SHANE J. NHO
MICHAEL LEUNIG
CHRISTOPHER M. LARSON
ASHEESH BEDI
BRYAN T. KELLY
EDITORS

Hip Arthroscopy and Hip Joint Preservation Surgery

EXTRAS ONLINE

Springer Reference

Hip Arthroscopy and Hip Joint Preservation Surgery

Shane J. Nho • Michael Leunig
Christopher M. Larson
Asheesh Bedi • Bryan T. Kelly
Editors

Hip Arthroscopy and Hip Joint Preservation Surgery

With 933 Figures and 86 Tables

Springer Reference

Editors
Shane J. Nho
Hip Preservation Center
Department of Orthopedic Surgery
Rush Medical College of Rush University
Rush University Medical Center
Chicago, IL, USA

Michael Leunig
Department of Orthopaedic Surgery Schulthess Klinik
Zurich, Switzerland

Christopher M. Larson
Minnesota Orthopedic Sports Medicine Twin Cities Orthopedics - Edina
Edina, MN, USA

Asheesh Bedi
Department of Orthopaedics
University of Michigan
Ann Arbor, MI, USA

Bryan T. Kelly
Sports Medicine and Shoulder Service and Center for Hip Preservation Surgery
Hospital for Special Surgery
New York, NY, USA

ISBN 978-1-4614-6964-3 ISBN 978-1-4614-6965-0 (eBook)
ISBN 978-1-4614-6966-7 (print and electronic bundle)
DOI 10.1007/978-1-4614-6965-0
Springer New York Heidelberg Dordrecht London

Library of Congress Control Number: 2014952885

Printed on acid-free paper

Springer is part of Springer Science+Business Media (www.springer.com)

Foreword

It is an honor to prepare the Foreword for this magnificent work. While the title covers the latest technology in hip arthroscopy, a topic near and dear to my heart, it is also the most comprehensive title ever published on the subject of hip preservation. The understanding of hip disorders and strategies of management is the most rapidly evolving field in orthopedics today. The knowledge sharing around the world transcends traditional political boundaries, and the lot of hip arthroscopists and hip preservationists truly is a global community. This expertly crafted title reflects this global nature as the editors have assembled the work of the world's thought leaders into a 99-chapter compendium on this complex and challenging discipline. The editors masterfully simplify the subject by breaking it down to its elements, and the authors deftly dissect the details of each topic.

This is a timely work when there is such a scientific and clinical explosion in the hip realm. Dysplasia and its consequences have been recognized for many years, and Bill Harris has been telling us for decades that there is no such thing as primary osteoarthritis of the hip. So why is it now that there has been such an influx? Three independent forces were at work in the world. Arthroscopists were looking in hips recognizing the existence of painful disorders that were previously undiagnosed. This forced more careful evaluation of hip complaints. Arthroscopic techniques have now evolved into endoscopic methods for disorders around the hip, providing a less invasive approach for traditionally recognized problems and also identifying new disorders. Meanwhile, Professor Ganz and his colleagues in Bern tied together the principles previously proposed by Dr. Harris and many others and gave us the concept of femoroacetabular impingement as a frequent causative factor in hip problems. Lastly, for years, general surgeons have been trying to meet the challenges of athletic pubalgia and other compensatory pelvic disorders. Eventually, we recognized that we were each identifying various components of a common problem, especially in the form of sports hip injuries.

Thus, with so much happening on the horizon, the users will find this comprehensive work to be a valuable resource for years to come. Congratulations to the editors and the authors. More importantly, thank you to their supportive families, tireless coworkers, and staff essential to each of them and, most importantly, the patients who honor us by putting their faith and trust in our care.

J. W. Thomas Byrd, MD

Preface

The diagnosis and treatment of hip pain and pathology, from infant to adult, was described years ago. This field has grown exponentially over the past decade largely as a result of Professor Ganz's description and research regarding the concept of femoroacetabular impingement (FAI). Dr. Poirier, in 1899, first wrote the description of mechanical hip joint impingement called "empreinte ilaque," or an impression of the anterior inferior iliac spine on the proximal femoral head–neck junction. In 1936, Dr. Smith-Petersen, at the Massachusetts General Hospital, described "impingement of the femoral neck on the anterior acetabular margin" leading to "traumatic arthritis." In 1965, Dr. Murray provided the description of the "tilt deformity" of the proximal femur that was associated with the development of osteoarthritis. It was not until 1974 when Drs. Stulberg and Harris and 1976 when Dr. Solomon looked at additional causes of hip osteoarthritis including pediatric deformities such as developmental hip dysplasia, slipped capital femoral epiphysis, Legg-Calve-Perthes disease, multiple epiphyseal dysplasia, and spondyloepiphyseal dysplasia.

The concept of FAI as defined by Professor Ganz in 2003 was a pivotal point in hip preservation. He defined the importance of subtle deformities of the proximal femur and acetabulum and the potential link to progressive chondrolabral damage and end-stage hip osteoarthritis. Drs. Bedi, Leunig, and Kelly described the static and mechanical causes of hip pain encompassing the spectrum of hip deformities causing either dynamic stress with the hip in motion or static overload stress due to hip instability. Meanwhile, orthopedic surgeons from many different disciplines were observing and describing hip deformities and treatment of nonarthritic hip pathology. Some hip surgeons were treating intra-articular pathology and deformities with surgical hip dislocation and corrective pelvic and femoral osteotomies. Other surgeons were developing arthroscopic techniques and were able to access the hip joint and perform correction of soft-tissue pathology and osseous deformities. Both open and arthroscopic surgical approaches as well as surgical indications continue to evolve, and the roles of these various procedures and approaches are becoming better defined.

As hip pathology affects many subspecialties of orthopedic surgery, our goal was to provide a definitive reference for any physician, orthopedic surgeon, or scientist regarding the treatment of the hip from infant to adult. There are textbooks on hip arthroscopy, pediatric hip surgery, adult hip, and open hip surgery. However, a comprehensive handbook was necessary as our understanding of hip pathology continues to grow at a very fast pace, and these fields are correspondingly becoming more closely and intricately linked with one another.

We chose a title that was meant to reflect the comprehensive nature of the handbook and to include open and arthroscopic hip preservation techniques and approaches. The handbook is designed to provide fundamental knowledge regarding basic science, imaging studies, and nonsurgical treatment of hip pain and pathology. The section on operative hip basics for arthroscopic and open hip preservation surgery is intended to provide a detailed description of the setup and surgical approach so that the subsequent chapters can focus on the operative technique relevant to the specific pathology. Each section is divided by subspecialty and organized by specific disorders, which are then followed by a series of surgical technique chapters for each specific disorder. The goal is to develop a reference for surgeons that incorporates the most current research in addition to a detailed description of the surgical technique by the preeminent experts in the field. In addition, many of the surgical techniques have accompanying multimedia content that can be accessed. The overall result is 99 chapters dedicated to hip disorders from hundreds of authors who are the current and future leaders in this field.

We believe that this handbook provides comprehensive information in an efficient manner resulting in an invaluable reference in the office, laboratory, clinic, and operating room. We are also delighted that *Hip Arthroscopy and Hip Preservation Surgery* will be accessible through SpringerReference, which allows the readers to access the content online as well. We had the great pleasure of combining the insights of the foremost orthopedic surgeons in the world to contribute to this handbook and believe that it will be the definitive reference for hip arthroscopy and hip preservation surgery.

September 2014

Shane J. Nho
Chicago, IL, USA
Michael Leunig
Zurich, Switzerland
Christopher M. Larson
Edina, MN, USA
Asheesh Bedi
Ann Arbor, MI, USA
Bryan T. Kelly
New York, NY, USA

About the Editors

Shane Nho is an Assistant Professor at Rush Medical College of Rush University and Rush University Medical Center in Chicago, Illinois, USA. He is a team physician for the Chicago Bulls, Chicago White Sox, Chicago Steel, and Roosevelt University. Dr. Nho specializes in arthroscopic and reconstructive surgery for the shoulder, hip, and knee.

Dr. Nho completed his undergraduate studies at Northwestern University and graduated with honors in Biology. He enrolled in the combined MD/MS program at Rush Medical College and the Graduate College of Rush University under the mentorship of Brian J. Cole, MD, MBA. He completed his surgical internship at New York Presbyterian Hospital of Weill Cornell Medical College and a residency in orthopedic surgery at the Hospital for Special Surgery in New York. Dr. Nho returned to Rush University Medical Center to complete a fellowship in sports medicine under the direction of Bernard R. Bach, Jr., MD.

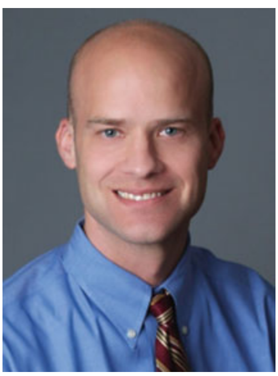
Dr. Nho was the recipient of the Herodicus Society Traveling Fellowship and has trained with hip arthroscopists and hip joint preservation surgeons from the United States and Switzerland. He was also selected as North American Traveling Fellow through the American Orthopaedic Association.

Dr. Nho is the cohead of the Hip Study Group, which is a multidisciplinary group of clinicians and scientists to conduct research directed at studying hip joint preservation and clinical outcomes after hip surgery at Rush University Medical Center. He has authored over 150 peer-reviewed articles, chapters, and publications in the field of hip preservation, sports medicine, and reconstructive surgery of the shoulder, hip, and knee.

Michael Leunig is currently Head of Orthopedics at the Schulthess Clinic in Zürich, Switzerland. The Schulthess Clinic is the largest orthopedic Hospital in Switzerland and one of the largest centers in Europe. Academically, Dr. Leunig has been affiliated with the University of Berne. His busy hip practice spans the entire spectrum from joint preservation to hip replacement and revision surgery. He received his orthopedic training under Professor Reinhold Ganz in Berne, Switzerland.

Based on his accomplishments, Dr. Leunig was elected a member of the International Hip Society in 2005 and currently serves on the Board. In addition, he is a member of several international research networks such as ANCHOR and MAHORN as well as a board member of the Müller Foundation North America and ISOT. His current research activities, inspired by his work with Professor Ganz, focus almost exclusively on the hip such as the prevalence of femoroacetabular impingement (FAI). He has described several surgical techniques for FAI and leads a research group focusing on the outcomes of FAI surgery.

So far, Dr. Leunig has published more than 200 PubMed-listed papers, his H-factor is 43, and he has edited several books and authored several book chapters. Besides, he has been an invited speaker at most large orthopedic conferences (EFORT, SICOT, AAOS, ISAKOS, etc.) and is a recipient of several awards recognizing his accomplishments. Apart from serving as reviewer for all large orthopedic journals, Dr. Leunig is International Associated Editor of *Clinical Orthopedics*, Associate Editor of *Orthopaedic Journal of Sport Medicine*, and Deputy Editor of *Journal of Hip Preservation Surgery*. His research has been funded by national agencies such as the Swiss National Science Foundation for many years.



Christopher M. Larson is an orthopedic sports medicine surgeon who specializes in hip and knee sports medicine. He serves as program director for the ACGME-accredited MOSMI/Fairview Orthopedic Sports Medicine Fellowship Program. He is recognized for his commitment to sports medicine research and education and has helped to develop and pioneer various sports medicine surgical techniques.

Dr. Larson has authored and published numerous textbooks and book chapters and has published over 80 articles in peer-reviewed journals regarding hip and knee sports medicine. In addition to sports medicine research, he lectures nationally and internationally, having been an invited lecturer in countries such as Switzerland, Germany, Denmark, France, Japan, Argentina, Turkey, Brazil, and Canada.

Dr. Larson also provides coverage for a number of sports teams. He was formerly a team physician for the Minnesota Vikings (NFL) and is currently a team physician for the Minnesota Wild (NHL). Dr. Larson's specialties include hip arthroscopy and arthroscopic hip joint preservation, ACL reconstruction and knee sports medicine, and proximal hamstring and hip abductor injuries/ surgery.

Asheesh Bedi is the Harold and Helen W. Gehring Early Career Professor of Orthopaedic Surgery and an Associate Professor of Sports Medicine and Shoulder Surgery at the University of Michigan and the MedSport Program. He is also an Adjunct Assistant Professor at the Hospital for Special Surgery/Weill Cornell Medical College. Dr. Bedi is a team physician for the University of Michigan and Eastern Michigan University. He specializes in arthroscopic hip preservation surgery as well as arthroscopic and open surgery for athletic injuries of the shoulder, elbow, and knee.

Dr. Bedi completed his undergraduate training at Northwestern University, where he graduated Summa Cum Laude. He graduated from the University of Michigan Medical School with AOA recognition, and remained in Ann Arbor to pursue residency training in Orthopaedic Surgery at the University of Michigan. After completing his training, Dr. Bedi completed a two-year fellowship in sports medicine and hip preservation at the Hospital for Special Surgery and Weill Cornell Medical College in New York. He has authored over 200 articles, chapters, and peer-reviewed publications on shoulder, elbow, knee, and hip injuries in athletes. Dr. Bedi has a number of ongoing funded translational research investigations studying joint preservation and novel methods to improve recovery after athletic injury.

Bryan T. Kelly is a specialist in sports medicine injuries and arthroscopic and open surgical management of nonarthritic disorders around the hip. He has faculty appointments at New York Presbyterian's Weill Cornell Medical College as well as the Hospital for Special Surgery. He cares for several sports teams, serving as an Associate Team Physician for the New York Giants and the New York Red Bull's MLS team as well as team Consultant for hip injuries for the New Jersey Nets and several collegiate teams in the tristate region.

After the completion of his residency at the Hospital for Special Surgery (HSS) in 2001, Dr. Kelly completed a 2-year fellowship at the same institution, specializing in Sports Medicine and Shoulder Surgery. He then completed a fellowship in Hip Sports Injuries and Arthroscopy at the University of Pittsburgh Medical Center, Center for Sports Medicine, under the direction of Dr. Marc J. Philippon, MD. Prior to starting his practice, he also completed an AO International Traveling Fellowship, whereby he spent time with Dr. Herbert Resch at the Landeskliniken Hospital in Salzburg, Austria. Dr. Kelly also spent time with Professor Reinhold Ganz and Dr. Michael Leunig in Bern and Zurich, Switzerland, studying advanced techniques in open surgical management of hip injury and deformity through an AO Surgical Preceptorship.

Dr. Kelly currently serves as Codirector for the Center for Hip Preservation at the Hospital for Special Surgery, which is designed to provide multidisciplinary care for patients at all levels with hip injuries. He has a broad range of both clinical and basic science research interests including the development of a clinical outcomes registry, biomechanical studies evaluating conflict patterns in femoroacetabular impingement and techniques in labral refixation, development of synthetic scaffolds for labral reconstruction and cartilage injuries in the hip, and the development of novel surgical techniques for managing soft tissue injuries around the hip joint. Dr. Kelly has authored over 120 scientific publications, book chapters, review articles, and books.

Contents

Volume 1

Contributors

Geoffrey D. Abrams Department of Orthopaedic Surgery, Stanford University, Palo Alto, CA, USA

Sol M. Abreu-Sosa Clinica de Medicina Fisica y Electrodiagnostico (CMFE), Aguada, PR, USA

Buen Samaritano Hospital, Aguadilla, PR, USA

William B. Acker II University of Michigan, Ann Arbor, MI, USA

Jeffrey Ackman Shriners Hospital for Children, Chicago, IL, USA

Ronald S. Adler Center for Musculoskeletal Care, NYU Langone Medical Center, New York, NY, USA

Jaimo Ahn Orthopaedic Trauma and Fracture Service, Department of Orthopaedic Surgery, University of Pennsylvania, Philadelphia, PA, USA

Christoph E. Albers Department of Orthopedic Surgery, Inselspital, University of Bern, Bern, Switzerland

Stephen K. Aoki Department of Orthopaedics, University of Utah Health Care, Salt Lake City, UT, USA

John M. Arnold The Hughston Clinic, PC, Columbus, GA, USA

Shin Azegami Royal National Orthopedic Hospital, Stanmore, Middlesex, UK

Keith Bachmann Cleveland Clinic Foundation, Cleveland, OH, USA

Ali S. Bajwa The Villar Bajwa Practice, Impington, Cambridge, UK

Champ L. Baker Jr. Department of Orthopaedics, Medical College of Georgia, Augusta, GA, USA

Champ L. Baker III The Hughston Clinic, PC, Columbus, GA, USA

Keith Baldwin Children's Hospital of Philadelphia, Philadelphia, PA, USA

Samuel J. Barke The EOC, Epsom General Hospital, Epsom, Surrey, England

Brian T. Barlow Orthopaedic Chief Resident, Naval Medical Center, San Diego, USA

Nicholas C. Barnthouse School of Medicine, University of Kansas City, Kansas City, KS, USA

Brandon Beamer Boston Children's Hospital, Boston, MA, USA

Paul E. Beaulé The Ottawa Hospital, Adult Reconstruction, University of Ottawa, Ottawa, ON, Canada

Martin Beck Department of Orthopaedics, Luzerner Kantonsspital, Luzern, Switzerland

James T. Beckmann Department of Orthopaedics, University of Utah Health Care, Salt Lake City, UT, USA

Asheesh Bedi Department of Orthopaedic Surgery, University of Michigan, Ann Arbor, MI, USA

Srino Bharam Department of Orthopaedic Surgery, Lenox Hill Hospital, New York, NY, USA

Patrick M. Birmingham Orthopaedic Sports Medicine, NorthShore University Health Systems, Chicago, IL, USA

Bernd Bittersohl Faculty, Orthopedic Department, University Hospital Dusseldorf, Dusseldorf, Germany

Edward D.R. Bray The EOC, Epsom General Hospital, Epsom, Surrey, England

Robert L. Buly Hospital for Special Surgery, Surgical Arthritis and Hip Preservation Services, Weill Medical College of Cornell University, New York, NY, USA

Charles A. Bush-Joseph Rush University Medical Center, Chicago, IL, USA

J. W. Thomas Byrd Nashville Sports Medicine Foundation, Nashville, TN, USA

K. Carpentier AZ St Blasius, Dendermonde, Heverlee, Belgium

Dominic Carreira Broward Health Imperial Point, Orthopedic Surgery – Foot and Ankle, Broward Health Medical Center, Fort Lauderdale, FL, USA

Antonia F. Chen The Rothman Institute, Thomas Jefferson University, Philadelphia, PA, USA

Jesse Chrastil University of Utah Health Care Orthopaedic Center, Salt Lake City, UT, USA

Struan H. Coleman Sports Medicine, Hospital for Special Surgery, New York, NY, USA

Geoffrey D. Cornelsen Minnesota Orthopedic Sports Medicine Institute, Twin Cities Orthopedics, Edina, MN, USA

K. Corten Department of Orthopaedic Surgery, Ziekenhuis Oost-Limburg, Genk, Belgium

Gregory Cvetanovich Department of Orthopaedic Surgery, Rush University Medical Center, Chicago, IL, USA

Benjamin G. Domb Hinsdale Orthopaedics Associates, Hinsdale, IL, USA

The American Hip Institute, Hinsdale, IL, USA

Loyola University, Stritch School of Medicine, Maywood, IL, USA

Sheila A. Dugan Rush Medical College, Departments of PM&R and Preventive Medicine, Rush University Medical Center, Chicago, IL, USA

Thomas J. Ellis Department of Orthopaedics, Ohio State University, Columbus, OH, USA

Brandon J. Erickson Department of Orthopaedic Surgery, Rush University Medical Center, Chicago, IL, USA

Jill Erickson University of Utah Health Care Orthopaedic Center, Salt Lake City, UT, USA

Scott C. Faucett Department of Hip Arthroscopy, Center for Outcomes-based Orthopaedic Research, Steadman Philippon Research Institute, Vail, CO, USA

Andrew E. Federer Midwest Orthopaedics at Rush, Hip Preservation Center, Rush University Medical Center, Chicago, IL, USA

Yale A. Fillingham Department of Orthopaedic Surgery, Rush University Medical Center, Chicago, IL, USA

Jared R.H. Foran Panorama Orthopaedics and Spine Center, Golden, CO, USA

Jonathan M. Frank Department of Orthopaedic Surgery, Rush University Medical Center, Chicago, IL, USA

Rachel M. Frank Department of Orthopaedic Surgery, Rush University Medical Center, Chicago, IL, USA

Luca Gala The Ottawa Hospital, Adult Reconstruction, University of Ottawa, Ottawa, ON, Canada

Reinhold Ganz Emeritus Inselspital Bern, Bern, Switzerland

Faculty of Medicine, University of Bern, Bern, Switzerland

Andrew G. Georgiadis Department of Orthopaedic Surgery, Henry Ford Hospital, Detroit, MI, USA

Philip J. Glassner Hunterdon Orthopedic Institute, Flemington, NJ, USA

Ryan Goodwin Cleveland Clinic Foundation, Cleveland, OH, USA

Carlos A. Guanche Southern California Orthopedic Institute, Van Nuys, CA, USA

Anil Gupta Department of Orthopaedic Surgery, Rush University Medical Center, Chicago, IL, USA

Asheesh Gupta Hinsdale Orthopaedics Associates, Hinsdale, IL, USA

The American Hip Institute, Hinsdale, IL, USA

Jason J. Halvorson Medical Center Boulevard, Hospital for Special Surgery, Wake Forest University School of Medicine, Winston-Salem, NC, USA

Mathew Hamula Department of Orthopaedic Surgery, Lenox Hill Hospital, New York, NY, USA

Joshua D. Harris Houston Methodist Center for Orthopaedics & Sports Medicine, Houston, TX, USA

Rush University Medical Center, Chicago, IL, USA

Thomas W. Hash II Musculoskeletal Section, Department of Radiology, Duke University Medical Center, Durham, NC, USA

Munif A. Hatem Baylor University Medical Center, Hip Preservation Center, Dallas, TX, USA

David L. Helfet Weill Medical College of Cornell University, Orthopaedic Trauma Service, Hospital for Special Surgery/New York Presbyterian Hospital, New York, USA

Michael D. Hellman Department of Orthopaedic Surgery, Rush University Medical Center, Chicago, IL, USA

Benton E. Heyworth Division of Sports Medicine, Boston Children's Hospital, Instructor in Orthopaedic Surgery, Harvard Medical School, Boston, MA, USA

Jenna Hodge Howard Head Sports Medicine, Vail Valley Medical Center, Vail, CO, USA

Harish S. Hosalkar Sharp Grossmont Hospital, La Mesa, Paradise Valley Hospital, National City, Vista, CA, USA

Matthew T. Houdek Department of Orthopedic Surgery, Mayo Clinic, Rochester, MN, USA

Elizabeth A. Howse Department of Emergency Medicine, Long Island Jewish Medical Center, Hofstra North Shore-LIJ School of Medicine, New Hyde Park, NY, USA

Atul F. Kamath Department of Orthopaedic Surgery, University of Pennsylvania, Philadelphia, PA, USA

Richard W. Kang Sports Medicine and Shoulder Surgery, Hospital for Special Surgery, New York, NY, USA

James S. Keene Division of Sports Medicine, University of Wisconsin, Madison, WI, USA

Bryan T. Kelly Department of Orthopaedic Surgery, Center for Hip Preservation Surgery, Hospital for Special Surgery, New York, NY, USA

Weill Medical College of Cornell University, New York, NY, USA

Michael Knesek Department of Orthopaedic Surgery, University of Michigan, Ann Arbor, MI, USA

Mininder S. Kocher Harvard Medical School Children's Hospital Boston, Boston, MA, USA

Division of Sports Medicine, Orthopedic Surgeon, Boston Children's Hospital, Boston, MA, USA

Monica Kogan Department of Orthopaedic Surgery, Rush University Medical Center, Chicago, IL, USA

Pediatric Orthopaedic Surgery, Rush University Medical Center, Chicago, IL, USA

Dirk Kokmeyer Howard Head Sports Medicine, Vail Valley Medical Center, Vail, CO, USA

Joel Kolmodin Cleveland Clinic Foundation, Cleveland, OH, USA

Aaron Krych Department of Orthopedic Surgery, Mayo Clinic, Rochester, MN, USA

Joseph Kuechle Department of Orthopedics, State University of New York, Buffalo, NY, USA

Catalina Larrain Orthopedics Department, Clínica Las Condes, Santiago, Chile

Christopher M. Larson Minnesota Orthopedic Sports Medicine Institute, Twin Cities Orthopedics, Edina, MN, USA

Nicholas J. Lash Department of Orthopaedics, Luzerner Kantonsspital, Luzern, Switzerland

Simon Lee Department of Orthopaedic Surgery, Midwest Orthopaedics at Rush, Rush University Medical Center, Chicago, IL, USA

Michael Leunig Department of Orthopaedic Surgery, Schulthess Clinic, Zürich, Switzerland

Travis G. Maak Department of Orthopaedics, University of Utah Health Care, Salt Lake City, UT, USA

G. Peter Maiers II Department of Orthopaedic Surgery, Indiana University School of Medicine, Indianapolis, IN, USA

Molly A. Malloy The University of Chicago Medical Center, Chicago, IL, USA

Philip J. Malloy Program in Exercise Science, Department of Physical Therapy Graduate Research Assistant, Marquette University, Milwaukee, WI, USA

John P. Manalo Department of Orthopaedic Surgery, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA

Anthony Marchie Mackenzie Health, Richmond Hill, ON, Canada

Rodrigo Mardones Orthopedics Department, Clínica Las Condes, Santiago, Chile

Niv Marom Department of Orthopaedic Surgery, Lenox Hill Hospital, New York, NY, USA

Department of Orthopaedic Surgery, Meir Medical Center, Kfar saba, Israel

Hal D. Martin Baylor University Medical Center, Hip Preservation Center, Dallas, TX, USA

John R. Martin Department of Orthopedic Surgery, Mayo Clinic, Rochester, MN, USA

Randy Mascarenhas Department of Orthopaedic Surgery, Midwest Orthopaedics at Rush, Rush University Medical Center, Chicago, IL, USA

Richard C. Mather III Department of Orthopaedic Surgery, Duke University School of Medicine, Durham, NC, USA

Dean K. Matsuda Department of Orthopedic Surgery, Kaiser West Los Angeles Medical Center, Los Angeles, CA, USA

Joseph C. McCarthy Harris Orthopaedic Laboratory, Massachusetts General Hospital, Boston, MA, USA

Joint Reconstruction Center, Newton–Wellesley Hospital, Boston, MA, USA

Frank McCormick Hip Preservation Center, Division of Sports Medicine, Department of Orthopaedic Surgery, Rush University Medical Center, Rush Medical College, Rush University, Chicago, IL, USA

Brian McGrath Department of Orthopedics, State University of New York, Buffalo, NY, USA

Samir Mehta Hospital of the University of Pennsylvania, Orthopaedic Surgery, Penn Orthopaedics, Philadelphia, PA, USA

Joshua Minori The Rothman Institute, Thomas Jefferson University, Philadelphia, PA, USA

Nicholas G. H. Mohtadi Sport Medicine Centre and Section of Orthopaedic Surgery, University of Calgary, Calgary, AB, Canada

Jozef Murar Orthopaedic Surgery Resident, Department of Orthopaedic Surgery, Northwestern University, Chicago, IL, USA

Shane J. Nho Department of Orthopaedic Surgery, Rush University Medical Center, Chicago, IL, USA

Midwest Orthopaedics at Rush, Hip Preservation Center, Rush University Medical Center, Chicago, IL, USA

Eziamaka Okafor Harvard's Brigham and Women's Hospital, Boston, MA, USA

Patrick O'Toole The Rothman Institute, Thomas Jefferson University, Philadelphia, PA, USA

Ian J. Palmer Baylor University Medical Center, Hip Preservation Center, Dallas, TX, USA

Ian Panuncialman Harris Orthopaedic Laboratory, Massachusetts General Hospital, Boston, MA, USA

Caroline Park Sports Medicine and Shoulder Surgery, Hospital for Special Surgery, New York, NY, USA

Javad Parvizi The Rothman Institute, Thomas Jefferson University, Philadelphia, PA, USA

Ronak M. Patel Department of Orthopaedic Surgery, Northwestern University – Feinberg School of Medicine, Chicago, IL, USA

Anay Patel Department of Orthopaedic Surgery, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA

Christopher Pelt University of Utah Health Care Orthopaedic Center, Salt Lake City, UT, USA

Christopher Peters University of Utah Health Care Orthopaedic Center, Salt Lake City, UT, USA

Marc J. Philippon Department of Hip Arthroscopy, Center for Outcomes-based Orthopaedic Research, Steadman Philippon Research Institute, Vail, CO, USA

David A. Podeszwa Department of Orthopaedic Surgery, University of Texas Southwestern Medical Center, Dallas, TX, USA

Texas Scottish Rite Hospital for Children, Dallas, TX, USA

Lazaros A. Poultsides Department of Orthopaedic Surgery, Center for Hip Pain and Preservation, Hospital for Special Surgery, New York, NY, USA

Anil Ranawat Sports Medicine and Shoulder Surgery, Hospital for Special Surgery, New York, NY, USA

Monica Rho Harvard's Brigham and Women's Hospital, Boston, MA, USA

Patrick Riley Jr. Akron Children's Hospital, Akron, OH, USA

Leon Root Hospital for Special Surgery, New York, NY, USA

James Rosneck Sports Health Center, Orthopaedic and Rheumatologic Institute, Cleveland Clinic, Garfield Heights, OH, USA

James R. Ross Sports Medicine and Shoulder Service, Department of Orthopaedic Surgery, University of Michigan, Ann Arbor, MI, USA

David S. Ruch Duke University Health System, Durham, NC, USA

John M. Ryan Department of Orthopaedics, Division of Hip Preservation, Ohio State University, Columbus, OH, USA

Marc R. Safran Stanford University School of Medicine, Redwood City, CA, USA

Michael J. Salata Department of Orthopaedic Surgery, Joint Preservation and Cartilage Restoration Center, University Hospitals Case Medical Center, Cleveland, OH, USA

Thomas G. Sampson Medical Director of Hip Arthroscopy, Post Street Surgery Center, San Francisco, CA, USA

Post Street Orthopaedics and Sports Medicine, San Francisco, CA, USA

Wudbhav N. Sankar Division of Orthopaedic Surgery, Perelman School of Medicine, University of Pennsylvania, Children's Hospital of Philadelphia, Philadelphia, PA, USA

Mara L. Schenker Orthopaedic Trauma and Fracture Service, Department of Orthopaedic Surgery, University of Pennsylvania, Philadelphia, PA, USA

Matthew Schmitz San Antonio Military Medical Center, Houston, TX, USA

Christopher M. Shaw University of Missouri-Kansas City, Kansas City, MO, USA

Klaus A. Siebenrock Department of Orthopedic Surgery, Inselspital, University of Bern, Bern, Switzerland

Rafael J. Sierra Department of Orthopedic Surgery, Mayo Clinic, Rochester, MN, USA

Jack Skendzel Summit Orthopedics, Woodbury, MN, USA

William Slikker III Midwest Orthopaedics at Rush, Chicago, IL, USA

Luke Spencer-Gardner Department of Orthopedic Surgery, Mayo Clinic, Rochester, MN, USA

David Spiegel Division of Orthopaedic Surgery, Children's Hospital of Philadelphia, Philadelphia, PA, USA

Giles H. Stafford The EOC, Epsom General Hospital, Epsom, Surrey, England

Michael Stanek University of Illinois, Champaign, IL, USA

Simon D. Steppacher Department of Orthopedic Surgery, Inselspital, University of Bern, Bern, Switzerland

Rebecca M. Stone Minnesota Orthopedic Sports Medicine Institute, Twin Cities Orthopedics, Edina, MN, USA

Michael D. Stover Department of Orthopaedic Surgery, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA

Allston J. Stubbs Surgical Sciences-Orthopaedics, Wake Forest Baptist Medical Center, Winston-Salem, NC, USA

Moritz Tannast Department of Orthopedic Surgery, Inselspital, University of Bern, Bern, Switzerland

Laura E. Thorp Department of Anatomy and Cell Biology, Rush University Medical Center, Chicago, IL, USA

Lisa M. Tibor Kaiser Permanente Medical Center, South San Francisco, CA, USA

Ronald Tsao Rush University Medical Center, Chicago, IL, USA

Craig J. Della Valle Midwest Orthopaedics at Rush, Rush University Medical Center, Chicago, IL, USA

Geoffrey S. Van Thiel Orthopedics and Orthopedic Surgery, Rockford Orthopedic Associates, Rockford, IL, USA

Rush University Medical Center, Chicago, IL, USA

Richard N. Villar The Richard Villar Practice, Spire Cambridge Lea Hospital, Impington, Cambridge, UK

Walter Virkus Department of Orthopaedic Surgery, Indiana University School of Medicine, IU Health Methodist Hospital, Indianapolis, IN, USA

James E. Voos Division of Sports Medicine, Department of Orthopaedic Surgery, University Hospitals Case Medical Center, Cleveland, OH, USA

Jonathan N. Watson Department of Orthopaedic Surgery, University of Illinois, Chicago, IL, USA

Alexander E. Weber Sports Medicine and Shoulder Service, Department of Orthopaedic Surgery, University of Michigan, Ann Arbor, MI, USA

Kathleen Weber Midwest Orthopaedics at Rush, Rush University Medical Center, Chicago, IL, USA

Jeffrey A. Weiss Departments of Bioengineering and Orthopaedics, and Scientific Computing and Imaging Institute, University of Utah, Salt Lake City, UT, USA

Ryan H. Wood The University of Chicago Medical Center, Chicago, IL, USA

Thomas H. Wuerz Hip Preservation Center, Department of Orthopaedic Surgery, Division of Sports Medicine, Rush University Medical Center, Rush Medical College of Rush University, Chicago, IL, USA

Adam B. Yanke Hip Preservation Center, Department of Orthopaedic Surgery, Division of Sports Medicine, Rush University Medical Center, Rush Medical College of Rush University, Chicago, IL, USA

Yi-Meng Yen Harvard Medical School Children's Hospital Boston, Boston, MA, USA

Division of Sports Medicine, Orthopedic Surgeon, Boston Children's Hospital, Boston, MA, USA

Ira Zaltz Department of Orthopaedic Surgery, William Beaumont Hospital, Royal Oak, MI, USA

Michael Zlowodzki Department of Orthopaedic Surgery, Indiana University School of Medicine, IU Health Methodist Hospital, Indianapolis, IN, USA

Part I

Hip Basic Science

Hip Anatomy

1

Laura E. Thorp

Contents

L.E. Thorp
Department of Anatomy and Cell Biology, Rush University Medical Center, Chicago, IL, USA
e-mail: laura_thorp@rush.edu

Abstract

This chapter reviews the bony, capsular, ligamentous, muscular, and neurovascular anatomies of the hip joint with specific attention to structures of greatest clinical relevance. The hip is a ball-and-socket joint formed by the union of the femoral head and acetabulum. As such it possesses the potential for triplanar motion similar to the shoulder joint, but because the hip functions primarily in closed kinematic chain motions, e.g., gait, a need for stability tempers the mobility available at this articulation. The capsule of the hip joint provides stability to the joint and is reinforced by the presence of the intrinsic capsular ligaments. Intra-articular structures such as the labrum and ligamentum teres are also elements of the hip joint anatomy which impact its function. Musculature of the hip joint contributes to both the mobility and stability of the hip and can be compartmentalized regionally into the gluteal muscles and the muscles of the anterior, medial, and posterior thighs. Innervation to the hip joint is provided by the nerves crossing the joint. Classically the obturator nerve is considered the primary source of innervation to the hip; however, branches of the femoral and sciatic nerves also contribute to its sensory innervation. The primary source of blood supply to the hip joint is the medial femoral circumflex artery with additional contributions from the femoral and gluteal vessels. Knowledge of the anatomic relationships between the

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_113

bony, ligamentous, muscular, and neurovascular structures is of great importance for surgical and nonsurgical treatments of hip joint pathology.

Introduction

The development of the extremities begins at approximately the fourth week of gestation. By the sixth week of development, the hyaline cartilage models of the limb bones are present in the developing embryo [1]. Joints of the limbs form when the process of chondrogenesis is interrupted and a joint interzone is induced to form. In this interzone region, cells initially increase in number and density, but subsequently a joint cavity is formed by the process of apoptosis [1]. Mesenchymal cells in the interzone differentiate into dense fibrous tissue which will form the articular cartilage at the end of the femur. The surrounding cells form the capsular structures [1]. While the molecular mechanisms behind the development of joints remain to be elucidated, it is believed that WNT14 plays a role in these molecular mechanisms [1]. Primary ossification centers in all long bones, including the femur, are present by 12 weeks of development, and the diaphysis of the femur should be completely ossified at birth, while the femoral epiphyses remain cartilaginous [1].

The hip joint is ball-and-socket-type class of synovial joint formed by the union of the acetabulum of the pelvis and the head of the femur (Fig. 1). The acetabulum itself is formed by the union of the three pelvis bones, the ilium, ischium, and pubis. The center of the hip joint lies approximately 1–2 cm distal to the middle third of the inguinal ligament. While described as a bony socket, the acetabulum is an incomplete sphere, with a gap at its inferior aspect (Fig. 2). This gap is spanned by the transverse acetabular ligament, which aids in maintaining the integrity of the articulation. As will be discussed further in this chapter, the acetabular labrum, joint capsule, capsular ligaments, and hip joint musculature all play a role in maintaining the integrity of this joint. Individual variability in hip joint range motion may be a consequence of individual differences in any of these anatomic components. The ball-and-socket architecture of the hip joint allows for triplanar motion in the sagittal, coronal, and horizontal/transverse planes. The joint arthrokinematic motions occurring between the acetabulum and the femoral head include spinning and gliding of the femoral head in the acetabulum. The resultant osteokinematic motions are flexion and extension in the sagittal plane, abduction and adduction in the coronal plane, and medial (internal) or lateral (external) rotation in the horizontal/transverse plane. During flexion and extension, respectively, there is spin of the head of the femur on the acetabulum accompanied by a slight posterior or anterior glide of the femoral head. In abduction the femoral head glides inferiorly in the acetabulum. Finally, the anterior or posterior glide of the femoral head on the acetabulum occurs at the joint surfaces during external (lateral) or internal (medial) rotation, respectively. Average active range of motion values are approximately 120 degrees of flexion and 20 degrees of extension in the sagittal plane, 40 degrees of abduction in the frontal plane, and 30 degrees of both internal and external rotation ranges of motion in the horizontal plane when measured in a seated position with 90 degrees of knee flexion [2]. When examined as a variable of age, variations in range of motion between the youngest and oldest individuals are small and probably of little clinical significance with the exception of hip extension where a 20 % decrease in range of motion has been observed in the older individuals as compared with the young [2]. The closed packed position of the hip, which yields its greatest stability, is a position of extension with some medial rotation and abduction. In this position, joint surfaces are maximally, though not completely, congruent.

Bony Anatomy and Alignment

Significant variability exists in the morphology of the proximal femur and acetabulum. Specific aspects of femoral and acetabular alignments have significant clinical relevance and impact joint biomechanics. Both the angle of inclination

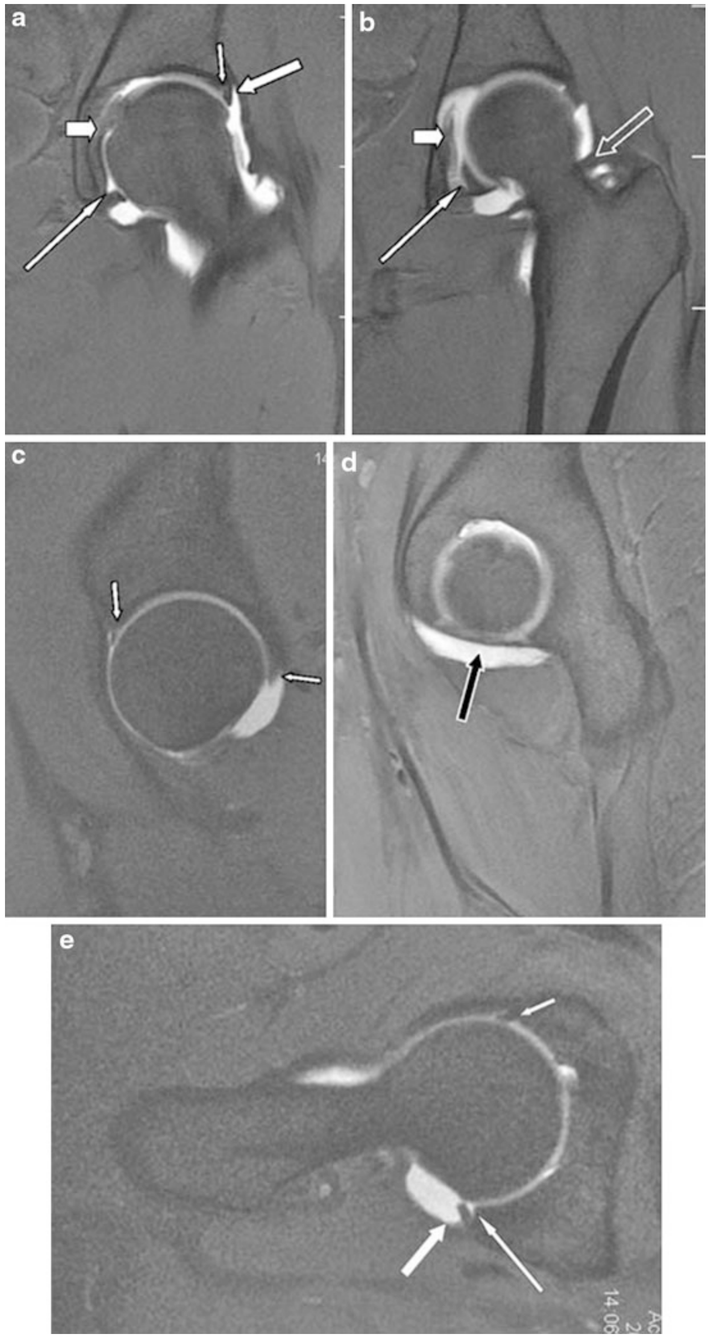


Fig. 1 Normal hip anatomy. Fat-suppressed T1-weighted coronal (**a**, **b**), sagittal (**c**, **d**), and axial oblique (**e**) images with intra-articular gadolinium. (**a**, **b**) The normal triangular-shaped labrum is shown by the *small thin arrow*. The joint capsule inserts several millimeters above the acetabular rim (*medium-sized arrow*) forming a normal recess. The ligamentum teres arises (*short thick arrow*) from the fovea capitis and extends to the transverse ligament. The *long thin arrows* depict the junction of the ligamentum teres and the transverse ligament. The circular fibers of the zona orbicularis (*open arrow*). (**c**) The triangular cross-section of the anterior and posterior labra (*arrows*). (**d**) The transverse ligament spans the acetabular notch (*arrow*). (**e**) The anterior triangular-shaped labrum (*small arrow*), the posterior joint capsule attachment at the base of the posterior labrum (*large thick arrow*), and the small posterior inferior sulcus at the junction of the transverse ligament and labrum (*long thin arrow*) (http://www.springerimages.com/Images/MedicineAndPublicHealth/1-10.1007_s00256-006-0105-5-0)

and the amount of femoral torsion are characteristics of femoral anatomy that affect the function of the hip joint. The angle of inclination, or neck-shaft angle, is formed by the axis of the head and neck of the femur with respect to the axis of the shaft of the femur. The normal angle is approximately 125 degrees [3]. The neck-shaft angle determines the size of the femoral offset, the distance from the center of rotation of the femoral head to a line bisecting the long axis of the femur. The neck-shaft angle of the femur and resultant femoral offset affect the mechanical

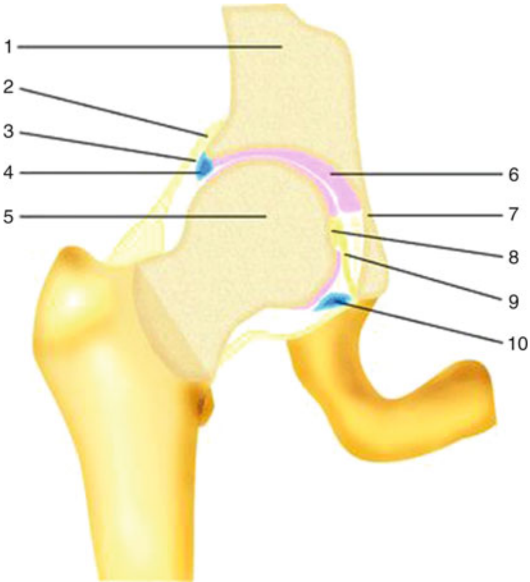


Fig. 2 Hip joint anatomy. (*1*) Ilium, (*2*) capsula articularis, (*3*) perilabral recess, (*4*) acetabular labrum, (*5*) head of femur, (*6*) articular cartilage, (*7*) acetabulum, (*8*) fovea, (*9*) ligamentum teres, (*10*) transverse ligament (http://www.springerimages.com/Images/MedicineAndPublicHealth/1-10.1007_s13244-010-0023-x-1)

advantage of the hip abductors. Decreased neck-shaft angles (coxa vara) increase the moment arm of the abductors and increase joint stability due to increased coverage of the femoral head in the acetabulum. A shorter, more valgus femoral neck reduces the offset and decreases the moment arm of the hip abductors requiring larger abductor forces and potentially increased contact forces at the hip joint. Femoral version (torsion) is defined as the angular difference between the axis of the femoral neck and transcondylar axis of the knee and, in adulthood, is normally about 15 degrees [3]. Anteversion is an increase in the angle of femoral torsion. While a decrease in the angle of femoral torsion is known as retroversion. Excessive anteversion and retroversion require compensatory rotation (medial and lateral, respectively) at the hip joint to maintain joint congruency. Excessive anteversion may also change the moment arm of the gluteus medius and thus decrease its efficiency. Specifically, increased femoral anteversion displaces the greater trochanter posteriorly thus decreasing the lever arm of the gluteus medius and subsequently its strength [4]. Hip contact forces have been shown to increase with increased femoral anteversion [5]. The angle formed by the line connecting the center of the femoral head and the lateral rim of the acetabulum forms an angle with vertical known as the central edge angle, with angles greater than 25 degrees being considered normal [3]. Examination of proximal femoral and acetabular alignment using computed tomography has demonstrated relationships between these measures [6]. Mean femoral neck version was found to be positively correlated with acetabular version [6]. A negative correlation was found between femoral version and acetabular inclination [6]. Femoral neck-shaft angle was positively correlated with acetabular version and negatively correlated with age [6]. Both acetabular version and central edge angle were observed to be positively correlated with female gender [6].

Capsular and Ligamentous Anatomy

The capsule of the hip joint consists of a cylindrical arrangement of dense fibers connecting the acetabulum and proximal femur through firm proximal attachments to the acetabular periosteum and distal attachments to the intertrochanteric line on the femur anteriorly [7]. The anterior attachments extend from the intertrochanteric line to the greater trochanter superiorly and to the lesser trochanter inferiorly [7]. Posteriorly it has been observed that the fibers of the capsule lack a direct distal attachment on the femur but the zona orbicularis forms "an arched free border" around the femoral neck medial to the intertrochanteric crest [7]. Where the acetabulum is incomplete inferiorly, the capsule is attached to the transverse acetabular ligament [7]. Thickness of the capsule has been observed to vary, but it is reinforced consistently by the capsular ligaments.

Anteriorly, the strongest of the capsular ligaments, the iliofemoral ligament, reinforces the capsule with longitudinally oriented fibers spanning between the intertrochanteric line and the ilial portion of acetabular rim and anterior inferior iliac spine (Fig. 3). It is suggested that this ligament is reinforced by the tendinous origin of the rectus femoris muscle [7].

Fig. 3 Anterior view of the right hip capsule. Observe the presence of the iliofemoral ligament with its two components: superior (*white arrows*) and inferior fascicles (*black arrows*) (http://www.springerimages.com/Images/MedicineAndPublicHealth/1-10.1007_978-1-4419-7925-4_7-8)

Fig. 4 Posterior view of the right hip joint. Observe the presence of the ischiofemoral ligament (*white arrows*) and femoral arcuate ligament (*black arrows*) that passes below the first one (http://www.springerimages.com/Images/MedicineAndPublicHealth/1-10.1007_978-1-4419-7925-4_7-9)

The iliofemoral ligament possesses two distinct thickened bands, superiorly and inferiorly along its course, with a thinner central region [7] (Fig. 4). Its fibers appear maximally taut in extension and lax in flexion of the hip [7]. It has been suggested that due to the position of this ligament anterior to the hip joint, it has a special role in the maintenance of erect posture, balancing the force of the body's weight on the femur during standing.

The pubofemoral ligament spans from its proximal attachment at the obturator crest and superior pubic ramus to the intertrochanteric fossa. An extracapsular portion of this ligament has been observed inferior to the acetabulum [7]. The intracapsular portion courses perpendicular to the zona orbicularis. Distally at the femoral attachment, the pubofemoral ligament blends with the inferior band of fibers of the iliofemoral ligament at the attachment site on the lesser trochanter [7]. This ligament has been observed to exhibit maximal tautness in abduction and laxity when the hip is in an adducted position [7].

The ischiofemoral ligament is attached to the acetabular rim and labrum and the inner surface of the greater trochanter. Posteriorly the ischiofemoral ligament reinforces the joint capsule and can be subdivided into two bands (superior and inferior) which both run in an oblique direction between the acetabular rim and labrum and the inner surface of the greater trochanter [7]. The thickness of the capsule is decreased posteriorly with thinner and looser fibers except at the distal attachment on the femur where the transversely oriented fibers of the zona orbicularis provide reinforcement [7]. This ligament has been suggested to restrict internal rotation due to the length and tautness of its fibers with this motion at the hip joint [7].

The zona orbicularis is a circumferential band of fibers which forms an annular ligament at the hip joint. It attaches superiorly on the femur at the greater trochanter, blending with the fibers of the ischiofemoral ligament [7]. It reinforces the hip joint capsule posteriorly, where its fibers create a sling around the femoral neck and comprise the distal free border of the joint capsule [7, 8].

The labrum and capsule with its associated ligaments and zona orbicularis provide stability to the hip joint during distraction [8]. The proximal to middle portions of the capsule and the zona orbicularis seem to have a particularly important role in resisting distractive forces [8]. Thickness of the joint capsule has also been linked to joint stability. The role of the joint capsule as a stabilizer is an especially important consideration in total hip arthroplasty (THA), as research has suggested that compromise of the capsule may be an important, if not the primary, cause of dislocation after arthroplasty surgery [9]. Because all of the ligaments are taught in hip extension, hip extension is a very stable, "closed packed" position of the joint.

The ligamentum teres was once considered a vestigial structure with no role in the stabilization or biomechanics of the hip joint; however, it is now suggested that this ligament is more than an embryonic remnant and may contribute to hip joint mechanics and pathology [10, 11]. The ligament teres arises from the transverse acetabular ligament as well as the ischial and pubic aspects of the acetabulum [11]. Its insertion site is into the fovea capitis of the femur which is a round area on the head of the femur devoid of articular cartilage. Histologically, the ligamentum teres is composed of well-organized bundles of type I, III, and IV collagen fibers [11]. It is invested by its own synovial membrane [10, 11]. An anterior branch of the posterior division of the obturator artery courses through the ligamentum teres, vascularizing it, and variable branches of this vessel extend into the femoral head. In some individuals these branches may contribute some, but not all, of the vascular support to the femoral head itself [10, 11]. It has been suggested that this ligament may function as an intrinsic stabilizer of the hip joint and may resist subluxation of the hip joint, but this remains to be definitively proven [10, 11].

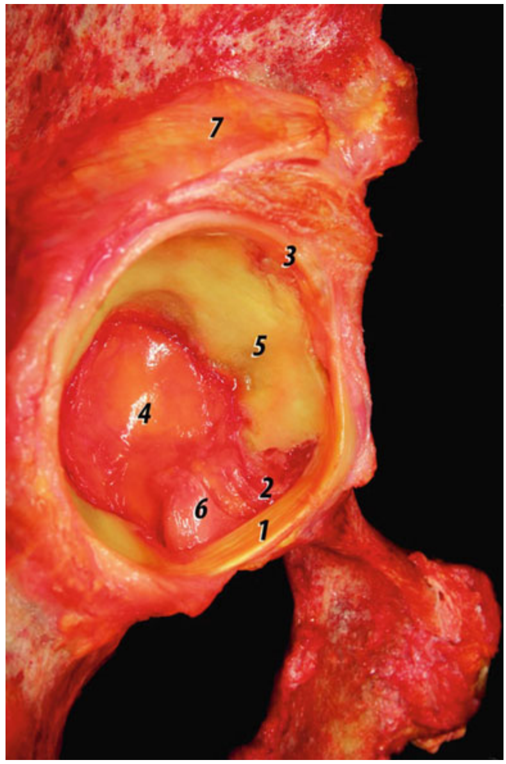


Fig. 5 Posterolateral view of a right acetabulum. Note the origin and attachment of the transverse acetabular ligament. (*1*) Transverse acetabular ligament. (*2*) Acetabular notch. (*3*) Acetabular labrum. (*4*) Acetabular fossa. (*5*) Anterior lunate cartilage. (*6*) Ligamentum teres (resected). (*7*) Reflected tendon of the rectus femoris muscle (http://www.springerimages.com/Images/MedicineAndPublicHealth/1-10.1007_978-1-4419-7925-4_7-7)

The acetabular labrum, also called the cotyloid ligament, is a fibrocartilagenous structure attached both along the perimeter of the bony acetabulum and to its articular surface via connections to the articular cartilage lining the acetabulum (Fig. 5). The labrum also has attachments to the transverse acetabular ligament anteriorly and posteriorly, and together these structures complete the socket of the hip joint [12, 13]. The labrum is primarily composed of type I collagen [12]; however, the attachment of these fibers to the acetabulum differs anteriorly and posteriorly in the joint. The anterior fibers are reported to attach parallel to the bony edge of the acetabulum making them susceptible to shear forces, while the posterior fibers attach perpendicular to the bony edge increasing their ability to resist shear stresses [13]. The labrum is separated from the joint capsule by the capsular recess [13]. The labrum appears horseshoe-shaped when viewed en face; however, in cross section, the labrum appears triangular in shape [12].

Muscular Anatomy

Muscles acting on the hip joint include muscles of the gluteal region (Fig. 6) as well as the muscles of the anterior, medial, and posterior compartments of the thigh. This section will review the attachment sites and actions of these regional groups of muscles with particular attention to those with the greatest clinical significance.

In the gluteal region muscles are classically divided into superficial and deep groups. The superficial group includes the gluteus maximus, gluteus medius, gluteus minimus, and the tensor fascia lata. Of this group, the gluteus maximus is the most superficial muscle with origins from the fascia of the gluteus medius, the ilium, the thoracolumbar fascia between the lower border of the posterior superior iliac spine and a point just lateral to the spinous process of the third sacral vertebrae, the aponeurosis of the erector spinae muscles, the sacrum, the coccyx, and the sacrotuberous ligament [14]. The insertion site for the majority of this muscle is on the iliotibial band at its aponeurotic origin on the greater trochanter of the femur; however, the inferior third of the muscular fascicles insert onto the gluteal tuberosity of the femur [14]. This muscle receives innervation from the inferior gluteal nerve, which arises from the ventral rami of the fifth lumbar as well as the first and second sacral spinal nerves. The inferior gluteal nerve emerges from the pelvis via the greater sciatic foramen inferior to the piriformis muscle. Attachments of the gluteus maximus to the thoracolumbar fascia and the aponeurosis of the erector spinae are not uniformly recognized in all anatomy texts. Recent literature suggests that these attachments and the large area

Fig. 6 Cadaveric dissection of the musculature of the gluteal region with the gluteus maximus reflected. The gluteus medius, piriformis, superior gemellus, tendon of the obturator internus, inferior gemellus, and quadratus femoris are visible in the dissection field. The sciatic nerve is visualized coursing inferior to the piriformis muscle (Photo reproduced with permission from Daniel Wasserman)

of the gluteus maximus which crosses posterior to the sacroiliac joint support a role for this muscle as a stabilizer of the sacroiliac joint via generation of compressive forces on this joint and may also assist in load transfer between the lower limbs and trunk [14]. These functions are in addition to this muscle's more classically described function of hip joint extension when the thigh is in a flexed position, such as in rising from a seated position. The gluteus maximus is also activated with forceful lateral rotation and abduction of the thigh. There is uniform agreement that the gluteus medius originates from the ilium between the anterior and posterior gluteal lines, while the iliac crest and the gluteal aponeurosis are also cited by some as proximal sites of attachment by some [15]. The gluteal minimus is classically described as arising from the external surface of the ilium between the anterior and inferior gluteal lines [15]. The gluteus minimus was extensively described in a cadaveric dissection study as proximally arising from the external ilium just anterior to the anterior superior iliac spine and then running parallel to the iliac crest up to the iliac tubercle [16]. Along the anterior gluteal line, the gluteus minimus is reported to extend to the greater sciatic notch and to have a proximal attachment to cover the posterior superior acetabulum extending to the anterior inferior iliac spine [15].

Fig. 7 Cadaveric dissection of the musculature of the gluteal region highlighting the tendinous insertions of the piriformis, superior gemellus, obturator internus, inferior gemellus, and quadratus femoris muscles onto the greater trochanter of the femur (Photo reproduced with permission from Daniel Wasserman)

It is agreed upon that the gluteus medius and minimus insert onto the greater trochanter of the femur, with the medius being most often reported as inserting onto the lateral aspect of the greater trochanter and the minimus onto its anterior surface [15]. It has been suggested that the gluteus minimus has an insertion into the hip joint capsule [16]. The final muscle of the superficial gluteal group, the tensor fascia lata, is commonly described as originating from the anterior lateral portion of the iliac crest and the lateral aspect of the anterior superior iliac spine. It is distally described as attaching into the fascia lata. There are differing descriptions as to how this distal attachment occurs, specifically if the tendon of the tensor muscle inserts immediately into the iliotibial band just beneath its muscular belly or if these fibers themselves continue inferiorly to insert into the Gerdy's tubercle at the lateral aspect of the tibia and the patellar retinaculum [15]. Innervation to the gluteus medius and minimus and tensor fascia lata is via the superior gluteal nerve, arising from ventral rami of the fourth and fifth lumbar and first sacral spinal nerves. The superior gluteal nerve emerges from the pelvis via the greater sciatic foramen in a position superior to the piriformis muscle. Together, the gluteus medius and minimus and the tensor fascia lata serve a primary function as abductors of the thigh. Of most clinical relevance is their function in closed chain during gait to stabilize the pelvis and prevent pelvic drop away from the stance limb as the contralateral limb swings through for limb advancement. In a position of hip flexion, the gluteus medius and minimus also function as medial rotators of the thigh. While classically less emphasized than the other gluteal muscles, results of cadaveric dissection suggest a potentially important function of the gluteus minimus, given its attachment to the articular capsule. It is suggested that the gluteus minimus may be to stabilize the head of the femur in the acetabulum by tightening the capsule and applying pressure on the femoral head [16]. Recent detailed study of the gluteal muscles suggests that both the gluteus medius and minimus, but not the tensor fascia lata, can be subdivided into distinct compartments based on fascicular organization within each muscle as well as the patterns of innervations via distinct branches of the superior gluteal nerve to these compartments [17]. Clinical relevance to this compartmental organization has been suggested, as rehabilitation of these gluteal muscles often utilizes exercises which target specifically the anterior or posterior aspects of the muscles [17].

The deep group of gluteal muscles consists of the six short rotators: the piriformis, the superior and inferior gemellus muscles, the obturator externus and internus muscles, and the quadratus femoris muscle (Fig. 7). As a group the short rotators (with the exception of the obturator externus) receive innervation from the ventral rami of the fifth lumbar as well as the first and second sacral spinal nerves. The piriformis muscle is an important surgical landmark. Its origin is from the anterior surface of the second through fourth sacral vertebrae, from which the muscle travels laterally to exit the pelvis via the greater sciatic foramen [18]. The most consistently observed variant in the anatomy of the piriformis muscle is a split of the muscle belly into superior and inferior portions by the sciatic nerve [18]. More typically the sciatic nerve will course inferior to the piriformis muscle, and the muscle

Fig. 8 Cadaveric dissection of the anterior and medial thighs. The sartorius, rectus femoris, iliopsoas, pectineus, adductor longus, and gracilis muscles are visible in the superficial plane of the dissection field. The femoral nerve, artery, and vein are visible in the femoral triangle between the iliopsoas and pectineus muscles. The obturator nerve is seen coursing between the adductor longus and the deeper adductor brevis muscles. Though classified as a gluteal muscle, the tensor fascia lata is visible at the lateral most aspect of the thigh, inserting into the iliotibial band (Photo reproduced with permission from Daniel Wasserman)

will possess a single belly. The distal attachment site of the piriformis has been most often been observed to be superior or both anterior and superior to the trochanteric fossa [19, 20]. The obturator internus muscle originates from the rami surrounding the obturator foramen and the quadrilateral plate and exits the bony pelvis via the lesser sciatic foramen [18]. The superior gemellus muscle is consistently described as originating from the ischial spine, while the origin of the inferior gemellus muscle is from the lateral surface of the ischial tuberosity. These two muscles are described as inserting into the tendon of the obturator internus muscle prior to inserting into the greater trochanter [20, 21]. The tendons of the piriformis and the obturator internus have also been reported to merge into a conjoined tendon prior to their insertion onto the superior and medial surfaces of the greater trochanter of the femur [18], though this is not uniformly reported in anatomic descriptions. Based on the finding of cadaveric dissection, this conjoined tendon, when present, has also been observed to attach to the hip joint capsule, the posterior margin of the gluteus medius, and the tendon of the obturator externus [18]. The obturator externus has a proximal attachment to the obturator foramen and courses laterally, inferior to the femoral neck, to insert into the trochanteric fossa [18, 20]. The tendon of the obturator externus has been observed to connect to the hip joint capsule and the conjoined tendon of the piriformis and obturator internus, though this is not uniformly reported in the literature [18]. The obturator externus muscle is technically considered a muscle of the medial thigh and receives innervation from the posterior division of the obturator nerve but is considered with the short rotators of the gluteal region given its shared function with these muscles. The quadratus femoris muscle has a proximal attachment to the lateral border of the ischial tuberosity and courses posterior to the head of the femur to insert onto the quadrate tubercle of the intertrochanteric crest as well as the quadrate line. It is not entirely surprising that descriptions of conjoined tendons for the short rotator muscles have been observed given the proximity of their classically reported distal attachment sites on the femur and their shared function of lateral rotation at the hip joint. The piriformis is perhaps the most consistently studied of the six short rotator muscles. It is suggested that this muscle potentially plays a role not only in lateral rotation of the hip but also in restricting posterior translation of the femoral head when the joint is flexed due to the shift towards a more posterior position of this muscle with respect to the hip joint in hip flexion [19].

Proximally, the anterior compartment of the thigh is comprised of the sartorius, rectus femoris portion of the quadriceps, iliopsoas, and pectineus muscles (though the pectineus is sometimes classified as a muscle of the medial thigh) (Fig. 8). Together these muscles contribute to flexion of thigh at the hip joint to varying degrees and in general receive innervation from ventral rami of second through fourth lumbar spinal nerves primarily via branches from the femoral nerve. Two exceptions to this innervation pattern include the psoas major portion of the iliopsoas which receives its innervation from the ventral rami of first and second lumbar spinal nerves where these nerves branch off the lumbar plexus and the pectineus which may receive innervation from either the femoral or accessory obturator nerves [22].

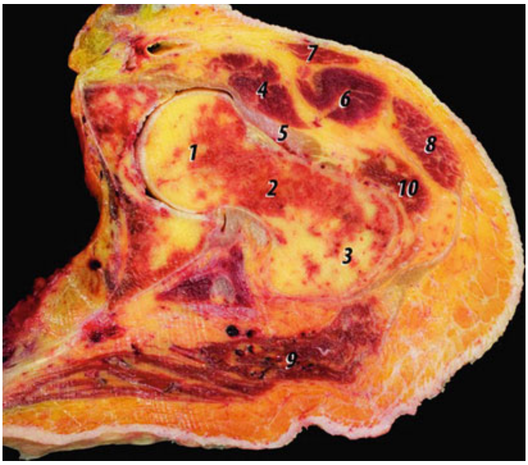


Fig. 9 Transverse cross section of a right hip at level of femoral neck showing the close relationship between the iliopsoas muscle and the anterior capsule of the hip. (*1*) Femoral head. (*2*) Femoral neck. (*3*) Greater trochanter. (*4*) Iliopsoas muscle. (*5*) Anterior capsule of the hip. (*6*) Rectus femoris muscle. (*7*) Sartorius muscle. (*8*) Tensor fasciae latae muscle. (*9*) Gluteus maximus muscle. (*10*) Gluteus medius muscle (http://www.springerimages.com/Images/MedicineAndPublicHealth/1-10.1007_978-1-4419-7925-4_7-20)

Of the muscles contributing to hip flexion, the iliopsoas is the most power flexor of the thigh at the hip joint. It originates as two muscles inside the abdomen and pelvis, the psoas major and the iliacus. The psoas major arises from the spinous processes of the 12th thoracic through the 5th lumbar vertebrae [23]. The iliacus muscle originates from the anterior iliac crest and the upper two-thirds of the iliac fossa [23]. The muscle bellies of the psoas and iliacus unite to form a shared tendon at the level of the inguinal ligament and pass deep to this structure [23]. The tendon of the iliopsoas has been observed to lie directly anterior to the anterior-superior aspects of the capsulolabral complex, and it is reported that the circumference of the tendon is greatest at this point [24] (Fig. 9). The sartorius muscle arises for the anterior superior iliac spine and courses obliquely inferior across the thigh to insert into proximal aspect of the medial tibia at the pes anserinus. It is a weak contributor to flexion at the hip given its length and span across multiple joints. The rectus femoris portion of the quadriceps muscle has a dual origin by a straight tendon arising from the anterior inferior iliac spine and a reflected tendon which arises from the groove above the rim of the acetabulum. This muscle shares a common tendinous insertion with the remaining three muscles of the quadriceps at the base of the patella and then indirectly via the shared patellar tendon (ligament) into the tibial tuberosity. The rectus femoris participates in hip flexion, but its power as a hip flexor is affected by the position of the knee; specifically, its action as a hip flexor is weakened due to active insufficiency, when the knee is extended. The pectineus originates from the pectineal line of the pubis and inserts distally into the pectineal line of the femur extending from the lesser trochanter to the linea aspera. The pectineus lies just medial to the iliopsoas muscles, and the two muscles border the femoral vessels within the femoral triangle. Together with the iliopsoas, the pectineus participates in flexion of the thigh as its primary action at the hip joint.

Collectively the muscles of the medial compartment of the thigh are described as adductors of the hip joint with accessory motions as flexors and medial rotators of the thigh at the hip. This group of muscles functionally consists of the adductor brevis, adductor longus, adductor magnus, and gracilis muscles. These muscles receive their innervation from the ventral rami of the second, third, and fourth lumbar spinal nerves via the anterior or posterior divisions of the obturator nerve. The origin of the adductor group is on the pubic bone with the adductor magnus having an additional proximal attachment on the ischial tuberosity. The medial compartment muscles have distal attachments onto the shaft of the femur with the exceptions of the gracilis which inserts onto the medial tibia at the pes anserinus and the adductor magnus which has an additional distal attachment to the adductor tubercle of the femur. The portion of the adductor magnus which originates from the ischial tuberosity is considered the "hamstring part" of this muscle as it receives its innervation via the tibial branch of the sciatic nerve and participates in some extension of the thigh at the hip with the hamstring group.

The hamstring muscles of the posterior thigh, semimembranosus, semitendinosus, and biceps femoris, are innervated by the ventral rami of the fifth lumbar as well as the first and second sacral spinal nerves via the tibial division of the sciatic nerve. The hamstrings share a common proximal attachment site to the ischial tuberosity. These muscles serve to extend the thigh at the hip, but since they are multi-joint muscles, their action at the hip is affected, via active insufficiency, by the position of the knee.

Blood Supply and Innervation

Hilton's law of innervation states, "The same trunks of nerves whose branches supply the groups of muscles moving a joint, furnish also a distribution of nerves to the skin over the insertion of the same muscles, and the interior of the joint receives its nerves from the same source." It is generally agreed upon that innervations to the hip joint therefore are provided by the nerves which cross this joint and supply the hip joint musculature. The obturator nerve, in particular, is most consistently described as the nerve supplying innervation to the hip joint and is a target of nerve blocks for control of hip joint pain [12, 25]. However, cadaveric studies have revealed that innervation to the hip joint extends beyond contributions from the obturator nerve, and innervation to the joint capsule can be broken down into regional designations. The anterior region of the hip joint capsule receives innervation from the femoral and obturator nerves with the femoral nerve contributing specifically to the anterior and anterolateral capsular regions [26]. Innervation from the obturator nerve supplies the anteromedial aspects of the hip joint. Both the anterior and posterior divisions of the obturator nerve have been observed to contribute branches to this region of the capsule [26]. The posterior elements of the joint capsule receive innervation from the superior gluteal nerve, the nerve to the quadrates femoris, and direct branches from the sciatic nerve [26]. Beyond innervations to the capsule itself, both the acetabular labrum [12] and ligamentum teres receive innervations from articular nerve fibers. Nociceptive and proprioceptive fibers have been identified in the acetabular labrum, with a high concentration of nociceptive and proprioceptive

Fig. 10 Magnetic resonance angiography (*MRA*)-reconstructed composite image, produced by fusing contrast (Gadovist)-enhanced MRA series with coronal high-resolution proton density (*PD*) view of the hip, to illustrate the presence and position of blood vessels visualized on MRA in relation to the anatomy of the hip. Normal hip. The medical circumflex femoral artery (*MCFA*) is well demonstrated. (Note: CE-MRA series acquired in coronal plane after 25-s delay after bolus 7.5 mg Gadovist (Bayer Schering Pharma AG, Germany).) In normal MRA studies, the vessels are demonstrated by following them sequentially through the coronal plane images. For illustrative purposes only, the single-plane coronal PD image has been fused with a composite of the coronal angiography images to provide a representation of the vascular anatomy, as seen on MRA, relative to the hip joint (http://www.springerimages.com/Images/MedicineAndPublicHealth/1-10.1007_978-1-84800-088-9_9-3)

fibers being found close the attachment site of the labrum on the acetabulum [27]. The ligamentum teres also receives nociceptive and proprioceptive innervations with the highest concentrations being found centrally within this structure [27]. It is suggested that there may be a decline in both nociceptive and proprioceptive innervations to the capsule with aging.

Based on the results of cadaveric dissection studies, it is generally agreed upon that the primary source of blood supply to the hip joint is medial femoral circumflex artery (MFCA), which typically arises as a branch of the deep femoral artery in the femoral triangle of the anterior thigh [28, 29] (Fig. 10). Results of anatomic dissection reveal several consistent branches of this vessel.

A superficial branch supplies the adductor musculature rather than the femoral head. As the main trunk of the MFCA courses towards the lesser trochanter, it has been observed to consistently divide into a descending branch and a deep branch, with the deep branch being described as the continuation of the main trunk and the main vessel supplying the femoral head [28, 29], while the descending branch courses to supply musculature, specifically of the posterior thigh. The deep branch has been observed to course posterior to the obturator externus and anterior to the quadratus femoris and then cross the tendons of the two gemellus muscles and the obturator internus prior to entering the hip capsule [28]. The relationship of the deep branch of the MFCA to hip musculature and the femoral neck is significant as posterior dislocation, particularly with fracture of the hip, can compromise this vessel resulting in avascular necrosis of the femoral head.

Other contributions to blood supply of the hip joint have been described. A small vessel has been observed to arise at the bifurcation of the MCFA which courses to the posterior inferior quadrant of the femoral head and neck [29]. Additionally, it has been suggested that an anastomosis exists between the deep branch of the femoral artery and the inferior gluteal artery which may be an additional source of vascular support. Due to the course of this vessel being along the inferior margin of the piriformis tendon, it has been described as the piriformis branch of the inferior gluteal artery [28, 30]. The acetabular labrum is vascularized by branches of the superior and inferior gluteal arteries as well as the obturator artery [12]. Blood supply to the labrum arises from small vessels on its capsular side, and these vessels do not penetrate deeply into the substance of the labrum itself. This leaves the majority of the labrum avascular and contributes to limitation in healing with labral tears [12].

Summary

In life the hip tends to operate in a closed kinematic chain, with the distal end of the chain, the foot, fixed in contact with the ground during weight bearing and the proximal end of the chain, the head, tending to remain upright and vertically oriented over the rest of the body. Proper functioning of the hip joint is essential for locomotion. The anatomic components of the hip joint as outlined in this chapter contribute to its function by balancing stability and mobility through its associated capsular, ligamentous, and muscular structures. Adequate blood supply and innervation also ensure proper functioning of this joint. Knowledge of these structures and the anatomic relationships that exist among them is essential for treating hip joint pathology.

References

1. Sadler TW. Limbs Medical embryology. Baltimore: Lippincott Williams & Wilkins; 2012. p. 151–61.
2. Roach KE, Miles TP. Normal hip and knee active range of motion: the relationship to age. Phys Ther. 1991;71:656–65.
3. Wheeless CR. Wheeless' textbook of orthopaedics. Nunley JA, Urbaniak JR. http://www.wheelessonline.com/ (2013). Data Trace Internet Publishing, LLC. 10-4-2013.
4. Heller MO, Bergmann G, Deuretzbacher G, Claes L, Haas NP, Duda GN. Influence of femoral anteversion on proximal femoral loading: measurement and simulation in four patients. Clin Biomech. 2001;16:644–9.
5. Lenaerts G, De Groote F, Demeulenaere B, et al. Subject-specific hip geometry affects predicted hip joint contact forces during gait. J Biomech. 2008;41:1243–52.
6. Buller LT, Rosneck J, Monaco FM, Butler R, Smith T, Barsoum WK. Relationship between proximal femoral and acetabular alignment in normal hip joints using 3-dimensional computed tomography. Am J Sports Med. 2012;40:367–75.
7. Wagner FV, Negrao JR, Campos J, et al. Capsular ligaments of the hip: anatomic, histologic, and positional study in cadaveric specimens with MR arthrography. Radiology. 2012;263:189–98.
8. Ito H, Song Y, Lindsey DP, Safran MR, Giori NJ. The proximal hip joint capsule and the zona orbicularis contribute to hip joint stability in distraction. J Orthop Res. 2009;27:989–95.
9. Elkins JM, Stroud NJ, Rudert MJ, et al. The capsule's contribution to total hip construct stability – a finite element analysis. J Orthop Res. 2011;29:1642–8.
10. Bardakos NV, Villar RN. The ligamentum teres of the adult hip. J Bone Joint Surg Br. 2009;91:8–15.
11. Cerezal L, Kassarjian A, Canga A, et al. Anatomy, biomechanics, imaging, and management of ligamentum teres injuries. Radiographics. 2010;30:1637–51.

12. Blankenbaker DG, Tuite MJ. Acetabular labrum. Magn Reson Imaging Clin N Am. 2013;21:21–33.
13. Grant AD, Sala DA, Davidovitch RI. The labrum: structure, function, and injury with femoro-acetabular impingement. J Child Orthop. 2012;6:357–72.
14. Barker PJ, Hapuarachchi KS, Ross JA, Sambaiew E, Ranger TA, Briggs CA. Anatomy and biomechanics of gluteus maximus and the thoracolumbar fascia at the sacroiliac joint. Clin Anat. 2014;27(2):234–40. PMID:23959791.
15. Flack NA, Nicholson HD, Woodley SJ. A review of the anatomy of the hip abductor muscles, gluteus medius, gluteus minimus, and tensor fascia lata. Clin Anat. 2012;25:697–708.
16. Beck M, Sledge JB, Gautier E, Dora CF, Ganz R. The anatomy and function of the gluteus minimus muscle. J Bone Joint Surg Br. 2000;82:358–63.
17. Flack NA, Nicholson HD, Woodley SJ. The anatomy of the hip abductor muscles. Clin Anat. 2014;27 (2):241–53. PMID:23625344.
18. Solomon LB, Lee YC, Callary SA, Beck M, Howie DW. Anatomy of piriformis, obturator internus and obturator externus: implications for the posterior surgical approach to the hip. J Bone Joint Surg Br. 2010;92:1317–24.
19. Roche JJ, Jones CD, Khan RJ, Yates PJ. The surgical anatomy of the piriformis tendon, with particular reference to total hip replacement: a cadaver study. Bone Joint J. 2013;95-B:764–9.
20. Ito Y, Matsushita I, Watanabe H, Kimura T. Anatomic mapping of short external rotators shows the limit of their preservation during total hip arthroplasty. Clin Orthop Relat Res. 2012;470:1690–5.
21. Aung HH, Sakamoto H, Akita K, Sato T. Anatomical study of the obturator internus, gemelli and quadratus femoris muscles with special reference to their innervation. Anat Rec. 2001;263:41–52.
22. Woodburne RT. The accessory obturator nerve and the innervation of the pectineus muscle. Anat Rec. 1960;136:367–9.
23. Blomberg JR, Zellner BS, Keene JS. Cross-sectional analysis of iliopsoas muscle-tendon units at the sites of arthroscopic tenotomies: an anatomic study. Am J Sports Med. 2011;39(Suppl):58S–63.
24. Alpert JM, Kozanek M, Li G, Kelly BT, Asnis PD. Cross-sectional analysis of the iliopsoas tendon and its relationship to the acetabular labrum: an anatomic study. Am J Sports Med. 2009;37:1594–8.
25. Locher S, Burmeister H, Bohlen T, et al. Obturator nerve block: a technique based on anatomical findings and MRI analysis. Pain Med. 2008;9:1012–5.
26. Birnbaum K, Prescher A, Hessler S, Heller KD. The sensory innervation of the hip joint–an anatomical study. Surg Radiol Anat. 1997;19:371–5.
27. Haversath M, Hanke J, Landgraeber S, et al. The distribution of nociceptive innervation in the painful hip: a histological investigation. Bone Joint J. 2013;95-B:770–6.
28. Gautier E, Ganz K, Krugel N, Gill T, Ganz R. Anatomy of the medial femoral circumflex artery and its surgical implications. J Bone Joint Surg Br. 2000;82:679–83.
29. Zlotorowicz M, Szczodry M, Czubak J, Ciszek B. Anatomy of the medial femoral circumflex artery with respect to the vascularity of the femoral head. J Bone Joint Surg Br. 2011;93:1471–4.
30. Grose AW, Gardner MJ, Sussmann PS, Helfet DL, Lorich DG. The surgical anatomy of the blood supply to the femoral head: description of the anastomosis between the medial femoral circumflex and inferior gluteal arteries at the hip. J Bone Joint Surg Br. 2008;90:1298–303.

Clinical Biomechanics of the Hip Joint 2

Philip J. Malloy and Shane J. Nho

Contents

P.J. Malloy (✉)
Program in Exercise Science, Department of Physical Therapy Graduate Research Assistant,
Marquette University, Milwaukee, WI, USA
e-mail: philip.malloy@marquette.edu

S.J. Nho
Department of Orthopaedic Surgery, Rush University Medical Center, Chicago, IL, USA

Midwest Orthopaedics at Rush, Hip Preservation Center, Rush University Medical Center, Chicago, IL, USA
e-mail: shane.nho@rushortho.com

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_2

Abstract

Biomechanics is a broad field that commonly analyzes the movement and forces generated and transmitted by and through the musculoskeletal system. Simple observation of movement is the most basic biomechanical analysis; however, laboratory methods often involve the use of complex measurement tools to quantify motion and forces on the body. Pathology of the hip joint oftentimes results in alterations of the biomechanics at the hip or other links in the kinetic chain. The understanding of the biomechanics of the hip joint is important from a clinical perspective as this information can assist in guiding clinical decision making for certain hip pathologies. Although an extensive body of literature exists on the biomechanics of the hip in individuals with osteoarthritis (OA), less is known about conditions such as femoroacetabular impingement (FAI) or acetabular labral tears, which have also been shown to influence the movement and forces at the hip joint. Understanding the normal osteokinematics, arthrokinematics, and muscle actions at the hip joint provides clinicians with the basic biomechanical background to detect impairments that may impact function and contribute to injury. Human gait serves as an excellent model in which to discuss the biomechanics of the lower extremity and has been extensively studied in patients with hip pathology.

Introduction

Biomechanics is part of the broader fields of exercise science and kinesiology. It has been defined as "the study of structure and function of biological systems by the means and methods of mechanics" [1]. The body systems that are often the focus of biomechanics research are the musculoskeletal and neurologic systems; therefore, the findings can have important implications for the field of orthopedics. Biomechanical analysis often involves the measurement of kinematics and kinetics. Kinematics is the description of motion without consideration of the forces causing the motion. Position, velocity, and acceleration are all examples of kinematic variables that are often studied in biomechanical research [2, 3]. Kinetics analyzes the relationship between the forces and motion of the system. Some of the common kinetic measurements are linear force, angular force (moments), impulse, power, work, and energy [2, 3]. The muscular contribution to kinematics and kinetics is of interest in biomechanical research. Electrical activity associated with muscle activation and the estimation of force-generating capacity are all commonly measured biomechanical variables. The information gained from orthopedic biomechanical analysis can contribute to the understanding of injury mechanisms, the physiologic impact of rehabilitation intervention, tissue behaviors and loading characteristics, and athletic performance.

Biomechanical Instrumentation and Measurement

The most basic level of biomechanical analysis involves general observations of movement during activity. This type of analysis is commonly performed in the orthopedic and sports medicine clinical setting. A high-speed digital camera can be used to record movement tasks such as running or jumping in order for two-dimensional analysis to be performed. Two-dimensional analysis movement analysis is an effective tool for analyzing movement patterns which may be related to injury risk factors [4]. Devices such as electrogoniometers, accelerometers, and motion capture systems are also commonly used in the research setting for the analysis of movement. Some of these measures such as electrogoniometers and accelerometers can be used in a clinical setting although they are often costly and require additional software for data analysis. Motion capture, such as near-infrared camera systems (Fig. 1), is most commonly used in the laboratory setting. However, these systems are very expensive and require a significant amount of data storage, as well as require an understanding of biomechanical research methods to be used appropriately [2, 3].

Fig. 1 Near-infrared motion capture camera (Vicon Motion Systems Ltd., Oxford, UK)

Fig. 2 Handheld dynamometer (Lafayette Instrument Company, Lafayette, IN)

Kinetic analysis at a basic level can involve drawing a free body diagram to estimate two-dimensional forces during activity. These diagrams require information of the body's anthropometrics in order to accurately estimate the forces acting on the segment. However, the calculations of these forces are limited to a static state of equilibrium and cannot be used for dynamic force estimates. A force transducer can be used to directly quantify a force and include devices such as a handheld dynamometer, which is commonly used in the clinical setting to measure muscle strength (Fig. 2). In the laboratory setting, a force plate is often used to measure three-dimensional forces during activity. The force plate data can be used for three-dimensional biomechanical analysis using an inverse dynamics approach by integrating the kinematic and kinetic data to calculate joint moments, powers, and energy in all three planes of motion.

Electromyography (EMG) and pressure sensors are also commonly used instruments to obtain information on muscle activation and pressure distribution patterns. EMG is a technique for recording and measuring electrical activity in the muscle, and the signal is a sum of all the recorded muscle fiber action potentials in the area of the recording electrode. Surface EMG involves the use of electrodes (Fig. 3) that are placed on the skin overlying the muscle of interest. Fine wire or intramuscular EMG involves the insertion of a small needle electrode or wires directly into the muscle fibers. Surface EMG measures a large sample of motor unit action potentials from the

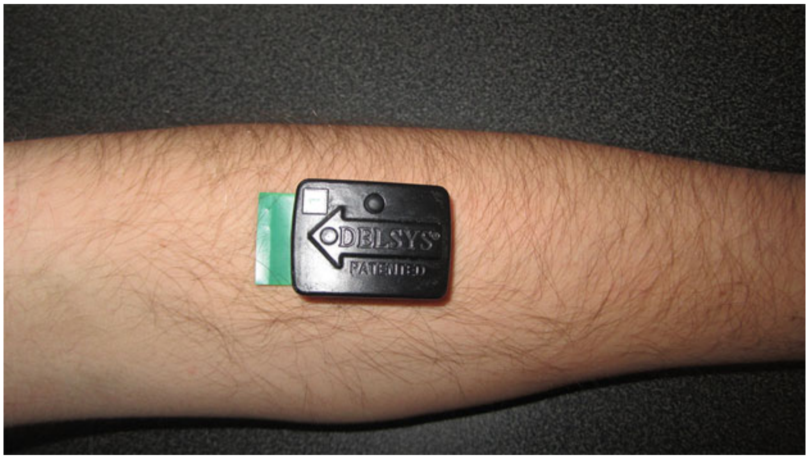


Fig. 3 Wireless EMG sensor (Delsys Inc., Natick, MA)

area under the electrodes, whereas fine wire EMG only records from a small number of motor units that are in close proximity to the electrode. There are many factors that can influence the signal recorded by EMG such as skin impedance, muscle fiber depth, adipose tissue, movement artifact, and muscle length; therefore, caution should be taken when interpreting the results. It also should be acknowledged that EMG signal is only directly proportional to muscle force output during an isometric contraction when muscle length does not change. Dynamic activities such as jumping or running cause a change in the number of motor units that are positioned under the electrode; therefore, a consistent measure from the same motor units is not being recorded throughout the movement which can influence the signal output. EMG data can also be used with advanced muscle measurement techniques such as musculoskeletal modeling which can provide a more accurate estimate of muscle forces during activity, although the use of such techniques requires a greater computational background and an understanding of programming to create a valid model.

Biomechanical Analysis of the Hip Joint

Early biomechanical investigations of the hip joint performed by Rydell described the complex structural anatomy of the femur as it pertains to accommodating load demand during weight bearing [5]. Crowninshield [6] performed a kinematic and kinetic analysis of the hip during the activities of walking, stair negotiation, and moving from sitting to standing. These seminal investigations of hip biomechanics provide the foundation for further investigation of hip biomechanics during functional activity in both the healthy and pathologic populations. Kinematic investigation in patients with hip osteoarthritis revealed an overall loss of hip range of motion during gait. Additionally patients with the greatest amount of motion loss and pain exhibited a kinematic alteration characterized by a "reversal" of sagittal plane motion as the hip was moved into extension [7]. Similarly this sagittal plane kinematic "reversal" was observed in patients with symptomatic femoroacetabular impingement (FAI) during the stance phase of gait [8]. Kennedy et al. [9] found that patients with symptomatic FAI demonstrate less hip abduction and pelvic frontal plane motion compared to controls during the swing phase of walking. Patients with FAI also demonstrate a reduced squat depth compared to controls during a maximal squat task [10]. These findings support the use of a maximal squat test during the evaluation of patients with symptomatic FAI [11].

Hip pathology has also been shown to cause kinetic alterations at the hip joint. Often structural abnormality associated with developmental deformity or degenerative osteoarthritis can have profound effect on force production and transmission at the hip joint. These structural abnormalities can lead to alterations of the hip joint axis of rotation which has been shown to have profound impact on the hip muscles moment arm length, muscle force production, and muscle moments produced at the hip joint [12]. An instrumented femoral prosthesis following a total hip replacement has been used to quantify in vivo contact forces during daily activities such as walking and stair climbing [13]. The hip joint contact forces during level walking were found to be approximately 1.5 times body weight and 2.5 times body weight for stair climbing [13]. Hip joint forces during common open chain hip exercises were also quantified using musculoskeletal modeling based on kinematic, kinetic, and EMG data from healthy individuals. This model simulated conditions of weakness in particular muscle groups and showed that a significant increase in force in the anterior hip occurs in the presence of hip extensor weakness [14]. These findings have implications for rehabilitation specialists when prescribing exercises for patients with anterior hip injury such as an acetabular labral tear. FAI has also been shown to alter kinetics at the hip joint during walking with a reduction of external moments for hip flexion and external rotation in symptomatic patients [15].

Muscle activation patterns may influence the kinetics at the hip joint, and changes in these patterns have been observed in patients with hip pathology such as osteoarthritis and FAI [16, 17]. A recent systematic review of hip muscle weakness in patients with OA revealed consistent evidence of reductions in muscle force output and fiber atrophy [16]. Similar findings were observed in patients with FAI where isometric muscle torque was reduced for hip adduction, flexion, external rotation, and abduction compared to healthy controls [17]. In addition to pathology, externally imparted forces change the muscle activation patterns at the hip. Neumann et al. [18, 19] found that changes in carrying load position result in changes in muscle activity of the gluteus medius muscle. These findings have practical significance for patient education and treatment interventions. Carrying a load at the side results in a greater amount of EMG activity of the ipsilateral gluteus medius muscle during walking [18]. Conversely, individuals who used an assistive device on the contralateral side demonstrated a reduction in gluteus medius activity compared to walking without an assistive device [20]. The basic biomechanical findings provide the rationale for physicians and rehabilitation specialists in instructing patients with hip pathology to use an assistive device such as a cane on the side contralateral the injury [18].

Hip pathomorphology is an abnormality in the three-dimensional geometry of the hip, whereas hip pathomechanics involve the understanding of how motion conflicts produce chondrolabral injury. Ganz and colleagues published the landmark paper describing the concept of femoroacetabular impingement (FAI) as a possible cause of end-stage hip osteoarthritis [21]. FAI consists of morphological pathology involving abutment of the femoral neck and the acetabular rim at the extremes of range of motion. Deformities can be primarily related to the acetabulum (pincer), femoral neck (CAM), or most commonly a combination of the two. FAI and other pathomorphologic hip deformities (i.e., dysplasia) can lead to labral injury, osseous change, and eventually osteoarthritis. Surgical correction of the deformities on the acetabulum, proximal femur, or both was originally described using an open surgical hip dislocation or osteotomies. More recently, hip arthroscopy has been widely utilized as a technique to correct the pathomorphology associated with FAI (Fig. 4).

There has been recent interest in the characterization of hip pathomorphology using three-dimensional imaging to assist identification of deformities and guide surgical treatment. Computer tomography (CT) scans allow for the precise evaluation of the three-dimensional morphology of the hip joint. Kang and colleagues have described a novel, automated method to describe aberrancies in the topography of the proximal femur and acetabulum [22]. The quantification of CAM and pincer deformities is critical for a more precise understanding of hip motion conflicts (Figs. 5 and 6). In addition, three-dimensional modeling is another technique to create a virtual hip model to identify areas of impingement with hip movement. More advanced three-dimensional imaging and dynamic imaging may provide surgeons information about

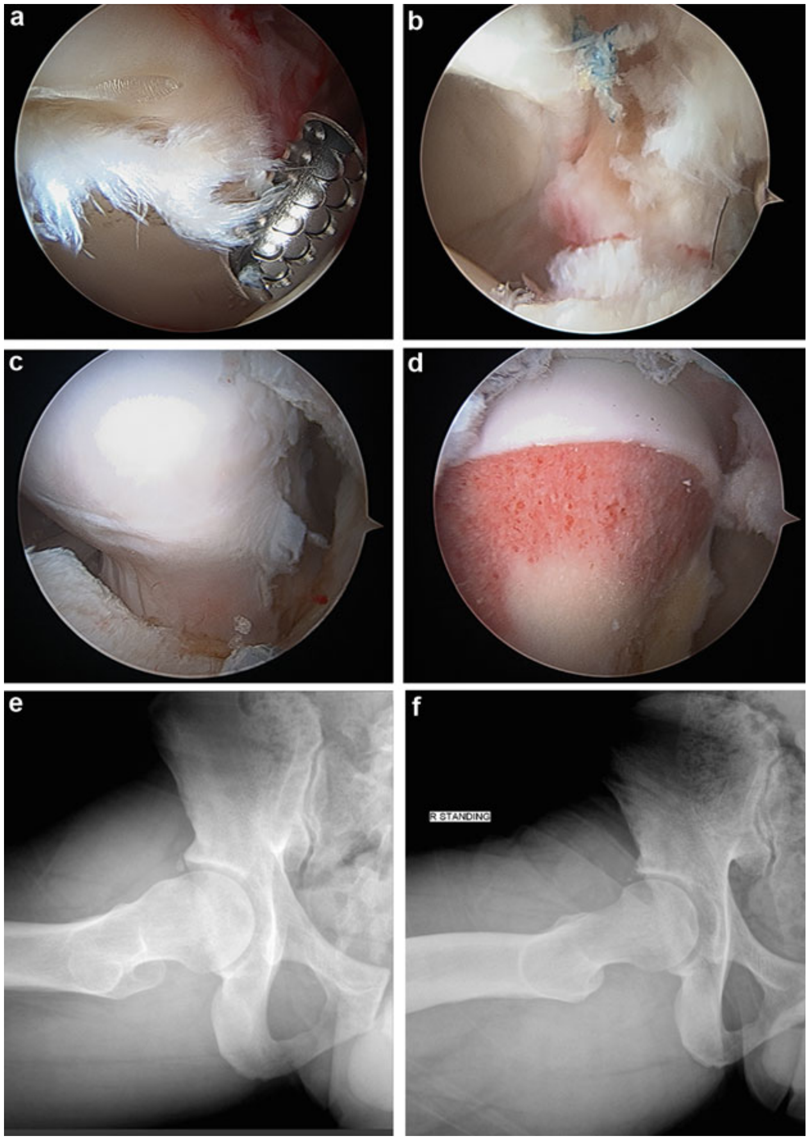


Fig. 4 Hip arthroscopy to treat chondrolabral injury and FAI. (**a**) Injury at the chondrolabral junction seen with CAM deformity. (**b**) Arthroscopic appearance after labral refixation and chondral debridement. (**c**) CAM deformity at the proximal femoral head-neck junction. (**d**) View of the proximal femoral head-neck junction after osteochondroplasty. (**e**) Plain radiograph using the Dunn lateral view of the CAM deformity prior to surgery. (**f**) Plain radiograph using the Dunn lateral view of the proximal femur after osteochondroplasty

the exact amount of osseous resection of the acetabulum and proximal femur to normalize the anatomy and eliminate motion conflict.

Controlled laboratory biomechanical investigation has established our foundational understanding of the biomechanics of the hip in the healthy and pathologic populations; however, the methods applied in these studies are often beyond the capability of the clinical setting. Therefore, it is essential that orthopedic clinicians have a manner in which to evaluate the biomechanics of a patient in order to assist in the clinical decision making process. Visual observation of movement patterns during common activities can provide practical information on the biomechanical implications of hip pathology. Evaluation of hip osteokinematics, arthrokinematics, and muscle activation can provide information about the movement and alteration in loading patterns that are associated with a certain injury. Additionally, this information provides a baseline comparison to assess improvement following a treatment intervention. Gait analysis is commonly performed in orthopedic practice as this activity is one of the most basic activities of daily living. Understanding the biomechanics of the gait cycle can assist clinicians in diagnosis and treatment of hip injury through the observation of deviations that are often associated with a particular pathology. The following sections of this chapter will discuss the clinical biomechanical analysis of the hip joint through the discussion of normal hip osteokinematics, arthrokinematics, and muscle actions in each of the planes of motion. Additionally, biomechanical analysis of the hip joint during normal gait will be discussed, and gait and deviations associated with hip pathology will be presented.

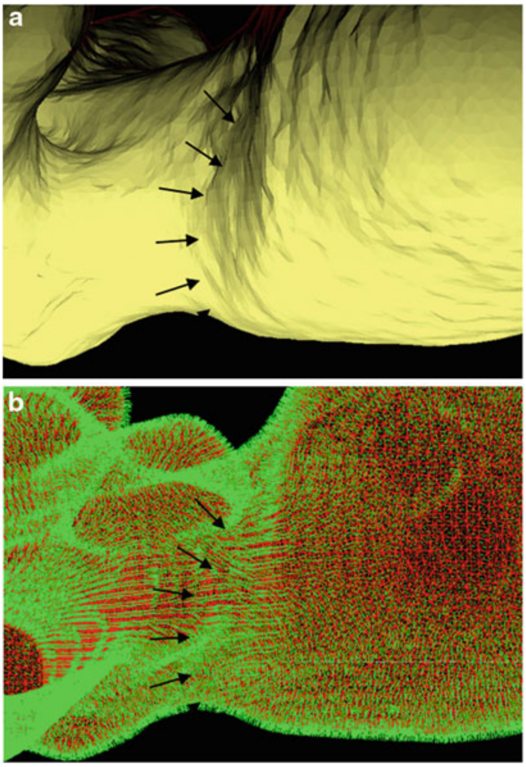


Fig. 5 Quantification of CAM deformity. (**a**) CAM lesion in the femoral neck appeared on the 3D polygon model (*arrows*). (**b**) Normal vectors of the polygons (*green lines*). The CAM lesion is shown by arrows

Osteokinematics, Arthrokinematics, and Muscle Actions of the Hip Joint

The ball and socket structure of the hip joint allows for six degrees of freedom around three axes of motion at the hip joint center. Although the hip is a relatively mobile joint, it is also incredibly stable because of its osseous architecture, joint articulation, and extensive surrounding soft tissue structures. Open chain activity of the hip joint is characterized by femoral-on-pelvic motion, whereas closed chain function often results in pelvic-on-femoral motion [23]. Femoral-on-pelvic movement will be referred to as open chain and pelvic-on-femoral movement as closed chain for the remainder of this discussion.

The movements and axis of rotation of the hip joint in each motion plane are listed in Table 1. Osteokinematics describe the motion of a body segment and in the case of the hip joint refers to motion of the femur or pelvis. Arthrokinematics refers to the movement that occurs between the two joint surfaces and is the motion occurring between the femoral head and acetabulum in the case of the hip joint. The arthrokinematics of the hip joint abide by the principles of the convex on concave and concave on convex rules [23].

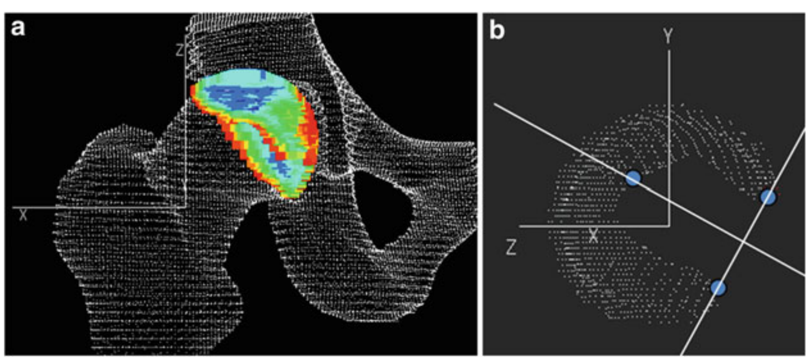


Fig. 6 In vivo zoning system based on the acetabulum. (**a**) Automatic segmentation of the acetabulum point clouds using the least distances in the hip joint.
(**b**) Determination of anatomical landmarks to define acetabulum reference lines

Table 1 Planes of motion, anatomical direction, axis of rotation, and muscle actions of the hip joint

Plane of motion	Movement	Axis of rotation	Muscles
Sagittal	Flexion and extension	Mediolateral	**Flexors** (iliopsoas, rectus femoris, sartorius, adductor longus)
			Extensors (gluteus maximus, adductor magnus; hamstrings)
Frontal	Abduction and adduction	Anterior-posterior	**Adductors** (adductor longus, brevis, and magnus, obturator externus, pectineus)
			Abductors (tensor fascia lata, gluteus medius and minimus, sartorius)
Transverse	Internal and external rotation	Longitudinal axis	**External rotators** (piriformis, gluteus maximus, gemelli superior and inferior, obturator internus, quadratus femoris)

These principles state that when a convex surface (i.e., femoral head) moves on a concave surface (i.e., acetabulum), the motion between the joint surfaces occurs in the opposite directions. Conversely, when the concave surface of the acetabulum moves on a convex surface of the femoral head, the motions at the joint will occur in the same direction (i.e., closed chain hip motion).

The hip consists of 25 muscles that cross the joint; therefore, the influence of these muscle actions on joint mechanics is profound [12]. Additionally, a number of the muscles span two joints and, therefore, influence multiple joints during activation. The muscle actions at the hip will be discussed in the context of the direction of force produced to create movement. Although these muscle actions involve angular and linear force production, the discussion on the quantification of these forces involves advanced biomechanical analysis and is beyond the scope of this chapter. The following sections will describe the osteokinematics, arthrokinematics, and muscle actions for the hip in each of the three planes of motion.

Sagittal Plane Osteokinematics

The normal osteokinematics of the hip joint in the sagittal plane for open chain flexion is approximately 120–125° with the knee in a flexed position but reduces to between 70° and 80° when the knee is in an extended position [23, 24]. During closed chain function, hip flexion corresponds to an anterior tilt of the pelvis which results in approximation of the anterior pelvis and the anterior aspect of the femoral neck and shaft. Motion in this plane has profound implication in hip pathology as hip flexion loss is associated with conditions such as osteoarthritis and femoroacetabular impingement [21, 25]. The normal value for open chain hip extension is between 10° and 15° [24]. A posterior pelvic tilt in the closed chain creates extension motion at the hip joint and impact gait in the presence of hip flexion contracture or intraarticular injury [7]. Closed chain motion at the hip joint has a direct consequence on the movement of the lumbar spine because of the strong attachment of the sacrum to the pelvis through the posterior pelvic ligaments and trunk extensor muscles [23]. The link that the hip joint provides between the trunk and lower extremities has profound implications for the diagnosis and treatment of low back and lower extremity kinetic chain injury.

Sagittal Plane Arthrokinematics

The bony structure and soft tissue support of the hip joint minimize translational motion of the femoral head on the acetabulum [23].The motion between the femur and acetabulum during the motions of hip flexion and extension occurs as a pure rotation around a medial lateral axis that passes through the center of the femoral head.

Open chain hip flexion causes a spin of the femoral head in the posterior, while an anterior pelvic tilt will cause an anteriorly directed rotation of both the femoral head and acetabulum. These motions occur in the opposite direction for open chain hip extension and a posterior pelvic tilt.

Sagittal Plane Muscle Actions

In order for a muscle to create motion at the hip in the sagittal plane, the line of pull from the muscle must lie either anterior or posterior to the joint axis of rotation [12]. Muscles that create action on the anterior side of the axis of rotation will result in the motion of flexion at the hip, while muscles that lie posterior to this axis cause hip extension motion. This information is important to clinicians because the understanding of the line of pull of a muscle in relation to the joints axis of rotation can provide a general idea of the direction of force at the joint during motion. This information can also be used to assist with muscle strengthening by changing joint positions to influence moment arm lengths and muscle force production [12].

The primary hip flexor muscles consist of the psoas major and iliacus (iliopsoas), tensor fascia lata (TFL), rectus femoris, sartorius, and adductor longus [23]. The iliopsoas muscle group can cause open chain hip flexion or closed chain anterior pelvic tilting. The iliopsoas attachment to the femur and pelvis and lumbar spine creates force transmission both across the hip joint and lumbar spine structures [26]. The tensor fascia lata muscle produces force through its attachment to the iliotibial band of the thigh (ITB). The ITB attaches distally to the proximal aspect of the tibia and to the linea aspera of the femur. The TFL creates tension through the fascial system of the thigh, thereby imparting a force on the soft tissues of the thigh and across the hip joint. The rectus femoris muscle crosses both the hip and knee joint and, therefore, transmits force across both joints [12]. The proximal attachment of the rectus femoris to the rim of the acetabulum may have clinical implications as the forces created by this muscle may contribute to pain and pathology of anterior structures such as the hip capsule and acetabulum labrum. Action of the sartorius muscle creates a combination of hip flexion, external rotation, and abduction based on its orientation passing distally and medially across the thigh and location of its distal attachment at the medial tibia.

The hip extensor muscles are comprised of the gluteus maximus, hamstrings, and adductor magnus. The hip extensors produce open chain posterior motion of the femur on pelvis as observed during the stance phase of gait. The hip extensors muscles work from a mechanical advantage with the femur in a position of flexion as these muscles moment arm is lengthened in this position [12]. The hip extensors also work to produce a posterior pelvic tilt with the axis of rotation being around a medial lateral axis through the femoral heads [23]. Forward leaning of the trunk also promotes activation of the hip extensor muscles as the vertical ground reaction force moves more anteriorly with respect to the hip joint facilitating activation of the hip extensors to maintain an upright position [23].

The adductor longus muscle functions in both the sagittal and frontal planes. In the sagittal plane, the adductor longus muscle works as a primary hip flexor as the moment arm of this muscle passes anterior to the axis of rotation of the hip when the joint is in a position of extension [23]. The moment arm changes to a posterior position in relation to the medial lateral axis of rotation when the hip is in a flexed position. Function such as walking, running, cycling, and squatting that involve a repetitive sagittal plane motion often involves force production from the adductor longus in this plane. The multiplanar demand placed on this muscle may be a reason that it is prone to overuse or overload injury during sports activity such as hockey [27].

Frontal Plane Osteokinematics

The frontal plane osteokinematics of the hip include the open chain motions of abduction and adduction. The closed chain motions at the hip involve pelvic elevation or depression on a fixed

Fig. 7 Closed chain hip adduction. This posture is also referred to as a Trendelenburg sign and is associated with weakness of the hip abductor muscles

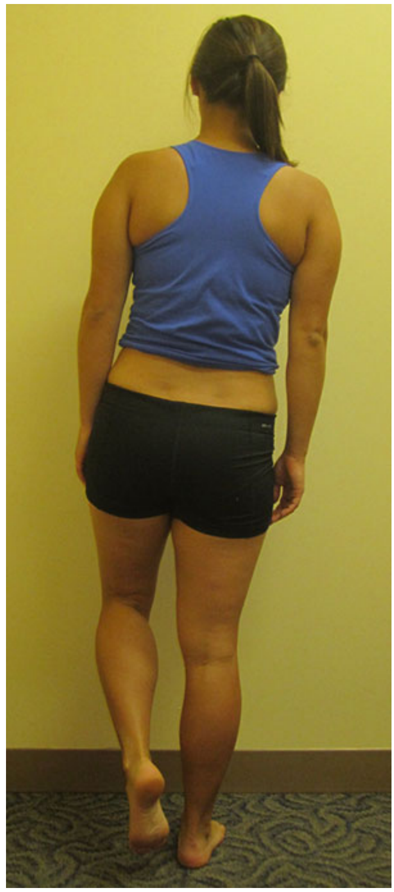


Fig. 8 Closed chain hip abduction. This posture is also referred to as a compensated Trendelenburg sign and is characterized by an elevation of the contralateral pelvis and lateral trunk lean over the stance side

femur which is characterized by the non-stance side pelvis being raised or lowered above the horizontal (Figs. 7 and 8). The normal open chain ranges of motion for hip abduction are 40° and 20° for adduction [23, 28]. Closed chain pelvic elevation on the non-stance side creates motion in the direction of abduction on the stance leg (Fig. 8). Closed chain hip adduction motion occurs as the non-stance side pelvis is lowered below the horizontal (Fig. 7). Alterations in closed chain frontal plane osteokinematics are commonly observed in the hip pathologies such as osteoarthritis (OA). The two most common osteokinematic compensations in the frontal plane are an excessive drop of the non-stance side pelvis (Trendelenburg sign) (Fig. 7) and excessive elevation of the non-stance pelvis with lateral trunk lean to the stance side (compensated Trendelenburg) (Fig. 8).

Frontal Plane Arthrokinematics

The frontal plane arthrokinematics of the hip joint follow a vertical path along the middle of the femoral head and acetabulum [23]. The motion at the articular surface during open and closed chain frontal plane motion is a combination of a translation and rotation of the surfaces. The translational motion of the hip joint is minimal in the frontal plane similar to that of sagittal plane arthrokinematic motion.

Frontal Plane Muscle Actions

The hip abductor and adductor muscles control the movement of the femur and pelvis in the frontal plane. The function of these muscles is extremely important for stabilization of the lower extremity and pelvis during gait. The hip adductors are one of the most commonly injured muscle groups during sports [27]. The hip abductors have been implicated in the development of overuse injuries of the lower extremity including patellofemoral pain and iliotibial band syndrome [29–31].

The hip adductor muscle group functions in all three planes of hip motion. The primary role of this muscle group in the frontal plane is to produce adduction motion in both the open and closed chains. Eccentric activation of the adductors in the closed chain assists with frontal plane control of the pelvis during single leg functional activity such as planting to kick a ball or changing directions while running. Open chain activation causes hip adduction as well as provides contribution to hip flexion, extension, and internal rotation. The multiplane function of this muscle group with a combination of simultaneous open and close chain function during weight-bearing activities may contribute to these muscles being commonly injured.

The hip abductor muscle group is comprised of the gluteus medius, gluteus minimus, and tensor fascia lata muscles. Each of the muscles produces open chain and closed chain motions of hip abduction. Additionally, the hip abductors promote frontal plane stability of the pelvis during the single limb support phase of gait [32]. The activation of the hip abductors prevents an excessive drop of the non-stance side pelvis (i.e., Trendelenburg gait). Hip abductor activation also results in a compressive force through the hip joint as the femoral head is approximated into the acetabulum to maintain frontal plane stability against the downward rotary torque created in the single leg stance position. Neumann [20] used a static equilibrium model to demonstrate the force interaction in the frontal plane. The hip abductor force (HAF) creates a counterclockwise force at the hip to counteract the clockwise force created by the force of gravity on the pelvis of the non-stance side. The balance of the forces results in a joint reaction force (JRF) that is directed through the hip joint as a result of establishing the balance of torques in the frontal plane [20].

Transverse Plane Osteokinematics

Hip motion in the transverse plane occurs around a longitudinal axis passing through the femoral head. The semispherical shape of the femoral head allows for a variable degree of rotation between individuals. Normal open chain hip internal rotation has been reported between 35° and 70° and external rotation between 45° and 90° [23, 24, 28]. The hip capsular ligaments and soft tissue structures are under greater tension when the hip is in a neutral or extended position; therefore, the amount of hip rotation increases as the hip is in a position of flexion. Closed chain transverse plane motion corresponds to anterior or posterior motion of the non-stance side pelvis. Hip internal rotation corresponds to an anterior rotation of the pelvis, while posterior motion of the pelvis causes hip external rotation [23]. Alterations in the normal osteokinematics of the hip in the transverse plane have been demonstrated in individuals with symptomatic FAI secondary to abnormal contact between the femur and acetabulum [15, 21].

Transverse Plane Arthrokinematics

Transverse plane hip arthrokinematics occur along a horizontal path along the femoral head and acetabulum [23]. The articular motion at the hip in the transverse plane is primarily a rotation and rollback with minimal translation, which is consistent with the other planes. It is for this reason that oftentimes hip instability is often referred to as a rotational instability as opposed to a translational instability as is observed at the shoulder joint [33].

Transverse Plane Muscle Actions

External rotation motion is caused by activation of the gluteus maximus and deep external rotator muscles during both open and closed chain functions (Table 1). Injury to the lower extremity kinetic chain has been linked to weakness of the hip external rotators resulting in a loss of transverse plane control during weight bearing [31].

The hip lacks a primary internal rotator muscle group secondary to the structural anatomy of the joint. However, multiple groups of muscles collectively contribute to internal rotation action at the hip. This internal rotation action that is created by these muscles increases is greater in a position of hip flexion as the moment arms of the hip abductors (i.e., anterior fibers of gluteus medius and minimus) are placed in a position of mechanical advantage to produce hip internal rotation [32]. In the neutral position, the adductor longus is in the most advantageous position to produce internal rotation as the line of muscle action is anterior to the longitudinal axis of rotation of the femur [32].

The Biomechanics of the Hip During Gait

Gait is one of the most common activities of daily living that begins early in the life span. The temporal and spatial parameters of human gait are usually established by the age of five [34]. Gait impairment can affect many aspects of physical function such as energy expenditure, mobility, strength, and neuromuscular control. These impairments can lead to an overall decrement of physical activity which has profound implications for the overall health and well-being of individuals of all ages. Injury and disease of multiple body systems can result in impairment of normal gait. This chapter will discuss musculoskeletal-derived gait impairments commonly encountered in orthopedic practice.

Walking is often thought as a relatively automatic and low-demand activity. However, human gait is extremely complex from a biomechanical perspective as it involves precise control and coordination between the nervous and musculoskeletal systems to be performed efficiently. The hip joint serves as the link between the trunk and lower extremities; therefore, coordinated action of this joint is required to maintain the appropriate coupling of motion between the upper and lower body regions. Injury or pathology of the hip joint can cause profound alterations to an individual's gait patterns. These gait impairments can cause significant functional limitations and eventual disability. It is important for orthopedic clinicians to understand biomechanical analysis of the gait cycle because the information may be used to guide evaluation and treatment.

Temporal and Spatial Parameters of Gait

The most basic biomechanical analysis of human gait involves measures of the temporal and spatial parameter (TSP) such as speed, stride length, and cadence [34]. Although a normal decline in these measures is seen with aging, they remain relatively stable throughout the life span. Many pathologies can lead to deterioration of the TSPs; therefore, they serve as a useful clinical measure of overall gait impairment. There are inherent associations between the TSPs of speed, cadence, and stride length. Cadence is a measure of steps per minute, such that a step is defined as one foot initial contact to the opposite foot initial contact. A stride consists of two consecutive steps or is defined as the time period from initial contact to initial contact of the same foot. A normal cadence for an uninjured individual is around 120 steps per minute or one stride per second. Inversely related to cadence is the measure of stride length which is defined as the distance covered during one stride. As stride length increases, cadence decreases to maintain what is known as "constant walk ratio" [34]. Gait speed is a measure of distance over time and is commonly reported in meters per second. Speed is a product of cadence, and stride length therefore is directly associated with each of these measures. Evaluation of the gait TSPs can assist in identifying a hip pathology through its impact on these measures.

Gait Analysis

The biomechanical variables that are often used to describe the features of the gait cycle are joint kinematics (i.e., angles) and joint kinetics (i.e., moments and power/energy). Muscle activation patterns are also measured to provide additional information on the muscular contributions to gait. The following section will describe the kinematics, kinetics, and muscle activations of the hip joint during the gait cycle.

Although the terminology used to describe the different phases of the gait cycle may vary between texts, the overall kinematic and kinetic patterns described are consistent [2, 3, 23, 34]. The phases of the gait cycle will be broken into the stance phase which encompasses from 0 % to 60 % of the cycle with two periods of double support lasting approximately 10 % each. The swing phase is defined as the last 60–100 % of the cycle with the end of the gait cycle being initial contact of the swing limb. The gait cycle will be discussed in reference to the right leg, and only the primary events of the cycle will be mentioned.

Sagittal Plane Gait Biomechanics

The gait cycle begins with initial contact (0 %) of the right foot heel strike with the hip in a position approximately 30–35° of flexion. This position places the ground reaction force anterior to the right hip joint creating a hip extension moment during the first 30 % of the cycle. The first double support phase is at between 10 % and 20 % of the gait cycle as both the right and left feet are in contact with the ground. Strong concentric action of the right hip extensors generates energy at the hip joint as the body is propelled forward over the limb. The hip moves into a near-neutral position in the sagittal plane (≈0°) at approximately 30 % of the gait cycle which is often referred to as mid-stance. Clinicians should be aware of the transition of the hip from a flexed position at initial contact to the neutral position at mid-stance because activation of the hip extensors has been shown to create a high degree of force across the anterior aspect of the hip joint in the open chain [14]. Therefore, individuals with injury to anterior structures such as the acetabular labrum or capsular ligaments may manifest an impairment during this phase of the gait cycle.

The right hip joint moves into a position of hip extension to approximately 10–15° from mid-stance through the second double support (50–60 %) as the right leg now prepares for the swing phase. As the vertical ground reaction force moves in the posterior direction during the cycle, a hip flexor moment is created which works to decelerate the trunk and pelvis over femur as the right hip joint moves into extension at the end of the stance phase. This moment and position create energy absorption through an eccentric contraction of the hip flexor muscles between approximately 30 % and 50 % of the gait cycle. The last portion of the stance phase (50–60 %) ends with the right hip in a position of maximal extension prior to initiating the swing phase. Individuals with intraarticular hip pathology such as OA may demonstrate gait impairments at the terminal stance portion of the gait cycle. A forward bend of the trunk during mid to terminal stance may indicate a loss of hip motion into extension as would be seen with intraarticular hip pathology. An excessive lumbar lordosis may also be observed in the case of intraarticular hip pathology which allows an individual to maintain the hip in a greater degree of hip flexion during the stance phase.

The swing phase of the gait cycle (60–100 %) in the sagittal plane is initiated through a brief concentric contraction of the right hip flexor muscles to initiate swing of the limb. This concentric muscle activation causes a short period of energy generation at the hip joint, but overall energy transfer following this initial burst is minimal during the rest of the swing phase. A small hip flexion moment is generated during this phase although it is thought to contribute minimally to swing. Hip pathology involving the hip flexor muscles could result in impairment during the swing phase. Reduced step length of the swing leg or a circumducted gait pattern may be observed if activation of the hip flexors is painful or reduced secondary to weakness.

Frontal Plane Gait Biomechanics

At initial contact, the right hip is in neutral position in the frontal plane and moves into the position of adduction through the closed chain motion of the left pelvic depression during the first 20 % of the gait cycle. The vertical ground reaction force is in a position that is medial to the axis of rotation of the right hip joint which creates a hip abductor moment during the early stance phase. This hip abductor moment drives eccentric activation of the right hip abductor muscles during the first 30 % of the cycle to prevent an excessive drop of the left pelvis. The hip abductor moment is maintained throughout the stance phase to maintain frontal plane pelvic control. The eccentric activation of the hip abductors corresponds with energy absorption at the right hip joint for the first 30 % of the cycle. Conversely, the right hip adductor muscles are active concentrically as they bring the right hip into adduction through the closed chain motion of the left pelvic depression. The right hip then moves toward a position of abduction just following mid-stance (≈45 %) as the left pelvis is elevated to a neutral position. This motion is driven through concentric activation of the right hip abductors during the latter half of the stance phase which contributes to a right hip abductor torque and a period of energy generation at the right hip joint.

The biomechanics of the hip in the frontal plane have been studied in relation to both hip and knee pathology [20, 29–31]. The most common gait deviations described in the frontal plane is the Trendelenburg or compensated Trendelenburg patterns. A Trendelenburg gait pattern is characterized by an excessive drop of the pelvis of the nonsupport limb during the stance phase of gait. A compensated Trendelenburg pattern is when a compensatory pelvic elevation of the nonsupport pelvis occurs with a simultaneous lateral trunk lean to the stance side. Both of these gait patterns are commonly observed in individuals who demonstrate considerable impairment of the hip abductor muscles. Weakness of the hip abductor musculature has been postulated as a contributing factor to overuse knee injury such as patellofemoral pain or iliotibial band syndrome (ITBS) [30, 31]. Frontal plane hip and pelvic biomechanics should be assessed during gait and other weight-bearing tasks because deviations of motion in the frontal plane may be indicative of hip injury.

The swing phase in the frontal plane is relatively uneventful in terms of forces at the hip joint. The hip moves toward a position of adduction during the swing phase (60–100 %). The moments, energy, and muscle activations are relatively minimal during the swing phase. Concentric activation of the hip adductors assists in driving the kinematics of the hip into adduction for the transition to initial contact at the beginning of the next gait cycle [34].

Transverse Plane Gait Biomechanics

The stance phase of the gait cycle begins with the right hip in a position of external rotation at initial contact (0 %). As the left pelvis begins to rotate forward, the right hip moves into internal rotation throughout much of the stance phase (10–50 %) although the overall magnitude of this rotation is less than 10°. The right hip transitions to external rotation motion at approximately 50 % of the stance phase and continues this external rotation through over half of the swing phase to approximately 85 % of the gait cycle. The right hip joint then transitions to internal rotation motion through the rest of the swing phase of the gait cycle (85–100 %).

The hip joint moment profiles in the transverse plane reveal a period of hip external rotation torque through the initial 30 % of the gait cycle to mid-stance with a hip internal rotation torque being generated from mid-stance to terminal stance (≈55 %) of the gait cycle. Swing phase hip joint moments are relatively minimal. The energy transfer at the hip joint follows the moment profile with a period of energy absorption associated with eccentric activity of the hip external rotators during early initial stance and the rest of the phases of gait demonstrating minimal energy transfer throughout the swing phase.

Alterations in transverse plane hip biomechanics have been observed in individuals with symptomatic FAI [15]. Individuals with symptomatic hip impingement demonstrated significantly less hip internal rotation motion and external rotation moments compared to healthy controls [15]. Additionally, changes in transverse plane control of the femur during single leg activities have been implicated in lower extremity kinetic chain injury [29, 31].

Summary

Biomechanical analysis can be extremely broad and considered to encompass everything from basic visual observation of movement to complex three-dimensional modeling and quantification of movement patterns. Orthopedic clinicians oftentimes measure biomechanical variables such as joint motion and muscle force in order to determine if impairment exists. The information provided by these measures is extremely important as it offers information on the functional impact of pathology such as deficiency with walking. The biomechanics of the hip joint have been extensively investigated especially with regard to normal gait and the pathology of hip osteoarthritis. These studies have revealed the impact that pathology can have on biomechanical alterations that effect normal function. Recent improvements in the diagnosis and treatment of hip pathology, such as with FAI and acetabular labral tears, have driven the need for a greater understanding of the impact these injuries may have on hip biomechanics during function.

References

1. Hatze H. The meaning of the term: "biomechanics". J Biomech. 1974;7:189–90.
2. Robertson DG, Caldwell GE, Hamill J, Kamen G, Whittlesey SL. Research methods in biomechanics. 2nd ed. Champaign: Human Kinetics; 2014.
3. Winter DA. Biomechanics and motor control of human movement. 3rd ed. Hoboken: Wiley; 2005.
4. Myer GD, Ford KR, Brent JL, Hewett TE. An integrated approach to change the outcome part I: neuromuscular screening methods to identify high ACL injury risk athletes. J Strength Cond Res. 2012;26:2265–71.
5. Rydell N. Biomechanics of the hip joint. Clin Orthop Relat Res. 1973;92:6–15.
6. Crowinshield RD, Johnston RC, Andrews JG, Brand RA. A biomechanical investigation of the human hip. J Biomech. 1978;11:75–85.
7. Hurwitz DE, Hulet CH, Andriacchi TP, Rosenberg AG, Galante JO. Gait compensations in patients with osteoarthritis of the hip and their relationship to pain and passive hip motion. J Orthop Res. 1997;15(4):629–35.
8. Rylander JH, Shu B, Andriacchi TP, Safran MR. Preoperative and postoperative hip kinematics in patients with femoroacetabular impingement during level walking. Am J Sports Med. 2011;39:36S–42.
9. Kennedy MJ, Lamontagne M, Beaule PE. Femoroacetabular impingement alters hip and pelvic biomechanics during gait: walking biomechanics of FAI. Gait Posture. 2009;30:41–4.
10. Lamontagne M, Kennedy MJ, Beaule P. The effect of cam FAI on hip and pelvic motion during maximum squat. Clin Orthop Relat Res. 2009;467:645–50.
11. Kivlan BR, Marin RL. Hip in athletes: a systematic review for reliability and validity. Int J Sports Phys Ther. 2012;7:402–12.
12. Delp SL, Maloney W. Effects of hip center location on the moment-generating capacity of the muscles. J Biomech. 1993;26:485–99.
13. Bergmann G, Deuretzbacher G, Heller M, et al. Hip contact forces and gait patterns from routine activities. J Biomech. 2001;34(7):859–71.
14. Lewis CL, Sahrmann SA, Moran DW. Anterior hip joint forces increases with hip extension, decreased gluteal force, and decreased iliopsoas force. J Biomech. 2007;40:3725–31.
15. Hunt MA, Gunether JR, Gilbart MK. Kinematic and kinetic differences during walking in patients with and without femoroacetabular impingement. Clin Biomech. 2013. http://dx.doi.org/10.1016/j.clinbiomech.2013.05.002.
16. Loureiro A, Mills PM, Barrett RS. Muscle weakness in hip osteoarthritis: a systematic review. Arthritis Care Res. 2013;65:340–52.
17. Casartelli NC, Maffiuletti NA, Item-Glatthorn JF, Bizzini SM, Impellizzeri FM, Leunig M. Hip muscle weakness in patients with symptomatic femoroacetabular impingement. Osteoarthritis Cartilage. 2011;19(7):816–21.
18. Neumann DA, Cook TM. Effect of load and carrying position on the electromyographic activity of the gluteus medius muscle during walking. Phys Ther. 1985;65:305–11.
19. Neumann DA, Cook TM, Sholty RL, Sobush DC. An electromyographic analysis of hip abductor muscle activity when subjects are carrying loads in one or both hands. Phys Ther. 1992;72:207–17.
20. Neumann DA. Biomechanical analysis of selected principles of hip joint protection. Arthritis Care Res. 1989;2:146–55.
21. Ganz R, Parvizi J, Beck M, Leunig M, Notzli H, Siebenrock KA. Femoroacetabular impingement: a

cause for osteoarthritis of the hip. Clin Orthop Relat Res. 2003;417:112–20.
22. Kang RW, Yanke AB, Espinoza Orias AA, Inoue N, Nho SJ. Novel 3-D quantification and classification of cam lesions in patients with femoroacetabular impingement. Clin Orthop Relat Res. 2013;471:358–62.
23. Neumann DA. Kinesieology of the musculoskeletal system foundations for rehabilitation. 2nd ed. St. Louis: Mosby Elsevier; 2010.
24. Polkowski GG, Clohisy JC. Hip biomechanics. Sports Med Arthrosc Rev. 2010;18:56–62.
25. Altman R, Alacaron G, Appelrouth D, Bloch D, Borenstein D, Brandt K, et al. The American college of rheumatology criteria for classification and reporting of osteoarthritis of the hip. Arthritis Rheum. 1991;34:505–14.
26. Arbanas J, Klasan GS, Nikolic M, Jerkovic R, Miljanovic I, Malnar D. Fibre type composition of the human psoas muscle with regard to the level of its origin. J Anat. 2009;215(6):636–41.
27. Tyler TF, Nicholas SJ, Campbell RJ, McHugh MP. The association of hip strength and symmetry with the incidence of adductor strains in professional ice hockey players. Am J Sports Med. 2001;29:124–8.
28. Dutton M. Orthopaedic examination, evaluation, & intervention. New York: McGraw Hill; 2004.
29. Niemuth PE, Johnson RJ, Myers MJ, Thieman TJ. Hip muscle weakness and overuse injuries in recreational runners. Clin J Sport Med. 2005;15:14–21.
30. Noehern B, Davis I, Hamill J. ASB clinical biomechanics award winner 2006 prospective study of the biomechanical factors associated with iliotibial band syndrome. Clin Biomech. 2007;22:951–6.
31. Souza RB, Powers CM. Differences in hip kinematics, muscle strength, and muscle activation between subjects with and without patellofemoral pain. J Orthop Sports Phys Ther. 2009;39:12–9.
32. Neumann DA. Kinesiology of the hip: a focus on muscular actions. J Orthop Sports Phys Ther. 2010;40:82–94.
33. Philippon MJ, Zehms CT, Briggs KK, Manchester DJ, Kuppersmith DA. Hip Instability in the athlete. Oper Tech Sports Med. 2010;15:189–94.
34. Kirtley C. Clinical gait analysis theory and practice. Philadelphia: Elsevier Churchill Livingstone; 2006.

Plain Radiographic Evaluation of the Hip

3

Simon D. Steppacher, Christoph E. Albers,
Moritz Tannast, and Klaus A. Siebenrock

Contents

S.D. Steppacher • C.E. Albers •
M. Tannast • K.A. Siebenrock (✉)
Department of Orthopedic Surgery, Inselspital,
University of Bern, Bern, Switzerland
e-mail: simon.steppacher@insel.ch; christoph.albers@insel.ch; moritz.tannast@insel.ch; klaus.siebenrock@insel.ch

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_3

Abstract

Plain radiographic imaging remains the standard imaging modality for the hip despite modern three-dimensional computer tomography or magnetic resonance imaging. To know the technical principles of radiographic imaging is essential for correct interpretation of plain radiographs. The anatomy of the hip on plain radiographs depends on the conical projection, film-tube and patient-film distance, centering and direction of the x-ray beam, and the pelvic orientation during radiograph acquisition. Standard radiographic evaluation of the hip comprises the anteroposterior pelvic radiograph and an axial view (e.g., cross-table). Hip-centered or deep-centered views are not recommended in hip-preserving surgery since the centering of the x-ray alters the projected anatomy on the radiograph. Additional views are performed to answer specific questions, e.g., a false profile view to judge the anterior acetabular coverage. This article summarizes and illustrates the most common radiographic parameters to describe the acetabular depth, acetabular coverage, acetabular orientation, head-neck sphericity, and joint congruency.

Introduction

Plain radiographs remain the standard imaging modality of the hip despite modern and three-dimensional modalities such as computer tomography (CT) or magnetic resonance imaging (MRI). Plain radiographs are relatively inexpensive, can be performed within a few minutes, and are widely available. A pelvic radiograph gives a good visualization of the overall morphology of the pelvis and the proximal femur. Additional projections can be performed to answer specific questions, e.g., a false profile to judge the anterior acetabular coverage. Plain radiographic imaging serves as the basis for surgical decision making in joint-preserving surgery, total hip arthroplasty, or trauma surgery. To know the technical principles of radiographic imaging is essential for correct interpretation. Plain radiographs are based on a point-shaped radiation source and a conical projection. The anatomy of the pelvis on the plain radiograph depends on technical and geometrical properties. To obtain reproducible radiographs of the hip, a standardized acquisition technique is mandatory.

This article describes (1) the technical properties of plain imaging affecting the anatomy of the hip on the radiograph, (2) summarizes the different radiographic projections of the hip, and (3) reports the radiographic parameters to describe the morphology of the acetabulum and proximal femur.

Technical Principles of Radiographic Imaging

For correct interpretation of plain radiographs, one has to understand the technical principles of radiographic imaging. In contrast to computer tomography (CT) or magnetic resonance imaging (MRI), a plain radiograph is a two-dimensional projection of the three-dimensional reality. The anatomy of the hip on a plain radiograph depends on multiple technical properties including the conical projection, film-tube distance, patient-film distance, centering and direction of the x-ray beam, and pelvic orientation during radiograph acquisition.

Conical Projection

Plain radiographs are based on a point-shaped x-ray source with conical projection. Therefore, distorsion of the pelvic anatomy on plain radiographs is unavoidable. Typically, the closer an object is located to the x-ray source, the more lateral it will be projected (Fig. 1). In contrast to the conical projection in plain radiographs, CT and MR imaging is performed with parallel x-ray beams not resulting in distorsion (Fig. 1).

Film-Tube Distance

The film-tube distance affects the anatomy of the hip on the radiograph, e.g., the acetabular

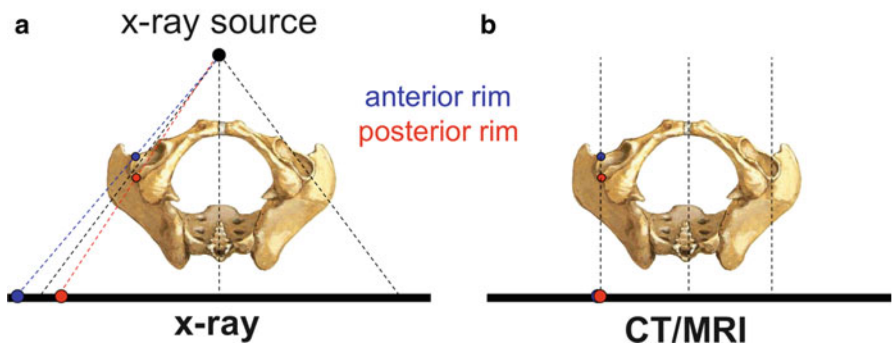


Fig. 1 (**a**) Plain radiographs are based on a point-shaped x-ray source and a conical projection. Therefore, distortion of the anatomy is unavoidable. Typically, the more anterior an object is located to the x-ray source (anterior rim in *blue*), the more lateral it is projected. (**b**) Computer tomography and magnetic resonance imaging are based on parallel rays and, therefore, these imaging modalities do not result in distortion (Reprinted with permission)

orientation. With increasing film-tube distance, the apparent acetabular anteversion increases. In contrast, with decreasing film-tube distance, the anteversion decreases and the acetabulum may appear retroverted (Fig. 2).

Patient-Film Distance

The patient-film distance has only a minor influence on the transformation of the hip on the radiograph [1]. The patient-film distance usually remains consistent for an individual patient. Interindividual differences exist in patient-film distance. But even in very obese patients, the distance between hip joints and the table shows minor variation since the increased body volume mainly affects the anterior part of the body (Fig. 3).

Centering and Direction of the X-Ray Beam

The centering of the x-ray beam is one of the most important factors influencing the anatomy of the hip on plain radiographs [1]. On a standard anteroposterior (AP) pelvic radiograph, the central beam is directed to the midpoint between the upper border of the symphysis and a line connecting both anterior superior iliac spines (Fig. 4). By lowering the center of the x-ray beam (low-centered AP pelvic radiograph), the apparent acetabular anteversion increases (Fig. 5). The acetabular anteversion also increases by moving the central beam from the center of the pelvis to the center of the hip (Fig. 6). Since the radiographic parameters to describe the morphology of the hip have been defined on pelvis-centered radiographs, it is not recommended to use low-centered or hip-centered radiographs for joint-preserving surgery.

Standard pelvic radiographs are performed with an AP direction of the x-ray beam. In contrast, chest radiographs are performed with a posteroanterior direction of the x-ray beam. Objects closer to the source of the x-rays (e.g., symphysis in AP direction) become magnified compared to those more distant (e.g., sacrum in AP direction). Since the hip is located in the middle of the pelvis, the direction of the x-ray beam has a minor effect on the projected anatomy on the plain radiograph.

Pelvic Orientation

The spatial orientation of the pelvis during radiograph acquisition can vary considerably which directly affects the projected morphology of the hip on the radiograph (Fig. 7). The orientation of the pelvis can vary in three dimensions: obliqueness, rotation, and tilt. While variations in pelvic obliqueness and rotation can be decreased by a

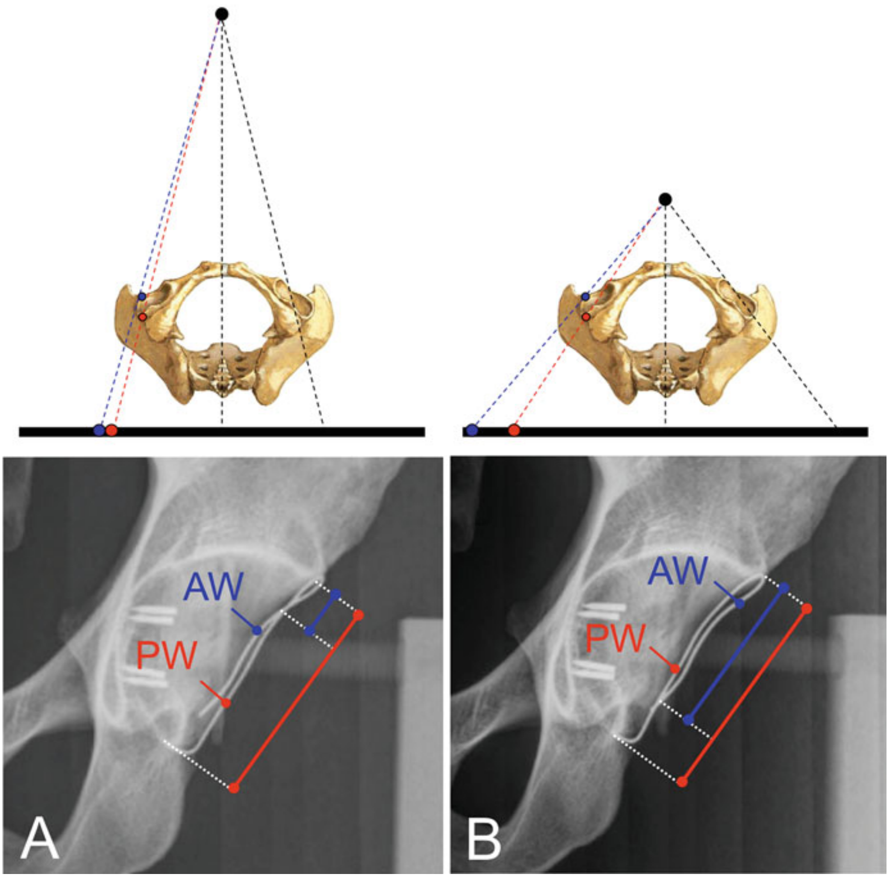


Fig. 2 The anatomy of the hip on a plain radiograph depends on the film-tube distance. (**a**) With a regular film-tube distance, a cranial retroversion exists indicated by a crossing of the anterior wall (*AW*) and posterior acetabular wall (*PW*). (**b**) By decreasing the film-tube distance, the apparent retroversion becomes more pronounced indicated by a more distal crossing of the *AW* and *PW* (Reprinted with permission)

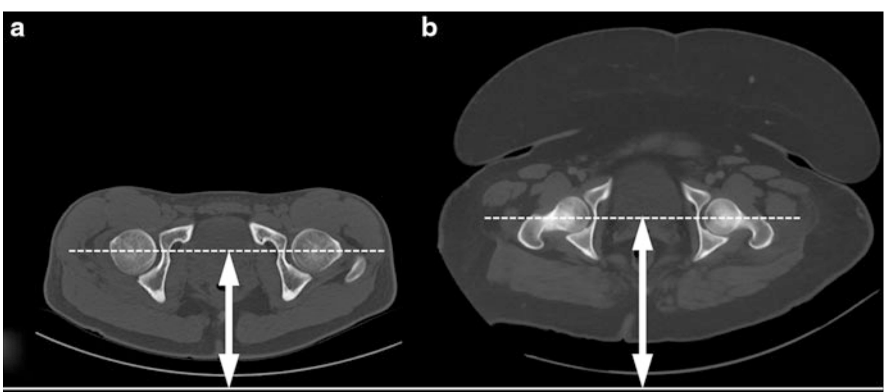


Fig. 3 The patient-film distance has a minor effect on the anatomy of the hip on the radiograph. (**a**) The patient-film distance shows a minor increase even in (**b**) obese patients since the increased body volume mainly affects the anterior part of the body

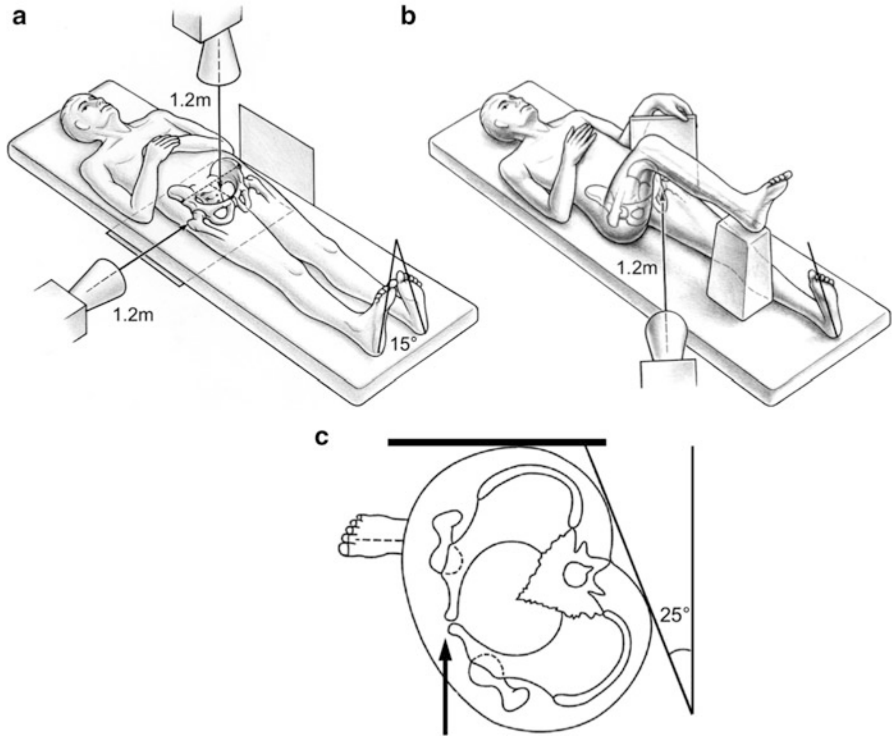


Fig. 4 Acquisition techniques for the different hip projections. (**a**) The anteroposterior (*AP*) pelvis radiograph is acquired with the patient supine, the legs 15° internally rotated to compensate for femoral antetorsion, and the x-ray beam centered to the midpoint between the upper part of the symphysis and a line connecting the anterior superior iliac spines. A concomitant true lateral view can be performed to evaluate pelvic tilt. The (**b**) cross-table axial view is performed with the patient supine, the x-ray beam angled in a 45° angle, and centered to the inguinal fold. The (**c**) false profile view is performed with the patient supine, with the contralateral hip tilted backward by 25°, and with the axis of the ipsilateral foot parallel to the film

standardized acquisition technique, pelvic tilt can vary substantially. An interindividual range of pelvic tilt up to 60° has been reported [2]. Pelvic tilt mainly affects the apparent anteversion of the acetabulum (Fig. 7).

An error due to pelvic obliqueness can simply be corrected by measuring the radiographic parameters according to an anatomical horizontal reference, e.g., the teardrop line. Pelvic rotation can be determined by the horizontal distance between the center of the sacrococcygeal joint and the center of the symphysis (Fig. 7). Ideally, these anatomical landmarks are in line (Fig. 7). Rotation to the right (left) side results in decreased (increased) acetabular anteversion on the right (left) side (Fig. 7). Pelvic tilt can be estimated by the vertical distance between the sacrococcygeal joint and the upper border of the symphysis (Fig. 7). In average this vertical distance is 47 mm in men and 32 mm in women [3]. However, to accurately determine pelvic tilt, a onetime true lateral pelvic view is needed [3]. With increasing pelvic tilt, the apparent acetabular anteversion decreases and vice versa (Fig. 7).

Fluoroscopy

Fluoroscopy differs in terms of acquisition technique compared to a regular AP pelvic view (Fig. 8). These differences have to be respected when interpreting fluoroscopic images. First, in fluoroscopy the x-ray beam is in posteroanterior direction instead of AP direction in a standard pelvic view. Next, the central beam is centered over the hip, and not the pelvis. Additionally, the

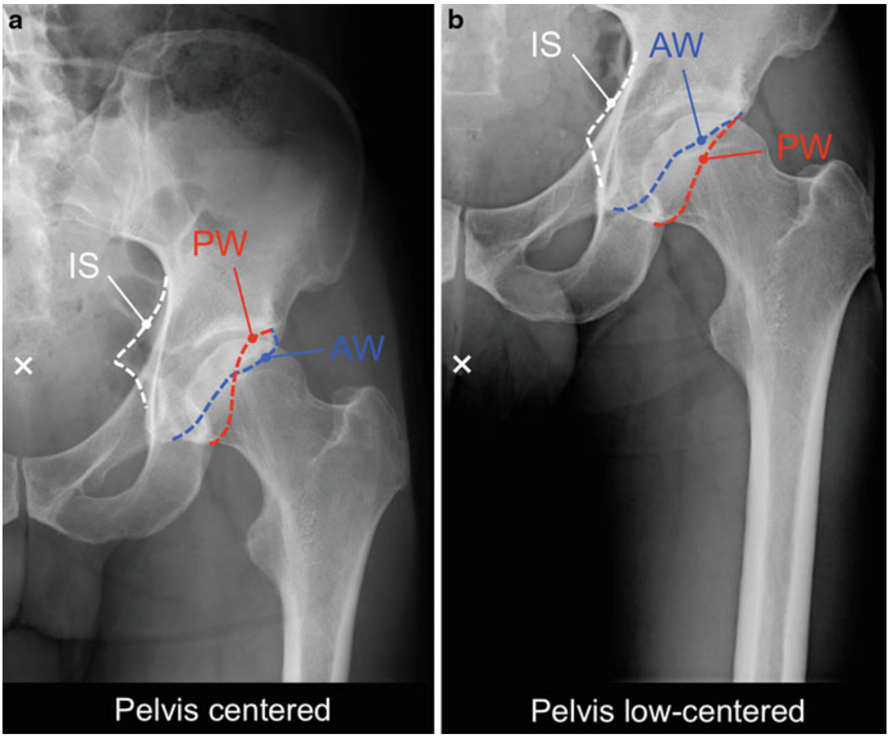


Fig. 5 Centering of the x-ray beam directly influences the anatomy of the hip on a plain radiograph, e.g., acetabular anteversion. (**a**) On the pelvis-centered radiograph (*white cross* represents central beam), the acetabulum is retroverted indicated by a crossing of the anterior (*AW*) and posterior acetabular wall (*PW*) and a positive ischial spine sign [ischial spine (*IS*) projected medially to the pelvic brim]. (**b**) On the low-centered radiograph, the crossover and the ischial spine signs are negative, and therefore, an acetabular retroversion would have been missed

tube-intensifier distance is decreased. This results in a more anteverted-appearing acetabulum on fluoroscopic images compared to regular pelvic radiographs (Fig. 8).

Radiographic Projections of the Hip

The standard radiographic evaluation of the hip includes two projections: the AP pelvic view and an axial view of the hip. In total hip arthroplasty, a deep-centered pelvic view or hip-centered view has been used. Due to the different centering of the x-ray beam in these views, the projected anatomy of the hip is significantly altered, and therefore, these views are not recommended in hip-preserving surgery. Different techniques have been described for the axial view of the hip including the axial cross-table, Dunn-Rippstein, Lauenstein, or frog-leg lateral views. In addition to the AP and axial view of the hip, additional projections exist which are performed to answer specific questions, e.g., the false profile to evaluate the anterior acetabular coverage.

Anteroposterior (AP) Pelvis View

Basic radiographic evaluation of the hip is performed with an AP pelvis view (Table 1). It gives a good visualization of the overall morphology of the pelvis and the proximal femur. It allows a comparison of the morphology of the symptomatic hip to the contralateral side. An AP pelvis

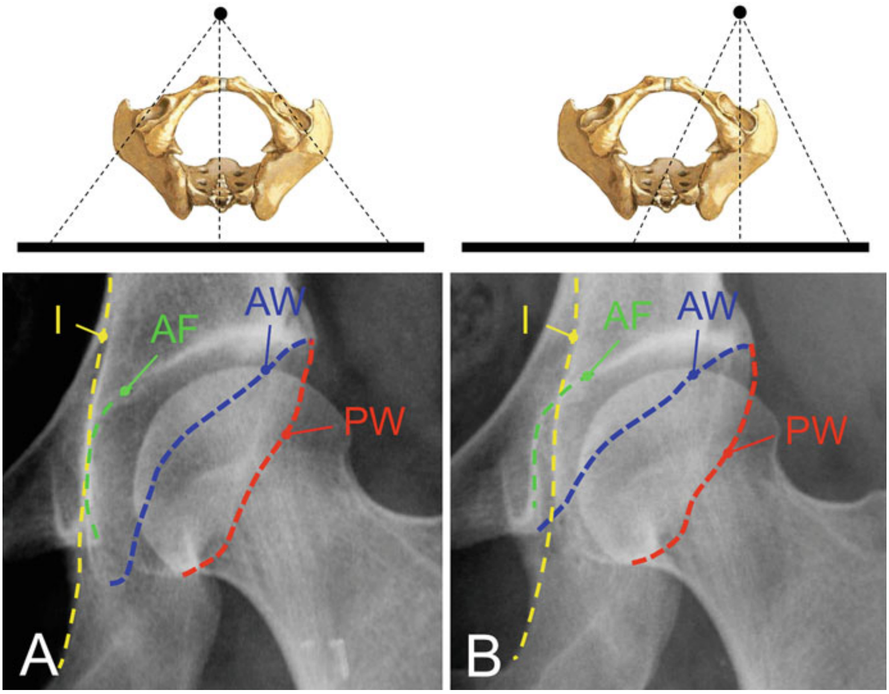


Fig. 6 The anatomy of the hip on a plain radiograph directly depends on the centering of the x-ray. When moving the center of the x-ray from (**a**) the center of the pelvis to the (**b**) center of the hip, the acetabulum becomes more anteverted indicated by a more distant course of the anterior (*AW*) and posterior acetabular wall (*PW*). In addition, the acetabulum appears deeper with a positive coxa profunda sign [acetabular fossa (*AF*) crossing the ilioischial line (*I*)]

view can be performed supine or standing. We favor pelvis radiographs performed in supine position because they can directly be compared to pelvic radiographs performed intraoperatively or at follow-up during early rehabilitation and restricted weight bearing. The legs are 15° internally rotated to compensate for femoral antetorsion (Fig. 4). The film-focus distance is 120 cm. The central beam is centered to the midpoint between the upper border of the symphysis and a line connecting the two anterior superior iliac spines.

Low-Centered Pelvis View

In total hip arthroplasty, low-centered pelvis views are performed to ensure that both cup and stem are entirely shown (Table 1). Due to different centering of the x-ray beam and its effect on the projected anatomy of the hip, low-centered pelvis views are not recommended for hip-preserving surgery (Fig. 5).

Anteroposterior (AP) Hip View

For the AP hip view, the central beam is centered to the hip (Table 1). Hip-centered views are mainly performed for total hip arthroplasty. The different centering of the x-ray results in an altered projection of the hip, and therefore, this view is not recommended for hip-preserving surgery (Fig. 6). On hip-centered radiographs, it is impossible to define the anatomical horizontal reference because of the missing contralateral landmark (e.g., teardrop figure). This makes the measurement of anatomical referenced cup inclination on

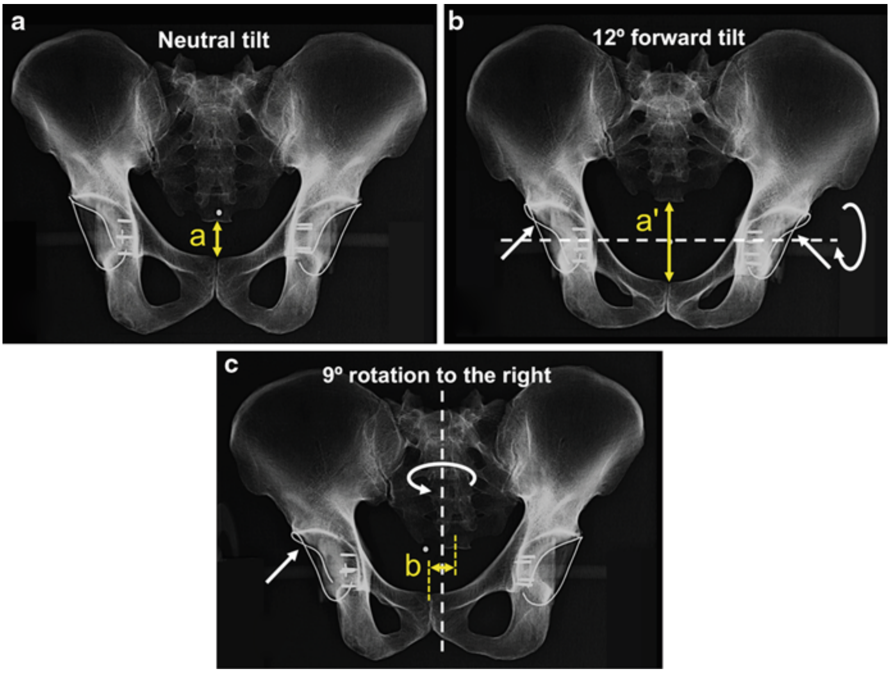


Fig. 7 Pelvic orientation during radiograph acquisition affects the projected anatomy of the hip. (**a**) A cadaver pelvis with metal-wire-marked acetabular walls is mounted in a holding device in neutral position. Both acetabula are anteverted. Pelvic tilt during radiograph acquisition is estimated by the vertical distance between sacrococcygeal joint and symphysis (distance a). (**b**) By a 12° forward tilt of the pelvis, both acetabula show a cranial retroversion (positive crossover signs indicated by *arrows*). The increased pelvic tilt is reflected by the increased distance a′. (*C*) A rotation of 9° towards the right side results in a cranial retroversion of the right acetabulum and an increased anteversion on the left side. Pelvic rotation is estimated by the horizontal distance b. Ideally, the distance b is 0 cm

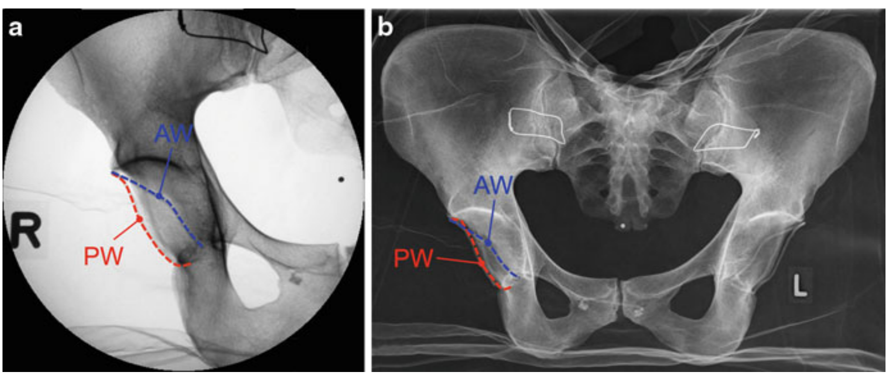


Fig. 8 (**a**) Fluoroscopic imaging is performed with a posteroanterior direction of the x-rays, a central beam centered over the hip, and a decreased tube-intensifier distance. This results in a more anteverted acetabular orientation. On the fluoroscopic image, the anterior and posterior acetabular walls do not cross, whereas on the (**b**) regular anteroposterior pelvis radiograph, there is a cranial crossover sign (*arrow*)

hip-centered radiographs impossible. In addition to an AP pelvis view, a hip-centered view can be used to differentiate whether a cup is anteverted or retroverted. While anteversion increases for an anteverted cup on a hip-centered view (Fig. 6), anteversion decreases for a retroverted cup on a hip-centered view compared to a pelvis-centered radiograph [4].

Table 1 Radiographic projections of the hip

Projection	Technique	Features
Anteroposterior (AP) pelvis	Patient supine or standing, legs 15° internally rotated, centering of x-ray beam to the midpoint between the upper border of symphysis and a line connecting both ASIS	Basic radiographic evaluation of the hip for hip-preserving surgery, total hip arthroplasty, or trauma surgery
Deep-centered pelvis	Patient supine or standing, legs 15° internally rotated, centering of the x-ray beam below the symphysis	Performed in total hip arthroplasty; not recommended for hip-preserving surgery due to different x-ray centering and altered morphology of the hip
Anteroposterior (AP) hip	Patient supine or standing, legs 15° internally rotated, hip-centered x-ray beam	Performed in total hip arthroplasty; not recommended for hip-preserving surgery due to different x-ray centering and altered morphology of the hip
Axial cross-table	Patient supine, ipsilateral leg 15° internally rotated, contralateral hip flex and elevated	Anterior and posterior femoral head-neck contour
Dunn-Rippstein	Patient supine, hips in 90° flexion and 20° abduction, knees in 90° of flexion, legs in holding device	Femoral antetorsion, anterior and posterior femoral head-neck contour
Modified Dunn	Patient supine, hips in 45° flexion and maximal abduction	Increased sensitivity to detect cam deformities in the anterosuperior head-neck area
Lauenstein	Patient supine, hip centered x-ray beam, hip and knee flexed, and hip in abduction	Anterior and posterior femoral head-neck contour
Frog-leg lateral	Patient supine, pelvis-centered x-ray beam, both hips and knee flexed, and the leg abducted so the soles of the feet contact	Anterior and posterior femoral head-neck contour
False profile	Patient standing, hip-centered x-ray beam, contralateral hip is tilted backward by 25°, ipsilateral foot remains parallel to radiographic table	Anterior acetabular coverage, anterosuperior subluxation, quantification of posteroinferior joint space
True lateral	Patient supine, horizontal x-ray beam from lateral and centered to the hip	Pelvic inclination; concomitantly performed with AP pelvis view to correlate pelvic inclination with vertical distance from symphysis to sacrococcygeal joint on AP pelvis view
Functional (abduction/ adduction)	Patient positioning and centering of x-ray for AP pelvis view; additional abduction or adduction of the hip, possible combination of abduction, internal rotation and flexion	Abduction view to differentiate between subluxation and true joint space narrowing in dysplastic hips, to simulate acetabular coverage following acetabular reorientation or femoral osteotomy

ASIS anterior superior iliac spine

Axial Cross-Table View

The axial cross-table view allows evaluating the anterior and posterior contour of the femoral head-neck junction (Fig. 9). This view is performed with the patient supine and the ipsilateral leg 15° internally rotated to compensate for femoral antetorsion (Fig. 4). The contralateral hip is flexed and the leg elevated (Fig. 4). The lateral x-ray beam is angled in a 45° angle and centered to the inguinal fold (Table 1).

Dunn-Rippstein View and Modified Dunn View

The Dunn-Rippstein view is performed with the patient supine (Table 1) [5, 6]. Both hips and knees are 90° flexed, and the legs are hold on a special holding device in 20° abduction. This hip view was originally introduced to measure exact femoral antetorsion [5, 6]. However, compared to CT-based measurements, this method is not free of error due to patient malpositioning [7].

Fig. 9 The axial cross-table view allows evaluating the anterior and posterior contour of the femoral head-neck junction. In hips with cam-type femoroacetabular impingement (*FAI*), the head-neck contour is aspherical (*arrow*)

The Dunn-Rippstein view can be used as an alternative to the axial cross-table view to evaluate the anterior and posterior contour of the femoral head-neck junction. Femoral head-neck asphericities in hips with FAI are most often localized in the anterosuperior region [8]. These asphericities are best shown with a "modified Dunn view" with the hips in 45° of flexion [9].

Lauenstein and Frog-Leg Lateral View

Both the Lauenstein [10] and frog-leg lateral views are performed with the patient supine (Table 1). The Lauenstein view is performed with the hip and knee flexed and the hip in abduction. The central beam is centered to the hip. If abduction of the hip is impaired, the pelvis can be tilted to the ipsilateral side. The frog-leg lateral view is taken with both hips and knee flexed and the leg abducted so the soles of the feet contact. Both views are used as an alternative to the axial cross-table view to evaluate the anterior and posterior contour of the femoral head-neck junction.

False Profile of Lequesne and de Sèze

The false profile view is performed with the patient standing (Table 1). The ipsilateral hip touches the radiographic table (Fig. 4). The contralateral hip is tilted backward by 25°. The axis of the ipsilateral foot remains parallel to the film. The false profile view is performed to evaluate the anterior acetabular coverage or anterosuperior subluxation of the femoral head which is of particular interest in dysplastic hips (Fig. 10). In addition, the false profile view allows to quantify the posteroinferior joint space (Fig. 10).

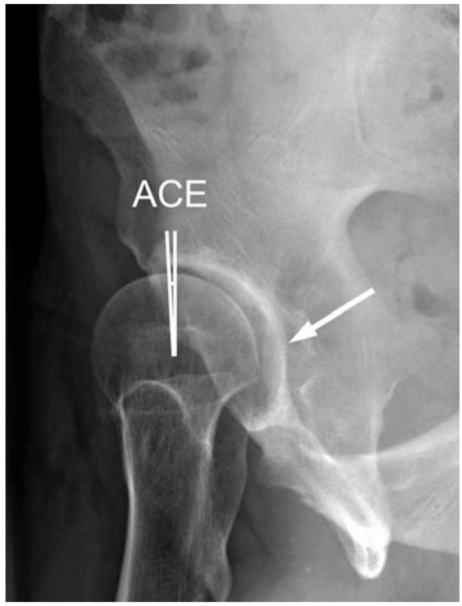


Fig. 10 A false profile view is performed to evaluate the anterior acetabular coverage by anterior center-edge (*ACE*) angle. In this patient with hip dysplasia, the anterior acetabular coverage is clearly deficient (ACE angle less than 25°). In addition, the false profile view allows to judge the posteroinferior joint space. Typically, dysplastic hips have a subluxation of the femoral head with an irregular joint space and widening of the posteroinferior joint space (*arrow*)

True Lateral Pelvis View

A true lateral pelvis radiograph can be performed to assess the pelvic tilt (Table 1). This view is acquired in the supine position and in addition to an AP pelvis view (Fig. 4). The pelvic tilt angle is constructed as the angle between a horizontal line and a line connecting the upper border of the symphysis and the sacral promontory (Fig. 11). Neutral tilt is defined as an angle of 60° [3, 11].

A linear correlation exists between the tilt angle (Fig. 11) and the vertical distance between the upper border of the symphysis and the sacrococcygeal joint on the AP pelvis radiograph (Fig. 7) [3]. Therefore, a true lateral view is only performed once for an individual patient. Exact tilt can be calculated from any other AP pelvis view using the linear correlation and the onetime determination of pelvic tilt on the true lateral view [3].

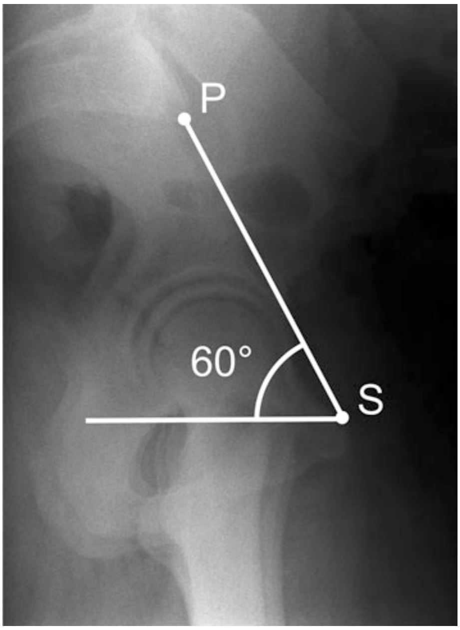


Fig. 11 On a true lateral pelvis radiograph, the pelvic tilt angle is constructed by a line connecting the sacral promontory (*P*) and the upper border of the symphysis (*S*) and a horizontal line. A pelvic tilt of 60° is defined as neutral position

Functional Views

Functional views of the hip are performed to assess joint congruency. The abduction view is used to differentiate between subluxation and joint space narrowing. In dysplastic hips there is typically an apparent joint space narrowing due to subluxation (Fig. 12). On the abduction view, these hips show a realignment of the femoral head with improvement of the joint space (Fig. 12). Sometimes flexion or internal rotation of the hip is needed to simulate an additional proximal femoral osteotomy. Persistent joint space incongruency on the abduction view represents cartilage loss and is a relative contraindication for joint-preserving surgery. Occasionally, an adduction view is indicated to simulate congruency following a valgus intertrochanteric osteotomy.

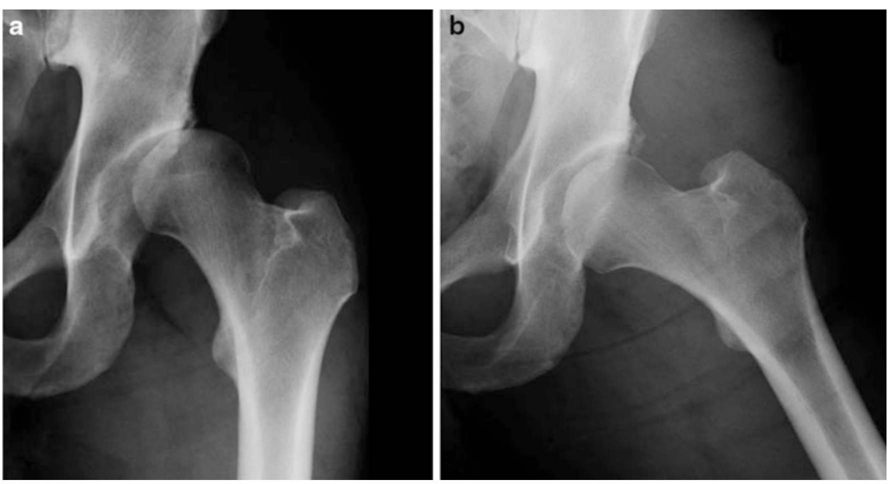


Fig. 12 (**a**) Dysplastic hips typically present with subluxation and decreased lateral joint space. To differentiate between subluxation and true joint space narrowing in dysplastic hips, an abduction view can be performed. (**b**) On the abduction view, the femoral head shows realignment and a regular joint space indicating subluxation without joint space narrowing. This hip would qualify for acetabular reorientation

Table 2 Radiographic parameters to describe the acetabular and proximal femoral morphology (see Fig. 13 for illustration)

Category	Parameter	Radiograph	Definition
Acetabular depth	**Coxa profunda [positive/ negative]**	AP pelvis	Acetabular fossa (AF) touches or crosses the ilioischial line (I)
	Protrusio acetabuli [positive/negative]	AP pelvis	Femoral head (F) touches or crosses the ilioischial line (I)
Acetabular coverage	**Lateral center-edge (LCE) angle [°]**	AP pelvis	Angle formed by a vertical line (v) and a line through the center of the femoral head (C) and the lateral edge of the acetabulum (L)
	Acetabular index [°]	AP pelvis	Angle formed by a horizontal line (h) and a line through the medial (M) and lateral edge (L) of the acetabular roof
	Extrusion index [%]	AP pelvis	Percentage of the femoral head width (w) which is not covered by the acetabulum (x)
	Sharp angle [°]	AP pelvis	Angle between a horizontal line (h) and a line connecting the acetabular teardrop (T) with the lateral edge of the acetabulum (L)
	Anterior center-edge (ACE) angle [°]	False profile	Angle formed by a vertical line (v) and a line through the center of the femoral head (C) and the anterior edge of the acetabulum (A)
Acetabular orientation	**Posterior wall sign [positive/ negative]**	AP pelvis	Positive if the posterior wall (PW) runs medially to the center of the femoral head (C)
	Anterior and posterior acetabular wall index [positive/negative]	AP pelvis	Ratio of the width of the anterior (AW)/posterior acetabular wall (PW) measured along the femoral head-neck axis (a) divided by the femoral head radius (r)
	Crossover sign [positive/ negative]	AP pelvis	Anterior wall (AW) crosses the posterior wall (PW)
	Retroversion index [%]	AP pelvis	Percentage of the retroverted acetabular opening (a) divided by the entire opening (b)
	Ischial spine sign [positive/ negative]	AP pelvis	Positive if the ischial spine (IS) is projected medially to the pelvic brim (PB)
Head-neck sphericity	**Alpha (beta) angle [°]**	Axial[a]	Angle formed by the femoral head-neck axis (a) and line through the center of the femoral head (C) and the point where the anterior (posterior) head-neck contour exceeds the head radius
	Gamma (delta) angle [°]	AP pelvis	Angle formed by the femoral head-neck axis (a) and line through the center of the femoral head (C) and the point where the cranial (caudal) head-neck contour exceeds the head radius
	Offset [mm]	Axial[a]	Difference (o) between the femoral head radius (r) and the neck radius
	Offset ratio []	Axial[a]	Ratio of offset (o) to the femoral head radius (r)
	Triangular index []	AP pelvis	A perpendicular line (p) is drawn at half the head radius (r). Distance (R) is measured from the femoral head center (C) to the point where p intersects the anterior femoral head-neck contour. The triangular index is positive if $R \geq r + 2$ mm
Joint congruency	**Shenton's line [intact/ interrupted]**	AP pelvis	Interrupted if the caudal femoral head-neck contour and the superior border of the obturator foramen do not form a harmonic arc
	Lateralization of femoral head [mm]	AP pelvis	Shortest distance between the medial aspect of the femoral head (F) and the ilioischial line (I)

(*continued*)

Table 2 (continued)

Category	Parameter	Radiograph	Definition
Additional findings	**Centrum collum diaphyseal (CCD) angle [°]**	AP pelvis	Angle formed by the femoral head-neck axis (a) and the femoral shaft axis (s)
	Fovea angle delta [°]	AP pelvis	Angle formed by a line through the medial edge of the acetabular roof (M) and the center of the femoral head (C) and a line through the lateral border of the fovea capitis femoris (F) and the center of the femoral head (C)

AP anteroposterior
[a]Cross-table, Dunn, or Lauenstein view

Radiographic Parameters

The radiographic parameters to describe the morphology of the acetabulum and proximal femur are grouped as follows: acetabular depth, acetabular coverage, acetabular orientation, head-neck sphericity, joint congruency, and additional parameters.

Acetabular Depth

Acetabular depth has been quantified using the coxa profunda sign and the protrusio sign (Table 2, Fig. 13). The coxa profunda sign is considered positive if the acetabular fossa touches or crosses the ilioischial line (Fig. 14). Recently, it could be shown that the prevalence of a positive coxa profunda sign is not increased in hips with pincer impingement compared to normal hips [12]. Therefore, the coxa profunda sign is considered a normal radiographic finding [12]. The protrusio sign is positive if the femoral head touches or crosses the ilioischial line (Fig. 14). In contrast to the coxa profunda sign, the protrusio sign only occurs in very deep hips. Primary hip protrusio occurs predominantly in females and bilateral. It has to be distinguished from secondary hip protrusio, e.g., following end-stage osteoarthrosis or a fracture [13, 14].

Acetabular Coverage

Lateral acetabular coverage is quantified by the lateral center-edge (LCE) angle, acetabular index (AI), or the extrusion index (Table 2, Fig. 13). The LCE angle is constructed by the lateral acetabular edge, the center of the femoral head, and the vertical anatomical reference (perpendicular line to the teardrop line). An LCE angle of less than 25° is defined as dysplastic [15], and an angle exceeding 39° is considered a pincer hip (Table 3) [16]. The AI, also known as acetabular roof angle, is constructed by a horizontal line and a line passing through the lateral and medial edge of the acetabular roof (Table 2, Fig. 13). An AI of 14° or more is defined as dysplastic [16] and a negative AI as a pincer hip (Table 3). The extrusion index is the percentage of femoral head width which is uncovered by the acetabulum (Table 2, Fig. 13). A normal extrusion index ranges from 17 % to 27 % (Table 3) [17].

The anterior acetabular coverage is quantified on the false profile view using the anterior center-edge (ACE) angle (Fig. 10). It is constructed by a vertical line and a line passing through the anterior acetabular edge and the center of the head (Fig. 10). A minimal angle of 25° is defined as normal (Table 3). The posterior acetabular coverage is judged using the posterior wall sign which is positive if the posterior wall runs medial to the femoral head center (Fig. 15) [18]. The acetabular wall index can be calculated to quantify either anterior or posterior acetabular coverage (Table 2) [19]. The anterior (posterior) acetabular wall index is calculated as the ratio of the width of the anterior (posterior) acetabular wall divided by the femoral head radius (Fig. 13). This index correlates strongly with area-based measurements of anterior and posterior acetabular coverage from a validated computer analysis model (Table 3) [19].

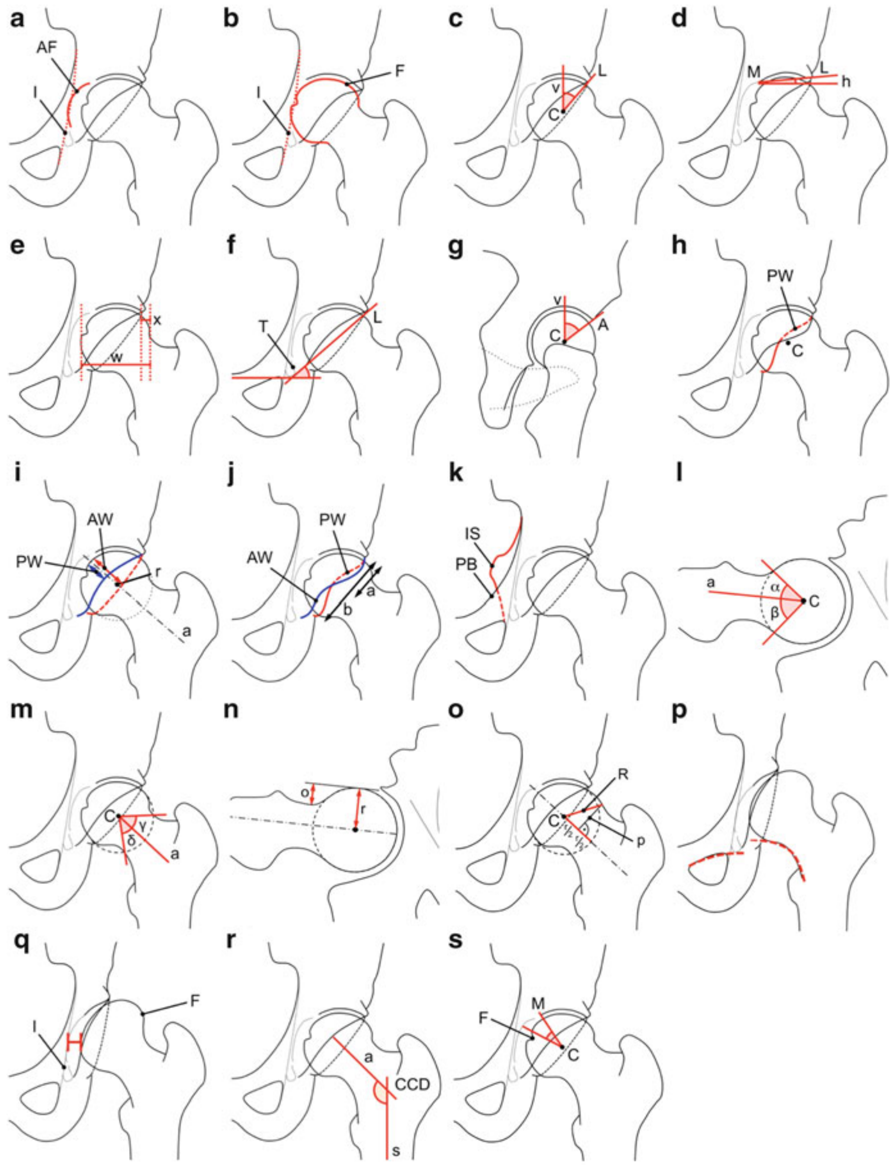


Fig. 13 Radiographic parameters to describe the acetabular and proximal femoral morphology. See Table 2 for definitions. *A* Coxa profunda, *B* protrusio, *C* lateral center-edge (*LCE*) angle, *D* acetabular index, *E* extrusion index, *F* sharp angle, *G* anterior center-edge (*ACE*) angle, *H* posterior wall sign, *I* anterior and posterior acetabular wall index, *J* crossover sign with extrusion index, *K* ischial spine sign, *L* alpha and beta angle, *M* gamma and delta angle, *N* offset and offset ratio, *O* triangular index, *P* Shenton's line, *Q* lateralization of femoral head, *R* centrum collum diaphyseal (*CCD*) angle, and *S* fovea angle delta

Acetabular Orientation

In a normal and anteverted acetabulum, the anterior acetabular rim runs medial to the posterior rim. In addition, the posterior acetabular rim runs lateral to the femoral head center (negative posterior wall sign [18]). Acetabular retroversion is an acetabular pathomorphology with malorientation of the acetabular opening which is partially or completely facing

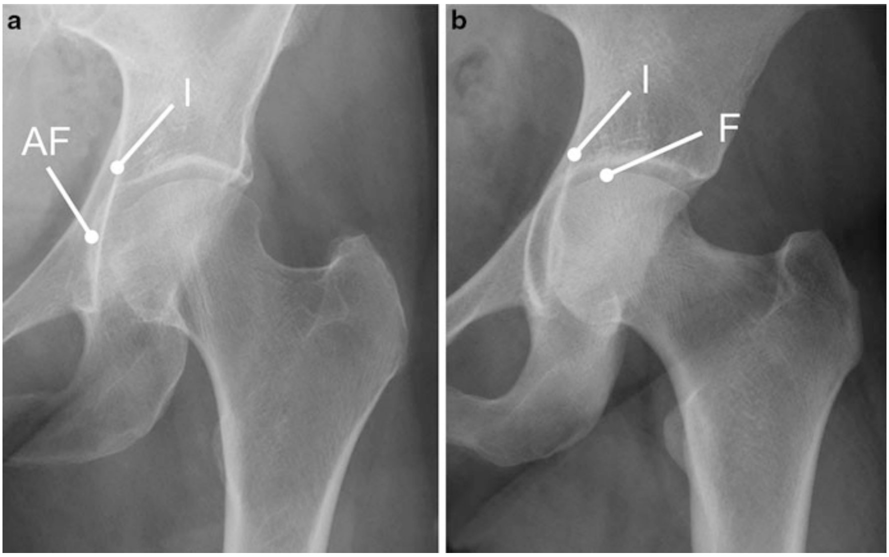


Fig. 14 (**a**) The coxa profunda sign is positive if the acetabular fossa (*AF*) touches or crosses the ilioischial line (*I*). The coxa profunda sign is considered a normal radiographic finding. (**b**) The protrusio acetabuli sign is positive if the femoral head (*F*) touches or crosses the ilioischial line (*I*)

posteriorly. Typical radiographic signs are a positive crossover sign [18], ischial spine sign [20], and posterior wall sign [18]. The crossover sign is positive if the anterior acetabular wall crosses the posterior acetabular wall (Fig. 15) [18]. The retroversion index quantifies the acetabular retroversion and is defined as the percentage of the retroverted cranio-lateral acetabular opening to the entire opening (Table 2; Fig. 13) [21]. The ischial spine sign is considered positive if the ischial spine is projected medially to the pelvic brim (Fig. 15) [20]. A more recent study could show that acetabular retroversion is not an isolated pathomorphology of the acetabulum, but rather a malorientation of the entire hemipelvis [22]. In hips with an acetabular retroversion, the entire innominate bone is externally rotated [22]. This explains the associations of acetabular retroversion with extra-articular anatomical landmarks such as the ischial spine sign [22].

Head-Neck Sphericity

Normally, the head-neck contour is spherical and concave. In hips with cam-type femoroacetabular impingement (FAI), the head-neck contour is aspherical (Fig. 9). The asphericity is typically located in the anterosuperior head-neck quadrant and limits internal rotation in flexion [23]. The head-neck sphericity can be quantified by the alpha angle, offset, offset ratio, or triangular index. Asphericities in the anterosuperior head-neck quadrant are best seen on an axial hip view (cross-table, Dunn, or Lauenstein view) and often hidden on the AP pelvis view. The alpha angle is defined by the femoral head-neck axis and a line passing through the center of the femoral head and the point where the anterior head-neck contour exceeds the head radius (Table 2; Fig. 13). A normal alpha angle is less than 50°, and an alpha angle exceeding 50° defines a cam-type morphology (Table 3) [24]. On the axial view, the angle describing the head-neck sphericity on the posterior side is the beta angle (Fig. 13) [25]. The asphericity of the femoral head-neck junction can also be characterized by the femoral offset. The offset is defined as the difference between the femoral head radius and the neck radius (Table 2; Fig. 13). The offset ratio is the ratio of the offset divided by the femoral head radius (Table 2; Fig. 13).

Table 3 Normal and pathological values of the described radiographic parameters

Category	Parameter	Normal	Hip dysplasia	Pincer impingement	Cam impingement
Acetabular depth	**Acetabular fossa**	Normal or coxa profunda	Normal or coxa profunda	Coxa profunda or protrusio	n.a.
Acetabular coverage	**Lateral center-edge (LCE) angle [°]**	20–39	<20	>39	n.a.
	Acetabular index [°]	0–14	>14	<0	n.a.
	Extrusion index [%]	17–27	>27	12–16	n.a.
	Sharp angle [°]	33–38	>47	Not described	n.a.
	Anterior center-edge (ACE) angle [°]	>25	<20	Not described	n.a.
Acetabular orientation	**Posterior wall sign []**	Negative	Often positive	Positive with retroversion	n.a.
				Negative with protrusion	
	Anterior acetabular wall index []	0.41 (Range, 0.30–0.51)	0.28 (Range, -0.06–0.52)	0.61 (Range, 0.24–0.89)	n.a.
	Posterior acetabular wall index []	0.91 (Range, 0.81–1.14)	0.81 (Range, 0.35–1.04)	1.15 (Range, 0.73–1.61)	n.a.
	Crossover sign []	Negative	Often positive	Positive with retroversion	n.a.
	Ischial spine sign []	Negative	Not described	Positive with retroversion	n.a.
Head-neck sphericity	**Alpha angle [°]**	<50	Often >50	n.a.	> 50
	Beta angle [°]	42 ± 7	Not described	Not described	Not described
	Gamma angle [°]	53 ± 13	Not described	Not described	Not described
	Delta angle [°]	43 ± 5	Not described	Not described	Not described
	Offset [mm]	>10 mm	Not described	>10 mm	<8 mm
	Offset ratio []	>0.20	Not described	>0.20	<0.18
	Triangular index []	Negative	Not described	Negative	Positive
Joint congruency	**Shenton's line [intact/ interrupted]**	Intact	Often interrupted	Intact	Intact
	Lateralization of femoral head [mm]	Not described	~16	Not described	Not described
Additional findings	**Centrum collum diaphyseal (CCD) angle []**	129–135	>135	<129	Not described
	Fovea angle delta [°]	26 ± 10	Not described	Not described	Not described

Adapted according to Tannast et al. [32], *n.a.* not applicable

An asphericity in the superior part of the femoral head-neck area is referred to as pistol grip deformity and detectable on the AP pelvis radiograph (Fig. 16). Analogously to the alpha angle on the axial view, the gamma (delta) angle describes the cranial (caudal) asphericity on the AP pelvis view (Table 2; Fig. 13) [25]. In addition, the triangular index has been described for quantification of asphericity in the superior part of the femoral head-neck area (Table 2; Fig. 13) [26]. The femoral neck radius is measured on the cranial side at a distance of half of the

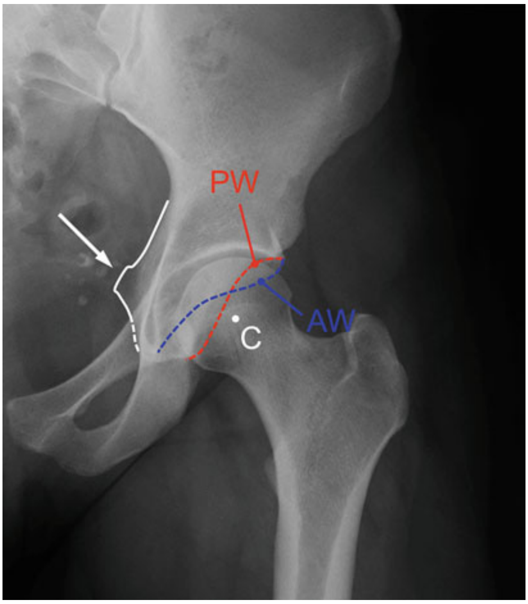


Fig. 15 Acetabular retroversion is defined by a positive crossover, posterior wall, and ischial spine sign. The crossover sign is positive if the anterior acetabular wall (*AW*) crosses the posterior acetabular wall (*PW*) [18]. The posterior wall sign is positive if the posterior wall (*PW*) runs medial to the femoral head center (*C*) [18]. The ischial spine sign is positive if the ischial spine (*IS*) is projected medially to the pelvic brim

femoral head radius (Table 2; Fig. 13). If the neck radius exceeds the head radius plus 2 mm, the triangular index is considered positive (Table 2; Fig. 13).

Joint Congruency

Joint incongruency due to subluxation in dysplastic hips is best assessed with a functional view with the hip in abduction (Fig. 12). Alternatively, the cranial subluxation can be assessed by Shenton's line [27]. Shenton's line is considered intact if the caudal femoral head-neck contour and the superior border of the obturator foramen build a harmonic arc on the AP pelvis view (Fig. 17). Lateralization of the femoral head can be quantified by the distance between the most medial aspect of the femoral head and the ilioischial line (Table 2; Fig. 13).

Fig. 16 A pistol grip deformity is an asphericity in the superior part of the femoral head-neck area which is detectable on an AP pelvis radiograph

Additional Findings

The relationship between the femoral neck and the femoral shaft in the frontal plane is quantified by the neck shaft angle (also known as centrum collum diaphyseal [CCD] angle; Fig. 13). In a normal hip, the neck shaft angle ranges from 129° to 135° (Table 3) [25, 28]. In a valgus hip, the CCD angle in increased and decreased in a varus hip. In a hip with a normal CCD angle, a horizontal line at the height of the tip of the greater trochanter runs approximately through the center of the femoral head. In a valgus (varus) hip, this horizontal line runs caudal (cranial) to the femoral head center (Fig. 17).

In a normal hip on the AP pelvis view, the fovea capitis femoris is not directly in contact with the weight-bearing area of the acetabulum (Fig. 13). The fovea angle delta is defined as the angle between the cranial extension of the fovea capitis femoris and the medial portion of the sclerotic zone (acetabular sourcil). In a normal population, this angle is 26 ± 10° (Table 3) [29].

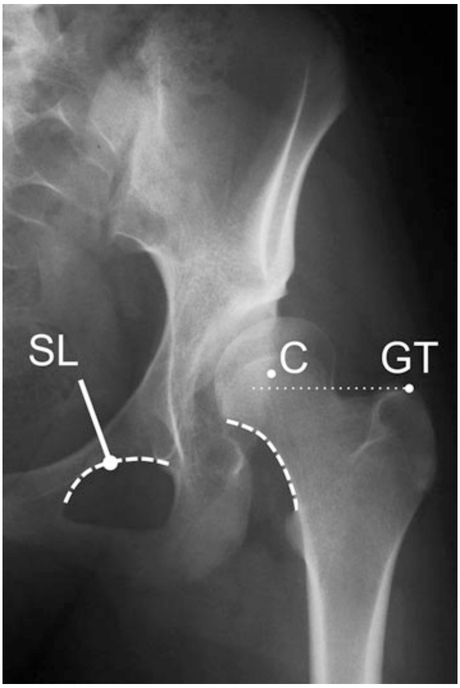


Fig. 17 Subluxation in hip dysplasia is assessed by Shenton's line (*SL*). If the caudal femoral head-neck contour and the superior border of the obturator foramen do not build a harmonic arc on the AP pelvis view, Shenton's line is considered interrupted. In a valgus hip a horizontal line at the height of the tip of the greater trochanter (*GT*) typically runs cranially to the femoral head center (*C*)

Computer-Assisted Evaluation of Plain Pelvis Radiographs

Parameters to describe acetabular coverage and orientation measured on a plain radiograph directly depend on the pelvic orientation during radiograph acquisition. In particular, pelvic tilt shows a large interindividual variability [2]. A validated computer software called *hip2norm* to correct the measured radiographic parameters for malorientation of the pelvis exists [30, 31]. Together with a onetime true lateral pelvis radiograph, the radiographic parameters can be corrected to the pelvis neutral position with 60° of inclination.

Summary

Correct interpretation of plain radiographs is not possible without knowing the technical principles of this imaging modality. The anatomy of the hip on plain radiographs depends on the conical projection, film-tube and patient-film distance, centering and direction of the x-ray beam, and the pelvic orientation during radiograph acquisition. The standard radiographic evaluation of the hip with an AP pelvis radiograph and an axial view can be completed with additional projections such as a false profile, true lateral, or functional view. Normal and pathological values for acetabular depth, acetabular coverage, acetabular orientation, head-neck sphericity, and joint congruency have been defined on plain radiographs. Plain radiographic imaging of the hip remains an essential imaging modality in everyday orthopedic practice despite modern three-dimensional modalities.

References

1. Burckhardt K. Theoretical study to the sub-project 'Interactive software for 2D and 3D standardization of pelvic radiographs and CT-scans for accurate evaluation of hip joint morphology' under CO-ME Project 4. Swiss Federal Institute of Technology. 2003; Report No.: 267.
2. Tannast M, Langlotz U, Siebenrock KA, Wiese M, Bernsmann K, Langlotz F. Anatomic referencing of cup orientation in total hip arthroplasty. Clin Orthop Relat Res. 2005;436:144–50.
3. Tannast M, Zheng G, Anderegg C, Burckhardt K, Langlotz F, Ganz R, Siebenrock KA. Tilt and rotation correction of acetabular version on pelvic radiographs. Clin Orthop Relat Res. 2005;438:182–90.
4. Seradge H, Nagle KR, Miller RJ. Analysis of version in the acetabular cup. Clin Orthop Relat Res. 1982;166:152–7.
5. Dunn DM. Anteversion of the neck of the femur; a method of measurement. J Bone Joint Surg Br. 1952;34 (2):181–6.
6. Rippstein J. Zur Bestimmung der Antetorsion des Schenkelhalses mittels zweier Röntgenaufnahmen. Z Orthop Ihre Grenzgeb. 1955;86:345–60.
7. Wissing H, Buddenbrock B. Determining rotational errors of the femur by axial computerized tomography in comparison with clinical and conventional radiologic determination. Unfallchirurgie. 1993;19(3):145–57.

8. Tannast M, Goricki D, Beck M, Murphy SB, Siebenrock KA. Hip damage occurs at the zone of femoroacetabular impingement. Clin Orthop Relat Res. 2008;466(2):273–80.
9. Meyer DC, Beck M, Ellis T, Ganz R, Leunig M. Comparison of six radiographic projections to assess femoral head/neck asphericity. Clin Orthop Relat Res. 2006;445:181–5.
10. Lauenstein C. Nachweis der "Kocherschen Verbiegung" des Schenkelhalses bei der Coxa vara durch Röntgenstrahlen. Fortschr Röntgenstr. 4:61–4.
11. Williams PL. The skeleton of the lower limb. In: Williams PL, Warkick R, Dyson M, Bannister LH, editors. Gray's anatomy. Edinburgh: Churchill Livingstone; 1989.
12. Nepple JJ, Brophy RH, Matava MJ, Wright RW, Clohisy JC. Radiographic findings of femoroacetabular impingement in National Football League combine athletes undergoing radiographs for previous hip or groin pain. Arthrosc J Arthrosc Relat Surg. 2012;28 (10):1396–403.
13. Alexander C. The aetiology of primary protrusio acetabuli. Br J Radiol. 1965;38:567–80.
14. Gilmour J. Adolescent deformities of the acetabulum an investigation into the nature of protrusio acetabuli. Br J Surg. 1939;26(104):670–99.
15. Murphy SB, Ganz R, Muller ME. The prognosis in untreated dysplasia of the hip. A study of radiographic factors that predict the outcome. J Bone Joint Surg Am. 1995;77(7):985–9.
16. Tonnis D, Heinecke A. Acetabular and femoral anteversion: relationship with osteoarthritis of the hip. J Bone Joint Surg Am. 1999;81(12):1747–70.
17. Tannast M, Albers CE, Steppacher SD, Siebenrock K. Hip pain in the young adult. In: Bentley G, editor. European instructional lectures, vol. 11. New York: Springer; 2011.
18. Reynolds D, Lucas J, Klaue K. Retroversion of the acetabulum. A cause of hip pain. J Bone Joint Surg Br. 1999;81(2):281–8.
19. Siebenrock KA, Kistler L, Schwab JM, Buchler L, Tannast M. The acetabular wall index for assessing anteroposterior femoral head coverage in symptomatic patients. Clin Orthop Relat Res. 2012;470:3355–60.
20. Kalberer F, Sierra RJ, Madan SS, Ganz R, Leunig M. Ischial spine projection into the pelvis: a new sign for acetabular retroversion. Clin Orthop Relat Res. 2008;466(3):677–83.
21. Siebenrock KA, Kalbermatten DF, Ganz R. Effect of pelvic tilt on acetabular retroversion: a study of pelves from cadavers. Clin Orthop Relat Res. 2003;407:241–8.
22. Tannast M, Pfannebecker P, Schwab JM, Albers CE, Siebenrock KA, Buchler L. Pelvic morphology differs in rotation and obliquity between developmental dysplasia of the hip and retroversion. Clin Orthop Relat Res. 2012;470(12):3297–305.
23. Tannast M, Siebenrock KA, Anderson SE. Femoroacetabular impingement: radiographic diagnosis–what the radiologist should know. AJR Am J Roentgenol. 2007;188(6):1540–52.
24. Notzli HP, Wyss TF, Stoecklin CH, Schmid MR, Treiber K, Hodler J. The contour of the femoral head-neck junction as a predictor for the risk of anterior impingement. J Bone Joint Surg Br. 2002;84 (4):556–60.
25. Toogood PA, Skalak A, Cooperman DR. Proximal femoral anatomy in the normal human population. Clin Orthop Relat Res. 2009;467(4):876–85.
26. Gosvig KK, Jacobsen S, Palm H, Sonne-Holm S, Magnusson E. A new radiological index for assessing asphericity of the femoral head in cam impingement. J Bone Joint Surg Br. 2007;89(10):1309–16.
27. Shenton EWH. Disease in bone and its detection by the X-rays. London: Macmillan; 1911.
28. Hoaglund FT, Low WD. Anatomy of the femoral neck and head, with comparative data from Caucasians and Hong Kong Chinese. Clin Orthop Relat Res. 1980;152:10–6.
29. Notzli HP, Muller SM, Ganz R. The relationship between fovea capitis femoris and weight bearing area in the normal and dysplastic hip in adults: a radiologic study. Z Orthop Ihre Grenzgeb. 2001;139 (6):502–6.
30. Tannast M, Mistry S, Steppacher SD, Reichenbach S, Langlotz F, Siebenrock KA, Zheng G. Radiographic analysis of femoroacetabular impingement with Hip2Norm-reliable and validated. J Orthop Res. 2008;26(9):1199–205.
31. Tannast M, Mistry S, Steppacher SD, Reichenbach S, Siebenroc KA, Zheng GA. A comprehensive validation of a new method for correction of radiographic hip parameters for pelvic malpositioning. J Bone Joint Surg Br. 2009;91-B(SUPP III):451.
32. Tannast M, Siebenrock K. Imaging: plain radiographs. In: Sekiya JK, Safran MR, Ranawat AS, Leunig M, editors. Techniques in hip arthroscopy and joint preservation. Philadelphia: Elsevier Saunders; 2011.

Computer Tomography Scan of the Hip and Pelvis

4

Richard W. Kang, Caroline Park, and Anil Ranawat

Contents

R.W. Kang • C. Park (✉) • A. Ranawat
Sports Medicine and Shoulder Surgery, Hospital for Special Surgery, New York, NY, USA
e-mail: rwkang.md@gmail.com; parkc@hss.edu; ranawatanil@hss.edu

Abstract

Accurate preoperative assessment of osseous morphology is critical in the diagnosis and management of various hip and pelvic conditions, such as femoroacetabular impingement (FAI), acetabular dysplasia, traumatic hip instability, and other complex pediatric disorders. Plain radiography, ultrasound, and magnetic resonance imaging (MRI) have all been used to assess abnormal hip morphology pre- and postoperatively; however, computed tomography (CT) has emerged as the gold standard for detecting osseous deformities of hip and pelvic pathologies. Technical advancements have allowed this imaging modality to provide more precise and accurate anatomic characterizations of complex, bony lesions with lower doses of radiation than historical scanning. This review focuses on the use of computed tomography scanning in the assessment of a variety of hip pathologies.

Introduction

Computed tomography (CT) scanning is an invaluable tool in evaluating the hip and pelvis. Both obvious and subtle osseous abnormalities are effectively identified by CT scans. Thus, CT scans are useful for evaluating a variety of hip and pelvis conditions including femoroacetabular impingement (FAI) and acetabular dysplasia (AD) and complex pediatric disorders including malalignment

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_4

syndromes, traumatic hip instability, acetabular and avulsion fracture, and the component positioning after total hip arthroplasty (THA).

Recent advancements in CT imaging techniques include the ability to reformat axial images into multiple planes to create complex 3-dimensional (3D) modeling. Surface rendering techniques have also helped to better define subtle osseous abnormalities at the proximal femur and acetabulum [1]. In addition to its clinical usefulness, the lower cost, relatively short scanning times, and new lower radiation dose scanning techniques make CT an ideal modality for imaging the hip and pelvis.

Scanning Protocol

As summarized in Table 1, there are several parameters that can be considered in the CT evaluation of patients. These parameters are useful to better define the osseous anatomy as well as diagnose specific disease pathology. The parameters will be briefly listed by anatomy, followed by a more detailed discussion in the proceeding sections organized by disease pathology. In the femur, parameters assessed include the cam location (clockface), alpha angle (swiss axial or radial planes), neck-shaft angle, and femoral version angle. In the acetabulum, parameters assessed include the coronal center-edge angle, sagittal center-edge angle, acetabular version (at 1 o'clock, 2 o'clock, and 3 o'clock), anterior inferior iliac spine (AIIS) width, distance from the distal base of AIIS to acetabular rim, and subspine morphology (type I, II, or III). The combined femoral and acetabular version is referred to as the McKibbin index. This index is important for assessing constrainment and instability and will be addressed later in this chapter. In certain cases, if rotational deformity is suspected, then spot scans of the proximal tibia are done for tibial version. Tibial version can be evaluated using the bimalleolar method as described by Goutallier et al. [2]. In this method, the version is measured as an angle between a line along the posterior contour of the proximal tibia and the intermalleolar axis (Fig. 1). At the author's institution, multidetector helical CT scans are obtained through the hip utilizing 0.625 mm slice thicknesses. Sagittal and coronal reformatted images are obtained in addition to 3D volume-rendered images. Axial images can be obtained through the knee and distal tibia for version analysis.

Table 1 Variables to measure with a CT scan for proper preoperative planning of a patient with FAI

Variable	Normal values	Abnormal values
CAM location		Lesion between 12 and 4 o'clock
Alpha angle		>42°
Coronal center-edge angle	41°	<20°
Sagittal center-edge angle	31°	<20°
Neck-shaft angle	126–139°	Coxa vara <126° Coxa valga >139°
Acetabular version 1 o'clock	5°	
Acetabular version 2 o'clock	10°	
Acetabular version 3 o'clock	15°	>30°
Femoral version angle	15–20°	>30°
McKibbin index	30–60°	>60 or <30°
AIIS width	1 cm	
Distal base of AIIS to acetabular rim	≥0.5 cm	<0.5 cm

Radiation Exposure

Radiation dose is measured in units of rem or sievert (Sv), where 100 rem is equivalent to 1 Sv. A single chest radiograph exposes the patient to approximately 0.02 mSv, while a CT scan of the pelvis exposes the patient to approximately 3–4 mSv [3]. This amount of exposure is significant in light of prior evidence demonstrating an increase in cancer risk with radiation doses in excess of 50 mSv [4]. New image reconstruction techniques, such as adaptive statistical iterative reconstruction (ASiR), have made it possible to reduce the radiation dose to patients up to 50 % [5, 6]. Several dose reduction strategies exist to

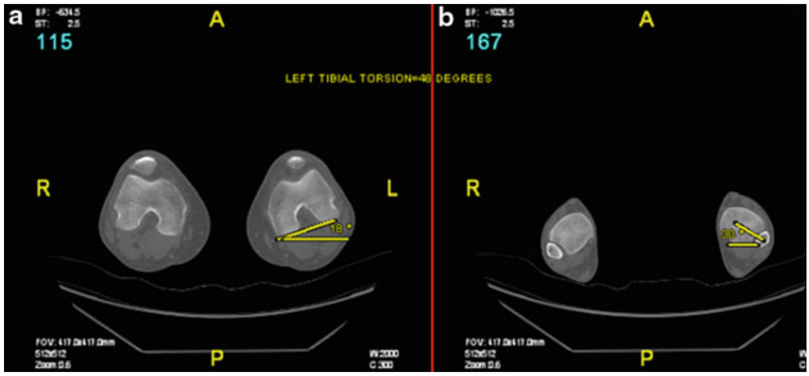


Fig. 1 Tibial version is measured by finding the angle between the line along the posterior contour of the proximal tibia (**a**) and the intermalleolar axis (**b**). Normal tibial version angle ranges between 18° and 47° in skeletally mature adults

achieve diagnostic image quality while minimizing patient radiation exposure. For routine contrast-enhanced CT of the pelvis, the CT protocol can be tailored based on patient size and clinical indications by modifying the maximum tube current (mA) and noise index (NI), which can generally be increased with increasing patient size [7]. The lower tube potential (kV) can also be adjusted based on patient size [8]. ASiR has demonstrated not only significant radiation reduction but also improved image quality in comparison with traditional filtered back-projection techniques [7, 8]. CT protocols can be customized in order to lower radiation doses particularly for pregnant or pediatric patients without compromising image quality.

Pathological Entities

Femoroacetabular Impingement (FAI)

Cam, pincer, and mixed types of impingement can occur between the proximal femur and the acetabulum. Cam-type FAI occurs when a nonspherical femoral head leads to an abnormal contour of the head-neck junction. This morphologic abnormality can impinge with the acetabulum and labrum when the hip is placed in positions of flexion, adduction, and internal rotation. Cam-type impingement may result from a variety of causes including distortion of the physis during growth, Legg-Calvé-Perthes syndrome, previous trauma, or slipped capital femoral epiphysis.

Preoperative planning with a 3D CT protocol can help tailor the operative approach (Table 1). Cam lesion location is commonly defined by using clockface terminology. Typically, 12 o'clock represents the most superolateral aspect, 3 o'clock represents the most anterior aspect, and 6 o'clock represents the most inferomedial aspect of the femoral head-neck junction. The cam is most commonly found between 12 o'clock and 4 o'clock [9]. Cam lesions that are beyond 12 o'clock (i.e., 11 o'clock) toward the posterior aspect of the hip are difficult to address arthroscopically and may be more amenable to an open procedure.

An objective method to quantify the size of the cam lesion is the alpha angle. The alpha angle is measured at the maximal offset where the abnormal bony contour of the femoral head-neck junction extends beyond the spherical confines of the femoral head. Using CT, the alpha angle can be measured on multiple planes. The measurement is an angle from the axis of the femoral neck to the point where the head-neck junction loses its spherical contour [10] (Fig. 2). An alpha angle greater than 42° is considered abnormal [10]. In a retrospective, comparative study, Hetsroni et al. found that the alpha angles for men and women presenting with hip pain and labral tears were $63.6 \pm 10.8°$ and $47.8 \pm 9.5°$, respectively [11].

The neck-shaft angle (NSA) is measured on the coronal view. This NSA angle is the angle subtended by the femoral neck and diaphyseal axes (Fig. 3). The neck axis is defined by the line connecting the center of the femoral head and the center of the femoral neck at its isthmus. The diaphyseal axis is defined by the line connecting the midpoint of two lines drawn

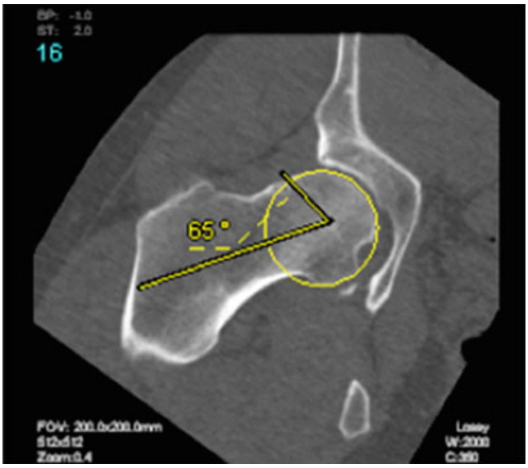


Fig. 2 Coronal oblique view alpha angle determined by measuring the angle subtended between a line along the axis of the femoral neck and the point at which the femoral head loses its sphericity at its maximal deformity. The alpha angle and cam location in this case are 65° and between 1 and 3 o'clock, respectively. Note that the alpha angle can be measured from the coronal, sagittal, and radial views. An angle > 42° is considered to be abnormal

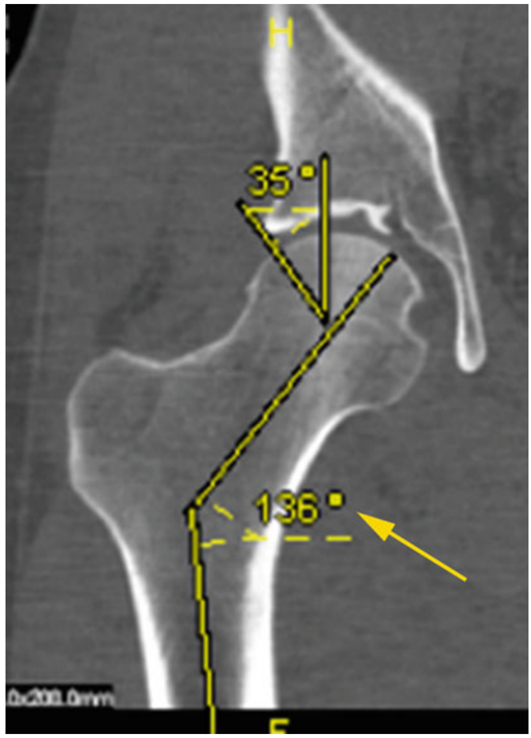


Fig. 3 Neck-shaft angle (*arrow*) is normally between 126° and 139°

perpendicularly across the diaphysis of the femur. A normal NSA is between 126° and 139°. Coxa vara is defined as a NSA <126° and is more consistent with impingement. Coxa valga is defined as NSA >139° and is more consistent with instability and dysplasia [12].

Pincer-type FAI occurs when the abnormal morphology is in the acetabulum. Several possible causes of abnormal acetabular morphology include coxa profunda, protrusio, rim prominence, and retroversion. Acetabular version angle is formed by the intersection of the line perpendicular to the line between the posterior pelvic margins and the line connecting the anterior and posterior rim of the acetabulum (Fig. 4) [13]. This measurement is conducted at 1 o'clock, 2 o'clock, and 3 o'clock with normal being approximately 5°, 10°, and 15°, respectively. Patients with retroverted acetabulums are associated with pincer-type FAI.

Subspine Impingement

Subspine impingement occurs when there is direct impingement of the inferior femoral neck along with the labrum, capsule, and indirect head of the rectus femoris against the anterior inferior iliac spine (AIIS). This type of impingement occurs in positions of straight hip flexion. AIIS morphology can be typed and subtyped based on the direction of slope of the AIIS as well as the presence or absence of a clear subspine space (Figs. 5 and 6) (Tables 2 and 3). AIIS type I has an upsloping morphology, which does not contribute to impingement. AIIS type III has a downsloping (crossing the rim) morphology that is more likely to cause impingement. AIIS subtype A has a clear subspine space, which does not contribute to impingement. However, AIIS subtype B has a subspine bone prominence or rim level-based AIIS that is more likely to cause impingement. Therefore, AIIS types II and III and subtype B have a higher predilection for subspine impingement [14]. The AIIS is also assessed to determine the degree of subspine impingement. The distance from the distal base of the AIIS to

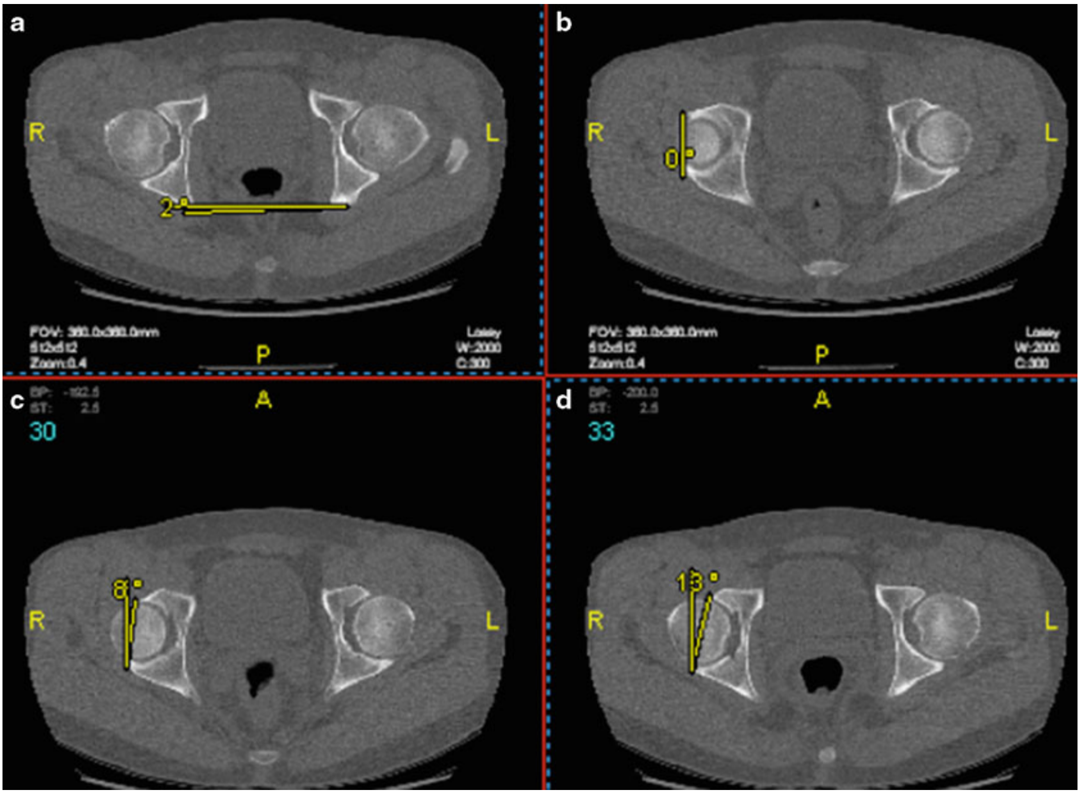


Fig. 4 Acetabular version. (**a**) The angle created from the line connecting the posterior pelvic margin and horizontal line of the image screen represents the amount of correction (2°) that will need to be added to subsequent measurements in B, C, and D. (**b**) Acetabular version at 1 o'clock measuring 2° (0° + 2° of correction). (**c**) Acetabular version at 2 o'clock measuring 10° (8° + 2° of correction). (**d**) Acetabular version at 3 o'clock measuring 15° (13° + 2° of correction). Acetabular version angle is normally 5°, 10°, and 15° at 1 o'clock, 2 o'clock, and 3 o'clock, respectively

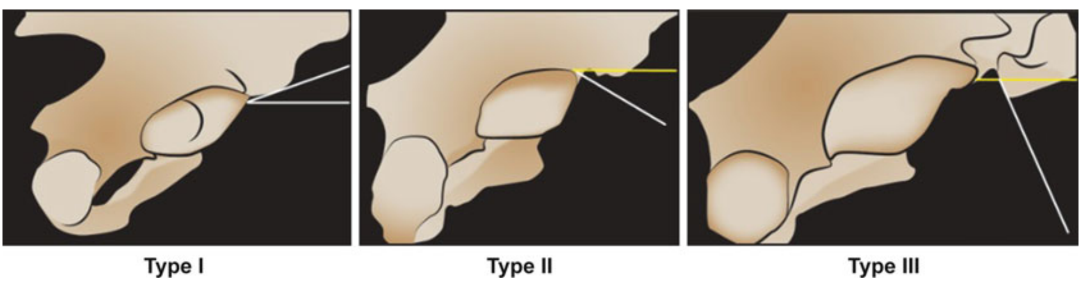


Fig. 5 AIIS morphology types. Type I with an upsloping AIIS morphology. Type II with a flat or downsloping AIIS morphology. Type III with a significantly downsloping AIIS morphology

the acetabular rim should be ≥0.5 cm. A measurement of <0.5 cm is consistent with subspine impingement. The width of the AIIS should be about 1 cm.

Acetabular Dysplasia

Acetabular dysplasia is defined as an abnormality with the version, volume, or inclination of the

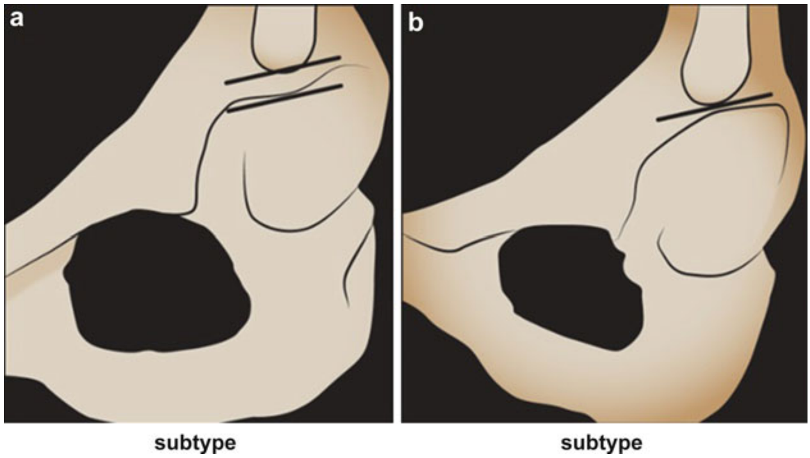


Fig. 6 AIIS morphology subtypes. Subtype A with a subspine clear space. Subtype B with a subspine bone prominence or rim level-based AIIS

Table 2 AIIS morphology

Type	Description	CT definitions	Clinical importance
I	Upsloping	Upsloping on Ischium view	AIIS does not contribute to impingement
II	Flat to downsloping	Flat to downsloping on ischium view, but does not cross the rim	AIIS may contribute to impingement
III	Significant downsloping	Downsloping and crosses the rim	AIIS may contribute to impingement

Table 3 AIIS Subtype Morphology

Subtype	Description	CT Definitions	Clinical Importance
A	Clear subspine space	No secondary extension to rim	AIIS does not contribute to impingement
B	Subspine bone prominence, or rim level-based AIIS	Caudal extension of AIIS on ilium wall to acetabular rim level	AIIS may contribute to impingement

acetabulum [15]. The morphologic deformity places a greater concentration of weight-bearing forces on a smaller area of articular cartilage, resulting in degenerative soft tissue injuries and osteoarthritic symptoms. Patients with subtle dysplasia are difficult to identify, particularly as a spectrum of disease involve both dysplasia and FAI, which often have overlapping symptoms [16]. Morphologically, acetabular dysplasia is one of the most complex 3D deformities. CT reconstruction circumvents the limitations of traditional 2D radiographic screening, allowing for more accurate estimations of the severity of dysplasia. Measurements such as center-edge angle (CEA), acetabular index (AI), and acetabular sourcil angles can be used to evaluate for dysplasia.

The CEA corresponds to the degree of coverage of the femoral head by the acetabulum. A higher CEA indicates a greater degree of coverage, whereas a lower CEA indicates a lesser degree of coverage. The center-edge angle can be measured on the sagittal or coronal views. The specific cuts chosen to measure these angles are through the center of the femoral head. The coronal CEA is measured by first drawing a line through the center of the femoral head perpendicular to the transverse pelvic axis. Next, a line is drawn through the center of the femoral head to the superolateral aspect of the acetabular roof. The angle between these lines defines the coronal CEA (Fig. 7a). This corresponds to the lateral CEA on radiographs [17], where a CEA $<20°$ is considered to be dysplastic [18]. The average CEA is approximately 41° [19]. The sagittal CEA is measured by first drawing a vertical line through the center of the femoral head. Next, a line is drawn through the center of the femoral head to the most

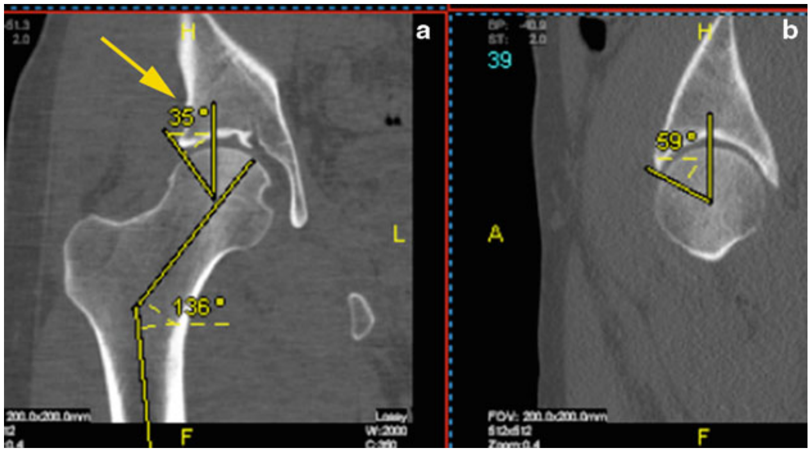


Fig. 7 Coronal center-edge angle (*arrow*) (**a**) and sagittal center-edge angle (**b**). A center-edge angle <20° is considered to be abnormal

anterior aspect of the acetabular rim. The angle between these lines defines the sagittal CEA (Fig. 7b). This corresponds to the anterior CEA on radiographs where an anterior CEA <20° is considered to be dysplastic [20]. The average anterior CEA angle is approximately 31° [19]. In patients with a CEA <20°, a procedure such as a periacetabular osteotomy (PAO) may be indicated.

The AI is represented by the angle between the pelvic horizontal line and the line through the lateral margin of the acetabulum and the superior edge of the fovea [21]. The AI is typically between 4° and 10°. Axial CT images can provide analysis of anterior, posterior, and global acetabular deficiencies by measuring the anterior, posterior, and horizontal acetabular sector angle (AASA, PASA, HASA) by using the axial CT slice located one cut above the greater trochanter (Fig. 8) [22]. AASA is measured by drawing a lines through the center of the femoral head and contralateral femoral head and tangential to the anterior lip of the acetabulum [23]. Anterior acetabular coverage ≤ 50° is indicative of dysplasia. PASA is measured by lines drawn through the center of the femoral head and contralateral femoral head and tangential to the posterior lip of the acetabulum. Abnormal posterior acetabular coverage is considered to be when the posterior acetabular sector angle is ≤ 90°. HASA is measured by lines drawn from the anterior lip of the acetabulum through the center of the femoral head and the posterior lip of the acetabulum. Angles ≤140° indicate inadequate global acetabular coverage [24].

Although instability/dysplasia can be diagnosed by radiographic findings, CT scans can be used to help objectify predictors for this condition. Measurements that can be used include acetabular version angle, femoral version angle, as well as the McKibbin index. Acetabular version angle is measured using the method previously described. Larger acetabular anteversion angles >30° can predispose the patient to instability. Femoral version angle is measured by first taking limited CT cuts from the femoral head and neck down to the distal femur. The femoral head center image is then superimposed on the transcondylar knee image. The femoral version angle is the angle subtended between the femoral head and neck axis and the posterior condylar angle (Fig. 9) [25, 26]. Normal femoral version angle ranges between 15° and 20° of anteversion [25, 26]. Femoral anteversion >30° is consistent with instability [27]. The femoral version angle is also important in determining the starting point of the anterolateral portal in hip arthroscopic procedures. More femoral retroversion should dictate a more anterior starting point for the anterolateral portal to avoid crowding with the greater trochanter.

The McKibbin index is the sum of the femoral version angle and the acetabular version angle measured at 3 o'clock. A normal McKibbin index ranges between 30° and 60°. A McKibbin

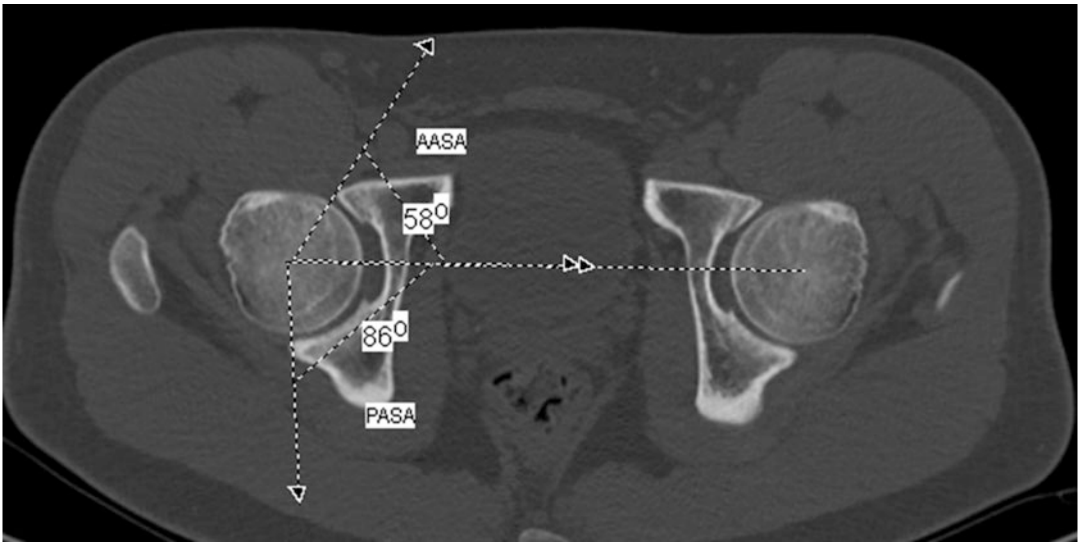


Fig. 8 Anterior acetabular sector angle (AASA) is measured by drawing lines through the center of the femoral head and contralateral femoral head and tangential to the anterior lip of the acetabulum. Anterior acetabular coverage ≤ 50° is indicative of dysplasia. Posterior acetabular sector angle (PASA) is measured by lines drawn through the center of the femoral head and contralateral femoral head and tangential to the posterior lip of the acetabulum. Abnormal posterior acetabular coverage is considered to be when the posterior acetabular sector angle is ≤ 90°. Horizontal acetabular sector angle (HASA) is measured by lines drawn from the anterior lip of the acetabulum through the center of the femoral head and the posterior lip of the acetabulum. Angles ≤ 140° indicate inadequate global acetabular coverage

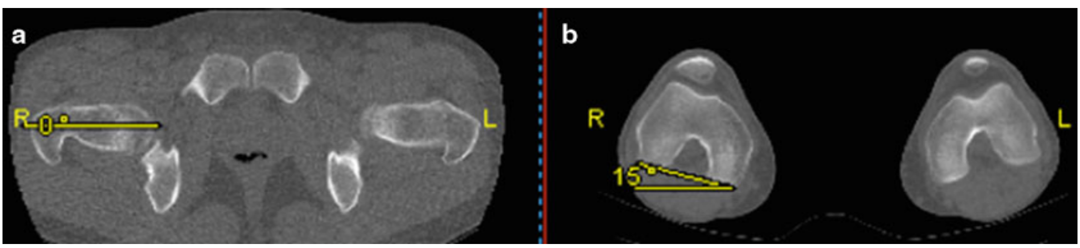


Fig. 9 Femoral version is measured by finding the angle between the femoral head and neck axis (**a**) and the posterior condylar angle (**b**). Normal femoral version angle ranges between 15° and 20° of anteversion

index >60° predisposes the hip to instability, while a McKibbin index <30° predisposes the hip to impingement [12]. Patients with a McKibbin index between 15° and 30° can be treated arthroscopically, while lower ranges (<15°) are amenable to an open procedure to derotate the femur [12, 28, 29]. Patients with a mid to high McKibbin index (45–60°) can be treated arthroscopically with minimal capsular cuts. Patients with a McKibbin index greater than 60° can be treated with a derotational osteotomy and/or periacetabular osteotomy (PAO) [12, 28, 29].

Pediatric Disorders (DDH, Perthes, SCFE)/Malalignment Syndromes

Common pediatric hip pathologies include developmental dysplasia of the hip (DDH), Legg-Calvé-Perthes (LCP) disease, and slipped capital femoral epiphysis (SCFE). While radiographic imaging remains the primary imaging modality used for diagnosis, CT imaging has proven useful in the workup of these conditions and during pre- and postoperative planning/management [30]. DDH is the underdevelopment of the acetabulum with unknown etiology. CT scans are

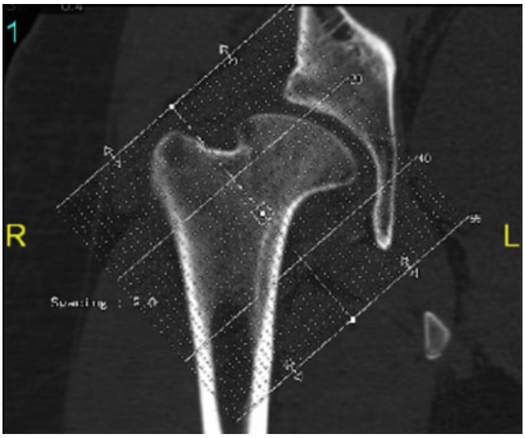


Fig. 10 Perthes of the right hip as demonstrated on a coronal CT scan

predominantly used to assess post-procedural outcome when assessing spica cast placement. CT imaging can help evaluate hip joint congruity in axial and coronal planes, acetabular morphology, and possible hardware complications [31]. LCP is characterized by idiopathic avascular necrosis of the epiphysis, impairing the endochondral ossification of the femoral head (Fig. 10) [32]. CT scans can allow early detection of bone collapse, sclerotic zones, and subtle changes in the bone trabecular pattern as well as intraosseous cysts in later stages of the disease [33]. SCFE is a type 1 fracture through the proximal femur physis causing relative posteromedial displacement of the epiphysis with respect to the metaphysis [34]. SCFE can lead to significant impingement lesions and symptoms (Fig. 11). Axial and sagittal oblique planes, parallel to the long axis of the femoral neck as seen on coronal images, are most helpful in defining the extent of posterior slip [35]. 3D CT scanning can be helpful diagnostically and in presurgical planning. Anterior knee pain and instability are symptoms of patellofemoral dysplasia, often referred to as miserable malalignment syndrome (MMS). MMS is characterized by a triad of anatomic abnormalities, which are excessive femoral anteversion, increased knee Q angle, and external tibial torsion [36]. CT scans can be used to accurately determine tibial torsion by scanning the tibial transmalleolar axis as well as femoral anteversion [37].

Fig. 11 SCFE of the left femoral head as seen on a coronal CT scan

Traumatic Hip Instability/Acetabular and Avulsion Fracture

Traumatic hip instability is characterized by hip dislocation or subluxation sustained from an acute event or series of repetitive events, most often stemming from a motor vehicle accident or sports injury. Hip dislocation can cause damage to osseous and/or soft tissue hip structures causing recurrent instability [38]. The spectrum of injury can range from dislocation or subluxation with or without concomitant injuries such as acetabular fractures or intra-articular injuries. CT scanning can be particularly useful when evaluating traumatic hip instability as it is more sensitive in detecting small, non-displaced acetabular fractures than radiographs. This is especially important as the characteristic triad of posterior hip subluxation includes posterior acetabular hip fractures in addition to hemarthrosis and ischiofemoral ligament disruption [39]. In the setting of traumatic hip injury and hip dislocations, CT is valuable due to its ability to detect osteochondral fragments, intra-articular loose bodies, and residual subluxation; to assess the femoral head and adequacy of reduction; and to detect cam lesions anteriorly that may cause the "lever effect." This can be done using 3 mm cuts through the hip [40].

Total Hip Arthroplasty

Although radiography is the standard method of evaluation of hip arthroplasty patients, CT does have a role in more complicated situations. For example, CT scans can be used to assess for component malposition, component wear, periprosthetic fracture, and infection [41, 42]. Although radiographs can be used to assess for osteolysis, CT scans can be used to measure the volume of osteolysis [43, 44]. CT scans also provide a way to image the surrounding soft tissues, which can be obscured with an MRI due to metal artifact.

Summary

CT scans are an effective tool in evaluating osseous lesions of the hip. FAI, dysplasia, pediatric disorders, and traumatic hip instability can be objectified with the variables described above. These measurements are useful in the preoperative planning stage of patient care. Continued advancements in 3D rendering of CT scans can further improve their usefulness in addressing hip pathology and ultimately help dictate care. New scanning protocols help to lower radiation exposure.

References

1. Kang RW, Yanke AB, Espinoza Orias AA, Inoue N, Nho SJ. Emerging ideas: novel 3-D quantification and classification of cam lesions in patients with femoroacetabular impingement. Clin Orthop Relat Res. 2013;471(2):358–62.
2. Goutallier D, Van Driessche S, Manicom O, Sariali E, Bernageau J, Radier C. Influence of lower-limb torsion on long-term outcomes of tibial valgus osteotomy for medial compartment knee osteoarthritis. J Bone Joint Surg Am. 2006;88:2439–47.
3. Flohr TG, Schaller S, Stierstorfer K, et al. Multi-detector row CT systems and image-reconstruction techniques. Radiology. 2005;235:756.
4. Amis Jr ES, Butler PF, Applegate KE, et al. American College of Radiology white paper on radiation dose in medicine. J Am Coll Radiol. 2007;4:272.
5. Gay F, Pavia Y, Pierrat N, Lasalle S, Neuenschwander S, Brisse HJ. Dose reduction with adaptive statistical iterative reconstruction for paediatric CT: phantom study and clinical experience on chest and abdomen CT. Eur Radiol. 2013;24(1):102–11.
6. Gervaise A, Osemont B, Louis M, Lecocq S, Teixeira P, Blum A. Standard dose versus low-dose abdominal and pelvic CT: comparison between filtered back projection versus adaptive iterative dose reduction 3D. Diagn Interv Imaging. 2013;95(1):47–53.
7. Goldman AR, Maldjian PD. Reducing radiation dose in body CT: a practical approach to optimizing CT protocols. Am J Roentgenol. 2013;200(4):748–54.
8. McCollough CH, Primak AN, Braun N, Kofler J, Yu L, Christner J. Strategies for reducing radiation dose in CT. Radiol Clin North Am. 2009;47(10):27–40.
9. Bedi A, Dolan M, Magennis E, Lipman J, Buly R, Kelly BT. Computer-assisted modeling of osseous impingement and resection in femoroacetabular impingement. Arthroscopy. 2012;28(2):204–10.
10. Notzli HP, Wyss TF, Stoecklin CH, Schmid MR, Treiber K, Hodler J. The contour of the femoral head-neck junction as a predictor for the risk of anterior impingement. J Bone Joint Surg Br. 2002;84:556–60.
11. Hetsroni I, Dela Torre K, Duke G, Lyman S, Kelly BT. Sex differences of hip morphology in young adults with hip pain and labral tears. Arthroscopy. 2013;29(1):54–63.
12. Tonnis D, Heinecke A. Acetabular and femoral anteversion: relationship with osteoarthritis of the hip. J Bone Joint Surg Am. 1999;81:1747–70.
13. Jacquemier M, Jouve JL, Bollini G, Panuel M, Migliani R. Acetabular anteversion in children. J Pediatr Orthop. 1992;12:373–5.
14. Hetsroni I, Poultsides L, Bedi A, Larson CM, Kelly BT. Anterior inferior iliac spine morphology correlates with hip range of motion: a classification system and dynamic model. Clin Orthop Relat Res. 2013;471(8):2497–503.
15. Stubbs AJ, Anz AW, Frino J, Lang JE, Weaver AA, Stitzel JD. Classic measures of hip dysplasia do not correlate with three-dimensional computer tomographic measures and indices. Hip Int. 2011;21(5):549–58.
16. Ranawat AS, Schulz B, Baumbach SF, Meftah M, Ganz R, Leunig M. Radiographic predictors of hip pain in femoroacetabular impingement. HSS J. 2011;7(2):115–9.
17. Monazzam S, Bomar JD, Cidambi K, et al. Lateral center-edge angle on conventional radiography and computed tomography. Clin Orthop Relat Res. 2013;471(7):2233–7.
18. Wiberg G. Studies on dysplastic acetabula and congenital subluxation of the hip joint: with special reference to the complication of osteoarthritis. Acta Chir Scand Suppl. 1939;83(58):1–135.
19. Tallroth K, Lepistö J. Computed tomography measurement of acetabular dimensions: normal values for correction of dysplasia. Acta Orthop. 2006;77(4):598–602.

20. Crockarell JR, Trousdale RT, Guyton JL. The anterior centre-edge angle: a cadaver study. J Bone Joint Surg Br. 2000;82-B:532–4.
21. Ito H, Matsuno T, Hirayama T, Tanino H, Yamanaka Y, Minami A. Three-dimensional computed tomography analysis of non-osteoarthritic adult acetabular dysplasia. Skeletal Radiol. 2009;38(2):131–9.
22. Haddad FS, Garbuz DS, Duncan CP, Janzen DL, Munk PL. CT evaluation of periacetabular osteotomies. J Bone Joint Surg Br. 2000;82:526–31.
23. Beltran LS, Rosenberg ZS, Mayo JD, Diaz de Tuesta M, Martin O, Neto LP, Bencardino JT. Imaging evaluation of developmental hip dysplasia in the young adult. AJR. 2013;200:1077–88.
24. Anda S, Svenningsen S, Dale LG, Benum P. The acetabular sector angle of the adult hip determined by computed tomography. Acta Radiol Diagn (Stockh). 1986;27:443–7.
25. Murphy SB, Simon SR, Kijewski PK, Wilkinson RH, Griscom NT. Femoral anteversion. J Bone Joint Surg Am. 1987;69:1169–76.
26. Weiner DS, Cook AJ, Hoyt Jr WA, Oravec CE. Computed tomography in the measurement of femoral anteversion. Orthopedics. 1978;1:299–306.
27. Grissom L, Harcke HT, Thacker M. Imaging in the surgical management of developmental dislocation of the hip. Clin Orthop Relat Res. 2008;466:791–801.
28. Ranawat CS, Maynard MJ. Modern techniques of cemented total hip arthroplasty. Tech Orthopedics. 1991;6:17–25.
29. Zaltz I, Kelly BT, Larson CM, et al. Surgical treatment of femoroacetabular impingement: what are the limits of hip arthroscopy. Arthroscopy. 2014;30(1):99–110.
30. Schlesinger AE, Hernandez RJ. Diseases of the musculoskeletal system in children: imaging with CT, sonography and MR. AJR. 1992;158:729–41.
31. Atweh LA, Kan JH. Multimodality imaging of developmental dysplasia of the hip. Pediatr Radiol. 2013;43 (1):S166–71.
32. Nelitz M, Lipppacher S, Krauspe R, Reichel H. Perthes disease- current principles of diagnosis and treatment. Dtsch Arztebl Int. 2009;106(31–32):517–23.
33. Dimeglio A, Canavese F. Imaging in Legg-Calvé-perthes disease. Orthop Clin North Am. 2011;42:297–302.
34. Gill KG. Pediatric hip: pearls and pitfalls. Semin Musculoskeletal Radiol. 2013;17:328–38.
35. Jarrett DY, Matheney T, Kleinman PK. Imaging SCFE: diagnosis, treatment and complications. Pediatr Radiol. 2013;43(1):S71–82.
36. Bruce WD, Stevens PM. Surgical correction of miserable malalignment syndrome. J Pediatr Orthop. 2004;24(4):392–6.
37. Eckhoff DG, Johnson KK. Three-dimensional computed tomography reconstruction of tibial torsion. Clin Orthop. 1997;339:152–5.
38. Boykin RE, Anz AW, Bushnell BD, Kocher MS, Stubbs AJ, Philippon MJ. Hip instability. J Am Acad Orthop Surg. 2011;19(6):340–9.
39. Moorman 3rd CT, Warren RF, Hershman EB, Crowe JF, Potter HG, Barnes R, O'Brien SJ, Guettler JH. Traumatic posterior hip subluxation in American football. J Bone Joint Surg Am. 2003;85(A7):1190–6.
40. Shindle MK, Ranawat AS, Kelly BT. Diagnosis and management of traumatic and atraumatic hip instability in the athletic patient. Clin Sports Med. 2006;25:309–26.
41. Wines AP, McNicol D. Computed tomography measurement of the accuracy of component version in total hip arthroplasty. J Arthroplasty. 2006;21:696.
42. van Holsbeeck MT, Eyler WR, Sherman LS, et al. Detection of infection in loosened hip prostheses: efficacy of sonography. AJR Am J Roentgenol. 1994;163:381.
43. Kitamura N, Pappedemos PC, Duffy 3rd PR, et al. The value of anteroposterior pelvic radiographs for evaluating pelvic osteolysis. Clin Orthop Relat Res. 2006;453:239.
44. Park JS, Ryu KN, Hong HP, et al. Focal osteolysis in total hip replacement: CT findings. Skeletal Radiol. 2004;33:632.

Magnetic Resonance Imaging of the Hip

5

Thomas W. Hash II

Contents

T.W. Hash II
Musculoskeletal Section, Department of Radiology,
Duke University Medical Center, Durham, NC, USA
e-mail: tom.hash@dm.duke.edu

Abstract

MRI provides an excellent noninvasive means of assessing pathology of the central and peripheral compartments and peritrochanteric space. It also provides accurate assessment of conditions about the hip, including hamstring tendon pathology and ischiofemoral impingement. It provides accurate assessment of athletic pubalgia, frequently coexisting with femoral acetabular impingement (FAI). Patient management can be driven or altered by MRI findings.

The hip's unique anatomy does pose challenges to MRI, particularly accurate imaging of the central compartment. Awareness of the limitations and pitfalls of hip MR imaging allows for more sensitive, specific, and accurate diagnoses.

Introduction

MR imaging of the hip is difficult due to its deep location, hindering optimal contrast and spatial resolution and magnetic field homogeneity. In other words, it is more difficult to obtain optimal resolution of the central compartment structures, particularly the cartilage than it is for the more superficially located knee, for example. Unlike other joints, there is no dedicated imaging coil for the hip. Furthermore, the closely applied, curved articular surfaces of the dome and head, covered by relatively thin cartilage, are

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_5

significantly more susceptible to volume averaging. Volume averaging occurs when more than one structure is included in the same voxel (i.e., location) encoded with signal (i.e., information); this blurred comingling of adjacent, different structures can degrade accurate assessment. As the curved articular surfaces of the dome and head do not fall neatly into the usual imaging planes, volume averaging occurs in all quadrants. Due to the current limitations of MR magnets and coils, this is unavoidable as extremely thin slices cannot be obtained without the loss of resolution.

Like other joints, certain imaging sequences are recommended for hip imaging. A T1-weighted sequence in at least one plane is recommended, mainly to evaluate for marrow pathology and muscle quality. On T1-weighted sequences, fat (e.g., subcutaneous fat, fatty marrow) is high/bright in signal, normal muscle is intermediate (in between bright and dark) in signal, and normal synovial fluid is low/dark in signal. High T1 signal in muscle usually indicates fatty atrophy and less frequently acute blood products after acute trauma. Low T1 signal in muscle can occur due to mineralization or older blood products. Fatty marrow has high T1 signal; hematopoietic marrow, often heterogeneous, is slightly higher in signal than normal muscle. Marrow replacement or infiltrative processes (e.g., metastatic disease) have T1 signal that is lower, or sometimes equivalent to, normal muscle.

In addition, at least one plane of imaging of the hip needs to be a fluid-sensitive sequence. Fluid-sensitive sequences, referred to throughout the chapter, include proton density- and T2-weighted sequences as well as short tau inversion recovery (STIR) sequences. On these sequences, normal fat-containing structures are low/dark in signal, muscle is intermediate in signal, and fluid is high/bright in signal. Fluid-sensitive sequences thus are sensitive in detecting marrow edema (e.g., stress fracture), muscle and other soft tissue edema, paralabral cysts, and bursal distention. The conspicuity of fluid or edema is increased by the use of spectral fat suppression on proton density-weighted and T2-weighted sequences due to the fact that the high signal is seen more easily with darkening the signal of fat-containing structures, particularly the marrow.

For accurate MR imaging of the hip, it is necessary to obtain images with substantial contrast resolution, spatial resolution, a small field of imaging, and a high signal-to-noise ratio. Traditionally, in the evaluation of the central compartment structures, MRI has been performed after arthrography where the joint is distended with a solution containing a small amount of gadolinium. The widely held benefit of intra-articular contrast distention, like in the shoulder, is improved detection of labral tears and chondral defects or delamination due to pressure-related flow of the solution into defects that may be intimately coapted so as not to allow the normal intravasation of joint fluid.

However, the author's experience and others suggests otherwise [1–4]. Accurate detection of labral tears and chondral defects can be made without the use of intra-articular gadolinium. Given the higher signal-to-noise ratios obtained with higher-strength magnets, imaging on a 3 Tesla (i.e., 3 T) magnet is strongly preferred over a 1.5 T magnet. Multichannel surface coils (instead of body coils) are recommended in dedicated imaging of the hip for improved signal detection. Femoral version measurement is obtained with fast low-resolution imaging of the proximal femora and knees. Additionally, three-dimensional (3D) reformats with volume rendering for morphologic evaluation of the acetabular rim and proximal femur are obtained with a volumetric interpolated breath-hold (VIBE) sequence (Fig. 1). An example of an imaging protocol (40- to 45-min total exam time) used at the author's institution is detailed in Table 1.

While high-resolution imaging is certainly preferred in the evaluation of the central compartment, patients may be imaged in a more limited fashion. Thus, scattered throughout the chapter, images will detail how pathology can be detected without the use of high-resolution imaging. Additionally, central compartment pathology will be shown with and without the use of intra-articular gadolinium.

When referencing the signal characteristics of structures, they will be in reference to normal muscle signal, whether there is fat suppression or not. Thus, something referred to as low signal means that it is lower in signal than normal muscle. Something referred to as intermediate signal has the same signal as muscle. And something that is high in signal is higher in signal than normal muscle.

Locations around joint will be referenced to the clockface, whether the right or left hip: 3 o'clock is directly anterior; 12 o'clock is superior; 9 o'clock is posterior; 6 o'clock is directly caudal/distal.

While not a part of the hip, the end of the chapter will discuss imaging of hamstring tendon pathology, athletic pubalgia, and ischiofemoral impingement given the propensity of these conditions to mimic and/or coexist with hip pathology.

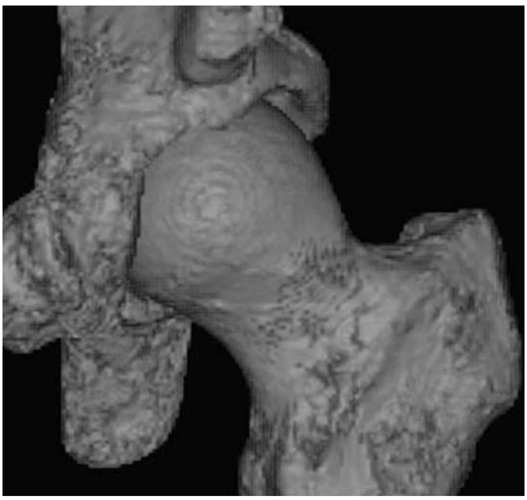


Fig. 1 3D volume-rendered reformation of the dome and proximal femur with rotation axis in the coronal plane obtained from the VIBE sequence

FAI Hip Pathomorphology

Cam femoral acetabular impingement (FAI) morphology is visualized well with MRI. The presence of a cystic or multicystic intraosseous ganglion, or focal fibrocystic change, is frequently seen on MRI at the site of head-neck asphericity. Oblique axial images, obtained parallel to the long axis of the femoral neck, have traditionally been obtained to evaluate proximal femur pathomorphology. The asphericity of the head-neck junction in cam FAI is quantified with the alpha angle, as can be done with radiographs or computed tomography (CT). A best-fit circle is obtained of the femoral head, and a line bisecting the neck is extended to the center of the circle; the angle formed between this line and the point where the aspherical head-neck junction departs from the circle is the alpha angle [5]. An abnormal alpha angle, signifying pathomorphology, is defined by various authors as greater than 42–60°, although angles greater than 65–74° were found in patients with anterior impingement in two case–control studies [6–10]. Of note, the oblique axial images may be obtained incorrectly due to failure by the MRI technologist to obtain them parallel to the femoral neck (Fig. 2).

The intraobserver agreement of the alpha angle on MRI has been questioned [11]. Additionally, as

Table 1 Hip MRI sequences and parameters

Sequence	FOV	Matrix	ST/G	TR	TE	NEX	BW	TF
COR T1	18 × 18	320 × 224	3.5/0	600	10	2	200	3
OBL AX FS PD	18 × 18	512 × 384	3.5/0	3,600	32	2	200	8–10
COR FS PD	18 × 18	512 × 358	3.5/0	3,600	32	2	200	8–10
SAG FS PD	18 × 18	512 × 384	3.5/0	3,600	32	2	200	8–10
SAG VIBE	18 × 18	256 × 256	0.7/0	12.3	4.92	1	150	–
AX HASTE	38 × 38	320 × 240	5/1	800	85	1	601	–

Description: *OBL AX* oblique axial, *COR* coronal, *SAG* sagittal, *FS* fat-suppressed, *PD* proton density, *HASTE* half-Fourier acquisition single-shot turbo spin-echo, *FOV* field of view (in cm), *ST/G* slice thickness/interslice gap (in mm), *TR* repetition time, *TE* echo time, *NEX* number of excitations, *BW* bandwidth (in Hertz/pixel), *TF* turbo factor

Fig. 2 Fat-suppressed coronal proton density-weighted image shows *dashed lines*, reflecting orientation of the oblique axial images, not paralleling the long axis of the neck

the asphericity is typically anterosuperior in location, the angle obtained with the use of oblique axial images has been questioned as these images best quantify the asphericity at the direct anterior location (i.e., 3 o'clock). The use of radial imaging has been found to be more accurate in the assessment of the asphericity [12]. The use of both oblique axial and radial images may be moot if 3D reformats with volume rendering are performed for presurgical planning.

Pincer FAI morphology is usually better evaluated with radiographs than MRI. The lateral center-edge angle and Tönnis index are accurately evaluated with MRI; the anterior center-edge angle, however, is not [13]. The assessment of acetabular retroversion can be made with true axial images; the anterior wall will project lateral to the posterior wall on these images. Far cranial retroversion requires evaluating the last image or two on which the central compartment is visualized.

The Central Compartment

The Labrum

Before beginning discussion of the labrum, although widely known, it should be emphasized that labral pathology has a high prevalence in the asymptomatic population and is frequently seen on imaging done for reasons other than hip pain. For example, in an MRI study of 45 patients without hip pain, Register and colleagues found that 69 % of hips had labral tears [2].

Many studies have been performed evaluating the accuracy of detecting labral tears using MRI with and without contrast (gadolinium). The majority of studies have been performed with the use of joint distention with arthrography. One study examined the detection of tears with the use of intravenous gadolinium [14]. The sensitivities and specificities in tear detection with MR arthrography (MRA) range from 69–100 % and 0–100 %, respectively [15]. The sensitivities and specificities in tear detection with conventional MRI range from 0–97 % to 33–100 %, respectively [15]. The wide range of the sensitivity and specificity for both reflect a great heterogeneity of the studies, particularly the fewer conventional MRI studies. It is difficult to draw a definite conclusion as to whether MR arthrography (MRA) or conventional MRI in the evaluation of labral tears.

On MRI, the normal labrum is homogeneously low in signal. With aging, intrasubstance degeneration is common, manifested as intermediate to even high signal contained within the labrum. A triangular shape in all quadrants is far and away the most common shape. It occasionally thin and elongated and not infrequently somewhat rounded in shape with aging. Intralabral ossification is frequently seen, often reflecting pincer FAI (Fig. 3).

There has been one grading scheme proposed using MRI and one using arthroscopy [16, 17]. A study directly comparing the schema found no significant correlation between the two [18]. Different descriptive terms have been and are currently used to describe tears. It is most important

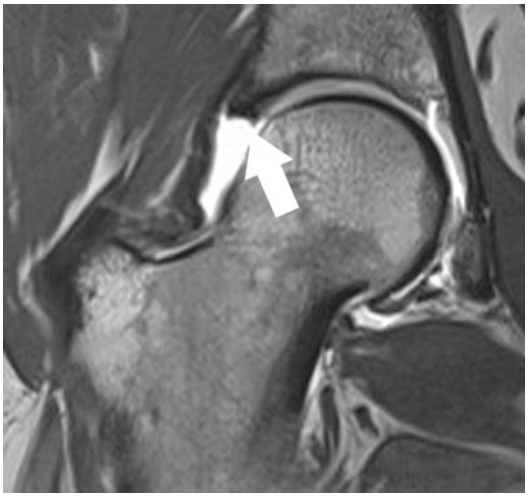


Fig. 3 Coronal T1-weighted post-arthrogram image shows subtotal ossification of the posterosuperior labrum with only a tiny residual degenerated fibrocartilaginous remnant (*arrow*)

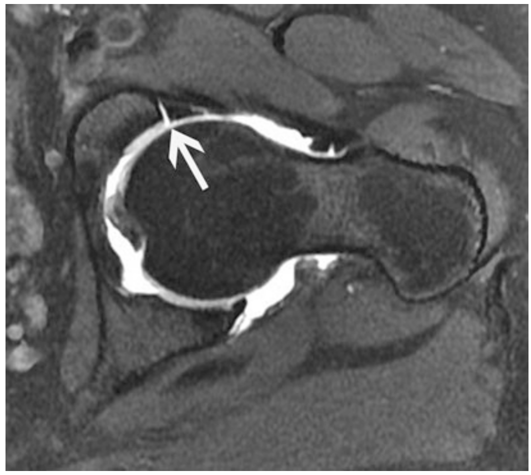


Fig. 4 Fat-suppressed oblique axial T1-weighted post-arthrogram image shows full-thickness linear fluid extension at the anterior chondrolabral junction (*arrow*)

for the radiologist and referring surgeon to use the same terminology.

On MRI, labral tears may be intermediate in signal. More often, tears are high in signal, similar to joint fluid (on both conventional MRI and MR arthrography (MRA)). Peripheral detachments (i.e., peripheral longitudinal tears) are seen when linear fluid or contrast insinuates between the labrum and rim at the chondrolabral junction (Fig. 4). Radial (or flap) tears are present when fluid or contrast extends into the substance of the joint side of the labrum, entering discretely away from the chondrolabral junction (Fig. 5). Sometimes multiple small radial tears are present, also called a radial fibrillated tear (Fig. 6). A complex tear is the preferred term when signal is branching and extending in multiple directions or multiple tear types coexist (Fig. 7). Occasionally, bucket-handle tears can be seen with frank displacement of a portion of the labrum (Fig. 8). Abnormal shape, particularly blunting, of the labrum is often a clue to a displaced flap. While the assessment of tear stability is very difficult on MRI without frank labral displacement, instability can be suggested if the labrum is thickened and distorted [18].

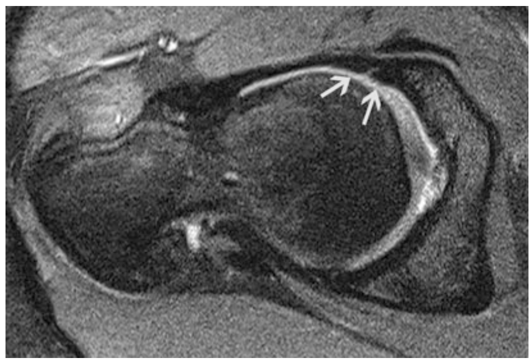


Fig. 5 Fat-suppressed oblique axial proton density-weighted image shows linear high signal separating the anterior labrum roughly in halves (*arrows*), a radial tear

Normal labral sulci, simulating peripheral detachments, are potential pitfalls on MRI. Sulci are normal variant clefts at the chondrolabral junction. They can be located at one position on the clockface (e.g., 8 o'clock) or extend over several positions (e.g., 8–9 o'clock). On MRI, they are well-defined linear areas of high signal located at the chondrolabral junction (Figs. 9 and 10). Importantly, they are partial-thickness clefts, distinguishing them from full-thickness peripheral detachments. There is no extension of the signal into the adjacent substance of the labrum. The adjacent labrum is usually normal in signal.

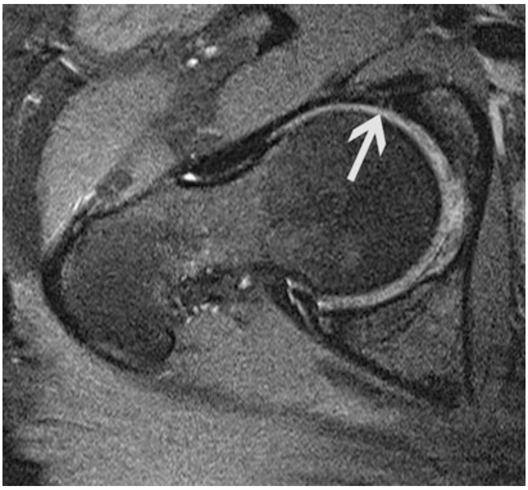


Fig. 6 Fat-suppressed oblique axial proton density-weighted image shows two small linear foci of slightly high signal entering the anterior labrum from the joint side (*arrow*), a radial fibrillated tear

Fig. 7 Coronal T1-weighted post-arthrogram image shows multiple tear configurations (*arrow*), reflecting a complex tear

Several studies have found very conflicting results as to the frequency of sulci at all different locations [19–22]. In the author's experience, sulci are frequent in all quadrants (particularly around 8 o'clock and 11 o'clock) except for anterosuperiorly. Given the majority of these studies have found normal sulci in the anterosuperior quadrant, it would be impossible to distinguish a focal partial-thickness peripheral detachment from a sulcus in this location. Fortunately, for MRI diagnosis, tears most often extend over multiple hours on the clockface in this quadrant (Fig. 11). Additionally, isolated tears in quadrants other than the anterosuperior quadrant are infrequent.

The suitability for tear repair or even refixation can be assessed by noting the underlying quality of the labrum. As mentioned previously, the normal labrum is homogeneously low in signal. A degenerated labrum is usually diffusely intermediate to slightly high in signal. Occasionally, a frank round fluid signal focus can be seen in the labral substance, consistent with focal mucoid/myxoid degeneration.

A paralabral cyst is pathognomonic for a labral tear, often seen in the setting of underlying extensive labral degeneration (Fig. 12). The vast majority of the cysts have the same signal intensity as the joint fluid. On occasion, they may have different signal than joint fluid, likely due to mucinous or proteinaceous contents. They are well circumscribed and may be multilobulated or multiseptated. While usually small, they may be large and elongated. Even rarer, large paralabral cysts have the potential to impinge on adjacent neurovascular structures. A connection/communication of these cysts with the labrum should be seen on MRI; the communication may be very small and only definitively visualized on one image of the entire exam. Nonvisualization of a discrete communication should raise the possibility of a cystic mass.

In using MRI for labral tear detection, it is useful to recognize the strengths and limitations for the standard imaging planes used in the evaluation. Coronal images are best to evaluate the superior labrum. Oblique axial images are best to evaluate the anterior and posterior labrum. Sagittal images are best to evaluate the anterior labrum around the 3 o'clock position. However, not infrequently anterior tears can be seen, occasionally to better advantage, on the coronal images (Fig. 13).

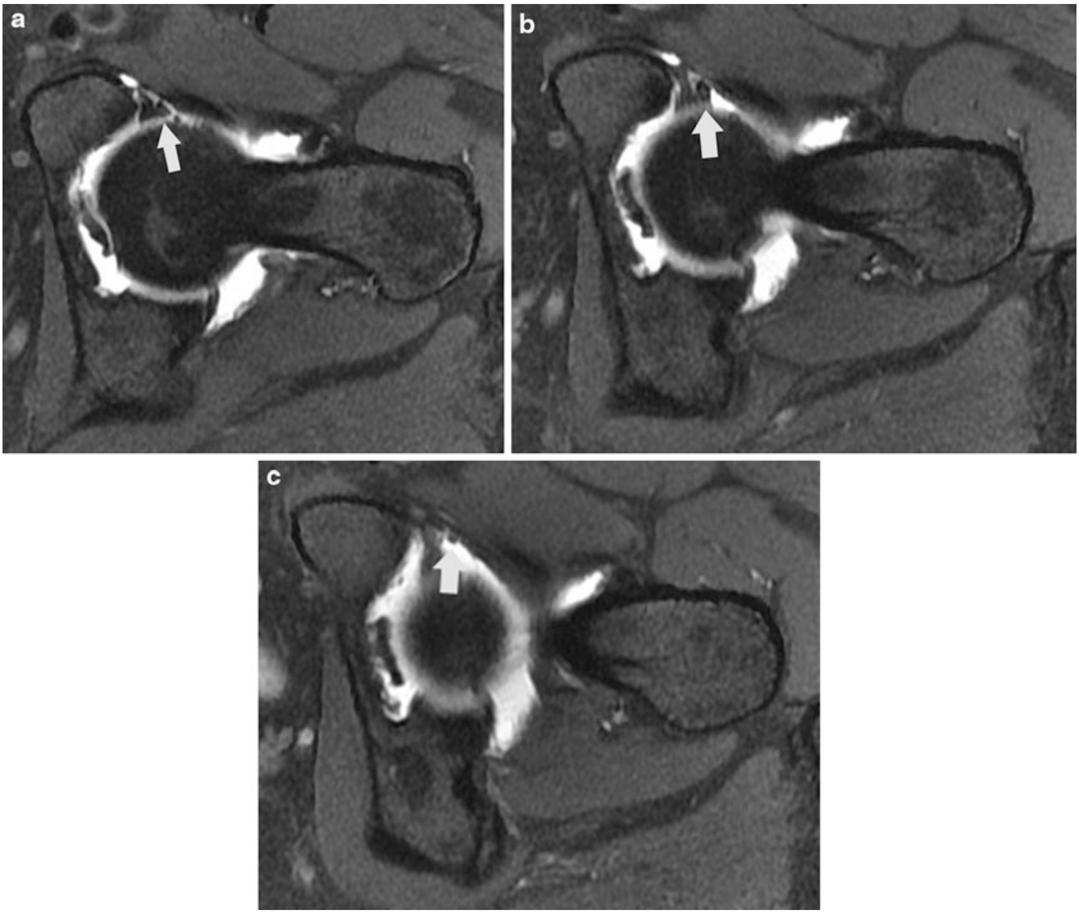


Fig. 8 (**a**–**c**) Sequential post-arthrogram fat-suppressed oblique axial T1-weighted images show a flap (*arrows*) emanating from the torn anteroinferior labrum

Additionally, superior tears can be seen on the oblique axial images. Due to the obliquity of the dome, the labrum is more difficult to evaluate accurately anterosuperiorly (particularly around 1 o'clock) and posterosuperiorly (particularly around 11 o'clock) in all standard imaging planes; the labrum is not crisply visualized due to the curvature and volume averaging in these locations. Given this relative limitation, as the vast majority of tears are located in the anterosuperior quadrant, particular scrutiny of all imaging planes should be made of this quadrant. Labral tears, including those in the anterosuperior quadrant, can be seen on low-resolution and/or large field of imaging studies (Figs. 14, 15, and 16).

Radial MR imaging has thus been utilized to evaluate the labrum. Radial imaging is performed by using a plane perpendicular to the acetabular rim, obtained from the oblique axial and coronal sequences. Slices in this plane are then obtained at a constant interval (usually 15° intervals) over 360°. This technique lessens the problem of volume averaging in standard imaging planes as the labrum can be imaged in a more direct, perpendicular fashion at all clockface locations. Somewhat surprisingly, however, the use of the sequence has been shown in one study to not improve the accuracy of labral tear detection [23].

Certain labral tear sites are associated more frequently with certain conditions. For example,

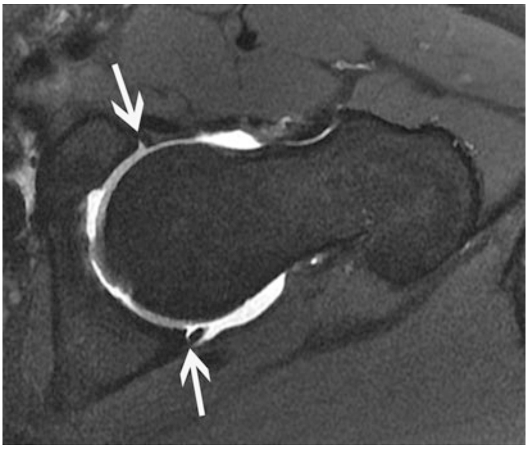


Fig. 9 Fat-saturated oblique axial T1-weighted post-arthrogram image shows partial-thickness linear high signal at the anteroinferior and posteroinferior chondrolabral junctions (*arrows*), representing normal sulci

Fig. 10 Fat-saturated coronal T1-weighted image shows a fluid-filled cleft at the posterosuperior chondrolabral junction (*arrow*), representing a sulcus

given the otherwise relative rarity, the presence of a posterior labral tear suggests prior posterior subluxation or dislocation. An isolated tear at or close to 3 o'clock may reflect iliopsoas impingement, particularly in the absence of FAI pathomorphology or its sequelae (Fig. 17) [24–26]. However, the labrum may appear entirely normal at MRI in iliopsoas impingement; no findings suggestive of the focal injection and inflammation seen at arthroscopy are detected with MRI. No specific findings related to the iliopsoas tendon have been found in the impingement patients [24, 25].

Cartilage

Morphologic Imaging

As the extent of chondral loss is a primary determinant of hip preservation surgery outcomes, accurate detection is paramount [27–30]. The accuracy in detection of cartilage defects has varied widely as has interobserver agreement [31–34]. More specifically pertaining to delamination, the sensitivity of its detection is poor to moderate at best [31, 32, 34]. Detection of chondral defects is particularly difficult on MRI due to the curved, closely opposed surfaces of the dome and head. Magnifying the difficulty is the relatively thin cartilage over both surfaces. Moreover, the majority of pathology is located anterosuperiorly, a site of prominent volume averaging on MRI.

Fluid-sensitive sequences are utilized for cartilage evaluation. Likewise, post-arthrogram T1-weighted sequences, typically with fat suppression, are often utilized. The oblique axial and coronal planes are complementary in evaluating the cartilage in the anterosuperior quadrant. The sagittal images may also be complementary, usually for evaluating the cartilage over the dome as volume averaging greatly impedes evaluation of the relatively thinner cartilage over the subjacent head. Given the unavoidable volume averaging that occurs at 1–2 o'clock (or in any location other than around 3, 12, and 6 o'clock), there should be particularly close scrutiny for subtle findings at these locations in all planes (Fig. 18). If chondral pathology is seen on coronal images at 12 o'clock, far and away more common over the dome, this should heighten the scrutiny of the 1–2 o'clock positions to detect extension.

The oblique axial sequence is best in evaluating the cartilage over the anterior dome and head as well as the anterior and posterior parafoveal region. The coronal sequence is best

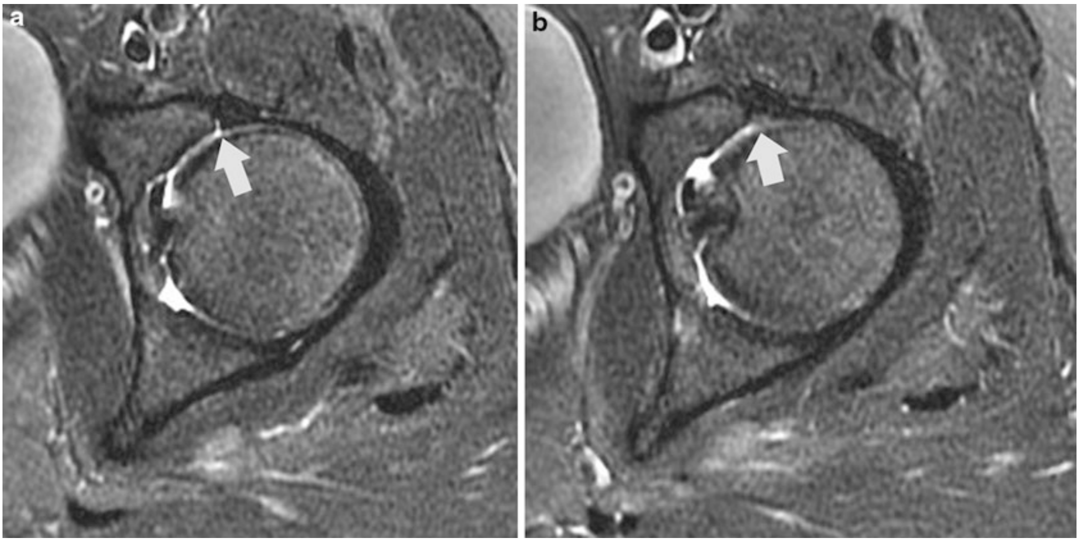


Fig. 11 Adjacent fat-suppressed axial T2-weighted images show linear partial-thickness fluid at the chondrolabral junction at 3 o'clock (*arrow* in **a**). If the adjacent labrum was normal, this could be a partial-thickness peripheral detachment or a normal sulcus. However, on the next most superior image at 2 o'clock, there are abnormal signal and shape of the labrum (*arrow* in **b**). Thus, the findings are consistent with tear

Fig. 12 Fat-suppressed sagittal proton density-weighted image shows a small well-circumscribed, homogeneous fluid collection, a paralabral cyst (*arrow*), communicating with an anterosuperior labral tear

in evaluating the cartilage over the medial dome and head. The sagittal sequence is complementary to the oblique axial sequence in evaluating the cartilage over the posterior dome and head; it is also complementary to the coronal sequence in the evaluation of the cartilage over the superior dome and head.

There are many grading systems for chondral loss with fairly extensive variations between multiple schema [35]. On MRI, defects can be classified as superficial, partial-thickness, or full-thickness/exposed subchondral bone. While it would be ideal to distinguish low-grade partial-thickness defects (less than 50 % thickness) from high-grade defects, in practicality this is often very difficult, particularly using studies with suboptimal resolution and at sites of volume averaging (e.g., 1 o'clock). For these same reasons, distinguishing between deep partial-thickness and full-thickness defects, as well as the extent of chondral loss, can be extremely difficult (Fig. 19). A clue to look closer for overlying chondral loss is a subchondral marrow edema pattern, sometimes relatively small, focal, and relatively innocuous in appearance (Fig. 20) [36].

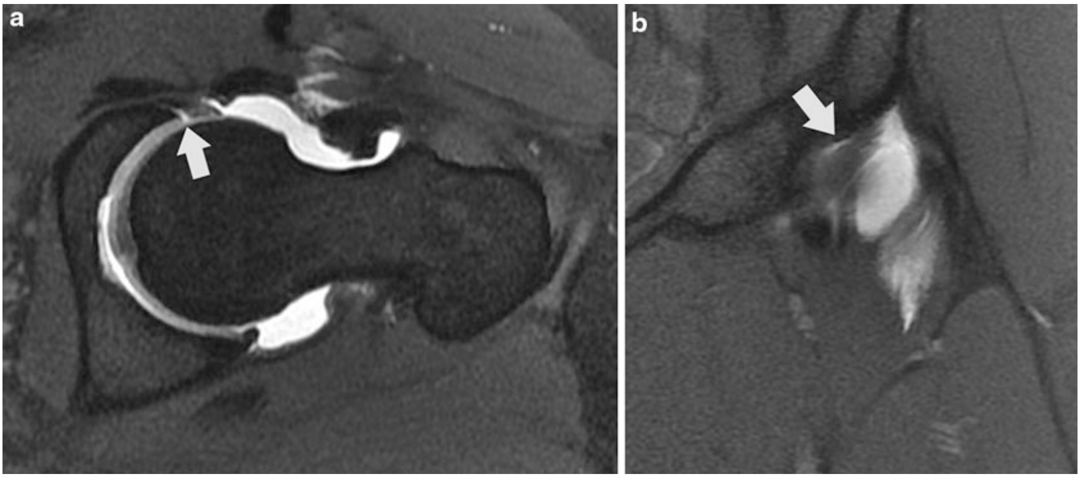


Fig. 13 Fat-suppressed oblique axial (**a**) and coronal (**b**) T1-weighted images in a patient after prior femoroplasty show a peripheral detachment centered around 3 o'clock. Note how the extent of the detachment is nicely displayed in (**b**). Also, note the diffuse intermediate signal and slightly irregular contour of the anterior labrum in (**a**), reflecting diffuse degeneration; this is in contrast to the normal low-signal posterior labrum

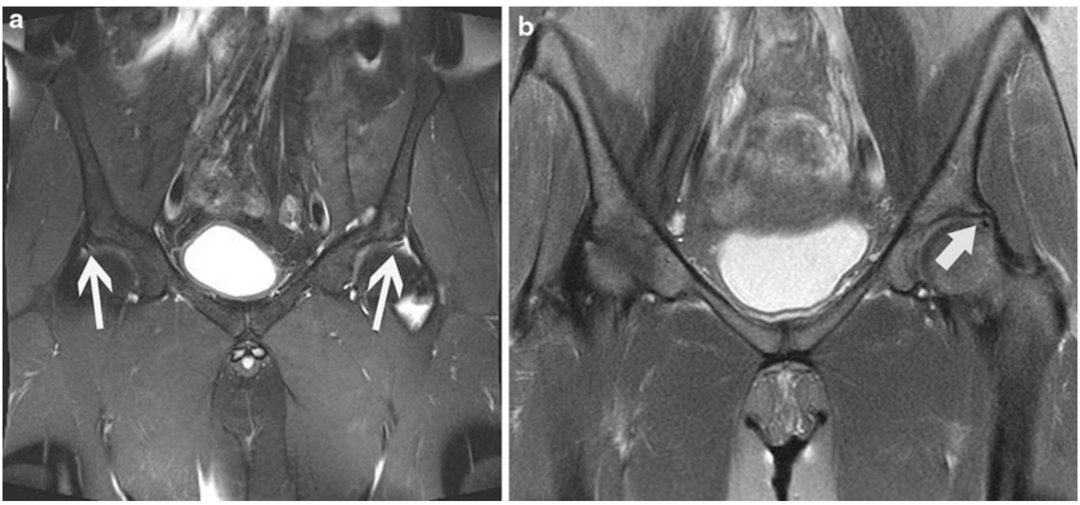


Fig. 14 (**a**, **b**) Fat-suppressed coronal T2-weighted images (different patients) on pelvis MRI show linear signal in the anterosuperior labrum or at the anterosuperior chondrolabral junction (*arrows*), representing tears

Chondral delamination (i.e., frank debonding from the subchondral plate) can clearly be seen at times on MRI when thin, elongated high signal on fluid-sensitive sequences, usually the same signal as joint fluid, is interposed between the subchondral plate and the overlying cartilage (Figs. 21 and 22). Delamination can also be detected when high chondral signal is present with overlying thin low signal; this has been described as the inverted "Oreo" cookie sign given the low signal is "sandwiched" between the deep high signal and the superficial joint fluid (Fig. 23) [37]. Although unclear as to the etiology of the superficial low signal, it could potentially reflect proteoglycan depletion or fibrous metaplasia [37].

Intrasubstance chondral delamination (carpet lesion) can also be seen but is often difficult to

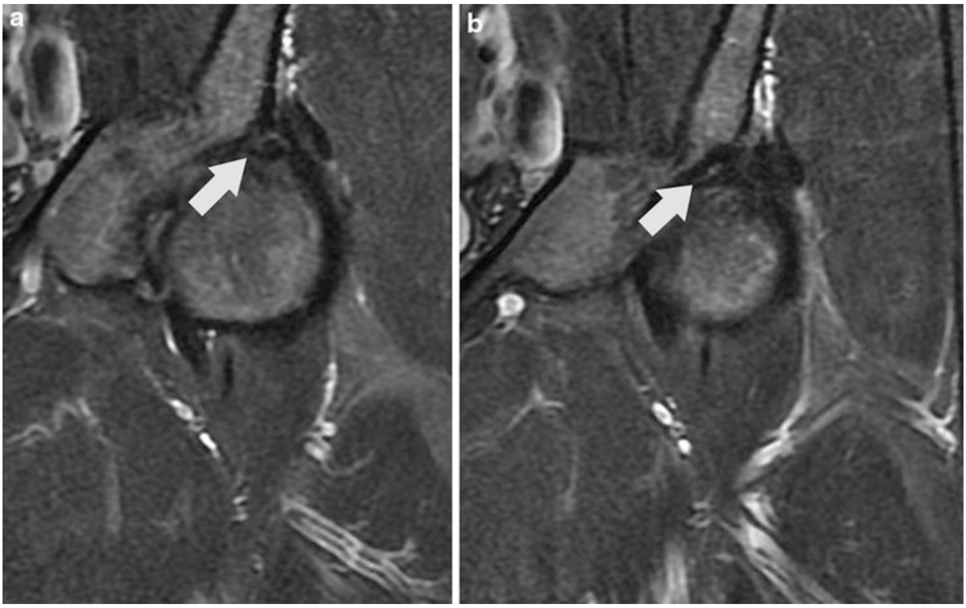


Fig. 15 (**a**, **b**) Successive fat-suppressed coronal T2-weighted images show very thin, linear, slightly high signal at the anterosuperior chondrolabral junction (*arrows*), a peripheral detachment

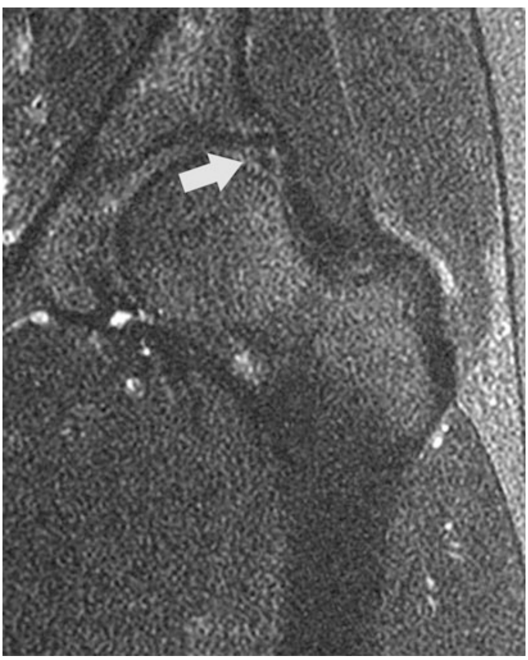


Fig. 16 Fat-suppressed coronal T2-weighted image, markedly "grainy" due to low signal-to-noise ratio, shows vague heterogeneous high signal at the chondrolabral junction at 1 o'clock (*arrow*), reflecting tear

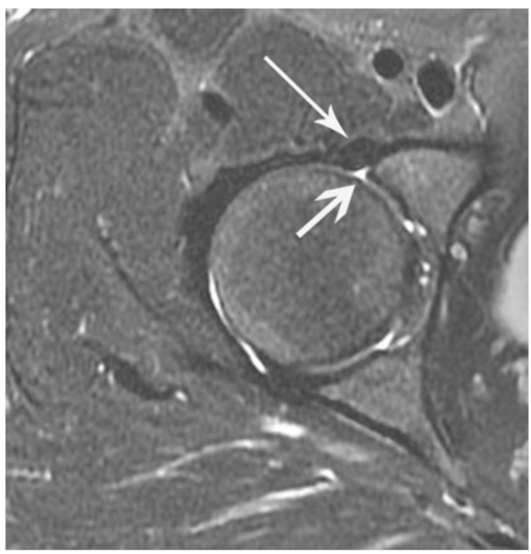


Fig. 17 Fat-suppressed oblique axial T2-weighted image shows a peripheral labral detachment at 3 o'clock (*shorter, thicker arrow*) with adjacent iliopsoas tendon (*longer, thinner arrow*) in a patient with iliopsoas impingement. Note that in many of patients with iliopsoas impingement, the tendon is not directly adjacent to the tear

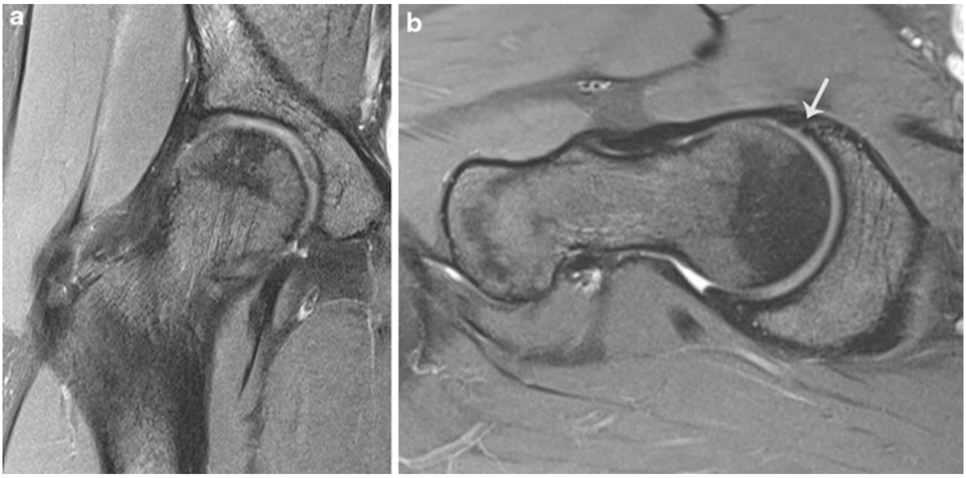


Fig. 18 Fat-suppressed coronal proton density-weighted images show loss of definition of the cartilage over the head and dome at 1 o'clock (**a**) and 2 o'clock (**b**). Note the anterior labral tear in (**b**) (*arrow*)

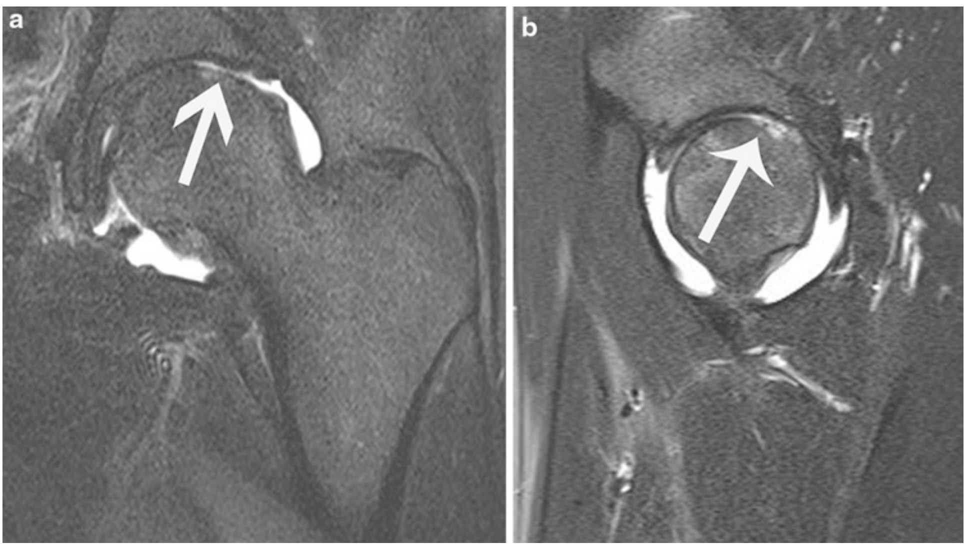


Fig. 19 (**a**, **b**) Post-arthrogram fat-suppressed coronal and sagittal T2-weighted images show extensive chondral loss over the posterosuperior head (*arrows*). While exposed subchondral bone is clearly seen, there are adjacent areas in which deep partial-thickness chondral loss appears to be present (particularly lateral to the exposed bone in (**a**). There was a 30 × 8 mm area of exposed bone over the head at arthroscopy; even in retrospect, the greatest extent of exposed bone that could be visualized on MRI was 20 mm

discern from frank basal delamination. This can be seen when there is elongated, linear high signal in the cartilage but with clearly identifiable cartilage located deep to the signal. Often the high signal extends peripherally to the chondrolabral junction and/or communicates with a fissure. Cartilage that is clearly heterogeneous, containing areas of slightly high signal without a frank fissure or superficial defect, likely reflects extracellular matrix depletion/degeneration. This may manifest at arthroscopy as chondral "softening" and the wave sign, consistent with chondral extracellular matrix disorganization/degeneration (Fig. 24). Importantly, it is often very difficult to discern

Fig. 20 Fat-suppressed sagittal T2-weighted sequence shows a very mild, irregularly shaped marrow edema pattern in the dome (*short*, *large arrow*) adjacent to elongated high signal at the adjacent tidemark (*long*, *thin arrow*), reflecting chondral delamination

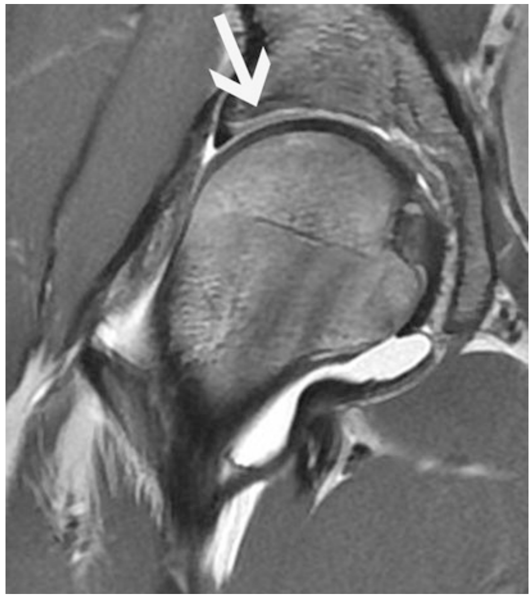


Fig. 21 Post-arthrogram coronal proton density-weighted image shows extensive linear, elongated fluid extending from the chondrolabral junction over the far anterosuperior dome (*arrow*) adjacent to the tidemark (represented by adjacent deep low-signal line), representing chondral delamination

whether elongated, linear high chondral signal represents delamination, whether frank debonding from the tidemark or within the substance of the cartilage, from chondral softening, particularly on low-resolution exams (Fig. 25).

Although less frequent, chondral defects can be seen on MRI over the head, particularly in the parafoveal region (Figs. 26, 27, and 28). Due to volume averaging, detecting and quantifying the depth and extent of loss of the thinner cartilage over the periphery of the head is frequently very difficult. Likewise, due to the relative decreased thickness over the central dome, defects in this location are usually more difficult to visualize. Posteroinferior chondral loss may be detected, usually indicating the effects of contrecoup impaction in pincer FAI (Fig. 29).

For reasons stated previously, high-resolution MR imaging is paramount for accurate detection of chondral pathology. While less specific due to the low resolution obtained with larger fields of imaging, chondral defects can be visualized, particularly higher-grade defects. In some cases, delamination can be suspected (Fig. 30).

One potential pitfall in cartilage evaluation is the supra-acetabular fossa (Fig. 31). This is a small, well-defined concavity of the subchondral plate of the dome, located at 12 o'clock. There is varying overlying chondral thickness, including absence of the overlying cartilage. In a study of 1,002 patients with MRA, it was present in 10.5 % of cases [38].

On a different note, it is important to note when ordering an MRI if metallic fixation hardware is present. While the hardware should be recognized by the radiologist protocoling the MRI, this presupposes that radiographs are viewed at the time of protocol determination which is not always the case. MRI technologists most often perform the exam as protocoled. Thus, images will be obtained with marked magnetic susceptibility artifact if the usual spectral fat-suppressed sequences are performed (Fig. 32). This artifact may greatly obscure the adjacent cartilage and labrum, particularly if situated close to the central compartment.

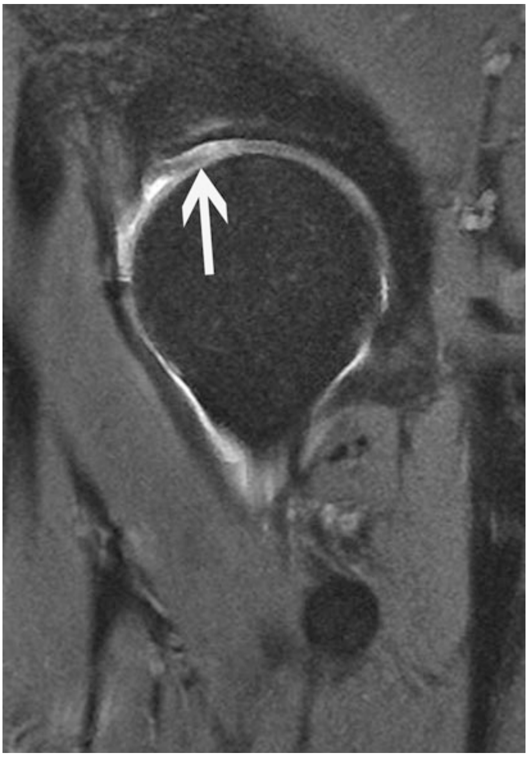


Fig. 22 Fat-suppressed post-arthrogram sagittal T2-weighted image shows linear fluid signal extending from the chondrolabral junction over the anterosuperior dome adjacent to the tidemark (*arrow*), consistent with chondral delamination. Note the adjacent subchondral high signal in the dome, representing an associated marrow edema pattern

In these cases, it is recommended that nonfat-suppressed sequences be performed for more accurate assessment of the cartilage and labrum.

Quantitative Imaging

Techniques have been used in MRI to more quantitatively assess the status of the cartilage extracellular matrix. They afford detection of early derangement of the matrix contents, a marker of early degeneration. These techniques include delayed gadolinium-enhanced MRI of cartilage (dGEMRIC), T2 mapping, and T1-rho imaging.

dGEMRIC imaging is performed with intravenous, or less frequently, intra-articular gadolinium. After administering gadolinium, the patient exercises for usually a 10- to 15-min period, and imaging is performed at 45–90 after the contrast is given. Given the negative charge of gadolinium, it accumulates within the cartilage in proportion to the degree of loss of negatively charged glycosaminoglycans (proteoglycan side chains), measured by the dGEMRIC index. T2 mapping assesses the degree of loss of collagen fiber orientation and subsequent increased mobility of water in the matrix; T2 values increase proportional to the degree of collagen disorganization. T1-rho imaging assesses the interactions between hydrogen and the macromolecules in water, correlating with proteoglycan content; T1-rho values increase as the proteoglycan content decreases [39].

dGEMRIC has been studied most frequently in the hip. In a study of patients with hip dysplasia, Kim et al. found the dGEMRIC index to correlate with pain and the severity of dysplasia [40]. In a study of 47 patients undergoing a Bernese periacetabular osteotomy for dysplasia by the same group, among radiographic evidence of osteoarthritis, subluxation, and the dGEMRIC index, the index was found to be the most important predictor of failure [41].

Ligamentum Teres

The contribution of ligamentum teres tears to pain is difficult to accurately pinpoint as most patients have other coexistent pain-generating pathology (e.g., labral tears, chondral lesions) [42–44]. Pain due to isolated tears without other intra-articular pathology can, however, occur [45]. Its contribution to hip stability is controversial [46]. If the ligament has a significant contribution to stability, it would be expected and make sense that patients having previously undergone surgical hip dislocation with usual sacrifice of the ligament would have subsequent instability; however, to the author's knowledge, no such literature of instability following surgical dislocation exists.

The ligament is usually composed of two bands, although it has been found to instead be composed of three bands [47]. Congenital absence of the ligament has been described [48]. The normal ligamentum teres is homogeneously low in signal on all sequences, extending from the fovea/bare area distally to insert on the

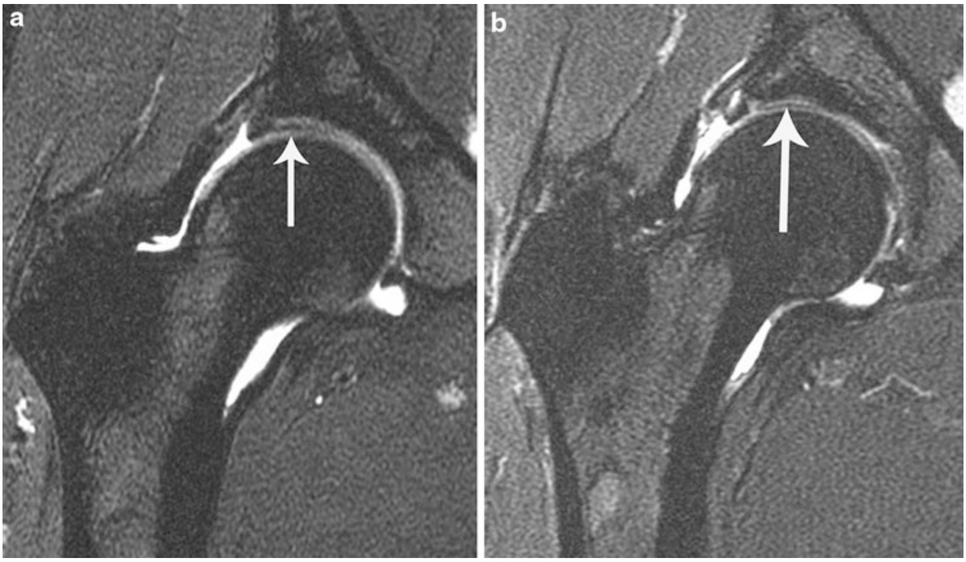


Fig. 23 Successive post-arthrogram fat-suppressed coronal T2-weighted images show surgically documented chondral delamination over the lateral dome, manifested as minimally high signal in the cartilage with adjacent superficial linear low signal (*arrow* in **a**) and high signal "sandwiched" between two linear areas of linear low signal (*arrow* in **b**). Also note the adjacent labral tear in (**b**)

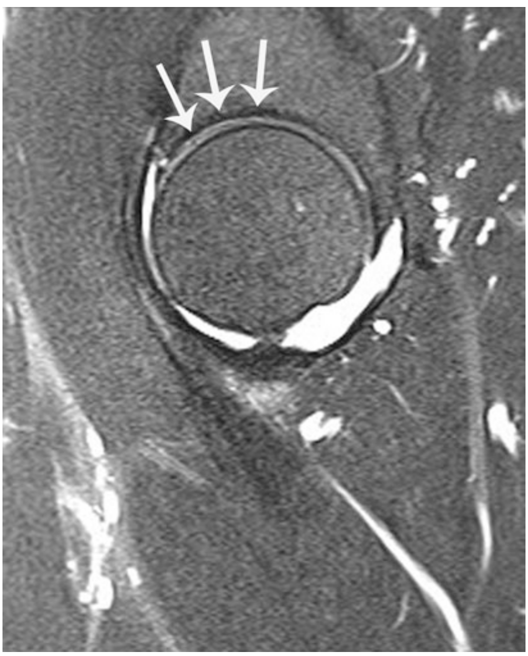


Fig. 24 Post-arthrogram fat-suppressed sagittal T2-weighted image shows extensive thin linear signal in the cartilage over the dome (*arrows*); a wave sign was noted at arthroscopy. As in this case, it is often difficult to differentiate between chondral delamination and "softening" on MRI

transverse acetabular ligament and adjacent pubis and ischium. It is more ovoid in shape proximally at the fovea; its midportions and distal portions are flatter and somewhat pyramidal in shape.

Like all ligaments, intrasubstance degeneration is common with aging, most frequently observed proximally, manifested as intermediate and high signal within its substance on fluid-sensitive sequences. Given the mucoid/myxoid and fatty degeneration, intrasubstance intermediate to high signal may be seen within the ligament on T1-weighted sequences. Additionally, occasionally osseous metaplasia can be seen in the ligament.

Partial tears of the ligament are fairly common (up to 46 %) and ruptures less so (4–15 %) [42, 45, 49, 50]. Partial tears are often difficult to differentiate from degenerative change of the ligament on MRI. The sensitivity and specificity in partial tear detection are subpar [51].

As most partial tears occur proximally at or near the fovea, this location should be most highly scrutinized. The ligament has normal striations of high to slightly high signal on fluid-sensitive sequences, reflecting interstitial fluid between its

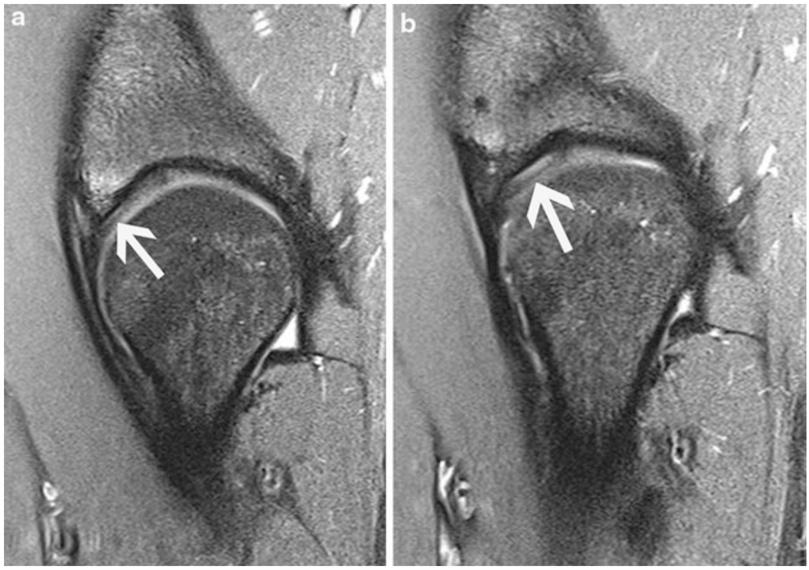


Fig. 25 (**a**, **b**) Successive fat-suppressed sagittal proton density-weighted images show linear high signal deep within the cartilage over the dome, adjacent to the tidemark (*arrows*). While this appears to be chondral delamination, only softening was found at arthroscopy

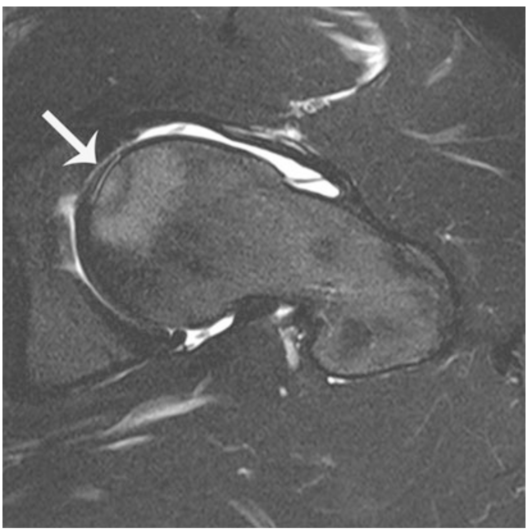


Fig. 26 Post-arthrogram fat-suppressed oblique axial T2-weighted image shows linear fluid signal adjacent to the tidemark over the anterior suprafoveal head (*arrow*), reflecting chondral delamination. Note the adjacent extensive subchondral high signal (i.e., marrow edema pattern). Note the large cam lesion

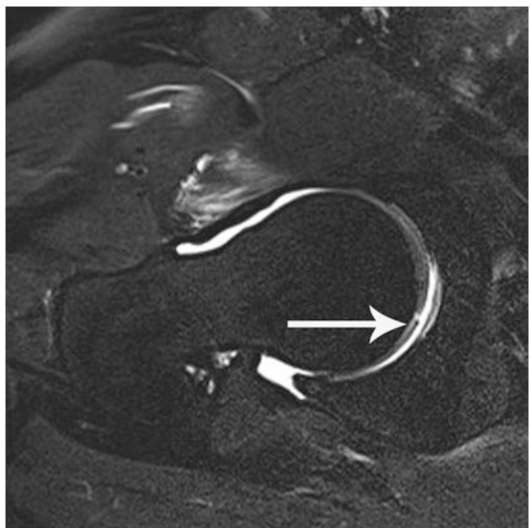


Fig. 27 Post-arthrogram fat-suppressed oblique axial T2-weighted image shows a chondral flap over the posterior suprafoveal head (*arrow*)

layers of collagen, throughout its course that usually become more conspicuous with aging; this signal is also located between its discrete bands. Both the intact degenerated ligament and partially torn ligament can have frayed margins and be attenuated or thickened on MRI (Fig. 33). Partial tears may be small, very thin, and focal. For definitive diagnosis of a partial tear, fluid signal should be seen extending to at least one margin of the ligament (Fig. 34).

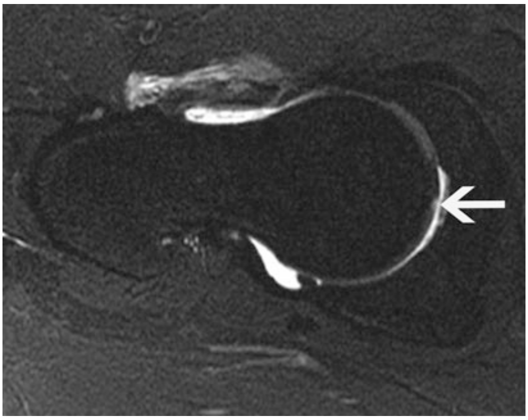


Fig. 28 Post-arthrogram fat-suppressed oblique axial T2-weighted image shows a tiny, focal partial-thickness chondral defect, less than 50 % in thickness, over the suprafoveal head (*arrow*)

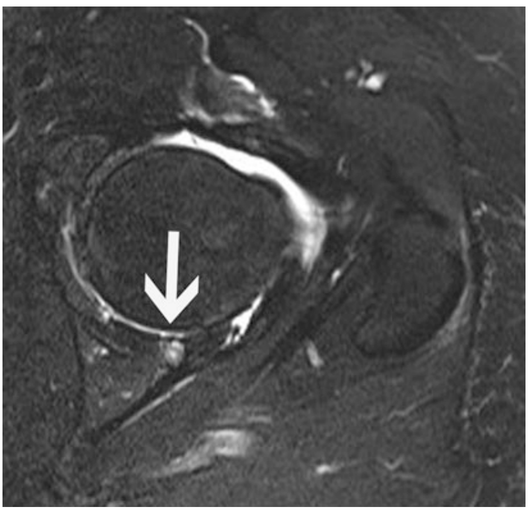


Fig. 29 Post-arthrogram fat-suppressed oblique axial T2-weighted image shows exposed bone over the posteroinferior dome (*arrow*) with associated subchondral cyst

However, partial tears can simply manifest with intrasubstance signal. In an MR arthrographic study, 10 (out of 12) known partial tears only had linear intrasubstance fluid signal, with or without irregular contour of the adjacent periphery of the ligament; intact ligaments also

Fig. 30 Post-arthrogram fat-suppressed sagittal proton density-weighted image shows vague slightly high signal deep in the cartilage over the dome, marginated by superficial linear low signal (*arrows*). This was delaminated cartilage at arthroscopy

had similar findings [51]. Hence, both false-positive and false-negative diagnoses on MRI are not unexpected. Further complicating matters of diagnosis, as Botser et al. state in their series delineating partial tears into less than or more than 50 % in thickness, "low-grade partial-thickness tears . . . may have been disregarded in previous literature" [42].

The oblique axial (or straight axial) plane is usually the best plane to detect partial tears as the ligament is seen in cross section. The coronal plane may also be useful, although the normal striations within and thin separation between the bundles can simulate tears. The sagittal plane is frequently unhelpful given relatively poor visualization of the ligament and volume averaging as this is the plane that the majority of the ligament traverses. In addition, the so-called ligamental plica can often be seen just medial to the ligament, particularly after arthrography and in the setting of a joint effusion; although rarely confusing due to its clear separation from the ligament and its

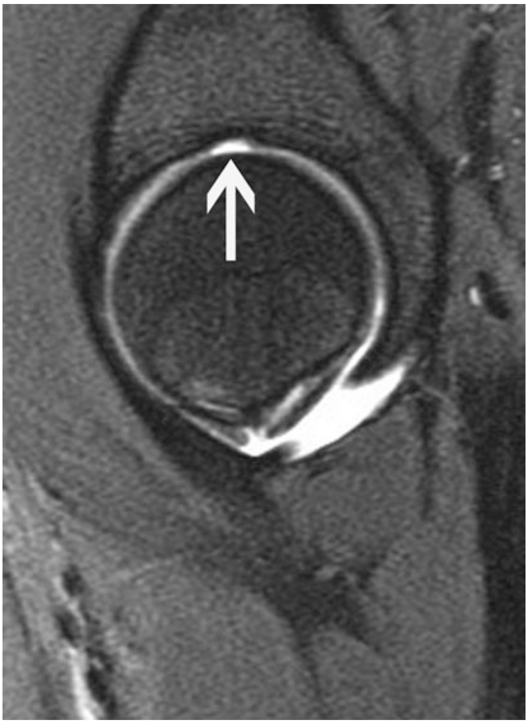


Fig. 31 Post-arthrogram fat-suppressed sagittal T2-weighted image shows a focal well-defined concavity of the subchondral plate with thin overlying cartilage at 12 o'clock (*arrow*), representing a normal supra-acetabular fossa

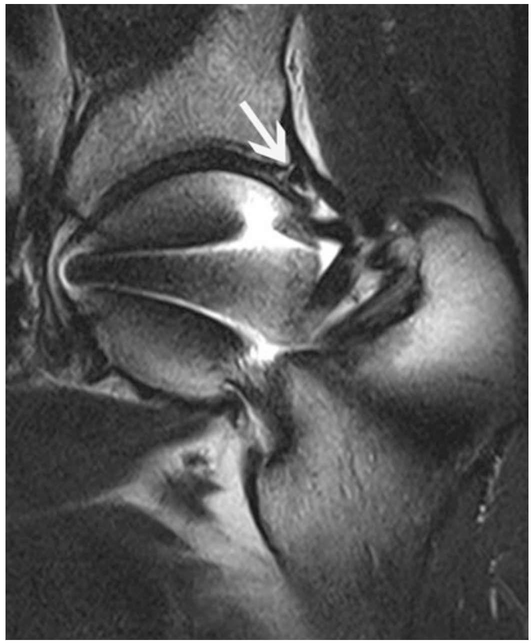


Fig. 32 Post-arthrogram fat-suppressed sagittal T2-weighted image shows marked magnetic susceptibility artifact associated with SCFE fixation screw. *Arrow* points to incompletely visualized anterior labrum, obscured by the artifact

elongated course, it should not be mistaken for a partial tear (Fig. 35) [52].

Rupture of the ligamentum teres is most often due to prior hip dislocation or in the setting of advanced degeneration [53]. Ruptures can occasionally be due to a twisting injury [45]. Rupture detection is straightforward. Frank discontinuity is present, usually at the fovea or in its midportion (Fig. 36). The ligament distal to the site is frequently attenuated and wavy. While occasionally reactive marrow edema, or a frank intraosseous ganglion, is present at its foveal insertion, almost always in the setting of ligament degeneration, parafoveal marrow edema can signify the presence of an acute rupture/avulsion in the setting of recent trauma.

No description of the MRI appearance of ligamentum teres reconstruction or tears of reconstructions in the literature is known to the author. Likely, similar to the appearance of the

Fig. 33 Post-arthrogram coronal proton density-weighted image shows marked fraying and heterogeneous, slightly high signal in the proximal ligamentum teres (*arrow*). This appearance can be seen in both the partially torn and degenerated, intact ligament

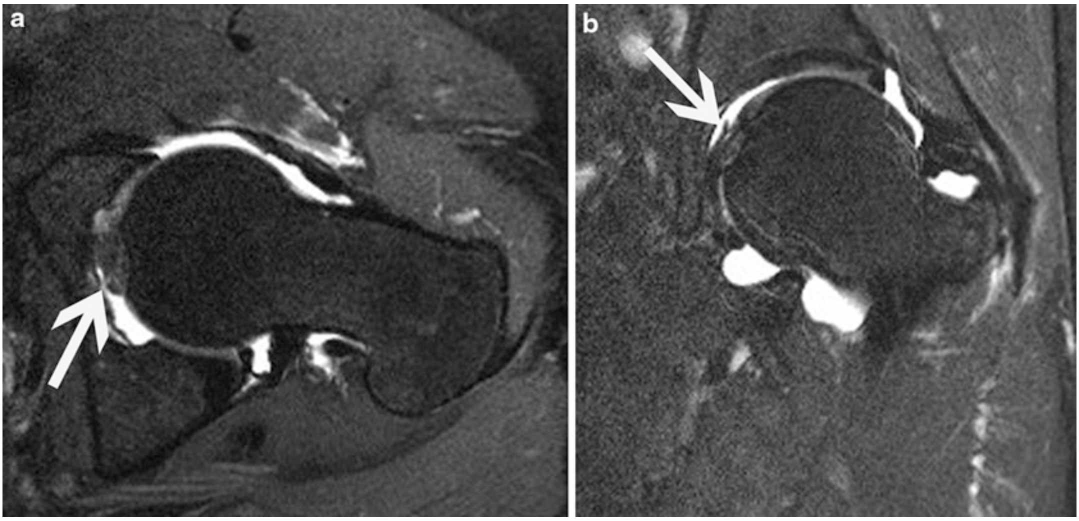


Fig. 34 Post-arthrogram fat-suppressed oblique axial (**a**) and coronal (**b**) T2-weighted images show focal, thin high signal in a portion of the proximal ligamentum teres, extending to its surface (*arrows*), consistent with partial tear as was found at surgery

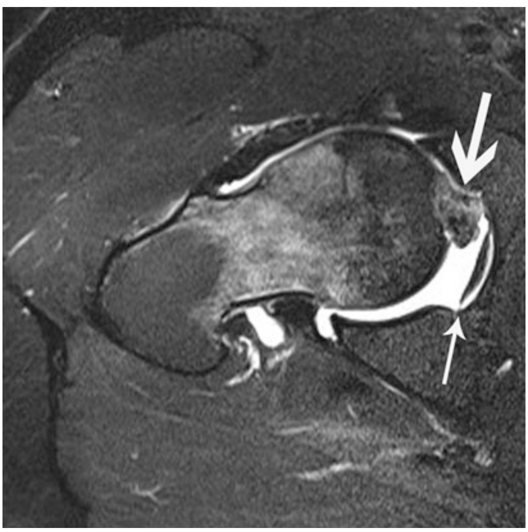


Fig. 35 Post-arthrogram fat-suppressed oblique axial T2-weighted image shows hypertrophy and heterogeneous signal in the proximal ligamentum teres with linear partial tear (*thicker arrow*). Also note the thin, elongated low-signal band-like structure deeper in the cotyloid fossa (*thinner arrow*), a ligamental plica. Note the prominent marrow edema pattern in the neck and distal head related to subchondral fracture associated with head avascular necrosis (not shown). There is also a large cam lesion

Fig. 36 Post-arthrogram fat-suppressed coronal T1-weighted image shows absence of the ligamentum teres at the fovea (*arrow*), representing ligament rupture

reconstructed or augmented labrum, heterogeneous signal is to be expected within the reconstructed ligament on MRI. Tears of the reconstructed ligament most likely will be those of any ligament, namely, fluid signal extending into the ligament with or without an irregular contour or frank displacement or absence of a portion of the ligament.

The Peripheral Compartment

The Capsule

The capsule of the hip includes four distinct thickenings: the iliofemoral, ischiofemoral, and pubofemoral ligaments and the zona orbicularis. The iliofemoral ligament is a stout thickening of the anterior and anterolateral capsule; it is readily visualized on all three usual planes of imaging on MRI. The ischiofemoral ligament is a posteroinferior thickening of the capsule; it is also visualized well on all three standard planes of imaging. The pubofemoral ligament, located caudally, is the most difficult of the capsular ligaments to image fully, best noted on the sagittal and axial sequences. The zona orbicularis, present as a circular sling-like structure about the femoral neck, can be seen in all three planes; it is better visualized in certain locations in different planes (e.g., due to volume averaging, the anterior and posterior portions of the ligament are better seen on the oblique axial sequence than on the coronal sequence).

In an MR arthrogram study of 30 patients, equally divided between men and women, men had a significantly thicker capsule only anteriorly [54]. However, a defined normal thickness of the capsular ligaments is not known. It is also not known what, if any, change in thickness of the ligaments occurs with capsular distention (e.g., after arthrography). However, although nonspecific, as in the glenohumeral joint, subjective capsular laxity suggests capsular instability; this subjective assessment most likely is more accurate in the eyes of an experienced radiologist who has seen many hip joints imaged and has a rough baseline as to the typical appearance of the capsule. In the author's experience, the native capsule in patients with capsular laxity/insufficiency has more prominent outward curvature, particularly evident near the insertions (Figs. 37 and 38). In the author's experience, unless a process is present predisposing the patient to capsular laxity (e.g., Ehlers-Danlos, Marfan, Down syndrome), the insufficient capsule usually subjectively appears normal in thickness.

The iliofemoral and/or ischiofemoral ligament can be disrupted during hip dislocation, as can, occasionally an avulsion fracture at the ligament's insertion. On MRI, the findings are not subtle and usually manifest as a large gap in the injured ligament, often with joint fluid extravasating into the adjacent soft tissues (Fig. 39).

Of note, the iliofemoral ligament can appear markedly irregular and usually abnormally thickened due to iatrogenic contrast injection during

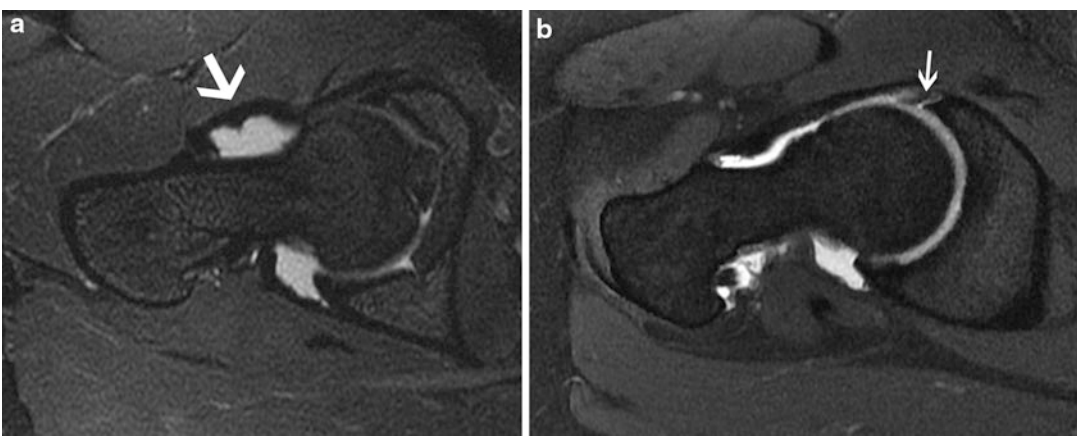


Fig. 37 Post-arthrogram fat-suppressed oblique axial T2-weighted images of two different patients. The patient in (**a**) had instability under anesthesia; note the prominent outpouching of the distal iliofemoral ligament (*arrow*). Compare this to the capsule of the normal patient without instability in (**b**). Incidentally, note the anterior labral tear in (**b**) (*arrow*)

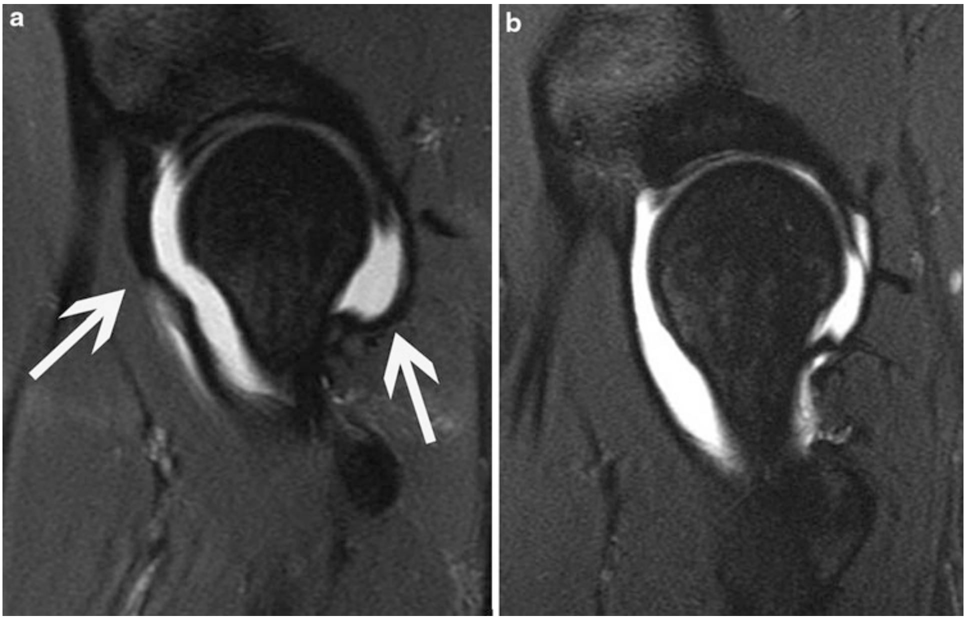


Fig. 38 Post-arthrogram fat-suppressed sagittal T2-weighted images of two different patients. Note the prominent diffuse irregular outpouching of the iliofemoral ligament (anterior *arrow*) and ischiofemoral ligament (posterior *arrow*) in (**a**); this patient had gross instability. Compare to the normal capsule appearance in the patient without instability (**b**)

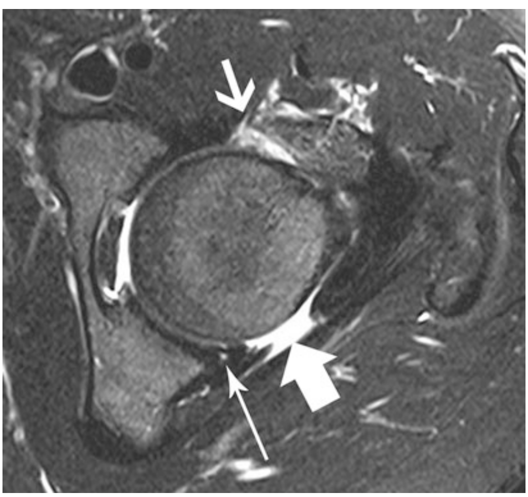


Fig. 39 Post-arthrogram fat-suppressed oblique axial T2-weighted image in a patient with multiple prior dislocations shows wide dehiscence of the ischiofemoral ligament (*thick arrow*), a tear of the iliofemoral ligament (*thinner arrow*) with diffuse heterogeneous thickening of the ligament distally, and small focus of fluid signal in the posteroinferior labrum (*long*, *thin arrow*), reflecting tear (present on other images)

arthrography (Fig. 40). While usually in its midportion, the abnormal appearance can be relatively diffuse. This spurious finding on MRI can usually be detected as the gadolinium-containing mixture is high in signal on the fat-suppressed T1-weighted sequences (unlike normal synovial fluid); the high signal on these sequences will be seen in the ligament at the site of abnormality, often extending into the adjacent soft tissues along the injection track.

Also note that cases of adhesive capsulitis of the hip have been reported [55]. However, to the author's knowledge, no literature exists describing MRI findings of adhesive capsulitis in the hip.

While much more frequently involving the gluteal tendons, calcium hydroxyapatite can form in the capsule. While usually quiescent and incidental finding, occasionally a calcium deposit can cause adjacent inflammation when undergoing a change in composition, associated with inflammation and causing intense pain. The findings on MRI during these episodes can include

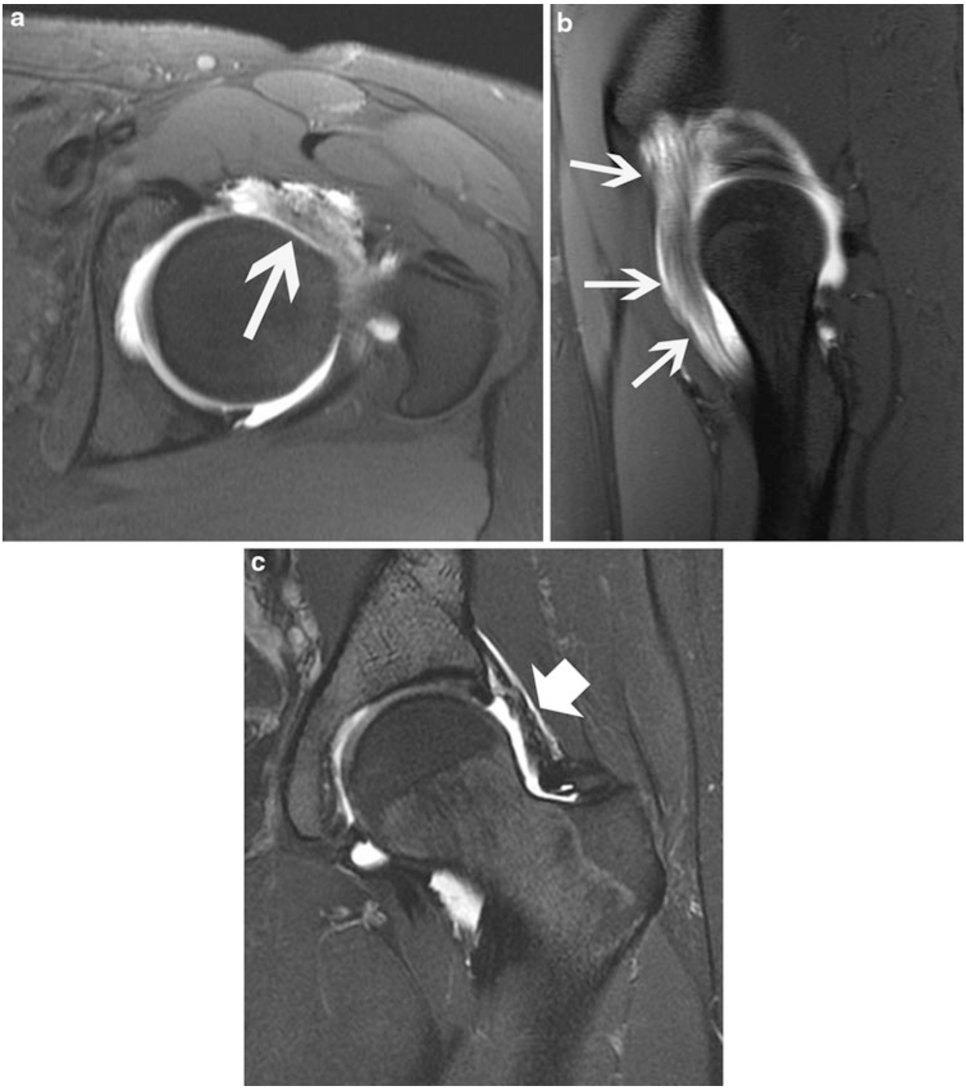


Fig. 40 Post-arthrogram fat-suppressed oblique axial (**a**) and sagittal (**b**) T1-weighted images as well as fat-suppressed coronal (**c**) T2-weighted image of the same exam show extensive, diffuse enlargement and high signal within the iliofemoral ligament (**a** and **b**). Note in (**a**) the proximal ligament appears to be discontinuous. Also, note the irregular, predominantly linear foci of high signal in the overlying superficial soft tissues in (**a**), representing the track of the needle (and contrast) during the arthrogram. T2-weighted images can help to clarify whether the abnormal signal and shape of the ligament are spurious as it will usually be heterogeneously lower in signal than the joint fluid, as in (**c**). Note that no capsular defect was present at surgery

marked soft tissue and marrow edema in and about the calcium deposit and potentially be confused with a neoplastic or infectious process. Thus, although sometimes subtle, close scrutiny for a small focus or clustered foci of very low signal on MRI, representing the calcium deposit(s), should be made. It is usually located within or immediately adjacent to the enthesis, amidst the surrounding edema. The reactive inflammation can cause adjacent osseous cystic and/or erosive change, further simulating an aggressive process (Fig. 41). If no low-signal focus is seen on MRI, comparison with radiographs and/or computed tomography (CT) can

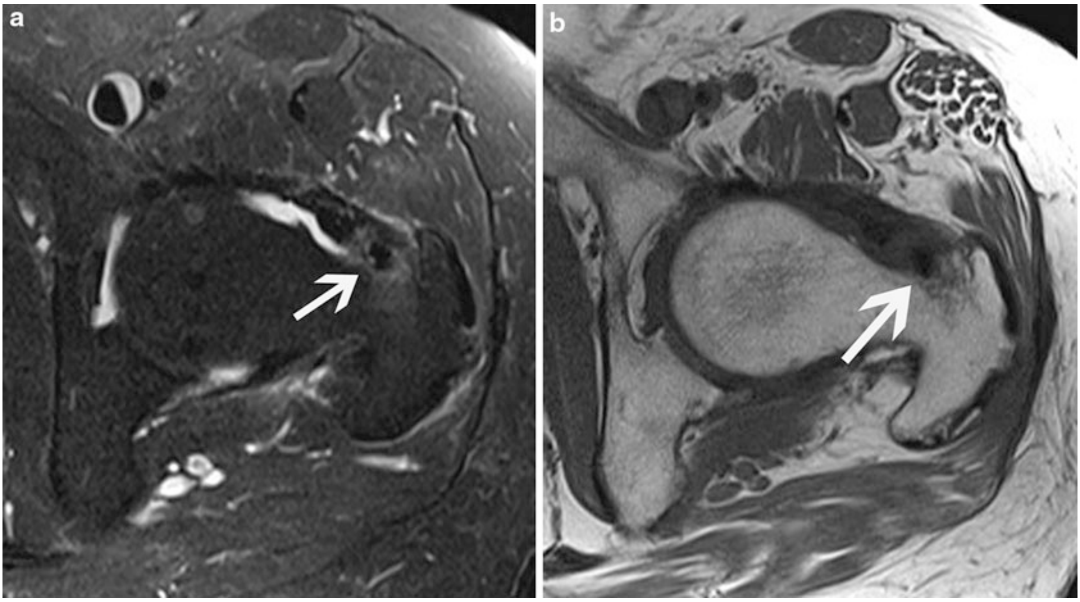


Fig. 41 Fat-suppressed axial T2-weighted image (**a**) and nonfat-suppressed T1-weighted image (**b**) of a patient with acute-onset hip pain. Note the very low-signal focus in the distal iliofemoral ligament at its insertion with surrounding soft tissue and marrow edema pattern (*arrow* in **a**). The low-signal focus of calcium hydroxyapatite can sometimes be better visualized on nonfat-suppressed T1-weighted images (*arrow* in **b**). Also note that a marrow edema pattern has heterogeneously intermediate signal on T1-weighted sequences, as in the intertrochanteric region adjacent to the calcium deposit in B. Note that no other cause for acute hip pain was found

be helpful in detecting calcium deposit(s). Radiographs and, particularly, CT are much more sensitive than MRI in their detection.

Plicae

Synovial plicae, or folds, are normal reflections of the synovial membrane [56]. Plicae are thin, elongated structures that are commonly seen on MRI. Three main plicae have been described: the neck plicae, located along the base of the femoral neck; the ligamental plica, located medial to the ligamentum teres; and the labral plica, located peripheral to the labrum [56]. While usually of no clinical import, there have been documented reports of symptomatic plicae [57, 58].

The plicae are often overlooked at MRI; they are innocuous in appearance and, frankly, often ignored. Plicae are thin, low-signal band-like, elongated structures. The most easily recognized plica is the neck plica known as the pectinofoveal fold. It is an elongated structure in the inferomedial aspect of the joint, usually measuring 2–3 mm in thickness on MRI. It extends from the inferomedial proximal femoral neck distally to insert on the capsule or distal femur [59]. It not infrequently can be slightly irregular in contour [59]. This irregularity in contour may manifest as focal ovoid-like thickening with intermediate to slightly high signal on fluid-sensitive sequences as it passes along the caudal aspect of the subcapital femoral neck (Fig. 42). Occasionally, there may be an adjacent reactive marrow edema pattern or intraosseous ganglion, likely the result of chronic abrasion (Figs. 43 and 44).

The ligamental plica is another commonly identified plica seen on MRI. It is located in the cotyloid fossa medial to the proximal ligamentum teres (see Figs. 35 and 42). It is likewise relatively thin and low in signal and courses distally along the medial aspect of the ligament.

Fig. 42 Post-arthrogram fat-suppressed coronal T1-weighted image shows a pectinofoveal fold that is thin distally (*small arrows*) with focal ovoid thickening proximally adjacent to the caudal subcapital neck (*intermediate-sized arrow*). The fold is unusually diffusely intermediate in signal, as opposed to the more common low signal. Also, incidentally note the high signal within and irregular shape of the proximal iliofemoral ligament (*large arrow*), reflecting iatrogenic contrast injection into the ligament. The very long, thin arrow points to an incidental ligamental plica

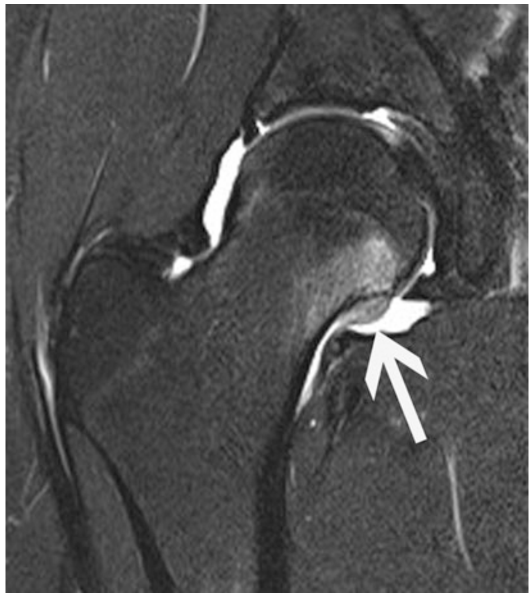


Fig. 43 Post-arthrogram fat-suppressed coronal T2-weighted image shows a marked marrow edema pattern within the femur adjacent to a slightly thickened proximal ligamentum teres with uncommon intermediate signal (*arrow*). The marrow edema pattern, presumably reactive in this young competitive breaststroke swimmer, had decreased in extent on a later MRI

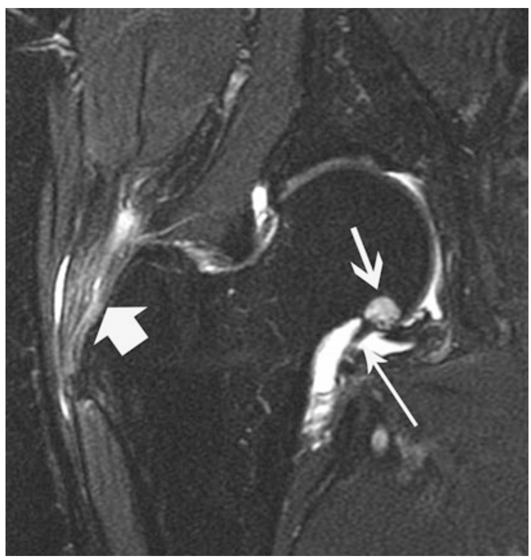


Fig. 44 Post-arthrogram fat-suppressed coronal T2-weighted image shows a well-circumscribed, slightly heterogeneous intraosseous ganglion in the caudal neck (*intermediate-sized arrow*) adjacent to the normal-appearing adjacent pectinofoveal fold (*long, thin arrow*). Incidentally note the extensive degeneration and partial-thickness tearing of the gluteus minimus and medius tendons (*short, thick arrow*)

The Peritrochanteric Space

The Hip Abductors

The gluteal tendons, the so-called rotator cuff of the hip, are important pain generators. The gluteus medius tendon inserts on both the superoposterior and lateral facets of the greater trochanter. The gluteus minimus tendon inserts on the anterior facet of the greater trochanter.

The gluteal tendons degenerate with age. Partial-thickness tears are not infrequent in the young adult. Full-thickness tears are uncommon in the young adult, even in the setting of significant trauma. Tears overwhelmingly occur in the distal tendon at or just proximal to the greater trochanter.

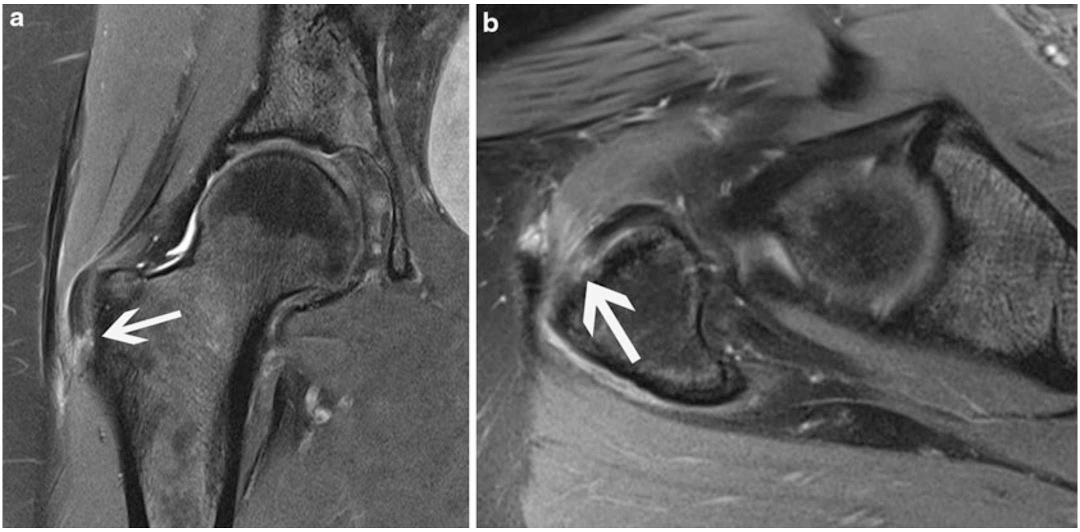


Fig. 45 Fat-suppressed coronal (**a**) and oblique axial (**b**) proton density-weighted images show linear partial-thickness fluid signal within the distal gluteus minimus tendon with adjacent peritendinous and soft tissue edema (*arrows*), representing a partial-thickness tear

Like all tendons, the normal gluteus medius and minimus tendons are homogeneously low in signal. Degeneration is seen as intermediate signal of the tendon, with or without enlargement. Partial tears manifest as linear or irregular fluid signal in the tendon, usually extending from the deep margin into the substance of the tendon (Figs. 45 and 46). Also, partial tears can manifest as subtle signal with focal, often irregular, thinning of the tendon fibers. Full-thickness tears manifest as frank complete fiber disruption, the tear site usually filled with fluid (Fig. 47). Commonly, there is peritrochanteric soft tissue edema and trochanteric bursal distention of some degree in the presence of gluteal tears. Recent tears can usually be distinguished from chronic ones by more extensive surrounding soft tissue edema, often extending into and about the distal muscle-tendon junction.

All planes are useful in assessing the gluteal tendons. The author finds cross-referencing the axial and coronal planes on the picture archiving and communications system (PACS) most helpful to fully delineate the depth and extent of tears, particularly in studies with a large field of imaging

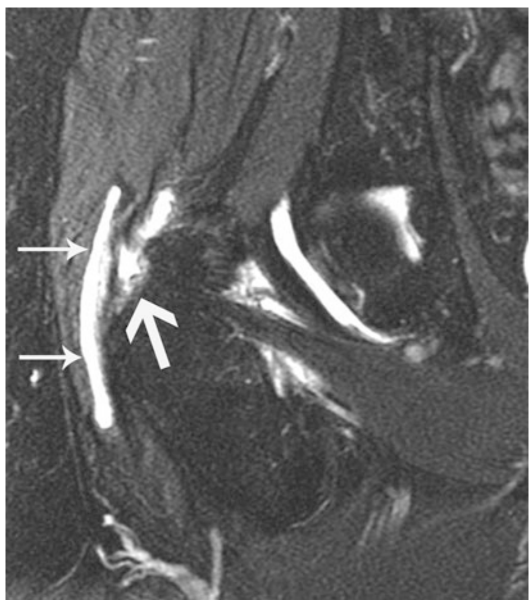


Fig. 46 Post-arthrogram fat-suppressed coronal T2-weighted image shows markedly irregular shape and interposed fluid within the distal gluteus medius tendon at the lateral trochanteric facet (*larger arrow*), representing a high-grade partial-thickness tear. Note the adjacent moderately distended trochanteric bursa (*thinner arrows*)

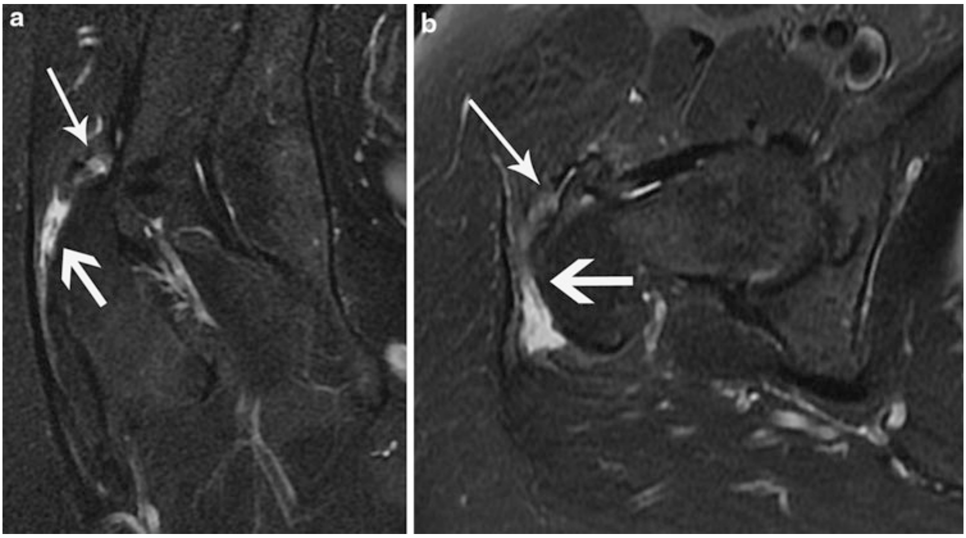


Fig. 47 Fat-suppressed coronal (**a**) and axial (**b**) T2-weighted images show a full-thickness tear of the gluteus medius tendon at the lateral facet ("bald" facet with adjacent high-signal fluid) in A. In B, the full-thickness gluteus medius tendon tear at the lateral facet is again shown (*larger arrow*); there is also a partial-thickness tear of the more anterior gluteus minimus tendon (*thinner arrow*), manifested by an irregular shape and thinning with interposed fluid

(e.g., whole-pelvis MRI). While frequently not an issue, the author finds true axial sequences more helpful than oblique axial sequences as the tendons are more obliquely oriented distally on the latter sequences, particularly the gluteus medius component inserting on the lateral trochanteric facet.

Like the Goutallier grading system of the rotator cuff musculature, the quality of the gluteal muscles can be assessed. While there is no known dedicated grading system known to the author, usually the degree of fat infiltration and muscle atrophy can be graded using a mild, moderate, or severe schema (i.e., fat less than, equal to, or greater than normal muscle signal and bulk, respectively). The degree of fat infiltration is most accurately assessed on T1-weighted sequences due to easy differentiation between high-signal fat and intermediate-signal muscle (Fig. 48). High signal in fluid-sensitive sequences in the musculature is nonspecific but may reflect edema due to recent strain.

In adults, particularly older adults, foci of calcium hydroxyapatite may form in the gluteal tendons. There is a relatively higher incidence of calcium formation in the distal gluteus maximus tendon at the proximal linea aspera. As discussed in the section describing the capsule, prominent marrow and soft tissue edema can be seen as inflammatory change in association with the calcium hydroxyapatite and simulate an infectious or neoplastic process.

The Trochanteric Bursa

There are several bursa about the hip, the two most important being the greater trochanteric and iliopsoas bursae. The greater trochanteric bursa, lying adjacent to the greater trochanter its posterolateral surface, is very frequently mildly thickened on MRI. Likewise, mild to moderate peritrochanteric soft tissue edema is often coexistent. Given the ubiquity of the findings, neither thickening or peritrochanteric edema has specificity and usually is ignored when reporting the MRI findings. Subjectively, mild to moderate greater trochanteric fluid distention can be seen.

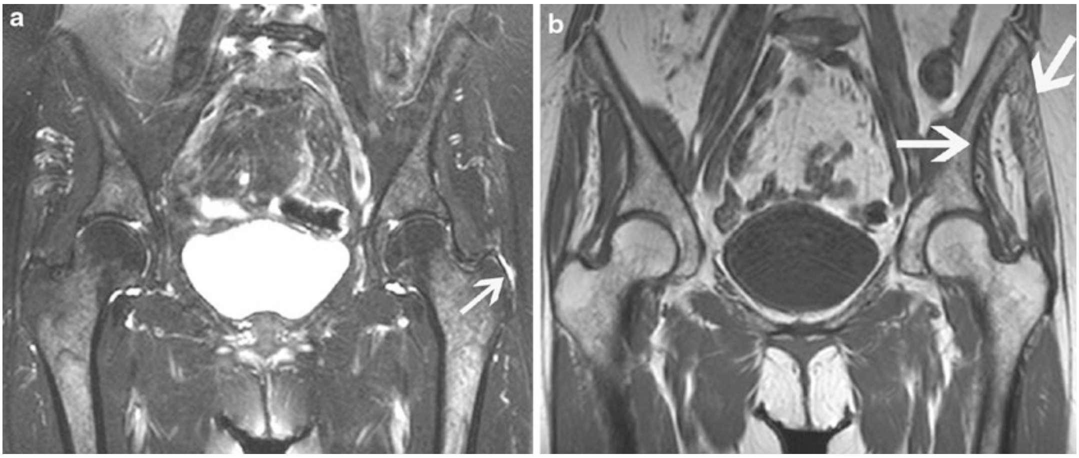


Fig. 48 Fat-suppressed coronal T2-weighted image (**a**) from pelvis MRI shows a full-thickness tear of the distal left gluteus medius tendon at the lateral trochanteric facet with fluid filling the tear site (*arrow*), compared to the normal contralateral tendon. Nonfat-suppressed coronal T1-weighted image (**b**) from this exam shows extensive linear high T1 signal areas within the left gluteus medius muscle and slightly less bulk as compared to the contralateral muscle (*superficial arrow*), reflecting moderate fatty atrophy; there is less extensive fat infiltration but more prominent atrophy of the left gluteus minimus muscle (*deep arrow*)

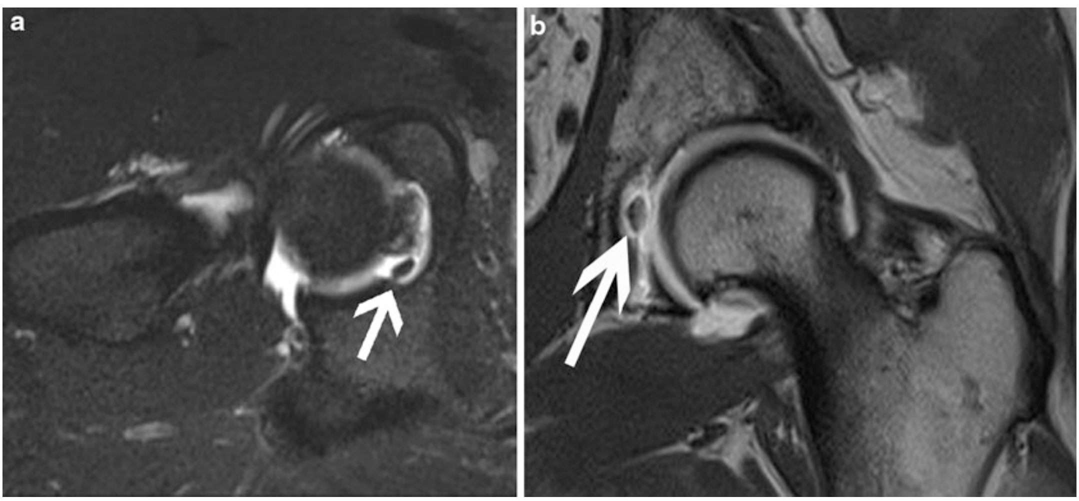


Fig. 49 Post-arthrogram fat-suppressed oblique axial image (**a**) and nonfat-suppressed coronal T1-weighted image (**b**) show an osseous body in the cotyloid fossa (*arrows*)

Rarely, marked distention of the bursa is seen, most often in the acute setting after gluteal tendon tear. It is important to note that the finding of bursal distention (i.e., "bursitis" based on MRI) has low specificity for pain. In a study of 256 hips imaged at MRI, the presence of trochanteric bursal distention, regardless of size, did not distinguish those with from those without trochanteric pain [60].

Postoperative Imaging

There are a multitude of causes of hip arthroscopy failure, including undercorrection of FAI morphology, labral tears, chondral defects, adhesions, instability, and iliopsoas pathology [61–63]. Intra-articular bodies may also be present (Fig. 49) [64]. The most frequent cause is FAI undercorrection [62, 63]. While MRI can detect undercorrection, it is definitely not needed as radiographs usually suffice.

The postoperative labrum is difficult to image frequently due to invariable irregular signal and variable irregular shape. The obliquity of the usual postoperative site in the anterosuperior quadrant only makes assessment more difficult. In a retrospective review of 70 revision arthroscopies, MRA had a low (39 %) negative predictive value in the diagnsosis of labral tear [64].

After prior resection, a tear of the blunted labrum can be diagnosed when fluid signal is seen extending into the articular side of the labrum or the chondrolabral junction, the labrum is thickened and irregular in contour, or when there is a new paralabral cyst [65]. Obviously, a tear is present if there is frank displacement of labral tissue. A new tear can be seen at a new site as well, not infrequently adjacent to the former surgical site (Fig. 50). As in all postoperative joints in which fibrocartilage has been resected or repaired, accuracy should be improved if comparison can be made with the preoperative MRI. To the author's knowledge, no MRI study has been performed evaluating labral repairs for failed repair or re-tear; presumably, diagnosis would be analogous to the meniscus (fluid imbibition/uptake into the site indicative of failed repair or re-tear).

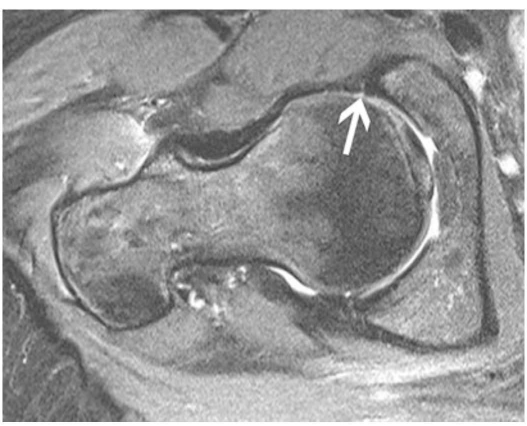


Fig. 50 Fat-suppressed oblique axial proton density-weighted image shows a radial split in the anterior labrum at 3 o'clock (*arrow*) adjacent to caudal aspect of previously repaired anterosuperior labral tear (not shown)

Intra-articular adhesions can be a cause of persistent pain and decreased range of motion after surgery. These can be seen as thin linear or irregular bands extending from the capsule to the neck at the site of osteochondroplasty or between the capsule and postoperative labrum; additionally, they may be so extensive in the paralabral recess as to obscure it (Figs. 51 and 52) [66, 67]. Note that as adhesions likely always occur to some degree after surgery, their presence on MRI may or may not be of significance [61]. Also note that MRA instead of conventional MRI should be considered as MRA may provide better evaluation due to capsular distention.

Capsular defects, either due to failed repair or lack of repair of the capsulotomy, can be easily identified after surgery on MRI. There is a focal or grossly wide gap in the iliofemoral

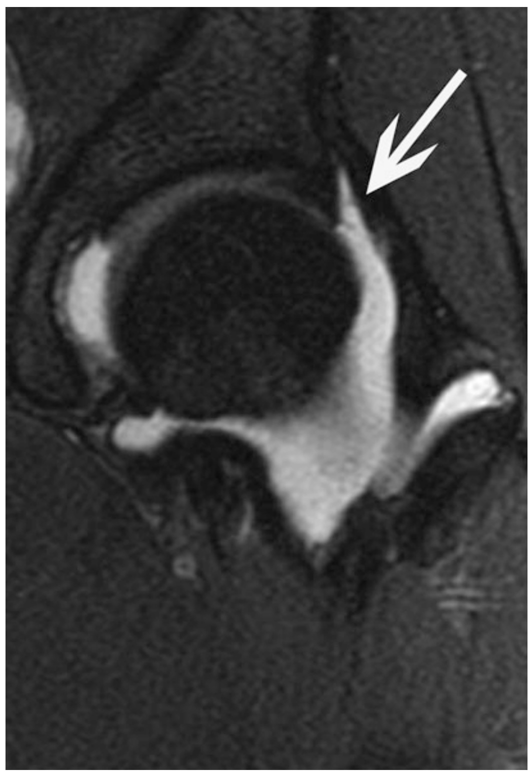


Fig. 51 Post-arthrogram fat-suppressed coronal T2-weighted image shows thin linear bands in the perilabral recess, extending from the iliofemoral ligament to the labrum (*arrow*), in this patient with pain after labral repair

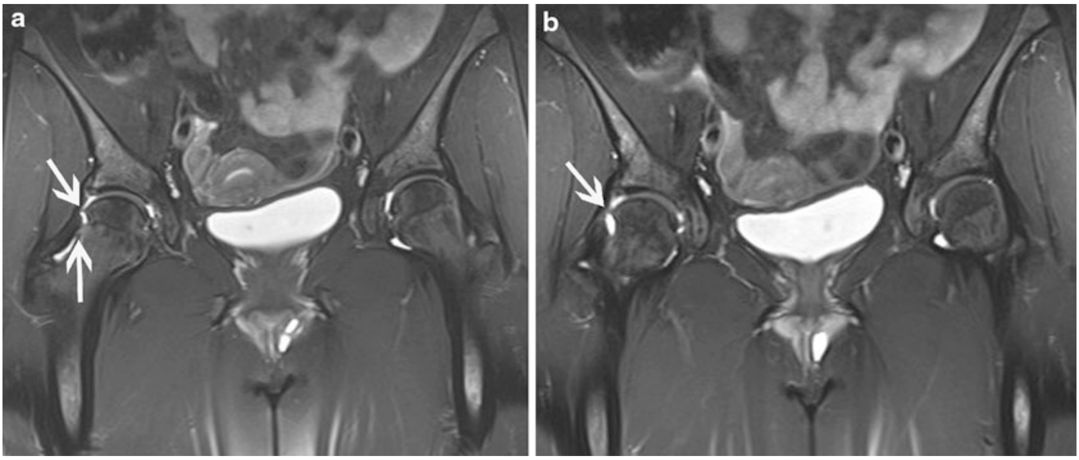


Fig. 52 (**a**, **b**) Fat-suppressed coronal T2-weighted images after femoral osteochondroplasty and rim trimming with labral refixation show short, slightly thick intermediate-signal bands extending from the ligament to the osteochondroplasty site (*arrows*), reflecting adhesions

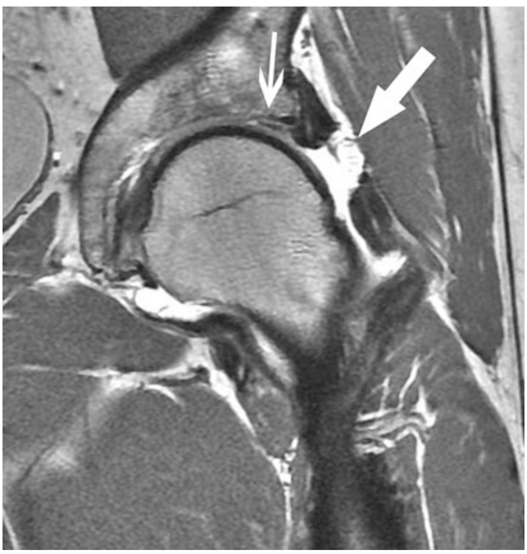


Fig. 53 Post-arthrogram coronal proton density-weighted image prior to revision surgery reveals a large defect in the iliofemoral ligament (*thick arrow*). Note the chondral delamination over the lateral dome (*thin arrow*)

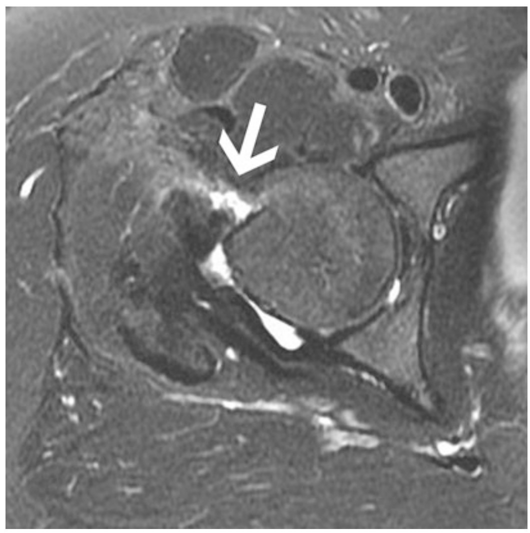


Fig. 54 Fat-suppressed axial T2-weighted image without intra-articular contrast at 3 months following capsular repair at the conclusion of arthroscopy. There is a defect in the iliofemoral ligament through which synovial fluid extends (*arrow*), reflecting failed repair or possibly interval tear of repaired capsule

ligament, noted best on oblique axial and coronal images (Figs. 53 and 54). These defects can predispose to instability, subluxation, and dislocation [68, 69].

As in all cases, chondral defects can be seen MRI after surgery. Iatrogenic chondral trauma during surgery can occur, particularly over the head [70]. Thus, particular scrutiny of the cartilage over the head should be made in postsurgical patients.

Insufficiency fractures after femoral osteochondroplasty are a rare cause of pain after surgery [71]. While radiographs are often sufficient for diagnosis, MRI has much better sensitivity and is recommended if a fracture is suspected in the presence of negative radiographs.

Although not usually a problem after FAI surgery, as stated previously, any metallic structure can cause significant distortion/misregistration and artifact on MRI, potentially obscuring much of the joint. Even tiny metallic foci can cause a significant artifact. This is particularly exaggerated on fat-suppressed sequences. Thus, if metallic structures are known to exist (e.g., fixation screws, anchors), it is strongly recommended that the radiologist is aware so that appropriate sequences can be arranged. Nonfat-suppressed fluid-sensitive sequences in multiple planes are recommended in these situations.

Miscellaneous

Synovial Processes

Synovitis (i.e., abnormal synovial proliferation) is associated with joint pain. Hip synovitis, whether preoperatively or postoperatively, is associated with decreased hip function scores and arthroscopic outcomes [72, 73]. The evaluation for synovitis traditionally has been performed after the intravenous administration of gadolinium, the degree and extent of synovial enhancement corresponding to synovial proliferation/synovitis. However, the degree of synovitis can also be assessed on noncontrast imaging. Synovial proliferation, whether using noncontrast imaging or distending the joint with fluid with arthrography, is present when there is abnormal signal within the joint fluid. Synovitis can be thin and irregular or relatively thick and conglomerate; it can be focal or, more frequently, diffuse (Figs. 55 and 56). While more easily identified in the presence of a joint effusion or after arthrography with related capsular distention, it can be detected when a normal volume of synovial fluid is present. Because of the tight confines of the central compartment, it is more readily identified in the peripheral compartment. Edema (i.e., high signal on fluid-sensitive sequences) within the cotyloid fossa on noncontrast imaging can be a sign of synovitis as well.

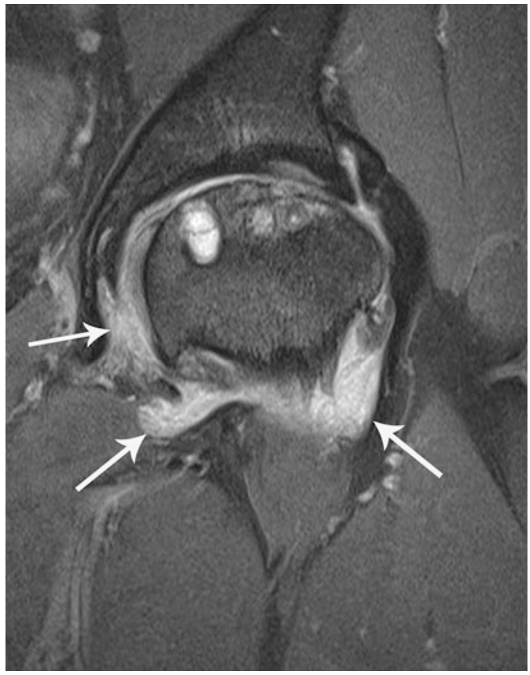


Fig. 55 Fat-suppressed coronal proton density-weighted image in a patient with obvious extensive exposed bone over the superior head shows extensive thick, elongated, high-signal foci within the synovial fluid (*arrows*), consistent with marked synovitis

The presence of synovitis is frequently a result of synoviocyte upregulation due to chondral loss. In an arthroscopic evaluation of 81 patients, the degree of synovitis had a linear correlation with the extent of synovitis [73]. While extensive synovitis is usually noted in the presence of obvious, extensive chondral loss, the presence of synovitis on MRI should elicit scrutiny for more subtle chondral pathology if overt evidence is not present.

Other conditions to be considered when non-mass-like synovitis is present include systemic inflammatory arthritides (e.g., rheumatoid arthritis) as well as crystalline arthropathies (e.g., gout). When mass-like intra-articular signal exists, the top two differential considerations are synovial chondromatosis and pigmented villonodular synovitis (PVNS).

Synovial osteochondromatosis is easily diagnosed if intra-articular mineralized bodies of similar size and shape are present. Often, osseous

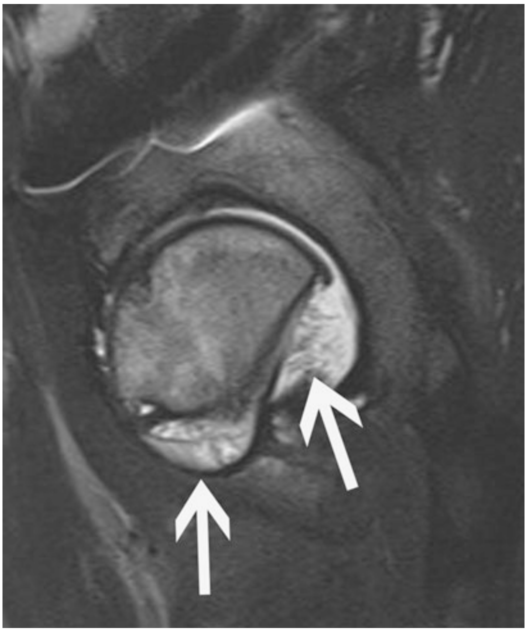


Fig. 56 Post-arthrogram fat-suppressed sagittal T2-weighted image after remote periacetabular osteotomy shows extensive linear intermediate- and low-signal foci within the synovial fluid (*arrows*), reflecting extensive synovitis

erosions are present. (Of course, if on radiographs frank intra-articular osseous bodies are present and are generally the same shape and size, with or without osseous erosion, a confident diagnosis of synovial osteochondromatosis can be made.) However, mineralization may not be present, and osseous erosions may be subtle or absent. Thus, MRI can play an important role in the diagnosis. On MRI, synovial chondromatosis is usually seen as focal or diffuse small bodies which are often heterogeneous in signal but predominantly intermediate in signal on T1-weighted sequences and intermediate to high in signal on fluid-sensitive sequences (i.e., largely the same signal as cartilage). In some cases, conglomerate, mass-like foci may be seen, sometimes diffusely filling the joint. Osseous erosions on either or both sides of the joint are more common in cases of extensive synovial chondromatosis.

PVNS manifests as focal or diffuse intra-articular mass-like signal. PVNS is typically heterogeneous in signal, predominantly low or intermediate in signal on T1-weighted sequences and variable (usually low or intermediate) on fluid-sensitive sequences. The extent of hemosiderin in pvns is generally proportional to the extent and degree of low signal. Due to hemosiderin (variable in extent) present in PVNS, gradient-echo sequences can be extremely helpful in the diagnosis; due to dephasing and magnetic susceptibility artifact associated with hemosiderin, hemosiderin-containing FOCI within PVNS will be more prominent and "bloom" on this sequence, clinching the diagnosis (Fig. 57). In addition, extensive PVNS not infrequently has extracapsular extension, the location and extent of which as well as potential involvement of adjacent neurovascular structures can be defined.

Like all synovial processes, synovial chondromatosis and PVNS can infrequently involve the hip bursa and tendon sheaths. Involvement of these structures is rare compared to intra-articular involvement of the hip.

Avascular Necrosis

Avascular necrosis (AVN) of the femoral head is an important cause of hip pain in young and middle-aged adults that frequently leads to subchondral collapse and osteoarthrosis if left untreated. There are a vast array of etiologies of AVN, including corticosteroids and alcohol.

The diagnosis of (AVN) of the femoral head is readily made with MRI. Circumscribed, irregularly shaped subchondral marrow is present on both T1-weighted and fluid-sensitive sequences. On T2-weighted sequences, classically a so-called double-line sign is seen where the margin of osteonecrosis has a high-signal inner portion adjacent to a low-signal outer margin, generally thought to reflect the interface between reparative and sclerotic trabeculae (i.e., creeping substitution). On other fluid-sensitive sequences (e.g., fat-suppressed T2-weighted sequences), often the signal is SIMPLY high about the margin [74]. The size and location of AVN varies. Additionally, multiple foci of AVN may be present. The extent of involvement of the weight-bearing surface and location is generally proportional to prognosis [75]. Collapse of the subchondral plate

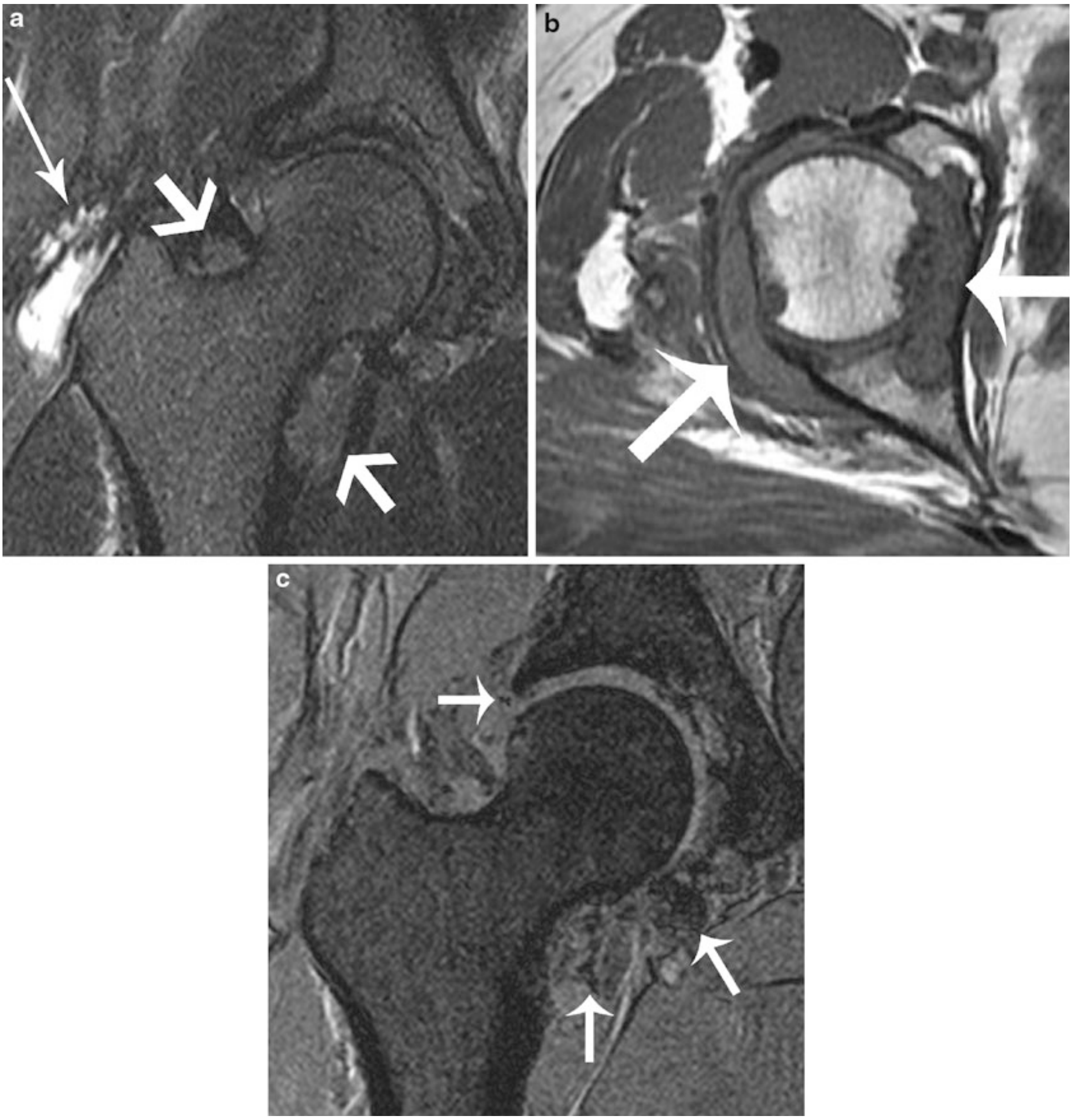


Fig. 57 Multiple images from a patient with PVNS, including fat-suppressed coronal T2-weighted image (**a**), axial T1-weighted image (**b**), and coronal gradient-echo image (**c**). There is extensive abnormal heterogeneously intermediate signal filling the joint in (**a**) (*shorter*, *thicker arrows*). In (**b**), there is also extensive abnormal intermediate signal filling the joint (*arrows*) with multiple erosions, extensively involving the cotyloid fossa and adjacent head. On gradient-echo sequences, prominent low-signal foci will invariably be present (*arrows* in **c**), reflecting hemosiderin. Incidentally, note the full-thickness gluteus medius tendon tear in (**a**) (*thin arrow*)

carries a poor prognosis. There are many classification systems of staging femoral head AVN, including the Association Research Circulation Osseous (ARCO) staging system.

MRI is usually reserved in the diagnosis and assessment of early- and mid-stage AVN as radiographs are clearly diagnostic of late-stage disease. On MRI, AVN may have a normal marrow signal, an edema-like signal, or fibrotic signal (low on all sequences), or a mixture. AVN that is diffusely low in signal on all sequences reflects completely devitalized bone. The extent of femoral head surface involvement can be made visually or directly with measurement on coronal and sagittal sequences. MRI has been found to add more accurate staging evaluation than radiographs in ARCO stages II (positive radiographic and MRI findings without subchondral fracture) and III (subchondral fracture and collapse with generally preserved head contour) [76]. Occasionally, small foci of AVN can be found, often located in a non-weight-bearing portion of the head, so-called “minimal” AVN; these small foci of AVN are thought to be due to a focal segmental ischemic insult and are not at risk of subchondral collapse [77].

The extent and even the presence of subchondral collapse can be difficult on pelvis MRI due to the large field of imaging. In the author’s experience, an extensive marrow edema pattern in and about the AVN, usually extending into the femoral neck, usually indicates a recent subchondral fracture. Subchondral fractures are best noted on fluid-sensitive sequences; look for thin, linear, or irregular fluid signal within the osteonecrotic area which may extend into and through the subchondral plate (Fig. 58). Due to the curvature of the head, subchondral fracture visualization can be difficult, particularly anterosuperiorly or posterosuperiorly, without the presence of a sagittal sequence. In this setting, the fracture may not be definitively visualized on either coronal or axial images due to volume averaging, particularly when it is not located around 12, 3, 6, or 9 o’clock. Regardless, in the setting of an extensive marrow edema pattern in the head and neck, particularly with recent onset or increase in pain, a subchondral fracture should be suspected.

Idiopathic Transient Osteoporosis of the Hip, Subchondral Fractures, and Stress Fractures

Idiopathic transient osteoporosis of the hip (ITOH) is a peculiar entity with unclear causation, potentially due to a transient reversible vasomotor process. It classically affects middle-aged males and females in the third trimester of pregnancy. It usually presents with a gradual onset of atraumatic hip pain, increasing in severity over several months. The pain then plateaus and gradually subsides, the entire duration usually lasting less than one year. On MRI, there is usually a diffuse, sometimes patchy, marrow edema pattern throughout the femoral head, extending distally into the neck. A joint effusion is usually present. Sometimes there is focal sparing of the subchondral plate from the marrow edema pattern [78]. There is classically no subchondral hypointense line to suggest subchondral fracture. There is no circumscribed subchondral bone as in AVN. Additionally, there is usually no subchondral marrow edema pattern in the dome, distinguishing ITOH from the typical findings of other intra-articular processes (e.g., septic arthritis).

Similar symptoms and MRI findings can occur in some patients affected by ITOH in the contralateral hip or either lower extremity (knee, ankle, foot). Occasionally, involvement of a lower extremity joint precedes that of hip involvement [79]. This constellation of findings carries the diagnosis of regional migratory osteoporosis. Regional migratory osteoporosis more frequently occurs in men, and the symptoms and findings in the subsequently affected joint occur within the first year after the onset of symptoms in the index joint [79].

It is plausible that some cases of ITOH are actually not idiopathic but due to a subchondral fracture. Subchondral fractures could be obscured by the extensive marrow edema pattern in ITOH given that the fracture signal is often similar to that of edema on both T1-weighted and fluid-sensitive sequences. In a study of 45 patients with early-stage femoral head AVN, CT had a higher sensitivity in the detection of subchondral fractures than MRI [80]. On occasion, a subchondral line or focal thickening of the subchondral plate, often very small and subtle, may be seen on MRI, reflecting a subchondral fracture. This leads to confusion as to whether the findings are due to ITOH or, instead, due to a subchondral fracture. In these cases, it is usually impossible to tell whether the subchondral fracture occurred due to subchondral bone weakened by the intense hyperemia of ITOH or whether the subchondral fracture occurred first. In other

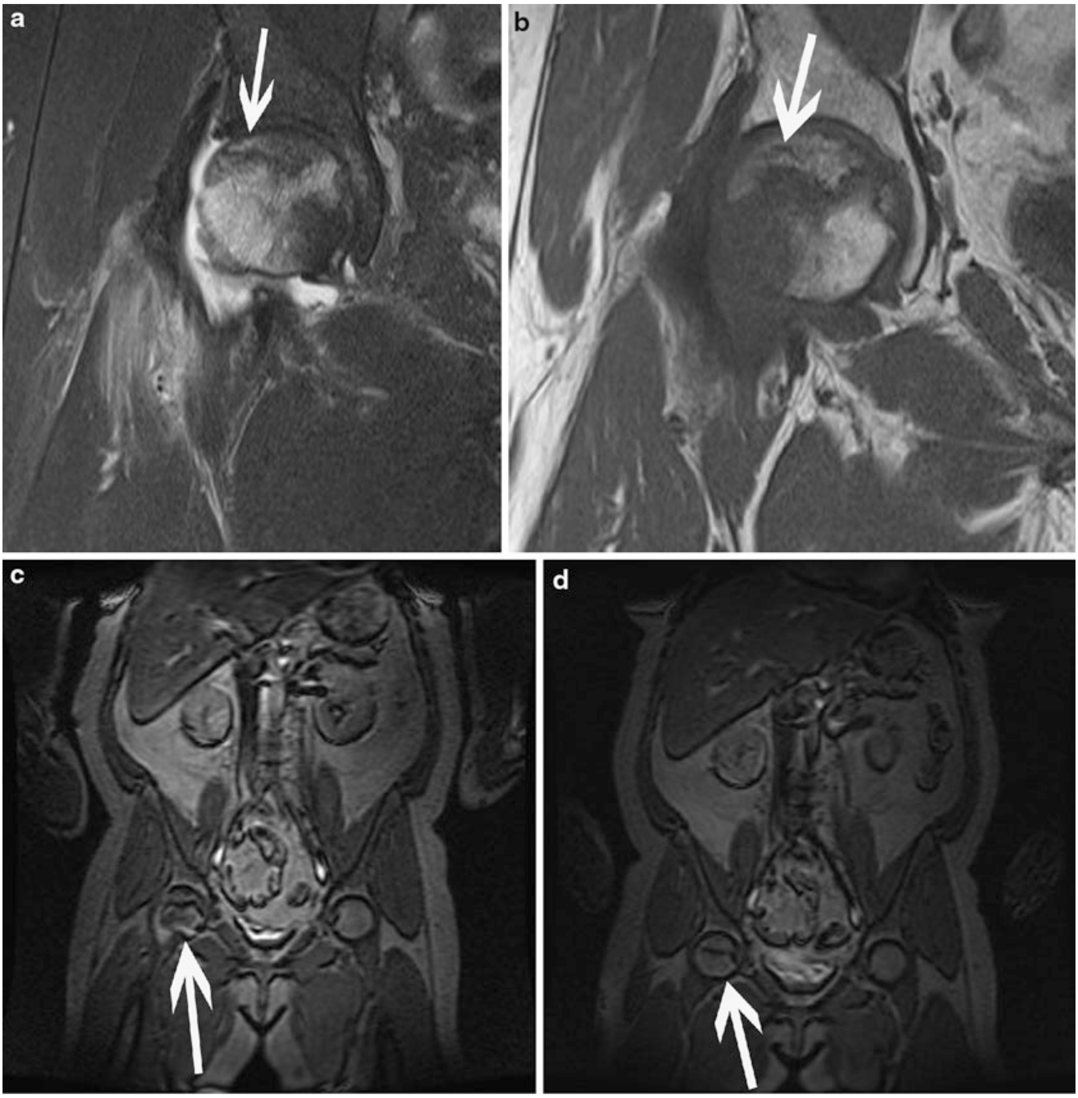


Fig. 58 Fat-suppressed coronal T2-weighted image (**a**) shows a small, roughly linear high-signal focus in the lateral aspect of AVN in the head (*arrow*), representing a subchondral fracture; note the extensive adjacent marrow edema pattern in the head and neck and the reactive joint effusion. In (**b**), the low-signal fracture line is also seen (*arrow*). Note that localizer sequences obtained on other MRI exams, most commonly lumbar spine MRIs, will include the pelvis and frequently the proximal femora. In (**c**) on a lumbar spine MRI obtained 1 month prior to the hip MRI in (**a**) and (**b**), circumscribed low signal, reflecting AVN, can be seen in the head (*arrow*); heterogeneously low signal in the osteonecrotic portion and in the adjacent head reflects the marrow edema pattern as seen in (**a**) and (**b**). Two years earlier, the AVN could also be seen (*arrow* in **d**); note that no low signal is present in the surrounding head and neck to suggest subchondral fracture

words, it may be impossible to tell which came first: the chicken or the egg?

This leads to a discussion of subchondral fractures of the femoral head. They occur in both young and old adults. The characteristic patient is an obese postmenopausal female with osteoporosis. However, subchondral fractures can occur in young adults (Fig. 59). On MRI, these fractures are thin, curvilinear, or linear, usually very close to and paralleling the subchondral plate. The

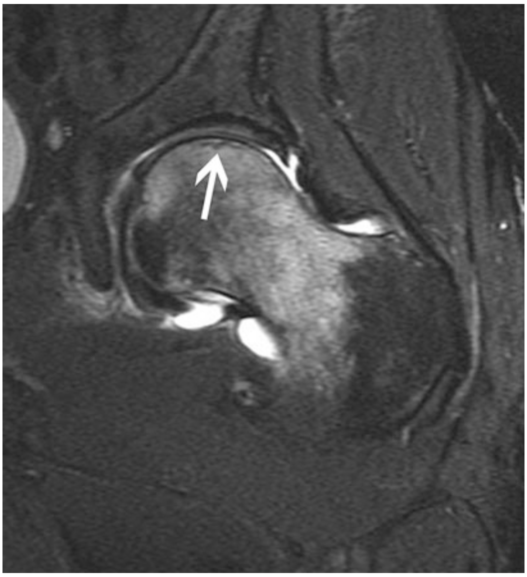


Fig. 59 Fat-suppressed coronal T2-weighted image shows a subchondral fracture line (*arrow*) with an associated extensive marrow edema pattern, extending distally to the intertrochanteric region. Note the associated reactive joint effusion

Fig. 60 Fat-suppressed coronal T2-weighted image shows a low-signal line in the medial basicervical neck (*arrow*), a typical compression-side stress fracture. There is an associated surrounding marrow edema pattern and adjacent periosteal edema

fracture may be best seen on T1-weighted sequences as thin intermediate to low signal. On fluid-sensitive sequences, they can be instead better visualized as thin low signal amidst an invariable extensive surrounding marrow pattern, sometimes extending distally to the intertrochanteric region.

The usual "treatment" for ITOH and subchondral fractures usually is protected weight-bearing and symptomatic treatment with nonsteroidal anti-inflammatory medications (NSAIDs). The majority of cases of ITOH as well as subchondral fractures of the femoral head in the young or middle-aged adult resolve without sequelae. However, the outcome of elderly patients with subchondral fractures is more guarded as subchondral collapse and progressive osteoarthrosis can occur [81].

Stress fractures of the femoral neck can be extremely subtle on radiographs. MRI has much better sensitivity in their detection and is the imaging modality of choice. The typical findings are an eccentric marrow edema pattern in the medial basicervical femoral neck, the site of the overwhelmingly more frequent compression-side fractures as opposed to the superolaterally located tension-side fractures. Close scrutiny should be made for a hypointense line amidst the edema which may be very small; at times it is better visualized on fluid-sensitive sequences, while at other it is better seen on T1-weighted sequences (Fig. 60). There is almost always adjacent periosteal and soft tissue edema. If a fracture line is not visualized at MRI despite close scrutiny, stress reaction is the diagnosis. Obviously, patients with stress reaction are at risk for progression to fracture, and restricted or protected weight-bearing should be instituted.

Bone Lesions

It is beyond the scope of this chapter to discuss the myriad bone lesions that can occur in the proximal femur or dome. Nonetheless, two lesions, one commonplace and the other much less frequent,

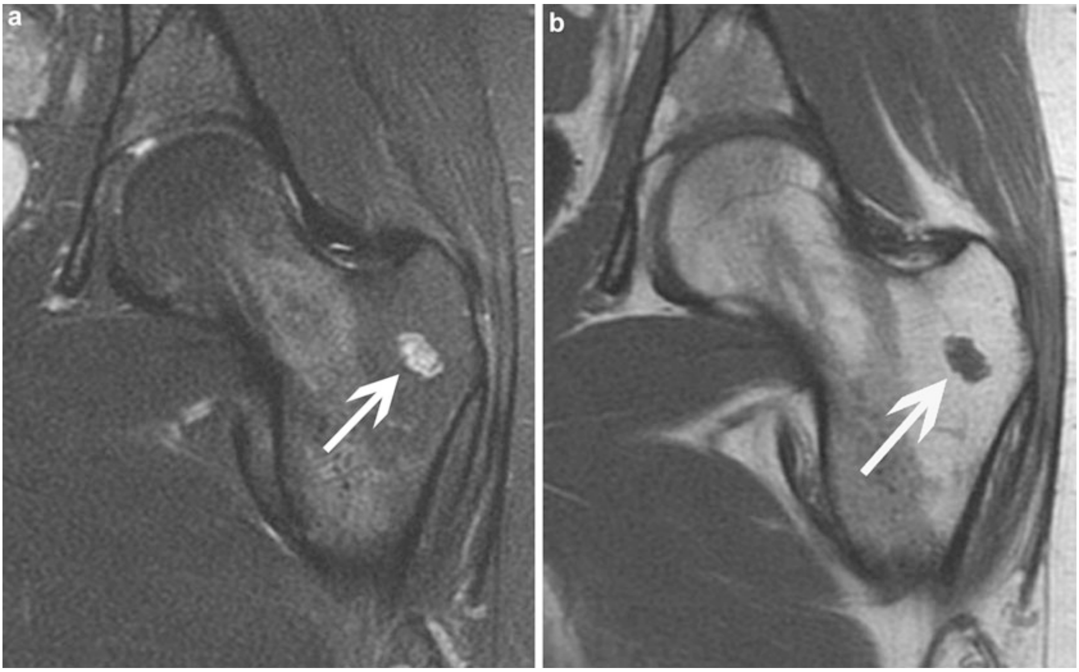


Fig. 61 Coronal fat-suppressed T2-weighted and nonfat-suppressed T1-weighted images (**a** and **b**, respectively) show a small, well-circumscribed, multilobulated lesion in the greater trochanter (*large arrows*) with mildly heterogeneous signal, predominantly high signal in (**a**) and intermediate signal in (**b**), representing an enchondroma

will be discussed. Benign chondroid lesions (e.g., enchondromas) are frequently present in the proximal femur (Fig. 61). Chondroid lesions are mildly heterogeneous in signal, predominantly intermediate in signal on T1-weighted sequences, and predominantly high in signal on fluid-sensitive sequences. They are well defined without a surrounding marrow edema pattern. They typically have microlobular margins and thin internal areas of low signal, particularly noticeable on fluid-sensitive sequences. As opposed to the typical benign chondroid lesions, chondrosarcomas characteristically present with pain; they tend to be larger and may have associated prominent endosteal scalloping, periosteal reaction, and adjacent soft tissue extension.

Osteoid osteomas are benign lesions that may be subperiosteal, intracortical, or intramedullary in location. They are usually found in patients in the second or third decades; in one series of 255 cases, the ages ranged from 19 months to 56 years [82, 83]. They have a distinct male predilection. The classic history of these lesions is night pain relieved with NSAIDs; however, this is nonspecific as many other lesions can present similarly.

Intra-articular lesions are frequently more difficult to diagnose at imaging, including MRI. When intra-articular, they more frequently present with a joint effusion, joint tenderness, swelling, and reduced range of motion; pain is less frequently present at night [82, 84]. On MRI, clues to the diagnosis include a prominent marrow edema pattern in an unusual location (e.g., not subchondral), joint effusion, synovitis, and often adjacent soft tissue edema. Note that there is usually minimal to no cortical or juxtacortical thickening associated with intra-articular osteoid osteomas as there is with extra-articular juxtacortical lesions. When the aforementioned findings are seen, close scrutiny should be made for a small well-circumscribed area of signal in or at the margin of the intra-articular edema, reflecting the nidus (Fig. 62). The nidus is usually

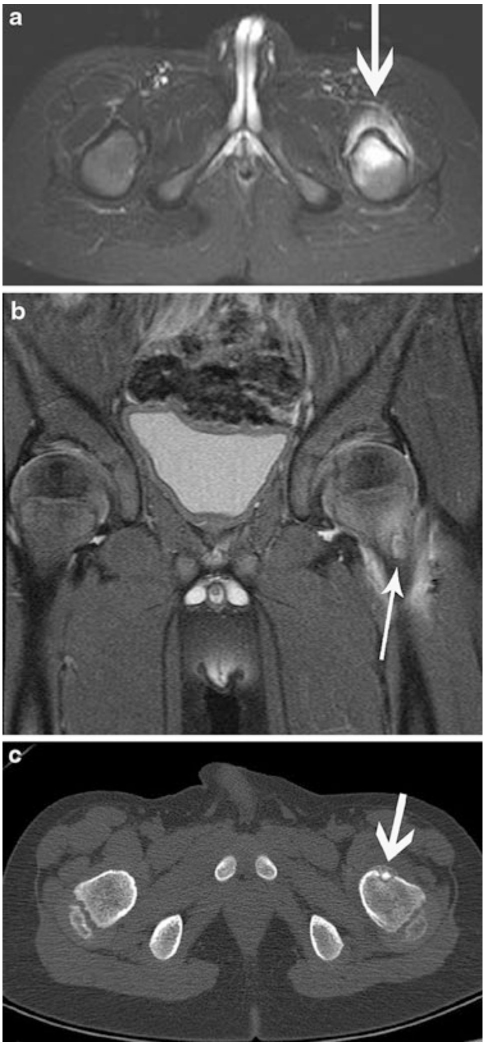


Fig. 62 Fat-suppressed axial T2-weighted image (**a**) shows an extensive marrow edema pattern in the anterior intertrochanteric femur with adjacent marked subperiosteal edema. Post-contrast, fat-suppressed coronal T1-weighted image (**b**) shows a small heterogeneous ovoid structure in the anterior intertrochanteric region (*arrow*) with surrounding extensive marrow enhancement, adjacent soft tissue enhancement, and a small joint effusion. Axial noncontrast CT (**c**) reveals a small intracortical focus of dense mineralization with surrounding thin lucency in the location of the ovoid structure in (**b**) with adjacent periosteal and subperiosteal mineralization. In this skeletally immature patient, the findings on MRI and confirmed with CT are extremely likely to represent an osteoid osteoma. Given the findings, particularly the lack of adjacent medullary sclerosis on CT, a Brodie's abscess with sequestrum is very unlikely. The patient's clinical history and negative laboratory findings corroborated the top differential consideration on imaging, an osteoid osteoma

round or ovoid, low or intermediate in signal on T1-weighted sequences, and variable in signal on fluid-sensitive sequences [84]. On high-resolution, small field of imaging studies, variable internal low signal reflecting mineralization of the nidus can be identified. However, on large field of view imaging (e.g., whole-pelvis MRI), detection of the nidus is often more difficult, particularly for juxta-cortical (i.e. subperiosteal or intracortical) osteoid osteomas; particularly close scrutiny on these large filed of imaging studies should be made. If a typical nidus is visualized, the diagnosis of an osteoid osteoma can be made. If a focal juxtacortical or intracortical signal abnormality is questionable or not definitely visualized, CT is recommended as the next step. Thin-section CT usually clearly will show the lucent nidus with variable internal calcification if an osteoid osteoma is present.

As a Brodie's abscess and osteoid osteoma can have a similar presentation and MR and CT findings, there should be a careful evaluation of the laboratory findings. Contrast-enhanced MRI can often distinguish between the avidly enhancing, hypervascular nidus and rim-enhancing Brodie's abscess. Additionally, the nidus typically has increased uptake on a nuclear medicine bone scan and lesser uptake about its periphery (the so-called double-density sign), as opposed to a Brodie's abscess.

The Iliopsoas Tendon and Bursa

The iliopsoas tendon is a well-known cause of hip snapping. Most imaging studies have involved sonography with dynamic imaging detailing the abrupt transition of the tendon, most commonly as it transits medially across the iliopectineal eminence and about the iliacus muscle [85–87]. The author is not aware of any specific findings on MRI.

On MRI, axial and coronal images provide excellent visualization of the tendon. The normal tendon is homogeneously low in signal. Degenerated tendons have intermediate signal and may be enlarged. Minimal peritendinous edema is commonplace and nonspecific. Iliopsoas tendon tears are not subtle; there is disruption and usually retraction of the tendon with surrounding soft tissue edema/hemorrhage. Associated avulsion fractures of the lesser trochanter are usually easily detected on MRI; small fractures may be difficult to detect if small and imaged with a large field of view (i.e., pelvis MRI). In the elderly, due to the high association of avulsion fractures with underlying neoplastic involvement of the trochanteric marrow, the assessment of trochanteric marrow signal is critical.

The iliopsoas bursa communicates normally with the hip joint in approximately 15 % of patients. It is not uncommon to see minimal distal peritendinous edema. Bursal distention is usually mild or moderate and seen usually most prominently anterior to the iliopectineal eminence. Rarely, marked bursal distention can be seen, most often in patients with rheumatoid arthritis. The bursal distention typically is located along the medial margin of the tendon, but may instead saddlebag around the tendon, centered posteriorly.

Like all bursae, frank synovial thickening can be seen on MRI in the iliopsoas bursa. The synovial thickening most often manifests as thin and irregular low signal amidst the bursal fluid. Occasionally, the bursae can contain more amorphous heterogeneous signal. Like all synovial fluid, whether in joints, bursae, or tendons, this heterogeneous signal most often signifies proteinaceous contents. However, blood products within a bursa can be seen; this should be suspected when high signal on T1-weighted sequences is present, particularly in the setting of a recent injury and local pain (Fig. 63).

The Hamstring Tendons

The hamstring tendons, except for the short head biceps femoris tendon, have a common origin at the ischial tuberosity. The semimembranosus tendon originates from the superolateral facet of the ischial tuberosity. The long head biceps femoris and semitendinosus tendons originate as a conjoint tendon from the inferomedial facet of the ischial tuberosity. Translating this to MRI, on axial sequences, the conjoint tendon arises more posteromedially on the tuberosity than the longer anterolaterally located semimembranosus tendon. On coronal sequences, the conjoint tendon is located more inferomedially than the more superolaterally located semimembranosus tendon at the tuberosity.

All three standard imaging planes on MRI are helpful in the evaluation of the tendons; typically the tendons are imaged in only the axial and coronal planes, however. The axial plane is most useful in their evaluation given that they are seen in cross section in this plane. However, the craniocaudal extent of abnormality is usually best assessed in the coronal plane. The evaluation for degeneration and tears of the proximal tendons is exactly as described for the gluteal tendons (Figs. 64 and 65). While there is significant overlap between asymptomatic and symptomatic hamstring tendinosis on MRI, increased size of the tendons, adjacent soft tissue edema, and a marrow edema pattern in the ischial tuberosity are more frequent in symptomatic patients [88].

There are some important differences between the proximal hamstring and gluteal tendons. Often the torn tendon(s) is retracted, the extent of which can easily be made on MRI, usually with the coronal sequence. Edema, hemorrhage, and/or a hematoma is often prominent in the setting of high-grade partial and, particularly, full-thickness tears of the hamstring tendons; this is seen as amorphous and/or well-defined high signal on fluid-sensitive sequences in the soft tissues about the site of tear. Given the proximity to the sciatic nerve, this edema, hemorrhage, and associated inflammatory change in the adjacent soft tissues can abut and sometimes encase the nerve (Fig. 66). This may lead to resultant nerve inflammation. More significantly, the resultant healing process can lead to scarring of the torn tendon(s) to the nerve, making surgery more complex and difficult if not performed early after injury. Thus, in the detection and localization of the extent of torn hamstring tendon(s) and the attendant edema/hemorrhage, MRI can play an important role in the timing of surgery.

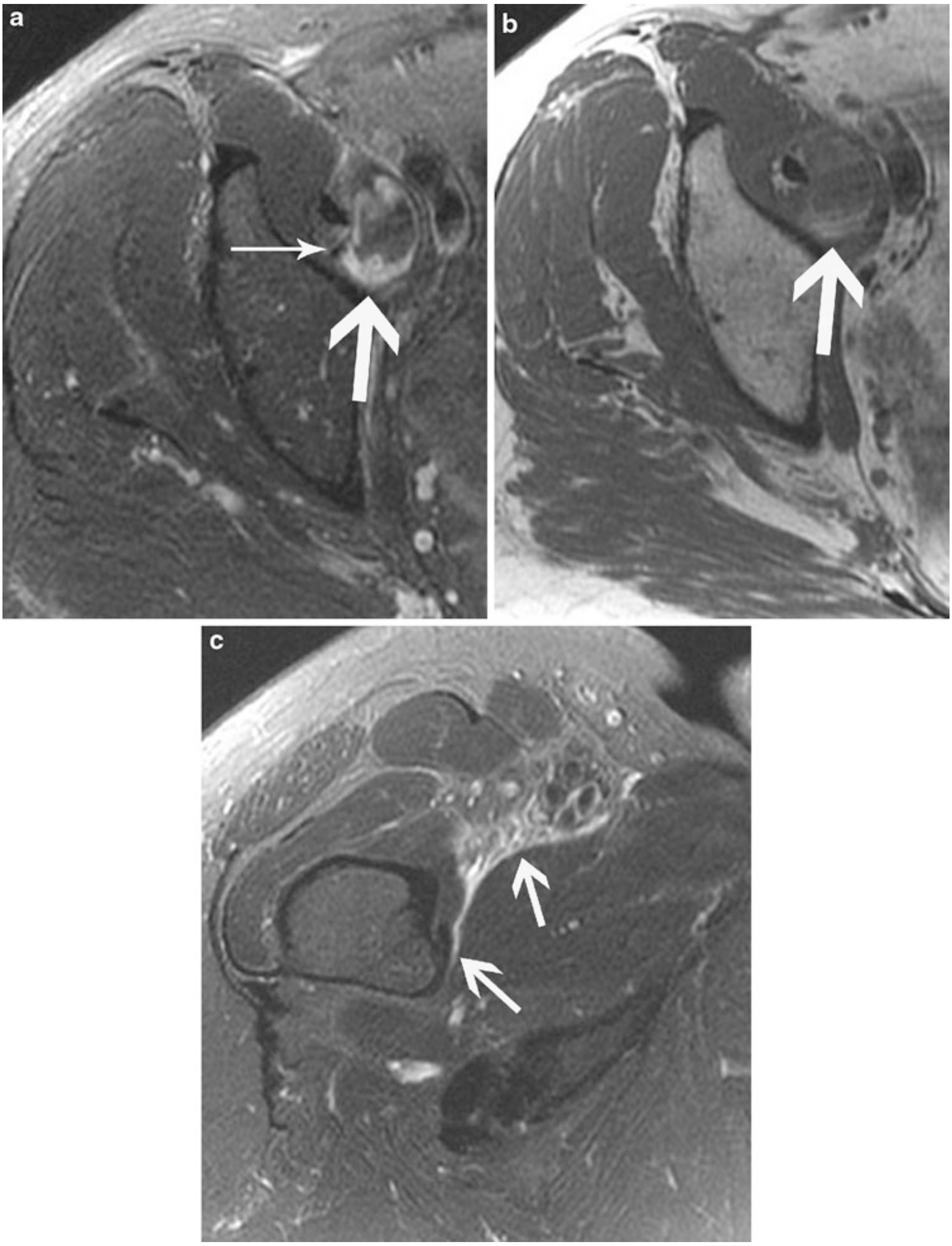


Fig. 63 Fat-suppressed axial T2-weighted image (**a**) and nonfat-suppressed T1-weighted image (**b**) show a prominent heterogeneous structure (*large arrows*) abutting the medial aspect of the iliopsoas tendon (*thin arrow*), representing a distended iliopsoas bursa; high signal on both images, in this case circumferential, is consistent with blood products/hemorrhage. Distally at the level of the insertion of the tendon on the trochanter, there is thin fluid extending from the bursa into the adjacent anterior soft tissues (*arrows*), reflecting bursal rupture (**c**). This is an unusual case of a ruptured, hemorrhagic iliopsoas bursa

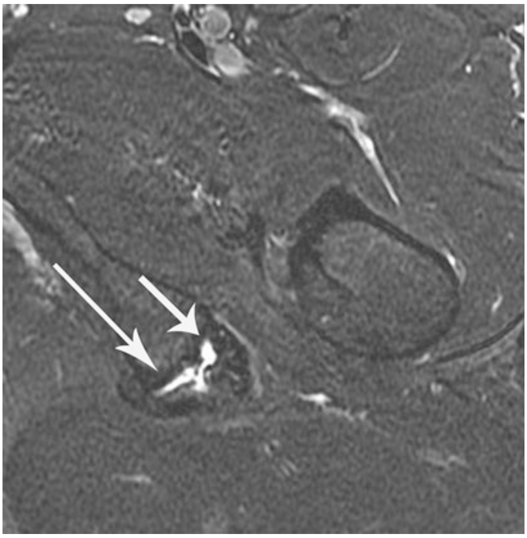


Fig. 64 Fat-suppressed axial T2-weighted image shows partial-thickness tears of both the semimembranosus tendon (*shorter arrow*) and conjoint tendon (*longer arrow*), manifested by prominent fluid interposed between the torn tendons and the tuberosity. Note heterogeneous signal in the tendons, reflecting degeneration/tendinosis

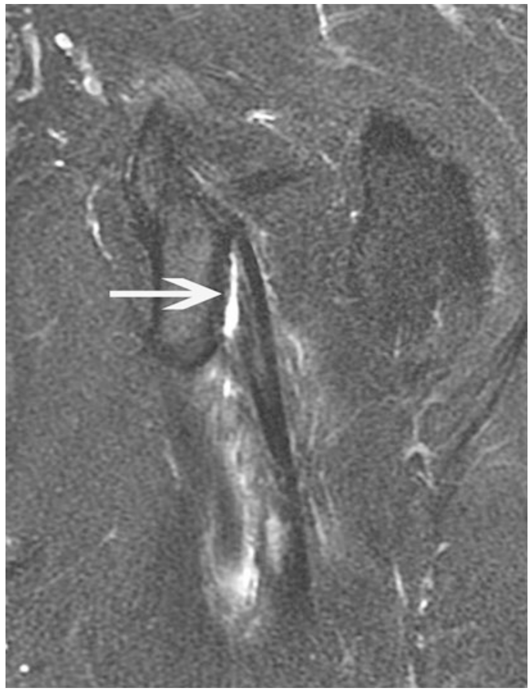


Fig. 65 Fat-suppressed coronal T2-weighted image shows a high-grade partial-thickness tear of the semimembranosus tendon, reflected by fluid interposed between the torn tendon and tuberosity (*arrow*). Note the heterogeneous signal of the tendon, representing degeneration. The surrounding prominent soft tissue edema indicates the tear occurred recently

Proximal muscle-tendon junction injury can be seen on hip and pelvis MRI, more often on pelvis MRI due to its larger field of imaging which usually extends more distally into the proximal thigh. Muscle-tendon junction injury ranges from mild strain to partial-thickness tear to full-thickness disruption. Mild strain is seen as feathery fluid about the junction, having a comblike appearance of edema with thin elongated edema oriented craniocaudally along the central tendon with fine bands of edema radiating along the muscle-tendon junction fibrils at acute angles with the central tendon. Partial-thickness tears manifest with more significant edema/hemorrhage along and about the muscle-tendon junction with thinning and irregular contour of the tendon, sometimes with a focal hematoma. Disruption is seen as frank discontinuity of the tendon, usually with significant surrounding edema/hemorrhage and sometimes also a frank hematoma.

Of note, as with all significant muscle-tendon junction or muscle tears, chronic hematomas can persist for years and simulate a cystic mass on MRI. About the hip, in the author's experience, this is most frequently seen in the rectus femoris muscle. This should be kept in mind when the diagnosis of a cystic intramuscular mass is made on MRI. Detailed patient questioning as to a prior injury in the location of the mass is recommended to prevent a misadventure.

While imperfect, MRI can have prognostic import as to time to return to sport. Some clinically diagnosed hamstring strains have no abnormal findings on MRI; these patients have a shorter time of recovery and returning to full activity than those with positive MRI findings [89]. Generally, involvement of the proximal tendon(s) indicates a more prolonged recuperation period and return to sport than those without injury to the proximal tendon(s) [89]. Likewise, the higher grade of hamstring muscle-tendon junction injury corresponds to a longer recovery period.

Although there are a number of mimickers of proximal hamstring injury, one that is seen

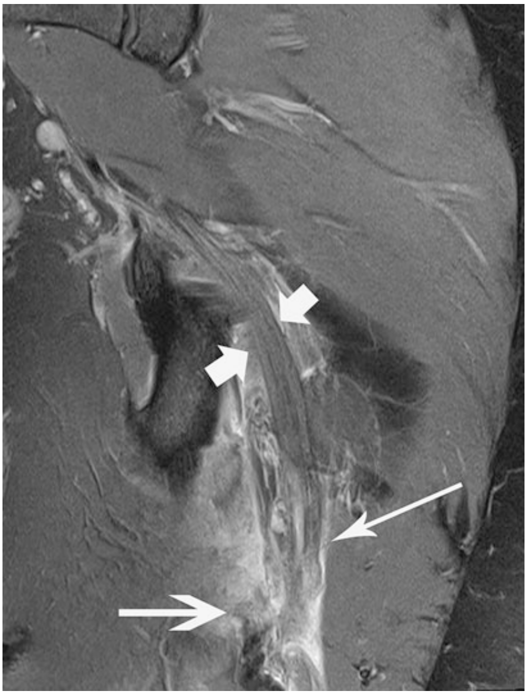


Fig. 66 Fat-suppressed coronal proton density-weighted image in a patient with a recent full-thickness tear/avulsion of the semimembranosus tendon. The torn tendon is retracted distally (*intermediate-sized arrow*). There is extensive surrounding high signal in the soft tissues (i.e., edema and hemorrhage), some of which extends around the sciatic nerve (*long*, *thin arrow*). The more proximal sciatic nerve is demarcated by the *short*, *thick arrows*

infrequently and usually affects older individuals is injury of the ischiopubic origin of the adductor magnus tendon (Fig. 67). The injured, more caudally located adductor magnus tendon arising from the ischial tuberosity can simulate a tear of the conjoint tendon on MRI if the axial images are not scrutinized.

Athletic Pubalgia

Athletic pubalgia, colloquially known as sports hernia, refers to gradual or acute pain in the inguinal region, lower abdomen, proximal adductor region, or a combination thereof. It is the result of symphyseal instability arising from chronic stresses and tearing of the insertion of the rectus abdominis tendons and/or HIP adductor tendon origins. The rectus abdominis tendons insert on the anteroinferior aspect of the pubis, in continuity with the origin of the adductor longus tendons and the symphyseal capsule and disk [90]. The adductor brevis and gracilis tendons may also have an attachment to the capsule [90].

MRI assessing the presence of athletic pubalgia should be performed with a small field of imaging and as high a signal-to-noise ratio to improve detection of pathology of both the rectus abdominis and adductor tendons. Both the axial and coronal sequences have their own strengths. Due to the muscle's coronal orientation, rectus abdominis tears are usually better seen on axial images. Adductor tendon origin tears are usually better seen on coronal images. To improve detection of both rectus abdominis and adductor tendon tears, a sagittal sequence is recommended. Not infrequently, given their close apposition, tears involve both tendons.

Adductor tendon pathology invariably involves the adductor longus tendon. Tears of the tendon are usually partial, but complete tears can occur. Partial tears are seen as thin fluid signal extending from the caudal aspect of the symphysis pubis inferolaterally into the tendon on coronal images, a so-called secondary cleft sign (Fig. 68). The partial tear may also be seen on axial images at the anteroinferior aspect of either pubic body, as well as on the sagittal images located just lateral to midline (Fig. 69). The tear may extend more posteriorly into the other adductor tendons, most frequently the adductor brevis tendon.

Rectus abdominis tendon tears are seen as thin fluid, irregular contour, or frank disruption of the tendon anterior to the pubis (Fig. 70). Tears can be seen on both axial and sagittal images, the sagittal images showing the tendon in an elongated fashion anterior to the pubis. A clue to a more chronic tear is fatty atrophy of either or both portions of the muscle; this is best detected on axial T1-weighted sequences where the affected portion(s) is relatively small in size and has a varying degree of high signal within it, reflecting fat infiltration (Fig. 71).

As mentioned earlier, given the close relationship of the rectus abdominis and adductor longus tendon at the pubis, tears frequently involve both.

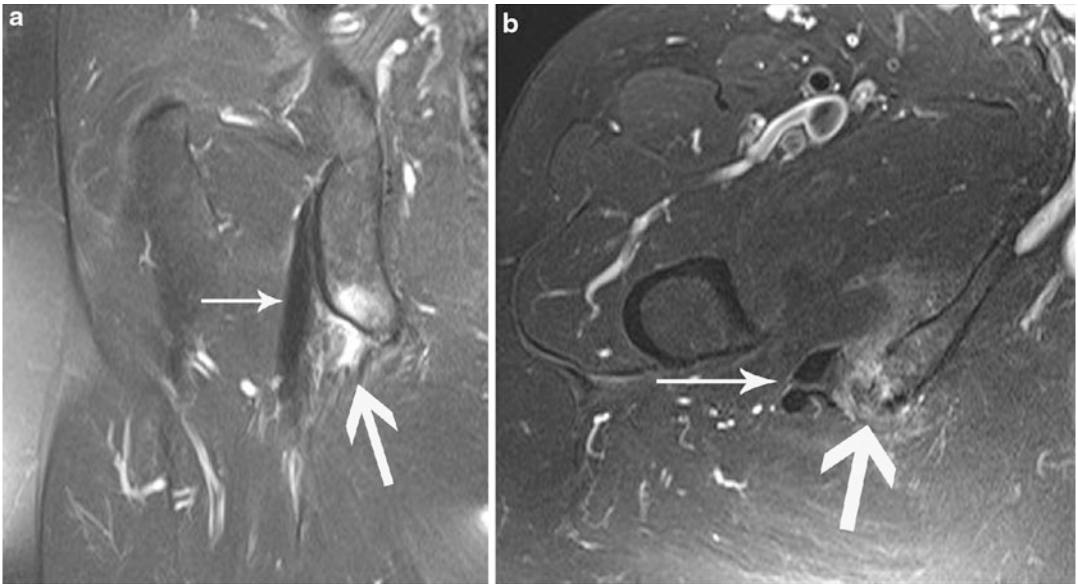


Fig. 67 Fat-suppressed coronal and axial T2-weighted images (**a** and **b**, respectively). In (**a**), there is prominent fluid signal adjacent to the tuberosity and marrow edema pattern in the tuberosity (*arrow*). Given the location of the fluid, this might be construed as a conjoint tendon tear if only the coronal images are utilized; however, it is more posterior in location than the origin of the conjoint tendon and is in the location of the origin of the adductor magnus tendon as noted on (**b**) (*thick arrow*). The normal semimembranosus and conjoint tendons are located lateral to the tear site in (**b**) (*thin arrow*)

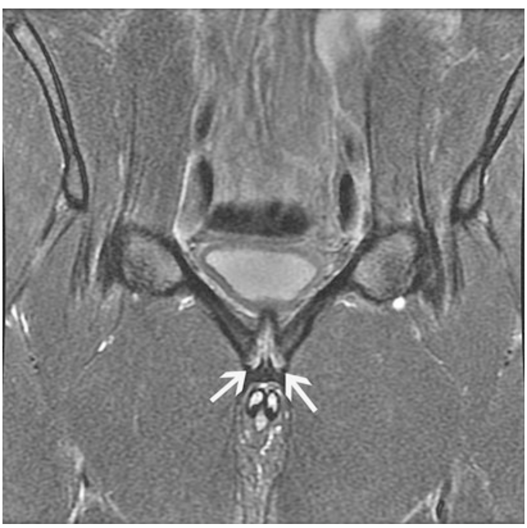


Fig. 68 Fat-suppressed coronal T2-weighted image shows thin fluid extending from the caudal symphysis into a portion of the adjacent adductor longus tendon bilaterally (*arrows*), representing the so-called secondary cleft sign of partial-thickness adductor longus tendon tears

The superficial fibers of the rectus abdominis and adductor longus tendons are in direct continuity [91]. A finding on MRI consistent with this combination tear is the so-called superior cleft sign [92]. (Radiologists just love naming imaging findings with a sign) This sign is present when there is fluid signal extending lateral to the symphysis at the level of the caudal margin of the superior pubic ramus; this linear fluid signal is located more anterosuperiorly at the pubis than the secondary cleft sign (Fig. 72).

Very frequently patients with athletic pubalgia and attendant symphyseal instability have abnormal findings at the symphysis. There is a frequent marrow edema pattern in the pubic bodies, often asymmetrically prominent on the side of tendon tearing if unilateral. Other frequent findings include marginal proliferative change, subchondral cysts, and subchondral erosions. Subchondral fractures can also occur about the symphysis. While a symphyseal marrow edema

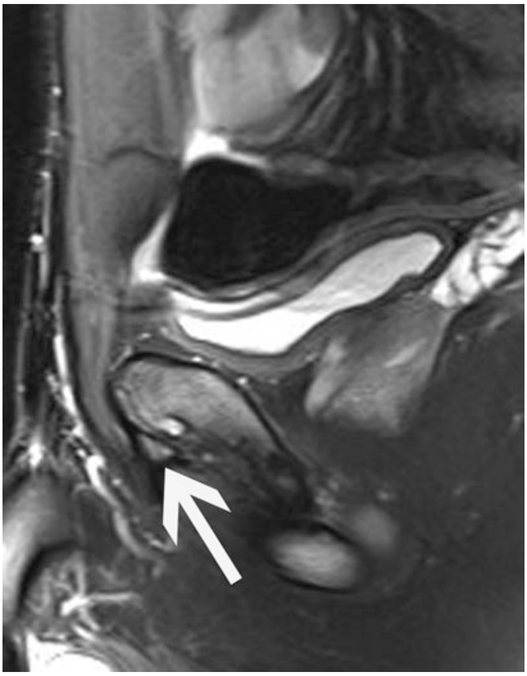


Fig. 69 Fat-suppressed sagittal T2-weighted image just off midline shows high signal at the origin of the adductor longus tendon (*arrow*), representing a partial-thickness tear

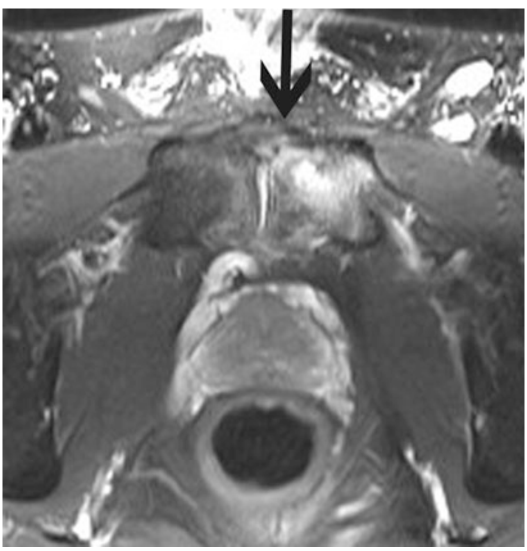


Fig. 70 Fat-suppressed axial T2-weighted image shows thin fluid undercutting the medial aspect of the left rectus abdominis tendon (*arrow*), representing a partial-thickness tear. Note the disproportionate marrow edema pattern in the adjacent pubic body. The patient underwent a modified Bassini repair of the tendon

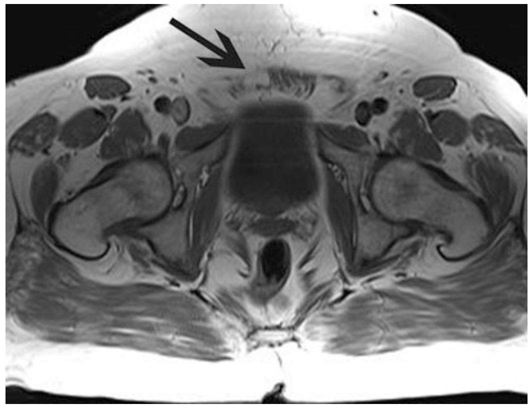


Fig. 71 Axial T1-weighted image shows disproportionate prominent fat infiltration in the right rectus abdominis muscle (*arrow*), reflecting a chronic tendon tear

Fig. 72 Fat-suppressed coronal T2-weighted image shows bilateral horizontally oriented high signal at the level of the inferior superior pubic ramus (*arrows*), reflecting bilateral tears at the junction of the rectus abdominis and adductor longus tendons

pattern is usually present, there may be none in patients with rectus abdominis tendon tears [93].

Athletic pubalgia and symptomatic FAI frequently coexist [94–96]. The moniker "sports hip triad" refers to the presence of a labral tear and rectus abdominis and adductor injury [94]. While not dedicated imaging of either hip, MRI performed in the evaluation of athletic pubalgia often reveals evidence of FAI (i.e., labral

Fig. 73 Fat-suppressed coronal T2-weighted image (**a**) shows high signal extending from the symphysis into the origin of the left adductor longus tendon (*arrow*), representing a partial-thickness tear. Another image from this same sequence (**b**) shows bilateral labral tears, as well as focal full-thickness chondral loss over the adjacent dome in the right hip (*arrows*)

tears, chondral defects) in either or both hips (Fig. 73).

While athletic pubalgia is not a true hernia by definition, the rectus abdominis tendon fibers have a close relationship with the internal oblique and transversus abdominis tendons at the posterior inguinal wall [91]. Thus, it may be difficult to separate some tears from a true hernia as there may be a related posterior wall defect or insufficiency.

Ischiofemoral Impingement

Ischiofemoral impingement is an infrequent cause of hip, buttock, or lower extremity pain. Most frequently, pain is localized to the posterior hip [97]. Occasionally, pain may radiate distally into the thigh or to the knee, possibly due to associated sciatic nerve involvement given its proximity to the distal quadratus femoris muscle [98]. It may cause a snapping sensation [99]. The majority of affected patients are middle-aged and older females [97, 99, 100].

Studies of affected patients have found the ischiofemoral and quadratus femoris spaces (i.e., distances) to be significantly narrower than in controls [97, 99]. The ischiofemoral distance is the distance between the lateral margin of the ischial tuberosity and the lesser trochanter. The quadratus femoris distance is the distance between the lateral margin of the proximal semimembranosus tendon and the lesser tuberosity or iliopsoas tendon. The quadratus femoris distance is usually less than the ischiofemoral distance due to the lateral location of the semimembranosus tendon relative to the tuberosity. However, note that the space distances obtained while supine at rest on MRI may not be entirely representative of that with dynamic hip motion.

Ischiofemoral impingement has also been documented in the posttraumatic and postsurgical setting, including after proximal femoral osteotomy and total hip arthroplasty [101]. In these cases, symptoms resolved after resection of the lesser trochanter.

On MRI, the typical finding of ischiofemoral impingement is edema about the distal quadratus femoris muscle-tendon junction, extending variably into the distal and midportion of the muscle (Fig. 74). Often, the patient has disproportionate fatty atrophy of the distal muscle. Subcortical cystic changes can be seen about the space [97, 98].

Quadratus femoris muscle-tendon junction strains as well as partial-thickness and

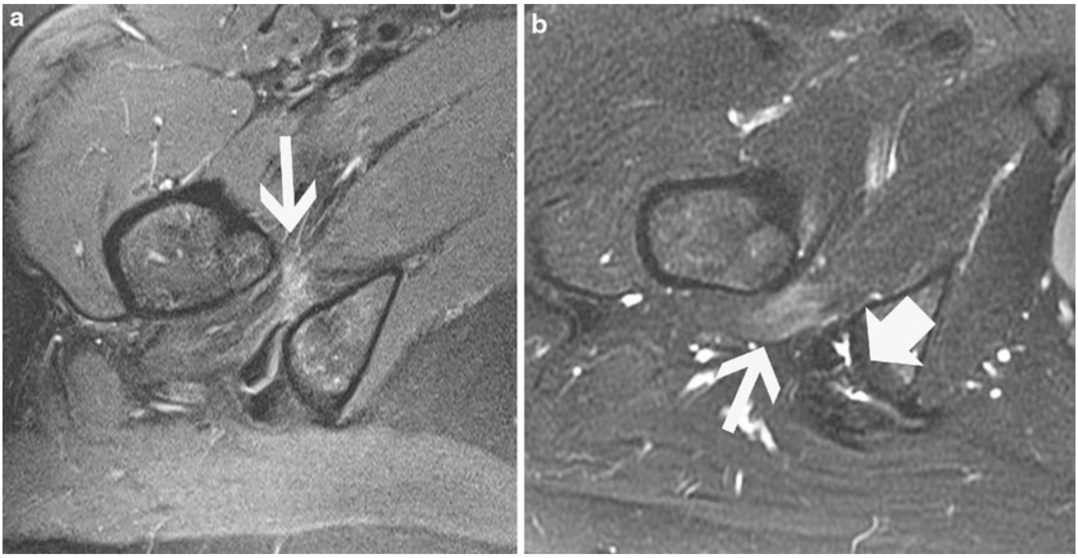


Fig. 74 Fat-suppressed oblique axial proton density-weighted image (**a**) and axial T2-weighted image (**b**) in different patients show edema in the quadratus femoris muscle as it courses between the trochanter and tuberosity (*arrow* in **a** and intermediate-sized *arrow* in **b**). Note the partial-thickness tear of the hamstring tendon origins in **b** (*short*, *thick arrow*)

full-thickness tears of the muscle-tendon junction are infrequent but can occasionally be seen [100, 102, 103]. Some patients with tears have chronic pain [100]. Thus, while not proven, it is plausible that chronic impingement could predispose to frank tearing of the tendon.

Summary

MRI is a useful noninvasive tool in the evaluation of hip and groin pain. It can help to clarify or diagnose a vast array of pathology in and about the hip. The shape of the dome and head and the relative hip's deep location present unique challenges in diagnosis of central compartment pathology. The accuracy of detecting chondral pathology is not optimal. With inevitable technological advancements in magnet and coil design, accurate detection of pathology should improve. Quantitative cartilage imaging can be used for the diagnosis of early, pre-morphologic chondrosis. One hopes that with improvements in the early detection of chondrosis, methods will be developed to impede the progression of or even halt the development of osteoarthritis.

MRI of the hip and hip preservation surgery is continuously evolving. There is no doubt that some of the current paradigms for both will be disproved in the years ahead. Likewise, undoubtedly new techniques and improvements in existing ones will occur. My hope is that orthopedic surgeons will be able to benefit from the images and discussion in this chapter for at least the next several years ahead.

References

1. Mintz DN, Hooper T, Connell D, Buly R, Padgett DE, Potter HG. Magnetic resonance imaging of the hip: detection of labral and chondral abnormalities using noncontrast imaging. Arthroscopy. 2005;21:385–93.
2. Register B, Pennock AT, Ho CP, Strickland CD, Lawand A, Philippon MJ. Prevalence of abnormal hip findings in asymptomatic participants: a prospective, blinded study. Am J Sports Med. 2012;40:2720–4. doi:10.1177/03635465124462124.
3. Robinson P. Conventional 3-T MRI and 1.5-T MR arthrography of femoroacetabular impingement. AJR. 2012;199:509–15.
4. Sundberg TP, Toomayan GA, Major NM. Evaluation of the acetabular labrum at 3.0-T MR imaging compared with 1.5-T MR arthrography: preliminary experience. Radiology. 2006;238:706–11.

5. Nötzli HP, Wyss TF, Stoecklin CH, Schmid MR, Treiber K, Hodler J. The contour of the femoral head-neck junction as a predictor for the risk of anterior impingement. J Bone Joint Surg Br. 2002;84-B:556–60.
6. Agricola R, Bessems J, Ginai AZ, Heijboer MP, van der Heijden RA, Verhaar J, et al. The development of cam-type deformity in adolescent and young male soccer players. Am J Sports Med. 2012;40:1099–106.
7. Barton C, Salineros MJ, Rakhra KS, Beaule PE. Validity of the alpha angle measurement on plain radiographs in the evaluation of cam-type femoroacetabular impingement. Clin Orthop Relat Res. 2011;469:464–9.
8. Clohisy JC, Nunley RM, Otto RJ, Schoenecker PL. The frog-leg lateral radiograph accurately visualized hip cam impingement abnormalities. Clin Orthop Relat Res. 2007;462:115–21.
9. Mast NH, Impiellizzeri F, Keller S, Leunig M. Reliability and agreement of measures used in radiographic evaluation of the adult hip. Clin Orthop Relat Res. 2011;469:188–99.
10. Philippon MJ, Ho CP, Briggs KK, Stull J, LaPrade RF. Prevalence of increased alpha angles as a measure of cam-type femoroacetabular impingement in youth hockey players. Am J Sports Med. 2013;41:1357–62.
11. Lohan DG, Seeger LL, Motamedi K, Hame S, Sayre J. Cam-type femoral-acetabular impingement: is the alpha angle the best MR arthrography has to offer? Skeletal Radiol. 2009;38:855–62.
12. Rakhra KS, Sheikh AM, Allen D, Beaule PE. Comparison of MRI alpha angle measurement planes in femoroacetabular impingement. Clin Orthop Relat Res. 2009;467(3):660–5.
13. Stelzeneder D, Hingsammer A, Bixby SD, Kim YJ. Can radiographic parameters for the hip be assessed on MRI? Clin Orthop Relat Res. 2013;471(3):989–99. doi:10.1007/s11999-012-2654-3.
14. Zlatkin MB, Pevsner D, Sanders TG, Hancock CR, Ceballos CE, Herrera MF. Acetabular labral tears and cartilage lesions of the hip: indirect MR arthrographic correlation with arthroscopy – a preliminary study. AJR. 2010;194(3):709–14. doi:10.2214/AJR.07.3669.
15. Smith TO, Hilton G, Toms AP, Donell ST, Hing CB. The diagnostic accuracy of acetabular labral tears using magnetic resonance imaging and magnetic resonance arthrography: a meta-analysis. Eur Radiol. 2011;21:863–74.
16. Czerny C, Hofmann S, Urban M, Tschauner C, Neuhold A, Pretterklieber M, Recht MP, Kramer J. MR arthrography of the adult acetabular capsular-labral complex: correlation with surgery and anatomy. AJR. 1999;173:345–9.
17. Lage LA, Patel JV, Viller RN. The acetabular labral tear: an arthroscopic classification. Arthroscopy. 1996;12:269–72.
18. Blankenbaker DG, De Smet AA, Keene JS, Fine JP. Classification and localization of acetabular labral tear. Skeletal Radiol. 2007;36:391–7.
19. Dinauer PA, Murphy KP, Carroll JF. Sublabral sulcus at the posteroinferior acetabulum: a potential pitfall in MR arthrography diagnosis of acetabular labral tears. AJR. 2004;183(6):1745–53.
20. Kim KW, Baek JH, Ha YC. Prevalence and locations of acetabular labral sulcus in patients undergoing arthroplasty for hip fracture. Arthroscopy. 2012;28(10):1373–80.
21. Saddik D, Troupis J, Tirman P, O'Donnell J, Howells R. Prevalence and location of acetabular sublabral sulci at hip arthroscopy with retrospective MRI review. AJR. 2006;187:W507–11.
22. Studler U, Kalberer F, Leunig M, Zanetti M, Hodler J, Dora C, et al. MR arthrography of the hip: differentiation between an anterior sublabral recess as a normal variant and a labral tear. Radiology. 2008;249:947–54.
23. Yoon LS, Palmer WE, Kassarjian A. Evaluation of radial-sequence imaging in detecting acetabular labral tears at hip MR arthrography. Skeletal Radiol. 2007;36:1029–33.
24. Aly AR, Rajasekaran S, Obaid H. MRI morphometric hip comparison analysis of anterior acetabular labral tears. Skeletal Radiol. 2013;42(9):1245–52.
25. Blankenbaker DG, Tuite MJ, Keene JS, del Rio AM. Labral injuries due to iliopsoas impingement: can they be diagnosed on MR arthrography? AJR. 2012;199(4):894–900.
26. Domb BG, Shindle MK, McArthur B, Vos JE, Magennis EM, Kelly BT. Iliopsoas impingement: a newly identified cause of labral pathology in the hip. HSS J. 2011;7(2):145–50.
27. Egerton T, Hinman RS, Takla A, Bennell KL, O'Donnell J. Intraoperative cartilage degeneration predicts outcome 12 months after hip arthroscopy. Clin Orthop Relat Res. 2013;471(2):593–9.
28. Farjo LA, Glick JM, Sampson TG. Hip arthroscopy for acetabular labral tears. Arthroscopy. 1999;15(2):132–7.
29. Murphy S, Tannast M, Kim YJ, Buly R, Millis MB. Debridement of the adult hip for femoroacetabular impingement. Clin Orthop Relat Res. 2004;429:178–81.
30. Streich NA, Gotterbarm T, Barie A, Schmitt H. Prognostic value of chondral defects on the outcome after arthroscopic treatment of acetabular labral tears. Knee Surg Sports Traumatol Arthrosc. 2009;17(10):1257–63.
31. Anderson LA, Peters CL, Park BB, Stoddard GJ, Erickson JA, Crim JR. Acetabular cartilage delamination in femoroacetabular impingement. J Bone Joint Surg Am. 2009;91:305–13.
32. Camacho-Alvarez D, Mardones-Peterman R. Femoroacetabular impingement: association between the over-coverage and acetabular cartilage delamination areas. Rev Esp Cir Ortop Traumatol. 2013;57(2):111–6.

33. James SLJ, Ali K, Malara F, Young D, O'Donnell J, Connell DA. MRI findings of femoroacetabular impingement. AJR. 2006;187:1412–9.
34. Pfirrmann CWA, Duc SR, Zanetti M, Dora C, Hodler J. MR arthrography of acetabular cartilage delamination in femoroacetabular cam impingement. Radiology. 2008;249:1236–41.
35. Spahn G, Klinger HM, Hofmann GO. How valid is the arthroscopic diagnosis of cartilage lesions? Results of an opinion survey among highly experienced arthroscopic surgeons. Arch Orthop Trauma Surg. 2009;129:1117–21.
36. Neumann G, Mendicuti AD, Zou KH, Minas T, Coblyn J, Winalski CS, et al. Prevalence of labral tears and cartilage loss in patients with mechanical symptoms of the hip: evaluation using MR arthrography. Osteoarthritis Cartilage. 2007;15(8):909–17.
37. Beaule PE, Zaragoza E, Copelan N. Magnetic resonance imaging with gadolinium arthrography to assess acetabular cartilage delamination: a report of four cases. J Bone Joint Surg Am. 2004;86-A:2294–9.
38. Dietrich TJ, Suter A, Pfirrmann CW, Dora C, Fucentese SF, Zanetti M. Supraacetabular fossa (pseudodefect of acetabular cartilage): frequency at MR arthrography and comparison of findings at MR arthrography and arthroscopy. Radiology. 2012;263 (2):484–91.
39. Jazrawi LM, Alaia MJ, Chang G, Fitzgerald EF, Recht MP. Advances in magnetic resonance imaging of articular cartilage. J Am Acad Orthop Surg. 2011;19(7):420–9.
40. Kim YJ, Jaramillo D, Millis MB, Gray ML, Burstein D. Assessment of early osteoarthritis in hip dysplasia with delayed gadolinium-enhanced magnetic resonance imaging of cartilage. J Bone Joint Surg Am. 2003;85-A:1987–92.
41. Cunningham T, Jessel R, Zurakowski D, Millis MB, Kim YJ. Delayed gadolinium-enhanced magnetic resonance imaging of cartilage to predict early failure of Bernese periacetabular osteotomy for hip dysplasia. J Bone Joint Surg Am. 2006;88:1540–8.
42. Botser IB, Martin DE, Stout CE, Domb BG. Tears of the ligamentum teres: prevalence in hip arthroscopy using 2 classification systems. Am J Sports Med. 2011;39:117S. doi:10.1177/0363546511413865.
43. Guanche CA, Sikka RS. Acetabular labral tears with underlying chondromalacia: a possible association with high-level running. Arthroscopy. 2005; 21(5):580–5.
44. Phillipon MJ, Kuppersmith DA, Wolff AB, Briggs KK. Arthroscopic findings following traumatic hip dislocation in 14 professional athletes. Arthroscopy. 2009;25(2):169–74.
45. Byrd JW, Jones KS. Traumatic rupture of the ligamentum teres as a source of hip pain. Arthroscopy. 2004;20:385–91.
46. Bardakos NV, Villar RN. The ligamentum teres of the adult hip. J Bone Joint Surg Br. 2009;91-B:8–15.
47. Demange MK, Kakuda CMS, Pereiera CAM, Sakaki MH, Albuqueque RFM. Influence of the femoral head ligament on hip mechanical function. Acta Orthop Bras. 2007;15:187–90.
48. Cerezal L, Kassarjian A, Canga A, Dobado MC, Montero JA, Llopis E, et al. Anatomy, biomechanics, imaging, and management of ligamentum teres injuries. RadioGraphics. 2010;30:1637–51.
49. Baber YF, Robinson AH, Villar RN. Is diagnostic arthroscopy of the hip worthwhile? A prospective review of 328 adults investigated for hip pain. J Bone Joint Surg Br. 1999;81(4):600–3.
50. Rao J, Zhou YX, Villar RN. Injury to the ligamentum teres. Mechanism, findings, and results of treatment. Clin Sports Med. 2001;20(4):791–9.
51. Blankenbaker DG, De Smet AA, Keene JS, Del Rio AM. Imaging appearance of the normal and partially torn ligamentum teres on hip MR arthrography. AJR. 2012;199(5):1093–8.
52. Bencardino JT, Kassarjian A, Vieira RL, Schwartz R, Mellado JM, Kocher M. Synovial plicae of the hip: evaluation using MR arthrography in patients with hip pain. Skeletal Radiol. 2011;40(4):415–21.
53. Gray AJR, Villar RN. The ligamentum teres of the hip: an arthroscopic classification of its pathology. Arthroscopy. 1997;13:575–8.
54. Weidner J, Buchler L, Beck M. Hip capsule dimensions in patients with femoroacetabular impingement: a pilot study. Clin Orthop Relat Res. 2012;470: 3306–12.
55. Byrd JW, Jones KS. Adhesive capsulitis of the hip. Arthroscopy. 2006;22(1):89–94.
56. Fu Z, Peng M, Peng Q. Anatomical study of the synovial plicae of the hip joint. Clin Anat. 1997;10(4):235–8.
57. Atlihan D, Jones DC, Guanche CA. Arthroscopic treatment of a symptomatic hip plica. Clin Orthop Relat Res. 2003;411:174–7.
58. Hélénon CH, Bergevin H, Aubert JD, Lebreton C, Hélénon O. Plication of hip synovia at upper border femoral neck. J Radiol. 1986;67:737–40.
59. Blankenbaker DG, Davis KW, De Smet AA, Keene JS. MRI appearance of the pectinofoveal fold. AJR. 2009;192:93–5.
60. Blankenbaker DG, Ullrick SR, Davis KW, De Smet AA, Haaland B, Fine JP. Correlation of MRI findings with clinical findings of trochanteric pain syndrome. Skeletal Radiol. 2008;37(10):903–9.
61. Beck M. Groin pain after open FAI surgery: the role of intraarticular adhesions. Clin Orthop Relat Res. 2009;467:769–74.
62. Heyworth BE, Shindle MK, Voos JE, Rudzki JR, Kelly BT. Radiologic and intraoperative findings in revision hip arthroscopy. Arthroscopy. 2007;23(12):1295–302.
63. Philippon MJ, Schenker ML, Briggs KK, Kuppersmith DA, Maxwell RB, Stubbs AJ. Revision hip arthroscopy. Am J Sports Med. 2007;35(11): 1918–21.

64. McCarthy JC, Glassner PJ. Correlation of magnetic resonance arthrography with revision hip arthroscopy. Clin Orthop Relat Res. 2013;471:4006–11.
65. Blankenbaker DG, De Smet AA, Keene JS. MR arthrographic appearance of the postoperative acetabular labrum in patients with suspected recurrent labral tears. AJR. 2011;197:W1118–22. doi:10.2214/AJR.11.6671.
66. Dudda M, Mamisch TC, Krueger A, Werlen S, Siebenrock KA, Beck M. Hip arthroscopy after surgical hip dislocation: is predictive imaging possible? Arthroscopy. 2011;27(4):486–92.
67. Krueger A, Leunig M, Siebenrock KA, Beck M. Hip arthroscopy after previous surgical hip dislocation for femoroacetabular impingement. Arthroscopy. 2007;23(12):1285–9.
68. McCormick F, Slikker W 3rd, Harris JD, Gupta AK, Abrams GD, Frank J, et al. Evidence of capsular defect following hip arthroscopy. Knee Surg Sports Traumatol Arthrosc. 2013 [Epub ahead of print].
69. Nepple JJ, Byrd JW, Siebenrock KA, Prather H, Clohisy JC. Overview of treatment options, clinical results, and controversies in the management of femoroacetabular impingement. J Am Acad Orthop Surg. 2013;21 Suppl 1:S53–8.
70. Dietrich TJ, Dora C, Pfirrmann CWA. Postoperative imaging in femoroacetabular impingement. Semin Musculoskelet Radiol. 2013;17:272–8.
71. Zingg PO, Buehler TC, Poutawera VR, Alireza A, Dora C. Femoral neck fractures after arthroscopic femoral neck osteochondroplasty for femoroacetabular impingement. Knee Surg Sports Traumatol Arthrosc. 2012 [Epub ahead of print].
72. Egerton T, Hinman RS, Takla A, Bennell KL, O'Donnell J. Intraoperative cartilage degeneration predicts outcome 12 months after hip arthroscopy. Clin Orthop Relat Res. 2013;471(2):593–9. doi:10.1007/s11999-012-2594-y.
73. Haviv B, O'Donnell J. Arthroscopic treatment for acetabular labral tears of the hip without bony dysmorphism. Am J Sports Med. 2011;39:79S. doi:10.1177/0363546511412915.
74. Malizos KN, Karantanas AH, Varitimidis SE, Dailiana ZH, Bargiotas K, Maris T. Osteonecrosis of the femoral head: etiology, imaging and treatment. Eur J Radiol. 2007;63:16–28.
75. Sugano N, Takaoka K, Ohzono K, Matsui M, Masuhara K, Ono K. Prognostication of nontraumatic avascular necrosis of the femoral head. Clin Orthop Relat Res. 1994;303:155–64.
76. Zibis AH, Karantanas AH, Roidis NT, Hantes ME, Argiri P, Moraitis T, Malizos KN. The role of MR imaging in staging femoral head osteonecrosis. Eur J Radiol. 2007;63:3–9.
77. Theodorou DJ, Theodorou SJ, Haghighi P, Resnick D. Distinct focal lesions of the femoral head: imaging features suggesting an atypical and minimal form of bone necrosis. Skeletal Radiol. 2002;31:435–44.
78. Malizos KN, Zibis AH, Dailiana Z, Hantes M, Karachalios T, Karantanas AH. MR imaging findings in transient osteoporosis of the hip. Eur J Radiol. 2004;50(3):238–44.
79. Cahir JG, Toms AP. Regional migratory osteoporosis. Eur J Radiol. 2008;67:2–10.
80. Stevens K, Tao C, Lee S-U, Salem N, et al. Subchondral fractures in osteonecrosis of the femoral head: comparison of radiography, CT, and MR imaging. AJR. 2003;180:363–8.
81. Yamamoto T, Takabatake K, Iwamoto Y. Subchondral insufficiency fracture of the femoral head resulting in rapid destruction of the hip joint: a sequential radiographic study. AJR. 2002;178:435–7.
82. Kransdorf MJ, Stull MA, Gilkey FW, Moser Jr RP. Osteoid osteoma. RadioGraphics. 1991;11:671–96.
83. Lee EH, Shafi M, Hui JH. Osteoid osteoma: a current review. J Pediatr Orthop. 2006;26(5):695–700.
84. Chai JW, Hong SH, Choi J-Y, Koh YH, Lee JW, Choi J-A, Kang HS. Radiologic diagnosis of osteoid osteoma: from simple to challenging findings. RadioGraphics. 2010;30:737–49.
85. Bankenbaker DG, Tuite MJ. Iliopsoas musculotendinous unit. Semin Musculoskelet Radiol. 2008;12(1):13–27. doi:10.1055/s-2008-1067934.
86. Blankenbaker DG, De Smet AA, Keene JS. Sonography of the iliopsoas tendon and injection of the iliopsoas bursa for diagnosis and management of the painful snapping hip. Skeletal Radiol. 2006;35(8):565–71.
87. Deslandes M, Guillin R, Cardinal E, Hobden R, Bureau NJ. The snapping iliopsoas tendon: new mechanisms using dynamic sonography. AJR. 2008;190(3):576–81. doi:10.2214/AJR.07.2375.
88. De Smet AA, Blankenbaker DG, Alsheik NH, Lindstrom MJ. MRI appearance of the proximal hamstring tendons in patients with and without symptomatic proximal hamstring tendinopathy. AJR. 2012;198:418–22.
89. Rubin DA. Imaging diagnosis and prognostication of hamstring injuries. AJR. 2012;199:525–33.
90. Robinson P, Salehi F, Grainger A, Clemence M, Schilders E, O'Connor P, Agur A. Cadaveric and MRI study of the musculotendinous contributions to the capsule of the symphysis pubis. AJR. 2007;188: W440–5. doi:10.2214/AJR.06.1238.
91. Koulouris G. Imaging review of groin pain in elite athletes: an anatomic approach to imaging findings. AJR. 2008;191:962–72.
92. Murphy G, Foran P, Murphy D, Tobin O, Moynagh M, Eustace S. "Superior cleft sign" as a marker of rectus abdominis/adductor longus tear in patients with suspected sportsman's hernia. Skeletal Radiol. 2013;42:819–25.
93. Zoga AC, Kavanagh EC, Omar IM, Morrison WB, Koulouris G, Lopez H, et al. Athletic pubalgia and the "sports hernia": MR imaging findings. Radiology. 2008;247(3):797–807. doi:10.1148/radiol.2473070049.

94. Freeley BT, Powell JW, Muller MS, Barnes RP, Warren RF, Kelly BT. Hip injuries and labral tears in the national football league. Am J Sports Med. 2008;36(11):2187–95.
95. Hammoud S, Bedi A, Magennis E, Meyers WC, Kelly BT. High incidence of athletic pubalgia symptoms in professional athletes with symptomatic femoroacetabular impingement. Arthroscopy. 2012;28(10):1388–95.
96. Larson CM, Pierce BR, Giveans MR. Treatment of athletes with symptomatic intra-articular hip pathology and athletic pubalgia/sports hernia: a case series. Arthroscopy. 2011;27(6):768–75. doi:10.1016/j.arthro.2011.01.018.
97. Tosun O, Algin O, Yalcin N, Cay N, Ocakoglu G, Karaoglanoglu M. Ischiofemoral impingement: evaluation with new MRI parameters and assessment of their reliability. Skeletal Radiol. 2012;41:575–87.
98. Patti JW, Ouellette H, Bredella MA, Torriani M. Impingement of lesser trochanter on ischium as a potential cause for hip pain. Skeletal Radiol. 2008;37:939–41.
99. Torriani M, Souto SC, Thomas BJ, Ouellette H, Bredella MA. Ischiofemoral impingement syndrome: an entity with hip pain and abnormalities of the quadratus femoris muscle. AJR. 2009;193(1):186–90. doi:10.2214/AJR.08.2090.
100. O'Brien SD, Bui-Mansfield LT. MRI of quadratus femoris muscle tear: another cause of hip pain. AJR. 2007;189:1185–9.
101. Johnson KA. Impingement of the lesser trochanter on the ischial ramus after total hip arthroplasty. Report of three cases. J Bone Joint Surg Am. 1977;59(2):268–9.
102. Peltola K, Heinonen OJ, Orava S, Mattila K. Quadratus femoris muscle tear: an uncommon cause for radiating gluteal pain. Clin J Sport Med. 1999;9:228–30.
103. Willick SE, Lazarus M, Press JM. Quadratus femoris strain. Clin J Sport Med. 2002;12:130–1.

Ultrasound of the Hip

6

Ronald S. Adler

Contents

Abstract

The adult hip poses several challenges to ultrasound evaluation. The structures are deeply situated requiring the use of relatively low-frequency transducers (5–8 MHz range), thereby limiting resolution. Variable amounts of abdominal fat often cause aberration of the ultrasound beam. Doppler assessment is also limited due to diminished flow sensitivity in evaluating deep structures. Despite these limitations, ultrasound can display a variety of pathologic conditions about the hip. The real-time capability allows assessment of conditions elicited by provocative maneuvers (i.e., snapping hip syndromes). Ultrasound provides guidance for performance of selective interventional procedures, and it is not subject to artifact introduced by indwelling metallic hardware. Ultrasound is, therefore, playing an increasingly important diagnostic and therapeutic role in patients with hip pain. Following a brief overview of ultrasound technique and normal sonographic anatomy, a variety of common clinical applications of ultrasound of the hip are discussed.

Introduction

The adult hip poses several challenges to ultrasound evaluation. The structures are deeply situated requiring the use of relatively low-frequency transducers (5–8 MHz range), thereby limiting

R.S. Adler
Center for Musculoskeletal Care, NYU Langone Medical Center, New York, NY, USA
e-mail: ronald.adler@nyumc.org

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_6

resolution. Variable amounts of abdominal fat often cause aberration of the ultrasound beam. Doppler assessment is also limited due to diminished flow sensitivity in evaluating deep structures. Nevertheless, ultrasound is playing an increasingly important role in hip imaging, particularly in the assessment of "dynamic disorders," such as the "snapping hip," as well as in the performance of a large variety of therapeutic injections, aspirations, or biopsies [1–9]. The combination of image fusion along with real-time ultrasound imaging may further increase the role of ultrasound in performing a variety of interventions about the hip.

Following a brief overview of ultrasound technique and normal sonographic appearances, a variety of common clinical applications of ultrasound of the hip will be discussed. Description of specific disorders will be included with respect to the relevant sonographic findings.

Technique

The hip is typically imaged using an intermediate frequency transducer (5–8 MHz). The best image quality is afforded by a linear phased array transducer, but often a curved or sector format provides a larger field of view to assess the anatomy of interest. The optimal frequency is often determined by patient body habitus, the nature of the subcutaneous fat, and the depth and extent of the area of interest [2]. Extended field-of-view (EFOV) imaging, present on many scanners, can include as much as 60 cm of in-plane information, providing a method to image pathology that exceeds the dimensions of the transducer footprint [10].

The advent of two-dimensional arrays will permit real-time biplanar imaging. Electromagnetic needle tracking and transducer tracking may ultimately allow real-time multimodality fusion imaging, further enhancing the role of ultrasound in evaluating and treating hip disorders.

Hip Joint

The hip joint should be scanned in long axis with the transducer positioned along the anterior aspect of the proximal thigh (Fig. 1). The cortical surfaces appear as an echogenic (bright) specular surface with posterior shadowing [2, 3, 6]. At diagnostic frequencies and power, only the cortical surfaces can be imaged. The femoral head is a convex structure adjacent to a short linear cortical surface, corresponding to the anterior margin of the acetabulum. The convex surface transitions to a concave surface, corresponding to the head-neck junction. A thin hypoechoic (dark) band contiguous with the femoral head relates to the visible portion of the articular cartilage, while an overlying triangular echogenic structure corresponds to the anterior superior labrum. A thin echogenic band, contiguous with the acetabulum, overlying the femoral head and neck relates to the anterior capsule. The interface between the capsule and labrum may be difficult to separate. The non-distended capsule will parallel the cortical margin of the femoral head and neck. Hypoechoic structures overlying the capsule relate to visualized portions of the iliopsoas, rectus femoris, and sartorius muscles.

In short axis, the femoral head appears convex with overlying hypoechoic articular cartilage and echogenic joint capsule (Fig. 2). The iliopectineal eminence of the acetabulum is visualized as a cortical specular reflector medial to the femoral head. An echogenic ellipse abutting the acetabulum, which may lie within a thin capsular depression, corresponds to the iliopsoas tendon. The pectineus muscle belly lies medial to the iliopsoas tendon and superficial to the acetabulum. Superficial and medial to the iliopsoas muscle/tendon complex lies the neurovascular structures, femoral artery, and vein (hypoechoic ellipses in short axis), adjacent to the femoral nerve (variable echogenicity and may have a fascicular appearance). The iliopsoas bursa lies deep and often medial to the tendon at this anatomical level and is generally not distended in asymptomatic individuals [9].

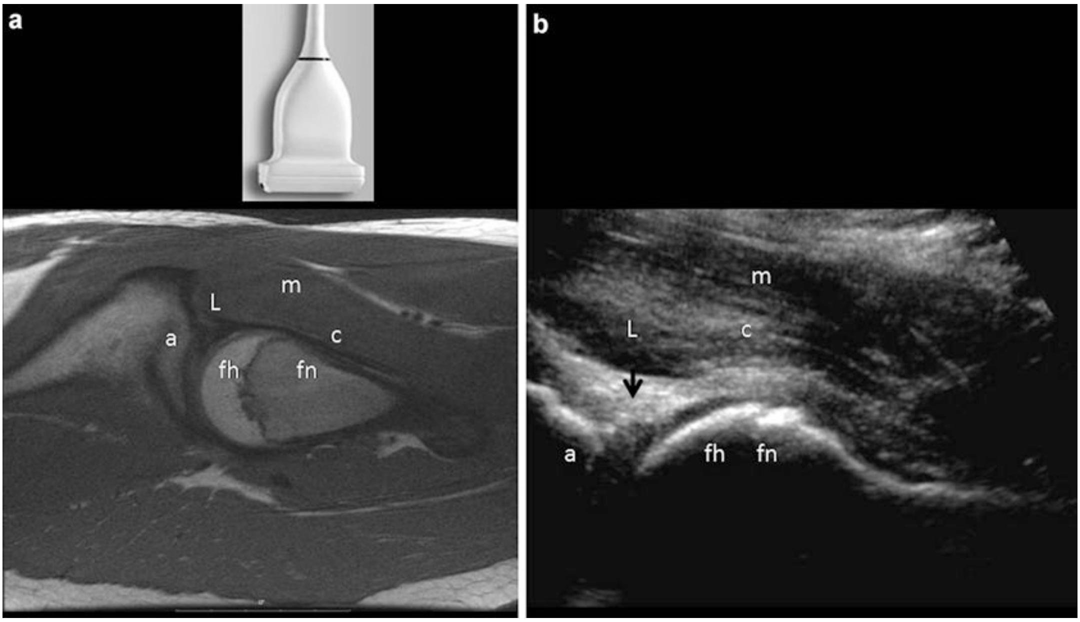


Fig. 1 (**a**) Representative long axis orientation obtained from a proton density acquisition of the hip showing the transducer position relative to the acetabulum and femoral head-neck junction. The femoral head (*fh*), femoral neck (*fn*), acetabulum (*a*), capsule (*c*), labrum (*L*) and iliopsoas muscle (*m*) are labeled. (**b**) Corresponding long-axis ultrasound image displaying the sonographic anatomy. A thin hypoechoic (*dark*) band overlies the femoral head, corresponding to the articular cartilage. Alternatively, fibrocartilage (labrum, *black arrow*) appears hyperechoic (*bright*) on ultrasound. Note the absence of echoes deep to the cortical surfaces due to the presence of posterior acoustic shadowing. The labels are similar to (**a**)

Distension of the joint capsule is appreciated as separation from the cortical margin, often appearing convex (Fig. 3). The nature of the material distending the joint is quite variable. Fluid is typically anechoic or hypoechoic, but can appear echogenic, such as in an acute hemarthrosis [11] Synovitis or capsular thickening may be difficult to distinguish from complex fluid. Various measurements of capsular distention have been suggested to assess for joint effusion [6]. Direct compression with the ultrasound transducer resulting in redistribution of the material or arthrocentesis is the only reliable method to distinguish fluid from other soft tissue processes. Material that is incompressible or demonstrates vascularity on Doppler imaging confirms its solid nature.

The cortical surface is normally smooth and continuous. The presence of contour deformities, osteophytic ridges, and joint bodies may be evident on sonography (Fig. 4). Diminished offset of the femoral head-neck junction may be measured sonographically in the setting of a CAM-type femoral acetabular impingement (FAI) [12, 13]. Likewise, the presence of labral pathology, which often affects the anterior superior labrum, may be conspicuous as intrasubstance hypoechoic clefts, a blunted or fragmented labrum (Fig. 5). Fluid imbibition into the labrum following intra-articular injection can result in improved conspicuity of the labral tear (Fig. 6) [14–16]. Observation of the hip during flexion with internal rotation (FADIR maneuver) may help assess dynamic impingement. Likewise, real-time observation in patients with snapping hip syndrome (below) may provide useful information as to the etiology.

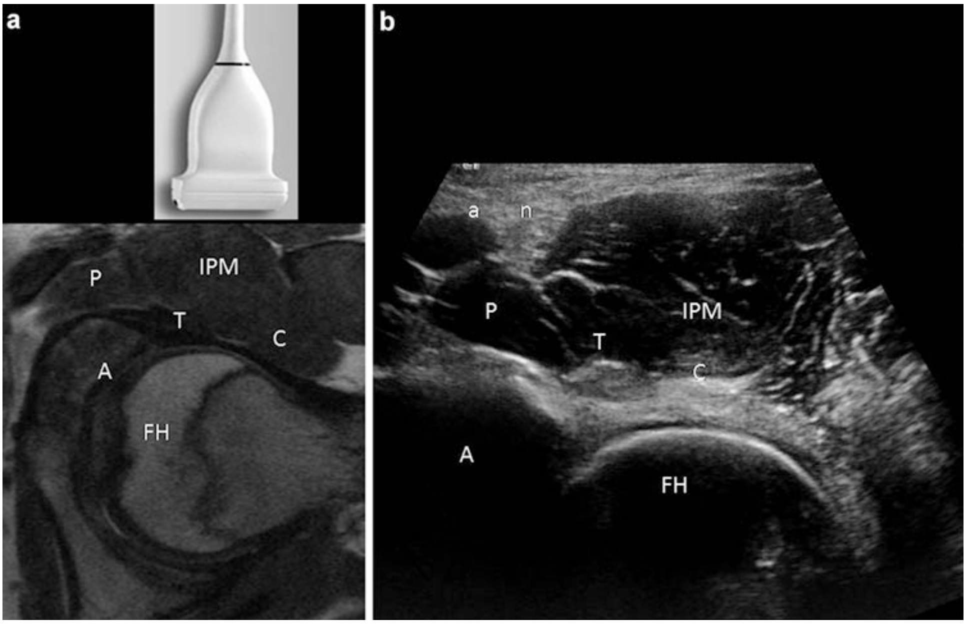


Fig. 2 Short-axis ultrasound of the hip. (**a**) Axial proton density image displaying the transducer position and relationship of the femoral head (*FH*), acetabulum (*A*), pectineus muscle (*P*), iliopsoas tendon (*T*), iliopsoas muscle (*IPM*) and capsule (*C*). (**b**) Corresponding ultrasound anatomy. Note that the iliopsoas tendon (*T*) appears hyperechoic in distinction to the low signal intensity of the tendon on MR. Muscle is hypoechoic, containing echogenic linear structures, corresponding to fascial boundaries and perimyseal connective tissue. The tendon sits in a small capsular depression and overlies the anterior labrum. The femoral artery (*a*) and nerve (*n*) are labeled. The remaining labels are similar to (**a**)

Muscles and Tendons

It is convenient to separate the muscles/tendons into anterior, medial, lateral, and posterior compartments. The anterior compartment consists of the iliopsoas, rectus femoris, and sartorius muscles and the remaining quadriceps compartment muscles distal to the hip joint proper. The medial compartment consists primarily of the gracilis and adductor muscles. The pectineus and obturator muscles are not frequently examined on ultrasound; the lateral compartment consists of the tensor fascia lata, inclusive of the iliotibial tract, and gluteus minimus, medius, and maximus. The posterior compartment primarily consists of the hamstring tendons and muscles, piriformis, and short external rotators.

Muscles appear hypoechoic containing fine linear echogenic structures corresponding to the perimyseal connective tissue [17, 18]. The epimyseal connective tissue appears echogenic (Fig. 7). The rectus femoris and sartorius muscles can be traced proximally to their respective tendons. It is important to recognize that in distinction to MR, tendons appear as compact echogenic and fibrillar on ultrasound and are subject to anisotropy (Fig. 8) [19].

The latter refers to the variable tendon echogenicity determined by the direction of insonation by the ultrasound transducer. The iliopsoas, direct head of the rectus femoris, and sartorius origins are well seen, while the indirect head of rectus femoris may appear artificially hypoechoic due to anisotropy, as it courses along the lateral margin of the acetabulum. Other tendons that are well evaluated on ultrasound are the adductor tendon origins (longus and brevis), abductor tendon insertions at the greater trochanter, and hamstring tendon origins at the ischium, including the adductor magnus, as well as the iliotibial tract (Fig. 9). Individual tendons may be traced to their respective muscles during real-time examination

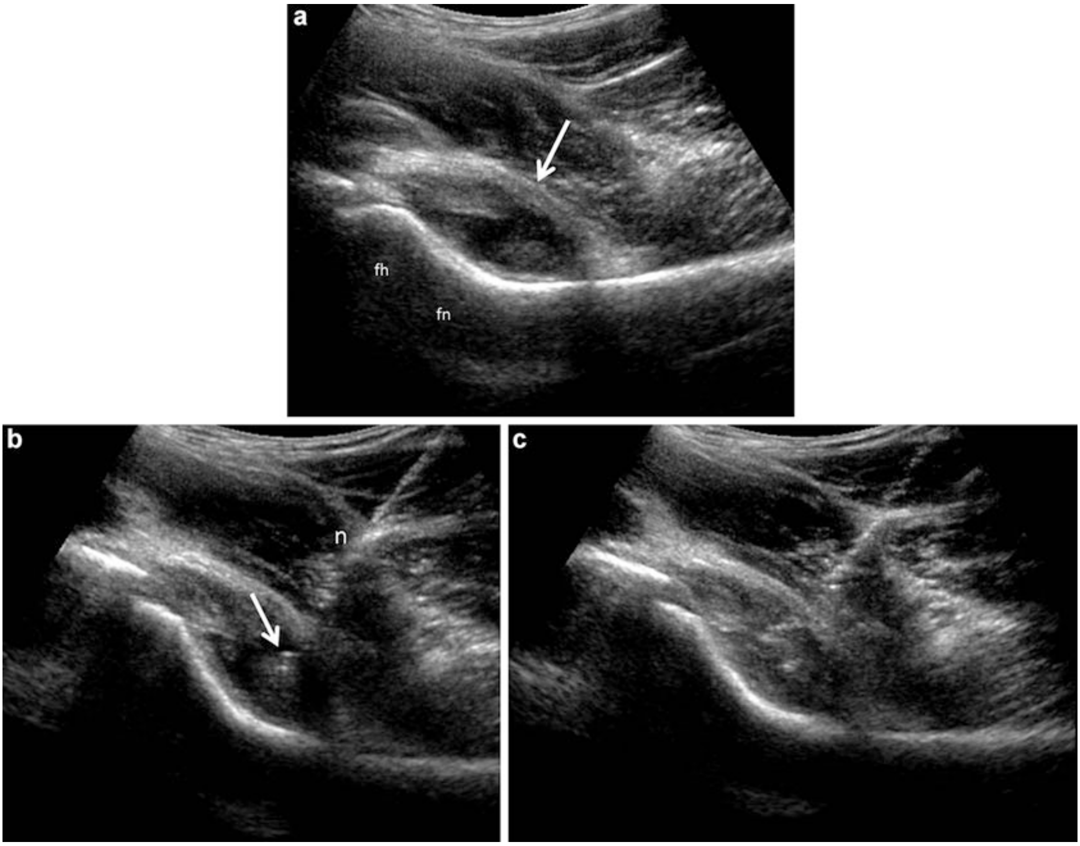


Fig. 3 Joint effusion with ultrasound guided aspiration. (**a**) Long-axis ultrasound image of the hip depicting distension of the anterior capsule (*arrow*), which assumes a convex margin. The material distending the capsule consists of hypoechoic effusion containing low level echoes as well as nodulular echogenic areas of synovial proliferation. The femoral head (*fh*) and femoral neck (*fn*) are labeled. (**b**) A 3.5 in. 20 gauge spinal needle (n) has been placed into the hip joint using a long-axis approach. The needle tip (*arrow*) is positioned within the hypoechoic material noted in (**a**). (**c**) Aspiration of the hypoechoic material confirmed its fluid composition, leaving only the echogenic proliferative synovium

[7–9]. EFOV imaging may be of value in order to better display the myotendinous unit [10].

Knowledge of normal anatomic relationships allows identification of these structures. The sciatic nerve is seen as an echogenic ellipse at the level of the hamstring origin situated superficial to the quadratus femoris and abutting the inferolateral margin of the conjoint tendon (Fig. 10). The nerve is usually well seen below this level as it courses in the intermuscular fascia between medial and lateral hamstrings. Proximal to the ischium, the nerve assumes a flatter configuration as it enters the sacrosciatic notch and courses deep to the piriformis muscle.

Tendinosis is characterized by loss of the normal compact fibrillar architecture [9, 19]. Tendons may appear enlarged and heterogeneous, containing intrasubstance fissures and areas of cystic degeneration (Figs. 11 and 12) [19]. Areas of intra- and peritendinous neovascularity may be apparent, although reduced Doppler sensitivity may be a limiting factor in assessing vascularity. Punctuate areas of dystrophic calcification may occur, as well as more globular deposits in the form of calcium hydroxyapatite. Enthesopatic ossification is often seen about the tendon origins and insertions and should not be mistaken for calcific tendinosis. Correlation with radiographs

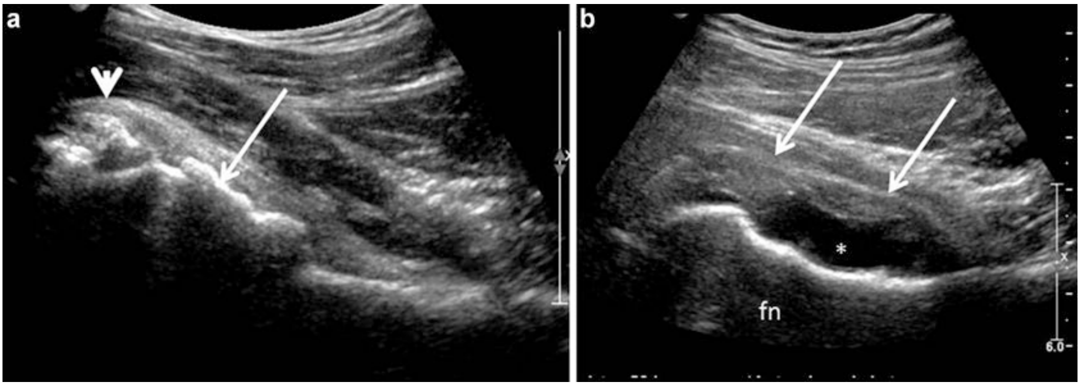


Fig. 4 (**a**) Osteoarthrosis. Hypertrophic changes at the femoral head-neck junction are evident (*arrow*) resulting in loss of the smooth convex/concave transition. In addition, a rounded echogenic area contiguous with the anterior joint capsule and overlying the anterior superior labrum (*arrowhead*) corresponds to a joint body. (**b**) Post-operative deformity. The patient has had previous resection of a CAM lesion with an irregular concave margin of the femoral head/neck (*fn*) junction. A moderate effusion (*) is present with proliferative synovium or thickened capsule (*arrows*)

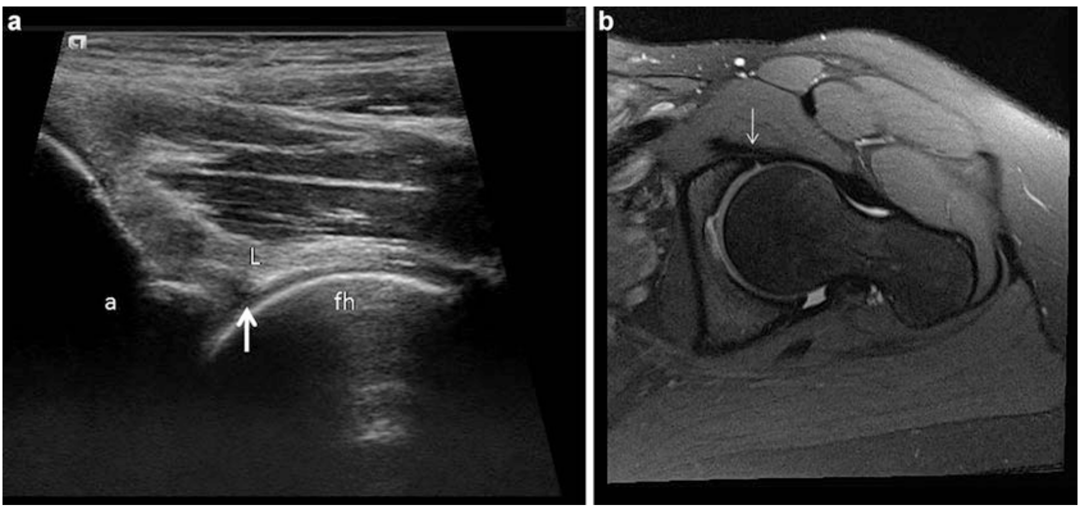


Fig. 5 Labral tear. (**a**) Long-axis ultrasound image of the hip showing an obliquely oriented hypoechoic fissure (*arrow*) extending through the deep surface of the anterior labrum (*L*). The acetabulum (*a*) and femoral head (*fh*) are labeled. (**b**) Corresponding oblique axial fat suppressed proton density image shows fluid imbibition into an obliquely oriented intrasubstance tear (*arrow*) in the same patient and at approximately the same anatomic level

may be necessary to distinguish these. Calcific tendinosis can occur about the hip, most frequently affecting the abductor insertions and rectus femoris origin, although these calcific deposits can occur anywhere about the musculoskeletal system (Fig. 13) [9, 20]. Tears appear as discrete hypoechoic defects within the tendon (Fig. 12). Chronic tears may also be attritional with diffuse tendon thinning. Complete tears are associated with retraction of the myotendinous unit often resulting in shadowing at the retracted stump.

In contrast to muscle, the myotendinous junction appears as a compact curvilinear echogenic reflector, which may be central or along the margin of the muscle and can be traced in continuity

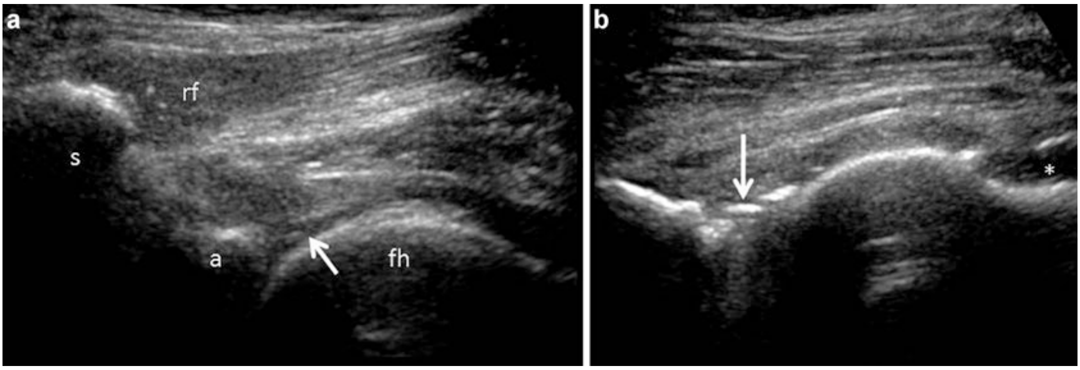


Fig. 6 Fluid/microbubble imbibition into a labral tear. (**a**) Patient with groin pain sent for ultrasound guided therapeutic injection. An obliquely oriented fissure (*arrow*) is present within the labrum, suggesting the possibility of a non-displaced labral tear. The direct head of the rectus femoris (*rf*) is present in this image and labeled. The anterior inferior iliac spine (*s*), acetabulum (*a*) and femoral head (*fh*) are labeled. (**b**) Following the therapeutic injection, there is fluid distension of the anterior joint capsule (*) and linear echogenic material present within the labral substance (*arrow*) that was not present on preliminary imaging, relating to fluid and microbubble imbibition into the labral substance and confirming the presence of a tear

Fig. 7 Professional football player with semitendinosus strain. (**a**) Transverse sonogram of the semitendinosus muscle belly contrasting edematous (*e*) and normal (*n*) muscle. Normal muscles appear as a hypoechoic (*dark*) background containing fine linear echogenic structures corresponding to perimyseal connective tissue. The epimyseal connective tissue appears echogenic boundary (*arrow*). Edematous muscle (*e*) due to a grade 1 strain, as in this case, appears echogenic and often displays increased volume. (**b**) Long-axis extended field-of-view sonogram in the same individual shows the normal appearing muscle (*n*) with linearly oriented perimyseal connective tissue with a hypoechoic background in contrast to the superficial grade 1 strain, displaying increased echogenicity (*e*). The epimyseal connective tissue is denoted by *arrows*

to their respective tendons. Infiltrative processes, such as edema and hemorrhage, can result in increased muscle size and echogenicity in either a heterogeneous or uniform distribution while maintaining the fascicular architecture of the muscle, such as is seen in a grade 1 muscle strain (Figs. 7 and 14). Thickening of the epimyseal connective tissue and/or hypoechoic fluid/edema tracking along the epimysium may be present. Higher-grade injuries are associated with disruption of muscle fibers resulting in the loss of normal fascicular architecture. Localized heterogeneous or hypoechoic collections may be seen with discrete tears or as heterogeneous masses with peripheral vascularity in the case of an organizing hematoma [21–24]. Ultrasound is a sensitive technique to detect calcification/ossification in the case of developing myositis ossificans, appearing as linear echoic foci often with posterior acoustic shadowing [25].

Seromas appear as discretely marginated intra- or perimuscular anechoic/hypoechoic collections

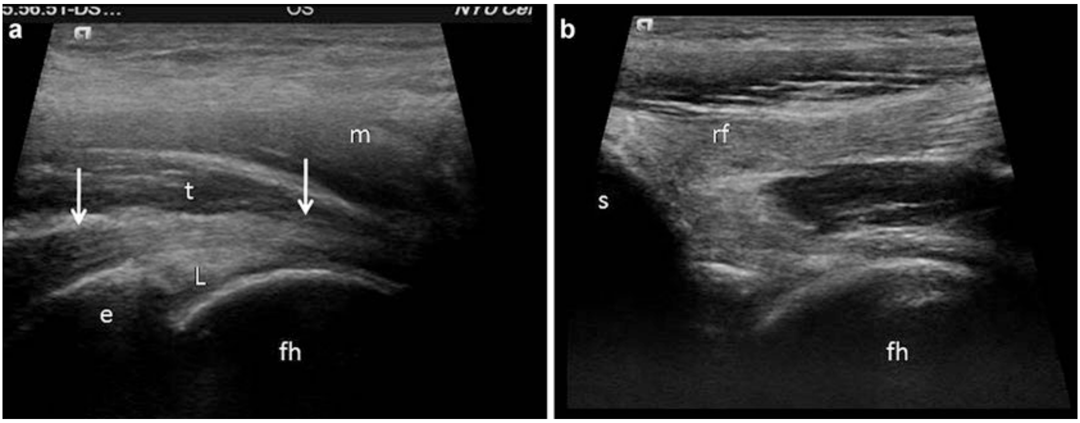


Fig. 8 Normal tendon. (**a**) Iliopsoas tendon. Longitudinal ultrasound image of the iliopsoas tendon (*t*) as it passess over the iliopectineal eminence (*e*) and capsular labral complex (*L*). The tendon appears as a compact linear fibrillar structure that displays greatest echogenicity centrally and is more hypoechoic on either proximal or distal end (*arrows*) due to anisotropy. The overlying muscle (*m*) appears hypoechoic. The femoral head (*fh*) is labeled. (**b**) Longitudinal ultrasound image in the same patient obtained slightly more laterally shows the origin of the direct head of the rectus femoris (*rf*) arising from the anterior inferior iliac spine (*s*). Again the tendon appears compact, echogenic and fibrillar. The femoral head (*fh*) is labeled

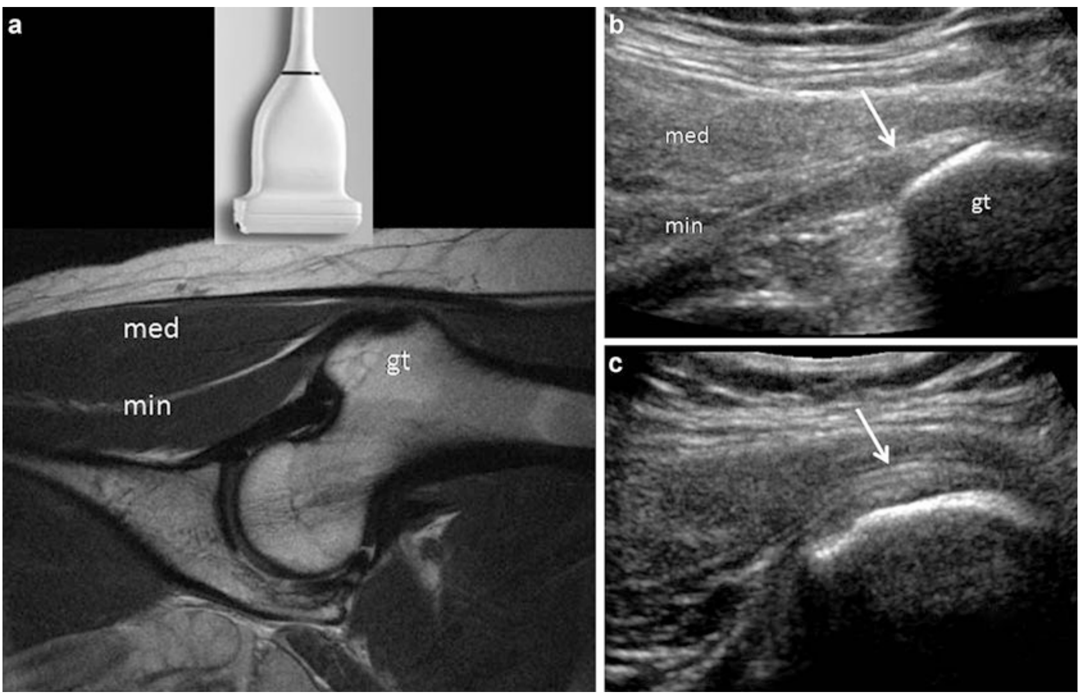


Fig. 9 Normal abductor muscles. (**a**) The abductor group is often best evaluated with the patient in a decubitus position and with the transducer positioned over the greater trochanter (*gt*), as depicted in this coronal proton density image. In this case the gluteus minimus tendon/muscle (*min*) is evident in the plane of the transducer, as is the gluteus medius muscle (*med*). (**b**) Sonogram obtained in the same plane as (**a**) depicts the echogenic gluteus minimus tendon (*arrow*) as it inserts on the anterior facet of the great trochanter (*gt*). The gluteus minimus (*min*) and medius (*med*) muscles are labeled. (**c**) Sonogram obtained in the logitudinal plane, slightly more posteriorly on the lateral facet shows part of the gluteus medius tendon insertion (*arrow*) onto the lateral facet of the greater trochanter

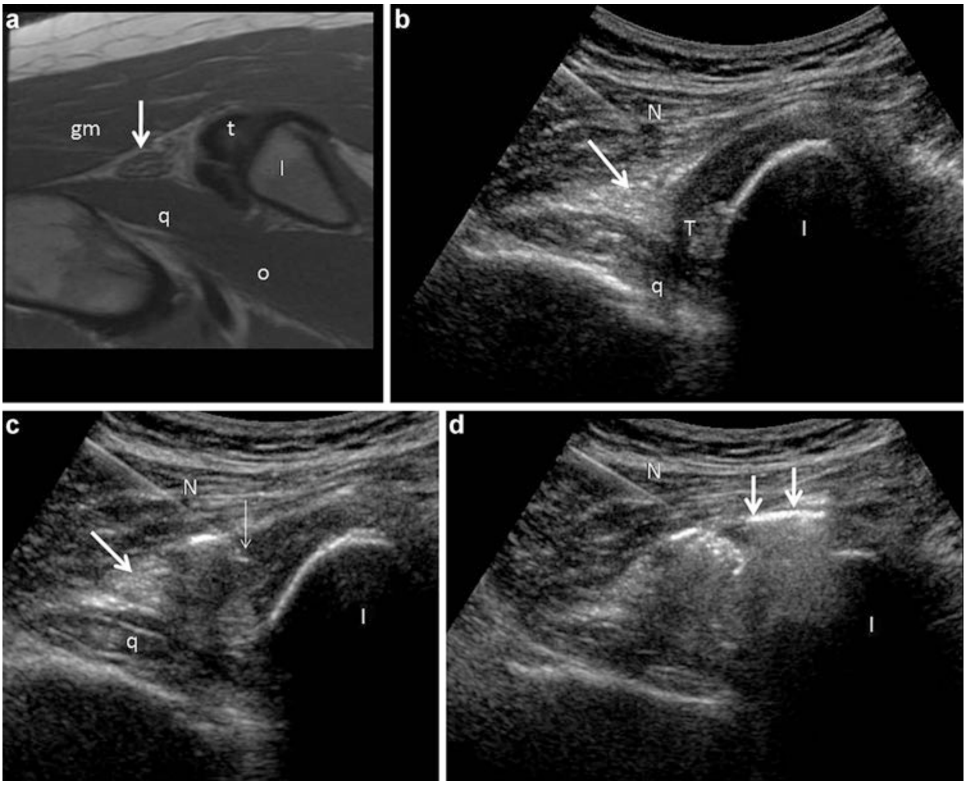


Fig. 10 Hamstring origin in short axis in 35 year old marathon runner. (**a**) Axial FSE proton density image depicts the hamstring origin (*t*), which appears tendinotic (containing intermediate background signal intensity), the sciatic nerve (*arrow*), the gluteus maximus (*gm*) and potions of the quadratus femoris (*q*) and obturator extenernus (*o*) muscle groups. The ischium (*I*) is labeled. (**b**) Transverse sonogram obtained in the same patient during a guided peritendinous injection, using a lateral approach. The needle (*N*) is positioned within the belly of the gluteus maximus above the sciatic nerve (*arrow*). The nerve appears as a heterogeneous echogenic ellipse overlying the quadratus femoris muscle (*q*) and closely related to the inferior margin of the hamstring tendon origin (*t*). The hamstring tendon origin appears largely hypoechoic, likely due to anisotropy. The ischium (*I*) is labeled. (**c**) The needle (*N*) has been advanced to the lateral margin of the hamstring tendon origin above the sciatic nerve (*arrow*). The tip of the needle (*thin arrow*) is depicted, as are the ischium (*I*) and quadrates femoris (*q*). (**d**) Microbubbles and fluid (*arrows*) from the injectate surround the tendon origin and adjacent sciatic nerve during this peritendinous injection. In the author's experience, the optimal distribution is peritendinous avoiding intratendinous and intramuscular intravasation. Portions of the ischium (*I*) and tendon are obscured because of artifact produced by the microbubbles

that may inhibit rehabilitation following a muscle injury (Fig. 15). Ultrasound provides a convenient method to perform percutaneous aspiration of these collections which has been postulated to help facilitate recovery [24]. Muscle atrophy can likewise present as increased muscle echogenicity but with diminished muscle volume. Differentiating atrophy and edema on ultrasound can be challenging in the setting of an acute or chronic injury. Magnetic resonance imaging provides a more sensitive assessment of muscle integrity in this setting. Dynamic assessment following muscle injury using ultrasound can be invaluable in determining the apposition of the torn muscle ends while performing provocative maneuvers [17].

Similarly, demonstrating muscle hernias through fascial defects may require provocative maneuvers to manifest [26]. Chronic muscle tears sometimes may become more conspicuous following or during injection therapy performed under ultrasound guidance as the injected material can decompress into the tear, taking a path of least resistance (Fig. 16).

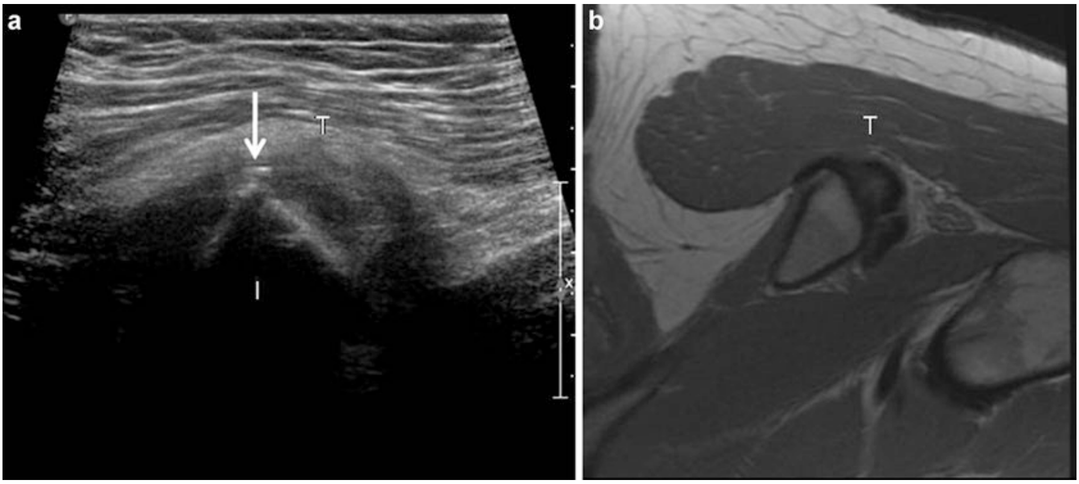


Fig. 11 Hamstring tendinosis. (**a**) short-axis ultrasound image of the hamstring origin (*T*) shows an inhomogeneous tendon with a small calcification (*arrow*) present along the superficial margin of the tendon. The ischium (*I*) is labeled. (**b**) Corresponding axial FSE proton density image show heterogeneous hamstring tendon origin (*T*), compatible with tendinosis. Small dystrophic calcifications are generally not appreciated on MR

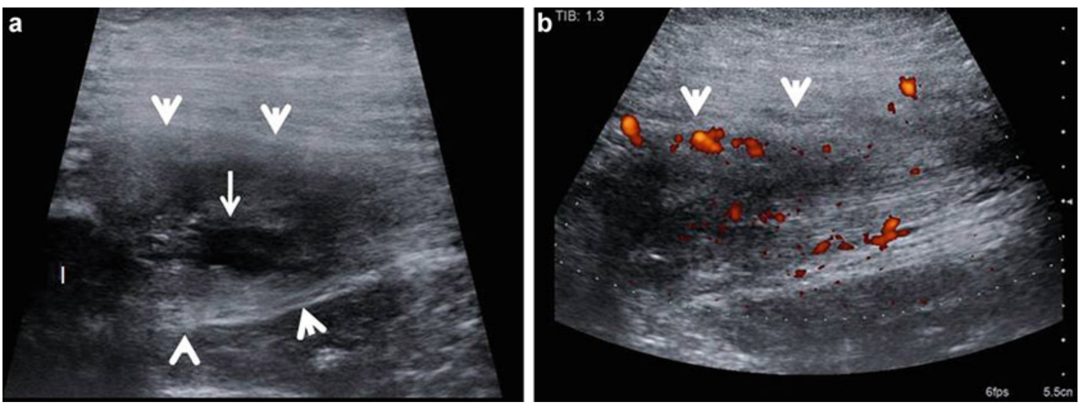


Fig. 12 Hamstring tendinosis with tear. (**a**) Longitudinal ultrasound image of the proximal hamstring tendon (*arrow heads*) origin shows it to be expanded and heterogeneous with a intrasubtance tear (*arrow*), appearing as a discretely marginated hypoechoic area within the tendon substance. The ischium (*I*) is labeled. (**b**) Longitudinal power Doppler ultrasound image of the proximal hamstring tendon (*arrow heads*) origin. Increased vascularity (*red* to *orange* hues) on power Doppler imaging can be seen due to angiofibroblastic proliferative response

Areas of focal scarring appear as poorly marginated echogenic areas within the muscle.

Fluid Collections

Aside from joint effusions, a variety of fluid collections can be seen about the hip. Paralabral cysts appear as hypoechoic uni- or multilocular collections that may be seen in continuity with a tear of the adjacent labrum and are contained within a fibrous pseudocapsule [27]. They typically contain gelatinous material and can be hard to palpation. Consequently these cysts will not redistribute following compression by the transducer. Ganglion cysts have a similar sonographic appearance and can be difficult to distinguish from a paralabral cyst apart from their location (Fig. 17).

Bursas are recognized by their characteristic anatomic locations [2–9]. The most commonly

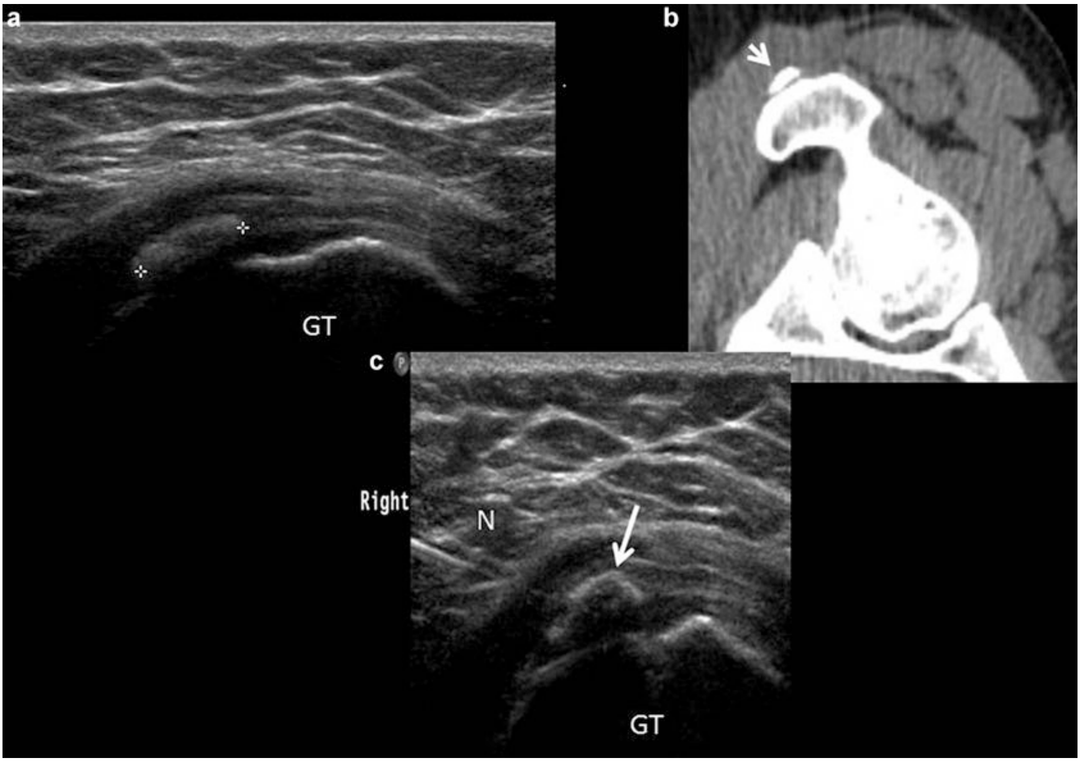


Fig. 13 Calcific tendinosis. (**a**) Long axis ultrasound image over the greater trochanter (*GT*) shows a globular echogenic area (+) within the substance of the gluteus medius tendon. The cortical echo below the echogenic focus is lost due to posterior acoustic shadowing, confirming the calcified nature of the echogenic material. (**b**) Computed tomography of the same hip shows the calcification (*arrow*) adjacent to the greater trochanter. (**c**) A 20 gauge spinal needle (*N*) has been placed into the calcification (*arrow*) using a short axis approach for purposes of aspiration and therapeutic injection. The greater trochanter (*GT*) is labeled

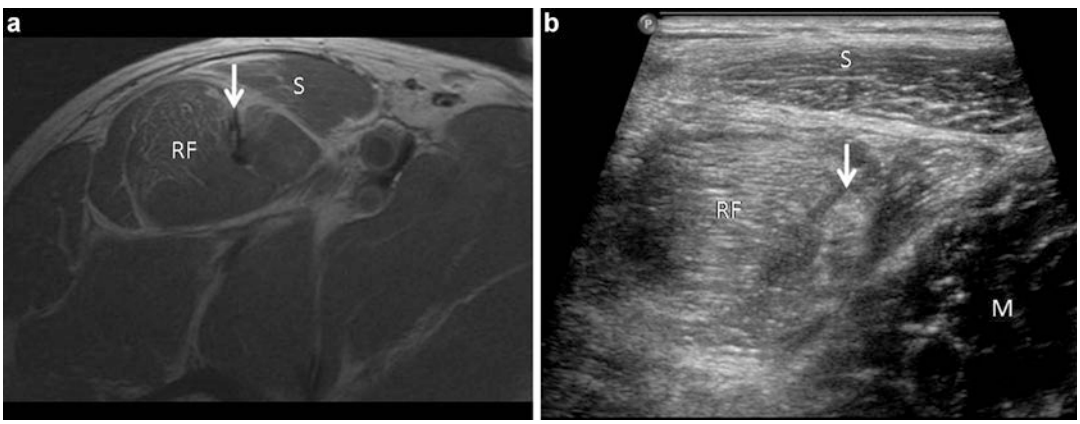


Fig. 14 Muscle edema pattern. (**a**) Axial FSE proton density image in a professional soccer player who sustained a strain injury to the rectus femoris (*RF*) and sartorious (*S*) muscles. Heterogeneous signal intensity is present within the tendon of the indirect head of the rectus femoris (*arrow*). (**b**) Corresponding ultrasound image shows both rectus femoris (*RF*) and sartorius (*S*) displaying increased echogenicity related to infiltrative edema and hemorrhage. Normal adjacent muscle (*M*) can be seen in the same image for comparison. The tendon of the indirect head (*arrow*) is inhomogeneous

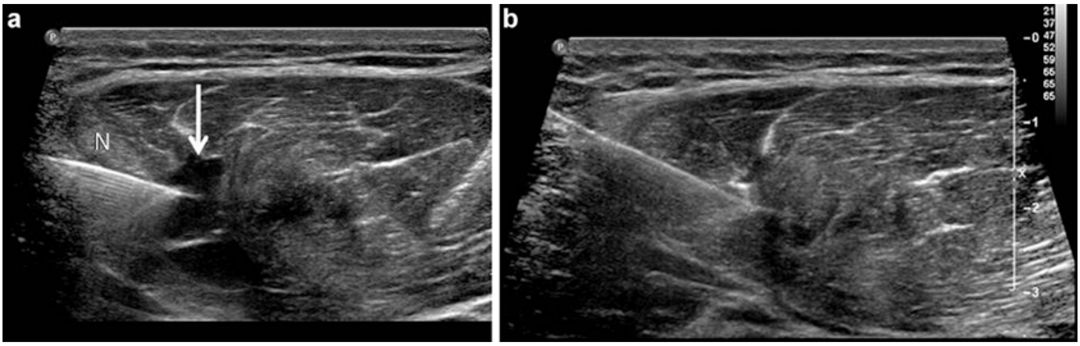


Fig. 15 Muscle tear with aspiration. (**a**) Professional baseball player with myotendinous junction injury to indirect head of the rectus femoris. Short axis ultrasound image of the rectus femoris displays distortion of muscle morphology with patchy areas of increased echogenicity (muscle edema pattern) and eccentric discretely marginated hypoechoic collection (*arrow*) corresponding to a muscle tear with seroma formation. A needle (*N*) has been placed into the collection under ultrasound guidance for purposes of aspiration. (**b**) Corresponding transverse sonogram following aspiration depicting decompression of the seroma. The patient was able to return to play 24 h following therapeutic aspiration

assessed bursas about the hip include iliopsoas, greater trochanteric, and ischiofemoral bursas (Figs. 18 and 19). The greater trochanteric bursa most often refers to the bursa situated deep to the gluteus maximus and overlying posterior facet bare area of the greater trochanter and gluteus medius muscles. Several bursas have been recognized about the greater trochanter, which may be distended individually or in combination with others. These may be targeted for percutaneous aspiration and/or injection under ultrasound guidance. Not infrequently, an adventitial bursa deep to the iliotibial tract may be present at the level of the greater trochanter.

Other miscellaneous collections include hematomas, seromas, and abscesses. The Morel-Lavallée lesion represents a large seroma at the junction of the subcutaneous fat and iliotibial tract, often the result of a degloving injury (Fig. 20) [28]. The presence of hyperemia on color flow imaging can be helpful in distinguishing whether a collection is infectious and/or inflammatory in etiology [29].

Injections

Ultrasound is well suited to performing a variety of selective injections [30]. These include trigger point injections, joint aspiration/injections, aspiration/injection of cysts, bursa and other fluid collections, percutaneous treatment of calcific tendinosis, as well as intratendinous therapy (i.e., injection of platelet-rich concentrates; Fig. 21).

Ultrasound-guided injection and/or aspiration allows administration of therapeutic agent without the use of ionizing radiation. This is particularly helpful in younger individuals with suspected labral pathology or in situations where multiple intra-articular injections are required, such as in patients receiving viscosupplementation [31, 32]. Ultrasound provides an ideal method to aspirate fluid collections about the hip [33]. The hip is imaged in long axis with the needle positioned in the plane of the transducer, thereby avoiding the neurovascular structures (Figs. 1 and 3) [31].

Observing in real time ensures that collections are aspirated to completion and allows one to observe the distribution of injected material. This is particularly important in cases where the collections may be multiloculated. Ultrasound is the method of choice to aspirate and inject paralabral cysts and other para-articular collections, such as iliopsoas bursa or ganglion cysts. Ultrasound guidance has also become the method of choice to perform a variety of peritendinous injections, where real-time monitoring of injected and/or aspirated material is of value [34–36].

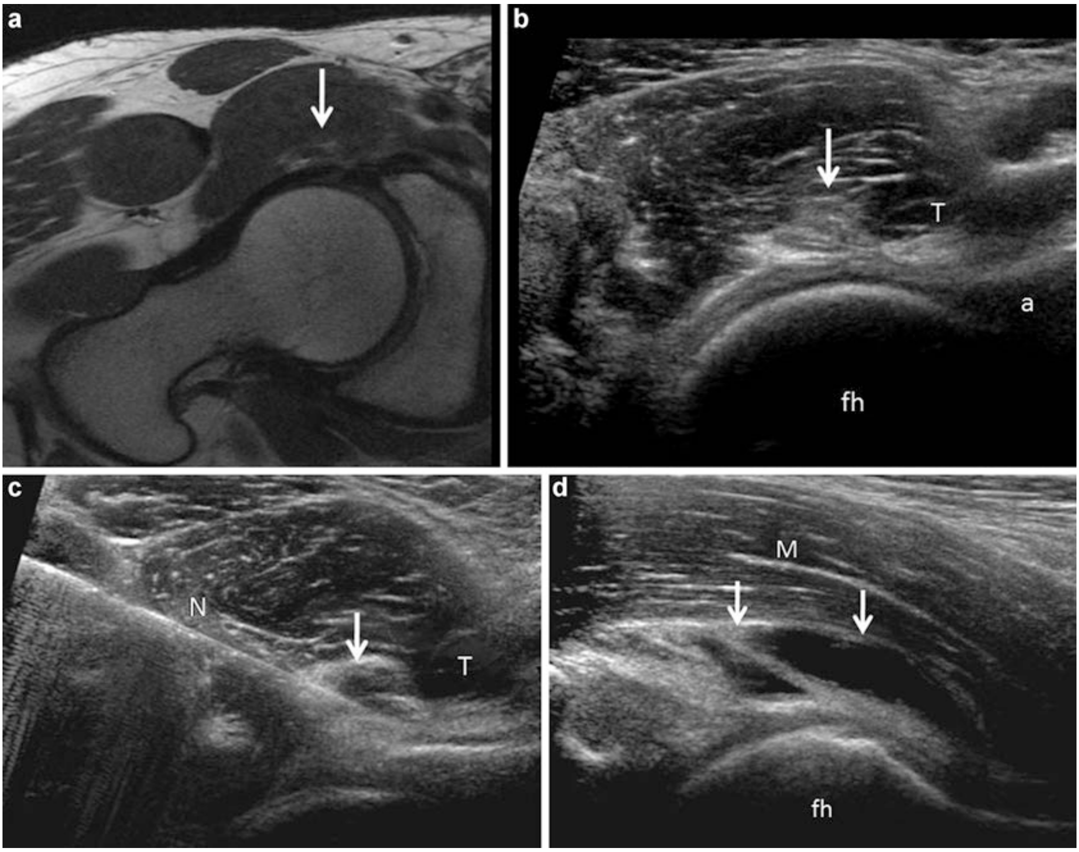


Fig. 16 Occult muscle tear in a high school soccer player with chronic groin pain. (**a**) Axial FSE proton density image shows myotendinous strain with increased signal intensity along the deep surface of the iliopsoas muscle at the level of the iliopectineal eminence (*arrow*). (**b**) Short axis ultrasound image showing an ill-defined area of increased echogenity (*arrow*), lateral to the tendon, corresponding to the abnormality seen on magnetic resonance imaging. The femoral head (*fh*) and iliopsoas tendon (*T*) and adjacent acetabulum (*a*) are labeled. (**c**) Short axis ultrasound image showing a 22 gauge spinal needle (*N*) positioned adjacent to the iliopsoas tendon (*T*) with fluid (*arrow*) beginning to preferentially distend an intrasubstance tear within the muscle. (**d**) Long axis view of the hip at the level of the region of muscle edema pattern depicted in (**a**). Following needle removal and therapeutic injection, distension of a thick-walled cavity (*arrows*) within the muscle became apparent separate from the liopsoas bursa and within the muscle substance, compatible with a chronic tear. The femoral head (*fh*) and overlying muscle (*M*) are labeled

The most commonly requested peritendinous injections in the author's experience are about the iliopsoas tendon, abductor tendon, and hamstring tendon origin (Figs. 10, 18, and 19). In the first two of these injections, the needle is directed to the iliopsoas or greater trochanteric bursa, respectively. These injections can be fairly straightforward when the bursa is distended. They become more challenging when there is no preexisting bursal distension. One must then employ anatomic landmarks and test injections with anesthetic for localization. Injections at the hamstring origin are generally peritendinous since no true anatomic bursa exists. An adventitial bursa may occasionally be present over the ischium.

A lateral approach, while scanning the tendons in short axis, is preferred for each of these injections [30]. The needle is positioned deep to the iliopsoas tendon at the level of the iliopectineal eminence, or it is directed toward the posterior

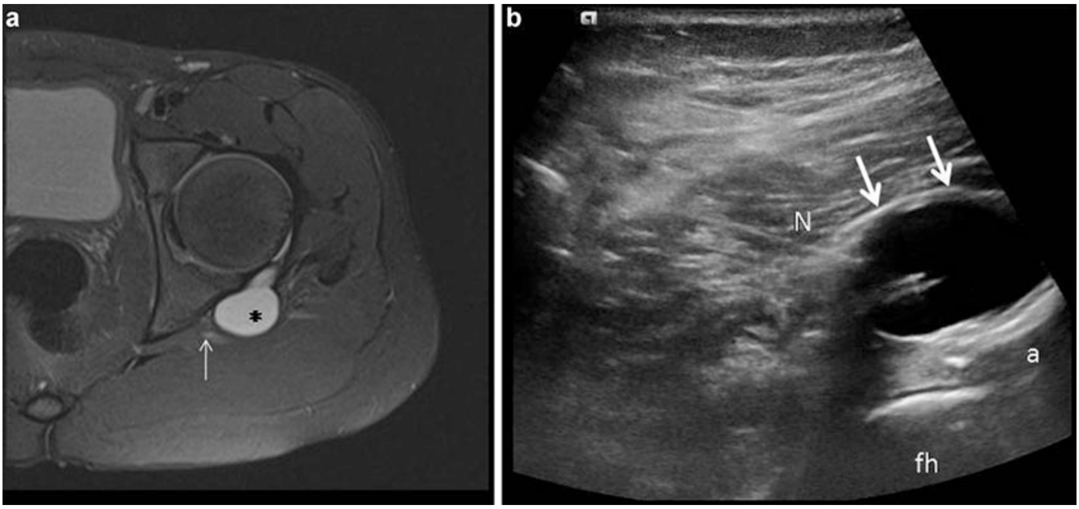


Fig. 17 Dancer with painful left posterior ganglion cyst compressing sciatic nerve. (**a**) Axial fat-suppressed T2 weighted image of the left hip showing a large unilocular posterior paralabral fluid collection (*) which abuts the sciatic nerve (*arrow*). (**b**) Short axis ultrasound of the posterior hip obtained at approximately the same anatomic level as depicted in (**a**). A 20 gauge spinal needle (*N*) has been positioned within the cyst (*arrows*) under ultrasound guidance with the patient in a prone position for purposes of therapeutic aspiration. The cyst appears as a unilocular anechoic (homogeneously dark) collection. The acetabulum (*a*) and fenoral head (*fh*) are partially visualized. The sciatic nerve is not visualized in this image

facet of the greater trochanter for a trochanteric bursal injection. When performing intratendinous therapy, the injected material may decompress into an adjacent bursa. The distribution of injected material during intratendinous therapy can often be determined by the pattern of intratendinous microbubbles within the myotendinous unit. This may obviate the need to perform a peppering procedure in order to distribute the injectate [37]. Likewise, aspiration of symptomatic calcific deposits can be performed under real-time guidance (Fig. 13) [38].

Single and dual-needle techniques have been described to lavage and aspirate the calcific material in patients with rotator cuff calcific tendinosis [39, 40]. Simple mechanical fenestration, using a 25-gauge needle alone, has also been advocated as a method of treatment. All these techniques appear to be comparably effective. Generally I use a single-needle technique, where the needle is positioned within the calcification, employing pulse lavage of the calcification initially using local anesthetic, followed by sterile saline. This will often decompress the calcification. The procedure is completed with local fenestration and peritendinous steroid/anesthetic injection.

Targeted injections can be performed in some cases of compressive neuropathy [41–43]. These include patients with meralgia paresthetica or piriformis-related symptoms (Figs. 22 and 23). The sciatic nerve can be traced proximally to the sacrosciatic notch and the level of the piriformis muscle where selective perineural/intramuscular injection can be performed. Likewise, the lateral femoral cutaneous nerve can be identified and injected near the level of the iliac crest.

Certain injections for which ultrasound guidance is currently not the method of choice may become available as multimodality registration techniques evolve. The most common method to accomplish this entails use of an electromagnetic tracker to localize the transducer orientation in space along with identification of at least three internal fiduciary markers in common to both the ultrasound and either MR or CT image volume (Fig. 24). The ultrasound and MR/CT image volumes can then be co-registered (usually with several mm accuracy) allowing the second modality

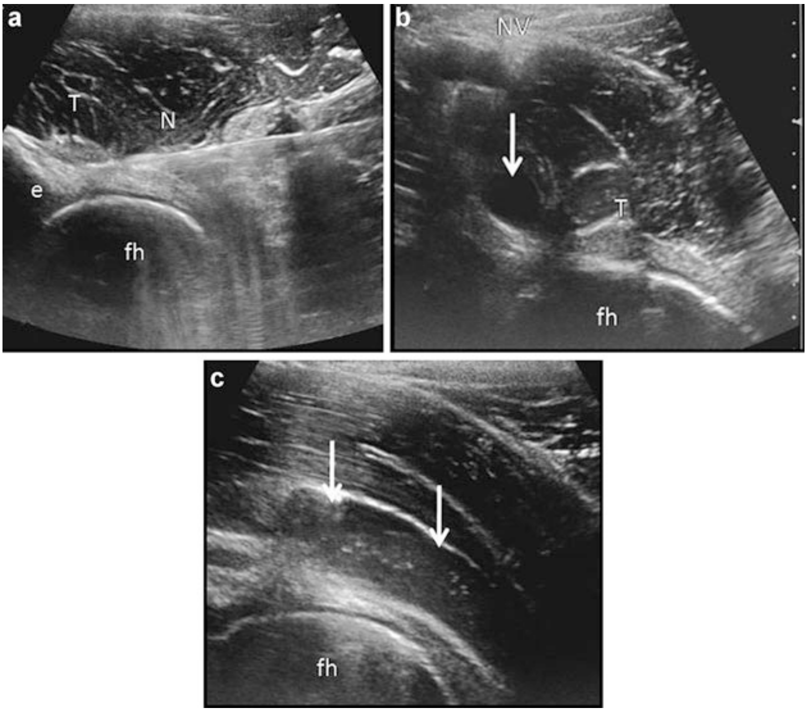


Fig. 18 Iliopsoas bursal injection. (**a**) A needle (*N*) is positioned deep to the iliopsoas tendon (*T*) using a short-axis approach at the level of the iliopectineal eminence (*e*). This approach enables one to avoid the neurovascular structures. The femoral head (*fh*) is labeled. (**b**) Short axis view of the hip at same level as depicted in (**a**). Distension of the iliopsoas bursa is seen as a fluid collection (*arrow*) medial to the tendon (*T*) and deep to the neurovascular structures (*NV*). The femoral head (*fh*) is labeled. (**c**). Long axis view of the hip. The distended bursa (*arrows*) is well seen on this long axis view, containing low-level echoes from the injected therapeutic mixture. The femoral head (*fh*) is labeled

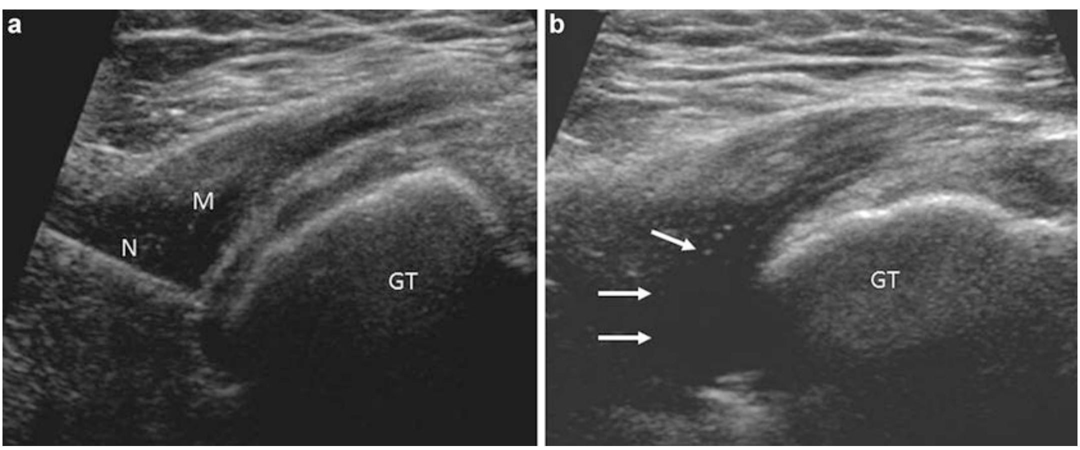


Fig. 19 Greater trochanteric bursa injection. (**a**) Transverse sonogram of the greater trochanter shows a needle (*N*) positioned at the posterior facet deep to the gluteus maximus muscle (M), corresponding to the bare area of the greater trochanter (*GT*). (**b**) Transverse sonogram at same level as (**a**). Following needle removal there is distension of the greater trochenteric bursa (*arrow*) with hypoechoic fluid (*arrows*). The greater trochanter (*GT*) is labeled

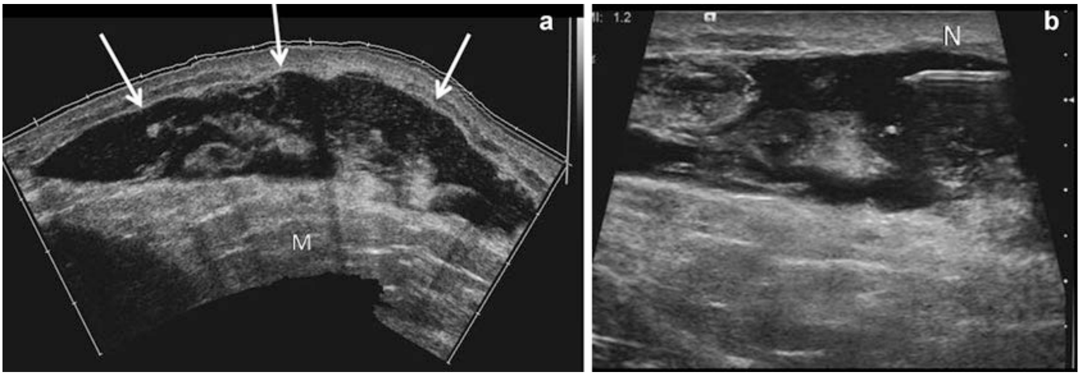


Fig. 20 Large hematoma/seroma over right buttock following motorcycle injury (Morel Lavallee). (**a**) Extended field-of-view transverse image over the right buttocks shows a large complex fluid collection (*arrows*) overlying the gluteal muscles (*M*) and iliotibial tract. The collection contains fluid and solid components. (**b**) Small field-of-view transverse image over the collection obtained during aspiration. A needle (*N*) is visualized within the fluid collection during ultrasound guided aspiration. These collections frequently recur following aspiration

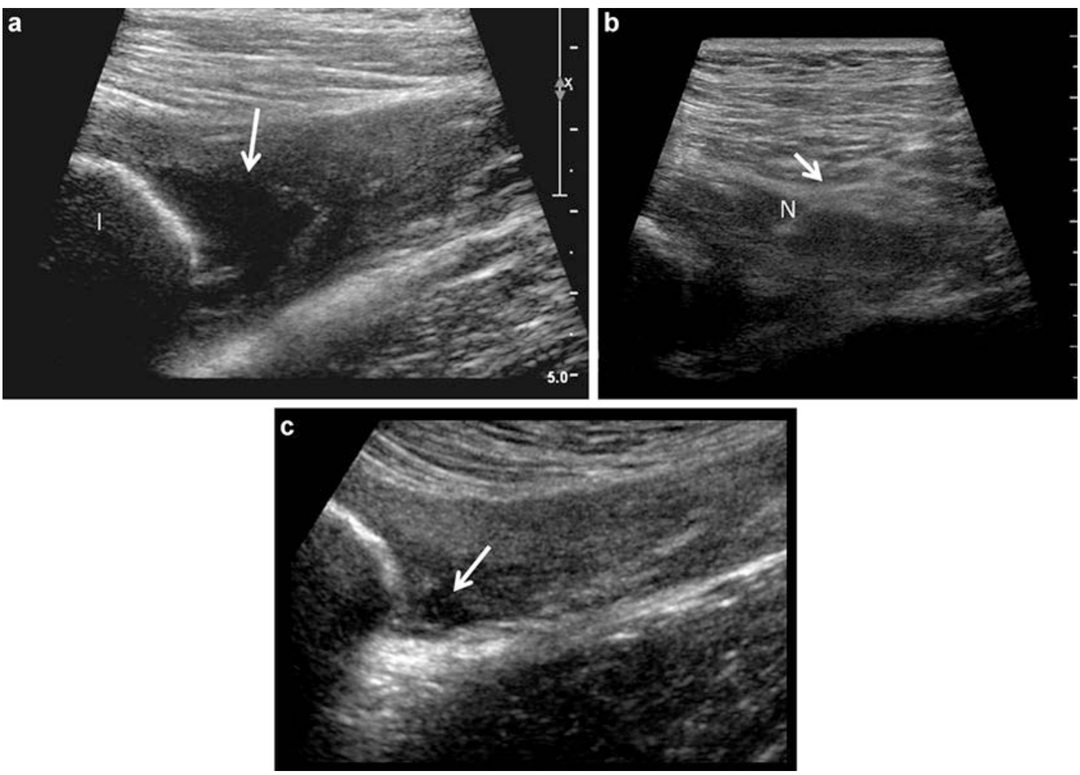


Fig. 21 Hamstring platelet rich plasma injection. (**a**) Initial longitudinal sonogram at the hamstring tendon origin shows a high grade partial tear (*arrow*) which is seen as a discretely marginated hypoechoic area in the otherwise echogenic tendon. The ischium (*I*) is labeled. (**b**) A needle (*N*, *arrow*) has been placed into the tendon in long axis for purposes of tendon fenestration and injection of the platelet rich concentrate. (**c**) Scan obtained at 3 months follow-up shows near complete resolution of the tear (*arrow*)

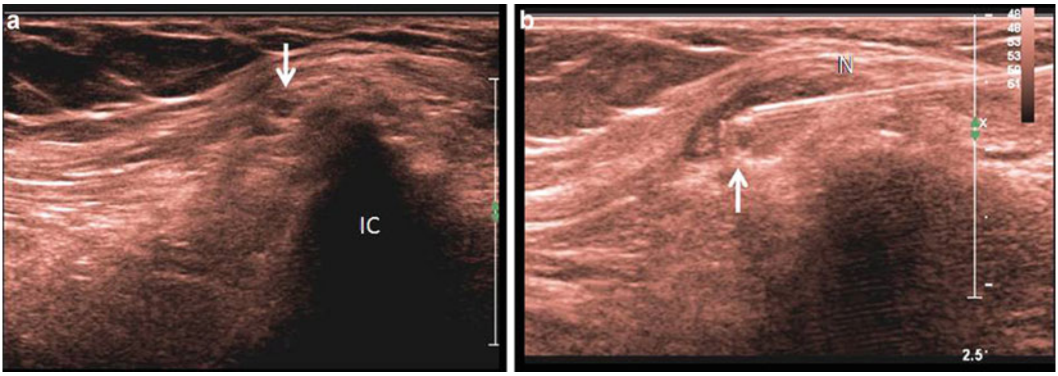


Fig. 22 Eighty four year old female with meralgia paresthetica. (**a**) Transverse sonogram obtained at the level of the anterior superior iliac crest (*IC*) shows a thickened hypoechoic lateral femoral cutaneous nerve in cross-section (*arrow*). The nerve can generally be located by following its course along the superficial margin of the sartorius muscle. (**b**) A needle (*N*) is placed superficial to the nerve (*arrow*) under ultrasound guidance for purposes of perineural injection

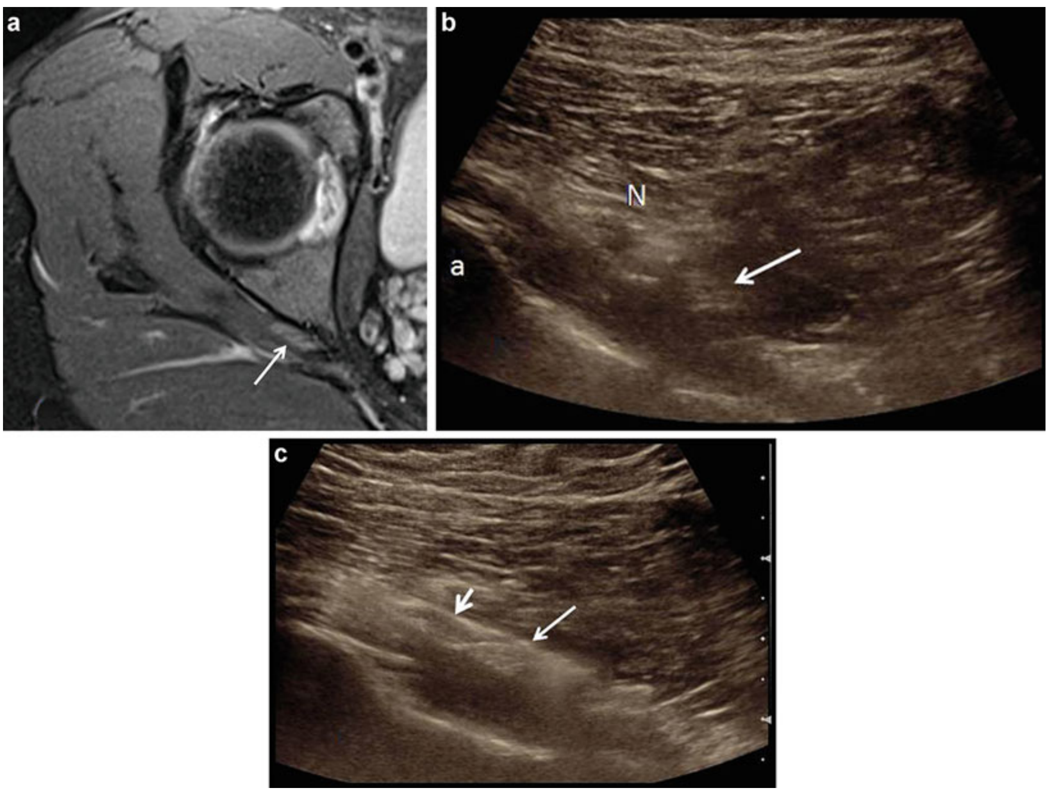


Fig. 23 Perineural injection in patient with buttock pain. (**a**) Axial T2-weighted fat-suppressed image of the right hip shows mild hyperintensity of the sciatic nerve (*arrow*) as it exits the sacrosciatic notch. (**b**) Colorized gray-scale ultrasound image in short axis in a plane slightly caudal the MR image (**a**). A needle (*N*), which is not well seen, has been positioned in the fat plane to the left of the nerve. The sciatic nerve (*arrow*) is partially obscured. The posterior acetabulum is labeled (**a**). (**c**) Transverse sonogram obtained at same anatomic level as (**b**). Following needle removal, the nerve (*arrow*) is more conspicuous. Injected material (*short arrow*) distends the fat plane and circumferentially surrounds the nerve. In the author's experience, the injectate will typically dissect along the path of the nerve near the injection site

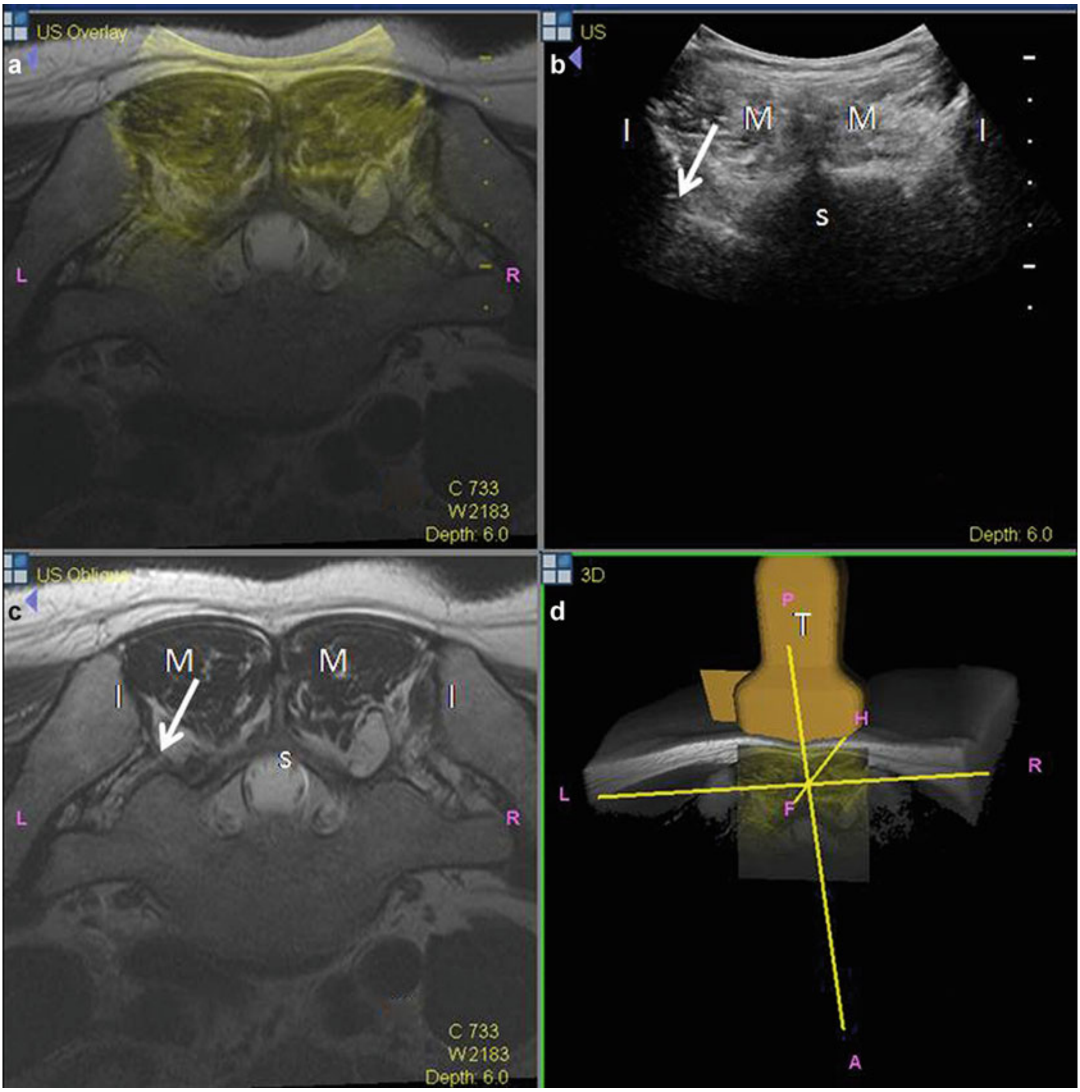


Fig. 24 Ultrasound MR fusion. (**a**) Co-registered axial ultrasound (**b**) and MR (**c**) images for purposes of displaying the sacro-iliac joints. (**b**) Short axis ultrasound image at the level of the lumbosacral junction, showing the posterior margins of the ilia (*I*) and sacrum (*s*). An *arrow* depicts the direction of approach to the left sacro-iliac joint. The paraspinous muscles (*M*) are labeled. (**c**) Corresponding axial proton density MR image showing the posterior ilia (*I*), sacrum (*s*) and paraspinous muscles (*M*). The left sacro-iliac joint (*arrow*) is depicted. (**d**) The transducer (*T*)r position relative to the body axis is displayed in this composite. The directions of the body axes are labeled in pink (*L* left, *R* right, *A* anterior, *P* posterior, *H* head, *F* foot)

to effectively guide needle placement for the procedure. These fusion techniques using ultrasound are still relatively early in their development. Papers have appeared demonstrating their potential application in the musculoskeletal system [44].

Dynamic Examination

The most commonly requested dynamic examinations assess both internal and external etiologies for snapping about the hip [44–48]. Snapping

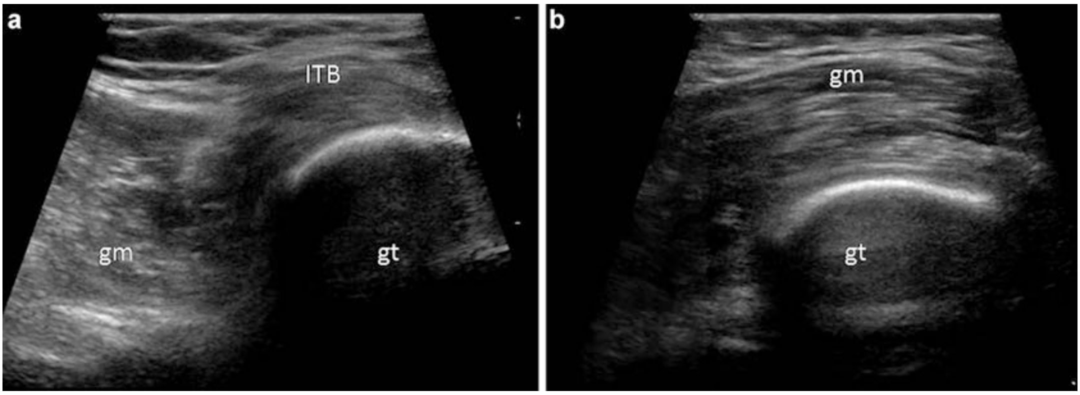


Fig. 25 Snapping ITB/Gluteus maximus in patient with painful snapping over the greater trochanter during hip flexion. (**a**) Short-axis ultrasound image over the greater trochanter (*gt*) shows the gluteus maximus (*gm*) and iliotibial band (*ITB*) in normal relationship. (**b**) During flexion, the iliotibial band and glutleus maximus (*gm*) translate anteriorly and displace the greater trochanter (*gt*) medially. There was a corresponding palpable and audible snap during this maneuver

along the anterior aspect of the hip may be due to intra-articular pathology or subluxation of the iliopsoas tendon (IPT). The latter is also referred to as internal snapping or coxa saltans. A variety of potential causes for internal snapping have been suggested. The diagnosis can be made during real-time observation of the IPT during hip flexion external rotation following by internal rotation extension. The transducer is positioned in the oblique axial plane at the level of the iliacus muscle while observing in real time. The lack of the smooth arc of motion during the latter stage of this maneuver is usually indicative of an internal snapping syndrome. The tendon will display a vertical displacement that corresponds to the palpable and sometimes audible snap. Intra-articular etiologies for snapping may not be evident sonographically, since they may result from cartilage flaps or joint bodies.

The most common cause for external snapping refers to the iliotibial tract/gluteus maximus abruptly and often painfully translating over the greater trochanter in going from hip extension to flexion (Fig. 25) [47, 48]. A transducer placed over the greater trochanter in the axial plane while observing flexion extension in real time will allow documentation of the translation. Due to the chronic friction, an adventitial bursa may form deep to the iliotibial tract. Other etiologies have been suggested relating to subluxation of various ligamentous attachments about the hip and are less common [9].

Mechanical impingement can be secondary to intervening bone or soft tissue preventing full range of motion or producing pain with certain maneuvers. Direct conflict between bony surfaces is thought to be the mechanism for pain in the case of femoral acetabular impingement during performance of a flexion/internal rotation (FADIR) maneuver. The real-time nature of ultrasound is amenable to demonstrating various types of mechanical impingement about the hip.

Hardware

Indwelling metallic hardware has a characteristic appearance on ultrasound [48–52]. Metal is a specular reflector often with either a strong reverberation artifact or “dirty” shadow deep to its surface (Fig. 26). This feature allows

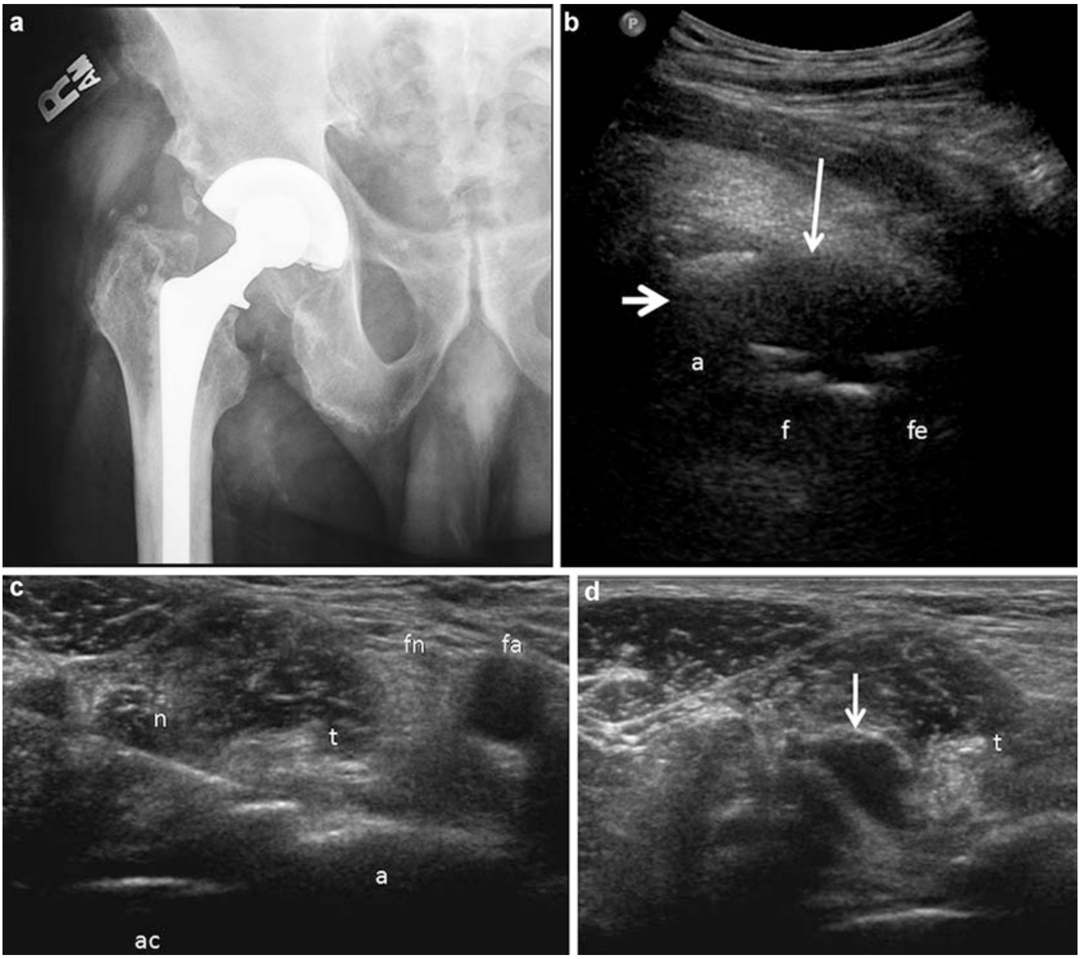


Fig. 26 Patient with hip replacement and groin pain thought to be due to iliopsoas tendinosis. (**a**) Radiograph of a patient with a hybrid right total hip arthroplasty without periprosthetic lucency. (**b**) Long-axis ultrasound image over the prosthesis in the same patient. Visualized acetabular component (*a*), femoral component (*f*) and native femur (*fe*) are labeled. There is distension of the joint capsule (*arrow*) by hypoechoic fluid and/or soft tissue. Notice the echoes deep to the metallic components (*short arrows*) are prominent due to ring-down artifact typically seen deep to metallic structures. (**c**) A needle (*n*) has been placed deep to the iliopsoas tendon (*t*) at the level of the native acetabulum (*a*) using a short-axis approach. A portion of the acetabular component (*ac*) is seen in short axis. The femoral nerve (*fn*) and artery (*fa*) are labeled. (**d**) Transverse ultrasound image near the same anatomic level as (**c**). Following injection and needle removal, a portion of the fluid distended bursa (*arrow*) is evident lateral to the tendon. The inhomogeneous nature of the tendon (t) is more conspicuous

recognition of arthroplasty components, side plates, the proximal portions of intramedullary nails, and screw heads. More importantly, the presence of metal does not interfere with evaluation of the overlying soft tissue, allowing recognition of soft tissue masses and fluid collections, which can occur as complications of indwelling hardware (Fig. 27). Real-time guidance for diagnostic aspirations, biopsies, as well as therapeutic injections can be performed. Likewise, the presence of mechanical impingement of the adjacent soft tissue

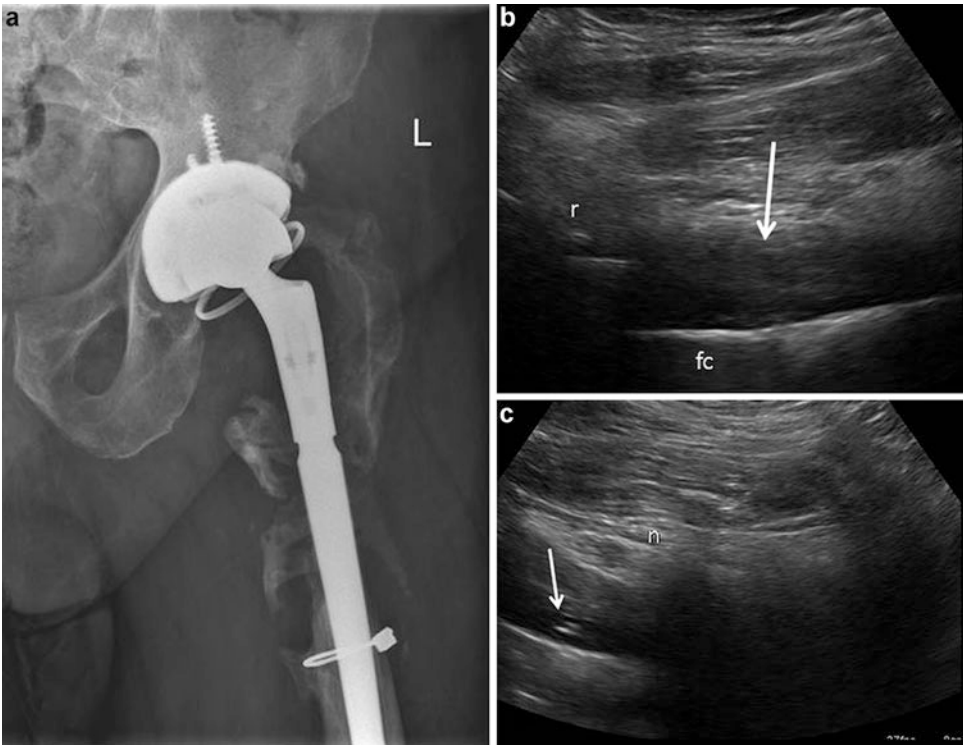


Fig. 27 Hip aspiration. (**a**) AP radiograph of a patient with a constrained left total hip arthroplasty showing severe osteolysis about the femoral component. (**b**) Longitudinal ultrasound of the hip showing the constrained femoral component (*fc*), a portion of the constraining ring (*r*) and distension of the anterior capsule (*arrow*) with fluid and soft tissue. (**c**) A needle (*n*) has been positioned into the fluid. The tip of the needle (*arrow*) is evident within the effusion. 45 cc of serosanguinous fluid was aspirated from the joint

can be determined, such as in IPT impingement by an oversized acetabular component.

Summary

Despite some limitations, ultrasound can display a variety of pathologic conditions about the hip. The real-time capability allows assessment of conditions elicited by provocative maneuvers (i.e., snapping syndromes). It provides guidance for performance of selective interventional procedures and it is not subject to artifact introduced by indwelling metallic hardware, which often limit the utility of computed tomography and MRI. It therefore will continue to play an increasingly important diagnostic and therapeutic role in patients with hip pain.

References

1. Klauser AS, Tagliafico A, Allen GM, Boutry N, Campbell R, Court-Payen M, Grainger A, Guerini H, McNally E, O'Connor PJ, Ostlere S, Petroons P, Reijnierse M, Sconfienza LM, Silvestri E, Wilson DJ, Martinoli C. Clinical indications for musculoskeletal ultrasound: a Delphi-based consensus paper of the European Society of Musculoskeletal Radiology. Eur Radiol. 2012;22(5):1140–8. doi:10.1007/s00330-011-2356-3.
2. Martinoli C, Garello I, Marchetti A, Palmieri F, Altafini L, Valle M, Tagliafico A. Hip ultrasound. Eur J Radiol. 2012;81(12):3824–31.
3. Jacobson JA, Bedi A, Sekiya JK, Blankenbaker DG. Evaluation of the painful athletic hip: imaging options and imaging-guided injections. AJR Am J Roentgenol. 2012;199(3):516–24.
4. Campbell R. Ultrasound of the athletic groin. Semin Musculoskelet Radiol. 2013;17(1):34–42.

5. Weber AE, Jacobson JA, Bedi A. A review of imaging modalities for the hip. Curr Rev Musculoskelet Med. 2013;6(3):226–34. doi:10.1007/s12178-013-9174-y. PMID: 23784063 [PubMed – in process].
6. Friedman T, Miller TT. MR imaging and ultrasound correlation of hip pathologic conditions. Magn Reson Imaging Clin N Am. 2013;21(1):183–94. doi:10.1016/j.mric.2012.09.002. Epub 2012 Oct 23. Review.
7. Finlay K, Friedman L. Ultrasonography of the lower extremity. Orthop Clin North Am. 2006;37(3):245–75. v. Review. PMID: 16846760.
8. Blankenbaker DG, De Smet AA. The role of ultrasound in the evaluation of sports injuries of the lower extremities. Clin Sports Med. 2006;25(4):867–97.
9. Bancroft LW, Blankenbaker DG. Imaging of the tendons about the pelvis. AJR Am J Roentgenol. 2010;195(3):605–17.
10. Lin EC, Middleton WM, Teefey SA. Extended field-of-view sonography in musculoskeletal imaging. J Ultrasound Med. 1999;18(2):147–52.
11. Weybright PN, Jacobson JA, Murry KH, Lin J, Fessell DP, Jamadar DA, Kabeto M, Hayes CW. Limited effectiveness of sonography in revealing hip joint effusion: preliminary results in 21 adult patients with native and postoperative hips. AJR Am J Roentgenol. 2003;181(1):215–8.
12. Buck FM, Hodler J, Zanetti M, Dora C, Pfirrmann CW. Ultrasound for the evaluation of femoroacetabular impingement of the cam type. Diagnostic performance of qualitative criteria and alpha angle measurements. Eur Radiol. 2011;21(1):167–75.
13. Lerch S, Kasperczyk A, Warnecke J, Berndt T, Ruhmann O. Evaluation of cam-type femoroacetabular impingement by ultrasound. Int Orthop. 2013;37(5):783–8.
14. Troelsen A, Mechlenburg I, Gelineck J, Bolvig L, Jacobsen S, Søballe K. What is the role of clinical tests and ultrasound in acetabular labral tear diagnostics? Acta Orthop. 2009;80(3):314–8.
15. Sofka CM, Adler RS, Danon M. Sonography of the acetabular labrum; visualization of labral pathology during intraarticular injections. J Ultrasound Med. 2006;25(10):1321–6.
16. Jin W, Kim KI, Rhyu KH, Park SY, Kim HC, Yang DM, Park JS, Park SJ. Ryu KN Sonographic evaluation of anterosuperior hip labral tears with magnetic resonance arthrographic and surgical correlation. J Ultrasound Med. 2012;31(3):439–47.
17. Campbell S, Adler RS, Sofka C. Ultrasound of muscle abnormalities. Ultrasound Q. 2005;21(2):87–94.
18. Hayashi D, Hamilton B, Guermazi A, de Villiers R, Crema MD, Roemer FW. Traumatic injuries of thigh and calf muscles in athletes: role and clinical relevance of MR imaging and ultrasound. Insights Imaging. 2012;3(6):591–601.
19. Adler RS, Finzel KC. The complementary roles of MR imaging and ultrasound of tendons. Radiol Clin N Am. 2005;43:771–807.
20. Sofka CM, Adler RS, Marx R. Utility of ultrasound for the diagnosis of adductor avulsion injury (thigh splints). J Ultrasound Med. 2006;25(7):913–6.
21. Mariani C, Caldera FE, Kim W. Ultrasound versus magnetic resonance imaging in the diagnosis of an acute hamstring tear. PM R. 2012;4(2):154–5. doi:10.1016/j.pmrj.2011.09.010.
22. Westacott DJ, Minns JI, Foguet P. The diagnostic accuracy of magnetic resonance imaging and ultrasonography in gluteal tendon tears–a systematic review. Hip Int. 2011;21(6):637–45.
23. Klauser AS, Martinoli C, Tagliafico A, Bellmann-Weiler R, Feuchtner GM, Wick M, Jaschke WR. Greater trochanteric pain syndrome. Semin Musculoskelet Radiol. 2013;17(1):43–8.
24. Rubin DA. Imaging diagnosis and prognostication of hamstring injuries. AJR Am J Roentgenol. 2012;199(3):525–33.
25. Abate M, Salini V, Rimondi E, Errani C, Alberghini M, Mercuri M, Pelotti P. Post traumatic myositis ossificans: sonographic findings. J Clin Ultrasound. 2011;39(3):135–40.
26. Kramer DE, Pace JL, Jarrett DY, Zurakowski D, Kocher MS, Micheli LJ. Diagnosis and management of symptomatic muscle herniation of the extremities: a retrospective review. Am J Sports Med. 2013. [Epub ahead of print].
27. Mervak BM, Morag Y, Marcantonio D, Jacobson J, Brandon C, Fessell D. Paralabral cysts of the hip: sonographic evaluation with magnetic resonance arthrographic correlation. J Ultrasound Med. 2012;31(3):495–500.
28. Neal C, Jacobson JA, Brandon C, Kalume-Brigido M, Morag Y, Girish G. Sonography of Morel-Lavallee lesions. J Ultrasound Med. 2008;27(7):1077–81. PMID: 18577672.
29. Newman JS, Adler RS, Bude RO, Rubin JM. "Power Doppler" sonography: applications in musculoskeletal inflammatory disease. Am J Roentgenol. 1994;163:385–9.
30. Sofka CM, Collins AJ, Adler RS. Utilization of ultrasound guidance in interventional musculoskeletal procedures: a review from a single institution. J Ultrasound Med. 2001;20:21–6.
31. Sofka C, Saboeiro G, Adler RS. Ultrasound-guided adult hip injections. J Vasc Interv Radiol. 2005;16:1121–3.
32. Mulvaney SW. A review of viscosupplementation for osteoarthritis of the hip and a description of an ultrasound-guided hip injection technique. Curr Sports Med Rep. 2009;8(6):291–4.
33. Wu HP, Vesely TM. Percutaneous drainage of fluid collections in the extremities. Radiology. 1998;208(1):159–65.
34. Adler RS, Buly R, Ambrose R, Sculco T. Diagnostic and therapeutic use of ultrasound guided Iliopsoas peritendinous injections. AJR. 2005;185(4):940–3.
35. Blankenbaker DG, De Smet AA, Keene JS. Sonography of the iliopsoas tendon and injection

of the iliopsoas bursa for diagnosis and management of the painful snapping hip. Skeletal Radiol. 2006;35(8):565–71.
36. McEvoy JR, Lee KS, Blankenbaker DG, Rio AM, Keene JS. Ultrasound-guided corticosteroid injections for treatment of greater trochanteric pain syndrome: greater trochanter bursa versus subgluteus medius bursa. AJR Am J Roentgenol. 2013;201(2): W313–7.
37. Loftus ML, Endo Y, Adler RS. Retrospective analysis of post-injection ultrasound imaging following platelet rich plasma or autologous blood: observational review of anatomic distribution of injected material. AJR. 2012;199(4):W501–5.
38. Crevenna R, Kéilani M, Wiesinger G, Nicolakis P, Quittan M, Fialka-Moser V. Calcific trochanteric bursitis: resolution of calcifications and clinical remission with non-invasive treatment. A case report. Wien Klin Wochenschr. 2002;114(8–9):345–8.
39. Lee KS, Rosas HG. Musculoskeletal ultrasound: how to treat calcific tendinitis of the rotator cuff by ultrasound-guided single-needle lavage technique. AJR Am J Roentgenol. 2010;195(3):638.
40. Sconfienza LM, Viganò S, Martini C, Aliprandi A, Randelli P, Serafini G, Sardanelli F. Double-needle ultrasound-guided percutaneous treatment of rotator cuff calcific tendinitis: tips and tricks. Skeletal Radiol. 2013;42(1):19–24.
41. Tagliafico A, Padua L, Martinoli C. High-resolution ultrasonography in the assessment of meralgia paresthetica: some clarifications are needed. Muscle Nerve. 2012;45(6):922. doi:10.1002/mus.23391. author reply 922. PMID: 22581555.
42. Tagliafico A, Serafini G, Lacelli F, Perrone N, Valsania V, Martinoli C. Ultrasound-guided treatment of meralgia paresthetica (lateral femoral cutaneous neuropathy): technical description and results of treatment in 20 consecutive patients. J Ultrasound Med. 2011;30(10):1341–6.
43. Martinoli C, Miguel-Perez M, Padua L, Gandolfo N, Zicca A, Tagliafico A. Imaging of neuropathies about the hip. Eur J Radiol. 2013;82(1):17–26.
44. Ewertsen C, Săftoiu A, Gruionu LG, Karstrup S, Nielsen MB. Real-time image fusion involving diagnostic ultrasound. AJR Am J Roentgenol. 2013;200(3): W249–55.
45. Wahl CJ, Warren RF, Adler RS, Hannafin JA, Hansen B. Internal coxa saltans (snapping hip) as a result of overtraining: a report of 3 cases in professional athletes with a review of causes and the role of ultrasound in early diagnosis and management. Am J Sports Med. 2004;32(5):1302–9. Epub 2004 May 18.
46. Deslandes M, Guillin R, Cardinal E, Hobden R, Bureau NJ. The snapping iliopsoas tendon: new mechanisms using dynamic sonography. AJR Am J Roentgenol. 2008;190(3):576–81.
47. Bureau NJ. Sonographic evaluation of snapping hip syndrome. J Ultrasound Med. 2013;32(6):895–900.
48. Fantino O, Borne J, Bordet B. Conflicts, snapping and instability of the tendons: pictorial essay. J Ultrasound. 2012;15(1):42–9.
49. Sofka CM, Potter HG, Adler RS, Pavlov H. Musculoskeletal imaging update: current applications of advanced imaging techniques to evaluate the early and long-term complications of patients with orthopaedic implants. HSS J. 2006;2(1):73–7.
50. Hoefnagels EM, Obradov M, Reijnierse M, Anderson PG, Swierstra BA. Sonography after total hip replacement: reproducibility and normal values in 47 clinically uncomplicated cases. Acta Orthop. 2007;78(1):81–5.
51. Fritz J, Lurie B, Miller TT. Imaging of hip arthroplasty. Semin Musculoskelet Radiol. 2013;17(3):316–27.
52. Miller TT. Imaging of hip arthroplasty. Eur J Radiol. 2012;81(12):3802–1.

Physical Examination of the Hip and Pelvis

7

Hal D. Martin, Ian J. Palmer, and Munif A. Hatem

Contents

H.D. Martin (✉) • I.J. Palmer • M.A. Hatem
Baylor University Medical Center, Hip Preservation Center, Dallas, TX, USA
e-mail: Hal.Martin@baylorhealth.edu; haldavidmartin@yahoo.com; ianjamespalmer@gmail.com; munifhatem@gmail.com

Abstract

The clinical examination of the hip is a five-layer comprehensive assessment of the hip joint. In order to appreciate this achievement of symphonic function, it is important to understand the balance and interrelationship that each layer has upon the other in both a static and dynamic fashion. Optimally, the hip will be recognized early as the source of the complaint which is dependent upon a consistent method of interpreting the history and clinical examination of the hip. The standardized physical examination produces a fixed background, on which to produce the shadow of hip pathology and allowing for a five-layer comprehensive diagnosis.

Introduction

The works of Galen (121–201 CE), Vesalius (1555), and Albinus (1749) were some of the earliest descriptions of the human hip joint. The 1800s brought medical books and medical journals providing publications of hip joint anatomy and surgery. In 1895, the invention of the X-ray propelled the study of hip joint pathology into the 1900s with a proliferation of journals and publications. As in prior times of technological advancement, hip arthroscopy and open surgical techniques have progressed allowing for a better understanding of the complex hip anatomy and biomechanics of each of the five layers: the

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_115

osteochondral, capsulolabral, musculotendinous, neurovascular, and kinematic chain. The interpretation of which is dependent on a comprehensive physical examination of the hip. The establishment of a group of diagnostic tests as the background on which to cast the shadow of the pathologic condition is a key in the recognition of any complex pattern. A comprehensive standardized battery of physical examination tests and techniques will provide reliability and lead to an accurate diagnosis in all layers of the hip.

The Layers Approach to Hip Evaluation

Indications for hip arthroscopy have quickly expanded from addressing intra-articular, extra-articular, peritrochanteric, and deep gluteal space pathology. Arthroscopic visualization of these areas has increased our understanding of hip anatomy and biomechanics which has led to important research developments. Advancements applied to clinical findings further aid in our interpretation of the physical examination. Much work has been done to associate labral damage to hip pain and early osteoarthritis and degenerative joint disease; however, addressing intra-articular pathology alone is only part of the equation. Structural abnormalities must also be assessed and corrected, whether it is structural overcoverage or undercoverage. Hip pain generators can also be linked to contractile tissues or neurovascular problems. Therefore, the history and physical examination of the hip requires a comprehensive assessment of four distinct layers: osteochondral, capsulolabral, musculotendinous, and neurovascular as described by Draovich et al. [1]. The central link of the hip between the trunk and lower extremity represents the fifth layer, the kinematic chain. Hip symptoms can arise from pathologic conditions in a single layer or in a combination of multiple layers due to their static and dynamic interaction.

Layer I, the osseous layer, includes the femur, pelvis, and acetabulum which involve congruency, version, stability, and alignment. Abnormalities of layer I can involve bony overcoverage or undercoverage of the hip joint. Whether the hip pain generator of osseous pathology is static or dynamic, the source of pain is largely due to layer II, capsulolabral insult. However, consideration should also be given to how osseous abnormalities can directly (snapping hip, nerve impingement) or indirectly (contracture, instability) involve the layer III and layer IV hip pain.

Layer II, the capsulolabral layer, involves the hip ligaments, capsule, and labrum. Layer II functions as hip joint stabilizers. The contribution to limiting range of motion for the ischiofemoral ligament, pubofemoral ligament, and medial and lateral arms of the iliofemoral ligament has been documented [2]. Recent research has shown the important role of the ligamentum teres in limiting hip rotation in hip flexion [3]. The acetabular labrum extends the acetabular coverage over the femoral head and maintains a seal within the joint critical to joint health. The most common source of layer II pain is the labrum which can lead to cartilaginous degeneration of the hip joint. Hip instability, which may involve all layers, translates more forces to the layer II structures.

Layer III, the musculotendinous layer, involves the musculature around the hip joint. Layer III functions as a dynamic stabilizer and moves the hip, pelvis, and trunk. Pain and weakness of hip joint musculature can be the primary source of pain as a muscle tear, tendonosis, or tendonitis. Layer III pathology can be secondary to layer I and II pathology due to a compensatory response to hip pain.

Layer IV, the neurovascular layer, involves the neural and vascular structures of the hip joint. Pain, motor control, and proprioception contribute to hip joint kinematics and health. Layer IV pain can be illusive, however easy to diagnose with a comprehensive physical exam and understanding of the neurovascular anatomy and biomechanics.

Layer V, the kinematic chain, encompasses the hip joint, lumbar spine, abdominal trunk, knee, and their interrelationship. The dynamic connection of the hip joint, lumbar spine, sacroiliac joint, and knee joint ultimately functions as a unit. An incongruity from any layer can result in a disruption of the kinematic chain.

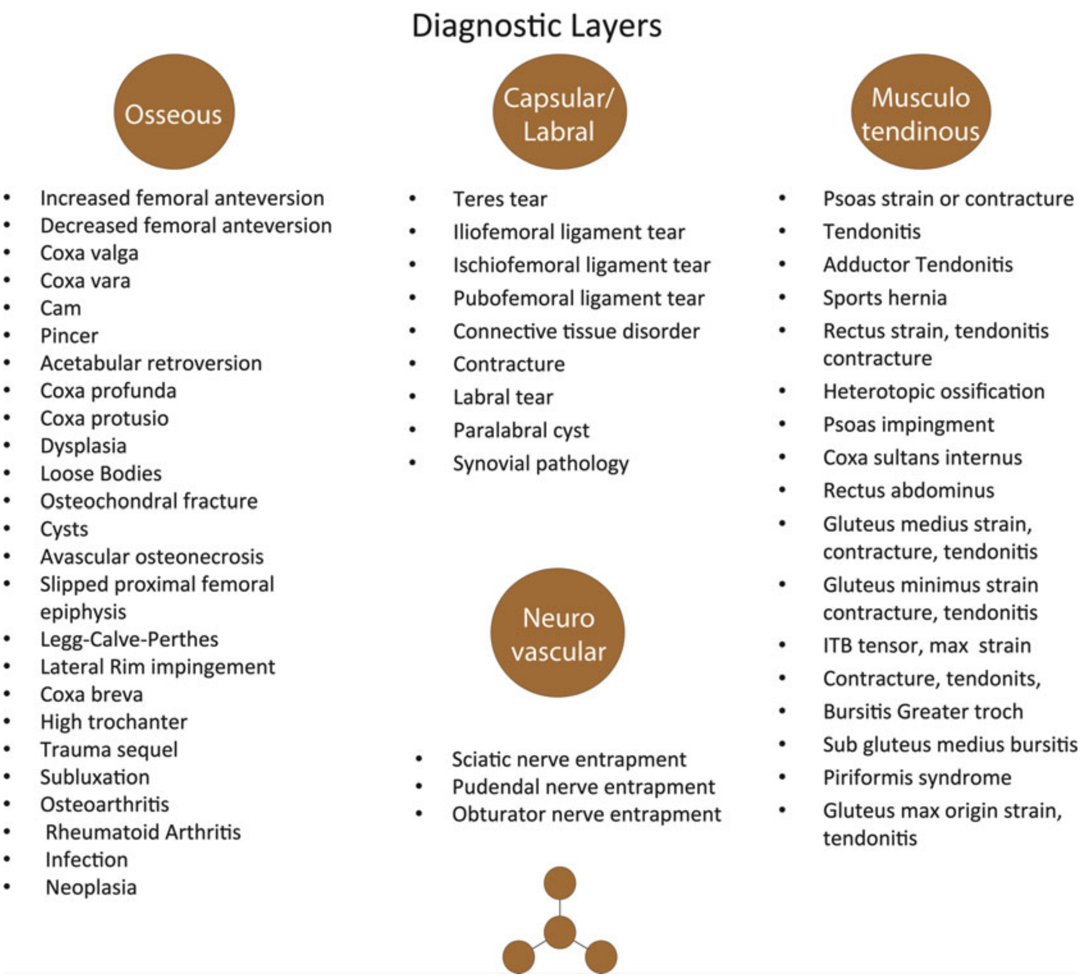


Fig. 1 Possible diagnoses by layer

Table 1 Example of a hip layered diagnosis

Layer 1	Femoral head cam deformity
Layer 2	Capsular laxity and labral tear
Layer 3	Gluteus medius tear
Layer 4	Deep gluteal syndrome/sciatic nerve entrapment
Layer 5	Lumbar spine arthrodesis

A standardized clinical evaluation will allow for the identification of pathologic conditions occurring in each of the five layers (Fig. 1, Table 1). As with the shoulder and knee examinations, there are critical steps that form the basis of the examination of the hip joint. This hip examination contains 21 steps, which compares well with the shoulder 20-step exam and the knee 33-step exam. A clinical evaluation of the hip that incorporates this layered concept will lead to an accurate diagnosis in a timely manner. This chapter describes the detailed history and 21 core examinations of a standardized clinical evaluation of the adult and adolescent hip.

History

Prior to the physical examination of the hip, a comprehensive history of the patient is obtained. Age and the presence or absence of trauma are the primary factors directing the examination.

Documentation includes the chief complaint, the date of onset, and mechanism of injury. Detail the description of pain according to location, severity, and factors that increase or decrease the pain. Referred pain to the knee or lumbar spine often accompanies hip pathology and should also be investigated.

In addition to the standard history, the patients past medical and lifestyle history is acquired. Treatment to date must be clearly defined, including all conservative and surgical therapies. Hip preservation surgery is becoming more common; therefore, detailed prior surgical treatment is necessary for any revision surgery consideration. The patient's functional status is assessed and current limitations are detailed, which may involve getting in or out of a bathtub or car, activities of daily living, jogging, walking, and/or climbing stairs. Related symptoms of the spine, abdomen, and lower extremity must be identified. Limitation of hip motion can lead to secondary problems in lumbar spine, sacroiliac joint, and pubic-inguinal musculotendinous structures [4–7], representing the fifth layer or kinematic chain. Knee ligament injury can also be associated to restrict hip mobility [8]. The presence of back pain and coughing or sneezing exacerbation help rule out thoracolumbar problems. Abdominal pain, anorectal and genitourinary complaints, and menses-related symptoms indicate intrapelvic or abdominal pathologies, which may coexist with hip problems. In addition, night pain, sitting pain, weakness, numbness, or paresthesia in the lower extremity may suggest neural compression, which can be located at the lumbar spine, inside the pelvis or within the subgluteal space. The following items must additionally be addressed: past injuries, childhood or adolescent hip disease, ipsilateral knee disease, suggestive history of inflammatory arthritis, and risk factors for osteonecrosis.

Hip pain often stems from some type of sports-related injury, mainly with rotational sports, such as golf, tennis, ballet, and martial arts. Five to six percent of adult sports injuries originate in the hip and pelvis [9]. Athletes participating in rugby and martial arts have also been reported to have an increased incidence of degenerative hip disease [10]. Documentation of sports activities can help determine the type of injury and guide the treatment considering the patient's goals and expectations. A complete review of the history is given in Table 2.

A questionnaire is utilized in order to score the patient's hip status. There are several questionnaires validated for the assessment of patient hip joint function and related pain. The Harris Hip Score (HHS) is the most documented hip score and was first described to evaluate patients with mold arthroplasty [11] and has been modified for more current use in hip arthroscopy [12]. As the hip joint questionnaires have evolved, the functional assessment has become more important, and self-administered questionnaires are preferred rather than observer administered [13]. The MAHORN (Multicenter Arthroscopy of the Hip Outcomes Research Network) Group has validated the International Hip Outcome Tool (iHOT-33) [14]. Developed through an international multicenter study, the iHOT-33 is a self-administered questionnaire, and its target population is active adult patients with hip pathology. While the iHOT-33 was developed for research purposes, a short form (iHOT-12) [15] was also described for use in routine clinical practice. The Short Form 36 (SF-36) [16] and 12 (SF-12) [17] are questionnaires used for general health survey and are sometimes associated with the more hip-specific evaluation tools. Final outcome is dependent upon the effects of treatment which should consider the mental and spiritual aspects of the patient. This assessment has proven useful in heart and cancer treatment and is required in complex hip pathology evaluation due to the critical role the hip plays in all activities of daily life.

The diagnostic and treatment tools for adult and adolescent hip disorders have improved over the past decade. Choosing the best approach for each patient depends mainly on the patient expectations and goals. The history of complaint is important not only to define the diagnosis but also to choose the most appropriate option for each patient among the treatment alternatives [18]. The use of a common language and standardized history and physical examination will aid in the understanding of patients with complex pathology.

Table 2 Complete review of patient history

NAME: ______________________ **DATE:** ______________ **AGE:** ________
DUTIES: ______________________ **REFERRED BY:** ______________________
CHIEF COMPLAINT: **L HIP** **R HIP** **OTHER:** ______________________
HISTORY OF PRESENT ILLNESS:

- Date of onset ______________
- Pain location ______________
- Traumatic/nontraumatic
- Mechanism of injury _________
- Popping/locking ___________
- Knee pain _____ Back pain ______
- Pain at Rest (VAS 0 -10) ________
- Pain With Activity(VAS 0 -10) ____
- Pain a.m. / p.m.____________
- Pain increased with _________
- Pain decreased with _________
- Have you ever been diagnosed with AVN/ONFH ? ______________________
  Alcohol use_______ Tobacco use ________Steroid use_______ Other __________
- Suggestive history of inflammatory arthritis______________________

TREATMENT TO DATE

Rest Ice Heat NSAIDS__________
Physiotherapy __________
Surgery______________
Chiropractic tx ______________
Injections______________
Support (cane, crutch)__________
Orthotics______________

TESTS AND EVALUATIONS:

MRI MRI Arthrogram CT X -rays Lab Biometrics Consults_____________

PAST INJURIES: _ ______________________

LIMITATIONS:

- Sitting
- Getting in or out of tub
- Getting in or out of car
- Jogging
- Length of time able to sit
- Sports
- Stairs
- Work
- ADL's
- Household activities

FUNCTION:

HHS m_______________iHOT -33__________________

ASSOCIATED

- Spine sacroiliac affection
- Night pain awakening
- Knee pathology
- Genitourinary complaints
- Numbness
- Abdominal Pain

SPORTS AND ACTIVITIES: ______________________
GOAL IN TREATMENT:______________________
REVIEW OF SYSTEMS:

VAS visual analog pain scale, *AVN* avascular necrosis of the femoral head, *ONFH* osteonecrosis of the femoral head, *ADL* activities of daily living

The Physical Examination of the Hip

The 21-step physical examination of the hip (Table 3) is a comprehensive assessment of four distinct layers: osteochondral, capsulolabral, musculotendinous, and neurovascular. A consistent hip examination is performed quickly and efficiently to find the comorbidities that coexist with complex hip pathology by assessing the hip, back, abdominal, neurovascular, and neurologic

Table 3 Twenty-one (21)-step physical examination of the hip

Standing	1. Gait	Pelvic tilt/rotation, stride length, stance phase, FPA
	2. Single-leg stance phase test	Neural loop/abductor strength
	3. Inspection	Leg lengths, forward bend/spine, body habitus, global laxity
Seated	4. Neurovascular/reflex	Skin, lymphedema, sensory, DTR
	5. ROM	Internal and external rotation
Supine	6. Palpation	Abdomen, adductor tubercle
	7. ROM	Abduction, adduction, flexion
	8. Hip flexor contracture test	Psoas/hip flexor contracture
	9. DIRI	FAI
	10. DEXRIT	FAI, anteroinferior instability, apprehension
	11. FADDIR	FAI
	12. FABER	Hip vs. SI
	13. Dial test	Laxity/instability
Lateral	14. Palpation	GT facets and bursae, glutei origin/insertion
	15. Strength	Abduction, gluteus medius, gluteus maximus
	16. Passive adduction tests	Tensor fascia lata, gluteus medius, gluteus maximus
	17. Lateral rim impingement test	FAI, laxity, apprehension
	18. Posterior rim impingement test	FAI, apprehension, contrecoup
	19. Apprehension test	Laxity, contrecoup
Prone	20. Rectus femoris contracture test	Rectus femoris contracture
	21. Femoral version test	Femoral anteversion

Reprint with permission from Martin and Palmer [20]

systems. Loose-fitting clothing about the waist is helpful for exposure and patient comfort. Documentation of the exam by an assistant on a standardized written form aids in accuracy and thoroughness. The use of a common language and specific technique for each examination test will eliminate multi-clinical discrepancy and improve reliability. The MAHORN (Multicenter Arthroscopy of the Hip Outcomes Research Network) Group outlined the common tests that form the basis of a multilayered hip evaluation [19]. This battery of tests covers all five layers of the hip.

Standardization enhances the physical examination reliability [21], and the most efficient order of examination begins with standing tests followed by seated, supine, lateral tests, and ending with prone tests [22].

Standing Examination

A general location of pain is noted by the patient pointing with one finger will usually help direct the examination. The groin region may be indicative of an intra-articular problem. Lateral-based pain may be associated with intra- or extra-articular aspects. A characteristic sign of patients with intra-articular hip pain is the "C Sign" [23]. The patient will hold his or her hand in the shape of a C and place it above the greater trochanter, with the thumb positioned posterior to the trochanter and fingers extending into the groin. Posterior-superior pain requires the differentiation of hip and back pain. Bilateral shoulder and iliac crest heights in the standing position are compared to evaluate leg-length discrepancies (Fig. 2a, b). Incremental heel lifts placed under the short side foot will help with orthotic considerations. Trunk bending (side to side and forward) is performed to evaluate the lumbar spine and to differentiate structural from nonstructural scoliosis (Fig. 2c, d). General body habitus and joint laxity are easily assessed in the standing examination (Fig. 2e).

Gait abnormalities often help detect hip pathology and the kinematic chain. The patient is taken to an area large enough to observe a full gait of six to eight stride lengths (Fig. 3). The key elements of gait evaluation include foot progression angle (FPA), pelvic rotation, stance phase, and stride length. The whole limb, including osseous and

Fig. 2 Standing evaluation. (**a**) and (**b**). Shoulder and iliac crest heights are examined with the patient with dynamic loading of the hip joint in the standing position. (**c**). Spinal alignment is assessed during a forward bending of the trunk, palpating spinal alignment and noting the degrees of trunk maximal flexion. (**d**). Tests for generalized joint laxity of the elbows. (**e**). Passive opposition of the thumb to the flexor aspect of the forearm

soft tissues, must be considered when evaluating any FPA abnormality. Increased femoral version can cause an internally rotated FPA, but it may be balanced by tibial lateral torsion or deficient medial hip rotators. The knee and thigh are observed simultaneously to assess any rotatory parameters. The knee may want to be held in internal or external rotation to allow proper patellofemoral joint alignment but may produce a secondary abnormal hip rotation. This abnormal motion is usually present in cases of severe increased femoral anteversion, precipitating a

Fig. 3 Gait evaluation. A full gait of six to eight stride lengths is evaluated from behind (**a**) and from the front (**b**) as the patient walks in the hallway. Fundamental points of gait evaluation include foot progression angle, pelvic rotation, stance phase, stride length, and arms swing

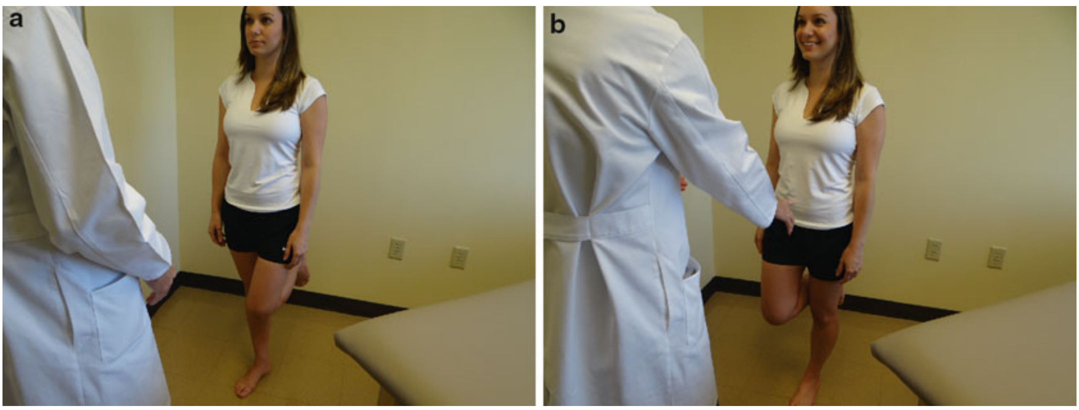


Fig. 4 Single-leg stance phase test. (**a**), right side. (**b**), left side. Bilateral assessment, observed from behind and in front of the patient. The patients hold this position for 6 s

battle between the hip and knee for a comfortable position, which will affect the gait. The following abnormal gait patterns can be associated with hip pathologies: winking gait with excessive pelvic rotation in the axial plane, abductor deficient gait (Trendelenburg gait or abductor lurch), antalgic gait with a shortened stance phase on the painful side, and short leg gait with dropping of the shoulder in the direction of the short leg.

The single-leg stance phase test (Trendelenburg test) is performed during the standing evaluation of the hip. The single-leg stance phase test is performed bilaterally, with the non-affected leg examined first, to establish a baseline (Fig. 4). A positive is noted if the pelvis drops toward the nonbearing side or shift of more than 2 cm toward the bearing (affected) side, which may indicate that the abductor musculature is weak or the neural loop of proprioception is disrupted on the bearing side. Trunk inclination for the bearing (affected) side is also noted in a positive single-leg stance test. This assessment is performed in a dynamic fashion by some examiners.

Seated Examination

The seated hip examination consists of a thorough vascular, lymphatic, and neurologic examination, which are performed in all individuals (Fig. 5). Vascular and lymphatic assessment includes the posterior tibial pulse, any swelling of the extremity, and inspection of the skin. The neurologic evaluation includes sensibility, motor function, and deep tendon reflexes (patellar and Achilles). The presence of radicular neurologic symptoms is detected by the straight leg raise test, performed by passively extending the knee into full extension.

The seated position provides a reproducible and reliable platform for the assessment of hip internal and external rotation (Fig. 6). The ischium is square to the table at 90° of hip flexion. Values of hip rotation measured in different positions (seated, prone, supine) can be compared for an assessment of ligamentous versus osseous abnormality. Passive internal and external rotation is performed until a gentle endpoint is obtained and compared bilaterally. Proper hip function requires sufficient internal rotation, and there should be at least 10° of internal rotation at the midstance phase of normal gait [24], but less than 20° is abnormal. Excessive femoral anteversion may be indicated by increased internal rotation combined with a decreased external rotation. In contrast, excessive femoral retroversion may be indicated by increased external rotation with decreased internal rotation.

Supine Examination

The supine examination begins with the inspection of leg-length discrepancy (Fig. 7). Tenderness with palpation is documented for the abdomen, pubic

Fig. 5 Vascular, lymphatic, and neurologic examination. (**a**). Palpation of posterior tibial pulse and skin inspection. (**b**). Skin and lymphedema inspection. (**c**). Straight leg raise, useful in detecting radicular neurological symptoms

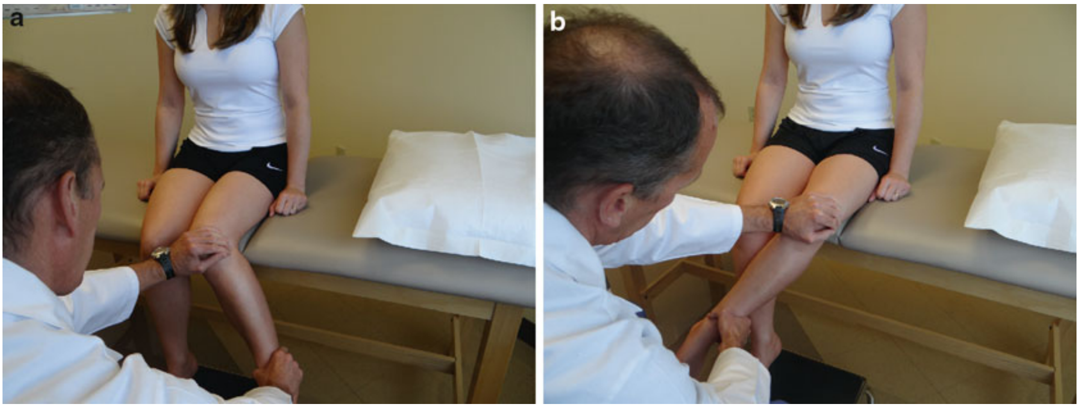


Fig. 6 Seated internal (**a**) and external (**b**) rotation range of motion. Passive internal and external rotation testing are compared from side to side. In the seated position, the ischium is square to the table, thus providing sufficient stability at 90° of hip flexion

Fig. 7 Supine assessment of leg lengths in a static position. Can be compared to the dynamic loading in the standing assessment

symphysis, and adductor tubercle. Differentiation of isolated adductor tendinitis and sports hernia may be made by a resisted sit-up torso flexion (Fig. 8).

Hip ranges of motion are recorded for passive abduction, adduction, and flexion (Fig. 9). Bring both of the patients' legs into flexion (knees to chest) and note the pelvic position because the hip may stop early in flexion resulting in pelvic rotation to achieve end range of motion. With both legs in flexion, the pelvis is in a zero set point (eliminating lumbar lordosis) important for the hip flexion contracture test (Thomas test). The patient holds one knee to their chest and passively moves the contralateral leg into extension (Fig. 10). Inability for the back of the thigh to reach the table indicates the presence of contracture and patients with hyperlaxity or lumbar spine hyperlordosis can result in a false negative. In patients with hyperlaxity or connective tissue disorders, the zero set point can be established with an abdominal contraction.

Hip joint versus sacroiliac joint pain is detected by the flexion, abduction, and external rotation test (FABER), historically known as the Patrick's test. The leg is placed in figure-4 position with the knee in flexion and hip in flexion, abduction, and external rotation so that the ankle rests on the contralateral thigh. Ipsilateral or contralateral sacroiliac discomfort may be felt. Recreation of hip pain can be associated with posterior femoroacetabular impingement, ligamentous injury, or trochanteric pathologies.

Several tests exist for the assessment of femoroacetabular congruence, instability, or intra-articular pathology. Ganz originally described the flexion, adduction, and internal rotation test [25], and McCarthy later described the dynamic assessment of the femoroacetabular congruence and relationship to the labrum [26].

The dynamic internal rotatory impingement (DIRI) test is the assessment of anterior femoroacetabular congruence (DIRI). The zero set point of the pelvis must be obtained by the patient holding the contralateral leg in flexion.

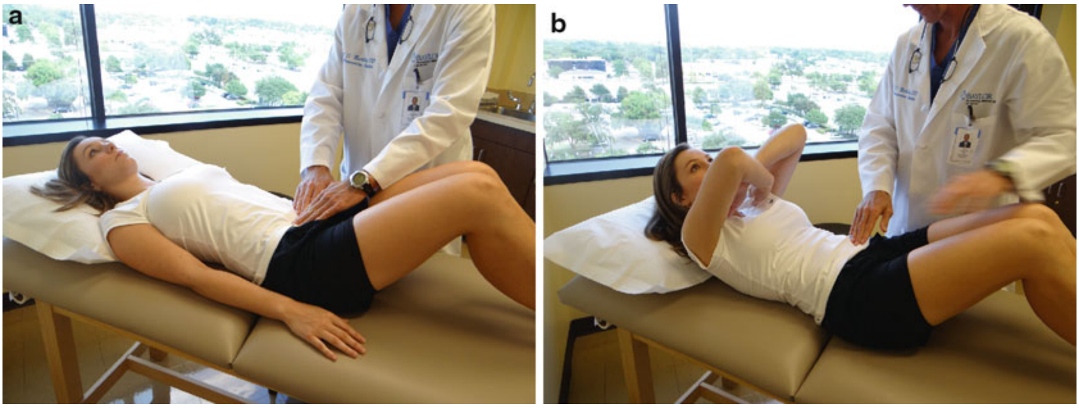


Fig. 8 Abdominal and pubic palpation. (**a**). Palpation in the relaxed supine position. (**b**). Palpation with abdominal contraction

Fig. 9 Passive abduction (**a**) and adduction (**b**) range of motion assessment

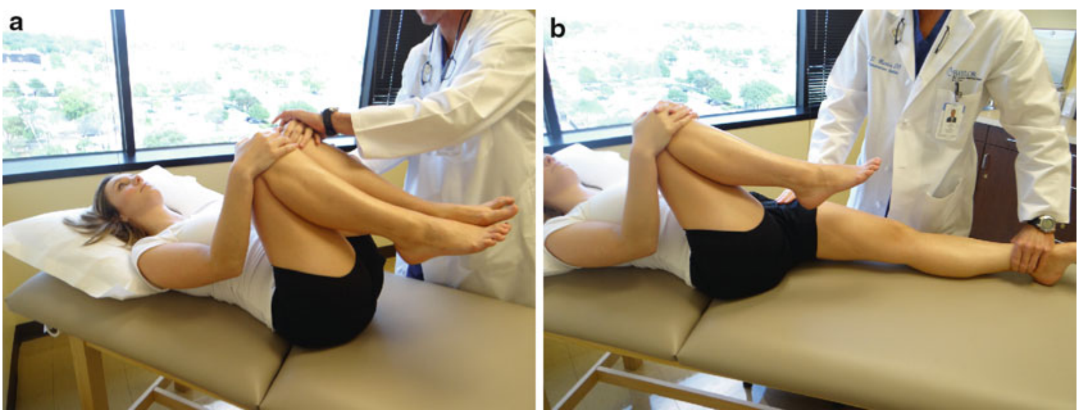


Fig. 10 Hip flexion contracture test. (**a**). Starting position. Zero set point of the pelvis is achieved by having the patient hold the contralateral leg in full flexion, thus establishing neutral pelvic inclination. (**b**). The examined hip is passively extended toward the table. Both hips are evaluated for side-to-side comparison

Fig. 11 Dynamic internal rotatory impingement (DIRI) test. (**a**). DIRI begins with the hip at 90° flexion or beyond. (**b**) and (**c**). It is then passively taken through a wide arc of adduction and internal rotation, checking the anterior congruence between the femoral neck and acetabulum

The hip is dynamically taken in a wide arc from abduction/external rotation to flexion, adduction, and internal rotation (Fig. 11). Recreation of the complaint pain is a positive result. Note the degree of flexion that causes impingement, which helps determine the degree, type, and location of anterior impingement. The Scour test is performed in the same manner as DIRI, while applying pressure at the knee to increase pressure on the hip joint.

The dynamic external rotatory impingement test (DEXRIT) includes a wide arc movement of passive abduction and external rotation. The patient holds the contralateral leg in flexion to establish the zero set point of the pelvis. The hip is dynamically taken from 90° flexion or beyond through an arc of abduction and external rotation (Fig. 12). The DEXRIT is an assessment of superolateral and posterior femoroacetabular impingement. Patients with anteroinferior hip instability, anteroinferior acetabular hypoplasia, torn teres ligament, and capsular laxity may also exhibit a positive DEXRIT. A positive result is noted with recreation of pain or feeling of instability. Both the DEXRIT and DIRI can be performed intraoperatively for direct visualization of femoroacetabular congruence.

The posterior rim impingement test is performed to assess the congruence between the posterior wall and femoral neck. The patient is positioned at the edge or end of the examination table so that the leg can hang freely to full extension. The patient established the pelvic zero set point with both legs held in flexion. The examined leg is allowed to reach full extension off the table and then taken into abduction and external rotation. Recreation of the symptoms is a positive test

Fig. 12 Dynamic external rotatory impingement test (DEXRIT). (**a**). DEXRIT begins with the hip at 90° flexion or beyond. (**b**, **c**). It is then dynamically taken through a wide arc of abduction and external rotation, in order to assess the superior posterior acetabular impingement and anteroinferior instability

and can also present as an apprehension sign in cases of anterior instability.

Other useful tests may be included during the supine assessment. The heel strike test (strike the heel abruptly) is performed in patients with a history of trauma. The straight leg raise against resistance or Stinchfield test (resisted hip flexion from 10° to 45° with knee extended is an assessment of hip flexor and psoas strength, Fig. 13) and is also useful for evaluation of psoas impingement, tendonitis, or intra-articular irritation, as the psoas places pressure on the labrum. The passive supine rotation or log roll (passive internal and external hip rotation with the leg in extension) is performed bilaterally (Fig. 14). Any side-to-side differences in this maneuver may indicate the presence of laxity, effusion, or intra-articular derangement. A modification of the log roll test is the dial test which is an assessment of iliofemoral laxity. It includes passive internal rotation of the leg, followed by releasing the leg and allowing it to external rotate. External rotation of more than 45°, relative to vertical, is a positive test [27].

Fig. 13 Straight leg raise against resistance test

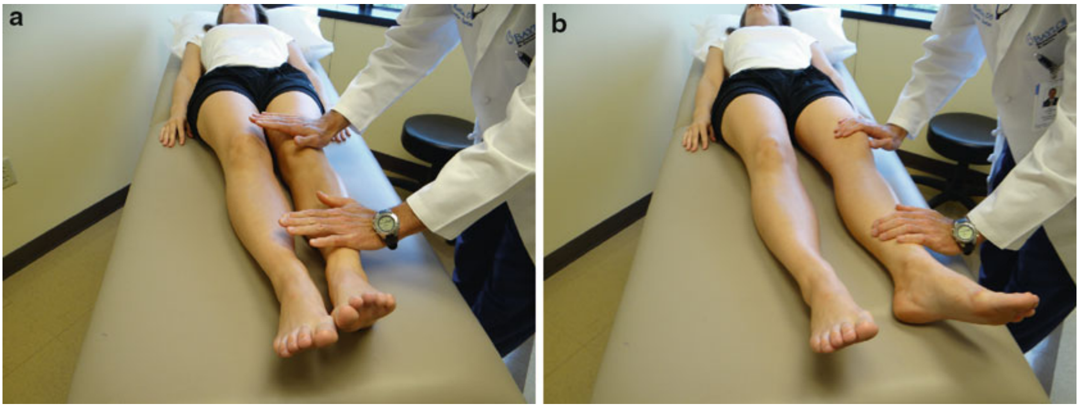


Fig. 14 The passive supine rotation test involves passive internal rotation (**a**) and external rotation (**b**) of the femur, with the leg lying in an extended position

Lateral Examination

Palpation in the lateral position includes suprasacroiliac area, sacroiliac (SI) joint, gluteus maximus origin, piriformis muscle, and sciatic nerve. The facets of the greater trochanter (anterior, lateral, supero-posterior, and posterior) are palpated. The insertion of the gluteus minimus located at the anterior facet, the gluteus medius at the supero-posterior and lateral facets, and the trochanteric bursa at the posterior facet. Strength is assessed with any type of lateral-based hip complaint. The tests are performed in lateral decubitus with the patient actively abducting the hip against resistance and graded with a standardized 5-point scale. The gluteus medius strength test (Fig. 15a) is performed with the knee in flexion to release the gluteus maximus contribution for the iliotibial band. The overall abductor strength (Fig. 15b) is evaluated with the knee in extension, and the gluteus maximus is tested asking the patient to abduct and extend the hip. A set of passive adduction tests (similar to Ober's test) is performed with the leg in three positions – extension (tensor fascia lata contracture test), neutral (gluteus medius contracture test), and flexion (gluteus maximus contracture test) (Fig. 16). If the hip does not come beyond the midline in the longitudinal axis of the torso, it is graded as 3+ restriction above torso, 2+ at the midline, and 1+ restriction below.

The lateral decubitus is also utilized for femoroacetabular congruence evaluation. The passive flexion adduction internal rotation (FADDIR) test is performed in a dynamic manner (Fig. 17a). Any reproduction of the patient's complaint and the degree of femoroacetabular impingement are noted. FADDIR is traditionally performed as part of the supine assessment (Fig. 17b).

The lateral rim impingement test is performed with the hip passively abducted and externally rotated (Fig. 18a). Reproduction of the patient's pain is scored positive and can be caused by anterior instability or posterior impingement. If the feeling of guarding or anterior pain is present, the test is positive for instability. An apprehension test with a provocative maneuver is also executed in the lateral position (Fig. 18b), including a forward force to test anteroinferior instability [28]. Beyond the capsular and teres ligament, this test is also useful to detect acetabular anteroinferior hypoplasia.

Prone Examination

In the prone position is also performed the rectus contracture test (also known as Ely test) (Fig. 19a). Passively bring the knee leg into

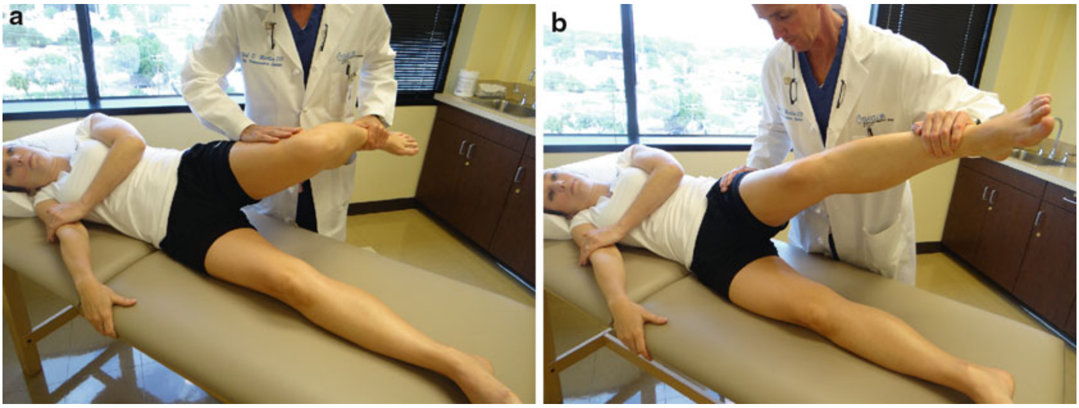


Fig. 15 Abductor strength assessment. The patient actively abducts the limb against the examiner, who utilizes his weight as force of resistance. (**a**). Gluteus medius strength is evaluated by having the patient perform active hip abduction with flexed knee. (**b**). Overall abductor strength evaluation

flexion noting the end range of motion. The femoral anteversion test, traditionally known as Craig's test, will give the examiner an idea of femoral anteversion and retroversion (Fig. 19b). Palpate the greater trochanter and internally/externally rotate the hip until the greater trochanter is in the most lateral position. Note the angle of the lower leg compared to vertical. Normally, femoral anteversion is between 10° and 20°.

Femoroacetabular Congruence and Hip Instability

Femoroacetabular impingement and instability are increasingly being recognized as a cause of more severe symptoms when they coexist. It is important to understand that femoroacetabular congruence tests assess not only femoroacetabular impingement but also hip instability. Beyond the osseous layer, these tests evaluate the contribution of the capsular and musculotendinous layers for the hip joint stability. Biomechanics is also considered when interpreting the results of the femoroacetabular congruence. For example, although the femoral head-neck transition anatomy and acetabular retroversion significantly affect the tests, decreased femoral anteversion [29] or increased pelvic tilt can cause anterior impingement without high degrees of hip flexion or internal rotation. With biomechanical advancements, clinical tests may be refined or excluded from the routine evaluation. Multiple examinations are important for the detection of intra-articular pathology, and femoroacetabular congruence tests can have reasonable predictive value for impingement or instability when performed by an experienced examiner. However, no single test is sensitive enough to be used exclusively and a battery is always used for assessment.

In addition to femoroacetabular congruence testing, tests of hip instability are also incorporated. The key tests for instability are the Beighton signs of global laxity, passive supine rotation/dial test, in-toeing gait, and flexion/abduction/external rotation test apprehension. Laxity may involve the stabilizing ligaments in extension, such as the iliofemoral ligament, and the stabilizing ligaments in flexion, such as the teres [2, 3, 28]. Hypoplasia of the anteroinferior horn is best detected by apprehension [27].

Table 4 is a summary of the main tests utilized for femoroacetabular congruence evaluation and hip instability considerations.

Snapping Hip Tests

Snapping iliopsoas tendon tests – a fan test (the patient circumducts and rotates the hip in a rotatory fashion) can help delineate the presence of the snapping iliopsoas tendon over the femoral

Fig. 16 Passive adduction tests (**a**). The tensor fascia lata contracture test: With the knee in extension, the examiner passively brings the hip into extension then adduction. (**b**). Gluteus medius contracture test is performed with knee flexion, thus eliminating the gluteus maximus contribution for the iliotibial band. The examiner passively adducts the hip toward the examination table. (**c**). The gluteus maximus contracture test is performed with the ipsilateral shoulder rotated toward the examination table. With the examined leg held in knee extension, the examiner passively brings the hip into flexion then adduction

head or the innominate. Often, this diminishes with an abdominal contraction. A flexion, abduction, external rotation maneuver, followed by extension, can be also performed in assessing the snapping of the iliopsoas tendon over the iliopectineal eminence.

Hip external snapping tests – a hula hoop maneuver, in which the patient stands and twists, or a bicycle test (performed in the lateral position), can help distinguish the pop internally from the external pop of coxa saltans externus caused by the subluxing iliotibial band over the greater trochanter. The bicycle test is performed with the patient in the lateral position. The motion of a bicycle pedaling pattern is recreated as the examiner monitors the iliotibial band for the detection of coxa saltans externus.

Tests Emphasized for Pain Location

As our understanding of hip pain has evolved, the concept of emphasized tests based upon pain location has emerged. Although the 21 tests are the

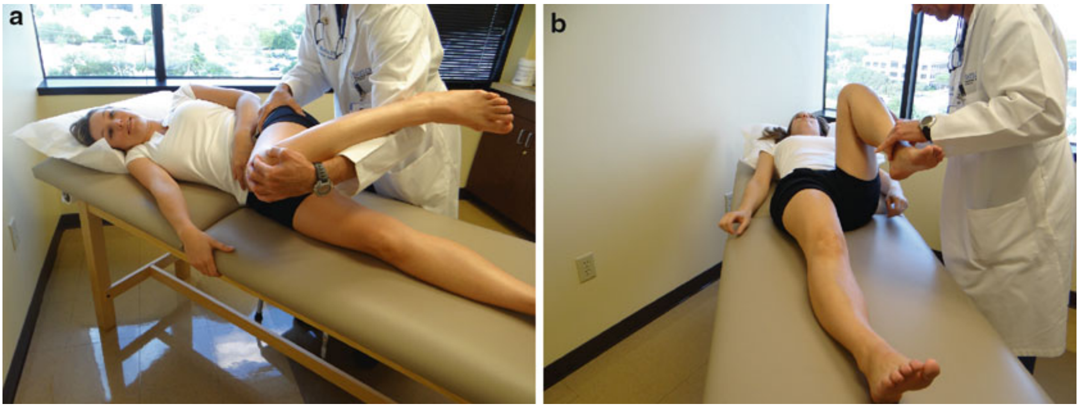


Fig. 17 Flexion, adduction, internal rotation (FADDIR). (**a**). In the lateral position, the examiner brings the examined hip into flexion, adduction, and internal rotation, while monitoring the superior aspect of the hip. (**b**). FADDIR can also be performed in the supine position

Fig. 18 Lateral rim impingement and apprehension tests. (**a**). The lateral rim impingement test is performed in the lateral position. The examiner cradles the patient's lower leg with one arm and monitors the hip joint with the opposing hand. The affected hip is passively brought through a wide arc from flexion to extension in continuous abduction while externally rotating the hip. (**b**). Apprehension test. The examiner brings the hip from flexion to extension in external rotation and abduction. With the opposite hand, the examiner forces forward the proximal femur to provoke anteroinferior subluxation of the femoral head

basics, specialized tests for specific locations of pain can be incorporated.

Anterior pain requires differentiation of soft tissue versus osseous etiology. Femoroacetabular impingement (cam/pincer) utilizes the congruence tests distinct from psoas impingement. Consideration is given to psoas impingement versus subspinous osseous congruence. The straight leg against resistance is helpful for psoas issues and palpation of the adductor tubercle and abdomen for sports hernia or pubic symphysis problems.

In patients with lateral pain, the palpation should be more specific. The greater trochanteric facets are assessed, with special attention to the gluteus minimus insertion in the anterior facet and gluteus medius insertion in the lateral facet. Patients with trochanteric bursitis have pain over the posterior facet. Abductor strength tests are important as a loss of strength with pain suggests gluteus medius/minimus tendon tears. Contracture tests aid in the detection of muscle contracture of the gluteus maximus, tensor fascia lata, or gluteus medius.

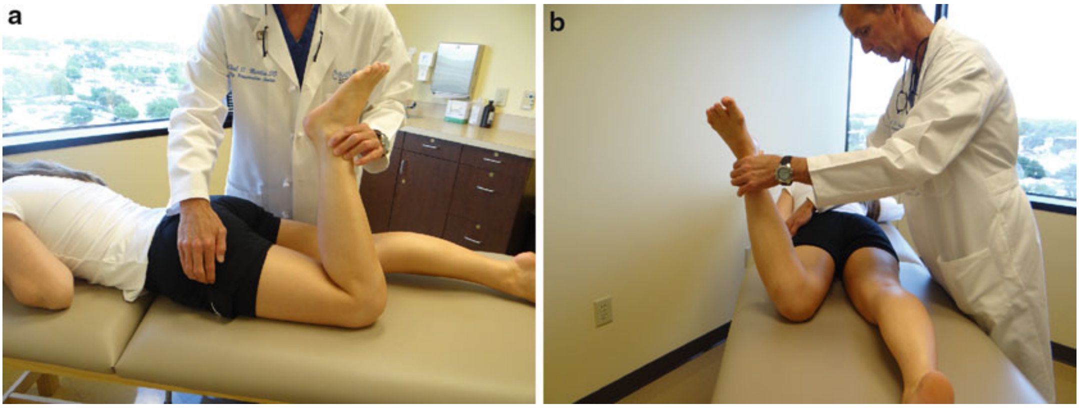


Fig. 19 Femoral anteversion and rectus contracture tests. (**a**). For the femoral anteversion test, the knee is flexed to 90°, and the examiner manually rotates the leg while palpating the greater trochanter. The examiner positions the greater trochanter so that it protrudes most laterally, noting the angle between the axis of the tibia and an imaginary vertical. (**b**). Rectus contracture test. The lower extremity is flexed toward the gluteus maximus. Any raise of the pelvis or restriction of knee flexion motion is indicative of rectus femoris muscle contracture

Table 4 Summary of femoroacetabular congruence and laxity tests

Test	Positioning	Tested impingement	Tested instability
Flexion, adduction, internal rotation	Lateral or supine	Anterior	
	Opposite hip in extension		
Dynamic internal rotatory impingement test (DIRI)	Supine	Anterior	Posterior
	Opposite hip in full flexion		
Dynamic external rotatory impingement test (DEXRIT)	Supine	Superior and posterior	Anteroinferior
	Opposite hip in full flexion		
Posterior rim impingement test	Supine	Posterior	Anterior
	Opposite hip in full flexion		
Lateral rim impingement	Lateral	Superior and posterior	Anterior and inferior
Apprehension test	Lateral	Posterior	Anterior and inferior

Posterior hip pain requires evaluation of the deep gluteal region and abnormalities of the sacroiliac joint. To aid in the differential diagnosis, palpation of the gluteal structures is performed. The patient is in the seated position with the pelvis square to the examination table and the ischial tuberosity (IT) serves as the reference point for palpation. Pain superolateral to the IT at the sciatic notch is characteristic of deep gluteal syndrome; pain lateral to the IT, ischial tunnel syndrome or ishiofemoral impingement is considered; pain at the IT, hamstrings tendons pathologies are possible; and pain medial to the IT, pudendal nerve entrapment is considered.

Deep gluteal syndrome examination includes passive and active testing. The seated piriformis stretch test (Fig. 20a) is a flexion, adduction with internal rotation test performed with the patient in the seated position. The examiner extends the knee (engaging the sciatic nerve) and passively moves the flexed hip into adduction with internal rotation while palpating 1 cm lateral to the ischium (middle finger) and proximally at the sciatic notch (index finger). A positive test is the recreation of the posterior pain at the level of the piriformis or external rotators. An active piriformis test (Fig. 20b) is performed by the patient pushing the heel down into the table, abducting and

Fig. 20 Posterior hip pain evaluation. (**a**) Seated piriformis stretch test. The patient is in the seated position with knee extension. The examiner passively moves the flexed hip into adduction with internal rotation while palpating 1 cm lateral to the ischium (middle finger) and proximally at the sciatic notch (index finger). (**b**) Active piriformis test. With the patient in the lateral position, the examiner palpates the piriformis. The patient drives the heel into the examining table thus initiating external hip rotation while actively abducting and externally rotating against resistance. (**c**) Ischiofemoral impingement test of the right hip. Impingement between the lesser trochanter and ischium is assessed by passive extension of the hip, reproducing the patient's symptoms with the examined hip adducted or neutral. The left index finger of the examiner is palpating the ischiofemoral space lateral to the ischium. Extension and abduction of the hip does not reproduce the symptoms

externally rotating the leg against resistance, while the examiner monitors the piriformis. In a recent published study, the combination of the seated piriformis stretch test with the piriformis active test presented a sensitivity of 91 % and specificity of 80 % for the endoscopic finding of sciatic nerve entrapment [30].

Patients with ischiofemoral impingement usually complain of deep gluteal pain and limitation on physical activities. Affected individuals also grab the symptomatic hip lateral to ischium during long stride walking, while pain is alleviated with short stride walking. The ischiofemoral impingement test is performed with the patient in contralateral decubitus and taking the affected hip into passive extension in neutral and adduction. A test is considered positive when reproduces the symptoms with the examined hip extended and adducted or neutral, while extension with abduction does not reproduce the symptoms (Fig. 20c).

Ischial tunnel syndrome or hamstring syndrome is described as pain in the lower buttock region that radiates down the posterior thigh to the popliteal fossa and is commonly associated with hamstring weakness. Patients experience pain with sitting, stretching, and with exercise, primarily running (sprinting and acceleration). Palpable tenderness is located around the ischial tuberosity in the proximal hamstring region. An active knee flexion test against resistance, with 30 degrees versus 90 degrees of knee flexion, can help evaluate the proximal hamstring tendons.

Abnormalities of the sacroiliac (SI) joint should be also considered when assessing patients with gluteal pain and lower lumbar pain [31]. In addition to the FABER test, four other tests are indicated on the evaluation of SI joint: thigh thrust test, Gaenslen test, distraction test, and compression test. The thigh thrust test is performed with the patient in the supine position. The ipsilateral hip is flexed at 90 degrees and the examiner applies axial pressure through the axis of the femur, provoking anteroposterior shear stress on the SI joint. For the Gaenslen test, the patient lies supine near the edge of the table with the contralateral hip is held in flexion. The examiner then extends the hip off table. This test applies torsional stress on the SI joints. Also in the supine position is the distraction test. The examiner applies pressure on the left and right anterior superior iliac spine in order to generate tensile forces on the anterior aspect of the SI Joint. The compression test is performed with patient in the lateral position, with the examiner compressing the anterior aspect of the lateral ilium. This test places lateral compression force across the SI joints.

More detailed information can be found in ▶ Chap. 71, "Deep Gluteal Syndrome" of this book.

Summary

The physical examination of the hip has evolved throughout history and continues to be refined as our understanding of pathologic and nonpathologic anatomy and biomechanics progresses. A common descriptive language for the clinical hip exam is necessary for international dialogue and the progression of research in this field. The core 21 hip physical examination tests are described in this chapter. A complete battery of tests will assess all layers of the hip, with 16 assessing the osteochondral layer, 13 the capsulolabral layer, 11 the musculotendinous layer, 7 the neurovascular layer, and 5 the kinematic chain. Sixteen maneuvers or group of maneuvers evaluate more than one layer. A five-layer diagnosis on each patient should be considered in order to be thorough and comprehensive in organizing diagnostic and treatment strategies. A well-organized and structured exam may last as long as 8 min or less depending on the examiner's practice. The standardized physical examination produces a fixed background, on which to produce the shadow of hip pathology and allowing for a five-layer comprehensive diagnosis.

References

1. Draovitch P, Edelstein J, Kelly BT. The layer concept: utilization in determining the pain generators, pathology and how structure determines treatment. Curr Rev Musculoskelet Med. 2012;5:1–8.
2. Martin HD, Savage A, Braly BA, Palmer IJ, Beall DP, Kelly B. The function of the hip capsular ligaments: a quantitative report. Arthroscopy. 2008;24:188–95.
3. Martin HD, Hatem M, Kivlan BR, Martin RL. The function of the ligamentum teres in limiting hip rotation. Arthroscopy. 2014 (in review).
4. Bedi A, Dolan M, Leunig M, Kelly BT. Static and dynamic mechanical causes of hip pain. Arthroscopy. 2011;27:235–51.
5. Birmingham PM, Kelly BT, Jacobs R, McGrady L, Wang M. The effect of dynamic femoroacetabular impingement on pubic symphysis motion: a cadaveric study. Am J Sports Med. 2012;40:1113–8.
6. Hammoud S, Bedi A, Magennis E, Meyers WC, Kelly BT. High incidence of athletic pubalgia symptoms in professional athletes with symptomatic femoroacetabular impingement. Arthroscopy. 2012;28(10):1388–95.
7. Verrall GM, Slavotinek JP, Barnes PG, Esterman A, Oakeshott RD, Spriggins AJ. Hip joint range of motion restriction precedes athletic chronic groin injury. J Sci Med Sport. 2007;10:463–6.
8. Powers CM. The influence of abnormal hip mechanics on knee injury: a biomechanical perspective. J Orthop Sports Phys Ther. 2010;40:42–51.

9. DeAngelis NA, Busconi BD. Assessment and differential diagnosis of the painful hip. Clin Orthop Relat Res. 2003;406(1):11–8.
10. Kujala UM, Kaprio J, Sarna S. Osteoarthritis of weight bearing joints of lower limbs in former elite male athletes. Br Med J. 1994;308:231–4.
11. Harris WH. Traumatic arthritis of the hip after dislocation and acetabular fractures: treatment by mold arthroplasty. An end-result study using a new method of result evaluation. J Bone Joint Surg Am. 1969;51:737–55.
12. Byrd JW, Jones KS. Prospective analysis of hip arthroscopy with 2-year follow-up. Arthroscopy. 2000;16:578–87.
13. Martin RL, Mohtadi NG, Safran MR, Leunig M, Martin HD, McCarthy J, Guanche CA, Kelly BT, Byrd JW, Clohisy JC, Philippon MJ, Sekiya JK. Differences in physician and patient ratings of items used to assess hip disorders. Am J Sports Med. 2009; 37:1508–12.
14. Mohtadi NG, Griffin DR, Pedersen ME, Chan D, Safran MR, Parsons N, Sekiya JK, Kelly BT, Werle JR, Leunig M, McCarthy JC, Martin HD, Byrd JW, Philippon MJ, Martin RL, Guanche CA, Clohisy JC, Sampson TG, Kocher MS, Larson CM. The development and validation of a self-administered quality-of-life outcome measure for young, active patients with symptomatic hip disease: the International Hip Outcome Tool (iHOT-33). Arthroscopy. 2012;28: 595–605. quiz 606–510 e591.
15. Griffin DR, Parsons N, Mohtadi NG, Safran MR. A short version of the International Hip Outcome Tool (iHOT-12) for use in routine clinical practice. Arthroscopy. 2012;28:611–6. quiz 616–618.
16. Ware Jr JE, Sherbourne CD. The MOS 36-item short-form health survey (SF-36). I. Conceptual framework and item selection. Med Care. 1992;30:473–83.
17. Ware Jr J, Kosinski M, Keller SD. A 12-Item Short-Form Health Survey: construction of scales and preliminary tests of reliability and validity. Med Care. 1996;34:220–33.
18. Clohisy JC, Keeney JA, Schoenecker PL. Preliminary assessment and treatment guidelines for hip disorders in young adults. Clin Orthop Relat Res. 2005;441: 168–79.
19. Martin HD, Kelly BT, Leunig M, Philippon MJ, Clohisy JC, Martin RL, Sekiya JK, Pietrobon R, Mohtadi NG, Sampson TG, Safran MR. The pattern and technique in the clinical evaluation of the adult hip: the common physical examination tests of hip specialists. Arthroscopy. 2010;26:161–72.
20. Martin HD, Palmer IJ. History and physical examination of the hip: the basics. Curr Rev Musculoskelet Med. 2013;6:219–25.
21. Cibere J, Thorne A, Bellamy N, Greidanus N, Chalmers A, Mahomed N, Shojania K, Kopec J, Esdaile JM. Reliability of the hip examination in osteoarthritis: effect of standardization. Arthritis Rheum. 2008;59:373–81.
22. Martin HD. Clinical examination of the hip. Operat Tech Orthop. 2005;15:177–81.
23. Byrd JW. Operative hip arthroscopy. New York: Springer; 2005. p. 78–83.
24. Perry J. Gait analysis normal and pathological function. Thorofare: SLACK; 1992.
25. Ganz R, Parvizi J, Beck M, Leunig M, Notzli H, Siebenrock KA. Femoroacetabular impingement: a cause for osteoarthritis of the hip. Clin Orthop Relat Res. 2003;417:112–20.
26. McCarthy JC, Busconi BD, Owens BD. Assessment of the painful hip. In: McCarthy JC, editor. Early hip disorders. New York: Springer; 2003. p. 3–6.
27. Philippon M, Zehms C, Briggs K, Manchester D, Kuppersmith D. Hip instability in the athlete. Operat Tech Sport Med. 2007;15:189–94.
28. Martin RL, Palmer I, Martin HD. Ligamentum teres: a functional description and potential clinical relevance. Knee Surg Sports Traumatol Arthrosc. 2012;20: 1209–14.
29. Audenaert EA, Peeters I, Vigneron L, Baelde N, Pattyn C. Hip morphological characteristics and range of internal rotation in femoroacetabular impingement. Am J Sports Med. 2012;40:1329–36.
30. Martin HD, Kivlan BR, Palmer IJ, Martin RL. Diagnostic accuracy of clinical tests for sciatic nerve entrapment in the gluteal region. Knee Surg Sports Traumatol Arthrosc. 2013; [Epub ahead of print].
31. Szadek KM, van der Wurff P, van Tulder MW, Zuurmond WW, Perez RS. Diagnostic validity of criteria for sacroiliac joint pain: a systematic review. J Pain. 2009. 10(4):354–68.

Selective and Image Guided Injections Around the Hip and Pelvis

8

Kathleen Weber

Contents

K. Weber
Midwest Orthopaedics at Rush, Rush University Medical Center, Chicago, IL, USA
e-mail: kweber@rushortho.com

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_8

Abstract

Common conditions that cause pain in the hip, groin, and pelvic regions may include bursitis, tendonitis, and intra-articular pathology. The mainstay of treatment for these conditions is typically conservative. Conservative management often includes activity modification, anti-inflammatory medications, and rehabilitation exercises. When symptoms persist despite this treatment, then injections may be indicated. The use of ultrasound or fluoroscopic guidance increases the accuracy of the injection. This chapter will review a variety of disorders in the hip and pelvis that can cause pain and disability. The conditions that will be discussed will include gluteus medius, hamstring, and adductor tendinosis; iliopsoas, greater trochanteric, and gluteal bursitis; piriformis and lateral cutaneous nerve syndrome; and intra-articular pathology. First, a brief overview of these conditions will be provided and followed by the injection technique.

Introduction

Hip and pelvis disorders have gained increasing attention in orthopedic and sports medicine practice. These disorders can be painful, limit function, and at times present as diagnostically challenging. Most of these conditions are initially treated conservatively with home or formal physical therapy, modalities, nonsteroidal anti-inflammatory medications, and when necessary localized injections. Intra-articular and soft tissue injections are typically corticosteroid based, but platelet-rich plasma has also been used in specific conditions. These injections may be used to confirm a diagnosis by injecting a combination of local anesthetic and corticosteroid solution into a symptomatic area resulting in immediate reduction or elimination of the pain. The goal for therapeutic use is to reduce or eliminate pain and to increase function and mobility.

This chapter will focus on specific hip and pelvis pathology and the use of injections, when deemed clinically appropriate. The information will be presented with a brief overview of the diagnosis, common causes, pertinent physical examination findings, treatment options, and the injection technique. The chapter will cover a variety of injections including bursae, tendons, and joints. The use of ultrasound and fluoroscopy can assist in the accuracy of the injection placement.

Greater Trochanteric Bursitis/Greater Trochanteric Pain Syndrome

Trochanteric bursitis commonly is implicated in the cause of lateral hip pain [1]. The greater trochanteric bursa lies between the greater trochanter and the iliotibial band and the tendon of the gluteus medius. The bursa can be a single bursa sac or multi-segmented and loculated. Other bursae have also been described along the greater trochanter. At least three bursae are present in most individuals at the lateral greater trochanter and associated with the gluteus tendons, ITB, and tensor fascia latae [2].

Greater trochanteric bursitis can be acute caused by a direct blow or fall onto the lateral hip or chronic and cause intermittent pain that is noted lateral to the greater trochanter [2, 3]. Overuse and direct chronic pressure such as lying in a recumbent position on the affected hip also have been implicated in the cause of this bursitis [2, 4].

Chronic iliotibial band tightness or hip adductor weakness may result in chronic irritation of the bursa resulting in painful bursitis. The gluteus medius muscle abducts and medially rotates the thigh and stabilizes the pelvis in ambulation. Gluteus medius muscle weakness may contribute to lateral hip pain and bursitis especially in repetitive activity such as walking and running. Running on uneven surfaces or poor mechanics also may contribute to bursitis.

It is common that individuals presenting with lateral hip pain also will have pain and tenderness with palpation of the greater trochanter region accompanied with palpable pain in the lateral thigh and buttock region. Greater trochanteric pain syndrome (GTPS) may better characterize this broader condition. GTPS can be referred to as a regional pain syndrome and may be associated with bursitis, tendinitis, tendinosis, trigger points, gluteus medius or minimus tendon tears, and iliotibial band syndrome [5]. The regional pain may be related to the surrounding tissue or pain from other sources such as degenerative joint disease, myofascial pain, or lumbar pathology. This syndrome is common and it is estimated that 10–25 % of individuals in industrial societies are affected by GTPS [6–8].

Patients typically present with lateral hip pain after initiating an exercise program, but gluteus medius tendonitis or tendinopathy can develop from trauma, overuse, daily activities, or sports. Chronic repetitive activities in a setting of gluteus medius weakness can contribute to the tendinopathy. Patients with greater trochanteric bursitis complain of pain at the greater trochanter or pain that radiates down the lateral side of their leg from the greater trochanteric region to the knee. This pain may mimic sciatic-type pain but it differs in that it does not extend distal to the proximal tibia attachment. Lying on the affected side, rising from a seated to a standing position, and ascending stairs may exacerbate pain. It is not uncommon for patients that have greater trochanteric bursitis also to have lateral hip pain associated with iliotibial band, tendinitis, and muscle strains/tears.

GTPS is suggested by point tenderness to the posterolateral area of the greater trochanter, gluteus medius, gluteus minimus tendon, or posterolateral trigger points [2, 10]. Physical examination typically reveals localized tenderness with palpation of the greater trochanter. Pain is reproduced with extremes of passive adduction, abduction, or rotation and active resistance of hip abduction. Pain may be elicited by flexion, abduction, and external rotation. Rasmussen [9] described criteria for the diagnosis of trochanteric bursitis. The criteria require lateral hip pain and pain with palpation of the greater trochanter. Additionally included is one of the following: pain with passive abduction; pain with extremes of rotation, abduction, or adduction; and pseudoradiculopathy [9]. Pain with resisted hip abduction helps establish the gluteus medius tendon as the pain generator rather than greater trochanteric bursitis.

MRI study results suggest that lateral hip pain more often is associated with gluteus medius tendinopathy and less often solely a result of greater trochanteric bursitis [14–16]. If greater trochanteric bursitis is noted on imaging, it is typically in combination with gluteus medius tendinopathy [14, 16]. The gluteus medius tendon is subject to tears, tendinitis, and tendinosis [16].

The treatment of GTPS includes topical or oral nonsteroidal anti-inflammatory medications, modalities, physical therapy, and activity modification. Physical therapy should focus on stretching, soft tissue mobilization, and gluteus medius strengthening. When symptoms are not responsive to or cannot tolerate physical therapy, a localized corticosteroid injection can be performed. Corticosteroid and local anesthetics have been shown to have positive response rates of 60–100 % for providing pain relief [9, 11, 13].

Greater Trochanteric Bursa Injection Technique

Position the patient on the unaffected side with the lower leg flexed and upper leg extended. Palpate and mark the greater trochanter at the maximal point of discomfort. Cleanse the site in typical sterile fashion. Insert the needle and slowly advance the needle perpendicular to the skin directed at the point of maximal tenderness on the greater trochanter (Fig. 1). Once the needle touches the bone, slightly withdraw the needle

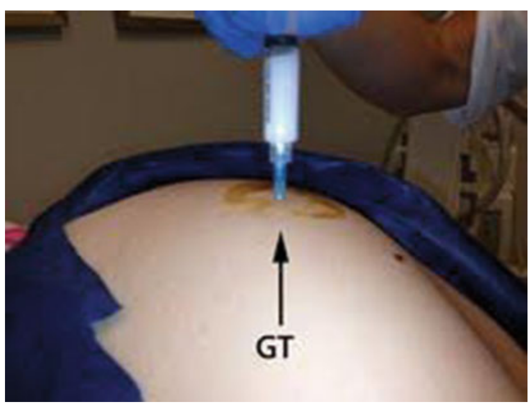


Fig. 1 Greater trochanteric bursa injection. The needle is inserted perpendicular to the skin and directed at the point of maximal tenderness at the greater trochanter (GT)

and slowly inject the solution without resistance. Adjust the needle position in different directions around the greater trochanter while distributing the medication to assure adequate spread of the steroid solution. If symptoms persist immediately after the injection of the corticosteroid and anesthetic injection, it may suggest an alternative diagnosis such as tendonitis, tendon tear, other bursa involvement, or inaccurate placement of the injection [12, 13].

Gluteus Medius Tendon Injection Technique

Position the patient in the lateral decubitus position on the unaffected side. Clean the area in the typical sterile fashion. Palpate the area of localized tenderness. Place the ultrasound probe in an oblique coronal plane using the long axis for needle advancement and medication placement. First, anesthetize the skin and subcutaneous affected area, using the ultrasound. Once the superficial area is anesthetized, slowly advance the 25-gauge needle to the lateral part of the tendon at the attachment on the greater trochanter using continuous ultrasound (Fig. 2). Position the needle tip and inject the corticosteroid solution under real-time ultrasound. Remove the needle and cover with a sterile dressing.

Fig. 2 Ultrasound imaging of a gluteus medius tendon injection. *GM* gluteus medius, *GT* greater trochanter

Aftercare

Avoid causative activity for 1 week and then gradually increase activity. Stretching of the iliotibial band and strengthening of the hip abductors should be encouraged. At times the addition of modalities, formal physical therapy for focused hip stretching and strengthening, NSAIDs – oral or local – and modified exercise can be helpful.

Gluteal Bursitis

Gluteal bursitis can occur as a result of acute trauma such as a direct fall onto the buttocks or repetitive overuse activities such as running. Typically, the patient will complain of pain over the upper outer quadrant of their buttock. The patient may complain of difficulty sleeping on the involved side.

Physical exam findings will reveal pain when palpating the upper outer quadrant of the affected buttock. Pain may be reproduced by passive flexion and adduction and resisted extension and abduction of the extremity.

The gluteal bursae vary in number and location and are located between the layers of the gluteal muscles and between the muscles and the surface of the ilium. When planning the placement of the injection, palpate the area and identify the location of the painful site.

Gluteal Bursa Injection Technique

Position the patient in a recumbent position on the unaffected side. Extend the lower leg and flex the upper leg. Identify and mark the tender area in the upper outer quadrant of the buttock. Cleanse the injection site in typical sterile fashion. Insert a

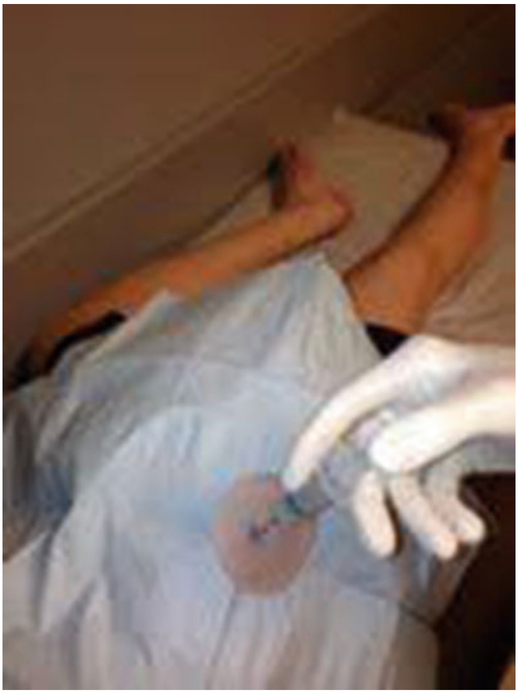


Fig. 3 Gluteal bursa injection. Positioned on the unaffected side. The needle is inserted at the point of maximal pain in the upper outer quadrant of the affected buttock

22- or 25-gauge needle perpendicular to the skin at the point of maximal pain until it touches the ilium (Fig. 3). Once the needle is positioned, aspirate. If no blood or paresthesias is noted, inject the contents of the syringe. There should be minimal resistance to injection flow. If paresthesias are noted during the procedure, immediately relocate the needle as it may have come into contact with the sciatic nerve. Remove the needle and place a sterile dressing over the site.

Aftercare

The patient should be instructed to observe for any signs of irregularity at the injection site. The patient should be instructed to modify activity to avoid excessive use of the lower extremities for 1 week and then gradually resume normal activities. Physical therapy may be indicated to address the etiology of the bursitis.

Iliopsoas Bursitis

The iliopsoas muscle is composed of the iliacus, psoas major, and psoas minor. The iliopsoas tendon inserts on the lesser trochanter and acts as a hip flexor. The iliopsoas bursa is positioned between the distal iliopsoas tendon and the anterior aspect of the femoral neck.

Iliopsoas bursitis and tendonitis are due to overuse and can be a source of hip and groin pain. These conditions are seen in activities that require repeated hip flexion. Runners, hurdlers, and ballet dancers are examples of athletes that may present with iliopsoas bursitis or tendonitis. Iliopsoas bursitis may occur after acute trauma, a total hip arthroplasty, or in rheumatoid arthritis patients [17, 18].

Patients likely will complain of groin pain and may note radiation of pain down the anterior thigh toward the knee. Pain is worsened by activities such as running or daily activities such as walking up stairs.

Physical examination may note tenderness in the area of the inguinal ligament. Pain may be elicited by passive hip flexion, adduction, abduction, and at times extension. Resisted hip flexion or passive hyperextension may reproduce pain. A snap may be heard or felt in the inguinal area when the hip is flexed, abducted, and externally rotated, and then the hip is extended.

Conservative treatment includes anti-inflammatories, modified activities, stretching, and physical therapy. If symptoms persist despite this management, then a corticosteroid anesthetic injection could be considered.

Iliopsoas Bursa Injection

Proper injection technique is critical since the bursa lies deep to the femoral vein, artery, and nerve. The use of ultrasound can allow for accurate placement of the injection [19].

Technique – Position the patient supine. Palpate the femoral artery. Cleanse the injection site in a typical sterile fashion. Place the ultrasound transducer in an oblique orientation that is parallel

Fig. 4 Iliopsoas bursa injection. Note the bursa is distended (Photo courtesy of John Hill, DO, University of Colorado)

to the femoral neck. Use a lateral to medial approach and real-time ultrasound to insert a 22- or 25-gauge spinal needle at the level of the iliopectineal eminence. The bursa lies between the iliopsoas tendon and the anterior hip capsule. Confirm proper placement by real-time ultrasound as the bursa fills (Fig. 4). When injecting the solution, no resistance should be noted. If resistance is met, withdraw further until fluid flows uninhibited. Remove the needle and place a sterile dressing over the injection site.

Aftercare

The patient should be instructed to observe for any signs of irregularity at the injection site. Instructions should be provided to limit causative activity for a week or longer depending on symptoms.

Potential Complications

Although uncommon, anesthesia of the lateral branch of the femoral nerve may occur. If this occurs the patient will experience temporarily anesthesia of the quadriceps. Keeping the injection lateral as described above will help avoid this rare complication. Additionally, it is advisable to have the patient report immediately any symptoms of tingling or nerve sensation while performing the injection. If these symptoms are reported, reposition the needle prior to injecting the solution.

Adductor Tendonitis

The adductor muscles of the hip include the adductor longus, adductor brevis, and the adductor magnus. This tendon group originates along the pubis and ischial tuberosity and is responsible for hip adduction. The tendon group is susceptible to developing tendonitis from overuse and traumatic stretching injuries. The tendon can be injured along the proximal tendon or at the more common area at the site of the attachment at the pubic area. Tendinitis can occur with repetitive overuse activity. The adductor longus muscle is most frequently injured. This muscle is susceptible to injury with activities that require sprinting with sudden directional changes such as basketball, soccer, or hockey.

Acute injuries typically occur at the muscle-tendon junction but can occur at the bone-tendon attachment [20] (Fig. 5). A partial or full thickness tears can occur and inspection of the region may demonstrate bruising on the inner aspect of the thigh.

Individuals with adductor injuries will complain of localized pain at the origins of the tendons and along the muscle with palpation. The pain can be sharp, constant, and severe. Pain is elicited with passive abduction or resisted adduction of the affected leg.

Initial treatment includes rest, ice, compression, and nonsteroidal anti-inflammatory medications [21]. Physical therapy may be helpful to assist with stretching, correct biomechanical abnormalities and functional deficits, and progress strengthening [22].

If chronic adductor pain persists despite conservative management, then a corticosteroid injection at the base of the tendon or at the site of inflammation may be considered. Schilders et al. [23] reported that a single entheseal pubic cleft injection provided short-term improvement, mean of 5 weeks, in a competitive athlete with

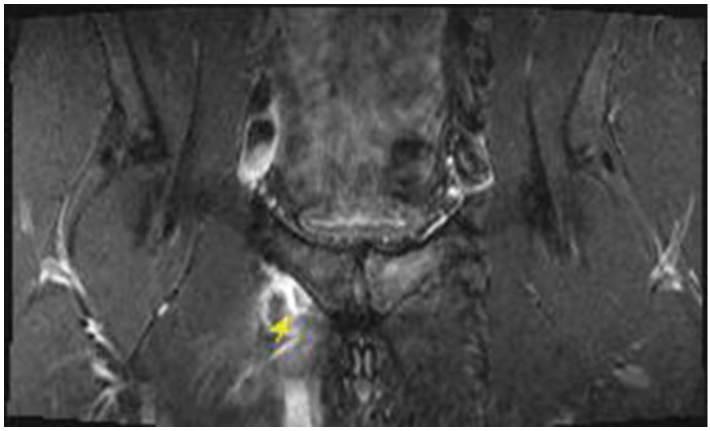


Fig. 5 Full thickness tear with retraction of the adductor longus and brevis muscle in a semiprofessional hockey player

evidence of enthesopathy on magnetic resonance imaging (MRI). They reported improved outcomes in recreational athletes even when abnormalities were found on MRI [23].

Adductor Tendon Injection Technique

Position the patient supine with the affected leg slightly abducted and externally rotated. Cleanse the area in the typical sterile fashion. Palpate the origin of the tendon. Insert the 25-guage needle directed at the pubic bone (Fig. 6). Once the needle touches the bone, pepper the solution into the area. Ultrasound or fluoroscopic guidance can be used for this procedure.

Aftercare

Educate the patient on the signs and symptoms of an infection. Activity should be limited following the injection and gradually increase as tolerated as prescribed by the treating physician. A combination of stretching and strengthening exercises should be incorporated to overcome any weakness or existing muscle imbalances.

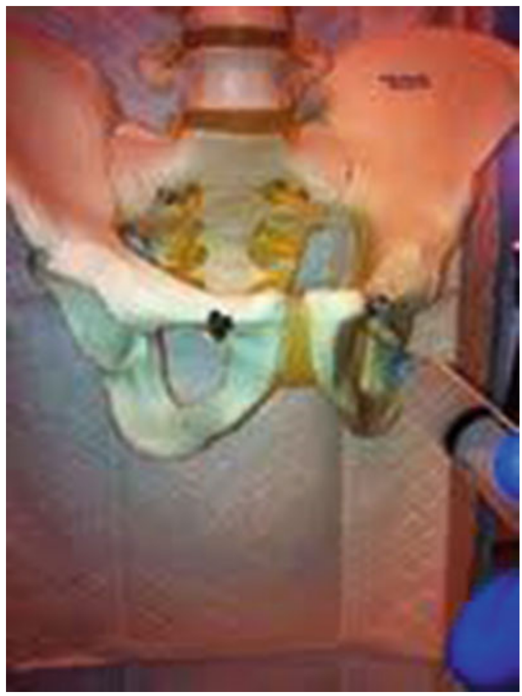


Fig. 6 Adductor tendon injection. Insert the 25-guage needle directed at the pubic bone. Once the needle touches the bone, pepper the solution into the area

Hamstring Tendon Injuries

Hamstring injuries are common [20]. Although the muscle-tendon junction is a common site for the hamstring to tear [20, 24], proximal hamstring injuries can occur and be a source of acute or chronic pain (Fig. 7). Proximal hamstring pain can be acute in sudden tendon rupture or chronic from overuse resulting in tendinosis [25].

The hamstring tendons originate on the ischial tuberosity. The conjoint tendon consisting of the biceps femoris and semitendinosus inserts posterior and inferior on the tuberosity, while the

Fig. 7 Proximal hamstring tendinosis with a partial tear (*arrow*). Ischial tuberosity (IT)

Fig. 8 Ultrasound-guided right semitendinosus injection. The needle is just superior to the ischial tuberosity (IT) (Photo courtesy of John Hill, DO, University of Colorado)

semimembranosus tendon attaches more proximal and anteriorly just medial to the sciatic nerve. Hamstring injuries can mimic sciatic-type symptoms and may coexist with sciatic nerve irritation. The ischial bursa can be found between the ischial tuberosity and the gluteus maximus and can be a source of pain.

Pain arising from the proximal hamstring tendon can occur suddenly, as in an acute eccentric strain or direct trauma, resulting in an acute rupture or incomplete tear. Chronic hamstring tendinosis is seen in overuse and repetitive activity such as running.

Patients typically present with activity-related soreness in the ischial region or inability to participate in sports secondary to pain. Sitting can become difficult especially noted with prolonged car rides or sitting for long periods. Pain is elicited on exam by palpation of the ischial tuberosity, passively stretching the hamstring, resisted flexion of the knee, or hip extension.

Treatment is nonoperative unless there is an indication for surgical treatment such as proximal hamstring avulsion fracture or failed conservative management [25–27]. Initially, treatment includes rest, ice, compression, and elevation. Physical therapy can be utilized for modalities, soft tissue mobilization, and gradual strengthening [28].

An MRI can be performed if symptoms persist despite conservative management or the clinical examination indicates a need for further evaluation of the tendon. If the MRI findings do not suggest that surgery is indicated, a corticosteroid injection can be considered [29].

Hamstring Injection Technique

Place the patient either in the prone position or on the unaffected side. Palpate and mark the affected area. Clean the area in typical sterile fashion. Using real-time ultrasound guidance in the long axis, insert the 22- or 25-gauge needle directed at an angle to the ischial tuberosity staying medial to the sciatic nerve (Fig. 8). Continue to insert the needle until the tuberosity is touched and then pepper the solution into the peritendinous-osseous region. If bursitis is suspected, deposit the solution into the bursa. If trauma preceded the pain, such as a hard fall onto the area, an aspiration of blood may be required prior to the injection of the steroid solution.

Aftercare

Educate the patient on the signs and symptoms of an infection. Avoid prolonged sitting or offending activities for at least a week. Thereafter, activity should be gradually increased as tolerated as prescribed by the treating physician.

Intra-articular Hip Conditions

Hip pain can be caused by numerous conditions including osteoarthritis, rheumatoid arthritis, labral tears, and, less commonly, collagen vascular disease, infection, and avascular necrosis and referred from the lumbar spine [30]. The most common of these diagnoses is osteoarthritis. The clinician should consider the differential and perform a diagnostic work-up based on the patient's history and presenting symptoms. Plain radiographs can help in the diagnosis. At times further imaging with MRI or MRI arthrogram may be needed in determining the diagnosis [30].

The majority of patients with intra-articular pathology present with hip or groin pain; however, some report thigh and/or knee pain. The clinician should do a thorough evaluation of the lumbar spine, hip, and knee when determining the source of the pain.

Common complaints of patients include symptoms of stiffness and pain after inactivity such as rising from bed, prolonged standing, or walking and transitional pain such as rising after sitting for a prolonged time. Rest typically relieves the pain. If pain is related to arthritis, patients may report gradual decrease in their functional activity. Over time range of motion will be diminished and they may report difficulty crossing their legs or putting on their shoes and socks.

Physical examination findings include limited hip range of motion, pain with flexion, and internal rotation (IR). Hip abduction and extension may also elicit pain.

Plain radiographs can help determine joint space, inflammatory changes, avascular necrosis, and other deformities such as hip dysplasia and hip impingement. However, further imaging with MRI or MRI arthrogram may be indicated based on clinical history and examination.

Intra-articular Hip Injection Technique

The use of fluoroscopic guidance or ultrasound assures the placement of the medication solution in the joint.

Position the patient on his back. Position either the fluoroscope or the ultrasound probe in the proper position to visualize the joint. Cleanse the area using sterile technique. Identify the landmarks of the ASIS and the greater trochanter. Palpate the femoral artery. Insert the needle along the line directed medially at the midportion of the femoral head. Advance the needle until the bone is touched. Aspirate to confirm no blood and then inject a few milliliters of air into the hip. Confirm placement with the joint using an air arthrogram, and then inject the steroid solution (Fig. 9). Remove the needle and place a sterile dressing over the site.

Fig. 9 Right intra-articular hip injection. Air arthrogram confirms the needle is intra-articular

Aftercare

Educate the patient on the signs and symptoms of an infection. Limit activities for approximately 1 week to activities of daily living. Encourage stretching and strengthening activities. Participation in aerobic activities especially non-weight-bearing may be found to be helpful. Weight loss, when appropriate, should be encouraged.

Sacroiliac Joint Dysfunction

Sacroiliac (SI) pain has a variety of potential causes including ligamentous strain, joint hypomobility or hypermobility, degenerative arthritis, trauma, inflammatory arthritis, muscular imbalances, infection, stress or insufficiency fracture, and/or abnormal shear forces resulting in joint inflammation and pain [31]. Pregnant patients or patients with prior lumbar spinal fusion may develop SI pain that may be related to the transfer of stress to the joints. The clinical history and presentation will determine if and when additional testing is required to determine the cause (i.e., imaging and cultures). A fluoroscopy-guided injection into the SI joint that relieves the patient's pain is considered diagnostic [32].

The SI joint forms an articulation between the sacral alae and the iliac bones and is diarthrotic. The joint is strengthened by its interosseous ligaments. The L3 to L5 nerve roots innervate the SI joint likely contributing to its ill-defined pain pattern. SI pain can be confused as originating from the lumbar spine. SI joint pain can be localized to the SI joint, referred into the buttock or into the lower extremity. The described pain typically does not extend below the knee but has been found in some patients to be the source of lower leg, foot, and ankle pain.

Patients may present with lower back pain, buttock, or radiating pain into the lower extremity. It is typically worse with activity but may be present at rest. The pain can be constant and achy and may interfere with sleep.

Physical examination should include a back, hip, and neurological examination. Pain is elicited by palpation of the SI joint region. Common assessment of the SI joint will include an evaluation of the pelvic alignment, Patrick test, Gaenslen test, Gillet test, distraction, compression, thigh thrust, and sacral thrust. Although a number of SI joint tests have been described in diagnosing SI joint disorders, none have strong scientific validation [33].

A diagnostic anesthetic block performed using fluoroscopic guidance with a placebo-controlled comparative local anesthetic is the best available reference standard for confirming SIJ-mediated pain [34]. A greater or equal to 80 % relief and ability to do previously painful movements following the injection is considered a positive response [34]. Long-term relief attributed to the corticosteroid should also be noted.

SI Injection Technique

Patients with suspected SI generated pain may benefit from an SI injection. However, there is no clear evidence from controlled trials that support a benefit to the use of steroid injections in non-spondyloarthropathic conditions [34, 35]. The use of fluoroscopic imaging is preferred as nonimage-assisted technique reveals a low target specificity [36].

Position the patient prone on the fluoroscopy table. Cleanse the injection area in the typical sterile fashion. Use the fluoroscopy tube to view the SI joint and position the placement of the injection at the inferior aspect of the joint. Anesthetize the skin and then advance a 22- or 25-gauge spinal needle through the posterior longitudinal ligament via fluoroscopic guidance. If bone is encountered while positioning the needle, withdraw into the subcutaneous skin and redirect. Contrast radiopaque dye can confirm placement in nonallergic patients (Fig. 10). Once needle placement is confirmed, inject the solution. If resistance to flow occurs, the placement may be in the ligament. Advance the needle into the joint until the fluid can flow without significant resistance. Remove the needle and apply a sterile dressing.

Fig. 10 Sacroiliac joint injection. Contrast material is noted to extend superior in the sacroiliac joint, confirming intra-articular placement

Aftercare

It may be helpful to have the patient fill out a pain diary following the injection to elicit and document the response to the injection. The patient should look for and report any signs of redness, warmth, or drainage from the site. The area should be kept clean. Normal daily activities are encouraged, but avoidance of invoking activities and activity modification should be encouraged for at least 1 week.

Osteitis Pubis

Osteitis pubis is a painful condition involving inflammation of the pubis symphysis joint and the surrounding area [37]. Pain typically is localized at the pubis but also can radiate into the inner thigh and lower abdominal wall. This process is more common in the second through fourth decades and in females. This syndrome should be considered in the differential diagnosis of athletes that present with groin pain especially if their pain is localized to the pubis symphysis. Athletes that are diagnosed and treated early can return to play sooner [38].

Osteitis pubis has been associated with urological, gynecological, inguinal, or prostate procedures [39, 40]. The occurrence of osteitis pubis in athletes may be related to periosteal trauma from direct injury or repetitive microtrauma from participation [41].

Physical examination findings reveal tenderness with palpation of the pubic symphysis. Tenderness may also be elicited with palpation of the origin of the rectus abdominus and adductor muscles or the adjacent pubic bones. Resisted strength testing of the adductor and lower abdominal may reproduce the pain. Pelvic alignment should be evaluated.

Plain radiographs are the first step in imaging. Radiographic findings that are pathognomonic for this condition are erosion, sclerosis, and widening of the pubic symphysis. However, not all patients will have radiographic changes. An MRI can be helpful as it can show bone edema in the pubic bones at the pubis [42].

The MRI can also help identify and eliminate other potential etiology for the groin pain such as sports hernia, stress fracture, or muscle strain. If osteomyelitis is suspected, a bone scan along with erythrocyte sedimentation rate may be utilized.

Osteitis pubis is typically treated with nonsteroidal anti-inflammatories, physical therapy, and activity modification. Physical therapy should include correction of pelvic malalignment, core, hip, and gluteal strengthening.

If the patient has persistent pain despite conservative management, then a pubic symphysis corticosteroid injection may be considered. Studies have demonstrated that subjects with both acute and chronic pain have noted improvement following injections [43]. Randomized control studies need to be done to further determine the efficacy and timing of these injections.

Pubic Symphysis Injection Technique

The injection can be without visual guidance. However, using ultrasound or fluoroscopic

Fig. 11 Pubic symphysis injection. Needle is advanced slowly and perpendicular to the skin directed at the symphyseal cleft

guidance improves injection accuracy and reduces complication risk. The technique described in this section uses fluoroscopic guidance.

Position the patient supine on the fluoroscopy table. The area is cleansed in typical sterile technique. Palpate the pubic symphysis and use fluoroscopic imaging to confirm injection location. Inject the skin and subcutaneous tissue to the level of the pubis with local anesthetic. Once the tissue is anesthetized, slowly advance a 25-gauge needle perpendicular to the skin directed at the symphyseal cleft (Fig. 11). If bone is encountered, reposition until a mild resistance is met, indicating the needle is just outside the joint. Advance the needle another centimeter to place the needle in the fibrocartilaginous disk. Be careful not to advance too far as the bladder may be punctured. Aspirate for blood, and if none, inject a small amount of nonionic contrast material to confirm placement as it outlines the disk. There should be minimal resistance while injecting. This may reproduce the patient's symptoms. Once placement is established, inject the corticosteroid solution into the joint. Remove the needle. Reexamination should reveal symptom resolution.

Aftercare

Inform the patient to keep the area clean and dry. A pain diary may be helpful to track the patient's response to the injection. The patient should be instructed to resume activities of daily living, but a return to more strenuous activity should be prescribed individually.

Piriformis Syndrome

The piriformis muscle is flat and pyramidal in shape and originates from the anterior sacrum, margin of the greater sciatic foramen, and the sacrotuberous ligaments anterior surface. It inserts at the upper border of the greater trochanter. The piriformis muscle is a lateral rotator of the hip. During hip extension the piriformis laterally rotates the femur and with hip flexion it abducts the femur. The function of this muscle is important in lower extremity movement as it stabilizes the hip, enables us to walk, and maintains balance.

The sciatic nerve as it passes through the gluteal region either pierces through the piriformis muscle or lies beneath the muscle as it extends down the leg. Piriformis syndrome results from the sciatic nerve being irritated by the piriformis muscle resulting in sciatic-type pain. The nerve can be compressed by a tight piriformis muscle or by external compression such as prolonged sitting or pressure from a wallet in the back pocket.

Clinically, patients may complain of buttock pain, tingling, numbness, and possibly radiation of pain posterior into the affected thigh and leg. Prolonged sitting or activities such as running can aggravate these symptoms. This syndrome must be differentiated from a lumbar etiology such as a radiculopathy, sacral iliac pain, hip, or hamstring pathology. Some clinicians consider piriformis syndrome a diagnosis of exclusion.

Physical examination often reveals tenderness and tightness of the piriformis muscle. Deep palpation of the piriformis muscle belly may illicit pain in the buttock and/or along the sciatic nerve distribution. Stretching of the muscle may cause or relieve symptoms. A lumbar, hip, and neurological examination should be performed to evaluate for other causes of patients' complaints.

Plain radiographs, magnetic resonance imaging (MRI), computed tomography (CT), or electromyography (EMG) may be necessary to rule out other sources of the pain. However, the diagnosis is typically made on the clinical history and examination findings.

This condition is typically responsive to conservative treatment. The mainstay of treatment should include stretching of the piriformis to relieve compression of the nerve. Additionally, modification or avoidance of activities that increase compression on the sciatic nerve such as prolonged sitting may be helpful. Rest, ice, NSAIDs, massage and formal physical therapy may be helpful. If these measures fail to relieve the pain and dysfunction, a therapeutic corticosteroid and anesthetic injection can be done. Although not commonly used, botulinum toxin (Botox) has been shown to be effective in relieving muscle tightness and minimizing pain. The combination of either the corticosteroid/anesthetic or Botox injection with physical therapy has been shown to provide symptomatic improvement [44, 45].

Piriformis Injection Technique

Inform the patient to report any numbness, tingling, or sciatic-type symptoms during the injection procedure.

Position the patient prone. The area is cleaned in the typical sterile fashion. Palpate the area of localized tenderness. The ultrasound probe is used to identify the greater trochanter. Place the probe in the long axis between the greater trochanter and the ischial tuberosity and identify the location of the sciatic nerve. Internally and externally rotate the hip to help identify the piriformis. Place the probe in the long axis parallel with the piriformis muscle. Anesthetize the skin and subcutaneous tissue. Once the superficial area is anesthetized, using real-time ultrasound, slowly advance the 25-gauge needle in the long axis from medial to lateral into the piriformis muscle (Fig. 12). Once placement is confirmed, inject the solution under real-time ultrasound into the muscle. Remove the needle and cover with a sterile dressing.

Fig. 12 Ultrasound-guided piriformis injection. The X marks an area of hypoechoicity in the piriformis muscle (Photo courtesy of John Hill, DO, University of Colorado)

Lateral Cutaneous Femoral Nerve Syndrome

Lateral femoral cutaneous nerve syndrome is also known as meralgia paresthetica. This syndrome is typically unilateral. Compression of this sensory nerve is most susceptible as it exits the pelvis medial to the anterosuperior iliac spine (ASIS) at the inguinal ligament. Compression of the nerve produces pain, dysesthesia, or hypesthesia over the anterolateral and lateral thigh [46]. Nerve conduction studies may be diagnostic for nerve compression, revealing prolonged latency or decreased velocity [47]. Meralgia paresthetica is a clinical diagnosis but can also be confirmed with a localized anesthetic injection.

Conditions that may result in the nerve injury or nerve compression include obesity, wearing tight pants, belts (weightlifting, tool), or direct trauma. Intra-abdominal or pelvic causes include pregnancy, tumors, or scar tissue. The nerve can also be injured during anterior surgical approaches to the pelvis, acetabulum, or hip. Walking or hip extension may make the symptoms worse.

Physical examination findings will display hypoesthesia or dysesthesia in the distribution of the lateral femoral cutaneous nerve. A positive Tinel's sign and applying pressure over the exiting nerve at the ASIS or just medial to the ASIS can produce pain and/or paresthesias in the nerve distribution.

Plain radiographs of the pelvis should be performed to rule out a bone abnormality. If the hip is suspected as a potential source of the pathology, then hip films should be obtained.

Treatment of meralgia paresthetica includes eliminating the source of compression (i.e., weightlifters belt and loosen clothing). If overweight or obese, weight loss should be encouraged. Neuropathic pain medications may reduce symptoms. Localized injection of a corticosteroid preparation at the exiting site of the nerve may be helpful.

Fig. 13 Lateral cutaneous femoral nerve injection. The skin is penetrated just medial and caudal to the anterior superior iliac spine (ASIS) and inferior to the inguinal ligament (*yellow line*)

Lateral Cutaneous Femoral Nerve Injection Technique

Position the patient supine on the table. The ASIS on the affected side is identified. Cleanse the area in typical sterile fashion. Use a 25-gauge needle to anesthetize the skin just medial and caudal to the ASIS and inferior to the inguinal ligament. Perform an injection using the corticosteroid solution 1–2 cm deeper at a 60° angle to the skin (Fig. 13). Remove the needle and place a sterile dressing over the injection site.

Summary

Determining the cause of hip and pelvis pain can be diagnostically challenging. Frequently, there can be underlying conditions that make diagnosis difficult. A targeted injection can be helpful in determining the source of pain and may be both diagnostic and potentially therapeutic. The use of ultrasound or fluoroscopic guidance can assist in proper placement of the injection.

References

1. Margo K, Drezner J, Motzkin D. Evaluation and management of hip pain: an algorithmic approach. J Fam Pract. 2003;52:607–17.
2. Shbeeb MI, Matteson EL. Trochanteric bursitis (greater trochanter pain syndrome). Mayo Clin Proc. 1996;71:565–9.
3. Alvarez-Nemegyei J, Canoso JJ. Evidence-based soft tissue rheumatology: III: trochanteric bursitis. J Clin Rheumatol. 2004;10:123–4.
4. Jones DL, Erhard RE. Diagnosis of trochanteric bursitis versus femoral neck stress fracture. Phys Ther. 1997;77:58–67.
5. Tortolani PJ, Carbone JJ, Quartararo LG. Greater trochanteric pain syndrome in patients referred to orthopedic spine specialists. Spine J. 2002;2:251–4.
6. Segal NA, Felson DT, Torner JC, Zhu Y, Curtis JR, Niu J, et al. Greater trochanteric pain syndrome: epidemiology and associated factors. Arch Phys Med Rehabil. 2007;88:988–92.

7. Lievense A, Bierma-Zeinstra S, Schouten B, Bohnen A, Verhaar J, Koes B. Prognosis of trochanteric pain in primary care. Br J Gen Pract. 2005;55:199–204.
8. Collee G, Dijkmans BA, Vandenbroucke JP, Cats A. Greater trochanteric pain syndrome (trochanteric bursitis) in low back pain. Scand J Rheumatol. 1991;20:262–6.
9. Rasmussen K, Fano N. Trochanteric bursitis. Treatment by corticosteroid injection. Scan J Rheumatol. 1985;14:417–420.
10. Gordon EJ. Trochanteric bursitis and tendinitis. Clin Orthop. 1961;20:193–202.
11. Shbeeb MI, O'Duffy JD, Michet Jr CJ, O'Fallon WM, Matteson EL. Evaluation of glucocorticosteroid injection for the treatment of trochanteric bursitis. J Rheumatol. 1996;23:2104–6.
12. Walker P, Kannangara S, Bruce WJ, Michael D, Van der Wall H. Lateral hip pain: does imaging predict response to localized injection? Clin Orthop Relat Res. 2007;457:144–9.
13. Cohen SP, Narvaez JC, Lebovits AH, Stojanovic MP. Corticosteroid injections for trochanteric bursitis: is fluoroscopy necessary? A pilot study. Br J Anaesth. 2005;94:100–6.
14. Kingzett-Taylor A, Tirman PF, Feller J, et al. Tendinosis and tears of gluteus medius and minimus muscles as a cause of hip pain: MR imaging findings. AJR Am J Roentgenol. 1999;173:1123–6.
15. Chung CB, Robertson JE, Cho GJ, Vaughan LM, Copp SN, Resnick D. Gluteus medius tendon tears and avulsive injuries in elderly women: imaging findings in six patients. AJR Am J Roentgenol. 1999;173:351–3.
16. Bird PA, Oakley SP, Shnier R, Kirkham BW. Prospective evaluation of magnetic resonance imaging and physical examination findings in patients with greater trochanteric pain syndrome. Arthritis Rheum. 2001;44:2138–45.
17. Johnston CA, Wiley JP, Lindsay DM, Wiseman DA. Iliopsoas bursitis and tendinitis. A review. Sports Med. 1998;25(4):271–83.
18. Trousdale RT, Cabanela ME, Berry DJ. Anterior iliopsoas impingement after total hip arthroplasty. J Arthroplasty. 1995;10(4):546–9.
19. Adler RS, Buly R, Ambrose R, Sculco T. Diagnostic and therapeutic use of sonography-guided iliopsoas peritendinous injections. AJR Am J Roentgenol. 2005;185:940–3.
20. Garrett Jr WE. Muscle strain injuries. Am J Sports Med. 1996;24(6 Suppl):S2–8.
21. Fricker PA. Management of groin pain in athletes. Br J Sports Med. 1997;31:97–101.
22. Hoelmich P. Adductor-related groin pain in athletes. Sports Med Arthroscopy Rev. 1997;5:285–91.
23. Schilders E, Bismil Q, Robinson P, O'Connor PJ, Gibbon WW, Talbot JC. Adductor-related groin pain in competitive athletes. Role of adductor enthesis, magnetic resonance imaging, and entheseal pubic cleft injections. J Bone Joint Surg Am. 2007;89(10):2173–8.
24. De Smet AA, Best TM. MR imaging of the distribution and location of acute hamstring injuries in athletes. Am J Roentgenol. 2000;174:393–9.
25. Cohen S, Bradley J. Acute proximal hamstring rupture. J Am Acad Orthop Surg. 2007;15(6):350–5.
26. Cross MJ, Vandersluis R, Wood D, Banff M. Surgical repair of chronic complete hamstring tendon rupture in the adult patient. Am J Sports Med. 1998;26:785–8.
27. Clanton TO, Coupe KJ. Hamstring strains in athletes: diagnosis and treatment. J Am Acad Orthop Surg. 1998;6:237–48.
28. Fredericson M, Moore W, Guillet M, Beaulieu C. High hamstring tendinopathy in runners: meeting the challenges of diagnosis, treatment and rehabilitation. Phys Sportsmed. 2005;33(5):32–43.
29. Zissen MH, Wallace G, Stevens KJ, Fredericson M, Beaulieu C. High hamstring tendinopathy: MRI and ultrasound imaging and therapeutic efficacy of percutaneous corticosteroid injection. AJR Am J Roentgenol. 2010;195(4):993–8.
30. Jacobson JA, Bedi A, Sekiya JK, Blankenbaker DG. Evaluation of the painful athletic hip: imaging options and imaging-guided injections. AJR Am J Roentgenol. 2012;199:516–24.
31. Tibor LM, Sekiya JK. Differential diagnosis of pain around the hip joint. Arthroscopy. 2008;24(12):1407–21.
32. Dreyfuss P, Dreyer SJ, Cole A, Mayo K. Sacroiliac joint pain. J Am Acad Orthop Surg. 2004;12(4):255–65.
33. Dreyfuss P, Michaelsen M, Pauza K, McLarty J, Bogduk N. The value of medical history and physical examination in diagnosing sacroiliac joint pain. Spine. 1996;21:2594–602.
34. Manchikanti L, Boswell MV, Singh V, Benyamin RM, Fellows B, Abdi S, et al. Comprehensive evidence-based guidelines for interventional techniques in the management of chronic spinal pain. Pain Physician. 2009;12(4):699–802.
35. Maugars Y, Mathis C, Berthelot JM, Charlier C, Prost A. Assessment of the efficacy of sacroiliac corticosteroid injections in spondylarthropathies: a double-blind study. Br J Rheumatol. 1996;35(8):767–70.
36. Rupert MP, Lee M, Manchikanti L, Datta S, Cohen SP. Evaluation of sacroiliac joint interventions: a systematic appraisal of the literature. Pain Physician. 2009;12(2):399–418.
37. Schnute WJ. Osteitis pubis. Clin Orthop Relat Res. 1961;20:187–92.
38. Choi H, McCartney M, Best TM. Treatment of osteitis pubis and osteomyelitis of the pubic symphysis in athletes: a systematic review. Br J Sports Med. 2011;45:57–64.
39. Lentz SS. Osteitis pubis: a review. Obstet Gynecol Surv. 1995;50(4):310–5.
40. Johnson R. Osteitis pubis. Curr Sports Med Rep. 2003;2(2):98–102.

41. Batt ME, McShane JM, Dillingham MF. Osteitis pubis in collegiate football players. Med Sci Sports Exerc. 1995;27(5):629–33.
42. Gibbon WW, Hession PR. Diseases of the pubis and pubic symphysis: MR imaging appearances. AJR Am J Roentgenol. 1997;169(3):849–53.
43. Holt MA, Keene JS, Graf BK, et al. Treatment of osteitis pubis in athletes. Results of corticosteroid injections. AM J Sports Med. 1995;23:601–6.
44. Fishman LM, Dombi GW, Michaelsen C, Ringel S, Rozbruch J, Rosner B, et al. Piriformis syndrome: diagnosis, treatment, and outcome–a 10-year study. Arch Phys Med Rehabil. 2002;83(3):295–301.
45. Fishman LM, Anderson C, Rosner B. BOTOX and physical therapy in the treatment of piriformis syndrome. Am J Phys Med Rehabil. 2002;81(12):936–42.
46. Grossman MG, Ducey SA, Nadler SS, Levy AS. Meralgia paresthetica: diagnosis and treatment. J Am Acad Orthop Surg. 2001;9(5):336–44.
47. Harney D, Patijn J. Meralgia paresthetica: diagnosis and management strategies. Pain Med. 2007;8(8): 669–77.

9 Hip Osteoarthritis: Definition and Etiology

Lisa M. Tibor and Reinhold Ganz

Contents

L.M. Tibor (✉)
Kaiser Permanente Medical Center, South San Francisco, CA, USA
e-mail: lisa.tibor@gmail.com

R. Ganz
Emeritus Inselspital Bern, Bern, Switzerland

Faculty of Medicine, University of Bern, Bern, Switzerland
e-mail: rd.ganz@bluewin.ch

Abstract

Hip osteoarthritis (OA) is a degenerative process where continued cartilage breakdown results from mechanical overload, causing secondary bony and synovial changes and characteristic clinical and radiographic findings. Evidence is accumulating that primary hip OA is actually secondary to a subtle mechanical problem like mild dysplasia or femoroacetabular impingement (FAI). Dysplasia causes increased cartilage stress at the lateral acetabular rim, with labral hypertrophy and cartilage breakdown. FAI causes damage when the hip is flexed. Cam-type FAI causes cartilage delamination and separation of the labral-chondral junction, while pincer-type FAI causes a crushing injury to the labrum and a linear pattern of cartilage damage. Family history is a known risk factor for hip OA, and both FAI and dysplasia can be inherited. In addition, certain genotypes appear to make the cartilage more vulnerable to mechanical overloading. Nonetheless, not all radiographic hip OA is symptomatic, and not everyone with FAI or dysplasia ultimately develops hip OA. Thus, it appears that end-stage hip OA is a multifactorial process, caused by a combination of a structural deformity, wear due to activity, the inherent "robustness" of the cartilage, and the amount of inflammation that the individual experiences. The understanding of the structural factors that contribute to hip OA is advancing rapidly. It also appears that

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_9

identification and treatment of FAI and dysplasia help symptoms that result from early chondrolabral damage.

Introduction

Both patients and physicians often use the terms arthritis, arthrosis, and osteoarthritis interchangeably. Arthritis is, however, a nonspecific term that denotes inflammation of a joint, whereas arthrosis is defined as a degenerative affliction of a joint [1]. In contrast, osteoarthritis is a distinct pathologic process: “arthritis characterized by erosion of articular cartilage, either primary or secondary to trauma or other conditions, which becomes soft, frayed, and thinned with eburnation of subchondral bone and outgrowths of marginal osteophytes; pain and loss of function results” [1]. In this vein, the terms degenerative joint arthritis, degenerative joint disease, and osteoarthrosis are true synonyms for osteoarthritis.

Although there is debate about the factors that initiate osteoarthritis, the pathologic process is characterized by progressive loss of the articular surface (Fig. 1) [2]. Initially there is cartilage fissuring, chondrocyte clustering, and some attempt at repair [2]. In this early state when the cartilage damage is confined to the articular surface and there is no associated subchondral reaction, this could also be considered “arthritis” as there is no “osteo” component. As the degenerative process progresses, the subchondral bone remodels and appears sclerotic on radiographs. Among osteoarthritis researchers, there are competing theories about the cause of these subchondral bone changes and whether they occur in response to the cartilage damage or if they occur in response to increased load even before the cartilage has been macroscopically damaged. Nonetheless, as the joint degenerates further, the process is consistent. The synovium and capsule thicken, marginal osteophytes form, and the subchondral bone may develop cysts.

Although osteoarthritis is not an inflammatory process in the same sense as the rheumatologic diseases that cause joint destruction, inflammation is clearly part of what causes radiographic osteoarthritis to become painful. When osteoarthritis becomes symptomatic, patients complain of joint pain, decreased motion, effusions, and crepitation and, in more advanced cases, may notice deformity due to ongoing bony destruction. Thus, to gather all of these concepts into a broad definition, osteoarthritis should be defined as a degenerative process where continued cartilage breakdown results from mechanical overload, causing secondary bony and synovial changes and characteristic clinical and radiographic findings.

In the hip, there are many new ideas about anatomic and biomechanical factors that may ultimately cause osteoarthritis (OA), and the basic science in this area is evolving rapidly. When evaluating a potential risk factor or cause of a disease, the Bradford-Hill criteria are helpful for determining if an association between a risk factor and a disease is actually a cause-and-effect relationship [3]. These criteria consist of the following:

- Strength of association: This refers to the relationship between the possible cause and effect. If there is a stronger relative risk of developing a disease for a patient with a particular risk factor, the risk factor is more likely to be a causal factor. Occasionally, however, the observed association is slight, and the risk factor is nonetheless proven to be a cause of a disease.
- Consistency: This means that the same association is observed repeatedly, in studies that occur in different populations, with different study designs, and by different observers.
- Specificity: This describes how precisely a potential risk factor can predict that the disease will occur. It is important, however, to keep in mind that diseases may have more than one cause and that one-to-one relationships between a risk factor and a disease are infrequent.
- Temporality: This means that the proposed risk factor for the disease always precedes the disease.
- Dose–response effect: This means that the frequency of the disease increases with the dose

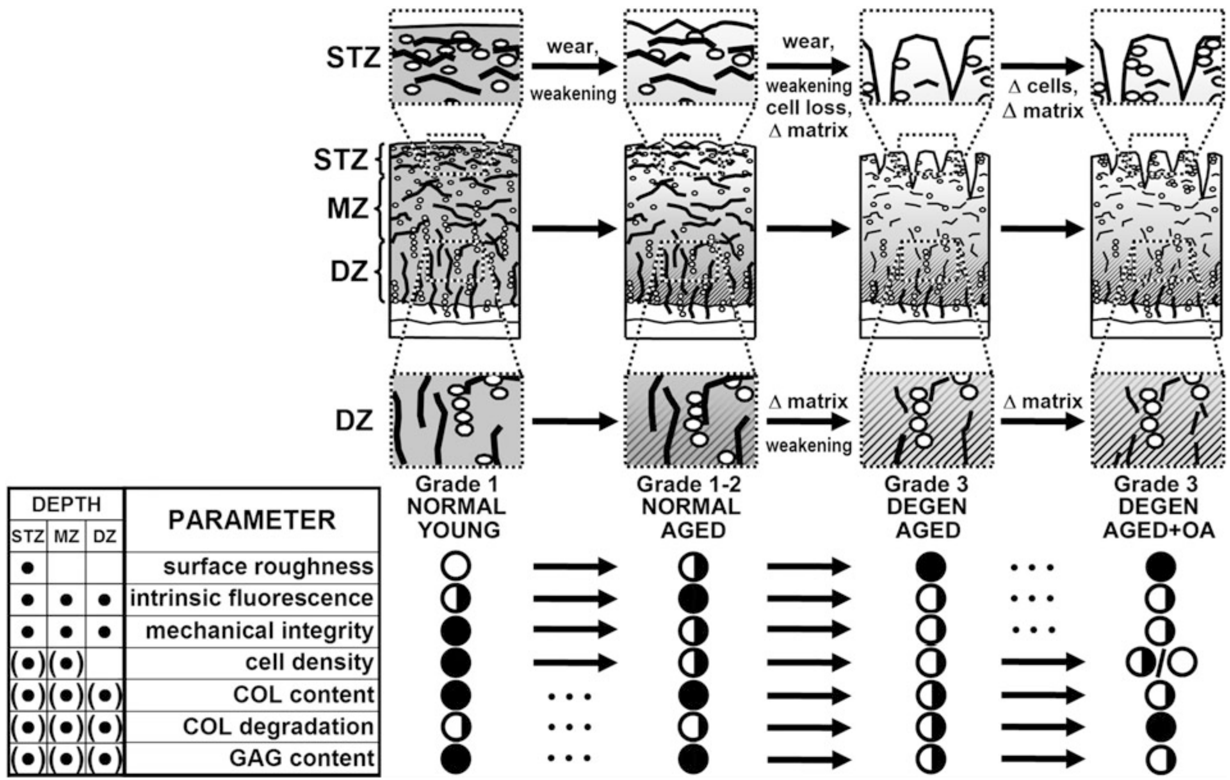


Fig. 1 Normal aged cartilage is biomechanically different from normal young cartilage. Over time, the surface (STZ: superficial tangential zone) becomes rougher and there are fewer chondrocytes. Degenerative aged and/or arthritic cartilage has deep surface clefts and fibrillation with chondrocyte clustering. In this diagram, the degree of *gray shading* indicates biomechanical integrity and loss of glycosaminoglycan (GAG). The table shows the location of changes in the STZ, middle zone (MZ), and deep zone (DZ). (•) indicates the full presence of changes, (o) the complete absence, with the *arrow* indicating a change between cartilage states (Reprinted from Osteoarthr Cartilage, Vol. 17, Temple-Wong MM, Bae WC, Chen MQ, Bugbee WD, Amiel D, Coutts RD, Lotz M, Sah RL. Biomechanical, structural, and biochemical indices of degenerative and osteoarthritic deterioration of adult human articular cartilage of the femoral condyle, 1469–1476, Copyright (2009), with permission from Elsevier)

or level of exposure. In orthopedics the dose or level exposure can also be the magnitude of a deformity.

- Biological plausibility: This means that, with what is currently known about biology or biomechanics, the proposed risk factor could reasonably cause the disease. Nonetheless, it is important to keep in mind that sometimes the basic science also needs to advance to elucidate the relationship between the cause and effect.
- Coherence: This means that the proposed association should not contradict current knowledge about the natural history and biology of the disease.
- Experimental evidence: This means that an experiment validates the cause-and-effect relationship in the expected manner. For example, modifying a risk factor decreases the frequency of a disease, or addressing the proposed cause of the disease brings about a cure.
- Analogy: This is the process of thinking about a proposed risk factor by comparing other similar and known cause-and-effect relationships for a particular disease. Reasoning by analogy can help to ascertain a cause-and-effect relationship when the observed association is slighter but similar to a known effect.

Hill himself cautioned, however, that these criteria are not necessary or sufficient for making a causal judgment and should be used more as guidelines for considering whether an observed risk factor truly causes a disease [3]. He also reminded the reader that "the 'cause' of illness may be immediate and direct, it may be remote and indirect underlying the observed association." Thus, returning to the question of the etiology of hip OA, it is more likely that the "cause" is multifactorial and different for different individuals. Furthermore, the Bradford-Hill criteria provide a useful framework for evaluating current hypotheses and evidence about the etiology of hip OA.

Although global prevalence of radiographic hip OA varies considerably, it is a common condition in the United States and Europe. The lifetime risk of developing symptomatic hip OA has been estimated to be as high as 25 % after adjusting for race, body mass index, sex, and prior injury [4]. However, not everyone who has radiographic evidence of hip OA becomes symptomatic. In one recent study, only 20 % of people with radiographic hip OA eventually became symptomatic enough to require total hip arthroplasty [5]. The natural history of asymptomatic radiographic hip OA is, however, difficult to elucidate because it requires long-term prospective cohort studies of large populations. Furthermore, the number of patients who progress to arthroplasty is likely to increase because many middle-aged and elderly patients expect to remain active indefinitely and would rather undergo arthroplasty than modify their activities. Age is one of the known risk factors for developing hip OA, and the incidence of hip OA increases with age. Not only does cartilage accumulate damage over time, but older mesenchymal stem cells also have less repair capacity and a decreased ability to protect cartilage from biomechanical stress [6]. Thus, as the expected human lifespan increases, the amount of hip OA and rates of hip arthroplasty are also projected to increase [7].

Other risk factors for hip OA include physical activity like long-term frequent lifting and standing [8] as well as intense or impact sports in young adulthood [9, 10]. There is an association between higher body mass index (BMI) and hip OA, although this association is much weaker than the association between BMI and knee OA [11]. Sex also appears to be a risk factor, with women having higher rates of hip OA than men [4]. Finally, family history and known congenital deformities have also been categorized as risk factors for hip OA.

Historically, hip OA was categorized as primary or idiopathic and secondary, meaning that the hip became arthritic as a result of a prior traumatic injury, pediatric deformity, or following infection. Primary or idiopathic hip OA was attributed to having "bad genes" – essentially that the patient had inherited weak cartilage. However, evidence is accumulating that primary hip OA is actually secondary to a subtle mechanical problem like femoroacetabular impingement (FAI) or mild dysplasia. The concept that hip OA is a mechanical process was proposed by a number of authors and summarized nicely by Ganz in 2008:

> Most, if not all, hip OA is secondary, often secondary to subtle but definite and commonly overlooked, ignored, or not recognized dysplasia or pistol grip deformities (FAI). [12]

The Genetics of Hip Osteoarthritis

While there is clearly an inheritance pattern to hip OA, the nature of the genetic contribution is not entirely known. Have family members with a history of hip OA all inherited a bone structure like dysplasia or FAI that causes chondral damage and subsequent OA, or have they simply inherited less rigorous cartilage that is more likely to be damaged in the setting of subtle FAI or dysplasia? Or, as seems likely, is it some combination of the two factors?

Twin studies done in Caucasian females found a genetic contribution of about 60 % for both center-edge angle (as a measure of acetabular depth) and radiographic hip OA [13]. The magnitude of the genetic contribution is not the same for other joints, meaning that the etiology of OA is likely specific to mechanical factors and anatomy at that joint. This also implicates morphology rather than poor-quality cartilage as the bigger risk factor for hip OA. Other studies have shown that femoral head shape is heritable in families with a history of arthroplasty for "idiopathic" OA. However, in one of these studies, patients with a positive family history were more symptomatic than patients with the same degree of FAI morphology but no family history of hip OA. This suggests that bony morphology may not be entirely responsible for symptom development [14]. Genes have been identified that are associated with both cartilage thickness and hip shape [15, 16]. These genes are expressed in developing limb buds and in developing cartilage as well as being expressed in response to increased biomechanical loads [15, 16]. Thus, the genes associated with hip OA could affect either the hip shape or the cartilage microstructure. Finally, genetic variability influences the association between hip OA and bony morphology, meaning that certain genotypes appear to make the cartilage more vulnerable to mechanical overloading from subtle FAI or dysplasia [15].

Acetabular Dysplasia

Acetabular dysplasia is defined as a shallow or small acetabulum that inadequately covers the femoral head. Moderate to severe acetabular dysplasia has long been recognized as a risk factor for the early development of hip OA [17]. The risk of developing hip OA due to mild or borderline dysplasia is less clear, however, and may be influenced by external factors like soft tissue laxity, femoral version, and sport or dance activities.

Although dysplasia has historically been thought of in the context of infantile hip subluxation or dislocation, there is growing recognition that adolescent- or adult-onset dysplasia may represent a developmental process distinct from infantile dysplasia [18]. Furthermore, very few younger adults undergoing hip arthroplasty for arthritis secondary to dysplasia are identified as neonates [19]. The demographics of the infant and adolescent dysplasia populations are different, with adolescent-onset dysplasia patients having more bilateral hip involvement, a stronger family history, and a higher proportion of male patients [18]. Infantile dysplasia may represent a "packaging problem," meaning that mechanical factors play a greater role in the shape of the neonatal acetabulum and containment of the femoral head. The risk factors for infantile dysplasia – breech positioning, left-sided laterality, and first-born females – implicate the intrauterine environment as a mechanical factor influencing acetabular development. Furthermore, the historical prevalence of dysplasia was substantially higher in populations that had a tradition of infant swaddling with the legs in extension. When this connection was recognized and parents were instructed not to swaddle their children, the incidence of dysplasia decreased [20].

The prevalence of acetabular dysplasia varies widely [20]. It is somewhat difficult to compare the prevalence of dysplasia between countries or regions because some studies have evaluated adults whereas for some populations the data are only available for infants. In addition, some studies have defined dysplasia as a center-edge angle of <25° whereas others have used a center-edge angle of <20°. Nonetheless, it is well recognized that the prevalence of dysplasia is higher in Asia and is the most common cause of hip OA in Japan [21].

A family history of dysplasia is a known risk factor for dysplasia and is consistent across all studied populations. Dysplasia is even more prevalent in areas where consanguinity (e.g., marriage between first cousins) is common [20]. Twin studies have revealed that the heritability of dysplasia is likely polygenic, with a higher incidence of dysplasia in monozygotic twins as compared to dizygotic twins. These findings have led investigators to propose that the genetic mechanism involves inheritance of excessive soft tissue laxity as well as acetabular shape [20].

Biomechanics of Dysplasia

In normal hips, the peak cartilage contact pressure when standing is located near the acetabular dome. The peak contact site varies between the lateral edge and the superior dome of the acetabulum, becoming more medial if the acetabulum is deeper and more lateral if the acetabulum is shallow [22, 23]. There is a direct relationship between the degree of acetabular coverage (as measured using the center-edge angle) and the contact area of the acetabular surface. As the contact surface area decreases, the peak contact pressure increases – meaning that a small center-edge angle is a marker for higher contact pressure [22]. This translates to increased force on the acetabular rim, particularly in stance, and causes characteristic chondrolabral pathology, including labral tears, ganglia, and, in some cases, acetabular rim fractures [24]. The tissue loss predictably occurs at the superior and anterosuperior regions of the acetabulum [24], which corresponds to the area of the highest load [22]. Acetabular version also influences contact pressures. Highly anteverted dysplastic hips have higher anterior contact stresses as a result of minimal anterior femoral head coverage [25]. In contrast, patients with dysplasia and retroversion have impingement-type contact stresses at the anterior edge of the acetabulum. Correcting the version and coverage with an acetabular reorientation osteotomy has been shown to decrease contact pressure by up to 50 % [23].

Natural History of Dysplasia

Radiographic dysplasia, variably defined as a center-edge angle of <20° or <25°, is clearly associated with an increased risk of hip OA [17]. The risk of developing hip OA is also clearly related to the grade of dysplasia, indicating that hips with worse biomechanics and higher contact pressures have a higher likelihood of sustaining joint damage and ultimately becoming arthritic (Fig. 2) [17]. If hip pain in a young person (<40) is considered to be a precursor of hip OA, it is notable that 25–35 % of young patients with hip pain have dysplasia [26]. Version may also play a role in the natural history of dysplasia. Patients who have retroversion and dysplasia experience an earlier onset of hip pain as compared to patients with normal anteversion [27].

If the loading biomechanics of a dysplastic hip are changed as a result of a femoral or acetabular osteotomy, the natural history of that hip appears to improve. The results are better for periacetabular or rotational acetabular osteotomy than for femoral osteotomy however. The long-term (20-year) survival rate of the native hip after a periacetabular osteotomy is about 60 % [28]. Even with the improvement in hip biomechanics, most patients have some progression of osteoarthritis and, on average, advance one radiographic Tönnis grade after 10 years [28]. Because dysplasia is largely a problem related to static loading across the hip, one might expect that weight loss in an overweight patient with dysplasia could improve hip pain and natural history because it decreases the overall static load. Although weight loss is known to improve pain and function in patients with knee OA, this has not been studied for patients with dysplasia. The

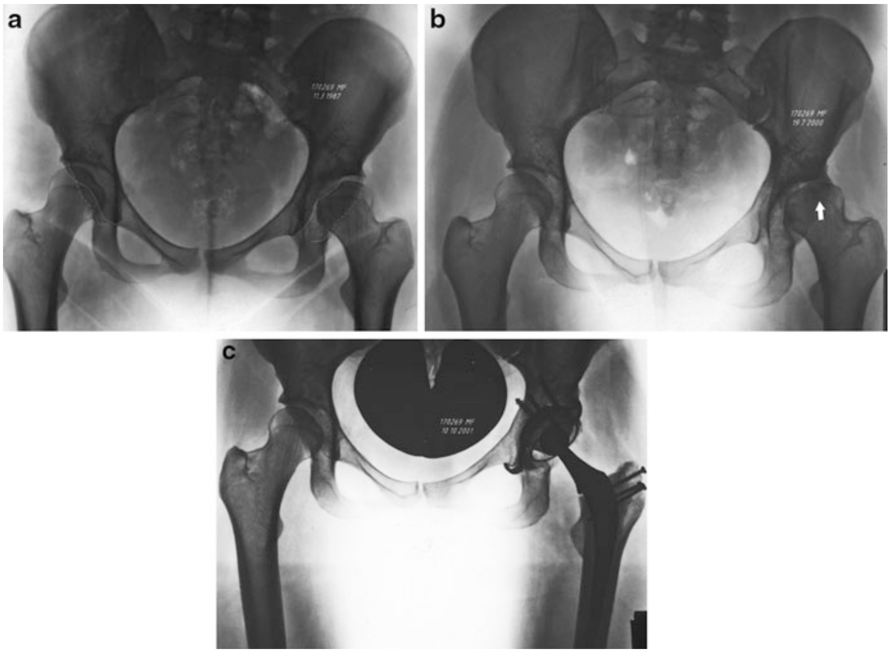


Fig. 2 The natural history of a patient with severe *left* anterior dysplasia (outline). At the time of presentation, the hip had already subluxed laterally, but the joint space was relatively preserved (**a**). Thirteen years later, there was advanced and severe cartilage degeneration on the femur, with a subchondral cyst and sclerosis (*arrow*) in the femoral head, as well as on the acetabulum (**b**). She underwent a total hip arthroplasty 1 year later (**c**)

potential for an osteotomy to improve hip function does have some limits. The success rates of osteotomy are poor for patients older than 35 with Tönnis grade 2 or more radiographic hip OA [28]. Thus, there appears to be a "tipping point" of cartilage damage, after which an osteotomy is unlikely to improve the natural history of a dysplastic hip.

Femoroacetabular Impingement (FAI)

Broadly speaking, FAI is defined as the abnormal contact between the femur and the acetabulum during hip motion that occurs as a result of a subtle deformity at the femoral head-neck junction or acetabular rim and that causes progressive chondrolabral damage [29]. Although Reinhold Ganz generally receives credit for describing FAI [29], the earliest description of impingement appears to have been in 1899 in the French literature, with the author noting "empreinte iliaque," or an impression on the head-neck junction produced by the ilium at the area of the anterior-inferior iliac spine with the hip in flexion [30]. Subsequent authors correctly described impingement in the context of coxa vara, severe protrusio deformities, and slipped capital femoral epiphysis [31–33]. Depending on the site of the deformity, these authors also recommended femoral neck osteoplasty and/or acetabular rim trimming to restore range of motion and provide pain relief. In contrast to all of the previous authors however, Ganz substantiated his ideas about FAI with observations of chondrolabral damage at the site of the impinging lesions and with the results of treatment [29, 34], both of which were made possible with the development of the technique for a safe surgical dislocation of the hip. The description of FAI also coincided with technical improvements in hip arthroscopy that resulted in an increase in hip arthroscopy for labral tears. As a result, arthroscopists began to recognize and describe early chondrolabral damage, which ultimately helped to substantiate the association between hip pain, impingement anatomy, and eventual hip OA [35].

FAI is broadly grouped into cam and pincer types of impingement, which have different mechanisms of damage and different prognoses for the cartilage. Considering the mechanical type of impingement injury is a useful way to think about FAI because it allows for the realization that different anatomic abnormalities can cause the same type of impingement. Although cam impingement can result from many distinct anatomic abnormalities, it ultimately causes an inclusion type of injury where a bony deformity at the head-neck junction enters the joint with hip flexion. Most commonly, the abnormality is a "cam deformity" which occurs as a result of an extension of the physis onto the femoral neck [36] causing either decreased head-neck offset or a prominence at the head-neck junction. However, the femoral head deformities that occur after Legg-Calve-Perthes' disease and mild or moderate slipped capital femoral epiphysis also cause cam-type impingement and can be considered extreme examples of cam impingement [33, 37]. In cam impingement, the deformity at the head-neck junction causes shear stress and delamination of the acetabular cartilage with separation at the chondrolabral junction [29, 34]. This type of impingement has a worse prognosis for the cartilage and can cause end-stage arthrosis in a relatively young (40–50-year-old) adult. Although the cartilage over the non-spherical portion of the femoral head is abnormal, with histologic changes like cell clustering and surface fibrillation that are consistent with early arthritis [38], the macroscopic chondral damage occurs initially on the acetabulum. The weight-bearing cartilage on the femoral head remains relatively preserved until the acetabular chondral defect advances to the point that the femoral head migrates into the defect. At this time, the chondral damage becomes radiographically apparent, with visible joint space narrowing on x-rays. Pincer impingement, in contrast, causes an impaction type of injury with hip flexion, with the acetabular rim contacting the femoral head, neck, or metaphysis. Global acetabular overcoverage and focal acetabular overcoverage from acetabular retroversion are the two more classic causes of pincer impingement. However, a prominent anterior-inferior iliac spine can also cause rim impingement [39], as can acetabular protrusio [40] and a severe SCFE deformity [33]. The rim impaction causes a crushing injury to the labrum and a linear wear pattern of cartilage damage and, over time, causes rim ossification [29, 34, 41]. In addition, a "pincer groove" is often visible on the femoral neck. Although pincer impingement may not cause chondral damage as rapidly as cam impingement, the crushing injury to the labrum appears to be quite painful for the patient. A smaller number of patients with pincer impingement have femoral levering on the acetabular rim, causing contrecoup injury to the cartilage in the posterior acetabulum [29, 34]. Patients with true acetabular protrusio also develop medial cartilage thinning [40], which may be a result of increased medial contact pressure. While the distinction between cam and pincer FAI helps to explain the nature of the observed cartilage injuries, in practicality most patients with FAI have mixed cam and pincer morphotypes [29].

Biomechanics of FAI

Impingement can be observed directly during a surgical hip dislocation. Nonetheless, these observations have also been validated with finite element analysis of the cartilage contact forces during hip flexion. When the deformity at the head-neck junction is increased (by increasing the alpha angle), the non-spherical portion of the head intrudes into the acetabulum, causing increased cartilage stress on the anterior acetabulum at the site of the cam deformity. In a similar manner, increasing the amount of acetabular coverage (by increasing the center-edge angle) causes higher contact stresses at the acetabular rim and contact with the femoral neck.

Natural History of FAI

There is clearly some heritability for impingement-type anatomy, although the genetic influence may not be as strong for FAI as it is for dysplasia. Interestingly, cam morphology seems to be more heritable than pincer morphology. One sibling study observed a relative risk of 2.8 for inheriting cam-type anatomy and a relative risk of 2.0 for inheriting pincer morphology [14].

Although the evidence that FAI ultimately causes hip OA seems convincing, it is indirect (Fig. 3). Labral tears and FAI morphology are frequent in asymptomatic volunteers [42, 43]. What remains unknown about these populations is whether the subjects are asymptomatic because they are in an early stage of the disease process and have minimal chondrolabral damage or if not all FAI ultimately progresses to become symptomatic. All of the currently available natural history studies are level III or IV prognostic studies based on pelvic radiographs [44, 45]. The rates of radiographic progression for patients with FAI are quite variable, ranging from 18 % to 73 % over 10 years [44, 45]. However, FAI morphology was found in nearly all (96–99 %) hip arthroplasty patients <55 years old who were previously diagnosed with primary or idiopathic hip OA [45].

Studies of hip OA in athletes provide evidence that OA may result from a combination of FAI and abnormal loading or motion requirements. Compared to the general population, both male and female athletes have higher rates of hip OA. Contact sports and higher exposure to sports increase the risk of hip OA [9, 10]. A few studies have looked at the prevalence of hip OA in former professional dancers. Here the effect is less clear; one study showed an increased incidence of hip OA in former dancers [46], whereas a later study found no difference in rates of hip OA between dancers and the general population [47]. One potential reason for this may be that the range of motion and the amount of trained soft tissue laxity

Fig. 3 This patient presented with bilateral FAI from cam deformities and acetabular retroversion on the *right*. At the initial presentation, there was acetabular subchondral sclerosis, but no joint space narrowing (**a**). Fifteen years later he had complete joint space destruction in both hips (**b**). Twenty years after his initial presentation, there are extensive cystic changes and femoral head collapse in both hips (**c**)

required for dancers weed out patients with impingement morphology before they reach elite or professional levels.

In addition to higher rates of hip OA, high-level athletes also have a higher prevalence of FAI morphology compared to the general population. This was actually first observed in the 1970s but was described as a "tilt deformity" and attributed to a mild subclinical SCFE [48]. More recently, cam deformities were found in 78 % of US collegiate football players, and a radiographic crossover sign was observed in 61 % of these same athletes [49]. Both professional and adolescent soccer players had high rates of FAI-type anatomy, with 72 % of the male professional players and 50 % of the females having some radiographic finding consistent with FAI [50]. Among asymptomatic professional and collegiate hockey players, 39 % had an elevated alpha angle but 77 % had hip and groin abnormalities on MRI [51]. Finally, a study of elite-level basketball players found that 89 % had an elevated alpha angle [52]. Cam deformities appear to occur from an extension of the femoral physis onto the femoral neck. Thus, one cause of high rates of FAI and cam deformities specifically in athletes may be the frequent high-intensity sporting activity itself. High-intensity sports have been shown to affect the proximal humeral physis and glenoid version in the young thrower as well as the distal radial physis in the gymnast. Similarly, repetitive rotational stress across the hip and proximal femoral physis as it is closing may cause the high rates of cam deformity seen in these athletes.

Summary and Conclusions

End-stage hip OA is caused by a combination of a structural deformity (either dysplasia or FAI), wear caused by the motion or activity required from an individual's hip, the inherent "robustness" of the individual's cartilage, and the amount of inflammation that the individual experiences. It is clear that not all radiographic hip OA is equally symptomatic and that many, but not all, people with FAI or dysplasia ultimately develop hip OA. The understanding of the structural factors that contribute to hip OA is advancing rapidly. It also appears that identification and treatment of FAI and dysplasia appears to help symptoms that result from early chondrolabral damage. There is good evidence that changing the biomechanics of the dysplastic joint with an acetabular osteotomy changes the natural history of the disease. If this occurs before the cartilage damage has advanced, acetabular reorientation may prevent end-stage OA or at least considerably delay an eventual arthroplasty. For FAI, there is evidence that correcting a cam or pincer deformity improves the symptoms from early OA. Although it seems likely, it is not yet known if surgical treatment can change the natural history of FAI and prevent progression of hip OA. One caveat, however, is that causing further chondral damage with surgery or incomplete treatment of these structural factors, e.g., not recognizing dysplasia in a patient with a cam deformity, might not be helpful and may incite the cascade of OA. Thus, the correct diagnosis and meticulous care of the cartilage are important when treating these patients in an attempt to prevent or delay the onset of hip OA.

References

1. Steadman's medical dictionary. 26th ed. Baltimore: Williams &Wilkins; 1995. Arthritis; p. 149, arthrosis; p. 151; osteoarthritis; p. 1267.
2. Temple-Wong MM, Bae WC, Chen MQ, Bugbee WD, Amiel D, Coutts RD, et al. Biomechanical, structural, and biochemical indices of degenerative and osteoarthritic deterioration of adult human articular cartilage of the femoral condyle. Osteoarthr Cartil. 2009;17: 1469–76.
3. Hill AB. The environment and disease: Association or causation? Proc R Soc Med. 1965;58:295–300.
4. Murphy LB, Helmick CG, Schwartz TA, et al. One in four people may develop symptomatic hip osteoarthritis in his or her lifetime. Osteoarthritis Cartilage. 2010;18:1372–9.
5. Franklin J, Ingvarsson T, Englund M, Ingimarsson O, Robertsson O, Lohmander LS. Natural history of radiographic hip osteoarthritis: a retrospective cohort with 11–28 years of followup. Arthritis Care Res. 2011;63: 689–95.
6. Payne KA, Didiano DM, Chu CR. Donor sex and age influence the chondrogenic potential of human femoral bone marrow stem cells. Osteoarthritis Cartilage. 2010;18:705–13.

7. Kurtz S, Ong K, Lau E, Mowat F, Halpern M. Projections of primary and revision hip and knee arthroplasty in the United States from 2005 to 2030. J Bone Joint Surg Am. 2007;89-A:780–5.
8. Sulsky SI, Carlton L, Bochmann F, et al. Epidemiological evidence for work load as a risk factor for osteoarthritis of the hip: a systematic review. PLoS ONE. 2012;7:e31521.
9. Vingard E, Alfredsson L, Goldie I, Hogstedt C. Sports and osteoarthritis of the hip. An epidemiologic study. Am J Sports Med. 1993;21:195–200.
10. Vingard E, Alfredsson L, Malchau H. Osteoarthrosis of the hip in women and its relationship to physical load from sports activities. Am J Sports Med. 1998;26:78–82.
11. Wang Y, Wluka AE, Simpson JA, et al. Body weight at early and middle adulthood, weight gain and persistent overweight from early adulthood are predictors of the risk of total knee and hip replacement for osteoarthritis. Rheumatology. 2013;52:1033–41.
12. Ganz R, Leunig M, Leunig-Ganz K, Harris WH. The etiology of osteoarthritis of the hip: an integrated mechanical concept. Clin Orthop Relat Res. 2008; 466:264–72.
13. MacGregor AJ, Antoniades L, Matson M, Andrew T, Spector TD. The genetic contribution to radiographic hip osteoarthritis in women. Results of a classic twin study. Arthritis Rheum. 2000;43:2410–6.
14. Pollard TCB, Batra RN, Judge A, et al. The hereditary predisposition to hip osteoarthritis and its association with abnormal joint morphology. Osteoarthritis Cartilage. 2013;21:314–421.
15. Baker-LePain JC, Lynch JA, Parimi N, et al. Variant alleles of the Wnt antagonist FRZB are determinants of hip shape and modify the relationship between hip shape and osteoarthritis. Arthritis Rheum. 2012;64:1457–65.
16. Castaño Betancourt MC, Cailotto F, Kerkhof HJ, et al. Genome-wide association and functional studies identify the DOT1L gene to be involved in cartilage thickness and hip osteoarthritis. Proc Natl Acad Sci USA. 2012;109:8218–23.
17. Murphy SB, Ganz R, Müller ME. The prognosis in untreated dysplasia of the hip. A study of radiographic factors that predict outcome. J Bone Joint Surg Am. 1995;77-A:985–9.
18. Lee CB, Mata-Fink A, Millis MB, Kim YJ. Demographic differences in adolescent-diagnosed and adult-diagnosed acetabular dysplasia compared with infantile developmental dysplasia of the hip. J Pediatr Orthop. 2013;33:107–11.
19. Engsæter IO, Lie SA, Lehmann TG, Furnes O, Vollset SE, Engesæter LB. Neonatal hip instability and risk of total hip replacement in young adulthood. Acta Orthop. 2008;79:321–6.
20. Loder RT, Skopelja EN. Epidemiology and demographics of hip dysplasia. ISRN Orthop. 2011; Article ID 238607, p. 47.
21. Jingushi S, Ohfuji S, Sofue M, et al. Multiinstitutional epidemiological study regarding osteoarthritis of the hip in Japan. J Orthop Sci. 2010;15:626–31.
22. Chegini S, Beck M, Ferguson SJ. The effects of impingement and dysplasia on stress distributions in the hip joint during sitting and walking: a finite element analysis. J Orthop Res. 2009;27:195–201.
23. Zhao X, Chosa E, Totoribe K, Deng G. Effect of periacetabular osteotomy for acetabular dysplasia by three-dimensional finite element analysis. J Orthop Sci. 2010;15:632–40.
24. Klaue K, Durnin CW, Ganz R. The acetabular rim syndrome: a clinical presentation of dysplasia of the hip. J Bone Joint Surg Br. 1991;73-B:423–9.
25. Matej D, Iglic A, Kral-Iglic V. Hip contact stress during normal and staircase walking: the influence of acetabular anteversion angle and lateral coverage of the acetabulum. J Appl Biomech. 2008;24:88–93.
26. Nogier A, Bonin N, May O, et al. Descriptive epidemiology of mechanical hip pathology in adults under 50 years of age. Prospective series of 292 cases: clinical and radiological aspects and physiopathological review. Orthop Traumatol Surg Res. 2010;96S:S53–8.
27. Fujii M, Nakashima Y, Yamamoto T, et al. Acetabular retroversion in developmental dysplasia of the hip. J Bone Joint Surg Am. 2010;92:895–903.
28. Steppacher SD, Tannast M, Ganz R, Siebenrock KA. Mean 20-year follow-up of Bernese periacetabular osteotomy. Clin Orthop Relat Res. 2008;466: 1633–44.
29. Ganz R, Parvizi J, Beck M, Leunig M, Nötzli H, Siebenrock KA. Femoroacetabular impingement: a cause for osteoarthritis of the hip. Clin Orthop Relat Res. 2003;417:112–20.
30. Poirier P. Traité d'anatomie humaine, vol. 1. Embryologie (Prenant A.) Ostéologie et Arthologie [Treatise of human anatomy, vol. 1 embryology (Prenant A) osteology and arthrology] (in French) Paris: Masson; 1899. pp. 516–26.
31. von Mikulicz J. In: von Bergmann E, von Bruns P, von Mikulicz J, editors. Handbuch der praktischen Chirurgie, Bd IV, Chirurgie der Extremitäten. [Handbook of surgical practice, vol. 4, Extremity surgery.] (in German). Stuttgart: Enke; 1903. p. 600.
32. Smith-Petersen MN. Treatment of malum coxae senilis, old slipped upper femoral epiphysis, intrapelvic protrusion of the acetabulum, and coxa plana by means of acetabuloplasty. J Bone Joint Surg. 1936; 18:869–80.
33. Leunig M, Casillas MM, Hamlet M, et al. Slipped capital femoral epiphysis. Early mechanical damage to the acetabular cartilage by a prominent femoral metaphysis. Acta Orthop Scand. 2000;71:370–5.
34. Beck M, Kalhor M, Leunig M, Ganz R. Hip morphology influences the pattern of damage to the acetabular cartilage: femoroacetabular impingement as a cause of osteoarthritis of the hip. J Bone Joint Surg Br. 2005;87(B):1012–8.
35. Kelly BT, Weiland DE, Schenker ML, Philippon MJ. Arthroscopic labral repair in the hip: surgical technique and review of the literature. Arthroscopy. 2005;21:1496–504.

36. Kienle KP, Keck J, Werlen S, Kim YJ, Siebenrock KA, Mamisch TC. Femoral morphology and epiphyseal growth plate changes of the hip during maturation: MR assessments in a 1-year follow-up on a cross-sectional asymptomatic cohort in the age range of 9–17 years. Skeletal Radiol. 2012;41:1381–90.
37. Eijer H, Podeszwa DA, Ganz R, Leunig M. Evaluation and treatment of young adults with femoroacetabular impingement secondary to Perthes' disease. Hip Int. 2006;16:273–80.
38. Wagner S, Hofstetter W, Chiquet M, et al. Early osteoarthritic changes of human femoral head cartilage subsequent to femoro-acetabular impingement. Osteoarthritis Cartilage. 2003;11:508–18.
39. Hetsroni I, Larson CM, Dela Torre K, Zbeda RM, Magennis E, Kelly BT. Anterior inferior iliac spine deformity as an extra-articular source for hip impingement: a series of 10 patients treated with arthroscopic decompression. Arthroscopy. 2012;11:1644–53.
40. Leunig M, Nho SJ, Turchetto L, Ganz R. Protrusio acetabuli: new insights and experience with joint preservation. Clin Orthop Relat Res. 2009;467:2241–50.
41. Corten K, Ganz R, Chosa E, Leunig M. Bone apposition of the acetabular rim in deep hips: a distinct finding of global pincer impingement. J Bone Joint Surg Am. 2011;93 Suppl 2:10–6.
42. Reichenbach S, Jüni P, Werlen S, et al. Prevalence of cam-type deformity on hip magnetic resonance imaging in young males. A cross-sectional study. Arthritis Care Res. 2010;62:1319–27.
43. Leunig M, Jüni P, Werlen S, et al. Prevalence of cam and pincer-type deformities on hip MRI in an asymptomatic young Swiss female population: a cross-sectional study. Osteoarthritis Cartilage. 2013;21:544–50.
44. Bardakos NV, Villar RN. Predictors of progression of osteoarthritis in femoroacetabular impingement: a radiological study with a minimum of ten years follow-up. J Bone Joint Surg Br. 2009;91:162–9.
45. Clohisy JC, Dobson MA, Robison JF, et al. Radiographic structural abnormalities associated with premature, natural hip-joint failure. J Bone Joint Surg Am. 2011;93 Suppl 2:3–9.
46. Andersson S, Nilsson B, Hessell T, et al. Degenerative joint disease in ballet dancers. Clin Orthop Relat Res. 1989;236:233–6.
47. van Dijk CN, Lim LSL, Poortman A, Strübbe EH, Marti RK. Degenerative joint disease in female ballet dancers. Am J Sports Med. 1995;23:295–300.
48. Murray RO, Duncan C. Athletic activity in adolescence as an etiological factor in degenerative hip disease. J Bone Joint Surg Br. 1971;53-B:406–19.
49. Kapron AL, Anderson AE, Aoki SK, et al. Radiographic prevalence of femoroacetabular impingement in collegiate football players. AAOS Exhibit Selection. J Bone Joint Surg Am. 2011;93:e111.
50. Gerhardt MB, Romero AA, Silvers HJ, Harris DJ, Watanabe D, Mandelbaum B. The prevalence of radiographic hip abnormalities in elite soccer players. Am J Sports Med. 2012;40:584–8.
51. Silvis ML, Mosher TJ, Smetana BS, et al. High prevalence of pelvic and hip magnetic resonance imaging findings in asymptomatic collegiate and professional hockey players. Am J Sports Med. 2011;39:715–21.
52. Siebenrock KA, Ferner F, Noble PC, Santore RF, Werlen S, Mamisch TC. The cam-type deformity of the proximal femur arises in childhood in response to vigorous sporting activity. Clin Orthop Relat Res. 2011;469:3229–40.

10 Layered Concept of the Hip and Pelvis

Alexander E. Weber, James R. Ross, Bryan T. Kelly, and Asheesh Bedi

Contents

A.E. Weber • J.R. Ross
Sports Medicine and Shoulder Service, Department of Orthopaedic Surgery, University of Michigan, Ann Arbor, MI, USA
e-mail: alexweb@med.umich.edu; orthodocjimross@gmail.com

B.T. Kelly
Department of Orthopaedic Surgery, Center for Hip Preservation Surgery, Hospital for Special Surgery, New York, NY, USA
e-mail: info@bryankellymd.com

A. Bedi (✉)
Department of Orthopaedic Surgery, University of Michigan, Ann Arbor, MI, USA
e-mail: abedi@umich.edu

Abstract

Providing appropriate care to young patients and athletes with hip pain is predicated upon a thorough and comprehensive diagnostic evaluation. A comprehensive approach to the evaluation of such a patient is paramount to successfully defining the source of the patient's symptoms and ultimately rendering appropriate treatment. Compartmentalization of the normal hip anatomy into layers from deep to superficial allows for a comprehensive and systematic evaluation and identification of all potential pain generators in patients with either hip or groin pain. This chapter will discuss a layered approach to the evaluation of the painful groin in a manner that allows the clinician to narrow the differential diagnosis of "hip" pain. The four layers are (1) the osteochondral layer, (2) the inert layer, (3) the contractile layer, and (4) the neurokinetic layer. The normal anatomy of each layer will be reviewed with attention paid to structure and function. The most common pathologic conditions affecting the young patient with hip pain will also be reviewed, with an emphasis on the interaction between the layers.

Introduction

Compartmentalization of the normal hip anatomy into layers from deep to superficial allows for a comprehensive and systematic evaluation and

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_10

identification of all potential pain generators in patients with either hip or groin pain (Table 1). A layered approach to the evaluation of hip pathologic entities can allow the clinician to narrow the differential diagnosis of hip pain (Table 2). The comprehensive history and physical examination that is performed as part of this approach will ultimately allow the physician to develop the most appropriate treatment plan for each individual patient.

Layer 1: Osteochondral Layer

This deep layer of the hip comprises the osseous and chondral structures, which function to provide joint congruence and osteoarticular kinematics in the normal hip. The bony structures that encompass this layer include the pelvis, acetabulum, and femur. Structural pathologies exist within this layer and can be classified into three distinct groups: (1) static overload, (2) dynamic impingement, and (3) dynamic instability [1–8]. Numerous anatomic variations may result in static overload, including lateral or anterior acetabular undercoverage (acetabular dysplasia), acetabular protrusio, excessive femoral anteversion, relative femoral retroversion, excessive acetabular retroversion/excessive anteversion, and coxa vara/valga. These anatomic variants change the structural mechanics and may predispose the articular cartilage to eccentric loading, with subsequent abnormal or increased stress and asymmetric loads between the femoral head and acetabular socket in the axially loaded, standing position.

During terminal hip motion, hip pain may present due to abnormal stress and contact between the femoral head and acetabular rim. Structural variants within layer 1 that may contribute to such dynamic impingement include femoroacetabular impingement (FAI; cam-type, pincer-type, or combined), relative femoral retroversion, and coxa vara. When the required range of motion for competition in sports or for daily activities exceeds the limits of an individual's physiologic motion imparted by the anatomic structures of the hip, a compensatory increase in motion may be provided through layer 1. Specifically, increased motion and consequential stresses through the pubic symphysis, sacroiliac (SI) joints, and lumbar spine may occur. Compensatory injuries in the form of lumbar spondylolysis, osteitis pubis, and SI joint strain have been well described in the setting of primary FAI. When functional range of motion requirements exceed the individual's normal motion limits, forceful anterior contact occurring at the end range of internal rotation may also lead to dynamic instability in the form of subtle repetitive posterior hip subluxations as the femoral head levers out of the hip socket [1, 9]. An appropriately aligned anteroposterior (AP) pelvic radiograph in addition to a lateral view of the proximal femur (modified Dunn or frog-leg lateral) and/or a false profile view allows the treating physician to systematically assess the bony anatomy of the acetabulum and proximal femur. Various described radiographic indices such as Tönnis osteoarthritis grade, minimum joint space, lateral center-edge angle, anterior center-edge angle, Tönnis angle (acetabular inclination), alpha and beta angles, and presence/absence of crossover, posterior wall, and prominent ischial spine signs may better delineate the bony anatomy and thus facilitate a correct mechanical diagnosis [10–16].

Topographic Bony Anatomy

The evaluation of the osteochondral layer of the young adult with hip pain begins superficially with the bony landmarks about the hip. The principal bony landmarks that are identified include the pubic symphysis, anterior superior iliac spine (ASIS), iliac crest, posterior iliac spine, greater trochanter, and ischium (Fig. 1). These landmarks serve as reference points for the deeper soft-tissue structures during the physical examination. The greater trochanter and the ASIS also have been described as the two key landmarks for accurately establishing portals during hip arthroscopy should surgical treatment be warranted.

Table 1 Overview of the layered approach to the anatomy and pathology of the hip

Layer	Name	Structure	Purpose	Pathology	
I	Osteochondral	Innominate	Joint congruity	*Developmental*	*Dynamic*
		Acetabulum	Joint kinematics/ biomechanics	Dysplasia	Cam/Rim impingement
		Femur		Acetabular Profunda/protrusio	Trochanteric impingement
					Sub-spine impingement
				Femoral/acetabular version	Delamination
				Femoral inclination	
II	Inert	Capsule	Static stability	Capsular instability	
		Labrum		Labral tear	
		Ligamentous complex		Ligamentum teres tear	
		Ligamentum teres		Adhesive capsulitis	
III	Contractile	Peri-articular Musculature	Dynamic stability	Hemi-pelvic pubalgia	
		Lumbosacral Musculature		Hip flexor strain	
		Pelvic Floor		Anterior enthesiopathy	
				Psoas impingement	
				Rectus femoris impingement	
				Medial enthesiopathy	
				Adductor tendinopathy	
				Rectus abdominus	
				Enthesiopathy	
				Posterior enthesiopathy	
				Hamstring strain	
				Lateral enthesiopathy	
				Peri-trochanteric disorders	
				Gluteus medius tear	
IV	Neuromechanical	Neurovasculature of the pelvic girdle and hip	Biofeedback	*Neural*	*Mechanical*
			Timing and sequencing of kinematic chain	Nerve entrapment	Scoliosis
				Referred spinal	Ambulation/ foot structure and mechanics
		Mechanoreceptors	Perfusion	Pathology	Pelvic posture
		Thoraco-lumbar and lower extremity mechanics		Neuromuscular dysfunction	Osteitis pubis
				Regional pain syndromes	Sacroiliac dysfunction
					Pubic symphysis dysfunction

Adapted from Draovitch et al. [72]. Copyright Springer

Table 2 Differential diagnosis of hip pain

Traumatic injury	Fracture or stress fracture
	Hip dislocation
	Soft tissue contusion or hematoma
Labral injury	Trauma
	Femoroacetabular impingement
	Hip joint hypermobility
	Dysplasia
Cartilage Injury	Lateral impaction
	Loose body
	Condral shear
	Osteoarthrosis
	Osteonecrosis
Capsule pathology	Capsular laxity
	Adhesive capsulitis
	Capsular inflammation/synovitis
Synovial proliferative disorders	Pigmented villonodular synovitis
	Synovial chondromatosis
	Chondrocalcinosis
Inflammatory conditions	Rheumatoid arthritis
	Reiter syndrome
	Psoriatic arthritis
Infection	Septic arthrosis
	Osteomyelitis
Tumor	Benign soft tissue neoplasm
	Benign osseous neoplasm
	Malignant soft tissue neoplasm
	Malignant osseous neoplasm
	Metastatic disease
Metabolic disorders	Paget's disease
	Primary hyperparathyroidism
Extra-articular musculoskeletal disorders	Coxa saltans
	Trochanteric bursitis
	Athletic pubalgia
	Abductor impingement
	Psoas impingement
	Ischial bursitis
	Osteitis pubis
	Tendonitis (flexors, abductors or adductors)
	Sacroiliac pathology
Non-musculoskeletal disorders	Inguinal hernia
	Spine pathology (myelopathy, radiculopathy) Iliopsoas muscle abscess
	Intra-abdominal pathology (endometriosis, ovarian cyst)
	Peripheral vascular disease
Unknown etiology	Transient osteoporosis of the hip
	Bone marrow edema syndrome

Osteology of the Hip Joint

Both the proximal femur and pelvis are preformed in cartilage. Three of the eight acetabular ossification centers ultimately fuse to create the osseous acetabulum. The iliac, ischial, and pubic ossification centers form the triradiate cartilage. The iliac ossification center is the first to appear at approximately 9 weeks of intrauterine development, while the ischial and pubic centers appear at 16 and 20 months, respectively. At birth, the acetabulum remains a cartilaginous structure, and it is not until 8–9 years of life that the acetabulum transitions from a cartilaginous to full osseous structure. Fusion of the cartilage centers reaches completion around 16–18 years of life [17, 18].

The two ossification centers of the proximal femur (the femoral epiphysis and the trochanteric apophysis) are not present during prenatal life; however, they both become apparent during the first year of life. The femoral epiphysis ossification center is offset laterally within the femoral head. In early development, the AP diameter is greater than the transverse dimension. At the age of 3 years, the AP and transverse dimensions are equivalent. After 3 years of age, the femoral head becomes ovoid with the transverse diameter becoming larger than the AP diameter [19, 20].

The developmental osteology of the hip and acetabulum has been studied from early postnatal life and into adulthood, and these changes have implications on the degree of femoral head coverage by the acetabulum. Horii et al. [21] demonstrated the difference in the degree of femoral head coverage between children, adolescents, and adults, suggesting that global osseous acetabular coverage of the femoral head is greater in adults than in children and adolescents. However, when taking into account the acetabular labrum, the total femoral head coverage is greater in children than in adults [21].

An understanding of the normal anatomic positions of the acetabulum and femoral head is also important. The acetabular articular surface, or lunate fossa, is horseshoe-shaped, with the central-inferior portion devoid of articular cartilage. This bare area is the location of the acetabular attachment of the ligamentum teres, which is

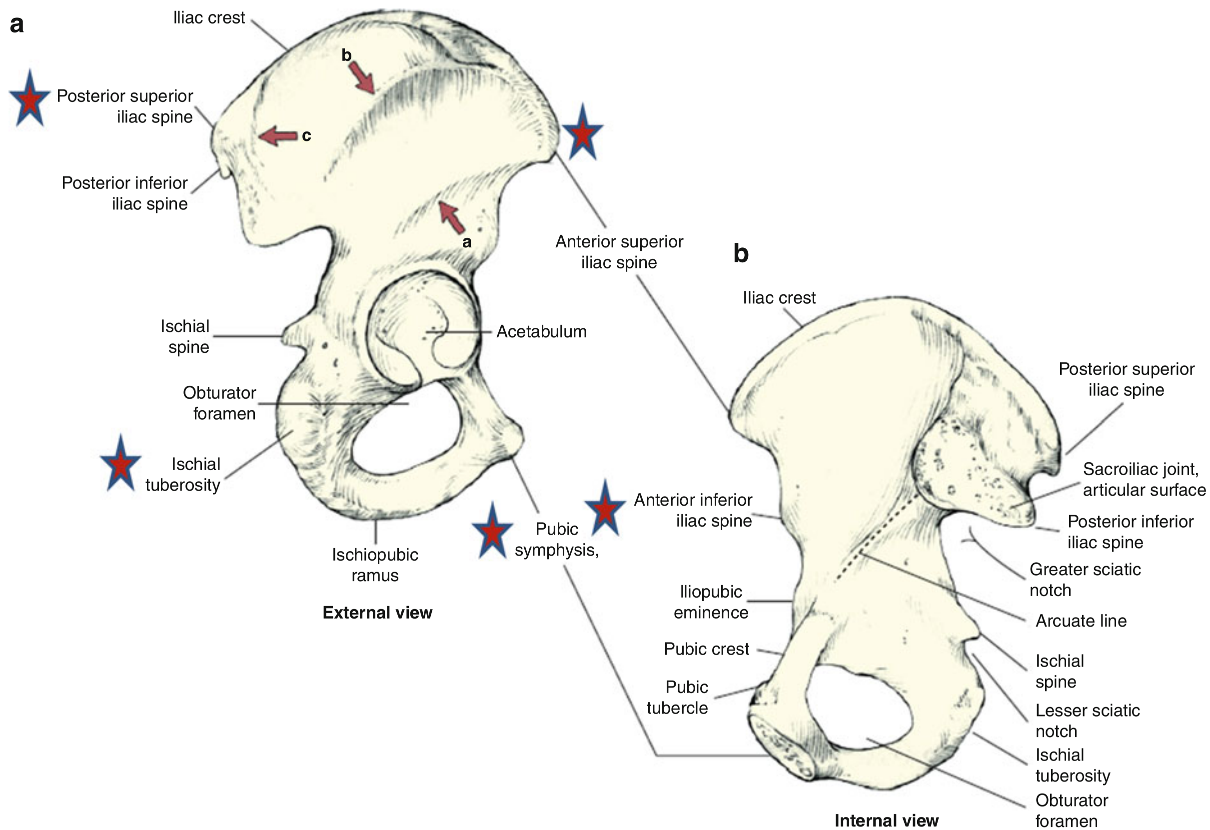


Fig. 1 Bony landmarks (marked with stars) which may be palpated during the examination of the hip to provide frame of reference include anterior superior iliac spine, pubic symphysis, iliac crest, posterior iliac spine, and ischial tuberosity (From Mathers LH, et al. Clinical Anatomy: Principles. St. Louis: Mosby; 1996. Copyright Elsevier)

surrounded by a synovial fat pad, also known as the pulvinar. The horseshoe-shaped acetabulum is completed by the transverse acetabular ligament inferiorly, which is continuous with the labrum (Fig. 2) [17].

The proximal femoral anatomy can be quite variable; however the neck-shaft angle averages 125° with approximately 14° of femoral neck anteversion [22]. The femoral head articular cartilage is often described as covering the equivalent of two-thirds of a sphere. The cartilage is congruous except for a shallow depression in the inferomedial aspect of the head. This small area devoid of cartilage is known as the fovea capitis and anatomically serves as the femoral attachment of the ligamentum teres [17].

Deviations from the normal development and morphologic relationships of the bony acetabulum and proximal femur may result in a mechanical conflict within the hip joint. This conflict can span a continuum from hip joint "undercoverage" (dysplasia) to hip joint "overcoverage" (femoroacetabular impingement) [1, 4, 5]. The specific pathology that results in a femoral and acetabular mismatch can be further differentiated by its primary factor, either static or dynamic [1].

Static mechanical factors may result in abnormal stress within the hip joint and asymmetric load across the hip joint either in the standing or axially loaded position. Bony pathologies most often associated with static mechanical stress are lateral acetabular undercoverage, anterior acetabular undercoverage, and excessive femoral anteversion and femoral valgus. The mechanical stresses in the aforementioned conditions may lead to asymmetric loading and thus premature wear of the chondral surfaces of the acetabulum and femur [1, 23–25]. This primary bone and cartilage mechanical stress may lead to compensatory muscular injury/overload (abductors, iliopsoas, and adductors) as the periarticular musculature attempts to stabilize the mismatched anatomy of the hip joint and subsequently affect the superficial layer 3, which will be discussed later in this chapter.

In the setting of dynamic mechanical disorders, range of motion of the hip causes contact between the femoral head and acetabular rim. The bony abnormalities most often associated with dynamic mechanical overload are a cam-type or pincer-type (focal, global retroversion, profunda, protrusio) deformity, relative femoral retroversion, or coxa vara. The mechanical stresses of the above pathologic conditions primarily result in hip and groin pain. When the athletic demands on the hip are greater than the functional range of motion, compensatory stresses and subsequent pain may develop in the lumbar spine, pubic symphysis, sacroiliac joints, and posterior acetabulum [1]. This compensatory stress also places demands on the periarticular musculature, which may also lead to muscle injury of the adductor longus, proximal hamstrings, abductors, iliopsoas, and hip flexors (layer 3) [26].

Layer 2: Inert Layer

The resultant mechanical stresses of the previously described layer 1 may lead to reactive hip pain related to insufficient congruency or impingement between the femoral head and the acetabulum. Thus, the morphology of layer 1 has a direct effect on the inert layer, or layer 2, of the hip.

Layer 2 includes the acetabular labrum, joint capsule, ligamentous complex, and ligamentum teres. These structures are all contributors to the static stability of the hip joint. When abnormal mechanical stresses are applied to the hip joint secondary to underlying abnormalities within layer 1, the resultant pathology may present in layer 2. These pathologies include labral injury, ligamentum teres tears, capsular injury, and consequent instability or adhesive capsulitis. Magnetic imaging resonance (MRI), usually with an arthrogram, is the best imaging modality to help evaluate the chondral, labral, and capsular damage. With an understanding of the underlying structural mechanics of layer 1, in combination with knowledge of the activity-specific range of hip motion, one can predict the type of injury to layer 2 structures.

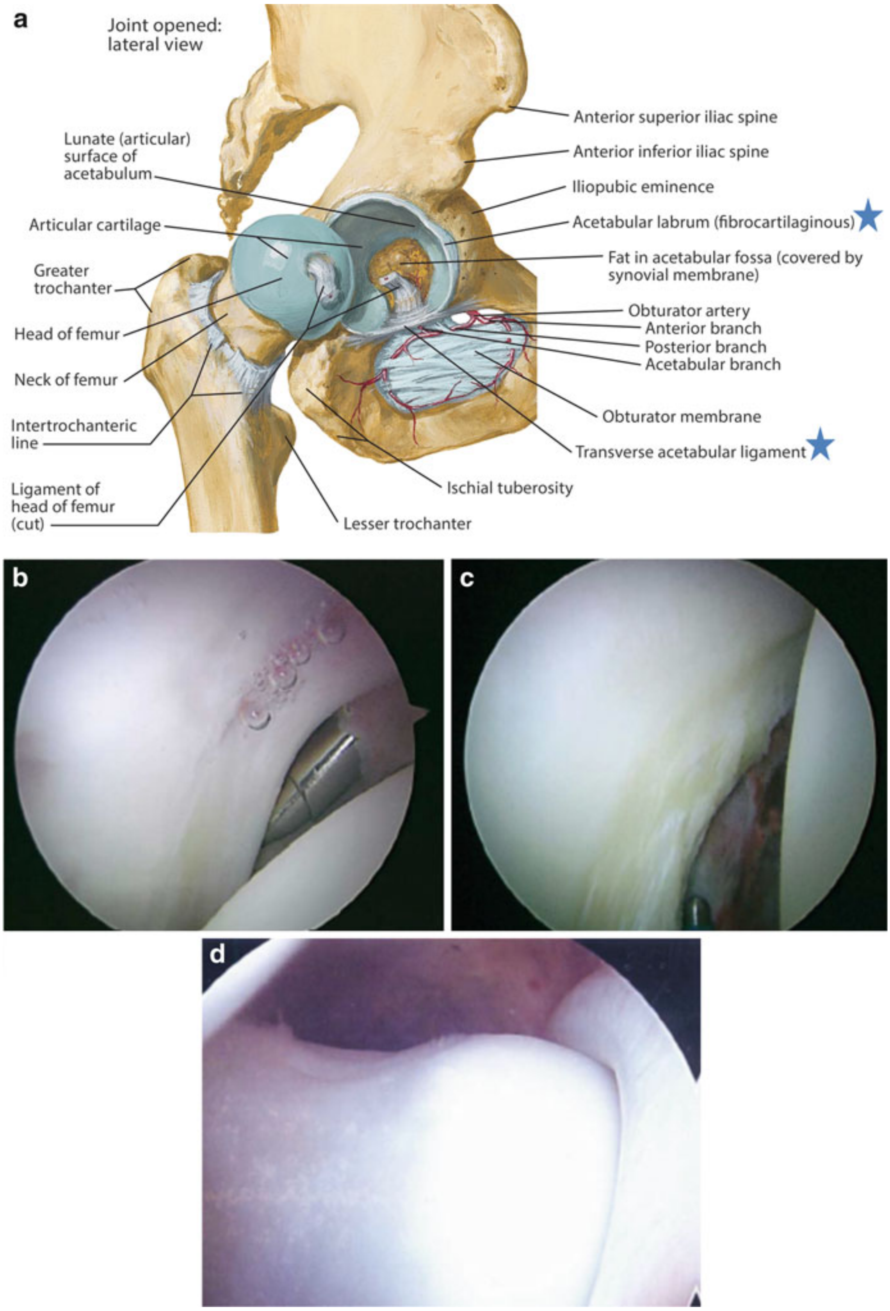


Fig. 2 (**a**) Illustration of the acetabulum and femoral head, with the hip dislocated to identify the acetabular labrum and transverse acetabular ligament (marked with stars). Netter illustration from www.netterimages.com. ©Elsevier Inc. All rights reserved. (**b**) Arthroscopic view of the anterior labrum from a posterolateral viewing portal. (**c**) Arthroscopic view of the posterior labrum from an anterolateral viewing portal. (**d**) Arthroscopic view of the "suction seal" effect of the labrum (**b**–**d**) (From Ranawat AS, Kelly BT. Anatomy of the hip: Open and arthroscopic structure and function. Oper Tech Orthop. 2005;15 (3):160–174. Copyright Elsevier)

Capsular Structure and Function

The capsular complex is mainly composed of three discrete thickenings. The three discrete bands of capsule are the iliofemoral, ischiofemoral, and pubofemoral ligaments (Fig. 3). These three ligaments each originate from one of the three named bones of the pelvis. Their function is to effectively encapsulate the hip joint from acetabulum to the intertrochanteric ridge. From the acetabular origins, the capsule extends laterally to surround the femoral head and neck and fans out to insert broadly on the proximal femur. The femoral insertions extend anteriorly onto the intertrochanteric line, superiorly onto the base of the femoral neck, posteriorly superomedial onto the intertrochanteric crest, and inferiorly onto the femoral neck [17].

The iliofemoral ligament (Y-ligament of Bigelow) has two arms, comprises the anterior portion of the capsule and derives its name from its appearance as an inverted "Y." From one common origin between the anterior-inferior iliac spine and the acetabular rim, it splits into a superior arm and an inferior arm as it crosses the joint [17, 27]. The superior arm travels horizontally and inserts proximally along the intertrochanteric line anterior to the hip joint. The inferior arm takes a vertical course and inserts caudally along the intertrochanteric line [28]. The iliofemoral ligament is the strongest and thickest of the three ligaments. Functionally, it provides restraint to anterior hip subluxation or dislocation, especially when the hip is in positions of extension and external rotation [17, 27]. In its contracted position, the iliofemoral ligament causes the hip to move into a flexed and internally rotated posture. Given its functionality, preservation or repair of the iliofemoral ligament at the conclusion of hip preservation surgery is preferred and may be especially relevant in patients with hyperlaxity, anterior instability, and/or acetabular undercoverage.

The ischiofemoral ligament comprises the majority of the posterior portion of the hip capsule. Generally, the ligament takes its origin from the ischial rim of the acetabulum and inserts

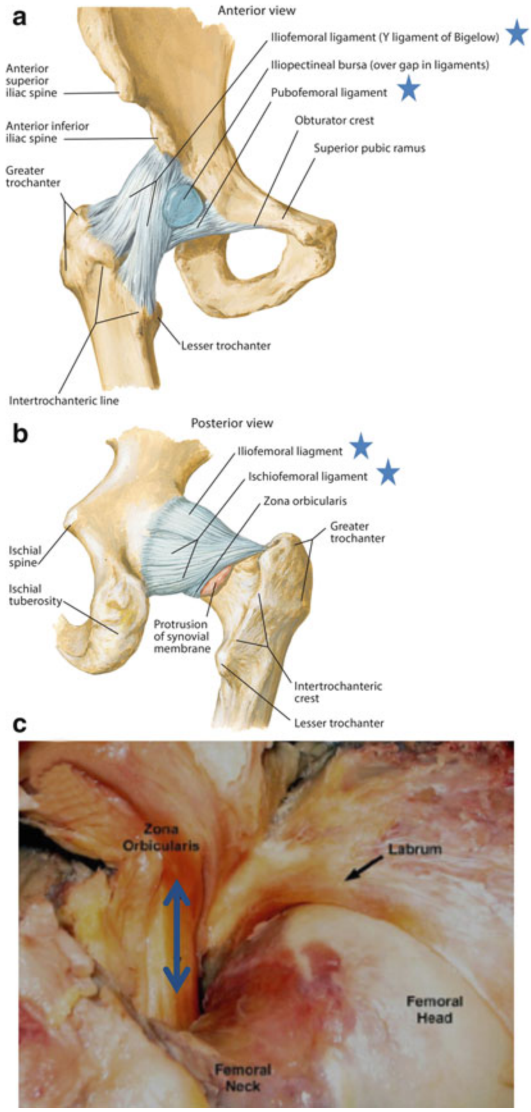


Fig. 3 Illustration of the (**a**) anterior capsular complex and (**b**) posterior capsular complex; iliofemoral ligament, ischiofemoral ligament, and pubofemoral ligament are marked with stars. Zona orbicularis present on the posterior view and denoted with *red underline*. Netter illustrations from www.netterimages.com. ©Elsevier Inc. All rights reserved. (**c**) Gross dissection of the capsular structures denoting the *inner surface* of the capsule and the *undersurface* of the zone orbicularis (marked with *blue arrow*) (From Bedi et al. Capsular management during hip arthroscopy: From femoroacetabular impingement to instability. Arthroscopy. 2011;27(12):1720–1731. Copyright Elsevier)

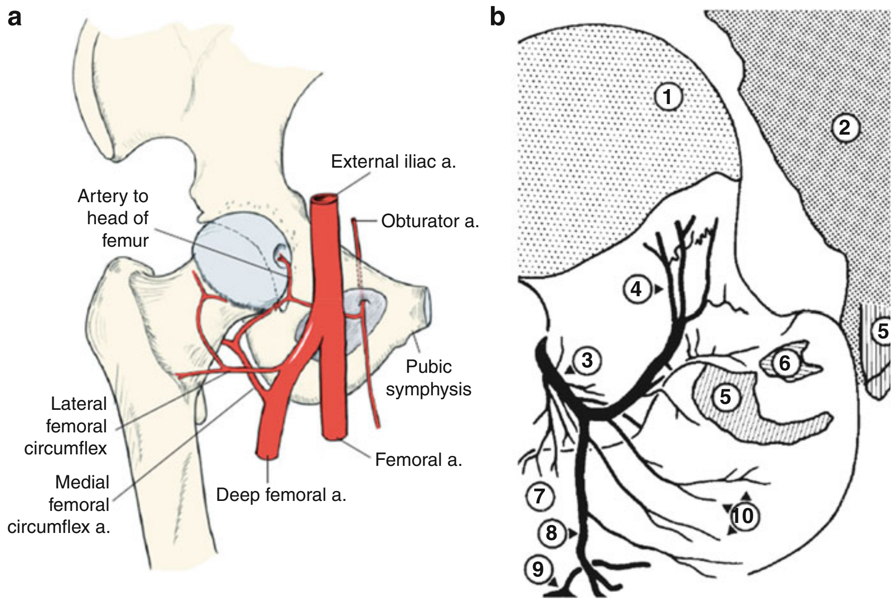


Fig. 4 (**a**) Illustration of the major blood vessels to the hip (From Mathers LH, et al. Clinical Anatomy: Principles. St. Louis: Mosby; 1996. Copyright Elsevier). (**b**) Detailed illustration of the terminal branches of the medial femoral circumflex artery (MFCA). (*1*) Femoral head, (*2*) gluteus medius, (*3*) deep branch of the MFCA, (*4*) the terminal subsynovial branches of the MFCA, (*5*) insertion of gluteus medius, (*6*) piriformis insertion, (*7*) nutrient vessels to lesser trochanter, (*8*) the trochanteric branch of MFCA, (*9*) the branch of the first perforating artery, and (*10*) the greater trochanteric branches (From Gautier et al. Anatomy of the medial femoral circumflex artery and its surgical implications. J Bone Joint Surg [Br]. 2000;82-B:679–683. Reproduced with permission and copyright © of the British Editorial Society of Bone and Joint Surgery)

around the posterior aspect of the femoral neck [27]. The ischiofemoral ligament also divides into two arms, with the more superior arm blending with the zona orbicularis fibers and the inferior arm inserting more posteriorly on the intertrochanteric crest [28]. In action, the main function of the ischiofemoral ligament is to resist internal rotation and adduction of the hip. In comparison to the iliofemoral ligament, the ischiofemoral ligament has less than half its ultimate load to failure [27–29].

The pubofemoral ligament takes its origin at the pubic portion of the acetabular rim and the obturator crest of the pubic bone before fanning out distally like in a sling-like shape to attach onto the femoral neck. Fibers of the pubofemoral ligament blend with the more medial arm of the iliofemoral ligament. As the ligament courses caudally, it inserts posteriorly on the femoral neck below the ischiofemoral ligament. In action, the pubofemoral ligament works in conjunction with the medial and lateral arms of the iliofemoral ligament, to control external rotation of the joint [17, 28].

In contrast to the longitudinally oriented fibers of the three constituents of the outer capsule (iliofemoral, ischiofemoral, and pubofemoral ligaments), the inner capsular fibers of the zona orbicularis run in a horizontal fashion as they circle the femoral neck (Fig. 3). This leash of capsule fibers acts like a locking ring around the femoral neck preventing femoral head distraction [30]. In a cadaveric study, Ito et al. [30] demonstrated that the zona orbicularis serves as the main hip stabilizer against distractive forces.

In addition to providing stability to the hip joint, the capsule provides protection to the articular cartilage and serves as the location for the blood supply to the acetabulum and femoral head. Four main blood vessels supply the hip capsule: the superior gluteal artery, the inferior gluteal artery, the medial femoral circumflex artery, and the lateral femoral circumflex artery (Fig. 4). The posterior hip capsule receives its major blood supply from the superior and inferior gluteal arteries as they descend from the pelvis. The anterior capsule is vascularized from the medial and lateral circumflex arteries as they ascend through the hip capsule [31]. In most cases, the medial femoral circumflex artery travels on to be the primary blood supply to the femoral head; however, a cadaveric study by Kalhor et al. [31] demonstrated the inferior gluteal artery to be the dominant blood supply to the femoral head in a minority of cases. In the majority of cases, the main blood supply to the femoral head originates distally at the capsular insertion, and thus overzealous dissection of the capsule either for exposure or instrumentation during hip procedures may lead to iatrogenic devascularization of the femoral head. Therefore, in cases where the capsule must be opened, either via capsulotomy or capsulectomy, the T-capsular split should be made between the medial and lateral synovial folds, which corresponds to the intermuscular plane between the iliocapsularis and gluteus minimus [17, 31].

Labral Structure and Function

The labrum is a horseshoe-shaped fibrocartilaginous tissue that attaches to the bony rim of the acetabulum and effectively deepens the acetabular socket. The labrum is triangular in cross section with the apex forming the free edge closest to the joint center. The labrum terminates inferiorly at the anterior and posterior edges of the acetabular fossa. At the terminal edges, the labrum becomes contiguous with the transverse acetabular ligament (Fig. 2) [17]. The base of the triangular labrum (edge furthest from the joint center) attaches to the acetabular articular cartilage through a transition zone of calcified cartilage with a distinct tidemark [32]. The average labral thickness is reported to be 5.3 ± 2.6 mm [33]. However, labral thickness varies by location; the largest diameter is located at the posterosuperior aspect and the smallest diameter is in the anteroinferior aspect of the acetabular rim. The labrum is a neurovascular structure that receives its vascular supply from the exterior capsular attachments, which penetrate the labral tissue at its base. This vascular configuration leaves the central most region of the labrum with rather poor vascular perfusion and thus limited healing potential. Conversely, in similar fashion to the knee meniscus, the peripheral capsulolabral junction has the highest healing potential [34]. The labrum also contains free nerve endings that carry both proprioceptive and nociceptive fibers. This likely substantiates clinical results that suggest athletes with torn labral tissue have decreased proprioception and pain [35].

In addition to the stability conferred by the osseous anatomy, more recent studies suggest that the soft-tissue envelope around the joint, the joint capsule and labrum, may contribute a large portion of total hip stability [36–38]. Tan et al. [33] found that total acetabular surface area coverage increased more than 25 % and the acetabular volume increased by approximately 20 % in the presence of an intact labrum as compared to a hip joint devoid of labrum. The efficacy of the labrum in providing stability likely stems from its ability to function as a “suction seal,” resisting fluid extravasation from between the femoral head and acetabulum. This labral seal creates the divide between the central intra-articular compartment and the peripheral intra-capsular, extra-articular compartment. Furthermore, the labrum increases the intra-articular hydrostatic pressure and uniformity of load distribution. Biomechanical studies have demonstrated that progressive damage to the labrum correlates with increased risk of hip instability [36, 38, 39].

Ligamentum Teres Structure and Function

The ligamentum teres is an intra-articular but extra-synovial structure that runs from the fovea capitis (area of the femoral head devoid of cartilage) to the acetabular fossa. With an average length of 35 mm, the ligament has two functional tissue bands, anterior and posterior. The two tissue bands diverge as they insert broadly in the acetabular fossa and blend with the transverse acetabular ligament. The ligament becomes taught in adduction, flexion, and external rotation of the hip, prompting some investigators to propose a hip stabilization role for the ligament. This secondary stabilizing effect may be most pronounced in patients with labral deficiency or a dysplastic hip [17, 40].

Layer 3: Contractile Layer

Layer 3 is the contractile layer of the hip and hemipelvis and it consists of all musculature around the hemipelvis, including the lumbosacral musculature and pelvic floor. Layer 3 is responsible for muscular balance and dynamic stability of the hip, pelvis, and trunk. Abnormal mechanics present within layer 1 (i.e., from FAI) can lead to increased mechanical stresses on the SI joint, pubic symphysis, and ischium. Likewise, FAI (layer 1 pathology) can lead to secondary increases in the strains of the muscles attached to these pelvic structures and thus compensatory injuries to layer 3 musculature. Furthermore, patients with FAI, when compared to a control group, have decreased maximal voluntary contraction levels in all the major muscle groups around the hip joint [41]. The decreased maximal voluntary contractions were most pronounced with hip abduction (28 % decrease), hip flexion (26 %), external rotation (18 %), and adduction (11 %). These results led the authors to conclude that structural pathology in layer 1 can lead to pain and muscle dysfunction [41].

Enthesopathies and/or tendinopathies related to the layer 3 anatomy can result in a variety of periarticular muscular injuries. Due to the large number of muscles in this body region, the muscle injuries are subcategorized based upon their location (origin or insertion) relative to the hip joint (anterior, medial, posterior, and lateral). Anterior enthesopathy describes injury to the following anatomic locations: hip flexor strains, psoas impingement, and subspine impingement. The medial enthesopathies include adductor and rectus abdominus tendinopathies, which have traditionally been described as athletic pubalgia or "sports hernia." Posterior enthesopathies include proximal hamstring strains but can also include injuries to the short external rotators, including the piriformis, and may involve a constellation of pain patterns described as "deep gluteal syndrome." Deep gluteal syndrome involves posterior soft-tissue injury and irritation or compression of the sciatic nerve [42]. Lateral enthesopathies involve strains of the gluteus medius or minimus and injuries within the peritrochanteric space. The muscles crossing the hip joint are numerous – 27 altogether; however, the large number of muscles can be compartmentalized into groups based on location and function (Table 3). The primary hip flexors are the iliacus, psoas, iliocapsularis, pectineus, direct and indirect rectus femoris, and sartorius. The hip extensors are gluteus maximus, semimembranosus, semitendinosus, short and long head of biceps femoris, and adductor magnus (ischiocondyle portion). Hip abductors include the gluteus medius, gluteus minimus, tensor fascia lata, and iliotibial band. The adductors are comprised of the adductor brevis, adductor longus, adductor magnus (anterior portion), and gracilis. The external rotators include the piriformis, quadratus femoris, superior and inferior gemellus, obturator externus, and obturator internus [17]. A detailed understanding of the hip and hemipelvic musculature, including the attachments, functions, and innervations, is critical for the accurate diagnosis of hip pain. Furthermore, knowledge of this anatomy is crucial to safely provide open or arthroscopic treatment to the hip while minimizing iatrogenic injury to the critical muscular envelope of the joint.

Iliopsoas Structure and Function

Based on its anatomic position, origins, and insertions, the iliopsoas is the only periarticular hip muscle that is able to contribute to stability and movement of the trunk, pelvis, and leg. Because the iliopsoas has two portions with separate innervations, the two muscles may act in unison or

Table 3 Overview of the peri-articular hip musculature and innervations

Muscle group – Action	Muscles (Innervation)
Hip flexors	Iliacus (Femoral nerve, L1-4)
	Psoas (L1-4)
	Iliocapsularis (L1-2)
	Pectineus (Femoral and obturator nerves, L2-4)
	Rectus femoris: direct and indirect heads (Femoral nerve, L2-4)
	Sartorius (Femoral nerve, L2-3)
Hip extensors	Gluteus maximus (Inferior gluteal nerve, L5-S2)
	Semimembranosus (Sciatic nerve – tibial division, L5-S2)
	Semitendinosus (Sciatic nerve – tibial division, L5-S2)
	Biceps femoris
	Short head (Sciatic nerve – fibular division, L5-S2)
	Long head (Sciatic nerve – tibial division, L5-S2)
	Adductor magnus: ischiocondylar (Sciatic nerve, L4)
Hip abductors	Gluteus medius (Superior gluteal nerve, L5,S1)
	Gluteus minimus (Superior gluteal nerve, L5,S1)
	Tensor fascia lata (Superior gluteal nerve, L4-5)
Hip adductors	Adductor brevis (Obturator nerve, L2-4)
	Adductor longus (Obturator nerve, L2-4)
	Adductor magnus: anterior (Obturator nerve, L2-4)
	Gracilis (Obturator nerve, L2-3)
Hip external rotators	Piriformis (Ventral rami, S1-2)
	Quadratus femoris (Nerve to quadratus, L5-S1)
	Superior gemellus (Nerve to obturator internus, L5-S1)
	Inferior gemellus (Nerve to quadratus, L5-S1)
	Obturator externus (Obturator nerve, L3-4)
	Obturator internus (Nerve to obturator internus, L5-S1)

separately at any given time. Work by Andersson et al. [43] examined the role of the iliacus and psoas separately in response to different body positions and actions via electromyography (EMG) and found that both muscles are involved in hip flexion and maximal thigh abduction. The iliacus is selectively activated for motion between the hip and pelvis, whereas the psoas is selectively activated for stabilizing the lumbar spine in the standing position in response to axial load applied to the contralateral side of the body [43]. The psoas tendon's close relationship with the anterior hip capsule allows it to function as a dynamic and static stabilizer during hip motion [44]. During normal ranges of hip flexion and extension, the psoas may be displaced laterally with flexion and displaced medially with hip extension. This motion may explain the pain associated with internal snapping of the psoas over the femoral head or iliopectineal eminence [45]. In a cohort of patients undergoing psoas lengthening for symptomatic snapping refractory to nonoperative measures, Fabricant et al. [46] demonstrated inferior clinical outcomes were associated with higher (greater than 25°) femoral anteversion. This finding highlights the importance of the psoas tendon in stabilizing the hip joint during functional range of motion and provides a key example of the interplay between the osteochondral (layer 1) and the contractile (layer 3) layers.

Iliocapsularis Structure and Function

This lesser-known muscle can be found overlying the anteromedial joint capsule. Research suggests that contraction of the iliocapsularis results in tightening of the hip capsule and subsequently imparts relative stability to the hip joint [47, 48]. Ward et al. [48] found that the iliocapsularis was more prominent in the dysplastic hip than their nondysplastic counterparts. In addition to its role as a dynamic stabilizer of the hip capsule, the iliocapsularis functions as an important landmark in hip arthroscopy when performing the vertical limb of a T-shaped capsulotomy. To avoid denervation or vascular injury to the capsule or labrum, the vertical limb of the T should be made when necessary in the intermuscular plane between the gluteus minimus and the iliocapsularis muscles.

Gluteus Minimus and Medius Structure and Function

The gluteus minimus muscle originates from the external ilium and the inside of the pelvis at the sciatic notch, and it inserts at both the greater trochanter and the anterosuperior hip capsule. The gluteus minimus can function as a hip flexor, hip internal rotator, hip external rotator, or hip abductor depending on the position of the lower extremity. During hip arthroscopy, the inserting fibers on the anterosuperior capsule serve as a reproducible landmark between the central and peripheral compartments. During open hip procedures, disruption of the gluteus minimus can lead to loss of anatomic and structural hip stability [49].

The gluteus medius muscle originates from the external superior surface of the ilium and the gluteal aponeurosis, and it inserts at the lateral aspect of the greater trochanter. Robertson et al. [50], in a cadaveric study, demonstrated that the medius has two distinct and consistent insertion sites on the greater trochanter. The larger of the two is the lateral facet and the smaller is the superoposterior facet [50]. The function of the gluteus medius is mainly hip abduction and hip internal rotation.

The gluteus medius and minimus have been termed "the rotator cuff of the hip," illustrating the importance of the hip abductors in normal hip function and their role in hip pathology, mainly greater trochanteric pain syndrome (GTPS) [51–54]. The incidence of gluteus medius tendinopathy and tearing far exceeds that of the gluteus minimus [55, 56]. Clinically, tears or weakening of the musculature may present with a Trendelenburg sign or difficulty with stair ascension. Recalcitrant GTPS due to medius tearing has been shown to be successfully managed with surgical repair, emphasizing the importance of knowing the native anatomic insertion [51]. Classically the surgical repair was done through an open approach; however, more recently studies have documented good and excellent outcomes following endoscopic management [57, 58].

Hamstring Structure and Function

Aside from the short head of the biceps femoris (linea aspera), the remainder of the hamstring complex originates from the ischial tuberosity and inserts distally below the knee on the proximal tibia. The tibial branch of the sciatic nerve is the innervation for the semitendinosus, the semimembranosus, and the long head of the biceps femoris. The short head of the biceps femoris is innervated by the peroneal branch of the sciatic nerve. The semitendinosus and long head of the biceps femoris share a common proximal tendon and origin footprint on the ischial tuberosity. The semimembranosus also arises from the ischial tuberosity but from a distinctly separate anterolateral footprint [59].

The characteristic mechanism of injury for a complete proximal hamstring tear involves forced hip flexion with the knee held in extension; however, acute tears or avulsions may occur with a number of sporting activities that require rapid acceleration or deceleration maneuvers [60–62]. In addition to the acute tear, proximal hamstring injuries can be related to increased stress and compensatory motion subsequent to decreased hip joint motion caused by FAI [63]. Nonoperative treatment consisting of rest, ice, physical therapy, and motion exercise is typically advocated for partial tears and tendinosis of the proximal hamstrings [64]. Steroid injections into the proximal tendon sheath have a role in patients who fail to improve with initial nonoperative measures. Lastly, surgical repair is indicated for full-thickness tears and partial-thickness tears or tendinosis recalcitrant to nonoperative interventions. Recently, Dierckman and Guanche [65] described an endoscopic technique for proximal hamstring repair, which may eliminate some of the morbidity associated with an open surgical approach; however, experience is required given the risk of injury to the sciatic nerve, which is in close proximity to the hamstring origin.

Additional Layer 3 Pathology

The general concept of compensatory motion and muscle strain plays a role in the layer 3 evaluation. Layer 3 injuries to the abdominal wall musculature and the development of sports hernias (athletic pubalgia) are often secondary injury patterns in response to restricted terminal range of motion caused by FAI, deviation from the normal anatomy in layer 1 [66]. The abnormal bony contact associated with FAI leads to a mechanical block to terminal hip joint motion, and in response alternate sites (lumbar spine, pubic symphysis, sacroiliac joint, posterior acetabulum) compensate by increasing their motion. The coexistence of FAI and athletic pubalgia and the compensatory injury patterns caused by FAI have been well documented [67, 68]. Hammoud et al. [68] evaluated 38 professional athletes with concomitant FAI and athletic pubalgia. They found that 32 % of their cohort had previously undergone surgery for athletic pubalgia and none of those patients were able to return to their previous level of sport. Once the FAI was addressed, all of these patients were able to return to their professional sporting activity [68]. Similarly, Larson et al. [67] examined a cohort of high-level athletes presenting with both intra-articular hip pathology and extra-articular athletic pubalgia and noted 89 % return to play at preoperative levels when both areas of pathology were addressed as compared to only a 25 % return-to-play rate when the extra-articular pathology was addressed in isolation and a 50 % return-to-play rate when the intra-articular pathology was addressed in isolation [67].

The concept of the kinetic chain (upstream muscle and joint activation affects downstream muscle and joint activation) can be applied to the hip and the adjacent muscle and joint structures. Weakness, limitations in motion, and pain in the hips (upstream component) caused by FAI can affect the biomechanics of rotation of the pelvis and trunk (downstream component) in the throwing athlete [69]. The result of limited pelvic and trunk rotation in the throwing athlete can be decreased accuracy and velocity as well as a predisposition to injury in the throwing shoulder and elbow [69, 70]. In addition to injuries in the throwing athlete, FAI has been linked to knee injuries in the kicking athlete, further reinforcing the importance of the kinetic chain [71]. In a cohort of 50 soccer players sustaining anterior cruciate ligament tears, greater than half had radiographic evidence of hip abnormalities [71].

Layer 4: Neurokinetic Layer

Layer 4 is the neurokinetic layer, comprised of the thoracolumbosacral plexus, lumbopelvic tissue, and lower extremity structures. Locally, at its most basic level, this layer is responsible for blood supply and innervation to the surrounding tissues. However, this layer also provides the mechanoreceptors and nociceptors that are responsible for proprioception and pain in and around the hip. This layer also serves as the neuromuscular link and thus dictates functional control of the entire segment as it moves within space. Globally, this layer also accounts for the posture of the pelvis with regard to the femur. Pathology within this layer includes nerve compression and pain syndromes, neuromuscular dysfunction, and spine radicular referral patterns. The most common peripheral nerve disorders adjacent to the hip include lateral femoral cutaneous neuropathy (meralgia paresthetica), femoral neuropathy, sciatic neuropathy (piriformis syndrome), obturator neuropathy, superior and inferior gluteal neuropathies, pudendal neuropathy, and ilioinguinal, iliohypogastric, and genitofemoral neuropathies [72–74].

All of the neural structures around the hip are paired with their vascular counterparts except in the case of the superficial lateral femoral cutaneous nerve (Fig. 5). The medial and lateral circumflex arteries supply blood to the hip joint. Both circumflex vessels typically originate from the deep

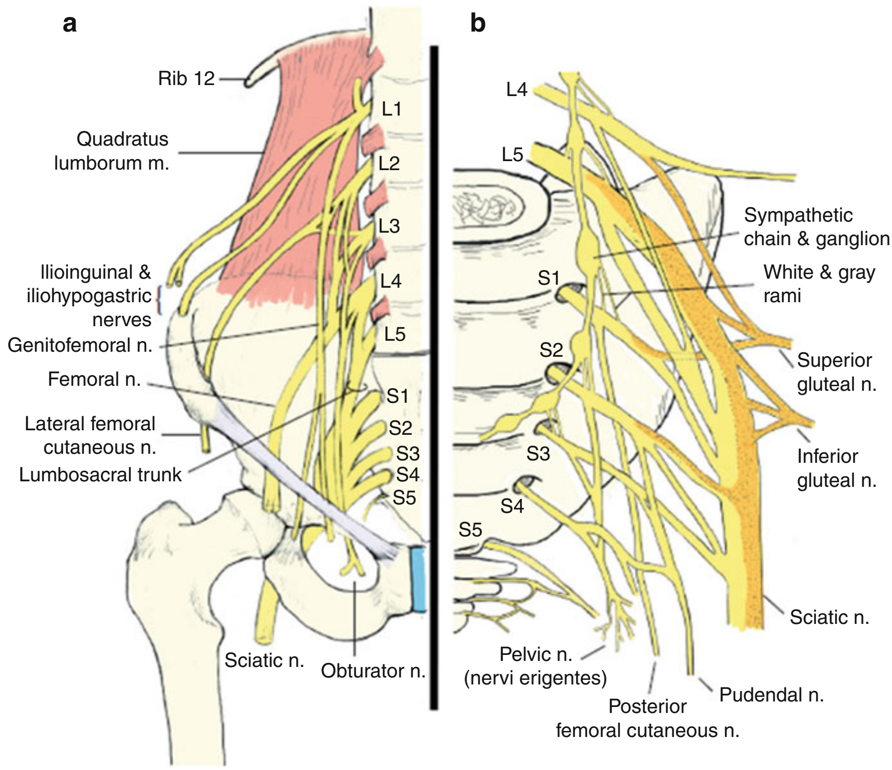


Fig. 5 Illustration of the lumbosacral plexus with emphasis on the lumbar nervous system (**a**) and sacral nervous system (**b**) (From Mathers LH, et al. Clinical Anatomy: Principles. St. Louis: Mosby; 1996. Copyright Elsevier)

femoral artery; however, variants originating from the common femoral artery have been described. A small branch off the posterior division of the obturator artery runs through the ligamentum teres and may contribute to femoral head vascularity. Although the labrum is not a well-vascularized structure, it derives its blood flow from the obturator, superior gluteal, and inferior gluteal arteries.

The femoral neurovascular structures (nerve, vein, and artery) exit the pelvis below the inguinal ligament approximately halfway between the pubic tubercle and the ASIS. The femoral nerve lies on the iliopsoas muscle and is the most lateral of the three structures and superficial in depth. The femoral nerve provides motor innervation to the psoas, iliacus, pectineus, sartorius and quadriceps muscles, while providing sensation to the anterior thigh via the anterior cutaneous branches. The lateral femoral cutaneous nerve (LFCN) of the thigh originates from the lumbar plexus and exits the pelvis deep to the inguinal ligament superior to the femoral nerve and in close proximity to the ASIS. Due to its location relative to the ASIS, the LFCN is predisposed to external compression or injury with anterior and mid-anterior arthroscopic portals (meralgia paresthetica). The obturator nerve originates from the lumbar plexus and exits the pelvis via the obturator canal before dividing into two divisions, anterior and posterior. The obturator nerve provides sensation to the inferomedial thigh via cutaneous branches and provides motor innervation to the gracilis, obturator externus, and adductor muscle group. Injury to the nerve has been reported with retractor placement posterior to the transverse acetabular ligament in open approaches to the hip joint. The sciatic nerve takes origin from the lumbosacral plexus and exits the pelvis through the greater sciatic foramen. In the majority of patients, the nerve lies deep to piriformis muscle before coursing superficially over the remainder of the short external rotators. The sciatic nerve provides motor innervation to the short external rotators and then branches distally into the tibial nerve and common peroneal nerve. Compression of the sciatic nerve at the level of the piriformis (piriformis syndrome) can cause a constellation of symptoms, including buttock pain, muscular tension around the hip, and radicular symptoms extending into the lower extremity. The superior and inferior gluteal nerves exit the pelvis through the greater sciatic foramen and course with their arterial counterparts. The superior gluteal nerve exits the pelvis at or above the level of the piriformis and provides innervation to the gluteus medius, gluteus minimus, and tensor fascia lata. The inferior gluteal nerve exits inferior to the piriformis and mainly innervates the gluteus maximus. Neither the superior nor the inferior gluteal nerve has a sensory role. Sensation overlying the gluteal region is provided by the cluneal nerves, which are branches of the lumbar and sacral dorsal rami. In addition to the compressive nerve syndromes and mononeuropathies, it is possible for hip and groin pain to be due to myelopathy or radiculopathy. Although not common, radiculopathy and myelopathy should be kept in mind whenever evaluating the athletic patient with a painful hip [17, 72]. A complete understanding of the neuromuscular relationships between the spine and the affected lower extremity is crucial to the successful diagnosis and management of the patient with hip pain.

Summary

Appropriate diagnosis and treatment for the young patient with hip pain can be challenging given the complex anatomy of the hip and hemipelvis. Dividing this complex anatomy into its functional layers – osteochondral layer 1, inert capsulolabral layer 2, contractile muscular layer 3, and neurokinetic layer 4 – assists in simplifying the diagnostic process for hip pain. The layered approach should be employed as a framework for pinpointing pain generators and pathology. Frequently patients will have related and symptomatic discomfort in multiple layers, and recognition of this pathology is critical for defining the correct treatment approach as well as counseling the patient regarding expectations of surgical interventions. The routine utilization of the layered approach is recommended for the evaluation of each patient with a chief complaint of groin pain that is potentially related to the hip or hemipelvis. Implementing this approach in a systematic and reproducible fashion will increase diagnostic accuracy and ultimately lead to successful treatment.

References

1. Bedi A, Dolan M, Leunig M, Kelly BT. Static and dynamic mechanical causes of hip pain. Arthroscopy. 2011;27(2):235–51.
2. Bredella MA, Stoller DW. MR imaging of femoroacetabular impingement. Magn Reson Imaging Clin N Am. 2005;13(4):653–64.
3. Dolan MM, Heyworth BE, Bedi A, Duke G, Kelly BT. CT reveals a high incidence of osseous abnormalities in hips with labral tears. Clin Orthop Relat Res. 2011;469(3):831–8.
4. Ganz R, Leunig M, Leunig-Ganz K, Harris WH. The etiology of osteoarthritis of the hip: an integrated mechanical concept. Clin Orthop Relat Res. 2008; 466(2):264–72.
5. Ganz R, Parvizi J, Beck M, Leunig M, Notzli H, Siebenrock KA. Femoroacetabular impingement: a cause for osteoarthritis of the hip. Clin Orthop Relat Res. 2003;417:112–20.
6. Leunig M, Beck M, Woo A, Dora C, Kerboull M, Ganz R. Acetabular rim degeneration: a constant finding in the aged hip. Clin Orthop Relat Res. 2003;413:201–7.
7. Leunig M, Werlen S, Ungersbock A, Ito K, Ganz R. Evaluation of the acetabular labrum by MR arthrography. J Bone Joint Surg Br. 1997;79(2):230–4.
8. Wenger DE, Kendell KR, Miner MR, Trousdale RT. Acetabular labral tears rarely occur in the absence of bony abnormalities. Clin Orthop Relat Res. 2004;426:145–50.
9. Voos JE, Mauro CS, Kelly BT. Femoroacetabular impingement in the athlete: compensatory injury patterns. Oper Tech Orthop. 2010;20(4):231–6.
10. Clohisy JC, Nunley RM, Otto RJ, Schoenecker PL. The frog-leg lateral radiograph accurately visualized hip cam impingement abnormalities. Clin Orthop Relat Res. 2007;462:115–21.
11. Garbuz DS, Masri BA, Haddad F, Duncan CP. Clinical and radiographic assessment of the young adult with symptomatic hip dysplasia. Clin Orthop Relat Res. 2004;418:18–22.
12. Lequesne M, Bellaiche L. Anterior femoroacetabular impingement: an update. Joint Bone Spine. 2012; 79(3):249–55.
13. Manaster BJ, Zakel S. Imaging of femoral acetabular impingement syndrome. Clin Sports Med. 2006; 25(4):635–57.

14. Meyer DC, Beck M, Ellis T, Ganz R, Leunig M. Comparison of six radiographic projections to assess femoral head/neck asphericity. Clin Orthop Relat Res. 2006;445:181–5.
15. Notzli HP, Wyss TF, Stoecklin CH, Schmid MR, Treiber K, Hodler J. The contour of the femoral head-neck junction as a predictor for the risk of anterior impingement. J Bone Joint Surg Br. 2002;84(4):556–60.
16. Tannast M, Siebenrock KA, Anderson SE. Femoroacetabular impingement: radiographic diagnosis–what the radiologist should know. AJR Am J Roentgenol. 2007;188(6):1540–52.
17. Hansen JT. Netter's clinical anatomy. Philadelphia: Saunders/Elsevier; 2010.
18. Lindstrom JR, Ponseti IV, Wenger DR. Acetabular development after reduction in congenital dislocation of the hip. J Bone Joint Surg Am. 1979;61(1):112–8.
19. Birkenmaier C, Jorysz G, Jansson V, Heimkes B. Normal development of the hip: a geometrical analysis based on planimetric radiography. J Pediatr Orthop B. 2010;19(1):1–8.
20. Carbonell PG, de Puga DB, Vicente-Franqueira JR, Ortuno AL. Radiographic study of the acetabulum and proximal femur between 1 and 3 years of age. Surg Radiol Anat. 2009;31(7):483–7.
21. Horii M, Kubo T, Hachiya Y, Nishimura T, Hirasawa Y. Development of the acetabulum and the acetabular labrum in the normal child: analysis with radial-sequence magnetic resonance imaging. J Pediatr Orthop. 2002;22(2):222–7.
22. Reikeras O, Bjerkreim I, Kolbenstvedt A. Anteversion of the acetabulum and femoral neck in normals and in patients with osteoarthritis of the hip. Acta Orthop Scand. 1983;54(1):18–23.
23. Leunig M, Huff TW, Ganz R. Femoroacetabular impingement: treatment of the acetabular side. Instr Course Lect. 2009;58:223–9.
24. Leunig M, Nho SJ, Turchetto L, Ganz R. Protrusio acetabuli: new insights and experience with joint preservation. Clin Orthop Relat Res. 2009;467(9): 2241–50.
25. Maquet P, Vu Anh T. On the forces exerted on the hip during gait. Arch Orthop Trauma Surg. 1981;99(1):53–8.
26. Nassif NA, Pekmezci M, Pashos G, Schoenecker PL, Clohisy JC. Osseous remodeling after femoral head-neck junction osteochondroplasty. Clin Orthop Relat Res. 2010;468(2):511–8.
27. Fuss FK, Bacher A. New aspects of the morphology and function of the human hip joint ligaments. Am J Anat. 1991;192(1):1–13.
28. Martin HD, Savage A, Braly BA, Palmer IJ, Beall DP, Kelly B. The function of the hip capsular ligaments: a quantitative report. Arthroscopy. 2008;24(2):188–95.
29. Hewitt J, Guilak F, Glisson R, Vail TP. Regional material properties of the human hip joint capsule ligaments. J Orthop Res. 2001;19(3):359–64.
30. Ito H, Song Y, Lindsey DP, Safran MR, Giori NJ. The proximal hip joint capsule and the zona orbicularis contribute to hip joint stability in distraction. J Orthop Res. 2009;27(8):989–95.
31. Kalhor M, Beck M, Huff TW, Ganz R. Capsular and pericapsular contributions to acetabular and femoral head perfusion. J Bone Joint Surg Am. 2009;91(2): 409–18.
32. Seldes RM, Tan V, Hunt J, Katz M, Winiarsky R, Fitzgerald Jr RH. Anatomy, histologic features, and vascularity of the adult acetabular labrum. Clin Orthop Relat Res. 2001;382:232–40.
33. Tan V, Seldes RM, Katz MA, Freedhand AM, Klimkiewicz JJ, Fitzgerald Jr RH. Contribution of acetabular labrum to articulating surface area and femoral head coverage in adult hip joints: an anatomic study in cadavera. Am J Orthop (Belle Mead NJ). 2001;30(11):809–12.
34. Kelly BT, Weiland DE, Schenker ML, Philippon MJ. Arthroscopic labral repair in the hip: surgical technique and review of the literature. Arthroscopy. 2005;21(12):1496–504.
35. Kim YT, Azuma H. The nerve endings of the acetabular labrum. Clin Orthop Relat Res. 1995;320:176–81.
36. Crawford MJ, Dy CJ, Alexander JW, et al. Frank Stinchfield Award. The biomechanics of the hip labrum and the stability of the hip. Clin Orthop Relat Res. 2007;465:16–22.
37. Dy CJ, Thompson MT, Crawford MJ, Alexander JW, McCarthy JC, Noble PC. Tensile strain in the anterior part of the acetabular labrum during provocative maneuvering of the normal hip. J Bone Joint Surg Am. 2008;90(7):1464–72.
38. Greaves LL, Gilbart MK, Yung AC, Kozlowski P, Wilson DR. Effect of acetabular labral tears, repair and resection on hip cartilage strain: a 7 T MR study. J Biomech. 2010;43(5):858–63.
39. Ferguson SJ, Bryant JT, Ganz R, Ito K. An in vitro investigation of the acetabular labral seal in hip joint mechanics. J Biomech. 2003;36(2):171–8.
40. Rao J, Zhou YX, Villar RN. Injury to the ligamentum teres. Mechanism, findings, and results of treatment. Clin Sports Med. 2001;20(4):791–9. vii.
41. Casartelli NC, Maffiuletti NA, Item-Glatthorn JF, et al. Hip muscle weakness in patients with symptomatic femoroacetabular impingement. Osteoarthritis Cartilage. 2011;19(7):816–21.
42. Martin HD, Shears SA, Johnson JC, Smathers AM, Palmer IJ. The endoscopic treatment of sciatic nerve entrapment/deep gluteal syndrome. Arthroscopy. 2011;27(2):172–81.
43. Andersson E, Oddsson L, Grundstrom H, Thorstensson A. The role of the psoas and iliacus muscles for stability and movement of the lumbar spine, pelvis and hip. Scand J Med Sci Sports. 1995;5(1):10–6.
44. Alpert JM, Kozanek M, Li G, Kelly BT, Asnis PD. Cross-sectional analysis of the iliopsoas tendon and its relationship to the acetabular labrum: an anatomic study. Am J Sports Med. 2009;37 (8):1594–8.

45. Allen WC, Cope R. Coxa Saltans: the snapping hip revisited. J Am Acad Orthop Surg. 1995;3(5):303–8.
46. Fabricant PD, Bedi A, De La Torre K, Kelly BT. Clinical outcomes after arthroscopic psoas lengthening: the effect of femoral version. Arthroscopy. 2012;28(7):965–71.
47. Babst D, Steppacher SD, Ganz R, Siebenrock KA, Tannast M. The iliocapsularis muscle: an important stabilizer in the dysplastic hip. Clin Orthop Relat Res. 2011;469(6):1728–34.
48. Ward WT, Fleisch ID, Ganz R. Anatomy of the iliocapsularis muscle. Relevance to surgery of the hip. Clin Orthop Relat Res. 2000;374:278–85.
49. Beck M, Sledge JB, Gautier E, Dora CF, Ganz R. The anatomy and function of the gluteus minimus muscle. J Bone Joint Surg Br. 2000;82(3):358–63.
50. Robertson WJ, Gardner MJ, Barker JU, Boraiah S, Lorich DG, Kelly BT. Anatomy and dimensions of the gluteus medius tendon insertion. Arthroscopy. 2008;24(2):130–6.
51. Kagan 2nd A. Rotator cuff tears of the hip. Clin Orthop Relat Res. 1999;368:135–40.
52. Bird PA, Oakley SP, Shnier R, Kirkham BW. Prospective evaluation of magnetic resonance imaging and physical examination findings in patients with greater trochanteric pain syndrome. Arthritis Rheum. 2001;44 (9):2138–45.
53. Kong A, Van der Vliet A, Zadow S. MRI and US of gluteal tendinopathy in greater trochanteric pain syndrome. Eur Radiol. 2007;17(7):1772–83.
54. Bunker TD, Esler CN, Leach WJ. Rotator-cuff tear of the hip. J Bone Joint Surg Br. 1997;79(4):618–20.
55. Kingzett-Taylor A, Tirman PF, Feller J, et al. Tendinosis and tears of gluteus medius and minimus muscles as a cause of hip pain: MR imaging findings. AJR Am J Roentgenol. 1999;173(4):1123–6.
56. Connell DA, Bass C, Sykes CA, Young D, Edwards E. Sonographic evaluation of gluteus medius and minimus tendinopathy. Eur Radiol. 2003;13(6):1339–47.
57. Voos JE, Shindle MK, Pruett A, Asnis PD, Kelly BT. Endoscopic repair of gluteus medius tendon tears of the hip. Am J Sports Med. 2009;37(4):743–7.
58. Domb BG, Botser I, Giordano BD. Outcomes of endoscopic gluteus medius repair with minimum 2-year follow-up. Am J Sports Med. 2013;41(5):988–97.
59. Miller SL, Gill J, Webb GR. The proximal origin of the hamstrings and surrounding anatomy encountered during repair. A cadaveric study. J Bone Joint Surg Am. 2007;89(1):44–8.
60. Blasier RB, Morawa LG. Complete rupture of the hamstring origin from a water skiing injury. Am J Sports Med. 1990;18(4):435–7.
61. Orava S, Kujala UM. Rupture of the ischial origin of the hamstring muscles. Am J Sports Med. 1995; 23(6):702–5.
62. Klingele KE, Sallay PI. Surgical repair of complete proximal hamstring tendon rupture. Am J Sports Med. 2002;30(5):742–7.
63. Voos JE, Mauro CS, Kelly BT. Femoroacetabular impingement in the athlete: compensatory injury patterns. Oper Techniq Orthop. 2010;20:231–6.
64. Tibor LM, Sekiya JK. Differential diagnosis of pain around the hip joint. Arthroscopy. 2008;24(12): 1407–21.
65. Dierckman BD, Guanche CA. Endoscopic proximal hamstring repair and ischial bursectomy. Arthrosc Tech. 2012;1(2):e201–7.
66. Brown SH, Ward SR, Cook MS, Lieber RL. Architectural analysis of human abdominal wall muscles: implications for mechanical function. Spine. 2011;36(5):355–62 (Phila Pa 1976).
67. Larson CM, Pierce BR, Giveans MR. Treatment of athletes with symptomatic intra-articular hip pathology and athletic pubalgia/sports hernia: a case series. Arthroscopy. 2011;27(6):768–75.
68. Hammoud S, Bedi A, Magennis E, Meyers WC, Kelly BT. High incidence of athletic pubalgia symptoms in professional athletes with symptomatic femoroacetabular impingement. Arthroscopy. 2012; 28(10):1388–95.
69. Robb AJ, Fleisig G, Wilk K, Macrina L, Bolt B, Pajaczkowski J. Passive ranges of motion of the hips and their relationship with pitching biomechanics and ball velocity in professional baseball pitchers. Am J Sports Med. 2010;38(12):2487–93.
70. Fleisig GS, Barrentine SW, Escamilla RF, Andrews JR. Biomechanics of overhand throwing with implications for injuries. Sports Med. 1996;21(6):421–37.
71. Ellera Gomes JL, Palma HM, Becker R. Radiographic findings in restrained hip joints associated with ACL rupture. Knee Surg Sports Traumatol Arthrosc. 2010;18(11):1562–7.
72. Draovitch P, Edelstein J, Kelly BT. The layer concept: utilization in determining the pain generators, pathology and how structure determines treatment. Curr Rev Musculoskelet Med. 2012;5(1):1–8.
73. Leunig M, Beck M, Stauffer E, Hertel R, Ganz R. Free nerve endings in the ligamentum capitis femoris. Acta Orthop Scand. 2000;71(5):452–4.
74. Zazulak BT, Hewett TE, Reeves NP, Goldberg B, Cholewicki J. Deficits in neuromuscular control of the trunk predict knee injury risk: a prospective biomechanical-epidemiologic study. Am J Sports Med. 2007;35(7):1123–30.

11 Athletic Populations of Interest in Hip Arthroscopy and Hip Preservation Surgery

Rachel M. Frank, Randy Mascarenhas, Simon Lee, Michael J. Salata, and Shane J. Nho

Contents

R.M. Frank (✉)
Department of Orthopaedic Surgery, Rush University Medical Center, Chicago, IL, USA

R. Mascarenhas • S. Lee
Department of Orthopaedic Surgery, Midwest Orthopaedics at Rush, Rush University Medical Center, Chicago, IL, USA
e-mail: simon.x.lee@rushortho.com

M.J. Salata
Department of Orthopaedic Surgery, Joint Preservation and Cartilage Restoration Center, University Hospitals Case Medical Center, Cleveland, OH, USA
e-mail: michael.salata@uhhospitals.org

S.J. Nho
Department of Orthopaedic Surgery, Rush University Medical Center, Chicago, IL, USA

Midwest Orthopaedics at Rush, Hip Preservation Center, Rush University Medical Center, Chicago, IL, USA

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_11

Abstract

Hip arthroscopy and hip preservation surgery have experienced a tremendous upsurge in the past two decades. As imaging modalities, diagnostic abilities, and surgical instrumentation/implants continue to improve, indications for arthroscopic hip surgery are expanding at a rapid rate. Understanding the complex anatomy of the hip joint and appreciating the pathology responsible for both intra-articular and extra-articular pathology are crucial to selecting the appropriate patients and procedures. This is of utmost importance in the athletic patient population. Depending on the activity or sport, young athletes with hip pain may present in different ways with a variety of complaints. This chapter will discuss specific populations of interest in hip arthroscopy and hip preservation surgery. Diagnoses pertinent to this patient population will be discussed, with an emphasis on the unique injury patterns encountered in these patients.

Introduction

Hip preservation surgery has undergone exponential growth over the past two decades. Advances in our understanding of hip pain in both the general patient population and also in multiple specific patient populations have revolutionized our ability to treat these patients. Further, substantial recent improvements in imaging modalities and surgical instrumentation have completely changed the surgical approach to these patients, from techniques that were previously open and invasive to techniques that are now more efficient and less invasive. One area of particular interest to surgeons performing hip arthroscopy and hip preservation surgery is the evaluation and treatment of the young, athletic patient population [1–5]. These patients can be challenging to diagnose and treat, as they are often high-demand, high-level patients who desire a quick return to athletic activity. Depending on the activity or sport, young athletes with hip pain may present in different ways with different complaints.

General Classifications of Hip Pain in Athletes

Hip pain in young athletes can be characterized in several different ways. Often, hip pain is classified by its location about the joint, including lateral hip pain, anterior hip pain, posterior pain, and medial hip, or groin, pain [6–8]. Common causes of lateral hip pain include greater trochanteric bursitis, gluteus medius dysfunction, iliotibial band syndrome, and meralgia paresthetica. Anterior hip pain is more variable and can result from osteoarthritis, hip flexor tendinopathy, iliopsoas bursitis, occult hip fracture or femoral neck stress fracture, acetabular labral pathology, and avascular necrosis of the femoral head. Femoroacetabular impingement (FAI) is a common source of anterior hip pain in this patient population, but FAI-related pain can be referred to other areas throughout the hip. Posterior hip pain, even in the athlete, is often referred from the lumbar spine and/or from the sacroiliac joint. Other sources of posterior hip pain include hip extensor or rotator muscle strains, proximal hamstring ruptures, and piriformis syndrome. In addition to geographic location, hip pain can also be classified as either intra-articular or extra-articular. Intra-articular sources of pain include labral tears, FAI, capsular laxity, cartilage damage, ligamentum teres pathology, and loose bodies. On the other hand, extra-articular sources of hip pain can include iliopsoas tendinitis, iliotibial band syndrome, gluteus medius and/or minimus tendinitis, greater trochanteric bursitis, femoral neck stress fracture, piriformis syndrome, and, as previously mentioned, referred pain from the lumbar spine or sacroiliac joint.

Femoroacetabular Impingement

Femoroacetabular impingement (FAI) is becoming increasingly recognized in both the general and athletic patient populations. FAI is a disorder characterized by abnormal contact between the femoral head and acetabulum, with associated labrum and articular cartilage damage. While some patients have isolated cam deformities and

Fig. 1 AP pelvis radiographs in competitive hockey player illustrate bilateral acetabular crossover signs indicative of femoroacetabular impingement

others that isolated pincer deformities (see Fig. 1), the majority of patients with FAI have a combination of both. Hips with impingement have been associated with limited range of motion, hyperlaxity, and athletic pubalgia, among other conditions. It is unclear as to whether FAI makes patients more susceptible to other injuries or if FAI simply develops concurrently in these patients. Regardless, athletes involved in a variety of activities, including contact athletes, pivoting athletes, overhead athletes, endurance athletes, and hypermobile athletes, are, depending on the sport, at high risk for certain hip disorders, with or without concomitant FAI. While FAI is becoming increasingly recognized in this patient population [9], it is critical to have a general appreciation for the different types of hip pain unique to this patient population in order to determine the correct diagnosis and ultimately the appropriate treatment plan.

The Role of Hip Arthroscopy and Hip Preservation Surgery

Given the wide variety of potential diagnoses in patients presenting with hip pain, the need for both an accurate diagnosis and an appropriate treatment algorithm has become apparent. Arthroscopy has become an extremely valuable diagnostic tool in the evaluation of these patients. Diagnostic hip arthroscopy allows the clinician to evaluate for both intra- and extra-articular sources of pain, and with recent advances in instrumentation and soft tissue management, the procedure has become much more tolerable for the patient. In addition to examining intra-articular structures, including the labrum, capsule, and articular cartilage, hip arthroscopy also allows for a full evaluation of the peri-trochanteric space. Finally, hip arthroscopy allows for a dynamic intraoperative assessment of mechanical pathology, which can be extremely problematic in athletes. Mechanical pain in athletes is often thought to be related to dynamic and/or static factors. Dynamic factors include femoral retroversion, femoral varus, cam lesions, and pincer lesions, while static factors include femoral anteversion, femoral valgus, lateral acetabular under-coverage, and anterior acetabular under-coverage [1, 2, 9].

Overall, arthroscopy is extremely helpful in both the diagnosis and treatment of athletes presenting with hip pain. Given the variety of stresses placed upon the hip joint during different athletic activities, patient presentations and underlying diagnoses are variable. Specifically, while athletes such as football players, hockey players, and runners, for example, may each present to the office complaining of hip pain, the underlying diagnoses are likely to be different in each situation. This chapter will focus on several specific athletic patient populations who commonly present with a need for hip arthroscopy and/or hip preservation surgery. Specific patient populations to be discussed include contact athletes, pivoting athletes, overhead athletes, endurance athletes, and hypermobile athletes.

The Contact Athlete

Athletes involved in contact sports are at risk for a variety of different hip disorders. Participation in athletic activities involving physical contact, such as football, rugby, and hockey, among others, places athletes at risk for both repetitive mechanical intra-articular pain and direct impact-based injuries. Common injuries in this patient population include traumatic instability, contusions and “hip pointers,” and fractures of the hip and pelvis.

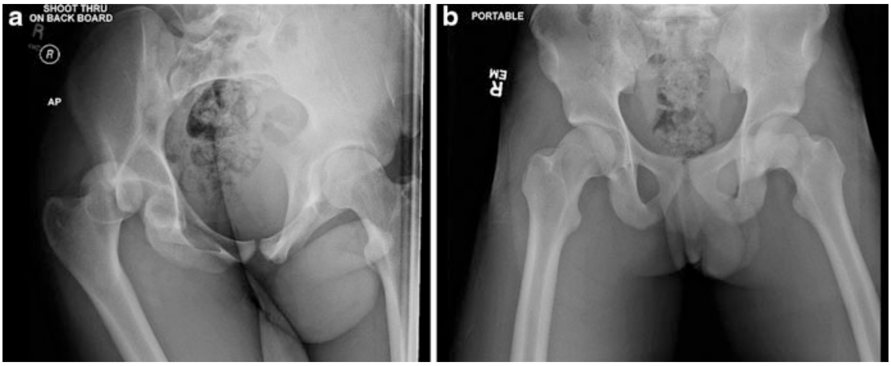


Fig. 2 (**a**) Portable AP pelvis radiographs show a posterior hip dislocation in a football player. (**b**) Postreduction films indicate a successful reduction of the dislocated hip

Of all athletes, those participating in contact sports are perhaps at the highest risk for traumatic hip instability, including subluxation and/or dislocation (see Fig. 2). Specifically, athletes most likely to sustain an injury resulting in hip instability include those participating in football and hockey [10]. It remains unclear as to whether these athletes are predisposed to hip instability due to underlying FAI. In a report of three cases of posterior hip instability by Berkes and colleagues [11], all three patients were found to have evidence of acetabular retroversion, a cam lesion, an elevated alpha angle, a labral injury, and a posterior acetabular rim fracture. While the presence of the bony fracture and possibly the labral injury can likely be attributed to the initial instability event, the other associated findings are clearly unrelated to the injury, and the question remains: do such anatomical abnormalities predispose these athletes to hip instability? Philippon and colleagues [10] performed hip arthroscopy on 14 professional athletes at a mean time of 125 days following traumatic hip dislocation and found all patients to have labral tears. Nine of the 14 athletes had evidence of FAI with cam- and/or pincer-type pathology. Despite the mean time from dislocation to reduction being only 3.56 h, all patients were found to have chondral defects at the time of arthroscopy, again suggesting possible preexisting intra-articular pathology from underlying FAI.

Hip pointer injuries are also common in contact athletes. The term “hip pointer” refers to an injury resulting from direct blunt trauma to the iliac crest. This type of injury is typically sustained only during contact sports after a direct blow to the hip and causes substantial pain and often an inability to continue participation [6, 12]. The trauma from the direct blow leads to subperiosteal edema with subsequent hematoma formation and a contusion of the iliac crest itself. In these situations, the history is usually diagnostic as patients are able to recall the specific inciting event and also point to where the pain is. Usually, plain radiographs are sufficient to rule out fracture and further diagnostic imaging is unnecessary. These patients will benefit from a course of activity modification, ice, and compression to the area. Occasionally, these patients may be managed temporarily with injections of local anesthetic to allow them quicker return to play [13]. While treatment for hip pointers is almost exclusively nonoperative, patients with persistent pain may be evaluated with hip arthroscopy in order to assess for possible concomitant intra-articular injuries.

Avulsion fractures near the hip represent another diagnosis commonly seen in the contact athlete, though noncontact athletes can sustain these injuries as well. Avulsion fractures are more common in the adolescent patient population and are typically the result of a sudden forceful contraction of a muscle attached to the apophysis. These injuries can be sustained with both forceful concentric and eccentric contractions. Common sites of injury include the anterior superior iliac spine (sartorius, tensor fascia lata), ischial tuberosity (hamstrings), anterior inferior iliac spine (direct head of rectus femoris), lesser trochanter (iliopsoas), greater trochanter (abductors), and iliac crest (from external and abdominal obliques) [14, 15]. While these injuries can usually be managed without surgery, nonunion or malunion can become problematic. For example, Matsuda reported on a case of a traumatic anterior iliac spine apophyseal avulsion fracture leading to secondary symptomatic FAI that ultimately required arthroscopic surgery with anterior iliac spinoplasty [16]. In this case, the patient was also noted to have acetabular retroversion and a cam deformity, which highlights the importance of evaluating for concomitant impingement-related symptoms in this patient population. Traumatic avulsion fractures/injuries of the ligamentum teres have also become increasingly recognized, and arthroscopic surgery is sometimes warranted. In a series including 23 patients with traumatic ligamentum teres ruptures undergoing hip arthroscopy, Byrd and Jones noted an average improvement in modified Harris Hip Score from 47 to 90 [17]. Interestingly, despite advanced imaging, the diagnosis of ligamentum teres rupture was made preoperatively in only 2 of the 23 cases, and thus arthroscopy proved to be both diagnostic and therapeutic for these patients.

The Pivoting Athlete

Athletes participating in sports that involve a substantial amount of pivoting are at increased risk for several specific hip injuries. Some of the more common sports involving pivoting include soccer, lacrosse, wrestling, Australian rules football, and field hockey. The amount of strain placed on the hip and pelvis during a forceful pivot motion can be significant, leading to multiple painful conditions, including FAI, athletic pubalgia, osteitis pubis, and muscle sprains secondary to overuse. The pivoting action itself is thought to translate force down the kinetic chain, causing a compensatory overload from the hip to the knee, ankle, and foot, potentially leading to injury in these areas as well. Athletes engaged in pivoting sports often develop hip pain localized to the groin. Given the complex anatomy in this region, the differential diagnosis in these patients is extremely broad. Common diagnoses include FAI, adductor muscle strains, and osteitis pubis. Other causes of groin pain in these athletes are summarized in Table 1.

Table 1 Common causes of groin pain in athletes

FAI
Adductor strains
Osteitis pubis
Athletic pubalgia
Ilioinguinal neuralgia
Avulsion fractures
Bursitis
Rectus femoris strain
Obturator nerve entrapment
AVN of the femoral head
SCFE
Snapping hip

Adductor muscle strains are common in the pivoting athlete patient population [18–22]. These injuries are typically referred to as groin strains and are most often encountered by athletes participating in ice hockey, soccer [23], and Australian football. Adductor strains are more likely to occur in athletes with decreased adductor strength relative to abductor strength, as noted by Tyler and colleagues [24]. Specifically, Tyler et al. examined 47 professional hockey players and noted that athletes were 17 times more likely to sustain an adductor strain if their adductor strength was less than 80 % that of the ipsilateral abductor [24]. In another study, decreased adductor strength as well as flexibility have been shown to be risk factors in athletes [19], though the study by Tyler did not find adductor flexibility (or lack thereof) to be a risk factor [24]. Osteitis pubis is another diagnosis commonly encountered in the pivoting athlete, with pain most often localized to the groin. Thought to be a chronic, overuse injury caused by overloading of the pubic symphysis and adjacent parasymphyseal bone, patients with osteitis pubis will eventually develop a bony stress reaction affecting the pubic symphysis and/or the parasymphyseal bone [25]. Arriving at this diagnosis can be challenging, as patients will often complain of vague groin pain with adduction-, rotation-, and flexion-type activities, but occasionally, patients will have pain directly over the pubic symphysis.

Athletic pubalgia, or sports hernia, has become an increasingly recognized diagnosis in the pivoting athlete. Occurring predominantly in men, athletic pubalgia is an occult hernia caused by weakness (or a tear) in the posterior inguinal wall. A variety of anatomical structures may be involved, including attenuation of the transversalis fascia, partial avulsion of the internal oblique muscle fibers at the pubic tubercle, and disruption of the body of the internal oblique muscle itself. The etiology is poorly understood but is thought to be related to repetitive twisting and turning at high speed, which places substantial stress on the inguinal wall musculature. Typical exacerbating motions in these athletes include trunk hyperextension and thigh hyperabduction [26, 27]. Patients with this injury often have pain relief with rest and symptoms only become apparent during exacerbating movements. Unlike other more classic hernia presentations, there is no palpable bulge or other clinically appreciable signs of hernia in these patients, making the diagnosis challenging. Patients will often have tenderness over the pubic tubercle, especially with resisted hip adduction and resisted sit-up motions.

Many of these athletes can have concomitant athletic pubalgia and FAI. It is unclear as to whether anatomical features of FAI predispose these patients to the development of a sports hernia. Certainly there is a relationship between limited hip range of motion, in particular flexion and internal rotation, and the development of symptomatic groin pain [28]. In these cases, it is imperative for the surgeon to appreciate the potential for underlying FAI, as both nonoperative and operative treatments aimed only at athletic pubalgia are unlikely to be beneficial [29, 30]. Hammoud and colleagues [31] reported on a series of 38 professional athletes with athletic pubalgia undergoing arthroscopic surgery for FAI. The authors noted 32 % of these athletes had previously undergone isolated surgical treatment for athletic pubalgia, with none of these patients returning to their previous level of competition after the index procedure. Following the arthroscopic procedure for FAI, 36 of 38 patients were able to return to their previous level of play at an average of 5.9 months postoperatively [31].

The Overhead Athlete

Overhead athletes are subject to the same subset of hip disorders commonly encountered in other athletic patient populations, including FAI. These athletes, however, may experience unique sequelae from FAI not typically found in other athletes, as alterations in their biomechanics often occur in a compensatory fashion up the kinetic chain, from the hip to the sacroiliac joint, spine, elbow, and shoulder. Thus, while many overhead athletes, including pitchers, quarterbacks, and volleyball players, present with elbow and/or shoulder injuries, some authors will argue that baseline hip pathology is ultimately responsible [32, 33]. The act of overhead throwing places a tremendous force on the hip at all stages in the pitching cycle. The biomechanics of the pitching cycle are complex, and while much attention is given to the force placed on both the shoulder and elbow, hip motion and stability also play a key role in stabilizing the shoulder/elbow and even generating ball velocity. In 2010, Robb and colleagues [34] examined 19 professional baseball pictures throughout the cycle with a focus on hip range of motion. The authors found a significantly decreased passive range of motion in the nondominant hip when compared to the dominant hip for all ranges. Further, the authors found that the total arc of rotation of the nondominant hand was correlated with ball velocity. They concluded that the motion disparity between the dominant and nondominant hips was correlated with various pitching biomechanical parameters [34]. Laudner et al. [35] found that pitchers have significantly smaller amounts of hip internal rotation compared to positional players and, as a result, rely more on energy created in the core and upper extremity to throw. This data suggests a correlation between upper extremity injuries in pitchers and abnormal hip kinematics (less range of motion).

In 2010, Scher and colleagues [33] found a possible relationship between hip extension and shoulder external rotation in baseball players with a history of shoulder injury. It is still unclear as if these athletes become injured due to the overhead throwing activity or if they have underlying anatomical features that place them at high risk for FAI and subsequent instability. Further, most high-level overhead athletes, including pitchers and quarterbacks, have intense practice regimens that require repetitive rotational loads on not only the shoulder but also the hip and other joints of the body involved in throwing. Hip arthroscopy is an extremely useful treatment in these patients, as noted by Klingenstein et al. [32]. In their retrospective study of 34 high-level baseball and lacrosse players with FAI, hip arthroscopy was able to bring 33 of the 34 athletes back to their pre-injury level of participation. The authors concluded that mechanical overload of the hip from FAI and subsequent secondary instability can have a detrimental effect on function, leading to potential decreased performance in these overhead athletes.

The Endurance Athlete

Hip pain in the endurance athlete can be extremely challenging to evaluate and treat. Often, these patients are high-demand, high-level athletes who are subject to a variety of overuse injuries. Further complicating the matter is the potential for these athletes to be unwilling to decrease or modify their training regimen. Athletes involved in running, rowing [36], and triathlon, as well as military recruits, are especially at risk for these injuries, which can include femoral neck stress fractures [37], proximal hamstring strains [38, 39], and abductor tears [40]. Femoral neck stress fractures remain one of the most commonly reported injuries in runners and military personnel. A high index of suspicion is necessary in endurance athletes presenting with vague complaints of groin pain, as physical examination may be nondiagnostic and radiographs may initially be normal. While it is still unclear as to whether these athletes have underlying FAI or whether underlying FAI makes them more susceptible to developing femoral neck stress fractures, recent reports have supported an association [41–43]. For example, Taylor-Haas et al. [42] described a marathon runner with 3 months of insidious hip pain, found to have a stress fracture at the inferomedial femoral neck on magnetic resonance imaging. In addition, this patient was found to have cam-type FAI and mild superolateral labral heterogeneity. In this case, it is possible that the patient's abnormal bony anatomy consistent with FAI altered the biomechanics of the hip joint, leading to abnormal stress placed on the femoral head-neck junction, potentially predisposing the patient to a stress fracture. Kuhn and colleagues [43] reported on 54 military recruits treated for femoral neck stress fractures and found a higher incidence of acetabular retroversion in these patients compared to controls. This data also indicates a potential association between abnormal hip orientation, altered hip mechanics, and possible increased susceptibility to femoral neck stress fracture. The authors concluded by discussing the possibility of imposing more aggressive screening of military recruits with known acetabular retroversion in order to identify those athletes that may be at higher risk for femoral neck stress fracture. Carrey and colleagues [41] noted similar findings in their review of 69 consecutive soldiers treated for femoral neck stress fracture. The authors found a radiographic crossover sign in 51 %, center edge angle over 40° in 47 %, and alpha angle over 50° in 55 % of the cohort. They concluded that underlying FAI may lead to abnormal stress across the femoral neck, placing these athletes at higher risk for femoral neck stress fractures [41]. Hip preservation surgery with closed reduction and percutaneous pinning or minimally invasive internal fixation is the treatment of choice for displaced stress fractures or those refractory to nonoperative management. In a series of 42 military recruits with femoral neck stress fractures, Lee et al. [44] reported fixation with either a compression hip screw or multiple cancellous screws to be an acceptable treatment strategy. The authors noted that delayed treatment and postoperative varus malalignment were risk factors for postoperative development of avascular necrosis of the femoral head.

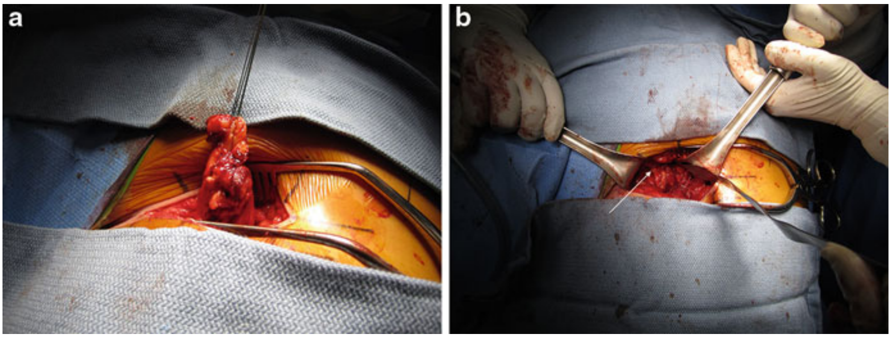


Fig. 3 (**a**) Proximal hamstring tendon rupture in a hurdler highlighted with tagging sutures. (**b**) Repair of the ruptured tendon illustrated with the *white arrow*

Endurance athletes are also at risk for muscle strains/tears. These are most often secondary to overuse, but acute injuries from explosive training exercises have also been reported. Two of the most common muscle groups involved are the hamstrings [38, 39] and the abductors [40]. While proximal hamstring tendon ruptures (See Fig. 3) are most often encountered in the acute setting due to a forceful eccentric contraction while sprinting, hurdling, or waterskiing, hamstring strains are more common in the runner or endurance athlete. Grade I and II strains are essentially incomplete tears of the muscle tendon, while Grade III strains represent a complete rupture of the musculotendinous unit. Previous laboratory work utilizing gait analysis has demonstrated that while running, the hamstrings undergo routine eccentric contraction during the last 25 % of the swing phase in order to assist proximally with hip extension while decelerating knee extension distally [38, 45]. The hamstrings then remain active during the first half of the stance phase via a concentric contraction to produce extension and resist the extension [38, 45]. While, often, patients with an acute tear are able to recall the original injury, patients with chronic strains due to overuse, such as those experienced by endurance athletes, may not recall any specific traumatic event. Minimally invasive endoscopic repair has been described for patients with proximal hamstring tendon ruptures [46, 47]. This type of surgical approach is advantageous over larger open approaches, due to improved visualization and the opportunity to perform a concomitant ischial bursectomy.

Recently, hip pathology in rowers has been described. This is a unique patient population that places a tremendous amount of force on the hip joint in a repetitive fashion. In a series by Boykin and colleagues [36], physical examination findings as well as imaging findings were used to confirm the presence of FAI in 18 rowing athletes (21 hips, 3 bilateral). Interestingly, the majority of the cohort was female (85 %), but it is unclear what role, if any, gender plays in the prevalence of FAI or in causing symptoms in patients with FAI who participate in rowing. In this study, arthroscopic intervention was employed in 18 hips; however, 6 of the 18 never returned to rowing.

The Hypermobile Athlete

Athletes requiring significant range of motion to perform their activity of choice, including dancers and gymnast, are at risk for a specific subset of hip disorders. Hip pathology can account for over 20 % of injuries in elite ballet dancers [48]. While the majority of these injuries likely occur from improper technique, muscle weakness, muscular imbalance, or overtraining, the movements and motions required for this sport place substantial demand on the hip joint. In ballet, for example, forcing a proper turnout can be extremely problematic for the hip joint. Given the extreme range of motion required to produce a

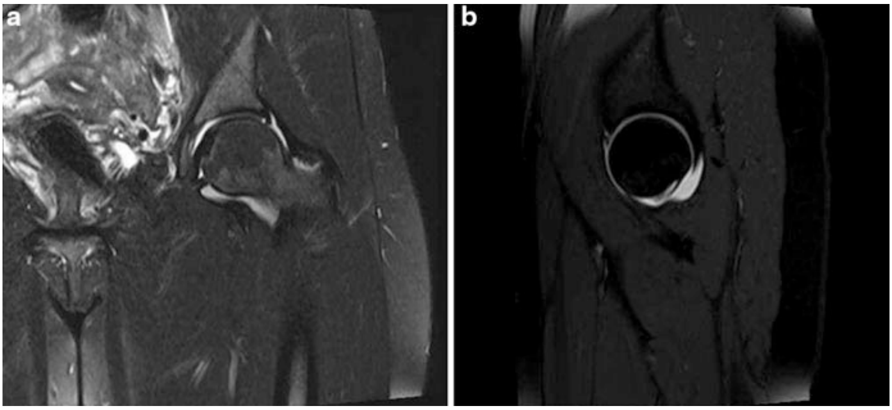


Fig. 4 (**a**) Coronal T2-weighted and (**b**) sagittal T1-weighted MRI sequences show a labral tear of the hip in a ballet dancer

satisfactory turnout, the labrum can often become pinched between the femoral head and acetabulum, leading to labral pathology (see Fig. 4) and symptoms consistent with FAI. This is especially problematic in patients with underlying FAI, as now abnormal stress (from the motion required to perform ballet) is placed on a hip with anatomy that is already abnormal.

High-level dancers are also at risk for a variety of coxa saltans or "snapping hip" syndromes [49–51]. External snapping hip occurs when the iliotibial band brushes against the greater trochanter, while internal snapping hip occurs when the iliopsoas tendon rubs against the anterior superior iliac spine, lesser trochanter, or iliopectineal ridge. Both these conditions are commonly encountered in ballet dancers. Snapping hip due to the iliotibial band can also lead to acute and/or chronic greater trochanter bursitis, which represents another source of lateral hip pain in this patient population. Multiple authors have described arthroscopic approaches for treating cases of snapping hip refractory to nonoperative management, with encouraging outcomes [50, 52]. For example, Anderson et al. [50] described clinical outcomes in 15 athletes undergoing arthroscopic iliopsoas tendon release for snapping hip. The authors reported excellent outcomes, with all 15 athletes returning to full participation at an average of 9 months following surgery.

Finally, both dancers and gymnasts may also experience piriformis syndrome, which represents a source of posterior hip pain in this patient population. With the substantial emphasis on hip range of motion and, in particular, rotation and turnout, the piriformis muscle can often become tight in these athletes, leading to irritation of the sciatic nerve as it courses behind the muscle. While most cases of piriformis syndrome can be treated without surgery, arthroscopic approaches have been described in refractory cases [53, 54].

Recently, some authors have suggested a correlation between dancers presenting with hip pain and underlying hip dysplasia [55]. Hip dysplasia is thought to be more prevalent in females than in males, which may explain the potential association with ballet dancers, the majority of which are female. Furthermore, when considering both male and female ballet dancers, female dancers may be at higher risk for hip pathology due to the use of their legs as levers for lifts and the typical higher height reached during certain maneuvers that require increased motion at the hip joint. Bauman and colleagues [56] reported on the radiographic and arthroscopic findings of 41 hips in professional dancers. The authors found that hip dysplasia was present in 55 % of patients, while cam impingement findings were seen in 25 % of patients. Thus, while additional research is needed in this area, hip dysplasia and a subsequent predisposition to hip pain may be seen in this special patient population.

Special Considerations

In-Season Management

Athletes presenting with hip pathology in the middle of their competitive season can be difficult to manage. While some injuries may be acute, such as a hip pointer as described above, other injuries are more of an acute or chronic exacerbation of an ongoing problem. In either situation, the athlete will typically demand the "quickest fix" possible with a rapid return to play. This can be exceptionally challenging in athletes presenting with hip pathology, as even arriving at the diagnosis can be difficult. Currently, there are no clear recommendations for in-season athletes presenting with symptomatic FAI regarding whether it is acceptable to have them continue with competitive play or sit out in order to address the underlying pathology with surgery. As described in the preceding sections, the majority of available studies do report good to excellent outcomes, including returned to pre-injury level of activity, in athletes undergoing hip arthroscopy for FAI [1–4]. The most appropriate timing for surgery, for those patients who require operative intervention, is nevertheless yet to be determined.

General Treatment Guidelines

In general, treatment for any athlete, whether in-season or off-season, should involve a stepwise approach to arrive at the correct diagnosis and formulate a treatment plan. All patients should undergo a thorough history that includes inquiring about the onset and duration of symptoms as well as previous treatments and traumatic events. A complete physical examination of both hips should be performed, assessing for both intra- and extra-articular hip pathology. Diagnostic work-up should continue with imaging studies including radiographs and advanced imaging including CT and/or MRI as appropriate. One helpful diagnostic modality is an intra-articular injection comprised of local anesthetic with or without corticosteroid, as this can be both diagnostic for intra-articular pathology and therapeutic [57]. Finally, the majority of athletes presenting with hip pain with appropriate surgical indications who either have exhausted nonoperative treatment or are otherwise not indicated for such treatment can ultimately undergo minimally invasive arthroscopy and hip preservation surgery.

Summary

The number of hip arthroscopy and hip preservation surgeries performed is increasing at a rapid rate. As the indications continue to evolve, pathologies that were once treated in an open, invasive fashion are now been addressed with minimally invasive techniques. As such, patients with both intra-articular and extra-articular hip disorders are becoming increasingly recognized, and these patients are now able to obtain surgical intervention that allows them quicker return to activity. Athletes, including those participating in contact sports, pivoting sports, overhead sports, endurance sports, and other unique activities, represent a challenging patient population to manage. Having a clear understanding of the specific pathologies unique to each of these patient populations is paramount to arriving at the correct diagnosis and ultimately the correct treatment decision.

References

1. Lynch TS, Terry MA, Bedi A, Kelly BT. Hip arthroscopic surgery: patient evaluation, current indications, and outcomes. Am J Sports Med. 2013; 41(5):1174–89.
2. Bedi A, Kelly BT, Khanduja V. Arthroscopic hip preservation surgery: current concepts and perspective. Bone Jt J. 2013;95-B(1):10–9.
3. Fabricant PD, Heyworth BE, Kelly BT. Hip arthroscopy improves symptoms associated with FAI in selected adolescent athletes. Clin Orthop Relat Res. 2012;470(1):261–9.
4. Kelly BT, Williams 3rd RJ, Philippon MJ. Hip arthroscopy: current indications, treatment options, and management issues. Am J Sports Med. 2003; 31(6):1020–37.

5. Tibor LM, Sekiya JK. Differential diagnosis of pain around the hip joint. Arthroscopy. 2008; 24(12):1407–21.
6. Frank RM, Slabaugh MA, Grumet RC, Bush-Joseph CA, Virkus WW, Nho SJ. Hip pain in active patients: what you may be missing. J Fam Pract. 2012;61(12):736–44.
7. Grumet RC, Frank RM, Slabaugh MA, Virkus WW, Bush-Joseph CA, Nho SJ. Lateral hip pain in an athletic population: differential diagnosis and treatment options. Sports Health. 2010;2(3):191–6.
8. Frank RM, Slabaugh MA, Grumet RC, Virkus WW, Bush-Joseph CA, Nho SJ. Posterior hip pain in an athletic population: differential diagnosis and treatment options. Sports Health. 2010;2(3):237–46.
9. Bedi A, Kelly BT. Femoroacetabular impingement. J Bone Jt Surg. 2013;95(1):82–92. American volume.
10. Philippon MJ, Kuppersmith DA, Wolff AB, Briggs KK. Arthroscopic findings following traumatic hip dislocation in 14 professional athletes. Arthroscopy. 2009;25(2):169–74.
11. Berkes MB, Cross MB, Shindle MK, Bedi A, Kelly BT. Traumatic posterior hip instability and femoroacetabular impingement in athletes. Am J Orthop. 2012;41(4):166–71.
12. Hall M, Anderson J. Hip pointers. Clin Sports Med. 2013;32(2):325–30.
13. Orchard JW, Steet E, Massey A, Dan S, Gardiner B, Ibrahim A. Long-term safety of using local anesthetic injections in professional rugby league. Am J Sports Med. 2010;38(11):2259–66.
14. Metzmaker JN, Pappas AM. Avulsion fractures of the pelvis. Am J Sports Med. 1985;13(5):349–58.
15. Rossi F, Dragoni S. Acute avulsion fractures of the pelvis in adolescent competitive athletes: prevalence, location and sports distribution of 203 cases collected. Skel Radiol. 2001;30(3):127–31.
16. Matsuda DK, Calipusan CP. Adolescent femoroacetabular impingement from malunion of the anteroinferior iliac spine apophysis treated with arthroscopic spinoplasty. Orthopedics. 2012;35(3):e460–3.
17. Byrd JW, Jones KS. Traumatic rupture of the ligamentum teres as a source of hip pain. Arthroscopy. 2004;20(4):385–91.
18. Padua DA, Bell DR, Clark MA. Neuromuscular characteristics of individuals displaying excessive medial knee displacement. J Athl Train. 2012;47(5):525–36.
19. Hrysomallis C. Hip adductors' strength, flexibility, and injury risk. J Strength Cond Res/Natl Strength Cond Assoc. 2009;23(5):1514–7.
20. Charnock BL, Lewis CL, Garrett Jr WE, Queen RM. Adductor longus mechanics during the maximal effort soccer kick. Sports Biomech/Int Soc Biomech Sports. 2009;8(3):223–34.
21. Grote K, Lincoln TL, Gamble JG. Hip adductor injury in competitive swimmers. Am J Sports Med. 2004;32(1):104–8.
22. Nicholas SJ, Tyler TF. Adductor muscle strains in sport. Sports Med. 2002;32(5):339–44.
23. Boykin RE, Stull JD, Giphart JE, Wijdicks CA, Philippon MJ. Femoroacetabular impingement in a professional soccer player. Knee Surg Sports Traumatol Arthrosc. 2013;21(5):1203–11.
24. Tyler TF, Nicholas SJ, Campbell RJ, McHugh MP. The association of hip strength and flexibility with the incidence of adductor muscle strains in professional ice hockey players. Am J Sports Med. 2001; 29(2):124–8.
25. Hiti CJ, Stevens KJ, Jamati MK, Garza D, Matheson GO. Athletic osteitis pubis. Sports Med. 2011;41(5):361–76.
26. Jakoi A, O'Neill C, Damsgaard C, Fehring K, Tom J. Sports hernia in National Hockey League players: does surgery affect performance? Am J Sports Med. 2013;41(1):107–10.
27. Farber AJ, Wilckens JH. Sports hernia: diagnosis and therapeutic approach. J Am Acad Orthop Surg. 2007;15(8):507–14.
28. Verrall GM, Hamilton IA, Slavotinek JP, et al. Hip joint range of motion reduction in sports-related chronic groin injury diagnosed as pubic bone stress injury. J Sci Med Sport/Sports Med Australia. 2005;8(1):77–84.
29. Larson CM, Pierce BR, Giveans MR. Treatment of athletes with symptomatic intra-articular hip pathology and athletic pubalgia/sports hernia: a case series. Arthroscopy. 2011;27(6):768–75.
30. Matsuda DK. Endoscopic pubic symphysectomy for reclacitrant osteitis pubis associated with bilateral femoroacetabular impingement. Orthopedics. 2010; 33(3):199.
31. Hammoud S, Bedi A, Magennis E, Meyers WC, Kelly BT. High incidence of athletic pubalgia symptoms in professional athletes with symptomatic femoroacetabular impingement. Arthroscopy. 2012; 28(10):1388–95.
32. Klingenstein GG, Martin R, Kivlan B, Kelly BT. Hip injuries in the overhead athlete. Clin Orthop Relat Res. 2012;470(6):1579–85.
33. Scher S, Anderson K, Weber N, Bajorek J, Rand K, Bey MJ. Associations among hip and shoulder range of motion and shoulder injury in professional baseball players. J Athl Train. 2010;45(2):191–7.
34. Robb AJ, Fleisig G, Wilk K, Macrina L, Bolt B, Pajaczkowski J. Passive ranges of motion of the hips and their relationship with pitching biomechanics and ball velocity in professional baseball pitchers. Am J Sports Med. 2010;38(12):2487–93.
35. Laudner KG, Moore SD, Sipes RC, Meister K. Functional hip characteristics of baseball pitchers and position players. Am J Sports Med. 2010;38(2):383–7.
36. Boykin RE, McFeely ED, Ackerman KE, Yen YM, Nasreddine A, Kocher MS. Labral injuries of the hip in rowers. Clin Orthop Relat Res. 2013; 471(8):2517–22.
37. Harrast MA, Colonno D. Stress fractures in runners. Clin Sports Med. 2010;29(3):399–416.

38. Ropiak CR, Bosco JA. Hamstring injuries. Bull NYU Hosp Jt Dis. 2012;70(1):41–8.
39. Askling CM, Tengvar M, Saartok T, Thorstensson A. Acute first-time hamstring strains during high-speed running: a longitudinal study including clinical and magnetic resonance imaging findings. Am J Sports Med. 2007;35(2):197–206.
40. Lachiewicz PF. Abductor tendon tears of the hip: evaluation and management. J Am Acad Orthop Surg. 2011;19(7):385–91.
41. Carey T, Key C, Oliver D, Biega T, Bojescul J. Prevalence of radiographic findings consistent with femoroacetabular impingement in military personnel with femoral neck stress fractures. J Surg Orthop Adv. 2013;22(1):54–8.
42. Taylor-Haas JA, Paterno MV, Shaffer MD. Femoral neck stress fracture and femoroacetabular impingement. J Orthop Sports Phys Ther. 2011;41(11):905.
43. Kuhn KM, Riccio AI, Saldua NS, Cassidy J. Acetabular retroversion in military recruits with femoral neck stress fractures. Clin Orthop Relat Res. 2010;468(3):846–51.
44. Lee CH, Huang GS, Chao KH, Jean JL, Wu SS. Surgical treatment of displaced stress fractures of the femoral neck in military recruits: a report of 42 cases. Arch Orthop Trauma Surg. 2003; 123(10):527–33.
45. Chumanov ES, Heiderscheit BC, Thelen DG. Hamstring musculotendon dynamics during stance and swing phases of high-speed running. Med Sci Sports Exerc. 2011;43(3):525–32.
46. Domb BG, Linder D, Sharp KG, Sadik A, Gerhardt MB. Endoscopic repair of proximal hamstring avulsion. Arthros Tech. 2013;2(1):e35–9.
47. Dierckman BD, Guanche CA. Endoscopic proximal hamstring repair and ischial bursectomy. Arthros Tech. 2012;1(2):e201–7.
48. Gamboa JM, Roberts LA, Maring J, Fergus A. Injury patterns in elite preprofessional ballet dancers and the utility of screening programs to identify risk characteristics. J Orthop Sports Phys Ther. 2008;38(3):126–36.
49. Lee KS, Rosas HG, Phancao JP. Snapping hip: imaging and treatment. Semin Musculoskelet Radiol. 2013;17(3):286–94.
50. Anderson SA, Keene JS. Results of arthroscopic iliopsoas tendon release in competitive and recreational athletes. Am J Sports Med. 2008;36(12):2363–71.
51. Idjadi J, Meislin R. Symptomatic snapping hip: targeted treatment for maximum pain relief. Phys Sports Med. 2004;32(1):25–31.
52. Zoltan DJ, Clancy Jr WG, Keene JS. A new operative approach to snapping hip and refractory trochanteric bursitis in athletes. Am J Sports Med. 1986; 14(3):201–4.
53. Dezawa A, Kusano S, Miki H. Arthroscopic release of the piriformis muscle under local anesthesia for piriformis syndrome. Arthroscopy. 2003;19(5):554–7.
54. Hwang DS, Kang C, Lee JB, Cha SM, Yeon KW. Arthroscopic treatment of piriformis syndrome by perineural cyst on the sciatic nerve: a case report. Knee Surg Sports Traumatol Arthros. 2010; 18(5):681–4.
55. Turner R, O'Sullivan E, Edelstein J. Hip dysplasia and the performing arts: is there a correlation? Curr Rev Musculoskelet Med. 2012;5(1):39–45.
56. Bronner S, Brownstein B. Profile of dance injuries in a Broadway show: a discussion of issues in dance medicine epidemiology. J Orthop Sports Phys Ther. 1997;26(2):87–94.
57. Jacobson JA, Bedi A, Sekiya JK, Blankenbaker DG. Evaluation of the painful athletic hip: imaging options and imaging-guided injections. AJR Am J Roentgenol. 2012;199(3):516–24.

Rehabilitation of Non-Operative Hip Conditions

12

Molly A. Malloy, Ryan H. Wood, and Philip J. Malloy

Contents

M.A. Malloy (✉) • R.H. Wood
The University of Chicago Medical Center, Chicago, IL, USA
e-mail: Molly.malloy@uchospitals.edu; Ryan.Wood@uchospitals.edu

P.J. Malloy
Program in Exercise Science, Department of Physical Therapy Graduate Research Assistant, Marquette University, Milwaukee, WI, USA
e-mail: Philip.malloy@marquette.edu

Abstract

In recent years, the recognition of intra- and extra-articular sources of hip pain and the understanding of the interaction between these conditions have led to improved diagnosis of these pathologies. This improved understanding has created a need for rehabilitation guidelines to appropriately manage these conditions and prevent reoccurring symptoms. Extra-articular hip pathologies include tendon injuries, greater trochanteric pain syndrome, internal and external snapping hip syndromes, hamstring injuries, athletic pubalgia, osteitis pubis, and adductor muscle strains. Intra-articular pathologies include femoroacetabular impingement, labral tears, chondral lesions, and osteoarthritis. The purpose of this chapter is to present guiding principles for conservative rehabilitation of hip pathologies. The rehabilitation guidelines discussed take into account structural, biomechanical, and physiologic factors that affect injury and healing.

Introduction

Hip injuries can give rise to considerable functional limitations, and most individuals will benefit from organized rehabilitation to return to optimal function. The most common hip injuries that are managed conservatively are extra-articular pathologies. Interventions for these injuries involve managing pain, reducing

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_12

inflammation, regaining dynamic stability, and normalizing movement of the joint. In recent years, considerable research has investigated diagnosis and management of intra-articular hip pathology. Surgery aimed at promoting joint preservation by addressing intra-articular pathologies has been shown to have high success rates at returning individuals to their prior functional levels [1, 2]. Rehabilitation guidelines have been extensively described for patients following hip surgery, and similar progressions have been applied when treating these pathologies nonoperatively [3–6].

Establishing conservative rehabilitation guidelines is of considerable importance in the management of hip pathology. Factors that may cause or contribute to hip injury can be classified as modifiable or non-modifiable. A non-modifiable pathology describes a deformity involving the osseous structures or soft tissue of the hip and is often associated with underlying biological, developmental, or genetic factors. These conditions result in non-modifiable movement compensations that require adaption to function over time. Rehabilitation may be effective to manage symptoms of these hip injuries, although the underlying structural contribution to injury will not be changed through conservative management. Some evidence suggests that conservative management for subtle anatomical abnormalities, such as femoroacetabular impingement (FAI), may be effective at restoring function, although the evidence is limited [7]. It has been shown that patients with signs and symptoms of prearthritic and intra-articular hip disorders, who were treated with physical therapy, demonstrated significant improvement in pain and functioning from baseline to 1 year. Forty-four percent of the patients benefited with conservative treatment alone [8]. This data suggests that a trial of conservative physical therapy management for persons with prearthritic, intra-articular hip disorders should be considered before engaging in surgical intervention. [8]. Most frequently, hip rehabilitation is directed at conditions that are associated with modifiable factors. Specific modifiable deficits include abnormal mobility, flexibility, strength, neuromuscular control, endurance, power, agility, and skill.

It is beneficial when formulating rehabilitation guidelines to look at what has been effective conservative management with the other joints of the body. Commonalities have been found between the shoulder and the hip joints due to the underlying ball and socket structural similarities. Parallels in the soft tissue structures have been reported comparing the gluteus medius tendons to the rotator cuff tendons as both tissues function as abductors and external rotators [9–11]. Injury to these structures can be commonly insidious and degenerative in nature [9, 12]. Parallels also exist with rehabilitation progressions, with emphasis on proximal closed-chain stabilization of the scapula and pelvis. It has been found that most patients with hip injury improve with pelvic stabilization prior to initiation of open-chain hip flexor or abductor strengthening. This is due to the control required to accommodate the forces that are generated across the hip during open-chain activities, which is similar to the shoulder joint complex.

Thorough evaluation to determine if a patient's impairments are related to extra-articular versus intra-articular pathology should guide the initial plan of care. Often intra-articular and extra-articular pathologies coexist; therefore, treatment plans must be based on the individual needs of each patient. The challenge lies within where to direct treatment initially if pathologies coexist. Patients with hip pathologies, regardless of the structures involved, can benefit from physical therapy to reduce soft tissue inflammation, decrease pain, improve muscle firing patterns, and prevent advancement of the present disease process. The purpose of this chapter is to discuss the guidelines for conservative management of extra-articular and intra-articular hip pathologies. It is important for clinicians to recognize that in the instance of both intra- and extra-articular involvement, the extra-articular tissues should be addressed first to assist in ruling out underlying intra-articular involvement.

Extra-articular Pathology

Overuse Tendon Injuries

Over 24 tendons cross the hip joint with the purpose of moving the trunk and lower extremity in all planes of motion. Due to the intricacies of these motions and forces, overuse injuries are common. Hip tendon injuries occur when the structure becomes overused with excessive tensile loading and subsequently breaks down [13]. Tendinopathies are considered overuse injuries and frequently occur when a person increases their training routine without allowing adequate healing time between sessions. Repeated micro-injuries occur when a tendon is not given adequate time to heal between bouts of exercise or training and may result in more extensive injuries [13]. Overloading the tendons beyond their tensile threshold is another mechanism of injury. This can happen with a single session of increased activity especially if sufficient underlying strength is lacking. Structural factors including malalignment of the lower extremity, leg length discrepancy, excessive body mass index, and generalized ligamentous laxity may contribute to overuse and overload injuries as well. Other modifiable factors, which may predispose one to tendon injury, include deficits of strength, joint mobility, flexibility, endurance, and motor control [14].

The healing phase of the injury needs to be considered when treating patients with tendon injury. Tendonitis refers to the acute phase of the injury with patients demonstrating symptoms of inflammation such as pain, circulatory changes, edema, and tissue temperature elevation [14, 15]. Treatment for acute tendonitis includes managing the inflammatory response and addressing the factors that contribute to the sustained injury. If acute management fails, the overall presentation will lead to tendinosis. Recognition of acute versus chronic tendinopathy is necessary for successful rehabilitation. Progressions for the treatment guidelines for specific tendon injuries will be discussed in detail according to region of the hip involved.

Anterior Hip Overuse Disorders

The most common structures involved in anterior hip overuse syndromes are the hip flexor tendons. These patients will exhibit anterior hip pain, which tends to increase with activity. The common pain distribution involves the attachment sites of the iliopsoas, rectus femoris, or sartorius muscles. Patients may complain of pain with functional activities requiring active hip flexion such as the swing phase of gait, rolling in bed, or crossing their legs in sitting. Patients also frequently experience pain when the hip flexors are eccentrically loaded and stretched during the terminal stance phase of gait. In standing the patient may demonstrate reduced hip extension [16]. Examination may reveal weakness of the hip flexors and of the antagonist hip extensor and external rotator musculature. Hypertonicity, defined as palpable firmness and tenderness of the muscle tissue, is usually present in both the hip flexors and hip adductor musculature. Many patients will also demonstrate poor abdominal control, which may be observed as an inability to stabilize the pelvis and lower trunk in neutral while performing open kinetic chain lower extremity motions.

Conservative management of hip flexor tendinopathy should focus on decreasing irritation of the anterior hip. Interventions can include manual therapy techniques, use of pain relieving modalities, and regaining normal range of motion into hip extension with joint mobilizations. Patients will experience pain relief when they alternate between sitting and standing work stations and adjusting car seat positions in order to avoid increased hip flexion angles for extended periods of time. Clinicians should also assess if the hip flexor musculature is adaptively shortened versus hypertonic. Patients may demonstrate full hip extension range of motion but have increased tone and tenderness with palpation to the hip flexor muscles. Stretching a muscle, which has full length, but increased tone, may aggravate the condition. If a patient has increased hip flexor tone without limited extension motion, these patients also often demonstrate weakness of the gluteal muscles. Once control of the antagonist extensor and abductor muscle groups is regained, the increased tone of the hip flexors typically normalizes. The initial phase of strengthening for the gluteal musculature is a supine bridging series. Encourage full hip extension and minimal hip rotation into the bridge position to facilitate lengthening of the hip flexors with abdominal control. Additional progressions of strengthening include prone hip extension exercises, squat progressions from double to single leg while maintaining pelvic alignment, side plank series, and resistive band retro walking. Therapist cueing should promote level pelvic alignment for all single leg positions and encourage activation of gluteals into full extension without compensations into lumbar extension.

Next, strength and endurance of the hip flexor and pelvic stabilizer musculature should be addressed. During initial strengthening of the hip flexors, it is recommended to work the hip flexors eccentrically by extending the trunk in a seated position. One should cue the patient to engage the abdominals to maintain lumbar neutral posture while maintaining feet on the floor [17] (Fig. 1). Supine walkouts on a therapy ball are an exercise progression that encourages dynamic control of the hip flexors (Fig. 2). This position allows the hip flexors to lengthen eccentrically and encourages initiation of gluteal control to maintain the hip extended position. It is important to retrain this muscle through multiple methods of strengthening due to the dual role of the psoas muscle group with the proximal and distal fibers working as both postural stabilizers of the lumbopelvic region and prime movers for hip flexion [18]. Long lever open kinetic chain hip flexion exercises including straight leg raises and side leg raises are not recommended for these patients, secondary to the increased forces put through the hip in these positions [19, 20]. Long lever open kinetic chain exercises may cause more inflammation and pain especially if performed without adequate pelvic control or when tissue irritation is present.

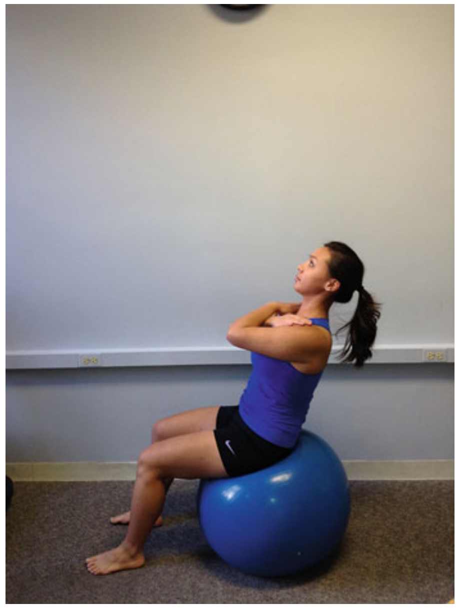


Fig. 1 Trunk lean

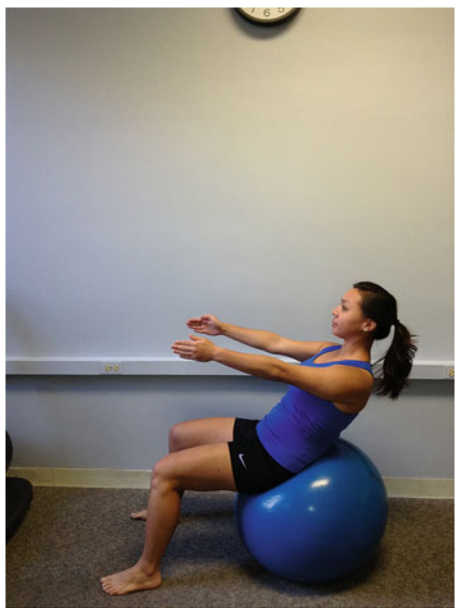


Fig. 2 Supine walkouts on ball

Greater Trochanteric Pain Syndrome

Greater trochanteric pain syndrome (GTPS) includes a number of disorders of the lateral hip, including trochanteric bursitis, tears of the gluteus medius and minimus muscles, iliotibial band syndrome, and external coxa saltans (snapping hip) [21]. Patients typically present with pain and tenderness in the region of the greater trochanter, buttocks, or lateral thigh. During clinical examination, passive range of motion often causes minimal pain, while active motions of the hip tend to be more painful. Muscle length assessments may show adaptive shortening especially of the hip abductor and hip flexor muscles. Weakness of the hip abductor muscles is common, and it is recommended to assess the patient's strength in both open- and closed-chain positions. Caution should be used to not overload joint structures if testing in long lever side-lying positions. Single limb stance may reveal a hip drop (Trendelenburg sign) (Fig. 3) or excessive elevation of the contralateral pelvis (compensated Trendelenburg sign) [22]. During gait assessment, one may observe movement compensations in all planes of motion but most commonly in the frontal plane. Patients often excessively rotate through the pelvis and lumbar spine in order to compensate for limited extension of the hip, especially noted during the late to terminal stance phases of gait.

Conservative management of GTPS starts with decreasing irritation of the lateral hip with manual therapy techniques, pain relieving modalities, and regaining normal range of motion. Manual techniques should include stretching into hip adduction, internal rotation and extension, cross friction mobilization to the gluteal tendons, and deep tissue techniques to the lateral hip musculature and tensor fasciae latae. Once acute irritation has been addressed and range of motion normalized, the clinician needs to assess the biomechanics of the trunk and lower extremity. Reduced neuromuscular control of the trunk commonly causes compensatory movements, throughout the lower extremity chain; therefore, core stabilization is very important for these patients. Exercise progressions to address these muscle imbalances include strengthening the hip muscles in the closed chain position using verbal, tactile, and/or demonstrative cues on pelvic, trunk, and lower extremity position to encourage appropriate activation patterns. Initial dynamic activities include gait retraining exercises and dynamic multiplane progressions. Other dynamic activities may include active pelvic depression and hip hiking standing on the involved side (Fig. 4). Appropriate hip strength progressions include supine bridging series, side plank series, and squats progressing from double to single leg focusing on control throughout the entire movement [23].

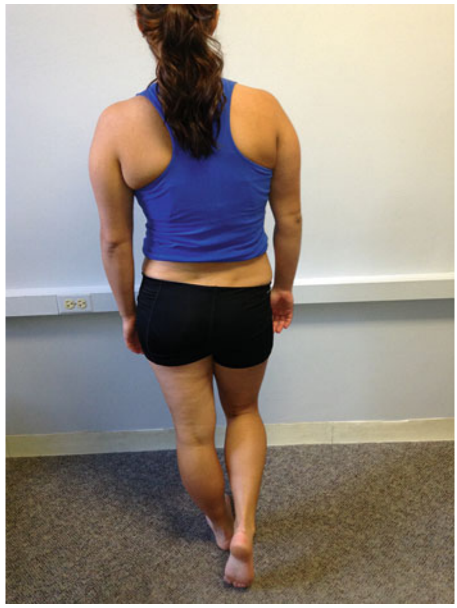


Fig. 3 Trendelenburg

Internal and External Snapping Hip Syndromes

Internal snapping hip syndrome results from the iliopsoas tendon snapping over the iliopectineal eminence of the femoral head as the femur moves from a position of flexion to extension. External snapping hip syndrome results from the iliotibial band snapping over the prominence of the greater trochanter while actively moving the thigh into flexion and then into extension [23]. These

Fig. 4 Hip hiking

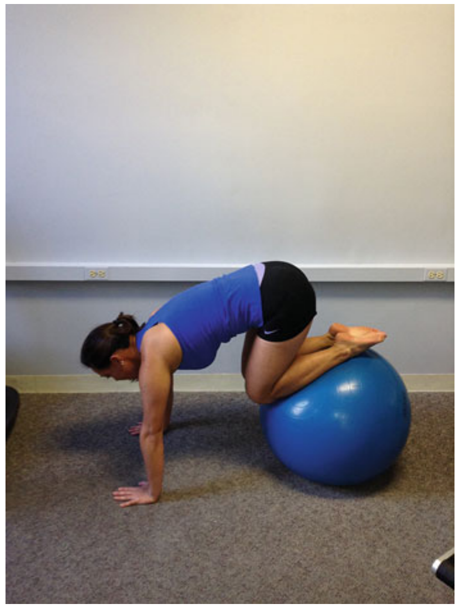


Fig. 5 Walkouts to ski tuck

syndromes are more prevalent in a younger population and typically occur between the ages of 15–40 years. Imbalance between the agonist and antagonist muscle groups of the hip and trunk often precedes this injury, and commonly patients demonstrate weakness or improper firing of the core musculature with functional movements.

Passive range of motion of these patients is commonly unremarkable. Mechanical symptoms are usually only reproduced with active movement, and external snapping can be reproduced in standing by moving the pelvis into hip adduction [23]. Initial treatment includes addressing muscular asymmetries of the deep abdominals, iliopsoas, and hip abductor muscles. It is critical if the patient demonstrates transverse abdominus weakness to retrain to maintain pelvic neutral with all movements of the pelvis over the femur. Initial core exercises consist of eccentric training of the hip flexors and abdominals by performing trunk leans with the feet on the floor while sitting on a stool or therapy ball. Progress the core exercises by adding short lever-arm lower extremity exercises such as bent knee flexion and small lower extremity movements while cueing the patient to maintain a neutral lumbar position by recruitment of the deep abdominals. Strengthening is progressed to involve more challenging abdominal tasks including plank progressions, prone walkouts on the therapy ball to tuck positions (Fig. 5), and abdominal exercises with the lower extremities lifted. For all strengthening exercises, it is required to cue the patient to maintain lumbar neutral with strong abdominal and gluteal control.

Hamstring Injuries

Injuries to the hamstring muscle and tendons can occur traumatically from an eccentric muscle overload at the end range of hip flexion with the knee in a position of extension or chronically through overuse. It is important for clinicians to fully identify the mechanism of injury in order to structure treatment interventions to address the underlying contributing impairments.

An acute hamstring muscle or tendon injury often results in ecchymosis of the posterior thigh and leg. Severe swelling and ecchymosis may indicate a torn proximal hamstring tendon and should be evaluated with an MRI. The indications

Fig. 6 Hamstring bridge on reformer

for proximal hamstring repair are rupture two or more tendons and/or greater than two centimeters of displacement from the ischial tuberosity. In general, orthopedic surgeons want to repair within 2 weeks to minimize risk to adhesions to nearby structures (i.e., sciatic nerve). Examination will reveal shortening of the hamstring musculature with pain and muscle guarding. Palpation will clarify the exact location of the tear. In severe cases, reduced hamstring strength when compared to the opposite side will be present.

Conservative management of acute hamstring injury includes addressing ecchymosis and edema through soft tissue massage, compression, and modalities such as electrical stimulation and pulsed ultrasound. Once the patient is out of the acute phase, 3–5 days post injury, gentle passive range of motion into hip flexion and extension may be initiated, followed by isometric strengthening in a closed-chain position. Caution should be taken in the acute phase to gently stretch the lower extremity to end range hip flexion while maintaining knee flexion to minimize tension through the proximal hamstring tendons.

Chronic proximal hamstring tendinopathy occurs as a result of repetitive microtrauma. Often these patients have a history of recurrent hamstring injury and underlying neuromuscular imbalance of the pelvis, trunk, or lower extremities. Patients will complain of a deep ache in the posterior hip and buttocks, which is commonly exacerbated with activity. Hip extension and knee flexion weakness and pain with palpation to the ischial tuberosity may be present. Passively moving the patient's hip and knee into full flexion and then slowly extending the knee will also reproduce the patients pain in the posterior buttocks.

Conservative management of chronic proximal hamstring injury should focus on soft tissue mobilization to the hamstring insertion at the ischial tuberosity. Initial strengthening exercises include hip extension bridges on a stable surface progressing to an unstable surface such as a therapy ball. The use of a pilates reformer for isolated and dynamic movements such as double leg hamstring curls and single leg marching while maintaining a bridge position is also an effective strengthening progression (Figs. 6 and 7). Additional exercises include standing double and single leg dead lifts with the knees in multiple angles of flexion. These exercises promote activation of the hamstring muscle across both the hip and knee joints simultaneously. It is important to note that because the hamstring is a two joint muscle, maximal stress is exerted in full knee extension and hip flexion. Multiplane strengthening in this lengthened position should precede the progression to jumping, landing, agilities, and cutting drills. When starting these drills, cueing the patient to land and slowly flex into the squat

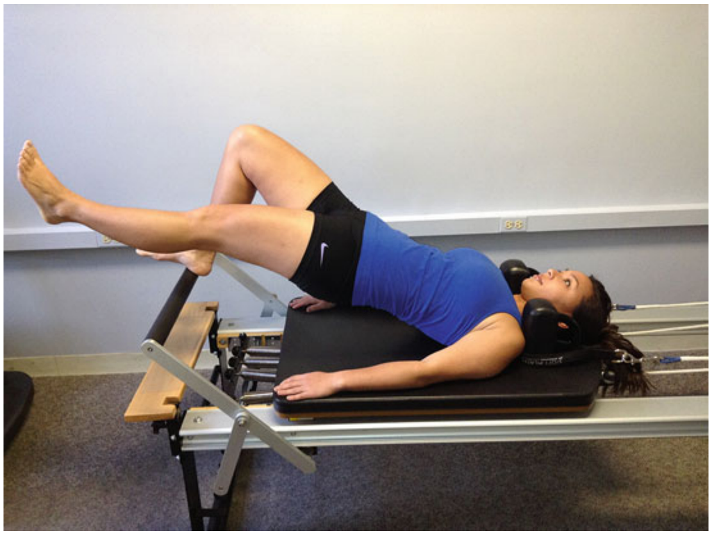


Fig. 7 Hamstring bridge with marching

position will retrain the involved hamstring muscle eccentrically and will encourage equal weight bearing through both extremities.

Athletic Pubalgia and Osteitis Pubis

Pelvic pain syndromes as a source of injury around the hip have been found to affect 5–18 % of athletes involved in competitive sports [24]. Athletic pubalgia, or "sports hernia," causes groin pain in the absence of an actual hernia. The etiology is an overuse injury related to repetitive rotation and commonly occurs during sports, such as hockey and tennis. Groin pain usually arises insidiously with an occasional patient report of feeling a tearing sensation in the groin. Underlying muscle imbalances in the trunk and pelvis lead to abnormal force transfers across the pelvis and lead to soft tissue tearing and injury. Initially, symptoms are reproduced only during high-level function, but as the severity progresses, pain may be reproduced during routine activities of daily living.

Osteitis pubis is defined as a separation of the pubic symphysis resulting from a series of repetitive twisting, cutting, and/or pivoting movements. Often this occurs in conjunction with frequent acceleration and deceleration that are necessitated in sports such as soccer, football, rugby, swimming, and hockey. During these repetitive movements, one can develop chronic insertional tendinosis of the adductor mass at the pubis. Weakness and poor neuromuscular control of the abdominals combined with adaptive shortening and/or hypertonicity of the adductor muscles may cause instability of the pubic symphysis [24]. These microtraumas can lead to symptom reproduction over the symphysis, medial groin, abdomen, and/or scrotum. Symptoms can be provoked with resisted adduction of the thigh, dynamic single leg activities, or dynamic abdominal activities such as a traditional sit-up [25].

Initial treatment of both athletic pubalgia and osteitis pubis should focus on minimizing aggravating positions until symptoms subside. Avoidance of lower extremity cutting, pivoting, and dynamic abdominal activities are important to allow proper healing of the pubic symphysis and tendinous insertions to the pelvis. Manual therapy can be utilized to correct any malalignment of the pelvis, decrease pain, improve muscle length, and to decrease abnormal muscle tone. Isometric strengthening can be initiated with emphasis on transverse abdominus, rectus femoris, gluteus medius, and hip adductor musculature when a patient tolerates. Once pain has decreased, progression into straight plane and subsequent multiplane strengthening should be initiated. These exercises should be performed within pain-free ranges of motion and intensity with cueing to maintain pelvic alignment. Higher-level core stabilization exercises such as planks, side planks, hip extension bridges, and trunk leans while sitting on a therapy ball are recommended as exercises which strengthen the abdominals without excessive activation of the hip flexors and adductor muscles. Often abdominal strengthening exercises in which the lower extremities are off of the ground cause more irritation to the healing pubic bones. This may be due to an increase in shearing across the pubic symphysis if imbalances are present between the agonist and antagonists muscle groups. At all times clinicians need to be assessing pubic bone and groin pain and modify the exercises if any activities or positions are painful. Once straight plane stability is established and the athlete is no longer having pain, higher-intensity training may be initiated. Sport-specific training of cutting, pivoting, kicking, skating, and jumping should always focus on maintaining proper form through the trunk and pelvis. The patient's training program, even after discharge from therapy, should always include maintaining abdominal and core strength.

Adductor Muscle Strains

Muscle strains of the adductors can occur traumatically from an excessive eccentric activation of the muscle during a slip of the planted foot while the body moves in an opposite lateral direction. This can also occur when pivoting on a planted foot and forcefully rotating the pelvis. The patient will often feel an immediate pull or strain through the groin with pain that may radiate down to the knee. Overuse adductor injuries occur from repeated contraction of the adductor muscles to stabilize the pelvis when there is limited motion of the opposite hip or from muscle imbalances in the pelvis. Initial rehabilitation includes manual therapy to reduce pain and swelling. Gentle stretching and hip range of motion can be performed within pain-free ranges during the first few weeks of rehabilitation. Once swelling and gentle mobility are regained, closed-chain strengthening may be initiated. Double leg squats, hip extension bridge series, and balance retraining

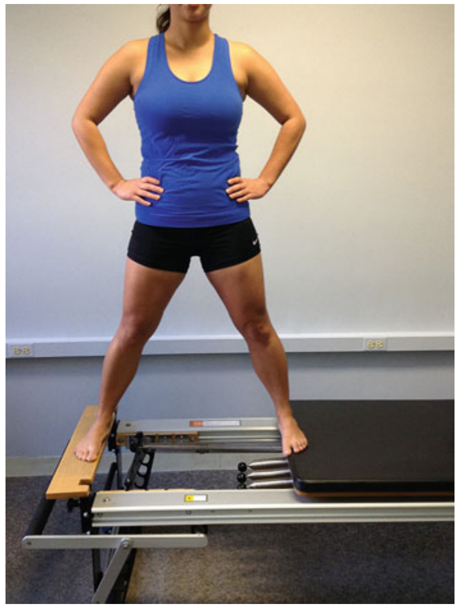


Fig. 8 Skater series knees extended

are all appropriate initial exercises. When sagittal plane motion is pain-free, the clinician can introduce lateral hip strengthening being sure to work the injured leg in both the open and closed chain. Initial lateral strengthening includes skater series exercises on a pilates reformer or slide board (Figs. 8 and 9), side stepping with a theraband at varied degrees of hip flexion, and progressing to lateral stepping over a step or balance board. Healing of injured muscles can take a number of weeks or months, and it is important to not push an athlete back into high-level activity prior to full healing. Use pain and strength as a guideline for the overall progression of return to full function.

Intra-articular Pathologies

The most common intra-articular hip injuries that can benefit from nonoperative rehabilitation include chondral defects, hip osteoarthritis, femoroacetabular impingements, and labral tears. When treating a patient with an underlying abnormal tissue or boney morphology, clinicians need to be cautious to not exacerbate symptoms due to the non-modifiable nature of these contributors to injury. To be successful with rehabilitation, it is

Fig. 9 Skater series side lunge

crucial to understand which positions, movements, and functional daily activities will likely increase joint irritation and exacerbate the underlying pathology. The role of the physical therapist is to educate the patient on how to modify activity to accommodate the underlying pathoanatomy. Many patients with intra-articular hip conditions can lead an active healthy lifestyle with minimal pain if they are taught a few modifications to reduce stress across involved hip joint tissues. Teaching patients the importance of supportive and shock absorbing shoe wear for both exercise and work can immediately decrease joint pain especially in the osteoarthritic population. Additionally, it may be beneficial to instruct patients to select low impact exercises such as swimming, biking, and walking. Balance exercises and improving lower extremity proprioception are important to improve overall function. Encouraging hiking with walking poles or using an assistive device in the community can greatly benefit patients and decrease forces through the lower extremities. These lifestyle modifications in addition to reduction in body weight may decrease joint pain and minimize subsequent extra-articular irritation of the muscles surrounding the joints due to increased demands with daily function.

Chondral Injury and Osteoarthritis

A multitude of factors including biochemical, biomechanical, and genetic abnormalities may contribute to osteoarthritis of the hip. Although the patho-mechanism of degeneration affecting the dysplastic hip is well understood, the exact pathogenesis for idiopathic osteoarthritis has not been established [26–28]. Researchers have demonstrated a correlation between osseous abnormalities of the acetabulum and femur and intra-articular hip injury including acetabular labral tears and osteoarthritis [26–30].

Acute chondral lesions resulting in a focal defect of the articular cartilage of the hip can occur during high-level activity such as sports or secondarily from a hip dislocation or subluxation. The common mechanical position for traumatic chondral injuries is rotation of the pelvis on a fixed femur under an axial load. Patients with chondral injuries will often complain of similar pain patterns of anterior or lateral hip pain, which increases with weight bearing, pivoting, and twisting positions. They may complain of mechanical symptoms such as clicking, locking, or catching if a loose body is present in the joint. Physical examination may reproduce pain with passive hip flexion and rotation and during a resisted straight leg raise.

Patients with moderate or severe arthritis will also demonstrate limited mobility commonly in flexion, internal rotation, external rotation, and abduction. Subjective complaints include pain with weight bearing, sitting on a low surface, difficulty crossing their legs, tying their shoes, and with getting into low cars. When assessing gait, patients with unilateral hip osteoarthritis may lean their trunk and pelvis toward the side of the painful hip during single limb support, described as uncompensated Trendelenburg [22]. This is done as a joint protection mechanism to reduce force across the hip joint that is created during contraction of the hip abductors. Muscular assessment often shows weakness of the hip abductor, abdominal, and hip extensor musculature [31].

Initial treatment for patients with the disease spectrum from chondral defects to severe arthritis begins with educating the patient in methods to reduce the forces across the hip. This education is applied in order to attempt to prevent excessive hip joint reaction forces that can exacerbate an acute condition and/or predispose further cartilage deterioration [22]. Education includes teaching the patient about decreasing body weight and encouraging activities and exercises that minimize forces through the hip. This can be done by normalizing gait, promoting the use of a cane, and performing low impact activities such as biking and aquatic therapy for exercise. Patients should be educated with regard to which positions may aggravate the chondral defect or hip OA including to avoid pivoting on the involved leg and deep squatting.

Addressing the limitations in ranges of motion through stretching, manual tissue mobilizations, and joint mobilizations is indicated. Clinicians should address joint and tissue restrictions and then stabilization and strengthening in the new available ranges of motion. Long axis joint distraction, lateral distraction, and inferiorly directed mobilizations are also beneficial for restoring range of motion. It is important to educate patients to first perform prescribed mobility and flexibility exercises prior to strengthening and stabilization when performing a home exercise program. Beneficial home joint mobility exercises include hands and knees rocking and teaching family members how to perform long axis distraction. It is important to teach patients not to push into pain during mobility and flexibility exercises. Although the goal is to increase available range of motion and muscle length, joint irritation and pain should always be avoided. Once mobility has been improved, closed-chain strengthening is initiated. It is recommended to start with body weight resistance progressing to single leg activities. Functional strengthening activities include double leg squatting, single leg balance squats, single leg squats, high-level balance activities, and the bridging hip extensor series all while maintaining neutral pelvic alignment core strengthening (Figs. 10, 11, and 12). Cardiovascular exercises can include walking on an inclined treadmill, biking with resistance, and swimming. It is important to always assess how the patient feels following treatment to assure that the progression of exercises is not too irritating.

Fig. 10 Balance squat

Fig. 11 Single leg squat

Fig. 12 Single leg balance on BOSU

Femoroacetabular Impingement

Femoroacetabular impingement (FAI) is a condition where abnormal boney contact results from a subtle abnormal morphology of the proximal femur and/or the acetabulum. These patients typically have otherwise normal or near normal anatomy of the hip joint [27, 28]. The types of FAI that have been described are cam type, which arises from a lack of offset at the head and neck junction of the femur, or pincer type, which occurs on the acetabular side of the hip and results in global over-coverage associated with coxa profunda or focal/isolated over-coverage as a result of acetabular retroversion or an os acetabuli [28]. Abnormal boney contact occurs during normal ranges of motion, most commonly flexion and internal rotation [27]. Conversely boney impingement between the femoral neck and acetabular rim can occur in individuals with normal morphologic features if they engage in activities that require supraphysiologic ranges of motion as observed during figure skating or ballet [32]. Although surgery is indicated for many of these patients, at times it is not indicated secondary to other comorbidities. In patients for whom surgery is not an option, optimum conservative management has yet to be established. Physical therapy may be beneficial for both groups of patients to learn how to accommodate their abnormal structure within the context of their individual functional demands [7].

Femoroacetabular impingement is commonly diagnosed in young active individuals with their initial complaint usually being an insidious onset of groin pain in the absence of an episode of acute trauma. Pain may be intermittent and exacerbated by sitting, walking, running, or performing in their specific sport. Clinical examination often reveals limitation of motion, particularly with internal rotation, flexion, and adduction [27]. Limitations in strength and muscle flexibility will depend on duration of symptoms, extra-articular involvement, and the severity of the FAI.

Initial management for patients with FAI includes education on avoiding positions that may cause hip pain and joint irritation. Modifications to activities of daily living may be required to avoid symptom exacerbation from engagement of the boney impingement. If exacerbating positions are unavoidable during required function, then encouraging exercises such as gentle stretching or self-mobilization may be beneficial in assisting with symptom management. Additionally, it is important for clinicians to avoid engaging these positions when attempting to reestablish range of motion during treatment. It is recommended to address flexibility of the surrounding hip musculature in the presence of adaptive shortening. For example, shortening of the hamstrings may pull the pelvis into a position of posterior tilt, which results in a position of flexion at the hip joint. In the presence of impingement, this may exacerbate anterior symptoms due this biased hip flexion position.

In the cases of functions that require supraphysiologic ranges of motion such as ballet dancing, figure skating, and hockey goal tending, teaching limitations to end ranges of motions and modifications of activities is warranted. Emphasis should be placed on pelvic and trunk control to prevent impingement due to poor proximal stability. It is beneficial to strengthen the hip abductors and extensor muscles while maintaining adequate core control and moving through mid ranges of hip motion as these are the most common during daily functions. Encouragement of strengthening through an aquatic program of aqua-jogging and flutter kicking can also allow cardiovascular training without movement into impingement positions while maintaining cardiovascular endurance.

Labral Tear

Mechanical impingement and instability of the hip joint are believed to be common causes of labral pathology [3, 33–36]. Athletic activities that involve repetitive pivoting movements or repetitive hip flexion are now recognized as additional causes of acetabular labral injury [37, 38]. Clinical signs of FAI have been found to be present in up to 95 % of patients with labral tears [39]. Lesions of the chondrolabral junction may also result from atraumatic hip instability with or without mechanical impingement [3]. While the postoperative rehabilitation guidelines following a labral repair have been established, limited research is present for patients who are managed conservatively [3]. Treatment of individuals with a labral tear secondary to femoroacetabular impingement should follow the rehabilitation guidelines associated with FAI. However, individuals with atraumatic hip instability as the cause of acetabular labral injury should be managed differently. It is important to note that these individuals may demonstrate excessive motion not only at the hip but also in other joints of the body. The labrum is important for overall hip joint stability, and therefore, the chondral and ligamentous structures can come under additional stress once a labral tear occurs. Greater stress is placed across articular cartilage and with a loss of the suction seal from a torn labrum can lead to greater movement at the joint [3].

The patient history of individuals with labral tears will commonly include complaints of groin pain with radiation of pain into the thigh, buttocks, or lateral hip. The pain is most commonly reproduced with the combined motion of hip internal rotation, adduction, and flexion, especially if FAI is present. Conversely, patients with an isolated labral tear may complain of pain with extension and external rotation [40]. Patients may feel joint irritation with walking, stairs, impact activities, pivoting, and prolonged sitting. The pain is frequently described as intermittent but can become constant as the injury progresses. Some patients will complain of mechanical symptoms such as popping, clicking, or locking [39]. Range of motion testing typically reveals excessive external rotation. Strength should be assessed with particular attention being paid to the hip flexors, abductors, and extensors because of the role these muscles play in movement and stability of the hip and pelvis. Neuromuscular control of the pelvis and trunk should also be observed to determine if motor control is contributing to pain.

Conservative treatment of acetabular labral tears should focus on lumbopelvic stabilization, correction of hip muscular imbalances, and sport-specific functional progressions [3]. Initially, patients are treated with modalities for pain control, education in trunk stabilization, and correction of abnormal movement. Abnormal movements commonly observed include contralateral hip drop during the stance phase of gait or with single limb stance. This again is usually a result of hip abductor weakness and lack of trunk stability which both need to be addressed. Rehabilitation should focus on regaining full strength without placing the labrum at risk of further injury. Axial loading and rotating around a fixed femur places increased stress on the labrum; therefore, education on appropriate patterns of movement in a closed-chain position should be emphasized to prevent compensations that may create increased labral strain. Strength and stabilization exercises include gluteus medius strengthening through bridge series, side stepping, and retro-stepping with resistance (Fig. 13). Trunk stabilization and core control should be encouraged through plank progressions as well as side plank progressions, all in hip neutral positions. Lower extremity strength training should begin with double leg squatting and

Fig. 13 Retro gait with band

then progress to single leg squatting. Single plane stability should be the initial focus with the integration of multiplane exercises as symptoms allow. High-level balance activities using altered surfaces such as with a BOSU or wobble board are also beneficial and improve the neuromotor control of the deep stabilizer musculature of the entire lower extremity and trunk (Fig. 12). It is encouraged to use pain as a guide for all activities during rehabilitation for labral tears. Patients should not push through pain, as they could be making the tear worse.

If a patient desires to return to athletics, it is important to address which positions in their sport may worsen the condition. Follow through during throwing places the hip in a position of significant internal rotation thereby potentially straining the labrum in throwers. Figure skaters and dancers often require supraphysiologic motions in both open- and closed-chain positions, and therefore, many positions can be irritating. Hockey players, especially goalies, require excessive and repetitive rotation of the hips during play. Many of these athletes cannot completely avoid these rotational positions so the role of therapy is to help to establish appropriate muscular control into the aggravating positions. The main treatment goal for athletes should be to establish strength, muscular endurance, and neuromuscular control to meet the demand of their particular sport in order to minimize hip joint stress and slow injury progression.

Summary

Conservative rehabilitation for hip pathology can be effective at eliminating impairments and restoring function in many patients. Although conservative management is unable to modify structural factors that may contribute to pathology, specific manual therapies, exercise prescription, and patient education are often able to improve overall function. Clinicians should have a thorough understanding of hip structural anatomy and kinesiology in order to develop an appropriate treatment plan that takes into account the individual needs of each patient. Additionally, clear communication between the patient, physician, physical therapist and rehabilitation specialist will facilitate each patient to return to realistic function.

References

1. Nho SJ, Magennis EM, Singh CK, et al. Outcomes after the arthroscopic treatment of femoroacetabular impingement in a mixed group of high-level athletes. Am J Sports Med. 2011;39:14S–919.
2. Philippon MJ, Briggs KK, Yen YM, et al. Outcomes following hip arthroscopy for femoroacetabular impingement and associated chondrolabral dysfunction: a minimum two year follow-up. J Bone Joint Surg (Br). 2009;91:16–23.
3. Yazbek PM, Ovanessian V, Martin R, et al. Nonsurgical treatment of acetabular labrum tears: a case series. J Orthop Sports Phys Ther. 2011;41:346–53.
4. Wahoff MS, Ryan M. Rehabilitation after hip femoroacetabular impingement arthroscopy. Cin Sports Med. 2011;30:463–82.
5. Edelstein J, Ranawat A, Enseki KR, et al. Post-operative guidelines following hip arthroscopy. Curr Rev Musculoskelet Med. 2012;5:15–23.
6. Malloy P, Malloy M, Draovitch P. Guidelines and pitfalls for the rehabilitation following hip arthroscopy. Curr Rev Musculoskeletal Med. 2013;6(3):235–41.

7. Emara K, Samir W, Motasem EH, Ghafar KAE. Conservative treatment for mild femoroacetabular impingement. J Orthop Surg (Hong Kong). 2011; 19(1):41–5.
8. Hunt D, et al. Clinical outcomes analysis of conservative and surgical treatment of patients with clinical indications of pre-arthritic, intra-articular hip disorders. Am Acad Phys Med Rehab. 2012;l4:479–87.
9. Dwek MR. Imaging of the hip abductors: normal anatomy and commonly encountered pathology at the greater trochanter. Magn Reson Imaging Clin N Am. 2005;13:691–704.
10. Bunker T, Esler C, Leach W, et al. Rotator-cuff tears of the hip. Bone Joint J. 1997;79:618–20.
11. Kagan A. Rotator cuff tears of the hip. Clin Orthop Relat Res. 1999;368:135–40.
12. Robertson W, Gardner M, Barker J, et al. Anatomy and dimensions of the gluteus medius tendon insertion. J Arthroscopic Relat Surg. 2008;24:130–6.
13. Maganaris CN, Narici MV, Almekinders LC, Maffulli N. Biomechanics and pathophysiology of overuse tendon injuries: ideas on insertional tendinopathy. Sports Med. 2004;34:1005–17.
14. Kannus P. Etiology and pathophysiology of chronic tendon disorders in sports. Scand J Med Sci Sports. 1997;7:78–85.
15. Reynolds N, Worrell T. Chronic Achilles peritendinitis: etiology, pathophysiology, and treatment. J Ortho Sports Phys Ther. 1991;13:171–6.
16. Kennedy MJ, Lamontagne M, Beaule PE. Femoroacetabular impingement alters hip and pelvic biomechanics during gait: walking biomechanics of FAI. Gait Posture. 2009;30:41–4.
17. Edelstein J, Ranawat A, Ensenki K, et al. Post-operative guidelines following hip arthroscopy. Curr Rev Musculoskeletal Med. 2012;5:15–23.
18. Arbanas J, Klason G, Nikolic M, et al. Fibre type composition of the human psoas major muscle with regard to the level of its origin. J Anat. 2009; 215:636–41.
19. Rydell N. Biomechanics of the hip joint. Clin Orthop. 1973;92:6–15.
20. Crowninsheild RD, Johnston RC, Andrews JG, et al. A biomechanical investigation of the human hip. J Biomech. 1978;11:75–85.
21. Voos JE, Rudzki JR, Shindle MK, et al. Arthroscopic anatomy and surgical techniques for peritrochanteric space disorders in the hip. Arthroscopy. 2007; 23:124el–6e5.
22. Neumann DA. Biomechanical analysis of selected principles of hip joint protection. Arthr Care Res. 1989;2:146–55.
23. Ilizaliturri VM, Camacho- Galindo J, Ramirez A, Ibarra Y, McMillan S, Busconi BD. Soft tissue pathology around the hip. Clin Sport Med. 2011;30:391–415.
24. Weber MA, Rehnitz C, Ott H, et al. Groin pain in athletes. Fortschr Röntgenstr. 2013;185:1139–48. doi:10.1055/s-0033-1335304.
25. Leblanc KE, Leblanc KA. Groin pain in athletes. Hernia. 2003;7:68–71.
26. Harris WH. Etiology of osteoarthritis of the hip. Clin Orthop Relat Res. 1986;213:20–33.
27. Ganz R, Parvizi J, Beck M, Leunig M, Notzli H, Siebenrock KA. Femoroacetabular impingement: a cause for osteoarthritis of the hip. Clin Orthop Relat Res. 2003;417:112–20.
28. Ganz R, et al. The etiology of osteoarthritis of the hip. Clin Orthop Relat Res. 2008;466:264–72.
29. Wenger DE, Kendell KR, Miner MR, Trousdale RT. Acetabular labral tears rarely occur in the absence of bony abnormalities. Clin Orthop Relat Res. 2004;426:145–50.
30. Ito K, Leunig M, Ganz R. Histopathologic features of the acetabular labrum in femoroacetabular impingement. Clin Orthop Relat Res. 2004;429:262–71.
31. Loureiro A, Mills PM, Barrett RS. Muscle weakness in hip osteoarthritis: a systematic review. Arth Care Res. 2013;65:340–52.
32. Charbonnier C, Kolo FC, Duthon VB, Magnenat-Thalmann N, Becker CD, Hoffmeyer P, Menetrey J. Assessment of congruence and impingement of the hip joint in professional ballet dancers- a motion capture study. Am J Sports Med. 2011;39:557–66.
33. Beck M, Kalhor M, Leunig M, Ganz R. Hip morphology influences the pattern of damage to the acetabular cartilage: femoroacetabular impingement as a cause for early osteoarthritis. J Bone J Surg [Br]. 2005; 87:1012–8.
34. Guevara CJ, Pietrobon R, Carothers J, et al. Comprehensive morphologic evaluation of the hip in patients with symptomatic labral tear. Clin Orthop Relat Res. 2006;453:277–85.
35. Martin RL, Enseki KR, Draovitch P, Trappuzzano T, Philippon MJ. Acetabular labral tears of the hip: examination and diagnostic challenges. J Orthop Sports Phys Ther. 2006;36:503–15.
36. Philippon MJ, Zehms CT, Briggs KK, Manchester DJ, Kuppersmith DA. Hip instability in the athlete. Oper Tech Sports Med. 2007;15:189–94.
37. De Paulis F, Cacchio A, Michelini O, Damiani A, Saggini R. Sports injuries in the pelvis and hip: diagnostic imaging. Eur J Radiol. 1998;27 suppl 1:S49–59.
38. Mason JB. Acetabular labral tears in the athlete. Clin Sports Med. 2001;20:779–90.
39. Lewis CL, Sahrmann SA. Acetabular labral tears. J Orthop Sport Phys Ther. 2006;86:110–21.
40. Burnett RS, Della Rocca GJ, Prather H, Curry M, Maloney WJ, Clohisy JC. Clinical presentation of patients with tears of the acetabular labrum. J Bone Joint Surg Am. 2006;88:1448–57.

Rehabilitation of Post-Operative Hip 13

Dirk Kokmeyer and Jenna Hodge

Contents

Abstract

Postoperative rehabilitation after hip arthroscopy can present several challenges to the physical therapist. The rehabilitation process calls for a delicate progression of exercise prescription to restore mobility, gait, strength, and neuromuscular control and return to normal activity while preventing excessive anterior hip joint forces that can lead to chronic anterior hip pain. Postoperative complications include, pain, joint effusion, tissue edema, and muscle atrophy, which lead to impairments, such as pain, range of motion limitations, and muscle weakness and neuromuscular deficits during active hip movements. These impairments contribute to functional impairments, such as gait deficits, difficulties in performing activities of daily living, and the inability to perform recreational or sports activities. A criteria-driven, phased rehabilitation program is recommended to address these impairments, restore normal function, and return patients to unrestricted daily activity, recreation, and sports. Furthermore, using criteria rather than time-specific benchmarks offers patients a more individualized rehabilitation.

Introduction

The purpose of the chapter is to outline the post-operative rehabilitation after hip arthroscopy. Four phases of rehabilitation will be presented to

D. Kokmeyer (✉) • J. Hodge
Howard Head Sports Medicine, Vail Valley Medical Center, Vail, CO, USA
e-mail: mail@kokmeyer.com; Kokmeyer@vvmc.com; hodge@vvmc.com

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_13

include several strengthening progressions of musculature about the hip. The authors of this guide recommend criteria-driven rehabilitation guidelines rather than a time-specific program. This is more pragmatic because of the variability of postoperative parameters among surgeons, concomitant procedures, and anthropometric, demographic, cultural, and behavioral factors of the patient. Furthermore, the physical therapist should defer to evidenced-based medicine and impose discretion when determining whether to progress a patient.

Typically, patients will begin formal outpatient therapy during their first postoperative week. Phase 1 begins with interventions tailored to reduce pain, edema, and joint effusion. Passive range of motion exercises and gentle strengthening exercises are initiated. Phase 2 may be considered the most critical phase of the rehabilitation process because the transition to weight bearing yields complex neuromuscular impairments that facilitate gait deficiencies and subsequent overloading of the anterior joint. It may commence when the patient is cleared to begin weight-bearing activities, the outlined criteria are met, and the physical therapist deems that it is appropriate to progress.

The time at which a patient may begin phase 2 is variable. For instance, a patient undergoing a concomitant microfracture procedure on a weight-bearing portion of the joint may be required to restrict weight bearing for 6–8 weeks. This means that phase 2 may not begin until the 6th postoperative week. In other instances, it may be prudent to limit weight-bearing activity until a patient can actively control hip extension due to the increase in anterior joint forces this produces [1]. Phase 3 entails restoring strength neuromuscular control and returning a patient to functional activities. For many patients, this may be the final phase of the rehabilitation if the patient's functional goals do not include returning to athletic performance. Phase 4 is reserved for athletes who wish to return to their previous level of sports participation and encompasses sports-specific drills and a return to sports interval program.

Phase 1: Protection Phase

During phase 1, the guiding principles include (1) protecting the integrity of repaired tissue, (2) reduction of pain and inflammation and prevention of fibrosis, (3) restoring passive and active mobility within restricted ranges, and (4) restoring proper neuromuscular control patterns.

Protection of Repaired Tissue

It is important to identify the involved tissue and quality of the repaired tissue. This contributes to the severity of the surgical repair and, therefore, determines the level of protection with postoperative rehabilitation. Many surgeons will provide restrictions and precautions dependent upon the surgical procedure performed. An example of commonly prescribed restrictions is outlined in Table 1.

Protection of the repaired tissue is achieved through range of motion restrictions, weight-bearing status, hip flexor protection, bracing, and an anti-rotational system. Hip extension and external rotation are typically restricted initially because they place stress on the anterosuperior portion of the joint capsule. Many arthroscopic procedures enter the joint using two to three arthroscopic portals placed anterolateral, mid-anterior to the hip joint [2]. Depending on joint laxity, closure to the capsule or a plication of the capsule may be performed in order to increase hip joint stability. The duration of restriction is variable depending upon the severity of the joint closure and quality of ligamentous integrity. Supplemental protection may be provided by the use of a hip brace, which limits extension, external rotation, and abduction.

Patients are advised to use different options to sleep comfortably while maintaining postoperative range of motion restrictions. One, patients may either sleep in a device to limit external rotation or sleep in a constant passive motion machine (CPM), if it is prescribed, which maintains a neutral position of the hip. Sleeping on the

Table 1 Surgical procedures and restrictions

Restrictions	Osteoplasty/rim trimming	Labral repair/reconstruction	Microfracture
Passive range of motion (PROM)	Extension to 0° × 21 days	Extension to 0° × 21 days	Extension to 0° × 21 days
	External rotation to 0° × 17–21 days	External rotation to 0° × 17–21 days	External rotation to 0° × 17–21 days
	Abduction 0–45 × 14 days	Abduction 0–45 × 14 days	Abduction 0–45 × 14 days
	Flexion, adduction, IR no limits within pain-free range	Flexion, adduction, IR no limits Within pain-free range	Flexion, adduction, IR no limits within pain-free range
Weight bearing (WB)	20 lb WB with crutches × 3 weeks, 50 % × 1 week, then wean gradually 10 %/day as tolerated until normal gait is achieved	20 lb WB with crutches × 3 weeks, 50 % × 1 week, then wean gradually 10 %/day as tolerated until normal gait is achieved	20 lb WB with crutches × 7 weeks, 50 % × 1 week, then wean gradually 10 %/day as tolerated until normal gait is achieved
Continuous passive motion (CPM)	6+ h/day at 10° abduction	6+ h/day at 10° abduction	8+ h/day at 10° abduction
Hip brace	Set at 0–105 used while ambulating × 21 days	Set at 0–105 used while ambulating × 21 days	Set at 0–105 used while ambulating × 21 days
Anti-rotational boots	Used while lying supine × 17–21 days, correlated to ER restriction	Used while lying supine × 17–21 days, correlated to ER restriction	Used while lying supine × 17–21 days, correlated to ER restriction

Resource: Philippon sports med arthroscopy rev

uninvolved side offers the patient an alternative sleeping position while gravity restricts abduction and external rotation from occurring. Patients are encouraged to use a large pillow under the involved extremity to limit excess adduction and internal rotation, which is typically painful.

Weight-bearing restrictions may vary among surgeons, the surgical procedure, concomitant procedures, and/or comorbidities. A single-subject study assessing in vivo acetabular contact pressures during gait showed that touchdown weight-bearing (TDWB) produced the least amount of acetabular contact pressure when compared to full weight-bearing (FWB), partial weight-bearing (PWB), and non-weight-bearing gait (NWB) [3]. Patients who do not undergo microfracture of a weight-bearing surface are typically limited to TDWB for 2–4 weeks. One of the primary reasons of this limitation is to reduce joint effusion and tissue edema. Joint effusion of the hip triggers arthrogenic inhibition of the gluteus medius (GM) [4], which is essential for normal gait and pelvic control. In later phases of rehabilitation, recurrent or chronic joint effusion may continue to contribute to GM inhibition, leading to increased anterior joint forces during hip extension and gait [1, 5].

Reduction of Pain, Inflammation, and Fibrosis

Postoperative pain and inflammation are controlled through medication, use of a continuous passive motion (CPM) machine, early nonresistance biking, and soft tissue modalities. Nonsteroidal anti-inflammatory (NSAIDS), muscle relaxants, and pain medication may be prescribed by the physician. Ice compression devices are recommended 4–5×/day or as needed for pain control and inflammation during the first 2 weeks of recovery. Cryotherapy has been shown to be an efficacious modality with reducing the use of analgesic medication and pain while improving comfort with sleeping and overall satisfaction in postoperative patients [6, 7].

Range of motion is initiated as early as postoperative day (POD) 0. This includes early stationary biking without resistance for 20 min, twice daily to assist with portal drainage. Passive circumduction range of motion performed by a therapist or caregiver is recommended daily. The circular motion performed during this exercise encompasses flexion, extension, abduction, and adduction of the hip and is simple to instruct to caregivers. Additionally, it has been theorized to reduce adhesions about the zona orbicularis of the hip because of the circular motion.

Manual physical therapy techniques, such as lymphatic massage or gentle joint mobilizations (grade 1 and 2), can be used to decrease postsurgical inflammation and control pain. These techniques are performed for 2 weeks after surgery or until inflammation has dissipated. Soft tissue mobilizations to lengthen tissue and decrease reactive muscle tone surrounding the hip joint are then initiated. Anterior musculature, such as the iliopsoas, rectus femoris, tensor fasciae latae, and adductor muscles, tends to respond to surgery with reactive shortening and hypertonicity. This may be due to effusion and positioning the hip in flexion for prolonged periods of time, such as when using the CPM machine. Previous authors have recommended that patients lay in a prone position daily during phase 1 [8].

Restoring Passive and Active Ranges of Motion

Passive range of motion is initiated on POD 0 and continued through phases 1 and 2 or until full range of motion is achieved. Initially, flexion, internal rotation (IR), abduction (abd), adduction (add), and circumduction are performed to help restore mobility, prevent fibrosis [8–10], and contribute to joint health [11]. Abduction is restricted to 45° for the first 2 weeks, and external rotation and extension are typically limited to neutral for 2–3 weeks. These limitations prevent capsular stretching at the surgical site. Excessive flexion and IR should be avoided to limit irritation due to compression at the surgical site [9]. The frequency of passive range of motion is varied; however, twice daily is recommended.

Active range of motion is initiated within the first week in order to promote neuromuscular control and normal muscle firing patterns. Exercises, such as cat and camel and quadruped rocking (Figs. 1 and 2), are initiated as early as postoperative day 4. These exercises are recommended to reestablish pelvic control through the stabilizing effect of co-contraction and recruitment of synergistic muscle groups. Quadruped rocking allows gravity-assisted hip flexion to occur while decreasing the pinching sensation a patient may experience in supine flexion. Hip flexor activity is typically limited during the initial 2 weeks of rehabilitation to avoid magnifying anterior hip pain. During week 3, patients may begin light, pain-free hip flexion exercises. Patients and therapists should be aware of compensation patterns, increased irritation of rectus femoris, hip adductors, and tensor fasciae latae (TFL) muscles and tendons; these muscles tend to substitute for iliopsoas weakness or inhibition

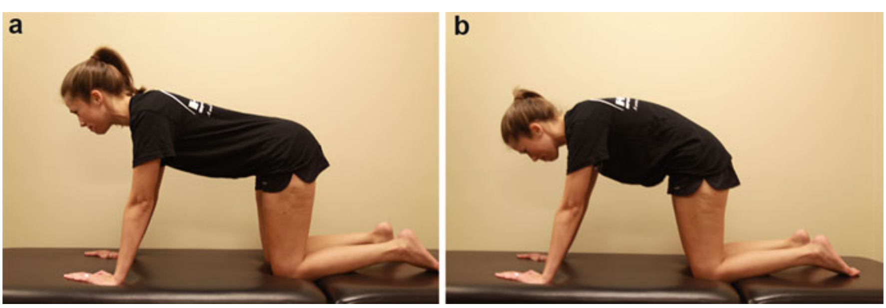


Fig. 1 (**a** and **b**) Cat and camel exercise. The patient assumes a quadruped position and performed lumbar flexion and extension and pelvis anterior and posterior rotation. This creates a hip flexion and extension moment while promoting gentle neuromuscular control in a closed kinetic chain

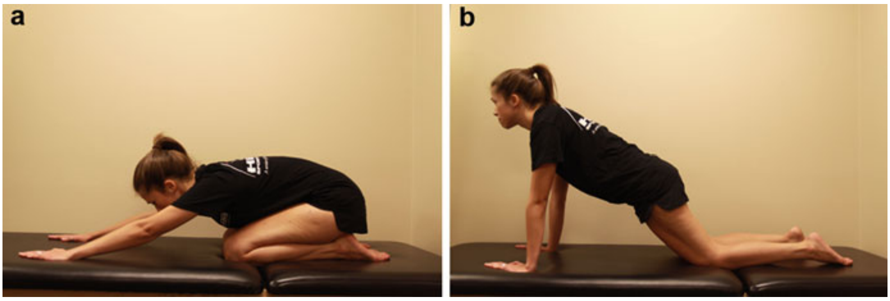


Fig. 2 (**a** and **b**) Quadruped rock exercise. The patient assumes the quadruped position and performs a gentle rocking motion forward and backward as tolerated. This exercise is an excellent exercise for patients to perform unassisted range of motion and gentle stretching of flexion and extension at home while promoting neuromuscular control of the hip, pelvis, and trunk

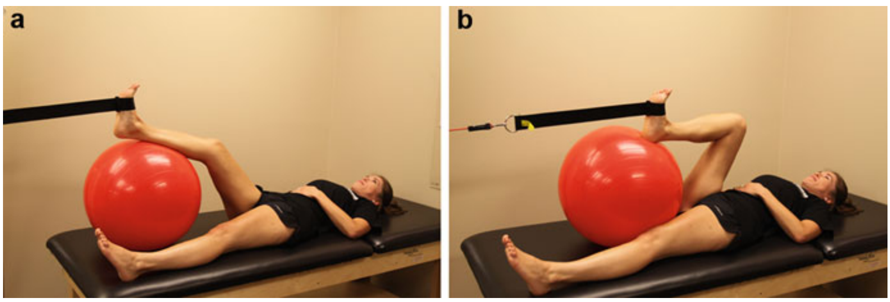


Fig. 3 (**a** and **b**) Supine hip flexion. This exercise is performed in a supine position with the involved extremity on a fit ball. As the patient stabilizes the trunk, active hip flexion and extension are performed to encourage light activity of the hip flexors and normal movement patterns. Resistance may be added to this exercise as pictured above

[1, 12]. Figure 3a, b demonstrates such an exercise. During this exercise, the patient lays supine while rolling a fit ball into hip flexion and extension while maintaining trunk control. This can be performed with (as pictured) or without light resistance.

Proper Neuromuscular Control Patterns

A key component to prepare the hip joint for weight-bearing and normal gait is reestablishing proper neuromuscular control patterns of the hip. Patients with FAI and labral pathology often develop compensation patterns of the hip, trunk, and lower extremity. These patterns continue after surgery has been performed, which may be harder to correct the longer they have been present. Common patterns that occur prior to surgery include:

Hypertonic muscles
- Iliopsoas
- TFL
- Adductors
- Piriformis

Inhibited/hypotonic muscles
- Gluteus medius
- Gluteus maximus
- Deep external rotators

In order to restore normal muscle activation, the musculature of the hip, trunk, and lower extremity must be addressed simultaneously. Initially, patients are instructed on isometric exercises that recruit the GM, GMax, quadriceps, hamstrings, and transverse abdominus. Patients are instructed to co-contract trunk musculature with hip and lower extremity musculature in order to promote trunk and pelvic stability.

As swelling and pain decrease, open chain, active assist, and active exercises are initiated with the goal of recruiting inhibited muscles and reducing activity of hypertonic muscles. Three studies have looked at rehabilitation exercises that target gluteal musculature while minimizing hypertonic musculature [13–15]. Selkowitz et al. [13] studied gluteal-specific exercises that reduce TFL activity. Philippon et al. [14] ranked 13 hip exercises in order of GM iliopsoas activity. In a similar study, Giphart et al. [15] ranked the same exercises with regard to gluteal piriformis and pectineus activity. While some exercises in these studies will reduce, for example, TFL activity, they may increase the activity of other muscles, such as the iliopsoas, pectineus, and piriformis. Therapists should use these exercises in the presence of hypertonicity of the specific muscles identified during a patient assessment.

Active assisted exercises include the slide board abduction exercise and can begin during the first postoperative week. Once gluteus medius isolation is mastered with slide board abduction, the patient can progress to standing abduction, and side lying gluteus medius holds. It is important to be aware of TFL substitution during these exercises. Early activation of the deep external rotators can begin week 2 with prone external rotator activation from the position of IR and stopping at neutral. This will activate the rotator muscles with respect to the ER rotation restriction. Furthermore, gluteus maximus activation and transverse abdominis stability can be progressed from an isometric exercise to quadruped hip extensions (Fig. 4).

Fig. 4 Quadruped hip extension. The patient assumed quadruped position and then extends the involved leg into hip extension. Careful attention should be placed on compensation patterns such as excessive lumbar lordosis and anterior pelvic tilting. The patient is instructed to recruit trunk musculature in order to counteract these compensatory patterns

Criteria to Advance to Phase 2

Criteria to advance to phase 2 include (1) minimal pain with all phase 1 exercises, (2) correct muscle firing patterns with all phase 1 exercises, and (3) minimal complaints of anterior hip pain prior to 100° of passive hip flexion.

Phase 2

Phase 2 begins when weight-bearing restrictions have been discontinued and phase 1 criteria have been met. The main goal of phase 2 is to restore normal gait. Secondary goals include returning the patient to weight-bearing activities of daily living, ascending and descending stairs with alternating gait, and double-limb squatting. Phase 2 may be considered the most critical phase to success after hip arthroscopy because of the neuromuscular challenges the rehabilitation specialist will encounter when returning a patient to unrestricted weight bearing. As patients progress to walking without an assistive device, they are more likely to experience increased anterior hip pain, which is commonly described as hip flexor tendonitis [8, 16]. This pain may be better explained as excessive anterior hip forces on the surgical site created by the femoral head due to insufficient muscle activity during hip extension and flexion motions. This pain may be difficult to control without rest and unloading of the joint until the pain subsides.

Gait Progression

Weaning a patient from assisted weight bearing to full weight bearing should be progressed carefully. Initially, weight bearing should be introduced with weight-shifting exercises in order to gradually promote co-contraction and muscle firing patterns that promote pelvic and hip stability. For instance, side-to-side leaning and forward and backward leaning in mini-lunge position, with the involved limb both forward and to the rear, allow the patient to experience variable static positions of gait in a closed kinetic chain. Progressive perturbation may be added during this exercise to promote increased neuromuscular recruitment (Fig. 5a, b).

It is critical that weight-bearing activities are performed with optimal neuromuscular control. The absence of adequate muscle recruitment of the iliopsoas, gluteus medius, and gluteus maximus muscles in weight-bearing leads to increased anterior joint forces. Using three-dimension computer modeling, Lewis et al. [1] found that anterior hip joint force increased with increasing hip extension. Furthermore, decreased force contribution from the gluteal muscles during hip extension and the iliopsoas muscle during active hip flexion resulted in higher anterior hip joint force. This results in greater susceptibility to irritation of the surgical site and subsequent increases in pain and inflammation.

As patients strive to walk normally, a typical gait abnormality will develop. Between mid-stance and toe-off, anterior pelvic rotation increases while lumbar extension increases. Patients may increase lateral pelvic rotation in place of anterior rotation in the sagittal plane. This is typically due to inadequate, active hip extension. Oftentimes, passive hip extension will present normally and symmetrical compared to

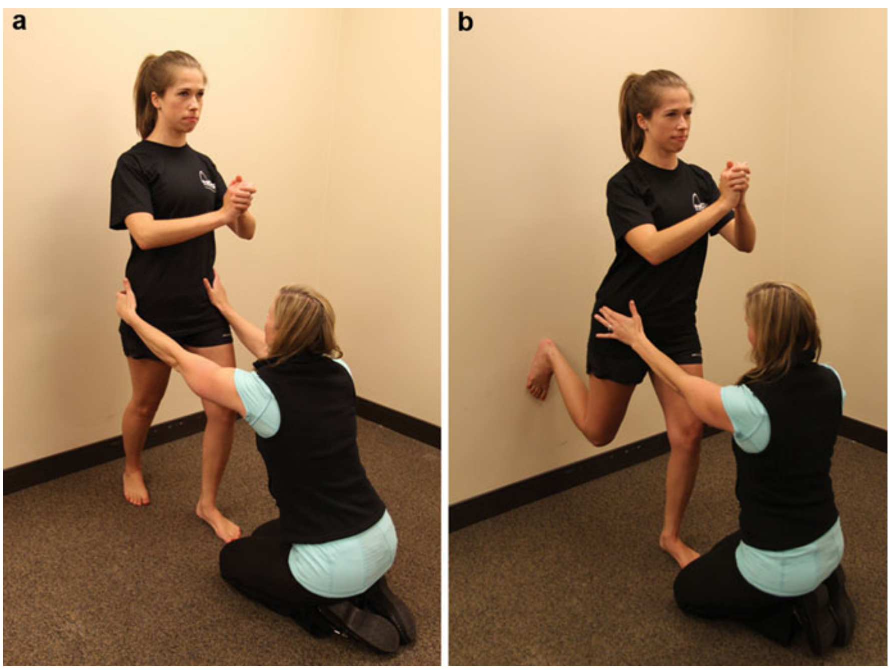


Fig. 5 (**a** and **b**) Mini-lunge weight bearing with perturbations provided by therapist. These exercises may be performed as weight-shifting exercises without intervention from the therapist or as a neuromuscular exercise where the therapist provides perturbations in arbitrary directions to promote stability and co-contraction of the trunk and lower limb. In either scenario, the patient attempts to maintain an upright position while weight shifting or reacting to the therapists perturbations

Table 2 Quick test screen for progression to gait phase

Test	Procedure	Advancement
Prone hip extension test	Patient is able to maintain proper gluteus maximus initiation and maintain activation beyond 0° hip extension	Patient can progress to weight-shifting exercises
Single-leg balance test	Patient is able to maintain level pelvis without pelvic drop or rotation for 30 s	Patient can progress to unassisted short-distance indoor walking[a]

Utilization of QuickScreen tests helps to assure that progression into the crutch weaning phase is appropriate for the individual patient and therefore decreases the risk of anterior joint irritation complicating success throughout this phase of rehabilitation

[a]Patients should demonstrate proper short-distance walking without a limp prior to performing it independently

the uninvolved hip. However, when active hip extension is tested (see Table 2), it is often deficient. If left unaddressed, this gait abnormality may increase anterior hip pain and subsequently lead to secondary low back and sacroiliac joint pain. Therefore, a detailed progression of gait exercises, temporary gait modifications, and continuous verbal and tactile cuing is recommended.

Once static double- and single-limb weight bearing are tolerated, gait may be introduced gradually and with caution. Devices such as an anti-gravity treadmill or walking in chest-deep water (see section "Aquatic Therapy") allow patients to exercise normal, double-limb gait with less weight-bearing load on the hip joint. If these devices are not available, slowly progressing from TDWB to PWB to FWB while using two and then one crutch is advised. Additionally, patients are advised to begin walking slowly, with a shorter stride length to reduce anterior hip joint force [5]; this decreases the amount of hip extension required in the presence of insufficient gluteal contribution. Furthermore, using increased ankle push-off during gait can help to offset weakened gluteal musculature and decrease potential anterior hip joint force [17]. Soreness that resolves within 24 h is considered acceptable, yet patients that experience pain longer than 24 h are advised to discontinue weight bearing until the pain subsides and return to the previous level before progressing.

Simple cuing techniques can promote normal sequencing of muscle activation with movement. During hip extension, for example, Lewis et al. [18] discovered that cuing gluteal activation during prone hip extension caused simultaneous activation of the gluteal and hamstrings muscles rather than early activation of the hamstrings. During forward walking, cuing a patient to place emphasis on trunk control to limit anterior pelvic tilt and lumbar extension during mid-stance to toe-off will help to decrease this compensatory pattern and improve pelvic control. Slow backward walking with an emphasis on controlled, active hip extension from mid swing to toe contact may assist in recruiting the gluteal musculature during hip extension. Side stepping offers patients increased tolerance to weight bearing while eliminating sagittal plane movements of the hip, thus decreasing the potential for anterior hip pain.

Aquatic Therapy

Aquatic therapy is a valuable adjunct to a land-based physical therapy program. Water buoyancy in varying water depths offer patients decreased load on weight-bearing joint. Additionally, water offers a more stable environment for patients to perform activities that may otherwise be too difficult or painful to perform on land. Lastly, the warmth and pressure of water may help to decrease pain and swelling in the hip joint.

Patients may begin aqua jogging in deep water using a buoyancy suit and waterproof dressings as early as POD 3. Patients are progressed to chest-deep walking approximately 2–3 weeks after surgery. A similar progression of side stepping, forward walking, and backward walking is instructed as mentioned above. Furthermore, strengthening exercises can be performed in waist-deep water 2 weeks prior to land training. Pool running without a floatation device should begin 4 weeks prior to starting a land-based progression. When a patient can perform water running without pain, an interval progression may be implemented on dry land.

Criteria to Progress to Phase 3

Once gait is normalized and the proper exercise progressions of phase 2 have been mastered by the patient, it is appropriate to progress to initial strength and endurance of phase 3. Specific criteria that should be accomplished in phase 2 in order to ensure progression to phase 3 is appropriate include (1) normalized gait without pain or limping, (2) pain-free performance of phase 2 exercises absent of excessive compensatory patterns, (3) therapist discretion based upon clinical screening tools, and (4) ability to perform single-leg squat test for 1 min.

Phase 3

The ultimate goal of phase 3 of the rehabilitation program is to restore muscle strength, power, and endurance. Activities of daily living and recreational activities should become relatively asymptomatic during this phase. The physical therapist should continue to monitor compensatory patterns previously described during the progression from open kinetic chain to closed kinetic chain exercises and as the demand for range of motion, joint stability, and neuromuscular control increases. Careful consideration should be taken to determine the best exercise prescription based on the demands and functional goals of the patient while applying basic training principles of load, volume, frequency, and periodization when designing exercise prescriptions [19, 20]. Exercise specificity should be considered with athletes seeking to return to sports participation; however, this should not be overridden by fundamental physiologic, metabolic, and biomechanical voids that must be fulfilled prior to prescribing more complex and specific exercises.

Exercises should emphasize muscles that stabilize the hip joint and reduce anterior joint forces. These muscles include the GM, gluteus maximus, and Iliopsoas [1, 21]. Table 3 presents a series of exercise progressions for each of these muscle groups, which are based on empirical evidence [13–15, 22, 23], clinical rationale, and anecdotal expertise. However, therapists should exercise discrimination when prescribing these exercises based on the individual differences of each patient and clinical findings.

Exercise parameters should take into account the level of previous training and current state of fitness an individual can endure. The American College of Sports Medicine [20] has defined specific parameters that relate to training level and optimal training parameters. These recommendations are based on large meta-analyses that calculated effect sizes for training dose–response relationships [24, 25]. An untrained individual, for instance, will have maximal strength gains by training at 60 % of 1 repetition maximum (1RM), 3 days per week for 4 sets per muscle group. Recreational athletes have maximal strength gains with an intensity of 80 % of 1RM, training 2 days per week at 4 sets per muscle group. Athletes benefit most with a training intensity of 85 % of 1RM, 2 days per week and a training volume of 8 sets per muscle group.

It should be noted that data derived for these studies is derived from healthy individuals. Factors such as disuse atrophy, arthrogenic inhibition, and pain inhibition should be taken into consideration when implementing exercise prescriptions using these recommendations. Furthermore, due to the altered physiologic environment of the hip joint and surrounding tissues, increases in temperature, pain, and inflammation about the joint should be monitored closely. These findings indicate that the patient has trained beyond the physiologic threshold of the joint and surrounding tissue and may cause further injury [26]. In the presence of these findings, the physical therapist should adjust the training regimen and allow for symptoms to subside.

Strength exercises should be progress from double-limb to single-limb exercises and single-plane to multiplane as the patient tolerates. During single-limb exercises, careful attention should be placed on limb alignment. Oftentimes, patients will exhibit excessive pelvic rotation with femoral internal rotation and tibial external rotation (dynamic valgus), which may lead to increased hip and/or knee pain. As strength improves, a patient may be slowly progressed to plyometric exercises, if necessary. In a patient who desires to

Table 3 Exercise progressions per muscle group

Exercise	Criteria to progress to exercise below	Compensatory patterns	Recommended phase
Gluteus maximus progression			
Glute isometrics	Ability to hold isolated glute max contraction for 3 sets of 30 s holds	Inability to isolate gluteus maximus due to iliopsoas/hamstring/ quadratus lumborum (QL) co-activation	Phase 1
Quadruped leg lift (Fig. 4)	1. Ability to maintain a level pelvis without pelvic rotation or lumbar extension	1. Inability to maintain pelvic control due to lack of transverse abdominis control	Phase 1
	2. Gluteus maximus is active from 30 deg flexion through 0 deg extension	2. Compensation with hamstring or QL for decreased glute max strength	
	3. Achievement of 2 sets of 10 reps prior to advancement to next exercise		
Shuttle squats with heel raise	1. Ability to achieve proper glute/ quad synergy activation	1. Anterior pelvic tilt due to inability to maintain proper core control with squat	Phase 2
	2. Patient is progressed to 50 % WB	2. Rectus femoris domination compensating for improper glute max activation	
	3. Achievement of 2 sets of 20 reps	3. Hamstring domination	
Prone short arc quad (SAQ)	1. Ability to maintain synergistic glute/quad activation	1. Excessive anterior pelvic tilt due to rectus femoris overactivation and lack of TA stabilizing pelvis	Phase 2
	2. Proper neuromuscular pattern (glute max > hamstring > QL)		
	3. Achievement of 2 sets of 20 reps holding 5–10 s	2. Inability to maintain glute max co-contraction with quad activation	
		3. QL initiates glute extension	
Weight shifting	1. Ability to maintain level pelvis without drop or pelvic rotation	1. Patient is unable to increase body weight load through the hip joint without pelvic drop, rotation	Phase 2
	2. Proper glute max activation stabilizes hip joint as body weight increases the joint load	2. Glute max does not activate properly as body weight loading increases into the joint	
	3. Proper performance of single-leg (SL) balance for 30 s without hip drop, rotation, knee valgus, or anterior hip pain	3. Patient is unable to maintain SL balance for 30s and cannot correct compensation patterns with cueing	
Double-leg (DL) bridging	1. Patient is able to initiate hip extension with glute max musculature while maintaining a level pelvis	1. Patient initiates hip extension with hamstrings, adductors, and QL	Phase 2
	2. Patient is able to maintain neutral spine and neutral pelvic tilt throughout exercise	2. Patient is unable to isolate and maintain proper glute max and glute med control resulting in rectus femoris co-activation leading to anterior pelvic tilt and lumbar lordosis	
	3. Proper performance of 3 sets of 15 reps		

(*continued*)

Table 3 (continued)

Exercise	Criteria to progress to exercise below	Compensatory patterns	Recommended phase
Double-knee bends	1. Patient is able to maintain a level pelvis, with neutral spine and neutral femoral internal rotation	1. Patient demonstrates hip drop or pelvic rotation and femoral internal rotation	Phase 2
	2. Patient is maintain a stable pelvis and produce a hinge movement through proper hip mobility and glute max activation to create the hip extension moment, without allowing knees to bend beyond their toes	2. Patient is unable to disassociate between stable pelvis and movable hip joint, creating lumbar lordosis and inactivity of glute max muscles	
	3. Proper performance of 3 sets of 20 reps		
Step-ups	1. Patient is able to perform exercise with level pelvis, proper glute activation for hip extension, glute/quad synergy without excessive fatigue	1. Patient exhibits contralateral pelvic drop and rotation and increased femoral IR, with step-up	Phase 2
	2. Proper performance of 3 sets of 20 rep while maintaining correct form	2. Patient shows upper body lateral shift to compensate for glute complex weakness	
Single-leg (SL) bridge	1. Patient is able to maintain neutral pelvis, neutral lumbar spine, and glute activation	1. Patient exhibits contralateral pelvic drop and decreased glute max and glute med activation with increase hamstring domination and quadratus lumborum	Phase 3
	2. Proper performance of 3 sets of 15 reps with proper form and no signs of anterior joint stress		
Balance squat	1. Patient is able to maintain neutral pelvis, without femoral IR, and glute/quad synergy	1. Patient is unable to maintain a neutral pelvis by displaying contralateral pelvic drop or rotation. Ipsilateral anterior tilt indicates improper glute/quad synergy	Phase 3
	2. Proper performance of 3 sets of 20 reps without anterior joint pain	2. Decreased glute med and rotator contribution leads to femoral IR	
Single-leg squat	1. Patient is able to maintain neutral pelvis, without femoral IR and glute/quad synergy	1. Patient is unable to maintain a neutral pelvis by displaying contralateral pelvic drop or rotation. Ipsilateral anterior tilt indicates improper glute/quad synergy	Phase 3
	2. Proper performance of 3 sets of 20 reps to progress to multiplanar activities	2. Decreased glute med and ER rotator contribution leads to femoral IR	
	3. Proper performance of 3 sets for 1 min maintaining proper form is necessary for progression to plyometric activity		

(*continued*)

Table 3 (continued)

Exercise	Criteria to progress to exercise below	Compensatory patterns	Recommended phase
Multiplanar lunges	1. Patient is able to maintain neutral pelvis, without femoral IR and glute/quad synergy	1. Patient is unable to maintain a neutral pelvis by displaying contralateral pelvic drop or rotation	Phase 3
	2. Proper performance of 3 sets of 20 reps	2. Patient exhibits femoral IR or lateral trunk lean with increased adductor and rectus femoris to compensate for inefficient glute max and glute med contribution	
Lateral and diagonal	1. Patient is able to maintain proper femoral alignment with 30° of knee absorption and explosion	1. Patient displays femoral IR and inability to absorb and explode due to inadequate eccentric glute complex and quad control	Phase 3
Agilities (Fig. 9a, b)	2. Patient is able to maintain proper pelvic alignment without anterior tilt, contralateral pelvic drop, or rotation	2. Patient is unable to maintain a neutral pelvis by displaying anterior pelvic tilt indicative of disproportionate glute/quad contribution and/or contralateral pelvic drop or rotation	
	3. Ability to perform 3 sets of 1 min prior to adding sport cord resistance		
Gluteus medius progression			
Slide board abduction	1. Patient is able to properly isolate the glute med while maintaining a neutral pelvic position	1. Patient is unable to isolate glute med and compensates with TFL, QL, or rectus femoris	Phase 1
	2. Ability to perform 2 sets of 15 reps with proper form		
Standing abduction	1. Patient is able to properly isolate the glute med while maintaining a neutral pelvic position	1. Patient is unable to isolate glute med and compensates with TFL, QL, or rectus femoris	Phase 1
	2. Ability to perform 3 sets of 10 reps with proper form	2. Patient is unable to maintain a level pelvis throughout the exercise	
Side lying glute med holds with glute max push backs	1. Patient is able to maintain a level pelvis with a stable lumbar spine	1. Patient cannot maintain a neutral spine and compensates with anterior pelvic tilt, posterior or anterior pelvic rotation	Phase 1
	2. Patient is able to maintain glute med activation throughout	2. Patient is unable to isolate glute med or glute max and compensates with TFL, rectus femoris, QL, or hamstring	
	3. Ability to perform 3 sets of 30 s holds with proper form		
Side lying neutral clams	1. Patient is able to maintain a level pelvis with a stable lumbar spine	1. Patient cannot maintain a neutral spine and compensates with anterior pelvic tilt, posterior or anterior pelvic rotation	Phase 2
	2. Patient is able to maintain glute med activation throughout	2. Patient is unable to activate glutes properly and compensates with TFL, rectus femoris, QL, or hamstring	
	3. Ability to perform 3 sets of 15 with proper form		
3-way glider (lateral, 45° posterior) (Fig. 8a, b)	1. Patient is able to maintain a level pelvis with a stable lumbar spine	1. Patient shows increased lumbar lordosis or superior tilt	Phase 2
	2. Patient is able to maintain glute med activation throughout	2. Patient is unable to isolate glute med and compensates with TFL, rectus, or QL	
	3. Ability to perform 3 sets of 15 with proper form and no anterior hip pain		

(*continued*)

Table 3 (continued)

Exercise	Criteria to progress to exercise below	Compensatory patterns	Recommended phase
Side planks	1. Patient is able to maintain a level pelvis with a stable lumbar spine	1. Patient is unable to maintain a neutral pelvis and compensates with an anterior tilt or rolling the pelvis forward	Phase 2
	2. Patient is able to maintain glute med activation	2. Patient compensates with over activating TFL and/or rectus femoris	
	3. Ability to perform 3 sets of 15 with proper form and no hip pain		
Hip hiker (Figs. 6 and 7)	1. Patient is able to maintain a level pelvis with a stable lumbar spine	1. Patient is unable to maintain a neutral pelvis and compensates with an anterior tilt or rolling the pelvis forward	Phase 3
	2. Patient is able to maintain glute med activation	2. Patient is unable to utilize glute med as the dominant muscle and compensates with over activating TFL, adductor and/or Rectus femoris	
	3. Ability to perform 3 sets of 15 with proper form and no hip pain		
Lateral step downs	1. Patient is able to maintain a level pelvis with a stable lumbar spine	1. Patient is unable to maintain a neutral pelvis and compensates with an anterior tilt, pelvic rotation, or contralateral pelvic drop	Phase 3
	2. Patient is able to maintain glute med activation	2. Patient is unable to utilize glute med and glute max as the dominant muscles and compensates with over activating TFL, adductor, and/or rectus femoris	
	3. Ability to perform 3 sets of 15 with proper form		
Lateral and diagonal agility (Fig. 9a, b)	1. Patient is able to maintain proper femoral alignment with 30° of knee absorption and explosion	1. Patient displays femoral IR and inability to absorb and explode due to inadequate eccentric glute complex and quad control	Phase 3
	2. Patient is able to maintain proper pelvic alignment without anterior tilt, contralateral pelvic drop, or rotation	2. Patient is unable to maintain a neutral pelvis by displaying anterior pelvic tilt indicative of disproportionate glute/quad contribution and/or contra lateral pelvic drop or rotation	
	3. Ability to perform 3 sets of 1 min prior to adding sport cord resistance		
Hip flexor progression			
Side lying hip flexion active assisted from 70° to 90°	1. Able to maintain proper pelvic alignment with iliopsoas initiating movement	1. Patient is unable to stabilize pelvis with proper core control	Phase 1
	2. Ability to perform 2 sets of 15 without anterior hip pain	2. Patient uses rectus femoris instead of iliopsoas to initiate and dominate movement pattern	
Side lying hip flexion active assisted from 0° to 100°	1. Able to maintain proper pelvic alignment with iliopsoas initiating movement	1. Patient is unable to stabilize pelvis with proper core control	Phase 1
	2. Ability to perform 2 sets of 15 without anterior hip pain	2. Patient uses rectus femoris instead of iliopsoas to initiate and dominate movement pattern	

(continued)

Table 3 (continued)

Exercise	Criteria to progress to exercise below	Compensatory patterns	Recommended phase
Supine hip flexion ball rolls (Fig. 3a, b performed without cord)	1. Able to maintain proper pelvic alignment with iliopsoas initiating movement	1. Patient is unable to stabilize pelvis with proper core control	Phase 1
	2. Ability to perform 2 sets of 15 without anterior hip pain	2. Patient uses rectus femoris instead of iliopsoas or TFL and adductor to initiate and dominate movement pattern due to a dysfunctional iliopsoas	
Seated inseam heel drag	1. Able to perform pain-free with proper control into Fig. 2b position	1. Patient is unable to slide leg up without compensating by arching lumbar spine or forward trunk lean	Phase 2
	2. Able to perform 2 sets of 20 into acceptable ranges without posterior lateral hip symptoms	2. Patient is unable to control the knee down into the Fig. 2b position and experiences post/lateral hip discomfort commonly due to anterior joint stiffness or iliopsoas weakness	
Standing march to 90°	1. Able to perform with proper pelvic stability	1. Patient is unable to maintain a level pelvis resulting in anterior tilt, ipsilateral hip hike or contralateral rotation	Phase 2
	2. Activation of the hip flexor group with increased iliopsoas activation as the leg is brought up to further ranges of flexion	2. Patient is unable to activate iliopsoas properly and compensates with adductor/TFL activation	
	3. Proper performance of 2 sets of 20 pain-free		
Resisted supine ball rolls (Fig. 3a, b)	1. Able to maintain proper pelvic alignment with iliopsoas initiating movement	1. Patient is unable to stabilize pelvis with proper core control	Phase 3
	2. Ability to perform 2 sets of 15 without anterior hip pain	2. Patient uses rectus femoris instead of iliopsoas or TFL and adductor to initiate and dominate movement pattern due to a dysfunctional iliopsoas	

run or return to higher-risk sports or performing arts, plyometric strength is recommended, and patients are encouraged to pass a functional performance test in order to progress into phase 4. These sports and performing arts include ballet, figure skating, ice hockey, martial arts, basketball, and football.

The Vail Hip Sports Test is a performance test that has been derived from the Vail Sports Test for the knee [27]. The utility of this test is to determine if a patient is prepared to begin integrating athletic performance into training. In particular situations, it may be used a criteria to begin a return to running program. The test is comprised of four exercises: (1) single-knee bends, (2) lateral agility, (3) diagonal agility (Figs. 9a, b and 2b), and (4) forward box lunge [8]. Preparation for this clearance exam begins with performing the exercises without resistance to ensure sufficient foundational strength has been achieved. Then, a

Fig. 6 Hip hiker exercise. The patient places the involved leg on a box and uninvolved hip against a ball that rests against the wall. A lateral pelvic drop is performed (Trendelenburg maneuver) in order to exercise the gluteus medius muscle on the involved leg

gradual progression of sport cord resistance is added over the course of 3–4 weeks to increase exercise intensity in conjunction with increasing duration, as appropriate with demonstration of proper form. This continues until the sports test guidelines are achieved with a passing score of 17/20 (see Table 4).

Criteria to Advance to Phase 4

It should be noted that not all patients are suitable for phase 4 exercise progressions due to the increased demands that will be placed on the hip and surrounding musculature. Therefore, gait and functional activities must be free of pain and compensatory movement patterns. The patient should demonstrate symmetrical isometric strength as compared to the uninvolved side. Handheld dynamometry is recommended to obtain an accurate assessment of isometric muscle strength because it provides a valid and reliable force analysis that is more sensitive than manual muscle testing [28, 29]. Additionally, patients should pass a functional performance assessment, such as the Vail Hip Sports Test (Table 4), before progressing to this phase. Lastly, light running, plyometric drills, and resisted closed kinetic chain exercises should be free of pain and symmetrical with the uninvolved side and minimal fear avoidance.

Phase 4

Phase 4 is characterized by returning patients to their previous level of sports activity. This phase is reserved for athletes who desire to return to their prior level of athletic performance. In order to safely progress a patient to full performance, sports-specific, interval programs must be designed, implemented, and periodized with a strength and conditioning program that is tailored to the demands of the sport. Because every sport has different metabolic and biomechanical demands, a needs analysis of these demands should drive the exercise prescription for this phase [25, 30].

The metabolic demands of a sport vary [30]. While some sports rely on the aerobic energy system, Kreb's cycle, and type 1 muscle fibers, others will rely on the phosphocreatine and glycolysis system and type 2 muscle fibers. Many sports have mixed demands based on muscle groups and the variability of the sport. Ice hockey, for instance, requires lower extremity endurance for skating (aerobic demand); however, the athlete must have the capacity to sprint several times during this time (anaerobic demand). With regard to the hip, the gluteal musculature must be trained for these demands because early fatiguing could be catastrophic, leading to tissue failure.

A biomechanical analysis encompasses an assessment of limb and body movement during a sport [30]. Variables such as specific joint

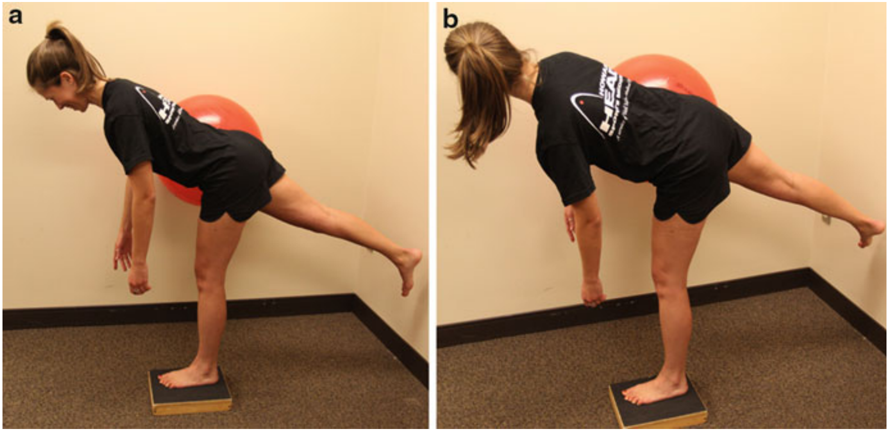


Fig. 7 (**a** and **b**) Advanced hip hiker exercise. The patient places the involved leg on a box and leans the uninvolved hip against a fit ball that is against a wall. The patient performs a partial Romanian dead lift and from this position performs lateral pelvis rotations to strengthen the gluteus medius muscle

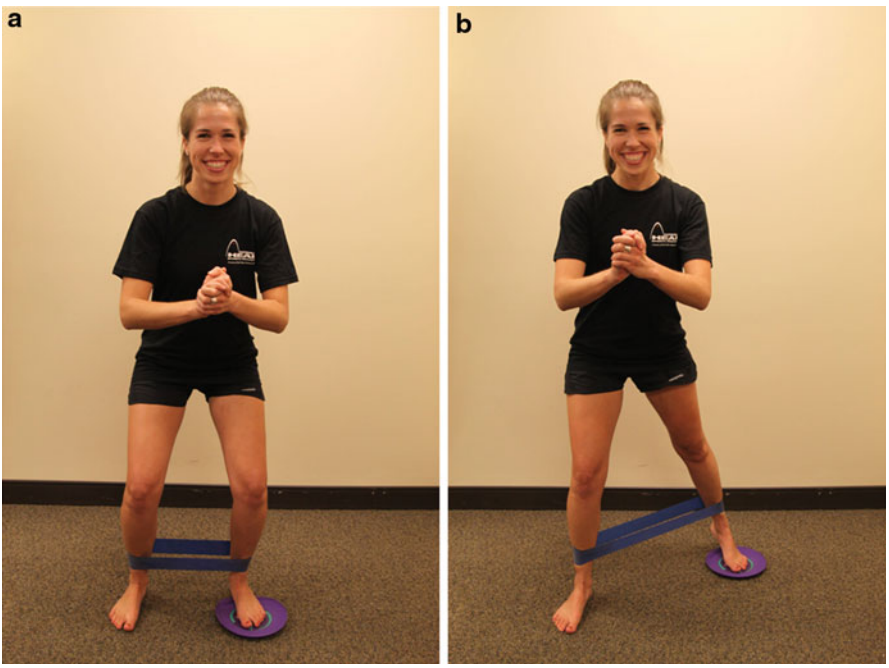


Fig. 8 (**a** and **b**) Three-way glider exercise. The patient stands in a mini-squat position with elastic tubing around his or her ankles (optional). Using a glider device under the involved limb (this may be switched for a different training effect), the patient performs hip abduction to the side, hip abduction and extension (45° to the rear), and sagittal plane extension

movements, speed of movement, closed versus open chain demands, and how these movements integrate with the trunk and other extremities are important in the development of a logical training program. With regard to the ice hockey athlete, there is a strong demand for single-limb, closed kinetic chain, and hip abduction movements. The speed at which movements occur is variable, but

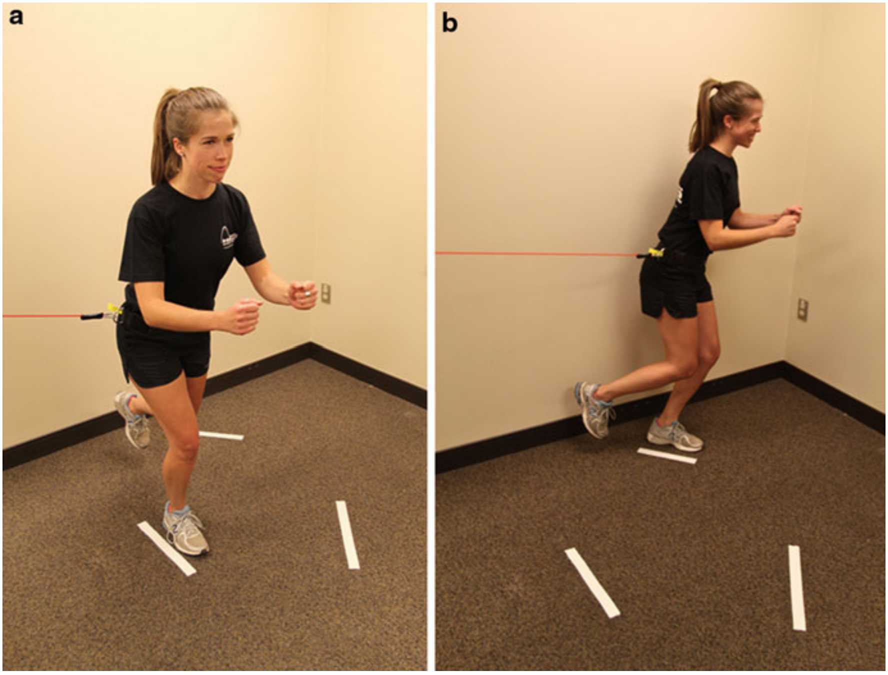


Fig. 9 (**a** and **b**) Sports cord lateral diagonal agility exercise. The patient attaches a sports cord to his or her waist. The start position acts as a pivot point and the point where the involved leg is placed (**a**). The patient then bounds laterally and diagonally 45° to the rear (**b**), returns to the start position (eccentric phase), and then bounds 45° to the front. The patient is instructed to absorb the impact of each bound at the start position before transitioning to the concentric phase of the exercise

Table 4 Vail Hip Sports Test [8]

Exercise	Goal	Resistance	Points
Single-knee bends	3 min	Single black sport cord	1 point for each 30 s completed
Lateral agility	100 s	Double black sport cord	1 point for each 20 s completed
Diagonal agility	100 s	Double black sport cord	1 point for each 20 s completed
Forward box lunge	2 min	Double black sport cord	1 point for each 30 s completed

collectively demands power and endurance and must integrate upper extremity and trunk movements associated with stickhandling. Lastly, the risk of re-injury should be assessed. Although phase 3 addresses strength endurance and power, while phase 4 addresses sports-specific demands, fatigue during performance can lead to irregular movement patterns, abnormal loading of the involved extremity, and, eventually, tissue failure [30, 31].

Based on a comprehensive needs analysis, the physical therapist should develop an interval strategy to return the patient to full sports participation while continuing exercises to improve strength and conditioning; workloads of exercise and sports participation should be balanced using the principles of periodization in order to enhance performance and prevent injury as a result of tissue overload [26, 32]. At this point, the physical therapist may coordinate with external training staff in order to develop such a program. However, high-level athletes not associated with professional organizations will necessitate the expertise of a physical therapist to safely return the patient to sports participation.

Summary

Rehabilitation after hip arthroscopy necessitates delicate and meticulous prescriptions of exercise progressions. Restoration of normal muscular function and neuromuscular control while preventing excessive anterior joint loading remains a paradoxical challenge during the course of rehabilitation and may obscure the clinical decision to advance a patient to higher-level activities. Criteria-driven parameters, empirical rationale, and evidenced-based medicine should precipitate these decisions in order to safely progress a patient toward achieving functional goals and ensuring a successful surgical outcome.

References

1. Lewis CL, Sahrmann SA, Moran DW. Anterior hip joint force increases with hip extension, decreased gluteal force, or decreased iliopsoas force. J Biomech. 2007;40(16):3725–31. PubMed PMID: 17707385. Pubmed Central PMCID: 2580726.
2. Philippon MJ, Schroder e Souza BG, Briggs KK. Labrum: resection, repair and reconstruction sports medicine and arthroscopy review. Sports Med Arthrosc Rev. 2010;18(2):76–82. PubMed PMID: 20473125.
3. Givens-Heiss DL, Krebs DE, Riley PO, Strickland EM, Fares M, Hodge WA, et al. In vivo acetabular contact pressures during rehabilitation. Part II: postacute phase. Phys Ther. 1992;72(10):700–5; discussion 6-10. PubMed PMID: 1528963.
4. Freeman S, Mascia A, McGill S. Arthrogenic neuromusculature inhibition: a foundational investigation of existence in the hip joint. Clin Biomech. 2013;28(2):171–177.
5. Lewis CL, Sahrmann SA, Moran DW. Effect of hip angle on anterior hip joint force during gait. Gait Posture. 2010;32(4):603–7. PubMed PMID: 20934338. Pubmed Central PMCID: 3063362.
6. Singh H, Osbahr DC, Holovacs TF, Cawley PW, Speer KP. The efficacy of continuous cryotherapy on the postoperative shoulder: a prospective, randomized investigation. J Should Elb Surg Am Should Elb Surg. 2001;10(6):522–5. PubMed PMID: 11743529.
7. Speer KP, Warren RF, Horowitz L. The efficacy of cryotherapy in the postoperative shoulder. J Should Elb Surg Am Should Elb Surg. 1996;5(1):62–8. PubMed PMID: 8919444.
8. Wahoff M, Ryan M. Rehabilitation after hip femoroacetabular impingement arthroscopy. Clin Sports Med. 2011;30(2):463–82. PubMed PMID: 21419967.
9. Enseki KR, Draovitch P. Rehabilitation for hip arthroscopy. Op Tech Orthop. 2010;20(4):278–81. PubMed PMID: 2010886012. Language: English. Entry Date: 20110204. Revision Date: 20111111. Publication Type: journal article. Journal Subset: Biomedical.
10. Stalzer S, Wahoff M, Scanlan M. Rehabilitation following hip arthroscopy. Clin Sports Med. 2006;25(2): 337–57, x. PubMed PMID: 16638496. Epub 2006/04/28. eng.
11. Nugent-Derfus GE, Takara T, O'Neill JK, Cahill SB, Gortz S, Pong T, et al. Continuous passive motion applied to whole joints stimulates chondrocyte biosynthesis of PRG4. Osteoarthritis Cartilage. 2007;15(5): 566–74. PubMed PMID: 17157538. Pubmed Central PMCID: 2680602.
12. Lewis CL, Sahrmann SA, Moran DW. Anterior Hip Joint Force Increases with Hip Extension, Decreased Gluteal Force, or Decreased Iliopsoas Force. J. Biomech. 2007;40(16):3725–3731.
13. Selkowitz DM, Beneck GJ, Powers CM. Which exercises target the gluteal muscles while minimizing activation of the tensor fascia lata? Electromyographic assessment using fine-wire electrodes. J Orthop Sports Phys Ther. 2013;43(2):54–64.
14. Philippon MJ, Decker MJ, Erik G, Torry M, Wahoff M, LaPrade RF. Rehabilitation Exercise Progresion for Gluteus Medius Muscle with Consideration for Iliopsoas Tendinitis: An in Vivo Electromyography Study. Am. J. Sports Med. 2011;39(8):1777–1785.
15. Giphart JE, Stull JD, Laprade RF, Wahoff MS, Philippon MJ. Recruitment and activity of the pectineus and piriformis muscles during hip rehabilitation exercises: an electromyography study. Am J Sports Med. 2012;40(7):1654–63. PubMed PMID: 22523373.
16. Enseki KR, Martin R, Kelly BT. Rehabilitation after arthroscopic decompression for femoroacetabular impingement. Clin Sports Med. 2010;29(2):247–55, viii. PubMed PMID: 20226317.
17. Lewis CL, Ferris DP. Walking with increased ankle pushoff decreases hip muscle moments. J Biomech. 2008;41(10):2082–9. PubMed PMID: 18606419. Pubmed Central PMCID: 2562040.
18. Lewis CL, Sahrmann SA. Muscle activation and movement patterns during prone hip extension exercise in women. J Athl Train. 2009;44(3):238–48. PubMed PMID: 19478835. Pubmed Central PMCID: 2681207.
19. National Strength & Conditioning Association (U.S.), Hoffman J. NSCA's guide to program design. Champaign: Human Kinetics; 2012.
20. American College of Sports M. American College of Sports Medicine Position Stand. Progression models in resistance training for healthy adults. Med Sci Sports Exerc. 2009;41(3):687–708. PubMed PMID: 19204579.
21. Lewis CL, Sahrmann SA, Moran DW. Effect of position and alteration in synergist muscle force

contribution on hip forces when performing hip strengthening exercises. Clin Biomech. 2009;24 (1):35–42. PubMed PMID: 19028000. Pubmed Central PMCID: 2677193.
22. Boren K, Conrey C, Le Coguic J, Paprocki L, Voight M, Robinson TK. Electromyographic analysis of gluteus medius and gluteus maximus during rehabilitation exercises. Int J Sports Phys Ther. 2011;6(3): 206–23. PubMed PMID: 22034614. Pubmed Central PMCID: PMC3201064. Epub 2011/10/29. eng.
23. Ekstrom RA, Donatelli RA, Carp KC. Electromyographic analysis of core trunk, hip, and thigh muscles during 9 rehabilitation exercises. J Orthop Sports Phys Ther. 2007;37(12):754–62. PubMed PMID: 18560185. Epub 2008/06/19. eng.
24. Peterson MD, Rhea MR, Alvar BA. Applications of the dose-response for muscular strength development: a review of meta-analytic efficacy and reliability for designing training prescription. J Strength Cond Rese Natl Strength Cond Assoc. 2005;19(4):950–8. PubMed PMID: 16287373. Epub 2005/11/17. eng.
25. Peterson MD, Rhea MR, Alvar BA. Maximizing strength development in athletes: a meta-analysis to determine the dose-response relationship. J Strength Cond Rese Natl Strength Cond Assoc. 2004;18(2):377–82. PubMed PMID: 15142003. Epub 2004/05/15. eng.
26. Dye SF. The knee as a biologic transmission with an envelope of function: a theory. Clin Orthop Relat Res. 1996;325:10–8. PubMed PMID: 8998861.
27. Garrison JC, Shanley E, Thigpen C, Geary R, Osler M, Delgiorno J. The Reliability of the Vail Sport Test as a Measure of Physical Performance Following Anterior Cruciate Ligament Reconstruction. International journal of sports physical therapy. 2012;7(1):20–30.
28. Thorborg K, Petersen J, Magnusson SP, Holmich P. Clinical assessment of hip strength using a hand-held dynamometer is reliable. Scand J Med Sci Sports. 2010;20(3):493–501. PubMed PMID: 19558384.
29. Arnold CM, Warkentin KD, Chilibeck PD, Magnus CR. The reliability and validity of handheld dynamometry for the measurement of lower-extremity muscle strength in older adults. J Strength Cond Rese Natl Strength Cond Assoc. 2010;24(3):815–24. PubMed PMID: 19661831. Epub 2009/08/08. eng.
30. Kraemer WJ, Comstock BA, Clark JE, Dunn-Lewis C. Athlete needs analysis. In: National Strength & Conditioning Association (U.S.), Hoffman J, editors. NSCA's guide to program design. Champaign: Human Kinetics; 2012. p. 1–21.
31. Green HJ. Mechanisms of muscle fatigue in intense exercise. J Sports Sci. 1997;15(3):247–56. PubMed PMID: 9232550. Epub 1997/06/01. eng.
32. Haff GG, Haff E. Training integration and periodization. In: National Strength & Conditioning Association (U.S.), Hoffman J, editors. NSCA's guide to program design. Champaign: Human Kinetics; 2012. p. 213–58.

Outcome Assessment of Non-Arthroplasty Hip Disease

14

Nicholas G. H. Mohtadi

Contents

N.G.H. Mohtadi
Sport Medicine Centre and Section of Orthopaedic Surgery, University of Calgary, Calgary, AB, Canada
e-mail: mohtadi@ucalgary.ca

Abstract

In order to assess the outcome of the treatment of non-arthroplasty hip disease, it is necessary to use validated patient-generated and patient-reported outcome tools. These measures should reflect the patient population with hip disorders who are typically younger and more active. The chapter addresses the who, why, when, where, and what of outcome assessment. It includes descriptions of the most up-to-date validated tools. This includes the NAHS, the iHOT-33 and iHOT-12, HAGOS, and Vail-10 questionnaires. Suggestions for the surgeon contemplating outcome assessment in their patients with hip disorders are made. Future research should provide direct comparison of the outcome measures to provide evidence-based recommendations.

Introduction

The basic tenet in understanding outcome assessment is to appreciate the patient population of interest. In the context of non-arthroplasty hip disease, the focus is patients who are typically younger and more active. The clinical conditions are not likely to be arthritic and the demands of the patients greater than one would expect from someone requiring hip replacement surgery. Defining the focus in terms of treatment (i.e., hip arthroscopy and hip preservation surgery) implies that the patients have the indications for surgical

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_14

treatment and that they have failed nonsurgical methods. However, when considering outcome assessment, it is necessary to start with the patient of interest. This could represent a patient with femoroacetabular impingement (FAI), a torn labrum, a mildly dysplastic hip, pelvic dysfunction, or one of other pre-arthritic conditions. Utilizing outcomes that are capable of measuring a variety of treatments is necessary. Using an outcome that is sensitive enough to measure across the whole spectrum of disease is an obligatory quality. Finally, using similar or identical outcome measures in order to understand the comparisons between nonsurgical and surgical treatment is critical. Without these principles in mind, the assessment of non-arthroplasty hip disease will continue to be an enigma or at worst a process of trial and error with the patients potentially suffering the consequences.

For the purposes of this chapter, outcome assessment will be used synonymously with patient-reported outcome (PRO) assessment. Focusing on PROs should in no way mitigate the importance of utilizing physical assessments, measured functions, or investigations such as pre- and postoperative x-ray evaluations. It is also important to measure the consequences of treatment such as complications or adverse effects. The hip preservation (arthroscopy) surgeon must always weigh the benefits of surgery with the risks. There are examples of where the risks may in fact outweigh the benefits of surgical treatment when randomized trials are conducted to look at surgical compared to nonsurgical treatments.

Outcome Assessment: Who, Why, Where, When, and What?

Who Should Measure Outcomes?

In the simplest sense, everyone should be interested in measuring and understanding outcomes from the patients. This would include allied healthcare practitioners, physicians, healthcare administrators, the healthcare system, and most importantly surgeons. Surgeons are in a unique position when it comes to treating their patients with hip disorders. The surgeon is obligated to provide informed consent. The process of informed consent includes a discussion of treatment alternatives, the risks of surgery, and the benefits of surgery including the expected outcome. It is reasonable for a physician prescribing a medication to expect a consistent effect and, therefore, outcome irrespective of whether the patient is living in South America or Europe. At least the effect on outcome that can be attributed to the individual physician is logically very small compared to a surgeon performing a "prescribed" operative procedure. The surgeon in Europe may have learned different nuances to the technique that are distinctive from the surgeon in North America. The skill of the surgeon clearly has a role to play in outcome, and it is very difficult to measure the magnitude of that role. A surgeon who quotes the risks, benefits, and expected outcomes based on a published report from an "expert" hip arthroscopist may in fact be misleading the patient. The issue to the practicing surgeon is whether they can duplicate or even improve on what is reported in the literature. This fact alone clearly defines one of the most important roles of a surgeon to measure his/her outcomes in a meaningful way in order to provide informed consent.

Why Should We Measure Patient Outcomes?

Notwithstanding the argument used in the previous section, there has been a strong movement toward evaluating outcomes in the last two to three decades. The concerns have been raised long before [1, 2]. In 1914 E.A. Codman endorsed his "end result" idea with respect to surgical outcomes [1, 2]. At the time he apparently urged his colleagues to do the same, which was considered heresy, and Codman was ostracized. Ultimately, he was only recognized for his understanding of "bone tumors and diseases of the shoulder," rather than his main thesis of measuring outcomes [2]. The field of outcome assessment developed slowly thereafter. In the 1970s and 1980s, the need to understand health outcomes led to the development of several outcome measures [3].

"In 1989 the U.S. Congress passed the Patient Outcome Research Act which called for the establishment of a broad-based, patient-centered outcomes research program" [4].

The main reason for measuring outcomes is that the morbidity of musculoskeletal disease in the modern world is a huge problem for healthcare systems. Specifically, hip arthroscopy and hip preservation surgery address conditions that affect patients' quality of life rather than the quantity of life. Understanding how these conditions affect patients and the outcomes of treatment are paramount in determining the allocation of sometimes scarce but ever-increasing costly treatments. Research agencies are also demanding that patient-reported outcomes become the standard when reporting treatment effects rather than traditionally used objective measures.

Furthermore, the science behind developing patient-reported outcomes is well defined, and systematic evaluation of outcomes is becoming commonplace.

Finally, the public is becoming increasingly aware of their individual health status rather than simply having their disease treated. Information available to the patient through the Internet places practitioners and surgeons in a position of rationalizing and explaining the treatments to a greater extent than ever before. Similarly, hospitals and healthcare systems are comparing surgeons and looking at "performance" statistics to allocate resources.

Where Should Patient-Reported Outcomes Be Measured?

Ideally, a patient-reported outcome should be determined in an environment independent of the clinician or surgeon. The majority of modern developed outcomes are self-administered questionnaires that are paper or computer based. More recently tablet or handheld devices and web-based formats are being utilized. This approach provides the patient with convenient options that are likely to benefit compliance and data collection. The most important attribute is to allow the patient to fill in their outcome information independently and in a neutral or nonthreatening environment. This approach reduces the bias of clinician influence or other outside pressures.

When Should Patient-Reported Outcomes Be Measured?

Ideally, the surgeon should measure the PRO at baseline prior to treatment and at an appropriate interval to allow for the patient's health status to change. In this way a well-validated outcome will be able to measure whether or not there is a benefit to the patient.

This question lends itself to a hypothetical example. A surgeon was trying to evaluate the benefit of microfracture treatment of grade III chondral damage on the acetabulum compared to a simple debridement of the damaged surface. The surgeon randomly assigned a sufficient number of patients to one or the other treatment. At baseline the patient groups were similar in all respects including average scores on the hypothetical outcome for Group 1 (debridement group – 35/100) and Group 2 (microfracture group, 37/100). The patients were followed at 6 weeks, 3 months, 6 months, and 1 year. At 6 weeks and 3 months, Group 1 was much better but by 1 year there were no statistical or clinically important differences. The surgeon concluded that microfracture was of no benefit and that debridement was better because of the short-term benefits in the first 3 months. This may be a reasonable and logical conclusion except for the fact that the microfracture group was placed on crutches with no weight bearing allowed for the first 6 weeks. Full weight bearing was achieved by 3 months. The debridement group was allowed full weight bearing by 10 days. Therefore, it is reasonable to assume that the early differences in outcome between the two groups were directly related to the weight bearing status and had nothing to do with the treatment. The 1-year results may be too soon to see any meaningful differences, and 2 years would have been more appropriate. Therefore, when an outcome is measured, it has a bearing on the inferences of treatment effect and the potential benefits to the patient.

What Outcomes Should Be Measured?

The answer to this question creates the most controversy and confusion. The main reason for this debate is that historically utilized outcome measures were nonspecific, developed for an older population with arthritis, evolved from clinician-reported tools, and not truly validated in the modern sense. Nevertheless, these outcomes remain in common usage and have been reported in the literature [5–7].

The first issue when determining the appropriate outcome is to understand what is a patient-reported outcome (PRO)? Outcome measures can be called instruments, tools, scales, scores, indices, measures, outcomes, or questionnaires. These terms are used interchangeably for the purpose of this chapter. Outcome measures can be classified in many ways. The purpose of outcome measures can be classified as either disease-specific, such as those tools created to assess osteoarthritis, or joint-specific, such as those created to assess the outcome of any pathology of the hip. These measures can also be classified according to the person who completes the assessment. Traditionally, outcomes have been assessed by clinicians and include objective measures such as radiographic assessments. The clinician also asks the patient about pain and other subjective measures. These "clinician-based" or "clinician-administered" tools may introduce bias due to the way they are administered but more importantly may not capture the patient's perceived outcomes. The Harris Hip Score was originally a clinician-based tool used to evaluate the outcome of hip arthroplasty surgery in patients with arthritis [7]. This tool was modified by Byrd to allow for a patient- or clinician-reported format [8]. More recently, patient-based and patient-administered tools have been created [9–12]. These patient-reported outcomes (PROs) are typically self-administered and can be completed in a nonthreatening environment to the patient. Patient-reported outcomes are considered to be the reference standard for reporting clinical trials. It is necessary to distinguish between self-administered (i.e., by the patient) outcomes from those which are not only self-administered but are also patient-derived or patient-determined outcomes. There is some debate regarding what constitutes a patient-reported outcome. The commonly accepted definition is "any report coming directly from patients, without interpretation by physicians or others, about how they function or feel in relation to a health condition and its therapy" [13]. This definition works very well for simple outcomes such as measuring pain intensity over time using a visual analogue scale. The PRO takes on a different context when one is attempting to measure more complicated concepts such as quality of life (QoL). A more complete definition of a PRO is that it is collected from a patient but more importantly the "information gained is necessarily of direct concern to the patient" [14]. It is well recognized that the patient perspective is different from that of the clinician and most importantly the surgeon [15]. Therefore, if we accept the first and simple definition of a patient-reported outcome, the patient is the source of the information and it becomes critical to define and/or label the content, construct, or concept of the specific PRO. Typically, this content has included measures "that includes direct subjective assessment by the patient of elements of their health including: symptoms, function, well-being, health-related quality-of-life (HRQoL), perceptions about treatment, satisfaction with care received, and satisfaction with professional communication. The patient is asked to summarize his or her evaluation of the disease, treatment, or health-care system interactions through various modes, providing perceptions related to the condition, its impact, and its functional implications" [16]. It is evident from the literature that there is discussion and debate regarding the definition of a PRO, what context it is measuring, the importance of patient input, and not to mention how it is analyzed and reported. If a surgeon is truly interested in determining the efficacy (i.e., ideal world) or effectiveness (i.e., real world) of a particular surgical treatment, then using a patient-generated and patient-reported outcome would be optimal.

The objective of the tool must also be considered. If the goal is to follow patients over time and to assess changes, an evaluative index is necessary, because it can measure the magnitude of longitudinal change in an individual or a group of individuals [17]. If the objective is to differentiate among patients to determine treatment, a discriminative index should be used, because it distinguishes between individuals or groups [17]. It is very important to understand that the properties of each outcome measure change depending on the objective of the tool. One of the key properties of an evaluative index is the demonstration of responsiveness. Responsiveness refers to the ability of the outcome measure or instrument **to detect within patient change over time** [18]. A discriminative index needs to **differentiate between patients at a particular point in time** [18], in other words being able to distinguish patients with more or less severe "disease" states. Guyatt has explained the differences between these two types of instruments by using the statistical concept of quantifying the signal-to-noise ratio [18]. The better the signal-to-noise ratio, the better the instrument. "If the variability between patients (the signal) is much greater than the variability within patients (the noise), an instrument will be deemed reliable." Discriminative instruments need to be highly reliable, and the questions included in these instruments must enhance the ability to measure variability. Evaluative instruments are subtly different in that they need to detect change over time and responsiveness is a reflection of that change. Responsiveness is "directly related to the magnitude of the difference in score in patients who have improved or deteriorated (the signal) and the extent to which patients who have not changed provide more or less the same scores (the noise)." If the change over time is clinically meaningful, then a responsive instrument will be able to measure whether or not specific treatment (i.e., surgery) has improved a patient's outcome.

Finally, it is very important to understand how each item in a PRO is determined. It is this initial item pool through the process of item generation that is critical [18, 19]. Once a comprehensive item pool is identified, then the final set of items is reduced and formulated into the questionnaire [19]. "The procedure for achieving comprehensiveness is different when selecting an item pool for an evaluative instrument than for either a discriminative or predictive tool" [17]. In a discriminative index it would be important to have the majority of the respondents answer all the questions. In this way, the questionnaire will be able to distinguish between patients by the differences in their scores. In an evaluative index all relevant and important items should be included irrespective of whether an individual patient answers every question. This results in a certain amount of redundancy in an evaluative questionnaire [17].

Patient-Reported Outcomes for Hip Joint Conditions

A recent systematic review of the literature identified three "patient-reported outcomes" for patients with femoroacetabular impingement (FAI) and labral tears [20]. The authors identified the Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC), the Non-arthritic Hip Score (NAHS), and the Hip Outcome Score (HOS). However, critical appraisal of these outcomes would come to the conclusion that the WOMAC is patient-based and self-administered but created for patients who are older than those with FAI and is disease-specific for osteoarthritis [5, 6]. The NAHS is made up of 20 questions, ten of which are taken directly from the WOMAC and the remaining questions determined by consensus from pilot test interviews with patients of varying educational levels as well as with health professionals [9]. The NAHS is designed for this population of patients, but the responsiveness of this questionnaire had not been reported [21]. The HOS was developed to assess activities of daily living and sport participation [22, 23]. However, the HOS did not have any patient involvement in determining the items in the two-component questionnaires [22]. The HOS is patient-reported but not patient derived [22].

A second systematic review focused on patient-reported outcome questionnaires when assessing hip and groin disability [21]. This review suggested that the Hip Dysfunction and Osteoarthritis Outcome Score (HOOS) [24] is recommended for evaluating patients with osteoarthritis and the Hip Outcome Score (HOS) is recommended for patients undergoing hip arthroscopy [21]. They resolved by stating "that a new PRO questionnaire focusing on the evaluation of hip and groin disability in young and physically active patients is needed" [21].

The latest systematic review looked at psychometric evidence of outcomes used in hip arthroscopy. The authors identified the Modified Harris Hip Scale (MHHS), the NAHS, and the HOS as the three possible outcome measures [25]. They evaluated each outcome utilizing the COSMIN checklist [26]. They concluded based on the available evidence for patients undergoing hip arthroscopy that a combination of the NAHS and the HOS should be used as outcome measures [25]. They also stated that "more studies on the validity and reliability of these questionnaires are warranted" [25].

The consistency of outcomes evaluating femoroacetabular impingement also reflects the above systematic reviews [27]. "There was a lack of consensus with regard to reported outcomes (clinical and radiographic) after arthroscopic treatment of FAI. Clinical outcomes reported include the Harris Hip Score (45 %) and the Non-Arthritic Hip Scale (28 %), range of motion (34 %), pain scores (24 %), and patient satisfaction (28 %). The most commonly reported radiographic outcomes included the alpha angle (38 %), head-neck offset (14 %), and degenerative changes (21 %)" [27]. They concluded that "there is significant variation in reported clinical and radiographic outcomes after arthroscopic treatment of FAI" [27].

The Modified Harris Hip Scale (MHHS) [8], the Merle d'Aubigne-Postel Scale (MAPS) [28, 29], the Non-arthritic Hip Score (NAHS), the Larson Hip Score (LHS) [30], the Hip Outcome Score (HOS) [22, 23], and the Rating Scale for Hip Disabilities (RSHD) [31] adopted by the Japanese Orthopaedic Association have all been used in published articles pertaining to hip arthroscopy or hip preservation surgery. Three additional outcomes have recently been reported: the Copenhagen Hip and Groin Outcome Score (HAGOS) [12], a derived score (i.e., the so-called Vail-10) [32], and the International Hip Outcome Tool (iHOT) [11]. Only the NAHS, HOS, HAGOS, IHOT, and the "Vail-10" have specifically targeted the young, active patient with a hip disorder. Ultimately these outcomes should be considered for use in randomized clinical trials and prospective cohort studies in order to evaluate treatments. The outcomes should not only be patient-reported but also reflect all of the important psychometric properties of any evaluative measurement tool.

Non-arthritic Hip Score (NAHS)

The **Non-arthritic Hip Score (NAHS)** was reported in 2003 to assess pain and function in young, active patients with activity-limiting hip pain, both pre- and postoperatively [9]. This tool is a patient-based, self-administered questionnaire (Table 1) that was developed as a modification of the Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC) [5, 6]. Ten questions came directly from the WOMAC, and the other ten were developed de novo. Four of the questions were specific to mechanical symptoms in the hip, and the other six were to measure the patient's activity level. The items were generated through pilot test interviews with patients of varying educational levels and with health professionals. The Non-arthritic Hip Score is intended for 20–40-year-old patients who are experiencing hip pain without an obvious radiographic diagnosis. The tool has been shown to be reproducible with test-retest reliability evaluated between 1 and 16 days. The tool has internal consistency, as assessed using the Cronbach's coefficient alpha. Construct validity was determined by comparing the Non-arthritic Hip Score to the Harris Hip Score and the Short Form-12 in 48 patients. Although this tool attempts to capture a younger population that has not been previously represented by other hip outcomes, the methodology is not ideal because the twenty questions were somewhat arbitrarily determined without statistical support. This may result in a misrepresentation of items that are relevant to a young, active patient with non-arthritic hip problems. In addition, half of the items were taken directly from the WOMAC index, which were generated in an older, more sedentary population. Therefore, the

Table 1 NAHS. Reproduced from [9] with permission from Wolters Kluwer Health

INSTRUCTIONS: The following 5 questions concern the amount of pain you are currently experiencing in the hip that you are having evaluated today. For each situation, please circle the response that most accurately reflects the amount of pain experienced in the past 48 hours. Please circle one answer that best describes your situation.

QUESTION: How much pain do you have-

1. Walking on a flat surface?
   4 = none
   3 = mild
   2 = moderate
   1 = severe
   0 = extreme
2. Going up or down stairs?
   4 = none
   3 = mild
   2 = moderate
   1 = severe
   0 = extreme
3. At night while in bed?
   4 = none
   3 = mild
   2 = moderate
   1 = severe
   0 = extreme
4. Sitting or lying?
   4 = none
   3 = mild
   2 = moderate
   1 = severe
   0 = extreme
5. Standing upright?
   4 = none
   3 = mild
   2 = moderate
   1 = severe
   0 = extreme

INSTRUCTIONS: The following 4 questions concern the symptoms that you are currently experiencing in the hip that you are having evaluated today. For each situation, please circle the response that most accurately reflects the symptoms experienced in the past 48 hours. Please circle one answer that best describes your situation.

QUESTION: How much trouble do you have with-

1. Catching or locking of your hip?
   4 = none
   3 = mild
   2 = moderate
   1 = severe
   0 = extreme
2. Your hip giving out on you?
   4 = none
   3 = mild
   2 = moderate
   1 = severe
   0 = extreme
3. Stiffness in your hip?
   4 = none
   3 = mild
   2 = moderate
   1 = severe
   0 = extreme
4. Decreased motion in your hip?
   4 = none
   3 = mild
   2 = moderate
   1 = severe
   0 = extreme

(*continued*)

Table 1 (continued)

INSTRUCTIONS: The following 5 questions concern your physical function. For each of the following activities, please circle the response that most accurately reflects the difficulty that you have experienced in the past 48 hours because of your hip pain. Please circle one answer that best describes your situation.

QUESTION: What degree of difficulty do you have with-

1. Descending stairs?
   4 = none
   3 = mild
   2 = moderate
   1 = severe
   0 = extreme
2. Ascending stairs?
   4 = none
   3 = mild
   2 = moderate
   1 = severe
   0 = extreme
3. Rising from sitting?
   4 = none
   3 = mild
   2 = moderate
   1 = severe
   0 = extreme
4. Putting on socks/stockings?
   4 = none
   3 = mild
   2 = moderate
   1 = severe
   0 = extreme
5. Rising from bed?
   4 = none
   3 = mild
   2 = moderate
   1 = severe
   0 = extreme

INSTRUCTIONS: The following 6 questions concern your ability to participate in certain types of activities. For each of the following activities, please circle the response that most accurately reflects the difficulty that you have experienced in the past month because of your hip pain. If you do not participate in a certain type of activity, please estimate how much trouble your hip would cause you if you had to perform that type of activity. Please circle one answer that best describes your situation.

QUESTION: How much trouble does your hip cause you when you participate in-

1. High demand sports involving sprinting or cutting (for example, football, basketball, tennis, and exercise aerobics)
   4 = none
   3 = mild
   2 = moderate
   1 = severe
   0 = extreme
2. Low demand sports (for example, golfing and bowling)
   4 = none
   3 = mild
   2 = moderate
   1 = severe
   0 = extreme
3. Jogging for exercise?
   4 = none
   3 = mild
   2 = moderate
   1 = severe
   0 = extreme
4. Walking for exercise?
   4 = none
   3 = mild
   2 = moderate
   1 = severe
   0 = extreme

(*continued*)

Table 1 (continued)

5. Heavy household duties (for example, lifting firewood and moving furniture)? 4 = none 3 = mild 2 = moderate 1 = severe 0 = extreme	6. Light household duties (for example, cooking, dusting, vacuuming, and doing laundry)? 4 = none 3 = mild 2 = moderate 1 = severe 0 = extreme

INSTRUCTIONS: Please add the numbers associated with each of your 20 answers to arrive at the raw score, Multiply the raw score by 1.25 to obtain your hip score.

outcome may be predisposed to ceiling effects, thus limiting its use in the younger, more active population. Baseline average scores on the NAHS are typically in the 50–70 out of 100 range. In addition the sections that address pain, mechanical symptoms, and physical function ask the patient to consider problems in the past 48 h, which may be too short of a time line to be truly representative of the problems that these patients are experiencing. Finally no measure of responsiveness has been reported.

Hip Outcome Score (HOS)

The **Hip Outcome Score (HOS)** was specifically developed for younger more active patients between the ages of 13 and 66 years [22]. The HOS is a patient-administered tool (Table 2) that was designed to assess self-reported functional status; therefore, symptoms were not considered part of the functional assessment. The HOS includes two subscales: activities of daily living (ADL) and sports. Items were generated by physicians and physical therapists and reduced by factor analysis. No patients were involved with item generation. The tool does show internal consistency, as determined by Cronbach's coefficients. True test-retest reliability was not measured since all patients had an intervention (i.e., arthroscopic surgery) between administrations at baseline and an average of 7 months follow-up [23]. The HOS demonstrated good construct validity, as measured by convergent and divergent validity with the Short Form 36 questionnaire using Pearson correlation coefficients. The items in both subscales were shown to be responsive. By design the HOS does not represent a true patient-derived outcome since it does not include items of specific concern to patients such as symptoms and work-related, social, or emotional issues. It should be considered a well-designed and evaluated functional outcome measure. The scoring of the HOS is somewhat complicated since each subscale is scored separately as a percentage score. The ADL subscale has 19 items but only 17 are scored. The items pertaining to sitting and putting on socks and shoes are not included. The sports subscale has 9 items. Each item on both scales is scored from 4 to 0, with 4 indicating "no difficulty" and 0 indicating "unable to do." There is a "not applicable" option as well. The percentage score is calculated by comparing the item total score divided by the highest potential score multiplied by 100.

Copenhagen Hip and Groin Outcome Score (HAGOS) and International Hip Outcome Tool (iHOT)

More recently two newer patient-reported outcomes have been developed: the **Copenhagen Hip and Groin Outcome Score (HAGOS)** [12] and the **International Hip Outcome Tool (iHOT)** [11]. The HAGOS (Table 3) has been

Table 2 HOS. Reprinted from [22], with permission from Elsevier

Please answer **every question** with one response that most closely describes to your condition within the past week.
If the activity in question is limited by something other than your hip mark not applicable (N/A).

Activities of Daily Living subscale

	No difficulty at all	Slight difficulty	Moderate difficulty	Extreme difficulty	Unable to do	N/A
Standing for 15 minutes	☐	☐	☐	☐	☐	☐
Getting into and out of an average car	☐	☐	☐	☐	☐	☐
Putting on socks and shoes	☐	☐	☐	☐	☐	☐
Walking up steep hills	☐	☐	☐	☐	☐	☐
Walking down steep hills	☐	☐	☐	☐	☐	☐
Going up 1 flight of stairs	☐	☐	☐	☐	☐	☐
Going down 1 flight of stairs	☐	☐	☐	☐	☐	☐
Stepping up and down curbs	☐	☐	☐	☐	☐	☐
Deep squatting	☐	☐	☐	☐	☐	☐
Getting into and out of a bath tub	☐	☐	☐	☐	☐	☐
Sitting for 15 minutes	☐	☐	☐	☐	☐	☐
Walking initially	☐	☐	☐	☐	☐	☐
Walking approximately 10 minutes	☐	☐	☐	☐	☐	☐
Walking 15 minutes or greater	☐	☐	☐	☐	☐	☐

Because of your hip how much difficulty do you have with:

	No difficulty at all	Slight difficulty	Moderate difficulty	Extreme difficulty	Unable to do	*N/A*
Twisting/pivoting on involved leg	☐	☐	☐	☐	☐	☐
Rolling over in bed	☐	☐	☐	☐	☐	☐
Light to moderate work (standing, walking)	☐	☐	☐	☐	☐	☐
Heavy work (push/pulling, climbing, carrying)	☐	☐	☐	☐	☐	☐
Recreational activities	☐	☐	☐	☐	☐	☐

(*continued*)

Table 2 (continued)

How would you rate your current level of function during your usual activities of daily living from 0 to 100 with 100 being your level of function prior to your hip problem and 0 being the inability to perform any of your usual daily activitie?

☐☐☐.0 %

Sports subscale
Because of your hip how much difficulty do you have with:

	No difficulty at all	Slight difficulty	Moderate difficulty	Extreme difficulty	Unable to do	N/A
Running one mile	☐	☐	☐	☐	☐	☐
Jumping	☐	☐	☐	☐	☐	☐
Swinging objects like a golf club	☐	☐	☐	☐	☐	☐
Landing	☐	☐	☐	☐	☐	☐
Starting and stopping quickly	☐	☐	☐	☐	☐	☐
Cutting/lateral movements	☐	☐	☐	☐	☐	☐
Low impact activities like fast walking	☐	☐	☐	☐	☐	☐
Ability to perform activity with your normal technique	☐	☐	☐	☐	☐	☐
Ability to participate in your desired sport as long as you would like	☐	☐	☐	☐	☐	☐

How would you rate your current level of function during your sports related activities from 0 to 100 with 100 being your level of function prior to your hip problem and 0 being the inability to perform any of your usual daily activities?

☐☐☐ 0 %

How would you rate your current level of function?

☐ Normal ☐ Nearly normal ☐ Abnormal ☐ Severely abnormal

Table 3 HAGOS. Reproduced from [12] with permission from BMJ Publishing Group Ltd

Today's date:_______/_______/_______Date of birth:_______/_______/_______

Name: ____

INSTRUCTIONS: This questionnaire asks for your view about your hip and/or groin problem. The questions should be answered considering your hip and/or groin function during the **past week** This information will help us keep track of how you feel, and how well you are able to do your usual activities.

Answer **every** question by ticking the appropriate box. Tick only one box for each question. If a question does not pertain to you or you have not experienced it in the past week please make your "best guess" as to which response would be the most accurate.

Symptoms

These questions should be answered considering your hip and/or groin **symptoms** and difficulties during the **past week.**

S1 Do you feel discomfort in your hip and/or groin?

Never	Rarely	Sometimes	Often	Always
☐	☐	☐	☐	☐

S2 Do you hear clicking or any other type of noise from your hip and/or groin?

Never	Rarely	Sometimes	Often	All the time
☐	☐	☐	☐	☐

S3 Do you have difficulties stretching your legs far out to the side?

None	Mild	Moderate	Severe	Extreme
☐	☐	☐	☐	☐

S4 Do you have difficulties taking full strides when you walk?

None	Mild	Moderate	Severe	Extreme
☐	☐	☐	☐	☐

S5 Do you experience sudden twinging/stabbing sensations in your hip and/or groin?

Never	Rarely	Sometimes	Often	All the time
☐	☐	☐	☐	☐

Stiffness

The following questions concern the amount of stiffness you have experienced during the **past week** in your hip and/or groin. Stiffness is a sensation of restriction or slowness in the ease with which you move your hip and/or groin.

S6 How severe is your hip and/or groin stiffness after first awakening in the morning?

None	Mild	Moderate	Severe	Extreme
☐	☐	☐	☐	☐

S7 How severe is your hip and/or groin stiffness after sitting, lying or resting **later in the day?**

None	Mild	Moderate	Severe	Extreme
☐	☐	☐	☐	☐

(*continued*)

Table 3 (continued)

Pain

P1 How often is your hip and/or groin painful?

Never	Monthly	Weekly	Daily	Always
☐	☐	☐	☐	☐

P2 How often do you have pain in areas other than your hip and/or groin that you think may be related to your hip and/or groin problem?

Never	Monthly	Weekly	Daily	Always
☐	☐	☐	☐	☐

The following questions concern the amount of pain you have experienced during the **past week** in your hip and/or groin. **What amount of hip and/or groin pain have you experienced during the following activities?**

P3 Straightening your hip fully

None	Mild	Moderate	Severe	Extreme
☐	☐	☐	☐	☐

P4 Bending your hip fully

None	Mild	Moderate	Severe	Extreme
☐	☐	☐	☐	☐

P5 Walking up or down stairs

None	Mild	Moderate	Severe	Extreme
☐	☐	☐	☐	☐

P6 At night while in bed (pain that disturbs your sleep)

None	Mild	Moderate	Severe	Extreme
☐	☐	☐	☐	☐

P7 Sitting or lying

None	Mild	Moderate	Severe	Extreme
☐	☐	☐	☐	☐

The following questions concern the amount of pain you have experienced during the **past week** in your hip and/or groin. **What amount of hip and/or groin pain have you experienced during the following activities?**

P8 Standing upright

None	Mild	Moderate	Severe	Extreme
☐	☐	☐	☐	☐

P9 Walking on a hard surface (asphalt, concrete, etc.)

None	Mild	Moderate	Severe	Extreme
☐	☐	☐	☐	☐

P10 Walking on an uneven surface

None	Mild	Moderate	Severe	Extreme
☐	☐	☐	☐	☐

Physical function, daily living

The following questions concern your physical function. **For each of the following activities please indicate the degree of difficulty you have experienced in the past week due to your hip and/or groin problem.**

(*continued*)

Table 3 (continued)

A1 Walking up stairs

None	Mild	Moderate	Severe	Extreme
☐	☐	☐	☐	☐

A2 Bending down, e.g. to pick something up from the floor

None	Mild	Moderate	Severe	Extreme
☐	☐	☐	☐	☐

A3 Getting in/out of car

None	Mild	Moderate	Severe	Extreme
☐	☐	☐	☐	☐

A4 Lying in bed (turning over or maintaining the same hip position for a long time)

None	Mild	Moderate	Severe	Extreme
☐	☐	☐	☐	☐

A5 Heavy domestic duties (scrubbing floors, vacuuming, moving heavy boxes etc)

None	Mild	Moderate	Severe	Extreme
☐	☐	☐	☐	☐

Function, sports and recreational activities

The following questions concern your physical function when participating in higher-level activities. Answer **every** question by ticking the appropriate box. If a question does not pertain to you or you have not experienced it in the past week please make your "best guess" as to which response would be the most accurate. **The questions should be answered considering what degree of difficulty you have experienced during the following activities in the past week due to problems with your hip and/or groin.**

SP1 Squatting

None	Mild	Moderate	Severe	Extreme
☐	☐	☐	☐	☐

SP2 Running

None	Mild	Moderate	Severe	Extreme
☐	☐	☐	☐	☐

SP3 Twisting/pivoting on a weight bearing leg

None	Mild	Moderate	Severe	Extreme
☐	☐	☐	☐	☐

SP4 Walking on an uneven surface

None	Mild	Moderate	Severe	Extreme
☐	☐	☐	☐	☐

SP5 Running as fast as you can

None	Mild	Moderate	Severe	Extreme
☐	☐	☐	☐	☐

SP6 Bringing the leg forcefully forward and/or out to the side, such as in kicking, skating etc.

None	Mild	Moderate	Severe	Extreme
☐	☐	☐	☐	☐

(*continued*)

Table 3 (continued)

SP7 Sudden explosive movements that involve quick footwork, such as accelerations, decelerations, change of directions etc.

None	Mild	Moderate	Severe	Extreme
☐	☐	☐	☐	☐

SP8 Situations where the leg is stretched into an outer position (such as when the leg is placed as far away from the body as possible)

None	Mild	Moderate	Severe	Extreme
☐	☐	☐	☐	☐

Participation in physical activities

The following questions are about your ability to participate in your preferred physical activities. Physical activities include sporting activities as well as all other forms of activity where you become slightly out of breath. **When you answer these questions consider to what degree your ability to participate in physical activities during the past week has been affected by your hip and/or groin problem.**

PA1 Are you able to participate in your preferred physical activities for as long as you would like?

Always	Often	Sometimes	Rarely	Never
☐	☐	☐	☐	☐

PA2 Are you able to participate in your preferred physical activities at your normal performance level?

Always	Often	Sometimes	Rarely	Never
☐	☐	☐	☐	☐

Quality of Life

Q1 How often are you aware of your hip and/or groin problem?

Never	Monthly	Weekly	Daily	Constantly
☐	☐	☐	☐	☐

Q2 Have you modified your life style to avoid activities potentially damaging to your hip and/or groin?

Not at all	Mildly	Moderately	Severely	Totally
☐	☐	☐	☐	☐

Q3 In general, how much difficulty do you have with your hip and/or groin?

None	Mild	Moderate	Severe	Extreme
☐	☐	☐	☐	☐

Q4 Does your hip and/or groin problem affect your mood in a negative way?

Not at all	Rarely	Sometimes	Often	All the time
☐	☐	☐	☐	☐

Q5 Do you feel restricted due to your hip and/or groin problem?

Not at all	Rarely	Sometimes	Often	All the time
☐	☐	☐	☐	☐

Thank you very much for completing all the questions in this questionnaire.

developed using standardized format by identifying a specific population of interest, generating items, item reduction, and determination of validity, reliability, and responsiveness. This instrument has the goal of evaluating "hip and/or groin disability related to impairment (body structure and function), activity (activity limitations) and participation (participation restrictions) according to the International Classification of Functioning, disability and health (ICF), in young to middle-aged physically active patients with hip and/or groin pain." The item generation process was determined as a result of a systematic review of the literature. The authors chose to include items from the HOOS and HOS questionnaires [22–24]. Forty-three questions in total (40 from the HOOS and 3 from the HOS) formed the basis of the HAGOS. An expert group, including two orthopaedic surgeons, one physician, and four physiotherapists, added an additional 8 questions. A representative focus group of 20 patients added 2 and removed one question, resulting in a 52-item questionnaire. The item reduction process involved 101 patients. A combination of the frequency and importance of each question to these patients, as well as reliability testing, was used to determine which items should be included. Fourteen items were subsequently removed as a result of further consensus among the authors. One final item was removed as a result of factor analysis resulting in a final questionnaire of 37 questions in six separate subscales: Pain (10 items), Symptoms (7 items), ADL (5 items), Sport/Rec (8 items), PA (2 items), and QOL (5 items) [33]. Content validity was considered due to the patient ($N = 25$) and expert group ($N = 7$) involvement. The questionnaire includes items that are related to soft tissue injury and clearly is distinct from the other questionnaires in this respect. Test-retest reliability was measured 1–3 weeks after baseline in 44 out of the 101 patients and deemed to be very high in all subscales, with intra-class correlation coefficients ranging from 0.82 to 0.91. The authors measured responsiveness at 4 months from baseline in 87 of the 101 patients. They compared the change scores to asking the patients on a 7-point Global Perceived Effect (GPE) score. The correlations with the HAGOS in each subscale were higher than hypothesized. They also measured the standardized response mean (SRM) and effect sizes (ES) on each subscale, which were noticeably higher in patients who had stated that they were "much better" and "better" on their GPE scores. The SRM and ES calculations ranged from 0.90 and 0.77, respectively, for the ADL subscale and 1.46 and 1.78 for the QOL subscale. Construct validity was determined by comparing the HAGOS to the SF-36, which has significant limitations. The SF-36 is a generic outcome measure with likely little relevance to the population at hand. Therefore it is not surprising that the comparison with respect to a priori correlations was satisfactory but not consistent. Finally, PROs should be able to measure the minimal important change (MIC) and/or minimal important difference (MID). The HAGOS showed that the MIC for each subscale ranged from 10 to 15 points based on using the estimate of one half of the reported standard deviation. The authors identified this limitation. If the HAGOS was used as the primary outcome measure in a clinical trial, more patients would be needed in order to achieve a meaningful sample size [12].

The latest PRO advocated for patients with hip disorders is the **International Hip Outcome Tool (iHOT)** [11]. This PRO has previously been called the Hip Quality-of-Life Questionnaire and the MAHORN Hip Outcome Tool (MHOT). Developed with the cooperation of the Multicenter Arthroscopy of the Hip Outcomes Research Network (MAHORN), this tool was designed to address the outcomes of treatment in young active patients with hip disorders. This outcome measure included patients from the USA, Canada, England, and Switzerland. This outcome measure was developed for active patients (18–60 years old; Tegner activity scale ≥ 4) presenting with a variety of hip conditions. This multicenter study recruited patients from the practices of a group of international hip arthroscopy and arthroplasty surgeons. The outcome was created using a process of item generation (51 patients, 4 orthopaedic surgeons, and 4 physiotherapists), item reduction (150 patients), and pretesting (31patients). The questionnaire was tested for test-retest reliability (123 patients), face, content and construct validity

Table 4 iHOT-12. Reprinted from [10], with permission from Elsevier

iHOT[12]

INTERNATIONAL HIP OUTCOME TOOL

NAME

DATE OF BIRTH

TODAY'S DATE

WHICH HIP IS THIS SURVEY ABOUT?

If we've asked you to tell us about one hip In particular, tick that. Otherwise. tick the one which causes most trouble.

O Left

O Right

QUALITY OF LIFE QUESTIONNAIRE FOR YOUNG, ACTIVE PEOPLE WITH HIP PROBLEMS

INSTRUCTIONS

- These questions ask about the problems you may be experiencing in your hip, how these problems affect your life, and the emotions you may feel because of these problems.
- Please indicate the severity by marking the line below each question with a slash.
  - » If you put a mark on the far **left**, it means that you **feel you are significantly Impaired**.
    For example:

    SIGNIFICANTLY IMPAIRED /____ NO PROBLEMS AT ALL

  - » If you put a mark on the far **right**. it means that you do not **think that you have any problems** with your hip. For example:

    SIGNIFICANTLY IMPAIRED____/ NO PROBLEMS AT ALL

  - » If the mark is placed in the middle of the line, this indicates that you are moderately disabled, or in other words, between the extremes of 'significantly impaired' and 'no problems at all'. It is important to put your mark at either end of the line if the extreme descriptions accurately reflect your situation.

TIP If you don't do an activity, imagine how your hip would feel if you had to try it.

- Please let your answers describe the typical situation in the last **month**.

Q1 Overall, how much pain do you have in your hip/groin?

EXTREME PAIN ____ NO PAIN AT ALL

Q2 How difficult is it for you to get up and down off the floor/ground?

EXTREMELY DIFFICULT____ NOT DIFFICULT AT ALL

Q3 How difficult is it for you to walk long distances?

EXTREMELY DIFFICULT____ NOT DIFFICULT AT ALL

Q4 How much trouble do you have with grinding, catching or clicking in your hip?

SEVERE TROUBLE-- NO TROUBLE AT ALL

(continued)

Table 4 (continued)

Q5	How much trouble do you have pushing, pulling, lifting or carrying heavy objects? SEVERE TROUBLE-- NO TROUBLE AT ALL
Q6	How concerned are you about cutting/changing directions during your sport or recreational activities? EXTREMELY CONCERNED-- NOT CONCERNED AT ALL
Q7	How much pain do you experience in your hip *after* activity? EXTREME PAIN-- NO PAIN AT ALL
Q8	How concerned are you about picking up or carrying children because of your hip? EXTREMELY CONCERNED-- NOT CONCERNED AT ALL
Q9	How much trouble do you have with sexual activity because of your hip? ☐ This is not relevant to me SEVERE TROUBLE-- NO TROUBLE AT ALL
Q10	How much of the time are you aware of the disability in your hip? CONSTANTLY AWARE-- NOT AWARE AT ALL
Q11	How concerned are you about your ability to maintain your desired fitness level? EXTREMELY CONCERNED-- NOT CONCERNED AT ALL
Q12	How much of a distraction is your hip problem? EXTREME DISTRACTION-- NO DISTRACTION AT ALL

(51 patients), and responsiveness over a 6-month period in post-arthroscopy patients (27 patients) for a total of 433 patients. Initially, 146 items identified through patient query were reduced to 60 through item reduction and categorized into four domains – (a) Symptoms and Functional Limitations, (b) Sport and Recreational Physical Activities, (c) Job-Related Concerns, and (d) Social, Emotional, and Lifestyle Concerns – and formatted using a visual analogue scale. Pretesting confirmed appropriate wording, content, and formatting. Test-retest reliability showed Pearson correlations greater than 0.80 for 33 of the 60 questions. These 33 questions were formulated into a self-administered questionnaire using a visual analogue scale response format from 0 to 100. A score of “0” reflects the worst possible quality of life and “100” the best possible score.

Table 5 iHOT-33. Reprinted from [11], with permission from Elsevier

iHOT33

INTERNATIONAL
HIP OUTCOME TOOL

NAME

DATE OF BIRTH

TODAY'S DATE

WHICH HIP IS THIS SURVEY ABOUT?
If we've asked you to tell us about one hip In particular, tick that. Otherwise. tick the one which causes most trouble.

O Left

O Right

QUALITY OF LIFE QUESTIONNAIRE FOR YOUNG, ACTIVE PEOPLE WITH HIP PROBLEMS

INSTRUCTIONS

- These questions ask about the problems you may be experiencing in your hip, how these problems affect your life, and the emotions you may feel because of these problems.
- Please indicate the severity by marking the line below each question with a slash.
  - » If you put a mark on the far **left**, it means that you **feel you are significantly impaired.** For example:

    SIGNIFICANTLY / NO PROBLEMS
    IMPAIRED______________________________ AT ALL

  - » If you put a mark on the far **right**. it means that you **do not think that you have any problems** with your hip. For example:

    SIGNIFICANTLY / NO PROBLEMS
    IMPAIRED______________________________ AT ALL

  - » If the mark is placed in the middle of the line, this indicates that you are moderately disabled, or in other words, between the extremes of 'significantly impaired' and 'no problems at all'. It is important to put your mark at either end of the line if the extreme descriptions accurately reflecty our situation.

TIP If you don't do an activity, imagine how your hip would feel if you had to try it.

- Please let your answers describe the typical situation in the last month.

SECTION1 I SYMPTOMS AND FUNCTIONAL LIMITATIONS

The following questions ask about symptoms that you may experience in your hip and about the function of your hip with respect to daily activities. Please think about how you have felt most of the time over the past month and answer accordingly

Q1 How often does your hip/groin ache?

CONSTANTLY______________________________ NEVER

Q2 How stiff is your hip as a result of sitting/resting during the day?

EXTREMELY STIFF______________________________ NOT STIFF AT ALL

Q3 How difficult is it for you to walk long distances?

EXTREMELY DIFFCULT------------------------------ NOT DIFFCULT AT ALL

(*continued*)

Table 5 (continued)

Q4 How much pain do you have in your hip while sitting?

EXTREME PAIN ____NO PAIN AT ALL

Q5 How much trouble do you have standing on your feet for long periods of time?

SEVERE TROUBLE____NO TROUBLE AT ALL

Q6 How difficult is it for you to get up and down off the floor/ground?

EXTREMELY DIFFCULT-- NOT DIFFCULT AT ALL

Q7 How difficult is it for you to walk on uneven surfaces?

EXTREMELY DIFFCULT--NOT DIFFCULT AT ALL

Q8 How difficult is it for you to lie on your affected hip side?

EXTREMELY DIFFCULT -- NOT DIFFCULT AT ALL

Q9 How much trouble do you have with stepping over obstacles?

SEVERE TROUBLE____NO TROUBLE AT ALL

Q10 How much trouble do you have with climbing up/down stairs?

SEVERE TROUBLE ____NO TROUBLE AT ALL

Q11 How much trouble do you have with rising from a sitting position?

SEVERE TROUBLE ____NO TROUBLE AT ALL

Q12 How much discomfort do you have with taking long strides?

EXTREME DISCOMFORT-- NO DISCOMFORT AT ALL

Q13 How much difficulty do you have with getting into and/or out of a car?

EXTREMELY DIFFCULTY--NOT DIFFCULTY AT ALL

Q14 How much trouble do you have with grinding, catching or clicking in your hip?

SEVERE TROUBLE ____NO TROUBLE AT ALL

Q15 How much difficulty do you have with putting on/taking off socks, stockings or shoes?

EXTREME DIFFCULTY-- NOT DIFFCULTY AT ALL

Q16 Overall, how much pain do you have in your hip/groin?

EXTREME PAIN____ NO PAIN AT ALL

(*continued*)

Table 5 (continued)

SECTION 2 I SPORTS AND RECREATIONAL ACTIVITIES

The following questions ask about your **hip** when you participate in sports and recreational activities. Please think about how you have felt most of the time over the past **month** and answer accordingly.

Q17 How concerned are you about your ability to maintain your desired fitness level?

EXTREMELY CONCERNED________________________________ NOT DIFFCULTY AT ALL

Q18 How much pain do you experience in your hip after activity?

EXTREME PAIN________________________________ NO PAIN AT ALL

Q19 How concerned are you that the pain in your hip will increase if you participate in sports or recreational activities?

EXTREMELY CONCERNED________________________________ NOT DIFFCULTY AT ALL

Q20 How much has your quality of life deteriorated because you cannot participate in sport/recreational activities?

EXTREMELY DETERIORATED________________________________ NOT DETERIORATED AT ALL

Q21 How concerned are you about cutting/changing directions during your sport or recreational activities?

☐ I do not do this action in my activities

EXTREMELY CONCERNED________________________________ NOT CONCERNED AT ALL

Q22 How much has your performance level decreased in your sport or recreational activities?

EXTREMELY DECREASED________________________________ NOT DECREASED AT ALL

SECTION 3 I JOB RELATED CONCERNS

The following questions relate to your hip with respect to your current work. Please think about how you have felt most of the time over the past month and answer accordingly.

☐ I do not work because of my hip *(please skip section)*

☐ I do not work for reasons other than my hip *(please skip section)*

Q23 How much trouble do you have pushing, pulling, lifting or carrying heavy objects at work?

☐ I do not do these actions in my activities

SERVICE TROUBLE ________________________________NOT TROUBLE AT ALL

Q24 How much trouble do you have with crouching/squatting?

SERVICE TROUBLE ________________________________NOT TROUBLE AT ALL

(*continued*)

Table 5 (continued)

Q25 How concerned are you that your job will make your hip worse?

EXTREMELY CONCERNED ____________________ NOT CONCERNED AT ALL

Q26 How much difficulty do you have at work because of reduced hip mobility?

EXTREME DIFFICULTY ____________________ NO DIFFICULTY AT ALL

SECTION 4 I SOCIAL, EMOTIONAL AND LIFESTYLE CONCERNS

The following questions ask about social, emotional and lifestyle concerns that you may feel with respect to your hip problem. Please think about how you have felt most of the time over the past month and answer accordingly.

Q27 How frustrated are you because of your hip problem?

EXTREMELY FRUSTRATED ____________________ NO FRUSTRATED AT ALL

Q28 How much trouble do you have with sexual activity because of your hip?

☐ This is not relevant to me

SERVICE TROUBLE ____________________ NOT TROUBLE AT ALL

Q29 How much of a distraction is your hip problem?

EXTREME DISTRACTION ____________________ NO DISTRACTION AT ALL

Q30 How difficult is it for you to release tension and stress because of your hip problem?

EXTREMELY DIFFICULT ____________________ NOT DIFFICULT AT ALL

Q31 How discouraged are you because of your hip problem?

EXTREMELY DISCOURAGED ____________________ NOT DISCOURAGED AT ALL

Q32 How concerned are you about picking up or carrying children because of your hip?

☐ I do not do this action in my activities

EXTREMELY CONCERNED ____________________ NOT CONCERNED AT ALL

Q33 How much of the time are you aware of the disability in your hip?

CONSTANTLY AWARE ____________________ NOT AWARE AT ALL

Intra-class correlation statistic as a reflection of reliability was 0.78, and Cronbach's alpha as a reflection of internal consistency was 0.99. Face and content validity were ensured because of the extensive involvement of patients, the expert developers, and the MAHORN group. Construct validity was demonstrated with a correlation of 0.81 to the Non-arthritic Hip Score. Responsiveness was demonstrated with a paired *t*-test ($p \leq 0.01$), effect size was 1.95, standardized response mean was 1.69, and the responsiveness ratio was 6.7. The calculated minimal clinical important difference (MCID) was 6 points out of the total 100-point scale. These properties make the iHOT very attractive as an outcome tool, since the MCID can be used interchangeably with the MIC, in calculating sample sizes for prospective research studies. As a result this highly validated, truly patient-based, and responsive questionnaire has been recommended for use in randomized clinical trials and prospective cohort studies [11]. An ongoing effort by the MAHORN group continued to develop a 12-item questionnaire, i.e., the iHOT-12 (Table 4), that reflects the longer version, the iHOT-33 [10] (Table 5). The IHOT-12 utilizes 12 of the same questions with similar properties, includes all four domains, and has been recommended for clinical use rather than for research purposes. In this way, busy clinicians with minimal resources can measure a PRO with their patients. The IHOT-12 should be simple enough for phone administration and paper- or computer-based formats and will minimize responder burden for the patients [10].

Vail-10 Questions

The ***"Vail-10 questions"*** represent a derived outcome based upon questions from the NAHS, the MHHS, and the HOS [32]. The authors retrospectively analyzed an extensive database of over 2,000 patients who had completed at baseline the NAHS, HOS, and MHHS and over 1,100 with 1-year follow-up data. They stated that the 10 questions demonstrated criterion validity with no floor or ceiling effects, responsiveness, and construct validity compared to the SF-12 questionnaire [32]. It is unclear at this time whether separating these questions and using them in a prospective way will prove to be valid and reliable.

Summary

The most commonly used outcome measures for patients undergoing nonsurgical, arthroscopic, or hip-preserving surgery have been disease specific (arthritis-based), generic, or derived from historical clinician-based outcome measures. It is important for the practitioner/surgeon to use an outcome that reflects the modern development principles. These would include patient-generated items, reliability, responsiveness, and validity. The practitioner/surgeon should consider the purpose of the outcome assessment. If the purpose is to follow patients over time, then a simple computer-/web-based tool would be best suited. The iHOT-12 represents such an instrument. If the purpose is to perform prospective cohort or randomized clinical trials, then a more robust tool such as the iHOT-33 would be recommended. The HAGOS may be more appropriate for patients with sport injuries since it has questions that address groin as well as hip problems or symptoms. The Vail-10 questionnaire represents a totally derived instrument and therefore is not truly patient derived. The HOS has very good properties but does not have patient input, and the NAHS is a reasonable compromise with half the questions derived and the other half representative of patient and practitioner input. Continued use of the MHHS is not recommended since there are much better tools available today to evaluate non-arthroplasty hip disease. The future research should address head to head comparisons of these questionnaires to determine which should be recommended for clinical and research purposes.

References

1. Codman E. The product of a hospital. Surg Gyn Obst. 1914;18:491–6.
2. Mallon W. E. Amory Codman, surgeon of the 1990s. J Shoulder Elbow Surg. 1998;7(5):529–36.

3. Bergner M, Rothman M. Health status measures: an overview and guide for selection. Annu Rev Public Health. 1987;8:191–210.
4. Ware J. The status of health assessment 1994. Annu Rev Public Health. 1995;16:327–54.
5. Bellamy N, Buchanan WW. A preliminary evaluation of the dimensionality and clinical importance of pain and disability in osteoarthritis of the hip and knee. Clin Rheumatol. 1986;5(2):231–41.
6. Bellamy N, et al. Validation study of WOMAC: a health status instrument for measuring clinically important patient relevant outcomes to antirheumatic drug therapy in patients with osteoarthritis of the hip or knee. J Rheumatol. 1988;15(12):1833–40.
7. Harris WH. Traumatic arthritis of the hip after dislocation and acetabular fractures: treatment by mold arthroplasty. An end-result study using a new method of result evaluation. J Bone Joint Surg Am. 1969;51(4): 737–55.
8. Byrd JW. Hip arthroscopy: patient assessment and indications. Instr Course Lect. 2003;52:711–9.
9. Christensen CP, et al. The nonarthritic hip score: reliable and validated. Clin Orthop Relat Res. 2003;406:75–83.
10. Griffin DR, et al. A short version of the International Hip Outcome Tool (iHOT-12) for use in routine clinical practice. Arthroscopy. 2012;28(5):611–6; quiz 616–8.
11. Mohtadi NG, et al. The Development and validation of a self-administered quality-of-life outcome measure for young, active patients with symptomatic hip disease: the International Hip Outcome Tool (iHOT-33). Arthroscopy. 2012;28(5):595–605; quiz 606–10 e1.
12. Thorborg K, et al. The Copenhagen Hip and Groin Outcome Score (HAGOS): development and validation according to the COSMIN checklist. Br J Sports Med. 2011;45(6):478–91.
13. Patrick DL, et al. Patient-reported outcomes to support medical product labeling claims: FDA perspective. Value Health. 2007;10 Suppl 2:S125–37.
14. Doward LC, McKenna SP. Defining patient-reported outcomes. Value Health. 2004;7 Suppl 1:S4–8.
15. Martin RL, et al. Differences in physician and patient ratings of items used to assess hip disorders. Am J Sports Med. 2009;37(8):1508–12.
16. Patrick DL. Patient-Reported Outcomes (PROs): an organizing tool for concepts, measures and applications. Qual Life Newslett. 2003;31:1–5.
17. Kirshner B, Guyatt G. A methodological framework for assessing health indices. J Chronic Dis. 1985;38(1): 27–36.
18. Guyatt GH, Feeny DH, Patrick DL. Measuring health-related quality of life. Ann Intern Med. 1993;118(8): 622–9.
19. Guyatt GH, Bombardier C, Tugwell PX. Measuring disease-specific quality of life in clinical trials. CMAJ. 1986;134(8):889–95.
20. Lodhia P, et al. Patient-reported outcome instruments for femoroacetabular impingement and hip labral pathology: a systematic review of the clinimetric evidence. Arthroscopy. 2011;27(2):279–86.
21. Thorborg K, et al. Validity, reliability and responsiveness of patient-reported outcome questionnaires when assessing hip and groin disability: a systematic review. Br J Sports Med. 2010;44(16):1186–96.
22. Martin RL, Kelly BT, Philippon MJ. Evidence of validity for the hip outcome score. Arthroscopy. 2006;22(12):1304–11.
23. Martin RL, Philippon MJ. Evidence of reliability and responsiveness for the hip outcome score. Arthroscopy. 2008;24(6):676–82.
24. Nilsdotter AK, et al. Hip disability and osteoarthritis outcome score (HOOS)–validity and responsiveness in total hip replacement. BMC Musculoskelet Disord. 2003;4:10.
25. Tijssen M, et al. Patient-reported outcome questionnaires for hip arthroscopy: a systematic review of the psychometric evidence. BMC Musculoskelet Disord. 2011;12:117.
26. Mokkink LB, et al. The COSMIN checklist for assessing the methodological quality of studies on measurement properties of health status measurement instruments: an international Delphi study. Qual Life Res. 2010;19(4):539–49.
27. Hetaimish BM, et al. Consistency of reported outcomes after arthroscopic management of femoroacetabular impingement. Arthroscopy. 2013;29(4):780–7.
28. Merle D'Aubigne R. Numerical evaluation of hip function. Rev Chir Orthop Reparatrice Appar Mot. 1970;56(5):481–6.
29. Merle D'Aubigne R, Cauchoix J, Ramadier J. Evaluation chiffree de la fonction de la hanch. Application a l'etude des resultats des. Rev Chir Orthop. 1949;35:5–12.
30. Larson CB. Rating scale for hip disabilities. Clin Orthop Relat Res. 1963;31:85–93.
31. Kim KC, et al. Influence of femoroacetabular impingement on results of hip arthroscopy in patients with early osteoarthritis. Clin Orthop Relat Res. 2007;456:128–32.
32. Briggs KK, Philippon MJ. A new validated score to document outcomes following hip arthroscopy. In: International society for hip arthroscopy. Cancun; 2010.

Part II

Operative Basics for Hip Arthroscopy and Open Hip Preservation Surgery

15 Operative Indications for Hip Arthroscopy and Open Hip Preservation Surgery

Asheesh Bedi, William B. Acker II, James R. Ross, and Christopher M. Larson

Contents

A. Bedi (✉)
Department of Orthopaedic Surgery, University of Michigan, Ann Arbor, MI, USA
e-mail: abedi@umich.edu

W.B. Acker II
University of Michigan, Ann Arbor, MI, USA
e-mail: wbacker@med.umich.edu

J.R. Ross
Sports Medicine and Shoulder Service, Department of Orthopaedic Surgery, University of Michigan, Ann Arbor, MI, USA
e-mail: rossjam@med.umich.edu

C.M. Larson
Minnesota Orthopedic Sports Medicine Institute, Twin Cities Orthopedics, Edina, MN, USA
e-mail: chrislarson@tcomn.com

Abstract

The use of hip arthroscopy has rapidly increased over the past 30 years and investigations into its use have seen an exponential increase over the past decade. Advancements in techniques and instrumentation have allowed an increasing number of orthopedists to perform this operation to address a rapidly expanding number of disorders of the hip and adjacent anatomy. Hip arthroscopy is most commonly performed for intra-articular conditions such as FAI, labral and chondral lesions, disorders of the synovium and capsule, loose bodies, ligamentum teres injuries, and septic arthritis and as a diagnostic aid in conjunction with other procedures. Periarticular conditions such as greater trochanteric pain syndrome, snapping hip, extra-articular FAI, and pathology in the posterior gluteal space are also increasingly addressed with the aid of hip arthroscopy. Contraindications to the procedure, as its strengths and limitations are better defined through increased study and use, have also evolved. These include the presence of advance stages of osteoarthritis, inflammatory arthritis, various forms of hip dysplasia, chronic muscle pathology, preexisting neurologic injury, and greater trochanter impingement, among others. This chapter will expand upon these indications and contraindications, reviewing evidence to help guide both

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_15

beginner and advanced hip arthroscopists. As the use of hip arthroscopy continues to expand, the utility and limitations of its use will continue to evolve.

Introduction

Although initially described in 1931, hip arthroscopy has been gaining popularity in the medical field only since the 1980s [1, 2]. Indeed, the overall incidence of its use has been estimated to have increased eighteen-fold among recently trained orthopedists [3]. Additionally, there has been an exponential increase in the number of investigations related to hip arthroscopy and hip preservation in the last 5–10 years. The use of hip arthroscopy was initially limited due to the technical difficulties presented by the anatomy of the hip joint, which, compared to other joints, presents additional challenges with respect to safe accessibility and maneuverability. Entry and exploration of the joint were more challenging due to the thick encompassing soft tissue envelope, thick joint capsule, constrained position of the femoral head within the acetabulum, proximity of neurovascular structures, and lack of instrumentation capable of handling the depth of the joint [4]. The development of specific instrumentation and improved techniques in exposure and positioning, including traction and joint distraction, as well as improved capsular management have allowed for greater accessibility to the joint and subsequently have expanded the indications for hip arthroscopy [5]. In the hands of experienced surgeons, hip arthroscopy is a minimally invasive procedure that may offer the potential for decreased morbidity to periarticular musculature, diminished risk of neurovascular injury, and shorter recovery periods compared to traditional open exposures to the hip [2].

Indications

See Table 1.

Table 1 Current indications of hip arthroscopy

Intra-articular	Periarticular
FAI (cam and pincer type)	Greater trochanteric pain syndrome
Labral pathology	Snapping hip syndromes
Chondral lesions	Proximal hamstring repair
Ligamentum teres injuries	Sciatic nerve entrapment
Loose bodies/synovial chondromatosis	FAI (ischiofemoral and AIIS/subspine type)
Septic arthritis	
Synovial-based diseases	
Adhesive capsulitis	
Capsular laxity and instability	
Staged interventions	
Adjunct to total hip replacement	

Intra-articular (Central Compartment) Pathology

Femoroacetabular Impingement (FAI)

Femoroacetabular impingement (FAI) is a disorder of the hip joint that results in abnormal osseous contact between the femoral head-neck junction and acetabulum that can lead to chondral and/or labral pathology [6]. Although some individuals with radiographic FAI are asymptomatic, recurrent cyclical impingement may result in pain and discomfort among patients and is one of the predominant causes of arthritis in the nondysplastic hip [7, 8].

Ganz and colleagues pioneered the open surgical dislocation approach via a trochanteric osteotomy as an effective and reproducible technique to safely address FAI in symptomatic patients [9]. Advances in arthroscopic techniques have allowed for equivalent correction of femoral offset and focal rim impingement when compared to surgical dislocation of the hip [10]. Recent literature even suggests that arthroscopy may provide equal or perhaps greater improvement in outcomes compared to open surgical dislocation for the treatment of FAI, with a lower reoperation and complication rate [11, 12]. Arthroscopy also minimizes trauma to the periarticular soft tissues without the need for trochanteric osteotomy, which may allow for an earlier return to full activities and decreased abductor dysfunction. Although superoposterior cam and even posterior and posterior-inferior acetabular deformities can be addressed arthroscopically in expert hands, global acetabular deformities (i.e., protrusio acetabuli, acetabular retroversion) are likely better addressed through open approaches to allow for comprehensive correction of these global and extensive pathomorphologies, which may contribute to hip pain [13].

Labral Pathology

The acetabular labrum is a ring of fibrocartilage that acts as a "suction seal" to ensure continuous lubrication of the hip joint and contributes to favorable joint stability and kinematics by distributing contact forces via load sharing and deepening of the hip joint [14, 15]. Labral damage may result in painful clicking and locking, reduced range of motion, and interference with daily activities [16, 17]. Labral pathology most commonly occurs in the form of a tear and can be secondary to FAI, dysplasia, degeneration, or both major and minor trauma. During surgical treatment of labral tears, the labrum is typically debrided or repaired and/or refixed based on tear pattern and healing potential, depending on tissue quality and vascularity [18].

While labral pathology has been found to occur most commonly in the anterior and superior margins of the acetabulum, the location typically reflects the areas of mechanical conflict between femoral and acetabular pathomorphology [19]. This osseous pathomorphology must be addressed in addition to the labral damage to avoid recurrent injury and a modest clinical outcome. In the setting of acetabular dysplasia, anterosuperior, superior, and posterior abnormalities of a hypertrophic labrum have been identified [20]. The objectives of labral preservation are to treat the symptoms and pain and restore the hip seal and thus stability. Additionally, labral refixation or repair is performed with the goal of potentially preventing the premature development of arthritis associated with abnormal contact forces on the articular cartilage, which has been shown to correlate with labral tears [21]. Studies have suggested that labral repair leads to superior outcomes when compared to debridement and/or excision as part of a joint preservation procedure [22–25]. Therefore, one should attempt to repair labral tears with good-quality tissue; if this is not possible, however, labral debridement has also been shown to have good clinical outcomes [26].

Chondral Lesions

The articular cartilage surfaces of the hip provide a smooth, almost frictionless articulation of the femoral head and acetabulum. Insults to the chondral surfaces can occur traumatically, both acutely from subluxation/dislocation/lateral impaction and chronically from cyclical femoroacetabular impingement (FAI) or as a result of a degenerative process, such as the static overload that occurs in dysplasia. Injury can occur on either the articular surface of the femoral head (more common with acute trauma) or on the chondral surface of the acetabulum, as is typical with FAI [1].

Chondral lesions of the acetabulum are commonly associated with intra-articular hip disorders and reflect a morphological incongruity between an aspherical femoral head-neck junction and the acetabular geometry. Loss of normal sphericity and offset at the head-neck junction, as is characteristic in the setting of FAI, can cause a delamination of the chondral surface of the acetabulum via cyclical wear. The resultant damaged cartilage can be a source of pain and mechanical symptoms. Arthroscopy allows for inspection of the chondral surfaces of the hip and identification of chondral lesions and any indicated debridement and/or marrow-stimulation techniques (e.g., microfracture or drilling) to treat these lesions [10, 27, 28]. As arthroscopic techniques evolve, whole-tissue transplantation of autograft or allograft to repair severe osteochondral defects may become possible.

Chondral lesions of the femoral head are less common than those on the acetabulum. The femoral head cartilage is thinner than the acetabular cartilage, and preparation of an adequate border for marrow-stimulation cartilage techniques is also more difficult. The success of mosaicplasty procedures in the knee has led to their application in other joints, including the femoral head [29, 30]. These procedures, however, were described via an open approach, and the application of this treatment method has yet to be described arthroscopically. As techniques continue to advance, it is likely that smaller, accessible lesions will be treated in this manner.

The risk of iatrogenic chondral injury during arthroscopy and the potential long-term consequences of such injury deserve special mention. These injuries to friable cartilage have been shown to heal poorly spontaneously, and no perfect arthroscopic treatment for smaller defects has been reported [31]. As hip arthroscopy continues to gain in popularity, great caution should be exercised by new and experienced hip arthroscopists during joint entry and surgical maneuvers to avoid creating new or exacerbating previous chondral lesions.

Ligamentum Teres Injuries

The ligamentum teres is a strong, intra-articular ligament that is thought to be important for the stabilization of the hip, particularly in adduction, flexion, and external rotation, the position of greatest posterior instability of the hip joint [32]. Lesions of the ligamentum teres include partial or complete traumatic tears, degenerative tears, and avulsion fractures at the foveal insertion of the femoral head [33]. Traumatic hip subluxations or dislocations have a high incidence of complete or partial tears of the ligamentum teres [34]. Chronic hip joint inflammation may also lead to degeneration of the ligament or alternatively can be indicative of hip instability [2].

Ligamentum teres injuries are difficult to diagnose and patients may present with mechanical hip pain and describe painful locking, clicking, or giving way. Arthroscopy is an effective technique that has greatly enhanced diagnosis of such injuries and is almost the single treatment modality used to resect or debride the ligament, although reconstruction techniques have been described despite limited evidence [32]. When significant tears in the ligamentum teres are encountered in the absence of degenerative change, traumatic (subluxation) and atraumatic (dysplasia/multidirectional instability) instability should be suspected. Foveal avulsions are associated with acute or repetitive hyperabduction of the hip. Classification systems of ligamentum teres injury via arthroscopic assessment have been described. Based on their arthroscopic findings, Gray and Villar [35] classified ligamentum teres tears into 3 types: type I, complete rupture; type II, partial rupture; and type III, degenerative tear. Botser et al. [36] proposed a new classification system to categorize the ligamentum teres tears, given the high prevalence of partial (type II) tears when classified according to Gray and Villar. This new classification includes grade 0, no tear; grade 1, low-grade partial tear <50 %; grade 2, high-grade partial tear >50 %; and grade 3, full-thickness tear [36].

Septic Arthritis

Septic arthritis is a bacterial, viral, or fungal infection of the hip joint. The infection can cause acute chondrolysis and irreversible damage to joint articular surfaces that initially causes pain, warmth, and swelling and, if left untreated, may lead to osteomyelitis, sepsis, and eventually osteoarthritis of the joint. Septic arthritis of the hip is common in young children, in whom it is thought to be secondary to hematogenous spread of the offending agent, aided by the child's rapid growth and robust vascularity, and in immunocompromised and elderly adults.

Open arthrotomy with adequate irrigation and debridement has been considered the standard treatment of patients with septic arthritis of the hip. Arthroscopic drainage of septic arthritis of the hip has been used as an alternative to open arthrotomy given the success that has been accomplished in eradicating septic arthritis of the knee with arthroscopic treatment [37]. Previous authors have reported case series on the effective treatment of septic arthritis of the hip with arthroscopy [38–43]. In a comparative study, El-Sayed showed equal eradication of infection at greater than 12-month follow-up with no recurrence or development of complications when comparing arthroscopic versus open treatment of septic arthritis [44]. The authors, however, emphasized early diagnosis and prompt treatment as the most important factors in successful treatment.

The patients in the arthroscopic group did have a significantly shorter duration of hospital stay; however, overall recovery time was not affected by the method of treatment. Arthroscopic drainage of septic arthritis of the hip appears to be a valid alternative to an open arthrotomy, especially in cases that are diagnosed in an acute setting and in the hands of an experienced arthroscopist.

Loose Bodies/Synovial Chondromatosis

Loose bodies are small fragments of bone, cartilage, or diseased synovium that are typically mobile within the hip, either in the central or peripheral compartments. These fragments may develop as a result of trauma or from reactive bone or synovial formation due to degenerative or inflammatory processes. Loose bodies typically cause mechanical symptoms such as popping, catching, and locking [16]. Due to the variable location and composition of loose bodies, physical exam and radiological imaging are unreliable diagnostic tools. Hip arthroscopy has become a valuable tool allowing for direct visualization and treatment of loose bodies in a minimally invasive fashion [45].

A large number of small loose bodies may also be the product of primary or secondary synovial chondromatosis or osteochondromatosis. Primary synovial chondromatosis is a proliferative disease affecting the joint synovium. Synovial membrane metaplasia enlarges and typically calcifies and breaks away, thus becoming free to enter the joint compartments. Once free, the loose body typically causes pain and mechanical symptoms [46]. Secondary synovial chondromatosis is more common and typically occurs secondary to trauma. Damage to articular cartilage as a result of trauma can result in loose chondral fragments that may or may not calcify. Hip arthroscopy allows for identification and removal of these fragments and also affords the opportunity for simultaneous treatment of the damaged chondral surface [46]. Radiologic imaging, despite the use of advanced techniques such as computed tomography and magnetic resonance arthrograms, frequently fails to identify these fragments, which may consist only of cartilage, leaving arthroscopy as the best tool for their identification and removal [1].

Synovial Diseases

The synovial membrane is a thin layer of soft tissue that lines the inner surface of the hip joint capsule and functions to produce and maintain the volume of lubricating joint fluid optimal for joint motion and function. The synovial lining of the hip can degenerate over time secondary to trauma, repetitive stress, and/or a variety of inflammatory arthropathies, such as synovial chondromatosis, rheumatoid arthritis (an autoimmune-induced synovial inflammation), and pigmented villonodular synovitis (PVNS, a synovial lining hypertrophy with synovial fluid overproduction).

Arthroscopy in the setting of synovial disease allows for minimally invasive treatment and also definitive diagnosis of the offending disorder. Arthroscopic synovectomy has been shown to slow deterioration of the articular cartilage and preserve hip function [45]. Focal PVNS located within the lunate fossa and inferomedial femoral neck region can be effectively treated and eradicated arthroscopically, while more diffuse disease, which commonly extends and proliferates outside the joint space into adjacent soft tissues, may warrant more thorough excision via surgical dislocation. Arthroscopy also provides an opportunity for biopsy of the synovium in a minimally invasive fashion to confirm the diagnosis of inflammatory arthropathy and guide subsequent treatment with appropriate disease-modifying agents.

Adhesive Capsulitis

Adhesive capsulitis of the hip, only recently recognized in 1999 and found to be more prevalent than previous literature indicated, is a newer indication for hip arthroscopy [47]. It is similar to adhesive capsulitis of the shoulder in that the patient experiences pain and loss of range of motion secondary to an inflammatory process of the joint capsule. However, this pathology can be nonspecific in the presence of other hip pathologies that cause pain and a decreased passive range of motion of the joint, such as FAI.

Adhesive capsulitis is likely underreported in the literature relative to shoulder adhesive capsulitis as decreased range of motion of the more constrained hip is perhaps less functionally noticeable and limiting for patients when compared to the shoulder [48]. Arthroscopy can effectively treat patients with adhesive capsulitis of the hip in a minimally invasive fashion via capsulotomy or capsulectomy of the pathologically thickened capsule and synovectomy of any associated intra-articular reactive tissue pathology.

Capsular Laxity and Instability

The cause of capsular laxity and hip instability can be divided into traumatic and atraumatic etiologies. Traumatic injuries can result in capsular incompetence with or without associated labral damage. Atraumatic hip instability can be the consequence of overuse and may result in anterior subluxation secondary to repetitive external rotation with axial loading. Other individuals may be predisposed to hip instability due to general ligamentous laxity, various degrees of acetabular dysplasia, or connective tissue disorders such as Ehlers-Danlos syndrome [48, 49].

Capsular laxity, regardless of cause, can be associated with hip pain, instability, and subluxation events. Arthroscopic capsular or labral repair or reconstruction may be beneficial for patients with recurrent hip instability, particularly in the setting of prior trauma [49]. Several recent case series have proposed that the structural abnormalities associated with FAI may predispose patients to traumatic posterior hip instability and subsequent subluxation events, with one series reporting favorable outcomes with arthroscopic osteoplasty and labral refixation [50, 51]. However, these procedures should be approached with caution in atraumatic cases and primarily utilized in patients with capsular or labral insufficiency in the setting of normal bony morphology. In the setting of acetabular dysplasia, the hip is best addressed via an open procedure, such as a periacetabular osteotomy, to restore the depth, congruity, and kinematics of the joint and correct the underlying structural pathomorphology that has compromised the soft tissue envelope.

Staged Interventions

Acetabular dysplasia, the most frequently encountered form of structural instability of the hip joint, can lead to static overload of the hip and resultant pain from damage to cartilaginous surfaces, hypertrophy and degeneration of the labrum, and hypertrophy and/or tearing of the ligamentum teres [52]. Correction of a shallow acetabulum is most commonly, completely successfully addressed with a periacetabular osteotomy (PAO), which, by reorienting the acetabulum into a better fit position, improves load sharing and reduces abnormal contact forces at the acetabular rim. The PAO may be performed with an anterior arthrotomy to address any intra-articular pathology or in the setting of combined cam-morphology, but arthroscopy in conjunction with a PAO is increasingly used because it affords an opportunity for more precise diagnosis, classification, and treatment of associated intra-articular pathology [52]. A recent study by Ross et al. demonstrated that labral and chondral pathology may be as high as 86 % and 69 %, respectively, which if left unaddressed could be a potential source of residual hip pain after PAO [53].

With any level of deformity, arthroscopy can be an effective adjunct tool to a more powerful extra-articular osteotomy; however, one should be cautious when utilizing this as an isolated treatment of dysplasia because extra-articular osteotomy typically provides more definitive correction of the acetabular and femoral morphology [54]. Isolated arthroscopy in the setting of acetabular dysplasia may result in iatrogenic instability with or without bony resections, labral debridements, and capsulotomies, which have been documented on multiple occasions [55–58].

Total Hip Replacement

Arthroscopy in the setting of a painful total hip arthroplasty may be used to evaluate the integrity of implants, assess component wear, remove loose acetabular screws, and perform debridements of soft tissue impingement or infection [16].

There have also been reports of psoas pain and impingement after hip arthroplasty with associated component malpositioning (relative cup retroversion, oversized components, and leg-length inequalities) [59]. Definitive treatment with psoas tenotomy or component revision and tendon debridement has proven successful, but revision arthroplasty is associated with a significant rate of complications and risks, particularly in elderly patients [60]. To correct this source of impingement, psoas lengthening or release may be performed arthroscopically to reliably improve pain and function. Similar outcomes have been documented after revision hip arthroplasty and psoas tenotomy in carefully selected individuals [61]. In some cases, psoas pain has been documented without any apparent impingement. A thickened and tensioned psoas may be seen in this setting, and an arthroscopic transcapsular or lesser trochanteric lengthening can be performed [59]. A release at the level of the lesser trochanter may be more desirable due to easier exposure without the risk of iatrogenic infection or instability secondary to capsular damage required to enter the joint.

Periarticular (Peripheral Compartment) Pathology

Greater Trochanteric Pain Syndrome

Greater trochanteric pain syndrome (GTPS) is an entity encompassing several pathologies that cause chronic lateral hip pain in the region of the greater trochanter [5]. GTPS is relatively common, reportedly affecting up to 10–25 % of the population [62]. Trochanteric bursitis is the most common form of GTPS and involves inflammation of the bursa between the trochanteric facets and the gluteus medius, the gluteus minimus, and the iliotibial band caused by repetitive trauma, commonly from a snapping tendon, as discussed below. In addition to chronic inflammation as a source of pain, tears in the abductor tendons and musculature can also result and contribute to pain generation, analogous to rotator cuff tears in the shoulder. The gluteus medius, which inserts on the lateral and posterior facets of the greater trochanter, is most commonly torn along its articular side, and akin to the shoulder, insertional medius tears can be partial (most often), intrasubstance, or complete [63]. GTPS can be effectively treated with arthroscopic bursectomy, iliotibial band release, and/or tendon repair to the greater trochanter depending on the offending anatomy [64]. Articular surface partial-thickness tears can be difficult or impossible to visualize from the peritrochanteric space; thus, recently transtendinous repair of these lesions has been advocated [63].

Snapping Hip Syndromes

Snapping hip syndrome is characterized by an audible (internal coxa saltans) or visible (external coxa saltans) snapping of the hip when the joint is in motion and may be accompanied by pain. Sources of the snapping can include loose bodies in the joint and extra-articular causes including a thickened iliotibial band or gluteus maximus (external coxa saltans) which may snap over the greater trochanter when the hip is flexed and then extended. A snapping hip may also be the result of the iliopsoas tendon (internal coxa saltans) displacing over the iliopectineal eminence, anterior inferior iliac spine (AIIS), acetabular rim, or femoral head. Friction from repetitive snapping leads to chronic inflammation and potential tears of the offending tendon. Asymptomatic snapping requires no treatment; however, arthroscopic procedures for recalcitrant symptoms are effective at addressing pain from a snapping tendon [65, 66]. Techniques include the removal of osseous impingements and/or the release or lengthening of the iliopsoas or iliotibial band to alleviate symptoms [67]. Care should be taken in patients with increased acetabular or femoral retroversion, however, as the psoas tendon may act as a dynamic stabilizer of the hip and lengthening procedures may lead to functional weakness and pain [18].

Proximal Hamstring Repair

Avulsion of the hamstring off the ischial tuberosity is a rare injury that occurs during forceful hip flexion and knee extension [68]. Waterskiing accidents are the most common cause of proximal hamstring avulsions. Reattachment of two or three tendon avulsion injuries with significant retraction is often performed in young and active patients with favorable outcomes and a high rate of return to sport [69, 70]. As the use of extra-articular arthroscopy about the hip continues to expand, the role of endoscopic repair of proximal hamstring tendon avulsions is currently being explored [71]. Arthroscopic approach to these injuries should be restricted to only those arthroscopists with adept skills and thorough anatomic knowledge of the deep gluteal space. There are currently no studies that have defined the outcomes and complications associated with arthroscopic repair and whether this technique offers any benefit over traditional open techniques. The presence of the sciatic nerve and gluteal vasculature in the deep gluteal space mandate that care be taken to visualize and protect these vital structures during this procedure. Currently, this application may be more predictably used for treatment of recalcitrant proximal hamstring tendinopathy with high-grade tears. Areas of tendinosis can be debrided, and creation of a bleeding bony bed at the ischial tuberosity aids with tendon repair. Chronic ruptures, which may require reconstruction with allograft material, are currently best treated via an open surgical approach [72].

Sciatic Nerve Entrapment

The sciatic nerve passes through the sciatic notch intimately in association with the piriformis muscle, and injury to this muscle, which results in spasm or contracture, may compress the nerve and lead to symptoms [73]. Approximately 17 % of patients may have variations in the course of the sciatic nerve in relation to the piriformis that must be considered [74]. The sciatic nerve may exit through the piriformis (0.5 %), a portion of the nerve may exit through the piriformis (13.7 %), or it may be double branched with the piriformis sandwiched between the nerve branches (1.3 %). Pain can also be secondary to nerve entrapment or compression by the hamstring origin and quadratus femoris/gemellus inferior and obturator internus/gemellus superior muscles or scar tissue, commonly described as fibrous bands [75]. Nerve entrapment is aggravated by hip flexion and internal or external rotation and can cause pain over the buttocks that can radiate down the dorsal thigh. The nerve normally accommodates hip movement, but nearby inflamed or contracted muscle reduces the space available for nerve excursion.

Endoscopic treatment has been used to reduce pain associated with sciatic nerve compression by addressing each offending structure; however, this treatment can be ineffective if tethering structures are too proximal in the pelvis or distal in the thigh [75]. Endoscopic decompression of the sciatic nerve requires accurate knowledge of the periarticular space and precise surgical technique to ensure all potential offending structures are addressed. Sciatic nerve decompression is a relatively new indication for hip endoscopy, and as such it is advisable that this approach be performed with great caution and only by surgeons familiar with the anatomy of the subgluteal space.

Extra-articular FAI

Extra-articular hip impingement commonly results from either ischiofemoral or AIIS/subspine impingement [76–78]. Ischiofemoral impingement occurs between the lesser trochanter and ischium, which may result in varying degrees of injury to the quadratus femoris [79]. Subspine impingement, on the other hand, is thought to result from osseous contact between a prominent AIIS and the femoral neck with hip flexion. Hetstroni et al. have recently classified the AIIS morphology, and one must be aware of the variations in the anatomy, as a low-lying AIIS may be mistaken as a false-positive crossover sign on an anteroposterior pelvic radiograph [80, 81]. Several articles have shown that decompression of the offending bony anatomy with arthroscopic or endoscopic approaches has led to improved outcomes [76–78].

Extra-articular greater trochanteric/pelvic impingement, classically seen in the setting of Legg-Calve-Perthes disease, is best treated with open surgical approaches that include relative femoral neck lengthening, greater trochanter distalization transfers, or both. Given that these sources of FAI are relatively new concepts, additional follow-up with larger patient populations is needed to better define outcomes and the optimal surgical procedures to address these sources of extra-articular impingement disorders.

Contraindications

Successful outcome of arthroscopy or endoscopy of the hip requires careful patient selection and a keen recognition of technical factors that may preclude the procedure or compromise optimal clinical outcomes (Table 2).

Absolute

In cases of severe osteoarthritis, where the articular cartilage has been fully denuded, arthroscopy should not be performed as universally poor results have been reported [49, 82, 83]. Outcomes after arthroscopic debridement or lavage in joints with severe osteoarthritic changes were found to be no better than those after a placebo procedure [84]. There is also good evidence demonstrating inferior outcomes and a higher rate of conversion to total hip arthroplasty in patients with a radiographic joint space less than 2 mm [85, 86]. In the case of a septic joint with infectious migration beyond the synovial matrix, arthroscopy should not be performed, and an open arthrotomy should be utilized if osteomyelitis is suspected [87]. Ankylosis of the joint is an important absolute contraindication, as arthroscopic instruments cannot be safely employed if the hip cannot be distracted or distended properly [1]. Dysplastic features with femoral head migration (>1 cm lateral or break in Shenton's line) indicate more severe structural instability and open, corrective approaches are more appropriate for the global correction required. Symptomatic greater trochanteric impingement cannot be reliably managed with an arthroscopic or endoscopic approach and open procedures should be considered in the presence of disabling extra-articular impingement. Finally, rim resection in the presence of severe acetabular retroversion can exacerbate instability from a posteriorly deficient acetabulum, and thus an anteverting periacetabular osteotomy should be considered in these situations.

Table 2 Contraindications of hip arthroscopy/endoscopy

Absolute contraindications	Relative contraindications
Advanced osteoarthritis	Obesity/deconditioning
Septic joint spread	Moderate osteoarthritis
Ankylosis	Dysplasia
Dysplasia with femoral head migration	Inflammatory arthritis
Greater trochanteric impingement	Neurological injury
Severe acetabular retroversion	Chronic proximal hamstring avulsions
	Chronic abductor avulsions with severe retraction and fatty atrophy
	Internal snapping hip with severe femoral neck anteversion

Relative

Obesity multiplies the technical challenges posed by hip arthroscopy and increases the risk of potential complications. Additionally, current instrumentation utilized for hip arthroscopy may not have sufficient length to access and instrument the hip joint [19]. Obesity and deconditioning also make full compliance with rigorous postoperative rehabilitation more difficult. Arthroscopic procedures are less effective in the presence of mild to moderate osteoarthritis, dysplasia, and inflammatory arthritis; indeed in the setting of preexisting radiographic degenerative change, high failure rates have been recorded [82]. These findings illustrate the importance of careful evaluation of osseous morphology and articular cartilage status on imaging studies preoperatively. Arthroscopy may be a useful adjunct tool for the diagnosis and treatment of intra-articular pathology in the setting of acetabular dysplasia but, as mentioned previously, is limited in its corrective ability and should typically be employed in combination with more powerful open procedures to address abnormal femoral and acetabular pathomorphology. Impingement resulting from more subtle dysplastic features, however, may be adequately addressed with carefully planned arthroscopy and care not to resect the acetabular rim and create iatrogenic instability. Capsular management and adequate repair is also important in these patients with borderline acetabular dysplasia, as they may rely on the capsule as a source of stability. Arthroscopic procedures may also be contraindicated for patients with known neurologic injury or disorders, such as pudendal nerve neuralgia, as hip traction may cause a "double-crush" phenomenon and risk further neurologic impairment [88]. Chronically retracted proximal hamstring and abductor tendon avulsions can be technically challenging scenarios, often requiring allograft reconstruction, and as such open approaches may more reliably address these situations. Finally, internal snapping hip caused by severe femoral neck anteversion may be more safely addressed with a derotational femoral osteotomy in an effort to avoid potentially further destabilizing the hip anteriorly by releasing the psoas tendon, which may be a secondary stabilizer in these patients.

Summary

Until recently, arthroscopy of the hip was not widely endorsed due to the complexity of a deep joint with thick surrounding tissue envelope and a constrained alignment of the osseous structures. Currently arthroscopic and endoscopic hip procedures are rapidly evolving into minimally invasive orthopedic procedures that can be safely and effectively performed to address a growing number of disorders involving the hip and pelvis. The use of hip arthroscopy and endoscopy has not only allowed for the precise diagnosis and treatment of hip injuries and disorders but has also advanced understanding of intra- and periarticular hip pathology. Indications and contraindications will continue to evolve as new technological advances and longer-term and larger-scale outcomes are reported. Adherence to evidence-based indications and contraindications will aid to optimize patient outcomes from arthroscopic and endoscopic procedures of the hip.

References

1. Byrd JW. Hip arthroscopy. J Am Acad Orthop Surg. 2006;14(7):433–44.
2. Burman MS. Arthroscopy or the direct visualization of joints. J Bone Joint Surg Am. 1931;4:669.
3. Colvin AC, Harrast J, Harner C. Trends in hip arthroscopy. J Bone Joint Surg Am. 2012;94(4):e23.
4. DeLee J, Drez D, Miller MD. DeLee & Drez's orthopaedic sports medicine principles and practice. Philadelphia: Saunders/Elsevier; 2010.
5. Rath E, Tsvieli O, Levy O. Hip arthroscopy: an emerging technique and indications. Isr Med Assoc J. 2012;14(3):170–4.
6. Larson CM, Giveans MR. Arthroscopic management of femoroacetabular impingement: early outcomes measures. Arthroscopy. 2008;24(5):540–6.
7. Byrd JW, Jones KS. Arthroscopic management of femoroacetabular impingement: minimum 2-year follow-up. Arthroscopy. 2011;27(10):1379–88.
8. Ganz R, Parvizi J, Beck M, et al. Femoroacetabular impingement: a cause for osteoarthritis of the hip. Clin Orthop Relat Res. 2003;417:112–20.
9. Leunig M, Beaule PE, Ganz R. The concept of femoroacetabular impingement: current status and future perspectives. Clin Orthop Relat Res. 2009; 467(3):616–22.
10. Bedi A, Zalta I, De La Torre K, Keely BT. Radiographic comparison of surgical hip dislocation and hip arthroscopy for treatment of cam deformity in femoroacetabular impingement. Am J Sports Med. 2011;39(Suppl):20S–8S.
11. Domb BG, Stake CE, Botser IB, Jackson TJ. Surgical dislocation of the hip versus arthroscopic treatment of femoroacetabular impingement: a prospective matched-pair study with average 2 year follow up. Arthroscopy. 2013;29(9):1506–13.
12. Harris JD, Erickson BJ, Bush-Joseph CA, Nho SJ. Treatment of femoroacetabular impingement: a systematic review. Curr Rev Musculoskelet Med. 2013;6(3):207–18.
13. Larson CM, Stone RM. Current concepts and trends for operative treatment of FAI: hip arthroscopy. Curr Rev Musculoskelet Med. 2013;6(3):242–9.
14. Ferguson SJ, Bryant JT, Ganz R, et al. An in vitro investigation of the acetabular labral seal in hip joint Mechanics. J Biomech. 2003;36:171–8.
15. Ferguson SJ, Bryant JT, Ganz R, Ito K. The influence of the acetabular labrum on hip joint cartilage consolidation: a poroelastic finite element model. J Biomech. 2000;33(8):953–60.
16. Diulus CA, Krebs VE, Hanna G, Barsoum WK. Hip arthroscopy technique and indications. J Arthroplasty. 2006;21(4 Suppl 1):68–73.
17. Philippon MJ, Faucet SC, Briggs KK. Arthroscopic hip labral repair. Arthrosc Tech. 2013;2(2):e73–6.

18. Lynch TS, Terry MA, Bedi A, Kelly BT. Hip arthroscopic surgery: patient evaluation, current indications, and outcomes. Am J Sports Med. 2013;41(5):1174–89.
19. Khanduja V, Villar RN. Arthroscopic surgery of the hip: current concepts and recent advances. J Bone Joint Surg Br. 2006;88(12):1557–66.
20. Resnick DL, Kang HS, Pretterklieber ML. Internal derangements of joints. Philadelphia: Saunders/Elsevier; 2007.
21. McCarthy JC, Noble PC, Schuck MR, Wright J, Lee J. The watershed labral lesion: its relationship on early arthritis of the hip. J Arthroplasty. 2001;16(8 Suppl 1):81–7.
22. Espinosa N, Rothenfluh DA, Beck M, Ganz R, Leunig M. Treatment of femoro-acetabular impingement: preliminary results of labral refixation. J Bone Joint Surg Am. 2006;88(5):925–35.
23. Larson CM, Giveans MR, Stone RM. Arthroscopic debridement versus refixation of the acetabular labrum associated with femoroacetabular impingement: mean 3.5 year follow-up. Am J Sports Med. 2012; 40(5):1015–21.
24. Larson CM, Giveans MR. Arthroscopic debridement versus refixation of the acetabular labrum associated with femoroacetabular impingement. Arthroscopy. 2009;25(4):369–76.
25. Schilders E, Dimitrakopoulou A, Bismil Q, et al. Arthroscopic treatment of labral tears in femoroacetabular impingement: a comparative study of refixation and resection with a minimum two-year follow-up. J Bone Joint Surg Br. 2011;93(8):1027–32.
26. Byrd JW, Jones KS. Hip arthroscopy for labral pathology prospective analysis with 10-year follow-up. Arthroscopy. 2009;25(4):365–8.
27. Yen YM, Kocher MS. Chondral lesions of the hip: microfracture and chondroplasty. Sport Med Arthroscop Rev. 2010;18(2):83–9.
28. Sampson TG. Arthroscopic treatment of chondral lesions of the hip. Clin Sports Med. 2011;30(2):331–48.
29. Hangody L, Fules P. Autologous osteochondral mosaicplasty for the treatment of full-thickness defects of weight-bearing joints: ten years of experimental and clinical experience. J Bone Joint Surg Am. 2003; 85-A Suppl 2:25–32.
30. Hart R, Janecek M, Visca P, Bucek P, Kocis J. Mosaicplasty for the treatment of femoral head defect after incorrect resorbable screw insertion. Arthroscopy. 2003;19(10):E1–5.
31. Jordan MA, Van Thiel GS, Chahal J, Nho SJ. Operative treatment of chondral defects in the hip joint: a systematic review. Curr Rev Musculoskelet Med. 2012; 5(3):244–53.
32. Bardakos NV, Villar RN. The ligamentum teres of the adult hip. J Bone Joint Surg Br. 2009;91:8–15.
33. Cerezal L, Kassarjian A, Canga A, Dobado MC, Montero JA, et al. Anatomy, biomechanics, imaging, and management of ligamentum teres injuries. Radiographics. 2010;30(6):1637–51.
34. Philippon MJ, Kuppersmith DA, Wolff AB, Briggs KK. Arthroscopic findings following traumatic hip dislocation in 14 professional athletes. Arthroscopy. 2009;25:167–74.
35. Gray AJ, Villar RN. The ligamentum teres of the hip: an arthroscopic classification of its pathology. Arthroscopy. 1997;13(5):575–8.
36. Botser IB, Martin DE, Stout CE, Domb BG. Tears of the ligamentum teres: prevalence in hip arthroscopy using 2 classification systems. Am J Sports Med. 2011;39(Suppl):117S–25S.
37. Jackson RW. The septic knee-arthroscopic treatment. Arthroscopy. 1985;1(3):194–7.
38. Chung WK, Slater GL, Bates EH. Treatment of septic arthritis of the hip by arthroscopic lavage. J Pediatr Orthop. 1993;13(4):444–6.
39. Hyman JL, Salvati EA, Laurencin CT, Rogers DE, Maynard M, Brause DB. The arthroscopic drainage, irrigation, and debridement of late, acute total hip arthroplasty infections: average 6-year follow-up. J Arthroplasty. 1999;14(8):903–10.
40. Yamamoto Y, Ide T, Hachisuka N, Maekawa S, Akamatsu N. Arthroscopic surgery for septic arthritis of the hip joint in 4 adults. Arthroscopy. 2001; 17(3):290–7.
41. Kim SJ, Choi NH, Ko SH, Linton JA, Park HW. Arthroscopic treatment of septic arthritis of the hip. Clin Orthop Relat Res. 2003;407:211–4.
42. Nusem I, Jabur MK, Playford EG. Arthroscopic treatment of septic arthritis of the hip. Arthroscopy. 2006;22(8):902.e1–3.
43. Blitzer CM. Arthroscopic management of septic arthritis of the hip. Arthroscopy. 1993;9(4):414–6.
44. El-Sayed AM. Treatment of early septic arthritis of the hip in children: comparison of results of open arthrotomy versus arthroscopic drainage. J Child Orthop. 2008;2(3):229–37.
45. Fu FH. Sports medicine. Master techniques in orthopaedic surgery, vol. xxiv. Philadelphia: Wolters Kluwer Health/Lippincott Williams & Wilkins; 2010. p. 640.
46. Helms CA, Major NM, Anderson MW, Kaplan P, Dussault R. Musculoskeletal MRI, vol. vii. 2nd ed. Philadelphia: Saunders Elsevier; 2009. p. 442.
47. Byrd JW, Jones K. Adhesive capsulitis of the hip. Arthroscopy. 2006;22(1):89–94.
48. Smith MV, Sekiya JK. Hip instability. Sports Med Arthrosc. 2010;18(2):108–12.
49. Tibor LM, Sekiya JK. Differential diagnosis of pain around the hip joint. Arthroscopy. 2008;24(12): 1407–21.
50. Berkes MB, Cross MB, Shindle MK, Bedi A, Kelly BT. Traumatic posterior hip instability and femoroacetabular impingement in athletes. Am J Orthop. 2012;41(4):166–71.
51. Krych AJ, Thompson M, Larson CM, Byrd JW, Kelly BT. Is posterior hip instability associated with cam and pincer deformity? Clin Orthop Relat Res. 2012; 470(12):3390–7.

52. Ross JR, Zaltz I, Nepple JJ, Schoenecker PL, Clohisy JC. Arthroscopic disease classification and interventions as an adjunct in the treatment of acetabular dysplasia. Am J Sports Med. 2011;39(Suppl):72S–8S.
53. Ross JR, Zaltz I, Nepple JJ, Schoenecker PL, Clohisy JC. Arthroscopic disease classification and interventions as an adjunct in the treatment of acetabular dysplasia. Am J Sports Med. 2011;39(Suppl):72S–8S.
54. Kim KI, Cho YJ, Ramteke AA, Yoo MC. Periacetabular rotational osteotomy with concomitant hip arthroscopy for treatment of hip dysplasia. J Bone Joint Surg Br. 2011;93(6):732–7.
55. Matsuda DK. Acute iatrogenic dislocation following hip impingement arthroscopic surgery. Arthroscopy. 2009;25(4):400–4.
56. Ranawat AS, McClincy M, Sekiya JK. Anterior dislocation of the hip after arthroscopy in a patient with capsular laxity of the hip. A case report. J Bone Joint Surg Am. 2009;91(1):192–7.
57. Benali Y, Katthagen BD. Hip subluxation as a complication of arthroscopic debridement. Arthroscopy. 2009;25(4):405–7.
58. Mei-Dan O, McConkey MO, Brick M. Catastrophic failure of hip arthroscopy due to iatrogenic instability: can partial division of the ligamentum teres and iliofemoral ligament cause subluxation? Arthroscopy. 2012;28(3):440–5.
59. Lachiewicz PF, Kauk JR. Anterior iliopsoas impingement and tendinitis after total hip arthroplasty. J Am Acad Orthop Surg. 2009;17(6):337–44.
60. Dora C, Houweling M, Sierra RJ. Iliopsoas impingement after total hip replacement: the results of non-operative management, tenotomy or acetabular revision. J Bone Joint Surg Br. 2007;89(8):1031–560.
61. Van Riet A, De Schepper J, Delport HP. Arthroscopic psoas release for iliopsoas impingement after total hip replacement. Acta Orthop Belg. 2011;77(1):41–6.
62. Strauss EJ, Nho SJ, Kelly BT. Greater trochanteric pain syndrome. Sports Med Arthrosc. 2010;18(2):113–9.
63. Domb BG, Nasser RM, Botser IB. Partial-thickness tears of the gluteus medius: rationale and technique for trans-tendinous endoscopic repair. Arthroscopy. 2010;26:1697–705.
64. Voos JE, Rudzki JR, Shindel MK, Martin H, Kelly BT. Arthroscopic anatomy and surgical techniques for peritrochanteric space disorders in the hip. Arthroscopy. 2007;23(11):1246.e1–5.
65. Voos JE, Shindle MK, Pruett A, Asnis PD, Kelly BT. Endoscopic repair of gluteus medius tendon tears of the hip. Am J Sports Med. 2009;37(4):743–7.
66. Contreras ME, Dani WS, Endges WK, De Araujo LC, Berral FJ. Arthroscopic treatment of the snapping iliopsoas tendon through the central compartment of the hip: a pilot study. J Bone Joint Surg Br. 2010; 92-B:777–80.
67. Kelly BT, Williams 3rd RJ, Philippon MJ. Hip arthroscopy: current indications, treatment options, and management issues. Am J Sports Med. 2003;31(6): 1020–37.
68. Beltran L, Ghazikhanain V, Padron M, Beltran J. The proximal hamstring muscle-tendon-bone unit: a review of the normal anatomy, biomechanics, and pathophysiology. Eur J Radiol. 2012;81:3772–9.
69. Birmingham P, Muller M, Wickiewicz T, Cavanaugh J, Rodeo S, Warren R. Functional outcome after repair of proximal hamstring avulsions. J Bone Joint Surg Am. 2011;93(19):1819–26.
70. Folsom GJ, Larson CM. Surgical treatment of acute versus chronic complete proximal hamstring ruptures: results of a new allograft technique for chronic reconstructions. Am J Sports Med. 2008;36(1):104–9.
71. Dierckman BD, Guanche CA. Endoscopic proximal hamstring repair and ischial bursectomy. Arthrosc Tech. 2012;1(2):e201–7.
72. Larson CM. Management of chronic proximal hamstring ruptures: surgical treatment. Operat Tech Sports Med. 2009;17(4):210–4.
73. Frontera WR, Micheli LJ, Herring SA, SIlver JK. Clinical sports medicine medical management and rehabilitation. Philadelphia: Saunders/Elsevier; 2007.
74. Smoll NR. Variations of the piriformis and sciatic nerve with clinical consequence: a review. Clin Anat. 2010;23(1):8–17.
75. Martin HD, Shears SA, Johnson JC, Smathers AM, Palmer IJ. The endoscopic treatment of sciatic nerve entrapment/deep gluteal syndrome. Arthroscopy. 2011;27(2):172–81.
76. Stafford GH, Villar RN. Ischiofemoral impingement. J Bone Joint Surg Br. 2011;93(10):1300–2.
77. Larson CM, Kelly BT, Stone RM. Making a case for anterior inferior iliac spine/subspine hip impingement: three representative case reports and proposed concept. Arthroscopy. 2011;27(12):1732–7.
78. Hetsroni I, Larson CM, Dela Torre K, Zbeda RM, Magennis E, Kelly BT. Anterior inferior iliac spine deformity as an extra-articular source for hip impingement: a series of 10 patients treated with arthroscopic decompression. Arthroscopy. 2012;28(11):1644–53.
79. Sussman WI, Han E, Schuenke MD. Quantitative assessment of the ischiofemoral space and evidence of degenerative changes in the quadratus femoris muscle. Surg Radiol Anat. 2013;35(4):273–81.
80. Hetsroni I, Poultsides L, Bedi A, Larson CM, Kelly BT. Anterior inferior iliac spine morphology correlates with hip range of motion: a classification system and dynamic model. Clin Orthop Relat Res. 2013; 471(8):2497–503.
81. Zaltz I, Kelly BT, Hetsroni I, Bedi A. The crossover sign overestimates acetabular retroversion. Clin Orthop Relat Res. 2013;471(8):2463–70.
82. Larson CM, Giveans MR, Taylor M. Does arthroscopic FAI correction improve function with radiographic arthritis? Clin Orthop Relat Res. 2011;469(6): 1667–76.
83. Farjo LA, Glick JM, Sampson TG. Hip arthroscopy for acetabular labral tears. Arthroscopy. 1999;15(2): 132–7.

84. Haviv B, O'Donnell J. The incidence of total hip arthroplasty after hip arthroscopy in osteoarthritic patients. Sports Med Arthrosc Rehabil Ther Technol. 2010;2:18.
85. Larson CM, Giveans MR, Taylor M. Does arthroscopic FAI correction improve function with radiographic arthritis? Clin Orthop Relat Res. 2011;469(6):1667–76.
86. Philippon MJ, Briggs KK, Carlisle JC, Patterson DC. Joint space predicts THA after hip arthroscopy in patients 50 years and older. Clin Orthop Relat Res. 2013;471(8):2492–6.
87. Kaminski A, Muhr G, Kutscha-Lissberg F. Modified open arthroscopy in the treatment of septic arthritis of the hip. Ortop Traumatol Rehabil. 2007;9(6):599–603.
88. Flierl MA, Stahel PF, Hak DJ, Morgan SJ, Smith WR. Traction table-related complications in orthopaedic surgery. J Am Acad Orthop Surg. 2010;18(11):668–75.

Hip Arthroscopy: Supine Approach to Patient Positioning, Set-up, and Traction

16

Allston J. Stubbs and Elizabeth A. Howse

Contents

Abstract

Hip arthroscopy can successfully be performed in either the supine or lateral position. Regardless of which method is chosen by the surgeon, proper positioning of the patient to achieve access to the hip through distraction and distention has proven to be essential for success. Like most aspects of medicine, having an informed team optimizes patient care, as positioning of monitoring and operative equipment should be carefully thought out prior to prepping the patient.

Introduction

In order to ensure a success in hip arthroscopy, the patient must be appropriately positioned for optimal access thereby allowing operative intervention. Traditionally, two positions have been utilized for hip arthroscopy, the supine position and the lateral position. The decision to choose one position over the other is largely dependent upon the surgeon's preference. This chapter describes the operating room setup and patient positioning for the supine position, detailing the benefits and potential risks that are associated with this approach. Setup and positioning for the lateral position are discussed in another chapter. As with the lateral position, the surgeon is afforded options in addition to which position he selects, such as the use of traction, and subsequently the type of distraction device used.

A.J. Stubbs (✉)
Surgical Sciences-Orthopaedics, Wake Forest Baptist Medical Center, Winston-Salem, NC, USA
e-mail: astubbs@wakehealth.edu

E.A. Howse
Department of Emergency Medicine, Long Island Jewish Medical Center, Hofstra North Shore-LIJ School of Medicine, New Hyde Park, NY, USA
e-mail: howseea@gmail.com

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_16

Fig. 1 Operative room layout

Preoperative Planning/Anesthesia/Setup

General anesthesia with muscle paralytics is the most predictable method to accomplish relaxation and simultaneously prevent intraoperative patient movement. Concomitant use of spinal anesthesia or regional anesthesia in the form of an epidural or lumbar plexus sciatic block may also be employed and adjunctively help with postoperative pain control. When necessary, an epidural may serve as the primary form of anesthesia; however, motor blockade is imperative to achieve appropriate muscle relaxation and subsequently a successful procedure [1, 2]. While positioning (supine versus lateral) is largely dependent upon surgeon's preference, it is imperative for the surgeon to communicate these preferences preoperatively with the anesthesia team in order to ensure a smooth execution on the operative day. Positioning will determine the appropriate means to secure the endotracheal tube, as well as determine access to the upper extremities thereby the appropriate locations for intravenous catheters and blood pressure monitoring. It is important for the anesthesiologist to know that the patient's mean arterial pressure (MAP) may affect arthroscopic visualization and should therefore be kept less than or equal to the arthroscopic pump pressure (generally 65 mmHg) whenever possible. Finally, the anesthesiologist should be prepared with tools to maintain euthermia such as warmed saline and/or a "Bair hugger" patient warming system (Arizant Healthcare Inc., St. Paul, MN, USA).

Once in the operative theater, prior to any intervention, a surgical "time-out" should be performed to confirm patient identification and operative hip. Performing the time-out early is beneficial for patient safety and affirms for the entire operating room staff, the laterality prior to patient, and equipment positioning. The arthroscopy monitors, fluid pump system, and fluoroscope should be positioned opposite to the operative side. This is necessary to allow room for the surgeon, his/her assistant, and scrub technician, whom will stand on the operative side, along with the primary instrument table, which will be behind the surgeon (Fig. 1). A standard fluoroscope is recommended, as the larger diameter is necessary to be able to go over the distraction system.

After anesthesia is induced and the airway is deemed secure, the OR staff can start to prepare for surgery as the anesthesiologist takes further protective measures, such as ocular lubrication.

Fig. 2 Patient positioning

As the patient may be under anesthesia for longer than 2 h, a Foley catheter may be considered prior to surgical positioning and operative prepping. Furthermore, the radio-frequency grounding pad should be attached to the patient's skin across the abdomen or contralateral thigh prior to creating a sterile field.

Positioning

The upper extremity (ipsilateral to the operative side) needs to be positioned away from the operative field. Concomitant papoose wrapping with the contralateral upper extremity or simply the ipsilateral arm draped over the patient's chest will accomplish the desired effect. Utilizing a small stack of blankets to allow less than 90° of flexion at the elbow reduces the risk of positional ischemia by optimizing the circulation to the arm(s). The use of a pulse oximeter on the ipsilateral arm may help confirm good perfusion intraoperatively when the arm cannot be otherwise evaluated. Keeping the needs of anesthesia in mind, the contralateral arm may alternatively be secured to an arm board. Regardless of which position is chosen, the flow of the IV catheter should be reassessed prior to establishing a sterile field.

The patient is repositioned on a standard fracture table or via a standard operative table with traction attachment. A padded perineal post is lateralized toward the operative side and subsequently secured. The patient is then moved so that the perineum is against a padded perineal post, which will contact the medial thigh of the operative hip and allow for controlled lateralization of the femur (Fig. 2). Additional padding of the post minimizes the potential of pudendal neurapraxia by providing a transverse force vector against the patient's thigh. While a commercially padded perineal post is not required, it is recommended in order to ensure that it is sized appropriately. The size of the perineal post corresponds with the degree of lateral force vector in place when the operative leg is placed in traction [3]. The use of oversized perineal post (commercial posts range between 9 and 12 cm) has been associated with less pudendal neurapraxia risk [1]. In a series of 20 patients, Byrd found this to occur in two patients with the use of "hand-devised" post, but not to occur in subsequent cases where a commercial post was used [1, 3, 4]. Careful attention should be paid toward the positioning of the scrotum to ensure that the genitalia are not compressed, as excessive pressure may result in edema, hematoma, or scrotal necrosis [5]. A glove may be left

Fig. 3 Preoperative fluoroscopic evaluation, with operative hip in flexion

on top of the post for the ease of the circulating nurse, who may need to reevaluate the positioning of the male genitalia with changes in traction throughout the case. Similar positioning care of the female patient should be observed as well.

Foam and/or towel padding is utilized over all bony prominences in order to prevent the formation of decubiti. In particular, the surgeon should pay specific attention to the contralateral ischial tuberosity and sacrum which depending on the operative table may rest upon metal or plastic bed parts. When positioning the nonoperative limb, the surgeon must consider stabilization of the pelvis and torso with a mild counterforce, providing adequate room for the C-arm fluoroscope to provide visualization of the operative hip while avoiding any unnecessary stress on the nonoperative hip limb. The patient's feet are placed in traction boots (after padding), and once the feet are secured in the traction boots, the pelvis and torso are stabilized by applying a delicate countertraction (10–20 lb/4.5–9 kg) to the nonoperative limb. Prior to applying traction, the operative hip is placed in 90° of flexion with approximately 20–30° of abduction; the traction boots enable the securement of the operative leg in this position by securing the boot along the rail. The C-arm is then brought in from the contralateral side, and a 45° Dunn lateral view of the hip is obtained. Fluoroscopic examination is then able to assess dynamic hip pathology that may not be evident on traditional static preoperative films (Fig. 3). Next, the operative hip is placed in a neutral rotation with approximately 0° of adduction and 15° of flexion in preparation for the application of traction. One may consider internal foot rotation of 10° to position the femoral neck more parallel to the operative horizon. Excessive flexion of the hip is avoided because tension on the sciatic nerve is increased during distraction and can result in sciatic injury. The contralateral limb is secured in its natural rotation for the remainder of the operative course.

Traction

In order to perform a successful operation, it is imperative for the hip arthroscopist to establish access to the hip joint. The complex soft tissue envelope that constrains the hip joint makes access difficult. Historically, entrance between

Fig. 4 Application of traction

the femoral head and acetabulum was thought "manifestly impossible" as the labral seal holds a vacuum force between 120 and 200 N; therefore, initial distraction forces are reported at around 200 lb (90 kg) [6–8]. It was later determined that this seal could be broken and the negative pressure vented by puncturing the joint and with an injection of normal saline causing distension to overcome part of these forces [5, 9, 10]. The addition of distension with proper anesthesia has reduced the amount of distraction force needed to less than 50 lb (23 kg) while still obtaining operative access [5]. Understanding all the aspects used to overcome the intra-articular force explains why the majority of complications related to hip arthroscopy are related to the duration and extent of traction applied [4, 11, 12]. Complications associated with excess traction include pudendal neurapraxia, sciatic nerve injury, genital injury, and lower extremity skin injury [1]. Careful monitoring of the amount of traction force applied and total traction time can avoid these potential complications [12, 13]. Intermittently removing traction throughout the case, in order to keep the total traction time under 2 h, and limiting continuous traction to 1 h minimize these risks [12].

Application of Traction

The use of traction should be applied gently and with considerable care. Applying traction prior to prepping and draping for the procedure establishes a secure operative setup and confirms adequate muscle paralysis, as well as ascertains the amount of distraction that will be necessary. The amount of traction required will be dependent upon each patient's pathophysiology; specifically, laxity requires less and arthritis more traction in order to distract the joint. Most patients will require 25–50 lb (11–23 kg) in order to achieve adequate distraction when appropriately relaxed [1, 14].

Traction is applied with the knee in full extension and a neutral rotation of the foot. Movement of the rail allows for changes in abduction and adduction of the hip without alteration of traction. Initially the operative lower extremity is placed against the padded perineal post in neutral adduction. Pulling on the operative leg prior to attaching the distraction clamp allows for the attachment to be maximally distal, thereby ensuring that the amount of distraction will not exceed the limitations of the device (Fig. 4). This will result in an

Fig. 5 A tensiometer can assist with monitoring the amount of traction applied

initial traction of 20–30 lb (9–14 kg). The use of a tensiometer can assist with monitoring the amount of traction utilized throughout the case (Fig. 5). If a tensiometer is employed, then the readings should be adjusted to account for the manually applied traction.

The C-arm is then used to determine that traction is adequate via the appearance of a crescent sign [15]. If a crescent sign is not originally visualized, the surgeon must choose between immediately prepping and draping the patient to allow for venting of the hip through use of a spinal needle in order to minimize additional force, or gently apply additional traction until the crescent sign becomes apparent. If traction is chosen, an initial "nominal" joint space of approximately 10–15 mm is necessary and sufficient for the hip capsule to accommodate the external forces applied to the joint and relax via physiological creep and ligamentotaxis [14]. Once this is visualized fluoroscopically, traction should remain while the surgeon is scrubbing for the case; it can then be relaxed while the operative leg is prepped and draped.

Regardless of whether the surgeon employs physiological creep, or venting, the hip should be in slight flexion and internal rotation to assist in relaxation of the anterior capsule prior to making an incision and ultimately penetrating the hip joint [5].

Tractionless Hip Arthroscopy

The hip can be divided into two compartments: central and peripheral, as described by Dorfmann and Boyer [7, 16]. The central compartment consists of the acetabular fossa, the ligamentum teres, the lunate cartilage, and the loaded articular surface of the femoral head, and access to the central compartment generally requires traction [7, 16].

The peripheral compartment is lateral to the labrum; it entails the anterior capsule including the intrinsic ligaments (zona orbicularis), the unloaded cartilage of the femoral head, and the femoral neck including the anterior, medial, and lateral synovial folds (Weitbrecht's ligaments) [7, 16]. Surgery on the peripheral compartment (hip endoscopy) is done with the hip flexed to approximately 40° to allow relaxation of the anterior capsule; therefore, it does not necessitate traction [7]. "Tractionless" hip arthroscopy initiates surgery in the peripheral compartment; the trajectory of the femoral neck is identified under fluoroscopic assistance, and the intracapsular space is entered via the Seldinger technique. Starting hip arthroscopy in the peripheral compartment minimizes the amount of initial traction necessary to enter the central compartment. When surgery is initiated in the central compartment (as described above), the peripheral compartment can be accessed under direct visualization (without fluoroscopic assistance); the hip is taken out of traction, thereby decreasing continuously traction time if the surgeon has not completed his work in the central compartment.

Distraction Options

The choice of distraction device is based on surgeon's preference, training, and availability, since these devices have not been comparatively reviewed. In considering which type of distraction device to use, it is necessary to know what type of table they were designed for, standard operating room table versus fracture table (Table 1).

There are two invasive hip distraction systems: the DR Medical Hip Distractor and the Dahners

Table 1 Distraction systems

Operative tables	
Maquet table	Getinge Group AB, Solna, Sweden
Amsco OrthoVision table	Steris, Inc., Mentor, OH, USA
Noninvasive hip distraction systems	
Smith and Nephew Hip Positioning System	Andover, MA, USA
OSI hip distractor	Union City, CA, USA
Arthrex Hip Distractor System	Arthrex, Inc., Naples, FL, USA
Spider Hip Position System	Tenet Medical, Calgary, Canada
McCarthy Hip Distractor (lateral only)	Innomed, Savannah, GA, USA
Invasive hip distraction systems	
DR Medical Hip Distractor	DR Medical AG, Solothurn, Switzerland
Dahners Hip Distractor (lateral only)	Medical Products Resource, Burnsville, MN, USA

Hip Distractor. The DR Medical Hip Distractor can be utilized in the supine position. It is applied by sterilely placing pins into the pelvis and femur and, therefore, does not require a perineal post to provide countertraction. The lack of a perineal post minimizes the risk of sciatic nerve stretch injury, as well as potential skin complications along the foot/ankle from the traction boots [17]. The DR Medical Hip Distractor may be beneficial when central compartment work is expected to be complex, or due to early surgeon experience, the resulting traction times will be over 2 h. The Dahners Hip Distractor affords similar benefits as the DR Medical Hip Distractor but instead applies traction through a distal femoral K-wire; however, it has only been used when the patient is laterally positioned [18, 19].

Summary

In order to ensure the optimal experience for a patient undergoing hip arthroscopy, the surgeon should pay careful attention to all aspects of the case. As this procedure is still considered new in many operating rooms, it is beneficial for the hip arthroscopist to educate the rest of the members of the operating team (anesthesia, scrub and circulating nurses, assistants, etc.) prior to the patient ever entering the operative theater. Since the supine position is utilized in all surgical subspecialties, it affords the benefit of being familiar to all of the operating room staff. Acquainting the team with the intricacies of hip arthroscopy reduces the operative risk and maximizes overall efficiency.

References

1. Stubbs AJ, Stone AV. Position and distraction options. In: Byrd JWT, editor. Operative hip arthroscopy. 3rd ed. New York: Springer; 2013. p. 121–9.
2. Byrd JWT. The supine approach. In: Byrd JWT, editor. Operative hip arthroscopy. 2nd ed. New York: Thieme; 2005. p. 145–69.
3. Kong KFJ, Chien P, Wong JWK, Yip DKH. Peroneal padding: an alternative to a peroneal post in hip arthroscopy. J Arthrosc. 2003;19(9):1027–9.
4. Byrd JWT. Hip arthroscopy using the supine position. J Arthrosc. 1994;10(3):275–80.
5. McCarthy JC, Day B, Busconi B. Hip arthroscopy: applications and technique. J Am Acad Orthop Surg. 1995;3:115–22.
6. Burman MS. Arthroscopy or direct visualization of the joints: an experimental cadaver study. J Bone Joint Surg Am. 1931;13A:669–95.
7. Dienst M. Hip arthroscopy without traction. In: Byrd JWT, editor. Operative hip arthroscopy. 2nd ed. - New York: Thieme; 2005. p. 170–88.
8. Byrd JWT, Chern KY. Traction versus distension for distraction of the joint during hip arthroscopy. J Arthrosc. 1997;13(3):346–9.
9. Eriksson E, Arvidsson I, Arvidsson H. Diagnostic and operative arthroscopy of the hip. Orthopedics. 1986;9:169–76.
10. Dienst M, Gödde S, Seil R, Hammer D, Kohn D. Hip arthroscopy without traction: in vivo anatomy of the peripheral hip joint cavity. J Arthrosc. 2001;17:924–31.
11. Clarke MT, Arora A, Villar RN. Hip arthroscopy: complications in 1054 cases. Clin Orthop Relat Res. 2003;406:84–8.
12. Byrd JWT. Complications associated with hip arthroscopy. In: Byrd JWT, editor. Operative hip arthroscopy. 2nd ed. New York: Thieme; 2005. p. 229–35.
13. Sampson TG. Complications of hip arthroscopy. Clin Sports Med. 2001;20(4):831–5.
14. Byrd JWT. Hip arthroscopy: the supine position. Clin Sports Med. 2001;20:703–31.
15. Philippon MJ, Stubbs AJ, Schenker ML, Maxwell RB, Ganz R, Leunig M. Arthroscopic management of

femoroacetabular impingement: osteoplasty technique and literature review. Am J Sports Med. 2007;35:1571–80.

16. Dorfmann H, Boyer T. Arthroscopy of the hip: 12 years of experience. J. Arthrosc. 1999;15:67–72.
17. Simpson J, Sadri H, Villar R. Hip arthroscopy techniques and complications. Orthop Traumatol Surg Res. 2010;96(8 Suppl):S68–76.
18. Smart LR, Oetgen M, Noonan B, Medvecky M. Beginning hip arthroscopy: indications, positioning, portals, basic techniques, and complications. J Arthrosc. 2007;23:1348–53.
19. Bushnell BD, Hoover SA, Olcott CW, Dahners LE. Use of an independent skeletal distractor in hip arthroscopy. J Arthrosc. 2007;23:106e1–4.

Hip Arthroscopy: Lateral Approach to Patient Positioning, Set-Up, and Traction

17

Thomas G. Sampson

Contents

Abstract

The lateral approach to hip arthroscopy was developed in the early 1980s by James M. Glick, M.D., and Thomas G. Sampson, M.D., as a response to poor access and reproducibility in accessing the central compartment of the hip. The procedure involves performing hip arthroscopy with the patient in the lateral decubitus position using a lateral distraction device, using common portals to the supine position, and allows access to all areas of the hip, including the central and peripheral compartments and the peritrochanteric space. This technique is indicated for all procedures in hip arthroscopy and has been widely accepted and allows for effective and reproducible results. The preference to perform hip arthroscopy in this position is based on surgeon's training, expertise, and comfort.

Introduction and Brief History

In 1982 there were no accepted techniques or hip-specific instruments for hip arthroscopy. The procedure was first done in our practice using the supine approach on a fracture table for distraction to enter the central compartment, then regarded as the intra-articular space. Simultaneously in Paris, Henri Dorfmann and Thierry Boyer were developing a method to get into the peripheral space without traction using the supine position [1, 2]. In our early experience, problems of getting

T.G. Sampson
Medical Director of Hip Arthroscopy, Post Street Surgery Center, San Francisco, CA, USA

Post Street Orthopaedics and Sports Medicine, San Francisco, CA, USA
e-mail: tgsampsonmd@gmail.com

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_27

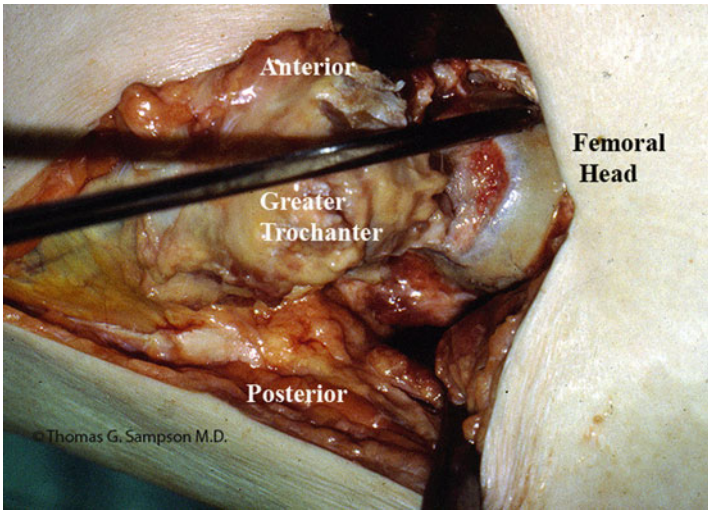


Fig. 1 Original cadaver dissection done by James M. Glick and Thomas G. Sampson to develop the portals for the lateral approach

into the hip joint and complications such as scuffing of articular cartilage, labral damage, poor maneuverability, and inability to achieve the anticipated result before extensive fluid extravasation made the procedure very difficult and unpredictable. Specific instruments were not developed and distraction parameters were not yet established. As a result, the procedure was neither predictable nor reproducible in entering the intra-articular space now known as the central compartment.

History has shown us that Burman [14] in 1931 was the first to arthroscope the hip in a cadaveric study. He was unable to enter the central compartment even with distraction. James M. Glick M.D (JMG) performed 11 cases between 1977 and 1982 and had difficulty getting in on two occasions [3]. Because of beneficial experience with the lateral decubitus positioning in total hip replacements, the idea of approaching hip arthroscopy with a similar approach was developed. A cadaver hip was dissected to determine the most direct access to the intra-articular space and to describe the anterior-peritrochanteric and posterior-peritrochanteric portals (Fig. 1). Later these have been referred to as the anterolateral and posterolateral portals. Distraction was introduced by Erikkson [4] using a fracture table to facilitate entering the central compartment in 1986. A rope and pulley system were developed with weights similar to what we used for shoulder arthroscopy as our first hip distractor (Fig. 2). The first patient JMG arthroscoped with the lateral approach was a massively obese woman with hip pain that JMG had previously arthroscoped without success in the supine position. In the lateral decubitus position, the obese portions of her thigh recede to expose a prominent greater trochanter. The neurovascular structures are safely away from the portals, and the surgeon is very familiar with their locations. These portals offer a direct entry into the femoroacetabular joint and do not require imagining the angles of entry as described by Byrd [5] for the supine approach. The few surgeons interested in hip arthroscopy at that time adopted the lateral technique and may continue to use it today.

With the interest by industry to develop arthroscopic instruments and distractors, the supine approach was once again used and described by Byrd [6–8]. There have been editorials, journal articles, and book chapters arguing the advantages of each method over the other, with the results that whichever approach to be used should be based on the surgeon's training and comfort. All procedures of hip arthroscopy are done equally well using either technique, and complications are not reported as technique specific. A distractor has been designed to be used on any operating room table for both techniques (Smith & Nephew, Inc.,

Fig. 2 The first hip distractor design using a Buck's traction as a leg holder suspended with ropes and pulleys from the ceiling and the wall with >45 lb of weight

Andover, MA), and all instruments designed for hip arthroscopy can be used for either technique; however, it was found that shorter traditional length instruments work well in the lateral position as the distance from the skin to the depths of the central compartment seems to be shorter due to the soft tissue dropping away from the portals.

The major advancements in getting into the central compartment came from a better understanding of distraction and the use of cannulated trochars and fluoroscopy. Later, the development of longer arthroscopes, slotted (half-pipe) cannulas, and curved and flexible instruments has allowed for advanced techniques following a similar but delayed enthusiasm as with knee and shoulder arthroscopy (Fig. 3a, b).

Preoperative Planning/Anesthesia/Setup

Most commonly general anesthesia is used. With regional anesthesia, there must be muscle relaxation. Often conscious sedation is employed as the downside shoulder may become sore. A protocol of antibiotic prophylaxis with one of the cephalosporins is given preoperatively. DVT prophylaxis is done using compression stockings (SCD) and a sequential pump on either legs or feet.

Positioning

The patient is placed on a well-padded operating room table in the lateral decubitus position (Fig. 4). An axillary roll or shoulder trough is positioned and hip positioners are used to support the pelvis. By preventing the pelvis from rolling backward on the peroneal post, the risk of pudendal neuropraxias may be reduced.

The foot is wrapped with padding then strapped into the holder taking care to avoid skin pinching and over compression of the foot by the device. The leg is held in abduction by the assistant for careful placement of the well-padded peroneal post. We have determined that the post should have an outer diameter greater than 9 cm for safety. Commercially available hip distractors all exceed this size. The genitalia are inspected to ensure they are free from compression. Distraction should be tested to ensure that the foot would not slip out of the holder. It is recommended to apply only enough traction to support the leg during the setup, preparation, and draping.

The fluoroscopic C-arm is brought in with the apex under the table and centered at the level of the greater trochanter. Multiple preoperative X-rays are done to check for positioning, head-neck/acetabular conflicts, and hip anatomy. A trial

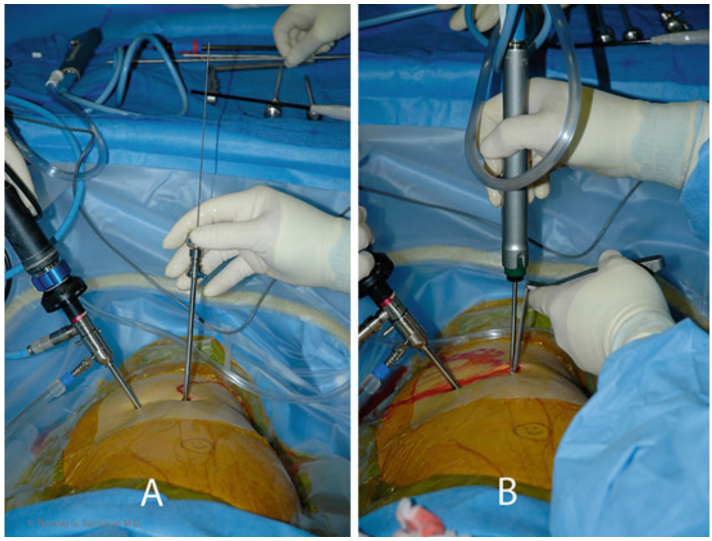


Fig. 3 The arthroscope has a shortened hub and the hip trochar is cannulated for a nitinol wire (Note the Mayo stand is above the patient's shoulder and is used to keep the instruments organized and for easy access by the surgeon)

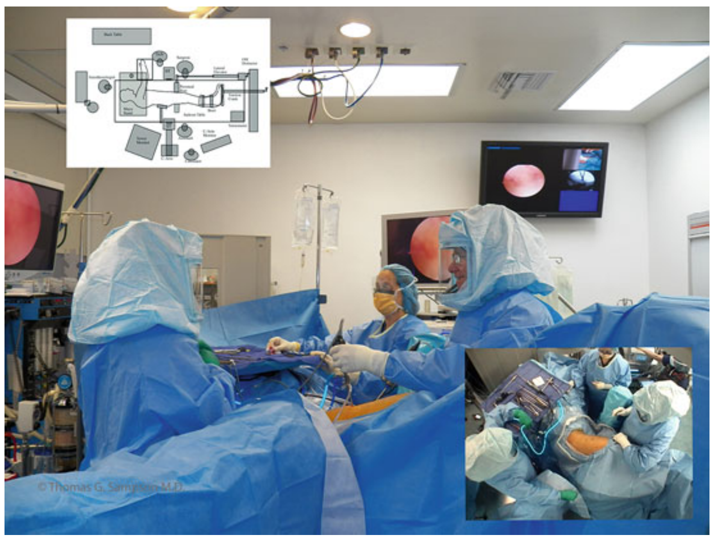


Fig. 4 Room setup in the lateral approach – the surgeon stands anterior (some prefer posterior if they do total hips from the posterior approach). The assistant works from the opposite side and the scrub technician to the side of the surgeon. The C-arm lies below the table throughout the case, and a Mayo stand is placed above the shoulder for instruments

of distraction will be of benefit for two reasons: (1) to check for the distractibility of the joint and (2) to ensure the foot is properly secured in the foot holder. If the hip does not distract well, it may be due to a tight or hypertrophic capsule, and a few minutes of traction may allow it to relax. Failure to distract may require greater forces of distraction and necessitate capsulotomy or acetabular rim trimming to avoid femoral head scuffing and iatrogenic labral damage. If the foot slips out of the holder during the trial, less padding and careful attention to strap placement and tightening may help prevent slippage. Adequate security of the foot in the holder is imperative to prevent an accidental release of distraction when the instruments are in the central compartment, which may result in iatrogenic articular cartilage damage.

Sticky towels or drapes are placed from the iliac crest to 6 in. below the greater trochanter and a sagittal line lateral to the anterior superior iliac spine anterior and the sciatic notch posterior.

The anesthesiologist is at the head of the table, the surgeon stands either anterior or posterior with the assistant on the opposite side. The scrub

Fig. 5 A commercially available lateral hip distractor made by Smith and Nephew®

technician stands next to the surgeon with the C-arm in between. A Mayo stand is placed above the patient's shoulder for easy accessibility to the instruments and organization of the arthroscopic cords.

Typically split sheets are used to drape the surgical site along with a large plastic pouch to catch fluids.

Distraction

For optimal viewing and safe surgery, at least 1.2 cm of distraction is required to enter the femoroacetabular joint (central compartment). Commercially available distractors for the lateral approach are available and have the ability to move the hip into various positions to optimize surgery in the central, peripheral, and peritrochanteric compartments during surgery (Fig. 5).

Not all distractors have a tensiometer to measure the force of distraction. Distraction should be thought of in the same way as a tourniquet. James M. Glick M.D., in a study using evoked potentials, originally concluded that forces less than 75 lb for less than 2 h were safe and did not cause permanent neuropraxias; however, on a later analysis of his original data using modern statistical analysis, the actual force is more pertinent than the time spent in traction [9]. Over 2,000 cases have been done by TGS and JMG without a tensiometer, taking care to keep the distraction time and the forces to a minimum resulting in few neuropraxias [10, 11]. A well-planned capsulotomy will reduce the forces of distraction necessary to enter the center compartment. The entire team should be aware of when the hip distraction begins and its duration. The surgeon must appreciate, however, that complications may occur from too little or too much traction, and to accomplish the procedure, the joint surfaces must be separated to introduce and properly use the instruments.

The peroneal post should have padding of at least 9 cm in diameter and positioned eccentrically over the pubic symphysis with little to no compression to the downside thigh (Fig. 6).

The operative distraction is initiated after the case is entirely set up and all the equipment have been turned on and are functioning. The traction time is recorded on a white board, which may be entered into the operative record. The abdomen is palpated under through the surgical drapes for distension from retroperitoneal extravasation every 15 to 30 minutes.

Once the surgery of the central compartment is completed, all of the distraction forces are released and the periarticular work in the peripheral compartment can be done without distraction

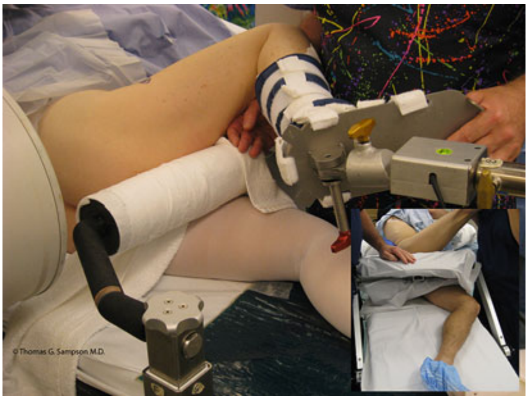


Fig. 6 A patient's right leg being placed in the hip distractor (Note the posterior hip positioner, the large peroneal pad, and the foot padding. Not seen is the axillary roll)

concerns. Distraction is then only instituted whenever the procedure requires entry into the central compartment such as labral repair or to protect the head from the burr during rim trimming.

OR Setup

Leg Position

In traction, the hip capsule is maximally relaxed in 15° of flexion, neutral rotation, and 15° of abduction [3, 12, 13]. This as a starting position and positional adjustments are made during the procedure to facilitate getting into different areas. Additionally, the peroneal post may be elevated laterally to add an abduction moment for better viewing.

Instruments

The 30° arthroscope is best for central viewing. It is easier to become oriented with this angle and is the best for getting started using the lateral approach. The 70° arthroscope is best for peripheral viewing, around the femoral head, deep in the fossa, to view the fovea and to create additional portals. On thin patients, standard arthroscopic equipment may be used if the sheath has a short hub. The advantages of commercially available hip kits are that they contain the proper sheath lengths and cannulated systems. The option for longer arthroscopes should be available for larger patients and for those cases in which excessive swelling occurs in the thigh during the procedure.

Both straight and curved graspers are necessary as well as straight and curved shavers. To insert curved instruments a slotted cannula or a flexible plastic sheath is used.

Radio-thermal probes are used for coagulation, cutting, and ablation of tissues such as the capsule or labrum. Many are curved or bendable and can reach lesions not accessible to straight shavers. Flexible wands for hips can be manually maneuvered with a trigger handle and curved shavers and burrs are now available, but not yet ideal in their designs for accessing the entire joint. The entire acetabulum and all but the femoral head fovea and portions of the posterior head are accessible by most instruments.

Angled neurocurrettes and angled picks are used to treat arthritic defects and remove attached and loose bodies located in difficult areas to reach such as the medial acetabular fossa and anterior medial acetabulum.

Pump

It is generally accepted to use a pump system since the exact pressure and flow can be controlled and monitored. We recommend using inflow/outflow pumps, which may reduce the amount of extravasation into the soft tissues. The pump pressure is set the same as shoulder settings, slightly above diastolic pressure, or as low as possible to achieve distention and a clear field from bleeding.

Tower

The arthroscopic tower with the monitor and instrument boxes should be placed opposite the surgeon and is slightly cephalad adjacent to the C-arm for optimal viewing of all the settings by the surgeon. The cords from the tower are brought onto the Mayo stand and organized for the surgeon to easily reach for the shaver and wands. It is more efficient and safe for the Mayo stand to act as neutral ground whereby only one person accesses it to avoid accidental glove punctures or lacerations (Fig. 7).

Procedure

The portals for the lateral approach are nearly identical to the supine approach except the anterior portal is located approximately 2 cm. lateral to the ASIS line. As a result, the anterior portal courses in an intermuscular plane between the sartorius and tensor fascia lata (Fig. 8).

Distraction is initiated after the patient has been prepped, draped and all equipment has been tested for proper functioning. For small and flexible patients, start with 25–50 lb of force, and with large stiff patients 50–75 lb if using a tensiometer or observing at least 1–2 cm of separation between the head and the acetabulum on the fluoroscopic view.

Fig. 7 The surgeon's view from the front of the patient. (**a**) There is a clear view of the tower's instrument boxes, the monitor, and the outflow-dependent pump. The Mayo stand is above the patient's shoulder and is used to organize and make easy access of the instruments. (**b**) A view of the fluoroscopic monitor is toward the foot of the table

Viewing with the C-arm fluoroscope starting with the anterolateral portal, the long 17 gauge needle is inserted while observing it pass between the head of the femur and acetabulum (closer to the femur to avoid puncturing the labrum). Listen for a hiss of sound as the joint suction seal is broken and room air is sucked into the joint. Observe for the traction forces to reduce on the tensiometer or a sudden distal subluxation of the hip. Obtain the desired distraction (usually greater than 1.2 cm). Insert a nitinol wire through the needle, and incise the skin with a #11 blade. Push the cannulated arthroscopic sheath over the wire and into the joint while advancing concentrically over the wire to prevent kinking and wire breakage. Backing the wire out slightly will reduce cartilage trauma.

If it is difficult to advance into the joint, suspect that the wire is going through the labrum. In such instances, it is best to start over and reposition the needle to avoid labral avulsions or tears. In some cases with stiff hips, the anterior capsule is very thick and very difficult to penetrate. In that situation it is best to begin with the posterolateral portal or gently cut the capsule with a long Beaver™

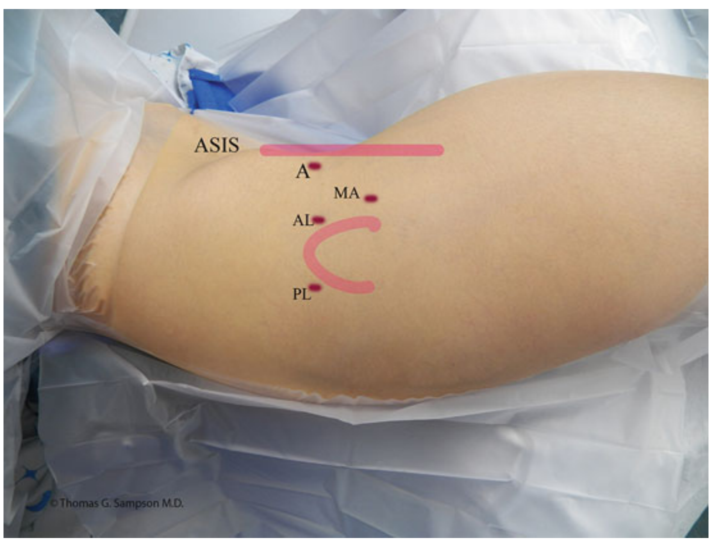


Fig. 8 Portals for the lateral approach (Right hip); anterior superior iliac spine (ASIS), posterolateral (PL), anterolateral (AL), anterior (A), and anteroinferior (AI)

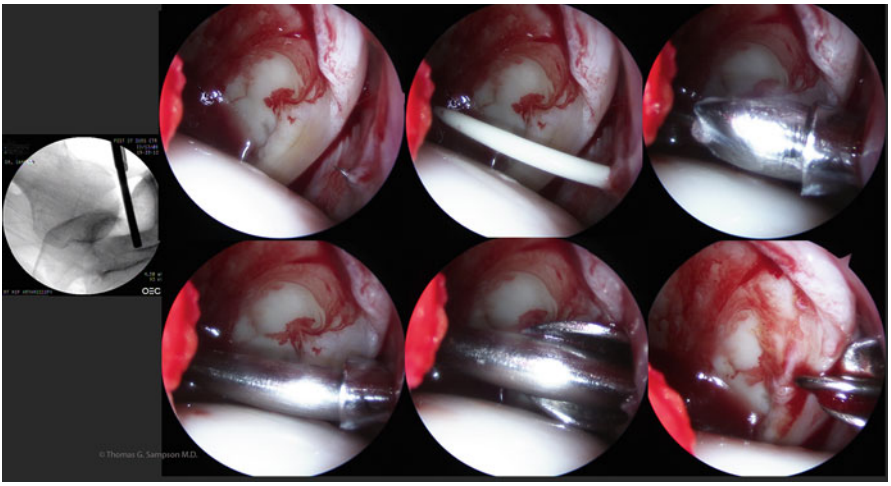


Fig. 9 Fluoroscopic view of distracting the hip joint and insertion of the cannulated trochar over the nitinol wire. Arthroscopic view from the anterolateral portal of the progression of steps inserting the needle, the nitinol wire, the cannulated trochar, switching stick, slotted (half-pipe) cannula, and finally an RF probe through the anterior portal

blade through the arthroscopic sheath prior to advancing into the joint. Entry into the joint should always be controlled to avoid damage to the labrum or scuffing of the cartilage. However, pushing a cannula through the anterior hip capsule is difficult and requires a lot of force when compared to any other joint in the body.

Introduce a 30° arthroscope and visually sweep the joint under air or fluid. Next, create the anterior and posterolateral portals using the same technique with the added benefit of viewing entry of the needle, nitinol wire, and instruments under direct vision to prevent injury to the cartilage and labrum. It is much safer with this approach in reducing iatrogenic injury. In creating the anterior portal, take care to only incise the skin superficially to avoid laceration of a branch of the lateral femoral cutaneous nerve. Spreading through the subcutaneous tissue with a clamp is also advised for this portal. Slotted or half-pipe cannulas may be used to insert larger or curved instruments (Fig. 9).

An alternate way to enter the peripheral and central compartments is through a capsulotomy first approach (Fig. 10). It is easier to teach hip arthroscopy to arthroplasty surgeons using this technique since it is essentially a submuscular arthrotomy of the hip. For most cases the use of the anterolateral and the mid-anterior portals are all that is necessary to complete the procedure for this method it is best to start with the hip in a relaxed position and no distraction. Using fluoroscopic control, a spinal needle is placed through the anticipated anterolateral portal and aimed at the superior lateral rim of the acetabulum about a half a centimeter toward the head. Using the 30° arthroscope the view of the anterior fat pad of the hip distal to the indirect head of the rectus femoris will identify the entry point just lateral to the origin of the iliofemoral ligament. The mid-anterior portal is created in the same fashion and a 4-mm shaver is used to clear a space anterolaterally. Using a radiofrequency device, the capsules are incised from the base of the neck longitudinally through the zona orbicularis to the acetabular rim, avoiding the labrum so as not to damage it. The capsule is then incised on the capsular junction along the rim laterally and anteriorly which then gives a global view of the femoral neck, head and neck junction, the labrum, the acetabular rim, the peripheral space, as well as the sub-spine space. Distraction is then initiated, requiring less force, and the arthroscope is driven into the central compartment under direct view reducing the incidence of chondral or labral damage. TGS has done over 1,500 capsulotomies in this fashion without the complication of

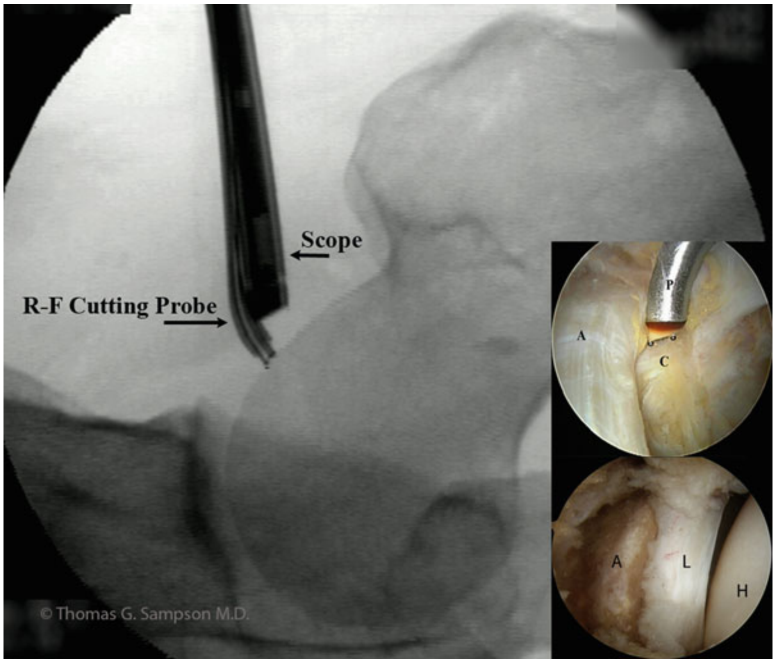


Fig. 10 Capsulotomy starting extracapsular is used to get into tight hips and for bone resection for femoroacetabular impingement. Fluoroscopic view placing the RF cutting probe on the anterolateral capsule. The RF cutting probe (P) on the capsule (C) (Note the reflected head of the rectus femoris (A). Arthroscopic view after capsulotomy; acetabular rim (A), labrum (L), and head of the femur (H))

subluxation or dislocation. The capsule may be repaired at the end of the case should be deemed necessary.

The central compartment should be inspected in a methodical fashion for damage and to plan the necessary treatment. Efficient management of the pathology, attention to distraction time, and monitoring the patients' temperature and abdominal distention will facilitate correction of the problem and reduce the potential for complications.

Arthroscopic Anatomy

The acetabulum and its structures are viewed first. Initially the femoral head cannot be entirely viewed with the hip distracted; however, the hidden portions will be observed when looking in the peripheral compartment later in the procedure (Fig. 11).

With the 30° scope, start with observing the acetabular fossa and the fat pad. Petechial hemorrhage is normal due to the traction forces pulling negative pressure on the vessels. Atrophy of the fat pad is abnormal. Look for loose bodies, rice bodies, and notch osteophytes or masses. Advance the scope deep to view the ligamentum teres. Inspect for tears or avulsions. The transverse acetabular ligament is very hard to see unless the patient has hyperlaxity.

Rotate the scope posterior and inferior and pick up the posterior labrum at the articular margin noting the posterior third. Check behind the labrum for loose bodies then follow the labrum lateral and anterior noting a normal cleft in the posterior articular margin with a small labral cartilage sulcus. The sulcus is not an old avulsion fracture nor from subluxation posterior. Note any labral fraying or tears and articular changes.

Scrutinize the mid-third and note any labral cartilage separations or fraying and degenerative changes. The surface may be smooth or have a cobblestone appearance in early degeneration.

As the scope is rotated to the anterior area, search for hypertrophy of the labrum in patients with dysplasia. The acetabular cartilage may be soft or may appear blistered or delaminated in dysplastics with anterior groin pain, instability, or popping. Look anterior beyond the labrum in the sulcus for synovitis and loose bodies. Move the scope to the superior sulcus of the joint to see the nonarticular side of the labrum in the

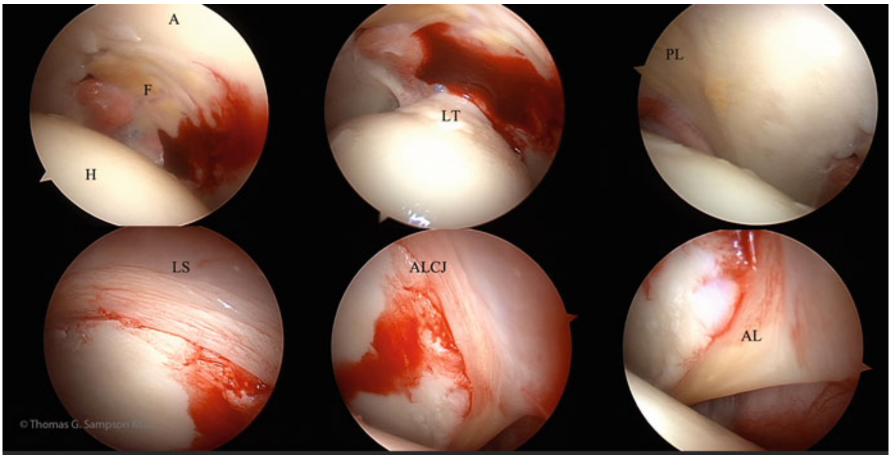


Fig. 11 Arthroscopic view of a *right* hip under room air. Acetabulum (A), head (H), labrum (L), fossa (F), ligamentum teres (LT), posterior labrum (PL), anterior labrum (AL), lateral sulcus (LS), anterior labrocartilaginous junction (ALCJ)

pericapsular space from anterior to posterior. Look for evidence of cysts, spurring, and labral tears. All the while, a probe or switching stick is used to probe.

Next observe as much of the femoral head with the same method, and if necessary rotate the leg while in traction. At this point we may switch to a 70° scope to look deeper into the notch and have a better view of the femoral head fovea with its ligamentum teres insertion.

After viewing from the anterolateral portal, the same procedure is carried out from the posterior portal or mid-anterior portal if not satisfied with the initial viewing. The corrective surgery is performed depending on the diagnosis and the distraction is completely released to allow the hip to be moved in rotation and flexion.

With the hip in slight flexion and neutral rotation, the 17 gauge needle is inserted through the anterolateral portal aiming along the femoral neck toward the head-neck junction to view the peripheral compartment. While observing under fluoroscopy, a small pop is felt as the needle passes through the capsule and the effusion dribbles out of the needle (Fig. 12). Pass a nitinol wire and bounce it off the medial capsule to confirm it is intra-articular. Advance the arthroscopic sheath over the wire and begin viewing the anterior, medial, inferior, and posterior peripheral spaces.

Fig. 12 Fluoroscopic view of a left hip with a nitinol wire in the peripheral compartment touching the medial capsule from the anterior portal

First note the femoral head seated in the labrum as it transforms into the transverse acetabular ligament. The zona orbicularis crosses the field and one may see the vincula-like vessel in the lateral synovial fold going into the femoral neck. Push the scope deep and posterior to view the sulcus and look for loose bodies.

As the scope is withdrawn, rotate it and advance it anterior medial and inferior to appreciate the reflection of the iliopsoas tendon on the capsule. The tendinous bulge on the capsule is

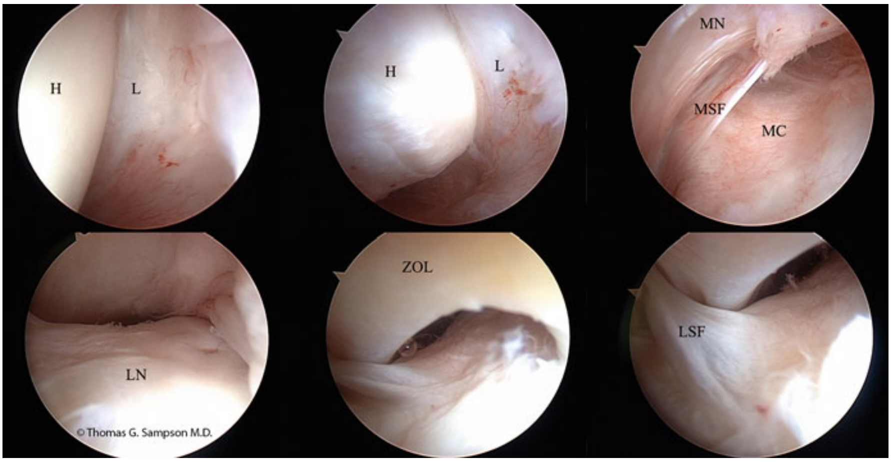


Fig. 13 Arthroscopic view of a right hip in the peripheral space. Head (H), labrum (L), zona orbicularis lateral portion (ZOL), medial neck (MN), medial synovial fold (MSF), medial capsule (MC), lateral neck (LN), lateral synovial fold (LSF)

usually opposite the inferior synovial fold at the head-neck junction and not to be mistaken for the zona orbicularis. Flexing the hip will relax the capsule for a larger field of view and improves the mobility of the scope and operative instruments (Fig. 13). A mid-anterior portal may be created at the level of the femoral neck midway between the head-neck junction and lesser trochanter for both the arthroscope and operative instruments. A far anteroinferior portal may be used at the level of the lesser trochanter for iliopsoas release.

At the completion of the procedure, close the wounds and apply a standard dressing. An intra-articular injection of a long-acting local anesthetic will make recovery and the trip home from the surgical center more tolerable.

In the recovery room, have the patient begin both passive and active range of motion of their hip. Crutches are used with the amount of weight bearing dependent on the diagnosis and treatment.

The dressings are removed in 24 h and the patient is allowed to shower. Therapy is started within a week to regain motion and strength.

Summary

Hip arthroscopy using the lateral approach was originally conceived as a method to access the central compartment of the hip reproducibly. Since inception, numerous advancements by surgeons and industry have created numerous improvements including cannulated instruments, distractors, longer arthroscopes, longer straight and curved shavers and burrs, and radiofrequency ablators as well as hip-specific instruments and anchors. All procedures described for hip arthroscopy can be achieved by this approach. The decision on whether to use the lateral or supine approach should be based on the surgeons training and comfort as the outcomes for either are similar and not position specific.

References

1. Dorfmann H, Boyer T, De Bie B. Arthroscopy of the hip. Rev Rhum Mal Osteoartic. 1988;55(1):33–6.
2. Dorfmann H, Boyer T, De Bie B. Arthroscopy of the hip. Methods and values. Rev Rhum Ed Fr. 1993;60(5):330–4.
3. Glick JM, et al. Hip arthroscopy by the lateral approach. Arthroscopy. 1987;3(1):4–12.
4. Eriksson E, Arvidsson I, Arvidsson H. Diagnostic and operative arthroscopy of the hip. Orthopedics. 1986;9(2):169–76.
5. Byrd JW. Hip arthroscopy utilizing the supine position. Arthroscopy. 1994;10(3):275–80.
6. Byrd JW. Hip arthroscopy. The supine position. Clin Sports Med. 2001;20(4):703–31.
7. Byrd JW. Hip arthroscopy: the supine position. Instr Course Lect. 2003;52:721–30.
8. Byrd JW. Hip arthroscopy by the supine approach. Instr Course Lect. 2006;55:325–36.

9. Telleria JJ, et al. Risk of sciatic nerve traction injury during hip arthroscopy-is it the amount or duration? An intraoperative nerve monitoring study. J Bone Joint Surg Am. 2012;94(22):2025–32.
10. Sampson TG. Complications of hip arthroscopy. Clin Sports Med. 2001;20(4):831–5.
11. Sampson TG. Complications of hip arthroscopy. Tech Orthop. 2005;20(1):63–6.
12. Gick JM, Sampson TG. Hip arthroscopy by the lateral approach. In: McGinty JB, editor. Operative arthroscopy. Philadelphia/New York: Lippincott-Raven; 1996. p. 1079–90.
13. Sampson TG, Glick JM. Indications and surgical treatment of hip pathology. In: McGinty JB, editor. Operative arthroscopy. Philadelphia/New York: Lippincott-Raven; 1996. p. 1067–78.
14. Burman MS. Arthroscopy or the direct visualization of joints: an experimental cadaver study. J Bone Joint Surg. 1931; 13:5–9.

Hip Arthroscopy: Portal Placement

18

Geoffrey D. Abrams, Joshua D. Harris, and Marc R. Safran

Contents

G.D. Abrams (✉)
Department of Orthopaedic Surgery, Stanford University, Palo Alto, CA, USA
e-mail: gabrams@stanford.edu; gabrams@gmail.com

J.D. Harris
Houston Methodist Center for Orthopaedics & Sports Medicine, Houston, TX, USA

Rush University Medical Center, Chicago, IL, USA
e-mail: joshuaharrismd@gmail.com

M.R. Safran
Stanford University School of Medicine, Redwood City, CA, USA
e-mail: msafran@stanford.edu

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_18

Abstract

The indications for hip arthroscopy are expanding. Initially, works within the central and peripheral compartments were described using a combination of three portals: anterolateral, anterior, and posterolateral. The anterolateral portal is typically the first portal made and is the only portal created without direct visualization. Fluoroscopy and tactile sensation are used to guide the spinal needle and cannula into the correct location, but iatrogenic chondral and labral injuries are potential risks. The anterior portal is in greatest proximity to a neurovascular structure, with the lateral femoral cutaneous nerve located more than 3 mm away. The mid-anterior portal is becoming more popular as it offers similar visualization as the anterior portal yet is more distant from the lateral femoral cutaneous nerve and offers an improved trajectory for suture anchor placement in labral repairs. Accessory portals and peritrochanteric space portals offer viewing and working abilities to address pathologies such as internal and external snapping hips, ischiofemoral impingement, gluteus medius and minimus repairs, and trochanteric bursectomy.

Introduction

When performing hip arthroscopy, portal placement is arguably the most important component of the procedure. Given the constrained nature of the hip joint, even small errors in the location of the portals can limit access to intra- and extra-articular structures. Anatomic landmarks are always utilized and fluoroscopy is commonly used to ensure accurate portal placement. This allows the surgeon to access all areas of the hip joint and surrounding structures as well as to avoid iatrogenic injury to neurovascular structures. There are numerous portals that have been described for hip arthroscopy in the central and peripheral compartment, including at least six different anterior portals. This chapter will review the senior author's portal preferences.

Anatomy

Among those that perform hip arthroscopy, a variety of portals have been described. One thing that remains constant, however, is the location of anatomic landmarks and neurovascular structures around the hip. A thorough knowledge of the location of these structures is important so they can guide positioning of the portals as well as help avoid complications.

The two most important surface landmarks that serve as reference points for portal placement are the anterior superior iliac spine (ASIS) and the greater trochanter. Both of these anatomic landmarks, particularly the greater trochanter, are accentuated when traction is applied to the extremity. In general, points medial and distal to the ASIS serve as potential locations for the femoral and lateral femoral cutaneous nerves (LFCN) as well as the ascending branch of the lateral femoral circumflex artery. The commonly utilized initial anterolateral portal is adjacent to the anterior-superior corner of the greater trochanter. Posterior to the greater trochanter lies the sciatic nerve, while superior to the tip of the trochanter is the superior gluteal nerve.

One difference between portal placement in the hip versus other joints is the large amount of soft tissue surrounding the hip. When placing the trocar into the hip joint, one must first penetrate the large muscular envelope around the joint. Following this, the thick hip capsule itself is encountered which may provide considerable resistance against the trocar [1]. The capsule contains discreet thickenings that comprise the capsuloligamentous stabilizers of the joint: iliofemoral (also known as the Y ligament of Bigelow), ischiofemoral, and pubofemoral ligaments [2, 3].

The hip joint itself is divided into the central and peripheral compartments, with the acetabular labrum marking the division between the two spaces. The central compartment contains the cartilage-covered articular surfaces of the femoral head and acetabulum, while the peripheral compartment contains the area surrounding the femoral neck, including the femoral head-neck junction. Thus, the central compartment is where the ligamentum teres as well as labral and articular cartilage injuries are identified and treated, whereas the peripheral compartment is where resection of a cam lesion may be performed. It is also one location for iliopsoas lengthening and where loose bodies and synovial pathology are often found [4]. The labrum is an important structure and serves to increase articular surface area [5] as well as oppose the flow of synovial fluid into and out of the central compartment [6, 7]. These functions allow for enhanced stability of the hip [8], maintain joint fluid within the central compartment for nutrition to the chondrocytes [7], provide more equal distribution of forces across the articular cartilage [9], and allow for a smooth gliding surface [10].

Patient Setup

Hip arthroscopy can be performed with equal efficacy in both the supine and lateral positions [11]. The senior author prefers placing the patient in the supine position on a fracture table so as to allow traction on the operative leg (Fig. 1). General anesthesia with muscle relaxation is recommended as well as a well-padded peroneal post to minimize the occurrence of postoperative pudendal nerve injury [12]. Utilizing the fracture table, gentle traction is placed on the operative leg to allow for approximately 1 cm of distance between the femoral head and acetabulum. This will allow space for the cannulae within the central compartment. The hip should be in neutral flexion-extension and slight abduction to neutral abduction-adduction. Some prefer approximately 15–20° internal rotation of the hip to offset the normal anteversion of the femoral neck, while others may place the hip in 10–20° of flexion. After proper positioning, the ASIS, greater trochanter, and future portal sites should be marked (Fig. 2). While it may seem obvious, the trochanter should not be outlined until after traction is applied, as it moves distal with traction.

Some prefer to "vent" the hip prior to prepping and draping to eliminate the vacuum phenomenon created with the leg in traction. This is done for two main reasons: (1) to assure the hip can be adequately distracted and (2) to reduce the amount of force necessary to distract the hip [13] This

Fig. 1 Intraoperative photo demonstrating patient setup following prepping and draping for a *right hip* arthroscopy. The *right leg* is in traction with the *left leg* abducted approximately 60°. The c-arm is positioned between the legs to aid in the establishment of portal placement as well as acetabular and cam lesion resection

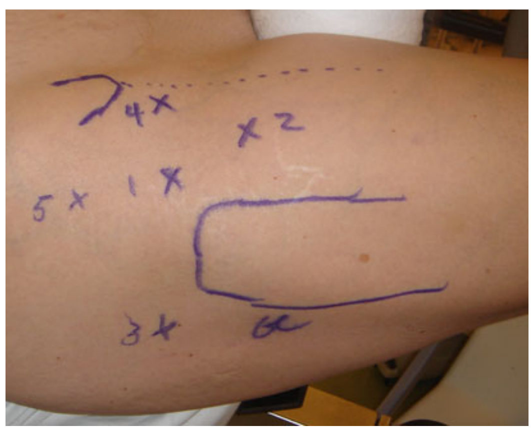


Fig. 2 Intraoperative photo showing provisional skin markings for portal placement. The anterior superior iliac spine (*ASIS*) and the greater trochanter are marked with a *dashed line* extending distally from the ASIS. The portals are (1) anterolateral, (2) mid-anterior, (3) posterolateral, (4) anterior, and (5) proximal anterolateral

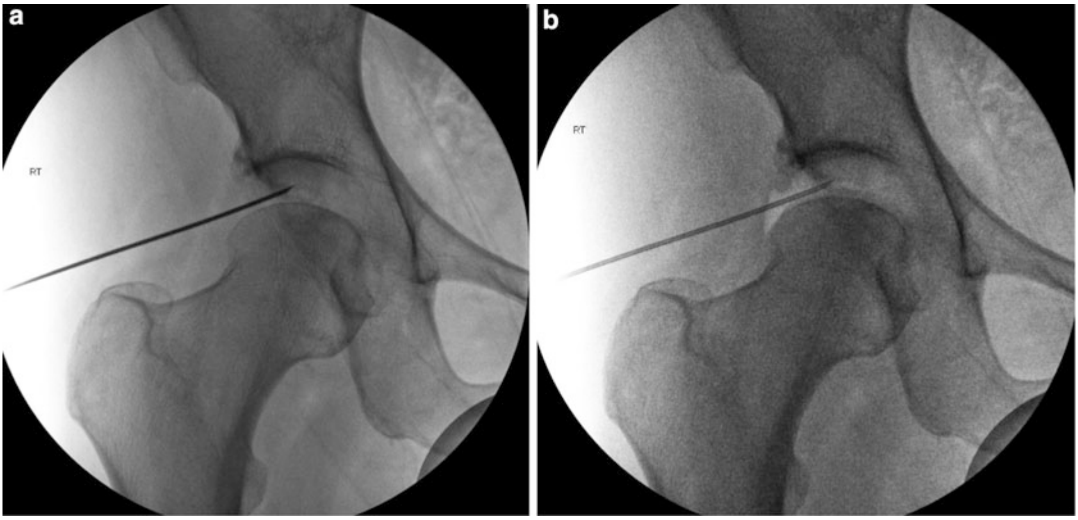


Fig. 3 (**a**) Fluoroscopic image of spinal needle used for localization of initial anterolateral portal following joint distraction. Note the trajectory of the needle adjacent to the femoral head (versus the acetabulum) so as to avoid penetration of the labrum. (**b**) Fluoroscopic image of spinal needle following removal of the stylus demonstrating the air arthrogram created and indicating intra-articular placement of the needle

involves placing the leg in traction and, using sterile technique and fluoroscopy, placing an 18-gauge 6-in. spinal needle at the location of the anterolateral portal (see below). The needle is introduced with the long end of the bevel away from the femoral head to reduce articular cartilage gouging. The path of the needle is such that the tip of the needle should end at the upper portion of the cotyloid fossa and the junction of the medial most aspect of the sourcil, with the shaft of the needle next to the femoral head. When the tip of the needle is in the correct location within the central compartment, the stylus is removed (Fig. 3a, b) to eliminate the negative intra-articular pressure created by the joint distraction. While confirming the air arthrogram with the fluoroscope, one will also notice the relaxation of the quadriceps muscle as air initially enters the joint, indicative of relaxation of the capsule and its proprioceptive mechanoreceptors.

Portal Overview

Hip arthroscopy portals utilized for working within the central compartment, peripheral compartment, and peritrochanteric space will be described. Most surgeons prefer two or three portals for standard hip arthroscopy involving the central and peripheral compartment.

The greatest risk in portal placement includes injury to neurovascular structures as well as iatrogenic injury to the cartilage and labrum. The structures at greatest risk will be discussed individually with each portal description. The most recent literature indicates major and minor complication rates following hip arthroscopy of 0.6 % and 7.5 %, respectively, with complications specifically related to portal placement of 0.5–0.6 % [12, 14].

Anterolateral Portal

Placement

The anterolateral portal is typically the first portal established as it lies most centrally in the safe zone for hip arthroscopy. Positioning of the other portals within the central compartment is facilitated by viewing through this portal. The closest structure is the superior gluteal nerve at an average of 4.4 cm proximal [15]. The entry point on the skin is adjacent to the anterior-superior border of the greater trochanter. Under fluoroscopic guidance

(Fig. 3a, b), a 6-in. 18-gauge spinal needle is directed through the gluteus medius muscle belly at approximately 15° cranially and 20–30° posteriorly to position the tip of the needle close to the femoral head . These are just general guidelines, as femoral version, femoral neck length, and neck-shaft angle vary. Another technique to identify the starting position and cranial-caudal angle is to overlay the needle on the skin. Using fluoroscopy, the needle is positioned to determine the skin entry point and angle of inclination by overlying the tip of the needle at the junction of the cotyloid fossa and sourcil, having the shaft of the needle next to the femoral head. Following the length of the needle to the skin will identify where the entry point should be along the length of the femur.

Fluoroscopy (the needle should be adjacent to the femoral head – Fig. 3a) and tactile sensation are utilized to avoid piercing the labrum with the spinal needle as subsequent placement of the trocar in this position will damage the labrum [13]. While minimal resistance is provided by the gluteus medius muscle belly, the hip capsule is a thick structure and provides greater resistance to needle penetration. Once the tip of the needle has penetrated the capsule, resistance should again be minimal as it enters the space of the central compartment. Again, the long end of the bevel of the needle should be away from the femoral head. Continued resistance will be experienced if the needle pierces the labrum or comes in contact with the femoral head or acetabulum.

The stylus of the spinal needle is then removed and a nitinol guide wire is placed through the needle. Leaving the guide wire in place, the needle is removed and a cannula with a cannulated obturator is placed over the guide wire. Some prefer to dilate the soft tissue around the portal with progressively larger obturators for easier placement of the final cannula (Fig. 4). After a second portal is established within the central compartment (the senior author prefers to create the mid-anterior portal next), a 70-degree arthroscope is inserted through this second portal to confirm appropriate placement of the anterolateral portal between the femoral head and acetabulum with avoidance of the labrum. If the anterolateral portal needs to be adjusted, this can be done with a spinal needle under direct visualization through the second portal.

Fig. 4 Fluoroscopic image of a cannula (with guide wire still in place) inserted into the anterolateral portal over a guide wire

Utility

Using a 70-degree arthroscope through the anterolateral portal, most of the structures within the central compartment can be visualized. Nearly the entire acetabular labrum can be seen; however, this portal provides the best visualization to the anterior and superior portions of this structure (Fig. 5). The articular cartilage surfaces of the femoral head and posterior-medial acetabulum are also well visualized (Fig. 6). Other structures that can be seen best through this portal are cotyloid fossa and ligamentum teres (Fig. 7), capsular-labral recess, and anterior triangle (Fig. 8). The anterior triangle is made up of the intra-articular portion of the iliofemoral ligament and capsule, the anterior superior portion of the femoral head, and the acetabular labrum. Visualization of this structure is particularly important as it allows for direct viewing of the spinal needle for creation of the anterior and/or mid-anterior portals.

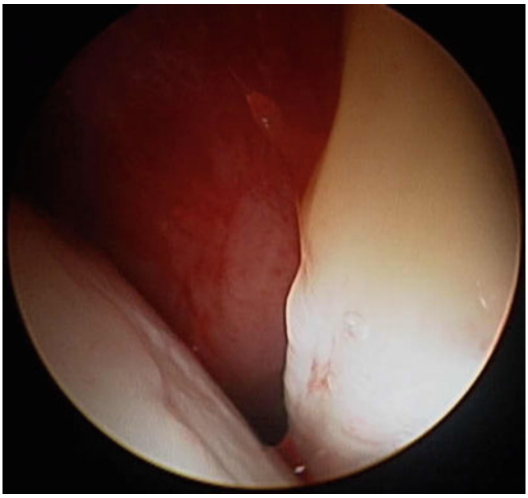


Fig. 5 Arthroscopic image with a 70-degree arthroscope of initial view through anterolateral portal showing the anterior-superior labrum on the *right*, femoral head on the *left*, and capsuloligamentous structure *centrally*

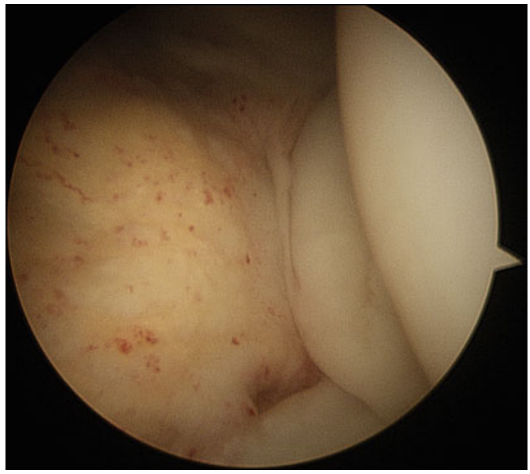


Fig. 7 Arthroscopic image with a 30-degree arthroscope from the anterolateral portal demonstrating the cotyloid fossa on the *left*, ligamentum teres *centrally*, and the femoral head on the *right*

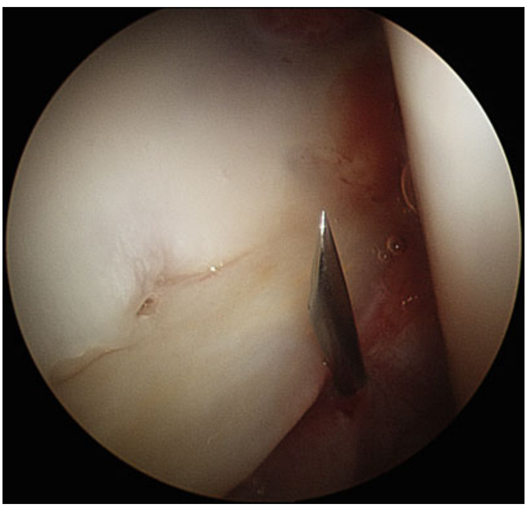


Fig. 6 Arthroscopic image with a 70-degree arthroscope from the anterolateral portal demonstrating spinal needle localization of the posterolateral portal. The acetabular cartilage and labrum can be seen on the *left* and the femoral head articular cartilage on the *right*

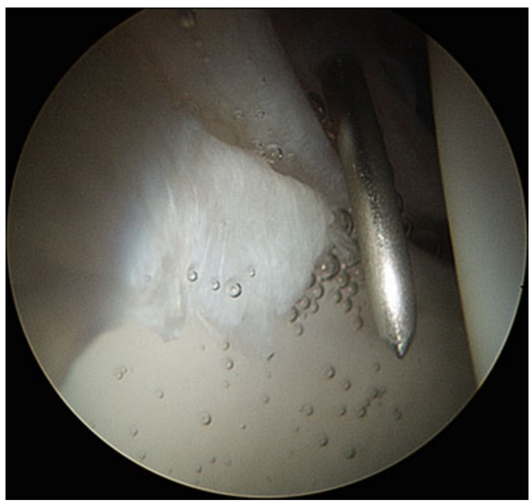


Fig. 8 Arthroscopic image with a 70-degree arthroscope from the anterolateral portal showing needle localization of the mid-anterior portal through the anterior triangle. The anterior-superior labrum is on the *left*, femoral head is on the *right*, and the needle is piercing the capsuloligamentous structure

The anterolateral portal can also be used to work and/or visualize within the peripheral compartment of the hip. To access this compartment, traction is released and the camera is placed through the anterolateral portal at the junction of the femoral head and neck. Fluoroscopy can be useful to confirm the correct location. The femoral neck and anterior and superior aspects of the non-articulating portion of the femoral head can be visualized for eventual cam resection.

Risks

The most significant risk in placement of anterolateral portal is not the superior gluteal nerve, which lies an average of over 4 cm proximal, but rather iatrogenic chondral-labral injury. This is due to the fact that it is the only portal that is placed without direct visualization. The utilization of both fluoroscopy and tactile sensation is important in the placement of this portal to avoid damage to intra-articular structures.

Anterior Portal

Placement

This portal originates at the intersection of a line drawn distally from the ASIS and a transverse line from the tip of the greater trochanter (Fig. 2). It is approximately 6.3 cm distal to the ASIS. When inserting the spinal needle in this location, the portal is aimed approximately 40° cephalad and 25–30° toward the midline. The muscle bellies of the sartorius and the rectus femoris are traversed before entering the hip through the anterior capsule. With the 70-degree arthroscope typically in the anterolateral portal and with a clear view of the anterior triangle (described above), this portal is made under direct visualization.

Utility

When it is the second portal created, one should first view through the anterior portal to confirm that the anterolateral portal is in the correct location and has not pierced the labrum. It can also be used as a working and/or viewing portal for evaluation and treatment of associated pathology. The anterior portion of the femoral head, the ligamentum teres, the acetabular fossa, and the superior labrum can be visualized from this location. The senior author typically creates a mid-anterior portal (see below) rather than a traditional anterior portal but does utilize the anterior portal position for labral takedown and retraction as an accessory portal when indicated (Fig. 9).

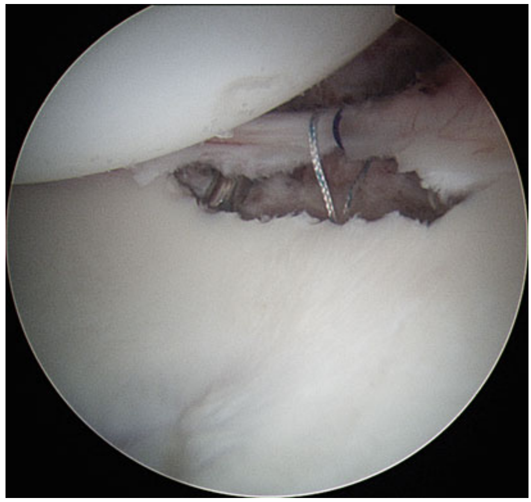


Fig. 9 Arthroscopic image from the posterolateral portal with a 70-degree arthroscope demonstrating labral repair. A suture anchor drill guide is placed through the mid-anterior portal in preparation for anchor placement. A traction stitch is exiting the anterior portal and providing retraction of the labrum for visualization

Risks

The LFCN is at greatest risk with this portal as it can be found within 3 mm [15]. The femoral nerve and the ascending branch of the lateral femoral circumflex artery (LFCA) are both approximately 4 cm away. Terminal branches of the LFCA, however, may be as close as 2 mm [15]. Because of the proximity of the LFCN, a superficial skin incision should be made followed by blunt dissection to protect the nerve. As long as the portal is not medial to the line drawn distally from the ASIS, the femoral artery and vein should not be at risk. Creation of this portal under arthroscopic visualization, typically through the anterolateral portal, minimizes the risk of cartilage or labral injury.

Mid-Anterior Portal

Given the proximity of the anterior portal to the LFCN and the recent interest in labral repair, the mid-anterior portal is becoming increasingly utilized instead of the traditional anterior portal. The senior author utilizes this portal, along with the anterolateral and posterolateral portals, when performing hip arthroscopy. This portal is placed using an outside-in technique similar to the placement of the anterior portal. It is located 5–7 cm distal to the anterolateral portal at a 45° angle (Fig. 2). This allows for the same visualization of structures as would be obtained through the anterior portal but with less risk to the LFCN [16] and an improved angle for placement of superior and anterior anchors for labral repair (Fig. 9) as well as easier and safer access in cases of pincer impingement with significant anterior acetabular overcoverage.

Posterolateral Portal

Placement

The posterolateral portal is placed 1 cm posterior to the superior-posterior tip of the greater trochanter and under arthroscopic visualization through one of the anterior portals (Fig. 6). The portal traverses just posterior to the gluteus medius and minimus and proximal to the piriformis tendon [17]. As the hip is more internally rotated, the trajectory of this portal becomes more parallel with the floor. However, internal rotation brings the sciatic nerve closer to the joint capsule, increasing risk of injury to the nerve. External rotation should also be avoided as that brings the greater trochanter more posterior. This reduces the zone of safety with regard to the sciatic nerve. It should also be approximately parallel to the anterolateral portal.

Utility

This portal provides visualization to the weight-bearing dome of the acetabulum, posterior-medial as well as anterolateral labrum, and the femoral head. The posterior labral recess, floor of the acetabular fossa, and inferior gutter can also be seen and are common areas for loose bodies to be found.

Risks

Minimal risks to neurovascular structures are present when the portal is in the appropriate position. The sciatic nerve is, on average, 2.9 cm away [15], while the deep branch of the medial femoral circumflex artery is just over 1 cm away, assuming normal trochanteric anatomy [18]. The risk to the sciatic nerve is increased with hip internal rotation or external rotation. Given that the portal is placed under direct arthroscopic visualization, the risk of iatrogenic injury to articular structures is small.

Accessory Portals

Proximal and Distal Anterolateral Portals

The senior author prefers to utilize proximal and/or distal anterolateral portals when working in the peripheral compartment for a cam lesion resection, iliopsoas release, or lesser trochanter resection for ischiofemoral impingement. For a cam resection, visualization with a 30-degree arthroscope is performed through the standard anterolateral portal. An accessory proximal anterolateral portal is created in line with and approximately 3–4 cm proximal to the anterolateral portal (Fig. 2). A shaver is first introduced through this proximal anterolateral portal to remove soft tissue overlying the capsule prior to capsulotomy. Once the cam lesion has been identified at the femoral head-neck junction, a burr is used for resection through this working portal.

The distal anterolateral portal is also in line with the anterolateral portal but 3–4 cm distal. It is often utilized for percutaneous suture anchor placement during anterior and anterolateral labral repair. For more distal pathology, including iliopsoas release and lesser trochanteric resection for ischiofemoral impingement, a distal anterolateral portal is made 3–5 cm distal to the anterolateral portal and 1–2 cm anterior. This allows easier access to the lesser trochanter or release of the iliopsoas at the level of the lesser trochanter.

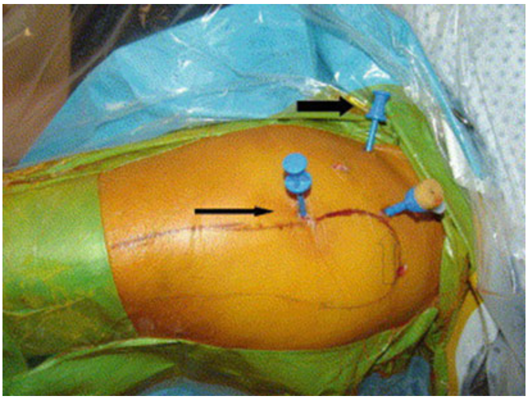


Fig. 10 Intraoperative photograph of a *left hip* demonstrating placement of peritrochanteric space portals with the anterior portal (*thick arrow*) and distal posterior portal (*thin arrow*). A third portal (*open arrow*) can be placed proximal to the tip of the greater trochanter for additional viewing and working (Reprinted from Voos et al. [19], with permission from Elsevier)

Peritrochanteric Space Portals

Anterior Portal

The anterior portal (different than described above) offers visualization of the peritrochanteric space. This portal is placed 1 cm lateral to the ASIS and inserted between the tensor fascia lata and sartorius (Fig. 10). With the leg in neutral flexion-extension and adduction-adduction and slight internal rotation, a small skin incision is made, and the cannula is bluntly directed posteriorly into the peritrochanteric space [19]. It is swept back and forth between the iliotibial band and the greater trochanter to open up this area. If done properly, one should clearly be able to see the greater trochanter and iliotibial band with the 70-degree arthroscope. Tears of the gluteus medius and minimus tendons can also be seen through this portal.

Distal Posterior Portal

This portal is placed midway between the tip of the greater trochanter and the vastus tubercle along the posterior one third of the greater trochanteric midline (Fig. 10). This working portal placement allows for access distally and proximally for procedures such as iliotibial band release, trochanteric bursectomy, and gluteus medius and minimus repairs. In addition, a third portal can be placed proximal to the tip of the greater trochanter in line with the distal posterior portal to further facilitate access to the peritrochanteric space and serve as an additional working portal for these procedures.

Summary

Given the articular congruity of the hip joint, accurate portal placement is critical to avoiding iatrogenic neurovascular injury as well as both visualizing and addressing hip pathology. The anterolateral portal is distant from neurovascular structures, but given the fact that it is the only portal placed under direct visualization, it has the highest risk of iatrogenic chondral and labral injury. The anterior portal provides the greatest risk for nerve injury during portal placement, with the LFCN an average of 3 mm away. While the traditional anterolateral and posterolateral portals are still commonly used and relatively safe, the mid-anterior portal is gaining in popularity over the anterior portal as it offers less risk of LFCN injury and offers an improved angle for suture anchor placement in labral repairs. Accessory portals include the proximal and distal anterolateral portals and they may be used for cam lesion resection, suture anchor placement for labral repair, iliopsoas release, and lesser trochanteric resection in ischiofemoral impingement. Peritrochanteric space portals prove useful when arthroscopically addressing gluteus medius and minimus tears, external snapping hip, and trochanteric bursitis.

References

1. Roy DR. Arthroscopy of the hip in children and adolescents. J Child Orthop. 2009;3:89–100.
2. Wagner FV, Negrao JR, Campos J, et al. Capsular ligaments of the hip: anatomic, histologic, and positional study in cadaveric specimens with MR arthrography. Radiology. 2012;263:189–98.

3. Telleria JJ, Lindsey DP, Giori NJ, et al. An anatomic arthroscopic description of the hip capsular ligaments for the hip arthroscopist. Arthroscopy. 2011;27:628–36.
4. Dienst M, Seil R, Kohn DM. Safe arthroscopic access to the central compartment of the hip. Arthroscopy. 2005;21:1510–4.
5. Seldes RM, Tan V, Hunt J, et al. Anatomy, histologic features, and vascularity of the adult acetabular labrum. Clin Orthop Relat Res. 2001;382:232–40.
6. Ferguson SJ, Bryant JT, Ganz R, et al. The acetabular labrum seal: a poroelastic finite element model. Clin Biomech (Bristol, Avon). 2000;15:463–8.
7. Ferguson SJ, Bryant JT, Ganz R, et al. An in vitro investigation of the acetabular labral seal in hip joint mechanics. J Biomech. 2003;36:171–8.
8. Crawford MJ, Dy CJ, Alexander JW, et al. The 2007 Frank Stinchfield Award. The biomechanics of the hip labrum and the stability of the hip. Clin Orthop Relat Res. 2007;465:16–22.
9. Safran MR. The acetabular labrum: anatomic and functional characteristics and rationale for surgical intervention. J Am Acad Orthop Surg. 2010;18:338–45.
10. Song Y, Ito H, Kourtis L, et al. Articular cartilage friction increases in hip joints after the removal of acetabular labrum. J Biomech. 2012;45:524–30.
11. Pollard TC, Khan T, Price AJ, et al. Simulated hip arthroscopy skills: learning curves with the lateral and supine patient positions: a randomized trial. J Bone Joint Surg Am. 2012;94:e68.
12. Harris JD, McCormick FM, Abrams GD, et al. Complications and reoperations during and after hip arthroscopy: a systematic review of 92 studies and more than 6,000 patients. Arthroscopy. 2013;29:589–95.
13. Byrd JW. Avoiding the labrum in hip arthroscopy. Arthroscopy. 2000;16:770–3.
14. Clarke MT, Arora A, Villar RN. Hip arthroscopy: complications in 1054 cases. Clin Orthop Relat Res. 2003;406:84–8.
15. Byrd JW, Pappas JN, Pedley MJ. Hip arthroscopy: an anatomic study of portal placement and relationship to the extra-articular structures. Arthroscopy. 1995;11:418–23.
16. Robertson WJ, Kelly BT. The safe zone for hip arthroscopy: a cadaveric assessment of central, peripheral, and lateral compartment portal placement. Arthroscopy. 2008;24:1019–26.
17. Byrd JW. Hip arthroscopy. J Am Acad Orthop Surg. 2006;14:433–44.
18. Sussmann PS, Zumstein M, Hahn F, et al. The risk of vascular injury to the femoral head when using the posterolateral arthroscopy portal: cadaveric investigation. Arthroscopy. 2007;23:1112–5.
19. Voos JE, Rudzki JR, Shindle MK, et al. Arthroscopic anatomy and surgical techniques for peritrochanteric space disorders in the hip. Arthroscopy. 2007;23:1246.e1–5.

Hip Arthroscopy: Central Compartment Access

19

Niv Marom, Mathew Hamula, and Srino Bharam

Contents

Abstract

Recently, the field and awareness of hip arthroscopy have significantly expanded amongst surgeons managing hip disorders. Pathologies arising from the intra-articular space of the hip joint, also referred to as the central compartment of the hip, remain the primary indications for this procedure. This chapter will focus on issues relevant to the arthroscopic access to the central compartment. These issues include the unique characteristics of the hip joint and surrounding tissues, relevant anatomical structures, the introduction of the standard portals used, their establishment and utilization, safety concerns and risks, the process of proper patient selection, and preoperative planning. This chapter provides the necessary base of knowledge needed prior to performing this procedure.

Introduction

Arthroscopic management of the hip has evolved significantly since hip arthroscopy was described in the early 1930s [1]. For many years, hip arthroscopy was primarily a diagnostic and therapeutic tool for pathology arising from the space within the hip joint. This space is commonly referred to as the central compartment of the hip. As hip arthroscopy evolved, two additional compartments for arthroscopic procedures were described: the peritrochanteric compartment [2]

N. Marom (✉)
Department of Orthopaedic Surgery, Lenox Hill Hospital, New York, NY, USA

Department of Orthopaedic Surgery, Meir Medical Center, Kfar saba, Israel
e-mail: niv.marom@gmail.com

M. Hamula • S. Bharam
Department of Orthopaedic Surgery, Lenox Hill Hospital, New York, NY, USA
e-mail: mhamula@gmail.com; srino.bharam@gmail.com

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_19

and the peripheral compartment, providing access to the femoral neck, outer acetabular rim, and peripheral labrum [3]. The advent of these compartments enabled the expansion of arthroscopic indications to treat a broader spectrum of pathologies within and around the hip joint.

Appropriate portal placement is fundamental to obtain adequate access to all compartments. Portal positioning is simultaneously a critical part of the procedure and, some might say, the most challenging part. Improper portal positioning has the potential to add complexity to the procedure as well as damage important and vital anatomical structures, hence compromising procedure safety and outcomes.

General Considerations

Hip arthroscopy can be performed with the patient positioned either in the supine or lateral decubitus position. The same portal placement is used in both positions [4–6]. The anterior superior iliac spine (ASIS) and tip of the greater trochanter are the most important superficial anatomical landmarks that serve as reference points in portal placement for the central compartment. Additional anatomical reference points include the symphysis pubis and femoral shaft.

Standard portals for the central compartment consist of the anterolateral (AL), anterior (AP), and posterolateral (PL) portals. More recently, the midanterior portal (MAP) is being increasingly utilized. The AL portal is the most commonly used introductory portal for hip arthroscopy and is the working portal for many arthroscopic hip procedures.

Portal placement is a demanding task that requires careful consideration of the unique anatomy of the hip joint and surrounding tissues. The hip joint is a constrained ball-and-socket joint with a relatively small intra-articular volume (normal range, 0.7–5.6 mL) [7]. Important intra-articular structures include the acetabular and femoral articular hyaline cartilage and the acetabular labrum, which effectively seals off the deep central part of the joint. Surrounding the hip joint are a thick soft-tissue mantle and the thickened hip joint capsule. Neurovascular structures in close proximity to the hip joint should also be taken into consideration. Important structures include the lateral femoral cutaneous nerve (LFCN), femoral nerve (the most lateral of the femoral neurovascular structures), sciatic nerve, superior gluteal nerve, and branches of the lateral circumflex femoral artery (LCFA) and medial circumflex femoral artery (MCFA) [8–12].

Initial entry into the central compartment of the hip carries a high risk of iatrogenic injury to the structures mentioned above. The intra-articular labrum is especially at risk due to its location directly along the path of the common initial portal placement. Scuffing the femoral head and/or acetabular articular cartilage is also possible when first entering the hip joint [8, 13–16].

Additional factors that might influence portal positioning and complicate access to the hip joint include coxa vara/valga, excessive bony abnormalities seen with femoroacetabular impingement, excessive version deformities, hip dysplasia, coxa profunda, hip joint arthritis with degenerative changes, and abnormal body habitus. These findings must be addressed and should be taken into consideration prior to arthroscopy.

Patient selection and preoperative planning are integral components to achieve successful outcomes in hip arthroscopy. Arthroscopic hip surgery should be advocated for patients with clinical and imaging findings suggestive of pathology amenable to arthroscopic intervention established by current evidence-based guidelines. It is imperative that the surgeon preoperatively plan the appropriate surgical procedure according to the underlying pathology and discuss with the patient the steps of the procedure and manage expectations in terms of outcomes [17].

Patient Setup

Hip arthroscopy is commonly performed under general anesthesia with muscle relaxation. Epidural anesthesia is an alternative option, but requires an adequate motor block to ensure optimal distractibility of the joint. Regional anesthesia can be used as an adjunct in postoperative pain

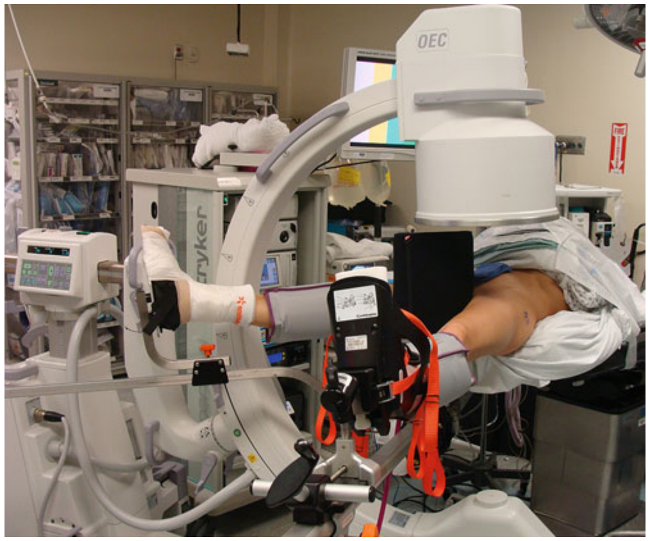


Fig. 1 Patient setup in the supine position for left hip arthroscopy

management [17, 18]. The patient can be placed either supine or in the lateral decubitus position for the procedure, according to surgeon preference. Both positions have been shown to yield comparable results. The supine position will be described, as it is perceived as simpler and more commonly utilized. The lateral approach may be preferable for patients with obesity or morbid obesity.

The arthroscopic and fluoroscopic towers are placed on the opposite side of the operative extremity, with the fluoroscopic monitor at the foot of the patient and the C-arm image intensifier centered over the operative hip. The patient is placed in the supine position on a fracture table or radiolucent table with traction capability. The feet are wrapped with padding and securely attached to the foot holders. A well-padded perineal post is attached to the operating table. The perineal post is positioned laterally against the proximal medial thigh of the surgical hip. This provides an optimal moment arm for distraction once traction forces are applied and reduces direct pressure on the perineum, minimizing the risk of neuropraxia of the pudendal nerve. Patient setup in the supine position is demonstrated in Fig. 1.

Gentle hip distraction is applied to obtain approximately 10 mm of joint distraction confirmed fluoroscopically (see Fig. 2a, b). The goal is to use the minimal force required to achieve adequate distraction and keep traction time as brief as possible (preferably less than 2 h). It is important that the distraction system employed allows for adequate mobility of the operative hip specifically in flexion, abduction, and/or adduction of the hip. Freedom of movement in these planes is essential to achieving sufficient visualization and optimal maneuverability during surgery [19, 20].

Portal Placement

After proper traction is applied, the operative extremity is placed in neutral abduction. Slight hip flexion can be added, as it may relax the anterior capsule and aid in portal placement. The principal landmarks are identified and marked including the greater trochanter, anterior superior iliac spine (ASIS), pubic symphysis, and femoral shaft. Arthroscopic access to the central compartment of the hip joint is based on two to three standard portals that are routinely established: anterolateral (AL), anterior, and/or posterolateral (PL) portals. A more commonly used portal in recent years is the midanterior portal (MAP). The AL portal is used as the introductory portal for virtually all routine hip arthroscopy. The anatomical landmarks and position of these portals are demonstrated in Fig. 3.

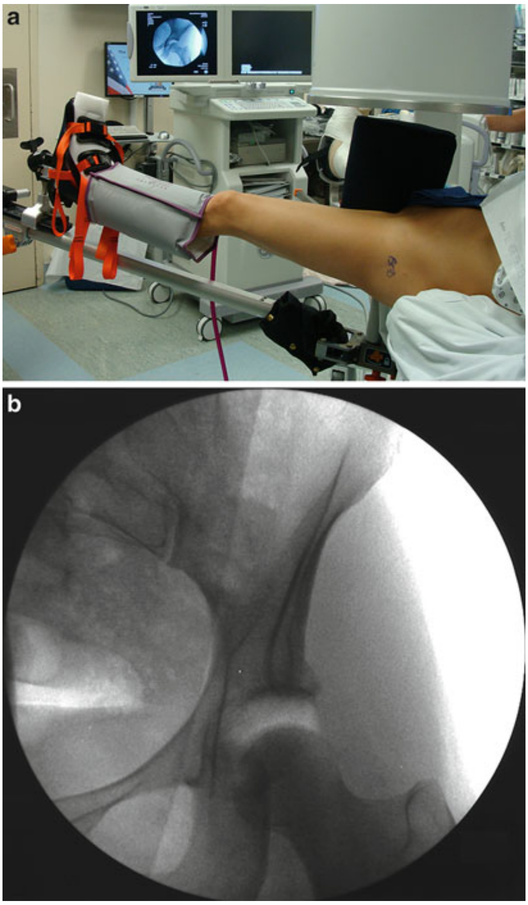


Fig. 2 (**a**) The left lower extremity is seen in approximately 10 mm of distraction and (**b**) confirmed fluoroscopically

Anterolateral Portal

The AL portal is usually established as the introductory portal. It is easier to access, reproducible, and presents relatively less risk to the surrounding neurovascular structures. The entry point on the skin is approximately 1 cm anterior and 1 cm superior to the anterosuperior tip of the greater trochanter. This portal was traditionally described as penetrating the gluteus medius muscle and entering the lateral capsule at its anterior margin [17]. Another well-described technique for establishing the AL portal is through the intermuscular interval between the abductors and the tensor fasciae latae. In some patients, when traction is applied to the operative extremity, a palpable ridge can be identified along the anterolateral thigh at the level of the AL portal insertion site. This palpable ridge is formed by the transition zone between the posterior border of the tensor fascia lata and the anterior border of the gluteus maximus fascia, which merge in line with the anterior aspect of the greater trochanter. When done properly, the portal will penetrate the gluteal fascia and then pass down to the joint capsule with minimal soft-tissue resistance as it passes atraumatically between the tensor fasciae latae anteriorly and the gluteus medius posteriorly [11].

The joint capsule is punctured with a large-gauge spinal needle (17 G or 16 G) under fluoroscopic guidance. The needle should be inserted through the AL portal insertion site with a slightly posterior (20–30°) and cephalad (10–20°) trajectory toward the hip joint and between the femoral head and acetabular labrum. A tactile feedback of a resistance felt when the needle penetrates the thick joint capsule with an immediate decrease of this resistance after penetration of the capsule can signify proper positioning of the needle without injury to the intra-articular structures at risk.

Successful entry into the central compartment of the hip joint is confirmed with an air arthrogram. This is performed by removal of the spinal needle stylus and breaking the vacuum seal of the joint. This should provide for additional distraction of the hip joint and often enables fluoroscopic visualization of a silhouette of the acetabular labrum. The joint can also be filled with saline at this time with observed backflow confirming the intra-articular position of the spinal needle. A nitinol guidewire is then placed through the spinal needle, and a stab incision is made through the skin adjacent to the needle. The needle is removed, leaving the nitinol wire and cannulated dilators placed over the guidewire to allow for easier passage of the final obturator. It is important to direct the various cannulas superiorly away from the convexity of the femoral head in order to avoid inadvertent scuffing of the articular cartilage [18].

A 70° arthroscope is then used to perform a diagnostic arthroscopic examination and to establish the other portal(s) under direct visualization.

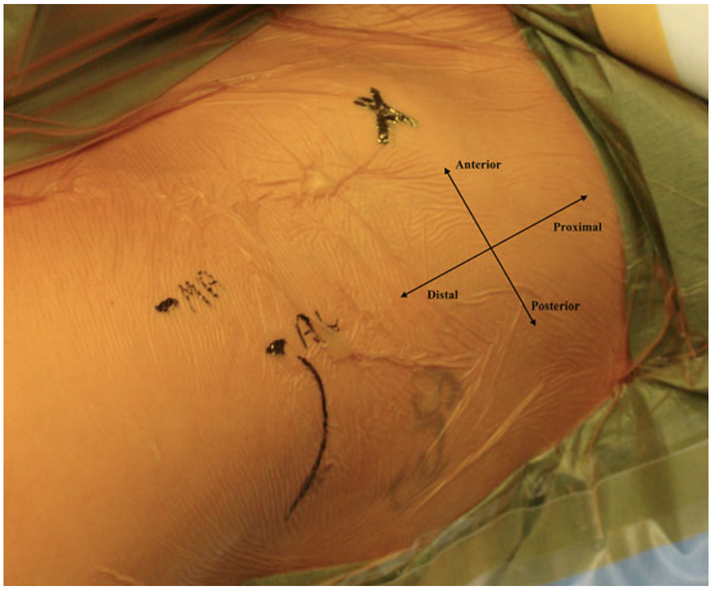


Fig. 3 Anatomical landmarks are outlined including the greater trochanter (*curved line*) and anterosuperior iliac spine (marked "X"); the anterolateral (*AL*) and midanterior (*MA*) portals are also labeled

This process of spinal needle, nitinol wire, dilators, and obturator placement is used for all subsequently portals and is an important aspect of safe portal placement. After placement of other portals, the arthroscope can be introduced into another portal in order to visualize the position of the anterolateral portal in relation to the acetabular labrum and make adjustments if necessary.

The anterolateral portal in the central compartment enables arthroscopic visualization of the following structures: cotyloid fossa, pulvinar, ligamentum teres, posterior medial acetabulum and labrum, anterior labrum, anterior capsule, paralabral sulcus, and the intra-articular portion of the psoas tendon and corresponding bursa. The anterior triangle, containing the anterior capsule, anterior labrum, and femoral head, is a very helpful landmark allowing for direct visualization of the spinal needle when establishing the anterior portal (see Fig. 4) [18].

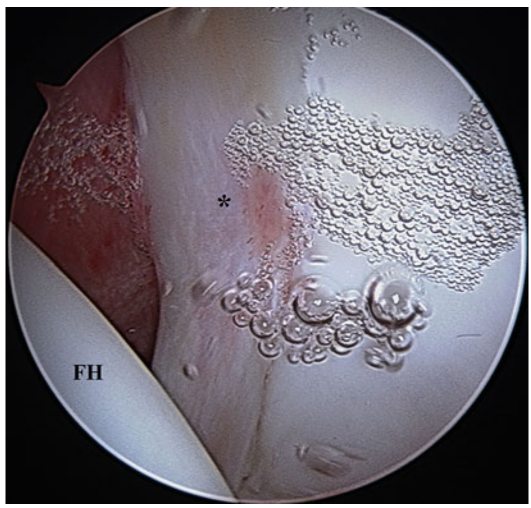


Fig. 4 Structures visualized through the anterolateral portal including the anterior triangle, anterior labrum/capsule (*asterisk*), and femoral head (*FH*)

Risks

The anterolateral portal is located centrally within the safe zone of access to the hip joint, minimizing the risk of damage to neurovascular structures. The superior gluteal nerve, coursing on the deep surface of the gluteus medius, and the sciatic nerve are the closest neurovascular structures to this portal located a mean distance of 64.1 and 40.2 mm, respectively, from the anterolateral portal [11].

The main concern when establishing the AL portal under solely fluoroscopic control is possible damage to the intra-articular structures such as scuffing the articular cartilage of the femoral head or perforating the acetabular labrum. A recent study of 250 patients reported an iatrogenic labral puncture rate of up to 20 % [13]. Strategies to minimize labral injury include using adequate distraction, joint distention, tactile feedback, reintroducing the spinal needle after distention of the joint with saline, as well as spinal needle repositioning with or without arthroscopic visualization from other portals. Careful attention to detail and proper technique can reduce the likelihood of iatrogenic injury [10, 13, 18].

In addition, novel techniques for minimizing the risks while accessing the central compartment of the hip joint have been described and published in recent years. One technique describes placing the arthroscope first in the peripheral compartment through the anterolateral portal followed by placement of a spinal needle and guidewire into the central compartment via an anterior portal under direct arthroscopic visualization [3]. Another technique describes directing the AL portal needle slightly anteroinferior to the clear space of the distracted joint overlapping the superior part of the femoral head, thereby directing the needle away from the labrum [14].

Anterior Portal

Several variations of positioning the anterior portal have been described. The traditional location of the skin entry point of the anterior portal was at the intersection of a sagittal line drawn down from the anterior superior iliac spine (ASIS) and a transverse line from the superior border of the greater trochanter. Insertion of the spinal needle is aimed approximately 45° cephalad and 30° toward the midline. It was found that when positioned this way, in line with the ASIS, this portal penetrates the sartorius muscle and the rectus femoris muscle before entering the anterior joint capsule and passes in dangerously close proximity to branches of the lateral femoral cutaneous nerve (3 mm) and the ascending lateral circumflex femoral artery (3 mm) [11, 12, 17]. Some have advocated placing the AP portal 1 cm lateral to its traditionally described location (i.e., 1 cm lateral to the ASIS) reducing the risk of lateral femoral cutaneous nerve (LFCN) and lateral circumflex femoral artery (LCFA) iatrogenic injury. When positioned laterally, the portal penetrates the muscle belly of the tensor fasciae latae (TFL) and passes through an interval between the gluteus minimus and rectus femoris before entering the joint through the anterior capsule. It courses a mean distance of 15 mm lateral to the LFCN and its branches, 31 mm proximal to the ascending LCFA, and 15 mm lateral to the ascending LCFA terminal branch [11].

The anterior portal is established under direct visualization using a 70° arthroscope in the anterolateral portal. The spinal needle is directed toward the anterior triangle. The anterior portal in the central compartment enables arthroscopic visualization of the following structures: ligamentum teres, posterior transverse ligament, posteromedial labrum, anterior transverse ligament, anterior labrum, superior articular cartilage, lateral labrum, and posterolateral capsule.

Risks

Special care and attention should be taken when inserting the AP portal. As mentioned, branches of the lateral femoral cutaneous nerve and lateral circumflex femoral artery are in close proximity to the portal and most at risk. Positioning the skin entry point 1 cm laterally places the portal path further away from these structures and may mitigate this risk. An important consideration is the anatomical variation of the proximal LFCN branching site, present in nearly 25 % of patients [19]. In these cases, the most lateral branch of the LFCN might be even closer to the AP portal. Stab incisions should be avoided in the anterior portal in an effort to prevent iatrogenic injury to these branches. The femoral neurovascular bundle is normally located a safe mean distance of more than 3 cm from the portal, and the vertical line marked from the ASIS distally can be used as the medial border of the safe zone. Because this portal is established under direct visualization, the risk to intra-articular structures should be minimal.

Midanterior Portal (MAP)

The midanterior portal is becoming more popular in recent years. Some have adopted this portal and the AL portal as part of the standard two-portal hip arthroscopy technique. The MAP was described in several variations including halfway between the anterior and AL portals and either 2–7 cm distally at a distal angle of 45° from the AL portal or as the third vertex of an equilateral triangle with the AP and AL portal as the remaining two proximal vertices [11, 18]. The placement of the midanterior portal may slightly vary depending on patient body habitus. This portal is also established under direct arthroscopic visualization and allows for a better-angled placement of superior and anterior anchors for labral repair. The midanterior portal in the central compartment enables arthroscopic visualization of the same structures as the anterior portal with the addition of the peripheral compartment.

Risks

The midanterior portal has been associated with penetration of the TFL before passing through the gluteus minimus–rectus femoris interval. There is a decreased risk of LCFN injury because this portal is placed laterally and distally to the traditional and modified anterior portal. The terminal branch of the ascending LCFA was found to be the closest neurovascular structure to the MAP with a mean distance of 10 mm (range, 1–23 mm). There is no report in the literature of significant intraoperative or postoperative bleeding using this portal. However, this theoretical risk should still be taken to consideration.

Posterolateral Portal

The posterolateral portal is placed 1 cm superior and 1 cm posterior to the tip of the greater trochanter or the posterior superior border of the greater trochanter. It is established under direct visualization from either the anterior or the anterolateral portal. The portal enters the hip joint at the posterior margin of the lateral capsule [17]. When the hip is in slight internal rotation, compensating for femoral neck anteversion, the needle is aimed almost parallel to the floor for joint entry [20]. The posterolateral portal provides visualization of the following structures within the central compartment of the hip joint: the inferior gutter of the hip joint, the weight-bearing dome of the acetabulum, anterolateral labrum, and femoral head (Fig. 5).

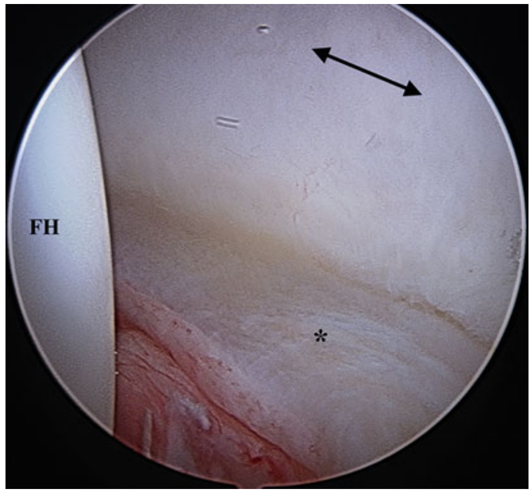


Fig. 5 Posterolateral compartment: the inferior gutter of the hip joint, the weight-bearing dome of the acetabulum (*two-sided arrow*), anterolateral labrum (*asterisk*), and femoral head (*FH*)

Risks

The sciatic nerve has been described as residing a safe mean distance of 22 mm from the PL portal to the central compartment [11, 12]. It is in closest proximity at the level of the joint capsule. The closest neurovascular structure to the PL portal was the deep branch of the medial circumflex femoral artery at a mean distance of 10.1 mm inferior to the PL portal as it passes through the piriformis tendon. The posterior tip of the greater trochanter seems to protect this important vessel, which is the main blood supplier to the femoral head, and functions as a bony boundary for the trocar. Any change of normal anatomy (pathologic or iatrogenic) of the posterior aspect of the greater trochanter must be evaluated properly prior to the surgery and addressed during the surgery [20–22]. Risk to intra-articular structures should be minimal, as this portal is established under arthroscopic visualization.

Challenges to Central Compartment Access

Portal placement can be complicated by several patient-specific pathologies. Detached anterosuperior labral tears can make it difficult to establish the anterior portal (Fig. 6). Large pincer lesions and global overcoverage may increase the amount of distraction needed for visualization. Structural bony abnormalities such as coxa vara/valga and focal retroversion may slightly alter optimal portal placement. An alternative is to enter into the peripheral compartment first and perform a peripheral labrum takedown and rim trimming to allow access to the central compartment [23]. Patients with hip dysplasia and a hypertrophic labrum are especially at risk of iatrogenic labral injury. Surgeons should be mindful of these challenges, plan preoperatively, and make the appropriate adjustments in order to optimize patient outcomes.

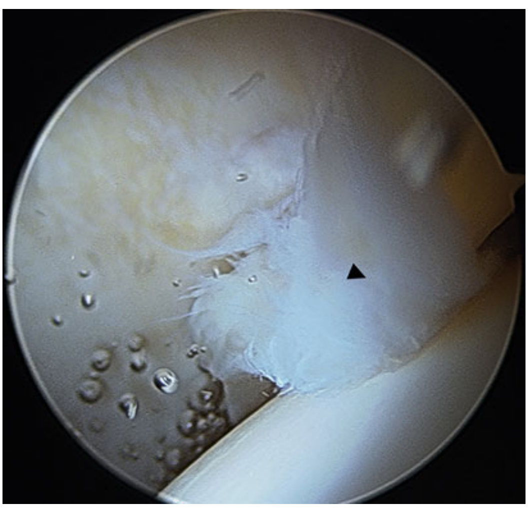


Fig. 6 Detached labral tear (*arrowhead*) obstructing visualization from the anterior portal

Capsulotomy

The strong fibrous capsule of the hip is a unique structure that provides stability, protection, and the blood supply for the hip joint. The capsule is composed of internal and external fibers. The internal fibers comprise the circular zona orbicularis, which forms a collar around the femoral neck. The external fibers run longitudinally and are made up of the iliofemoral, ischiofemoral, and pubofemoral ligaments. The anteriorly located and inverted "Y"-shaped iliofemoral ligament was found to be stiffer and more resilient to force than the ischiofemoral and pubofemoral ligaments [24]. This highlights the importance of preserving or restoring its anatomical and functional characteristics at the end of the procedure.

Capsular management during hip arthroscopy is critical to allow for better exposure without compromising hip stability, kinematics, and blood supply [25].

An anterior capsulotomy connecting the anterolateral and anterior portals can be very helpful in terms of visualization, exposure, instrument maneuverability, and safety. The interportal capsulotomy is created carefully within the plane between the labrum and femoral head. The length of the capsulotomy depends on findings in the central compartment and can be extended as posteriorly as the piriformis tendon and as anteromedially as the psoas tendon as needed. The interportal capsulotomy allows for arthroscopic visualization of the extra-articular side of the labrum, rim, and pathologic impingement lesions related to the anterior inferior iliac spine [6, 25].

There is no consensus on the optimal way to address the capsulotomy at the termination of the procedure. The capsule can either be repaired or left alone. Many hip surgeons traditionally have not repaired the capsulotomy with favorable results and no significant sequelae. Furthermore, it was suggested that when preoperative hip stiffness was encountered, the capsulotomy might even be therapeutic [6, 26]. Others believe that changing the anatomy of the capsule might result in instability, less restraint to external rotation of

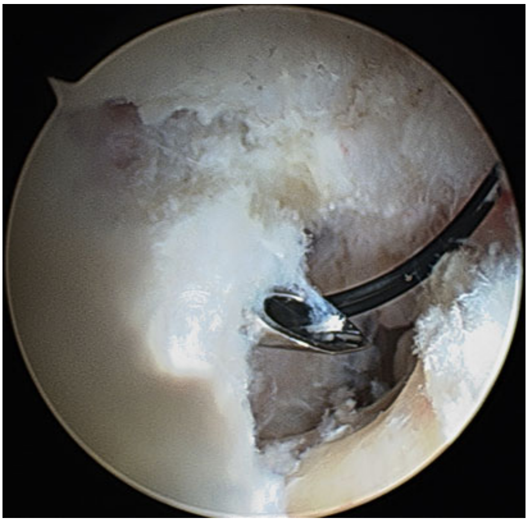


Fig. 7 Capsulotomy and repair

the hip, capsular scarring, and/or postoperative pain, particularly in patients with underlying hyperlaxity or dysplasia. Repair of the capsulotomy is done by anatomical reduction of the medial and lateral capsular flaps and repair with anatomical side-to-side stitches until a complete closure is achieved (Fig. 7).

Summary

Hip arthroscopy is becoming a more common treatment with encouraging outcomes for a wider range of pathologies within and around the hip joint. Pathologies arising from the intra-articular space of the hip joint, also referred to as the central compartment of the hip, remain the primary indications for this procedure. Access to the central compartment, as for other compartments as well, is based on proper portal positioning. Understanding the unique characteristics of the hip joint and surrounding tissues and anatomical structures is a key factor for a successful and safe procedure. Other pivotal components include proper patient selection and preoperative planning. The standard portals of the central compartment are the anterolateral (AL), anterior (AP), and posterolateral (PL) portals. The midanterior portal (MAP) is becoming a more frequently utilized portal. The AL portal is the most commonly used introductory portal for hip arthroscopy. Hip arthroscopy is considered a relatively safe procedure and surgeons should be cognizant of the possible sequelae and ways to mitigate the risk of complications.

References

1. Burman M. Arthroscopy or the direct visualization of joints. J Bone Joint Surg. 1931;4:669–95.
2. Voos JE, Rudzki JR, Shindle MK, Martin H, Kelly BT. Arthroscopic anatomy and surgical techniques for peritrochanteric space disorders in the hip. Arthroscopy. 2007;23:1246e1–1246e5.
3. Dienst M, Godde S, Seil R, Hammer D, Kohn D. Hip arthroscopy without traction: in vivo anatomy of the peripheral hip joint cavity. Arthroscopy. 2001;17:924–31.
4. Byrd JW. Hip arthroscopy by the supine approach. Instr Course Lect. 2006;55:325.
5. Glick JM. Hip arthroscopy by the lateral approach. Instr Course Lect. 2006;55:317–23.
6. Lynch TS, Terry MA, Bedi A, Kelly BT. Hip arthroscopic surgery: patient evaluation, current indications, and outcomes. Am J Sports Med. 2013;41(5):1174–89.
7. Moss SG, Schweitzer ME, Jacobson JA, Brossmann J, Lombardi JV, Dellose SM, Coralnick JR, Standiford KN, Resnick D. Hip joint fluid: detection and distribution at MR imaging and US with cadaveric correlation. Radiology. 1998;208(1):43–8.
8. Dienst M, Seil R, Kohn DM. Safe arthroscopic access to the central compartment of the hip. Arthroscopy. 2005;21(12):1510–4.
9. Dorfmann H, Boyer T. Arthroscopy of the hip: 12 years of experience. Arthroscopy. 1999;15(1):67–72.
10. Byrd JW. Avoiding the labrum in hip arthroscopy. Arthroscopy. 2000;16(7):770–3.
11. Robertson WJ, Kelly BT. The safe zone for hip arthroscopy: a cadaveric assessment of central, peripheral, and lateral compartment portal placement. Arthroscopy. 2008;24(9):1019–26.
12. Byrd JW, Pappas JN, Pedley MJ. Hip arthroscopy: an anatomic study of portal placement and relationship to the extra-articular structures. Arthroscopy. 1995;11:418–23.
13. Badylak JS, Keene JS. Do iatrogenic punctures of the labrum affect the clinical results of hip arthroscopy? Arthroscopy. 2011;27(6):761–7.
14. Aoki SK, Beckmann JT, Wylie JD. Hip arthroscopy and the anterolateral portal: avoiding labral penetration and femoral articular injuries. Arthrosc Tech. 2012;1(2):e155–60.
15. Byrd JWT. Complications associated with hip arthroscopy. In: Byrd JWT, editor. Operative hip arthroscopy. New York: Thieme; 1998. p. 171–9.

16. Byrd JWT, Jones KS. Hip arthroscopy for labral pathology: prospective analysis with 10-year follow-up. Arthroscopy. 2009;25:365–8.
17. Byrd JW. Hip arthroscopy. J Am Acad Orthop Surg. 2006;14(7):433–44.
18. Bond JL, Knutson ZA, Ebert A, Guanche CA. The 23-point arthroscopic examination of the hip: basic setup, portal placement, and surgical technique. Arthroscopy. 2009;25(4):416–29.
19. Grothaus MC, Holt M, Mekhail AO, Ebraheim NA, Yeasting RA. Lateral femoral cutaneous nerve: an anatomic study. Clin Orthop Relat Res. 2005;437:164–8.
20. Sussmann P, Zumstein M, Hahn F, Dora C. The risk of vascular injury to the femoral head when using the posterolateral arthroscopy portal: cadaveric investigation. Arthroscopy. 2007;23:1112–5.
21. Sevitt S, Thompson RG. The distribution and anastomoses of arteries supplying the head and neck of the femur. J Bone Joint Surg Br. 1965;47:560–73.
22. Gautier E, Ganz K, Krugel N, Gill T, Ganz R. Anatomy of the medial femoral circumflex artery and its surgical implications. J Bone Joint Surg Br. 2000;82:679–83.
23. Deinst M, Seil R, Kohn DM. Safe arthroscopic access to the central compartment of the hip. Arthroscopy. 2005;21(12):1510–4.
24. Hewitt JD, Glisson RR, Guilak F, Vail TP. The mechanical properties of the human hip capsule ligaments. J Arthroplasty. 2002;17(1):82–9.
25. Bedi A, Galano G, Walsh C, Kelly BT. Capsular management during hip arthroscopy: from femoroacetabular impingement to instability. Arthroscopy. 2011;27(12):1720–31.
26. Martin HD, Savage A, Braly BA, Palmer IJ, Beall DP, Kelly B. The function of the hip capsular ligaments: a quantitative report. Arthroscopy. 2008;24(2):188–95.

Hip Arthroscopy: Peripheral Compartment Access

20

Richard C. Mather III and Andrew E. Federer

Contents

Abstract

Femoroacetabular impingement (FAI) has recently been recognized as a source of hip pain and motion restrictions. Femoral morphology contributes to both cam and pincer FAI and is surgically addressed by accessing the peripheral compartment to restore normal femoral offset and sphericity. One of the more difficult aspects of hip arthroscopy is visualization and treatment of increased femoral head-neck offset and cam lesions in the peripheral compartment. The extracapsular technique explained in this chapter uses the anterior portal, anterolateral portal (ALP), and distal anterolateral access (DALA) portal. These portals allow access to the zona orbicularis and anterolateral aspect of the labrum and femoral neck while giving ergonomic access for cam lesion osteotomies. Moreover, the DALA portal offers the best condition for positioning suture anchors while allowing for an easy additional lengthwise incision of the joint capsule.

While several techniques are utilized, the purpose of this chapter is to detail our technique for exposure and 180° visualization of the femoral neck in the peripheral compartment using a T-capsulotomy.

R.C. Mather III (✉)
Department of Orthopaedic Surgery, Duke University School of Medicine, Durham, NC, USA
e-mail: chad.mather@gmail.com

A.E. Federer
Midwest Orthopaedics at Rush, Hip Preservation Center, Rush University Medical Center, Chicago, IL, USA
e-mail: afederer34@gmail.com

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_121

Introduction

Hip arthroscopy continues to be an evolving technique. Improved instrumentation and understanding of hip biomechanics have allowed orthopedic surgeons to address hip and groin pain through minimally invasive arthroscopic techniques [1–3]. However, arthroscopy of the hip joint is technically challenging – the learning curve for hip arthroscopy is much steeper than for shoulder or knee arthroscopy [4].

Femoroacetabular impingement (FAI) has recently been recognized as a source of hip pain and motion restrictions [5–7]. There have been multiple techniques, both open and arthroscopic, to treat this condition [8–11]. Additionally, FAI is now widely recognized as a precursor to early osteoarthritis [7, 12–15]. Femoral morphology contributes to both cam and pincer FAI and is surgically addressed by accessing the peripheral compartment to restore normal femoral offset and sphericity [16].

Classically, there have been two approaches to accessing the peripheral compartment, the extracapsular and intracapsular techniques, which can make use of multiple portal sites including anterolateral, anterior, posterolateral, distal anterolateral, and proximal anterolateral portals. The intracapsular technique is the traditional method that can avoid extended capsulotomies in cases of intra-articular pathology [17]. The extracapsular, or outside-in, technique described by Sampson [18] and Horisberger et al. [19] allows for better visualization of extra-articular pathology and can be employed when intracapsular access is not possible (e.g., deep overcoverage). In regard to peripheral compartment access, the definition of standard portals is difficult and of minor clinical relevance as different surgeons use a variety of approaches based on comfort and pathologic condition [20]. The extracapsular technique explained in this chapter uses the anterior portal, anterolateral portal (ALP), and distal anterolateral access (DALA) portal. According to Thorey et al. [20], the anterior, ALP, and DALA portals allow for a combined field of vision ranging from 9:00 to 7:30 o'clock with instrument accessibility of 11:00–4:30 o'clock. These portals allow access to the zona orbicularis and anterolateral aspect of the labrum and femoral neck while giving ergonomic access for cam lesion osteotomies. Moreover, the DALA portal offers the best condition for positioning suture anchors while allowing for an easy additional lengthwise incision of the joint capsule [20].

In using this extracapsular approach, the ALP is considered the safest portal with a mean distance of 54 and 42 mm from nearby structures of the lateral cutaneous femoral nerve and the superior gluteal nerve, respectively [20]. The anterior and DALA portals require a more meticulous placement with an average distance of 8 and 5 mm, respectively, from the lateral cutaneous femoral nerve [20]. Notably, lesions of the lateral femoral cutaneous nerve without complete resolution are rare and are generally not damaged with either dull or sharp trocar placement [21].

Access to the peripheral compartment begins with the transverse interportal capsulotomy. Several additional techniques can be utilized to gain further access to the femoral neck. In early hip arthroscopy, wide debridement of the capsule was utilized, but growing concerns about iatrogenic instability have made this technique less reliable. Many surgeons utilize a combination of changing the leg position and capsular retraction. An additional capsulotomy, termed a T-capsulotomy, is another technique that allows outstanding, reproducible access to the peripheral compartment if careful technique is followed. A T-capsulotomy is performed between the iliocapsularis insertion on the medial limb of the iliofemoral ligament and the gluteus minimus insertion on the lateral limb of the iliofemoral ligament. This incision allows for capsule preservation and improved visualization of the peripheral compartment superolaterally and distally [16]. Though there is no consensus in the literature, the authors choose to repair the capsulotomy as to help maintain translation and rotation as well as prevent increased external rotation [22]. Moreover, careful capsule repair can limit postoperative instability and anterior scarring [16].

One of the more difficult aspects of hip arthroscopy is visualization and treatment of increased femoral head-neck offset and cam lesions in the peripheral compartment [23–25]. While several techniques are utilized, the purpose of this chapter is to detail our technique for exposure and 180° visualization of the femoral neck in the peripheral compartment using a T-capsulotomy.

Methods

The patient is anesthetized and placed on a traction table with a well-padded perineal post. Traction is established by adduction and cantilever force over the perineal post. Image intensifier confirms appropriate joint distraction. Under fluoroscopic visualization, a standard anterolateral portal (ALP) is utilized just proximal to the greater trochanter and anterior to the iliotibial band. Needle localization is utilized to establish an anterior portal with an outside-in technique. The anterior portal is approximately 1 cm lateral to the ASIS and in line with the ALP. A capsulotomy is then performed connecting the two portals. The reflected capsule on the superior acetabular rim is released from the level of the chondrolabral junction to the anterior inferior iliac spine and indirect head of the rectus femoris insertion proximally. The central compartment is visualized and all indicated procedures are performed.

We routinely utilize a peritrochanteric distal anterolateral accessory portal (DALA) for anchor placement and later for peripheral compartment work. The arthroscope is placed in the anterolateral portal (ALP), and needle localization is utilized to establish a DALA portal aiming for the acetabular rim. This portal is in line with the ALP and approximately 4–6 cm distal.

The arthroscope is switched to the anterior portal. The light source is directed distally and laterally along the femoral neck. The traction is released and the hip is flexed approximately 30° to release tension on the anterior capsule and increase peripheral compartment volume.

A cannula is placed through the DALA portal, and a shaver and radiofrequency device along with blunt dissection are used to identify the correct interval (Fig. 1). This is a critical step – if the T-capsulotomy is performed outside of the interval between the iliocapsularis medially and the gluteus minimus laterally, access to the femoral neck will be compromised and the technique cannot be fully leveraged (Fig. 2). Often a small amount of fat is debrided to further expose the interval for both the cuts and to facilitate repair later. The starting point for the T-capsulotomy is approximately equidistant from either end of the transverse capsulotomy (Fig. 3). If one finds himself significantly closer to one end, the position

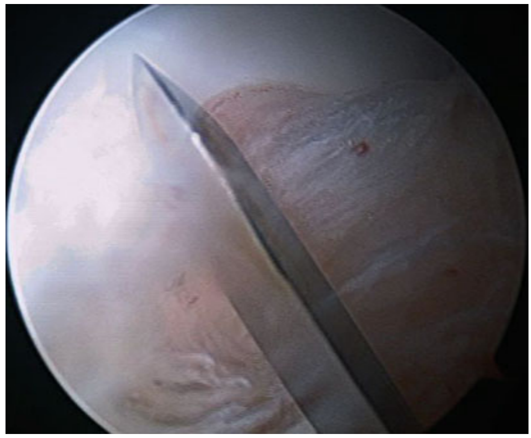


Fig. 1 T-capsulotomy perpendicular to interportal capsulotomy

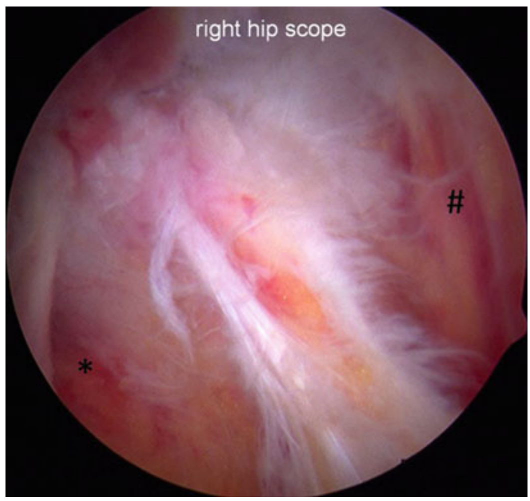


Fig. 2 Interval of the iliofemoral ligament between gluteus minimus (*) and iliocapsularis (#)

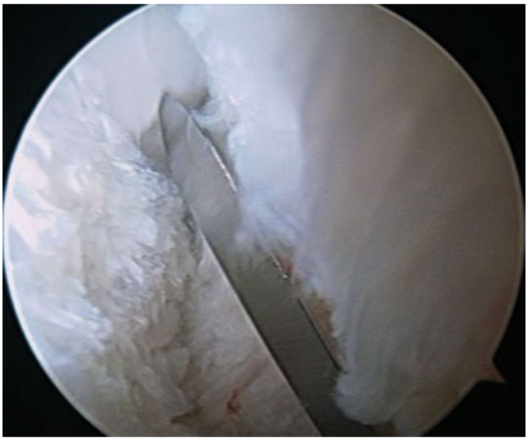


Fig. 3 Vertical incision through the iliofemoral ligament (IFL)

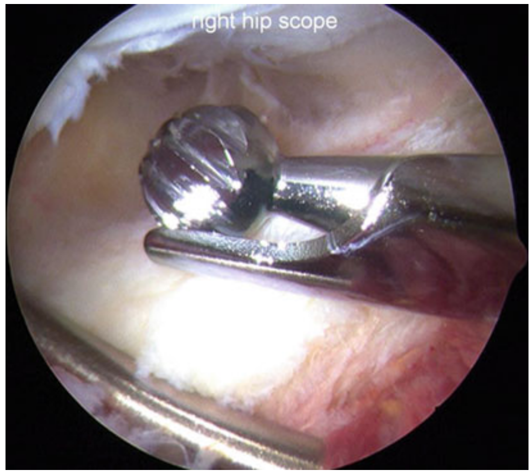


Fig. 5 Switching stick through the anterolateral portal (ALP) to retract the lateral leaflet of the iliofemoral ligament (IFL) and burr through the DALA portal

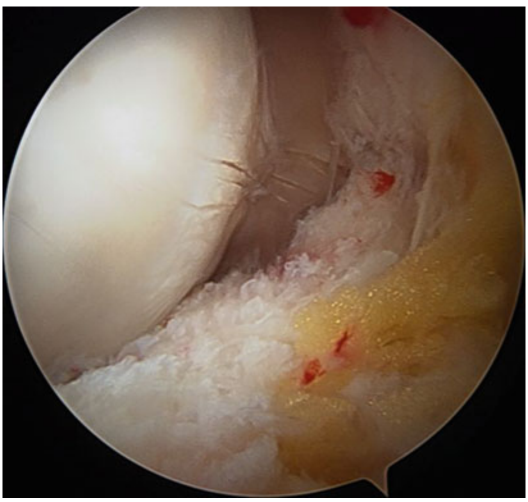


Fig. 4 Completed T-capsulotomy to expose the cam deformity

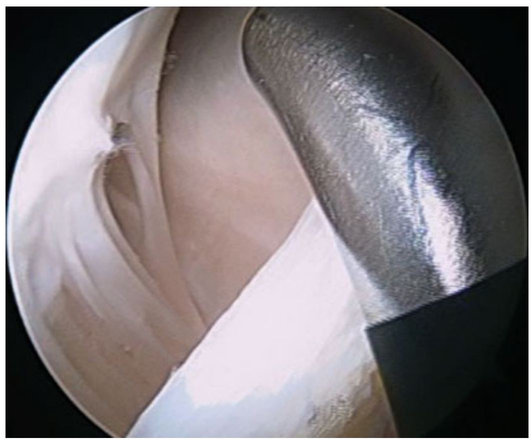


Fig. 6 Retraction of the medial leaflet of the iliofemoral ligament (IFL) to expose the medial synovial fold

should be rechecked before the cut is made. Next, an arthroscopic beaver blade is introduced through the DALA portal. The capsule is then cut from the femoral head-neck junction to the intertrochanteric line through the zona orbicularis (Fig. 4). The capsular cut should be made along the center of the femoral neck. Skiving the blade in either direction can lead to injury to the femoral vessels.

Once the capsulotomy is complete, a switching stick is introduced in the ALP (Fig. 5). The switching stick is used as a retractor of the superior and inferior leaflets of the capsule to improve visualization of the cam lesion (Fig. 6). Fluoroscopic images taken at a 45° angle to obtain a Dunn lateral view of the hip are frequently utilized to assess the extent of the cam lesion and retractor placement. The arthroscopic burr is inserted through the DALA portal for cam resection in a more ergonomic position (Fig. 7).

Improved exposure superior and inferior can be obtained with repositioning of the switching stick either above or below the femoral neck

Fig. 7 Cam lesion preparation

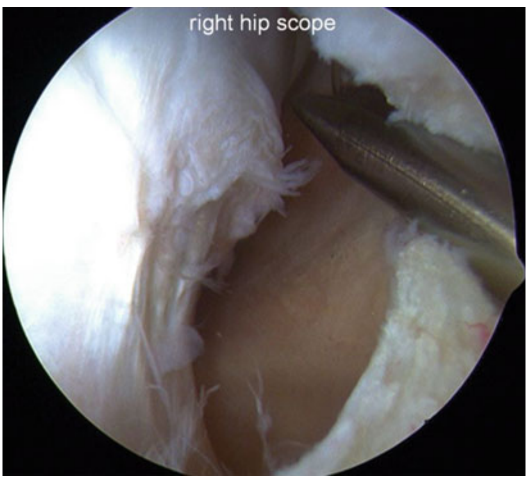


Fig. 8 Retraction of the medial leaflet to expose the anteromedial cam

(Figs. 8 and 9). Increasing hip flexion angle to 45° allows improved access to the distal femoral neck for larger cam lesions and visualization of the entire osteochondroplasty. Rotation of the hip in conjunction with fluoroscopic images helps improve access to anterior and posterior extents of the cam lesion (Fig. 10). This technique provides at least 180° visualization of the cam lesion and osteochondroplasty without extensive capsulectomy (Table 1).

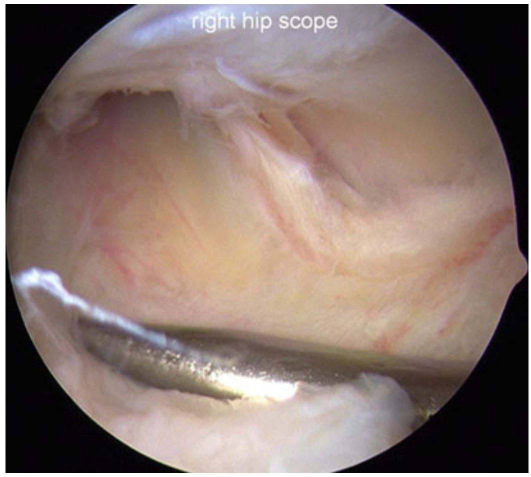


Fig. 9 Retraction of the lateral leaflet to expose posterolateral cam

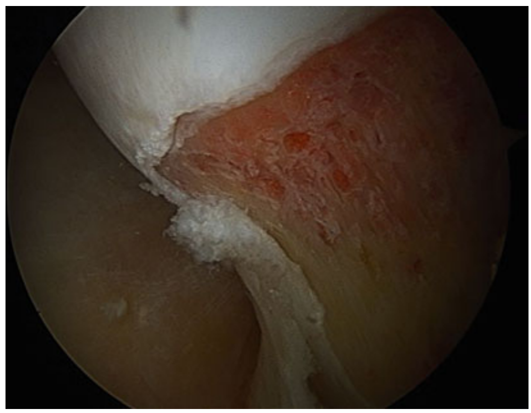


Fig. 10 View showing lateral extent of cam lesion

An alternative method of retracting the capsule is to place retraction suture in each leaflet (Fig. 11). The authors' preferred technique for this is to place one suture in the medial limb near the proximal corner and to retrieve it through the DALA portal. Two sutures are placed in the lateral limb with one in the proximal corner and one at the base. These stitches are retrieved through the ALP. The sutures are then snapped with tension against the skin. The sutures are placed typically through a cannula in the DALA portal but can also be placed through the ALP. The sutures can also be used for repair after completion of the femoral osteochondroplasty. This technique allows access

Table 1 Technical pearls (Reprinted from Suslak et al. [26] with permission from Elsevier)

Interportal capsulotomy	Provides adequate visualization and mobilization in the central compartment
T-capsulotomy	Provides adequate visualization to fully evaluate the proximal femur
Arthroscope in anterior portal	Directing the light source down the femoral neck allows at least 180° visualization of the cam lesion
Retraction	(1) Switching stick through the anterolateral portal to retract the medial and lateral leaflets of the capsule for improved exposure
	(2) Traction sutures in each limb
DALA portal	Allows ergonomic osteochondroplasty of the femoral head-neck junction

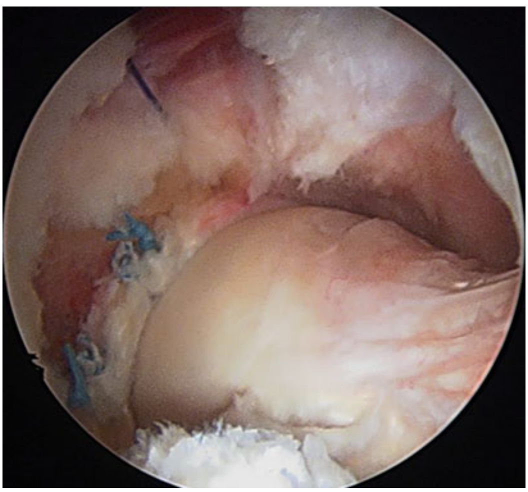


Fig. 11 Capsular suspension by placing retraction suture in each leaflet

to approximately 90 % of most cam lesions and requires no additional surgical assistance.

After the femoral head-neck osteochondroplasty is complete (Fig. 12), a dynamic arthroscopic exam of the hip is performed (Fig. 13). The hip is flexed to 60° and rotated internally and externally, ensuring there is no impingement on the labrum, liftoff, or subluxation of the femoral head. Also the proximal extent of the cam resection should not violate the suction seal of the hip joint. Fluoroscopic images are used to assess

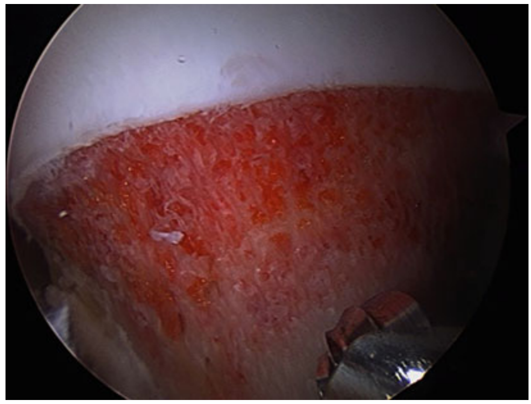


Fig. 12 Completed femoral osteochondroplasty

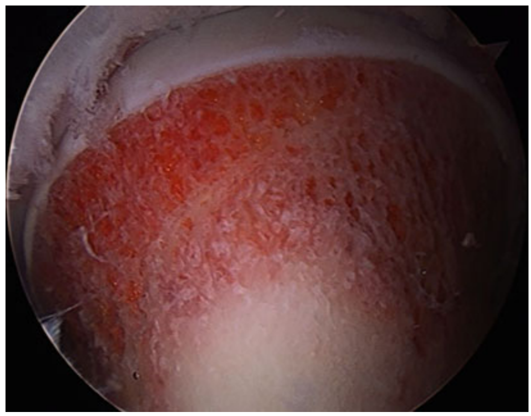


Fig. 13 Dynamic examination after completed femoral osteochondroplasty

anatomic recontouring of the femoral head-neck junction.

We routinely repair the capsule after the capsulotomy. This is done with the arthroscope in the anterior portal, utilizing the same visualization as for cam osteochondroplasty. A cannula is introduced in the DALA portal. A commercially available suture-passing device designed for closure of the hip capsule allows for a single portal technique. It is introduced through the cannula, and a suture is passed through the medial and lateral capsule leaflets starting distally and working proximally. The knot is tied on the outside of the capsule through the DALA portal (Fig. 14). We have found that it typically takes three to four passes to adequately close the capsule to the level

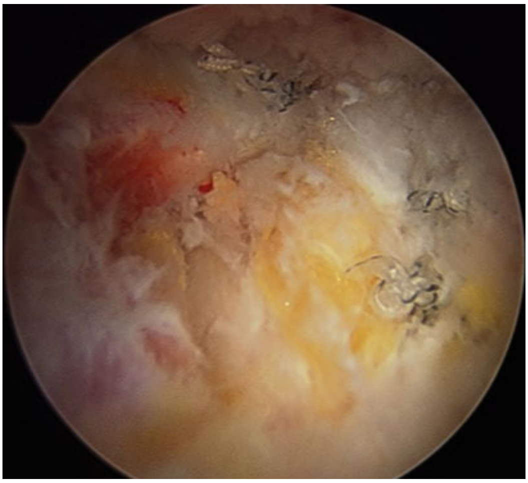


Fig. 14 Complete closure of the iliofemoral ligament (IFL)

of the interportal capsulotomy and cover the femoral osteochondroplasty. The authors' currently prefer to close the transverse capsulotomy. This is typically done through the ALP with two simple stitches. An alternative technique for capsular closure is to utilize a suture lasso technique. A suture passer is utilized through the DALA portal. The suture can be passed through the ALP and then tied through the DALA portal.

Summary

Hip arthroscopy continues to be an evolving technique. Improved instrumentation and understanding of hip biomechanics have allowed orthopedic surgeons to address hip and groin pain through minimally invasive arthroscopic techniques [1–3]. One of the more difficult aspects of hip arthroscopy is visualization and treatment of increased femoral head-neck offset and cam lesions in the peripheral compartment [23–25]. While several techniques are utilized, our technique using the anterior portal as the viewing portal and DALA portal as the working portal in conjunction with a T-capsulotomy allows for dynamic exposure and 180° visualization of the femoral neck in the peripheral compartment.

References

1. Stevens MS, Legay DA, Glazebrook MA, Amirault D. The evidence for hip arthroscopy: grading the current indications. Arthroscopy. 2010;26(10):1370–83.
2. McCarthy JC, Lee JA. Hip arthroscopy: indications, outcomes, and complications. Instr Course Lect. 2006;55:301–8.
3. Byrd JW, Jones KS. Hip arthroscopy in athletes: 10-year follow-up. Am J Sports Med. 2009;37(11):2140–3.
4. Konan S, Rhee SJ, Haddad FS. Hip arthroscopy: analysis of a single surgeon's learning experience. J Bone Joint Surg Am. 2011;93 Suppl 2:52–6.
5. Ito K, Minka 2nd MA, Leunig M, Werlen S, Ganz R. Femoroacetabular impingement and the cam-effect. A MRI-based quantitative anatomical study of the femoral head-neck offset. J Bone Joint Surg Br. 2001;83(2):171–6.
6. Murphy S, Tannast M, Kim YJ, Buly R, Millis MB. Debridement of the adult hip for femoroacetabular impingement: indications and preliminary clinical results. Clin Orthop Relat Res. 2004;429(429):178–81.
7. Lavigne M, Parvizi J, Beck M, Siebenrock KA, Ganz R, Leunig M. Anterior femoroacetabular impingement: part I. techniques of joint preserving surgery. Clin Orthop Relat Res. 2004;418(418):61–6.
8. Byrd JW, Jones KS. Arthroscopic management of femoroacetabular impingement: minimum 2-year follow-up. Arthroscopy. 2011;27(10):1379–88.
9. Fabricant PD, Heyworth BE, Kelly BT. Hip arthroscopy improves symptoms associated with FAI in selected adolescent athletes. Clin Orthop Relat Res. 2012;470(1):261–9.
10. Bedi A, Zaltz I, De La Torre K, Kelly BT. Radiographic comparison of surgical hip dislocation and hip arthroscopy for treatment of cam deformity in femoroacetabular impingement. Am J Sports Med. 2011;39(Suppl):20S–8.
11. Byrd JW, Jones KS. Arthroscopic management of femoroacetabular impingement in athletes. Am J Sports Med. 2011;39(Suppl):7S–13.
12. Bedi A, Dolan M, Hetsroni I, et al. Surgical treatment of femoroacetabular impingement improves hip kinematics: a computer-assisted model. Am J Sports Med. 2011;39(Suppl):43S–9.
13. Larson CM, Giveans MR, Taylor M. Does arthroscopic FAI correction improve function with radiographic arthritis? Clin Orthop Relat Res. 2011;469(6):1667–76.
14. Barros HJ, Camanho GL, Bernabe AC, Rodrigues MB, Leme LE. Femoral head-neck junction deformity is related to osteoarthritis of the hip. Clin Orthop Relat Res. 2010;468(7):1920–5.
15. Ng VY, Arora N, Best TM, Pan X, Ellis TJ. Efficacy of surgery for femoroacetabular impingement: a systematic review. Am J Sports Med. 2010;38(11):2337–45.

16. Lynch TS, Terry MA, Bedi A, Kelly BT. Hip arthroscopic surgery: patient evaluation, current indications, and outcomes. Am J Sports Med. 2013;41(5):1174–89.
17. Gutierrez RC, Iban MA, Ayestaran AC, Sobrino AS, Oteo-Alvaro A. Peripheral compartment as the initial access for hip arthroscopy in complex cases: technical note. Orthopedics. 2013;36(6):456–62.
18. Sampson TG. Arthroscopic treatment of femoroacetabular impingement. Am J Orthop (Belle Mead NJ). 2008;37(12):608–12.
19. Horisberger M, Brunner A, Herzog RF. Arthroscopic treatment of femoroacetabular impingement of the hip: a new technique to access the joint. Clin Orthop Relat Res. 2010;468(1):182–90.
20. Thorey F, Ezechieli M, Ettinger M, Albrecht UV, Budde S. Access to the hip joint from standard arthroscopic portals: a cadaveric study. Arthroscopy. 2013;29 (8):1297–307.
21. Dienst M, Grun U. Complications of hip arthroscopies. Orthopade. 2008;37(11):1108–9, 1111–5.
22. Myers CA, Register BC, Lertwanich P, et al. Role of the acetabular labrum and the iliofemoral ligament in hip stability: an in vitro biplane fluoroscopy study. Am J Sports Med. 2011;39(Suppl): 85S–91S.
23. Ejnisman L, Philippon MJ, Lertwanich P. Femoroacetabular impingement: the femoral side. Clin Sports Med. 2011;30(2):369–77.
24. Vaughn ZD, Safran MR. Arthroscopic femoral osteoplasty/chielectomy for cam-type femoroacetabular impingement in the athlete. Sports Med Arthrosc. 2010;18(2):90–9.
25. Matsuda DK. The case for cam surveillance: the arthroscopic detection of cam femoroacetabular impingement missed on preoperative imaging and its significance. Arthroscopy. 2011;27(6):870–6.
26. Suslak AG, Mather 3rd RC, Kelly BT, Nho SJ. Improved arthroscopic visualization of peripheral compartment. Arthrosc Tech. 2012;1(1):e57–62.

Hip Arthroscopy: Peritrochanteric Space Access

21

James T. Beckmann, Travis G. Maak, and Stephen K. Aoki

Contents

Abstract

Endoscopic techniques accessing the peritrochanteric space have enabled more effective identification and treatment of disorders including external coxa saltans, trochanteric bursitis, and gluteus medius and minimus tears when conservative treatment has failed. A thorough understanding of the anatomy of the hip and proper placement of portals facilitates access to the peritrochanteric compartment and procedural ease. Outcome-based studies and improved surgical instrumentation will continue to allow more effective management of extra-articular hip pain.

Introduction

Hip arthroscopy is used increasingly to treat disorders about the hip where hip-preserving techniques are indicated. Enthusiasm for arthroscopic and endoscopic techniques is reflected by a nearly twentyfold increase in the number of hip arthroscopy procedures performed for the American Board of Orthopaedic Surgery examination during the 10-year period from 1999 to 2009 [1]. Hip arthroscopy, in conjunction with advances in magnetic resonance imaging of the hip, has increased understanding of both intra-articular and extra-articular hip pathologies. Structural anatomy and function have been described in detail by Bryd [2, 3] and Voos [4], allowing surgeons to broaden arthroscopic applications. Many

J.T. Beckmann • T.G. Maak • S.K. Aoki (✉)
Department of Orthopaedics, University of Utah Health Care, Salt Lake City, UT, USA
e-mail: james.beckmann@hsc.utah.edu; travis.maak@gmail.com; stephen.aoki@hsc.utah.edu

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_21

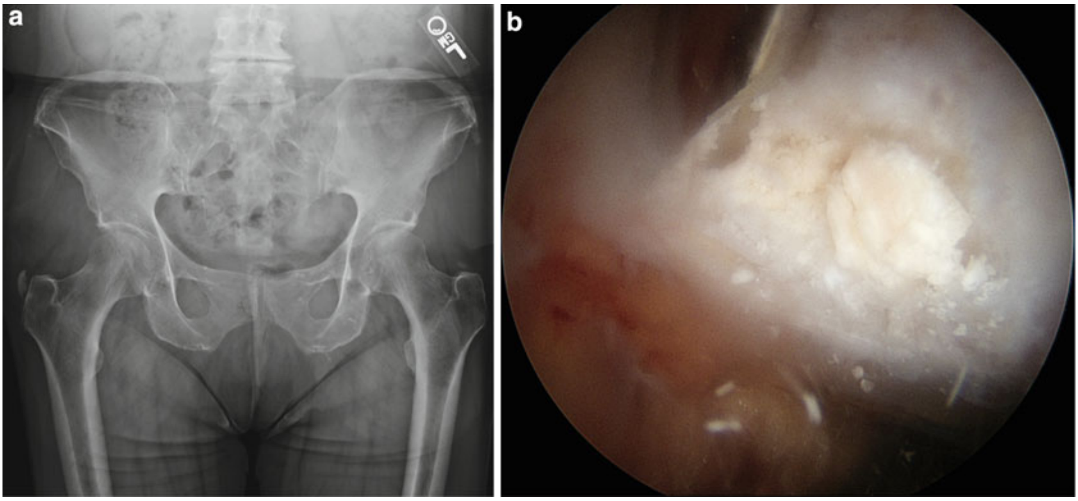


Fig. 1 (**a**, **b**) AP pelvis radiograph with bilateral gluteus medius calcific tendonitis. Corresponding arthroscopic view demonstrating evacuation of the calcific deposit

disorders previously performed through open procedures can be addressed in a minimally invasive fashion with more recent arthroscopic techniques.

Arthroscopic techniques originally designed to address intra-articular pathology have been expanded to include soft tissue pathology about the hip. In particular, endoscopy of the peritrochanteric compartment has evolved to treat greater trochanteric pain syndrome (GTPS). Disorders included in GTPS include (1) greater trochanteric bursitis, (2) external snapping hip syndrome, and (3) gluteus medius and minimus tendinopathies. Initially, the preferred treatment modality for GTPS is nonoperative therapy consisting of corticosteroid and anesthetic injections [5] combined with a structured physical therapy regimen. Surgery is reserved only for patients who fail an extensive course of conservative treatment [6–8]. Open surgical techniques have been described for disorders of the peritrochanteric space prior to the widespread use of hip arthroscopy but varied in efficacy and have been associated with significant postoperative morbidity.

Recent advances in hip arthroscopy have led to surgical treatment descriptions for many of the peritrochanteric compartment disorders. Recalcitrant trochanteric bursitis can be managed with arthroscopic trochanteric bursectomy [5, 9]. Arthroscopic release of the iliotibial band can effectively treat external coxa saltans [10]. Calcific tendonitis within the gluteus medius and minimus tendons can be removed endoscopically [11] (Fig. 1a, b). Repair of gluteus medius and minimus tears, which may cause much of the pain previously associated with refractory trochanteric bursitis, has also been reported [12]. These advances have increased the understanding of hip pain and have provided surgeons with more effective tools to address extra-articular hip pathology.

This chapter describes the evaluation of the lateral aspect of the hip, the peritrochanteric space anatomy, the associated pathologic entities, and the endoscopic techniques used to effectively address them.

Anatomy of the Peritrochanteric Compartment

An understanding of the relevant anatomy of the trochanteric space is requisite for treating associated pathology. The peritrochanteric compartment is an anatomic region of the lateral aspect of the hip between the borders of the greater trochanter

Fig. 2 Coronal T1-weighted MRI image showing the normal gluteus minimus tendon (*arrows*) inserting on the anterior facet of the greater trochanter

Fig. 3 Coronal T1-weighted MR image in a patient with a normal gluteus medius tendon. The lateral part of the gluteus medius tendon (*arrow*) inserts at the lateral facet of the greater trochanter. The *open arrowhead points* to the trochanteric bald spot

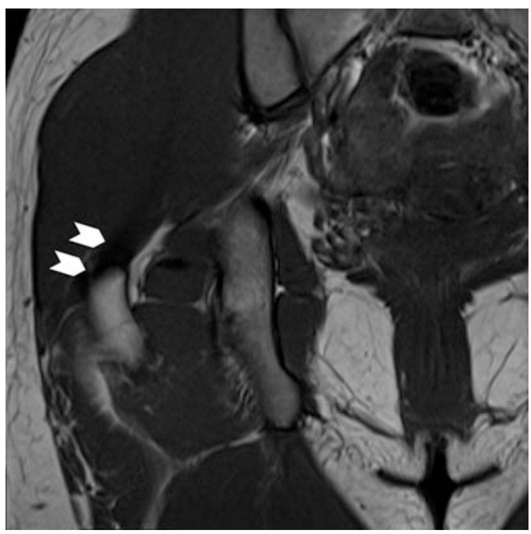


Fig. 4 The main portion of the normal gluteus medius tendon (*arrow heads*) inserts on the posterior superior facet of the greater trochanter

and iliotibial band (ITB) comprising the trochanteric bursa. Anteriorly, the compartment is bordered by the proximal sartorius and tensor fascia latae musculature. Medially, its limits are defined by the gluteus medius and minimus superiorly, extending distally past the vastus ridge to include the vastus lateralis. The lateral border is comprised of the fibers of the iliotibial band, and the peritrochanteric compartment terminates distally at the level of the gluteal sling insertion.

The greater trochanter has four distinct facets consisting of the anterior facet, superoposterior facet, lateral facet, and posterior facet. The gluteus minimus can be divided into a lateral and medial portion. The lateral portion arises from the superficial muscle belly and contains the main tendinous insertion on the anterior facet (Fig. 2), while the medial portion inserts on the anterior and superior capsules of the hip joint. The gluteus medius is comprised of an anterior, lateral, and posterior portion. The strong posterior portion inserts on the posterosuperior facet, while the lateral portion inserts distally on the lateral facet (Figs. 3 and 4). The anterior portion is mainly muscular and joins the gluteus minimus tendon interiorly [13].

Three distinct bursas are consistently present in most individuals, although bursa anatomy can be variable. The trochanteric bursa encompasses the posterior facet of the greater trochanter and is typically the largest bursa encountered. The subgluteus medius bursa lies between the superior portion of the lateral facet and the lateral portion of the gluteus medius tendon. The subgluteus minimus bursa lies beneath the gluteus minimus tendon medial and superior to its insertion [14].

Endoscopic Techniques

There is certainly an improved understanding of hip pathology given the advancements in hip imaging and surgical techniques. Specialized arthroscopic hip instrumentation has enabled surgeons to access the hip and perform procedures that were once only possible with open techniques. Voos et al. [12] described disorders of the peritrochanteric hip and an endoscopic technique to address this extra-articular compartment.

In most individuals, the intra-articular compartment is first inspected to assess for concomitant hip pathology prior to examining the peritrochanteric space. With this completed, attention is directed to the extra-articular spaces. It is important to understand the anatomy of the peritrochanteric compartment. The borders consist of the iliotibial band and tensor fascia lata laterally, vastus lateralis inferomedially, abductor tendons superomedially, gluteus maximus muscle superiorly, and gluteus maximus tendon insertion on the linea aspera posteriorly. Within this space, the trochanteric bursa can be found.

A consistent, routine endoscopic evaluation assures that all pathology is addressed. Examination of the space is generally performed through three portals, although portal placement for the peritrochanteric space can vary among surgeons.

Once the procedures in the central and peripheral compartments are completed, the leg is positioned in full extension, held in 0° of adduction and 10–15° of internal rotation. The first portal is the mid anterior (MA) portal, which is used to first establish access to the peritrochanteric space (Fig. 5). The portal is placed 1 cm lateral to a line extending distally from the anterior superior iliac spine (ASIS) in the soft spot interval between the tensor fascia latae and sartorius. The cannula is directed into the peritrochanteric space with the

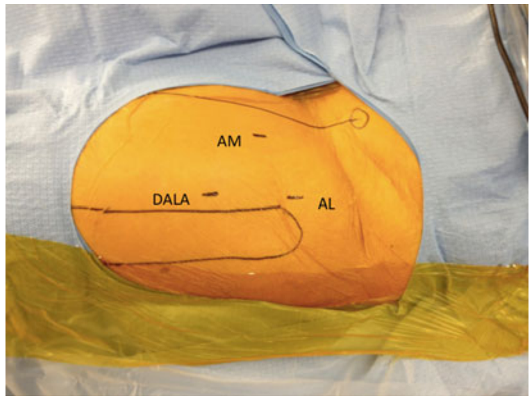


Fig. 5 Portals for peritrochanteric space access: *AL* antero lateral, *MA* mid-anterior, *DALA* distal antero lateral accessory

tip directed toward the vastus ridge and the cannula swept between the iliotibial band and the greater trochanter once in the peritrochanteric space. This technique is analogous to accessing the subacromial space in the shoulder where the iliotibial band is analogous to the undersurface of the acromion. When the portal is placed appropriately, a clear space lying between the iliotibial band and the greater trochanter can be easily identified. If in doubt, the cannula should be placed directly lateral to the greater trochanter and confirmed on fluoroscopy if needed (Fig. 6). A second, anterolateral (AL) portal is utilized just proximal to the tip of the greater trochanter. This portal is similar to the standard anterolateral portal established for central compartment visualization. This is utilized as a proximal working portal and can also be utilized to visualize distally. A third, distal anterolateral accessory (DALA) portal is used for diagnostic evaluation and operative intervention. The distal anterolateral portal is placed in line with the proximal anterolateral portal. If standard portals do not allow clear access to the peritrochanteric space, further portals can be established for visualization and instrumentation. Spinal needle localization can assist in obtaining the correct positioning of these portals. Oftentimes, these three portals are the same portals utilized for intra-articular compartment access.

The 70-degree scope is first placed into the MA portal. Identifying the borders of the peritrochanteric space is important in properly

Fig. 6 Fluoroscopic confirmation of cannula placement into the peritrochanteric space, just lateral to the greater trochanter

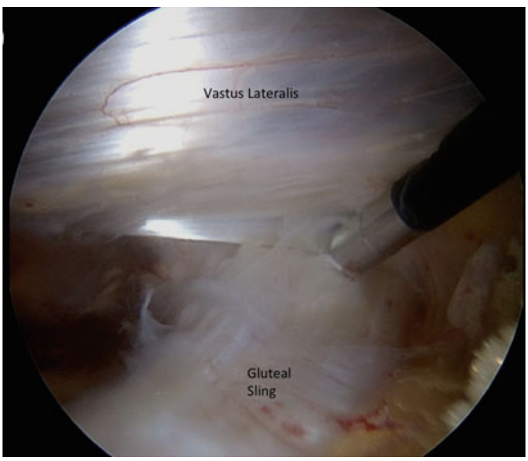


Fig. 7 Inferior border of the greater trochanteric space: fibers of the vastus lateralis and gluteal sling

assessing this compartment. An important anatomic landmark is the gluteus maximus insertion ("gluteal sling") onto the linea aspera (Fig. 7). Inspection should begin with identification of the gluteal sling, as this will assist in providing a landmark to confirm placement into the peritrochanteric space. In order to view the distal structures, the light source and camera are oriented distally. The distal anterolateral portal is

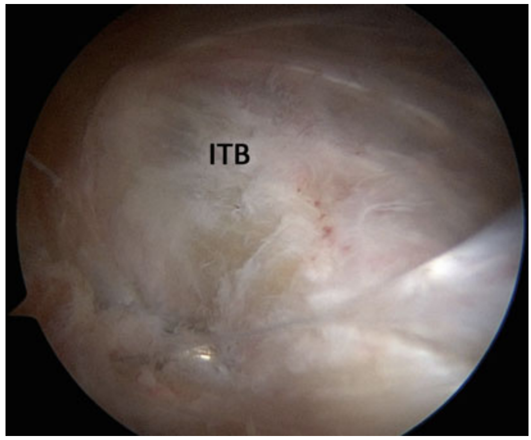


Fig. 8 IT band undersurface wear associated with a full-thickness myotendinous gluteus tear

then utilized as the working portal. Hemostasis can be obtained with radiofrequency ablation. Sometimes fibrinous bands in this area will require excision in order to begin identifying anatomic landmarks.

Just anterior to the gluteus maximus insertion, the longitudinal lines of the vastus lateralis can be seen as the scope camera is advanced in a circular fashion (Fig. 7). These fibers can be traced proximally up to the insertion at the vastus tubercle. The gluteus minimus musculotendinous complex attachment onto the anterior facet of the greater trochanter is visualized with the camera placed laterally looking anterosuperior. As the scope is directed superiorly, the gluteus medius will come into view with its insertion onto the lateral greater trochanter. It should be thoroughly probed and visualized to identify the presence of full-thickness tendon insertion tears. Further cleaning of the trochanteric bursal fibrinous bands may be required for better access and visualization. Use of electrocautery or an arthroscopic shaver is useful to remove this tissue. A non-toothed shaver is safer in this region as it is more protective against iatrogenic injury to gluteal and tensor fascia lata muscle tissue in the proximal space. Finally, the scope should be turned toward the iliotibial band. In particular, the posterior one third of the iliotibial band is implicated in coxa saltans externus and may be causing direct abrasive wear to the greater trochanter (Fig. 8).

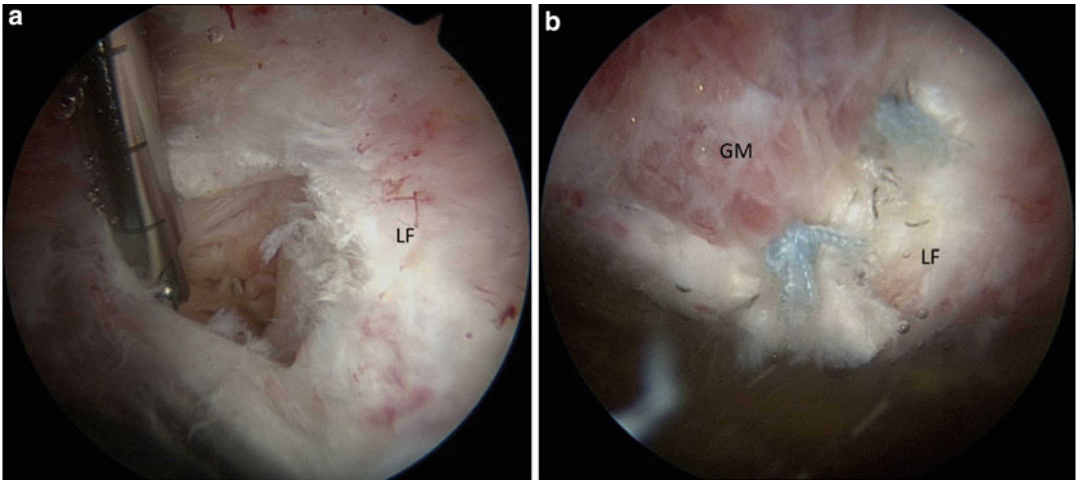


Fig. 9 (**a**) Coronal T2-weighted MRI image of a 52-year-old woman demonstrating a focal full-thickness tear of the gluteus medius tendon. (**b**) Endoscopic image of gluteus medius tear as visualized from the peritrochanteric compartment. Fibers are disrupted from their insertion on the lateral facet (*LF*) of the greater trochanter. (**b**) Endoscopic repair of the gluteus medius (*GM*) tendon using two double-loaded suture anchors

If painful snapping of the iliotibial band has been refractory to nonoperative treatment, a release may be required with the use of an electrocautery device or beaver blade. The release should be performed along the posterolateral portion of the iliotibial band beginning at the vastus tubercle insertion extending to the tip of the greater trochanter. The goal of surgical treatment should be to relax the fibers that are under the greatest amount of tension. Various techniques have been described in the literature including z-plasties, diamond-shaped resections, and step cuts [15, 16]. Polesello et al. also describe releasing the gluteal sling at the linea aspera as a treatment option for external snapping hip [17].

Patients who fail conservative treatment and have evidence of an abductor tendon tear on MRI may benefit from surgical management. Adequate visualization of the gluteus attachment sites may require resection of overlying fibrinous bands. When a repairable tear is identified, the edges are debrided and prepared for repair (Fig. 9a). The attachment site of the tendon at the greater trochanter is prepared with a full-radius shaver. Suture anchors can be placed into the footprint of the abductor tendons in a standard arthroscopic fashion (Fig. 9b). Fluoroscopic guidance may be helpful in directing the anchors in the appropriate direction and location. Once the anchors are placed, the sutures are retrieved and passed sequentially through the edges of the prepared gluteus tendon with a suture-passing device and tied under arthroscopic visualization.

Summary

Advancement in arthroscopic techniques has now enabled more effective identification and treatment of peritrochanteric compartment disorders including external coxa saltans, trochanteric bursitis, and gluteus medius and minimus tears when conservative treatment has failed. A thorough understanding of arthroscopic anatomy of the hip and proper placement of portals is crucial for surgical ease and outcome success. Continued improvements through clinical studies and instrumentation will expand the viable treatment options and allow more effective management of extra-articular hip pain.

References

1. Colvin AC, Harrast J, Harner C. Trends in hip arthroscopy. J Bone Jt Surg Am Vol. 2012;94(4):e23. PubMed PMID: 22336982.

2. Byrd JW. Hip arthroscopy. J Am Acad Orthop Surg. 2006;14(7):433–44. PubMed PMID: 16822891.
3. Byrd JW. Hip arthroscopy: surgical indications. Arthroscopy. 2006;22(12):1260–2. PubMed PMID: 17165215.
4. Voos JE, Rudzki JR, Shindle MK, Martin H, Kelly BT. Arthroscopic anatomy and surgical techniques for peritrochanteric space disorders in the hip. Arthroscopy. 2007;23(11):1246 e1–5. PubMed PMID: 17986418. Epub 2007/11/08. eng.
5. Fox JL. The role of arthroscopic bursectomy in the treatment of trochanteric bursitis. Arthroscopy. 2002;18(7):E34. PubMed PMID: 12209419.
6. Govaert LH, van der Vis HM, Marti RK, Albers GH. Trochanteric reduction osteotomy as a treatment for refractory trochanteric bursitis. J Bone Jt Surg Br. 2003;85(2):199–203. PubMed PMID: 12678352.
7. Zoltan DJ, Clancy Jr WG, Keene JS. A new operative approach to snapping hip and refractory trochanteric bursitis in athletes. Am J Sports Med. 1986;14 (3):201–4. PubMed PMID: 3752359.
8. Brooker Jr AF. The surgical approach to refractory trochanteric bursitis. Johns Hopkins Med J. 1979;145 (3):98–100. PubMed PMID: 470296.
9. Wiese M, Rubenthaler F, Willburger RE, Fennes S, Haaker R. Early results of endoscopic trochanter bursectomy. Int Orthop. 2004;28(4):218–21. PubMed PMID: 15197493.
10. Ilizaliturri Jr VM, Villalobos Jr FE, Chaidez PA, Valero FS, Aguilera JM. Internal snapping hip syndrome: treatment by endoscopic release of the iliopsoas tendon. Arthroscopy. 2005;21(11):1375–80. PubMed PMID: 16325091.
11. Kandemir U, Bharam S, Philippon MJ, Fu FH. Endoscopic treatment of calcific tendinitis of gluteus medius and minimus. Arthroscopy. 2003;19(1): E4. PubMed PMID: 12522397.
12. Voos JE, Rudzki JR, Shindle MK, Martin HM, Kelly BT. Arthroscopic anatomy and surgical techniques for peritrochanteric space disorders in the hip. Arthroscopy. In Press 2007.
13. Robertson WJ, Gardner MJ, Barker JU, Boraiah S, Lorich DG, Kelly BT. Anatomy and dimensions of the gluteus medius tendon insertion. Arthroscopy. 2008;24(2):130–6. PubMed PMID: 18237695. Epub 2008/02/02. eng.
14. Hoffmann A, Pfirrmann CW. The hip abductors at MR imaging. Eur J Radiol. 2012;81(12):3755–62. PubMed PMID: 20399581. Epub 2010/04/20. eng.
15. White RA, Hughes MS, Burd T, Hamann J, Allen WC. A new operative approach in the correction of external coxa saltans: the snapping hip. Am J Sports Med. 2004;32(6):1504–8. PubMed PMID: 15310578.
16. Ilizaliturri Jr VM, Camacho-Galindo J. Endoscopic treatment of snapping hips, iliotibial band, and iliopsoas tendon. Sports Med Arthrosc Rev. 2010;18 (2):120–7. PubMed PMID: 20473131.
17. Polesello GC, Queiroz MC, Domb BG, Ono NK, Honda EK. Surgical technique: endoscopic gluteus maximus tendon release for external snapping hip syndrome. Clin Orthop Relat Res. 2013;471(8):2471–6. PubMed PMID: 23086213. Pubmed Central PMCID: 3705038.

Hip Arthroscopy Techniques: Deep Gluteal Space Access

22

Carlos A. Guanche

Contents

Abstract

With the expansion of endoscopically exploring various areas around the hip have come new areas to define. The area posterior to the hip joint, known as the subgluteal space or deep gluteal space (DGS), is one such area. This chapter will summarize the relevant anatomy and pathology commonly found in the DGS. It is hoped that this will allow the reader to further explore the area and treat the appropriate pathological areas.

Introduction

With the increasing abilities gained in exploring various areas endoscopically has come an expansion of what can be explored. The area posterior to the hip joint, known as the subgluteal space or deep gluteal space (DGS), is one such area. It has been known for many years that there is a significant cohort of patients that have persistent posterior hip and buttocks pain, whose treatment has been very difficult. Part of the difficulties have stemmed from poor understanding of the anatomy and pathology of this area. With endoscopic exploration of DGS, orthopedic surgeons have been able to visualize the pathoanatomy and, therefore, have a better understanding of the pathologies in a part of the body that has been historically ignored.

The complexity of the area makes diagnosis difficult, as there are osseous, vascular, neural, and muscular elements to the pathological

C.A. Guanche
Southern California Orthopedic Institute, Van Nuys, CA, USA
e-mail: cguanche@scoi.com

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_22

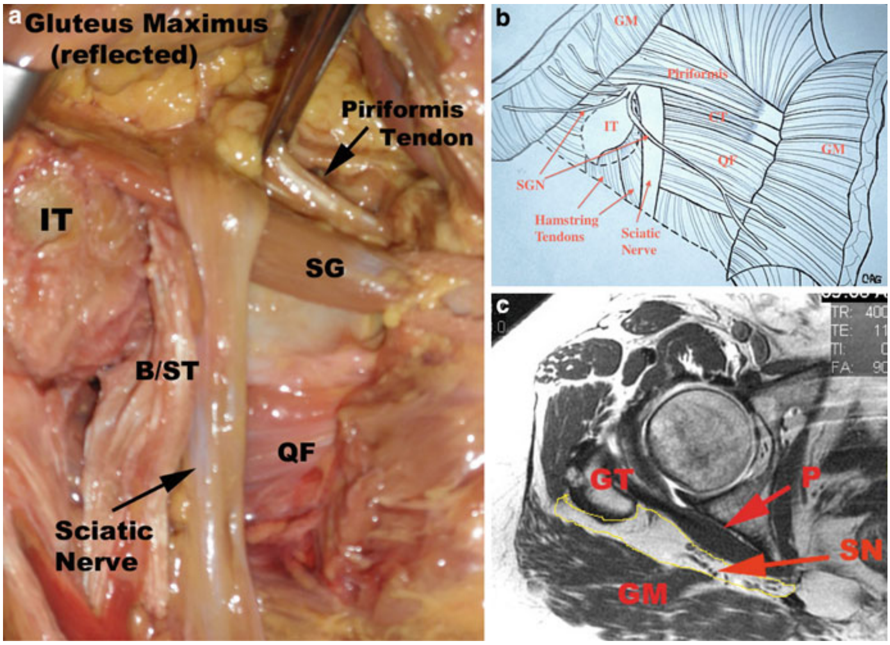


Fig. 1 The borders of the subgluteal space. (**a**) Cadaveric dissection of a right subgluteal space, visualized from posterior. Note the entrance of the sciatic nerve just below the piriformis tendon and lying on the common tendon (*B/ST* common tendon of biceps and semitendinosus that has been partially detached and slightly everted, *IT* ischial tuberosity, *QF* quadratus femoris). (**b**) A diagrammatic representation of a right subgluteal space (*CT* conjoint tendon of the superior and inferior gemelli, along with the obturator internus, *GM* gluteus maximus muscle, everted, *IT* ischial tuberosity, *QF* quadratus femoris). (**c**) An axial, T1-weighted image of a right hip. The *yellow line outlines* the borders of the space (*SN* sciatic nerve, *P* piriformis muscle, *GT* greater trochanter, *GM* gluteus maximus)

processes in the space. The anatomy is intricate, and the pathological processes are poorly understood, with only small case series available in the literature [1, 2]. The primary goal of this chapter is to serve as a baseline description for access and exploration of the deep gluteal space. Some of the common entities encountered will also be briefly discussed.

Anatomy of Deep Gluteal or Subgluteal Space

The DGS is the posterior extension of the peritrochanteric space and is largely the potential space deep to the gluteus maximus muscle. More specifically, the posterior border of the space is the anterior surface of the gluteus maximus with the distal margin beginning inferiorly at the femoral insertion site of the gluteus maximus tendon on the linea aspera and proximal margin at the origin of the gluteus maximus on the iliac crest. Anteriorly, the space is bordered by the sacrotuberous and falciform fascia medially and the ischium, hamstring origin, and the inferior margin of the sciatic notch laterally [3]. Finally, the posterior femoral neck is the most lateral portion (Fig. 1).

The contents of the space include the sciatic nerve, the piriformis, the obturator internus/externus, the gemelli, the quadratus femoris, the hamstrings, the superior and inferior gluteal nerves, the lateral ascending vessels of the medial femoral circumflex artery, the ischium, the sacrotuberous and sacrospinous ligaments, and the origin of the ischiofemoral ligament.

Some specifics of the anatomy are critical to understanding the pathological processes that are encountered within the space. From the sciatic notch, the piriformis muscle originates from the ventrolateral surface of the sacrum and courses between the iliotibial band and inserts on superior and posterior aspect of the greater trochanter (Fig. 1). Distal to the piriformis is the cluster of short external rotators: the gemellus superior, obturator internus, and gemellus inferior (Fig. 2). The gemelli blend with the obturator internus onto the anterior aspect of the medial surface of the greater trochanter [3]. The piriformis tendon can be partially blended with this common tendon in its insertion [4]. The obturator internus arises from the inner surface of the anterolateral wall of the pelvis and exits the pelvis through the lesser sciatic foramen. The superior gemellus arises from the outer surface of the ischial spine, and the inferior gemellus arises from the ischial tuberosity. Inferior to this complex is the quadratus femoris, which arises from the upper part of the external border of the ischial tuberosity and inserts on the posterior surface of the femur, along the intertrochanteric ridge. The quadratus assists in external rotation, while the piriformis and short external rotators assist external rotation and abduction of the flexed hip. At the ischium, the biceps femoris and semitendinosus have a common tendinous origin that separates about nine cm from the proximal border of the origin [5] (Fig. 3).

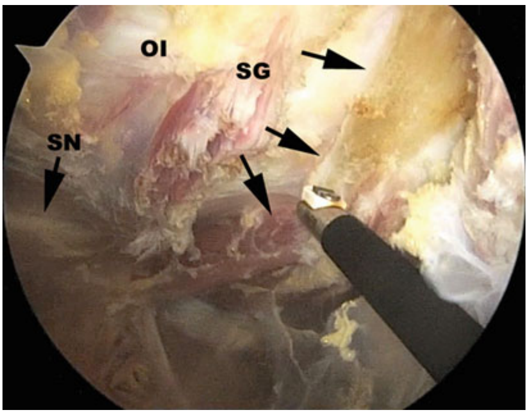


Fig. 2 The sciatic nerve (SN) resting on the common tendon and exiting below the piriformis (*arrows*) (*OE* obturator externus, *SG* superior gemellus)

Six neural structures exit the pelvis through the greater sciatic notch. The neural structures include the sciatic, pudendal, posterior femoral cutaneous, superior gluteal, and inferior gluteal nerves and the nerve to the obturator internus. In addition, the superior and inferior gluteal arteries also exit through the greater sciatic notch. The sciatic nerve courses distally through the space anterior to the piriformis muscle and posterior to the obturator/gemelli complex as well as the quadratus femoris. There are, however, a number of anomalies that are commonly encountered that include entry into the space either through or posterior to the piriformis. These have been documented in up to 17 % of cases in several cadaveric studies [6]. The superior gluteal artery and nerve divide 1–2 cm above the superior border of the piriformis and fan out in a course

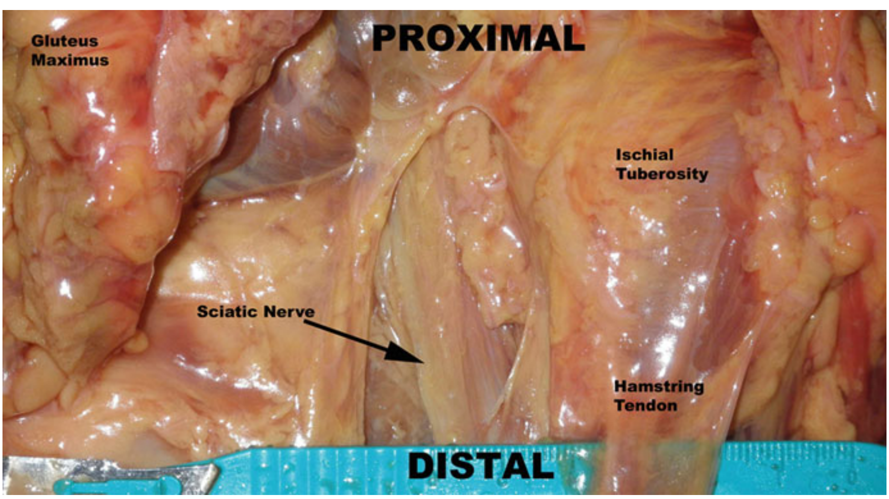


Fig. 3 The lower portion of the deep gluteal space (the view is of a left hip, from the posterior aspect)

anterior and distal to the greater sciatic foramen between the gluteus minimus and medius, supplying those muscles and the tensor fascia femoris [7]. The inferior gluteal nerve and artery enter the pelvis at the greater sciatic notch medial to the sciatic nerve between the piriformis and coccygeus muscles [3]. It descends, along with the sciatic and posterior femoral cutaneous nerves, between the greater trochanter and the ischial tuberosity. Clinically, this nerve is found penetrating the gluteus maximus five cm above its inferior border.

The medial circumflex artery is also relevant within the space. If follows the inferior border of the obturator externus and crosses over its tendon and under the external rotators and piriformis muscle. This vessel terminates as the lateral retinacular vessels, which are the principal blood supply of the femoral head in adults.

The sciatic nerve is located at an average of 1.2 ± 0.2 cm from the most lateral aspect of the ischial tuberosity. Under normal conditions, the sciatic nerve is able to stretch and glide to accommodate strain or compression that occurs with hip motion. One study has documented that with a straight leg raise and knee extension, the sciatic nerve experiences a proximal excursion of 28 mm medial toward the hip joint [8]. Any entrapment of the nerve, therefore, may increase the likelihood of decreased translation of the tissues and subsequent development of pain in the nerve's distribution. Sources of sciatic nerve entrapment include hamstring tendon disruptions and their consequent scar formation immediately adjacent to the nerve. The piriformis tendon is also commonly implicated in compression of the nerve. In addition, malunited ischial avulsion or lesser trochanteric fractures can lead to perineural scar formation. Other etiologies include vascular anomalies, tumors, as well as the gluteus maximus tendon in cases of prior iliotibial band releases. Remote acetabular fractures can also lead to nerve impingement.

One other source that is starting to be understood is ischiofemoral impingement. The disorder has been described in the radiological literature to some extent [9–11]. The mechanism is that of entrapment of the nerve between some portion of the posterior femur and the ischium. The anatomy of this space makes it susceptible to impingement as the clearance between the structures is minimal, especially at the extremes of motion (Fig. 4).

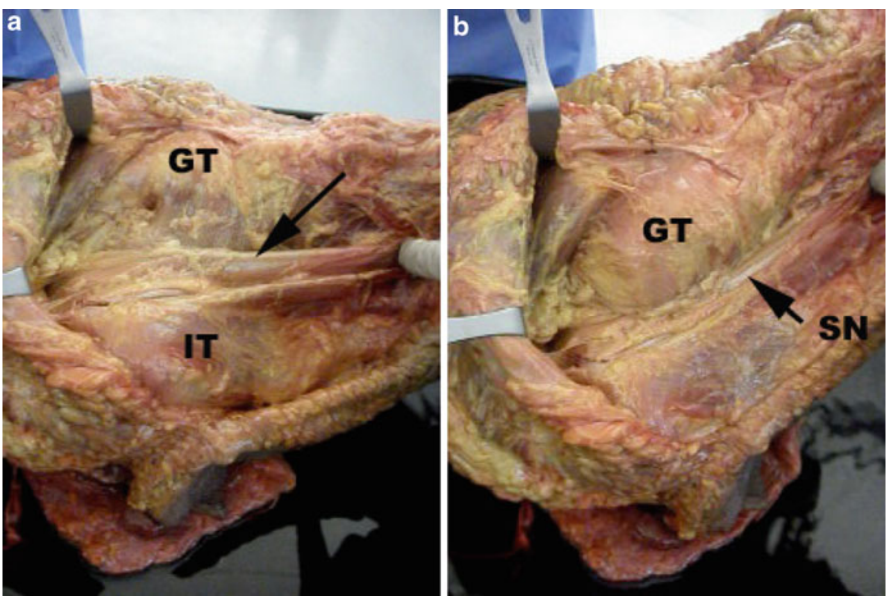


Fig. 4 Ischiofemoral impingement in a cadaver. The view is of a right hip and the proximal portion is to the *left*. (**a**) With the leg in neutral rotation that is the space between the trochanter (GT) and the ischium for the sciatic nerve (SN) to glide. (**b**) With external rotation, there is a diminished space between the GT and the ischium (IT)

Fig. 5 Positioning for access to the deep gluteal space in the lateral position. Note the leg is abducted about 30°

In some cases, the quadratus femoris is hypertrophied, and surgical release is indicated. As well, the lesser trochanter, lying underneath this muscle, may be prominent. Care must be taken during the release of this muscle, as the medial circumflex femoral vessels are within the surgical field. It is vitally important not to compromise these, since they are the primary blood supply to the femoral head, as stated previously.

Surgical Technique

In most cases, the procedure is performed in the supine position and may be performed concomitant to a hip arthroscopy of the central and/or peripheral compartments, if indicated. The procedure is performed with the 30° arthroscope. In some cases, however, it is useful to employ the 70° device for added visualization. It is also possible to require the use of a longer arthroscope (probably a 70° device) in larger patients. The procedure can also be performed in the lateral position with the leg in a slightly abducted position (Fig. 5). This is done in cases where there is no central or peripheral compartment pathology that needs to be addressed.

In the supine position, following the completion of the central and peripheral work, any traction is discontinued, and the leg is abducted to about 30° in order to open the interval between the trochanter and the iliotibial band. The leg is internally rotated, for the same reason. Entrance into the subgluteal space is accomplished by traveling through the peritrochanteric space, which is between the greater trochanter and the iliotibial band. A modified anterior (MA) portal, which has been used for anterior visualization of the central and peripheral compartments, is used to enter the peritrochanteric space between the tensor fascial femoris (laterally) and the rectus femoris (medially) [1] (Fig. 6). This is accomplished by palpating the interval between these two muscle bellies with the blunt arthroscopic probe and cannula.

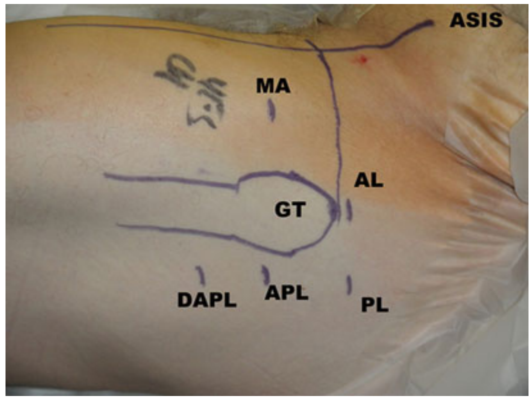


Fig. 6 The typically employed portals for access to the deep gluteal space in a left hip (Note: the leg is to the *left*) (*AL* anterolateral portal, *MA* modified anterior portal, *APL* auxiliary posterolateral portal, *DAPL* distal auxiliary posterolateral portal, *GT* greater trochanter, *ASIS* anterior superior iliac spine)

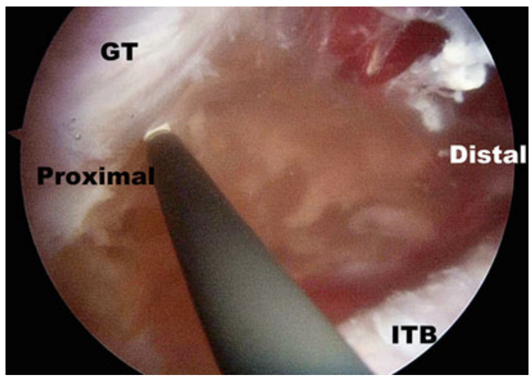


Fig. 7 Visualization of the greater trochanteric bursa in a right hip, with the distal aspect to the right. The visualization portal is the MA, and the instrument is being inserted via the AL portal (*GT* greater trochanter, *ITB* iliotibial band)

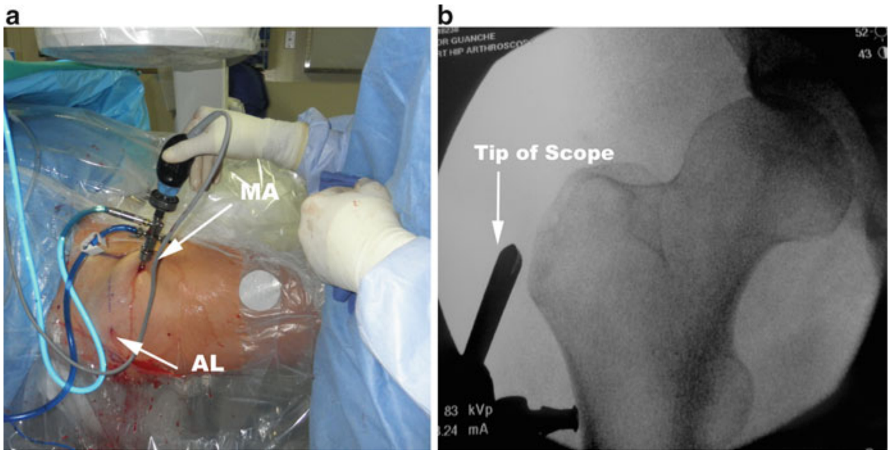


Fig. 8 Appropriate position of the arthroscope in a right hip in order to locate the gluteal sling. The camera is positioned parallel to the body, and the scope is visualizing distally. (**a**) Distal visualization allows one to identify the gluteal sling for orientation. Note that visualization is from the modified anterior (MA) portal and the working portal is the anterolateral (AL) portal. (**b**) Fluoroscopic view of the arthroscope in the right trochanteric bursa

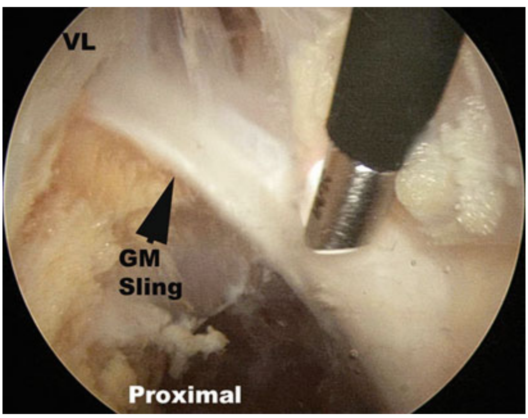


Fig. 9 The gluteal sling in a right hip. Note the fatty tissue immediately posterior to the sling, where the sciatic nerve resides (*VL* vastus lateralis, *GM* sling, gluteus maximus sling)

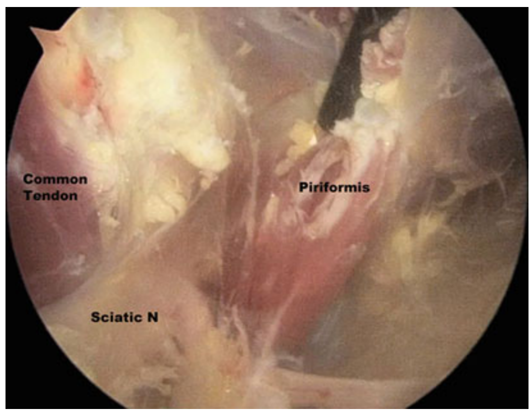


Fig. 10 Piriformis release for sciatic nerve entrapment in a left hip. Note: this is the release of the tendon previously seen in Fig. 2

Ultimately, the space is successfully entered when the lateral aspect of the greater trochanter is palpated and can be confirmed with the use of fluoroscopic guidance. An anterolateral (AL) portal is then employed as a working portal in the trochanteric bursa. The procedure then continues by exposure of the bursa and resection of abnormal bursal tissue, as necessary (Fig. 7).

Once the peritrochanteric space is cleared and any encountered pathology is addressed, the more posterior aspect is identified, and the subgluteal space is formally entered. With respect to orientation, a predictable technique is to place the arthroscope perpendicular to the patient and look in a distal direction in order to identify the gluteus maximus tendon inserting into the linea aspera of the femur posteriorly (Fig. 8). Once this structure is identified, the area of the sciatic nerve can then be known. It lies directly posterior to this structure as it exits the subgluteal space (Fig. 9).

In most cases, an auxiliary posterolateral (APL) portal is created about three cm posterior

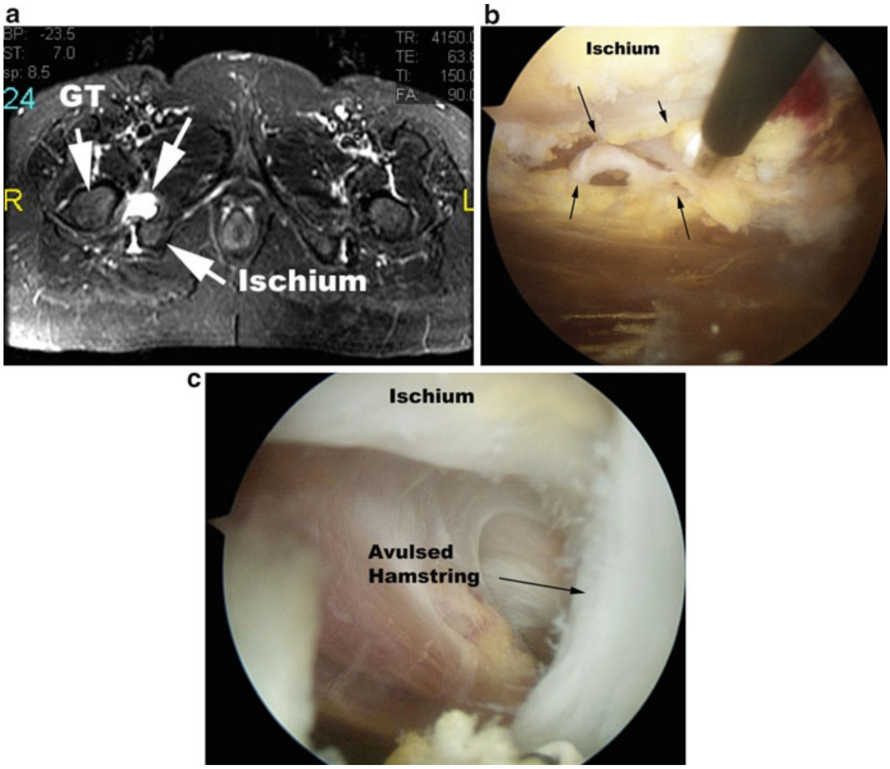


Fig. 11 Ischiofemoral impingement in a right hip. (**a**). Axial, T2-weighted MRI showing fluid in the interval between the ischium and the greater trochanter. The *arrow* is indicating the fluid collection between the greater trochanter and the ischium (*GT* greater trochanter). (**b**) Visualization of the area of hamstring impingement. The *arrows* indicate the rent in the hamstring sheath. The sciatic nerve is immediately posterior in the visualized fatty tissues. (**c**) With retraction of the rent in the sheath, the deep avulsion of the tendon can be seen

to the posterior aspect of the greater trochanter. This serves as a further working portal, while continuing to visualize from the anterior (MA) portal. In some cases, it is necessary to establish an additional, more distal portal for more posterior visualization around the greater trochanter and toward the piriformis. This portal, termed the distal auxiliary posterolateral (DAPL), is created in parallel with the APL and is about four cm distal to that portal (Fig. 6).

The primary area of interest in most of these cases is the sciatic nerve. Secondarily, the area of the hamstring origin can also be a source of pathology and is certainly part of this space. However, a separate chapter in this book serves to describe a more effective way to address primary hamstring and ischial pathology (which includes that the patient be positioned prone), and readers are referred to that section of the textbook for further discussion of this topic.

As previously stated, the nerve is known to be immediately posterior to the gluteal sling so that it can be traced proximally from that point. Inspection of the sciatic nerve then begins distal to the quadratus femoris, just above the gluteal sling. A blunt probe or surgical dissector can then be employed to expose the sciatic nerve and any vascular scar bands over the quadratus femoris and the conjoint tendon of the gemelli and obturator internus. Finally, the piriformis muscle is identified, and any abnormal anatomical variants are identified. In cases where a piriformis nerve release will be performed, the muscle belly is followed laterally into its insertion into the apex of the greater trochanter (Fig. 2). The tendon is typically confluent with the common tendon and

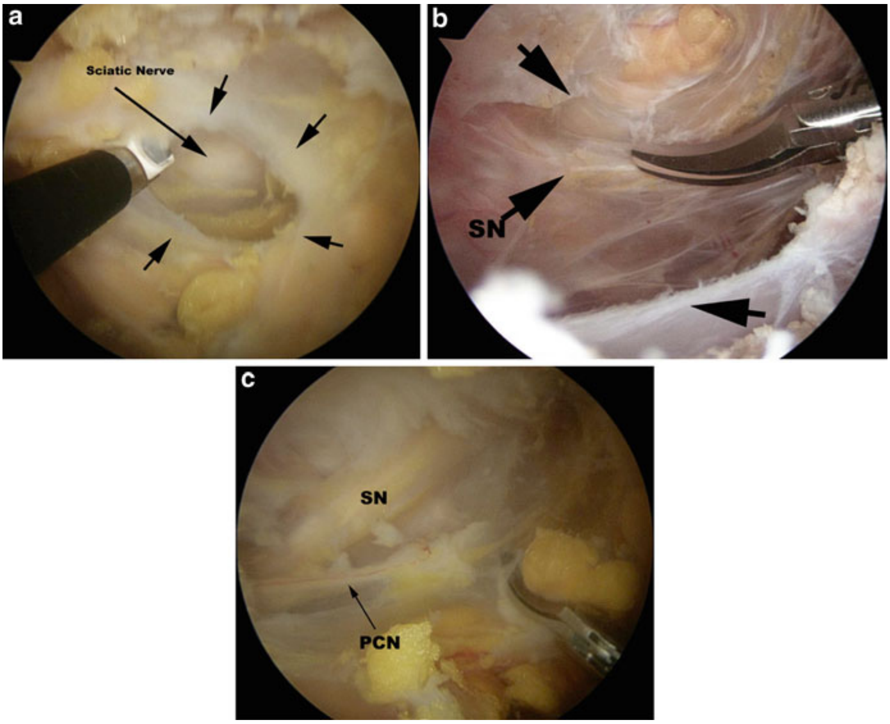


Fig. 12 Dissection of sciatic nerve scar tissue in a patient with ischiofemoral impingement. This is a right hip procedure. (**a**) Visualization via the DAPL and working via the PL portal. The *arrows* are pointing to the areas of scar tissue with the sciatic nerve in the depths of the field. (**b**) With resection of some of the scar tissue, the sciatic nerve is more obvious within the field of view. The *arrows* are pointing to the scar that is being resected (*SN* sciatic nerve). (**c**) The completed nerve decompression. The sciatic nerve (SN) is clearly seen as well as the posterior cutaneous nerve (PCN) that runs parallel to the sciatic, slightly more posteriorly

must be separated from that structure in order to completely release it and allow medial retraction of the belly of the muscle (Fig. 10). It is important to assess the sciatic nerve for its mobility prior to beginning any surgical dissection. With the arthroscope visualizing the nerve, the hip can be flexed and rotated in any direction in order to assess not only the mobility but also for any evident impingement. This can occur anywhere along the posterior aspect of the femur (from the greater to the lesser trochanter) and also against the ischium and hamstring origin (Fig. 11). Following decompression of the nerve, an assessment of the nerve mobility can be done by repeating the active hip motion assessment (Fig. 12).

In general, all of the structures along the course of the sciatic nerve have been implicated as causative factors in chronic sciatic symptoms [1]. The findings by Martin et al. included adhesions over the ischium posteriorly and inferiorly, multiple sciatic nerve branches with multiple branches encased in scar tissue, adhesions of the nerve lateral to ischium with no excursion, and a hypovascular appearance in some nerves. Interestingly, 27 patients in their study had greater trochanteric bursal adhesions that were excessively thickened and appeared to extend to near the sciatic nerve. They also found the sciatic nerve entrapped by the piriformis tendon in 18 patients. Characteristics of the piriformis muscle included splits of the muscle in several cases [1].

Most of the time, a blunt dissector, such as a switching stick, can be employed for release of scar bands. It is recommended that arthroscopic dissection scissors also be available for the dissection of finer tissues that are more adherent to the nerve (Fig. 12). Fibrovascular tissue can also be cauterized with a radiofrequency probe. Constant attention must be paid to the branches of the inferior gluteal artery lying in proximity to the piriformis muscle, as these are critical to the blood supply of the femoral head [1].

One aspect that needs to be taken into consideration is the potential complications in that may occur in the DGS and the lack of historical knowledge of the pitfalls in the treatment of these entities. The most obvious issue is damage to the sciatic nerve. Clearly, this is a critical structure to the function of the entire lower extremity, and damage to it can cause innumerable complications as it relates to function of the extremity. The role of devascularization of the nerve following surgical dissection needs to be evaluated, and parameters need to be established with respect to that issue [1].

Another area that deserves special mention is abdominal (retroperitoneal) fluid extravasation (Fig. 13). This is monitored by maintaining fluid inflow at the minimum pressure that allows good visualization, along with the use of hypotensive anesthesia, when not clinically contraindicated. Other safeguards include the regular monitoring of the patient for any obvious signs of fluid distension as well as the continued awareness of any decrease in body temperature while being monitored by the anesthesia team [12].

Fig. 13 Case of abdominal extravasation. CT scan of a patient who underwent a decompression of the sciatic nerve in the supine position. Examination at the end of the procedure revealed a significant amount of abdominal distension. The CT examination revealed the extent of the fluid extravasation in the retroperitoneal space (*arrows* are pointing to areas of diffuse extravasation) with displacement of the abdominal contents anteriorly (*yellow line* indicates the posterior and inferior extent of the displacement of the abdominal contents)

Summary

As a result of the expanding interest in hip arthroscopy and, more generally, hip pathologies, this area is a recently defined anatomical region that is very amenable to endoscopic access and evaluation. Currently, the techniques available are limited by the lack of insight into the pathologies that are present and how to effectively treat them. However, there is an explosion of knowledge that is taking place as it relates to the diagnosis and treatment of the entities in this space. Further refinement in the diagnosis and management of deep gluteal space pathologies will certainly be seen in the future.

The further improvement of these procedures will most certainly provide a less invasive approach for disorders presently addressed with major open procedures. While conventional open techniques can also address these pathologies, the use of the magnification inherent to arthroscopy adds significant value to any procedure performed in that space, given the delicate nature of the structures contained as well as less overall morbidity. Finally, there is sure to be an expansion of procedures to address some of these previously hard to define disease entities, leading to overall better care of these complex patients.

References

1. Martin HD, Shears SA, Johnson JC, Smathers AM, Palmer IJ. The endoscopic treatment of sciatic nerve entrapment/deep gluteal syndrome. Arthroscopy. 2011;27:172–81.
2. Dezawa A, Kusano S, Miki H. Arthroscopic release of the piriformis muscle under local anesthesia for piriformis syndrome. Arthroscopy. 2003;19:554–7.
3. Guvencer M, Akyer P, Iyem C, Tetik S, Naderi S. Anatomic considerations and the relationship between the piriformis muscle and the sciatic nerve. Surg Radiol Anat. 2008;3:467–74.
4. Meknas K, Christensen A, Johansen O. The internal obturator muscle may cause sciatic pain. Pain. 2003;104:375–80.
5. Miller SL, Webb GR. The proximal origin of the hamstrings and surrounding anatomy encountered during repair. Surgical technique. J Bone Joint Surg Am. 2008;90(Suppl 2 Pt 1):108–16.
6. Smoll NR. Variations of the piriformis and sciatic nerve with clinical consequence: a review. Clin Anat. 2010;23:8–17.
7. Jacobs LG, Buxton RA. The course of the superior gluteal nerve in the lateral approach to the hip. J Bone Joint Surg. 1989;71:1239–43.
8. Coppieters MW, Alshami AM, Babri AS, Souvlis T, Kippers V, Hodges PW. Strain and excursion of the sciatic, tibial, and plantar nerves during a modified straight leg-raising test. J Orthop Res. 2006;24:1883–9.
9. Miller A, Stedman GH, Beisaw NE, Gross PT. Sciatica caused by an avulsion fracture of the ischial tuberosity. A case report. J Bone Joint Surg. 1987;69:143–5.
10. Patti JW, Ouellette H, Bredella MA, Torriani M. Impingement of lesser trochanter on ischium as a potential cause for hip pain. Skeletal Radiol. 2008;37:939–41.
11. Torriani M, Souto SC, Thomas BJ, Ouellette H, Bredella MA. Ischiofemoral impingement syndrome: an entity with hip pain and abnormalities of the quadratus femoris muscle. Am J Roentgenol. 2009;193:186–90.
12. Kocher MS, Frank JS, Nasreddine EY, Safran MR, Philippon MJ, Sekiya JK, Kelly BT, et al. Intra-abdominal fluid extravasation during hip arthroscopy: a survey of the MAHORN group. Arthroscopy. 2012;28:1654–60.

23 Posterolateral Approach to the Hip

Jared R.H. Foran and Craig J. Della Valle

Contents

J.R.H. Foran (✉)
Panorama Orthopaedics and Spine Center, Golden, CO, USA
e-mail: jaredforan@yahoo.com

C.J.D. Valle
Midwest Orthopaedics at Rush, Rush University Medical Center, Chicago, IL, USA
e-mail: craigdv@yahoo.com; craig.dellavalle@rushortho.com

Abstract

The modern posterolateral approach to the hip developed from the posterior approaches described by Kocher and Langenbeck and from subsequent modifications. It is a utilitarian approach to the proximal femur and acetabulum. This approach has a relatively easy learning curve and can be minimally invasive or extensile. There are many indications for the posterolateral approach, and it remains the most common approach for total hip arthroplasty in the United States. With modern techniques and modifications, the dislocation rate associated with this approach rivals that of other common approaches. There is no true internervous or intramuscular plane. To avoid complications, the key vascular and nervous anatomy must be thoroughly understood and respected.

Introduction

The posterolateral approach to the hip affords excellent exposure to both the proximal femur and the acetabulum. Because of its utility, anatomic simplicity, preservation of the abductor musculature, and low complication rate, it is the most commonly used approach for total hip arthroplasty (THA) in the United States [1]. The posterolateral approach is also ideal for any procedure that requires excellent exposure of

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_23

the proximal femur, the acetabulum, or both. A thorough understanding of the key anatomy will help avoid complications when utilizing this approach.

History

What is generally referred to today as the "posterolateral" approach developed from the posterior approaches described by Langenbeck in 1873 [2] and Kocher in 1887 [3]. These approaches were described to aid in the surgical treatment of suppurative and tuberculous infections about the hip, respectively. In 1950 Gibson described a modification, which involved releasing the gluteus minimus and medius muscles to improve acetabular exposure [4]. In 1954 Marcy and Fletcher described a modification, which preserved the abductor musculature and allowed for insertion of an endoprosthesis [5]. Harris, in 1980, described a modification in which the distal end of the incision was extended and angled 45° posteriorly that improved both femoral and acetabular exposure by relieving tension on the soft tissues, particularly in the setting of challenging cases [6].

In more recent years, so-called "minimally invasive" modifications of the posterolateral approach have been described that decrease incision length and soft tissue dissection and purportedly allow for faster postoperative recovery. The ability to perform minimally invasive posterolateral approaches is in large part due to improvements in instrumentation, retractors, and soft tissue handling techniques.

The posterolateral approach is often simply referred to as the "posterior" approach to the hip. However, technically, this is a misnomer. The posterior approach to the hip, also known as the "Southern" approach, which was originally described by Austen Moore [7] and later modified by Iyer [8] and Shaw [9] is less utilitarian than what is now known as the posterolateral approach, because of the relatively limited exposure to the acetabulum that it affords.

Indications

The posterolateral approach is ideal for any procedure that requires excellent exposure of the proximal femur, the acetabulum, or both. Depending on the size of the incision, it provides limited or extensile exposure for total hip arthroplasty, hemiarthroplasty, hip resurfacing arthroplasty, revision hip arthroplasty, resection arthroplasty, osteochondral grafting, surgical dislocation of the hip, removal of intra-articular loose bodies, drainage of intra-articular sepsis, treatment of proximal femoral or acetabular osteomyelitis, tumor resection, fixation of posterior acetabular and posterior column pelvic fractures, open reduction of posterior hip dislocations, and takedown of arthrodesed hips. The posterolateral approach can also be easily extended distally in cases of periprosthetic fracture, where exposure to the femoral shaft is required or in cases in which an extended trochanteric osteotomy is required.

Contraindications

There are few contraindications to the posterolateral approach to the hip. The blood supply to the femoral head originates largely from the medial circumflex and the ascending cervical arterial branches. As such, for procedures that aim to preserve the femoral head, the surgeon must be conscious of this vascular anatomy. In order to avoid the development of osteonecrosis due to vascular compromise, care should be taken to preserve a soft tissue cuff around the femoral neck, including leaving the attachments of the obturator internus and externus, the gemelli inferior and superior, and the quadratus femoris. Alternatively, another approach to the hip should be considered. This is especially true for the open reduction and internal fixation of femoral neck fractures in young patients, in which preservation of the vasculature is paramount. Historically, the posterior approach has been associated with a higher rate of dislocation when used for THA as

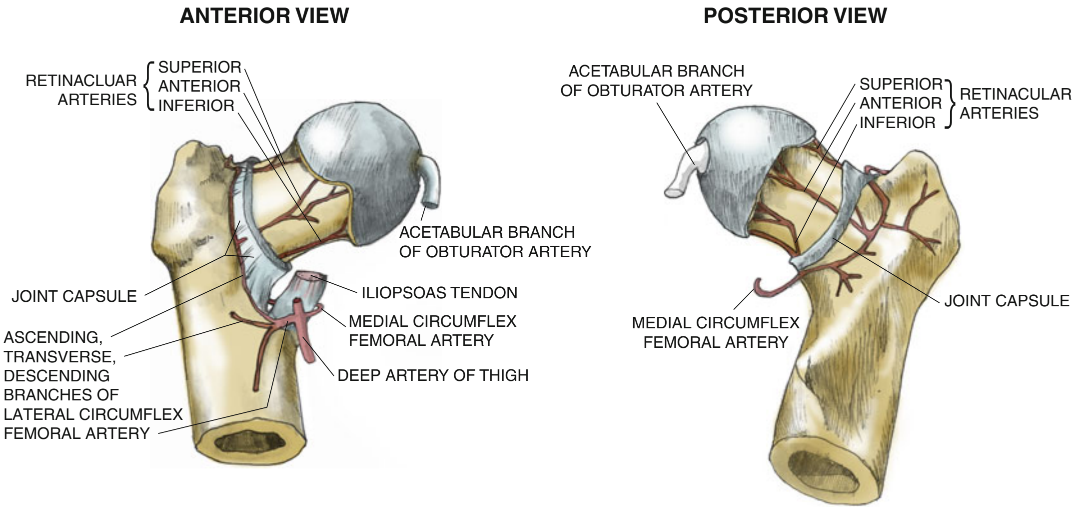


Fig. 1 The vascular supply to the femoral head

compared to the lateral, anterolateral, and anterior approaches. However, with modern tissue-sparing methods, the widespread use of larger femoral heads, increased femoral offset options, and posterior capsular repair, the dislocation rate has been dramatically reduced and in many series, the dislocation rate of less than 1 % rivals that of the other approaches [10, 11].

Key Anatomy

The main blood supply to the femoral head arises from an extracapsular vascular ring at the base of the femoral neck that is supplied posteriorly by the medial femoral circumflex artery and anteriorly by the lateral femoral circumflex artery (minor contribution). The superior and inferior gluteal arteries also have minor contributions. The extracapsular ring gives rise to the ascending cervical branches, which then give rise to the retinacular arteries and the subsynovial intraarticular vascular ring. The superior blood supply to the femoral head arises from the artery of the ligamentum teres (acetabular branch of the obturator artery), which, while variable in size, typically provides only a small amount of blood supply, and is inadequate to nourish the femoral head in the setting of disrupted inferior supply (Fig. 1).

There is no true internervous, intervascular, or intramuscular plane in the posterolateral approach. The gluteus maximus, which is split in line with its fibers is not significantly denervated by the approach, because it receives its nerve supply (from the inferior gluteal nerve), proximal to the split. Care should be taken therefore to avoid aggressive proximal dissection of the gluteus maximus. The vascular supply to the gluteus maximus (from the superior and inferior gluteal arteries) is redundant, and vascular compromise is therefore uncommon. Branches of the inferior gluteal artery (which supply the distal two third of the muscle) are invariably cut when splitting the gluteus maximus, so care should be taken to identify and coagulate them before they are avulsed.

The sciatic nerve is the major nerve at risk in the posterolateral approach. The sciatic nerve is rarely exposed during this approach, but knowledge of its location is crucial. Injury to the sciatic nerve is generally due to compression from retractors or excessive stretching (such as with lengthening of the limb); direct transection is rare. When viewing the field from the surgeon's perspective, the sciatic nerve (or its tibial and peroneal

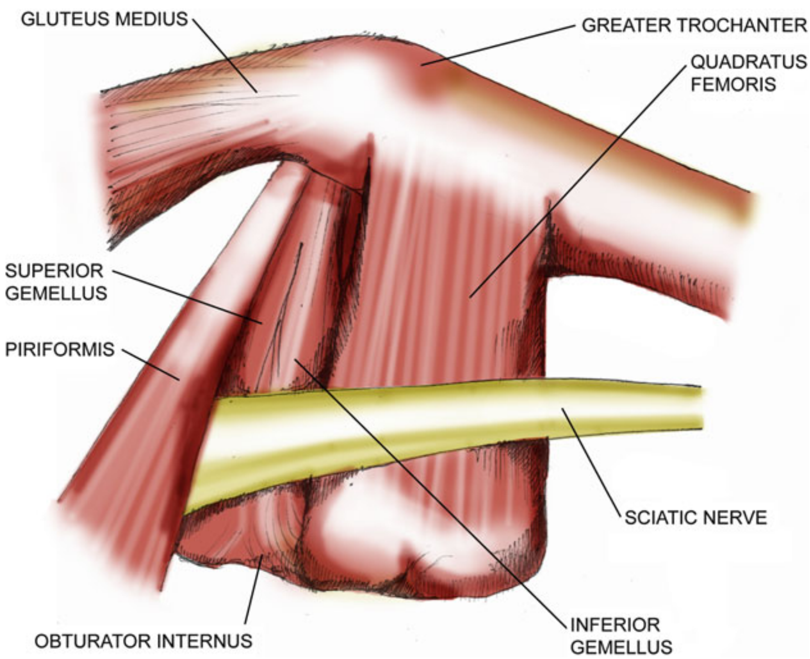


Fig. 2 The course of the sciatic nerve as viewed from the surgeon's perspective

branches) enters the field deep to the piriformis muscle belly. It then continues its course (viewed from the surgeon) superficial to the remainder of the short external rotators (the superior gemellus, obturator internus, inferior gemellus, and the quadratus femoris) and then runs deep to the tendinous insertion of the gluteus maximus (Fig. 2). The femoral nerve is also at risk of indirect injury, usually from anterior acetabular retractor placement. In placing the anterior retractor, care must be taken to hug the bony anterior wall and avoid impinging soft tissue between the retractor and the anterior walls of the acetabulum.

Surgical Technique

Positioning

The patient is positioned in the lateral decubitus position. Care must be taken to ensure that the pelvis is level and secure, particularly in the setting of total hip arthroplasty, as a tilted or rotated pelvis may lead to improper positioning of the acetabular component. The pelvis is secured using well-padded hip positioners on the sacrum posteriorly and the pubis and/or iliac crests anteriorly. Alternatively, a bean bag may be used. Using the floor as an external reference, care is taken to ensure that the gluteal crease is parallel to the floor. As an additional check, the anterior superior iliac spines may be palpated and interspinous line should be perpendicular to the floor. An axillary roll is placed just distal to the axilla on the proximal chest wall to minimize neurovascular compromise on the dependent upper extremity. The dependent leg is well padded to minimize pressure injury to this extremity or compression of the superficial peroneal nerve (Fig. 3).

Superficial Exposure

Proper placement of the skin incision facilitates exposure of the deeper structures, enables the use of more limited incision sizes, and avoids the need for aggressive soft tissue retraction. Begin by palpating and outlining the greater trochanter. Palpation of the trochanter can be facilitated by abduction and adduction of the hip by an assistant. This is particularly helpful in obese patients. In very large patients, a 22-guage spinal needle can be helpful in sounding the proximal femur and

Fig. 3 Patient positioning for the posterolateral approach. The contact points of the hip positioner are well padded and an axillary roll has been placed (not shown). Care is taken to ensure that the pelvis is secure and perpendicular to the ground

identifying the borders of the greater trochanter. A longitudinal incision (ranging from 7 to 20 cm) is made over the posterior one third of the greater trochanter. Too anterior an incision makes retraction of the posterior flap difficult, particularly in the setting of an obese or muscular patient. Too posterior an incision puts the sciatic nerve at risk. Proximally, the incision may be directed toward the posterior superior iliac spine, and distally it is directed in line with the femoral shaft. A good rule of thumb for total hip arthroplasty is that one third of the incision should be proximal to the proximal tip of the greater trochanter and two third should be distal. The skin incision is performed sharply and care is taken to cauterize subcuticular vessels (Fig. 4).

Next, the subcutaneous tissues are dissected using a knife or electrocautery. Care should be taken to identify and cauterize subcutaneous vessels. When performing the subcutaneous dissection, the surgeon should pause frequently to palpate the greater trochanter, which becomes more easily palpable with increasing dissection. The goal is to create one subcutaneous plane that is directed toward the midline of the greater trochanter in the anteroposterior plane. Once the fascia lata is identified, a Cobb elevator may be used to gently clear the remaining subcutaneous tissues in order to expose the fascia and to

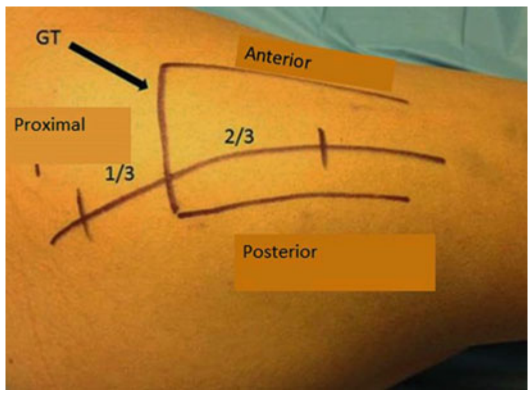


Fig. 4 The incision is centered at the posterior one third of the trochanter. Two-thirds of the incision is distal to the tip of the trochanter (*GT* greater trochanter)

facilitate closure of full thickness fascial flaps at the conclusion of the case (Fig. 5).

Next the fascia is incised in line with the incision. Starting at the distal aspect of the incision approximately 3 cm of fascia is incised from distal to proximal (Fig. 6). Next the surgeon uses the index finger to sweep 360° underneath the fascial incision in order to break up bursal adhesions. Manually releasing these adhesions facilitates the creation of full thickness anterior and posterior fascial and gluteal flaps. Next the fascial incision is carried proximally. The gluteus maximus is split in line with its fibers, and electrocautery is used to

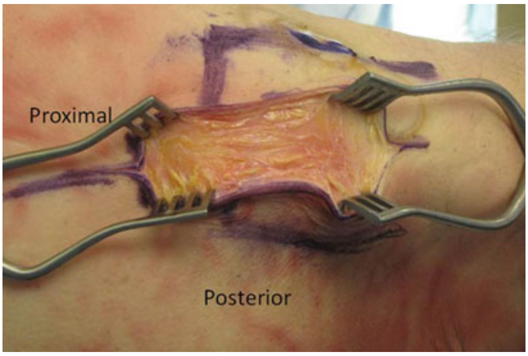


Fig. 5 Subcutaneous dissection is carried out with electrocautery. Care is taken to aim the plane of dissection toward the midline of the greater trochanter in the anteroposterior plane

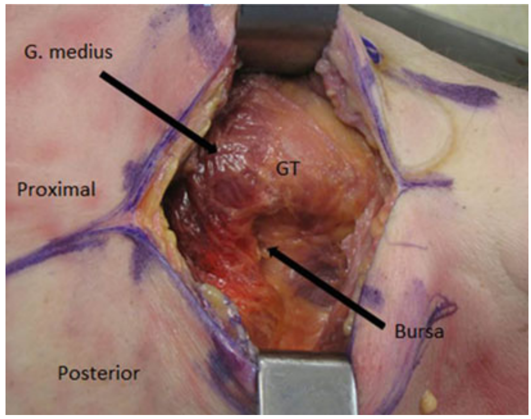


Fig. 7 A Charnley retractor is placed. The gluteus medius is identified and the trochanteric bursa is seen (*GT* greater trochanter)

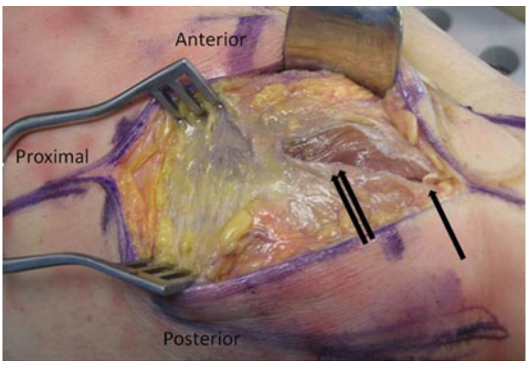


Fig. 6 An incision is made in the distal aspect of the fascia lata and the dissection is carried proximally where the gluteus maximus is split in line with its fibers (*Single arrow*: distal extent of fascial incision; *Double arrow*: gluteus maximus)

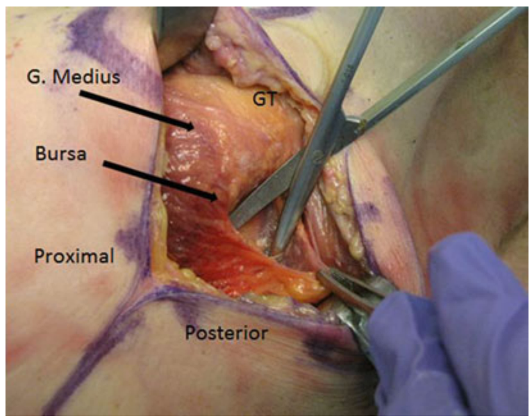


Fig. 8 The bursa is incised from anterior to posterior. Care is taken to cauterize blood vessels around the bursa (*GT* greater trochanter)

coagulate branches of the inferior gluteal artery which are invariably cut. Approximately 5 cm of gluteus maximus splitting is generally adequate to provide excellent exposure of both the femur and acetabulum. Further proximal dissection of the gluteus maximus risks injury to the inferior gluteal nerve and denervation of the muscle. A Charnley retractor is then placed, first on the posterior flap (where the sciatic nerve is at risk if the retractor is placed to deeply) and then the anterior flap (Fig. 7).

Next, the knee is flexed to 90°, and the hip is maximally internally rotated. Exposure is enhanced by ensuring that the thigh is held in extension and not allowed to drift into flexion as is its natural tendency. The bursa is then incised (not excised) from anterior to posterior. Scissor dissection allows for the development of a single plane and for the identification and cauterization of peribursal vessels, which may be copious (Fig. 8). Electrocautery may also be used to incise the bursa. The surgeon should not carry the bursal incision too far posteriorly as the sciatic nerve is at risk. Movement of the foot at any time during the deep dissection is a sign the dissection is close to the sciatic nerve.

The piriformis and remaining short external rotators (superior and inferior gemelli, obturator

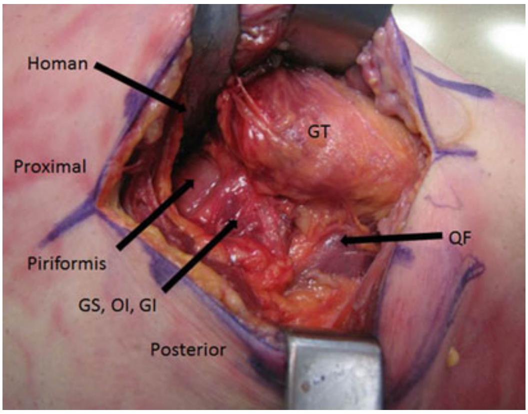


Fig. 9 A bent Homan retractor is placed superior to the piriformis and deep to the gluteus minimus. The short external rotators are clearly identified now (*GT* greater trochanter, *GS* gemellus superior, *OI* obturator internus, *GI* gemellus inferior, *QF* quadratus femoris)

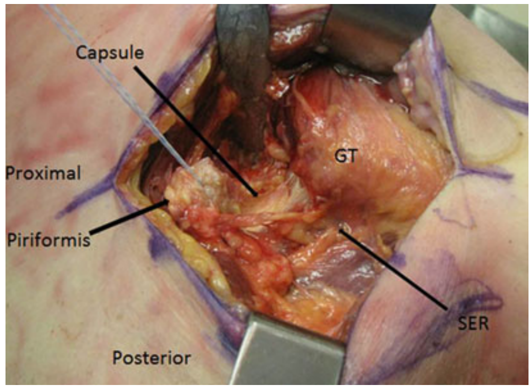


Fig. 10 The piriformis is incised from its insertion in the piriformis fossa and tagged with non-absorbable suture. The capsule is now visible (*GT* greater trochanter, *SER* short external rotators)

internus, and quadratus femoris) are identified. A plane is made superior to the piriformis tendon. The piriformis and gluteus minimus are often confluent at the level of the piriformis tendon, and a Cobb elevator is useful in defining this plane. Next a bent Homan retractor is placed superior to the piriformis and deep to the gluteus minimus and medius, taking care to not retract vigorously as this may damage the abductor musculature and increase the risk for heterotopic ossification (Fig. 9). The tip of the Homan is directed toward the anterior-superior acetabulum and "toed-in." In most cases the piriformis is then taken down at its insertion at the piriformis fossa and tagged with a large gauge nonabsorbable suture. Some surgeons prefer to preserve the piriformis insertion to augment stability (Fig. 10).

Next, with the hip under maximal internal rotation, the hip capsule and short external rotators are taken directly off their insertion to the proximal femur as one layer; while it is possible to take them in separate layers, one layer is simpler and yields a stronger flap for later repair. Distally the quadratus femoris may be spared from dissection or the proximal one third may be released. Care must be taken when dissecting the proximal quadratus, because the medial femoral circumflex artery may be cut and may retract, causing troublesome bleeding. Slowly cauterizing the quadratus during this dissection helps avoid this problem.

There are several described methods of performing the capsulotomy. A single longitudinal capsulotomy allows for easy exposure and affords a strong posterior capsular closure at the conclusion of the case. The capsulotomy is carried from distal to proximal, incising the capsule and external rotators as close to their femoral insertion as possible. The proximal extent of the capsulotomy is carried just proximal to the posterior superior aspect of the acetabulum, generally following the posterior border of the gluteus medius and concluding once the posterosuperior acetabular labrum has been transected. Two or three additional nonabsorbable tag sutures are placed in the capsule and rotators. When tagging these structures the needle should be directed from posterior to anterior to avoid injury to the sciatic nerve. Once tagged, the capsule and rotators can be reflected posteriorly to protect the sciatic nerve and to enhance acetabular exposure (Fig. 11).

It should be noted that if a femoral head preserving procedure is being undertaken, then the capsule and short external rotators should not be incised directly from the femur. Instead, a cuff of 1–2 cm of this soft tissue should be left on the femoral insertion in order to preserve the blood supply to the femoral head.

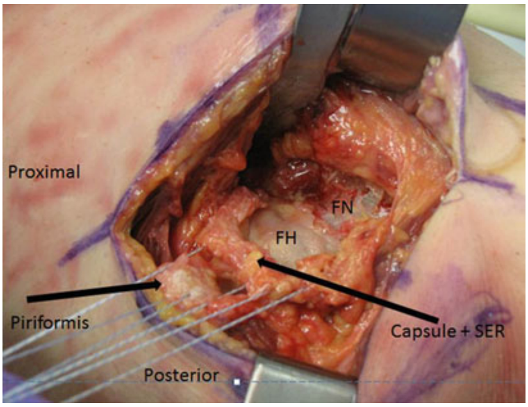


Fig. 11 The capsule and short external rotators are incised off the insertion to the proximal femur from distal to proximal and tagged with non-absorbable suture (*FH* femoral head, *FN* femoral neck, *SER* short external rotators)

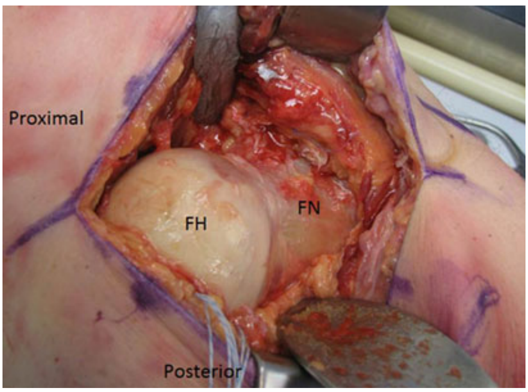


Fig. 12 The hip is dislocated and retractors are placed anteriorly around the femoral neck (*FH* femoral head, *FN* femoral neck)

Depending on the procedure being performed, several options are now possible. The hip may now be dislocated at this time (Fig. 12). If hip dislocation is difficult at this point, it is often due to residual inferior capsular attachments to the posteroinferior femoral neck. Complete detachment of this inferior capsular reflection (the zona orbicularis) should allow for hip dislocation without resistance. When resistance is encountered, forceful dislocation may result in fracturing of the femur and should be avoided.

If hip arthroplasty is being performed, then retractors are placed inferior and superior to the femoral neck and an oscillating and/or reciprocating saw is used to cut the neck. Acetabular exposure is greatly enhanced by cutting the femoral neck, but it is possible to expose the acetabulum with the head in place (such as may be necessary in hip resurfacing, acetabular tumor resection, or loose body removal). In these cases, it is usually necessary to create a "pocket" anterosuperiorly under the abductors in order to place the femoral head.

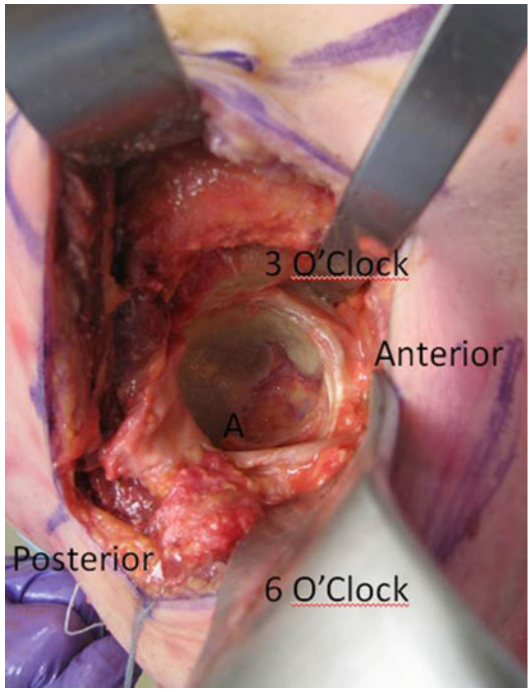


Fig. 13 The acetabulum is exposed. Retractors are placed around the acetabulum (*A* acetabulum)

To expose the acetabulum, retractors are placed anteriorly, at approximately the 3 o'clock position, and posteroinferiorly (Fig. 13). The use of Charnley pins or Steinman pins in the superior ilium and ischium may enhance superior and posterior exposure, respectively. The labrum and pulvinar are excised for total hip arthroplasty, but the labrum is retained for hemiarthroplasty to improve stability.

Exposure of the proximal femur for arthroplasty is readily achieved with two Mueller-type femoral neck elevators. One elevator is placed anteromedially deep to the calcar, and

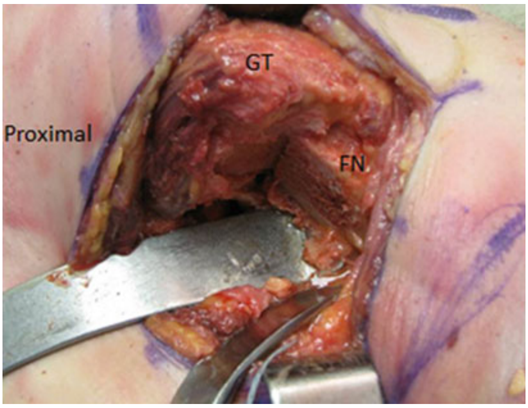


Fig. 14 Preparation of the femur: The femoral head has been osteotomized. The proximal femur is elevated with two Mueller- type retractors (*GT* greater trochanter, *FN* femoral neck after osteotomy)

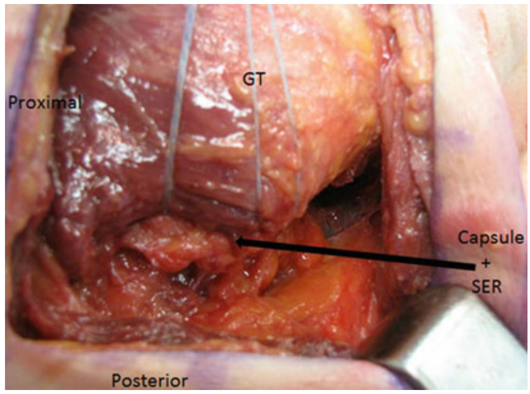


Fig. 15 Closure of the capsule and short external rotators (*GT* greater trochanter, *SER* short external rotators)

the second is placed directly anteriorly. The hip is held in maximum internal rotation and flexion (Fig. 14).

Capsular closure can be achieved in multiple ways. A direct capsule to capsule closure (which requires preservation of some anterior capsule at the time of capsulotomy) may be performed. This closure has the benefit of being "tension free" in that it is less affected by the tension created with hip internal rotation and flexion. It is also possible to approximate the capsule and short external rotators through drill holes in the greater trochanter. An equally reasonable closure is achieved by approximating the capsule and rotators to the posterior border of the abductor muscle and tendon insertion just superior to the greater trochanter. The disadvantage of drill holes are the possibility of trochanteric fracture, while suturing into the abductor tendon has the disadvantage of compromising this important tendinous insertion. Each method however provides an excellent checkrein to posterior dislocation (Fig. 15).

Next the deep fascia and gluteus maximus is approximated. Care is then taken to eliminate dead space in the subcutaneous tissue with layered deep sutures if necessary. The skin is approximated based on the surgeon's preference.

Avoiding Complications

The most common complications associated with the posterolateral approach to the hip are sciatic and femoral nerve injury, dislocation, and heterotopic ossification. A thorough knowledge of the anatomic course of the sciatic nerve, careful posterior retractor placement, and minimizing undue stretch on the nerve (such as what occurs with excessive limb lengthening in THA or with the intraoperative position of flexion, internal rotation, and adduction during femoral preparation) will help minimize direct and indirect injury to the sciatic nerve. Careful placement of the anterior acetabular retractor (avoiding trapping soft tissue between the anterior retractor and the anterior wall) will help minimize femoral nerve injury. Appropriate exposure to allow optimal component position in THA as well as preservation and repair of the posterior capsule and short external rotators decreases the dislocation risk. Finally, meticulous dissection and avoidance of unnecessary damage to the gluteus medius and minimus has been reported to minimize the formation of heterotopic ossification.

Summary

The posterolateral approach to the hip is a utilitarian approach that provides excellent exposure to the proximal femur and acetabulum. This approach is useful for many procedures, including primary and revision hip arthroplasty, osteochondral grafting, surgical dislocation of the hip, surgical treatment of periarticular infection, tumor resection, and fixation of posterior acetabular and posterior column pelvic fractures. There are few contraindications, but preservation of the vascularity to the femoral head must be considered in head sparing procedures. The superficial dissection involves skin and subcutaneous dissection, a longitudinal incision of the fascia lata, and splitting of the gluteus maximus fibers. Deep dissection involves incising the insertion of the short external rotators and the posterior hip capsule. Complications including sciatic or femoral nerve injury, dislocation, and heterotopic ossification can be avoided with a thorough understanding of the applied anatomy and with meticulous capsular repair.

References

1. Berry DJ, Bozic KJ. Current practice patterns in primary hip and knee arthroplasty among members of the American Association of Hip and Knee Surgeons. J Arthroplast. 2010;25(6 Suppl):2–4.
2. VonLangenbeck B. Congress of German society for surgery, 4th session, 1873. Klin Chir. 1874;16:263.
3. Kocher T. In: Stiles H, editor. Textbook of operative surgery. 4th ed. Edinburgh: Adam and Charles Black; 1903.
4. Gibson A. Posterior exposure of the hip joint. J Bone Jt Surg Br Vol. 1950;32-B(2):183–6.
5. Marcy GH, Fletcher RS. Modification of the posterolateral approach to the hip for insertion of femoral-head prosthesis. J Bone Jt Surg Am Vol. 1954;36-A (1):142–3.
6. Harris WH. Advances in surgical technique for total hip replacement: without and with osteotomy of the greater trochanter. Clin Orthop Relat Res. 1980;146:188–204.
7. Moore AT. The self-locking metal hip prosthesis. J Bone Jt Surg Am Vol. 1957;39-A(4):811–27.
8. Iyer KM. A new posterior approach to the hip joint. Injury. 1981;13(1):76–80.
9. Shaw JA. Experience with a modified posterior approach to the hip joint. A technical note. J Arthroplast. 1991;6(1):11–8.
10. Sierra RJ, Raposo JM, Trousdale RT, Cabanela ME. Dislocation of primary THA done through a posterolateral approach in the elderly. Clin Orthop Relat Res. 2005;441:262–7.
11. White Jr RE, Forness TJ, Allman JK, Junick DW. Effect of posterior capsular repair on early dislocation in primary total hip replacement. Clin Orthop Relat Res. 2001;393:163–7.

Surgical Approach to Open Hip Surgical Dislocation

24

Thomas J. Ellis and John M. Ryan

Contents

T.J. Ellis (✉)
Department of Orthopaedics, Ohio State University, Columbus, OH, USA
e-mail: tomellis1234@gmail.com

J.M. Ryan
Department of Orthopaedics, Division of Hip Preservation, Ohio State University, Columbus, OH, USA
e-mail: johnm.ryan@osumc.edu

Abstract

First described by Ganz, surgical dislocation of the hip provides a safe and effective means of gaining direct access to femoral head and neck and the acetabulum, labrum, and articular surfaces for a variety of surgical indications.

Introduction

Surgical hip dislocation is a powerful technique employed by hip preservation surgeons for treatment of a variety of hip pathologies, including correction of femoroacetabular impingement pathoanatomy: cam and pincer lesions, (acetabular overcoverage retroversion or coxa profunda) [1, 2], repair of the hip labrum, repair of focal chondral lesions of the femoral head or acetabulum [3], and in rare cases femoral neck osteotomy or trochanteric advancement.1 The technique provides the surgeon outstanding visualization of the entire acetabular and femoral surface and has therefore also been employed to aid in anatomic reduction of acetabular and femoral head fractures, [4] as well as treatment of relatively uncommon conditions such as pigmented villonodular synovitis, synovial chondromatosis and osteochondromas of the femoral neck [5].

Traditionally, surgical hip dislocation was an infrequently utilized surgery with persistent concerns regarding the risk of iatrogenic AVN

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_24

Fig. 1 The patient is placed in the lateral decubitus position with posts or beanbag. Surgical prep should extend from above iliac crest and include entire operative extremity (Copyright Thomas J. Ellis)

due to compromise of the femoral head vascular supply. However, in 2001, Ganz provided a description of a new surgical hip dislocation technique utilizing a trochanteric osteotomy approach, and showed that this approach was safe and effective, with no AVN of the femoral head reported in over 200 cases [6]. The critical element of this approach is a detailed understanding of the blood supply to the femoral head, which takes its most important contribution from the medial femoral circumflex artery (MFCA). Gautier and colleagues provided a detailed description of the vascular anastomoses and branches MFCA with their relationship to relevant surgical landmarks [7]. These anatomic data form the basis of this technique. Further evidence demonstrating the safety of the procedure was provided by Notzli et al in a study that used laser Doppler flowmeter to assess blood flow in the femoral head at various points of the dislocation procedure. While blood flow in the femoral head was noted to be transiently reduced in some positions of the hip after dislocation, it returned to normal following reduction of the head back into the acetabulum [8]. This chapter provides a step by step description of this technique with emphasis on relevant surgical landmarks.

Setup

A standard OR table may be used. Intraoperative fluoroscopy and radiographs are typically not utilized.

Positioning and Draping

The patient is positioned in the lateral decubitus position (Fig. 1). The patient's position should be stabilized with side posts positioned at the sacrum posteriorly and pubic symphysis anteriorly or a bean bag. Care should be taken prior to the prep and drape to ensure that the posts or bean bag do not impede a full range hip motion or encroach on the operative field. The surgical prep should include the entire operative extremity and should be extended proximally above the iliac crest. A standard hip drape may be utilized; however, it is helpful to have a drape with a large pocket located on the anterior aspect of the patient to allow for placement of the leg and foot during dislocation of the hip later in the procedure. The draping should allow for access from the iliac crest to mid thigh. The leg should be draped free to allow for unimpeded range of motion.

Bony Landmarks

The greater trochanter should be palpated carefully to establish its anterior, posterior, and superior extent. This should be outlined with a marking pen. The iliac crest should be palpated and marked as well with particular note of the anterior superior iliac spine (ASIS) and anterior inferior iliac spine (AIIS). The latter will be only palpable in thin individuals.

Incision

A straight lateral skin incision is used. The length of the incision depends on the size of the patient but is typically 20–25 cm in length. The incision is centered over the anterior third of the greater trochanter and is oriented in a slightly proximal/posterior to distal/anterior direction.

Superficial Dissection

The skin and subcutaneous tissues are incised sharply. A Gibson or Kocher-Langenbeck approach may be used; however, the Gibson approach, which utilizes the interval between gluteus maximus and gluteus medius, lowers the risk of injury to the inferior gluteal nerve. The interval between gluteus maximus and medius can be identified by a number of small perforating vessels which are branches of the inferior gluteal artery typically run in this area. The overlying fascia is incised sharply to reveal the fibers of the two muscles. This fascial incision is carried longitudinally down over the trochanter and vastus lateralis to the inferior aspect of the operative field. As the fascia is opened, the insertion of the gluteus maximus tendon on the posterior aspect of the femur should be noted in the inferior aspect of the field.

Deep Dissection

At this point, internal rotation of the hip aids in visualization as the dissection progresses. Returning to the proximal portion of the field, close inspection of the gluteus medius reveals a thin fascial layer overlying this muscle which may be incised and elevated at its posterior aspect. Any bursal tissue should be excised from the greater trochanter. This will reveal the trochanteric branch which arises from the deep branch of the medial femoral circumflex artery running along the posterior aspect and up onto the lateral surface of the greater trochanter. Attention is turned to the posterior aspect of the gluteus medius near its trochanteric insertion. A strip of fatty tissue will be noted here, and the tissue should be carefully spread to reveal the piriformis tendon underneath running toward the piriformis fossa (Fig. 2). Care should be taken when dissecting as this area is quite vascular as it contains an anastomosis between the inferior gluteal artery and the deep branch of the medial circumflex artery.

Fig. 2 The piriformis tendon, running toward the piriformis fossa, is revealed. This region is highly vascular and care must be taken during dissection (Copyright Thomas J. Ellis)

Fig. 3 Posterior aspect of the greater trochanter. Electrocautery is used to mark the osteotomy path (Copyright Thomas J. Ellis)

Trochanteric Osteotomy

The hip is further internally rotated to bring the posterior aspect of the trochanter into full view in the center of the field. In preparation for the osteotomy, electrocautery can be used to mark the path of the osteotomy along the posterior aspect of the greater trochanter (Fig. 3). Vessels

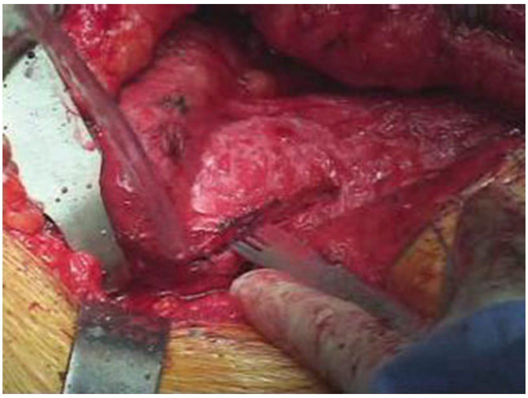


Fig. 4 The osteotomy progresses posterior to anterior across the greater trochanter toward the vastus ridge (Copyright Thomas J. Ellis)

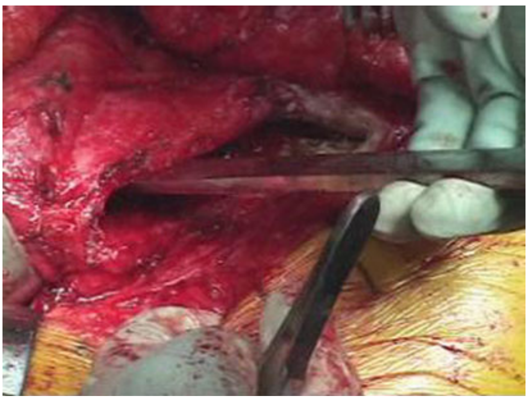


Fig. 5 An osteotome should be used to complete the osteotomy at the anterior cortex instead of the sagittal saw (Copyright Thomas J. Ellis)

running along the posterior trochanter can be coagulated as well. The osteotomy should be made such that the majority of the gluteus medius insertion and vastus lateralis origin remain attached to the trochanteric fragment. The hip is internally rotated, and the blade of the sagittal saw should be roughly parallel to the leg to maintain the proper trajectory for the osteotomy as it progresses from posterior to anterior across the greater trochanter from the posterior superior trochanter toward the vastus ridge (Fig. 4). A finger may be placed over the top of the piriformis tendon during the cut to ensure that this critical area is not violated by the saw blade. The piriformis

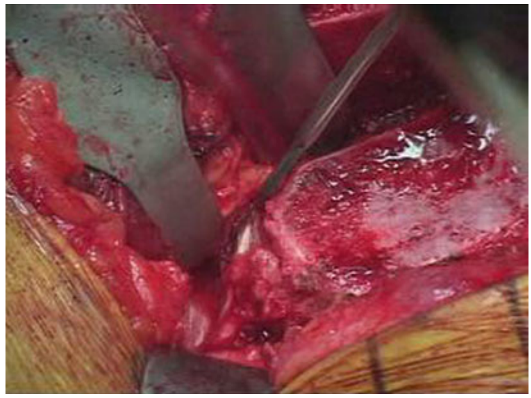


Fig. 6 Remaining posterior fibers of the gluteus medius should be incised from the trochanteric remnant. Care should be taken to keep the knife blade between the medius tendon fibers and the piriformis tendon to protect the MFCA (Copyright Thomas J. Ellis)

tendon and other short external rotators should remain attached to the femur to protect the vascular supply to the femoral head via the deep branch of the medial femoral circumflex artery (MFCA). This vessel travels proximal to the quadratus femoris, crosses the obturator externus, and continues anterior to the superior and inferior gemellus and obturator internus.

The majority of the trochanteric osteotomy can be accomplished with the sagittal saw; however, it is advisable to leave the anterior cortex of the trochanter intact and to finish this part with an osteotome (Fig. 5). This leaves an anterior cortical ridge which may increase stability of the trochanteric fragment for later refixation. Next, a broad osteotome is used to carefully lever the trochanter anteriorly completing the osteotomy. At the distal aspect, the vastus lateralis fibers are carefully elevated off of the femur with either scalpel or electrocautery. Proximally the remaining posterior fibers of gluteus medius are incised sharply from the trochanteric remnant (Fig. 6). In order to protect the MFCA, identify the piriformis tendon and keep the knife blade between the medius tendon fibers and the piriformis tendon. It is advisable to avoid electrocautery in this area due to the close proximity to the medial circumflex artery. Identify the interval between the gluteus minimums and piriformis. The inferior margin of the gluteus minimus often lies underneath the piriformis

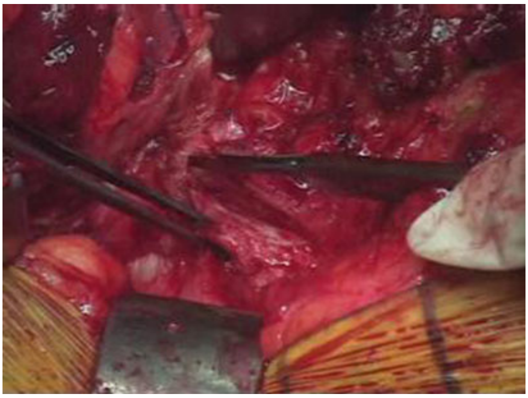


Fig. 7 In the “z” shape capsulotomy, the initial incision is made longitudinally down the neck along the posterior aspect of the iliofemoral ligament (Copyright Thomas J. Ellis)

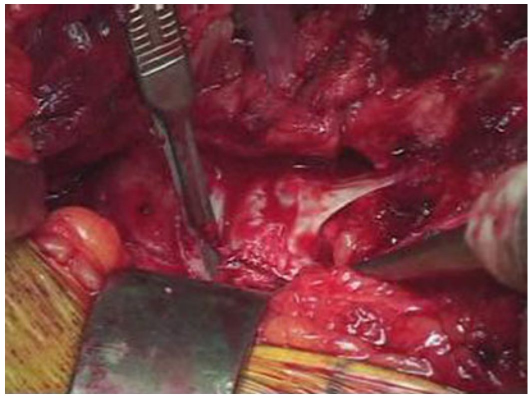


Fig. 8 Second limb of “z” capsulotomy – extending posteriorly along capsulolabral junction (Copyright Thomas J. Ellis)

muscle. This should allow for a blunt, curved Hohmann-type retractor to be slid from posterior to anterior into the interval and be positioned along the lateral and anterior joint capsule. Care should be taken to avoid damaging the minimus muscle belly during this maneuver. Flexing and externally rotating the hip will aid in anterior retraction of the medius, minimus, and trochanteric fragment. Placement of a pointed Hohmann-type retractor along the anterior aspect of the femur at the level of the osteotomy will also aid in flipping the trochanteric fragment anteriorly. Using cautery or sharp dissection, release remaining fascial attachments of the minimus muscle from the intact trochanter to expose the joint capsule. Flexing and externally rotating the leg will “open up” the anterior joint and facilitate this portion of the exposure. A self retaining Charnley-type retractor can be placed between the gluteus maximus and the trochanteric fragment to maintain capsular exposure.

Capsulotomy

With elevation of the gluteus minimus off of the joint capsule and reflected anteriorly, a good view of the capsule from posterior to anterior should be obtained. A “z”-shaped capsulotomy is then performed. First, a longitudinal cut is made down the neck just along the posterior aspect of the iliofemoral ligament (Fig. 7). The proximal aspect of this is connected with a second cut that extends posteriorly along the capsulolabral junction (Fig. 8). The posterior cut is placed proximally to ensure that the MFCA is not damaged during the capsulotomy. Care must be taken at this point to ensure that the labrum or articular cartilage is not violated by the scalpel. The third limb of the capsulotomy is created extending medially along the anterior aspect of the neck following intertrochanteric line, but stopping before the lesser trochanter is reached (Fig. 9). A small cuff of capsule should be left attached to the femur distally to aid in closure. Elevate the anterior capsular flap, and place a sharp Hohmann retractor into the iliopectineal fossa between the labrum

Fig. 9 A third capsulotomy limb extends medially along the anterior aspect of the neck, following the intertrochanteric line, but stopping short of the lesser trochanter (Copyright Thomas J. Ellis)

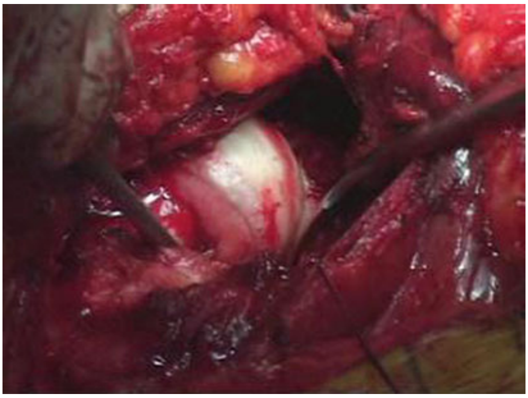


Fig. 10 The hip is dislocated with flexion and external rotation. A bone hook is used to assist in delivering the femoral head out of the acetabulum (Copyright Thomas J. Ellis)

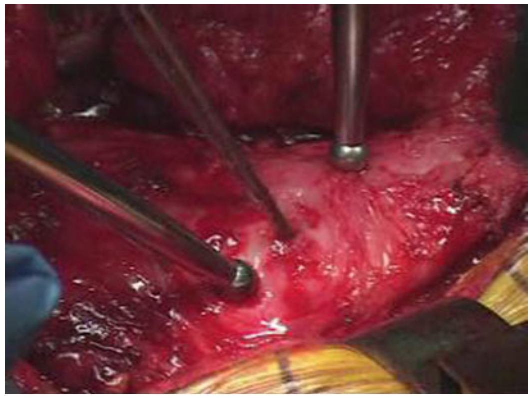


Fig. 11 The greater trochanter is then repositioned and reattached with a pair of parallel screws 3.5 cortical screws. Ball spike pushers are useful for holding provisional reduction (Copyright Thomas J. Ellis)

and capsule. In addition, blunt Hohmann retractors can be placed intra-articularly against the medial and lateral neck to improve visualization.

Dislocation and Femoral Head and Acetabular Exposure

After completion of the capsulotomy, the femoral head can be dislocated out of the joint with flexion and external rotation of the hip (Fig. 10). The foot can be kept sterile by placing it in the pouch that is part of the anterior drape. A bone hook can be used to assist in subluxing the femoral head out of the acetabulum. By lowering the knee the femoral head is further delivered out of the field to allow for excellent visualization and access to the femoral head and neck.

Visualization of the acetabulum is accomplished by flexing the hip and pushing the femoral head posteriorly. This is augmented by placing a femoral elevator retractor inferiorly near the acetabular fossa and levering off of the femoral neck with the back of the retractor, to further displace the femur posteriorly. If more exposure is needed, a curved Mayo-type scissor can be used to cut the ligamentum teres and allow for full dislocation of the hip. This provides excellent visualization of the majority of the acetabular surface. The MFCA is readily visualized in the posterolateral femoral neck synovial fold.

Capsular Closure

The capsule is reapproximated with absorbable sutures. Avoid overtensioning the capsule as this may impede blood flow from the MFCA to the femoral head.

Trochanter Reattachment

The greater trochanter is repositioned in its anatomic location and fixed with two parallel screws 3.5 mm cortical screws (Fig. 11). The screw trajectory should be planned to engage the far cortex at a point at or just below the lesser trochanter to eliminate the possibility of the screw tip irritating the psoas tendon near its attachment on the lesser trochanter (Fig. 12).

Pearls

A straight skin incision centered over the greater trochanter coupled with a careful layer closure of the fascial and subcutaneous layers gives a better cosmetic result by decreasing the occurrence of the “saddlebag deformity” or drooping of the subcutaneous tissue, particularly in females.

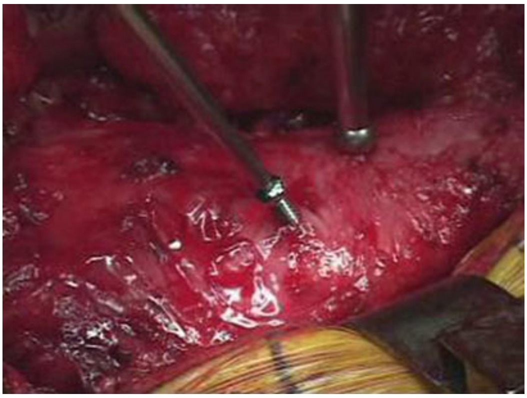


Fig. 12 Screws should be angled at or just below the lesser trochanter to avoid irritation of the psoas tendon (Copyright Thomas J. Ellis)

Exposure is facilitated by flexing and adducting the hip during anterior mobilization of the trochanteric, gluteus medius, gluteus minimus, and vastus lateralis.

Remember the MCFA penetrates the joint capsule between the piriformis and superior gemellus muscle. Proximal to the piriformis, submuscular/extracapsular dissection can be safely performed without risk to the artery.

Summary

Surgical hip dislocation is a well-described procedure that allows the surgeon outstanding access to the femoral head and acetabular surfaces. It is useful for a variety of surgical indications. Clear understanding of the complex anatomy about the hip coupled with a careful surgical dissection, precise placement of retractors, and positioning of the operative leg during various parts of the procedure is crucial for safe and effective implementation of this procedure.

References

1. Beck M, Puloski S, Leunig M, Siebenrock K, Ganz R. Surgical dislocation of the adult hip. A technique for the treatment of articular pathology of the hip. Semin Arthroplasty. 2005;16(1):38–44.
2. Ellis TJ, Beck M. Trochanteric osteotomy for acetabular fractures and proximal femur fractures. Orthop Clin North Am. 2004;35(4):457–61.
3. Ganz R, Gill T, Gautier E, Ganz K, Krügel N, Berlemann U. Surgical dislocation of the adult hip a technique with full access to the femoral head and acetabulum without the risk of avascular necrosis. J Bone Joint Surg Br. 2001;83(8):1119–24.
4. Gautier E, Ganz K, Krügel N, Gill T, Ganz R. Anatomy of the medial femoral circumflex artery and its surgical implications. J Bone Joint Surg Br. 2000;82(5):679–83.
5. Kim Y, Millis MB. Safe surgical dislocation of the hip. Oper Tech Orthop. 2005;15(4):338–44.
6. Lavigne M, Parvizi J, Beck M, Siebenrock KA, Ganz R, Leunig M. Anterior femoroacetabular impingement: part I. techniques of joint preserving surgery. Clin Orthop. 2004;418:61–6.
7. Nötzli H, Siebenrock K, Hempfing A, Ramseier L, Ganz R. Perfusion of the femoral head during surgical dislocation of the hip. Monitoring by laser Doppler flowmetry. J Bone Joint Surg Br. 2002;84(2):300–4.

Smith-Petersen Approach to the Hip

25

Ronak M. Patel and Michael D. Stover

Contents

Abstract

The anterior approach to the hip is a muscle-sparing exposure that preserves the blood supply to the native femoral head. From its earliest description, the anterior approach has been utilized from irrigation of the hip joint to surgical dislocations of the hip and, more recently, to total hip arthroplasty. Modifications to the true Hueter or Smith-Petersen anterior approach have led to fewer complications with the lateral femoral cutaneous nerve. This chapter will describe the surgical technique for the modified Hueter/Smith-Petersen anterior approach to the hip.

Introduction

Many surgical approaches to the hip have been described, including posterior, anterolateral, lateral, lateral transtrochanteric, medial, and anterior. An optimal approach prevents muscle splitting or detachment and preserves the blood supply to the femoral head. The anterior approach meets these requirements and is the only described approach to the hip that is muscle sparing.

The anterior approach was first described by German surgeon Carl Hueter in 1881. However, it was Norwegian-born American surgeon Marius N. Smith-Petersen who popularized this approach in the English-speaking world [1, 8]. His extensive use of the anterior approach for open reduction of congenital hip dislocations at

R.M. Patel (✉)
Department of Orthopaedic Surgery, Northwestern University – Feinberg School of Medicine, Chicago, IL, USA
e-mail: r-patel7@md.northwestern.edu

M.D. Stover
Department of Orthopaedic Surgery, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA
e-mail: mstover@nmff.org

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_25

Table 1 Procedures utilizing the anterior approach

Total hip arthroplasty
Open reduction internal fixation of the femoral neck/head
Surgical dislocation of the hip
Open reduction of congenitally dislocated hip
Labral repair
Femoral or acetabular osteo-/chondroplasty
Irrigation and debridement of septic hip
Biopsy
Excision of ectopic bone
Hip resurfacing
Osteotomies for the treatment of developmental hip dysplasia
Decompression and bone grafting for avascular necrosis of the femoral head
Hip arthrodesis

Massachusetts General Hospital established it as the "Smith-Petersen" approach. Since then many surgeons have championed and modified the approach for various orthopedic procedures (Table 1). French surgeon Emile Letournel described an extension of the anterior approach for the treatment of acetabular fractures known as the extended iliofemoral approach.

The true Hueter or Smith-Petersen approach utilizes an 8–10 cm incision from 2 to 3 cm lateral to the anterior superior iliac spine (ASIS) toward the lateral patella. The superficial internervous plane is between the sartorius (femoral nerve) and tensor fascia lata (TFL, superior gluteal nerve). The deep internervous plane lies between the rectus femoris (RF, femoral nerve) and the gluteus medius and minimus (GMed and GMin, superior gluteal nerve). Developing the interval between the TFL and sartorius directly places the lateral femoral cutaneous nerve (LFCN) at risk. The LFCN typically crosses under the inguinal ligament medial to the ASIS and courses over the TFL 1.5–5 cm distal to the ASIS [2]. However, many anatomical variants have been described and impairment of the LFCN after anterior approach for total hip arthroplasty has been reported to be up to 14.8 % [3, 4]. Matta [5] has described a modification of the Hueter approach with a more lateral skin incision, which utilizes the same superficial and deep internervous planes but protects against injury to the LFCN.

The primary goal of this chapter is to describe, in detail, the surgical technique of this modified Hueter/Smith-Petersen anterior approach to the hip.

Surgical Technique

The patient can be placed supine on a standard or orthopedic table depending on the procedure and surgeon preference. The surgical side of the patient may also be elevated or the table tilted in a semi-lateral position, which can help in obtaining a lateral fluoroscopic image of the hip. The area between the xiphoid cranially and mid-femur as well as the pubic symphysis to the posterior buttock should be prepped to allow for possible extension of the exposure (Fig. 1a). Smaller areas of interest may be draped for specific procedures (Fig. 1b).

Landmarks and Incision

The iliac crest, greater trochanter, and the ASIS are identified and marked. The incision starts approximately 2 cm lateral and 1 cm distal to the ASIS, depending on the size of the patient. Continue the incision distally and posteriorly for 8–10 cm toward the anterior border of the femur in a direction parallel to the fibers of the TFL. The distal end of the incision typically lies 2–3 cm anterior to the greater trochanter. Sharply traverse the subcutaneous tissue with a scalpel and coagulate any small superficial vessels. Conversely, a transverse incision can be made in the inguinal fold [6], which can improve cosmesis. Dissection will continue down to muscular fascia. With a transverse incision, proximal and distal cutaneous flaps are developed. The translucent fascia overlying the fibers of the TFL is identified (Fig. 2).

Exposure

A soft tissue protector and retractor may be inserted into the wound (Alexis Wound Protector, Rancho Santa Margarita, CA). Identify the

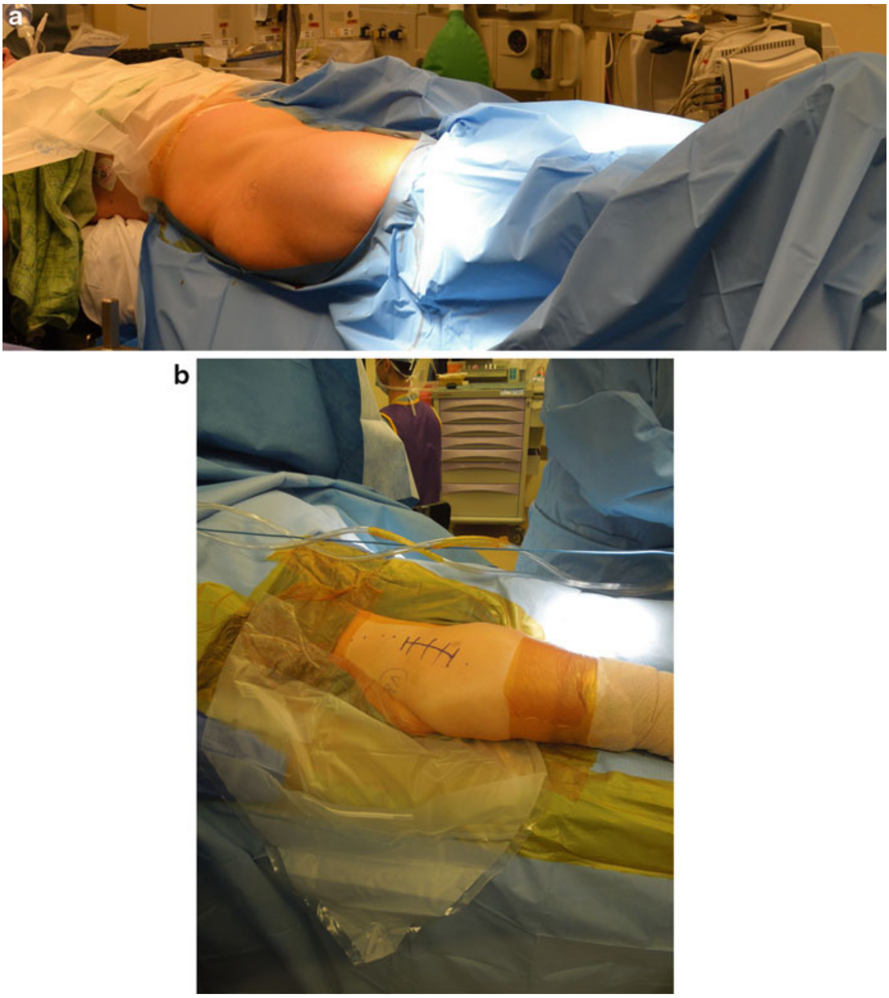


Fig. 1 (**a**) Initial positioning for the anterior approach on the orthopedic table. (**b**) Final field preparation and draping with leg free on radiolucent table

junction of the anterior two-thirds and posterior one-third of the TFL. Make an incision in the fascia over the TFL parallel to its fibers, and this may be extended both distally and proximally beyond the length of the skin incision. The location of the fascial incision over the muscle helps protect the LFCN.

Pick up the anterior flap of the fascia (Fig. 3). Dissect muscle fibers sharply from the fascia or bluntly using finger dissection to delineate the anterior and medial borders of the tensor muscle belly. A medial retractor will retract the rectus femoris. A blunt-tipped retractor can be placed between the superior hip capsule and the gluteus minimus to aid in retracting the TFL. Placement of the retractor directly onto the posterior femoral neck should be avoided in preservation cases. Incise longitudinally the fascia over the lateral rectus, which will facilitate its medial retraction. Split the retinaculum overlying the hip, and the lateral circumflex vessels to the hip are visualized (Fig. 4). At the anterior inferior iliac spine (AIIS), the two heads of the RF can be seen.

The lateral femoral circumflex vessels are now visualized and controlled. Ligate with suture or cauterize the individual branches as inadequate

Fig. 2 Translucent fascia overlying tensor fascia lata (TFL) muscle

Fig. 3 Medial dissection under TFL fascia, mobilizing the muscle laterally

hemostasis will lead to excessive intraoperative or postoperative bleeding. Avoid passing a retractor under the iliocapsularis and RF to further expose the capsule. Lateral retraction of the distal TFL will allow improved visualization of the capsule and proximal vastus intermedius. Relaxation of the TFL and rectus with slight flexion and abduction of the hip can facilitate deep retraction.

Capsulotomy

Some of the adipose tissue on the anterior hip capsule can be removed to better visualize the capsule. The capsulotomy is made parallel to the neck at the junction of the anterior and

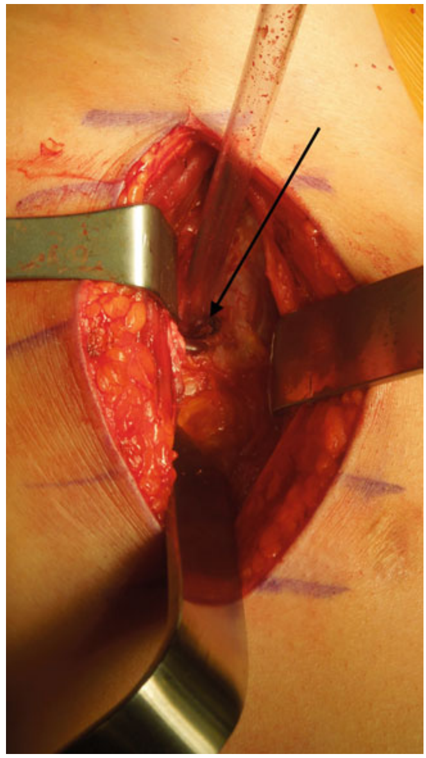


Fig. 4 Lateral femoral circumflex vessels (*arrow*) under deep retinacular fascia

superolateral capsule between the caudal border of the gluteus minimus and lateral iliocapsularis (Fig. 5). Working from distal to proximal along a line from just medial to the anterior tubercle of the greater trochanter to the medial border of the AIIS, the capsule is opened. Extending the capsulotomy medially along the intertrochanteric line at the base of the neck will improve initial intra-articular visualization and help protect the labrum from injury as the capsulotomy is extended toward the acetabular rim. The capsulotomy can be extended along the intertrochanteric line to the lesser trochanter and can be elevated from the acetabular rim medially and cranially under the reflected head to improve exposure. The femoral head can be subluxed for intra-articular access or dislocated to address injuries to the femoral head. Traction (if positioned on an orthopedic table) or a femoral distractor can facilitate this. If greater mobility of the proximal femur will be required, the remainder of the

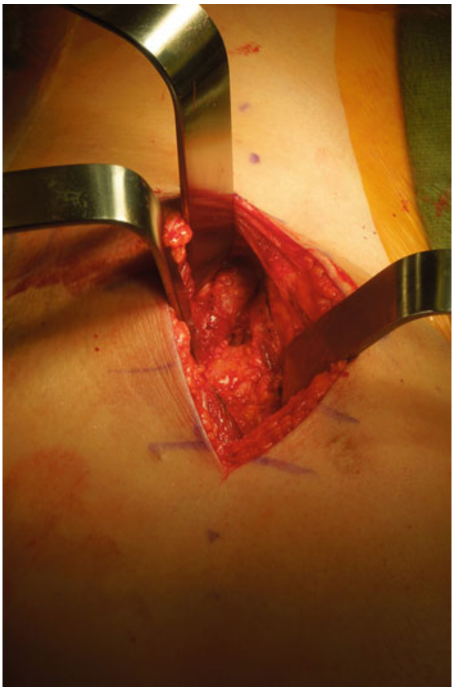


Fig. 5 Interval between gluteus minimus (*left*) and iliocapsularis muscle (*right*) defined for capsulotomy to be performed

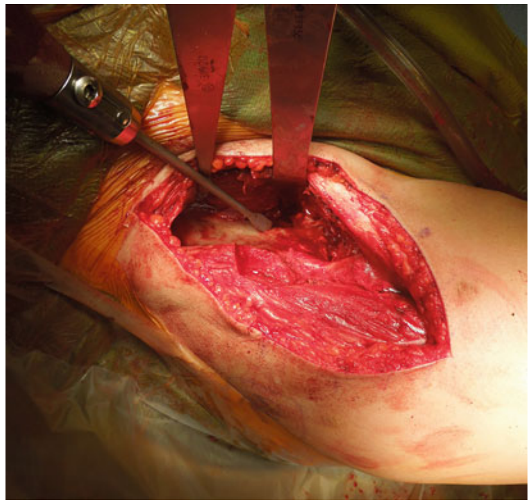


Fig. 6 Intrapelvic extension with ASIS osteotomy

capsule can be released from the greater trochanter, but this is usually reserved for arthroplasty. Retractors can be safely placed onto the medial femoral neck, acetabular rim, and over the posterior superior labrum to further retract the capsule. Release of the RF is rarely necessary unless dislocation is difficult. Proximal extension into the pelvis or outer ilium can be done to gain access to the innominate bone for fractures or osteotomies (Fig. 6) [6]. Osteotomy of the ASIS for proximal extension will maintain soft tissue attachments, viability, and contour of the pelvis (Fig. 7).

Closure

After completion of the intra-articular procedure, the capsule is closed with interrupted absorbable suture. The fascia, subcutaneous tissue, and skin are closed in routine serial fashion.

Conclusion

The Hueter/Smith-Petersen anterior approach to the hip provides exposure to the anterior capsule through the internervous plane of the femoral and superior gluteal nerves. The described approach also avoids the LFCN through a more lateral incision. While other approaches to the hip are widely used, the disadvantages of each provide rationale for the anterior approach. Posterior approaches require splitting the gluteus maximus and tenotomy of the short external rotators and potentially the quadratus femoris. Splitting the gluteus maximus puts the inferior gluteal nerve at risk of injury. On the other hand, the lateral approaches can injure the superior gluteal nerve as the gluteus minimus and medius muscles are split and detached. Injury to the superior gluteal nerve and incomplete healing of the gluteus medius tendon can lead to significant abductor dysfunction and Trendelenburg gait.

However, the anterior approach has known complications, including potential injury to the LFCN. Failure to correctly identify the translucent TFL fascia and the underlying TFL can lead to dissection at the sartorius or even medial to the sartorius at the femoral triangle. This can damage not only the LFCN but also the femoral neurovascular bundle through aberrant medial

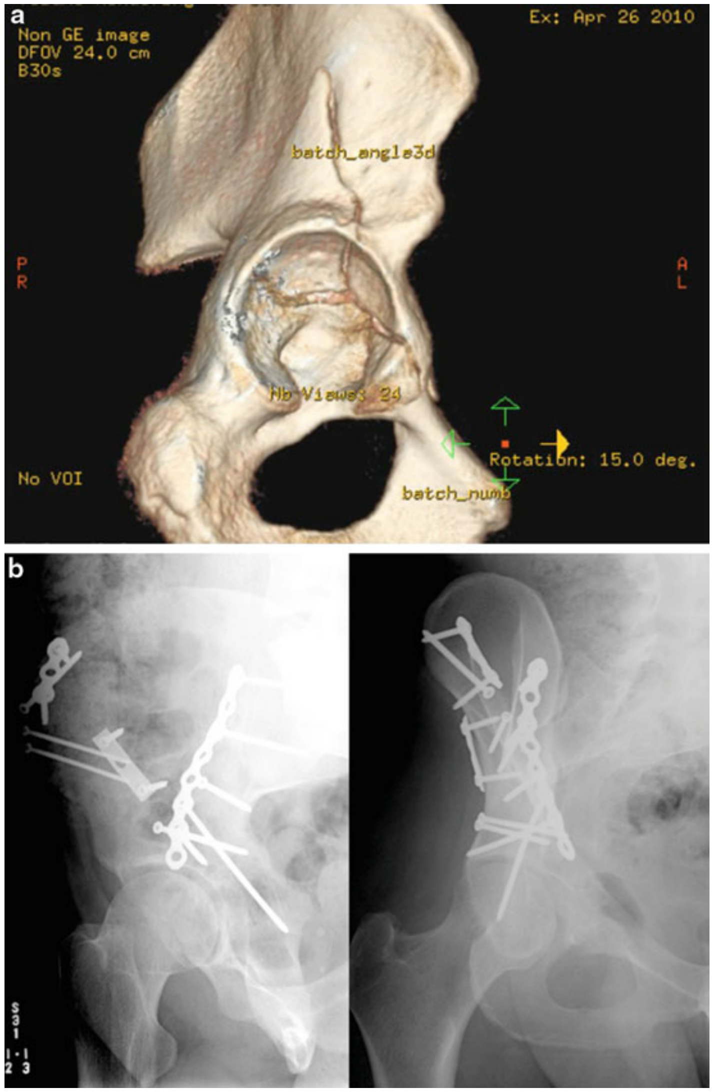


Fig. 7 (**a**–**b**) Anterior posterior hemitransverse variant fracture, with posterior wall, approached through extended Smith-Petersen, exposing both medial and lateral surfaces of the bone

dissection or retractor placement. Intraoperative hemorrhage and postoperative hematoma are also possible complications if the lateral circumflex vessels are not properly identified and ligated. Proximal femoral visualization is also limited with a conventional anterior approach. Lastly, while the approach is muscle sparing, overzealous retraction can damage the muscle bellies of the sartorius, RF, and TFL. Some postulate that excessive retraction of these muscles, particularly the TFL, may lead to the not uncommon postoperative incidence of heterotopic ossification [7].

Summary

The direct anterior approach described by Hueter/Smith-Petersen takes advantage of an internervous plane between the femoral and superior gluteal nerves while not detaching any muscles or tendons. The modification described here utilizes a more lateral incision to avoid the superficial LFCN and exposes the sartorius-TFL interval deep to the TFL fascia. This approach is becoming increasingly common in the use of

THA but serves as a versatile tool in the surgeon's armamentarium in addressing intra-articular hip and periacetabular pathology.

References

1. Rachbauer F, Kain MS, Leunig M. The history of the anterior approach to the hip. Orthop Clin North Am. 2009;40(3):311–20.
2. Victor M, Ropper AH (ed.), Diseases of the peripheral nerves. Adams and Victor's Principles of Neurology. 7th ed. New York, NY: McGraw-Hill. 2001;1370–1446.
3. Bhargava T, Goytia RN, Jones LC, Hungerford MW. Lateral femoral cutaneous nerve impairment after direct anterior approach for total hip arthroplasty. Orthopedics. 2010;33(7):472. PubMed PMID: 20608633.
4. Aszmann OC, Dellon ES, Dellon AL. Anatomical course of the lateral femoral cutaneous nerve and its susceptibility to compression and injury. Plast Reconstr Surg. 1997;100(3):600–4. PubMed PMID: 9283556.
5. Matta JM, Shahrdar C, Ferguson T. Single-incision anterior approach for total hip arthroplasty on an orthopaedic table. Clin Orthop Relat Res. 2005;441:115–24. PubMed PMID: 16330993.
6. Leunig M, Faas M, von Knoch F, Naal FD. Skin crease 'bikini' incision for anterior approach total hip arthroplasty: surgical technique and preliminary results. Clin Orthop. 2013;471(7):2245–52.
7. Barton C, Kim PR. Complications of the direct anterior approach for total hip arthroplasty. Orthop Clin North Am. 2009;40(3):371–5. PubMed PMID: 19576405.
8. Smith-Petersen, M.N. A new new supra-articular subperiosteal approach to the hip joint. J Bone & Joint Surg. 1917. S2–15(8):592–595.

Mini-Open Approach to the Hip

26

Antonia F. Chen, Patrick O'Toole, Joshua Minori, and Javad Parvizi

Contents

Abstract

The mini-open surgical approach is a favorable approach for treating femoroacetabular impingement (FAI) to perform labral refixation, acetabular rim trimming, and femoral osteochondroplasty. It is performed through an internervous plane, minimizes muscle damage, and reduces the traction necessary for accessing the hip joint compared to a hip arthroscopy. Caution must be taken to prevent damage to the lateral femoral cutaneous nerve, as well as the lateral femoral circumflex vessels. The mini-open approach is a very successful approach for providing adequate visualization of the hip joint to allow treatment for hip pathology present from FAI.

Introduction

Surgical approaches for treating femoroacetabular impingement (FAI) include surgical dislocation, hip arthroscopy and the mini-open surgical approach [1]. Of these three approaches, the mini-open approach has gained favor among hip preservation surgeons that also perform total hip arthroplasty (THA) [2, 3]. The mini-open surgical approach is very similar to the direct anterior (DA), or Smith-Petersen, approach for performing a THA, as it uses a smaller skin incision that has also been used to perform irrigation and debridements of a native hip [4]. This approach has the same benefits of a DA approach, as it uses the

A.F. Chen • P. O'Toole • J. Minori • J. Parvizi (✉)
The Rothman Institute, Thomas Jefferson University, Philadelphia, PA, USA
e-mail: antoniachen1@gmail.com; pojotoole@yahoo.com; Joshua.Minori@rothmaninstitute.com; parvj@aol.com

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_26

internervous plane between the femoral nerve (sartorius and rectus femoris muscles) and superior gluteal nerve (tensor fascia lata and gluteus medius muscles). The mini-open approach allows for direct visualization of the femoral head-neck junction without the need for surgical dislocation and the risk of avascular necrosis. There is minimal use of traction in comparison to hip arthroscopy, and there is no use of a traction post, which reduces the risk of pudendal nerve injury and other associated issues with the use of traction. Studies have demonstrated that the use of the mini-open approach to treat FAI improves patient function scores and allows athletes to return to a high level of activity [5, 6]. This chapter provides a step-by-step guide for performing a mini-open hip approach to facilitate access to perform acetabular rim trimming, labral refixation, and femoral osteochondroplasty.

Patient Positioning and Preparation

For proper positioning, the patient is positioned supine on the operating table. Care is taken to ensure that all bony prominences are well padded. The procedure can be performed with or without a gel bump under the hip. If a rectangular gel bump is used, it is placed under the pelvis so that the anterior superior iliac spines are positioned in the middle of the bump from a proximal to distal direction. This allows the operative hemipelvis to be elevated approximately 5° from the horizontal plane. The patient is then moved towards the operative side so that the patient's greater trochanter is at the edge of the operating table. If the patient is excessively tilted after these maneuvers, the table can be counter-tilted to restore alignment. The hip should be flexed and the distal portion of the rectangular bump should be at the distal level of the ischium. A leg holder can be used to keep the foot and leg elevated for skin preparation, and this also facilitates application of a non-sterile U-drape that is placed proximally encircling the groin and the posterolateral thigh. A non-sterile 10 × 10 cm drape is used to drape the area between the two limbs of the U-drape, and a second 10 × 10 cm drape is wrapped around the foot starting in the mid-calf. For the instrument trays, the basic set required for performing acetabular rim trimming, labral refixation, and femoral osteochondroplasty should include two blunt and two sharp Hohmann retractors, a Cobb elevator, nerve hook and a pituitary rongeur.

Approach

After routine prepping and draping, the surgical incision should be marked approximately 1 cm lateral to and 1 cm inferior from the anterior superior iliac spine (ASIS) (Fig. 1). The length of the surgical incision is approximately 4 cm long, which should provide adequate access to the hip joint.

Using a 10 blade scalpel, the incision should be extended through the subcutaneous tissue and bleeding should be controlled by electrocautery. The teeth of a Hibbs retractor can be used on the medial side for retraction, and should be held by one assistant. For deeper subcutaneous tissue, a second Hibbs retractor can be used on the lateral side and should be held by a second assistant. Careful dissection using a scalpel should be performed to avoid damage to the lateral femoral cutaneous nerve and until the tensor fascia lata (TFL) is identified (Fig. 2).

Fig. 1 Mini-open incision. The *circle* is marked around the anterior superior iliac spine (*ASIS*) and the surgical incision starts 1 cm lateral and 1 cm inferior to the ASIS

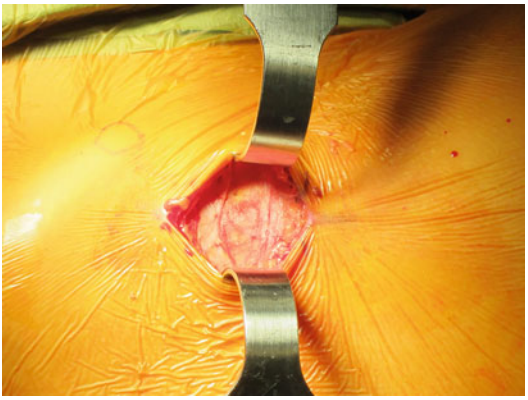


Fig. 2 The fascia of the tensor fascia lata is the first layer of the approach

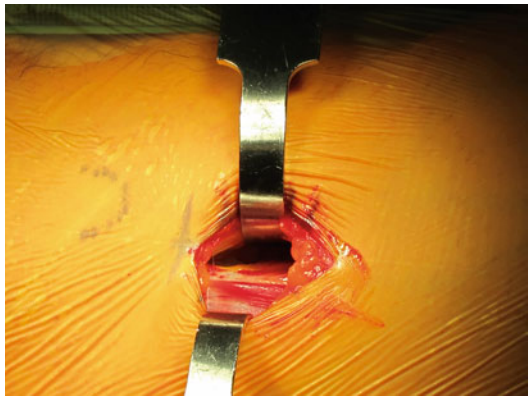


Fig. 4 The second layer of the approach is the first internervous interval between the sartorius and TFL

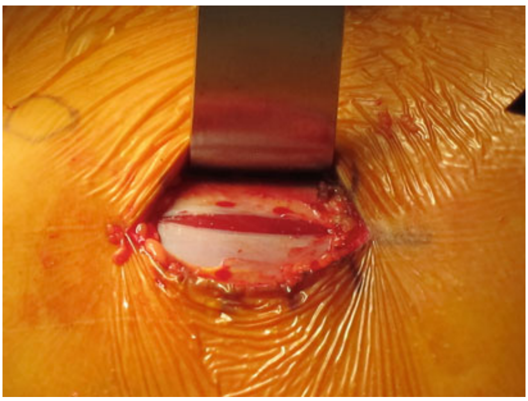


Fig. 3 A vertical incision is carried through the TFL using a 10 blade scalpel

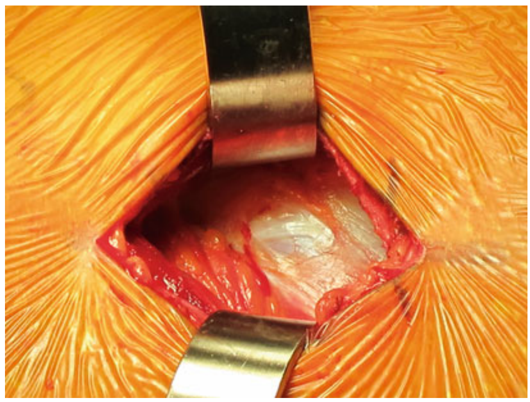


Fig. 5 The silver muscle belly of the rectus femoris denotes the second internervous interval before the hip capsule

The TFL is then cut longitudinally inline with the fibers (Fig. 3) and the index finger is used to sweep underneath the fascia. The Hibbs retractors should be reversed and the hook end should be used to retract the fascia on either side. The interval between the TFL and sartorius should be identified by finding two distinct muscle bellies (Fig. 4). Finger dissection should be performed through this interval and the TFL muscle should be swept laterally and the sartorius should be swept medially. The Hibbs retractors should be replaced deeper with the hook ends to retract the TFL and sartorius.

Since this is the mini-open approach, the ascending branches of the lateral femoral circumflex artery and vein may not be exposed in the distal region of the incision between the TFL and sartorius. If the blood vessels are not visualized, they do not need to be cauterized. If they are exposed, then the ascending branches of the lateral femoral circumflex vessels should be cauterized prior to proceeding, to prevent bleeding that may compromise visualization of the surgical field.

Dissection through the TFL and sartorius are carried out until the silver muscle belly of the rectus femoris can be identified (Fig. 5). The plane between the gluteus medius and rectus femoris can be identified.

Care should be taken to preserve the straight head of the rectus femoris that is attached to the

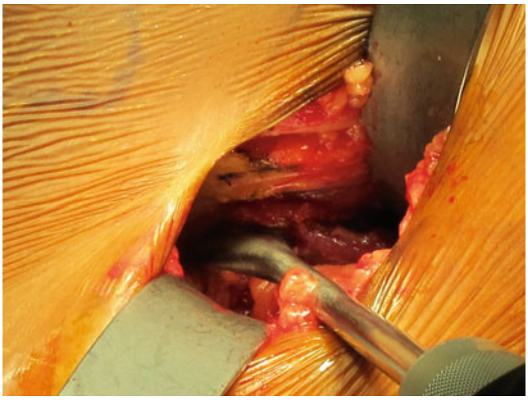


Fig. 6 A Cobb elevator is used to develop a plane above the straight head of the rectus femoris to allow for placement of a lighted superior retractor with a tooth

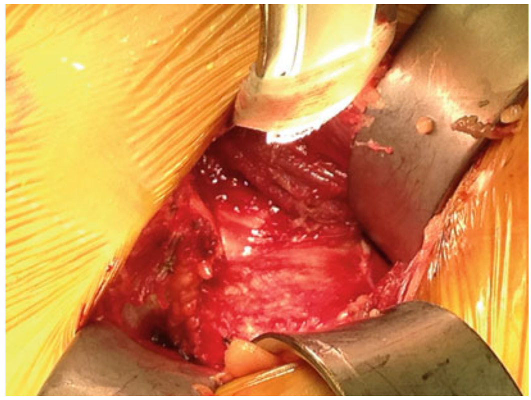


Fig. 7 Visualization of the hip capsule. Note the use of the three main retractors: one long handled blunt Hohmann retractor around the calcar, one long handled blunt Hohmann retractor around the greater trochanter, and one lighted superior retractor above the anterior inferior iliac spine (*AIIS*)

anterior inferior iliac spine (AIIS) and the reflected head that is attached to the superior acetabular brim. Occasionally, there are tight bands of the rectus femoris superiorly that can be released using electrocautery without releasing the entire origin of the muscle. Once the plane between the rectus femoris and gluteus medius is developed, the hip capsule should be visible. To provide adequate visualization of the hip capsule, one long handled narrow blunt Hohmann retractor should be placed around the calcar and another long handled narrow blunt Hohmann retractor should be placed around the greater trochanter. To gain visualization superiorly, a Cobb elevator should be used to develop the plane above the AIIS for placement of a lighted superior retractor with a tooth (Fig. 6).

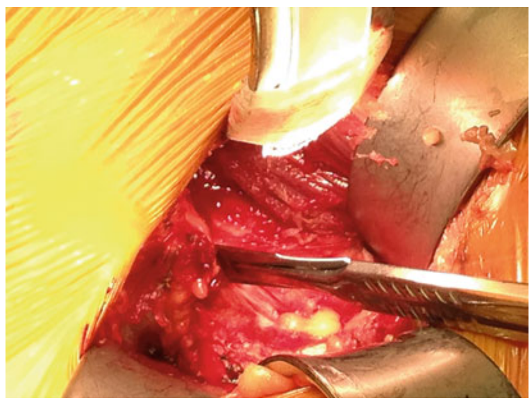


Fig. 8 The first incision of the capsulotomy is made in the vertical plane

Capsulotomy

Once the hip capsule has been adequately exposed (Fig. 7), a capsulotomy should be performed to allow for visualization of the labrum and the hip joint. The capsulotomy should be performed in a T or I shape, depending on the amount of dissection needed to gain adequate view of the surgical field. The initial vertical incision with a long handled 10-blade is through the middle of the capsule (Fig. 8). This is followed by the superior horizontal incision of the T shaped incision (Fig. 9a, b). This must be done very carefully to avoid damage to the labrum, and can be facilitated by resting the handle of the long handled scalpel against the Hohmann retractors. If additionally visualization is required, the inferior horizontal incision may be performed to make an I shaped incision. This is done approximately at the level of the calcar, to only allow access to the lateral femoral neck and not to permit dissection too inferiorly. Once the capsulotomy has been performed, the two long handled narrow blunt Hohmann retractors should be repositioned inside the capsule, with one

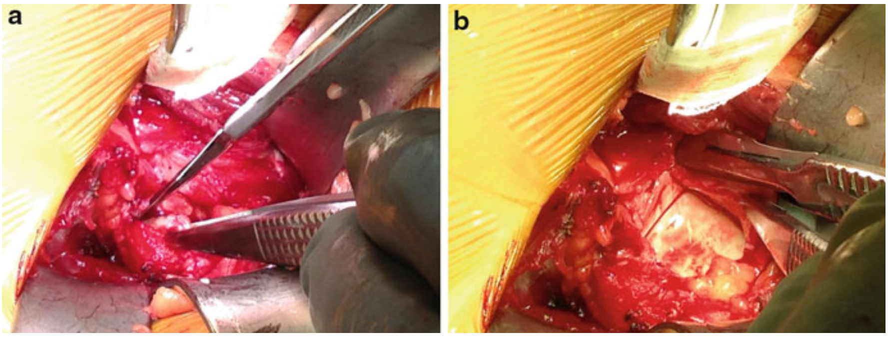


Fig. 9 The capsulotomy is performed in a T or I shaped manner to gain adequate visualization of the labrum and access to the lateral femoral neck to allow for surgical management. The (**a**) lateral flap is elevated, then the (**b**) medial flap is elevated

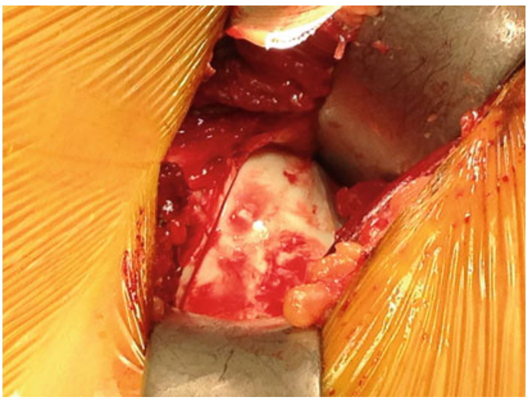


Fig. 10 The completed capsulotomy allows adequate visualization of the labrum, femoral head and acetabulum. The long handled narrow blunt Hohmann retractors should be repositioned under the capsule to provide improved visualization of the surgical field

inferiorly around the calcar and one superiorly within the hip capsule (Fig. 10). This provides a good view of the surgical field, with access to the labrum, acetabulum, femoral head, and lateral femoral neck.

Conclusion

The mini-open approach is a useful surgical approach that utilizes the intranervous plane between the TFL and sartorius muscules, then the gluteus medius and rectus femoris muscles. When dissecting, care must be taken to try to avoid damage to the lateral femoral cutaneous nerve and the ascending branches of the lateral circumflex vessels. When the mini-open approach is performed with a capsulotomy, it provides great visualization of the hip joint that can then permit acetabular rim trimming, labral refixation, and femoral osteochondroplasty.

References

1. Wilson AS, Cui Q. Current concepts in management of femoroacetabular impingement. World J Orthop. 2012;3 (12):204–11.
2. Parvizi J, Huang R, Diaz-Ledezma C, Og B. Mini-open femoroacetabular osteoplasty: how do these patients do? J Arthroplasty. 2012;27(8 Suppl):122–5.
3. Ribas M, Ledesma R, Cardenas C, Marin-Peña O, Toro J, Caceres E. Clinical results after anterior mini-open approach for femoroacetabular impingement in early degenerative stage. Hip Int. 2010;20 Suppl 7: S36–42.
4. Malik AK, Chou DT, Witt JD. Anterior approaches to the hip for the treatment of femoro-acetabular impingement: a cadaveric study. Hip Int. 2010;20(4):482–8.
5. Laude F, Sariali E, Nogier A. Femoroacetabular impingement treatment using arthroscopy and anterior approach. Clin Orthop Relat Res. 2009;467(3):747–52.
6. Cohen SB, Huang R, Ciccotti MG, Dodson CC, Parvizi J. Treatment of femoroacetabular impingement in athletes using a mini-direct anterior approach. Am J Sports Med. 2012;40(7):1620–7.

Surgical Approach for Periacetabular Osteotomy

27

Michael Zlowodzki and Walter Virkus

Contents

Abstract

The surgical technique for the Bernese periacetabular osteotomy was developed by Prof. Reinhold Ganz in 1984 for the treatment of hip dysplasia (Ganz R, Klaue K, Vinh TS, Mast JW. Clin Orthop Rel Res 232:26–36, 1988). The purpose of the surgical approach is to expose the innominate bone in order to enable an osteotomy which allows for a complete detachment of the acetabulum while leaving the posterior 50 % of the posterior column intact. The dissection resembles the well-known Smith-Petersen approach with a few modifications. Anatomical structures at risk during surgical exposure and osteotomy include the lateral femoral cutaneous nerve, the femoral nerve, the sciatic nerve, the obturator artery and nerve, and the medial femoral circumflex artery. This chapter describes in detail the surgical setup, the planes of dissection, the location of the neurovascular structure at risk, and how to avoid complications.

Introduction

The surgical technique for the Bernese periacetabular osteotomy was developed by Prof. Reinhold Ganz in 1984 for the treatment of hip dysplasia [1]. The purpose of the surgical approach is to expose the innominate bone to enable four separate bone osteotomies

M. Zlowodzki (✉) • W. Virkus
Department of Orthopaedic Surgery, Indiana University School of Medicine, IU Health Methodist Hospital, Indianapolis, IN, USA
e-mail: zlowo001@umn.edu; wvirkus@iuhealth.org

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_17

which allow for a complete detachment of the acetabulum from the intact pelvis while leaving the posterior 50 % of the posterior column intact:

1. Ischial osteotomy just inferior to the posterior horn of the acetabulum at the level of the subcotyloid groove
2. Superior ramus osteotomy medial to the iliopectineal eminence
3. Anterior iliac wing osteotomy
4. An ischial osteotomy posterior to the acetabulum through the quadrilateral surface dividing the posterior column of the acetabulum in half by connecting the iliac wing osteotomy with the infra-acetabular ischial osteotomy

(see Chap. 45, "▶ Surgical Technique: Periacetabular Osteotomy")

Setup and Positioning

After an optional insertion of an epidural catheter for postoperative pain control as well as an insertion of a Foley catheter, the patient is positioned supine on a flattop radiolucent table. Typically, general anesthesia is used. The combination of general and epidural anesthesia optimally allows for a hypotensive anesthetic technique, which significantly reduces blood loss. Some surgeons like to use a bump under the affected hip, while others prefer not to use it in order to keep the pelvis parallel to the floor. This facilitates the intraoperative assessment of the orientation of the osteotomized acetabulum. The surgeon requires at least one and ideally two surgical assistants. Intraoperative imaging is required, and a C-arm is set up on the contralateral side perpendicular to the patient's body at the level of the pelvis. The operative leg is draped free to allow for hyperflexion of the hip during the procedure, and a foot rest can be fixed to the table prior to draping to facilitate hyperflexion of the hip if preferred by the surgeon.

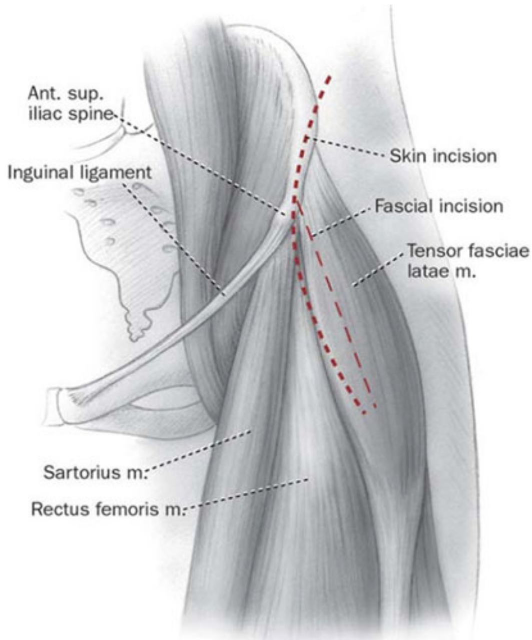


Fig. 1 Overview of skin incision and the underlying muscles (Figure taken with permission from "Periacetabular osteotomy in the treatment of severe acetabular dysplasia. Surgical technique." Clohisy JC, Barrett SE, Gordon JE, Delgado ED, Schoenecker PL. J Bone Joint Surg Am. 2006 Mar; 88 Suppl 1 Pt 1:65–83)

Skin Incision

The landmarks for the incision include the anterior superior iliac spine (ASIS), the iliac crest, and the sometimes palpable interval between the tensor fasciae latae muscle belly and the sartorius muscle belly (Figs. 1 and 2).

The original incision described by Ganz uses the skin incision of the Smith-Petersen approach [1, 2]. The incision is started along the gluteal tubercle of the iliac crest and then turned at the level of the ASIS to follow the course of the underlying tensor fascia latae muscle fibers. Proximally, the incision is made over the iliac crest and just lateral to the ASIS to avoid a painful surgical scar formation over bony prominences and to avoid injuring the lateral femoral cutaneous nerve which runs just medial to the ASIS deep to the inguinal ligament. Some surgeons advocate at this stage to identify and dissect out the lateral

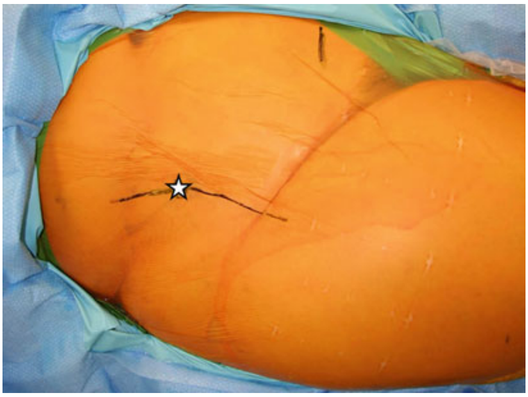


Fig. 2 **Skin incision.** The skin incision is located over the iliac crest proximally and the palpable anterior border of the tensor fascia latae muscle belly distally. *Star*, anterior superior iliac spine; *black line*, skin incision

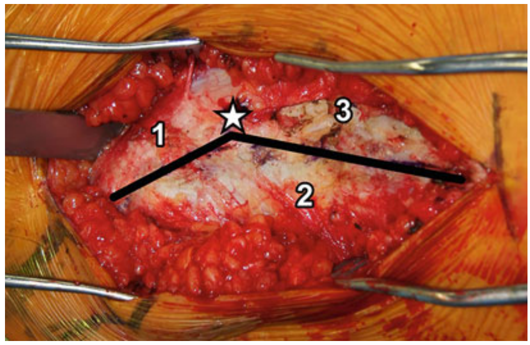


Fig. 3 **Fascial incision.** Proximally, the facial incision is located over the iliac crest where the abdominal muscle fascia meets the origin of the tensor fascia latae. Distally, the fascial incision lies over the anterior border of the tensor fascia latae muscle belly. *Star*, anterior superior iliac spine; *black line*, fascial incision; *1*, external oblique abdominal muscle; *2*, tensor fasciae latae; *3*, sartorius femoris

femoral cutaneous nerve to avoid its entrapment during closure of the incision; however, it is sufficient to keep the dissection lateral to it so that the nerve remains uninjured in the more medial soft tissues.

Alternatively in thin patients, the incision does not have to angle at the ASIS but can remain straight and follows the course of the inguinal ligament just distal to it. This type of incision allows for a more cosmetic “bikini line” scar but makes the subsequent steps of the surgical approach more difficult especially in more muscular or obese patients.

Deep Dissection

Proximally, the interval between the origin of the tensor fasciae latae muscle and the abdominal muscle aponeurosis consistent of the external oblique, internal oblique, and transverse muscle layers is identified (Fig. 3). As the abdominal muscle aponeurosis wraps around the iliac crest, this interval is located proximally just distal to the iliac crest and at the level of the ASIS right over the iliac crest. The interval is incised down to the periosteum, and dissection is carried out towards the inner iliac table just across the iliac crest leaving the origin of the tensor fasciae latae untouched. In the early developmental phases of the this approach, Ganz et al. have detached the origin of the tensor fasciae latae of the anterior part of the outer pelvic table [1]; however, they have later on abandoned it as it was deemed unnecessary [3, 4].

Care is taken not to cut into the external oblique muscle belly which forms the superficial layer of the abdominal muscle aponeurosis and to remain subperiosteal to avoid unnecessary bleeding. At this point the dissection of the iliacus muscle from the inner pelvic table is continued. As the periosteum thins out on the inner pelvic table, the dissection of the iliacus origin from the inner pelvic table can be associated with substantial blood loss from blood vessels from the muscle belly as well as the nutrient artery of the ilium which is a branch of the superior gluteal artery and enters the ilium approximately 2 cm anterior to the sacroiliac joint and 2 cm proximal to the pelvic brim [5, 6]. Inevitably the nutrient vessel of the ilium is severed and bone wax is necessary for hemostasis. Dissection is carried out along the inner table of the pelvis until the pelvic brim is visualized. This area can be packed with lap pads to allow hemostasis to occur while the more distal dissection is completed.

Next, further distal dissection is performed. The fascia over the tensor fascia lata muscle belly is incised just posterior to the anterior edge

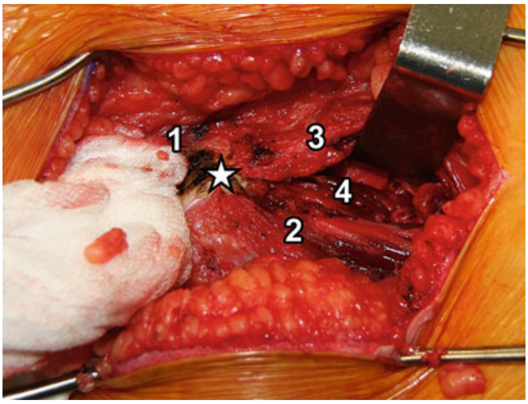


Fig. 4 Deep muscular dissection. Dissection is performed between the tensor fascia latae laterally and the sartorius medially. The sartorius is taken off the anterior superior iliac spine to expose the straight head of the rectus and the iliopsoas. *Star*, anterior superior iliac spine; *black line*, fascial incision; *1*, external oblique abdominal muscle; *2*, tensor fasciae latae; *3*, sartorius femoris; *4*, straight head of rectus femoris

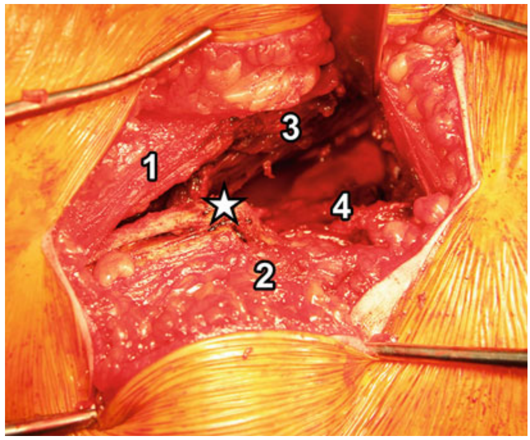


Fig. 5 Exposure of pubic ramus. The pubic ramus is exposed by retracting the sartorius and iliopsoas muscles. *Star*, anterior superior iliac spine; *black line*, fascial incision; *1*, external oblique abdominal muscle; *2*, tensor fasciae latae; *3*, sartorius femoris; *4*, straight head of rectus femoris

of the muscle belly. The tensor fascia lata muscle belly can sometimes be difficult to identify, and it is advisable to err posteriorly. The interval between the sartorius and the tensor fasciae latae is easier to identify proximally near the ASIS. A helpful landmark is a leash of vessels at the posterior aspect of the tensor fasciae latae muscle belly which pierces the fascia to supply the overlying skin [7]. The fascia lata itself is posterior to the tensor fasciae latae muscle belly. The dissection is too posterior if the surgeon encounters increased bleeding or visualizes the fascia lata at this stage. After proper incision of the fascia over the tensor fasciae latae at its anterior edge, the muscle belly is retracted laterally within its sheath. Proximally, a tissue sleeve including the sartorius and the inguinal ligament is dissected off the ASIS (Fig. 4). Alternatively, as originally described by the ASIS can be osteotomized with the origin of the sartorius and the inguinal ligament. The fascia at the floor of the tensor sheath is incised exposing the rectus femoris. The sartorius is retracted medially fully exposing the rectus. Dissection between the rectus femoris and the sartorius is carried out (Fig. 4). After release of the sartorius, exposure of the inner table of the supraacetabular region, iliopectineal eminence, and superior pubic ramus continues from lateral to medial (Fig. 5). It is important to remove all periosteal and soft tissue connections to the brim of the acetabulum, quadrilateral plate, and proximal portion of the superior ramus; otherwise, these will interfere with the mobility of the fragment after the osteotomies are performed. The origin of the straight head of the rectus from the anterior inferior iliac spine is visualized. Medial to the rectus femoris, the iliopsoas is visualized as it comes across the pelvic brim. The fascia overlying the psoas is carefully incised to allow increased mobilization of the psoas. This fascia should be divided under direct visualization distally, as the femoral nerve will lie directly under the fascia. At this point the hip is hyperflexed and adducted to relax the iliopsoas muscle. The iliopsoas has contributing muscle fibers that originate from the anterior hip capsule. Those muscle fibers have been named the iliocapsularis muscle [8]. Dissection is carried out medial to the straight head of the rectus femoris and lateral to the iliocapsularis and iliopsoas. The iliocapsularis muscle fibers are dissected off their origin at the anterior hip capsule. Dissection is carried out between the hip capsule and the iliopsoas proximal to the crossing fibers of the obturator externus down to the subcotyloid groove of the ischium just inferior to the posterior horn of the acetabulum. This is done somewhat blindly and is most easily performed by directing a curved Mayo scissors between the capsule over the femoral neck and the psoas until the ischium is encountered. This is typically deeper and more medial than expected. Fluoroscopic guidance is often helpful in locating the anterior edge of the ischium for those with less experience in the procedure. Care is taken not to stray medially as the obturator neuromuscular bundle is close as it exits the inner pelvis underneath the obturator canal and pierces the obturator membrane. In a study of 29 cadaveric hemipelvises, the distance between the inferior ischial osteotomy site and the obturator artery has been shown to be an average of 36mm with a minimum of 22 mm [9]. It is also important to not dissect distal to the cephalad margin of the obturator externus in order to not jeopardize the medial femoral circumflex artery which constitutes the main blood supply to the femoral head [10]. Optionally, the straight head of the rectus femoris muscle can be taken off the anterior inferior iliac spine to facilitate the exposure; however, in our opinion that is not routinely necessary. It can be useful to perform a capsulotomy to assess the labrum and the femoral neck offset for possible impingement after the acetabular correction.

Anatomical Structures at Risk During Exposure

In general, the described approach for the Bernese periacetabular osteotomy is safe if the surgeon is aware of and protects the following anatomical structures:

1. Lateral femoral cutaneous nerve during exposure and closure
2. Obturator neurovascular bundle during dissection for the inferior ischial cut and the superior pubic ramus cut
3. Medial femoral circumflex artery during dissection for the inferior ischial cut
4. Femoral nerve during retraction for the superior pubic ramus exposure
5. Sciatic nerve during ischial cuts (see Chap. 45, "▶ Surgical Technique: Periacetabular Osteotomy")

In a review of 1,760 patients at five institutions, 36 patients (2.1 %) developed a sciatic (1.6 %) or femoral nerve (0.5 %) deficit. Full recovery occurred in 17/36 patients at an average of 5.5 months postoperatively. All cases of none or incomplete recovery involved the sciatic nerve [11]. In a consecutive series of 508 cases by Ganz, postoperative symptoms related to the lateral femoral cutaneous nerve occurred in approximately 30 % of patients [3]. There was one case of femoral head osteonecrosis in a patient who underwent a periacetabular and femoral osteotomy [3]. To our knowledge, obturator nerve injuries have not been reported in literature. Pring et al. used intraoperative electromyographic monitoring in a consecutive series of 140 patients and reported no postoperative obturator nerve injuries [12].

Avoiding Pitfalls

1. Place the skin incision just lateral to the anterior superior iliac spine to avoid injuring branches of the lateral femoral cutaneous nerve and to avoid a painful scar over the anterior superior iliac spine.
2. Place the distal fascial incision over the muscle fibers of the tensor fascia latae, and retract the muscle fibers within its fascial sheet as opposed to dissecting more anterior between the sartorius and tensor fasciae latae.
3. Remain subperiosteal when exposing the superior pubic ramus, and protect the obturator neurovascular bundle with retractors like blunt Hohmanns or Crezos within the obturator canal at the inferolateral aspect of the superior pubic ramus.
4. Flex and adduct the hip and minimize traction on the femoral nerve during superior pubic ramus exposure.
5. Adduct the leg during both ischial osteotomies to increase the distance of the osteotomies to the sciatic nerve.

Summary

In summary, the surgical approach for periacetabular osteotomy is safe as long as the surgeon has a good knowledge of pelvic anatomy and is aware of the neurovascular structures at risk.

References

1. Ganz R, Klaue K, Vinh TS, Mast JW. A new periacetabular osteotomy for the treatment of hip dysplasias. Technique and preliminary results. Clin Orthop Rel Res. 1988;232:26–36.
2. Smith-Petersen MN. Approach to and exposure of the hip joint for mold arthroplasty. J Bone Jt Surg. 1949;31A(1):40–6.
3. Hussell JG, Rodriguez JA, Ganz R. Technical complications of the Bernese periacetabular osteotomy. Clin Orthop Rel Res. 1999;363:81–92.
4. Leunig M, Ganz R. Evolution of technique and indications for the Bernese periacetabular osteotomy. Bull NYU Hosp Jt Dis. 2011;69(Suppl 1):S42–6.
5. Hempfing A, Leunig M, Notzli HP, Beck M, Ganz R. Acetabular blood flow during Bernese periacetabular osteotomy: an intraoperative study using laser Doppler flowmetry. J Orthop Res. 2003;21 (6):1145–50.
6. Beck M, Leunig M, Ellis T, Sledge JB, Ganz R. The acetabular blood supply: implications for periacetabular osteotomies. Surg Radiol Anat. 2003;25(5–6):361–7.
7. Nogler M. The direct anterior approach to the hip. Mahwah: HSC Press; 2011.
8. Ward WT, Fleisch ID, Ganz R. Anatomy of the iliocapsularis muscle. Relevance to surgery of the hip. Clin Orthop Rel Res. 2000;374:278–85.
9. Kamada S, Naito M, Shiramizu K, Nakamura Y, Kinoshita K. Is the obturator artery safe when performing ischial osteotomy during periacetabular osteotomy? Int Orthop. 2011;35(4):503–6.
10. Gautier E, Ganz K, Krugel N, Gill T, Ganz R. Anatomy of the medial femoral circumflex artery and its surgical implications. J Bone Jt Surg Br. 2000;82 (5):679–83.
11. Sierra RJ, Beaule P, Zaltz I, Millis MB, Clohisy JC, Trousdale RT. Prevention of nerve injury after periacetabular osteotomy. Clin Orthop Rel Res. 2012;470(8):2209–19.
12. Pring ME, Trousdale RT, Cabanela ME, Harper CM. Intraoperative electromyographic monitoring during periacetabular osteotomy. Clin Orthop Rel Res. 2002;400:158–64.

Complications with Hip Arthroscopy and Open Hip Surgery

28

Joshua D. Harris, Christopher M. Larson, and Shane J. Nho

Contents

Abstract

Recognition of femoroacetabular impingement as a potential precursor to hip osteoarthritis has led to the development of both open and arthroscopic hip preservation surgery. Successful short- and midterm clinical outcomes have been reported following hip preservation surgery. Improvements in technique and instrumentation have led to a dramatic increase in the number of surgeons performing hip arthroscopy and the number of cases performed internationally. However, there is a significant learning curve associated with hip arthroscopy. Although the rate of minor complications is low (7.5 %), it is largely related to the learning curve. The two most common minor complications are iatrogenic chondrolabral injury and temporary neuropraxia. Open surgical hip dislocation permits a 360° view of the femoral head and acetabulum but requires a larger incision, greater soft tissue dissection, and a trochanteric osteotomy. Although the rate of minor complications is reportedly higher following open surgical hip dislocation due to the occasional development of painful hardware requiring removal, the rate of major complications is less than 1 % in both open and arthroscopic hip preservation surgery. Thus, both open and arthroscopic hip preservation surgeries appear to be safe. Lack of clarity in reporting complications within orthopedic surgery has spurred academic hip surgeons to adapt and test a general surgery-validated complication reporting system for use in hip preservation.

J.D. Harris (✉)
Houston Methodist Center for Orthopaedics & Sports Medicine, Houston, TX, USA

Rush University Medical Center, Chicago, IL, USA
e-mail: joshuaharrismd@gmail.com

C.M. Larson
Minnesota Orthopedic Sports Medicine Institute, Twin Cities Orthopedics, Edina, MN, USA
e-mail: chrislarson@tcomn.com

S.J. Nho
Department of Orthopaedic Surgery, Rush University Medical Center, Chicago, IL, USA

Midwest Orthopaedics at Rush, Hip Preservation Center, Rush University Medical Center, Chicago, IL, USA
e-mail: shane.nho@rushortho.com

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_28

Introduction

Hip preservation surgery encompasses both arthroscopic and open non-arthroplasty approaches. The role of hip arthroscopy has been rapidly evolving for the treatment of a variety of hip disorders including femoroacetabular impingement (FAI) and labral tears [1–3]. Recognition of the steep learning curve associated with hip arthroscopy and new techniques for managing hip disorders has led to better recognition and increased efforts to avoid complications [4–6]. The prevalence of complications associated with arthroscopy has been reported to be 8.1 % (7.5 % minor; 0.58 % major) in a comprehensive systematic review of over 6,000 subjects [7]. Iatrogenic chondrolabral injury and temporary neuropraxia were the two most common minor complications. The reoperation rate was 6.3 %, and the most common reason for reoperation was conversion to total hip arthroplasty. Minor complications and reoperation rates were directly related to the learning curve of hip arthroscopy. However, there have been no published prospective studies that specifically and comprehensively assess complications at predetermined time points. This has prompted a prospective analysis performed by surgeons at different institutions in the ANCHOR study group (Academic Network of Conservational Hip Outcomes Research) that should optimally determine the true rate of complications after hip arthroscopy using a validated and reliable classification system [8].

Despite the dramatic increase in the number of arthroscopic hip procedures performed, there are clearly indications for open hip preservation techniques (e.g., surgical hip dislocation and mini-open anterior approach) that are largely based on the complexity of hip pathomorphology and ability to access and correct these regions arthroscopically. Although open approaches are more invasive than arthroscopy and have their own inherent unique complications, an extensive degree of soft tissue trauma can result from arthroscopic procedures with improper technique. The incidence of complications and reoperations has recently been reported comparing arthroscopic and open approaches [9]. The rate of reoperation following surgical dislocation was 41 %, which was significantly greater than that of arthroscopic-assisted mini-open (19 %), mini-open (10 %), and arthroscopy (3 %). Ninety-five percent of reoperations following surgical dislocation were for painful hardware removal. Although there were significantly more temporary nerve palsies following arthroscopy (1.7 %) versus surgical dislocation (0.17 %), they are still uncommon. Other complications after arthroscopic and/or open hip preservation surgery include heterotopic ossification, avascular necrosis, fluid extravasation, infection, instability, femoral neck fracture, venous thromboembolism, among others.

Surgical Anatomy

The hip joint is deep, with a highly congruent articulation between the femoral head and acetabulum and a thick capsuloligamentous and muscular covering. Thus, access via arthroscopy is technically more demanding compared to knee or shoulder arthroscopy. Access via open surgical approaches requires larger degrees of soft tissue dissection and mobilization with the need for trochanteric osteotomy for surgical hip dislocation. Any surgical approach to the hip mandates that the surgeon be comfortable with the pathoanatomy being treated to avoid complications and persistent disability from residual deformity. Protection of the medial femoral circumflex vessels and terminal vessels reduces the risk of avascular necrosis. The risk of neural injury can be minimized with safe arthroscopic portal placement, reduced magnitude and duration of traction during arthroscopy, and meticulous layer-by-layer dissection with open incisions. Furthermore, a degree of capsulotomy is required to access FAI deformities, and failure to close capsulotomies in certain situations may result in postoperative instability [10, 11].

Strict adherence to appropriate surgical indications may improve postoperative outcomes. Given that there is a high prevalence of abnormal radiographic findings suggestive of FAI in asymptomatic patients, understanding the various pain

Table 1 Layer concept of hip anatomy and pathoanatomy [12]

Layer	Anatomy	Pathoanatomy
I Osteochondral	Femoral head	Cam impingement
	Acetabulum	Pincer impingement
		Sub-spine (AIIS) impingement
II Inert	Joint capsule	Instability
	Labrum	Labral tear, degeneration, ossification
	Ligamentum teres	Ligamentum teres tear
III Contractile	Musculature crossing hip	Muscle strain
	Musculature crossing lumbosacral spine	Muscle tear
	Musculature crossing pelvic floor	Tendinopathy
IV Neuromechanical	Neurovascular structures	Nerve injury
	Axial/appendicular coordination and mechanics	Spine and lower extremity malalignment
		Pain syndromes

Table 2 Potential nerves injured and mechanisms during hip arthroscopy

Nerve	Mechanism
Pudendal	Pressure due to perineal post
Lateral femoral cutaneous	Direct injury due to portal placement
Common peroneal	Traction
Sciatic	Traction or portal placement
Femoral	Traction or portal placement
Superior gluteal	Portal placement

generators around the hip is paramount with regard to patient selection. The "layer concept" allows the hip surgeon to understand the pathology around the hip that may contribute to a patient's pain (Table 1) [12]. Unique complications encountered during arthroscopy are generally iatrogenic and related to the learning curve of the technique. These include, but are not limited to, iatrogenic chondrolabral injury, various motor and sensory neuropraxias (Table 2), skin damage due to excessive traction against the perineal post, and traction injuries to the foot and ankle. Complications encountered during open hip surgery are painful hardware, greater trochanteric pain syndrome, greater trochanteric osteotomy delayed/nonunion, heterotopic ossification (not unique to open surgery), avascular necrosis of the femoral head (although no reported cases in the literature for treatment of FAI with surgical hip dislocation), femoral neck fracture (not unique to open surgery), infection, and excessive blood loss.

Complications

Iatrogenic Chondrolabral Injury

The overall incidence of iatrogenic chondral and labral injury during hip arthroscopy is 3.8 % and 0.9 %, respectively, but have been reported to be as high as 20 % and 20 %, respectively [7, 13]. To obtain joint access, sharp instrumented joint entry is required for visualization, instrumentation, and mobilization. Unintentional injury to the labrum or articular cartilage may occur during initial portal placement from spinal needle entry, dilation, cannulation, or capsulotomy. During the early learning curve of hip arthroscopy, the rate of iatrogenic chondrolabral injury is greater with earlier time points [5]. Although there is published literature demonstrating that iatrogenic labral punctures have no significant effect on short-term clinical outcome [13], various studies have shown improved results with labral preservation compared to excision/debridement, and longer-term studies might shed further light on this subject [14–16]. Other recent investigations offer techniques to achieve a very low rate of chondrolabral injury (Table 3) [17, 18]. The latter recommend positioning the hip in mild flexion (15–20°), internal rotation, adduction, and traction to break the suction seal and achieve approximately 10 mm of distraction (Fig. 1). The safety of a blind anterolateral portal usually makes it the initial portal placed. A

Table 3 Step-by-step technique to reduce risk of iatrogenic chondrolabral injury [17]

Large-bore spinal needle joint entry with the bevel facing up to avoid the labrum
Stylet removed to permit an air arthrogram image
Stylet reinserted and the needle brought just outside of capsule
Fluoroscopically confirm needle outside of arthrogram
Reinsert needle back into joint with bevel facing labrum
As soon as "pop" is felt (penetration of capsule), needle rotated 180° to avoid femoral head articular cartilage
Stylet removed and nitinol wire placed intra-articular
Needle removed, followed by dilation, cannulation, and arthroscope insertion

70° arthroscope is used to directly visualize extra-labral anterior portal placement. The arthroscope is switched to the anterior portal to verify that the anterolateral portal is extra-labral. The arthroscope is switched back to the anterolateral portal, and a transverse interportal capsulotomy is made. This step requires precision to avoid labral and chondral injury and to permit capsulotomy closure at the end of the procedure. Thus, the interportal capsulotomy should be made 5–10 mm from the labrum and 2–4 cm long, from approximately 10 to 2 o'clock for a left hip (Fig. 2), but the interportal capsulotomy may need to be extended depending on the size of the pincer deformity. Diagnostic arthroscopy of the central compartment can then be performed safely. Acetabuloplasty rim trimming and labral treatment are performed with the hip in traction. In cases with excessive rim over-coverage or in the presence of a large hypertrophic labrum, there is greater risk of iatrogenic chondrolabral injury, and making the capsulotomy from outside in or beginning in the peripheral compartment might allow for safer entry into the central compartment under direct visualization.

During labral refixation or reconstruction, the surgeon must be cognizant of the appropriate drill angle for anchor placement to avoid penetration through the acetabular cartilage into the joint. Using three-dimensional acetabular models of computed tomography scans of 20 cadaveric hips, the acetabular rim angle was defined and evaluated [19]. This angle quantifies the amount of acetabular bone available for drill bit and suture anchor penetration and creates a safety margin for the surgeon.

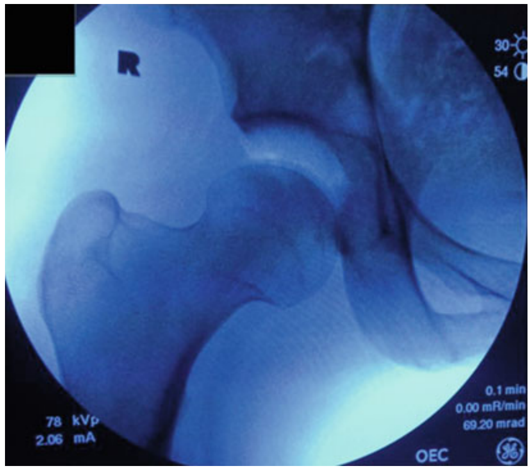


Fig. 1 Intraoperative *C*-arm fluoroscopy of a right hip in the supine position. A vacuum phenomenon is demonstrated after the suction seal is broken with application of traction

Using drill bits of length between 10 and 25 mm and acetabular rim trimming amounts of 0, 2.5 and 5.0 mm, the investigation demonstrated that clock position, drill depth, and amount of rim trimming all had significant effects on the acetabular rim angle. The angle was greatest at 2 o'clock but smallest at 3 o'clock. While greater drill depth significantly reduced the rim angle, greater amounts of rim trimming significantly increased rim angle. Thus, anterosuperiorly, the surgeon must take care in drilling the minimum amount necessary to insert the anchor, especially near the 3 o'clock position. Ultimately making the portals used for placing anchors further distal typically gives a better angle for drilling and anchor placement with less risk for penetration of the acetabular articular cartilage. Beyond drilling and anchor placement, the surgeon must pass suture around or through the labrum. If suture retrieval is lost, this creates an opportunity for multiple passes of the suture-passing device through the labrum, which can potentially lead to biological disruption.

Iatrogenic Instability

There have been nine reported cases of post-arthroscopy hip dislocation [4, 20–25]. Due to publication bias, this is likely a significant

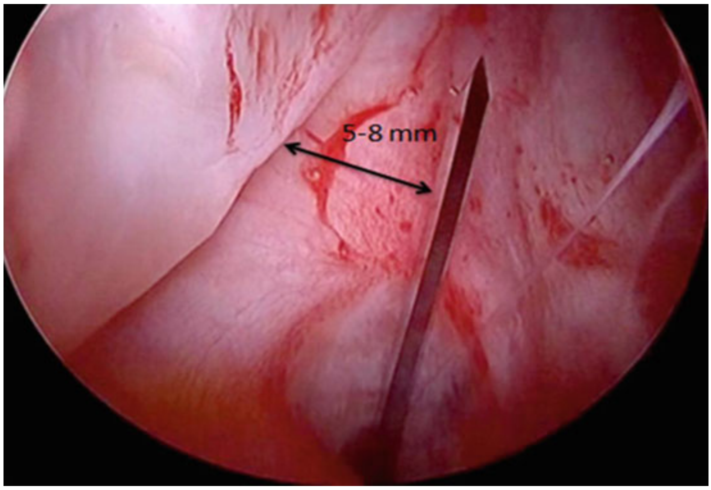


Fig. 2 Interportal capsulotomy creation may be made 5–8 mm from the acetabular labrum with an arthroscopic scalpel. This amount of acetabular side capsule permits tissue for capsular repair or plication at the conclusion of the case. The patient is in the supine position, with the right hip being viewed from the anterolateral portal

underestimate of the true incidence of instability following hip arthroscopy [7]. In addition, it is likely that a number of patients have persistent disability from subtle instability postoperatively without frank dislocation which is much more difficult to define (Figs. 3 and 4). The risk of postoperative instability is related to the following patient-, hip-, and surgical technique-specific factors: type and size of capsulotomy or capsulectomy without repair, labral resection (versus refixation or repair), overaggressive rim trimming, overall capsular laxity, and psoas tenotomies [10]. Capsulotomy (interportal with or without "T" extension) permits visualization and instrumentation of the central and peripheral compartments. However, the iliofemoral ligament is the strongest of four discrete hip ligaments, and its primary purpose is to restrain external rotation and extension of the hip. This part of the capsule is vital to stability in the latter provocative positions (Fig. 5). Multiple cadaveric biomechanical studies have demonstrated that iliofemoral ligament sectioning results in increased external rotation, extension, and anterior translation [26–28]. Further, no difference exists between the repaired and intact state. Thus, unrepaired capsulotomies have the potential for postoperative instability in some situations, which falls along a spectrum of "microinstability" to frank dislocation [29–32]. Although technically demanding, there are several pearls and pitfalls to assist the surgeon in performing repair or plication (Table 4). Following open hip preservation surgery, instability has not been reported in the literature. In this situation, capsulotomies are, for the most part, repaired once the intra-articular work is complete, reducing the risk of instability. It is imperative for the surgeon to have an understanding of normal acetabular anatomy, hip dysplasia, and dysplastic variants when performing FAI corrective procedures. Excessive rim resections should be avoided in all patients, and the capsule and labrum should be repaired/preserved in borderline dysplastic hips. Psoas tenotomies should also be performed with caution as psoas impingement is frequently seen in the setting of acetabular dysplasia and excessive femoral neck anteversion, both of which can be associated with anterior hip instability. Psoas tenotomies in the presence of anterior instability can further destabilize the hip. Prior studies have reported inferior outcomes after psoas tenotomy in the presence of excessive femoral neck anteversion as well as postoperative hip dislocation after psoas tenotomy and capsulotomy performed arthroscopically [33].

Neurovascular Injury

Nerve or blood vessel injury is uncommon during both arthroscopic and open hip surgery [7, 9]. In hip arthroscopy, the incidence of nerve damage is 1 %, with temporary (recovery room to 4 months following surgery) neuropraxia accounting for

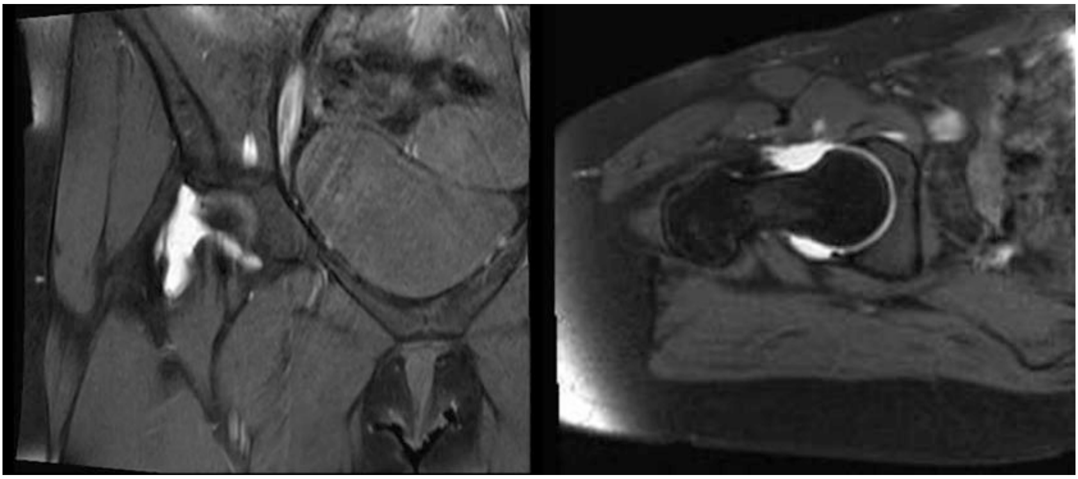


Fig. 3 MRA evidence of capsular defects after hip arthroscopy. *Left*: T2 coronal images demonstrating gadolinium extravasation due to capsular defect. *Right*: T2 axial oblique images demonstrating capsular defect with retraction of the iliofemoral ligament

Fig. 4 Arthroscopic evaluation of capsular defect involving the entire iliofemoral ligament

nearly all cases. The most commonly reported affected nerve is the pudendal (40 %), followed by lateral femoral cutaneous (21 %), sciatic (17 %), common peroneal (17 %), and femoral (4.7 %) [7]. The pudendal nerve (sacral plexus; S2–S4) is both a somatic and autonomic nerve that provides sensory, motor, sympathetic, and parasympathetic function to both male and female external genitalia and sphincters of the bladder and rectum. Nerve compression between the perineal post and the inferior pubic ramus may lead to a neuropraxia, with subsequent perineal numbness, and less commonly difficulty with erection and ejaculation [34]. Urinary and/or fecal incontinence have not been reported following hip arthroscopy, likely due to the relevant innervation of the structures controlling these functions proximal to the zone of compression and the fact that bilateral nerve injury would be required in order to cause incontinence [34]. Further, inferior pubic rami anatomy is unique between genders (steep course of ramus from ischial tuberosity to pubic symphysis in males versus more rounded, gentler, and straighter course in females) [34]. Although the magnitude of traction while using a perineal post has been shown to significantly affect the incidence of pudendal nerve injury, the effect of duration of traction is less clear [35]. Additionally, a lower extremity adduction moment increases the traction force [35] and the force around the post [36]. Therefore, in order to reduce the risk of pudendal nerve compression in the perineum, the following can be helpful: general anesthesia with muscle relaxation in particular when longer traction times are required, sufficient padding of the perineal post, joint distention, and application of the least amount of traction force necessary to distract the hip sufficiently (less than 50 lb) [37]

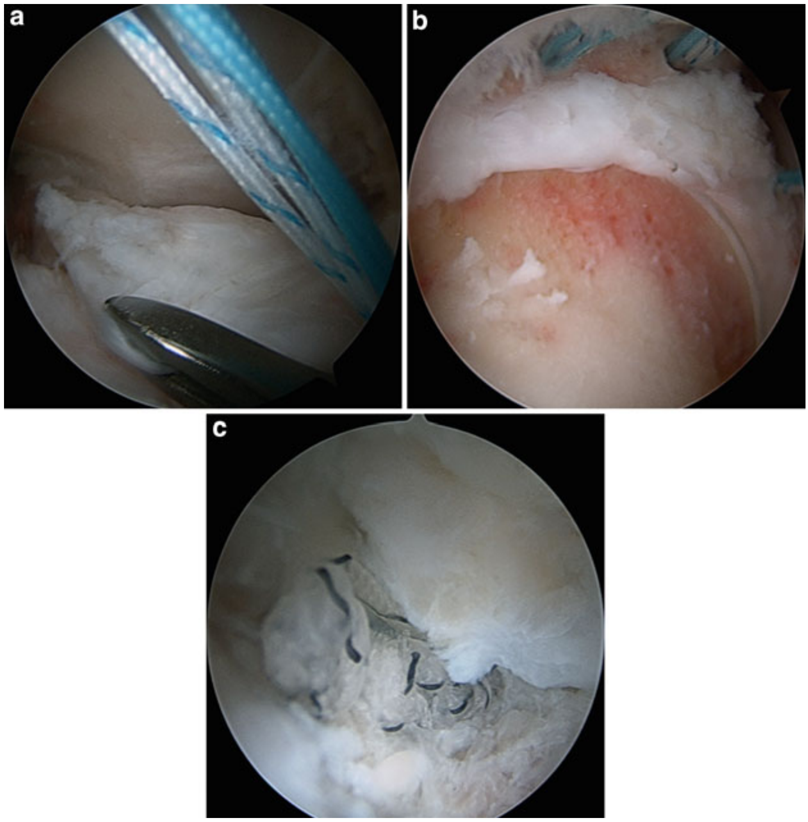


Fig. 5 (**a**) Arthroscopic view of the anterior aspect of the femoral neck. Using an arthroscopic grasper to mobilize the retracted iliofemoral ligament. (**b**) Arthroscopic revision femoral osteochondroplasty with three sets of double-loaded suture anchors for capsular reconstruction. (**c**) Arthroscopic view of the completed capsular reconstruction with suture anchors

Table 4 Pearls and pitfalls for hip capsulotomy and capsular repair or plication. DALA (distal anterolateral accessory) portal; IFL (iliofemoral ligament) [10]

Pearls	Pitfalls
Interportal and "T" capsulotomy	Poor visualization
Enhanced central and peripheral compartment visualizatio	Poor portal placement
Refixation of labrum	Failure to address bony pathology
Suture anchor based; as close as possible to articular margin using DALA portal	Femoral cam and acetabular pincer
Static stability restoration	Stresses labral/capsular repair
Femoral and acetabular osteochondroplasty	Too aggressive capsulectomy
Reduces/eliminates impingement	Prevents complete closure or requires too much tension upon repair that predisposes to stiffness postoperatively
Complete capsular closure	Damaged capsular edges from mechanical shaver devices may preclude secure "bite" with sutures
Avoid aggressive capsulectomy	Avoid iatrogenic articular cartilage damage with passage of tissue penetrator/suture passer devices
Begin closure at distal base of IFL "T'd" capsule and progress proximally toward interportal capsulotomy	Postoperative rehabilitation
Customize degree of plication/"bite" based on patient's ligamentous laxity status	Hip extension or external rotation that stresses capsulolabral repair, with potential disruption
Postoperative rehabilitation	Poor patient selection
Avoid hip extension, external rotation	Dysplasia, hyperlaxity, coxa magna

and adduction necessary to achieve joint visualization for the least amount of time. Some surgeons have even successfully performed arthroscopy without a perineal post [38].

Sciatic nerve (L4–S3) neurophysiologic monitoring during supine arthroscopy has revealed that approximately half (54 %) of subjects experience significant somatosensory evoked potential (SSEP) waveform changes [39]. These changes (signal loss) occur from 7 to 46 min from traction onset and are recovered after 2–15 min of traction release. The latter investigation's time dependency has not been observed during lateral arthroscopy, where SSEP and also tcMEP (transcranial motor evoked potential) have been used to study this phenomenon [40]. The risk of a sciatic nerve event significantly increased 4 % with every 0.45 kg increase in traction ($p = 0.043$), while an increase in traction time did not significantly increase the risk of a nerve event ($p = 0.201$). However, only 7 % of subjects had a clinically detectable postoperative nerve injury. Historically, the 2-h time limit on duration of hip arthroscopy traction was extrapolated from the time-dependent ischemia threshold associated with tourniquet use [41]. It appears that the time dependency of neural injury is related to the perineum and pudendal nerve due to a compression or ischemic effect while in the supine position versus the magnitude dependency of neural injury which is related to the sciatic nerve and axial distraction while in the lateral position [41]. Thus, the surgeon should be mindful of both traction magnitude, especially while lateral, and traction duration, especially while supine [41].

Sufficient padding of the lower extremity in order to reduce neural compression and injury applies to the foot and ankle boot as well, not just the perineum. The superficial peroneal nerve is at risk of compression injury if the foot/ankle is improperly padded and also if the duration/magnitude of traction is excessive [37]. Femoral nerve (L2–L4) injury has also rarely been reported, secondary to either traction, fluid extravasation, or excessively medial placement of the anterior portal [7]. The lateral femoral cutaneous nerve (L2–L3) is at risk with anterior arthroscopic portal placement and open anterior approaches to the hip due to its directly subcutaneous extrafascial location. Although the nerve course is variable, it tends to pass medial to the anterior superior iliac spine (ASIS), under the inguinal ligament, on the superficial surface of sartorius muscle approximately 10–15 mm distal to the ASIS [42]. This places the nerve proper or one of its branches in very close proximity to a deep stab incision for anterior portal placement. Lateral femoral cutaneous nerve injury may cause a spectrum of symptoms ranging from benign or bothersome numbness to debilitating painful dysesthesias. Lateral femoral cutaneous neuropraxia is likely underreported, as the majority may not be noticed by the patient and found on inspection of the anterolateral thigh with nearly all resolving within 6 months post-surgery. The superior gluteal nerve limits the safe zone of the anterolateral and posterolateral portals proximally at approximately 4–7 cm [43, 44]. The sciatic nerve limits the safe zone of the posterolateral portal posteriorly at approximately 2–5 cm [43, 44]. Thus, avoidance of hip external rotation is recommended as the greater trochanter moves posteriorly blocking access for the portal and putting the nerve at greater risk.

The most common vascular structure at risk for injury anteriorly is the ascending terminal branch of the lateral femoral circumflex artery, which may be as close as 10 mm from an anterior portal [44]. However, this artery is commonly ligated during a Smith-Peterson approach just deep to the interval between the tensor fascia lata and sartorius. Due to the fact that the medial femoral circumflex is the largest contributor to the head, lateral femoral circumflex injury or ligation is likely of minimal consequence [45]. During surgical hip dislocation, when preparing to perform greater trochanteric osteotomy, it is critically important to protect the medial circumflex vessels by leaving the external rotators intact. These small muscles should remain attached to the non-osteotomized femur. When dissecting near the insertion of the external rotators on the proximal femur, the surgeon must identify the superior margin of quadratus femoris, which is marked by the trochanteric branch of the deep branch of the medial circumflex artery [45]. The trochanteric branch marks the level of the tendon of the obturator externus, which is crossed posteriorly by the deep branch of the medial circumflex. The obturator externus is responsible for protecting this vessel from either tension or compression during surgical hip dislocation. The deep branch ascends superiorly and pierces the capsule at the level of the superior gemellus. Once intracapsular, 2–4 subsynovial superior retinacular vessels pierce the head approximately 2–4 mm from the head-neck junction. Damage to the medial femoral circumflex artery or one of its branches may cause variable degrees of femoral head avascular necrosis. Following hip arthroscopy, the risk of avascular necrosis is less than 1 % (10 cases reported out of 6,334 hips) [7]. Following surgical dislocation, the risk is also significantly less than 1 % [9]. However, the intent of the surgery is to access 360° of the femoral head and acetabulum with a 0 % risk (not just less than 1 %) of avascular necrosis, implying that this complication is completely preventable with attention to appropriate technique [46]. Although avascular necrosis has been reported following surgical hip dislocation for the treatment of Perthes disease, slipped capital femoral epiphysis, and developmental hip dysplasia [47], it has not been reported for the treatment of femoroacetabular impingement. It must be recognized, however, that the surgeons performing these techniques are experienced surgeons that either developed the technique or have trained with the developers, performed hip vascular supply research with the developers, and performed a high volume of the technique. The femoral artery proper is at risk only with far medial straying of the anterior portal (3.5–4 cm medial).

Heterotopic Ossification

Heterotopic ossification may complicate the postoperative outcome after hip preservation surgery. Open approaches have significantly higher rates of heterotopic bone formation versus arthroscopic approaches although in the majority of cases it is not clinically relevant [9]. In a recent systematic review of 29 studies and over 2,500 hips, the rate of heterotopic ossification following surgical dislocation was 15 %, followed by mini-open (13 %), arthroscopic plus mini-open (3 %), and arthroscopy (<1 %) ($p < 0.05$) [9]. The use of the Brooker grading system revealed that most cases are grade 1 (72 %), followed by grade 2 (20 %), grade 3 (7 %), and grade 4 (1 %). Neither prophylaxis nor treatment was discussed for management of the ectopic bone formation in most studies. However, in the studies that did report the presence or absence of symptoms, the majority of subjects did not require further treatment, as the mild grades of ossification were largely not bothersome (Figs. 6 and 7).

Heterotopic ossification prophylaxis has been studied in two recent large investigations. In a retrospective comparative case series of 300 subjects with 18-month outcome after hip arthroscopy, the use of oral nonsteroidal anti-inflammatory drug (NSAID) prophylaxis was evaluated (naproxen 500 mg twice daily for 3 weeks in 248 subjects; indomethacin, ketoprofen, or etoricoxib for 3 weeks in 37 subjects) [48]. Fifteen subjects received no NSAID prophylaxis (control). Five cases of heterotopic ossification occurred following surgery, and all were in the control group (33 % rate without prophylaxis). No heterotopic ossification occurred if the patient received prophylaxis. In a separate comparative cohort study of 616 subjects after hip arthroscopy for FAI or peritrochanteric disorders, the addition of indomethacin (Indocin SR, Merck, Whitehouse Station, New Jersey, USA; 75 mg orally once daily for 4 days) to a naproxen-only (500 mg orally twice daily for 30 days) protocol reduced the risk of heterotopic ossification from 8.3 % to 1.8 % ($p < 0.05$) [49]. The latter analysis showed that patients that received only naproxen following surgery were 4.4 times more likely to develop ectopic bone formation. All cases of heterotopic ossification occurred in the setting of osteoplasty for FAI. One percent of subjects needed revision surgery for excision of heterotopic bone. Of note, patients receiving indomethacin also received omeprazole (20 mg orally once daily for 4 days following surgery) for gastric protection. Patients that develop heterotopic ossification are more

Fig. 6 A 20-year-old collegiate soccer player 1-year status post hip arthroscopy with heterotopic ossification. *Left*, pre-op. *Right*, post-op

Fig. 7 Arthroscopic removal of heterotopic ossification

frequently male (80 % in the former and 72 % in the latter studies) [48, 49]. Removal of all bony debris and coagulation of vessels within the periarticular muscles at the conclusion of FAI corrective procedures might also decrease the potential for HO development.

Other Minor Complications

Skin injury following hip arthroscopy is uncommon, with an incidence of 0.16 % (10 cases out of 6,334 hips; 6 labial and/or vaginal and 4 scrotal) [7]. Reduction in the duration and magnitude of traction, placement of a large (greater than 9 in. diameter to distribute pressure) perineal post more lateral (on the medial thigh) than central in the perineum, and visualization of safety of external genitalia upon traction initiation without any excessive pressure or malpositioning may reduce the already low incidence of skin injury following hip arthroscopy.

The rate of postoperative superficial infection (requiring only antibiotic therapy without surgery) is very low following hip arthroscopy (0.11 %). Only one case of septic arthritis requiring surgical arthrotomy and drainage has been reported in the literature following hip arthroscopy [50]. Despite a larger incision, the rate of infection following open approaches for hip preservation is very low and not significantly different from that of arthroscopy [9]. The deep nature of the hip mandates longer instruments with longer lever arms to visualize and instrument the joint. Thus, instrument breakage is a potential adverse event that is uncommon (9 cases out of 6,334 reported) [7] but largely preventable with safe, meticulous, and non-forceful cannulation and instrumentation. Although stiffness following hip preservation surgery is not commonly reported, the rate of lysis of adhesions for post-arthroscopic arthrofibrosis is 0.47 % [7].

Other Rare, but Severe, Complications

Despite their infrequency, rare complications that are potentially harmful to life or limb merit specific consideration, and steps should be taken to avoid them at all costs. Following hip preservation surgery (either open or arthroscopic), only two deaths have been reported (one in a polytrauma patient due to pulmonary embolus [51] and the other "an unrelated cause") [7, 9]. Despite extensive guidelines by multiple organizations in different fields of medicine regarding thromboembolic disease and its prophylaxis following hip arthroplasty, the literature following hip preservation surgery is scarce. Following both arthroscopic and open surgery, the literature contains only isolated case reports of superficial and deep vein thromboses and/or pulmonary emboli [7, 9]. The only guidelines published for hip arthroscopy thromboembolic disease prevention were released by the Italian Intersociety Working Group [52]. Despite no studies to support guidelines, the workgroup recommended thorough preoperative assessment of thrombophilic and bleeding risk factors and postoperative use of mechanical (compression stockings, intermittent sequential compression devices) and pharmacologic (low-molecular-weight heparin) measures for patients undergoing arthroscopy. Although the current rates of thromboembolic events after hip arthroscopy do not support the routine use of DVT prophylaxis, one might consider this for patients with significant risk factors such as those with clotting cascade disorders and those traveling long distances or flying in the early postoperative period.

Following femoral head-neck junction cam osteochondroplasty, there have been only five cases of proximal femur fracture reported in the literature. Three cases occurred following arthroscopy (all with femoral osteochondroplasty), with two being successfully treated nonoperatively and one requiring reduction and sliding hip screw fixation [4, 53, 54]. One subtrochanteric femur fracture has been reported following mini-open femoral osteochondroplasty, and one femoral neck fracture has been reported following arthroscopic-assisted mini-open femoral osteochondroplasty [9]. Regardless of the type of approach used, basic science literature has demonstrated up to 30 % of the femoral neck diameter may be removed without increased risk of fracture [55]. While fracture is rare and the optimal resection amount is still unknown, the surgeon must be cognizant that over-resection or resections in the presence of relatively osteopenic bone may increase the risk of fracture.

Intra-abdominal or intrathoracic extravasation of fluid may lead to abdominal compartment syndrome, cardiovascular collapse, and death. This complication is exclusively related to arthroscopy. Twenty-two cases have been reported in the literature (19 intra-abdominal; 3 intrathoracic) [7]. One of these patients did experience transient, yet prolonged, asystole, with subsequent successful resuscitation [56]. This was a case of a trauma patient with an acetabular fracture in which fluid extravasated through the fracture. This did require emergent exploratory laparotomy and decompression. A second case exhibited temporary apneic pulseless electrical activity that only responded to emergent laparotomy and decompression [57]. The other reported cases have demonstrated risk factors of longer operative time and performance of iliopsoas release [37, 58]. Thus, keeping intra-articular pressure as low as possible, performing the surgery as efficiently as possible without compromising quality, frequently monitoring the abdomen and peek ventilatory inspiratory pressure, and performing iliopsoas releases when indicated at the conclusion of the case might help to minimize the risk for this complication.

Classification Systems in Orthopedic and Hip Preservation Surgery

The lack of clear definitions and classification of complications in orthopedic surgery prompted the ANCHOR group to adapt the validated the Clavien-Dindo classification system to hip preservation surgery [8]. The original Clavien system was a four-grade classification used to assess complications following cholecystectomy [59]. This was modified by Dindo to a five-grade system, now utilized by general surgery and urology (Table 5) [60]. Ten hip surgeons from eight centers

Table 5 Modified Clavien-Dindo complication classification system [8]

	Definition	Example
Grade I	Requires no treatment	Asymptomatic heterotopic ossification
	Has no clinical relevance	Mild postoperative fever
	No deviation from routine follow-up	Simple wound problem not requiring intervention
Grade II	Deviation from normal postoperative course	Additional clinic visits
		Oral antibiotic treatment for superficial infection
	Requires outpatient treatment	Transient asymptomatic neuropraxia
Grade III	Treatable but requires surgical intervention	Deep infection requiring drainage
		Trochanteric nonunion requiring fixation
Grade IV	Life-threatening complication	Permanent nerve injury
	Requires intensive care unit	Avascular necrosis
	May result in permanent disability	Pulmonary embolism
Grade V	Death	

in three countries adapted the modified system to hip preservation surgery and reported a high inter- and intraobserver reliability [8]. The aim of the latter was to standardize the reporting of complications, improve the quality of evidence, and allow for valid comparison of outcome studies.

Summary

Recognition of femoroacetabular impingement as a potential precursor to hip osteoarthritis has led to the development of the field of both open and arthroscopic hip preservation within orthopedic surgery. The use of hip arthroscopy is rapidly increasing internationally. There is a significant learning curve associated with hip arthroscopy. The rate of minor complications is low (7.5 %) and is largely related to the learning curve. The two most common minor complications are iatrogenic chondrolabral injury and temporary neuropraxia. Although the rate of minor complications is higher following open surgical hip dislocation due to the development of painful hardware requiring removal, the rate of major complications is less than 1 % in both open and arthroscopic hip preservation surgeries. Lack of clarity in reporting complications within orthopedic surgery has spurred academic hip surgeons to adapt and test a general surgery-validated complication reporting system for use in hip preservation.

References

1. Bozic KJ, Chan V, Valone FH 3rd, Feeley BT, Vail TP. Trends in hip arthroscopy utilization in the United States. J Arthroplasty. 2013;28(8 Suppl):140–3.
2. Montgomery S, Ngo S, Hobson T, Nguyen S, Alluri R, Wang J, et al. Trends and demographics in hip arthroscopy in the United States. Arthroscopy. 2013;29(4):661-5.
3. Colvin AC, Harrast J, Harner C. Trends in hip arthroscopy. J Bone Joint Surg Am. 2012;94(4):e23.
4. Souza BG, Dani WS, Honda EK, Ricioli Jr W, Guimaraes RP, Ono NK, et al. Do complications in hip arthroscopy change with experience? Arthroscopy. 2010;26(8):1053–7.
5. Konan S, Rhee SJ, Haddad FS. Hip arthroscopy: analysis of a single surgeon's learning experience. J Bone Joint Surg Am. 2011;93 Suppl 2:52–6.
6. Lee YK, Ha YC, Hwang DS, Koo KH. Learning curve of basic hip arthroscopy technique: CUSUM analysis. Knee Surg Sports Traumatol Arthrosc. 2013;21(8):1940–4.
7. Harris JD, McCormick FM, Abrams GD, Gupta AK, Ellis TJ, Bach Jr BR, et al. Complications and reoperations during and after hip arthroscopy: a systematic review of 92 studies and more than 6,000 patients. Arthroscopy. 2013;29(3):589–95.
8. Sink EL, Leunig M, Zaltz I, Gilbert JC, Clohisy J. Reliability of a complication classification system for orthopaedic surgery. Clin Orthop Relat Res. 2012;470(8):2220–6.
9. Harris JD, Erickson BJ, Bush-Joseph CA, Nho SJ. Treatment of femoroacetabular impingement: a systematic review. Curr Rev Musculoskelet Med. 2013;6(3):207–18.
10. Harris J, Slikker W, Gupta A, Abrams G, Nho S. Routine complete capsular closure during hip arthroscopy. Arthrosc Tech. 2013;2(2):e89–94.

11. McCormick F, Slikker W 3rd, Harris JD, Gupta AK, Abrams GD, Frank J, et al. Evidence of capsular defect following hip arthroscopy. Knee Surg Sports Traumatol Arthrosc. 2013 Jul 13. [Epub ahead of print]. PMID: 23851921.
12. Draovitch P, Edelstein J, Kelly BT. The layer concept: utilization in determining the pain generators, pathology and how structure determines treatment. Curr Rev Musculoskelet Med. 2012;5(1):1–8.
13. Badylak JS, Keene JS. Do iatrogenic punctures of the labrum affect the clinical results of hip arthroscopy? Arthroscopy. 2011;27(6):761–7.
14. Krych AJ, Thompson M, Knutson Z, Scoon J, Coleman SH. Arthroscopic labral repair versus selective labral debridement in female patients with femoroacetabular impingement: a prospective randomized study. Arthroscopy. 2013;29(1):46–53.
15. Espinosa N, Beck M, Rothenfluh DA, Ganz R, Leunig M. Treatment of femoro-acetabular impingement: preliminary results of labral refixation. Surgical technique. J Bone Joint Surg Am. 2007;89(Suppl 2 Pt.1):36–53.
16. Larson CM, Giveans MR, Stone RM. Arthroscopic debridement versus refixation of the acetabular labrum associated with femoroacetabular impingement: mean 3.5-year follow-up. Am J Sports Med. 2012; 40(5):1015–21.
17. Domb B, Hanypsiak B, Botser I. Labral penetration rate in a consecutive series of 300 hip arthroscopies. Am J Sports Med. 2012;40(4):864–9.
18. Domb BG, Botser IB. Iatrogenic labral puncture of the hip is avoidable. Arthroscopy. 2012;28(3):305–7; author reply 7–8.
19. Lertwanich P, Ejnisman L, Torry MR, Giphart JE, Philippon MJ. Defining a safety margin for labral suture anchor insertion using the acetabular rim angle. Am J Sports Med. 2011;39(Suppl):111S–6.
20. Benali Y, Katthagen BD. Hip subluxation as a complication of arthroscopic debridement. Arthroscopy. 2009;25(4):405–7.
21. Matsuda DK. Acute iatrogenic dislocation following hip impingement arthroscopic surgery. Arthroscopy. 2009;25(4):400–4.
22. Mei-Dan O, McConkey MO, Brick M. Catastrophic failure of hip arthroscopy due to iatrogenic instability: can partial division of the ligamentum teres and iliofemoral ligament cause subluxation? Arthroscopy. 2012;28(3):440–5.
23. Ilizaliturri Jr VM. Complications of arthroscopic femoroacetabular impingement treatment: a review. Clin Orthop Relat Res. 2009;467(3):760–8.
24. Ranawat AS, McClincy M, Sekiya JK. Anterior dislocation of the hip after arthroscopy in a patient with capsular laxity of the hip. A case report. J Bone Joint Surg Am. 2009;91(1):192–7.
25. Sansone M, Ahlden M, Jonasson P, Sward L, Eriksson T, Karlsson J. Total dislocation of the hip joint after arthroscopy and ileopsoas tenotomy. Knee Surg Sports Traumatol Arthrosc. 2013;21(2):420–3.
26. Myers CA, Register BC, Lertwanich P, Ejnisman L, Pennington WW, Giphart JE, et al. Role of the acetabular labrum and the iliofemoral ligament in hip stability: an in vitro biplane fluoroscopy study. Am J Sports Med. 2011;39(Suppl):85S–91.
27. Martin HD, Savage A, Braly BA, Palmer IJ, Beall DP, Kelly B. The function of the hip capsular ligaments: a quantitative report. Arthroscopy. 2008;24(2):188–95.
28. Hewitt JD, Glisson RR, Guilak F, Vail TP. The mechanical properties of the human hip capsule ligaments. J Arthroplasty. 2002;17(1):82–9.
29. Philippon MJ. The role of arthroscopic thermal capsulorrhaphy in the hip. Clin Sports Med. 2001;20 (4):817–29.
30. Blakey CM, Field MH, Singh PJ, Tayar R, Field RE. Secondary capsular laxity of the hip. Hip Int. 2010;20(4):497–504.
31. Bellabarba C, Sheinkop MB, Kuo KN. Idiopathic hip instability. An unrecognized cause of coxa saltans in the adult. Clin Orthop Relat Res. 1998;355:261–71.
32. Shu B, Safran MR. Hip instability: anatomic and clinical considerations of traumatic and atraumatic instability. Clin Sports Med. 2011;30(2):349–67.
33. Fabricant PD, Bedi A, De La Torre K, Kelly BT. Clinical outcomes after arthroscopic psoas lengthening: the effect of femoral version. Arthroscopy. 2012;28(7):965–71.
34. Baca V, Bacova T, Grill R, Otcenasek M, Kachlik D, Bartoska R, et al. Pudendal nerve in pelvic bone fractures. Injury. 2013;44(7):952–6.
35. Brumback RJ, Ellison TS, Molligan H, Molligan DJ, Mahaffey S, Schmidhauser C. Pudendal nerve palsy complicating intramedullary nailing of the femur. J Bone Joint Surg Am. 1992;74(10):1450–5.
36. Kruger DM, Kayner DC, Hankin FM, Falahee MH, Kaufer H, Matthews LS, et al. Traction force profiles associated with the use of a fracture table: a preliminary report. J Orthop Trauma. 1990;4(3):283–6.
37. Sampson TG. Complications of hip arthroscopy. Clin Sports Med. 2001;20(4):831–5.
38. Mei-Dan O, McConkey MO, Young DA. Hip arthroscopy distraction without the use of a perineal post: prospective study. Orthopedics. 2013;36(1):e1–5.
39. Ochs BC, Herzka A, Yaylali I. Intraoperative neurophysiological monitoring of somatosensory evoked potentials during hip arthroscopy surgery. Neurodiagn J. 2012;52(4):312–9.
40. Telleria JJ, Safran MR, Harris AH, Gardi JN, Glick JM. Risk of sciatic nerve traction injury during hip arthroscopy-is it the amount or duration? An intraoperative nerve monitoring study. J Bone Joint Surg Am. 2012;94(22):2025–32.
41. Birmingham P. Hip arthroscopy neurapraxia: is it only about weight of traction? J Bone Joint Surg Am. 2012;94(22):e169.
42. Kosiyatrakul A, Nuansalee N, Luenam S, Koonchornboon T, Prachaporn S. The anatomical variation of the lateral femoral cutaneous nerve in relation

to the anterior superior iliac spine and the iliac crest. Musculoskelet Surg. 2010;94(1):17–20.
43. Byrd JW, Pappas JN, Pedley MJ. Hip arthroscopy: an anatomic study of portal placement and relationship to the extra-articular structures. Arthroscopy. 1995; 11(4):418–23.
44. Robertson WJ, Kelly BT. The safe zone for hip arthroscopy: a cadaveric assessment of central, peripheral, and lateral compartment portal placement. Arthroscopy. 2008;24(9):1019–26.
45. Gautier E, Ganz K, Krugel N, Gill T, Ganz R. Anatomy of the medial femoral circumflex artery and its surgical implications. J Bone Joint Surg Br. 2000; 82(5):679–83.
46. Ganz R, Gill TJ, Gautier E, Ganz K, Krugel N, Berlemann U. Surgical dislocation of the adult hip a technique with full access to the femoral head and acetabulum without the risk of avascular necrosis. J Bone Joint Surg Br. 2001;83(8):1119–24.
47. Alves C, Steele M, Narayanan U, Howard A, Alman B, Wright JG. Open reduction and internal fixation of unstable slipped capital femoral epiphysis by means of surgical dislocation does not decrease the rate of avascular necrosis: a preliminary study. J Child Orthop. 2012;6(4):277–83.
48. Randelli F, Pierannunzii L, Banci L, Ragone V, Aliprandi A, Buly R. Heterotopic ossifications after arthroscopic management of femoroacetabular impingement: the role of NSAID prophylaxis. J Orthop Traumatol. 2010;11(4):245–50.
49. Bedi A, Zbeda RM, Bueno VF, Downie B, Dolan M, Kelly BT. The incidence of heterotopic ossification after hip arthroscopy. Am J Sports Med. 2012;40 (4):854–63.
50. Clarke MT, Arora A, Villar RN. Hip arthroscopy: complications in 1054 cases. Clin Orthop Relat Res. 2003;406:84–8.
51. Bushnell BD, Dahners LE. Fatal pulmonary embolism in a polytraumatized patient following hip arthroscopy. Orthopedics. 2009;32(1):56.
52. Randelli F, Romanini E, Biggi F, Danelli G, Della Rocca G, Laurora NR, et al. II Italian intersociety consensus statement on antithrombotic prophylaxis in orthopaedics and traumatology: arthroscopy, traumatology, leg immobilization, minor orthopaedic procedures and spine surgery. J Orthop Traumatol. 2013;14(1):1–13.
53. Gedouin JE, May O, Bonin N, Nogier A, Boyer T, Sadri H, et al. Assessment of arthroscopic management of femoroacetabular impingement. A prospective multicenter study. Orthop Traumatol Surg Res. 2010; 96(8 Suppl):S59–67.
54. Ayeni OR, Bedi A, Lorich DG, Kelly BT. Femoral neck fracture after arthroscopic management of femoroacetabular impingement: a case report. J Bone Joint Surg Am. 2011;93(9):e47.
55. Mardones RM, Gonzalez C, Chen Q, Zobitz M, Kaufman KR, Trousdale RT. Surgical treatment of femoroacetabular impingement: evaluation of the effect of the size of the resection. Surgical technique. J Bone Joint Surg Am. 2006;88(Suppl 1 Pt 1):84–91.
56. Bartlett CS, DiFelice GS, Buly RL, Quinn TJ, Green DS, Helfet DL. Cardiac arrest as a result of intraabdominal extravasation of fluid during arthroscopic removal of a loose body from the hip joint of a patient with an acetabular fracture. J Orthop Trauma. 1998;12(4):294–9.
57. Sharma A, Sachdev H, Gomillion M. Abdominal compartment syndrome during hip arthroscopy. Anaesthesia. 2009;64(5):567–9.
58. Haupt U, Volkle D, Waldherr C, Beck M. Intra- and retroperitoneal irrigation liquid after arthroscopy of the hip joint. Arthroscopy. 2008;24(8):966–8.
59. Clavien PA, Sanabria JR, Strasberg SM. Proposed classification of complications of surgery with examples of utility in cholecystectomy. Surgery. 1992;111(5):518–26.
60. Dindo D, Demartines N, Clavien PA. Classification of surgical complications: a new proposal with evaluation in a cohort of 6336 patients and results of a survey. Ann Surg. 2004;240(2):205–13.

Part III

Pediatric Hip Conditions

Surgical Technique: Pavlik Harness and Closed Reduction for Developmental Dysplasia of the Hip

29

Yale A. Fillingham, Brandon J. Erickson, Michael D. Hellman, Gregory Cvetanovich, and Monica Kogan

Contents

Abstract

Developmental dysplasia of the hip (DDH) is a common pediatric disorder with 1 % of children experiencing subluxation or dysplasia and 0.1 % of children having a dislocated hip. Diagnosis of the disorder is initially found on physical exam, but further confirmation is done with radiographic and ultrasonographic imaging of the hip. Treatment of DDH is essential to prevent significant future disability. Regardless of the treatment modality, the goal of treatment has been to maintain a stable concentrically reduced hip joint to promote acetabular remodeling and long-term stability. Several treatments exist with nonsurgical options including the Pavlik harness and closed reduction of the hip with spica casting. Surgical choices are open reduction of the hip with the option of a femoral or acetabular osteotomy. The choice of treatment is based on several factors with the primary factors including age of the patient and degree of hip stability. Among the nonsurgical options, the Pavlik harness has become the standard treatment for infants with dysplasia and reducible hips. The outcomes have demonstrated success between 53 % and 99 % with infrequent complications of osteonecrosis, femoral nerve palsy, and Pavlik harness disease. When children are considered too old for the harness or have failed the Pavlik harness, the alternative treatment is closed reduction of the hip and spica casting. However, closed reduction of the hip and spica

Y.A. Fillingham (✉) • B.J. Erickson • M.D. Hellman • G. Cvetanovich
Department of Orthopaedic Surgery, Rush University Medical Center, Chicago, IL, USA
e-mail: yale.fillingham@gmail.com; berickso.24@gmail.com; mdhellman@gmail.com; gregory.cvetanovich@gmail.com

M. Kogan
Department of Orthopaedic Surgery, Rush University Medical Center, Chicago, IL, USA

Pediatric Orthopaedic Surgery, Rush University Medical Center, Chicago, IL, USA
e-mail: mkogan@rushortho.com

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_30

casting are associated with a high rate of conversion to open reduction and a 22–66 % need for future treatment or surgery.

Introduction

Developmental dysplasia of the hip (DDH) has evolved to reference a spectrum of pathologic conditions of the developing pediatric hip. The conditions collectively referred to as DDH include the range of acetabular dysplasia to hip subluxation to irreducible hip dislocation. Regardless of where patients present along the spectrum of DDH, early identification and treatment are necessary to prevent future disability. As a result of this disorder, it has been implicated in up to 9 % of all primary hip replacements and up to 29 % of all hip replacements in patients under the age of 60 years [1].

Physical examination and ultrasonography are important diagnostic tools to aid in the early identification of DDH. Despite agreement for newborn screening of DDH, the screening protocol has dramatic geographical differences in the utilization of ultrasonography. Central Europe has an aggressive protocol with universal ultrasonography of newborns, whereas it is used more sparingly in English-speaking countries [2, 3]. Universal screening has been thought to be essential in Central Europe due to the relatively higher incidence of DDH [2]. However, the only two randomized control trials published on the utilization of ultrasonography in the screening of DDH demonstrated no significant reduction in the rates of late presenting cases with universal screening but did have higher rates of treatment [4, 5]. As a result, selective ultrasonographic screening has become the primary protocol in the United States.

The goal of treatment in DDH is to obtain and maintain a stable concentrically reduced hip joint to allow adequate acetabular remodeling and formation of long-term stability [6]. Predictors of a successful outcome include not only the severity of the disorder but the age at reduction of the hip. Because acetabular remodeling cannot be ensured after the age of 18 months, it is important to initiate treatment while the child still has the ability to form long-term stability of the hip joint [7].

Historically, maintaining reduction of the hip while the acetabulum undergoes remodeling had been one of the challenges to the successful treatment of DDH. In the 1950s, Arnold Pavlik of Czechoslovakia published articles on the treatment of hip dysplasia using a harness consisting of a chest strap, shoulder straps, and anterior and posterior stirrup straps with the purpose of maintaining the hips in flexion and abduction [8–15]. Pavlik reported promising outcomes of good results for 85 % of the patients with hip dislocations and only a 2.8 % rate of osteonecrosis [8]. As a result of his work, the Pavlik harness has become a standard in the treatment of DDH [2, 8, 16–19].

In the treatment of DDH, the orthopedist has available both nonsurgical and surgical methods of treatment. Nonsurgical options include the Pavlik harness and closed reduction of the hip with spica casting, while surgical choices are open reduction of the hip with the option of a femoral or acetabular osteotomy. Due to the extensive awareness among the medical community and early detection of DDH, most children are capable of solely being treated with nonsurgical options. Consequently, this chapter will discuss the indications, techniques, complications, and outcomes in the nonsurgical treatment of DDH.

Indications

The choice of treatment is typically based on age of the patient, stability of the hip, the ability to reduce the dislocated hip, and the degree of acetabular dysplasia as measured on ultrasound. Because age of the patient at the time of initiating treatment has a significant influence on the mode and success of treatment, it often guides the clinical decision-making process. Consensus among the orthopedic literature has been to group patients into three categories based on age: newborn to 6 months of age, 6–18 months of age, and 18 months of age and older.

Newborn to 6 Months of Age

When an infant has a physical exam suggestive of DDH (Figs. 1 and 2), the diagnosis is typically confirmed with routine radiography and ultrasonography of the hips and pelvis [20, 21]. The foundation of the Graft Classification is the patient's age and the measured α and β angles, which correspond to types I, II, D, III, and IV hips (Table 1) [20, 21]. The patients are further characterized based on the severity of the disorder, which guides the treatment protocol (Fig. 3) [16]. Since patients who experience subluxation of the hip can have spontaneous resolution, it is acceptable to observe and reevaluate the patient in 3 weeks. If the physical exam and ultrasonographic reevaluation demonstrate a stable hip, no further treatment is warranted for the patient. In the event the patient continues to experience subluxation of the hip, a Pavlik harness is indicated to treat the patient.

If the hip has a greater degree of instability and is dislocated or dislocatable, the patient should be placed in a Pavlik harness [2, 8, 16]. Alternatively in the setting of an unreliable family or unfavorable social situation, a spica cast would be the initial choice of treatment instead of the Pavlik harness [16]. Within 2–3 weeks of stabilizing the hip in a Pavlik harness, the patient must be observed for spontaneous reduction of the hip. If the hip has been reduced, the patient should undergo a full treatment course with the Pavlik harness and periodic observation. When the hip fails to spontaneously reduce in the Pavlik harness, the patient should be considered a candidate for a closed reduction of the hip and spica casting with the option of prereduction overhead traction.

6 to 18 Months of Age

Patients between the ages of 6 and 18 months retain the ability to readily remodel the acetabulum and initially warrant nonsurgical treatment. Unfortunately, use of the harness is often precluded in children who begin to wear it after the age of 6 months. The difficulty with initiating the required immobilization in the harness has led to failure rates exceeding 50 % in older patients [18, 22]. The treatment of choice for patients 6–18 months of age would be closed reduction of the hip and spica casting with the option of an adductor tenotomy to aid the reduction (Fig. 4) [22]. Regrettably, patients with an irreducible hip or failure to have a medial dye pool of less than 7 mm on arthrography (refer to section on closed reduction and spica casting for definition of medial dye pool) following closed reduction must proceed with open reduction and spica casting [22, 23].

Fig. 1 Observation of the skin folds on the patient's legs will often demonstrate asymmetry of the skin folds

Fig. 2 Examination of the patient shows a positive Galeazzi sign with a difference in knee height. After further investigation, it was confirmed that the patient had a dislocated left hip

Table 1 Graf classification[a]

Graf hip type	Descriptive term	α angle (°)	β angle (°)	Age (years)	Recommended treatment	Equivalent radiographic description
I a	Normal	≥60	>70	Any	No	Concentrically reduced
I b	Normal	≥60	<70	Any	No	Concentrically reduced
II a[b]	Physiologically immature	50–59	na[c]	0–12 weeks	Depends on age and α angle	Concentrically reduced
II b	Immature	50–59	na[c]	>12 weeks	Yes	Concentrically reduced
II c	Dysplastic with risk of dislocation	43–49	<77	Any	Yes	Centered on medial wall of acetabulum with sloping of roof
D	Decentered	43–49	>77	Any	Yes	Subluxated
III a	Decentered	<43	na[d]	Any	Yes	Dislocated
III b	Decentered	<43	na[d]	Any	Yes	Dislocated
IV	Decentered	<43	na[d]	Any	Yes	Dislocated

[a]Reproduction from Roposch et al. [21]
[b]After the age of 6 weeks, type II a is subclassified in II a plus (still considered physiologically immature and no treatment is mandated) and II a minus (immaturity is not considered to be physiologic anymore and treatment is mandated) according to the α angle
[c]Defined by α angles only; *na* not applicable
[d]Beta angles must not be measured in decentered hips

18 Months of Age and Older

Older patients have a reduced capacity to remodel the acetabulum, which inhibits successful nonsurgical treatment of DDH. Despite the increased failure of closed reduction of the hip and spica casting, it is recommended that patients undergo a trail of nonsurgical treatment (Fig. 5) [22]. Because certain studies have suggested that acetabular remodeling can occur up to the age of 8 years, a trial of closed reduction and spica casting can limit the associated morbidity of an extensive open procedure [24, 25].

Nonsurgical Techniques

Pavlik Harness

Since Arnold Pavlik published articles on the harness that would bear his name, other devices have been available but all have failed to offer the same flexibility of use and stability of the hip [8, 16, 26]. The original harness has evolved over the last half-century from its original construct of leather straps; however, the concept of maintaining the hips in flexion and abduction while limiting extension and adduction continues to be the foundation of today's Pavlik harness. The use of the Pavlik harness has been accepted as the standard of treatment for DDH among infants [8, 16].

Technique

In general, the Pavlik harness consists of a chest strap, shoulder straps, and anterior and posterior stirrup straps (Fig. 6). Several manufactures offer a preassembled Pavlik harness, which all slightly differ in the application of the harness. Because the Pavlik harness can appear complicated due to the multiple straps, it is recommended to be familiar with the manufacture's handbook and how to properly apply the specific harness. Once the Pavlik harness has been applied to the infant, the straps should be adjusted to situate the hips into the optimal position within the "safe zone" of Ramsey [16, 19]. In order to determine the "safe zone," position the infant's hips in 90° of flexion and passively adduct the hips noting when the femoral head displaces from the acetabulum [16, 19]. The "safe zone" is the degree difference between maximal passive abduction and when the femoral head dislocates during passive adduction

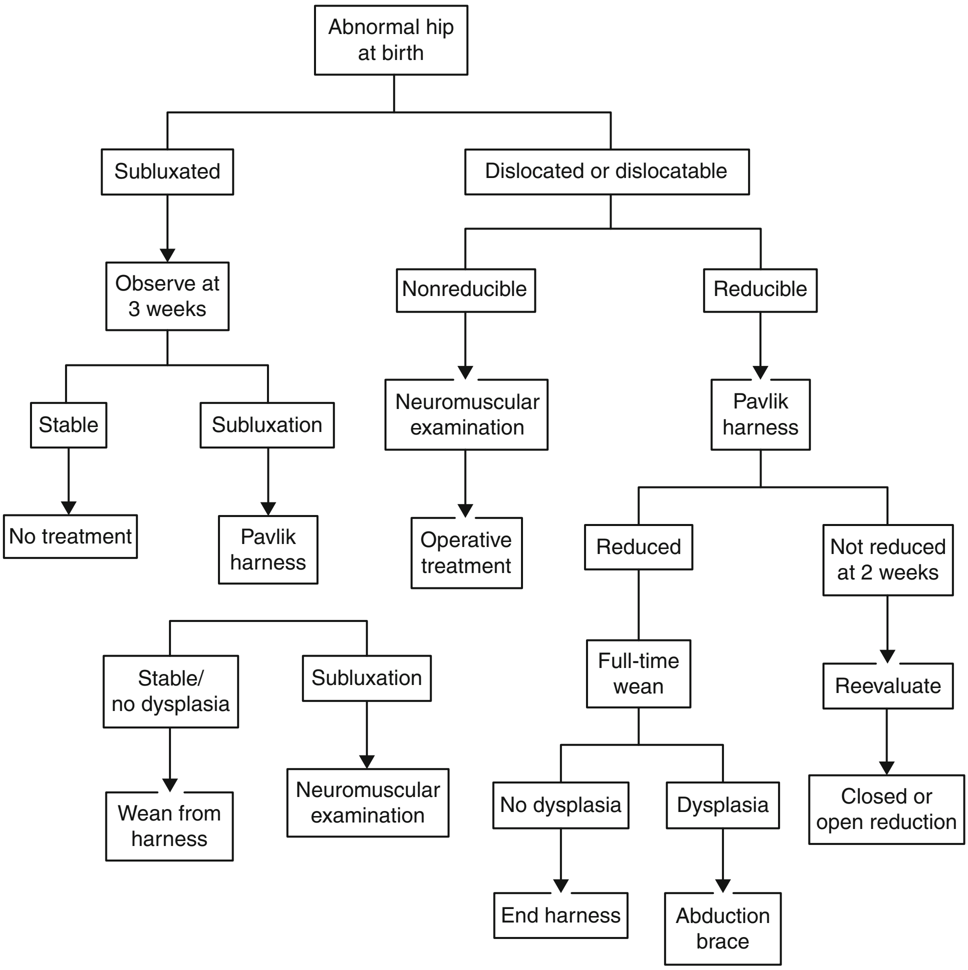


Fig. 3 Algorithm for evaluation and treatment of DDH in the newborn to 6 months of age (© 2000 American Academy of Orthopaedic Surgeons. Reprinted from the *Journal of the American Academy of Orthopaedic Surgeons*, volume 8(4), pp. 232–242 with permission)

[16, 19]. If the infant has a narrow "safe zone" of less than 40°, there should be consideration of an adductor tenotomy [19]. After the straps have been adjusted, no actual reduction maneuver of the hip is required. The normal movement of the infant in the harness will usually cause the hip to spontaneously reduce [2]. When infants failed to achieve hip reduction with the Pavlik harness in Ramsey's series, he noted the most common cause of failure was inadequate hip flexion [16].

Before patients leave the office, the parents must be properly trained to remove, apply, and adjust the harness straps. It will often take significant counseling of the parents until they are comfortable with the harness. Since parents will need to be capable of adjusting the straps but likely would not be able to accurately assess the infant's "safe zone," typically the proper position to instruct parents is to position the hips between 100° and 110° of flexion with mild abduction [2, 17, 19]. When parents appear to have significant difficulty in the office with the harness, it is recommended to provide closer follow-up. If the parents continually have difficulty with the harness, it would be considered an indication to transition the infant to a spica cast.

Multiple variables will determine the duration of treatment and monitoring of the infant in a

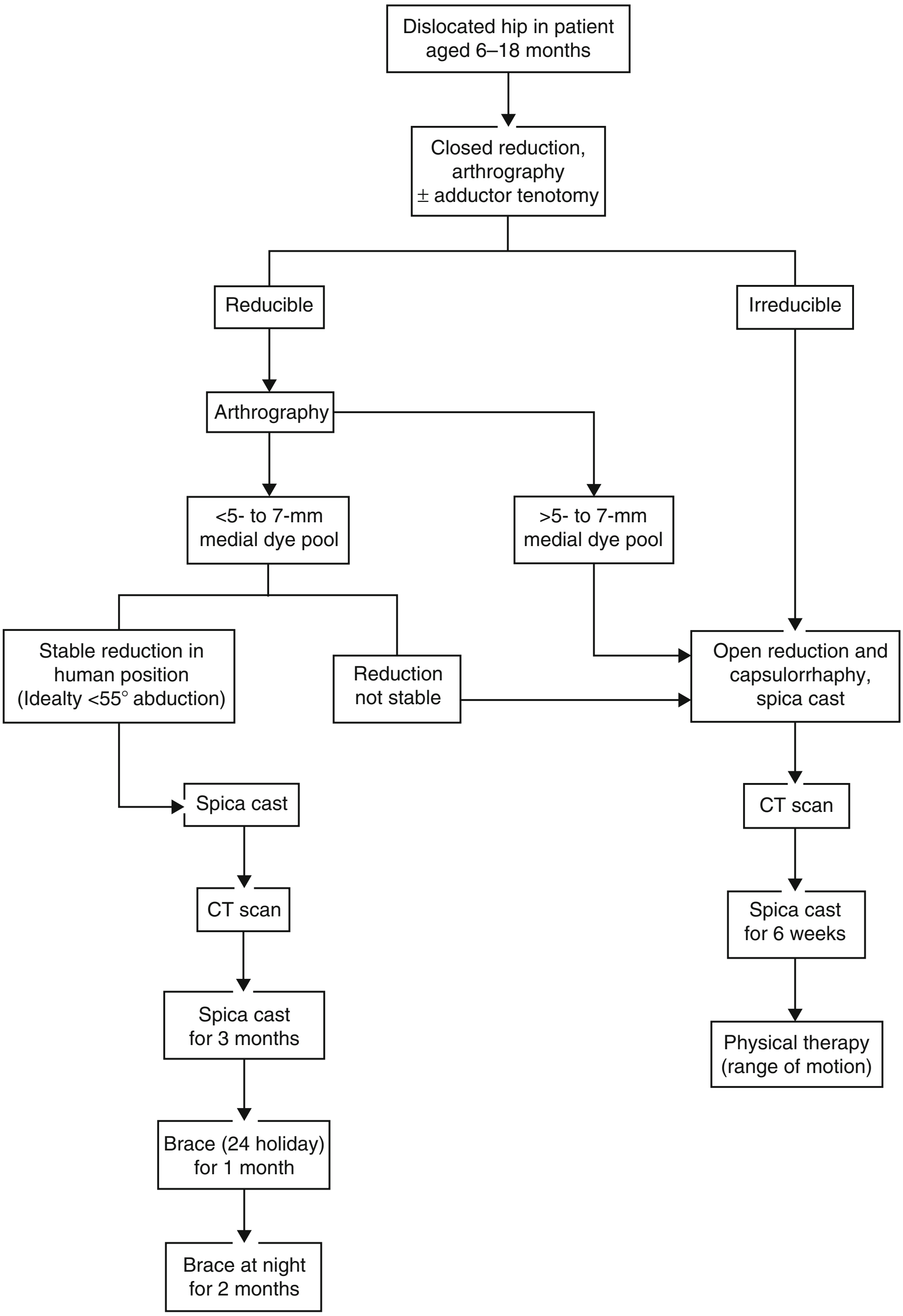


Fig. 4 Algorithm for evaluation and treatment of DDH in children aged 6–18 months of age (© 2001 American Academy of Orthopaedic Surgeons. Reprinted from the *Journal of the American Academy of Orthopaedic Surgeons*, volume 9(6), pp. 401–411 with permission)

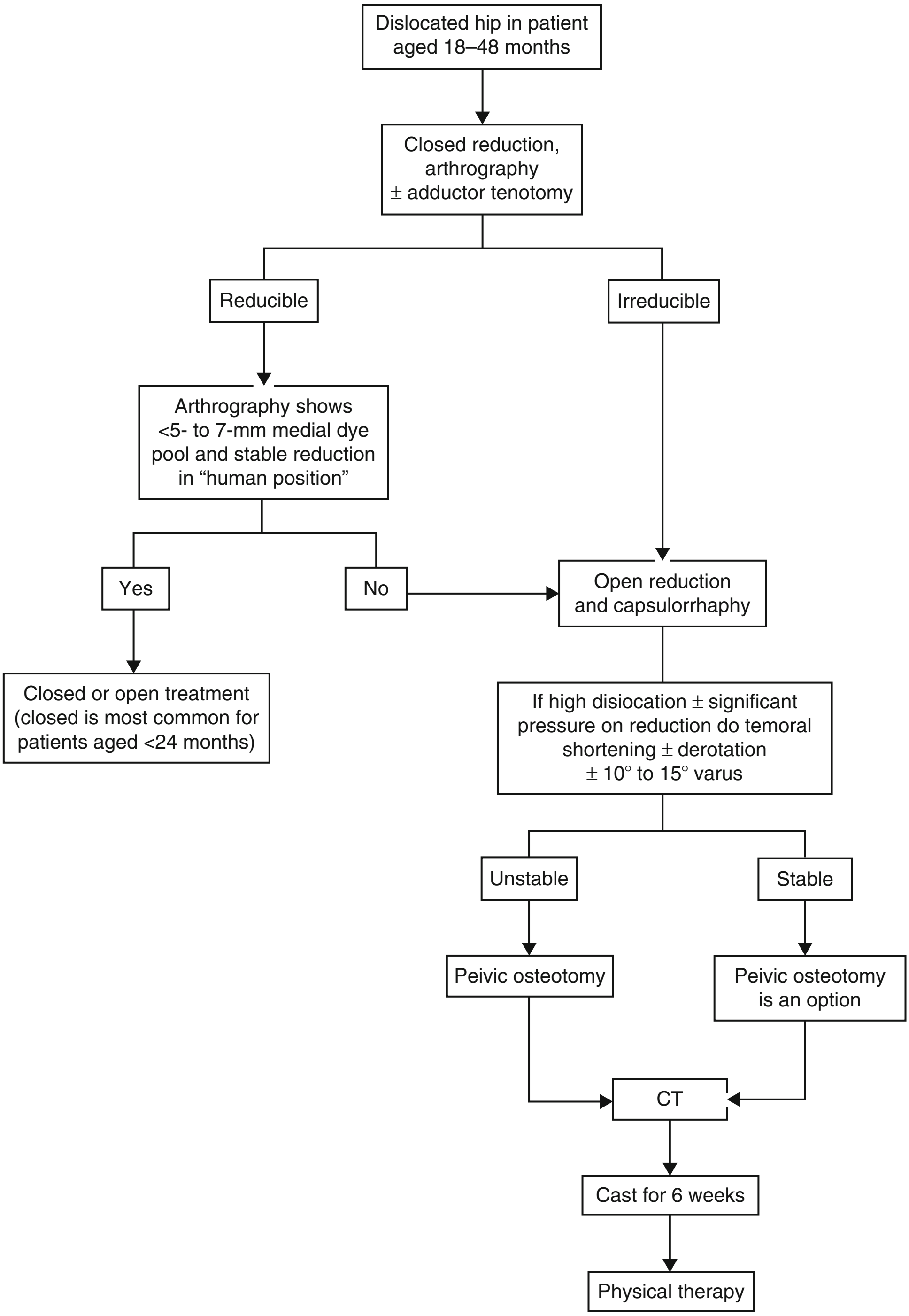


Fig. 5 Algorithm for evaluation and treatment of DDH in children aged more than 18 months (© 2001 American Academy of Orthopaedic Surgeons. Reprinted from the *Journal of the American Academy of Orthopaedic Surgeons*, volume 9(6), pp. 401–411 with permission)

Fig. 6 Infant in the Pavlik harness demonstrates proper placement of the chest strap, shoulder straps, and anterior and posterior stirrup straps with the hips in adequate flexion and abduction

Pavlik harness. Once the infant has been placed in the harness, ultrasonographic evaluation should document adequate flexion and direction of the femoral head toward the triradiate cartilage [27]. The infant should wear the harness for at least 23 h a day until clinical and ultrasonographic examinations are within normal limits [8, 28]. Infants with a dislocated hip should be reevaluated every 1–2 weeks to observe for reduction of the hip, while some recommend that the infants with adequate parental support do not require further evaluation for 3–4 weeks [8, 17, 19]. If the hip remains dislocated or demonstrates no improvement in the degree of dysplasia on ultrasound after 3–4 weeks, the harness should be discontinued to prevent Pavlik harness disease and the infant is considered a candidate for closed reduction and spica casting [8, 17].

After successful reduction of the hip, the infant only needs follow-up every 3–4 weeks for reevaluation. In general, the duration of treatment in the Pavlik harness is a minimum of 3 months for infants 3 months of age or younger, but older children should remain in the harness for approximately double their age [8]. It is not necessary for the harness to be worn 23 h a day during the entire duration of treatment. Beginning at the midpoint of treatment, the hours spent in the harness can gradually be weaned [16]. Prior to weaning the harness, the parents should take the patient out of the harness the night before the office visit [16]. If the ultrasonographic evaluation the following day is consistent with a stable hip, the infant can increase to 4 hours per day spent out of the harness for the first third of the remaining treatment period, then 8 hours per day for the second third of the treatment period, and then 12 hours per day for the last third of the duration of treatment. At the end of the weaning process, check for residual acetabular dysplasia with the use of ultrasonography. If present, continue to wear the harness 12 h per day until the patient has radiographic resolution of the dysplasia [16].

Several contraindications exist in the use of a Pavlik harness to treat DDH. Because reduction of the hip is reliant on normal movements of the infant, any delay in motor skills or major muscle imbalance would preclude the use of the Pavlik harness [2, 8]. After reduction of the hip, the Pavlik harness along with the soft tissue helps to maintain the reduction. Therefore, infants with ligamentous laxity as in Ehlers-Danlos syndrome would not benefit from the harness [8]. As previously stated, the infant's age and family situation would be limiting factors and an indication for another treatment option [2, 28]. Although not contraindications, historically cited risk factors for failure of the Pavlik harness are an absent Ortolani sign at the initial evaluation, bilateral dislocation, and age of greater than 7 weeks before initiation of treatment [29]. More recently, a systematic review on the Pavlik harness identified the radiographic distance from the midpoint of the proximal metaphyseal border of the femur to Hilgenreiner's line on the initial radiograph as predictive of success and failure of the Pavlik harness [30]. The literature has demonstrated that a value greater than 8 mm is associated with a reduced risk of AVN and a value greater than 6 mm was indicative of a satisfactory outcome [31–33].

Closed Reduction and Spica Casting

When children fail or exhibit a contraindication to the Pavlik harness, the remaining alternative to surgical treatment is closed reduction and spica casting. Historically, closed reduction of the hip was preceded by the use of longitudinal or overhead traction. More recently, the use of traction has become controversial due to complications of skin breakdown and the belief that it does not aid in the reduction [22]. Advocates for prereduction traction believe the gentle stretching of the soft tissue structures improves the success of closed reduction and reduces the risk of osteonecrosis. However, the current literature has conflicting reports on the use of prereduction traction [22, 34, 35].

Technique

Closed reduction of the hip should be performed under general anesthesia in the operating room to provide adequate muscle paralysis. The reduction maneuver involves longitudinal traction, flexion, and abduction of the hip, all while applying posterior pressure on the greater trochanter [16]. Frequently an adductor tenotomy is necessary via an open or percutaneous technique, which relieves one of the opposing forces and widens the "safe zone." After reduction of the hip, intraoperative arthrography will confirm a concentric reduction of the femoral head by demonstrating a collection of dye in the space between the femoral head and medial border of the acetabulum of less than 5–7 mm (Figs. 7 and 8) [23]. The previously described collection of dye is often referred to as the "medial dye pool." If the medial dye pool measures greater than 7 mm, it is an indication to proceed with an open reduction [23].

Once reduction of the hip has been documented, the stable zones of the hip in all planes of direction (abduction/adduction, flexion/extension, internal/external rotation) should be identified to ensure stability of the hip in the "human position" prior to applying the spica cast. The purpose of the spica cast is to maintain the hip in 100° of flexion and 40–50° of abduction, which is commonly referred to as the "human position" of the hip (Fig. 9). The spica cast is technically demanding, but close attention to detail can ensure hip positioning and maintenance of the reduction. Because the padding over the anterior aspect of the hip has a tendency to extend the hip, it is prudent to maintain necessary flexion until the casting material has hardened.

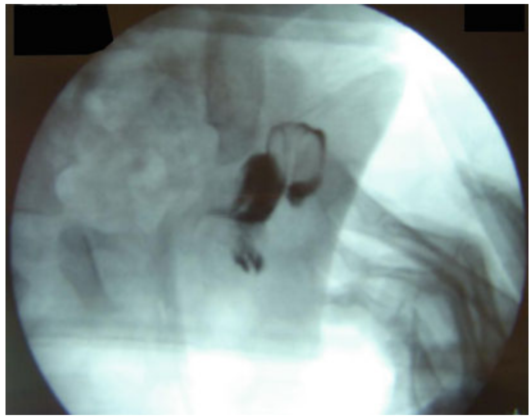


Fig. 7 Intraoperative arthrography prior to closed reduction of the hip shows the medial dye pool confirming the presence of a dislocated hip

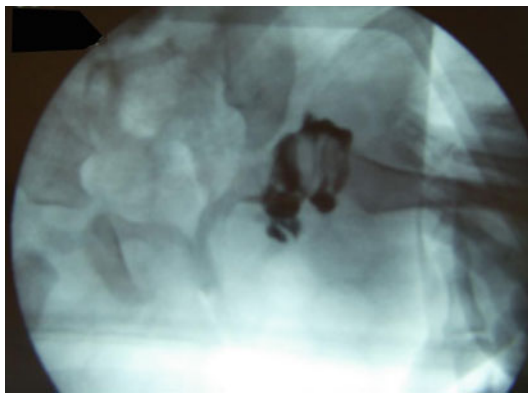


Fig. 8 Intraoperative arthrography following closed reduction of the hip demonstrates resolution of the medial dye pool confirming reduction of the hip

The femoral head will often migrate posteriorly leading to a loss in the reduction, but the use of a greater trochanter mold can help prevent the migration (Fig. 10b) [22].

Because the hip can dislocate in the process of the cast application, it is important to confirm reduction of the hip in the spica cast (Fig. 10a, b). The standard imaging modality has been computed tomography (CT) scan but an alternative is magnetic resonance imaging (MRI) [17, 19]. After reduction of the hip in the spica cast has been documented, children are generally immobilized for 3–4 months regardless of the child's age with periodic plain film radiographs and spica cast changes as needed for hygiene and growth of the child (Fig. 11) [17, 22]. When the cast is removed, the children are further immobilized with a removable abduction brace. The abduction brace is used either until the acetabulum normalizes or for 4 weeks followed by 4 weeks at night time [17, 22]. During the treatment of closed reduction and casting, it is possible to use the change in acetabular angle to track the child's progress. If the acetabular angle has not decreased by at least 4° by 6 months after the reduction, abandonment of close reduction for surgical correction should be considered [23]. The measurement of acetabular angle has demonstrated intraobserver variability, which would affect its interpretation [36]. Therefore, it is recommended to only use acetabular angles as a guiding tool in the larger clinical setting. In the event of surgical correction, a femoral osteotomy is primarily used in children with acetabular remodeling potential because it redirects the femoral head into the acetabulum [22]. Older children without acetabular remodeling potential will often require the addition of a pelvic osteotomy; however, certain authors have advocated for pelvic osteotomy as the index procedure for patients older than 18 months [22].

Fig. 9 Child was placed in the spica cast following closed reduction with the hips located in the human position

Discussion

The Pavlik harness and closed reduction of the hip with spica casting have proven to be reliable nonsurgical treatment options for DDH in infants and children. However, they have been associated

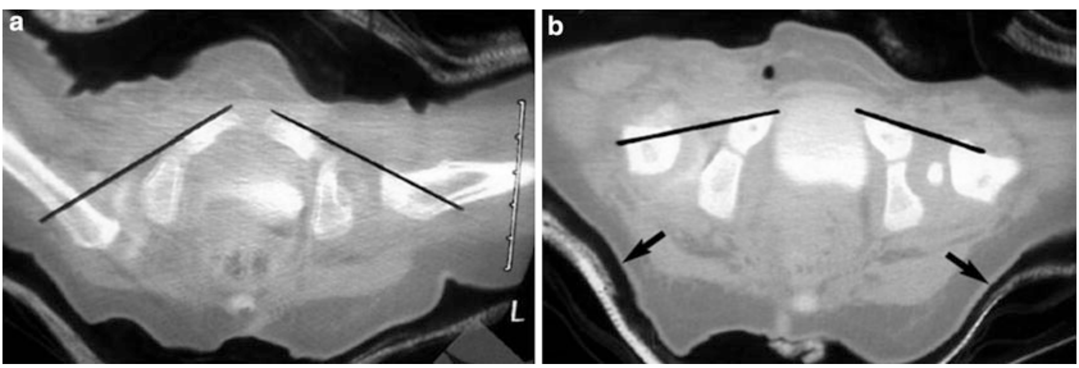


Fig. 10 (**a**) CT scan obtained after attempted closed reduction of a dislocated right hip. *Line* drawn parallel to the *right* pubic ramus misses the proximal metaphysis. The hip was not reduced, and the patient was immediately taken back to the operating room. (**b**) CT scan obtained after reduction of the hip. *Lines* drawn along the pubic rami are continuous with the proximal metaphyses on both sides. Note the concentric reduction and well-molded greater trochanter mold to help prevent hip migration (*arrows*) (© 2001 American Academy of Orthopaedic Surgeons. Reprinted from the *Journal of the American Academy of Orthopaedic Surgeons*, volume 9(6), pp. 401–411 with permission)

with the complications of femoral nerve palsy from excessive flexion and osteonecrosis from excessive abduction [17]. The prevalence of osteonecrosis is higher when treating a dislocation versus dysplasia as well as when undergoing closed reduction and spica casting versus the Pavlik harness (Tables 2 and 3). The cause of osteonecrosis can result from excessive abduction leading to impingement of a branch of the medial femoral circumflex artery and pronounced increased intra-articular pressure constricting the intra-epiphyseal vessels of the femoral head [2, 19]. By placing the infant or child in less than 55–60° of abduction, it will reduce the risk of osteonecrosis [17, 37]. Complications specifically described to the Pavlik harness has been Pavlik harness disease and improper application of the harness by the parents. Pavlik harness disease results from the dislocated femoral head sitting up against the edge of the acetabulum leading to worsening of the acetabulum dysplasia via erosion of the posterolateral rim [17, 19, 38]. These complications are rare and avoidable by providing close follow-up and encouraging parental involvement [28].

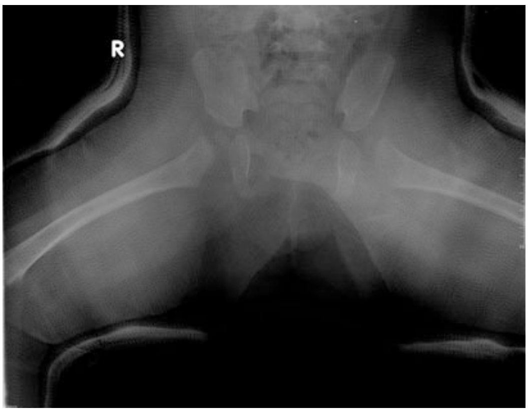


Fig. 11 Routine evaluation of the patient following closed reduction and spica casting involves plain film radiographs. Patient demonstrates sustained reduction of the right hip in the spica cast

The clinical outcomes of the Pavlik harness reported in the orthopedic literature have a success rate between 53 % and 99 % (Table 2) [14, 39–46]. In general, the accepted success rate for infants under the age of 6 months with a dysplastic hip is greater than 90 % with a recurrence rate of approximately 10 % [17]. When the infant has a dislocated hip, the success rate is expected to drop to 85 % with further declines correlating with an increasing age of the child [17, 28]. Due to multiple factors, there is a high variability in the success rate of closed reduction and spica casting. Approximately 22–66 % of these children will require a future intervention (Table 3) [47–50]. Given the possibility of

Table 2 Outcome and complication rate of Pavlik harness

Author	Number of hips	Average age	Average follow-up	Rate of success (%)	Rate of osteonecrosis (%)	Other complications
Filipe et al.	112	NR	NR	94	4	
Harding et al.	55	1 month	23 months	53	NR	
Harris et al.	720	2 months	25 months	89	1	1 %, transient pain or limited ROM of the hip
Iwasaki[a]	201	4 months	>1 year	84	7	
Johnson et al.	91	2 months	8 months	99	0	1 %, skin breakdown from the harness straps
Kalamchi et al.	122	5 months	5 years	97	0	
Pavlik[b]	632	NR	NR	84	0	
Ramsey et al.	27	<6 months	3 years	89	NR	
Suzuki et al.	233	4 months	NR	94	16	

[a]Only reporting outpatient results
[b]Only reporting dislocated hips

Table 3 Outcome and complication rate of closed reduction and spica casting

Author	Number of hips	Average age	Average follow-up	Converted to open reduction (%)	Rate of additional treatment (%)[a]	Rate of osteonecrosis (%)[a]
Ishii et al.	40	<1 year	15 years	NR	NR	30
Kahle et al.	47	NR	43 months	43	22	7
Schoenecker et al.	38	23 months	>3 years	39	52	4
Zionts et al.	51	20 months	12 years	25	66	3

[a]Rate calculation excludes hips converted to open reduction

recurrence or need for further action after either treatment, the children should be followed periodically until maturation.

Summary

Developmental dysplasia of the hip is a common pediatric condition. Timely diagnosis and treatment of hip dysplasia or dislocation are critical to prevent disability and needed for the morbid procedure of an open reduction with a femoral or acetabular osteotomy. Primarily, the nonsurgical treatment options include the Pavlik harness and closed reduction with spica casting. The preferred treatment method is based on age of the patient, stability of the hip, the ability to reduce the dislocated hip, and the degree of acetabular dysplasia, but the most important factor is patient age. Children younger than 6 months of age are ideally treated with the Pavlik harness, which has provided rates of reduction as high as 99 % in the orthopedic literature. When children are between the ages of 6 and 18 months, they are ideal candidates for closed reduction and spica casting. As children grow older, they have a diminished capacity to remodel the acetabulum that results in the need for surgical treatment. Once the children are older than 18 months, they will nearly always require surgical treatment. Despite success of nonsurgical treatment options, proper positioning of the hip in the Pavlik harness or spica cast is necessary to prevent associated complications.

References

1. Dezateux C, Rosendahl K. Developmental dysplasia of the hip. Lancet. 2007;369(9572):1541–52.
2. Hefti F, Brunner R, Hasler CC, Jundt G. Pediatric orthopedics in practice. Heidelberg/New York: Springer Medizin; 2007. http://dx.doi.org/10.1007/978-3-540-69964-4.
3. Weinstein SL, Mubarak SJ, Wenger DR. Developmental hip dysplasia and dislocation: part I. Instr Course Lect. 2004;53:523–30.
4. Rosendahl K, Markestad T, Lie RT. Ultrasound screening for developmental dysplasia of the hip in the neonate: the effect on treatment rate and prevalence of late cases. Pediatrics. 1994;94(1):47–52.
5. Holen KJ, Tegnander A, Bredland T, et al. Universal or selective screening of the neonatal hip using ultrasound? A prospective, randomised trial of 15,529 newborn infants. J Bone Jt Surg Br Vol. 2002;84(6):886–90.
6. Malvitz TA, Weinstein SL. Closed reduction for congenital dysplasia of the hip. Functional and radiographic results after an average of thirty years. J Bone Jt Surg Am Vol. 1994;76(12):1777–92.
7. Salter RB, Dubos JP. The first fifteen year's personal experience with innominate osteotomy in the treatment of congenital dislocation and subluxation of the hip. Clin Orthop Relat Res. 1974;98:72–103.
8. Weinstein SL, Mubarak SJ, Wenger DR. Developmental hip dysplasia and dislocation: part II. Instr Course Lect. 2004;53:531–42.
9. Pavlik A. A harness for treatment of congenital hip dislocation in infants. Acta chirurgiae orthopaedicae et traumatologiae Cechoslovaca. 1953;20(5–6):93–100.
10. Pavlik A. Method of functional therapy with strap braces as a principle of conservative therapy of congenital dislocation of the hip in infants. Zeitschrift fur Orthopadie und ihre Grenzgebiete. 1957;89(3):341–52.
11. Pavlik A. Functional treatment of congenital femur dislocation in infants. Acta chirurgiae orthopaedicae et traumatologiae Cechoslovaca. 1955;22(1–2):33–40.

12. Pavlik A. Experience with the treatment of congenital hip dysplasias by active movements in stirrups. Acta chirurgiae orthopaedicae et traumatologiae Cechoslovaca. 1959;26:432–5.
13. Pavlik A. Stirrups as an aid in the treatment of congenital dysplasias of the hip in children. By Arnold Pavlik, 1950. J Pediatr Orthop. 1989;9(2):157–9.
14. Pavlik A. The functional method of treatment using a harness with stirrups as the primary method of conservative therapy for infants with congenital dislocation of the hip. 1957. Clin Orthop Relat Res. 1992;281:4–10.
15. Pavlik A. To the question of originality of treatment of congenital hip dysplasias by active movement in the stirrups. 1959. J Pediatr Orthop Part B. 2001;10(3):165–8.
16. Guille JT, Pizzutillo PD, MacEwen GD. Development dysplasia of the hip from birth to six months. J Am Acad Orthop Surg. 2000;8(4):232–42.
17. Lieberman JR, American Academy of Orthopaedic Surgeons. AAOS comprehensive orthopaedic review. Rosemont: American Academy of Orthopaedic Surgeons; 2009.
18. Lovell WW, Winter RB, Morrissy RT, Weinstein SL. Lovell and Winter's pediatric orthopaedics. 4th ed. Philadelphia: Lippincott-Raven; 1996.
19. Miller MD, Hart JA. Review of orthopaedics. 5th ed. Philadelphia: Saunders/Elsevier; 2008.
20. Graf R. The diagnosis of congenital hip-joint dislocation by the ultrasonic Combound treatment. Arch Orthop Trauma Surg. 1980;97(2):117–33.
21. Roposch A, Graf R, Wright JG. Determining the reliability of the Graf classification for hip dysplasia. Clin Orthop Relat Res. 2006;447:119–24.
22. Vitale MG, Skaggs DL. Developmental dysplasia of the hip from six months to four years of age. J Am Acad Orthop Surg. 2001;9(6):401–11.
23. Race C, Herring JA. Congenital dislocation of the hip: an evaluation of closed reduction. J Pediatr Orthop. 1983;3(2):166–72.
24. Lindstrom JR, Ponseti IV, Wenger DR. Acetabular development after reduction in congenital dislocation of the hip. J Bone Jt Surg Am Vol. 1979;61(1):112–8.
25. Harris NH. Acetabular growth potential in congenital dislocation of the hip and some factors upon which it may depend. Clin Orthop Relat Res. 1976;119:99–106.
26. von Rosen S. Diagnosis and treatment of congenital dislocation of the hip joint in the new-born. J Bone Jt Surg Br Vol. 1962;44-B:284–91.
27. Suzuki S. Reduction of CDH by the Pavlik harness. Spontaneous reduction observed by ultrasound. J Bone Jt Surg Br Vol. 1994;76(3):460–2.
28. Mubarak S, Garfin S, Vance R, McKinnon B, Sutherland D. Pitfalls in the use of the Pavlik harness for treatment of congenital dysplasia, subluxation, and dislocation of the hip. J Bone Jt Surg Am Vol. 1981;63(8):1239–48.
29. Viere RG, Birch JG, Herring JA, Roach JW, Johnston CE. Use of the Pavlik harness in congenital dislocation of the hip. An analysis of failures of treatment. J Bone Jt Surg Am Vol. 1990;72(2):238–44.
30. Tibrewal S, Gulati V, Ramachandran M. The Pavlik method: a systematic review of current concepts. J Pediatr Orthop Part B. 2013;22(6):516–20.
31. Suzuki S, Seto Y, Futami T, Kashiwagi N. Preliminary traction and the use of under-thigh pillows to prevent avascular necrosis of the femoral head in Pavlik harness treatment of developmental dysplasia of the hip. J Orthop Sci Off J Jpn Orthop Assoc. 2000;5(6):540–5.
32. Kitoh H, Kawasumi M, Ishiguro N. Predictive factors for unsuccessful treatment of developmental dysplasia of the hip by the Pavlik harness. J Pediatr Orthop. 2009;29(6):552–7.
33. Inoue T, Naito M, Nomiyama H. Treatment of developmental dysplasia of the hip with the Pavlik harness: factors for predicting unsuccessful reduction. J Pediatr Orthop Part B. 2001;10(3):186–91.
34. Gage JR, Winter RB. Avascular necrosis of the capital femoral epiphysis as a complication of closed reduction of congenital dislocation of the hip. A critical review of twenty years' experience at Gillette Children's Hospital. J Bone Jt Surg Am Vol. 1972;54(2):373–88.
35. Brougham DI, Broughton NS, Cole WG, Menelaus MB. Avascular necrosis following closed reduction of congenital dislocation of the hip. Review of influencing factors and long-term follow-up. J Bone Jt Surg Br Vol. 1990;72(4):557–62.
36. Skaggs DL, Kaminsky C, Tolo VT, Kay RM, Reynolds RA. Variability in measurement of acetabular index in normal and dysplastic hips, before and after reduction. J Pediatr Orthop. 1998;18(6):799–801.
37. Smith BG, Millis MB, Hey LA, Jaramillo D, Kasser JR. Postreduction computed tomography in developmental dislocation of the hip: part II: predictive value for outcome. J Pediatr Orthop. 1997;17(5):631–6.
38. Jones GT, Schoenecker PL, Dias LS. Developmental hip dysplasia potentiated by inappropriate use of the Pavlik harness. J Pediatr Orthop. 1992;12(6):722–6.
39. Suzuki S, Yamamuro T. Avascular necrosis in patients treated with the Pavlik harness for congenital dislocation of the hip. J Bone Jt Surg Am Vol. 1990;72(7):1048–55.
40. Ramsey PL, Lasser S, MacEwen GD. Congenital dislocation of the hip. Use of the Pavlik harness in the child during the first six months of life. J Bone Jt Surg Am Vol. 1976;58(7):1000–4.
41. Kalamchi A, MacFarlane 3rd R. The Pavlik harness: results in patients over three months of age. J Pediatr Orthop. 1982;2(1):3–8.
42. Johnson AH, Aadalen RJ, Eilers VE, Winter RB. Treatment of congenital hip dislocation and dysplasia with the Pavlik harness. Clin Orthop Relat Res. 1981;155:25–9.
43. Iwasaki K. Treatment of congenital dislocation of the hip by the Pavlik harness. Mechanism of reduction and usage. J Bone Jt Surg Am Vol. 1983;65(6):760–7.

44. Harris IE, Dickens R, Menelaus MB. Use of the Pavlik harness for hip displacements. When to abandon treatment. Clin Orthop Relat Res. 1992;281:29–33.
45. Harding MG, Harcke HT, Bowen JR, Guille JT, Glutting J. Management of dislocated hips with Pavlik harness treatment and ultrasound monitoring. J Pediatr Orthop. 1997;17(2):189–98.
46. Filipe G, Carlioz H. Use of the Pavlik harness in treating congenital dislocation of the hip. J Pediatr Orthop. 1982;2(4):357–62.
47. Zionts LE, MacEwen GD. Treatment of congenital dislocation of the hip in children between the ages of one and three years. J Bone Jt Surg Am Vol. 1986;68 (6):829–46.
48. Schoenecker PL, Dollard PA, Sheridan JJ, Strecker WB. Closed reduction of developmental dislocation of the hip in children older than 18 months. J Pediatr Orthop. 1995;15(6):763–7.
49. Kahle WK, Anderson MB, Alpert J, Stevens PM, Coleman SS. The value of preliminary traction in the treatment of congenital dislocation of the hip. J Bone Jt Surg Am Vol. 1990;72(7):1043–7.
50. Ishii Y, Ponseti IV. Long-term results of closed reduction of complete congenital dislocation of the hip in children under one year of age. Clin Orthop Relat Res. 1978;137:167–74.

30 Surgical Technique: Anterior Open Reduction for Developmental Dysplasia of the Hip and Salter Innominate Osteotomy

Wudbhav N. Sankar

Contents

Abstract

An open reduction is indicated for those developmental hip dislocations that have failed conservative attempts at reduction including Pavlik harness and closed reduction or for those children who present initially at an older age. The goal of an open reduction is to remove the obstacles blocking the access of the femoral head to the depths of the acetabulum. Achieving a deep and concentric reduction minimizes the risk of re-dislocation and maximizes future remodeling of the hip joint. The anterior approach is the most common technique used to perform an open reduction. This exposure also allows a pelvic osteotomy to be performed (if necessary) to augment coverage and facilitate acetabular remodeling. A concomitant femoral shortening osteotomy may need to be performed if excessive tension is present at the time of open reduction, doing so reduces the risk of avascular necrosis. In addition, femoral version should be assessed to determine the need for femoral derotation.

Introduction

An open reduction for a congenital or developmental dislocation of the hip is indicated for younger infants who have failed more conservative measures to relocate the femoral head within the acetabulum including a Pavlik harness, abduction

W.N. Sankar
Division of Orthopaedic Surgery, Perelman School of Medicine, University of Pennsylvania, Children's Hospital of Philadelphia, Philadelphia, PA, USA
e-mail: sankarw@email.chop.edu

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_31

brace, or closed reduction and spica casting. In general, an open reduction becomes more likely after a year of age and is certainly the treatment of choice beyond 18 months [1, 3, 5]. Because the natural history of a dislocated hip is superior to that of a subluxated or severely dysplastic hip and because acetabular remodeling potential decreases with age, there should be an upper age limit for attempting an open reduction [9]. The exact threshold is somewhat controversial, but in general, one should consider *not* performing surgery in children older than 5 or 6 with bilateral dislocations and in children older than 8 with unilateral dislocations.

A concomitant pelvic osteotomy should be performed at the time of open reduction for most infants older than 18 months and in those younger infants in whom additional coverage is needed to maintain femoral head stability [5]. The pelvic osteotomy that is classically performed in conjunction with open reduction for the treatment of DDH is Salter innominate osteotomy, which provides anterior and lateral femoral head coverage and promotes remodeling of the acetabulum [5]. Alternatively, a Pemberton or Dega pelvic osteotomy (discussed in chapter on Neuromuscular Hip Disorders: Focus on Cerebral Palsy) can be performed to achieve similar goals.

The primary indication for performing a concomitant femoral osteotomy along with an open reduction is when excessive tension is needed to bring the femoral head into the acetabulum [6, 8]. In this situation, performing a femoral shortening osteotomy can allow a tension-free reduction, which reduces the risk of avascular necrosis. The second indication for performing a femoral osteotomy is when excessive femoral anteversion is present. In this situation, derotation of the femur can help center the femoral head within the acetabulum, improving the quality of reduction and the position of spica casting [6]. The author prefers to derotate the femur whenever the anteversion is estimated to exceed 50°. Varus correction as part of a femoral osteotomy is generally not needed for hip stability. Unnecessary varus of the proximal femur can cause a Trendelenburg gait and abductor dysfunction and is typically avoided except in unusual cases.

Planning and Positioning

After induction and prior to positioning and draping, the child is turned 90° while supine on the operating table such that the affected limb can be flexed 90° at the knee over the end of the bed. The femur is then rotated externally until the ossific nucleus lines up with the femoral shaft on the image intensifier (Fig. 1). Femoral anteversion can be estimated by subtracting the amount of external rotation necessary to obtain this view from 90°. As mentioned, the author generally performs a femoral derotation whenever the anteversion exceeds 50°, or if excessive internal hip rotation is needed to obtain stability intraoperatively.

For the surgical procedure, the patient is positioned supine on a radiolucent operating table with a small bump placed under the ipsilateral iliac crest. Split drapes are used to allow access to the entire hemipelvis with care taken to place the posterior limb as posterior as possible to

Fig. 1 Estimating femoral anteversion

prevent draping out the ilium. Paralysis is avoided to facilitate identification of the femoral nerve if need be.

If the patient has already failed an attempt at closed reduction at a prior date, then preoperative arthrography is typically not performed. If this is not the case, an arthrogram can be performed to determine whether closed treatment is possible or if the open reduction is necessary. In general, the need for an open reduction increases beyond a year of age [1, 3, 5].

Open Reduction

A 6–8 cm incision is made parallel and 1 cm distal to the iliac crest, with approximately 3 cm of the incision extending medial to the anterior superior iliac spine (ASIS) (Fig. 2). The subcutaneous tissues are divided in line with the incision to expose the iliac crest and its overhanging external oblique muscle. Using Bovie cautery, the obliques can be feathered up and off the crest, taking care not to burn too much of the apophyseal cartilage (Fig. 3).

Next, the Smith-Peterson interval is found on a line connecting the ASIS and the patella, usually 1–2 cm distal to the ASIS itself. It is helpful to palpate the muscle bellies of the sartorius and the tensor fascia lata and find the soft depression in between. One can then spread bluntly down through the interval to the level of the rectus tendon (Fig. 4). Alternatively, one can access the interval by opening the sheath of the tensor fascia lata a few millimeters lateral to the interval itself. Once opened, the muscle belly can be retracted laterally allowing entry into interval. This approach is theoretically more protective to the lateral femoral cutaneous nerve and has been described for periacetabular osteotomies. Visualization of the direct head of the rectus itself can be improved by incising the thin fascia overlying the tendon.

Using a 15 blade scalpel, the iliac crest apophysis is then split down its midline from the ASIS to the point at which the crest begins to swing back medially. The medial and lateral halves of the apophysis can then be “popped” off of the boney ilium using fingers or a freer elevator

Fig. 2 Incision

Fig. 3 Exposure of iliac crest apophysis

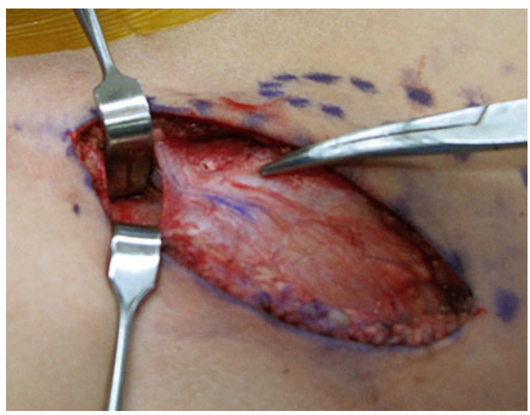


Fig. 4 Smith-Peterson interval

(Fig. 5). Sponges may be packed subperiosteally along both the inner and outer tables.

The iliac exposure and the Smith-Peterson interval are now connected by sharply dissecting

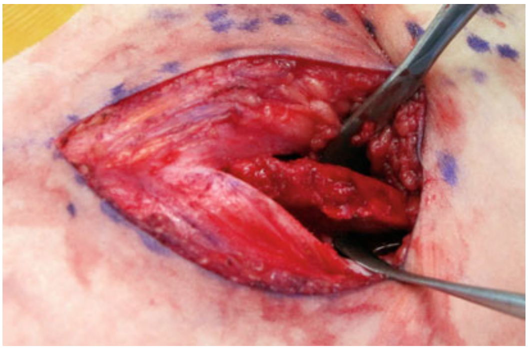


Fig. 5 Splitting of apophysis to reveal underlying iliac crest

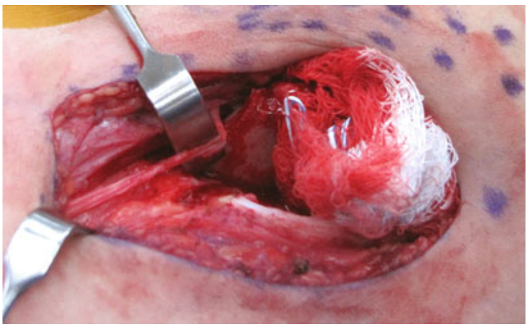


Fig. 6 Lateral femoral cutaneous nerve

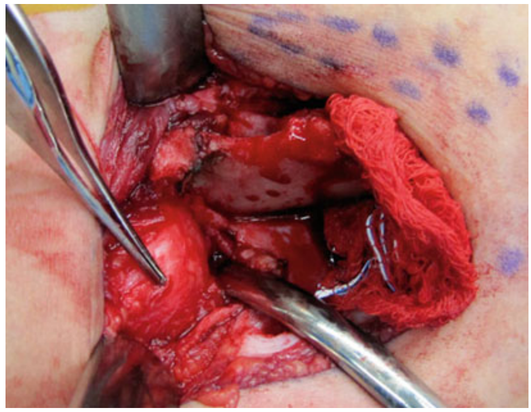


Fig. 7 Developing the pericapsular interval with Crego. Forceps point to anterior capsule overlying femoral head

Fig. 8 Psoas tendon

along the prow of the ilium connecting the ASIS and the anterior inferior iliac spine (AIIS). Care should be taken at this point to protect the lateral femoral cutaneous nerve, which passes from medial proximal to distal lateral (Fig. 6). Once identified, the nerve should be mobilized and retracted medially. The direct head of the rectus should be fully defined and then transected just distal to its insertion on the AIIS. It can be tagged for later repair.

The anterior capsule most now be exposed using a Cobb elevator or sharp dissection to clear off residual fibers of the iliocapsularis muscle. Proximally and laterally, one must define the interval overlying the capsule but underneath the abductor complex. This is best done with a Crego elevator using it to not only enter the interval but to define it medially all the way to the upper border of the false acetabulum (Fig. 7). The periosteum overlying the outer table of the ilium can now be elevated more distally through the pseudo-acetabulum. The anterior and superior exposure of the capsule is completed by partially releasing the junction of the lateral subperiosteal interval and the pericapsular interval from front to back using a curved Mayo scissor.

Working medially, the iliopsoas is identified as it crosses into the pelvis just on the medial size of the periosteum overlying the inner table. The tendon lies distal to the pubis and posterior in relation to the muscle belly itself. It is best exposed by rolling the muscle medially on itself using forceps, and then capturing the tendon with a right angle clamp inserted from medial to lateral (Fig. 8). If there is any confusion, the psoas tendon can be distinguished from the femoral nerve by noting muscle fibers entering the structure, checking that it tightens with internal rotation of the hip, and confirming that there is no reaction to light touch with Bovie cautery (assuming that the patient is not paralyzed). Release of the tendon may then be performed with cautery. Once released, retraction of the psoas becomes quite easy, and the hip capsule can now be exposed as medially as possible along and inferior to the pubic ramus.

The capsulotomy is performed in a “T” shape with the top of the incision running along the acetabular margin and the stem of “T” brought out horizontal roughly over the center of the dislocated femoral head (Fig. 9). It is important that the capsule be released as medial as possible to allow full exposure of the true acetabulum. Using a scalpel, the initial incision into the capsule is best performed medially over the empty acetabulum to prevent inadvertent injury to the articular cartilage of the femoral head. The capsulotomy can then be safely extended using scissors. The redundant corner may be excised to facilitate later capsulorrhaphy (Fig. 10).

The first structure to identify upon opening the hip joint is the ligamentum teres. This can be sharply transected off of the femoral head using a scalpel and then followed down to the true acetabulum before being excised with scissors (Fig. 11). The pulvinar can be removed from the acetabulum with a pituitary rongeur to expose the acetabular cartilage (Fig. 12). Finally, the transverse

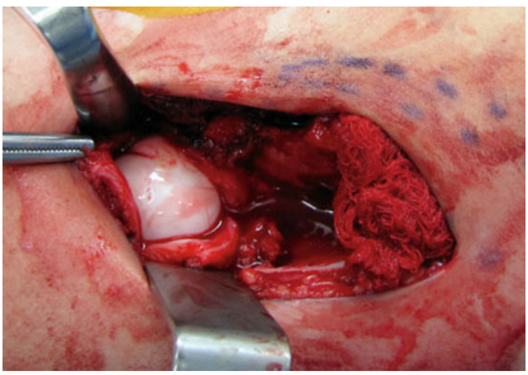


Fig. 9 Capsulotomy

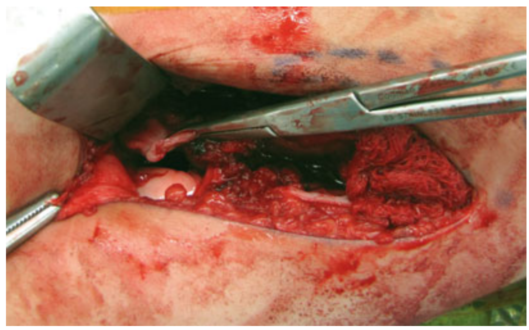


Fig. 11 The ligamentum teres is transected and followed into the true acetabulum

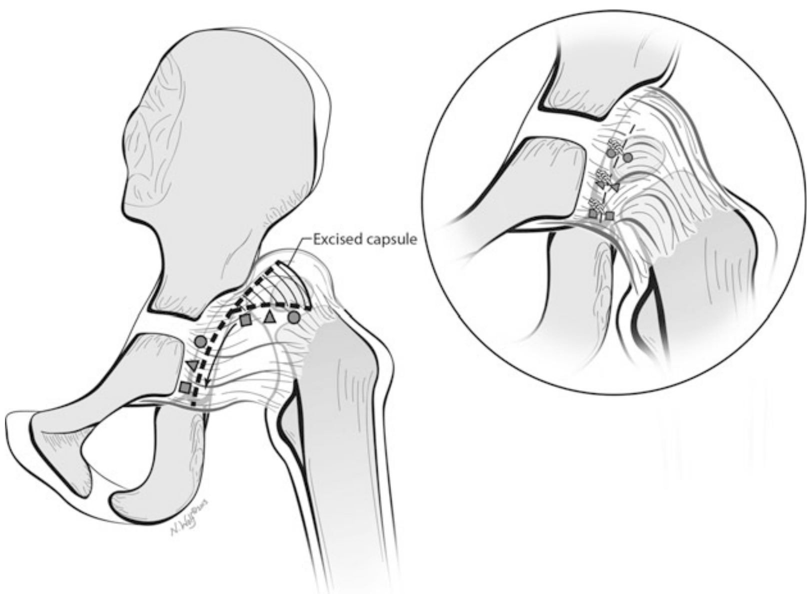


Fig. 10 Schematic of capsulotomy and repair

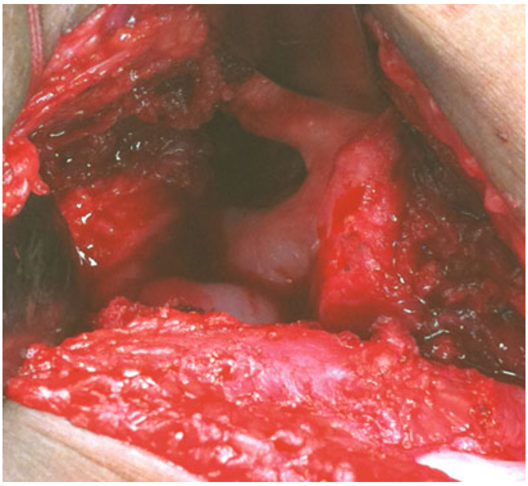


Fig. 12 Acetabular exposure

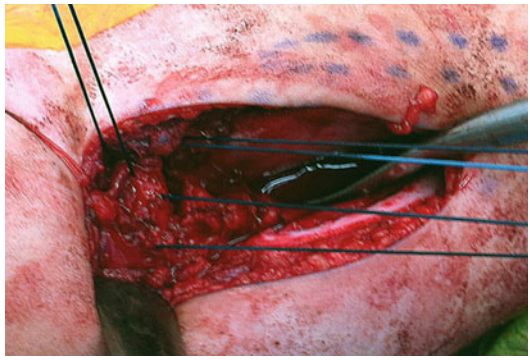


Fig. 13 Capsule sutures

acetabular ligament should be released from the depths of the acetabulum where it joins both ends of the labrum as it crosses the cotyloid fossa. This is an extremely important step to allow relaxation and expansion of the labrum, which widens the acetabular opening to accommodate the femoral head.

At this point a trial reduction can be performed. Generally the femoral head will reduce with a satisfying "clunk." The stability of the reduction can be assessed as well as the tension required to achieve reduction. If the adductors are felt to be tight, the adductor longus can be percutaneously transected with a beaver blade. At this point the surgeon must decide if the tension on the femoral head is excessive and whether a femoral shortening osteotomy is necessary. Regardless of whether or not concomitant osteotomies are planned, it is easiest to place capsular sutures at this time when the femoral head can be easily dislocated to allow wide access to the acetabulum. Three #2 Ethibond sutures or similar nonabsorbable material are placed as medial as possible on the superior capsule (on the acetabular side) and brought out lateral to the apex of the inferior capsular cut (on the femoral side) (Fig. 13). These are tagged but not tied at this time as it is best to tie the capsulorrhaphy after all osteotomies are performed and the femoral head and pelvis are in their final positions.

Femoral Osteotomy

If a femoral osteotomy is warranted, an approximately 7 cm incision is made overlying the lateral aspect of the proximal femur. Dissection is carried down through the subcutaneous tissues and the fascia lata incised in line with the incision. The vastus lateralis is elevated off of the lateral aspect of the femur in an "L" fashion using Bovie cautery with the posterior limb running a few millimeters anterior to the posterior margin of the muscle and the vertical limb running up the vastus ridge. Both cuts should leave a small cuff of muscle for later repair. The periosteum can now be split and elevated circumferentially around the femur using Crego elevators.

For the size patient that typically undergoes these procedures, a one third tubular plate is generally sufficient for fixation, although a larger 3.5 mm DCP plate or blade plate can also be used. The five-hole one third tubular plate is typically cut down to four holes with a bolt cutter to minimize exposure. The plate is applied to the lateral aspect of the femur such that it ends just at the trochanteric flare. The proximal two screw holes are drilled and measured, and a single screw is placed loosely in the most proximal hole. This allows the plate to be rotated out of the way for the osteotomy. An oscillating saw is used to perform a transverse cut between the 2nd and 3rd holes of the plate in the subtrochanteric region of the femur. Performing the osteotomy in this region is easier given the sizing of the patient and the typical need to customize the amount of femoral shortening. The distal fragment can be delivered out of the wound to allow the shortening cut. Generally it is sufficient to remove 1.5–2 cm of the femur, although an alternative technique is to reduce the femoral head into the acetabulum and resect the amount of overlap between the two femoral segments. The plate is now rotated back into position and the proximal screw tightened in place followed by placement of the second screw. If necessary, derotation of the femur can be performed by externally rotating the distal segment the desired amount. A Verbrugge clamp is used to reduce the plate to the shaft of the femur, and the distal two screws are drilled and placed. The incision is closed in a layered fashion including sutures in the fascia lata, dermal tissue, and subcutaneous layer.

Salter Innominate Osteotomy

To perform the Salter osteotomy, it is best to first confirm that the femoral head is reduced. The outer table of the ilium should then be exposed subperiosteally into the sciatic notch (Fig. 14). A matching channel is created along the inner table, although it is difficult to visualize the notch itself from this side because it is hidden by the pelvic brim. A right angle clamp can be used to confirm that the two exposures are continuous. Specialized Rang retractors can now be placed along the inner and outer table; these provide a safe slot for passage of the Gigli saw (Fig. 15). Alternatively, Chandler retractors can be used. The Gigli saw is passed from medial to lateral and pulled through.

To perform the iliac osteotomy, the arms of the Gigli saw should be positioned such that the cut will exit just proximal to the AIIS. It is often helpful to lower the bed when making the cut to allow better control. The classic technique for harvesting bone graft for the osteotomy involves cutting the anterior part of the iliac crest that includes the ASIS and prow of the ilium using an oscillating saw and trimming this into a 30° wedge. This technique, however, distorts the ilium and can potentially lead to abductor dysfunction. An alternative method is to harvest a triangular wedge from a more proximal location in the ilium; this does not affect the contour of the iliac crest, and the donor site is quickly filled in by the thick layers of periosteum on either side (Figs. 16 and 17).

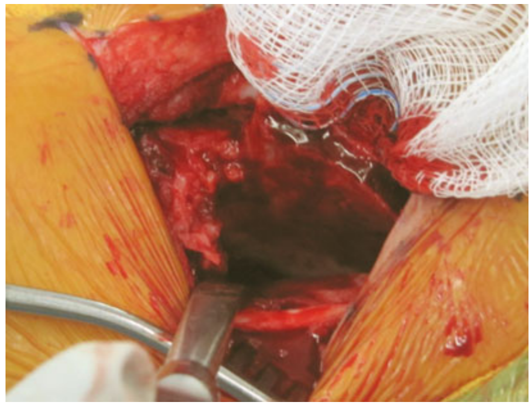


Fig. 14 Location of sciatic notch identified along outer table using Crego elevator

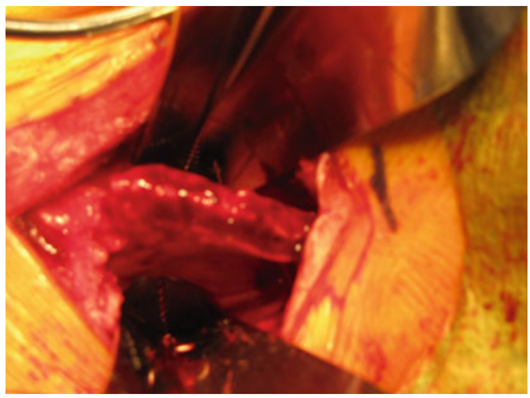


Fig. 15 Gigli saw is passed from medial to lateral through channel created by Rang retractors

The osteotomy is opened by applying a penetrating towel clip to the proximal iliac segment and the distal segment deep to the AIIS apophysis (Fig. 18). The 30° bone wedge is inserted along the medial edge of the osteotomy and fixed in place using two threaded Kirschner wires (Fig. 19). The image intensifier is used to confirm the length and position of the pins (away from the joint and the triradiate cartilage). The pins can be cut just above the bone or left longer if removal is planned.

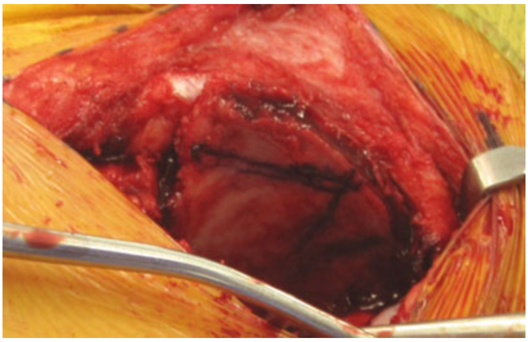


Fig. 16 A triangle of autograft can be taken from the anterior aspect of the crest or as a wedge from the proximal ilium

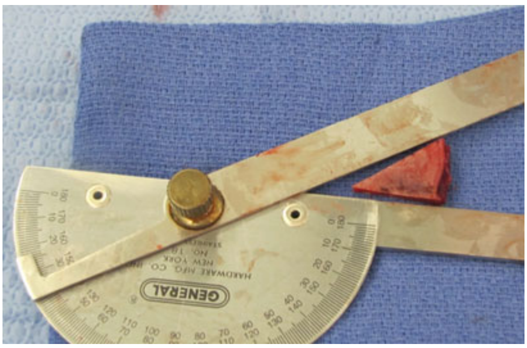


Fig. 17 Bone graft is typically a 30° wedge

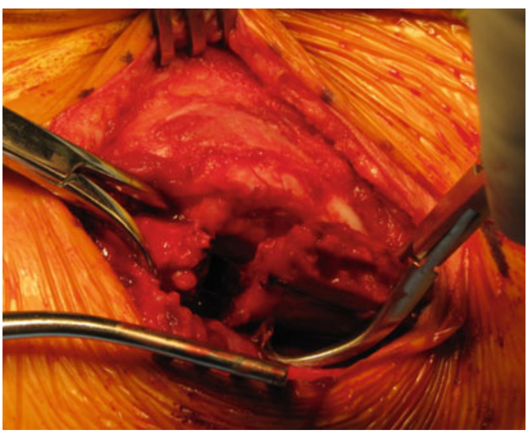


Fig. 18 Osteotomy site is opened using towel clamps

One criticism of the Salter osteotomy is the risk of anterior overcoverage and iatrogenic retroversion that can be result when the procedure is performed at an age in which the ossified rims of the acetabulum are difficult to visualize. This can

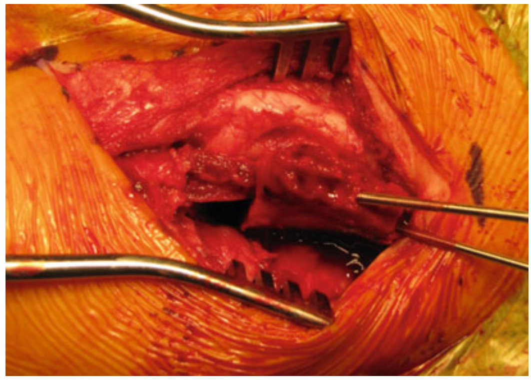


Fig. 19 Fixation using threaded K-wires

be minimized by not using an excessively large bone graft and by checking hip range of motion after correction. Alternatively, a Pemberton or Dega pelvic osteotomy (discussed in chapter on Neuromuscular Hip Disorders: Focus on Cerebral Palsy) can be performed.

Closure

After the necessary pelvic and/or femoral osteotomies have been performed, reduction is confirmed, and the limb is held with the hip in approximately 30° of flexion and 30° of abduction. The previously placed capsular sutures are tied and cut. The direct head of the rectus is repaired to the AIIS stump using a heavy absorbable suture. The apophysis of the iliac crest is repaired with a deep-deep, superficial-superficial double throw absorbable 0 or 1-0 suture, which improves apposition of the cartilage surfaces. The external oblique is repaired with a running 2-0 suture followed by the dermis and the subcutaneous layer.

Postoperative Management

Following open reduction, the child is placed into a one-and-a-half legged spica cast with the hip held in the same position of 30° of flexion, 30° of abduction, and slight internal rotation. The use of plaster allows a superior mold upward on the greater trochanter. A CT or MRI is done within a few hours of surgery to confirm reduction in the axial plane.

The cast is maintained for 6 weeks before being replaced with an abduction brace, which is worn full time for 2 additional weeks. Continued use of the abduction brace at night after this period may help continued acetabular remodeling.

Complications

The primary significant risks of an open reduction are re-dislocation, avascular necrosis, and need for additional surgery to address residual acetabular dysplasia. The incidence of re-dislocation following a technically proficient open reduction has been reported to be approximately 6 % [7]. Risk factors for re-dislocation include inadequate release of the capsule or transverse acetabular ligament, inadequate capsulorrhaphy, and abnormal femoral version [7]. Avascular necrosis (AVN) can be the most devastating complication of an open hip reduction with a wide frequency reported in the literature ranging from 3 % to 60 % [2, 4]. Mild forms of AVN may cause no clinical sequelae, while more extensive forms can cause significant morbidity. The risk of AVN is best minimized by meticulous surgical technique, appropriate femoral shortening, and proper positioning within the spica cast. Finally, the need for additional subsequent procedures to address residual dysplasia has been estimated to be 42 % and 49 % in two recent series [2, 4]. Even if a pelvic osteotomy is routinely performed after 18 months of age as advocated by Salter himself, continued follow-up through skeletal maturity is imperative to identify patients in need of further surgery to address residual acetabular dysplasia [5].

Case Example

A 2-year-old female presented with a limp. An AP pelvic radiograph clearly demonstrates a complete dislocation of the left hip (Fig. 20). She underwent an open reduction and Salter osteotomy but did not require a femoral osteotomy. AP pelvic radiograph taken 6 weeks after surgery in the spica cast demonstrates a reduced left hip with healing Salter osteotomy (Fig. 21). Three years after

Fig. 20 AP radiograph demonstrates left hip dislocation

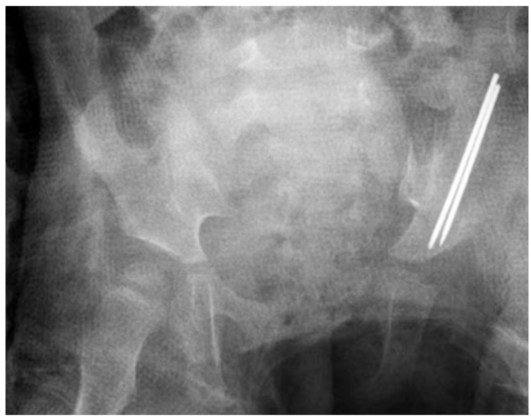


Fig. 21 AP radiograph in spica cast 6 weeks after open reduction and Salter osteotomy

surgery (and pin removal), a follow-up AP pelvis x-ray demonstrates a well-reduced left hip with excellent acetabular remodeling (Fig. 22).

Summary

An open reduction is the treatment of choice for those developmental hip dislocations that have failed conservative attempts at reduction including Pavlik harness and closed reduction, or for those children who present initially at an older age. The purpose of an open reduction is to remove the obstacles blocking the access of the femoral head to the depths of the acetabulum,

Fig. 22 AP radiograph 3 years following surgery (and pin removal)

which can include the capsule, pulvinar, psoas tendon, and transverse acetabular ligament among others. The goal is a deep and concentric reduction of the femoroacetabular articulation: this minimizes the risk of re-dislocation and maximizes future remodeling of the hip joint. The described anterior approach is the most common technique used to perform an open reduction; this exposure also allows a pelvic osteotomy to be performed (if necessary) to augment coverage and facilitate acetabular remodeling. A concomitant femoral shortening osteotomy may need to be performed if excessive tension is present at the time of open reduction, doing so reduces the risk of avascular necrosis. In addition, femoral version should be assessed to determine the need for femoral derotation.

References

1. Galpin RD, Roach JW, Wenger DR, et al. One-stage treatment of congenital dislocation of the hip in older children, including femoral shortening. J Bone Joint Surg Am. 1989;71(5):734–41.
2. Gholve PA, Flynn JM, Garner MR, Millis MB, Kim YJ. Predictors for secondary procedures in walking DDH. J Pediatr Orthop. 2012;32:282–9.
3. Haidar RK, Jones RS, Vergroesen DA, et al. Simultaneous open reduction and Salter innominate osteotomy for developmental dysplasia of the hip. J Bone Joint Surg Br. 1996;78(3):471–6.
4. Holman J, Carroll KL, Murray KA, MacLeod LM, Roach JW. Long term follow-up of open reduction surgery for developmental dislocation of the hip. J Pediatr Orthop. 2012;32:121–4.
5. Salter RB. Role of innominate osteotomy in the treatment of congenital dislocation and subluxation of the hip in the older child. J Bone Joint Surg Am. 1966;48:1413.
6. Sankar WN, Neubuerger CO, Moseley CF. Femoral anteversion in developmental dysplasia of the hip. J Pediatr Orthop. 2009;29(8):885–8.
7. Sankar WN, Young CR, Lin AG, Crow SA, Baldwin KD, Moseley CF. Risk factors for failure after open reduction for DDH: a matched cohort analysis. J Pediatr Orthop. 2011;31:232–9.
8. Wenger DR, Lee CS, Kolman B. Derotational femoral shortening for developmental dislocation of the hip: special indications and results in the child younger than 2 years. J Pediatr Orthop. 1995;15:768–79.
9. Zionts LE, MacEwen GD. Treatment of congenital dislocation of the hip in children between the ages of one and three years. J Bone Joint Surg Am. 1986;68(6):829–46.

31 Surgical Technique: Modified Bernese Triple Innominate Osteotomy

Wudbhav N. Sankar

Contents

W.N. Sankar
Division of Orthopaedic Surgery, Perelman School of Medicine, University of Pennsylvania, Children's Hospital of Philadelphia, Philadelphia, PA, USA
e-mail: sankarw@email.chop.edu

Abstract

The triple innominate osteotomy is a complete redirectional acetabular osteotomy, consisting of cuts through the ilium, ischium, and pubis. This osteotomy spares the triradiate cartilage and allows the acetabulum to be completely freed from the rest of the pelvis which in turn allows the surgeon to obtain large corrections and control the final position of the acetabulum in multiple planes. This procedure is best indicated for skeletally immature patients with congruent or near-congruent joints that require large degrees of correction and/or changes in acetabular version. The modified Bernese triple osteotomy is performed through two incisions. The medial approach allows access for the ischial osteotomy which is oriented toward the proximal aspect of the ischial spine. The pubis is cut with a Gigli saw and is accessed through the interval between the rectus femoris and iliacus. The ilium is osteotomized in a modified manner from the typical Salter osteotomy. Once mobilized, the acetabulum is carefully positioned with the help of a Schanz screw and pointed tenaculum before definitive fixation is achieved with long 3.5 mm pelvic screws.

Introduction

The triple innominate pelvic osteotomy is indicated for the school-aged child or young adolescent with acetabular dysplasia who is still

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_32

skeletally immature. First described by Le Coeur and later popularized by Steel and Tönnis, the triple innominate osteotomy involves complete cuts in the ilium, ischium, and pubis that spare the triradiate cartilage (unlike the Ganz periacetabular osteotomy which crosses the triradiate growth center) [1–3]. By doing so, the acetabulum is completely freed from the rest of the pelvis, which allows the surgeon to obtain large corrections and control the final position of the acetabulum in multiple planes. The triple osteotomy is a true redirectional pelvic osteotomy; unlike reshaping procedures like the Pemberton or the Dega, it does not change the size or shape of the acetabulum but does reorient the acetabulum in space [4]. Because the acetabulum is rotated around the femoral head to increase coverage, triple osteotomies require congruency or near congruency of the joint.

Triple osteotomies are best indicated in older children and adolescents with open triradiates who would not achieve sufficient correction from a Salter or Dega osteotomy, but whose acetabular growth potential may preclude a periacetabular osteotomy (which cuts directly through the triradiate cartilage). The exact age at which a PAO may be safely performed instead of a triple osteotomy is somewhat uncertain, but it is known that posttraumatic acetabular dysplasia occurs rarely after the age of 10 years, suggesting that the growth contribution of even an open physis is not significant at that age.

Triple osteotomies (similar to the PAO in the skeletally matured patient) are the osteotomies of choice for children in whom the surgeon wants to correct acetabular version or address dysplasia in the setting of a hypoplastic acetabulum. In this latter situation, a reshaping osteotomy would not improve lateral coverage but would only succeed in further reducing the volume of the acetabulum and placing excessive pressure on the femoral head [5]. In contrast, the triple osteotomy can reposition the existing sourcil (albeit short) more centrally over the weight-bearing zone, thereby improving coverage.

Planning and Positioning

After induction with general anesthesia, an epidural is typically placed for postoperative analgesia. Paralysis is avoided as both the sciatic nerve and obturator nerve are at risk during the procedure. The patient is then positioned supine on a radiolucent operating table. A bump is generally not used as it can distort intraoperative imaging. Both arms are positioned out laterally to facilitate access to the proximal pelvis by the surgeon (on the ipsilateral side) and the image intensifier (from contralateral side). Split drapes are used to allow access to the entire hemipelvis with care taken to place the posterior limb as posterior as possible to prevent draping out the ilium. In addition, the medial limb should be medial enough to allow access to the adductor region.

Medial Exposure and Ischial Cut

With the limb in a frog position, the first of two incisions needed to perform the modified Bernese triple osteotomy is made approximately 5 cm long, in line with the adductor longus muscle [6] (Fig. 1). The fascia is split in line with the skin incision, and the adductor longus tendon is isolated and retracted proximally to reveal the underlying adductor brevis (Fig. 2). The anterior branch of the obturator nerve must then be identified, mobilized, and traced proximally to the point at which it exits the pelvis through the obturator foramen. The anterior surface of the ischium lies just lateral to this point. A curved clamp is used to enlarge this opening along the lateral side of the nerve, and a lane retractor is passed through the foramen along the periosteum of the quadrilateral plate until it “hooks” on the ischial spine (Fig. 3). The image intensifier can be used with both anteroposterior (AP) and false profile views to confirm appropriate position of the lane. The lateral edge of the ischium should be palpated and a second lane passed around the edge in a similar trajectory as the first. Peanuts can now be used to

Fig. 1 Medial skin incision for ischial cut (Reproduced with permission from the Division of Orthopaedic Surgery, Children's Hospital of Philadelphia)

Fig. 2 The adductor longus tendon (*AL*) is identified and retracted proximally to reveal the underlying anterior branch of the obturator nerve (*ON*) (Reproduced with permission from the Division of Orthopaedic Surgery, Children's Hospital of Philadelphia)

clear fibers of the obturator externus as well as a leash of vessels off the periosteal surface of the ischium.

The correct starting point for the ischial cut is just inferior to the lip of the acetabulum; this can both be palpated and confirmed with the false profile view [6]. The cut should be aimed toward the proximal aspect of the ischial spine, as this negates the ability of the sacrospinous ligament to alter the mobility of the acetabular fragment (Fig. 4). A straight Ganz osteotome is used to make the osteotomy, starting on the medial side of the ischium followed by a second pass to cut the lateral side (Fig. 5). The position and depth of the cut can be periodically confirmed on the false profile view; throughout, the osteotome should be externally rotated such that the blade is on the "edge" on the image intensifier (Fig. 6). This results in a cut that is somewhat oblique in the coronal plane, angling from proximal medial to distal lateral. It is important that the medial lane retractor be kept in place during the medial cut to protect the contents of the sciatic notch. Similarly, the lateral lane retractor should be in place when cutting the lateral aspect of the ischium. Although use of these retractors obscures visualization on the image intensifier, the position of the osteotome can be confirmed by periodically withdrawing the lane (Fig. 7). Completion of the cut is confirmed by placing a Cobb in the osteotomy site and twisting to confirm fragment mobility. The wound is packed with a moist sponge in the event that the cut needs to be revisited.

Anterior Exposure and Pubis Cut

Similar to the exposure for an open reduction of the hip, an 8 cm incision is made parallel and 1–2 cm distal to the iliac crest, with approximately

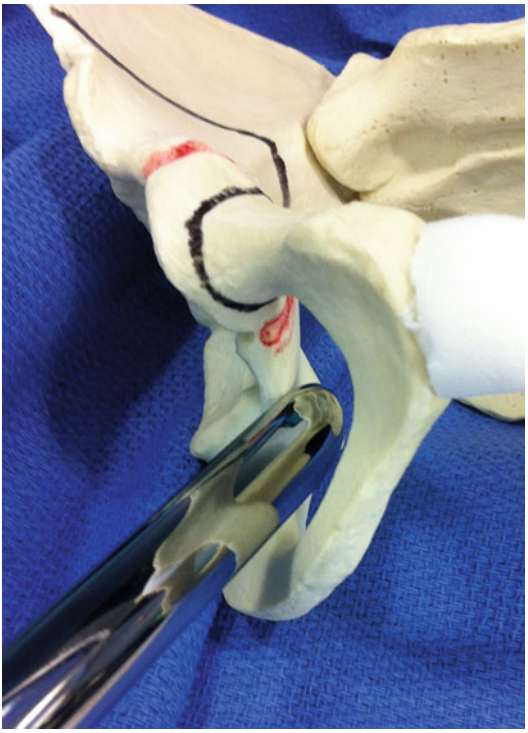


Fig. 3 The lane retractor is slid just lateral to the nerve through the obturator foramen along the quadrilateral plate until it "hooks" on the ischial spine (Reproduced with permission from the Division of Orthopaedic Surgery, Children's Hospital of Philadelphia)

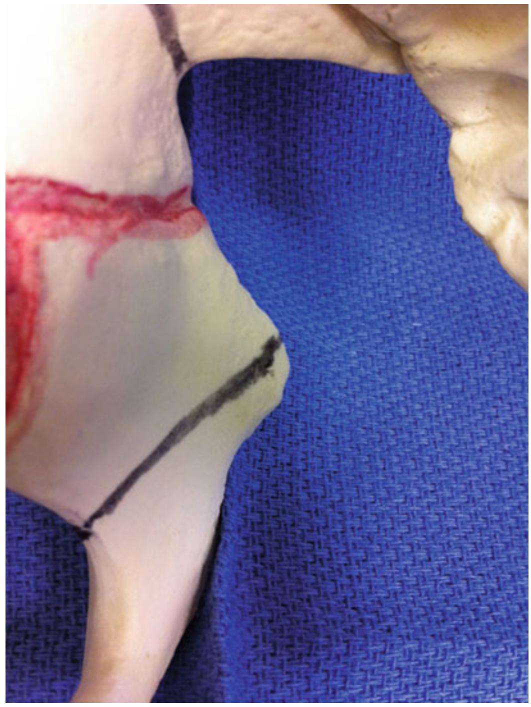


Fig. 4 The ischial cut is aimed toward the proximal aspect of the ischial spine (Reproduced with permission from the Division of Orthopaedic Surgery, Children's Hospital of Philadelphia)

3 cm of the incision extending medial to the anterior superior iliac spine (ASIS) (Fig. 8). The subcutaneous tissues are divided in line with the incision to expose the ilium; the overhanging external oblique muscle is feathered up and over the crest using bovie cautery.

Next, the Smith-Peterson interval is entered indirectly by incising the fascia overlying the tensor fascia lata muscle belly at approximately a 30° angle to the ASIS. The muscle can be separated from the medial compartment fascia and retracted laterally. One can then bluntly dissect down through the interval to the level of the rectus tendon. This indirect approach to the interval helps minimize risk of injury to the lateral femoral cutaneous nerve. Visualization of the direct head of the rectus itself can be improved by incising the thin fascia overlying the tendon.

Using a no. 15 blade scalpel, the iliac crest apophysis is then split down its midline from the ASIS to the point at which the crest begins to swing back medially (Fig. 9). Only the medial half of the apophysis is elevated with a Cobb or Freer elevator in a subperiosteal fashion down the inner table of the iliac wing. Minimizing exposure of the outer table helps preserve abductor function postoperatively. The iliac exposure and the Smith-Peterson interval are now connected by sharply dissecting along the prow of the ilium connecting the ASIS and the anterior inferior iliac spine (AIIS). In spite of the indirect access to the interval, care should still be taken at this point to protect the lateral femoral cutaneous nerve, which can cross the surgical field from medial proximal to distal lateral. If identified, the nerve should be mobilized and retracted medially.

The most bloodless interval with which to access the pubis is between the medial edge of the rectus tendon and the iliacus muscle (Fig. 10). The iliacus can sometimes adhere to the underlying periosteum, but lifting up and away with a Hibbs or Aufranc retractor can help clearly define

Fig. 5 A straight Ganz osteotome is used to make the ischial osteotomy, starting on the medial side of the ischium followed by a second pass to cut the lateral side. A lane is used for protection (Reproduced with permission from the Division of Orthopaedic Surgery, Children's Hospital of Philadelphia)

the surgical plane. Sharp extraperiosteal dissection with a scalpel can release any remaining muscle fibers. The psoas can then be retracted to reveal the pubic eminence, an important landmark that constitutes the next "bump" medial to the AIIS. In order to avoid injury to the triradiate cartilage and the acetabulum, the pubis cut must stay medial to this structure (Fig. 11). The iliopectineal fascia is first opened cephalad to the pubic ramus using a right-angle clamp. A lane retractor can then be passed around the superior edge of the ramus in an extraperiosteal fashion. The inferior edge of the ramus is slightly more difficult to define, but similar use of a Crego elevator followed by a lane can help create this channel. A Statinski vessel clamp can then be passed extraperiosteally from distal to proximal through the obturator foramen; a long Schnitt can be used to transfer a suture loop to the Statinski which can in turn be used to shuttle a Gigli saw around the pubic ramus (Fig. 12). Before the saw

Fig. 6 The position and depth of the cut can be periodically confirmed on the false profile view. The osteotome should be externally rotated such that the blade is on the "edge" on the fluoroscopic image (Reproduced with permission from the Division of Orthopaedic Surgery, Children's Hospital of Philadelphia)

Fig. 7 Retractors are needed for protection but obscure visualization. The position of the osteotome can be confirmed by periodically withdrawing the lane (Reproduced with permission from the Division of Orthopaedic Surgery, Children's Hospital of Philadelphia)

is passed, the shuttle suture is pulled taut to be sure that the adductor musculature does not contract which would indicate entrapment of the obturator nerve. While the pubis can be cut with other

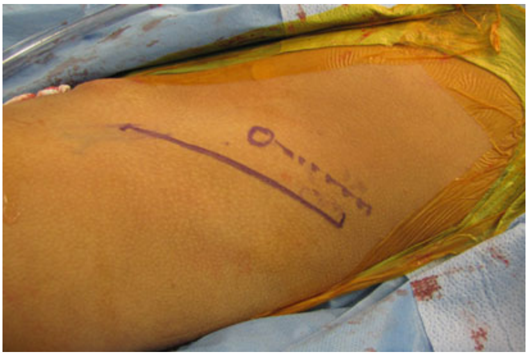


Fig. 8 Skin incision for the anterior exposure (Reproduced with permission from the Division of Orthopaedic Surgery, Children's Hospital of Philadelphia)

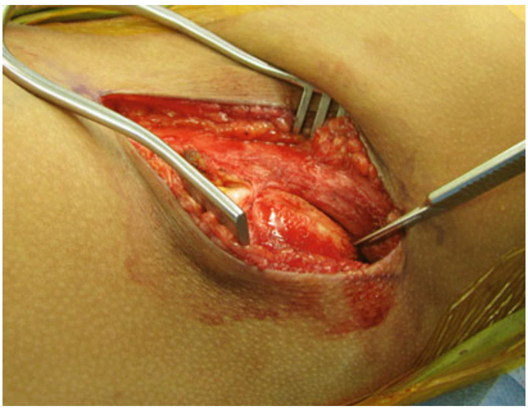


Fig. 9 Splitting of the apophysis (Reproduced with permission from the Division of Orthopaedic Surgery, Children's Hospital of Philadelphia)

techniques (e.g., osteotome), use of a Gigli saw ensures a complete cut including the overlying and often thick periosteum.

Iliac Cut

The iliac cut generally starts just distal to the ASIS and is shaped somewhat similar to Kalamchi's modification of the straight Salter osteotomy [7]. A 3.2 mm drill hole is made approximately 1 cm above the pelvic brim in line with superior aspect of the sciatic notch on the false profile view (Fig. 13). An oscillating saw is used to perform

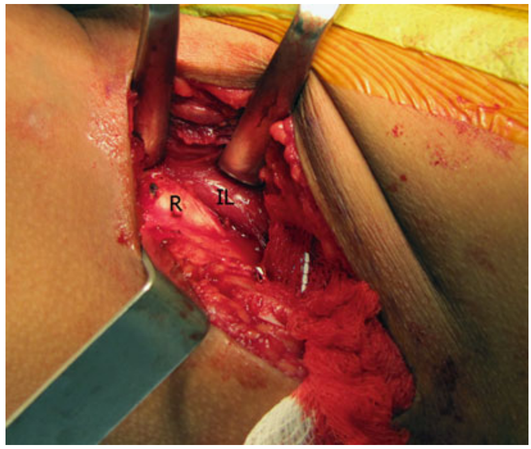


Fig. 10 The interval between the rectus tendon (*R*) and the iliacus (*IL*) is identified (Reproduced with permission from the Division of Orthopaedic Surgery, Children's Hospital of Philadelphia)

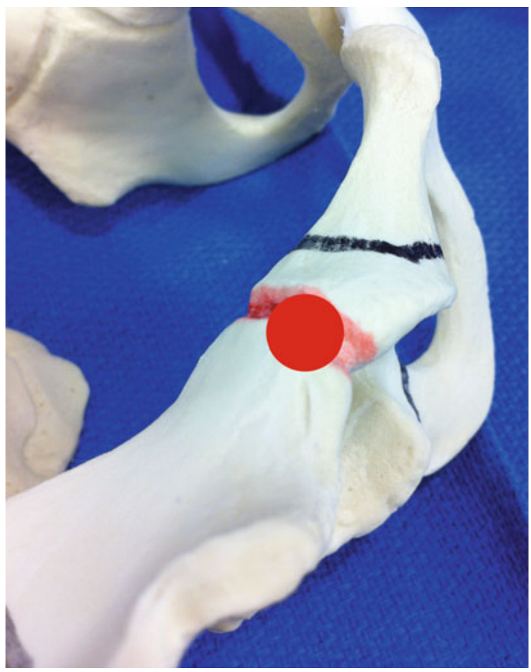


Fig. 11 The pubis cut (*black line*) must be made medial to the pubic limb of the triradiate cartilage (*red line*). The pubic eminence (*red dot*) can be palpated easily and is an important landmark for this cut (Reproduced with permission from the Division of Orthopaedic Surgery, Children's Hospital of Philadelphia)

Fig. 12 A Statinski clamp is used to shuttle a suture (and then a Gigli saw) around the pubic ramus (Reproduced with permission from the Division of Orthopaedic Surgery, Children's Hospital of Philadelphia)

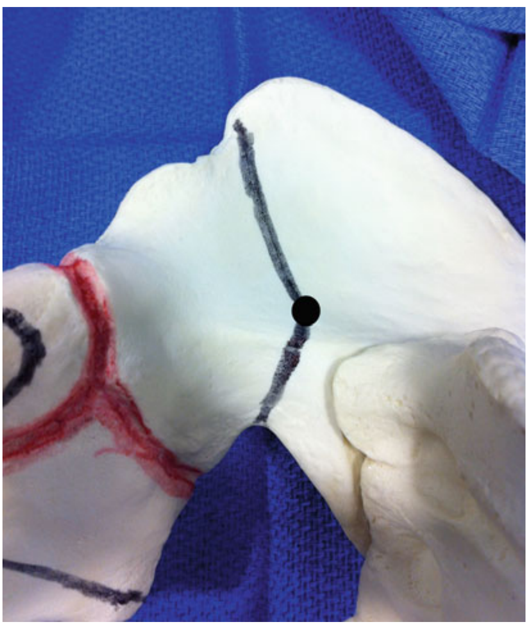


Fig. 13 Orientation of iliac osteotomy. A drill hole (*black dot*) is placed 1 cm above the pelvic brim. This allows the surgeon to control the directional change in the osteotomy (Reproduced with permission from the Division of Orthopaedic Surgery, Children's Hospital of Philadelphia)

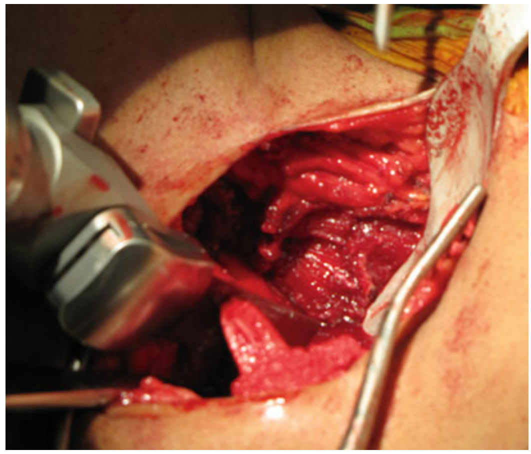


Fig. 14 The first segment of the iliac osteotomy is performed with an oscillating saw (Reproduced with permission from the Division of Orthopaedic Surgery, Children's Hospital of Philadelphia)

the first segment of the osteotomy connecting the starting point and the drill hole (Fig. 14). With a reverse Homan in place to protect the contents of the sciatic notch, the corner of a straight Ganz or flat chisel is placed in the drill hole and aimed directly into the notch (Fig. 15). Use of a laminar spreader can facilitate completion of the osteotomy. Performing this angled cut allows bony contact close to the point in the pelvis where the mechanical forces are transmitted, thereby improving stability and fixation and obviating the need for a large structural bone graft.

Mobilization of the Fragment and Fixation

Assuming all of the cuts are complete, a 4 mm Schanz screw is placed into the acetabular fragment just distal and parallel to the first segment of the iliac cut. A Weber tenaculum is then placed around the superior pubic ramus exiting proximal to the Schanz screw. Grasping the screw with a T-handle chuck in one hand and the Weber with the opposite hand allows complete control over the acetabular fragment. Similar to a periacetabular osteotomy, positioning

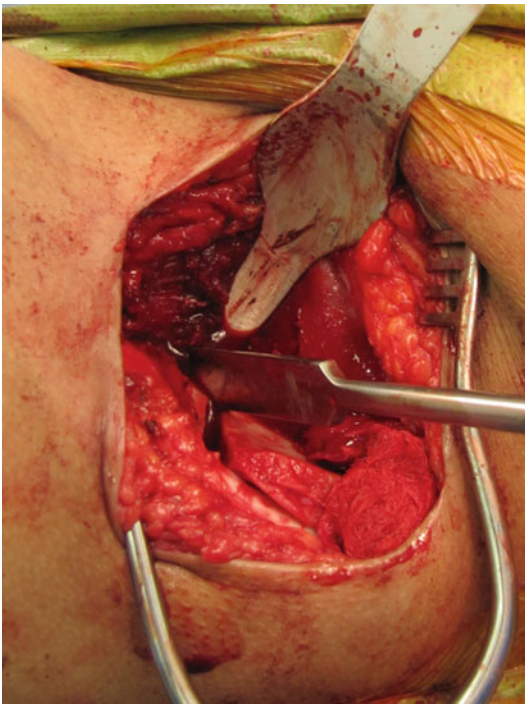


Fig. 15 With a reverse Homan in place to protect the contents of the sciatic notch, the corner of a straight Ganz or flat chisel is placed in the drill hole and aimed directly into the notch (Reproduced with permission from the Division of Orthopaedic Surgery, Children's Hospital of Philadelphia)

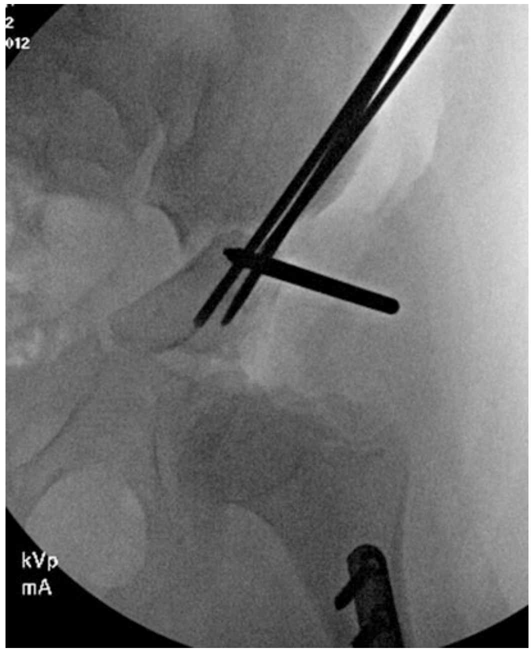


Fig. 16 The acetabular fragment is positioned with the help of a Schanz screw and held provisionally with K wires (Reproduced with permission from the Division of Orthopaedic Surgery, Children's Hospital of Philadelphia)

of the acetabular fragment is critical and should be tailored to the particular pattern of deficiency. In general, anterior coverage is provided by angling the Schanz screw distal, anteversion by angling the screw medial, and lateral coverage by rotating the fragment externally about the axis of the screw itself. Three to four 3/32 Kirschner (K) wires are inserted through the proximal ilium and into the acetabular fragment to get provisional fixation (Fig. 16). AP and false profile views of the operative hip and the entire pelvis using the image intensifier are necessary to confirm proper coverage, version, and sourcil position. In addition one should confirm that the wires are positioned away from the joint and the triradiate cartilage. The length of the K wires are then measured by placing an additional K wire next to the exiting portion and measuring the difference with a ruler. Each K wire can then be sequentially exchanged for a long 3.5 mm pelvic screw for definitive fixation (Fig. 17).

Closure

If a sharp spike is present on the acetabular fragment, this can be trimmed at the level of the AIIS and used to bone graft the iliac osteotomy. A drain is placed along the inner table and brought out through the Smith-Peterson interval. The apophysis of the iliac crest is repaired with a deep-deep, superficial-superficial double throw using absorbable 0 or 1-0 suture which improves apposition of the cartilage surfaces (Fig. 18). The external oblique is repaired with a running 2-0 suture followed by additional running layers in the dermis and the subcutaneous layer.

Postoperative Management

Depending on the patient's ability to comply with postoperative weight-bearing restrictions, a single-leg spica cast can be used for postoperative immobilization. Otherwise, the patient may be

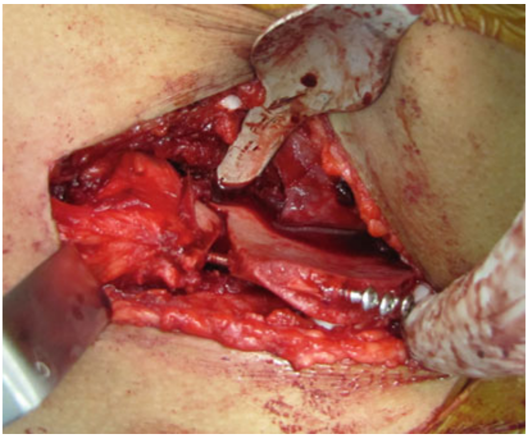


Fig. 17 Definitive fixation of the osteotomy with 3.5 mm pelvic screws (Reproduced with permission from the Division of Orthopaedic Surgery, Children's Hospital of Philadelphia)

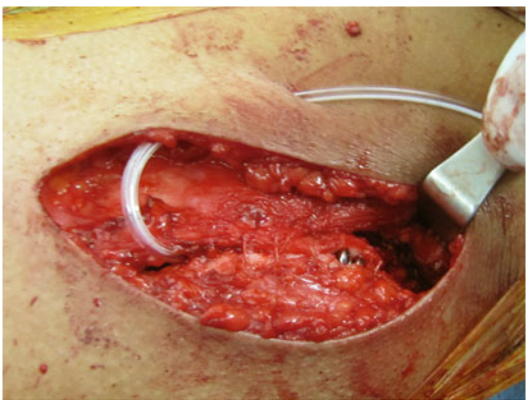


Fig. 18 Closure and drain placement (Reproduced with permission from the Division of Orthopaedic Surgery, Children's Hospital of Philadelphia)

kept touchdown weight bearing. At 4–6 weeks depending on the degree of bony healing, weight bearing is generally progressed and physical therapy started for gait training and abductor strengthening.

Pearls and Pitfalls

- Angling the ischial cut above the ischial spine removes the tethering effects of the sacrospinous ligament and aids in the mobilization of the acetabular fragment.

Fig. 19 Preoperative anteroposterior (*AP*) radiograph of the pelvis (Reproduced with permission from the Division of Orthopaedic Surgery, Children's Hospital of Philadelphia)

- Using a Gigli saw passed extraperiosteally around the pubic ramus ensures that the pubis (which can be thin and flexible in this age group) is completely cut and freed from its thick periosteal connections.
- Performing the Kalamchi modification of the Salter iliac cut allows bony contact close to the point in the pelvis where the mechanical forces are transmitted, thereby improving stability and fixation and obviating the need for a large structural bone graft.
- Unlike the PAO, intraoperative imaging to assess acetabular position after correction can be quite difficult in the triple osteotomy because the bony acetabular rim is not well defined in these younger patients. As a result, one must be quite careful to avoid overcoverage. It is sometimes helpful to place a clamp at the edge of the palpated acetabular rim and use the image intensifier to help estimate coverage.

Case Example

An 11-year-old female presented with a pain and a limp. An AP pelvic radiograph demonstrates subluxation and acetabular dysplasia of the left hip (Fig. 19). She underwent a modified Bernese triple osteotomy. AP pelvic radiograph taken

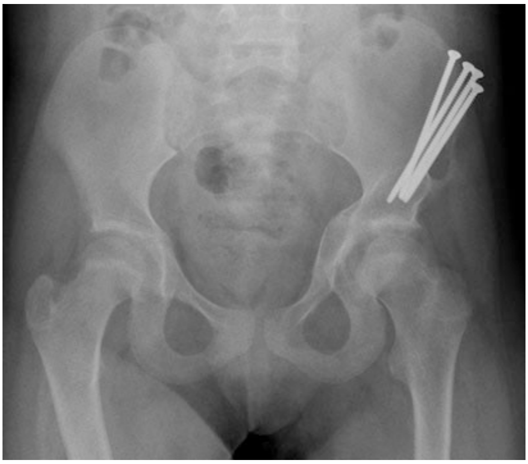


Fig. 20 AP pelvic radiograph 2 years following surgery (Reproduced with permission from the Division of Orthopaedic Surgery, Children's Hospital of Philadelphia)

two years after surgery demonstrates a healed osteotomy. The left hip is now reduced with improved femoral head coverage and a horizontal sourcil (Fig. 20).

Summary

The triple innominate osteotomy is a complete redirectional acetabular osteotomy, consisting of cuts through the ilium, ischium, and pubis. Unlike a periacetabular osteotomy (Ganz), the triple does not violate the triradiate cartilage and can therefore be performed in the skeletally immature. In contrast to most other pelvic osteotomies, this procedure completely frees the acetabulum from the rest of the pelvis which in turn allows the surgeon to obtain large corrections and control the final position of the acetabulum in multiple planes. The modified Bernese triple osteotomy is performed through two incisions. The medial approach allows access for the ischial osteotomy, while the anterior exposure is used for the osteotomies of the pubis and ilium. Definitive fixation is obtained with long pelvic screws.

References

1. Le Coeur P. Correction of the abnormal acetabular orientation with isthmic osteotomy of the ilium [in French]. Rev Chir Orthop. 1965;51:211–2.
2. Steel HH. Triple osteotomy of the innominate bone. J Bone Joint Surg. 1973;55:343–50.
3. Tonnis D, Behrens K, Tscharani F. A modified technique of triple pelvic osteotomy: early results. J Pediatr Orthop. 1981;1:241–9.
4. Gillingham BL, Sanchez AA, Wenger DR. Pelvic osteotomies for the treatment of hip dysplasia in children and young adults. J Am Acad Orthop Surg. 1999;7(5):325–37.
5. Sankar WN. Complete redirectional acetabular osteotomies for neurogenic and syndromic hip dysplasia. J Pediatr Orthop. 2013;33(Suppl1):S39–44.
6. Rebello G, Zilkens C, Dudda M, Matheney T, Kim YJ. Triple pelvic osteotomy in complex hip dysplasia seen in neuromuscular and teratologic conditions. J Pediatr Orthop. 2009;29:527–34.
7. Kalamchi A. Modified Salter osteotomy. J Bone Joint Surg Am. 1982;64(2):183–7.

Surgical Technique: Arthroscopic Treatment of Perthes Disease

32

Patrick Riley Jr., Mininder S. Kocher, and Yi-Meng Yen

Contents

Abstract

Legg-Calve-Perthes disease (LCPD) is an idiopathic hip disorder that causes ischemic necrosis of the growing femoral head. It is a known cause of femoroacetabular impingement (FAI) in older children and adolescents because of the development of a misshapen head and corresponding acetabular deformity. Moreover, hip instability, extra-articular impingement, hinge abduction, and versional deformities are found in Perthes hips. These all have the potential to lead to debilitating arthritis later in life. Although hip arthroscopy has proven to be successful in the treatment of FAI, its role and indications in LCPD-associated FAI remain unclear. Historically, the most common indication for hip arthroscopy in the pediatric and adolescent population has been in LCPD, for both the diagnosis of the severity of the disease and the removal of loose bodies. With improved techniques and instrumentation, however, the indications have expanded to include femoral head/neck reshaping, labral repair, and acetabular trimming. Short-term outcomes studies have been promising; however, longer-term studies are needed to see if the natural long-term history of LCPD can be affected.

P. Riley Jr. (✉)
Akron Children's Hospital, Akron, OH, USA
e-mail: patrick.riley@childrens.harvard.edu

M.S. Kocher • Y.-M. Yen
Harvard Medical School Children's Hospital Boston, Boston, MA, USA

Division of Sports Medicine, Orthopedic Surgeon, Boston Children's Hospital, Boston, MA, USA
e-mail: mininder.kocher@childrens.harvard.edu; yi-meng.yen@childrens.harvard.edu

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_35

Introduction

Legg-Calve-Perthes disease (LCPD) is an idiopathic self-limiting condition involving avascular necrosis of the femoral epiphysis. The femoral head eventually heals but may result in complex deformities resulting in FAI, instability, or both. These deformities have been attributed to early development of hip osteoarthritis [1, 2]. Since its first description almost 100 years ago, there has been little insight into the etiology and pathophysiology of this condition [3]. There is some evidence that a mutation in type II collagen may play a role [4] and suggestions of thrombophilias causing LCPD [5, 6].

Epidemiology

LCPD is the most common form of pediatric hip disorders ranging from 2 to 294 per 1,000,000 children. Typically, LCPD presents within the first decade of life occurring predominantly in males aged 4–8 but can affect a much wider age range. The male to female ratio is around 5:1 and affects bilateral hips in 10–15 % of patients [7]. LCPD seems to have a predilection for children who are hyperactive and have delayed skeletal bone ages [8]. It is a diagnosis of exclusion as other etiologies of femoral head osteonecrosis such as steroid use, sickle cell disease, or skeletal dysplasias must be ruled out.

Presentation

The pathogenesis of the disease passes through multiple stages: the initial avascular necrosis, fragmentation of the epiphysis, resorption, and collapse, followed by re-ossification [9].

LCPD is specific to the hip joint and typically presents as an insidious, unilateral relatively painless limp with the earliest complaint commonly being "stiffness." If groin pain is present, it usually is mild and is exacerbated with increasing physical activity and sitting for prolonged periods. The earliest physical examination findings include decreased internal rotation and loss of abduction of the hip. A positive Trendelenburg test is frequently seen as well. Initial irritability of the hip is likely from synovitis, but as the head heals with resultant deformity, pain may also be elicited in positions of impingement such as hip flexion with internal rotation [10]. Patients may complain of catching or locking of the hip during prolonged sitting or standing, squatting, ascending stairs, and bending down to put on shoes [11].

A diagnosis of LCPD is made with an anteroposterior and lateral X-ray view of the hip and femoral head. Initial severity of the disease is graded by the Catterall or Herring classification systems, which give a prognosis based upon the location of the avascular necrosis and the percentage of head involvement [12, 13]. After the femoral head re-ossifies and matures, a final assessment is graded by the Mose or Stulberg classification system [1, 14]. The Stulberg classification is useful because it serves as a predictor for symptoms and function with time. The pathology of the hip varies from a Stulberg I hip (nearly normal) to a Stulberg V (severe femoral head deformity associated with significantly altered acetabular morphology and an incongruous joint).

Pathoanatomy

One of the difficulties in the decision making for treatment options in LCPD is searching for the source of pain. Catterall postulated that hinge abduction and limb length discrepancy were the main causes of pain [12]. Grossbard described osteochondritis dissecans, hinging hip, torn acetabular labrum, and meralgia paresthetica as causes of pain [15]. Finally, abductor insufficiency and fatigue have also been recognized as a cause of pain associated with overgrowth of the greater trochanter and coxa brevis [16].

LCPD nearly always results in a deformity of the femoral head and neck and corresponding acetabulum. Snow et al. were the first to publish on impingement as a cause of groin pain in the later stages of LCPD [17]. They described impingement of the anterior femoral head against the anterior acetabulum on internal rotation of the leg as cause of groin pain after re-ossification of the femoral head. The combination of a misshapen femoral head and acetabular remodeling can lead to symptoms due to femoroacetabular impingement (FAI). In general, in LCPD, there is nearly always a component of FAI, which can be cam or pincer, or a combination of both [17, 18]. This abnormal contact between the anterior femoral head-neck junction and the anterior aspect of the acetabular rim has been linked to the onset of early OA of the hip [19].

Indications and Decision Making

The ultimate goal, regardless of treatment method, in patients with FAI secondary to LCPD is to improve impingement free range of motion and restore joint congruency and stability [20, 21]. Therefore, arthroscopy may benefit these patients by reduction of the femoral head deformity and its resultant acetabular incongruity. However, as in all surgery, careful patient selection is critical. A detailed history and physical examination is, therefore, necessary to identify the patients who will most benefit from arthroscopic surgery. However, whether arthroscopic treatment of this disease affects long-term outcomes and the natural history of this disease remains to be seen.

An approach to deciding which LCPD patient will benefit from arthroscopic surgery first depends on the patients' complaints. Patients with mechanical symptoms such as clicking, catching, or locking and physical exam findings of FAI can benefit from hip arthroscopy. Oftentimes, these symptoms are caused by a torn labrum, chondral flaps, chondral loose bodies, or a combination of these. Treatment of these conditions is very suitable for arthroscopic intervention.

If the patient has signs of FAI but not significant mechanical symptoms, then imaging and classifying the hip based on the Stulberg classification is performed. A Stulberg I or II with FAI findings is a good arthroscopic candidate because treatment with femoral head-neck osteoplasty may be no more difficult than those patients with "primary" FAI. Stulberg IV or V patients, in general, are not as amenable to arthroscopic treatment because of the severity of the deformity and the presence of coexisting acetabular retroversion or dysplasia with resultant instability (Fig. 1). Once a patient has developed secondary instability, or a complex mismatch between the enlarged femoral head and misshapen acetabulum, arthroscopic treatment is unlikely to be of benefit [20, 21]. These patients are more likely to benefit from open procedures. Stulberg III patients are a gray area but have the potential to do very well with arthroscopic hip surgery as demonstrated by Roy et al. [22].

Other sources of pain and dysfunction in patients with Perthes disease include extra-articular impingement and version problems in both the femur and acetabulum. Perthes patients may develop a relatively high-riding greater trochanter as a consequence of the proximal femoral physis arrest and collapse. This extra-articular impingement can be a cause of limited range of motion and dysfunction. Arthroscopy has little role in this instance and would better be treated

Fig. 1 Stulberg IV hip that is not amenable to arthroscopic intervention. Clinical symptoms of greater trochanteric impingement. Note the amount of bone that would need to be removed to make the femoral head truly spherical (*dotted circle*). The patient was scheduled for an open surgical dislocation with osteochondroplasty and trochanteric transfer for a relative head-neck lengthening

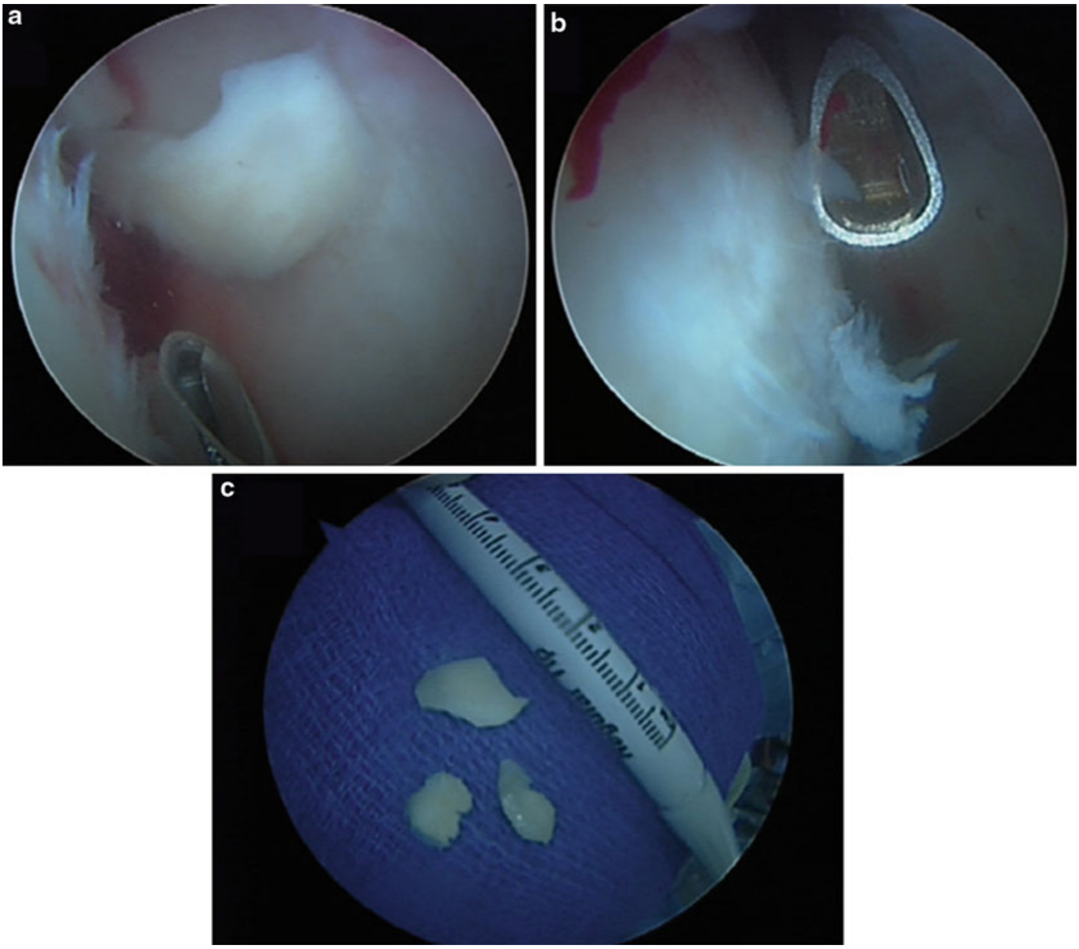


Fig. 2 Central compartment access of a Perthes hip. (**a**) Chondral flap of the acetabulum. (**b**) Chondral fraying of the femoral head. (**c**) Removal of loose bodies from the central compartment

by an open procedure, such as a trochanteric advancement with relative neck lengthening.

Perthes patients, historically, have had higher than normal femoral anteversion when compared with normal controls [23]. It has been suggested that this is a secondary deformation which may predispose the hip to further instability and pain. Additionally, there have been studies that have shown an increase in acetabular retroversion, while other studies have refuted this [24–26]. "Functional retroversion" of the femoral head has also been described when the posteromedial portion of the femoral head has remodeled in a retroverted position in relation to the anterolateral portion. This may help explain the paradox of why a Perthes patient walks with an externally rotated gait [27]. Arthroscopy would not have a role in these cases and open osteotomies would be recommended to correct significant version deformities.

Surgical Technique

Standard portals may need to be modified in LCPD patients secondary to trochanteric overgrowth, coxa magna, and surgical reconstruction of the acetabulum [22]. The anterolateral peritrochanteric portal may be more proximal than normal, and the use of accessory portals may be more common. The use of fluoroscopy is recommended to avoid iatrogenic damage to the hip joint. Occasionally, an outside-in technique may need to be utilized to gain access to the peripheral compartment first before entry into the central compartment particularly in cases in which acetabular reorientation has already been performed [28].

After access to the central compartment, inspection of the joint can be conducted. Commonly, loose bodies may be encountered, most times originating from the femoral head. They should be removed with arthroscopic graspers, shavers, or suction (Fig. 2). The chondral surfaces can be inspected and chondroplasty or microfracture can be performed on the acetabulum, femoral head, or both. Labral pathology is also commonly found and may be addressed by either labral debridement in the cases of a hypertrophied labrum, an infolded labrum, or labral fraying or, more commonly, repair of the labrum with suture anchors (Fig. 3).

Once work is completed in the central compartment, the peripheral compartment work can be addressed. A generous capsulotomy is helpful with the increased in the femoral head-neck size. Any loose bodies in the peripheral compartment should be removed at this time (Fig. 4). Recontouring of the femoral head-neck junction will depend on the Stulberg classification, with Stulberg I/II hips more amenable to a suitable osteochondroplasty (Fig. 5). With the Perthes hip, the amount of recontouring that is able to be conducted will depend upon preoperative templating and how ovoid the femoral head-neck junction is. The use of liberal fluoroscopy and dynamic examination will guide the osteochondroplasty. It is very rare that a Perthes head can be actually made truly spherical, particularly a Stulberg III–V femoral head (Fig. 6). A dynamic and fluoroscopic examination should be conducted to ensure adequate bony resection. A capsule closure can be performed, particularly in patients who are ligamentously lax or have complaints of instability.

Postoperative management is similar to that of patients treated with idiopathic FAI, with foot-flat weight bearing for 2–4 weeks followed by a rigorous physical therapy regimen [29]. A continuous passive motion machine is used for 2–3 weeks unless a microfracture was done in which case the CPM is used for 6 weeks. Generally, return to activity occurs at 4–6 months.

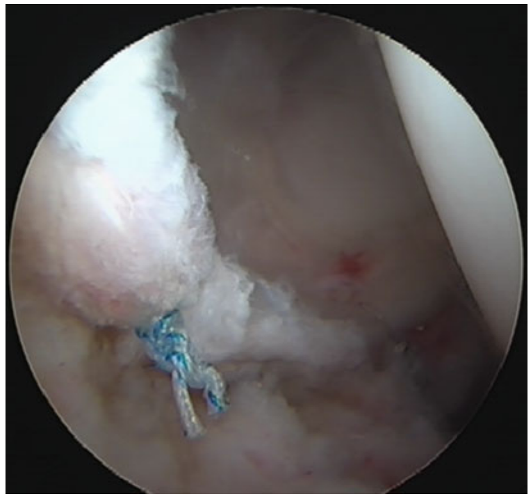


Fig. 3 Labral repair in Perthes hip utilizing suture anchors into the acetabulum

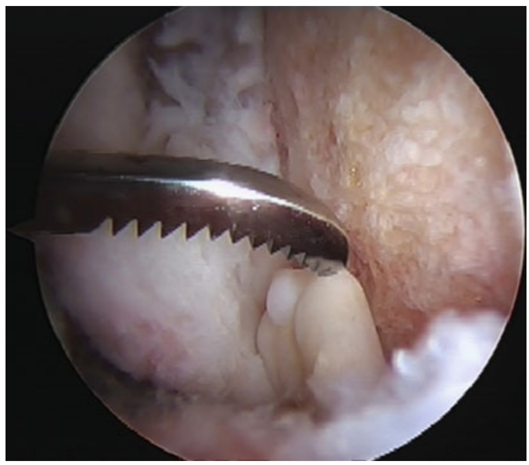


Fig. 4 Removal of loose bodies in the peripheral compartment. Osteochondroplasty has already been performed of the femoral head-neck junction

Pearls and Pitfalls

- Proper preoperative planning is paramount. Identification of the Stulberg classification of the LCPD is used to decide upon open vs. arthroscopic procedures.
- Removal of all loose bodies is critical and thorough examination of the central and peripheral compartment is necessary.
- A generous capsulotomy or “T-shaped” capsulotomy can be used because of the

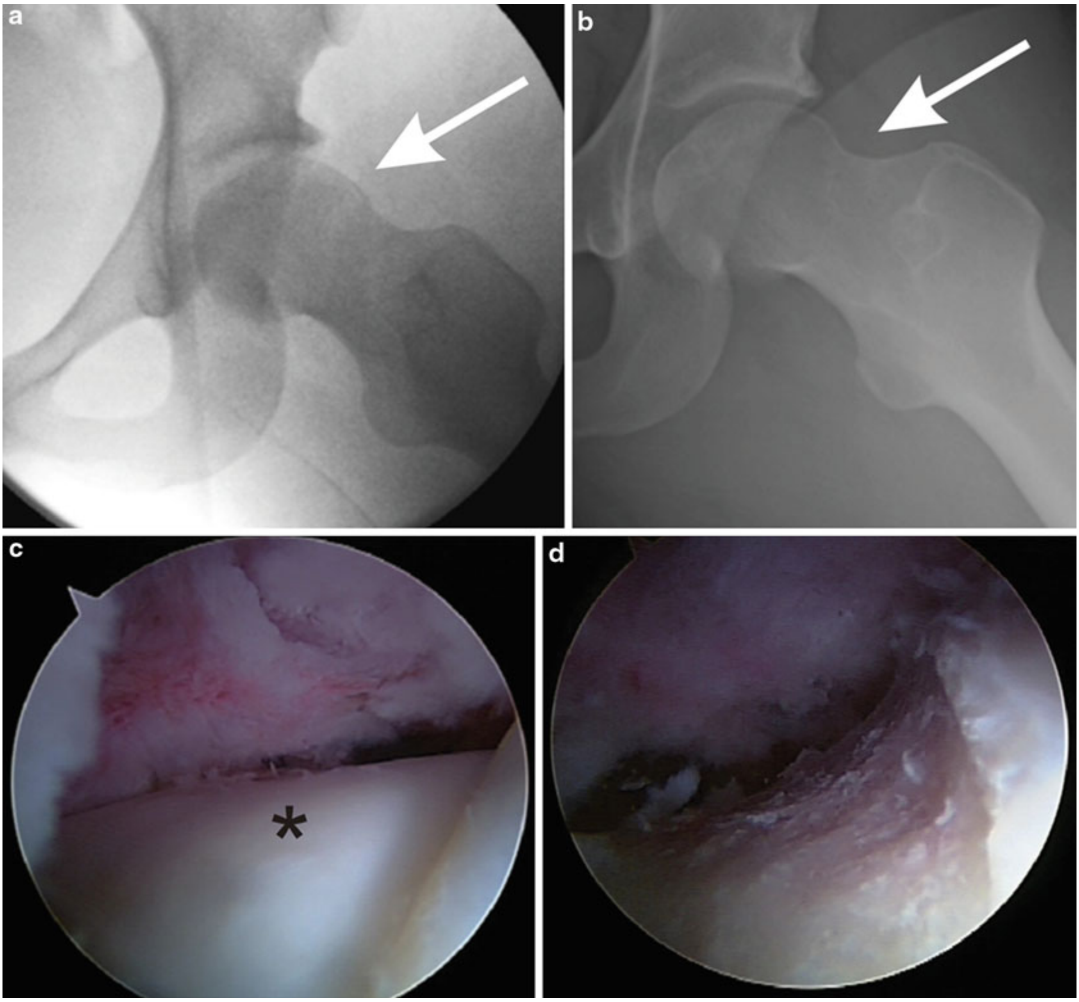


Fig. 5 Osteochondroplasty of a Stulberg I hip with only minimal femoral head-neck deformity. (**a**) Preoperative head-neck junction showing decreased head-neck offset with no acetabular deformity. (**b**) Postoperative head-neck junction after osteochondroplasty. (**c**) Intraoperative photo of preoperative head-neck junction prior to osteochondroplasty (*asterisk*). (**d**) Intraoperative photo following osteochondroplasty

enlarged femoral head-neck junction. Consideration can be made to close the capsulotomy.

- Liberal use of fluoroscopy to identify the exact areas of resection and prevent under- or over-resection.

Outcomes

Despite multiple studies, an optimal treatment for LCPD has yet to be found. Historically, outcomes from LCPD demonstrated overall good results especially for Stulberg I and II hips with variable results for Stulberg III and poor results for IV and V [1, 12, 30]. In a recent multicenter prospective study, Larson et al. reported on 20 years of follow-up for 56 LCPD patients in their third decade of life that were treated nonoperatively [31]. They showed that at least half of these young patients had poor or fair outcomes and that hip pain was associated with positive impingement signs on physical exam. The study also showed that 60 % of Stulberg III, IV, and V hips had evidence of osteoarthritis, similar to Stulberg's initial study, but at a much younger age.

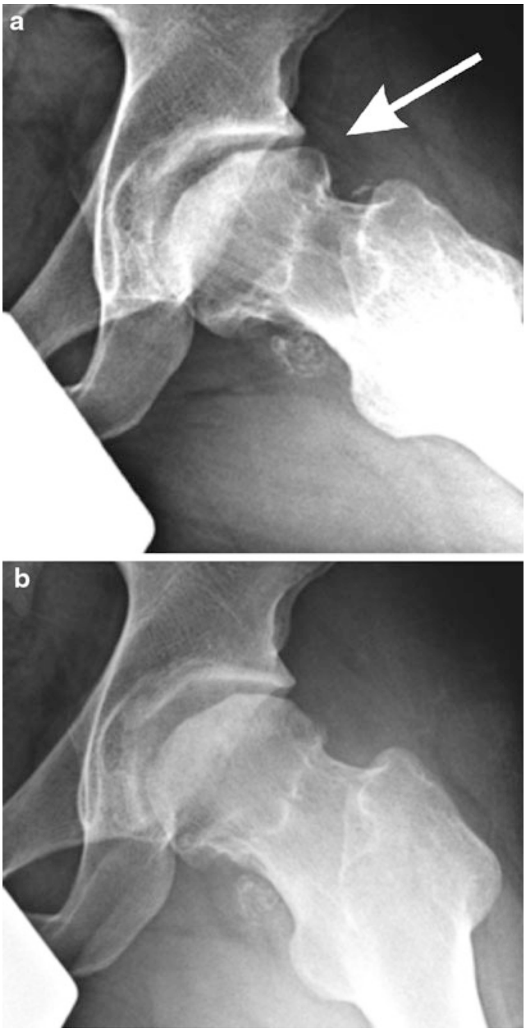


Fig. 6 Osteochondroplasty of a Stulberg IV Hip. (**a**) Preoperative Dunn-lateral radiograph (*arrow* depicts the osseous bump at the femoral head-neck junction). (**b**) Postoperative Dunn-lateral radiograph after osteochondroplasty of the femoral head-neck junction

With the femoral head-neck “bump” being recognized as a source of impingement and resultant dysfunction in late LCPD, initial studies reported on performing “cheilectomies” with highly satisfactory results 2–3 years postoperatively [32]. However, in a 25-year follow-up of five LCPD patients who underwent cheilectomy, all patients had developed osteoarthritis and three of the five had poor results, while the other two had fair or good results [33].

Several studies have looked at results of arthroscopic management of patients with LCPD. Roy et al. performed nine hip arthroscopies on LCPD patients with one being a Stulberg II, one a Stulberg IV, and the rest Stulberg III [22]. The surgery was conducted during adolescence following a pain-free interval. Eight of the nine patients had abnormalities noted on arthroscopy and seven of these eight improved their symptoms enough to play sports. The one patient that experienced no improvement had osteoarthritis, had significant femoral head deformity, and was classified as a Stulberg IV. Within 2 years, two of the seven underwent repeat arthroscopy due to recurrence of symptoms (one at 18 months, one at 2 years). A third patient underwent total hip arthroplasty after 2 years of no symptoms. Kocher et al. reported on 54 hip arthroscopies in children and adolescents, including eight LCPD patients [34]. Their mean mHHS improved by a mean of over 30 points, which was statistically significant. Bowen et al. reported on their experience of hip arthroscopy for OCD after LCPD [35]. The three patients who had the OCD fragment removed were asymptomatic at 1 year, while the two without removal had severe degeneration of the femoral head. O’Leary et al.’s series of 84 patients included 9 who had LCPD. Eighty-eight percent of the LCPD had improvement postoperatively [36]. More recently, Freeman et al. reported on 22 patients ranging from Stulberg I-IV with a median age of 27. Eighteen labral tears were identified, along with 17 torn ligamentum teres, 17 chondral lesions, 5 loose bodies, 3 osteochondral defects, and 2 cam lesions. All patients improved with a modified Harris hip score of 82 postoperatively compared to 56.7 preoperatively which did not correlate with the Stulberg classification. Two patients underwent repeat arthroscopy for failure of pain relief [37].

Summary

LCPD is an idiopathic disease of necrosis of the femoral head. The resultant proximal femur deformity and corresponding acetabular deformity are best classified by the Stulberg system. Stulberg I and II hips are very amenable to arthroscopic intervention, while Stulberg IV and V hips often require open surgical procedures to adequately address the bony deformities. Good short-term outcomes have been obtained from arthroscopic approaches.

References

1. Stulberg SD, Cooperman DR, Wallensten R. The natural history of Legg-Calve-Perthes disease. J Bone Joint Surg Am. 1981;63(7):1095–108.
2. Weinstein SL. Legg-Calvé-Perthes disease: results of long-term follow-up. Hip. 1985:28–37.
3. Legg A. An obscure affection of the hip-joint. Boston Med Surg J. 1910;162:202.
4. Miyamoto Y, Matsuda T, Kitoh H, Haga N, Ohashi H, Nishimura G, Ikegawa S. A recurrent mutation in type II collagen gene causes Legg-Calve-Perthes disease in a Japanese family. Hum Genet. 2007;121(5):625–9.
5. Glueck CJ, Glueck HI, Greenfield D, Freiberg R, Kahn A, Hamer T, Stroop D, Tracy T. Protein C and S deficiency, thrombophilia, and hypofibrinolysis: pathophysiologic causes of Legg-Perthes disease. Pediatr Res. 1994;35(4 Pt 1):383–8.
6. Hresko MT, McDougall PA, Gorlin JB, Vamvakas EC, Kasser JR, Neufeld EJ. Prospective reevaluation of the association between thrombotic diathesis and legg-perthes disease. J Bone Joint Surg Am. 2002;84-A (9):1613–18.
7. Molloy MK, MacMahon B. Incidence of Legg-Perthes disease (osteochondritis deformans). N Engl J Med. 1966;275(18):988–90.
8. Kim HK. Legg-Calve-Perthes disease. J Am Acad Orthop Surg. 2010;18(11):676–86.
9. Waldenström H. The definitive forms of coxa plana. Acta Radiol. 1992;1:384.
10. Eijer H, Berg RP, Haverkamp D, Pecasse GA. Hip deformity in symptomatic adult Perthes' disease. Acta Orthop Belg. 2006;72(6):683–92.
11. Eijer H, Podeszwa DA, Ganz R, Leunig M. Evaluation and treatment of young adults with femoro-acetabular impingement secondary to Perthes' disease. Hip Int. 2006;16(4):273–80.
12. Catterall A. The natural history of Perthes' disease. J Bone Joint Surg Br. 1971;53(1):37–53.
13. Herring JA, Kim HT, Browne R. Legg-Calve-Perthes disease. Part I: classification of radiographs with use of the modified lateral pillar and Stulberg classifications. J Bone Joint Surg Am. 2004;86-A(10):2103–20.
14. Mose K, Hjorth L, Ulfeldt M, Christensen ER, Jensen A. Legg Calve Perthes disease. The late occurrence of coxarthrosis. Acta Orthop Scand Suppl. 1977;169:1–39.
15. Grossbard GD. Hip pain during adolescence after Perthes' disease. J Bone Joint Surg Br. 1981;63B (4):572–4.
16. Catterall A. Adolescent hip pain after Perthes' disease. Clin Orthop Relat Res. 1986;209:65–9.
17. Snow SW, Keret D, Scarangella S, Bowen JR. Anterior impingement of the femoral head: a late phenomenon of Legg-Calve-Perthes' disease. J Pediatr Orthop. 1993;13(3):286–9.
18. Sucato DJ. Role of femoral head surgery in skeletally mature Perthes disease. J Pediatr Orthop. 2013;33 Suppl 1:S70–5.
19. Ganz R, Parvizi J, Beck M, Leunig M, Notzli H, Siebenrock KA. Femoroacetabular impingement: a cause for osteoarthritis of the hip. Clin Orthop Relat Res. 2003;417:112–20.
20. Kim YJ, Novais EN. Diagnosis and treatment of femoroacetabular impingement in Legg-Calve-Perthes disease. J Pediatr Orthop. 2011;31(2 Suppl):S235–40.
21. Novais EN, Clohisy J, Siebenrock K, Podeszwa D, Sucato D, Kim YJ. Treatment of the symptomatic healed Perthes hip. Orthop Clin North Am. 2011;42 (3):401–17, viii.
22. Roy DR. Arthroscopic findings of the hip in new onset hip pain in adolescents with previous Legg-Calve-Perthes disease. J Pediatr Orthop B. 2005;14 (3):151–5.
23. Upadhyay SS, Burwell RG, Moulton A. Femoral anteversion in Perthes' disease with observations on irritable hips. Application of a new method using ultrasound. Clin Orthop Relat Res. 1986;209:70–6.
24. Sankar WN, Flynn JM. The development of acetabular retroversion in children with Legg-Calvé-Perthes disease. J Pediatr Orthop. 2008;28(4):440–3. doi:10.1097/BPO.0b013e318168d97e.
25. Ezoe M, Naito M, Inoue T. The prevalence of acetabular retroversion among various disorders of the hip. J Bone Joint Surg Am. 2006;88(2):372–9.
26. Kawahara S, Nakashima Y, Oketani H, Wada A, Fujii M, Yamamoto T, Mawatari T, Motomura G, Sato T, Akiyama M, Fujii T, Takamura K, Iwamoto Y. High prevalence of acetabular retroversion in both affected and unaffected hips after Legg-Calvé-Perthes disease. J Orthop Sci. 2012;17(3):226–32. doi:10.1007/s00776-012-0213-0. Epub 2012 Mar 20.
27. Kim HT, Wenger DR. "Functional retroversion" of the femoral head in Legg- Calvé-Perthes disease and epiphyseal dysplasia: analysis of head-neck deformity and its effect on limb position using three-dimensional computed tomography. J Pediatr Orthop. 1997;17 (2):240–6.
28. Gutierrez RC, Iban MA, Ayestaran AC, Sobrino AS, Oteo-Alvaro A. Peripheral compartment as the initial access for hip arthroscopy in complex cases: technical note. Orthopedics. 2013;36(6):456–62.
29. Stalzer S, Wahoff M, Scanlan M. Rehabilitation following hip arthroscopy. Clin Sports Med. 2006;25 (2):337–57, x.

30. McAndrew MP, Weinstein SL. A long-term follow-up of Legg-Calve-Perthes disease. J Bone Joint Surg Am. 1984;66(6):860–9.
31. Larson AN, Sucato DJ, Herring JA, Adolfsen SE, Kelly DM, Martus JE, Lovejoy JF, Browne R, Delarocha A. A prospective multicenter study of Legg-Calve-Perthes disease: functional and radiographic outcomes of nonoperative treatment at a mean follow-up of twenty years. J Bone Joint Surg Am. 2012;94(7):584–92.
32. Garceau G. Surgical treatment of coxa plana. J Bone Joint Surg Br. 1964;46B:779.
33. Rowe SM, Jung ST, Cheon SY, Choi J, Kang KD, Kim KH. Outcome of cheilectomy in Legg-Calve-Perthes disease: minimum 25-year follow-up of five patients. J Pediatr Orthop. 2006;26(2):204–10.
34. Kocher MS, Kim YJ, Millis MB, Mandiga R, Siparsky P, Micheli LJ, Kasser JR. Hip arthroscopy in children and adolescents. J Pediatr Orthop. 2005;25 (5):680–6.
35. Bowen JR, Kumar VP, Joyce III JJ, Bowen JC. Osteochondritis dissecans following Perthes' disease. Arthroscopic-operative treatment. Clin Orthop Relat Res. 1986;209:49–56.
36. O'Leary JA, Berend K, Vail TP. The relationship between diagnosis and outcome in arthroscopy of the hip. Arthroscopy. 2001;17(2):181–8.
37. Freeman CR, Jones K, Byrd JW. Hip arthroscopy for Legg-Calve-Perthes disease: minimum 2-year follow-up. Arthroscopy. 2013;29(4):666–74.

Slipped Capital Femoral Epiphysis: Acute and Chronic

33

Andrew G. Georgiadis and Ira Zaltz

Contents

Abstract

Slipped capital femoral epiphysis (SCFE) involves displacement of the proximal femoral metaphysis relative to a fixed epiphysis, generally during a period of rapid growth and unique physeal susceptibility. Affected patients have characteristic clinical, histological, and radiologic features that contribute to the displacement. A number of concomitant clinical features and medical diagnoses should heighten a physician's suspicion of SCFE, prompting appropriate radiologic and laboratory workup. Limp and hip or knee pain in a patient between the ages of 10 and 16 should always include SCFE in the differential until proven otherwise. Once the diagnosis is made, appropriate treatment involves proximal femoral physeal stabilization by a number of surgical methods. The optimal surgical treatment of severe SCFE and its late sequelae remain an evolving and controversial subject.

Introduction

The earliest report of slipped capital femoral epiphysis (SCFE) is widely attributed to a 1572 French text, *Cinq Livres de Chirurgie*, in which Ambroise Paré, a barber surgeon for the King of France, reviewed his first-person experience with hip fractures and dislocations [1]. He warned, "the epiphysis of the head of the femur sometimes separates in such a way that the surgeon is misled,

A.G. Georgiadis
Department of Orthopaedic Surgery, Henry Ford Hospital, Detroit, MI, USA

I. Zaltz (✉)
Department of Orthopaedic Surgery, William Beaumont Hospital, Royal Oak, MI, USA
e-mail: zaltzira@gmail.com

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_36

thinking there is a dislocation instead of a separation of the epiphysis." Müller first described a deformity he termed "bending of the neck of the femur in adolescence," which included cadaveric specimens and a histological description of trabecular remodeling in four patients with clinically adducted hips [2]. Before and shortly after the invention of roentgen pictures in 1895, many diagnoses were characterized broadly as simple coxa vara, which described patients with an adducted, externally rotated, extended femur. In 1898, there were 22 publications on coxa vara in the German literature alone, many of which are now recognized to represent slipped proximal femoral epiphyses. The suspected source of deformity in these early studies included fracture [3], rickettsial disorders [2], infection [4], endocrine disturbances, and "periosteal atrophy ··· tending to produce a point of weakness at the epiphyseal line" [5].

Sprengel was the first to cadaverically prove epiphyseal separation and proposed that such cases stemmed from fracture [6]. The first thorough etiologic categorization was performed by Key, in which he comprehensively classified proximal femoral varus due to Perthes, infection, Charcot arthropathy, rickets, congenital deformities, and arthritic causes, with an emphasis on slipped epiphyses [5]. Despite incomplete understanding of its true pathology, late nineteenth- and early twentieth-century authors did differentiate between traumatic and insidious proximal femoral epiphyseal separation, with early treatment often including three or more months of hip spica casting in a position of abduction and external rotation [3, 7]. Subtrochanteric cuneiform osteotomies were being performed as early as 1900 for a variety of fixed proximal femoral deformities in adolescents. Many such cases likely represented healed or remodeled SCFE.

Pathophysiology

The underlying pathology in SCFE involves a mechanical overload to the proximal femoral physis causing anterior translation and external rotation of the metaphysis with respect to the upper femoral epiphysis that remains located in the acetabulum. At-risk patients are of a characteristic age and physeal susceptibility, with a number of attendant epidemiologic, anatomical, histological, and endocrinologic factors.

Vascular Anatomy

The vascular supply to the proximal femoral epiphysis undergoes a characteristic series of developmental stages. Trueta elegantly investigated the vascular investment of the developing femoral head in 46 proximal femoral specimens from birth until adulthood [8]. Before 3 months of age, the developing hip has a significant contribution from the artery of the ligamentum teres, a robust and nearly vertical ascending metaphyseal circulation, and a horizontal precursor to the lateral epiphyseal arteries emanating from the greater trochanter. By 18 months of age, the lateral epiphyseal arteries have become the dominant contributors to the femoral head, and the ascending metaphyseal arteries no longer cross the physeal plate (Fig. 1). The artery of the ligamentum teres largely disappears between 6 months and 3 years of age, reappearing by ages 7–10 with an anastomosis with the lateral epiphyseal vessels. Complete independence of the metaphyseal and epiphyseal circulations persists. During adolescence and immediately preceding physeal closure, an increasingly rich metaphyseal circulation begins to invest the subphyseal region, ascending to terminate in the hypertrophic zone of the physeal plate (Fig. 2). The metaphyseal circulation has implications for a vulnerable physis that can "slip" through the hypertrophic zone of the physis. This metaphyseal supply to the neck arises from the extracapsular arterial ring, distinct from the epiphyseal circulation.

Preceding the above experiments, Trueta described the intra- and extraosseous proximal femoral vascular anatomy in adult hips through a series of detailed histological dye studies [9]. The medial circumflex femoral artery (MFCA) branches from the profunda femoris artery, has a predictable branching pattern, and supplies lateral epiphyseal branches which become the dominant

Fig. 1 Specimen from an 18-month-old human cadaver. Evident is the complete independence of the lateral epiphyseal arteries investing the epiphysis and the ascending metaphyseal vessels ending at the physeal plate. This independence persists until physeal closure in adolescence (From Trueta, J: The normal vascular anatomy of the human femoral head during growth. J Bone Joint Surg Br 1957;39-B(2):358–394, with permission)

vascular contribution to the femoral head by age of 18 months. This dominance persists into adulthood. The MFCA ascends the posterolateral femoral neck and, once intracapsular, is invested by a fibrous sheath and adjacent venular system. The artery generally divides into 2–6 spiral branches and heads toward a point between fovea and the inferior articular margin (Fig. 3). These arteries undergo a characteristic arborization in both the coronal and sagittal planes, almost always branching at 90° and directed toward the chondral surface, providing a robust subchondral circulation (Fig. 4). In adulthood, little ascending metaphyseal contribution exists to the femoral head, and only a small anastomosis with the artery of the ligamentum teres (the acetabular branch of the obturator artery) may remain. The artery of the ligamentum teres was found in 100 % of cadaveric specimens aged 13–80 years in a study by Wertheimer et al. [10], but more than two third did not fill with dye on histological section, could not be visualized angiographically, or were of such small caliber to be deemed clinically insignificant. In 18 % of specimens, an anastomosis of the lateral epiphyseal with the ascending metaphyseal arteries was present and was more common in females. Such an anatomical variant is thought to be protective and to explain the lower

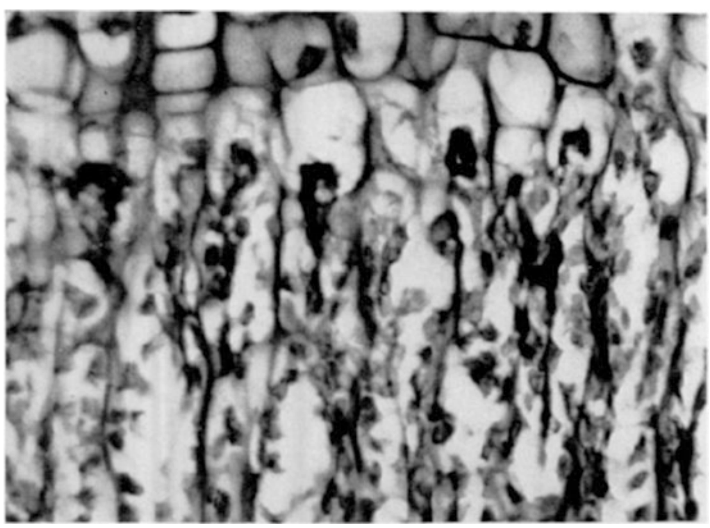


Fig. 2 During adolescence, terminal branches of the ascending metaphyseal circulation do not cross the closing physis, instead ending in the hypertrophic zone of the growth plate (From Trueta J: The normal vascular anatomy of the human femoral head during growth. J Bone Joint Surg Br 1957;39-B(2): 358–394, with permission)

rate of avascular necrosis (AVN) in female patients with SCFE [11]. Ganz and colleagues performed detailed dye investigations of the MFCA and lateral epiphyseal vessels, the protection of which can allow for complex surgical exposure of the hip by preservation of the epiphyseal circulation [12].

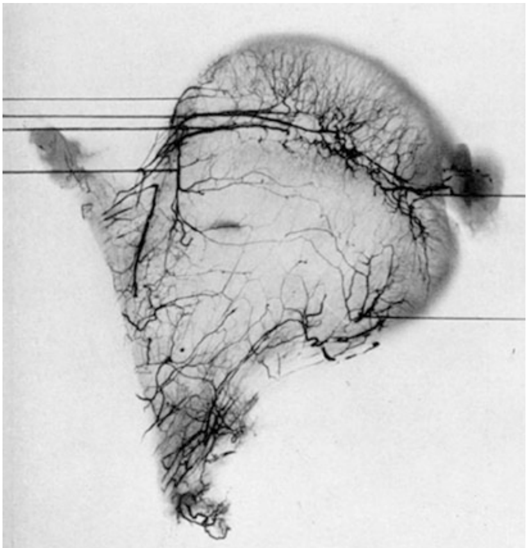


Fig. 3 The medial circumflex femoral artery becomes intracapsular in the posterolateral femoral neck, and its lateral epiphyseal vessels provide the majority of the nutrition to the femoral head after physeal closure. There is minimal ascending metaphyseal contribution and varying but small anastomoses with the artery of the ligamentum teres (From Trueta J and Harrison MH: The normal vascular anatomy of the femoral head in adult man. J Bone Joint Surg Br 1953;35-B(3): 442–461, with permission)

Osseous and Physeal Anatomy

At first consideration, a slipped capital femoral epiphysis may appear similar to a Salter-Harris I growth plate injury. However, multiple factors differentiate SCFE from an acute fracture, including antecedent physeal lysis, slower displacement, and intact periosteum. There are a number of anatomical characteristics that likely contribute to the pathogenesis of SCFE and physeal instability, including variations in proximal femoral and acetabular anatomy.

Gelberman et al. [13] compared torsional profiles from axial CT scans of femora with unilateral slipped epiphyses and described relative femoral retroversion in affected hips (averaging 1.0 vs. 6.3° of anteversion). He postulated that abnormal torsional stresses, stemming from decreased anteversion, could contribute to rotational instability across the developing growth plate. Later, Sankar investigated acetabular anatomy after treatment of unilateral SCFE, demonstrating acetabular retroversion and over-coverage in the unaffected hip, a finding with implications for

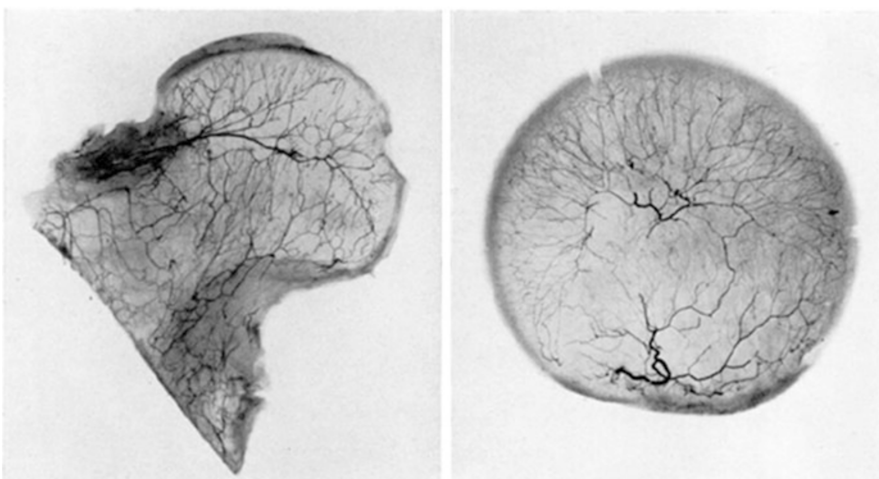


Fig. 4 The intraosseous lateral epiphyseal vessels undergo characteristic branching at right angles in all planes and point toward the chondro-osseous junction (From Trueta J and Harrison MH: The normal vascular anatomy of the femoral head in adult man. J Bone Joint Surg Br 1953;35-B(3): 442–461, with permission)

SCFE development and the development of posttreatment impingement in the unaffected hip [14].

The femoral neck-shaft angle decreases from 160° at birth to an average of 125º by adolescence with changes in orientation of the physis. Speer et al. described a growth spurt around age 7 resulting in asymmetric neck lengthening and increased verticality of the physis [15]. Mirkopoulos et al. [16] found that patients with unilateral SCFE had steeper physes than both their unaffected contralateral hips and age-matched controls. A 14° increase in radiographic slope occurred between 1 and 18 years, with the most rapid increase occurring between ages 9 and 12 with no relationship to sex or other demographic factor. This higher "physeal inclination angle" and resultant increase in shear vector parallel to the growth plate may contribute to epiphyseal translation and development of an SCFE [15, 17]. Since physiologic loads can create shear forces in excess of six times body weight, obesity further contributes to epiphyseal instability.

The perichondrial ring also contributes to the load-carrying capacity of the physeal plate. The perichondrial ring thins and attenuates during development, and consequently less shear force is necessary to cause epiphyseal displacement by early adolescence [15, 18]. Physiologic shear forces alone can displace an adolescent proximal femoral epiphysis ex vivo [18, 19], furthering the mechanical contribution to SCFE development.

Although the exact pathogenesis is unclear, physeal columnar height and organization are significantly altered in SCFE. Because of the already thinned perichondrial ring after 3 years of age, the large surface area of the undulating, interlocking mammillary processes provides the greatest internal support of the epiphysis. SCFE is characterized by physeal widening up to 12 mm (normal range, 2–6 mm), a widened hypertrophic zone comprising 60–80 % of the physeal height, enlargement of chondrocytes, columnar disorganization, cleft formation, higher proteoglycan and extracellular matrix concentrations throughout the physis, and a general disruption in orderly chondrocyte differentiation and endochondral ossification (Fig. 5) [15, 17, 20]. Radiographic physeal widening implies a mechanically weakened physis susceptible to "unlocking" of the mammillary processes and further destabilization.

The epiphyseal tubercle is an anatomical feature receiving increased attention. It is a prominence consistently located among the mammillary processes of the posterosuperior quadrant of the

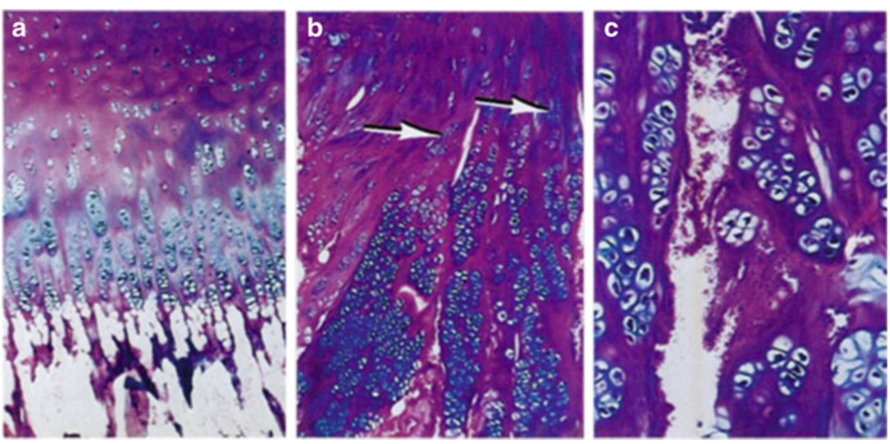


Fig. 5 Normal proximal femoral physis with extracellular matrix primarily in the resting zone and excellent columnar organization of developing chondrocytes (**a**). Proximal femoral physis of an SCFE patient showing ECM in the proliferating and hypertrophic zones (**b**) and a frank cleft in the hypertrophic zone with disorganized ECM and erythrocyte invasion (**c**) (From Ippolito E, Mickelson MR, Ponseti IV: A histochemical study of slipped capital femoral epiphysis. J Bone Joint Surg Am 1981;63(7):1109–13, with permission)

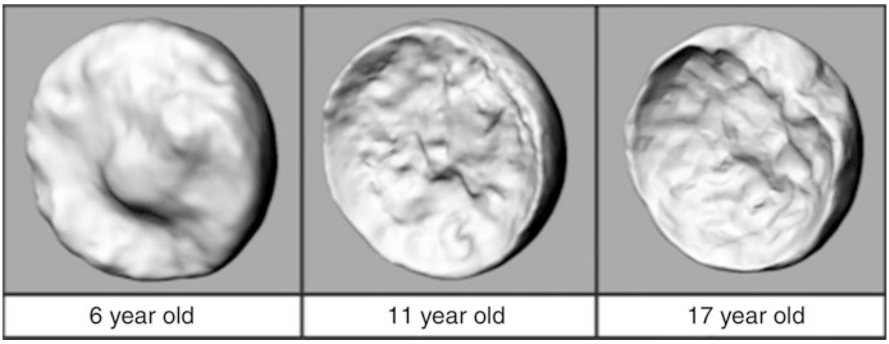


Fig. 6 Three-dimensional CT reconstructions of the epiphyseal tubercle in the posterosuperior epiphysis, decreasing in normative size as a child ages. Also appreciable is the increase in physeal cupping over time (From Liu RW, Armstrong DG, Levine AD et al.: An Anatomic Study of the Epiphyseal Tubercle and Its Importance in the Pathogenesis of Slipped Capital Femoral Epiphysis. J Bone Joint Surg Am 2013;95:e34(1–8), with permission)

epiphysis [21]. The tubercle averages 4 mm in height, is always below the foraminae for the lateral epiphyseal vessels, and is postulated to confer mechanical strength to the physeal plate. It is considered a possible “keystone” for physeal stability, decreasing in normative size and surface area during childhood and adolescence with a concomitant increase in peripheral physeal cupping, which is thought to provide compensatory stability. Liu et al. postulate that the epiphysis internally rotates on the epiphyseal tubercle and that a widened physis could contribute to epiphyseal dislodgement [22]. Since the lateral epiphyseal arteries are immediately adjacent to and above the epiphyseal tubercle, this could explain the low rate of osteonecrosis in chronic, stable slips (i.e., minimal displacement of the lateral epiphyseal vessels; Fig. 6).

Related Conditions

Suspicion of an endocrinologic disturbance in the pathogenesis of SCFE arose due to the known stippling effect of congenital hypothyroidism, as thyroid hormone (T3) is necessary for normal skeletal development and specifically chondrocyte differentiation. While a common presentation of SCFE is that of an obese, “hypogonadal” male during the adolescent growth spurt, the majority of SCFEs occur in the absence of endocrinopathy [23, 24].

A stature test has been used to identify patients with SCFE and a concomitant endocrine abnormality [25]. Patients with SCFE who were below the 10th percentile for height comprised 90.9 % of all endocrinopathies, and all such patients remained below the 10th percentile throughout adolescence. These children could be identified with a sensitivity of 90 % and negative predictive value (NPV) of 98.6 % based on height alone and hence could be targeted with screening laboratory tests and appropriate workup. Loder et al. described and later reaffirmed the prognostic implications of an “age-weight” and “age-height” test to distinguish a typical (idiopathic) from an atypical (usually endocrine-related) SCFE [26]. Patients were grouped into six categories based on age (<10 years, 10–16 years, >16 years) and weight or height (greater or less than the 50th percentile). In combined variable models, extremes of age and weight; height and weight; and age, height, and weight were associated with increased odds ratios of an atypical slipped epiphysis (Table 1). As these tests all have high NPV, an orthopedic surgeon can feel reasonably confident that an SCFE is idiopathic when the age-weight, age-height, and stature tests are negative. Laboratory screening will not be cost-effective unless it is guided by specific patient characteristics and appropriate clinical suspicion.

Loder et al. [27] published a review of all previously reported slipped capital epiphyses in patients with endocrine disturbances, separating

Table 1 Odds ratios of atypical SCFE based on deviations from norms of age, weight, and height (Adapted from Loder RT, Starnes T, Dikos G: Atypical and typical (idiopathic) slipped capital femoral epiphysis. Reconfirmation of the age-weight test and description of the height and age-height tests. J Bone Joint Surg Am 2006;88 (7):1574–81, with permission)

Group	Odds ratio
Age <10 or >16 years	7.4
Height <50th percentile	6.0
Age and height	
Age <10 or >16 years	5.8
Height <50th percentile	15.0
Weight and height	
Weight <50th percentile	7.4
Height <50th percentile	12.8
Age, weight, and height	
Age <10 or >16 years	4.9
Weight <50th percentile	4.5
Height <50th percentile	14.1

patients into three groups: hypothyroidism, growth hormone deficiency, and all others. Hypothyroidism was the most common diagnosis (40 % of 85 patients), and in his series, the development of SCFE usually antedated the diagnosis of thyroid disturbance that was generally made during the same hospitalization. All patients with growth hormone deficiency (25 %) that had been diagnosed prior to developing an SCFE experienced the shortest symptom duration before a slip diagnosis, and 92 % developed a slip during their hormone replacement therapy. All patients with other endocrine disturbances such as panhypopituitarism, craniopharyngioma, and multiple endocrine neoplasia (35 %) were diagnosed later in adolescence (average age 17.4 years) and an average of 3 years from first SCFE symptoms. Bilaterality has been reported more commonly in endocrine-related SCFE [27, 28], with many unilateral presentations progressing to contralateral involvement within the first 18 months. Increased rates of SCFE have also been demonstrated in patients with renal osteodystrophy and secondary hyperparathyroidism [29], postradiation [30], hypogonadism [31], and Down syndrome [32].

A unique combination of histological, vascular, anatomical, and endocrinologic factors all contribute to physeal stability and the pathogenesis of SCFE. The proximal femoral physis represents an area of rapid cellular proliferation more vulnerable to instability than areas of slower turnover, is characterized by a unique temporal susceptibility that can be heightened by the body's endocrinologic milieu, and is nourished by a fragile blood supply. A thorough understanding of the complexity of proximal femoral anatomy is necessary before any surgical treatment of SCFE is undertaken.

Clinical Evaluation

Patients with slipped capital epiphyses can present to a physician in a delayed or acute fashion, often providing clues to chronicity and stability. Patients may have long-standing symptoms lasting months prior to physician evaluation. The most common presentations include limp and pain in the affected groin, lateral or posterior hip, thigh, or ipsilateral knee. Knee pain in SCFE is referred, which implies a reflex arc involving somatic sensory nerves ending at the same spinal level (Fig. 7). This differs from radiating pain, which is thought to be caused by irritation of obturator nerve branches that course to the medial knee. Knee pain, present in 15–50 % of SCFE, leads to high rates of misdiagnosis, extra radiographs, and higher-grade SCFE at treatment [33, 34] and could result in errant surgical procedures directed at nonexistent knee pathology or a second knee diagnosis such as Osgood-Schlatter [35], reinforcing the need for careful evaluation. A stable slip, Medicaid insurance, and distal thigh/knee pain are the strongest independent predictors of a delay in diagnosis of SCFE [34].

Altered gait patterns in SCFE include components of antalgic, "waddling," or Trendelenburg gait, with an externally rotated foot progression angle. Only a small percentage of limp in SCFE is painless [36]. Motion can be severely restricted in multiple planes, and rotational profiles should be compared bilaterally. Patients may have weak hip abduction, decreased hip flexion, decreased internal rotation, obligate external rotation with hip flexion (Drehmann sign), and the development of synovitis precipitating a flexed posture or even hip flexion contracture. A positive

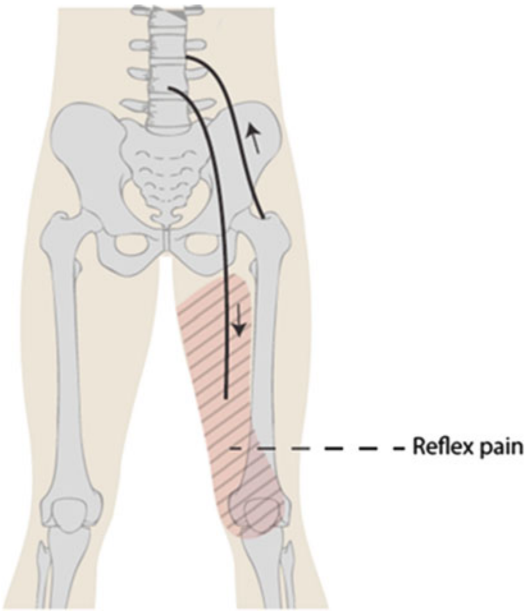


Fig. 7 Reflex arc of referred pain in SCFE, in which a reflex of afferent somatic sensory nerves from the hip terminates at a spinal level in proximity to efferent pain signals to the knee and thigh

Drehmann sign is elicited when the examiner passive flexes the supine patient's hip, which then falls into obligate external rotation and abduction [37].

Reviews of large national databases report an SCFE incidence rate of 10 per 100,000, with a 1.4:2.0 female/male ratio. The average age of diagnosis is 12 years, and most patients presenting outside of ages 10 and 16 represent atypical SCFE that may be associated with endocrinologic diagnoses [26]. Blacks, Hispanics, and Pacific Islanders have significantly greater incidence rates than Whites. There has been a consistent finding of higher SCFE incidence north of 40° latitude, with the greatest incidences in the United States found in the Northeast and West and the lowest rates in the Midwest and South. A seasonal incidence pattern has also been noted, with most northern latitudes presenting in the summer months and most southern latitudes in the winter [38, 39]. Recent data have indicated a trend of decreasing age and increasing frequency of bilaterality at first presentation, with a suspected correlation with increasing rates of childhood obesity [40].

Bilateral SCFE at first presentation has traditionally been reported in approximately 20 %, with higher rates in patients with endocrinopathy [25, 26]. The incidence of metachronous slip, affecting 15–36 % of SCFE, may provide justification for prophylactic pinning of the unaffected hip in at-risk patients [41, 42]. Almost 90 % of metachronous SCFE presents within the first 18 months after treatment of the index slip [43].

Radiographic Evaluation

The primary radiographic test used in suspected SCFE remains an AP and frog pelvis radiograph. Klein et al. [44] described a line constructed along the superior neck on the AP image that should intersect the epiphysis. Failure to do so comprises a positive "Trethowan sign" and may indicate a slipped femoral epiphysis. The sensitivity of this line has been questioned, reportedly missing 61 % of SCFE and underdiagnosing patients in a "pre-slip" phase [45]. Green et al. found similarly low sensitivity of this line, proposing a modification wherein the amount of the epiphysis lateral to Klein's line was compared between the two hips on the AP image, with a 2 mm side-to-side difference highly suspicious of SCFE [46]. There are numerous subtle radiographic findings indicative of early slippage, including widening and irregularity of the affected physis, sharpening of the metaphyseal border of the head, loss of anterior concavity to the head-neck junction on the lateral view, and subtle periosteal elevation [44, 47]; however, lateral radiographs are generally more sensitive than the AP images, particularly in the earliest phases of slipping (Fig. 8). Most SCFE is characterized by anterior translation and external rotation of the metaphysis relative to the epiphysis, which, on an AP projection, is located posterior and inferior to the metaphysis. As a result, the total epiphyseal height may appear decreased and a double-density or "metaphyseal blanch" sign described by Steel [48] can be present. Chronic SCFE can result in uncoverage and resorption of the superior metaphysis, with periosteal reaction, osseous formation, and beaking along the inferomedial neck (Fig. 9).

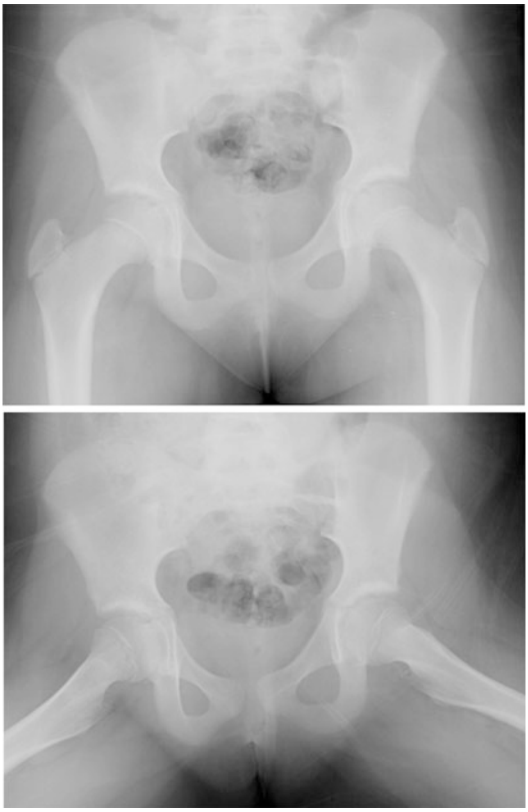


Fig. 8 AP and lateral radiographs of a right slipped capital epiphysis in a 10-year-old female. On the AP projection, Klein's line intersects both epiphyses. A modified Klein's line would reveal asymmetry in the quantity of epiphysis intersected. The right physis is obviously widened compared to the unaffected left. A frog-lateral projection reveals posterior slippage and loss of anterior convexity of the head-neck junction courtesy of Ira Zaltz, M.D.

The role of advanced imaging is controversial and generally used on a case-by-case basis. Ultrasound can detect the presence of hip effusion and a step-off at the epiphyseal-metaphyseal junction [49], but provides little of the morphological detail necessary for surgical planning. Computed tomography (CT) allows for the assessment of physeal closure or morphological assessment of proximal femoral deformity, particularly with three-dimensional reconstruction if complex osteotomy is planned. Intra-articular hardware penetration is also best assessed by CT [50]. Magnetic resonance imaging (MRI) can assess vascularity to the femoral head or the degree of AVN, although hardware scatter can compromise image quality.

Fig. 9 AP hip radiographic demonstrating chronic remodeling in chronic SCFE. There are periosteal reaction and beaking along the inferior neck and rounding and blunting of the superior metaphysis due to resorption courtesy of Ira Zaltz, M.D.

Classification

Symptom duration has been used to describe SCFE as acute, chronic, or acute on chronic. Acute SCFE comprises 10–15 % of all SCFEs and presents within 3 weeks of the onset of symptoms. Acute presentation is associated with a higher rate of AVN [51] and can present after trauma or an identifiable inciting injury. Chronic SCFE, comprising 85 % of cases, implies at least 3 weeks of symptoms, often presenting after prior physician evaluation or incorrect diagnoses, when symptoms fail to resolve. Patients with prodromal symptoms of pain and limp have been known to experience a new injury with immediate worsening, a so-called acute-on-chronic slip [11]. Temporal classifications are useful in describing chronicity but have less prognostic value.

Loder introduced the concept of physeal stability in his classification, categorizing SCFE as either "stable" or "unstable" based on the patient's ability to bear weight, even with crutches [43]. Physeal stability was predictive of osteonecrosis rates, with 47 % of unstable and 0 % of stable slips developing AVN within 6–18 months. There is marked heterogeneity in the application of the stable/unstable classification in the literature [52]. More recently analyzed historical data suggests the rate of osteonecrosis in unstable SCFE is closer to 23.9 % [53].

Radiographic severity was originally described by epiphyseal displacement as a fraction of total physeal diameter, expressed as a percentage [47, 54]. By this grading, slips can be mild (<33 %), moderate (33–50 %), or severe (>50 %). A more commonly used classification is the slip angle of Southwick [55], in which the difference in the angle subtended by the proximal femoral physis and the ipsilateral femoral shaft is compared between affected and unaffected sides. Differences less than 30° are mild, between 30° and 50° are moderate, and greater than 50° are severe. Since femoral rotation can vary depending on radiographic technique and patient comfort, the Southwick grading is inconsistent. Cross-table and frog-lateral projections have been found to optimally quantify the magnitude of SCFE by the Southwick angle when the femur is abducted to 45° with less than 30° of external rotation [56]. In cases where a contralateral comparison is unavailable (i.e., contralateral slip), Southwick described "normal" head-shaft angles for ready comparison.

Treatment

Slipped capital femoral epiphysis demands surgical management except in rare instances, as stabilization of the epiphysis and early fusion of the proximal femoral physis prevents further displacement and is a common goal of all treatment modalities. The preferred treatment of SCFE has undergone dramatic change as surgical techniques have become more refined, and improved imaging has allowed complex definition of deformity and precise surgical planning. There remains considerable debate about the optimal treatment of stable SCFE. Recommendations will evolve if long-term, prospective data becomes available and as surgeons are trained in increasingly complex hip surgery.

Closed Reduction and Spica Casting

Deliberate manipulation and closed reduction of a slipped epiphysis is largely historical. Some authors have reported low rates of osteonecrosis if gentle, deliberate manipulation was performed within 24 h of presentation, presumably because contracture and shortening of the ascending cervical branches of the MFCA had yet to occur [57]. "Spontaneous reductions" refer to a nondeliberate change in metaphyseal-epiphyseal relationships that usually occurs during patient positioning on a surgical table immediately preceding treatment. Conversely, further displacement can also occur during positioning for a frog-leg lateral radiograph, leading some to advocate against these images in the unstable slip. Spontaneous reduction has been reported to occur in up to 90 % of unstable and 8 % of stable slips [43, 52].

Casting as a sole treatment modality or in conjunction with closed reduction was almost entirely abandoned in the developed world by the late 1950s. Historically, patients were immobilized in a position of extension, abduction, and external rotation. Many series have reported high rates of slip progression, chondrolysis, pressure ulcers [3, 7], and recurrence of slip even up to 11 months after recumbent spica immobilization [47]. Long-term Swedish data with an average of 46-year follow-up revealed a harmful influence of both closed reduction and hip spica casting when compared to symptomatic or no treatment [58]. Reduction and hip spica casting resulted in higher rates of subsequent hip surgery and more late arthrosis.

Open Reduction and Physiodesis

As the primary goal of SCFE treatment is stabilization and arrest of the proximal femoral physis, surgeons have attempted to achieve this with formal open physiodesis, usually using a corticocancellous bone graft. Since the rates of physiodesis with the earliest, large metallic implants (Smith-Peterson nails, Hagie pins) were unsatisfactory, Heyman and Herndon described manipulative reduction and bone peg epiphysiodesis [59]. Their patients experienced rapid fusion of the proximal femoral physis (2.3 months), much shorter than classic conservative treatment, and the authors hypothesized that the inducement of vascular channels to the epiphysis was responsible. Comparably good functional series with open epiphysiodesis were reproduced for decades thereafter [11]. A fundamental drawback to the procedure was a larger and more morbid anterior exposure, and it subsequently became less popular as screw fixation methods improved.

In Situ Fixation

In situ screw fixation to prevent further epiphyseal displacement and to induce physeal arrest is currently the most common treatment for SCFE of all types (stable or unstable), regardless of the degree of deformity [52]. Correct insertion requires placing the screw perpendicular to the epiphysis, crossing the physis into the geometric center of the femoral head [60]. With increasing slip displacement, the epiphysis location is more posterior relative to the femoral neck, requiring a more anterior starting point on the neck in order to cross perpendicularly into the epiphysis (Fig. 10). Percutaneous fixation was accomplished exclusively with solid or threaded pins until the introduction of screws with both threaded and unthreaded segments. There are many current variations in the technique and controversies regarding placement [61].

Percutaneous in situ fixation can be performed by positioning the patient supine either on a radiolucent operating table or on a fracture table. The main difference in the technique using these approaches is that the limb is moved to obtain lateral x-rays when using a radiolucent table, whereas the fluoroscope is moved when a fracture table is used. Advantages of the fracture table technique include less movement of the patient, lower chance of bending a pin, and consistent and reproducible AP and lateral images. The main disadvantage of the fracture table technique is difficulty obtaining clear lateral radiographs of the hip joint, especially in extremely obese

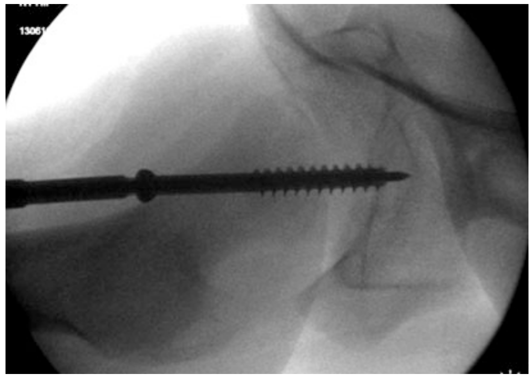
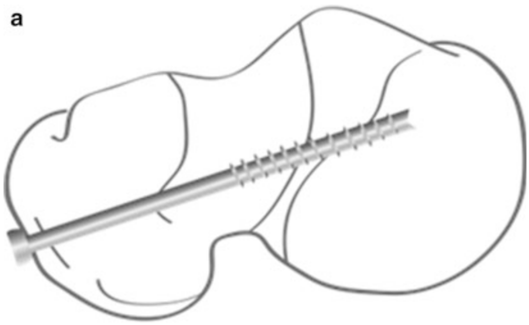


Fig. 10 Pictured is an intraoperative fluoroscopic image of a moderate slip with relative posterior epiphyseal displacement. The posteriorly displaced epiphysis necessitates an anterior starting point at the base of the femoral neck cross the physis perpendicularly and engage center-center in the head courtesy of Ira Zaltz, M.D.

patients with severe epiphyseal displacement. Most authors use cannulated screws to increase the chances of center-center fixation [62]. There is no biomechanical or clinical advantage to the use of multiple screws versus a single screw [63, 60]. Liu et al. suggest that single in situ screw fixation should not be placed in the posterosuperior quadrant of the femoral head, the location of the epiphyseal tubercle, because the epiphysis could theoretically pivot around this single, fixed point [22].

The most important technical considerations during percutaneous in situ fixation are center-center fixation within the epiphysis and avoiding intra-articular penetration. A lateral femoral starting point may appear radiographically central on an AP image though maldirected on a lateral image, distinguishing in situ SCFE fixation technique from pinning an adult hip fracture (Fig. 11). Walters and Simon [64] geometrically detailed the manner by which joint penetration could be overlooked, even with seemingly intraosseous metallic pins on AP, lateral, and frog-lateral images. They described a “safe zone” for placement when static images are obtained. Despite exacting radiographic techniques, Senthi demonstrated that screws terminating closer than 4 mm to the subchondral bone on lateral images may penetrate the joint [50]. Other methods including injection of dye into a cannulated screw were a



b

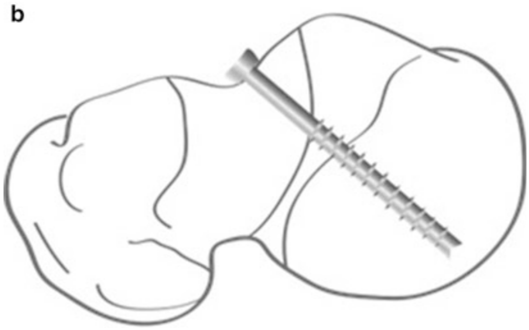


Fig. 11 Pictured are (**a**) an erroneous lateral starting point, which would represent failure to appreciate proximal femoral deformity and which could appear correctly positioned on an AP radiograph, and (**b**) a properly positioned screw that would penetrate the anterior femoral neck and be oriented posteriorly

developed to mitigate risk of screw penetration and development of chondrolysis [65].

Prophylactic pinning of a radiographically and clinically normal hip is generally reserved for patients with non-idiopathic SCFE, i.e., those <10 or >16 years of age, SCFE associated with endocrinopathy, or in obese patients with delayed presentation in whom the surgeon suspects a delay would ensue in the presentation of metachronous SCFE. Even in the case of unilateral SCFE in a patient with endocrinopathy, wherein the metachronous slip rate is 70 %, routine prophylactic pinning of the asymptomatic hip has been contested [25]. There is a growing body of evidence that supports routine prophylactic in situ fixation in unilateral SCFE as the associated complication rate is acceptable compared to the high incidence of complications from a contralateral SCFE [66–68].

Contemporary Surgical Treatments

Modern open surgery for treatment of SCFE can be technically challenging, and its role remains controversial. Unsatisfactory clinical results reported in 10–20 % of patients treated with in situ pin fixation have led some to advocate that for severe deformities, surgeons should attempt to downgrade the degree of deformity or to correct the deformity in order to mitigate the risk of cartilage damage [69–72]. For mild SCFE or healed proximal femoral deformity producing impingement, surgical dislocation and limited anterior open or arthroscopic approaches with osteochondroplasty have been reported [73–75].

Osteotomy can be performed at the intertrochanteric, basicervical, or subcapital level depending on the extent of physeal healing and degree of deformity. Goals of this treatment include redirection of the femoral head to minimize impingement and to improve functional range of motion. Contracture of the posterior retinacular vessels occurs in the setting of a chronic slip, necessitating femoral neck shortening to avoid tension causing occlusion and AVN if a head-neck osteotomy is to be performed (Fig. 12). The modified Dunn procedure, an intracapsular wedge osteotomy of the femoral neck with reduction of the head, has been reported in the treatment of unstable SCFE with short-term results [76–79].

Loder suggests that a surgical dislocation approach has insufficient evidence in either stable or unstable situations, citing an increased rate of AVN using surgical dislocation/modified Dunn as a treatment for stable SCFE (7 % vs. 0 %). He recommends that select centers undertake a prospective evaluation of surgical dislocation and modified Dunn osteotomy, cautioning against its routine use [52].

Complications

All treatments of slipped capital femoral epiphysis share the common goals of epiphyseal stabilization and preventing serious complications including chondrolysis and osteonecrosis of the femoral

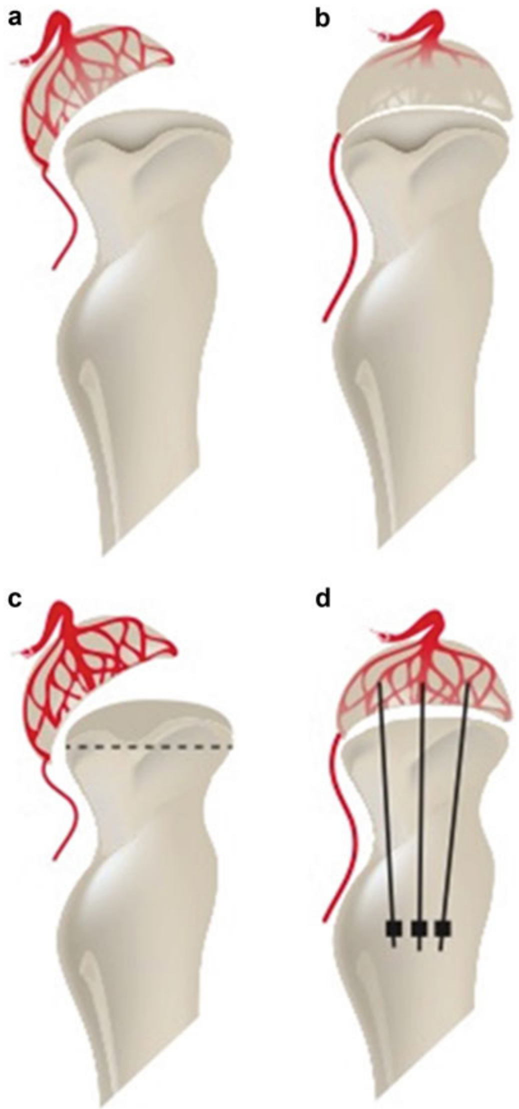


Fig. 12 In chronic SCFE, the posterior vessels are shortened and contracted (**a**), and acute reduction results in high rates of AVN (**b**). A functional shortening and relaxation of the epiphyseal vasculature can be achieved with a cuneiform neck osteotomy (**c**) preceding epiphyseal reduction (**d**) (Adapted from Dunn, DM: The Treatment of Adolescent Slipping of the Upper Femoral Epiphysis. J Bone Joint Surg Br 1964;46:621–9, with permission)

epiphysis. Loder's original description of physeal stability reported 0 % AVN in stable and 47 % in unstable slips [43]. Recently reported composite rates from 15 clinical studies place the osteonecrosis rate of unstable slips at 23.9 % [53]. It remains controversial whether acute reduction contributes to or could improve vascularity of the epiphysis, as superselective angiography of the superior retinacular vessels has shown restoration of epiphyseal flow after acute reduction of the epiphysis [80].

Chondrolysis, a loss of the cartilaginous surface of the femoral head and acetabulum, has been reported with all methods of treatment of SCFE. Clinically, patients present with global loss of motion and pain. Radiographically, the diagnosis is suspected when there is 50 % loss in joint space compared to the unaffected side or less than 3 mm of measurable joint space. The highest rates occur following nonoperative treatment [81], especially high-grade slips [54] and in 1.5 % of patients treated with percutaneous in situ fixation [82]. Chondrolysis can occur with or without evidence of guidewire or implant penetration during or following surgery, though most authors recommend avoiding intra-articular penetration regardless of its causative role [83]. Anterior femoroacetabular impingement as a result of residual femoral metaphyseal prominence has been implicated in late-presenting chondrolysis [84].

Recurrence or progression of a slipped epiphysis has been reported even after prolonged spica cast treatment [47] and other modes of stabilization [85]. It is more likely to occur in non-idiopathic SCFE or following in situ stabilization of severe deformities. The true prevalence of this complication is not known with specificity, though this occurs infrequently with the use of modern imaging and accurate screw placement techniques.

Summary

SCFE is a common adolescent hip disorder. The physis is uniquely susceptible to lysis during specific periods of growth, and the risk of epiphyseal displacement is compounded by normal proximal femoral development, physeal orientation, acetabular morphology, and endocrinologic factors. It is most clinically useful to classify SCFE by physeal stability, determined by the patient's ability to bear weight. Rapid diagnosis can be made by careful clinical history, examination, and performance of AP and frog-lateral radiographs. Single in situ screw fixation across the physis predictably stabilizes the epiphysis and accelerates physeal closure. There is increasing use of open reduction especially following acute SCFE and reconstruction for symptomatic chronic SCFE though the precise role for these procedures is not yet defined.

References

1. Paré A. Cinq Livres De Chirurgie. Wechel; Paris 1572.
2. Müller E. The classic: 1 on the deflection of the femoral neck in childhood a new syndrome. Clin Orthop Relat Res. 1966;48:7–10.
3. Whitman RI. Further observations on depression of the neck of the femur in early life; including fracture of the neck of the femur, separation of the epiphysis and simple coxa vara. Ann Surg. 1900;31:145–62.
4. Frohelich. Ztschr F Orthop Chir. 1904;12:80.
5. Key JA. Epiphyseal coxa vara or displacement of the capital epiphysis of the femur in adolescence. J Bone Joint Surg. 1926;8:53–117.
6. Sprengel. Arch F Klin Chir. 1898;57:805.
7. Wilson PD. Displacement of upper epiphysis of femur treated by open reduction. J Am Med Assoc. 1924;83:1749–56.
8. Trueta J. The normal vascular anatomy of the human femoral head during growth. J Bone Joint Surg Br. 1957;39-B:358–94.
9. Trueta J, Harrison MH. The normal vascular anatomy of the femoral head in adult man. J Bone Joint Surg Br. 1953;35-B:442–61.
10. Wertheimer LG, Lopes Sde L. Arterial supply of the femoral head. A combined angiographic and histological study. J Bone Joint Surg Am. 1971;53:545–56.
11. Aadalen RJ, Weiner DS, Hoyt W, Herndon CH. Acute slipped capital femoral epiphysis. J Bone Joint Surg Am. 1974;56:1473–87.
12. Gautier E, Ganz K, Krugel N, Gill T, Ganz R. Anatomy of the medial femoral circumflex artery and its surgical implications. J Bone Joint Surg Br. 2000;82:679–83.
13. Gelberman RH, Cohen MS, Shaw BA, Kasser JR, Griffin PP, Wilkinson RH. The association of femoral retroversion with slipped capital femoral epiphysis. J Bone Joint Surg Am. 1986;68:1000–7.
14. Sankar WN, Brighton BK, Kim YJ, Millis MB. Acetabular morphology in slipped capital femoral epiphysis. J Pediatr Orthop. 2011;31:254–8.
15. Speer DP. The John Charnley award paper. Experimental epiphysiolysis: etiologic models slipped capital femoral epiphysis. Hip. 1982:68–88.
16. Mirkopoulos N, Weiner DS, Askew M. The evolving slope of the proximal femoral growth plate relationship to slipped capital femoral epiphysis. J Paediatr Orthop. 1988;8:268–73.
17. Ippolito E, Mickelson MR, Ponseti IV. A histochemical study of slipped capital femoral epiphysis. J Bone Joint Surg Am. 1981;63:1109–13.
18. Sm C, Batterman SC, Brighton CT. Shear strength of the human femoral capital epiphyseal plate. J Bone Joint Surg Am. 1976;58:94–103.
19. Litchman HM, Duffy J. Slipped capital femoral epiphysis: factors affecting shear forces on the epiphyseal plate. J Pediatr Orthop. 1984;4:745–8.
20. Ponseti IV, Mcclintock R. The pathology of slipping of the upper femoral epiphysis. J Bone Joint Surg Am. 1956;38-A:71–83.
21. Tayton K. Does the upper femoral epiphysis slip or rotate? J Bone Joint Surg Br. 2007;89:1402–6.
22. Liu RW, Armstrong DG, Levine AD, Gilmore A, Thompson GH, Cooperman DR. An anatomic study of the epiphyseal tubercle and its importance in the pathogenesis of slipped capital femoral epiphysis. J Bone Joint Surg Am. 2013;95:E341.
23. Cd R, Nelson C, Eversman J. Growth hormone levels in slipped capital femoral epiphysis. J Bone Joint Surg Am. 1972;54:1224–6.
24. Mann DC, Weddington J, Richton S. Hormonal studies in patients with slipped capital femoral epiphysis without evidence of endocrinopathy. J Paediatr Orthop. 1988;8:543–5.
25. Burrow SR, Alman B, Wright JG. Short stature as a screening test for endocrinopathy in slipped capital femoral epiphysis. J Bone Joint Surg Br. 2001;83:263–8.
26. Loder RT, Starnes T, Dikos G. Atypical and typical (idiopathic) slipped capital femoral epiphysis. Reconfirmation of the age-weight test and description of the height and age-height tests. J Bone Joint Surg Am. 2006;88:1574–81.
27. Loder RT, Wittenberg B, Desilva G. Slipped capital femoral epiphysis associated with endocrine disorders. J Paediatr Orthop. 1995;15:349–56.
28. Wells D, King JD, Roe TF, Kaufman FR. Review of slipped capital femoral epiphysis associated with endocrine disease. J Pediatr Orthop. 1993;13:610–4.
29. Loder RT, Hensinger RN. Slipped capital femoral epiphysis associated with renal failure osteodystrophy. J Paediatr Orthop. 1997;17:205–11.
30. Wolf E, Berdon WE, Cassady JR, Baker DH, Freiberger R, Paviov H. Slipped femoral capital epiphysis as a sequela to childhood irradiation for malignant tumors. Radiology. 1977;125:781–4.
31. Hirsch PJ, Hirsch SA. Slipped capital femoral epiphysis. Occurrence after treatment with chorionic gonadotropin. JAMA. 1976;235:751.
32. Stack RE, Peterson LF. Slipped capital femoral epiphysis and Down's disease. Clin Orthop Relat Res. 1966;48:111–7.
33. Matava MJ, Patton CM, Luhmann S, Gordon JE, Schoenecker PL. Knee pain as the initial symptom of slipped capital femoral epiphysis: an analysis of

initial presentation and treatment. J Pediatr Orthop. 1999;19:455–60.
34. Kocher MS, Bishop JA, Weed B, Hresko MT, Millis MB, Kim YJ, Kasser JR. Delay in diagnosis of slipped capital femoral epiphysis. Pediatrics. 2004;113:E322–5.
35. Kaplan SR, Klinghoffer L. Knee pain in slipped femoral capital epiphysis causing a delay in diagnosis. Am J Surg. 1961;101:798–802.
36. Cowell HR. The significance of early diagnosis and treatment of slipping of the capital femoral epiphysis. Clin Orthop Relat Res. 1966;48:89–94.
37. Drehmann F. Drehmann's sign. A clinical examination method in epiphysiolysis (slipping of the upper femoral epiphysis). Description of signs, aetiopathogenetic considerations, clinical experience (author's translation). Z Orthop Ihre Grenzgeb. 1979;117:333–44.
38. Loder RT. The demographics of slipped capital femoral epiphysis. An international multicenter study. Clin Orthop Relat Res. 1996;322:8–27.
39. Lehmann CL, Arons RR, Loder RT, Vitale MG. The epidemiology of slipped capital femoral epiphysis: an update. J Paediatr Orthop. 2006;26:286–90.
40. Nasreddine AY, Heyworth BE, Zurakowski D, Kocher MS. A reduction in body mass index lowers risk for bilateral slipped capital femoral epiphysis. Clin Orthop Relat Res. 2013;471(7):2137–44.
41. Hurley JM, Betz RR, Loder RT, Davidson RS, Alburger PD, Steel HH. Slipped capital femoral epiphysis. The prevalence of late contralateral slip. J Bone Joint Surg Am. 1996;78:226–30.
42. Baghdadi YM, Larson AN, Sierra RJ, Peterson HA, Stans AA. The fate of hips that are not prophylactically pinned after unilateral slipped capital femoral epiphysis. Clin Orthop Relat Res. 2013;471 (7):2124–31.
43. Loder RT, Richards BS, Shapiro PS, Reznick LR, Aronson DD. Acute slipped capital femoral epiphysis: the importance of physeal stability. J Bone Joint Surg Am. 1993;75:1134–40.
44. Klein A, Joplin RJ, Reidy JA, Hanelin J. Roentgenographic features of slipped capital femoral epiphysis. Am J Roentgenol Radium Ther. 1951;66:361–74.
45. Pinkowsky GJ, Hennrikus W. Klein line on the anteroposterior radiograph is not a sensitive diagnostic radiologic test for slipped capital femoral epiphysis. J Pediatr. 2013;162:804–7.
46. Green DW, Mogekwu N, Scher DM, Handler S, Chalmers P, Widmann RF. A modification of Klein's line to improve sensitivity of the anterior-posterior radiograph in slipped capital femoral epiphysis. J Pediatr Orthop. 2009;29:449–53.
47. Wilson PD. The treatment of slipping of the upper femoral epiphysis with minimal displacement. J Bone Joint Surg. 1938;20:379–99.
48. Steel H. The metaphyseal blanch sign of slipped capital femoral epiphysis. J Bone Joint Surg Am. 1986;68:920–2.
49. Kallio PE, Paterson DC, Foster BK, Lequesne GW. Classification in slipped capital femoral epiphysis. Sonographic assessment of stability and remodeling. Clin Orthop Relat Res. 1993;294:196–203.
50. Senthi S, Blyth P, Metcalfe R, Stott NS. Screw placement after pinning of slipped capital femoral epiphysis: a postoperative CT scan study. J Pediatr Orthop. 2011;31:388–92.
51. Aronsson DD, Loder RT. Treatment of the unstable (acute) slipped capital femoral epiphysis. Clin Orthop Relat Res. 1996;322:99–110.
52. Loder RT, Dietz FR. What is the best evidence for the treatment of slipped capital femoral epiphysis? J Paediatr Orthop. 2012;32 Suppl 2:S158–65.
53. Zaltz I, Baca G, Clohisy JC. Unstable SCFE: review of treatment modalities and prevalence of osteonecrosis. Clin Orthop Relat Res. 2013;471(7):2192–8.
54. Wilson PD, Jacobs B, Schecter L. Slipped capital femoral epiphysis: an end-result study. J Bone Joint Surg Am. 1965;47:1128–45.
55. Southwick WO. Osteotomy through the lesser trochanter for slipped capital femoral epiphysis. J Bone Joint Surg Am. 1967;49:807–35.
56. Loder RT. Effect of femur position on the angular measurement of slipped capital femoral epiphysis. J Paediatr Orthop. 2001;21:488–94.
57. Peterson MD, Weiner DS, Green NE, Terry CL. Acute slipped capital femoral epiphysis: the value and safety of urgent manipulative reduction. J Pediatr Orthop. 1997;17:648–54.
58. Ordeberg G, Hansson LI, Sandstrom S. Slipped capital femoral epiphysis in Southern Sweden. Long-term result with closed reduction and hip plaster spica. Clin Orthop Relat Res. 1987;220:148–54.
59. Heyman CH, Herndon CH. Epiphy seodesis for early slipping of the upper femoral epiphysis. J Bone Joint Surg Am. 1954;36-A:539–55.
60. Kibiloski LJ, Doane RM, Karol LA, Haut RC, Loder RT. Biomechanical analysis of single- versus double-screw fixation in slipped capital femoral epiphysis at physiological load levels. J Paediatr Orthop. 1994;14:627–30.
61. Gourineni P. Oblique in situ screw fixation of stable slipped capital femoral epiphysis. J Pediatr Orthop. 2013;33:135–8.
62. Aronson DD, Loder RT. Slipped capital femoral epiphysis in black children. J Paediatr Orthop. 1992;12:74–9.
63. Karol LA, Doane RM, Cornicelli SF, Zak PA, Haut RC, Manoli II A. Single versus double screw fixation for treatment of slipped capital femoral epiphysis: a biomechanical analysis. J Pediatr Orthop. 1992;12:741–5.
64. Walters R, Simon SR. Joint destruction: a sequel of unrecognised pin penetration in patients with slipped capital femoral epiphysis. In: The hip: proceedings of the eighth open scientific meeting of the Hip Society, St Louis: Mosby 1980:145–64.
65. Lehman WB, Grant A, Rose D, Pugh J, Norman A. A method of evaluating possible pin penetration in slipped capital femoral epiphysis using a cannulated

internal fixation device. Clin Orthop Relat Res. 1984;186:65–70.
66. Seller K, Raab P, Wild A, Krauspe R. Risk-benefit analysis of prophylactic pinning in slipped capital femoral epiphysis. J Pediatr Orthop B. 2001;10:192–6.
67. Woelfle JV, Fraitzl CR, Reichel H, Nelitz M. The asymptomatic contralateral hip in unilateral slipped capital femoral epiphysis: morbidity of prophylactic fixation. J Pediatr Orthop B. 2012;21:226–9.
68. Sankar WN, Novais EN, Lee C, Al-Omari AA, Choi PD, Shore BJ. What are the risks of prophylactic pinning to prevent contralateral slipped capital femoral epiphysis? Clin Orthop Relat Res. 2013;471:2118–23.
69. Millis MB, Zaltz I. Emerging concepts in slipped capital femoral epiphysis: editorial comment. Clin Orthop Relat Res. 2013;471(7):2083–4.
70. Witbreuk MM, Bolkenbaas M, Mullender MG, Sierevelt IN, Besselaar PP. The results of downgrading moderate and severe slipped capital femoral epiphysis by an early imhauser femur osteotomy. J Child Orthop. 2009;3:405–10.
71. Sink E, Zaltz I, Heare T, Dayton M. Acetabular cartilage and labral damage observed during surgical hip dislocation for stable slipped capital femoral epiphysis. J Pediatr Orthop. 2010;30:26–30.
72. Lee CB, Matheney T, Yen YM. Case reports: acetabular damage after mild slipped capital femoral epiphysis. Clin Orthop Relat Res. 2013;471:2163–72.
73. Clohisy JC, Zebala LP, Nepple JJ, Pashos G. Combined hip arthroscopy and limited open osteochondroplasty for anterior femoroacetabular impingement. J Bone Joint Surg Am. 2010;92:1697–706.
74. Leunig M, Horowitz K, Manner H, Ganz R. In situ pinning with arthroscopic osteoplasty for mild SCFE: a preliminary technical report. Clin Orthop Relat Res. 2010;468:3160–7.
75. Leunig M, Slongo T, Kleinschmidt M, Ganz R. Subcapital correction osteotomy in slipped capital femoral epiphysis by means of surgical hip dislocation. Oper Orthop Traumatol. 2007;19:389–410.
76. Ziebarth K, Zilkens C, Spencer S, Leunig M, Ganz R, Kim YJ. Capital realignment for moderate and severe SCFE using a modified Dunn procedure. Clin Orthop Relat Res. 2009;467:704–16.
77. Sankar WN, Vanderhave KL, Matheney T, Herrera-Soto JA, Karlen JW. The modified Dunn procedure for unstable slipped capital femoral epiphysis: a multicenter perspective. J Bone Joint Surg Am. 2013;95:585–91.
78. Slongo T, Kakaty D, Krause F, Ziebarth K. Treatment of slipped capital femoral epiphysis with a modified Dunn procedure. J Bone Joint Surg Am. 2010;92:2898–908.
79. Huber H, Dora C, Ramseier LE, Buck F, Dierauer S. Adolescent slipped capital femoral epiphysis treated by a modified Dunn osteotomy with surgical hip dislocation. J Bone Joint Surg Br. 2011;93:833–8.
80. Maeda S, Kita A, Funayama K, Kokubun S. Vascular supply to slipped capital femoral epiphysis. J Pediatr Orthop. 2001;21:664–7.
81. Waldenstrom H. On necrosis of the joint cartilage by epiphyseolysis capitis femoris. Clin Orthop Relat Res. 1930;1996:3–7.
82. Kennedy JP, Weiner DS. Results of slipped capital femoral epiphysis in the black population. J Pediatr Orthop. 1990;10:224–7.
83. Rooks MD, Schmitt EW, Drvaric DM. Unrecognized pin penetration in slipped capital femoral epiphysis. Clin Orthop Relat Res. 1988;234:82–9.
84. Leunig M, Casillas MM, Hamlet M, Hersche O, Notzli H, Slongo T, Ganz R. Slipped capital femoral epiphysis: early mechanical damage to the acetabular cartilage by a prominent femoral metaphysis. Acta Orthop Scand. 2000;71:370–5.
85. Boyer DW, Mickelson MR, Ponseti IV. Slipped capital femoral epiphysis. Long-term follow-up study of one hundred and twenty-one patients. J Bone Joint Surg Am. 1981;63:85–95.

Surgical Technique: Open Reduction Using the Modified-Dunn Technique

34

Ira Zaltz

Contents

Abstract

The modified Dunn technique is the most comprehensive approach to managing femoral deformity and intra-articular damage associated with slipped capital femoral epiphysis. It is a technically demanding procedure that requires an understanding of the SCFE deformity, upper femoral vascular anatomy, and expertise performing the surgical dislocation.

Introduction

The displaced unstable slipped capital femoral epiphysis or the high-grade stable SCFE is a serious orthopedic condition that has been associated with substantial complications including osteonecrosis [1], slip progression [2], chondrolysis, and severe deformity that may lead to significant clinical dysfunction [3, 4]. The accepted approach to managing acute unstable SCFE for several generations of pediatric orthopedic surgeons has been based on the philosophy of minimizing the rate of osteonecrosis by in situ stabilization, an approach that accepted severe deformity as a favorable alternative to the probability of osteonecrosis, often an unsalvageable complication. Selected surgeons have devised approaches intended to minimize the degree of deformity while protecting the posteriorly located vascular retinaculum. These include limited anterior open reduction [5], femoral neck osteotomy through an anterior approach to the hip joint [6, 7], gentle reduction using either

I. Zaltz
Department of Orthopaedic Surgery, William Beaumont Hospital, Royal Oak, MI, USA
e-mail: zaltzira@gmail.com

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_37

manipulation or traction, and, most recently, the modification of the Dunn procedure [8] using the surgical dislocation approach [9].

Technical Evolution

In 2000, Ganz et al. described the practical anatomy of the medial femoral circumflex artery [10] and a surgical approach that could be used to safely dislocate the hip without the risk of iatrogenic osteonecrosis [9]. This technique facilitated an improved understanding of hip mechanics, the role of the acetabular labrum, and the association between specific femoral morphologies and damage to the acetabular rim and labral-chondral complex [11]. Utilization of the surgical dislocation technique in the setting of chronic SCFE substantiated damage to the anterior acetabular articular cartilage and labrum induced by impingement between the upper femoral metaphysis and the anterior acetabulum [12]. Further application of the surgical dislocation approach enabled modification of the Dunn technique of subcapital reorientation [6, 8]. The concept of a safe and reproducible technique to reorient a chronically or acutely displaced upper femoral epiphysis is attractive for surgeons because it has the potential to favorably influence the natural history of the severe SCFE deformity and to prevent complications associated with treatment of high-grade slips including chondrolysis, osteonecrosis, slip progression, hip pain, functional disability, and osteoarthritis. The literature supporting routine use of the modified Dunn technique is generally supportive. It seems that in experienced centers, the technique is associated with an acceptable complication rate when compared to complications that are associated with more traditional management of acute unstable SCFE [1]. Ziebarth et al., Slongo et al., and Huber et al. collectively reported 83 combined stable and unstable SCFE treated using the modified Dunn technique with two cases of osteonecrosis [13, 14, 8]. In contrast, Sankar et al., reporting results from several US center, reported that 7 out of 27 patients developed osteonecrosis [15]. This disparity suggests that careful training and supervised practice may be necessary to prevent an unacceptable rate of complications especially during implementation of this technically demanding procedure.

Surgical Indications

There are no clear surgical indications for the modified Dunn approach. When one considers that the majority of patients treated by in situ fixation function reasonably well for many decades following initial treatment [2, 16], the surgeon must justify the additional risk and higher magnitude of this procedure upon a high likelihood of a poor result following in situ fixation or persistence of a severe deformity. As such, SCFE that are at higher risk for failure are those that may benefit from this procedure. These may include:

1. Displaced, unstable slips
2. High-grade or moderate-grade slips with incomplete physeal healing
3. Hips with progressive displacement following in situ fixation

Patient Evaluation

A patient who presents with a severe slipped epiphysis must be thoroughly evaluated prior to surgical treatment. A thorough history should be used to elucidate factors that may be associated with non-idiopathic SCFE. These include age, weight, past medical history, and family history. If there is a reason to suspect a non-idiopathic SCFE, proper management will increase the likelihood of successful healing and minimize the probability of complication following treatment. Complete radiographic evaluation is necessary to fully evaluate the degree of deformity, chronicity of the SCFE, and state of the contralateral hip. In certain cases of chronic SCFE, a CT scan will permit assessment of physeal healing, enable 3-dimensional visualization of the deformity, and facilitate preoperative planning. The acetabular morphology is becoming increasingly appreciated as a factor in symptomatic SCFE mechanics and can be analyzed preoperatively [17].

Surgical Technique

The technique of the modified Dunn was first used by Leunig et al. to describe acetabular damage associated with SCFE [12] and was first described in detail by Leunig and Ganz [18, 19] and most recently by Ziebarth et al. [8]. It is recommended that surgeons using this technique have familiarity with the vascular anatomy of the upper femur and experience using the surgical dislocation approach.

Setup and Equipment

Since the patient is placed in a lateral decubitus position, an operating table that will permit intraoperative radiographic evaluation of the operative hip is helpful. While it is not necessary to use a radiolucent operating table, the table must permit the surgeon to obtain fluoroscopic images which enable assessment of epiphyseal position, placement of fixation devices, and trochanteric positioning and refixation.

Since most patients with SCFE are obese, surgical visualization can be challenging. It is necessary to have proper retractors that enable both acetabular and femoral exposures. A complement of Langenbeck and Hohmann retractors designed for retraction of the hip joint facilitates difficult exposures. In addition, sharp periosteal elevators, pointed bone-holding forceps, and thin osteotomes are required during various steps of the procedure.

Fixation can be accomplished using cannulated screws, long 4.5 or 6.5 mm fully or partially threaded screws, or Steinmann pins either alone or in combination. There is no uniform method of reliable fixation, and each published series has reports of hardware failure. Surgeons have reported using threaded and smooth wires, solid screws, and cannulated screws. It is best to have various fixation options depending upon the size of the patient, the size of the epiphysis, and the surgeon's ability to visualize the epiphysis intraoperatively.

Intraoperative fluoroscopic imaging capability is very helpful in order to gauge the orientation of the epiphysis following reduction, the location of the trochanter relative to the femoral epiphysis, and the position and orientation of the fixation devices.

Procedure

After the induction of anesthesia and the administration of appropriate preoperative antibiotics, the patient is placed in a lateral decubitus position with an axillary roll placed beneath the contralateral chest and pads beneath the leg in order to protect the brachial plexus and to prevent peroneal nerve injury. While a beanbag can be used to maintain the lateral position, rigid holders such as a peg board or Montreal system permit more rigid positioning and minimize the risk of patient movement. Positioning the operative extremity on a support is necessary in order to relax the fascia lata and to permit proper femoral positioning during exposure and dissection. While prefabricated foam limb supports are commercially available, they may be too wide to permit sufficient femoral flexion, adduction, and external rotation that are required to mobilize the femoral epiphysis. Consequently, positioning the operative leg on blanket rolls will permit necessary mobility.

After preparing the skin the affected limb, hip, and entire hemipelvis should be draped as a sterile field. A sufficiently long skin incision specific to the patient's size is required. In contrast to the described technique for a surgical dislocation, it is helpful to curve the incision slightly posteriorly in order to facilitate access to the upper femur. While surgical dislocation procedures can be performed through relatively small incisions in asthenic individuals, many patients with SCFE have plethoric and dense fatty tissue that requires mobilization in order to safely expose the upper femur in a manner that will allow safe dissection and room for inserting fixation.

The fascia lata is then incised in line with the skin incision and the gluteus fascia is incised between the anterior gluteus maximus and the posterior tensor fascia muscle in order to avoid dividing fibers of the gluteus maximus. The gluteus maximus will require sufficient mobility to access the upper femur, and this can be addressed by either releasing the fascial interval proximal to the ilium or releasing the gluteus maximus tendon (with a cuff for subsequent repair). The trochanteric bursa is then incised which will allow the muscle to be safely retracted posteriorly.

The objective of the surgical exposure of the capsule is to preserve the ascending branch of the medial circumflex artery while providing access to the upper femur through a generous capsulotomy. The anatomy and course of the artery has been described in detail [10]. Capsular exposure is accomplished by first reflecting the gluteus minimus and vastus lateralis anteriorly with both attached to a mobilized fragment of the greater trochanter and then elevating the gluteus minimus muscle and remaining vastus lateralis muscles from the capsule. The anatomic landmarks defining the extent of capsule exposure are the piriformis tendon posteriorly, reflected head of the rectus femoris proximally, direct head of rectus tendon medially, and vastus intermedius inferiorly.

The vastus lateralis fascia is then incised, and the muscle is dissected from the intermuscular septum in order to access the lateral femur. It is important to expose the lateral femur in an extraperiosteal fashion because the fatty tissue that is deep to the vastus muscle is continuous with the pericapsular fat around the hip joint, thus providing a safe and avascular plane for exposure.

The interval between the gluteus minimus and piriformis tendon is used to access the hip joint. If visible, developing the interval can be started prior to trochanteric osteotomy; however, the external rotators and hip capsule are contracted in chronic SCFE making the interval difficult to access from the posterior hip joint.

An osteotomy of the greater trochanter enables efficient anterior retraction of the gluteus medius and vastus lateralis muscles. In order to safely osteotomize the trochanter, the depth and trajectory should be carefully assessed and fluoroscopy used if necessary. In order to protect the medial femoral circumflex, the depth of the osteotomy should not be medial to the gluteus medius tendon attachment to the posterosuperior trochanter. Following the osteotomy, the vastus lateralis fibers that remain attached to the anterior femur and hip capsule can be released, and the interval between the gluteus minimus and piriformis tendon developed to complete capsular exposure.

Once the capsular exposure is completed, the capsulotomy is performed in a specific pattern that avoids lateral capsular incisions that could endanger the retinaculum. The anterolateral capsule is incised parallel to the anterolateral femoral neck. For convenience when developing the retinacular flap, it is helpful to terminate the femoral limb of the anterolateral capsulotomy at the anterior aspect of the trochanteric apophysis. The cephalad limb is parallel to the labrum and may be extended posteriorly by retracting the piriformis muscle and carefully incising the capsule further distal along the ischium. The caudal limb extends from the inferior aspect of the anterolateral capsulotomy parallel to the intertrochanteric line and then diverges in a more cephalad position directed toward the base of the acetabulum.

In order to develop the retinacular flap, the entire upper femur is exposed in a subperiosteal fashion, and beginning this dissection prior to dislocating the hip is safer. The greater trochanter should be dissected posteriorly and superiorly as completely as possible prior to actually dislocating the hip. If the SCFE is chronic, the hip can be safely dislocated; however, if it is acute, temporary stabilization of the epiphysis is required prior to dislocating the hip joint in order to prevent disrupting the retinaculum. The epiphysis is then dislocated by placing a blunt bone hook around the medial femoral neck and applying traction to the ligamentum teres. External rotation and flexion will enable visualization and release of the ligament. After inspecting and addressing any chondral injury to the labrum and acetabulum, several sponges should be placed into the acetabulum in order to prevent inadvertent relocation of the epiphysis.

The next step is to complete the removal of the greater trochanteric apophysis. This is accomplished by osteotomizing through the apophysis parallel to the posterior neck of the femur, mobilizing the trochanter so that it can be removed and the periosteum of the upper femur and neck mobilized with the epiphysis attached. In order to complete developing the retinacular flap, the periosteum of the femoral neck is incised and reflected medially along the neck and upper femur and laterally posterior to the neck of the femur. If the SCFE is chronic and partially healed posteroinferiorly, mobilization of the epiphysis may require careful osteotomy through the healed bone, whereas if the SCFE is acute, mobilization can be accomplished by gently prying the epiphysis from the neck of the femur maintaining continuity of the periosteal cuff that is attached to the epiphysis.

Once the epiphysis has been mobilized on the retinacular flap, the goal is a tension-free reduction. This requires removing all periosteal new bones that formed along the posterior and superior femoral neck as well as any new bone and remaining physeal tissue within the epiphysis. Trial reductions can be performed periodically in order to assess the position of the epiphysis and tension generated in the periosteum. Most surgeons will also attempt to monitor the blood flow within the epiphysis either by drilling a small hole in the epiphysis to observe active bleeding or by using an intracranial pressure measuring probe calibrated to detect intraosseous blood flow.

Fixation of the epiphysis is performed using either antegrade or retrograde smooth or threaded wires, cannulated or solid screws, or their combination. Following fixation, the periosteum should be loosely secured in order to avoid kinking or buckling of the lateral periosteum and the hip reduced into the acetabulum. The capsule of the hip joint is not completely closed as it is usually contracted, and attempts to close it may tamponade the retinacular vessels. The last step is to repair the trochanter, which is positioned carefully, especially when significant neck shortening is necessary for cases of chronic SCFE. Care is exercised to position the proximal tip of the trochanter at approximately the center of the femoral head, usually located at the level of the physis in a skeletally immature patient. Generally, the trochanter is secured using two or three screws.

Postoperative Care

Patients are hospitalized for analgesia and therapeutic crutch training. A toe-touch gait pattern must be maintained until the physis is healed radiographically and sufficient time has passed to permit femoral neck remodeling. Most surgeons wait approximately three months before permitting unprotected weight-bearing.

Summary

The modified Dunn technique is an approach to correcting upper femoral deformity that is associated with SCFE. It is a technically demanding procedure that requires technical expertise and intimate knowledge of the vascular anatomy of the upper femur. The modified Dunn approach is currently the most comprehensive method that enables full correction of the femoral deformity and treatment of labral and chondral injury associated with SCFE. The precise role amongst the variety of approaches that are used to treat both stable and unstable SCFE and qualifications that are necessary to perform the procedure remains to be clarified.

References

1. Zaltz I, Baca G, Clohisy JC. Unstable SCFE: review of treatment modalities and prevalence of osteonecrosis. Clin Orthop Relat Res. 2013;471(7):2192–8.
2. Boyer DW, Mickelson MR, Ponseti IV. Slipped capital femoral epiphysis. Long-term follow-up study of one hundred and twenty-one patients. J Bone Joint Surg Am. 1981;63(1):85–95.
3. Larson AN, Sierra RJ, Yu EM, Trousdale RT, Stans AA. Outcomes of slipped capital femoral epiphysis treated with in situ pinning. J Pediatr Orthop. 2012;32(2):125–30.
4. Castañeda P, Ponce C, Villareal G, Vidal C. The natural history of osteoarthritis after a slipped capital femoral epiphysis/the pistol grip deformity. J Pediatr Orthop. 2013;33 Suppl 1:S76–82.
5. Parsch K, Weller S, Parsch D. Open reduction and smooth Kirschner wire fixation for unstable slipped capital femoral epiphysis. J Pediatr Orthop. 2009;29(1):1–8.

6. Dunn DM. The treatment of adolescent slipping of the upper femoral epiphysis. J Bone Joint Surg Br. 1964;46:621–9.
7. Fish JB. Cuneiform osteotomy of the femoral neck in the treatment of slipped capital femoral epiphysis. J Bone Joint Surg Am. 1984;66(8):1153–68.
8. Ziebarth K, Zilkens C, Spencer S, Leunig M, Ganz R, Kim YJ. Capital realignment for moderate and severe SCFE using a modified Dunn procedure. Clin Orthop Relat Res. 2009;467(3):704–16.
9. Ganz R, Gill TJ, Gautier E, Ganz K, Krugel N, Berlemann U. Surgical dislocation of the adult hip: a technique with full access to the femoral head and acetabulum without the risk of avascular necrosis. J Bone Joint Surg Br. 2001;83(8):1119.
10. Gautier E, Ganz K, Krügel N, Gill T, Ganz R. Anatomy of the medial femoral circumflex artery and its surgical implications. J Bone Joint Surg Br. 2000;82 (5):679–83.
11. Ganz R, Parvizi J, Beck M, Leunig M, Nötzli H, Siebenrock KA. Femoroacetabular impingement: a cause for osteoarthritis of the hip. Clin Orthop Relat Res. 2003;417:112–20.
12. Leunig M, Casillas MM, Hamlet M, Hersche O, Nötzli H, Slongo T, Ganz R. Slipped capital femoral epiphysis: early mechanical damage to the acetabular cartilage by a prominent femoral metaphysis. Acta Orthop Scand. 2000;71(4):370–5.
13. Huber H, Dora C, Ramseier LE, Buck F, Dierauer S. Adolescent slipped capital femoral epiphysis treated by a modified Dunn osteotomy with surgical hip dislocation. J Bone Joint Surg Br. 2011;93(6):833–8.
14. Slongo T, Kakaty D, Krause F, Ziebarth K. Treatment of slipped capital femoral epiphysis with a modified Dunn procedure. J Bone Joint Surg Am. 2010;92 (18):2898–908.
15. Sankar WN, Vanderhave KL, Matheney T, Herrera-Soto JA, Karlen JW. The modified Dunn procedure for unstable slipped capital femoral epiphysis: a multicenter perspective. J Bone Joint Surg Am. 2013;95(7):585–91.
16. Carney BT, Weinstein SL, Carney J. Long-term follow-up of slipped capital femoral epiphysis. J Bone Joint Surg Am. 1991;73-A(5):667–74.
17. Sankar WN, Brighton BK, Kim Y-J, Millis MB. Acetabular morphology in slipped capital femoral epiphysis. J Pediatr Orthop. 2011;31(3):254–8.
18. Leunig M, Slongo T, Kleinschmidt M, Ganz R. Subcapitale Korrekturosteotomie bei der Epiphysiolysis capitis femoris mittels chirurgischer Hüftluxation. Oper Orthop Traumat. 2007;19:389–410.
19. Leunig M, Slongo T, Ganz R. Subcapital realignment in SCFE. Instr Course Lect. 2008;57:499–507.

Surgical Technique: In-Situ Pinning of Unstable Slipped Capital Femoral Epiphysis

35

David A. Podeszwa

Contents

Abstract

An unstable slipped capital femoral epiphysis (SCFE) is one typically associated with prodromal hip pain and/or limp for days or weeks followed an acute event resulting in the patient's inability to bear weight. The patient will present with severe fracture-like pain. An AP pelvic radiograph is the only radiograph needed to diagnose an unstable SCFE, but a lateral radiograph of the contralateral hip is mandatory to screen for a contralateral SCFE if not obvious on the AP radiograph. Unstable SCFE has been associated with a high rate of avascular necrosis (AVN) and slip progression with in situ pinning of the epiphysis. Biomechanical data from animal models suggests that double-screw fixation is superior to single-screw fixation, but the mechanical superiority must be weighed against the potential complications of a second screw. The author's preferred technique is to accept the reduction obtained from positioning the patient on the operative table, perform double-screw fixation of the femoral epiphysis with 6.5 mm fully threaded cannulated screws, and decompress the hip capsule. Clinical data suggests that this technique offers superior results (lower rate of AVN and chondrolysis) compared to previous studies.

D.A. Podeszwa
Department of Orthopaedic Surgery, University of Texas Southwestern Medical Center, Dallas, TX, USA

Texas Scottish Rite Hospital for Children, Dallas, TX, USA
e-mail: david.podeszwa@tsrh.org

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_38

Introduction

An unstable slipped capital femoral epiphysis (SCFE) is relatively rare compared to a stable SCFE and one of the most challenging of all adolescent hip conditions to treat. The femoral epiphysis actually maintains its normal relationship within the acetabulum, and it is the femoral neck and shaft that displace relative to the femoral epiphysis. Most commonly, the metaphysis slips upward and anteriorly and induces a shortening and external rotation of the femur.

Etiology

The exact cause of an unstable SCFE is most likely multifactorial, with mechanical, endocrine, and genetic factors probably involved. There are several mechanical factors unique to the adolescent hip that may predispose or cause them to have a SCFE. The perichondral ring of the proximal femoral physis thins with maturation, thus altering the strength of the physis. The femoral neck of the adolescent with an SCFE has been shown to have relative or absolute retroversion, and the inclination of the proximal femoral physis is more vertical in those with a SCFE which predisposes the hip to increased shear stress across the physis [1–3]. A deep acetabulum may also influence physeal stability; a deep acetabulum has been identified more commonly in an unstable SCFE than a stable one [4]. A study comparing the contralateral hip in patients with a unilateral SCFE to age matched normal hips, the inclination of the physis in the SCFE group was 8.9° more vertical than the control group. Mathematic modeling demonstrated increased shear stress and contact stress in the hips of the SCFE group relative to the normal group, even when normalized for body weight [5].

Hypothyroidism, chronic renal failure (with secondary hyperparathyroidism), and disorders requiring growth hormone replacement are the most common endocrine disorders associated with SCFE [6]. One must have a high index of suspicion for undiagnosed endocrine disorders, especially hypothyroidism, in the very young patient or a patient presenting with severe bilateral SCFE. General anesthesia in the face of a significant hormone imbalance can lead to a catastrophic outcome.

Classification

The classic clinical classification of SCFE is based on the patient's clinical and radiographic evaluation. The SCFE was defined as acute (symptoms <3 weeks with severe displacement), chronic (symptoms >3 weeks, often months or years), and acute-on-chronic. Physeal stability is determined based on the patient's ability to bear weight on the affected lower extremity at the time of presentation to the emergency department or clinic [7]. A patient with an unstable SCFE will not be able to bear any weight with or without crutches. Physeal stability is prognostic of avascular necrosis (AVN). The risk of AVN for a patient with a stable SCFE is nearly 0, while the rate of AVN for a patient with an unstable SCFE is 20–47 % [7–9].

The sensitivity and specificity of the chronologic classification (acute, acute-on-chronic, chronic) and the stable/unstable classification of Loder et al. [7] may limit the ability to predict a truly unstable SCFE [10]. The physis was directly visualized and evaluated in 82 patients with severe SCFE undergoing a modified Dunn procedure (open reduction) through a surgical dislocation approach. The unstable/stable classification preoperatively had poor specificity (39 %) and limited sensitivity (76 %) for identifying a complete physeal disruption. Chronologic classification did not significantly improve the results (specificity 44 %, sensitivity 82 %).

Clinical Presentation

A patient with an unstable SCFE will present severe, sudden-onset pain after a relatively trivial trauma, such as a fall during athletics or a twisting injury after stepping into a hole or off a curb. Most children will have had some prodromal symptoms: hip pain, thigh pain, knee pain, and/or limp for days, weeks, or even months. The patient will prefer to lay supine on a stretcher as any hip range of motion will result in severe, fracture-like pain. The hip is typically held in mild flexion and external rotation. If an unstable SCFE is suspected, the hip should not be manipulated for fear of further displacement of the epiphysis.

Bilateral SCFE can be identified in 20–40 % of all patients, with about half having both hips involved at initial presentation. More than 80 % of patients with unilateral involvement who subsequently develop a contralateral SCFE will do so within 18 months of presentation for treatment of the first hip. A high index of suspicion must be maintained for younger patients and those with endocrine or metabolic abnormalities, as they are at highest risk for developing a subsequent SCFE. However, there are no definitive guidelines for prophylactic pinning of the contralateral hip. The overall modified Oxford bone age and an open triradiate cartilage have been identified as the best predictors of a contralateral SCFE [11–13].

An anteroposterior (AP) pelvic radiograph is the only radiograph needed to identify an unstable SCFE. There is typically marked displacement of the epiphysis relative to a stable SCFE. A lateral radiograph of the effected hip is not recommended so as not to manipulate the hip and risk further displacement of the epiphysis. A lateral radiograph of the contralateral hip, either preoperatively or intraoperatively, is required to evaluate for a contralateral SCFE. There is no current role for preoperative CT scan, MRI, or ultrasound of the hip.

Treatment

The gold standard surgical treatment for an unstable SCFE is in situ pinning using single- or double-screw fixation, accepting the reduction achieved with positioning of the patient. The reduction achieved can be quite variable (Figs. 1 and 2). Single-screw fixation has been associated with postoperative slip progression [14], while double-screw use may have an increased risk of screw penetration and chondrolysis.

Early studies of epiphyseal fixation in a bovine shear model demonstrated double-screw fixation to be stiffer than single-screw fixation, but there was no difference when tested under physiologic cyclical loading (no torsional testing performed). As a result, the authors recommended single-screw fixation as the risk of intra-articular placement of the second screw outweighed the mechanical benefit [15, 16]. Subsequent torsional testing in a nonreduced, immature bovine model demonstrated that double-screw fixation resulted in a 25 % increase in axial stiffness when tested under shear and a 312 % increase in torsional stiffness when compared to single-screw fixation [17]. In an immature porcine model of mild to moderate unstable SCFE, single versus double 7.3 mm (16 mm thread length) cannulated screws were tested with a posterior-inferior directed force instead of a pure shear load. Double-screw constructs were 66 % stiffer and 66 % stronger when compared to single-screw fixation. Therefore, a double-screw construct for an unstable SCFE is biomechanically superior to a single-screw construct, but this benefit must be weighed against the potential complications of a second screw, i.e., intra-articular penetration/chondrolysis [18].

Capsular decompression has been recognized as an important step when performing an in situ fixation of an unstable SCFE. Herrera-Soto et al. [19] demonstrated a significantly elevated intracapsular pressure in the effected hip when compare to the normal hip. In addition, there was a further significant increase in pressure after attempted manipulation. Given the risk of the increased intracapsular pressure causing a tamponade effect on the retinacular vessels and causing AVN, a capsular decompression is recommended.

There are multiple decompression techniques that can be utilized. An 18 gauge spinal needle can be placed percutaneously anterolaterally through the proximal thigh into the hip capsule under C-arm guidance. If there is a large traumatic effusion, this can be aspirated to decrease the intra-articular pressure and protect the posterior superior retinacular vessels from intra-articular compression. Alternatively, a large hemostat can be placed through one of the incisions created for screw placement and placed onto the femoral neck and

Fig. 1 (**a**) AP radiograph left hip demonstrating a severe unstable SCFE. (**b**) Frog lateral radiograph right hip confirming no contralateral SCFE. Note that the left hip was not manipulated. (**c**) Intraoperative image demonstrating incomplete reduction after positioning patient. (**d**) AP radiograph left hip (**e**) Frog lateral radiograph left hip following in situ fixation with two 6.5 mm fully threaded cannulated screws. Note the residual proximal femoral deformity but no AVN

through the capsule, guided by fluoroscopic imaging. The capsule can then be opened and decompressed. With this method, it is difficult to estimate how much fluid is decompressed from the hip. Another alternative is a formal Smith-Peterson anterior approach to the hip for a capsulotomy and decompression under direct visualization.

Author's Preferred Operative Technique

The patient is brought into the operating room and general anesthesia is induced while the patient is on the stretcher. The patient is not moved to the operating table prior to the induction of the

Fig. 2 (**a**) AP radiograph left hip demonstrating severe unstable SCFE. (**b**) AP pelvic radiograph (**c**) Frog lateral radiograph left hip. Note near anatomic alignment with no AVN 7 months following positional reduction and in situ pinning

anesthesia to minimize the patient's discomfort. Once asleep, the ipsilateral foot and ankle are well padded. The patient is then gently transferred to the fracture table. The fracture table is selected so that the affected hip can be maintained still during the procedure while moving the image intensifier for the AP and lateral views of the hip. Pinning the hip on a flattop table would require increased manipulation of the hip, which could be detrimental to the position of the epiphysis and potentially to its blood supply. The affected foot and ankle are placed into the traction boot of the fracture table, but no formal manipulation is formed. The contralateral hip is flexed and abducted and the leg placed on a well-padded leg holder. The effected lower extremity is positioned so the knee cap is pointed toward the ceiling. The image intensifier is then used in the AP and lateral views to confirm that appropriate imaging can be achieved. Any reduction achieved while positioning the patient is accepted, but no further reduction maneuver is performed. The patient is then prepped and draped in the usual sterile fashion.

Under fluoroscopic guidance, a 2.8 mm threaded guide pin is placed percutaneously into the center of the femoral epiphysis on the AP and lateral view of the hip. Commonly, the unstable SCFE has severe posterior displacement, and placing the guide pin in the center of the femoral head would require placing the pin quite anterior on the femoral neck (Fig. 1c). This can result in postsurgical screw impingement [20]. In this case, the guide pin is started more laterally on the femoral neck with the goal of creating the starting point at or lateral to the intertrochanteric line on the AP image. When planning the exact placement of the pin, thought should be given to placement of a second guide pin and a second screw. Two 6.5 mm fully threaded, cannulated screws are recommended. When compared to partially threaded screws, the fully threaded screws have superior purchase in the metaphyseal bone of the proximal femur and are easier to remove if needed. Once fluoroscopy has confirmed appropriate placement of the first guide pin in the center of the head, a second guide pin can be placed, most commonly in the posterior-inferior aspect of the femoral epiphysis. Placement of the second screw anteriorly may make the screw prone to cutting out of the anterior neck because of the limited metaphyseal bone anteriorly, and the screw may be more likely to become intra-articular if there is collapse of the anterior epiphysis with the onset of AVN. After both guide pins are placed in the appropriate position and confirmed under fluoroscopy in AP and lateral views, a 1 cm incision is made around each guide pin. Blunt dissection is taken down to bone along the guide pins. The appropriate screw lengths are measured, and a 5.0 mm cannulated drill is used to over-drill the guide pin up to, but not across, the physis. The appropriate length screw is placed by hand over the guide pin into the epiphysis. The goal is to have at least four threads of the screw across the physis into the epiphysis. The second guide pin is then over-drilled, and the second screw is placed in a similar fashion. Once both screws are secured, fluoroscopic images are obtained through a full arc of motion, from a true lateral to a true AP of the hip, confirming that neither screw is encroaching the subchondral bone of the epiphysis. Because of the residual malposition of the femoral epiphysis, it is frequently helpful to rotate the image intensifier beyond a true AP angle so that the femoral epiphysis can be imaged in an orthogonal manner.

Capsular decompression is then performed by placing a large hemostat through one of the incisions into the capsule under fluoroscopic guidance. Once the hip is decompressed, the guide pins can be safely removed. The wounds are copiously irrigated with normal saline. The deep tissues are closed with a 2-0 Vicryl suture and the skin approximated with a 2-0 Monocryl suture in a subcuticular fashion. Bupivacaine (0.25 %) is injected around the incision for local anesthetic. The incisions are covered with a gauze dressing that can be removed in 48 h.

If the contralateral hip requires in situ pinning, the contralateral foot and ankle are then padded and placed into the traction boot of the fracture table. The index hip is gently abducted, flexed, and placed onto a padded well-leg holder. Care should be taken to avoid maximal hip flexion and/or hip external rotation to avoid stressing the screw fixation of the hip and potentially impinging the retinacular vessels of the femoral epiphysis.

Postoperatively, the patient remains toe-touch weight bearing on the affected side (full weight bearing on the contralateral side) for 3 months while performing self-guided hip range of motion exercises. If there is any sign of AVN at 3 months, the patient will remain toe-touch weight bearing. If no evidence of AVN, the patient will be full weight bearing but not allowed to participate in running/jumping or sporting activities until 6 months from surgery and radiographs confirm no evidence of AVN.

Double-screw in situ fixation after positional reduction may prove to be superior to previous reports of in situ fixation for unstable SCFE. Chen et al. [21] reported 28 consecutive patients with 30 unstable SCFE (per the Loder et al. criteria) [7] who underwent urgent reduction (generally within 12–24 h of the onset of acute symptoms) and fixation with two 6.5 mm cannulated screws. Slip severity was mild in 13 patients, moderate in 9, and severe in 8. Positional reduction was accepted in 25 cases, while 5 cases underwent an open arthrotomy with reduction to the “pre-acute position” under direct visualization. A percutaneous arthrotomy was performed in 16 hips in addition to those performed as part of the open arthrotomy [21].

At short-term follow-up (average of 5.5 years, range 2–11.2 years), four patients (13 %) had radiographic findings consistent with AVN, and three of these patients required at least one additional surgery. All patients with AVN had undergone in situ pinning. One patient experienced slip progression, and no patient developed chondrolysis.

Two additional patients (7 %) reported mild pain with prolonged sitting, while six patients (20 %) reported a mild limp and two (7 %) reported a moderate limp. There was no statistical association between the development of AVN and age, duration of prodromal symptoms, time to reduction, severity of slip, presence of an arthrotomy, or noncompliance with weight bearing.

Summary

An unstable SCFE is a challenging condition to treat and is fraught with potential complications. Despite being the gold standard treatment for an unstable SCFE, severe residual displacement, limited hip range of motion, chondrolysis, and AVN can result from in situ pinning and cause long-term pain and dysfunction. Biomechanical testing and clinical data suggest that double-screw in situ fixation after positional reduction may result in lower rates of chondrolysis and AVN than previously reported in the literature.

References

1. Chung SM, Batterman SC, Brighton CT. Shear strength of the human femoral capital epiphyseal plate. J Bone Joint Surg Am. 1976;58(1):94–103.
2. Gelberman RH, et al. The association of femoral retroversion with slipped capital femoral epiphysis. J Bone Joint Surg Am. 1986;68(7):1000–7.
3. Mirkopulos N, Weiner DS, Askew M. The evolving slope of the proximal femoral growth plate relationship to slipped capital femoral epiphysis. J Pediatr Orthop. 1988;8(3):268–73.
4. Podeszwa DA, et al. Increased acetabular depth may influence physeal stability in slipped capital femoral epiphysis. Clin Orthop Relat Res. 2013;471 (7):2151–5.
5. Zupanc O, et al. Shear stress in epiphyseal growth plate is a risk factor for slipped capital femoral epiphysis. J Pediatr Orthop. 2008;28(4):444–51.
6. Loder RT, Wittenberg B, DeSilva G. Slipped capital femoral epiphysis associated with endocrine disorders. J Pediatr Orthop. 1995;15(3):349–56.
7. Loder RT, et al. Acute slipped capital femoral epiphysis: the importance of physeal stability. J Bone Joint Surg Am. 1993;75(8):1134–40.
8. Loder RT. What is the cause of avascular necrosis in unstable slipped capital femoral epiphysis and what can be done to lower the rate? J Pediatr Orthop. 2013;33 Suppl 1:S88–91.
9. Sankar WN, et al. The unstable slipped capital femoral epiphysis: risk factors for osteonecrosis. J Pediatr Orthop. 2010;30(6):544–8.
10. Ziebarth K, et al. Clinical stability of slipped capital femoral epiphysis does not correlate with intraoperative stability. Clin Orthop Relat Res. 2012;470(8):2274–9.
11. Stasikelis PJ, et al. Slipped capital femoral epiphysis. Prediction of contralateral involvement. J Bone Joint Surg. 1996;78(8 (Print)):1149–55.
12. Zide JR, Popejoy D, Birch JG. Revised modified Oxford bone score: a simpler system for prediction of contralateral involvement in slipped capital femoral epiphysis. J Pediatr Orthop. 2011;31(2):159–64.
13. Popejoy D, Emara K, Birch J. Prediction of contralateral slipped capital femoral epiphysis using the modified Oxford bone age score. J Pediatr Orthop. 2012;32 (3):290–4.
14. Sanders JO, et al. Progressive slippage after pinning for slipped capital femoral epiphysis. J Pediatr Orthop. 2002;22(2):239–43.
15. Karol LA, et al. Single versus double screw fixation for treatment of slipped capital femoral epiphysis: a biomechanical analysis. J Pediatr Orthop. 1992;12(6):741–5.
16. Kibiloski LJ, et al. Biomechanical analysis of single- versus double-screw fixation in slipped capital femoral epiphysis at physiological load levels. J Pediatr Orthop. 1994;14(5):627–30.
17. Segal LS, Jacobson JA, Saunders MM. Biomechanical analysis of in situ single versus double screw fixation in a nonreduced slipped capital femoral epiphysis model. J Pediatr Orthop. 2006;26(4):479–85.
18. Kishan S, et al. Biomechanical stability of single-screw versus two-screw fixation of an unstable slipped capital femoral epiphysis model: effect of screw position in the femoral neck. J Pediatr Orthop. 2006;26(5):601–5.
19. Herrera-Soto JA, et al. Increased intracapsular pressures after unstable slipped capital femoral epiphysis. J Pediatr Orthop. 2008;28(7):723–8.
20. Goodwin RC, et al. Screw head impingement after in situ fixation in moderate and severe slipped capital femoral epiphysis. J Pediatr Orthop. 2007;27 (3):319–25.
21. Chen RC, et al. Urgent reduction, fixation, and arthrotomy for unstable slipped capital femoral epiphysis. J Pediatr Orthop. 2009;29(7):687–94.

Surgical Technique: Proximal Femoral Osteotomies in Residual Childhood Disease

36

Brian T. Barlow, Bernd Bittersohl, Matthew Schmitz, and Harish S. Hosalkar

Contents

B.T. Barlow (✉)
Orthopaedic Chief Resident, Naval Medical Center, San Diego, USA
e-mail: brianbarlow05@gmail.com

B. Bittersohl
Faculty, Orthopedic Department, University Hospital Dusseldorf, Dusseldorf, Germany

M. Schmitz
San Antonio Military Medical Center, Houston, TX, USA
e-mail: mattrschmitz@gmail.com

H.S. Hosalkar
Sharp Grossmont Hospital, La Mesa, Paradise Valley Hospital, National City, Vista, CA, USA
e-mail: hhpedpod@gmail.com

Abstract

The proximal femoral osteotomy is an instrumental tool for hip deformity correction. The goal of a proximal femoral osteotomy is to return the patients' abnormal morphology to an anatomic alignment of the proximal femur. Proximal femoral osteotomy has also been used to change the otherwise normal proximal femoral anatomy in such a way to positively benefit the hip joint through improved mechanics as well as correct global femoral malrotation.

Proximal femoral osteotomy has been used to address problems such as severe slipped capital femoral epiphysis disease, Legg-Calvé-Perthes disease, developmental dysplasia of the hip, congenital malrotation, and posttraumatic malunion of the proximal femur.

The decision to operate with a proximal femoral osteotomy is driven by patient symptoms in conjunction with altered proximal femoral anatomy and alignment. Plain radiographs and long leg alignment imaging are the key diagnostic imaging techniques when planning a proximal femoral osteotomy. The indications for surgical intervention are poorly defined by previous literature and require a complete assessment of the patient's symptoms and diagnostic imaging.

Recently there has been renewed interest and development of new techniques of proximal femoral osteotomy to more directly address pathoanatomy. Focused research on the vascular supply to the femoral head has provided the opportunity to directly treat hip deformity that

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_39

previously would have been left to natural history and inevitable coxarthrosis [1]. While these new techniques have engendered considerable interest, the long-term outcomes are not available at this point. The techniques described in the following chapter are among the most technically challenging in all of orthopedic surgery.

Introduction

Since its original introduction under the title *Orthopédie* in 1741 by Nicolas Andry, orthopedics as a subspecialty continues to focus on the art and science of identifying, assessing, and planning deformity corrections [2]. Proximal femoral osteotomy is a powerful tool available for correcting deformities around the hip joint.

In the last decade, orthopedists have furthered our understanding related to the vascularity, mechanics, and imaging of the hip joint, leading to bolder surgical interventions that may be challenging to many surgeons. This has provided the opportunity to address hip deformity that previously would have been left to natural history and inevitable early arthritis. While these new techniques have engendered considerable interest, long-term outcomes demonstrating benefit are not available at this point. It is important to understand that hip joint deformities may not be isolated to the proximal femur and may additionally include intra- and periarticular deformities including impingement, acetabular dysplasia, fixed pelvic deformities, and occasionally suprapelvic deformities as well. Opposite limb involvement in the form of limb length discrepancy may play a significant role in ipsilateral hip problems.

The focus of this section is limited to specific osteotomies around the proximal femur only. Within the spectrum of proximal femoral deformity correction, the focus will be on the technique, indications, and outcomes of femoral head reduction osteotomy, intertrochanteric osteotomy, and subtrochanteric rotational osteotomy.

Femoral Head Reduction Osteotomy

Certain conditions such as Legg-Calvé-Perthes (LCP) disease can lead to extreme deformation of the femoral head and subsequent femoroacetabular incongruity [3] (Fig. 1). Not only is this symptomatic in terms of pain, but it also limits range of

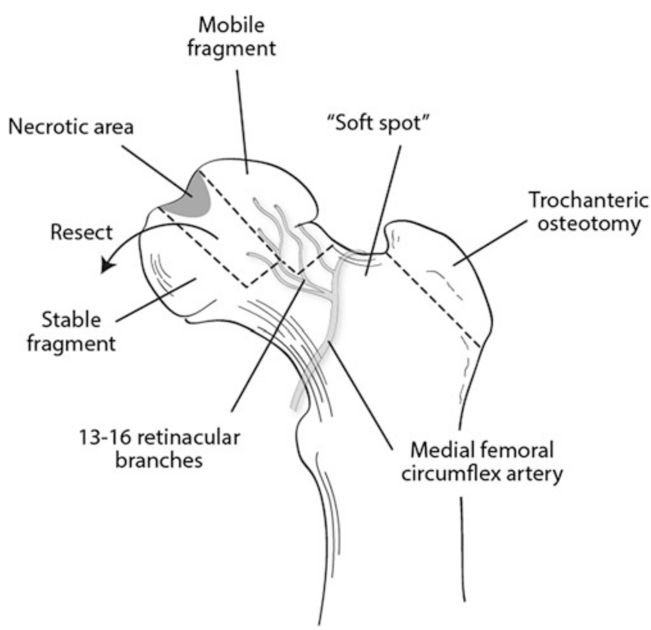


Fig. 1 Typical deformity of severe Perthes disease with a mushroom deformity and an area of central necrosis. Frequently, the deformity in the medial to lateral plane is greater than the deformity in the anterior to posterior plane. The planned osteotomies are shown by the *dotted lines*. The blood supply to the mobile fragment is maintained by retinacular terminal branches of the medial femoral circumflex artery

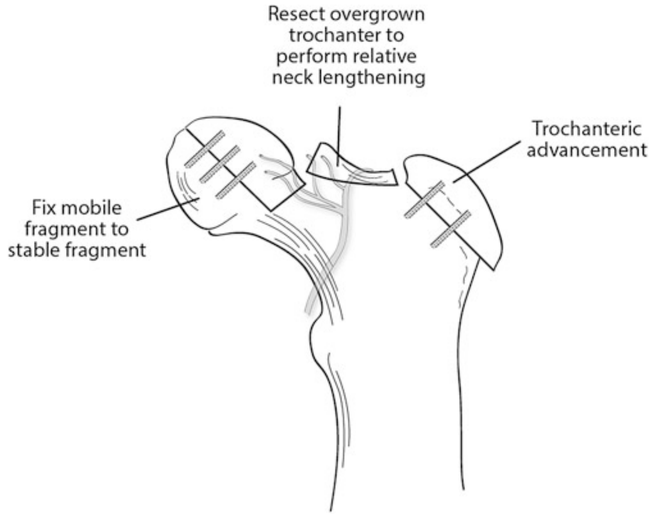


Fig. 2 The final construct after completion of the femoral head reduction osteotomy. The mobile and stable fragments are joined with headless compression screws. The trochanter is advanced distally and a relative neck lengthening is achieved by resecting excess bone to recontour the superior femoral neck while carefully preserving the blood supply in the retinacular flap

motion and function and creates an environment for rapid progression of degenerative arthritis. The notably young age of these patients (most often skeletally immature) makes them poor candidates for any form of arthroplasty.

The following section describes the technique for femoral head reduction osteotomy in a skeletally immature patient with LCPD (Fig. 2). There is a tremendous learning curve for such a procedure and the authors advise against performing this procedure for surgeons early in their practice of hip surgery.

Patient Selection

Patient selection is extremely important for this osteotomy and compliance is important as well.

Indications

- Healed LCPD hip. This procedure should not be performed in active stage of the disease.
- Central head necrosis with well-maintained articular cartilage over the rest of the femoral head. Oblong femoral head with greater medial-lateral diameter than anteroposterior diameter is ideal for this procedure.
- Acetabular cartilage damage must be absent or minimal for optimal results.
- Full strength should be present in major muscle groups about the hip. The procedure is not a good option for patients with neuromuscular disease conditions that cause weakness.
- Patient is toe-touch weight bearing for about 10–12 weeks and compliance is paramount and should be discussed preoperatively with the patient and caregivers.

Relative Contraindications

- Advanced articular degeneration of the femoral head or acetabulum
- Extremely misshapen femoral head not amenable to any symmetric head reduction osteotomy
- Noncompliant patient
- Extreme obesity
- Neuromuscular disease etiology for hip instability, subluxation, and deformity

Preoperative Imaging

- Standardized pelvis radiographs including AP and either Frog or Dunn lateral views (Figs. 3 and 4)
- Full-length standing alignment radiographs of the lower extremities
- MR arthrogram with radial neck sequencing to study the cartilage and labral status in detail, as well as to rule out changes of AVN preoperatively (Fig. 5)
- CT with 3D reconstruction to analyze the complete nature of the femoral head shape for purposes of planning of the osteotomy

Fig. 3 Legg-Calvé-Perthes disease of the right hip in a 13-year-old male. Lateral pillar classification C and has several "head at risk" signs including lateral calcification, Gage's sign, and head extrusion

Fig. 4 Frog lateral radiograph of LCP hip with mushroom deformity

Fig. 5 Coronal MR image of LCP hip demonstrating maintained vascularity to medial portion of epiphysis

Procedure

Room and Equipment Setup

- Neuromonitoring is preferred for this operation since the hip stays dislocated for a longer period in this case as compared to a safe surgical dislocation for a routine case of FAI. The leads on the opposite leg are placed prior to prepping and on the operative leg are placed in a sterile manner.
- Fluoroscopic examination may be valuable in the placement of the trochanteric fixation at the conclusion of the procedure, but is not required as the remainder of the procedure is completed under direct visualization.
- In cases when the procedure is being performed with a concomitant acetabular osteotomy, a cell-saver may be valuable.

Patient Positioning

- Patient is positioned in a lateral decubitus position. Additionally, the procedure can be carried out on a regular table or radiolucent flat-top table. The use of a radiolucent flat-top table offers the advantage of easier and faster setup, ease of imaging, and the potential for multiple

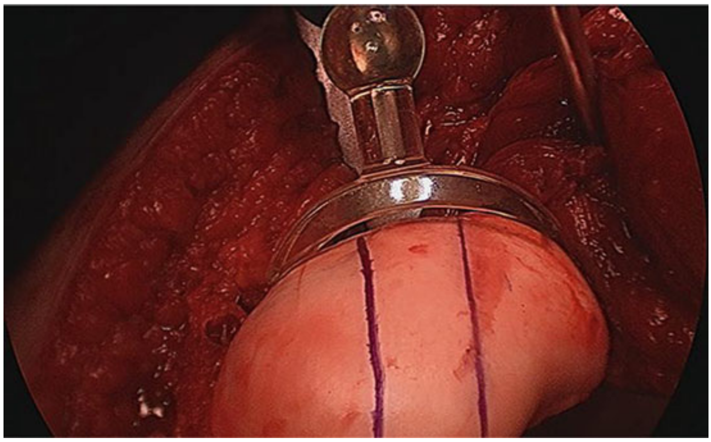


Fig. 6 Intraoperative photograph of femoral head sizing and planning for femoral head reduction osteotomy (FHRO)

surgeries to be performed simultaneously (e.g., concomitant proximal femoral osteotomy and acetabular osteotomy in complex cases).

- Standard hip positioners for lateral positioning are preferred over a beanbag to allow free manipulation of the operative limb. Commercial hip positioners more securely stabilize the pelvis than a beanbag in preparation for surgical hip dislocation.
- The entire limb is then prepped in a standard fashion from the iliac crest to the toes. Neuromonitoring wires are placed in a sterile manner and baseline readings are obtained.
- A 15 cm incision is obtained centered over the trochanter and curving a bit anteriorly in the proximal part. After superficial dissection, the tensor fascia lata is split in the line of the mid-trochanter (distal to trochanteric tip) and along the mid-femur in the distal part of the incision. Proximally it is split in line with the zone between tensor fascia lata and gluteus maximus fascia.
- The anterior and posterior margins of the trochanter are palpated and care is taken to protect the posterior vessels. A trochanteric flip osteotomy is now performed taking care to keep the gluteus medius, gluteus minimus, and vastus lateralis attached to the greater trochanteric fragment. Gluteus minimus tendon is released from its insertion over the trochanter and the trochanteric osteotomy is flipped anteriorly with the digastric attachment.
- A “z” capsulotomy (right hip) is now performed as the standard technique of safe hip dislocation and the femoral head is dislocated anterosuperiorly with adduction and external rotation. The ligamentum teres (which is almost always hypertrophied in these cases) is divided to facilitate a gentle dislocation.
- The femoral head is carefully examined and using the head-sizing jigs, the central femoral osteotomy is outlined (Fig. 6).
- The “soft spot” with the retinacular vessels is carefully protected in the superior fragment. The central fragment with the necrotic area of femoral head, in the saddle-shaped area, is now completely osteotomized with two osteotomies made parallel to the neck (Fig. 7).
- The deformed femoral head is usually saddle shaped on the surface and an ellipse in its circumference with the medial-lateral dimension larger than the anteroposterior dimension. Determination of the correct wedge resection is a combination of geometric resection and direct visualization. The goal is to restore the long-axis measure to as close to the shorter anteroposterior width (with a 10 % maximum difference between the medial-lateral and anteroposterior widths). For example, if the width is 5.5 cm and the long axis is 7.5 cm, the wedge should be at least 1.5 cm to bring the length to 6 cm and have an approximate 10 % maximum difference. In our experience, all osteotomy

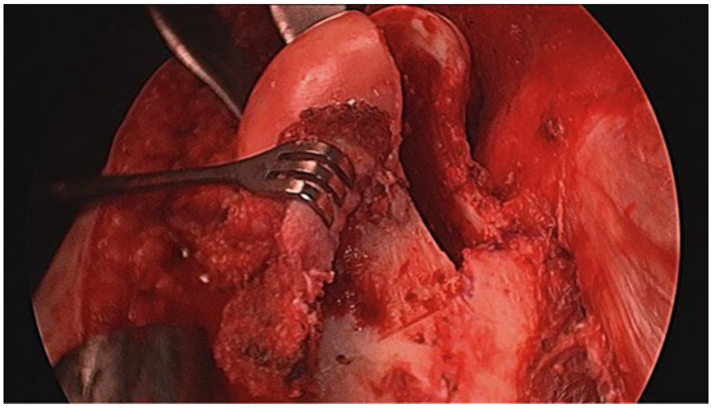


Fig. 7 Clinical photograph of FHRO with superior retinacular flap maintaining perfusion to the mobile, superior segment

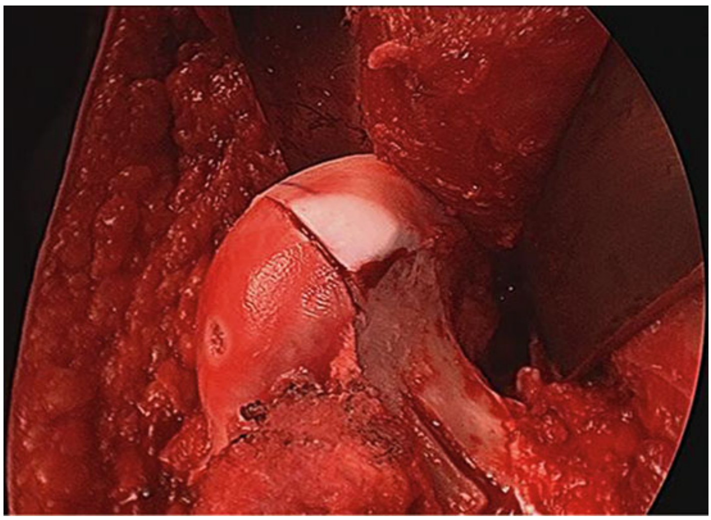


Fig. 8 Clinical photograph of FHRO following resection of central portion and matching of the superior mobile fragment and the inferior stable fragment to create a spherical femoral head

resections have included the fovea or the attachment of the ligamentum teres.

- The intraoperative templates (head sizers) are invaluable in determining the sphericity as determination of the adequacy of the resection by direct visualization obviously comes with experience.
- The initial superior osteotomy is made with an oscillating saw and finished with an osteotome to prevent damage to the posterior retinacular vessels. This creates the superior mobile fragment with blood supplied by the posterior retinacular vessels [4].
- The second osteotomy is inferior to the initial cut and is made in similar fashion to above. This creates the stable fragment which is still in continuity with the metaphysis and blood supply is provided by a constant posteromedial branch of the medial femoral circumflex artery that travels in the ligament of Weitbrecht [4].
- The superior (mobile) fragment with the attached retinacular blood vessels is now approximated to the inferior (stable) fragment and contour matched. Three or four headless cannulated screws are placed to achieve compression. The overhang (should be minimal) is carefully shaved off (Fig. 8).
- Femoral head-neck osteochondroplasty is performed in the anterior zone as necessary (between 12 and 6 o' clock).
- Acetabulum is inspected and labral pathology is treated, if present. Concomitant acetabular

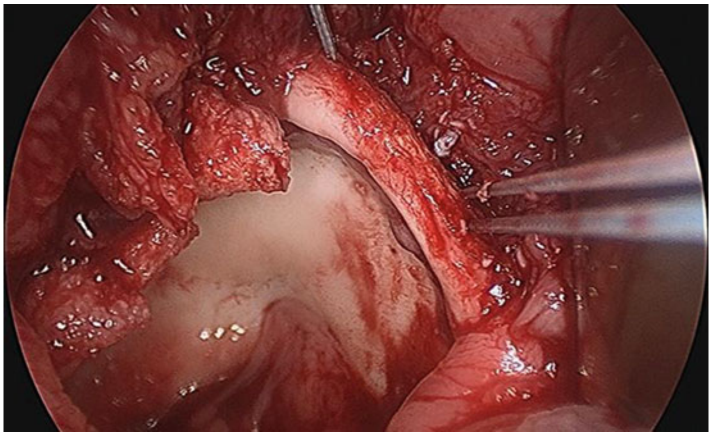


Fig. 9 Clinical photograph of acetabular rim resection and labral reattachment with suture anchors following FHRO

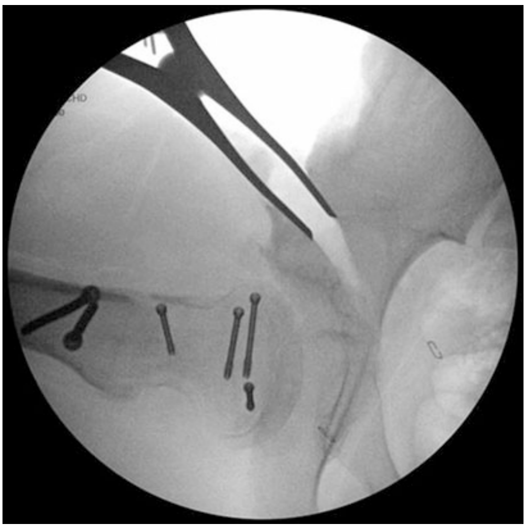


Fig. 10 Intraoperative fluoroscopy demonstrated inadequate acetabular coverage following FHRO. The acetabulum had remodeled to the deformed, mushroom-shaped LCP head and was too capacious following FHRO. A Pemberton pelvic osteotomy was performed to improve congruity of the hip. The authors recommend a volume reducing pelvic osteotomy when combined with a FHRO

Fig. 11 Intraoperative fluoroscopy demonstrating improved lateral hip coverage following Pemberton osteotomy

osteotomy, if required, is performed at this point (Figs. 9, 10, and 11).

- Relative neck lengthening is performed by appropriately distalizing and lateralizing the greater trochanteric fragment to restore the anatomy and proper biomechanics. LCP hips frequently have high trochanters and need to have the trochanter distalized such that the tip of the trochanter is equal with the approximate center of the femoral head. The trochanteric fragment is fixed using two screws (3.5 cortical fully threaded). Wound is closed in layers.
- After sterile dressings and a compression wrap are applied, the limb is placed in a knee immobilizer and a small abduction pillow is placed between the legs. Cast immobilization is discouraged for these cases (patients are typically above 12 years of age). Final AP pelvis and lateral radiographs of the affected hip are obtained in the OR prior to transporting the patient to PACU (Figs. 12 and 13).

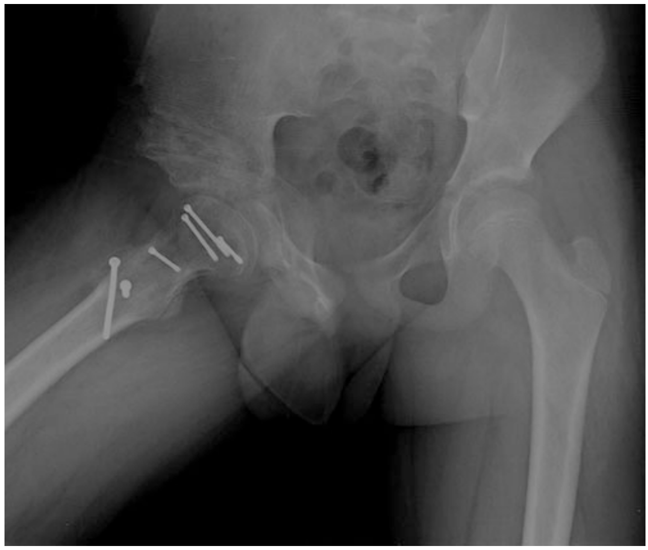


Fig. 12 Early postoperative radiographs. The FHRO was stabilized with four subchondral compression screws

Fig. 13 Early postoperative radiograph. The trochanteric slide osteotomy was secured with two 4.5 mm cortical screws

Complications

- Complete AVN of femoral head has been reported in less than 5 % in experienced centers [4–7].
- Sectoral avascular necrosis of the femoral head related to damage during dissection or moving the superior fragment.
- Nerve palsy due to prolonged position of dislocation.
- Distraction at the osteotomy leading to delayed union or nonunion.

The femoral head reduction osteotomy requires a surgical hip dislocation that has its own specific complication profile which may include:

- Osteonecrosis
- Trochanteric nonunion
- Infection/osteomyelitis
- Damage to trochanteric physis symptoms/painful hardware
- Chondrolysis

Postoperative Care and Rehabilitation

- Knee immobilizer for 2 weeks to limit hip flexion from 0 to 40° to limit stress against the capsular repair. Excessive flexion of the hip joint is nearly impossible in the early postoperative period with the knee in extension.
- Toe-touch weight bearing for 10–12 weeks with crutches for assistance.
- Progressive advancement of weight bearing for the next 4 weeks, followed by weight bearing as tolerated.
- First post-op visit at 2 weeks to assess wound healing, followed by visit at 6 weeks (with radiographs), and then at 3 months (Figs. 14 and 15).

Rehabilitation

- Isometric quads and ankle pump exercises starting POD #0
- Knee ROM exercises (with extended hip) starting after 3 weeks
- Quadriceps strengthening starting at 4 weeks
- Hip abductor strengthening to start at 6 weeks
- Hamstring and IT band stretching to begin when patient is FWB

Fig. 14 AP hip radiograph 30 months following FHRO and Pemberton pelvic osteotomy demonstrating healed osteotomy with no evidence of avascular necrosis. Patient had returned to sporting activities and underwent trochanteric screw removal due to symptomatic bursitis

Outcomes

Leunig and Ganz reported their results of femoral head reduction osteotomy (FHRO) and relative neck lengthening for severe Legg-Calvé-Perthes (LCP) disease in 14 patients with a minimum follow-up of 3 years. Eight patients had a concomitant periacetabular osteotomy (PAO) procedure. There were no major complications to include osteonecrosis or implant failure. All patients reported improved range of motion and decreased pain; but no validated outcome measurement was obtained [7].

Paley reported on his series of 21 patients who underwent FHRO secondary to a misshapen femoral head from LCP disease, adolescent avascular necrosis (AVN), and hip dysplasia who were followed for a mean of 2.7 years. The average age at surgery was 14 years and indications included pain, positive Trendelenburg sign and gait, and reduced range of motion. All patients achieved union, although one patient sustained a femoral neck fracture during surgery requiring conversion to total hip arthroplasty. Three additional patients have gone on to conversion to total hip arthroplasty. An additional patient underwent osteonecrosis at 18 months post-FHRO. Five patients had residual instability requiring an articulated external fixator for 6 weeks without recurrent hip instability. Overall 66 % (14/21) patients achieved good to excellent outcomes [4].

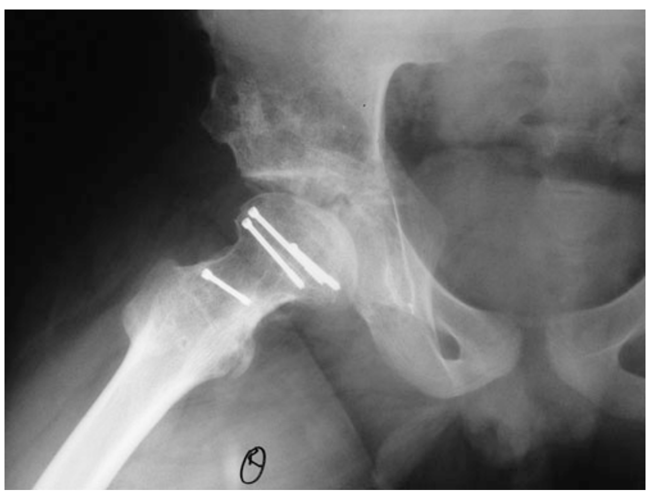


Fig. 15 Lateral hip radiograph demonstrating maintained joint space, contained hip, and well-healed pelvic osteotomy

A recent series by Burien et al. from the Czech Republic describes the use of an anteromedial wedge reduction osteotomy for the treatment of nonspherical femoral head in seven patients with late LCP or multiple epiphyseal dysplasia. The indications were decreased abduction and limp with saddle deformity and extrusion seen on radiographs (all Stulberg class V hips). At a mean follow-up of 17 months, the Harris hip score had improved significantly (55.4 pre-op to 84.8 post-op). No patients sustained osteonecrosis and all improved in their radiographic Stulberg classification [8].

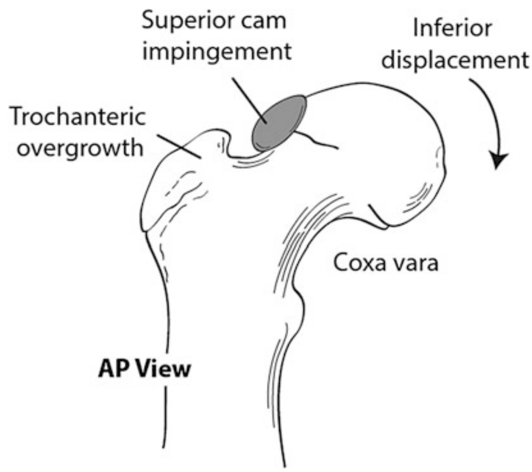


Fig. 16 Anterior view of the typical femoral deformity in the healed slipped capital femoral epiphysis (SCFE). The femoral metaphysis displaces superiorly and anteriorly which produces a relative displacement of the epiphysis in a medial, inferior, and posterior direction. This produces the combined deformity of coxa vara and a cam lesion that is centered anterosuperiorly

Proximal Femoral Intertrochanteric Osteotomy for Chronic Healed SCFE

Intertrochanteric osteotomies to treat deformities of the proximal femur have been a workhorse operation for the hip surgeon for many decades. For this reason, the long-term outcomes of these procedures are well described in the literature. Advantages of the intertrochanteric osteotomy included reliable healing and low risk of osteonecrosis. One of the disadvantages of the intertrochanteric osteotomy is that it does not address the pathoanatomy directly; therefore, the correction may be incomplete and may also introduce a new deformity to the proximal femur that can complicate revision or conversion to a total hip arthroplasty if required. The FHRO osteotomy for LCP disease or the modified Dunn procedure for severe SCFE can both be easily converted to a THA if avascular necrosis or end-stage degenerative changes develop. Additionally, intertrochanteric osteotomy is not immune from osteonecrosis of the femoral head.

Chronic healed slipped capital femoral epiphysis leads to a complex three-dimensional deformity of the proximal femur [10]. The main deforming components are posterior, medial, and inferior displacement of the femoral head in relation to the femoral neck, relatively short femoral neck, relative coxa vara, trochanteric overriding, and excessive intorsion of the femur (Figs. 16 and 17). There are components of femoroacetabular impingement (FAI) that need to be identified and addressed as well [11].

Many hip surgeons recognize the healed SCFE as an impingement lesion and address both impingement and deformity in the same setting. The following section describes a technique for corrective proximal femoral osteotomy while addressing femoroacetabular impingement in a case of chronic healed SCFE.

Very good indications for intertrochanteric osteotomy in the young patient include congenital coxa vara, limited avascular necrosis of the femoral head, femoral neck fracture nonunion, and limb length discrepancy up to 2.5 cm in length. Good indications for intertrochanteric osteotomy include coxa valga with subluxation, chronic SCFE, excessive version in a child, Perthes disease, spastic cerebral palsy, femoral neck fracture, and abduction deficiency after healed acetabular fracture or posttraumatic avascular necrosis. A salvage procedure for osteoarthritis in a young patient is a questionable indication for intertrochanteric [12].

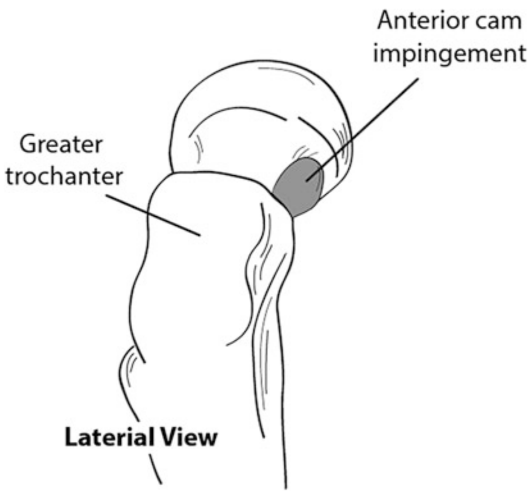


Fig. 17 Lateral view of the typical femoral deformity in the healed slipped capital femoral epiphysis (SCFE). The anterosuperior cam lesion is the product of the healing callus after separation of the epiphysis and metaphysis. The femoral metaphysis displaces anteriorly which produces the relative posterior displacement of the epiphysis

Patient Selection

Indications

- Healed SCFE hip. This procedure should not be performed in acute or unstable slipped epiphysis. The authors' algorithm for acute SCFE is outlined in a previous publication [13].
- Coxa vara.
- Partial osteonecrosis of the femoral head.
- The surgeon should assess the articular cartilage over the femoral head and acetabulum as minimal damage is optimal for results.
- Patient is toe-touch weight bearing for about 4–6 weeks and compliance is important.
- Other indications for general intertrochanteric osteotomies include coxa valga, coxa vara, avascular necrosis of the femoral head, femoral neck fracture nonunion or malunion, limb length discrepancy, cerebral palsy, version deformity, Perthes disease, and as treatment for osteoarthritis [12]. However, the technique described below is relatively specific for healed SCFE and has only been used by the senior author for this indication.

Relative Contraindications

- Advanced degenerative articular changes of the femoral head and/or acetabulum
- Noncooperative patient
- Extreme obesity
- Neuromuscular involvement leading to instability and subluxation

Preoperative Imaging

- Standardized AP radiograph of the pelvis and lateral including Frog lateral or Dunn lateral.
- Full-length standing alignment radiographs of the lower extremities.
- MR arthrogram with radial neck sequence imaging to study the cartilage and labral status in detail, as well as to rule out changes of AVN preoperatively. MR torsional profile may be obtained in the same setting if possible [14].
- CT with 3D reconstruction to analyze the complete nature of the proximal femoral deformity and torsional profile for purposes of planning of the osteotomy (Figs. 18, 19, and 20).

Procedure

Room and Equipment Setup

- The intertrochanteric osteotomy procedure is usually performed with fluoroscopy to visualize the implantation of internal fixation. The flexion, valgus, and derotational osteotomy described below can all be completed with direct visualization in experienced hands using anatomic landmarks and careful measurement preoperatively. Many surgeons will prefer to visualize the osteotomy using fluoroscopy, but it is not mandatory.
- In case the procedure is being performed in combination with an acetabular osteotomy, a cell-saver may be used.
- The choice of implants for stabilizing this osteotomy can include both traditional femoral blade plates and newer proximal femoral locking plates, which also provide fixed angle fixation. The authors' preference is for proximal femoral locking plates due to ease of use and use of a drill rather than a chisel to establish fixation.

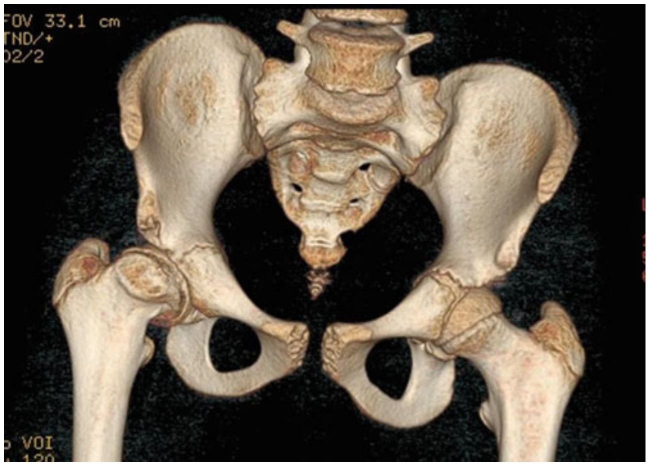


Fig. 18 3D reconstruction computed tomography (CT) of pelvis of moderate-severe SCFE with posterior and inferior displacement of the right femoral epiphysis. Notice the shortening of the proximal femur with a moderate-severe slip

Fig. 19 Isolated proximal femur CT demonstrating the inferior displacement of the femoral epiphysis as well as the prominent anterior metaphysis

Patient Positioning

- Patient is positioned in a lateral decubitus position on a radiolucent flat-top table. The use of a radiolucent flat-top table offers the advantage of easier and faster setup, ease of imaging, and the potential for multiple surgeries to be performed simultaneously (e.g., concomitant acetabular osteotomy in complex cases or concomitant 8-plate growth modulation around the knee in cases of associated genu valgum or LLD).
- Standard hip positioners for lateral positioning are preferred over a beanbag to allow free manipulation of the operative limb. Commercial hip positioners more securely stabilize the pelvis than a beanbag.
- The entire limb is then prepped in a standard fashion from the iliac crest to the toes.
- A 15 cm incision is obtained almost centered over the trochanter and curving a bit anteriorly in the proximal part. After superficial dissection, the tensor fascia lata is split in the line of the mid-trochanter (distal to trochanteric tip) and along the mid-femur in the distal part of the incision. Proximally it is split in line with the zone between tensor fascia lata and gluteus maximus fascia.
- The anterior and posterior margins of the trochanter are palpated and care is taken to protect the posterior vessels. A trochanteric flip osteotomy is now performed taking care to keep the gluteus medius, gluteus minimus, and vastus lateralis attached to the greater trochanteric fragment (Fig. 21). Gluteus minimus tendon is released from its insertion over the trochanter, and the trochanteric osteotomy is flipped anteriorly with the digastric attachment (Fig. 22).
- A “z” capsulotomy is now performed as the standard technique of safe hip dislocation and the femoral head is dislocated anterosuperiorly with hip adduction and external rotation. The ligamentum teres is divided.
- The femoral head is carefully examined, and the severity of intra-articular impingement is examined and documented with pictures (cam and pincer impingement, labral tears, acetabular and femoral cartilage status).

Fig. 20 Axial image of the proximal femur through 3D CT demonstrating the posterior displacement of the femoral epiphysis relative to the femoral metaphysis

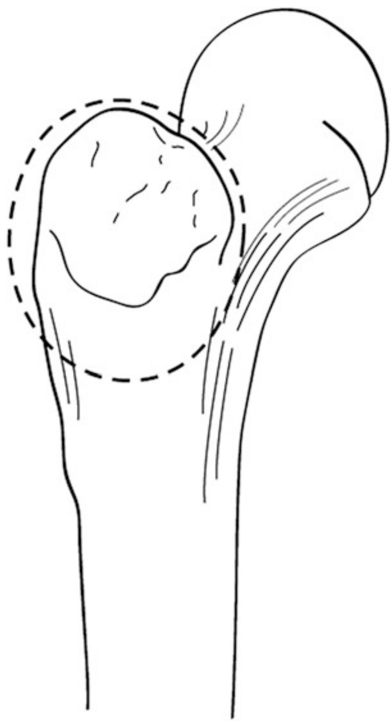


Fig. 21 Planned slide osteotomy of the greater trochanter to expose the proximal femur

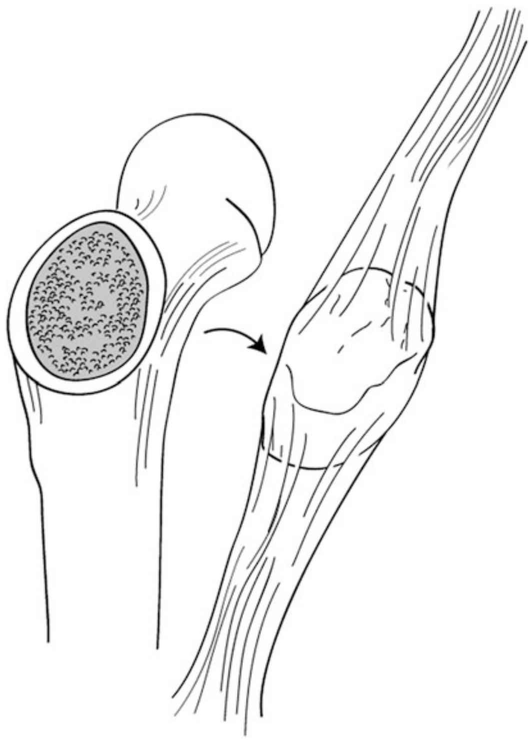


Fig. 22 Digastric osteotomy of the greater trochanter with the gluteus medius attached proximally and the vastus lateralis attached distally

- The "soft spot" with the retinacular vessels is carefully protected in the superior fragment. Posterior trochanter is osteotomized, while protecting the retinacular sleeve and soft spot, relative neck lengthening is performed.
- A femoral head-neck osteochondroplasty is performed at this point to restore the offset (especially in the 90° arc between the superolateral head-neck junction and the anterior head-neck junction). If there is a zonal pincer, it is addressed now with rim trim and labral reattachment.

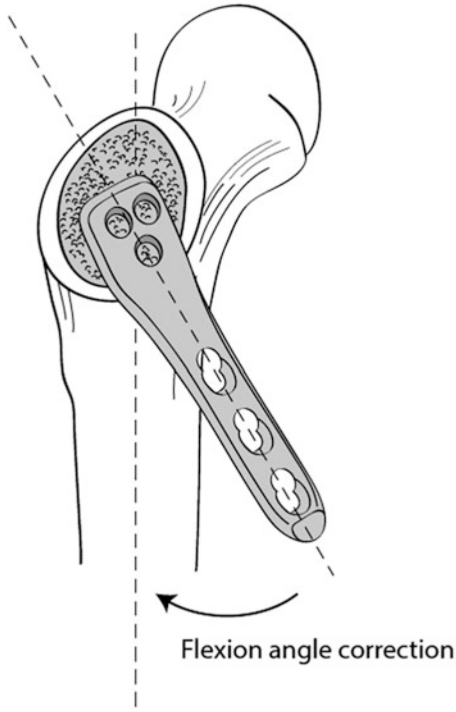


Fig. 23 The proximal femur plate is applied with the planned flexion angle correction in mind. The flexion angle correction is determined by preoperative planning

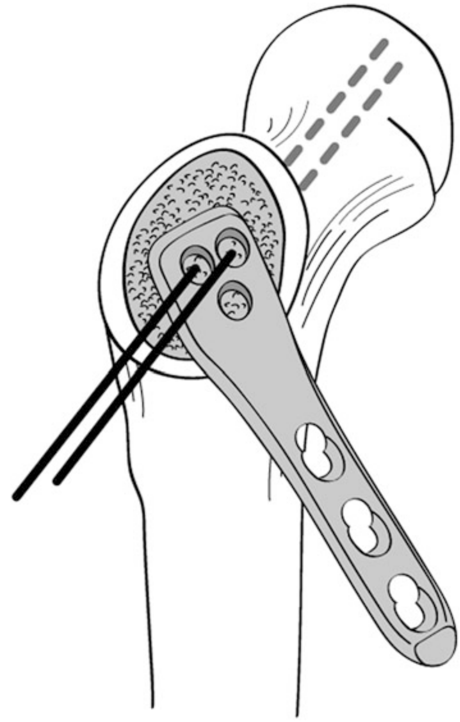


Fig. 24 With the predetermined flexion correction accounted for, the two guide wires are placed into the proximal holes of the proximally femur locking plate

- Having completed the management of intra-articular impingement, femoral head is relocated into the acetabulum. The "flexion-valgus-derotation" osteotomy is now planned.
- The level of the osteotomy is "intertrochanteric" and decided by the offset in the plate.
- Recognize that the trochanter is currently flipped and lying anteriorly. The fixation plate is placed on the osteotomized proximal femoral fragment in a position of flexion (to accommodate for the flexion component of the osteotomy) (Fig. 23). The guide wires are placed in the proximal fragment such that they are well located in both AP and lateral planes (Fig. 24). The plate is now removed and guide pins are left in situ.
- The level of osteotomy perpendicular to the femoral shaft is marked. Rotation is marked with drill holes to accommodate for the external rotation of the distal fragment (usually 30°) (Fig. 25).
- The osteotomy cut is made such that the distal fragment is cut perpendicular to the axis of the femur (Fig. 25). The flexion cut is made on the distal end of the proximal fragment (Fig. 26). Then the distal fragment is appropriately derotated to correct the intorsion (Fig. 26). The valgus cut is now finally made on to the proximal fragment to complete the correction (Fig. 27).
- The osteotomy is temporarily held with a threaded Kirschner wire placed out of the field of the incoming osteotomy fixation plate.
- The trochanteric fragment is pulled distal to its previous location, and two holes are placed in the fragment to match the guide pins previously placed in the proximal fragment (Fig. 28).
- The locking plate is now positioned over the two guide pins and held to the diaphysis with a plate-reduction clamp.
- Initially, locking screws are placed in the proximal fragment to securely fix the femoral head

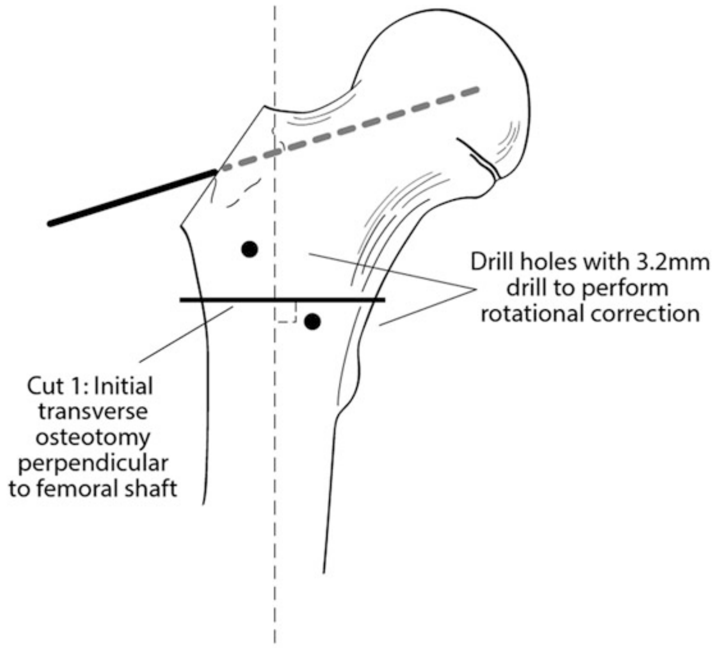


Fig. 25 The next step is to correct the torsional deformity which is accomplished by offsetting two drill bits by the preoperatively determined torsional deformity. Subsequently, an osteotomy is created which is perpendicular to the anatomic axis of the femur. This osteotomy should be made above the level of the lesser trochanter and the drill bits should be on either side of the planned transverse osteotomy

fragment. Next, a single non-locking bicortical screw is placed eccentrically in the plate along the femoral shaft to compress the osteotomy. Locking screws are subsequently placed in the shaft fragment to further stabilize the fixation. The non-locking bicortical screw is exchanged for a locking screw (Fig. 29).

- Bone graft from the osteotomy wedge is obtained and placed around the osteotomy. Hip stability and ROM is checked and documented.
- The joint capsule is loosely approximated so as not to put undue tension on the retinacular vessels.
- The wound is closed in layers.
- If any additional growth modulation procedure is required on the ipsilateral extremity (related to genu valgum in these cases) or contralateral extremity related to limb length discrepancy, it is performed at this time.
- After sterile dressings and a compression wrap are applied, the limb is placed in a knee immobilizer and a small abduction pillow is placed between the legs. Cast immobilization is not recommended (patients are typically above 12 years of age). A knee immobilizer and abduction pillow are used to help prevent dislocation by restricting the patient from placing the hip into flexed and adducted position. Final AP pelvis and lateral radiographs of the affected hip are obtained in the OR before moving the patient to PACU.

Complications

- Partial osteonecrosis of the femoral head secondary to iatrogenic damage during dissection or manipulation of the superior mobile fragment
- Complete osteonecrosis of femoral head
- Distraction at the osteotomy leading to delayed union or nonunion
- Infection/osteomyelitis nerve palsy due to prolonged position of dislocation
- Symptoms/painful hardware
- Chondrolysis
- Hardware failure
- Malrotation or malunion

Postoperative Care and Rehabilitation

- Knee immobilizer for support and to prevent hip flexion (10–14 days)
- Crutch walking as soon as POD #1
- Toe-touch weight bearing for 4 weeks

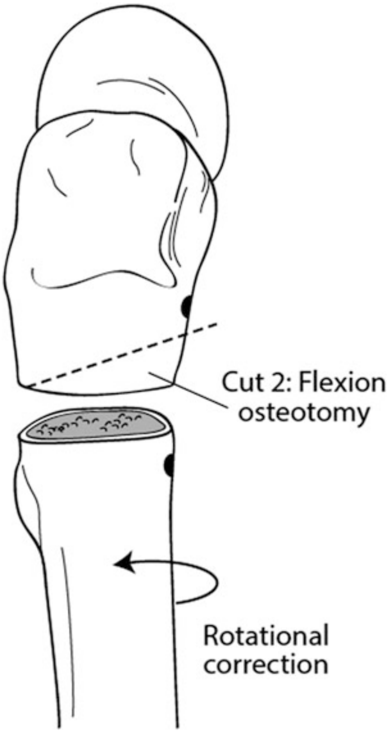


Fig. 26 Subsequently, the planned flexion osteotomy is performed after the rotational correction. This flexion osteotomy should begin at the posterior corner of the proximal fragment. The flexion osteotomy was determined by the preoperative imaging and is determined by the amount of posterior displacement of the epiphysis

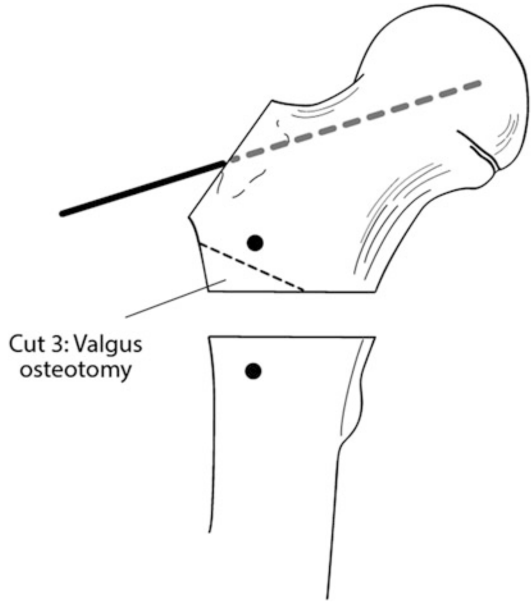


Fig. 27 The final osteotomy is a valgus-producing osteotomy to correct the coxa vara to a normal neck-shaft angle

- Partial weight bearing (20–50 %) for the next 2 weeks, followed by weight bearing as tolerated depending on healing
- First post-op visit at 10 days to 2 weeks for wound healing, followed by visit at 6 weeks (with radiographs), and then at 3 months (Figs. 30 and 31)

Outcomes

Schai and Exner reported the 24-year outcome of the Imhäuser intertrochanteric osteotomy in 51 patients treated for chronic SCFE. The indication was a moderately healed SCFE as determined by Southwick angle (>30°). At final follow-up, 55 % of patients had no osteoarthritis, 28 % developed moderate degenerative changes, and 17 % had developed severe coxarthrosis [15].

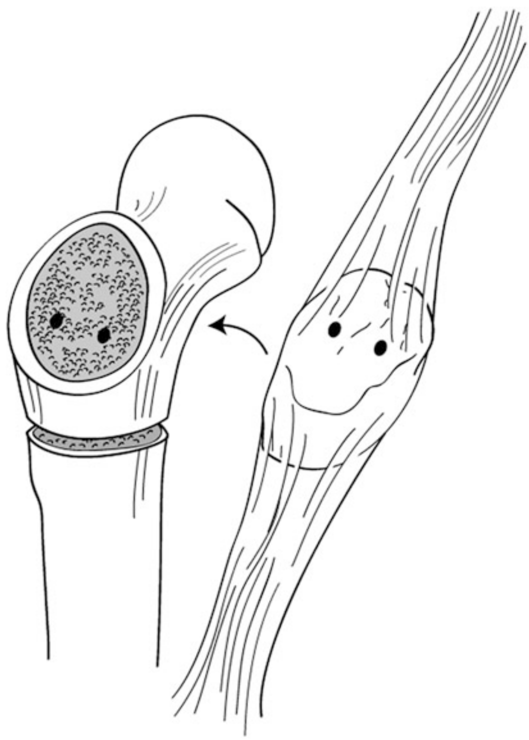


Fig. 28 The trochanter is nonanatomically reattached to the proximal femur. The trochanter is fixed in a distal position to increase the abduction force

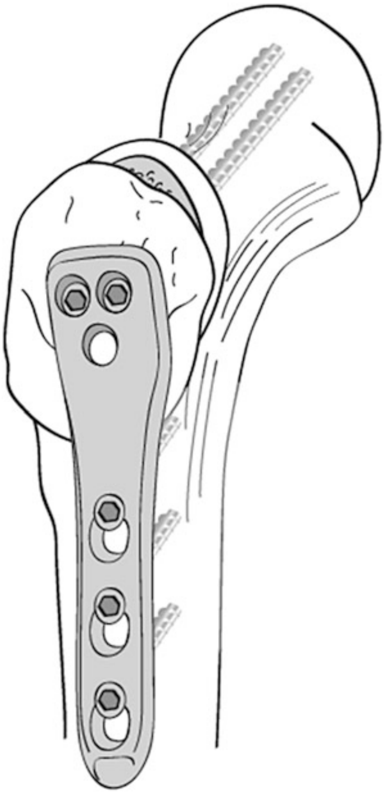


Fig. 29 The proximal femoral plate (preferably locking plate) is placed over the trochanteric fragment in its distalized position. The oblong holes in the distal plate are used to sequentially compress the proximal and distal fragments together to increase stability

Lino et al. retrospectively reviewed the radiographic outcomes of 37 patients with moderate-severe chronic SCFE treated with a Southwick intertrochanteric osteotomy over a 20-year period. They noted no osteonecrosis as well as a significant 30° improvement of the Southwick angle in their series. The authors did report chondrolysis in eight patients following Southwick osteotomy. Clinical outcomes were not reported [16].

Kartenbender et al. retrospectively reviewed the clinical and radiographic outcomes of 35 patients treated with the Imhäuser intertrochanteric osteotomy at a mean follow-up of 23.4 years. The average age at surgery was 13.7 years and the surgical indication was a slip angle >40°. Seventy-seven percent of patients achieved good-excellent clinical outcomes and 67 % of patients had good-excellent radiographic outcomes. Two patients underwent osteonecrosis and three patients developed severe coxarthrosis [17].

Merchan et al. reported on the clinical and radiographic outcomes of 36 patients with chronic moderate SCFE (as determined by Southwick angle >30°) treated with an intertrochanteric osteotomy. The average age at index surgery was 14.1 years and the mean follow-up was 7.5 years. The group reported a 10 % incidence of complications which included two cases of osteonecrosis, four cases of residual coxa vara, and loss of fixation in two patients requiring reoperation. Clinical results were good in 14 patients and fair-poor in 22 patients. Radiographic outcomes were good in 13 patients and fair-poor in 23 patients. The average correction in this series was 30°, and patients in whom the postoperative Southwick angle was <10° had improved outcomes [18].

Femoral Derotation Osteotomy in Adolescents and Adults

Femoral version is a dynamic process that begins with increased femoral anteversion at birth and through the early years of development. Increased femoral anteversion normalizes in the vast majority of children by 8 years of age [19–24]. However, femoral version is also relatively static after 8 years of age [19]. The range of normal femoral anteversion in adolescents and adults is 14° in men and 18° in women with a range of +/−10° [25]. Femoral deformity in the form of malrotation/malorientation is a common cause of hip and knee pain in teenagers and adults. The malrotation may lead to symptomatic intoeing gait and patellofemoral malalignment. Patellofemoral malalignment may be a root cause of patellar instability and secondary chondromalacia and arthrosis of the patellofemoral joint. Surgery may be required in a small subset of patients who fail to respond to conservative treatment [19, 26, 27]. Increased femoral anteversion in the form of femoral malrotation has also been associated as a cause of hip pain in young adults.

The diagnosis of pathologic femoral anteversion starts with a clinical examination. Patients at

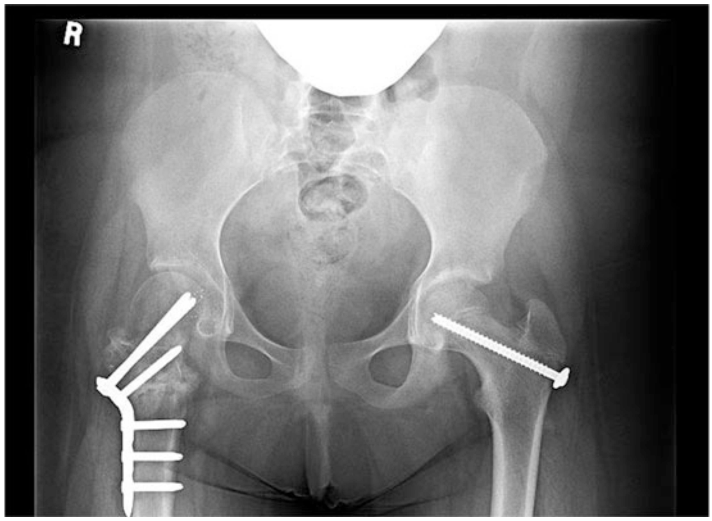


Fig. 30 Postoperative radiograph of the AP pelvis following correction with a valgus-producing osteotomy for moderate-severe SCFE. Notice the relative lengthening of the shortened femur with the valgus osteotomy. The contralateral hip has also been pinned due to increased risk of SCFE bilaterality

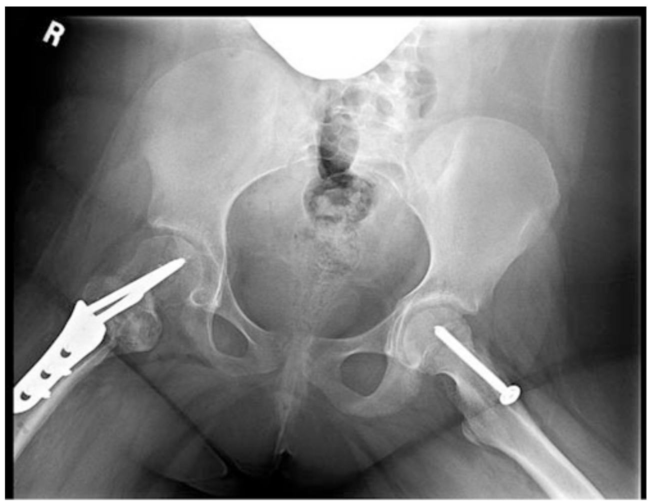


Fig. 31 Frog lateral radiograph of the pelvis demonstrating adequate fixation of the osteotomy. Notice the femoral head-neck junction with restored head-neck offset with a primary osteochondroplasty

skeletal maturity should have an equal range of motion with respect to internal and external rotation. Excessive internal rotation is often the first clue of excessive femoral anteversion. Full-length alignment radiographs are necessary for assessing for other sources of patellar maltracking including genu valgum. The gold standard for assessing rotational profile is a computed tomography torsional profile ("gunsight CT") which measures the femoral neck angle, posterior condylar angle, proximal tibial axis, and intermalleolar angle. The range of normal femoral anteversion in adolescents and adults is 14° in men and 18° in women with a range of +/−10° [25]. The current literature does not support a specific value of femoral anteversion above which a rotational osteotomy should be performed. The predominance of series include patients who failed conservative management and had gross inequality of internal to external rotation [19, 26, 27].

The following section describes a technique for femoral derotation osteotomy over an intramedullary nail, as well as provided some tips for technical ease. This procedure provides a safe and reliable method to correct femoral malrotation in adolescents and adults with a rigid intramedullary nail [28–30].

Patient Selection

Indications

- Excessive femoral ante- or retroversion
- Rotational malunions
- Patellofemoral malalignment or instability with excess femoral anteversion and tibial torsion (external rotation)
- Developmental proximal femoral deformity with component of excessive torsion

Relative Contraindications

- Juvenile patients (young patients have potential risk to trochanteric physis)
- Intramedullary fixation in patients with femoral canal diameter less than 8 mm (technical challenge)
- Extreme obesity
- Patellar instability cases where there is already advanced arthritis

Preoperative Imaging

- Standardized AP radiograph of the pelvis.
- Full-length standing alignment radiographs of the lower extremities.
- MRI torsional profile is the preferred method due to lack of radiation exposure and equal accuracy and reliability [14]. CT can be used in centers where MRI torsional profile is not available or feasible.

Procedure

Room Setup

- Prior to prepping and draping of the affected limb, it is essential to ensure that there is adequate space for the image intensifier.
- Due to the size of the instrumentation sets, as well as the length of the guide wire being utilized, it is important to perform this procedure in a large enough operating room, having adequate room for the table, instruments, image, and OR personnel.
- The drapes separating the surgical field and the anesthesia personnel should be high enough that the intramedullary nail guide wire does not have the risk of contamination during the process of reaming and nailing.

Patient Positioning

- Derotational femoral osteotomy over an antegrade intramedullary nail can be performed in either a supine or a lateral decubitus (author's preference) position. Additionally, the procedure can be carried out on a fracture table or radiolucent flat-top table. The use of a radiolucent flat-top table offers the advantage of easier and faster setup, ease of imaging, and the potential for multiple surgeries to be performed simultaneously (e.g., concomitant tibial derotational osteotomy in complex malalignment syndrome).
- The authors' preference for intramedullary fixation over plate osteosynthesis is due to load-sharing properties, smaller surgical exposure, decreased blood loss, and local reaming material at the osteotomy benefits of intramedullary fixation. From a mechanical perspective, IM nails are load-sharing devices and plates are load bearing. These load-sharing mechanics allow earlier weight bearing with the intramedullary nails than with the plates. Additionally, exposure for an IM nail is fairly limited and blood loss is frequently decreased which may be relevant in multistage procedures. Similarly, IM implant removal is achieved with smaller exposure compared to plates which often need larger incisions for removal of implants. IM reamings produce autologous bone graft at the osteotomy site, and reamed material removed can be replaced at the osteotomy site to help ensure uneventful union. Finally, in the event that the osteotomy is unintentionally oblique, rotation at the osteotomy can cause distraction at the osteotomy. Plating the oblique osteotomy in a distracted position without appropriate compression may lead to nonunion, especially in the absence of local bone graft. IM nails are more forgiving to slightly oblique osteotomies as compression can be generated with early weight bearing to promote union. For these reasons listed, the authors' preference is for intramedullary fixation of rotational femoral osteotomies.
- Standard hip positioners for lateral positioning are preferred over a beanbag to allow free

manipulation of the operative limb. Commercial hip positioners more securely stabilize the pelvis than a beanbag.

The procedure will be described in the lateral position utilizing the Synthes Adolescent Lateral Femoral Nail System (West Chester, Pennsylvania)

Surgical Technique

- The entire limb is then prepped in a standard fashion from the iliac crest to the toes.
- A small (<5 cm) incision is made over the tip of the greater trochanter in line with the femoral shaft. The fascia and abductor muscles are split longitudinally to expose the proximal trochanter. The trochanter is most prominent in the lateral position, thus making exposure easier. The anterior and posterior margins of the trochanter are palpated and care is taken to protect the posterior vessels.
- An awl or guide pin is then placed in the starting position for the lateral entry antegrade nail, roughly 10° lateral to the tip of the trochanter. If a guide pin is used, its position can be confirmed under fluoroscopic guidance. When a guide pin is used, an additional step of preparing the proximal femur with a cannulated drill is necessary.
- A flexible, ball-tipped guide wire is now inserted in the canal and its position in the canal is confirmed with the image intensifier. Care should be taken to avoid violating the distal femoral physis in those patients with an open distal femoral physis. After confirming the guide wire's position in the distal femur, length and size of the nail are then determined.
- If reaming is desired, canal reaming is initiated with the smallest end-cutting reamer and progressed up to the desired width. The guide wire must be stabilized when backing the reamer from the canal. For derotation osteotomies, we ream the distal fragment 1 mm more than the nail diameter and the proximal fragment at least 2 mm more than the diameter to allow for nail accommodation with altered morphology of the canal after derotation.
- Reaming material is collected as it is removed from the femur for later bone grafting of the osteotomy site. A small open incision to create the osteotomy allows for placement of the reaming material around the osteotomy to promote bone healing.
- The osteotomy site is now determined with fluoroscopic guidance. Osteotomy within the subtrochanteric region is preferred so that there is sufficient proximal length for a locking screw and an isthmic fit can be achieved distally.
- Once the osteotomy site is determined, a small (<5 cm) incision is made along the lateral aspect of the thigh and careful dissection is performed down to the femur. The iliotibial band and vastus lateralis fascia are handled with care to allow for good repair at the conclusion of the procedure. Care is taken to coagulate any perforating vessels along the posterior aspect of the femur. The periosteum is incised and retractors are placed around the femur subperiosteally to provide sufficient exposure of the planned osteotomy site.
- Two unicortical drill holes are now made with a 3.2 mm drill bit (for marking the rotational alignment in the proximal as well as distal fragment), taking care to make the distal hole at least 2 cm from the planned osteotomy site. The proximal hole is made along the lateral aspect of the femur, while the distal hole is offset anteriorly or posteriorly by the appropriate distance (using drill bit in the hole to estimate the angle), such that rotation of the distal fragment brings this hole in line with the proximal hole. The senior author prefers the drill hole technique as compared to a saw cut or proximal/distal K-wire placement technique to quantify the rotation of the femur.
- The guide wire is now removed, and the osteotomy is now performed with an oscillating saw, taking care to place the cut exactly perpendicular to the femur, and avoiding any bending or rotational torque moment until the cut is complete to prevent spike formation, obliquity, or spiraling of the cut.
- Once the osteotomy is complete, the guide wire is again placed in the canal, and position and length are reconfirmed. The intramedullary

nail is inserted and advanced appropriately in the proximal fragment while making sure that the osteotomy is not distracted, and is advanced distally taking care not to penetrate the distal femoral physis.
- Appropriate derotation (usually external rotation) of the distal femur is now accomplished and confirmed with the position of the previously made alignment drill holes. A 4.5 mm fully threaded cortical screw (usually a 16 mm unicortical screw) is placed through the previously made distal alignment hole which was created with a 3.2 mm drill bit to temporarily hold the derotation. This screw engages one of the grooves on the surface of the Synthes Adolescent Lateral Femoral Nail and locks the nail in the canal and to the bone in this rotated position during the rest of the procedure.
- With the nail in place, locking screws are placed. The proximal locking screw is usually performed using the guide on the nail insertion handle. The locking screw is preferentially placed obliquely from the greater trochanter to the lesser trochanter to avoid instrumentation of the femoral neck.
- The proximal locking screw is preferentially placed prior to the placement of the temporary derotation holding screw. Distal locking screws are usually placed using the perfect circles technique. With the nail secured in position, the insertion handle is removed. An end cap is recommended to facilitate later removal of the nail.
- With adequate derotation dialed into the femur, the final nail position and femoral alignment are confirmed using fluoroscopic image intensification. The distal interlock is placed using a perfect circles technique to fix the derotation correction. The surgical wounds at the nail entry site, osteotomy site, and proximal and distal locking screws are closed in layers.
- After sterile dressings and a compression wrap are applied, the limb is placed in a knee immobilizer for support. Full-length AP and lateral radiographs of the affected limb are obtained postoperatively.

Complications

- Inadequate rotation/malposition
- Distraction at the osteotomy leading to delayed union or nonunion
- Infection/osteomyelitis
- Damage to trochanteric physis or the distal femoral physis
- Avascular necrosis of the femoral head related to damage during dissection or inappropriate entry point
- Nerve palsy
- Compartment syndrome and vascular deficit
- Symptoms/painful hardware

Postoperative Care and Rehabilitation

- Knee immobilizer for 1 week – for soft tissue rest and pain control
- Crutch walking as soon as patient can tolerate (POD #1 or 2)
- Toe-touch weight bearing for 2 weeks
- Partial weight bearing (20–50 %) for the next 4 weeks, followed by weight bearing as tolerated
- First post-op visit at 10–14 days to assess wound healing, followed by next visit at 6 weeks (with radiographs)

Rehabilitation

- Isometric quads and ankle pump exercises starting POD #0
- Knee ROM exercises starting after 2 weeks (I start mine at 1 week, any reason to delay to 2 weeks?)
- Quadriceps strengthening starting at 4 weeks
- Hip abductor strengthening and hamstring and IT band stretching to start at 6 weeks
- WBAT or FWB from 6 weeks
- Full release to sports not before 12 weeks (and solid radiographic healing with 5/5 muscle strength in lower extremity)

Outcomes

Gordon et al. retrospectively reviewed their series of rotational femoral osteotomies in 13 patients with 21 affected limbs treated surgically. The average patient age was 10.7 years and the mean follow-up was 2.6 years. The surgical indication was frequent tripping interfering with activities of daily living and sport. The mean preoperative hip internal rotation was 77° and the mean external rotation was 15°. Following rotational osteotomy, the mean internal rotation was 40° and the mean external rotation was 57°. The authors reported no complications and all osteotomies healed. The patients reported no limp and no tripping interfering with activities of daily living or sports [19].

Bruce et al. also retrospectively reviewed their series of 14 patients with 27 symptomatic limbs with miserable malalignment syndrome as defined by excessive femoral anteversion, increased tibial external torsion, and patellofemoral pain. The average patient age was 14.9 years and the mean follow-up was 5.2 years. Preoperatively, the average hip internal rotation was 85° and the mean hip external rotation was 33°. A preoperative CT demonstrated a mean femoral anteversion of 35°. The average rotational correction was 35°. The authors reported one femoral shaft fracture through a screw hole requiring revision. All femoral osteotomies healed at an average of 3 months. All patients reported no limb and resolved knee pain. Eleven of fourteen participated in organized sports.

Summary

The proximal femoral osteotomy is an instrumental tool of the hip surgeon and has been a gold standard technique for hip deformity correction since the orthopedic community began addressing these problems surgically. Recently there has been renewed interest and development of new techniques of proximal femoral osteotomy to more directly address pathoanatomy. Focused research on the vascular supply to the femoral head has provided the opportunity to directly treat hip deformity that previously would have been left to natural history and inevitable coxarthrosis. While these new techniques have engendered considerable interest, long-term outcomes demonstrating benefit are not available at this point. The previous chapter's focus on the technique, indications, and outcomes of femoral head reduction osteotomy, intertrochanteric osteotomy, and subtrochanteric rotational osteotomy should serve as a base upon which further study begins. The techniques described in the previous chapter are among the most technically challenging in all of orthopedic surgery. The consequences of poorly performed (or poorly planned) surgery are considerable. Finally, the previous chapter only focuses on three techniques, yet there are a number of other osteotomies of the proximal femur that the hip surgeon should be aware of such that the correct procedure can be indicated for the correct patient.

References

1. Gautier E, Ganz K, Krugel N, Gill T, Ganz R. Anatomy of the medial femoral circumflex artery and its surgical implications. J Bone Joint Surg. 2000;82(5):679–83.
2. Peltier LF. Nicolas Andry. The designer of orthopedic iconography. Clin Orthop Relat Res. 1985;200:54–6.
3. Tannast M, Macintyre N, Steppacher SD, Hosalkar HS, Ganz R, Siebenrock KA. A systematic approach to analyse the sequelae of LCPD. Hip Int J Clin Exp Res Hip Pathol Ther. 2013;23 Suppl 9:61–70.
4. Paley D. The treatment of femoral head deformity and coxa magna by the Ganz femoral head reduction osteotomy. Orthop Clin North Am. 2011;42 (3):389–99. viii.
5. Ganz R, Horowitz K, Leunig M. Algorithm for femoral and periacetabular osteotomies in complex hip deformities. Clin Orthop Relat Res. 2010;468(12):3168–80.
6. Ganz R, Huff TW, Leunig M. Extended retinacular soft-tissue flap for intra-articular hip surgery: surgical technique, indications, and results of application. Instr Course Lect. 2009;58:241–55.
7. Leunig M, Ganz R. Relative neck lengthening and intracapital osteotomy for severe Perthes and Perthes-like deformities. Bull NYU Hosp Jt Dis. 2011;69 Suppl 1:S62–67.
8. Burian M, Dungl P, Nanka O, et al. Anteromedial wedge reduction osteotomy for the treatment of femoral head deformities. Hip Int J Clin Exp Res Hip Pathol Ther. 2013;23(3):281–6.
9. Alshryda S, Tsnag K, Ahmed M, Adedapo A, Montgomery R. Severe slipped upper femoral epiphysis; fish osteotomy versus pinning-in-situ: an eleven year perspective. Surg J R Coll Surg Edinb Irel. 2013. p. 1–5
10. Monazzam S, Dwek JR, Hosalkar HS. Multiplanar CT assessment of femoral head displacement in slipped capital femoral epiphysis. Pediatr Radiol. 2013;43 (12):1599–605.
11. Ramachandran M, Azegami S, Hosalkar HS. Current concepts in the treatment of adolescent femoroacetabular impingement. J Child Orthop. 2013;7(2):79–90.

12. Mueller ME. Intertrochanteric osteotomy: indication, preoperative planning, technique. In: Schatzker J, editor. The intertrochanteric osteotomy, vol. 1. 1st ed. New York: Springer; 1984. p. 22–66.
13. Hosalkar HS, Pandya NK, Bomar JD, Wenger DR. Hip impingement in slipped capital femoral epiphysis: a changing perspective. J Child Orthop. 2012;6 (3):161–72.
14. Muhamad AR, Freitas JM, Bomar JD, Dwek J, Hosalkar HS. CT and MRI lower extremity torsional profile studies: measurement reproducibility. J Child Orthop. 2012;6(5):391–6.
15. Schai PA, Exner GU. Indication for and results of intertrochanteric osteotomy in slipped capital femoral epiphysis. Orthopade. 2002;31(9):900–7.
16. Lino Jr W, Akkari M, Waisberg G, Braga SR, Santili C. Chronic slipped capital femoral epiphysis: a radiographic evaluation of the Southwick osteotomy. J Pediatr Orthop B. 2013;22(6):536–41.
17. Kartenbender K, Cordier W, Katthagen BD. Long-term follow-up study after corrective Imhauser osteotomy for severe slipped capital femoral epiphysis. J Pediatr Orthop. 2000;20(6):749–56.
18. Merchan EC, Na CM, Munuera L. Intertrochanteric osteotomy for the treatment of chronic slipped capital femoral epiphysis. Int Orthop. 1992;16(2):133–5.
19. Gordon JE, Pappademos PC, Schoenecker PL, Dobbs MB, Luhmann SJ. Diaphyseal derotational osteotomy with intramedullary fixation for correction of excessive femoral anteversion in children. J Pediatr Orthop. 2005;25(4):548–53.
20. Crane L. Femoral torsion and its relation to toeing-in and toeing-out. J Bone Joint Surg Am. 1959;41-A(3):421–8.
21. Engel GM, Staheli LT. The natural history of torsion and other factors influencing gait in childhood. A study of the angle of gait, tibial torsion, knee angle, hip rotation, and development of the arch in normal children. Clin Orthop Relat Res. 1974;99:12–7.
22. Fabry G, Cheng LX, Molenaers G. Normal and abnormal torsional development in children. Clin Orthop Relat Res. 1994;302:22–6.
23. Kling Jr TF, Hensinger RN. Angular and torsional deformities of the lower limbs in children. Clin Orthop Relat Res. 1983;176:136–47.
24. McSweeny A. A study of femoral torsion in children. J Bone Joint Surg. 1971;53(1):90–5.
25. Braten M, Terjesen T, Rossvoll I. Femoral anteversion in normal adults. Ultrasound measurements in 50 men and 50 women. Acta Orthop Scand. 1992;63(1):29–32.
26. Delgado ED, Schoenecker PL, Rich MM, Capelli AM. Treatment of severe torsional malalignment syndrome. J Pediatr Orthop. 1996;16(4):484–8.
27. Bruce WD, Stevens PM. Surgical correction of miserable malalignment syndrome. J Pediatr Orthop. 2004;24(4):392–6.
28. Keeler KA, Dart B, Luhmann SJ, et al. Antegrade intramedullary nailing of pediatric femoral fractures using an interlocking pediatric femoral nail and a lateral trochanteric entry point. J Pediatr Orthop. 2009;29 (4):345–51.
29. Gordon JE, Swenning TA, Burd TA, Szymanski DA, Schoenecker PL. Proximal femoral radiographic changes after lateral transtrochanteric intramedullary nail placement in children. J Bone Joint Surg Am. 2003;85-A(7):1295–301.
30. Momberger N, Stevens P, Smith J, Santora S, Scott S, Anderson J. Intramedullary nailing of femoral fractures in adolescents. J Pediatr Orthop. 2000;20(4): 482–4.

Surgical Technique: Arthroscopic Treatment of Chronic Slipped Capital Femoral Epiphysis

37

Brandon Beamer, Benton E. Heyworth, Yi-Meng Yen, and Mininder S. Kocher

Contents

B. Beamer (✉)
Boston Children's Hospital, Boston, MA, USA
e-mail: brandon.beamer@childrens.harvard.edu

B.E. Heyworth
Division of Sports Medicine, Boston Children's Hospital, Instructor in Orthopaedic Surgery, Harvard Medical School, Boston, MA, USA
e-mail: benton.heyworth@childrens.harvard.edu

Y.-M. Yen
Harvard Medical School Children's Hospital Boston, Boston, MA, USA
e-mail: yi-meng.yen@childrens.harvard.edu

M.S. Kocher
Harvard Medical School Children's Hospital Boston, Boston, MA, USA

Division of Sports Medicine, Orthopedic Surgeon, Boston Children's Hospital, Boston, MA, USA
e-mail: mininder.kocher@childrens.harvard.edu

Abstract

Slipped capital femoral epiphysis (SCFE) is the most common pediatric hip disorder and is a potential precursor to early hip arthritis in severely affect patients. The offending lesion is the prominent anterosuperior femoral neck metaphysis that displaces anteriorly during epiphyseal slippage. Though patients may be able to bear weight after healing the acute SCFE, eventual hip pain, decreased range of motion, and intra-articular derangement may develop. Arthroscopic treatment of proximal femoral deformity resulting from SCFE has been described as a joint preservation technique and has been demonstrated to be a safe and effective alternative to open surgical dislocation and osteoplasty.

Introduction

Epidemiology

Slipped capital femoral epiphysis (SCFE) is the most common pediatric hip disorder, affecting approximately 11 in every 100,000 skeletally immature children [1]. The average age of onset for males is 12.7 years and for female is 11.1 years, reflecting the differential rates of skeletal maturity between the sexes [1]. Black, Hispanic, and Native American children are more often affected than White and Asian children [1], and the causality may be likely linked to higher

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_118

rates of obesity in these populations [2]. Other environmental factors may also contribute to SCFE. For example, the Northeastern United States has the highest incidence of SCFE [1], which may be attributable to the limited capacity for cutaneous vitamin D production due to decreased sun exposure [3].

Presentation

Patients with SCFE may present in one of three different temporal classifications. Acute SCFE cases occur with a patient describing new hip, thigh, and/or knee pain, a limp, and difficult weight bearing (Fig. 1). These patients may present with or without a discrete traumatic event. Chronic SCFE patients often present with a history of remote or ongoing mild to moderate hip or knee pain with associated decreases in hip range of motion and signs and symptoms of femoroacetabular impingement. Lastly, acute-on-chronic presentation involves elements of both forms, in which acute difficulty weight bearing or refusal to bear weight follows a history of chronic hip pain. Because 26 % of children with chronic SCFE present with isolated knee or medial thigh pain, delays in diagnosis are common with the conditions, with all three forms of presentation being associated with potential sequelae such as labral tears, articular cartilage damage, further slip, and potential avascular necrosis (AVN) [4].

A second classification system developed for the presentation of SCFE was proposed by Loder et al. and has seen widespread acceptance and utilization. The Loder classification describes patients as having either stable SCFE, in which a child is willing to bear weight – with or without the presence of a limp – or unstable SCFE, in which there is refusal to bear weight. Importantly, the rates of avascular necrosis (AVN) of the hip have been clearly correlated with stability, with unstable SCFE having a 47 % rate of subsequent AVN, compared with no cases of AVN in stable SCFE in his study [5].

Pathoanatomy

Slipped capital femoral epiphysis accurately describes the disease process in which the structural integrity of the proximal femoral physis is compromised, leading to the epiphysis remaining reduced within the acetabulum, while the pull of the hip flexors displaces the proximal femoral metaphysis anteriorly. The resulting deformity leads to a loss of sphericity of the femoral head-neck junction, thereby decreasing the head-neck offset (Fig. 2). This is described by the alpha angle (Fig. 3a–b). A cam lesion forms at the anterosuperior head-neck junction of the proximal femur and impacts the acetabular rim in flexion and internal rotation. Alpha angles greater than 60° describe cam lesions that have been associated with a greater degree of labral and articular cartilage damage [6]. SCFE severity is generally described by the degree of slippage or the slip angle, described by Southwick [7]. The slip angle is formed by the long axis of the femoral neck and a line perpendicular to the

Fig. 1 Frog leg radiograph demonstrating a severe acute SCFE slip

Fig. 2 AP radiograph illustrating a mild SCFE slip with CAM impingement

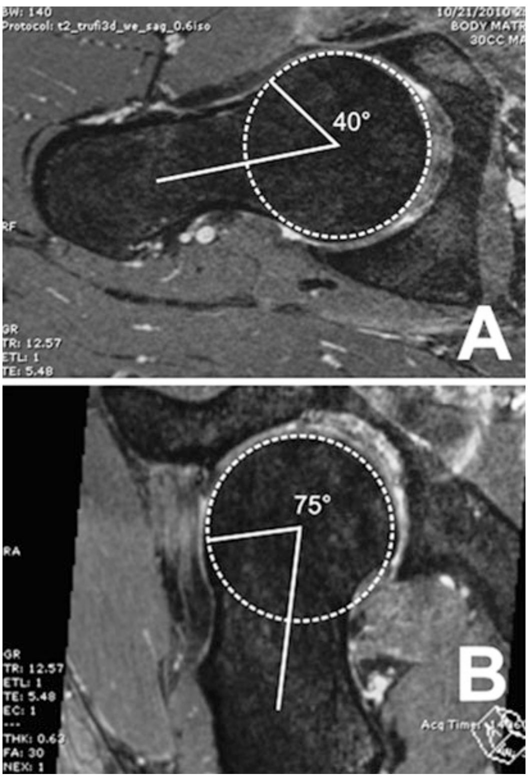


Fig. 3 Hip radiographs illustrating (**a**) normal and (**b**) abnormal alpha angles

physis and bisecting the epiphysis. Angles are measured on Dunn lateral, or frog leg lateral radiographs are categorized as mild (0–30°), moderate (30–60°), or severe (>60°) (image) [8]. Alternatively, the alpha angle may be measured on axial MRI or low-dose CT scan if hardware artifact impedes measurement on MRI.

Rab et al. performed computer modeling of SCFE cases, demonstrating that even mild slips lead to impaction of the proximal femur against the acetabular rim, and patients walk with increasing degrees of external rotation with increasing slip angle to prevent impingement with ambulation alone [9]. Leunig's intraoperative findings confirm that patients with even mild slips and less than 3 months of symptoms all had some degree of labral damage [10]. With SCFE, two types of impingement may occur. Minor slips, and those that have remodeled to smaller cam lesions, may lead to "inclusion" type impingement. This occurs when the prominent metaphyseal region enters the hip joint with range of motion, increasing shear forces across the articular cartilage. On the other hand, moderate and severe slips have greater metaphyseal prominence at the femoral head-neck junction that limits range of motion, and the lesion impacts the acetabular rim rather than entering the joint. This type of impingement is known as "impaction" impingement and leads to labral derangement and levering of the femoral head posteriorly in acetabulum resulting in countercoup impaction of the femoral head against the posterior acetabulum [8, 11–13]. The natural history of untreated SCFE or in situ pinning without addressing the underlying deformity may lead to the development of arthrosis in a proportion of patients. Some degree of arthrosis was reported in 92 % of patients at an 11-year follow-up in one study [14], with another report of approximately 5 % of patients requiring a total hip arthroplasty by 16 years of follow-up [15].

Indications

Patients with symptomatic femoral acetabular impingement (FAI) from a chronic, stable SCFE may be amenable to treatment with arthroscopic proximal femoral osteoplasty to increase the proximal femoral head-neck offset. Surgical dislocation and open proximal femoral osteoplasty remains a viable and, often times, preferable approach for more significant deformity resulting from severe slips [10, 16, 17]. Femoral neck osteoplasty allows for greater range of motion without pathological abutment of the prominent metaphyseal deformity against the acetabular rim. Patients with chronic slips may simply complain of decreased range of motion as compared to the contralateral side, rather than joint pain [4]. Pain with activities or positions that place the hip in high flexion are common. Intra-articular derangement often presents with pain radiating to the groin or thigh, as well as referred pain to the knee. Patients may demonstrate the "C-sign," as described by Byrd [18], when discussing their pain. Notably, some patients may be asymptomatic, but may have radiographic or MRI evidence of a labral tear or early acetabular cartilage delamination or degeneration. Given the natural history of this disease process, arthroscopy with the goal of joint preservation, removal of the offending bony lesion, and repair or debridement of damaged labral tissue is a reasonable surgical option.

Patients with mild chronic SCFE (slip angle <30°) may have alterations in joint biomechanics significant enough to create chondral and labral derangements due to inclusion impingement [10]. Joint space narrowing, signs, and symptoms of arthrosis may be evident clinically and radiographically. The earliest sign of arthrosis may currently be detected on delayed gadolinium-enhanced magnetic resonance imaging of cartilage (dGEMRIC). This study utilizes indirect contrast enhancement of articular cartilage by intravenously administered gadolinium. Gadolinium is a negatively charged element that binds to positively charged glycosaminoglycans (GAG) that comprises the matrix of articular cartilage. Areas of early arthrosis will have a relatively lower concentration of GAG and, thus, less contrast enhancement. Zilkens et al. studied 32 hips with varying severity of SCFE (10 mild, 22 moderate) treated with in situ pinning, on average 11 years prior to dGEMRIC imaging [19]. The authors correlated postoperative alpha angles with dGEMRIC findings. Though 85 % of the hips were Tonnis grade 0, showing no signs of joint space narrowing, those with alpha angles >60° did have significantly decreased gadolinium uptake and therefore greater early cartilage degeneration than those with normal proximal femoral head-neck offset (alpha angle <50°). The subset of patients in this study with alpha angles >60°, meeting FAI criteria, had a mean Harris Hip Score greater than 95, yet had degenerative changes on dGEMRIC. While patients with chronic, stable SCFE or those post-pinning with residual decrease in head-neck offset may be asymptomatic, subtle degenerative changes in the articular cartilage may be occurring. This may set the cascade of degenerative joint disease in motion. The goal of hip arthroscopy after SCFE is to restore the head-neck offset (alpha angle <50°) to prevent, or slow, these changes (Fig. 4a–b).

Provided adequate healing of any acute or unstable SCFE lesion has occurred, hip arthroscopy need not be delayed until skeletal maturity, as the technique has been shown to be a relatively safe procedure and effective approach for skeletally immature patients. Cam lesions typically develop at the level of the proximal femoral physis in the skeletally immature patient and can been safely debrided [20, 21]. No cases of physeal separation, growth arrest, femoral neck fracture, or AVN have been described after hip arthroscopy in the skeletally immature patient [22–25].

In summary, the indications for hip arthroscopy in patients after in situ pinning or untreated residual SCFE with uncorrected deformity may include symptomatic FAI, decreased range of motion, pain with activities that place the hip in high flexion, early degenerative changes on plain films or dGEMRIC imaging, and/or evidence of intra-articular derangement including labral and articular cartilage lesions. Contraindications to hip arthroscopy for chronic SCFE include significant arthrosis (joint space <2 mm), concomitant hip dysplasia, patient who are incapable of

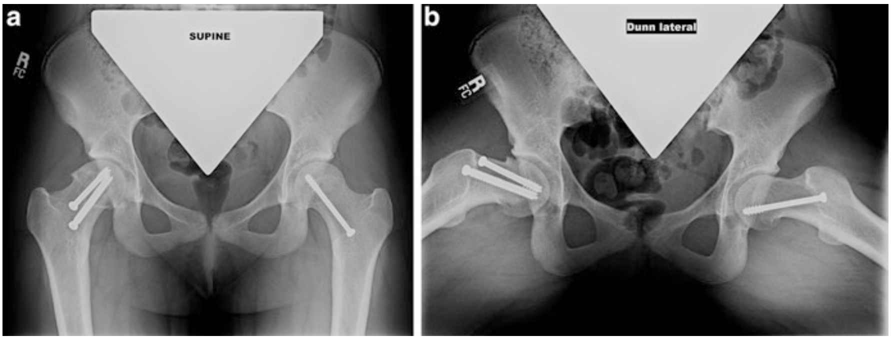


Fig. 4 Radiographs demonstrating a CAM deformity after SCFE treatment. (**a**) AP radiograph and (**b**) lateral Dunn radiograph

complying with the postoperative rehabilitation regimen, and severe SCFE, which may be better treated with open intertrochanteric osteotomy.

Technique

Setup and Diagnostic Arthroscopy

Hip arthroscopy is most commonly performed in the supine position on a traction table. A well-padded, large circumference pudendal post is placed between the legs, and the patient's feet are placed in well-padded boots at the end of the traction table. Typically 50–75 lbs of traction is applied, or enough to distract the hip 1 cm and observe a vacuum sign within the joint. Fluoroscopy is utilized to confirm joint distraction and the beginning of traction time is noted. The first portal is placed using anatomic landmarks and fluoroscopy assistance. A spinal needle is placed into the joint either from the anterolateral or posterolateral portal entry site. The negative pressure of the joint under traction will create an air arthrogram on fluoroscopy if the needle is within the joint. Other portals utilized include the accessory mid-anterior, direct anterior, and Dienst portals. The Dienst portal is located in the soft spot 1/3 of the way between the ASIS and the greater trochanter [26]. Typically, two to three portals are utilized. A flexible, blunt-tipped guide wire is inserted through the spinal needle, and its placement at the medial wall of the acetabulum is confirmed with fluoroscopy. The needle is removed over the guide wire, and a cannula with obturator is placed over the guide wire into the joint, taking care not to entrap the labrum. The position again is checked with fluoroscopy and the 70° arthroscope inserted in the scope cannula. The joint is insufflated with saline with the arthroscopic pump set at 45–60 mmHg, on average. The second portal is made under direct arthroscopic visualization within the central compartment, in a similar fashion to the first portal placement. Given the limitations in motion that often occur with SCFE, and postoperative changes after pinning, the joint capsules tend to be thick and stiff. Maneuverability within the joint is facilitated by establishing and extending an anterolateral capsulotomy with an arthroscopic beaver blade and shavers (Fig. 5a–c).

Central Compartment

A diagnostic arthroscopy of the central or articular compartment is performed. The second portal allows for probing of the labrum, acetabular and femoral articular cartilage, and ligamentum teres. Patients with large cam lesions secondary to chronic SCFE should have the integrity of the labrum and acetabular cartilage closely scrutinized. In the case of a labral tear, a third portal can be made if necessary as a distal accessory portal to facilitate an appropriate vector for anchor placement and suture passage. The labrum can be elevated from the acetabular rim sharply or recessed subperiosteally as described by Martin [27], theoretically preserving the blood supply to

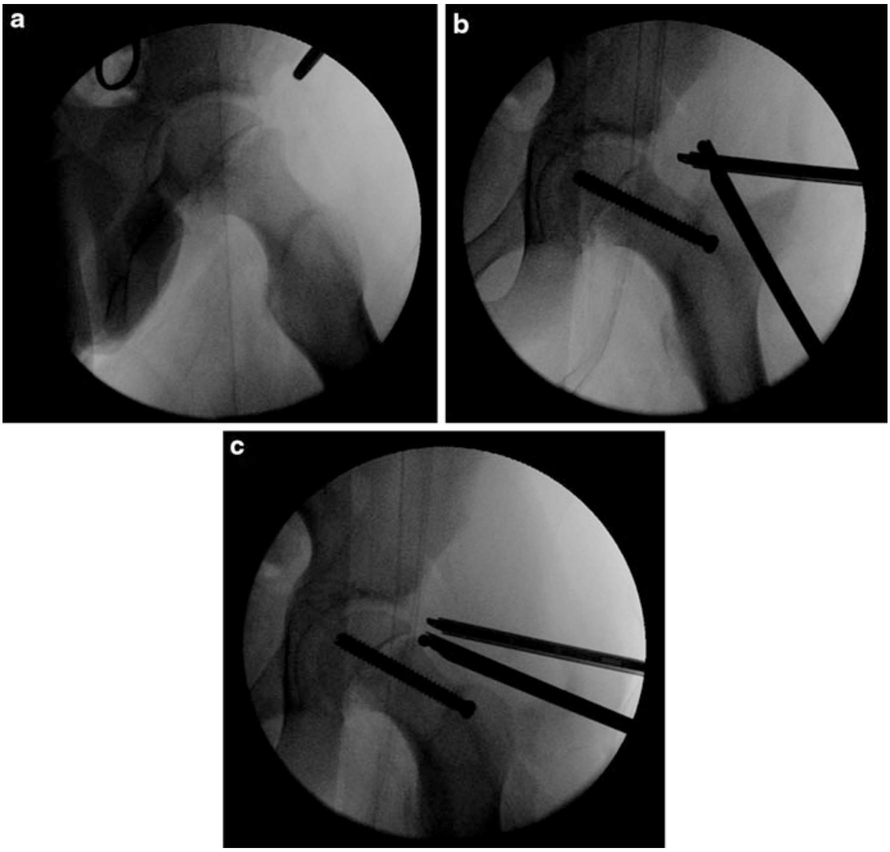


Fig. 5 Intraoperative fluoroscopy images illustrating a chronic SCFE slip with CAM lesion. (**a**) Frog leg images demonstrating a mild SCFE slip with CAM lesion prior to pinning and arthroplasty. (**b**) Pinned hip. (**c**) Pinned hip with femoroplasty

the labrum. If the structure of the labrum is preserved, yet it is separated from the chondrolabral junction, a repair is pursued rather than debridement. After preparing the acetabular rim creating a bleeding bony surface with an arthroscopic shaver or burr, enough anchors are placed to adequately reattach the torn portion. Nonabsorbable sutures from the anchors are passed either around the labrum encircling it or through the labrum in a mattress fashion and tied, reapproximating the labrum to the bleeding acetabular rim. Patients with post-SCFE impingement, whether resulting from untreated and healed slips or residual deformity after pinning, often develop articular cartilage changes. Acetabular cartilage delamination often accompanies labral damage at the anterosuperior acetabular articular margin and can be addressed with chondroplasty or microfracture.

Peripheral Compartment

Attention is then turned to the peripheral compartment where the offending cam lesion is addressed. With the arthroscope placed in the anterolateral portal, traction is removed; the hip is flexed to 45° and adducted while the arthroscope is maneuvered around the femoral head to view the femoral head-neck junction. Post-pinning patients often have capsular adhesions to the anterior femoral neck that limits maneuverability. The central compartment capsulotomy can be extended by performing a T-cut along the femoral neck to increase maneuverability in these cases. The position of the retinacular vessels supplying the femoral head when working in the peripheral compartment should be identified in order to avoid iatrogenic AVN. An MRI vascular study by Martin et al. confirmed that in 97 % of cases, these vessels are posterior to the 12 o'clock position of the femoral head. Lavigne et al. similarly showed that the majority of the vessels travel along the superior portion of the femoral neck, with 77 % found between the 9 and 2 o'clock positions [28]. The lateral synovial fold serves as an anatomic landmark for the posterior limit of safe cam lesion resection [29].

A 5.5 mm arthroscopic burr is then placed through the anterior portal in the region of abnormal femoral head-neck offset. Often, the presence of synovial herniation pits can direct the arthroscopist to the area of symptomatic impingement. Fluoroscopy is used to confirm the placement of the burr on the offending cam lesion. Recontouring of the proximal head-neck junction with gradual removal of the cam lesion and restoration of the offset is performed with frequent spot images taken to achieve adequate resection but prevent over-resection. The cam lesion is often at the level of the physis in younger, skeletally immature patients, and osteoplasty can be performed here safely (Fig. 6a–b). Biomechanical studies suggest up to 30 % of the anterosuperior femoral head-neck junction can be resected without increasing the risk for femoral neck fracture [30]. Increasing the degree of flexion allows for better visualization of the anterior femoral neck. Conversely, extension allows for better visualization posteriorly.

Prior SCFE fixation with a cannulated screw might present a problem when performing a proximal femoral osteoplasty as the screws are placed deep within the femoral neck. If removal of hardware is pursued, this can be performed prior to osteochondroplasty on the traction table with the patient in the supine position. Capsulotomies are closed when possible, especially for patients with generalized ligamentous laxity and/or any degree of dysplasia. Furthermore, if a T-shaped capsulotomy is created, this is routinely closed given its size and compromise of the iliofemoral ligament. This is performed with arthroscopic suture passers and a heavy, braided, absorbable suture.

Post Operative Care

Most patients are discharged on the day of surgery, though a minority stay overnight for pain control. All patients who have a proximal femoral osteochondroplasty are made touchdown weight bearing with crutches with a flat foot gait. Patients are kept on crutches for 2–3 weeks. Continuous passive motion (CPM) is generally utilized for 6–8 h a day for 3 weeks; unless microfracture was performed in which case the duration may be increased to 8 weeks. There have been no reported cases of deep vein thrombosis (DVT) or pulmonary embolism (PE) in pediatric patients undergoing hip arthroscopy, though there has been one reported fatal PE in the adult population. Patients who are 13 years old or older or any patient on oral contraception can be prescribed Aspirin 81 mg daily. Physical therapy is initiated early in the postoperative period, and the principles of rehabilitation described by Stalzer are

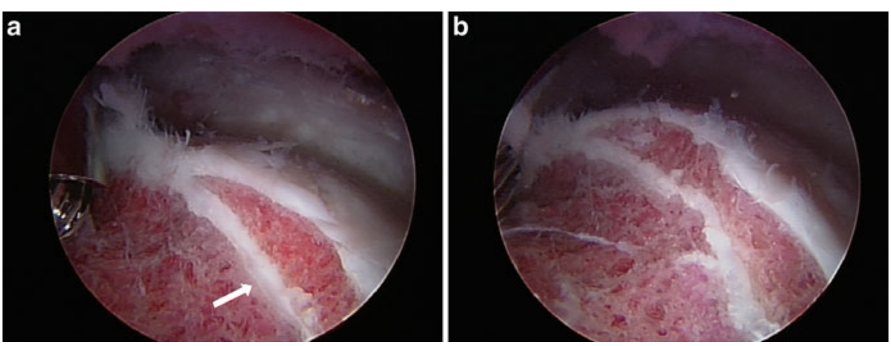


Fig. 6 (**a**) Intraoperative image following arthroscopic osteoplasty. The images demonstrate the close proximity of the cam lesion to the open femoral physis. The *white arrow* is pointing to the growth plate. (**b**) Final intraoperative arthroscopic view demonstrates the completed femoral osteoplasty

followed [31]. Patients undergo four phases of rehabilitation, first focusing on ROM exercises, then muscle endurance, strengthening, and eventual sport-specific exercises. Impact sports are limited for at least 4 months postoperatively.

Pearls and Pitfalls

- SCFE screw removal is recommended prior to attempted hip arthroscopy to prevent the phenomenon of the screw head tethering the capsule. Screw removal can be challenging with partially threaded, 7.3 mm cannulated screws.
- Because CAM morphology associated with SCFE may be different and/or more extensive than other forms of FAI, adequate osteoplasty/cam resection is essential, as studies have shown inadequate osteoplasty to be the most common technical error leading to poor outcomes requiring revision arthroscopy [32, 33].
- Because overweight patients are common in the SCFE population, additional portals may be necessary for optimization of access to different structures in the central compartment.
- Given the significant osteoplasty that may be required in an overweight SCFE patient, consideration should be given toward prolonged postoperative weight-bearing protection (e.g., ~6 week).

Outcomes

Reported outcomes in hip arthroscopy for the treatment of deformity associated with chronic SCFE are lacking as this is an emerging technique, though encouraging results from small case series have been reported. Lee reported outcomes for 5 patients with SCFE pinned in situ an average of 18 months prior to arthroscopic surgery [8]. All patients had clinical signs and symptoms of FAI, limitations in range of motion, and hip pain. All 5 patients were found to have some degree of chondral or labral damage at the time of surgery. The average alpha angle measured was >80° preoperatively. All patients underwent arthroscopic femoral osteoplasty and labral repair/debridement as indicated. Postoperatively, all patients had measured alpha angles less than 50°, resolution of their hip pain, and return to sports [8].

Though there is a paucity of reported outcomes for hip arthroscopy in the setting of chronic SCFE, there are many reports of clinical and radiographic outcomes for young patients with FAI treated with arthroscopic surgery. Philippon recently reported outcomes for children and adolescents who underwent arthroscopic surgery for FAI. 65 hips were treated and followed for an average of 3.5 years post-op. All patients had evidence of labral damage at the time of surgery; most underwent labral repair rather than debridement. At latest follow-up, the average improvement in Modified Harris Hip Scores (MHHS) was 34 points, from a mean of 57 pre-op to 91 post-op. The sport subset of the Hip Outcome Score (HOS) showed similar large gains after surgery. Patient satisfaction with the procedure was a median value of 10 out of 10 [34]. Other authors have reported similar improvements in outcomes and a rate of 80 % for return to full sporting activities in a similar cohort [35].

Summary

Slipped capital femoral epiphysis has long been known to be a precursor to arthrosis of the hip. Even mild slips (<30°) have a high potential to lead to chondral and labral damage. The offending lesion is the prominent proximal femoral metaphysis that displaces anteriorly as the femoral epiphysis separates forming a cam lesion. Alpha angles >60° have been associated with early degenerative changes noted on dGEMRIC imaging. Mild to moderate slips, and the resulting cam lesion that develops, can be addressed safely with arthroscopic femoral osteoplasty with a low complication rate and lower perioperative morbidity than open procedures. Severe slips (>60°) are best treated open. Early reports of arthroscopic femoral osteochondroplasty outcomes are encouraging for decreasing pain and increasing range of motion. Long-term outcome studies are needed to assess whether rates of arthrosis and eventual hip replacement are lower in these patients. Severe slips are likely best addressed by experienced hip surgeons facile in surgical hip dislocation and open osteoplasty or osteotomy.

References

1. Lehmann CL, et al. The epidemiology of slipped capital femoral epiphysis: an update. J Pediatr Orthop. 2006;26(3):286–90.
2. Wang Y. Disparities in pediatric obesity in the United States. Adv Nutr. 2011;2(1):23–31.
3. Webb AR, Kline L, Holick MF. Influence of season and latitude on the cutaneous synthesis of vitamin D3: exposure to winter sunlight in Boston and Edmonton will not promote vitamin D3 synthesis in human skin. J Clin Endocrinol Metab. 1988;67(2):373–8.
4. Kocher MS, et al. Delay in diagnosis of slipped capital femoral epiphysis. Pediatrics. 2004;113(4):e322–5.
5. Loder RT, et al. Acute slipped capital femoral epiphysis: the importance of physeal stability. J Bone Joint Surg Am. 1993;75(8):1134–40.
6. Johnston TL, et al. Relationship between offset angle alpha and hip chondral injury in femoroacetabular impingement. Arthroscopy. 2008;24(6):669–75.
7. Southwick WO. Osteotomy through the lesser trochanter for slipped capital femoral epiphysis. J Bone Joint Surg Am. 1967;49(5):807–35.
8. Lee CB, Matheney T, Yen YM. Case reports: acetabular damage after mild slipped capital femoral epiphysis. Clin Orthop Relat Res. 2013;471(7):2163–72.
9. Rab GT. The geometry of slipped capital femoral epiphysis: implications for movement, impingement, and corrective osteotomy. J Pediatr Orthop. 1999;19 (4):419–24.
10. Leunig M, et al. In situ pinning with arthroscopic osteoplasty for mild SCFE: a preliminary technical report. Clin Orthop Relat Res. 2010;468(12):3160–7.
11. Hosalkar HS, et al. Hip impingement in slipped capital femoral epiphysis: a changing perspective. J Child Orthop. 2012;6(3):161–72.
12. Millis MB, et al. Legg-Calve-Perthes disease and slipped capital femoral epiphysis: major developmental causes of femoroacetabular impingement. J Am Acad Orthop Surg. 2013;21 Suppl 1:S59–63.
13. Mamisch TC, et al. Femoral morphology due to impingement influences the range of motion in slipped capital femoral epiphysis. Clin Orthop Relat Res. 2009;467(3):692–8.
14. Hagglund G, et al. Slipped capital femoral epiphysis in southern Sweden. Long-term results after femoral neck osteotomy. Clin Orthop Relat Res. 1986;210:152–9.
15. Larson AN, et al. Outcomes of slipped capital femoral epiphysis treated with in situ pinning. J Pediatr Orthop. 2012;32(2):125–30.
16. Kocher MS, et al. Hip arthroscopy in children and adolescents. J Pediatr Orthop. 2005;25(5):680–6.
17. Futami T, et al. Arthroscopy for slipped capital femoral epiphysis. J Pediatr Orthop. 1992;12(5):592–7.
18. Byrd JW. Hip arthroscopy: patient assessment and indications. Instr Course Lect. 2003;52:711–9.
19. Zilkens C, et al. Delayed gadolinium-enhanced magnetic resonance imaging of cartilage (dGEMRIC), after slipped capital femoral epiphysis. Eur J Radiol. 2011;79(3):400–6.
20. Carter CW, Bixby S, Nasreddine AY, Yen Y-M, Kocher MS. The relationship between cam lesion and physis in skeletally immature patients. J Ped Orthop. 2014.
21. Siebenrock KA, et al. Abnormal extension of the femoral head epiphysis as a cause of cam impingement. Clin Orthop Relat Res. 2004;418:54–60.
22. Nwachukwu BU, et al. Complications of hip arthroscopy in children and adolescents. J Pediatr Orthop. 2011;31(3):227–31.
23. Papavasiliou AV, Bardakos NV. Complications of arthroscopic surgery of the hip. Bone Joint Res. 2012;1(7):131–44.
24. Ilizaliturri Jr VM. Complications of arthroscopic femoroacetabular impingement treatment: a review. Clin Orthop Relat Res. 2009;467(3):760–8.
25. Clarke MT, Arora A, Villar RN. Hip arthroscopy: complications in 1,054 cases. Clin Orthop Relat Res. 2003;406:84–8.
26. Dienst M, Seil R, Kohn DM. Safe arthroscopic access to the central compartment of the hip. Arthroscopy. 2005;21(12):1510–4.
27. Syed HM, Martin SD. Arthroscopic acetabular recession with chondrolabral preservation. Am J Orthop (Belle Mead NJ). 2013;42(4):181–4.
28. Lavigne M, et al. Distribution of vascular foramina around the femoral head and neck junction: relevance for conservative intracapsular procedures of the hip. Orthop Clin North Am. 2005;36(2):171–6. viii.
29. McCormick F, et al. Vascular safe zones in hip arthroscopy. Am J Sports Med. 2011;39(Suppl):64S–71.
30. Mardones RM, et al. Surgical treatment of femoroacetabular impingement: evaluation of the effect of the size of the resection. J Bone Joint Surg Am. 2005;87(2):273–9.
31. Stalzer S, Wahoff M, Scanlan M. Rehabilitation following hip arthroscopy. Clin Sports Med. 2006;25 (2):337–57. x.
32. Heyworth BE, et al. Radiologic and intraoperative findings in revision hip arthroscopy. Arthroscopy. 2007;23(12):1295–302.
33. Philippon MJ, et al. Revision hip arthroscopy. Am J Sports Med. 2007;35(11):1918–21.
34. Philippon MJ, et al. Outcomes 2 to 5 years following hip arthroscopy for femoroacetabular impingement in the patient aged 11 to 16 years. Arthroscopy. 2012;28 (9):1255–61.
35. Tran P, Pritchard M, O'Donnell J. Outcome of arthroscopic treatment for cam type femoroacetabular impingement in adolescents. ANZ J Surg. 2013;83 (5):382–6.

38 Neuromuscular Hip Disorders: Focus on Cerebral Palsy

Michael D. Hellman, Leon Root, and Monica Kogan

Contents

Abstract

Cerebral palsy (CP) is the most frequent cause of chronic orthopedic disorders in children. Hip dysplasia occurs secondary to delayed walking and excessive muscle tone. The hip flexors and adductors overpower the hip extensors and abductors causing the femoral head to pull out of socket, most commonly in a posterolateral direction. Children with CP should be evaluated on a regular basis – both clinically and radiographically. Walking by 30 months significantly lowers the child's risk of hip problems. An anteroposterior pelvis radiograph is used to assess for hip subluxation. A young patient with an at-risk hip (Migration Index <30 %) should undergo a soft-tissue operation. If subluxation is significant, a varus derotation osteotomy is indicated along with a soft-tissue operation. If the acetabulum is dysplastic, then an acetabuloplasty is also indicated. For chronic non-reducible painful hips, a salvage operation should be considered.

Introduction

Muscle paralysis often leads to hip instability in young growing children. The paralysis may be spastic or athetoid (dystonic) as occurs in cerebral palsy (CP), or flaccid paralysis in poliomyelitis or meningomyelocele. Since a vaccine has prevented polio and meningocele occurrence is decreasing,

M.D. Hellman (✉)
Department of Orthopaedic Surgery, Rush University Medical Center, Chicago, IL, USA
e-mail: mdhellman@gmail.com

L. Root
Hospital for Special Surgery, New York, NY, USA
e-mail: rootl@hss.edu

M. Kogan
Department of Orthopaedic Surgery, Rush University Medical Center, Chicago, IL, USA

Pediatric Orthopaedic Surgery, Rush University Medical Center, Chicago, IL, USA
e-mail: mkogan@rushortho.com

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_42

this discussion shall concentrate on the etiology and description of hip instability in the CP population.

Epidemiology and Classification

Cerebral palsy is the most frequent cause of chronic orthopedic disorders in children and can be attributed to perinatal abnormalities, the most common of which is prematurity and low birth weight (under 2,500 g). The incidence of CP is approximately 1–3 per 1,000 live births [1]. Fifteen percent of infants born under 2,500 g develop CP, and the incidence goes up 100-fold compared to terminal deliveries [1].

Cerebral palsy is usually described by topographical involvement: hemiplegia (involvement of only one side of the body), diplegia (symmetric lower extremity involvement), triplegia (involvement of both lower extremities and one upper extremity), or quadriplegia (total body involvement). The neurological consequence is also described and can be spasticity, athetosis (dystonia), or ataxia. The Gross Motor Function Classification System (GMFCS) helps categorize patients with CP: Level 1, walks without limitation; Level 2, walks with limitations; Level 3, walks using handheld mobility device; Level 4, self-mobility with limitations, may require a wheelchair, and able to control neck; and Level 5, transported in a wheelchair, without neck control [2].

The more involved the patient, the more likely they will develop hip instability. Hip abnormalities are approximately 7 % in independent ambulators and up to 60 % of non-independent sitters [3]. Soo and colleagues found that hip dysplasia was present in 0 % of children with GMFCS Level 1 and 90 % for those with GMFCS Level 5 [4]. Therefore, the more severely involved person must be followed very carefully for hip problems.

Pathogenesis

Children with cerebral palsy have normal hip joints at birth. Hip subluxation/dislocation is acquired secondary to delayed weight bearing and excessive muscle tone [5]. Unlike classic hip dysplasia, a subluxated CP hip does not have laxity or gross instability. A delay or complete lack of ambulation leads to persistent neonatal femoral anteversion and a delay in acetabular development [5]. Further, the muscular forces across the hip are up to six times greater than normal which causes it to be stiff and overtime forcefully pulls it out of socket [6]. Both the femur and the acetabulum are adversely affected. The femur is overpowered by the hip adductors and hip flexors and driven into the posterolateral acetabulum and labrum. This causes a dunce-cap deformation of the femoral head between the lateral acetabular margin and the hip capsule [7]. Ultimately this causes the femur to become irreducible and destroys the articular cartilage [8]. The acetabulum usually begins to show radiographic changes at about 30 months [9]. A defect in the acetabulum is formed by constant irregular contact of the femur eventually leading to acetabular insufficiency [10].

Clinical Evaluation

A child with cerebral palsy should have regular comprehensive clinical evaluations. The initial assessment includes a thorough history and physical examination. Important information includes the patient's birth history such as weight, gestational age at time of birth, and complications after birth; the patient's motor milestones history such as head control, sitting, standing, cruising, and walking; the patient's preference in side such as left- or right-handedness; and the patient's history on any associated conditions such as speech development disorders and seizure disorders [11]. Ambulatory patients should be asked about their walking status. Children that walk by 30 months are at low risk of developing hip problems [12]. Delayed standing/walking or changes in standing/walking are signs of possible hip abnormalities. Non-ambulatory patients should be asked about comfort and pain, sitting, contractures, and hygiene issues. Family and caregivers are always very helpful at identifying problems and should be an integral part of every evaluation.

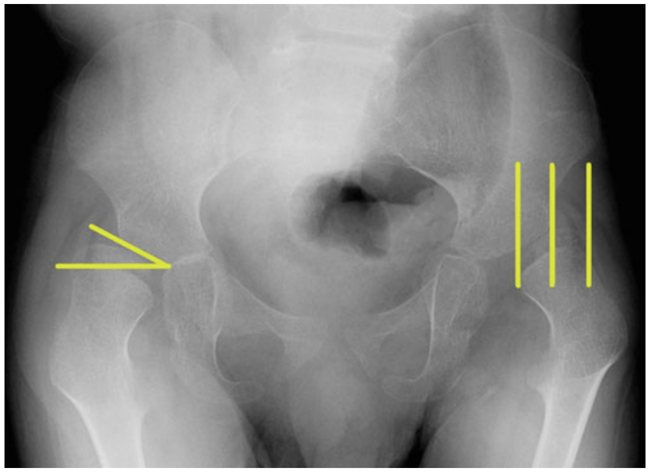


Fig. 1 Acetabular Index (right hip) is the angle between Hilgenreiner's line and a line drawn from the triradiate epiphysis to the lateral edge of the acetabulum. Reimers' Migration Index (left hip) is the percent of the transverse diameter of the femoral head that lies lateral to Perkin's line

Physical examination includes testing hip abduction in flexion and extension. The patient may show a positive Galeazzi sign secondary to hip dislocation [13]. With the hips in flexion, passive abduction less than 40° should alert the examiner to hip problems [12]. The examiner should also focus on the knees, ankles, and spine. Non-ambulatory patients should be assessed both on the exam table and in their wheelchair [8].

Radiologic Evaluation

A standard supine anteroposterior pelvic radiograph is used to assess for hip subluxation/dislocation in children with cerebral palsy. Obtaining properly positioned radiographs may be challenging secondary to pelvic obliquity, lumbar lordosis, and muscle contractures. Flexing the contralateral hip and knee while actively positioning the hip of interest into a neutral position (abduction/adduction) will allow for more accurate measurements [8, 14]. Fluoroscopy may be the only way to obtain an adequate radiograph in a patient with very high muscle tone [15].

Subluxation of the hip can be first seen as a break in Shenton's line. Shenton described a line formed by the upper margin of the obturator foramen and the inner margin of the femoral neck. A break in the continuity of this line indicates subluxation/dislocation of the hip. Reimers' Migration Index (MI) and the Acetabular Index (AI) are the two most useful measurements to assess for degree of hip subluxation. Reimers' Migration Index is the percent of the transverse diameter of the femoral head that lies lateral to Perkin's line (see Fig. 1) [14]. Acetabular Index is the angle between Hilgenreiner's line and a line drawn from the triradiate epiphysis to the lateral edge of the acetabulum (see Fig. 1). There is a strong correlation between AI and MI – AI gradually increases as MI increases [5]. The neck-shaft angle and the femoral head to teardrop distance should also be assessed. An elevated neck shaft angle represents coxa valga and increased femoral anteversion [5].

Both Reimers' Migration Index and the Acetabular Index are reproducible measurements. The experienced physician is able to measure the migration percentage within 5.8 % of its true value and measure the Acetabular Index within 2.6° of its true value [16]. In normal children, the 90th percentile for Migration Index is 10 % [14]. An MI greater than or equal to 33 % is recommended as a threshold for intervention or at least more intensified observation. Hips with an MI greater than or equal to 40 % have a very high risk for progressive displacement indicating the need for a surgical procedure at that threshold [15]. An Acetabular Index of greater than 30° in children greater than or equal to 4 years old is a good predictor for progressive acetabular insufficiency [17].

There is no consensus on how often to perform hip surveillance. Gordon and colleagues performed a systematic review and found six articles discussing hip surveillance protocols in hip patients with CP [18]. All patients with bilateral cerebral palsy should have a screening pelvic radiograph by 30 months. Acetabular Index and Reimers' Migration Index are useful measurements, and a migration percentage of 15 % or more needs careful monitoring. Hips with an MI greater than or equal to 60 % require immediate attention. Any hip with an AI greater than or equal to 30° or an MI greater than 33 % will likely require further intervention. Screening radiographs should happen at least on an annual basis, and any progression of MI greater than 7 % requires very close monitoring [18].

Treatment

Surgical treatment can be divided into three categories:

1. **The at-risk hip**: In the young child before subluxation occurs.
2. **Hip subluxation without acetabular dysplasia**: Hip muscle imbalance is present, subluxation has occurred, but the acetabulum appears normal.
3. **Hip subluxation with acetabular dysplasia**: Hip subluxation/dislocation is present with acetabular dysplasia.

The At-Risk Hip

The "at-risk hip" is thought to be in children less than five with a hip that has significant adduction and flexion contractures, minimal subluxation, and a Migration Index less than 30 % [19]. Early treatment is thought to be mandatory to prevent progression of subluxation. Almost always, bilateral surgery is indicated in the setting of spasticity even if only one hip is showing signs of early subluxation [20, 21]. Hip flexor releases include tenotomy of the rectus femoris origin, the tensor fasciae latae, and a release of the psoas tendon over the brim of the pelvis. Adductor tenotomy, usually just the longus and brevis, may also be necessary. When the hamstrings are also contracted as demonstrated by decreased straight leg raise and a decreased popliteal angle, distal hamstring lengthening is also indicated. Muscles are usually released until 50° of symmetric hip abduction is obtained. Infection and hematoma are the two most common complications of this procedure and are relatively rare [22]. Results of these procedures are fairly good. Silver and colleagues reported only 20 % of hips progressed to subluxation [23]. Cornell and colleagues found that 83 % of patients who underwent adductor tenotomies had hips that remained stable [24]. It is clear that the degree of subluxation of the hip at the time of tenotomy plays a big role in the outcome of soft-tissue-only procedures, and so strict indications for this treatment group should not be compromised [25].

Hip Subluxation Without Acetabular Dysplasia

When muscle imbalance persists and subluxation is present, a varus derotation osteotomy (VDRO) of the hip and appropriate muscle releases are necessary. A varus closing wedge osteotomy is made at the intertrochanteric level, and usually a 90° blade plate is used for internal fixation. Neck-shaft angle should be corrected to 90–100° of varus [26]. Concurrent tenotomies are performed to balance the forces around the hip joint. The most common complications of a VDRO are loss of fixation and fracture [8]. Many patients develop prominence of hardware and removal is frequent [11]. Brunner and Baumann noted that children less than 4 years old lost varus correction 96 % of the time and recommended a delay in surgery if possible until 8–10 years old [27]. Hoffer and colleagues reported on VDRO outcomes and concluded that it was a good procedure for hip subluxation but was inadequate to treat CP hip dysplasia [28]. Tylkowski and colleagues were able to keep reduction of 16/18 hips after a 3-year follow-up [29]. Overall, a VDRO used in concert with other procedures adequately reorients the center of the hip away from the lesser trochanter and tips the femoral head into the acetabulum.

Hip Subluxation with Acetabular Dysplasia

In addition to the muscle procedures and a VDRO, acetabular insufficiency must be addressed by an osteotomy, either a periacetabular (e.g., Dega or Ganz) or an innominate osteotomy (e.g. Salter). There are more complications associated with these surgeries. Postoperative fracture, pathologic fracture of the femur, pulmonary complications, and decubitus ulcers have all been reported [11]. Dietz and Knutson found that 79 % of hips undergoing a Chiari-type pelvic osteotomy were completely joint pain-free at 7-year follow-up [30]. Further, Osterkamp and colleagues reported that only two of nine hips redislocated after an acetabular osteotomy [31]. Shelf procedures in general appear to do well in patients with CP hip dysplasia. Overall, hip stability was obtained in 83–95 % of patients that underwent a shelf type acetabular osteotomy [32]. When the femoral head and acetabulum are deformed to the point that they are nonreconstructable, which occurs over time when the head is no longer protected in the acetabulum, reducing it into the acetabulum becomes more complex. Painful dislocated hips in the adult or young adult may require a Castle procedure (hip resection), a Schantz osteotomy (valgus osteotomy), a total hip replacement, or a hip fusion. These procedures are salvage operations that should be reserved only for patients with severely deformed, irreducible, and painful hips.

Summary

Hip subluxation/dislocation in the CP patient is an acquired condition and therefore preventable. The most severely involved child is at greatest risk. Frequent clinical and radiographic observation is essential. Before subluxation, the at-risk hip can be treated with muscle releases, but once subluxation occurs, VDRO and possible acetabular procedures are required to provide a stable and painless hip.

References

1. Washburn LK, Dillard RG, Goldstein DJ, Klinepeter KL, deRegnier RA, O'Shea TM. Survival and major neurodevelopmental impairment in extremely low gestational age newborns born 1990–2000: a retrospective cohort study. BMC Pediatr. 2007;7:20.
2. Palisano RJ, Cameron D, Rosenbaum PL, et al. Stability of the gross motor function classification system. Dev Med Child Neurol. 2006;48:424e8.
3. Lonstein JE, Beck RPT. Hip dislocation and subluxation in cerebral palsy. J Pediatr Orthop. 1986;6(5):521–6.
4. Soo B, Howard JJ, Boyd RN, et al. Hip displacement in cerebral palsy. J Bone Joint Surg Am. 2006;88:121–9.
5. Beals RK. Developmental changes in the femur and acetabulum in spastic paraplegia and diplegia. Dev Med Child Neurol. 1969;11:303–13.
6. Miller F, Slomczkowski M, Cope R, et al. Computer modeling of the pathomechanics of spastic hip dislocation in children. J Pediatr Orthop. 1999;19:486.
7. Abel MF, Wenger DR, Mubarak SJ, Sutherland DH. Quantitative analysis of hip dysplasia in cerebral palsy: a study of radiographs and 3-D reformatted images. J Pediatr Orthop. 1994;l14:283–9.
8. Flynn JM, Miller F. Management of hip disorders in patients with cerebral palsy. J Am Acad Orthop Surg. 2002;10:198–209.
9. Cornell MS. The hip in cerebral palsy. Dev Med Child Neurol. 1995;37:3–18.
10. Brunner R, Picard C, Robb J. Morphology of the acetabulum in hip dislocations caused by cerebral palsy. J Pediatr Orthop B. 1997;6:207–11.
11. Herring JA. Tachdjian's pediatric orthopaedics. 4th ed. Philadelphia: Saunders; 2008.
12. Scrutton D, Baird G, Smeeton N. Hip dysplasia in bilateral cerebral palsy: incidence and natural history in children 18 months to 5 years. Dev Med Child Neurol. 2001;43:586–600.
13. Nwaobi OM, Sussman MD. Electromyographic and force patterns of cerebral palsy patients with windblown hip deformity. L Pediatr Orthop. 1990;10:382–8.
14. Reimers J. Stability of the hip in children: a radiological study of results of muscle surgery in cerebral palsy. Acta Orthop Scand Suppl. 1980;184:1–100.
15. Hagglund G, Lauge-Pedersen H, Persson M. Radiographic threshold values for hip screening in cerebral palsy. J Child Orthop. 2007;1:43–7.
16. Parrott J, Boyd RN, Dobson F, Lancaster A, Love S, Oates J, Wolfe R, Nattrass GR, Graham HK.

Hip displacement in spastic cerebral palsy: repeatability of radiologic measurement. J Pediatr Orthop. 2002;22:660–7.
17. Cooke PH, Cole WG, Carey RP. Dislocation of the hip in cerebral palsy: natural history and predictability. J Bone Joint Surg Br. 1989;71:441–6.
18. Gordon GS, Simkiss DE. A systematic review of the evidence for hip surveillance in children with cerebral palsy. J Bone Joint Surg [Br]. 2006;88-B:1492–6.
19. Kalan V, Conklin MM, Sherman FC. Prevention of spastic paralytic dislocation of the hip. Dev Med Child Neurol. 1985;27:17.
20. Carr C, Gage JR. The fate of the nonoperated hip in cerebral palsy. J Pediatr Orthop. 1987;7(3):262–7.
21. Noonan KJ, Walker TL, Kayes KJ, et al. Effect of surgery on the nontreated hip in severe cerebral palsy. J Pediatr Orthop. 2000;20(6):771–5.
22. Root L, Spero CR. Hip adductor transfer compared with adductor tenotomy in cerebral palsy. J Bone Joint Surg. 1981;63-A:1133.
23. Silver RL, Rang M, Chan J, et al. Adductor release in nonambulant children with cerebral palsy. J Pediatr Orthop. 1985;5:672.
24. Cornell MS, Hatrick NC, Boyd R, et al. The hip in children with cerebral palsy: predicting the outcome of soft tissue surgery. Clin Orthop. 1997;340:165.
25. Miller F, Cardoso DR, Dabney KW, et al. Soft-tissue release for spastic hip subluxation in cerebral palsy. J Pediatr Orthop. 1997;17:571.
26. Eilert RE, MacEwen GD. Varus derotational osteotomy of the femur in cerebral palsy. Clin Orthop. 1977;125:168.
27. Brunner R, Baumann JU. Long-term effects of intertrochanteric varus-derotation osteotomy on femur and acetabulum in spastic cerebral palsy: an 11- to 18-year follow-up study. J Pediatr Orthop. 1997;17:585–91.
28. Hoffer MM, Stein GA, Koggman M, et al. Femoral varus-derotation osteotomy in spastic cerebral palsy. J Bone Joint Surg. 1985;67-A:1229.
29. Tylkowski CM, Rosenthal RK, Simon SR. Proximal femoral osteotomy in cerebral palsy. Clin Orthop. 1980;151:183.
30. Dietz FR, Knutson LM. Chiari pelvic osteotomy in cerebral palsy. J Pediatr Orthop. 1995;15:372.
31. Osterkamp J, Caillouette JT, Hoffer MM. Chiari osteotomy in cerebral palsy. J Pediatr Orthop. 1988;8:274.
32. McCall RE, Lillich JS, Harris JR, et al. The Grice extraarticular subtalar arthrodesis: a clinical review. J Pediatr Orthop. 1985;5:442.

Surgical Technique: Tendon Releases

39

Joel Kolmodin, Keith Bachmann, and Ryan Goodwin

Contents

Abstract

Adductor and hip flexor releases are commonly used to treat pathologic hip contractures about the hip in children with neuromuscular conditions such as cerebral palsy. Surgery is performed for a variety of reasons, including efforts to avoid hip subluxation or dislocation, to aid in patient positioning, or to address abnormalities in gait. Psoas tendon releases are typically performed at the pelvic brim, though they occur at the lesser trochanter in some circumstances. Adductor tendon releases occur at their origins, and they can involve a solitary tendon or numerous tendons, depending on the degree of contracture severity. Depending on the approach, caution must be taken to avoid injury to the femoral neurovascular bundle, the obturator nerve, or the lateral femoral cutaneous nerve. Postoperative immobilization focuses on maintaining an appropriate degree of normal hip extension and abduction, usually through the use of casting and intermittent stretching. Complications are few but can involve wound infection, neurovascular injury, excessive hip flexor or adductor weakness, and early contracture recurrence.

Introduction

Tendon releases are commonly employed as reliable methods to correct pathologic contractures about the hip joint in patients with neuromuscular

J. Kolmodin (✉) • K. Bachmann • R. Goodwin
Cleveland Clinic Foundation, Cleveland, OH, USA
e-mail: kolmodj@ccf.org; bachmak@ccf.org; goodwir@ccf.org

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_43

conditions, most commonly in the patient with cerebral palsy. Depending on the age of the patient and the severity of their hypertonicity, contracture can be very debilitating and deleterious to their care. Primary contractures are those of the iliopsoas and adductor muscles, causing pathologic flexion and adduction deformities at the hip joint. Thus, in an effort to directly address these pathologies, numerous approaches to the hip are used to access the psoas and adductor tendons. Handling of the tendons involves complete tenotomy, musculotendinous recession, or tendon lengthening. This chapter will focus on the anatomy surrounding psoas and adductor tendon releases, a utilitarian surgical approach to the adductor and psoas tendons, postoperative management, and complications.

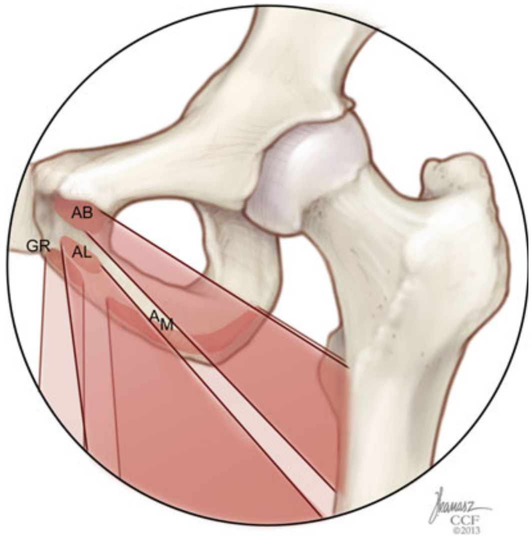


Fig. 1 Origins of the hip adductor muscles (Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography © 2013. All rights reserved)

Anatomic Considerations

The psoas major muscle takes its origin from the transverse processes and vertebral bodies of T12-L5, as well as their associated intervertebral disks. Coursing through the pelvis, the psoas muscle joins the iliacus muscle at approximately the level of the inguinal ligament. The psoas tendon crosses the hip joint anteriorly and inserts onto the lesser trochanter of the femur. The psoas muscle is innervated by the L1–L4 roots of the lumbar plexus.

The iliacus muscle originates in a broad fashion from the iliac fossa. As noted above, it joins the psoas muscle at the level of the inguinal ligament. The fibers of the iliacus muscle are located anterior to the psoas tendon and insert into the lesser rochanter, though some fibers extend further distal.

The adductor muscles (adductor longus, adductor brevis, adductor magnus, gracilis, pectineus) originate from various sites along the inferior pubic ramus (Fig. 1). The majority of the adductors insert at the linea aspera of the femur, the primary exception being the gracilis, which is a component of the pes anserinus and inserts into the proximal tibia. All of the adductor muscles are innervated by the obturator nerve (L2–L4), with the exception of the pectineus, which is innervated by the femoral nerve.

Numerous nerves and other vascular structures are at risk in dissection to perform tendon releases about the hip joint. Risk for their injury during dissection has been well documented [1]. The femoral nerve, artery, and vein lie rather superficially in the interval between the sartorius and the adductor longus/pectineus. The lateral femoral cutaneous nerve runs deep to the inguinal ligament at a point 2–3 cm medial to the ASIS. The two main branches of the obturator nerve surround the adductor brevis muscle in the groin. The anterior branch runs along the anterior portion of the adductor brevis, while the posterior branch runs posteriorly.

Surgical Management

Decisions to intervene surgically are patient specific, taking into consideration goals of treatment and function. In some cases, tendon releases about the hip joints are undertaken to avoid hip subluxation or dislocation, such as a young child with progressive subluxation. In other instances, tendon releases are performed to aid in ambulation. Some releases, such as complete psoas release from the lesser trochanter, are contraindicated in ambulatory patients. Regardless of the surgical indication, a thorough physical examination is required preoperatively to verify the character and extent of adduction or flexion contracture at the hip joint.

Routine preoperative planning usually involves supine AP radiographs (neutral/abduction), with standing radiographs obtained if patient function allows. In ambulatory patients, preoperative gait analysis can be extremely beneficial as well, giving insight into the pathologic portions of the patient's gait. It is also recommended that a thorough examination under anesthesia be performed prior to incision. At this point, while the patient is fully anesthetized and spasticity is minimized, it is possible to differentiate between decreased range of hip motion that is caused by abnormal muscle tone and that which is caused by true musculotendinous contracture (i.e., Thomas test).

Open Adductor Lengthening and Psoas Release

The patient is placed in a supine position, with the operative leg isolated so that the hip can be ranged freely. A 2–3 cm transverse incision is made in the groin crease directly overlying the adductor longus tendon (Fig. 2). This is made 1 cm distal to the groin crease. After isolating the adductor longus tendon through careful dissection, a right-angle clamp is passed around the tendon, separating it from the underlying adductor brevis muscle. Using electrocautery, the tendon is divided at the most proximal point possible. The adductor brevis muscle can then be isolated and divided in a similar manner. Care should be taken to avoid the anterior branch of the obturator nerve, which runs along the anterior portion of the adductor brevis, and the posterior branch of the obturator nerve, which lies posterior to the adductor brevis (Fig. 3). If further release is indicated, the gracilis can be divided. This muscle is found posterior to the adductor brevis, and it tends to be much smaller and flatter in nature. If psoas release is desired, the pectineus can be identified at this point. Using cautious blunt dissection, the interval between the pectineus and neurovascular bundle is developed, revealing the underlying psoas tendon. Exposure of the psoas tendon involves dissection around the medial femoral circumflex artery (which is often ligated) and removal of a fatty collection that invariably surrounds the tendon. The psoas tendon can then be released from the lesser trochanter [2].

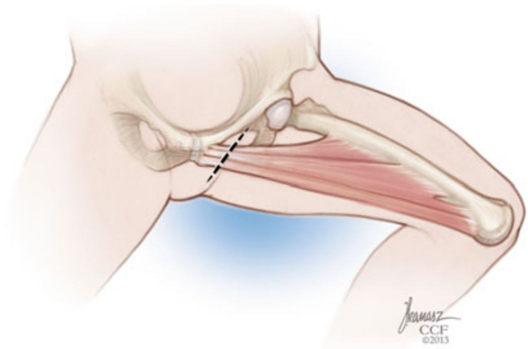


Fig. 2 Transverse incision for open procedure (Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography © 2013. All rights reserved)

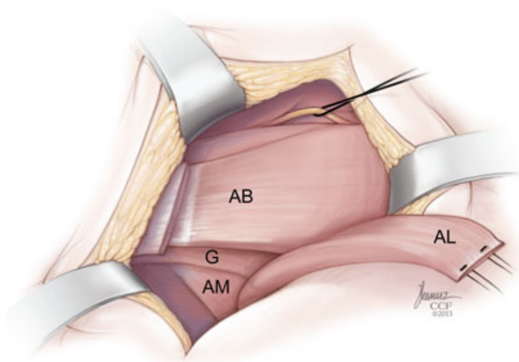


Fig. 3 Division of the adductor longus and exposure of the adductor brevis (Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography © 2013. All rights reserved)

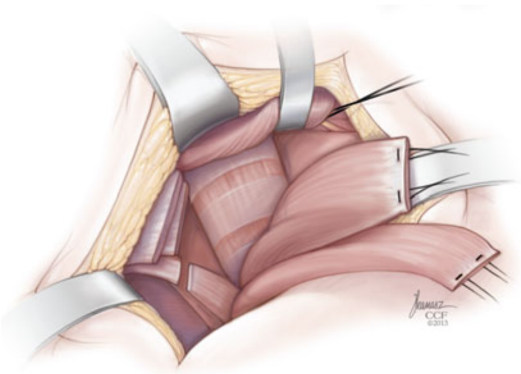


Fig. 4 Division of the adductor brevis and recession of the psoas tendon (Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography © 2013. All rights reserved)

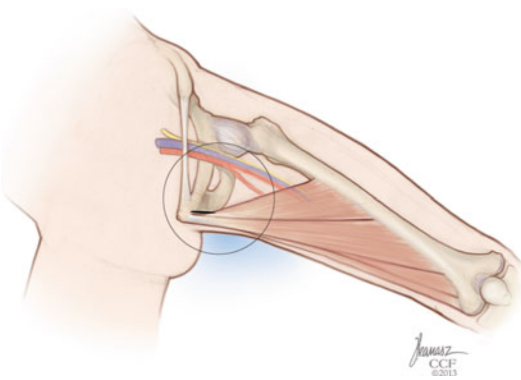


Fig. 5 Location of the percutaneous procedure (Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography © 2013. All rights reserved)

In addition, a musculotendinous recession can be carried out from this approach, as a full release is not recommended in an ambulatory child [3, 4] (Fig. 4).

Percutaneous Adductor Lengthening

The patient is placed in a supine position. The surgeon isolates the adductor longus origin between his or her thumb and index finger. A number 15 blade is introduced horizontally underneath the tendon [5] (Figs. 5, 6). Turning the

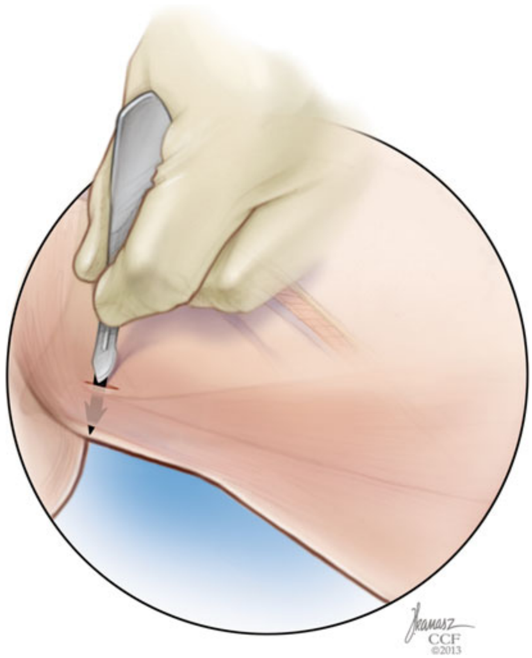


Fig. 6 The scalpel is inserted in a horizontal manner from lateral to medial, immediately deep to the adductor longus tendon (Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography © 2013. All rights reserved)

blade 90°, the surgeon cuts the adductor longus tendon as close to its origin as is possible (Fig. 7). The cut generally moves from deep to superficial, and it occurs away from the neurovascular bundle (lateral to medial). Remaining fibers are then divided by tactile movements of the knife blade against the firm residual tendon fibers (Figs. 8, 9). The knife blade is then turned 90° once again and withdrawn from the wound; in this way a "T" wound is not made as the blade is removed.

Postoperative Care

Immobilization is typically recommended after tendon releases about the hip in this patient population. The most useful mode of immobilization is usually bilateral long-leg casts separated by an abduction bar. Correction of hip flexion contracture is achieved postoperatively by intermittent passive hip extension exercises by a caregiver or by placing the child in the prone position

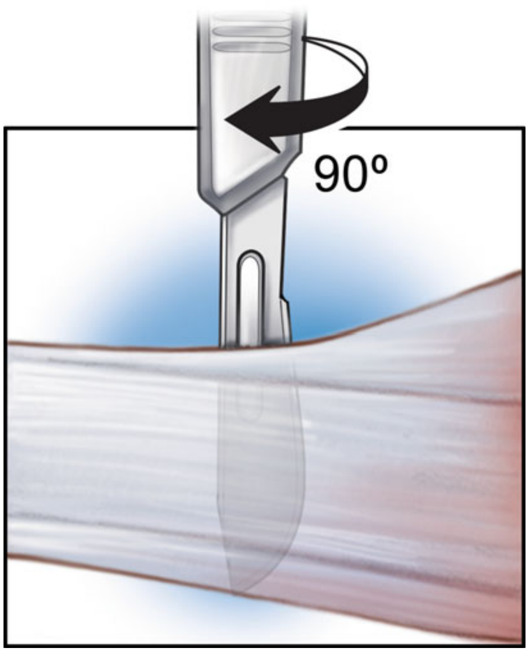


Fig. 7 The scalpel blade is rotated 90°, with the blade now pointing vertically (Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography © 2013. All rights reserved)

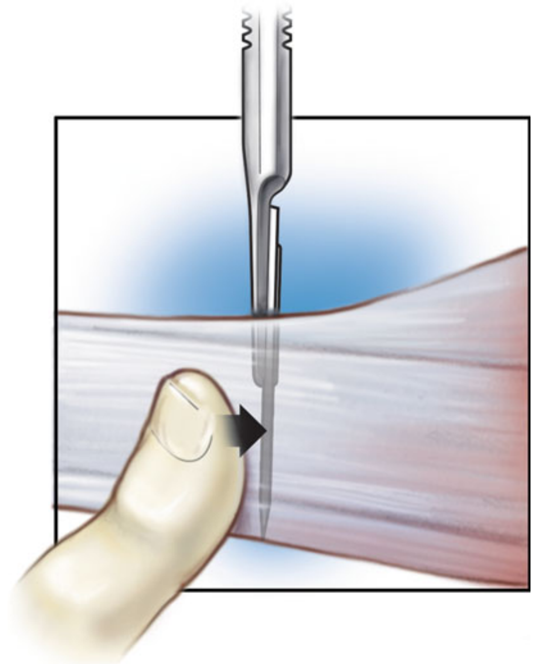


Fig. 8 As firm opposing pressure is applied by the surgeon's contralateral thumb, the scalpel blade is advanced through the tendon (Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography © 2013. All rights reserved)

frequently throughout the day. It is encouraged that the abduction bar be removed at regular intervals to perform hip range-of-motion exercises.

Complications

Complications of tendon releases about the hip are not common. Though not an overt complication, early recurrence of the flexion-adduction contracture is seen. This can necessitate further, more aggressive surgical intervention. As emphasized prior, there is a well-documented risk for excessive hip flexor weakness with complete tenotomy of the psoas tendon at the lesser trochanter. This is especially important to avoid in ambulatory patients. There is also a risk for pelvifemoral instability if adductor release is excessive or used inappropriately. Additionally, risk for neurovascular injury exists, most importantly of the femoral neurovascular bundle. Finally, wound drainage or dehiscence occurs with some frequency. This is due to the fact that

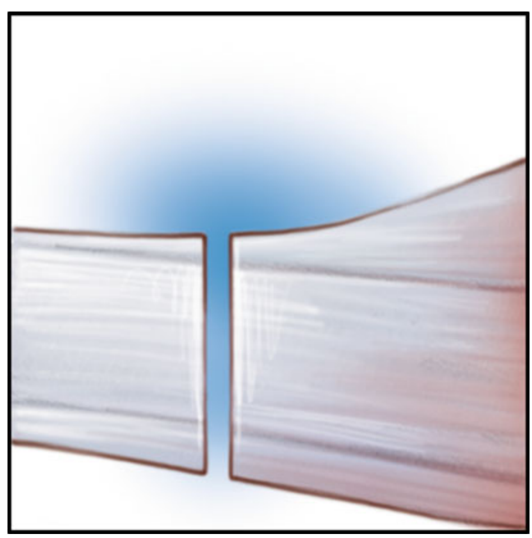


Fig. 9 With the tendon divided, the scalpel blade is rotated 90° and withdrawn from the wound (Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography © 2013. All rights reserved)

incisions lie within the groin crease, and proper wound care can be challenging in this patient population. Wound infection, however, is not common due to the abundant blood supply to this area.

Summary

Tendon releases about the hip joint remain a mainstay of treatment for flexion and adduction contractures in neuromuscular conditions, most notably in cerebral palsy. Understanding patient and caregiver goals for function can help to determine the appropriate surgical intervention, and a thorough physical examination gives great insight into pathologies that cause deformity or limitation. Psoas or adductor tendon releases can be performed in a variety of manners and via a number of approaches. Safe and reliable correction of abnormal hip contractures can be obtained.

References

1. Skaggs DL, et al. Psoas over the brim lengthenings: anatomic investigation and surgical technique. Clin Orthop Relat Res. 1997;339:174–9.
2. Poonnoose PM, et al. The anteromedial approach to the psoas tendon in patients with cerebral palsy. J Child Orthop. 2007;1:249–52.
3. Presedo A, et al. Soft-tissue releases to treat spastic hip subluxation in children with cerebral palsy. J Bone Joint Surg Am. 2005;87A(4):832–41.
4. Bleck E. Postural and gait abnormalities caused by hip-flexion deformity in spastic cerebral palsy: treatment by iliopsoas recession. J Bone Joint Surg Am. 1971;53A:1468–88.
5. Hage S, et al. Is percutaneous adductor tenotomy as effective and safe as the open procedure? J Pediatr Orthop. 2010;30:485–8.

Surgical Technique: Reconstruction with a Proximal Varus Derotational Osteotomy with Blade Plate Fixation and a Volume Reducing Pelvic Osteotomy for Neuromuscular Hip Dysplasia

40

Keith Baldwin and David Spiegel

Contents

K. Baldwin (✉)
Children's Hospital of Philadelphia, Philadelphia, PA, USA
e-mail: baldwink@email.chop.edu

D. Spiegel
Division of Orthopaedic Surgery, Children's Hospital of Philadelphia, Philadelphia, PA, USA
e-mail: spiegeld@email.chop.edu

Abstract

Neuromuscular hip dysplasia is a disorder of the hip joint caused by abnormal pull of spastic hip flexor and adductor muscles. It is common in children with cerebral palsy, and the incidence and severity increases with increasing neurologic involvement. Goals of management include maintaining a mobile painless located hip. The varus derotational osteotomy and volume-reducing pelvic osteotomy are the workhorses of reconstruction of this disorder. This chapter describes the technique of management of the reconstructible neuromuscular hip with blade plate fixation.

Introduction

Hip dysplasia is a common problem in cerebral palsy and other neuromuscular disorders. In cerebral palsy, the prevalence of hip displacement is related to the degree of neuromuscular impairment. The average migration percentage varies from 8.1 % GMFCS I to 46.2 % in GMFCS V patients [1]. Lonstein and Beck found hip subluxation and dislocation in 7 % of ambulators but 60 % of dependent sitters [2]. Pathogenesis of hip displacement involves dynamic imbalance between the muscles of flexion and adduction, with dynamic imbalance resulting in myostatic contracture over time. In the setting of fixed adduction and flexion deformity, ongoing muscle activity promotes gradual superior and posterior

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_44

Fig. 1 Chronic dislocations may be associated with erosion of the femoral head by the indirect head of the rectus femoris and/or other deformations resulting in mild to severe loss of sphericity and degenerative changes. There are no absolute guidelines to determine which hips are reconstructible, and direct inspection may be required to assess the degree of damage and decide what to do

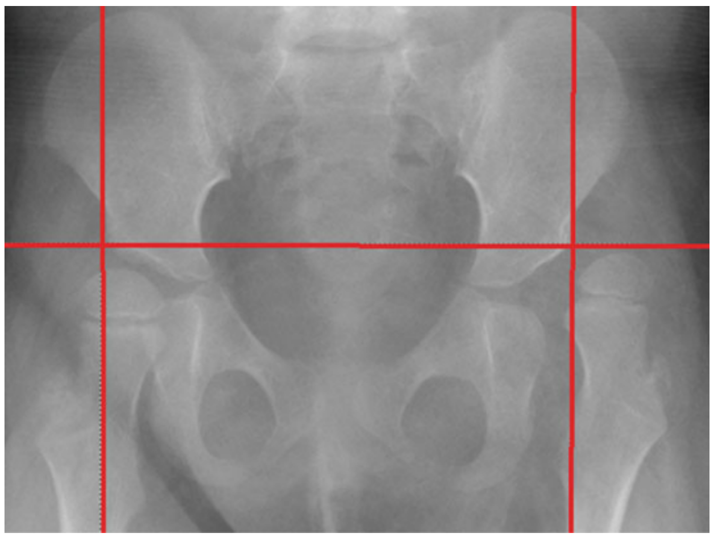


Fig. 2 AP x-ray of the pelvis showing a migration percentage of 20 % on the right and >90 % on the left

migration or displacement of the femoral head as well as progressive dysplasia of the acetabulum. Muscle imbalance also prevents the normal derotation of the femur or a failure to remodel femoral anteversion from approximately 45° at birth to 10–15° as an adult. The neck-shaft angle may be normal in some cases, but coxa valga or an increased neck-shaft angle will be observed in most cases. As the dysplasia progresses, and the femoral head subluxates or dislocates, the femoral head may become deformed due to compression by the indirect head of the rectus femoris by muscle pull, leading to erosion or loss of the superolateral portion of the femoral head (Fig. 1).

Displacement of the femoral head is assessed using the Reimer's migration index (Fig. 2). This measurement is taken by first drawing a line horizontal through the triradiate cartilage (Hilgenreiner's line) and then a line vertical at the edge of the lateral margin of the acetabulum (Perkin's line). The percent of displacement of the capital femoral epiphysis is then calculated as the percentage of the capital femoral epiphysis that lies outside Perkin's line. If the migration percentage is over 50 % [3], subluxation and dislocation is highly likely [4]. The acetabular index is also evaluated and is felt to be abnormal if >25°. The characteristics of the sourcil should also be noted.

Surveillance programs have been set up to screen for dysplasia and institute early treatment with the goal of preventing dislocation and minimizing the magnitude to surgery required to achieve this goal. These surveillance systems involve repeating the physical examination, typically every 6 months, and repeating radiographs at an interval defined by the relative clinical risk of progressive dysplasia.

Surgical treatment strategies for neuromuscular hip dysplasia may be (1) prophylactic, (2) reconstructive, or (3) salvage. Prophylactic procedures are indicated with younger patients who have developed soft tissue contractures but do not have significant subluxation. These patients typically exhibit clinical "at risk" signs (passive abduction in extension <45°) and minimal elevation in the migration percentage, <40–50 %. In such cases, an open adductor release with or without psoas tenotomy may prevent the need for reconstructive surgery in more than 50 % of patients at 9 years follow-up. Even when soft tissue surgery fails to eliminate the need for bony reconstructive surgery, the reconstructive procedures are performed at a later age which should reduce the chance or resubluxation or dislocation with growth and remodeling. Reconstructive surgery is performed when significant bony abnormalities are present (significant subluxation and acetabular dysplasia), ideally in a child older than 4 years of age. Salvage surgery is considered in hips that are felt to be non-reconstructible, in which femoral head deformity is severe or arthritic changes are present, typically in patients with long-standing dislocation and chronic pain. The degree of femoral head deformity beyond which reconstructive surgery should not be considered remains unknown, and there is no scientific evidence to guide this decision.

In this chapter reconstructive surgery will be considered which typically involves a soft tissue release, correction of proximal femoral deformity (excessive anteversion and valgus) by a varus derotational osteotomy (VDRO), and correction of coexisting acetabular dysplasia with a volume-reducing, incomplete pelvic osteotomy. In some cases a medial capsulotomy or an open reduction may also be required as a component of this reconstruction. The first goal of the femoral osteotomy is to restore normal rotational alignment to the proximal femur by correcting excessive femoral anteversion, which will improve abductor mechanics in the ambulatory, and lower extremity positioning in the nonambulator. The second goal is correction of coxa valga, recognizing that the desired end point for neck-shaft angle differs in ambulators versus nonambulators. The goal of the acetabular osteotomy is to restore acetabular morphology and improve coverage of the femoral epiphysis.

Indications for Surgery

1. Progressive subluxation or dislocation in a hip that is reconstructible (Fig. 1)

Equipment

1. Blade plate set: Comes with infant, child, adolescent, and adult sizes
2. Sterile goniometer
3. K wires
4. Oscillating saw
5. Cobb and Crego elevators
6. Hohman retractors
7. Lamina spreader
8. Bone holding forceps or clamps (Verbrugge, Lowman)
9. Metallic wedges of predetermined angles

Technique of Femoral Varus Derotational Osteotomy

Positioning and Preparation

1. Either supine or prone positioning may be chosen based on whether a pelvic osteotomy is required and the surgeon's experience and preference. When a pelvic osteotomy is not performed, typically in the ambulatory population, the procedure can be performed in the prone position. This position is familiar as the clinical assessment of lower extremity rotational alignment is usually carried out in this

position. There are several technical advantages, especially with regard to clinically estimating femoral anteversion. For one, it is easier to insert and maneuver the chisel for the blade plate when it is facing upwards, and fine-tuning of the chisel's trajectory on the lateral image can be made with simple positioning of the leg rather than while an assistant holds up the leg while you are striking the chisel close to the surface of the table. It is also possible to clinically estimate femoral anteversion using a guide pin up the femoral neck (Fig. 3).

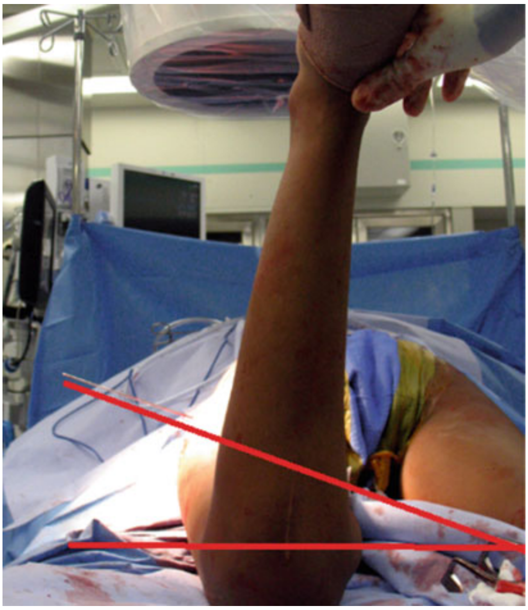


Fig. 3 Intraoperative assessment of femoral anteversion pin placed in center position on the lateral compared to the transepicondylar axis. In this instance the patient is in the prone position, but a similar assessment can be conducted in the supine position by holding the knee flexed to 90°

A supine position can also be used in nonambulators since they frequently require a pelvic osteotomy. In this position, the patient is placed with a bump under the ipsilateral buttock. If a bilateral procedure is to be performed, then a small square bump under the sacrum is helpful, particularly if performing pelvic-sided procedures (Fig. 4).

2. If a bilateral procedure is being done, a folded surgical towel can be placed over the genitalia and sealed using an occlusive dressing. The prep may be done over this sealed dressing.
3. The entire leg is prepped out with care to provide an adequate superior margin, up to the lower ribs, and also an adequate posterior margin.

Adductor Tenotomy

1. An adductor release is routinely performed with the VDRO in the nonambulatory population. This is rarely needed in an ambulatory patient and if required may often be performed as a percutaneous release of the fascia overlying the adductor longus rather than the more

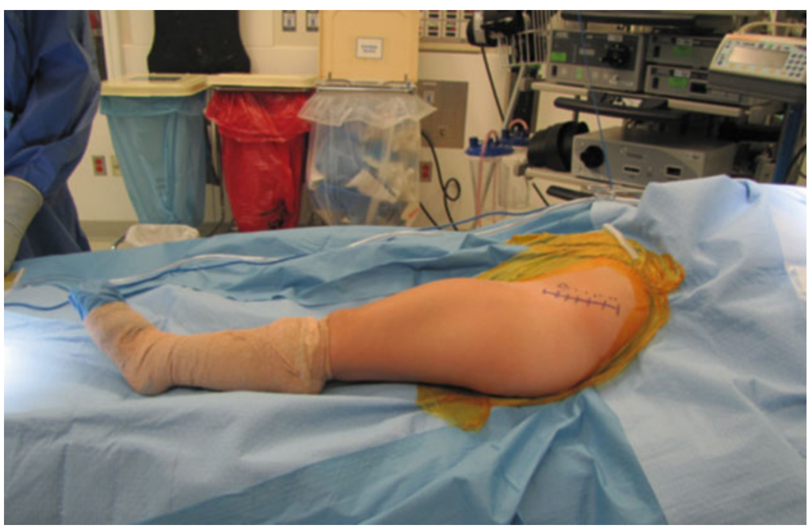


Fig. 4 Positioning of patient for cerebral palsy unilateral hip reconstruction following draping (photograph courtesy of Wudbhav Sankar, MD)

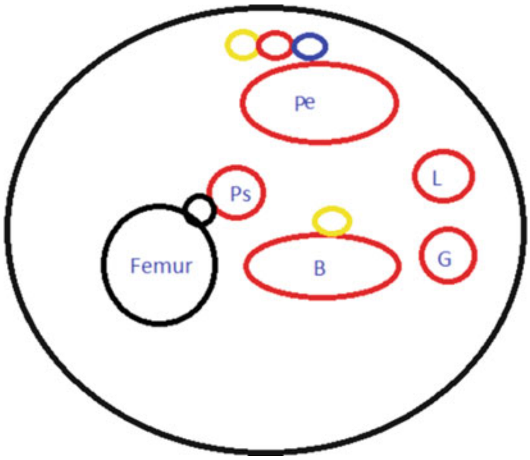


Fig. 5 Cross-sectional schematic of the proximal femur at the level of the lesser trochanter" *B* adductor brevis, *G* gracilis, *L* adductor longus, *Pe* pectineus, *Ps* psoas, *yellow* nerve, *blue* vein, *black* bone

extensive open release. In the ambulatory patients, correction of derotation is usually the major aim and little if any varus is required.

2. A transverse incision is made in the groin crease directly overlying the adductor longus tendon, about two fingerbreadths distal to the superior pubic ramus.
3. A longitudinal incision is made in the deep fascia overlying the adductor longus tendon, and the subfascial interval developed which exposes the adductor longus and surrounding muscles (Fig. 5).
4. A right angle retractor is passed around the longus tendon, and the tendon is then released with electrocautery. The anterior neurovascular bundle must be identified on the surface of the adductor brevis and protected. The gracilis is then bluntly identified and released with electrocautery. This muscle is placed under tension by abducting the hip with both the hip and knee extended.
5. A partial myotomy of the adductor brevis may be performed if additional passive abduction is required.
6. If a psoas tenotomy is planned at this stage, the interval between the pectineus and the brevis is used and widened laterally. The psoas tendon is identified as it attaches to the lesser trochanter. The sheath is opened up and a right angle is passed around it. It is released with electrocautery, while being careful to protect the medial femoral circumflex artery.
7. When the desired amount of hip abduction is obtained, the wound is irrigated and close in layers once hemostasis has been achieved.

Surgical Approach for VDRO

1. A longitudinal incision is made over the lateral proximal femur extending from the trochanteric flare or slightly above down along the femoral shaft. Sufficient length must be made to accommodate the length of the plate taking into account shortening from the varus with or without removal of any additional bone wedges.
2. Dissect through the skin subcutaneous tissue down to the level of the fascia lata. The fascia lata is then divided in line with the skin incision. A self-retaining retractor is placed in the fascia lata.
3. The fascia overlying the vastus lateralis is incised longitudinally approximately 5 mm anterior to the intramuscular septum. The muscle is teased off the posterior fascia until the periosteum is visualized, and then the periosteum is incised to subperiosteally expose the proximal femur. This cuff of remaining posterior fascia is utilized for the repair.
4. Proximally, the origin of the vastus lateralis is incised transversely (perpendicular to the longitudinal cut in the fascia) along the trochanteric flare and distal to the trochanteric apophysis, to allow the muscle to be reflected anteriorly (Fig. 6). Care is made not to plunge posteriorly with the cautery as the sciatic nerve is very close.
5. The periosteum is incised to expose the femur subperiosteally. Hohman retractors are placed anteriorly and posteriorly for protection.

Osteotomy Technique (90° Blade Plate)

Determining Rotation and Neck-Shaft Angle

The first component is to estimate or measure the true neck-shaft angle and the degree of anteversion. Recognizing that a more important

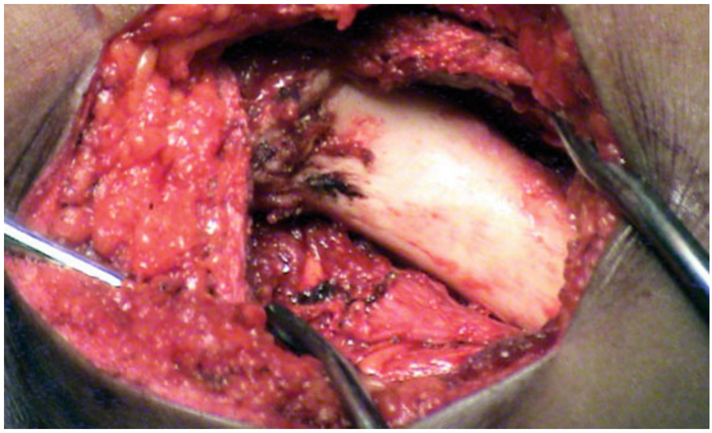


Fig. 6 The lateral approach to the femur. The proximal femur has been subperiosteally exposed. The lateral aspect of the greater trochanter apophysis is visible in the proximal aspect of the wound

concern is how to accurately achieve targets for final anteversion and neck-shaft angle, it is advantageous to get baseline measurements for completeness. These measurements can be made either before or after the patient is prepped and draped. With regard to neck-shaft angle, the hip is internally rotated until a true AP is obtained, typically when the length of the femoral neck appears greatest on the AP image. This image can be saved and measured with a goniometer. The degree of anteversion may be been measured using several techniques. Some prefer to obtain a CT scan preoperatively; however, anteversion may be measured intraoperatively. In the first, prior to prepping and draping, the patient is slid down to the end of the table, and the knee is flexed 90° over the edge. The hip is then medially rotated until a true AP is obtained, and the version is then measured as the angle between the tibial axis or shank and the vertical. Alternatively, version may be measured during the procedure.

Achieving Varus: Guidewire Placement

(A) The guide pin is placed using fluoroscopy. The starting point is typically just inferior to the trochanteric apophysis when the cannulated blade plate device is utilized; otherwise, the guidewire can be placed above the insertion point of the chisel. The guidewire must be placed to allow for maximal length of the blade without violating the femoral neck or the medial calcar.

(B) The next step is to determine what sized plate to use, and this can be done intraoperatively (or could have been done preoperatively). Options include the toddler, child, and adolescent sizes. The chisel is the same for the child and adolescent plates. The larger chisel can be held over the femur on the frog lateral view to determine if there is sufficient anteroposterior diameter in the femoral neck to accommodate the larger chisel. If the larger chisel can be accommodated, choosing between the child and adolescent plate will be based on comparison of these two plates on fluoroscopic images.

(C) Determine the correct angle of placement.

(C1) Using the *femoral neck as a reference* to achieve varus. If the chisel is placed parallel with the femoral neck, and a 90° blade plate is utilized, then the ultimate neck-shaft angle should be 90° (a 100° blade plate would place the neck-shaft angle at 100°). If the 90° blade plate is placed 10° lower in the neck than the neck-shaft angle, a 100° final neck-shaft angle will result (Fig. 7). Every degree higher will result in a degree lower neck-shaft angle, and every degree lower will result in a higher final neck-shaft angle. In order to use this technique, a true AP must be obtained, and a goniometer is used to determine the degree difference between the pin and the superior neck.

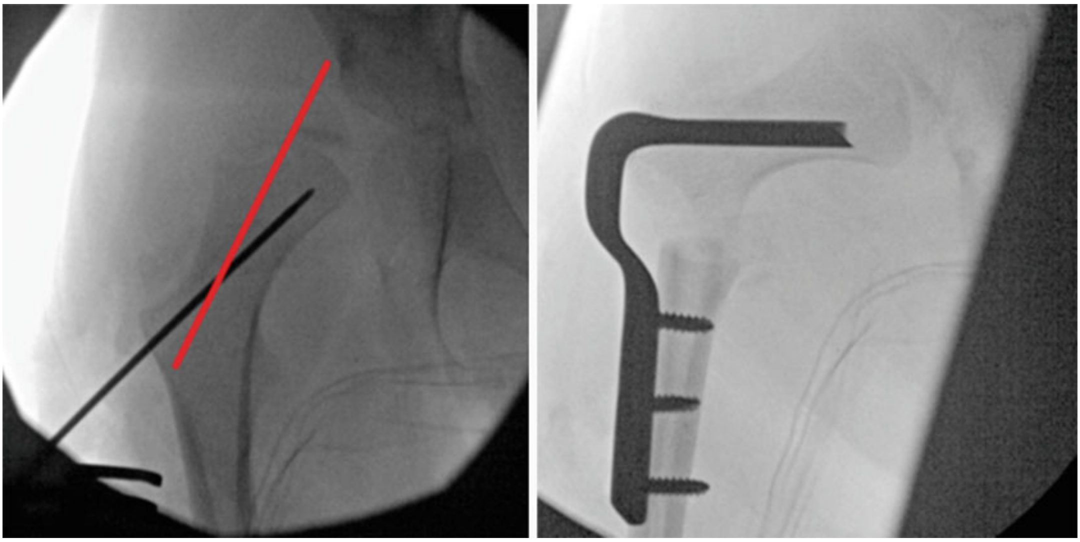


Fig. 7 On a perfect AP, this guide pin (*left panel*) is placed at 20° off the neck-shaft angle (*red line*). Using a 90° blade plate, this results in a 110° final neck-shaft angle (*right panel*)

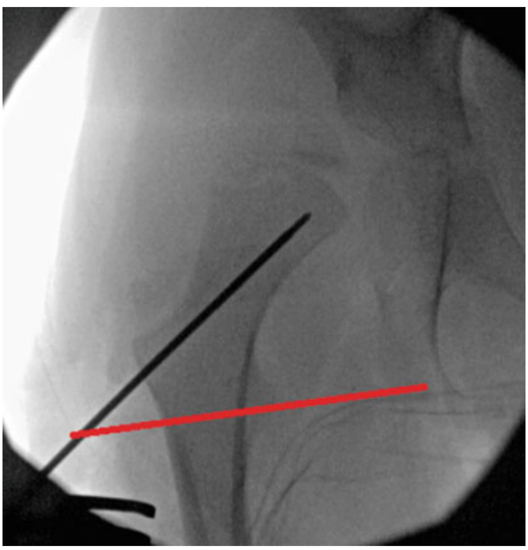


Fig. 8 Using the second technique, the guide pin placement indicates that 30° of varus correction will be obtained (*red line* perpendicular to the femoral shaft)

(C2) Using the *femoral shaft as a reference*. Some feel that the femoral neck is less consistent because the neck-shaft angle varies with subtle degrees of rotation (Fig. 8). The femoral shaft may be used as well. The angle between a line is drawn perpendicular to the femoral shaft, and a second line extending vertically (more parallel with the neck) will correspond to the amount of varus that is produced (Fig. 8). If the femoral shaft method is being used, use a radiopaque triangle to determine trajectory of the guide pin. Another reference can be the lateral femoral cortex. If the angle between the guide pin and the lateral femoral cortex is 60°, then 60 minus the 90° blade plate is 30° of correction.

Chisel Placement and Osteotomy

(A) The chisel is impacted either over the guidewire if a cannulated system is utilized or parallel to the guidewire.

(B) While the guidewire provides a reference in the coronal plane, care must be taken to orient the chisel perpendicular to the axis of femoral shaft to avoid introducing flexion or extension at the osteotomy. The implant manufacturer may also include a guide that sits on the femoral shaft to assess flexion/ extension.

(C) The chisel is lightly malleted into the bone, with periodic checks of the fluoro to make sure that the chisel has not deviated from expected path, which can occur even when a cannulated system is used. Periodically, the chisel should be backslapped to assure that it does not become incarcerated especially when bone quality is good. Avoid applying torque to the chisel especially in osteopenic bone.

(D) The osteotomy is completed with an oscillating saw proximal to the end of the shoulder of the plate (1–1.5 cm). A transverse osteotomy may be completed just above the lesser trochanter, and then the medial corner of the proximal fragment may be placed within the canal of the distal fragment. This achieves some medial translation at the osteotomy, which is essential in an ambulatory patient to restore the mechanical axis. In the nonambulator, either this technique or a medially based closing wedge osteotomy may be performed. The proximal saw cut is parallel to the chisel, while the distal cut is perpendicular to the femoral shaft. Additional shortening may be achieved, and bone wedge may then be utilized as graft for the pelvic osteotomy (if required). The psoas must be released to deliver the resected bone.

Placement of the Blade Plate and Achieving Derotation

(A) Remove the guidewire and the chisel, and gently insert the plate manually. Light taps with a mallet may be utilized to insert the plate, and the plate should go in easily. Make sure that the plate clears any soft tissues in the distal part of the wound; otherwise, it may be forced off the trajectory resulting in malpositioning. Fluoroscopic images are usually required to confirm proper orientation before final seating of the plate.

(B) Reduce the plate to the bone, and apply the desired amount of rotational correction. There are various opinions regarding the desired amount of derotation. It is important to recognize that the rotational orientation of the femur during gait does not directly correlate with the degree of anteversion. The goal is for restoration of the normal degree of anteversion, and so the target anteversion is generally 15–20°, in both ambulators and

Fig. 9 The examiner palpates the greater trochanter prominence of the patient. The internal rotation of the leg at the point of greatest prominence is the femoral anteversion (Reproduced with permission and copyright © of the British Editorial Society of Bone and Joint Surgery [Robin J, Graham HK, Selber P, et al. Proximal femoral geometry in cerebral palsy: a population-based cross-sectional study. J Bone Joint Surg [Br] 2008;90-B:1372–1379. (Fig. 1)])

nonambulators, based on intraoperative measurements as outlines below [5]. Other studies in ambulators have suggested (1) more than 20° of external rotation greater than internal rotation [6], (2) 50° external rotation and less than 30° internal rotation [7], and (3) the "clinical midpoint [8, 9]."

(C) The degree of femoral anteversion may be estimated on *bench examination* by the trochanteric prominence test (Fig. 9), in which the patient is placed prone and the knee is flexed 90°. The lower leg is internally or medially rotated until the greater trochanter is felt to be most prominent, and then the angle between the vertical and the tibial axis is measured as the degree of anteversion. The degree of anteversion should not be confused with the degree of medial rotation, and the medial rotation will always be greater than the anteversion. For example, a patient with 80–90° of medial rotation may have a femoral anteversion of only 40–50°. The trochanteric prominence test is less reliable in patients who are obese or when there is a lateral scar from previous surgery. In addition, the most prominent point of the trochanter is anterior which may also impact the accuracy clinical estimation using this technique [10].

(C1) Anteversion may be measured ***intraoperatively*** using a variation of this concept, by placing a guide pin parallel to the femoral neck, or using the chisel inserted for the blade plate, assuming it is parallel to the femoral neck. If the surgery is performed in the *prone* position, the leg can be medially rotated until the guide pin or chisel is parallel with the floor, and then the ankle between the tibial axis (shank) and the vertical represents the degree of anteversion. This may also be measured by keeping the lower leg axis vertical and then measuring the angle between the horizontal axis and the axis of the guide pin (Fig. 3). If the patient is in the *supine* position, the knee can be flexed 90° and the angle measured between the guide pin or chisel and a line perpendicular to the lower leg (knee joint axis) will represent the degree of anteversion.

(C2) Another technique involves placement of a Kirschner wire in the distal femur and a second in the greater trochanter parallel to each other, and the angle between the Kirschner wires can be measured with a goniometer as the degree of derotation. Alternatively, if a cannulated system is used, the distal femur pin can be compared to the pin in the plate to see what the anteversion is following osteotomy.

(C3) Another technique is to have premade metallic wedges of different angles to guide placement of the two Kirschner wires at the desired angle of correction, and then derotate until the two Kirschner wires are parallel.

(D) When the desired degree of derotation has been achieved, then place the first screw in the plate in compression mode, then place the other two screws. Sometimes it is easiest to hold the plate to the bone manually when placing the first screw, since some bone holding clamps may be difficult to securely clamp down depending on the amount of rotational correction achieved. Consider the Lowman ("turkey claw") clamp if available. Take stock in the rotational correction, and obtain final fluoroscopic imaging prior to irrigating and closing the wound in layers.

Discussion Points

1. Younger patients (four and under) have a greater chance of recurrence, and parents should be advised as such.
2. The operation shortens the limb, and the parents should be advised of this.
3. Each degree of varus results in one degree less of passive abduction, so a significant adductor release, and occasionally a medial capsulotomy, is required to retain adequate passive abduction after surgery. Otherwise, diapering and perineal care may remain or become a challenge for families.

4. Recognizing that remodeling of the neck-shaft angle will occur depending on the age of the patient, with a goal of 90–100° of varus in nonambulators [11]. The goal for ambulatory patients is 115–120°, as lower values may interfere with abductor mechanics.
5. There has been some controversy in the literature as to whether to perform a bilateral VDRO in patients with unilateral subluxation or dislocation. There is no definitive evidence to suggest that the natural history of the "normal" side hip is poor or that there is an absolute indication for bilateral surgery. Bilateral surgery will certainly address the asymmetry in thigh contour and leg lengths seen when a unilateral procedure is performed, and may improve the appearance of "windswept hips" especially when there is an abduction contracture on the normal side. It is often prudent to discuss these issues with each family and allow them to participate in the decision making process.
6. While the weight of the available evidence supports the use of a pelvic osteotomy when the acetabular index is greater than 25°, and liberal use of a volume-reducing pelvic osteotomy has been reported in the majority of case series, recent studies have questioned this practice and suggested that the pelvic osteotomy may not be required as frequently as previously thought [12].

Technique of Pelvic Osteotomy

Indications

1. A pelvic osteotomy is indicated if there is significant acetabular dysplasia, >25–30° (Fig. 10). Some authors prefer to do an intraoperative exam under anesthesia (EUA and arthrogram) in the setting of a large MP (<50–60%). If the EUA shows a nonconcentric reduction, then open reduction is performed. If it shows decreased coverage, a pelvic osteotomy is performed. Some authors advocate always performing a pelvic-sided procedure [13].
2. The acetabular deficiency in neuromuscular hip dysplasia is typically global or posterior, in contrast to developmental dysplasia in which the deficiency is anterior and lateral. Some authors favor routine the use of computed tomography preoperatively to better define the deficiency.

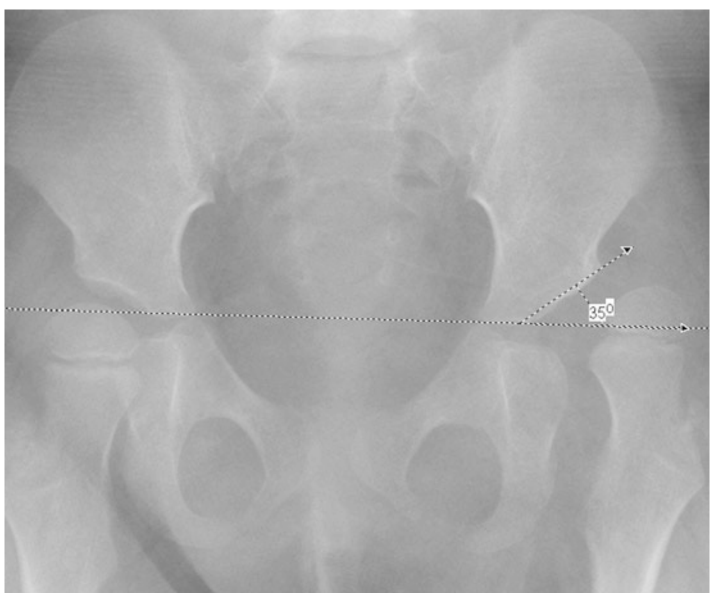


Fig. 10 Increased acetabular angle in a patient undergoing hip reconstruction

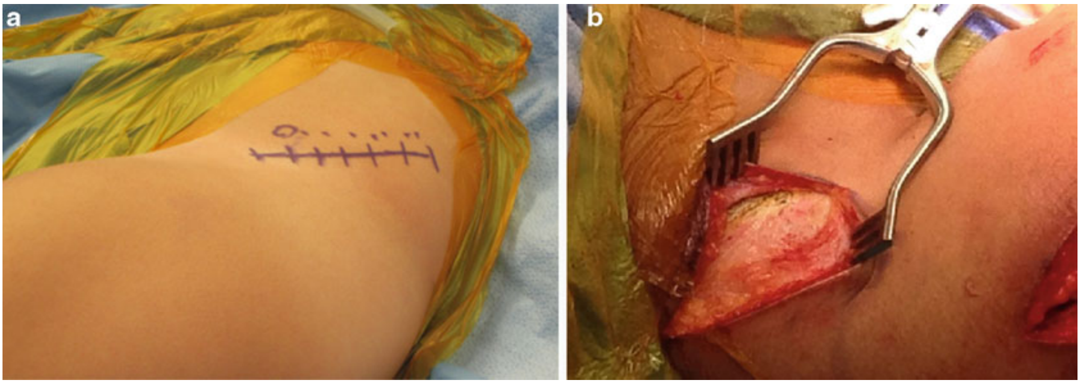


Fig. 11 Standard exposure for the pelvic osteotomy (Fig. X2a, b)

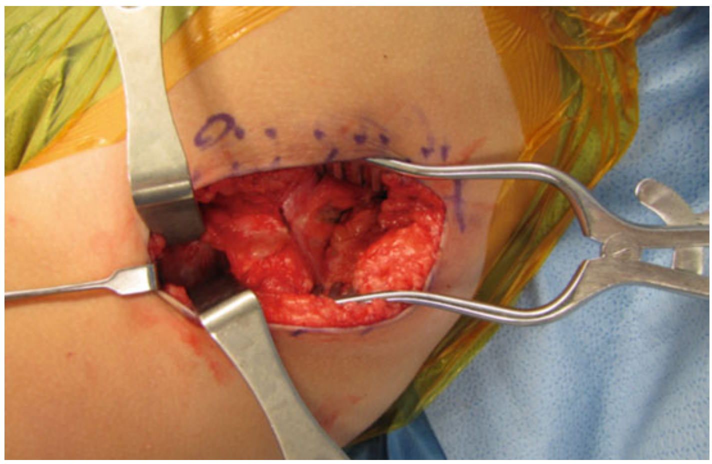


Fig. 12 The deep Smith-Petersen interval (Photograph courtesy of Wudbhav Sankar, MD)

Approach

1. An incision is made drawn parallel to the iliac crest, 1–2 fingerbreadths distal to the crest, beginning over the sartorius and extending along to the posterior iliac crest (Fig. 11a, b).
2. Dissect through the skin and subcutaneous tissue. The external oblique fascia is reflected exposing the iliac crest, and the apophysis of the ilium is split.
3. Distally, the interval in between the sartorius and the tensor fascia lata is identified, and the fascia overlying this interval is split longitudinally. The lateral femoral cutaneous nerve is identified and protected. The interval is developed bluntly down to the level of the rectus femoris (Fig. 12).
4. The outer table of the pelvis is exposed subperiosteally down to the sciatic notch, and the capsular margin is identified. Subperiosteal dissection is often completed superior to the anterior inferior iliac spine around the corner. The inner table can be exposed if it is the surgeon's preference or when an open reduction is required.
5. If a medial capsulotomy or an open reduction is to be performed, the skin incision may need to be extended medially, with or without a distal curved component as in the full Smith-Peterson approach. Both the direct and the indirect heads of the rectus are released and tagged, and the hip capsule can then be identified. The sartorius can be released, or reflected along with the soft tissues along the inner table of the ilium, when required for a formal open reduction.

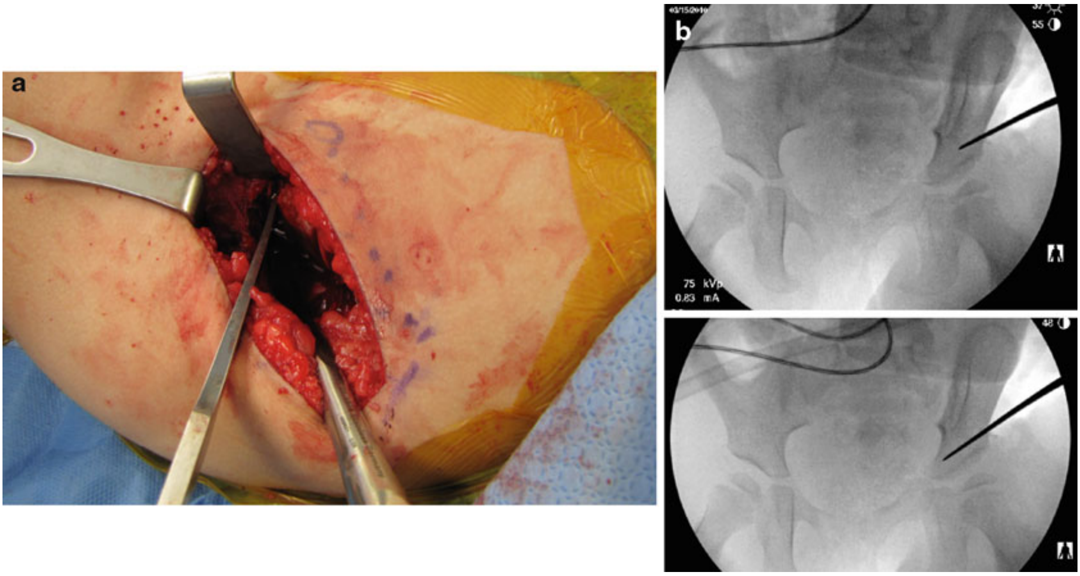


Fig. 13 The chisel is inserted for the periacetabular osteotomy (Photograph courtesy of Wudbhav Sankar, MD)

Blunt dissection with a curved hemostat will allow the medial capsule to be freed from the overlying soft tissues, and then a small Hohmann retractor can be placed. A medial capsulotomy can then be performed which will often increase passive abduction by 20–30°. In the case of an open reduction, the technique for opening the capsule and for capsulorrhaphy will be similar to that utilized for open reduction of a developmentally dysplastic hip.

Technique

1. The osteotomy is made 1–1.5 cm above the capsular insertion, and it is often helpful to take a fluoro image if the capsular margin is not obvious under direct vision.
2. As the location varies from anterior to posterior, the pelvis is scored at group of points at the same distance from the capsule as a guide for the osteotomy. Some authors prefer to place guidewires first and obtain fluoroscopy to assure that the osteotomy is appropriately directed. These guidewires are directed towards the triradiate cartilage.
3. The osteotomy is curvilinear and will require multiple passes of the osteotome under fluoro. The osteotomy is started just above the AIIS and continues posteriorly 1–1.5 cm in front of the sciatic notch. It may be curved distally if more posterior coverage is desirable.
4. Cuts are made with a thin, narrow, straight osteotome to connect the different points and then proceed to using a thin, curved osteotome. The cuts are made under fluoroscopic guidance, extending across towards the medial cortex and then curving down towards (but not into) the triradiate cartilage (Fig. 13a, b). A retractor is placed in the sciatic notch for protection when the posterior cuts are made.
5. Next an osteotome is used to cut the ilium medially and inferiorly in line with the guidewire down through the inner wall. The posterior third of the inner wall is left intact.
6. The cortex is then levered down with a wide osteotome and held open with the bone graft previously obtained with the femoral osteotomy. The less inner wall that is taken, the more lateral coverage will be obtained (Fig. 14).
7. Irrigate and close in layers, and close the iliac apophysis with heavy vicryl or ethibond suture.
8. Immobilization is either with a soft abduction pillow or a spica cast depending on the

Fig. 14 The acetabulum is levered down using the triradiate cartilage as a hinge, and an appropriate size graft is placed in the osteotomy

circumstances of each case. If fixation is adequate and an open reduction was not performed, a soft abduction wedge is preferred. A spica cast is used when an open reduction has been performed. The use of a spica cast has been associated with a greater risk of fractures postoperatively.

Summary

Hip dysplasia is a common problem in cerebral palsy. The incidence of this issue increases with increasing neurologic involvement. Surveillance is necessary to prevent salvage surgery and maintain mobile located hips. Bony and soft tissue reconstruction is often necessary, and pelvic- and femoral-sided procedures may be used in a comprehensive or ala carte approach to this complex population of patients.

References

1. Robin J, Graham HK, Selber P, Dobson F, Smith K, Baker R. Proximal femoral geometry in cerebral palsy: a population-based cross-sectional study. J Bone Joint Surg Br. 2008;90(10):1372–9.
2. Lonstein JE, Beck K. Hip dislocation and subluxation in cerebral palsy. J Pediatr Orthop. 1986;6(5):521–6.
3. Reimers J. The stability of the hip in children. A radiological study of the results of muscle surgery in cerebral palsy. Acta Orthop Scand Suppl. 1980;184:1–100.
4. Bagg MR, Farber J, Miller F. Long-term follow-up of hip subluxation in cerebral palsy patients. J Pediatr Orthop. 1993;13(1):32–6.
5. Gage JR. The treatment of gait problems in cerebral palsy. London: Mac Keith/Distributed by Cambridge University Press; 2004.
6. de Morais Filho MC, Kawamura CM, dos Santos CA, Mattar Jr R. Outcomes of correction of internal hip rotation in patients with spastic cerebral palsy using proximal femoral osteotomy. Gait Posture. 2012;36 (2):201–4.
7. Ounpuu S, DeLuca P, Davis R, Romness M. Long-term effects of femoral derotation osteotomies: an evaluation using three-dimensional gait analysis. J Pediatr Orthop. 2002;22(2):139–45.
8. Kim H, Aiona M, Sussman M. Recurrence after femoral derotational osteotomy in cerebral palsy. J Pediatr Orthop. 2005;25(6):739–43.
9. Dreher T, Wolf S, Braatz F, Patikas D, Doderlein L. Internal rotation gait in spastic diplegia–critical considerations for the femoral derotation osteotomy. Gait Posture. 2007;26(1):25–31.
10. Davids JR, Benfanti P, Blackhurst DW, Allen BL. Assessment of femoral anteversion in children with cerebral palsy: accuracy of the trochanteric prominence angle test. J Pediatr Orthop. 2002;22(2):173–8.
11. Mazur JM, Danko AM, Standard SC, Loveless EA, Cummings RJ. Remodeling of the proximal femur after varus osteotomy in children with cerebral palsy. Dev Med Child Neurol. 2004;46(6):412–5.
12. Huh K, Rethlefsen SA, Wren TA, Kay RM. Surgical management of hip subluxation and dislocation in children with cerebral palsy: isolated VDRO or combined surgery? J Pediatr Orthop. 2011;31(8):858–63.
13. McNerney NP, Mubarak SJ, Wenger DR. One-stage correction of the dysplastic hip in cerebral palsy with the San Diego acetabuloplasty: results and complications in 104 hips. J Pediatr Orthop. 2000;20(1): 93–103.

41 Surgical Technique: Resection Arthroplasty

Jeffrey Ackman

Contents

Abstract

Treatment of the subluxated or dislocated hip in a spastic patient is still controversial, though the untreated hip dislocation has a poor natural history. Goals of treatment are to relieve pain and to improve care and function. Our preferred treatment is the McHale osteotomy which combines femoral head and neck resection with adductor release and proximal femoral valgus osteotomy with internal fixation. This is a one-stage operation that meets the goals of surgery and improves the biomechanics of the hips.

Introduction

Treatment of the subluxated/dislocated hip in the spastic patient population has long caused a dilemma in orthopedics (Figs. 1 and 2). Questions arise as to whether anything should be done operatively, and if so, should attempts be made to preserve the hip with containment procedures such as muscle releases and femoral and/or innominate osteotomies, or try to relieve pain and contractures with abduction osteotomies or proximal femoral resection.

The untreated hip dislocation in the spastic patient population has a poor natural history [1] and the results of all procedures have poor outcomes in the growing child vs. the young adult.

The goals of surgery for the spastic dislocated hip are fourfold [2]: (1) relieve adduction

J. Ackman
Shriners Hospital for Children, Chicago, IL, USA
e-mail: jdackman@shrinenet.org

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_45

Fig. 1 Typical appearance of the dislocated hip with adduction contracture in a spastic patient

Fig. 2 X-ray showing the dislocated left hip

contractures to facilitate perineal hygiene and nursing care, (2) restore hip mobility enough so as not to interfere with positioning in bed or chair, (3) simple procedure so as to minimize complicated postop care and prolonged hospitalization, and (4) one-stage operation.

The majority of these patients are non-ambulatory, so the procedure is one to improve the quality of life, with its inherent economic and humanitarian implications.

Prevention is obviously the preferred treatment for the patient with spastic hip subluxation/dislocation, with early surgical intervention and the appropriate soft tissue releases and femoral and/or pelvic osteotomies. However, for long-standing cases with significant acetabular dysplasia as well as a deformed and eroded femoral head, measures other than containment are necessary. Options include simple soft tissue release, valgus osteotomy, Girdlestone procedure of femoral head and neck resection, proximal femoral resection with interposition arthroplasty, arthrodesis, and total hip arthroplasty.

Soft tissue release alone is insufficient to relieve pain and deformity, and valgus/abduction osteotomies often result in a stiff hip with skin breakdown over the prominent femoral head and difficulty with positioning [2–4]. The Girdlestone procedure is complicated by heterotopic ossification and proximal femoral migration with recurrent pain and deformity [2, 5–7]. These procedures are no longer recommended.

Proximal femoral resection with interposition arthroplasty [2, 5] has generally provided good pain relief and positioning ability, but still has problems with heterotopic ossification and requires traction postoperatively, requiring additional time in recumbency or rigging of the wheelchair. Hip arthrodesis [7] is only for patients with a normal contralateral hip and spine, often requires a concomitant proximal femoral osteotomy for positioning, and may develop heterotopic ossification and pseudarthrosis and maybe a hard sell to the patient and family. Total hip arthroplasty [7] often requires a custom stem and difficulty with positioning the acetabular component at its true level, requires a spica cast or brace for immobilization postoperatively, and has the inherent problems of dislocation, loosening, and wear.

Our preferred treatment is the McHale osteotomy, which combines a femoral head and neck resection with adductor release and a proximal femoral valgus osteotomy with internal fixation [4]. This is a one-stage operation that fulfills all the goals of surgery.

Technique

The patient is positioned supine under general anesthesia on the operating table with a bump or roll under the hip. Additional caudal or epidural block is at the discretion of the surgeon and the anesthesiologist. An adductor release of the

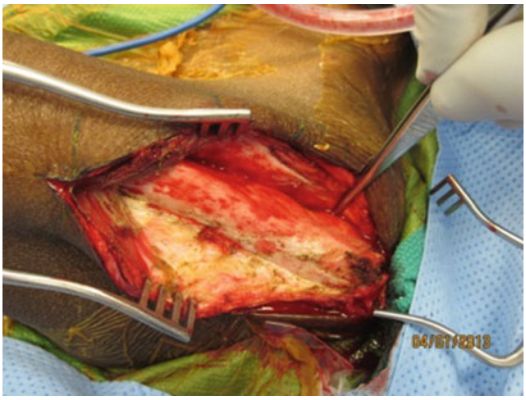


Fig. 3 The proximal femur is exposed subperiosteally through an anterolateral approach

Fig. 4 The abductors have been tagged and detached and a wide capsulotomy performed exposing the head/neck

longus and brevis is performed through a longitudinal incision in the groin over the adductor longus tendon, and dissection carried through to release the adductor longus and brevis. Obturator neurectomy is not routinely performed. The incision is closed in a standard fashion and attention is then turned laterally to the hip joint itself.

A straight lateral incision is made from approximately level with the anterior superior iliac spine, across the greater trochanter, and down the femoral shaft. An anterolateral approach to the hip, i.e., Watson-Jones, is used (Fig. 3). The fascia is exposed in line with the skin incision and split longitudinally, taking care to remain posterior to the posterior border of the tensor fascia lata proximally. Cutting too far anteriorly in the muscle belly of the tensor may make it difficult to discern the plane between the gluteus medius and tensor fascia lata.

Dissect with your finger under the cut fascia anteriorly and posteriorly and retract the fascial edges with a self-retainer. Identify the interval between the retracted tensor fascia and the gluteus medius. Place the retractor deep to the gluteus medius and retract superiorly and laterally to expose the fat pad overlying the hip capsule.

Incise the vastus lateralis with electrocautery starting at the vastus ridge and moving distally down the shaft of the femur. Either split the vastus lateralis or lift it anteriorly to expose the periosteum of the femur and then expose the femur subperiosteally.

Returning proximally, place a stay suture in the anterior tendinous portion of the gluteus medius just above the insertion into the greater trochanter, and then cut the anterior portion of the tendon. Identify the tendinous portion of the gluteus minimus as it inserts onto the greater trochanter, tag it, and detach it as well.

Flex, adduct, and externally rotate the hip and then dissect up towards the acetabulum, remaining just on top of the head and capsule. The indirect head of the rectus femoris may need releasing to gain more medial exposure of the hip capsule, and flexing the hip relieves tension to allow you to stay deep to the rectus and psoas, affording protection to the neurovascular bundle.

You should then be able to palpate the anterior rim of the acetabulum deep to the rectus and psoas. Expose and identify the hip capsule and then incise it in a T-fashion, with the longitudinal limb along the axis of the femoral neck and the transverse limb on the femoral attachment. If additional exposure of the head and neck is needed, a transverse limb can be added on the acetabular side, converting the T configuration to an H. You now should have exposure of the proximal femur from the greater trochanter to an area of 5–10 cm distal to the lesser trochanter (Fig. 4).

With an oscillating saw and/or osteotome, make a complete osteotomy at the base of the femoral neck and remove the femoral head, trying to leave as much ligamentum teres as possible (Figs. 5 and 6). A laterally based closing wedge osteotomy

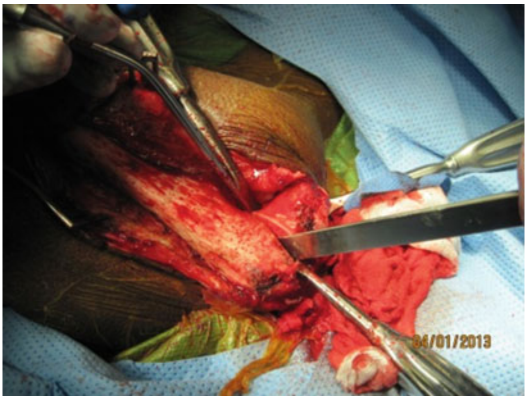


Fig. 5 Osteotomy is performed at the base of the femoral neck to resect the femoral head and neck

Fig. 6 The deformed and eroded femoral head

is then performed at or just below the level of the lesser trochanter to allow at least 45° of abduction. Internal fixation is provided by a plate and screws and either contouring of the plate with a plate bender or selecting a pre-bent plate (Fig. 7).

After fixation is complete, attempts are made to attach the ligamentum teres to the psoas tendon at the lesser trochanter, and a capsulorrhaphy is performed to help hold the lesser trochanter in the acetabulum. The abductor tendons are then sutured back to the greater trochanter.

The wound is then thoroughly irrigated to wash out any bone debris, a Hemovac drain may then be placed if necessary, and routine closure is performed. An abduction pillow is used postoperatively. The patient may be up to a wheelchair and gentle range of motion is begun as soon as tolerated. The osteotomy usually heals in 6–8 weeks.

Fig. 7 After femoral head and neck resection and valgus osteotomy with plate fixation

Summary

With this technique, as opposed to simple valgus osteotomy, Girdlestone stone head/neck resection, or complete proximal femoral resection, there appears to be fewer complications in the long term and there is no need for traction or external fixation as is necessary for femoral resection/interposition arthroplasty. Complications such as proximal femoral migration and recurrent pain and/or adduction contracture appear to be less than other procedures. Though heterotopic ossification is still a risk, it rarely interferes with function or requires resection later. Biomechanically the abductor insertion is moved laterally, akin to varus osteotomy. The improved moment arm increases the vector force of the abductors to oppose the deforming forces of the adductors and iliopsoas.

References

1. Cooperman DR, Bartucci E, Dietirck E, Millar EA. Hip dislocation in spastic cerebral palsy; long term consequences. J Paediatr Orthop. 1987;7:268–76.
2. Castle ME, Schneider C. Proximal femoral resection interposition arthroplasty. J Bone Joint Surg Am. 1978;60:1051–4.

3. Samilson R, Tsou P, Aamof G, Greeen W. Dislocation and subluxation of the hip in cerebral palsy. J Bone Joint Surg Am. 1972;54A:863–73.
4. McHale KA, Bagg M, Nasson SS. Treatment of the chronically dislocated hip in adolescents with cerebral palsy with femoral head resection and subtrochanteric valgus osteotomy. J Paediatr Orthop. 1990;10:504–9.
5. McCarthy RE, Simon S, Douglas B, Zawacki R, Reese N. Proximal femoral resection to allow adults who have severe cerebral palsy to sit. J Bone Joint Surg Am. 1988;70:1011–16.
6. Perlmutter M, Synder M, Miller F, Bisbal R. Proximal femoral resection for older children with spastic hip disease. Dev Med Child Neurol. 1993;35:525–31.
7. Root L, Goss J, Mendes J. The treatment of the painful hip in cerebral palsy by total hip replacement or hip arthrodesis. J Bone Joint Surg Am. 1986;68:590–8.

Additional Reference

8. Barrie JL, Galasko CSB. Surgery for unstable hips in cerebral palsy. J Paediatr Orthop. 1996;5 (Part B 4):225–31.

Part IV

Approach to Hip and Pelvis Disorders

Introduction to Static and Dynamic Overload of Hip Pathology

42

Michael Knesek, Jack Skendzel, and Asheesh Bedi

Contents

Abstract

Diagnosis and management of hip pain in the absence of osteoarthritic changes is challenging for the orthopedic surgeon and requires a thorough understanding of the various static and dynamic mechanical factors that affect the hip joint. Dynamic factors occur as a result of abnormal contact between the femoral head and acetabular rim throughout the hip arc of motion, whereas static factors occur while a patient is standing or in an axially loaded position and undergoes asymmetric or increased loading between the femoral head and acetabulum. When considering surgical treatment for these patients, one must appreciate that compensatory motion due to altered hip mechanics can cause specific injury patterns and pain. This chapter aims to clearly describe the static and dynamic factors associated with mechanical hip pain and the importance of addressing all concomitant pathology to develop a thoughtful and effective treatment plan for this patient population.

Introduction

The development of symptomatic hip pain in the absence of osteoarthritic changes is often a challenge to the treating physician. The pathology is related to the underlying altered structural hip morphology of both the acetabulum and proximal femur. As a result of these structural

M. Knesek (✉) • A. Bedi
Department of Orthopaedic Surgery, University of Michigan, Ann Arbor, MI, USA
e-mail: mknesek@med.umich.edu; abedi@umich.edu

J. Skendzel
Summit Orthopedics, Woodbury, MN, USA
e-mail: jskendzel@gmail.com

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_46

abnormalities, there is cyclic overloading of the articular surfaces with athletic activities or even activities of daily living [1–3]. These patients can therefore be predisposed to the development of early-onset hip osteoarthritis due to increased contact pressures and asymmetric loads across the hip chondral surfaces. Soft tissue structures, such as the acetabular labrum, are also more frequently damaged in this patient population. Recent studies have shown that greater than 90 % of patients with labral tears diagnosed by magnetic resonance imaging have underlying bony structural abnormalities [4]. These alterations in hip joint mechanics that predispose to osteoarthritis should be evaluated along a spectrum of bony "undercoverage" (dysplasia) and "overcoverage" (femoroacetabular impingement).

A complex combination of both static and dynamic factors impacts hip joint mechanics. A thorough understanding of both the static and dynamic mechanical forces that contribute to hip pain ensures that a proper treatment plan can be created for each patient. Dynamic factors result from abnormal contact and stress between the femoral head and acetabular rim during the terminal ends of hip motion. The abnormal contact and mechanical stresses most often present with reactive hip pain during positions of hip flexion where there is abnormal engagement between the femoral head and acetabulum [1, 2, 5]. Dynamic impingement most often presents with groin pain; however, compensatory changes from restricted hip terminal motion can lead to pain in the lumbar spine, sacroiliac joint, pubic symphysis, or posterior acetabulum [5]. These changes in motion and hip mechanics lead to compensatory changes that result in injury to the surrounding joints and to muscles such as the iliopsoas, rectus femoris, and the adductors. Static factors are due to overload of the chondral surfaces when the patient is in a standing position and the hip is axially loaded. These patients can have pain at rest, although activity-related pain due to overcompensation from surrounding hip musculature is not uncommon.

This chapter focuses on the following concepts:

1. Dynamic factors
   - (A) Loss of offset and asphericity of the femoral head-neck junction (cam-type lesions)
   - (B) Acetabular overcoverage
     - (i) Focal rim impingement lesion
       - (a) Cephalad retroversion (focal rim lesion)
       - (b) True acetabular retroversion
       - (c) Anterior inferior iliac spine (AIIS) impingement
     - (ii) Global overcoverage of the acetabulum – profunda and protrusio
   - (C) Femoral retroversion
2. Extra-articular impingement
   - (A) Coxa vara/trochanteric impingement
   - (B) Ischiofemoral impingement
3. Static factors
   - (A) Anterior or lateral undercoverage of acetabulum (dysplasia)
   - (B) Femoral anteversion
   - (C) Femoral valgus
4. Combined patterns
   - (A) Impingement with femoral retroversion
   - (B) Acetabular dysplasia with femoroacetabular impingement (FAI)

Dynamic Factors

Cam Lesions (Loss of Femoral Head-Neck Junction and Loss of Offset)

The loss of normal contour and femoral head-neck offset is a common source of prearthritic hip pain [6]. Beck et al. [7] described the process of cam-type impingement as a process in which the abnormal bone in the nonspherical portion of the femoral head can cause shear forces to the anterosuperior acetabular cartilage with resultant separation of the labrum from adjacent cartilage within the acetabulum during the terminal ranges of hip motion. Repetitive engagement of the cam lesion with the acetabulum, typically in deeper degrees of flexion and internal rotation, leads to a characteristic pattern of injury to the transition zone articular cartilage (Fig. 1). This results in chondral delamination that can progress with further loss of joint space over time [8]. The adjacent

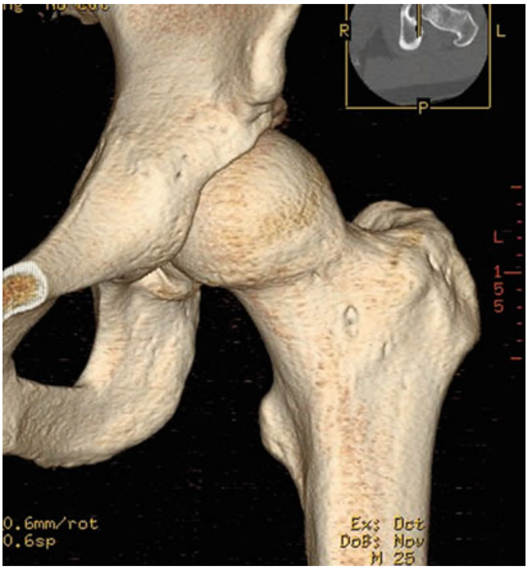


Fig. 1 Three-dimensional computed tomography reconstruction of left hip with prominent cam lesion as cause of femoroacetabular impingement. Note the loss of offset of typical anterosuperior location of femoral head-neck junction

labrum is also at risk to develop secondary degenerative changes and tears [8–10]. These cartilage lesions initially involve the non-weight-bearing portions of the femoral head; however, when large enough, the spherical (weight-bearing area) may become involved [9]. Several surgical options are available to treat these cartilage lesions, including simple debridement, abrasion chondroplasty, or microfracture; however, the long-term prevention of osteoarthritis from these interventions remains to be determined [6, 9, 11, 12]. The labral degeneration and tears predictably occur based on the location of the cam lesions, as the labrum adjacent to the cam lesion is compressed between the aspherical femoral head and acetabular rim. The tears are common at the transition zone cartilage, as opposed to intrasubstance tears, which is thought to be favorable for healing due to the improved vascular supply from the capsule at these more peripheral locations [9].

A recent review of 113 patients who had symptomatic cam-type impingement demonstrated that bilateral cam-type deformity was present in 88 patients (77.9 %), while only 23 (26.1 %) patients demonstrated pain in both hips [13]. A higher alpha angle was associated with a significantly higher percentage of symptoms (69.9° vs. 63.1°, $p < 0.001$) [13]. Johnston et al. [14] recently evaluated the relationship between the size of the cam lesion and damage to the labrum and articular cartilage. In 82 patients who eventually underwent operative intervention, a higher alpha angle was associated with full-thickness cartilage delamination and greater detachment of the base of the labrum (Fig. 2).

The orthopedic surgeon can address cam lesions and the loss of head-neck offset with either arthroscopic or open approaches [15]. Mardones et al. [16] evaluated both open and arthroscopic femoral head-neck osteoplasty in a cadaveric model to determine any variations in efficacy of bony resection between the two approaches. The authors concluded that either method results in improved head-neck offset, although procedure time was significantly shorter in the open group. While depth and width of the osteoplasty were reliable with an arthroscopic approach, there was a tendency to underestimate the length of bony resection needed.

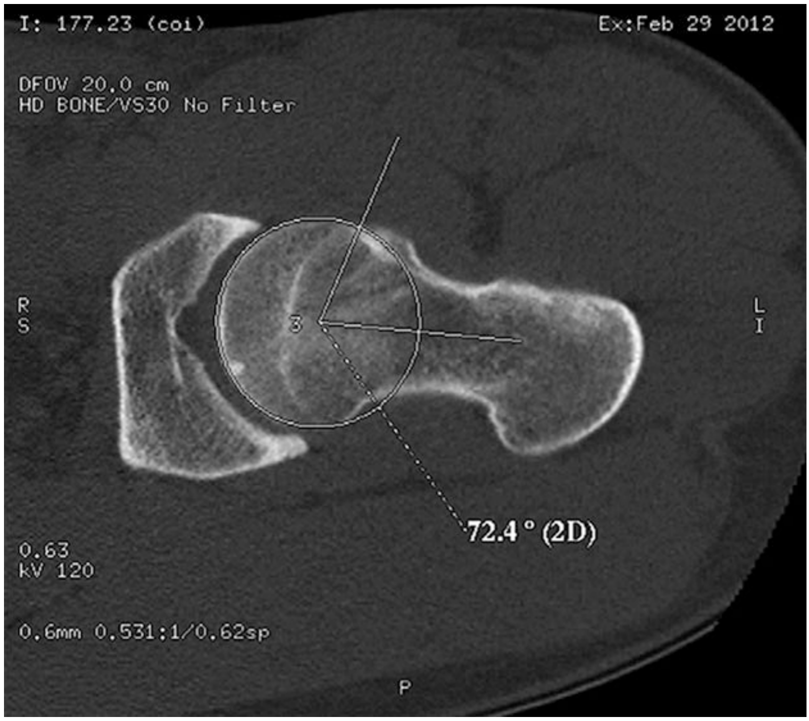
Acetabular Overcoverage

Focal Rim Impingement

Cephalad retroversion of the acetabulum, or a focal rim lesion, is another dynamic factor that is responsible for hip pain in the younger patient. The abnormal bone on the acetabular rim comes into contact with the normal femoral neck and results in tearing of the anterosuperior labrum. Focal rim lesions can be identified in anteroposterior (AP) radiographs as a crossover sign (Fig. 3). Other radiographic findings that may indicate impingement include the presence of os acetabuli or fractures of the acetabular rim, ossification of the labrum, and a pincer groove or trough sign laterally on the femoral head-neck junction. Patients with developmental dysplasia and Legg-Calve-Perthes disease may be more likely to have focal or global acetabular retroversion, or focal rim lesions [17].

Labral injury in these patients is often seen as intrasubstance fissuring and can be difficult to

Fig. 2 Axial computed tomography image of patient with cam-type femoroacetabular impingement and abnormal alpha angle (72.4°)



repair due to the poor tissue quality from repetitive impingement between the femoral head-neck and the acetabular rim. As the labral tissue undergoes degeneration, bony apposition increases between the femoral head and the anterosuperior acetabulum. Eventually, the labrum undergoes ossification and callus may form at sites of repetitive contact [7, 9]. Chronic impingement also creates a "contrecoup" pattern of cartilage loss within the posteroinferior edge of the acetabulum due to levering of the head on the overhanging acetabular rim with subsequent shearing of the acetabular cartilage [18, 19].

Byrd and Jones [20] have demonstrated good results with arthroscopic treatment of pincer-type lesions at 2-year follow-up. In their study of 100 patients, 79 % had good-to-excellent results with an overall median improvement in Harris Hip Scores (HHS) of 21.5 points. Zumstein et al. [21] performed a cadaveric study demonstrating that focal rim lesions located more posterosuperiorly are less accurately resected during hip arthroscopy performed through anterior or anterolateral portals due to difficulty in identifying the posterior starting point for resection.

The anterior inferior iliac spine (AIIS) prominence has recently been recognized as a cause of extra-articular impingement. Abnormal morphology of the AIIS is a form of focal rim impingement as extra bone formation around the insertion of the rectus femoris is a block to mechanical motion and can serve as a pain generator (Fig. 4). When this abnormal bone is identified as a source of impingement, surgical options in the form of arthroscopic resection exist. Hetsroni et al. [22] recently reported on a series of ten patients who underwent arthroscopic decompression of AIIS-type deformity. AIIS deformity is often not present in isolation, as anterior cam lesions may also be present and addressed at the time of surgery. In nine patients, an anterior cam lesion was also identified and decompressed. Postoperatively, terminal hip flexion improved from 99° to 117°, with an average improvement in modified HHS of 34 points (64–98) [22]. More recently, Hetsroni et al. [23] classified various types of AIIS (subspine) morphology depending on the extent of bony deformity based on computed tomography (CT) and dynamic software analysis. In type 1, the distal AIIS ends proximal to the acetabular rim. Type 2 subspine

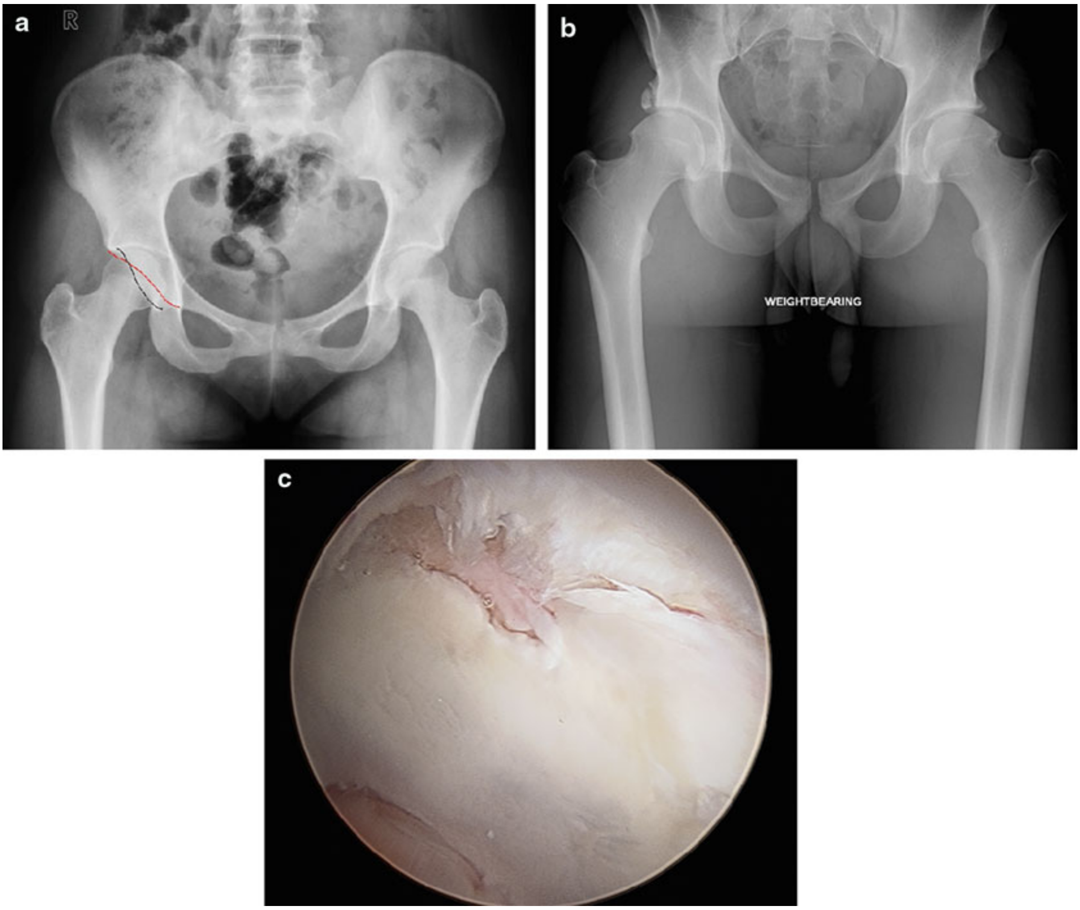


Fig. 3 (**a**) Anteroposterior (*AP*) pelvis radiograph demonstrating a crossover sign where a portion of the anterior acetabular wall lies lateral to the posterior wall causing "overcoverage" and impingement. (**b**) AP pelvis radiograph demonstrating right acetabular rim fracture in patient with pincer-type femoroacetabular impingement (FAI). (**c**) Intraoperative view of patient with cam-type FAI as seen from the anterolateral portal demonstrating area of chondral injury at the chondrolabral junction. Such lesions are characteristic of cam lesions

deformity is described in cases when the AIIS extends up to the acetabular rim, and type 3 subspine deformity extends beyond the acetabular rim. The AIIS can be successfully decompressed arthroscopically; however, the deformity must be clearly delineated preoperatively [23].

Mixed Impingement

Mixed impingement consists of hips with both femoral and acetabular deformities, and it is the most common form of FAI [9, 18]. A large population-based prospective survey of 2,081 young adults (874 males and 1,207 females) demonstrated cam-type deformities in 868 and 1,192 male and female subjects, respectively, with 187 males (21.5 %) and 39 females (3.3 %) demonstrating a pistol grip deformity with focal femoral neck prominence [24]. Pincer deformities were also seen in this group of males and females. Examination of AP radiographs revealed 203 (23.4 %) posterior wall signs, and the crossover sign was seen in 446 males (51.4 %) and 542 females (45.5 %) [24]. It is thus important to recognize the coexistence of femoral and acetabular deformities, and that deformity is often present bilaterally even in patients with only one symptomatic hip. Nepple and colleagues noted similar findings in a retrospective review of

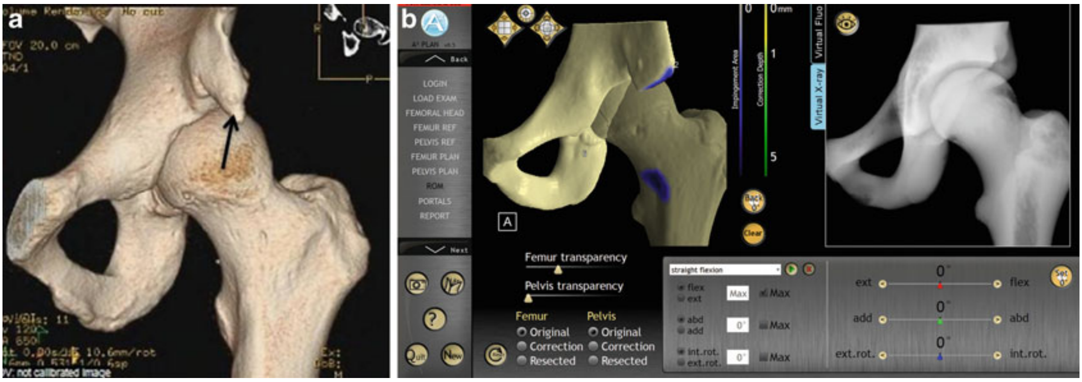


Fig. 4 (**a**) Three-dimensional computed tomography imaging demonstrating abnormal morphology of the anterior inferior iliac spine (AIIS), which is a cause of focal rim impingement due to extra bone formation around the insertion of the rectus femoris. (**b**) This bone is a clear block to mechanical motion, as demonstrated in this computer templating image whereby the anterior inferior neck impinges on the prominent AIIS and can serve as a pain generator, which is a very common finding in subspine impingement

football players with a history of groin pain or injury at the National Football League Combine [25]. Radiographic evidence of isolated cam-type deformity was present in only 12 hips (9.8 %), while isolated pincer-type FAI deformity was seen in 28 hips (22.8 %). There was, however, a high incidence of radiographic pincer- and cam-type FAI in this population (116 of 123 players, 94.3 % of hips). In another recent hospital-based study of asymptomatic patients, a review of radiographs of 522 hips showed a high prevalence of FAI, affecting over 90 % of the cohort. Signs of mixed-type FAI were seen in 82 hips, or 15.7 % of the patients evaluated [26]. Combined impingement lesions present with different patterns of cartilage and labral pathology, and thus it is important to correctly identify and treat all pathology at the time of surgical intervention. It is well reported that in cam-type impingement, the majority of damage to the labrum occurs anterosuperiorly, while in pincer impingement the damage to the labrum and underlying cartilage occurs more circumferentially, although the greatest damage occurs between 11 and 1 o'clock [7]. It is thus important to recognize the coexistence of femoral and acetabular deformities as this has implications for localization of cartilaginous and labral damage, which can be present bilaterally even in patients with only one symptomatic hip.

Global Acetabular Retroversion

Global retroversion of the acetabulum may present with very similar symptomatology to focal rim lesions; however, it represents an entirely different clinical problem of overcoverage anteriorly and undercoverage posteriorly [5]. This clinical entity can be recognized on the preoperative AP pelvis radiograph by the so-called posterior wall sign, whereby the outline of the posterior wall passes medial to the center of the femoral head [27]. Another radiographic sign, the ischial spine sign, also indicates acetabular retroversion and represents a prominent ischial spine as seen on the AP pelvis radiograph (Fig. 5) [28]. This combination of anterior overcoverage and posterior undercoverage can predispose these patients to posterior instability or dislocation. Surgical treatments that involve aggressive anterior rim decompression may put the patient at higher risk by creating global undercoverage with resultant instability. Although rare, symptomatic posterior instability can be addressed surgically with a reverse "anteverting" periacetabular osteotomy [7].

Global Acetabular Overcoverage

Coxa profunda or protrusio deformities are characterized by a deeper-than-normal acetabulum. Radiographic evaluation of the normal hip on an AP pelvis radiograph demonstrates a teardrop that is lateral to the ilioischial line. However, in cases

Fig. 5 Standard (anteroposterior) radiograph of patient with femoroacetabular impingement, demonstrating crossover sign and bilateral retroverted acetabula as evidenced by prominent ischial spines bilaterally (*black outline*)

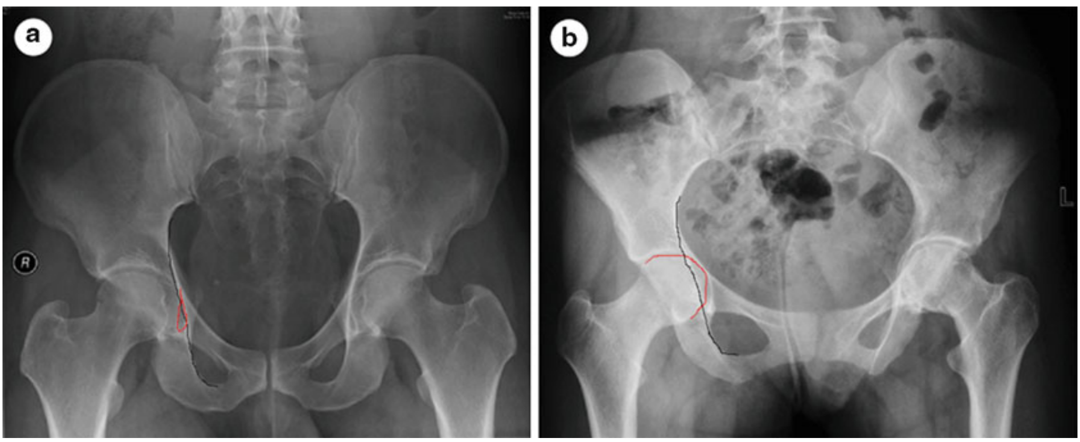


Fig. 6 (**a**) Anteroposterior pelvis radiograph showing deeper-than-normal acetabulum with medicalization of the teardrop (*red outline*) to the ilioischial line (*black line*). (**b**) Protrusio deformity results from additional medicalization of the femoral head (*red outline*) medial to the ilioischial line (*black line*)

of coxa profunda and protrusio, the teardrop or femoral head touches or crosses medial to the ilioischial line, respectively (Fig. 6). As a result of the altered acetabular morphology, the medial joint space is more susceptible to osteoarthritis from altered load transmission patterns, whereas the superior joint space initially remains unaffected [29, 30]. Leunig et al. [29] recently described a number of important morphologic findings in patients with protrusio-related osteoarthritis of the hip. Compared to patients without protrusio deformity who develop hip osteoarthritis, those with protrusio demonstrate significantly decreased medial joint space with increased superior joint space. In addition, the ilioischial line was lateral to the acetabular fossa in patients with protrusio, and the neck-shaft angle was substantially lower in this group than in the osteoarthritis control group (121° vs. 130°). Lastly, a "contrecoup lesion" was identified as a significant cause of osteoarthritis in the posteroinferior joint in hips with protrusio deformity and was proposed to initiate the process of cartilage degeneration [29].

Although global acetabular overcoverage can cause symptoms that are similar to focal rim lesions, the pattern of impingement is different and instead occurs circumferentially around the acetabular rim. Therefore, careful review of preoperative radiographs is necessary to recognize this pattern and allow the treating surgeon to understand that arthroscopic rim resection may address only a small portion of the mechanical zone of impingement. Some authors have recommended a surgical hip dislocation to address global overcoverage, whereas others have described variation of dual-portal arthroscopy to address the pincer impingement [29, 31]. In addition to performing an acetabular rim trim with labral refixation, some hips also require an osteochondroplasty of the head-neck junction to improve femoral head-neck offset and restore the normal concavity. Also, depending on articular cartilage damage present, a valgus intertrochanteric or pelvic-sided osteotomy may be indicated to shift the weight-bearing zone and prevent further degeneration of the articular cartilage [9, 32].

Femoral Retroversion

Retroversion of the femur has been described as a distinct dynamic factor that can cause mechanical hip pain in the young patient. In the adult male, the mean femoral anteversion angle measures approximately 15° [17]. Relative (<15°) or absolute (<0°) retroversion can increase functional external rotation while reducing the amount of hip internal rotation. Patients with both cam-type impingement and femoral retroversion will have pain earlier in the hip flexion arc of motion, as the retroverted femur rotates the cam lesion into the socket at lower angles of hip flexion. This places patients at higher risk for impingement symptoms during daily activities like sitting at a desk or getting in and out of a car [1, 5]. On the other hand, a patient with excessive femoral anteversion may not have symptoms of impingement until terminal hip flexion and have fewer painful restrictions in motion.

Surgical treatment consisting of cam decompression is usually successful in alleviating pain in patients with a loss of femoral head-neck offset and relative femoral retroversion. Kelly et al. [15] recently presented a series of patients with femoral retroversion who underwent arthroscopic decompression and had an overall improvement in hip range of motion, specifically internal rotation, after arthroscopic surgery. However, when a significant portion of the cam lesion is located more posterolaterally, open surgical dislocation is necessary to perform a complete resection and protect the retinacular vessels [7, 33]. If arthroscopic techniques are utilized for more posterolateral lesions, the hip should be placed in traction and extension to more safely visualize this area [34]. Isolated femoral retroversion, in the absence of concurrent cam or pincer lesions, can be treated with a femoral derotational osteotomy.

Extra-articular Impingement

Coxa Vara and Trochanteric-Pelvic Impingement

Coxa vara is another dynamic factor that must be recognized in the evaluation of mechanical hip pain and can be the result of multiple etiologies, including slipped femoral epiphysis, Perthes disease, trauma, or previous infection. It also may be caused by a developmental abnormality of the femur [35–37]. Femoral varus results in a relative shortening of the femoral neck and prominence of the greater trochanter as a result of a reduced neck-shaft angle. This can result in extra-articular lateral impingement of the greater trochanter on the AIIS (Fig. 7). Greater trochanteric impingement against the pelvis is classically seen in the setting of coxa vara and in individuals with a prominent greater trochanter [38]. The version of the femur is important to recognize when considering the diagnosis of greater trochanteric impingement. In the setting of femoral retroversion, patients have a functional decrease in internal rotation [1, 5], which can manifest itself as anterior trochanteric impingement with deeper degrees of flexion and internal rotation. Similarly, those patients with femoral anteversion have a functional increase in internal rotation and may

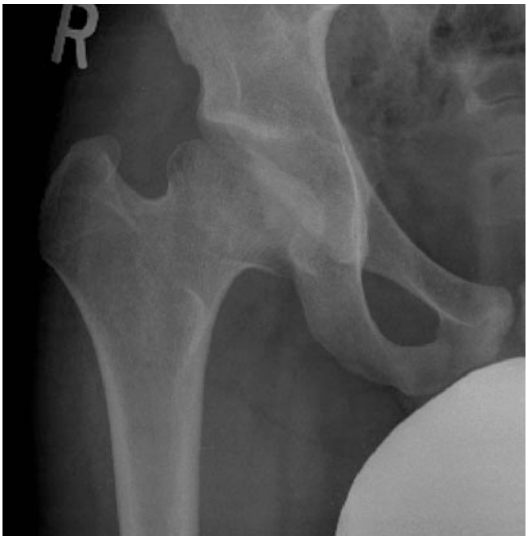


Fig. 7 Anteroposterior image of a right hip demonstrating coxa vara, which results in relative shortening of the femoral neck and prominence of the greater trochanter. This prominence leads to extra-articular lateral impingement of the greater trochanter on the anterior inferior iliac spine

manifest symptoms of posterior trochanteric impingement with external rotation. In cases of mild coxa vara, osteoplasty of the cam lesion and/or acetabular rim lesion is usually sufficient to resolve the impingement; however, in cases of significant varus deformity (<125°), a more extensive procedure such as relative femoral neck lengthening with a trochanteric osteotomy and distal trochanteric advancement may be required to address the severity of the mechanical impingement [32].

Ischiofemoral Impingement

Ischiofemoral impingement is a less commonly described source of extra-articular impingement and is caused by impingement between the ischium and femur, often in the setting of previous operations or trauma [39, 40]. Radiographic imaging often demonstrates abnormality of the quadratus femoris muscle, which becomes injured as a result of impingement between the ischial tuberosity and lesser trochanter [39]. A recent cadaveric study evaluating quadratus femoris injury within the ischiofemoral space found that while degenerative changes were present within the majority the muscles analyzed, there was no association between injury and degeneration with the size of the ischiofemoral space [41]. These patients typically have pain with hip extension as well as pain that can radiate toward the knee and may be the result of prior myotendinous injuries at the ischial tuberosity from proximal hamstring injury [42, 43] (Fig. 8). Initial treatment should be focused on conservative measures, and surgical decompression at the level of the ischial tuberosity and lesser trochanter is reserved for cases where nonoperative measures fail to relieve pain [42, 44].

Static Factors

Acetabular Dysplasia

Hip dysplasia is a condition in which the hip joint develops incorrectly during early infancy and childhood, with eventual abnormal morphology of the acetabulum, femoral head, or both [45]. As a result, anterior or lateral undercoverage of the acetabulum can lead to elevated contact pressures toward the posterosuperior rim of the acetabulum (Fig. 9). This reduced contact area contributes to premature cartilage and labral degeneration and ultimately osteoarthritis [46–49]. Another complicating factor is that global undercoverage results in structural instability and allows the femoral head to migrate into regions of acetabular deficiency. These recurrent subluxation events can also lead to degeneration and chondral injury [50].

Acetabular redirection osteotomies, including periacetabular osteotomy (PAO), are technically challenging procedures for treatment of painful hip dysplasia that can greatly improve patient function [46]. The goals of PAO surgery include reorientation of the acetabulum to distribute contact pressures more equally across the weight-bearing surface and to better contain the femoral head within the acetabulum while preventing subluxation events [10, 46, 51–58]. Preoperative CT is necessary to adequately plan surgery to correct the relationship of the abnormal femoral head and

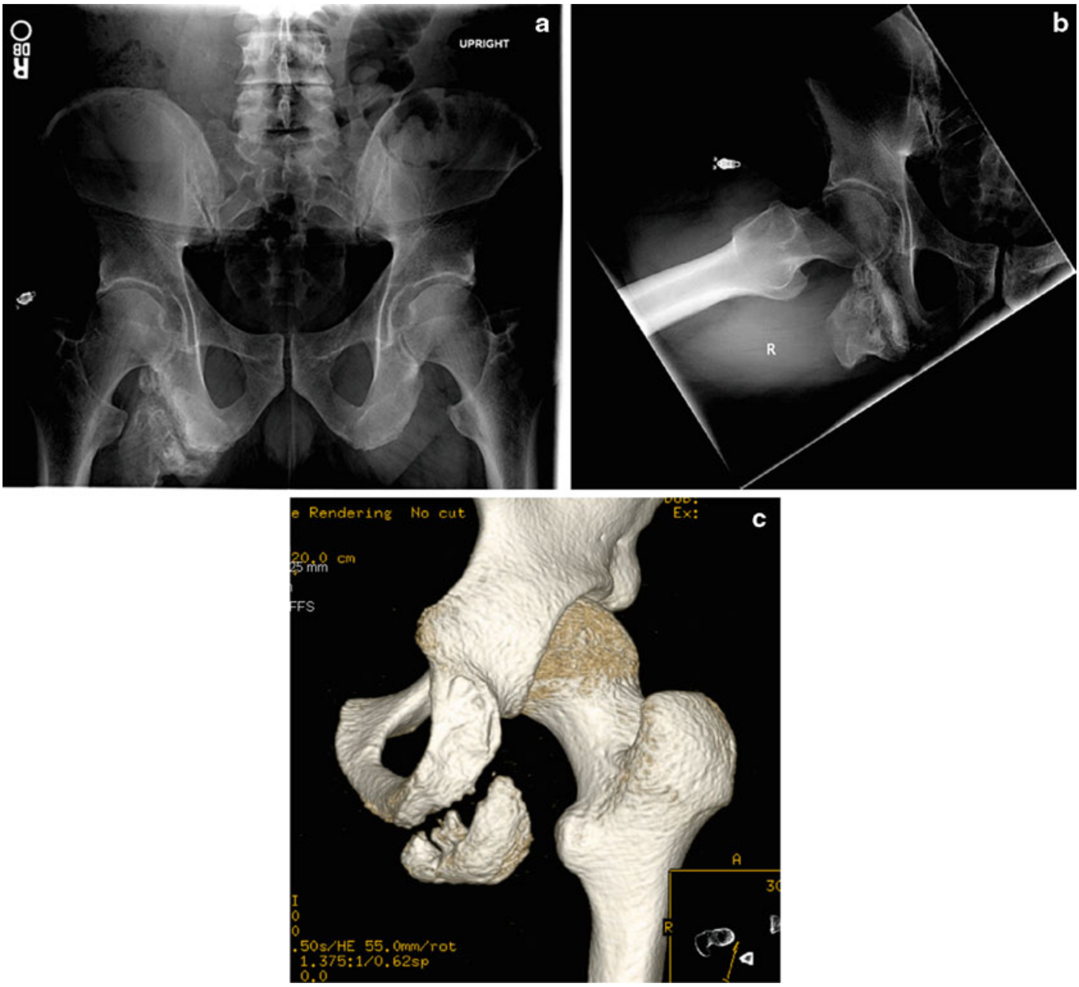


Fig. 8 Ischiofemoral impingement is commonly seen in the setting of previous surgery or trauma. (**a**) Anteroposterior pelvis radiograph demonstrating significantly diminished clearance between the ischium and proximal femur. (**b**) Lateral radiograph demonstrating significantly decreased space between posteroinferior proximal femur and the abnormal ischial bone, resulting in a significantly limited arc of motion and impingement. (**c**) Three-dimensional computed tomography image of previous bony avulsion injury of the ischial spine in a patient with ischiofemoral impingement

acetabulum. When performed on a suitable patient, correction of hip morphology through osteotomy can lead to more equal contact distribution on the articular cartilage and improved coverage of the hip socket. Associated femoral deformity is also not uncommon in patients with dysplasia, and concomitant procedures such as femoral head-neck osteochondroplasty, trochanteric advancement procedures, femoral neck lengthening, and varus or valgus proximal femoral osteotomies may be indicated to optimize hip mechanics [53, 54, 59].

Excessive Femoral Anteversion

On average, femoral anteversion ranges from 30° to 40° at birth and decreases progressively throughout growth to a mean anteversion angle of 9° [60, 61]. Anteversion angles greater than 15° should be considered abnormal and can cause mechanical hip pain. A recent CT study suggests that femoral anteversion can be found in isolation or with other associated hip morphology such as global acetabular deficiency associated with

Fig. 9 Anteroposterior radiograph of patient with acetabular undercoverage (dysplasia) of the right hip. The reduced weight-bearing surface of the femoral head leads to static overload and increased contact stresses with eventual cartilage damage

dysplasia [62]. These patients clinically demonstrate a functional increase in hip joint internal rotation with a corresponding decrease in external rotation, placing them at risk for developing both intra-articular and extra-articular symptomatic impingement [15, 63–65]. During simultaneous hip flexion and external rotation, extra-articular impingement from the greater trochanter engagement of the pelvis can occur. Similarly, during hip extension and simultaneous external rotation, the femoral neck and acetabular rim can engage causing intra-articular impingement. Fabricant et al. [66] recently investigated 67 patients with coxa saltans after arthroscopic treatment, including psoas release and acetabular rim decompression, showing inferior clinical outcomes in the setting of increased femoral anteversion due to the chronic overload of the anterior stabilizers of the hip joint. The authors suggest that patients with femoral anteversion greater than 30° should be considered for a derotational femoral osteotomy [66].

Femoral Valgus

Coxa valga is defined as a neck-shaft angle greater than 135° and is commonly seen in combination with femoral anteversion, acetabular dysplasia, or neuromuscular conditions [59]. Static overload of the anterosuperior head and dome may precipitate hip pain in this patient population and eventually lead to early degeneration of articular cartilage. In a study by Siebenrock et al. [67], the authors utilized CT scan reconstructions to evaluate the range of motion and impingement patterns in patients with coxa valga. They concluded that those patients with increased antetorsion are predisposed to posterior extra-articular FAI as well as anteroinferior spine impingement [67]. Patients with refractory symptoms from femoral valgus may benefit from a varus derotational osteotomy [63].

Combined Patterns

Femoroacetabular impingement can be caused by a combination of static and dynamic morphologic abnormalities. Relative or absolute femoral retroversion in association with cam or pincer deformities can alter the hip arc of motion, so that reduced amounts of hip flexion and internal rotation are necessary for engagement of the cam or pincer deformity. These patients must be considered for arthroscopic or open procedure depending on the degree of retroversion and size of cam or pincer deformity. Similarly, dysplasia can be seen in association with femoroacetabular impingement, although it remains uncommon. This complex hip deformity should be addressed with a periacetabular osteotomy combined with open or arthroscopic femoral procedures to provide comprehensive deformity correction [54].

Compensatory Injury Patterns

Alterations in static and dynamic hip joint mechanics can cause a change in various forces across the pelvis that can then predispose the patient to compensatory injuries. Anterior enthesopathy can result in hip flexor strains, psoas impingement, or subspine impingement from prior muscle injury or avulsion. Medial enthesopathy often involves injury to the adductor complex in the form of athletic pubalgia or osteitis pubis. In addition, the "sports hip triad" has been described and involves labral tearing, adductor strain, and rectus strain [5]. Injuries to the posterior structures of the pelvis can eventually result, including proximal hamstring strains, deep gluteal syndrome (sciatic nerve entrapment), or dysfunction of the sacroiliac joint. Similarly, the lateral structures of the pelvis and hip can lead to compensatory injury to the abductor complex or iliotibial band. These compensatory injury patterns are often recurrent unless treatment involves concomitant surgery for the underlying static and dynamic morphologic hip abnormality [68].

Summary

A thorough understanding of the complex constellation of static and dynamic factors that contribute to mechanical abnormalities and alterations in the normal hip arc of motion and loading patterns is imperative for the treating physician when evaluating younger patients with prearthritic hip. Compensatory changes within the hip joint and surrounding musculature can lead to additional pathology that must be recognized at the time of treatment to ensure the overall best outcome for patients. A combination of history, physical examination, and appropriate imaging is paramount for the treating physician to accurately diagnose and surgically treat this patient population.

References

1. Bedi A, Kelly BT. Femoroacetabular impingement. J Bone Joint Surg Am. 2013;95A(1):82–92.
2. Bedi A, Kelly BT, Khanduja V. Arthroscopic hip preservation surgery: current concepts and perspective. Bone Joint J. 2013;95B(1):10–9.
3. Knesek MJ, Skendzel JG, Kelly BT, Bedi A. Approach to the patient evaluation using static and dynamic hip pathomechanics. Oper Tech Sports Med. 2012;20(4):260–72.
4. Dolan MM, Heyworth BE, Bedi A, Duke G, Kelly BT. CT reveals a high incidence of osseous abnormalities in hips with labral tears. Clin Orthop Relat Res. 2011;469(3):831–8.
5. Bedi A, Dolan M, Leunig M, Kelly BT. Static and dynamic mechanical causes of hip pain. Arthroscopy. 2011;27(2):235–51.
6. Clohisy JC, Knaus ER, Hunt DM, Lesher JM, Harris-Hayes M, Prather H. Clinical presentation of patients with symptomatic anterior hip impingement. Clin Orthop Relat Res. 2009;467(3):638–44.
7. Beck M, Kalhor M, Leunig M, Ganz R. Hip morphology influences the pattern of damage to the acetabular cartilage – femoroacetabular impingement as a cause of early osteoarthritis of the hip. J Bone Joint Surg Br. 2005;87B(7):1012–8.
8. Leunig M, Beck M, Kalhor M, Kim YJ, Werlen S, Ganz R. Fibrocystic changes at anterosuperior femoral neck: prevalence in hips with femoroacetabular impingement. Radiology. 2005;236(1):237–46.
9. Ganz R, Leunig M, Leunig-Ganz K, Harris WH. The etiology of osteoarthritis of the hip – an integrated mechanical concept. Clin Orthop Relat Res. 2008;466(2):264–72.
10. Nepple JJ, Zebala LP, Clohisy JC. Labral disease associated with femoroacetabular impingement: do we need to correct the structural deformity? J Arthroplasty. 2009;24(6):114–9.
11. Larson CM, Giveans MR. Arthroscopic debridement versus refixation of the acetabular labrum associated with femoroacetabular impingement. Arthroscopy. 2009;25(4):369–76.
12. Larson CM, Giveans MR, Stone RM. Arthroscopic debridement versus refixation of the acetabular labrum associated with femoroacetabular impingement mean 3.5-year follow-up. Am J Sports Med. 2012;40(5):1015–21.
13. Allen D, Beaule PE, Ramadan O, Doucette S. Prevalence of associated deformities and hip pain in patients with cam-type femoroacetabular impingement. J Bone Joint Surg Br. 2009;91B(5):589–94.
14. Johnston TL, Schenker ML, Briggs KK, Philippon MJ. Relationship between offset angle alpha and hip chondral injury in femoroacetabular impingement. Arthroscopy. 2008;24(6):669–75.
15. Kelly BT, Bedi A, Robertson CM, Dela Torre K, Giveans MR, Larson CM. Alterations in internal rotation and alpha angles are associated with arthroscopic cam decompression in the hip. Am J Sports Med. 2012;40(5):1107–12.
16. Mardones R, Lara J, Donndorff A, et al. Surgical correction of "cam-type" femoroacetabular impingement: a cadaveric comparison of open versus arthroscopic debridement. Arthroscopy. 2009;25(2):175–82.
17. Ezoe M, Naito M, Inoue T. The prevalence of acetabular retroversion among various disorders of the hip. J Bone Joint Surg Am. 2006;88A(2):372–9.
18. Ganz R, Parvizi J, Beck M, Leunig M, Notzli H, Siebenrock KA. Femoroacetabular impingement – a cause for osteoarthritis of the hip. Clin Orthop Relat Res. 2003;417:112–20.
19. Ito K, Minka-Ii MA, Leunig M, Werlen S, Ganz R. Femoroacetabular impingement and the cam-effect – a MRI-based quantitative anatomical study of the femoral head-neck offset. J Bone Joint Surg Br. 2001;83B(2):171–6.

20. Byrd JWT, Jones KS. Arthroscopic management of femoroacetabular impingement: minimum 2-year follow-up. Arthroscopy. 2011;27(10):1379–88.
21. Zumstein M, Hahn F, Sukthankar A, Sussmann PS, Dora C. How accurately can the acetabular rim be trimmed in hip arthroscopy for pincer-type femoral acetabular impingement: a cadaveric investigation. Arthroscopy. 2009;25(2):164–8.
22. Hetsroni I, Larson CM, Dela Torre K, Zbeda RM, Magennis E, Kelly BT. Anterior inferior iliac spine deformity as an extra-articular source for hip impingement: a series of 10 patients treated with arthroscopic decompression. Arthroscopy. 2012;28(11):1644–53.
23. Hetsroni I, Poultsides L, Bedi A, Larson CM, Kelly BT. Anterior inferior iliac spine morphology correlates with hip range of motion: a classification system and dynamic model. Clin Orthop Relat Res. 2013;471 (8):2497–503.
24. Laborie LB, Lehmann TG, Engester IO, Eastwood DM, Engester LB, Rosendahl K. Prevalence of radiographic findings thought to be associated with femoroacetabular impingement in a population-based cohort of 2081 healthy young adults. Radiology. 2011;260(2):494–502.
25. Nepple JJ, Brophy RH, Matava MJ, Wright RW, Clohisy JC. Radiographic findings of femoroacetabular impingement in National Football League Combine athletes undergoing radiographs for previous hip or groin pain. Arthroscopy. 2012;28(10):1396–403.
26. de Bruin F, Reijnierse M, Farhang-Razi V, Bloem JL. Radiographic signs associated with femoroacetabular impingement occur with high prevalence at all ages in a hospital population. Eur Radiol. 2013;23(11):3131–9.
27. Jamali AA, Mladenov K, Meyer DC, et al. Anteroposterior pelvic radiographs to assess acetabular retroversion: high validity of the "cross-over-sign". J Orthop Res. 2007;25(6):758–65.
28. Kakaty DK, Fischer AF, Hosalkar HS, Siebenrock KA, Tannast M. The ischial spine sign does pelvic tilt and rotation matter? Clin Orthop Relat Res. 2010;468 (3):769–74.
29. Leunig M, Nho SJ, Turchetto L, Ganz R. Protrusio acetabuli: new insights and experience with joint preservation. Clin Orthop Relat Res. 2009;467(9):2241–50.
30. Maquet P, Anh TV. On the forces exerted on the hip during gait. Arch Orthop Trauma Surg. 1981;99(1):53–8.
31. Matsuda DK. Protrusio acetabuli: contraindication or indication for hip arthroscopy? And the case for arthroscopic treatment of global pincer impingement. Arthroscopy. 2012;28(6):882–8.
32. Turgeon TR, Phillips W, Kantor SR, Santore RF. The role of acetabular and femoral osteotomies in reconstructive surgery of the hip and beyond. Clin Orthop Relat Res. 2005;(441):188–99.
33. Beck M, Leunig M, Parvizi J, Boutier V, Wyss D, Ganz R. Anterior femoroacetabular impingement Part II. Midterm results of surgical treatment. Clin Orthop Relat Res. 2004;418:67–73.
34. Philippon MJ, Stubbs AJ, Schenker ML, Maxwell RB, Ganz R, Leunig M. Arthroscopic management of femoroacetabular impingement – osteoplasty technique and literature review. Am J Sports Med. 2007;35(9):1571–80.
35. Eijer H, Podeszwa DA, Ganz R, Leunig M. Evaluation and treatment of young adults with femoroacetabular impingement secondary to Perthes' disease. Hip Int. 2006;16(4):273–80.
36. Wenger DR, Kishan S, Pring ME. Impingement and childhood hip disease. J Pediatr Orthop B. 2006;15 (4):233–43.
37. Tanzer M, Noiseux N. Osseous abnormalities and early osteoarthritis. Clin Orthop Relat Res. 2004;429:170–7.
38. Macnicol MF, Makris D. Distal transfer of the greater trochanter. J Bone Joint Surg Br. 1991;73(5):838–41.
39. Taneja AK, Bredella MA, Torriani M. Ischiofemoral impingement. Magn Reson Imaging Clin N Am. 2013;21(1):65.
40. Lee S, Kim I, Lee SM, Lee J. Ischiofemoral impingement syndrome. Ann Rehabil Med. 2013;37(1):143–6.
41. Sussman WI, Han E, Schuenke MD. Quantitative assessment of the ischiofemoral space and evidence of degenerative changes in the quadratus femoris muscle. Surg Radiol Anat. 2013;35(4):273–81.
42. Tosun O, Cay N, Bozkurt M, Arslan H. Ischiofemoral impingement in an 11-year-old girl. Diagn Interv Radiol. 2012;18(6):571–3.
43. Askling CM, Koulouris G, Saartok T, Werner S, Best TM. Total proximal hamstring ruptures: clinical and MRI aspects including guidelines for postoperative rehabilitation. Knee Surg Sports Traumatol Arthrosc. 2013;21(3):515–33.
44. Ali AM, Whitwell D, Ostlere SJ. Case report: imaging and surgical treatment of a snapping hip due to ischiofemoral impingement. Skeletal Radiol. 2011;40 (5):653–6.
45. Dezateux C, Rosendahl K. Developmental dysplasia of the hip. Lancet. 2007;369(9572):1541–52.
46. Hipp JA, Sugano N, Millis MB, Murphy SB. Planning acetabular redirection osteotomies based on joint contact pressures. Clin Orthop Relat Res. 1999;364:134–43.
47. Mavcic B, Iglic A, Kralj-Iglic V, Brand RA, Vengust R. Cumulative hip contact stress predicts osteoarthritis in DDH. Clin Orthop Relat Res. 2008;466(4):884–91.
48. Millis MB, Murphy SB. Use of computed tomographic reconstruction in planning osteotomies of the hip. Clin Orthop Relat Res. 1992;274:154–9.
49. Russell ME, Shivanna KH, Grosland NM, Pedersen DR. Cartilage contact pressure elevations in dysplastic hips: a chronic overload model. J Orthop Surg Res. 2006;1:6.
50. Jager M, Wild A, Westhoff B, Krauspe RD. Femoroacetabular impingement caused by a femoral osseous head-neck bump deformity: clinical, radiological, and experimental results. J Orthop Sci. 2004;9(3):256–63.
51. Clohisy JC, Keeney JA, Schoenecker PL. Preliminary assessment and treatment guidelines for hip disorders

in young adults. Clin Orthop Relat Res. 2005;441:168–79.
52. Schoenecker PL, Clohisy JC, Millis MB, Wenger DR. Surgical management of the problematic hip in adolescent and young adult patients. J Am Acad Orthop Surg. 2011;19(5):275–86.
53. Clohisy JC, Schutz AL, John LS, Schoenecker PL, Wright RW. Periacetabular osteotomy: a systematic literature review. Clin Orthop Relat Res. 2009;467 (8):2041–52.
54. Clohisy JC, St John LC, Nunley RM, Schutz AL, Schoenecker PL. Combined periacetabular and femoral osteotomies for severe hip deformities. Clin Orthop Relat Res. 2009;467(9):2221–7.
55. Hsieh PH, Huang KC, Lee PC, Chang YH. Comparison of periacetabular osteotomy and total hip replacement in the same patient: a two- to ten-year follow-up study. J Bone Joint Surg Br. 2009;91B(7):883–8.
56. Millis MB, Murphy SB. The Boston experience. Periacetabular osteotomy and anterior arthrotomy via an abductor-sparing surgical approach. Orthopade. 1998;27(11):751–8.
57. Nehme A, Trousdale R, Tannous Z, Maalouf G, Puget J, Telmont N. Developmental dysplasia of the hip: is acetabular retroversion a crucial factor? Orthop Traumatol Surg Res. 2009;95(7):511–9.
58. Peters CL, Erickson JA, Hines JL. Early results of the Bernese periacetabular osteotomy: the learning curve at an academic medical center. J Bone Joint Surg Am. 2006;88A(9):1920–6.
59. Clohisy JC, Nunley RM, Carlisle JC, Schoenecker PL. Incidence and characteristics of femoral deformities in the dysplastic hip. Clin Orthop Relat Res. 2009;467(1):128–34.
60. Ejnisman L, Philippon MJ, Lertwanich P, et al. Relationship between femoral anteversion and findings in hips with femoroacetabular impingement. Orthopedics. 2013;36(3):E293–300.
61. Tayton E. Femoral anteversion – a necessary angle or an evolutionary vestige? J Bone Joint Surg Br. 2007;89B(10):1283–8.
62. Akiyama M, Nakashima Y, Fujii M, et al. Femoral anteversion is correlated with acetabular version and coverage in Asian women with anterior and global deficient subgroups of hip dysplasia: a CT study. Skeletal Radiol. 2012;41(11):1411–8.
63. Atecs Y, Omeroglu H. Varus derotational osteotomy for persistent dysplasia in CDH. J Bone Joint Surg Br. 1996;78B(5):853.
64. Guenther KP, Thielemann F, Hartmann A, Bernstein P. Combined hip-dysplasia and femoroacetabular impingement. Diagnosis and simultaneous surgical treatment. Orthopade. 2008;37(6):577–86.
65. Audenaert EA, Peeters I, Vigneron L, Baelde N, Pattyn C. Hip morphological characteristics and range of internal rotation in femoroacetabular impingement. Am J Sports Med. 2012;40(6):1329–36.
66. Fabricant PD, Bedi A, De La Torre K, Kelly BT. Clinical outcomes after arthroscopic psoas lengthening: the effect of femoral version. Arthroscopy. 2012;28(7):965–71.
67. Siebenrock KA, Steppacher SD, Haefeli PC, Schwab JM, Tannast M. Valgus hip with high antetorsion causes pain through posterior extraarticular FAI. Clin Orthop Relat Res. 2013;471(12):3774–80.
68. Larson CM, Pierce BR, Giveans MR. Treatment of athletes with symptomatic intra-articular hip pathology and athletic pubalgia/sports hernia: a case series. Arthroscopy. 2011;27(6):768–75.

Skeletally Mature Acetabular Dysplasia: Anatomy, Pathomorphology, Pathomechanics, Clinical Presentation, and Imaging Studies

43

Jesse Chrastil, Christopher Pelt, Jill Erickson, and Christopher Peters

Contents

Abstract

Acetabular dysplasia is a developmental disorder that affects approximately 4 % of the population and can lead to hip pain in the young adult. Over the last decade, the field of hip preservation has evolved with an increased understanding of the underlying pathomorphology and biomechanics of dysplasia. Significant advancements have also been made in the surgical treatment. The treating physician should be well versed in the clinical exam findings and pertinent history to accurately make a diagnosis. There is often significant delay in patient presentation to final diagnosis. Plain radiographs provide significant information and can confirm the diagnosis of dysplasia. Advanced imaging may provide additional information about intra-articular processes and can be useful for preoperative planning. Goals of the surgical treatment of acetabular dysplasia include improving acetabular coverage, medializing the center of rotation, decreasing the joint reactive forces at the hip, and improving overall joint congruity. Left untreated, acetabular dysplasia and associated deformities may be precursors for hip joint arthrosis, early hip pain, and the need for hip arthroplasty in the young active patient.

Introduction

Acetabular dysplasia, defined as a volumetrically deficient and/or maloriented acetabulum, is a cause of hip pain and dysfunction in young

J. Chrastil (✉) • C. Pelt • J. Erickson • C. Peters
University of Utah Health Care Orthopaedic Center, Salt Lake City, UT, USA
e-mail: jchrastil@hotmail.com; chris.pelt@hsc.utah.edu; jill.erickson@hsc.utah.edu; chris.peters@hsc.utah.edu

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_122

adult patients. The incidence of radiographic acetabular dysplasia in population studies has been reported to be 4.3 % in males and 3.6 % in females [1]. Dysplasia and associated acetabular deformities have been recognized to be potential precursors for development of early hip arthrosis and need for subsequent hip arthroplasty in young adults. Early recognition and treatment may delay or prevent degeneration of the hip joint, increasing quality of life and reducing the need for early arthroplasty.

Hip dysplasia has been recognized as a pathologic entity for nearly 80 years. Wiberg, in 1939, identified an association between dysplasia and hip degeneration, with greater than 25 % of patients with hip osteoarthritis having evidence of premorbid dysplasia [2]. Similarly, Gade, in 1947, attributed 48 % of hip arthrosis patients to underlying hip dysplasia [3]. The understanding of acetabular dysplasia continued to progress with advances in diagnosis and surgical interventions, in effort to postpone or prevent coxarthrosis. In 1965, Le Coeur described the original triple innominate osteotomy (TIO) to improve acetabular congruency and modify the hip biomechanics [4]. This surgical technique was later modified in 1973 by Steel [5], in 1981 by Tönnis [6], and 1982 by Carlioz [7] with the intention to facilitate correction of the acetabular fragment, increase osteotomy stability, and decrease perioperative morbidity.

In the 1980s, Reinhold Ganz and colleagues in Berne, Switzerland, introduced a new technique for acetabular reorientation termed the Bernese periacetabular osteotomy (PAO), which proved to be a major advancement in treatment [8]. Most notably, the PAO preserved the posterior column of the innominate bone, allowing for improved pelvic stability and early patient mobilization and functional recovery. All osteotomies of the PAO could also be performed through a single incision in the supine position, decreasing the morbidity and operative time of the procedure. Because the bone cuts were performed very close to the joint or acetabular center of rotation, the Bernese PAO allowed for significantly greater movement of the acetabular fragment, facilitating the complex multiplanar correction needed in patients with various forms and magnitudes of dysplasia [9]. The Bernese PAO also reliably resulted in medialization of the hip's center of rotation, thus improving the biomechanics of the dysplastic hip joint when compared to previously described techniques. Additionally, the shape of the true pelvis was not significantly altered, which prevented secondary effects on childbirth. The PAO also preserved the vascularity of the acetabular fragment via the inferior gluteal artery, allowing simultaneous hip arthrotomy without concern for further devascularization of the acetabular fragment. These improvements have made the Bernese PAO, and subsequent modifications of the original technique, the most widely utilized osteotomy in the North America and Europe for the surgical treatment of acetabular dysplasia.

PAO in the skeletally mature patient has shown a total hip arthroplasty conversion rate at 10 years of 16 % [10] and 40 % [11] at 20 years postoperatively, suggesting that durable results and the potential to delay or avoid total hip arthroplasty may be expected in appropriately selected patients. Ongoing advances in the early detection and treatment of acetabular dysplasia have continued to show advantages for the skeletally mature patient. With the goal of an enduring result, ideal candidates for hip preservation treatments for acetabular dysplasia are cited to be less than age 40 with minimal arthrosis and good or excellent hip joint congruency. Hip preservation is an evolving field, and with continued advances, we may see further improvements in these outcomes.

Anatomy

Acetabular dysplasia is characterized by a shallow acetabulum with insufficient acetabular coverage and often a lateralized hip center. Dysplasia is classically associated with bone deficiency anteriorly and laterally. There is often an increased abduction angle in the articular slope of the superior weight-bearing zone of the dysplastic acetabulum. Although classic acetabular dysplasia exhibits excessive anteversion, acetabular retroversion has been reported in as many as 40 % of dysplastic hips [12, 13] and has been suggested as a cause of earlier patient pain and morbidity [14]. Identification of retroversion is paramount during preoperative planning, as failure to account for its presence may result in insufficient correction. Iatrogenic femoroacetabular impingement following PAO is felt to be one cause of failure and later need for conversion to total hip arthroplasty [15, 16]. Indeed, contemporary management of acetabular dysplasia with reorientation must consider the potential to move a hip from a state of instability (dysplasia) to a state of impingement (FAI) by virtue of acetabular position alone or in combination with femoral-sided pathomorphology such as lack of femoral head-neck offset.

The pathomechanics associated with acetabular dysplasia are thought to be consistent with hip instability produced by an insufficiently contained femoral head [4, 17]. Dynamic instability of the hip joint results in shear across the chondral surface along with static and dynamic overload of the acetabular rim and labrum. In response to the instability and mechanical overload, the labrum and capsule frequently show signs of hypertrophy and eventual failure [4]. Labral hypertrophy and damage are common [18–20] and related to overload as the labrum shares a portion of the load with the dysplastic weight-bearing dome. Labral damage often occurs anterolaterally, associated with the area of most severe osseous undercoverage.

Extracapsular soft tissues may also become involved. Periarticular musculature becomes contracted and stiff with stabilizer muscles such as the iliopsoas, rectus, adductors, and hamstrings most affected. Though rare, significant limb shortening associated with severe dysplasia and associated subluxation or dislocation may pose additional challenges, with considerations of the surrounding muscles and neurovascular structures being important [21, 22].

Femoral-sided pathomorphology commonly coexists with acetabular dysplasia and may include excessive femoral neck anteversion, coxa valga, and a posteriorly located greater trochanter. The femoral head may be small and aspherical. The femoral canal is frequently narrow and straight with loss of metaphyseal flare, narrower in the medial-lateral than the anterior-posterior planes. The amount of femoral dysplasia often correlates to the degree of acetabular dysplasia. Decreased femoral head-neck offset may represent an additional source of potential femoroacetabular impingement (FAI) and may need to be addressed before, during, or after acetabular reorientation [16]. Concomitant femoral osteotomy is sometimes necessary during hip preservation procedures [23]. When hip replacement is required, these femoral deformities can require alternative modular instrumentation and implants, thus must be recognized during preoperative planning.

Biomechanics

Generalized joint instability, repeated sheer stress across the joint, acetabular rim overload due to a smaller contact area, and increased joint reactive forces due to a relatively lateralized hip center of rotation are the inciting insults to the articular cartilage leading to early degeneration and premature coxarthrosis in the dysplastic hip. Finite element analysis has shown that relatively small incongruencies between femoral and acetabular cartilage largely effect the perceived contact stresses [24]. The aim of reorientation osteotomies is to correct these biomechanics by removing the anomalous load across the acetabular rim, creating a broader surface to distribute weight-bearing forces, medializing the femoral center of rotation, and consequently decreasing the patients' joint reactive forces through the dysplastic acetabula.

In addition to the hyaline cartilage involvement, the labrum also plays an important role in the biomechanics of the dysplastic hip. Acetabular dysplasia leads to an increased incidence of labral hypertrophy, labral calcification, and labral tears [18–20]. Henak at al. utilized finite element analysis to evaluate load across the labrum. The labrum in morphologically normal hips often shares 1–2 % of the transferred load of the hip, while increasing levels of acetabular dysplasia resulted in a two to ninefold increase in labral load [17]. After labral tear, lack of load sharing may accelerate the rate of joint degeneration [18]. Unfortunately labral repair alone, whether open or arthroscopic, is rarely effective in isolation and may accelerate the degenerative process in some cases. The underlying pathomorphology of the hip must be addressed to remove excessive labral stresses and improve native hip survivorship [25, 26].

Clinical Presentation

History

Young patients require a thorough history be performed. Because there is a genetic predisposition for dysplasia, specific questioning for family history of hip problems or early hip replacement surgery in close relatives should be sought [27].

Nunley et al. have shown that 97 % of patients that present with hip dysplasia have insidious and persistent pain with the majority (77 %) having moderate to severe pain on a daily basis. The majority of patients (88 %) stated pain was activity related; however, 59 % endorsed some level of nighttime pain symptoms as well. In this series 72 % described the classic anterior groin pain, and 66 % described pain that was located around the lateral aspect of the hip. Buttock pain was only identified in 18 % and anterior thigh pain in 29 %. Mechanical symptoms of locking, catching, or popping were endorsed in 80 % of patients evaluated [28].

In the presence of mild acetabular dysplasia, the average age at presentation is often late twenties after two to three decades of normal activities with an asymptomatic hip [29]. Often, patients have seen several providers. It can be common for patients to have delays in diagnosis, inaccurate diagnoses, and ineffective treatments for hernias or muscle strains. One series reported the mean time from the onset of symptoms until the definitive diagnosis of acetabular dysplasia was over 5 years [28].

In 1991 Klaue et al. coined the phrase “the acetabular rim syndrome.” His report describes pain caused by overloading the outer rim and labrum of the acetabulum causing the patient vague and sometimes sharp groin pains. Often the pain, which may be a sharp stabbing sensation in the groin, is associated with clinical instability and various degrees of hip subluxation initially. Later, degenerative pain caused by labral and chondral injuries, possible rim fractures, and cystic degeneration may ensue [19]. The most common clinical presentation is a chief complaint of groin pain that increases with activity; however, even this has variability. Intra-articular pain can be both anterior and posterior with a patient denoting a positive “C” sign in which the patient cups their hand around the greater trochanter in order to demonstrate a deep internal pain [30]. Referred pain to the knee may also be seen, particularly in younger patients. Occasionally the patient will describe a clicking or catching sensation, which may be associated with an intracapsular injury such as chondral delamination or labral tear.

Exam

Physical examination of young patients with hip pain requires a thorough and thoughtful clinical inspection. Clinical exam can identify up to 98 % of intra-articular hip pathology, although the exact etiology often requires additional studies [31]. Both extremities must be examined to assess the symptoms and function of the affected limb compared to the asymptomatic or less affected hip. Initial inspection of gait must be performed, as 85 % have been described to have a limp on the affected side while the patient walks a short distance [28]. Posturing or protective maneuvers to alleviate stress on the hip joint should be noted. A standing leg length examination should always be obtained along with spinal alignment to assist in determining the reason for pain or limp. A single leg stance may be useful, as 38 % of dysplasia patients have been reported to have a positive Trendelenburg sign. Palpation can rule out pathology such as trochanteric bursitis, snapping hip syndrome, or muscle strain/inflammation. Evaluation of muscle strength is often normal. Although resisted active range of motion may produce joint pain, this can often be distinguished from muscle strains with variances between active and passive range of motion. A full hip range of motion examination must be performed bilaterally, keeping in mind that motion is often preserved. Ninety-seven percent of patients endorse a positive impingement sign with flexion, adduction, and internal rotation testing (FADIR test), which often reproduces anterior groin symptoms [28]. Anterior instability, if present, can be reproduced with the anterior apprehension test, performed by the examiner extending, adducting, and externally rotating the hip which can reproduce clinical instability and acetabular rim overloading.

Further testing to rule out other etiology for hip pain must be performed prior to completion of the physical exam. Both internal and external snapping hip can cause patient symptoms and bring the patient into the clinic for evaluation. External snapping is secondary to the posterior portion of the tensor fascia lata or anterior border of the gluteus maximus “snapping” over the prominent greater trochanter. This can often be reproduced upon command in the clinic, and the examiner can feel or see the lateral snapping as the patient flexes and extends the adducted hip. Internal snapping due to the iliopsoas tendon moving across the anterior inferior iliac spine (AIIS), the lesser trochanter, or the iliopectineal ridge can be elicited by passive movement of the leg from an initial position of flexion and external rotation to a final position of extension and internal rotation. With overuse or repetitive snapping, the resultant friction may cause painful symptoms resulting from bursitis or muscle inflammation in the area.

Spine and sacroiliac (SI) joint pathology must also be evaluated during a thorough hip examination. Paraspinal musculature palpation, straight leg raise, scoliosis evaluation, and neurological examination should be included in the initial workup. Passive straight leg raise should be performed to rule out lumbar nerve root irritation or “sciatica.” Direct palpation over the SI joint or performing provocative maneuvers like Patrick’s test or FABER (Flexion, Abduction, and External Rotation) testing can aid the clinician in determining if SI joint pain is an underlying source of the pain.

Athletic pubalgia can often be difficult to distinguish from hip pain. Tenderness to palpation of the abdominal and adductor musculature and the absence of discomfort with passive range of motion are findings seen with this condition, while resisted sit-ups or hip adduction may precipitate the patient’s symptoms.

If the clinical diagnosis remains unclear at the end of a thorough examination, a diagnostic intra-articular injection can aid in the final identification of the patient’s symptomology, which proves to have 90 % accuracy of detecting intra-articular pathology [31].

Imaging

There have been significant advances in the understanding and diagnosis of the symptomatic hip in the young adult over the last two decades; however, the diagnosis is often elusive, especially in the setting of mild structural abnormalities. In order to effectively make a diagnosis of dysplasia in a timely fashion, physicians must have common and reliable radiographic images for accurate diagnosis and disease classification. Accepted, valid, and reproducible measurements of acetabular coverage in both coronal and sagittal planes are necessary to tailor the surgical approach for individual complex acetabular pathomorphologies [32]. Table 1 lists the classic radiographic measurements and accepted parameters obtained in the evaluation of a dysplastic hip.

The most commonly utilized radiographs for evaluation of the hip include an anteroposterior (AP) view of the pelvis [8, 33], a cross-table lateral view [34], a false-profile view [35], a frog-leg lateral view [36], and a 45° or 90° Dunn view [37]. Each view provides important information about the pathomorphology. They are highly technique dependent, and standardized views must be provided in order to improve diagnostic accuracy and disease classification.

The anteroposterior pelvic radiograph can provide significant information when obtained using these standardized techniques. The subject’s lower extremities should be oriented in 15° of internal rotation in order to maximize the femoral neck length. The image should be centered between the superior border of the pubic symphysis and a line drawn connecting the anterior superior iliac spines to visualize the entire bony pelvis

Table 1 Classic radiographic measurements seen in the dysplastic hip. The common measurements include lateral center-edge angle of Wiberg, anterior center-edge angle of Lequesne, acetabular index or acetabular angle of Sharp, and the acetabular roof angle of Tönnis

Radiographic measurement	Normal range	Dysplastic hip	Miscellaneous
Lateral center-edge angle	25–50°	<20°	AP radiograph
Anterior center-edge angle	24–45°	<20°	False profile
Acetabular angle	33–38°	>42°	AP radiograph
Tönnis angle	<10°	>10°	AP radiograph
% head coverage	80 %	<70 %	AP radiograph
Teardrop	Narrow teardrop	Widened "U" morphology	AP radiograph
Medial space	10–15 mm	>15 mm	AP radiograph

and to minimize scatter and parallax effect. A good AP pelvic radiograph should have neutral tilt, and the distance between the sacrococcygeal junction should be 2.5–4 cm above the superior end of the symphysis in males and between 4 and 5.5 cm in females [14]. Some authors prefer using the tip of the coccyx as opposed to the sacrococcygeal junction due to increased ease in identifying this anatomic landmark. In this scenario, the tip of the coccyx should be centered 1–3 cm above the pubic symphysis to ensure appropriate pelvic inclination [38]. This assures that pathology identified is true and not a rotational misinterpretation.

A dysplastic hip on AP radiograph can be represented by a shallow acetabulum with a widened teardrop and lateralized center of rotation, with anteversion or retroversion. The acetabulum is considered anteverted if there is no evidence of crossover sign. This is determined by the line of the anterior aspect of the rim *not* crossing the line of the posterior aspect of the rim before reaching the lateral aspect of the sourcil. If the acetabulum is retroverted, the line of the anterior aspect of the rim *does* cross the line of the posterior aspect of the rim before reaching the lateral edge of the sourcil (Fig. 1). Retroversion is also frequently associated with a prominent extension of the ischial spine into the pelvis [39]. There is often an upsloping sourcil with a dysplastic hip; the sourcil consists of the radiodense subchondral bone of the weight-bearing dome of the acetabulum. This finding can be quantified by the acetabular roof angle of Tönnis or the acetabular index of the weight-bearing zone; this is the angle

Fig. 1 Acetabular retroversion with a crossover and prominent ischial spines

formed between a horizontal line to the pelvis and a line extending from the medial to the lateral edge of the sourcil (Fig. 2). A Tönnis angle of 0–10° is considered normal, whereas an angle of >10° would be considered abnormally steep and consistent with dysplasia [40]. Similarly, the acetabular angle of Sharp, also referred to as the acetabular index or acetabular inclination (Fig. 3), identifies the inclination of the acetabulum by measuring the angle formed between a horizontal line spanning along the base of the two radiographic acetabular teardrops and a line from the ipsilateral inferior teardrop to the lateral sourcil. Dysplastic hips often have acetabular angles

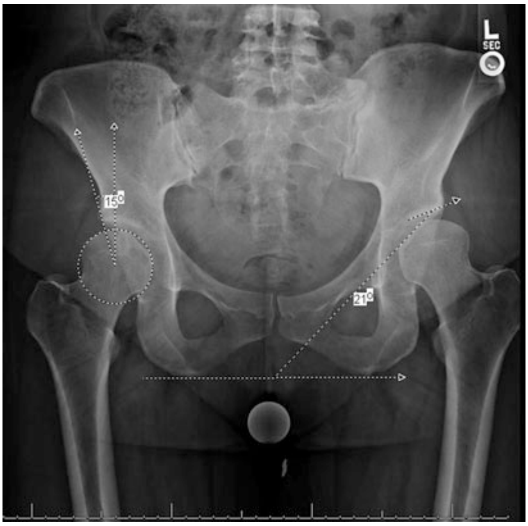


Fig. 2 Demonstrates a lateral center-edge angle of the right hip of 15°and acetabular roof angle or Tönnis angle of 21°. These measurements are both considered abnormal and consistent with acetabular dysplasia

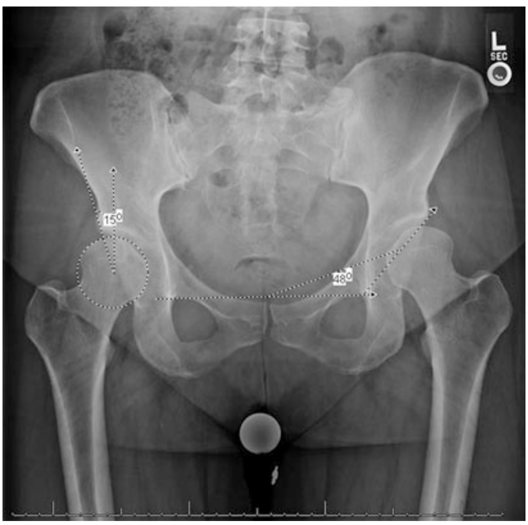


Fig. 3 Demonstrates a lateral center-edge angle of the right hip of 15° and Sharp's acetabular angle of the left hip of 48°. These measurements are both considered abnormal and consistent with acetabular dysplasia

greater than 42° [41]. The lateral center-edge angle of Wiberg on the AP radiograph assesses the acetabular superolateral coverage (Fig. 3). This is the angle formed by the intersection of a vertical line through the center of the femoral head and a line extending through the center of the

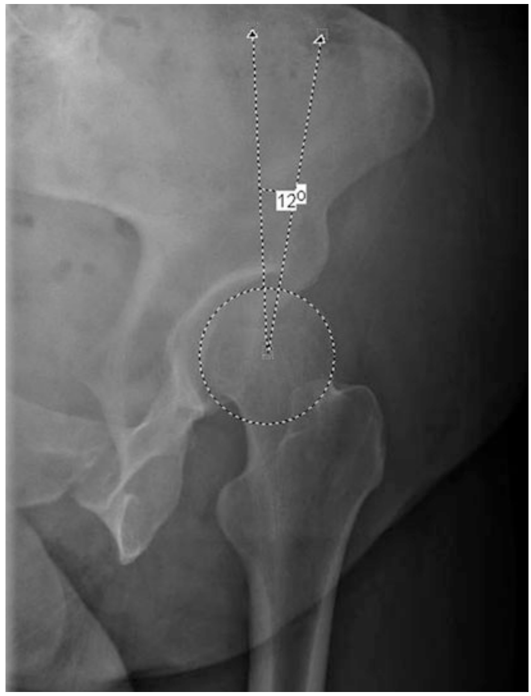


Fig. 4 Demonstrates an anterior center-edge angle of Lequesne on a false-profile radiograph

femoral head to the lateral sourcil. Hip dysplasia is defined as a lateral center-edge angle less than 20° [2].

A false-profile view [35] is obtained by positioning the patient standing with the symptomatic hip against the cassette with the foot parallel to the cassette. The pelvis is rotated 65°, so the radiograph is centered at the femoral head. This view shows anterior acetabular coverage of the femoral head and can show anterior subluxation during weight bearing in the dysplastic hip. The examiner can measure the anterior center-edge angle of Lequesne by identifying the angle formed by the intersection of a vertical line through the center of the femoral head to the anterior sourcil. Dysplastic hips often have measurements less than 20° [35] (Fig. 4).

A cross-table lateral radiograph [42] is obtained with the patient supine on the x-ray table. The contralateral hip and knee are flexed up out of the field of the hip being imaged. The imaged extremity is internally rotated 15° to expose the full anterolateral and posterolateral

Table 2 Hip dysplasia classification systems

	Femoral head subluxation	Proximal displacement
Crowe classification [43]		
Crowe I	<50 % subluxation	<10 %
Crowe II	50–75 % subluxation	10–15 %
Crowe III	75–100 % subluxation	15–20 %
Crowe IV	>100 % subluxation	>20 %
Hartofilakidis classification [44]		
Type A (dysplasia)	Femoral head within acetabulum despite some subluxation. Segmental deficiency of the superior wall. Inadequate true acetabulum depth	
Type B (low dislocation)	Femoral head creates a false acetabulum superior to the true acetabulum. There is complete absence of the superior wall. Inadequate depth of the true acetabulum	
Type C (high dislocation)	Femoral head is completely uncovered by the true acetabulum and has migrated superiorly and posteriorly. There is a complete deficiency of the acetabulum and excessive anteversion of the true acetabulum	
Tönnis classification of osteoarthritis by radiographic changes [40]		
Grade 0	No signs of OA	
Grade 1	Increased sclerosis, slight joint space narrowing, no or mild loss of head sphericity	
Grade 2	Small cysts, moderate joint space narrowing, moderate loss of head sphericity	
Grade 3	Large cysts, severe joint space narrowing, severe head deformity	

femoral head-neck junction. The x-ray tube is orientated parallel to the table at a 45° angle to the imaged limb and centered on the femoral head.

The frog-leg lateral view [36] has the patient positioned supine on the x-ray table with the symptomatic hip abducted to 45° with the knee bent. The radiograph is centered midway between the pubic symphysis and the anterior superior iliac spine.

Finally, the Dunn lateral view [37] has been described with the radiograph taken either with the hip flexed to 45° or 90° and is referred to as the 45° Dunn lateral or the 90° Dunn lateral, respectively. The radiograph is obtained the same way despite the degree of hip flexion. The symptomatic hip is flexed to either 45° or 90° with 20° of abduction. The limb is placed in a neutral rotation. The beam is centered at a point midway between the pubic symphysis and the anterior superior iliac spine.

The cross-table lateral, the frog lateral, and the Dunn lateral views have slight projectional variations and highlight certain pathology on the proximal femur such as head sphericity, anterior and posterior outlines of the femoral neck, alpha angle, and head-neck offset. The radiograph of choice is often physician specific, however should be kept constant throughout the practice.

There are several described radiographic classification systems associated with acetabular dysplasia; Table 2 outlines the three more commonly used classifications. The first is the Crowe classification [43] in which Crow I describes minimal to no abnormal development, with <50 % subluxation of the hip joint and <10 % proximal femoral displacement. Crowe II describes dysplastic morphology with 50–75 % subluxation and 10–15 % proximal femoral displacement. Crowe III shows severe acetabular dysplasia often with the femur dislocated or near dislocated with 15–20 % proximal femoral displacement. Finally Crowe IV is considered a high dislocation with >20 % proximal migration. The second classification system noted in Table 2 is the Hartofilakidis classification, which was described to predict the bony deficiencies encountered during the operation from preoperative pelvis radiographs. Hartofilakidis type A is described as a dysplastic acetabulum, type B is a low lying dislocation, and type C describes a high riding dislocation.

The degree of hip osteoarthritis must also be evaluated and graded based on the radiographic series that will dictate the potential treatment options. The Tönnis classification (Table 2) of osteoarthritis ranges from 0 to 3, with Grade 0 indicating no signs of osteoarthritis. Grade 1 shows increased sclerosis of the acetabulum, slight joint space narrowing, and slight lipping. Grade 2 shows small cysts in the acetabulum, moderate joint space narrowing, and moderate loss of sphericity of the head. Grade 3 is end-stage arthrosis with large cysts in the acetabulum, joint space obliteration or severe joint space narrowing, severe deformity of the femoral head, or evidence of necrosis [40]. Improved outcomes have been shown with hip-preserving procedure performed for Tönnis 1 or less [45].

Computed tomography (CT) scans are often not necessary in the diagnosis and treatment of acetabular dysplasia however do provide excellent three-dimensional osseous anatomy to thoroughly understand the underlying pathology. CT scan with femoral subtraction 3D reconstruction provides an unobstructed evaluation of acetabular morphology, the ability to quantify and determine the type or location of acetabular retroversion, and may aid in clinician understanding during preoperative planning. CT arthrogram also provides additional clinical information about labral injuries where magnetic resonance imaging is either not readily available or there are patient contraindications.

Magnetic resonance imaging (MRI) and magnetic resonance arthrogram (MRA) are commonly the most effective nonoperative ways of identifying suspected intra-articular pathology such as labral tears, chondral defects, synovial disease, muscle or capsular irritation, and inflammation and loose bodies. However, even with this advanced imaging, there are limitations. MRI has shown a 42 % false-negative rate and a 10 % false-positive rate while MRA increases the sensitivity with an 8 % false-negative interpretation but a 20 % false-positive rate [31]. The indications for obtaining MRI or MRA vary among clinicians, and there are currently no widely accepted algorithms.

Summary

Acetabular dysplasia in the skeletally mature patient remains an evolving discipline. Given its prevalence, morbidity if left untreated, and potential for intervention, there remains a need for improved awareness by clinicians, especially in the setting of radiographically mild disease. Subtle and insidious clinical symptoms often lead the patient to see multiple physicians, obtain multiple diagnoses, and undergo multiple treatments prior to final diagnosis of skeletally mature acetabular dysplasia. There is often greater than a 5-year delay from symptom onset to final diagnosis and possible intervention. Young patients with complaints of insidious-onset, activity-related groin and hip pain should be carefully investigated so that a focused evaluation and diagnosis are obtained. After an accurate diagnosis of acetabular dysplasia, the patients should be counseled regarding disease prognosis and treatment options.

The treatment of acetabular dysplasia ranges from conservative management, to osteotomies, to hip replacement depending upon patient specific variables. Asymptomatic patients with borderline radiographs picked up incidentally may be treated conservatively with close clinical and radiographic follow-up. Nonsteroidal anti-inflammatory agents can be used, and high-impact activities should be avoided with radiographs every 1–2 years. Symptomatic patients without evidence of osteoarthritis may be treated with a variety of treatments ranging from arthroscopy to remove loose bodies or labral repair, to pelvic osteotomies, to historically arthrodesis in the young dysplastic hip. The main goal for these procedures is to reduce pain and symptoms. Some osteotomies aim to decrease the progression of degenerative changes by increasing hip congruency, decreasing the acetabular edge loading, and better distributing the forces applied through the symptomatic hip. The various osteotomies will be described in a later chapter. Patients with late radiographic findings and severe osteoarthritis are best treated with total hip arthroplasty. There remains a vast area of clinically borderline symptomatic patients with young age and early osteoarthritis that still remains a topic of controversy.

References

1. Gosvig KK, Jacobsen S, Sonne-Holm S, Palm H. Prevalence of malformations of the hip joint and their relationship to sex, groin pain, and risk of

osteoarthritis: a population-based survey. J Bone Joint Surg Am. 2010;92(5):1162–9.
2. Wiberg GGF, Helen D. Studies on dysplastic acetabula and congenital subluxation of the Hip joint, with special reference to the complication of osteo-arthritis. (translated from the Swedish by Helen Frey). Stockholm: P. A. Norstedt & Sönep; 1939. p. 135.
3. Gade HG. A contribution to the surgical treatment of osteoarthritis of the Hip-joint: a clinical study. Oslo: Grøndahl & søns boktr; 1947.
4. Le Coeur P. Correction des défauts d'orientation de l'articulation coxofémorale par ostéotomie de l'isthme iliaque. Rev Chir Orthop. 1965;51:211–2.
5. Steel HH. Triple osteotomy of the innominate bone. J Bone Joint Surg Am. 1973;55(2):343–50.
6. Tönnis D, Behrens K, Tscharani F. A modified technique of the triple pelvic osteotomy: early results. J Pediatr Orthop. 1981;1(3):241–9.
7. Carlioz H, Khouri N, Hulin P. Triple juxtacotyloid osteotomy. Rev Chir Orthop Reparatrice Appar Mot. 1982;68(7):497–501 (PubMed – NCBI. … et réparatrice de l'appareil moteur. 1982.).
8. Ganz R, Klaue K, Vinh TS, Mast JW. A new periacetabular osteotomy for the treatment of hip dysplasias technique and preliminary results. Clin Orthop Relat Res. 1988;232:26–36.
9. Aminian A, Mahar A, Yassir W, Newton P, Wenger D. Freedom of acetabular fragment rotation following three surgical techniques for correction of congenital deformities of the Hip. J Pediatr Orthop. 2005;25(1):10.
10. Matheney T, Kim Y-J, Zurakowski D, Matero C, Millis M. Intermediate to long-term results following the Bernese periacetabular osteotomy and predictors of clinical outcome. J Bone Joint Surg Am. 2009;91 (9):2113–23.
11. Steppacher SD, Tannast M, Ganz R, Siebenrock KA. Mean 20-year followup of Bernese periacetabular osteotomy. Clin Orthop Relat Res. 2008;466(7): 1633–44.
12. Mast JW, Brunner RL, Zebrack J. Recognizing acetabular version in the radiographic presentation of hip dysplasia : section I symposium. Clin Orthop Relat Res. 2004;418:48–53.
13. Nehme A, Trousdale R, Tannous Z, Maalouf G, Puget J, Telmont N. Developmental dysplasia of the hip: is acetabular retroversion a crucial factor? Orthop Traumatol Surg Res. 2009;95(7):511–9.
14. Fujii M, Nakashima Y, Yamamoto T, Mawatari T, Motomura G, Matsushita A, et al. Acetabular retroversion in developmental dysplasia of the hip. J Bone Joint Surg Am. 2010;92(4):895–903.
15. Myers SR, Eijer H, Ganz R. Anterior femoroacetabular impingement after periacetabular osteotomy. Clin Orthop Relat Res. 1999;363:93.
16. Ginnetti JG, Pelt CE, Erickson JA, Van Dine C, Peters CL. Prevalence and treatment of intraarticular pathology recognized at the time of periacetabular osteotomy for the dysplastic hip. Clin Orthop Relat Res. 2013;471(2):498–503.
17. Henak CR, Ellis BJ, Harris MD, Anderson AE, Peters CL, Weiss JA. Role of the acetabular labrum in load support across the hip joint. J Biomech. 2011;44 (12):2201–6.
18. Dorrell JH, Catterall A. The torn acetabular labrum. J Bone Joint Surg. 1986;68(3):400–3.
19. Klaue K, Durnin CW, Ganz R. The acetabular rim syndrome. A clinical presentation of dysplasia of the hip. J Bone Joint Surg Br. 1991;73(3):423–9.
20. Guevara CJ, Pietrobon R, Carothers JT. Comprehensive morphologic evaluation of the hip in patients with symptomatic labral tear. Clin Orthop Relat Res. 2006;453:277–85.
21. Wu X, Li S-H, Lou L-M, Cai Z-D. The techniques of soft tissue release and true socket reconstruction in total hip arthroplasty for patients with severe developmental dysplasia of the hip. Int Orthop. 2012;36(9):1795–801.
22. Paavilainen T, Hoikka V, Solonen KA. Cementless total replacement for severely dysplastic or dislocated hips. J Bone Joint Surg Br. 1990;72(2):205–11.
23. Clohisy JC, Barrett SE, Gordon JE, Delgado ED, Schoenecker PL. Periacetabular osteotomy for the treatment of severe acetabular dysplasia. J Bone Joint Surg Am. 2005;87(2):254–9.
24. Harris MD, Anderson AE, Henak CR, Ellis BJ, Peters CL, Weiss JA. Finite element prediction of cartilage contact stresses in normal human hips. J Orthop Res. 2012;30(7):1133–9.
25. Peters CL, Erickson J. The etiology and treatment of Hip pain in the young adult. J Bone Joint Surg Am. 2006;88(suppl_4):20–6.
26. Parvizi J, Bican O, Bender B, Mortazavi SMJ, Purtill JJ, Erickson J, et al. Arthroscopy for labral tears in patients with developmental dysplasia of the hip: a cautionary note. J Arthroplasty. 2009;24 (6 Suppl):110–3.
27. Feldman GJ, Peters CL, Erickson JA, Hozack BA, Jaraha R, Parvizi J. Variable expression and incomplete penetrance of developmental dysplasia of the hip: clinical challenge in a 71-member multigeneration family. J Arthroplast. 2012;27(4):527–32.
28. Nunley RM. Clinical presentation of symptomatic acetabular dysplasia in skeletally mature patients. J Bone Joint Surg Am. 2011;93 Suppl 2:17.
29. Peters CL. Hip-preserving surgery: understanding complex pathomorphology. J Bone Joint Surg Am. 2009;91 Suppl 6:42.
30. Byrd JT. Physical examination. In: Byrd JWT, editor. Operative hip arthroscopy. 2nd ed. New York: Springer; 2005. p. 36–50.
31. Byrd JWT, Jones KS. Diagnostic accuracy of clinical assessment, magnetic resonance imaging, magnetic resonance arthrography, and intra-articular injection in hip arthroscopy patients. Am J Sports Med. 2004;32(7):1668–74.
32. Anderson LA, Gililland J, Pelt C, Linford S, Stoddard GJ, Peters CL. Center edge angle measurement for hip preservation surgery: technique and caveats. Orthopedics. 2011;34(2):86.

33. Jamali AA, Mladenov K, Meyer DC, Martinez A, Beck M, Ganz R, et al. Anteroposterior pelvic radiographs to assess acetabular retroversion: high validity of the "cross-over-sign". J Orthop Res. 2007;25(6):758–65.
34. Eijer H, Leunig M, Mahomed MN, Ganz R. Cross-table lateral radiographs for screening of anterior femoral head-neck offset in patients with femoro-acetabular impingement. Hip Int. 2001;11(1):37–41.
35. Lequesne M, Djian A. New radiographic angles for the study of the hip. The"false profile" of the pelvis. Vie médicale (Paris, France: 1920). 1961;42:1629.
36. Clohisy JC, Nunley RM, Otto RJ, Schoenecker PL. The frog-leg lateral radiograph accurately visualized hip cam impingement abnormalities. Clin Orthop Relat Res. 2007;462:115–21.
37. Dunn DM. Anteversion of the neck of the femur; a method of measurement. J Bone Joint Surg Br. 1952;34-B(2):181–6.
38. Clohisy JC. A systematic approach to the plain radiographic evaluation of the young adult hip. J Bone Joint Surg Am. 2008;90 Suppl 4:47.
39. Kalberer F, Sierra RJ, Madan SS, Ganz R, Leunig M. Ischial spine projection into the pelvis. Clin Orthop Relat Res. 2008;466(3):677–83.
40. Tönnis D. Clinical and radiographic schemes for evaluating therapeutic results. In: Congenital dysplasia and dislocation of the hip in children and adults. New York: Springer; 1987. p. 165–71.
41. Sharp IK. Acetabular dysplasia the acetabular angle. J Bone Joint Surg Br. 1961;43(2):268–72.
42. Laage H, Barnett JC, Brady JM, Dulligan PJ, Fett HC, Gallagher TF, et al. Horizontal lateral roentgenography of the hip in children; a preliminary report. J Bone Joint Surg Am. 1953;35-A(2): 387–98.
43. Crowe JF, Mani VJ, Ranawat CS. Total hip replacement in congenital dislocation and dysplasia of the hip. J Bone Joint Surg Am. 1979;61(1):15–23.
44. Hartofilakidis G, Stamos K, Karachalios T, Ioannidis TT, Zacharakis N. Congenital hip disease in adults. Classification of acetabular deficiencies and operative treatment with acetabuloplasty combined with total hip arthroplasty. J Bone Joint Surg Am. 1996;78 (5):683–92.
45. Ganz R, Horowitz K, Leunig M. Algorithm for femoral and periacetabular osteotomies in complex Hip deformities. Clin Orthop Relat Res. 2010;468(12): 3168–80.

Skeletally Mature Acetabular Dysplasia: Epidemiology, Natural History, Clinical Presentation, Imaging Studies, Non-Operative Treatment, Operative Treatment

44

Anay Patel, John P. Manalo, and Michael D. Stover

Contents

Abstract

Skeletally mature acetabular dysplasia (SMAD) is a recognized cause of hip pain in active young adults. The majority of hip dysplasia is treated in childhood by improving the physiologic and anatomic environment of the hip allowing it to develop into a morphologically sound structure. The abnormal morphology of the mature dysplastic hip can be subtle both clinically and radiographically which can often lead to a delay in diagnosis.

The goal of treatment with SMAD is first and foremost to recognize the specific pathology causing the common complaints of hip pain. Hip pain, limp, and mechanical symptoms that can present with SMAD may be the same presenting complaints in patients with either femoroacetabular impingement (FAI) or mild osteoarthritis (OA). Plain radiographs and MRI provide the diagnosis, which in turn allows the treating physician to choose the appropriate treatment course.

Treatment of SMAD generally falls into one of two categories. For patients who present without radiographic signs of OA, the goal is to preserve the native cartilage of the hip by creating a more mechanically sound hip. The periacetabular osteotomy (PAO) reorients the acetabulum, providing stability to the hip and decreasing cartilage stress. Patients who present with moderate to severe OA generally do not obtain good results with hip preservation procedures and, therefore, may require total

A. Patel • J.P. Manalo • M.D. Stover (✉)
Department of Orthopaedic Surgery, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA
e-mail: mstover@nmff.org

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_47

hip arthroplasty to address their hip pain. Although results are excellent with total joint replacement, the mainstay of treatment for SMAD should be recognition via clinical and radiographic examination followed by prompt treatment, if indicated, to help preserve the native hip joint.

Introduction

The term acetabular dysplasia is often used to describe a variety of abnormal acetabular morphologies leading to pathomechanical injury to the cartilage and labrum and, ultimately, osteoarthritis (OA) of the hip. The spectrum of pathologic morphologies is diverse, with variable areas of acetabular deficiency and version abnormalities, which may result in subluxation of the hip. There can be associated pathomorphologies of the femur. Together they result in abnormal force transmission at the acetabular rim, resulting in labral and cartilage damage, and eventual end stage OA of the hip (Figs. 1 and 2).

Fig. 1 This image shows a morphologically normal hip with adequate coverage of the femoral head

Pathoanatomy of the Dysplastic Hip

Normal development of the hip joint is determined by numerous factors that are present during both the gestation and postpartum periods of a child's life. The major driving force for proper morphologic development is concentric reduction of a spherical femoral head into the concave acetabulum. The femoral head provides a foci around which the acetabulum is shaped while the properly contained femoral head is in turn molded by the native acetabulum. Failure to achieve this interdependent concentric reduction will influence the ability of the native hip to develop appropriately.

Surrounding the edge of the femoral-acetabular junction is the labrum of the hip. This cartilaginous structure serves multiple functions in the healthy hip. It effectively increases the contact area between the acetabulum and the femoral head providing a greater surface area for force distribution. The labrum also creates a tight seal for the hip joint, which helps maintain a stable hip reduction, allows for smooth motion of the hip, and optimizes the fluid-film mechanics [1–3].

Skeletally mature acetabular dysplasia can prevent proper physiologic function due to the morphologic abnormalities affecting hip joint mechanics. The most common morphologic abnormality in SMAD is the presence of a shallow acetabulum with a lack of both anterior and lateral femoral head coverage; however, different morphologies also exist (Table 1) [4]. Approximately 40 % of patients will present with some degree of retroversion [5]. Dysplastic hips may also be associated with increased femoral anteversion and femoral neck valgus [6].

The ramifications of these morphologic abnormalities on the soft tissues of the hip are what cause the symptomatic nature of the condition. The deficient coverage leads to decreased surface area for force transmission and increased stress

Fig. 2 This image shows the shallow acetabulum and decreased coverage consistent with skeletally mature acetabular dysplasia

Table 1 Distribution of acetabular deficiency

Location of deficiency	Percentage (%)
Lateral	7
Anterior	8
Posterolateral	18
Anterolateral	72

across a focused area of the joint cartilage and labrum. Increased loads at these focal areas lead to both physical microtrauma and physiologic changes within the soft tissues that lead to their degeneration [7, 8]. Decreased coverage and increased inclination of the articular surface along with anteversion and valgus of the femur can result in variable degrees of joint instability. Normal repetitive activities such as walking, running, and climbing stairs lead to increased shear forces on the cartilage and labrum in the dysplastic hip. Eventually these repetitive shear forces cause tearing within the labrum itself [9]. The regions of acetabular bone undercoverage correlate anatomically with the location of the labral tear. This repetitive shearing and physical overload may also explain the presence of a hypertrophied labrum in dysplastic hips [10].

As the labrum degenerates, pathologic changes can also be seen in the articular cartilage of the hip joint. Dysplastic hips may already be at a disadvantage as the quality of the cartilage differs from that of a normal hip [11]. The increased loads at the cartilage of the chondrolabral junction in conjunction with a compromised labrum will exacerbate the pathologic process. Early alterations occur at the physiologic level, with cartilage specimens showing increased content of inflammatory cytokines and catabolic enzymes. These alterations lead to cleavage of proteoglycans, decreased water content, and further loss of the mechanical integrity [11]. While abnormal morphology is the root of this physical and physiologic degeneration, it is the injury to the soft tissues that is thought to be the genesis of symptoms in many patients [12].

The process of chondrolabral degeneration is not unique to SMAD. The treating surgeon should be mindful that FAI encompasses a number of femoral and acetabular morphologies. The specific morphology of the pathologic hip will not only affect the location and pattern of chondrolabral damage but will influence the presentation of the patient and will significantly impact the treatment options available. Hip dysplasia represents a focal process generally due to undercoverage in the anterolateral acetabulum; however, unique populations such as cerebral palsy patients will present with focal posterolateral deficiencies. An aspherical femoral head, diminished femoral torsion, or acetabular retroversion may potentiate motion-induced impingement even in the setting of focal insufficient coverage and can result in motion-induced instability. While all of these pathologies lead to differing abnormal forces at the hip joint, the specific morphologic pattern determines presentation and, more importantly, their treatment. Overcorrection of dysplasia may exacerbate an unrecognized or potential impingement.

Epidemiology

The exact prevalence of SMAD is difficult to determine due to the variable nature of symptomatology, access to healthcare, and failure to recognize the problem. Returning to the root of the problem, developmental dysplasia of the hip is estimated to have an incidence of 5.5 % in neonates based on studies performed in the United States (US) [13]. Currently, physical examination screening is performed on the majority of children in the US and this allows for both prompt identification of the problem and early treatment if necessary. This broad-based screening may have decreased the prevalence of SMAD by identifying the problem during infancy. While 80 % of neonatal hip dysplasia will spontaneously resolve, those who require treatment can be confident excellent results can be achieved with the Pavlik Harness [14].

What is difficult to discern is whether a direct correlation or even progression between DDH and SMAD exists. Developmental dysplasia of the hip has been shown to be associated with predominantly female sex, breech presentation, the left hip, and a family history of DDH. Patients with SMAD have a higher proportion of males, bilateral involvement, and a family member who underwent THA before age 65 [15] (Table 2). Larger population-based studies done for FAI are now shedding more light into the prevalence of SMAD and the prevalence of both asymptomatic and symptomatic patients. The rate of SMAD based on lateral center-edge angle (LCEA) places the prevalence at somewhere between 5 % and 20 % of the population having an LCEA $<25°$ and approximately 3–6 % having an LCEA $< 20°$ [16].

These studies have also shown that only a small minority of those patients with radiographic abnormalities will go on to develop symptoms. The connection of SMAD to OA is based on studies of small populations or selective cohorts. While decreased LCEA is associated with an increased risk for OA, the cutoff values used to define pathologic LCEA varies from study to study, and therefore, broad conclusions cannot be drawn. Females are thought to be at higher risk for OA than males, but this is based on limited study information [17]. Further complicating the issue is that SMAD represents only a small portion of the large variety of morphologic acetabular anomalies that have been described.

Table 2 Comparison of developmental dysplasia and skeletally mature acetabular dysplasia

Developmental dysplasia of the hip	Skeletally mature acetabular dysplasia
Female sex (98 %)	Female sex (88 %)
Breech presentation	
Left hip	Bilateral involvement
Family history of DDH	Family member with THA before age 65
Oligohydramnios	

Clinical Evaluation

Clinical diagnosis of the young patient with SMAD can be a difficult task. Most patients will present with the ambiguous complaint of hip pain. “Hip pain” is a vague term that can encompass many pathologies that may or may not be associated with the hip itself. Many patients who present with hip dysplasia have often had a long-standing history of hip pain with one study showing on average 5 years from onset of pain to the time of diagnosis [18]. Most patients had seen a primary care or sports medicine physician and been diagnosed with a variety of pathologic processes. Some will also have tried physical therapy with no improvement or possibly exacerbation of their symptoms with stretching programs [19]. Thus, the surgeon who chooses to evaluate hip pain in the young patient should be fully aware of the range of clinical presentations and how they relate to both intra-articular and extra-articular pathologies.

Evaluation should begin with a thorough history of the presenting complaint. Specific details should be determined including duration of the symptoms and the history of the symptoms. Most patients will present with an insidious onset; however, some will relate it to a traumatic cause [18]. Exacerbating factors should be determined as many patients will complain of pain with activities such as running or sports, but some may notice it with activities of daily living such as getting in and out of a car, prolonged sitting, or rising from a seated position. Pain with prolonged sitting is more often seen with FAI, whereas patients with SMAD may complain of pain with prolonged standing due to chronic abductor weakness [20]. The location of the pain is also variable, but will predictably be present in the anterior groin, the lateral side of the hip, or both. Laterally based pain can be associated with abductor weakness, while groin pain in SMAD may be due to a labral tear or instability. About 75 % of patients will find their pain to be either moderate or severe, and a similar percentage will either report or present with limping when walking even short distances [18, 20]. Nunley and colleagues reported an inability to correlate specific pathology with complaints. The constellation of symptoms seen in SMAD is also seen in FAI and mild osteoarthritis, which may lead to a delay in diagnosis if a thorough evaluation is not performed [18].

The majority of patients with either FAI or SMAD will present with mechanical symptoms during activities specifically those with labral tears [18, 20]. These mechanical symptoms may be described as snapping or popping and are generally sensations felt in the groin, not in the lateral thigh as is seen in iliotibial band (ITB) syndrome. Most will not be using walking aids. Rest is generally their most reliable method of symptom relief, and many will report having decreased or modified their activities to prevent pain [18, 20].

Thorough investigations should be performed into the patient's medical history. Specifically, their birth history is important to identify risk factors for DDH such as breech presentation, first born, or pregnancy-related issues such as oligohydramnios. Mode of delivery can also be useful as a patient may have been born via Cesarean section but not know the inciting reason for the non-vaginal delivery. Childhood history should also be evaluated to determine if any interventions were performed such as double diapers, Pavlik harness, brace wear, or any imaging that may have been done. Finally, family history should be explored to determine if there is any history of hip problems in the family. Physicians should specifically ask about family members who may have undergone THA and the ages at which those procedures were performed. Recent evidence shows patients with SMAD are more likely to have a family member who underwent total hip arthroplasty (THA) at an age less than 65 [15].

Physical exam should begin by evaluating the patient's gait. The presence of a Trendelenburg gait suggests abductor weakness, internal foot-progression angle may indicate increased femoral anteversion, and a limp may be due to pain or limb length equality. Limb lengths should be measured as they may indicate a possible subluxed or dislocated hip. Range of motion, typically evaluated in the supine position, should be documented and assessed for any limitation of abduction, flexion, or internal rotation. Patients with SMAD will generally have a slightly increased range of motion which may help distinguish it from FAI. Pain or clicking at the extremes of motion may be indicative of a labral tear. Patients should be evaluated for ITB tightness (Ober test) or abductor tightness which can occur in the setting of a patient with instability associated with dysplasia [21]. The patient should also be examined for a generalized ligamentous laxity.

A number of provocative maneuvers have been described to evaluate hip pain in the young patient. All patients should be palpated and asked about abdominal or inguinal tenderness to rule out a possible hernia. The anterior apprehension test is performed by having the patient extend their hip while the examiner adducts and external rotates the hip. Recreation of groin pain or guarding can be a sign of instability [21]. The anterior impingement test with flexion, adduction, and internal rotation of the hip has good positive predictive value but low sensitivity for labral tears [22]. The FABER test, with flexion, abduction, and external rotation, was initially described for the assessment of sacroiliitis; however, reproduction of groin pain has high positive predictive value for intra-articular hip pathology.

Finally, the examiner should attempt to reproduce any snapping sensation in the hip or the patient should be asked to reproduce them. Lateral snapping can be due to movement of the anterior

Fig. 3 Proper AP pelvis radiographs should have the tip of the coccyx centered of the pubic symphysis and within 2 cm. Additionally, the obturator foramen should appear symmetric

border of the gluteus maximus or the posterior border of the tensor fascia lata (TFL) over the greater trochanter. The psoas test, by which one will flex the hip, external rotate, abduct, and then slowly extend the leg with internal rotation, may reproduce snapping associated with the psoas tendon and the iliopectineal eminence.

Radiographic Evaluation

The cornerstone of treatment of SMAD is not only the clinical diagnosis but identification of specific anatomic abnormalities. Perhaps no portion of the workup plays a greater role than imaging. Although the clinical exam can be obvious at times, accurate interpretation of proper radiographs is crucial to determine the specific morphologic abnormalities that may be present and, subsequently, the treatment options that may or may not benefit the patient. Although a patient may present with an exam and history strongly suggestive of a labral detachment, plain films should always be closely scrutinized for morphologic abnormalities, as labral tears rarely in their absence.

All imaging begins with a proper AP pelvis plain radiograph. Patient positioning and beam placement is essential to obtaining an image that has accurate rotation and tilt. Films can be taken in the supine position or the standing position with approximately 120 cm between the tube and the film distance [23]. Standing films may lead to slight extension of the hip that can effect pelvic tilt and diminish the acetabular retroversion appreciated on supine films [24–26]. Both legs should be internally rotated approximately 15° to take into consideration femoral anteversion. In males, the proper tilt is obtained when the tip of coccyx is approximately 1–3 cm above the top of the pubic symphysis. In females, up to 4 cm of space may be seen in a normal AP pelvis radiograph [23]. The film should then be inspected for proper rotation. Rotation can be assessed by the symmetry of the obturator foramen, the teardrop, and the iliac wings [27]. The tip of the coccyx should also be centered over the pubic symphysis (Fig. 3).

After confirming that an appropriate film has been obtained, the physician should look for obvious pathology that could result in pain such as frank osteoarthritis, fracture, or any sclerosis or bone reaction in the region of the femoral head or neck that may signal a stress fracture or other pathologic process. The femoral head should be evaluated for possible subchondral radiolucency or collapse as seen in avascular necrosis. Subluxation can be assessed using Shenton's line. Joint space narrowing, osteophyte formation, subchondral cysts, and subchondral sclerosis should all be evaluated with respect to the Tonnis grading system for osteoarthritis of the hip.

Concurrent use of the joint space width (JSW) of ≥ 2 mm or <2 mm has been used to help

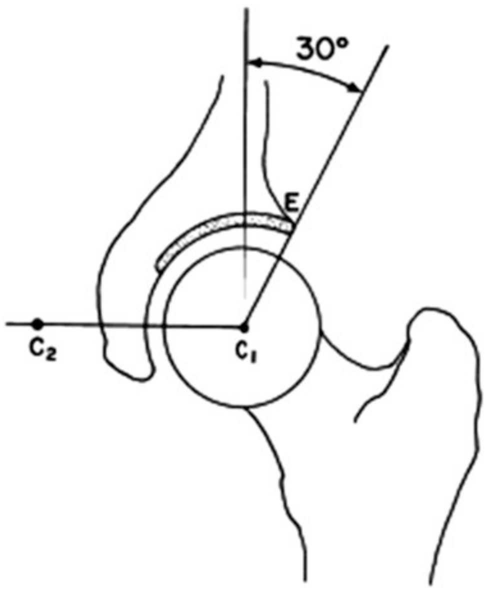


Fig. 4 This illustration shows the lateral center-edge angle of Wiberg (Reprinted with permission from Delaunay, S., RG Dussault, PA Kaplan, and BA Alford. "Radiographic Measurements of Dysplastic Adult Hips." *Skeletal Radiology* 26.2 (1997): 75–81.)

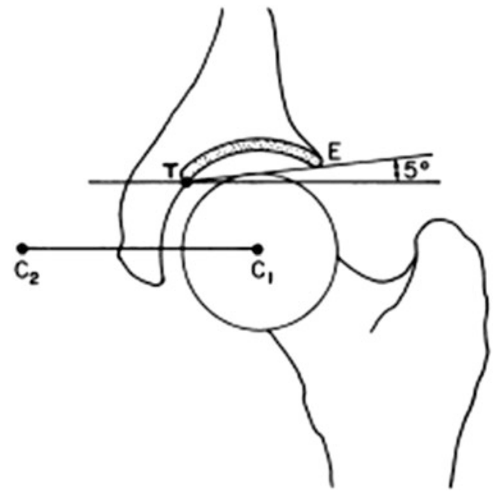


Fig. 5 This illustration shows the acetabular index or tonnis angle (Reprinted with permission from Delaunay, S., RG Dussault, PA Kaplan, and BA Alford. "Radiographic Measurements of Dysplastic Adult Hips." *Skeletal Radiology* 26.2 (1997): 75–81.)

dichotomize patients into those with Grade 0 or 1 changes and JSW >2 mm compared to a second group of patients with Grade 2 or 3 changes and JSW <2 mm [28–30]. Radiographic grading is important as the presence of Grade 2 or 3 changes have been associated with inferior outcome, following joint preservation procedures.

Next, parameters of the hip are determined to define any pathomorphologies. The lateral center-edge angle (LCEA) of Wiberg evaluates the lateral coverage of the hip. A line, which is perpendicular to the inter-teardrop line, is drawn through the center of the femoral head to the acetabulum, and a second line is drawn from the center of the femoral head to the lateral margin of the acetabulum. The angle created by this arc is the LCEA, and normal values range from 25° to 40°. Less than 20° is considered acetabular undercoverage, while greater than 40° indicates overcoverage [16] (Figs. 4 and 6).

The acetabular index of Tonnis assesses the slope of the weight-bearing surface. A line that is parallel to the inter-teardrop line is drawn across the top of the femoral head. A second line is then drawn from the medial aspect of the sourcil to the lateral aspect of the sourcil. An angle greater than 10° is commonly associated with dysplasia (Figs. 5 and 6).

A common finding in SMAD is lateralization of the hip center due to a shallow acetabulum. The amount of hip joint lateralization should be quantified to not only compare to the contralateral hip but also as a marker for how much medialization is needed at the time of surgery. Lateralization is commonly associated with subluxation, defined by a break in Shenton's line. The distance between the medial aspect of the femoral head and the ilioischial line is measured. A normal value is generally less than 10 mm [31].

Careful evaluation of acetabular version is important as this may indicate an isolated pathology and may be associated with posterior superior coverage deficiencies or a more classic lateral and anterior deficiency with concomitant retroversion. First the anterior acetabular rim should be evaluated in its relationship to the posterior acetabular rim. In a normal hip, the anterior rim is at all times medial to the posterior rim with both originating at the superior aspect of the acetabulum. If the anterior rim is seen to cross over the posterior rim, this is suggestive of acetabular retroversion (Fig. 7). In SMAD, the crossover is generally seen in the

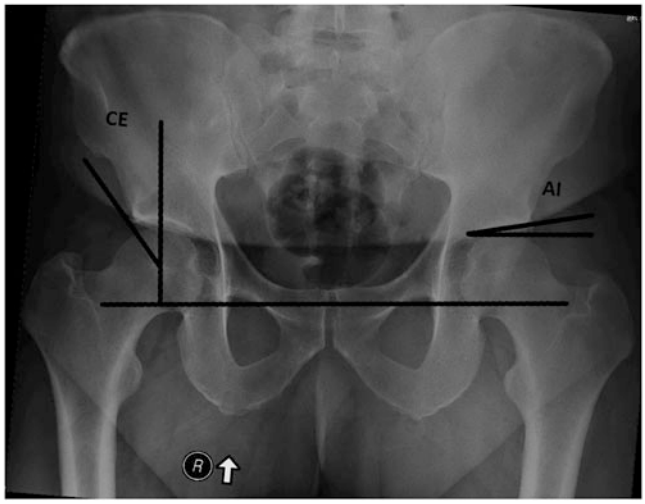


Fig. 6 A radiograph outlined with the radiographic measurements of the lateral center-edge angle and acetabular index

Fig. 7 This radiograph shows a prominent right ischial spine, crossover sign, and a large iliac wing consistent with focal acetabular retroversion

cranial third of the acetabulum [32]. Concurrently, the posterior wall sign must be evaluated as the normal posterior wall will intersect or be lateral to the center of the femoral head. Deficient posterior coverage is defined as the rim being medial to the center of the femoral head [33]. Another indicator of acetabular retroversion is the ischial spine sign which is demonstrated as any portion of the ischial spine projecting into the pelvic on an AP pelvis image [34]. Retroversion must be evaluated prior to undertaking surgery as failure to recognize and address its presence can lead to acetabular malposition and impingement symptoms even after acetabular redirecting surgery [35].

Lateral views of the hip will provide additional information on associated femoral morphologies. The 45° Dunn lateral has been shown to be the most sensitive for evaluating femoral CAM deformity [36]. The image is obtained with the patient supine, the hip flexed to 45° and abducted 20° with neutral rotation. The beam should be aimed at a point between the anterior superior iliac spine (ASIS) and the pubic symphysis [23]. Cross-table lateral images may help determine femoral torsion

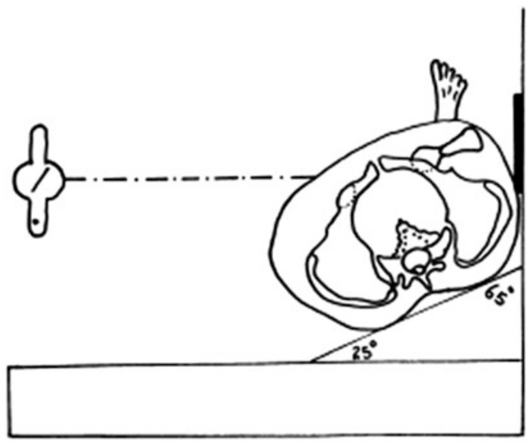


Fig. 8 Schematic of technique to obtain false profile view of the hip (Reprinted with permission from Delaunay, S., RG Dussault, PA Kaplan, and BA Alford. "Radiographic Measurements of Dysplastic Adult Hips." *Skeletal Radiology* 26.2 (1997): 75–81.)

in addition to femoral head neck abnormalities, but commonly advanced imaging will be necessary to better define this highly variable parameter.

The false profile view is used to determine anterior acetabular coverage of the femoral head [37]. It is obtained by having the patient stand against the film with the cassette at the side of their hip. The patient then turns 25° posterior and away from the coronal plane. The radiographic beam should be centered on the femoral head (Figs. 8 and 10) [23]. Similar to the LCEA, the anterior center-edge angle (ACEA) is determined on the false profile. A value less than 20° is suggestive of deficient anterolateral coverage, while values greater than 40° indicate possible overcoverage (Figs. 9 and 10). Furthermore, a prominent anterior inferior iliac spine is profiled on this view.

Computed tomography (CT) is still being defined in the treatment of both FAI and SMAD. The risks of increased radiation exposure must be weighed against the possible benefits a CT scan and 3-dimensional reconstruction may offer. CT has shown improved ability to evaluate OA compared to plain radiographs [28] decreased variability compared to plain radiographs in the measurement of LCEA and ACEA and may allow for better assessment of anterior coverage than ACEA alone [38]. This may indicate a role

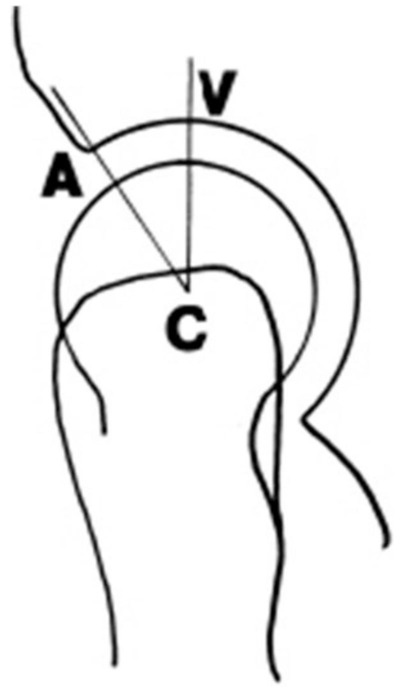


Fig. 9 The anterior center-edge angle is measured on the false profile view (Delaunay, S., RG Dussault, PA Kaplan, and BA Alford. "Radiographic Measurements of Dysplastic Adult Hips." *Skeletal Radiology* 26.2 (1997): 75–81.)

Fig. 10 Radiograph showing the false profile view of the right hip

for CT in a symptomatic patient who presents with borderline measurements (CEA between 20° and 25°) suggestive of undercoverage [26, 39]. Computed tomography can also allow for concurrent measurement of femoral head/neck offset, femoral torsion, and head tilt. This information can be helpful in preoperative planning when determining measurements and landmarks for both proximal femoral osteotomy (PFO) and PAO.

Magnetic resonance imaging (MRI) is the most utilized imaging modality to assess for labral tears and intra-articular hip pathology. Symptomatic patients with radiographic signs of dysplasia who respond to diagnostic injections are good candidates for MRI arthrograms (MRA) as labral tears and cartilage damage are found in the majority of these patients [40]. MRA utilizes an injection of approximately 4–6 cc of contrast dye into the joint and have sensitivities greater than 90 % for diagnosing labral tears [41, 42].

While excellent at demonstrating labral pathology, MRI shows much less sensitivity, approximately 50 %, for cartilage damage [43]. New protocols such as dGEMRIC (delayed gadolinium-enhanced MRI of cartilage) can help evaluate cartilage degeneration in dysplastic hips. This advanced protocol provides an assessment of the glycosaminoglycan (GAG) content and quality within cartilage and thus is an assessment of overall cartilage health. Loss or decreased quality of GAG in cartilage leads to water loss and decreased health of cartilage tissues. Increased gadolinium uptake corresponds to decreased GAG content in cartilage and is thought to indicate pathologic changes in the physiology of the cartilage which may precede physical changes. dGEMRIC protocols have already been shown to be predictive of patients who may experience early failure following PAO due to pathologic changes in cartilage that are not demonstrated on conventional MRI [44].

A unique finding in unstable dysplastic hips is presence of a hypertrophied iliocapsularis muscle. MRI can be utilized to evaluate the thickness and width in addition to the degree of fatty infiltration in the iliocapsularis muscle. Patients with dysplasia demonstrate a larger iliocapsularis muscle with far less fatty atrophy than patients with acetabular overcoverage [45]. The current hypothesis is that the muscle may have a function in hip stability in dysplastics.

Ultrasound (US) has also been utilized in the diagnosis of labral tears. Recent studies have shown high sensitivity in the diagnosis of labral tears [22, 46]; however, MRI remains the gold standard. While US is cheaper than MRI and dynamic, it is operator dependent and most facilities do not have access to technician or radiologist who are well versed in US-based assessment of hip labral tears. However, it remains a viable alternative in patients who are unable to undergo an MRI due to retained implants or claustrophobia.

Treatment

The treatment of SMAD depends on several factors. The degree to which a patient is limited by their symptoms will help determine the appropriate treatment course. Conservative management has a role in the treatment of SMAD; however, studies have shown that patients who are high activity level patients are not satisfied with the results of nonoperative management [47]. Furthermore, many patients have often seen one or more physicians over a course of several years prior to obtaining the correct diagnosis [18], and therefore, may have already failed nonsteroidal anti-inflammatory (NSAID) drugs and physical therapy. Perhaps the most important factor in the treatment is the current status of the hip joint cartilage. Joint preservation procedures have less reliable results in patients with Tonnis Grade 2 and 3 hips, and therefore, those patients with advanced OA may benefit from arthroplasty or arthrodesis options.

Conservative management of SMAD begins with NSAIDs to minimize inflammation, activity modification, and physical therapy (PT) [47]. Early studies have begun to elucidate a few helpful goals to optimize therapy [19]. The goals of physical therapy should be to focus on core strengthening of the trunk and pelvic muscles. Strengthening of these muscles may provide added stability to the unstable dysplastic hip with the goal of minimizing femoral head gliding and symptoms of instability [48]. PT should also focus on helping patients work on ADLs such as getting in and out of a car or tying a shoe which can provoke symptoms. Working on passive ROM or stretching exercises is a common reason for failure of PT. These exercises generally aggravate symptoms of FAI or dysplasia, and patients have not been found to gain ROM with their implementation. Rather, the overall goal of PT is to provide hip stability through strengthening while finding a comfortable ROM and appropriate activities that allow them to live their lives in a satisfactory manner [19].

Patients who fail conservative management should be counseled about the benefits of a diagnostic hip injection. Radiographic guided intra-articular injection of local anesthetic has very high positive predictive value for determining the source of a patient's pain. For patients with complex hip/spine complaints, a diagnostic injection can be a very effective way to determine the pain generator [49, 50]. Again it is best to not inject more than 4–6 ml of fluid into the joint as this may cause stretching of the hip capsule and exacerbate the situation [27]. Patients can be seen on the day of their hip injection so they can be reexamined. Alleviation of pain both with activity and provocative maneuvers is a strong indicator of intra-articular pathology. Patients are also instructed to keep a pain diary so as to determine the degree and duration of their symptom relief. Concomitant injection of analgesic and arthrogram dye can be performed for the sake of minimizing the number of injections if an MRA is indicated. This method may lead to either decreased amount of both dye and analgesic injected or the injection may result in too much fluid injected, possibly compromising the effect of the anesthetic or the MRA imaging.

Patients who respond well to diagnostic injections generally undergo an MRA to help determine the specific internal derangements of the hip. Low Tonnis grades (0–1) open the door for possible hip preservation surgery with the goal of repairing both the internal hip derangement and correcting the abnormal morphology of the hip. The least invasive surgical option is generally arthroscopy for labral repair. CAM deformities can also be addressed; however, the role of arthroscopy in SMAD may be limited as repair without deformity correction is unlikely to result in favorable outcomes [51]. Isolated labral repair may alleviate the symptoms of hip pain, but it will not address the underlying morphologic abnormalities that are placing pathologic stress on the labrum. Arthroscopy can be used as an adjunct to open procedures to allow more thorough access and evaluation of intra-articular pathologies, while the open portion of the procedure addresses the larger morphologic abnormalities [52].

The most commonly performed procedure for the correction of SMAD is the periacetabular osteotomy (PAO). The Bernese or Ganz PAO is the most commonly described and has the most evidence to supports its use. The osteotomy is a series of cuts are made around the acetabulum, freeing it from the pelvis while maintaining pelvic stability, allowing for freedom to reposition the hip socket (Figs. 11 and 12). A thorough explanation of the technical details of procedure is beyond the scope of this chapter; however, there are a few principles the surgeon should keep in mind when performing this procedure.

The Ganz osteotomy allows mobilization and reorientation of the acetabulum to address deficiencies and/or focal areas of overcoverage (i.e., retroversion). In the most common scenario of SMAD, there is anterolateral acetabular deficiency, and coverage is gained by laterally tilting and inwardly rotating the osteotomy. Intraoperative correction should be aimed at obtaining an LCEA of $>30°$. Concurrently extension of the osteotomy fragment will provide anterior coverage, aiming for an ACEA $>25°$. Finally the PAO also allows for medialization of the joint. Medial migration of fragment and lateral tilt will help medialize the femoral head. The goal should be to decrease the distance between the ilioischial line and the medial aspect of the femoral head to <1 cm [53].

After the osteotomy is performed, mobilized, and temporarily fixed, an image intensifier and/or a plain intraoperative radiograph should be obtained to confirm achievement of the above parameters (Figs. 13 – 15). Concurrently, a

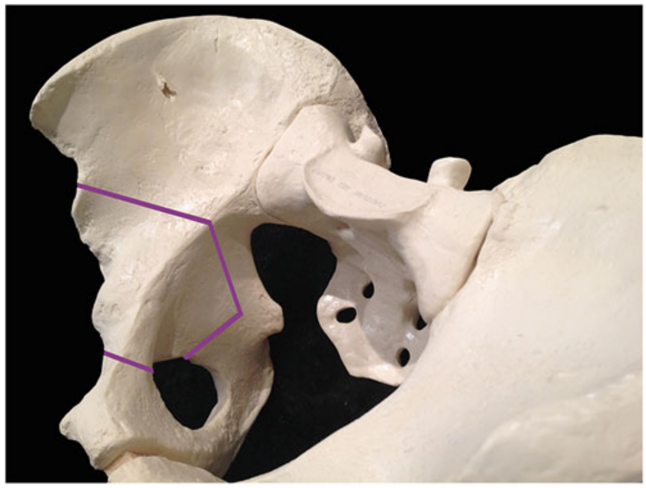


Fig. 11 Bernese osteotomy: Inner view of the pelvis

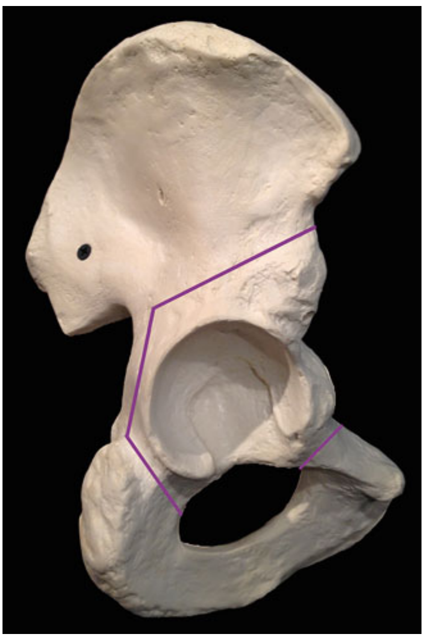


Fig. 12 Bernese osteotomy: outer view of the pelvis

Fig. 13 AP view of a dysplastic right hip prior PAO

physical exam should be performed in the OR to confirm if the patient can achieve 90° of hip flexion, 25–30° of hip abduction, and minimal loss of internal rotation. Inability to obtain appropriate range of motion can be associated with improper positioning of the acetabular fragment. After securing the osteotomy site, a capsulotomy may be performed to inspect the femoral neck, acetabular margin, and the labrum and assess for impingement. Labral tears can be debrided or repaired, although there is no data to support its improving outcomes. A femoral neck osteoplasty should be performed if appropriate positioning of the acetabular fragment results in decreased motion or obvious impingement. Finally, although the PAO is a powerful procedure, some patient may still have lateralization of the femoral head. If associated with coxa valga or excessive femoral antetorsion, a proximal femoral osteotomy may be necessary to achieve a congruent, stable

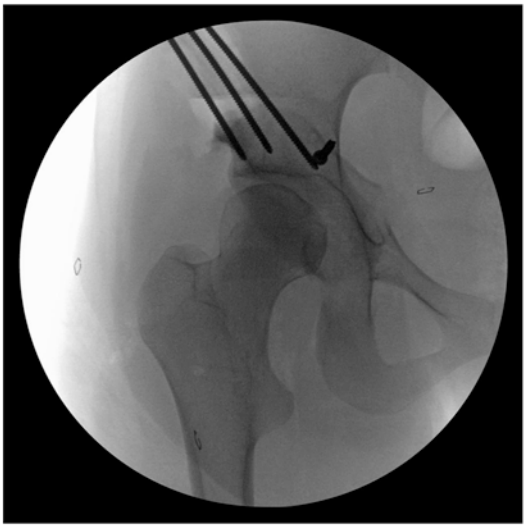


Fig. 14 Intraoperative AP view of a dysplastic right hip after reorientation of the acetabulum

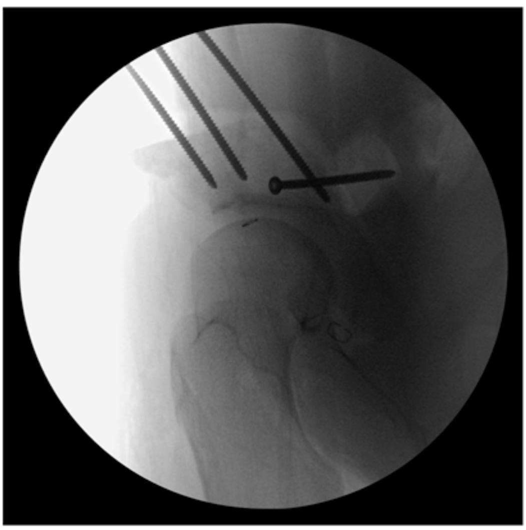


Fig. 15 Intraoperative false profile view of a right hip after a PAO showing increased coverage of the femoral head

articulation. In the author's experience, PFO is more commonly needed in the setting of more severe acetabular dysplasias (Figs. 16 and 17).

Patients with more significant osteoarthritis (Tonnis Grade the higher risk of early failure 2 or 3) have less predictable, outcomes with joint preservation surgery and should be counseled about arthroplasty or arthrodesis options [30]. Depending on age and associated musculoskeletal or medical comorbidities, arthroplasty or arthrodesis may be the best option in patients with advanced arthrosis (Grade 2–3). Total hip arthroplasty is one of the most successful procedures in modern medicine and good results have been obtained in patients with complex dysplasia, although increasingly complex substitute dysplasia may compromise outcomes [6, 54].

In conclusion, skeletally mature acetabular dysplasia is an architectural problem of the acetabulum that can lead to pathophysiologic changes in the cartilage and labrum of the hip. Increased load at the hip joint can lead to pain and eventual cartilage damage and OA of the hip joint. Careful clinical and radiographic workup is necessary to evaluate the subtle but significant abnormalities that distinguish SMAD from FAI patterns. Early intervention to preserve the hip joint, specifically PAO, can help improve the mechanics of the hip joint to a more anatomic position to allow for proper force transfer across the hip joint. Restoration of hip mechanics can help restore a physiologically sound environment for the patient with the goal of alleviating symptoms and prolonging the functional life of the hip joint.

Summary

Skeletally mature acetabular dysplasia (SMAD) is becoming a more recognized cause of pain in active young adults. Abnormal force transmission through the hip joint may result in labral pathology, cartilage damage, and, eventually, osteoarthritis. The constellation of symptoms seen in SMAD is similar to those seen in FAI and mild osteoarthritis.

The cornerstone of management of SMAD is not only treatment but proper identification of specific anatomic abnormalities. Imaging studies should include plain radiographs of the involved hip. Radiographic parameters such as the anterior center-edge angle, the lateral center-edge angle of Wiberg, and the acetabular index of Tonnis should be measured. Careful evaluation of acetabular version is also important as this may indicate an isolated pathology and may be associated with posterior superior coverage deficiencies or a

Fig. 16 Preoperative X-Rays of a patient with severe right acetabular dysplasia and coxa valga

Fig. 17 Post-operative X-Rays of a patient with left acetabular dysplasia after a PAO and PFO. Rim fracture reduced and stabilized

more classic lateral and anterior deficiency with concomitant retroversion. Classical signs of acetabular retroversion include a prominent ischial spine, a crossover sign, and a relatively enlarged profile of the ipsilateral iliac wing.

Treatment of SMAD depends on the condition of the hip joint at the time of diagnosis. The goal for patients without radiographic signs of osteoarthritis is hip joint preservation. Generally, a reorientation procedure such as a periacetabular osteotomy (PAO) is performed. The Bernese periacetabular osteotomy has been extensively described and has the potential for global repositioning of the acetabulum, allowing for correction to address areas of undercoverage or overcoverage. This procedure also medializes the femoral head. Patients who present with moderate to severe OA generally do not improve with hip preservation procedures, and these patients are better candidates for arthroplasty. Although THA has excellent results, prompt recognition and treatment of SMAD with hip preservation procedures is the mainstay of treatment in young individuals without evidence of OA.

References

1. Crawford MJ, Dy CJ, Alexander JW, Thompson M, Schroder SJ, Vega CE, et al. The 2007 Frank Stinchfield award. The biomechanics of the hip labrum and the stability of the hip. Clin Orthop Relat Res. 2007;465:16–22.
2. Ferguson SJ, Bryant JT, Ganz R, Ito K. An in vitro investigation of the acetabular labral seal in hip joint mechanics. J Biomech. 2003;36(2):171–8.
3. McCarthy JC, Noble PC, Schuck MR, Wright J, Lee J. The watershed labral lesion: its relationship to early arthritis of the hip. J Arthroplasty. 2001;16(8 Suppl 1):81–7.
4. Li PL, Ganz R. Morphologic features of congenital acetabular dysplasia: one in six is retroverted. Clin Orthop Relat Res. 2003;416:245–53.
5. Mast JW, Brunner RL, Zebrack J. Recognizing acetabular version in the radiographic presentation of hip dysplasia. Clin Orthop Relat Res. 2004;418:48–53.
6. MacKenzie JR, Kelley SS, Johnston RC. Total hip replacement for coxarthrosis secondary to congenital dysplasia and dislocation of the hip. Long-term results. J Bone Joint Surg Am. 1996;78(1):55–61.
7. Murphy SB, Ganz R, Muller ME. The prognosis in untreated dysplasia of the hip. A study of radiographic factors that predict the outcome. J Bone Joint Surg Am. 1995;77(7):985–9.
8. Jessel RH, Zurakowski D, Zilkens C, Burstein D, Gray ML, Kim YJ. Radiographic and patient factors associated with pre-radiographic osteoarthritis in hip dysplasia. J Bone Joint Surg Am. 2009;91(5): 1120–9.
9. Ganz R, Leunig M, Leunig-Ganz K, Harris WH. The etiology of osteoarthritis of the hip: an integrated mechanical concept. Clin Orthop Relat Res. 2008;466 (2):264–72.
10. Klaue K, Durnin CW, Ganz R. The acetabular rim syndrome. A clinical presentation of dysplasia of the hip. J Bone Joint Surg Br. 1991;73(3):423–9.
11. Hashimoto S, Rai MF, Gill CS, Zhang Z, Sandell LJ, Clohisy JC. Molecular characterization of articular cartilage from young adults with femoroacetabular impingement. J Bone Joint Surg Am. 2013;95(16): 1457–64.
12. Troelsen A. Assessment of adult hip dysplasia and the outcome of surgical treatment. Dan Med J. 2012;59(6): B4450.
13. Peled E, Eidelman M, Katzman A, Bialik V. Neonatal incidence of hip dysplasia: ten years of experience. Clin Orthop Relat Res. 2008;466(4):771–5.
14. Howorth B. “Congenital dislocation” of the hip . . . its early diagnosis and treatment. Arch Pediatr. 1962;79: 1–17.
15. Lee CB, Mata-Fink A, Millis MB, Kim YJ. Demographic differences in adolescent-diagnosed and adult-diagnosed acetabular dysplasia compared with infantile developmental dysplasia of the hip. J Pediatr Orthop. 2013;33(2):107–11.
16. Gosvig KK, Jacobsen S, Sonne-Holm S, Palm H, Troelsen A. Prevalence of malformations of the hip joint and their relationship to sex, groin pain, and risk of osteoarthritis: a population-based survey. J Bone Joint Surg Am. 2010;92(5):1162–9.
17. Sankar WN, Nevitt M, Parvizi J, Felson DT, Agricola R, Leunig M. Femoroacetabular impingement: defining the condition and its role in the pathophysiology of osteoarthritis. J Am Acad Orthop Surg. 2013;21 Suppl 1:S7–15.
18. Nunley RM, Prather H, Hunt D, Schoenecker PL, Clohisy JC. Clinical presentation of symptomatic acetabular dysplasia in skeletally mature patients. J Bone Joint Surg Am. 2011;93 Suppl 2:17–21.
19. Wall PD, Fernandez M, Griffin DR, Foster NE. Nonoperative treatment for femoroacetabular impingement: a systematic review of the literature. PM & R J Inj Funct Rehabil. 2013;5(5):418–26.
20. Clohisy JC, Knaus ER, Hunt DM, Lesher JM, Harris-Hayes M, Prather H. Clinical presentation of patients with symptomatic anterior hip impingement. Clin Orthop Relat Res. 2009;467(3):638–44.
21. Shu B, Safran MR. Hip instability: anatomic and clinical considerations of traumatic and atraumatic instability. Clin Sports Med. 2011;30(2):349–67.
22. Troelsen A, Mechlenburg I, Gelineck J, Bolvig L, Jacobsen S, Soballe K. What is the role of clinical tests and ultrasound in acetabular labral tear diagnostics? Acta Orthop. 2009;80(3):314–8.
23. Clohisy JC, Carlisle JC, Beaule PE, Kim YJ, Trousdale RT, Sierra RJ, et al. A systematic approach to the plain radiographic evaluation of the young adult hip. J Bone Joint Surg Am. 2008;90 Suppl 4:47–66.
24. Tannast M, Zheng G, Anderegg C, Burckhardt K, Langlotz F, Ganz R, et al. Tilt and rotation correction of acetabular version on pelvic radiographs. Clin Orthop Relat Res. 2005;438:182–90.
25. Siebenrock KA, Kalbermatten DF, Ganz R. Effect of pelvic tilt on acetabular retroversion: a study of pelvis from cadavers. Clin Orthop Relat Res. 2003;407:241–8.
26. Troelsen A, Jacobsen S, Romer L, Soballe K. Weightbearing anteroposterior pelvic radiographs are recommended in DDH assessment. Clin Orthop Relat Res. 2008;466(4):813–9.
27. Nepple JJ, Prather H, Trousdale RT, Clohisy JC, Beaule PE, Glyn-Jones S, et al. Clinical diagnosis of femoroacetabular impingement. J Am Acad Orthop Surg. 2013;21 Suppl 1:S16–9.

28. Troelsen A, Romer L, Kring S, Elmengaard B, Soballe K. Assessment of hip dysplasia and osteoarthritis: variability of different methods. Acta Radiol. 2010;51(2): 187–93.
29. Troelsen A, Elmengaard B, Soballe K. Medium-term outcome of periacetabular osteotomy and predictors of conversion to total hip replacement. J Bone Joint Surg Am. 2009;91(9):2169–79.
30. Hartig-Andreasen C, Troelsen A, Thillemann TM, Soballe K. What factors predict failure 4 to 12 years after periacetabular osteotomy? Clin Orthop Relat Res. 2012;470(11):2978–87.
31. Clohisy JC, Barrett SE, Gordon JE, Delgado ED, Schoenecker PL. Medial translation of the hip joint center associated with the Bernese periacetabular osteotomy. Iowa Orthop J. 2004;24:43–8.
32. Fujii M, Nakashima Y, Yamamoto T, Mawatari T, Motomura G, Matsushita A, et al. Acetabular retroversion in developmental dysplasia of the hip. J Bone Joint Surg Am. 2010;92(4):895–903.
33. Reynolds D, Lucas J, Klaue K. Retroversion of the acetabulum. A cause of hip pain. J Bone Joint Surg Br. 1999;81(2):281–8.
34. Kalberer F, Sierra RJ, Madan SS, Ganz R, Leunig M. Ischial spine projection into the pelvis: a new sign for acetabular retroversion. Clin Orthop Relat Res. 2008;466(3):677–83.
35. Peters CL, Erickson JA, Hines JL. Early results of the Bernese periacetabular osteotomy: the learning curve at an academic medical center. J Bone Joint Surg Am. 2006;88(9):1920–6.
36. Meyer DC, Beck M, Ellis T, Ganz R, Leunig M. Comparison of six radiographic projections to assess femoral head/neck asphericity. Clin Orthop Relat Res. 2006;445:181–5.
37. Lequesne M, Djian A. New radiographic angles for the study of the hip. The "false profile" of the pelvis. Vie Med. 1961;42:1629–41.
38. Sakai T, Nishii T, Sugamoto K, Yoshikawa H, Sugano N. Is vertical-center-anterior angle equivalent to anterior coverage of the hip? Clin Orthop Relat Res. 2009;467(11):2865–71.
39. Troelsen A, Romer L, Jacobsen S, Ladelund S, Soballe K. Cranial acetabular retroversion is common in developmental dysplasia of the hip as assessed by the weight bearing position. Acta Orthop. 2010;81(4): 436–41.
40. Leunig M, Podeszwa D, Beck M, Werlen S, Ganz R. Magnetic resonance arthrography of labral disorders in hips with dysplasia and impingement. Clin Orthop Relat Res. 2004;418:74–80.
41. Toomayan GA, Holman WR, Major NM, Kozlowicz SM, Vail TP. Sensitivity of MR arthrography in the evaluation of acetabular labral tears. AJR Am J Roentgenol. 2006;186(2):449–53.
42. Freedman BA, Potter BK, Dinauer PA, Giuliani JR, Kuklo TR, Murphy KP. Prognostic value of magnetic resonance arthrography for Czerny stage II and III acetabular labral tears. Arthroscopy. 2006;22(7):742–7.
43. Keeney JA, Peelle MW, Jackson J, Rubin D, Maloney WJ, Clohisy JC. Magnetic resonance arthrography versus arthroscopy in the evaluation of articular hip pathology. Clin Orthop Relat Res. 2004;429:163–9.
44. Cunningham T, Jessel R, Zurakowski D, Millis MB, Kim YJ. Delayed gadolinium-enhanced magnetic resonance imaging of cartilage to predict early failure of Bernese periacetabular osteotomy for hip dysplasia. J Bone Joint Surg Am. 2006;88(7):1540–8.
45. Babst D, Steppacher SD, Ganz R, Siebenrock KA, Tannast M. The iliocapsularis muscle: an important stabilizer in the dysplastic hip. Clin Orthop Relat Res. 2011;469(6):1728–34.
46. Troelsen A, Jacobsen S, Bolvig L, Gelineck J, Romer L, Soballe K. Ultrasound versus magnetic resonance arthrography in acetabular labral tear diagnostics: a prospective comparison in 20 dysplastic hips. Acta Radiol. 2007;48(9):1004–10.
47. Hunt D, Prather H, Harris Hayes M, Clohisy JC. Clinical outcomes analysis of conservative and surgical treatment of patients with clinical indications of prearthritic, intra-articular hip disorders. PM & R J Inj Funct Rehabil. 2012;4(7):479–87.
48. Lewis CL, Sahrmann SA. Acetabular labral tears. Phys Ther. 2006;86(1):110–21.
49. Byrd JW, Jones KS. Diagnostic accuracy of clinical assessment, magnetic resonance imaging, magnetic resonance arthrography, and intra-articular injection in hip arthroscopy patients. Am J Sports Med. 2004;32(7):1668–74.
50. Pateder DB, Hungerford MW. Use of fluoroscopically guided intra-articular hip injection in differentiating the pain source in concomitant hip and lumbar spine arthritis. Am J Orthop (Belle Mead NJ). 2007;36(11):591–3.
51. Bogunovic L, Gottlieb M, Pashos G, Baca G, Clohisy JC. Why do hip arthroscopy procedures fail? Clin Orthop Relat Res. 2013;471(8):2523–9.
52. Clohisy JC, Beaule PE, O'Malley A, Safran MR, Schoenecker P. AOA symposium. Hip disease in the young adult: current concepts of etiology and surgical treatment. J Bone Joint Surg Am. 2008;90(10): 2267–81.
53. Schoenecker PL, Clohisy JC, Millis MB, Wenger DR. Surgical management of the problematic hip in adolescent and young adult patients. J Am Acad Orthop Surg. 2011;19(5):275–86.
54. Numair J, Joshi AB, Murphy JC, Porter ML, Hardinge K. Total hip arthroplasty for congenital dysplasia or dislocation of the hip. Survivorship analysis and long-term results. J Bone Joint Surg Am. 1997;79(9): 1352–60.

Surgical Technique: Periacetabular Osteotomy

45

Martin Beck, Nicholas J. Lash, and Reinhold Ganz

Contents

M. Beck (✉) • N.J. Lash
Department of Orthopaedics, Luzerner Kantonsspital, Luzern, Switzerland
e-mail: martin.beck@luks.ch; nicholas.lash@xtra.co.nz

R. Ganz
Emeritus Inselspital Bern, Bern, Switzerland

Faculty of Medicine, University of Bern, Bern, Switzerland
e-mail: rd.ganz@bluewin.ch

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_48

Abstract

Hip dysplasia and acetabular retroversion are two common causes of hip pain and dysfunction and are potential precursors to degenerative joint disease. If these disorders are addressed in a timely fashion, then intra-articular damage, or the potential thereof, can be curtailed. The manner in which these disorders are treated includes periacetabular osteotomy (PAO) as one option.

Periacetabular osteotomy to reorientate the acetabulum remains a powerful tool in the armamentarium of the hip surgeon. The benefits of PAO include osteotomies made close to the joint allowing substantial corrections to be achieved, leaving the posterior column intact creating enhanced stability requiring less internal fixation, and not altering the internal diameter of the pelvis for childbearing. The approach is soft tissue friendly, and crucial blood supply is maintained allowing the ability to perform capsulotomy and address coexisting intra-articular pathology. The PAO is technically demanding and has an inherent learning curve, but long-term results show excellent outcomes in properly selected patients.

Introduction

Hip pain and osteoarthritis (OA) were once considered the realm of the elderly patient. Unfortunately, both of these entities are becoming more common to the young (<50-year-old) patient. Whether the presence of hip pain in this group was always evident and just underreported or whether lifestyle changes and patient's acceptance of disability is diminishing, it is apparent that more young patients are seeking treatment for painful hips.

The cause of hip pain in the young adult is invariably related to mechanical causes. Once considered "primary idiopathic osteoarthritis," Harris [1] identified that in hips originally thought to have primary osteoarthritis, an identifiable structural abnormality can be present in up to 90 % of cases. Thirty-nine percent of the structural abnormalities were attributed to developmental dysplasia. This is supported by Stulberg et al. [2], who found over 40 % of hips with OA had unreported features of dysplasia.

Hip dysplasia is a spectrum of deficiency of the acetabulum. It can exist as a combination of several changes. Most commonly there is anterolateral deficiency in the acetabulum, generally accompanied with a steep angle of roof inclination. The femoral head can be subluxed or even dislocated with or without a false acetabulum. Associated femoral neck deformities are possible, increased coxa valga and asphericity of the femoral head being common [3].

The loss of femoral head coverage leads to altered biomechanics with instability and subluxation of the head. The changed force vectors can lead to abductor muscle fatigue, a source of limp and pain. With the acetabular deficiency and reduced femoral head coverage, there are increased stress and contact pressures at the acetabular margin, which lead to injury of the labrum and the articular cartilage, potentially ending in osteoarthritis [4, 5].

The natural history of dysplasia with hip subluxation is well recognized. Stulberg et al. [2] and Wiberg [6] have demonstrated that the dysplastic hip with subluxation has a high chance of progressing to osteoarthritis. It is known that patients with subluxation will often have symptoms ensuing in mid-30s for females and mid-40s for males. It is also known that over time, this group will have demonstrable degeneration on radiographs on average one decade later. However, the natural history of dysplasia without subluxation is still uncertain. Wiberg in his original series noted a linear relationship between the severity of dysplasia as measured by the center-edge angle (CEA) and the rates of developing osteoarthritis [6]. On the contrary, Cooperman et al. [7] found that in 32 hips with dysplasia, monitored over 22 years, a clear linear correlation between the severity of dysplasia and development of degenerative changes was not evident. This highlights that currently, the natural history of dysplastic hips without subluxation is not entirely clear. It is estimated that between 40 % and 50 % of dysplastic hips will develop

OA before 50 years of age, with 50 % of some studied groups requiring their first reconstructive procedure before the age of 60 [1].

In addition to dysplasia of the hip, a relatively new entity known as acetabular retroversion is becoming more recognized. Originally identified as cause of osteoarthritis by Preiser in 1907 [8] and confirmed by Teinturier in 1970 [9], Reynolds et al. [10] described it as "a separate entity or part of a complex dysplasia." It exists with the structural abnormality being that the opening, or "mouth," of the acetabulum faces in a posterolateral fashion, instead of the usual anterolateral orientation. In its more severe forms, retroversion of the acetabulum causes anterior over-coverage and posterior deficiency. The anterior aspect of the acetabulum or "rim" becomes prominent and acts as a source of impingement. Hence, retroversion of the acetabulum is one common form of femoroacetabular impingement (FAI) [11, 12]. With bone impingement, repetitive contact stresses over time can cause injury to the chondrolabral complex. In addition to this anterosuperior zone of injury, posterior capsular strain can occur as the femoral head levers out posteriorly due to posterior acetabular wall deficiency. With repeated translations, the capsule can sustain injury and is a potential source of pain. Although a relatively newly identified pathology, the incidence of acetabular retroversion has been reported as between 6 % and 15 % [13, 14].

Basic Science

The structural differences between dysplastic and retroverted acetabula and normal acetabula need to be understood prior to committing to corrective surgery. In dysplastic hips, the acetabulum is most often deficient anterolaterally, and the roof of the acetabulum can be short and inclined at a higher angle. It is also important to recognize that the dysplastic acetabulum is retroverted in about one in six [15] to one-third of the hips [16], while in non-dysplastic hips, the incidence of acetabular retroversion has been reported as between 6 % and 15 % [13, 14]. The lack of coverage over the femoral head and the increased angle of the acetabulum create a situation where the femoral head becomes unstable. If not fully dislocated or subluxed, as can happen in severe dysplasia, the femoral head can sit in a more lateral position. This creates excess load and contact pressure at the margin of the residual acetabulum.

Under shear stress, the labrum will hypertrophy. With the increased tissue mass, relative stability may be achieved; however, over time with exposure to chronic shear stress, the labrum can sustain injury. Tears can occur within the substance of the labrum or the labrum itself can tear off the acetabular margin occasionally together with the adjacent acetabular cartilage. Fractures of the acetabular rim may occur and usually are propagated through preexisting bone cysts. Labral injury causes pain with flexion and internal rotation, and as the compensating soft tissues fail, the femoral head is prone to subluxation [17].

Histomorphologically, the labrum undergoes myxoid degeneration. In this cellular form, the collagen structure is weaker and prone to tears, with the myxoid material potentially leading to the formation of labral and para-labral cysts. The articular surface cartilage is also prone to injury. The chondrolabral junction in particular sustains increased load and stress resulting in thinning or even delamination of the articular cartilage. In addition to the cartilage pathology, the subchondral bone undergoes a reactive increase in density. This can be seen in plain radiographs as increased opacity or sclerosis at the acetabular margin. Overtime, subchondral cyst formation may occur. This is a marker of significant intra-articular pathology and, as mentioned, is a potential source of rim fracture.

While retroversion of the acetabulum has a different orientation abnormality to dysplasia, many of the pathological changes are similar. Instead of anterosuperior deficiency, in retroversion, there is excess bone anteriorly. It has been shown that in retroversion, the acetabulum has a normal shape and volume, but the acetabulum is oriented posteriorly [18]. The anterior femoral head coverage is more than usual, and this reduces the space between the femoral neck and acetabular rim. Because of its location, during hip flexion and internal rotation, the femoral neck impinges at reduced ranges of motion. With impingement, there is a propensity for the labrum to become compressed between the femur and acetabulum. Over time, this can lead to labral tears and, again, damage at the chondrolabral junction and articular cartilage. Chronic impingement against the acetabular rim will lead to bone apposition followed by thinning of the labrum, the end result being an osseous rim [19]. This further increases the "pincer" effect, and the joint loses the suction effect leading to poor lubrication and further encouraging cartilage degeneration [20].

Principles of Pelvic Osteotomy

Regardless of the indication, pelvic osteotomies are used to correct for incorrect orientation of the acetabulum. Most commonly used in hip dysplasia, the history of pelvic osteotomies is of either reshaping, augmentation/salvage, or reorientation.

Reshaping osteotomies as the Pemberton [21] and Dega [22] osteotomies rely on plastic deformation through the triradiate cartilage and are thus important in the pediatric patient population. They are not considered part of the scope of this text.

Augmentation procedures which include the Chiari osteotomy [23], the shelf procedure [24], and the capsular arthroplasty [25] look to augment the deficient acetabulum by increasing the weight-bearing area by using the pelvic bone. This can be done by osteotomizing the pelvis and medializing the acetabulum creating additional bone laterally, as in the Chiari osteotomy. Conversely, with the shelf procedure, free bone graft can be placed laterally. With the capsular arthroplasty, the femoral head covered entirely with capsular tissue is relocated in a socket reamed out at the original level. The interposed hip capsule acts as a weight-bearing surface and will undergo metaplasia to fibrocartilage. While an alternative, the mechanical properties of the metaplastic fibrocartilage are inferior to those properties of the native hyaline cartilage and subchondral bone. For these reasons, augmentation procedures are considered salvage procedures.

Reorientation osteotomies have largely been developed for dysplastic hips, and majority of the literature revolves around dysplasia management. Single, double, triple, spheric, and periacetabular osteotomies have been performed with a variety of bone cuts. Single osteotomies, such as the single innominate osteotomy described by Salter [26], have been widely used for dysplastic hips in children. The single bone cut proximal to the acetabulum allows for rotation of the acetabulum anteriorly and laterally to correct anterosuperior deficiency. This osteotomy relies on mobility in the symphysis pubis to gain correction. Due to age-related stiffness in the symphysis, this osteotomy becomes less powerful in adolescents and adults. Rotation around the symphysis can cause the acetabular fragment to lateralize the hip joint and to create posterior acetabular deficiency. A variety of double and triple osteotomies have also been described [27–30]. The addition of supplementary cuts allows greater mobility of the acetabular fragment. However, in an attempt to gain extra mobility to gain correction, the additional bone cuts will often render the pelvic ring unstable, and therefore, these procedures require greater fixation to achieve stability. The bone cuts are made distant to the acetabulum, which has several implications. The size of the fragment remains large, and the attachment of the sacropelvic ligaments can make the degree of correction difficult. The size of the fragment also has the potential to alter the internal diameter of the true pelvis; this can have an impact for female patients of childbearing age. In an attempt to negate some of these issues, osteotomies can be made closer to the acetabulum [31, 32]. This allows for greater mobility to achieve correction but does not change the need for increased fixation or to alter the internal pelvic diameter. Spherical osteotomies [33–35] have been used with corrections being able to be achieved due to the close proximity to the joint. The correction of anterolateral coverage is quite achievable; however, there are disadvantages. The ability to medialize the joint for better joint reaction forces and correction of version are limited, and the proximity to the joint renders the blood supply to the acetabular fragment to be at risk. The fragment relies on the retention of blood supply through the acetabular branch of the obturator artery and the blood supply of the joint capsule. Capsulotomy is, therefore, not recommended as an adjunct with spherical osteotomies.

Given the listed limitations of the various osteotomies mentioned above, the Bernese periacetabular osteotomy (PAO) was developed [36]. This juxta-articular, polygonally shaped osteotomy has many advantages. Because of its location close to the joint, it permits substantial correction. Rotation to give anterior and superior coverage is freely possible. In cases of retroversion, the fragment is able to be mobilized to correct for anterior over-coverage and posterior deficiency. The ability to medialize the joint to enhance joint reaction forces is also possible. Because the posterior bone cuts leave the posterior column of the acetabulum intact, there is enhanced stability. This often means minimal fixation is needed to secure the fragment. The intact posterior column also acts to protect the sciatic nerve during osteotomy. The location of the bone cuts respects the blood supply to the acetabular fragment from the branches from the gluteal vessels and also allows an anterior capsulotomy to be performed if required to address concomitant intra-articular pathology or impingement sources [37].

Preoperative Assessment and Planning

History

The history for a patient with a symptomatic hip may vary according to the underlying pathology. Most patients undergoing PAO will be young and have hip dysplasia. These patients may present with one or more of several symptoms. Laterally located hip pain can be present due to abductor muscle fatigue, gluteal muscle tendonitis, and trochanteric bursitis. Anterior hip or groin pain is common and is often due to labral pathology. It can present as a sharp stabbing pain, which comes on acutely and will abate as fast as it occurred. Labral tears can occasionally cause a sensation of a click or feeling of obstruction of motion, this may be relieved by attempts at freeing the motion of the hip. With progressive degenerative changes, arthritic pain can be present. This can be felt as a dull, deep ache in the groin with or without radiation to the thigh and knee. It is usually brought on by activity and relieved by rest, but in its most symptomatic state will present as nocturnal pain. In severe dysplasia, the patient or clinician may also notice leg length discrepancy, which if significant can also cause compensatory changes in the lumbar spine, resulting in lower back pain and perhaps scoliosis. Abductor fatigue and leg length discrepancy are two causes of limp, a common symptom. Patients with dysplasia may even get symptoms of instability. Typically, this would occur with the hip in extension and external rotation. It may cause anterior discomfort and feelings of apprehension.

Patients with retroversion of the acetabulum will also feel pain in similar locations to dysplastic patients. This is often due to underlying damage to the labrum. However, the mechanism of labral injury is quite different. The cause of labral injury in the retroverted acetabulum is femoroacetabular impingement. Patients will usually be able to identify certain activities that encourage the onset of pain, particularly activity that involves repeated flexion and rotation. While mechanical locking or clicking due to chondrolabral pathology is possible, abductor fatigue, subluxation, and leg length discrepancy are not an issue in retroversion of the acetabulum. Patients with high femoral anteversion but also patients with prominent posterior wall as seen in deep sockets and protrusio may have posterior pain; when posterior osteoarthritis is already present, night pain is a typical indicator. Ballet dancers may complain about posterior pain, because they impinge when professionally performing extreme external rotation in full extension.

Notwithstanding the considerable symptoms endured by these patients, they are usually young and can often present due to the functional disability caused by their pathology. A functional history on the impact of their hip disorder is also an important part of the history taking.

Finally, it should be also recognized that hip preservation surgery is performed in patients that at times have very little subjective symptoms. This may be due to only very early or little intra-articular damage or also in the case of significant subluxation/dislocation of the femoral without the formation of a false acetabulum, which can surprisingly be free of pain. However, limited internal rotation or migration of a femoral head in an acetabular socket is also a symptom, severe enough to discuss surgery in a given case even when pain or discomfort is negated.

Physical Examination

As with any patient with hip complaints, a standard examination of the hip consisting of inspection, palpation, and motion assessment is performed. The gait and lumbar spine should also be considered in the examination process. Patients with hip dysplasia may also have several additional features that could be present. The abductor mechanism should be examined for weakness. A Trendelenburg test should be performed, and additionally the power of the abductors should be assessed with the patient in the lateral position with resisted abduction. While in this position, the trochanteric area can be palpated for bursitis. Systematic palpation of the anterior, lateral, and posterior facets of the trochanter can be made. The patient can also be asked to "cycle" their leg as if they were on a bicycle, while the clinician palpates for tenderness and snapping of the tractus. Very commonly, there is increased internal rotation of the hip joint. This is best assessed with the patient supine with the hip flexed to 90°. Damage of the acetabular rim is assessed with the *impingement test*. With the hip flexed to 90°, additional adduction is performed while at the same time internal rotation is imparted. As the femoral neck rotates and flexes, any labral tear is compressed between the femoral neck and acetabular margin and may cause pain. The triggering of pain is considered as positive impingement test and is very sensitive for intra-articular pathology but unspecific regarding the cause. In significant dysplasia, an *apprehension sign* may be present. The patient lies supine, and the leg is moved into extension and simultaneously adducted and externally rotated. This can cause discomfort as the femoral head tries to sublux anteriorly and in doing so creates "apprehension" for the patient; this test can also be significative in borderline dysplastic hips, painful after arthroscopy. If the femoral head does indeed sublux, then it can be palpated as a mass in the anterior groin. This is usually only palpable in slim patients but when present is known as a *lump sign*. Lastly, disorders of the psoas tendon are possible and should be examined. Psoas inflammation is present if there is deep groin pain, and in slim patients the tendon can be palpated with the hip in flexion. Audible or palpable "snapping" of the tendon can be elicited by active circumduction. This is performed by simultaneously flexing, externally rotating, abducting, and extending the leg. The "snap" usually occurs at the transition from abduction-external rotation back into hip extension.

Medical Imaging/Diagnostic Studies

Conventional Radiography

There are four plain radiographs which are mandatory in preoperative assessment of a patient undergoing PAO. All must be taken with care so ensure that the radiograph is of appropriate quality to be making preoperative observations and measurement.

Anteroposterior Pelvis Radiograph

An appropriate AP pelvis radiograph needs to include both iliac crests down to the lesser trochanters. The femur should be held in neutral or even slight internal rotation. Malrotation should not be present and can be checked for by assessing the symmetry of the obturator foramina, the tear drops, and the symphysis pubis being in line with the coccyx. Appropriate tilt is required for accurate measurement, and ideally the sacrococcygeal junction should be 2 cm above the symphysis pubis. Adequate exposure on the film is necessary to be able to identify the anterior and posterior acetabular walls to identify acetabular version.

The radiographic parameters for dysplasia that should be measured include: lateral center-edge angle (LCEA) (normal between 25° and 33°), roof angle (normal between 0° and 10°), extrusion index, neck shaft angle, anterior and posterior rim, acetabular retroversion, Shenton's line, and medialization/lateralization of the head by checking and comparing the distance between the ilioischial line either to the medial border of the head or to the center of rotation. The inferior acetabular rim is identified and its distance to the inferior border of the head is estimated.

The radiographic parameters for acetabular retroversion include: LCEA, roof angle, crossover sign, ischial spine sign, acetabular retroversion index, posterior wall sign, and distance between the head and ilioischial line in coxa profunda and protrusio.

Cross-Table Lateral Radiograph

This radiograph gives a direct lateral view of the femoral head. It will allow the detection of aspheric anterior head neck junction or a nonexisting anterior wasting. If the positioning of the thigh is appropriately performed with the patella facing directly anteriorly, then an estimation of the femoral antetorsion can also be made.

False Profile View

This gives the clinician an oblique radiograph that allows assessment of the anterior margin of the acetabulum. Originally described by Lequesne [38], measurement of the anterior CEA (ACEA) is made; if the measurement is less than 20°, then there is a deficient anterior acetabulum. In hips with coxa profunda or protrusio, the false profile view is best to show the eventual posteroinferior joint space narrowing.

Abduction AP Hip Radiograph

It is only meaningful when the femoral rotation is neutral or even slightly internally rotated. This radiograph will assess whether the femoral head can be concentrically maintained or reduced within the acetabulum. The abduction moment should cause the femoral head to rotate and translate medially into the acetabulum. However, in cases of hinge abduction, the femoral head can "lever out" staying in a lateral position and not achieving a concentric reduction. This is a critical investigation, especially in dysplastic hips with laterally displaced femoral heads starting in a nonconcentric position. When the abduction view shows narrowing of the lateral joint space, this space must become wider with a repeated radiography with additional flexion of the femur to qualify for a PAO indication.

Computed Tomography (CT)

Good standardized radiographs allow already for correct assessment of acetabular anatomy. Occasionally, additional CT imaging can be helpful to visualize complex deformities. CT-based imaging can be of use in patients with acetabular retroversion. The axial slices are best used to make angular measurements from the anterior wall to the posterior wall. The angle subtended in relation to the sagittal plane gives the anteversion of the acetabulum. This can be correlated with the contralateral hip. It is also helpful to create 3D reformats that can demonstrate associated cam deformities.

Magnetic Resonance Imaging

MRI and MRA (magnetic resonance arthrography) of the diseased hip only are the current standard of assessing the periarticular soft tissues of the hip. Non-contrast-enhanced MRI is useful for assessing anatomical structures such as the psoas tendon, gluteus medius tendon, trochanteric bursa, and femoral head vascularity. Anatomical structures are usually best seen on T1-weighted sequences with the T2-weighted sequences better suited for identifying pathology. High signal on T2 will identify fluid/inflammation of any of the above structures and any labral or bone cysts. It will also identify intra-articular synovitis. MRA has the benefit of intra-articular contrast enhancement with gadolinium-based contrast medium. This allows visualization of the intra-articular structures, namely, the labrum and ligamentum teres. With specific proton density-weighted radial sequences made in the axis of the femoral neck, a circumferential view of the acetabular margin and the femoral neck can be made. This is useful for identifying labral tears and cartilage loss/damage.

Indications for Periacetabular Osteotomy

- Primary and secondary hip dysplasia close to (10 years of age) or after closure of physes/skeletal maturity
- AP pelvis abduction radiograph with a maintained or achieved congruent hip in 20–30° of hip abduction
- Acetabular retroversion in the presence of symptoms and signs of FAI
- Additional indication as reverse PAO for deep socket with negative roof angle and protrusio without major osteoarthritis

PAO in combination with femoral osteotomy has to be considered for severe dysplasia, aspheric congruency, high-riding trochanter, and high femoral anteversion.

Contraindications for Periacetabular Osteotomy

- High subluxation with a head articulation against a secondary acetabulum
- Dislocated femoral head with or without a false acetabulum
- Established osteoarthrosis
- Worsening concentricity of joint/hinge abduction on abduction films (if not treatable with concomitant femoral osteotomy)
- Older age

While a definite degree of osteoarthrosis at which a PAO should not be performed has not been defined, it is well documented in the literature that with worsening Tonnis grades, the outcome of PAO surgery is poorer. In general, a Tonnis grade ≥ 2 is a relative contraindication for PAO.

There is no ceiling age restriction for PAO; however, it is rare for this surgery to be performed in >50-year-old age group. Unlike distant osteotomies, the PAO does cross the posterior aspect of the triradiate cartilage and, therefore, is not indicated in young patients with substantial growth remaining; experience shows no negative influence from age 10 years.

Competence of the Surgeon

Periacetabular osteotomy is a demanding but learnable procedure; correct execution depends on a multitude of details performed in the right order. Exact knowledge of the bony, vascular, and neural anatomy of the hip and pelvis is indispensable. Experience with acetabular and pelvic fractures is a good precondition. Cadaver dissections of the procedure are mandatory. The best way to learn the technique is at a center with proper mentorship. A case load of at least 20–25 PAO procedures per year maintains and increases experience.

Surgical Technique for Periacetabular Osteotomy

Performing a periacetabular osteotomy should be considered as a stepwise series of events that allow progressive mobilization of the acetabulum. The steps can be considered as preoperative, intraoperative, and postoperative.

Preoperative requisites are a radiolucent operating table, operating instruments including a combination of curved and straight osteotomes, a selection of curved blunt and pointed Homan retractors, 30° angled notched osteotome, sterile leg holder or equivalent, Schanz pins, toothed lamina spreaders, 2.5 mm threaded tip Kirschner wires, 3.5 mm screws ranging from 40 to 120 mm in length and appropriate drill, oscillating power saw and blades, bone wax, and an image intensifier.

The anesthetized patient is positioned on a radiolucent operating table. A crucifix position will allow enough room for surgeon and operative assistants. Sterile preparation is in a hindquarter fashion extending to the umbilicus including the buttock. Access is required from the anterior ½ of the iliac crest down to mid thigh. The thigh and leg are free draped to allow mobilization of the lower limb for access.

The dissection is performed in a modified Smith–Petersen approach. The skin incision follows the contour of the iliac crest starting at the gluteal tubercle extending down to the anterior superior iliac spine (ASIS) curving lateral to this structure by 1–2 cm. The incision then follows a curvilinear course distally down the thigh over the anterolateral surface. Full-thickness subcutaneous flaps are made laterally. The sartorius has its origin from the anterior aspect of the ASIS and the tensor fasciae latae (TFL) muscle from the lateral surface of the anterior aspect of the iliac wing. A seam of fatty tissue lies between these two muscles in which runs the lateral femoral cutaneous nerve. By having the fascial incision over the belly of TFL laterally biased by 1.5–2 cm from the ASIS, this should be protected. By lifting up the medial cut edge of the fascia, dissection of the TFL muscle fibers off the fascia both superiorly and medially can be performed. The fibers freely part from their fascial attachment, and with lateral retraction with a handheld retractor, the interval is expanded. With combined medial retraction of the sartorius muscles fibers and slight abduction of the leg, the floor of the TFL muscle compartment is visualized. The floor is incised to expose the muscle and tendon of the direct head of the rectus femoris muscle. The floor is a fascial layer of varied thickness; once incised, the rectus will lie medially, and laterally fibrofatty tissue will be located in which the ascending branch of the lateral circumflex artery will be found. This vessel runs transversely in the interval and should be protected for its blood supply to the TFL muscle. With the distal dissection performed, attention is turned to the proximal segment.

The overhanging muscle fibers of the external oblique muscle are detached in a subperiosteal fashion from the iliac crest down to within 2 cm of the ASIS. A block of bone is then osteotomized from the ASIS 1.5–2 cm proximal to the palpable anterior prominence. This fragment with sartorius and ilioinguinal ligament is mobilized medially and protects the lateral cutaneous femoral nerve. To facilitate refixation of the osteotomy, predrilling can be performed. With the osteotomy of the ASIS performed, the inner aspect of the iliac wing can be exposed. Subperiosteal elevation of the iliacus muscle is performed from mid-ilium working distally toward the previously exposed interval between the sartorius and TFL. Staying subperiosteal in this dissection protects the iliacus muscle and intrapelvic structures. The proximal extent of this dissection should be up to, but not into, the greater sciatic foramen. During elevation of the periosteum, it is possible to encounter the nutrient vessel branch of the iliolumbar artery. In 50 % of patients, it will enter lateral to the pelvic brim [37], and when encountered, considerable backflow of blood can occur. This is often best controlled by applying bone wax to the nutrient foramina. In the other 50 % of cases, the vessel enters the ilium medial to the pelvic brim and its intraosseous course can be cut during the supra-acetabular osteotomy resulting in substantial blood loss. This cannot be accessed until full mobilization of the acetabular fragment when bone wax can be applied.

Distal extension of the elevation should be performed, but taking care to maintain the periosteal and muscular attachments medially on the ASIS. Distal to the ASIS osteotomy site the anterior inferior iliac spine (AIIS) will be located. With medial retraction of the sartorius, the underlying rectus is again visualized, and its direct head attachment to the AIIS is identified. The direct and reflected head of the rectus tendon are tenotomized. With distal medial retraction of the rectus femoris, the final structure before the capsule is encountered. The iliocapsularis muscle is the distal femoral expansion of the iliacus muscle and lies directly over the capsule. It can be hypertrophied in the dysplastic hip. It must be elevated from the capsule to allow access to the iliopectineal bursa. The elevation is performed sharply, working in a lateral to medial direction, in doing so, detaching its origin from the distal margin of the AIIS. Once the iliopectineal bursa is encountered, it is opened and the psoas major tendon is identified. This structure is a useful boundary marker and protector of the medially located femoral neurovascular bundle. It can be retracted by placing a pointed Hohmann under it and hammering the Hohmann into the superior pubic ramus 1–1.5 cm medial to the iliopectineal eminence. The dissection of the rectus and iliocapsularis should be done with the hip in approximately 40° of flexion; this relaxes the hip flexor musculature and allows easier retraction. Placing the thigh in a sterile leg holder maintains the hip flexion and is a useful surgical maneuver.

With the iliocapsularis completely mobilized, the inferior border of the capsule will be exposed. This outlines the inferior aspect of the femoral head and calcar region of the femoral neck. If an inadvertent puncture of the capsule occurs, closure should be performed as a closed capsule placed under tension facilitates the next step, dissection of the ischial ramus. The interval to gain access to the ischial ramus is between the inferior margin of the hip joint capsule and the obturator externus muscle. The orientation of the muscle fibers of the obturator externus lie in a transverse fashion, and the surgeon should stay proximal to the muscle belly as the medial circumflex artery runs distal to its inferior margin. By dissecting over the tough fibrous edge of the capsule, a plane between the two structures can be made. A Cobb elevator can be used to find this plane. Once opened, a large curved and smooth tipped pair of dissecting scissors can be introduced (Fig. 1). The scissors palpate the ischium in the area adjacent to the infracotyloid notch. With gentle probing, the width of the ramus can be estimated by entering the obturator foramen followed by the lateral aspect of the ramus. Laterally, the ischiocrural muscle origins lie as a protective layer between the osteotome and sciatic nerve, especially when the leg hip is flexed. The obturator externus is retracted distally (usually with a Cobb or wide periosteal elevator), and a 15 mm curved osteotome is introduced. Again, palpation onto the ramus is performed, and attention should be made to get the osteotome resting as close to the infracotyloid notch as possible (Fig. 2). Soft tissue tension can cause the osteotome to slip inferiorly down the ramus making the cut too distal, which can compromise correction. At this stage, an image intensifier can be used to check that the initial cut is not too distal. Starting with the handle of the osteotome aiming first posteriorly with a rotation of the blade toward the contralateral shoulder, the initial cut is made driving the osteotome to a depth of approximately 20–25 mm. As the osteotome is advanced, the handle should be turned downward to aim in

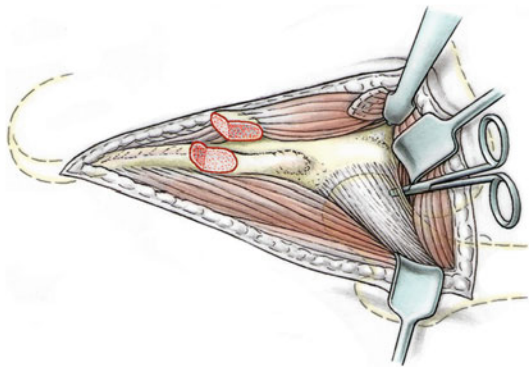


Fig. 1 Schematic of a right hip after the approach. A pointed Hohmann retractor sits on the superior pubic ramus, medial to the iliopectineal eminence. The blunt scissors follow the capsule posteromedially to palpate the obturator foramen, ischium, and infracotyloid notch

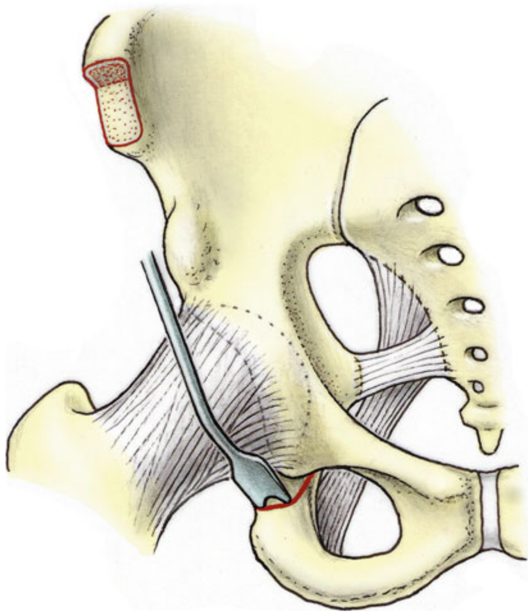


Fig. 2 A narrow curved double tipped osteotome is introduced into the space opened by the scissors. The ischial osteotomy is performed stepwise, leaving the lateral cortex intact (Reprinted with permission from SLACK Incorporated: RANAWAT, Anil (MD). KELLY, Bryan T. (MD). *Musculoskeletal Examination of the Hip and Knee: Making the Complex Simple*. Thorofare, NJ; Slack Incorporated: 2011)

a posterosuperior direction. The aim is to completely cut the medial surface of the ramus while scoring the lateral surface; in doing so, the sciatic nerve is protected. While careful notching of the lateral surface, the hip is slightly extended and abducted and the knee is flexed, a move which will relax the soft tissues including the sciatic nerve, drawing them away from the ramus. A particular technique during the ischial ramus cut is to advance the osteotome and then withdraw the instrument using a side-to-side "wiggling" motion. This allows palpation of the medial and lateral walls of the ramus, and the surgeon can repeat cuts if resistance is felt. Under no circumstances should repeated cuts be made especially laterally once there is a loss of resistance, risk of sciatic nerve damage is possible. It is also important to realize this first cut is incomplete, and the ischial ramus is only partially osteotomized at this stage of the operation; however, a sufficiently deep-enough cut is critical for complete mobilization of the acetabular fragment, and a deep-enough osteotomy must be ensured.

The second osteotomy is made in the superior pubic ramus with the location of the cut being just medial to the iliopectineal eminence. Subperiosteal elevation is performed, and blunt Hohmann retractors are placed superiorly and inferiorly for protection of the obturator nerve and vessels lying just on the other side of the pubic bone. A pointed Hohmann retractor can be placed further medial into the ramus to allow at least 1 cm bone substance between the planned osteotomy and the hole of the impacted retractor. This will help to avoid fissuring of this bone bridge, losing purchase of the retractor before the osteotomy is completed. A 15 mm Lexer osteotome is then used to osteotomize the ramus with a cut that runs perpendicular to the long axis of the ramus but is inclined 45° medially. With enlarging the cut to 2 mm in the cis cortex, fissuring of the medial part of the pubis is further prevented. Before advancing the osteotome, the position of the retractors at the opposite side should be checked for optimal protection; ideal is to cut against the retractors. The second osteotomy is complete when the levering osteotome creates a visible gap.

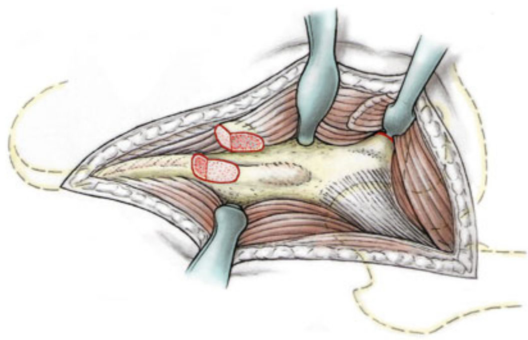


Fig. 3 Schematic after completion of the approach. A reverse Hohmann retractor sits medially on the ischial spine, retracting the iliacus and obturator internus muscle medially, giving view to the quadrilateral space. A second reverse Hohmann retractor is introduced from the outside into the sciatic foramen to protect the sciatic nerve

The remaining three osteotomies are performed through the supra-acetabular region and posterior column. To perform these cuts, further dissection of the quadrilateral surface, and tunnelling of the lateral side of the iliac wing, is required. The periosteum medial to the iliopectineal eminence is opened, and elevation of the periosteum and soft tissue should be performed. This occurs in a medial and inferior fashion over the quadrilateral plate, and the use of curved elevators facilitates this preparation. One key point is to limit the elevation back only as far as the greater sciatic notch but not into this area, preserving the periosteum prevents slippage of instruments into the foramen. This increases the stability of the reversed Hohmann retractor, which is now placed on the ischial spine. In cases where the PAO is being made for retroversion, the surgeon may find that the access to the quadrilateral plate is easier due to the orientation of acetabulum and spine. Preparation of the lateral aspect of the ilium should be limited to a space large enough to place a second reversed Hohmann retractor; the tip is placed into the greater sciatic notch (Fig. 3). The gluteus minimus muscle fibers are sharply elevated off the external iliac wing creating a tunnel for the Hohmann to be placed back as far as the greater sciatic notch to protect the sciatic nerve. An important part of the dissection is to elevate a tunnel 3 cm wide limiting the distal dissection leaving the distal 3 cm untouched. In

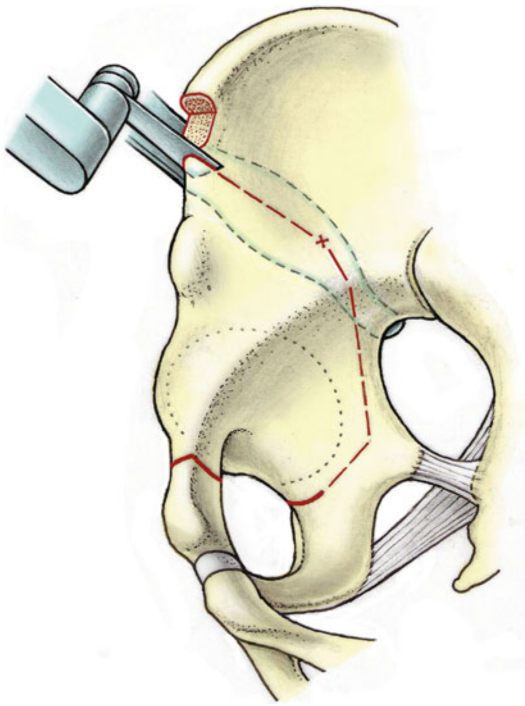


Fig. 4 Supra-acetabular osteotomy with the oscillating saw, starting just distal to the osteotomy of the ASIS. It stops about 1 cm from the pelvic brim

doing so, the remaining attachment of tensor fasciae latae is preserved, and the distal origin of gluteus minimus on the acetabular fragment remains undisturbed. The value of limiting the distal/inferior dissection is that the supra-acetabular branch of the superior gluteal artery runs within this muscle and will be an important supply to the acetabular fragment once mobilized.

The third and fourth supra-acetabular and retroacetabular osteotomies proceed sequentially with the supra-acetabular cut being made starting just inferior to the ASIS osteotomy site (Fig. 4). The direction of the cut is made perpendicular to the recumbent patient, heading posteriorly toward the pelvic brim. This osteotomy should stop 1.5 cm anterior to the pelvic brim. To facilitate the cut, prescoring of the line of the osteotomy on the medial aspect of the ilium should be made with a straight Simal osteotome prior to the formal osteotomy. At the end of the planned supra-acetabular osteotomy, the direction changes to begin the posterior osteotomy. The direction changes by approximately 110–120° as the posterior cut is made inferiorly along the posterior column. This angle and apex can vary as per patient morphology, but always the cut is made at a distance of approximately 1.5 cm anterior to the posterior margin of the posterior column. Both osteotomies are performed with a reversed Hohmann retractor placed within the lateral "tunnel" heading posteriorly back to include the greater sciatic foramen, protecting the lateral soft tissues and sciatic nerve. The supra-acetabular cut is made with an oscillating bone saw following the planned score in the ilium. At this point, a curved Simal osteotome is used to create the beginning of the posterior cut at the angle of 110–120°; this part of the posterior cut should end at least 1.5 cm anterior to the greater sciatic foramen (Fig. 5). The osteotome is directed to cut the outer cortex at the same level. It is not completed before the next branch along the quadrilateral surface is osteotomized using a straight Simal osteotome heading along the posterior column, 1.5 cm anterior to the posterior margin and ischial spine (Fig. 6). This cut usually only needs to be between 30 and 40 mm and does not need to connect immediately with the first ischial ramus cut because this bone will soon be fractured in a controlled fashion. Only thereafter the cut produced by the curved osteotome is completed with separating the outer cortex.

The bone density and strength of the acetabulum and ischial spine means that keeping the posterior cut between these two structures allows the controlled fracture to pass predictably between these two structures. To perform the controlled fracture, a 15 mm Lexer osteotome is placed within the second part of the posterior osteotomy, and by pulling the handle of the instrument anteriorly, an anterior directed force is applied, fracturing the remaining posteroinferior bone bridge. If this maneuver is unsuccessful with moderate force, then the posterior osteotomy will need to be extended. Because the original ischial ramus osteotomy aims to completely cut the medial surface but only score the lateral aspect of the ramus, there is a retained bone bridge posteroinferiorly between ischial and posterior osteotomies. It is this bone that may require additional attention,

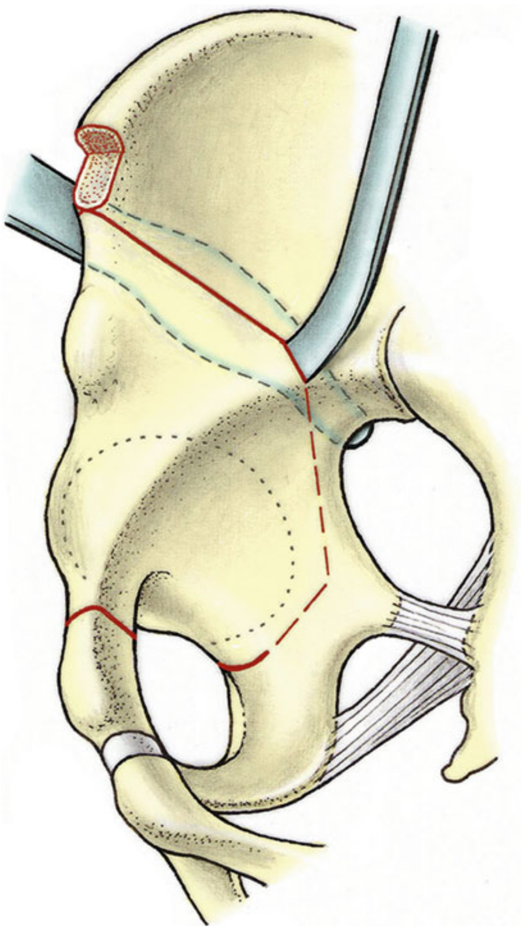


Fig. 5 A curved Simal osteotome is used to cut through the lateral cortex. The sciatic nerve is protected with the reversed Hohmann retractor

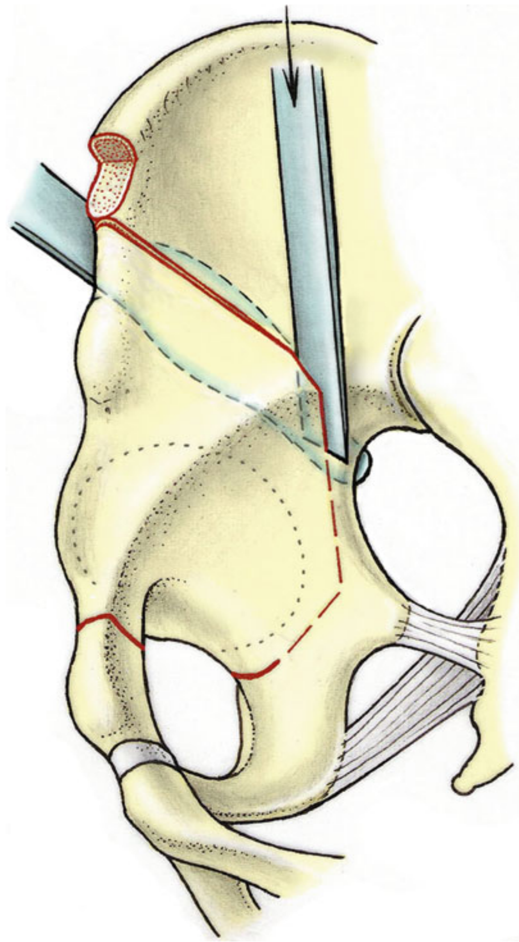


Fig. 6 Course of the straight Simal osteotome about 1.5 cm anterior to the posterior margin of the quadrilateral plate (Reprinted with permission from SLACK Incorporated: RANAWAT, Anil (MD). KELLY, Bryan T. (MD). *Musculoskeletal Examination of the Hip and Knee: Making the Complex Simple*. Thorofare, NJ; Slack Incorporated: 2011)

and completion of the osteotomy may require that this is cut with an osteotome.

To aid in this additional extension of the posterior cut, a Schanz screw is placed into the AIIS with a universal chuck connected serving as a handle. A large toothed laminar spreader is placed superiorly in the supra-acetabular osteotomy. With a pulling force on the handle of the Schanz screw and opening of the spreader, a distal–lateral moment is created, causing the acetabulum to rotate in that direction. This should make the quadrilateral surface more easily visible to allow the introduction of "special" angled osteotomes into the posterior osteotomy. This must be 4 cm below the pelvic brim, and the angle of the cut is made at 50° to complete the posterior cut through the retained posteroinferior bone bridge. During the completion of the posterior osteotomy, gentle hammer blows on the angled osteotome are made. Attention to loss of resistance is important as these cuts have to potential to injure the sciatic nerve if repeated blows through already completed bone cuts are made. As during the first ischial cut, decreasing the hip flexion together with increasing the knee flexion will relax the sciatic nerve. Signs of loss of bone resistance at the osteotome, less tension on the Schanz crew, and loosening of the laminar spreader indicate sufficient

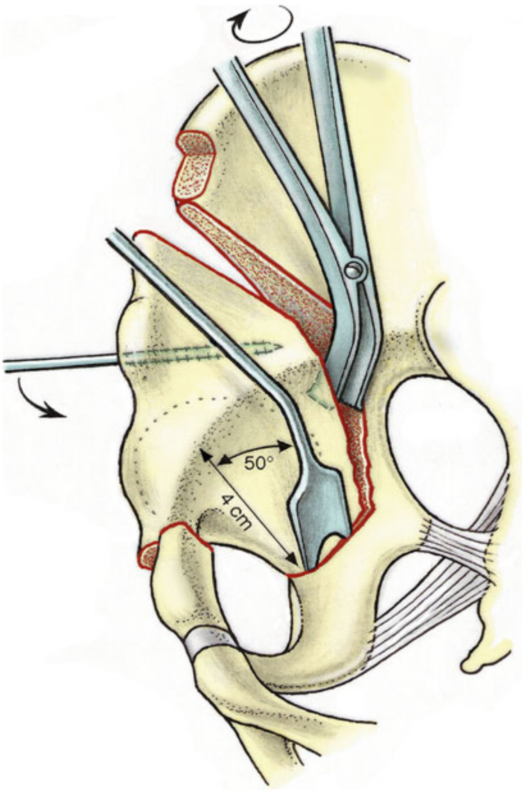


Fig. 7 Execution of the infra-acetabular osteotomy and maneuver to mobilize the acetabular fragment (Reprinted with permission from SLACK Incorporated: RANAWAT, Anil (MD). KELLY, Bryan T. (MD). *Musculoskeletal Examination of the Hip and Knee: Making the Complex Simple*. Thorofare, NJ; Slack Incorporated: 2011)

disengagement of the acetabular fragment. The osteotomy of the remaining posteroinferior bone bridge is completed with a controlled fracture by rotating the Schanz screw medially while externally rotating the laminar spreader (Fig. 7).

With the acetabular fragment free, correction can now be performed. Depending on the severity of dysplasia, correction of the deficiency is made by rotation of the acetabular fragment around the femoral head. This will work in the cases without substantial lateralization; however, if the femoral head does lie more laterally than usual, the joint and acetabular fragment should be medialized to correct for this. The most common form of deficiency in dysplasia is anterolateral loss of coverage which requires an anterior correction with internal rotation of the acetabular fragment. The correction is temporarily held with 2.5 mm threaded tipped K-wires from the ilium into the fragment. Preoperative assessment of the radiographs will help identify posterior deficiency (acetabular retroversion), which can occur in up to 15 % of dysplastic cases. The aim of the correction is to have an adequate acetabular roof angle (0–10°) with the posterior and anterior walls meeting at the lateral edge of the acetabulum, i.e., no crossover sign, and the femoral head sitting neutrally, not too medial as seen by the medial head being lateral to the ilioischial line. Inexperienced surgeons may be at risk of several errors. On attempting correction, the presence of a gap in the supra-acetabular region is generally considered a sign of "hinging" and an incomplete osteotomy/retained posteroinferior bone bridge that requires further completion (Fig. 8). In an attempt to get lateral coverage, the acetabular roof should never be placed at an angle past the horizontal (0°). Also in trying to achieve anterolateral coverage, there is a risk of retroverting the acetabulum and generating anterior impingement. It may require several redirection attempts until the ideal spatial orientation of the acetabulum is achieved. At this stage, a "T capsulotomy" is performed. Any prominence of the femoral head neck junction can be addressed concurrently as can labral surface tears, ganglions, or rim fragments requiring removal or refixation. Visualization of the joint during motion can be done, looking for impingement. Flexion past 90° and balanced internal and external rotation are ideal.

If the surgeon achieves the desired correction radiographically and on intraoperative assessment of the articulation, then definitive fixation is performed with three 3.5 mm screws. Two are directed from superiorly in the ilium into the acetabular fragment, with a third critical screw from the AIIS aimed at the sacroiliac joint, this screw gaining excellent purchase in the bone of the "sciatic buttress." The rotation of the fragment often leaves a prominent piece of bone anteriorly which should be trimmed and then placed within the supra-acetabular defect as bone graft. The capsulotomy, if performed, is loosely closed with absorbable sutures, and the rectus femoris

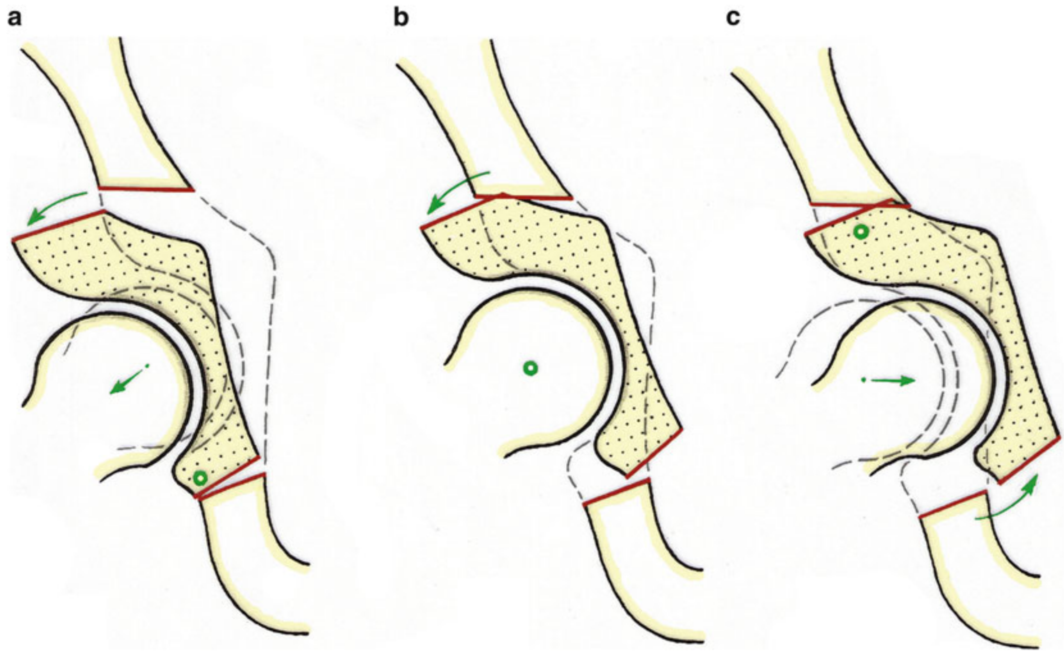


Fig. 8 (**a**) Inferior hinge (*green circle*) in the case of incomplete inferior osteotomy. The center of rotation is lateralized. (**b**) Rotation of the fragment around the femoral head center. The center of rotation remains anatomical. (**c**) Superior hinge leading to medialization of the fragment

direct head is reattached to the AIIS with transosseous nonabsorbable sutures. The ASIS osteotomy is fixed with a 2.7–3.5 mm screw with closure of the soft tissues in layers with superficial drains. Deep drains are avoided because at the completion of the osteotomy, large bare areas of cancellous bone are exposed, and deep drain placement could allow for the patient to lose an undesired amount of blood.

In the setting of an additional femoral osteotomy, it has to be decided before surgery whether this should be executed before or after the PAO. In the case of a high-riding trochanter, the femoral procedure has to be completed before; otherwise, the trochanter may limit the optimal acetabular correction. This is best performed in a lateral decubitus. However, the patient can be prepared and draped in such a way that changing the patient into supine position is possible without redraping. After trochanteric advancement and relative neck lengthening, the lateral approach can be used to perform the first ischial cut of the PAO under visual control of the sciatic nerve. Femoral neck or head osteotomies are also best performed before the PAO. Femoral derotation or intertrochanteric varus osteotomies can be performed before or after the PAO [39].

Postoperative Rehabilitation

Immediately post operation, the patient has their leg held in a foam splint in extension and neutral rotation. The patient has routine postoperative blood analysis and removal of drains on day 1–2 coinciding with dressing changes. The patient will mobilize either day one or two depending on comfort. During mobilization, crutches are used to allow touch-weight bearing for a total of 6 weeks. During the first 6 weeks of rehabilitation, active hip flexion with the knee extended is discouraged; this allows time for the disrupted rectus femoris to heal. At the 6-week follow-up, a radiograph is taken to assess bone healing; if adequate, then increased weight bearing and abductor strengthening can commence. Additional follow-ups are scheduled as needed.

Results of Periacetabular Osteotomy

Results and Outcomes for Dysplasia

Long-Term Results

Periacetabular osteotomy as a treatment of hip dysplasia originated in the mid-1980s [36]. Coined the “Bernese osteotomy” after the city it was established in, the senior author and colleagues reported on the initial results of PAO. In this paper headed by Siebenrock et al. [40], 75 dysplastic hips in 63 patients treated with PAO are reported on. This case series includes the first group of patients operated on from between 1984 and 1987. The average age of the patients was 29 years (13–56 years), with majority being female (3.4:1 F:M). There was a high rate of previous surgery for dysplasia with 31 % of patients having had prior surgery (14 hips had acetabular surgery, 9 hips femoral surgery). In addition, there were also high rates of moderate to severe dysplasia with 50 % Severin grade III and 44 % Severin grade IV; it was also deemed that 51 % of patients showed signs of preoperative osteoarthrosis. Average follow-up was achieved at 11.3 years (10–13.8 years) in 71 hips (95 %) of which at the last time of follow-up 82 % had retained hips of which 73 % were functioning with good to excellent results. Subgroup analysis showed that patients with Tonnis grade of ≤ 1 had 88 % good to excellent results. It was identified that poorer outcomes were associated with increasing age, presence of degenerative joint disease at the time of surgery, and inadequate correction with a suboptimal acetabular index. Being the first group of patients in a learning curve, major complications were reported in 18 patients including intra-articular cut (2), loss of position (2), and femoral head subluxation (3).

The same group of patients were reevaluated at an average of 20 years (19–23 years) by Steppacher et al. [41], with 68 hips in 58 patients available for assessment. Of the 68 hips, 41 (60 %) survived leaving 27 (40 %) that had required conversion to total hip arthroplasty (26) and one arthrodesis. The Kaplan–Meier survivorship was 60.5 % at 20 years. Of those hips surviving to this point, the average Merle d’ Aubigne score was 15.6, and 81 % (33 hips) were graded as good to excellent, 15 % fair (6 hips), and 5 % poor (2 hips). Again, major prognostic indicators of achieving poor outcome were increasing age, lower preoperative Merle d’Aubigne scores, osteoarthrosis, limp, increased extrusion index, and positive anterior impingement sign.

Intermediate Results

Trousdale et al. [42] reported on 42 patients who had undergone PAO during a 6-year period in the mid- to late 1980s. The average age was 47 years, and the average follow-up was 4 years (2–8 years). Ten patients had concomitant femoral osteotomy, and the average preoperative Harris Hip score (HHS) was 62. There was a range of osteoarthritic change from Tonnis grade I (15 patients), grade II (18 patients), and grade III (9 patients). Of the 33 patients with grade I–II changes, 32 had a good or excellent outcome on HHS. However, eight of the 9 patients with grade III changes preoperatively only achieved an average score of 70 on the HHS, only eight points increased over preoperative scores. This study shows a direct correlation between preoperative degenerative joint disease, as measured by Tonnis grade, and achieving poorer postoperative hip function as measured by HHS. These patients eventually required further procedures including total hip arthroplasty or further femoral osteotomy.

Periacetabular Osteotomy for Acetabular Retroversion

Since its description in 1999 by Reynolds, acetabular retroversion has become a topic of increasing investigation. Not to be confused with anterior rim prominence in its isolation, acetabular retroversion involves the entire acetabulum being malrotated and, therefore, not amenable to anterior rim resection alone. To perform an isolated anterior rim resection in a true case of retroverted acetabulum has the potential to leave the patient with a posterior deficiency.

Siebenrock et al. [43] have reported on 29 hips in 22 patients that underwent PAO for retroverted but otherwise normal acetabula. The average age was 23 years (14–41) with 19 male patients and 10 female patients. Preoperative symptoms persisted for 17 months on average before surgery was performed, and preoperative hip function was documented with Merle d' Aubigne scores which averaged 14 points (range 12–16 points). The presence of anterior impingement signs and range of motion of the hip was documented, and preoperative radiographs were analyzed for crossover and posterior wall signs. At average follow-up of 30 months (range 24–49 months), the average Merle d' Aubigne score had significantly increased to 16.9 points. Range of motion significantly improved with internal rotation in flexion increasing on average from 10° to 20°. Anterior over-coverage decreased significantly by 8° from 36° to 28° as measured by anterior center-edge angle based on false profile view of Lequesne. Of the 26 patients that underwent capsulotomy, all had labral pathology present that was addressed. Three patients required further treatment, one for screw bending from early weight bearing, one patient had delayed onset posteroinferior impingement treated with posterior rim resection, and one patient had ongoing impingement due to insufficient correction and underwent revision surgery to correct. Twenty-five of the 29 patients had no crossover sign at follow-up, and 27 of the 29 no longer had a positive posterior wall sign. At last follow-up, no patient had any radiographic signs of osteoarthritis or osteonecrosis. In a recent publication, the role of acetabular positioning and correction of an aspheric head neck junction was investigated. It was shown that proper acetabular reorientation and the creation of a spherical femoral head neck junction improve long-term survivorship and decelerate OA progression in patients with developmental hip dysplasia [44].

Complications

As with any surgical procedure, there are risks of infection, blood loss requiring transfusion, and thromboembolic events. However, there are certain specific intraoperative and postoperative complications that are known risks during or after PAO [45]. Overall, substantial complications account for about 4 % [46, 47].

Intraoperative complications comprise of soft tissue injuries to the neurological and vascular structures. Injury to the nutrient vessel branch of the iliolumbar artery has been mentioned and is source of excessive bleeding. The use of bone wax is useful to tamponade this vessel when intraosseous bleeding is brisk. Injury to the medial circumflex femoral artery is uncommon and avoided by dissecting the plane between the capsule and obturator externus staying close to the capsular side of the plane. Injury to the femoral vessels was so far not a complication of PAO using the Smith–Petersen approach. Neurological injury can occur in four nerves: lateral femoral cutaneous nerve, femoral nerve, obturator nerve, or sciatic nerve [48]. While the lateral femoral cutaneous nerve (LFCN) is the most commonly injured nerve, morbidity in the proper sense has not been reported; the frequency can be limited by opening the fascia overlying the TFL longitudinally over the muscle belly proper rather than in the fatty plane between the TFL and the sartorius. In doing so, the dissection is lateral to the LFCN, and injury to the nerve is less likely. Because of the associated morbidity, sciatic nerve injury poses the most concern for the surgeon, but in the senior author's series of more than 1,000 PAO's, only seven had permanent loss of sciatic nerve function. Femoral nerve injury has been observed after the acetabular fragment has been medialized excessively as necessary in posttraumatic dysplasia.

Bone injuries can also occur with unplanned fracture of the posterior column or inadvertent entering the hip joint during any of the osteotomies. Overcorrection and undercorrection are possible, but intraoperative imaging and attention to detail make these less likely.

Postoperative complications are related to the loss of reattachment of the rectus femoris muscle and hip flexors. This can create significant pain and weakness, and it is generally considered appropriate to perform refixation if this occurs. Heterotopic ossification is also a potential risk due to large areas of exposed bone and hematoma formation in the periarticular tissues. If this occurs near the AIIS, then it is possible for extra-articular bone impingement, requiring resection, to occur.

Delayed bone complications include loss of fixation of the correction and delayed or nonunion of the osteotomies/column fractures if present. Loss of fixation is negated by using three screws for fixation with the essential screw being the AIIS to the ilium screw creating enhanced stability, making loss of correction a rare event. If loss of correction does occur (usually due to excessive weight bearing at a too early stage) then re-correction should be taken into consideration at the earliest possible stage. Additional fixation with pelvic reconstruction plating superiorly should be considered. Nonunion of the pubic ramus can occur, with this usually happening in the setting of large corrections. As with the pubis, the posterior column, if fractured, can occasionally go on to nonunion and if symptomatic may require posterior column plating and bone grafting.

Implant-related irritation from the iliac crest is possible, and removal of the offending screw will resolve this issue, although routine removal is not necessary.

Finally, with the achieved correction, the patient may notice a slight global decrease in range of motion; this may not be considered a complication and more a flow on effect of creating a more normal joint orientation.

Summary

The PAO is an extremely powerful technique for reorientation of the acetabulum, suited for the treatment of hip dysplasia as well as acetabular retroversion. The technique, however, is demanding and requires a high understanding of muscular, bony, vascular, and neural anatomy. In the hands of the experienced surgeon, excellent long-term results can be achieved.

References

1. Harris W. Etiology of osteoarthritis of the hip. Clin Orthop Relat Res. 1986;213:20–33.
2. Stulberg S, Harris W. Acetabular dysplasia and the development of osteoarthritis of the hip. In: Harris, WH (ed). The hip, proceedings of the second open scientific meeting of the hip society. St. Louis: C.V. Mosby; 1974. p. 83.
3. Clohisy J, Nunley R, Carlisle J, Schoenecker P. Incidence and characteristics of femoral deformities in the dysplastic hip. Clin Orthop Relat Res. 2009;467:128–34.
4. Kummer B. [Biomechanical aspects of hip dislocation] [in German]. Orthopade. 1988;17:452–62.
5. Pauwels F. [Biomechanics of the abnormal and diseased hip. Theoretical foundation, technique and results of treatment: an atlas][in German]. Berlin: Springer; 1976.
6. Wiberg G. Studies on dysplastic acetabula and congenital subluxation of the hip joint. Acta Chir Scand. 1939; (Suppl.) 83:58.
7. Cooperman D, Wallensten R, Stulberg SD. Acetabular dysplasia in the adult. Clin Orthop Relat Res. 1983;175:79–85.
8. Preiser G. [Arthritis of the hip and its relation with abnormal version of the acetabulum][in German]. Dtsch Zeitschr Chirurg. 1907;89:541.
9. Teinturier P. [Abnormal orientation of the femur neck and acetabulum. Statistical study of 200 centered arthrosic hips][in French]. Rev Chir Orthop Repar Appar Mot. 1970;56:219–30.
10. Reynolds D, Lucas J, Klaue K. Retroversion of the acetabulum. A cause of hip pain. J Bone Joint Surg. 1999;81-B:281–8.
11. Ganz R, Parvizi J, Beck M, Leunig M, Notzli H, Siebenrock KA. Femoroacetabular impingement: a cause for osteoarthritis of the hip. Clin Orthop Relat Res. 2003;417:112–20.
12. Ganz R, Leunig M, Leunig-Ganz K, Harris WH. The etiology of osteoarthritis of the hip: an integrated mechanical concept. Clin Orthop Relat Res. 2008;466:264–72.
13. Ezoe M, Naito M, Inoue T. The prevalence of acetabular retroversion among various disorders of the hip. J Bone Joint Surg. 2006;88-A:372–9.
14. Kim W, Hutchinson C, Andrew J, Allen P. The relationship between acetabular retroversion and osteoarthritis of the hip. J Bone Joint Surg. 2006;88-B:727–9.
15. Li PL, Ganz R. Morphologic features of congenital acetabular dysplasia: one in six is retroverted. Clin Orthop Relat Res. 2003;416:245–53.
16. Mast JW, Brunner RL, Zebrack J. Recognizing acetabular version in the radiographic presentation of hip dysplasia. Clin Orthop Relat Res. 2004;418:48–53.
17. Klaue K, Durnin C, Ganz R. The acetabular rim syndrome. J Bone Joint Surg. 1991;73-B:423–9.
18. Kohnlein W, Ganz R, Impellizzeri FM, Leunig M. Acetabular morphology: implications for joint-preserving surgery. Clin Orthop Relat Res. 2009;467: 682–91.
19. Corten K, Ganz R, Chosa E, Leunig M. Bone apposition of the acetabular rim in deep hips: a distinct finding

of global pincer impingement. J Bone Joint Surg Am. 2011;93 Suppl 2:10–6.
20. Ferguson SJ, Bryant JT, Ganz R, Ito K. An in vitro investigation of the acetabular labral seal in hip joint mechanics. J Biomech. 2003;36:171–8.
21. Pemberton PA. Pericapsular osteotomy of the ilium for the treatment of congenitally dislocated hips. Clin Orthop Relat Res. 1974;98:41–54.
22. Dega W. [Anatomical and functional restitution in congenital hip dislocation by one stage surgical procedure] [in German]. Arch Orthop Unfallchir. 1966;60:16–29.
23. Chiari K. Medial displacement osteotomy of the pelvis. Clin Orthop Relat Res. 1974;98:55–71.
24. Spitzy H. Artificial acetabular roof: osseous bolts for temporary fixation][in German]. Z Orthop Chir. 1923;43:284–94.
25. Ganz R, Slongo T, Siebenrock KA, Turchetto L, Leunig M. Surgical technique: the capsular arthroplasty: a useful but abandoned procedure for young patients with developmental dysplasia of the hip. Clin Orthop Relat Res. 2012;470:2957–67.
26. Salter RB. Innominate osteotomy in the treatment of congenital dislocation and subluxation of the hip. J Bone Joint Surg. 1961;43-B:518–39.
27. LeCoeur P. [Acetabular reorientation with a triple osteotomy of the iliac isthmus][in French]. Rev Chir Orthop Repar Appar Mot. 1965;51:211–2.
28. Hopf A. [Hip acetabular displacement by double pelvic osteotomy in the treatment of hip joint dysplasia and subluxation in young people and adults][in German]. Z Orthop Grenzgeb. 1966;101:559–86.
29. Steel HH. Triple osteotomy of the innominate bone. J Bone Joint Surg Am. 1973;55:343–50.
30. Sutherland DH, Greenfield R. Double innominate osteotomy. J Bone Joint Surg Am. 1977;59:1082–109.
31. Tönnis D, Behrens K, Tscharani F. A modified technique of the triple pelvic osteotomy: early results. J Pediatr Orthop. 1981;1:241–9.
32. Carlioz H, Khouri N, Hulin P. [Triple juxtacotyloid osteotomy][in French]. Rev Chir Orthop Repar Appar Mot. 1982;68:497–501.
33. Eppright RH. Dial osteotomy of the acetabulum in the treatment of dysplasia of the hip. Abstract. J Bone Joint Surg Am. 1975;57:1172.
34. Wagner H. Osteotomies for congenital hip dislocation. In: The hip. Proceedings of the fourth open scientific meeting of the hip society. St Louis: Mosby, C.V.; 1976. p. 45–66.
35. Ninomiya S, Tagawa H. Rotational acetabular osteotomy for the dysplastic hip. J Bone Joint Surg Am. 1984;66:430–6.
36. Ganz R, Klaue K, Vinh TS, Mast JW. A new periacetabular osteotomy for the treatment of hip dysplasias. Technique and preliminary results. Clin Orthop Relat Res. 1988;232:26–36.
37. Beck M, Leunig M, Ellis T, Sledge JB, Ganz R. The acetabular blood supply: implications for periacetabular osteotomies. Surg Radiol Anat. 2003;25:361–7.
38. Lequesne M, Djian A. New radiographic angles for the study of the hip. The "false profile" of the pelvis. Vie Med. 1961;42:1629–41.
39. Ganz R, Horowitz K, Leunig M. Algorithm for femoral and periacetabular osteotomies in complex hip deformities. Clin Orthop Relat Res. 2010;468:3168–80.
40. Siebenrock KA, Schöll E, Lottenbach M, Ganz R. Bernese periacetabular osteotomy. Clin Orthop Relat Res. 1999;363:9–20.
41. Steppbacher SD, Tannast M, Ganz R, Siebenrock KA. Mean 20-year follow-up of Bernese periacetabular osteotomy. Clin Orthop Relat Res. 2008;466:1633–44.
42. Trousdale RT, Ekkernkamp A, Ganz R, Wallrichs SL. Periacetabular and intertrochanteric osteotomy for the treatment of osteoarthrosis in dysplastic hips. J Bone Joint Surg. 1995;77-A:73–85.
43. Siebenrock KA, Schoeniger RA, Ganz R. Anterior femoro-acetabular impingement due to acetabular retroversion treatment with periacetabular osteotomy. J Bone Joint Surg. 2003;85-A:278–86.
44. Albers CE, Steppacher SD, Ganz R, Tannast M, Siebenrock KA. Impingement adversely affects 10-year survivorship after periacetabular osteotomy for DDH. Clin Orthop Relat Res. 2013;471:1602–14.
45. Davey JP, Santore RF. Complications of periacetabular osteotomy. Clin Orthop Relat Res. 1999;363:33–7.
46. Clohisy JC, Schutz AL, St John L, Schoenecker PL, Wright RW. Periacetabular osteotomy: a systematic literature review. Clin Orthop Relat Res. 2009;467(8):2041–52.
47. Hussell JG, Rodriguez JA, Ganz R. Technical complications of the Bernese periacetabular osteotomy. Clin Orthop Relat Res. 1999;363:81–92.
48. Sierra RJ, Beaule P, Zaltz I, Millis MB, Clohisy JC, Trousdale RT, ANCHOR group. Prevention of nerve injury after periacetabular osteotomy. Clin Orthop Relat Res. 2012;470:2209–19.

Acetabular Retroversion

46

Benjamin G. Domb, Jonathan N. Watson, and Asheesh Gupta

Contents

B.G. Domb (✉)
Hinsdale Orthopaedics Associates, Hinsdale, IL, USA

The American Hip Institute, Hinsdale, IL, USA

Loyola University, Stritch School of Medicine, Maywood, IL, USA
e-mail: benjamindombmd@gmail.com

J.N. Watson
Department of Orthopaedic Surgery, University of Illinois, Chicago, IL, USA
e-mail: jonwatsonmd@gmail.com

A. Gupta
Hinsdale Orthopaedics Associates, Hinsdale, IL, USA

The American Hip Institute, Hinsdale, IL, USA
e-mail: asheeshg26@gmail.com

Abstract

Acetabular retroversion is a complex deformity that can present in a variety of clinical settings. While it may be associated with posterior wall undercoverage, a detailed examination must be performed to elucidate the underlying pathomechanics of hip. Common findings on diagnostic imaging include an increased anterior center edge angle, positive posterior wall sign, positive crossover sign, a positive ischial spine sign, and herniation pits in the femoral neck. Acetabular retroversion can be associated with anterosuperior pincer-type impingement, posterolateral instability, labral tears, chondral damage, and posterior undercoverage. Treatment options include nonoperative therapy, open surgical dislocation, reverse periacetabular osteotomy (PAO), and hip arthroscopy. Early treatment and correct diagnosis are essential in treating this increasingly recognized phenomenon. The goals of treatment are to correct or address over/undercoverage and labral treatment and to address pathology of the bone and soft tissues.

Introduction

Acetabular retroversion was first recognized as a cause of hip pain in 1999 [1]. Acetabular retroversion is a descriptive term used to describe the version of the acetabular in relation to the sagittal

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_50

plane. There are varying degrees of retroversion which can be a normal variant or pathologic. It is important to note that acetabular retroversion does not imply anterior overcoverage. There can be normal anterior coverage with posterior undercoverage. Hip dysplasia can present with retroversion, resulting in posterior undercoverage without anterior overcoverage. This may result in posterolateral instability or posterior overload. Conversely, coxa profunda where the acetabular fossa is medial to the ilioischial line can present with anterosuperior overcoverage without posterior undercoverage in the setting of acetabular retroversion. Treatment options range from nonoperative measures including therapy such as rest and pain medication to operative intervention including reverse periacetabular osteotomy (PAO), surgical hip dislocation, and hip arthroscopy to correct associated bony and soft tissue pathology.

Epidemiology

Acetabular retroversion can be found in isolation as well as in association with other conditions. Wenger et al. studied 31 patients with acetabular labral tears and found 87 % had at least one structural abnormality and 35 % had more than one abnormality [2]. Of these, ten patients had a retroverted acetabulum. In 2003 Giori et al. studied the relationship between osteoarthritis and acetabular retroversion and found 20 % of the patients with apparently "idiopathic" osteoarthritis had retroversion [3]. Ezoe et al. also found a 20 % prevalence of acetabular retroversion in hips with osteoarthritis, as well as 18 % in those with dysplasia and 42 % in those with LCP disease, all of which were significantly increased from normal hips [4]. Examining a smaller subset of their results, they found a significantly higher incidence of retroversion in LCP hips with a nonspherical femoral head; however, it is unclear whether or not the acetabular deformity is a cause or an effect of the femoral head deformity. In addition, there was no significant association between retroversion and osteonecrosis of the femoral head. Fujii et al. examined the version of hips in patients with dysplasia and correlated it with their level of pain [5]. They found a significant increase in positive crossover and posterior wall signs in the dysplastic hips as well as a significant decrease in age at onset of hip pain in those hips with retroversion. There has also been shown an association between SCFE and acetabular retroversion. Sankar et al. found an increased prevalence of acetabular overcoverage in the contralateral (normal) hip in patients with a previously treated SCFE [6]. In a separate study, they measured acetabular version and compared normal hips with those with dysplasia and Down syndrome [7]. They found significantly more retroversion in the hips of patients with Down syndrome than either dysplasia or normal controls. However, this study only examined hips in patients with instability, so it is unclear whether or not the posterior instability is caused by retroversion or vice versa.

Few studies have examined the prevalence of acetabular retroversion among different gender and racial populations in asymptomatic individuals. Werner et al. examined the radiographs of 1,325 individuals presenting at a trauma center and found a crossover sign indicating retroversion in 52 % of men and 48 % of women, which was significant [8]. There was no significant difference between ethnic groups. A similar study was performed by Tannenbaum et al.; however, their study was performed on cadaver pelves [9]. They also showed an increased prevalence of retroversion in male specimens with no difference between ethnicities.

Pathogenesis

Etiology of Retroversion

Acetabular retroversion can also occur in dysplastic hips secondary to a developmental deformity, such as a hypoplastic posterior wall, prominence of the anterior wall, or a rotational abnormality of the acetabulum [5]. There is an increased prevalence of AR with LCP disease; however, it is unclear whether the presence of AR is secondary to or a result of deformation of the femoral head. Acetabular retroversion can also be caused iatrogenically.

While over-resection of the posterior wall can lead to relative posterior undercoverage and potential posterior instability, the most common cause of iatrogenic retroversion is from corrective osteotomy [10, 11]. Pelvic osteotomies are traditionally used to treat dysplastic hips and can aggravate any posterior deficiency if preoperative imaging is not studied. Certain types of dysplasia do not involve deficient anterior coverage, so if an osteotomy that provides additional anterior coverage, such as a Salter osteotomy, is used, it can worsen the overcoverage.

Pathology of Retroversion

As mentioned previously, Reynolds postulated AR as a cause of hip pain in 1999 [1]. The authors felt that with this anatomic variant the edge of the anterior wall and the anterosuperior roof of the acetabulum were vulnerable to impingement. In 2003 Ganz et al. proposed FAI as a cause for osteoarthritis in a younger patient population [12]. They thought the impingement led to chondral and labral damage, which in turn led to accelerated appearance of osteoarthritis. The pincer subtype of FAI is a result of linear contact of the femoral head–neck junction and the acetabular rim. Persistent impaction of the labrum leads to degeneration, cystic changes, and ossification of the rim, which can lead to deepening of the acetabulum and worsening overcoverage. Contrecoup lesions can also occur in the posteroinferior acetabulum secondary to chronic leverage of the head in the acetabulum. This combination of labral degeneration and contrecoup chondral damage likely leads to progression of osteoarthritis. Henak et al. compared the cartilage contact mechanics between normal and subjects with acetabular retroversion [13]. They found a more localized contact force superiorly and medially in the retroverted hips compared to a wide distribution for the control group. In dysplastic acetabuli, there is static overload of the articular cartilage, and in the setting of acetabular retroversion, there is dynamic impingement between the prominent anterosuperior aspect of the acetabulum and the femoral head–neck junction.

Presentation

A comprehensive history and physical examination are essential when evaluating a patient presenting with hip or groin pain [14]. Upon presentation, it is important to characterize the onset, location, quality, any radiation of the pain, any associated symptoms, and any aggravating or alleviating factors related to the complaint. A true, intra-articular source of hip pain is localized to the groin, while buttock or thigh pain is more often associated with extra-articular hip or lumbar spine pathology. After the chief complaint and history of present illness are determined, past medical and surgical history, social history, and any relevant family history should be reviewed. Prior hip surgery or bracing can point towards a diagnosis of DDH. Alcohol abuse or corticosteroid use is typical of osteonecrosis. Mechanical symptoms can denote a loose body or labral tear. Multiple joint involvement, stiffness, or recurrent swelling can occur in the setting of an inflammatory disorder.

A thorough physical exam is essential when evaluating a patient with hip pain. Similar to other areas, it is useful to group your exam maneuvers into different areas, such as standing, seated, supine, lateral, and prone. It is helpful to obtain vital signs, especially temperature, prior to the start of an exam that could alert the examiner about a febrile or infectious cause of pain. The standing exam should begin with examination of gait. The foot progression angle can denote any rotational abnormalities. The stance and swing phases of gait can also be examined. A decrease in stance phase, or antalgic gait, can signify a variety of pathology, including trauma, leg length discrepancy, or neuromuscular deficit. A Trendelenburg gait, or abductor lurch, can indicate abductor weakness. During the stance phase, the weakened hip causes the pelvis to drop to the opposite side, which in turn causes the trunk to lean back towards the affected side to compensate. A pelvic rotational wink, or excess rotation towards the affected hip causing lumbar extension and rotation, is associated with laxity or hip flexion contracture. Overall alignment should also be observed. Pelvic obliquity can be caused by

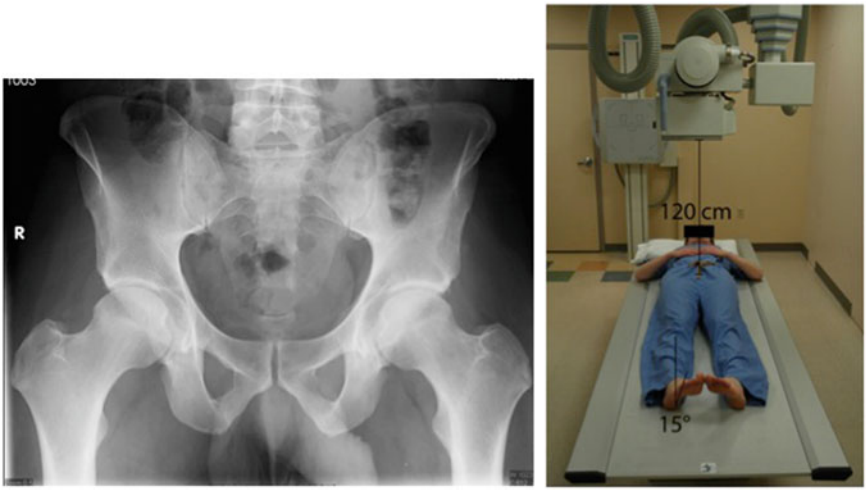


Fig. 1 The flexion–adduction internal rotation (*FADDIR*) (anterior impingement test) test is useful in diagnosing FAI with reproduction of pain in the groin/hip indicating a positive test

scoliosis or a leg length discrepancy. A single leg stance phase test can be performed. It is similar to the Trendelenburg test, as the patient flexes the knee and hip 45° and holds for 6 s. A pelvic shift or decrease by 2 cm is a positive test.

The seated examination can begin with a thorough neurovascular exam, including strength and sensation testing as well as documentation of peripheral pulses. A positive straight leg raise can detect any concomitant lumbar radiculopathy. Skin inspection and examination of lower extremity edema can again be performed in the seated position. Hip range of motion can be documented and compared to supine.

The next portion of the exam is performed supine. Palpation of various osseous and soft tissue locations should be performed, including the greater trochanter, ASIS, sciatic notch, ischial tuberosity, piriformis tendon and gluteus maximus insertions, the abdomen, pubic symphysis, and adductor tubercle. Resisted torso flexion with palpation of the abdomen can diagnose a sports hernia. Range of motion in flexion, extension, abduction, adduction, and internal and external rotation should be documented. Rotation testing is performed with the hip flexed to 90°. The Thomas test is used to rule out a hip flexion contracture. Both hips are brought to the chest, and the hips are sequentially brought into extension. Inability of the thigh to reach the table indicates a positive test. There are a wide variety of special exam maneuvers used in order to diagnose hip pathology [15]. The flexion–adduction–internal rotation (FADDIR) test is useful in diagnosing FAI (Fig. 1), with reproduction of pain indicating a positive test. In the dynamic external rotatory impingement test (DEXRIT), the contralateral hip is flexed past 90°, and then the ipsilateral hip is flexed to 90° or greater and taken through an arc of abduction and external rotation. A positive test indicates a pop or recreation of pain with the maneuver. The dynamic internal rotatory impingement test (DIRI) is similar to the DEXRIT; however, the hip is taken through an arc of adduction and internal rotation. The flexion–abduction–external rotation (FABER), or Patrick’s, test can differentiate a sacroiliac origin of pain from hip pain. The leg is brought into approximately 45° of flexion and externally rotated to place the foot on the contralateral knee, with a positive test causing posterior pain. A straight leg raise against resistance, or Stinchfield test, can cause pain secondary to the psoas placing pressure onto the labrum. The patient flexes the hip to 45° with the knee extended and resists the examiner’s hand applying a downward pressure. In the posterior rim impingement test, the patient is placed at the end

Fig. 2 The standard AP pelvis is taken with the patient lying supine with the lower extremities internally rotated approximately 15° to adjust for femoral anteversion. The beam should be directed to the *midpoint* of the symphysis and a *line* that connects the anterior superior iliac spines (*ASIS*) 120 cm from the patient

of the exam table with the legs hanging freely. The hips are flexed up to the chest, and the ipsilateral leg is extended off the table and simultaneously abducted and externally rotated.

The patient is placed in the lateral decubitus position for the next section of the physical exam. This position can aid in the palpation of the greater trochanter, piriformis, abductors, hamstring origin, and tensor fascia lata (TFL). Passive adduction or abduction contractures can be tested, including Ober's test, along with adduction with the hip in extension, neutral, and flexion. The TFL is tested in extension, the gluteus medius in neutral, and the gluteus maximus in flexion with the shoulders rotated back towards the table. The FADDIR test can be performed in the lateral position as well. The lateral rim impingement is a variation of the posterior rim impingement test, which is performed in the supine position. The hip is brought through a range of passive extension and flexion while the hip is abducted, with reproduction of pain indicating a positive sign. The piriformis test is also performed in the lateral position and involves active abduction and external rotation of the leg against resistance. Finally, extension, abduction, and external rotation of the hip with an anteriorly directed force and reproduction of pain can denote anterior laxity or ligamentum teres injury.

The final portion of the exam is conducted with the patient in the prone position. Femoral anteversion is tested by flexing the knee to 90° and rotating the hip until the greater trochanter is palpated directly lateral, measuring the angle between the tibia and a vertical line. A rectus contracture is tested by flexing the lower extremity towards the gluteus maximus. A restriction of motion or rising of the pelvis is a positive sign.

Imaging

In the diagnostic workup for hip or groin pain, X-rays should be the initial study of choice. In addition to a standard anteroposterior (AP) pelvis view, cross-table lateral, frog-leg lateral, Dunn lateral, false profile, and abduction–internal rotation (ABIR) views can be obtained [16]. The standard AP pelvis is taken with the patient lying supine with the lower extremities internally rotated approximately 15° to adjust for femoral anteversion. The beam should be directed to the midpoint of the symphysis and a line that connects the anterior superior iliac spines (ASIS) (Fig. 2). The cross-table lateral is used to visualize the contours of the femoral head–neck junction. This view is also obtained with the patient supine and the ipsilateral leg internally rotated. The

Fig. 3 The Dunn view can be obtained at either 45° or 90°. In this example, the symptomatic hip is flexed to 45°, abducted 20°, and maintained in neutral rotation with the beam directed at a *midpoint* between the ASIS and symphysis

contralateral hip is flexed and the beam is directed towards the inguinal fold at 45°. Alternatives to the cross-table lateral include the frog-leg lateral and the Dunn lateral view (Fig. 3). In order to obtain a frog-leg lateral view, the beam is directed vertically or with a slight (10–15°) cephalic tilt while the patient is supine with the knees flexed, soles of feet together, and thighs maximally abducted. The Dunn view can be obtained at either 45° or 90°. The symptomatic hip is flexed to either 45° or 90°, abducted 20°, and maintained in neutral rotation with the beam directed similar to the AP pelvis at a midpoint between the ASIS and symphysis. The false profile view is performed with the patient standing and the ipsilateral hip against the X-ray cassette and the pelvis rotated 65° in relation to the wall stand (Fig. 4). The ipsilateral foot is parallel to the cassette and the beam centered on the femoral head. The X-ray tube to film distance should be approximately 120 cm for the above studies. The ABIR view is useful to examine congruency when planning a rotational osteotomy about the hip. It is obtained with the patient supine and hips in approximately 20° of abduction and internal rotation.

When evaluating imaging of a patient with hip pain or possible acetabular retroversion, there are also several radiographic parameters that need to be determined, including acetabular depth and coverage and congruency of the joint. Acetabular depth is often related to overcoverage or pincer-type FAI. Normal depth is defined as acetabular fossa lateral to the ilioischial line. Coxa profunda (Fig. 5) is defined by the fossa touching or crossing the ilioischial line. Protrusio acetabula (Fig. 6) is defined as the femoral head touching or crossing the ilioischial line. Anterior, lateral, and posterior acetabular coverage are also determined on X-rays. Anterior coverage is examined on the false profile view using the anterior center edge angle (CEA). This angle is formed by a vertical line and a line connecting the center of the femoral

Fig. 4 The false profile view is performed with the patient standing and the ipsilateral hip against the X-ray cassette and the pelvis rotated 65° in relation to the wall stand. The ipsilateral foot is parallel to the cassette and the beam centered on the femoral head

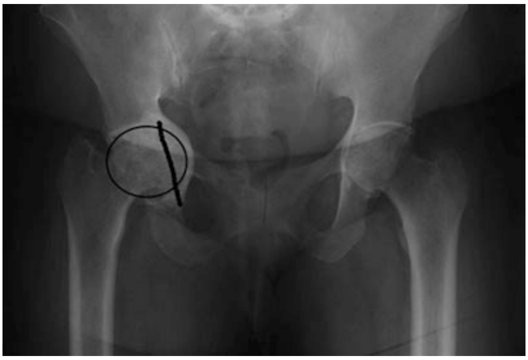


Fig. 5 Coxa profunda is defined by the floor of the acetabular fossa (*B*) touching or crossing the ilioischial line (*A*)

Fig. 6 Protrusio acetabula is defined as the femoral head (*black circle*) touching or crossing the ilioischial line (*black line*)

head and the anterior portion of the acetabular roof (Fig. 7). Lateral coverage is best determined by the acetabular index, extrusion index and lateral CEA, or center edge angle of Wiberg (Fig. 8). The acetabular index, or acetabular roof angle, is formed by a horizontal line and a line through the medial edge of the sclerotic zone and the lateral edge of the acetabulum (Fig. 9). The extrusion index is the ratio of the uncovered femoral head part to the total femoral head width, with pincer-type FAI having values less than 10 %. The lateral CEA is used to assess superolateral coverage and is formed by a vertical line and a line connecting the center of the femoral head to the lateral portion of the acetabular edge on an AP view. Values greater than 39° are typical in pincer impingement. Posterior coverage is viewed on the AP pelvis view. Reynolds, et al. originally described the crossover sign and the posterior wall sign [1]. The crossover sign was described as a line representing the lateral limit of the anterior wall that lies lateral to a similar point for the posterior wall. With normal anterversion of the acetabulum, on radiographs the anterior wall and posterior wall meet at the sourcil (Fig. 10). The posterior wall sign is present when the visible edge of the outline of the posterior wall descends medial to the center of the femoral head (Fig. 11). The ischial spine sign is also a marker of retroversion, which occurs when the ischial spine projects into the pelvis on an AP view (Fig. 12). It is important to determine the pelvic tilt on the imaging prior to any surgical decision making, as that can affect the version of the acetabulum based on varying degrees of tilt [17, 18]. An increase in the amount of pelvic inclination can show a positive crossover sign in the absence of acetabular retroversion. This pelvic inclination can be determined by measuring the distance between the symphysis pubis and the sacrococcygeal joint, with approximately 3.2 cm for men and 4.7 cm for women [17]. A larger distance indicates more of an inlet view and therefore shows a false increase in anterior coverage. Rotation towards the ipsilateral hip can also cause a false increase in retroversion. Neutral pelvic inclination is approximately 60° and should be standardized when obtaining radiographs to prevent any false appearance of overcoverage or undercoverage [18]. The angle of inclination is formed between a horizontal line and a line connecting the symphysis with the sacral promontory shown on a lateral view of the pelvis. Herniation pits in the femoral neck have also been associated with acetabular retroversion. Ji et al. performed a case–control study compared CT images in asymptomatic and symptomatic patients and found a significant association between herniation pits and central

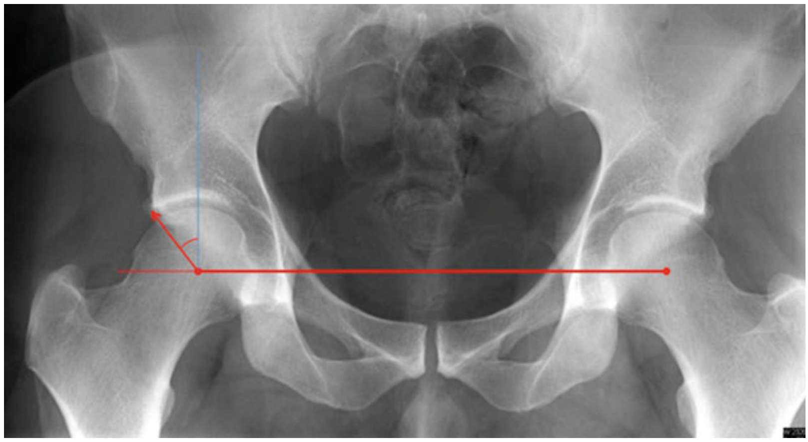


Fig. 7 The anterior center edge angle (*CEA*) is formed by a *vertical line* and a line connecting the *center* of the femoral head and the anterior portion of the acetabular roof

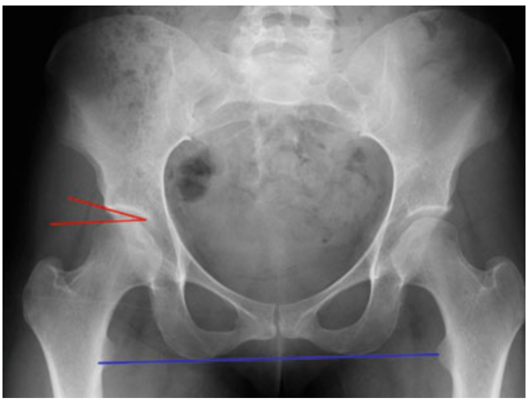


Fig. 8 The lateral center edge angle (*LCEA*) is calculated by creating a *horizontal line* connecting the *center* of the two femoral heads. A *vertical line* (*blue line*) is then drawn from the *center* of the femoral head to the acetabulum. A *second line* (*red arrow*) is then drawn to the lateral aspect of the sourcil. The angle subtended by these two lines is the LCEA

acetabular retroversion and pincer-type FAI [19]. Examination of joint congruity can help differentiate a pincer-type impingement or overcoverage from hip dysplasia. Shenton's line is formed by the top of the obturator foramen and the inner femoral neck. A break in this line denotes hip subluxation or dislocation. Lateralization of the femoral head based on the position of the medial aspect of the head relative to the ilioischial line also denotes an incongruous joint if greater than 10 mm (Fig. 13). Finally, the centrum collum diaphyseal angle can be measured. This is the angle formed by the femoral head–neck axis and the femoral shaft axis. Normal values are approximately 125–130°, with dysplastic hips having elevated values, and pincer-type FAI hips have decreased values.

In addition to radiographs, advanced imaging such as computed tomography (CT) and magnetic resonance imaging (MRI) can also be used to measure version of the acetabulum. Dandachli et al. used three-dimensional (3D) CT scans to determine version of the acetabulum [20]. They compared their results to radiographs and found 92 % sensitivity and 55 % specificity for the crossover sign and 81 % and 53 %, respectively, for the posterior wall sign. Kang et al. performed a similar study but found improved accuracy of radiographs when compared with CT scan, with a 71 % sensitivity and 88 % specificity for detecting retroversion [21]. Two separate methods have also been described for measurement of acetabular version using MRI [22]. Muhamad et al. used compared bony landmark measurements to soft tissue (labrum) measurements to measure acetabular version. They found that acetabular version remained consistent independent of the method used and can provide adequate information for calculating acetabular version.

In summary, there are a wide variety of diagnostic modalities available in the patient with hip pain. Hips with acetabular retroversion can have an increased lateral center edge angle, positive posterior wall sign, positive crossover sign, a positive ischial spine sign, and herniation pits in the femoral neck.

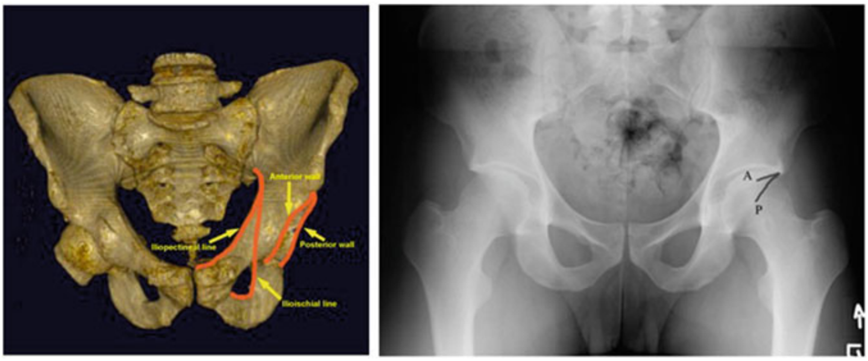


Fig. 9 The acetabular index, or acetabular roof angle, is formed by a *horizontal line* (*red line* parallel to *blue line* connecting center of femoral heads) and a line through the medial edge of the sclerotic zone and the lateral edge of the sourcil

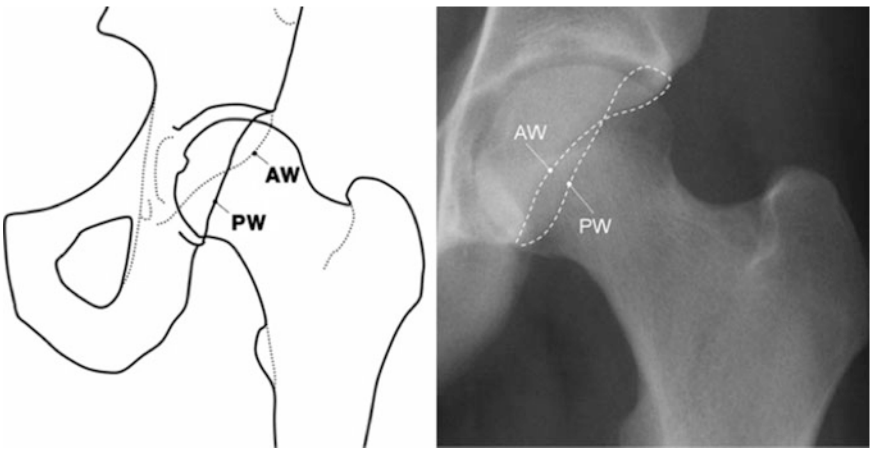


Fig. 10 Example of pelvic model with normal anteversion of the acetabulum. The posterior wall is lateral to the anterior wall. Radiographs of a normal anteverted acetabulum showing the anterior wall (*A*) and posterior wall (*P*) meet at the sourcil

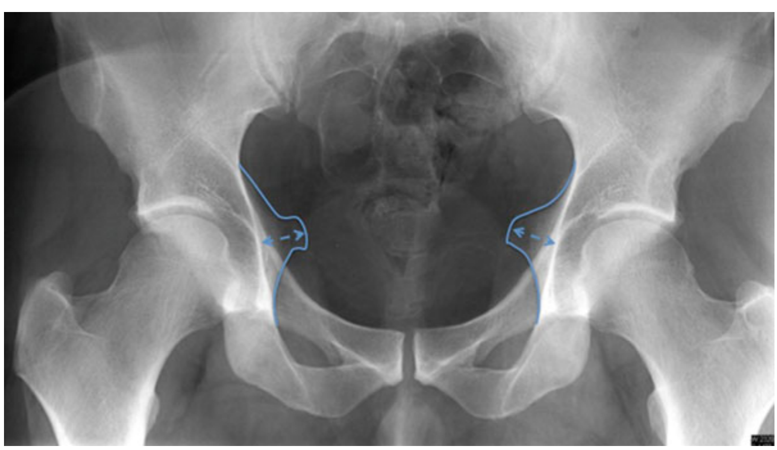


Fig. 11 The posterior wall sign is present when the visible edge of the outline of the posterior wall (*PW*) descends medial to the *center* of the femoral head

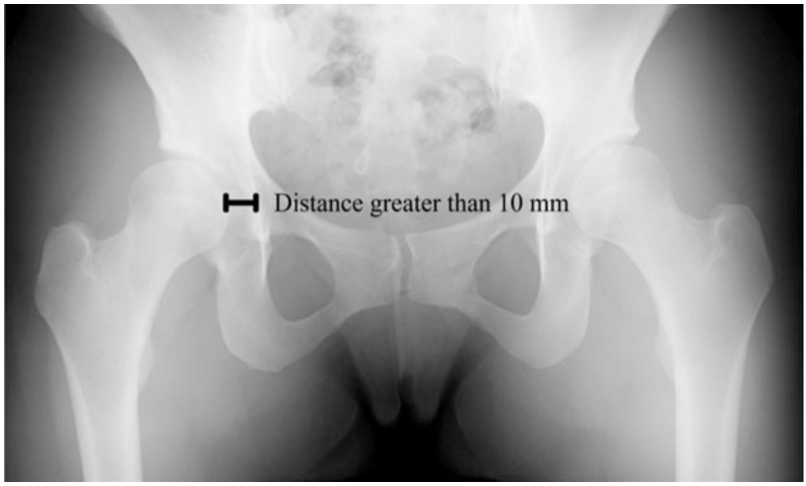


Fig. 12 The ischial spine sign is a marker of acetabular retroversion, which occurs when the ischial spine projects into the pelvis on an AP view (*blue outline*)

Fig. 13 Lateralization of the femoral head based on the position of the medial aspect of the head relative to the ilioischial line also denotes an incongruous joint if greater than 10 mm

Treatment

Nonoperative management is typically the initial treatment of choice for FAI [23, 24]. These modalities include activity modification, anti-inflammatory medications, and physical therapy. Therapy focuses on range of motion and strengthening of the hip muscles, specifically the hip abductors. Postural and core strengthening exercises can be implemented for those with concomitant lumbar spine pathology. Aquatic therapy can be included to lessen impact on the joints.

Operative treatment is divided into open, mini-open, and arthroscopic approaches. The open approach includes both surgical dislocation for management of FAI and a PAO. The mini-open approach is a combination of arthroscopy and a Hueter approach for osteochondroplasty and can also be used to address acetabular pathology if traction is applied. Arthroscopy allows visualization and management of the entire intra-articular compartment utilizing a minimally invasive approach.

For hip arthroscopy, the patient can be placed into the supine or lateral decubitus position for arthroscopic access to the hip, depending on surgeon preference. A fracture table or one specific for hip arthroscopy can be used. The feet are wrapped in protective boots and placed into foot holders.

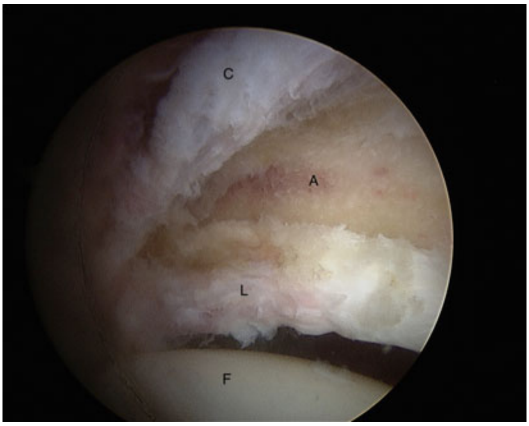


Fig. 14 During positioning for hip arthroscopy on a standard hip distractor table, the feet are wrapped in padded protective boots and placed into foot holders. A well-padded perineal post is placed as well to decrease incidence of pudendal neuropraxia

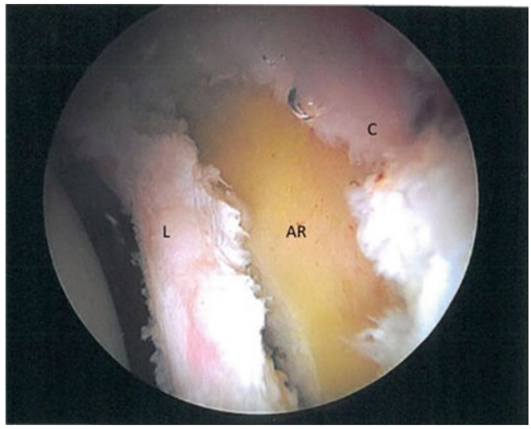


Fig. 15 Example of acetabular rim trimming. Femur (*F*), acetabular rim (*A*), labrum (*L*), and capsule (*C*) are identified

A well-padded perineal post is placed as well (Fig. 14). Prior to beginning the procedure, the ipsilateral extremity is internally rotated and fluoroscopy is used to determine the appropriate amount of distraction. Venting of the joint is performed using an 18-gauge spinal needle with further traction applied. A minimum of two portals (standard anterolateral and mid-anterior) are used with a capsulotomy performed to allow a diagnostic arthroscopy. Upon completion of the diagnostic arthroscopy and confirmation of acetabular retroversion, acetabular rim trimming can begin (Fig. 15). Similar to open techniques, it may be necessary to detach the labrum prior to rim trimming if resection of more than 3 mm of acetabular rim is planned (Fig. 16) [25]. The capsulolabral junction must be developed and care taken to avoid the reflected head of the rectus. A motorized burr is used to remove bone from the anterosuperior rim. A profile fluoroscopic view can be used to guide the resection. Reattachment of the labrum to the acetabular rim must be performed after completion of acetabular work (Fig. 17). Two to three anchors of surgeon choice (knotless vs. knotted) are used to reattach the labrum with care not to leave the knots on the intra-articular side if using knotted anchors. After the repair is completed, traction can be

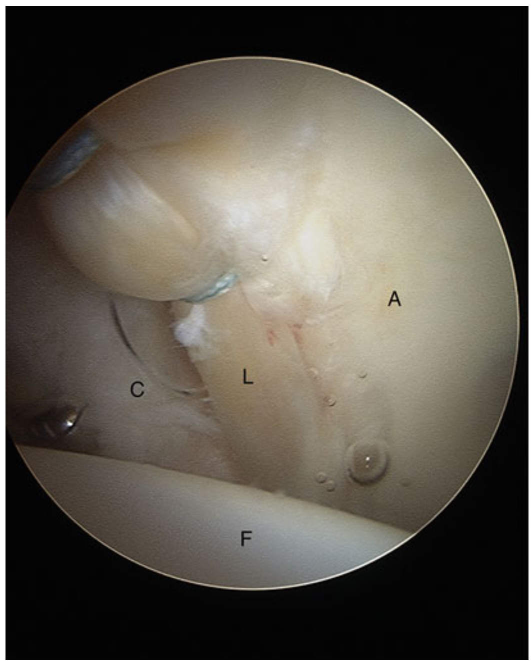


Fig. 16 Labral (*L*) detachment from acetabular rim (*AR*) if planned acetabular resection >3 mm. Capsule (*C*) is identified

released and a dynamic examination performed in order to determine the appropriate integrity of the labrum.

Open surgical dislocation of the hip for treatment of FAI has the longest track record of the three methods described. Preservation of the

Fig. 17 Reattachment of labrum (*L*) to the acetabular rim (*A*) using nonabsorbable sutures and anchors. Femoral head (*F*) and capsule (*C*) are identified

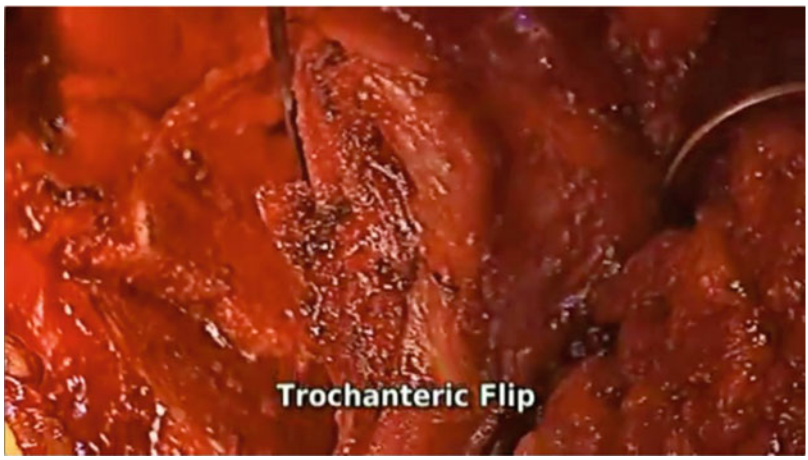


Fig. 18 Placement of the patient in the lateral decubitus position for open surgical hip dislocation

medial femoral circumflex artery (MFCA) is the key technical consideration for this approach [26]. The patient is placed into the lateral decubitus position with padded bolsters and the nonoperative limb protected. The patient is prepped and draped in a sterile fashion with a sterile bag drape opposite the table in order to receive the leg after dislocation (Fig. 18). A Gibson approach is made with split of the fascia lata. The leg is internally rotated to identify the posterior border of the gluteus medius. An incision is made from the posterosuperior edge of the greater trochanter distal to the posterior border of the ridge of the vastus lateralis. Next, a trochanteric osteotomy is made along the previously mentioned line. Initially Ganz described a single-plane osteotomy cut; however, recently a triplanar cut has been utilized to improve the stability of the fragments after fixation (Fig. 19). Proximally, the osteotomy should exit anterior to the posterior-most insertion of the gluteus medius in order to protect the deep branch of the MFCA. The greater trochanter fragment is mobilized anteriorly after release along the posterior border of the vastus lateralis to the middle of the gluteus maximus tendon. The posterior fibers of the gluteus medius are also released from the remaining trochanteric fragment. The leg is flexed and slightly externally rotated and the vastus lateralis and intermedius are elevated from the anterior and lateral proximal femur. The inferior gluteus medius is separated from the piriformis and capsule. This entire flap is then retracted superiorly and anteriorly to expose the superior portion of the capsule with

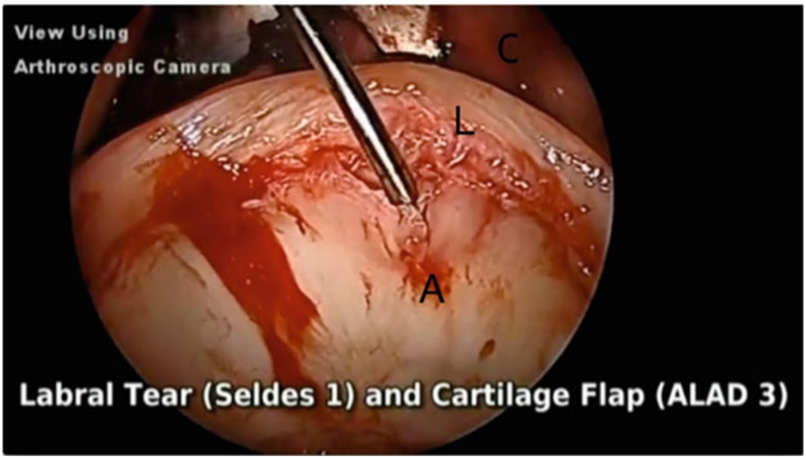


Fig. 19 A trochanteric osteotomy is performed. Initially Ganz described a single-plane osteotomy cut; however, recently a triplanar cut has been utilized to improve the stability of the fragments after fixation. Proximally, the osteotomy should exit anterior to the posterior-most insertion of the gluteus medius in order to protect the deep branch of the medial femoral circumflex artery. The greater trochanter fragment is mobilized anteriorly after release along the posterior border of the vastus lateralis to the *middle* of the gluteus maximus tendon. The posterior fibers of the gluteus medius are also released from the remaining trochanteric fragment

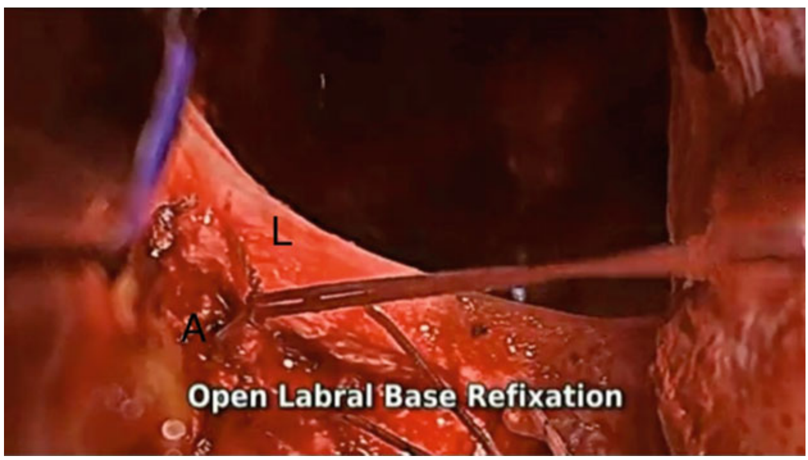


Fig. 20 Full examination of the femur and acetabulum (*A*) showing labral tear (Seldes) (*L*) and cartilage flap (*ALAD*). The capsule (*C*) is also visualized

progressive flexion and external rotation of the hip. An anterolateral incision along the capsule aligned with the long axis of the femoral neck is made, followed by an anteroinferior capsular incision remaining anterior to the lesser trochanter to protect the MFCA. Elevation of this flap allows visualization of the labrum. The initial capsular incision is extended to the acetabular rim and turned posteriorly. Next, the hip is dislocated by flexing, externally rotating, and placing it in front of the table in the sterile bag. Full examination of the femur and acetabulum is possible (Fig. 20). Acetabular rim trimming can be performed; however, labral detachment is necessary. The rim segment can be removed with a curved osteotome, with intraoperative evaluation necessary to determine the amount that is resected. After trimming is complete, the labrum is reattached with two to four anchors with the knots seated firmly and lying on the nonarticular surface of the labrum (Fig. 21). The hip is then reduced and capsular closure performed without excess tension. The osteotomy fragment is reduced and fixed with

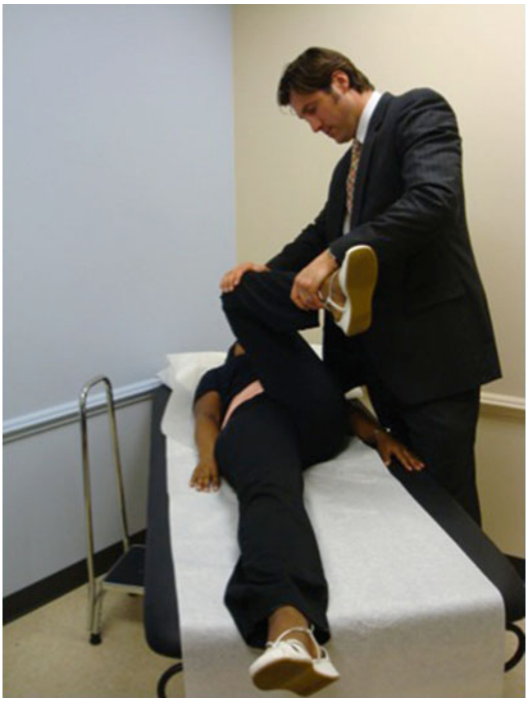


Fig. 21 Labrum (*L*) is reattached with two to four anchors placed in the acetabulum (*A*) with nonabsorbable suture. The *knots* are seated firmly, lying on the nonarticular surface of the labrum

either 3.5 millimeter (mm) or 4.5 mm screws. The wounds are irrigated and closed in a layered fashion.

The reverse PAO is another commonly performed procedure for open correction of acetabular retroversion [17]. The patient is first placed supine with the extremity draped free. A modified Smith-Peterson approach is performed, with the incision from the gluteal tubercle of the iliac crest to the lateral proximal thigh. The sartorius–tensor fascia lata (TFL) interval is identified and the TFL is split. The floor of the muscle compartment is incised longitudinally, and the rectus femoris is identified. The external oblique muscle is lifted and detached subperiosteally from the iliac crest 1.5 centimeters (cm) distal to the ASIS. Osteotomy of the ASIS is performed 1.5–2 cm proximal to its tip. The fragment is mobilized medially and subperiosteal dissection of the iliac wing to the pelvic brim is performed. The origin of the iliacus muscle is detached along the interspinous crest until the direct and reflected heads of the rectus femoris are visualized. The leg is then flexed to approximately 40° to relax the medial soft tissues. The direct head of the rectus is detached from the anteroinferior iliac spine (AIIS), and the indirect head is divided. The rectus is retracted medially and the iliocapsular muscle is detached from the capsule by lateral to medial dissection. The psoas tendon is then retracted medially. The space between the capsule and obturator externus is enlarged with scissors. Next, the osteotomies are performed with the hip in 45° of flexion. Partial osteotomy of the ischium is performed. Blunt dissection down to the ischial ramus is performed, and the width of the ischium is assessed. A curved pelvic osteotome is used with the osteotome pointing towards the contralateral shoulder and the handle posteroinferior. It is advanced 20–25 mm changing the direction of the handle to point posterosuperior. The medial bone bridge is cut and the lateral bridge is notched only to protect the sciatic nerve. Flexion, abduction, and external rotation of the hip also protect the sciatic nerve. The second osteotomy is of the superior pubic ramus just medial to the iliopectineal eminence. A complete cut is made perpendicular to the long axis of the pubic ramus with the osteotome pointing medially 45°. The approach to the quadrilateral plate is completed prior to the next osteotomies, and the gluteus minimus is detached between the ASIS and AIIS. The next osteotomies performed are the supra-acetabular and retro-acetabular. The osteotomy is marked from the lower border of the ASIS posteriorly and perpendicular at a distance of 1.5 cm from the pelvic brim. It is then continued distally at 110–120°. The supra-acetabular osteotomy is performed 1 cm from the greater sciatic notch. Using a curved osteotome, the remaining 1.5 cm to the brim is osteotomized at an angle of 110–120° from the initial cut. Using a straight osteotome, 20–30 mm of the quadrilateral plate is cut 1.5 cm anterior to the greater sciatic notch. An osteotome is then introduced into the posterior portion and levered against the acetabulum to break the sciatic spine. A Schanz pin is inserted through the AIIS into supra-acetabular bone and a spreader is inserted into the posterior part of the osteotomy and pulled distally and laterally. A 20 mm pelvic

Fig. 22 Postoperative AP pelvis of a reverse periacetabular osteotomy. After reorientation of the acetabulum to the desired position, definitive fixation is obtained with two 3.5 mm screws from the iliac crest into the acetabular fragment. Another screw is directed from the AIIS through the fragment towards the sacroiliac joint. Both heads of the rectus are reinserted with transosseous sutures and the ASIS is fixed with a 2.7 mm cortical screw

osteotome is advanced at an angle of 50° in the direction of the first ischial osteotomy 4 cm below the pelvic brim. The osteotome is advanced until decrease in tension is felt. The Schanz pin is rotated medially and spreader rotated externally to fracture the remaining bone bridge. The acetabulum is then reoriented as desired and provisionally stabilized with two 2.5 mm Kirschner wires. Definitive fixation is obtained with two 3.5 mm screws from the iliac crest into the acetabular fragment. Another screw is directed from the AIIS through the fragment towards the sacroiliac joint. Both heads of the rectus are reinserted with transosseous sutures, and the ASIS is fixed with a 2.7 mm cortical screw. The abdominal muscles are reattached to the iliac crest and fascia of the thigh approximated (Fig. 22).

Complications

General complications pertinent to all surgical procedures including infection, blood loss, and DVT are rare with the abovementioned procedures. Open surgical dislocation carries a risk of osteonecrosis, femoral neck fracture, symptomatic hardware, undercorrection or overcorrection of deformity, heterotopic ossification, and nonunion of the osteotomy site. Risks associated with the PAO include neurovascular injury, heterotopic ossification, avulsion of reattached muscles, posterior column fracture, loss of fixation, symptomatic hardware, nonunion, and delayed union. Complications after hip arthroscopy include capsulolabral adhesions, neurovascular injury, perineal numbness and genital injury, iatrogenic labral injury, heterotopic ossification, and postoperative instability.

Rehabilitation

Postoperative rehab protocols after arthroscopic surgery vary between surgeons; however, there are a few standard principles that the majority of surgeons follow [27]. Weight bearing can be initiated when the patient is able. Continuous passive motion or stationary biking begins as soon as the patient can tolerate. External rotation is usually limited for the first 2–3 weeks, as well as flexion past 90° and abduction. A specialized hip brace can be worn postoperatively to protect these motions. High-impact activities are generally avoided for the first 6 weeks, as is formal physical therapy.

After PAO or open surgical dislocation, touch-down weight bearing with crutches is permitted. Supine hip flexion is avoided with a PAO due to the reattachment of the hip flexors. If there is evidence of bony union at 8 weeks postoperatively, weight bearing and hip abductor strengthening are begun. For limited open techniques, abductor strengthening is begun immediately and 50 % weight bearing is allowed. Active hip flexion is also prohibited for the first 6 weeks. Postoperative methods of deep vein thrombosis (DVT) prophylaxis are also variable among surgeons, with the majority implementing foot pumps or aspirin.

Outcomes

Emara et al. followed 37 patients using nonoperative therapy for FAI and had an 11 % incidence of failure requiring surgery; however, 18 %

Table 1 Comparison of the advantages/disadvantages of open surgical hip dislocation and hip arthroscopy in the treatment of femoroacetabular impingement (*FAI*)

	Advantages	Disadvantages
Open surgical dislocation	Good visualization of joint	Major operation
	360° joint access	Soft tissue damage
	Enables treatment of all pathologies	Trochanteric osteotomy – risk of nonunion and hardware pain
	Templates can be used for femoral osteoplasty to ensure precise sphericity	Need to sacrifice ligamentum teres
		Increased to blood loss
		Longer rehabilitation
Arthroscopic surgery	Minimally invasive	Difficult access to ligamentum teres and inferior portion of joint
	Outpatient surgery	Traction complications – genital and perineal injury, pudendal neurapraxia
	Minor soft tissue damage	LFCN neurapraxia (portal injury)
	Faster rehabilitation	Abdominal compartment syndrome
	Easy approach to peripheral compartment and soft tissues	

LFNC lateral femoral cutaneous nerve

had recurrent hip pain [23]. Hunt et al. found a 44 % satisfaction rate with nonoperative therapy, with 56 % of patients eventually choosing surgery, but both groups improved significantly from their baseline status [24]. Studies examining the conservative treatment of FAI and other hip conditions are limited by lack of long-term follow-up and small sample sizes.

The outcomes of operative management have been better studied. Siebenrock et al. retrospectively reviewed 29 hips that underwent reverse PAO for treatment of anterior FAI due to AR [17]. They obtained 26 good or excellent outcomes overall, with three patients requiring reoperations and resolution of the crossover and posterior wall signs in all but four patients. Peters et al. studied 30 hips who underwent reverse PAO and 30 hips who underwent open surgical hip dislocation and osteochondroplasty for acetabular retroversion [28, 29]. They found that the Harris Hip Score improved from 52 to 90 in the hips treated with surgical dislocation and 72–91 in those treated with periacetabular osteotomy. Elimination of crossover sign and correction of posterior wall sign occurred in greater than 90 % of all patients when present.

Botser et al. also compared outcomes after open versus arthroscopic techniques [30]. They found a higher complication rate for the mini-open technique (16 %) as opposed to the open (9.2 %) and arthroscopic (1.7 %) approaches; however, the majority of these were neuropraxia in the mini-open group as opposed to major complications. Table 1 displays their algorithm for comparing open versus arthroscopic treatment. Matsuda performed a systematic review comparing outcomes after open, mini-open and arthroscopic management of FAI [31]. They concluded that after short- to midterm follow-up, there was improvement of pain and function in all techniques. The open approach had the highest complication rates secondary to trochanteric osteotomy and implants. The mini-open approach had the highest incidence of lateral femoral cutaneous nerve (LFCN) palsies. The arthroscopic approach had the lowest rate of complications with equivalent clinical outcomes. Recent studies with early to midterm results for the arthroscopic treatment of femoroacetabular impingement have shown promising results. Larson et al. followed 96 consecutive patients who underwent arthroscopic management of femoroacetabular impingement and found significant improvement for all outcome measures including Harris Hip Score, Short Form 12, and visual analog score with a mean of 9.9 months' follow-up [32]. Philippon et al. evaluated clinical outcomes after treatment for femoroacetabular impingement in the pediatric and adolescent population with a minimum of 2 years' follow-up [33]. They found that their modified Harris Hip Score increased from a mean of 57–91 ($P < 0.001$).

Summary

Acetabular retroversion is an increasingly recognized phenomenon that appears in a variety of disorders about the hip that can lead to pain, disability, and degenerative changes. A careful detailed assessment of both static and dynamic structures is essential for the correct treatment of this abnormality. Improved diagnostic methods and early treatment can lead to enhanced outcomes and likely delay or prevent salvage procedures in the future. When considering surgical intervention, careful analysis of the pathomechanics of the hip must be undertaken to ensure a successful outcome.

References

1. Reynolds D, Lucas J, Klaue K. Retroversion of the acetabulum. A cause of hip pain. J Bone Joint Surg Br. 1999;81(2):281–8.
2. Wenger DE, Kendell KR, Miner MR, Trousdale RT. Acetabular labral tears rarely occur in the absence of bony abnormalities. Clin Orthop Relat Res. 2004;426:145–50.
3. Giori NJ, Trousdale RT. Acetabular retroversion is associated with osteoarthritis of the hip. Clin Orthop Relat Res. 2003;417:263–9.
4. Ezoe M, Naito M, Inoue T. The prevalence of acetabular retroversion among various disorders of the hip. J Bone Joint Surg Am. 2006;88(2):372–9.
5. Fujii M, Nakashima Y, Yamamoto T, Mawatari T, Motomura G, Matsushita A, et al. Acetabular retroversion in developmental dysplasia of the hip. J Bone Joint Surg Am. 2010;92(4):895–903.
6. Sankar WN, Millis MB, Kim YJ. Instability of the hip in patients with Down syndrome: improved results with complete redirectional acetabular osteotomy. J Bone Joint Surg Am. 2011;93(20):1924–33.
7. Sankar WN, Schoenecker JG, Mayfield ME, Kim YJ, Millis MB. Acetabular retroversion in Down syndrome. J Pediatr Orthop. 2012;32(3):277–81.
8. Werner CM, Copeland CE, Ruckstuhl T, Stromberg J, Seifert B, Turen CH. Prevalence of acetabular dome retroversion in a mixed race adult trauma patient population. Acta Orthop Belg. 2008;74(6):766–72.
9. Tannenbaum E, Kopydlowski N, Smith M, Bedi A, Sekiya JK. Gender and racial differences in focal and global acetabular version. J Arthroplast. 2014;29(2): 373–6.
10. Kiyama T, Naito M, Shiramizu K, Shinoda T. Postoperative acetabular retroversion causes posterior osteoarthritis of the hip. Int Orthop. 2009;33(3): 625–31.
11. Dora C, Mascard E, Mladenov K, Seringe R. Retroversion of the acetabular dome after Salter and triple pelvic osteotomy for congenital dislocation of the hip. J Pediatr Orthop B. 2002;11(1):34–40.
12. Ganz R, Parvizi J, Beck M, Leunig M, Notzli H, Siebenrock KA. Femoroacetabular impingement: a cause for osteoarthritis of the hip. Clin Orthop Relat Res. 2003;417:112–20.
13. Henak CR, Abraham CL, Anderson AE, Maas SA, Ellis BJ, Peters CL, et al. Patient-specific analysis of cartilage and labrum mechanics in human hips with acetabular dysplasia. Osteoarthr Cartil/OARS, Osteoarthr Res Soc. 2013.
14. DeAngelis NA, Busconi BD. Assessment and differential diagnosis of the painful hip. Clin Orthop Relat Res. 2003;406:11–8.
15. Martin HD, Shears SA, Palmer IJ. Evaluation of the hip. Sports Med Arthrosc Rev. 2010;18(2):63–75.
16. Clohisy JC, Carlisle JC, Beaule PE, Kim YJ, Trousdale RT, Sierra RJ, et al. A systematic approach to the plain radiographic evaluation of the young adult hip. J Bone Joint Surg Am. 2008;90 Suppl 4:47–66.
17. Siebenrock KA, Kalbermatten DF, Ganz R. Effect of pelvic tilt on acetabular retroversion: a study of pelves from cadavers. Clin Orthop Relat Res. 2003;407:241–8.
18. Tannast M, Murphy SB, Langlotz F, Anderson SE, Siebenrock KA. Estimation of pelvic tilt on anteroposterior X-rays–a comparison of six parameters. Skelet Radiol. 2006;35(3):149–55.
19. Ji HM, Baek JH, Kim KW, Yoon JW, Ha YC. Herniation pits as a radiographic indicator of pincer-type femoroacetabular impingement in symptomatic patients. Knee Surg Sports Traumatol Arthrosc. 2013.
20. Dandachli W, Ul Islam S, Tippett R, Hall-Craggs MA, Witt JD. Analysis of acetabular version in the native hip: comparison between 2D axial CT and 3D CT measurements. Skelet Radiol. 2011;40(7):877–83.
21. Kang AC, Gooding AJ, Coates MH, Goh TD, Armour P, Rietveld J. Computed tomography assessment of hip joints in asymptomatic individuals in relation to femoroacetabular impingement. Am J Sports Med. 2010;38(6):1160–5.

22. Muhamad AR, Freitas JM, Bomar JD, Hosalkar H. Acetabular version on magnetic resonance imaging: analysis of two different measuring techniques. Hip Int J Clin Exp Res Hip Pathol Ther. 2012;22(6): 672–6.
23. Emara K, Samir W, el Motasem H, Ghafar KA. Conservative treatment for mild femoroacetabular impingement. J Orthop Surg. 2011;19(1):41–5.
24. Hunt D, Prather H, Harris Hayes M, Clohisy JC. Clinical outcomes analysis of conservative and surgical treatment of patients with clinical indications of prearthritic, intra-articular hip disorders. PM & R J Inj Funct Rehabil. 2012;4(7):479–87.
25. Fry R, Domb B. Labral base refixation in the hip: rationale and technique for an anatomic approach to labral repair. Arthrosc J Arthrosc Relat Surg. 2010;26 (9 Suppl):S81–9.
26. Ganz R, Gill TJ, Gautier E, Ganz K, Krugel N, Berlemann U. Surgical dislocation of the adult hip a technique with full access to the femoral head and acetabulum without the risk of avascular necrosis. J Bone Joint Surg Br. 2001;83(8): 1119–24.
27. Enseki KR, Martin R, Kelly BT. Rehabilitation after arthroscopic decompression for femoroacetabular impingement. Clin Sports Med. 2010;29(2):247–55, viii.
28. Peters CL, Erickson JA, Hines JL. Early results of the Bernese periacetabular osteotomy: the learning curve at an academic medical center. J Bone Joint Surg Am. 2006;88(9):1920–6.
29. Peters CL, Erickson JA, Anderson L, Anderson AA, Weiss J. Hip-preserving surgery: understanding complex pathomorphology. J Bone Joint Surg Am. 2009;91 Suppl 6:42–58.
30. Botser IB, Smith Jr TW, Nasser R, Domb BG. Open surgical dislocation versus arthroscopy for femoroacetabular impingement: a comparison of clinical outcomes. Arthrosc J Arthrosc Relat Surg. 2011;27(2):270–8.
31. Matsuda DK, Carlisle JC, Arthurs SC, Wierks CH, Philippon MJ. Comparative systematic review of the open dislocation, mini-open, and arthroscopic surgeries for femoroacetabular impingement. Arthrosc J Arthrosc Relat Surg. 2011;27(2):252–69.
32. Larson CM, Giveans MR. Arthroscopic management of femoroacetabular impingement: early outcomes measures. Arthrosc J Arthrosc Relat Surg. 2008;24(5):540–6.
33. Philippon MJ, Ejnisman L, Ellis HB, Briggs KK. Outcomes 2 to 5 years following hip arthroscopy for femoroacetabular impingement in the patient aged 11 to 16 years. Arthrosc J Arthrosc Relat Surg. 2012;28(9):1255–61.

Surgical Technique: Reverse Periacetabular Osteotomy

47

Christoph E. Albers, Simon D. Steppacher, Moritz Tannast, and Klaus A. Siebenrock

Contents

C.E. Albers • S.D. Steppacher • M. Tannast •
K.A. Siebenrock (✉)
Department of Orthopedic Surgery, Inselspital, University of Bern, Bern, Switzerland
e-mail: christoph.albers@insel.ch; simon.steppacher@insel.ch; moritz.tannast@insel.ch; klaus.siebenrock@insel.ch

Abstract

Acetabular retroversion is the result of an externally rotated hemipelvis rather than a focal overgrowth of the anterior wall and/or hypoplasia of the posterior wall. Acetabular retroversion is a cause of pincer impingement which, if left untreated, can lead to hip pain and osteoarthritis. The causal surgical treatment in hips with acetabular retroversion is acetabular reorientation with a reverse periacetabular osteotomy (PAO). Indication is based on a positive correlation among symptoms (typically groin pain), physical findings on examination (positive anterior impingement test and decreased flexion and internal rotation), and radiographic signs for acetabular retroversion. These include a positive crossover, posterior wall, and ischial spine sign. A reverse PAO is performed with four osteotomies and a controlled fracture. Unlike reorientation of the acetabular fragment in dysplastic hips, correction for acetabular retroversion is achieved by a combined extension and internal rotation of the acetabular fragment. Typically, a small supra-acetabular wedge resection is required to allow sufficient extension of the fragment. The quality of acetabular reorientation is evaluated by intraoperative AP pelvic radiographs. In addition, intraoperative testing of range of motion following acetabular reorientation is mandatory. An arthrotomy and offset correction of the femoral head-neck area is indicated in hips with decreased internal rotation following

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_116

acetabular reorientation. In a 10-year follow-up study of reverse PAO, a favorable outcome with preservation of all native joints was found. Correct acetabular orientation and, if necessary, a concomitant offset correction were the keys of successful outcome.

Introduction

Correct three-dimensional acetabular orientation, and in particular acetabular version, is crucial for appropriate range of motion of the hip. Acetabular retroversion is an acetabular pathomorphology with malorientation of the acetabular opening which is partially or completely facing posteriorly [1]. As a result, an early pathologic contact between the prominent anterior acetabular rim and the femoral neck occurs during range of motion (pincer type of femoroacetabular impingement) [1–4]. Patients typically present with hip pain and restricted internal rotation and flexion [4]. Pincer impingement, if left untreated, can result in early osteoarthritis of the hip [2, 5]. Radiographic diagnosis of acetabular retroversion is defined by a positive crossover [1, 4, 6], posterior wall [1], and ischial spine sign [7]. Acetabular retroversion has been associated with developmental dysplasia of the hip [8], Legg-Calvé-Perthes disease [9], and proximal femoral focal deficiency [10] but also occurs as an isolated entity with a prevalence of 5 % [3].

The association of extra-articular morphological landmarks like the ischial spine with a retroverted acetabulum suggests that acetabular retroversion is a pelvic dysmorphism rather than an isolated malorientation of the acetabulum [7, 11]. There is increasing evidence that acetabular retroversion is the result of an externally rotated hemipelvis [7, 12–15] rather than an anterior wall prominence and/or a posterior rim hypoplasia as suggested previously [3]. Therefore, the causal surgical treatment option to restore normal anatomy in hips with acetabular retroversion is an anteverting acetabular reorientation, e.g., a reverse periacetabular osteotomy (PAO) [16–18]. Trimming of the anterior rim in retroverted hips with a hypoplastic posterior wall could potentially decrease the size of the articular surface to a critical level and result in a dysplastic hip.

This chapter describes the radiographic parameters in hips with acetabular retroversion, reports the indications for a reverse PAO and its postoperative regime, and illustrates the steps of this surgical procedure.

Radiographic Evaluation

Conventional imaging consists of an anteroposterior (AP) pelvic radiograph and an axial view of the hip (e.g., cross-table lateral view). Correct patient positioning and centering of the X-ray beam are mandatory since the projected acetabular orientation on the AP pelvic radiograph directly relies on these factors [4, 19]. In an anteverted hip, the posterior rim is projected laterally to the anterior rim and both converge towards the cranio-lateral edge of the acetabulum (Fig. 1). Acetabular retroversion is defined by a positive crossover [1, 4, 6], posterior wall [1], and ischial spine sign [7]. A crossover sign is considered positive if the anterior acetabular rim crosses the course of the posterior rim (Fig. 1). In hips with a mild acetabular retroversion, the crossover sign typically is found close to the cranio-lateral edge of the acetabulum (Fig. 1). With increasing retroversion of the acetabulum, the crossover sign is projected more caudally. The retroversion index quantifies the extent of acetabular retroversion. It is defined as the ratio of the retroverted cranio-lateral acetabular opening to the entire opening (Fig. 1). The posterior acetabular coverage in hips with acetabular retroversion is typically deficient. The posterior acetabular wall sign is positive if the deficient posterior acetabular wall runs medial to the femoral head center (Fig. 1) [1]. The ischial spine sign is considered positive if the ischial spine is projected medially to the pelvic brim (Fig. 1) [7]. The association of acetabular retroversion with an extra-articular anatomical landmark such as the ischial spine shows that acetabular retroversion is not an isolated pathomorphology of the acetabulum itself but rather involves the entire hemipelvis. In a morphometric study of the pelvis, it could be shown that acetabular retroversion is

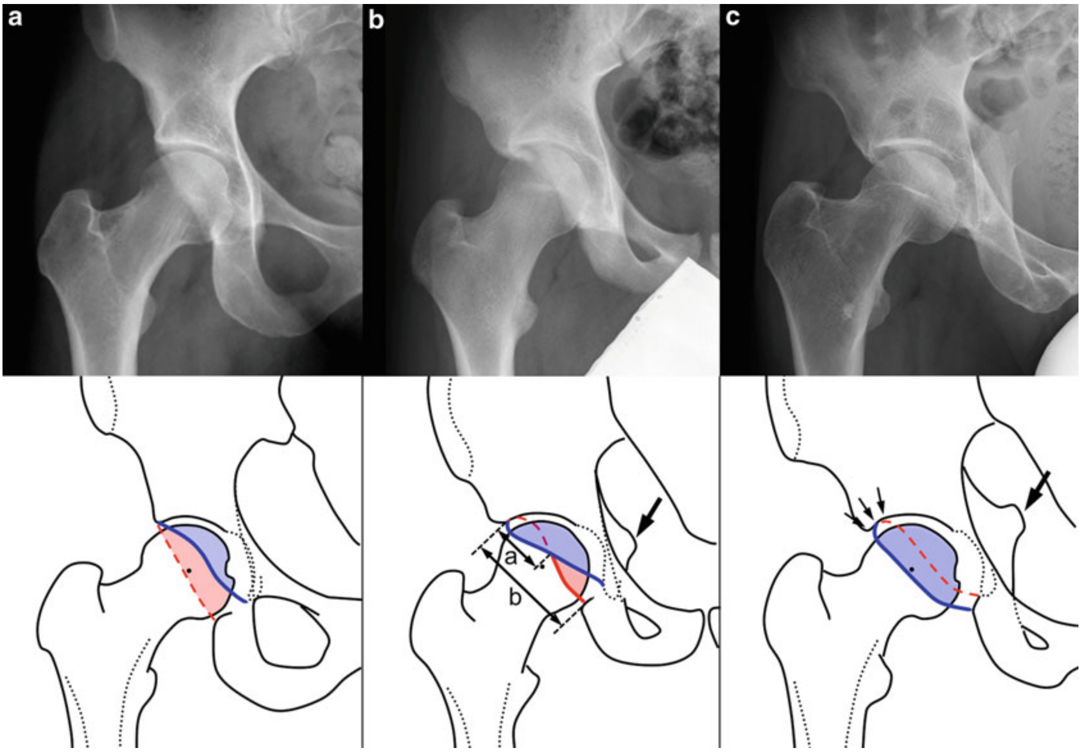


Fig. 1 (**a**) In an anteverted acetabulum, the posterior rim (red line) is projected laterally to the anterior rim (blue line) and both converge towards the cranio-lateral edge of the acetabulum. (**b**) Acetabular retroversion is defined by a positive crossover [1, 4, 6], posterior wall [1], and ischial spine sign [7]. A crossover sign is considered positive if the anterior acetabular rim crosses the course of the posterior rim [1]. With increasing retroversion of the acetabulum, the crossover sign is projected more caudally. The retroversion index (ratio of distance "a" to "b") quantifies the extent of acetabular retroversion. The posterior acetabular wall sign is positive if the deficient posterior acetabular wall runs medial to the femoral head center [1]. The ischial spine sign is considered positive if the ischial spine (arrow) is projected medially to the pelvic brim [7]. (**c**) Rarely total acetabular retroversion occurs with the entire acetabulum facing posteriorly. These cases can be missed because of the missing crossover sign. The entire anterior acetabular rim is projected laterally to the posterior wall on the AP pelvic radiograph. Typically, the anterior and posterior acetabular walls converge in the cranio-lateral edge and from an obtuse angle (*arrows*) instead of a sharp angle like in anteverted hips

due to an externally rotated hemipelvis [15]. Acetabular retroversion was associated with a lateral protruding iliac wing, a narrow ischium, a prominent anterior inferior iliac spine, and a steep ilioischial line (Fig. 2) [15]. These radiographic parameters of the pelvis can be used to assure the diagnosis of acetabular retroversion.

Very rarely total acetabular retroversion occurs with the entire acetabulum facing posteriorly (Fig. 1). These cases can be missed because of an absent crossover sign. The entire anterior acetabular rim is projected laterally to the posterior wall on the AP pelvic radiograph (Fig. 1). Typically, the anterior and posterior acetabular walls converge in the cranio-lateral edge and from an obtuse angle instead of a sharp angle in anteverted hips (Fig. 1).

The axial view (e.g., cross-table axial) is used to evaluate the femoral head-neck offset. A cam-type deformity is often present in hips with acetabular retroversion [2]. An untreated cam-type deformity can adversely affect the long-term outcome following acetabular reorientation [17, 20].

Magnetic resonance (MR) arthrography of the hip with radial sequences, intra-articular contrast

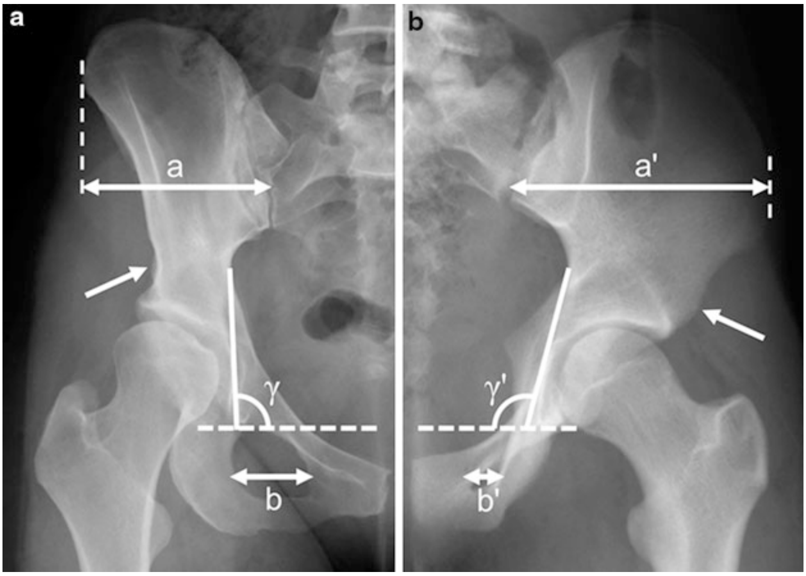


Fig. 2 Acetabular retroversion is the result of an externally rotated hemipelvis [7, 12–15] rather than an anterior wall prominence and/or a posterior rim hypoplasia as suggested previously [3]. Several extra-articular anatomical landmarks associated with acetabular retroversion exist. (**a**) Half an AP pelvic radiograph with a dysplastic hip in comparison with (**b**) half an AP pelvic radiograph with a retroverted hip. (**a**) The pelvis with an acetabular retroversion typically has a lateral protruding iliac wing ($a' > a$), a narrow obturator foramen ($b' < b$), a prominent anterior inferior iliac spine (*arrows*), and a less steep ilio-ischial line ($\gamma' > \gamma$) [15] (Figure reprinted with permission)

agent (gadolinium), and flexible surface coils is the current gold standard to detect chondrolabral pathologies in hips [21]. This imaging modality with radial sequences also allows exact location of additional cam-type deformities around the femoral neck. At the author's institution some additional MR slices at the height of the distal femur are performed which allows evaluating femoral antetorsion [22]. MR arthrography is not performed routinely in patients with acetabular retroversion but might be indicated in older patients to quantify chondrolabral damage.

Although computed tomography (CT) imaging allows exact three-dimensional evaluation of acetabular orientation, it is not performed on regular basis for diagnosis of acetabular retroversion. It can be used to determine femoral antetorsion and to evaluate whether a concomitant intertrochanteric femoral osteotomy is indicated [23]. Recently, a CT-based method has been introduced for three-dimensional planning of PAO with simulation of postoperative range of motion and possible impingement conflicts [24]. Accurate realization of the planned acetabular reorientation is accomplished using CT-based navigation [24].

Indication for Reverse PAO

Indication for acetabular reorientation is based on a positive correlation of symptoms, physical findings, and radiographic findings. Typically, patients with acetabular retroversion present with groin pain and decreased flexion and internal rotation [17, 18]. The anterior impingement test, performed with the patient supine and internal rotation in 90° of flexion, results in reproducible hip pain [4]. In extreme forms of acetabular retroversion, the anterior impingement test might not be possible, and there is unavoidable passive external rotation in flexion (positive Drehmann's sign [25]). Radiographically, indication is based on a combination of positive crossover [1, 4, 6], posterior wall [1], and ischial spine sign [7] with an acetabular retroversion index exceeding 30 % (Fig. 1). Typically, these patients are young with the majority aged less than 35 years [18].

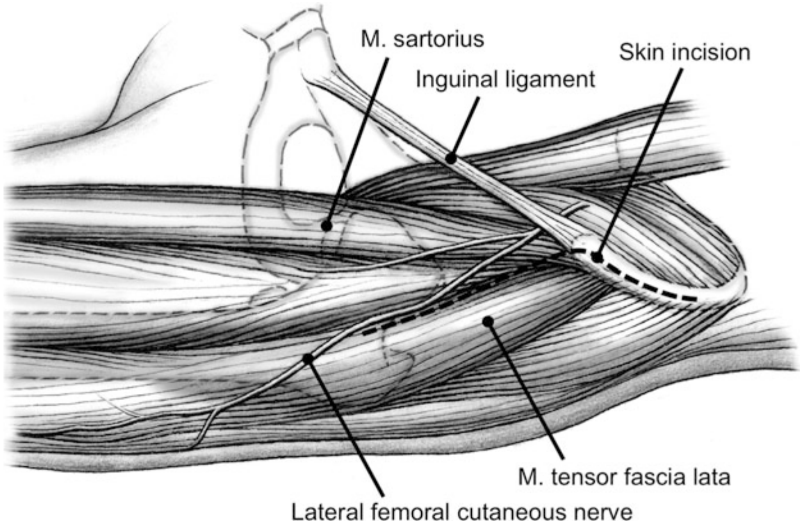


Fig. 3 The periacetabular osteotomy (PAO) is performed through a modified Smith-Petersen approach. The incision includes the anterior third of the iliac crest, is curved at the anterior superior iliac spine (ASIS), and runs distally along the anterior boarder of the tensor muscle. Care must be taken not to injure the lateral femoral cutaneous nerve, which pierces the fascia of the thigh close to the intermuscular interval of tensor and sartorius muscles

A contraindication is advanced osteoarthritis Tönnis grade > 1 [26]. Ideally, the arthro-MRI shows no or minor chondrolabral damage.

Concomitant surgical procedures comprise the arthrotomy with offset correction or intertrochanteric osteotomies. Offset of the anterior femoral head-neck junction is judged on the axial view of the hip. An alpha angle exceeding 50° is considered insufficient offset [27]. However, intraoperative testing of range of motion following acetabular reorientation is crucial. Often a cam-type deformity limits internal rotation. If a minimum of 30° of internal rotation in 90° of flexion is not achieved, an arthrotomy with offset correction is indicated. A concomitant intertrochanteric osteotomy in hips with acetabular reorientation due to retroversion is rarely indicated. A high femoral antetorsion could potentially result in a posterior impingement following acetabular reorientation [28]. A derotational femoral osteotomy could be indicated in these hips.

PAO Versus Surgical Hip Dislocation

Trimming of the prominent anterior acetabular wall in retroverted hips can potentially decrease the size of the articular surface to a critical level resulting in a dysplastic hip. However, surgical hip dislocation with acetabular rim trimming can be an alternative in some selected hips with a lower degree of acetabular retroversion. Hips with a retroversion index of less than 30 % and a negative posterior wall sign are eligible for rim trimming [17, 18]. In these hips a sufficient size of the articular surface can be obtained following acetabular rim trimming [17, 18]. In contrast, a reverse PAO resulted in a too prominent posterior wall and a posterior impingement [17, 18].

Surgical Technique

In general, the approach and the sequence of osteotomies do not differ in a PAO performed for acetabular retroversion compared to a PAO for hip dysplasia [16]. The surgery is performed under general anesthesia with full muscle relaxation. The patient is positioned supine on a radiolucent table. Sterile draping of the leg should allow mobility of the leg during surgery. The entire iliac crest and femur should be accessible. A blood salvage device may be used to encounter excessive blood loss.

A modified Smith-Petersen approach [29] is used with an incision reaching from the anterior third of the iliac crest to the anterior superior iliac spine. The incision is then curved and extended caudally along the anterior boarder of the tensor muscle for approximately 10 cm (Fig. 3). Care must be taken not to injure the lateral femoral cutaneous nerve (Fig. 3). The fascia is incised at

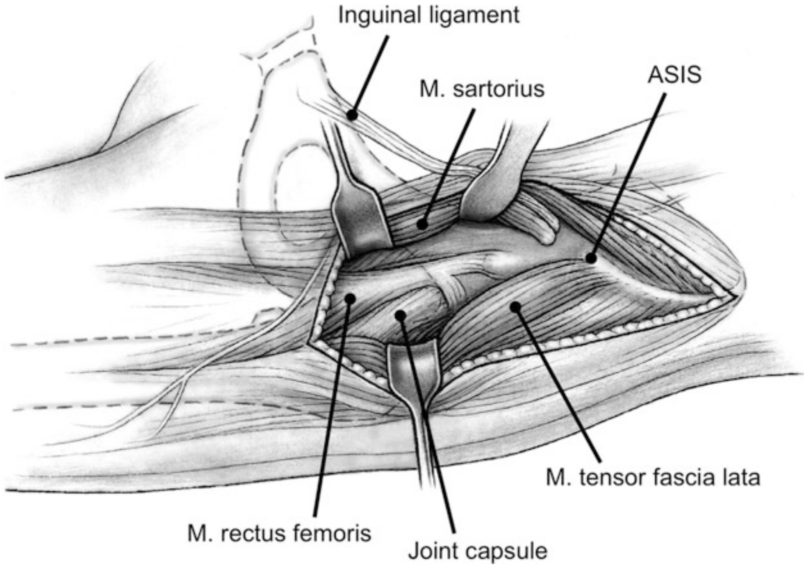


Fig. 4 The inguinal ligament and the sartorius muscle are detached from its origin at the anterior superior iliac spine (ASIS) and retracted medially. By retracting the tensor fascia lata muscle laterally, the joint capsule and the origin of the rectus muscle with its direct and reflected portions are exposed

the anterior border of the tensor muscle. Staying within the fascial sheath of this muscle will protect the lateral femoral cutaneous nerve. The tensor fascia lata muscle is retracted laterally. Flexion of the leg decreases tension of the tensor muscle. A further deep fascia layer is incised longitudinally, and the reflected tendinous portion of the origin of the rectus femoris muscle is exposed. Distally, the ascending branch of the lateral femoral circumflex artery crosses the intermuscular interval of tensor and rectus muscles. This vessel should be preserved and it usually represents the distal end of the exposure.

At this point the aponeurosis of the abdominal muscles is detached from the anterior third of the iliac crest sharply with a knife like it is done for exposure of the first window in an ilioinguinal approach. With the hip in flexion and adduction, the iliacus muscle is bluntly mobilized from the iliac bone. The inguinal ligament and the sartorius muscle are detached subperiosteally from its origin at the anterior superior iliac spine (ASIS; Fig. 4). This step has replaced the osteotomy of the ASIS, which was performed routinely in earlier years [16]. Dissecting the inguinal ligament and the sartorius muscle of the ASIS connects the first ilioinguinal window with the previously performed exposure of the anterior aspect of the hip joint area. Medial retraction of the rectus muscle with a blunt retractor exposes the iliocapsularis muscle which is attached to the anterior aspect of the capsule. Sharp dissection of its lateral border from the capsule allows medial mobilization. In order to preserve the rectus muscle attachment, the proximal aspect of this muscle is now retracted laterally in order to continue complete sharp dissection of the iliocapsularis muscle from the capsule (Fig. 5). Additional medial retraction of the iliopsoas muscle is facilitated by adduction and flexion of the hip joint. The psoas tendon is retracted medially with a Hohmann retractor which is placed into the superior pubic ramus medially to the eminentia iliopubica (Fig. 5). This protects the femoral nerve and vessels that are located medially to the psoas tendon. This will lead to exposure of the anterior inferior iliac spine and of the corpus pubis and the psoas tendon surrounded by the iliopectineal bursa.

Finally, blunt dissection of the infra-articular space between the psoas tendon and the capsule with a pair of scissors is performed until the tip of the instrument touches the base with the medial aspect of the ischial bone lateral to the obturator foramen (Fig. 5).

For preparation of the outer aspect of the pelvis, subperiosteal dissection is performed starting between the superior and inferior anterior iliac spine. Only limited dissection of the outer part of the pelvis is necessary to place a blunt pelvic retractor in the sciatic notch, thereby protecting

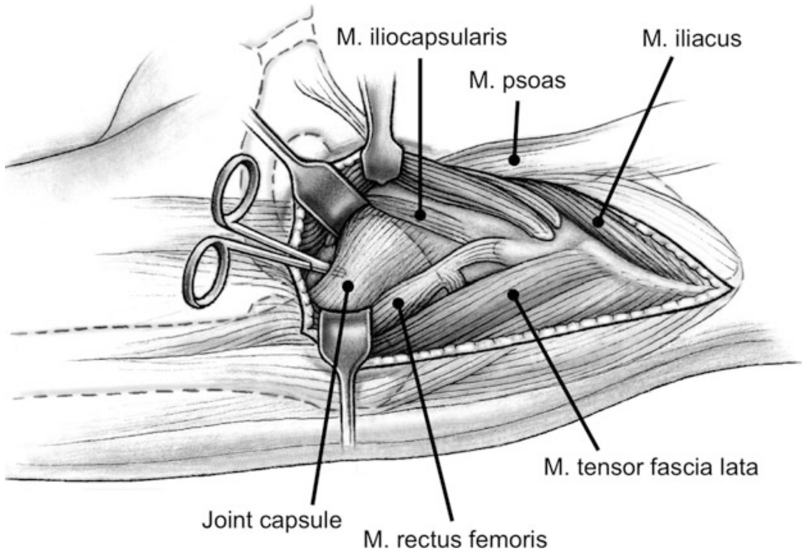


Fig. 5 The iliocapsularis muscle is dissected sharply from the anterior joint capsule, but its origin at the anterior inferior iliac spine (AIIS) is left intact. The iliocapsularis muscle is retracted medially. The psoas tendon is retracted medially with a Hohmann retractor which is brought into the superior pubic ramus medially to the eminentia iliopubica. The infra-articular space between the psoas tendon and the capsule is dissected bluntly with a pair of scissors until the tip of the instrument touches the base with the medial aspect of the ischial bone lateral to the obturator foramen

the musculature and the sciatic nerve during supra-acetabular und retroacetabular osteotomy. In addition, limited dissection allows to preserve the proximal remaining fibers of the origin of the tensor fascia muscle and the origin of the gluteus minimus containing the inferior branch of the superior gluteal artery providing blood supply to the supra-acetabular bone.

In total, four periacetabular osteotomies and a controlled retroacetabular fracture are performed to mobilize the acetabular fragment. The ischial osteotomy is done first (Fig. 6). It is performed with a special osteotome (Ganz osteotome) with a notched 15 mm blade that is 30° angulated towards the shaft. The osteotome is placed into the space between the distal joint capsule and the psoas tendon with the hip flexed and abducted to lateralize the position of the sciatic nerve (Fig. 6). The infracotyloid groove at the level of the inferior border of the acetabulum is palpated with the osteotome (Fig. 6). The correct position of the blade can be verified with an image intensifier. While the shaft of the osteotome points towards the patient's contralateral shoulder, the blade is hammered into the ischium to a depth of 2.5 cm. The shape of the ischial body is triangular with its base posterior. A complete osteotomy of the medial cortex is more important than of the thinner lateral cortex as the lateral cortex eventually breaks while the acetabular fragment is mobilized. The osteotome is carefully retrieved. Palpation with the osteotome assures whether the medial and lateral aspects of the ischial body are osteotomized correctly. The ischial osteotomy is incomplete to maintain the posterior column and the continuity of the pelvic ring (Fig. 6).

The second osteotomy is done through the medial aspect of the superior pubic ramus just medial to the eminentia pubica (Fig. 7). A Hohmann retractor is placed medially into the superior pubic ramus to retract the soft tissue medially and to protect the neurovascular structures (Fig. 7). The hip is flexed and adducted in order to relax the soft tissues. Subperiosteal placement of two blunt retractors around the origin of the superior pubic ramus helps to avoid damage to the adjacent obturator nerve and vessels. A complete, transverse osteotomy of the pubic ramus medially to the pubic eminentia is performed with 15 mm Lexer chisel (Fig. 7).

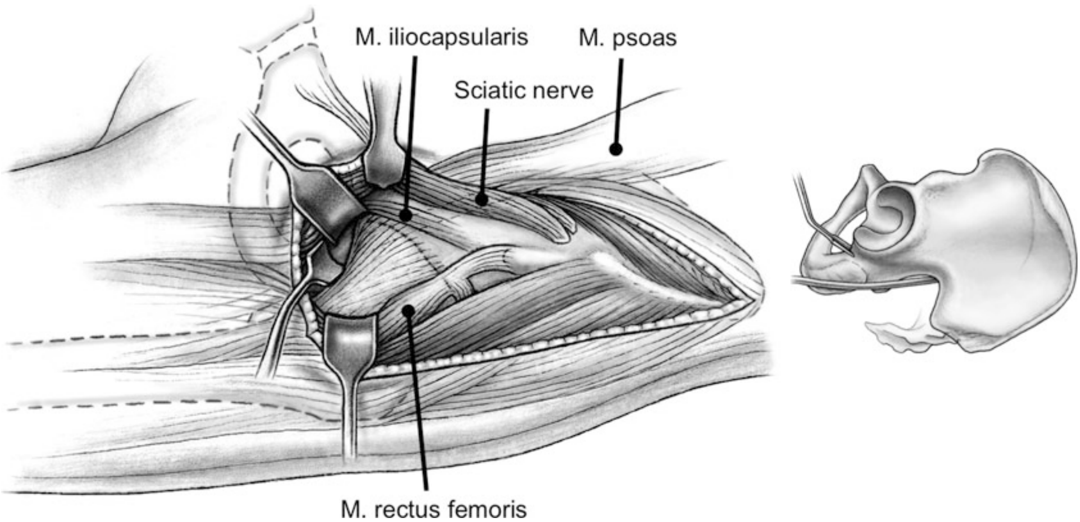


Fig. 6 The ischial osteotomy is performed with the Ganz osteotome which is placed in the infracotyloid groove. The hip is flexed and abducted to lateralize the position of the sciatic nerve. The shaft of the Ganz osteotome is pointed towards the patient's contralateral shoulder. The ischial osteotomy is incomplete to maintain the posterior column

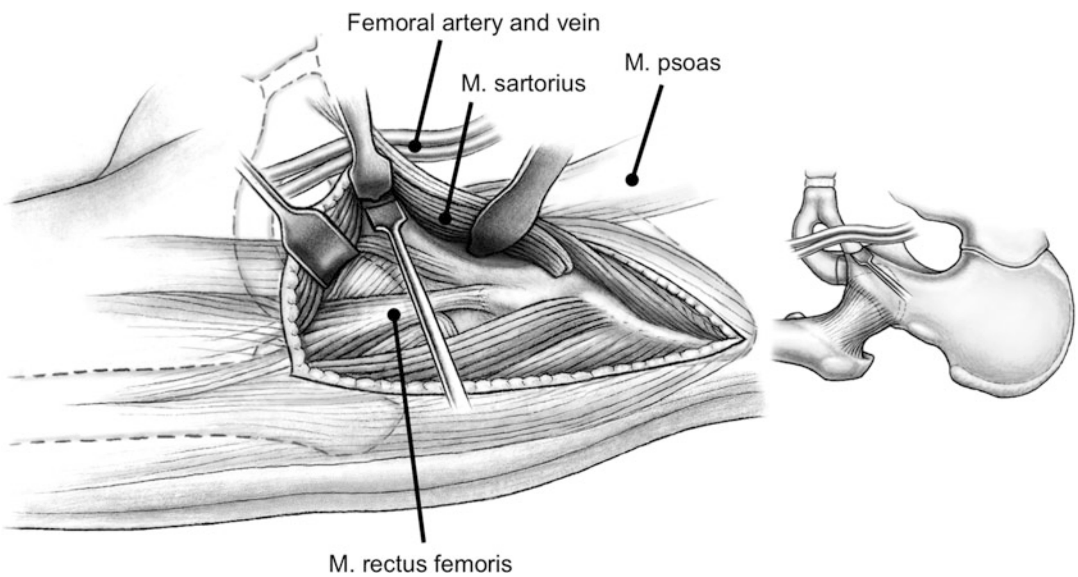


Fig. 7 The second osteotomy is the osteotomy of the superior pubic ramus just medial to the eminentia pubica. A Hohmann retractor is placed medially into the superior pubic ramus to retract the soft tissue medially and to protect the neurovascular structures. The osteotomy is performed with a Lexer chisel

The supra- and retroacetabular osteotomy are performed in two steps. First, the iliac bone is osteotomized with an oscillating saw (Fig. 8). The starting point is located at the lower margin of the anterior superior iliac spine. It is important to choose the entry point as cranially as possible to protect the gluteus minimus that encloses the supra-acetabular branch of the superior gluteal artery providing vascular supply to the acetabular fragment. In addition, sufficient supra-acetabular bone stock should be maintained for sufficient purchase of the Schanz screws for

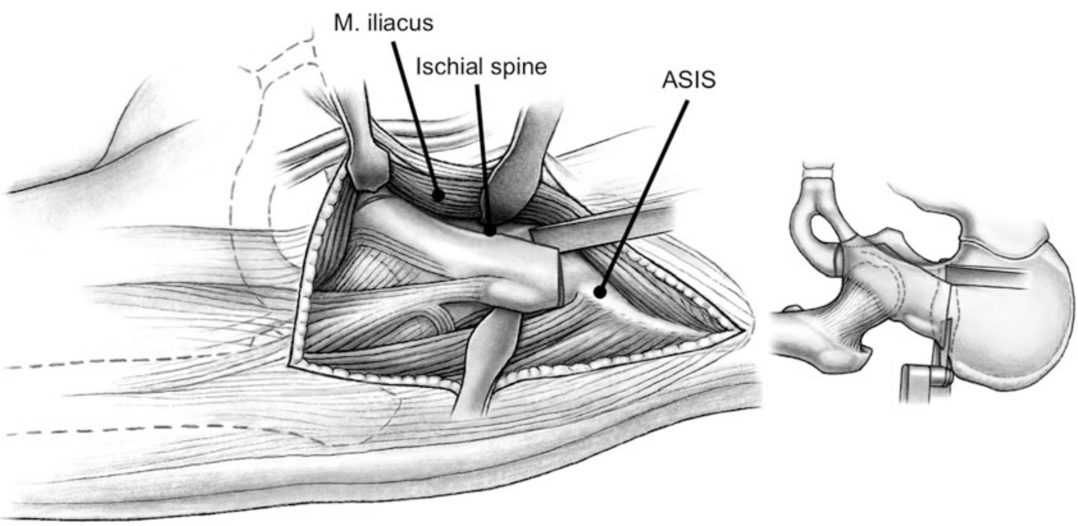


Fig. 8 To access the iliac bone, the aponeurosis of the abdominal muscles is sharply detached from the anterior third of the iliac crest. The iliac muscle is bluntly mobilized from the iliac bone. The supra-acetabular osteotomy is performed with an oscillating saw. The starting point is located at the lower margin of the anterior superior iliac spine (ASIS). The soft tissues on the inner and outer aspect of the ilium are protected by blunt retractors. This osteotomy ends about 2 cm lateral to the pelvic brim to leave the posterior column intact. At this point the osteotome is angled about 110° distally aiming at the tip of a blunt retractor which is placed on the inner aspect of the ischial spine

later mobilization and manipulation of the acetabular fragment. The soft tissue on the outer aspect of the ilium is protected by a blunt retractor. This osteotomy ends about 2 cm lateral to the pelvic brim. At this point the osteotome is angled about 110° distally aiming at the tip of a blunt retractor which is placed on the inner aspect of the ischial spine (Fig. 8). It is important that this retroacetabular osteotomy is performed through the inner aspect of the quadrilateral plate maintaining an osseous bridge of about 2 cm to the border of the greater sciatic notch. This will ensure the continuity of the posterior column. Ideally, the distal end of this osteotomy meets the previously performed incomplete cut of the ischial osteotomy. An image intensifier can be used to verify the extra-articular position and length of the retroacetabular osteotomy.

To mobilize the acetabular fragment, a 5 mm Schanz screw is driven into the supra-acetabular bone of the osteotomized fragment at the level of the anterior inferior iliac spine and pointing towards the vertex between the supra-acetabular and retroacetabular osteotomy (Fig. 9). Care must be taken not to penetrate the joint. A laminar spreader is positioned in the retroacetabular osteotomy and spread open. Moving the Schanz screw distally and medially helps to fracture the remaining bone bridges in a controlled fashion. Cutting of the remaining bone bridges can be facilitated by a Ganz osteotome which is placed 4 cm inferior of the linea terminalis at an angle of 50° to the quadrilateral surface aiming towards the endpoint of the first ischium osteotomy. The osteotome is carefully scored into the bone and can be transferred distally towards the end of the ischium osteotomy with careful protection of the adjacent sciatic nerve (Fig. 9). Decreasing resistance of the Schanz screw and loosening of the laminar spreader indicates yielding of the acetabular fragment. Before acetabular reorientation, the fragment must be completely free. This is best achieved by a counter-directed movement of the Schanz screws internally and the laminar spreader externally.

Unlike reorientation of the acetabular fragment in dysplastic hips, correction for acetabular retroversion is achieved by a combined extension

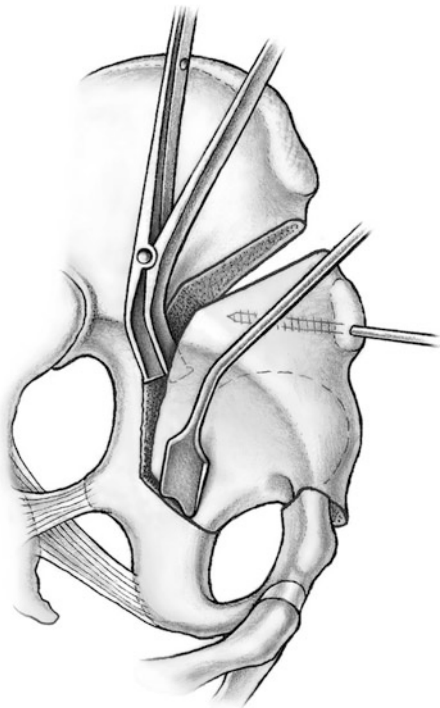


Fig. 9 To mobilize the acetabular fragment, a Schanz screw is driven into the supra-acetabular bone of the osteotomized fragment at the level of the anterior inferior iliac spine. A laminar spreader is positioned in the retroacetabular osteotomy. Cutting of the remaining bone bridges can be facilitated by a Ganz osteotome. Before acetabular reorientation, the fragment must be completely free. This is best achieved by a counter-directed movement of the Schanz screws internally and the laminar spreader externally

around the transverse axis and internal rotation around the longitudinal axis of approximately 10–20° each (Fig. 10). Increasing the angle of inclination of the acetabular fragment may add to prevent lateral overcoverage. Extension of the acetabular fragment is often limited due to a supra-acetabular impingement, requiring an additional supra-acetabular wedge resection of about 10° at the cranial aspect of the acetabular fragment with a lateral base of the wedge (Fig. 10). The goal of reorientation is an anteversion of the acetabulum without increasing the lateral coverage.

The reoriented acetabular fragment is temporarily fixed with K-wires. The quality of acetabular reorientation has to be evaluated by means of a correct intraoperative AP radiograph of the pelvis. The radiographic crossover sign should be eliminated (Fig. 11). Lateral acetabular coverage must not be increased excessively. This can be evaluated by the lateral center edge (LCE) angle which should be within a range of 23–33° and the acetabular index resulting within a range of 3–13° [30, 31]. The posterior wall sign should be negative (Fig. 11). However, it is important to avoid posterior overcoverage to prevent posterior impingement after reorientation. The amount of anterior and posterior acetabular coverage can be evaluated by the acetabular wall index [32]. The ischial spine sign does not change following acetabular reorientation since the ischial spine is not included in the osteotomies (Fig. 11). It is not uncommon that more than one attempt has to be made to achieve the desired degree of reorientation. After correct three-dimensional reorientation of the acetabular fragment, definitive fixation is performed with three cortical screws. Additional fixation of the acetabular fragment with a plate positioned on the inner aspect of the ileum is rarely necessary and only in cases with excessive internal rotation of the fragment.

After reorientation of the acetabular fragment, hip range of motion should be tested intraoperatively. A minimal internal rotation of 30° in 90° flexion should be achieved with a correctly anteverted acetabulum. Frequently, the source of limited internal rotation is a cam-type deformity of the femoral head (Fig. 12). In this case an anterior capsulotomy with osteochondroplasty with alternating medial or lateral retraction of the rectus femoris muscle should be performed (Figs. 12 and 13). Capsulotomy with osteochondroplasty was indicated in 92 % of our reported series of reverse PAO [17, 18]. The prominent head-neck offset can be trimmed with a chisel or high-speed burr after acetabular reorientation until impingement free range of motion up to 30° internal rotation in 90° flexion is achieved. This can be verified under direct visualization after the capsulotomy (Fig. 13).

After capsular closure the edges of the acetabular fragment can then be trimmed and the resected bone is interposed to fill up gaps of the

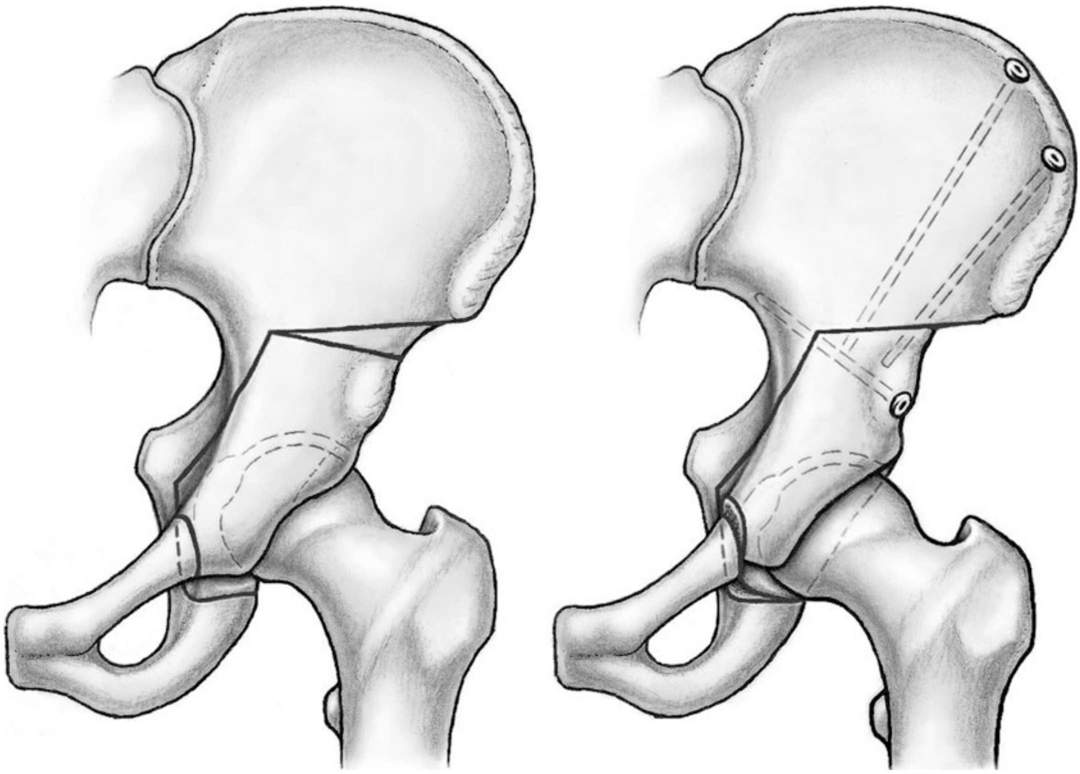


Fig. 10 Unlike reorientation of the acetabular fragment in dysplastic hips, correction for acetabular retroversion is achieved by a combined extension and internal rotation of the acetabular fragment of approximately 10–20° each. Extension of the acetabular fragment is often limited due to a supra-acetabular impingement, requiring an additional supra-acetabular wedge resection of about 10° at the cranial aspect of the acetabular fragment with a lateral base of the wedge

osteotomies and improve consolidation. The origins of the sartorius muscle with the attached inguinal ligament are reinserted with transosseous fixation. Suction drains are routinely positioned in the true pelvis.

Postoperative Regimen

After surgery, the operated leg is positioned in a neutral position in a soft splint. Suction drains are routinely removed after 48 h. Because of the preserved continuity of the pelvic ring, patients are early mobilized with crutches. Partial weight bearing with 15 kg on the operated side has to be maintained within the first 8 weeks after surgery. The hospital stay is around 5 days. Prophylaxis of deep venous thrombosis is performed until full weight bearing without crutches is reached. Continuous passive motion of the hip is performed from the first postoperative day to prevent capsular adhesions. Typically, stepwise increasing weight bearing is allowed when signs of callus formation are visible on radiographs 8 weeks postoperatively. Physiotherapy to improve range of motion and muscle strength is usually performed over a 2–3-month period. Additional femoral osteotomies usually do not change the postoperative regimen.

Results in Literature

The only long-term study of reverse PAO reported a favorable outcome with preservation of the native joint in all 29 hips [17, 18]. In 24 hips

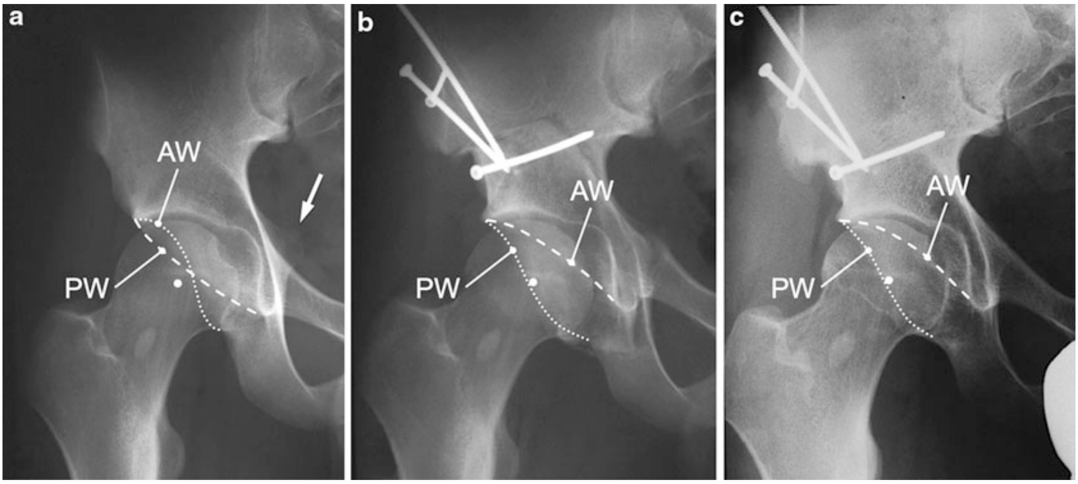


Fig. 11 (**a**) Twenty-nine-year-old male patient with acetabular retroversion indicated by a positive crossover sign (crossing of anterior [AW] and posterior [PW] acetabular wall), a positive posterior wall sign (posterior wall runs medial of femoral head center), and a positive ischial spine sign (*arrow*). (**b**) A reverse periacetabular osteotomy was performed and the crossover and posterior wall signs were eliminated. (**c**) At 8-year follow-up homogeneity and width of the joint space present unchanged

(83 %) a concomitant arthrotomy with offset correction was performed. Pain and function, including flexion and internal rotation, improved at a mean follow-up of 11 years. There was no significant increase in osteoarthritis. Correct acetabular orientation and, if necessary, a concomitant offset correction of the femoral head-neck junction were the keys for successful outcome. Overcorrection resulting in deficient anterior coverage and/or excessive posterior coverage was related to impaired outcome. In two hips with preoperatively a normal posterior wall (negative posterior wall sign), the reverse PAO resulted in an excessive posterior coverage and posterior impingement. These hips were treated with secondary surgical hip dislocation and posterior wall trimming. Therefore, the authors concluded that hips with acetabular retroversion and a normal posterior wall (negative posterior wall sign) are best treated with anterior rim trimming instead of acetabular reorientation. In addition, hips with untreated cam-type deformity were associated with an unfavorable outcome. Intraoperative evaluation of range of motion following acetabular reorientation and correction of the femoral head-neck offset is essential for good long-term outcome.

Summary

Reverse PAO is the causal surgical treatment for hips with acetabular retroversion. It is a technical demanding procedure that belongs in the hand of the well-experienced hip surgeon. Indication is based on a positive correlation among symptoms, physical findings, and radiographic signs for acetabular retroversion. Plain radiographs usually are sufficient for diagnosis, and a MR arthrography is only performed if uncertainties exist about chondrolabral damage of the joint. The reverse PAO is performed with four periacetabular osteotomies, some of them without direct visualization, and a controlled fracture. The quality of acetabular reorientation is evaluated by intraoperative pelvic radiographs. Concomitant capsulotomy with osteochondroplasty is performed in a majority of the cases. With a correct acetabular orientation and sufficient femoral head-neck offset, a good long-term result with

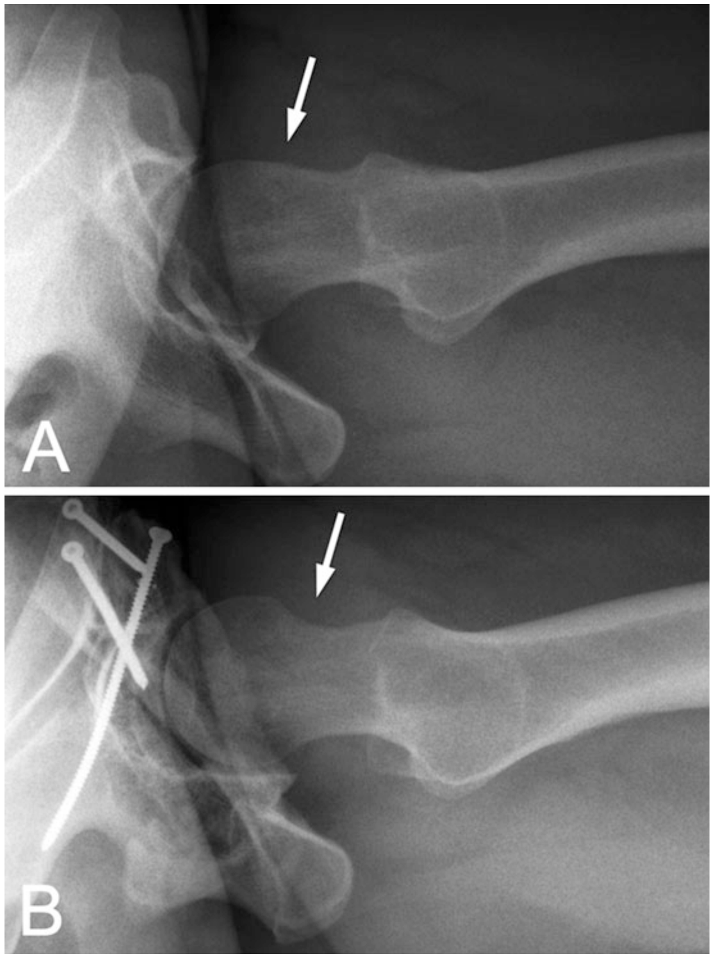


Fig. 12 (**a**) Hips with acetabular retroversion often are associated with an anterior cam-type deformity (*arrow*). (**b**) A reverse periacetabular osteotomy with concomitant anterior offset correction was performed. An untreated cam-type deformity can adversely affect the long-term outcome following acetabular reorientation [17, 20]

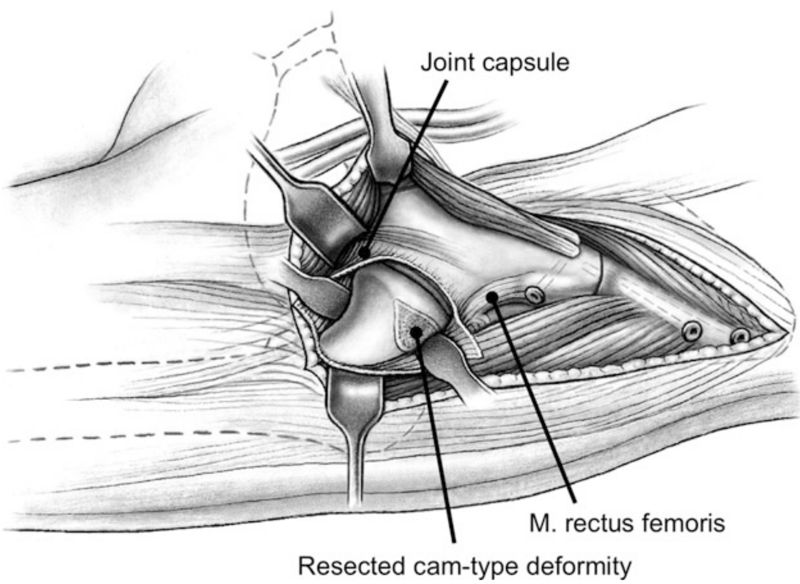


Fig. 13 After acetabular reorientation a minimal internal rotation of 30° in 90° of flexion should be achieved. Frequently, the source of limited internal rotation is a cam-type deformity of femoral head-neck area. An anterior capsulotomy with osteochondroplasty is performed with alternating medial or lateral retraction of the rectus femoris muscle. The prominent offset can be trimmed with a chisel or high-speed burr until impingement free range of motion up to 30° of internal rotation in 90° of flexion is achieved

preservation of the native joint for more than 10 years can be achieved [17, 18].

References

1. Reynolds D, Lucas J, Klaue K. Retroversion of the acetabulum. A cause of hip pain. J Bone Joint Surg Br. 1999;81(2):281–8. Epub 1999/04/16.
2. Ganz R, Parvizi J, Beck M, Leunig M, Notzli H, Siebenrock KA. Femoroacetabular impingement: a cause for osteoarthritis of the hip. Clin Orthop Relat Res. 2003;417:112–20. Epub 2003/12/04.
3. Giori NJ, Trousdale RT. Acetabular retroversion is associated with osteoarthritis of the hip. Clin Orthop Relat Res. 2003;417:263–9. Epub 2003/12/04.
4. Tannast M, Siebenrock KA, Anderson SE. Femoroacetabular impingement: radiographic diagnosis – what the radiologist should know. AJR Am J Roentgenol. 2007;188(6):1540–52. Epub 2007/05/23.
5. Beck M, Leunig M, Parvizi J, Boutier V, Wyss D, Ganz R. Anterior femoroacetabular impingement: part II. Midterm results of surgical treatment. Clin Orthop Relat Res. 2004;418:67–73. Epub 2004/03/27.
6. Siebenrock KA, Kalbermatten DF, Ganz R. Effect of pelvic tilt on acetabular retroversion: a study of pelvis from cadavers. Clin Orthop Relat Res. 2003;407: 241–8. Epub 2003/02/05.
7. Kalberer F, Sierra RJ, Madan SS, Ganz R, Leunig M. Ischial spine projection into the pelvis: a new sign for acetabular retroversion. Clin Orthop Relat Res. 2008;466(3):677–83. Epub 2008/02/12.
8. Li PL, Ganz R. Morphologic features of congenital acetabular dysplasia: one in six is retroverted. Clin Orthop Relat Res. 2003;416:245–53. Epub 2003/12/04.
9. Larson AN, Stans AA, Sierra RJ. Ischial spine sign reveals acetabular retroversion in Legg-Calve-Perthes disease. Clin Orthop Relat Res. 2011;469(7):2012–8. Epub 2011/02/01.
10. Dora C, Buhler M, Stover MD, Mahomed MN, Ganz R. Morphologic characteristics of acetabular dysplasia in proximal femoral focal deficiency. J Pediatr Orthop B. 2004;13(2):81–7. Epub 2004/04/13.
11. Kakaty DK, Fischer AF, Hosalkar HS, Siebenrock KA, Tannast M. The ischial spine sign: does pelvic tilt and rotation matter? Clin Orthop Relat Res. 2010;468 (3):769–74. Epub 2009/08/08.
12. Fujii M, Nakashima Y, Sato T, Akiyama M, Iwamoto Y. Pelvic deformity influences acetabular version and coverage in hip dysplasia. Clin Orthop Relat Res. 2011;469(6):1735–42. Epub 2011/01/05.
13. Jamali AA, Mladenov K, Meyer DC, Martinez A, Beck M, Ganz R, et al. Anteroposterior pelvic radiographs to assess acetabular retroversion: high validity of the “cross-over-sign”. J Orthop Res. 2007;25 (6):758–65. Epub 2007/03/09.
14. Kohnlein W, Ganz R, Impellizzeri FM, Leunig M. Acetabular morphology: implications for joint-preserving surgery. Clin Orthop Relat Res. 2009;467 (3):682–91. Epub 2009/01/09.
15. Tannast M, Pfannebecker P, Schwab JM, Albers CE, Siebenrock KA, Buchler L. Pelvic morphology differs in rotation and obliquity between developmental dysplasia of the hip and retroversion. Clin Orthop Relat Res. 2012;470(12):3297–305. Epub 2012/07/17.
16. Ganz R, Klaue K, Vinh TS, Mast JW. A new periacetabular osteotomy for the treatment of hip dysplasias. Technique and preliminary results. Clin Orthop Relat Res. 1988;232:26–36. Epub 1988/07/01.
17. Siebenrock KA, Schaller C, Tannast M, Keel M, Büchler L. 10-year follow-up after anteverting periacetabular osteotomy for symptomatic acetabular retroversion. J Bone Joint Surg Am. 2014;submitted (submitted):submitted. doi:10.2106/JBJS.M.00842
18. Siebenrock KA, Schoeniger R, Ganz R. Anterior femoro-acetabular impingement due to acetabular retroversion. Treatment with periacetabular osteotomy. J Bone Joint Surg Am. 2003;85-A(2):278–86. Epub 2003/02/07.
19. Tannast M, Zheng G, Anderegg C, Burckhardt K, Langlotz F, Ganz R, et al. Tilt and rotation correction of acetabular version on pelvic radiographs. Clin Orthop Relat Res. 2005;438:182–90. Epub 2005/09/01.
20. Albers CE, Steppacher SD, Ganz R, Tannast M, Siebenrock KA. Impingement adversely affects 10-year survivorship after periacetabular osteotomy for DDH. Clin Orthop Relat Res. 2013;471 (5):1602–14. Epub 2013/01/29.
21. Leunig M, Werlen S, Ungersbock A, Ito K, Ganz R. Evaluation of the acetabular labrum by MR arthrography. J Bone Joint Surg Br. 1997;79(2): 230–4. Epub 1997/03/01.
22. Murphy RJ, Subhawong TK, Chhabra A, Carrino JA, Armand M, Hungerford M. A quantitative method to assess focal acetabular overcoverage resulting from pincer deformity using CT data. Clin Orthop Relat Res. 2011;469(10):2846–54. Epub 2011/07/13.
23. Murphy SB, Simon SR, Kijewski PK, Wilkinson RH, Griscom NT. Femoral anteversion. J Bone Joint Surg Am. 1987;69(8):1169–76. Epub 1987/10/01.
24. Ecker TM, Tannast M, Puls M. Computer assisted Orthopaedic Surgery. In: Jolesz F, editor. Intraoperative Imaging and Image-Guided Therapy. New York: Springer; 2014;1:661–675.
25. Kamegaya M, Saisu T, Nakamura J, Murakami R, Segawa Y, Wakou M. Drehmann sign and femoro-acetabular impingement in SCFE. J Pediat Orthop. 2011;31(8):853–7. Epub 2011/11/22.
26. Tönnis D. General radiography of the hip joint. In: Tönnis D, editor. Congenital dysplasia, dislocation of the hip. New York: Springer; 1987. p. 100–42.
27. Notzli HP, Wyss TF, Stoecklin CH, Schmid MR, Treiber K, Hodler J. The contour of the femoral head-neck junction as a predictor for the rick of anterior

impingement. J Bone Joint Surg Br. 2002;84(4): 556–60.
28. Siebenrock KA, Steppacher SD, Haefeli PC, Schwab JM, Tannast M. Valgus hip with high antetorsion causes pain through posterior extraarticular FAI. Clin Orthop Relat Res. 2013;471:3774–80. Epub 2013/03/07.
29. Smith-Petersen MN. Approach to and exposure of the hip joint for mold arthroplasty. J Bone Joint Surg Am. 1949;31(1):40–6. Epub 1949/01/01.
30. Tannast M, Kakaty DK, Zheng G, Siebenrock KA. What is a normal acetabulum? A computer-assisted radiographic study. In: Davies BL, Joskowicz L, Murphy SB, editors. 9th annual meeting of CAOS-international proceedings. Berlin: Pro Business; 2009. 5 p.
31. Tannast M, Albers CE, Steppacher SD, Siebenrock KA. Hip pain in the young adult. In: Bentley G, editor. European Instructional Lectures. Berlin: Springer; 2011:141–154.
32. Siebenrock KA, Kistler L, Schwab JM, Buchler L, Tannast M. The acetabular wall index for assessing anteroposterior femoral head coverage in symptomatic patients. Clin Orthop Relat Res. 2012;470(12): 3355–60. Epub 2012/07/17.

Acetabular Protrusion and Surgical Technique

48

Michael Leunig and Reinhold Ganz

Contents

M. Leunig (✉)
Department of Orthopaedic Surgery, Schulthess Clinic, Zürich, Switzerland
e-mail: michael.leunig@kws.ch

R. Ganz
Emeritus Inselspital Bern, Bern, Switzerland

Faculty of Medicine, University of Bern, Bern, Switzerland
e-mail: rd.ganz@bluewin.ch

Abstract

Acetabular protrusion is a severe hip deformity and remains even today difficult to treat, due to the complexity of deformities and the early occurrence of hip osteoarthritis (OA). This chapter outlines an algorithmic approach to the surgical treatment of acetabular protrusion. Individual treatment plans need to be based upon the entire clinical presentation including the appreciation of all skeletal deformities and the degree of OA. In this technical report, the focus is on protrusion in the young adult after closure of the growth plate and in the absence of advanced OA. The most severe protrusion not only reveals global acetabular overcoverage (pincer impingement), but can occur with a high-riding trochanter and even a negatively tilted acetabular roof with medially shifting femoral head. Surgical treatment needs to be tailored according to the presented deformities and usually begins with a surgical hip dislocation to address pincer impingement including labral reconstruction. Concomitant procedures such as relative neck lengthening for trochanteric advancement and periacetabular and even intertrochanteric osteotomies might become necessary to normalize joint mechanics. The amount of applied surgery needs to be well balanced with respect to age and symptoms of the patients, since the outcome will be largely determined by the preexisting OA of these hips. Minimally invasive surgical methods such as hip arthroscopy are unable to cope with structural deformities and should be carefully considered.

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_51

Introduction

The pathomorphology of an acetabulum protruding into the true pelvis was first described by Otto in 1816 [1]. The consequence of acetabular protrusion mainly affecting women [2] is secondary osteoarthritis (OA) and has been characterized by a loss of medial and posterior joint space, while the craniolateral (superior) joint space remains largely unaltered even until end-stage disease. The mechanism has been explained by higher load transmission through the medial aspect of the joint [3, 4]. Even in minimal primary protrusion, the femoral head has been observed to migrate medially over time due to medial dysplasia. Potentially even more important for OA development, a direct abutment of the femoral neck against the acetabular rim called "pincer impingement" occurs in protrusion causing direct damage to the labrum and acetabular cartilage and indirect posteroinferior cartilage damage by leverage [5]. The latter is visible on a false profile radiograph or by MRI but not on anteroposterior (AP) pelvic radiograph.

During the first 100 years after Otto's first description, the interest in hip protrusion was focused on etiology and classification, while treatment recommendations developed only more recently. For the skeletally immature hip, surgical closure of the triradiate cartilage has been proposed [6]. However, this approach has not been widely adopted, in part because of the inability to predict which hips will undergo disease progression. For the young adult under the age of 40 years without the presence of arthritic changes, valgus intertrochanteric osteotomy has been the recommended treatment option for a long time [7–9]; today it is understood that the resulting medialization of the femur increases the risk for posterior impingement. The standard surgical treatment for the middle and older age with evidence of osteoarthritis is total hip replacement (THR), while resection arthroplasty and even arthrodesis are considered to be historical treatment options. Anterior acetabuloplasty was first anecdotally performed by Smith-Petersen [10] to increase motion in older patients with marked stiffness, but this approach has not become a clinical routine.

Table 1 Algorithmic surgical approach to protrusion

Protrusion in young adults without OA	
Deformities	Treatment
(i) Global overcoverage	Rim trim ± labral refixation/ reconstruction
(ii) High trochanter	Neck lengthening + trochanteric advancement
(iii) High fossa with negative roof angle	PAO
(iv) Medial shift of femoral head[a]	Intertrochanteric valgization osteotomy

[a]If an intertrochanteric osteotomy is performed, neck lengthening (ii) is not required

The appropriate treatment must be tailored upon the age and skeletal maturity of the patient, the degree of protrusion, and the extent of degenerative changes of the hip. Patient selection remains challenging, since adaptive and arthritic alterations can be present in young patients, and the amount of required surgery to address deformities might be extensive. Severe protrusion might not only reveal acetabular overcoverage (pincer impingement) but also a high-riding trochanter, a negatively tilted acetabular roof, and even a medially shifted femoral head requiring concomitant bony procedures (Table 1). Despite anecdotal reports on the use of hip arthroscopy [11], minimally invasive surgical options cannot address the complexity of structural deformities and therefore should be not considered as a curative approach [12–14]. In this technical report, the focus is on open surgery for the treatment of protrusion in the young adult after closure of the growth plate and in the absence of advanced OA.

Surgical Technique

The technique of surgical hip dislocation (SHD) is the workhorse to address acetabular overcoverage in protrusion and is covered in detail in the literature [15] and in Chap. 51, "Surgical Technique: Open Acetabular Rim Trimming, Labral Refixation, and Open Femoral Osteochondroplasty." Based upon coexisting deformities, relative neck lengthening with trochanteric advancement [16, 17], periacetabular osteotomy (PAO) [18], and intertrochanteric valgus osteotomy are potential concomitant procedures that may become necessary. Since all of these procedures have been described in the literature, only critical aspects relevant to the treatment of protrusion will be covered.

Surgical Hip Dislocation

To address acetabular overcoverage (pincer impingement), surgery usually starts with an SHD. The first important aspect to appreciate during SHD is that protrusion hips can reveal a relatively high-riding greater trochanter; oftentimes relative lengthening of the femoral neck and distal advancement of the greater trochanter is required to increase the clearance on the femoral side. In such instances, a classic trochanteric slide osteotomy as originally described [15] should be preferred over the more recently introduced step-cut osteotomy. The latter one provides better mechanical conditions after refixation [19, 20] but does not allow to distally advance the trochanter. Another consideration in hips revealing a high-riding trochanter is that the gluteus minimus muscle extends distally under the piriformis muscle, which might make the dissection off the capsule more difficult. The remaining dissection of the capsule and capsulotomy is not different from standard surgical dislocations.

Head Dislocation and Rim Trimming

The second critical step is the dislocation of the femoral head itself, which, similar to THR, is more difficult in protrusion and has to be performed carefully so as not to forcefully deliver the femoral head. When performed after relative neck lengthening, external rotation is increased and facilitates anterior dislocation. In the authors' experience, rim resection prior to dislocation had never to be performed, but might be considered. Technically, however, rim resection with the dislocated head will allow for a more precise assessment and conduction of the rim resection. The third important consideration is the size of horseshoe or acetabular articular surface. The fact that there is global overcoverage does not mean that excess articular cartilage is present. In fact, often the opposite is true. The acetabular fossa in patients with protrusion is typically enlarged and runs very high (negative roof tilt), and the horseshoe may even be narrowed (Table 1). In such patients, especially with a negative roof tilt, conservative trimming of the acetabular rim is indicated, and a PAO needs to be considered to reorient and conserve the horseshoe. The head neck junction in protrusion frequently shows an indentation line (rather than a cam deformity) at the head neck junction which typically is more peripheral than the classic cam deformity. Additional femoral neck osteoplasty does not significantly improve the situation, but might be considered. Adaptive head neck changes on the posterior neck also have to be considered and if present should be addressed. These are sometimes ossifications at the head neck junction that can be found as a result of chronic neck abutment.

Labral Refixation/Reconstruction

Although sufficient coverage after rim trimming might not demand labral preservation, it is the authors' recommendation to preserve the native labrum, if it is of sufficient quality [21]. However, chronic periosteal irritation followed by bone apposition and overgrowth of the labrum might have stretched the labrum with a resultant thinning impairing the sealing function especially when the acetabular rim had been surgically removed and the remnants of the labrum are fixed to the acetabular bone [22]. In these cases, circumferential labral reconstruction [23] might become necessary as described in another chapter (Chap. 97, "Surgical Technique: Open Hip HS Allograft"). Usually, multiple bone anchors between 10 and 15 mm apart are required for circumferential refixation (Fig. 1).

Relative Neck Lengthening and Trochanteric Advancement

If not already performed to facilitate the dislocation, trimming of the stable trochanter for relative lengthening may be executed after addressing

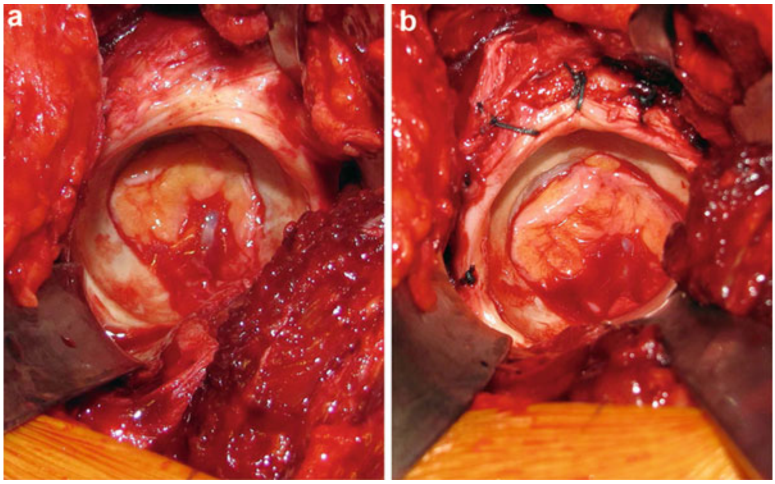


Fig. 1 (**a**) The acetabular fossa in patients with protrusion is typically enlarged and runs very high, and the horseshoe may even be narrowed. (**b**) In such patients, especially with a negative Tönnis angle, conservative trimming of the acetabular rim is indicated

intraarticular impingement. The gain in range of motion might minimize the potential for pelvi-trochanteric impingement, specifically, in hips with a high trochanter. In these hips, relative neck lengthening with trochanteric advancement can improve muscular biomechanics and resolve the extra-articular impingement. The described technique is called "relative neck lengthening," because only the superior circumference of the neck becomes longer by the trimming away of most of the trochanteric portion in its posterosuperior circumference, such that it becomes continuous with the smaller contour of the neck [16, 17]. Leg length remains the same, but muscle biomechanics and clearance for hip motion is improved [24]. At the end of the procedure, the distal advancement of the trochanteric fragment is facilitated by a release of the long head of the gluteus minimus muscle, which runs along the anterior border of the mobile trochanteric fragment. The definitive cephalad-caudad position of the trochanteric fragment, and therefore the amount of the trochanteric advancement, is aimed to distalize the tip of the trochanter to the level of the center of rotation of the femoral head avoiding overdistalization (Fig. 2).

Concomitant PAO/ITO: If it is anticipated that rim trimming alone will not leave enough cartilage, concomitant periacetabular osteotomy (PAO) becomes necessary. The PAO is done during the same surgical session after repositioning of the patient from lateral decubitus to supine. During the first step of the procedure, with the patient in a lateral decubitus position, the first incomplete cut of the PAO of the ischium can be made with direct visual observation of the sciatic nerve [25]. All remaining cuts are done through the modified Smith-Petersen approach as described previously [18]. The goal for the correction is to lateralize the acetabular fragment and rotate it toward the midline (clockwise in a right hip, counterclockwise in a left hip) so that the roof angle and the acetabular version become normal. This is a technically difficult procedure, and the proximal displacement of the acetabular fragment can put the femoral nerve at risk of injury. As the femoral nerve courses with the iliopsoas tendon, these structures become draped over the step created by the rotation of the fragment, and the nerve is susceptible to a tension injury. Direct observation of the nerve and optimal rotation of the fragment are possible with an inguinal extension of the Smith-Petersen approach, allowing a ball spike to be placed onto the pubic portion of the acetabular fragment [26]. In this manner, the formation of a medial step is prevented, while the osteotomized fragment is rotated with a supra-acetabular Schanz screw. If the femur is in varus with a medial shift the femoral head, an intertrochanteric valgus osteotomy may be considered, performed though the SHD approach. This can be performed through the same lateral incision used for the surgical dislocation using a blade plate.

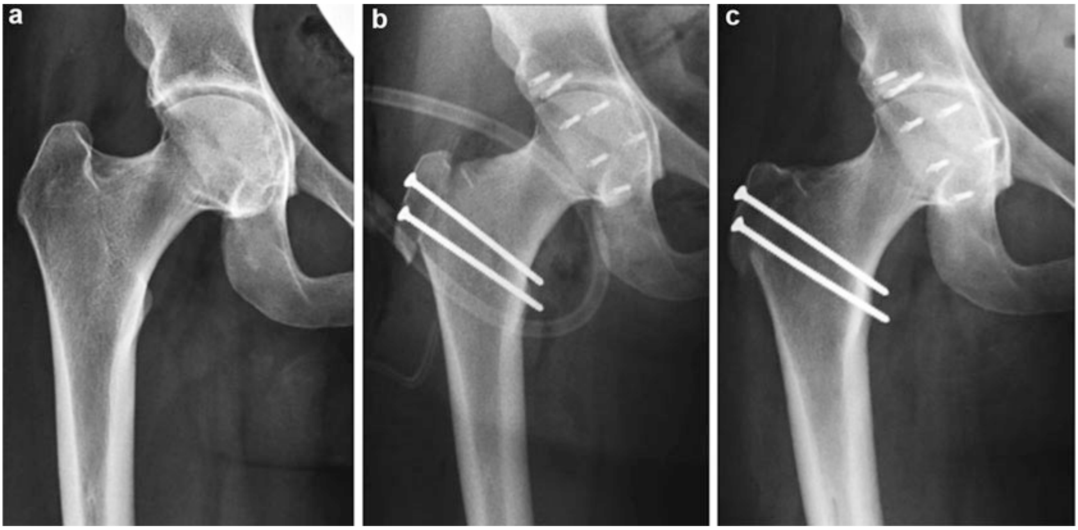


Fig. 2 (**a**) A twenty-year-old female with acetabular protrusion and high-riding trochanter and severe pain. The roof tilt is only mildly negative, but the femoral head remains centered. (**b**) A surgical dislocation was performed with an acetabular rim trimming, labral reconstruction using a hamstring allograft combined with a relative femoral neck lengthening and trochanteric advancement. (**c**) Three months later, the trochanter is united and the patient fully weight bearing, and pain has completely subsided

The difficulty is that after a surgical dislocation, the trochanteric osteotomy needs to be fixed before blade insertion. The addition of an intertrochanteric osteotomy to a periacetabular osteotomy has been quite rare in our experience. If a relative neck lengthening is considered, valgus intertrochanteric osteotomy has never been considered.

Summary

Acetabular protrusion remains a severe hip deformity, and even today, treatment options are limited due to the complexity of deformities and the early occurrence of hip OA. This chapter proposes a simplified algorithmic approach to the surgical treatment of acetabular protrusion. Surgical hip dislocation is the workhorse of the surgical treatment, while less invasive surgical techniques are incapable to address the existing deformities. Individual treatment plans need to be based upon the entire clinical picture of each patient including the appreciation of all skeletal deformities and degree of OA.

References

1. Otto AW. Ein Becken, mit kugelformig augedehnten Pfannen. In: Heft E, editor. Seltene Beobachtungen zur Anatomie, Physiologie und Pathologie gehorig, vol. IX. Breslau: Verlag Willibald August Holaufer; 1816. p. 19–20.
2. Gilmour J. Adolescent deformities of the acetabulum: an investigation into the nature of protrusio acetabuli. Br J Surg. 1939;26:670–99.
3. Crowninshield RD, Brand RA, Pedersen DR. A stress analysis of acetabular reconstruction in protrusio acetabuli. J Bone Joint Surg Am. 1983;65:495–9.
4. Maquet P. Biomechanics of the hip. Berlin: Springer; 1985.
5. Ganz R, Parvizi J, Beck M, Leunig M, Notzli H, Siebenrock KA. Femoroacetabular impingement: a cause for osteoarthritis of the hip. Clin Orthop Relat Res. 2003;417:112–20.
6. Steel HH. Protrusio acetabuli: its occurrence in the completely expressed Marfan syndrome and its musculoskeletal component and a procedure to arrest the course of protrusion in the growing pelvis. J Pediatr Orthop. 1996;16:704–18.
7. Dunlop CC, Jones CW, Maffulli N. Protrusio acetabuli. Bull Hosp Jt Dis. 2005;62:105–14.
8. McBride MT, Muldoon MP, Santore RF, Trousdale RT, Wenger DR. Protrusio acetabuli: diagnosis and treatment. J Am Acad Orthop Surg. 2001;9:79–88.

9. Rosenmeyer B, Viernstein K, Schuhmann HJ. Mittelfristige ergebnisse der valgisierenden und medialisierenden intertrochanteren osteotomie mit verkurzung des coxalen femurendes bei der primaren protrusio acetabuli. Arch Orthop Unfall Chir. 1973;77:138–48.
10. Smith-Petersen MN. Treatment of malum coxae senilis, old slipped upper femoral, intrapelvic protrusion of the acetabulum, and coxa plana by means of acetabuloplasty. J Bone Joint Surg Am. 1936;18:869–80.
11. Matsuda DK. Protrusio acetabuli: contraindication or indication for hip arthroscopy? And the case for arthroscopic treatment of global pincer impingement. Arthrosc J Arthrosc Relat Surg. 2012;28:882–8.
12. Leunig M, Huff TW, Ganz R. Femoroacetabular impingement: treatment of the acetabular side. Instr Course Lect. 2009;58:223–9.
13. Leunig M, Nho S, Tuchetto L, Ganz R. Protrusio acetabuli: new insights and experience with joint preservation. Clin Orthop Relat Res. 2009;467:2241–50.
14. Larson C. Protrusio acetabuli: some questions. Arthrosc J Arthrosc Relat Surg. 2012;28:1193–4.
15. Ganz R, Gill TJ, Gautier E, Ganz K, Krugel N, Berlemann U. Surgical dislocation of the adult hip a technique with full access to the femoral head and acetabulum without the risk of avascular necrosis. J Bone Joint Surg (Br). 2001;83:1119–24.
16. Ganz R, Huff TW, Leunig M. Extended retinacular soft tissue flap for intraarticular hip surgery. Instr Course Lect. 2009;58:241–55.
17. Leunig M, Ganz R. Relative neck lengthening and intracapital osteotomy for severe Perthes and Perthes-like deformities. Bull NYU Hosp Jt Dis. 2011;69 Suppl 1:S62–7.
18. Ganz R, Klaue K, Vinh TS, Mast JW. A new periacetabular osteotomy for the treatment of hip dysplasias. Technique and preliminary results. Clin Orthop Relat Res. 1988;232:26–36.
19. Schöniger R, Lafrance A, Oxland TR, Guy P, Ganz R, Leunig M. Does trochanteric step osteotomy provide better stability than classic slide osteotomy: a preliminary study. Clin Orthop Relat Res. 2009;467:775–82.
20. Bastian JD, Wolf AT, Wyss TF, Notzli HP. Stepped osteotomy of the trochanter for stable, anatomic refixation. Clin Orthop Relat Res. 2009;467:732–8.
21. Tibor L, Leunig M. Systematic review: treatment of hip labrum pathology: resection vs labral preservation. HSS J. 2012;8:225–9.
22. Corten K, Ganz R, Chosa E, Leunig M. Bone apposition of the acetabular rim in deep hips: a distinct finding of global pincer impingement. J Bone Joint Surg Am. 2011;93S:10–6.
23. Costa Rocha P, Klingenstein G, Ganz R, Kelly BT, Leunig M. Complete allograft reconstruction of the acetabular labrum: case series and surgical technique. Hip Int 2013;23:S42–53.
24. Eijer H, Podeszwa DA, Ganz R, Leunig M. Evaluation and treatment of young adult with femoroacetabular impingement secondary to Perthes' disease. Hip Int. 2006;16:273–80.
25. Leunig M, Rothenfluh D, Beck M, Nork SE, Gautier E, Kerboull M, Ganz R. Surgical dislocation and periacetabular osteotomy through a posterolateral approach: a cadaveric feasibility study and initial clinical experience. Op Tech Orthop. 2004;14:4957.
26. Letournel E. The treatment of acetabular fractures through the ilioinguinal approach. Clin Orthop Relat Res. 1993;292:62–76.

49 Femoral Deformities: Varus, Valgus, Retroversion, and Anteversion

Robert L. Buly

Contents

R.L. Buly
Hospital for Special Surgery, Surgical Arthritis and Hip Preservation Services, Weill Medical College of Cornell University, New York, NY, USA
e-mail: bulyr@hss.edu

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_52

Abstract

Osteoarthritis of the hip is almost always due to a structural abnormality. These abnormalities may include any of the following, often in bewildering combinations: acetabular dysplasia, femoroacetabular impingement (FAI), coxa valga, coxa vara, and retroversion or excessive anteversion of the femur or acetabulum. Femoral osteotomy is a powerful tool to correct these deformities. The most commonly employed types are varus or valgus intertrochanteric, with or without derotation. Extension or flexion of the intertrochanteric osteotomy may be added if it is desirous to change the head position in the sagittal plane. Pure derotation osteotomies may be preformed alone if abnormal version is the sole deformity.

Historically, femoral osteotomies were employed in a "salvage" mode for pain relief. Since the advent of total hip arthroplasty, femoral osteotomy is now best suited to a "hip preservation" mode. It is important to determine which of these abnormalities may be present, with the realization that it may require additional radiographic studies, such as CT scans, to make the correct diagnosis. This is especially important with version abnormalities, which can be very difficult to diagnose on plain radiographs. A thorough physical examination is critical to assess range of motion, leg length, and the foot-progression angle, since these may all be altered with a corrective osteotomy. It is important to remember that correcting one deformity (such as excessive femoral anteversion) may exacerbate another, such as FAI.

When correctly executed, femoral osteotomy can provide long-term pain relief and functional improvement. The procedure may also obviate the necessity of total hip arthroplasty, especially if performed before the onset of significant articular cartilage damage.

Introduction

With the increasing understanding of conditions such as acetabular dysplasia, coxa valga, version abnormalities of the femur and acetabulum, and femoroacetabular impingement (FAI), only a small minority of cases can now be classified as "idiopathic" osteoarthritis of the hip. These conditions may occur in isolation or may occur together in a bewildering combination. Hip impingement, whatever the cause, proceeds along the final common pathway of labrum and articular cartilage damage, ultimately leading to osteoarthritis if uncorrected. Conditions such as acetabular dysplasia and femoral anteversion are usually associated with hip instability, shear damage to the labrum, and articular cartilage overload [1–7].

It is incumbent upon the orthopedic surgeon performing hip preservation surgery to not only correctly diagnose the underlying problem but also to perform the correct anatomical correction to relieve pain, improve function, and hopefully prolong the life of the hip.

The History of Femoral Osteotomy

Pauwels has documented the inevitable progression of osteoarthritis in untreated dysplasia [8]. Efforts have been made by surgeons since then to intervene to halt this disturbing progression. Dysplasia has certainly been the most obvious deformity and efforts have been made to treat the deformity on both the acetabular and femoral sides of the joint. The Bernese periacetabular osteotomy (PAO) has evolved and become the most widely used procedure to treat acetabular dysplasia [9]. In the era proceeding total hip arthroplasty (1959–1960), femoral osteotomy was used in a "preserving" as well as "salvage" mode. Voss (1956) described a "hanging hip" procedure where the circumferential muscles of the hip joint were sectioned in an effort to lower joint reaction forces and relieve pain. A trochanteric osteotomy, left unfixed, was usually included [10].

McMurray in 1939 described a medial displacement intertrochanteric osteotomy. The idea was to unload the forces in the hip joint by transferring weight-bearing from the ischium to the femoral shaft [11].

Pauwels proposed using either varus or valgus producing intertrochanteric osteotomies as an alternative to improve joint biomechanics [12–14]. These techniques were further advanced by Swiss surgeons such as Mueller and Schneider, who not only standardized the techniques but developed implants and instrumentation that facilitated the operations [15, 16].

Pauwels and Bombelli proposed with the use of a valgus-producing intertrochanteric osteotomy in which the head was reoriented to include a "capital drop" osteophyte in the weight-bearing process to make the joint surface more congruent [13, 17, 18].

Derotation osteotomies have evolved along with varus or valgus producing procedures because of the coexisting deformities of either excessive anteversion or femoral retroversion. Isolated derotation osteotomies have been employed in the treatment of posttraumatic deformities following intramedullary nailing [19–22]. Increasingly, this technique is being used to treat naturally occurring isolated deformities of femoral version where it may not be necessary to alter the neck-shaft angle.

Patient History

The underlying condition leading to these abnormalities may have been present since birth (breech birth, prematurity, congenital dislocation) or maybe due to some dramatic event that occurs later in life (infection, Perthes disease, slipped capital femoral epiphysis). A limp may be present constantly or might only occur after prolonged walking. Femoral version abnormalities will often lead to an unusual foot-progression angle. Patients with excessive femoral anteversion will usually report a history of an in-toeing gait, which can be accentuated with increasing pain or fatigue. Patients with femoral retroversion will usually report an out-toeing type of gait. Because of the excessive external rotation of the hip associated with femoral retroversion, these patients are usually more comfortable sitting cross-legged on the floor. In contrast, patients with excessive femoral anteversion are often more comfortable sitting "W style" with the hips flexed and internally rotated. Patients with femoroacetabular impingement will often report that their hips have always had restricted range of motion compared to their peers. There may be a history of concomitant knee or ankle pain. Some patients report difficulty with bicycle riding because the knees and ankles seemed to be moving in discordant planes. Patients with labral tears will often report "mechanical" symptoms: locking, catching, giving way, or a sharp pain with certain movements, especially flexion and rotation. As hip joint deterioration progresses, patients may report diminished walking ability.

Physical Examination

In addition to a thorough history, a detailed physical examination is critical. It is often helpful to exam the gait of the patient. There may be an antalgic component, a Trendelenburg limp indicative of abductor weakness, both, or no limp at all. The foot-progression angle should also be noted. Patients with excessive femoral anteversion will usually have an in-toeing gait. At times, there may be a compensatory external tibial torsion along with the excessive femoral anteversion in which the patellae will squint inward but the foot-progression angle is straight or even externally rotated. In contrast, patients with femoral retroversion will usually walk with an external foot-progression angle, which at times may be marked [23]. Careful note should be made of the leg lengths. Certain conditions such as slipped capital femoral epiphysis (SCFE), Perthes disease, infantile growth arrest of the proximal physis, and coxa vara will usually cause shortening of the involved extremity. The range of motion should be measured carefully. If the overall flexion arc is less than 90°, degenerative changes may be too advanced to make an osteotomy a reasonable option. Since varus and valgus intertrochanteric osteotomies are essentially abduction and abduction osteotomies, respectively, there should be adequate motion remaining in the opposing axis. Intra-articular pain may prohibit an accurate measurement. For example, a painful impingement test at 90° of flexion may mask the real degree of internal rotation, there may not be an actual bony block. It is often helpful to measure the motion if the patient has received an intra-articular injection. Intra-articular injections are also extremely valuable in determining whether the source of pain is intra-articular or extra-articular.

Rotation of the femur can be measured in both flexion and extension, but impingement is usually much more notable with the hip flexed. If there is femoroacetabular impingement, internal rotation at 90° of hip flexion is usually limited or even completely absent. This may be true no matter which anatomic defect is causing the impingement [24]. On the other hand, patients with excessive femoral anteversion will usually have more internal rotation than external rotation. In certain cases, external rotation may be completely lacking, making it difficult for patient to sit cross-legged on the floor.

Patients with excessive femoral anteversion will often have a positive apprehension test when the leg is extended and externally rotated. This is due to a feeling of subluxation that is accentuated with this provocative maneuver. It is especially impressive if there is excessive acetabular anteversion, acetabular dysplasia, capsular laxity, or iatrogenic capsular deficiency. On the other hand, patients may feel much more comfortable with the leg in certain positions: abducting an uncovered femoral head will usually provide relief. Patients may adopt a certain foot-progression angle to minimize subluxation or impingement.

With extreme coxa vara or a high-riding greater trochanter due to a short femoral neck, hip abduction will be limited or blocked secondary to impingement against the lateral ilium.

It is important to realize that not all hip pain is intrinsic to the joint itself. A radiculopathy from the lumbar spine, especially upper lumbar, can certainly cause groin or buttock pain mimicking a hip source. Because of the proximity, an inguinal hernia can easily be confused with hip pain. Tendinopathies of the hip flexors and adductors, such as athletic pubalgia or sports hernia, may add to the confusion [25]. Again, selective local anesthetic injections will help to differentiate extra-articular pain from an intra-articular source.

Imaging

Plain Radiographs

Most anatomic deformities are readily visible on plain radiographs. The AP pelvis x-ray should not be rotated. Excessive lumbar lordosis may cause the pelvis to have the appearance of an inlet view. This may give the appearance of anterior acetabular over-coverage or a pincer lesion. The coccyx should appear to be 1–2 cm above the pubic symphysis. Reshooting the film with a cephalic tilt will often correct the appearance so that what was thought to be a pincer lesion may now be absent [26]. An "elongated neck" or Dunn view lateral will best demonstrate the presence of a cam lesion [27]. The false-profile view allows an assessment of anterior and posterior femoral head coverage, whether it is deficient or excessive. In addition, joint space narrowing may be present only in the anterior or posterior portions of the joint and therefore may not be readily apparent on the AP view [28]. Radiographs should be assessed for the presence of osteoarthritis (Tonnis classification) and joint space narrowing [29]. The radiographic parameters to be measured may include, but not be limited to, center-edge angle of Wiberg [30], anterior center-edge angle [28], neck-shaft angle [29], roof slope angle [31], and the alpha angle [32]. The presence of osteophytes and a "crossover sign" indicative of pincer impingement should be noted as well [33]. Os acetabuli may be present in cases of either hip impingement or dysplasia [34].

If a varus or valgus producing femoral intertrochanteric osteotomy is being considered, functional radiographs should be taken. For a varus derotation osteotomy, an AP pelvis x-ray should be taken with the legs abducted and internally rotated (if correcting excessive anteversion, otherwise slight external rotation may be better). For a valgus osteotomy, the involved leg should be adducted. The joint space should appear to be improved in the corrected position. The femoral head should rotate within the socket and not hinge or gap open [16].

Long-leg films should be obtained if there are concerns about the mechanical axis of the knee and how a varus or valgus osteotomy may alter alignment. A scanogram should be obtained if there are concerns about leg length inequality, keeping in mind that a varus or valgus intertrochanteric osteotomy will change the length by approximately 1 cm depending upon the degree of correction. An AP and lateral radiograph of the entire femur is useful for templating if a derotation osteotomy is to be done over an intramedullary nail.

MRI Scanning

MRI scans are not a replacement for plain radiographs; they should clarify the clinical picture. Scans should not be a pelvic MRI only but a dedicated surface coil study of the involved hip that clearly demonstrates the status of the labrum, synovium, and articular cartilage. Assessment of capsular integrity is important if an arthroscopic procedure with a capsule release has been performed. High-resolution studies are possible with and without the administration of intra-articular gadolinium [35, 36]. Increasingly, cartilage mapping studies such as T2 relaxation or dGEMRIC are useful to determine the overall health of the articular cartilage and therefore the probability of success with hip-preserving procedures [37, 38].

CT Scan

CT scans not only allow the viewing of axial, coronal, and sagittal slices but also detailed 3-D images that may be viewed from various angles which are helpful to understand the more subtle forms of hip impingement [39, 40]. These 3-D images may also be manipulated with range of motion simulation software to assess the effect of various corrective procedures [41, 42]. Useful measurements include the alpha angle, coronal and sagittal center-edge angles, neck-shaft angle, acetabular version, femoral version, and anterior inferior iliac spine size and position. The alpha angle can be measured on the "Swiss" or radial axial images to find the apex of a cam deformity on the femoral neck, expressed as a clock-face position (i.e., an alpha angle of 67° at the 2:00 position) [43].

Pathoanatomy of Femoral Deformity

Coxa valga is a problem because the steep neck-shaft angle causes a relative uncovering of the femoral head. The contact area between the acetabulum and femoral head is diminished, subjecting the articular cartilage to higher loads. Contributing to cartilage overload is the fact that there may be concomitant acetabular dysplasia, usually with anterior and lateral socket deficiency. The labrum, overloaded and subjected to shear stress, undergoes degeneration and tearing. Coxa valga may be associated with normal version, excessive femoral anteversion, or retroversion [24]. Compounding the problem is that a valgus neck-shaft angle decreases the abductor lever arm, raising joint reaction forces. Adding insult to injury, there may be coexisting femoroacetabular impingement, due to either a femoral cam lesion or acetabular pincer lesion.

Abnormalities of femoral version can affect not only the hip joint but also the patellofemoral joint and gait [44]. Excessive femoral anteversion overloads the front of the acetabulum and can result in labral degeneration. Femoral retroversion can create havoc, similar to the problems that occur if the femoral component of a hip replacement is retroverted. Femoral retroversion causes femoroacetabular impingement with resultant damage to the labrum and articular cartilage [24, 45, 46]. The femur and acetabulum can have completely different version measurements, either compounding the problem or offsetting each other. Diminished anteversion of the acetabulum or femur appears to be both more common and more damaging than excessive anteversion [24]. These abnormalities may appear as isolated entities or as part of a constellation of anatomic deformities [6, 7].

In cases of SCFE, the deformity also creates hip impingement and ultimately osteoarthritis with more damage occurring with larger deformities [3, 47].

A growth arrest of the capital femoral physis in infancy leads to a short femoral neck, a deformed femoral head, and overgrowth of the greater trochanter [48–51]. This condition is associated with abductor weakness and fatigue with ambulation, along with impingement of the greater trochanter against the ilium with abduction, but there may be considerable intra-articular deformities as well [52].

With Perthes disease there is often a resultant "coxa magna" deformity which is analogous to a square peg in a round hole [53, 54]. This deformity may also be associated with acetabular dysplasia along with premature cartilage degeneration, labral tears, and osteoarthritis.

The Rationale of Femoral Osteotomy

Femoral intertrochanteric osteotomy is a very important tool in the armamentarium of the hip-preserving surgeon. Depending upon the abnormality, the goal may be to reduce the load on the articular cartilage, alleviate impingement, improve the biomechanics and deficiency of the periarticular musculature, alter the leg length, increase range of motion, or a combination of any of these. Numerous studies have documented the fact that "joint-preserving" hip surgeries are most predictable and durable when performed before there is irreversible articular cartilage loss [55–58]. This is the opposite tact taken compared to a patient that requires a total hip arthroplasty, who is told to wait as long as possible. That strategy of watchful waiting may close the window of opportunity. It can be difficult to make a decision for patients in the ambiguous middle ground: some arthritis but not hopeless, not a child or young adult but not elderly either. Pain at rest, stiffness (less than 90° of flexion, a flexion contracture, limited rotation), and joint space narrowing are all worrisome signs that may contraindicate a femoral osteotomy. Even with an excellent range of motion and an acceptable joint space on plain radiographs, MRI scans may reveal profound cartilage loss and synovitis. Any osteotomy may be contraindicated in this situation.

It is also important to remember that numerous anatomic defects may coexist [7, 59, 60]. Dysplasia may have elements of impingement [61]. Historically, it was thought that dysplasia is nearly always present with excessive femoral or acetabular anteversion. In fact, Ganz has reported that as many as one out of six dysplastic hips may actually have elements of acetabular retroversion [62]. Coxa valga was usually thought to be associated with excessive femoral anteversion. Tonnis noted that 47 % sign of patients with coxa valga actually had diminished femoral anteversion of 15° or less [24].

Because of the fact that multiple anatomic abnormalities may be present, the surgeon has to consider the effects of one correction upon a different defect. For example, it is not unusual for acetabular dysplasia and coxa valga to coexist with femoral retroversion or a cam lesion. If the socket is reoriented, will dysplasia now be swapped for impingement? Should the cam lesion be addressed at the same time? Will the correction of excessive femoral anteversion cause a previously innocuous cam lesion to now impinge? 3-D CT modeling can be done to predict range of motion with excellent clinical validation [41, 42]. It is possible to simulate various corrections to predict the effect of correction upon the residual deformities remaining in the hip.

While it is hoped that a corrective osteotomy will preserve the hip indefinitely, a hip arthroplasty may eventually be required. Consideration must be made regarding the need for subsequent hardware removal and whether or not altering the femoral anatomy will compromise the placement of a femoral component. These concerns, however, must not prevent the execution of a femoral osteotomy when there is a clear indication.

Patients and surgeons must view a femoral osteotomy as an investment in the future. It may take months or even a year to recover fully from a femoral osteotomy. A period of protective weight-bearing is often required. This is in contrast to the fairly rapid recovery and pain relief of a total hip arthroplasty.

Varus Intertrochanteric Osteotomy

The primary goal of this osteotomy is to improve the coverage of the femoral head. The neck-shaft angle should be between 125° and 140° [63]. Tonnis considered coxa valga to be anything over 135° [24]. The problem with coxa valga is that it minimizes contact at the articular surfaces, causing cartilage overload and subsequently degeneration and loss. This can be accentuated by the shallow socket of dysplasia with lateral and anterior acetabular deficiency. The labrum and cartilage are both subjected to shear stress as well. This can be accentuated with excessive femoral anteversion, which has the effect of overloading the anterior rim. Historically, coxa valga has been associated with excessive femoral anteversion; performing a correction of the deformity would usually involve rotating the femur as well, giving rise to the term VRO: varus rotation osteotomy. Subsequent studies have shown that coxa valga may be associated with femoral retroversion instead [24]. From a biomechanical standpoint, a valgus neck-shaft angle will increase the overall joint reaction force by diminishing the length of the abductor lever arm. Placing the proximal femur into more varus should help to unload the articular cartilage [8, 14]. The varus correction should not be overdone to avoid excessive shortening of the leg and abductor muscle fibers. The most widely use device for this osteotomy is the 90° blade plate (Synthes, Paoli PA) which was specifically designed by the Swiss AO group to facilitate the procedure [15]. The plate incorporates an offset that medially displaces the femoral shaft. The rationale of this is to avoid displacing the mechanical axis of the leg into the medial compartment of the knee [16]. Because of the change in the neck-shaft angle, shortening of about 1 cm will usually occur provided that a wedge is not removed at the osteotomy site. This operation is therefore ideal if the involved leg is already longer.

Before the more widespread use of the Bernese periacetabular osteotomy (PAO), VRO alone was often used to treat dysplasia. While decreasing the neck-shaft angle will improve coverage, PAO is a more invasive but more powerful procedure to increase femoral head coverage. If the major defect is on the acetabular side, PAO is a more appropriate option. PAO also avoids deforming the proximal femur, facilitating femoral stem insertion should a future total hip arthroplasty be required. PAO also makes it easier to access the joint through an anterior arthrotomy, should a labral reattachment or femoral neck osteochondroplasty be required [64]. If a VRO is the correct procedure to be done, but there are concerns that the abductor muscle fibers will be excessively shortened, a simultaneous greater trochanter advancement can be done with the blade inserted through the trochanter. It is also possible to perform a surgical hip dislocation with a VRO should extensive intra-articular work be required such as cartilage grafting. To address most intra-articular problems, hip arthroscopy will usually suffice and can be performed on the same day immediately prior to the osteotomy.

Results of Varus Intertrochanteric Osteotomy

Haverkamp and Marti reported on the long-term results of bilateral varus intertrochanteric osteotomy for coxa valga in 26 patients. In this study, the surgeons treated the more painful hip (the "therapeutic" group) first. Patients were then encouraged to have the less damaged contralateral side done early, if the hip was at risk but minimally symptomatic (the "early osteotomy" group). The center-edge angle and neck-shaft angles were comparable in the 2 groups. At a follow-up of 20 years (range 15–26 years) the "therapeutic" group had a 15-year survivorship of 56 %, with 14 of 26 hips undergoing replacement at an average of 9.9 years. In the "early" group, the survivorship was 76 % with 6 hips undergoing replacement. These results were statistically significant. Better results were associated with a younger age at the time of surgery, a lower Tonnis arthritis score, and a better Merle d'Aubigne score [65].

Voos reported on 45 hips in 40 patients who underwent a varus-producing intertrochanteric osteotomy for coxa valga at an average follow-up of 22.6 years (range: 15–34 years). By 10 years follow-up, approximately one-third of patients had undergone total hip arthroplasty. By 20 years follow-up, this figure increased to two-thirds. Many of these patients in retrospect were not ideal VRO candidates: they may have had too much arthritis or may have been better candidates for PAO, which was not in widespread use at the time. In a subset of patients with a better initial HSS hip score, younger age, minimal osteoarthritis, or subluxation, the long-term survivorship was much better [55].

Ito reported on 55 VRO in 46 patients with coxa valga at an average follow-up of 17 years (range 6–28 years). Failure was considered to be a hip replacement or a Harris Hip Score of 70 or less. The survivorship was 81 % at 10 years, 60 % at 20 years, and 50 % at 25 years. The best results were obtained in patients with Tonnis grade arthritis of 0, 1, or 2, spherical heads, and only mild dysplasia at the time of osteotomy [66].

Iwase studied varus osteotomies at an average follow-up of 21 years and 58 valgus osteotomies at an average of 20 years. At the 10- and 15-year marks, the survivorship was 89 % and 87 %, respectively, for varus osteotomies. Survivorship was less impressive for valgus osteotomies, 66 % and 38 % at the 10- and 15-year marks, respectively [67].

Ansari et al. reported on 26 VRO in 24 patients at an average follow-up of 5 years (range: 1.6–23 years). There were no reoperations and one nonunion. The average Harris Hip Score improved from 72 to 97 points [68].

Zweifel reported on 52 VRO for dysplastic hips at an average follow-up of 17.8 years. Only 9.6 % of these hips did not require a total hip arthroplasty after a period of 15 years. However, this study reveals that the chance for long-term survival is best when there are minimal arthritic changes at the time of osteotomy [69].

In contrast, Koulouvaris demonstrated how good results could be with strict selection criteria. Inclusion criteria consisted of Tonnis grade 1 arthritis or less, spherical femoral heads, good congruency, deformity on the femoral side only, and age less than 60. Survivorship at 15 years was 96.5 %. In addition, by not removing a wedge at the osteotomy site, it was noted that femoral shortening was not apparent [70].

Valgus Intertrochanteric Osteotomy

A valgus femoral intertrochanteric osteotomy may be indicated for cases of coxa vara where there is a low neck-shaft angle (less than 125°) with a high greater trochanter. Correcting the neck-shaft angle will normalize the relationship between the center of rotation and the tip of the greater trochanter, placing the abductors and a more efficient length thereby improving abductor function. As with a varus intertrochanteric osteotomy, coexisting rotational abnormalities can be corrected as well. Blade plates ranging from 95° to 130° are available; the correct selection depends upon the degree of deformity and can be ascertained from preoperative planning.

The valgus intertrochanteric osteotomy had also been popularized in Europe for the treatment of hips with more advanced osteoarthritic changes and a “capital drop” osteophyte. The rationale of this osteotomy is to improve the contact area between the head and socket [13, 17]. This technique was usually combined with an extension osteotomy to improve congruency. Since most surgeons today would consider these types of hips to be in a “salvage” situation due to loss of joint space and osteophyte formation, it has fallen out of widespread use. This is particularly true when considering that function and longevity has increased considerably with the use of total hip arthroplasty.

With the deformity of slipped capital femoral epiphysis (SCFE), a large cam lesion is nearly always present on the femoral neck. In certain cases, it may be sufficient to remove a cam lesion alone. This may be done open or arthroscopically. However, if there is a coxa vara deformity as well, it may be beneficial to combine this with an intertrochanteric osteotomy to improve the biomechanics of the joint. Often, a flexion osteotomy is used to reorient a deformed head into a better position within the socket [71–73]. Smaller SCFE deformities (<30°) might be managed with a femoral neck debridement, while larger deformities might be better managed with a femoral osteotomy. For deformities greater than 60°, it may be necessary to perform a neck debridement along with a femoral osteotomy [74]. Again, version abnormalities can be corrected at the same time. If the neck-shaft angle is not sufficiently altered, pure rotation correction can be achieved with a rotational osteotomy over an intramedullary nail. Alternatively, SCFE deformities can be performed at the level of the femoral neck with a subcapital corrective osteotomy. The risk of avascular necrosis has to be considered [75].

The technique of “relative lengthening” of the femoral neck has also been used to correct a short femoral neck with overgrowth of the greater trochanter [52].

Valgus intertrochanteric osteotomy has also been a useful technique for femoral neck nonunions. The rationale is that the shear forces associated with a Pauwels type III fracture are converted to beneficial compressive forces by elevating the neck-shaft angle. The “double-angle” plates of the 110–130° range are usually most suited for this application [76–78].

Valgus intertrochanteric osteotomy has also been proposed for treating the protrusio deformity of the hip caused by the “medializing” forces on the femoral head. Approximately 20–30° of valgus correction should be employed to sufficiently lateralize weight-bearing forces [8, 79].

Results of Valgus Intertrochanteric Osteotomy

Maistrelli and Bombelli detailed the results of 277 intertrochanteric valgus-extension osteotomies performed for primary or secondary osteoarthritis between 1973 and 1975. The average age was 51 years and the follow-up range was 11–15 years. At latest follow-up, 67 % were rated as good or excellent. The results were best with age under 40, unilateral involvement, an elliptical head, minimal subluxation, adequate preoperative range of motion, and a mechanical etiology of the arthritis [80].

Kawate reported on 127 hips with arthritis treated with valgus intertrochanteric osteotomy. The average age at surgery was 42 years. At an average follow-up of 25 years, survivorship was 69 %. Results were better with lesser degrees of arthritis, age less than 50 years, and unilateral involvement [81].

Bartonicek reported on the use of valgus intertrochanteric osteotomy for iatrogenic coxa vara in patients older than 30 years who had been treated for the infantile treatment of developmental dysplasia of the hip (DDH). These patients all had a short neck and high trochanter along with shortening of the involved leg. The average age was 43 (range 31–60 years) with shortening of 2–4 cm. The average follow-up was 10 years (range 5–20 years). Three patients underwent total hip arthroplasty at 7.5–12 years after osteotomy and the average postoperative Harris Hip Score was 93 [82].

Schai reported the results of the Imhäuser intertrochanteric osteotomy in 51 slipped capital femoral epiphysis patients followed for an average of 24 years (range 20–29 years). At latest follow-up, 55 % were clinically asymptomatic with minimal adverse radiographic changes, while 28 % had moderate changes and 17 % had advanced changes [73].

Additional studies on the use of flexion intertrochanteric osteotomy for SCFE have reported good or excellent results in the range of 71–85 % with moderate to severe slip angles [83–86].

In the treatment of femoral neck nonunions with valgus intertrochanteric osteotomy, Varghese reported on 32 patients at an average age of 43 (range 14–60 years) at an average follow-up of 5 years range 2–12 years. Bone union rate was 91 % with an average Harris Hip Score of 82. Risk factors for failure were associated with the creation of excessive valgus and a neck resorption ratio less than 0.5 [76].

Gadegone reported on 41 hips with nonunion at an average age of 45. At a follow-up of 33 months (range 24–54 months), the union rate was 95 % at an average Harris Hip Score of 91 [77].

Other studies by Said and Kahn also reported union rates of 97 % and 88 %, respectively [78, 87].

Rosemeyer reported the results of valgus intertrochanteric osteotomy for the treatment of protrusio deformity. At an average follow-up of 6 years, good and excellent results were obtained in 21 of 25 hips (84 %) [88].

McBride reported the results of 19 hips in 12 patients treated at various institutions with various implants with a protrusio deformity. A follow-up of 2–33 years, 8 hips had undergone total hip replacement [89].

Femoral Osteotomy for the Correction of Retroversion or Excessive Anteversion

As noted above, rotational abnormalities may occur alone or in association with acetabular dysplasia, coxa valga, coxa vara, SCFE, or femoroacetabular impingement (FAI). Patients with FAI may have a fairly high incidence of dysplasia or coxa valga [7, 60]. Rotational deformities can be simultaneously corrected when a blade plate is applied to correct the neck-shaft angle or with the flexion osteotomy correction of SCFE. However, if there is no need to correct the neck-shaft angle or to add flexion/extension to an intertrochanteric osteotomy, a pure derotation osteotomy can be performed over an intramedullary nail. These techniques were initially employed to correct the rotational malunions of femoral shaft fractures. Corrections can be done in a minimally invasive fashion since it is not necessary to strip the muscles and apply a plate an articulated tensioning device. The transverse osteotomy can even be performed with a Winquist cam/blade hand saw (Biomet, Warsaw, Indiana) from within the canal obviating the need to expose the osteotomy site.

Why should there be concern regarding version abnormalities of the femur? Patients may present with gait abnormalities, and those with excessive femoral anteversion tend to walk with an in-toeing gait of the feet and a "squinting in" of the patellae. This adapted maneuver is thought to be an attempt to center the femoral heads and therefore minimize symptoms. Certain patients have adapted a compensatory external tibial torsion. These patients may have squinting of the patellae, but with an external foot-progression angle. Therefore, the CT scan should include the ankle as well as the hip and knee axial slices. This is important because if a derotation femoral osteotomy is performed and this is ignored, then the external rotation of the distal femur used to correct the excessive anteversion will cause a marked external foot-progression angle. In these cases, it may be necessary to perform a simultaneous correction of tibial torsion.

Excessive anteversion of the femoral component in a total hip arthroplasty can cause anterior dislocation. In the unreplaced native hip, there is shear stress applied to the anterior labrum and cartilage, analogous to the lateral overload of the dysplastic socket. This will be accentuated if there is concomitant excessive acetabular anteversion or deficiency of the anterior socket associated with dysplasia. The McKibbin index is the summation of femoral version plus acetabular version at the three o'clock position [90]. Excessive anteversion also places strain on the anterior soft tissues, capsule, and psoas tendon, all of which are attempting to restrain the subluxing anterior forces. Releasing the anterior capsule (as is often done with arthroscopic surgery) or the psoas tendon may increase the anterior instability of the hip.

Retroversion of the femur causes impingement, similar to the impingement and posterior instability that occurs in total hip arthroplasty. The impingement is accentuated if there is concomitant acetabular retroversion, a cam or pincer lesion, coxa profunda, or protrusio. At times, pure femoral retroversion may be the only source of hip impingement. If CT scans are not performed as part of the routine radiographic workup, these important findings will be missed. Increasingly, MRI scans can be employed to measure femoral version, but because of the much longer acquisition times compared to CT scans, the results may be less accurate if the patient rotates the legs at all between the time of capture of the hip and knee axial slices.

If there is a large cam or pincer lesion that can be addressed arthroscopically, this can be performed before proceeding with a more invasive femoral osteotomy. This may succeed if the degree of femoral retroversion is mild. However, for larger degrees of retroversion (more than 10° of retroversion), it is often preferable to proceed with the osteotomy instead. Hip arthroscopy can be performed immediately prior to the osteotomy to address any intra-articular issues such as labrum and cartilage damage.

Normal femoral version is approximately 12–15° [24, 91, 92]. There can be a wide variation; femoral retroversion detected on CT scan in patients with hip impingement has shown a range of 20° retroversion to 52° of anteversion [7]. Normal acetabular version is approximately +5°, +10°, and +15° at 1, 2, and 3 o'clock [33, 93].

Femoral rotational correction is usually in the range of 15–25°. Simultaneous tibial correction, if required, can be done acutely with an intramedullary nail or gradually with a spatial frame. Tibial correction is easier in the supramalleolar region, but it may be performed in the proximal tibia if it is desirable to correct the femoral-tibial angle as with a varus or valgus knee.

One advantage of this technique of derotation osteotomy is that it does not alter leg length as does intertrochanteric osteotomy because the neck-shaft angle is left unchanged.

The interplay between various coexisting deformities has to be kept in mind. If excessive femoral anteversion is corrected but a cam or pincer lesion is present, the anterior overload may be alleviated but impingement may now occur. For that reason, the cam or pincer should be prophylactically debrided. Femoral retroversion can coexist with acetabular dysplasia; if the socket is rotated anteriorly to improve coverage, more impingement may occur.

Results of Derotation Femoral Osteotomy

Winquist reported on the ability to perform a closed osteotomy and intramedullary nailing to correct simple rotational deformities [19].

Chapman reported on closed osteotomy nailing performed in 37 patients for leg length inequality or rotational deformities. Shortening osteotomies were done in 31 patients, derotation in 6. Preoperative rotational deformities average to 58°; all were corrected to within 5° of normal. There were no nonunions, no infections [20].

Stahl treated 14 patients with posttraumatic rotational deformities of the femur. The deformities ranged from 26° to 63°. Surgery was performed with a closed technique over an intramedullary nail. Postoperative CT scans revealed excellent correction of the deformity within 4° in all cases. All osteotomies healed eventually [22].

Varus Intertrochanteric Osteotomy: Surgical Technique

(Representative case examples are shown in Figs. 1 and 2.)

All appropriate radiographic studies should be performed. High-resolution MRI scans are needed to assess the degree of intra-articular damage. While it is possible to open the hip joint during a VRO, it makes for a more extensive dissection and it may still be very difficult to get an excellent view of intra-articular pathology. It is usually preferable to perform hip arthroscopy instead to address the intra-articular problems, either staged or simultaneously immediately before the osteotomy. Plain radiographs should include an AP pelvis, Dunn view lateral, and a false-profile view of the involved hip. In addition, "functional" radiographs should be obtained to simulate the osteotomy. For a varus osteotomy, the AP view with legs abducted will simulate how the coverage will appear after adding varus; if there is excessive anteversion, the leg should also be internally rotated with the abduction to simulate the derotation component of the VRO.

A 3-D CT scan will provide excellent visualization of the deformity and is the most accurate method to measure acetabular and femoral version. Preoperative templating and planning should not be omitted. The surgeon can think through the steps involved, decide on the degree of angular correction, select the correct blade plate, and predict the degree of shortening or lengthening. It is also possible to predict the length of the blade and where it will come to rest in the femoral head.

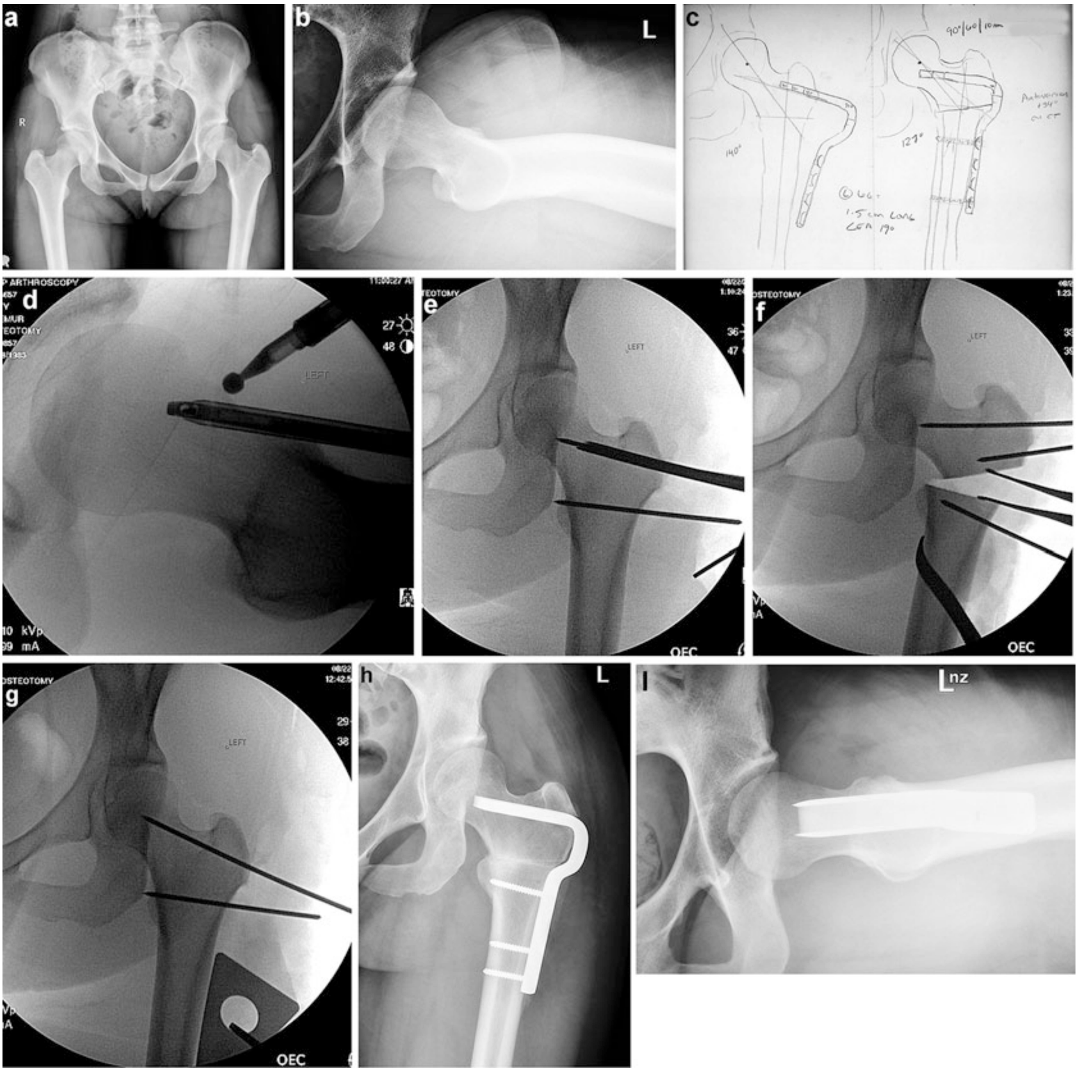


Fig. 1 Varus derotation intertrochanteric osteotomy. The patient is a 28-year-old female with left hip pain for over 2 years. The left leg is 1.5 cm long. The left leg has an in-toeing gait. There is borderline hip dysplasia with the center-edge angle 20° on the AP view and 23° on the false-profile view. Coxa valga is present with a neck-shaft angle of 140°. In addition, there is excessive femoral anteversion of 34° along with increased acetabular anteversion of 27°. A femoral neck cam lesion, with an alpha angle of 64°, is also present. A varus derotation intertrochanteric osteotomy was performed along with a concomitant hip arthroscopy and femoral neck osteochondroplasty to avoid further impingement after diminishing femoral anteversion. It was felt that a femoral intertrochanteric osteotomy would best correct the various deformities and reduce cartilage overload. A 13° varus correction was performed along with a 20° reduction of the femoral anteversion. (**a**) AP pelvis radiograph. (**b**) Dunn view lateral left hip showing cam lesion. (**c**) Preoperative plan tracing for a VRO with a 90° blade plate. (**d**) Arthroscopic femoral neck osteochondroplasty performed immediately prior to the osteotomy. (**e**) Steinmann pin placement at the osteotomy site and into the femoral neck at the desired angle of varus correction. (**f**) Chisel insertion hugging the proximal Steinmann pin, location confirmation should be made on both the AP and lateral views. (**g**) Lamina spreader used to open the osteotomy and mobilize the fragments. Rotation control Steinmann pins have been placed on either side prior to performing the osteotomy. (**h**) AP radiograph at follow-up. (**i**) Lateral radiograph at follow-up. The operation not only provided pain relief but also restored normal hip rotation and foot-progression angle, prevented hip impingement, and equalized leg lengths

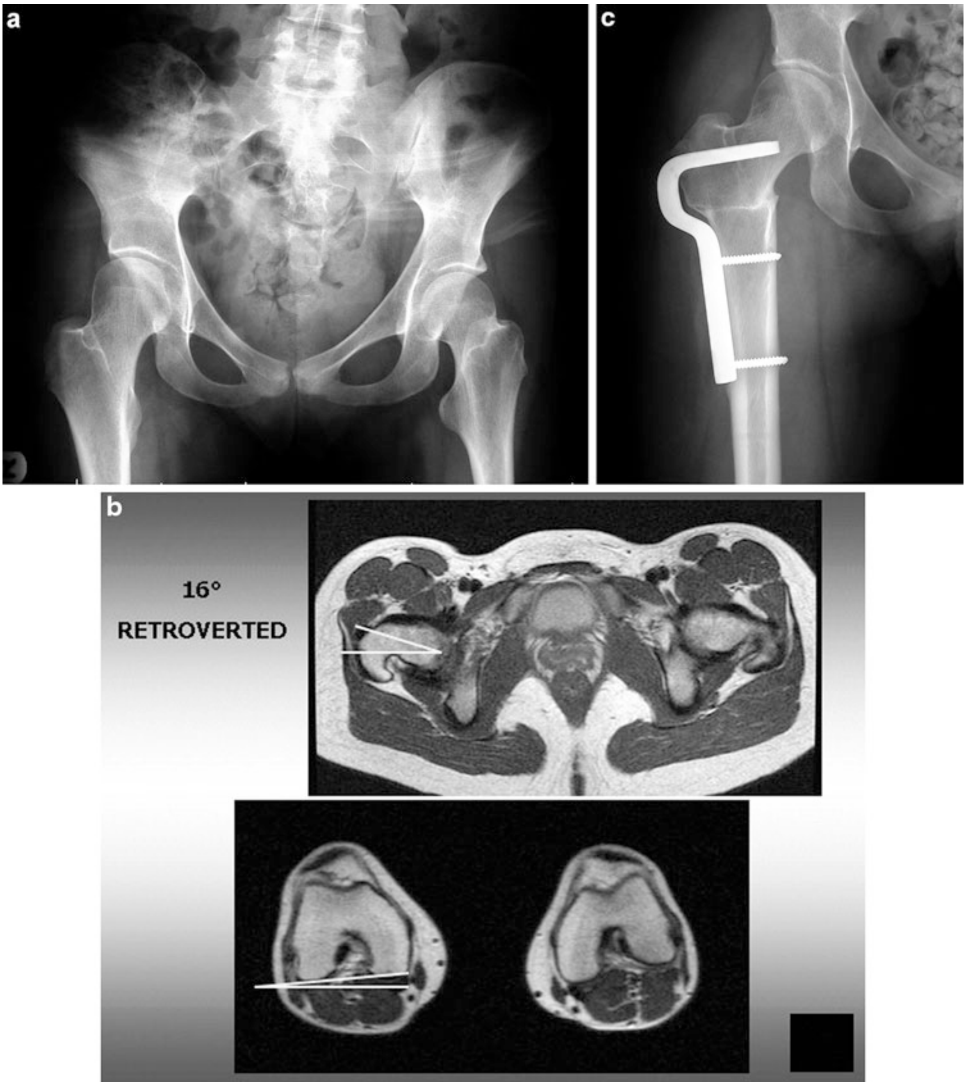


Fig. 2 Varus derotation intertrochanteric osteotomy with femoral retroversion. The patient is a 19-year-old female who was born with a dislocated right hip. She was treated with an open reduction and bracing in infancy. Pain in the hip has been present for over 1 year, limiting activities. The right leg is 1.5 cm longer than normal. She has limited internal rotation, a positive impingement test, excessive external rotation, and minimal internal rotation. There is coxa valga with a neck-shaft angle of 146°, borderline acetabular dysplasia, and 16° of femoral retroversion. A varus derotation intertrochanteric osteotomy was performed to correct valgus and retroversion of the femur. (**a**) Preoperative AP pelvis radiograph. (**b**) Axial MRI images through the hip and knee demonstrating 16° of retroversion of the right femur. The left hip has normal femoral anteversion. (**c**) Postoperative AP radiograph of the right hip. The osteotomy has relieved pain, corrected the foot-progression angle, and restored normal hip range of motion and leg lengths

The operation should be performed on a radiolucent table. Either the supine or lateral position may be used. In the supine position, a radiolucent bump or float beneath the involved buttock makes the exposure easier.

A straight lateral incision is used. A larger incision may be required if the articulated tensioning device is to be used distally to compress the osteotomy site. The vastus lateralis is elevated from the femur near the linea aspera. Perforating vessels are identified and coagulated. It is helpful to angle the fascia incision anteriorly just distal to the vastus ridge, this will keep adequate tissue at the vastus lateralis origin to permit a complete closure of the vastus fascia over the blade plate. The vastus origin is then elevated proximally to allow the plate to be fully opposed to the femoral cortex.

Two smooth Steinmann pins are placed, each approximately 7/64 or 1/8 in. One is placed transversely from lateral to medial at the superior border of the lesser trochanter at the proposed site of the osteotomy. The second is placed just proximal to the proposed path of the chisel. The proximal pin has to be placed at such an angle (usually 105–110° to the shaft) so that the chisel that hugs the pin will make the correct path for the blade plate. It is helpful to use the various metal triangles in the Synthes blade plate set to achieve the correct angle in the coronal plane. Care must be taken to confirm that the pin is properly seated in the lateral projection as well. If there is excessive external rotation at 90°, a frog-type view may show the lateral projection. If the patient has excessive anteversion, then it may be easier to fully internally rotate and cant the fluoroscope over the involved hip to achieve the lateral view. Placing the chisel is the most crucial part of the operation. After placing the pin, compare it to the preoperative tracing. Are the two comparable? It is not necessary to be concerned with rotational control at this point. The chisel should be inserted at the correct angle on the AP projection so that once the femur is sectioned and the 90° blade plate inserted, it will affect the correct coronal plane correction. The chisel should hug the Steinmann pin; this will assure not only the correct varus/valgus angle but also the correct anterior/posterior placement in the femoral head and neck (provided that this was controlled fluoroscopically with the pin).

The 90° adult blade plate has blade lengths of 40, 50, 60, and 70 mm. One should have an idea of the correct length from the preoperative planning. The offsets available are 10, 15, or 20 mm. Again, preoperative tracing will help to determine which angled plate should fit the contour of the femur best after correction. The blade plate was designed with this offset to cause an intentional medial displacement of the femoral shaft so as not to overload the medial compartment of the knee.

There should be at least 15 mm from the osteotomy to the site of chisel insertion. Any less than this may cause the blade to break into the osteotomy site when compression is applied.

The harder the bone, the more important it is to extract the chisel partially after each advancement of approximately 1 cm. If the chisel is inserted to the complete 40–70 mm in one thrust, it may be extremely difficult to extract the chisel.

Before inserting the chisel, determine whether flexion or extension will be needed. If neither is desired, then the handle of the chisel guide should be parallel to the shaft of the femur during insertion. If 10–30° of flexion or extension are desired (as in cases of SCFE or AVN of the femoral head), this must also be taken into account before inserting the chisel. Flexion osteotomies have the effect of rotating the femoral head anteriorly, vice versa for an extension osteotomy. During insertion, periodic AP and lateral fluoroscopic images should be taken to make sure the chisel is following a path closely applied to the Steinmann pin. To control rotation, Steinmann pins are placed anteriorly both proximal and distal to the osteotomy site. The pins must be placed at the desired angle of correction. It is usually easier to set the pins at the desired degree of correction (i.e., 20° apart from each other; the triangles are used to determine this) so that making them parallel to each other during blade plate fixation will have affected the desired rotational correction. Only then can the transverse osteotomy be performed, otherwise there is no control of rotation. The osteotomy is made parallel to the previously placed Steinmann pin at the superior border of the lesser trochanter.

After performing the osteotomy of the femur, a lamina spreader helps to mobilize the fragments sufficiently. If it is not in the way, the chisel can be kept in the proximal fragment until this moment to visualize the correct path for blade plate insertion. The proximal Steinmann pin will help to ensure the correct path as well. The blade plate is then inserted and impacted with the pencil punch until the plate comes to rest against the lateral cortex of the proximal fragment. The distal fragment is brought to rest against the plate and secured with a Verbrugge clamp. Prior to tightening the clamp, the distal femur is rotated to align the rotation control pins parallel to each other.

The articulated tensioning device helps to compress the osteotomy site. Historically, a medial wedge of bone was removed to achieve a greater surface area of bony apposition. However, this tends to shorten the femur needlessly. Instead, a single transverse osteotomy is made. With compression, the medial cortex of the proximal fragment is impacted into the canal of the distal fragment, due to the intentional medial displacement. An incision approximately 5 cm longer may be required to use the articulated tensioning device that attaches to the distal screw hole. Alternatively, compression can be achieved by placing the plate screws in compression mode.

With harder bone, it may not be necessary to fill all four screw holes on the plate. The proximal screw just below the blade at the corner of the blade plate should be used if there is any compromise of bone quality.

At this point, the AP and lateral fluoroscopic images should be inspected. Is the osteotomy well opposed and well aligned? Does it compare favorably with the preoperative plan tracing? Are all the screws in optimal position?

The hip range of motion should now be tested after removing the Steinmann pins and tensioning device. At 90° of flexion, there should now be internal rotation if correcting retroversion and improved external rotation if excessive anteversion was corrected.

If it is felt that excessive varus was required to correct the neck-shaft angle, the greater trochanter tip may be too high compared to the level of the center of rotation. A greater trochanteric advancement may be done as well. The chisel is inserted into the greater trochanter at a 90° angle (the position it will be and when the blade plate was inserted). It is inserted initially only to the depth of the greater trochanter osteotomy. The trochanter osteotomy is performed and then the Steinmann pin and chisel inserted as needed to correct the neck-shaft angle.

After the osteotomy, the blade can be inserted first into the greater trochanter, which is then advanced distally before finishing the blade insertion into the head/neck fragment. This initial insertion into the greater trochanter has also to consider flexion/extension corrections. It may be desirable to augment the greater trochanter fixation with supplemental screws.

The vastus fascia is then closed over top of the plate. Drains are used if required and the wound closed in layers. Postoperatively, the patient can be out of bed as soon as possible. A cast or brace is not required. There are no range of motion precautions. The patient is kept at 20° toe-touch weight-bearing until 6 weeks postoperatively, at which time progression is made to full weight-bearing if bone consolidation is evident.

Valgus Intertrochanteric Osteotomy: Surgical Technique

(A representative case example is shown in Fig. 3.)

The valgus osteotomy is performed in a similar fashion to the varus osteotomy, with certain differences. The valgus osteotomy tends to lengthen the leg; therefore, it may be desirable to remove a lateral-based wedge from the osteotomy site. This will not only improve bony apposition but will also help to minimize undesired leg lengthening.

If the involved leg is already short, then the lengthening effect may be desirous.

Instead of using the 90° blade plate commonly used with varus intertrochanteric osteotomy, the plates ranging from 95° to 130° are used instead. The correct plate can be determined from preoperative planning and tracing.

Care must be taken when using the higher angled plates with the articulated tensioning device. With a 90° plate, almost pure compression is generated. However, with more valgus angles, the device may have the effect of pulling the blade out of the proximal fragment. This must be monitored carefully fluoroscopically.

As opposed to the varus intertrochanteric osteotomy, some lateral displacement of the distal

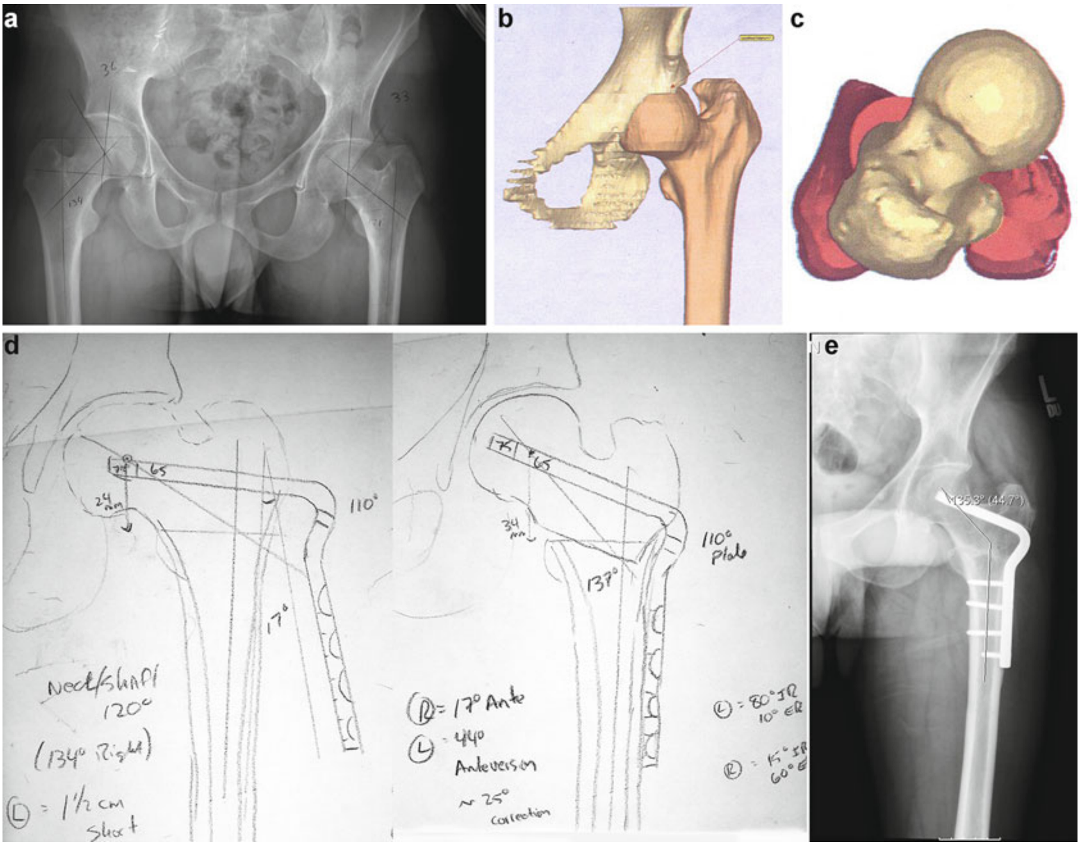


Fig. 3 **Valgus derotation intertrochanteric osteotomy**. The patient is a 30-year-old male who presented with several years of increasing left hip pain. In addition, he noted a pronounced lack of external hip rotation since the age of 6. The left leg is 1.5 cm shorter than the right leg. A valgus derotation intertrochanteric osteotomy was preformed. (**a**) AP radiograph of the pelvis demonstrating coxa vara with a neck-shaft angle of 121° with a relatively high greater trochanter. (**b**) 3-D CT scan image of the left hip demonstrating the excessive femoral anteversion as well as coxa vara. (**c**) Superimposed axial images of the proximal and distal femur demonstrate 44° of femoral anteversion. (**d**) Preoperative plan tracing for a valgus derotation intertrochanteric osteotomy employing a 110° blade plate. (**e**) Correction of the neck-shaft angle to 135°. In addition, approximately 25° of femoral anteversion was reversed. The procedure relieved pain, restored normal hip rotation and equilibrated leg lengths. Hip abductor strength also improved

fragment is usually desirous to centralize the leg axis within the knee joint.

Derotation Femoral Osteotomy: Surgical Technique

(A representative case example is shown in Fig. 4.)

If there is no need to correct the coronal neck-shaft angle or to add flexion or extension to an intertrochanteric osteotomy, then a pure rotational osteotomy can be performed.

With this technique, the anatomy of the proximal femur is unaltered, minimizing the effect upon the stem placement should a future total hip arthroplasty be required. Because the neck-shaft angle is unaltered, there is no change in leg length. The abductors are not shortened as with a varus osteotomy; therefore, the abductors recover more quickly. Because of the stability with a transverse osteotomy fixed with an intramedullary nail, weight-bearing as tolerated may commence immediately. There is considerably less muscle dissection then with the use of a blade plate.

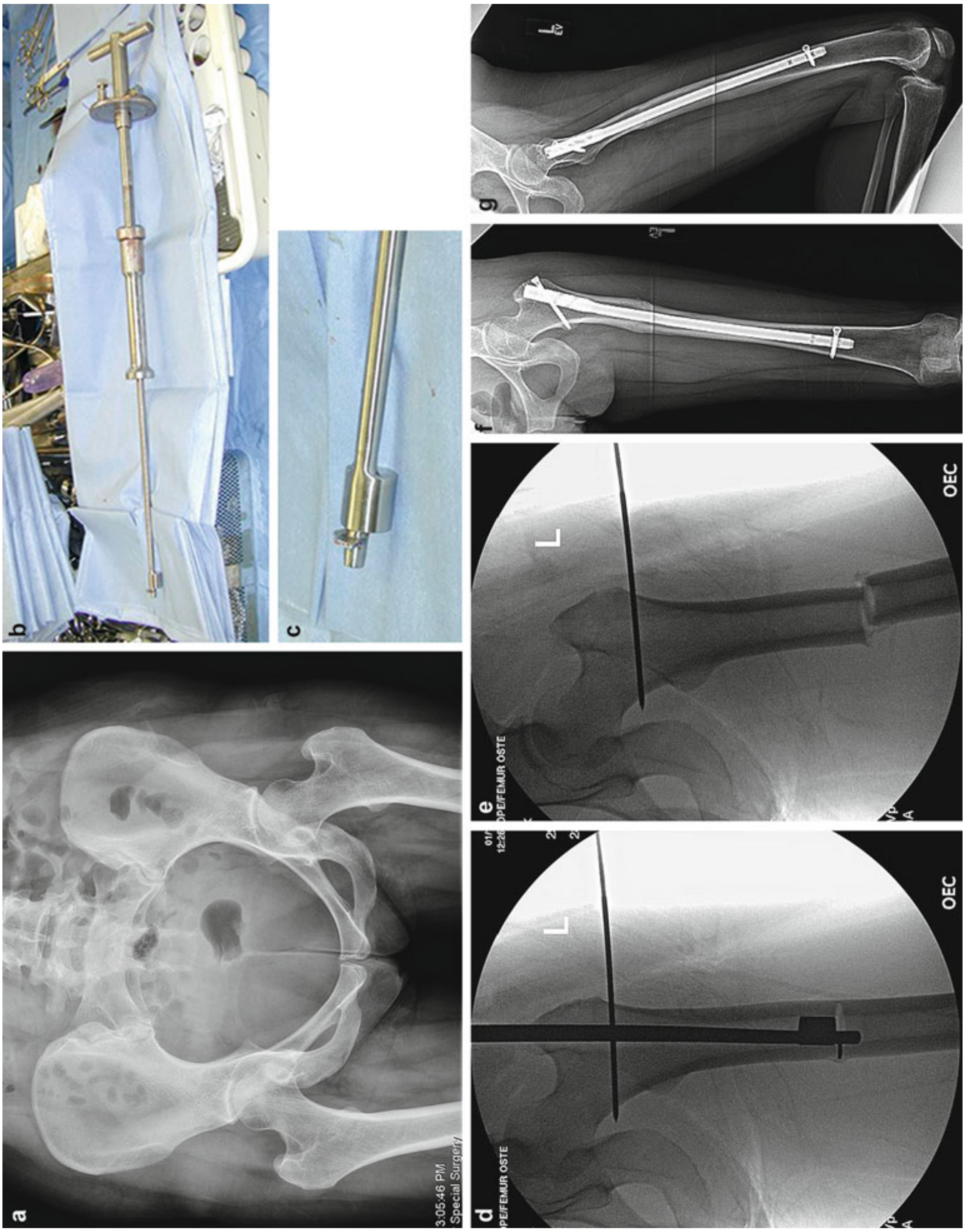


Fig. 4 (continued)

Hardware removal, if required, is also less invasive.

Piriformis fossa or trochanteric entry nails can be used. The osteotomy is made in the subtrochanteric region. Shorter femoral nails, such as the Gamma or Synthes TFN, may be used, which facilitates placement of the distal interlocking screw. However, these nails were designed for fracture fixation and have a larger diameter at the proximal end of the nail (approximately 17 mm), which may remove excessive bone in younger patients.

Full-length trochanteric entry nails tend be easier to insert and come in smaller diameters. Adolescent nails may be required in smaller patients.

The patient is placed in the supine position with a radiolucent float beneath the involved buttock, and the ipsilateral arm suspended over the face with a McConnell device to allow unencumbered access to the proximal greater trochanter. The entire leg is prepped and draped.

A small incision is made just proximal to the greater trochanter approximately 6 cm in size. The gluteus maximus fibers are split bluntly and any adhesions between the fascia lata and greater trochanter/abductors are freed with digital pressure. The canal is entered with the threaded guide pin from the apex of the greater trochanter, staying in the more posterior bald spot of the trochanter to avoid disruption of the abductor fibers. The pin has to be placed with sufficient room remaining between it and the posterior edge of the greater trochanter so that the reamers or nail does not violate the posterior cortex.

After the guide pin is advanced into the canal, AP and lateral fluoroscopic images must confirm optimal position.

A starter reamer is then inserted only to the level of the lesser trochanter. This must be done to accommodate the proximal width of the IM nail. A helpful step is to insert the proximal smooth Steinmann pin (usually 7/64 or 1/8 in.) that will serve as a rotational control, at this point. Since the reamers and nail enter in the more posterior half of the trochanter, the Steinmann pin is brought to rest against the lateral cortex of the greater trochanter through a separate stab incision. After feeling the anterior cortex with the pin, it is slid posteriorly as it is drilled all the way to the medial cortex. In this fashion, it avoids the path of the reamers, Winquist saw and intramedullary nail. It is placed parallel to the floor and perpendicular to the shaft.

The femoral shaft is then prepared with flexible reamers, usually 0.5 mm over the proposed nail diameter. The length and diameter of the nail can be predicted by preoperative templating of the full-length femur radiographs.

A second Steinmann pin is placed in the supracondylar region, also perpendicular to the shaft. It must be sufficiently distal to avoid impingement from the intramedullary nail. It is much easier to insert the distal pin so that after the osteotomy, making the pins parallel will achieve the desired rotational correction.

The subtrochanteric region is then reamed 0.5 mm larger than the proposed Winquist saw. This can usually be determined from preoperative planning. The Winquist saw has diameters ranging from 12 to 17 mm. Each millimeter increase in saw size will provide an additional 3 mm of cutting diameter, starting at 20.5 mm.

The Winquist saw is inserted to the desired location in the subtrochanteric region and the osteotomy completed. Now there will be nearly complete free rotation of the distal femur.

Fig. 4 **Derotation osteotomy of the femoral shaft.** The patient is a 28-year-old female with several years of left hip pain. She had previously undergone arthroscopy and labral debridement, which was minimally effective. The center-edge angle and neck-shaft angle are in normal range, but femoral anteversion measures 38°. A derotation subtrochanteric osteotomy was preformed. The osteotomy was performed closed with an intramedullary Winquist saw, fixation achieved with a trochanteric entry intramedullary nail. (**a**) AP pelvis radiograph. (**b**) Winquist saw. (**c**) Close-up of Winquist cam/blade assembly. (**d**) Intraoperative fluoroscopic image showing the femur nearly transected. The Steinman pin is for rotation correction control, the distal control pin (not seen in the figure) is located in the supracondylar region. (**e**) The femur now transected, allowing free femoral rotation. The saw has been removed. (**f** and **g**) AP and lateral postoperative radiographs of the femur showing bony union

The guide wire is reinserted into the canal. The rod is inserted over the guide wire while an assistant monitors a rotational correction by keeping the Steinmann pins parallel to each other.

Two interlocking screws are usually fixed sufficient, one proximal and one distal. If using fluoroscopy for the distal screw, the proximal screw is inserted first through the handle jig. While keeping the pins parallel, the distal interlock is placed, preferably in dynamic mode. Axial compression of the osteotomy during this step can be performed by pushing proximally on the foot. If an intramedullary electromechanical guidance device (i.e., SureShot, Smith and Nephew, Memphis, TN) is to be used to facilitate placing the distal interlock, it must be placed first since the probe travels distally inside the nail. The proximal interlocking screw that passes from the greater to lesser trochanter is typically used. If for some reason fixation is not sufficient, the proximal screws can be passed up the femoral neck. A second distal interlocking screw may be placed if there is any micromotion with rotational testing.

After the insertion handle and Steinmann pins are removed, the correction is evaluated by flexing the hip to 90°. There should now be a dramatic difference in rotation: more internal rotation if there was correction of retroversion, the opposite for the correction of excessive anteversion.

Summary

In the management of femoral deformities, it is critical to make the correct diagnosis. A thorough history and physical is vital. One must appreciate the degree of coronal and sagittal plane deviation, as well as the rotational version of both the femur and acetabular. Obtaining the proper radiographic studies is crucial to making an accurate assessment. Keep in mind that numerous deformities may coexist and that correcting one may exacerbate another. Surgical correction should achieve the goal of not only pain relief but long-term hip preservation as well. All hip-preserving surgeries have a greater likelihood of success if performed before there is irreversible articular cartilage loss.

References

1. Boyer DW, Mickelson MR, Ponseti IV. Slipped capital femoral epiphysis. Long-term follow-up study of one hundred and twenty-one patients. J Bone Joint Surg Am. 1981;63(1):85–95.
2. Harris WH. Etiology of osteoarthritis of the hip. Clin Orthop. 1986;213:20–33.
3. Goodman DA, Feighan JE, Smith AD, Latimer B, Buly RL, Cooperman DR. Sublinical slipped capital epiphysis. Relationship to osteoarthrosis of the hip. J Bone Joint Surg Am. 1997;79:1489–97.
4. Harris WH. The correlation between minor or unrecognized developmental deformities and the development of osteoarthritis of the hip. Instr Course Lect. 2009;58:257–9.
5. Ganz R, Parvizi J, Beck M, Leunig M, Notzli H, Siebenrock KA. Femoroacetabular impingement: a cause for osteoarthritis of the hip. Clin Orthop. 2003;417:112–20.
6. Allen D, Beaule PE, Ramadan O, Doucette S. Prevalence of associated deformities and hip pain in patients with cam-type femoroacetabular impingement. J Bone Joint Surg Br. 2009;91(5):589–94.
7. Moya LE, Molisani D, Henn F, Ma Y, Kelly BT, Buly RL. Femoral retroversion in patients with femoroacetabular impingement: a cofactor in the development of hip osteoarthritis. In: Orthopaedic Research Society 55th annual meeting, Feb 2009, Las Vegas; 2009.
8. Pauwels F. Biomechanics of the normal and diseased hip. Theoretical foundation, technique and results: an atlas. Berlin: Springer; 1976.
9. Ganz R, Klaue K, Vinh TS, Mast JW. A new periacetabular osteotomy for the treatment of hip dysplasias. Technique and preliminary results. Clin Orthop. 1988;232:26–36.
10. Voss C. Coxarthrosis; the temporary hanging hip, a new procedure for operative treatment of painful hip in the aged and other chronic deforming diseases of the hip. Munch Med Wochenschr. 1956;98(28):954–6.
11. McMurray TP. Osteo-arthritis of the hip joint [classical article]. 1939. Clin Orthop. 1990;(261):3–10.
12. Pauwels F. Diseases of the hip of mechanical origin and their treatment by adduction osteotomy. Rev Chir Orthop Reparatrice Appar Mot. 1951;37(1):22–30.
13. Maquet P. Valgus intertrochanteric osteotomy and tenotomy (Pauwels II). In: Biomechanics of the hip. Berlin: Springer; 1985a. p. 102–33.
14. Maquet P. Varus intertrochanteric osteotomy (Pauwels I). In: Biomechanics of the hip. Berlin: Springe; 1985b. p. 76–101.
15. Muller ME. Intertrochanteric osteotomy: indication, preoperative planning, technique. In: Schatzker J, editor. The intertrochanteric osteotomy. Berlin: Springer; 1984. p. 25–66.
16. Schneider R. Intertrochanteric osteotomy in osteoarthritis of the hip joint. In: Schatzker J, editor.

The intertrochanteric osteotomy. Berlin: Springer; 1984. p. 135–77.
17. Bombelli R, Gerundini M, Aronson J. The biomechanical basis for osteotomy in the treatment of osteoarthritis of the hip: results in younger patients. Hip. 1984;18–42.
18. Santore RF, Bombelli R. Long-term follow-up of the Bombelli experience with osteotomy for osteoarthritis: results at 11 years. Hip. 1983;106–28.
19. Winquist RA. Closed intramedullary osteotomies of the femur. Clin Orthop. 1986;212:155–64.
20. Chapman ME, Duwelius PJ, Bray TJ, Gordon JE. Closed intramedullary femoral osteotomy. Shortening and derotation procedures. Clin Orthop. 1993;287:245–51.
21. Jaarsma RL, van Kampen A. Rotational malalignment after fractures of the femur. J Bone Joint Surg Br. 2004;86(8):1100–4.
22. Stahl JP, Alt V, Kraus R, Hoerbelt R, Itoman M, Schnettler R. Derotation of post-traumatic femoral deformities by closed intramedullary sawing. Injury. 2006;37(2):145–51.
23. Crane L. Femoral torsion and its relation to toeing-in and toeing-out. J Bone Joint Surg Am. 1959;41-A(3):421–8.
24. Tonnis D, Heinecke A. Acetabular and femoral anteversion: relationship with osteoarthritis of the hip. J Bone Joint Surg Am. 1999;81(12):1747–70.
25. Meyers WC, McKechnie A, Philippon MJ, Horner MA, Zoga AC, Devon ON. Experience with “sports hernia” spanning two decades. Ann Surg. 2008;248 (4):656–65.
26. Tannast M, Zheng G, Anderegg C, Burckhardt K, Langlotz F, Ganz R, et al. Tilt and rotation correction of acetabular version on pelvic radiographs. Clin Orthop Relat Res. 2005;438:182–90.
27. Meyer DC, Beck M, Ellis T, Ganz R, Leunig M. Comparison of six radiographic projections to assess femoral head/neck asphericity. Clin Orthop Relat Res. 2006;445:181–5.
28. Lequesne M, de Seze S. La faux profil du bassin: nouvelle incidence radiographique pour l’etude de la hanche. Son utilite dans les dysplasies et les differentes coxopathies. Rev Rhum. 1961;28:643–52.
29. Tonnis D. Congenital dysplasia and dislocation of the hip in children and adults. Berlin: Springer; 1987. p. 121–3.
30. Wiberg G. Studies on the dysplastic acetabulum and congenital subluxation of the hip joint. Acta Chir Scand. 1939;83 Suppl 58:1–135.
31. Tonnis D. Normal values of the hip joint for the evaluation of X-rays in children and adults. Clin Orthop Relat Res. 1976;119:39–47.
32. Notzli HP, Wyss TF, Stoecklin CH, Schmid MR, Treiber K, Hodler J. The contour of the femoral head-neck junction as a predictor for the risk of anterior impingement. J Bone Joint Surg Br. 2002;84(4):556–60.
33. Reynolds D, Lucas J, Klaue K. Retroversion of the acetabulum. A cause of hip pain. J Bone Joint Surg Br. 1999;81(2):281–8.
34. Martinez AE, Li SM, Ganz R, Beck M. Os acetabuli in femoro-acetabular impingement: stress fracture or unfused secondary ossification centre of the acetabular rim? Hip Int. 2006;16(4):281–6.
35. Toomayan GA, Holman WR, Major NM, Kozlowicz SM, Vail TP. Sensitivity of MR arthrography in the evaluation of acetabular labral tears. AJR Am J Roentgenol. 2006;186(2):449–53.
36. Mintz DN, Hooper T, Connell D, Buly R, Padgett DE, Potter HG. Magnetic resonance imaging of the hip: detection of labral and chondral abnormalities using noncontrast imaging. Arthroscopy. 2005;21(4):385–93.
37. Kim YJ, Jaramillo D, Millis MB, Gray ML, Burstein D. Assessment of early osteoarthritis in hip dysplasia with delayed gadolinium-enhanced magnetic resonance imaging of cartilage. J Bone Joint Surg Am. 2003;85-A(10):1987–92.
38. Bittersohl B, Miese FR, Hosalkar HS, Mamisch TC, Antoch G, Krauspe R, et al. T2* mapping of acetabular and femoral hip joint cartilage at 3 T: a prospective controlled study. Invest Radiol. 2012;47(7):392–7.
39. Beaule PE, Zaragoza E, Motamedi K, Copelan N, Dorey FJ. Three-dimensional computed tomography of the hip in the assessment of femoroacetabular impingement. J Orthop Res. 2005;23(6):1286–92.
40. Heyworth BE, Dolan MM, Nguyen JT, Chen NC, Kelly BT. Preoperative three-dimensional CT predicts intraoperative findings in hip arthroscopy. Clin Orthop Relat Res. 2012;470(7):1950–7.
41. Bedi A, Dolan M, Hetsroni I, Magennis E, Lipman J, Buly R, et al. Surgical treatment of femoroacetabular impingement improves hip kinematics: a computer-assisted model. Am J Sports Med. 2011;39 (Suppl):43s–9.
42. Bedi A, Dolan M, Magennis E, Lipman J, Buly R, Kelly BT. Computer-assisted modeling of osseous impingement and resection in femoroacetabular impingement. Arthroscopy. 2012;28(2):204–10.
43. Milone MT, Bedi A, Poultsides L, Magennis E, Byrd JW, Larson CM, et al. Novel CT-based three-dimensional software improves the characterization of cam morphology. Clin Orthop Relat Res. 2013;471(8):2484–91.
44. Eckhoff DG. Effect of limb malrotation on malalignment and osteoarthritis. Orthop Clin North Am. 1994;25(3):405–14.
45. Tonnis D, Heinecke A. Diminished femoral antetorsion syndrome: a cause of pain and osteoarthritis. J Pediatr Orthop. 1991;11(4):419–31.
46. Tonnis D, Heinecke A. Decreased acetabular anteversion and femur neck antetorsion cause pain and arthrosis. 2: Etiology, diagnosis and therapy. Z Orthop Ihre Grenzgeb. 1999;137(2):160–7.
47. Cooperman DR, Charles LM, Pathria M, Latimer B, Thompson GH. Post-mortem description of slipped capital femoral epiphysis. J Bone Joint Surg Br. 1992;74(4):595–9.
48. Iwersen LJ, Kalen V, Eberle C. Relative trochanteric overgrowth after ischemic necrosis in congenital dislocation of the hip. J Pediatr Orthop. 1989;9(4):381–5.

49. Wagner H. Treatment of osteoarthritis of the hip by corrective osteotomy of the greater trochanter. In: Schatzker J, editor. The intertrochanteric osteotomy. Berlin: Springer; 1984. p. 179–201.
50. Lloyd-Roberts GC, Wetherill MH, Fraser M. Trochanteric advancement for premature arrest of the femoral capital growth plate. J Bone Joint Surg Br. 1985;67 (1):21–4.
51. Macnicol MF, Makris D. Distal transfer of the greater trochanter. J Bone Joint Surg Br. 1991;73(5):838–41.
52. Ganz R, Huff TW, Leunig M. Extended retinacular soft-tissue flap for intra-articular hip surgery: surgical technique, indications, and results of application. Instr Course Lect. 2009;58:241–55.
53. McAndrew MP, Weinstein SL. A long-term follow-up of Legg-Calve-Perthes disease. J Bone Joint Surg Am. 1984;66(6):860–9.
54. Stulberg SD, Cooperman DR, Wallensten R. The natural history of Legg-Calve-Perthes disease. J Bone Joint Surg Am. 1981;63(7):1095–108.
55. Voos JE, Ranawat AS, Pellicci PM, Buly RL, Salvati EA. Varus rotational osteotomies for adults with hip dysplasia: a 20-year followup. Clin Orthop Relat Res. 2007;457:138–43.
56. Trousdale RT, Ekkernkamp A, Ganz R, Wallrichs SL. Periacetabular and intertrochanteric osteotomy for the treatment of osteoarthrosis in dysplastic hips. J Bone Joint Surg Am. 1995;77(1):73–85.
57. Larson CM, Giveans MR, Taylor M. Does arthroscopic FAI correction improve function with radiographic arthritis? Clin Orthop Relat Res. 2011;469(6):1667–76.
58. Murphy S, Tannast M, Kim YJ, Buly R, Millis MB. Debridement of the adult hip for femoroacetabular impingement: indications and preliminary clinical results. Clin Orthop. 2004;1(429):178–81.
59. Ipach I, Mittag F, Walter C, Syha R, Wolf P, Kluba T. The prevalence of acetabular anomalies associated with pistol-grip-deformity in osteoarthritic hips. Orthop Traumatol Surg Res. 2013;99(1):37–45.
60. Dolan MM, Heyworth BE, Bedi A, Duke G, Kelly BT. CT reveals a high incidence of osseous abnormalities in hips with labral tears. Clin Orthop Relat Res. 2011;469(3):831–8.
61. Ganz R, Leunig M. Morphological variations of residual hip dysplasia in the adult. Hip Int. 2007;17 Suppl 5: S22–8.
62. Li PL, Ganz R. Morphologic features of congenital acetabular dysplasia: one in six is retroverted. Clin Orthop. 2003;416:245–53.
63. Tannast M, Kubiak-Langer M, Langlotz F, Puls M, Murphy SB, Siebenrock KA. Noninvasive three-dimensional assessment of femoroacetabular impingement. J Orthop Res. 2007;25(1):122–31.
64. Leunig M, Ganz R. The evolution and concepts of joint-preserving surgery of the hip. Bone Joint J. 2014;96(1):5–18.
65. Haverkamp D, Marti RK. Bilateral varus osteotomies in hip deformities: are early interventions superior? A long-term follow-up. Int Orthop. 2007;31(2):185–91.
66. Ito H, Matsuno T, Minami A. Intertrochanteric varus osteotomy for osteoarthritis in patients with hip dysplasia: 6 to 28 years followup. Clin Orthop Relat Res. 2005;433:124–8.
67. Iwase T, Hasegawa Y, Kawamoto K, Iwasada S, Yamada K, Iwata H. Twenty years' followup of intertrochanteric osteotomy for treatment of the dysplastic hip. Clin Orthop. 1996;331:245–55.
68. Ansari A, Jones S, Hashemi-Nejad A, Catterall A. Varus proximal femoral osteotomy for hip dysplasia in adults. Hip Int. 2008;18(3):200–6.
69. Zweifel J, Honle W, Schuh A. Long-term results of intertrochanteric varus osteotomy for dysplastic osteoarthritis of the hip. Int Orthop. 2011;35(1):9–12.
70. Koulouvaris P, Stafylas K, Aznaoutoglou C, Zacharis K, Xenakis T. Isolated varus intertrochanteric osteotomy for hip dysplasia in 52 patients: long-term results. Int Orthop. 2007;31(2):193–8.
71. Southwick WO. Osteotomy through the lesser trochanter for slipped capital femoral epiphysis. J Bone Joint Surg Am. 1967;49(5):807–35.
72. Imhauser G. Late results of Imhauser's osteotomy for slipped capital femoral epiphysis (author's transl). Z Orthop. 1977;115(5):716–25.
73. Schai PA, Exner GU. Corrective Imhauser intertrochanteric osteotomy. Oper Orthop Traumatol. 2007;19(4):368–88.
74. Kuzyk PR, Kim YJ, Millis MB. Surgical management of healed slipped capital femoral epiphysis. J Am Acad Orthop Surg. 2011;19(11):667–77.
75. Leunig M, Slongo T, Ganz R. Subcapital realignment in slipped capital femoral epiphysis: surgical hip dislocation and trimming of the stable trochanter to protect the perfusion of the epiphysis. Instr Course Lect. 2008;57:499–507.
76. Varghese VD, Boopalan PR, Titus VT, Oommen AT, Jepegnanam TS. Indices affecting outcome of neglected femoral neck fractures following valgus intertrochanteric osteotomy. J Orthop Trauma. 2014;28(7):410–6.
77. Gadegone WM, Ramteke AA, Lokhande V, Salphade Y. Valgus intertrochanteric osteotomy and fibular strut graft in the management of neglected femoral neck fracture. Injury. 2013;44(6):763–8.
78. Said GZ, Farouk O, Said HG. Valgus intertrochanteric osteotomy with single-angled 130 degrees plate fixation for fractures and non-unions of the femoral neck. Int Orthop. 2010;34(8):1291–5.
79. Poss R. The intertrochanteric osteotomy. In: Sledge CB, editor. The hip: techniques in orthopaedic surgery. Philadelphia: Lippincott-Raven; 1998. p. 183–96.
80. Maistrelli GL, Gerundini M, Fusco U, Bombelli R, Bombelli M, Avai A. Valgus-extension osteotomy for osteoarthritis of the hip. Indications and long-term results. J Bone Joint Surg Br. 1990;72(4):653–7.
81. Kawate K, Tanaka Y, Ohmura T, Hiyoshi N, Yajima H, Tomita Y, et al. Twenty-five years followup of patients who had valgus osteotomy for arthritic hips. Clin Orthop. 2004;426:151–8.

82. Bartonicek J, Vavra J. Valgus intertrochanteric osteotomy for coxa vara of Bucholz-Ogden Types II and III in patients older than 30 years. Arch Orthop Trauma Surg. 2011;131(9):1211–7.
83. Rao JP, Francis AM, Siwek CW. The treatment of chronic slipped capital femoral epiphysis by biplane osteotomy. J Bone Joint Surg Am. 1984;66(8):1169–75.
84. Maussen JP, Rozing PM, Obermann WR. Intertrochanteric corrective osteotomy in slipped capital femoral epiphysis. A long-term follow-up study of 26 patients. Clin Orthop. 1990;259:100–10.
85. Kartenbender K, Cordier W, Katthagen BD. Long-term follow-up study after corrective Imhauser osteotomy for severe slipped capital femoral epiphysis. J Pediatr Orthop. 2000;20(6):749–56.
86. Witbreuk MM, Bolkenbaas M, Mullender MG, Sierevelt IN, Besselaar PP. The results of downgrading moderate and severe slipped capital femoral epiphysis by an early Imhauser femur osteotomy. J Child Orthop. 2009;3(5):405–10.
87. Khan AQ, Khan MS, Sherwani MK, Agarwal R. Role of valgus osteotomy and fixation with dynamic hip screw and 120 degrees double angle barrel plate in the management of neglected and ununited femoral neck fracture in young patients. J Orthop Traumatol Off J Ital Soc Orthop Traumatol. 2009;10(2):71–8.
88. Rosemeyer B, Viernstein K, Schumann HJ. Follow up study of intertrochanteric valgus osteotomy with medial displacement in cases of primary protrusio acetabuly (author's transl). Arch Orthop Unfallchir. 1973;77(2):138–48.
89. McBride MT, Muldoon MP, Santore RF, Trousdale RT, Wenger DR. Protrusio acetabuli: diagnosis and treatment. J Am Acad Orthop Surg. 2001;9(2):79–88.
90. McKibbin B. Anatomical factors in the stability of the hip joint in the newborn. J Bone Joint Surg Br. 1970;52 (1):148–59.
91. Maruyama M, Feinberg JR, Capello WN, D'Antonio JA. The Frank Stinchfield Award: morphologic features of the acetabulum and femur: anteversion angle and implant positioning. Clin Orthop Relat Res. 2001;393:52–65.
92. Reikeras O, Bjerkreim I, Kolbenstvedt A. Anteversion of the acetabulum and femoral neck in normals and in patients with osteoarthritis of the hip. Acta Orthop Scand. 1983;54(1):18–23.
93. Jamali AA, Mladenov K, Meyer DC, Martinez A, Beck M, Ganz R, et al. Anteroposterior pelvic radiographs to assess acetabular retroversion: high validity of the "cross-over-sign". J Orthop Res. 2007;25 (6):758–65.

Femoro-acetabular Impingement: Definition, Etiology, Pathophysiology

50

Michael Leunig, Shin Azegami, Atul F. Kamath, and Reinhold Ganz

Contents

M. Leunig
Department of Orthopaedic Surgery, Schulthess Clinic, Zürich, Switzerland
e-mail: michael.leunig@kws.ch

S. Azegami
Royal National Orthopaedic Hospital, Stanmore, Middlesex, UK
e-mail: azegami@hotmail.com

A.F. Kamath (✉)
Department of Orthopaedic Surgery, University of Pennsylvania, Philadelphia, PA, USA
e-mail: akamath@post.harvard.edu

R. Ganz
Emeritus Inselspital Bern, Bern, Switzerland

Faculty of Medicine, University of Bern, Bern, Switzerland
e-mail: rd.ganz@bluewin.ch

Abstract

Femoroacetabular impingement (FAI) is a pathoanatomic condition which may cause pain and early degeneration of the hip, particularly in young, active adults. It has gained significant attention as a link not only to hip pain but also to the pathogenesis of early hip osteoarthritis. Hip impingement represents a pathological repetitive contact between the proximal femur and/or acetabulum which may result in injury to the labrum and/or articular cartilage. FAI tends to occur as a combination of morphological factors relevant to hip damage, in combination with vigorous activity (particularly sports) or extremes in range of motion. In some cases of FAI, the underlying structural abnormality is secondary to residual childhood hip disorders, such as Legg-Calve-Perthes disease, slipped capital femoral epiphysis, and others. In the majority of cases, there is no obvious history of previous hip pathology, and the impingement is referred to as primary FAI.

Introduction

Femoroacetabular impingement (FAI) is a condition which has received increasing recognition for its association with pain and early degeneration of the hip, particularly in young, active adults. Building upon few previous anecdotal descriptions [1–6] of hip impingement, FAI was formally

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_53

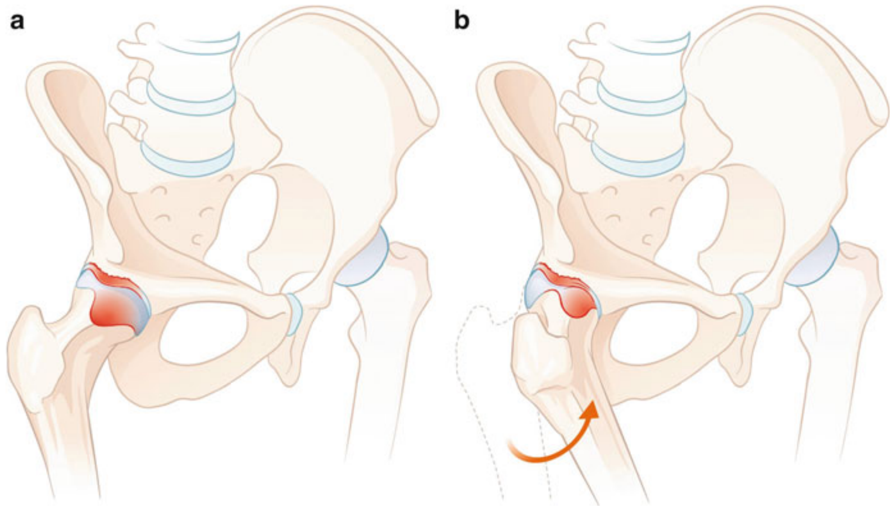


Fig. 1 (**a**–**b**). Illustration demonstrating the biomechanical concept of femoroacetabular impingement (FAI). As the hip moves from a neutral position (Fig. 1a) towards the extremes of flexion and internal rotation (Fig. 1b), there is a collision between the femur and the acetabulum. Areas particularly affected by this conflict, including the acetabular labrum, are indicated in *red*

conceptualized in 2003 [7]. FAI is a dynamic phenomenon, which results in inclusion of the femoral head and neck into the acetabulum and/or impaction of the neck against the acetabular rim secondary to abnormal morphology of the femoral head-neck junction or acetabulum or a combination of both (Fig. 1). The abnormal morphology of the femoral head-neck junction can be due to an asphericity of the femoral head and neck, termed a cam-type deformity, and/or a decreased head-neck offset. On the acetabular side, the so-called pincer-type deformity is overcoverage (focal or global) of the femoral head.

Pathological contact between the femur and acetabulum, when repetitive, may lead to damage of articular structures, including the acetabular labrum and cartilage. Individuals affected by this condition typically present with hip or groin symptoms and painful limitations in range of motion, particularly with hip internal rotation. At present, emerging evidence suggests that (1) FAI may instigate osteoarthritis (OA) of the hip and that (2) adolescents and active adults with groin pain might be successfully treated by addressing the deformities associated with FAI. The pathological hip mechanics of FAI may lead to associated dysfunction of the periarticular musculature. Increased stress loading across ligaments and other soft tissue structures about the lumbar spine, sacroiliac joints, and pubic symphysis may challenge identification of groin-related symptoms [8].

Evidence from a number of population studies, including the Cohort Hip and Cohort Knee (CHECK), Chingford cohort, and Rotterdam study, showed associations between deformities seen in FAI and later development of hip OA [9–14]. However, some patients exhibiting such hip deformities do not necessarily reach the end points of total hip arthroplasty or radiographic OA [15, 16], which suggests that other factors are involved. Moreover, in most patients presenting with hip pain, more than one pathological factor is present, pointing towards the complex interplay between impingement and even instability in many of these hips [17, 18].

Definition

The definition of the clinical condition of FAI has recently been described in detail by Sankar et al. [19]. The Medical Subject Headings thesaurus of the United States National Library of Medicine defines FAI as a clinical entity in which a pathological mechanical process causes hip pain when morphological abnormalities of the femur and/or acetabulum, combined with vigorous hip motion (especially at the extremes), lead to repetitive collisions that damage the soft tissue structures within the joint itself. This definition contains five essential elements: (1) abnormal morphology of the femur and/or acetabulum; (2) abnormal contact between the two structures; (3) vigorous, supra-physiologic motion which underlies the latter; (4) repetitive motion resulting in the continuous insult; and (5) the presence of soft tissue damage. In order to diagnose an individual with FAI accurately, three criteria must be met: the patient must (1) be symptomatic in the affected hip, as well as exhibit (2) clinical and (3) radiological features consistent with FAI. Clearly, the spectrum of disease presentation must be weighed against the signs and symptoms of the individual patient when considering the diagnosis of FAI.

Pathophysiology

Femoroacetabular impingement is not a disease per se but rather a process by which the human hip can fail [20]. A variety of abnormalities of the bony acetabulum and/or femur, combined with terminal and/or rigorous hip motion, can lead to repetitive collisions that damage the soft tissue structures (labrum and/or cartilage) at the acetabular rim. More than one century ago, collisions between the femur and the acetabulum had been reported anecdotally [1–6]; these reports described intra-articular damage of the hip caused by a pathological relationship between the proximal femur and the acetabulum as sequelae of childhood disorders and femoral neck fracture. As these reports were merely single observations without conceptual follow-up, it was not until the introduction of a surgical technique for dislocating the hip that direct observations could be made of such pathomechanical process [7]. These observations and clinical follow-up lead to the development of the concept of FAI as a mechanical cause of hip OA. The concept of FAI, as pioneered by Ganz et al. [7], associates subtle, often unrecognized developmental alterations of the hip with the subsequent development of OA. This theory has been compared to previous work [1, 3, 5, 21] which has described secondary OA in the setting of grossly visible deformities (e.g., acetabular dysplasia, femoral pistol grip, and head tilt).

Based upon intraoperative inspection of patients suffering from hip pain, Ganz et al. [7] described two distinct types of intra-articular pathologies frequently found in these patients. The pathomechanical concept was called FAI based upon the mechanism of joint damage leading to a distinct pattern of chondral and labral lesions of the hip. Femoral-sided pathomorphologies were called cam-type FAI, and acetabular-sided alterations were termed pincer-type FAI. These two types of impingement can coexist, and a wide variation in the frequency may also exist across different ethnic and racial subgroups.

Another mechanical distinction within the diagnosis of FAI is inclusion injuries (cam-type FAI) and impaction injuries, which are due to pincer FAI but also may be caused by a decreased head-neck offset. As the terms cam- and pincer-type FAI are well established in the literature, this report will stay within the confines of this terminology. In a Swiss cohort described by Beck et al., 47 % (27 of 57 hips) with cam-type impingement had an associated acetabular deformity, and 63 % (34 of 54 hips) with pincer-type impingement had an abnormally shaped femoral head [22].

Cam-type impingement is caused by insufficient concavity of the femoral head-neck junction anterolaterally. Therefore, this region of the femoral head has an increased radius of curvature that cannot be accommodated by the tightly congruent acetabulum. As a consequence, the femoral head, with its abnormal morphology, repetitively collides with the rim of the acetabulum during supra-physiologic motions. These collisions lift up the labrum; rather than predominantly direct damage, a shearing of the adjacent articular cartilage may potentially disrupt the chondrolabral junction. This process can cause the labrum and articular cartilage to delaminate from the subchondral bone, which is seen most frequently at the anterosuperior aspect of the acetabular rim

[23]. Intraoperative findings suggest that the predominant lesion due to cam FAI is delamination of the cartilage, while the labral tissue is relatively spared [23].

Pincer-type impingement is an acetabular-based deformity caused by over-coverage of the femoral head by the acetabulum. The over-coverage may be focal (cephalad retroversion or true acetabular retroversion) or global (acetabular protrusion), depending on the underlying pathoanatomy. The deformity leads to contact of the labrum against the femoral neck during hip motion [24]. The mechanical features of pincer-type FAI are different from those of cam-type FAI: the contact between the proximal femur and the acetabulum is linear, leading to failure of the labrum, but damage to the articular cartilage is initially limited to the acetabular rim [23]. Pincer-type impingement caused by focal over-coverage may be secondary to cephalad over-coverage or true acetabular retroversion, with only the posterior wall insufficiently covering the posterior femoral head. Both conditions present with similar clinical symptoms; however, true acetabular retroversion represents an entirely different mechanical problem of over-coverage anteriorly combined with under-coverage posteriorly e.g., in PFFD hips or posttraumatic dysplasia (Lit). In the case of acetabular protrusion, global over-coverage of the hip leads to impingement that occurs circumferentially around the rim.

Using the cam-/pincer-type classification, not all damage mechanisms are sufficiently covered, as they describe merely bony morphological abnormalities, such as the asphericity of the femoral head (cam type) or the focal/global over-coverage (pincer type). This schema does not include extracapsular alterations, such as varus or valgus femoral deformity or femoral torsion. Using inclusion-type or impaction-type joint damage places focus on the damage mechanism, and therefore, all impingement-causing deformities should be covered in theory. Of note, in the presence of combinations of deformities, it may be difficult to decide which condition (impaction or inclusion) predominates.

The prevalence of cam and pincer impingement has been found to be substantially higher in college football players compared to the reported prevalence in the general population [25]. This may suggest that the former group may have a higher propensity to develop symptomatic FAI related to higher body mass index (BMI) and activity level, leading to greater loads on the hip and with abnormal morphology contributing to the labral and articular cartilage damage. In one study, 39 % of asymptomatic professional and collegiate hockey players were found to have an abnormally high alpha angle [26]. Seventy-two percent of male and 50 % of female professional soccer players had at least one radiographic abnormality that could cause FAI [27]. Although these athletes were asymptomatic at the time of radiographic study, 50 % of men and 25 % of women reported a past groin or hip injury [27].

There is some evidence that increased stress on the proximal femoral physis around the time of closure is responsible for the cam deformity [28]. In a study of elite adolescent soccer players, 26 % had an alpha angle $>60°$, 13 % had a prominence at the head-neck junction, and 53 % had flattening; this was in comparison to rates of 17 %, 0 %, and 18 %, respectively, for these measures in nonathlete control patients [29]. Elite club basketball players who had played basketball year-round since age 8 were found to have greater mean alpha angles than controls, as well as higher mean alpha angles after physeal closure [30]. Clinical examination of these athletes revealed that 48 % had pain on impingement testing, and 19 % of the athletes reported hip or groin pain in the preceding 6 months [30]. In comparison, 1.3 % of the controls had a positive impingement test. In a case-control study of elite basketball players and age-matched nonathletes, Siebenrock et al. presented evidence for growth plate alterations, rather than simply reactive bone formation, contributing to cam-type deformity in the athletic subgroup [31].

Additional Causative Factors for Impingement

Relative (less than 15° but greater than 0°) or absolute (less than 0°) retroversion of the femur is a distinct dynamic factor that should be considered in the evaluation of mechanical causes of hip pain. Even in the absence of a cam or pincer lesion, femoral retroversion can reduce the internal rotation of the hip [32]. Femoral retroversion can amplify the effect of a coexisting cam or pincer lesion by rotating the cam into the acetabular rim at an earlier phase of hip flexion. Conversely, femoral anteversion may mitigate the effect of cam-type impingement but may cause mechanical hip pain through increased static stress or instability at the anterior acetabulum [8].

Femoral varus (low femoral neck-shaft angle) may aggravate a preexisting cam deformity, similar to femoral retrotorsion, but, in addition, varus leads to relative shortening of the femoral neck and prominence of the greater trochanter. This can result in extra-articular impingement of the greater trochanter against the anterior inferior iliac spine and soft tissues [8]. The deformity can also amplify the intra-articular, lateral impingement of a superolateral cam and/or rim impingement lesion.

Etiology

In many cases of FAI, the underlying structural abnormality is secondary to residual deformity from the sequelae of childhood hip disorders, such as Legg-Calve-Perthes disease (LCPD), slipped capital femoral epiphysis (SCFE), and hip dysplasia. However, in most cases of FAI, there is no history of previous hip pathology, and the impingement etiology may be considered as primary FAI. The prevalence of radiographic signs of classic FAI (ranging between 10 % and 74 %) is high in the asymptomatic, or pain-free, population. Asphericities of the femoral head-neck (cam-type deformities) vary between the sexes, with males having three to five times the rates of those seen in females [33–36] and bilateral cam-type deformities being more prevalent in males [36]. Pincer-type deformities, such as acetabular over-coverage or increased acetabular depth, are observed in 10–15 % of Caucasian hips and, when present, were bilateral in 75–77 % of cases [34–37].

There are various theories to explain the development of the so-called primary FAI. However, the etiology of this condition is still open to speculation. The morphological abnormalities seen in primary cam-type impingement is similar to the deformity seen in SCFE and LCPD. Therefore, it has been suggested that these conditions may have occurred subclinically during development. Other propositions include abnormal bone extension from the epiphysis, abnormal patterns of ossification owing to congenital factors, low-grade infection, or autoimmune reactions [30, 31, 38]. High-intensity sports activity causing physeal stress surrounding adolescence has also been suggested as a risk factor for the development of cam-type deformity: observations have found FAI more frequently in athletes than in age-matched individuals not participating in high-level sports [30, 31, 38].

Genetic influences have also been implicated in the development of primary FAI [39]. Pollard et al. screened 96 siblings of 64 patients with primary FAI clinically and radiologically for the presence of cam and pincer lesions. They found that the siblings of patients with a cam deformity or a pincer deformity had a relative risk ratio of 2.8 and 2.0, respectively, of having the same deformity. It is unclear whether the genetic component determines the deformity at conception or if it predisposes to abnormal development or subclinical hip pathology before skeletal maturity.

Childhood diseases, in particular SCFE and LCPD, can cause secondary pincer- and cam-type FAI. The same is true for acetabular protrusion, which leads to global pincer FAI. Protrusion may be caused by metabolic or inflammatory disease, although for some no such definitive etiology can be found. In SCFE, FAI occurs between the prominent metaphysis and the acetabular rim, as confirmed by intraoperative findings during surgical hip dislocation from various reports [40, 41]. This has revealed that, even in mild stable slips, there is evidence of labral injury and chondromalacia in the anterosuperior quadrant of the acetabulum. These studies highlight the potential need to address proximal femoral deformity leading to cam and pincer FAI (impaction and inclusion) in SCFE, in order to prevent subsequent OA due to FAI. There exists, however, controversy surrounding the remodeling potential of residual SCFE deformity. Some authors believe that metaphyseal remodeling can be expected with time, resulting in minimal consequence of the deformity, and that FAI will not develop in most patients. The latter concept is supported by a long-term follow-up study by Boyer et al. [42].

Residual deformity in LCPD is a cause of hip pain in adolescents and young adults owing to the altered mechanics of the hip joint. The condition results in abnormal femoral head shape, growth disturbance of the proximal femoral physis, and acetabular dysplasia due to secondary remodeling processes. The symptoms may be related to hip joint instability, FAI, or a combination of both [43]. The cause of FAI in LCPD is the due to the aspherical femoral head [44, 45], acetabular retroversion [46, 47], or secondary to surgical procedures like innominate osteotomy [48]. These can lead to intra-articular impingement, but extra-articular impingement can also result from overgrowth of the greater trochanter or, less commonly, impingement of the lesser trochanter due to a shortened femoral neck [43].

The majority of studies on the progression of deformity in children with SCFE and LCPD have focused on the proximal femur, but a number of more recent studies have demonstrated that FAI can occur in both conditions as a result of acetabular retroversion, which places the patient at risk for developing pincer-type impingement [46, 47, 49]. Sankar et al. have found that the prevalence of acetabular retroversion is rare in children with LCPD before skeletal maturity; rather, this retroversion developed over time in a child with a deformed femoral head and suggested a cause-and-effect relationship [47]. A high prevalence of acetabular retroversion in proximal femoral focal deficiency and bladder exstrophy has also been reported [43].

The mechanism of hip damage in acetabular protrusion has been explained by higher load transmission through the medial aspect of the joint. Even in minimal primary protrusion, the femoral head has been observed to migrate medially over time due to medial dysplasia. Of potential more importance for the development of OA, a direct abutment of the femoral neck against the acetabular rim causing pincer FAI occurs in protrusion and causes direct damage to the labrum and acetabular cartilage, with indirect posteroinferior cartilage damage by leverage [50]. Pincer impingement is seen in young patients secondary to a relatively deep acetabulum, such as coxa profunda or acetabular protrusion and acetabular retroversion.

There are additional posttraumatic and iatrogenic causes of FAI, such as acetabular dysplasia [48], femoral neck fracture [51], and iatrogenic deformities created by procedures such as femoral varus osteotomy and pelvic osteotomy [52], that can lead to secondary FAI. Other reported causes of secondary FAI include exostosis and osteoid osteoma [53].

Summary

FAI is a pathoanatomic mechanical hip condition which has received significant attention due to growing evidence that it predisposes to early-onset hip osteoarthritis in a subgroup of patients. FAI may occur as the sequelae of previous childhood hip disorders, such as SCFE or LCPD (secondary FAI). More frequently, it occurs in the absence of any obvious history (primary FAI). Because a significant proportion of patients with primary FAI may go on to develop hip OA, it will be important to determine which individuals with FAI-related morphological abnormalities are at greatest risk of developing hip OA at a relatively young age.

References

1. Harris WH. Etiology of osteoarthritis of the hip. Clin Orthop Relat Res. 1986;213:20–33.
2. Heyman CH, Herndon CH, Strong JM. Slipped femoral epiphysis with severe displacement; a conservative operative treatment. J Bone Joint Surg Am. 1957;39-A (2):293–303. passim.
3. Murray RO. The aetiology of primary osteoarthritis of the hip. Br J Radiol. 1965;38(455):810–24.
4. Smith-Petersen MN. Treatment of malum coxae senilis, old slipped upper femoral epiphysis, intrapelvic protrusion of the acetabulum, and coxa plana by means

of acetabuloplasty. J Bone Joint Surg Am. 1936;18 (4):869–80.
5. Solomon L, Schnitzler CM, Browett JP. Osteoarthritis of the hip: the patient behind the disease. Ann Rheum Dis. 1982;41(2):118–25.
6. Vulpius O, Stoffel A. Orthopädische operationslehre. 3rd ed. Stuttgart: Enke; 1924.
7. Ganz R, Parvizi J, Beck M, Leunig M, Notzli H, Siebenrock KA. Femoroacetabular impingement: a cause for osteoarthritis of the hip. Clin Orthop Relat Res. 2003;417:112–20.
8. Bedi A, Dolan M, Leunig M, Kelly BT. Static and dynamic mechanical causes of hip pain. Arthrosco J Arthrosc Relat Sur. 2011;27(2):235–51.
9. Agricola R, Heijboer MP, Bierma-Zeinstra SM, Verhaar JA, Weinans H, Waarsing JH. Cam impingement causes osteoarthritis of the hip: a nationwide prospective cohort study (CHECK). Ann Rheum Dis. 2013;72(6):918–23.
10. Allen D, Beaule PE, Ramadan O, Doucette S. Prevalence of associated deformities and hip pain in patients with cam-type femoroacetabular impingement. J Bone Joint Surg Br. 2009;91(5):589–94.
11. Bardakos NV, Villar RN. Predictors of progression of osteoarthritis in femoroacetabular impingement: a radiological study with a minimum of ten years follow-up. J Bone Joint Surg Br. 2009;91(2):162–9.
12. Clohisy JC, Dobson MA, Robison JF, Warth LC, Zheng J, Liu SS, Yehyawi TM, Callaghan JJ. Radiographic structural abnormalities associated with premature, natural hip-joint failure. J Bone Joint Surg Am. 2011;93 Suppl 2:3–9.
13. Nicholls AS, Kiran A, Pollard TC, Hart DJ, Arden CP, Spector T, Gill HS, Murray DW, Carr AJ, Arden NK. The association between hip morphology parameters and nineteen-year risk of end-stage osteoarthritis of the hip: a nested case–control study. Arthritis Rheum. 2011;63(11):3392–400.
14. Reichenbach S, Leunig M, Werlen S, Nuesch E, Pfirrmann CW, Bonel H, Odermatt A, Hofstetter W, Ganz R, Juni P. Association between cam-type deformities and magnetic resonance imaging-detected structural hip damage: a cross-sectional study in young men. Arthritis Rheum. 2011;63(12):4023–30.
15. Audenaert EA, Peeters I, Van Onsem S, Pattyn C. Can we predict the natural course of femoroacetabular impingement? Acta Orthop Belg. 2011;77(2):188–96.
16. Hartofilakidis G, Bardakos NV, Babis GC, Georgiades G. An examination of the association between different morphotypes of femoroacetabular impingement in asymptomatic subjects and the development of osteoarthritis of the hip. J Bone Joint Surg Br. 2011;93 (5):580–6.
17. Tibor LM, Ganz R, Leunig M. Case reports: anteroinferior acetabular rim damage due to femoroacetabular impingement. Clin Orthop Relat Res. 2013;471(12):3781–7.
18. Tibor LM, Liebert G, Sutter R, Impellizzeri FM, Leunig M. Two or more impingement and/or instability deformities are often present in patients with hip pain. Clin Orthop Relat Res. 2013;471(12):3762–73.
19. Sankar WN, Nevitt M, Parvizi J, Felson DT, Agricola R, Leunig M. Femoroacetabular impingement: defining the condition and its role in the pathophysiology of osteoarthritis. J Am Acad Orthop Surg. 2013;21 Suppl 1:S7–15.
20. Leunig M, Beaule PE, Ganz R. The concept of femoroacetabular impingement: current status and future perspectives. Clin Orthop Relat Res. 2009;467(3): 616–22.
21. Stuhlberg SD. Unrecognized childhood hip disease: a major cause of idiopathic osteoarthritis of the hip. In: Cordell LD, Harris WH, Ramsey PL, McEwen GD, editors. The hip: proceedings of the third open scientific meeting of the hip society. St Louis: CV Mosby; 1975. p. 212–228.
22. Beck M, Leunig M, Parvizi J, Boutier V, Wyss D, Ganz R. Anterior femoroacetabular impingement: part II. Midterm results of surgical treatment. Clin Orthop Relat Res. 2004;418:67–73.
23. Beck M, Kalhor M, Leunig M, Ganz R. Hip morphology influences the pattern of damage to the acetabular cartilage: femoroacetabular impingement as a cause of early osteoarthritis of the hip. J Bone Joint Surg Br. 2005;87(7):1012–8.
24. Siebenrock KA, Schoeniger R, Ganz R. Anterior femoro-acetabular impingement due to acetabular retroversion. Treatment with periacetabular osteotomy. J Bone Joint Surg Am. 2003;85-A(2):278–86.
25. Kapron AL, Anderson AE, Aoki SK, Phillips LG, Petron DJ, Toth R, Peters CL. Radiographic prevalence of femoroacetabular impingement in collegiate football players: AAOS Exhibit Selection. J Bone Joint Surg Am. 2011;93(19):e111(111–110).
26. Silvis ML, Mosher TJ, Smetana BS, Chinchilli VM, Flemming DJ, Walker EA, Black KP. High prevalence of pelvic and hip magnetic resonance imaging findings in asymptomatic collegiate and professional hockey players. Am J Sports Med. 2011;39(4):715–21.
27. Gerhardt MB, Romero AA, Silvers HJ, Harris DJ, Watanabe D, Mandelbaum BR. The prevalence of radiographic hip abnormalities in elite soccer players. Am J Sports Med. 2012;40(3):584–8.
28. Kienle KP, Keck J, Werlen S, Kim YJ, Siebenrock KA, Mamisch TC. Femoral morphology and epiphyseal growth plate changes of the hip during maturation: MR assessments in a 1-year follow-up on a cross-sectional asymptomatic cohort in the age range of 9–17 years. Skeletal Radiol. 2012;41(11): 1381–90.
29. Agricola R, Bessems JH, Ginai AZ, Heijboer MP, van der Heijden RA, Verhaar JA, Weinans H, Waarsing JH. The development of cam-type deformity in adolescent and young male soccer players. Am J Sports Med. 2012;40(5):1099–106.
30. Siebenrock KA, Ferner F, Noble PC, Santore RF, Werlen S, Mamisch TC. The cam-type deformity of the proximal femur arises in childhood in response to

vigorous sporting activity. Clin Orthop Relat Res. 2011;469(11):3229–40.
31. Siebenrock KA, Behning A, Mamisch TC, Schwab JM. Growth plate alteration precedes cam-type deformity in elite basketball players. Clin Orthop Relat Res. 2013;471(4):1084–91.
32. Yoo WJ, Choi IH, Cho TJ, Chung CY, Park MS, Lee DY. Out-toeing and in-toeing in patients with Perthes disease: role of the femoral hump. J Pediatr Orthop. 2008;28(7):717–22.
33. Hack K, Di Primio G, Rakhra K, Beaule PE. Prevalence of cam-type femoroacetabular impingement morphology in asymptomatic volunteers. J Bone Joint Surg Am. 2010;92(14):2436–44.
34. Kang AC, Gooding AJ, Coates MH, Goh TD, Armour P, Rietveld J. Computed tomography assessment of hip joints in asymptomatic individuals in relation to femoroacetabular impingement. Am J Sports Med. 2010;38(6):1160–5.
35. Gosvig KK, Jacobsen S, Sonne-Holm S, Palm H, Troelsen A. Prevalence of malformations of the hip joint and their relationship to sex, groin pain, and risk of osteoarthritis: a population-based survey. J Bone Joint Surg Am. 2010;92(5):1162–9.
36. Laborie LB, Lehmann TG, Engesaeter IO, Eastwood DM, Engesaeter LB, Rosendahl K. Prevalence of radiographic findings thought to be associated with femoroacetabular impingement in a population-based cohort of 2081 healthy young adults. Radiology. 2011;260(2):494–502.
37. Leunig M, Juni P, Werlen S, Limacher A, Nuesch E, Pfirrmann CW, Trelle S, Odermatt A, Hofstetter W, Ganz R, Reichenbach S. Prevalence of cam and pincer-type deformities on hip MRI in an asymptomatic young Swiss female population: a cross-sectional study. Osteoarthritis Cartilage. 2013;21(4):544–50.
38. Murray RO, Duncan C. Athletic activity in adolescence as an etiological factor in degenerative hip disease. J Bone Joint Surg Br. 1971;53(3):406–19.
39. Pollard TC, Villar RN, Norton MR, Fern ED, Williams MR, Murray DW, Carr AJ. Genetic influences in the aetiology of femoroacetabular impingement: a sibling study. J Bone Joint Surg Br. 2010;92(2):209–16.
40. Leunig M, Casillas MM, Hamlet M, Hersche O, Notzli H, Slongo T, Ganz R. Slipped capital femoral epiphysis: early mechanical damage to the acetabular cartilage by a prominent femoral metaphysis. Acta Orthop Scand. 2000;71(4):370–5.
41. Sink EL, Zaltz I, Heare T, Dayton M. Acetabular cartilage and labral damage observed during surgical hip dislocation for stable slipped capital femoral epiphysis. J Pediatr Orthop. 2010;30(1):26–30.
42. Boyer DW, Mickelson MR, Ponseti IV. Slipped capital femoral epiphysis. Long-term follow-up study of one hundred and twenty-one patients. J Bone Joint Surg Am. 1981;63(1):85–95.
43. Kim YJ, Novais EN. Diagnosis and treatment of femoroacetabular impingement in Legg-Calve-Perthes disease. J Pediatr Orthop. 2011;31(2 Suppl):S235–40.
44. Eijer H, Berg RP, Haverkamp D, Pecasse GA. Hip deformity in symptomatic adult Perthes' disease. Acta Orthop Belg. 2006;72(6):683–92.
45. Snow SW, Keret D, Scarangella S, Bowen JR. Anterior impingement of the femoral head: a late phenomenon of Legg-Calve-Perthes' disease. J Pediatr Orthop. 1993;13(3):286–9.
46. Ezoe M, Naito M, Inoue T. The prevalence of acetabular retroversion among various disorders of the hip. J Bone Joint Surg Am. 2006;88(2):372–9.
47. Sankar WN, Flynn JM. The development of acetabular retroversion in children with Legg-Calve-Perthes disease. J Pediatr Orthop. 2008;28(4):440–3.
48. Dora C, Mascard E, Mladenov K, Seringe R. Retroversion of the acetabular dome after Salter and triple pelvic osteotomy for congenital dislocation of the hip. J Pediatr Orthop B. 2002;11(1):34–40.
49. Monazzam S, Krishnamoorthy V, Bittersohl B, Bomar JD, Hosalkar HS. Is the acetabulum retroverted in slipped capital femoral epiphysis? Clin Orthop Relat Res. 2013;471(7):2145–50.
50. Leunig M, Nho SJ, Turchetto L, Ganz R. Protrusio acetabuli: new insights and experience with joint preservation. Clin Orthop Relat Res. 2009;467(9):2241–50.
51. Eijer H, Myers SR, Ganz R. Anterior femoroacetabular impingement after femoral neck fractures. J Orthop Trauma. 2001;15(7):475–81.
52. Myers SR, Eijer H, Ganz R. Anterior femoroacetabular impingement after periacetabular osteotomy. Clin Orthop Relat Res. 1999;363:93–9.
53. Herget GW, Sudkamp NP, Bohm J, Helwig P. Osteoid osteoma of the femoral neck mimicking monarthritis and causing femoroacetabular impingement. Acta Chir Orthop Traumatol Cech. 2012;79(3):275–8.

Surgical Technique: Open Acetabular Rim Trimming, Labral Refixation, Open Femoral Osteochondroplasty

51

Luca Gala and Paul E. Beaulé

Contents

L. Gala • P.E. Beaulé (✉)
The Ottawa Hospital, Adult Reconstruction, University of Ottawa, Ottawa, ON, Canada
e-mail: lgala@toh.on.ca; pbeaule@ottawahospital.on.ca

Abstract

Femoroacetabular impingement (FAI) of the hip is an osseous abnormality of the proximal femur and/or acetabulum as described by Ganz. Although no longer the most commonly used approach for the treatment of cam FAI, open surgical dislocation is the most established technique for hip preservation surgery and is a more powerful tool than arthroscopy as it allows the surgeon to address any atypical or complex intra- and extra-articular pathology and can be combined to a concomitant femoral or pelvic osteotomy. This surgery is performed through a Gibson approach, the femoral head is dislocated after a trochanteric osteotomy (trochanteric slide), and the whole head-neck junction is perfectly visualized as well as the acetabulum. This chapter describes the surgical setup, the dissection, the location of the neurovascular structure at risk, and how to avoid complications.

Introduction

Femoroacetabular impingement (FAI) of the hip is an osseous abnormality of the proximal femur and/or acetabulum as described by Ganz et al. [1–3]. The process of motion leads to a forceful contact between the femoral head and neck with the acetabulum that may cause hip pain, labral tears, cartilage delamination, and potentially osteoarthritis later in life [4–8]. The prevalence has

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_54

been shown to be 10–15 % in young active patients and up to 94 % of young patients with hip pain [9]. Although the pathomechanism leading to the onset of symptoms is still being studied, it is clear that the severity of the cam lesion is a significant risk factor for the development of hip pain as well as cartilage damage.

In a follow-up study, 170 of 200 returned the questionnaire in regard to a new onset of hip pain, and those with internal rotation of less than 20° had an odds ratio of 3.1 ($p = 0.006$) of developing hip pain [10]. These findings are also consistent with the recent paper by Kapron et al. [11] in collegiate football athletes. There is a consistent biological gradient associated with the alpha angle severity and risk of articular pathology. In several studies, the severity of the alpha angle was predictive of intraoperative severity of acetabular cartilage damage (as well as the risk of developing hip pain in individuals with a previously asymptomatic cam deformity [12–16]). Alpha angle values greater than 60° represented the greatest risk of developing hip pain as well as leading to more severe acetabular cartilage damage.

Although no longer the most commonly used approach for the treatment of cam FAI, the surgical dislocation approach described by Ganz [31] was pivotal in our understanding of the pathomechanism of FAI as well as establishing its principals of treatment. And even though hip arthroscopy is now being hailed as the preferred technique to treat cam-type FAI as well as minor forms of pincer, one should not underestimate the technical demands of this surgical technique [2, 3, 17, 18].

Philippon et al. [19] and Heyworth et al. [20] reported that the most common reason for revision surgery is the failure to completely address the bony impingement lesions of the hip. Open surgical dislocation is the most established technique for hip preservation surgery and is a more powerful tool than arthroscopy as it allows the surgeon to address any atypical or complex intra- and extra-articular pathology and can be combined to a concomitant femoral or pelvic osteotomy [21]. Favorable good to excellent midterm clinical outcomes of 70–80 % have been reported in the literature [22–30].

Surgical Technique

The patient is placed in a lateral decubitus position as described by Ganz et al. [31]. A Gibson approach is performed so the anterior muscle fibers of the gluteus maximus are not split and the neurovascular supply is not at risk [32, 33]. The skin incision should be centered over the tip of the greater trochanter (GT) and runs through the anterior one-third of the GT (approximately 20 cm). The subcutaneous tissue is then cut until the iliotibial band and the fascia over the gluteus maximus muscle are reached. Branches from the inferior gluteal artery that run within the fascia between the tensor and the gluteus maximus can help us identifying the anterior border of the gluteus maximus. This fascia is kept with the gluteus maximus in order to protect branches of the inferior gluteal nerve that accompany the vessels. The tissue over the GT is exposed and incised at its posterior border, then reflected anteriorly, away from the trochanteric crest, allowing visualization of the vastus lateralis ridge. After exposure the trochanteric branch of the medial femoral circumflex artery can be seen and coagulated before performing the trochanteric flip. The hip is prepared for the trochanteric osteotomy by internal rotation of the joint (20–30°) and identification of the posterior borders of the gluteus medius and GT; it is sized at a thickness of approximately 15 mm to allow stable reattachment to the trochanteric base. To facilitate the reduction of the trochanteric fragment, one can predrill one of the screw holes. Two 4.5 mm screws are typically used. The goal is to keep the insertions of the gluteus medius, gluteus minimus, and vastus lateralis on the trochanteric fragment. The major part of the piriformis insertion as well as the other external rotators remains on the femoral side of the osteotomized trochanter (stable trochanter). It is important to keep in mind that the deep branch of the medial femoral circumflex artery reaches the trochanter just proximal to the quadratus femoris [34]. The osteotomy follows a line that starts at the posterosuperior edge of the GT and is extended distally toward the posterior border of the vastus lateralis muscle, with a plane parallel to the lower limb. Proximally, the osteotomy starts about 5 mm anterior to the most posterior insertion of the gluteus medius muscle onto the tip of the trochanter. A thin oscillating saw is used to perform the osteotomy, but it should stop at the anterior cortex, and then an osteotome is used to complete the osteotomy [31, 35]. To enhance stability and facilitate anatomic reduction of the trochanteric fragment, Notzli introduced a stepped osteotomy. This osteotomy allows a more anatomic fixation and a more aggressive postoperative mobilization [28, 36]. A small Hohmann retractor is then placed over the anterior edge of the stable trochanter, and the trochanteric fragment is flipped anteriorly with the attached medius proximally and vastus lateralis distally. Eventually, fibers of the piriformis tendon that remain attached to the trochanteric fragment must be cut to allow for its further mobilization. The lower limb is now flexed and externally rotated, allowing more anterior retraction of the mobile trochanter. The vastus lateralis and the vastus intermedius are lifted off the lateral and anterior aspects of the proximal part of the femur. The gluteus medius muscle is gently retracted in an anterosuperior direction. The capsule is approached within the interval between the piriformis and the gluteus minimus. The gluteus minimus tendon is detached from the capsule. Now, the posterior, superior, and anterior aspects of the joint capsule are exposed. The insertions of the short external rotator muscles and the piriformis muscle are left intact, protecting the deep branch of the medial femoral circumflex artery. A z-shaped capsulotomy is performed with the leg flexed and externally rotated, with an inside-out technique to avoid injury to the labrum or cartilage. The incision starts at the posterior acetabular rim and then along the anterolateral femoral neck to the anteromedial femoral neck; extreme care should be taken to avoid injury to the medial femoral circumflex artery, which runs posterosuperior to the lesser trochanter. Injury to the small branches of the lateral femoral circumflex artery can result from incision of the inferomedial aspect of the capsule, but they do not contribute to the perfusion of the femoral head. Additionally, care must be taken not to damage the underlying labrum. The hip should be taken through a full range of passive motion to most precisely locate the area of impingement. This step should always be performed prior to dislocation so that the surgeon may better visualize the area where osteochondroplasty must be performed. To assess the acetabulum, the femoral head is subluxated anteriorly by flexion and external rotation with the use of a bone hook that is placed around the calcar. A curved pair of scissors is used to cut the ligamentum teres allowing a complete anterior dislocation. External rotation aids in opening up the anterior joint space and tensioning the ligament for easier transection. Now, it is possible to fully evaluate the femoral head-neck junction as well as to probe the labrum and adjacent acetabular cartilage. Lowering the knee lets the femoral head rise automatically out of the surgical site, permitting its full inspection (Fig. 1). For visualization of the acetabulum, the knee is brought higher than the pelvis, and a gentle axial push allows the head to go posteriorly, creating enough space to view the acetabulum in its entirety. Three retractors are inserted. One double-angled Hohmann retractor is placed over the anterior rim of the acetabulum, a second straight Hohmann on the anterosuperior rim, and a cobra retractor into the teardrop to retract the femoral neck posteroinferiorly. With a blunt probe, the integrity of the labrum and the articular cartilage is determined. The labrum can be released temporarily and preserved for later refixation and the osseous overcoverage can be resected [37, 38]. If the labrum appears significantly diseased, it should be resected and reconstructed using IT band or the ligamentum teres; otherwise, attempts to mobilize and preserve the labrum should be attempted in order to preserve normal function of the hip joint [39–41].

The amount of acetabular rim resection is determined on the basis of the magnitude of the damage to the acetabular cartilage and/or the degree of overcoverage, but resection should be less rather than more in order to avoid any instability of the hip joint. Currently, the senior author will perform rim resection for exposed subchondral bone secondary to acetabular cartilage delamination if associated with acetabular

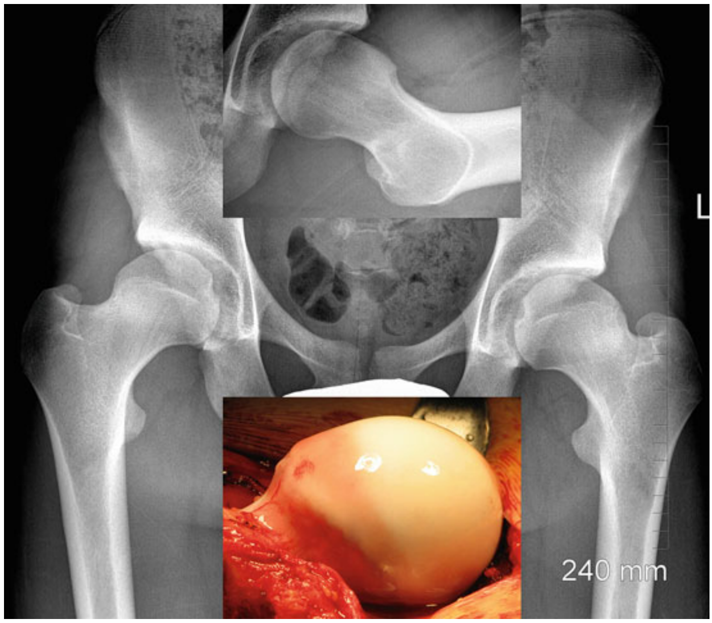


Fig. 1 Surgically dislocated hip demonstrating CAM lesion (*inset-bottom*) with preoperative anteroposterior radiograph with Dunn view (*inset-top*) and of a 24-year-old male

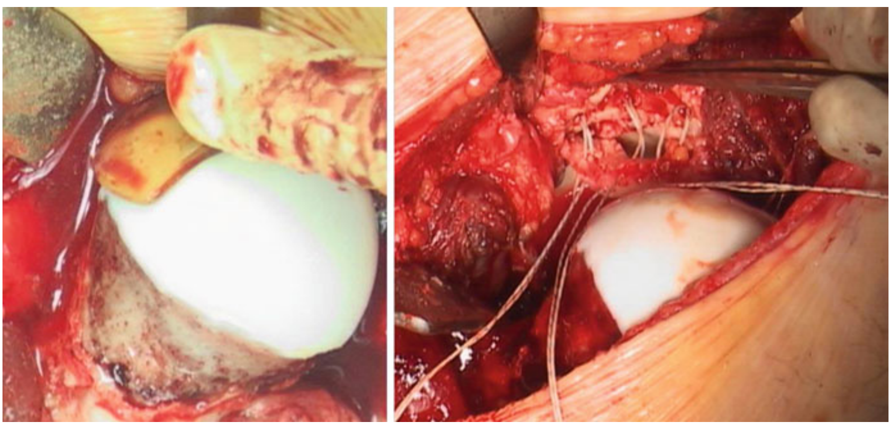


Fig. 2 Surgically corrected CAM lesion with suture anchors has been placed after acetabular rim trimming

retroversion and >3 mm in depth. In the absence of cartilage delamination, acetabular rim trimming with labral refixation will be performed if the crossover sign extends more than 5 mm from acetabular roof. The labrum is taken down using an 11 blade and then retracted using a blunt probe.

The resection is performed with use of a curved 10-mm osteotome or high-speed burr, normally with exposure of the well-bleeding cancellous bone of the acetabular rim. If an area of acetabular cartilage damage persists, microfracture can be performed. Most acetabular rim lesions are located anterosuperiorly and require two to four bone anchors to reattach the labrum. It is important to note that the refixation of the tip of the labrum (not labral repair) requires a base of bleeding cancellous bone. Positioning of the anchors is performed under direct vision, about 2 mm away from the bone-cartilage interface. Knots are tied on the outer surface, with the suture being passed through the base of the labrum (Fig. 2). After acetabular rim trimming and labral refixation, the acetabulum is carefully irrigated to remove all osseous and fibrous debris. The nonspherical portion of the femoral head is assessed with the use of transparent spherical templates and usually is located anterosuperiorly and with a reddish appearance of the cartilage. Gentle removal of excess bone and recreation of a smooth femoral neck are performed with small curved chisels and repetitive assessment with templates. Excessive bone removal during the offset procedure should be avoided, although a resection of <30 % of the neck diameter has been reported to not weaken the femoral neck [42]. Furthermore, excessive resection may compromise the sealing function of the labrum. To address cam lesions extending superiorly or posterosuperiorly, the penetrating retinacular vessels were elevated and protected under direct visualization during the resection. Perfusion of the femoral head is checked by observation of the bleeding coming from the foveolar artery or the resection surface, and laser Doppler flowmetry is also possible [43]. Although in the initial experience of surgical dislocation a thin layer of bone wax was placed over the bleeding cancellous bone to avoid adhesions to the capsule, a recent report on allergic reaction has led to the abandonment of this technique [44].

Following reduction, the hip should once again be taken through a full range of motion paying special attention to those positions that were noted to cause impingement prior to dislocation [45]. If adequate resection of the offending lesions has been performed, the hip should be able to be taken through a full range of motion with no further impingement. The capsule is closed avoiding any tension because this may stretch the retinaculum and adversely influence the perfusion of the femoral head. The trochanteric fragment is anatomically reduced and fixed with two or three 4.5-mm cortical screws; the screws are best aimed toward the lesser trochanter. An intraoperative radiograph is used to confirm proper screw length/position and trochanteric reduction. Thereafter, the various soft tissue layers are closed with running or single sutures. Potential complications include trochanteric nonunion, heterotopic ossification, trochanteric bursitis, sciatic nerve palsy, osteonecrosis of the femoral head, femoral neck fractures, as well as intra-articular adhesions [22, 46, 47].

Postoperatively the patient should be restricted to toe-touch weight bearing on the operative extremity for 6–8 weeks until there is solid union of the trochanteric osteotomy site. During the same period, the patient receives deep vein thrombosis prophylaxis. Nonsteroidal anti-inflammatory drugs were given for 2 weeks postoperatively as heterotopic ossification prophylaxis. Physical therapy may be started immediately using passive range of motion for the first and second weeks postoperatively. At 10–14 days active motion may begin; however, flexion should be limited 70° for the first 6–8 weeks. Early range of motion will help prevent the formation of adhesions. Return to competitive sports depended on recovery of muscle function and strength, especially of the abductors. Typically, patients returned to competitive sports after 4–6 months [26, 48].

Summary

Surgical dislocation is a safe and powerful tool that allows open acetabular rim trimming, labral refixation, and femoral osteochondroplasty. One should not underestimate the technical demands of this surgical technique. Favorable good to excellent midterm clinical outcomes of 70–80% have been reported in the literature.

References

1. Ganz R, Parvizi J, Leunig M, Siebenrock KA. Femoroacetabular Impingement: a cause for osteoarthritis of the hip. Clin Orthop. 2003;417(417):112–20.
2. Mei-Dan O, McConkey MO, Brick M. Catastrophic failure of hip arthroscopy due to iatrogenic instability: can partial division of ligamentum teres and iliofemoral ligament cause subluxation? Arthrosc. 2012;28(3): 440–5.
3. Ranawat AS, McClincy M, Sekiya JK. Anterior dislocation of the hip after arthroscopy in a patient with capsular laxity of the hip. A case report. J Bone Joint Surg. 2009;91(1):192–7.
4. Ayeni OR, Naudie D, Crouch S et al. Surgical indications for treatment for femoroacetabular impingement with surgical hip dislocation. Knee Surg Sports Traumatol Arthrosc. 2013;21(7):1676–83.
5. Leunig M, Beck M, Woo A, et al. Acetabular rim degeneration. A constant finding in the aged hip. Clin Orthop. 2003;413:201–7.

6. Matsuda DK, Carlisle JC, Arthurs SC, et al. Comparative systematic review of the open dislocation, mini-open, and arthroscopic surgeries for femoroacetabular impingement. Arthroscopy. 2011;27(2):252–69.
7. Wenger DE, Kendall KR, Miner M, Trousdale RT. Acetabular labral tears rarely occur in the absence of bony abnormalities. Clin Orthop. 2004;426(426):145–50.
8. Beck M, Kalhor M, Leunig M, Ganz R. Hip morphology influences the pattern of damage to the acetabular cartilage: femoroacetabular impingement as a cause of early osteoarthritis of the hip. J Bone Joint Surg Br. 2005;87(7):1012–8.
9. Hack K, Diprimio G, Rakhra K, Beaule PE. Prevalence of CAM type femoroacetabular impingement in asymptomatic volunteers. J Bone Joint Surg. 2010; 92A(14):2436–44.
10. Khannas V, Caragianis A, DiPrimio G et al. Incidence of hip pain in a prospective cohort of asymptomatic volunteers: is the cam deformity a risk factor for hip pain? Am J Sports Med. 2014;42(4):793–7, http://www.ncbi.nlm.nih.gov/pubmed/24481825
11. Kapron AL, Anderson AE, Peters CL, et al. Hip internal rotation is correlated to radiographic findings of cam femoroacetabular impingement in collegiate football players. Arthroscopy. 2012;28(11):1661–70.
12. Beaule PE, Hynes K, Parker G, Kemp KA. Can the alpha angle assessment of cam impingement predict acetabular cartilage delamination? Clin Orthop Relat Res. 2012;470(12):3361–7.
13. Nepple JJ, Carlisle JC, Nunley RM, Clohisy JC. Clinical and radiographic predictors of intra-articular hip disease in arthroscopy. Am J Sports Med. 2011;39(2):296–303.
14. Johnston TL, Schenker ML, Briggs KK, Philippon MJ. Relationship between offset angle alpha and hip chondral injury in femoroacetabular impingement. Arthroscopy. 2008;24(6):669–75.
15. Sutter R, Dietrich TJ, Zingg PO, Pfirrmann CW. How useful is the alpha angle for discriminating between symptomatic patients with cam-type femoroacetabular impingement and asymptomatic volunteers? Radiology. 2012;264(2):514–21.
16. Allen DJ, Beaule PE, Ramadan O, Doucette S. Prevalence of associated deformities and hip pain in patients with cam type femoroacetabular impingement. J Bone Joint Surg. 2009;91B(5):589–94.
17. Sansone M, Ahlden M, Jonasson P, Sward L, Eriksson T, Karlsson J. Total dislocation of the hip joint after arthroscopy and ileopsoas tenotomy. Knee Surg Sports Traumatol Arthrosc. 2013;21(2):420–3.
18. Ilizaliturri Jr VM. Complications of arthroscopic femoroacetabular impingement treatment: a review. Clin Orthop Relat Res. 2009;467(3):760–8.
19. Philippon MJ, Schenker ML, Briggs KK, et al. Revision hip arthroscopy. Am J Sports Med. 2007;35(11): 1918–21.
20. Heyworth BE, Shindle MK, Voos JE, Rudzki JR, Kelly BT. Radiologic and intraoperative findings in revision hip arthroscopy. Arthroscopy. 2007;23(12):1295–302.
21. Bedi A, Zaltz I, De La Torre K, Kelly BT. Radiographic comparison of surgical hip dislocation and hip arthroscopy for treatment of cam deformity in femoroacetabular impingement. Am J Sports Med. 2011;39(Suppl):20S–8.
22. Beaule PE, LeDuff MJ, Zaragoza EJ. Quality of life outcome of femoral head/neck offset correction for femoroacetabular impingement. J Bone Joint Surg. 2007;89A(4):773–9.
23. Beck M, Leunig M, Parvizi J, et al. Anterior femoroacetabular impingement: part II. Midterm results of surgical treatment. Clin Orthop Relat Res. 2004;418:67–73.
24. Espinosa N, Beck M, Rothenfluh DA, et al. Treatment of femoro-acetabular impingement: preliminary results of labral refixation. Surgical technique. J Bone Joint Surg Am. 2007;89(Suppl 2 Pt 1):36–53.
25. Murphy SB, Tannast M, Kim Y-J, et al. Debridement of the adult hip for femoroacetabular impingement. Indications and preliminary clinical results. Clin Orthop. 2004;429(429):178–81.
26. Naal FD, Miozzari HH, Wyss TF, Notzli HP. Surgical hip dislocation for the treatment of femoroacetabular impingement in high-level athletes. Am J Sports Med. 2011;39(3):544–50.
27. Naal FD, Miozzari HH, Wyss TF, Notzli HP. Midterm results of surgical hip dislocation for the treatment of femoroacetabular impingement. Am J Sports Med. 2012;40(7):1501–10.
28. Sink EL, Fabricant PD, Pan Z, Daytonm MR, Novais EN. Results of treatment of femoroacetabular impingement in adolescents with a surgical hip dislocation approach. Clin Orthop Relat Res. 2013;471(8): 2563–9. Ref Type: Generic.
29. Tanzer M, Noiseux N. Osseous abnormalities and early osteoarthritis: the role of hip impingement. Clin Orthop. 2004;429(429):170–7.
30. Wilson AS, Cui Q. Current concepts in management of femoroacetabular impingement. World J Orthop. 2012;3(12):204–11.
31. Ganz R, Gill TJ, Gautier E, et al. Surgical dislocation of the adult hip. A new technique with full access to the femoral head and acetabulum without the risk of avascular necrosis. J Bone Joint Surg Br. 2001;83(8):1119–24.
32. Gibson A. Posterior exposure of the hip joint. J Bone Joint Surg Am. 1950;32:36–53.
33. Nork SE, Schar M, Pfander G, et al. Anatomic considerations for the choice of surgical approach for hip resurfacing arthroplasty. Orthop Clin North Am. 2005;36(2):163–70.
34. Gautier E, Ganz K, Krugel N, et al. Anatomy of the medial circumflex artery and its surgical implications. J Bone Joint Surg. 2000;82B(5):679–83.
35. Lavigne M, Parvizi J, Beck M, et al. Anterior femoroacetabular impingement. Part I: Techniques of joint preserving surgery. Clin Orthop. 2004;418(418):61–6.
36. Bastian JD, Wolf AT, Wyss TF, Notzli H. Stepped osteotomy of the trochanter for stable, anatomic refixation. Clin Orthop Relat Res. 2009;467:732–8.

37. Burnett SJ, Della Rocca GJ, Prather H, et al. Clinical presentation of patients with tears of the acetabular labrum. J Bone Joint Surg. 2006;88(7):327–33.
38. Ito K, Leunig M, Ganz R. Histopathologic features of the acetabular labrum in femoroacetabular impingement. Clin Orthop Relat Res. 2004;429 (429):262–71.
39. Geyer MR, Philippon MJ, Fagrelins TS, Briggs KK. Acetabular labral reconstruction with an iliotibial band autograft: outcome and survivorship analysis at minimum 3-year follow-up. Am J Sports Med. 2013;41(8):1750–6.
40. Philippon MJ, Briggs KK, Hay CJ, et al. Arthroscopic labral reconstruction in the hip using iliotibial band autograft: technique and early outcomes. Arthroscopy. 2010;26(6):750–6.
41. Sierra RJ, Trousdale RT. Labral reconstruction using the ligamentum teres capitis: report of a new technique. Clin Orthop Relat Res. 2009;467(3):753–9.
42. Mardones RM, Gonzalez C, Chen Q, et al. Surgical treatment of femoroacetabular impingement: evaluation of the effect of the size of the resection. J Bone Joint Surg. 2005;87A(2):273–9.
43. Notzli HP, Siebenrock KA, Hempfing A, et al. Perfusion of the femoral head during surgical dislocation of the hip. Monitoring by laser Doppler flowmetry. J Bone Joint Surg Br. 2002;84(2):300–4.
44. Lavigne M, Boddu Siva Rama KR, Doyon J, Vendittoli PA. Bone-wax granuloma after femoral neck osteoplasty. Can J Surg. 2008;51(3):58–60.
45. Lamontagne M, Brisson N, Kennedy MJ, Beaulé PE. Preoperative and postoperative lower-extremity joint and pelvic kinematics during maximal squatting of patients with cam femoro-acetabular impingement. J Bone Joint Surg. 2011;93A Suppl 2:40–5.
46. Beck M. Groin pain after open FAI surgery: the role of intraarticular adhesions. Clin Orthop Relat Res. 2009;467(3):769–74.
47. Hellman MD, Riff AJ, Haughom BD, Patel R, Stover MD, Nho SJ. Operative treatment of FAI: open hip preservation surgery. Curr Rev Musculoskelet Med. 2013;6(3):258–63.
48. Bizzini M, Notzli HP, Maffiuletti NA. Femoroacetabular impingement in professional ice hockey players: a case series of 5 athletes after open surgical decompression of the hip. Am J Sports Med. 2007;35(11):1955–9.

Surgical Technique: Mini-open Acetabular Rim Trimming, Labral Refixation, Femoral Osteochondroplasty

52

Patrick O'Toole, Antonia F. Chen, Joshua Minori, and Javad Parvizi

Contents

Abstract

Femoroacetabular impingement is a condition that results from a mismatch of congruity between the femoral head and the acetabulum. This incongruency leads to repetitive trauma to the intervening soft tissues leading inevitably to labral and chondral injuries that can subsequently result in degenerative joint disease of the hip. Early appropriate treatment of the lesion may preserve the longevity of the patient's native joint. The hip joint is exposed and the labral pathology is assessed. A femoral neck osteoplasty is then performed followed by labral reattachment. Postoperatively, the patient can be weight bearing as tolerated using crutches or another assistive walking device for 6 weeks to protect the labral repair.

Introduction

Femoroacetabular impingement (FAI) is a condition that results from a mismatch of congruity between the femoral head and the acetabulum. This incongruency leads to repetitive trauma to the intervening soft tissues leading inevitably to labral and chondral injuries that can subsequently result in degenerative joint disease of the hip. This condition was first described by Ganz [1] and is a cause of hip pain in younger patients. As FAI can lead to premature degenerative joint disease, early appropriate treatment of the lesion may preserve the longevity of the patient's native joint. While different

P. O'Toole • A.F. Chen • J. Minori • J. Parvizi (✉)
The Rothman Institute, Thomas Jefferson University, Philadelphia, PA, USA
e-mail: pojotoole@yahoo.com; antoniachen1@gmail.com; Joshua.Minori@rothmaninstitute.com; parvj@aol.com

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_55

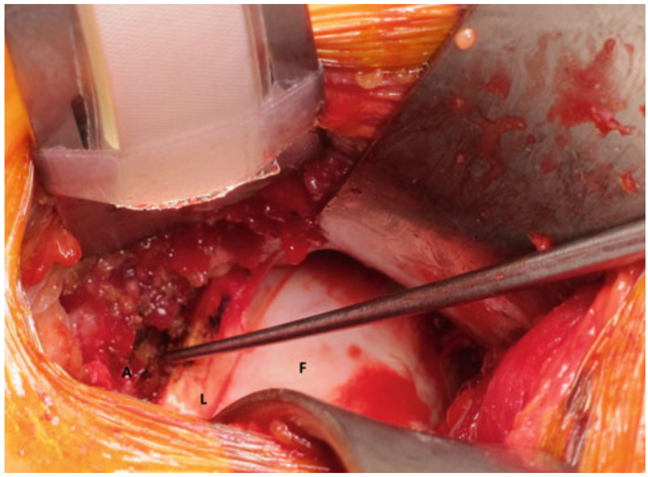


Fig. 1 Exposure of the femoral head (*F*), labrum (*L*), and acetabular rim (*A*), with probing of the labrum using a nerve hook to determine labral pathology

approaches and techniques exist for the treatment of FAI, at our institution a technique that utilizes a mini-Hueter anterior approach is performed.

Preoperative Planning

A prerequisite of any successful surgical technique includes appropriate patient selection, which is directed by specific findings on history, physical examination, and special investigations including plain radiography and magnetic resonance imaging. Adequate exposure and visualization is essential so that the offending labral and chondral lesions are easily accessible for treatment. Although Malik et al. [2] showed that the accessible acetabular rim surface with the mini-open technique is only 1.9 cm with a range of 1.1–2.4 cm, which is a smaller area when compared to that of surgical dislocations and hip arthroscopy, Beck et al. [3], however, revealed that the majority of labral and chondral lesions were located within the accessible area offered by this approach. Although the mini-Hueter approach is not ideal for more extensive lesions, it is adequate for cam-type lesions and cases where acetabular over-coverage due to retroversion needs to be addressed.

Surgical Technique

Once the surgical approach is complete, adequate visualization of the acetabular margin, labrum, anterior femoral head cartilage, and femoral neck should be achieved (Fig. 1). Flexion of the hip now allows direct visualization of the offending lesion causing labral damage secondary to femoral neck abutment on the acetabular rim. Next, the labrum is carefully inspected for evidence of a tear with or without degeneration. This can be done using the nerve hook in a probing fashion to allow inspection of the affected labrum (Fig. 1). The acetabular cartilage now needs to be inspected; however in order to do this, the labrum must be detached from the rim; this can be performed de novo or by extending a preexisting tear. Careful excision of any calcified lesions within the labrum can be performed at this stage (Fig. 2). The labrum can now be retracted sufficiently to expose the acetabular margin. At this point the assistant can gently exert longitudinal traction on the distal limb to allow the surgeon to insert a blunt spacer, usually a blunt Cobb elevator, into the joint space between the femoral head and the acetabulum (Fig. 3). Extreme care should be taken with this step so as not to further damage the articular cartilage.

Acetabular Rim Debridement

Inspection and assessment of the articular cartilage of the femoral head and acetabulum is now performed using a nerve hook to gently probe the surfaces for chondral flaps (Fig. 3). Occasionally, in the presence of a significant full-thickness flap, micro-fracturing is performed. The acetabular rim can now be addressed. If over-coverage of the

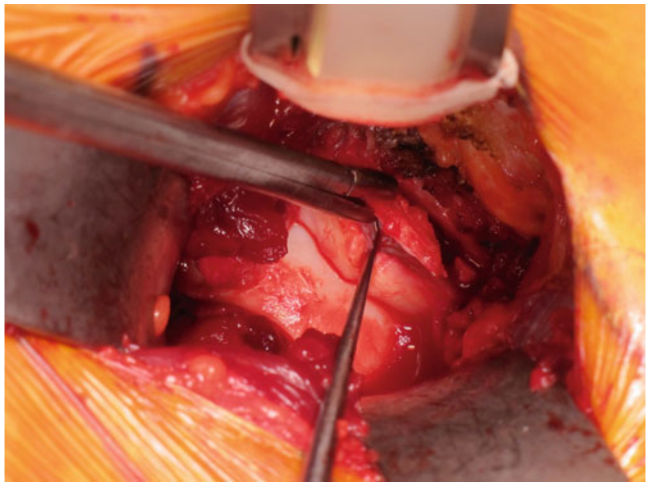


Fig. 2 Calcified labral lesions are carefully excised after the labrum is exposed

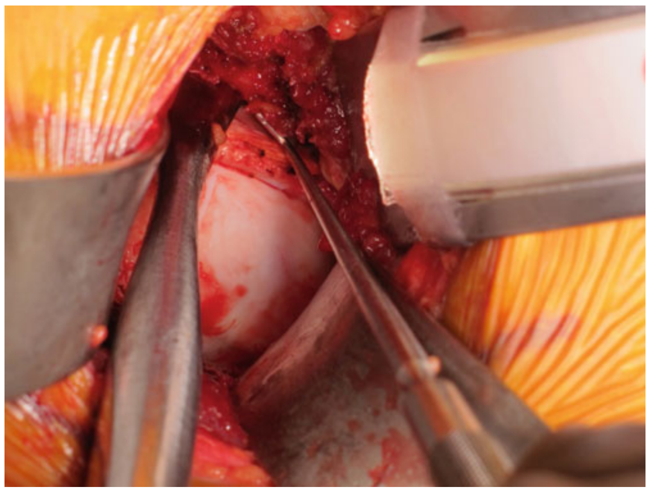


Fig. 3 Longitudinal traction on the limb allows a blunt Cobb elevator to be inserted into the joint. This aids exposure of the cartilaginous surfaces allowing a nerve hook to be used to gently probe any chondral flaps

femoral head by the acetabular rim is present, as a result of acetabular retroversion, this redundant or excess rim can be excised. A 10 mm curved osteotome in combination with a 5 mm pneumatic burr is used to achieve adequate debridement. The amount of debridement varies depending on the extent of the chondral lesion, but care must be taken not to remove more than 1 cm from the rim as this may lead to joint instability postoperatively.

Femoral Neck Osteoplasty

Next the femoral head-neck junction is examined. When a cam-type lesion is present, it is usually located on the anterosuperior region of the neck and often exhibits a characteristic reddish appearance. The assistant should now place the hip in internal rotation, which simulates the impingement test and allows the surgeon to clearly define the extent of the cam lesion (Fig. 4). It is now imperative to define the head-neck junction and also to denude the cam-type lesion of its cartilage covering. Now the neck osteoplasty can be safely performed, again using the 10 mm osteotome and the 5 mm burr. The osteoplasty is only complete when once again the smooth contour of the femoral head and neck is recreated. The preoperative decision regarding the extent of the resection can be difficult as FAI is a dynamic process. The resection should always be less than 30 % of the neck area [4]. A reference guide to the desired

Fig. 4 To assess the adequacy of the neck osteoplasty, internal rotation of the limb is performed to simulate the impingement test, with the absence of impingement signifying sufficient resection

Fig. 5 Pre-op anteroposterior (**a**) and lateral (**b**) radiographs. The location and extent of the osteoplasty resection on the post-op anteroposterior radiograph (**c**) is shown

resection based on the anteroposterior radiograph of the pelvis starts the bony correction 15 mm away from the acetabular rim, with extension of it 10 mm anterior to the 12 o'clock position and 10 mm to the lateral border (Fig. 5). On the Lowenstein lateral view, a resection depth of 5 mm is acceptable to restore the femoral head-neck offset, with extension distally in the axial plane of up to 20 mm. The adequacy of the neck osteoplasty is assessed by once again performing internal rotation of the limb to simulate the impingement test, with absence of impingement signifying sufficient resection (Fig. 4). The surgeon should aim to get hip flexion of 100° and internal rotation of 15° without impingement occurring. Three-dimensional assessment of the neck osteoplasty can be performed at the time of surgery. Computer-assisted osteoplasty has been described to aid with the desired level of resection [5–7]; however this should not be solely relied upon.

Labral Reattachment

The next portion of the surgical technique is related to the reattachment of the labrum to the acetabular rim. The cancellous acetabular rim can be burred, under direct vision, to bleeding bone. The surgeon should try to use at least three suture anchors for labral reattachment as this allows for a more robust repair (Fig. 6) (Lupine™ loop anchor system, DePuy Mitek, Raynham, MA, USA). The suture anchors are predrilled using a stop drill with the anchors themselves inserted freehand. An inside-out technique should be used when suturing the labrum in place with these anchors to allow the knots to rest in

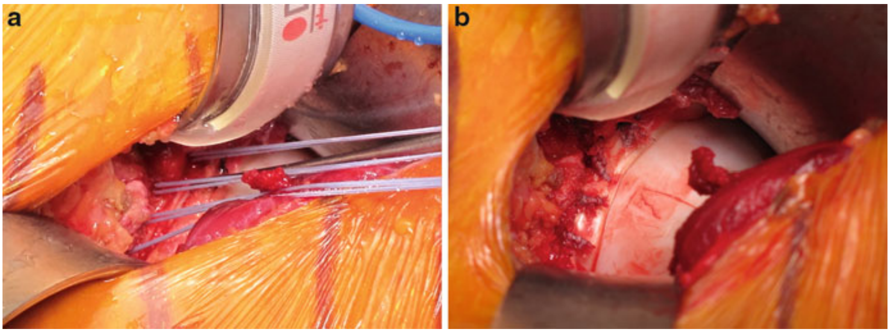


Fig. 6 (**a**) Multiple suture anchors used for labral reattachment and (**b**) after fixation is complete

an extra-articular location. The repair is tested using the nerve hook, and all exposed cancellous bone is sealed using bone wax in order to prevent excess bleeding. Thorough irrigation of the hip joint is now performed. It should be noted that the involved labrum is not always amenable to reattachment and may need to be debrided to stable tissue.

Finally, the range of motion is once again assessed, and closure then proceeds in a predictable fashion with the capsule closed with interrupted sutures and the remainder of the wound closed in layers.

Postoperative Management

Analgesia is based on a multimodal regime, while aspirin and mechanical pneumatic devices are used to prevent thromboembolic events. Heterotopic ossification prophylaxis is not routinely used, as the incidence of this is quite low. Patient's weight bearing is weight bearing as tolerated with crutches or other assistive walking devices for up to 6 weeks postoperatively. This protects the labral repair and helps protect against femoral neck fractures. The majority of patients are discharged home on the day of surgery and are followed up at 6 weeks, 3 months, and 1 year post procedure with radiographs obtained at each visit.

Summary

Femoroacetabular impingement is a condition that results from a mismatch of congruity between the femoral head and the acetabulum. This incongruency may lead to degenerative joint disease of the hip. Early appropriate treatment of the lesion may preserve the longevity of the patient's native joint. A femoral neck osteoplasty is often required followed by a labral repair.

References

1. Ganz R, Parvizi J, Beck M, Leunig M, Notzli H, Siebenrock KA. Femoroacetabular impingement: a cause for osteoarthritis of the hip. Clin Orthop Relat Res. 2003;417:112–20.
2. Malik AK, Chou DT, Witt JD. Anterior approaches to the hip for the treatment of femoro-acetabular impingement: a cadaveric study. Hip Int. 2010;20(4): 482–8.
3. Beck M, Kalhor M, Leunig M, Ganz R. Hip morphology influences the pattern of damage to the acetabular cartilage: femoroacetabular impingement as a cause of early osteoarthritis of the hip. J Bone Joint Surg Br. 2005;87 (7):1012–8.
4. Mardones RM, Gonzalez C, Chen Q, Zobitz M, Kaufman KR, Trousdale RT. Surgical treatment of femoroacetabular impingement: evaluation of the effect of the size of the resection. J Bone Joint Surg Am. 2005;87(2):273–9.
5. Bedi A, Dolan M, Magennis E, Lipman J, Buly R, Kelly BT. Computer-assisted modeling of osseous impingement and resection in femoroacetabular impingement. Arthroscopy. 2012;28(2):204–10.
6. Ecker TM, Puls M, Steppacher SD, Bastian JD, Keel MJ, Siebenrock KA, et al. Computer-assisted femoral head-neck osteochondroplasty using a surgical milling device an in vitro accuracy study. J Arthroplasty. 2012;27(2):310–6.
7. Mofidi A, Shields JS, Tan JS, Poehling GG, Stubbs AJ. Use of intraoperative computed tomography scanning in determining the magnitude of arthroscopic osteochondroplasty. Arthroscopy. 2011;27(7): 1005–13.

Surgical Technique: Arthroscopic Management of the Acetabulum

53

Michael J. Salata and Christopher M. Larson

Contents

Abstract

Femoroacetabular impingement (FAI) can occur due to acetabular or pelvic morphologies that predispose to a mechanical mismatch during functional hip range of motion. This can take the form of pincer-type FAI due to socket orientation or excessive depth or can arise from either bony or soft tissue impingement that may result from extra-articular structures such as anterior inferior iliac spine (AIIS) pathomorphology. Surgical management of these conditions can be successful if a thorough and careful preoperative evaluation is performed and the patient is properly indicated for these procedures. This chapter will discuss the surgical technique for addressing these various pathoanatomies.

Introduction

Traditionally, pincer-driven FAI has been described secondary to acetabular overcoverage. This overcoverage can be further subclassified as true overcoverage or relative overcoverage based on a retroverted socket orientation. Most commonly focal acetabular overcoverage involves the anterior aspect of the acetabulum [1–6]. True overcoverage can be global and depending on the depth of the socket is termed coxa profunda or protusio acetabula [4]. When anterior overcoverage is present, it results in abnormal contact between the rim of the acetabulum and the femoral

M.J. Salata (✉)
Department of Orthopaedic Surgery, Joint Preservation and Cartilage Restoration Center, University Hospitals Case Medical Center, Cleveland, OH, USA
e-mail: michael.salata@uhhospitals.org

C.M. Larson
Minnesota Orthopedic Sports Medicine Institute, Twin Cities Orthopedics, Edina, MN, USA
e-mail: chrislarson@tcomn.com

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_56

neck during functional hip motion. This has two major sequelae. The first is that the acetabular labrum becomes ensnared and crushed between the rim of the acetabulum and the femoral head-neck junction, which can lead to labral ecchymosis, degeneration, tearing, and in some cases ossification [1–3, 5]. A second source of damage that may lead to clinical symptoms of posterior discomfort is a phenomenon termed "contrecoup" damage to the posterior aspect of the joint which results from a levering of the femoral head out of the socket that can lead to both labral and chondral damage [1–3]. Extra-articular sources of pelvic impingement have been more recently been elucidated. A prominent AIIS that can result from a developmental anatomic variant, from previous apophyseal avulsions of the rectus femoris or in the setting of a previous periacetabular osteotomy, can lead to bony or soft tissue impingement in the anterior aspect of the hip joint [7, 8].

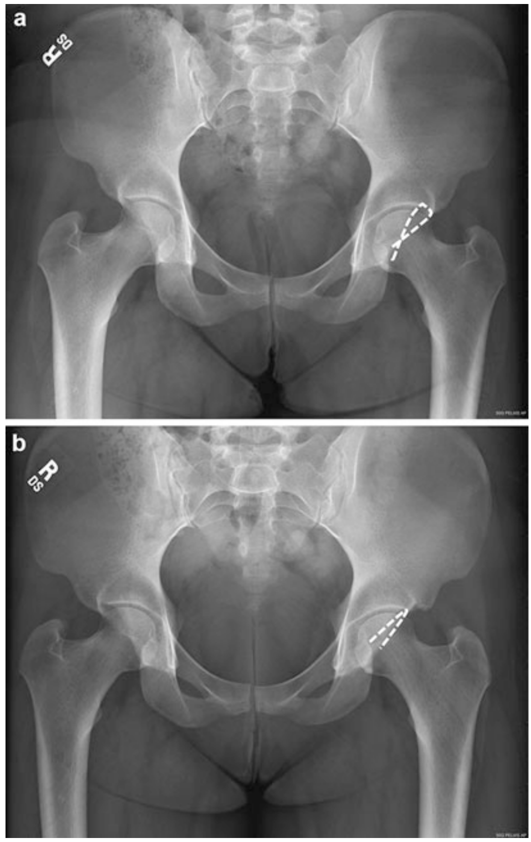


Fig. 1 (**a, b**) Anteroposterior radiograph of the left hip reveals evidence of a crossover sign (*dashed line*) and low AIIS in Fig. 1a indicating acetabular retroversion. Figure 1b after correction of the pincer-type FAI with elimination of the crossover sign and decompression of the AIIS

Patient Evaluation and Selection

Anterior hip pain is the most common symptom that patients with this condition will present with. The pain is often exacerbated with certain activities. Leg positions that combine flexion, adduction, and internal rotation (FADIR) are the most common aggravating motions for anteriorly based overcoverage. Abduction and extension/external rotation pain can be seen in the setting of lateral and posteriorly based acetabular overcoverage, respectively. While each surgeon will have their individual preferences for radiographic evaluation, a baseline series of radiographs including a well-aligned AP pelvis, a lateral view of the hip (i.e., cross table or 45° Dunn view), and a false profile view to evaluate anterior hip coverage should be considered. Further imaging including MRI and 3D CT scans can be helpful for confirming the diagnosis of labral and articular cartilage pathology and can be helpful to more accurately define acetabular anatomy and plan for surgical bony resections. While some controversy exists regarding definitive indications for arthroscopic rim resections, relative indications include focal anterior overcoverage (Fig. 1a, b), mild to moderate retroversion of the acetabulum, coxa profunda (Fig. 2a, b), and subspinous/AIIS impingement. Contraindications for rim recession include advanced arthrosis with higher Tonnis grades, acetabuli with excessively depth (protusio) combined with a large notch leading to deficient volume of articular cartilage, significant dysplasia/low volume acetabula, as well as severe acetabular retroversion.

Technique

Intraoperative Setup and Assessment

While both the supine and lateral position have been described, this chapter will discuss the supine position. Either a commercially available

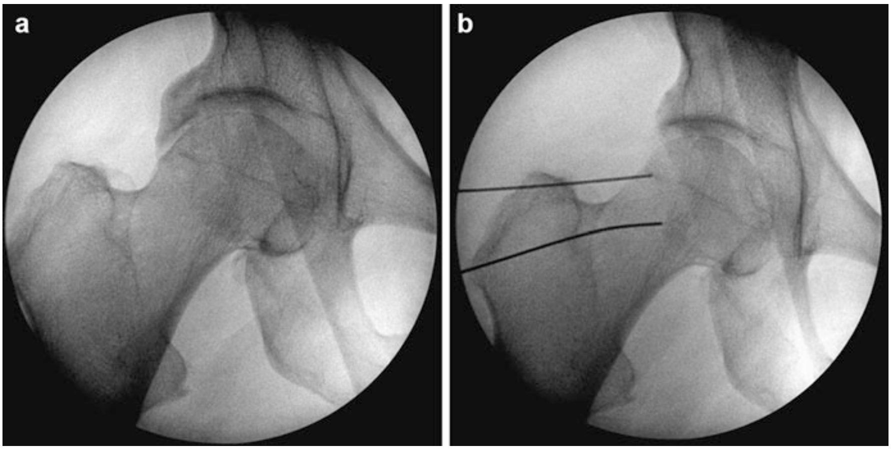


Fig. 2 (**a, b**) Intraoperative fluoroscopic image reveals coxa profunda with global overcoverage and labral/rim ossification in Fig. 2a. Correction of the overcoverage after anterior/lateral/posterior (global) rim resection arthroscopically in Fig. 2b (nitinol guidewires maintaining portal positions)

table attachments or a standard fracture table can be utilized to provide the distraction forces required to perform this procedure safely. Once anesthetized, an examination under anesthesia is performed to assess for hypermobility and to document preoperative range of motion with particular emphasis placed on the internal and external hip rotation at 90° and in extension. Well-padded boots and a well-padded offset perineal post are utilized to decrease the risk of neurologic injury [9]. The operative limb is placed into a position of slight hip flexion and roughly 15° of internal rotation to account for normal femoral anteversion and is placed in neutral abduction/adduction. Depending on the orientation of the pelvis after positioning, the operative bed may need to be tilted or "airplaned" with variable Trendelenburg/reverse Trendelenburg to achieve neutral pelvic tilt in order to create a fluoroscopic recreation of a well-centered preoperative AP pelvic radiograph. Once the patient is appropriately positioned and oriented, several fluoroscopic views of the hip are performed, a technique that has been described as an "around the world" assessment (Fig. 3). The hip is evaluated in neutral rotation and maximal internal and external rotation while both flexed and extended for a total of six views. This allows for an assessment of the medial and lateral femoral head-neck junction (hip extension) and anterior and posterior femoral head-neck junction (hip flexion).

Surgical Technique

The hip is gradually distracted under fluoroscopy with care to avoid over-distraction and a goal of 8–10 mm of joint space opening. With excessively deep sockets or in the presence of large anterior/lateral acetabular fragments/overcoverage, it may be necessary to consider beginning in the peripheral compartment or outside the capsule and entering from an "outside-in" approach after a rim recession is performed. More commonly, a standard anterolateral (AL) portal is created beginning with a 16 gauge spinal needle and introduction of a cannula over a nitinol guidewire. A modified mid-anterior portal (MMA) is then created under direct visualization [10, 11]. Rarely a posterolateral (PL) portal will need to be created if a significant posterior rim recession is planned. During the diagnostic portion of the procedure, the clinical diagnosis of a pincer-type FAI is confirmed by the presence of labral ecchymosis, degeneration, ossification, posterior acetabular wear consistent with a contrecoup lesion, and an acetabular rim that extends well beyond the chondrolabral junction.

Labral preservation is preferred if possible and has been shown to have superior results to labral resection [2, 10]. If possible, the chondrolabral junction can be left intact as it can help to stabilize the labrum in a more anatomic position and more predictably results in a repair with maintenance of the labral seal (Fig. 4). When this technique is utilized, the leading edge of the acetabular rim is resected behind the labrum to achieve the desired level of resection. This technique is quite effective for focal anterior-based overcoverage. Alternatively a formal labral takedown can be performed sharply and the rim recessed and is more often used in the setting of greater degrees of

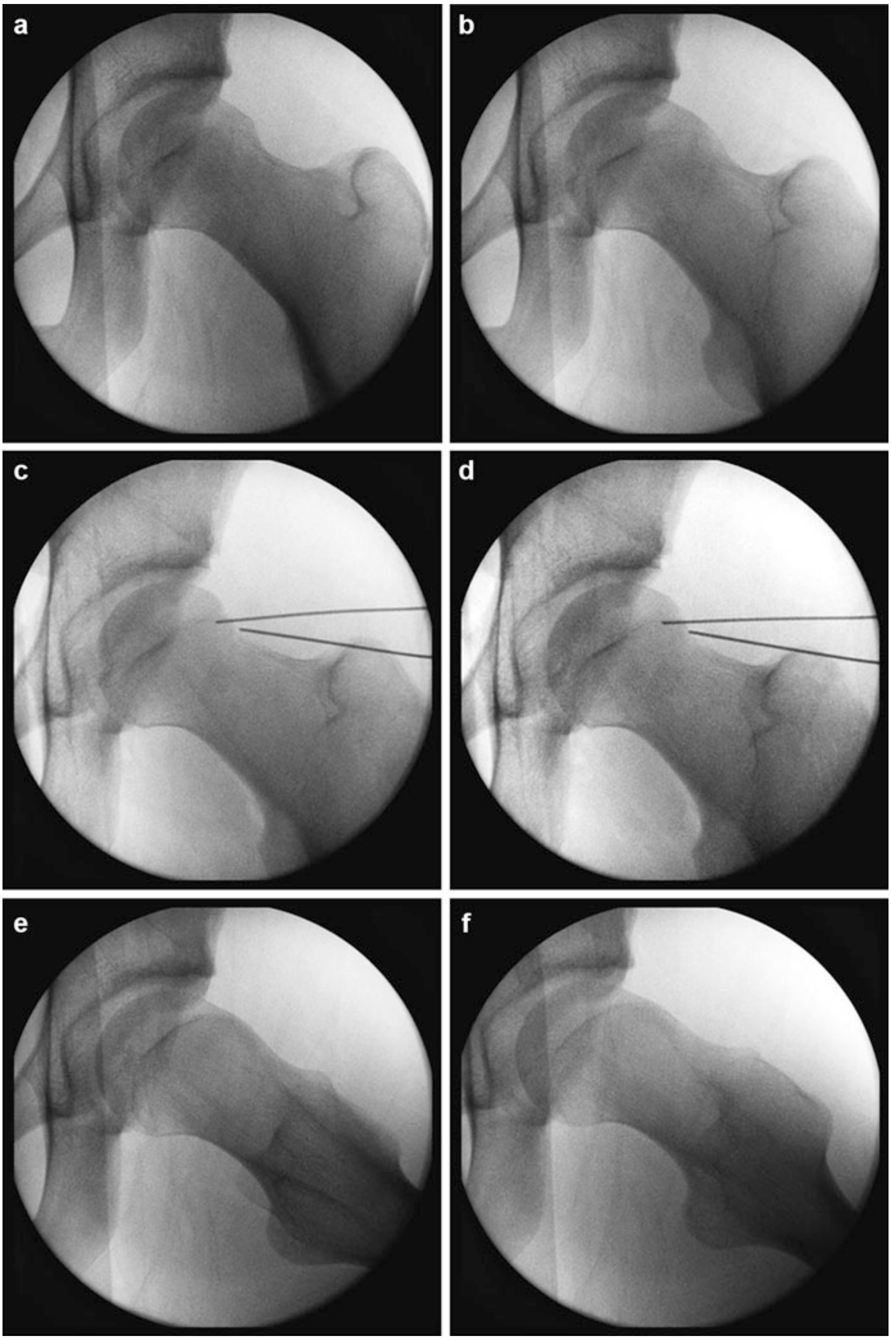


Fig. 3 (continued)

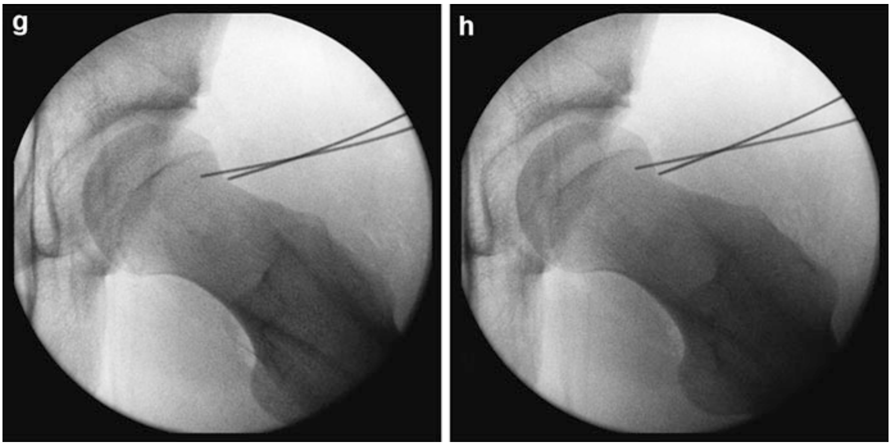


Fig. 3 Intraoperative pre-resection (**a, b, e, f**) and post-resection (**c, d, g, h**) AP and lateral fluoroscopic images demonstrate cam decompression

overcoverage in order to resect excess acetabular articular cartilage allowing for better advancement of the labrum to the acetabular rim (Fig. 5) [6]. Using preoperative information including a 3D CT scan and the information gathered at the time of the diagnostic arthroscopy, the length and depth of the rim resection can be determined and can vary significantly between patients. Typically the amount of resection for focal overcoverage is between 3 and 6 mm in depth. When a deeper resection is necessary, it is helpful to occasionally release traction to assess for proper acetabular coverage as over-resection can lead to edge loading of the acetabular articular surface and the potential for iatrogenic dysplasia/instability.

For both techniques, the rim recession is performed with a motorized high-speed burr and the starting point for the resection is confirmed with fluoroscopy. Work is initiated through the modified mid-anterior portal and viewing through the AL portal and begins with the recession just inferior to the area of acetabular overcoverage seen on the fluoroscopic image. For more posterior resections the burr can be introduced though the PL portal and AL portal while viewing from the MMA and anterolateral portal. As the recession continues it is continually assessed with fluoroscopy to confirm the regions of the acetabulum being resected. A combination of direct visualization and intraoperative fluoroscopy allows for a more accurate resection in the current authors experience.

If a labral takedown has been performed, or if the amount of rim resected has led to a destabilization of the labrum, labral refixation is then performed at this point of the procedure. The number of anchors utilized will depend on the length of rim resected and is determined by the anatomy of the pincer lesion. Placement of the first anchor is at the more superior aspect of the labral takedown when more lateral rim resection is performed. This can be accomplished by drilling through the AL portal or alternatively a distal anterolateral portal (DALA) can be created to improve the angle for drilling allowing the surgeon to place the anchor as close to the acetabular rim as possible while not penetrating the articular surface. The DALA portal is localized in line with the standard AL portal and is between 2 and 3 cm distal to it. Otherwise, the anchors are placed from anteromedial to superolateral with anchors placed through the MMA portal and arthroscopic visualization via the AL portal. It is critical to place the anchor on the bony rim 1–2 mm off the articular margin in order to avoid eversion of the labrum, which will enable the surgeon to more accurately recreate the suction seal effect of the labrum (Fig. 6). With the anchor placed and tested for pullout, a tissue-penetrating device is used to place one limb of

Fig. 4 Rim recession depicted with chondrolabral junction left intact

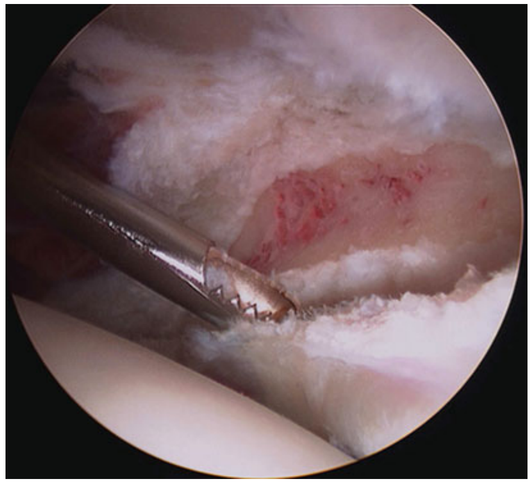


Fig. 5 Rim recession with a formal labral takedown

the suture between the acetabular rim and the labrum. When possible it is beneficial to then re-pierce the labrum and retrieve this suture creating a mattress-type or base refixation configuration for the repair. Some surgeons prefer to loop the suture around the labrum and currently there is no clear agreement about which technique is superior. Typically anchors are placed roughly 1 cm apart and will typically use 3 anchors for refixation, though for more extensive lesions between 5 and 8 anchors may be required. If there is associated AIIS/subspine impingement, the AIIS can be decompressed through the MMA portal and occasionally may be helpful to make an additional window through the direct head of the rectus femoral and capsular in order to preserve the capsular anteriorly (Fig. 7a, b). Rim fractures and/or os acetabula can typically be resected. In cases of fragments contributing to hip stability, however, arthroscopic-assisted cannulated screw fixation can be considered if the fragment is unstable (Fig. 8a, b).

Once the repair is completed and probed for stability, the traction is released and the construct is evaluated for maintenance of the suction seal. A thorough dynamic exam is then performed. Although surgeons can release the foot from the holder to perform the dynamic assessment, this can also be done in various positions of hip flexion, abduction, adduction, and internal/external rotation with the foot remaining in the boot. In addition to the traditional FADIR position which assesses anterior rim and anterior femoral head-neck junction impingement, we assess for impingement with the hip in greater degrees of extension with maximum internal and external rotation as well as abduction and adduction. Hip extension and abduction assesses for impingement between the lateral acetabular rim and lateral femoral head-neck junction. Hip abduction with the hip in 30–40° of flexion assesses for impingement between the lateral femoral head-neck junction and supero-posterior acetabulum.

At this time if there is noted to be any residual impingement coming from the femoral side, it is addressed with femoral osteoplasty (Fig. 9). Capsular closure is a debatable topic and there is no clear consensus with respect to this topic at this time. If a T-limb is utilized to aid in exposure, most surgeons would agree that at a minimum this should be repaired. Currently closure of the majority of the capsule in the majority of cases and in particular for females and cases with associated borderline dysplastic findings is preferred. Closure of the intra-portal capsulotomy can be

Fig. 6 The labral seal is maintained after release of traction and rim resection and labral repair with mattress-type suture technique

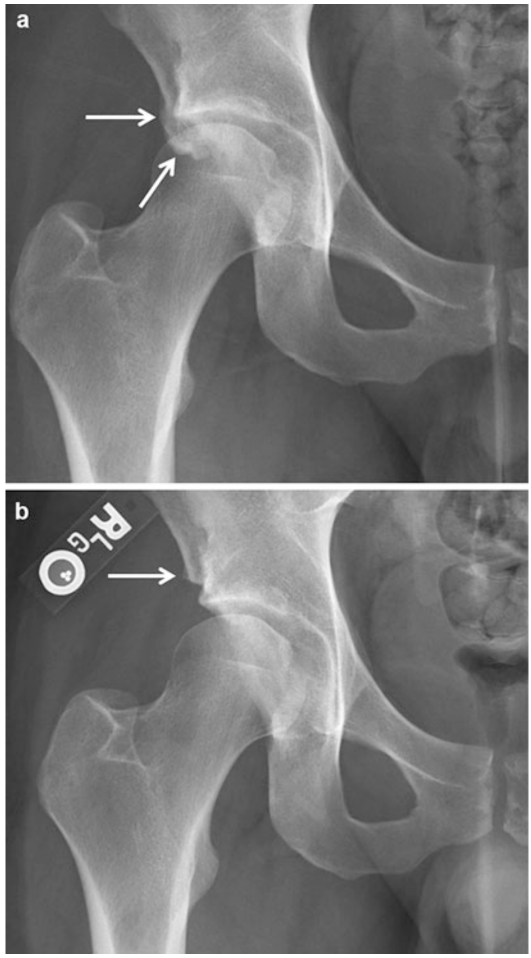


Fig. 7 (**a, b**) AIIS deformity is shown (*arrows*) in (**a**), and AIIS decompression and elimination of subspinous impingement noted in (**b**)

performed if desired with between 2 and 5 #2 sutures (Fig. 10). The portals are closed with nonabsorbable sutures and a soft dressing is applied.

Postoperative Management

Each surgeon may have slight variances in their postoperative rehabilitation protocols. Weight-bearing restrictions are based on the type of bony resection that is performed. Either heel touch or toe touch weight-bearing for 2–3 weeks can be utilized with the use of crutches until the patient walks without a limp to minimize overuse of the surrounding muscular envelope of the hip or increased stress to the femoral resection that might predispose to femoral neck stress reactions. In general extremes of motion especially the combination of passive hip hyperextension and the extremes of external rotation are avoided for 2–3 weeks in order to protect the capsular repair. Some surgeons choose to utilize a hip brace to help limit these activities for the first 2–3 weeks postoperatively, but the current authors reserve this for revision cases with capsular incompetence and patients with connective tissue disorders.

Some form of early passive ROM is critical in the immediate postoperative setting to minimize symptomatic intra-articular and capsulolabral adhesions. Some surgeons recommend the use of a continuous passive motion machine (CPM), while others favor 10–20 mins of cycling on a stationary bike 2x/day or circumduction ROM in lieu of the CPM. Initiation of physical therapy early can help to speed the recovery process. In general it is recommended that therapy begin on either the day of surgery or postoperative day 1. It is ideal if the patient can be treated by a therapist with experience in managing patients after hip arthroscopy, as this can be incredibly beneficial to help avoid pitfalls such as hip flexor tendonitis or motion limitations which can develop if activities are added too quickly or motion is initiated too late during this phase.

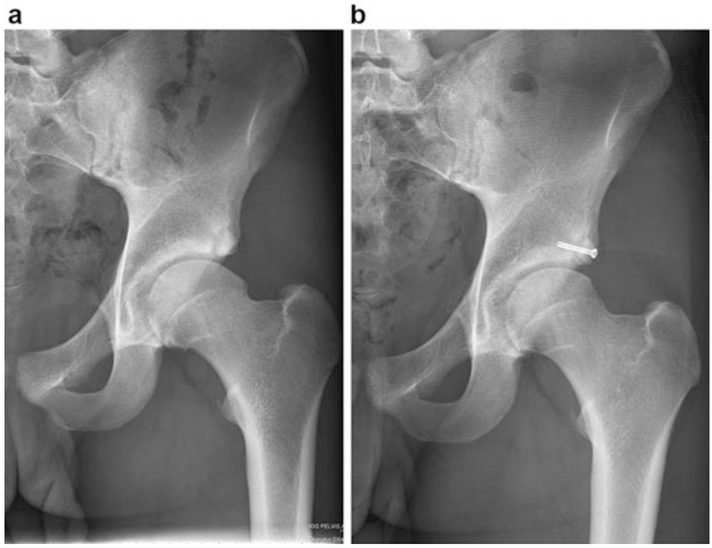


Fig. 8 (**a, b**) AP pelvis radiograph of the left hip reveals lateral cam-type morphology and a rim fracture that contributes to hip stability (**a**). Postoperative AP pelvis radiograph reveals femoral resection and cannulated screw fixation of the rim fracture (**b**)

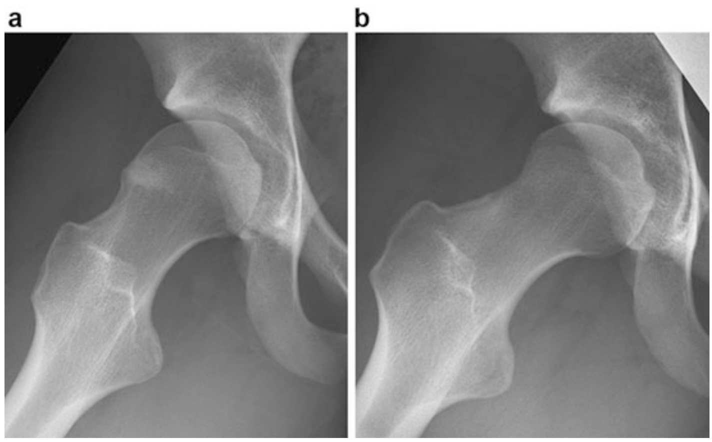


Fig. 9 (**a, b**) Preoperative (**a**) lateral radiograph and lateral radiograph after cam decompression (**b**) reveal restoration of femoral head-neck sphericity and offset

Outcomes

There are several studies in the literature that discuss the results of arthroscopic management of FAI and pincer impingement [2, 10–14]. Most studies show at least equivalent results of arthroscopic treatment of these conditions when compared to open surgical dislocation, with some studies showing arthroscopy might have some advantages regarding rehabilitation time or additional surgical procedures required which are primarily secondary to hardware removal or infrequent trochanteric nonunions. As previously discussed, several studies that compare labral refixation/repair to resection/debridement have shown that the refixation/repair results are superior to the resection/debridement in terms of outcomes studies and show less degenerative changes on follow-up X-rays [2, 11, 14].

Summary

Surgery to address the acetabular side of the joint can be performed successfully in the properly indicated patient. Care to technical detail and accurate diagnosis are key to a successful outcome in this patient population.

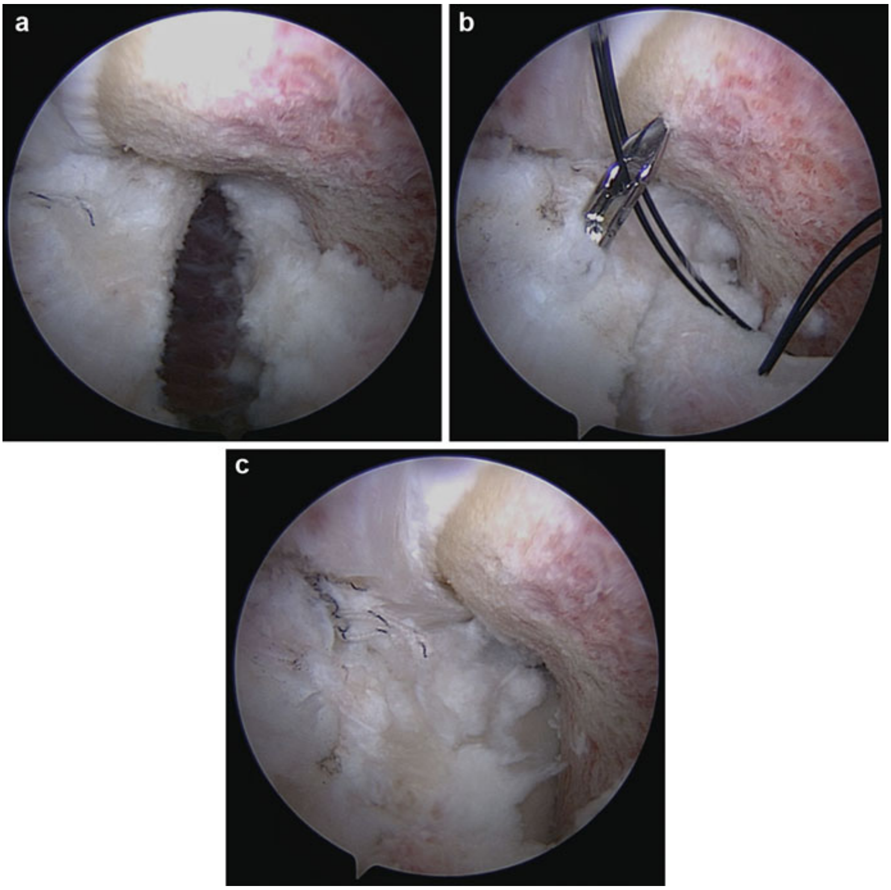


Fig. 10 Repair of the interportal capsulotomy is depicted (**a–c**)

References

1. Beck MLM, Parvizi J, et al. Anterior femoroacetabular impingement: Part II. Midterm results of surgical treatment. Clin Orthop Relat Res. 2004;418:67–73.
2. Espinosa N, Rothenfluh DA, Beck M, et al. Treatment of femoro-acetabular impingement: preliminary results of labral refixation. J Bone Joint Surg Am. 2006;88(5): 925–35.
3. Ganz RPJ, Beck M, et al. Femoroacetabular impingement: a cause for osteoarthritis of the hip. Clin Orthop Relat Res. 2003;417:112–20.
4. Larson CM. Arthroscopic management of pincer-type impingement. Sports Med Arthrosc. 2010;18(2):100–7.
5. Leunig MBM, Dora C, et al. Femoroacetabular impingement: etiology and surgical concept. Oper Tech Orthop. 2005;15:247–55.
6. Philippon MJ, Schenker ML. A new method for acetabular rim trimming and labral repair. Clin Sports Med. 2006;25(2):293–7.
7. Hetsroni I, Larson CM, Dela Torre K, et al. Anterior inferior iliac spine deformity as an extra-articular source for hip impingement: a series of 10 patients treated with arthroscopic decompression. Arthroscopy. 2012;28(11):1644–53.
8. Larson CM, Kelly BT, Stone RM. Making a case for anterior inferior iliac spine/subspine hip impingement: three representative case reports and proposed concept. Arthroscopy. 2011;27(12):1732–7.
9. Larson CM, Wulf CA. Intraoperative fluoroscopy for evaluation of bony resection during arthroscopic management of femoroacetabular impingement in the supine position. Arthroscopy. 2009;25(10):1183–92.
10. Larson CM, Giveans MR. Arthroscopic management of femoroacetabular impingement: early outcomes measures. Arthroscopy. 2008;24(5):540–6.
11. Larson CMGM. Arthroscopic debridement versus refixation of the acetabular labrum associated with femoroacetabular impingement. Arthroscopy. 2009;25: 369–76.
12. Bedi A, Chen N, Robertson W, et al. The management of labral tears and femoroacetabular impingement of

the hip in the young, active patient. Arthroscopy. 2008;24(10):1135–45.
13. Byrd JW, Jones KS. Arthroscopic femoroplasty in the management of cam-type femoroacetabular impingement. Clin Orthop Relat Res. 2009;467(3):739–46.
14. Philippon MJ, Briggs KK, Yen YM, et al. Outcomes following hip arthroscopy for femoroacetabular impingement with associated chondrolabral dysfunction: minimum two-year follow-up. J Bone Joint Surg Br. 2009;91(1):16–23.

Surgical Technique: Arthroscopic Labral Management

54

Frank McCormick, Andrew E. Federer, and Shane J. Nho

Contents

F. McCormick (✉)
Hip Preservation Center, Division of Sports Medicine, Department of Orthopaedic Surgery, Rush University Medical Center, Rush Medical College, Rush University, Chicago, IL, USA
e-mail: drfrankmccormick@yahoo.com

A.E. Federer
Midwest Orthopaedics at Rush, Hip Preservation Center, Rush University Medical Center, Chicago, IL, USA
e-mail: afederer34@gmail.com

S.J. Nho
Department of Orthopaedic Surgery, Rush University Medical Center, Chicago, IL, USA

Midwest Orthopaedics at Rush, Hip Preservation Center, Rush University Medical Center, Chicago, IL, USA
e-mail: shane.nho@rushortho.com

Abstract
The acetabular labrum is instrumental in maintaining the suction seal of the hip and thus allowing for cartilage load disbursement, synovial fluid distribution, and adds to joint stability. Acetabular labral tears are the most common indication for hip arthroscopy and are highly associated with femoral acetabular impingement (FAI). The intrinsic labral blood supply is poor, so acetabular bony decortication is used to access biologic healing factors, and the nervous pain generators are of highest density at the anterosuperior labral region. Recent studies demonstrate benefit to repairing labral defects over debridement. A reproducible, reliable, and successful surgical technique for arthroscopic labral repair and the intraoperative surgical decision-making is described.

Introduction

Acetabular labral tears are the most common indication for hip arthroscopy [1]. Most tears are caused by either trauma or an underlying hip pathomorphology such as dysplasia or femoral acetabular impingement (FAI) [2]. FAI is increasingly recognized and considered to be present in nearly of 90 % of patients undergoing hip arthroscopy for nontraumatic labral tears [2]. Following FAI, the second most common indication for patients undergoing hip arthroscopy is a history of trauma [3]. Additional and less common causes

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_57

of labral tears include capsular laxity, atraumatic microinstability, psoas impingement, and symptomatic internal snapping hip [1].

Labral tears of the hip are most commonly classified as either type 1 or type 2 based on anatomic and histological features determined by Seldes et al. [4]. Type 1 tears consist of detachment of the labrum at the articular cartilage surface, most often at the transition zone between the fibrocartilaginous labrum and the articular hyaline cartilage. These tears are perpendicular to the articular surface and can extend down to subchondral bone. Type 2 tears consist of one or more cleavage planes of variable depth within the substance of the labrum [4]. Labral tears can also be characterized using the more comprehensive Lage classification: radial flap, radial fibrillated, abnormally mobile, and longitudinally peripheral [5, 6]. Radial flap is the most common type and is considered a disruption in the free edge of the labrum. Radial fibrillated is associated with chondromalacia and consists of fraying of the labral free edge. Abnormally, mobile tears are often seen in labral detachment, similar to the Bankart lesion of the shoulder [6]. Longitudinally, peripheral tears occur along the labral edge and are the least common type of lesion.

Anatomy

The labrum is a horseshoe-shaped structure, blending into the transverse ligament inferiorly. It comprises two parts: the articular (fibrocartilage) and capsular (dense connective tissue) components [7]. The labrum is widest anteriorly but thickest superiorly and laterally, where maximum weight bearing occurs [6]. Anteriorly the labral-chondral transition zone is sharp, whereas it is gradual posteriorly [4].

The acetabular labrum has no intrinsic vasculature and derives most of its blood supply from the capsule and synovium [4, 7–10]. The resulting blood supply to acetabular labrum comes from a vascular anastomotic ring that surrounds the site of capsular attachment to the labrum. Thus, there is improved grade mean vascular supply to the capsular portion rather than the articular side of the labrum [9]. This ring is composed of the superior gluteal vessels, obturator artery, and one ascending branch of the medial femoral circumflex artery [10].

The correlation between labral integrity and hip pain can be understood by appreciating the nervous supply to the labrum. Its innervation by a diverse range of nerve types allow for detection of pressure, deep sensation, temperature, proprioception, and pain. The vast majority (86 %) of the nerve endings are located near to the articular surface, in contrast to any blood supply [9, 11]. Importantly, the density of unmyelinated free nerve endings, which detect pain, is highest in the anterior and superior regions of the labrum. The amount of nervous supply to the labrum does not change with age [11].

Biomechanical Considerations

A better anatomic understanding coupled with a growing body of biomechanical and clinical research continues to support the many roles of the acetabular labrum. Anatomically, it increases the articular surface by 22 % and the volume of the acetabulum by 33 %, therefore, increasing joint contact surface area [4]. Seldes et al. [4] demonstrated that when the labrum is torn, there is an increased association with cartilage defects. Crawford et al. [12] demonstrated that femoral head micro-motion within the acetabulum increased with labral tears and, therefore, highlights the role of the labrum in hip joint stability. Structurally, it confers a seal to the central compartment, which is the intra-articular space confined by the labrum and acetabulum. This seal has several functions including creating a suction environment with a negative pressure that increases the difficulty of dislocating the hip. It also helps retain synovial fluid within the central compartment, which has many benefits in its own right. This retention of synovial fluid acts to efficiently provide nutrients to the articular cartilage of the hip while maintaining a smooth gliding environment [13–15]. For this reason, a labral tear increases the resistance to rotation within the hip joint [16]. In a cadaveric study following repair of

Table 1 Summary of studies evaluating outcomes for labral repair versus debridement

Study	No. of patients	Study design	Mean age	Mean follow-up	Scoring scales	Outcomes
Espinosa et al. [23]	60	Level III retrospective comparative	30 year	12 and 24 months	Merle d'Aubigné clinical score Tönnis arthrosis classification	Open repair had statistically significant better outcomes
Larson et al. [24]	94	Level III retrospective comparative	30 year	40 months	Harris Hip Score (HHS), short form 12 (SF-12), and a visual analog scale (VAS)	Repair had statistically significant better outcomes
Krych et al. [28]	36	Level I RCT	39 year	36 months	Hip Outcomes Score (HOS)	Repair had statistically significant better outcomes
Philippon et al. [29]	122	Level II prospective cohort	40.6 year	27 months	Modified Harris Hip Score (mHHS)	Repair had statistically significant better outcomes with FAI

3 cm labral-chondral separation defects, the mean and maximal chondral strains of repaired labrums were less than that of labral-chondral separated or resected specimens and not different from intact labrums [17]. A more equally distributed pressure throughout the femoroacetabular joint prevents early arthritic wear.

Surgical Considerations

Recent studies demonstrate the benefit to repairing labral defects (Table 1). Surgical treatment for patients with labral tears includes either partial labrectomy, labral repair, or labral reconstruction. When hip arthroscopy was introduced, primary partial labrectomy was initially described for the treatment of labral tears; however, a number of recent studies have demonstrated improved outcomes with labral repair compared to labral labrectomy. In patients who have undergone partial labrectomy, Shindle et al. [18] have shown an improvement in modified Harris hip scores 31–40 % and reduction of hip pain in 91 % at 2–3.5 years follow-up. Importantly, however, many of these earlier studies saw a dramatic decline in clinical outcome if there was a concomitant chondral defect with the labral tear or if untreated FAI was present following a partial labrectomy [19–22]. More recent research comparing labral refixation compared to labral resection and correction FAI morphology has reported improved clinical and radiological outcomes with labral refixation [23, 24]. Philippon et al. [25] have proposed an algorithm for the treatment of labral injury based on location, size, and tissue quality. Labral debridement is recommended when the labrum demonstrates fraying on the periphery of the labrum but retains enough structural support to maintain normal labral function. In cases of labral detachment or after acetabular rim trimming, the labrum is refixed whenever possible with either a mattress stitch or simple loop stitch according to tissue quality. In the setting of severely degenerative tissue quality or after a prior partial labrectomy, a labral reconstruction can be considered.

Labral Refixation Technique

Once adequate distraction has been obtained, the anterolateral portal is established approximately 1 centimeter (cm) anterior and proximal to the anterior aspect of the greater trochanter. A spinal needle is introduced into the hip under fluoroscopic guidance, and a guidewire is placed through the spinal needle. A 4.5 cannula passed over the guidewire into the hip joint between the labrum and the femoral head. The 70° arthroscope is inserted through the cannula, and the anterior hip triangle is visualized. An anterior portal is created about 1 cm inferior and 1 cm lateral from the intersection of the vertical line from the anterior superior iliac spine (ASIS) and the horizontal line from the tip of the greater trochanter under direct visualization. At this point, the anteromedial aspect of the labrum can be visualized, and a probe can be used to palpate potential areas of detachment as well as cartilage delamination. Switch the arthroscope into the anterior portal to visualize the anterolateral extent of the labrum. Using a long handle arthroscopic scalpel (Samurai, Pivot Medical Sunnyvale CA), incise the hip capsule 5–8 mm away from the labrum to connect the anterolateral and anterior portals. The arthroscope needs to be switched back to the anterolateral cannula to complete the interportal capsulotomy. The length of the capsulotomy may vary depending on the extent of pathology, but generally about 4 cm is required. Be sure to incise the entire width of the capsule to allow for instrument exchange and excursion.

The labrum is evaluated according to the Seldes criteria [4]. The size and location of the chondrolabral injury can be approximated with a probe. The tissue quality and the labral width are also considerations when deciding how to treat the labrum. Based on preoperative imaging as well as the labral evaluation, the area of pincer deformity that requires rim trimming can be determined (Fig. 1).

A small shaver is introduced through the anterior portal, and the capsular tissue adjacent to the labrum is gently debrided. The shaver is positioned next to the capsular tissue, and the suction will allow the capsular tissue to be selectively debrided avoiding iatrogenic labral damage. It is critical to avoid injury to the superficial layer of the labrum to maintain the integrity of the labrum. A radiofrequency device (HipVac, ArthroCare, Austin, TX) is then introduced into the joint to ablate the remaining capsular tissue from the acetabular rim and the meticulously peel back the labrum from the acetabulum. Through the anterior portal, a 5.5 mm arthroscopic spherical burr is used to resect the anteromedial aspect of the

Fig. 1 Acetabular labral tear. An arthroscopic view of a *right hip* with a 70° scope in the anterolateral portal demonstrates a labral tear within the anterosuperior acetabular region with a wave sign of delaminated cartilage

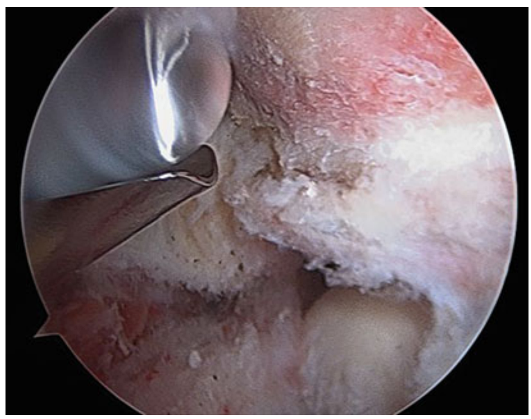


Fig. 2 Acetabular rim preparation. An arthroscopic view of a *right hip* with a 70° scope in the anterior portal with the arthroscopic burr through the anterolateral portal

pincer deformity (Fig. 2). Next, the arthroscopic burr is placed through the anterolateral portal to access the anterolateral aspect of the pincer deformity. If there are areas of anteroinferior iliac spine (AIIS) impingement, subspine decompression can be performed prior to labral refixation. The authors prefer to avoid labral takedown in most cases to preserve the chondrolabral transition zone in order to maintain the biologic interface and to facilitate more anatomic labral refixation and restoration of suction seal. In some cases, a formal labral takedown may be necessary in cases of global acetabular overcoverage. The fluoroscopic imaging can also be used to confirm the extent of bony rim resection.

Anchor placement can begin after the adequate osseous and soft tissue preparation. With the arthroscope in the anterior portal, 8 × 110 mm clear plastic cannula (Smith & Nephew, Andover, MA) is placed over a Wissinger rod in the anterolateral portal to refix the anterolateral labrum between 10 o'clock and 1 o'clock for a right hip. The drill guide is positioned through the cannula on the acetabulum as close to the articular cartilage surface as possible without penetration at the 11–12 o'clock position. The authors recommend using as small a suture anchor as possible to allow for accurate placement of anchors. While the assistant is drilling, the drill tip can be sounded to prevent violation of the subchondral plate. Meanwhile, the arthroscope is placed into the joint to view the cartilage surface to ensure that the drill is not between the subchondral bone and cartilage surface. Once the suture anchor (1.4 mm Nanotack, Pivot Medical, Sunnyvale, CA) is malleted into place, the sutures can be passed through the labrum. Anchor pull-out strength is always gently checked to confirm anchor seating. If the labrum size and tissue quality are normal, then mattress stitches are preferred with the first pass using a tissue-penetrating device (Nanopass, Pivot Medical, Sunnyvale, CA) at the chondrolabral junction, and the suture is retrieved through the widest aspect of the labrum. If the labrum is either hypoplastic or the tissue appears degenerative, then a simple loop is passed around the labrum. Next, the sutures are tied using reverse half hitches and alternating posts. Another anchor can be placed from this portal at the 12–1 o'clock location.

To access the anteromedial acetabulum, the arthroscope is placed in the anterolateral portal, and the clear plastic cannula is placed in the anterior portal. A percutaneous distal accessory anterolateral (DALA) portal is placed 4 cm distal and in line with the anterolateral portal. A drill guide is passed over a guidewire and placed on the acetabular rim (Fig. 3a). The drill is advanced using the same techniques previously described with a trajectory parallel to the floor. Once drilling begins, the cartilage within the joint is viewed ensuring there is no cartilage breaching with the drill. The anchor is inserted through the guide and gently malleted within the bone. Through the anterior cannula, the tissue penetrator is used to retrieve the suture closest to the labrum, and the labrum is pierced at the chondrolabral junction (Fig. 3b). The penetrator is passed into the joint and releases the suture. If performing a mattress configuration, the labrum is pierced in the widest aspect of the labrum with the penetrator (Fig. 3c). If performing a simple loop stitch, the penetrator is passed into the joint, and the sutures are then retrieved through the labrum and out of the cannula. Using the other suture as the post, a series of reverse half hitches and alternating posts are used to tie the knot (Fig. 3d–f). Labral refixation is completed when the labrum is firmly reattached to the acetabulum, and the suction seal of the joint is reestablished (Fig. 4). If a femoral osteoplasty is to be performed, the traction is taken off and the joint reduced. At the completion of the surgery, a dynamic assessment of the hip range of motion and the labral seal is confirmed.

Postoperative rehabilitation for labral tear repair is performed in a stepwise fashion. Immediately postoperation, patient are 20 lb foot flat weight bearing (FFWB) for 3–4 weeks with flexion limited to 90° and no hip extension or external rotation. Early passive range of motion with either continuous passive motion machine or stationary upright bicycle is recommended. In addition, passive circumduction of the operative extremity may also prevent capsulolabral adhesions. The authors prefer to use a hip orthosis and abduction pillow worn at night. A guided physical therapy program is strongly encouraged with stepwise milestones on restoring core and pelvic stability, passive range of motion, manual therapy, muscle activation, neuromuscular retraining, and strengthening. Running activities are allowed at 12 weeks if proper strength and motor control have been established and full return to sport at 6 months.

Summary

The scientific evidence supporting arthroscopic labral tear repair is growing [24, 26, 27]. Within increasing levels of evidence, labral repair

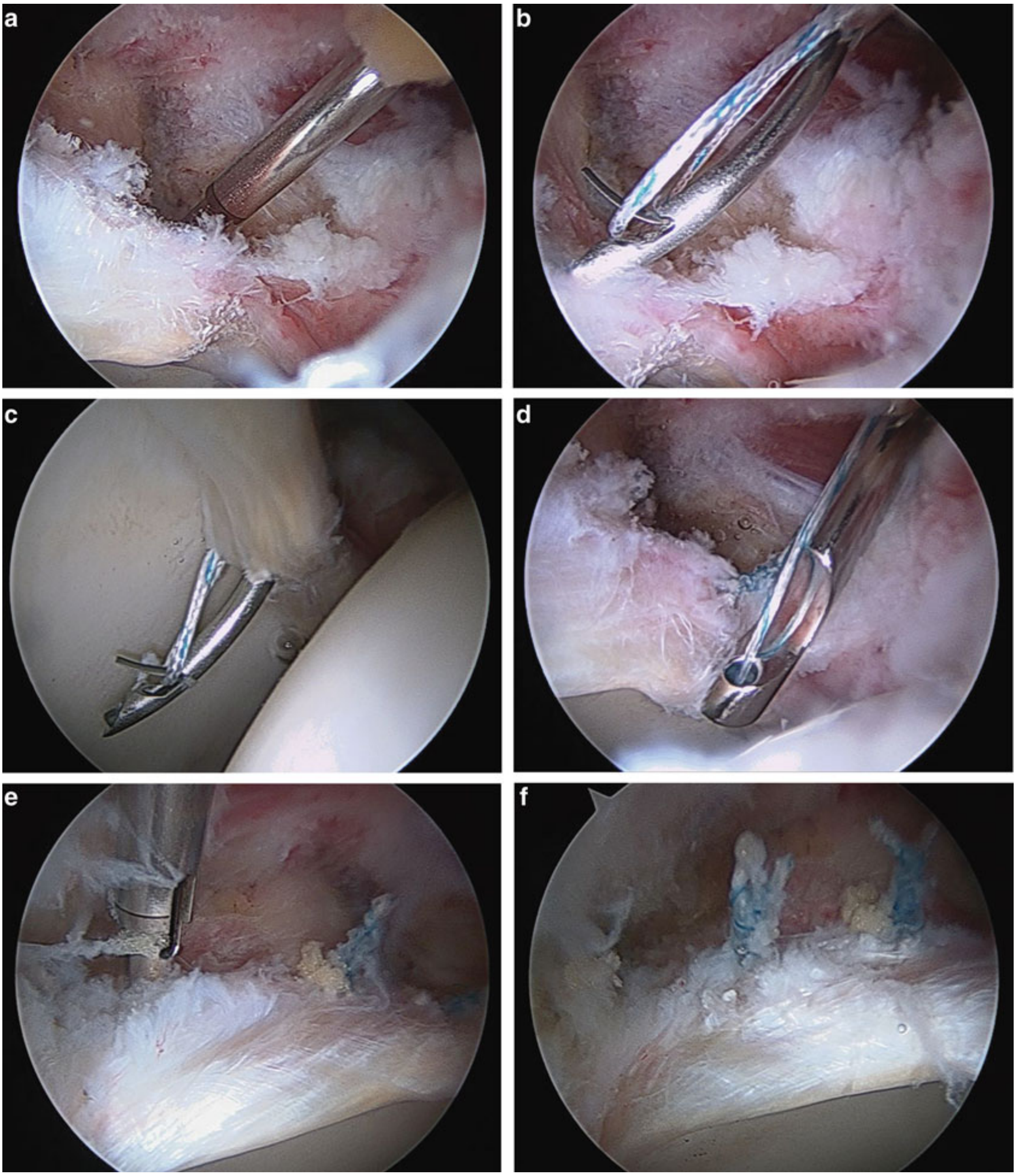


Fig. 3 Labral refixation. (**a**) Suture anchor insertion. The drill guide is placed through the DALA portal with the guide positioned 2–3 mm above the acetabular rim and parallel to the floor to avoid chondral penetration. (**b**) Tissue penetrator is introduced through the anterior portal to retrieve the suture and pass through the chondrolabral junction. (**c**) Tissue penetrator is used to make a second pass through the base of the labrum then used to retrieve the suture limb out of the cannula. (**d**) Sutures can be tied using arthroscopic knot tying techniques. (**e**) Tied anterior suture anchor. (**f**) Final appearance

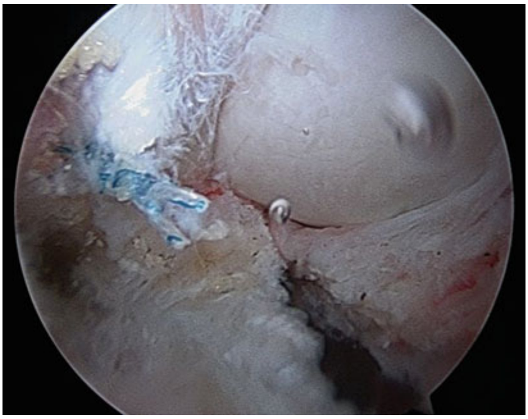


Fig. 4 Suction seal

consistently demonstrates superior outcomes compared to debridement [23, 24, 28, 29] (Table 1).

The surgical technique goals for labral repair are to restore the anatomy and, thus, biomechanical function. The authors' recommended surgical technique is as follows: (1) preserve labral tissue integrity; (2) assess labral tear size, location, and tissue quality (detachment, tear, degeneration, bruising, or diminutive/hypoplastic) to guide treatment; (3) preserve chondrolabral transition; (4) use smallest anchor as possible; (5) anchor placement as close to articular cartilage surface without penetration or delamination; and (6) mattress stitch when possible to avoid labral eversion.

The acetabular labrum is instrumental to maintain the suction seal of the hip joint, increased joint contact area, synovial fluid distribution, and joint stability. Acetabular labral tears are the most common indication for hip arthroscopy and are highly associated with femoral acetabular impingement (FAI) and other hip deformities. Recent clinical studies demonstrate improved functional outcome after labral refixation compared to labral debridement. Anatomic labral refixation is recommended whenever possible to preserve the structure and functional roles of the labrum.

References

1. Safran MR. The acetabular labrum: anatomic and functional characteristics and rationale for surgical intervention. J Am Acad Orthop Surg. 2010;18:338–45.
2. Wenger DE, Kendell KR, Miner MR, et al. Acetabular labral tears rarely occur in the absence of bony abnormalities. Clin Orthop Relat Res. 2004;426:145–50.
3. Burnett RS, Della Rocca GJ, Prather H, et al. Clinical presentation of patients with tears of the acetabular labrum. J Bone Joint Surg Am. 2006;88:1448–57.
4. Seldes RM, Tan V, Hunt J, et al. Anatomy, histologic features, and vascularity of the adult acetabular labrum. Clin Orthop Relat Res. 2001;382:232–40.
5. Lage LA, Patel JV, Villar RN. The acetabular labral tear: an arthroscopic classification. Arthroscopy. 1996;12:269–72.
6. Martin RL, Enseki KR, Draovitch P, et al. Acetabular labral tears of the hip: examination and diagnostic challenges. J Orthop Sports Phys Ther. 2006;36:503–15.
7. Petersen W, Petersen F, Tillmann B. Structure and vascularization of the acetabular labrum with regard to the pathogenesis and healing of labral lesions. Arch Orthop Trauma Surg. 2003;123:283–8.
8. McCarthy J, Noble P, Aluisio FV, et al. Anatomy, pathologic features, and treatment of acetabular labral tears. Clin Orthop Relat Res. 2003;406:38–47.
9. Kelly BT, Shapiro GS, Digiovanni CW, et al. Vascularity of the hip labrum: a cadaveric investigation. Arthroscopy. 2005;21:3–11.
10. Putz R, Schrank C. Anatomy of the labro-capsular complex. Orthopade. 1998;27:675–80.
11. Kim YT, Azuma H. The nerve endings of the acetabular labrum. Clin Orthop Relat Res. 1995;320:176–81.
12. Crawford MJ, Dy CJ, Alexander JW, et al. The 2007 Frank Stinchfield Award. The biomechanics of the hip labrum and the stability of the hip. Clin Orthop Relat Res. 2007;465:16–22.
13. Ferguson SJ, Bryant JT, Ganz R, et al. The influence of the acetabular labrum on hip joint cartilage consolidation: a poroelastic finite element model. J Biomech. 2000;33:953–60.
14. Ferguson SJ, Bryant JT, Ganz R, et al. The acetabular labrum seal: a poroelastic finite element model. Clin Biomech (Bristol, Avon). 2000;15:463–8.
15. Ferguson SJ, Bryant JT, Ganz R, et al. An in vitro investigation of the acetabular labral seal in hip joint mechanics. J Biomech. 2003;36:171–8.
16. Song Y, Ito H, Kourtis L, et al. Articular cartilage friction increases in hip joints after the removal of acetabular labrum. J Biomech. 2012;45:524–30.
17. Greaves LL, Gilbart MK, Yung AC, et al. Effect of acetabular labral tears, repair and resection on hip cartilage strain: a 7T MR study. J Biomech. 2010;43:858–63.
18. Shindle MK, Voos JE, Nho SJ, et al. Arthroscopic management of labral tears in the hip. J Bone Joint Surg Am. 2008;90 Suppl 4:2–19.
19. Farjo LA, Glick JM, Sampson TG. Hip arthroscopy for acetabular labral tears. Arthroscopy. 1999;15:132–7.
20. Santori N, Villar RN. Acetabular labral tears: result of arthroscopic partial limbectomy. Arthroscopy. 2000;16:11–5.

21. Kim KC, Hwang DS, Lee CH, et al. Influence of femoroacetabular impingement on results of hip arthroscopy in patients with early osteoarthritis. Clin Orthop Relat Res. 2007;456:128–32.
22. Tanzer M, Noiseux N. Osseous abnormalities and early osteoarthritis: the role of hip impingement. Clin Orthop Relat Res. 2004;429:170–7.
23. Espinosa N, Rothenfluh DA, Beck M, et al. Treatment of femoro-acetabular impingement: preliminary results of labral refixation. J Bone Joint Surg Am. 2006;88:925–35.
24. Larson CM, Giveans MR, Stone RM. Arthroscopic debridement versus refixation of the acetabular labrum associated with femoroacetabular impingement: mean 3.5-year follow-up. Am J Sports Med. 2012;40:1015–21.
25. Philippon MJ, Peixoto LP, Goljan P. Acetabular labral tears: debridement, repair, reconstruction. Op Tech Sports Med. 2012;20:281–6.
26. Byrd JW, Jones KS. Arthroscopic management of femoroacetabular impingement: minimum 2-year follow-up. Arthroscopy. 2011;27:1379–88.
27. McCormick F, Nwachukwu BU, Alpaugh K, et al. Predictors of hip arthroscopy outcomes for labral tears at minimum 2-year follow-up: the influence of age and arthritis. Arthroscopy. 2012;28:1359–64.
28. Krych AJ, Thompson M, Knutson Z, et al. Arthroscopic labral repair versus selective labral debridement in female patients with femoroacetabular impingement: a prospective randomized study. Arthroscopy. 2013;29:46–53.
29. Philippon MJ, Briggs KK, Yen YM, et al. Outcomes following hip arthroscopy for femoroacetabular impingement with associated chondrolabral dysfunction: minimum two-year follow-up. J Bone Joint Surg (Br). 2009;91:16–23.

55 Surgical Technique: Arthroscopic Femoral Osteochondroplasty

J. W. Thomas Byrd

Contents

Abstract

Ganz termed cam impingement "the silent killer of the hip" because advanced damage occurs to the aneural acetabular articular surface before the densely innervated labrum starts to fail, creating symptoms. There is a high predilection for active young adult males where breakdown occurs as the joint exceeds its diminished physiologic limits imposed by the altered morphology. The clinical assessment and imaging are detailed in this chapter. The arthroscope is an important part of the surgical treatment algorithm, identifying the secondary damage that indicates pathological impingement and the need for correction of the underlying cam bump. Most can be corrected arthroscopically and the technique is detailed. With proper patient selection, the results are quite favorable with few complications.

Introduction

Professor Ganz has referred to cam impingement as the silent killer of the hip. That is because the cam lesion results in preferential damage to the aneural articular surface of the acetabulum long before the labrum, with its dense nociceptive innervation, starts to fail, sounding the alarm to the patient that a problem exists.

This author has identified a bimodal population of patients with cam-type FAI [1]. One is the typical middle-aged patient (average age 43 years, with a 1.9:1 male/female ratio) who presents with

J.W.T. Byrd
Nashville Sports Medicine Foundation, Nashville, TN, USA
e-mail: info@nsmfoundation.org; byrd@nsmoc.com

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_59

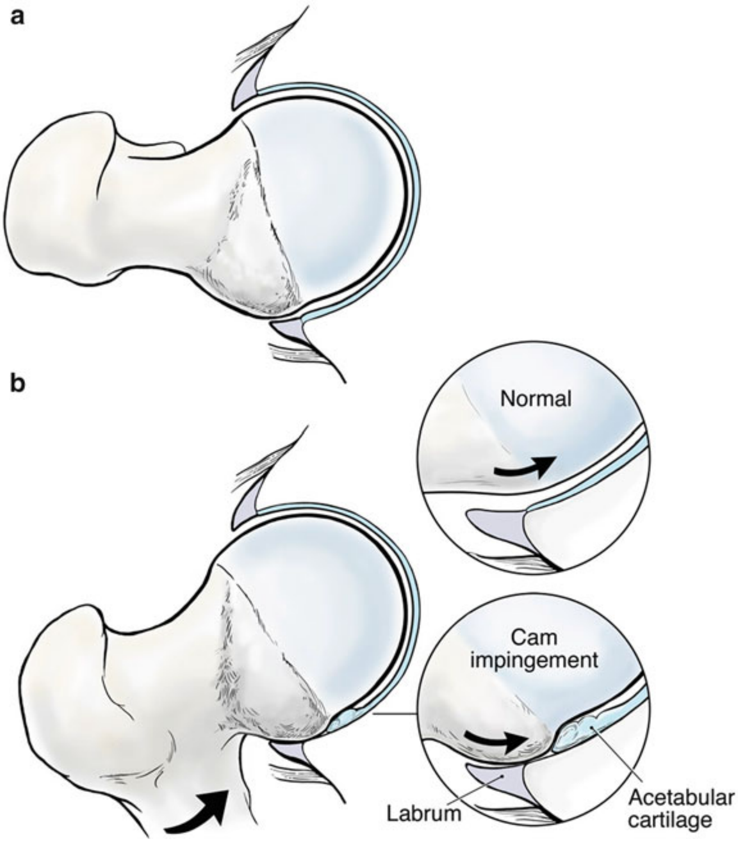


Fig. 1 Cam impingement occurs with hip flexion as the bony prominence of the nonspherical portion of the femoral head (cam lesion) glides under the labrum engaging the edge of the articular cartilage and results in progressive delamination. Initially, the labrum is relatively preserved, but secondary failure occurs over time (© J. W. Thomas Byrd, reprinted with permission)

early age onset osteoarthritis. The second population is much younger, with an even greater male preponderance (average age 20 years, with 3.1:1 male/female ratio), and most (70 %) are involved in athletic activities. These are active individuals who push their hips beyond the diminished physiologic limits and sustain substantial joint breakdown at a young age.

Anatomy/Pathoanatomy

Cam-type femoroacetabular impingement refers to the cam effect created by a nonspherical femoral head. During flexion, the prominence of the out-of-round portion rotates into the acetabulum, engaging against its surface, resulting in delamination and failure of the acetabular articular cartilage (Fig. 1). Early in the disease process, the labrum is relatively preserved but, with time, it begins to sustain secondary damage.

Cam impingement is classically attributed to a slipped capital femoral epiphysis, resulting in a bony prominence of the anterior and anterolateral head/neck junction. However, the most common cause is the pistol grip deformity, attributed to a developmental abnormality of the capital physis during growth. The exact etiology is unclear; it may represent premature asymmetric closure of the physis, and it has been postulated that this could be due to late separation of the common proximal femoral growth plate that forms the physis of the greater trochanter and femoral head [2].

Femoroacetabular impingement is still incompletely understood. The pathomechanics explain the observations of secondary joint pathology caused by the impingement. However, some individuals with impingement-shaped hips may never become symptomatic due to secondary damage. Thus, it is possible to have impingement *morphology* without impingement *pathology*. The arthroscope has become invaluable in the treatment

Fig. 2 The impingement test is performed by provoking pain with flexion, adduction, and internal rotation of the symptomatic hip (© J. W. Thomas Byrd, reprinted with permission)

Fig. 3 Abduction and external rotation may elicit pain with laterally based cam lesions (© J. W. Thomas Byrd, reprinted with permission)

algorithm for patients with FAI. Arthroscopic observations on the secondary articular and labral damage associated with pathological impingement dictate the need for correcting the underlying bony abnormalities.

Patient Selection

History and Physical Examination

Patients with cam impingement have typical hip joint-type symptoms [3]. The onset may be gradual or associated with an acute episode, which is the culmination of altered wear developing over a protracted period of time. Patients with cam impingement usually have reduced joint motion which can result in other secondary disorders. Athletes compensate with increased pelvic motion, often resulting in problems with athletic pubalgia [4]. More stress is placed on the lumbar spine, resulting in concomitant lumbar disease.

Pain with flexion, adduction, and internal rotation is almost uniformly present and is referred to as the "impingement test" (Fig. 2) [5]. However, in this author's experience, this maneuver is uncomfortable for most irritable hips, regardless of the underlying etiology, and thus is not specific for impingement. Laterally based cam lesions may result in painful abduction or external rotation (Fig. 3). Internal rotation of the flexed hip is usually diminished but may be preserved in some patients (Fig. 4). Limited range of motion may be present bilaterally as the morphological variation is often present in both hips.

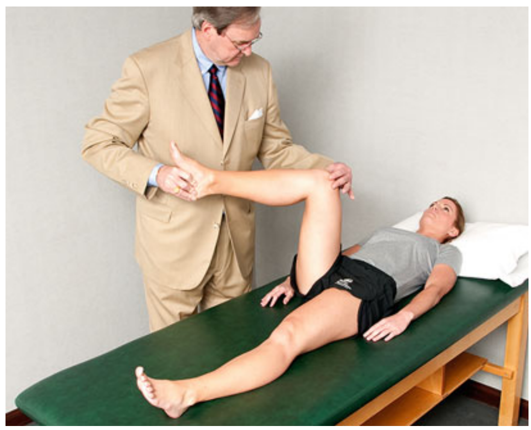


Fig. 4 Internal rotation is checked with the hip in a 90° flexed position (© J. W. Thomas Byrd, reprinted with permission)

Diagnostic Imaging

Radiographs are essential to the routine evaluation of impingement. A well-centered AP pelvis X-ray is important for assessing the acetabular indices of pincer impingement but also allows observations

Fig. 5 A properly centered AP radiograph must be controlled for rotation and tilt. Proper rotation is confirmed by alignment of the coccyx over the symphysis pubis (*vertical line*). Proper tilt is controlled by maintaining the distance between the tip of the coccyx and the superior border of the symphysis pubis at 1–2 cm (© J. W. Thomas Byrd, reprinted with permission)

Fig. 6 The frog lateral radiograph is convenient because it is simple to obtain in a reproducible fashion. The cam lesion (*arrow*) is evident as the convex abnormality at the head/neck junction where there should normally be a concave slope of the femoral neck (© J. W. Thomas Byrd, reprinted with permission)

on the cam lesion by comparing both hips (Fig. 5) [6]. The epicenter and shape of the cam lesion are variable. Thus, while the 40° Dunn view has been reported as the best image, in this author's experience, no single lateral radiographic view is reliable for optimally assessing the cam lesion in all cases (Fig. 6) [7]. Sometimes the cam lesion is more anteriorly based and sometimes more lateral. The characteristic feature is loss of sphericity of the femoral head. The alpha angle has been described to quantitate this observation (Fig. 7) [8]. However, imaging will under interpret this measurement unless it catches the maximal location of the cam lesion. No studies have shown a significant correlation between the amount of alpha angle correction and the results of surgery, indicating that there may be other factors at play; but higher alpha angles have been associated with more clinically relevant lesions [9, 10].

Magnetic resonance imaging (MRI) and gadolinium arthrography with MRI (MRA) aid in assessing secondary damage to the articular cartilage and labrum associated with cam impingement [11]. These studies are better at detecting labral pathology and less often reveal the severity of articular involvement. Alpha angle can again be recorded but is still variable depending on whether the cross-sectional images catch the maximal location of the cam lesion.

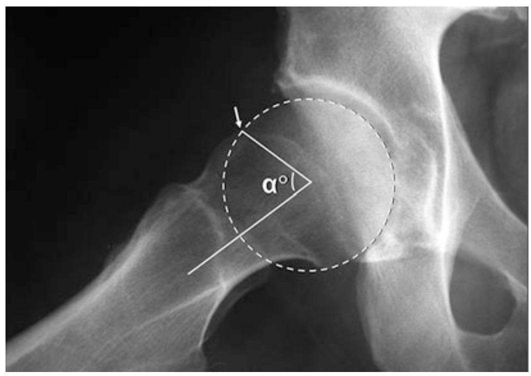


Fig. 7 The alpha angle is used to quantitate the severity of the cam lesion. A circle is placed over the femoral head. The alpha angle is formed by a line along the axis of the femoral neck (1) and a line (2) from the center of the femoral head to the point where the head diverges outside of the circle (*arrow*) (© J. W. Thomas Byrd, reprinted with permission)

Computed tomography with 3-D reconstruction provides great clarity in evaluating the shape, size, and location of the cam lesion. This is quite valuable for the arthroscopic management of this condition. Exposure of the abnormal bone is simplified by knowing its exact appearance.

Indications/Contraindications

The indication for hip arthroscopy is imaging evidence of intra-articular pathology amenable to arthroscopic intervention, or sometimes simply recalcitrant hip pain that remains refractory to efforts at conservative treatment, keeping in mind that imaging studies may often underestimate the severity of intra-articular pathology. Correction of the cam lesion is performed, especially when there is arthroscopic evidence that it is responsible for the concomitant joint pathology. This secondary joint damage is best characterized by failure of the anterolateral acetabular articular surface. The failure is most typically represented by articular delamination with the peel-back phenomenon but, earlier in the disease process, may be characterized by simply deep closed Grade I articular blistering, referred to as the wave sign [12].

It is this author's opinion that simply radiographic findings of impingement, in the absence of clinical findings of a joint problem, are not an indication for arthroscopy. Some individuals with impingement morphology may function for decades without developing secondary joint damage and symptoms. For some it is unclear when, or if, they will develop problems warranting surgical intervention. For example, many individuals may present with symptoms in one hip when radiographic findings of impingement are present in both. While intervention in the asymptomatic joint would not be appropriate, it is important to educate the patient about warning signs of progressive joint damage. It is a clinical challenge in the decision not to intercede too early or too late. In this author's experience, 93 % of patients undergoing arthroscopy for cam impingement demonstrate Grade III and Grade IV articular damage, reflecting that the disease process is already substantially advanced at the time of intervention [1].

Objective contraindications include advanced disease states characterized by Grade 3 Tonnis changes, or less than 2 mm remaining joint space [13–15]. Prominent cam lesions, almost by definition, constitute a Grade 2 Tonnis change and broad spectrum of disease. Larson has subcategorized Tonnis 2 into those with greater or less than 50 % joint space remaining, showing poorer results among those with less than 50 % residual space [15]. Subjective contraindications may include the patient's expectations of surgery. If the patient has unreasonable goals of what the procedure may accomplish, then surgery may not be the best option. Also, in the presence of secondary degenerative disease, the potential advantages of a joint preserving procedure must be weighed against the high level of satisfaction associated with joint arthroplasty.

Conservative Treatment

Conservative management begins with an emphasis on early recognition of the underlying impingement disorder. The mainstay of treatment is identifying and modifying offending activities that precipitate symptoms. Some individuals can modify their lifestyles and stabilize the process for years. Efforts can be made to optimize mobility of the joint, but these are only modestly effective since motion is limited by the bony architecture which cannot be corrected with manual techniques. Decompensatory disorders are those secondary problems that develop as individuals struggle to compensate for the chronic limitations imposed by the impingement. A conservative strategy must include assessment and treatment of the secondary problems, which can contribute substantially to the patient's symptoms.

For patients with degenerative disease, treatment may simply be lifestyle modifications to keep the symptoms manageable. For athletes pushing the joint beyond its diminished physiologic limitations, a specific program becomes more important. Optimizing core strength can aid in regaining the athlete's ability to properly compensate. Loading of a flexed hip can be particularly destructive in the presence of impingement; thus, repetitive training activities such as squats and lunges should be avoided, or modified, to limit hip flexion.

Surgical Technique

The procedure begins with arthroscopy of the central compartment to assess for the pathology associated with cam impingement. This is carried out with the standard supine three-portal technique that has been well described in the literature

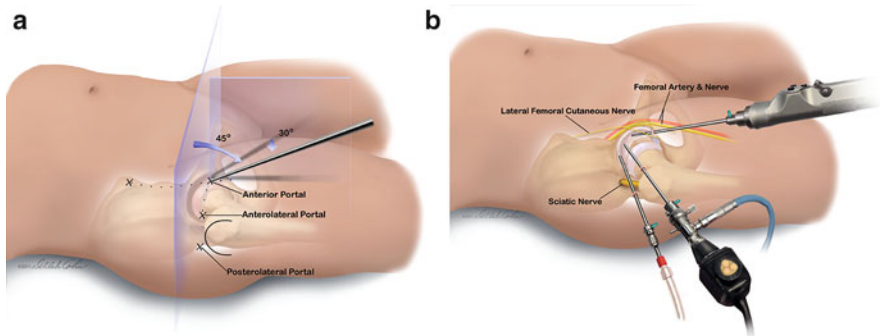


Fig. 8 (**a**) The site of the anterior portal coincides with the intersection of a *sagittal line* drawn distally from the anterior superior iliac spine and a transverse line across the superior margin of the greater trochanter. The direction of this portal courses approximately 45° cephalad and 30° toward the midline. The anterolateral and posterolateral portals are positioned directly over the superior aspect of the trochanter at its anterior and posterior borders. (**b**) The relationship of the major neurovascular structures to the three standard portals is illustrated. The femoral artery and nerve lie well medial to the anterior portal. The sciatic nerve lies posterior to the posterolateral portal. The lateral femoral cutaneous nerve lies close to the anterior portal. Injury to this structure is avoided by using proper portal placement. The anterolateral portal is established first because it lies most centrally in the safe zone for arthroscopy (© J. W. Thomas Byrd, reprinted with permission)

(Fig. 8) [16–18]. The characteristic feature of pathological cam impingement is articular failure of the anterolateral acetabulum. The femoral head remains well preserved until late in the disease course. Early stages of the disease are characterized by closed Grade I chondral blistering, which sometimes must be distinguished from normal articular softening (Fig. 9). This author's experience has been that most patients already have Grade III or Grade IV acetabular changes by the time of surgical intervention [1]. The articular surface is seen to separate or peel away from its attachment to the labrum (Fig. 10) and is caused by the shear effect of the cam lesion. The labrum may be relatively well preserved but, with time, progressive fragmentation occurs.

If the labrum is patent, it is left alone. Often its articular edge is exposed by delamination of the adjacent acetabular cartilage separating away, and the edge can be conservatively smoothed off. If the labral damage is substantial, most can be repaired. Commonly, a combined pincer lesion is also present and is reshaped in conjunction with labral refixation. The articular pathology is addressed with chondroplasty and microfracture as dictated by its severity.

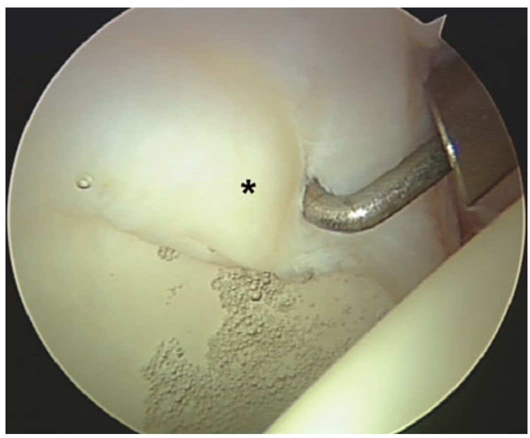


Fig. 9 Pathological chondral blistering (*asterisk*) is being probed from the anterior portal of this right hip. This indicates sublaminar shearing of the articular cartilage associated with pathological cam impingement. Unroofing the blister may reveal partial or full-thickness articular loss (© J. W. Thomas Byrd, reprinted with permission)

After completing arthroscopy of the central compartment, the cam lesion is addressed from the peripheral compartment. A capsulotomy is created by connecting the anterior and anterolateral portals (Fig. 11). The posterolateral portal is removed and the anterior and anterolateral

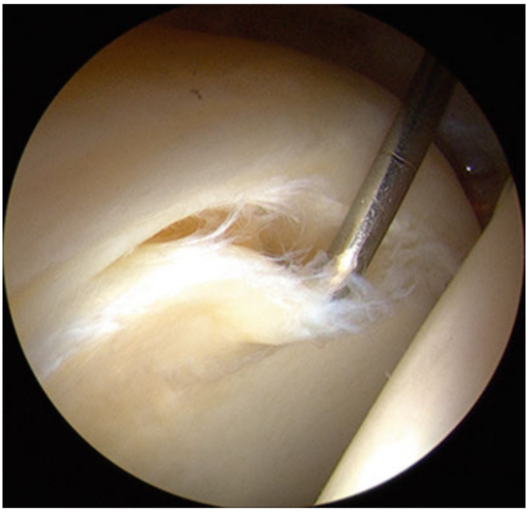


Fig. 10 Viewing a right hip from the anterolateral portal, a probe identifies articular delamination consistent with pathological cam impingement (© J. W. Thomas Byrd, reprinted with permission)

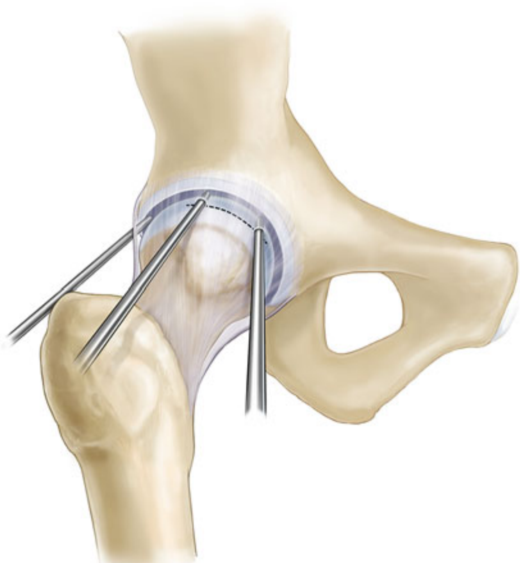


Fig. 11 A capsulotomy is performed by connecting the anterior and anterolateral portals (*dotted line*). This is geographically located adjacent to the area of the cam lesion. This capsulotomy is necessary in order for the instruments to pass freely from the central to the peripheral compartment as the traction is released and the hip flexed (© J. W. Thomas Byrd, reprinted with permission)

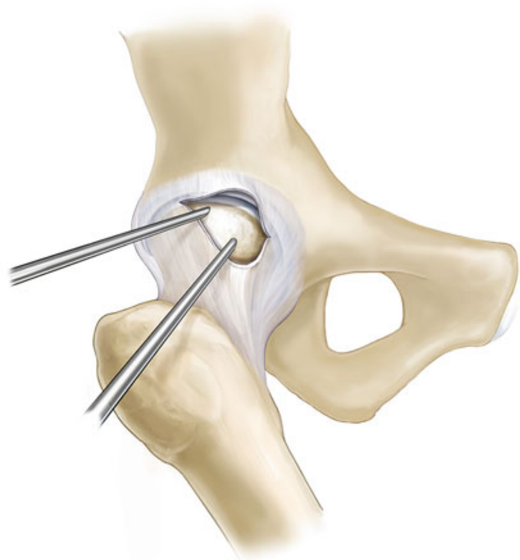


Fig. 12 With the hip flexed, the anterolateral portal is now positioned along the neck of the femur. A cephalad (proximal) anterolateral portal has been placed. These two portals allow access to the entirety of the cam lesion in most cases. Their position also allows an unhindered view with the c-arm (© J. W. Thomas Byrd, reprinted with permission)

cannulas are simply backed out of the central compartment. The traction is released and the hip flexed approximately 35°. As the hip is flexed under arthroscopic visualization, the line of demarcation between healthy femoral cartilage and abnormal fibrocartilage that covers the cam lesion can usually be identified.

A cephalad anterolateral portal is established approximately 5 cm above the anterolateral portal, entering through the capsulotomy that has already been established. These proximal and distal anterolateral portals work well for accessing and addressing the cam lesion (Fig. 12). The anterior portal can be removed or maintained if it is needed for better access to the medial side of the femoral neck.

Most of the work for performing the recontouring of the cam lesion (femoroplasty) lies in the soft tissue preparation. This includes capsular debridement as necessary to assure complete visualization of the lesion and then removal of the fibrocartilage and scar that covers the abnormal

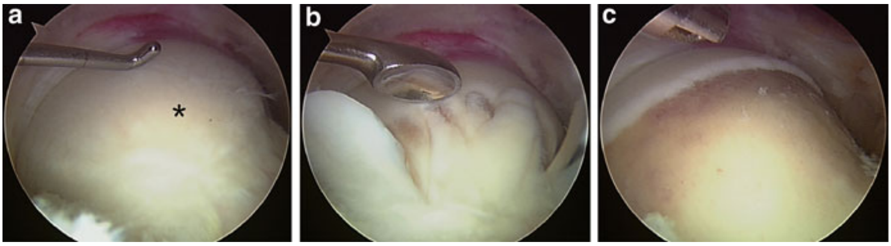


Fig. 13 The right hip is viewed from the anterolateral portal. (**a**) The cam lesion is identified, covered in fibrocartilage (*asterisk*). (**b**) An arthroscopic curette is used to denude the abnormal bone. (**c**) The area to be excised has been fully exposed. The soft tissue preparation aids in precisely defining the margins to be excised (© J. W. Thomas Byrd, reprinted with permission)

bone (Fig. 13a–c). With the hip flexed, the proximal portal provides better access for the lateral and posterior portions, while the distal portal is more anterior relative to the joint and provides best access for the anterior part of the lesion. The lateral synovial fold is identified as the arthroscopic landmark for the retinacular vessels and care is taken to preserve this structure during the recontouring (Fig. 14). Switching between the portals is important for full appreciation of the three-dimensional anatomy of the recontouring.

Once the bone has been fully exposed, recontouring is performed with a spherical burr. The goal is to remove the abnormal bone, identified on the preoperative CT scan, and recreate the normal concave relationship that should exist where the femoral neck meets the articular edge of the femoral head. It is best to begin by creating the line and depth of resection at the articular margin. The resection is then extended distally, tapering with the normal portion of the femoral head (Figs. 15a, b and 16a, b). It is recommended that the resection begin at the lateral/posterior limit of the cam lesion with the arthroscope in the more distal portal and instrumentation in the more proximal portal. The posterior extent of the resection is usually the most difficult; the resection is also the most critical to avoid notching the tensile surface of the femoral neck; and particular attention must be given to avoiding and preserving the lateral retinacular vessels. Then, switching the arthroscope to the proximal portal, the burr is introduced distally, and the reshaping is completed along the anterior head and neck junction. Lastly, attention is given to make sure that all bone debris is removed as thoroughly as possible to lessen the likelihood of developing heterotopic ossification. The quality of the recontouring is assessed, and preservation of the lateral retinacular vessels is confirmed (Fig. 17a–c).

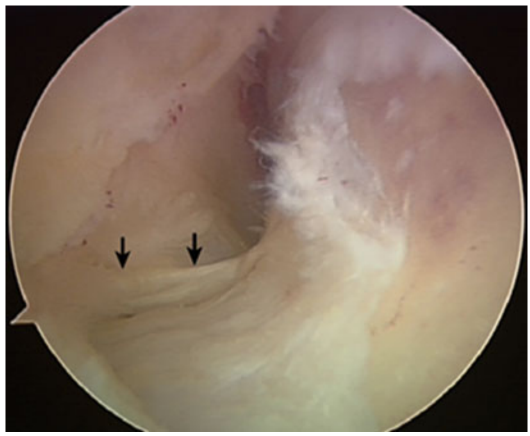


Fig. 14 Viewing laterally, underneath the area of the lateral capsulotomy, the lateral synovial fold (*arrows*) is identified along the lateral base of the neck, representing the arthroscopic landmarks of the lateral retinacular vessels (© J. W. Thomas Byrd, reprinted with permission)

Thoughtful capsular management is important in respect to the risk of creating iatrogenic instability. The slit created by connecting the anterior and anterolateral portals is approximately 1.5 cm and unlikely to be a problem. The capsulotomy is often extended for exposure, and, in some tight hips, this is transformed into a more formal capsulectomy with the hope of providing better mobility. If instability is a concern, most of the capsule can be preserved and closed when the case is completed. This may be a concern in cases

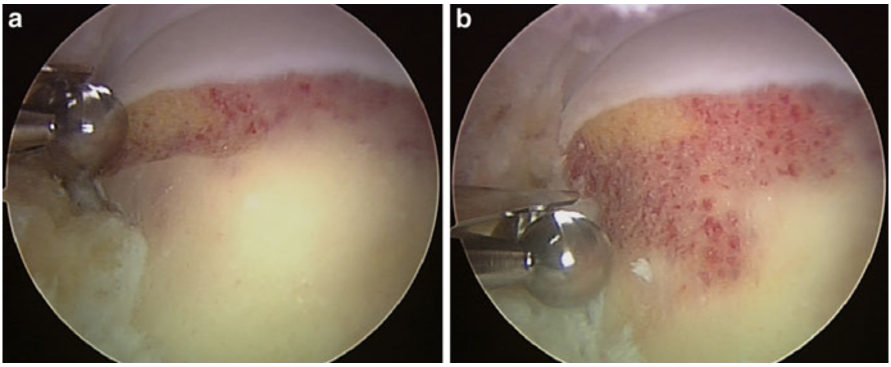


Fig. 15 The arthroscope is in the more distal (anterolateral) portal with the instrumentation placed from the proximal portal. (**a**) Bony resection is begun at the articular margin. (**b**) The resection is then carried distally, recreating the normal concave relationship (© J. W. Thomas Byrd, reprinted with permission)

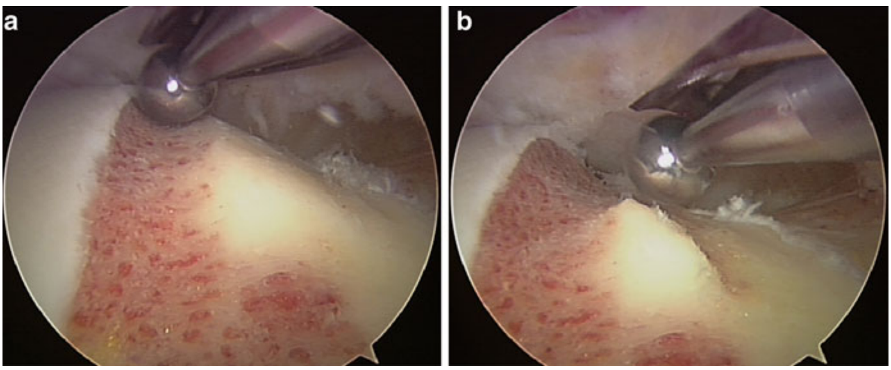


Fig. 16 The arthroscope is now in the proximal portal with the instrumentation introduced distally. (**a**) The line of resection is continued along the anterior articular border of the bump. (**b**) The recontouring is completed (© J. W. Thomas Byrd, reprinted with permission)

where (1) a cam lesion is corrected in conjunction with a shallow acetabulum, (2) patients have global laxity, (3) individuals are returning to activities that require extreme range of motion, and (4) there are some large cam lesions where decompression results in a relative increased capsular volume. With capsular closure, #2 absorbable sutures are used to avoid retained foreign material that can result in capsular scarring and thickening.

Case Reports

A 20-year-old hockey player with a 40-year history of worsening right groin pain was evaluated. Examination revealed diminished internal rotation of both hips (10°). Forced flexion, adduction, and internal rotation of the right hip recreated the characteristic pain that he experienced with activities (Fig. 18a–g).

A 15-year-old female level 10 gymnast presented with a 6-month history of left hip pain unresponsive to conservative treatment including a protracted period of rest. Examination revealed a 20° loss of motion of the left hip compared to the right with pain recreated on flexion, adduction, and internal rotation (Fig. 19a–g).

Rehabilitation

Formal supervised physical therapy begins within 1 or 2 days following surgery. An emphasis is on optimizing range of motion with early

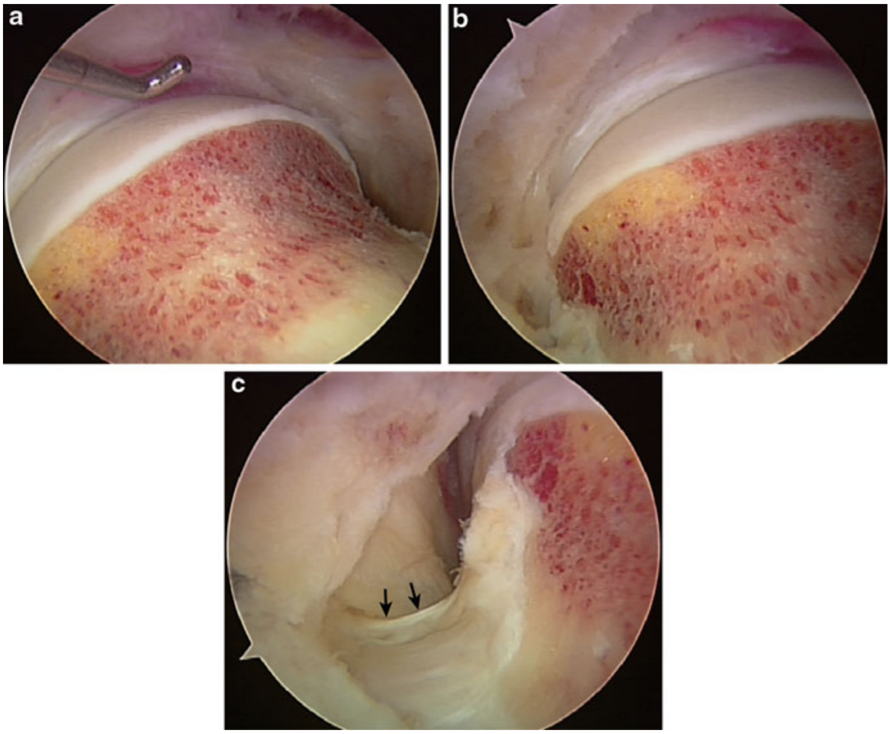


Fig. 17 The arthroscope has been returned to the distal portal for final survey. (**a**) Viewing medially; (**b**) viewing laterally; (**c**) confirming preservation of the lateral retinacular vessels (*arrows*) (© J. W. Thomas Byrd, reprinted with permission)

implementation of closed chain joint stabilization and core strengthening exercises. The patient is allowed to weight bear as tolerated, but crutches are used for 4 weeks as a precautionary measure to protect the femoroplasty site against any awkward twisting episodes. Once normal muscle tone and response patterns have been regained, these will adequately protect the joint for normal forces. Impact loading is avoided for 3 months while the bone fully remodels. The rehab protocol is modified for microfracture by keeping the patient on a strict protected weight-bearing status for 2 months. The patient is allowed to place the weight of the leg on the ground which provides optimal neutralization of forces across the joint. Also, if a labral repair has been performed, excessive flexion and external rotation are avoided for the first 4 weeks. A formal structured rehab protocol is continued for 3 months. For athletes, functional progression is then advanced as tolerated. While some athletes may resume unrestricted activities quickly, it is anticipated that, usually, another 1–3 months are necessary for full participation.

Results

This author reported on 200 patients (207 hips) with 100 % follow-up at 1–2 years.[1] The average age was 33 years (range 13–63). There were 138 males and 62 females with 120 right and 87 left hips. 163 patients underwent femoroplasty to correct cam impingement alone while 44 patients underwent femoroplasty in combination with correction of associated pincer impingement. Overall, the average improvement was 20 points (preop 66; postop 86). 83 % were improved with 83 % good and excellent results using the Harris classification. Viewing the results over time (Fig. 8), continued improvement was noted throughout the first year with results

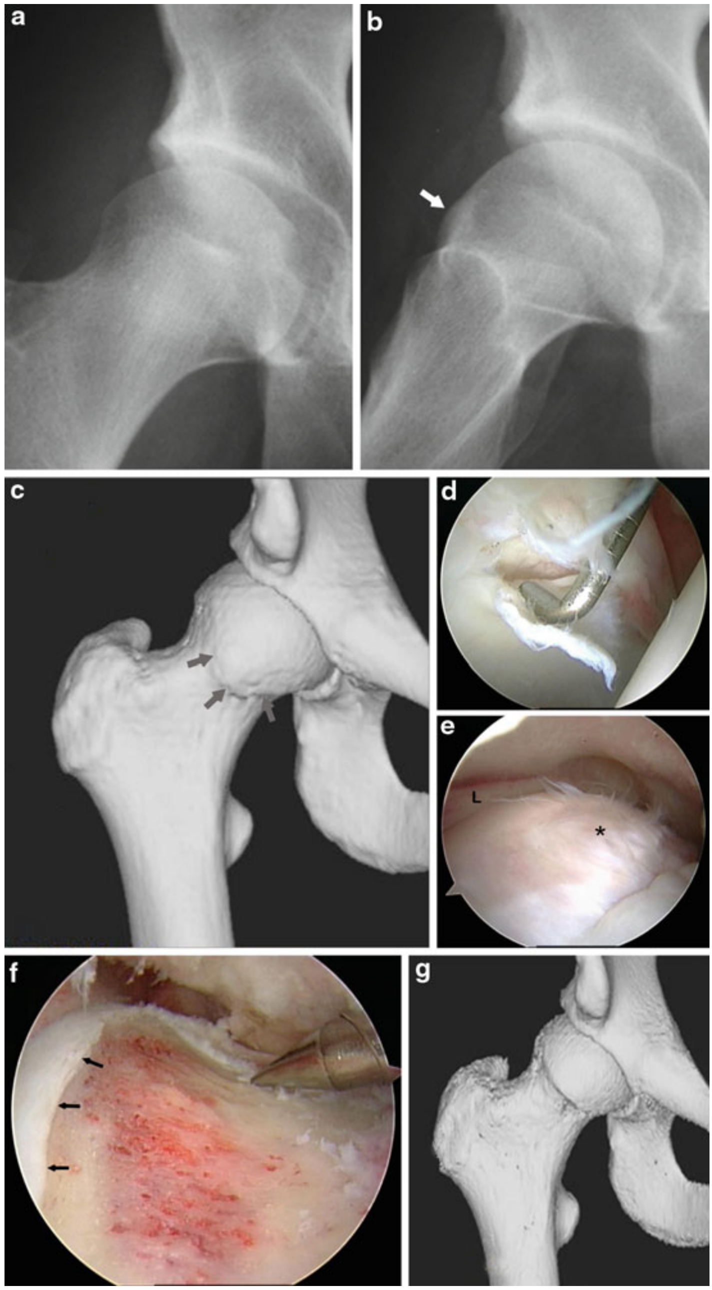


Fig. 18 Images illustrate the case of a 20-year-old hockey player with a 4-year history of right hip pain. (**a**) AP radiograph is unremarkable. (**b**) Frog lateral radiograph demonstrates a morphologic variant with bony buildup at the anterior femoral head/neck junction (*arrow*) characteristic of cam impingement. (**c**) A 3-D CT scan further defines the extent of the bony lesion (*arrows*). (**d**) Viewing from the anterolateral portal, the probe introduced anteriorly displaces an area of articular delamination from the anterolateral acetabulum characteristic of the peel-back phenomenon created by the bony lesion shearing the articular surface during hip flexion. (**e**) Viewing from the peripheral compartment, the bony lesion is identified (*asterisk*) immediately below the free edge of the acetabular labrum (*L*). (**f**) The lesion has been excised, recreating the normal concave relationship of the femoral head/neck junction immediately adjacent to the articular surface (*arrows*). Posteriorly, resection is limited to the midportion of the lateral neck to avoid compromising blood supply to the femoral head from the lateral retinacular vessels. (**g**) A postoperative 3-D CT scan illustrates the extent of bony resection (© J. W. Thomas Byrd, reprinted with permission)

maintained in those with 2-year follow-up. Most had Outerbridge Grade IV (107) or Grade III (83) articular damage on at least one side of the joint. Fifty-eight underwent microfracture with an average 20-point improvement (preop 65; postop 85). Comparing the results of cam and combined lesions, the results were comparable with 20- vs. 19-point improvement while the cam patients tended to be slightly younger, with an average age of 33 vs. 35 for combined lesions. A bimodal age distribution was identified with a peak at age 20 and a second peak at age 43. Ninety-four (45 %) were athletic related. For those patients under age 30 ($n = 88$), 62 (70 %) were associated

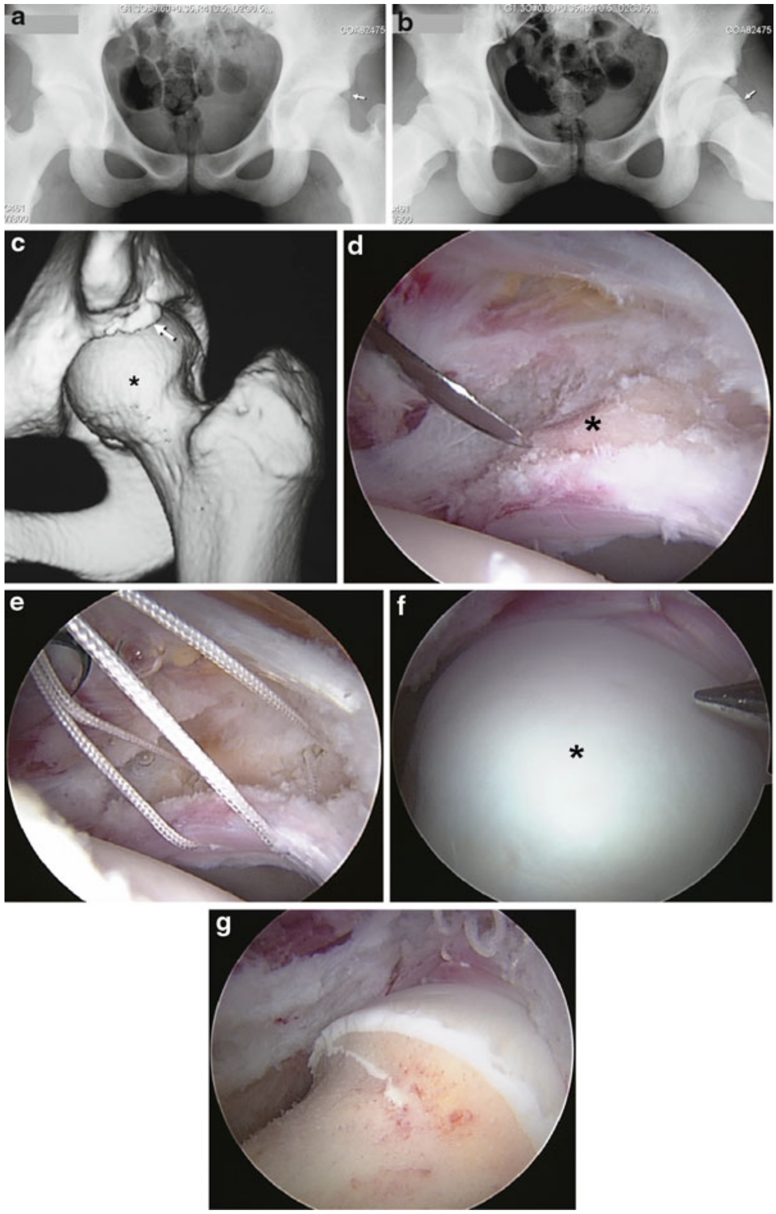


Fig. 19 (**a**–**g**) Images illustrate the case of a 15-year-old female gymnast with pain and reduced internal rotation of the left hip. (**a**) AP pelvis radiograph demonstrates a crossover sign of the left hip with an associated os acetabulum (*arrow*). (**b**) Frog lateral view illustrates asymmetric cam lesion (*arrow*) present in the left hip and not the right. (**c**) A 3-D CT scan further defines the pincer lesion with os acetabulum (*arrow*) and cam lesion (*asterisk*). (**d**) Viewing from the anterolateral portal, the pincer lesion and os acetabulum (*asterisk*) are exposed, with the labrum being sharply released with an arthroscopic knife. (**e**) The acetabular fragment has been removed and the rim trimmed with anchors placed for repair of the labrum. (**f**) Viewing from the periphery, the cam lesion is identified (*asterisk*) covered in fibrocartilage. (**g**) The cam lesion has been excised, recreating the normal concave contour of the head/neck junction adjacent to the site of labral refixation (© J. W. Thomas Byrd, reprinted with permission)

with athletic activities, while after the third decade ($n = 119$) only 30 (25 %) were associated with athletics. The under 30 group was also more male-dominated with a male to female ratio of 3.1:1 compared to the over 30 group with a ratio of 1.9:1. One patient (0.5 %) was converted to a total hip arthroplasty and three patients underwent a second arthroscopic procedure. There were three complications, but none significant. There was one each of a transient neuropraxia of the pudendal nerve and lateral femoral cutaneous nerve, which resolved uneventfully. One case was incidentally noted to have developed heterotopic ossification within the capsule, which did not preclude a successful outcome.

Looking at a more recent cohort of athletes, 163 had cam-type FAI (141 isolated; 22 combined with pincer) in which there was 100 % minimum one-year follow-up [19]. The average age was 29 years with 119 males and 44 females. There were 18 professional, 49 intercollegiate, and 96 high school or recreational athletes. The average improvement was 22 points (preop 71; postop 93) with 89 % of professional and 90 % of intercollegiate athletes returning to their previous level of athletic competition.

Other authors have reported outcomes of arthroscopic management of FAI specifically in regard to correction of the cam lesion. Ilizaliturri, et al. reported improvement in 16 of 19 patients (84 %) with minimum 2-year follow-up [20]. Villar and coauthors reported on femoral osteoplasty in 24 patients with 1-year follow-up compared to a control group in which arthroscopic debridement was performed without excising the impingement lesion [21]. The modified Harris hip score was better in the study group (83) compared to the control group (77); and there was a significantly higher proportion of good/excellent results in the study group (83 %) compared to the control (60 %). Brunner, et al. reported on 45 athletically active individuals from whom 31 (69 %) were able to resume these activities with average 2.4 years of follow-up [22].

Summary

The arthroscope is instrumental in the treatment algorithm for cam impingement. Since cam morphology can exist in the absence of secondary associated joint pathology, the arthroscopic findings substantiate the need for correction of the underlying impingement. Most cases of cam impingement can be addressed arthroscopically. This is a technically demanding procedure that requires meticulous preparation for visualizing the cam lesion and careful orientation in recontouring of the bone to avoid inadequate or excessive resection. The results of the arthroscopic approach are at least comparable to those of the open method, with few complications. Successful results with low morbidity can be expected in the majority of patients, including athletes and individuals seeking to return to an active lifestyle.

References

1. Byrd JWT, Jones KS. Arthroscopic “femoroplasty” in the management of cam-type femoroacetabular impingement. Clin Orthop Relat Res. 2009;467:739–46.
2. Siebenrock KA, Wahab KHA, Werlen S, Kalhor M, Leunig M, Ganz R. Abnormal extension of the femoral head epiphysis as a cause of cam impingement. Clin Orthop Relat Res. 2004;418:54–60.
3. Byrd JWT. Physical examination. In: Byrd JWT, editor. Operative hip arthroscopy. 2nd ed. New York: Springer; 2005. p. 36–50.
4. Meyers WC, Foley DP, Garrett WE, Lohnes JH, Mandlebaum BR. Management of severe lower abdominal or inguinal pain in high-performance athletes. PAIN (Performing Athletes with Abdominal or Inguinal Neuromuscular Pain Study Group). Am J Sports Med. 2000;28(1):2–8.
5. Ganz R, Parvizi J, Beck M, Leunig M, Notzli H, Siebenrock KA. Femoroacetabular impingement: a cause for osteoarthritis in the hip. Clin Orthop. 2003;417:112–20.
6. Parvizi J, Leunig M, Ganz R. Femoroacetabular impingement. J Am Acad Orthop Surg. 2007;15(9):561–70.
7. Meyer DC, Beck M, Ellis T, Ganz R, Leunig M. Comparison of six radiographic projections to assess femoral head/neck asphericity. Clin Orthop. 2006;445:181–5.
8. Notzli H, Wyss TF, Stoecklin CH, Schmid MR, Treiber K, Hodler J. The contour of the femoral head/neck junction as a predictor for the risk of anterior impingement. J Bone Joint Surg Br. 2002;84(4):556–60.
9. Philippon MJ, Wolff AB, Briggs KK, Zehms CT, Kuppersmith DA. Acetabular rim reduction for the treatment of femoroacetabular impingement correlates with preoperative and postoperative center-edge angle. Arthroscopy. 2010;26(6):757–61.
10. Larson CM, Sikka RS, Sardelli MC, Byrd JWT, Kelly BT, Jain RK, Giveans MR. Increasing alpha angle is predictive of athletic-related “hip” and “groin” pain in collegiate national football league prospects. Arthroscopy. 2013;29(3):405–10.
11. Byrd JWT, Jones KS. Diagnostic accuracy of clinical assessment, MRI, gadolinium MRI, and intraarticular injection in hip arthroscopy patients. Am J Sports Med. 2004;32(7):1668–74.
12. Philippon MJ, Schenker ML. Arthroscopy for the treatment of femoroacetabular impingement in the athlete. Clin Sports Med. 2006;25(2):299–308.
13. Tonnis D, Heinecke A. Acetabular and femoral anteversion: relationship with osteoarthritis of the hip. J Bone Joint Surg Am. 1999;81:1747–70.
14. Philippon MJ, Briggs KK, Yen YM, et al. Outcomes following hip arthroscopy for femoroacetabular

impingement with associated chondrolabral dysfunction: minimum two-year follow-up. J Bone Joint Surg Br. 2009;91:16–23.
15. Larson CM, Giveans MR, Taylor M. Does arthroscopic correction improve function with radiographic arthritis? Clin Orthop Relat Res. 2010;468: 555–64.
16. Byrd JWT. Hip arthroscopy utilizing the supine position. Arthroscopy. 1994;10(3):275–80.
17. Byrd JWT. Routine arthroscopy and access: central and peripheral compartments, iliopsoas bursa, peritrochanteric, and subgluteal spaces. In: Byrd JWT, editor. Operative hip arthroscopy. 3rd ed. New York: Springer; 2012. p. 131–60.
18. Byrd JWT. Hip access: fundamental principles. Sports medicine & arthroscopy multimedia education center, American academy of orthopaedic surgeons 2012 annual meeting, San Francisco, 7–11 Feb; 2012.
19. Byrd JWT, Jones KS. Arthroscopic management of Femoroacetabular Impingement (FAI) in athletes. Am J Sports Med. 2011;39:7S–13.
20. Ilizaliturri VM, Orozco-Rodriguez L, Acosta-Rodriguez E, Camacho-Galindo J. Arthroscopic treatment of cam-type femoroacetabular impingement. J Arthroplasty. 2008;23(3):226–34.
21. Bardakos NV, Vasconcelos JC, Villar RN. Early outcome of hip arthroscopy for femoroacetabular impingement: the role of femoral osteoplasty in symptomatic improvement. J Bone Joint Surg Br. 2008;90:1570–5.
22. Brunner A, Horisberger M, Herzog RF. Sports and recreation activity of patients with femoroacetabular impingement before and after arthroscopic osteoplasty. Am J Sports Med. 2009;37:917–22.

Part V

Enthesopathy and Neuromuscular Conditions

Osteitis Pubis

56

Jozef Murar and Patrick M. Birmingham

Contents

Abstract

Osteitis pubis refers to lower abdominal or groin pain arising from hypermobility of the pubic symphysis, degenerative change to the symphyseal cartilaginous disk, and stress reaction in the peri-symphyseal bone. It is initially treated conservatively, but if symptoms persist after 12 weeks, surgical intervention is an option. A number of procedures have been described, but they all involve either resecting the symphyseal cartilaginous disk or fusing the joint. Both types of procedures have reasonable return to play rates.

Introduction

Groin pain is a frequent problem in athletes, particularly in those involved in sports that require kicking, twisting, cutting, sprinting most frequently seen in soccer, Australian rules rugby, ice hockey, and long distance running [1–5]. In fact, sports-related injury rates of the groin range from 0.5 % to 6.2 % [6]. In soccer up to 13 % of injuries involve the groin, and in one series, 58 % of soccer players have experienced a groin injury [7–9].

Osteitis pubis is one of many etiologies of groin pain in athletes and has been shown to be the third most common cause of groin pain in the athlete, preceded only by sports hernia and adductor pathology [10]. It was first described in 1924 by Beer et al. as a complication of suprapubic

J. Murar (✉)
Orthopaedic Surgery Resident, Department of Orthopaedic Surgery, Northwestern University, Chicago, IL, USA
e-mail: j-murar@md.northwestern.edu

P.M. Birmingham
Orthopaedic Sports Medicine, NorthShore University Health Systems, Chicago, IL, USA
e-mail: patrickbirmingham@gmail.com

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_60

surgery and later described in athletes in 1932 by Spinelli et al. [6].

In athletes, osteitis pubis is defined as a painful overuse stress injury of the pubic symphysis and parasymphyseal bone due to chronic overloading stresses. These stresses lead to altered biomechanics due to core muscle injury, which can lead to symphyseal instability [11, 12]. The chronic overuse injury initially causes stress reaction in the pubic bone, and later degenerative changes to the symphysis [13]. Interestingly, bone biopsies of patients with osteitis pubis showed formation of new woven bone, osteoblasts, neovascularization, and stellate fibroblasts with a complete absence of any inflammatory cells or signs of osteonecrosis [11]. Similarly, biopsies of the symphyseal cartilage disk at the time of curettage for osteitis pubis showed degenerative cartilage with complete absence of inflammatory cells [13].

It is important to be aware, however, that there are different etiologies of osteitis pubis. For example, osteitis pubis has also been associated with a number of clinical scenarios including vaginal birth, pelvic and perineal surgery, infection, and rheumatoid arthritis [4, 11]. These may have a different underlying pathology compared to osteitis pubis in athletes [4, 11]. Coventry and Mitchell published a series of studies in 1961 where they described osteitis pubis as an inflammatory disease, as seen with histology. Their study population was composed of patients with osteitis pubis and a history of infection or pelvis surgery rather than athletes [14]. Although there are multiple causes of osteitis pubis, this chapter will focus on the pertinent anatomy, patient presentation, physical examination, imaging, and treatment options of athletic osteitis pubis.

Anatomy

The complexity of the anatomy of the hip joint, pelvis, pubic symphysis, and associated abdominal wall necessitates careful evaluation to accurately diagnose the source of an athlete's groin pain. The bony pelvis has two principal functions: to transfer weight and to withstand compression forces resulting from its support of the weight [15]. It is made up of two innominate bones anteriorly and the sacrum and coccyx posteriorly. The anterior aspect of the pelvis is where the center of core injuries occurs given multiple muscle attachments in the area as well as the pubic symphysis. The pubic symphysis is a non-synovial amphiarthrodial joint that is stabilized by the fibrocartilaginous articular disk between the pubic bones and four ligaments as well as multiple tendon attachments [16].

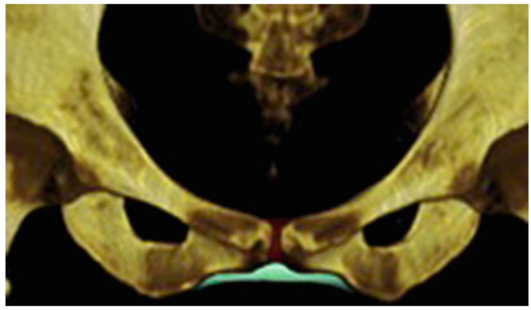


Fig. 1 Diagram of the pubic symphysis demonstrates the midline fibrocartilage pubic disk in *red* and the anteroinferior arcuate ligament in *green* (Reprinted from [17] with permission from Elsevier)

The ligamentous attachments of the pubic symphysis include the arcuate (inferior), superior, anterior, and posterior pubic ligaments. The arcuate and the superior ligaments are the most functionally important for stability and in resisting shear forces. The arcuate ligament lines the inferior aspect of the pubic symphysis superficial to the articular disk and deep to the rectus abdominis/adductor aponeurosis (Fig. 1). The superior ligament spans the space between the pubic tubercles. The anterior ligament blends with fibers of the external oblique and rectus abdominis superficially. The deep portion of the anterior ligament attaches to the intra-articular disk. The posterior ligament is thin and poorly developed providing the least support for stability [16]. The symphysis is innervated with branches of the pudendal and genitofemoral nerves. Its blood supply is derived from branches of all

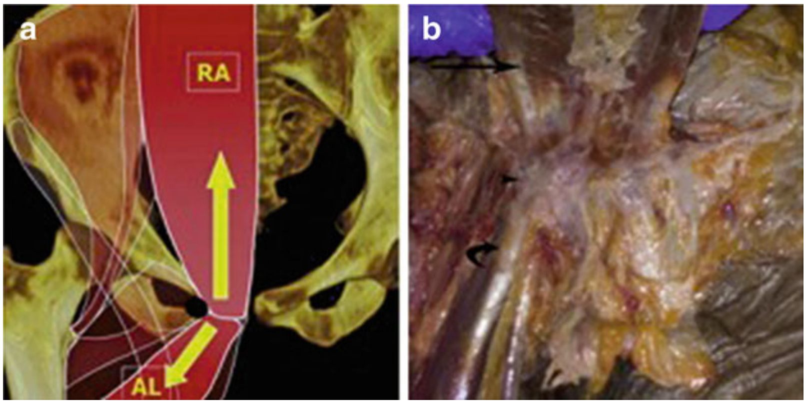


Fig. 2 (**a**) Diagram of the opposing forces of the rectus abdominis (*RA*) and adductor longus (*AL*) at the pubic tubercle. The rectus abdominis creates superoposterior tension, whereas the adductor longus creates inferoanterior tension. Disruption of either leads to altered biomechanics. The *black circle* represents the superficial inguinal ring. (**b**) Gross specimen demonstrates the rectus abdominis (*arrow*), the adductor longus (*curved arrow*), and the pubic tubercle attachment of the rectus abdominis/adductor aponeurosis (*arrowhead*) (Reprinted from [17] with permission from Elsevier)

major vessels in the area including the obturator, internal pudendal, inferior epigastric, and medial femoral circumflex arteries [16].

The pubic symphysis acts as a fulcrum for forces generated at the anterior pelvis. Musculoaponeurotic plate attachments at the pubic symphysis are important for core stability, and coordinated contraction of the muscles that directly attach to the fulcrum produces a slight anterior tilt of the pelvis. The abdominal muscle attachments include the rectus abdominis and internal oblique, external oblique, and transversus abdominis. The medial thigh compartment attachments include the pectineus, adductor longus, adductor brevis, adductor magnus, and gracilis. Functionally, the most important attachments for anterior pelvis stabilization are where the rectus abdominis and the three adductor muscles join to the fibrocartilage plate of the pubic symphysis [16, 18, 19]. The rectus abdominis attaches to the anterior and anteroinferior aspects of the pubic symphysis. The origin of the pectineus and adductors is confluent with the rectus insertion and is defined as the rectus abdominis/adductor aponeurosis [19].

During core rotation and extension, the rectus abdominis and the adductor longus act as antagonists (Fig. 2). The rectus elevates the pelvis, while the adductor depresses it. Injuring one of the components tends to cause abnormal biomechanical forces on opposing muscles and tendons leading to further injury at the aponeurosis and the tenoperiosteal attachments [20]. Detachment of the aponeurosis itself can lead to instability of the pubic symphysis, as can injury to the arcuate or anterior pubic ligaments. Performing activities such as running with an unstable pubic symphysis can lead to detachment of the fibrocartilage plate from the periosteum of the pubis, exacerbating the instability and increasing shearing forces. This may cause an increased stress reaction in the bone and can ultimately lead to fluid crossing the cortex, cyst formation, and symphyseal arthritis [7, 11–13, 17, 21].

Differences in anatomy of males and females exist due to demands of childbirth on the female anatomy. The female pelvis is a more stable pelvis in that it has fewer shifts in forces and has a relatively wider subpubic angle leading to a different distribution of forces. These forces are likely protective with regard to the development of core injuries in women [22]. The fibrocartilaginous disk and the inner dimensions are also wider in women. The symphysis has 2–3 mm more mobility in women as well, which can increase by up to 10 mm during pregnancy [16].

Patient Presentation

Groin injuries are most common in athletes who participate in sports that require repetitive twisting, pivoting, cutting motions, and frequent acceleration and deceleration. Ice hockey, soccer, and rugby have a particularly high incidence [1–5]. Initial presentation of osteitis pubis typically includes gradual onset of pain over the medial and anterior groin. In some cases pain is present directly over the pubic symphysis, which may be tender to palpation. Other times, tenderness may be palpated over the superior pubic rami, and pain may be felt in the adductor musculature, lower abdominal muscles, perineal region, inguinal region, or scrotum. This pain can progress to the point where the patient is unable to participate in sports. Pain is usually aggravated by athletic activity, particularly with cutting, twisting, and kicking activities as well as with resisted hip adduction or flexion and eccentric loads to the rectum abdominis [1, 2, 15, 23, 24].

It is important to consider the differential diagnosis of osteitis pubis given it shares many of its symptoms with other groin injuries. These include other musculoskeletal etiologies of groin pain such as athletic pubalgia, adductor strain, stress fracture, inguinal hernia, intra-articular hip pathology, femoroacetabular impingement, iliopsoas injury, and abnormality of the lower lumbar spine.

Perhaps the most important differential diagnosis is osteomyelitis of the pubic symphysis, which presents in a similar fashion as osteitis pubis and has been reported in the literature to spontaneously occur in athletes [6, 25, 26]. Osteomyelitis can be differentiated based on the acute, atraumatic onset of symptoms that mimic osteitis pubis, along with signs of systemic signs of infection, such as fever, or elevated markers of inflammation such as erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), and leukocytosis [25, 26]. It is important to differentiate the two because the treatments for the two conditions radically differ.

Other atraumatic acute onset groin pains that must be ruled out include testicular torsion, cystitis, appendicitis, nephrolithiasis, and other intra-abdominal and pelvic non-musculoskeletal conditions.

Physical Exam

After a thorough history, a comprehensive physical examination is required to evaluate a patient with groin pain. The physical exam can help with the diagnosis as well as which particular type or sequence of diagnostic imaging to obtain.

Assessment for osteitis pubis should begin with palpation of the pubic symphysis and pubic rami, insertion of the rectus abdominis, adductor origin, external and internal obliques, transversus abdominis, pectineus, gracilis, and inguinal ring for areas of tenderness. Exam findings for osteitis pubis frequently overlap with athletic pubalgia and include tenderness of the pubic symphysis (67 %), adductor origin tenderness (59 %), pain with adductor squeeze test (96 %), and apprehension throughout hip range of motion, particularly with internal rotation. Pain can also be elicited by hip flexion or eccentric loading of the rectus abdominis and with resisted adduction [1, 2, 27–29]. More severe cases may present with a typical "waddling" gait pattern [23].

It is important to rule out intra-articular hip pathology with a thorough physical exam of the hip including hip range of motion (flexion, extension, adduction, and abduction), internal and external rotation, and any provocative testing, i.e., to assess for femoroacetabular impingement with the anterior impingement test (flexion, adduction, and internal rotation) which can suggest intra-articular pathology [30]. Palpation of the insertion of the gluteus medius and minimus, short external rotators, and trochanteric bursa should be performed as well, and a lower extremity neurologic exam and straight leg raise may be useful for ruling out lumbar spine pathology.

A diagnosis of osteitis pubis can be confirmed by anesthetic and/or corticosteroid injection into the symphysis [13, 20, 31]. Some centers use this routinely prior to surgical planning to confirm the diagnosis [5].

Imaging

Radiographic Analysis

Plain radiographs obtained for athletes presenting with groin pain are essential for proper diagnosis. They should be evaluated for osteoarthritis, femoroacetabular impingement, dysplasia, fracture, apophyseal avulsion, and osteitis pubis. The series should include an appropriately oriented weight-bearing anteroposterior (AP) pelvis, a Dunn lateral, and a false profile view [32, 33].

The AP view should be used to evaluate for a crossover sign (cephalad acetabular retroversion), the center edge angle of Wiberg (dysplasia and lateral overcoverage), the acetabular index (dysplasia), the joint space (arthritis), and the pubic symphysis. The Dunn lateral should be used to evaluate the alpha angle for any decreased head-neck offset (cam), pincer trough, or synovial herniation pits. The false profile should be used to evaluate for anterior overcoverage or dysplasia, for anterior center edge angle (dysplasia), for anterior and posterior joint space, and for the morphology of the anteroinferior iliac spine.

Some authors suggest the use of a "flamingo view," which is a one-legged AP view of the symphysis used to evaluate for pubic instability. Vertical shift of greater than 2 mm or widening greater than 7 mm indicates instability (Figs. 3 and 4) [4, 28].

Osteitis pubis appears normal in acute cases, but in chronic cases (>6 months), radiographs show cystic changes, sclerosis, fragmentation, widening, or narrowing of the symphysis, and one-legged stance films may suggest instability (Figs. 3 and 4) [13, 31]. These changes need to be correlated with the patient's symptoms as similar findings can be seen in asymptomatic athletes or athletes with different pathologies [7]. One recent study evaluating radiographic predictors of groin pain found that radiographic evidence osteitis pubis (sclerosis, lytic changes, and cystic changes) was symptomatic only 64.2 % of the time, and it was not independently predictive of groin/hip pain [34].

Fig. 3 Standing AP plain film of the pelvis illustrating the classic radiographic features of osteitis pubis: sclerosis, cystic change, and rarefaction of the medial portions of the pubic rami. Reprinted from [28] with permission from Elsevier

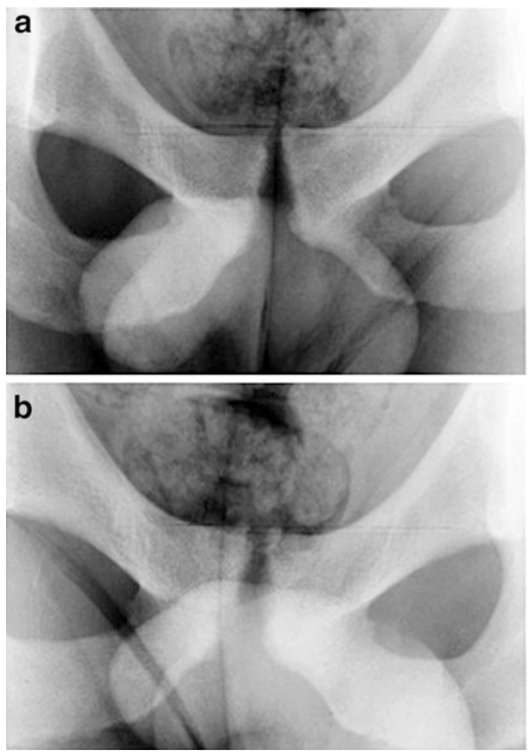


Fig. 4 Vertical symphyseal instability demonstrated by AP flamingo view radiographs. (**a**) Patient standing on left leg. (**b**) Patient standing on right leg. Reprinted from [28] with permission from Elsevier

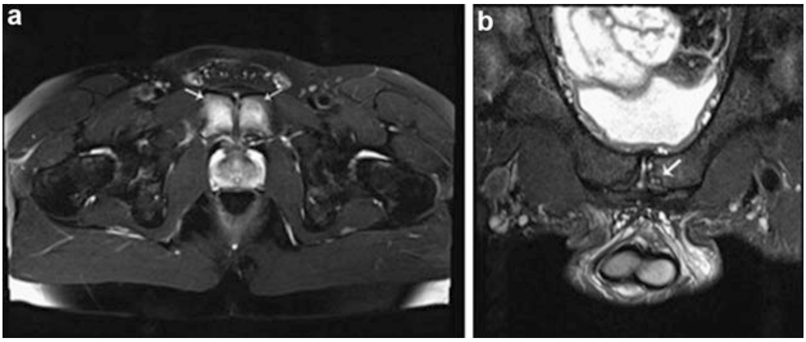


Fig. 5 (**a**) Bone marrow edema spanning the subchondral region of the pubic symphysis anterior to posterior on an axial FSE fat-saturated T2-weighted image (*arrows*) typical for severe osteitis pubis. (**b**) Coronal oblique FSE fat-saturated T2-weighted image shows chronic osteitis pubis with osseous productive change and subchondral cyst formation (*arrow*) (Reprinted from [35] with permission from Elsevier)

Magnetic Resonance Imaging (MRI)

MRI has become the diagnostic modality of choice when advanced imaging is required. It has excellent visualization of soft-tissue abnormalities as well as bone marrow changes, and it can help diagnose core injuries of the abdominal musculature and throughout the pelvis region [6]. When patients present with anterior pelvic pain with concern of groin pathology outside of the hip joint, the MRI study should include dedicated high-resolution imaging of the pubic symphysis and its musculotendinous attachments, as well as a large field view of the entire pelvis, to help exclude other pathologies that can mimic osteitis pubis [17, 21].

Osteitis pubic has acute and chronic forms that can manifest differently on imaging. Acute osteitis pubis on MRI shows bone marrow edema involving the subchondral bone spanning the entire symphysis. The edema is usually bilateral but often asymmetric with more advanced edema involving the symptomatic side (Fig. 5a) [13, 20, 31]. This is best seen using STIR (short tau inversion recovery) or T2 fat-suppression sequences in the coronal plane [36]. The increased signal may be noted over a broad area of the parasymphyseal bone. It can also occur as a hyperintense line paralleling the subchondral bone plate of the pubis that has been found in the majority of symptomatic athletes and may prove to be more clinically relevant than bone marrow edema alone, which can be seen in asymptomatic athletes [27]. This should be differentiated from focal marrow edema at the pubic tubercles, which can be seen with avulsive injuries of the rectus abdominis-adductor aponeurosis.

Severe osteitis pubis can show extensive subchondral marrow edema with articular erosion similar to distal clavicular osteolysis at the acromioclavicular joint. With theses severe lesions, healing potential is unknown [37, 38]. An MRI of chronic osteitis pubis can show less edema but more osseous productive changes with osteophytes, sclerosis, and subchondral cysts secondary to chronic instability at the symphysis (Fig. 5b) [37].

In some cases, on the axial sequences, an abnormal inferior extension of the cleft in the symphyseal fibrocartilage can be seen and has been called a secondary cleft sign (Fig. 5) [20, 39]. This likely represents a microtear of the adductor enthesis. This is important because adductor dysfunction and osteitis pubis frequently coexist, and it has been speculated that adductor dysfunction may precede the development of osteitis pubis [39, 40]. If both pathologies are present, treatment options may need to expand to focus on both pain generators (Fig. 6).

Intra-articular hip pathology such as acetabular labral tears are very common in athletes and are a common differential diagnostic consideration that should be ruled out in patients with groin pain. MRI arthrography has been used for the assessment of intra-articular hip pathology as it has been shown to be superior to conventional MRI in detecting labral

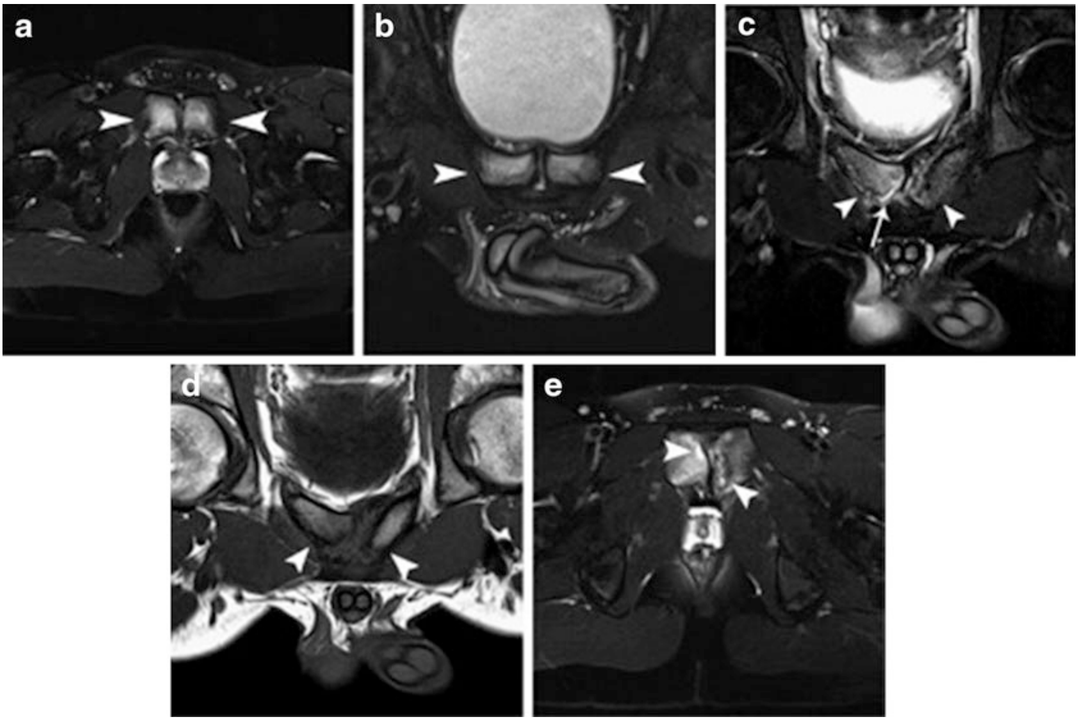


Fig. 6 Osteitis pubis. (**a**, **b**) College football player with typical appearance of severe acute osteitis pubis. (**a**) Large field-of-view axial T2 fat-saturated FSE image of the bony pelvis shows severe acute osteitis pubis (*arrowheads*). Osteitis pubis extends in the anteroposterior direction involving the pubic body rather than the focal edema localized to the pubic tubercle, which is often seen in unilateral caudal rectus abdominis detachments. (**b**) Small field-of-view coronal oblique T2 fat-saturated FSE image using a pubalgia protocol demonstrates the severe acute osteitis pubis (*arrowheads*). No chronic changes are seen, such as degenerative cysts, sclerosis, or osteophytes in this case. (**c**–**e**) College soccer player with severe acute or chronic osteitis pubis. (**c**) Small field-of-view coronal oblique T2 fat-saturated FSE image using a pubalgia protocol demonstrates severe acute or chronic osteitis pubis (*arrowheads*) with disorganization and resorption predominantly affecting the left pubis. A right secondary cleft is present (*arrow*). (**d**) Small field-of-view coronal oblique proton density image using a pubalgia protocol demonstrates severe acute or chronic osteitis pubis (*arrowheads*) with disorganization and resorption predominantly affecting the left pubis. (**e**) Large field-of-view axial T2 fat-saturated FSE image of the bony pelvis demonstrates severe acute or chronic osteitis pubis (*arrowheads*) with disorganization and resorption predominantly affecting the left pubis. Reprinted from [17] with permission from Elsevier

tears (87 % vs. 66 % sensitivity and 64 % vs. 79 % specificity, respectively) [41]. The addition of a local anesthetic to the directly injected contrast can also help distinguish intra-articular from extra-articular causes of pain [42].

CT

Computed tomography can be used to more accurately evaluate the bony morphology of the hip joint in the setting of femoroacetabular impingement or osteitis pubis. Measurements of acetabular and femoral neck version and the alpha angle are taken in addition to three-dimensional reconstructions to better characterize pincer and cam morphology. In osteitis pubis, the CT scan may also show marginal “stamp erosions” of the parasymphyseal pubis better than plain X-rays and MRI (Fig. 7) [43].

Bone Scan

Historically the ^{99m}Tc-methylene diphosphonate triple-phase bone scan has been used with

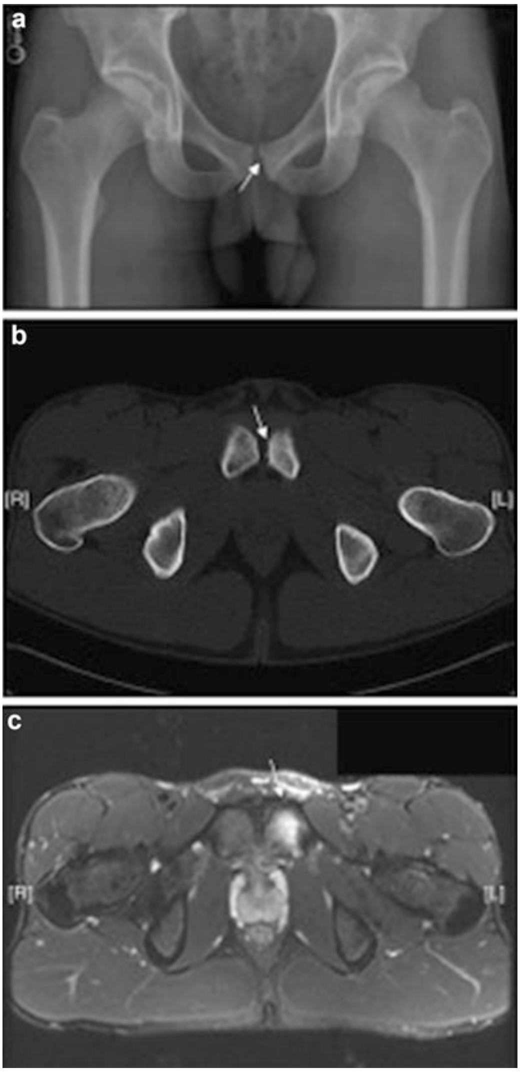


Fig. 7 (**a**) Anteroposterior radiograph and (**b**) axial CT of the pelvis demonstrate stamp erosions of the pubic symphysis on the *left* (*arrow*). (**c**) Axial T2-weighted image with fat saturation demonstrates prominent bone marrow edema within the left pubis (*arrow*). Reprinted from [23] with permission from Elsevier

standard radiographs to evaluate osteitis pubis. The bone scan can also be a helpful screening tool for acute bony pathology including stress fracture or osteomyelitis. Positive bone scans show increased tracer uptake in the region of the pubic symphysis and the parasymphyseal bone, especially on delayed images, but the degree of uptake is poorly correlated with the duration and severity of symptoms [4, 23, 38].

Treatment

Nonoperative

Initial treatment of osteitis pubis should begin with conservative measures typically including rest or activity modification, nonsteroidal anti-inflammatory drugs (NSAIDs), and physical therapy [26]. As mentioned previously, one theory is that osteitis pubis is caused by increased stress at the pubic symphysis due to imbalance between hip adductors and abdominal muscles. It has been shown that active rehabilitation programs that improve coordination and strength of the counterbalancing muscles that attach to the pelvis can help decrease pain in patients with chronic groin pain [44]. Physical therapy should progress from trunk, pelvic, and hip range of motion to stability exercises and to a more complex strength program. Sport-specific exercises are then introduced. Return to activity is usually based on incremental improvement in pain and the athletes' willingness to continue with nonsurgical treatment [44]. One case series demonstrated that earlier diagnosis and initiation of treatment led to fewer symptoms and quicker return to play in athletes [44]. Another small case series demonstrated that use of compression shorts reduced groin pain during exercise in 11 patients with osteitis pubis [45].

There are several studies examining the effectiveness of rehabilitation programs for osteitis pubis [27, 44, 46]. They all propose a graded or progressive approach where a patient progresses with therapy from simple, light exercises to more complex, stressful exercises as mentioned above. However, each study had extremely variable treatments with a wide range of return to activity ranging from 3 days up to 1 year. Symptomatic relief also varied significantly from 41 % to 100 % complete resolution of symptoms [27, 44, 46]. These highly variable treatments and results make these studies difficult to interpret, and further investigation is needed to examine the exact nature of the intervention, as well as functional and clinical criteria for progression and successful treatment.

If the initial period of rest and NSAIDs does not reduce symptoms, a symphyseal corticosteroid injection may be considered. Although no ideal strategy has been published with regard to strength or quantity of steroid medication, multiple studies have showed favorable results with direct injections of one to three cc's of lidocaine and/or bupivacaine with a corticosteroid [3, 24, 47]. Corticosteroid injections are an additional treatment modality that can be an adjunct in accelerating return to play. The timing of the injection may play a role in the overall success rate of the therapy. It has been shown that corticosteroid injection directly into the symphyseal cleft results in a quicker return to sport; however, in most patients, the symptoms return and require additional treatment or another injection [3, 47]. In one case series, patients who received the injection within the first 2 weeks of the diagnosis had a faster return to sport when compared to patients with more chronic symptoms (>16 weeks) who required multiple injections and up to 2 years to recover [24]. Nevertheless, up to 20.7 % of athletes did not respond to injection and were unable to return to sport at all [26]. Given athletic osteitis pubis is a bony stress response rather than an inflammatory process may help explain the marginal results of corticosteroid injections.

After corticosteroid injections, it is important to continue physical therapy since strengthening the core and pelvic musculature helps prevent the recurrence of the condition [12].

Another novel option for conservative treatment of osteitis pubis is prolotherapy or "regenerative injection therapy" consisting of a dextrose and lidocaine injection [48, 49]. The theory behind this therapy is that since osteitis pubis is a degenerative process, an injection of dextrose into the symphysis and surrounding tissues promotes tendon, ligament, and cartilage repair. Topol et al. published two case series demonstrating the efficacy of local dextrose injection in 24 and 72 Australian rules rugby and soccer athletes with good results [48, 49]. Twenty-two of 24 patients were fully participatory in sports within 3 months, and only 20 of 24 had no pain with full participation [49]. In the second study, 66 of 72 athletes returned to full play in a mean time of 3 months with an average of three prolotherapy treatments [48]. Of note, all patients had no improvement after conservative treatment prior to prolotherapy treatment. Additionally, posttreatment follow-up (from 6 to 73 months) demonstrated a significant reduction in pain ranging from 78 % to 82 % [48]. Despite encouraging results, there have been some critiques of this method citing the multitude of injection sites and lack of specific pathology targeting (injections varied and included the pubic symphysis and nearly all muscle attachment sites of the ischiopubic ramus) [50]. Further studies are necessary to replicate the success achieved by Topol et al. and to create a useful protocol for using prolotherapy [23].

Operative

The majority of patients with osteitis pubis respond well to conservative management with rest, physical therapy, oral medications, or local injections, although return to sports can take a considerable amount of time [27, 44, 46]. Nevertheless, 5–10 % of patients fail nonoperative management and may need surgical treatment [31]. A variety of surgical options have been described in the literature including wedge resection, arthrodesis, curettage of the pubic symphysis through open and arthroscopic approaches, and application of retropubic polypropylene mesh to reinforce the abdominal and pelvic musculature [5, 13, 40, 51–53].

Wedge resection of the pubic symphysis was first described in 1961 by Schnute. Use of the wedge resection resulted in significant clinical improvement patients who were severely limited by pain. Grace et al. also reported on ten patients who underwent wedge resection at an average of 32 months after onset of symptoms [52]. All patients improved significantly during the first year after surgery and were able to resume their pre-injury level of activity. At 92 months after surgery, seven patients remained satisfied; however, three patients developed chronic pain and one required fusion of the sacroiliac joints for posterior instability that developed secondary to the wedge resection. Moore et al. reported two additional patients who developed sacroiliac instability after wedge resection. Both of those patients responded well to fusion of the pubic symphysis and bilateral sacroiliac joints. The use of this procedure in high-level athletes has not been performed; however, given the relatively high risk of developing late posterior instability, this technique should be used very cautiously in the athletic population.

Fusion of the pubic symphysis with compression plating and bone grafting has been described as a viable treatment option for chronic osteitis pubis with excellent results [28]. Williams et al. reported on seven rugby players with chronic groin pain who were diagnosed with osteitis pubis and failed at least 13 months of nonoperative management, including at least a 6-month hiatus from rugby. Surgical treatment involved excision of the articular surface of the pubis, cancellous bone grafting and application of a compression plate. Postoperatively the athletes were non-weight bearing for 1 week and 50 % weight bearing for an additional 2 weeks. Light training began 2–3 months postoperatively with return to sport at around the 6-month mark. All seven athletes were able to return to sport. At an average of 52 months after surgery, all were pain free.

Curettage is the most straightforward surgical option; however, the results have been mixed in the athletic population [31]. The procedure was first reported as a small case series by Mulhall et al. in 2002 [51]. They reported on two soccer players who failed conservative therapy and underwent curettage of the symphysis pubis and successfully returned to play within 6 months of the procedure [51]. A larger trial of 23 athletes was performed by Radic et al. in 2008 to evaluate the efficacy of symphyseal curettage [13]. Surgical treatment involved curettage of the articular surface until all articular cartilage was removed and bleeding bone was encountered. The bone was also drilled to promote additional bleeding. No stabilization of the joint was performed. Initially, 70 % of patients were able to return to their previous level of activity at an average of 5.6 months (2.5–12 months) after surgery. However, with longer follow-up, only 39 % remained asymptomatic and 26 % had experienced a one-time recurrence of their symptoms that resolved with rest. Additionally, one patient underwent late fusion of the pubic symphysis.

In 2010, Hechtman at al. reported on a minimally invasive, arthroscopically assisted symphyseal curettage that was successfully performed on four athletes with osteitis pubis. The surgical technique aimed to preserve the adjacent pubic ligaments to ensure they are not debrided so the pelvis will remain stable. Patients returned to full sports on average at 3 months and remained pain-free at final follow-up (average 50 months).

Mesh reinforcement of the pubic symphysis was described by Paajanen et al. in 2005 [40]. They identified 16 athletes with clinical history suggestive of osteitis pubis and MRI demonstrating pubic bone marrow edema and a positive bone scan. All athletes were treated initially with rest, physical therapy, NSAIDs, and injection of the pubic symphysis. Eight patients with more severe symptoms, who failed 6 months of conservative management, elected to undergo surgical intervention which involved laparoscopic placement of mesh behind the pubis held in place with titanium tacks. Two athletes had concomitant adductor or gracilis release. Full activity was allowed 4–8 weeks after surgery. Seven of eight operatively treated patients returned to sport at an average of 2 months postoperatively. One patient required a second procedure, drilling of the pubis, at 6 months after the index procedure. Imaging in that patient suggested pelvic ring instability. At an average of 2.7 years after surgery, all surgically treated patients were competing at their pre-injury level in their pre-injury sport. Of the eight patients treated nonoperatively, four were able to return to their pre-injury level of activity after 1–1.5 years of conservative treatment, and four continued to have some pain at one level below their pre-injury level [40]. For the properly selected patient, surgical management can allow quicker return to sport with better resolution of pain than conservative management. The advantage of this surgical technique is the mesh support to the conjoint tendon and the posterior pubic symphysis, as well as minimal postoperative pain due to laparoscopic nature of the procedure.

There has been one report of treatment of osteitis pubis with an endoscopic decompression of the pubic symphysis in a 31-year-old female with recalcitrant osteitis pubis which occurred in association with FAI [53]. The FAI was also treated arthroscopically at the same sitting. To access the pubic symphysis, two midline portals were used. The first was 2 cm proximal to the superior border of the symphysis, and the second was directly anterior to the symphysis. Ossified symphyseal fibrocartilage and heterotopic bone were removed, and the inferior (arcuate) and posterior pubic ligaments were preserved. Twelve months following this single-stage surgery, the patient reported high satisfaction with decreased pain and improved function and resolution of a classic waddling gait.

Most recently a novel surgical technique introduced by Hopp et al. was published involving five competitive soccer players who suffered from osteitis pubis and concomitant adductor longus tendinopathy [5]. After failure of conservative therapy for at least 12 months, the patients underwent resection of degenerative soft and bone tissues of the adductor longus origin and subsequent reattachment with suture anchors. The symphysis pubis was addressed as well via a two-portal arthroscopic curettage of the degenerative fibrocartilaginous disk. Postoperative rehabilitation involved immediate full weight bearing with limited abduction and adduction for 6 weeks, subsequent increased mobility, and core-strengthening exercises, followed by sport-specific exercises at 3 months post-op. All patients recovered to full activity after an average of 14.4 weeks and remained pain free or with only mild symptoms before and during activity at the last follow-up (average 18.2 months). One patient did have a bleeding complication intraoperatively with successful ligation of the bleeding vessel.

Complications

Surgical interventions are not without complications, and patients must be made aware of the risks of surgery. Complications associated with arthrodesis of the symphysis for osteitis pubis that have been reported include hemospermia, intermittent scrotal swelling, and a stress fracture through the symphyseal arthrodesis requiring a secondary arthrodesis [23, 28].

Late-onset complications such as pubic symphyseal instability have also been reported where the patient required a subsequent fusion of the pubic symphysis or SI joint [13]. Several patients that underwent wedge resection of the pubic symphysis subsequently developed posterior SI joint instability requiring SI joint fusion and symphyseal fusion [52].

The underlying reasons for these complications lie in the inherent risk of such surgery. Damage to one or more of the pubic-stabilizing ligaments can result in an increased pathologic magnitude of motion in the anterior pelvic ring, which can lead to overall instability of the entire pelvis. Great care must be taken to avoid damaging these pelvic stabilizers in order to prevent such complications. More novel arthroscopically assisted repairs aim to minimize damage to the important pubic-stabilizing ligaments to prevent complications of pubic instability; however, larger studies and long-term follow-up are not yet available to fully evaluate the long-term complication rate.

Summary

Osteitis pubis is a painful overuse stress injury that develops due to biomechanical overload of the pubic symphysis and the parasymphyseal bone. Although the differential diagnosis of groin and lower abdominal pain is diverse, a thorough history, physical examination, and appropriate use of imaging can help accurately diagnose osteitis pubis as well as other related conditions. Most of the time, nonoperative treatment is successful and consists of rest, NSAIDs, physical therapy, and occasionally steroid or dextrose injections into the symphysis. There are multiple rehabilitation protocols that all focus on improving stability and strength of core musculature and improving hip range of motion; however, it is unknown which specific protocol works best.

A number of surgical treatments for recalcitrant osteitis pubis have been described; however, most have been described in small studies and some with significant long-term complications. Future studies need to be performed to identify specific diagnostic criteria and therapeutic protocols for athletic osteitis pubis. Until that time, the variability of treatments will remain broad, and deciding which to use will continue to be difficult.

References

1. Macintyre J, Johson C, Schroeder EL. Groin pain in athletes. Curr Sports Med Rep. 2006;5(6):293–9.
2. Verrall GM, et al. Hip joint range of motion reduction in sports-related chronic groin injury diagnosed as pubic bone stress injury. J Sci Med Sport. 2005;8(1): 77–84.
3. Batt ME, McShane JM, Dillingham MF. Osteitis pubis in collegiate football players. Med Sci Sports Exerc. 1995;27(5):629–33.
4. Fricker PA, Taunton JE, Ammann W. Osteitis pubis in athletes. Infection, inflammation or injury? Sports Med. 1991;12(4):266–79.
5. Hopp SJ, et al. Osteitis pubis and adductor tendinopathy in athletes: a novel arthroscopic pubic symphysis curettage and adductor reattachment. Arch Orthop Trauma Surg. 2013;133(7):1003–9.
6. Johnson R. Osteitis pubis. Curr Sports Med Rep. 2003;2(2):98–102.
7. Harris NH, Murray RO. Lesions of the symphysis in athletes. Br Med J. 1974;4(5938):211–4.
8. Ekstrand J, Gillquist J. The avoidability of soccer injuries. Int J Sports Med. 1983;4(2):124–8.
9. Ekstrand J, Hilding J. The incidence and differential diagnosis of acute groin injuries in male soccer players. Scand J Med Sci Sports. 1999;9(2):98–103.
10. Atkins JM, Taylor JC, Kane SF. Acute and overuse injuries of the abdomen and groin in athletes. Curr Sports Med Rep. 2010;9(2):115–20.
11. Verrall GM, et al. Bone biopsy of the parasymphyseal pubic bone region in athletes with chronic groin injury demonstrates new woven bone formation consistent with a diagnosis of pubic bone stress injury. Am J Sports Med. 2008;36(12):2425–31.
12. Beatty T. Osteitis pubis in athletes. Curr Sports Med Rep. 2012;11(2):96–8.
13. Radic R, Annear P. Use of pubic symphysis curettage for treatment-resistant osteitis pubis in athletes. Am J Sports Med. 2008;36(1):122–8.
14. Coventry MB, Mitchell WC. Osteitis pubis: observations based on a study of 45 patients. JAMA. 1961;178:898–905.
15. Meyers WC, et al. Understanding "sports hernia" (athletic pubalgia): the anatomic and pathophysiologic basis for abdominal and groin pain in athletes. Op Tech Sports Med. 2012;20(1):33–45.
16. Gamble JG, Simmons SC, Freedman M. The symphysis pubis. Anatomic and pathologic considerations. Clin Orthop Relat Res. 1986;203:261–72.
17. Palisch A, Zoga AC, Meyers WC. Imaging of athletic pubalgia and core muscle injuries: clinical and therapeutic correlations. Clin Sports Med. 2013;32(3): 427–47.
18. Feeley BT, et al. Hip injuries and labral tears in the national football league. Am J Sports Med. 2008;36(11):2187–95.
19. Mullens FE, et al. Review of MRI technique and imaging findings in athletic pubalgia and the "sports hernia". Eur J Radiol. 2012;81(12):3780–92.
20. Zoga AC, et al. Athletic pubalgia and the "sports hernia": MR imaging findings. Radiology. 2008;247 (3):797–807.
21. Zoga AC, Mullens FE, Meyers WC. The spectrum of MR imaging in athletic pubalgia. Radiol Clin N Am. 2010;48(6):1179–97.
22. Litwin DE, et al. Athletic pubalgia (sports hernia). Clin Sports Med. 2011;30(2):417–34.
23. Hiti CJ, et al. Athletic osteitis pubis. Sports Med. 2011;41(5):361–76.
24. Holt MA, et al. Treatment of osteitis pubis in athletes. Results of corticosteroid injections. Am J Sports Med. 1995;23(5):601–6.
25. Pauli S, et al. Osteomyelitis pubis versus osteitis pubis: a case presentation and review of the literature. Br J Sports Med. 2002;36(1):71–3.
26. Choi H, McCartney M, Best TM. Treatment of osteitis pubis and osteomyelitis of the pubic symphysis in athletes: a systematic review. Br J Sports Med. 2011;45(1):57–64.
27. Verrall GM, et al. Outcome of conservative management of athletic chronic groin injury diagnosed as pubic bone stress injury. Am J Sports Med. 2007; 35(3):467–74.
28. Williams PR, Thomas DP, Downes EM. Osteitis pubis and instability of the pubic symphysis. When nonoperative measures fail. Am J Sports Med. 2000;28(3):350–5.
29. Verrall GM, et al. Hip joint range of motion restriction precedes athletic chronic groin injury. J Sci Med Sport. 2007;10(6):463–6.
30. DeAngelis NA, Busconi BD. Assessment and differential diagnosis of the painful hip. Clin Orthop Relat Res. 2003;406:11–8.
31. Mehin R, et al. Surgery for osteitis pubis. Can J Surg. 2006;49(3):170–6.
32. Clohisy JC, et al. A systematic approach to the plain radiographic evaluation of the young adult hip. J Bone Joint Surg Am. 2008;90 Suppl 4:47–66.
33. Dunn DM. Anteversion of the neck of the femur; a method of measurement. J Bone Joint Surg (Br). 1952;34-B(2):181–6.
34. Larson CM, et al. Increasing alpha angle is predictive of athletic-related "hip" and "groin" pain in collegiate

national football league prospects. Arthroscopy. 2013;29(3):405–10.
35. Khan W, Zoga AC, Meyers WC. Magnetic resonance imaging of athletic pubalgia and the sports hernia: current understanding and practice. Magn Reson Imaging Clin N Am. 2013;21(1):97–110.
36. Meyers WC, et al. Experience with "sports hernia" spanning two decades. Ann Surg. 2008;248(4): 656–65.
37. Kunduracioglu B, et al. Magnetic resonance findings of osteitis pubis. J Magn Reson Imaging. 2007;25(3): 535–9.
38. Verrall GM, Slavotinek JP, Fon GT. Incidence of pubic bone marrow oedema in Australian rules football players: relation to groin pain. Br J Sports Med. 2001;35(1):28–33.
39. Brennan D, et al. Secondary cleft sign as a marker of injury in athletes with groin pain: MR image appearance and interpretation. Radiology. 2005;235(1):162–7.
40. Paajanen H, Hermunen H, Karonen J. Pubic magnetic resonance imaging findings in surgically and conservatively treated athletes with osteitis pubis compared to asymptomatic athletes during heavy training. Am J Sports Med. 2008;36(1):117–21.
41. Smith TO, et al. The diagnostic accuracy of acetabular labral tears using magnetic resonance imaging and magnetic resonance arthrography: a meta-analysis. Eur Radiol. 2011;21(4):863–74.
42. Safran M. Evaluation of the hip: history, physical examination, and imaging. Op Tech Sports Med. 2005;13(1):2–12.
43. De Paulis F, et al. Sports injuries in the pelvis and hip: diagnostic imaging. Eur J Radiol. 1998;27 Suppl 1: S49–59.
44. Wollin M, Lovell G. Osteitis pubis in four young football players: a case series demonstrating successful rehabilitation. Phys Ther Sport. 2006;7(3):153–60.
45. McKim KR, Taunton JE. The effectiveness of compression shorts in the treatment of athletes with osteitis pubis. N Z J Sports Med. 2001;29(4):70–3.
46. Rodriguez C, et al. Osteitis pubis syndrome in the professional soccer athlete: a case report. J Athl Train. 2001;36(4):437–40.
47. O'Connell MJ, et al. Symphyseal cleft injection in the diagnosis and treatment of osteitis pubis in athletes. AJR Am J Roentgenol. 2002;179(4):955–9.
48. Topol GA, Reeves KD. Regenerative injection of elite athletes with career-altering chronic groin pain who fail conservative treatment: a consecutive case series. Am J Phys Med Rehabil. 2008;87(11):890–902.
49. Topol GA, Reeves KD, Hassanein KM. Efficacy of dextrose prolotherapy in elite male kicking-sport athletes with chronic groin pain. Arch Phys Med Rehabil. 2005;86(4):697–702.
50. Rabago D, et al. A systematic review of prolotherapy for chronic musculoskeletal pain. Clin J Sport Med. 2005;15(5):376–80.
51. Mulhall KJ, et al. Osteitis pubis in professional soccer players: a report of outcome with symphyseal curettage in cases refractory to conservative management. Clin J Sport Med. 2002;12(3):179–81.
52. Grace JN, et al. Wedge resection of the symphysis pubis for the treatment of osteitis pubis. J Bone Joint Surg Am. 1989;71(3):358–64.
53. Matsuda DK. Endoscopic pubic symphysectomy for reclacitrant osteitis pubis associated with bilateral femoroacetabular impingement. Orthopedics. 2010; 33(3):199.

Surgical Technique: Osteosynthesis for Pelvic Instability and Osteitis Pubis

57

Jason J. Halvorson and David L. Helfet

Contents

Abstract

Pelvic instability, including osteitis pubis, can be challenging for physicians to diagnose and treat. While initial management of these conditions is almost a universally conservative treatment (including physical therapy, injections, anti-inflammatories, etc.), a subset of patients appear to benefit from surgical intervention as guided by the proposed treatment algorithm. When planning surgery, it is important to remember "the ring" structure of the pelvis, taking into account both the anterior and the posterior pelvic ring. Numerous surgical techniques for both anterior and posterior fixation, with or without formal fusion, have been proposed with varying results in the literature. A brief overview of pelvic instability is provided followed by review of the currently published surgical techniques. Finally, surgical technique regarding both anterior and posterior fixation is described.

Introduction

The treatment of pelvic instability (including osteitis pubis) is initially conservative. However, a select group of patients may benefit from surgical stabilization of their pelvis to improve pain and function. Both conservative treatment measures and surgical techniques for these conditions are varied in the literature. Therefore, a review of the overall treatment strategy and methods of surgical stabilization is presented.

J.J. Halvorson (✉)
Medical Center Boulevard, Hospital for Special Surgery, Wake Forest University School of Medicine, Winston-Salem, NC, USA
e-mail: jhalvorsnd@gmail.com

D.L. Helfet
Weill Medical College of Cornell University, Orthopaedic Trauma Service, Hospital for Special Surgery/New York Presbyterian Hospital, New York, USA
e-mail: helfetd@hss.edu

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_61

Pelvic instability can be challenging for physicians to diagnose and treat, partly due to lack of a unifying and consensus definition or nomenclature. In an effort to clarify and standardize the diagnosis, European physicians attempted to define and put forward guidelines for diagnosis and treatment strategies for pelvic girdle pain (as it was termed) in order to better clarify and standardize the various patients, symptoms, and treatment strategies for this patient group [1]. Multiple etiologic factors have been proposed including sequelae of trauma, infection, postpartum, rheumatologic, sports related (i.e., osteitis pubis), and idiopathic. Osteitis pubis classically occurs in those patients with a history of athletic activity involving primarily kicking (such as soccer) or sharp cutting with acceleration/deceleration [2–6] and is now thought to occur secondary to microtrauma and stress to the pubic symphysis secondary to strains upon the abdominal and/or hip musculature (especially hip adductors) [7–12]. Pelvic girdle relaxation is a term specifically utilized to describe pregnancy-related pelvic pain, defined by Larsen et al. as "a condition developing during pregnancy or delivery. This is characterized by disabling pain located in the sacroiliac joints and/or the pubic symphysis" [13]. Postpartum instability can be further divided into those cases which occur traumatically before or at the time of delivery (acting similar to traumatic pelvic lesions) and those which appear secondary to pelvic girdle relaxation with an apparent maintenance of ligamentous laxity after childbirth [13]. The incidence of acute traumatic injury to the pelvis postpartum has been suggested as 1/600–1/30,000 [14–16]. The exact incidence of pregnancy-related pelvic pain and/or persistent pelvic instability is reported to occur with a wide variation in the literature, most likely secondary to diagnosis and definitions used for inclusion criteria, time point of pain measurement (both pre and post partum), and length of follow-up. Of 855 women followed by Ostgaard et al. during pregnancy and evaluated for high back pain, low back pain, or sacroiliac pain, 27 % of patients reported "back pain," with the majority of these attributed to SI joint pain [17]. In addition, a strong correlation between pubic symphysis pain was found for those patients with SI joint pain. Given the overall definitions, the true incidence of pregnancy-related pelvic instability is likely around 14–20 %. Of this group, it appears approximately 2–24 % of those women continue to have debilitating pain at 12 months [1, 13, 17–20]. In addition, reports of up to one in four women with debilitating postpartum pelvic pain will go on to have chronic pelvic pain [18]. Risk factors for postpartum pelvic pain are varied but include a previous history of pain either prior to or during a previous pregnancy, family members with similar conditions, age, education level, heavy workload, smoking, increased weight, daily stress and work satisfaction, and multiparae [13, 17, 20–22]. While pregnancy-related pelvic pain can begin at any point in pregnancy and as early as the 3rd or 4th month [23], it normally presents within the 5–8th month of pregnancy [13, 24]. Patients with earlier onset pelvic pain during pregnancy or more severe pain during pregnancy are at risk for continued pain postpartum [25]. Likewise, the history of an acute "trauma" or pain at the time of delivery should alert the obstetrician or evaluating orthopedic surgeon to the possibility of a pelvic ring injury similar to that experienced in a traumatic situation with disruption of the anterior pubic symphysis, SI ligaments, and pelvic floor.

Initial radiographic evaluation of any patient with suspected pelvic pathology should include an anterior-posterior, inlet, and outlet pelvis views, allowing the treating physician to assess global pelvic stability in the vertical (outlet view) and anterior-posterior plane (inlet view) along with pubic diastasis, if present. In osteitis pubis, radiographic imaging classically demonstrates bony sclerosis/erosions/lysis as well as cystic changes [2, 26]. Flamingo views are beneficial in assessing abnormal motion at the pubic symphysis [27, 28], though the absence of instability on flamingo views does not rule out the presence of osteitis pubis [7] and up to 36 % of patients may present with persistent postpartum pain but have negative "flamingo" views [23]. Radiographically, it has been suggested that a pubic diastasis of >10 mm and vertical displacement >5 mm [27] is an indication for surgery, though wider displacements may be followed conservatively with good functional outcomes. Therefore, it is imperative that the radiographs be interpreted in association with the clinical history and exam before interventions are considered [16, 29–32]. If further imaging is required, ultrasound, MRI, and CT scan have all been proposed as potential modalities for assessment. Garagiola et al. examined the CT scans of 14 postpartum patients in comparison to 15 controls (CT scan done for trauma, abdominal pain, or evaluation of mass – all of which reported as normal) in an effort to identify "normal" postdelivery anatomy [33]. They found that roughly half (46 %) of patients had an increased pubic symphysis width (average 6.5 mm) compared to controls and that one patient had SI joint widening compared with controls. Gas could be identified in 31 % of patients within the pubic symphysis and 42 % of patients within the SI joint (Garagiola). However, no correlations to patient symptoms were made making conclusions about the relevance of these findings in clinical practice difficult. Elgafy et al. examined patients with clinical exam findings consistent with SI joint pain and patients with improvement of pain after SI joint injection. While CT scan was positive after review (including subchondral sclerosis, osteophytes, cyst formation) in 57.5 % of patients with symptomatic SI joints, there were no findings in 42.5 % of symptomatic patients. Because of this, the authors concluded limited diagnostic value for CT scan in sacroiliac joint disease [34]. Therefore, the role of CT scan in pelvic instability has yet to be determined [1]. Major et al. documented SI lesions (erosions, sclerosis, osteophytic change) in four of nine patients with osteitis pubis in addition to increased uptake on bone scan in two patients with normal radiographic SI joints [35]. They highlight the "ring structure" and that increased stress in the anterior of the pelvis inherently places increased stress on the back. MRI has been examined by Wurdinger et al. who examined 19 postpartum patients, 6 of whom reported debilitating pelvic pain and compared them to 11 healthy volunteers [36]. No differences in pubic gap were found between those patients with and without symptoms postpartum. Likewise, pubic symphyseal edema was frequently found in those patients who had just delivered, regardless of symptoms. In addition, edema within the pubic symphysis has been shown in asymptomatic controls in studies evaluating osteitis pubis, questioning the role of MRI for diagnosis of this condition [6, 37, 38]. Bjorklund et al. examined the width of the pubic symphysis during pregnancy (at two time points prior to delivery and then at one follow-up at 5 months) utilizing ultrasound [39]. Despite a 49 % reported incidence of pelvic pain during pregnancy, no correlation could be found with either width or vertical displacement of the symphysis at follow-up in those patients with persistent pelvic pain following delivery [39]. Conversely, Scriven et al. performed ultrasound on nine women who complained of pubic symphysis pain following delivery and compared this group to 42 women who did not complain of pain [40]. Average symphyseal gap in symptomatic women was 20 mm vs 4.5 mm in the asymptomatic cohort. However, as with the CT and MRI findings, while imaging may help aide the diagnosis, the clinician should not rely on imaging to confirm the diagnosis which stems mainly from history and clinical exam.

In the patient with pelvic instability, whether from osteitis pubis, postpartum, or otherwise, treatment initially consists of conservative measures [41]. Conservative treatment consists of physical therapy focusing on pelvic floor and abdominal/core strengthening [1]. Anti-inflammatories are used routinely as appropriate. If the patient is postpartum and is still nursing, efforts are made to cease breast-feeding to eliminate the possible effects of birth hormones on pelvic relaxation since some have postulated that hormones are a potential contributing factor for continued pain and instability. While there is some data to support the claim that higher relaxin levels correlate with pubic symphyseal pain [1, 42], other studies have found minimal correlation with this specific hormone [43]. The use of a pelvic binder may be used for support of the pelvic ring [1, 41], although it does not appear to ultimately change the natural history and eventual outcome. If improvement with conservative measures occurs, continued conservative management is appropriate.

Corticosteroids and local anesthetic injections may also be used for pelvic instability. Injections can be used anteriorly at the pubic symphysis and can be particularly helpful in the case of associated SI joint dysfunction when a diagnosis is in question [12, 44–46]. Injections may not only provide therapeutic pain relief (albeit potentially short term); they can also be diagnostic for diagnosing SI joint pain as previously mentioned. Indeed, many include injections and their subsequent response within the treatment algorithm leading up to potential surgery, requiring multiple injections with benefit prior to offering a surgical treatment option [47]. If >75 % reduction in pain is observed within the first hour from a local anesthetic injection, and pain relief for greater than a week ensues after corticosteroid injection, the diagnosis of SI joint pain is highly probable. Therefore, a recommendation of three consecutive injections with benefit from each injection is recommended prior to surgical treatment to confirm the diagnosis. A CT-guided injection is preferable to assure accurate placement within the joint. If no improvement is noted after injection, surgical intervention is unlikely to improve the patient's condition.

Attempting to predict who will improve and who will require surgical intervention in the setting of pelvic instability (either from postpartum or from trauma, joint dysfunction, etc.) continues to be difficult. Good functional outcomes and resolution of the diastasis with conservative measures has been reported in patients with up to 4–6 cm of pubic diastasis noted at the time of delivery [31, 32]. Figure 1 demonstrates a patient who, despite marked instability on flamingo views and widening of the pubic symphysis after delivery, was successfully managed with physical therapy and conservative measures. Indeed, the vast majority of women suffering from pelvic instability secondary to pregnancy will improve within the first 4–6 weeks postpartum [1, 18, 31]. Elden et al. examined postpartum pain and divided women into a prospective trial comparing standard treatment, acupuncture, and pelvic exercises/physical therapy. At 12 weeks postpartum, no differences in pain scores/diaries were seen between the groups, although the patients with daily pelvic exercises had less provocative pain-producing tests [48]. Albert et al. noted the most dramatic reduction in pain was seen at the 3-month follow-up visit, after which improvement was limited in all patients [18].

Surgery should be considered only when conservative treatment fails. A trial of nonoperative therapy should be attempted for at least 3 months in the initial phase of injury (if seen within 6 months of initial symptoms) including therapy, injections, and anti-inflammatories. However, after 4–6 months from injury or onset of symptoms, less significant improvements with conservative therapy are typically seen (especially with postpartum instability) [18, 30], and a discussion with the patient as to continued conservative vs surgical management is appropriate. Clinical history and physical exam are key for surgical planning. If the patient has pain subjectively in the anterior pelvis/groin as well as posteriorly (right or left SI joint), then consideration to anterior pelvic stabilization as well as posterior SI joint stabilization should be given. Flamingo views are routinely used for assessment and documentation of hemi-pelvic instability, with focus on differential hemi-pelvic motion as opposed to exact numerical measurements of that motion. The one exception to the above "watchful waiting" is the case of acute traumatic pelvic disruption following childbirth. In this case, many authors have suggested this pelvic injury sustained during parturition should be considered along the same lines as traumatic pelvic disruption and classified within the spectrum of open book pelvic injuries [49]. Figure 2 shows a patient without pain prior to delivery. Given the wide separation anteriorly as well as disruption of bilateral SI joints (essentially an anterior-posterior compression type II injury), surgical intervention was undertaken with good early results. Table 1 lists potential surgical indications.

Surgical treatment of pelvic instability and osteitis pubis is limited to case series alone and can be divided into anterior and posterior fixation strategies. Anteriorly, reports of surgery including curettage of the symphysis, polypropylene mesh, and stabilization with or without bone grafting have been reported [26, 50]. Choi et al.

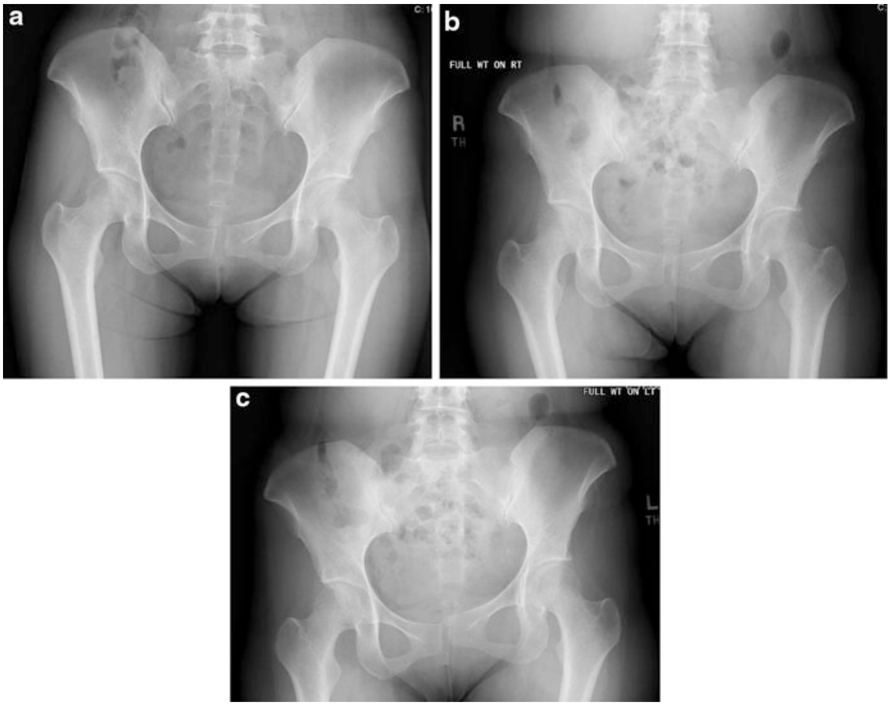


Fig. 1 "Flamingo views" can be beneficial as this is a dynamic maneuver demonstrating potential instability, re-creating a vertical shear-type moment on the pubic symphysis and SI joint. The definition of instability is debatable with 2 mm of vertical translation or 7 mm of widening being abnormal by most reports [2, 19, 52]. Garras et al. found statistically significant differences in multiparous patients when compared to men and nulliparous patients on flamingo views, with an average of 3.1 mm of translation vs 1.4 and 1.6 mm, respectively [28]. Siegel et al. examined the use of flamingo views in a series of 38 patients referred for "pelvic pain" and history consistent with instability [27]. They defined abnormal motion at the symphysis with flamingo views as >5 mm. In total, 66 % of patients had more than 5 mm of motion. Perhaps more importantly is the fact that supine or two-legged stance films were unable to pick up this instability, enhancing the potential benefit to dynamic radiographs in aiding the diagnosis. (**a**–**c**) demonstrates an AP pelvis followed by standing on the left and then the right leg with the shift in the pubic symphysis. Despite this instability, the patient was managed conservatively and did well from a clinical standpoint

identified six case series of surgical intervention for osteitis pubis [4]. A total of 25 athletes have been reported as treated with simple curettage of the pubic symphysis [4]. Of those patients, 72 % were able to return to sport at an average of 5.6 months [4]. Radic et al. examined the role of pubic symphysis curettage in 23 patients who had failed conservative therapy [51]. While pain scores did not statistically differ pre- and postoperatively, 61 % of patients were able to return to full sporting activity. However, 26 % of patients had recurrence of symptoms which resolved with rest and therapy in a range of 8–18 months postoperatively. At final follow-up, 30 % of patients following the procedure were unable to return to their previous level of sporting activity. Surgical stabilization of the anterior pelvic ring with or without bone grafting has been reported in two series with eight patients [4]. Return to sport was noted an average of 6.6 months in those eight patients. Williams et al. treated seven rugby players with osteitis pubis over the course of 12 years who were recalcitrant to conservative therapy [52]. Surgery consisted of pubic symphysis resection, tricortical iliac crest autograft, and plate fixation. Return to play ranged from 5 to 9 months and all patients reported their pain had disappeared and no recurrences were noted at an average of 64 months postoperatively. No major complications were reported. Wedge resection alone of the

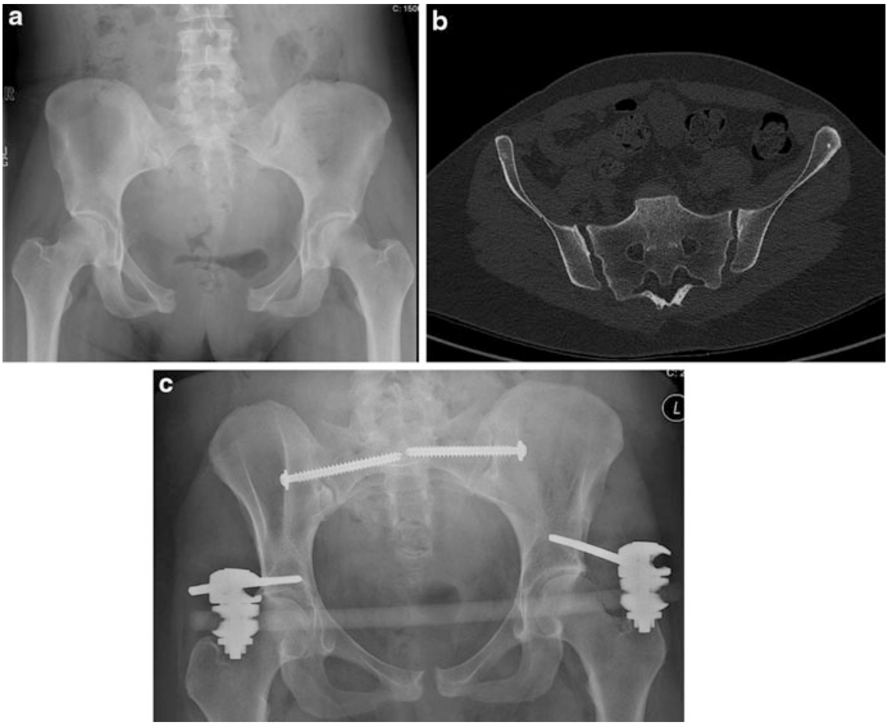


Fig. 2 (**a**) AP pelvis of a patient who present 10 days after vaginal delivery and 8 cm pubic diastasis with bilateral SI joint widening. (**b**) CT scan of the same patient demonstrating widening of the bilateral SI joints. (**c**) Postoperative AP after placement of external fixator and bilateral SI screws. Given the time from delivery, an open anterior approach was contraindicated given the potential for significant bleeding secondary to pregnancy changes

Table 1 Surgical considerations for pelvic instability

1. Failure of conservative therapy (including physical therapy, corticosteroid/analgesic injection, cessation of lactation if postpartum pain, etc.) >3 months
2. >6 months of pain since onset of symptoms, especially postpartum, without significant improvement along with radiographic instability
3. Radiographic instability on flamingo views with a clinical history of anterior pubic pain/groin pain +/− posterior pain
4. Acute, traumatic postpartum injury (i.e., APC II pelvic ring injury)
5. Response to therapeutic/diagnostic corticosteroid/analgesic injection to the pubic symphysis or SI joint with resolution of symptoms (typically series of three injections)

pubic symphysis has been advocated by some [26, 50]. Grace et al. identified 10 patients with recalcitrant osteitis pubis in which wedge resection of the pubic symphysis was undertaken. While all 10 patients were able to return to their previous level of activities and all reported subjective decreases in pain in the short term, three patients continued to complain of groin pain and one patient had instability at the SI joint following their anterior Resection [26]. It should also be noted there have been reports of posterior instability following anterior wedge resection of the pelvis, resulting in arthrodesis of the SI joints [19]. While the use of bone graft with decortication of the pubic symphysis has been reported with good clinical outcomes in some series [52], it should be noted that series report that up to 30 % of patients may have evidence of hardware loosening with/without hardware failure [29]. Therefore, some authors advocate pubic symphyseal plate fixation with compression alone, finding no benefit or need of formal fusion [19, 53]. Rommens presented three patients in which conservative therapy failed following birth trauma, one of which included posterior instability/injury diagnosed on CT scan [49]. In all patients, anterior plating (with one patient receiving sacral bars) lead to resolution of pain and return to function [49]. In the specific case of osteitis pubis, there is some data to suggest that elite athletes operatively managed fair better and return to sport quicker than those treated conservatively [37]. In Paajanen et al.'s examination of eight patients treated operatively vs eight patients treated conservatively, all eight surgically treated patients had returned to their elite level of athletic competition at 1-year follow-up compared with only 50 % of those treated conservatively. In a separate evaluation, polypropylene mesh was placed in five patients with recalcitrant osteitis pubis [54]. All five patients were pain-free at one month and had returned to full activity by 1 year.

Numerous techniques for posterior fixation/fusion have been described. Buchowski et al. examined 20 patients in which a formal SI joint fusion via plating was performed through a Smith-Peterson approach [47]. Improvement in functional outcomes occurred in all patients after surgery. However, three nonunions occurred along with two deep infections, indicating the procedure is not without morbidity. Kibsgard et al. reported on open SI joint fusions (unilateral, bilateral, or with anterior plating) in which 84 % of patients had an underlying diagnosis of SI joint pain either secondary to pregnancy or "idiopathic" [55]. They noted no significant difference in functional outcomes between surgical and nonsurgical management with up to 23-year follow-up [55]. More importantly, long-term outcomes trended toward outcomes seen at 1-year follow-up, highlighting that recovery may progress for up to 1 year post surgery with little improvement after that time. Some authors have suggested percutaneous screw fixation alone is sufficient to limit motion as opposed to formal fusion with good clinical results [23, 53]. Van Zwienen et al. described their technique of anterior plating (either with or without wedge iliac crest grafting) along with posterior percutaneous screw placement in a cohort of 58 patients with postpartum pelvic pain [23]. In their series, statistical improvement in functional outcome was found at 12 and 24 months. Improvements from 20 wheelchair-bound and eight bed-bound patients fell to four in each group, respectively. However, 30 % of patients improved less than ten points in the Majeed score. Recently, hollow screws filled with bone graft have been utilized in a percutaneous fashion to treat SI joint pathology, including pelvic dysfunction and instability [56–58]. Improvements were seen in 87 % of patients utilizing validated outcome questionnaires with minimal complications [56]. In the patient with acute birth trauma and pelvic ring disruption, surgical decision-making should follow that of pelvic ring trauma. As these injuries are almost universally open book (anterior-posterior compression injuries), fixation of the anterior pelvis with or without sacral fixation is warranted. External fixation is typically reserved for patients who present immediately postpartum as the anterior approach/Pfannenstiel incision can cause significant bleeding secondary to venous engorgement within the space of Retzius immediately postpartum.

Surgical Technique

The treatment of both osteitis pubis and pelvic instability involves, essentially, the limitation of motion/movement at the pubic symphysis and the SI joint to allow healing and scarring and prevent pathologic motion. As such, both conditions are surgically treated similar.

The patient is placed supine on a radiolucent table to allow for ease of intraoperative fluoroscopy. A stack of towels or sheets is placed under the lumbar spine to slightly elevate the patient off the bed to allow increased access to the posterior buttock region. If percutaneous SI screw placement is planned, routine use of non-sterile neuromonitoring is recommended. A Foley catheter is always placed to deflate and protect the bladder. Preoperative antibiotics are administered. The abdomen is prepped cranially to the nipple line, caudally to just above the base of the penis in males or above the labia in females, and as posterior on the buttock as can be safely obtained, as can be seen in Fig. 3. A standard Pfannenstiel incision is then made approximately one fingerbreadth above the pubic symphysis centered

Fig. 3 Standard prep and drape for an anterior approach to the pubic symphysis as well as percutaneous screw placement of the SI joints. Feet are located to the right, the head to the left

on midline. Dissection is carried down to the rectus abdominis musculature and the linea alba. Care is taken to not stray too far lateral in the incision for fear of injuring either the inferior epigastric vessels or round ligament/spermatic cord. Once midline is identified, a split is made within the rectus abdominis musculature vertically and blunt dissection is taken to the space of Retzius. Often, significant scarring/adhesions are noted along the posterior aspect of the pubic symphysis from chronic, abnormal motion, and care should be taken to release/protect the bladder during dissection. A malleable retractor with wet sponge is placed to protect the bladder once mobilized. Dissection is carried along the superior border of the pubic symphysis to the pectineal eminence. Anteriorly, the rectus abdominis may be released to gain visualization if necessary. Figure 4 demonstrates the radiograph of a patient with osteitis pubis along with the initial dissection down to the pubic symphysis, looking onto the superior aspect of the pubic symphysis. Often, osteophytes can be identified (Fig. 5) and gross motion appreciated at the pubic symphysis without taking down any ligamentous structures. In addition, gross motion may be appreciated. The pubic symphysis is cleaned of debris/scar tissue followed by reduction with a large Weber clamp placed either within the obturator foramen or just inferior to the pubic tubercle. Care should be taken when placing this clamp as bone quality of the patient may cause clamp cutout if not careful. If formal fusion is deemed necessary, tricortical iliac crest graft harvest may be performed in the standard fashion for structural support. This may be either placed into the pubic symphysis or inlaid along the superior aspect with fixation placed over the top [52]. Figure 5 demonstrates an example of wedge resection of the pubic symphysis followed by placement of graft material. Once the pubic symphysis is reduced and compressed, either a 4- or 6-hole 3.5 mm reconstruction plate or pre-contoured pubic symphysis plate is chosen and placed (Fig. 4). Dual plating may be performed if necessary for (a) poor bone quality or (b) to aide and hold reduction in cases of chronic instability/deformity. Intraoperative fluoroscopy can confirm both reduction of the symphysis and plate/screw placement. The Foley catheter is checked for any signs of blood and to assure that urine output has occurred since closing down/reducing the pelvis to confirm no bladder injury or incarceration. The wound is lavaged and a deep drain is placed within the space of Retzius. The rectus abdominis muscle layer is closed followed by the skin.

The use of external fixation may be considered for anterior stabilization in certain cases. Given the reliability and track record of anterior plate fixation, external fixation is now primarily used in the setting of acute postpartum trauma in which a Pfannenstiel incision could result in increased risk of bleeding secondary to venous congestion within the space of Retzius. If an external fixator is placed for this indication, it is typically removed within 4–6 weeks after close radiographic follow-up of no changes to the overall pelvic ring.

If the patient requires posterior pelvic ring stabilization as deemed necessary by preoperative evaluation, the surgery continues with placement of SI joint screws. This can only be performed if adequate visualization of bone landmarks can be assessed on AP, inlet, outlet, and lateral radiographs intraoperatively and is often checked prior to prep and drape not only to assure visualization but also to mark out these angles to save time intraoperatively (inlet/outlet study). This technique should not be undertaken by

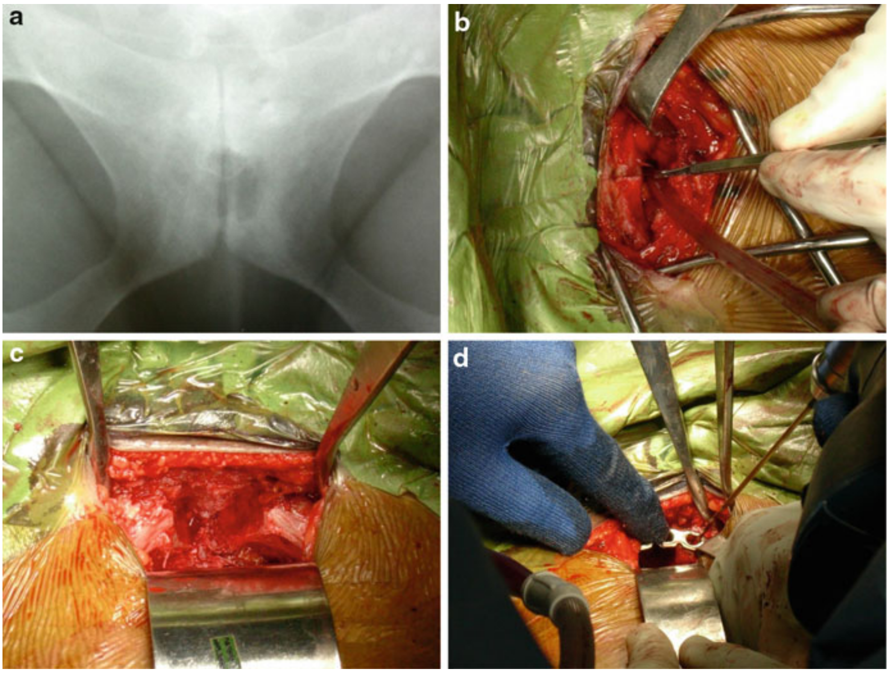


Fig. 4 (**a**) Radiograph of a patient with classic signs of osteitis pubis. (**b**) Dissection down to the superior and anterior border of the pubic symphysis. The patient's feet are to the left of the image and the head to the right. (**c**) View of the pubic symphysis after wedge resection. In this case, the patient was bone grafted with iliac crest. Note the malleable retractor posterior to the pubic symphysis protecting the bladder. (**d**) Placement of 3.5 mm reconstruction plate with drilling

those who have not been trained in placement of percutaneous SI joint screws. Preoperative planning and review of the patient radiographs and CT scan are critical to assess for safety of screw placement and corridors [59–61]. S1 or S2 screws may be used to "lock" the SI joint. These screws are placed as a true "SI joint" screw (posterior-to-anterior and often caudal-to-cranial direction as dictated by preoperative planning) as opposed to a "straight across" sacral screw. Often, two screws are used to help control rotation about the SI joint if patient anatomy allows and is deemed safe. Guide pins are placed utilizing the inlet (used for assessment of anterior-posterior pin placement), outlet (used for assessment of cranial/caudal placement in relation to the sacrum and the neuroforamen), and lateral (used to confirm pin placement within the body and no cortical perforation anteriorly) projections. The pin is then measured with a pre-calibrated measuring depth gauge. A cannulated drill is then used to drill over the pin, and the measured screw is then placed under fluoroscopy and confirmed to be in adequate position in all planes. The wounds are then closed in standard fashion. Figure 6 demonstrates an example of anterior plate fixation followed by percutaneous posterior pelvic fixation. Two screws were used posteriorly to help control rotation of the SI joint. If a formal fusion of the SI joint is deemed necessary, the patient is positioned similar to the above. The lateral window of the ilioinguinal approach is undertaken with skin incision following the level of the iliac crest. Dissection is carried down to the intermuscular and sharply defined plane between the gluteus maximus muscle and external abdominal oblique muscle. With use of knife or electrocautery, the external oblique and abdominal muscles are taken off the iliac crest from the level of the anterior superior iliac spine (ASIS) working posteriorly. Incision distal to the ASIS will help relax

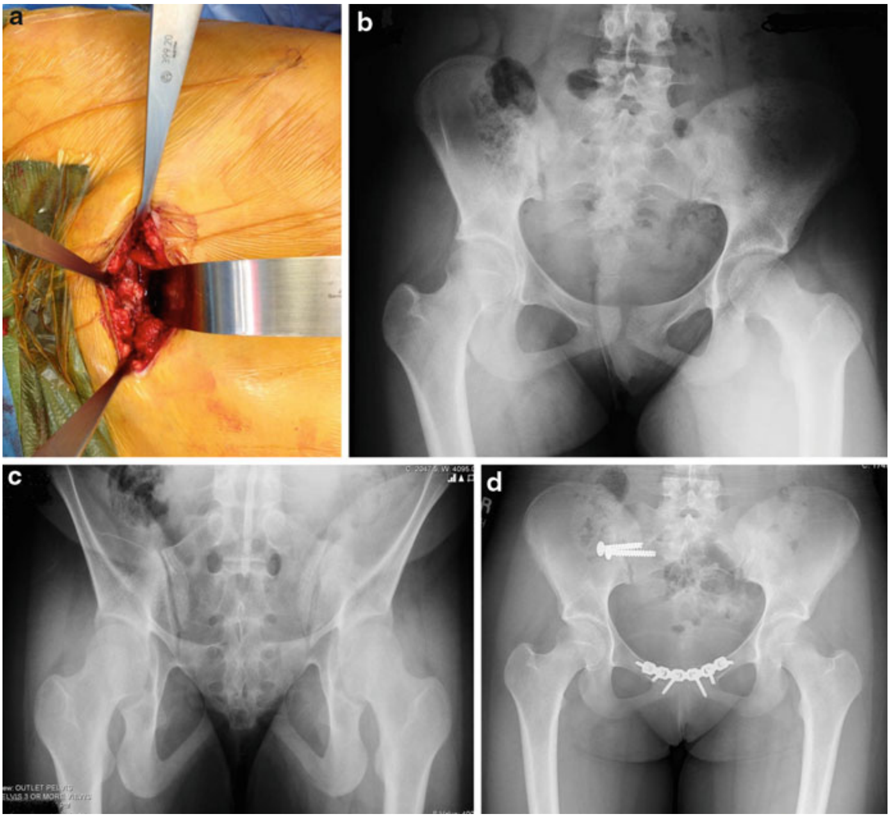


Fig. 5 Intraoperative photo of a 14-year-old with longstanding pelvic pain and instability after being hit into the perineum from a teeter-totter. Pain was located both anteriorly within the groin and posteriorly with feelings of "moving." (**a**) Gross motion was identifiable at the pubic symphysis along with osteophytes about the superior pubic ramus. (**b**) Due to preoperative planning, the patient's sacrum was quite dysmorphic on both the AP and outlet view (**b**, **c**) and would not safely allow screw passage into either S1 or S2. Smaller screws were placed across the SI joint along with anterior plate fixation (**d**) with resolution of the patient's feelings of instability and pelvic discomfort at latest follow-up

skin tension and allow better access to the SI joint. The iliacus muscle is then easily dissected bluntly from the inner table/iliac fossa of the acetabulum and packed with lap sponges. Bone wax should be readily available for any bone bleeders of significance. Dissection is carried back to the SI joint, and retractors can be placed medial to the joint into the sacrum. Care should be taken to not travel too medial onto the sacrum as the L5 nerve root lies within 1–2 cm of the SI joint. Curettage and debridement of the SI joint is then undertaken and bone graft placed. Typically, two 3.5 mm recon plates, placed at 90° to one another, are then used to secure the iliac wing to the sacrum. This can be further stabilized by placement of an SI screw as described above. Figure 7 demonstrates an example of formal SI fusion with this technique.

The debate for formal fusion of either the anterior or posterior pelvis is yet unresolved. Use of bone graft with decortication of the pubic symphysis has been reported with good clinical outcomes in some series [29, 52]. While some reports demonstrate no problems with nonunion or hardware failure [52], others demonstrate up to 30 % incidence of hardware loosening with/without hardware failure [29]. Attempts at formal fusion of either the anterior or posterior joints has largely been abandoned as it is, in the author's experience, extremely difficult to achieve. In addition, as

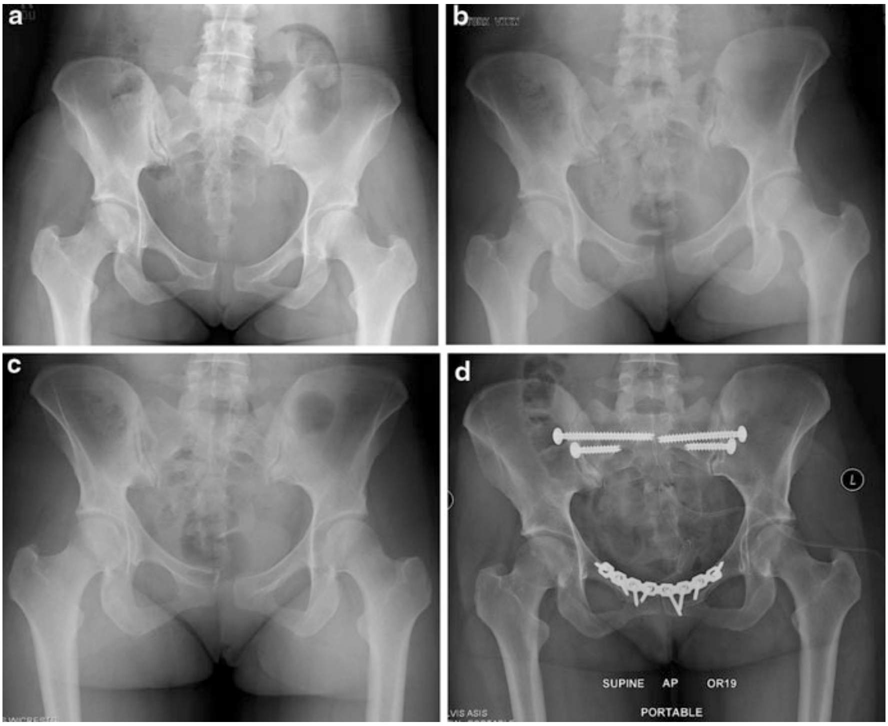


Fig. 6 A patient with 5 years of pelvic pain (both anteriorly and bilateral posterior) and instability postpartum. AP pelvis and flamingo views demonstrate instability (**a**–**c**). In addition, patient complained of significant back pain bilaterally located at the SI joints. (**d**) Postoperatively, patient's pain was greatly improved after anterior plating and placement of SI joint screws. Two screws on each side were used to help control rotation about the SI joint

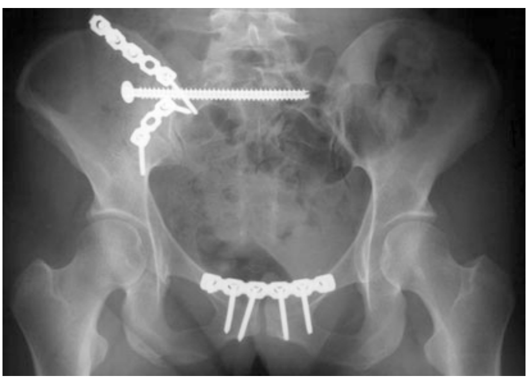


Fig. 7 Example of final pelvic radiograph of formal right-sided SI joint fusion with two 3.5 reconstruction plates with SI screw placement. Patient had an excellent functional recovery

the goal of surgery is to limit motion anteriorly and posteriorly, this is accomplished by plate and screw fixation without the larger surgery needed for formal fusion, negating the risk of nonunion which is not insignificant. This is highlighted by previous reports with symphysiodesis resulting in a 27 % nonunion and even tricortical wedge bone grafting resulting in as high as an 8 % nonunion [23]. In addition, the large and relatively morbid procedure to obtain bony fusion of an SI joint carries little clinical benefit when compared to percutaneous fixation when the goal is to prevent further "abnormal" or pathologic motion which can be obtained with screw placement. Thus, percutaneous fixation of the SI joint and anterior plate fixation of the pubic symphysis decreases motion both in the front and the back, alleviating the patient's pain and allowing healing to occur. Patients are partial weight bearing to the affected extremity if unilateral and "four-point gait" if bilateral for approximately 8 weeks postoperatively. Pending radiographs and clinical exam, progressive partial weight bearing is then instituted to full weight bearing by 12 weeks.

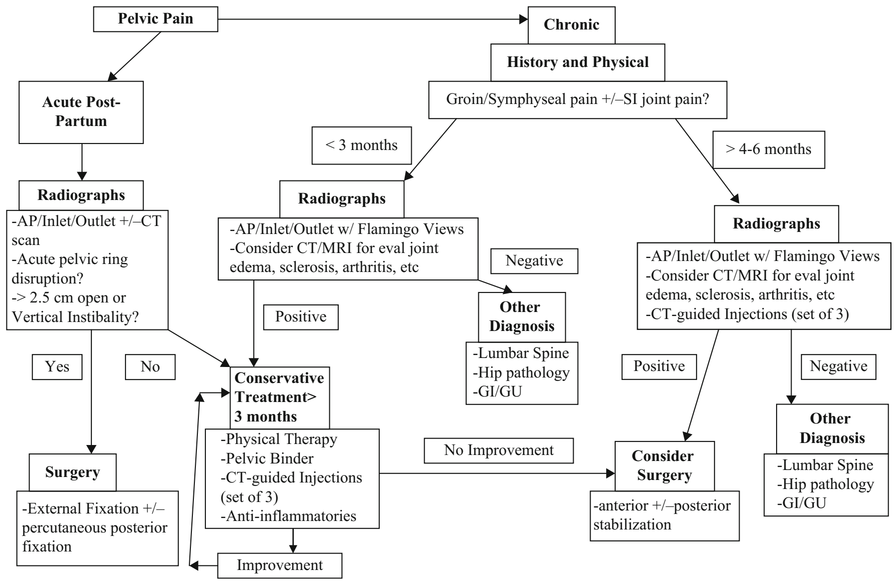


Fig. 8 Proposed treatment algorithm for a patient presenting with potential pelvic instability

Summary

The vast majority of patients with osteitis pubis and pelvic instability can be treated conservatively, and surgery should be used sparingly. Figure 8 provides a proposed flow chart for evaluation and treatment of pelvic instability, as partially described by Weil et al. [53]. If surgical stabilization is planned, an examination of both the anterior and posterior pelvic ring is paramount with surgical intervention focused on addressing both anterior and posterior instability/pain as necessary. Numerous methods have been described to treat both osteitis pubis and pelvic instability with little concrete, absolute data currently available to guide the clinician as to the best surgical treatment strategy. However, with patient participation and understanding of the disease process and which options are available with realistic expectations, good functional outcomes can be obtained.

References

1. Vleeming A, Albert HB, Ostgaard HC, Sturesson B, Stuge B. European guidelines for the diagnosis and treatment of pelvic girdle pain. Eur Spine J. 2008;17(6):794–819.
2. Beatty T. Osteitis pubis in athletes. Curr Sports Med Rep. 2012;11(2):96–8.
3. Bradshaw CJ, Bundy M, Falvey E. The diagnosis of longstanding groin pain: a prospective clinical cohort study. Br J Sports Med. 2008;42(10):851–4.
4. Choi H, McCartney M, Best TM. Treatment of osteitis pubis and osteomyelitis of the pubic symphysis in athletes: a systematic review. Br J Sports Med. 2011;45(1):57–64.
5. Fricker PA, Taunton JE, Ammann W. Osteitis pubis in athletes. Infection, inflammation or injury? Sports Med. 1991;12(4):266–79.
6. Lovell G, Galloway H, Hopkins W, Harvey A. Osteitis pubis and assessment of bone marrow edema at the pubic symphysis with MRI in an elite junior male soccer squad. Clin J Sport Med. 2006;16(2):117–22.
7. Batt ME, McShane JM, Dillingham MF. Osteitis pubis in collegiate football players. Med Sci Sports Exerc. 1995;27(5):629–33.
8. Rodriguez C, Miguel A, Lima H, Heinrichs K. Osteitis pubis syndrome in the professional soccer athlete: a case report. J Athl Train. 2001;36(4):437–40.
9. Omar IM, Zoga AC, Kavanagh EC, Koulouris G, Bergin D, Gopez AG, Morrison WB, Meyers WC. Athletic pubalgia and "sports hernia": optimal MR imaging technique and findings. Radiographics. 2008;28(5):1415–38.
10. Macintyre J, Johson C, Schroeder EL. Groin pain in athletes. Curr Sports Med Rep. 2006;5(6):293–9. Review.
11. Cunningham PM, Brennan D, O'Connell M, MacMahon P, O'Neill P, Eustace S. Patterns of bone and soft-tissue injury at the symphysis pubis in soccer players: observations at MRI. AJR Am J Roentgenol. 2007;188(3):W291–6.

12. Wollin M, Lovell G. Osteitis pubis in four young football players: a case series demonstrating successful rehabilitation. Phys Ther Sport. 2006;7:153–60.
13. Larsen EC, Wilken-Jensen C, Hansen A, Jensen DV, Johansen S, Minck H, Wormslev M, Davidsen M, Hansen TM. Symptom-giving pelvic girdle relaxation in pregnancy. I: Prevalence and risk factors. Acta Obstet Gynecol Scand. 1999;78(2):105–10.
14. Reis RA, Baer JL, Arens RA, Stewart E. Traumatic separation of the symphysis pubis during spontaneous labor: with a clinical and x-ray study of the normal symphysis pubis during pregnancy and the puerperium. Surg Gynecol Obstet. 1932;55:336–54.
15. Taylor RN, Sonson RD. Separation of the pubic symphysis. An underrecognized peripartum complication. J Reprod Med. 1986;31:203–6.
16. Snow RE, Neubert AG. Peripartum pubic symphysis separation: a case series and review of the literature. Obstet Gynecol Surv. 1997;52(7):438–43. Review.
17. Ostgaard HC, Andersson GB, Karlsson K. Prevalence of back pain in pregnancy. Spine (Phila Pa 1976). 1991;16(5):549–52.
18. Albert H, Godskesen M, Westergaard J. Prognosis in four syndromes of pregnancy-related pelvic pain. Acta Obstet Gynecol Scand. 2001;80(6):505–10.
19. Moore Jr RS, Stover MD, Matta JM. Late posterior instability of the pelvis after resection of the symphysis pubis for the treatment of osteitis pubis. J Bone Joint Surg Am. 1998;80(7):1043–8.
20. Wu WH, Meijer OG, Uegaki K, Mens JM, van Djeen JH, Wuisman PI, Ostgaard HC. Pregnancy-related pelvic girdle pain (PPP), I: Terminology, clinical presentation, and prevalence. Eur Spine J. 2004;13(7): 575–89. Epub 2004 Aug 27.
21. Albert HB, Godskesen M, Korsholm L, Westergaard JG. Risk factors in developing pregnancy-related pelvic girdle pain. Acta Obstet Gynecol Scand. 2006;85(5): 539–44.
22. Hansen A, Jensen DV, Wormslev M, Minck H, Johansen S, Larsen EC, Wilken-Jensen C, Davidsen M, Hansen TM. Symptom-giving pelvic girdle relaxation in pregnancy. II: Symptoms and clinical signs. Acta Obstet Gynecol Scand. 1999;78(2):111–5.
23. van Zwienen CM, van den Bosch EW, Snijders CJ, van Vugt AB. Triple pelvic ring fixation in patients with severe pregnancy-related low back and pelvic pain. Spine (Phila Pa 1976). 2004;29(4):478–84.
24. Owens K, Pearson A, Mason G. Symphysis pubis dysfunction–a cause of significant obstetric morbidity. Eur J Obstet Gynecol Reprod Biol. 2002;105(2): 143–6.
25. Damen L, Buyruk HM, Güler-Uysal F, Lotgering FK, Snijders CJ, Stam HJ. The prognostic value of asymmetric laxity of the sacroiliac joints in pregnancy-related pelvic pain. Spine (Phila Pa 1976). 2002;27 (24):2820–4.
26. Coventry MB, Mitchell WC. Osteitis pubis: observations based on a study of 45 patients. JAMA. 1961;178:898–905.
27. Siegel J, Templeman DC, Tornetta 3rd P. Single-leg-stance radiographs in the diagnosis of pelvic instability. J Bone Joint Surg Am. 2008;90(10):2119–25.
28. Garras DN, Carothers JT, Olson SA. Single-leg-stance (flamingo) radiographs to assess pelvic instability: how much motion is normal? J Bone Joint Surg Am. 2008;90(10):2114–8.
29. Najibi S, Tannast M, Klenck RE, Matta JM. Internal fixation of symphyseal disruption resulting from childbirth. J Orthop Trauma. 2010;24(12):732–9.
30. Hagen R. Pelvic girdle relaxation from an orthopaedic point of view. Acta Orthop Scand. 1974;45(4):550–63.
31. Kharrazi FD, Rodgers WB, Kennedy JG, Lhowe DW. Parturition-induced pelvic dislocation: a report of four cases. J Orthop Trauma. 1997;11(4):277–81.
32. Hou Z, Riehl JT, Smith WR, Strohecker KA, Maloney PJ. Severe postpartum disruption of the pelvic ring: report of two cases and review of the literature. Patient Saf Surg. 2011;5(1):2.
33. Garagiola DM, Tarver RD, Gibson L, Rogers RE, Wass JL. Anatomic changes in the pelvis after uncomplicated vaginal delivery: a CT study on 14 women. AJR Am J Roentgenol. 1989;153(6):1239–41.
34. Elgafy H, Semaan HB, Ebraheim NA, Coombs RJ. Computed tomography findings in patients with sacroiliac pain. Clin Orthop Relat Res. 2001;382:112–8.
35. Major NM, Helms CA. Pelvic stress injuries: the relationship between osteitis pubis (symphysis pubis stress injury) and sacroiliac abnormalities in athletes. Skeletal Radiol. 1997;26(12):711–7.
36. Wurdinger S, Humbsch K, Reichenbach JR, Peiker G, Seewald HJ, Kaiser WA. MRI of the pelvic ring joints postpartum: normal and pathological findings. J Magn Reson Imaging. 2002;15(3):324–9.
37. Paajanen H, Hermunen H, Karonen J. Pubic magnetic resonance imaging findings in surgically and conservatively treated athletes with osteitis pubis compared to asymptomatic athletes during heavy training. Am J Sports Med. 2008;36(1):117–21.
38. Verrall GM, Slavotinek JP, Fon GT. Incidence of pubic bone marrow oedema in Australian rules football players: relation to groin pain. Br J Sports Med. 2001;35(1):28–33.
39. Björklund K, Nordström ML, Bergström S. Sonographic assessment of symphyseal joint distention during pregnancy and post partum with special reference to pelvic pain. Acta Obstet Gynecol Scand. 1999;78(2):125–30.
40. Scriven MW, Jones DA, McKnight L. The importance of pubic pain following childbirth: a clinical and ultrasonographic study of diastasis of the pubic symphysis. J R Soc Med. 1995;88(1):28–30.
41. Luger EJ, Arbel R, Dekel S. Traumatic separation of the symphysis pubis during pregnancy: a case report. J Trauma. 1995;38(2):255–6.
42. Kristiansson P, Svardsudd K, von Schoultz B. Serum relaxin, symphyseal pain, and back pain during pregnancy. Am J Obstet Gynecol. 1996;175:1342–7.

43. Bjorklund K, Bergstrom S, Nordstrom M-L. Symphyseal distension in relation to serum relaxin levels and pelvic pain in pregnancy. Acta Obstet Gynecol Scand 2000;79:269–275.
44. Katz V, Schofferman J, Reynolds J. The sacroiliac joint: a potential cause of pain after lumbar fusion to the sacrum. J Spinal Disord Tech. 2003;16(1):96–9.
45. Slipman CW, Patel RK, Whyte II WS, et al. Diagnosing and managing sacroiliac pain. J Musculoskelet Med. 2001;18:325–32.
46. O'Connell MJ, Powell T, McCaffrey NM, O'Connell D, Eustace SJ. Symphyseal cleft injection in the diagnosis and treatment of osteitis pubis in athletes. AJR Am J Roentgenol. 2002;179(4):955–9.
47. Buchowski JM, Kebaish KM, Sinkov V, Cohen DB, Sieber AN, Kostuik JP. Functional and radiographic outcome of sacroiliac arthrodesis for the disorders of the sacroiliac joint. Spine J. 2005;5(5):520–8.
48. Elden H, Hagberg H, Olsen MF, Ladfors L, Ostgaard HC. Regression of pelvic girdle pain after delivery: follow-up of a randomised single blind controlled trial with different treatment modalities. Acta Obstet Gynecol Scand. 2008;87(2):201–8.
49. Rommens PM. Internal fixation in postpartum symphysis pubis rupture: report of three cases. J Orthop Trauma. 1997;11(4):273–6.
50. Grace JN, Sim FH, Shives TC, Coventry MB. Wedge resection of the symphysis pubis for the treatment of osteitis pubis. J Bone Joint Surg Am. 1989;71(3):358–64.
51. Radic R, Annear P. Use of pubic symphysis curettage for treatment-resistant osteitis pubis in athletes. Am J Sports Med. 2008;36(1):122–8.
52. Williams PR, Thomas DP, Downes EM. Osteitis pubis and instability of the pubic symphysis. When nonoperative measures fail. Am J Sports Med. 2000;28(3):350–5.
53. Weil YA, Hierholzer C, Sama D, Wright C, Nousiainen MT, Kloen P, Helfet DL. Management of persistent postpartum pelvic pain. Am J Orthop (Belle Mead NJ). 2008;37(12):621–6.
54. Paajanen H, Heikkinen J, Hermunen H, Airo I. Successful treatment of osteitis pubis by using totally extraperitoneal endoscopic technique. Int J Sports Med. 2005;26(4):303–6.
55. Kibsgård TJ, Røise O, Sudmann E, Stuge B. Pelvic joint fusions in patients with chronic pelvic girdle pain: a 23-year follow-up. Eur Spine J. 2013;22(4):871–7.
56. Khurana A, Guha AR, Mohanty K, Ahuja S. Percutaneous fusion of the sacroiliac joint with hollow modular anchorage screws: clinical and radiological outcome. J Bone Joint Surg Br. 2009;91(5):627–31.
57. Wise CL, Dall BE. Minimally invasive sacroiliac arthrodesis: outcomes of a new technique. J Spinal Disord Tech. 2008;21(8):579–84.
58. Al-Khayer A, Hegarty J, Hahn D, Grevitt MP. Percutaneous sacroiliac joint arthrodesis: a novel technique. J Spinal Disord Tech. 2008;21(5):359–63.
59. Miller AN, Routt Jr ML. Variations in sacral morphology and implications for iliosacral screw fixation. J Am Acad Orthop Surg. 2012;20(1):8–16.
60. Routt Jr ML, Simonian PT, Agnew SG, Mann FA. Radiographic recognition of the sacral alar slope for optimal placement of iliosacral screws: a cadaveric and clinical study. J Orthop Trauma. 1996;10(3):171–7.
61. Gardner MJ, Morshed S, Nork SE, Ricci WM, Chip Routt Jr ML. Quantification of the upper and second sacral segment safe zones in normal and dysmorphic sacra. J Orthop Trauma. 2010;24(10):622–9.

Athletic Pubalgia

58

Jozef Murar and Patrick M. Birmingham

Contents

J. Murar (✉)
Orthopaedic Surgery Resident, Department of Orthopaedic Surgery, Northwestern University, Chicago, IL, USA
e-mail: j-murar@md.northwestern.edu

P.M. Birmingham
Orthopaedic Sports Medicine, NorthShore University Health Systems, Chicago, IL, USA
e-mail: patrickbirmingham@gmail.com

Abstract

Athletic pubalgia refers to lower abdominal and groin pain arising from an injury to the congruent fascial sheath of the rectus abdominis and the adductor complex. Its cause can be multifactorial, but is mainly an overuse injury from high-level athletic activity. It is initially treated conservatively, but if symptoms persist after 6–12 weeks, surgical intervention is an option. A number of different procedures have been described, but they all have similar return to play rates. Femoroacetabular impingement can coexist and should be ruled out, because if untreated it leads to a high failure rate.

Introduction

Athletic pubalgia, also known as sports hernia, is a syndrome of chronic lower abdominal or groin pain that can occur in both athletes and nonathletes. It is defined as weakness or tearing of the rectus abdominis insertion onto the superior pubic ramus and sometimes tearing or incompetence of the abdominal obliques and/or transversus abdominis. It has also been used, however, as an umbrella term that describes several anatomic injury patterns specifically involving the thigh and abdominal wall musculature [1]. A number of different terms, including "sports hernia," "Gilmour's" groin, osteitis pubis, "slap-shot gut," "sportsman's hernia," and adductor or rectus

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_62

strain, have been utilized to describe a wide variety of pathologies around the groin [2–5]. Although none of these terms is perfect, they all attempt to describe this poorly understood disease complex.

Moreover, chronic lower abdomen and groin pain can have several causes including but not limited to intra-articular hip joint pathology, osteitis pubis, fractures, hernias, entrapment neuropathies, pain referred from the SI joint or the spine, and nonmusculoskeletal conditions including gynecologic, urologic, and gastrointestinal etiologies [6]. Because of its broad definition and complicated anatomy in the groin and lower abdomen, correctly diagnosing and treating true athletic pubalgia can be challenging [4, 7].

There is growing recognition that groin injuries in athletes comprise a complex set of injuries to the musculature of the abdominal wall, the adductors, the hip joint, the pubic symphysis, and the sacroiliac joint that can be a source of significant disability [2, 8, 9]. A relationship between femoroacetabular impingement syndrome and athletic pubalgia has been identified as well [9–11]. The various described entities likely represent a spectrum of pathology with a common etiology. Frequent failure of nonsurgical management to predictably return athletes with groin injuries to sport has stimulated significant interest in determining the underlying etiology, pathology, and development of effective surgical management strategies [5, 12]. This chapter will discuss the current concepts in evaluating and treating athletic pubalgia including pertinent anatomy, history, patient evaluation, imaging, and treatment options.

Anatomy

The complexity of the anatomy of the hip joint, pelvis, pubic symphysis, and the associated abdominal wall necessitates careful evaluation to accurately diagnose the source of an athlete's pain. The bony pelvis has two principal functions: to transfer weight and to withstand compression forces resulting from its support of the weight [4]. It is made up of two innominate bones anteriorly and the sacrum and coccyx posteriorly. The anterior aspect of the pelvis is where the center of core injuries and athletic pubalgia occur given the multiple muscle attachments in the area as well as the pubic symphysis. The pubic symphysis is a non-synovial amphiarthrodial joint that is stabilized by the fibrocartilaginous articular disk between the pubic bones and four ligaments as well as multiple tendon attachments [13].

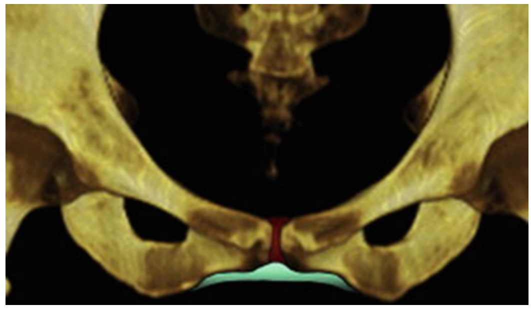


Fig. 1 Diagram of the pubic symphysis demonstrating the midline fibrocartilage disk in *red* and the anteroinferior arcuate ligament in *green*. Reproduced with permission from Springer [17]

The ligamentous attachments of the pubic symphysis include the arcuate (inferior), superior, anterior, and posterior pubic ligaments. The arcuate and the superior ligaments are the most functionally important for stability and in resisting shear forces. The arcuate ligament lines the inferior aspect of the pubic symphysis superficial to the articular disk and deep to the rectus abdominis/adductor aponeurosis (Fig. 1). The superior ligament spans the space between the pubic tubercles. The anterior ligament blends with fibers of the external oblique and rectus abdominis superficially. The deep portion of the anterior ligament attaches to the intra-articular disk. The posterior ligament is thin and poorly developed providing the least support for stability [13].

The pubic symphysis acts as a fulcrum for forces generated at the anterior pelvis (Fig. 2). Musculoaponeurotic plate attachments at the pubic symphysis are important for core stability, and coordinated contraction of the muscles that directly attach to the fulcrum produces a slight anterior tilt of the pelvis. The abdominal muscle

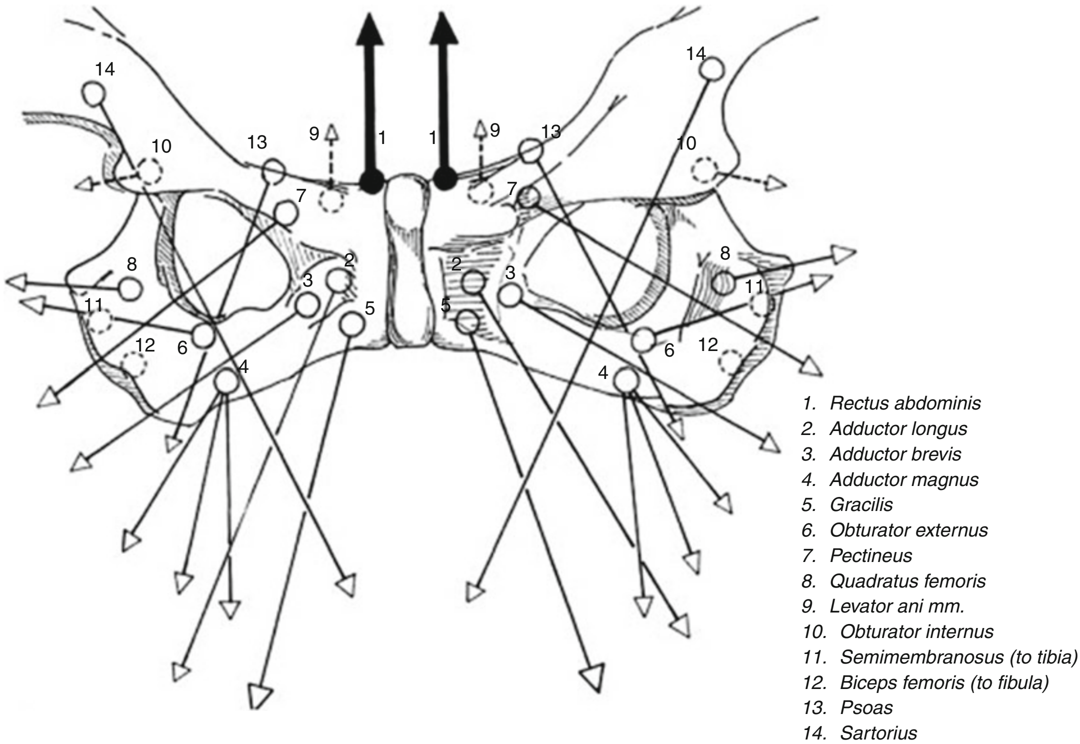


Fig. 2 Diagram of forces generated at the anterior pelvis that have to be counterbalanced by the pubic symphysis. Reproduced with permission from Springer[14]

attachments include the rectus abdominis, internal oblique, external oblique, and transversus abdominis. The medial thigh compartment attachments include the pectineus, adductor longus, adductor brevis, adductor magnus, and gracilis. Functionally, the most important attachments for anterior pelvis stabilization are where the rectus abdominis and the three adductor muscles join to the fibrocartilage plate of the pubic symphysis [10, 13, 14]. The rectus abdominis attaches to the anterior and anterior–inferior aspects of the pubic symphysis. The origin of the pectineus and adductors is confluent with the rectus insertion and is defined as the rectus abdominis/adductor aponeurosis [14].

The abdominal wall has a layered structure. From superficial to deep, the structures of the abdominal wall are skin, fascia, external oblique fascia and muscle, internal oblique fascia and muscle, transversus abdominis muscle and fascia, and the transversalis fascia. The posterior fascia is deficient in the lower 1/3 of the rectus, so descriptions of tears of the posterior sheath in this region are not accurate. Fibers from the rectus, conjoint tendon (fusion of the internal oblique and transversus abdominis fascia), and external oblique merge to form the pubic aponeurosis which is confluent with the adductor and gracilis origin. The conjoint tendon inserts anterior to the rectus abdominis on the pubis [15].

During core rotation and extension, the rectus abdominis and the adductor longus act as antagonists (Fig. 3). The rectus elevates the pelvis while the adductor depresses it. Injuring one of the components tends to cause abnormal biomechanical forces on opposing muscles and tendons leading to further injury at the aponeurosis and the tenoperiosteal attachments [16]. Detachment of the aponeurosis itself can lead to instability of the pubic symphysis, as injury to the arcuate or anterior pubic ligaments. Altering the biomechanics of the core may subsequently lead to abnormal

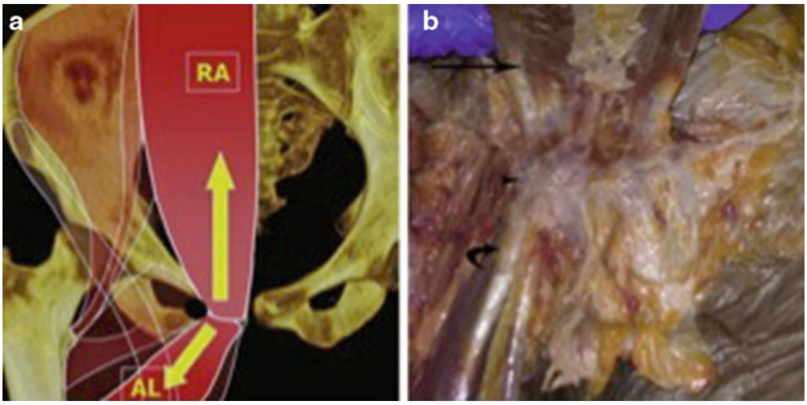


Fig. 3 (**a**) Diagram of the opposing forces of the rectus abdominis (*RA*) and adductor longus (*AL*) at the pubic tubercle. The rectus abdominis creates superoposterior tension, whereas the adductor longus creates inferoanterior tension. Disruption of either leads to altered biomechanics. The *black circle* represents the superficial inguinal ring. (**b**) Gross specimen demonstrates the rectus abdominis (*arrow*), adductor longus (*curved arrow*), and the pubic tubercle attachment of the rectus abdominis/adductor aponeurosis (*arrowhead*). Reproduced with permission from Springer [17]

forces throughout the upper and lower extremities that may ultimately lead to aggravation of hip impingement, labral tears, knee ligament injuries, and even ankle sprains [17].

Disorders with innervation of the anterior pelvis may also be a pain generator that can present as groin pain. In particular, pain may be caused by nerve entrapment of the genital branches of the ilioinguinal or genitofemoral nerves due to weakness in the posterior wall of the inguinal canal [18]. The symphysis itself is innervated by branches of the pudendal and genitofemoral nerves [13]. Other reports have suggested that the iliohypogastric or obturator nerves could also be involved [19].

It should be noted that there are differences in male versus female anatomy with regard to the pelvis and the hips. Historically, <1 % of patients with athletic pubalgia were female as reported by Meyers, but this has changed dramatically over the last two decades, and now approximately 15 % of patients with athletic pubalgia are female [20]. Differences in anatomy include a more slender and lighter pelvis in the female with fewer shifts in forces, a relatively wider subpubic angle leading to a different distribution of forces, and a relatively wider, more stable pelvis resulting in the transfer of destabilizing forces to the narrow-based lower extremities (Fig. 4) [4]. These anatomic differences are likely responsible for the difference in injury incidence in males compared to females. The forces are likely protective with regard to development of athletic pubalgia, but may also contribute to the increased incidence of anterior cruciate injuries in women [1].

History of Groin Injury

Reports of groin injuries have appeared in the medical literature as early as 1932 when Spinelli reported on pubic pain in fencers [21]. In 1966, Cabot reported on Spanish soccer players with groin pain [22]. In 1980 Gilmore recognized and surgically repaired groin disruption in a group of athletes with chronic lower abdominal and groin pain. He identified a triad of pathology including injuries to the external oblique aponeurosis and conjoint tendon, avulsion of the conjoint tendon from the pubic tubercle, and dehiscence of the conjoint tendon from the inguinal ligament [2]. This entity of groin disruption associated with groin pain in the athlete was subsequently termed Gilmore's groin. A traditional hernia was not identified in this series of patients. Gilmore later presented a larger series of patients in which he described an anatomical repair of the injured structures with a six-layer suture repair. He reported a 97 % return to sport rate with this technique [5].

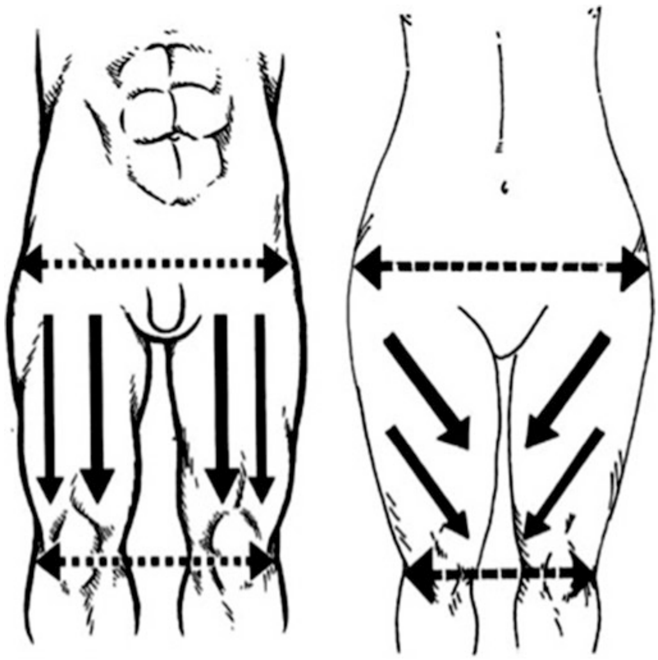


Fig. 4 Basic differences in male versus the female anatomy that relate to the pubic joint and injury. Note the differences in width between the pelvis and knees of the two genders. These differences suggest a different distribution of forces during extremes of exertion, for example, more lateral forces emanate from the female pelvis and more acutely angled forces are transmitted to female knees during landing. Reproduced with permission from Springer [4]

In 1993 the term "sports hernia" was first coined by Hackney to describe a syndrome of groin pain in athletes that had failed nonsurgical management. During surgery, he identified weakening of the transversalis fascia with separation of that fascia from the conjoint tendon, dilation of the inguinal ring, and one case of a small direct hernia. He treated all patients with a surgical repair of the posterior inguinal wall and obtained an 87 % return to sport rate in 15 athletes [12].

In 2001 Irshad et al. described the "hockey groin syndrome" in 22 National Hockey League players and found tearing of the external oblique aponeurosis and entrapment of the ilioinguinal nerve. Surgical management included mesh repair of the external oblique aponeurosis and ablation of the ilioinguinal nerve [23].

Meyers has proposed that use of the term "athletic pubalgia," and more recently "Core injury," is more appropriate for the constellation of injuries to the abdominal wall, hip flexors, adductors, and pubic symphysis than the more commonly used "sports hernia" [4]. He introduced the idea of a "pubic joint" as a complex structure consisting of the anterior pelvic ring and associated musculotendinous attachments. He proposed that the primary pathology in athletic pubalgia is an imbalance between the strong adductors and the relatively weak abdominal muscles, which are then predisposed to strain during the abdominal hyperextension/hip abduction mechanism that is commonly associated with the onset of groin pain in athletes. Based on findings at surgery, Meyers describes 17 different variants of athletic pubalgia, the most common of which are multiple tears or detachment of the anterior and anterolateral fibers of the rectus abdominis from the pubis and combined injuries to the rectus and adductors [20].

As the understanding of intra-articular hip pathology has improved, there has been increasing recognition of labral pathology and femoroacetabular impingement coexisting with athletic pubalgia [9, 10]. Larson et al. reported surgical treatment in a subset of athletes with coexistent femoroacetabular impingement and athletic pubalgia. Failure to treat both pathologies resulted in a low return to sport rate (25 % of athletic pubalgia was addressed in isolation, and 50 % of the intra-articular hip pathology was addressed in isolation). This resulted in the development of a surgical protocol to address both etiologies under the same anesthesia when athletes present with both symptomatic athletic pubalgia and intra-articular hip disorders (FAI). Using this approach, they achieved an 85–93 % return to sport rate [9].

Presentation

Groin injuries are most common in athletes who participate in sports that require repetitive twisting, pivoting, and cutting motions, as well as activities requiring frequent acceleration and deceleration. Ice hockey, soccer, and rugby have a particularly high incidence [2, 23, 24]. Up to 13 % of soccer injuries involve the groin, and in one series, 58 % of soccer players have experienced a groin injury [25].

Patients with an isolated sports hernia usually present with a dull, chronic pain in the groin. Occasionally the pain radiates to the perineum, inner thigh, or occasionally the scrotum. They generally report an insidious onset of groin pain that intensifies with athletic activity and is relieved with rest [1, 8, 26–28]. Pain is usually most aggravated during sudden acceleration, twisting and turning, pivoting, cutting, kicking, sit-ups, coughing, or sneezing [8, 24, 27, 28]. Meyers et al. found that 92 % of their athletes had minimal to no pain at rest and 100 % reported pain with exertion [8]. Night pain that awakens the athlete from sleep and severe rest pain are atypical and should raise concern for tumors and other nonmusculoskeletal pathology.

Usually groin pain starts unilaterally, but 43 % of patients developed bilateral symptoms. Sixty-seven percent developed adductor pain after the onset of lower abdominal pain [9].

Some patients remember a specific instance when the pain began; however, others do not recall one particular event. There is some discrepancy within the literature regarding the acuity of presentation, with 6–71 % of athletes recalling an inciting event [7, 8, 29]. If an inciting event occurs, two types of pain syndrome may emerge: (1) The patient is unable to participate in sport after the first 5 min of exertion due to incapacitating pain and despite conservative care and rehabilitation remains disabled, or (2) the athlete can play through the pain, but often at less than 100 % capacity. At the conclusion of the season, the athlete may rest for a few months and anticipate resolution of the pain. Often, however, upon returning to training, they note recurrence of pubalgia symptoms [1].

Physical Examination

After a thorough history, a comprehensive physical examination is required to distinguish between intra-articular and extra-articular hip pathology. This will help dictate the next most appropriate workup with regard to imaging studies.

Assessment for athletic pubalgia should begin with palpation of the pubic symphysis, insertion of the rectus abdominis, adductor origin, external and internal obliques, transverses abdominis, pectineus, gracilis, and inguinal ring for areas of tenderness. Upon palpation, there is no detectable true inguinal hernia; however, there is typically tenderness to palpation of the conjoint tendon and sometimes the pubic tubercle (22 %), adductor longus (36 %), superficial inguinal ring, or posterior inguinal canal [8, 30, 31]. Pain can be elicited with provocative testing such as with simulated coughing, resisted sit-ups (46 %), and resisted hip adduction or Valsalva [8]. One study found that more patients had pain with resisted adduction (88 %) than pubic tenderness (22 %) [8]. Patients can also have pain with resisted hip flexion (9 %) [8, 27].

Kachingwe and Grech noted five signs and symptoms that they felt most indicative of a sports hernia. These include (1) a subjective complaint of deep groin/lower abdominal pain; (2) exacerbation of the pain with sport-specific sprinting, kicking, cutting, and/or sit-ups and relief of pain with rest; (3) palpable tenderness over the pubic ramus at the insertion of the rectus abdominis and/or conjoined tendon; (4) pain with resisted hip adduction at 0°, 45° and/or 90° of hip flexion; and (5) pain with resisted abdominal curl up [27].

It is important to rule out intra-articular hip pathology with a thorough physical exam of the hip including hip range of motion (flexion, extension, adduction, and abduction), internal and external rotation, and provocative testing to assess for femoroacetabular impingement. Typically, the anterior impingement test (flexion, adduction, and internal rotation) or FADIR test is performed which can suggest intra-articular hip pathology [32]. Palpation of the insertion of the gluteus medius and gluteus minimus, short external rotators, and trochanteric bursa should be performed as well, and a lower extremity neurologic exam and straight leg raise may be useful to rule out lumbar spine pathology.

Imaging

Radiographic Analysis

Plain radiographs are recommended for the initial evaluation of the athlete with hip or groin pain. Although they cannot specifically identify athletic pubalgia, they are excellent at visualizing and ruling out other pathologies including osteitis pubis, avulsion fractures, stress fractures, apophysitis, osteoarthritis, and femoroacetabular impingement/ dysplasia. It is important to obtain good quality, properly oriented images according to an established imaging protocol. The initial series should include an anteroposterior (AP) pelvic radiograph, a modified Dunn, or elongated neck lateral [33, 34].

The AP view may be used to evaluate the pubic symphysis for evidence of osteitis pubis, including sclerosis, fragmentation, and cyst formation within the pubic ramus, as well as symphyseal widening. When evaluating for femoroacetabular impingement, femoral head neck deformities and acetabular depth and version are assessed utilizing the alpha angle, femoral head/neck offset, neck shaft angle, lateral center edge angle (LCE), anterior center edge angle (ACE), Tonnis angle, and presence of a crossover sign. In the adolescent athlete, the AP view can be useful to identify apophyseal injuries. Occasionally, stress fractures of the femoral neck and pubic rami and sacroiliitis may be identified if they are well established [15].

Stability of the pubic symphysis can be determined on single leg stance AP views. Symphyseal widening greater than 7 mm or vertical translation greater than 2 mm on a single leg stance view suggests instability of the pubic symphysis [35].

Magnetic Resonance Imaging (MRI)

While plain x-rays and CT scans can be helpful to rule out bony abnormalities, magnetic resonance imaging plays a critical role in the accurate diagnosis of core injuries of the abdominal musculature in the pelvic region.

A dedicated athletic pubalgia MRI protocol has evolved as the imaging standard for core muscle injuries, particularly if the pathology is felt to be extrinsic to the hip joint. It includes large field-of-view sequences of the bony pelvis as well as smaller field-of-view sequences of the pubic symphysis [36]. Meyers et al. have put forth such a specific technique for MRI evaluation of sports hernia, which correlates well with demonstrable injury [17]. This technique uses a surface coil, a send–receive body coil, as well as oblique planes to maximize the evaluation of the osseous and musculotendinous pathology of the pelvis [20]. If the radiologist is unfamiliar with such a protocol, guidance can be found in multiple published reviews [16, 36].

Ideally, coronal oblique and axial oblique sequences through the rectus insertion and pubic symphysis should be obtained in addition to standard sagittal, coronal, and axial sequences. Short tau inversion recovery (STIR) and proton density fat-suppression imaging are useful in identifying bone and soft tissue edema [37]. MRI is 68 % sensitive and 100 % specific for rectus abdominis pathology when compared with findings at surgery and 86 % sensitive and 89 % specific for adductor pathology. It is 100 % sensitive for osteitis pubis [16]. Non-arthrogram studies may be preferred for in-season athletes to avoid the potential for irritation secondary to intra-articular contrast administration.

The MRI should be evaluated in a systematic fashion. Working in a systematic fashion can help identify not only injuries related to athletic

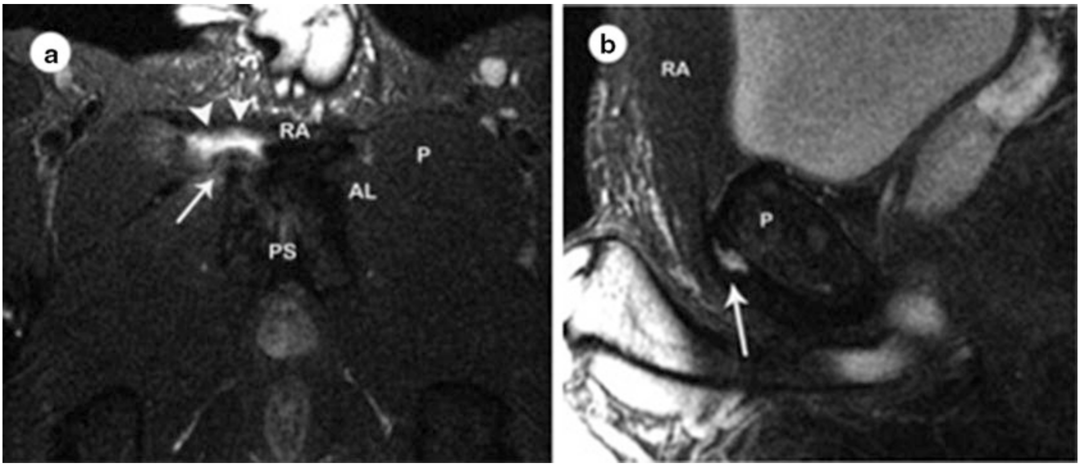


Fig. 5 Axial (**a**) and sagittal (**b**): T_2-weighted fast spin echo fat suppressed images from a noncontrast MRI dedicated to the pelvis using an athletic pubalgia protocol acquired at 1.5 T in a professional football player with refractory right-sided groin pain: on the axial image, the left rectus abdominis (*RA*), pectineus (*P*), and adductor longus (*AL*) are intact, and the pubic symphysis (*PS*) is normal. On the *right*, the rectus abdominis is amputated (*arrowheads*) and the adductor longus is retracted (*arrow*). On the sagittal image, the rectus abdominis is disrupted at its anteroinferior pubic attachment (*arrow*). On this lateral representation of anatomy one cm lateral to the pubic symphysis, "*P*" denotes the pubic bone and "*RA*" the rectus abdominis muscle. Reproduced with permission from Springer [4]

pubalgia but other etiologies of groin pain as well including pelvic muscle strains and tears, osteitis pubis, fracture, sacroiliitis, visceral pathology, and intrinsic hip pathology.

When suspecting athletic pubalgia clinically, the rectus abdominis/adductor longus aponeurosis injuries are the most commonly encountered lesions on MRI. The injury generally involves the caudal aspect of the rectus abdominis insertion, the adductor longus origin, and the pubic tubercle periosteum [14, 16, 20]. Frequently, interstitial tearing of the lateral aspect of the caudal rectus abdominis can be seen, with occasional involvement of the adductor longus tendon (Fig. 5). The injury is usually unilateral and does not extend across the midline. Edema in the anteroinferior aspect of the superior pubic ramus is most consistent with injury to the common adductor–rectus aponeurosis. Frequent findings include fluid signal within the rectus abdominis or adductor insertion, thickening of either structure, peritendinous fluid, or partial or complete disruption of either tendon. Most commonly, there is confluent fluid signal extending from the anterior–inferior insertion of the rectus abdominis into the adductor origin, with corresponding fluid signal in the pubis (Figs. 6, 7, and 8) [16].

Another common pattern of athletic pubalgia injury involves injury to the midline pubic plate. This can be seen on the MRI as fluid extending from the midline pubic symphysis bilaterally [14, 17, 36]. Abnormal T2 hyperintense signal on MRI generally undercuts the rectus abdominis attachments medially, extends inferiorly to involve both the rectus abdominis and adductor aponeurosis, and often extends all the way into one adductor longus tendon where there can be interstitial tearing (Fig. 9). The other adductor tendon is generally spared [14, 17]. This injury seems to be due to a disruption of the entire apparatus at the pubic symphysis. With this type of lesion, osteitis pubis can be a concomitant disorder and the pubic bones should be evaluated for edema, subchondral sclerosis, and cysts suggestive of osteitis pubis (Fig. 10) [14, 36]. As mentioned previously, other pathologies such as rectus abdominis strain, adductor injury, fracture, apophysitis, or bursitis are occasionally present.

Intra-articular hip pathology such as acetabular labral tears are very common in athletes and are a

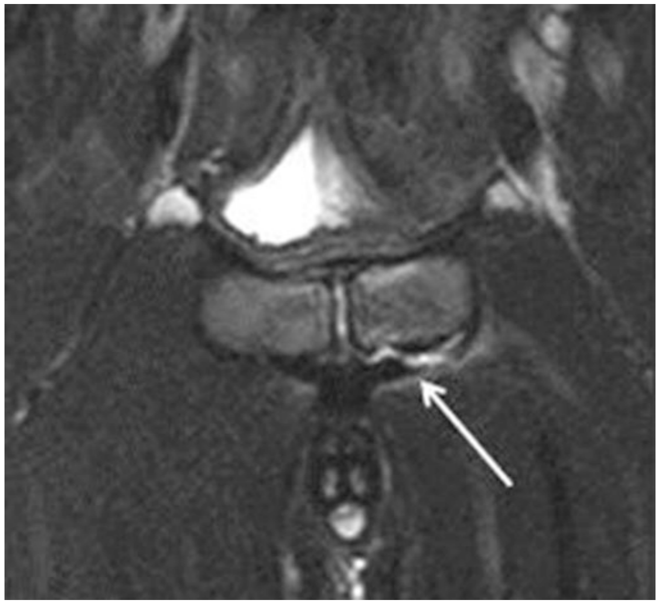


Fig. 6 Oblique axial fat-suppression T2 MRI of the pubic symphysis. There is disruption of the left rectus aponeurosis as it inserts on the anterior aspect of the superior pubic ramus (tip of the *white arrow*). Reproduced with permission from Springer

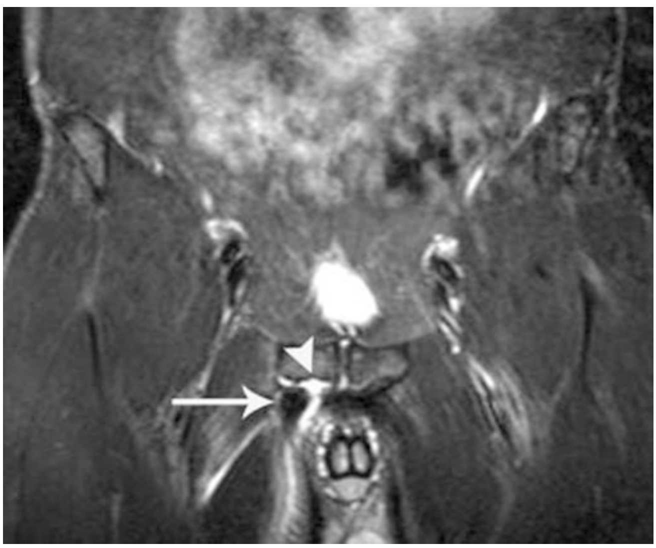


Fig. 7 Coronal short tau inversion recovery image of the pelvis acquired at 1.5 T using an athletic pubalgia protocol in a professional baseball player with an acute right-sided groin injury while fielding a bunt: The brightest signal represents fluid on this fluid-sensitive sequence. Note the abnormal fluid signal tracking inferolaterally from the pubic symphysis (*arrowhead*), sometimes referred to as a secondary cleft sign and often indicating a tear at the rectus abdominis attachment on the pubic bone. The adductor longus tendon has been avulsed and is retracted caudally and laterally (*arrow*). Reproduced with permission from Springer [4]

common differential diagnostic consideration. MR arthrography has been advocated for the assessment of intra-articular hip pathology and has been shown to be superior to conventional MRI for detecting labral tears (87 % vs. 66 %, sensitivity and 64 % vs. 79 % specificity, respectively) [38]. The addition of a local anesthetic to the directly injected contrast can also help distinguish intra-articular from extra-articular causes of pain [39].

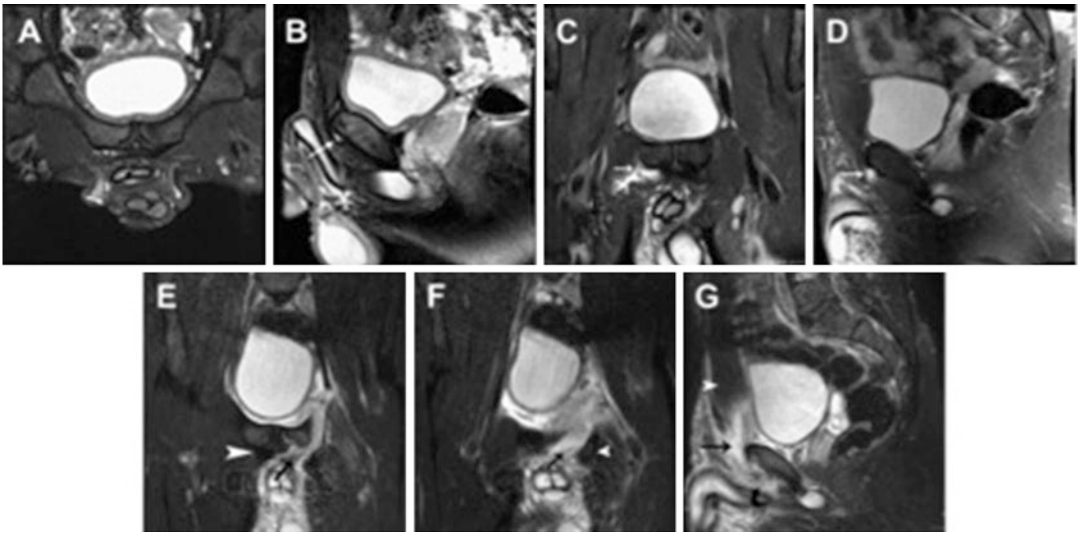


Fig. 8 (**a**) Coronal oblique FSE fat-saturated T2-weighted image from a dedicated athletic pubalgia MRI protocol shows a small tear at the lateral edge of the right rectus abdominis–adductor aponeurosis in a football player (*arrow*). (**b**) Sagittal FSE fat-saturated T2-weighted image from the same examination as (**a**) again shows the lateral edge aponeurosis tear (*arrow*). (**c**) Coronal oblique FSE fat-saturated T2-weighted image demonstrates a large tear at the lateral edge of the right aponeurosis (*arrow*), with retraction of the adductor longus tendon (*arrowhead*), and an interposed hematoma in a professional basketball player with an acute-on-chronic injury. (**d**) Sagittal FSE fat-saturated T2-weighted image from the same examination as (**c**) shows the elongated tenoperiosteal detachment of the rectus abdominis (*arrow*). (**e**) Coronal oblique FSE fat-saturated T2-weighted image in a football player shows a very large aponeurotic lesion with an adductor avulsion on the *left* (*black arrow*). Normal right aponeurosis is seen on the *right* (*white arrowhead*). (**f**) More anterior image from the same examination and sequence as (**e**) confirms an avulsed osseous fragment (*white arrowhead*) arising from the pubic attachment site of the aponeurosis, while the *black arrow* shows hematoma. (**g**) Sagittal fat-saturated T2-weighted FSE image from same patient reveals torn (*black arrow*) and partially retracted rectus abdominis (*white arrowhead*). There is complete avulsion at the level of the aponeurosis (*curved arrow*). Reproduced with permission from Springer [36]

Fig. 9 (**a**) Coronal oblique FSE fat-saturated T2-weighted image shows a midline rectus abdominis–adductor aponeurotic plate lesion (*arrow*) in a professional female distance runner. (**b**) Another midline aponeurotic plate defect (*arrow*) in a 17-year-old male basketball player. (**c**) A large midline aponeurotic plate defect extending to the lateral edges of both aponeurosis (*arrows*) in a middle-aged recreational athlete. Reproduced with permission from Springer [36]

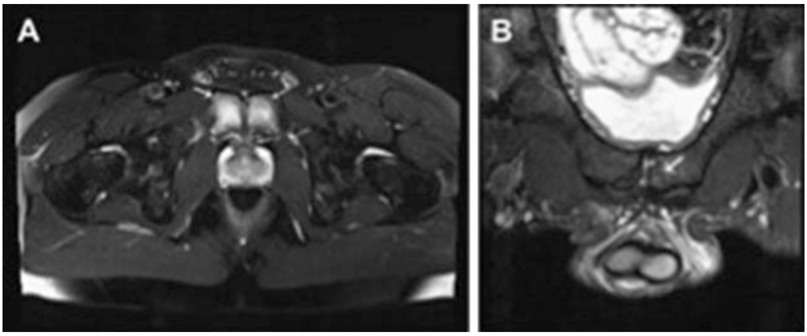


Fig. 10 (**a**) Bone marrow edema spanning the subchondral region of the pubic symphysis anterior to posterior on an axial FSE fat-saturated T2-weighted image (*arrows*) typical for severe osteitis pubis. (**b**) Coronal oblique FSE fat-saturated T2-weighted image shows chronic osteitis pubis with osseous productive change and subchondral cyst formation (*arrow*). Reproduced with permission from Springer [36]

Herniography

Herniography has been used in the past because it allows a dynamic assessment of the abdominal wall musculature. The exam includes an intraperitoneal injection of contrast followed by fluoroscopic evaluation. The patient then performs a Valsalva maneuver several times while in the supine position. Subtle hernias or defects in the abdominal musculature may be identified with this technique [2, 7, 12]. It is not routinely used due to the invasive nature of this study.

More recently, dynamic ultrasound performed during a Valsalva maneuver has been utilized to identify occult hernias. The goal of dynamic ultrasound is to establish posterior inguinal wall deficiency. These methods are less invasive than herniography and may be more appropriate for evaluation of the patient in whom occult hernia is suspected. It is also important to remember that ultrasound is operator dependent [28, 37, 40].

Nonsurgical Treatment

Groin pain due to abdominal wall injury is sometimes self-limited and resolves. At other times, disability persists and surgical intervention is required. Nevertheless, a trial of conservative management should be the first line of treatment, and surgery should be reserved for failures of conservative management.

Conservative management for athletic pubalgia is similar to other causes of groin pathology. Generally, a brief period of rest is indicated. Physical therapy should emphasize core strengthening and identification and treatment of weakness and restricted motion in the musculature of the hip and pelvis. Ice and NSAIDs can be helpful for managing pain. Therapeutic modalities including ultrasound and electrical stimulation may also be useful [24].

Woodward et al. described a nonoperative treatment protocol for acute sports hernia that allowed them to return a professional hockey player to sport without surgical intervention or recurrence of his symptoms [41]. Their protocol involved three phases. Phase 1 emphasized pain control with ice and electrical stimulation, manual therapy, and pool therapy targeting lumbar, hip, and pelvis control. Phase 2 began when the athlete had no pain at rest and minimal pain with activity and had normal motion and mild strength deficit. This phase consisted of a strength and stability progression. Phase 3 was initiated when the athlete had very minimal pain with high-intensity straight line activities. Sport-specific training was initiated, and the athlete's pain level was carefully monitored to prevent a worsening of symptoms. The entire progression took approximately 7 weeks, and the athlete did not have a recurrence of his symptoms during eight ensuing seasons in the National Hockey League. This is an isolated case report, however, and there is a paucity of evidence regarding the efficacy of nonsurgical treatment of athletic pubalgia.

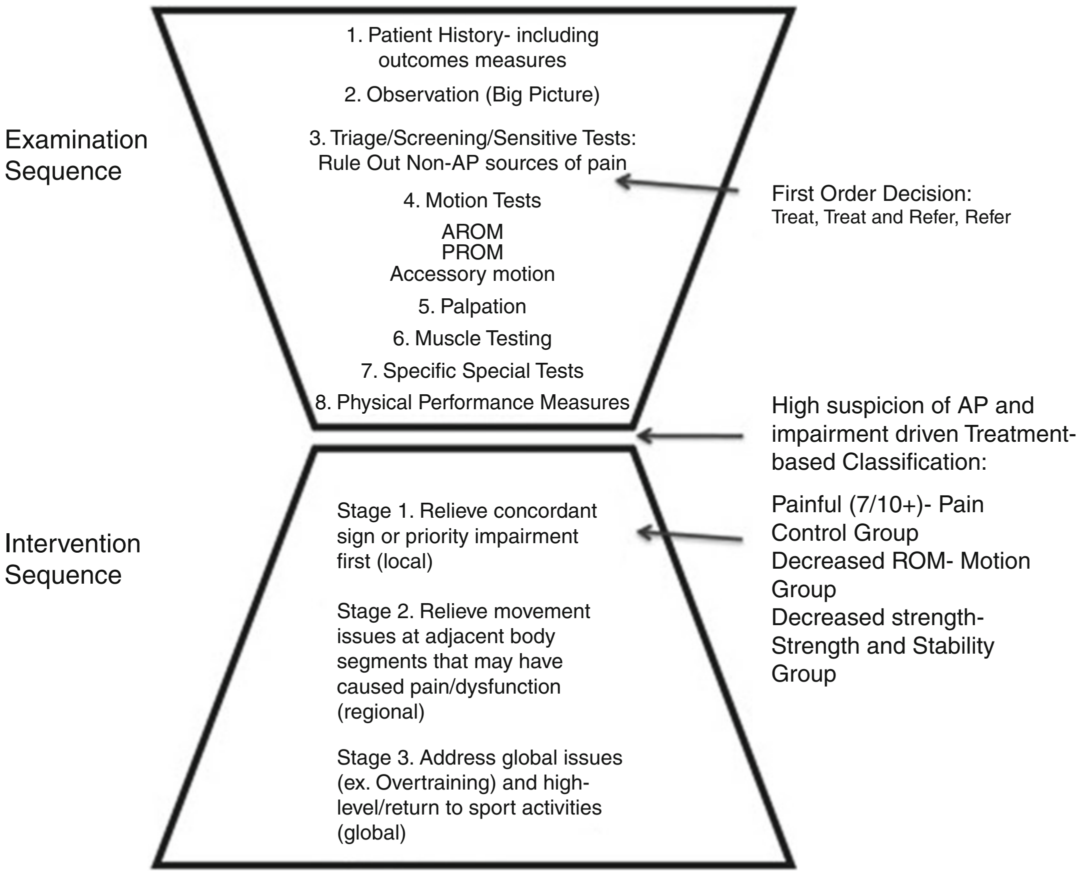


Fig. 11 A proposed paradigm for examination and treatment of patients with athletic pubalgia. Reproduced with permission from Springer [42]

The physical therapy literature has recently published another suggested model for physical examination and conservative treatment of athletic pubalgia [42]. Interventions are based on patient impairments and are divided into local, regional, and global interventions. Local interventions consist of managing pain, range of motion limitation, strength, and stability limitations via manual interventions including joint mobilization, manipulation, and passive range of motion techniques. A passive range of motion and stretching exercise program has been recommended as part of an injury prevention program; however, more evidence suggests that active exercise programs may improve abduction range of motion without specifically stretching the adductors [43]. With regard to strengthening, strengthening of the hip adductors, rectus abdominis, abdominal obliques, and transverse abdominis muscles is advocated. Lastly, global interventions are implemented that focus on rehabilitating athletes and preparing them for a return to sport by focusing on sport-specific functional rehabilitation (Fig. 11) [42].

One of the downsides of conservative management is the length of recovery. Patients may be required to have 6–8 weeks of rest followed by progressive physical therapy and strengthening [44]. With traditional conservative management, it may be up to 10–12 weeks after initiation of treatment prior to return to sports [44].

Operative Treatment

When conservative treatment measures fail, surgical treatment may be pursued [8, 16, 45]. Many elite athletes may choose to wait until the off-season to opt for surgery in order to minimize time missed during the regular season. This is a reasonable option for athletes who are able to continue with their athletic activity despite disability. Waiting has not been shown to worsen outcomes with surgical repair.

A variety of surgical procedures have been described for the treatment of athletic pubalgia. Although initially procedures were performed with open surgical techniques, they historically required a prolonged time for recovery. A number of laparoscopic and minimally invasive procedures have been developed in an effort to decrease overall recovery time [7]. One series of 35 professional soccer players treated with laparoscopic mesh repair of the posterior wall showed 87 % excellent results and return to play at 10 days [46]. Gentisaris et al. reported on 131 professional athletes who underwent laparoscopic mesh repair and found that 97 % returned to full sporting activities within 2–3 weeks [47]. Caudill's systematic review of the literature found very good and comparable results between open and laparoscopic repairs based on return to sports activity (92.8 % vs. 96 %, respectively) [44].

One study found that of 43 professional National Hockey League (NHL) players who underwent a "sports hernia" operation, 80 % would return to play two or more full seasons [48]. Additionally, the younger players (those that played less than seven seasons prior to injury) were shown to return to their pre-injury level of production, while more veteran players had a decrease in their production after injury. Nevertheless, the study demonstrates that surgical repair is a viable option and allows players to return to play at the elite level.

Additionally, one randomized control trial has shown that laparoscopic surgery for athletic pubalgia is more effective than nonoperative treatment [49]. In this study, 60 athletes with athletic pubalgia were randomized into operative and nonoperative arms. Thirty athletes who underwent an operation had a statistically significant decrease in groin pain at 1 and 12 months ($p < 0.0001$), and 90 % (27 patients) returned to sporting activity within 3 months compared to just 27 % (8 patients) in the nonoperative group ($p < 0.0001$) [49]. Seven patients in the nonoperative arm chose to undergo surgical treatment after 6 months, and all returned to the same level of sports they were at prior to the groin injury.

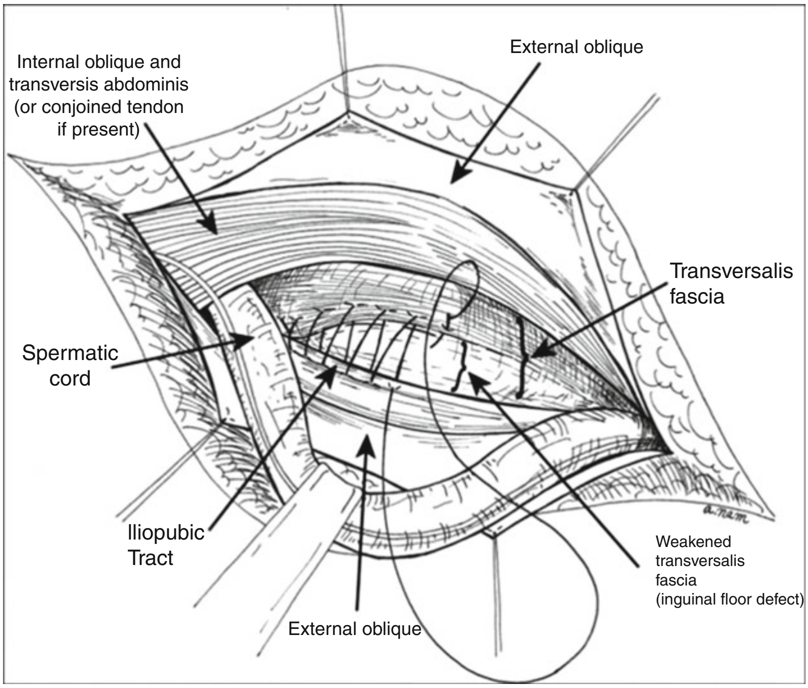
Laparoscopic Repair

Laparoscopic treatment of sports hernia can be approached by one of two ways: transabdominal preperitoneal approach (TAPP) and totally extraperitoneal (TEP). Both approaches are well described in the literature [50]. In the TAPP approach, the peritoneal cavity is entered, a flap of peritoneum is raised, and a piece of polypropylene mesh is placed in the preperitoneal space. In the TEP approach, the peritoneal space is not entered and a mesh is placed in the inguinal region [1]. Both approaches have reportedly similar outcomes. One study showed that return to full training resumes at an average of 5 weeks after open approach compared with 3 weeks after laparoscopic approaches [51].

Open Repair

Open repairs of sports hernias date back to the 1980s when Gilmore first started treating Gilmore's groin. Repairs with and without the use of mesh have been described. Mesh repairs are considered to be tension-free repairs as opposed to suture repairs which are under tension [1]. Generally, anatomic defects found intraoperatively consisted of a tear in the external oblique aponeurosis weakness in the posterior wall of the inguinal canal, a tear in the transversalis fascia, a tear in the conjoined tendon, or an inguinal hernia [26]. Two examples of open repairs include the Modified Bassini repair which uses mesh (Fig. 12) and the

Fig. 12 Modified Bassini repair described by Steele and colleagues. The transversalis fascia is approximated to the iliopubic tract from the internal to external ring with a running Prolene suture. Steele and colleagues then reinforced the floor of the inguinal canal by placing a mesh on the floor (not shown). The standard Bassini repair is performed by suturing the transversus abdominis and internal oblique musculoaponeurotic arches to the iliopubic tract. Reproduced with permission from Springer [26]



Modified Shouldice repair, which does not use mesh (Fig. 13). These repairs have been well described in the literature and used successfully in the past [5, 12, 26].

Two series on open repair of posterior wall deficiency showed excellent results in 89 % and 93 %, respectively, with return to pre-injury level of competition [40]. Hackney et al. showed 87 % of players return to play at 6 weeks after open repair. The repair included reconstruction of the internal inguinal ring, plication of the transversalis fascia, and apposition of the conjoint tendon to the internal inguinal ring [12].

Meyers et al. described their open approach, which consisted of a broad pelvic floor repair involving reattachment of the anteroinferior rectus to the pubis and a variation of an adductor release. Mesh was not utilized. In a series of 157 athletes treated with this technique, 88 % and 96 % performed at or above their pre-injury level by 3–6 months, respectively. The 96 % success rate group was made up of patients who underwent both a rectus repair and an adductor release [8].

Another technique described involves a tension-free suture repair of the weakened posterior wall of the transversalis fascia and decompression or ablation of the genital branch of the genitofemoral nerve. Return to sport occurred at day 14, and 83.7 % of professional athletes were fully competitive in their sport at 28 days postoperatively [52].

The use of a polypropylene mesh and an internal oblique flap to reinforce the inguinal floor and rebalance the rectus abdominis origin from the pubic tubercle has also been described [31].

Overall, in the literature there does not appear to be a significant difference in outcome between laparoscopic and open treatment of athletic pubalgia or between the use of a mesh or not. However, as mentioned previously, there does appear to be a quicker return to sport with laparoscopic procedures [7]. Most evident is the lack of validated outcome measures and long-term follow-up in order to determine the optimal surgical technique.

Complications

The most common postoperative complaint is minor bruising or edema involving the abdomen, thighs, genitals, and perineum. Postoperative hematoma requiring reoperation occurred in

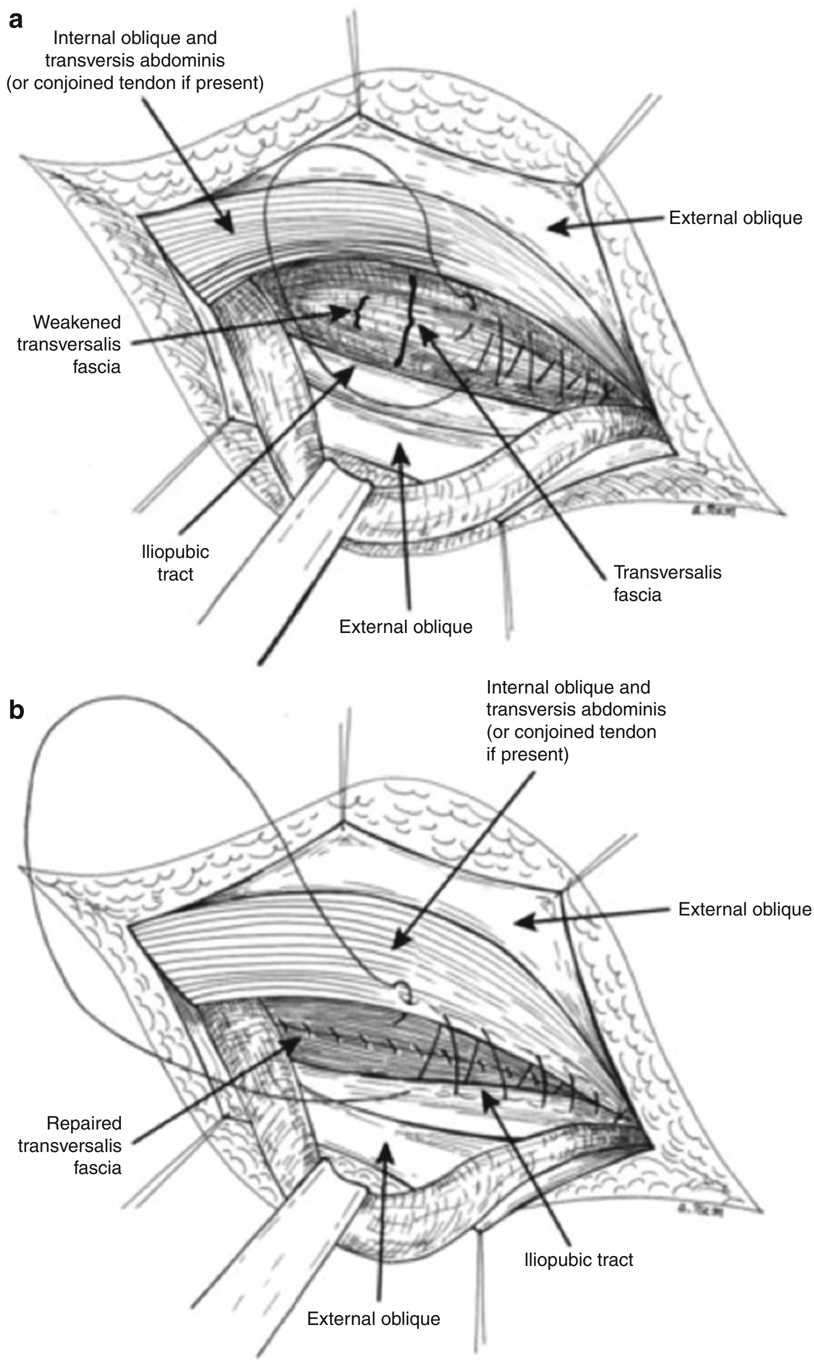


Fig. 13 Modified Shouldice repair. (**a**) The transversalis fascia is plicated with nonabsorbable running suture. (**b**) The next layer approximates the internal oblique and transversus abdominis muscles (or the conjoined tendon if present) to the iliopubic tract and covers the plicated transversalis fascia. Reproduced with permission from Springer [26]

0.3 % of patients, and the wound infection rate was 0.4 % [20]. Nerve dysesthesia of the ilioinguinal, genitofemoral, anterior, or lateral femoral cutaneous nerve distribution occurred in 0.3 % of patients [20]. Penile vein thrombosis occurred in 0.1 % of patients but all resolved [20]. There is also the potential for postoperative scar tissue and subsequent neural dysesthesias.

The most common reason for reoperation was development of similar symptoms on the contralateral side. The second most common was for adductor release not carried out at the first surgery [20]. Another common reason for continued disability after surgical treatment results from failure to identify associated intra-articular hip pathology (i.e., FAI) [9, 20].

Summary

Athletic pubalgia is a syndrome of chronic lower abdominal or groin pain that is caused by an injury to the fascial sheath of the rectus abdominis and adductor complex. Its causes are multifactorial, but generally, it is an overuse injury from high-level athletic activity. Although the differential diagnosis of groin pain is diverse, a thorough history, physical examination, and appropriate use of imaging can help accurately diagnose athletic pubalgia. Sometimes, nonoperative treatment is successful and consists of rest, NSAIDs, and physical therapy. If symptoms persist after 6–12 weeks of conservative treatment, surgical intervention is an option and has been shown to be successful. A variety of surgical procedures have been described for the treatment of athletic pubalgia, and most of them have similar outcomes. It is important to remember to evaluate for concomitant femoroacetabular impingement as it can frequently coexist with athletic pubalgia and lead to a high failure rate if left untreated.

References

1. Litwin DE, et al. Athletic pubalgia (sports hernia). Clin Sports Med. 2011;30(2):417–34.
2. Gilmore J. Groin pain in the soccer athlete: fact, fiction, and treatment. Clin Sports Med. 1998;17(4):787–93, vii.
3. Verrall GM, et al. Outcome of conservative management of athletic chronic groin injury diagnosed as pubic bone stress injury. Am J Sports Med. 2007;35(3):467–74.
4. Meyers WC, et al. Understanding "sports hernia" (athletic pubalgia): the anatomic and pathophysiologic basis for abdominal and groin pain in athletes. Op Tech Sports Med. 2012;20(1):33–45.
5. Gilmore O. Gilmore's groin: ten years experience of groin disruption – a previously unsolved problem in sportsmen. Sports Med Soft Tissue Trauma. 1991;3(3):12–4.
6. Morelli V, Espinoza L. Groin injuries and groin pain in athletes: part 2. Prim Care. 2005;32(1):185–200.
7. Swan Jr KG, Wolcott M. The athletic hernia: a systematic review. Clin Orthop Relat Res. 2007;455:78–87.
8. Meyers WC, et al. Management of severe lower abdominal or inguinal pain in high-performance athletes. PAIN (Performing Athletes with Abdominal or Inguinal Neuromuscular Pain Study Group). Am J Sports Med. 2000;28(1):2–8.
9. Larson CM, Pierce BR, Giveans MR. Treatment of athletes with symptomatic intra-articular hip pathology and athletic pubalgia/sports hernia: a case series. Arthroscopy. 2011;27(6):768–75.
10. Feeley BT, et al. Hip injuries and labral tears in the national football league. Am J Sports Med. 2008;36(11):2187–95.
11. Birmingham PM, et al. The effect of dynamic femoroacetabular impingement on pubic symphysis motion: a cadaveric study. Am J Sports Med. 2012;40(5):1113–8.
12. Hackney RG. The sports hernia: a cause of chronic groin pain. Br J Sports Med. 1993;27(1):58–62.
13. Gamble JG, Simmons SC, Freedman M. The symphysis pubis. Anatomic and pathologic considerations. Clin Orthop Relat Res. 1986;203:261–72.
14. Mullens FE, et al. Review of MRI technique and imaging findings in athletic pubalgia and the "sports hernia". Eur J Radiol. 2012;81(12):3780–92.
15. Birmingham PL, C. Soft tissue injuries of the hip: adductor strain and athletic pubalgia.
16. Zoga AC, et al. Athletic pubalgia and the "sports hernia": MR imaging findings. Radiology. 2008;247(3):797–807.
17. Palisch A, Zoga AC, Meyers WC. Imaging of athletic pubalgia and core muscle injuries: clinical and therapeutic correlations. Clin Sports Med. 2013;32(3):427–47.
18. Akita K, et al. Anatomic basis of chronic groin pain with special reference to sports hernia. Surg Radiol Anat. 1999;21(1):1–5.
19. Ziprin P, Williams P, Foster ME. External oblique aponeurosis nerve entrapment as a cause of groin pain in the athlete. Br J Surg. 1999;86(4):566–8.
20. Meyers WC, et al. Experience with "sports hernia" spanning two decades. Ann Surg. 2008;248(4):656–65.
21. Spinelli A. A new sports injury pubic pain in fencers. Orthop Trauma dell'Apparato Motore. 1932;4:111–27.
22. Cabot J. Osteopatica dinamica del pubis. Proceedings of the XVI World Congress of Sports Medicine; 1966. Hannover.
23. Irshad K, et al. Operative management of "hockey groin syndrome": 12 years of experience in National Hockey League players. Surgery. 2001;130(4):759–64, discussion 764–6.
24. Minnich JM, et al. Sports hernia: diagnosis and treatment highlighting a minimal repair surgical technique. Am J Sports Med. 2011;39(6):1341–9.
25. Ekstrand J, Gillquist J. The avoidability of soccer injuries. Int J Sports Med. 1983;4(2):124–8.

26. Nam A, Brody F. Management and therapy for sports hernia. J Am Coll Surg. 2008;206(1):154–64.
27. Kachingwe AF, Grech S. Proposed algorithm for the management of athletes with athletic pubalgia (sports hernia): a case series. J Orthop Sports Phys Ther. 2008;38(12):768–81.
28. Garvey JF, Read JW, Turner A. Sportsman hernia: what can we do? Hernia. 2010;14(1):17–25.
29. Engebretsen AH, et al. Intrinsic risk factors for groin injuries among male soccer players: a prospective cohort study. Am J Sports Med. 2010;38(10):2051–7.
30. Farber AJ, Wilckens JH. Sports hernia: diagnosis and therapeutic approach. J Am Acad Orthop Surg. 2007;15(8):507–14.
31. Ahumada LA, et al. Athletic pubalgia: definition and surgical treatment. Ann Plast Surg. 2005;55(4): 393–6.
32. DeAngelis NA, Busconi BD. Assessment and differential diagnosis of the painful hip. Clin Orthop Relat Res. 2003;406:11–8.
33. Clohisy JC, et al. A systematic approach to the plain radiographic evaluation of the young adult hip. J Bone Joint Surg Am. 2008;90 Suppl 4:47–66.
34. Dunn DM. Anteversion of the neck of the femur; a method of measurement. J Bone Joint Surg (Br). 1952;34-B(2):181–6.
35. Williams PR, Thomas DP, Downes EM. Osteitis pubis and instability of the pubic symphysis. When nonoperative measures fail. Am J Sports Med. 2000;28(3):350–5.
36. Khan W, Zoga AC, Meyers WC. Magnetic resonance imaging of athletic pubalgia and the sports hernia: current understanding and practice. Magn Reson Imaging Clin N Am. 2013;21(1):97–110.
37. Davies AG, et al. Review: imaging of groin pain in the athlete. Skelet Radiol. 2010;39(7):629–44.
38. Smith TO, et al. The diagnostic accuracy of acetabular labral tears using magnetic resonance imaging and magnetic resonance arthrography: a meta-analysis. Eur Radiol. 2011;21(4):863–74.
39. Safran M. Evaluation of the hip: history, physical examination, and imaging. Op Tech Sports Med. 2005;13(1):2–12.
40. Orchard JW, et al. Groin pain associated with ultrasound finding of inguinal canal posterior wall deficiency in Australian Rules footballers. Br J Sports Med. 1998;32(2):134–9.
41. Woodward JS, Parker A, Macdonald RM. Non-surgical treatment of a professional hockey player with the signs and symptoms of sports hernia: a case report. Int J Sports Phys Ther. 2012;7(1):85–100.
42. Hegedus EJ, et al. A suggested model for physical examination and conservative treatment of athletic pubalgia. Phys Ther Sport. 2013;14(1):3–16.
43. Holmich P, et al. Effectiveness of active physical training as treatment for long-standing adductor-related groin pain in athletes: randomised trial. Lancet. 1999;353(9151):439–43.
44. Caudill P, et al. Sports hernias: a systematic literature review. Br J Sports Med. 2008;42(12):954–64.
45. Anderson K, Strickland SM, Warren R. Hip and groin injuries in athletes. Am J Sports Med. 2001;29 (4):521–33.
46. Susmallian S, et al. Laparoscopic repair of "sportsman's hernia" in soccer players as treatment of chronic inguinal pain. Med Sci Monit. 2004;10(2): CR52–4.
47. Genitsaris M, Goulimaris I, Sikas N. Laparoscopic repair of groin pain in athletes. Am J Sports Med. 2004;32(5):1238–42.
48. Jakoi A, et al. Sports hernia in National Hockey League players: does surgery affect performance? Am J Sports Med. 2013;41(1):107–10.
49. Paajanen H, et al. Laparoscopic surgery for chronic groin pain in athletes is more effective than nonoperative treatment: a randomized clinical trial with magnetic resonance imaging of 60 patients with sportsman's hernia (athletic pubalgia). Surgery. 2011;150(1):99–107.
50. Memon M, Fitzgibbons RJ. Laparoscopic inguinal hernia repair: transabdominal preperitoneal (TAPP) and totally extraperitoneal (TEP). In: Scott-Conner CEH, editor. The SAGES manual. New York: Springer; 2006. p. 467–81.
51. Ingoldby CJ. Laparoscopic and conventional repair of groin disruption in sportsmen. Br J Surg. 1997;84 (2):213–5.
52. Muschaweck U, Berger L. Minimal repair technique of sportsmen's groin: an innovative open-suture repair to treat chronic inguinal pain. Hernia. 2010;14(1):27–33.

Pelvic Floor Dysfunction

59

Sheila A. Dugan and Sol M. Abreu-Sosa

Contents

S.A. Dugan (✉)
Rush Medical College, Departments of PM&R and Preventive Medicine, Rush University Medical Center, Chicago, IL, USA
e-mail: Sheila_Dugan@rush.edu

S.M. Abreu-Sosa
Clinica de Medicina Fisica y Electrodiagnostico (CMFE), Aguada, PR, USA

Buen Samaritano Hospital, Aguadilla, PR, USA
e-mail: sol_abreu@yahoo.com

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_63

Abstract

The specific aim of this chapter is to introduce the role of the pelvic floor and pelvic floor dysfunction for arthroscopic and open hip joint preservation surgery patients presenting with pain and functional impairments. Painful pelvic floor muscles may occur as a result of inherent musculoskeletal causes or may be the result of somatic dysfunction occurring in the hip. An overview of the muscles, bones, and nerves of the pelvis and pelvic floor will be presented. Visceral and somatic referral patterns and biomechanical links between the hip, pelvis, pelvic floor, and lumbar spine will be pointed out, in particular in the context of the patient with chronic symptoms and/or increased tone and muscle dysfunction. The reader will be exposed to the epidemiology, pathophysiology, and presentation of pelvic floor dysfunction in order to raise awareness of the pelvic floor as a symptom generator in "hip" pain patients. Patient evaluation, including the pelvic floor examination, will be presented to increase understanding of how to utilize the physical examination to identify dysfunctions and guide in comprehensive treatment planning. Although some clinicians may prefer to refer pelvic floor patients to expert providers, this chapter will provide knowledge regarding the evaluation such providers will perform; collaborative care with physicians and physical therapists treating pelvic floor patients is invaluable in this patient population. Ultimately, knowledge of pelvic floor function and dysfunction will broaden the differential diagnosis for patients presenting with hip- and pelvic-related pain increasing the accuracy of diagnosis and help dictate the most appropriate treatment for these patients.

Introduction

The goal of this chapter is to raise awareness of pelvic floor dysfunctions that may present in patients referred for arthroscopic and open hip joint preservation surgery. The term pelvic floor refers collectively to the pelvic diaphragm, the sphincter mechanism of the lower urinary tract, the upper and lower vaginal supports (in women), and the internal and external anal sphincters [1]. Painful pelvic floor muscles may occur as a result of inherent musculoskeletal causes in the pelvic floor muscles, ligaments, and tendons or as a functional adaptation to other disorders within the pelvis-hip-spine complex [2]. Painful muscles may also be secondary to gynecological, gastrointestinal/colorectal, gyn-urological/urological, and psychological disorders [2]. The orthopedic provider must be cognizant of the role of the pelvic floor in order to provide comprehensive care for patients being considered for hip arthroscopy and open hip joint preservation surgery. The reader is likely well acquainted with a broad differential diagnosis for pelvic pain patients but may not have been exposed to the presentation, physical examination, and treatment of pelvic floor dysfunction. This chapter aims to fill this gap in evaluating and treating the patient presenting for management of hip and pelvic pain.

Pathophysiology

Dysfunction of the pelvic floor muscles, pelvic floor dysfunction (PFD), is a term that may be applied to a group of clinical disorders that include urinary incontinence, anal incontinence, pelvic organ prolapse, sensory and emptying abnormalities of the lower urinary tract, defecatory dysfunction, sexual dysfunction, and several chronic pain syndromes [3]. Clinically, PFDs are commonly divided into hypertonic and hypotonic. Patients presenting to a clinic for hip arthroscopy and hip joint preservation surgery evaluation or follow-up with PFD will typically present with painful pelvic floor muscles with hypertonic dysfunction. Hypertonic or high-/increased-tone dysfunctions include pain and excessive muscle tension and can present with associated constipation and dyspareunia (pain with intercourse). Hypotonic or low-/decreased-tone dysfunctions can present with incontinence and may be related to collagen changes, previous childbirth or gynecological surgery, or peripheral nerve injury, among other causes [2].

Epidemiology and Risk Factors for PFD

The medical literature provides insights into the epidemiology of PFD; however, hypotonic dysfunctions and female subjects have been the focus of the majority of studies. The most prevalent PFDs like urinary incontinence, fecal incontinence, and pelvic organ prolapse afflict women three to seven times more often than men. The highest gender disparity is seen between the ages of 45 and 69 years [4] Overall, 23.7 % of women had symptoms consistent with at least 1 pelvic floor disorder. Of these, 15.7 % experienced urinary incontinence, 9.0 % experienced fecal incontinence, and 2.9 % experienced symptomatic pelvic organ prolapse. The proportion of women that reported at least 1 pelvic floor disorder increased with age; 9.7 % in women aged 20–39 years, 26.5 % in women aged 40–59 years, 36.8 % in women aged 60–79 years, and 49.7 % in women aged 80 years or older. Other characteristics that were significantly associated with at least 1 pelvic floor disorder were (1) family poverty income ratio, 20.8 % if>2-fold above the poverty threshold vs 28.8 % if below the poverty threshold and 29.7 % if 1–2 $\times$ above the poverty threshold; (2) body mass index 15.1 % for underweight/normal weight, 26.3 % for overweight, and 30.4 % for obese women; and (3) parity for nulliparous women vs 18.4 %, 24.6 %, and 32.4 % for those women with 1, 2, and $\geq$3 deliveries, respectively. Race/ethnicity and education were not significantly associated with having at least 1 pelvic floor disorder [5]. These problems are currently underreported, often embarrassing for the patient, and undertreated [6].

For hip arthroscopy and open hip joint preservation patients, PFDs to consider include primarily hypertonic ones, presenting with painful pelvic floor muscles and associated impairments like constipation or dyspareunia. While both genders can present with pelvic floor pain, a women's unique anatomy and biomechanics place her at increased risk to develop pain in the pelvic region as compared to men [7]. Hip structural abnormalities also place women at increased risk for pelvic pain, particularly pelvic floor pain. Muscles involved in hip function often respond to injury or dysfunction by guarding and may even rest in a contracted or overactive position. The increase in resting state in turn may lead to pelvic floor pain [2]. As with any musculoskeletal (MSK) patient presentation, the differential diagnosis is initially broad and rules out other possible pain generators, based on history and physical examination, before deciding on PFD as the symptom generator. For example, lumbar radiculopathy might also lead to pelvic floor pain since the lower lumbar roots and sacral roots innervate the pelvic floor. A comprehensive review of the causes, presentation, physical examination, and treatment of MSK pelvic pain is beyond the scope of this chapter.

Although not widely discussed, the pelvic floor has an important role in the function of core muscle stabilization provided by the muscles of the trunk (abdominals, quadratus lumborum, spinal muscles including the multifidus, and hip muscles, including iliopsoas), the diaphragm, and the pelvic floor. An increase in the resting state of the muscles of the pelvic floor can result in increased pressure within the spinal column that can contribute to low back pain [2].

Interstitial cystitis, irritable bowel syndrome, endometriosis, dysmenorrhea, nonbacterial prostatitis, fibroids, constipation, pregnancy and/or delivery, physical inactivity, history of sexual abuse, and other chronic pelvic pain syndromes are all factors that need to be considered when evaluating a patient for pelvic floor dysfunction [2, 6]. For example, a previous history of coccydynia may be a clue to a history of sustained muscle contraction that can play a role in persistent muscle guarding patterns. In patients with sustained contractions, visceral somatic reflexes previously triggered by the colon, uterus, bladder, prostate, or other organ source may set the stage for somatic reflexes. As has been described for visceral reflexes, referred pain from somatic structures, in this case, the hip, cause skeletal muscles attaching at or near the hip joint to be tender and tense. Referred pain from somatic structures has been attributed mainly to central hyperexcitability, triggered by the primary pain generator, in this case the hip, and peripheral reflex arc activation [8]. See Fig. 1

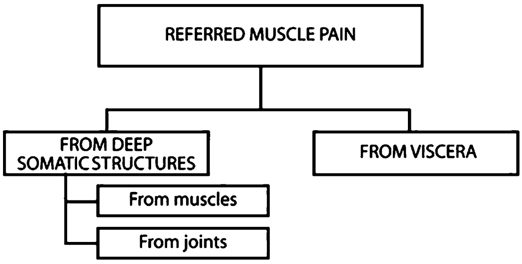


Fig. 1 Referred muscle pain from somatic and visceral sources (From Giamberardino M in Encyclopedia of Pain, SpringerReference, 2013 [23])

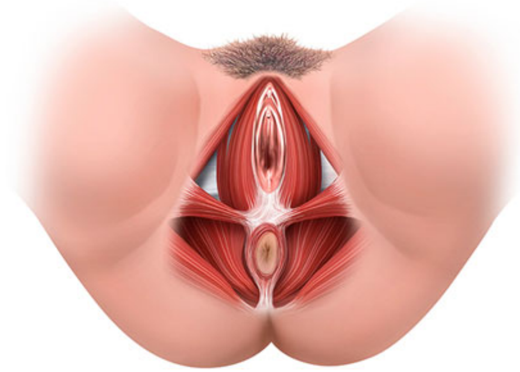


Fig. 2 Muscles of the pelvic floor (With permission from PIXOLOGICSTUDIO/Science Source) (Female pelvic floor. Computer artwork of the female pelvic floor muscles seen from below. The muscles maintain continence as part of the urinary and anal sphincters. At *center top* is the clitoris, with the urethra, vagina and rectum below it. The bulbospongiosus muscle, surrounds the urethra and vagina. The external anal sphincter controls the anal opening. Either side of the anus are the levator ani, which support the pelvic organs and surrounds the anus and vagina. Running across center is the deep transverse perineal muscle, which also supports the pelvic organs)

Anatomy of the Pelvic Floor

The pelvic floor complex consists of muscles, ligaments, and fascia that form a multilayered support for the inferior pelvis in order to perform three important functions: supportive, sphincteric, and sexual [9]. It is composed of visceral pelvic fascia and pelvic diaphragm, the urogenital and anal triangles with superficial and deep genital muscles, and the sphincters of the urethra and anus. The pelvic diaphragm muscles can be subdivided into the levator ani group, the coccygeus, and the obturator internus. The perineum can be divided into the urogenital and anal triangle regions, with the perineal body the anchor point between them. Superficial external genital muscles include the external anal sphincter in the anal triangle and the superficial transverse perineal, bulbocavernosus, and ischiocavernosus in the urogenital triangle. In part they form a figure of eight around the front passages (vaginal and urethral) and the back passage, the anus [9]. See Fig. 2. The interaction between the pelvic floor muscles and the supportive ligaments is critical to pelvic organ support [10]. The urogenital diaphragm (the perineal membrane) is deeper in the urogenital triangle. It is anterior to and more superficial than the pelvic diaphragm and oriented transversely with fascia and muscle that spans from the ischiopubic rami bilaterally. It consists of the striated urogenital sphincters: sphincter urethrae, compressor urethrae, and urethrovaginal sphincter muscles [9]. Female urethral sphincters include the striated rhabdosphincter sphincter (the sphincter urethrae) and two distal urethral sphincters, the intrinsic sphincter, and external sphincters. The levator ani and the compressor urethrae muscles assist the sphincters in urethral closing. The psoas muscle invests into the pelvic fascia, affording a direct biomechanical link between function or psoas dysfunction and PFD. See Fig. 3.

The pelvic walls are formed by bones and ligaments partly lined with muscles and covered with fascia. The anterior pelvic wall is a shallow wall formed by the posterior surfaces of the pubic bones and symphysis pubis. The posterior pelvic wall is a more extensive wall, consisting of the sacrum, coccyx, and piriformis muscle. The lateral pelvic wall is a component of the pelvis formed by part of the innominate bone, the obturator foramen, the sacrotuberous and sacrospinous ligaments, and the obturator internus muscle and fascia. The inferior pelvic wall, or pelvic floor, consists of the levator ani muscles, coccygeus, and pelvic fascia. It is accessible to palpation only through internal vaginal (women) or rectal (women and men) examination [2].

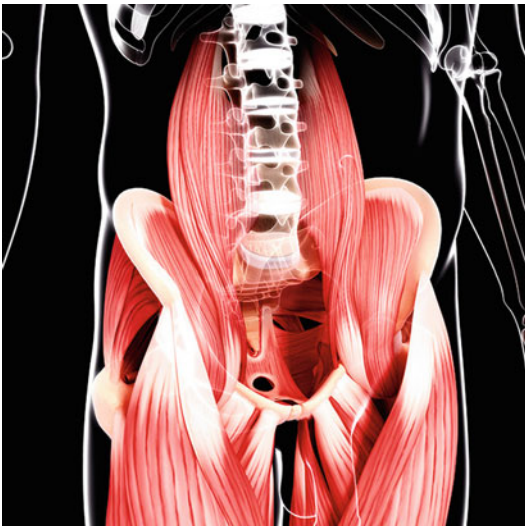


Fig. 3 Psoas muscle (With permission from PIXOLOGICSTUDIO/Science Source) (Human hip musculature, computer artwork)

Bones and Ligaments That Form the Pelvic Floor

The pelvis is a ring and the base of support for the upper and lower torso and extremities. Located in the posterior pelvic ring are the sacroiliac joints. These are synovial joints inferiorly, have anterior and posterior capsules, and are surrounded by ligaments: the anterior sacroiliac ligament, the dorsal longitudinal sacroiliac joint (SIJ) ligament, and the interosseous sacroiliac ligaments, which could contribute to posterior pelvic pain [2]. Ligaments extrinsic to the SIJ but that contribute to posterior pelvic pain include the iliolumbar and sacrotuberous ligaments. The iliolumbar ligament absorbs and distributes forces from the lumbar spine and ilium. The sacrotuberous ligament absorbs and distributes forces from the lower extremity through direct and fascial attachments to the hamstrings and thoracodorsal fascia.

The symphysis pubis is a cartilaginous joint between the two pubic bones. It is surrounded by ligaments that allow subtle motion in frontal, inferior, and superior sheer motion and rotational planes. This joint is subjected to substantial mechanical stresses during pregnancy. Muscles from the abdomen (rectus abdominis) and lower extremities (adductors) insert or originate at the pubis assisting with load transfer, providing a direct pathway for myofascial overload and pain referral patterns from the pelvis cephalad to the abdomen or caudad to the lower extremities. Lastly the sacrococcygeal joint is a cartilaginous joint that is joined by ligaments. Much movement is possible at this joint, although this varies among individuals [2]. The joint can be palpated both externally and internally, with internal palpation via the rectum affording the best assessment of pain, mobility, and alignment.

Nerves to the Pelvic Floor

The lumbosacral trunk passes into the pelvis and joins the sacral nerves as they emerge from the anterior sacral foramina. From a clinical perspective, the important nerve branches associated with clinical syndromes at or near the pelvic floor include sciatic, obturator, femoral, lateral femoral cutaneous, and pudendal nerve [2]. The clinical anatomy of the pudendal nerve and its branches make it prone to damage during complicated vaginal childbirth and surgical interventions. The pudendal nerve arises from the sacral plexus from the ventral branches S2, S3, and S4. It supplies sensation to the external genitalia and motor function to the urinary and external anal sphincters and muscles responsible for ejaculation in men and orgasm in both genders. Because of its complex and comprehensive function, the pudendal nerve is implicated in PFD related to pain, incontinence, and sexual dysfunction [2]. Recent studies have shown that the levator ani muscles were innervated by a nerve that originated from S2–4 that branches at a point proximal to the ischial spine (before the pudendal nerve roots reach the sacrospinous ligament) and then innervates the levator ani muscles on their pelvic surface [11]. As noted previously, the pelvic floor reflex innervation affords wide representation of referred pain from primary pain generators in visceral and somatic structures via mechanisms such as activation of peripheral reflex arcs and central hyper excitability. Pelvic floor hypertonic dysfunction can present with dysfunction of the viscera that are controlled by the pelvic floor and may present as a primary pain generator or solely as a component of a chronic pain syndrome [12].

Understanding the Presentation of Arthroscopic and Open Hip Joint Preservation Surgery Patients with Pelvic Floor Dysfunction

As noted earlier in the chapter, muscles involved in hip function often respond to injury or dysfunction by guarding and may even rest in a contracted or overactive position. While this may be easily recognized on history and physical examination in muscle that attached externally to the hip and pelvic girdle, one must entertain a high level of suspicion for pelvic floor dysfunction in certain patients. For example, external muscle guarding in the gluteus medius muscle can present with pain with ipsilateral side lying, be associated with greater trochanter bursitis, and present with an antalgic gait. This would likely be easily recognized by physician, physician extender, or physical therapist and addressed efficiently. If the health-care provider does not include the entire array of muscles that provide stability and mobility to the hip joint as part of the comprehensive assessment of the arthroscopic and open hip joint preservation surgery patient with persistent pain and/or impairments pre- or post successful surgery, they will fail at providing comprehensive care. As the old adage goes, overactive or hypertonic pelvic floor muscles have likely "seen you" but you "have not seen them." If one were not aware that the pelvic floor muscles' adaptation of prolonged contraction prior to surgery had perpetuated post surgery, the examiner may miss historical and physical exam findings to diagnose the pelvic floor dysfunction and offer appropriate treatment. Further, patients may not offer key historical information as they may not be aware of the link between pelvic floor symptoms and their "hip" pain. Patients with missed pelvic floor presentations often undergo additional imaging and procedures that do not shed further light on the problem and have inherent risks of radiation, medication, and other unnecessary exposures. In addition, patients presenting with certain imaging findings such as labral tears, which are sometimes asymptomatic findings, may be misdiagnosed and not receive appropriate treatment to address the primary pain generator, for example, the pelvic floor muscles, and may even undergo surgery without success in these circumstances.

Frequently, there is muscle hyperalgesia, typically proportional to the extent of the primary injury. The hyperalgesia can be associated with trophic muscle changes, including loss of muscle mass [13]. Myofascial trigger points, taut bands of muscle fibers, can develop causing local and referred pain patterns. Referred muscle pain and loss of muscle mass can compound gait abnormalities and other functional impairments, perpetuating disability.

History

To increase recognition of PFD, a more extensive review of symptoms (ROS) beyond the comprehensive orthopedic history must be obtained, querying for changes in bowel, bladder, and sexual function. These complaints provide insight into contracted pelvic floor muscles that are not relaxing properly during defecation, allowing for complete bladder emptying or causing sexual dysfunction or pain. Because of the embarrassing aspects of such complaints, it is especially important for the health-care provider to query patients about bowel, bladder, and sexual functioning symptoms directly. Reports of constipation outlasting narcotic use, painful intercourse, reduced or absent orgasm or ejaculation, inability to tolerate vaginal tampon, frequent urination, and other symptoms can be clues to hypertonic pelvic floor muscles. Patients with painful pelvic floor muscles may also report referred pain to the lower back, gluteus, groin, or lower extremities. In keeping with clinical reasoning, questions about pain onset, aggravating and relieving factors, quality and intensity, and others must be included in the complete history. Information about local patterns of hypo- or hyperalgesia can implicate associated peripheral nerve involvement.

Physical Examination

As with all orthopedic patients, the physical exam of the patient with PFD includes observation, palpation, neurological testing, joint and muscle testing, and other special tests. See Table 1 for an overview of the physical examination of the pelvic floor. The pelvic floor examination is done in addition to the complete lumbopelvic and LE examination, as directed by the patient's history.

Pelvic floor examination requires specific training that may not be part of the usual and customary training of many musculoskeletal providers. Partnering with trained pelvic floor providers such as physiatrists and physical therapists can allow for recognition and management of PFD. For interested practitioners, pelvic floor training can be pursued through professional organizations like the Women's Health Section of the American Physical Therapy Association (APTA) or the American Academy of Physical Medicine and Rehabilitation (AAPM&R). Continuing education is also offered by several national and international expert PT's in the field. Specialized EMG training is available to physiatrists and neurologists through the Bowel and Bladder Special Interest Group of the American Association of Neuromuscular and Electrodiagnostic Medicine (AANEM).

Before performing the pelvic floor examination, consent is obtained. Explaining the purpose of the examination is best done using a model of the pelvis complete with pelvic floor muscles. Most adult patients are familiar with the digital vaginal and/or rectal examination from previous experience with gynecological and prostate exams. It is important for the musculoskeletal practitioner to differentiate the focus of the pelvic floor exam to evaluate the pelvic floor musculoskeletal and neurological systems rather than organs per se.

Table 1 Physical examination of the pelvic floor

1. Obtain verbal consent
2. Inspect perineum and genitalia for scarring, defects, lesions, and skin changes; identify pelvic organ prolapse-rectocele, cystocele, etc.
3. Neurological examination
(a) Sensory exam, S2–5 light touch, pinprick
(b) Anal wink
(c) Tinel test – pudendal nerve
4. Test for allodynia
(a) Q-tip test
5. Observe pelvic floor function externally
(a) Voluntary contraction and relaxation
(b) Involuntary contraction (cough) and relaxation (Valsalva)
6. External and internal examination of superficial and deep pelvic floor muscles – performed via digital examination both vaginally (in women) and rectally (in both genders)
(a) Assess tenderness and referral pattern
(b) Assess muscle tone
(c) Assess ability to relax
(d) Assess ability to contract-lift and squeeze, "Kegel"
i. Repeated quick flick
ii. 10 s hold
7. Internal and external examination of obturator internus muscle
(a) Assess tenderness and referral pattern
(b) Assess muscle tone
(c) Assess ability to relax
(d) Assess ability to contract-ipsilateral hip abduction
8. Assess rectal tone
9. Coccyx/sacrococcygeus joint
(a) Assess for tenderness
(b) Assess for mobility
(c) Assess for deviation

Adapted from Ref. [2]

Observation

The genitalia, skin, and sphincters should be inspected for any scars, muscle or tendinous defects, skin lesions, or any signs of pelvic organ prolapse. Having the patient bear down can accentuate a rectoceles or cystoceles and give insights into connective tissue weakening.

Neurological Exam

As in the limbs or trunk, the neurological examination includes sensory, reflex and muscular strength, coordination, and endurance testing. Sensory testing includes the distribution of the bilateral pudendal nerves, S2–4, using light touch and pinprick and checking for the anal wink. A Q-tip can be used to lightly stroke the labia or penis to evaluate for hyperpathia or allodynia. If there is a history of perineal paresthesias, tapping lightly over the pudendal nerves just inferior to the ischial spines can be performed during the internal exam. Pelvic floor muscle testing will be described in the next section on muscle exam.

Pelvic Floor Muscle Exam

The examiner will then inspect the pelvic floor muscles externally as he/she cues the patient to contract (lift) then relax (lower) the pelvic muscles. Next the patient is asked first to cough and then to Valsalva while the examiner checks for the involuntary pelvic floor lift and descent, respectively. The muscle palpation examination includes external and internal examination of superficial and deep pelvic floor muscles – performed via digital examination – either vaginally (in women) and rectally (in both genders). The use of a figurative clock face may be employed to describe local areas of tenderness, with the pubic arch 12 o'clock and the coccyx at 6 o'clock. In keeping with the model, the levator ani muscle on the patient's left is at the 3–5 o'clock position while on the right at the 7–9 o'clock position. The palpation examination reveals pelvic floor muscle tenderness, ability to contract and relax, and quality and coordination of the contraction and relaxation. The levator ani lift is considered in four quadrants, left and right, both anterior and posterior, as well as with quick flicks for coordination and 10 s holds for endurance. The muscles are graded using the Modified Oxford Scale (MOS) from 0/5 for no discernible contraction to 5/5 with strong resistance felt with two fingers inserted being approximated and the contraction held for 10 s. Correlations between strength testing using the MOS and manometry are better than between MOS and ultrasound [14, 15]. The International Incontinence Society recommends a simpler four-point scale: absent, weak, normal, and strong to reflect the total behavior of tightening, lifting, and squeezing [16]. Initial and follow-up muscle testing can be used to gauge progress with pelvic floor rehab.

The muscle belly of the obturator internus muscles is found lateral to the arcus tendineus, and resisted hip abduction will result in muscle contraction beneath the examiner finger as the lateral aspect of the pelvic muscles are palpated vaginally or rectally. Other areas to palpate on the digital exam include the arcus tendineus separating the levator ani from the obturator internus, ischial spines, bladder and urethra and their fascial supports, and rectum. Joints of the pelvis and muscle attachments can also be palpated. Due to the rectovaginal septum in women, the posterior extension of the levator ani and obturator internus muscles and the coccygeus are best palpated on rectal exam. The rectal exam is done in side lying, with the patient on their left side for a right-handed examiner, with copious amounts of lubricant and slow digital insertion. The examiner can check for anal sphincter tone and note any defects in the sphincter. Along with muscle palpation for tenderness, contraction, relaxation, and endurance, the coccyx can be palpated for tenderness and alignment in addition to evaluating passive movement of the sacrococcygeus joint.

Joint Range of Motion/Special Tests

Hypertonic muscles of the pelvic floor can lead to reduced hip abduction and internal or external rotation range of motion. Likewise, the sacroiliac and sacrococcygeus joints may have reduced accessory joint movement when pelvic floor muscles maintain a contracted state or are shortened. Psoas and iliopsoas tightness may present with reduced hip extension range of motion; this tightness may be due in part of fascial restrictions mitigated by the pelvic floor muscles. Special tests of hip flexibility may be positive depending on the muscles involved.

Treatment of Pelvic Floor Pain

Due to their training and experience in treating musculoskeletal and neurological disorders, acute and chronic pain, and the associated impairments and disabilities that result, the physiatrist is in an excellent position to manage pelvic floor issues for those patients being considered for arthroscopic and open hip joint preservation procedures. Physiatric care centers around identifying functional goals and providing pain management once the pain generator has been identified. If comfortable with pelvic floor presentation and diagnosis, the orthopedic provider might refer the patient directly to pelvic floor PT.

Medications

Topical medication can be delivered via gel, liquid, or suppository, including anesthetic agents and muscle relaxants, to the vagina or rectum. Topical administration can limit side effects relative to oral dosing. Compounding pharmacists can combine medications to address hypertonicity, pain, and peripheral nociceptor upregulation. Medications prescribed should be tailored to the presumed type of pain. In a retrospective chart review of patients with high-tone PFD, women who used 10 mg valium vaginal suppositories at bedtime for 30 days, in addition to other treatments, reported subjective improvement in their PFD [17]. A more recent randomized placebo controlled subject of 21 women found that 10 mg of vaginal diazepam used nightly for 4 weeks was not associated with any change in subjective complaints on validated surveys or any change in resting EMG parameters compared with placebo suppository [18]. The authors concluded that suppository therapy alone may be insufficient in treating high-/increased-tone pelvic floor dysfunction.

Most adjunctive pain medications used after arthroscopic and open hip joint preservation surgery for patients presenting with high-tone PFD are used for off-label indications, addressing the myofascial pain. For instance, tricyclic antidepressants (TCA) or similar agents can be used topically via compounds or orally, closely monitoring for worsening constipation or urinary retention in pelvic floor patients already managing these issues. Antiepileptic medications have been used for patients with hypertonic PFD. Even though transitory side effects such as dizziness may initially be functionally limiting, a trial may prove to be helpful in achieving pain relief. Narcotics and related medications such as tramadol may be necessary for refractory pain but should be used for a predetermined period of time to limit drug tolerance and dependency. Muscle relaxants may help reduce overall muscle tone, but the sedating aspect might reduce overall activity, thereby having a detrimental impact on function. In addition, anticholinergic side effects can add to constipation and urinary retention. As with any painful condition, sleep regulation is important. Medications that initiate onset or assist in maintaining sleep are useful. Finally, patients with chronic pain can present with reactive depression that may need to be managed for full participation in the rehabilitation program.

Physical Therapy

Referral to physical therapists with specialty training through the treatment Women's Health Section of the APTA is frequently initiated and is the mainstay of hypertonic PFD. [Note: although trained through the Women's Health Section, Pelvic Floor PT's are trained to care for both men and women.] The treatment strategy is directed by identifying the type of tissue alterations that have resulted in the patient, related to somatic reflexes, sustained postures, and altered movement patterns. The role of the physiatrist or referring orthopedic provider is to provide a PT prescription summarizing the musculoskeletal findings of the history, physical examination, and diagnostic testing. Details of surgical intervention provide insight into tissue healing and aid in developing short- and long-term rehabilitation goals.

Along with identified pelvic floor muscle dysfunction, linked musculoskeletal findings in the lumbar spine, pelvic joints, and muscles must be targeted by the therapist. For example, in the arthroscopic and open hip joint preservation surgery patient, the ipsilateral psoas muscle is frequently painful and shortened due to somatic reflex contraction and altered gait patterns or maladaptive sitting or standing postures. Painful pelvic floor, hip, and lumbopelvic muscles can be treated with direct soft tissue release techniques, both externally and internally, as tolerated by the patient. If direct external or internal techniques are not well tolerated, topical lidocaine can be used prior to treatment. Soft tissue work such as mobilization has been suggested to address adhesions, diminish trigger points, and desensitize tissue [19–21]. Beyond addressing local soft tissue restrictions, physical therapy must address joint range of motion, muscle length and strength, and movement patterns related to the spine, pelvis, hip, and lower extremity [22]. Manual techniques such as scar release, myofascial release, acupressure, muscle energy, strain-counterstrain, and joint mobilization may all be indicated, based on the particular needs of the patient and skill of the practitioner.

Modalities can be used as an adjunct to support functional goals. Biofeedback can be useful in reducing the pelvic floor muscle hypertonic resting state and improve muscle firing patterns, particularly in the setting of hypertonic muscle dysfunction and dyssynergic relaxation. Electrical stimulation may facilitate pelvic floor pain control and relaxation [7]. As in trunk and extremity muscle dysfunction, ice and heat can be used locally.

Dry needling and soft tissue injections may facilitate pain reduction, in turn assisting with other therapeutic interventions and functional gains. Muscles with trigger points, especially the levator ani and obturator internus muscles, that respond only transiently to soft tissue techniques may benefit from injections of short-acting anesthetic agents, such as 1 % or 2 % lidocaine or dry needling [2]. Injections should be done in conjunction with manual techniques and PT to further benefit the patient.

Summary

This chapter has introduced the role of the pelvic floor and associated pelvic floor dysfunction that can be encountered in patients being considered for or post arthroscopic and open hip joint preservation procedures. Health-care providers for these patients should be able to evaluate and plan appropriate treatments for patient presenting with hypertonic PFD or alternatively seek out appropriate referrals. Collaborative care with physicians and physical therapists treating pelvic floor patients is invaluable in this patient population.

References

1. Maglinte DDT, Kelvin FM, Fitzgerald K, Hale DS, Benson JT. Association of compartments defects in pelvic floor dysfunction. Am J Roentgenol. 1999;172:439–44.
2. Prather H, Dugan S, Fitzgerald C, Hunt D. Review of anatomy, evaluation and treatment of musculoskeletal pelvic floor pain in women. Phys Med Rehab. 2009;1:346–58.
3. Bump RC, Norton PA. Epidemiology and natural history of pelvic floor dysfunction. Obstet Gynecol Clin North Am. 1998;25:723–46.
4. Thomas TM, Plymat KR, Blannin J, Meade TW. Prevalence of urinary incontinence. Br Med J. 1980;281:1243–5.
5. Nygaard I, Barber MD, Burgio KL, Kenton K, Meikle S, Schaffer J, Spino C, Whitehead WE, Wu J, Brody DJ. Pelvic floor disorders network. Prevalence of symptomatic pelvic floor disorders in US women. JAMA. 2008;300(11):1311–6.
6. Butrick CW. Pelvic floor hypertonic disorders: identification and management. Obstet Gynecol Clin North Am. 2009;39(3):707–22.
7. Hunt D, Clohisy J, Prather H. Acetabular labral tears of the hip in women. Phys Med Rehab Clin North Am. 2007;18:497–520.
8. Arendt-Nielsen L, Svensson P. Referred muscle pain: basic and clinical findings. Clin J Pain. 2001;17:11–9.
9. Wallace K. Female pelvic floor functions, dysfunctions, and behavioral approaches to treatment. Clin Sports Med. 1994;13(2):459–81.
10. Barber MD, Bremer RE, Thor KB, Dolber PC, Kuehl TJ, Coates KW. Innervation of the female levator ani muscles. http://www.ncbi.nlm.nih.gov/pubmed/12114890 Am J Obstet Gynecol. 2002 Jul;187 (1):64–71.
11. Ashton-Miller JA, DeLancey JO. Functional anatomy of the female pelvic floor. Ann N Y Acad Sci. 2007;1101:266–96.
12. Bump RC, Mattiasson A, Bo K, Brubaker LP, DeLancey JO, Klarskov P, Shull BL, Smith AR. The standardization of terminology of female pelvic organ prolapse and pelvic floor dysfunction. Am J Obstet Gynecol. 1996;175:10–77.
13. Galletti R, Obletter M, Giamberardino MA, Formica LM, Cicchitti G, Vicchiet L. Pain from osteoarthritis of the knee. Adv Pain Res Ther. 1990;3:183–91.

14. Isherwood PJ, Rane A. Comparative assessment of pelvic floor strength using a perineometer and digital examination. BJOG. 2000;107(8):1007–11.
15. Thompson JA, O'Sullivan PB, Briffa NK, Neumann P. Assessment of voluntary pelvic floor muscle contraction in continent and incontinent women using transperineal ultrasound, manual muscle testing and vaginal squeeze pressure measurements. Int Urogyneco J Pelvic Floor Dysfunct. 2006;17(6):624–30. Epub 2006 Mar 11.
16. Frawley H. Pelvic floor muscle strength testing. Aust J Physiother. 2006;52(4):307.
17. Rogalski MJ, Kellogg-Spadt S, Hoffmann AR, Fariello JY, Whitmore KE. Retrospective chart review of vaginal diazepam suppository use in high-tone pelvic floor dysfunction. Int Urogynecol J. 2010;21(7):895–9.
18. Crisp CC, Vaccaro CM, Estanol MV, Oakley SH, Kleeman SD, Fellner AN, Pauls RN. Intra-vaginal diazepam for high-tone pelvic floor dysfunction: a randomized placebo-controlled trial. Int Urogynecol J. 2013 May 17. [Epub ahead of print].
19. Markwell SJ. Physical therapy management of pelvi/perineal and perianal pain syndromes. World J Urol. 2001;19(3):194–9.
20. Sinaki M, Merritt JL, Stillwell GK. Tension myalgia of the pelvic floor. Mayo Clin Proc. 1977;52(11):717–22.
21. Slocumb JC. Neurological factors in chronic pelvic pain: trigger points and the abdominal pelvic pain syndrome. Am J Obstet Gynecol. 1984;149(5):536–43.
22. Prather H, Spitznagle TM, Dugan SA. Recognizing and treating pelvic pain and pelvic floor dysfunction. Phys Med Rehabil Clin N Am. 2007;18(3):477–96. ix.
23. Giambernardino M. Muscle pain, referred pain. In: Schmidt R, Gebhart G editors. Encyclopedia of pain: SpringerReference (www.springerreference.com). Springer-Verlag Berlin Heidelberg, 2013. DOI:10.1007/SpringerReference_334843 2012-07-16 14:05:41 UTC.

Coxa Saltans: Iliopsoas Snapping and Tendonitis

60

James S. Keene

Contents

J.S. Keene
Division of Sports Medicine, University of Wisconsin, Madison, WI, USA
e-mail: Keene@ortho.wisc.edu

Abstract

Painless snapping of the iliopsoas tendon occurs in 10 % of the general population during sports activities and to a lesser extent, activities of daily living. In asymptomatic individuals, no treatment is required. Painful snapping iliopsoas tendons and the associated tendinitis are common in young, physically active individuals that participate in sports that demand repeated abduction of the leg above waist level. When the snapping is symptomatic, distinguishing between the internal (snapping iliopsoas tendon) and intra-articular causes (e.g., labral tears) often is difficult because these two conditions have many of the same clinical findings. Thus, the goal of this chapter is to define the unique characteristics of a snapping iliopsoas tendon that distinguish it from the intra-articular causes of snapping through a review of the (1) pertinent anatomy, (2) clinical presentation, (3) role of the various imaging modalities, and (4) outcomes and complications of open and arthroscopic treatment of the painful, snapping iliopsoas tendon.

Introduction

Painful snapping hips are due to external, internal, and intra-articular causes [1–7]. The external and most common type is the result of the iliotibial band (i.e., the convergence site of the tensor fascia lata and gluteus maximus muscles) snapping

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_64

across the greater trochanter as the hip moves from flexion to extension and is evident on clinical examination. The intra-articular variety is due to labral tears, loose bodies, and articular cartilage flaps within the hip joint proper. However, the term "intra-articular snapping hip" is rarely used in current literature because a snapping mechanism is not really involved, and diagnosis of intra-articular pathology is now more accurate. The third type or internal snapping hip, which is the focus of this chapter, is the result of the iliopsoas tendon catching at the iliopectineal eminence [7, 8], snapping across the femoral head [9], or flipping over the iliac muscle [10] when the hip is brought from a flexed-abducted-externally rotated position into extension during athletic activities and, in some cases, during activities of daily living. Recurrent snapping of the tendon often causes a secondary iliopsoas tendinitis and bursitis and may produce labral injuries due to impingement of the tendon at the acetabular rim [11]. Distinguishing between the internal (snapping iliopsoas tendon) and intra-articular causes (e.g., labral tears) often is difficult because these two conditions have many of the same clinical findings. Thus, the goal of this chapter is to define the unique characteristics of a snapping iliopsoas tendon that distinguish it from the intra-articular causes of snapping. To accomplish this goal, the chapter will focus on the (1) pertinent anatomy, (2) clinical presentation, (3) role of the various imaging modalities, and (4) outcomes and complications of open and arthroscopic treatment of the snapping iliopsoas tendon.

Pertinent Anatomy

The psoas muscle originates from the spinous processes of the 12th thoracic and the fifth lumbar vertebrae, and the iliacus muscle originates from the anterior iliac crest and the upper twothirds of the inner table of the ilium. The iliopsoas tendon is formed from a sequential confluence of the psoas and iliacus muscle bellies at the level of the inguinal ligament [12]. However, at the inguinal ligament level where the snapping occurs, the iliac muscle remains separated from the psoas tendon and muscle, and it merges more distally with its tendon and then joins the psoas tendon to become the "iliopsoas" tendon (Fig. 1). Based on dynamic ultrasound evaluations, Deslandes et al. concluded that it is the psoas tendon trapping and then abruptly rolling over the medial part of the iliacus muscle onto the superior pubic ramus that produces the snap [10]. In normal hips, the tendon slides back smoothly medially over the iliac muscle and ends up on the pubic bone without a snap [10].

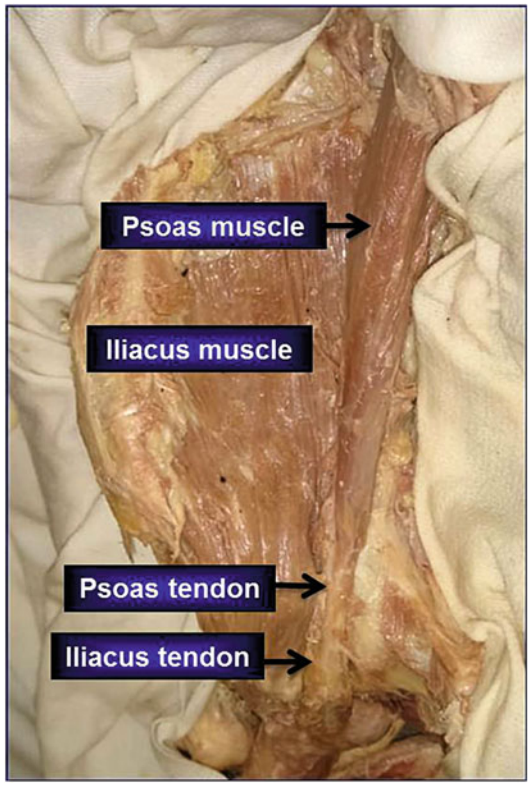


Fig. 1 The iliopsoas tendon is formed from a confluence of the psoas and iliacus muscles at the level of the inguinal ligament. However, at the inguinal ligament level where the snapping occurs, the iliac muscle remains separated from the psoas tendon and muscle and then merges more distally with its tendon and joins the psoas tendon to become the "iliopsoas" tendon

Below the level of the inguinal ligament, the iliac and psoas muscle-tendon complex, which will be referred to as the iliopsoas muscle-tendon unit (MTU), crosses anterior to the pelvic brim and hip joint capsule in a groove between the

Table 1 The average circumference of the 40 iliopsoas tendons and muscle-tendon units (MTUs) at the level of the labrum, the transcapsular release site, and the lesser trochanter

Level of measurement	Tendon average circumference (mm)	MTU average circumference (mm)	Tendon percentage of the MTU (%)
Labrum	27.1	68.3[a]	39.7[b]
Transcapsular	31.0	58.0[a]	53.4[b]
Lesser trochanter	27.5	45.7[a]	60.2[b]

[a]The average circumferences of the complete iliopsoas muscle-tendon units at the level of the labrum (68.3 mm), the capsule (57.9 mm), and the lesser trochanter (45.7 mm) were significantly different from each other ($p < 0.05$)
[b]The tendon made up a significantly higher percentage of the MTU at the transcapsular (53 %) and lesser trochanteric (60 %) levels than it did at the level of the labrum (40 %, $p < 0.001$)

iliopectineal eminence medially and the anterior inferior iliac spine laterally [12]. The iliopsoas MTU is separated from these structures by the iliopsoas bursa. The iliopsoas bursa, which is 5–6 cm long and 3–4 cm wide, extends from the level of the iliopectineal eminence to the lesser trochanter and is the largest bursa in the body.

It is important to note that the iliopsoas bursa communicates directly with the articular cavity of the hip joint in 15 % of individuals [12]. The presence of this communication should be documented by the radiologist at time of the patient's MR arthrography. This is an important finding with regard to assessing the results of diagnostic, anesthetic hip joint injections. In patients with this communication, an anesthetic hip joint injection would anesthetized both the hip joint and the iliopsoas bursa, and thus, the anesthetic hip joint injection would be of little value for determining if one or both of these structures are the source of the patient's "hip pain."

The composition of the iliopsoas muscle-tendon unit (MTU), which varies throughout its course from its origin at the level of the inguinal ligament to its insertion on the lesser trochanter, has been defined at the level of the labrum, the femoral neck, and the lesser trochanter, the three sites where arthroscopic tenotomies currently are performed. Alpert et al. found that the iliopsoas tendon makes up 44 % of the MTU at the level of the labrum, and they advocated performing tenotomies at the labral level because they believed that releasing the tendon at the lesser trochanter would release the entire iliopsoas muscle belly-tendon complex [13]. However, Alpert's study did not define the composition of the iliopsoas MTU or the percentage of the MTU that was released when the tendon was cut at the level of the femoral neck or lesser trochanter. Blomberg and associates performed a cross-sectional analysis of the iliopsoas MTU at the level of the labrum, the femoral neck, and the lesser trochanter and found that at the level of the labrum, the transcapsular (femoral neck) release site, and lesser trochanter, the iliopsoas MTU was composed of 40 % tendon/60 % muscle belly, 53 % tendon/47 % muscle belly, and 60 % tendon/40 % muscle belly, respectively (Table 1) [14]. Blomberg's results documented that there is a significant (40 %) muscular component to the iliopsoas MTU attachment to the lesser trochanter and that performing a tenotomy at that site does not release the entire muscle belly-tendon complex.

Blomberg's findings are in agreement with the results of an earlier anatomical study by Tatu et al. which noted that the most lateral fibers of the iliac muscle inserted, without any tendon, on the anterior surface of the lesser trochanter and infratrochanteric region [12]. They also identified an ilio-infratrochanteric muscular bundle that ran along the anterolateral edge of the iliacus muscle and inserted without any tendon onto the anterior surface of the lesser trochanter and the infratrochanteric region. Thus, they documented that the iliopsoas MTU had both a muscular and tendinous attachment to the lesser trochanter. It is clear from the results of these two studies that performing a tenotomy at that lesser trochanter will not release the entire muscle belly-tendon complex.

Clinical Presentation

The internal (iliopsoas) snapping hip syndrome is characterized by an audible or palpable snap in the anterior area of the hip, and it occurs without symptoms in 10 % of young active individuals. In asymptomatic individuals, no treatment is required. Symptomatic individuals with this problem typically report a painful snapping in their hip that they localize to the anterior and medial groin area. Their "hip" pain is usually exacerbated by active hip flexion and activities that require extension of the flexed, abducted, and externally rotated hip. Painful snapping iliopsoas tendons most often occur in young, physically active individuals and are common in sports that demand repeated abduction of the leg above waist level. This may be the due to the wide range of hip motion that individuals must generate in such activities as karate and ballet. Other aggravating activities include: walking, running, kicking a ball, climbing stairs, putting on socks, and rising from a chair. Patients often note that they have dull, achy anterior groin pain immediately following the "snapping" of their hip and that their pain and snapping diminish with decreased activity and rest.

On physical examination, patients with a painful, snapping tendon may keep their knee flexed during heel-strike and midstance phases of gait. The painful snapping can often be reproduced by placing the affected hip in a flexed (30°), abducted, and externally rotated position and then extending the hip to a neutral position. Patients with associated iliopsoas tendinitis will have pain with a resisted straight-leg test and tenderness to palpation of the psoas muscle-tendon complex just below the inguinal ligament [4, 6, 15–17].

Individuals with iliopsoas impingement (IPI) may or may not report snapping in their hip. Domb et al. noted that all patients with iliopsoas impingement have anterior hip pain and pain with active flexion, but most do not experience snapping sensations [11]. In these patients, physical examination reveals focal tenderness over the iliopsoas tendon, a positive impingement test, and pain with a resisted straight-leg test. However, this constellation of clinical findings is common with labral pathology and femoroacetabular impingement and is not specific to the diagnosis of iliopsoas impingement. Thus, the diagnosis of iliopsoas impingement is made at arthroscopy and is based on the findings of an isolated, anterior (3 o'clock) labral injury that is located directly adjacent to the iliopsoas tendon [11].

Imaging Studies

As previously noted, distinguishing between the internal (snapping iliopsoas tendon) and intra-articular (e.g., labral tear) causes of a snapping hip often is difficult because these two conditions have many of the same clinical findings. Thus, most patients with chronic painful snapping hips and a clinical examination consistent with either an internal or intra-articular causation have both magnetic resonance arthrography of their hip and an ultrasound evaluation of the iliopsoas tendon performed. However, standard radiographs of the involved hip should be obtained prior to ordering any ancillary imaging studies.

Standard Radiographs

All individuals with painful, snapping hips should have standard radiographs of the symptomatic hip that include an anteroposterior view of the pelvis and a cross-table or elongated-neck lateral view (hip in 90° of flexion and 20° of abduction) of the affected hip. These radiographic views may identify such intra-articular causes of snapping as loose bodies, os acetabuli, or a calcified labrum. The plain radiographs also will evaluate the patient's pelvis and proximal femur for evidence of femoroacetabular (bony) impingement, degenerative joint disease, and other bony abnormalities.

Ancillary Imaging Studies

After plain films have been completed and evaluated, magnetic resonance imaging usually is the first ancillary study performed. The details of the

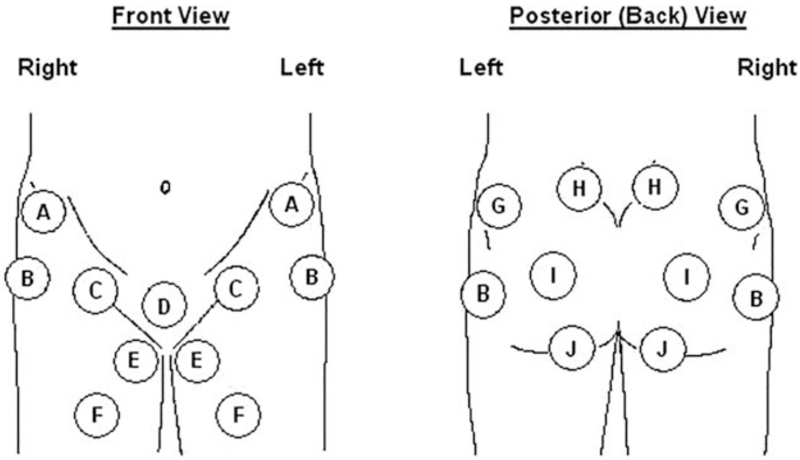


Fig. 2 The pain "circle" diagram (PCD) has circles that the patients put an "X" in to indicate if they have pain in that area. The areas include the anterior superior spine (*A*), greater trochanter (*B*), central groin (*C*), symphysis pubis (*D*), proximal inner thigh (*E*), anterior thigh (*F*), posterior iliac crest (*G*), sacroiliac joint (*H*), sciatic notch (*I*), and ischial tuberosity (*J*). The *circled areas* represent the anatomic locations associated with hip pain

author's MRI and ultrasound imaging protocols have been previously reported [4, 17] and are summarized below. Immediately preceding the MR arthrography and ultrasound-guided psoas bursa injections, patients are given a pain "circle" diagram (PCD) to complete that indicates the sites of their "hip pain." Ten to 30 min after their injection, they complete a second PCD that indicates at which sites their pain was relieved (Fig. 2) [18]. The patients are then counseled that their areas of "hip pain" that were not transiently relieved by the injections will not be relieved by hip arthroscopy. The PCD combined with the anesthetic injections of the hip joint and iliopsoas bursa has helped physicians reconcile the often unrealistic expectations of those patients with labral tears and snapping tendons who believe that hip arthroscopy also will treat all of their pelvic, buttock, and back pain.

Magnetic Resonance Imaging

Prior to imaging, the hip is injected with a standard solution of a long-acting anesthetic and contrast. MR arthrography is preferred because several studies have shown that (1) MR imaging without intra-articular contrast has a lower sensitivity and accuracy for detecting labral pathology [19–21] and (2) pain relief from an intra-articular anesthetic injection confirms that the source of pain is from within the hip joint [21–25]. MR arthrography (MRA) also is useful for detecting labral tears and other intra-articular causes of joint pain. Labral tears have been diagnosed on preoperative MRA and found during hip arthroscopy in up to 70 % of patients with a painful, snapping iliopsoas tendon [4, 17].

Ultrasound Evaluations

If a patient has no or minimal relief of their hip pain after their MRA, they subsequently (1 or more weeks after the hip arthrogram) have an ultrasound evaluation of their hip that includes static and dynamic (real-time imaging) of their psoas tendon and injection of anesthetic and steroid into their psoas bursa. Static images of the iliopsoas tendon are obtained to evaluate for tendon thickening, an enlarged bursa, and a peritendinous fluid collections. Color Doppler imaging of the iliopsoas may be performed to look for hyperemia as a sign of tendinous or peritendinous inflammation. Imaging of the asymptomatic contralateral hip should be performed to assess for any differences in appearance of the iliopsoas tendon. Studies have found that in a majority (95 %) of the cases, static images were normal. In the remaining 5 %, the static images demonstrated iliopsoas bursitis and/or tendinitis [4, 17].

Dynamic, real-time imaging of the tendon is performed because prior sonography studies have documented that an abnormal, sudden jerky motion of the iliopsoas tendon occurs in patients with a snapping hip [7, 26]. Several studies have found that in symptomatic patients, real-time imaging of the tendon will demonstrate snapping of the tendon in ~85 % of the cases [27–29]. This abnormal motion of the iliopsoas tendon can also be demonstrated radiographically by iliopsoas bursography or tenography followed by fluoroscopy [27, 28]. However, sonography is the preferred technique for examining the iliopsoas tendon because it allows both static and dynamic evaluation of the tendon [7, 29].

Following the dynamic evaluation of the iliopsoas tendon, a diagnostic, anesthetic injection of the iliopsoas bursa often is performed with the following protocol [30]. Using real-time imaging, a 22-gauge needle is passed into the iliopsoas bursa, and a 7 cc mixture of 3.5 cc of 0.5 % bupivacaine hydrochloride (Abbott Laboratories, N Chicago, IL) and 3.5 cc of 1 % lidocaine hydrochloride (Abbott Laboratories, N Chicago, IL) is injected into the bursa. Patients are asked to rate their pain level (scale 0–10) prior to the injection and immediately following the injection. If their pain is relieved, they are instructed to perform, within the next 2–4 h, the activities that previously had been painful and record their level of discomfort with each activity. These ultrasound-guided anesthetic injections will produce immediate pain relief in 89 % of patients with sonographically demonstrated snapping tendons [30]. With the addition of 1 cc of Kenalog-40 to the bupivacaine-lidocaine mixture, the injections have produced good and sustained (4 months–1 year) pain relief in a high percentage (~88 %) of patients with painful, snapping tendons [30–32]. If the ultrasound-steroid injections fail to produce sustained pain relief from the snapping of the tendon, further nonoperative treatment is pursued.

Nonoperative Treatment

Initial treatment for a painful, snapping iliopsoas tendon is rest, stretching exercises, oral anti-inflammatory medications, and focal treatment including iontophoresis and ultrasound [1, 5, 9, 33, 34]. Jacobson and Allen reported that "...stretching exercises involving hip extension for 6–8 weeks are generally successful in alleviating symptoms" [9]. Taylor and Clarke reported that 8 (36 %) of the 22 patients referred to them for a "snapping psoas" improved and did not require surgery after 6 weeks of rest and physiotherapy, which included assisted extension and ultrasound [34]. In Gruen et al.'s series of 30 patients, all patients received at least a 3-month period of stretching of the iliopsoas tendon, concentric strengthening of the hip internal/external rotators, and eccentric strengthening of the hip flexors and extensors. Nineteen patients (63 %) improved and did not require further intervention [33].

Injection of the iliopsoas bursa with anesthetic and steroids also has been advocated for treatment of this problem [28, 30, 31, 34, 35]. Wahl et al. reported on the results of bursal injections in two professional athletes who developed progressive iliopsoas tendon snapping as a result of vigorous drills and exercises. In both cases, ultrasound-guided intrabursal injections of steroids resulted in a return to football 4 weeks later without pain and long-term (26 months) resolution of the snapping [31]. Vaccaro et al. reported similar results on eight patients with painful snapping iliopsoas tendons that had cortisone injections into their psoas bursa [28]. Although 7 of the 8 patients obtained 2–8 months of pain relief from the injection, four patients eventually underwent surgery. In the largest series of bursal injections published to date, Blankenbaker et al. reported that 16 of 18 patients with painful snapping tendons had prolonged ($\geq$3 months) relief after steroid injections into their iliopsoas bursa [30]. In those cases where steroid injections and physical therapy fail to relieve painful snapping of the tendon, operative treatment often is recommended.

Operative Treatment

The traditional surgical techniques described for the treatment of painful hips due to recurrent snapping of the iliopsoas tendon have been open procedures that include either a release of the tendon [8, 34, 35] or a lengthening of the iliopsoas muscle-tendon unit [1, 5, 9, 27, 28, 33]. Complications from these procedures have been

Table 2 Comparison of the postoperative complications reported with the open surgical procedures that have been used to treat painful snapping of the iliopsoas tendon

Study (year)	No. hips	Procedure	Persistent pain	Recurrent snapping	Flexor weakness	Wound problems
Taylor (1995) [34]	16	Tendon release (medial approach)	5	6	2	0
Dobbs (2002) [3]	11	Tendon lengthening (iliofemoral incision)	1	1	0	2
Gruen (2002) [33]	12	Tendon lengthening (ilioinguinal incision)	5	0	5	0
Hoskins (2004) [5]	92	Tendon lengthening (iliofemoral incision)	6	20	3	11
Totals	**131**		**17**	**27**	**10**	**13**

Table 3 Comparison of the published results of arthroscopic iliopsoas tenotomies performed at the lesser trochanter

Study (year)	No.	Procedure	Follow-up (years)	Recurrent snapping	Results (good/exc)
Byrd (2005) [15]	12	Trochanteric release (supine position)	1	0	12
Ilizaliturri (2005) [6]	6	Trochanteric release (lateral position)	1	0	6
Flanum (2006) [4]	6	Trochanteric release (supine position)	1	0	6
Anderson (2008) [17]	15	Trochanteric release (supine position)	1	0	15
Ilizaliturri (2009) [36]	10	Trochanteric release (lateral position)	1	0	10
Ludwig (2013) [37]	60	Trochanteric release (supine position)	3	3	55
Totals	**109**			**3**	**104** (95 %)

reported to occur in 43–50 % of the patients [1, 5, 9]. The problems reported with these surgical approaches are summarized in Table 2 and include: (1) recurrent snapping of the tendon, (2) persistent hip pain, and (3) sensory nerve injuries due to the surgical incision. In Hoskins et al.'s study of 92 patients that had open fractional lengthening of their iliopsoas tendon, there were 40 complications, and 28 % of the complications were related to the incision required to perform the procedure [5]. Thus, an arthroscopic release of the iliopsoas tendon was introduced as an adjunct to hip arthroscopy for operative treatment of this problem, and to date, the results of this minimally invasive arthroscopic procedure have been excellent and better than those reported for open methods [4, 6, 15–17].

The first arthroscopic iliopsoas tendon releases were performed at the lesser trochanter, and published studies have documented that an arthroscopic release of the tendon at this level will provide long-term relief from painful snapping of the tendon [4, 6, 15, 16]. The results of arthroscopic iliopsoas tenotomies performed at the lesser trochanter are summarized in Table 3. In 2005, Byrd published two articles describing the technique of endoscopic release of the tendon [15, 16] and noted that his preliminary experience in nine cases "had been quite good." Although the length of follow-up was not stated, he reported that there was 100 % resolution of snapping and patient satisfaction, with no complications. In that same year, Ilizaliturri et al. reported on six patients who underwent an endoscopic lesser trochanteric release of the iliopsoas tendon for internal snapping hip syndrome [6]. Over the 10–27-month follow-up period, none of the patients had recurrence of their snapping symptoms. Although all patients noted a significant loss of flexion strength after surgery, their strength had improved by 8 weeks. In a subsequent publication, Flanum et al. reported that six patients who had an endoscopic iliopsoas tendon release had excellent results [4]. Their preoperative modified Harris hip scores averaged 58 points. After surgery, all patients had hip flexor weakness, used crutches for 4–5 weeks, and had 6-week scores that averaged 62 points. The patients continued to improve, and at 6 and 12 months, their scores averaged 90 and 96 points, respectively, and none had recurrence of their snapping or pain [4].

However, a major question that was not addressed in the aforementioned studies was whether athletes could return to college, high school, or recreational sports after this procedure. This question was answered by Anderson and Keene's study in 2008 [17]. They evaluated the outcomes of five competitive and ten recreational athletes that had arthroscopic iliopsoas tenotomy and concluded that a return to college, high school, and recreational sports activities can be expected after an arthroscopic release of the tendon. Although all 15 athletes could not actively flex their hip immediately following surgery, they all regained active hip flexion power by 6 weeks and were off of their crutches by 8 weeks after surgery. Six months after surgery, they had 5 out of 5 hip flexor strength on manual muscle testing, and all 15 felt their legs were of equal strength. Of the five competitive athletes, two returned to full participation in Division 1-A, college sports, and three returned to high school varsity sports. One of the two college athletes returned to soccer at her prior right defense position, 4 months after surgery, and played 90 min per game. One of the high school athletes, who had both iliopsoas tendons released, returned to be the starting catcher on her varsity softball team 9 months after her last release. All ten recreational athletes returned to their sports, which included softball, jogging, power walking, water skiing, and swimming at an average of 9 months (range 5–11 months) after their surgery [17].

The success of the arthroscopic tenotomy technique is due in part to the fact that it does not require the medial, iliofemoral, or ilioinguinal open approaches to the hip joint described above and thus avoids the wound complications inherent with their use. In addition, it is performed in conjunction with arthroscopy of the hip which permits direct examination of the joint for intra-articular pathology. Several of the aforementioned arthroscopic iliopsoas tenotomy studies have documented the coexistence of labral tears in a high percentage of the patients [4, 6, 15–17]. In Anderson and Keene's study, 12 of the 15 athletes (80 %) had labral tears, and two of these individuals also had partial tears of the ligamentum teres that were found and treated at the time of their iliopsoas release [17]. These tears would have gone undetected and untreated with open techniques.

Arthroscopic iliopsoas tenotomies (AIT's) also are done with the so-called "transcapsular" technique at the level of the femoral neck through an anterior hip capsulotomy [36, 38]. The published results of this technique are summarized in Table 4. Wettstein et al. originally described this technique in 2006 and reported resolution of the pain and snapping and no complications in the nine patients in which this procedure was performed [38].

In 2009, Ilizaliturri et al. preformed a prospective randomized clinical study that compared the results and complications of the transcapsular and lesser trochanteric arthroscopic techniques of iliopsoas release for the treatment of internal snapping hip syndrome [36]. There were 19 patients in the study; 10 had lesser trochanteric releases and 9 had transcapsular releases. Improvements in WOMAC scores were statistically significant in both groups, and there was no difference in the postoperative WOMAC scores between the groups. There were no complications in either group. They found no clinical difference in the results of both techniques and concluded that an arthroscopic iliopsoas tendon release at the level of the lesser trochanter or at the transcapsular head-neck level is effective and reproducible [36].

The only detractors from performing iliopsoas tenotomies in the peripheral hip joint at the head-neck level are the difficulties of isolating

Table 4 Comparison of the published results of transcapsular arthroscopic iliopsoas tenotomies performed in the peripheral hip at the head-neck level

Study (year)	No.	Procedure	Follow-up (years)	Recurrent snapping	Results (good/exc)
Wettstein (2006) [38]	9	Transcapsular release (supine position)	1	0	9
Byrd [39]	47	Transcapsular release (supine position)	2	0	45
Ilizaliturri (2009) [36]	9	Transcapsular release (lateral position)	1	0	9
Totals	**65**			**52**	**63** (97 %)

the tendon in obese patients and identifying both components of the tendon in those individuals with bifid tendons. As noted previously, bifid iliopsoas tendons are present in ~15 % of the general population [12], and Shu an Safran reported a case of a failed transcapsular psoas release due to not visualizing and releasing both parts of a bifid tendon. They recommended a capsulotomy of at least 1 cm when performing a release in the peripheral compartment to avoid this complication [40].

Recently, arthroscopic labral-level iliopsoas tenotomies have been recommended for the treatment of impingement and snapping of the tendon [11, 41, 42]. In 2011, Domb et al. introduced the concept of central iliopsoas tendon impingement. They noted that in addition to causing hip pain from snapping of the tendon, the iliopsoas muscle-tendon unit can cause symptomatic labral injuries due to impingement of the tendon [11]. The impingement occurs where the tendon crosses the acetabular rim, and it causes a distinct 3 o'clock labral injury at the iliopsoas notch which the authors concluded is too focal to be related to femoroacetabular impingement or dysplasia. The authors refer to this new cause of labral pathology as iliopsoas impingement (IPI) [11]. Their treatment of these labral injuries included debridement or repair of the labrum and a release of the iliopsoas tendon at the level of the labrum. They reported good results (1-year postoperative modified Harris hip scores averaged 86 points) in the 25 patients in whom these procedures were performed [13]. Although the authors noted that "some patients experienced snapping sensations" preoperatively, they did not comment on whether these sensations were relieved by the release. The authors did state that none of the patients had audible snapping of the hip preoperatively.

In 2010, Contreras et al. reported the results of a pilot study on arthroscopic treatment of a snapping iliopsoas tendon through the central compartment of the hip [41]. They released the tendon at the level of the labrum in seven patients that had painful, snapping iliopsoas tendons that were recalcitrant to five months of nonoperative treatment. They performed the release with the patient in the supine position and the hip in traction. The tendon was identified through an anterior capsulotomy and released with a radiofrequency probe. At their 12- and 24-month follow-up visits, none of the patients had recurrent snapping, and their 1- and 2-year modified Harris hip scores, which improved 32 points, averaged 88 points. There were no postoperative complications, but two patients reported no improvement in pain despite resolution of their snapping.

In 2013, Arabia et al. reported on the 1-year results of 19 patients that had arthroscopic iliopsoas tenotomies at the level of the labrum to treat a snapping hip. All of these patients also had symptomatic femoroacetabular impingement as well, and femoral head-neck osteoplasties also were performed in these patients [42]. All 19 patients had resolution of their snapping and 18 had resolution of their pain. At the time of their follow-up visits (mean 23 ± 12.9 months), hip flexion strength was 5 of 5 in all patients, including the patient who continued to have pain [42].

In 2013, Nelson and Keene reported on 30 patients that had an arthroscopic release of their iliopsoas tendon at the level of the labrum

Table 5 Comparison of the published results of transcapsular arthroscopic iliopsoas tenotomies performed at the level of the labrum

Study (year)	No.	Procedure	Follow-up (years)	Recurrent snapping	Results (good/exc)
Contreras (2010) [41]	7	Labral-level release (supine position)	1	0	7
Nelson (2012) [43]	**5**	Labral-level release (supine position)	2	3	2
Arabia (2013) [42]	19	Labral-level release (supine position)	2	0	19
Totals	**31**			**3**	**28** (90 %)

to treat iliopsoas impingement [43]. All of the patients had a tight psoas tendon that was found at arthroscopy to be impinging upon a torn or inflamed labrum. After surgery, the patients had 6-month scores that averaged 73 points and their 12-month scores that averaged 82 points (range 40–100 points). However, over the first postoperative year, three of the five patients whose preoperative ultrasound imaging demonstrated snapping of the tendon developed recurrent painful snapping of their hip. All three had iliopsoas bursa injections and experienced immediate relief of their hip pain. In two patients, the relief was temporary and an arthroscopic release of the tendon at the lesser trochanter was performed. The authors concluded that an arthroscopic release of the iliopsoas tendon at the level of the labrum is effective for alleviating hip pain from labral lesions caused by impingement of the tendon. In those patients with hip pain due to snapping of the tendon, the results of a central compartment release are less predictable, and recurrent snapping may occur in a high percentage of these patients [43]. The published results of arthroscopic iliopsoas labral-level tenotomies are summarized in Table 5.

There appear to be several reasons for the higher incidence of recurrent snapping with labral-level iliopsoas tenotomies. First are the findings of Arabia et al. who evaluated the degree of regeneration after labral-level tenotomies in 19 patients by comparing measurements of the circumferences of the psoas tendon on the preoperative and postoperative MRIs of these patients [42]. They found that tendon regeneration occurred in all patients and that mean postoperative circumference of the tendon at the site of the tenotomy was 84 % of its original circumference (55.4 ± 5.7 mm vs. 46.7 ± 6.1 mm). The authors concluded that after an arthroscopic (central compartment) iliopsoas tenotomy, the patients recover their flexion strength and have a regeneration of over 80 % of the circumference of the psoas tendon.

Second is the study of Bayer and Keene that evaluated the amount of tendon separation that occurred in 80 patients, 40 with labral-level (LL) tenotomies and 40 with lesser trochanteric (LT) tenotomies [44]. They found that there was a significant difference in the average tendon separation in the two groups. The average tendon separation in the LL patients was 8.3 ± 2.04 mm. The average tendon separation in the LT patients was 13.3 ± 3.01 mm (Fig. 3). The 0.5 cm difference in the amount of tendon separation between the two groups was statistically significant at the $p < 0.001$ level. They suggested that the difference in the amount of tendon separation that occurs at these two sites may explain the high rates of recurrent snapping (~50 %) reported with arthroscopic and open labral-level releases.

Third are the results of Blomberg et al.'s study that determined the composition of the iliopsoas muscle-tendon unit (MTU) at all three sites where arthroscopic iliopsoas tenotomies are performed [14]. They found that the diameter of the iliopsoas MTU at the level of the labrum was 68 mm and that tendon made up 40 % of the MTU (Table 1). At the level of the lesser trochanter, the diameter

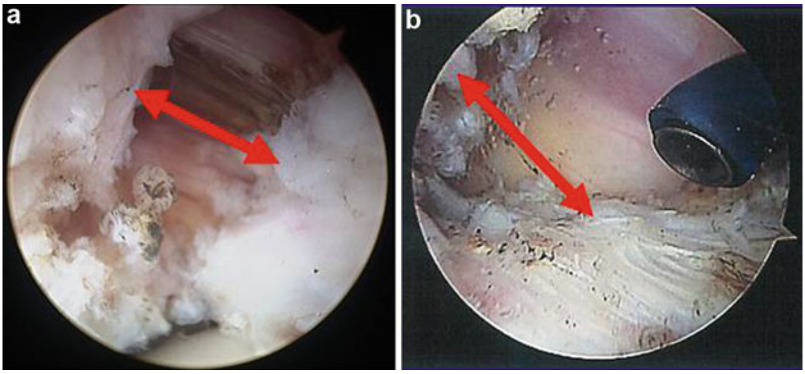


Fig. 3 (**a**, **b**) Arthroscopic views of the tendon separation that occurred after labral level (**a**) and lesser trochanteric (**b**) iliopsoas tenotomies. The *arrows* indicate where the separation measurements were made. After a labral-level tenotomy, the tendon separated an average of 0.83 cm. After a lesser trochanteric tenotomy was performed, the tendon separated an average of 1.33 cm. The 0.5 cm difference in the amount of tendon separation between the two groups was statistically significant at the $p < 0.001$ level

of the iliopsoas MTU was 46 mm, and the tendon made up 60 % of the iliopsoas MTU. This percentage is significantly higher than the 40 % the tendon made up of the MTU at the level of the labrum ($p < 0.001$). One could speculate that the 20 % increase in the amount of muscle that remains after a labral-level tenotomy may more effectively tether and limit the amount of tendon separation that occurs and was observed with a tendon release at that level [44].

Conditions That Compromise Outcomes

There are several conditions that adversely affect the outcomes of arthroscopic iliopsoas tenotomies (AITs). These include heterotopic bone formation (HO), femoral anteversion, and psoas and iliacus muscle atrophy. Heterotopic ossification is a reported complication of both open and endoscopic iliopsoas releases. In 2009, Byrd reported three cases of HO that required excision in 21 cases of lesser trochanteric tenotomies [39]. He had no cases of HO in the 34 patients in which he performed the tenotomy through a transcapsular approach in the peripheral hip joint. No other studies on arthroscopic lesser trochanteric tenotomies have reported cases of HO [4, 6, 17, 36, 37], but in all of these studies, patients were put on anti-inflammatory medications (400 mg of celecoxib daily for 21 days [36] or 800 mg. ibuprofen for 14 days [4, 17, 37]) to prevent this complication.

Excessive femoral anteversion also adversely affects the outcomes of AITs. In a recent study, Fabricant et al. found that excessive femoral anteversion (>25º) was associated with significantly lower 1-year MHH scores (77 vs. 86 points) compared to normal anteversion patients [45]. The authors concluded that (1) femoral version is a measurable prognostic factor that must now be considered; (2) preoperative evaluations of iliopsoas release patients should include radiographic measurements of femoral anteversion; and (3) patients with excessive (>25°) femoral anteversion are at risk for inferior clinical results.

Chronic psoas and iliacus muscle atrophy have also been reported. In a recent MRI study, Hahn et al. [46] documented the presence and severity of the iliopsoas muscle atrophy found in 20 patients 1–5 years after lesser trochanteric arthroscopic psoas tenotomies. Sixteen (80 %) had atrophy of both muscles, and 8 had grade 4 atrophy of one or both muscles. The average age of the patients with grade 4 atrophy (41 years) was significantly higher than the average age of those with minimal (grade 1) atrophy (26 years).

The average 1-year MHH scores of the eight patients with grade 4 atrophy was 79 points, and the average MHH scores for the six patients with grades 2–3 and the six patients with grade 1 atrophy were 84 and 89 points, respectively. The 10-point difference in the average scores of the grade 4 and grade 1 patients was due to lower scores in the categories of distance walked (6 vs. 10 points) and presence and severity of a limp (6 vs. 10 points).

In contrast to Arabia et al.'s study, the iliopsoas tendons were disrupted in 35 % of the cases, distorted (irregular/deformed) in 50 % of the cases, and normal in only 15 % of the cases. In Arabia et al.'s study which was cited previously, the authors also evaluated the degree of regeneration of the tenotomized tendons by comparing measurements of the circumferences of the psoas tendon on the preoperative and postoperative MRIs of their 19 patients [42]. They found that tendon regeneration occurred in all patients and that mean postoperative circumference of the tendon at the site of the tenotomy was 84 % of its original circumference (55.4 ± 5.7 mm vs. 46.7 ± 6.1 mm). The authors concluded that after an arthroscopic (central compartment) iliopsoas tenotomy, the patients recover their flexion strength and have a regeneration of over 80 % of the circumference of the psoas tendon [42]. The differences in the results of these studies may be due to the sites (trochanteric vs. labral level) at which the tenotomies were performed.

Some studies have speculated that iliopsoas tenotomies, specifically those performed at the lesser trochanter, will produce permanent weakness in hip flexion and not allow athletes to return to their sport [13]. However, all published studies to date have documented a full recovery of hip flexion strength [4, 6, 17, 36, 42] and a full return of all athletes to competitive and recreational sports [17].

Summary

Snapping of the iliopsoas tendon occurs without symptoms in 10 % of the general population and no treatment is required. Painful snapping iliopsoas tendons occur in young, physically active individuals that participate in sports that demand repeated abduction of the leg above waist level. Initial treatment for a painful, snapping iliopsoas tendon is rest, stretching exercises, activity modification, and oral anti-inflammatory medications, and these modalities are successful in alleviating the snapping and pain in ~50 % of the individuals with this problem. Injection of the iliopsoas bursa with anesthetic and steroids is an effective adjunct to the above methods of treatment and will provide prolonged (≥3 months) relief in up to 85 % of individuals treated in this manner. Ultrasound evaluation of the iliopsoas tendon and anesthetic injection of the psoas bursa also will confirm that snapping of the tendon is the cause of a patient's hip pain. In those cases where steroid injections and physical therapy fail to relieve painful snapping of the tendon, operative treatment is recommended. The traditional surgical techniques have been open procedures that include either a release of the tendon or a lengthening of the iliopsoas muscle-tendon unit, but complications from these procedures occur in 43–50 % of the patients in which they are performed. Thus, an arthroscopic release of the iliopsoas tendon was introduced as an adjunct to hip arthroscopy for operative treatment of this problem, and to date, the results of this minimally invasive procedure have been excellent. An arthroscopic release of the tendon is now the surgical treatment of choice because it is a safe outpatient procedure that will avoid the complications of open procedures and provide long-term relief from painful snapping of the tendon.

References

1. Allen WC, Cope R. Coxa saltans: the snapping hip revisited. J Am Acad Orthop Surg. 1995;3:303–8.
2. Brignall CG, Stainby GD. The snapping hip: treatment by Z-plasty. J Bone Joint Surg Br. 1991;73:253–4.
3. Dobbs MB, Gordon E, Luhmann SJ, et al. Surgical correction of the snapping iliopsoas tendon in adolescents. J Bone Joint Surg Am. 2002;84:420–4.
4. Flanum ME, Keene JS, Blankenbaker DG, DeSmet AA. Arthroscopic treatment of the painful "internal" snapping hip: results of a new endoscopic technique and imaging protocol. Am J Sports Med. 2007;35:770–9.

5. Hoskins JS, Burd TA, Allen WC. Surgical correction of internal coxa saltans: a 20-year consecutive study. Am J Sports Med. 2004;32:1–4.
6. Ilizaliturri Jr V, Villalobos Jr F, Chaidez P, et al. Internal snapping hip syndrome: treatment by endoscopic release of the iliopsoas tendon. Arthroscopy. 2005;21(11):1375–80.
7. Lyons JC, Peterson FAL. The snapping iliopsoas tendon. Mayo Clin Proc. 1984;59:327–9.
8. Rotini R, Spinozzi C, Ferrari A. Snapping hip: a rare form of internal etiology. Ital J Orthop Traumatol. 1991;17:283–8.
9. Jacobson T, Allen WC. Surgical correction of the snapping iliopsoas tendon. Am J Sports Med. 1990;18:470–4.
10. Deslandes M, Guillin R, Cardinal E, Hobden R, Bureau NJ. The snapping iliopsoas tendon: new mechanisms using dynamic sonography. AJR. 2008;190:576–81. doi:10.2214/AJR.07.2375.
11. Domb BG, Shindle MK, McArthur B, Boos JE, Magennis EM, Kelly BT. Iliopsoas impingement: a newly identified cause of labral pathology in the hip. HSSJ. 2011;7:145–50. doi:10.1007/s11420-011-9198-z.
12. Tatu L, Parratte B, Vuillier M, et al. Descriptive anatomy of the femoral portion of the iliopsoas muscle: anatomical basis of anterior snapping of the hip. Surg Radiol Anat. 2001;23:371–4.
13. Alpert JM, Kozanek M, Li G, Kelly BT, Asnis PD. Cross-sectional analysis of the iliopsoas tendon and its relationship to the acetabular labrum: an anatomic study. Am J Sports Med. 2009;37:1594–8.
14. Blomberg JR, Zellner BS, Keene JS. Cross-sectional analysis of iliopsoas muscle-tendon units at the sites of arthroscopic tenotomies. Am J Sports Med. 2011;39 Suppl 1:58S–63S. doi:10.1177/0363546511412162.
15. Byrd JWT. Snapping hip. Oper Tech Sports Med. 2005;13(1):46–54.
16. Byrd JWT. Evaluation and management of the snapping iliopsoas tendon. Tech Orthop. 2005;20(1):45–51.
17. Anderson SA, Keene JS. Results of arthroscopic iliopsoas tendon release in competitive and recreational athletes. Am J Sports Med. 2008;36:2363–71.
18. Arnold DR, Keene JS, Blankenbaker DG, DeSmet AA. Hip pain referral patterns in patients with labral tears: analysis based on intra-articular anesthetic injections, hip arthroscopy and a new pain "circle" diagram. Phys Sportsmed. 2011;39(1):1–7.
19. Czerny C, Hofmann S, Neuhold A, et al. Lesions of the acetabular labrum: accuracy of MR imaging and MR arthrography in detection and staging. Radiology. 1996;200:225–30.
20. Hodler J, Yu JS, Goodwin D, Haghighi P, Trudell D, Resnick D. MR Arthrography of the hip: improved imaging of the acetabular labrum with histologic correlation in cadavers. AJR. 1995;165:887–91.
21. Petersilge CA, Haque MA, Petersilge WJ, et al. Acetabular labral tears: evaluation with MR arthrography. Radiology. 1996;200:231–5.
22. Byrd JWT. Labral lesions: an elusive source of hip pain. Arthroscopy. 1996;12:603–12.
23. Byrd JWT, Jones KS. Diagnostic accuracy of clinical assessment, magnetic resonance imaging, magnetic resonance arthrography, and intra-articular injection in hip arthroscopy patients. Am J Sports Med. 2004;32:1668–74.
24. Petersilge CA. Chronic adult hip pain: MR arthrography of the hip. Radiographics. 2000;20:S43–52.
25. Petersilge CA. MR arthrography for evaluation of the acetabular labrum. Skeletal Radiol. 2001;30:423–30.
26. Cardinal E, Buckwalter KA, Capello WN, et al. US of the snapping iliopsoas tendon. Radiology. 1996;198:521–2.
27. Schaberg JE, Harper MC, Allen WC. The snapping hip syndrome. Am J Sports Med. 1984;12:361–5.
28. Vaccaro JP, Sauser DD, Beals RK. Iliopsoas bursa imaging: efficacy in depicting abnormal iliopsoas tendon motion in patients with internal snapping hip syndrome. Radiology. 1995;197:853–6.
29. Pelsser V, Cardinal E, Hobden R, Aubin B, Lafortune M. Extraarticular snapping hip: sonographic findings. Am J Roentgenol. 2001;176:67–73.
30. Blankenbaker DG, DeSmet AA, Keene JS. Sonography of the iliopsoas tendon and injection of the iliopsoas bursa for diagnosis and management of the painful snapping hip. Skel Radiol. 2006;35(8):565–71. doi:10.1007/s00256-006-0084-6.
31. Wahl CJ, Warren RF, Adler RS, Hannafin JA, Hansen B. Internal coxa saltans (snapping hip) as a result of overtraining. Am J Sports Med. 2004;32(5):1302–9.
32. Adler RS, Buly R, Ambrose R, Sculco T. Diagnostic and therapeutic use of sonography-guided iliopsoas peritendinous injections. AJR Am J Roentgenol. 2005;185:940–3.
33. Gruen GS, Scioscia TN, Lowenstein JE. The surgical treatment of internal snapping hip. Am J Sports Med. 2002;30:607–13.
34. Taylor GR, Clarke NMP. Surgical release of the "snapping iliopsoas tendon". J Bone Joint Surg Br. 1995;77:881–3.
35. Harper MC, Schaberg JE, Allen WC. Primary iliopsoas bursography in the diagnosis of disorders of the hip. Clin Orthop. 1987;221:238–41.
36. Ilizaliturri Jr VM, Chaidez C, Villegas P, et al. Prospective randomized study of 2 different techniques for endoscopic iliopsoas tendon release in the treatment of internal snapping hip syndrome. Arthroscopy. 2009;25(2):159–63.
37. Ludwig BJ, Keene JS. Long-term outcomes of arthroscopic iliopsoas tenotomies: results in 60 consecutive cases. www.ishameetings.net/meetings/2013/paper/presentations
38. Wettstein M, Jung J, Dienst M. Arthroscopic psoas tenotomy. Arthroscopy. 2006;22:907.e1–4.
39. Byrd JWT, Polkowski G, Jones KS. Endoscopic management of the snapping iliopsoas tendon. Arthroscopy Association of North American 2009 Annual meeting, San Diego, CA.

40. Shu B, Safran MR. Bifid iliopsoas tendon causing refractory internal snapping hip. Clin Orthop. 2011;469(1):289–93.
41. Contreras MEK, Dani WS, Endges WK, Araujo LCT, Berral FJ. Arthroscopic treatment of the snapping iliopsoas tendon through the central compartment of the hip. J Bone Joint Surg Br. 2010;92(6):777–80. doi:10.1302/o301-620X.9286.22797.
42. Arabia WHM, Gómez J, Serna JFL, Bohorquez BA, Barrera JMN, et al. Regrowth of the psoas tendon after arthroscopic tenotomy: a magnetic resonance imaging study. Arthroscopy. 2013;29(8):1308–13. http//dx.doi.org/10.10161/j.arthro.2013.05.002.
43. Nelson IR, Keene JS. Results of labral-level arthroscopic iliopsoas tenotomies for treatment of labral impingement. Arthroscopy. 2014;30(6):688–694.
44. Bayer JL, Keene JS. Comparison of the amount of iliopsoas tendon lengthening that occurs after arthroscopic labral-level and lesser trochanteric tenotomies. www.sportsmed.org/apps/aossm_2013/scientific_posters.html.
45. Fabricant PD, Bedi A, De La Torre K, Kelly BT. Clinical outcomes after arthroscopic psoas lengthening: the effect of femoral anteversion. Arthroscopy. 2012 (Online).
46. Hain KS, Blankenbaker DG, De Smet AA, Keene JS, Munoz del Rio A. MR appearance and clinical significance of changes in the hip muscles and iliopsoas tendon after arthroscopic iliopsoas tenotomy in symptomatic patients. HSSJ (Hospital Special Surg J). 2013;9(3):236–241.

Surgical Technique: Arthroscopic Iliopsoas Lengthening in the Central and Peripheral Compartments

61

Geoffrey S. Van Thiel and Michael Stanek

Contents

Abstract

Iliopsoas tendinitis or the "snapping hip" can be a very complex and perplexing clinical problem for the treating orthopedic surgeon. Nonoperative modalities have and will continue to be the mainstay of the treatment algorithm. However, there are a small subset of patients that continue to have functionally limiting pain despite appropriate conservative management. These patients may then be candidates for arthroscopic management of their condition. This includes an arthroscopic iliopsoas tendon release in addition to the treatment of any other presenting pathology. Arthroscopic release can be performed with two different techniques. One technique initiates the release from the central compartment with the use of traction, whereas the second technique requires no traction with the release performed from the peripheral compartment. Both techniques have been shown to have good results. The release from the central compartment is the most common method chosen, but technique can be surgeon and patient dependent.

Introduction

The surgical release of the iliopsoas tendon has been used in patients that have failed conservative management for conditions such as internal snapping hip, impingement syndrome, iliopsoas

G.S. Van Thiel (✉)
Orthopedics and Orthopedic Surgery, Rockford Orthopedic Associates, Rockford, IL, USA

Rush University Medical Center, Chicago, IL, USA
e-mail: gvanthiel@rockfordortho.com; gsvanthiel@gmail.com

M. Stanek
University of Illinois, Champaign, IL, USA
e-mail: mtstanek1@gmail.com

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_65

tendinitis, and iliopsoas tendinitis after total hip arthroplasty as well as in the setting of specific labral tears [1–6]. Multiple surgical approaches have been described for the release or recession of the iliopsoas tendon, each of which has various results and complications. Through advancements in surgical and arthroscopic techniques, the arthroscopic release of the iliopsoas tendon is now favored over the traditional open approach. This is predominantly due to the decreased complication rate as well as the ability to treat concomitant intra-articular pathologies [1, 7]. The most common technique utilizes an anterior window from the central compartment; however, the release from the peripheral compartment does have significant advantages in select patient populations.

Iliopsoas Tendinitis

Numerous causes can contribute to pathology of the iliopsoas tendon. The most commonly encountered problem is iliopsoas tendinitis with or without internal "snapping hip." This condition typically presents with an audible snap when the patient brings their hip from flexion to extension [4, 8, 9]. It can often cause significant pain; however, it is also an incidental finding in 5–10 % of the population for which the snapping occurs without symptoms [8]. Pain and functional limitations are the main symptoms that warrant treatment. This condition can also occur in the setting of total hip arthroplasty. The increase in head sizes available in contemporary hip replacement has led to an increase in iliopsoas tendon problems. Specifically, patients with a resurfacing arthroplasty can be particularly prone to developing this pathology.

History and Physical Exam Findings

The diagnosis of iliopsoas tendinitis or snapping hip is one that relies heavily on the patient's history and physical examination. Patients are typically young and active and will complain of anterior groin pain that may or may not be accompanied by an audible snap. Congenital predisposition and/or the overuse phenomenon with specific activities such as dancing, running, or cycling have all been implicated in the theoretical development of iliopsoas tendinitis [7, 8]. Patients should be asked whether or not they can voluntarily reproduce snapping. Included in the thorough history should be the specific events causing symptoms such as athletic activities, climbing stairs, rising from a seated position, etc.

The physical exam begins with taking the hip through a full range of flexion and extension to see what movements elicit the symptoms. A more specific test includes bringing the hip from a flexed, abducted, and externally rotated position to full extension with internal rotation. This can cause pain or "popping." Applying pressure over the iliopsoas tendon during this maneuver can be used as an apprehension test if a palpable snap or pain is appreciated. The pressure applied will hinder the tendons ability to snap and can aid in your diagnosis [8].

The iliopsoas tendon may also be implicated as a cause of persistent hip pain following total hip arthroplasty. Patients will report months of anterior groin pain refractory to conservative measures. Pain is often present with hip flexion activities such as climbing stairs and rising from a seated position. An audible snap is rarely appreciated in this condition. Tenderness to palpation over the anterior groin especially with resisted hip flexion is the most common finding on physical exam [3, 5].

Lastly, a study by Domb et al. attributed the iliopsoas tendon to a unique labral pathology located in the direct anterior (3 o'clock) position (Fig. 1) [1]. History in these patients includes painful hip flexion with or without snapping. Physical examination reflects findings seen with femoroacetabular impingement. However, patients will have pain on flexion, adduction, and internal rotation (impingement test) or a resisted straight leg raise. Although rare, iliopsoas impingement should still be considered when labral lesions are found outside of their normal anterior superior location.

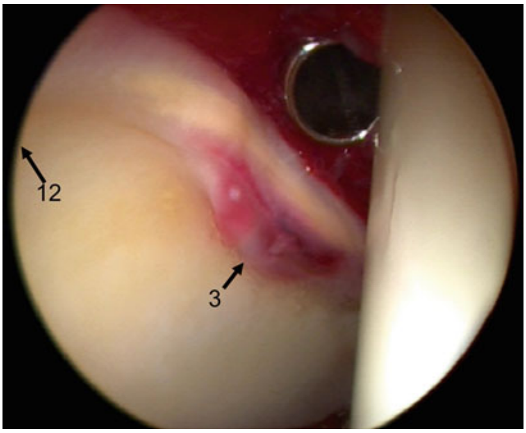


Fig. 1 Arthroscopic view of the hip joint from the anterolateral portal. The cannula is in the anterior portal. At the 3 o'clock position on the acetabulum, significant inflammation and an unstable labrum are observed. The iliopsoas crosses behind the labrum at the 3 o'clock position

Imaging

Most studies report that diagnostic imaging provides little insight into the diagnosis of iliopsoas pathology. Despite this, X-rays, ultrasounds, and MRIs are frequently ordered to rule out affiliated hip pathology [4]. Radiographs are indispensible and can exclude osteoarthritis, impingement lesions, AVN, as well as other pathologies. The use of static/dynamic sonography of the iliopsoas tendon as well as bursography or tenography has been historically reported with various results. Presently, dynamic sonography has replaced both bursography and tenography. The patient's ability to reproduce the snapping may be correlated to the frequency of pathologic findings on sonographic evaluation [10]. In addition to being cost-effective and noninvasive, sonography can be both diagnostic and therapeutic if an injection is used to alleviate symptoms. Much like its use in other aspects of medicine, results are heavily weighted on the experience of the examiner. Although MRI is very nonspecific for cases involving iliopsoas tendinitis, it is frequently utilized by clinicians due to the high incidence of associated intra-articular injuries [2].

Indications for Arthroscopic Release

Indications for iliopsoas release include tendinitis or "snapping" with pain that has failed conservative treatment and possibly atypical anterior labral pathology with significant inflammation at the anterior notch of the acetabulum in approximately the 2–3 o'clock position [1, 3, 5, 8, 11–13]. Patients should have failed injections and physical therapy before surgical release is indicated.

Surgical Techniques

Release from the Central Compartment

Arthroscopic iliopsoas tendon release from the central compartment is the most common method utilized. It can be performed as a component to a standard arthroscopic procedure. Traction is placed across the hip joint, and fluoroscopy is used to ensure that there is approximately 1 cm of distraction. A standard anterolateral portal, as described by Byrd, is established under spinal needle localization at the anterior superior corner of the greater trochanter (arthroscopic evaluation and portal placement is discussed in other chapters). A 70° scope is inserted into this portal and the anterior hip capsule/triangle is visualized. A standard anterior portal is created under spinal needle localization. After completion of a standard transportal capsulotomy, the iliopsoas tendon is exposed. This is completed by extending the anterior capsulotomy to the 2–3 o'clock location on the acetabulum from the anterior portal with visualization through the anterolateral portal (Fig. 2). There is often a notch in the acetabulum/labrum at this position that corresponds to the location of the iliopsoas tendon. There is also a definitive distinction between the hip capsule and the shiny white longitudinal fibers of the iliopsoas. A knife can be used for the capsulotomy; however, it can occasionally be difficult to achieve the appropriate angle on the capsule at the 3 o'clock position. In these cases, a radiofrequency device can be used to release the capsule. With the iliopsoas now exposed

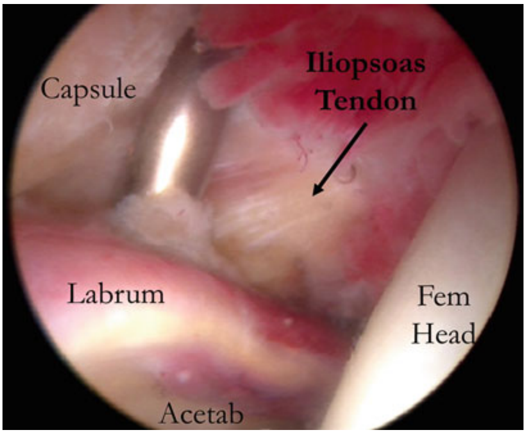


Fig. 2 Arthroscopic view from the anterolateral portal. The capsulotomy was extended to the 3 o'clock position on the acetabulum, and the iliopsoas tendon has been exposed

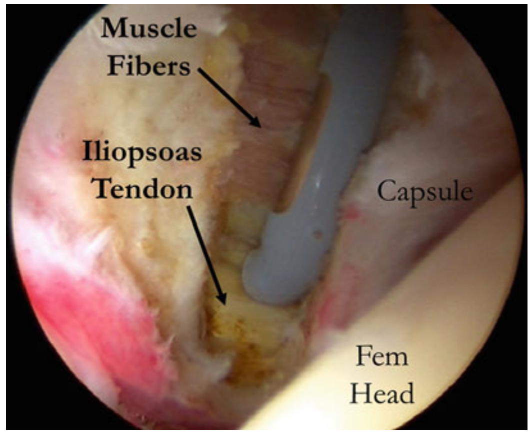


Fig. 4 Arthroscopic view from the anterolateral portal with the RF device in the anterior portal. The iliopsoas tendon is being released with the exposure of the muscle portion of the iliopsoas behind in

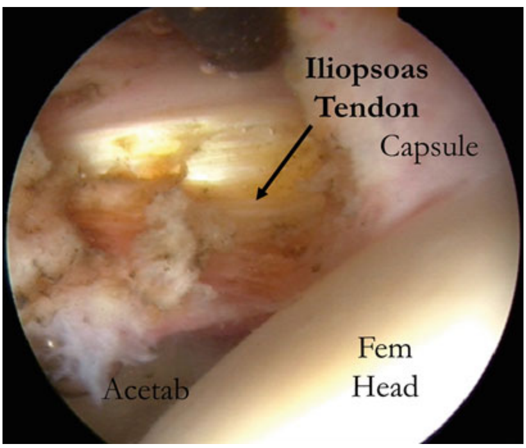


Fig. 3 Arthroscopic view from the anterolateral portal. The iliopsoas tendon has been exposed, and the release is initiated with an RF device from the anterior portal (*black* instrument)

(Fig. 3), the radiofrequency device can be used to release the iliopsoas fibers layer by layer. The release is completed once the muscular portion of the iliopsoas is visualized (Fig. 4).

Release from the Peripheral Compartment

Arthroscopic iliopsoas tendon release from the peripheral compartment can be completed without the use of traction (can also be done at the end of a standard arthroscopic procedure). This technique is a good option for the release of the iliopsoas tendon in the setting of a hip arthroplasty. A 70° or a 30° arthroscope can be used. The hip is flexed 30° and slightly externally rotated. The anterolateral portal can often be utilized to access the peripheral compartment at the anterior inferior femoral neck. However, occasionally an accessory portal is required that is slightly anterior and approximately 2 cm distal. Fluoroscopy is used to direct the spinal needle to the appropriate position at the anterior/inferior femoral neck. The cannulated trocar is then inserted. In a native hip, peripheral compartment landmarks can be visualized. These include the medial synovial fold (which represents the 6 o'clock position on the femoral neck), zona orbicularis, and the femoral head/neck junction (Fig. 5). However, in the setting of a hip arthroplasty, these landmarks will be obscured secondary to scar tissue. In these cases, the medial collar of the prosthesis can serve as a useful landmark. In the release from the peripheral compartment, a mid-anterior portal (distal and lateral compared to a standard anterior portal) is established (Fig. 6). It is more functional and allows appropriate utilization of instruments. Once visualization is achieved, a capsulotomy just proximal to the zona orbicularis in the anterior

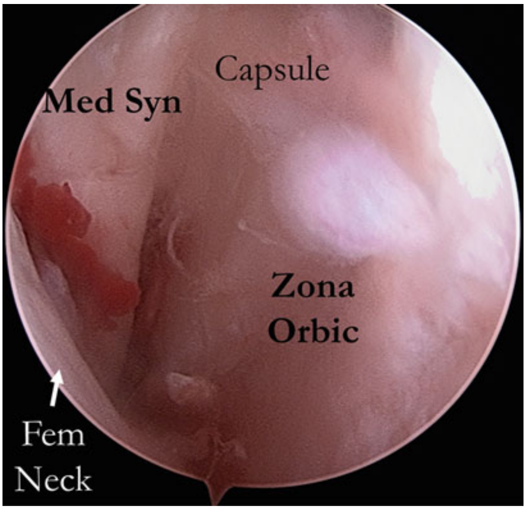


Fig. 5 Arthroscopic view from the anterolateral portal into the peripheral compartment of the hip. The femoral neck (fem neck), medial synovial fold (med syn), and the zona orbicularis (zona orbic) can be identified

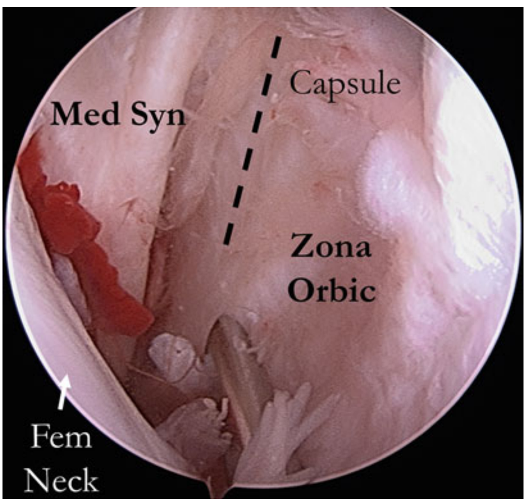


Fig. 6 Arthroscopic view of the peripheral compartment. The spinal needle is localizing an appropriate new anterior portal that is more distal. The *dotted line* represents the location of the capsulotomy for exposure and release of the iliopsoas from the peripheral compartment

aspect of the joint will reveal the iliopsoas tendon (Fig. 6). In the setting of hip arthroplasty, access can be more difficult. The capsule is often scarred to the prosthesis and distension with a spinal needle can be difficult. In these cases it is best to use fluoroscopy to locate the head/neck junction.

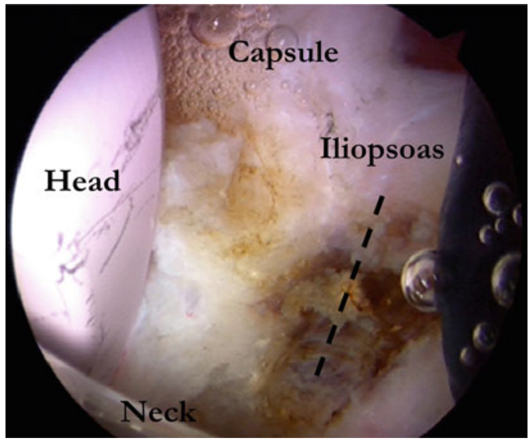


Fig. 7 Arthroscopic view of the peripheral compartment in the total hip arthroplasty. No traction is applied to the hip. The femoral head and neck are visualized, and a capsulotomy has been completed along the *dotted line* to expose the iliopsoas tendon

Use the trocar to probe this area under fluoroscopic guidance. Once palpation and X-ray confirm the appropriate location, insert the scope extracapsular. Use triangulation and a spinal needle to establish an additional anterior portal. The shaver can then clear some of the adhesions from the extra-articular portion of the capsule. An RF device can then be used to create a portal or a small capsulotomy in the anterior capsule. The scope can then follow this path into the peripheral compartment (Fig. 7). The first landmark exposed is the femoral neck. The iliopsoas can be difficult to identify in these cases secondary to significant fraying and adherence to the capsule. Once it is visualized and palpated (the iliopsoas will have increased tension across it comparted to capsular tissue), it can then be released with the use of a radiofrequency device again exposing the muscle layer behind it (Figs. 8 and 9).

Postoperative Rehabilitation

Due to decreased flexion strength frequently reported up to 8 weeks post-op, the rehab protocol as well as timeline for specific activity goals should be discussed in detail prior to surgery. Patients are allowed a free range of motion

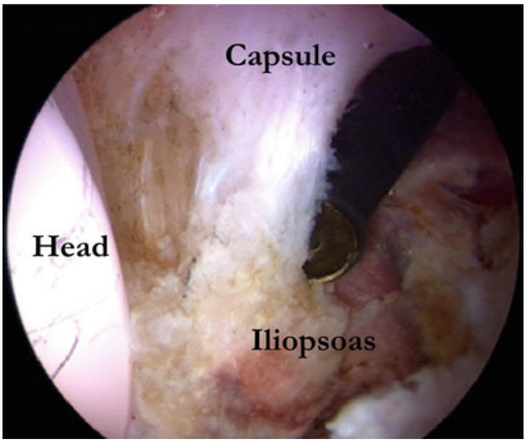


Fig. 8 Arthroscopic view showing the release of the iliopsoas from the peripheral compartment using a flexible RF device

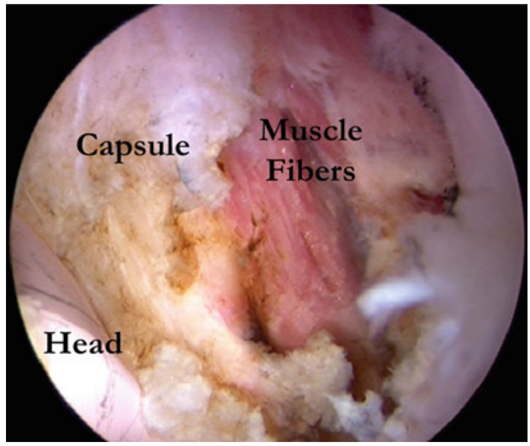


Fig. 9 Arthroscopic view showing complete release of the iliopsoas tendon with exposure of the iliopsoas muscle fibers

following iliopsoas release. Isometric contractions of the hip, hamstring, and quad muscles should be started as early as post-op day 1. Active assist ranges of motion as well as strengthening exercises are introduced in a stepwise fashion beginning in week 3. Partial weight bearing using crutches or other assist devices is continued until active hip flexion is obtained, usually in weeks 2–3. Active flexion is obtained when a patient is able to ambulate pain-free without a limp as well as climb stairs or rise from a chair without support. The rehabilitation is complete when a patient has returned to his or her functional baseline or participation in sporting activities [9, 11, 14].

Results

Dating back to 1951, the first cases describing iliopsoas release for the surgical treatment of the snapping hip can be attributed to Nunziata and Blumenfeld [15]. Historically, open approaches using either release or lengthening of the tendon have been described, all of which have shown to improve symptoms [8, 12, 16, 17]. Similar results have also been published with the use of arthroscopic techniques. The superiority of this approach can be seen when the results of recurrent snapping, post-op complications, and the time it takes to return to baseline functional activates are compared with open approaches. Byrd et al. reported 100 % resolution of symptoms along with patient satisfaction and no complications in nine total patients [8]. Ilizaliturri et al. have also reported equivalent results in early studies on endoscopic techniques. All seven patients in their study were relieved of symptoms with no recurrence. Muscle weakness, however, was reported when tested at 6–8 weeks. Half of their patients were also reported to have intra-articular pathology [18]. The ability to simultaneously identify and treat intra-articular pathologies is yet another advantage with the arthroscopic approach.

Even when open techniques were the preferred approach, much debate revolved around the specific location to perform the tenotomy. This debate still continues with the arthroscopic approach. However, recent studies have helped to shed additional light on this subject. In a study by Ilizaliturri et al., they concluded there was no difference in the results of a release performed at either the lesser trochanter or the transcapsular site [14]. Furthermore, Blomberg et al. evaluated the composition of the muscle tendon unit at common release sites. The authors concluded that no significant difference exists in the composition of the transcapsular or lesser trochanteric muscle tendon unit as the tendon comprises 53 % and 60 % of the muscle tendon unit, respectively. Thus, release at either level would yield similar results such as those seen in the previous study by Ilizaliturri et al. [14, 19].

Interestingly, Márquez Arabia et al. evaluated the reformation of the iliopsoas tendon at an average of 23 months post-release from the central compartment with MRI. The authors found that tendon regeneration occurred in all patients with an average of 84 % reconstitution and full recovery of strength [20, 21]. Regardless, iliopsoas release has been shown to be an effective treatment modality in the appropriately selected patient.

Complications

The complication rate of open techniques has been reported in as many as 40–50 % of the cases and includes subjective weakness, sensory loss, as well as the need for reoperation. Arthroscopic techniques to date report no incidence of major complications such as infection, sensory loss, or the need for reoperation. What they do have in common to open techniques is the loss of subjective flexion strength that ordinarily subsides by 8 weeks post-op. Although the incidence is decreased with an arthroscopic approach, the complication still exists [8, 12–14, 18].

Less common complications, but reported nonetheless, include heterotopic bone formation as well as complete reformation of the iliopsoas tendon following a release. Massive heterotopic ossification was reported in one case just 3 months following an open approach [7]. Although relatively new, there have been no reported cases of heterotopic ossification using an arthroscopic approach [2, 4, 8, 9, 11, 14, 18]. Some researchers have suggested the use of prophylactic NSAIDS to eliminate any such complications although there is no current evidence to support this [14].

Summary

Iliopsoas release can be accomplished from both the central and the peripheral compartment. The technique is relatively straightforward. However, the decision to proceed with an iliopsoas release or recession is more complicated. Snapping hip, iliopsoas tendinitis, and specific labral tears have all been reportedly treated with an iliopsoas release with good results. However, the majority of these cases are recalcitrant that have failed conservative management. Thus, it should be noted that the first-line treatment for many of these conditions is nonoperative management. The more common approach to the release of the iliopsoas tendon is done from the central compartment at the anterior aspect of the acetabulum. The release of the tendon at this level maintains a muscular portion that continues to attach at the lesser trochanter. The theory is that there is less of a loss of strength when performed at this level. However, there is also some early research that suggests that the tendon will "regrow" regardless of where the release is performed. There is continuing research focused on the role of the iliopsoas tendon as a pain generator in the setting of anterior hip pain.

References

1. Domb BG, Shindle MK, McArthur B, Voos JE, Magennis EM, Kelly BT. Iliopsoas impingement: a newly identified cause of labral pathology in the hip. HSS J. 2011;7(2):145–50.
2. Ilizaliturri Jr VM, Camacho-Galindo J. Endoscopic treatment of snapping hips, iliotibial band, and iliopsoas tendon. Sports Med Arthrosc Rev. 2010;18 (2):120–7.
3. Jerosch J, Neuhauser C, Sokkar SM. Arthroscopic treatment of iliopsoas impingement (IPI) after total hip replacement. Arch Orthop Trauma Surg. 2013;133(10):1447–54.
4. Kroger EW, Griesser MJ, Kolovich GP, Ellis TJ. Efficacy of surgery for internal snapping hip. Int J Sports Med. 2013;34:851–5.
5. Lachiewicz PF, Kauk JR. Anterior iliopsoas impingement and tendinitis after total hip arthroplasty. J Am Acad Orthop Surg. 2009;17(6):337–44.
6. Taylor GR, Clarke NM. Surgical release of the 'snapping iliopsoas tendon'. J Bone Joint Surg Br Vol. 1995;77(6):881–3.
7. McCulloch PC, Bush-Joseph CA. Massive heterotopic ossification complicating iliopsoas tendon lengthening: a case report. Am J Sports Med. 2006;34 (12):2022–5.
8. Byrd JWT. Snapping hip. Oper Tech Sports Med. 2005;13(1):46–54.

9. Flanum ME, Keene JS, Blankenbaker DG, Desmet AA. Arthroscopic treatment of the painful "internal" snapping hip: results of a new endoscopic technique and imaging protocol. Am J Sports Med. 2007;35(5):770–9.
10. Blankenbaker DG, De Smet AA, Keene JS. Sonography of the iliopsoas tendon and injection of the iliopsoas bursa for diagnosis and management of the painful snapping hip. Skeletal Radiol. 2006;35(8):565–71.
11. Anderson SA, Keene JS. Results of arthroscopic iliopsoas tendon release in competitive and recreational athletes. Am J Sports Med. 2008;36(12):2363–71.
12. Gruen GS, Scioscia TN, Lowenstein JE. The surgical treatment of internal snapping hip. Am J Sports Med. 2002;30(4):607–13.
13. Hoskins JS, Burd TA, Allen WC. Surgical correction of internal coxa saltans: a 20-year consecutive study. Am J Sports Med. 2004;32(4):998–1001.
14. Ilizaliturri Jr VM, Chaidez C, Villegas P, Briseno A, Camacho-Galindo J. Prospective randomized study of 2 different techniques for endoscopic iliopsoas tendon release in the treatment of internal snapping hip syndrome. Arthroscopy. 2009;25(2):159–63.
15. Nunziata A, Blumenfeld I. Snapping hip; note on a variety. Prensa Med Argent. 1951;38(32):1997–2001.
16. Dobbs MB, Gordon JE, Luhmann SJ, Szymanski DA, Schoenecker PL. Surgical correction of the snapping iliopsoas tendon in adolescents. J Bone Joint Surg Am Vol. 2002;84-a(3):420–4.
17. Jacobson T, Allen WC. Surgical correction of the snapping iliopsoas tendon. Am J Sports Med. 1990;18(5): 470–4.
18. Ilizaliturri Jr VM, Villalobos Jr FE, Chaidez PA, Valero FS, Aguilera JM. Internal snapping hip syndrome: treatment by endoscopic release of the iliopsoas tendon. Arthroscopy. 2005;21(11):1375–80.
19. Blomberg JR, Zellner BS, Keene JS. Cross-sectional analysis of iliopsoas muscle-tendon units at the sites of arthroscopic tenotomies: an anatomic study. Am J Sports Med. 2011;39(Suppl):58s–63.
20. Garala K, Power RA. Iliopsoas tendon reformation after psoas tendon release. Case Rep Orthop. 2013; 2013:361087.
21. Marquez Arabia WH, Gomez-Hoyos J, Llano Serna JF, Aguilera Bohorquez B, Nossa Barrera JM, Marquez Arabia JJ, et al. Regrowth of the psoas tendon after arthroscopic tenotomy: a magnetic resonance imaging study. Arthroscopy. 2013;29(8): 1308–13.

Surgical Technique: Arthroscopic Iliopsoas Lengthening After THA

62

Ali S. Bajwa and Richard N. Villar

Contents

Abstract

A small proportion of total hip arthroplasty (THA) patients remain symptomatic with persistent groin discomfort may have an underlying iliopsoas impingement. Other causes of residual hip pain such as loosening, infection, aseptic lymphocyte-dominated vasculitis-associated lesion (ALVAL), and gross component malposition are first excluded. In patients with positive diagnostic block of iliopsoas and failure of nonoperative rehabilitation, an arthroscopic iliopsoas release and lengthening can be safely undertaken. Iliopsoas muscle-tendon complex is formed of psoas major, psoas minor (60 % of individuals), and iliacus. Relative retroversion, decreased abduction angle, and oversizing of prosthetic acetabular component may predispose the patient to iliopsoas pathology. The iliopsoas impingement may occur at the acetabular component margin or over the large prosthetic femoral head. A two-portal arthroscopic approach is employed, and iliopsoas lengthening is undertaken using radiofrequency probe either at the acetabular component margin or anterior to the prosthetic femoral head-neck junction in the peripheral compartment depending on the site of impingement. Distal release at the lesser trochanter is also feasible. Only tendinous portion of the iliopsoas muscle-tendon complex is released. A structured rehabilitation plan ensures functional recovery within 12 weeks for the majority of the patients.

A.S. Bajwa (✉)
The Villar Bajwa Practice, Impington, Cambridge, UK
e-mail: alibajwa@doctors.org.uk; alibajwa@villarbajwa.com

R.N. Villar
The Richard Villar Practice, Spire Cambridge Lea Hospital, Impington, Cambridge, UK
e-mail: rnv1000@aol.com

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_67

Introduction

Total hip arthroplasty (THA) is generally considered a very successful procedure in improving a patient's quality of life [1]. The patient satisfaction rate is reportedly quite high. However, a subset of THA patient cohort is afflicted with pain and discomfort in the groin [2]. The persistence of such symptoms can be for a variety of reasons including iliopsoas pathology [3, 4]. The iliopsoas is a principal hip flexor and contributes to hip stability. The anatomy of the iliopsoas muscle-tendon complex renders it prone to injury and impingement [5]. This can happen in a native hip joint but in particular after THA. In selected patients, this can be addressed by arthroscopic iliopsoas release or lengthening [6]. The arthroscopic surgical technique and consideration for this approach are discussed in this chapter.

Anatomical Considerations

Psoas muscle joins the iliacus at the level of the inguinal ligament to form the iliopsoas muscle-tendon complex, which inserts onto the lesser trochanter of the femur (Fig. 1). Psoas major takes origin from the lateral surfaces of T12 and L1–5 vertebral bodies including the corresponding intervertebral discs and tendinous arches [7]. The iliacus muscle originates from the iliac fossa. Psoas minor, absent in 40 % individuals, originates from the T12 rib and L1 vertebra. It lies anterior to psoas major and inserts on iliopectineal eminence [7].

The cross section of the iliopsoas at different levels delineates a higher tendon to muscle fiber ratio closer to its insertion [8]. The iliopsoas tendon-muscle complex at the level of the labrum, transcapsular iliopsoas release site in the peripheral compartment, and the level of the lesser trochanter is composed of 40 % tendon/60 % muscle belly, 53 % tendon/47 % muscle belly, and 60 % tendon/40 % muscle belly, respectively [9]. This has an implication on the site of iliopsoas tendon release or lengthening when it is planned arthroscopically.

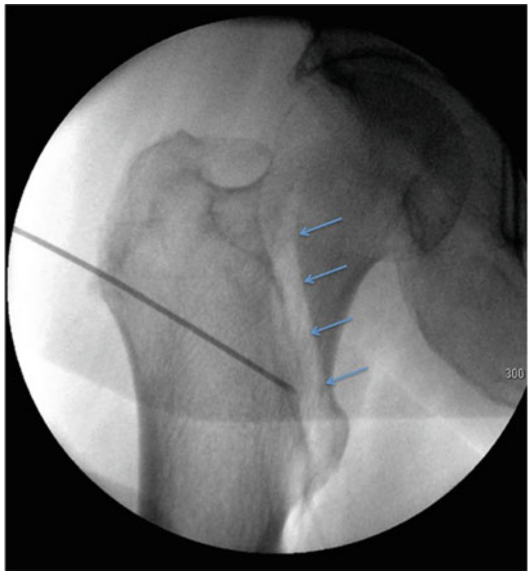


Fig. 1 An image intensifier picture of right hip joint showing the air shadow (*blue arrows*) along iliopsoas tendon course during iliopsoas air bursography (Arthroscopic image Courtesy Mr. Ali Bajwa)

Total Hip Arthroplasty and Iliopsoas Impingement

Iliopsoas impingement can occur in the native hip, the internal snapping of the hip. It has been well described as a cause of clunking and pain in the native hip joint. The presence of THA can predispose a patient to iliopsoas problems. These can result from dynamic or static issues [10]. As a result the implant position merits careful evaluation.

Static Issues

The implanted acetabular component may be relatively retroverted compared the native acetabulum. Likewise, the abduction angle may be more closed or reduced than the anatomical position. Both of the above factors bring into play the anterior and anterosuperior walls of the acetabular component, which may rub on the undersurface of the iliopsoas tendon as it glides across the hip joint [11]. Acetabular component malposition is particularly problematic in the uncemented hips, which have a metal edge on the acetabular implant and even more critical in hip resurfacing [12]. Likewise, the version of the femoral component has an impact on the position of the lesser trochanter in relation to the acetabulum, which in turn affects the mechanics of the iliopsoas tendon. The problem, as expected, is worse when position of both the components is compromised. In addition to version, the over sizing of the acetabular component or a large femoral head may increase the risk of iliopsoas impingement especially in hip extension phase of the gait [10].

Dynamic Issues

The dynamic problem of iliopsoas in association is poorly understood. Iliopsoas muscle group has predominantly red muscle fibers (slow twitch), thereby making it suitable for maintaining spinal posture [13] and controlled hip flexion [14]. Hence, if there is a deficit in the other muscle groups that contribute to posture, then iliopsoas pathology may result from overuse.

Clinical Assessment

Prior to undertaking arthroscopic surgery for iliopsoas pathology in the presence of THA, a full clinical evaluation is mandatory. It is ascertained that whether the onset of symptoms is following THA or lingering on from the preoperative period. Clinical examination typically reveals hip flexor irritability against resisted flexion but relatively pain-free passive range of motion. Occasionally, clunking or snapping can be elicited. Investigations are performed with two aims: Firstly, as a diagnostic tool to rule out implant loosening, infection, metal ion-related reaction, or referred pain and secondly, to confirm the iliopsoas pathology [11, 15]. The standard work-up will include full blood count (FBC), C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), cobalt and chromium ion levels in case of metal-on-metal articulation, dynamic ultrasound scan, metal artifact reduction sequence magnetic resonance imaging (MARS MRI), and plain radiographs in anteroposterior and cross-table lateral views. If iliopsoas pathology is visualized on imaging studies, a trial of ultrasound-guided steroid and local anesthetic is undertaken. This serves not only as a diagnostic test but occasionally is therapeutic when combined with a structured physical therapy rehabilitation program [16]. In selected cases where other causes of groin discomfort have been excluded and the diagnostic injection is positive but without sustained pain relief, an arthroscopic iliopsoas release is undertaken [15].

Arthroscopic Intervention

Arthroscopic intervention is planned under general anesthesia, intravenous antibiotic cover, and deep venous thrombosis (DVT) prophylaxis; however, the evidence for the latter is inconclusive. Based on the surgeon's experience and preference, a lateral or supine position is used [11]. The patient is placed on Maquet table with a dedicated hip arthroscopy positioning system. The use of the hip arthroscopy distractor system enables superior patient positioning and protection of the pressure areas; however, no distraction is required for this procedure. The hip to be operated upon is on top in the lateral position. The knee joint is flexed to 40° and the hip joint flexed to 35° with 15° of abduction to relax the anterior hip capsule. After careful prep and drape, per-operative image intensifier views are obtained. A spinal needle (24G) is used to access the hip from the direct lateral side, an aspirate is attempted in case of an effusion; otherwise insufflation of the hip is carried out with 20–40 ml of normal saline. Next the access portals are established. The superior portal is 3 cm superior and 2 cm anterior to the tip of the greater trochanter (GT). The needle tip is directed to the neck of the femoral component to avoid scratching of the prosthetic articulating surfaces. In the case of a resurfacing hip arthroplasty, the needle is aimed at the implant-neck junction anteriorly. After confirmation of the access needle position under radiographic control, a guide wire is inserted followed

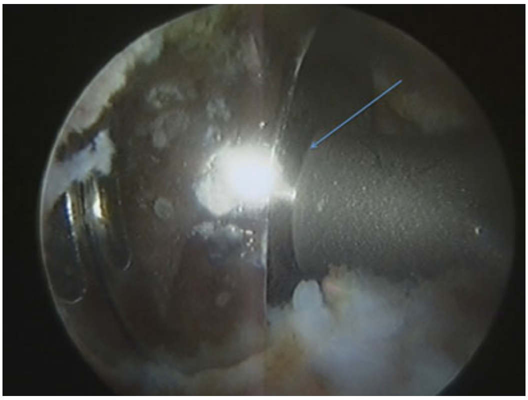


Fig. 2 An arthroscopic image of prosthetic femoral head-neck junction in THA (Arthroscopic image Courtesy Mr. Ali Bajwa)

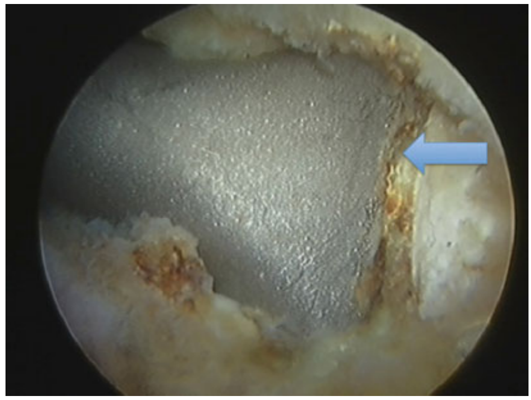


Fig. 3 An arthroscopic image showing bone-prosthesis interface (*blue arrow*) of a well-fixed THA femoral component (Arthroscopic image Courtesy Mr. Ali Bajwa)

by a 4.5 mm trocar and cannula system using Seldinger technique [17]. It requires careful visualization under image intensifier to avoid breakage of the guide wire since it may get trapped against the metal prosthesis or pushed against the scar tissue. The superior portal is used as the viewing portal. Anterolateral portal is next established 5 cm anterior and 2 cm distal to the tip of the greater trochanter. This portal is established under direct vision and 5.0 mm trocar-cannula system is used. This portal can be substituted for a distal anterolateral portal as required. An imaginary line is drawn perpendicularly distal to the anterior superior iliac spine (ASIS) and the anterolateral portal should not cross anterior to this line to avoid neurological damage [18]. Following access, the joint aspirate is collected for microscopy, culture, and sensitivity. This may be sent in aerobic and anaerobic culture bottles to decrease the risk of a false-negative result.

A radiofrequency probe is inserted from the working portal (AL) and a capsulotomy is performed. At this stage, further tissue samples are taken for histology and microbiology examination. The femoral prosthesis is examined at the head-neck (Fig. 2) interface as well as femoral stem-bone interface (Fig. 3). Acetabular component is assessed at the implant-bone interface (Fig. 4). Acetabular liner assessment is carried

Fig. 4 An arthroscopic image showing acetabular component-bone (*yellow arrow*) and femoral component-bone (*blue arrow*) interface in a resurfacing hip arthroplasty (Arthroscopic image Courtesy Mr. Richard Villar)

out at the periphery, but no attempt is made to distract the hip or examine the central part of the acetabular liner (Fig. 5). Both femoral and acetabular components can be stressed with a blunt and rigid instrument such as a Trethowan bone lever to assess for loosening. Dynamic testing is carried out to rule out any macro- or micromotion at the implant-bone interface and impingement of soft tissues on implant. In a case of typical iliopsoas impingement, the anterior capsule in the vicinity of the iliopsoas tendon shows fraying (Fig. 6) and synovitis. Occasionally the iliopsoas can be seen

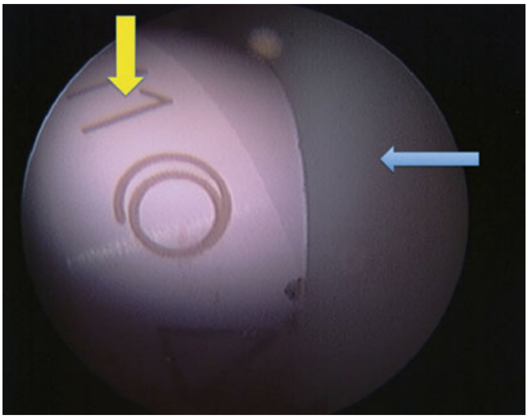


Fig. 5 Ceramic femoral head (*yellow arrow*) articulating with ultra high molecular weight polyethylene (*UHMWP*) acetabular liner (*blue arrow*) being inspected at the time of arthroscopy (Arthroscopic image Courtesy Mr. Richard Villar)

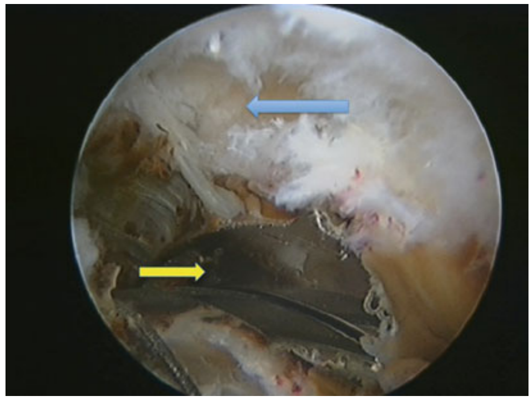


Fig. 7 Iliopsoas tendon (*blue arrow*) is seen catching on the edge of the acetabular component of resurfacing hip arthroplasty (*yellow arrow*) seen at the time of hip arthroscopy (Arthroscopic image Courtesy Mr. Ali Bajwa)

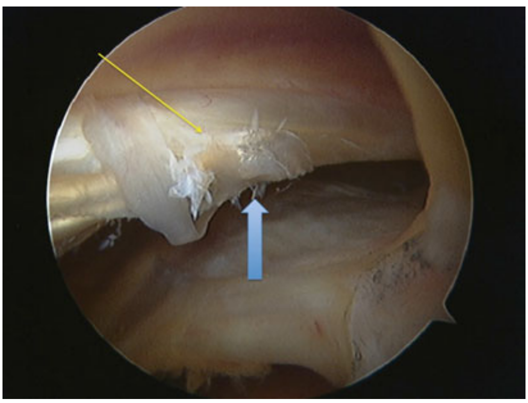


Fig. 6 An arthroscopic image of iliopsoas tendon (*yellow arrow*) with frayed undersurface (*blue arrow*) (Arthroscopic image Courtesy Mr. Ali Bajwa)

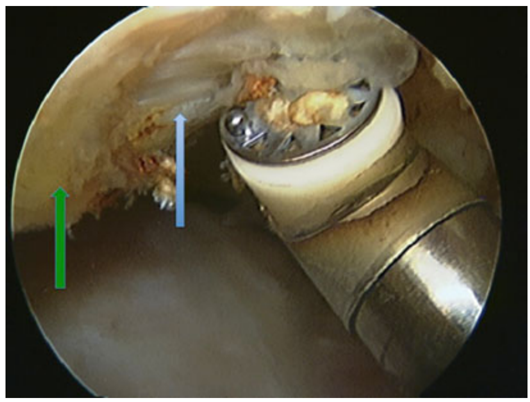


Fig. 8 Iliopsoas tendon (*blue arrow*) is being released using radiofrequency probe after making a window in the anterior hip capsule (*green arrow*) (Arthroscopic image Courtesy Mr. Ali Bajwa)

impinging on the hip prosthesis. The impingement may be at the rim of the acetabular component (Fig. 7), femoral head, or a combination. The iliopsoas course is identified both arthroscopically and with the aid of an image intensifier. If the hip arthroplasty was undertaken using a posterior approach, then a clear visualization of zona orbicularis is possible; however, medial synovial fold is often difficult to view. A window is made in the anterior capsule with a radiofrequency probe and iliopsoas tendon is identified (Fig. 8). This is carried out either at the acetabular margin or further distal depending on the site of impingement and scarring. The iliopsoas tendon is identified and carefully dissected from the often-adherent capsule. Histology samples are routinely taken (Fig. 9) and assessed for aseptic lymphocyte-dominated vasculitis-associated lesion (ALVAL) [10]. If there is a large iliopsoas bursa or significant metal debris in cases of MoM hip arthroplasty, it is cleared away (Fig. 10). Once the iliopsoas tendon is isolated, the lengthening is undertaken by releasing the tendinous part of the iliopsoas only (Fig. 11). The muscle fibers are

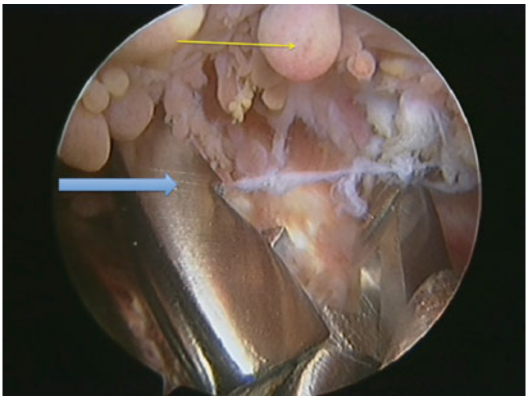


Fig. 9 Specimen of inflamed synovium (*yellow arrow*) being biopsied (*blue arrow*) for histological examination at the time of hip arthroscopy (Arthroscopic image Courtesy Mr. Richard Villar)

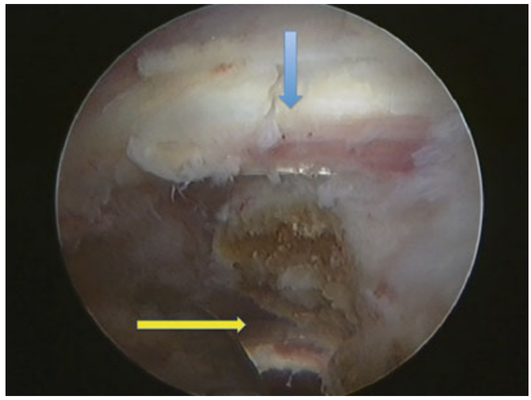


Fig. 11 Tendinous portion of the Iliopsoas tendon (*blue arrow*) is being inspected after making a window in the anterior hip capsule in front of femoral prosthetic component (*yellow arrow*) of a resurfacing hip arthroplasty (Arthroscopic image Courtesy Mr. Richard Villar)

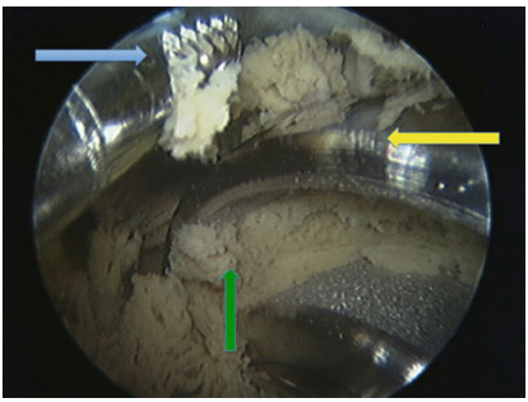


Fig. 10 Metal debris (*green arrow*) being cleared away using an arthroscopic shaver (*blue arrow*) from the vicinity of MoM THA (*yellow arrow*) (Arthroscopic image Courtesy Mr. Ali Bajwa)

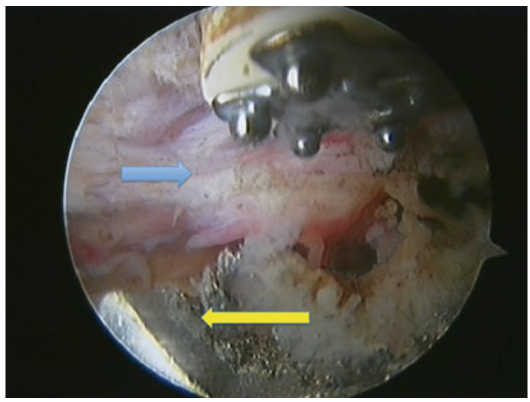


Fig. 12 Fibrosed iliopsoas tendon (*blue arrow*) impinging on the prominent anterior edge of an uncemented acetabular component (*yellow arrow*) (Arthroscopic image Courtesy Mr. Ali Bajwa)

preserved, which avoids total transection of the iliopsoas tendon-muscle complex as well as safeguards against damage to the neurovascular structures. The iliopsoas tendon release is undertaken using radiofrequency probe. Depending on the site and angle required, a 90° RF probe (Fig. 8) or a hooked RF probe can be used. The fibers are released from deep to superficial under direct vision and the extent of lengthening is noted, which ranges from 2 to 3 cm (Fig. 12). Care is taken to avoid cutting the muscle fibers in the iliopsoas muscle-tendon complex (Fig. 13). This is followed by dynamic assessment, checking the extent of clearance achieved. The opportunity is taken to document any prosthetic overhang either due to oversizing the components or malposition. This is important for further surgical planning should the arthroscopic surgery fail to resolve the whole problem.

Alternatively, iliopsoas release can be performed at the level of the lesser trochanter. This has the advantage of avoiding both iatrogenic instability secondary to capsulotomy and iatrogenic introduction of infection in the hip joint proper. The inability to access the impingement

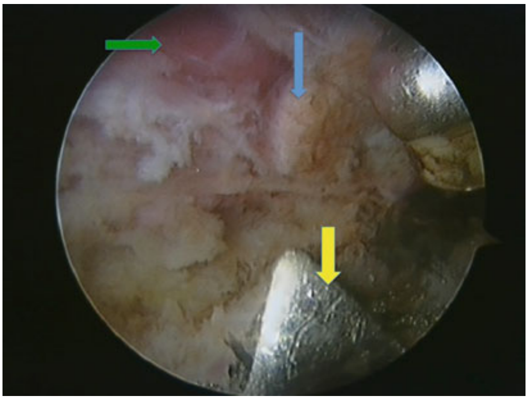


Fig. 13 Tendinous portion of the iliopsoas (*blue arrow*) being arthroscopically released while preserving the muscle fibers (*green arrow*) to achieve iliopsoas lengthening to help relieve impingement on the prominent anterior edge of uncemented acetabular component (*yellow arrow*). The alternative solution would be revision arthroplasty of the acetabular component (Arthroscopic image Courtesy Mr. Ali Bajwa)

site and the prosthetic components are potential disadvantages. This approach also precludes biopsy for histology and microbiology samples in the vicinity of the prosthesis. The optimal site of iliopsoas release, however, remains a subject for further study.

Tips and Tricks

The anterior hip capsule can be quite thick and adherent in post-arthroplasty patients. Insufflation with normal saline prior to proceeding with portal development is helpful. Consider the use of serial dilators when establishing portals. Occasionally an extracapsular approach may be required to gain safe access. Breakage of guide wire carries an extra risk in this patient group, and careful monitoring under image intensifier is needed while introducing the trocar and cannula system. In MoM hips, the polished surfaces become reflective and adjustment of light source is often required for optimal visualization. It is advisable to use switching stick and open slotted cannula while exchanging instruments to avoid losing the portals. The prophylactic antibiotics are best used after the microbiology specimens have been taken.

Postoperative Rehabilitation

This includes full weight bearing as able with crutches used only as required [19]. Full and free range of motion is permitted within the constraints of the arthroplasty in situ. Physical therapy is instituted. The aims are muscle activation and gait reeducation in the first 6 weeks. To help prevent the contractures and adhesions of the lengthened iliopsoas tendon, passive and active range of motion is encouraged as well as lying prone for 30–45 min every day. Hydrotherapy is instituted as soon as the wounds have sealed. Core stability work is commenced within 24 h after surgery while resisted hip flexor strengthening work is delayed for 6 weeks. With structured rehabilitation of patients undergoing iliopsoas lengthening, a full functional recovery is anticipated within 12 weeks postoperatively [11, 15].

Summary

Iliopsoas tendinopathy and impingement is a recognized cause of groin pain after total hip arthroplasty. There is a role for arthroscopic iliopsoas release in carefully selected cases where prosthetic components are well fixed and reasonably well positioned, infection has been ruled out, and clinically iliopsoas pathology is diagnosed and remains resistant to nonoperative measures. MARS MRI and USS provide valuable information in not only diagnosing iliopsoas pathology but also in ruling out other problems such as ALVAL. Prior to arthroscopic intervention, a trial of iliopsoas injection therapy is recommended both as a diagnostic and potentially therapeutic tool. In refractory cases, hip arthroscopy is undertaken using two-portal technique with a choice from AL, AL, and distal DAL portals. The iliopsoas lengthening is carried out by release of tendinous part while preserving the muscle fibers. The site of release is individualized and hence can be transcapsular at the level of impingement or distal at the level of lesser trochanter. Authors prefer to undertake iliopsoas release at the level of impingement.

This can be safely carried out between prosthetic acetabular margin and mid-neck region of the femoral component. This allows for assessment of prosthetic components and obtaining optimal samples for histological and microbiological analyses. Distal release at the lesser trochanter has also been reported as a viable option. The main advantages for distal release are the prevention of iatrogenic instability, reduced risk of iatrogenic infection to hip joint proper, and protection of prosthetic components from iatrogenic scratching during instrumentation. The preferred site of release thus remains a subject of further research. Rehabilitation is generally quicker than the arthroscopy of the native hip joint since no traction is used during arthroscopy of hip arthroplasty.

References

1. Santić V, Legović D, Sestan B, Jurdana H, Marinović M. Measuring improvement following total hip and knee arthroplasty using the SF-36 Health Survey. Coll Antropol. 2012;36(1):207–12.
2. Piscitelli P, Iolascon G, Innocenti M, Civinini R, Rubinacci A, Muratore M, D'Arienzo M, Leali PT, Carossino AM, Brandi ML. Painful prosthesis: approaching the patient with persistent pain following total hip and knee arthroplasty. Clin Cases Miner Bone Metab. 2013;10(2):97–110.
3. Henderson RA, Lachiewicz PF. Groin pain after replacement of the hip: aetiology, evaluation and treatment. J Bone Joint Surg Br. 2012;94(2):145–51.
4. Wuenschel M, Kunze B. Iliopsoas cyst causing persistent pain after total hip arthroplasty. Orthopedics. 2011;34(5):396.
5. Braun KF, Siebenlist S, Sandmann G, Martetschläger F, Kraus T, Ehnert S, Neumaier M, Freude T, Stöckle U. Recurrent hematomas of the iliopsoas muscle after total hip replacement as a differential diagnosis for chronic groin pain: case series report. Orthopade. 2012;41(3):212–6.
6. Van Riet A, De Schepper J, Delport HP. Arthroscopic psoas release for iliopsoas impingement after total hip replacement. Acta Orthop Belg. 2011;77(1):41–6.
7. Van Dyke JA, Holley HC, Anderson SD. Review of iliopsoas anatomy and pathology. Radiographics. 1987;7(1):54–84.
8. Chang CY, Huang AJ. MR imaging of normal hip anatomy. Magn Reson Imaging Clin N Am. 2013; 21(1):1–19.
9. Blomberg JR, Zellner BS, Keene JS. Cross-sectional analysis of iliopsoas muscle-tendon units at the sites of arthroscopic tenotomies: an anatomic study. Am J Sports Med. 2011;39(Suppl):58S–63.
10. Cobb JP, Davda K, Ahmad A, Harris SJ, Masjedi M, Hart AJ. Why large-head metal-on-metal hip replacements are painful: the anatomical basis of psoas impingement on the femoral head-neck junction. J Bone Joint Surg Br. 2011;93(7):881–5.
11. Bajwa AS, Villar RN. Arthroscopy of the hip in patients following joint replacement. J Bone Joint Surg Br. 2011;93(7):890–6.
12. Kajino Y, Kabata T, Maeda T, Iwai S, Kuroda K, Fujita K, Tsuchiya H. Strict component positioning is necessary in hip resurfacing. J Orthop Sci. 2013;18(2): 290–7.
13. Andersson E, Oddsson L, Grundstrom H, Thorstensson A. The role of the psoas and iliacus muscles for stability and movement of the lumbar spine, pelvis and hip. Scand J Med Sci Sport. 1995;5 (1):10–6.
14. Bogduk N, Pearcy M, Hadfield G. Anatomy and biomechanics of psoas major. Clin Biomech. 1992;7:109–19.
15. Jerosch J, Neuhäuser C, Sokkar SM. Arthroscopic treatment of iliopsoas impingement (IPI) after total hip replacement. Arch Orthop Trauma Surg. 2013;133(10):1447–54.
16. Byrd JW. Evaluation and management of the snapping iliopsoas tendon. Instr Course Lect. 2006;55:347–55.
17. Seldinger SI. Catheter replacement of the needle in percutaneous arteriography; a new technique. Acta Radiol. 1953;39(5):368–76.
18. Robertson WJ, Kelly BT. The safe zone for hip arthroscopy: a cadaveric assessment of central, peripheral, and lateral compartment portal placement. Arthroscopy. 2008;24(9):1019–26.
19. Jayasekera N, Aprato A, Villar RN. Are crutches required after hip arthroscopy? A case-control study. Hip Int. 2013;23(3):269–73.

Subspine Impingement and Surgical Technique

63

Lazaros A. Poultsides and Bryan T. Kelly

Contents

Abstract

Femoroacetabular impingement is one of the causes of hip pain leading to acetabular labral tears and cartilage damage via mechanical overload that may lead to the development of early osteoarthritis. Recently, other causes of impingement have been described that may be associated with the painful nonarthritic hip. Anterior inferior iliac spine (AIIS)/subspine impingement is caused by abnormal contact between the AIIS and proximal femur with straight hip flexion. Recently, a classification system of the AIIS morphology has been proposed which may provide valuable information for the preoperative surgical plan. Radiographs, magnetic resonance imaging, ultrasound, or computed tomography may help to better elucidate the problem and differentiate between intra- and extra-articular pathology. The rationale of arthroscopic subspine decompression procedure has been supported recently demonstrating favorable results. Complex AIIS morphologies combined with significant intra-articular pathology can make the arthroscopic procedure challenging. Since long-term outcomes of arthroscopic subspine decompression are still forthcoming, safety should be the first priority. This can be accomplished by following specific principles such as detailed preoperative planning by utilizing advanced imaging modalities, avoidance of long traction times, and fluoroscopic imaging intraoperatively to assure adequate and accurate AIIS and cam resection.

L.A. Poultsides (✉)
Department of Orthopaedic Surgery, Center for Hip Pain and Preservation, Hospital for Special Surgery, New York, NY, USA
e-mail: PoultsidesL@HSS.EDU

B.T. Kelly
Department of Orthopaedic Surgery, Center for Hip Preservation Surgery, Hospital for Special Surgery, New York, NY, USA

Weill Medical College of Cornell University, New York, NY, USA

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_68

Introduction

Hip pain and dysfunction in a nonarthritic joint have typically been associated with two distinct mechanical types of femoroacetabular impingement (FAI) [1]. Cam impingement can also be described as an inclusion type of injury [1–3], where a bony deformity at the femoral head–neck junction enters the joint with hip flexion [3–5]. Pincer impingement – also known as an impaction type of injury [1–3] – occurs as a result of focal or global acetabular overcoverage, causing the neck, head–neck junction, or femoral head to impact the acetabular rim, with hip flexion [1, 6–8]; symptomatic patients most commonly have features of both types of FAI [9–13].

Furthermore, nonarthritic hip joint pain has been associated either with dynamic factors resulting in abnormal contact between the femoral head and acetabular rim when the hip is in motion or with static overload stresses related to undercoverage of the femoral head in the axially loaded position [14]. Mechanical factors related to dynamic impingement include variations in anatomy such as the cam deformity [1, 11, 15, 16], lack of head–neck offset [15], increased acetabular depth or protrusio deformity [17, 18], acetabular retroversion [1, 19–22], and, at the extremes of this spectrum, slipped capital femoral epiphysis (SCFE) [2, 3] and the sequelae of childhood Perthes' disease [23]. Recently, a newly recognized cause of pincer-/impaction-type impingement has been described, presenting as an extra-articular form of FAI and occurs when a prominent anterior–inferior iliac spine (AIIS) or subspine region impinges against the anterior or inferior/medial part of the femoral neck in straight hip flexion to over 90° (subspine impingement) [6–8].

Rectus femoris AIIS avulsion injuries leading to a deformity of the AIIS have been described as the most common cause of these AIIS deformities [24–26] and specifically between the ages of 13 and 23 years, when the ratio of muscle to physeal strength is greatest [24, 25, 27, 28]. Rectus femoris anatomy and relevant biomechanics may elucidate the pathomechanics of this type of injuries. It is fusiform in shape; its superficial fibers are arranged in a bipenniform manner, whereas the deep fibers run straight down to the deep aponeurosis. It arises by two tendons: one, the anterior or straight, from the AIIS and the other, the posterior or reflected, from a groove above the rim of the acetabulum [29]. A recently published cadaveric study by Hapa et al. [30] showed that the direct head of the rectus tendon has a broad insertion on the AIIS; in 11 male cadaveric hips, the mean proximal–distal and medial–lateral distances for the rectus origin footprint were 2.2 ± 0.1 cm (range, 2.1–2.4 cm) and 1.6 ± 0.3 cm (range, 1.2–2.3 cm), respectively. In addition, on the clock face, the lateral margin (1 o'clock to 1:30 position) and medial margin (2 o'clock to 2:30 position) of the AIIS and the indirect head of the rectus (12 o'clock) were consistent for all specimens. Authors found that the AIIS typically extended further anterior and inferomedial than the rectus footprint, leaving a typical bare area devoid of tendon in this region. This footprint anatomy may have significant clinical relevance, in cases of symptomatic AIIS impingement, regarding the safe extent of subspine decompression with respect to maintaining the integrity of the origin of the direct head of the rectus femoris tendon [30]. These two tendons unite at an acute angle and spread into an aponeurosis which is prolonged downward on the anterior surface of the muscle; from this aponeurosis, the muscular fibers arise. Rectus femoris is innervated by two branches of the femoral nerve with fibers from L3 and L4. Its function is to extend the knee by lifting the lower leg. Because of the biarticular nature of rectus femoris, the associated passive insufficiency may explain the predisposition of specific sports, requiring repetitive kicking or sprinting (i.e., soccer, football, and basketball), to avulsion injuries; rectus femoris may not be able to stretch out enough to allow knee flexion while the hip is in extension. Recently, a potentially developmental – rather than an avulsion-type injury – form of AIIS deformity and subsequent subspine impingement was described and was associated with acetabular retroversion and may in fact be the most prevalent form of subspine

Fig. 1 CT AP view of type I AIIS variant in a left hip. There is a smooth ilium wall between the caudad level of the AIIS and the acetabular rim

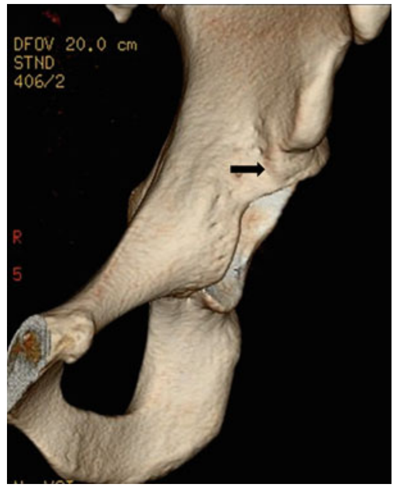


Fig. 2 CT AP view of type II AIIS variant in a left hip. Bony prominences (*black arrow*) are present on the ilium wall extending from the caudad area of the AIIS to the acetabular rim

impingement [6]. The relevant three-dimensional (3D) CT reconstruction views revealed a low-set AIIS with smooth and rounded borders extending to the level of the acetabular rim [6], as opposed to the spiky-spur appearance of the AIIS observed in another series [8].

In order to delineate these distinct etiologies contributing to AIIS impingement, a specific classification system has been proposed lately attempting to define indications and facilitate the surgical decision-making when managing this clinical entity [7]. Three-dimensional CT reconstructions of 53 hips (53 patients) with symptomatic FAI were evaluated [7] defining three morphological AIIS variants: type I when there was a smooth ilium wall between the AIIS and the acetabular rim, characterized by the lack of contribution from the AIIS to hip impingement (Fig. 1); type II when the AIIS extended to the level of the rim (Fig. 2); and type III when the AIIS extended distally to the acetabular rim (Fig. 3). Type III, and to a lesser extent type II variants, contributed to FAI, documented by the limitation in flexion and internal rotation and the bone contact seen between the AIIS and the femoral neck at terminal hip positions. In both type II and III cases, the AIIS may be considered and consequently has to be critically assessed as a potential contributor to hip impingement [7].

Fig. 3 CT AP view of type III AIIS variant in a left hip. The AIIS (*white arrow*) extends distally to the anterosuperior acetabular rim, characterized by a downward "spur appearance"

Systematic Approach to the Evaluation and Surgical Treatment

A systematic approach for each patient who presents with symptomatic FAI and possible subspine impingement should include combined information of a detailed history, an anatomically based clinical examination, and the interpretation of clinically relevant findings of all available imaging modalities. These diagnostic tools will enable the clinician to successfully make a "four-layered" – osteochondral, inert, contractile, and neuromechanical layer – diagnosis, which is essential to formulate a safe and successful treatment plan [31–33].

Clinical Presentation

The clinical presentation in patients with subspine impingement includes tenderness to palpation over the AIIS that recreates typical pain and anterior hip or groin pain with straight or prolonged hip flexion [34]. On physical examination, hip flexion range of motion is limited. Partial pain relief and persistent hip flexion limitations after intra-articular anesthetic hip injection may be observed [6]. This may be explained by the fact the AIIS deformity may not have been the single cause for the preoperative symptoms since studies have shown the concomitant presence of abnormal cam morphology [35]. Furthermore, cam lesions may have contributed to anterior impingement against the AIIS, consistent with recent studies that showed that impingement in such cases, when the hip was flexed to greater than 90°, occurred between the AIIS prominence and the anterior aspect of the femoral neck [6–8, 36]. In the presence of intra-articular FAI, the minimal relief of groin and/or anterior pain during straight hip flexion after intra-articular injection of a local anesthetic implies the coexistence of extra-articular subspine impingement [37]. Such cases combining both intra- and extra-articular deformity/impingement underscore the significant advantage of using the arthroscopic approach to decompress an abnormal AIIS, which enables the surgeon to address simultaneously all potential intra- and extra-articular sources of hip pathology.

Imaging Modalities

Radiographs may reveal calcification within the direct or indirect head of the rectus femoris origin, evidence of prior AIIS avulsion injury (Fig. 4), a prominent AIIS deformity, extending to the level of or caudad to the level of the anterior–superior acetabular rim (on AP view of the pelvis and lateral view of the femur), excessive anterior and distal extension of AIIS viewed on false-profile view, crossover sign with increased anterior acetabular rim sclerosis (on AP view of the pelvis), and impingement cysts located at the distal femoral neck (Fig. 5) [6–8, 38]. Impingement cysts in AIIS/subspine impingement are located more distal on the femoral neck than typically seen with FAI impingement [6–8, 39]. The 3D computed tomography (CT) images are invaluable tool for preoperative assessment [6–8]. The 3D surface rendering computed tomography images could delineate the shape and deformity of the AIIS facilitating the classification and consequently generating the appropriate for each patient surgical plan. They could provide the clinician with the specific location of osseous impingement and the unique pattern of FAI impingement in each patient (Figs. 1, 2, 3, 6, and 7a, b). Magnetic resonance imaging (MRI) may also demonstrate abnormalities of the AIIS or subspine area. This area should always be evaluated on hip MR examinations in addition to the cartilage, labrum, capsule, and periarticular soft tissues such as tendons, muscles, and bursae (Fig. 8) [34]. Except for radiography, CT, and MRI, advanced dynamic imaging

Fig. 4 AP view of the pelvis demonstrating calcification of the rectus femoris origin (*arrow*) in the left hip in an athlete with a history of chronic avulsion of the direct head of the rectus femoris that was treated nonoperatively

Fig. 5 Lateral view of the left hip of a patient with a history of chronic avulsion of the direct head of the rectus femoris. Prominent AIIS (*arrow*) extending caudad to the level of the anterosuperior acetabular rim, and herniation pits (*dotted arrow*) at the distal femoral neck confirm the presence of subspine impingement

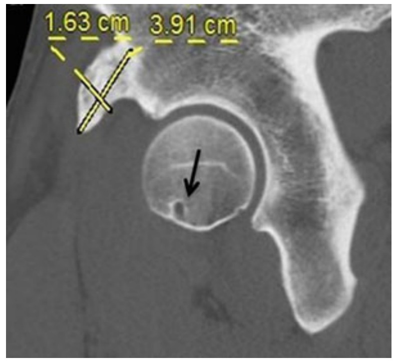


Fig. 6 CT sagittal view of a left hip (same patient with Figs. 4 and 5) showing the distal and anterior extension of the AIIS and the associated impingement cysts (*arrow*) at the distal femoral neck

modalities such as dynamic ultrasonography and three-dimensional dynamic imaging analysis could facilitate the assessment of subspine impingement. A CT-based, dynamic computer model software program (A2 Surgical, Saint-Pierred'Allevard, France) [6–8, 40] allows the execution of motion paths that may be unique for each painful hip. Reproduction of zones of proximal femoral and prominent AIIS and/or acetabular bone-to-bone contact, via straight flexion beyond 90°, offers great insights for preoperative planning and could tailor the appropriate treatment of each patient (Fig. 9a, b) [41].

Treatment: Arthroscopic Approach

Decompression of a symptomatic AIIS prominence was described in the past, through the Smith-Petersen approach, either as a single procedure [24] or after arthroscopic exploration of the joint [36], but recently, the concept of arthroscopic decompression has been popularized in several studies demonstrating satisfactory short-term results [6–8, 30]. The rationale of arthroscopic decompression of prominent AIIS is supported by short-term outcomes series showing improvement in hip function and ROM [8]. This type of extra-articular arthroscopic procedure appears to be safe given that no associated complications have been reported. Despite the proximal and possible medial dissection of the capsule, no cases of fluid extravasation into the abdomen or retroperitoneum were observed. Furthermore, no cases of postoperative hip flexion weakness, complete detachment of the direct head of rectus femoris insertion from the AIIS, or formation of heterotopic ossification (HO) have been described. The concept of arthroscopic subspine decompression in AIIS variants that extend to and below the acetabular rim is further emphasized by a recent study [7]. In a cohort where FAI patients were matched for age, femoral version, and alpha angles, a CT-based dynamic computer model revealed that these prominent types of AIIS were associated with a decrease in hip flexion and internal rotation compared to the normal AIIS variants [7].

The significant advantage of arthroscopic approach is that it allows the hip surgeon to address patients with coexisting intra- and extra-articular causes of hip pain by utilizing a single arthroscopic procedure [6–8]. Preoperative planning is of paramount importance and should include 3D CT reconstruction views to evaluate the extension of the AIIS prominence, both anteriorly and distally. Should a cam deformity be present, concomitant decompression could be performed to increase the range of straight flexion without bone-to-bone contact. Any intra-articular

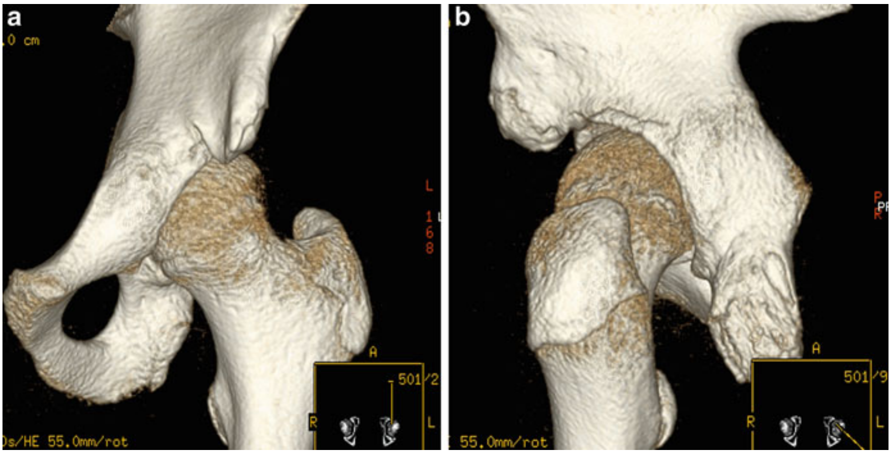


Fig. 7 **a**–**b** The prominent AIIS is well demonstrated in the 3D CT reconstruction AP view (**a**) and the "ischium AIIS" view (**b**) of this left hip before arthroscopic subspine decompression was performed

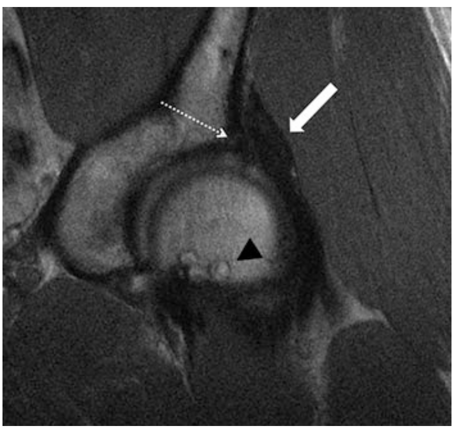


Fig. 8 MRI coronal view of a left hip (same patient with Figs. 4, 5, and 6) revealing thickening of the rectus femoris (*arrow*) secondary to chronic avulsion of the direct head of the rectus femoris that was treated nonoperatively, anterosuperior labral tear (*dotted arrow*) and associated herniation pits (*arrowheads*) at the distal femoral neck

abnormalities, such as labral or chondral injury, should be addressed as well to optimize the surgical outcome. The development of advanced and less invasive arthroscopic techniques, including extensile arthroscopic capsulotomies, has improved the central and peripheral compartment access and visualization facilitating, therefore, acetabular rim and AIIS evaluation and resection, treatment of labral and chondral injury, and osteoplasty for cam decompression.

Surgical Technique

The positioning of the patient depends on surgeon preference, but for both supine and lateral positions, the feet should be well padded, and a large perineal pad should be used to optimize distraction and to minimize traction-related complications, such as pudendal nerve injury. Adequate distraction results in approximately 10 mm of joint space opening confirmed by fluoroscopy. Majority of surgeons utilize either two or three portals depending on their preoperative plan and preference. The two most widely accepted portals are the anterolateral (lateral) and the true anterior or modified anterior portal. Additional used portals are the distal anterolateral accessory portal (DALA) and various percutaneous distal portals that facilitate the anchor placement, especially in the anterior acetabular rim, suture management, and work (femoroplasty and capsule closure) in the peripheral compartment. The aforementioned portals allow better visualization and safe access to the hip joint [42]. The anterolateral pertrochanteric portal is established first under fluoroscopic guidance, followed by the mid-anterior portal (slightly more lateral to the traditional anterior to avoid injury to the lateral femoral cutaneous nerve) under arthroscopic visualization from the lateral portal. The mid-anterior portal may be placed more distally in cases with

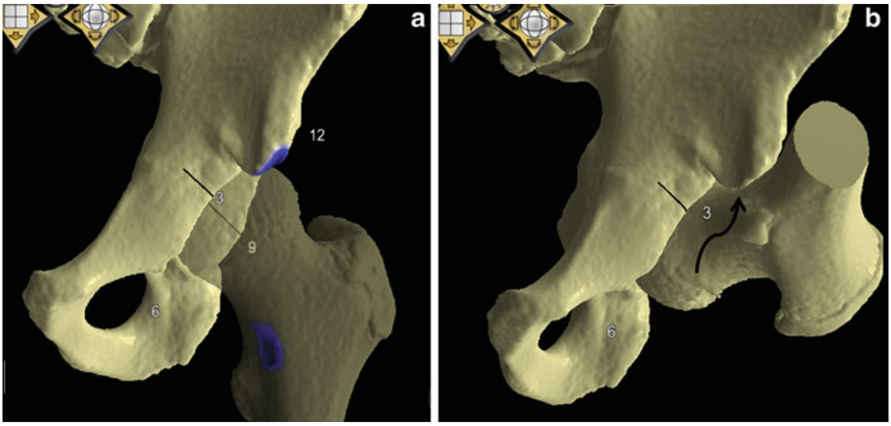


Fig. 9 **a**–**b** CT-based dynamic software images of a left hip showing areas of impingement between a type III AIIS (extending caudad to the level of the anterosuperior acetabular rim) and the distal femoral neck. (**a**) Hip in neutral position. The *blue* color highlights the area of bony impingement of the AIIS against the inferior part of the femoral neck with straight flexion. (**b**) Hip in 112° of flexion. The *curved arrow* illustrates the area of AIIS impingement

AIIS impingement in order to facilitate access to the anterior portion of the joint. Care should be taken to keep minimum 6–7 cm between the portals, which will allow sufficient working space between instruments. Having established the lateral and mid-anterior portals, based on each patient's individual bone anatomy, a diagnostic arthroscopy is performed to evaluate the labrum, capsule, femoral head and acetabular cartilage, and ligamentum teres (Fig. 10a). The interpretation and correlation of intraoperative findings with the clinical examination and imaging findings will confirm the layered diagnosis and enable the surgeon to follow the preoperative surgical plan. Afterwards, the interportal cut is performed, connecting the anterolateral with the mid-anterior portal. This capsular cut will improve the visualization and enable the surgeon to work on the acetabular rim and subspine area. It should be limited only to the area of labral injury because unnecessary capsular cutting beyond the two portals may lead to postoperative capsular instability, especially in the setting of static overload such as acetabular undercoverage, increased femoral or acetabular version, femoral valgus, and dynamic instability [43, 44].

Based on preoperative imaging and intraoperative visualization, the margins of the AIIS abnormality and associated capsular-sided labral damage are defined. Depending on the distal and anterior extension of the AIIS in relation to the acetabular rim, the degree of labral damage and capsular-sided erythema may vary in severity. The capsule is then elevated off in this area using both low-energy radiofrequency ablation and motorized shavers bur (extra-long 5.5-mm full radius), but care is taken not to primarily detach the labrum from the rim. A flexible radiofrequency probe may be helpful to dissect the capsule around the AIIS and in the area of acetabular rim if focal overcoverage coexists (Fig. 10b). After the capsule has been elevated, and the AIIS is fully exposed, subspine decompression can be performed utilizing motorized burs (5.5 mm in diameter) (Fig. 10c). Resection of the prominent AIIS can be confirmed both arthroscopically and under fluoroscopic imaging (Figs. 10d, 11). Over-resection proximally should be avoided in order not to endanger the insertion of the direct head of rectus femoris; if it is significantly destabilized, reattachment may be required although this complication has not been reported in the literature to date. If the prominence of the AIIS extends medially as well, decompression of the medial border should be performed especially if there are clinical, radiological, and intraoperative findings of symptomatic psoas impingement against the AIIS. The medial portion

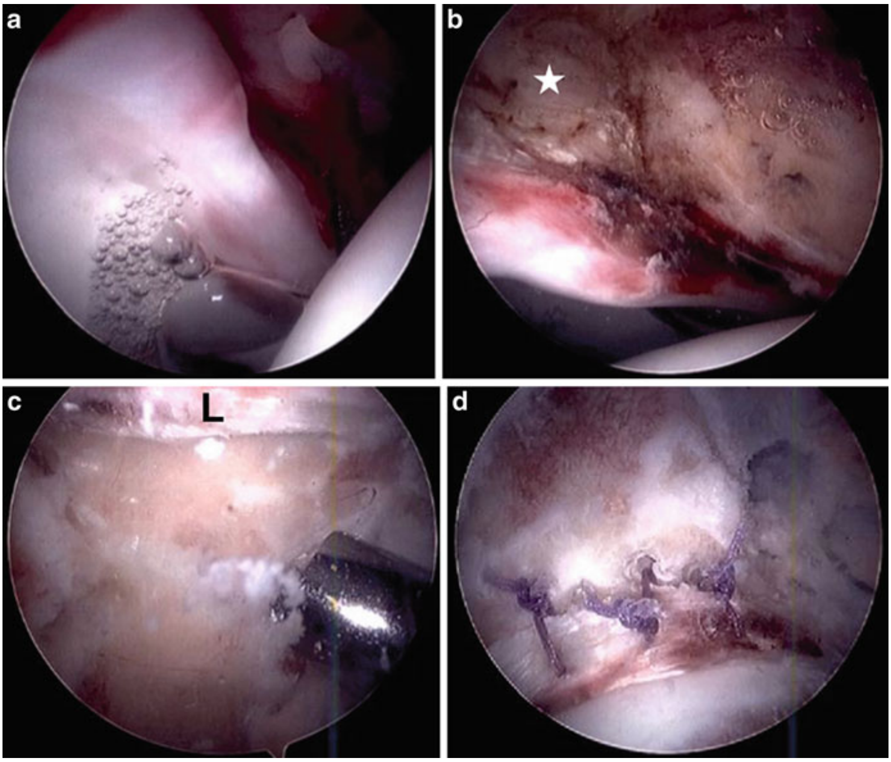


Fig. 10 **a**–**d** (**a**) View from the anterolateral portal in a right hip showing no discreet labral detachment, no cartilage wear on the femoral head, early grade 1 chondral delamination on transition zone cartilage from cam lesion, and anterior capsular inflammation. (**b**) View from the anterolateral portal demonstrating significant capsular-sided labral erythema, rectus inflammation, and distal extension of the AIIS (*white asterisk*) below the acetabular rim. (**c**) Subspine decompression with a 5.5 mm bur. View from the mid-anterior portal confirms that the resection of the acetabular rim distally to the AIIS extends all the way to the transition zone of the chondrolabral junction. (**d**) Arthroscopic view showing adequate subspine decompression, rim correction, and labral refixation. *L* labrum

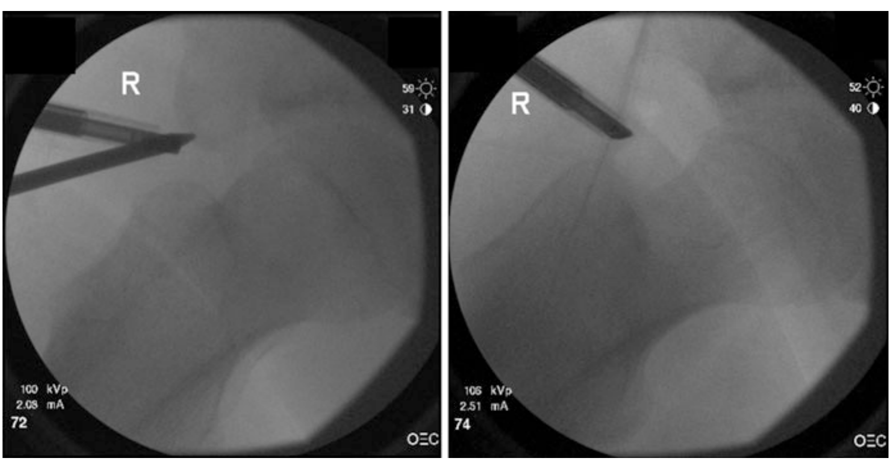


Fig. 11 *Left*, intraoperative fluoroscopic view of the same patient (Fig. 10a–d) demonstrating the prominent AIIS and anterior cam lesion. *Right*, after subspine and cam decompression, the AIIS shape is no longer extending distally and anteriorly, and the head–neck offset is recreated along the anterior femoral neck

of the AIIS has been shown to be devoid of tendinous origin and a safe zone for decompression [30]. If the estimated time of traction will exceed 90 min due to significant work required in the central compartment (rim trimming, AIIS resection, labral repair), then the AIIS decompression can be performed or completed without traction to decrease the incidence of extended traction-related complications [45]. Potential disadvantage of this approach is that after completion of the AIIS decompression, labral attachment integrity should be reassessed. When the AIIS prominence extends straight distally to the rim level and therefore significant bone resection may be required, the labrum may need to be repaired at the completion of the decompression, which is not possible without traction. The subspine decompression is considered successful when the resection of the acetabular rim distally to the AIIS extends all the way to the transition zone of the chondrolabral junction minimizing, thus, the likelihood for residual postoperative rim-impaction impingement (Fig. 10c). However, intraoperative fluoroscopy should be used to confirm the extent of AIIS resection, especially distally (Fig. 11). Radiological and intraoperative recognition of the extent of the AIIS prominence relative to the acetabular rim both anteriorly and distally is of paramount importance. It has been shown that AIIS extending to or below the level of the anterior–superior acetabular rim may be partially or completely responsible for the appearance of a radiographic crossover sign in hips with anteverted acetabulum [46]. The use of fluoroscopic imaging may prevent unnecessary resection of acetabular hyaline cartilage and production of iatrogenic acetabular dysplasia. In the setting of normal acetabular version, preoperative crossover sign on a well-positioned AP view of the pelvis may be corrected after adequate isolated AIIS resection [46]. Extended subspine decompression combined with rim resection and damage to the transition zone cartilage may necessitate labral refixation. Destabilized labrum should be reattached to the rim with modern arthroscopic techniques (Fig. 10d). At this point, a third portal is established; the DALA portal is placed in line with the anterolateral portal, approximately 5 cm distally which will enable the positioning of the anchor/anchors along the acetabular rim and could be used as a working portal for the femoroplasty in the peripheral compartment later, if needed. Depending on the femoral torsion, the DALA portal may be placed slightly more anterior in retroverted femurs to reduce the possibility of instruments' impaction against the anterior facet of the greater trochanter. Whether the labrum should be debrided or repaired, it is based on the size of the tear, the degree of detachment, and the quality of the labral tissue, aiming to preserve as much labral tissue possible and reestablish the normal seal effect of the labrum. Anatomic labral refixation can be accomplished with small diameter anchors. Labral eversion should be avoided, and depending on labral tissue quality, sutures should be placed either intrasubstance or circumferentially around the labrum (Fig. 10d).

After the central compartment is addressed and reevaluated for any residual sites of pathology, the hip is taken out of traction. If residual AIIS decompression is required, it is completed. The hip is flexed to evaluate for remaining subspine impingement. Should preoperative imaging and intraoperative findings document the presence of coexisting cam deformity, femoroplasty must follow in order to restore the normal offset of the head–neck junction and treat the intra-articular FAI (Fig. 11). Although femoroplasty can be performed effectively without capsulotomy, the T-capsulotomy leads to greater visualization of the peripheral compartment, allowing the surgeon to perform osteoplasty medially, laterally, and distally with greater ease up to the intertrochanteric line. Decompression of the anterior facet of the greater trochanter in certain cases of likely extra-articular impingement can be accomplished as well with this approach. Recent data from a CT-based, dynamic computer model showed that in straight flexion, impingement occurred most often on the inferior/medial region of the femoral head–neck junction along the medial synovial fold, whereas the average location of impingement on the acetabulum occurred at 1:30 (range, 12:30–2:15), corresponding to the area distal to the subspine region. T-capsulotomy may be required to address these “bump” locations. Fluoroscopy confirms at the end the adequate cam decompression (Fig. 11). Since these arthroscopic procedures are usually lengthy in time and require extensive soft-tissue dissection, it is critical to monitor the patient’s abdominal pressure in order to observe for potential intra-abdominal extravasations of fluids, which may evolve to a serious complication. Finally, the T-capsulotomy is repaired in a side-to-side fashion with approximately 4–6 No. 2 nonabsorbable sutures depending upon the degree of inherent structural instability (static overload) or capsular laxity. Postoperative management should include 2–4 weeks of protected weight bearing with crutches and ROM exercises until protective muscle strength is regained. Strengthening and proprioception exercises may enhance the rehabilitation. No specific changes in postoperative rehabilitation are required when an AIIS decompression has been performed as part of an arthroscopic FAI corrective procedure. Anti-inflammatory medications appear to decrease the risk of HO [47], especially if aggressive subspine decompression has been performed. In the case of positive history for HO or intolerance to anti-inflammatories, one dose of radiation is recommended on postoperative day one. AP pelvis and lateral hip radiographs should be obtained at the 6-week follow-up visit and then at 1 year and 2 years after the operation to assess for potential development of HO, bone regrowth, or joint degradation.

Outcomes Following Arthroscopic AIIS/Subspine Decompression

The rationale of arthroscopic subspine decompression is supported by short-term outcomes series revealing improvement in hip function and ROM [6, 8, 30, 48]. Two studies are limited to small case series [6, 8] and have shown significant improvements in outcomes scores and hip flexion ROM, whereas a case report has shown similar results [48]. Larson et al. [6] introduced the concept of AIIS impingement and included 3 representative cases after arthroscopic subspine decompression, the mHHS improved from a mean of 76 points preoperatively to 94 points postoperatively with minimum 1-year follow-up. Hetsroni et al. [8] published the largest series of arthroscopic subspine decompression in 10 hips due to prior AIIS avulsion injury. At a mean of 14.7 months’ follow-up, the mHHS improved from a mean of 64 points preoperatively to 98 points postoperatively; an improvement of a mean 18° in hip flexion range of motion was recorded as well. Matsuda and Calipusan [48] reported a case of arthroscopic AIIS decompression with 18 months’ follow-up in a 13-year-old track athlete with a prior apophyseal avulsion injury that led to a resolution of symptoms. He returned to football with no symptoms with a terminal hip flexion of 120°, whereas his self-assessed nonarthritic hip score improved from 22 preoperatively to 98 postoperatively. Hapa et al. [30] recently published the results of the largest consecutive series to date in the literature. In this clinical series, 163 (150 patients) AIIS decompressions were performed for symptomatic subspine impingement. At a mean follow-up of 11.1 months, the mean mHHS significantly improved from 63.1 points preoperatively to 85.3 points. Short Form 12 scores improved significantly from a mean of 70.4 preoperatively to a mean of 81.3 postoperatively. Similarly, the mean pain score on a visual analog scale improved significantly from a mean of 4.9 preoperatively to a mean of 1.9 postoperatively. All published data highlight the low risk for postoperative hip flexion weakness and rectus femoris avulsion/rupture after such decompressions. Table 1 summarizes in detail the findings (number of patients, length of follow-up, and improvements in hip flexion ROM and in patient-reported scores) of the three published cases series.

Summary

Arthroscopic decompression of a symptomatic AIIS deformity is a reproducible and safe procedure that has shown to provide excellent outcomes at short-term follow-up. An arthroscopic approach may be advantageous in patients with

Table 1 Short-term outcomes of clinical series following arthroscopic subspine decompression for symptomatic AIIS impingement

	Number of patients/ hips	Follow-up in months, mean (min–max)	Preoperative hip flexion, mean (min–max)	Postoperative hip flexion, mean (min–max)	Preoperative hip and/or health survey and/or pain score, mean (min–max)	Postoperative hip and/or health survey and/or pain score, mean (min–max)
Larson et al. [6]	3/3	16 (12–18)	105° (100–110)	126.7° (125–130)	mHHS: 75.7 (74–79)	mHHS: 85.3 (37–100)
					VAS: 6.2 (4.8–8.0)	VAS: 1.1 (0–1.7)
Hetsroni et al. [8]	10/10	14.7 (6–26)	98.5° (90–110)	117° (110–130)	mHHS: 64.2 (41–96)	mHHS: 98.4 (96–100)
Hapa et al. [30]	150/163	11.1 (6–24)	N/R	N/R	mHHS: 63.1 (21–90)	mHHS: 85.3 (37–100)
					SF-12: 70.4 (34–93)	SF-12: 81.3 (31–99)
					VAS: 4.9 (0.1–8.6)	VAS: 1.9 (0–7.8)

mHHS modified Harris hip score, *SF-12* short form 12, *VAS* pain score on a visual analog scale, *N/R* not reported

mixed intra- and extra-articular causes of hip pain and dysfunction, because it enables the surgeon to address all pathologies with a single arthroscopic procedure. Preoperative planning to assess the morphology of the AIIS prominence with regard to location and required amount of decompression is of paramount importance. The use of fluoroscopy during surgery may prevent over- or under-resection of the AIIS distal or anterior extension, avoiding thus iatrogenic dysplasia or residual impingement, respectively. Long traction times should be avoided, and when extensive work in the central compartment is anticipated, the AIIS resection can be performed without traction. Because these procedures are lengthier and require extensive soft-tissue dissection, postoperative anti-inflammatory protocol is essential for the prevention of heterotopic bone formation. Adherence to these principles is associated with effectiveness and safety following arthroscopic subspine decompression.

References

1. Ganz R, Parvizi J, Beck M, Leunig M, Notzli H, Siebenrock KA. Femoroacetabular impingement: a cause for osteoarthritis of the hip. Clin Orthop Relat Res. 2003;417:112–20. doi:10.1097/01.blo.0000096804.78689.c2.
2. Rab GT. The geometry of slipped capital femoral epiphysis: implications for movement, impingement, and corrective osteotomy. J Pediatr Orthop. 1999;19:419–24.
3. Leunig M, Casillas MM, Hamlet M, Hersche O, Notzli H, Slongo T, Ganz R. Slipped capital femoral epiphysis: early mechanical damage to the acetabular cartilage by a prominent femoral metaphysis. Acta Orthop Scand. 2000;71:370–5. doi:10.1080/000164700317393367.
4. Toogood PA, Skalak A, Cooperman DR. Proximal femoral anatomy in the normal human population. Clin Orthop Relat Res. 2009;467:876–85. doi:10.1007/s11999-008-0473-3.
5. Siebenrock KA, Wahab KH, Werlen S, Kalhor M, Leunig M, Ganz R. Abnormal extension of the femoral head epiphysis as a cause of cam impingement. Clin Orthop Relat Res. 2004;418:54–60.
6. Larson CM, Kelly BT, Stone RM. Making a case for anterior inferior iliac spine/subspine hip impingement: three representative case reports and proposed concept. Arthroscopy. 2011;27:1732–7. doi:10.1016/j.arthro.2011.10.004.
7. Hetsroni I, Poultsides L, Bedi A, Larson CM, Kelly BT. Anterior inferior iliac spine morphology correlates with hip range of motion: a classification system and dynamic model. Clin Orthop Relat Res. 2013;471:2497–503. doi:10.1007/s11999-013-2847-4.
8. Hetsroni I, Larson CM, Dela Torre K, Zbeda RM, Magennis E, Kelly BT. Anterior inferior iliac spine deformity as an extra-articular source for hip impingement: a series of 10 patients treated with arthroscopic decompression. Arthroscopy. 2012;28:1644–53. doi:10.1016/j.arthro.2012.05.882.
9. Sink EL, Gralla J, Ryba A, Dayton M. Clinical presentation of femoroacetabular impingement in adolescents.

J Pediatr Orthop. 2008;28:806–11. doi:10.1097/BPO.0b013e31818e194f.
10. Anderson LA, Peters CL, Park BB, Stoddard GJ, Erickson JA, Crim JR. Acetabular cartilage delamination in femoroacetabular impingement. Risk factors and magnetic resonance imaging diagnosis. J Bone Joint Surg Am. 2009;91:305–13. doi:10.2106/JBJS.G.01198.
11. Dudda M, Albers C, Mamisch TC, Werlen S, Beck M. Do normal radiographs exclude asphericity of the femoral head-neck junction? Clin Orthop Relat Res. 2009;467:651–9. doi:10.1007/s11999-008-0617-5.
12. Cobb J, Logishetty K, Davda K, Iranpour F. Cams and pincer impingement are distinct, not mixed: the acetabular pathomorphology of femoroacetabular impingement. Clin Orthop Relat Res. 2010;468:2143–51. doi:10.1007/s11999-010-1347-z.
13. Beck M, Kalhor M, Leunig M, Ganz R. Hip morphology influences the pattern of damage to the acetabular cartilage: femoroacetabular impingement as a cause of early osteoarthritis of the hip. J Bone Joint Surg Br. 2005;87:1012–8. doi:10.1302/0301-620X.87B7.15203.
14. Bedi A, Dolan M, Leunig M, Kelly BT. Static and dynamic mechanical causes of hip pain. Arthroscopy. 2011;27:235–51. doi:10.1016/j.arthro.2010.07.022.
15. Ito K, Minka II MA, Leunig M, Werlen S, Ganz R. Femoroacetabular impingement and the cam-effect. A MRI-based quantitative anatomical study of the femoral head-neck offset. J Bone Joint Surg Br. 2001;83:171–6.
16. Notzli HP, Wyss TF, Stoecklin CH, Schmid MR, Treiber K, Hodler J. The contour of the femoral head-neck junction as a predictor for the risk of anterior impingement. J Bone Joint Surg Br. 2002;84:556–60.
17. Leunig M, Nho SJ, Turchetto L, Ganz R. Protrusio acetabuli: new insights and experience with joint preservation. Clin Orthop Relat Res. 2009;467:2241–50. doi:10.1007/s11999-009-0853-3.
18. Smith-Petersen MN. The classic: treatment of malum coxae senilis, old slipped upper femoral epiphysis, intrapelvic protrusion of the acetabulum, and coxa plana by means of acetabuloplasty. 1936. Clin Orthop Relat Res. 2009;467:608–15. doi:10.1007/s11999-008-0670-0.
19. Reynolds D, Lucas J, Klaue K. Retroversion of the acetabulum. A cause of hip pain. J Bone Joint Surg Br. 1999;81:281–8.
20. Siebenrock KA, Schoeniger R, Ganz R. Anterior femoro-acetabular impingement due to acetabular retroversion. Treatment with periacetabular osteotomy. J Bone Joint Surg Am. 2003;85-A:278–86.
21. Jamali AA, Mladenov K, Meyer DC, Martinez A, Beck M, Ganz R, Leunig M. Anteroposterior pelvic radiographs to assess acetabular retroversion: high validity of the “cross-over-sign”. J Orthop Res. 2007;25:758–65. doi:10.1002/jor.20380.
22. Fujii M, Nakashima Y, Yamamoto T, Mawatari T, Motomura G, Matsushita A, Matsuda S, Jingushi S, Iwamoto Y. Acetabular retroversion in developmental dysplasia of the hip. J Bone Joint Surg Am. 2010;92:895–903. doi:10.2106/JBJS.I.00046.
23. Eijer H, Podeszwa DA, Ganz R, Leunig M. Evaluation and treatment of young adults with femoro-acetabular impingement secondary to Perthes’ disease. Hip Int. 2006;16:273–80.
24. Rajasekhar C, Kumar KS, Bhamra MS. Avulsion fractures of the anterior inferior iliac spine: the case for surgical intervention. Int Orthop. 2001;24:364–5.
25. Rossi F, Dragoni S. Acute avulsion fractures of the pelvis in adolescent competitive athletes: prevalence, location and sports distribution of 203 cases collected. Skeletal Radiol. 2001;30:127–31.
26. Mader TJ. Avulsion of the rectus femoris tendon: an unusual type of pelvic fracture. Pediatr Emerg Care. 1990;6:198–9.
27. Reina N, Accadbled F, de Gauzy JS. Anterior inferior iliac spine avulsion fracture: a case report in soccer playing adolescent twins. J Pediatr Orthop B. 2010;19:158–60. doi:10.1097/BPB.0b013e32833399a4.
28. Gomez JE. Bilateral anterior inferior iliac spine avulsion fractures. Med Sci Sports Exerc. 1996;28:161–4.
29. Gray H. Muscles and fasciae. In: Pickering T, Howden R, editors. Gray’s anatomy. New York: Crown; 1977. p. 415–31.
30. Hapa O, Bedi A, Gursan O, Akar MS, Guvencer M, Havitcioglu H, Larson CM. Anatomic footprint of the direct head of the rectus femoris origin: cadaveric study and clinical series of hips after arthroscopic anterior inferior iliac spine/subspine decompression. Arthroscopy. 2013;29:1932–40. doi:10.1016/j.arthro.2013.08.023.
31. Draovitch P, Edelstein J, Kelly BT. The layer concept: utilization in determining the pain generators, pathology and how structure determines treatment. Curr Rev Musculoskelet Med. 2012;5:1–8. doi:10.1007/s12178-011-9105-8.
32. Poultsides LA, Bedi A, Kelly BT. An algorithmic approach to mechanical hip pain. HSS J. 2012;8(3):213–24.
33. Skendzel JG, Weber AE, Ross JR, Larson CM, Leunig M, Kelly BT, Bedi A. The approach to the evaluation and surgical treatment of mechanical hip pain in the young patient: AAOS exhibit selection. J Bone Joint Surg Am. 2013;95:e1331–16. doi:10.2106/JBJS.L.01611.
34. Blankenbaker DG, Tuite MJ. Non-femoroacetabular impingement. Semin Musculoskelet Radiol. 2013;17:279–85. doi:10.1055/s-0033-1348094.
35. Notzli HP, Wyss TF, Stoecklin CH, Schmid MR, Treiber K, Hodler J. The contour of the femoral head-neck junction as a predictor for the risk of anterior impingement. J Bone Joint Surg Br. 2002;84:556–60.
36. Pan H, Kawanabe K, Akiyama H, Goto K, Onishi E, Nakamura T. Operative treatment of hip impingement caused by hypertrophy of the anterior inferior iliac spine. J Bone Joint Surg Br. 2008;90:677–9. doi:10.1302/0301-620X.90B5.20005.

37. Kivlan BR, Martin RL, Sekiya JK. Response to diagnostic injection in patients with femoroacetabular impingement, labral tears, chondral lesions, and extra-articular pathology. Arthroscopy. 2011;27:619–27. doi:10.1016/j.arthro.2010.12.009.
38. Amar E, Druckmann I, Flusser G, Safran MR, Salai M, Rath E. The anterior inferior iliac spine: size, position, and location. An anthropometric and sex survey. Arthroscopy. 2013;29:874–81. doi:10.1016/j.arthro.2013.01.023.
39. Leunig M, Beck M, Kalhor M, Kim YJ, Werlen S, Ganz R. Fibrocystic changes at anterosuperior femoral neck: prevalence in hips with femoroacetabular impingement. Radiology. 2005;236:237–46. doi:10.1148/radiol.2361040140.
40. Milone MT, Bedi A, Poultsides L, Magennis E, Byrd JW, Larson CM, Kelly BT. Novel CT-based three-dimensional software improves the characterization of cam morphology. Clin Orthop Relat Res. 2013;471:2484–91. doi:10.1007/s11999-013-2809-x.
41. Bedi A, Thompson M, Uliana C, Magennis E, Kelly BT. Assessment of range of motion and contact zones with commonly performed physical exam manoeuvers for femoroacetabular impingement (FAI): what do these tests mean? Hip Int. 2013. doi:10.5301/hipint.5000060.
42. Robertson WJ, Kelly BT. The safe zone for hip arthroscopy: a cadaveric assessment of central, peripheral, and lateral compartment portal placement. Arthroscopy. 2008;24:1019–26. doi:10.1016/j.arthro.2008.05.008.
43. Matsuda DK. Acute iatrogenic dislocation following hip impingement arthroscopic surgery. Arthroscopy. 2009;25:400–4. doi:10.1016/j.arthro.2008.12.011.
44. Ranawat AS, McClincy M, Sekiya JK. Anterior dislocation of the hip after arthroscopy in a patient with capsular laxity of the hip. A case report. J Bone Joint Surg Am. 2009;91:192–7. doi:10.2106/JBJS.G.01367.
45. Konan S, Rhee SJ, Haddad FS. Hip arthroscopy: analysis of a single surgeon's learning experience. J Bone Joint Surg Am. 2011;93 Suppl 2:52–6. doi:10.2106/JBJS.J.01587.
46. Zaltz I, Kelly BT, Hetsroni I, Bedi A. The crossover sign overestimates acetabular retroversion. Clin Orthop Relat Res. 2013;471:2463–70. doi:10.1007/s11999-012-2689-5.
47. Bedi A, Zbeda RM, Bueno VF, Downie B, Dolan M, Kelly BT. The incidence of heterotopic ossification after hip arthroscopy. Am J Sports Med. 2012;40:854–63. doi:10.1177/0363546511434285.
48. Matsuda DK, Calipusan CP. Adolescent femoroacetabular impingement from malunion of the anteroinferior iliac spine apophysis treated with arthroscopic spinoplasty. Orthopedics. 2012;35:e460–3. doi:10.3928/01477447-20120222-44.

Greater Trochanteric Pain Syndrome

64

Randy Mascarenhas, Michael J. Salata, and Shane J. Nho

Contents

R. Mascarenhas (✉)
Department of Orthopaedic Surgery, Midwest Orthopaedics at Rush, Rush University Medical Center, Chicago, IL, USA

M.J. Salata
Department of Orthopaedic Surgery, Joint Preservation and Cartilage Restoration Center, University Hospitals Case Medical Center, Cleveland, OH, USA
e-mail: michael.salata@uhhospitals.org

S.J. Nho
Department of Orthopaedic Surgery, Rush University Medical Center, Chicago, IL, USA

Midwest Orthopaedics at Rush, Hip Preservation Center, Rush University Medical Center, Chicago, IL, USA

Abstract

As techniques in minimally invasive hip surgery evolve, there is increasing opportunity to treat peritrochanteric hip conditions endoscopically. The collective diagnosis of greater trochanteric pain syndrome (GTPS) includes trochanteric bursitis, gluteus medius and minimus tears, and external snapping hip syndrome (coxa saltans). Most of these conditions can be accurately diagnosed with routine history and physical examinations aided by plain radiographs, MRI, CT scans, and ultrasonography. Nonsurgical treatment is generally the first line of treatment and most conditions will improve with oral anti-inflammatories and directed physical therapy programs. Diagnostic and therapeutic injections are useful in narrowing down the diagnosis and may also provide treatment for many ailments outside the hip joint. Minimally invasive surgical interventions via endoscopy have expanded dramatically in this area and continue to grow as we further understand the treatment pathology and select appropriate patients for surgery.

Introduction

Indications for hip arthroscopy have expanded greatly over the past decade, and it remains one of the areas with greatest growth in orthopedic surgery. Current diagnostic and treatment regimens have allowed for considerable

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_69

advancements in treatment for both intra-articular and extra-articular hip conditions. With the growth seen in the minimally invasive treatment of intra-articular hip conditions, surgeons are now venturing extracapsular to treat soft tissue ailments. Greater trochanteric pain syndrome is a relatively common clinical entity that is seen in 10–25 % of the general population [1] that encompasses disorders of the lateral, peritrochanteric space of the hip including trochanteric bursitis, tears of the gluteus medius and minimus tendons, and external coxa saltans (snapping hip). Patients generally experience reproducible pain over the greater trochanter, buttocks, or lateral thigh, and the condition is seen with increased incidence in patients with low back pain [2] and increased prevalence in women [1]. Endoscopy provides improved visualization of extra-articular pathology that previously required large open incisions for diagnosis and treatment. Specific to greater trochanteric pain disorders, endoscopic approaches are now effective in providing extra-articular access to the iliotibial band in external coxa saltans, debridement of recalcitrant trochanteric bursitis, and endoscopic repair of gluteus medius tears. The purpose of this chapter is to characterize the anatomy and diagnosis of greater trochanteric pain disorders while providing nonoperative and operative treatment options.

Relevant Anatomy

The greater trochanter lies at the junction of the femoral neck and shaft and is the site of attachment of the gluteal, obturator, and piriformis tendons. The peritrochanteric space consists of the gluteus medius and minimus tendons, iliotibial band, and greater trochanter with its associated bursa. The gluteus medius inserts into the superoposterior and lateral facets of the greater trochanter while the gluteus minimus tendon attaches to the anterior facet. A fibromuscular sheath composed of the gluteus maximus, tensor fascia lata (TFL), and iliotibial band (ITB) lies superficial to these tendons.

Three bursae in the vicinity of the greater trochanter serve to cushion the gluteal tendons, the ITB, and the TFL. The subgluteus medius bursa lies over the lateral facet with the subgluteus minimus bursa sitting deep to the tendon around the anterior facet and anterior hip capsule. The largest bursa, the trochanteric or subgluteus maximus bursa, sits deep to the fibers of the gluteus maximus and tensor fascia lata (TFL) as they form the iliotibial band (ITB). This bursa sits on top of the posterior facet of the greater trochanter, the distal-lateral gluteus medius tendon at the lateral facet, and the proximal vastus lateralis insertion [3]. The gluteus medius and minimus tendons function in a similar manner to the rotator cuff, helping to stabilize the hip joint and initiate abduction [4]. Tears generally occur in the footprint on the greater trochanter and can be intrasubstance, partial, or complete [5]. External snapping hip generally occurs secondary to thickening of the posterior IT band, tensor fascia lata, or gluteus maximus as they slide over the greater trochanter during hip flexion.

Disease Presentation

Greater trochanteric pain syndrome (GTPS) encompasses a broad category of diagnoses and is characterized by lateral-sided hip pain and includes trochanteric bursitis, gluteus medius or minimus tears, and external coxa saltans [6, 7]. Trochanteric bursitis refers to inflammation of at least one of the three aforementioned trochanteric bursae and is thought to result from gait abnormalities, trauma, or repetitive activity [8]. It often involves pain localized to the greater trochanter that radiates down the lateral thigh or into the buttocks caused by repetitive friction between the ITB and greater trochanter with hip flexion and extension. Patients presenting with trochanteric bursitis generally have other associated pathology such as osteoarthritis of the ipsilateral hip or lumbar spine [9], but the disease is now being seen more commonly in younger patient populations [10, 11].

Gluteus medius or minimus tears are the most common presentation and are often found in patients with recalcitrant trochanteric bursitis [12, 13]. Recent studies have suggested that tears will occur in 25 % of middle-aged women and 10 % of men of similar age [14]. This increased incidence in women may be secondary to the wider female pelvis [15]. The natural progression of hip abductor tendinopathy is similar to the pathogenesis of tendon degeneration elsewhere in the body and generally begins with bursitis before progressing down the spectrum of tendonitis, tendinopathy, partial thickness tearing, full thickness tearing, and massive tears. Chronic massive tears of the abductors may lead to significant fatty infiltration and atrophy of the gluteal muscles, which may lead to a guarded functional prognosis following repair.

External snapping hip syndrome is often secondary to thickening of the posterior third of the IT band (ITB) that lies posterior to the greater trochanter when the hip is in a neutral position [16]. Repeated flexion and extension of the overly taut ITB will result in mechanical symptoms as the ITB catches on the greater trochanter. Further tightening of the ITB and its resultant "snapping" is exacerbated by hip adduction and extension at the knee [17]. Women with prominent greater trochanters or a larger pelvis have a predilection for external snapping hip. This is most commonly seen in women who adduct beyond their midline during gait [17]. The snapping of the ITB itself is usually non-painful [16] but can lead to inflammation of the trochanteric bursa from the abrasive wear [18]. Kingzett-Taylor et al. [7] suggested that both abductor tendinopathy and trochanteric bursitis could be secondary to frictional trauma caused by high ITB tension. This supports the notion that causes of GTPS are multifactorial and can influence each other.

Patient History

Inflammation in the region of the greater trochanter can result in radiating pain and paresthesias that can lead to a wide and confusing initial differential diagnosis list. Patients may report a constellation of varying degrees of buttock, lateral hip, and groin pain which is due, in part, to the varying nervous supply of the peritrochanteric compartment. Patients may complain of lateral hip pain that is exacerbated by direct pressure, prolonged standing or upright activity, and activities that engage the hip abductors such as getting up from a seated position or climbing stairs. They may also report pain in a lateral decubitus position that wakes them up at night. Additionally, in most cases, the onset of symptoms is insidious, but there are some patients that report an acute exacerbation of symptoms after a specific event [19]. Patients presenting with external snapping hip syndrome may describe a snapping sensation that occurs with exercise or routine daily activities.

Physical Examination

Physicians must be certain to rule out spine pathology as a cause of symptoms. Patients suffering from a gluteus medius avulsion may present with an obvious limp [20, 21]. Additionally, certain anatomic abnormalities such as a high valgus knee angles and leg length discrepancies have been known to cause mechanical abrasion and subsequent abductor tears due to increased ITB tension [16]. Physical examination specific to the hip begins with observation of the patient for Trendelenburg gait followed by performing the Trendelenburg fatigue test. A distinct drop of the unsupported side of the pelvis indicates weakness or loss of function of the abductors. Positive findings on this test may be suggestive of possible abductor insufficiency. With the patient in a lateral decubitus position, the anterior, lateral, and posterior aspect of the greater trochanter is palpated for tenderness. Abductor strength testing can be performed in both knee flexion and extension to enable gravity strength testing.

Provocative maneuvers can be performed including the trochanteric pain sign, which is performed with the patient in a supine position. With the hip flexed to 90°, it is then abducted and externally rotated (see Fig. 1). The test is considered to be positive if there is pain with external rotation. Additionally, flexion, abduction, external rotation, and extension (FABERE) testing may also elicit pain in patients with GTPS (see Fig. 2). Resisted external rotation should also be

Fig. 1 The trochanteric pain sign is elicited with the patient supine and the hip in 90° of flexion by abducting and externally rotating the hip

Fig. 2 FABERE testing is performed with flexion, abduction, external rotation, and extension of the hip

performed while the patient is in the supine position with the hip flexed at 90° [5, 22].

The physical exam for external snapping hip syndrome generally reveals a palpable or observable snapping of the ITB over the greater trochanter upon flexion and subsequent extension. The patient can be placed in a lateral decubitus position, and a single leg bicycle maneuver can be performed which may reproduce ITB snapping. The diagnosis can be confirmed if pressure applied over the superior greater trochanter prevents snapping with repeated hip flexion. A positive OBER test resulting in significant tightening of the ITB may be seen and would qualify the patient as a good candidate for ITB release along with removal of the symptomatic bursa [23].

Diagnostic Imaging

Plain radiographs should be obtained to rule out osteoarthritis and can also demonstrate evidence of calcific tendonitis or intra-bursal calcifications. They are generally low yield for gluteus medius and minimus tears but can identify trochanteric osteophytes that the gluteal tendons may be draped over. MRI and ultrasonography are the primary imaging modalities used to diagnose tendon pathology. Specific findings in GTPS may include enthesopathic changes along the trochanteric insertion, subminimus and submedius bursitis, and fatty atrophy of the associated muscle bellies [16]. On T2-weighted MRI, hyperintensity superior and lateral to the greater trochanter is often seen due to either thickened hip abductor tendons, tendinopathy, or tendon tears (see Fig. 3a and b). Tendon discontinuity may be seen on T1 images. MRI can also be used to gauge the degree of fatty infiltration and atrophy of the abductor muscle complex. Though the overall specificity of MRI is debated [24], Kingzett-Taylor et al. found T2-weighted MRI of the superior greater trochanter to be diagnostic for partial abductor tendon tears with high sensitivity (73 %) and specificity (95 %) [7], but false positive rates have been reported to be as high as 88 % in one series [25]. Ultrasound may be a reliable alternative to MRI in the diagnosis of gluteus medius and minimus tears, with a sensitivity of 79 % and a positive predictive value of 100 % [26]. Dynamic real-time ultrasound can also be used to visualize snapping hip and identify inflamed trochanteric bursa.

Nonoperative Treatment

Nonoperative treatment modalities are the first line of treatment for GTPS. Rest, avoidance of exacerbating activities, anti-inflammatories, weight loss, and physical therapy are generally recommended before consideration for surgical intervention. Physical therapy protocols for GTPS should focus on stretching, flexibility, strengthening, and gait

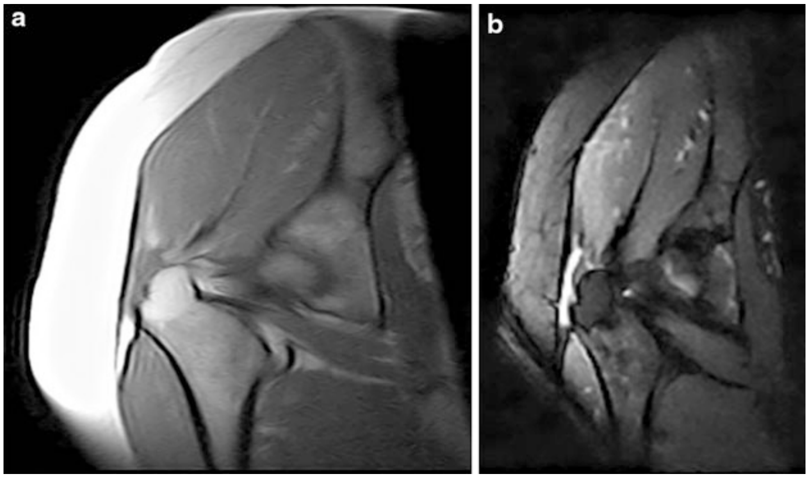


Fig. 3 Coronal T1 (**a**) and T2 (**b**) MRI sequences of the right hip show hyperintensity lateral to the greater trochanter suggestive of gluteus medius tendinopathy or tearing

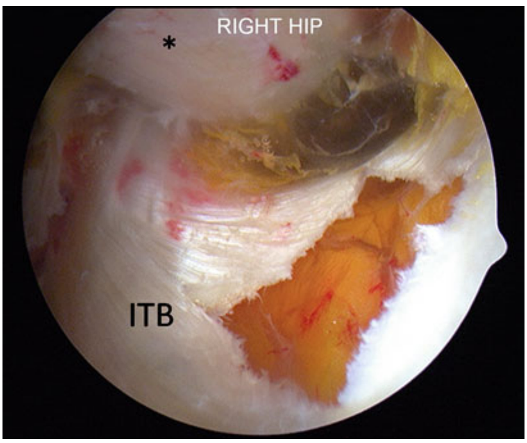


Fig. 4 Endoscopic diamond-shaped release of the iliotibial band for external coxa saltans

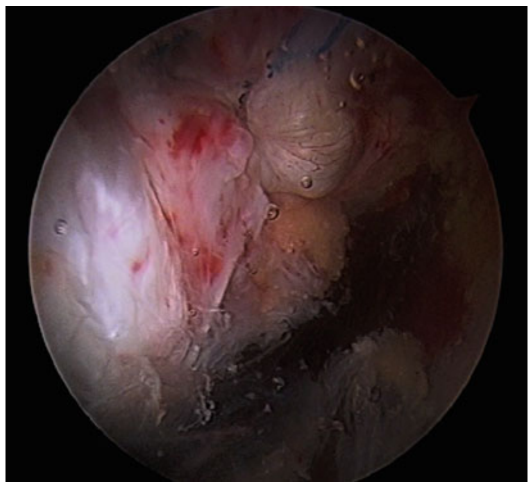


Fig. 5 Endoscopic completion of a gluteus medius tendon repair

mechanics [9]. Persistent pain despite noninvasive treatments can be treated with an injection of cortisone and local anesthetic as both a diagnostic and therapeutic treatment measure. In a study following 75 patients with trochanteric bursitis, Shbeeb et al. showed that lidocaine/betamethasone injections were effective at relieving pain secondary to GTPS in 77.1 % of patients at 1 week and 61.3 % of patients at 6 months [27]. Failure of these treatments to alleviate symptoms leads to consideration of endoscopic treatment for GTPS, which is discussed in more detail in the surgical technique chapters on trochanteric bursectomy, external snapping hip release (see Fig. 4), gluteus medius repair (see Fig. 5), and gluteus maximus transfer.

Surgical Outcomes

Baker et al. [10] reported on 25 consecutive patients undergoing endoscopic trochanteric bursectomy with mean follow-up greater than 2 years. The authors found statistically significant improvements in VAS pain scores, Harris Hip scores, and SF-36 scores when compared to preoperative scores. Similarly, Fox et al. retrospectively followed 27 patients following endoscopic bursectomy and found that 23 of 27 patients had good or excellent results at 1 year postoperatively and only two patients had recurrence of symptoms at 5 years postoperatively [31].

Voos et al. [19] reported prospectively on ten patients undergoing endoscopic gluteus minimus repair with follow-up greater than 2 years. The authors found resolution of pain and return of abductor strength in 100 % of patients and excellent postoperative Harris Hip and Hip Outcome scores. Similarly, Domb et al. reported prospectively on 15 patients undergoing arthroscopic repair of gluteus medius tears and reported improvements on all hip outcome scores in 14 out of 15 patients at greater than 2 years follow-up [32].

Studies have proven endoscopic treatment of external coxa saltans to provide excellent results. Ilizaliturri et al. [33] reported on 11 patients undergoing endoscopic treatment of external coxa saltans in the lateral position. A diamond-shaped resection/release of the ITB followed by debridement of the trochanteric bursa was performed with 10 of 11 patients still relieved of pain at minimum 1 year follow-up and all patients returning to preoperative levels of activity. No patients required revision surgery. Similarly, Polelsello et al. [34] performed an endoscopic gluteus maximus tenotomy in nine patients with refractory external snapping hip and found that all patients returned to their preoperative activity levels and none complained of weakness at almost 2 years follow-up. Seven of nine patients were relieved of pain and one patient required revision surgery. The successful outcomes seen with these studies parallel the excellent results seen with open treatment of external snapping hip by multiple authors [35–37].

Whiteside has published on a technique involving anterior transfer of the gluteus maximus and tensor fascia lata for massive, irreparable tears of the abductors [38]. Five patients were evaluated at mean 28 months follow-up after the procedure, with 3 patients having complete resolution of pain and no limp, while one patient had residual symptoms and another suffered a fall leading to fracture of the greater trochanter and persistent pain and weakness. He has also published similar results when using the transfer in patients with abductor insufficiency in the setting of total hip arthroplasty with favorable results [39, 40].

Limitations and Potential Complications of Surgery

Endoscopy for extra-articular hip conditions is a relatively new frontier. Surgical techniques and instrumentation will continue to evolve with further clinical and scientific research. Complications following endoscopy for extra-articular hip conditions have not been well described but are thought to be less frequent and less severe than complications after hip arthroscopy. The peritrochanteric space is outside the pelvis, and thus potentially severe complications such as fluid extravasation have not been reported. Other major complications such as avascular necrosis, femoral neck fracture, postoperative instability, or adhesions have also not been reported. Iatrogenic injury to the gluteal vessels or sciatic nerve is a possibility if entry into the space is either too proximal or too posterior. Walsh et al. [41] reported on 89 patients receiving endoscopic repair for abductor tendon tears from 2000 to 2008, in which the most frequent complication was deep vein thrombosis (6 %), which is low relative to other lower-extremity orthopedic procedures.

Summary

Greater trochanteric pain syndrome represents a common source of hip pain and consists of a spectrum of disease including trochanteric bursitis, gluteus medius and minimus tears, and external snapping hip. Diagnosis and indications for surgery continue to expand as advances are made in imaging and hip arthroscopy. While GTPS can often be successfully treated noninvasively, surgical intervention can lead to pain relief and return to function in patients with refractory symptoms. Endoscopic techniques for greater trochanteric pain syndrome are considered to be relatively safe with few reported complications. Nevertheless, additional clinical and scientific research in this field is paramount to the assessment and advancement of diagnosis and treatment of these extra-articular hip disorders.

References

1. Segal NA, Felson DT, Torner JC, Zhu Y, Curtis JR, Niu J, Nevitt MC. Multicenter Osteoarthritis Study Group. Greater trochanteric pain syndrome: epidemiology and associated factors. Arch Phys Med Rehabil. 2007;88(8):988–92.
2. Tortolani PJ, Carbone JJ, Quartararo LG. Greater trochanteric pain syndrome in patients referred to orthopedic spine specialists. Spine J. 2002;2(4):251–4.
3. Dwek J, Pfirrmann C, Stanley A, Pathria M, Chung CB. MR imaging of the hip abductors: normal anatomy and commonly encountered pathology at the greater trochanter. Magn Reson Imaging Clin N Am. 2005;13 (4):691–704, vii.
4. Bunker TD, Esler CN, Leach WJ. Rotator-cuff tear of the hip. J Bone Jt Surg Br. 1997;79(4):618–20.
5. Domb BG, Nasser RM, Botser IB. Partial-thickness tears of the gluteus medius: rationale and technique for trans-tendinous endoscopic repair. Arthroscopy. 2010;26(12):1697–705.
6. Bard H. Tendinopathy of the gluteus medius tendon. Rev Prat. 2009;59:463–8.
7. Kingzett-Taylor A, Tirman PF, Feller J, et al. Tendinosis and tears of gluteus medius and minimus muscles as a cause of hip pain: MR imaging findings. AJR Am J Roentgenol. 1999;173:1123–6.
8. Fearon AM, Scarvell JM, Cook JL, Smith PN. Does ultrasound correlate with surgical or histologic findings in greater trochanteric pain syndrome? A pilot study. Clin Orthop Relat Res. 2010;468(7):1838–44.
9. Schapira D, Nahir M, Scharf Y. Trochanteric bursitis: a common clinical problem. Arch Phys Med Rehabil. 1986;67(11):815–7.
10. Baker Jr CL, Massie RV, Hurt WG, Savory CG. Arthroscopic bursectomy for recalcitrant trochanteric bursitis. Arthroscopy. 2007;23(8):827–32.
11. Farr D, Selesnick H, Janecki C, Cordas D. Arthroscopic bursectomy with concomitant iliotibial band release for the treatment of recalcitrant trochanteric bursitis. Arthroscopy. 2007;23(8):905.e1–5.
12. Kagan 2nd A. Rotator cuff tears of the hip. Clin Orthop Relat Res. 1999;368:135–40.
13. Lachiewicz PF. Abductor tendon tears of the hip: evaluation and management. J Am Acad Orthop Surg. 2011;19:385–91.
14. Robertson WJ, Gardner MJ, Barker JU, Boraiah S, Lorich DG, Kelly BT. Anatomy and dimensions of the gluteus medius tendon insertion. Arthroscopy. 2008;24(2):130–6. doi:10.1016/j.arthro.2007.11.015.
15. Tibor LM, Sekiya JK. Differential diagnosis of pain around the hip joint. Arthroscopy. 2008;24 (12):1407–21.
16. Ilizaliturri Jr VM, Camacho-Galindo J, Evia Ramirez AN, et al. Soft tissue pathology around the hip. Clin Sports Med. 2011;30:391–415.
17. Anderson K, Strickland SM, Warren R. Hip and groin injuries in athletes. Am J Sports Med. 2001;29:521–33.
18. Voos JE, Rudzki JR, Shindle MK, et al. Arthroscopic anatomy and surgical techniques for peritrochanteric space disorders in the hip. Arthroscopy. 2007;23:1246.e1–1246.e5.
19. Voos JE, Shindle MK, Pruett A, et al. Endoscopic repair of gluteus medius tendon tears of the hip. Am J Sports Med. 2009;37:743–7.
20. Svensson O, Skold S, Blomgren G. Integrity of the gluteus medius after the transgluteal approach in total hip arthroplasty. J Arthroplasty. 1990;5:57–60.
21. Weber M, Berry DJ. Abductor avulsion after primary total hip arthroplasty. Results of repair. J Arthroplasty. 1997;12:202–6.
22. Bird PA, Oakley SP, Shnier R, et al. Prospective evaluation of magnetic resonance imaging and physical examination findings in patients with greater trochanteric pain syndrome. Arthritis Rheum. 2001;44:2138–45.
23. Strauss EJ, Nho SJ, Kelly BT. Greater trochanteric pain syndrome. Sports Med Arthrosc. 2010;18:113–9.
24. Blankenbaker DG, Ullrick SR, Davis KW, et al. Correlation of MRI findings with clinical findings of trochanteric pain syndrome. Skeletal Radiol. 2008;37:903–9.
25. Kong A, Van der Vliet A, Zadow S. MRI and US of gluteal tendinopathy in greater trochanteric pain syndrome. Eur Radiol. 2007;17(7):1772–83.
26. Westacott DJ, Minns JI, Foguet P. The diagnostic accuracy of magnetic resonance imaging and ultrasonography in gluteal tendon tears–a systematic review. Hip Int. 2011;21(6):637–45.
27. Shbeeb MI, O'Duffy JD, Michet Jr CJ, O'Fallon WM, Matteson EL. Evaluation of glucocorticosteroid injection for the treatment of trochanteric bursitis. J Rheumatol. 1996;23(12):2104–6.
28. Shbeeb MI, O'Duffy JD, Michet Jr CJ, O'Fallon WM, Matteson EL. Evaluation of glucocorticosteroid injection for the treatment of trochanteric bursitis. J Rheumatol. 1996;23(12):2104–6. PubMed PMID: 8970048.
29. Kandemir U, Bharam S, Philippon MJ, Fu FH. Endoscopic treatment of calcific tendinitis of gluteus medius and minimus. Arthroscopy. 2003;19(1):E4.
30. Miozzari HH, Dora C, Clark JM, et al. Late repair of abductor avulsion after the transgluteal approach for hip arthroplasty. J Arthroplasty. 2010;25:450–7.e1.
31. Fox JL. The role of arthroscopic bursectomy in the treatment of trochanteric bursitis. Arthroscopy. 2002;18(7):E34.
32. Domb BG, Botser I, Giordano BD. Outcomes of endoscopic gluteus medius repair with minimum 2-year follow-up. Am J Sports Med. 2013;41(5):988–97.
33. Ilizaliturri Jr VM, Martinez-Escalante FA, Chaidez PA, Camacho-Galindo J. Endoscopic iliotibial band release for external snapping hip syndrome. Arthroscopy. 2006;22(5):505–10.
34. Polesello GC, Queiroz MC, Domb BG, Ono NK, Honda EK. Surgical technique: endoscopic gluteus maximus tendon release for external snapping hip syndrome. Clin Orthop Relat Res. 2013;471(8):2471–6.

35. Brignall CG, Stainsby GD. The snapping hip. Treatment by Z-plasty. J Bone Jt Surg Br. 1991;73(2):253–4.
36. Provencher MT, Hofmeister EP, Muldoon MP. The surgical treatment of external coxa saltans (the snapping hip) by Z-plasty of the iliotibial band. Am J Sports Med. 2004;32(2):470–6.
37. White RA, Hughes MS, Burd T, Hamann J, Allen WC. A new operative approach in the correction of external coxa saltans: the snapping hip. Am J Sports Med. 2004;32(6):1504–8.
38. Whiteside LA. Surgical technique: gluteus maximus and tensor fascia lata transfer for primary deficiency of the abductors of the hip. Clin Orthop Relat Res. 2013.
39. Whiteside LA. Surgical technique: transfer of the anterior portion of the gluteus maximus muscle for abductor deficiency of the hip. Clin Orthop Relat Res. 2012;470(2):503–10.
40. Whiteside LA, Nayfeh T, Katerberg BJ. Gluteus maximus flap transfer for greater trochanter reconstruction in revision THA. Clin Orthop Relat Res. 2006;453:203–10.
41. Walsh M, Walsh NA, Walton J et al. Surgical repair of the abductor mechanism of the hip. 2009;463.

Surgical Technique: Endoscopic Trochanteric Bursectomy

65

Michael J. Salata and Shane J. Nho

Contents

Abstract

As arthroscopic procedures for intra-articular hip pathology have increased in popularity and frequency, there has been a natural progression of thought to expand the reach of this minimally invasive technique to other areas of the pelvic region. With improvements in surgical instruments, fluid management systems, and improved diagnostics, endoscopy of the peritrochanteric space has become technically possible, and pathology in this compartment has been found to be amendable to arthroscopic treatment. Greater trochanteric bursitis (GTB) is an important cause of hip pain and is readily treatable via endoscopic techniques. This pathology may be evaluated with plain radiographs including an AP pelvis and 45° Dunn lateral and weight-bearing false profile, as well as CT, ultrasound, and MRI. Nonoperative management of GTB is preferred and can be successful the majority of the time, and when pain persists despite the above management techniques, an injection of local anesthetic and corticosteroid directed to the GTB area can be effective. Failure of conservative treatment is an indication for endoscopic treatment which can be a reliable and successful treatment in the properly indicated patient.

Electronic supplementary material: The online version of this chapter (doi:10.1007/978-1-4614-6965-0_70) contains supplementary material, which is available to authorized users.

M.J. Salata (✉)
Department of Orthopaedic Surgery, Joint Preservation and Cartilage Restoration Center, University Hospitals Case Medical Center, Cleveland, OH, USA
e-mail: Michael.Salata@uhhospitals.org

S.J. Nho
Department of Orthopaedic Surgery, Rush University Medical Center, Chicago, IL, USA

Midwest Orthopaedics at Rush, Hip Preservation Center, Rush University Medical Center, Chicago, IL, USA
e-mail: snho@rushortho.com

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_70

Introduction

As arthroscopic procedures for intra-articular hip pathology have increased in popularity and frequency, there has been a natural progression of thought to expand the reach of this minimally invasive technique to other areas of the pelvic region. With improvements in surgical instruments, fluid management systems, and improved diagnostics, endoscopy of the peritrochanteric space has become technically possible, and pathology in this compartment has been found to be amendable to arthroscopic treatment. The obvious advantages of arthroscopy include its minimal invasiveness and improved visualization not possible with limited open approaches. As with any emerging technology, understanding the indications for these techniques and their limitations is important in continuing to show successful outcomes for these procedures.

Patient History and Examination

Greater trochanteric bursitis (GTB) is a diagnosis of exclusion. Clinically there are numerous other potential causes of lateral-sided hip pain, and an exhaustive and detailed work-up should be performed to rule out such entities as gluteus medius and minimus tears, lumbar radiculopathies, and external coxa saltans. The majority of patients will present with an insidious onset of lateral-sided hip discomfort. Pain due to direct trauma to the lateral side of the hip can lead to traumatic bursitis or rupture of the hip abductors, and in patients with associated weakness, a timely diagnosis of abductor tendon tear is very important. Many patients will complain of an inability to lie comfortably on the affected side and tenderness to touch in the lateral aspect of the hip. Anterior discomfort should prompt a further evaluation of the hip joint proper, and entities such as hip arthritis and labral pathology can coexist with GTB.

Imaging

As with any condition of the hip, radiographic evaluation should be undertaken in this patient population. Plain radiographs including an AP pelvis and 45° Dunn lateral and weight-bearing false profile are preferred as a baseline evaluation. This condition can often be seen in the younger patient population when hip dysplasia is also present as forces can be placed on the supporting hip musculature in this instance and can lead to pain. The presence and severity of hip osteoarthritic changes should be documented, and assessment for intra-bursal calcifications may indicate a more severe bursitis, but are considered nonspecific [1–4]. Features that can be seen frequently on plain radiographs are calcifications located on the greater trochanter known as calcific tendonitis of the gluteus medius and minimus. These lesions may be a sign of partial undersurface tearing of the abductors or may lead to tenting of the gluteal tendons and can be a source of lateral-sided peritrochanteric pain. Other modalities that have been demonstrated to be of some use in excluding other sources of lateral-sided hip pain include CT and dynamic ultrasound [5–7]. Increased signal in the bursal tissues on T2-weighted imaging is considered diagnostic for this condition.

Treatment

Nonoperative management of GTB is preferred and can be successful the majority of the time. As this can often be an overuse injury in patients, rest, NSAIDs, icing techniques, and stretching of the iliotibial band can often lead to resolution of symptoms. Weight reduction and a dedicated course of physical therapy can also help to limit discomfort in patients that are over their ideal BMI or in those found to have weakness in their supporting pelvic musculature [4, 8, 9].

When pain persists despite the above management techniques, an injection of local anesthetic and corticosteroid directed to the GTB area can be effective. As this space is often deep, use of a longer spinal needle is often required to place the injection accurately, and this can further be augmented with ultrasound guidance if desired. This injection can serve two purposes. It can be therapeutic in many patients and can also be diagnostic in those patients where it is difficult to determine the overall pain generator in the hip area [9]. Surgical intervention should be a last resort but can be effective in patients where nonoperative management has not provided long-term resolution of symptoms. Failure of at least two injections is required prior to offering surgical treatment.

Surgical Technique

Endoscopic bursectomy has been described in the literature as an effective method for recalcitrant cases [10–13]. The potential for heterotopic ossification should be discussed, as should postoperative prophylaxis. Patients take 10 days of 75 mg-sustained release Indocin to minimize the risk of this potentially painful and debilitating side effect. Endoscopy of the peritrochanteric space can be performed in either the supine or lateral position depending on surgeon preference as described in the literature by Byrd [14] and Glick et al. [15], respectively. If any intra-articular or peripheral compartment work is to be done, this should be accomplished first moving to the peritrochanteric compartment upon completion of these procedures.

The supine position is preferred for this procedure. The patient is placed into well-padded boots and a well-padded and offset perineal post is placed. A commercially available table attachment designed for hip arthroscopy is used. An SCD is placed on the nonoperative limb, and antibiotics are infused intravenously prior to skin incision. If intra-articular procedures are planned, the hip is distracted; if an isolated bursectomy is planned, no traction is applied to the limb. The pad on the perineal post is often omitted or removed if an isolated procedure of the bursa is planned as it allows for more normal hip range of motion. The leg is abducted approximately 15° and placed in neutral extension with the foot in slight internal rotation to present the trochanter.

Portal Placement

The standard anterolateral (AL) portal and a distal anterolateral accessory (DALA) portal are effective for a diagnostic arthroscopy of this compartment and for performing a thorough bursectomy (Fig. 1). Additional portals can be added as necessary for more involved techniques such as gluteus medius repair. The use of a high flow bridge and a pump that controls both inflow and outflow maximizes visualization in this space. The location of the AL portal is located 1 cm proximal and 1 cm medial to the anterior aspect of the greater trochanter. Fluoroscopy can be helpful to localize the AL portal, and in some instances when a gluteus medius repair is considered, a more proximal location may maximize visualization. A 5.0 mm metal cannula placed over a blunt trochar is introduced posteriorly between the lateral aspect of the greater trochanter and the iliotibial band and swept back and forth to clear a space and establish a "room with a view" in this compartment. Fluoroscopy is used to confirm that the cannula is adjacent to the greater trochanter. Initially this space may be filled with a thickened and inflamed bursa. To establish the DALA portal, a spinal needle is placed approximately 5 cm distal to the AL portal. A nitinol wire can be placed at this point and an incision made over the wire with a #11 blade. A cannulated switching stick can be then passed over the wire and a slotted cannula over this to accurately prepare for introduction of instruments. Alternatively, a plastic cannula can be used in this setting but may limit the excursion of instruments and is often unnecessary.

Diagnostic Arthroscopy and Bursectomy

Once the initial AL portal is established, a diagnostic arthroscopy is performed with a 70° arthroscope. Initially the camera and light source is directed inferiorly, and the insertion of the gluteus maximus tendon under the vastus lateralis is identified; this will be the distal extent of the

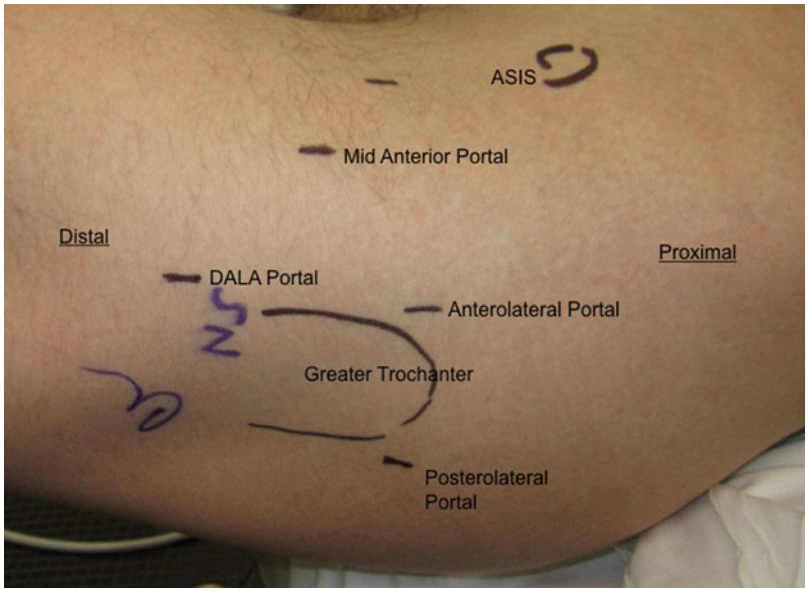


Fig. 1 Portal placement for trochanteric bursectomy. A preferred technique is to use the anterolateral portal and the DALA portal. Other portals can be used according to surgeon preference

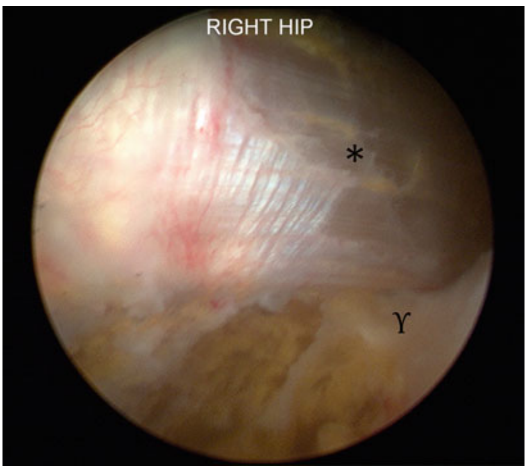


Fig. 2 Distal extent of the peritrochanteric space. * vastus lateralis, *ϒ* gluteus maximus insertion

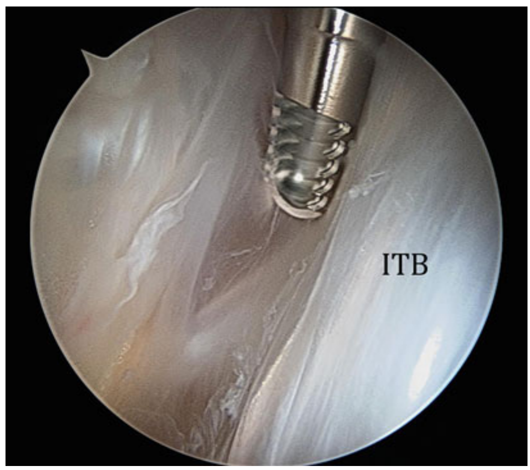


Fig. 3 Bursa in the peritrochanteric space. *ITB* iliotibial band

bursectomy (Fig. 2). An initial bursectomy may be required to maximize visualization of this structure. There are three bursas that have been described, and a thorough bursectomy requires that the subgluteus maximus bursa, the subgluteus medius bursa, and the gluteus minimus bursa are all debrided (Fig. 3). The camera is then directed toward the femur and moved proximally; this will bring the longitudinal fibers of the vastus lateralis into view. The fibers of the vastus lateralis can then be traced up to the insertion at the vastus tubercle. By looking immediately anterior to this structure, the anterior facet of the greater trochanter can be easily identified, as can be the insertions of the gluteus medius and minimus [10, 11, 13].

A thorough bursectomy is complete when it extends to the insertion of the gluteus maximus and when the gluteus medius and minimus

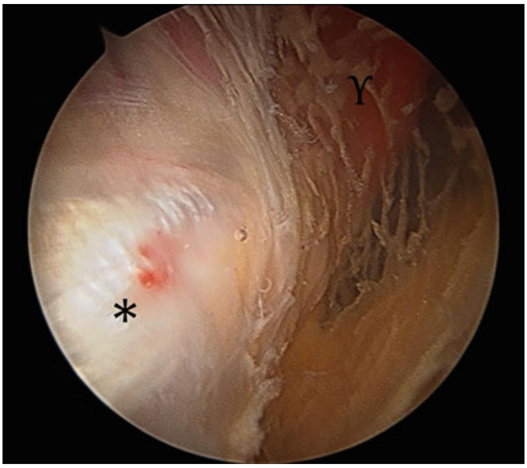
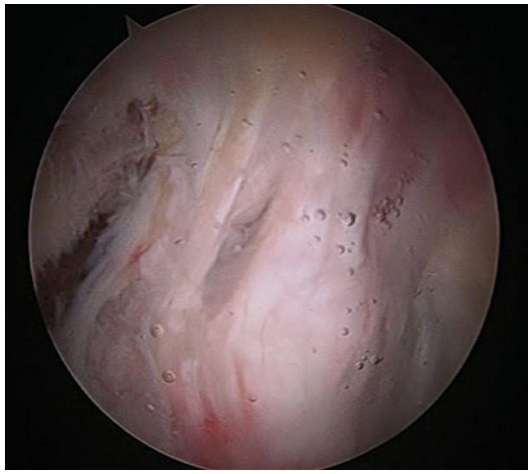


Fig. 4 Proximal extent of the peritrochanteric space. * gluteus medius tendons, *Y* gluteus medius muscle belly



Video 1 Appearance of the peritrochanteric space after a complete bursectomy

insertions are clearly visualized (Fig. 4). This step is accomplished with a motorized shaver and a radiofrequency device to maintain hemostasis. In addition to a thorough bursectomy, the posterior third of the ITB will often be released (Video 1). There is evidence that this portion of the procedure can be beneficial in cases where direct wear on the greater trochanter is seen or in patients with known recalcitrant external snapping hip symptoms [11]. This can be performed with either a radiofrequency device, a beaver blade, or a shaver.

Results

Few studies are available that assess the effectiveness of this procedure. Release of the posterior third of the ITB in conjunction with bursectomy has been shown to have favorable results [11, 16]. In a prospective study of 25 patients, Baker et al. showed significant improvement in visual analog pain scores, Harris hip scores, and improvements in SF 36 [10]. As previously described, calcifications can be seen within the gluteal tendons in a similar manner as calcific tendonitis of the rotator cuff. Kandemir has demonstrated effective treatment of this condition with debridement of the calcific deposits and bursectomy for the diagnosis of GTB [17].

Summary

GTB is a very common condition that can be difficult to treat. As it is a diagnosis of exclusion, a thorough and detailed work-up of all associated conditions must be performed. Nonoperative therapy is often quite effective in managing this condition, but when conservative measures fail to produce long-term benefit, surgical intervention can be a reliable and successful treatment in the properly indicated patient.

References

1. Del Buono A, Papalia R, Khanduja V, et al. Management of the greater trochanteric pain syndrome: a systematic review. Br Med Bull. 2012;102:115–31.
2. Fearon AM, Scarvell JM, Neeman T, et al. Greater trochanteric pain syndrome: defining the clinical syndrome. Br J Sports Med. 2013;47(10):649–53.
3. Tibor LM, Sekiya JK. Differential diagnosis of pain around the hip joint. Arthroscopy. 2008;24(12):1407–21.
4. Williams BS, Cohen SP. Greater trochanteric pain syndrome: a review of anatomy, diagnosis and treatment. Anesth Analg. 2009;108(5):1662–70.
5. Blankenbaker DG, Ullrick SR, Davis KW, et al. Correlation of MRI findings with clinical findings of trochanteric pain syndrome. Skeletal Radiol. 2008;37(10):903–9.

6. Kong A, Van der Vliet A, Zadow S. MRI and US of gluteal tendinopathy in greater trochanteric pain syndrome. Eur Radiol. 2007;17(7):1772–83.
7. Westacott DJ, Minns JI, Foguet P. The diagnostic accuracy of magnetic resonance imaging and ultrasonography in gluteal tendon tears–a systematic review. Hip Int. 2011;21(6):637–45.
8. Lustenberger DP, Ng VY, Best TM, et al. Efficacy of treatment of trochanteric bursitis: a systematic review. Clin J Sport Med. 2011;21(5):447–53.
9. Shbeeb MI, O'Duffy JD, Michet Jr CJ, et al. Evaluation of glucocorticosteroid injection for the treatment of trochanteric bursitis. J Rheumatol. 1996;23(12):2104–6.
10. Baker Jr CL, Massie RV, Hurt WG, et al. Arthroscopic bursectomy for recalcitrant trochanteric bursitis. Arthroscopy. 2007;23(8):827–32.
11. Farr D, Selesnick H, Janecki C, et al. Arthroscopic bursectomy with concomitant iliotibial band release for the treatment of recalcitrant trochanteric bursitis. Arthroscopy. 2007;23(8):e901–5.
12. Fox JL. The role of arthroscopic bursectomy in the treatment of trochanteric bursitis. Arthroscopy. 2002;18(7):E34.
13. Voos JE, Rudzki JR, Shindle MK, et al. Arthroscopic anatomy and surgical techniques for peritrochanteric space disorders in the hip. Arthroscopy. 2007;23 (11):1246 e1241–1245.
14. Byrd JW. Hip arthroscopy utilizing the supine position. Arthroscopy. 1994;10(3):275–80.
15. Glick JM, Sampson TG, Gordon RB, et al. Hip arthroscopy by the lateral approach. Arthroscopy. 1987;3(1): 4–12.
16. Govaert LH, van Dijk CN, Zeegers AV, et al. Endoscopic bursectomy and iliotibial tract release as a treatment for refractory greater trochanteric pain syndrome: a new endoscopic approach with early results. Arthrosc Tech. 2012;1(2):e161–4.
17. Kandemir U, Bharam S, Philippon MJ, et al. Endoscopic treatment of calcific tendinitis of gluteus medius and minimus. Arthroscopy. 2003;19(1):E4.

Surgical Technique: Endoscopic Hamstring Repair and Ischial Bursectomy

66

Carlos A. Guanche

Contents

Abstract

With the expansion of knowledge regarding hip pathologies resulting from the arthroscopic treatment of hip disorders have come expanded arthroscopic indications and treatment of injuries previously treated through open methods. The treatment of symptomatic ischial bursitis and hamstring injuries is one such area. In this chapter, the author describes the surgical procedure and discusses the findings and preliminary outcomes in a cohort of the author's first 15 patients undergoing the procedure. The clinical rationale associated with the treatment algorithm is also discussed.

Introduction

Proximal hamstring injuries have been effectively addressed in the past with a variety of open methods [1, 2]. However, the endoscopic management of certain hip and pelvis pathology previously treated with more invasive, open approaches has evolved. The technique described in this chapter is another such evolution. Hamstring injuries are common and can affect all levels of athletes [3–7]. There is a continuum of hamstring injuries that can range from musculotendinous strains to avulsion injuries [3, 4]. Most hamstring strains do not require surgical intervention and resolve with a variety of modalities and rest [3–7]. In some patients, chronic pain can occur at the hamstring origin

C.A. Guanche
Southern California Orthopedic Institute, Van Nuys, CA, USA
e-mail: cguanche@scoi.com

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_77

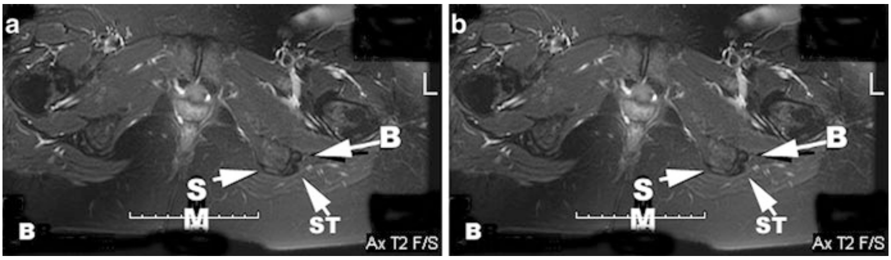


Fig. 1 Normal anatomy of hamstring origin on ischium (**a**) Cadaveric dissection of the ischium in a right hip, viewed from the posterior aspect (Note the everted tendon of the biceps/semitendinosus (B/ST) that is elevated and retracted laterally. *SN* Sciatic nerve). (**b**) Axial T2 weighted magnetic resonance image depicting the anatomy of the hamstring origin in a left hip. *SM* Origin of semimembranosus, *B* Biceps origin, *ST* Semitendinosus

from either partial or complete tears as well as from chronic ischial bursitis. The technique described in this chapter allows for the treatment of many of these problems in an endoscopic fashion, with a minimum risk and maximum diagnostic capability.

The hamstrings originate from the ischial tuberosity, with the exception of the short head of the biceps femoris, and insert distally below the knee on the proximal tibia. The tibial branch of the sciatic nerve innervates the semitendinosus and semimembranosus, and the peroneal branch of the sciatic nerve innervates the long head of the biceps femoris [5].

The proximal hamstring complex has a strong bony attachment on the ischial tuberosity (Fig. 1). The footprint on the ischium includes the semitendinosus and the long head of biceps femoris, which begin as a common proximal tendon and footprint and a distinct semimembranosus footprint [8]. The semimembranosus footprint is medial (and distal) to the crescent-shaped footprint of the common origin of the semitendinosus and long head of the biceps femoris, which is more lateral (Fig. 1).

Most acute injuries usually involve a traumatic event that includes forced hip flexion and knee extension. Most commonly, this has been described in waterskiing [9, 10]. However, the injury can result from any sporting activity that involves rapid acceleration and deceleration [10, 11].

Proximal hamstring tears can be categorized as complete avulsions, partial avulsions, apophyseal avulsions, and degenerative (tendinosis) avulsions [11]. Degenerative tears of the hamstring origin are insidious in onset and are commonly seen as an overuse injury, especially in runners. The mechanism of injury, presumably, is repetitive irritation of the medial aspect of the hamstring tendon (along the lateral aspect of the tuberosity, where the bursa resides). This causes an attritional tear of the tendon and chronic pain.

Commonly, athletes with proximal hamstring tendon tears describe a popping or tearing sensation with associated acute pain and bruising over the posterior hip and thigh [12, 13]. Occasionally, patients who present with either acute or chronic tears may complain of a pin and needle sensation in the sciatic nerve distribution [13, 14]. This may be due to acute compression from hematoma in the proximity of the sciatic nerve, acute traction-type injury in addition to the tendinous rupture, or chronic scarring and tethering of the tendon to the nerve. Similarly, symptoms of ischial bursitis include buttock or hip pain, as well as localized tenderness overlying the ischial tuberosity. Additional symptoms of chronic ischial bursitis may include tingling into the buttock with radiation down the leg [13].

Advanced imaging is crucial to obtain in cases of partial tears with chronic pain. Standard radiographs of the pelvis and a lateral of the affected hip are performed first to rule out any bony problems, in particular apophyseal avulsions of the ischial tuberosity in younger patients (Fig. 2). Most commonly, no fractures are identified and MRI is utilized to assess the proximal hamstring origin. A complete rupture of all three tendons is common and easily identified on MRI.

Partial hamstring origin tears, however, are more difficult to discern. For two tendon tears,

Fig. 2 AP radiograph of right hip showing a bony avulsion of the ischial tuberosity (*arrow*) in a 13-year-old runner

which commonly have an associated musculotendinous junction injury to the third tendon (semimembranosus), this is especially difficult. Most commonly, there is an avulsion of the common semitendinosus and biceps origin, with the semimembranosus remaining intact [7]. In addition, partial tears without any significant retraction can be seen on MRI as a sickle sign and are partial avulsions of the semimembranosus (Fig. 3).

Nonoperative treatment is most commonly recommended in the setting of low-grade partial tears and insertional tendinosis. Initial treatment consists of relative rest, oral nonsteroidal anti-inflammatory medications, and a physical therapy regime [15]. An ultrasound-guided corticosteroid injection may be used and has been shown to provide initial relief in up to 50 % of patients at 1 month [16]. Although evidence is lacking, platelet rich plasma (PRP) injections might also be considered for recalcitrant cases. Partial tears that remain symptomatic may benefit from surgical debridement and repair, similar to other commonly seen partial tendon tears (patella, quadriceps, and biceps) [17].

There are several series and descriptions of open surgical techniques [12, 13, 18–22]. To date, there has been no report of endoscopic management of these injuries. After developing experience in the open management of these injuries, the author has developed an endoscopic technique that allows a safe approach to the pathologic area. The benefits of this endoscopic approach include avoiding the need for excessive retraction or splitting the gluteus maximus and the use of endoscopic magnification to identify and protect the sciatic nerve. This technique with its inherent advantages may improve the management of these injuries and reduce morbidity.

Surgical Technique

The patient is positioned prone on a standard table that is flat. Bolsters are placed under the chest to support the torso. No effort is made to flex the hips, as this may decrease the potential distance between the ischium and the gluteus maximus, thus obliterating the potential working space. The posterior aspect of the hip is then draped assuring that the leg and thigh are free and can be freely manipulated during the procedure (Fig. 4).

Two portals are created; they are 2 cm medial and lateral to the palpable ischial tuberosity (Fig. 5). A blunt arthroscopic cannula is inserted into the lateral portal and a blunt switching stick in the medial portal. The instruments aim to localize the most prominent aspect of the ischium where the sciatic nerve is furthest away. The lateral portal is first used for the arthroscope and the plane between the gluteus maximus and ischium is developed. The prominence of the ischial tuberosity is identified and the medial and lateral borders are delineated. A 30° arthroscope is employed from the lateral portal and an electrocautery device is placed in the medial portal. Any remaining fibrous attachments between the ischium and the gluteus muscle are released, staying along the central and medial portions of the ischium to avoid coming near the sciatic nerve, which is well lateral (and anterior) to the ischial prominence. Once the tip and medial

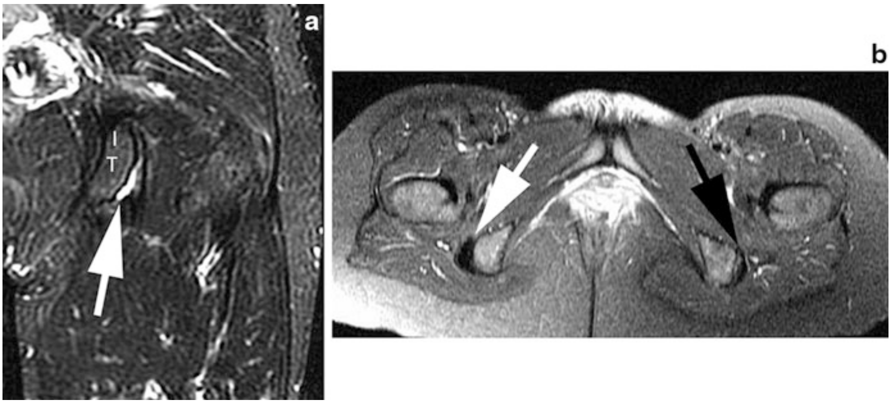


Fig. 3 MRI views of a partial insertional tear with a sickle sign, indicating fluid within the ischial bursa. (**a**) A coronal T2 weighted view of a right hip showing the sickle sign (*white arrow*). *IT* Ischial tuberosity. (**b**) Axial T2 weighted view showing both ischial tuberosities (Note the side with the *black arrow* showing the sickle sign and the normal side for comparison (*white arrow*))

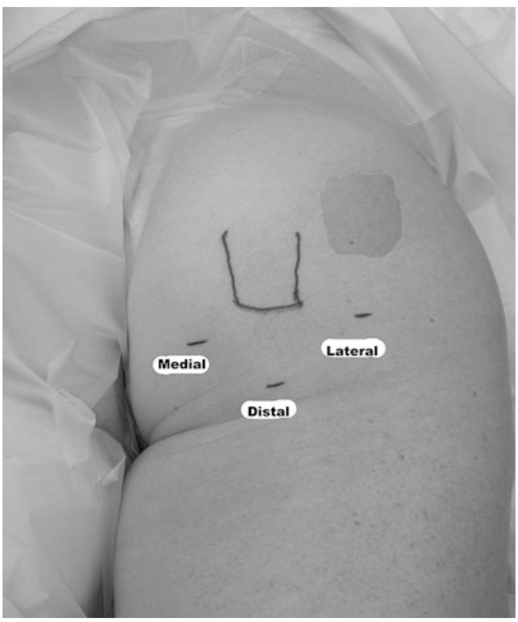


Fig. 4 The patient is positioned prone with the left hip and leg draped free. The outlined prominence is the palpable ischial tuberosity

Fig. 5 In the prone position, the standard portals for endoscopic hamstring repair include a medial and lateral portal that are about 2 cm on either side of the palpable ischium. A third portal can be established about 4 cm distal to the tip of the ischium. The arthroscope is in the medial portal and the cautery is in the lateral portal

aspect of the ischium are delineated, the lateral aspect is then exposed with the use of a switching stick as a soft tissue dissector. With the lateral aspect identified, the dissection continues anteriorly and laterally toward the sciatic nerve (Fig. 6). A careful release of any soft tissue bands is performed in a proximal to distal direction in order to mobilize the nerve and protect it throughout the procedure. This is especially important in cases of acute avulsions, where the nerve may be more difficult to identify secondary to hematoma, early adhesions, or tethering by the retracted tendons. The posterior femoral cutaneous nerve is often the first branch of the nerve identified during dissection and serves as a harbinger of the main sciatic nerve.

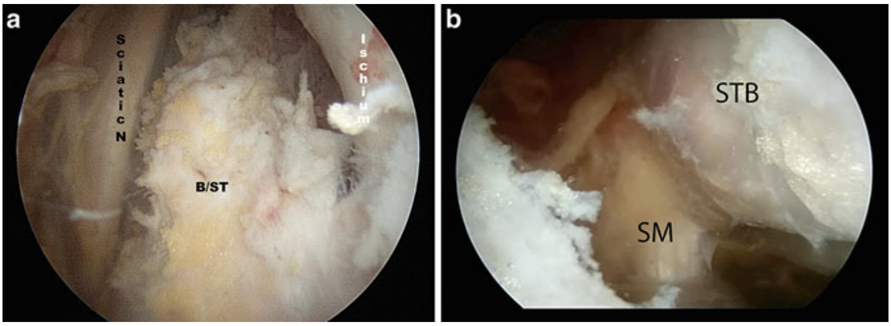


Fig. 6 Endoscopic views of the subgluteal space in a left hip. The arthroscope is in the lateral portal. (**a**) The sciatic nerve (SN) has been exposed along with biceps and semitendinosus avulsion (BST) and the lateral ischium (I). (**b**) The separate attachments of the semimembranosus (SM) tendon, which is more anterior and medial, and the common biceps and semitendinosus (BST) tendon more posterior and lateral

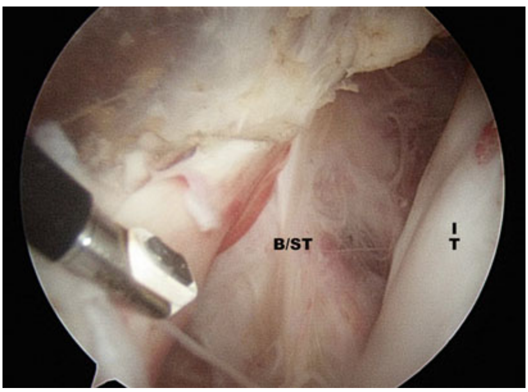


Fig. 7 Endoscopic view of a proximal hamstring origin avulsion in a left hip with the arthroscope in the lateral portal. The distal end of the ischium is seen with fibrous tissue attachment (IT) and the avulsed ischial origin of the common tendon is seen to the left (B/ST)

With the nerve identified and protected, the tendinous origin is then inspected for any obvious tearing (Fig. 7). In acute tears, the tear is obvious and the tendon is often retracted distally. In these cases, there may be a large hematoma that requires evacuation.

Once the area of pathology is identified (in incomplete tears), an endoscopic knife can be employed to longitudinally split the tendon along its fibers (Fig. 8). This area can be identified through palpation, as there is typically softening over the detachment, making the tissue ballotable against the ischium. The hamstring footprint is then undermined and the partial tearing and lateral ischial wall are debrided with an oscillating shaver and burr, if necessary. The devitalized tissue is removed and a bleeding cancellous bed is created. The more distal and inferior ischium and bursa can also be resected and cleared of inflamed tissue. By retracting the tissues, the bursa can be entered and resected (Fig. 9).

A more inferior portal can then be created approximately 4 cm distal to the tip of the ischium and equidistant from the medial and lateral portals (Fig. 5). This portal is employed either for insertion of suture anchors or suture management. Suture passing devices and instruments typically used in arthroscopic rotator cuff repair are then employed to prepare for the tissue approximation. Once all of the sutures are passed through the tissue of the avulsed hamstring, the sutures are tied. In general, one suture anchor is used per centimeter of detachment (Fig. 10).

Postoperatively, the patient is fitted with a hinged knee brace that is fixed at 90° of flexion for 4 weeks to maintain the patient non-weight-bearing. The brace will also serve to restrict excursion of the hamstring tendons and protect the repair. In larger repairs, consideration may be given to using a hip, knee, ankle, and foot orthosis (HKAFO). At 4 weeks, the knee is gradually extended by about 30° per week in order to allow full weight-bearing by 6–8 weeks while maintaining the use of crutches. Physical therapy is instituted at this point, with the initial phase

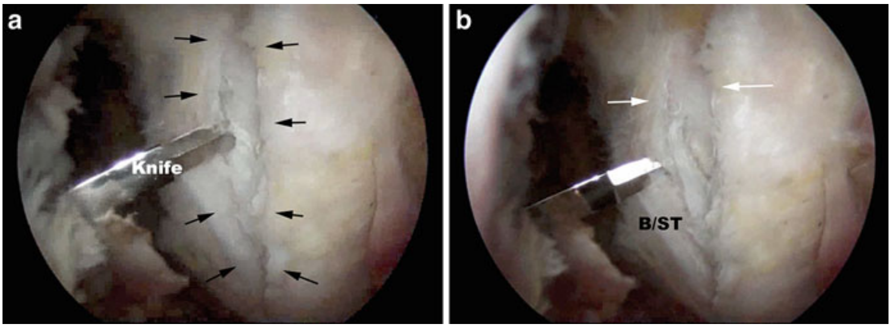


Fig. 8 Endoscopic views of the proximal hamstring origin in a left hip with the arthroscope in the lateral portal. (**a**) Knife has been inserted through the distal portal and the tendon (biceps/semitendinosus) attachment has been incised (along the *arrows*). (**b**) The common biceps/semitendinosus origin has been elevated (seen between the *arrows*). *B/ST* Biceps/semitendinosus complex

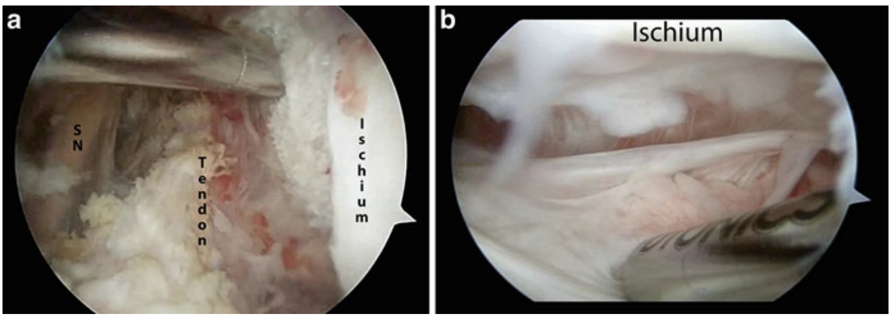


Fig. 9 Endoscopic view of the proximal hamstring origin in a left hip with the arthroscope in the lateral portal. (**a**) The common BST (tendon) origin has been incised and elevated (Note the tool is serving to retract the detached tissue. *SN* Sciatic nerve). (**b**) Ischial bursa prior to debridement (Note the hypertrophic inflammatory tissue in the bursal space)

focused on hip and knee range of motion. Hamstring strengthening is begun at 10–12 weeks, predicated on full range of motion and a painless gait pattern. Full, unrestricted activity is allowed at approximately 4 months.

Patients and Methods

Over the last 18 months, the procedure has been employed by the current author in a group of 15 patients. The first indication is an acute hamstring avulsion in an active patient with greater than 2 cm of retraction (three patients). In nine patients, there was a clinically evident partial hamstring avulsion involving the biceps/semitendinosus tendon, with refractory ischial pain and inability to return to high-level sports. The final three patients had a history of refractory ischial bursitis, with no discernable tear with a failure of conservative treatment including at least 6 weeks of physical therapy and two guided (ultrasound) ischial injections.

Results

At the index procedure, all patients underwent the surgery as described with no need to abandon the procedure as a result of failure of visualization of any of the structures. All of the patients underwent suture anchor fixation with no anchor complications to date. There were two patients that initially complained of numbness over the posterior thigh

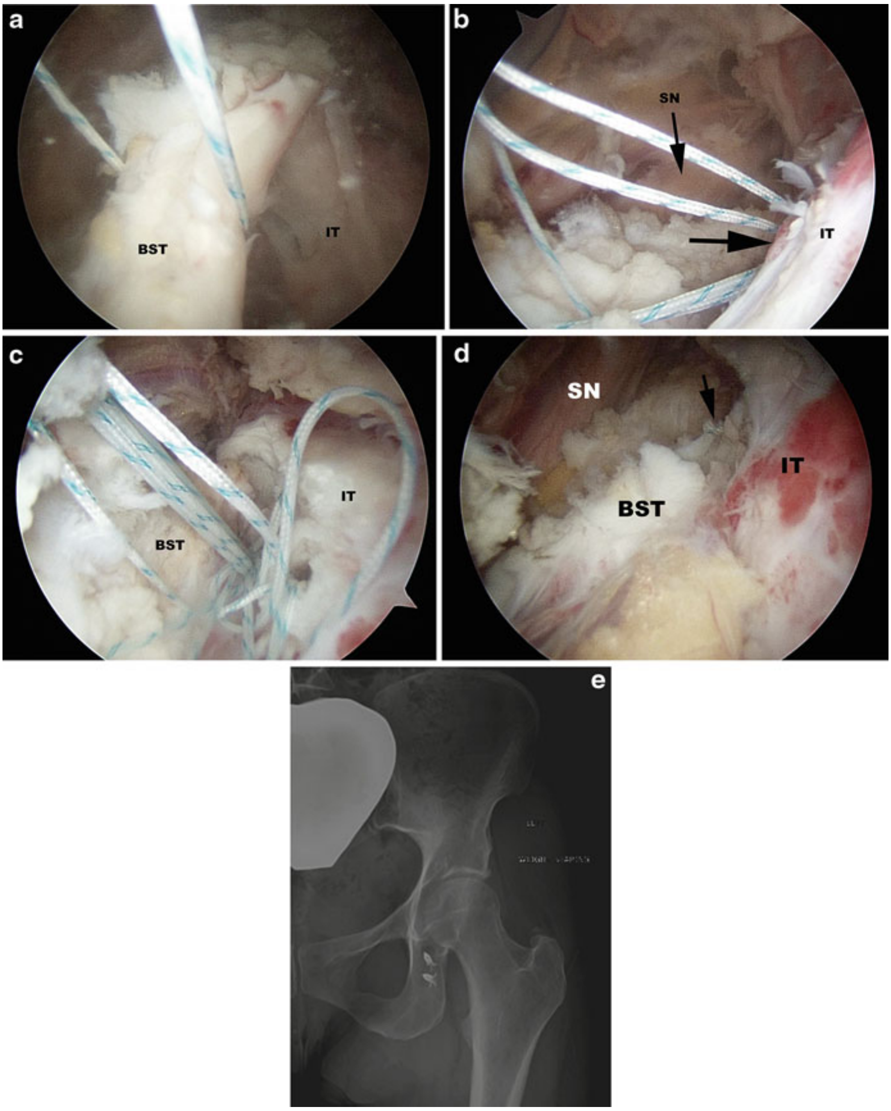


Fig. 10 Endoscopic view of repair of the proximal hamstring origin in a left hip with the arthroscope in the lateral portal. (**a**) The ischium has been prepared and the first suture is in place, retracting the avulsed tendon (BST). *IT* Ischial tuberosity. (**b**) Multiple sutures in place and passed through the tendon (Note the proximity of the sciatic nerve (SN) to the repair site. The *arrow* at the ischial tuberosity (IT) indicates the first anchor insertion point). (**c**) Final mattress sutures in place in the substance of the tendon. Multiple sutures are in place and are to be tightened with knotless anchor configuration (*IT* Ischial tuberosity, *BST* Biceps/semitendinosus). (**d**) Final tendon repair with the tendon edges reattached to the ischial footprint with a suture visualized (*arrow*). The sciatic nerve (SN) is seen to the *left* (*IT* Ischial tuberosity, *BST* Biceps/semitendinosus). (**e**) Radiograph of suture anchors in place in the ischial tuberosity

with resolution of their symptoms by 6 weeks postoperatively. There were no wound complications and no sciatic nerve dysfunction. One patient (with preoperative refractory ischial bursitis) has had a subsequent guided injection as a result of recurrent ischial pain.

Summary

The surgical approach to hamstring repairs has received limited attention. Those patients with partial tears and chronic bursitis are an even smaller percentage of hamstring problems, with few clinical studies available [23]. With the advances seen in hip arthroscopy, further development of endoscopic techniques about the hip and pelvis has allowed us to explore the use of the arthroscope in these previously uncharted areas.

One of the most important aspects in the treatment of proximal hamstring ruptures is early recognition and treatment. With the endoscopic technique, the management may certainly evolve to quicker repair of the problem. Patients with acute repairs have had better outcomes in the literature, when compared to chronic repair [12, 13]. Delayed complications of nonoperative treatment of proximal hamstring ruptures have been described, and these include knee flexion and hip extension weakness, difficulty sitting, and hamstring deformity [24]. The author has employed this technique successfully for several acute ruptures, as well as chronic partial tears.

Surgical repair of proximal hamstring ruptures also has its inherent risks. With open methods, superficial as well as deep wound infections can occur similar to other surgeries; however, the location of the incision can potentially increase this risk. With the endoscopic technique, this possibility may be substantially lessened. Additionally, the three main nervous structures at risk of iatrogenic injury are the posterior femoral cutaneous, inferior gluteal, and sciatic nerves [17, 25]. The sciatic nerve is in close proximity to the ischial tuberosity, running along its lateral aspect. With the endoscopic technique, there is no need for retraction, as the nerve is identified, visualized, and protected during the repair without the need for retraction.

A concern unique to the endoscopic approach is fluid extravasation into the pelvis as a result of the fluid used in the distension of the potential space around the hamstring tendon. The fluid inflow pump is maintained at the lowest possible setting throughout the procedure. In addition, cannulas are established with outflow suction attached at an early stage to prevent fluid egress into the soft tissues around the pathology. An effort should be made to keep track of the total fluid ingress and egress during the case. In addition the operative team should regularly check the abdomen for any evidence of distension. Likewise, any unusual blood pressure decreases may be due to fluid compression from retroperitoneal extravasation and should be closely monitored.

Through the appropriate application of this technique, many chronic hamstring injuries and some acute injuries previously addressed through more invasive, open methods can be effectively addressed endoscopically. Further outcomes research is certainly required to determine the optimal surgical approaches for proximal hamstring disorders.

References

1. Birmingham P, Muller M, Wickiewicz T, et al. Functional outcome after repair of proximal hamstring avulsions. J Bone Joint Surg Am. 2011;93:1819–26.
2. Blasier RB, Morawa LG. Complete rupture of the hamstring origin from a water skiing injury. Am J Sports Med. 1990;18:435–7.
3. Brown T. Thigh. In: Drez DD, DeLee JC, Miller MD, editors. Orthopaedic spors medicine. Principles and practice, vol. 2. Philadelphia: Saunders; 2003. p. 1481–523.
4. Byrd JWT, Polkowski G, Jones KS. Endoscopic management of the snapping iliopsoas tendon. Arthroscopy. 2009;25:e18.
5. Chakravarthy J, Ramisetty N, Pimpalnerkar A. Surgical repair of complete proximal hamstring tendon ruptures in water skiers and bull riders: a report of four cases and review of the literature. Brit J Sports Med. 2005;39:569–72.
6. Clanton TO, Coupe KJ. Hamstring strains in athletes: diagnosis and treatment. J Am Acad Orthop Surg. 1998;6:237–48.
7. Cohen S, Bradley J. Acute proximal hamstring rupture. J Am Acad Orthop Surg. 2007;15:350–5.
8. Cross MJ, Vandersluis R, Wood D, et al. Surgical repair of chronic complete hamstring tendon rupture in the adult patient. Am J Sports Med. 1998;26:785–8.
9. Elliott MC, Zarins B, Powell JW, Kenyon CD. Hamstring muscle strains in professional football players: a 10-year review. Am J Sports Med. 2011;39:843–50.
10. Garrett Jr WE. Muscle strain injuries. Am J Sports Med. 1996;24(6 Suppl):S2–8.
11. Garrett Jr WE, Rich FR, Nikolaou PK, et al. Computed tomography of hamstring muscle strains. Med Sci Sports Exerc. 1989;2:506–14.
12. Klingele KE, Sallay PI. Surgical repair of complete proximal hamstring tendon rupture. Am J Sports Med. 2002;30:742–7.

13. Konan S, Haddad F. Successful return to high level sports following early surgical repair of complete tears of the proximal hamstring tendons. Int Orthop. 2010;34:119–23.
14. Lempainen L, Sarimo J, Heikkila J, et al. Surgical treatment of partial tears of the proximal origin of the hamstring muscles. Brit J Sports Med. 2006;40:688–91.
15. Lempainen L, Sarimo J, Mattila K, et al. Proximal hamstring tendinopathy: results of surgical management and histopathologic findings. Am J Sports Med. 2009;37:727–34.
16. Martin HD, Shears SA, Johnson JC, et al. The endoscopic treatment of sciatic nerve entrapment/deep gluteal syndrome. Arthroscopy. 2011;27:172–81.
17. Mendiguchia J, Brughelli M. A return-to-sport algorithm for acute hamstring injuries. Phys Ther Sport. 2011;12:2–14.
18. Harris JD, Griesser MJ, Best TM, et al. Treatment of proximal hamstring ruptures – a systematic review. Inter J Sports Med. 2011;32:490–5.
19. Mica L, Schwaller A, Stoupis C, et al. Avulsion of the hamstring muscle group: a follow-up of 6 adult non-athletes with early operative treatment: a brief report. World J Sur. 2009;33:1605–10.
20. Miller SL, Gill J, Webb GR. The proximal origin of the hamstrings and surrounding anatomy encountered during repair. A cadaveric study. J Bone Joint Surg Am. 2007;89A:44–8.
21. Orava S, Kujala UM. Rupture of the ischial origin of the hamstring muscles. Am J Sports Med. 1995;23:702–5.
22. Puranen J, Orava S. The hamstring syndrome. A new diagnosis of gluteal sciatic pain. Am J Sports Med. 1988;16:517–21.
23. Sallay PI, Friedman RL, Coogan PG, et al. Subjective and functional outcomes following surgical repair of complete ruptures of the proximal hamstring complex. Orthopedics. 2008;31:1092–6.
24. Sarimo J, Lempainen L, Mattila K. Complete proximal hamstring avulsions: a series of 41 patients with operative treatment. Am J Sports Med. 2008;36:1110–5.
25. Zissen MH, Wallace G, Steven KJ, et al. High hamstring tendinopathy: MRI and ultrasound imaging and therapeutic efficacy of percutaneous corticosteroid injection. Am J Roentgen. 2010;195:993–8.

67 Surgical Technique: Endoscopic Gluteus Medius Repair

James E. Voos and Christopher M. Shaw

Contents

J.E. Voos (✉)
Division of Sports Medicine, Department of Orthopaedic Surgery, University Hospitals Case Medical Center, Cleveland, OH, USA
e-mail: jvoos@osmckc.com; voosjames@gmail.com

C.M. Shaw (✉)
University of Missouri-Kansas City, Kansas City, MO, USA
e-mail: cmshaw5@gmail.com

Abstract

The constellation of hip abductor pathology, trochanteric bursitis, and a tight iliotibial band has been termed "greater trochanteric pain syndrome." Gluteus medius and minimus tendon tears have been referred to as "rotator cuff tears of the hip." Conservative treatment consists of relative rest, NSAIDs, physical therapy, and trochanteric bursa injections. When conservative treatment fails and hip abductor pathology has been confirmed on examination and MRI, surgery is indicated. Endoscopic gluteus medius and minimus tendon repair has been described with good early clinical outcomes with minimal complications reported.

Introduction

It is common, when describing something that is foreign, to put it in terms that are more familiar. It is exactly this practice which has led some authors to call tears of the gluteus medius and minimus tendons "rotator cuff tears of the hip" [1–3]. Tears of the "rotator cuff of the hip" have been identified during open treatment of femoral neck fractures [1], open debridement of recalcitrant trochanteric bursitis [2], total hip arthroplasty [4], and treatment of gluteus medius calcific tendinitis [5, 6]. While the anatomy of both areas leads to natural comparison, it is only with the recent improvements in arthroscopic equipment and techniques

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_71

that endoscopic treatment of hip pathology has been described [7, 8].

Along with the increased interest in arthroscopic treatment of hip pathology has come the advancement of endoscopic techniques of peritrochanteric disorders such as trochanteric bursitis, calcific tendonitis, snapping iliotibial band, and full- and partial-thickness tears of the gluteus complex. Collectively, these disorders causing lateral hip pain have been termed “greater trochanteric pain syndrome” [9]. In most instances, these entities have effectively been treated with nonoperative means. Open trochanteric bursectomy has been described for refractory cases [4, 10]. Baker et al. published a prospective follow-up in 25 patients treated with endoscopic bursectomy at a mean follow up of 26.1 months. Using visual analog pain scores, modified Harris Hip Scores, and SF-36 scoring, significant improvement was noted [11]. Kandemir et al. described endoscopic treatment of trochanteric bursitis and debridement of calcific tendonitis of the gluteus medius and minimus tendons in 2003 [12]. More recently, in 2007, Voos et al. published the description of the endoscopic evaluation of the peritrochanteric space including the gluteus medius and minimus tendons [13]. A second study by Voos et al. reported on 10 patients who underwent endoscopic gluteus medius tendon repair with an average of 2 years follow-up. Sixty percent of patients treated endoscopically reported an acute injury as the cause of lateral hip pain. All patients had complete resolution of pain, maintained full hip range of motion, and 9 out of 10 achieved 5 out of 5 abductor muscle strength [7]. Subsequently, Domb et al. have reported an additional technique for treatment of partial-thickness tears [8]. Domb et al. also published outcomes of endoscopic gluteus medius tendon repairs in 15 patients with an average of 27.9 months follow-up [14]. Six cases were partial-thickness tears and nine were full thickness treated with suture anchor repair. Fourteen of the 15 patients showed postoperative improvement in hip-specific scores used to assess outcome, and good/excellent satisfaction rates after surgery were reported for 14 of 15 patients.

Indications

A thorough patient history is critical when evaluating hip pain. Patients with tears of the gluteus medius often report lateral hip pain with activity with additional discomfort sleeping on the affected hip at night. The most common cause of lateral sided hip pain is trochanteric bursitis and/or a tight iliotibial band. Referred pain from the lumbar spine should also be on the differential diagnosis. Gluteus medius tendon tears are suspected when treatment of lateral sided hip pain has failed to respond to rest, physical therapy, corticosteroid injections, and anti-inflammatory medications [7].

In conjunction with a history of failed conservative treatment, a focused physical examination should be performed. The gluteus medius should be palpated from its origin on the anterior and middle aspect of the ileum to its two insertions on the middle and superoposterior facets of the greater trochanter [13]. Physical examination evaluating muscle strength can help decipher abductor strength with or without pain. Patients with tears of the gluteus complex will often have weakness with resisted hip abductor testing, and they may demonstrate a positive Trendelenburg sign.

Radiographs of the hip are often normal but may show calcifications at the level of the greater trochanter. Magnetic resonance imaging confirms the diagnosis. As with rotator cuff tears, the gluteus medius is assessed for tendon quality, tendon retraction, and the presence of muscle atrophy.

Endoscopic gluteus medius tendon repair is indicated in patients who have failed conservative treatment and who demonstrate positive lateral hip pain and gluteus medius tendon weakness and patient willingness to undergo the necessary postoperative rehabilitation. Contraindications to endoscopic repair include full-thickness tears with retraction and/or significant fatty atrophy of the gluteus medius/minimus muscles. Open gluteus medius/minimus tendon repair is indicated in these cases. In contrast to open rotator cuff repair where violation of the deltoid may have detrimental functional consequences, open abductor repair with division of the iliotibial band is less morbid and can be performed through a small incision. Additional relative contraindications to an endoscopic approach include a positive smoking status, severe degenerative changes in the hip, and inability to comply with postoperative restrictions. In addition, if there is concern for length of operative time or soft tissue swelling, then open repair is again considered. As in the rotator cuff, platelet-rich plasma (PRP) augmentation of repairs may be considered although there is no clinical data to support its use at this point. Finally, when an irreparable tear is present with retraction and muscle atrophy, an open gluteus maximus tendon transfer is a salvage option.

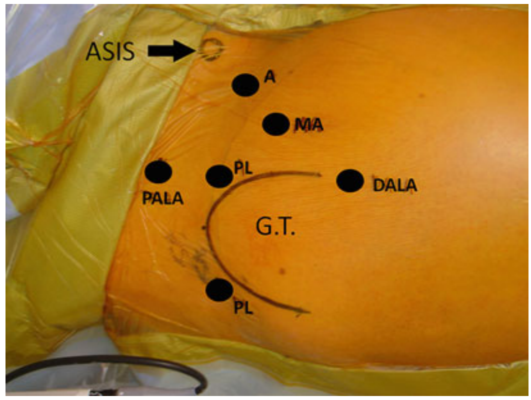


Fig. 1 Intraoperative image of a right hip demonstrating the placement of the anterolateral (AL), mid-anterior (MA), distal anterolateral accessory (DALA), posterolateral (PL), and proximal anterolateral accessory (PALA) portals for use in the peritrochanteric space. Anterior superior iliac spine (*Black arrow*; ASIS). Greater Trochanter (G.T.)

Surgical Technique

Diagnostic Endoscopy and Trochanteric Bursectomy

At the initiation of the procedure, the central compartment is accessed under traction in cases of coexisting/concomitant labral or chondral pathology. After evaluation of the intra-articular hip joint proper is complete, traction is released. The pudendal post is removed in order to relieve pressure on the skin and reduce the risk of neuropraxia. Peritrochanteric endoscopy can be performed in either the supine [7–9, 12–15] or lateral [16–20] position. The senior author performs the procedure in a modified supine position with the table rotated 30° laterally away from the surgical limb.

Accurate placement of the portals is paramount to successfully perform the procedure in a timely manner. Both standard portals used during arthroscopy and additional accessory portals to access the peritrochanteric space are employed (Fig. 1). Access to the space is achieved through a mid-anterior portal established slightly more lateral and distal than the traditional anterior portal in order to decrease the risk of injury to the lateral femoral cutaneous nerve. Fluoroscopy is used to assist in placement of the mid-anterior portal over the lateral prominence of the greater trochanter. The pump pressure is set at 40–50 mmHg to avoid over distension of the space and prevent excessive fluid extravasation. A blunt trochar is then inserted into the mid-anterior portal. The trochar is used to sweep between the iliotibial band and the vastus ridge in a manner similar to clearing the subacromial space during shoulder arthroscopy. Care is taken to avoid placing the trochar too proximal in order to avoid damage to the gluteus medius musculature or too distal to avoid damage to the fibers of the vastus lateralis. Slight traction without distraction of the joint may be utilized to tension the abductors during initial portal placement. Gentle tension on the abductors is performed either by slightly adducting the leg or retaining the pudendal post.

After the peritrochanteric space has been accessed, visualization is achieved with a 70° arthroscope in the mid-anterior portal. Diagnostic evaluation begins with visualization of the gluteus maximus tendon distally as it inserts on the femur just below the vastus lateralis (Fig. 2). Exploration posterior to the tendon is cautioned to avoid injury to the sciatic nerve. The camera is then directed towards the lateral femur and the longitudinal fibers of the vastus lateralis. The tendinous insertion and muscle of the gluteus medius is identified as the camera is moved further proximal.

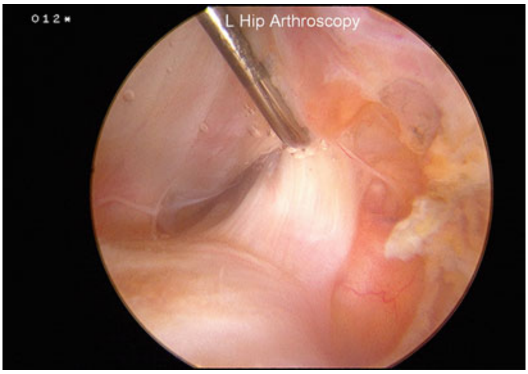


Fig. 2 Intraoperative image of a left hip demonstrating the probe at the insertion of the gluteus maximus tendon to the posterior portion of the femur. This is the distal extent of the peritrochanteric space

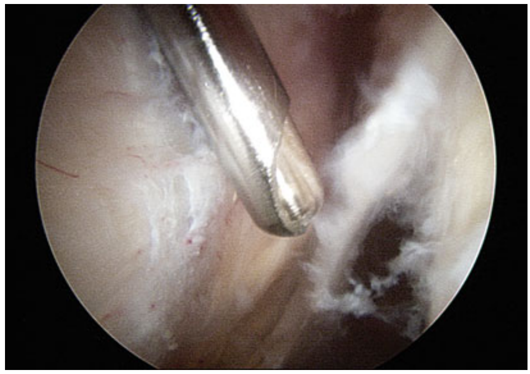


Fig. 3 Intraoperative image of a left hip demonstrating thick bands of trochanteric bursa tissue removed by the arthroscopic shaver. The vastus lateralis is to the *left*

Inspection of the peritrochanteric space is complete as the iliotibial band is identified by looking proximal and lateral.

Additional authors have reported alternative portals to access the peritrochanteric space in order to perform a trochanteric bursectomy and or iliotibial band lengthening [16–20]. Farr et al. described a technique involving two incisions: one 4 cm proximal to the greater trochanter along the anterior border of the iliotibial band and the other 4 cm distal and along the posterior border. A 30° arthroscope was introduced through the distal portal for viewing while the proximal portal was used as the working portal [20].

A thorough trochanteric bursectomy is performed first (Fig. 3). A spinal needle is placed under direct visualization 4–5 cm distal to the anterolateral portal to create a distal anterolateral accessory portal. The arthroscopic shaver is inserted through this portal, and a thorough trochanteric bursectomy is performed over the distal portion of the space. The bursectomy is performed from distal to proximal. Thickened bursal tissue and fibrinous bands are cleared off of the gluteus maximus tendinous insertion distally. A second accessory portal is created 2–3 cm proximal to the anterolateral portal called the proximal anterolateral accessory portal to remove the most proximal portions of the inflamed bursal tissue. Arthroscopic electrocautery can also be used to cauterize bursal vessels and complete the bursectomy.

If concomitant symptomatic external "snapping hip" or coxa saltans is present, lengthening of the iliotibial band is performed. A beaver blade or arthroscopic cautery device is used to create a transverse or cruciate-style lengthening of the iliotibial band [9, 12, 18].

Gluteus Medius Repair

Prior to repair of the hip abductor tendons, it is important to understand the anatomy and insertional footprint of the gluteus medius and minimus. Robertson et al. described the anatomy and dimensions of the abductor footprint insertion in a cadaveric study [21]. The four facets of the greater trochanter include the anterior, lateral, superoposterior, and posterior facets. The gluteus medius inserts onto the superoposterior and lateral facets of the trochanter, with the most robust portion of the tendon inserting on the superoposterior facet in a circular footprint pattern. The superoposterior insertion has a circular shape with an approximate diameter of 17 mm and a mean area of insertion of 196.5 mm^2, while the lateral facet footprint is approximately 35 mm long and 11 mm wide with a mean area of insertion of 438.0 mm^2. The gluteus minimus originates on the outside of the ilium between the anterior and inferior gluteal lines and from the sciatic notch, where the muscle protects the superior gluteal nerve and artery. The gluteus minimus has a broad insertion running from the hip capsule anterosuperiorly to its major insertion on the anterior facet of the greater trochanter deep to the gluteus medius. A bald spot of the trochanter, where there are no distinct tendon insertions, exists between the insertion of the gluteus minimus on the anterior facet and the gluteus medius on the lateral facet. The posterior facet of the greater trochanter lacks a tendon insertion and lies posterior to the lateral facet and inferior to the superoposterior facet with the trochanteric bursa overlying it.

Slight traction of the hip is needed to place the gluteus medius muscle fibers on tension in order to delineate proximal bursal tissue from gluteus medius muscle fibers. The 70° arthroscope is placed in the proximal anterolateral accessory portal to achieve a more global view of the abductors, while the working instruments can be placed in the mid-anterior and distal anterolateral accessory portal. The tears can be classified as intrasubstance, partial thickness, or full thickness [7, 8, 15, 21]. The tear location is often on the undersurface analogous to an articular sided rotator cuff tear, which extends posteriorly demonstrating a full-thickness tear.

Endoscopic techniques and fundamentals for gluteus tendon repair are analogous to rotator cuff repairs with the goal of a secure, tension-free repair, with restoration of the tendon footprint.

After the tendon is identified, the torn edge of the tendon is gently debrided to stable tissue. Next, a grasper is used to manually reduce the tear to its anatomic position on the footprint. An arthroscopic burr is used to decorticate the greater tuberosity footprint to a bleeding base (Fig. 4). A spinal needle is placed to localize the best angle for anchor placement. Bioabsorbable or metallic suture anchors are then inserted into the footprint. Fluoroscopic guidance is useful to confirm proper anatomic positioning of the anchors.

Arthroscopic plastic cannulas 7–8.25 mm in size are then inserted through the working portals for instrumentation and suture management. A suture-passing device is used to pass suture through the tendon from posterior to anterior sequentially. Voos et al. described using single- or double-loaded suture anchors with passage of

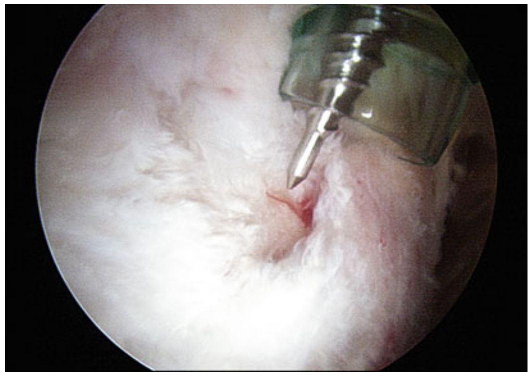


Fig. 4 Intraoperative image of a left hip demonstrating a longitudinal tear in the gluteus medius tendon. The footprint has been decorticated in preparation for anchor placement. An awl is utilized to prepare a pilot whole for the anchor

sutures in a simple fashion [7, 9]. Domb et al. published a technique for repair of partial-thickness undersurface gluteus medius tendon tears [8, 14]. In the technique described by Domb et al., a beaver blade is used to fashion a longitudinal split in the midsubstance of the lateral facet insertion of the gluteus medius. Through this split, the undersurface tearing and pathologic tendon tissue can be assessed. A suture passer used to pass one limb of each suture through the anterior part of the tendon and one limb of each suture through the posterior part. The steps may be repeated with a second anchor if necessary. This technique results in a side-to-side repair of the longitudinal tendon split. An arthroscopic suture-passing device that retrieves the suture (Fig. 5) such as the Scorpion (Arthrex Naples, Florida) and suture penetrators (Fig. 6) assists in ease of maneuvering in the peritrochanteric space.

After all sutures are passed, arthroscopic sliding, locking knots are tied with a knot pusher securing the medius back to its native footprint on the greater trochanter (Fig. 7). Transosseous and double-row techniques have also been described in recent studies [14, 15]. Dishkin-Paset et al. reported the results of a cadaveric biomechanical study comparing double-row – massive cuff stitch constructs to double row – knotless anchor constructs [15]. The biomechanical stability of the two constructs for gluteus medius

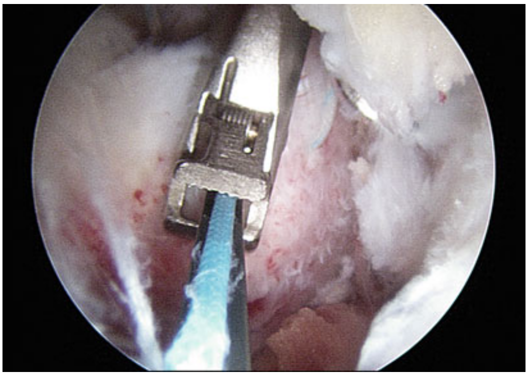


Fig. 5 Intraoperative image of a left hip demonstrating suture passage through the anterior and posterior margins of the gluteus medius tendon tear with a suture-passing device and penetrator

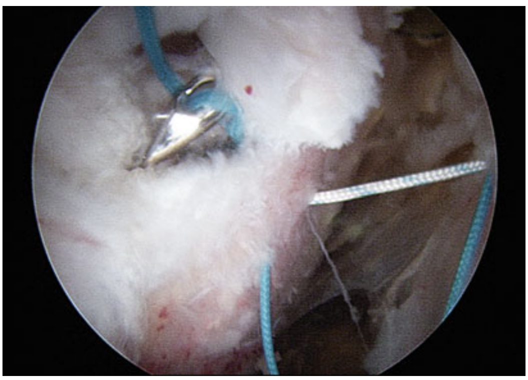


Fig. 6 Intraoperative image of a left hip demonstrating suture passage through the anterior and posterior margins of the gluteus medius tendon tear with a suture-passing device and penetrator

tendon repair was similar. The study also cautioned that performance of the knotless anchors was affected by bone mineral density.

Rehabilitation

The postoperative course following this endoscopic technique consists of 6 weeks of 20 lb foot flat weight bearing with crutches. A hip abduction brace is employed postoperatively and is worn for 6 weeks to prevent active abduction, while hip flexion and extension are permitted. At the end of 6 weeks, isometric strengthening of hip abductors is initiated. At 12 weeks, plyometric and progressive strengthening is begun. Once the patient can demonstrate equivalent strength of abduction bilaterally, they are then released to progressive sports-specific activities.

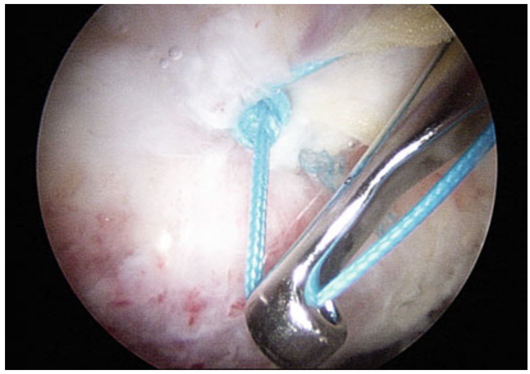


Fig. 7 Intraoperative image of a left hip demonstrating the final gluteus medius tendon repair after the sutures have been tied

Summary

Partial-thickness and full-thickness tears of the gluteus medius tendon can be addressed endoscopically with good clinical results in small case series. Appropriate patient selection and rehabilitation are critical to a successful clinical outcome. Surgical principals employed in shoulder rotator cuff repair of tendon mobilization, footprint preparation and restoration, and tension-free repair are translated to the hip in this setting.

References

1. Bunker TD, Esler CN, Leach WJ. Rotator-cuff tear of the hip. J Bone Joint Surg Br. 1997;79:618–20.
2. Kagan II A. Rotator cuff tears of the hip. Clin Orthop Relat Res. 1999 Nov;368:135–40.
3. Dwek J, Pfirrmann C, Stanley A, Pathria M, Chung CB. MR imaging of the hip abductors and commonly encountered pathology at the greater trochanter. Magn Reson Imaging Clin N Am. 2005; 13:691–704. vii.
4. Govaert LH, van der Vis HM, Marti RK, Albers GH. Trochanteric reduction osteotomy as a treatment for refractory trochanteric bursitis. J Bone Joint Surg Br. 2003;85(2):199–203.

5. Callaghan BD. Unusual calcification in the region of the gluteus medius and minimus muscles. Australas Radiol. 1977;21(4):362–6.
6. Gordon EJ. Trochanteric bursitis and tendinitis. Clin Orthop. 1961;20:193–202.
7. Voos J, Shindle M, Pruett A, Asnis P, Kelly B. Endoscopic repair of gluteus medius tendon tears of the hip. Am J Sports Med. 2009;37(4):743–7.
8. Domb BG, Nasser RM, Botser IB. Partial-thickness tears of the gluteus medius: rationale and technique for trans-tendinous endoscopic repair. Arthrosc J Arthrosc Relat Surg. 2010;26(12):1697–705.
9. Barnthouse NC, Wente TM, Voos JE. Greater trochanteric pain syndrome: endoscopic treatment options. Oper Tech Sports Med. 2012;20:320–4.
10. Zoltan DJ, Clancy Jr WG, Keene JS. A new operative approach to snapping hip and refractory trochanteric bursitis in athletes. Am J Sports Med. 1986;14(3):201–4.
11. Baker Jr CL, Massie RV, Hurt WG, Savory CG. Arthroscopic bursectomy for recalcitrant trochanteric bursitis. Arthroscopy. 2007;23(8):827–32.
12. Kandemir U, Bharam S, Phillipon MJ, Fu FH. Endoscopic treatment of calcific tendonitis of gluteus medius and minimus. Arthroscopy. 2003;19(1):E4.
13. Voos JE, Rudzki JR, Shindle MK, Martin HM, Kelly BT. Arthroscopic anatomy and surgical techniques for peritrochanteric space disorders in the hip. Arthrosc J Arthrosc Relat Surg. 2007;23(11): 1246e1. 5.
14. Domb BG, Botser I, Giordano BD. Outcomes of endoscopic gluteus medius repair with minimum 2-year follow-up. Am J Sports Med. 2013;41(5):988–97.
15. Dishkin-Paset JG, Salata MJ, Gross CE, Manno K, Shewman EF, Wang VM, Bush-Joseph CA, Nho SJ. A biomechanical comparison of repair techniques for complete gluteus medius tears. Arthroscopy. 2012. [Epub ahead of print]
16. Wiese M, Rubenthaler F, Willburger RE, Fennes S, Haaker R. Early results of endoscopic trochanter bursectomy. Int Orthop. 2004;28(4):218–21.
17. Baker Jr CL, Massie RV, Hurt WG, Savory CG. Arthroscopic bursectomy for recalcitrant trochanteric bursitis. Arthroscopy. 2007;23(8):827–3.
18. Ilizaliturri Jr VM, Martinez-Escalante FA, Chaidez PA, Camacho-Galindo J. Endoscopic iliotibial band release for external snapping hip syndrome. Arthroscopy. 2006;22(5):505–10.
19. Ilizaliturri Jr VM, Camacho-Galindo J. Endoscopic treatment of snapping hips, iliotibial band, and iliopsoas tendon. Sports Med Arthrosc. 2010;18(2):120–7.
20. Farr D, Selesnick H, Janecki C, Cordas D. Arthroscopic bursectomy with concomitant iliotibial band release for the treatment of recalcitrant trochanteric bursitis. Arthroscopy. 2007;23(8):905.e1. e5.
21. Voos JE, Maak T, Kelly BT. The peritrochanteric space. In: Sekiya J, Safran M, Ranawat A, Leunig M, editors. Techniques in hip arthroscopy and joint preservation surgery. London: Elsevier; 2010.

Surgical Technique: Endoscopic Iliotibial Band Lengthening

68

John M. Arnold, Champ L. Baker III, and Champ L. Baker Jr.

Contents

Abstract

Disorders of the peritrochanteric region include the following three entities: external coxa saltans/external snapping hip, greater trochanteric bursitis, and gluteus medius or minimus tears, or both. Previously, these disorders have been grouped together and called "greater trochanteric pain syndrome." In most cases, nonoperative treatment, consisting of local corticosteroid and anesthetic injections combined with structured physical therapy regimens, provides a successful outcome. When nonoperative treatment fails, endoscopic trochanteric bursectomy with iliotibial band lengthening, and/or abductor tendon repair can be performed. This chapter will focus on surgical management of iliotibial/external snapping hip and greater trochanteric bursitis.

Introduction

Endoscopic iliotibial band lengthening and trochanteric bursectomy is an effective surgical treatment for greater trochanteric bursitis and external coxa saltans refractory to nonoperative management. This minimally invasive procedure reliably alleviates lateral hip pain and allows expedient return to both normal and athletic activities [1–4].

J.M. Arnold • C.L. Baker III (✉)
The Hughston Clinic, PC, Columbus, GA, USA
e-mail: arnoljm@gmail.com; cbaker3@hughston.com

C.L. Baker Jr.
Department of Orthopaedics, Medical College of Georgia, Augusta, GA, USA
e-mail: cbaker@hughston.com

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_72

Technique

After induction of general anesthesia, the patient is placed in the lateral decubitus position with the affected extremity up and stabilized with an inflatable beanbag or hip positioner system (Fig. 1). Pillows are used to pad all bony prominences. The affected hip and leg are draped freely to allow an assistant to take the hip through a full range of motion. The greater trochanter of the hip is palpated and marked at the most superior, anterior, and posterior aspects. A spinal needle is inserted until it touches the trochanteric prominence. After the needle is withdrawn a few millimeters, approximately 30 mL of saline solution is injected into the bursa. The spinal needle is left embedded in the trochanteric prominence for localization purposes.

Fig. 1 The patient is positioned in the lateral decubitus position with the entire hip and leg draped free. Proximal and distal portals are marked on the skin, and a spinal needle marks the trochanteric prominence

Next, two portals are created: a proximal portal is made approximately 2–3 cm proximal to the most superior aspect of the greater trochanter and a distal portal is made 2–3 cm distal to the trochanteric prominence. The portals are made in line with the needle, slightly anterior to the midpoint, and parallel to the fibers of the IT tract. A 4-mm, 30° arthroscope is then inserted into the subcutaneous tissues directly above the iliotibial band (ITB). A 4.5-mm shaver is inserted into the other portal aiming toward the superior tip of the greater trochanter using triangulation to find the spinal needle. The subcutaneous fat immediately adjacent and adherent to the ITB over the greater trochanter is cleared by the use of the 4.5-mm shaver. Alternatively, an arthroscopic ablator can be used to clear the subcutaneous fat or a combination of shaver and ablator can be used depending on the preference of the surgeon (Figs. 2 and 3). When the ITB can be well visualized, an approximately 5–6-cm longitudinal incision is made with the ablator in line with the ITB fibers slightly posterior to the midportion of the band and the trochanteric prominence (Figs. 4 and 5). This incision exposes the trochanteric bursa. The surgical assistant abducts the leg to further relax the incised ITB and to allow the arthroscope and instruments to be advanced underneath the ITB. A common pitfall is to make the incision too posterior, making it difficult to access the bursa properly. Using the spinal needle to localize the most prominent aspect of the trochanter and completely expose the ITB prior to making the split helps to prevent this problem. If muscle is visualized while making the ITB incision, the surgeon is too posterior and should reassess for proper position.

The bursa and its thick fibrous bands and adhesions are now able to be easily visualized. Debridement of these bands and bursal tissue is performed with the arthroscopic shaver and primarily with the arthroscopic ablator (Fig. 6). Coagulation with the ablator is frequently necessary to maintain hemostasis and visualization. The assistant slowly internally and externally rotates the abducted leg to expose the posterior and anterior portions of the bursa, respectively. Care must be taken with extreme internal rotation because this places the sciatic nerve at risk.

After completion of the bursectomy, the tendinous attachments of the gluteus medius and minimus are inspected for partial- or full-thickness tears. Small partial tears are simply debrided while high-grade partial-thickness and full-thickness tears are repaired with the use of suture anchors as described in the respective

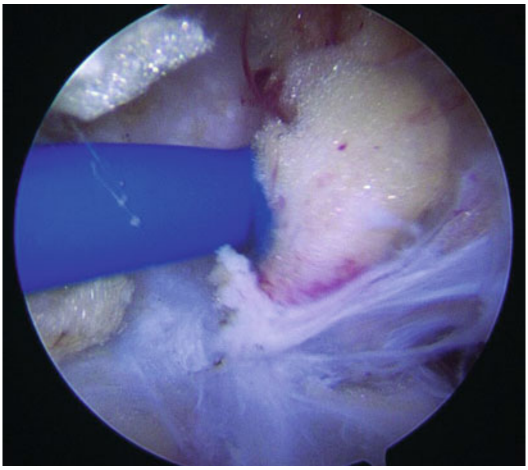


Fig. 2 An arthroscopic ablator is utilized to clear the subcutaneous fat from the underlying iliotibial band

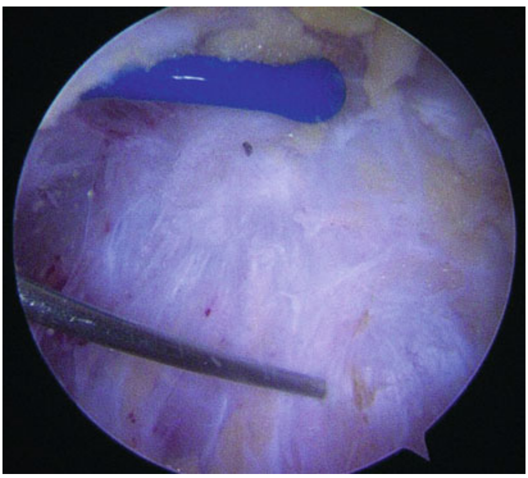


Fig. 3 The iliotibial band is exposed. The spinal needle localizes the most prominent aspect of the greater trochanter

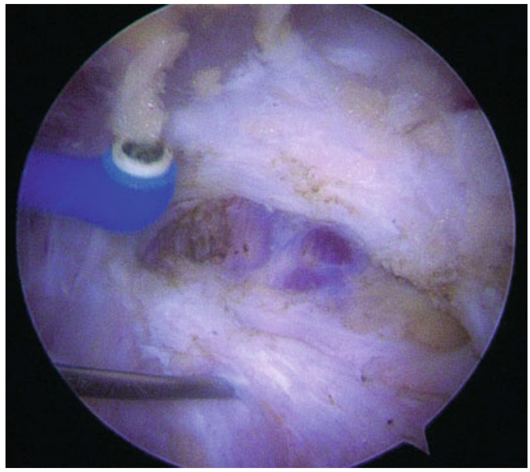


Fig. 4 The arthroscopic ablator is utilized to incise the iliotibial band in *line* with its fibers

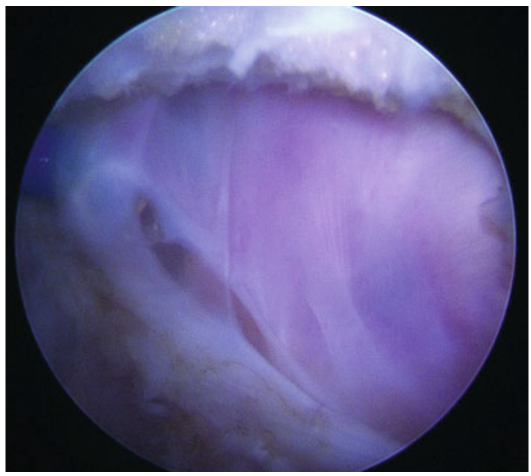


Fig. 5 The iliotibial band is incised for a length of 5–6 cm to expose the trochanteric bursa

chapter. The hip is taken through a gentle range of motion to ensure a complete bursectomy and decompression has been performed.

For patients with an associated external coxa saltans, an ITB lengthening procedure is necessary in addition to the standard trochanteric bursectomy. Several options exist, including an ellipsoidal or diamond-shaped resection of the ITB or a cruciate-type release of the ITB. After making the longitudinal incision in the ITB with the arthroscopic ablator, the anterior and posterior edges of the incised ITB are resected further with the shaver. Additional tissue removal is performed near the center of the midline cut to create an elliptical resection. Because the snapping usually results from a thickened posterior portion of the ITB flipping over the greater trochanter, the posterior resection is slightly greater than the anterior resection. Alternatively, a transverse secondary crisscross incision may be performed creating a cruciate-type release (Figs. 7, 8, and 9).

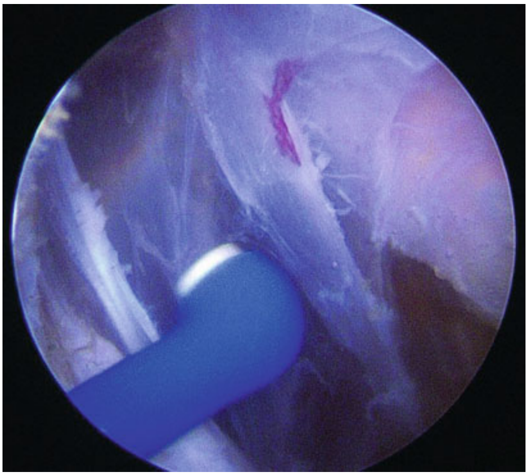


Fig. 6 Fibrous adhesions are released and bursal tissue is resected with the arthroscopic ablator

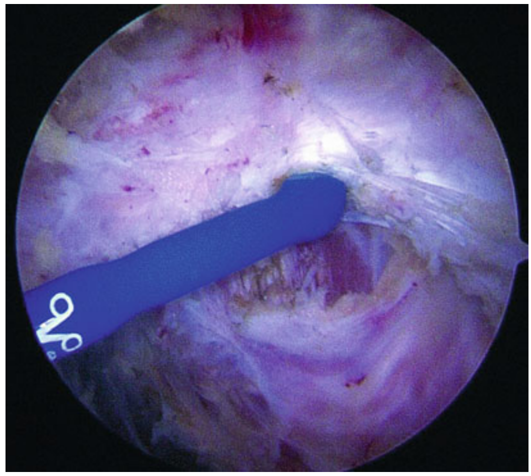


Fig. 8 A transverse cut is made in the anterior iliotibial band

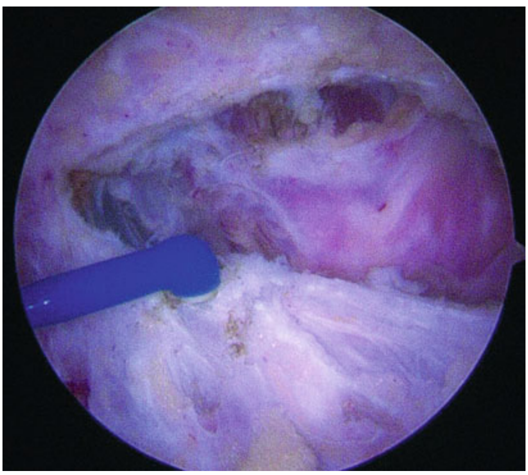


Fig. 7 A transverse cut is made in the posterior iliotibial band

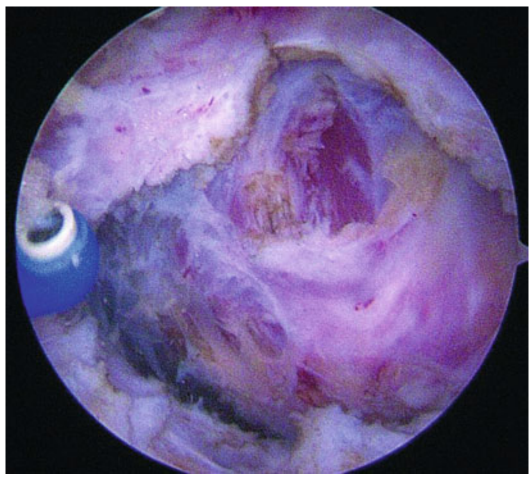


Fig. 9 A cruciate-type release of the iliotibial band has been performed for completion of the release procedure

After completion of the procedure and when hemostasis is achieved, the instruments are withdrawn and any excess fluid is expressed from the portals. The portals are closed with simple nylon suture, and a hip spica compression dressing is applied.

Postoperative Care

The procedure is performed on an outpatient basis. Patients are allowed immediate weight bearing as tolerated with crutches or a walker. Assistive devices are discontinued when the patient demonstrates an independent, nonantalgic gait. Sutures are removed within 1 week of surgery. Strengthening the hip musculature begins as pain tolerance allows, which is typically 1–2 weeks after surgery. After the immediate postoperative period, therapy continues to ensure lumbar spine flexibility, pelvic balance, and appropriate gluteal strength. Activities are allowed as tolerated, and patients with external coxa saltans are allowed a return to full athletic activities at approximately 6 weeks.

The senior author published a retrospective review of 25 patients treated with an endoscopic bursectomy at an average follow-up of 26 months [1]. Pain scores on the visual analog scale (0, no pain; 10, worst pain) improved from a preoperative mean of 7.2 to a postoperative mean of 3.1 at final follow-up. There was marked improvement in the mean Harris Hip Score from 51 preoperatively to 77 at final follow-up. One complication of a seroma occurred requiring drainage, and one patient with continued pain after endoscopic surgery was treated with an open bursectomy with successful resolution of symptoms.

Summary

Most patients with greater trochanteric pain syndrome respond to nonoperative treatment that typically consists of activity modification; nonsteroidal anti-inflammatory medications (NSAIDs); stretching of a tight ITB; physical therapy with modalities, such as heat and ultrasound; and local injections of corticosteroids and anesthetics directly into the bursa. When nonoperative treatment fails, endoscopic trochanteric bursectomy, iliotibial band lengthening, and/or gluteus medius tendon repair can be performed. In patients with associated external *coxa saltans*, an ITB lengthening procedure is necessary in addition to the standard trochanteric bursectomy.

References

1. Baker Jr CL, Massie RV, Hurt WG, Savory CG. Arthroscopic bursectomy for recalcitrant trochanteric bursitis. Arthroscopy. 2007;23:827–32.
2. Fox JL. The role of arthroscopic bursectomy in the treatment of trochanteric bursitis. Arthroscopy. 2002;18:E34.
3. Govaert LHM, van Dijk CN, Zeegers AVCM, Albers GHR. Endoscopic bursectomy and iliotibial tract release as a treatment for refractory greater trochanteric pain syndrome: a new endoscopic approach with early results. Arthrosc Tech. 2012;1:e161–4.
4. Ilizaliturri Jr VM, Martinez-Escalante FA, Chaidez PA, Camacho-Galindo J. Endoscopic iliotibial band release for external snapping hip syndrome. Arthroscopy. 2006;22:505–10.

69 Surgical Technique: Open Gluteus Medius Repair

Thomas H. Wuerz, Adam B. Yanke, and James Rosneck

Contents

Abstract
Gluteus medius tears can be a source of lateral thigh pain. They typically can present as chronic tears, associated with femoral neck fractures or osteoarthritis, or as avulsion after total hip arthroplasty. Various open and endoscopic repair and reconstruction techniques have been described. The focus in this chapter is on an open repair technique utilizing suture anchors and on postoperative rehabilitation.

Introduction

Gluteus medius tears are increasingly being recognized as the source of lateral thigh pain and abduction weakness. Pain over the greater trochanter was previously presumed to be largely from bursitis, but recently gluteus medius tears have been shown to be an etiology [1]. Tears at the insertion of the gluteus medius can be intrasubstance, partial, or complete and can occur either spontaneously or traumatically (see Figs. 1 and 2). Described as the "rotator cuff tears of the hip" and as an underlying cause of chronic greater trochanteric pain syndrome, gluteus medius and minimus avulsions have been reported most commonly in women ranging in age from 40 to 60 years, causing debilitating pain and reduced mobility [2, 3]. Gluteus medius tears have been reported to occur more often in women than in men at a ratio of up to 4:1, occurring in almost 25 % of women in their 60s [4].

T.H. Wuerz (✉) • A.B. Yanke
Hip Preservation Center, Department of Orthopaedic Surgery, Division of Sports Medicine, Rush University Medical Center, Rush Medical College of Rush University, Chicago, IL, USA
e-mail: Thomas_H_Wuerz@rush.edu; thomas.wuerz@googlemail.com; basworth@mac.com

J. Rosneck
Sports Health Center, Orthopaedic and Rheumatologic Institute, Cleveland Clinic, Garfield Heights, OH, USA
e-mail: rosnecj@ccf.org

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_73

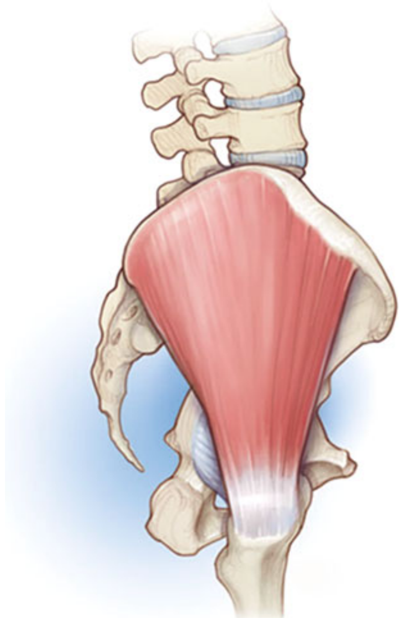


Fig. 1 Lateral view of gluteus medius anatomy and insertion site (Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography © 2013–2014. All rights reserved)

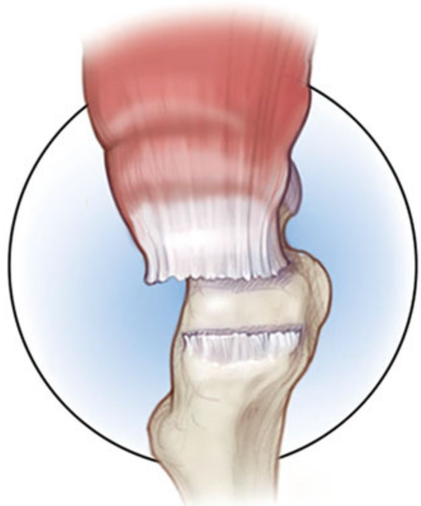


Fig. 2 Schematic depiction of gluteus medius tear (Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography © 2013–2014. All rights reserved)

The incidence of these tears has been shown to increase with history of osteoarthritis and femoral neck fracture. The gluteus medius can be separated into an anterior, middle, and posterior aspect. The anterior and middle aspect consists of vertical fibers. Both are involved in initiating hip abduction and insert at the lateral facet of the greater trochanter [5]. The posterior fibers run horizontally and insert onto the superoposterior facet of the trochanter. The anterior fibers tear most commonly, particularly at the musculotendinous junction. This has proposed to be most likely from chronic microtrauma and degeneration [6]. Local ischemia and differences in anatomy between men and women have also been implicated as risk factors, possibly causing increased breakdown of collagen fibers at the insertion sites on the greater trochanter. Tears can be partial or full. They typically occur at the dual insertion of the anterior and middle portion of the gluteus medius onto the superoposterior, anterior, and lateral facets of the greater trochanter [5, 7, 8].

Gluteus medius tendon tears are typically seen in three scenarios: (1) chronic, nontraumatic tear of the anterior fibers of the gluteus medius tendon; (2) abductor tendon tears found in patients with femoral neck fractures or osteoarthritis; and (3) avulsion after total hip arthroplasty performed through an anterolateral or transgluteal approach. The most common of these scenarios is chronic, nontraumatic tears.

Several repair techniques have been described including repairs using both transosseous sutures [2, 9, 10] and suture anchors [11]. Endoscopic repair techniques include gluteal debridement or repairs, bursectomy, and iliotibial band release [1, 12–15]. Gluteus maximus muscle transfers [16–19] and vastus lateralis muscle transfers [20, 21], dermal matrix allograft augmented repair techniques [22], and Achilles tendon allograft [23] have been described as well.

Few reports have been published on the outcomes of open repairs. The limited data from small patient series mainly presented as part of surgical technique papers suggest overall good outcomes, however often requiring extensive rehabilitation and long recovery times [12, 24].

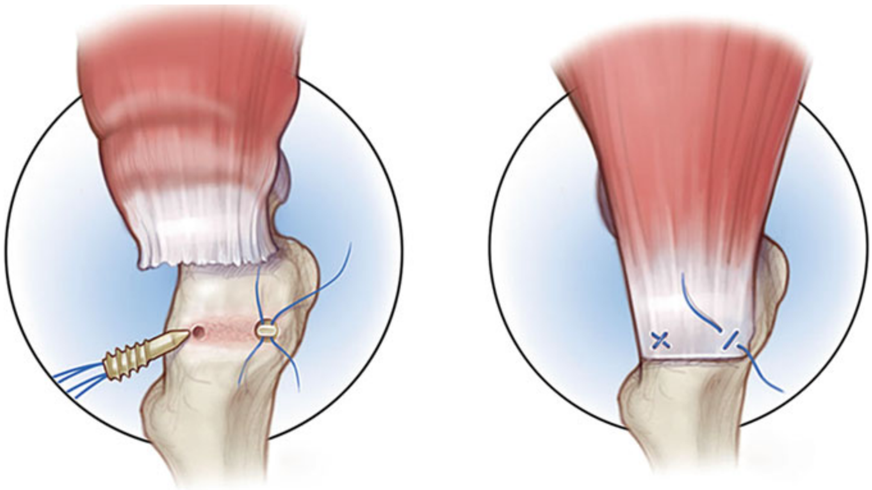


Fig. 3 Schematic depiction of gluteus medius repair with anchors (Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography © 2013–2014. All rights reserved)

Surgical Technique

The patient is placed supine on a standard operating room table. After induction of general endotracheal anesthesia, the patient is turned to a lateral decubitus position on a well-padded peg board. All bony prominences are well padded and a pneumatic compression device is placed on the well leg. After standard preparation of the operative extremity and sterile draping, a lateral skin incision centered over the greater trochanter is used to develop the interval between subcutaneous tissue and underlying iliotibial band. The iliotibial band is split longitudinally in line with its fibers, and appropriate retractors are placed, permitting visualization of the vastus ridge and the lateral facet of the greater trochanter. This allows entry into the peritrochanteric space to assess the gluteus medius tear (Fig. 4). The hip is internally rotated, exposing the trochanteric bursa at the posterior facet. The short external rotators are assessed for integrity. The sciatic nerve is typically not visualized or exposed.

The extremity is then externally rotated revealing the anterior trochanteric facet at the former insertion site of the gluteus minimus. Residual soft tissue remnants are thoroughly debrided off the footprint of the gluteus medius and minimus using a rongeur and round burr, promoting a favorable biologic healing response (Fig. 5). The retracted gluteus medius and gluteus minimus tendons are identified and assessed for visible degenerative changes in either tendon. Suture anchors are then placed into the peripheral zones of the footprint (see Fig. 3). The number of anchors placed is based on the size for the tear. Sutures can be passed in multiple manners. Options include simple, mattress, modified Mason-Allen, or a running locking fashion throughout the periphery of the crescent-shaped tear in the gluteus medius and minimus. Suture configuration is guided by surgeon preference and tear configuration. As in the shoulder, double-row fixation or transosseous equivalent type configurations can also be used. Sutures are tied with the leg held in slight abduction (10–15°) to limit tension at the repair site and reduce the tendon onto the prepared footprint (Fig. 6). On completion of the repair, the hip is gently brought through a broad physiologic range of motion and rotation. The footprint of the gluteus medius should be intact and stable throughout this range of motion.

After copious irrigation, the IT band layer is then closed using Vicryl absorbable sutures placed in a figure of eight fashion. A running subcuticular closure of the skin is finally completed [5, 25].

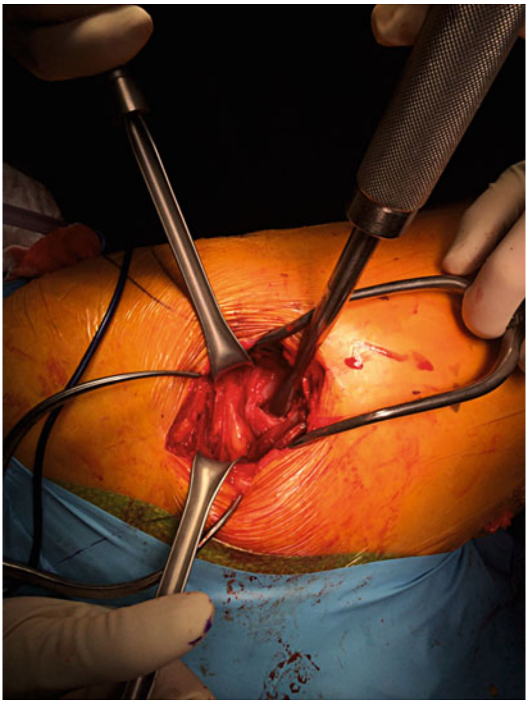


Fig. 4 Visualization of gluteus tear (Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography © 2013–2014. All rights reserved)

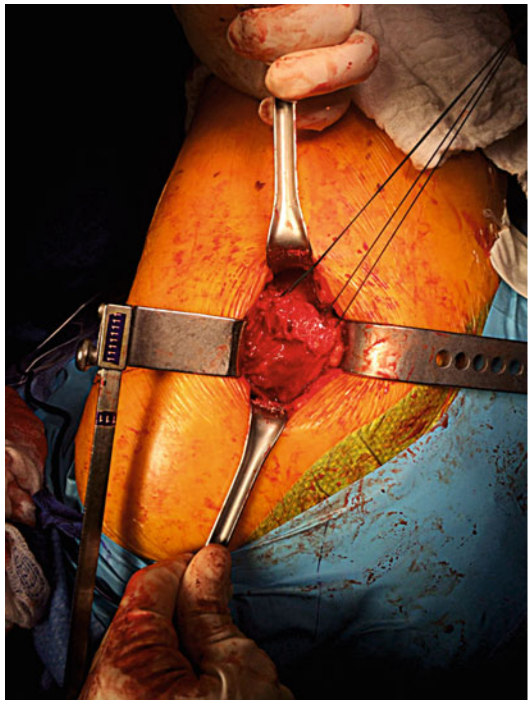


Fig. 5 Mobilization of gluteus medius (Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography © 2013–2014. All rights reserved)

Rehabilitation

Postoperatively, the patient is restricted to 20-lb flat-foot weight bearing for 6 weeks with no active abduction or passive adduction. A hip abduction brace is used during this time period, with a pillow between the legs while sleeping. The patient is advanced to weight bearing as tolerated by 8 weeks and full weight bearing without assistive devices by 12 weeks. Active hip abduction is allowed after the 8-week mark and progressive strengthening employed once full range of motion attained.

Outcomes

There are several small case reports in the literature on patients treated with open surgical repair of hip abductor tears [2, 11, 13, 26–29]. Most reports show good pain relief and some improvement in function after surgical repair. Clinical follow-up and long-term outcome assessment is limited, though. Davies et al. reported a retrospective review of a case series including 22 patients (23 hips). The mean Harris hip score improved from 53 points preoperatively to 87 points at 1 year and 88 points at 5 years. The mean lower-extremity activity scale score improved from 6.7 points preoperatively to 8.9 points at 1 year and 8.8 points at 5 years. There was no significant difference in the degree of clinical improvement in relation to the severity of the tear. However, three patients had poor results and were part of the group with the largest tears. Sixteen of nineteen patients were satisfied with their outcome and willing to undergo the procedure again. Davies et al. reported on 16 patients who underwent open surgical repair [26]. There were 4 re-ruptures, 3 of which were revised and 1 deep infection requiring debridement. The remaining

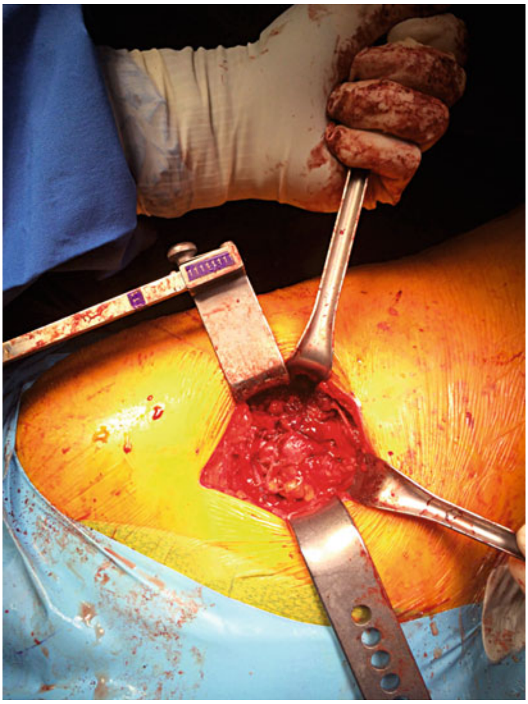


Fig. 6 Completed double-row repair (Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography © 2013–2014. All rights reserved)

11 patients had statistically significant improvements in hip symptoms. The mean change in visual analogue score was reported as 5 out of 10 ($p = 0.0024$). The mean change of Oxford hip score was 20.5 ($p = 0.00085$). The mean improvement in SF-36 PCS was 8.5 ($p = 0.0020$) and MCS 13.7 ($P = 0.134$). Six patients with preoperative Trendelenburg gait had normal gait 1 year following surgery. They concluded that surgical repair is overall successful for reduction of pain and improvement of function, but that there is a relatively high failure rate in chronic tears. Walsh et al. reported results of open surgical repair in 72 patients with a minimum follow-up of 1 year [28]. Improvement in both function and pain over time was seen in 95 % of their patients. Voos et al. reported good pain relief 2 years after arthroscopic repair in ten patients with low-grade tears [13]. Open repair was recommended for larger tears.

Summary

This chapter provides a brief overview of the anatomy, pathology, and epidemiology of gluteus medius tears and current repair techniques. The surgical technique of open repair is then described in detail (Figs. 4, 5, and 6).

References

1. Domb BG, Nasser RM, Botser IB. Partial-thickness tears of the gluteus medius: rationale and technique for trans-tendinous endoscopic repair. Arthroscopy. 2010;26(12):1697–705.
2. Bunker TD, Esler CN, Leach WJ. Rotator-cuff tear of the hip. J Bone Joint Surg Br. 1997;79(4):618–20.
3. Lachiewicz PF. Abductor tendon tears of the hip: evaluation and management. J Am Acad Orthop Surg. 2011;19(7):385–91.
4. Howell GE, Biggs RE, Bourne RB. Prevalence of abductor mechanism tears of the hips in patients with osteoarthritis. J Arthroplasty. 2001;16(1):121–3.
5. Robertson WJ, Gardner MJ, Barker JU, Boraiah S, Lorich DG, Kelly BT. Anatomy and dimensions of the gluteus medius tendon insertion. Arthroscopy. 2008;24(2):130–6.
6. Connell DA, Bass C, Sykes CAJ, Young D, Edwards E. Sonographic evaluation of gluteus medius and minimus tendinopathy. Eur Radiol. 2003;13(6):1339–47.
7. Flack NAMS, Nicholson HD, Woodley SJ. A review of the anatomy of the hip abductor muscles, gluteus medius, gluteus minimus, and tensor fascia lata. Clin Anat. 2012;25(6):697–708.
8. Flack NAMS, Nicholson HD, Woodley SJ. The anatomy of the hip abductor muscles. Clin Anat. 2014;27(2):241–253.
9. Miozzari HH, Dora C, Clark JM, Nötzli HP. Late repair of abductor avulsion after the transgluteal approach for hip arthroplasty. J Arthroplasty. 2010;25(3):450–1.
10. Lübbeke A, Kampfen S, Stern R, Hoffmeyer P. Results of surgical repair of abductor avulsion after primary total hip arthroplasty. J Arthroplasty. 2008;23(5):694–8.
11. Davies H, Zhaeentan S, Tavakkolizadeh A, Janes G. Surgical repair of chronic tears of the hip abductor mechanism. Hip Int. 2009;19(4):372–6.
12. Voos JE, Shindle MK, Pruett A, Asnis PD, Kelly BT. Endoscopic repair of gluteus medius tendon tears of the hip. Am J Sports Med. 2009;37(4):743–7.
13. Voos JE, Rudzki JR, Shindle MK, Martin H, Kelly BT. Arthroscopic anatomy and surgical techniques for peritrochanteric space disorders in the hip. Arthroscopy. 2007;23(11):1246.e1–5.

14. Domb BG, Carreira DS. Endoscopic repair of full-thickness gluteus medius tears. Arthrosc Tech Arthrosc Assoc North Am. 2013;2(2):e77–81.
15. Yanke AB, Hart MA, McCormick F, Nho SJ. Endoscopic repair of a gluteus medius tear at the musculotendinous junction. Arthrosc Tech Arthrosc Assoc North Am. 2013;2(2):e69–72.
16. Whiteside LA, Nayfeh T, Katerberg BJ. Gluteus maximus flap transfer for greater trochanter reconstruction in revision THA. Clin Orthop Relat Res. 2006;453:203–10.
17. Whiteside LA. Treating abductor deficiency: a transference technique. Orthopedics. 2011;34(9):e470–2.
18. Whiteside LA. Surgical technique: transfer of the anterior portion of the gluteus maximus muscle for abductor deficiency of the hip. Clin Orthop Relat Res. 2012;470(2):503–10.
19. Whiteside LA. Surgical technique: gluteus maximus and tensor fascia lata transfer for primary deficiency of the abductors of the hip. Clin Orthop Relat Res. 2014;472(2):645–53.
20. Beck M, Leunig M, Ellis T, Ganz R. Advancement of the vastus lateralis muscle for the treatment of hip abductor discontinuity. J Arthroplasty. 2004;19(4):476–80.
21. Kohl S, Evangelopoulos DS, Siebenrock KA, Beck M. Hip abductor defect repair by means of a vastus lateralis muscle shift. J Arthroplasty. 2012;27(4):625–9.
22. Rao BM, Kamal TT, Vafaye J, Taylor L. Surgical repair of hip abductors. A new technique using Graft Jacket allograft acellular human dermal matrix. Int Orthop. 2012;36(10):2049–53.
23. Fehm MN, Huddleston JI, Burke DW, Geller JA, Malchau H. Repair of a deficient abductor mechanism with Achilles tendon allograft after total hip replacement. J Bone Joint Surg Am. 2010;92(13):2305–11.
24. El-Husseiny M, Patel S, Rayan F, Haddad F. Gluteus medius tears: an under-diagnosed pathology. Br J Hosp Med (Lond). 2011;72(1):12–6.
25. Stanton MC, Maloney MD, DeHaven KE, Giordano BD. Acute traumatic tear of gluteus medius and minimus tendons in a patient without antecedant peritrochanteric hip pain. Geriatr Orthop Surg Rehab. 2012;3(2):84–8.
26. Davies JF, Stiehl JB, Davies JA, Geiger PB. Surgical treatment of hip abductor tendon tears. J Bone Joint Surg Am. 2013;95(15):1420–5.
27. Fisher DA, Almand JD, Watts MR. Operative repair of bilateral spontaneous gluteus medius and minimus tendon ruptures. A case report. J Bone Joint Surg Am. 2007;89(5):1103–7.
28. Walsh MJ, Walton JR, Walsh NA. Surgical repair of the gluteal tendons: a report of 72 cases. J Arthroplasty. 2011;26(8):1514–9.
29. Kagan A. Rotator cuff tears of the hip. Clin Orthop Relat Res. 1999;368:135–40.

Surgical Technique: Gluteus Maximus Transfer

70

Jonathan M. Frank, Andrew E. Federer, Simon Lee, and Shane J. Nho

Contents

Abstract

Abductor deficiency secondary to total hip arthroplasty or primary abductor disruption is a well-known cause of a Trendelenburg gait with associated pain and instability. The history, clinical exam, and supplementary imaging studies aid in establishing this diagnosis. Gluteus maximus transfer is a proven and effective treatment strategy for severe abductor deficiency.

Introduction

Hip abductor deficiency can result from primary abductor disruption or secondary to total hip arthroplasty (THA). Primary tears of the gluteus medius and minimus are often secondary to greater trochanteric pain syndrome (GTPS) and associated disorders such as chronic degeneration and tendinopathy of the gluteal tendons, trochanteric bursitis, and iliotibial band disorders [1–6]. Abductor deficiency secondary to THA is more common and results from rupture of the abductor tendon insertion, proximal femoral bone stock loss in association with failed THA due to osteolysis, inflammatory changes to abductor mechanism from hypersensitivity to metal-on-metal wear, as well as iatrogenic damage to the superior gluteal nerve (SGN) causing gluteal nerve palsy [1, 7–17]. Disruption or tears of the hip abductors post-THA are a well-recognized cause of hip pain and limp, with reported

J.M. Frank (✉)
Department of Orthopaedic Surgery, Rush University Medical Center, Chicago, IL, USA
e-mail: jon.m.frank@gmail.com

A.E. Federer
Midwest Orthopaedics at Rush, Hip Preservation Center, Rush University Medical Center, Chicago, IL, USA
e-mail: afederer34@gmail.com

S. Lee
Department of Orthopaedic Surgery, Midwest Orthopaedics at Rush, Rush University Medical Center, Chicago, IL, USA
e-mail: simon.x.lee@rushortho.com

S.J. Nho
Department of Orthopaedic Surgery, Rush University Medical Center, Chicago, IL, USA

Midwest Orthopaedics at Rush, Hip Preservation Center, Rush University Medical Center, Chicago, IL, USA
e-mail: shane.nho@rushortho.com

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_74

incidence between 0.08 % and 22 % [7–9, 18, 19]. The risk is increased in revision THA and is often associated with degradation of bone stock quality [20]. The decreased hip stability that follows is secondary to loss of ligamentous and capsular integrity as well as loss of muscle control [13, 21, 22]. The changes in abductor function nearly always result in a Trendelenburg gait or pelvic sag and predispose the hip to dislocations after THA revision.

Clinical Exam and Imaging

Physical exam includes gait assessment, limb length discrepancy, range of motion, and abductor strength testing. Patients with abductor insufficiency demonstrate Trendelenburg gait. Some patients may have already begun to use assistive devices for ambulation. The Trendelenburg test is a useful exam finding for abductor weakness, especially when coupled with history of trauma, subluxation, or dislocation [23, 24]. Motor strength testing in a lateral decubitus position with the knee extended will accentuate abductor insufficiency as most patients will not be able to maintain hip abduction against gravity. Post-THA abductor weakness warrants gathering a history regarding surgical approach, intraoperative events, and postoperative recovery [1].

Plain radiographs are initially used for the assessment and diagnosis of abductor deficiency and are able to detect implant orientation and position, as well as signs of loosening which is indicative of abductor weakness [1]. Plain radiographs can also rule out obvious femoral neck, intertrochanteric, and proximal shaft fractures. Magnetic resonance imaging (MRI) is considered to be the most useful imaging modality for detecting abductor tendon tears. The diagnosis, however, still requires supporting clinical findings [1, 25, 26]. Cvitanic et al. noted MRI T2-weighted hyperintensity just superior to the greater trochanter as having an accuracy of 91 %, with sensitivity and specificity of 75 % and 93 %, respectively [27]. Furthermore, T1-weighted sequences are useful for evaluating the quality of the gluteal muscles using the Goutallier or

Table 1 Goutallier classification of fatty muscle degeneration [28]

Grade	Amount of fat within muscle
0	Normal muscle
1	Some fatty streaks
2	More muscle than fat
3	Equal amounts of muscle and fat
4	More fat than muscle

Table 2 Quartile classification system for fatty degeneration of gluteal muscles [29]

Grade	Percent fat in muscle
0	Normal muscle
1	<25 %
2	25–50 %
3	50–75 %
4	75–100 %

Quartile classification systems to quantify the amount of fatty infiltration (Tables 1 and 2) [28, 29]. Much like rotator cuff tears, grade 2 or worse muscle degeneration is a potential contraindication to primary abductor repair [30, 31]. If the patient has had a prior THA, then metal suppression sequences are required to visualize the gluteus medius and minimus tendons. Ultrasonography is also a useful adjunct though its functionality is highly operator dependent [32]. There is a paucity of data on its sensitivity and specificity in diagnosing abductor deficiency.

In patients with abductor deficiency in the absence of radiographic or imaging findings, SGN palsy can be diagnosed and followed with electromyography (EMG). Close observation is encouraged as 95 % of patients are expected to spontaneously recover by 24 months post-THA. Serial EMG is a useful tool for the assessment of patient recovery during this time [33].

Surgical Management

Depending on the type and degree of abductor deficiency, different abductor repair or transfer techniques are warranted. The main indication for anterior gluteus maximus transfer is abductor deficiency secondary to loss of gluteus medius and minimus in the presence of a normal functioning gluteus maximus [13]. The current author's indication for gluteus maximus transfer is massive gluteus medius and minimus tears with greater than 50 % fatty infiltration or failed primary repair of gluteus medius and minimus tendons with a normal gluteus maximus muscle. Fortunately, gluteus maximus function is spared in most cases of abductor deficiency. Though large studies have yet to be conducted, anterior gluteus maximus transfer is believed to be particularly useful in patients with severe destruction of the abductor mechanism. This is often seen in patients with abductor avulsion, as well as osteolytic and inflammatory damage secondary to THA and THA revisions [9, 13]. Surgical reattachment of avulsed abductors is often difficult, and inflammatory destruction secondary to THA metal-on-metal hypersensitivity often results in complete loss of abductors and capsular instability. In these cases, anterior gluteus maximus transfer can restore stability and abductor function.

The anterior gluteus maximus originates at the anterior half of the iliac crest, and in hip extension, its fibers are positioned parallel to the femoral shaft. Since its SGN innervation enters the muscle proximally and posteriorly, the anterior detachment from the fascia lata during flap creation limits iatrogenic nerve damage [13, 34]. In the technique described by Whiteside [13], the anterior half of the gluteus maximus is transferred to the proximal femur and attached underneath the vastus lateralis. In cases of more severe deficiency of gluteus minimus and posterior capsule, a second triangular flap from the posterior gluteus maximus can be transferred under both the greater trochanter and the primary flap to the anterior capsule. This partially compensates for absence of short external rotators and posterior instability and is the subject of the surgical technique section.

In cases where the greater trochanter is displaced from the remaining bone stock but still has abductor attachments, a posterior gluteus maximus transfer technique can be used. Whiteside et al. [13] note that in most cases of tendon or greater trochanter avulsion associated with THA, surgeons cannot reattach the gluteus medius and minimus to the femur. Moreover, the abductor muscles and hip capsule may be completely absent in cases of destructive inflammation associated with failed THA [13]. In this technique, a triangular muscle flap from the middle posterior gluteus maximus is created, without disrupting the tendon insertion, and used to span the gap between the displaced greater trochanter and the remaining bone stock [20]. When indicated, this procedure can increase abduction and pelvic stability but often coincides with decreased range of motion. However, this posterior transfer technique cannot be used in cases of severe abductor deficiency or absence secondary to osteolytic degradation or prolonged displacement of the greater trochanter [13, 20]. Without gluteus medius or minimus attachment to the greater trochanter or proximal femur, complete abductor substitution is necessary [13]. In these cases, anterior gluteus maximus transfer would be more useful.

Other surgical options for less severe abductor deficiency include direct transosseous repair with or without incorporation of fascia lata and Achilles tendon allograft. These procedures have been met with mixed results [1, 7–10]. The authors' preferred technique includes a gluteus maximus transfer using a docking technique with double-row fixation with suture bridge.

Surgical Technique

The patient is placed in the lateral decubitus position. The greater trochanter is outlined with an incision that begins 3 cm proximal to the tip of the greater trochanter and extends 3 cm distally (Fig. 1). Sharp dissection is performed down to the fascial layer of the iliotibial band, which is then incised just distal to the iliac crest to the level of the greater trochanter. A flap is created in the posterior aspect of the gluteus maximus and mobilized (Fig. 2). A second flap is made just distal to the first to recreate the short external rotators. After adequately mobilizing these flaps (Fig. 3), two sets of Krackow stitches (No. 2 FiberWire, Arthrex, Naples, FL) are placed in each flap. If the

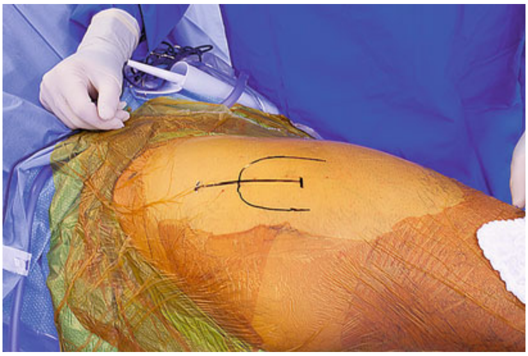


Fig. 1 The greater trochanter is outlined with a 6–8-cm incision centered at the tip of the greater trochanter

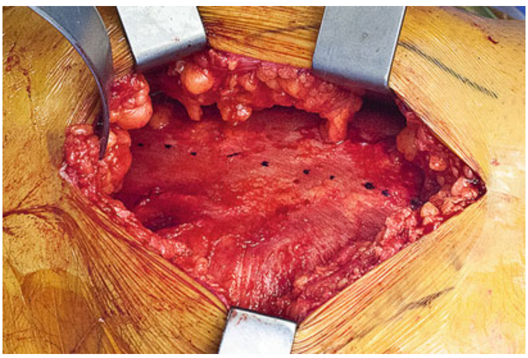


Fig. 2 The gluteus maximus is visualized. The incisions are marked for the posterior flap (*dotted line*) as well as the flap for the short external rotators (*solid line*)

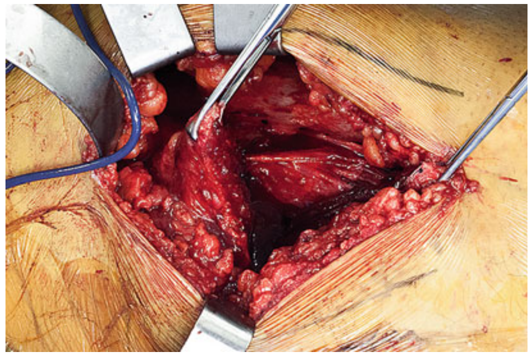


Fig. 3 The flaps are fashioned and mobilized. Once this is completed, two sets running Krackow sutures are placed in each flap

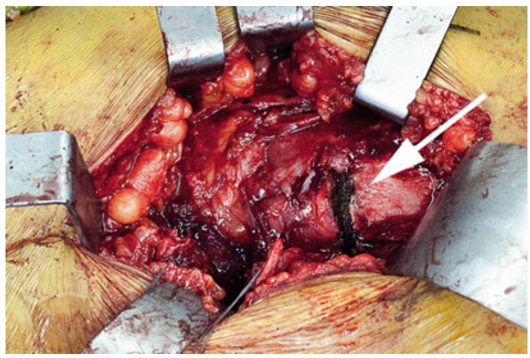


Fig. 4 A bone trough (*arrow*) is developed in the greater trochanter so that the gluteus maximus flap can be docked into the bone trough and tied over a bone bridge

gluteus medius tendon appears to be torn and retracted, a primary repair of the tendon can also be performed in addition to the gluteus maximus transfer. The greater trochanter is decorticated with a 4.0 mm oval burr (Stryker, Kalamazoo, MI) that is then used to create a trough within the greater trochanter (Fig. 4). The burr is placed on the tip of the greater trochanter over the superoposterior and lateral facets and directed approximately 45° away from the lateral cortex. Two drill holes are created to communicate with the posterior aspect of the trough and allow the sutures from the gluteus maximus flap to dock into the bone trough. Two additional drill holes are placed on the anterior aspect of the trough so that there are a total of four drill holes. Passing sutures are placed through the drill holes, and a suture loop will exit through the trough entry. Once this is completed, the hip is placed in an abducted position. Two suture anchors (4.5 Bio-Corkscrew, Arthrex, Naples, FL) are placed on the medial border of the gluteus medius footprint to primarily repair the gluteus medius and, in some cases, the gluteus minimus tendons. The Krackow sutures are passed into the trough via the passing sutures, and the initial gluteus maximus flap is docked into the bone trough and subsequently tied over a bone bridge (Fig. 5). Stitches from the suture anchors are passed through the flap, and these sutures with those tied over the bone bridge are subsequently incorporated into a lateral row suture anchor (4.5 SwiveLock, Arthrex, Naples, FL).

Next, the short external rotator flap is passed just deep to the initial flap. A knotless suture anchor is tapped into the anterior aspect of the

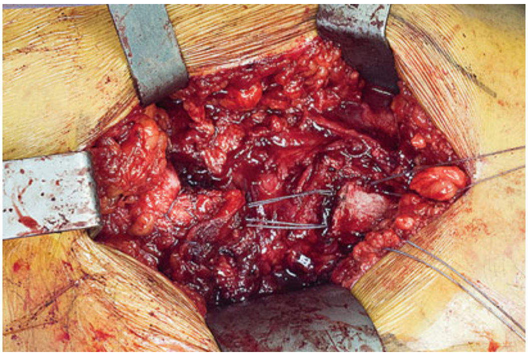


Fig. 5 Sutures from the gluteus maximus flap are passed through the bone trough for docking

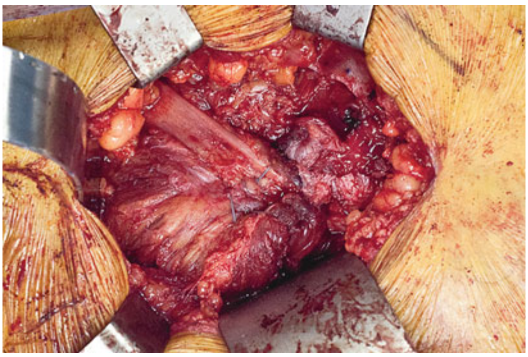


Fig. 6 View of the complete gluteus maximus transfer

greater trochanter. The free Krackow stitches from the short external rotator flap are fixated into a knotless anchor (4.5 SwiveLock, Arthrex, Naples, FL) to the anterior aspect of the greater trochanter (Fig. 6). The proximal ends of the vastus lateralis are closed over the gluteus maximus transfer. Once this is completed, the wound is copiously irrigated, and platelet-rich plasma is injected around the gluteus maximus transfer. Next, a deep drain is placed followed by closure of the fascia, deep dermal layer, and skin. A sterile dressing is applied, and an abduction pillow is placed.

In terms of the rehabilitation protocol, the patient is placed in a hip abduction brace with protected weight bearing with a walker for 2 months. The authors prefer to send patients home with a continuous passive motion machine for the first 2–3 weeks. The patient will begin supervised physical therapy at 6 weeks after surgery with instructions to continue gait training with a walker and no active hip abduction. The patient will progress from a walker to a cane at approximately 3 months with cane assistance for 5–6 months after surgery.

Summary

Abductor deficiency is a well-recognized condition secondary to primary abductor insufficiency or seen after THA and can result in severe gait dysfunction and instability. The use of a gluteus maximus muscle flap is effective for restoring hip abduction in the majority of patients, and inclusion of the short external rotator flap has the added benefit of increased stability, in particular for cases of severe capsular deficiency. With the steady projected increase in an aging population as well as increased number of primary and revision THA procedures in the near future [35], gluteus maximus transfer for severe abductor insufficiency is likely to increase.

References

1. Odak S, Ivory J. Management of abductor mechanism deficiency following total hip replacement. Bone Joint J. 2013;95-B(3):343–7.
2. Bunker TD, Esler CN, Leach WJ. Rotator-cuff tear of the hip. J Bone Joint Surg Br. 1997;79(4):618–20.
3. Howell GE, Biggs RE, Bourne RB. Prevalence of abductor mechanism tears of the hips in patients with osteoarthritis. J Arthroplasty. 2001;16(1):121–3.
4. Segal NA, Felson DT, Torner JC, Zhu Y, Curtis JR, Niu J, et al. Greater trochanteric pain syndrome: epidemiology and associated factors. Arch Phys Med Rehabil. 2007;88(8):988–92.
5. Lievense A, Bierma-Zeinstra S, Schouten B, Bohnen A, Verhaar J, Koes B. Prognosis of trochanteric pain in primary care. Br J Gen Pract. 2005; 55(512):199–204.
6. Williams BS, Cohen SP. Greater trochanteric pain syndrome: a review of anatomy, diagnosis and treatment. Anesth Analg. 2009;108(5):1662–70.
7. Weber M, Berry DJ. Abductor avulsion after primary total hip arthroplasty. Results of repair. J Arthroplasty. 1997;12(2):202–6.
8. Miozzari HH, Dora C, Clark JM, Notzli HP. Late repair of abductor avulsion after the transgluteal approach for hip arthroplasty. J Arthroplasty. 2010; 25(3):450–457.e1.

9. Fehm MN, Huddleston JI, Burke DW, Geller JA, Malchau H. Repair of a deficient abductor mechanism with Achilles tendon allograft after total hip replacement. J Bone Joint Surg Am. 2010;92(13):2305–11.
10. Lubbeke A, Kampfen S, Stern R, Hoffmeyer P. Results of surgical repair of abductor avulsion after primary total hip arthroplasty. J Arthroplasty. 2008;23(5): 694–8.
11. Baker AS, Bitounis VC. Abductor function after total hip replacement. An electromyographic and clinical review. J Bone Joint Surg Br. 1989;71(1):47–50.
12. Khan T, Knowles D. Damage to the superior gluteal nerve during the direct lateral approach to the hip: a cadaveric study. J Arthroplasty. 2007;22(8):1198–200.
13. Whiteside LA. Surgical technique: transfer of the anterior portion of the gluteus maximus muscle for abductor deficiency of the hip. Clin Orthop Relat Res. 2012;470(2):503–10.
14. Carr AM, DeSteiger R. Osteolysis in patients with a metal-on-metal hip arthroplasty. ANZ J Surg. 2008; 78(3):144–7.
15. Evans EM, Freeman MA, Miller AJ, Vernon-Roberts B. Metal sensitivity as a cause of bone necrosis and loosening of the prosthesis in total joint replacement. J Bone Joint Surg Br. 1974;56-B(4):626–42.
16. Killampalli VV, Reading AD. Late instability of bilateral metal on metal hip resurfacings due to progressive local tissue effects. Hip Int. 2009;19(3):287–91.
17. Park YS, Moon YW, Lim SJ, Yang JM, Ahn G, Choi YL. Early osteolysis following second-generation metal-on-metal hip replacement. J Bone Joint Surg Am. 2005;87(7):1515–21.
18. Masonis JL, Bourne RB. Surgical approach, abductor function, and total hip arthroplasty dislocation. Clin Orthop Relat Res. 2002;405:46–53.
19. Iorio R, Healy WL, Warren PD, Appleby D. Lateral trochanteric pain following primary total hip arthroplasty. J Arthroplasty. 2006;21(2):233–6.
20. Whiteside LA, Nayfeh T, Katerberg BJ. Gluteus maximus flap transfer for greater trochanter reconstruction in revision THA. Clin Orthop Relat Res. 2006; 453:203–10.
21. Coventry MB. Late dislocations in patients with Charnley total hip arthroplasty. J Bone Joint Surg Am. 1985;67(6):832–41.
22. Whiteside LA. Major femoral bone loss in revision total hip arthroplasty treated with tapered, porous-coated stems. Clin Orthop Relat Res. 2004;429:222–6.
23. Pai VS. Significance of the Trendelenburg test in total hip arthroplasty. Influence of lateral approaches. J Arthroplasty. 1996;11(2):174–9.
24. Hardcastle P, Nade S. The significance of the Trendelenburg test. J Bone Joint Surg Br. 1985;67(5): 741–6.
25. Pfirrmann CW, Notzli HP, Dora C, Hodler J, Zanetti M. Abductor tendons and muscles assessed at MR imaging after total hip arthroplasty in asymptomatic and symptomatic patients. Radiology. 2005;235(3): 969–76.
26. Muller M, Tohtz S, Winkler T, Dewey M, Springer I, Perka C. MRI findings of gluteus minimus muscle damage in primary total hip arthroplasty and the influence on clinical outcome. Arch Orthop Trauma Surg. 2010;130(7):927–35.
27. Cvitanic O, Henzie G, Skezas N, Lyons J, Minter J. MRI diagnosis of tears of the hip abductor tendons (gluteus medius and gluteus minimus). AJR Am J Roentgenol. 2004;182(1):137–43.
28. Goutallier D, Postel JM, Bernageau J, Lavau L, Voisin MC. Fatty muscle degeneration in cuff ruptures. Pre- and postoperative evaluation by CT scan. Clin Orthop Relat Res. 1994;304:78–83.
29. Engelken F, Wassilew GI, Kohlitz T, Brockhaus S, Hamm B, Perka C, et al. Assessment of fatty degeneration of the gluteal muscles in patients with THA using MRI: reliability and accuracy of the goutallier and quartile classification systems. J Arthroplasty. 2013;(1):149–53.
30. Mellado JM, Calmet J, Olona M, Esteve C, Camins A, Perez Del Palomar L, et al. Surgically repaired massive rotator cuff tears: MRI of tendon integrity, muscle fatty degeneration, and muscle atrophy correlated with intraoperative and clinical findings. AJR Am J Roentgenol. 2005;184(5):1456–63.
31. Goutallier D, Postel JM, Gleyze P, Leguilloux P, Van Driessche S. Influence of cuff muscle fatty degeneration on anatomic and functional outcomes after simple suture of full-thickness tears. J Should Elb Surg. 2003; 12(6):550–4.
32. Garcia FL, Picado CH, Nogueira-Barbosa MH. Sonographic evaluation of the abductor mechanism after total hip arthroplasty. J Ultrasound Med. 2010;29(3):465–71.
33. Picado CH, Garcia FL, Marques Jr W. Damage to the superior gluteal nerve after direct lateral approach to the hip. Clin Orthop Relat Res. 2007;455:209–11.
34. Gray H. Gray's anatomy. Elsevier, USA. 1901/1974.
35. Bang H, Chiu YL, Memtsoudis SG, Mandl LA, Della Valle AG, Mushlin AI, et al. Total hip and total knee arthroplasties: trends and disparities revisited. Am J Orthop (Belle Mead NJ). 2010;39(9): E95–102.

Deep Gluteal Space

71

Hal D. Martin, Ian J. Palmer, and Munif A. Hatem

Contents

Abstract

Deep gluteal syndrome is characterized by nondiscogenic, extrapelvic sciatic nerve compression presenting with symptoms of pain and dysesthesias in the buttock area, hip, or posterior thigh and/or as radicular pain. The piriformis muscle and tendon are the most common source of extrapelvic sciatic nerve entrapment. However, a number of structures can entrap the sciatic nerve in the deep gluteal space, including bone structures, fibrous scar bands, and muscular structures other than the piriformis. The main differential diagnoses are intra-articular hip pathologies, spine issues, intrapelvic abnormalities, ischiofemoral impingement, hamstring's origin tendinopathy, and pudendal nerve entrapment. A comprehensive history and physical examination is crucial for the diagnosis of deep gluteal syndrome. Guided injections and magnetic resonance imaging are useful complementary diagnostic tools. The nonoperative treatment of deep gluteal syndrome is successful in most patients. Endoscopic sciatic nerve decompression may be indicated in cases of failure of the conservative treatment.

Introduction

Posterior hip pain often represents a diagnostic challenge and the examiner must be aware of the deep gluteal space abnormalities in order to obtain

H.D. Martin (✉) • I.J. Palmer • M.A. Hatem
Baylor University Medical Center, Hip Preservation Center, Dallas, TX, USA
e-mail: Hal.Martin@baylorhealth.edu; ianjamespalmer@gmail.com; munifhatem@gmail.com

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_75

a correct diagnosis and treatment plan. The sources of symptoms can include conditions in one or more of the following hip layers: osseous, capsulolabral, musculotendinous, and neurovascular.

Deep gluteal syndrome is characterized by nondiscogenic, extrapelvic sciatic nerve compression presenting with symptoms of pain and dysesthesias in the buttock area, hip, or posterior thigh and/or as radicular pain [1]. The nomenclature piriformis syndrome was widely utilized in the early years to characterize patients with deep gluteal pain, since the piriformis muscle was considered the only structure to compress the sciatic nerve in the deep gluteal space. However, the progress in diagnostic and surgical techniques has demonstrated a number of structures entrapping the sciatic nerve: fibrous bands containing blood vessels [2, 3], gluteal muscles [1], hamstring muscles [4, 5], the gemelli-obturator internus complex [6, 7], bone structures [8], vascular abnormalities [9, 10], and space-occupying lesions [11, 12]. Considering the variation of anatomical entrapment, the term "deep gluteal syndrome" [1] is preferred to describe the entrapment of the sciatic nerve in the deep gluteal space. The sciatic nerve can be also affected in locations above and below the deep gluteal space, as in intrapelvic vascular and urogynecologic abnormalities [13]. Furthermore, entrapments can occur in more than one place in the same nerve fiber or coexist with lumbosacral root compression. Considering the sciatic nerve can be entrapped by structures in each layer of the hip, a comprehensive physical examination with a thorough understanding of anatomy and biomechanics is critical in cases of deep gluteal pain.

Deep Gluteal Space Anatomy

A complete review of anatomy is outside the scope of this chapter; however, a short review of the deep gluteal space and sciatic nerve anatomy will be given. The deep gluteal space is anterior to the gluteus maximus muscle and posterior to the acetabular column, hip joint capsule, and proximal femur. Other anatomical limits include the linea aspera (lateral), the sacrotuberous ligament and falciform fascia (medial), the inferior margin of the greater sciatic notch (superior), and the distal border of the ischial tuberosity (inferior) (Fig. 1). The sacrotuberous and sacrospinous ligaments create the greater and lesser sciatic foramen, which communicate the deep gluteal space with the true pelvis and ischioanal fossa. The sacrotuberous ligament is normally composed of two parts: a ligamentous band and a membranous falciform process [14] (Fig. 2). Both sacrospinous and sacrotuberous ligaments are anatomically close to the pudendal nerve and may be involved in the entrapment of this nerve.

The piriformis muscle occupies a central position in the buttock and is an important reference for identifying the neurovascular structures emerging above and below it (Fig. 3). This muscle arises from the ventrolateral surface of the sacrum, gluteal surface of the ileum, and sacroiliac joint capsule. The distal attachment of the piriformis is at the medial side of the upper border of the greater trochanter, often partially blended with the common tendon of obturator/gemelli complex [15–17]. Distal to the piriformis muscle is the cluster of short external rotators: the gemellus superior, obturator internus, gemellus inferior, and quadratus femoris muscle. At the ischial tuberosity, the long head of biceps femoris and semitendinosus have a common tendinous origin. The semimembranosus muscle also originates from the ischium, laterally and anteriorly to the long head of the biceps/semitendinosus muscles common origin [18] (Fig. 4).

Seven neural structures exit the pelvis through the greater sciatic notch: posterior femoral cutaneous nerve, superior gluteal nerve, inferior gluteal nerve, nerve to obturator internus, nerve to quadratus femoris muscle, pudendal nerve, and sciatic nerve (Fig. 3). Table 1 is a summary of the usual motor and sensory functions for each nerve. Accompanying the respective nerves are the superior gluteal vessels, inferior gluteal vessels, and internal pudendal vessels.

The anatomic positions of the inferior gluteal artery (IGA) and medial circumflex femoral artery (MCFA) are relevant within the deep gluteal space. The IGA enters the deep gluteal space

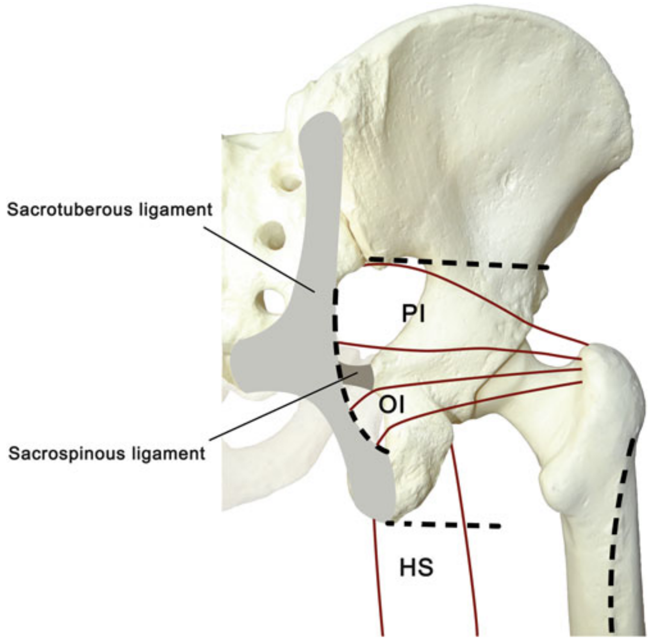


Fig. 1 Limits (*dashed lines*) of the deep gluteal space beneath the gluteus maximus muscle: lateral, linea aspera; medial, sacrotuberous ligament and falciform fascia; superior, inferior margin of the greater sciatic notch; inferior, the distal border of the ischial tuberosity. *PI* piriformis muscle, *OI* obturator internus muscle, *HS* hamstring muscles

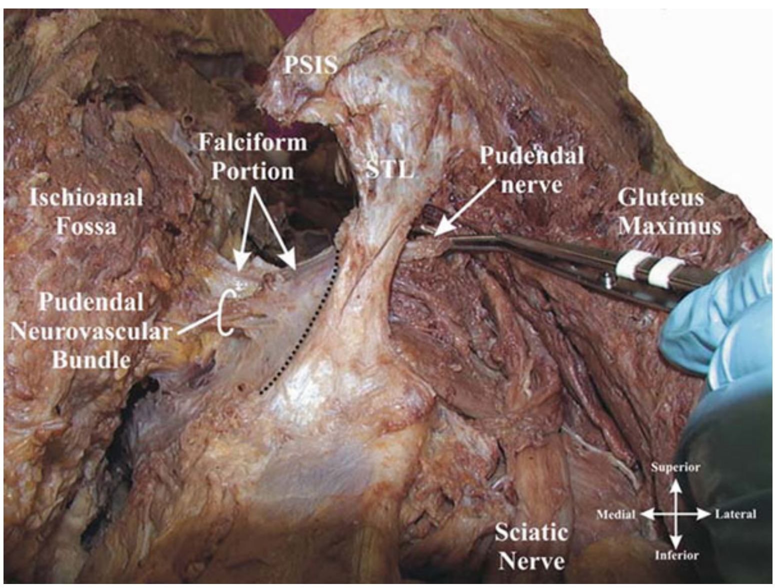


Fig. 2 Sacrotuberous ligament and the falciform process (Reprinted with permission from Loukas et al. [14])

with the inferior gluteal nerve and supplies the gluteus maximus muscle. This artery also gives a superficial arterial branch that crosses the sciatic nerve laterally between the piriformis and superior gemellus muscles. Another branch of the IGA is the descending branch, which runs along the posterior femoral cutaneous nerve in a frequency of 72 % according to a cadaveric study [21]. The MCFA follows the inferior border of the obturator externus and crosses over its tendon and under the external rotators and piriformis muscle (Fig. 5) [22]. The existence of an anastomosis between

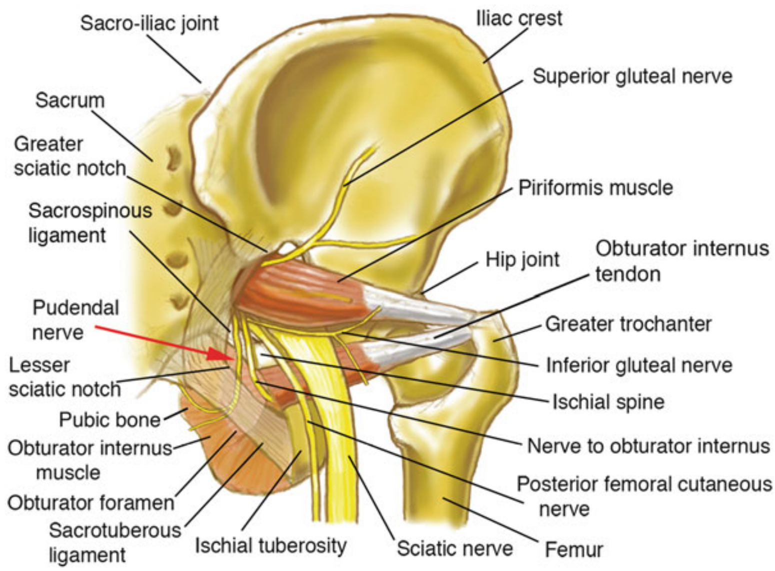


Fig. 3 Schematic illustrating the nerve anatomy of the deep gluteal space (Reprinted from Filler [19])

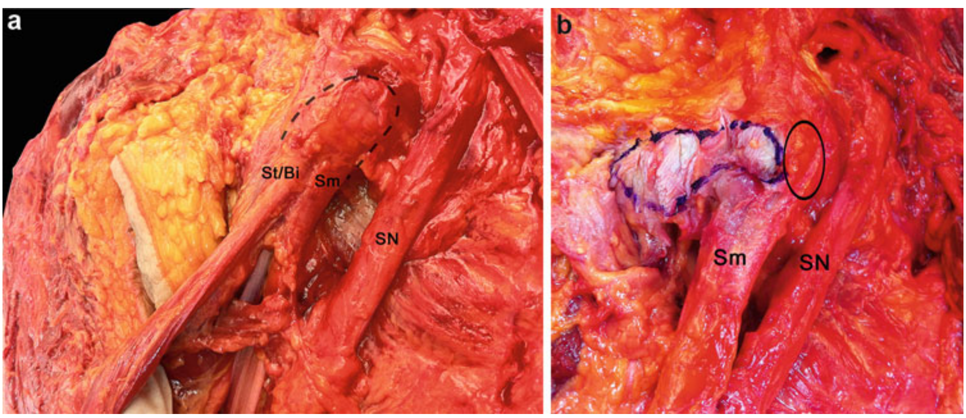


Fig. 4 Origin of the hamstrings muscles at ischial tuberosity. (**a**) Posterolateral view of the hamstrings origin at the ischial tuberosity (*dashed line*) in a cadaver specimen. The semimembranosus muscle origin (*Sm*) is anterior and lateral to the conjoint origin of the semitendinosus and long head of the biceps femoris muscles (St/Bi). *SN* sciatic nerve. (**b**) Posterior view after detachment of the semitendinosus and long head of the biceps femoris muscles from the ischial tuberosity (*blue demarked area*). The semimembranosus origin (*Sm*) was preserved. Solid line marking the origin of the sm located lateral to the St/Bi origin at the ischial tuberosity

the inferior gluteal artery and the medial femoral circumflex artery is frequent [23].

Sciatic Nerve Anatomy and Biomechanics

The sciatic nerve is formed by the L4–S3 ventral rami in the sacral plexus. Nerve fibers of the fibular and tibial components maintain a pattern of fiber separation in these branches and in the sciatic nerve. The sciatic nerve physically splits in tibial and fibular divisions at highly variable locations from the pelvis to the popliteal fossa, although this split is more frequent at the distal thigh [24]. Often, the split is oblique and may not be seen in a uniplanar MRI view [25]. Most sciatic neural fibers are destined to motor and sensory innervation distal to the knee. However, important branches arise from the nerve in the deep gluteal

Table 1 Summary of function of the nerves in the deep gluteal space

Nerve	Motor innervation	Sensory innervation
Posterior femoral cutaneous nerve		Gluteal region, perineum, and posterior thigh and popliteal fossa
Superior gluteal nerve	Gluteus medius, gluteus minimus, and tensor fascia lata	
Inferior gluteal nerve	Gluteus maximus	
Nerve to obturator internus	Superior gemellus and obturator internus	
Nerve to quadratus femoris	Inferior gemellus and quadratus femoris	Hip capsule
Pudendal nerve	Perineal muscles, external urethral sphincter, and external anal sphincter	Perineum, external genitalia
Sciatic nerve	Semitendinosus, biceps femoris, semimembranosus, extensor portion of the adductor magnus, and leg and foot musculature	Leg and foot, except for the saphenous nerve dermatome

Source: Moore [20] and Standring (Gray's Anatomy) [17]

space and thigh. A summary of the sciatic nerve branches in the thigh is depicted in Fig. 6 according to Seidel et al. [26] and Sunderland and Hughes [25].

Neural tissue and nonneural tissue compose the sciatic nerve. The ratio neural/nonneural tissue changes from 2/1 at the level of piriformis muscle to 1/1 at the midfemur, i.e., there is an increase in the nonneural tissue contribution as the sciatic nerve courses distally [27] (Fig. 7). The composition of the sciatic nerve also varies during the aging process, with increase in connective tissue and decrease of myelinated nerve fibers [28].

The nerve fibers of the sciatic nerve do not course between the tibial and fibular divisions [17]. However, fibers are often changing from one fascicle to another within each division [25]. Sunderland reported 6 mm as the maximum length of nerve trunk with a constant fascicular pattern, although an individual fascicle can maintain the same neural fibers for greater distances [25]. In general, most fascicles contain fibers for the majority, if not all, of the peripheral branches. Nevertheless, there is a tendency of grouping fibers for different muscles with similar function, for example, the fibers for the hamstring muscles are located anterior-medially in the proximal portion of the sciatic nerve. A progressive arrangement is found until the appearance of fascicles with nervous fibers exclusively destined to specific branches [25].

The sciatic nerve has a segmental arterial supply by branches of the IGA, MCFA, and perforating arteries of the thigh (usually the first and second) [29–31] (Fig. 8). The venous drainage of the sciatic nerve is performed through the perforators to the femoral profunda system in the thigh and to the popliteal vein at the knee [32] (Fig. 9). Nonfunctioning sciatic veins have been related to sciatic nerve symptoms [9].

The sacral plexus is anatomically close to the internal iliac vessels, their branches, and tributaries. The superior gluteal vessels run either between the lumbosacral trunk (L4–L5 ventral rami) and first sacral ventral ramus or between the first and second sacral rami, whereas the inferior gluteal vessels lie between either the first and second or second and third sacral rami (Fig. 10) [17, 33]. The ovaries are close to the sacral plexus, although on the left side the sigmoid is usually between the ovary and sacral plexus. The intimate anatomic relation between the iliac vessels, ovaries, and sacral plexus is an important consideration in sciatica caused by sacral plexus vascular compression and endometriosis [34].

The sciatic nerve is the terminal branch of the sacral plexus and courses anterior to the piriformis muscle in the pelvis. Variation in the relationship between the sciatic nerve and the piriformis muscle is present in 16–17 % of the subjects and can be a cause of sciatic nerve entrapment [35, 36]. After leaving the piriformis muscle, the sciatic nerve runs posteriorly to the obturator/gemelli

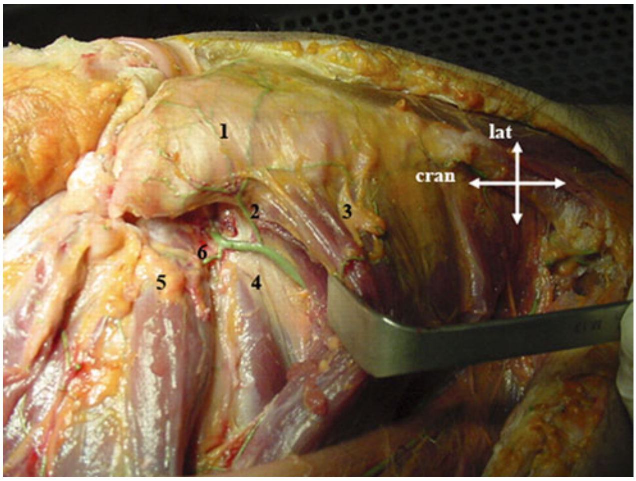


Fig. 5 Deep branch of the medial femoral circumflex artery. Posterior aspect of the right hip, demonstrating the anatomic position of the deep branch of the medial femoral circumflex artery. (*1*) greater trochanter, (*2*) trochanteric branch of the medial femoral circumflex artery, (*3*) quadratus femoris muscle, (*4*) obturator externus muscle, (*5*) obturator internus and gemellus muscles, and (*6*) anastomotic branch to the inferior gluteal artery. *Cran* cranial, *Lat* lateral (Reprint with permission from Kahlor et al. [22])

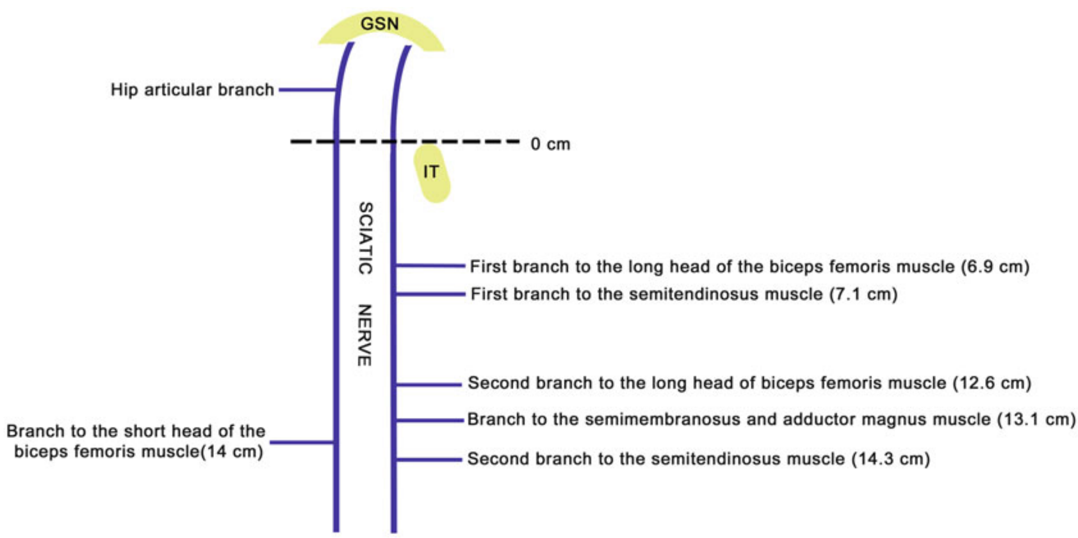


Fig. 6 Schematic showing the branches of the sciatic nerve before the physical separation in tibial and fibular nerves. The mean distance from the ischial tuberosity (*IT*) to the branch emergence is described between brackets. *GSN* greater sciatic notch, *IT* ischial tuberosity. Sunderland and Hughes [24] served as reference for the location of the BSH branch and Seidel et al. [26] for the other branches

complex and quadratus femoris muscle, located at an average of 1.2 ± 0.2 cm from the most lateral aspect of the ischial tuberosity and maintaining an intimate relationship with the hamstring origin [18] (Fig. 4). The sciatic nerve then enters the thigh posteriorly to the adductor magnus muscle and crosses anteriorly the long head of the biceps femoris. Next, the nerve runs between the semimembranosus and biceps before accessing the popliteal fossa.

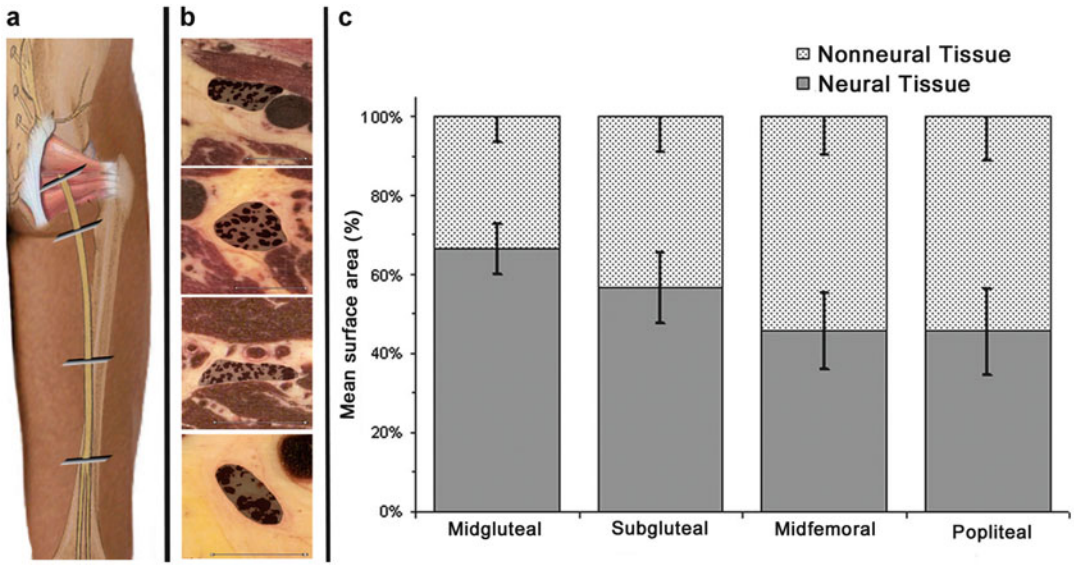


Fig. 7 Nonneural and neural tissue composition of the sciatic nerve at different locations. (**a**) Schematic diagram showing four locations of analysis: midgluteal, subgluteal, midfemoral, and popliteal sciatic nerve. (**b**) Transversal view of the sciatic nerve at the four locations, with details of the demarcated neural contents (*right*; *black dots*) and epineural areas (*gray fields*). (**c**) Relative values (percentages) of neural versus nonneural tissue inside the epineurium (means SDs) (Reprint with permission from: Moayeri and Groen [27])

Under normal conditions, the sciatic nerve is able to stretch and glide in order to accommodate moderate strain or compression associated with joint movement. During a straight leg raise with knee extension, the sciatic nerve experiences a proximal excursion of 28.0 mm [37] at 70–80° of hip flexion. Strain of the sciatic nerve increases 6.6 % relative to the extended hip [37]. Fleming et al. measured the sciatic nerve strain throughout ten hip arthroplasty procedures [38]. The strain increased on average 26 % during hip flexion with the knee in extension. This amount of strain is significant and may cause nerve dysfunction. An animal study reported the nerve conduction was completely blocked after stretching of 12 % of the nerve length for 1 h [39]. At 6 % strain, the authors found a decrease of 70 % in amplitude of the action potential after 1 h [39]. The changes in femoral bone morphology may influence sciatic nerve kinematics during hip mobilization [2]. Therefore, it is always important to assess osseous parameters, including femoral and acetabular version (Figs. 11 and 12). Hip flexion, adduction, and internal rotation increases the distance between the greater trochanter and posterior superior iliac spine and the distance between the greater trochanter and ischial tuberosity. This hip position stretches the piriformis muscle and causes a narrowing of the space between the inferior border of the piriformis, the superior gemellus, and the sacrotuberous ligament [40].

Etiology

The piriformis muscle and tendon are the most common source of extrapelvic sciatic nerve impingement. Yeoman first described the possibility of sciatic nerve entrapment by the piriformis muscle in 1928 [41]. The introduction of the term "piriformis syndrome" has been credited to Robinson, in 1947 [42]. The diagnostic resources have improved over the past decades, and a number of structures have been associated with sciatic nerve entrapment within the deep gluteal space: the piriformis muscle [2, 3, 11, 12, 43–49], fibrous bands containing blood vessels [2, 3, 43] (Fig. 13), gluteal muscles [1], gemelli-obturator internus complex [6, 7], hamstring muscles [4, 5], ischial tuberosity [8, 50], and space-occupying lesions

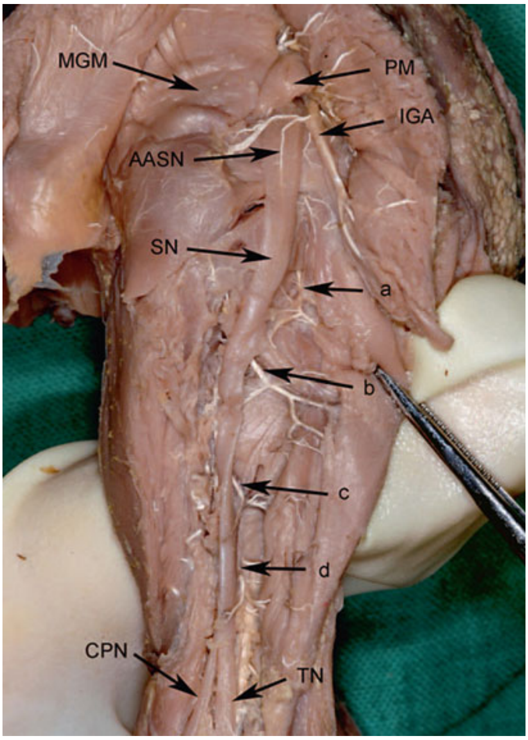
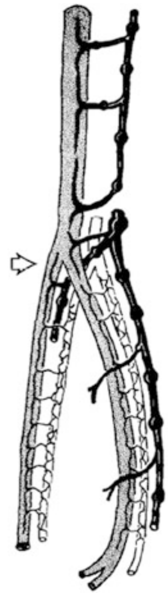


Fig. 8 Nutrient arteries of the fetal sciatic nerve in the gluteal and posterior femoral region; left fetal lower extremity. *MGM* Gluteus medius muscle, *PM* piriform muscle, *IGA* inferior gluteal artery, *AASN* accompanying artery of sciatic nerve, *SN* sciatic nerve, *CPN* common peroneal nerve, *TN* tibial nerve. a = Branch of medial circumflex femoral artery, *b* = first perforating artery, *c* = second perforating artery, *d* = third perforating artery (Reprint with permission from Ugrenovic et al. [31])

Fig. 9 Schematic diagram of the venous drainage of the sciatic nerves. *Arrows* designate the level of the knee. From proximal to distal the dominant venous drainage of the sciatic nerve is via the perforators of the profunda system in the thigh and directly to the popliteal vein at the knee. In the leg, the tibial and peroneal nerves drain predominantly to the plexus around their accompanying arteries as well as to muscular veins (Reprint with permission from Del Pinãl and Taylor [32])



[11, 12]. Additionally, vascular abnormalities [10, 47], prolonged surgery in the seated position [51], acetabular reconstruction surgery [52], and total hip replacement [53] have been reported to cause compression of the sciatic nerve. Considering the variation of anatomical structures causing the entrapment, the term "deep gluteal syndrome" [1] seems to be a more accurate description of this nondiscogenic sciatica.

The piriformis muscle is the most common source of sciatic nerve entrapment [2, 3, 11, 12, 43–49]. The risk of nerve compressive symptoms is increased by the existence of variation in the relationship between the piriformis muscle and the sciatic nerve. Six categories of piriformis-sciatic nerve variations have been identified [35] (Fig. 14). The prevalence of anomalies was 16.9 % in a meta-analysis of cadaveric studies [36] and 16.2 % in a review of published surgical case series [36] (Table 2). It is important to mention that the anomaly itself may not be the etiology of the Deep Gluteal Syndrome symptoms. Martin et al. [2] reported on 35 patients endoscopically treated for deep gluteal syndrome. Eighteen patients involved the piriformis muscle as etiology, including the sciatic nerve passing through the piriformis muscle or a portion of piriformis muscle/tendon passing through or anterior to the sciatic nerve [2]. A thick piriformis tendon hidden under the piriformis belly has been indentified as a cause of sciatic nerve compression (Fig. 15). Hypertrophy of the piriformis muscle has also been associated with sciatic nerve compression [12, 46, 47, 54]. However, of 14 patients with posttraumatic piriformis syndrome, Benson and Schutzer found that only two had larger piriformis muscles on the symptomatic side and seven appeared smaller than the unaffected side [44].

Atypical fibrovascular scar bands and hypertrophy of the greater trochanteric bursae have been reported in many cases of sciatic nerve entrapment [2, 3] (Fig. 13). In 27 of the 35 patients previously described by Martin et al. the greater trochanteric bursa was found to be excessively thickened, and large fibrovascular scar bands were present in many patients [2]. The fibrovascular bands

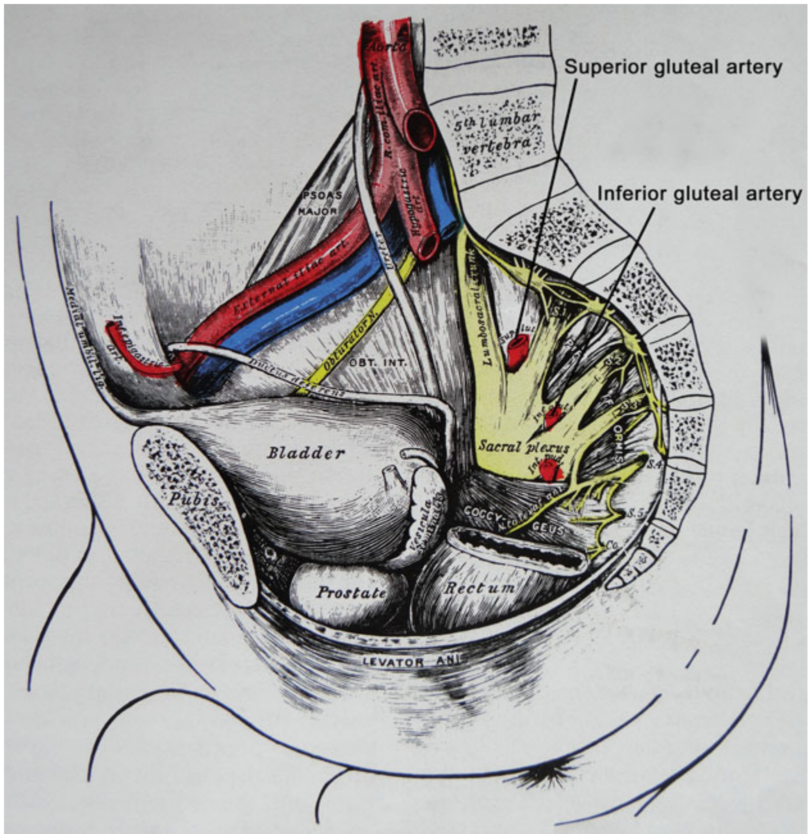


Fig. 10 Superior and inferior gluteal arteries crossing the sacral plexus before accessing the deep gluteal space

extended from the posterior border of the greater trochanter to the gluteus maximus on to the sciatic nerve and then proximally to the greater sciatic notch [2]. The obturator internus/gemelli complex is commonly overlooked in association with sciatica-like pain [6, 7, 15]. As the sciatic nerve passes under the belly of the piriformis and over the superior gemelli/obturator internus, a scissor effect between the two muscles can be the source of entrapment. In one case, Martin et al. found the obturator internus penetrating the sciatic nerve.

The sciatic nerve courses close to the hamstrings origin at the most lateral aspect of the ischial tuberosity (Fig. 4). Avulsions of the hamstring tendons or congenital fibrotic bands can affect the sciatic nerve causing symptoms of entrapment [4, 5, 55–57]. Other sources of sciatic nerve entrapment within the deep gluteal space include malunion of the ischium or healed avulsions, greater trochanter ischium impingement (Fig. 16), tumor, sciatic nerve venous varicosities [9] (Fig. 17), and gluteus maximus (from a prior iliotibial band release). Intra-articular hip disorders may also be involved with sciatic nerve symptoms. Patients submitted to surgical treatment of femoroacetabular impingement often recover hip mobility or can move the hip without having intra-articular pain. Considering that neural structures are sensitive to strain [39], increased mobility can lead to strains greater than normal in the sciatic nerve, triggering the sciatic nerve entrapment symptoms in patients with variations in the piriformis-sciatic nerve relationship. This factor may be even more important in patients with capsular laxity and abnormal bone morphologies, such as increased femoral version, retroversion, or greater trochanteric dysmorphism.

The fibers of the sciatic nerve can be also entrapped in the lumbar spine, pelvis, extrapelvic, and thigh. A discussion regarding intrapelvic etiologies of sciatic nerve entrapment will be provided in the differential diagnoses section.

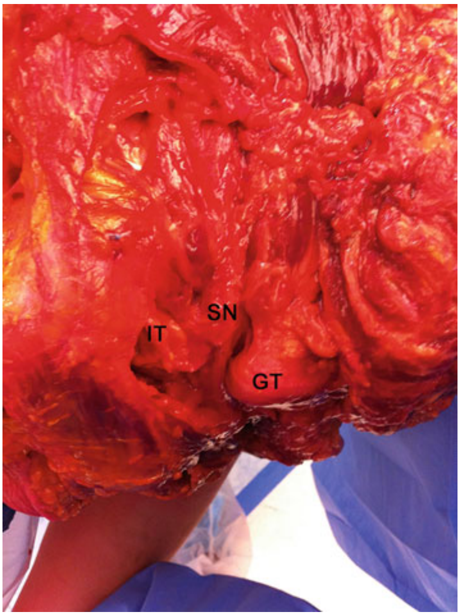


Fig. 11 Superior view of the sciatic nerve (*SN*) excursion between the greater trochanter (*GT*) and ischial tuberosity (*IT*) in a cadaveric specimen, right hip. The sciatic nerve runs in a tunnel formed by the greater trochanter and ischial tuberosity when the hip is flexed with the knee extended. This pattern of excursion of the sciatic nerve may change according to the bone morphology, adjacent soft tissue restriction, and knee position (flexion or extension)

The table 3 summarizes the etiologies of sciatic nerve entrapment reported within the literature.

Clinical Presentation and Ancillary Testing

History and Physical Examination

A comprehensive physical examination, a detailed history, and standardized radiographic interpretation are paramount in evaluating hip pain [2, 58, 59]. When assessing posterior hip pain, the physical examination will allow for an assessment of osseous, capsular labral, musculotendinous, and neurovascular etiologies. Additionally, it is important to recognize the coexistence of these pathologies. The lumbar spine, abdominal, and genitourinary problems are ruled out by history, physical examination, and ancillary testing. It is important to consider intrapelvic causes of sciatic nerve entrapment, particularly in patients with previous gynecologic surgical procedures and menses-related pain [13, 34]. In all cases of suspected sciatic nerve entrapment, the spine must first be ruled out by MRI and history/physical examination.

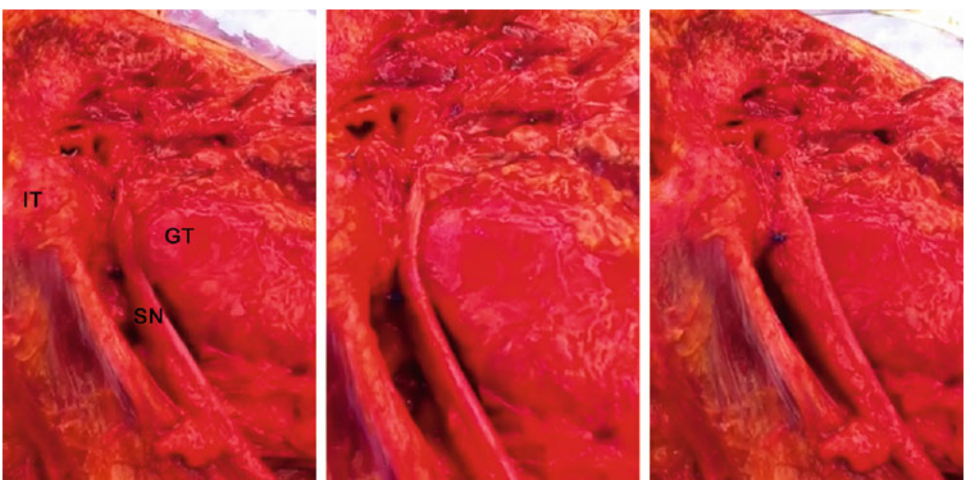


Fig. 12 Posterior view of the sciatic nerve (*SN*) excursion between the greater trochanter (*GT*) and ischial tuberosity (*IT*) in a cadaveric specimen, right hip. The sciatic nerve is forced posterior to the GT and ischium during increasing hip flexion and external rotation (images from *left* to *right*). The knee was flexed and the last figure represents a seated position. This pattern of excursion of the sciatic nerve may change according to the bone morphology, adjacent soft tissue restriction, and knee position (flexion or extension)

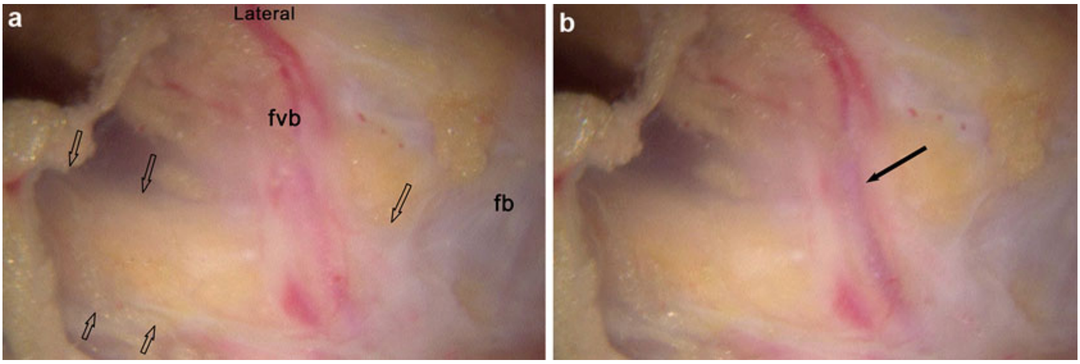


Fig. 13 Entrapment of the sciatic nerve by fibrovascular scar band, endoscopic visualization. (**a**) The sciatic nerve is indicated by the open arrows and is anterior to a fibrovascular band (*fvb*) and another fibrous band (*fb*). (**b**) Same view demonstrating the blood flow through a vessel in the fibrovascular band (*black arrow*)

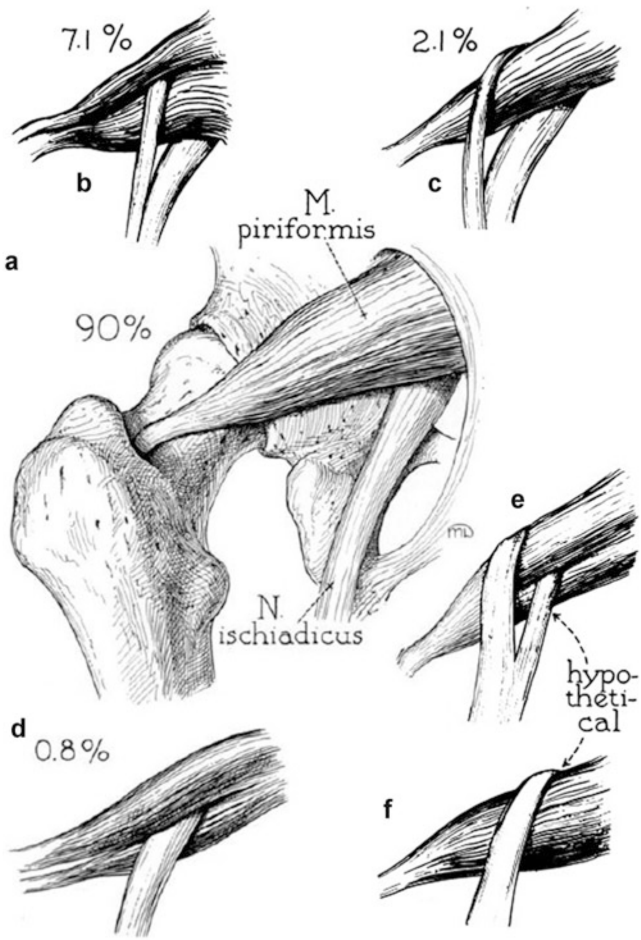


Fig. 14 Schematic of piriformis/sciatic nerve variants. Six types of arrangement of the sciatic nerve, or of its subdivisions in relation to the piriformis muscle, arranged in the order of frequency [35]. Gluteal (external) view. The percentage incidence in 240 examples is indicated. Figures **e** and **f** were hypothetical in 1938 [35]. (**a**) Nerve undivided passes out of greater sciatic foramen, below piriformis muscle, (**b**) divisions of nerve pass through and below heads of muscle, (**c**) divisions above and below undivided muscle, (**d**) nerve undivided between the heads of muscle, (**e**) divisions of nerve between and above heads, and (**f**) undivided nerve above undivided muscle (Figure reprinted from Beaton and Anson [35])

Table 2 Characteristics of cadaveric studies: piriformis and sciatic nerve variants

Investigator	Number of cadavers (number of sides included)		Percentage of female cadavers	Laterality of the anomalies		Notes
				Unilateral	Bilateral (%)[a]	
Parsons and Keith [91]	69	(138)	"Mostly men"	–	–	
Bardeen [92]	123	(246)[b]	30	–	–	
Trotter [93]	232	(464)	21	24	48 (63.6)	
Beaton and Anson 1937[d]	60[b]	(120)	–	5	14 (73.6)	
Beaton and Anson [35][c]	60	(120)	–	3	2 (40.0)	Results presented in this study included data from a previous study
Ming-Tzu [95]	70	(140)	"Mostly men"	22	24 (52.2)	
Misra [96]	150	(300)	–	–	–	
Anson and McVay [97][c]	1,004[b]	(2,000)	–	–	–	This data set incorporated data from Beaton and Anson's two previous studies
Nizankowski et al. [98]	100	(200)	45	–	–	
Lee and Tsai [99]	84	(168)	13	–	–	
Pecina [100]	65	(130)	–	–	–	
Chiba [101]	257	(511)	46	–	–	Three lower limbs of males excluded
Chiba et al. [102]	221	(442)	–	–	–	
Pokorny et al. [103]	51	(102)	–	–	–	
Fishman et al. [104]	38	(76)	–	1	10 (91.0)	This data was found in a study presenting data on the usage of H-reflex latencies for diagnosing piriformis syndrome
Benzon et al. [71]	36	(66)	–	1	0	In six cadavers, only one side was studied
Agur and Dalley [105]	320[b]	(640)	–	–	–	
Ugrenovic et al. [106]	100	(200)	–	–	–	This study was carried out on human fetuses
Pokorny et al. [107]	91	(182)[b]	–	–	–	
Guvencer et al. [108]	25	(50)	0	–	–	
Total	See Table 3			56	98 (63.6)[e]	

[a]Total number of cadavers used as a denominator
[b]Information derived from data presented in the text
[c]This study had inconsistencies in its reporting of statistics
[d]Omitted from total as these results have been included in Anson and McVay (1971)
[e]95 % confidence interval = 55.5–71.2 %. Reprinted with permission from Smoll [36]

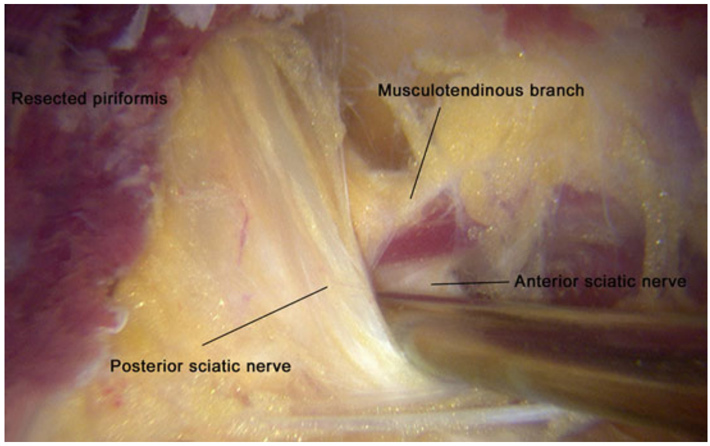


Fig. 15 Ancillary musculotendinous branch through the sciatic nerve

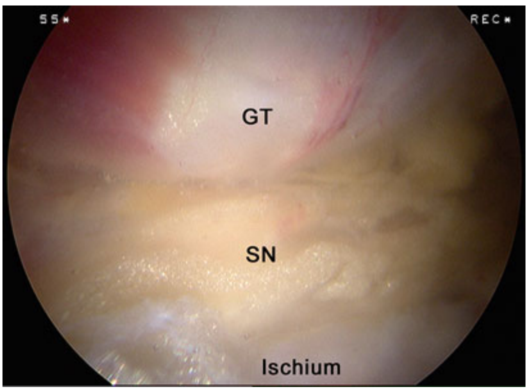


Fig. 16 Endoscopic view of sciatic nerve (*SN*) compression between the greater trochanter (*GT*) and ischial tuberosity. With hip flexion and external rotation, the sciatic nerve was not able to move due to the ischial outgrowth of bone

Patients presenting with sciatic nerve entrapment often have a history of trauma and symptoms of sit pain (inability to sit for more than 30 min), radicular pain of the lower back or hip, and paresthesias of the affected leg [2, 44]. Patients may present with neurological symptoms of abnormal reflexes or motor weakness [60]. Some symptoms may mimic a hamstring tear or intra-articular hip pathology such as aching, burning sensation, or cramping in the buttock or posterior thigh. Symptoms of sit pain can also be caused by pudendal nerve entrapment, in which the pain is medial to the ischium and will be discussed later in this chapter. Upon palpation of the piriformis, Robinson described a tender sausage-shaped mass as a key feature of what he termed "piriformis syndrome" [42]. Physical examination tests that have been used for the clinical diagnosis of sciatic nerve entrapment include passive stretching tests and active contraction tests. The space between the piriformis and obturator internus muscles narrows with flexion, adduction, and internal rotation [40].

The seated piriformis stretch test (Fig. 18a) is a flexion, adduction with internal rotation test performed with the patient in the seated position [58]. The examiner extends the knee (engaging the sciatic nerve) and passively moves the flexed hip into adduction with internal rotation while palpating 1 cm lateral to the ischium (middle finger) and proximally at the sciatic notch (index finger). A positive test is the recreation of the posterior pain at the level of the piriformis or external rotators. An active piriformis test (Fig. 18b) is performed by the patient pushing the heel down into the table, abducting and externally rotating the leg against resistance, while the examiner monitors the piriformis. In a recent published study, the combination of the seated piriformis stretch test with the piriformis active test presented a sensitivity of 91 % and specificity of 80 % for the endoscopic finding of sciatic nerve entrapment [61].

The palpation of the gluteal structures is fundamental for the diagnosis of gluteal and sit pain. The patient sits with the pelvis square to the examination table and the ischial tuberosity (IT) serves as the reference point for palpation

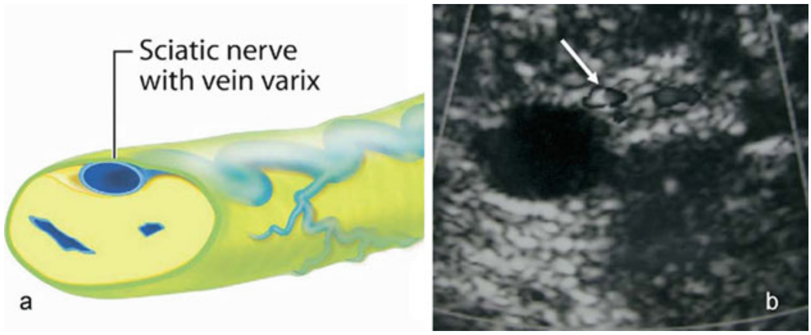


Fig. 17 Varicose veins within the sciatic nerve. (**a**) Schematic drawing of varicose veins within the perineurium and the sciatic nerve. (**b**) Sciatic nerve at midthigh with varicose veins within the nerve (*arrow*) in a patient who presented with pain and swelling. A larger refluxing vein is seen as an adhesion with the nerve (Reprinted with permission from Labropoulos et al. [9])

(Fig. 19). Pain superolateral to the IT at the sciatic notch is characteristic of deep gluteal syndrome [2]; pain lateral to the IT, ischial tunnel syndrome or ischiofemoral impingement is considered; pain at the IT, hamstrings tendons pathologies are possible; and pain medial to the IT, pudendal nerve entrapment is considered. An active knee flexion test against resistance, at 30° symptomatic with pain relief at 90°, can help evaluate the proximal hamstring tendons and sciatic nerve subluxation into the ischial tunnel [5].

Ischial tunnel syndrome or hamstring syndrome is described as pain in the lower buttock region that radiates down the posterior thigh to the popliteal fossa and is commonly associated with hamstring weakness [4]. This syndrome is related to sciatic nerve entrapment by scarring or a fibrotic band at the lateral insertion of the hamstring tendons to the ischial tuberosity [4, 5]. Patients experience pain with sitting, stretching, and with exercise, primarily running (sprinting and acceleration) [5, 62]. Palpable tenderness is located around the ischial tuberosity in the proximal hamstring region. Clinically, Young et al. reported that the straight leg raise test (Lasègue test) is slightly positive without neurological deficit. Marked weakness of the hamstring muscle at 30° knee flexion yet normal strength at 90° knee flexion is a suggestive finding in diagnosis [5].

Symptoms related to other nerves may be observed in cases of sciatic nerve entrapment, such as weakness of the gluteus medius and minimus muscles (superior gluteal nerve), weakness of the gluteus maximus (inferior gluteal nerve), perineal sensory loss (pudendal nerve), or loss of posterior cutaneous sensation (posterior femoral cutaneous nerve) (Table 1) (Fig. 20).

Ancillary Testing

Guided injections are useful to support the diagnosis of DGS, mainly when the piriformis is involved. Computed tomography, fluoroscopy, ultrasonography, electroneuromyography, or magnetic resonance imaging is useful to obtain more precise injections [46]. The results and techniques for injections in deep gluteal space will be discussed in more detail in the treatment section. The association of physical examination and injection is also utilized to rule out intra-articular hip pathologies, nerve root compression at lumbar spine, and pudendal nerve entrapment.

Electromyography and nerve conduction studies may assist with the diagnosis of deep gluteal syndrome. Piriformis entrapment of the sciatic nerve is often indicated by H-reflex disturbances of the tibial and/or fibular nerves [64, 65]. It is important to compare side to side and perform a dynamic test with the knee in extension and hip in adduction with internal rotation and held for a minimum of three minutes in this position. This position will tighten the piriformis muscle compressing the sciatic nerve sufficiently to disturb nerve conduction distally. Patients presenting with symptoms of sciatic nerve entrapment may fail to exhibit paraspinal denervation even when radiculopathy coexists [64]. Although

Table 3 Entrapments of the sciatic nerve within the deep gluteal space in key publications

Piriformis muscle	Martin et al. [2], Guvencer et al. [40], Papadopoulos and Kahn [60], Adams [43], Beauchesne and Schutzer [11], Benson and Schutzer [44], Chen [12], Dezawa et al. [45], Filler et al. [46], Hughs et al. [47], Mayrand et al. [48], Sayson et al. [48], Vandertop and Bosma [3], McCrory and Bell [1]
Hamstring muscles	Martin et al. [2], Puranen and Orava [4], Young et al. [5]
Gemelli-obturator internus complex	Martin et al. [2], Meknas et al. [15], Cox and Bakkum [6], Meknas et al. [7]
Fibrous bands containing blood vessels	Martin et al. [2], Adams [43], Vandertop and Bosma [3]
Ischial tuberosity	Miller et al. [8], Patti et al. [78], Torriani et al. [75]
Sciatic varicosities and vascular abnormalities	Martin et al. [2], Papadopoulos and Kahn [60], Hughs et al. [47], Papadopoulos, et al. 60], Labopoulos 2009
Gluteal muscles	Martin et al. [2], McCrory and Bell [1]
Acetabular reconstruction surgery	Issack et al. [52]
Prolonged surgery in the seated position	Brown et al. [51]
After total hip replacement	Uchio et al. [53]
Secondary to space-occupying lesions	Beauchesne and Schutzer [11], Chen [12]
Intrapelvic gynecologic and vascular abnormalities	Possover [13] and Possover et al. [34]

electrodiagnostic assessment can be useful when associated with adequate physical examination and injection tests, obesity, edema, and age can impair the acquisition of sensory nerve action potentials in the lower limb, principally for the proximally located nerves [66]. Moreover, asymptomatic patients (usually elderly) often present neurogenic changes in electrodiagnostic studies [66]. These features may be problematic for the differential diagnosis between lumbosacral and peripheral entrapment [66].

High resolution magnetic resonance imaging (MRI) is a useful imaging method for evaluation of sciatic nerve entrapment. The sciatic nerve anatomy and potential sources of compression can be assessed through this imaging method, including anomalies of the piriformis muscle, scar from proximal hamstring avulsion, osseous compression (Fig. 21), and intrapelvic abnormalities (Fig. 22). The MRI is also helpful in detecting direct and indirect signs of nerve injury [67]. Hyperintensity on fluid-sensitive images that are focal or similar to that of adjacent vessels is more likely to be significant [67]. However, hyperintensity in peripheral nerves may be seen in normal nerves due to the artifact known as magic angle effect [68]. Abnormalities in nerve size, fascicular pattern or blurring of the perineural fat tissue are suggestive of neural injury, although those features are difficult to note in small diameter nerves [67]. The main indirect sign of nerve entrapment injury is the muscular denervation edema [69]. In addition to sciatic nerve compression assessment, the MRI is important to rule out spine issues, intra-articular hip pathology, and other differential diagnoses. Despite the usefulness of MRI in the diagnosis of deep gluteal pain, the potential false-positive and false-negative results reinforce the importance of a proficient physical examination. Ultrasonography is a valuable method to guide nerve blocks and has been increasingly utilized for nerve assessment, with the advantages of dynamic evaluation and Doppler assessment of the vascular nerve supply [9].

The diagnosis of sciatic nerve entrapment is established through the combination of physical examination, imaging studies, and piriformis injection test.

Treatment

Nonoperative Treatment

The nonoperative treatment for deep gluteal syndrome begins addressing the suspected site of entrapment. Compression from a hypertrophied,

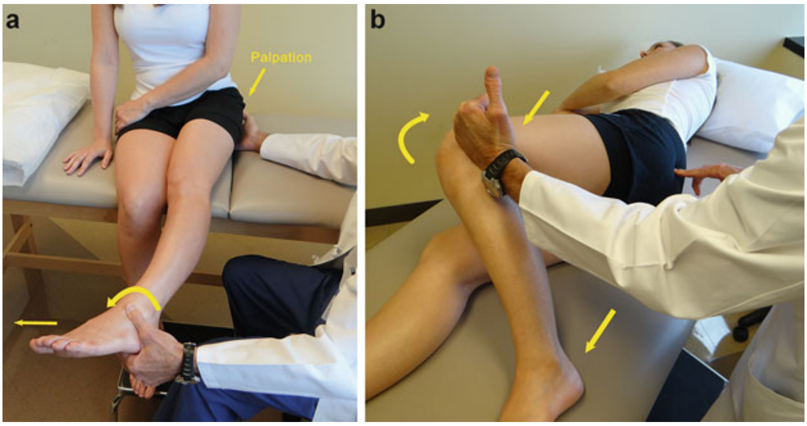


Fig. 18 (**a**) Seated piriformis stretch test. The patient is in the seated position with knee extension. The examiner passively moves the flexed hip into adduction with internal rotation while palpating 1 cm lateral to the ischium (middle finger) and proximally at the sciatic notch (index finger). (**b**) Active piriformis test. With the patient in the lateral position, the examiner palpates the piriformis. The patient drives the heel into the examining table thus initiating external hip rotation while actively abducting and externally rotating against resistance

Fig. 19 Palpation of the deep gluteal space. The examiner palpates the gluteal area using the ischial tuberosity (*IT*) as reference: (*1*) superolateral at the piriformis muscle/sciatic nerve (index finger); (*2*) moving the index finger to palpate lateral to the IT, ischiofemoral impingement and ischial tunnel syndrome; (*3*) at the IT, hamstrings origin tendinopathy, and avulsion (middle finger); (*4*) medial at the obturator internus/pudendal nerve (ring finger)

contracted, or inflamed muscle (piriformis, quadratus femoris, obturator internus, superior/inferior gemellus) is initially treated with rest, anti-inflammatories, muscle relaxants, and physical therapy. The physical therapy program should include stretching maneuvers aimed at the external rotators. The piriformis stretch, or FAIR, involves placing the hip in flexion, adduction, and internal rotation (Fig. 23a). Patients with CAM impingement, anterior pincer impingement, or acetabular retroversion may not be able to stretch adequately into this position and should be evaluated and treated primarily for the intra-articular pathology, as most will resolve with appropriate surgical intervention. In a seated position, the patient brings the knee into the chest and across midline and pulls the knee to the opposite shoulder for 20 seconds. Sciatic nerve glides and hip circumduction exercises are useful to maintain the sciatic nerve excursion and should be gently performed (Fig. 23b and c). Additional physical therapy techniques that may be helpful include ultrasound and electrical stimulation. Patients with more intense or acute symptoms may not

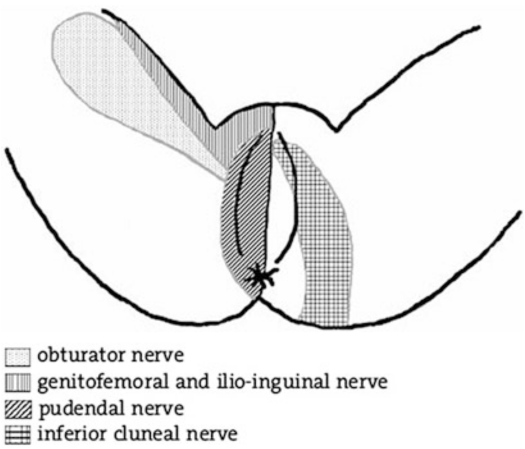


Fig. 20 Sensory zones of the perineum in female. The sensitive innervation territory is marked according to the nerve. The *dotted area* represents the obturator nerve territory. The *vertical lines* represent the genitofemoral and ilioinguinal nerves. The *oblique lines* represent the pudendal nerve. The *crossed lines* denote the inferior cluneal nerve innervation. Although the figure illustrate well-defined areas of innervation, it is important to remember that an overlap in dermatomes is frequent (Figure reprinted with permission from Labat et al. [63])

tolerate positions of hip flexion associated with knee extension. In this situation, a knee brace to avoid knee extension and relieve the sciatic nerve of tension may be helpful for some patients. The brace is adjusted according to the straight leg raise test and gradual extension of the knee is performed toward full extension over the course of 4–6 weeks.

Guided injections of anesthetic or corticosteroid into the piriformis muscle can provide pain relief in patients not responding to physical therapy. It is important to administer the injection to the correct site and different techniques can be utilized for guidance, including fluoroscopy, CT, ultrasound, electromyography, and MRI. A trial of three injections has been recommended before opting for more aggressive therapy, taken on a case-by-case basis [46, 60, 70]. The literature has reported variable results for piriformis injection [46, 71, 72]. Pace and Nagle reported a double injection technique of Kenalog and Xylocaine toward the piriformis muscle which relieved the pain in 41 out of 45 patients [72]. Filler et al. reported lasting pain relief in 37 out of 162 patients following one or two injections of Marcaine and Celestone [46]. The piriformis muscle may be also injected with botulinum toxin [73]. Another alternative is the perisciatic nerve injection of anesthetic and corticosteroid instead of intra-piriformis muscle [74]. Many cases of deep gluteal syndrome/sciatic nerve entrapment will respond to nonoperative measures.

Operative Treatment

The operative treatment for deep gluteal syndrome is discussed in Chap. 75, "► Surgical Technique: Endoscopic Sciatic Nerve Release" describing the endoscopic technique for sciatic nerve decompression.

Differential Diagnoses

Numerous muscular and neurovascular structures course between the deep gluteal space and adjacent anatomical areas. This complex anatomy makes the diagnosis of sciatic nerve entrapment challenging in some patients. Lumbar spine problems are the most frequent cause of sciatica and can manifest with gluteal pain. Intra-articular hip abnormalities can also produce posterior hip pain. Both lumbar spine and intra-articular pathology should be always ruled out through a comprehensive physical examination, imaging studies, and injection tests. The following section will discuss the main differential diagnoses for sciatic nerve entrapment located in the deep gluteal space.

Ischiofemoral Impingement

Ischiofemoral impingement (IFI) is described as abnormal osseous contact between the ischium and the lesser trochanter of the femur. This abnormality is associated with narrowing of the ischiofemoral space and inflammatory changes in the quadratus femoris muscle [75]. Johnson, in 1977, first described IFI in three patients following osteotomy or arthroplasty of the hip [76]. Only recently IFI has been identified as a

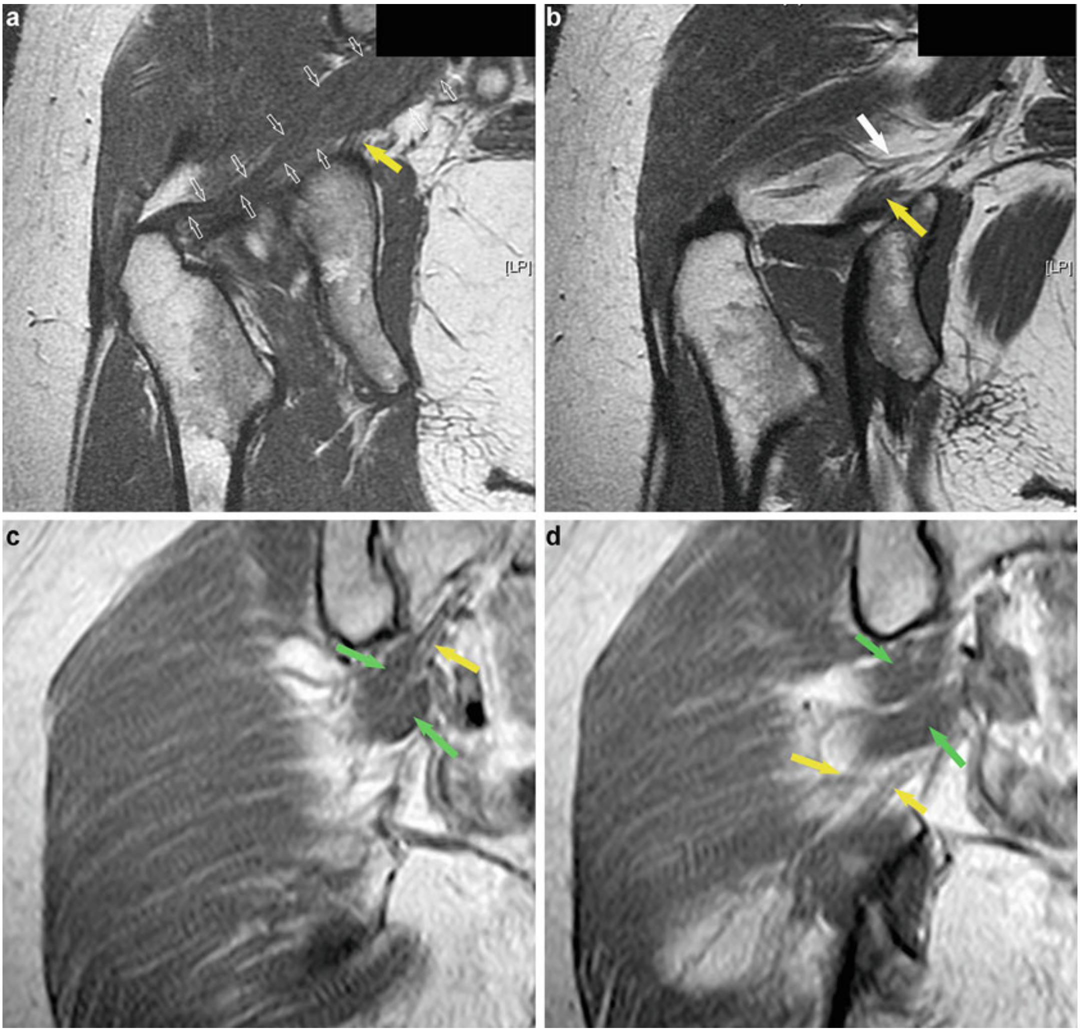


Fig. 21 Magnetic resonance images of deep gluteal space, coronal view of the right hip. (**a**) Normal relationship between the piriformis muscle (*open arrows*) and the sciatic nerve (*yellow arrow*). (**b**) More posterior cut of the same hip demonstrating the inferior gluteal artery (*white arrow*) leaving the sciatic notch close to the sciatic nerve (*yellow arrow*). (**c**) and (**d**) Variation of the sciatic nerve/piriformis relationship in a patient with deep gluteal syndrome. The superior division of the sciatic nerve (*yellow arrow* in **c**) is demonstrated crossing between the two piriformis muscle portions (*green arrows*)

source of hip symptoms in patients without trauma or previous surgery [77, 78]. Patients with IFI usually complain of deep posterior gluteal pain lateral to the ischium and limitation on physical activities. Symptomatic individuals grab the hip lateral to ischium during long-stride walking, while pain is alleviated with short-stride walking. In the physical examination, there is pain on palpation lateral to the ischium tuberosity and a positive ischiofemoral impingement test. This test is performed with the patient in contralateral decubitus and taking the affected hip into passive extension in neutral and adduction. A test is considered positive when it reproduces the symptoms with the examined hip extended and adducted or neutral, while extension with abduction does not reproduce the symptoms (Fig. 24).

The imaging findings of IFI include narrowing of the ischiofemoral space and quadratus femoris space associated with inflammatory findings in

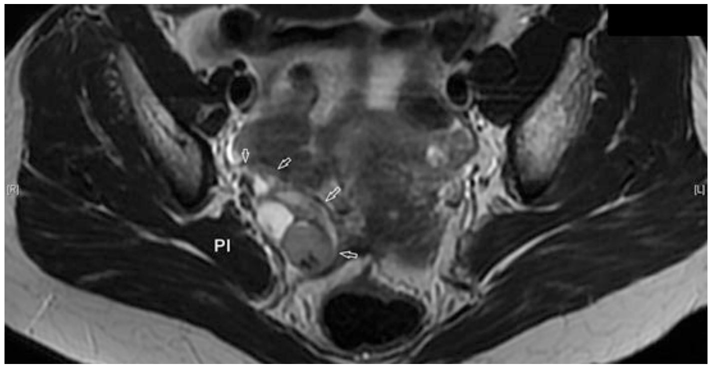


Fig. 22 Magnetic resonance imaging of the pelvis demonstrating an ovarian cyst in the right side (*open arrows*). The sacral plexus and gluteal vessels are located between the piriformis muscle (*PI*) and the ovarian cyst

Fig. 23 Piriformis stretch, sciatic nerve glides, and hip circumduction. (**a**) The piriformis stretch is performed in a seated position. The patient brings the knee toward the opposite shoulder. (**b**) For the sciatic nerve glides, the patient first performs cervical extension and plantar flexion of the ankle, followed by cervical flexion with ankle dorsiflexion. (**c**) Circumduction: performed in supine position with gentle passive movements, with the knee and ankle parallel to the body longitudinal axis Begin at 45° flexion with the leg abducted to the side at 90°, then adduct moving the hip into internal rotation toward midline allowing for the lateralization of the sciatic nerve (depicted in Figure 12)

the quadratus femoris muscle [75]. The ischiofemoral and quadratus femoris spaces are measured in the MRI and utilized for the diagnosis [75] (Fig. 25). Suggested cutoff values are ≤17 mm for the ischiofemoral space and ≤8 mm for the quadratus femoris space [75]. Abnormalities of the quadratus femoris muscle included edema, partial tear, and fatty infiltration [75]. The hamstring tendons of affected subjects may present edema and partial tears [75].

There are only a few case reports of open surgical treatment for correction of IFI [76, 79, 80]. Johnson was the first to describe lesser trochanter resection for IFI using open approach in three patients with previous hip surgery [76]. Open resection of lesser trochanter [79] or ischium tuberosity [80] has recently been described for IFI in patients with no prior history of surgery. Martin et al. reported on five cases of endoscopic partial resection of the lesser trochanter [81]. The mean mHHS improved from 51.3 points (range 34.1–73.7 points) preoperatively to 92.4 points (range 78.1–100 points) at 2 year follow-up. All patients improved their mHHS and the mean improvement was 41.1 points (range, 26.3–61.6 points).

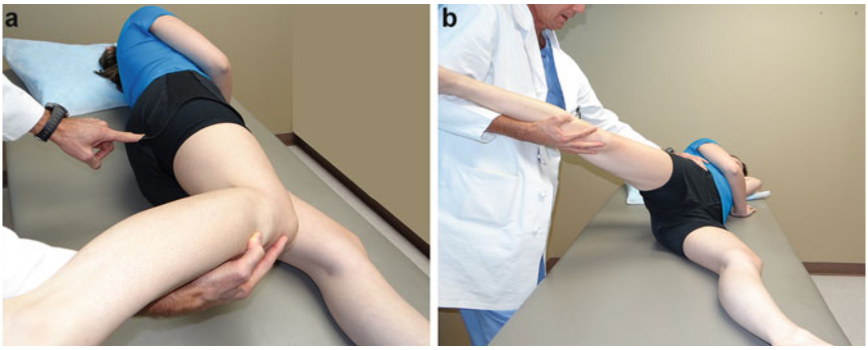


Fig. 24 The ischiofemoral impingement test is utilized in the differential diagnosis of posterior hip pain. (**a**) Impingement between the lesser trochanter and ischium is assessed by passive extension of the hip, reproducing the patient's symptoms with the examined hip adducted or neutral. The *left index finger* of the examiner is palpating the ischiofemoral space lateral to the ischium. (**b**) Extension and abduction of the hip does not reproduce the symptoms

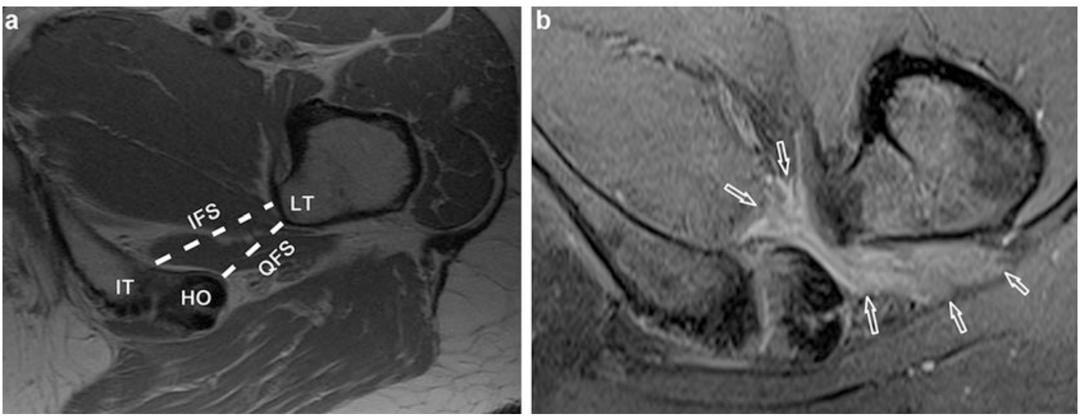


Fig. 25 MRI assessment for ischiofemoral impingement. (**a**) Normal ischiofemoral space (*IFS*) and quadratus femoris space (*QFS*), *IT* ischial tuberosity, *HO* hamstrings origin, *LT* lesser trochanter. (**b**) MRI of a patient with ischiofemoral impingement showing a narrowed ischiofemoral space with edema of the quadratus femoris muscle (*arrows*)

Proximal Hamstring Tendon Lesions

Gluteal pain can be result of avulsion and tendinopathy of the proximal hamstrings origin at ischial tuberosity (IT). Avulsions may have an acute or chronic presentation and cause gluteal pain with long-term functional disability [57]. Although runners are more often affected, proximal hamstring avulsion may be present without a clear history of sports injury. The symptoms include pain with sitting, stretching, and exercising. Snapping of the avulsed hamstring tendon over the IT has also been described [82]. In the physical examination, pain is elicited by stretching of the hamstrings and palpation on the IT. Active knee flexion against resistance exacerbates the symptoms, with knee at 30° of flexion rather than 90° of flexion secondary to the change in vector forces of subluxation that can irritate the sciatic nerve [5]. The proximal origin of the hamstrings has an intimate relationship with the sciatic nerve [18] and this nerve can be entrapped in advanced stages of inflammation and fibrosis, representing the ischial tunnel syndrome or hamstring syndrome [4, 15, 83]. MRI findings may include partial tearing (Fig. 26), tendon thickening, fatty infiltration, fluid collection at the

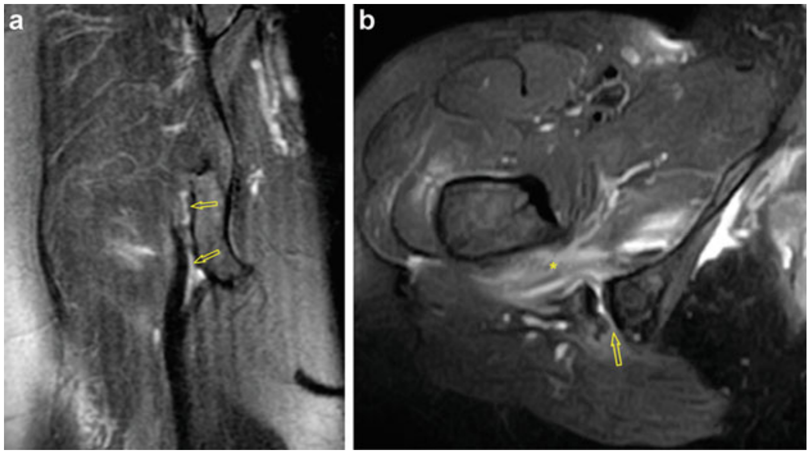


Fig. 26 MRI of partial detachment of the hamstring tendon from ischium. (**a**) Sagittal view of the avulsion (*arrows*). (**b**) Axial view of the same hip confirming the detachment (*arrow*) and the edema in adjacent structures (*)

margins of the tendon, and in advanced stages tethering of the sciatic nerve. Ischial bursitis should also be considered a cause of pain at the ischial tuberosity [84] and may be related with hamstring proximal avulsions. Considering hamstring avulsions can lead to sciatica by scarring around the sciatic nerve, surgical repair has been proposed early in order to avoid involvement of the sciatic nerve [5, 55, 57].

Pudendal Nerve Entrapment

The goal in developing this section is to raise awareness of a very disabling form of posterior hip pain. Neuropathic pain in the distribution of the pudendal nerve with sensations of burning, tearing, stabbing lightning-like, electrical, sharp shooting, and/or foreign body sensation [85]. Pain is made worse with sitting (alleviated with toilet stool sitting), reduced with standing, and absence upon awakening and progressing through the day. The common historical etiologies include childbirth, prolonged sitting, trauma, and cycling. Pelvic inclination, sacral slope orientation, or sacral dysmorphism may have an effect on the development of pudendal nerve pain. The etiology of pudendal nerve entrapment requires further definition. However, suspicion of this pathology can be validated through a comprehensive history, physical examination, and appropriate ancillary tests, especially imaging-guided injections.

The pudendal nerve can become entrapped in several locations from the greater sciatic notch to the lesser sciatic notch and even distally to the obturator internus/levator ani fascia (Fig. 27). Four main types of pudendal nerve entrapment are based upon the location of entrapment, which is very important for injections [19]: type I, at the exit of the greater sciatic notch accompanied by piriformis muscle spasm; type II, at the ischial spine, sacrotuberous ligament, and lesser sciatic notch entrance; type III, at the entrance of the Alcock canal associated with the obturator internus muscle spasm; and type IV, distal entrapment of terminal branches [19].

The diagnosis of pudendal neuralgia has been primarily clinical and empirical [63]; however, progress in clinical nerve imaging and injection techniques is aiding in the differential diagnosis of pudendal nerve entrapment [19]. In 2008, Labat et al. described the Nantes Criteria with five essential diagnostic criteria: (1) pain in the anatomical territory of the pudendal nerve (Fig. 20), (2) worsened by sitting (although pain is relieved when sitting on a toilet seat), (3) the pain does not wake the patient at night, (4) pain with no objective sensory impairment, and (5) pain is relieved by diagnostic pudendal nerve block [63]. Also defined in the report are complementary diagnostic criteria, exclusion criteria, and associated signs not excluding the diagnosis [63]. Neurophysiologic testing techniques have been used to aid in diagnosis [85]. The physical examination is useful for preliminarily sorting patients into four categories: type I, sciatic notch tenderness only; type II, midischial tenderness; type IIIa, obturator

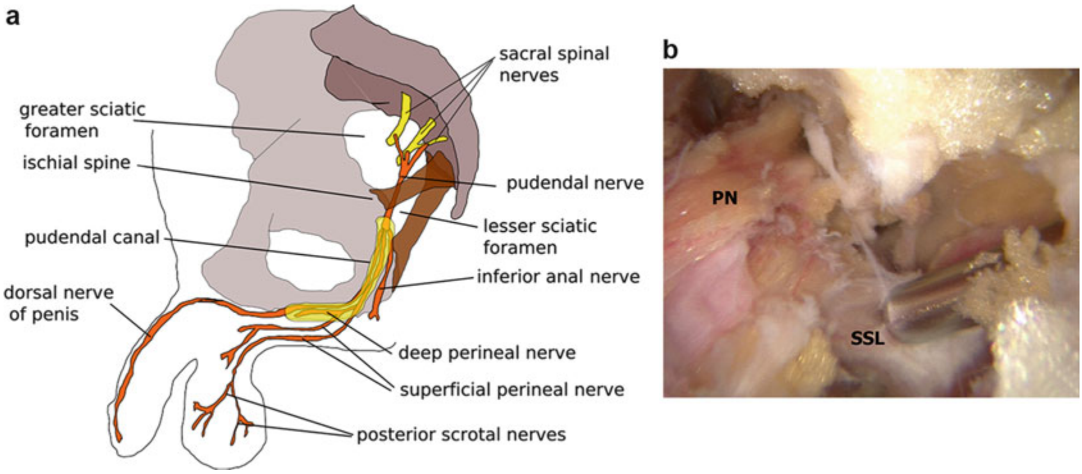


Fig. 27 The course of the pudendal nerve. (**a**) The pudendal nerve exits the pelvis through the greater sciatic notch passing over the sacrospinous ligament and reenters the pelvis through the lesser sciatic notch. In sequence, the nerve enters the Alcock canal (*yellow*) formed by the obturator fascia and sacrotuberous ligament (Reprinted with permission from Moore and Dalley [20]). (**b**) Endoscopic view of a cadaveric sacrospinous ligament (*SSL*) and its relationship to the pudendal nerve (*PN*)

internus muscle tenderness only; type IIIb, obturator and piriformis muscle tenderness; and type IV, no palpable tenderness. MRI can then be helpful to identify abnormalities in nerve or adjacent muscles/vessels [19].

The conservative treatment is based on pain medications, physical therapy of the pelvic floor muscles, and guided nerve anesthetic blocks including steroids [19]. The point of injection may be defined according the categories previously described [19]. The published outcomes of serial pudendal nerve blocks in controlling pain after 1 year range from 12 % to 87 % [19, 86, 87]. Surgical decompression may be considered when nonsurgical treatments have failed and is traditionally performed through trans-gluteal open approaches [19, 85, 88, 89].

Intrapelvic Sciatic Entrapment

The sciatic nerve is anatomically close to vascular and urogynecologic structures in the pelvis. This relationship is a potential source of nerve entrapment and sciatica can be caused by vascular entrapment of sacral neural roots or abnormalities of the ovary. Endometriosis should particularly be considered in patients with menses-related sciatica [13]. The endometriotic tissue can cause sciatica without a direct contact with the neural tissue, and the suggested cause is the stimulation of the sacral plexus by inflammation of the retroperitoneum [90].

Summary

Intra-articular and extra-articular hip pathologies may coexist. A comprehensive history and physical examination is required to distinguish osseous, capsular labral, musculotendinous, and neurovascular etiologies. A detailed understanding of anatomy, biomechanics, and pathokinematics is required to appreciate disorders of the deep gluteal space and to direct the appropriate treatment of this complex pathology.

References

1. McCrory P, Bell S. Nerve entrapment syndromes as a cause of pain in the hip, groin and buttock. Sports Med. 1999;27:261–74.
2. Martin HD, Shears SA, Johnson JC, Smathers AM, Palmer IJ. The endoscopic treatment of sciatic nerve entrapment/deep gluteal syndrome. Arthroscopy. 2011;27:172–81.
3. Vandertop WP, Bosma NJ. The piriformis syndrome. A case report. J Bone Joint Surg Am. 1991;73:1095–7.

4. Puranen J, Orava S. The hamstring syndrome. A new diagnosis of gluteal sciatic pain. Am J Sports Med. 1988;16:517–21.
5. Young IJ, van Riet RP, Bell SN. Surgical release for proximal hamstring syndrome. Am J Sports Med. 2008;36:2372–8.
6. Cox JM, Bakkum BW. Possible generators of retrotrochanteric gluteal and thigh pain: the gemelli-obturator internus complex. J Manipulative Physiol Ther. 2005;28:534–8.
7. Meknas K, Kartus J, Letto JI, Christensen A, Johansen O. Surgical release of the internal obturator tendon for the treatment of retro-trochanteric pain syndrome: a prospective randomized study, with long-term follow-up. Knee Surg Sports Traumatol Arthrosc. 2009;17:1249–56.
8. Miller A, Stedman GH, Beisaw NE, Gross PT. Sciatica caused by an avulsion fracture of the ischial tuberosity. A case report. J Bone Joint Surg Am. 1987;69:143–5.
9. Labropoulos N, Tassiopoulos AK, Gasparis AP, et al. Veins along the course of the sciatic nerve. J Vasc Surg. 2009;49(3):690–6.
10. Papadopoulos SM, McGillicuddy JE, Albers JW. Unusual cause of 'piriformis muscle syndrome'. Arch Neurol. 1990;47:1144–6.
11. Beauchesne RP, Schutzer SF. Myositis ossificans of the piriformis muscle: an unusual cause of piriformis syndrome. A case report. J Bone Joint Surg Am. 1997;79:906–10.
12. Chen WS. Sciatica due to piriformis pyomyositis. Report of a case. J Bone Joint Surg Am. 1992;74:1546–8.
13. Possover M. Laparoscopic management of endopelvic etiologies of pudendal pain in 134 consecutive patients. J Urol. 2009;181:1732–6.
14. Loukas M, Louis Jr RG, Hallner B, Gupta AA, White D. Anatomical and surgical considerations of the sacrotuberous ligament and its relevance in pudendal nerve entrapment syndrome. Surg Radiol Anat. 2006;28:163–9.
15. Meknas K, Christensen A, Johansen O. The internal obturator muscle may cause sciatic pain. Pain. 2003;104:375–80.
16. Solomon LB, Lee YC, Callary SA, Beck M, Howie DW. Anatomy of piriformis, obturator internus and obturator externus: implications for the posterior surgical approach to the hip. J Bone Joint Surg Br. 2010;92:1317–24.
17. Standring S. Grays anatomy the anatomical basis of clinical practice. London: Churchill Livingstone Elsevier; 2008.
18. Miller SL, Gill J, Webb GR. The proximal origin of the hamstrings and surrounding anatomy encountered during repair. A cadaveric study. J Bone Joint Surg Am. 2007;89:44–8.
19. Filler AG. Diagnosis and treatment of pudendal nerve entrapment syndrome subtypes: imaging, injections, and minimal access surgery. Neurosurg Focus. 2009;26:E9.
20. Moore K, Dalley A. Essential clinical anatomy. Philadelphia: Lippincott Williams and Wilkins; 1999.
21. Windhofer C, Brenner E, Moriggl B, Papp C. Relationship between the descending branch of the inferior gluteal artery and the posterior femoral cutaneous nerve applicable to flap surgery. Surg Radiol Anat. 2002;24:253–7.
22. Kalhor M, Beck M, Huff TW, Ganz R. Capsular and pericapsular contributions to acetabular and femoral head perfusion. J Bone Joint Surg Am. 2009;91:409–18.
23. Gautier E, Ganz K, Krugel N, Gill T, Ganz R. Anatomy of the medial femoral circumflex artery and its surgical implications. J Bone Joint Surg Br. 2000;82:679–83.
24. Sunderland S, Hughes ES. Metrical and non-metrical features of the muscular branches of the sciatic nerve and its medial and lateral popliteal divisions. J Comp Neurol. 1946;85:205–22.
25. Sunderland S, Ray LJ. The intraneural topography of the sciatic nerve and its popliteal divisions in man. Brain. 1948;71:242–73.
26. Seidel PM, Seidel GK, Gans BM, Dijkers M. Precise localization of the motor nerve branches to the hamstring muscles: an aid to the conduct of neurolytic procedures. Arch Phys Med Rehabil. 1996;77:1157–60.
27. Moayeri N, Groen GJ. Differences in quantitative architecture of sciatic nerve may explain differences in potential vulnerability to nerve injury, onset time, and minimum effective anesthetic volume. Anesthesiology. 2009;111:1128–34.
28. Sladjana UZ, Ivan JD, Bratislav SD. Microanatomical structure of the human sciatic nerve. Surg Radiol Anat. 2008;30:619–26.
29. Georgakis E, Soames R. Arterial supply to the sciatic nerve in the gluteal region. Clin Anat. 2008;21:62–5.
30. Karmanska W, Mikusek J, Karmanski A. Nutrient arteries of the human sciatic nerve. Folia Morphol (Warsz). 1993;52:209–15.
31. Ugrenovic SZ, Jovanovic ID, Vasovic LP, Stefanovic BD. Extraneural arterial blood vessels of human fetal sciatic nerve. Cells Tissues Organs. 2007;186:147–53.
32. Del Pinal F, Taylor GI. The venous drainage of nerves; anatomical study and clinical implications. Br J Plast Surg. 1990;43:511–20.
33. Testut L, Latarjet A. Traite d'anatomie humaine. Paris: G. Doin & Cie; 1949.
34. Possover M, Schneider T, Henle KP. Laparoscopic therapy for endometriosis and vascular entrapment of sacral plexus. Fertil Steril. 2011;95:756–8.
35. Beaton L, Anson B. The sciatic nerve and the piriformis muscle: their interrelation and possible cause of coccygodynia. J Bone Joint Surg Am. 1938;20:686–8.
36. Smoll NR. Variations of the piriformis and sciatic nerve with clinical consequence: a review. Clin Anat. 2010;23:8–17.

37. Coppieters MW, Alshami AM, Babri AS, Souvlis T, Kippers V, Hodges PW. Strain and excursion of the sciatic, tibial, and plantar nerves during a modified straight leg raising test. J Orthop Res. 2006;24:1883–9.
38. Fleming P, Lenehan B, O'Rourke S, McHugh P, Kaar K, McCabe JP. Strain on the human sciatic nerve in vivo during movement of the hip and knee. J Bone Joint Surg Br. 2003;85:363–5.
39. Wall EJ, Massie JB, Kwan MK, Rydevik BL, Myers RR, Garfin SR. Experimental stretch neuropathy. Changes in nerve conduction under tension. J Bone Joint Surg Br. 1992;74:126–9.
40. Guvencer M, Akyer P, Iyem C, Tetik S, Naderi S. Anatomic considerations and the relationship between the piriformis muscle and the sciatic nerve. Surg Radiol Anat. 2008;30:467–74.
41. Yeoman W. The relation of arthritis of the sacro-iliac joint to sciatica, with an analysis of 100 cases. Lancet. 1928;2:1119–22.
42. Robinson DR. Pyriformis syndrome in relation to sciatic pain. Am J Surg. 1947;73:355–8.
43. Adams JA. The pyriformis syndrome – report of four cases and review of the literature. S Afr J Surg. 1980;18:13–8.
44. Benson ER, Schutzer SF. Posttraumatic piriformis syndrome: diagnosis and results of operative treatment. J Bone Joint Surg Am. 1999;81:941–9.
45. Dezawa A, Kusano S, Miki H. Arthroscopic release of the piriformis muscle under local anesthesia for piriformis syndrome. Arthroscopy. 2003;19:554–7.
46. Filler AG, Haynes J, Jordan SE, Prager J, Villablanca JP, Farahani K, McBride DQ, Tsuruda JS, Morisoli B, Batzdorf U, Johnson JP. Sciatica of nondisc origin and piriformis syndrome: diagnosis by magnetic resonance neurography and interventional magnetic resonance imaging with outcome study of resulting treatment. J Neurosurg Spine. 2005;2:99–115.
47. Hughes SS, Goldstein MN, Hicks DG, Pellegrini Jr VD. Extrapelvic compression of the sciatic nerve. An unusual cause of pain about the hip: report of five cases. J Bone Joint Surg Am. 1992;74:1553–9.
48. Mayrand N, Fortin J, Descarreaux M, Normand MC. Diagnosis and management of posttraumatic piriformis syndrome: a case study. J Manipulative Physiol Ther. 2006;29:486–91.
49. Sayson SC, Ducey JP, Maybrey JB, Wesley RL, Vermilion D. Sciatic entrapment neuropathy associated with an anomalous piriformis muscle. Pain. 1994;59:149–52.
50. Bano A, Karantanas A, Pasku D, Datseris G, Tzanakakis G, Katonis P. Persistent sciatica induced by quadratus femoris muscle tear and treated by surgical decompression: a case report. J Med Case Rep. 2010;4:236.
51. Brown JA, Braun MA, Namey TC. Pyriformis syndrome in a 10-year-old boy as a complication of operation with the patient in the sitting position. Neurosurgery. 1988;23:117–9.
52. Issack PS, Kreshak J, Klinger CE, Toro JB, Buly RL, Helfet DL. Sciatic nerve release following fracture or reconstructive surgery of the acetabulum. Surgical technique. J Bone Joint Surg Am. 2008; 90(Suppl 2 Pt 2):227–37.
53. Uchio Y, Nishikawa U, Ochi M, Shu N, Takata K. Bilateral piriformis syndrome after total hip arthroplasty. Arch Orthop Trauma Surg. 1998;117:177–9.
54. Filler AG. Piriformis and related entrapment syndromes: diagnosis & management. Neurosurg Clin N Am. 2008;19:609–22, vii.
55. Chakravarthy J, Ramisetty N, Pimpalnerkar A, Mohtadi N. Surgical repair of complete proximal hamstring tendon ruptures in water skiers and bull riders: a report of four cases and review of the literature. Br J Sports Med. 2005;39:569–72.
56. Orava S. Hamstring syndrome. Op Tech Sports Med. 1997;5:143–9.
57. Wood DG, Packham I, Trikha SP, Linklater J. Avulsion of the proximal hamstring origin. J Bone Joint Surg Am. 2008;90:2365–74.
58. Martin H. Clinical examination and imaging of the hip. In: Byrd J, Guanche C, editors. AANA advanced arthroscopy: the hip. Philadelphia: Saunders; 2010.
59. Martin HD, Kelly BT, Leunig M, Philippon MJ, Clohisy JC, Martin RL, Sekiya JK, Pietrobon R, Mohtadi NG, Sampson TG, Safran MR. The pattern and technique in the clinical evaluation of the adult hip: the common physical examination tests of hip specialists. Arthroscopy. 2010;26:161–72.
60. Papadopoulos EC, Khan SN. Piriformis syndrome and low back pain: a new classification and review of the literature. Orthop Clin North Am. 2004;35: 65–71.
61. Martin HD, Kivlan BR, Palmer IJ, Martin RL, Hatem M. Diagnostic accuracy of clinical tests for sciatic nerve entrapment in the gluteal region. Knee Surg Sports Traumatol Arthrosc. 2014;22:882–888.
62. Migliorini S, Merlo M. The hamstring syndrome in endurance athletes. Br J Sports Med. 2011;45:363.
63. Labat JJ, Riant T, Robert R, Amarenco G, Lefaucheur JP, Rigaud J. Diagnostic criteria for pudendal neuralgia by pudendal nerve entrapment (Nantes criteria). Neurourol Urodyn. 2008;27:306–10.
64. Fishman LM, Wilkins AN. Piriformis syndrome: electrophysiology vs. anatomical assumption. In: Fishman LM, Wilkins AN, editors. Functional electromyography. New York: Springer; 2011.
65. Jawish RM, Assoum HA, Khamis CF. Anatomical, clinical and electrical observations in piriformis syndrome. J Orthop Surg Res. 2010;5:3.
66. Katirji B. Electrodiagnostic approach to the patient with suspected mononeuropathy of the lower extremity. Neurol Clin. 2002;20:479–501, vii.
67. Petchprapa CN, Rosenberg ZS, Sconfienza LM, Cavalcanti CF, Vieira RL, Zember JS. MR imaging of entrapment neuropathies of the lower extremity. Part 1. The pelvis and hip. Radiographics. 2010;30: 983–1000.

68. Chappell KE, Robson MD, Stonebridge-Foster A, Glover A, Allsop JM, Williams AD, Herlihy AH, Moss J, Gishen P, Bydder GM. Magic angle effects in MR neurography. AJNR Am J Neuroradiol. 2004;25:431–40.
69. Kim SJ, Hong SH, Jun WS, Choi JY, Myung JS, Jacobson JA, Lee JW, Choi JA, Kang HS. MR imaging mapping of skeletal muscle denervation in entrapment and compressive neuropathies. Radiographics. 2011;31:319–32.
70. Barton PM. Piriformis syndrome: a rational approach to management. Pain. 1991;47:345–52.
71. Benzon HT, Katz JA, Benzon HA, Iqbal MS. Piriformis syndrome: anatomic considerations, a new injection technique, and a review of the literature. Anesthesiology. 2003;98:1442–8.
72. Pace JB, Nagle D. Piriform syndrome. West J Med. 1976;124:435–9.
73. Porta M. A comparative trial of botulinum toxin type A and methylprednisolone for the treatment of myofascial pain syndrome and pain from chronic muscle spasm. Pain. 2000;85:101–5.
74. Hanania M, Kitain E. Perisciatic injection of steroid for the treatment of sciatica due to piriformis syndrome. Reg Anesth Pain Med. 1998;23:223–8.
75. Torriani M, Souto SC, Thomas BJ, Ouellette H, Bredella MA. Ischiofemoral impingement syndrome: an entity with hip pain and abnormalities of the quadratus femoris muscle. AJR Am J Roentgenol. 2009;193:186–90.
76. Johnson KA. Impingement of the lesser trochanter on the ischial ramus after total hip arthroplasty. Report of three cases. J Bone Joint Surg Am. 1977;59:268–9.
77. Kassarjian A. Signal abnormalities in the quadratus femoris muscle: tear or impingement? Am J Roentgenol. 2008;190:W380–1.
78. Patti JW, Ouellette H, Bredella MA, Torriani M. Impingement of lesser trochanter on ischium as a potential cause for hip pain. Skeletal Radiol. 2008;37:939–41.
79. Ali AM, Whitwell D, Ostlere SJ. Case report: imaging and surgical treatment of a snapping hip due to ischiofemoral impingement. Skeletal Radiol. 2011;40:653–6.
80. Truong W, Murnaghan L, Hopyan S, Kelley S. Ischioplasty for femoroischial impingement. J Bone Joint Surg Am Case Connect. 2012;2:2–6.
81. Martin HD, Palmer IJ, Hatem M. Diagnosis and 2-year outcomes of endoscopic treatment for ischiofemoral impingement. Arthroscopy 2014. (Accepted in production).
82. Scillia A, Choo A, Milman E, McInerney V, Festa A. Snapping of the proximal hamstring origin: a rare cause of coxa saltans: a case report. J Bone Joint Surg Am. 2011;93:e1251–3.
83. Lempainen L, Sarimo J, Mattila K, Vaittinen S, Orava S. Proximal hamstring tendinopathy: results of surgical management and histopathologic findings. Am J Sports Med. 2009;37:727–34.
84. Kim SM, Shin MJ, Kim KS, Ahn JM, Cho KH, Chang JS, Lee SH, Chhem RK. Imaging features of ischial bursitis with an emphasis on ultrasonography. Skeletal Radiol. 2002;31:631–6.
85. Popeney C, Ansell V, Renney K. Pudendal entrapment as an etiology of chronic perineal pain: diagnosis and treatment. Neurourol Urodyn. 2007;26:820–7.
86. Hough DM, Wittenberg KH, Pawlina W, Maus TP, King BF, Vrtiska TJ, Farrell MA, Antolak Jr SJ. Chronic perineal pain caused by pudendal nerve entrapment: anatomy and CT-guided perineural injection technique. AJR Am J Roentgenol. 2003;181:561–7.
87. Vancaillie T, Eggermont J, Armstrong G, Jarvis S, Liu J, Beg N. Response to pudendal nerve block in women with pudendal neuralgia. Pain Med. 2012;13:596–603.
88. Hibner M, Desai N, Robertson LJ, Nour M. Pudendal neuralgia. J Minim Invasive Gynecol. 2010;17:148–53.
89. Robert R, Labat JJ, Bensignor M, Glemain P, Deschamps C, Raoul S, Hamel O. Decompression and transposition of the pudendal nerve in pudendal neuralgia: a randomized controlled trial and long-term evaluation. Eur Urol. 2005;47:403–8.
90. Vilos GA, Vilos AW, Haebe JJ. Laparoscopic findings, management, histopathology, and outcome of 25 women with cyclic leg pain. J Am Assoc Gynecol Laparosc. 2002;9:145–51.
91. Parsons FG, Keith A. Sixth annual report of the committee of collective investigation of the anatomical society of Great Britain and Ireland, 1895–96. J Anat Physiol. 1896;31(Pt 1):31–44.
92. Bardeen K. A statistical study of the variations in the formation and position of the lumbosacral plexus in man. Anat Anz. 1901;9:209–38.
93. Trotter M. The relation of the sciatic nerve to the piriformis muscle in American Whites and Negroes. Anat Rec. 1932;52:321–3.
94. Beaton L, Anson B. The relation of the sciatic nerve and of its subdivisions to the piriformis muscle. Anat Rec. 1937;70:1–5.
95. Ming-Tzu P. The relation of the sciatic nerve to the piriformis muscle in the Chinese. Am J Phys Anthropol. 1941;28:375.
96. Misra B. The relations of the sciatic nerve to the piriformis in Indian cadavers. J Anat Soc India. 1954;3:28–33.
97. Anson B, McVay C. Surgical anatomy. Philadelphia: W.B. Saunders; 1971.
98. Nizankowski C, Slociak J, Szybejko J. Variations in the anatomy of the sciatic nerve in man. Folia Morphol (Warsz). 1972;31(4):507–13.
99. Lee CS, Tsai TL. The relation of the sciatic nerve to the piriformis muscle. Taiwan Yi Xue Hui Za Zhi. 1974;73(2):75–80.
100. Pecina M. Contribution to the etiological explanation of the piriformis syndrome. Acta Anat (Basel). 1979;105(2):181–7.

101. Chiba S. Multiple positional relationships of nerves arising from the sacral plexus to the piriformis muscle in humans. Kaibogaku Zasshi. 1992;67(6): 691–724.
102. Chiba S, Ishibashi Y, Kasai T. Perforation of dorsal branches of the sacral nerve plexus through the piriformis muscle and its relation to changes of segmental arrangements of the vertebral column and others. Kaibogaku Zasshi. 1994;69(3):281–305.
103. Pokorny D, Sosna A, Veigl P, et al. Anatomic variability of the relation of pelvitrochanteric muscles and sciatic nerve. Acta Chir Orthop Traumatol Cech. 1998;65(6):336–9.
104. Fishman LM, Dombi GW, Michaelsen C, et al. Piriformis syndrome: diagnosis, treatment, and outcome – a 10-year study. Arch Phys Med Rehabil. 2002;83(3):295–301.
105. Agur A, Dalley A. Grant's atlas of anatomy. Baltimore: Lippincott Williams & Wilkins; 2005.
106. Ugrenovic S, Jovanovic I, Krstic V, et al. The level of the sciatic nerve division and its relations to the piriform muscle. Vojnosanit Pregl. 2005;62(1):45–9.
107. Pokorny D, Jahoda D, Veigl D, et al. Topographic variations of the relationship of the sciatic nerve and the piriformis muscle and its relevance to palsy after total hip arthroplasty. Surg Radiol Anat. 2006;28(1): 88–91.
108. Guvencer M, Iyem C, Akyer P, et al. Variations in the high division of the sciatic nerve and relationship between the sciatic nerve and the piriformis. Turk Neurosurg. 2009;19(2):139–44.
109. Labropoulos N, Tassiopoulos AK, Gasparis AP, et al. Veins along the course of the sciatic nerve. J Vasc Surg. 2009;49(3):690–6.

72 Surgical Technique: Open Proximal Hamstring Repair

Joshua D. Harris, Shane J. Nho, and Charles A. Bush-Joseph

Contents

Abstract

The proximal hamstring is a common location for athletic injuries. A complete, three-tendon (semitendinosus, semimembranosus, long head biceps femoris) tear may occur in sports that involve eccentric contractions of the hamstrings with a rapid hip flexion and knee extension. Surgical treatment of multi-tendon tears with retraction in young active patients has demonstrated significantly better subjective and objective outcomes at short- and mid-term follow-up. A safe surgical approach is via the prone positioning, transverse or longitudinal skin incision, avoidance of the posterior femoral cutaneous nerve, exposure of the ischial tuberosity and proximal hamstring anatomic footprint, avoidance of the sciatic nerve (lateral to tuberosity), secure fixation via two or three double-loaded suture anchors, and meticulous hemostasis and closure. Chronic repair often requires extensive adhesiolysis, sciatic neurolysis, tendon mobilization, and possible allograft augmentation. Postoperative rehabilitation should avoid undue stress on the repair via avoiding hip flexion and knee extension.

Introduction

The proximal hamstring is a common location for injuries in athletes. The spectrum of injury at this location ranges from mild muscle strain to

J.D. Harris (✉)
Houston Methodist Center for Orthopaedics & Sports Medicine, Houston, TX, USA

Rush University Medical Center, Chicago, IL, USA
e-mail: joshuaharrismd@gmail.com

S.J. Nho
Department of Orthopaedic Surgery, Rush University Medical Center, Chicago, IL, USA

Midwest Orthopaedics at Rush, Hip Preservation Center, Rush University Medical Center, Chicago, IL, USA
e-mail: shane.nho@rushortho.com

C.A. Bush-Joseph
Rush University Medical Center, Chicago, IL, USA
e-mail: cbj@rushortho.com

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_78

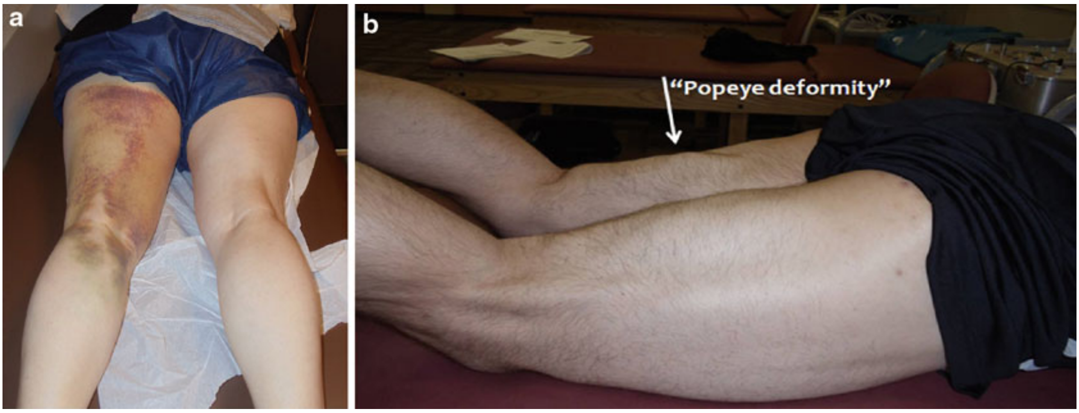


Fig. 1 (**a**) Prone position of a patient with a complete proximal hamstring rupture of the left lower extremity. (**b**) Prone position of a patient with a complete proximal hamstring rupture of the left lower extremity demonstrating a "popeye deformity"

complete three-tendon ruptures with retraction (Fig. 1a, b). The most common predictors of injury include prior hamstring injury [1], age [2], body mass index [3], decreased hip flexor and hamstring flexibility [4], and participation in sports requiring sprinting, jumping, accelerating, and decelerating [1]. Injury mechanism is usually an eccentric load with a flexed hip and extended knee. Complete ruptures are reported to be most common after waterskiing injuries [3]. In comparison to nonoperative management, recent evidence has reported significantly better subjective clinical outcomes, greater rate of return to pre-injury level of sport, and greater strength and endurance with surgical treatment of complete tendon tears [3, 5, 6]. The rate of persistent disability after complete proximal hamstring ruptures treated nonsurgically, however, remains unclear [7]. Furthermore, acute surgery (within 4 weeks of injury) had significantly better patient satisfaction, subjective clinical outcomes, pain relief, strength and endurance, and rate of return to pre-injury level of sport than chronic repair (more than 4 weeks from date of injury) [3]. In addition, acute repair had a significantly lower rate of complications and re-ruptures. Recent magnetic resonance imaging (MRI) investigations have reported that 100 % of subjects' repairs were still attached and in continuity at 36 months following surgery [8].

Indications for Surgery

Surgical management of proximal hamstring ruptures is indicated for two- or three-tendon ruptures with greater than 1–2 cm of retraction in active, healthy individuals. With only one tendon involved or even multiple tendon involvement without significant retraction, nonoperative management may be highly successful [7]. For patients with significant comorbidities, or without a desire to return to athletic activities, nonsurgical treatment is considered. Although less successful and predictable, chronic repair or reconstruction may also be warranted secondary to pain, weakness, and loss of function after failed conservative treatment or delay in diagnosis [9, 10]. In the chronic repair or reconstruction setting, there is a possibility for increased surgical difficulty due to adhesions around the sciatic nerve, preventing sufficient tendon mobilization and warranting reconstruction with allograft [11].

Surgical Anatomy

The hamstring musculature is composed of the semimembranosus, semitendinosus, and long and short heads of the biceps femoris. The "anatomic footprint" of the proximal hamstring origin has a common semitendinosus/long head of the biceps femoris origin and separate semimembranosus origin. The semitendinosus and biceps femoris long head tendons originate on an oval-shaped, more superficial and medial area that is longer than wide (2.8 cm proximal to distal; 1.7 cm medial to lateral) [12]. The semimembranosus origin is crescent shaped, deeper, and lateral to the semitendinosus/long head of the biceps origin (3.1 cm proximal to distal; 1.1 cm medial to lateral) [12]. The mean distance from the most proximal aspect of the semitendinosus and biceps origin to the inferior margin of gluteus maximus is 6.3 cm [12]. The mean distance from the inferior gluteal nerve and artery to the inferior margin of gluteus maximus is 5.0 cm [12]. The mean distance from the sciatic nerve (lateral to the tendinous origin, entering surgical field under piriformis) to the lateral-most margin of the ischial tuberosity is 1.2 cm [12]. The posterior femoral cutaneous nerve also enters the surgical field from under the piriformis but then travels deep to the gluteus maximus and then deep to the gluteal fascia and fascia lata down the posterior thigh.

Surgical Technique

General endotracheal anesthesia with muscle relaxation is commenced, and the patient is then positioned prone on a well-padded operative table (Fig. 2a). The table is then broken at the pelvis to allow for hip flexion and forward tilting of the pelvis to rotate the ischial tuberosity into the field more clearly (Fig. 2b). Alternatively two large rolls can be used with one placed under the chest and the other under the anterior pelvis. A limb support device is necessary for sterile limb preparation and draping (Fig. 2c). Either a longitudinal (proximal to distal) or transverse (medial to lateral) incision may be used. A transverse incision in the gluteal fold/crease often provides sufficient exposure and cosmesis (Fig. 3). Even a significantly retracted (up to 5–7 cm) tendon may be adequately visualized and mobilized because of excellent skin mobility in this area. Subdermal and subcutaneous infiltration of local anesthetic can be performed prior to skin incision. The skin incision is made and sharp dissection carried down through subcutaneous tissue to identify the gluteal fascia. A transverse incision is made in the fascia with care taken to avoid the posterior femoral cutaneous nerve and its branches. A blunt retractor is placed at the inferior margin of gluteus maximus and retracted superiorly to expose the hamstring fascia. One should avoid over-retraction of the gluteus maximus to avoid inferior gluteal nerve injury. The deep hamstring fascia is incised longitudinally and a large hematoma or seroma depending on timing of surgery is often expressed and evacuated. Avoid lateral placement of this fascial exposure as the sciatic nerve is lateral to the tendinous origin. The ischium should now be visible, but deep in the surgical field (a headlamp can be used for optimal visualization). Given that the most proximal aspect of the origin is 6.3 cm from the inferior margin of the unretracted gluteus maximus, one must retract the gluteus maximus in order to provide adequate exposure and allow for anatomic reattachment of the hamstrings.

After adequate deep exposure, the retracted tendon is identified and grasped with an Allis clamp. Tagging stay sutures placed in the proximal tendon edge can aid in exposure (Fig. 4). Mobility is ensured by advancement to its anatomic footprint. Adhesiolysis is carefully performed and can be assisted with Loupe and/or microscopic magnification as needed to avoid sciatic nerve injury. The sciatic nerve should be repeatedly visualized or palpated throughout the case to avoid iatrogenic injury. The ischial footprint is next prepared with a rongeur to a light bleeding surface (Fig. 5). Double-loaded suture (nonabsorbable) anchors are then placed (usually two or three depending on patient's anatomy and tear pattern) (Fig. 6). The surgeon should assure that the most lateral anchor is proximo-lateral enough to re-create the anatomy of the footprint. Only one limb of each suture is placed in a Krackow or locking pattern to allow for a "pulley technique." Mark the post limb of all sutures and pull the post limbs to reduce the torn proximal

Fig. 2 (**a**) Operating room table prepared to pad the right and left chest walls and the right and left knees and a padded roll to elevate the ankles and flex the knees. (**b**) Break in the operating room table at the pelvis to allow the hips to flex and rotate the pelvis to bring the ischial tuberosity into clearer view in the operative field. (**c**) Lower extremity limb supporter "candy cane" to allow for sterile preparation and draping of the leg

Fig. 3 Transverse skin incision at gluteal fold/crease marked for right hip in prone position

Fig. 4 Tagging suture and clamp on most proximal end of torn proximal hamstring tendon. After adhesiolysis, excellent excursion and tendon mobility is achieved

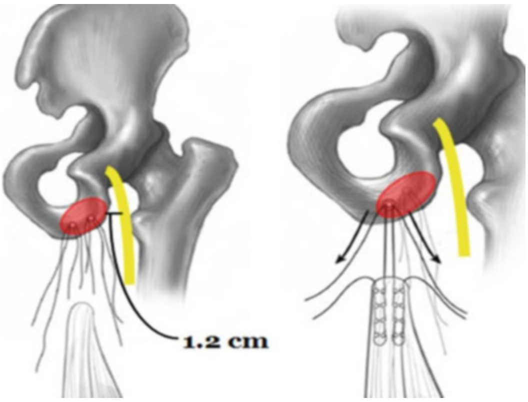


Fig. 5 Posterior view of pelvis and proximal hamstring footprint (*red*). Semitendinosus and long head biceps origin is more medial and distal relative to the semimembranosus, which is more lateral and proximal. The sciatic nerve (*yellow*) is 1.2 cm lateral to the most lateral edge of the ischial tuberosity

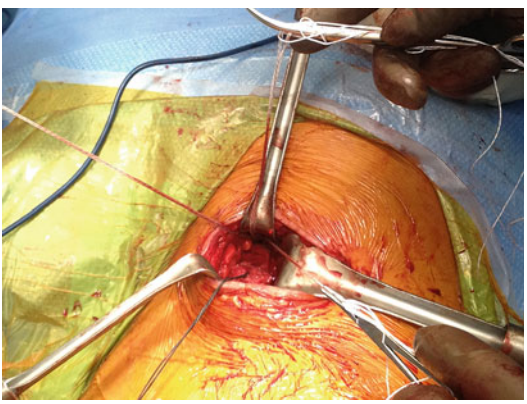


Fig. 6 Three double-loaded suture anchors with nonabsorbable suture placed in ischial tuberosity

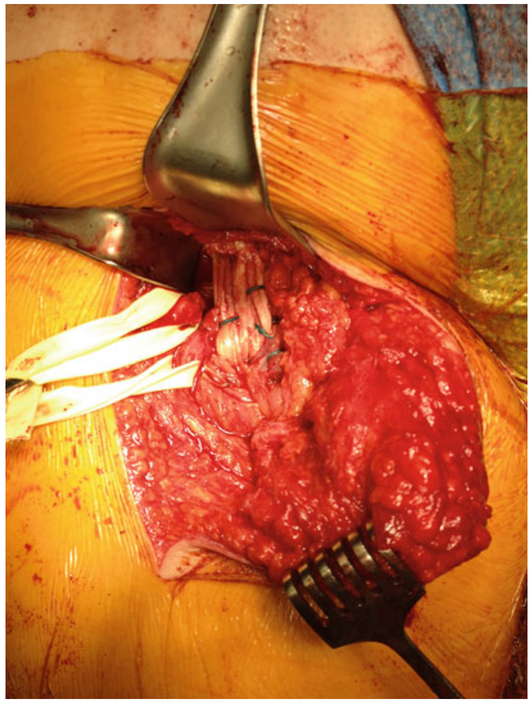


Fig. 7 Achilles allograft augmentation of a chronic proximal hamstring repair. After extensive sciatic neurolysis (Penrose drain around nerve), the native tendon was unable to be fully mobilized to the ischial tuberosity, thus requiring allograft augmentation

tendon to the anatomic footprint (Fig. 5). When final knot tying is performed, place the hip in neutral extension and the knee flexed to 90° to provide minimal tension on the repair, if necessary. Knots are securely tied. Medial knot placement precludes contact with the sciatic nerve (lateral).

In the setting of a chronic retracted injury, a longitudinal skin incision may be advantageous to facilitate exposure, adhesiolysis, and sciatic neurolysis [9, 11, 13]. Tendon and osseous preparation is accomplished in a similar fashion as described above for an acute repair. If the tendon is irreparable secondary to significant retraction and scarring, an allograft may be necessary to augment or reconstruct the proximal hamstring complex (Fig. 7) [14]. An Achilles tendon allograft has a similar thickness to that of the proximal hamstring and has adequate length to reattach even significantly residually retracted tendons. In cases of extensive adhesions to the sciatic nerve, a neurolysis may be required to dissect the hamstring away from the sciatic nerve in order to avoid undue tension on the nerve and improve mobility of the proximal hamstrings.

After the tendon is satisfactorily reattached, the wound is copiously irrigated. Hemostasis must be complete to avoid postoperative hematoma and sciatic nerve compression. The deep fascia and gluteal fascia/fascia lata can be closed with absorbable suture. The subdermal and subcuticular layers are closed in routine fashion. A sterile dressing is applied. A hip or knee orthosis may be applied to prevent undue stress on the repair.

Postoperative Rehabilitation

Patients ambulate with crutches and a brace can be used for 4–6 weeks following surgery and is based on the quality and tension of the repair noted at the time of surgery. Positions of repair tension are to be avoided (hip flexion and knee extension) for 4–6 weeks following surgery. If a knee orthosis is used, an extension stop is used and is gradually brought to full extension over 4–6 weeks. Flexion can be left open to allow for ease of transition between sitting and standing. Active hamstring motion is avoided. Advances in hip and knee motion and weight-bearing begin at 4–6 weeks after surgery. Very light hamstring concentric motion may also begin at 6 weeks. Core and pelvic strengthening may also ensue at this time. Concentric strengthening is advanced after 8 weeks and eccentric strengthening may be initiated at 3 months postoperatively. Light jogging, light short sprints, and closed-chain plyometrics also begin between 3 and 6 months postoperatively. Sport-specific drills and activities begin at 4–6 months after surgery, with return to competitive sports after 6 months.

Summary

Proximal hamstring injuries are common in sports and recreation. However, complete proximal hamstring ruptures are reported to be uncommon. Significantly retracted multi-tendon tears in young active patients may be successfully treated with surgical repair. Surgical treatment results in predictable improvements in pain, strength, endurance, function, and return to sport. A safe and reproducible surgical approach and technique for acute proximal hamstring ruptures with postoperative rehabilitation has been presented.

References

1. Engebretsen A, Myklebust G, Holme I, Engebretsen L, Bahr R. Intrinsic risk factors for hamstring injuries among male soccer players. Engebretsen is Am J Sports Med 2010;38(6):1147–53.
2. Arnason A, Sigurdsson S, Gudmundsson A, Holme I, Engebretsen L, Bahr R. Risk factors for injuries in football. Am J Sports Med. 2004;32(Suppl):5S–16.
3. Harris JD, Griesser MJ, Best TM, Ellis TJ. Treatment of proximal hamstring ruptures – a systematic review. Int J Sports Med. 2011;32(7):490–5.
4. Witvrouw E, Danneels L, Asselman P, D'Have T, Cambier D. Muscle flexibility as a risk factor for developing muscle injuries in male professional soccer players: a prospective study. Am J Sports Med. 2003;31:41–6.
5. Klingele K, Sallay P. Surgical repair of complete proximal hamstring tendon ruptures. Am J Sports Med. 2002;30:742–7.
6. Wood D, Packham I, Trikha S, Linklater J. Avulsion of the proximal hamstring origin. J Bone Joint Surg Am. 2008;90:2365–74.
7. Sallay PI, Friedman RL, Coogan PG, Garrett WE. Hamstring muscle injuries among water skiers. Functional outcome and prevention. Am J Sports Med. 1996;24(2):130–6.
8. Chahal J, Bush-Joseph CA, Chow A, Zelazny A, Mather RC, Lin E, et al. Clinical and magnetic resonance imaging outcomes after surgical repair of complete proximal hamstring ruptures: does the tendon heal? Am J Sports Med. 2012;40:2325–30.
9. Folsom GJ, Larson CM. Surgical treatment of acute versus chronic complete proximal hamstring ruptures: results of a new allograft technique for chronic reconstructions. Am J Sports Med. 2008;36(1):104–9.
10. Gidwani S, Bircher M. Avulsion injuries of the hamstring origin – a series of 12 patients and management algorithm. Ann R Coll Surg Engl. 2007;89:394–9.
11. Konan S, Haddad F. Successful return to high level sports following early surgical repair of complete tears of the proximal hamstring tendons. Int Orthop. 2010;34:119–23.
12. Miller SL, Gill J, Webb GR. The proximal origin of the hamstrings and surrounding anatomy encountered during repair. A cadaveric study. J Bone Joint Surg Am. 2007;89(1):44–8.
13. Sarimo J, Lempainen L, Mattila K, Orava S. Complete proximal hamstring avulsions: a series of 41 patients with operative treatment. Am J Sports Med. 2008;36:1110–5.
14. Folsom G, Larson C. Surgical treatment of acute versus chronic complete proximal hamstring ruptures- results of a new allograft technique for chronic reconstructions. Am J Sports Med. 2007;36:104–9.

Surgical Technique: Allograft Augmentation of Chronic Hamstring Repair

73

Christopher M. Larson, Geoffrey D. Cornelsen, and Rebecca M. Stone

Contents

Abstract

Proximal hamstring avulsion injuries are more commonly being recognized and can cause significant disability for the active patient. Patients frequently experience a loss of leg control and difficulties decelerating the leg, making athletic activities difficult. Unfortunately, patients may present late due to either misdiagnosis or failed attempts at conservative treatment. Retraction and scarring of the hamstring tendons can limit the surgeon's ability to repair the tendon to the ischial tuberosity without excessive tension. Use of an Achilles tendon allograft, can obviate the need for distal fractional lengthening or tenodesis and provides improved outcomes for activities of daily living and sports-related activities.

This chapter provides key history and exam findings to aid in diagnosis and describes the surgical procedure for allograft reconstruction for irreparable ruptures. Tendon fixation is described through use of either suture anchors or bone plug and interference screw depending on patient characteristics and intraoperative visualization. Postoperative protocols including hinged knee bracing are outlined. Most patients return to sport by 6 months although will improve for a year following the surgery. Posterior thigh numbness and superficial skin infection are the most common adverse events. Return to sports is inferior to acute

C.M. Larson (✉) • G.D. Cornelsen • R.M. Stone
Minnesota Orthopedic Sports Medicine Institute, Twin Cities Orthopedics, Edina, MN, USA
e-mail: chrislarson@tcomn.com; gcornel1@fairview.org; beckystone@tcomn.com

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_79

repair, but return to activities of daily living is comparable to acute repairs with universally high satisfaction rates after allograft reconstruction.

Introduction

There are an increasing number of studies regarding the treatment of proximal hamstring ruptures. While hamstring injuries are common sporting injuries, proximal hamstring avulsion occurs much less frequently. Avulsions of the proximal hamstring can present late due to misdiagnosis and can pose a challenging treatment dilemma if disability persists.

Due to their biarticular nature, the hamstring muscles are susceptible to eccentric forces, especially when the patient is in a position of hip flexion and knee extension placing the hamstring musculotendinous unit on maximal stretch [1]. The most frequently reported cause of proximal hamstring avulsion is a waterskiing injury although other causes have been reported including falls, powerlifting, badminton, judo, bull riding, and other athletic activities [2–5].

Surgical treatment for proximal hamstring avulsion was first described by Ishikawa in 1988 [4]. Since that time, a number of articles have been published on the subject [1–21]. However, fewer articles have addressed repair of chronic rupture [3, 5–15] and, even fewer, tendon reconstruction [8, 10, 11, 15–17].

Patient Evaluation

At the time of initial injury, patients will frequently feel and may even hear an audible "pop." Bruising and pain typically ensue in the buttock and posterior thigh. However, in the case of chronic ruptures, the history will be remote, and the examination much less dramatic. The injury is typically followed by a period of varying disability. While patients will improve somewhat over the following months, it is unclear how many patients will regain satisfactory function regarding activities of daily living and sports. Those with persistent disability typically have difficulties with regard to leg control and in particular for sports-related activities. There is typically a lack of control through hamstring eccentric phase of gait, and they may feel weakness or cramping. Patients can also experience radicular symptoms if the sciatic nerve is tethered. These symptoms can begin at the time of injury or develop over time.

On physical examination, patients often have a visible or palpable retracted proximal hamstring stump at variable points along the thigh with a proximal defect. They typically have decreased hamstring tension to palpation with passive hip flexion and knee extension in the supine position on the side of the rupture. The tendon will feel less taut than the contralateral side in particular for the medial hamstrings at the level of the posterior knee. Weakness is best demonstrated with resisted knee flexion while the patient is in the prone position and knees flexed to 90°. MRI with surface coil should be ordered for confirmation of prior rupture, degree of retraction, and proximity and potential scarring of the adjacent sciatic nerve (Fig. 1).

Operative Indications

Indications for repair of chronic proximal hamstring avulsions include two- or three-tendon complete avulsions from the ischial tuberosity or incomplete avulsion that fails conservative treatment with associated poor leg control, recurrent spasm and cramping, and sciatic nerve symptoms [10]. In the chronic setting, the hamstring muscles are retracted distally, scarred in place, and often cannot be mobilized sufficiently to obtain a primary repair. If this is the case, three options remain including tenodesis to the adjacent hamstring musculature, distal fractional lengthening and primary repair, and allograft reconstruction. Tenodesis is reported to result in unsatisfactory results [3]. Distal fractional lengthening produces better results but requires an additional incision and is technically demanding [6]. Reconstruction using Achilles tendon allograft has thus become the author's preferred method as this allows for

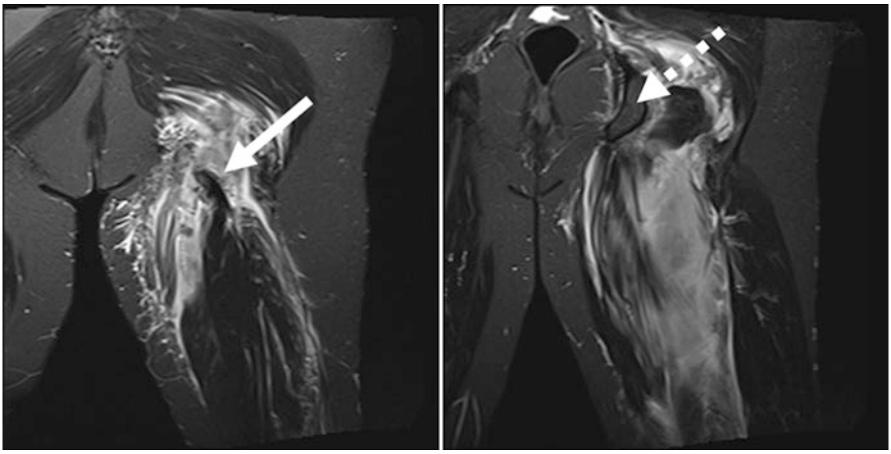


Fig. 1 Coronal MRI of a 45-year-old female with acute proximal hamstring avulsion (*solid arrow*) and 6 cm of distal retraction from the ischial tuberosity (*dashed arrow*) (Taken from Larson [10])

restoration of hamstring complex, crossing two joints, avoids the need for additional distal incisions and dissection without placing undue tension on the adjacent sciatic nerve.

Intraoperative Setup

The patient is positioned prone on the operating table. The use of a laminectomy frame eases positioning although it can be accomplished safely and conveniently with the use of large rolls, one under the chest and a second under the pelvis. Pillows or stacked blankets are placed under the patient's tibiae and feet with the goal of approximately 20–30° of hip flexion and 50° of knee flexion. The operative leg should be prepped and draped free from the posterior pelvis to the foot. Keeping the foot uncovered will allow more accurate assessment of sciatic nerve function intraoperatively with the use of a nerve stimulator.

Surgical Technique

While acute ruptures can be performed through transverse incisions, for chronic ruptures, a longitudinal incision is preferred and allows for more predictable access to the retracted hamstring stump (Fig. 2). The degree of retraction is assessed by MRI and with resisted knee flexion

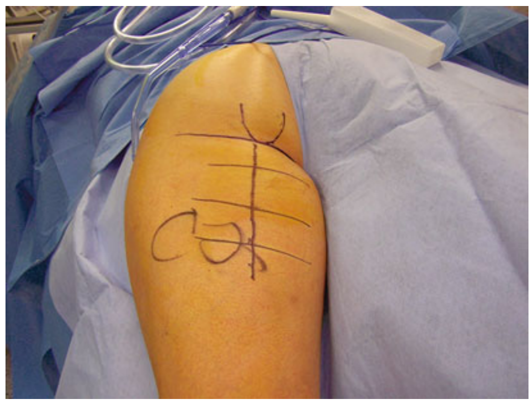


Fig. 2 The patient's left leg has been draped for surgery. The planned longitudinal incision has been drawn down the posterior thigh from the center of the ischial tuberosity, starting at the gluteal crease (Taken from Larson [10])

in the holding area and marked prior to the procedure. An incision is made from the gluteal crease to the level of the retracted hamstring tendons. The length of the incision will vary depending on retraction of the hamstring tendons. Sharp dissection is carried through the skin, subcutaneous tissue, and to the level of the hamstring fascia. The gluteus maximus is not incised but retracted proximally in order to identify and expose the ischial tuberosity. The sciatic nerve is identified and lies deep and lateral to the hamstring tendons followed by a formal neurolysis (Fig. 3). Locating the nerve distally in an area of more normal anatomy and/or

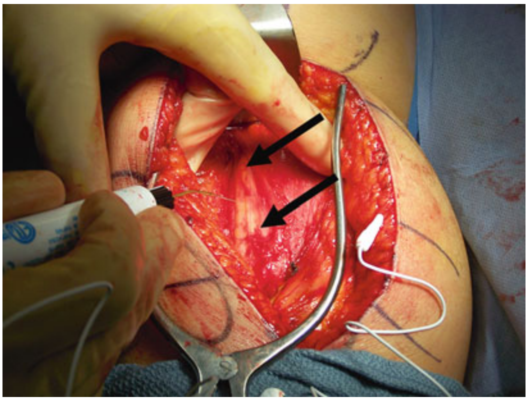


Fig. 3 After dissection is taken though the skin and subcutaneous tissue, a nerve stimulator is used in the sciatic nerve (identified by *arrows*) neurolysis (Taken from Larson [10])

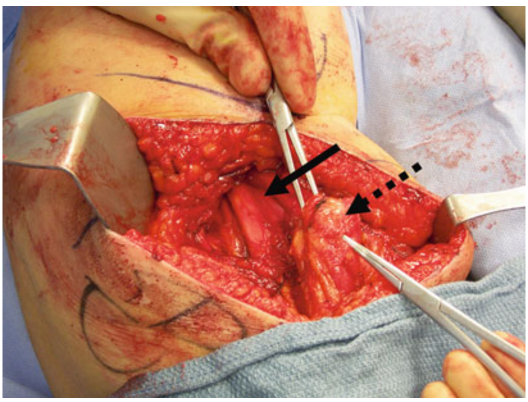


Fig. 4 The hamstring tendon (*dashed arrow*) is mobilized after the sciatic nerve identified (*solid arrow*) and neurolysis performed (Taken from Larson [10])

the use of a nerve stimulator to differentiate from surrounding scar tissue is recommended. The retracted hamstring tendon stump is then dissected free and mobilized. The ischial tuberosity is then identified and exposed to a bleeding bony bed (Fig. 4). It is important to recognize the anatomic origin of the proximal hamstring complex as it is fairly proximal on the ischial tuberosity.

Next, the method of proximal fixation must be made. If the patient is a small female with an ischial tuberosity that is not of sufficient size to accommodate a bone plug or if the patient has a large gluteus maximus muscle that limits proximal exposure, two to three large suture anchors

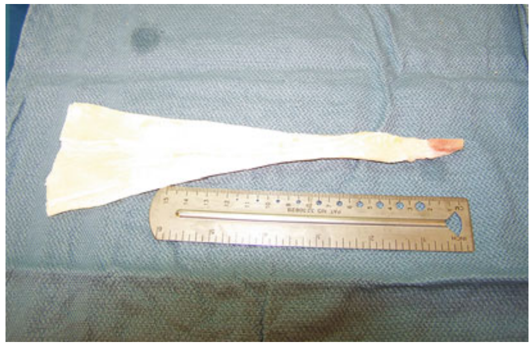


Fig. 5 The Achilles allograft prepared with bone plug for fixation with interference screw (Taken from Larson [10])

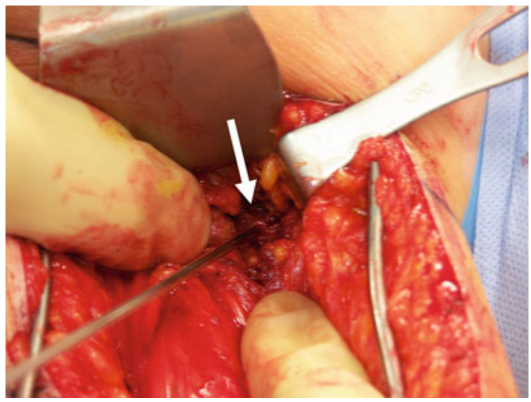


Fig. 6 Insertion of the guidewire into the prepared ischial tuberosity (*white arrow*) in preparation for reaming and graft insertion (Taken from Larson [10])

can be used for tendon reattachment. The anchors are placed in the anatomic footprint of the proximal hamstrings, lateral on the ischial tuberosity. The graft is then prepared and the Achilles bone block excised from the tendon (Fig. 5). One limb of each suture is placed in locking fashion through the graft. The second limb is used to secure the allograft to the ischial tuberosity.

If there is adequate bone and proximal exposure, an 8 × 20 mm bone plug is fashioned from the calcaneal allograft and fixed to the ischium with an interference screw. If this method is selected, a guidewire for an 8 mm reamer is drilled into the center of the hamstring footprint (Fig. 6). It is critical to verify that at least 20 mm of the wire is within the bone to ensure the entire bone plug and interference screw will seat and not be

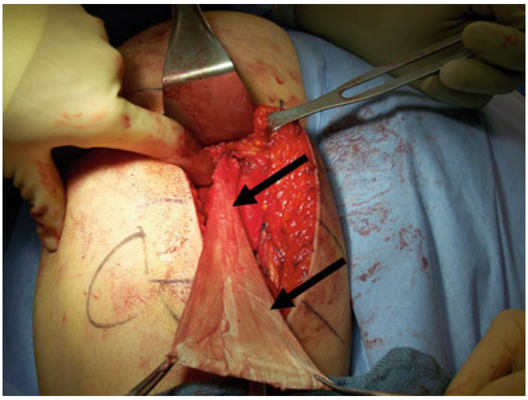


Fig. 7 The Achilles allograft (*solid arrows*) after fixation with metal interference screw (Taken from Larson [10])

prominent. Ream over the guide pin to the appropriate depth, insert the bone plug, and secure it into the tuberosity with a 7 or 8 × 20 mm interference screw depending on bone density.

While ensuring the hips are flexed 20–30° and the knees at least 45°, the graft is pulled distally and the hamstrings proximally. Under tension, the two are provisionally secured together with heavy absorbable suture. The tension is then evaluated. The newly reattached hamstrings should be taut in 45° of knee flexion but allow for knee extension to within 10° of full extension. If too much or too little tension is noted, simply repeat the previous step until appropriate tension is achieved. Finally, secure the graft to the tendon in a locking fashion with nonabsorbable sutures (Fig. 7). Check the sciatic nerve to be sure there has been an adequate neurolysis and there is minimal tension on the nerve with knee motion. The wound is then irrigated, hemostasis obtained, and the wound is closed in standard fashion (Fig. 8a, b).

Postoperative Management

A hinged knee brace is placed on the patient while prone on the operating table. The brace is worn at all times except showering with a 90° extension stop for the first 2 weeks, 60° for the next 2 weeks, and 30° for the final 2 weeks after which the brace is discontinued and crutches are weaned. If a drop-lock style brace is used, it allows free passive flexion for ease of sitting and standing. A hip orthosis is cumbersome for the patient and, with this protocol, has not been necessary in the senior author's (CML) experience. Physical therapy begins at 6–8 weeks postoperatively. Weeks 6–10 consist of range of motion activities, core strengthening, and stationary bike not using toe clips. At 10–12 weeks, resisted hamstring strengthening is begun. At 6 months, patients are typically allowed to return to sport; however, they will continue to improve for at least a year in terms of strength and function.

Complications

Posterior thigh/incisional numbness is the most common adverse event. Superficial wound problems requiring the use of oral antibiotics are not uncommon, but rarely require incision and drainage. Re-rupture has been reported after falls postoperatively but are rare [7, 11]. Complications after allograft reconstruction, however, are no greater than after repair of either acute or chronic injuries [11]. Transient sciatic nerve palsy has also been reported after hamstring surgery but has not been encountered by the senior author after hamstring reconstruction [7].

Outcomes

A number of authors have reported inferior outcomes with delayed repair of proximal hamstring ruptures [3, 7, 10, 12, 14, 18]. Allograft reconstruction of the proximal hamstrings, however, allows a predictable procedure for patients with chronic retracted proximal hamstring ruptures with regard to improved outcomes in the senior author's (CML) experience. Patient outcomes approach those seen after acute repair for activities of daily living with inferior but improved outcomes for sports-related activities [11]. In a recent study comparing outcomes of acute repair, chronic repair, and allograft reconstruction by the senior author (CML), patients were asked to rate their disability in sports participation and ADLs before and after surgery using Single Alpha

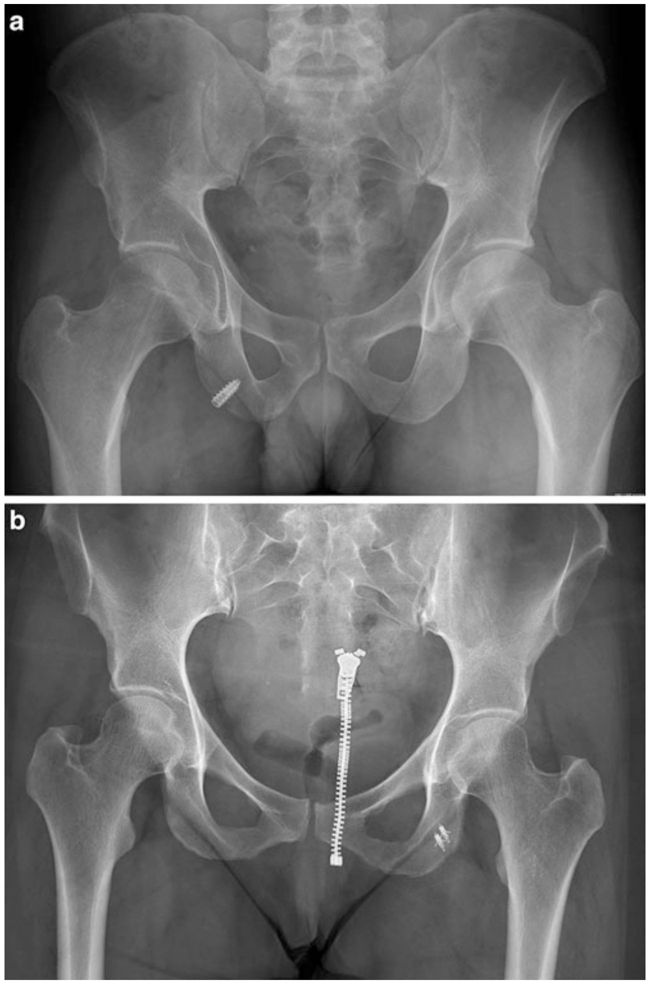


Fig. 8 (**a, b**) Postoperative anteroposterior pelvic radiographs after fixation either with bone plug and interference screw (**a**) or with suture anchors (**b**) (Published with permission from Am J Sports Med article (Rust et al. [11]))

Numeric Evaluation (SANE) scores, with 100 % being normal [11]. Patients with chronic tears had inferior results, scoring [70.2 % vs. 80.3 % ($p = 0.026$)] for sports participation and without statistically significant differences [86.5 % vs. 93.3 % ($p = 0.085$)] for ADLs compared with acute tears. When compared to their preoperative assessment, however, patients with chronic tears reported significant improvement in both sports activity score [30.3–70.2 % ($p = <0.01$)] and ADL score [56.1–86.5 % ($p = <0.01$)]. When comparing chronic injuries undergoing primary repair vs. allograft reconstruction, patients requiring allograft reported worse function preoperatively and had greater absolute improvements in their scores postoperatively [11]. Therefore, acute repair is preferred in particular for athletically active individuals; however, even patients with chronic injuries can be expected to improve significantly.

Isokinetic muscle strength testing and hamstring to quadriceps ratio in patients with allograft reconstruction has shown weakness compared to the contralateral side without a statistically difference to that seen after acute repairs in one clinical series [15, 17].

Summary

Chronic hamstring ruptures can lead to weakness and significant functional disability. These injuries can be identified through a detailed history and physical examination. An MRI confirms the diagnosis and aids in preoperative planning. Reconstruction with the use of Achilles allograft in chronic retracted irreparable injuries provides a reproducible technique and avoids the need for distal fractional lengthening or tenodesis. Patient outcomes to date have been favorable with the return of activities of daily living comparable to acute primary repair. Return to sports-related activities is also significantly improved after allograft reconstruction; however, these rates are still inferior to acute primary proximal hamstring repair.

References

1. Sallay PI, Friedman RL, Coogan PG, Garrett WE. Hamstring muscle injuries among water skiers. Functional outcome and prevention. Am J Sports Med. 1996;24(2):130–6.
2. Blasier RB, Morawa LG. Complete rupture of the hamstring origin from a water skiing injury. Am J Sports Med. 1990;18(4):435–7.
3. Orava S, Kujala UM. Rupture of the ischial origin of the hamstring muscles. Am J Sports Med. 1995; 23(6):702–5.
4. Ishikawa K, Kai K, Mizuta H. Avulsion of the hamstring muscles from the ischial tuberosity: a report of two cases. Clin Orthop. 1988;232:153–5.
5. Chakravarthy J, Ramisetty N, Pimpalnerkar A, Mohtadi N. Surgical repair of complete proximal hamstring tendon ruptures in water skiers and bull riders: a report of four cases and review of the literature. Br J Sports Med. 2005;39(8):569–72.
6. Klingele KE, Sallay PI. Surgical repair of complete proximal hamstring tendon rupture. Am J Sports Med. 2002;30(5):742–7.
7. Wood DG, Packham I, Trikha SP, Linklater J. Avulsion of the proximal hamstring origin. J Bone Joint Surg Am. 2008;90(11):2365–74.
8. Marx RG, Fives G, Chu SK, Daluiski A, Wolfe SW. Allograft reconstruction for symptomatic chronic complete proximal hamstring tendon avulsion. Knee Surg Sports Traumatol Arthrosc. 2009; 17(1):19–23.
9. Carmichael J, Packham I, Trikha SP, Wood DG. Avulsion of the proximal hamstring origin. Surgical technique. J Bone Joint Surg Am. 2009;91 Suppl 2:249–56.
10. Larson CM. Management of chronic proximal hamstring ruptures: surgical treatment. Op Tech Sports Med. 2009;17(4):210–4.
11. Rust DA, Giveans MR, Stone RM, Samuelson KM, Larson CM. Functional outcomes and return to sports after acute repair, chronic repair, and allograft reconstruction for proximal hamstring ruptures [published online ahead of print April 3, 2014]. Am J Sports Med. doi: 10.1177/0363546514528791.
12. Cohen SB, Rangavajjula A, Vyas D, Bradley JP. Functional results and outcomes after repair of proximal hamstring avulsions. Am J Sports Med. 2012;40(9):2092–8.
13. Brucker PU, Imhoff AB. Functional assessment after acute and chronic complete ruptures of the proximal hamstring tendons. Knee Surg Sports Traumatol Arthrosc. 2005;13(5):411–8.
14. Sarimo J, Lempainen L, Mattila K, Orava S. Complete proximal hamstring avulsions: a series of 41 patients with operative treatment. Am J Sports Med. 2008; 36(6):1110–5.
15. Folsom GJ, Larson CM. Surgical treatment of acute versus chronic complete proximal hamstring ruptures: results of a new allograft technique for chronic reconstructions. Am J Sports Med. 2008;36(1):104–9.
16. Kirkland A, Garrison JC, Singleton SB, Rodrigo J, Boettner F, Stuckey S. Surgical and therapeutic management of a complete proximal hamstring avulsion after failed conservative approach. J Orthop Sports Phys Ther. 2008;38(12):754–60.
17. Murray PJ, Lowe WR. Achilles allograft reconstruction of a chronic complete proximal hamstring rupture. Knee Surg Sports Traumatol Arthrosc. 2009;17(11): 1360–3.
18. Colosimo AJ, Wyatt HM, Frank KA, Mangine RE. Hamstring avulsion injuries. Op Tech Sports Med. 2005;13(1):80–8.
19. Domb BG, Linder D, Sharp KG, Sadik A, Gerhardt MB. Endoscopic repair of proximal hamstring avulsion. Arthrosc Tech. 2013;2(1):e35–9.
20. Skaara HE, Moksnes H, Frihagen F, Stuge B. Self-reported and performance-based functional outcomes after surgical repair of proximal hamstring avulsions. Am J Sports Med. 2013;41(11):2577–84.
21. Pombo M, Bradley JP. Proximal hamstring avulsion injuries: a technique note on surgical repairs. Sports Health. 2009;1(3):261–4.

Nerve Injuries Around the Hip

74

Monica Rho and Eziamaka Okafor

Contents

Abstract

Nerve injuries around the hip can have variable clinical presentations depending on the location of the lesion, the sensorimotor function of the nerve, and the severity of the injury. Nerve injury can be classified as neurapraxia, axonotmesis, and neurotmesis, which correlates to ascending levels of severity. History, physical examination, and electrodiagnostic studies aid in the diagnosis and the classification of neural injury. Basic knowledge of electrodiagnostic studies and its utility for each mononeuropathy is beneficial in determining location and severity of the injury, as well as a prognosis for recovery. The timing of when to order the electrodiagnostic study is critical to the proper interpretation of results. The etiology of the nerve injury often directs management. There are three principles of mononeuropathy treatment: facilitation of nerve healing, relief of symptoms, and restoration of function. Ultimately, recovery from a nerve injury occurs over time, and providing patients with realistic expectations for recovery is an important part of the management process. Each specific mononeuropathy of the hip has distinct clinical features. This chapter will cover the anatomic, etiologic, clinical presentation, and diagnostic features of the following nerves of the hip: iliohypogastric, ilioinguinal, genitofemoral, lateral femoral cutaneous, obturator, femoral, sciatic, superior gluteal, inferior gluteal, and pudendal.

M. Rho (✉) • E. Okafor
Harvard's Brigham and Women's Hospital, Boston, MA, USA
e-mail: mrho@ric.org; eokafor1@partners.org

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_80

Introduction

A complex circuitry of peripheral and spinal nerves exist within the lumbopelvic region. The location and course of each nerve may predispose it to damage by compression, stretch, transection, or ischemia. The function of the peripheral nerve can be disrupted by damage to any of its components: the cell body, axon, myelin sheath, connective tissue, or blood supply. According to the Seddon classification system, there are three different types of nerve injury: neurapraxia, axonotmesis, and neurotmesis [1]. Neurapraxia occurs when there is local myelin damage with preserved axon continuity. Axonotmesis describes discontinuity of the axons with variable preservation of the connective tissue elements of the nerve. Neurotmesis is the complete disruption of the nerve involving the myelin, axon, and the supporting neural structures.

The lumbosacral spinal nerves divide into dorsal and ventral rami which contain both motor and sensory fibers. The ventral rami of each lumbosacral spinal nerve will join together to form the lumbosacral plexus. All of the peripheral nerves of the lower extremity originate from the lumbosacral plexus, and each carries contributions from multiple spinal nerves. This particular organization enables a single spinal nerve to supply multiple muscles each innervated by a different peripheral nerve (Table 1). Similarly, sensory fibers from the same spinal nerve also branch into different peripheral nerves to supply different areas of cutaneous sensation. Given this high degree of overlap, a single spinal nerve lesion seldom results in complete sensory loss or complete muscle paralysis [2]. In contrast, a severe peripheral nerve lesion usually results in severe sensory and motor deficits because that sensory area or muscle is losing the collective input from all the spinal nerves to that specific location.

Injury to peripheral nerves can be broken down into two pathologic changes: damage to the myelin or damage to the axon (i.e., Wallerian degeneration). In demyelination, destruction of the myelin sheath occurs without axonal damage. Demyelinating injuries can slow electrical conduction over the entire length of the nerve, multiple segments of the nerve, a focal area of the nerve, or produce a conduction block (when focal demyelination is so severe that nerve action potential propagation across that segment does not occur) [3]. In Wallerian degeneration, the axon degenerates distally following transection or severe injury to the nerve. The time required for degeneration varies between sensory and motor segments and is related to the size and myelination of the fiber [4].

Mononeuropathy is characterized by injury to a single peripheral nerve. Nerve damage may be caused by vascular, muscular, or tumor compression; bony entrapment of the nerve; trauma (sharp, blunt, repetitive stress); toxins; metabolic syndromes (diabetes); vascular ischemia; and iatrogenic injury. The aim of this chapter is to provide a comprehensive overview of mononeuropathies around the hip with discussion of the evaluation of nerve injury, relevant anatomy, etiology, clinical presentation, and management. The mononeuropathies of the hip that will be discussed in this chapter include:

1. Iliohypogastric neuropathy
2. Ilioinguinal neuropathy
3. Genitofemoral neuropathy
4. Lateral femoral cutaneous neuropathy
5. Obturator neuropathy
6. Femoral neuropathy
7. Sciatic neuropathy
8. Superior gluteal neuropathy
9. Inferior gluteal neuropathy
10. Pudendal neuropathy

Evaluation of a Nerve Injury

The most important element in evaluating a peripheral nerve injury is precise knowledge of the course of the nerve, the terminal muscle innervations of the nerve, the spinal nerves that contribute to its motor branches, and the other muscles that these spinal nerves innervate. Knowledge of dermatomes, although important, often does not provide as clear of a distinguishing picture between peripheral nerve injury and

Table 1 Peripheral nerves arising from each spinal nerve

Lumbosacral Plexus	T12	L1	L2	L3	L4	L5	S1	S2	S3	S4
Anterior Division										
Obturator n.			■	■	■					
Iliohypogastric n.	■	■								
Ilioinguinal n.		■								
Genitofemoral n.		■	■							
Pudendal n.								■	■	■
Posterior Division										
Lateral Femoral Cutaneous n.			■	■						
Femoral n.			■	■	■					
Superior gluteal n.					■	■	■			
Inferior gluteal n.						■	■	■		
Anterior/Posterior Divisions										
Sciatic nerve					■	■	■	■	■	

radiculopathy, unless it is a purely sensory nerve. The knowledge of nerve anatomy and associated motor function will enhance the physical examination of the patient as well as the interpretation of the electrodiagnostic studies (Table 2). Most mononeuropathies can be preliminarily diagnosed by history and clinical exam; however, the gold standard for diagnosis of a mononeuropathy is electrodiagnostic (EDX) studies involving electromyography (EMG) and nerve conduction studies (NCS).

Physical Examination

The evaluation of a mononeuropathy is anchored on a through neurologic exam that includes manual muscle testing, sensation testing to light touch, and reflex testing. Recommended manual muscle testing for a mononeuropathy of the hip includes hip flexor, hip abductor, hip adductor, knee extensor, knee flexor, ankle dorsiflexor, ankle plantar flexor, and great toe extensor muscle testing. Sensation testing to light touch should include L2–S1 dermatomes. Reflex testing should include bilateral patellar, medial hamstring, and Achilles reflexes. A Tinel's sign over the specific nerve in question is onsidered special testing for a mononeuropathy. A Tinel's sign is elicited by gentle percussion by a finger or hammer along the course of an injured nerve. A positive Tinel's sign is acquired by the presence of transient tingling or pain in the distribution of the injured nerve that recreates typical pain. Tingling or pain isolated to the focal area of percussion does not constitute a positive Tinel's sign. When suspicious for a nerve injury, the physical examination should be used to help distinguish between mononeuropathy, mononeuropathy multiplex, peripheral neuropathy, and radiculopathy.

Electrodiagnosis

EDX studies are the gold standard for diagnosis of most mononeuropathies. An EDX examination is not necessary for every patient with suspected nerve injury, but can be extremely helpful in localizing the lesion, assessing severity of the injury, and estimating prognosis for recovery. EDX studies involve two types of examination: EMG and NCS. In many of the smaller and deep nerves around the hip, it is very difficult to obtain reliable NCS; therefore, EMG is needed to confirm the diagnosis and rule out other potential pathologies (e.g., radiculopathy, plexopathy, or

Table 2 Motor and sensory innervation of each peripheral nerve around the hip

Lumbosacral plexus			
Peripheral nerve	Root level	Muscle innervation	Cutaneous innervation
Iliohypogastric	T12–L1	• Transversus abdominis • Abdominal internal oblique	Anterior cutaneous branch: • Small area of the skin above the pubis Lateral cutaneous branch: • Upper buttock as far as the greater trochanter
Ilioinguinal	L1	• Transversus abdominis • Abdominal internal oblique	• Groin • Proximal medial thigh • Base of the penis and the upper part of the scrotum in men • Mons pubis and labium majorum in women
Genitofemoral	L1–L2	• Cremaster muscle in males • No motor innervations in women	Genital branch: • Pubis • Scrotum in men or mons pubis and labium majorum in women Femoral branch: • Upper part of the femoral triangle • Small patch of the skin on the proximal anterior thigh
Lateral femoral cutaneous	L2–L3	• No motor innervations	• Anterolateral aspect of the thigh
Obturator	L2–L4	Anterior branch: • Adductor longus • Adductor brevis • Gracilis • Pectineus Posterior branch: • Obturator externus • Adductor brevis • Proximal portions of adductor magnus	Anterior branch: • Distal 2/3rds of the medial thigh Posterior branch: • Articular capsule, cruciate ligaments, and synovial membrane of the knee joint
Femoral	L2–L4	• Iliopsoas • Pectineus Anterior branch • Sartorius Posterior branch • Quadriceps: rectus femoris, vastus lateralis, vastus medialis, vastus intermedius	Anterior branch (intermediate and medial cutaneous nerves) • Anteromedial aspect of the thigh Posterior branch (saphenous nerve) • Anteromedial aspect of the leg, medial malleolus, and arch of the foot
Sciatic	L4–S3	Tibial division: • Semitendinosus • Semimembranosus • Long head of biceps femoris • Ischiocondylar part of adductor magnus • Gastrocnemius/soleus muscles • Posterior tibialis • Long toe flexor Peroneal division: • Short head of biceps femoris • Anterior tibialis • Long toe extensor • Peroneus longus/brevis	• Posterior gluteal region • Posterior thigh • Entire lower leg, ankle, and foot (except for the medial lower leg)

(*continued*)

Table 2 (continued)

Lumbosacral plexus			
Peripheral nerve	Root level	Muscle innervation	Cutaneous innervation
Superior gluteal	L4–S1	• Gluteus medius • Gluteus minimus • Tensor fasciae latae	• No sensory innervations
Inferior gluteal	L5–S2	• Gluteus maximus	• No sensory innervation
Pudendal	S2–S4	Inferior rectal nerve • External anal sphincter Perineal nerve • Muscles of the perineum • Erectile tissue of penis • External urethral sphincter	Inferior rectal nerve • Lower anal canal and perianal skin Perineal nerve • Perineum • Scrotum/labia Dorsal nerve of penis or clitoris • Skin of penis/clitoris

myopathy). There are many nuances to the performance and analysis of EDX testing that make it complicated and beyond the scope of this chapter. There is some variability in the interpretation of EDX studies. It is important to refer the EDX studies to physiatrists or neurologists that are comfortable in performing advanced EDX for the hip. A basic understanding of electrophysiology can help one assess the quality of the EDX study.

Nerve Conduction Studies

There are two different types of NCS: motor and sensory. Motor NCS involves the electrical stimulation of a peripheral nerve at two points (proximal and distal). The stimulation of the nerve generates action potentials that propagate down to evoke a response in the muscle distally. The amplitudes, onset latencies, and velocities of the compound muscle action potential (CMAP) for each motor nerve tested are recorded and compared to normative data or to the asymptomatic contralateral side. The CMAP represents the summation of electrical signals discharged by the muscle fibers' depolarizing membranes. Sensory NCS records the sensory nerve action potential (SNAP) on the skin after proximal stimulation of the nerve. The SNAP is the summation of electrical signals discharged by the sensory neurons' depolarizing membranes. The peak latency and conduction velocity of the SNAP is also compared to normative data. The amplitudes of SNAP vary widely and, therefore, are not useful in the analysis for neuropathy.

Prolonged onset latency (motor NCS) or peak latency (sensory NCS) implies that the conduction of the electrical impulse down the nerve was delayed. This can mean that there is some injury to the myelin; however, the preferred way to assess the integrity of the myelin sheath is by conduction velocity. A prolonged motor NCS conduction velocity is more specific for a demyelinating injury and can even localize the specific segment of nerve that is injured.

The amplitudes of the CMAP are used to determine the presence of axon loss, conduction block, or both. Axonal loss is characterized by decreased CMAP amplitudes at both the distal and proximal sites of stimulation to the motor nerve. There is no axonal loss associated with conduction block, but the injury to the myelin is so severe that it essentially blocks most, if not all, conduction down the nerve giving the false appearance that the axons are no longer intact. It is characterized by diminished proximal and preserved distal CMAP amplitudes. A mixed picture of conduction block with axonal loss is associated with a pattern of decreased distal CMAP amplitude and an even greater decrease in proximal CMAP amplitude.

Electromyography

EMG involves insertion of a needle electrode directly into the muscle. A comprehensive EMG involves testing numerous muscles specifically selected based on the suspected pathology in question. There are two portions of an EMG conducted for each muscle tested: insertional activity (irritation of the sarcolemma during a resting state of the muscle) and muscle activation (voluntary activation of the muscle at varying degrees of effort). During the insertional activity phase of testing, abnormal spontaneous discharges implies acute denervation of that muscles. During the muscle activation phase of testing, the patient is asked to engage the muscle at minimum and maximum strength. This will lead to active firing of motor units. The motor unit action potential (MUAP) morphology and recruitment patterns can distinguish muscles with chronic denervation and potential reinnervation.

The role of EMG in the diagnosis of mononeuropathy is straightforward. For a purely demyelinating lesion of the nerve, you would expect a completely normal EMG. Severe axonal pathology accompanying a mononeuropathy will result in abnormal EMG findings in all the muscles innervated by that nerve. For a comprehensive EMG exam, one would also test muscles of different peripheral nerves, but common spinal nerve roots to rule out a lumbosacral radiculopathy, mononeuropathy multiplex, or a lumbosacral plexopathy.

Ordering Electrodiagnostic Studies

The timing of the EDX study is critical to the interpretation of the study. False conclusions can occur if the EDX study is not ordered at the appropriate time. The ideal time to order an EDX study is approximately four weeks after the onset of symptoms. One can order EDX studies earlier as long as they understand the limitations of the study at each time point. Immediately after injury to a nerve, EDX findings are subtle for both demyelinating and axonal lesions and can be missed. Seven days after a complete nerve lesion Wallerian degeneration will have progressed to the point where distal stimulation of motor axons elicits no motor response. Ten days after onset of a complete lesion, SNAPs will be absent as well. Demyelinating injuries can be distinguished from axonal injuries at 7–10 days after onset of injury. The needle EMG portion will start to show active signs of denervation 2–3 weeks after onset of injury [5].

Interpreting the Electrodiagnostic Study

Understanding the varying degrees of demyelinating injuries due to a mononeuropathy and how to interpret them on EDX studies can help with prognosis. For instance, some subtle neurapraxias can demonstrate completely normal EDX results; therefore, they carry the most favorable prognosis for recovery. A demyelinating injury to the nerve with the presence of prolonged latencies and conduction velocities on NCS is the least severe type of EDX-confirmed mononeuropathies. The quantitative difference between the observed abnormal latencies and conduction velocities compared to the normative standards or contralateral asymptomatic side can also provide information on the severity of the injury. Evidence of a conduction block is considered a more severe injury to the myelin. A mixed conduction block with axonal loss is considered a progressively worse injury to the nerve. Finally, EMG evidence of denervation in a muscle supplied by the peripheral nerve in question connotes the most severe type of mononeuropathy, with the worst prognosis for recovery. In the setting of severe axonal injury, MUAP morphology can reveal acute denervation, chronic denervation, and evidence of reinnervation. These features can be used to follow a nerve injury over time and add to the prognosis for recovery.

Clinical Presentations of Nerve Injury Around the Hip

Nerve injury can result in considerable morbidity. Due to the diverse features of every nerve, the distinct clinical presentation of each mononeuropathy of the hip varies widely. All nerves around the hip originate from the lumbosacral plexus which is formed within the psoas major muscle by the merging of the lumbar plexus (anterior primary rami of L1–L4) and the sacral plexus (anterior primary rami of L5–S3) (Fig. 1).

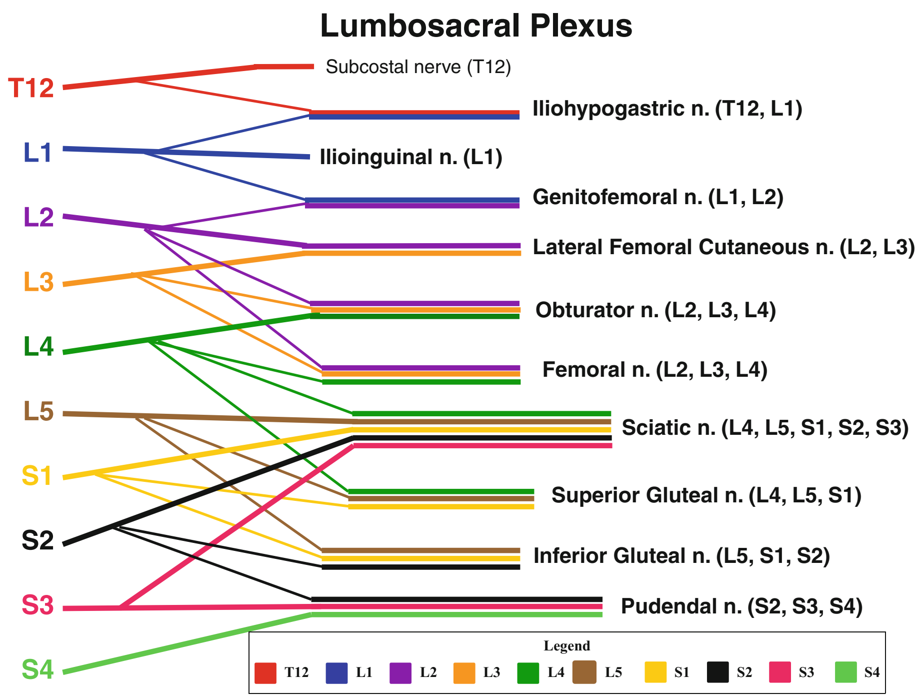


Fig. 1 Lumbosacral Plexus Anatomy. Pictoral representation of the lumobosacral root contribution to each peripheral nerve

Iliohypogastric Neuropathy

Anatomy

The iliohypogastric nerve (IHN) is a branch of the lumbar plexus arising from the primary ventral rami of L1 and a communicating branch of T12. The IHN passes through the psoas muscle and then descends. Approximately halfway between the anterior superior iliac spine (ASIS) and the highest point of the iliac crest, the nerve pierces the muscles of the abdominal wall [6]. The nerve supplies the lower fibers of the transversus abdominis and internal oblique muscles. It divides into lateral and anterior cutaneous branches. The lateral cutaneous branch crosses the iliac crest to innervate a patch of skin in the upper buttock, and the anterior cutaneous branch courses just above the inguinal ligament to supply a small area of skin above the pubis.

Etiology

Disorders of the IHN are rare. The most common causes of injury are surgical procedures involving transverse lower abdominal incisions such as hysterectomy, inguinal herniorrhaphy, and appendectomies. The main trunk of this nerve can be damaged by retroperitoneal tumors or large surgical incisions, producing sensory abnormalities in the distribution of the nerve and bulging of the lower abdominal muscles [6].

Clinical Presentation

Injury to the IHN is characterized by anesthesia, pain, and paresthesias to the lower abdomen and groin. Patients with injury to the anterior branch of the IHN report a suprapubic sensory disturbance, whereas patients with injury to the lateral branch report an isolated sensory disturbance over the upper buttock. There is some dermatomal overlap with the ilioinguinal nerve, which makes it clinically difficult to differentiate them. Weakness of the lateral abdominal wall musculature can be present; however, these muscles receive other innervation from the lower intercostals nerves. Furthermore, lateral abdominal muscle testing is difficult to reliably assess.

Diagnosis

The gold standard of diagnosis is a local anesthetic block of the nerve. Pain relief after the block confirms the diagnosis. These blocks are currently being performed under ultrasound guidance. There is no reliable EDX that can be performed to validate the diagnosis. Needle EMG of the lower abdominal musculature may serve as an adjunct in the diagnosis, although it is not commonly performed. Differential diagnosis includes upper lumbar or lower thoracic radiculopathy.

Ilioinguinal Neuropathy

Anatomy

The ilioinguinal nerve (IIN) arises as a branch of the lumbar plexus and is derived from the L1 ventral rami. The nerve pierces through the psoas muscle and follows a parallel course to the IHN as it runs distally and anteriorly, staying deep to the abdominal musculature. Adjacent to the ASIS, the nerve sends motor contributions to the inferior portions of the transversus abdominis and internal oblique muscles and cutaneous contributions to innervate a strip of skin over the iliac crest. The remainder of the nerve enters the inguinal canal and divides to provide cutaneous innervation for the groin, proximal medial thigh, base of the penis, and upper part of the scrotum, mons pubis, and labium majorum.

Etiology

IIN entrapment can occur proximally due to scarring from prior surgical incisions. Nerve injury can also develop from iliac crest bone harvesting, blunt nonsurgical trauma to the lateral abdominal wall, and hypertrophied abdominal muscles. Common surgical procedures resulting in ilioinguinal neuropathy are appendectomy, herniorrhaphy, and hysterectomy. Tumors and endometriosis can also lead to compressive neuropathies of the IIN.

Clinical Presentation

Pain is present in the inguinal region with potential radiation into the genitals. Sensory abnormalities in the distribution of this nerve have also been noted. On examination, tenderness to palpation may be elicited 2–3 cm medial and inferior to the ASIS. The clinical presentation of IIN injury is similar to IHN injury, and often both clinical entities are difficult to distinguish from each other.

Diagnosis

Similar to IHN injury, a local anesthetic block is the gold standard for diagnosis of injury to the IIN. No EDX studies are readily available to test this nerve. As with the IHN injury, lateral abdominal needle EMG may be helpful as an adjunct to diagnosis, but it is neither sensitive nor specific.

Genitofemoral Neuropathy

Anatomy

The genitofemoral nerve (GFN) is a small nerve that arises from the ventral rami of the L1–L2 spinal nerves and pierces the anterior surface of the psoas muscle at the level of L3–L4. It then descends retroperitoneally until the inguinal ligament, where it divides into the genital and femoral branches. The genital branch continues along the psoas major muscle and enters the inguinal canal. In males, it supplies the cremaster muscle, the skin of the pubis, and scrotum. In females, it travels with the round ligament of the uterus and provides cutaneous sensation to the labia majora and mons pubis. The terminal genital branch runs a course similar to the terminal IIN. The femoral branch travels lateral to the femoral artery and passes under the inguinal ligament to enter the proximal thigh. It pierces the sartorius muscle distal to the inguinal ligament and supplies a small patch of skin on the proximal anterior thigh and the upper part of the femoral triangle.

Etiology

Injury to this nerve is rare. The most common reported cause of entrapment of the GFN is surgical trauma (e.g., hernia repair, appendectomy, biopsies, and cesarean delivery). Other rare case reports include wearing tight clothing and direct trauma to the groin resulting in local scarring [6].

Clinical Presentation

Injury to GFN or genital branch causes groin pain which may be worse with internal or external rotation of the hip, prolonged walking, or even with light touch. Injury to the femoral branch causes pain, paresthesias, or anesthesia over the anterior thigh below the inguinal ligament, which distinguishes it from IHN and IIN injuries.

Diagnosis

It is often difficult to accurately separate GFN, IIN, and IHN lesions on clinical grounds as their terminal cutaneous sensory distribution commonly overlaps. There are no specific distinguishing tests of motor function. An image-guided diagnostic local anesthetic block may be performed to assist in diagnosis. The differential diagnosis includes injury to the IIN, IHN, and L1–L2 radiculopathies.

Lateral Femoral Cutaneous Neuropathy

Anatomy

The lateral femoral cutaneous nerve (LFCN) is a pure sensory nerve that receives fibers from the L2–L3 spinal nerves. The nerve passes through or under the psoas muscle [7] and across the iliacus muscle. It exits the pelvis under the lateral end of the inguinal ligament through a tunnel created by the inguinal ligament and the ASIS [4, 6, 8]. Approximately 10–12 cm distal to the inguinal ligament in line with the ASIS, the nerve divides into anterior and posterior branches that pierce the fasciae latae and innervates the skin of the lateral aspect of the thigh [8, 9]. The size of the area innervated varies among individuals [7–10].

Etiology

Entrapment of the LFCN, also known as meralgia paresthetica, is a focal injury causing pain and sensory loss in the lateral thigh of the affected individual. Most cases of meralgia paresthetica are caused by entrapment at the inguinal ligament or at the tensor fasciae latae [11]. This disorder can be precipitated by obesity, diabetes, and pregnancy [8, 10, 12, 13]. Additional causes of injury include: (1) pelvic and retroperitoneal tumors, (2) stretching of the nerve due to prolonged leg and trunk hyperextension, (3) rapid weight loss, (4) surgical complication, and (5) external compression by belts, weight gain, or tight clothing. It is rare for direct pelvic trauma to cause injury to the LFCN. LFCN injury is common after hip arthroscopy and pelvic surgery. The terminal branches of the LFCN are in close proximity to the anterior portal for hip arthroscopic surgery.

Clinical Presentation

Patients complain of lateral thigh pain, numbness, or paresthesias. The pain is often burning in quality but may also be described as sharp, dull, or aching. The patient should not complain of weakness because the LFCN has no motor component. Pain is aggravated by sitting [14]. In 80 % of cases, symptoms are unilateral [10]. On examination, the classic finding is decreased sensation in the lateral thigh. The area of decreased sensation is variable in size. Palpation or Tinel's over LCFN at the ASIS may exacerbate symptoms.

Diagnosis

History and physical examination are the most important diagnostic tools. Sensory NCS of the LFCN is feasible, but technically difficult even in the best circumstances. The difficulty of this test rises significantly in obese patients, who are at increased risk for entrapment of the nerve. EDX testing of the LFCN would demonstrate absent sensory response or prolonged nerve conduction velocity and latency with a normal EMG test. Femoral neuropathy, lumbar plexopathy, and an upper lumbar radiculopathy can mimic symptoms from meralgia paresthetica. EDX testing is helpful if these other etiologies are suspected. Imaging with ultrasound, computed tomography (CT), or magnetic resonance imaging (MRI) of the abdomen and pelvis is not common unless there is suspicion of a mass lesion causing the nerve impingement. A local anesthetic block of the nerve using imaging guidance for the injection can also aid in the diagnosis, although history and physical are the most common diagnostic tools for injury to the LFCN.

Obturator Neuropathy

Anatomy

The obturator nerve originates from the ventral rami of the L2–L4 spinal nerves. It enters the pelvis through the medial border of the psoas muscle at the level of the sacroiliac joint. The nerve runs distally on the lateral side of the ureter and the internal iliac artery. It exits the pelvis through the obturator foramen splitting into anterior and posterior divisions. The anterior branch runs in front of the adductor brevis and adductor magnus muscles and behind the adductor longus muscle. It provides motor innervation to adductor longus, adductor brevis, gracilis, and pectineus muscles. It provides cutaneous innervation to portions of the distal two-thirds of the medial thigh. The posterior division runs between the adductor brevis and adductor magnus muscles. It supplies the adductor brevis, obturator externus, and proximal portions of the adductor magnus muscles, which is also innervated by the sciatic nerve. Its sensory division supplies the articular capsule, cruciate ligaments, and synovial membrane of the knee joint [6].

Etiology

A common place for obturator nerve entrapment is a fascial band at the level of the obturator foramen and proximal thigh [14]. The nerve may also be compressed by a hematoma, obturator hernia, tumor, endometriosis, or pelvic fracture [13, 15, 16]. In pelvic fractures, the obturator nerve can be injured because of its close relationship to the pelvic bones. Traumatic injuries (e.g., pelvic fractures, gunshot wounds) with isolated obturator nerve injury are relatively uncommon because they typically will involve other nerves as well [16]. Surgically, obturator neuropathy can result from pelvic or hip procedures secondary to stretch, compression from a retractor, or encasement by cement [16].

Clinical Presentation

Pain, paresthesias, or anesthesia may occur in the mid- and lower-thirds of the medial thigh but can also radiate to the groin or knee. The pain is often exacerbated with activities that stretch the nerve such as hip extension or abduction. Patients may complain of difficulty walking because they have weakness with hip adduction. This weakness presents clinically as a circumducting wide-based gait [16]. Hip adduction weakness is the most prominent sign of obturator neuropathy. Weakness of the obturator externus (lateral rotation of the thigh) and gracilis (flexion and internal rotation of the leg) muscles occur to a lesser degree because these movements are adequately compensated by muscles innervated by other nerves.

Diagnosis

Due to the deep location of the obturator nerve, NCS is not performed. Obturator neuropathy can be confirmed by needle EMG with evidence of denervation in the hip adductor muscles, but normal EMG of other L2–L4 innervated muscles supplied by different peripheral nerves such as iliopsoas or the quadriceps muscles [16, 17]. The EMG will help differentiate between obturator neuropathy, upper lumbar radiculopathy, and lumbar plexopathy. Additional diagnostic testing searching for the etiology of nerve injury is sometimes needed. Suspicion of a mass lesion entrapping the obturator nerve would call for further examination with CT, MR, or ultrasound imaging studies.

Femoral Neuropathy

Anatomy

The femoral nerve arises from the posterior divisions of the ventral rami of the L2–L4 spinal nerves in the lumbar plexus. Motor branches of the femoral nerve initially supply the psoas and iliacus muscle. From the psoas major muscle, it passes distally deep to the inguinal ligament and gives off a motor branch to the pectineus muscle. The nerve remains lateral to the femoral artery as it enters the thigh and divides into anterior and posterior branches approximately 4 cm distal to the inguinal ligament [4, 16]. The sensory portion of the anterior branch divides into the intermediate cutaneous and medial cutaneous nerves to supply the anteromedial aspect of the thigh. The motor portion of anterior branch supplies the sartorius muscle. The posterior branch of the femoral nerve gives off the saphenous nerve, the largest cutaneous branch, which continues distally and supplies the sensory innervations to the anteromedial aspect of the leg, medial malleolus, and arch of the foot [4, 16]. The motor portion of the posterior branch supplies the quadriceps: rectus femoris, vastus lateralis, vastus medialis, and vastus intermedius muscles.

Etiology

The femoral nerve is often injured in the pelvis as it passes beneath the inguinal ligament [12]. Many of the causes of femoral neuropathy are iatrogenic [16]. The femoral nerve is vulnerable to injury during surgical procedures involving the abdomen, pelvis, inguinal area, and hip. These injuries are often due to the fact that the femoral nerve lies near the anterior capsule of the hip joint. The nerve is separated from the joint capsule only by the iliopsoas muscle and tendon. After hip arthroplasty, the incidence of femoral neuropathy has been reported to range from 0.1 % to 2.3 % [16, 18]. The various mechanisms of injury following surgery include pressure from the retractors, entrapment by bone cement, thermal injury, hematoma, or postoperative scar formation [16]. Sustained postures of the hip in extreme abduction and external rotation are also known to compress the femoral nerve beneath the inguinal ligament. This is often seen in obstetric and gynecologic procedures requiring the lithotomy position. Nonsurgical causes of femoral neuropathy include direct trauma to the nerve, stretching injuries from prolonged hyperextension, compression injuries during hip flexion, hematoma, or mass lesions.

Clinical Presentation

The femoral nerve is a large nerve that supplies many other peripheral nerves; therefore, damage can produce a variety of different clinical presentations. The manifestation of femoral neuropathy commonly presents as a mixed motor and sensory deficit. The location of injury to the nerve often dictates its clinical manifestation. Patients will often complain of dull, aching pain in the groin that radiates to the anterior thigh. Quadriceps weakness is most notable and can be accompanied with thigh atrophy in severe cases. Patients may notice difficulty with functional activities such as ambulation, getting out of a chair, and climbing stairs or inclines [4, 13, 19–21]. Sensory symptoms may be mild or absent [16]. If present, numbness is commonly noted over the anteromedial thigh, lower leg, and may extend into the medial aspects of the ankle and foot.

A severe femoral nerve lesion produces wasting and weakness of the quadriceps muscles, absent patellar reflex, and sensory impairment over the anteromedial thigh, anteromedial calf, medial foot, and the great toe [16]. This patient is typically able to walk, especially on level surfaces, because the gastrocnemius, tensor fasciae latae, gracilis, and gluteus maximus muscles aid in stabilizing the limb and keeping the knee hyperextended [4, 16]. The severely affected patients with femoral neuropathy will often describe buckling of the knee due to quadriceps weakness.

In partial lesions of the femoral nerve, various combinations of motor and sensory loss in part or all of the femoral distribution can be seen. Quadriceps weakness may be noted, but this muscle group is so strong that the examiner may not be able to detect a deficit [20]. An intact iliopsoas muscle masks any hip flexion weakness caused by rectus femoris and sartorius dysfunction. Quadriceps strength should be compared with adductor strength, which is expected to be normal. Lower leg muscles must be examined to ensure that the muscles in the sciatic distribution are normal [20].

Diagnosis

EDX studies are the gold standard to confirm the presence of a femoral nerve injury. They also play a role in establishing a prognosis for recovery. The routine EDX examination includes side-to-side comparison of femoral motor NCS and saphenous sensory NCS. The diagnosis of femoral neuropathy is supported by EMG evidence of denervation in the quadriceps muscles, but not in the lower leg or posterior thigh muscles [12, 20]. The adductors are especially important to test because they are innervated by the same nerve roots as the quadriceps muscles, but are supplied by the obturator nerve [12]. A complete needle EMG evaluation should include the iliopsoas muscle, at least 2 quadriceps muscles, 1–2 adductor muscles, gluteus minimus, 3 muscles between the knee and the ankle, and lumbar paraspinal muscles [19]. Thorough EDX testing is important to rule out other possible etiologies, which include: a different mononeuropathy, lumbar plexopathy, L4 radiculopathy, or neuromuscular junction disorder.

Sciatic Neuropathy

Anatomy

The sciatic nerve has contributions from the L4–S3 nerve roots. It exits through the greater sciatic notch inferior to the piriformis muscle. At this level, the sciatic nerve has peroneal and tibial divisions, and it is the largest nerve in the body with its transverse diameter being 2.0–2.5 cm [4]. The nerve descends deep to the gluteus maximus to the level of the inferior gluteal fold, where it lies between the ischial tuberosity and the greater trochanter. Just proximal to the popliteal fossa, the sciatic nerve divides into its two large divisions forming the common peroneal nerve, which deviates laterally, and the larger tibial nerve, which continues distally in the midline of the limb [4]. The tibial division of the sciatic nerve innervates the semitendinosus, semimembranosus, long head of biceps femoris, and a portion of adductor magnus muscles. Of the hamstring muscles, only the short head of the biceps femoris is supplied by the peroneal division of the sciatic nerve. Through the common peroneal and tibial nerves, the sciatic nerve innervates all the muscles of the lower leg and foot. Sensory distribution of the sciatic nerve involves the posterior gluteal region, posterior thigh, and entire lower leg, ankle, and foot, except for the medial lower leg that is supplied by the saphenous branch of the femoral nerve. The sural nerve is a pure sensory nerve that branches from the tibial portion of the sciatic nerve and supplies the skin on the posterior lateral calf and foot.

Etiology

The majority of sciatic neuropathies occur in the hip and gluteal region, rather than in the thigh [22]. The sciatic nerve is most likely to be injured as it leaves the sciatic notch and descends into the upper leg [12]. Compressive injuries to the nerve can be caused by anatomic variations in the relationship of the nerve to the piriformis muscle (i.e., piriformis syndrome) and to the sciatic notch [4]. The sciatic nerve is susceptible to injury from pelvic and sacral fractures; hip surgery or posterior hip dislocation; repetitive trauma, as seen in cyclists; intramuscular buttock needle injection injuries; hematoma or hemorrhages; nerve infarction from thromboembolic events; mass lesions; and any penetrating injury. It is important to note that the peroneal division is more susceptible to injury than the tibial division based on anatomic features and location [22].

Clinical Presentation

Pain, weakness, numbness, and paresthesias are the main presenting symptoms of sciatic neuropathy. Patients present with pain that is localized close to the level of the sciatic nerve lesion, although substantial radiation of the pain may be a feature. Weakness can affect all muscles of the lower leg and the hamstring muscles. One would expect to see weakness of knee flexion, ankle dorsiflexion, ankle plantar flexion, ankle eversion, toe extension, and toe flexion. A comprehensive battery of strength and sensory testing in the lower extremity can help localize the lesion of the sciatic nerve. The muscle groups that can be tested accurately arising from the tibial portion of the sciatic nerve include the hamstrings, gastrocnemius/soleus complex, posterior tibialis, and long toe flexors muscles. The muscles supplied by the peroneal division of the sciatic nerve are further divided into the deep peroneal nerve (anterior tibialis and long toe extensor muscles) and the superficial peroneal nerve (peroneus longus and brevis muscles). Loss of sensation can occur below the knee, but it will uniquely spare the medial lower leg (the territory of the saphenous branch of the femoral nerve). If the peroneal division is exclusively involved, sensory loss is primarily over the lateral aspect of the leg and dorsum of the foot [4]. If the tibial nerve is exclusively involved, the sensory deficit is primarily over the plantar aspect of the foot [4]. Achilles reflexes are usually diminished or absent in severe sciatic neuropathy. Some dramatic observational physical findings after a sciatic nerve injury include equinus deformity of the foot, clawing of the toes, and atrophy of the muscles innervated by the sciatic nerve.

Diagnosis

EDX studies play a critical role in the diagnosis and localization of a sciatic nerve injury. Motor and sensory NCS can be performed on the terminal nerve branches from the sciatic nerve (common peroneal, tibial, superficial peroneal, and sural nerves). EMG can confirm the distribution of denervation. Suggested muscles to test for needle EMG include peroneal- and tibial-innervated muscles above and below the knee and several non-sciatic-innervated muscles, including gluteal muscles, femoral-innervated muscles, and lumbar paraspinal muscles. MRI and CT are primarily reserved to rule out tumors and other causes of sciatic nerve or plexopathy. Given the length and complicated nature of the sciatic nerve, a MR neurography can also be helpful in the localization of nerve injury. Again, history and physical examination should be the first line to the diagnosis of a sciatic neuropathy.

Superior Gluteal Neuropathy

Anatomy

The superior gluteal nerve (SGN) is derived from L4–S1 spinal nerves. It leaves the pelvis through the greater sciatic foramen superior to the piriformis. The SGN is a purely motor nerve that supplies the gluteus medius and gluteus minimus muscles and tensor fasciae latae. There are no cutaneous sensory fibers.

Etiology

Injuries to the SGN are rare, but have been reported after local buttock trauma, pelvic fractures, and hip surgery [6]. Because of its separate course above the piriformis, injuries to this nerve may occur in isolation from other deep gluteal nerves [6]. With respect to hip arthroplasty, the SGN is most susceptible to injury with anterolateral approaches that split the gluteus medius muscle [18]. Other maneuvers that may injure this nerve include vigorous acetabular retraction for component insertion and extreme leg positioning for femoral preparation [18].

Clinical Presentation

The clinical manifestation of damage to the SGN is pain in the gluteal region and hip abduction and external rotation weakness. If the injury is severe enough, a Trendelenburg gait can be seen. There is no concomitant sensory loss or paresthesias associated with superior gluteal neuropathy.

Diagnosis

Isolated weakness in hip abduction will be the clinical hallmark on physical examination. To best test hip abduction strength, the patient should be placed in side-lying with the strong hip in contact with the table. Testing hip abduction with the hip in neutral will give a generalized impression of hip abductor strength without specificity. However, testing the hip abduction strength with the hip in 15–20° of hip extension will best challenge the strength of the gluteus medius muscle specifically. Tenderness to palpation of the upper gluteal region corresponding to the greater sciatic notch can also be seen. Additionally, EMG can be performed specifically on the gluteus medius and tensor fasciae latae muscles to look for denervation. Other L4–S1 muscles and lower lumbar paraspinal muscles should be tested by EMG to rule out plexopathy or radiculopathy. There are no NCS available to aid in this diagnosis.

Inferior Gluteal Neuropathy

Anatomy

The inferior gluteal nerve (IGN) arises from ventral rami of L5–S2 spinal nerves. It leaves the pelvis through the greater sciatic foramen, inferior to the piriformis, and supplies only the gluteus maximus muscle via several branches. The IGN has no cutaneous sensory distribution.

Etiology

Injury to the IGN rarely occurs without iatrogenic causes. The IGN is usually injured in variable combination with the other nerves exiting below piriformis, namely, the sciatic, pudendal, and posterior femoral cutaneous nerve of the thigh [6]. The role of piriformis hypertrophy as the compressive structure in producing this condition is controversial [6]. Isolated injury to the IGN has also been reported to result from local trauma or space-occupying lesions.

Clinical Presentation

Severe injury to the IGN will result in atrophy of the gluteus maximus and weakness of hip extension. There should be no sensory changes accompanying an isolated IGN injury; however, since the IGN is in close proximity to the posterior femoral cutaneous nerve, they are often injured together. Patient may also complain of a deep ache in the buttock.

Diagnosis

Clinical examination confirming hip extension weakness and gluteus maximus atrophy will be the most useful elements for diagnosis. Needle EMG testing of the gluteus maximus can also be helpful to determine denervation. Other L5–S1 muscles and lower lumbar paraspinal muscles should also be tested by EMG to rule out plexopathy or radiculopathy. There are no NCS available to aid in this diagnosis.

Pudendal Neuropathy

Anatomy

The pudendal nerve is the principal nerve of the perineum originating in the sacral plexus with contributions from the S2–S4 spinal nerve. It carries motor, sensory, and autonomic fibers. The pudendal nerve exits the pelvis through the greater sciatic foramen, traveling anterior to the piriformis muscle. It winds posteriorly around the ischial spine, swings anteriorly, and reenters the pelvis through the lesser sciatic foramen. It emerges below the pubic bone to enter the perineum and gives off its terminal branches: the dorsal nerve of the penis or clitoris, the inferior rectal nerve (innervates the external anal sphincter and sensory fibers to lower anal canal and perianal skin), and the perineal nerve (innervates muscles of the perineum; the erectile tissue of the penis; the external urethral sphincter; and the skin of the perineum, scrotum, or labia).

Etiology

Pudendal nerve compression can occur anywhere along the course of the nerve. Direct injuries to this nerve are rare because it is deeply situated. The pudendal nerve has also been reported to be damaged by deep buttock injections, prolonged childbirth, surgical manipulation, and pelvic fractures [6]. In cyclists, this nerve may be compressed with prolonged riding which has been reported to result in sensory loss or impotence in severe cases.

Clinical Presentation

Patients complain of burning pain or paresthesias in the perineum with potential numbness in the shaft of the penis and perineum. Some patients can present with impotence, urinary frequency or urgency, dyspareunia, or persistent sexual arousal [23]. Symptoms are worsened with sitting and relieved with lying down or standing [24]. History usually reveals activity that involves prolonged compression of the perineum such as long-distance cycling.

Diagnosis

The diagnosis of pudendal neuropathy is inherently difficult due to the overlap of innervation it shares with other pelvic nerves. There are many other clinical entities that mimic the symptoms of pudendal neuralgia including vulvodynia, pelvic floor dysfunction, interstitial cystitis, and nerve injury to the obturator, genitofemoral, or ilioinguinal nerves. History and physical examination is the hallmark for diagnosis and typically includes an internal bimanual pelvic floor examination of the pelvic floor muscles and ligaments.

The insertion site for the sacrospinous ligament at the ischial spine is often an area of maximal tenderness for patients with pudendal neuropathy. A Tinel's sign can be performed at this site [25]. Motor NCS can be performed on the pudendal nerve; however, the results are not reliable and subject to high interobserver variability. Local anesthetic blocks of the pudendal nerve can aid in the diagnosis.

Management of a Nerve Injury

Treatment of a mononeuropathy is focused on three separate areas: facilitation of nerve healing, relief of symptoms, and restoration of function.

Nerve Healing

Removing the inciting cause of nerve injury or avoiding conditions of compression is the first step in allowing the nerve to heal. The etiology and exact location of the nerve injury becomes important because in acute cases in which hemorrhage, trauma, scar tissue, or mass lesion is the cause, surgical intervention may be the initial treatment. However, more frequently, there is little that can be done to facilitate healing from a surgical standpoint. Oftentimes, the injury to the nerve is a transient moment, but the effects of the injury are longer-lasting. Therefore, it is important to educate the patient about the realistic expectations for nerve healing. Depending on the severity of injury, nerve healing has a variable time course. Nerve recovery can occur in as little as a few days in mild cases to over a year in severe cases. Additionally, it is important to counsel patients with severe or longstanding injury that full healing may never occur and there may be permanent loss of strength and sensation as well as continued pain symptoms.

Symptom Relief

Relief of pain and numbness is attempted with modalities and medications which include: ice in the acute setting; heat in the subacute stage; nonsteroidal anti-inflammatory drugs (NSAIDs); oral corticosteroids; and neuropathic pain medications such as antiseizure medications (e.g., gabapentin, pregabalin), serotonin-norepinephrine reuptake inhibitors (e.g., duloxetine, venlafaxine), or tricyclic antidepressants (e.g., amitriptyline, nortriptyline, desipramine, and imipramine). Image-guided local anesthetic blocks with and without corticosteroid have been used to break the pain cycle for the patient and have mixed results. These injections tend to be preferentially used in the smaller nerves (IIN, IHN, GFN, LFCN, gluteal nerves, and pudendal nerve) rather than the larger nerves (obturator, femoral, or sciatic). Surgical exploration is rarely used as a last resort for symptom relief.

Restoration of Function

The rehabilitation phase focuses on the restoration of function. Physical and occupational therapy will guide patients to improve range of motion, strength, and functional mobility. If there is severe weakness affecting gait, the physical therapist will improve proper gait mechanics and assess for safety with ambulation. Sometimes an assistive device for ambulation is necessary for profound weakness. In patients with chronic or severe neuropathic pain, therapy can be aimed at desensitizing the nerves by progressively increasing tolerance to touch and textures over the area of pain.

Summary

Nerve injuries can occur commonly around the hip. Physical examination and precise knowledge of anatomy and function of the nerve can aid in the diagnosis and localization of a nerve injury; however, in most cases, electrodiagnostic studies are the gold standard for diagnosis. Isolated mononeuropathies of the nerves around the hip can cause a characteristic constellation of sensory symptoms and patterns of weakness that can help the healthcare provider identify the etiology of nerve injury. Electrodiagnostic studies can also aid in the prognosis for recovery. Awareness of the potential for nerve injuries when performing hip surgery is an important consideration.

References

1. Seddon HJ. A Classifcation of Nerve Injuries. Br Med J. 1942:29;2(4260):237–9.
2. Preston DC. Approach to nerve conduction studies and electromyography. In: Preston DC, Shapiro BE, editors. Electromyography and neuromuscular disorders. 3rd ed. London: W.B. Saunders; 2013. p. 1–7.
3. Weiss L. Injury to peripheral nerves. In: Weiss L, Silver JK, Weiss J, editors. Easy EMG. Edinburgh: Butterworth-Heinemann; 2004. p. 81–6.
4. Jobe MT, Martinez SF. Peripheral nerve injuries. In: Canale ST, Beaty JH, editors. Canale & Beaty: Campbell's operative orthopaedics. 12th ed. Philadelphia: Elsevier Mosby; 2012. p. 3062–123.e6.
5. Pease WS, Lew HL, Johnson EW. Johnson's practical electromyography. 4th ed. Philadelphia: Lippincott Williams & Wilkins; 2007.
6. McCrory P, Bell S. Nerve entrapment syndromes as a cause of pain in the hip, groin and buttock. Sports Med. 1999;27(4):261–74.
7. Delaunay L, Jochum D. Anatomy of the lumbar and sacral plexus. In: Chelly JE, editor. Peripheral nerve blocks: a color atlas. 2nd ed. Philadelphia: Lippincott Williams & Wilkins; 2004. p. 74–86.
8. Craig EJ, Clinchot DM. Lateral femoral cutaneous neuropathy. In: Frontera WR, Silver JK, Rizzo TD, editors. Frontera: essentials of physical medicine and rehabilitation. 2nd ed. Philadelphia: Saunders Elsevier; 2008. p. 283–86.
9. Stevens H. Meralgia paresthetica. AMA Arch Neurol Psychiatry. 1957;77(6):557–74.
10. Aminoff MJ. Neurologic disorders. In: Creasy RK, Resnik R, Iams JD, Lockwood CJ, Moore TR, editors. Creasy and Resnik's maternal-fetal medicine: principles and practice. Philadelphia: Saunders Elsevier; 2009. p. 1089–112.
11. Beltran LS, Bencardino J, Ghazikhanian V, Beltran J. Entrapment neuropathies III: lower limb. Semin Musculoskelet Radiol. 2010;14(5):501–11. doi:10.1055/s-0030-1268070.
12. Misulis KE. Lower back and lower limb pain. In: Daroff RB, Fenichel GM, Jankovic J, editors. Bradley's neurology in clinical practice. 6th ed. Philadelphia: Elsevier; 2012. p. 349–60.
13. Shah A, Busconi B. Hip, pelvis, and thigh. In: DeLee JC, Drez Jr D, Miller MD, editors. DeLee and Drez's orthopaedic sports medicine. 3rd ed. Philadelphia: Saunders Elsevier; 2009. p. 1451–512.
14. Richer LP, Shevell MI, Stewart J, Pouli C. Pediatric meralgia paresthetica. Pediatr Neurol. 2002;26:321–3.
15. Bradshaw C, McCrory P, Bell S, Brukner P. Obturator nerve entrapment. A cause of groin pain in athletes. Am J Sports Med. 1997;25(3):402–8.
16. Busis NA. Femoral and obturator neuropathies. Neurol Clin. 1999;17(3):633–53.
17. Freeman TL, Johnson E, Freeman ED, Brown DP. Lower limb mononeuropathy. In: Cuccurullo S, editor. Physical medicine and rehabilitation board review. New York: Demos Medical Publishing; 2004.
18. Harkness JW, Crockarell JR. Arthroplasty of the hip. In: Canale ST, Beaty JH, editors. Campbell's operative orthopaedics, vol. 1. 11th ed. Philadelphia: Mosby Elsevier; 2008. p. 312–481.
19. Craig EJ, Clinchot DM. Femoral neuropathy. In: Frontera WR, Silver JK, Rizzo TD, editors. Frontera: essentials of physical medicine and rehabilitation. 2nd ed. Philadelphia: Saunders Elsevier; 2008. p. 277–81.
20. Misulis KE. Hemiplegia and monoplegia. In: Daroff RB, Fenichel GM, Jankovic J, editors. Bradley's neurology in clinical practice. 6th ed. Philadelphia: Elsevier; 2012. p. 273–85.
21. Rutkove SB. Peripheral neuropathies. In: Frontera WR, Silver JK, Rizzo TD, editors. Frontera: essentials of physical medicine and rehabilitation. 2nd ed. Philadelphia: Saunders Elsevier; 2008. p. 767–71.
22. Yuen EC, Yuen TS. Sciatic neuropathy. Neurol Clin. 1999;17(3):617–31.
23. Waldinger MD, Venema PL, van Gils AP, Schweitzer DH. New insights into restless genital syndrome: static mechanical hyperesthesia and neuropathy of the nervus dorsalis clitoridis. J Sex Med. 2009;6:2778–87.
24. Ramsden CE, McDaniel MC, Harmon RL, Renney KM, Faure A. Pudendal nerve entrapment as a source of intractable perineal pain. Am J Phys Med Rehabil. 2003;82:479–84.
25. Hibner M, Desai N, Robertson L, Nour M. Pudendal neuralgia. J Minimally Inv Gynecol. 2010;17(2):148–53.

Surgical Technique: Endoscopic Sciatic Nerve Release

75

Hal D. Martin, Munif A. Hatem, and Ian J. Palmer

Contents

Abstract

The deep gluteal space has increasingly received attention since the evolution in the knowledge of deep gluteal pain etiologies and their diagnoses. The endoscopic technique is being utilized to assess and treat deep gluteal space pathologies, improving the understanding of the sciatic nerve anatomy and biomechanics. This chapter presents a "10-step technique" for deep gluteal space endoscopic assessment and sciatic nerve decompression.

Introduction

The etiology of sciatic nerve entrapment can be diverse and may occur in one or more locations within the deep gluteal space. The suggested term to describe the clinical manifestation of these various points of nervous compressive pathology is deep gluteal syndrome (DGS) [1]. Of utmost importance to the diagnosis of DGS, is the exclusion of other sources of sciatica, most commonly the lumbar spine. Sources of posterior hip pain can mimic sciatica; however, a comprehensive history and physical examination of the hip will guide the examiner toward a differential diagnosis (covered in ▶ Chap. 7, "Physical Examination of the Hip and Pelvis"). The entire extrapelvic course of the sciatic nerve within the deep gluteal space can be assessed via an endoscopic surgical technique, allowing the diagnosis and treatment of the causes of DGS. The experienced surgeon must

H.D. Martin (✉) • M.A. Hatem • I.J. Palmer
Baylor University Medical Center, Hip Preservation Center, Dallas, TX, USA
e-mail: Hal.Martin@baylorhealth.edu; munifhatem@gmail.com; ianjamespalmer@gmail.com

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_117

Table 1 Summary of published results of open decompression of the sciatic nerve

Author	Number of procedures	Results
Miller et al. [6]	1	Immediate pain relief, 2.5 years post-op no pain yet decreased sensation over the posterolateral aspect of thigh
Vandertop and Bosma [3]	1	4 years post-op, doing well
Chen [7]	1	Pain resolved in 1 week. Motor weakness of the ankle extensors and toes for 3 months. 4 years post-op asymptomatic
Hughes et al. [8]	5	At 1 year: (1) No pain, slight residual tenderness in buttock, (2) asymptomatic, (3) no pain, slight residual tenderness in buttock, (4) no pain, (5) excellent
Sayson et al. [9]	1	6 months post-op: no pain
Benson and Schutzer [4]	15	2 years post-op: 11 excellent, 4 good
Meknas et al. [10]	12	No pain decrease at 6 months. 8 years post-op: significant decrease in pain
Filler et al. [2]	64	2 years post-op: excellent: 59 %, good: 23 %, no benefit: 17 %, worse: 2 %
Lewis et al. [11]	4	2 months post-op: 3 excellent, 1 still experiencing pain
Issack et al. [12]	10	1 year post-op: partial to complete relief of radicular pain, of diminished sensation, and of paresthesias
Young et al. [5]	44 hamstring	53 months post-op: 33 satisfied, 5 somewhat satisfied, 6 not satisfied
Beauchesne and Schutzer [13]	1	Immediate pain relief, residual numbness and limp resolved in 4 weeks
Jones et al. [14]	1	Immediate pain relief, 6 weeks post-op: complete resolution of symptoms

have a detailed understanding of the anatomy and biomechanics of all four layers of the hip joint and their interrelationships to accurately supply a comprehensive diagnosis and treatment plan (covered in ▶ Chap. 7, "Physical Examination of the Hip and Pelvis"). The open transgluteal approach has been described to effectively perform piriformis muscle resection and neurolysis of the sciatic and posterior femoral cutaneous nerves [2, 3]. Open operative treatment has been successful in a number of case studies [2, 4, 5]. Additionally, neurolysis of the sciatic nerve near the hamstrings origin at the level of the ischial tuberosity has been performed with satisfactory results [5]. Table 1 is a summary of open technique results for treatment of sciatic nerve entrapment.

Endoscopic Decompression

Endoscopy is an effective approach for the treatment of deep gluteal syndrome [15]. Advantages of endoscopy include minimally invasive procedure, magnified view of the sciatic nerve, and potential to evaluate the entire deep gluteal space from the sciatic notch to the proximal thigh, including sciatic nerve biomechanics. Dezawa et al. first reported on six cases of endoscopic piriformis muscle release [16]. Martin et al. reported an endoscopic technique for sciatic nerve decompression in 35 patients presenting with deep gluteal syndrome [15], and this technique has been utilized internationally [17].

The supine technique is utilized and modified by positioning the orthopedic table in maximal contralateral patient tilt. The supine position, as opposed to prone, allows for manual manipulation of the lower limb at the knee and hip joints for the full assessment of sciatic nerve kinematics. A relative contraindication for sciatic nerve decompression in this supine technique is knee recurvatum, considering the increased strain on the sciatic nerve. Nerve conduction and EMG are usually monitored intraoperatively and can demonstrate immediate improvement or change post-release. Mild acute changes in nerve conduction during the course of surgery may be observed due to fluid accumulation over time. For deep gluteal space visualization, a 70° high-definition

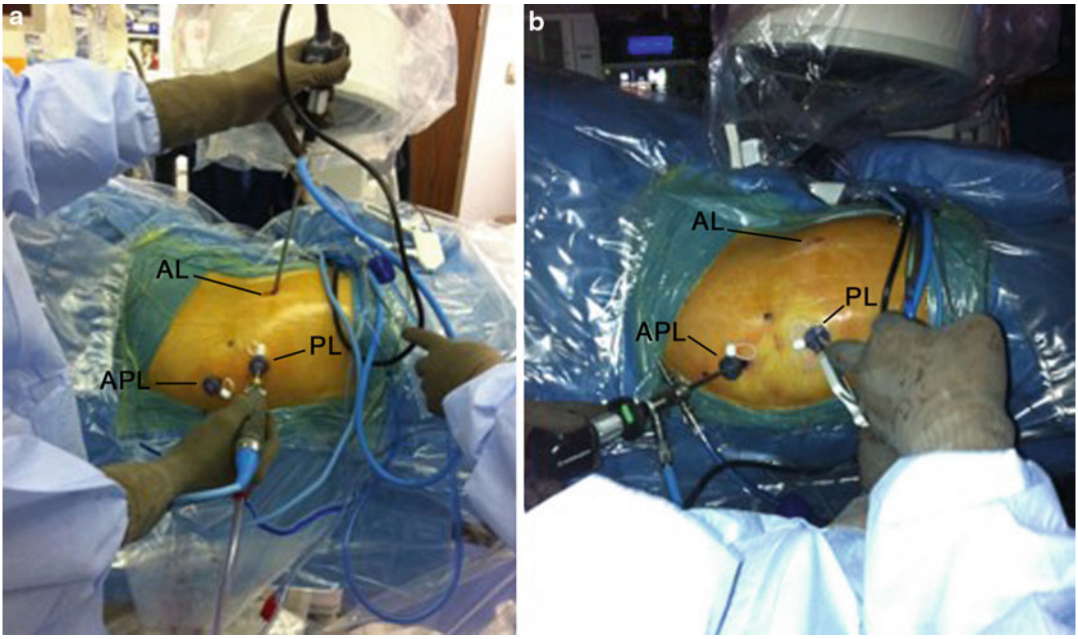


Fig. 1 Portal placement. (**a**) Use of long scopes from the anterolateral (*AL*) portal and shaver through the posterolateral (*PL*) portal. *APL* auxiliary posterolateral portal. (**b**) The arthroscope can be also positioned in the auxiliary posterolateral portal (*APL*) during the procedure (Reprint with permission TIO [19])

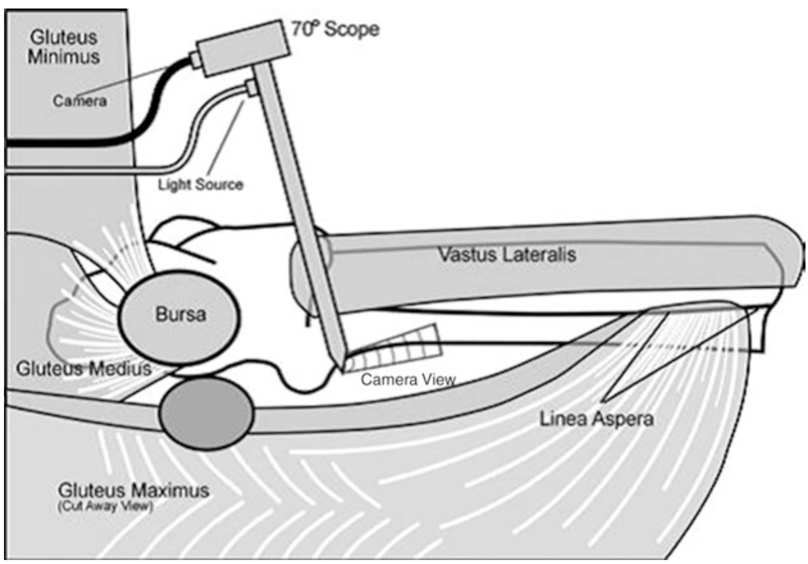


Fig. 2 Starting position and orientation. Establish orientation within the peritrochanteric space based upon anatomical landmarks. The 70° arthroscope is initially looking distal, oriented like a flag with the light cord proximal along with the lens focus distal (Reprint with permission from Slack Inc. [20])

long arthroscope with adjustable and lengthening cannulas is utilized [15]. The cannulas are opened to maintain the fluid flow, when utilizing the radiofrequency probe [18]. Fluid pressure is set to 60 mmHg with intermittent pressure increases up 80 mmHg. Three portals are utilized: anterolateral, posterolateral, and an auxiliary posterolateral portal positioned 3 cm posterior and 3 cm superior to the greater trochanter (Fig. 1) [15, 19]. Frequent use of intraoperative fluoroscopy will confirm the proper location of the endoscopic view.

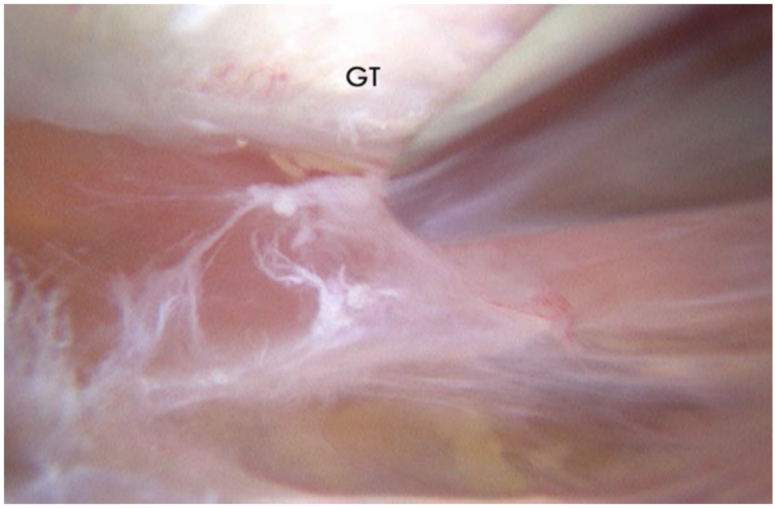


Fig. 3 The first step of the deep gluteal space endoscopy includes the greater trochanteric bursectomy. The bursa and fibrous bands are resected utilizing a shaver before identifying the quadratus femoris muscle. *GT* greater trochanter

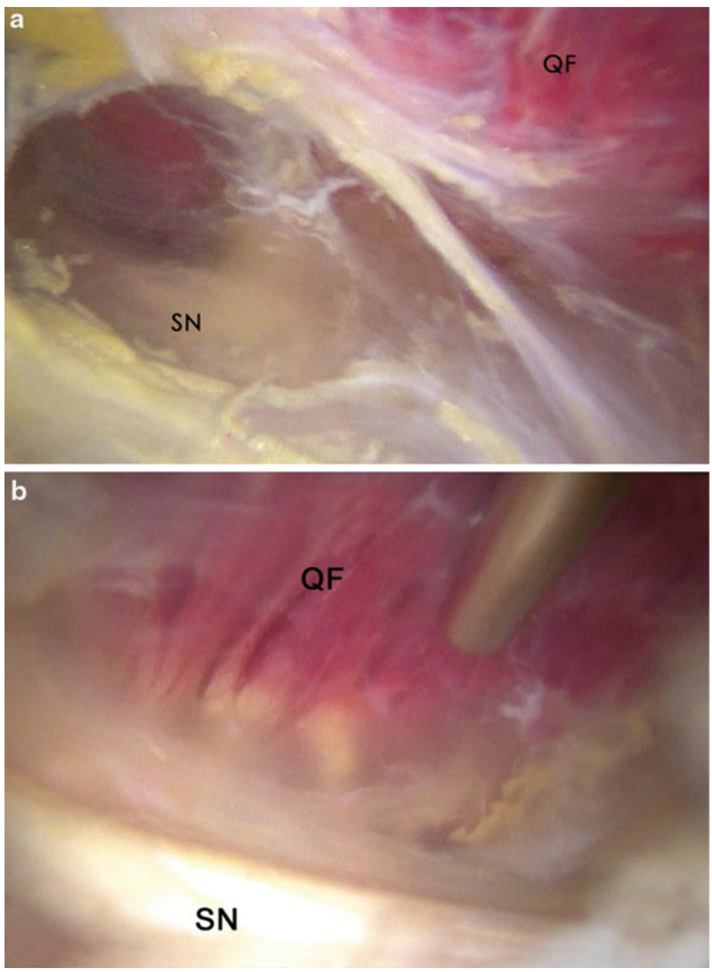


Fig. 4 Identification of the quadratus femoris muscle and sciatic nerve. (**a**) Internal rotation of the hip allows the visualization of the quadratus femoris muscle (*QF*) and sciatic nerve (*SN*). (**b**) Focused view of the sciatic nerve and quadratus femoris muscle

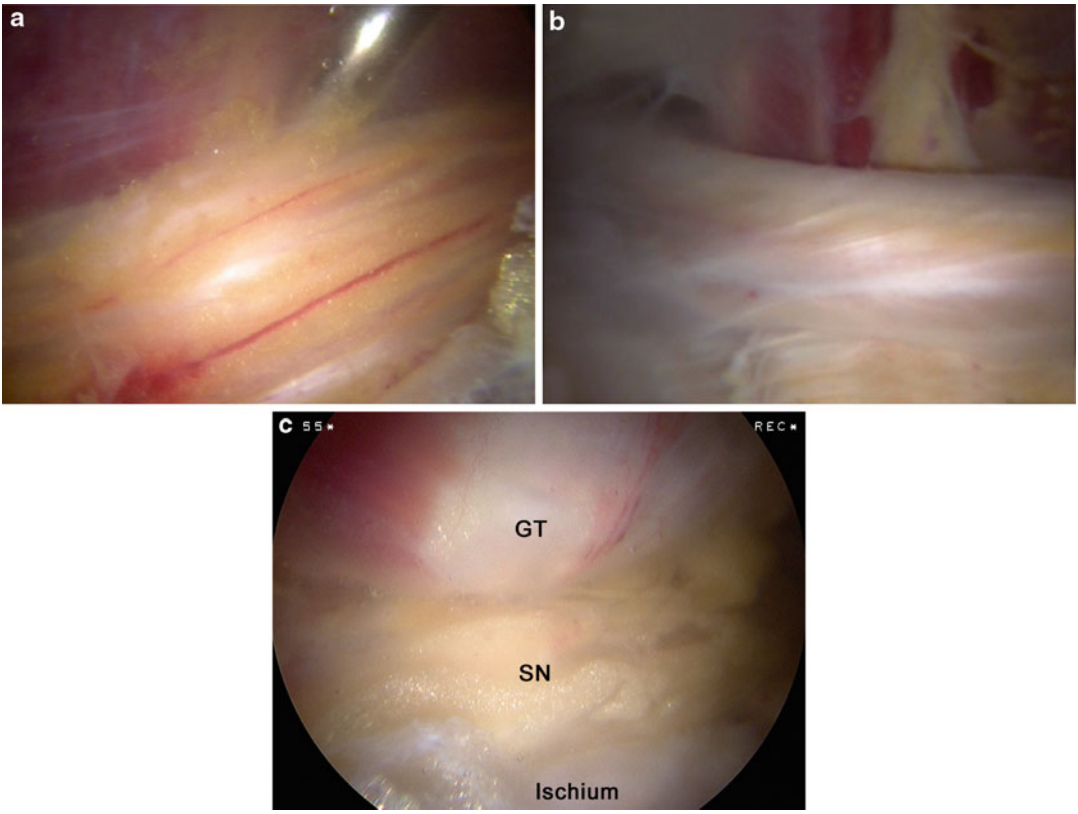


Fig. 5 Sciatic nerve inspection. (**a**) Normal sciatic nerve appearance with presence of blood flow and epineural fat. (**b**) Abnormal sciatic nerve with white shoestring appearance and no epineural fat. (**c**) Assessment of sciatic nerve (*SN*) compression between the greater trochanter (*GT*) and *ischium*

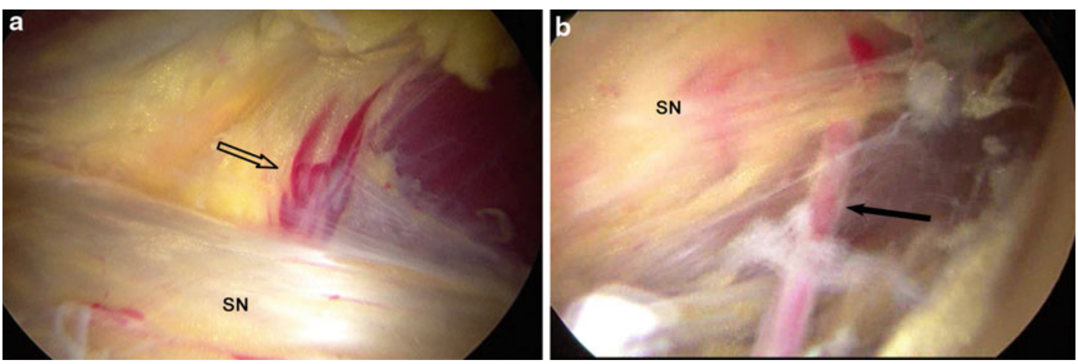


Fig. 6 Inspection of the vascular supply to the sciatic nerve. (**a**) Vascular supply (*open arrow*) reaching the sciatic nerve (*SN*) on its anterior surface. (**b**) Vessel accessing the sciatic nerve posteriorly (*arrow*)

A "10-step technique" allows for a complete sciatic nerve assessment and safe nerve decompression in the deep gluteal space. The starting position [20] is shown in Fig. 2, and once orientation has been established, the arthroscope is rotated to allow proximal viewing. **Step 1**: Inspect the peritrochanteric space and perform a greater trochanteric bursectomy [21], utilizing the anterolateral and posterolateral portals with the hip in neutral position (Fig. 3). Utilize the arthroscopic shaver for the bursectomy, releasing fibrous bands until the quadratus femoris is identified. Replace the shaver with the blunt probe and release any fibrous bands at the level of the quadratus femoris. **Step 2**: Identify the quadratus femoris muscle and the sciatic nerve with the hip in internal rotation (Fig. 4). **Step 3**: Evaluate the sciatic nerve color, epineural blood flow, epineural fat, and nerve motion. Normal sciatic nerve appearance (Fig. 5a) will have noticeable epineural blood flow and epineural fat and normal motion with internal/external rotation gliding along the border of the external rotator muscles. An abnormal sciatic nerve (Fig. 5b) will appear white resembling a shoestring and will not move with rotation and feel tight with probing. With deep hip flexion and external rotation, check for greater trochanteric impingement (Fig. 5c). In cases of sciatic nerve entrapment by the greater trochanter or ischium, greater trochanteric osteoplasty or osteotomy may be a

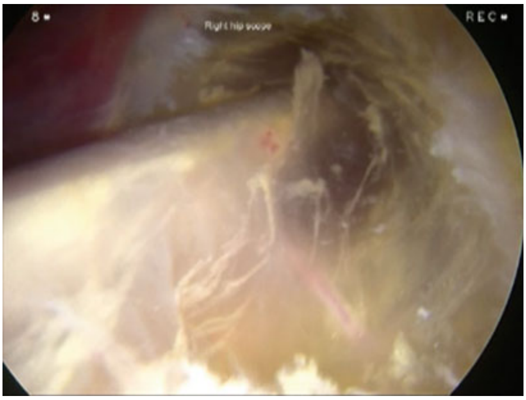


Fig. 7 Distal view of the deep gluteal space

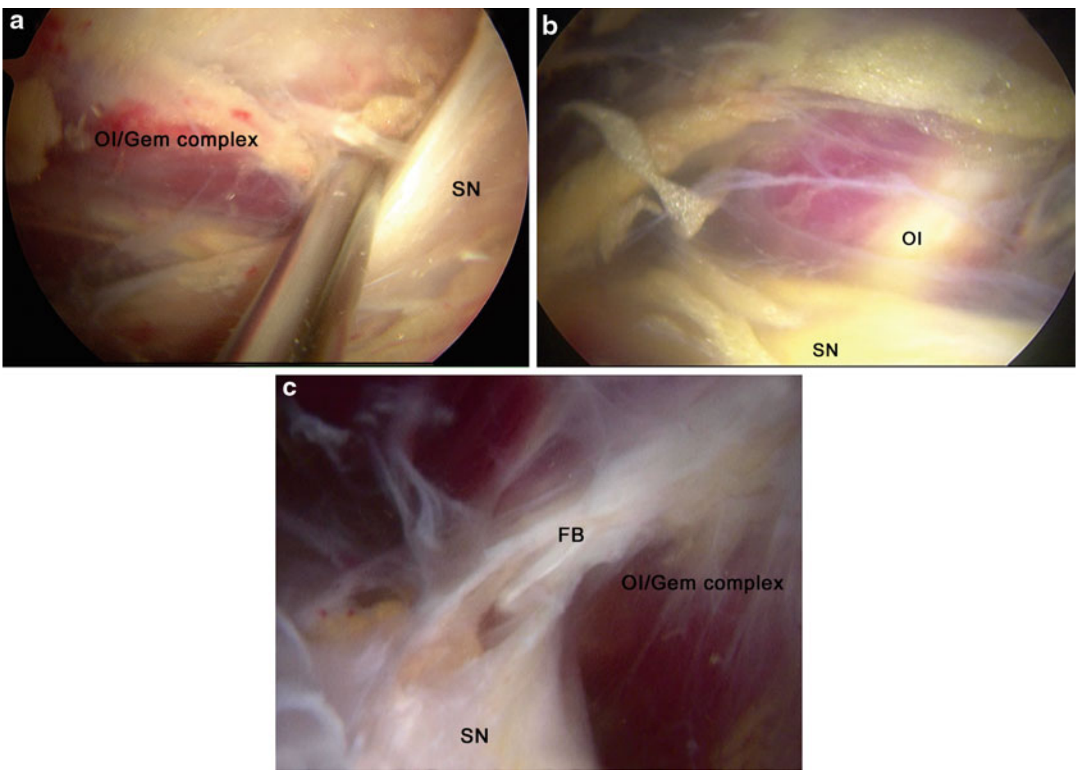


Fig. 8 Proximal view of the deep gluteal space. (**a**) The inspection of the sciatic nerve (*SN*) includes a gentle probing of the nerve. *OI/Gem*: obturator internus/gemellus. (**b**) Identification of the obturator internus tendon (*OI*). Sciatic nerve (*SN*) with normal appearance. (**c**) Abnormal sciatic nerve appearance at the level of the obturator Internus/gemellus (*OI/Gem*). *FB* fibrous scar band

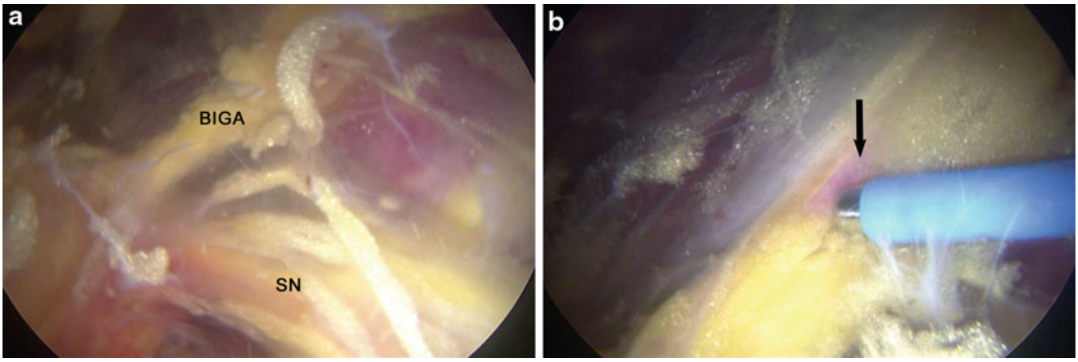


Fig. 9 (**a**) Proximal view with identification of a branch of the inferior gluteal artery (*BIGA*), which crosses posteriorly the sciatic nerve (*SN*). (**b**) Cauterization of the crossing branch (*arrow*) of the inferior gluteal artery utilizing a radiofrequency device

Table 2 Temperature assessed at the sciatic nerve after different times of continuous probe activation

Activation Time (sec)	Temperature in relation to the sciatic nerve	
	On the surface	In the perineurium
0	19.9 °C (range, 19–21 °C)	18.4 °C (range, 17–20 °C)
2	20.5 °C (range, 19–22 °C)	18.5 °C (range, 17–20 °C)
5	20.7 °C (range, 19–24 °C)	18.6 °C (range, 17–21 °C)
10	20.8 °C (range, 20–22 °C)	19.1 °C (range, 17–22 °C)

Reprint with permission from Arthroscopy [18]

Table 3 Fluid temperature assessed at the sciatic nerve at different intervals of distance and duration

Activation Time (sec)	Distance from the sciatic nerve		
	10 mm	5 mm	3 mm
0	20.6 °C (range, 20–21 °C)	20.6 °C (range, 20–21 °C)	20.6 °C (range, 20–21 °C)
2	21 °C (range, 20–22 °C)	21.6 °C (range, 20–23 °C)	21.8 °C (range, 21–23 °C)
5	21.8 °C (range, 21–24 °C)	22.3 °C (range, 21–25 °C)	22.8 °C (range, 21–25 °C)
10	21.8 °C (range, 21–23 °C)	22.6 °C (range, 21–24 °C)	23.7 °C (range, 22–28 °C)

Reprint with permission from Arthroscopy [18]

consideration. **Step 4**: Identify the vascular branches penetrating the sciatic nerve anteriorly just proximal to the quadratus femoris muscle (Fig. 6a). These branches originate from inferior gluteal artery, medial circumflex, and first perforating femoral artery and may sometimes reach the sciatic nerve on its posterior surface (Fig. 6b). **Step 5**: Endoscopic neurolysis of the sciatic nerve. This step requires gentle dissection of the fibrous bands in the area of the nerve along its course in the deep gluteal space. Utilize a blunt probe for dissection while probing the sciatic nerve to protect the perineural sheath. Some fibrous bands may extend down from the greater trochanteric bursa to the sciatic nerve [15]. Fibrovascular scar bands are often present, which will require cauterization prior to release, utilizing arthroscopic scissors. In cases when the fibrovascular bands are thick and clustered, resembling a bird's nest, combinations of cauterization and ligature may be necessary [15]. **Step 6**: Rotate the arthroscope for distal viewing and inspect the sciatic nerve at the ischial tunnel, hamstring origin, and sacrotuberous ligament (Fig. 7), releasing any fibers from the sciatic nerve. **Step 7**: Rotate the arthroscope for proximal viewing and identify the obturator internus muscle and tendon (Fig. 8). Release fibrous bands and probe and check for the relation between the sciatic nerve and obturator internus tendon, which may penetrate the sciatic nerve. **Step 8**: Move the long scope to the auxiliary or posterolateral portal when required (usually in larger patients). Identify the branch of

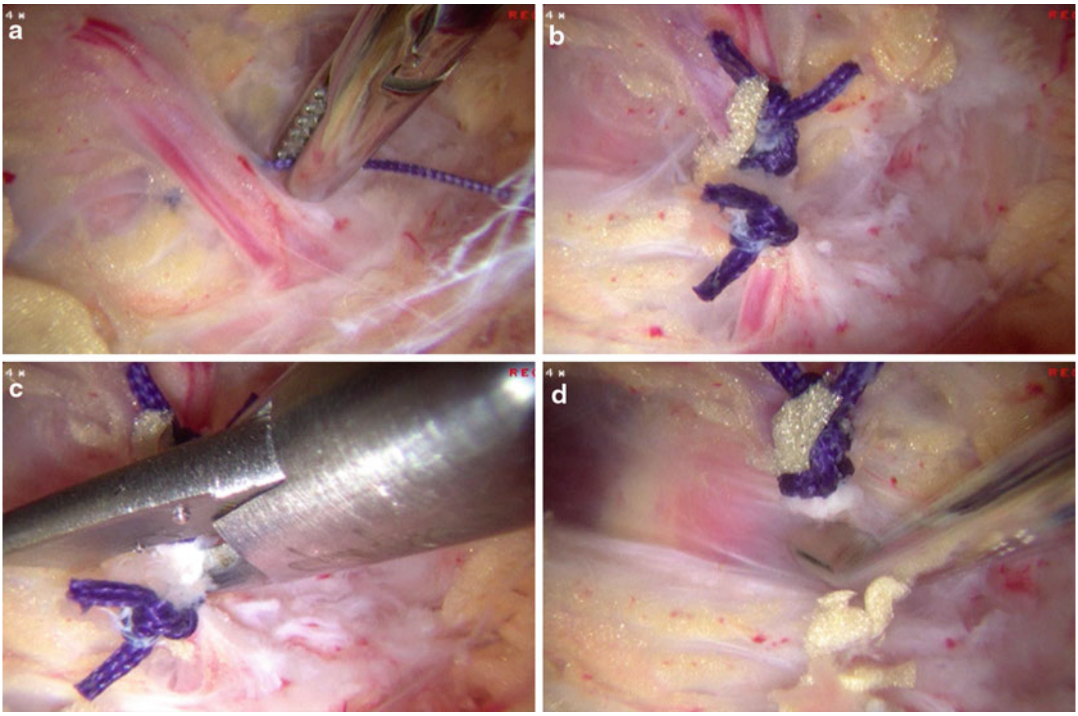


Fig. 10 Ligature of a vessel in a fibrovascular scar band. For vessels larger than 2 mm, a ligature is utilized rather than of long periods of radiofrequency activation

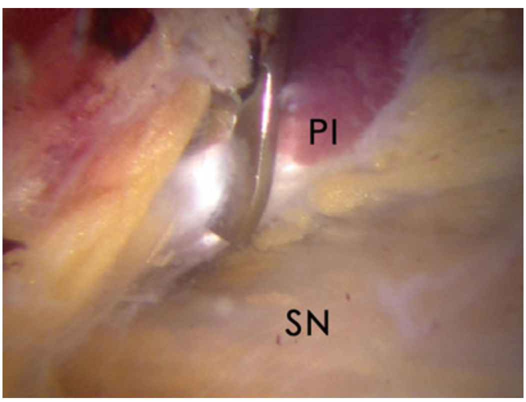


Fig. 11 Piriformis tendonotomy. *PI* piriformis muscle and tendon, *SN* sciatic nerve

the inferior gluteal artery, which crosses posterior to sciatic nerve distal to the border of the piriformis muscle. This branch must be cauterized or ligated (when larger than 2 mm using 4/0 PDS) and released before the inspection of the piriformis muscle and tendon (Fig. 9a, b). A recent study reported a mean distance of 8 mm (4–14 mm) between the sciatic nerve and the crossing branch of the inferior gluteal vessel [18]. The same study evaluated the fluid temperature during activation of a monopolar radiofrequency device around the sciatic nerve [18]. The temperature profile was safe to the sciatic nerve during the tested activation times of 3, 5, and 10 s (Tables 2 and 3). The standard approach to vessel cauterization is a? three-second interval of radiofrequency activation, maintaining continuous irrigation. Ten seconds is three times longer than standard activation time. For vessels larger than 2 mm, a ligature (Fig. 10) is utilized rather than longer activation time. **Step 9**: Identify and resect the piriformis muscle and tendon. The tendon is often hidden under the belly of the muscle which will require probing under the piriformis or shaving the distal border if necessary. The utilization of arthroscopic scissors for tendon release pulling the scissors toward you adds safety and ensures that only the tendon is released (Fig. 11). Any time the shaver or scissors are utilized in the vicinity of the sciatic nerve, a curved probe can be used for sciatic nerve retraction and safety. Following tendon resection, shave the tendinous stump back 1–2 cm. **Step 10**: Probe the sciatic nerve for tension and look for hidden muscle or tendon branches traversing the nerve. Perform the dynamic testing of sciatic nerve kinematics probing the nerve during hip internal and external rotation with flexion and extension. Cautiously probe the sciatic nerve up to the sciatic notch and confirm the location with fluoroscopy. Carefully note that the superior gluteal neurovascular structures exit the sciatic notch superior to the piriformis muscle. Using the curved probe, thoroughly explore the retrosciatic region to identify and release any ancillary musculotendinous branches that may be binding the nerve (Fig. 12). Internal and external rotation of the hip is helpful to identify these branches. Any restriction of nerve motion should suggest a penetrating branch, intrapelvic entrapment, or osseous abnormality.

A standardized technique and surgical experience for diagnosis and sciatic nerve decompression are mandatory in order to identify the sciatic nerve anatomy, to avoid iatrogenic injury, and to not overlook potential sources of sciatic nerve entrapment.

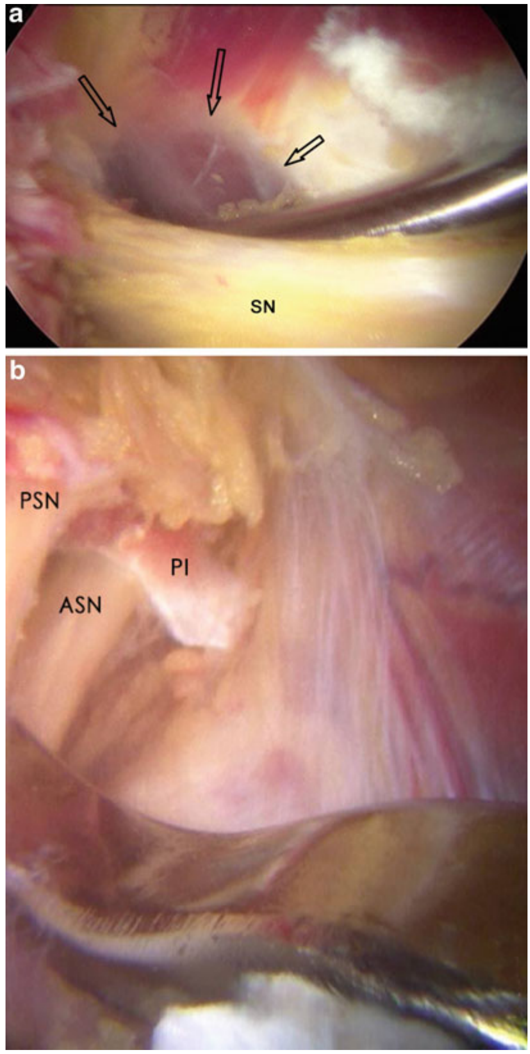


Fig. 12 (**a**) Cautious palpation of the sciatic nerve (SN) at the sciatic notch (*open arrows*) utilizing fluoroscopic guidance to confirm location. (**b**) Ancillary musculotendinous branch through the sciatic nerve identified after the resection of the posterior head of the piriformis muscle

Physical Therapy

Critical to the outcomes of endoscopic sciatic nerve decompression is the physical therapy rehabilitation and patient compliance. The goal of rehabilitation is to gain mobility and maintain movement of the hip joint. Patients feel much better after surgery and may want to take an aggressive approach regarding stretching and rehabilitation. However, stretch injury can cause neuropraxia and neuralgia. Six to twelve percentage increased in nerve stretch can cause decreased nerve conduction [22, 23]. To avoid overstretch injury, the Ilizarov osteogenesis principles of limb lengthening can be applied to rehabilitation by a slow progression of increased stretching. Kinematic motion of the sciatic nerve with knee flexion is different than with knee extension. This factor is important to understand in physical therapy principles. The following is an outline for postoperative sciatic nerve decompression rehabilitation.

Full circumduction of the hip with knee flexion can begin on day one. A knee brace is used to avoid knee extension and maintain a relaxed sciatic nerve when necessary. The utilization of the knee brace is dependent upon the strain of the sciatic nerve, which is influenced by degree of femoral anteversion and the number of sites of

Fig. 13 Rehabilitation stretching exercises. (**a**) The piriformis stretch is performed in the seated position. The patient brings the knee toward the opposite shoulder. (**b**) Sciatic nerve glides: the patient first performs cervical extension and plantar flexion of the ankle followed by cervical flexion with ankle dorsiflexion. (**c**) Hip circumduction: performed in supine position with gentle passive circular movements, with knee and ankle parallel to the body longitudinal axis (avoiding hip rotation)

entrapment. If increased tension is noticed postoperatively, the knee is locked at 45° for 3 weeks applying only nerve glides and circumduction. After week four, increase up to 10° of knee extension every two weeks as tolerated. Maintain circumduction, gentle nerve glides, and include stretching maneuvers aimed at the external rotators (Fig. 13). The piriformis stretch involves placing the hip in flexion, adduction, and internal rotation. In a seated position, the patient brings the knee into the chest and across midline and pulls the knee to the opposite shoulder for 20 seconds. Gradually progress the stretching by increasing duration and intensity until a maximal stretch is obtained. Standard physical therapy protocol can begin as early as 4 weeks. Again, a word of caution in cases of previous abdominal surgery and femoral retroversion as strain parameters will be a dependent factor and the nerve may be impinged in more than one location. The therapist should be diligent in recognizing these potential outcome factors.

The use of steroidal and nonsteroidal anti-inflammatories has been found useful after day two. Additional physical therapy techniques may be helpful including ultrasound and electrical stimulation. With a proper rehabilitation protocol, good to excellent outcomes can be achieved. The advancement in rehabilitation may prove beneficial to improve the outcomes of endoscopic sciatic nerve decompression in some patients.

Results

Martin et al. reported on a case series of 35 patients presenting with deep gluteal syndrome [15]. Average duration of symptoms was 3.7 years with an average preoperative verbal analog score of 7, which decreased to 2.4 postoperatively. Preoperative modified Harris Hip Score was 54.4 and increased to 78 postoperatively. Twenty-one patients reported preoperative use of narcotics for pain; 2 remained on narcotics postoperatively (unrelated to initial complaint). Eighty-three percent of patients had no postoperative sciatic sit pain (inability to sit for >30 min) [15]. Five patients experienced low mHHS scores and modest pain relief postoperatively. This poor outcome group is shown in Table 4 and may be related to femoral retroversion and previous abdominal surgery.

Table 4 Characteristics of five low-improvement cases

Case no.	Gender	Trauma	DOS (mo)	Surgical history	MHHS pre	MHHS post	BOR	VAS pre	VAS post	NSA	FV	PL-A portal	Sciatic nerve entrapment
1	F	No	16	Laparoscopy, breast	53.9	61.6	Poor	6	8	130	1	No	SN was completely bound down in scar tissue beneath the gluteus maximus and laid on top of the piriformis muscle, hypovascular in appearance
7	F	Yes	51	Hysterectomy, spinal fusion, appendectomy	53.9	44.0	Poor	7	5.5	139	18	No	Thick scar tissue on the anterior and posterior aspect of the ischium, piriformis was involved, significant adhesions proximally and distally, fibrinous lesion over the top of the SN, adhesed on the surface of the ischium
19	F	No	6	Hip arthroscopy, gall bladder	54.0	64.9	N/A	6	5	133	22	No	Large fibrinous bands, piriformis tendon was involved
23	M	Yes	10	None	35.2	46.2	Fair	10	5	134	−1	Yes	GT bursal hypertrophy, gluteus medius tear, the SN had significant scarring and a posteriorly occurring inferior gluteal branch which tethered the SN
28	M	Yes	10	Jaw, knee, tonsillectomy	35.0	57.2	Poor	10	4	142	9	Yes	Massive amount of scar tissue proximal to the quadratus femoris extending up proximal to mid hip, thickened piriformis tendon, adhesion of the entire proximal aspect of SN, hypovascular, the SN tethered along proximal border of the quadratus up into the piriformis

Note. Case numbers are presented in chronological order. *DOS* duration of symptoms, *BOR* Benson outcome rating, *VAS* verbal analog scale, *NSA* neck shaft angle, *FV* femoral version, *PL-A* portal, posterolateral auxiliary, *SN* sciatic nerve, *GT* greater trochanter (Reprint with permission from Martin et al. [18])

Now, among 200 national and international cases, complications continue to be extremely low. It is very important to assess acetabular and femoral version which has an effect on sciatic nerve biomechanics. To help avoid postsurgical stretch injury, it is recommended that intra-articular work be performed separately from extra-articular work. Due to the length of time from diagnosis to treatment and recovery, the psychological toll of the pain cycle can be frustrating. Psychological testing can be helpful pre and post-operatively. Participation in a pain reinforcing group is not recommended and may be a negative outcome predictor. Complications have involved hematoma brought on by early postoperative use of NSAIDs with excessive postoperative activity. Concomitant pudendal nerve and sciatic nerve complaints are often resolved; however, in two cases the pudendal complaints worsened.

Summary

The "10-step technique" for the deep gluteal space provides a standardized approach to sciatic nerve assessment and decompression. This endoscopic approach appears useful in detecting sciatic nerve pathology and treatment, and further studies are underway. By understanding the anatomy and biomechanics and applying clinical tests and diagnostic strategies, adequate treatment of all four layers can be obtained as a part of a comprehensive plan of treatment and rehabilitation.

References

1. Papadopoulos EC, Khan SN. Piriformis syndrome and low back pain: a new classification and review of the literature. Orthop Clin North Am. 2004;35:65–71.
2. Filler AG, Haynes J, Jordan SE, Prager J, Villablanca JP, Farahani K, McBride DQ, Tsuruda JS, Morisoli B, Batzdorf U, Johnson JP. Sciatica of nondisc origin and piriformis syndrome: diagnosis by magnetic resonance neurography and interventional magnetic resonance imaging with outcome study of resulting treatment. J Neurosurg Spine. 2005;2:99–115.
3. Vandertop WP, Bosma NJ. The piriformis syndrome. A case report. J Bone Joint Surg Am. 1991;73:1095–7.
4. Benson ER, Schutzer SF. Posttraumatic piriformis syndrome: diagnosis and results of operative treatment. J Bone Joint Surg Am. 1999;81:941–9.
5. Young IJ, van Riet RP, Bell SN. Surgical release for proximal hamstring syndrome. Am J Sports Med. 2008;36:2372–8.
6. Miller A, Stedman GH, Beisaw NE, Gross PT. Sciatica caused by an avulsion fracture of the ischial tuberosity. A case report. J Bone Joint Surg Am. 1987;69:143–5.
7. Chen WS. Sciatica due to piriformis pyomyositis. Report of a case. J Bone Joint Surg Am. 1992; 74:1546–8.
8. Hughes SS, Goldstein MN, Hicks DG, Pellegrini Jr VD. Extrapelvic compression of the sciatic nerve. An unusual cause of pain about the hip: report of five cases. J Bone Joint Surg Am. 1992;74:1553–9.
9. Sayson SC, Ducey JP, Maybrey JB, Wesley RL, Vermilion D. Sciatic entrapment neuropathy associated with an anomalous piriformis muscle. Pain. 1994; 59:149–52.
10. Meknas K, Kartus J, Letto JI, Christensen A, Johansen O. Surgical release of the internal obturator tendon for the treatment of retro-trochanteric pain syndrome: a prospective randomized study, with long-term follow-up. Knee Surg Sports Traumatol Arthrosc. 2009; 17:1249–56.
11. Lewis AM, Layzer R, Engstrom JW, Barbaro NM, Chin CT. Magnetic resonance neurography in extraspinal sciatica. Arch Neurol. 2006;63:1469–72.
12. Issack PS, Kreshak J, Klinger CE, Toro JB, Buly RL, Helfet DL. Sciatic nerve release following fracture or reconstructive surgery of the acetabulum. Surgical technique. J Bone Joint Surg Am. 2008;90(Suppl 2 Pt 2): 227–37.
13. Beauchesne RP, Schutzer SF. Myositis ossificans of the piriformis muscle: an unusual cause of piriformis syndrome. A case report. J Bone Joint Surg Am. 1997; 79:906–10.
14. Jones HG, Sarasin SM, Jones SA, Mullaney P. Acetabular paralabral cyst as a rare cause of sciatica. A case report. J Bone Joint Surg Am. 2009;91:2696–9.
15. Martin HD, Shears SA, Johnson JC, Smathers AM, Palmer IJ. The endoscopic treatment of sciatic nerve entrapment/deep gluteal syndrome. Arthroscopy. 2011;27:172–81.
16. Dezawa A, Kusano S, Miki H. Arthroscopic release of the piriformis muscle under local anesthesia for piriformis syndrome. Arthroscopy. 2003;19:554–7.
17. Polesello GC, Rosa JM, Queiroz MC, Honda EK, Guimarães RP, Junior WR. Deep gluteal pain: common problem in the office – review of literature and report of endoscopic treatment of 3 cases. Rev Bras Ortop. 2011;46:56–63.
18. Martin HD, Palmer IJ, Hatem M. Monopolar radiofrequency usage in deep gluteal space endoscopy: sciatic nerve safety and fluid temperature. Arthroscopy. 2014;30(1):60–4.

19. Martin HD, Hatem MA, Champlin K, Palmer IJ. The endoscopic treatment of sciatic nerve entrapment/deep gluteal syndrome. Tech Orthop. 2012;27:172–83.
20. Martin H. Diagnostic arthroscopy. In: Kelly BT, Philippon MJ, editors. Arthroscopic techniques of the hip: a visual guide. Thorofare: Slack; 2009.
21. Voos JE, Rudzki JR, Shindle MK, Martin H, Kelly BT. Arthroscopic anatomy and surgical techniques for peritrochanteric space disorders in the hip. Arthroscopy. 2007;23:1246.e1–5.
22. Coppieters MW, Alshami AM, Babri AS, Souvlis T, Kippers V, Hodges PW. Strain and excursion of the sciatic, tibial, and plantar nerves during a modified straight leg raising test. J Orthop Res. 2006;24:1883–9.
23. Wall EJ, Massie JB, Kwan MK, Rydevik BL, Myers RR, Garfin SR. Experimental stretch neuropathy. Changes in nerve conduction under tension. J Bone Joint Surg Br. 1992;74:126–9.

Part VI

Hip Instability and Fractures

76 Traumatic Instability: Acute and Delayed Management

J. W. Thomas Byrd and G. Peter Maiers II

Contents

Abstract

Sports-related traumatic instability of the hip is generally the result of a low-velocity mechanism compared to vehicular trauma. Most are posteriorly directed because of the typical mechanisms of injury. Anterior instability is less common and more frequently associated with atraumatic instability. Femoroacetabular impingement (FAI) morphology can create a fulcrum, placing the joint at a mechanical disadvantage and making it more susceptible to posterior instability. Following traumatic subluxations, the symptoms are sometimes not severely disabling and may belie the magnitude of injury. Thus, it is important to be keen in interpreting radiographs when looking for posterior rim fractures and performing appropriate diagnostic studies when there is an index of suspicion.

Most cases of traumatic instability can be treated conservatively with a high likelihood of returning to athletic endeavors. The presence of entrapped fragments, a nonconcentric reduction, or recurrent instability may force early surgical intervention. For cases treated conservatively, 3 or 4 months may be necessary for resolution of the acute injury phase. By then, if mechanical symptoms persist, this may indicate a role for arthroscopy. Standardized methodologies have been developed for the management of these injuries.

J.W.T. Byrd (✉)
Nashville Sports Medicine Foundation, Nashville, TN, USA
e-mail: info@nsmfoundation.org

G.P. Maiers II
Department of Orthopaedic Surgery, Indiana University School of Medicine, Indianapolis, IN, USA
e-mail: gpmaiers@gmail.com

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_81

Introduction

Sports-related traumatic instability of the hip is a relatively low-velocity injury compared to high-velocity vehicular trauma [1]. Thus, the characteristics may be different.

Most are posteriorly directed because this is the most common mechanism of injury [1]. Among these traumatic posterior instability problems, subluxations are most common with or without an accompanying fracture of the posterior rim.

Less frequent are dislocations that require emergent reduction [2]. Preexisting femoroacetabular impingement (FAI) morphology, including both pincer and cam, is often found in association with traumatic instability [3]. It is postulated that the FAI predisposes the joint to posterior instability [4]. With forced flexion, as the impingement lesions collide anteriorly, it creates a fulcrum, levering the femoral head posteriorly. However, it must also be kept in perspective that there is a high normal prevalence of FAI morphology in many athletic populations [5, 6].

Anteriorly directed instability is less common. This is often associated with dysplasia and activities that demand supraphysiological joint motion such as gymnastics, dance, and figure skating. Stress on the anterior capsulolabral structures has been associated with an increasing McKibbin's index, which is a summation of the femoral and acetabular anteversion [7]. Other osseous risk factors of instability include increasing neck shaft angle and diminishing center edge (CE) angle and acetabular index [8]. These considerations are more commonly associated with atraumatic instability [9].

Inherent joint stability is primarily provided by bony architecture of the hip [10]. It exhibits constrained joint architecture, but some normal translation occurs [11]. It is certainly more constrained than the shoulder, which is inherently unstable. There are numerous secondary stabilizers. The capsule is an important static stabilizer, especially the iliofemoral ligament [12]. The labrum is another static stabilizer [13]. It contributes very little to mechanical stability of the joint but has an important hydraulic effect through its labral seal [14–16]. As the morphology of the labrum is variable, its contribution may vary as well. It is generally perceived that the ligamentum teres does not contribute to joint stability but, in some circumstances, it may have some secondary stabilizing function [8]. Dynamic stability is provided by the surrounding musculature [17]. This is an especially important consideration in rehabilitation following injury or surgery [18].

Mechanism of Injury

The most common mechanism for posterior instability is axial loading of the flexed hip, usually with the knee flexed [1]. This is commonly encountered, for example, with tackling in football. However, this is often observed as a noncontact injury, such as planting the foot for rapid deceleration with the trunk being thrown forward axially loading the flexed hip with the knee extended [19]. Anterior instability occurs with extension and a component of external rotation [2].

Evaluation

History

The athlete should be quizzed about the mechanism of injury. Often, they will describe signs of hip irritability characterized by groin or anterolateral hip pain. Posterior discomfort may variably be present in association with injury to the posterior soft tissues or the posterior acetabular rim.

Even with significant subluxation episodes, or subluxation with accompanying fracture of the posterior rim, the symptoms may not be entirely disabling [1, 19]. Occasionally, athletes will actually return to play or, at least, attempt to return. The seemingly modest level of accompanying dysfunction may belie the seriousness and the magnitude of the injury. Severe symptoms will trigger a more thorough evaluation, but sometimes it is simply persistence of discomfort that precipitates the evaluation.

Physical Exam

Gait should be inspected, looking for a limp. Findings of hip joint irritability or apprehension are assessed. Commonly, flexion with internal rotation will be painful. Axial loading of the flexed hip may elicit posterior discomfort accompanying a posterior instability episode. Conversely, forced abduction with external rotation may be helpful for assessing anterior instability.

Imaging

Plain radiographs should be obtained in any case of suspected traumatic instability. Assessment should be made for a concentrically reduced joint with no evidence of joint space widening or entrapped fragment. Assessment is also made for any accompanying fracture. It is important to make sure that the radiographs are of adequate quality. For example, films obtained at a stadium can sometimes be suboptimal. Posterior rim fractures can be inadvertently overlooked simply because the athlete's level of function may not suggest such an injury.

MRI is sensitive at detecting accompanying soft tissue injury and signs of a significant subluxation episode. However, posterior rim fractures can be incorrectly interpreted as posterior labral detachments (Fig. 1). The bone is cortical and avascular and, thus, does not demonstrate any bony edema, making it difficult to discern as a fracture. High-resolution imaging with proper sequencing is necessary to search for other labral pathology, chondral injury, or intra-articular debris.

Computed tomography (CT) can confirm the presence of a fracture and may be helpful for assessing the congruency of joint space reduction and some intra-articular fragments.

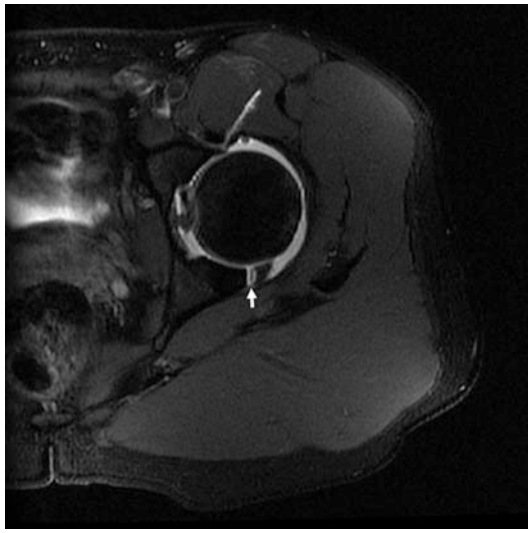


Fig. 1 MRA image of a left hip reveals disruption of the posterior acetabulum (*arrow*). This was initially interpreted as a posterior labral tear but actually represents a posterior rim fracture (©2014 J. W. Thomas Byrd, MD)

Treatment

Nonoperative Management

Most traumatic instability can be treated nonoperatively. Of course, dislocations require emergent reduction to minimize the risk of developing avascular necrosis of the femoral head. Unless there is a compelling reason for arthroscopic intervention, such as a nonconcentric reduction or obvious entrapped fragment, an initial trial of conservative treatment is preferable. Three months is a reasonable time frame for resolution of the acute phase of the injury and a clearer assessment of whether an athlete continues to have mechanical symptoms or clinical findings that may warrant arthroscopic intervention. In addition to subsidence of acute symptoms, this is an opportunity for some intra-articular damage to declare itself. For example, impaction injury to the articular surface of the femoral head may lead to subsequent chondrocyte cell death and articular delamination that may not be evidently close to the time of the initial injury. Arthroscopy, at a later date, may provide a clearer reflection of the severity of intra-articular pathology. Often the injuries heal, arthroscopy is not necessary, and athletes can resume sporting activities without symptoms. Long-term consequences may still be less certain, and thus athletes are educated about warning signs of symptoms that might warrant reevaluation in the future.

The presence of a posterior rim fracture does little to alter the treatment strategy, or likely outcomes, of posterior subluxation episodes. The fracture may only partially heal or develop a fibrous union, but this is rarely a source of residual symptoms or cause for surgical intervention. Surgery is usually necessitated due to other intra-articular pathology.

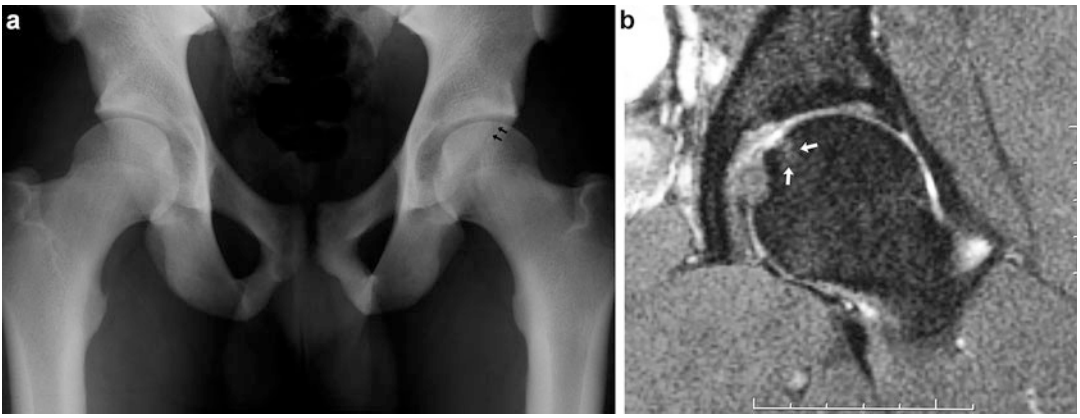


Fig. 2 A 20-year-old NFL rookie defensive back sustained an acute injury to his left hip in the first quarter of a game. He completed the game and then was evaluated for persistent pain. (**a**). AP radiograph reveals a posterior rim fracture (*arrows*). (**b**). Follow-up MRI at 6 weeks reveals localized superomedial area of subchondral edema in the femoral head on T2 coronal images indicative of concomitant impaction injury to the femoral head. This had no residual consequences as he returned to play at 7 weeks post-injury and has had a 10+-year career including several Pro Bowl appearances (©2014 J. W. Thomas Byrd, MD)

Fig. 3 A 31-year-old NFL wide receiver sustained an acute injury to his left hip. (**a**). 3D-CT image reveals a small posterior rim fracture (*arrows*). (**b**). However, within 3 weeks, an AP radiograph shows evidence of early joint space narrowing of the left compared to the uninjured right hip. (**c**). Follow-up coronal MRI shows significant subchondral edema of the femoral head with concomitant articular loss and persistent pronounced effusion. He developed quickly progressive posttraumatic osteoarthritis culminating in a resurfacing arthroplasty 1 year following his index injury (©2014 J. W. Thomas Byrd, MD)

Table 1 Hip Luxation Protocol

Hip Luxation Protocol (Stable Posterior Acetabular Fracture)
Initial x-rays, CT scan & MRI (high-resolution!)
Stable fracture pattern without obvious major intraarticular damage, then candidate for conservative treatment
Crutches, partial WB 4 weeks
Minimize risk of fracture displacement
Slight loading stimulates healing process
At 4 weeks fracture should be "sticky"
4 weeks
Limited CT scan
Check for signs of displacement
Alignment good & painfree, then transition off crutches
Follow-up MRI
Check for resolution of soft tissue edema
Check for early signs of subchondral femoral edema (bone bruise) indicative of FH impaction injury
May necessitate more conservative approach because of uncertain prognostic significance
4–6 weeks, avoid loading of flexed hip
Functional progression at 6 weeks, if pain-free
Return to unrestricted activities 8–10 weeks, if pain-free
F/U x-rays at 1, 2, 3 & 6 months
F/U MRI 3 & 6 months
Assess for early signs of AVN
Assess for progression/resolution of subchondral edema, if present
F/U CT scan, if needed due to symptoms
Bony bridging of fracture often partial/incomplete
May develop asymptomatic fibrous union

Most patients with posterior subluxations recover well and can resume athletic activities (Fig. 2). However, some do poorly, with rapidly progressive joint deterioration, and can be facing an arthroplasty even within a year of the index injury (Fig. 3). There are few indicators to predict which will do well and which may rapidly decline. Thus, a deliberate, responsible rehab strategy is important for all cases. A protocol is outlined in Table 1. Protective weight bearing is maintained for 4 weeks, during which time hip flexion is limited to 90°. A follow-up MRI is performed at 1 month, looking for resolution of soft tissue edema and any joint effusion. Subchondral edema may start to develop in the femoral head as an indication of the impaction injury to the joint surface. This is common and is not necessarily a harbinger of poor results or an indication of early AVN [19]. In the cases of posterior rim fractures, a follow-up CT scan can be helpful just to assure that no further displacement has occurred. Loaded hip flexion is avoided until 6 weeks, and then the athlete is progressed with a functional program as symptoms allow. Return to unrestricted activities at 8–10 weeks is not unreasonable, as long as the athlete remains pain-free. A later MRI may be obtained to rule out signs of late onset avascular necrosis.

Acute Surgical Management

There are many complex fractures that may warrant early ORIF, but that is not the subject of this chapter. Early arthroscopy may be indicated for a nonconcentric reduction or entrapped fragments (Fig. 4). Commonly, arthroscopy will reveal more extensive damage than that identified by the imaging. This tendency of MRI to underestimate the damage should be kept in mind, as some athletes who fail to respond to conservative care may have more occult joint pathology. The timing of surgery should also take into consideration concerns about soft tissue extravasation. Normally, the risk of extravasation should not be any greater than that encountered in association with other extensive arthroscopic procedures to correct FAI that often involve large capsular exposures. Early arthroscopy should be avoided when there is an acetabular fracture that communicates with the intrapelvic region [20].

Posterior labral detachments are common but rarely require repair. When observed in conjunction with early arthroscopy, repair may make sense if it can be performed without inordinately adding to the length of the procedure (Fig. 5). Additionally, since many cases of traumatic instability demonstrate underlying FAI, associated pathology may be encountered including anterior labral tearing or articular delamination [3]. With

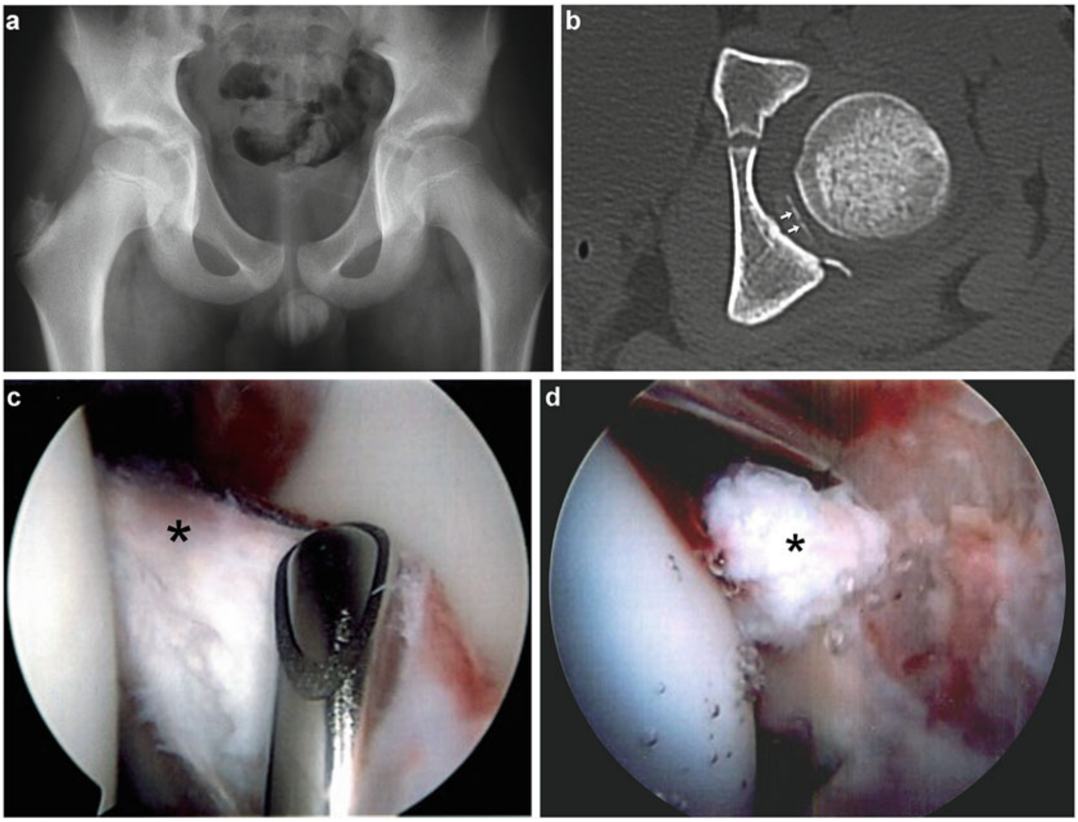


Fig. 4 A 10-year-old male sustained an acute left hip injury when involved in a pileup during a football game. Subsequently, he was unable to bear weight because of pain. (**a**). AP radiograph reveals open physes and a seemingly concentric reduction. (**b**). CT scan reveals an entrapped fragment (*arrows*). (**c**). Arthroscopic view from the anterolateral portal reveals the fragment (*asterisk*). This was attached to the posterior capsule, which was also entrapped within the joint. The fragment was excised and capsule reduced with an uneventful recovery. (**d**). Also noted was a rupture of the ligamentum teres (*asterisk*) which was debrided (©2014 J. W. Thomas Byrd, MD)

these observations, a decision must be made on whether to correct the preexisting FAI. This determination is often individualized to the athlete's circumstances. For example, on close questioning, some athletes may express having experienced joint symptoms prior to their acute traumatic episode.

Delayed Surgical Management

With delayed surgical management, the course of treatment is usually clearer. The extent of joint damage has declared itself, and the source of symptoms is usually more evident. If athletes continue to have posterior symptoms or a sense of subluxation, the need for a Bankart-type repair of posterior labral detachment is more evident (Fig. 6). Articular damage is addressed as indicated with chondroplasty or microfracture for Grade IV lesions. FAI can be present as an incidental finding but, if anterior acetabular pathology indicative of problematic FAI is present, it may warrant bony correction. A stump of the ligamentum teres can sometimes be a source of mechanical symptoms, and previously unrecognized intra-articular fragments or debris may be identified [21]. The peripheral compartment must be thoroughly inspected for fragments that can be a source of symptoms, undetected by arthroscopy of the central compartment alone (Fig. 7).

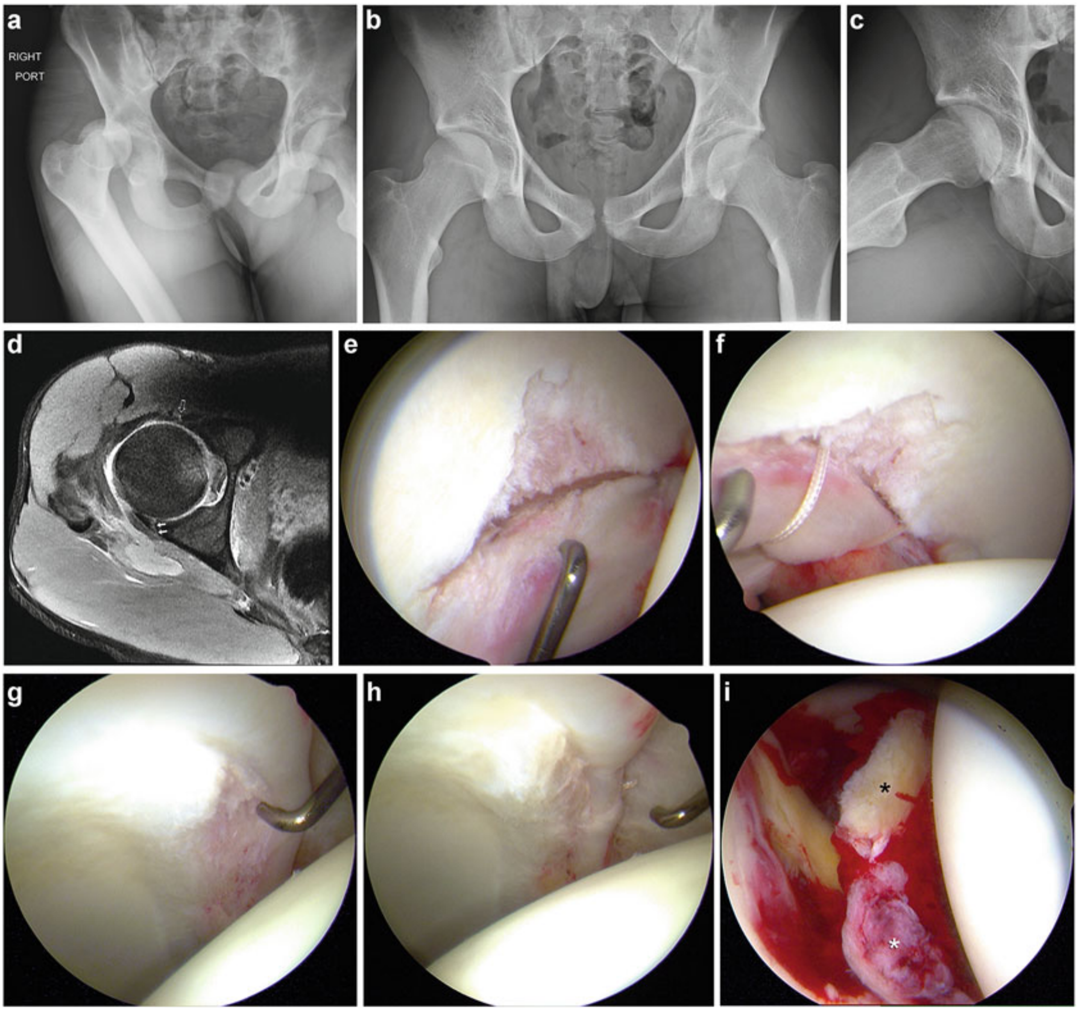


Fig. 5 An 18-year-old high school quarterback sustained a posterior hip dislocation getting tackled, with several players piling on top. (**a**). A single AP radiograph reveals the posterior dislocation for which he underwent prompt closed reduction. (**b**, **c**). Subsequent AP and lateral radiographs demonstrate a concentric reduction, but he continued to have symptoms of posterior instability despite normal precautions. (**d**). Follow-up MRI two and a half weeks post-injury reveals a posterior Bankart-type labral tear (*white arrows*) as well as increased signal in the anterior labrum (*open arrow*) on this axial image. Because of uncontrollable symptomatic instability, he underwent arthroscopic surgery. Views are from the anterolateral portal. (**e**). Viewing posteriorly, the detached labrum is probed from the posterolateral portal along with an adjacent chondral defect in the posterior rim. (**f**). The labrum was repaired with two suture anchors using simple loop sutures to restore and bolster the labrum to the posterior rim. (**g**). Viewing anteriorly, a separate detachment of the anterior labrum is probed from the anterior portal. (**h**). This was repaired with two suture anchors using horizontal mattress sutures to anatomically reapproximate the labrum. (**i**). Viewing medially, also noted was a rupture of the ligamentum teres (*white asterisk*) with attached articular fragment (*black asterisk*) which was excised to prevent this from creating mechanical symptoms. The athlete subsequently returned uneventfully to high school and then intercollegiate sports (©2014 J. W. Thomas Byrd, MD)

For anterior instability, the pathophysiology, associated pathology, and strategy of treatment may be completely different [9, 17]. Careful assessment for dysplasia and insufficient bony stability must be made. This cannot be corrected with arthroscopic intervention and may necessitate a PAO to restore stability. Accounting must be given to all secondary

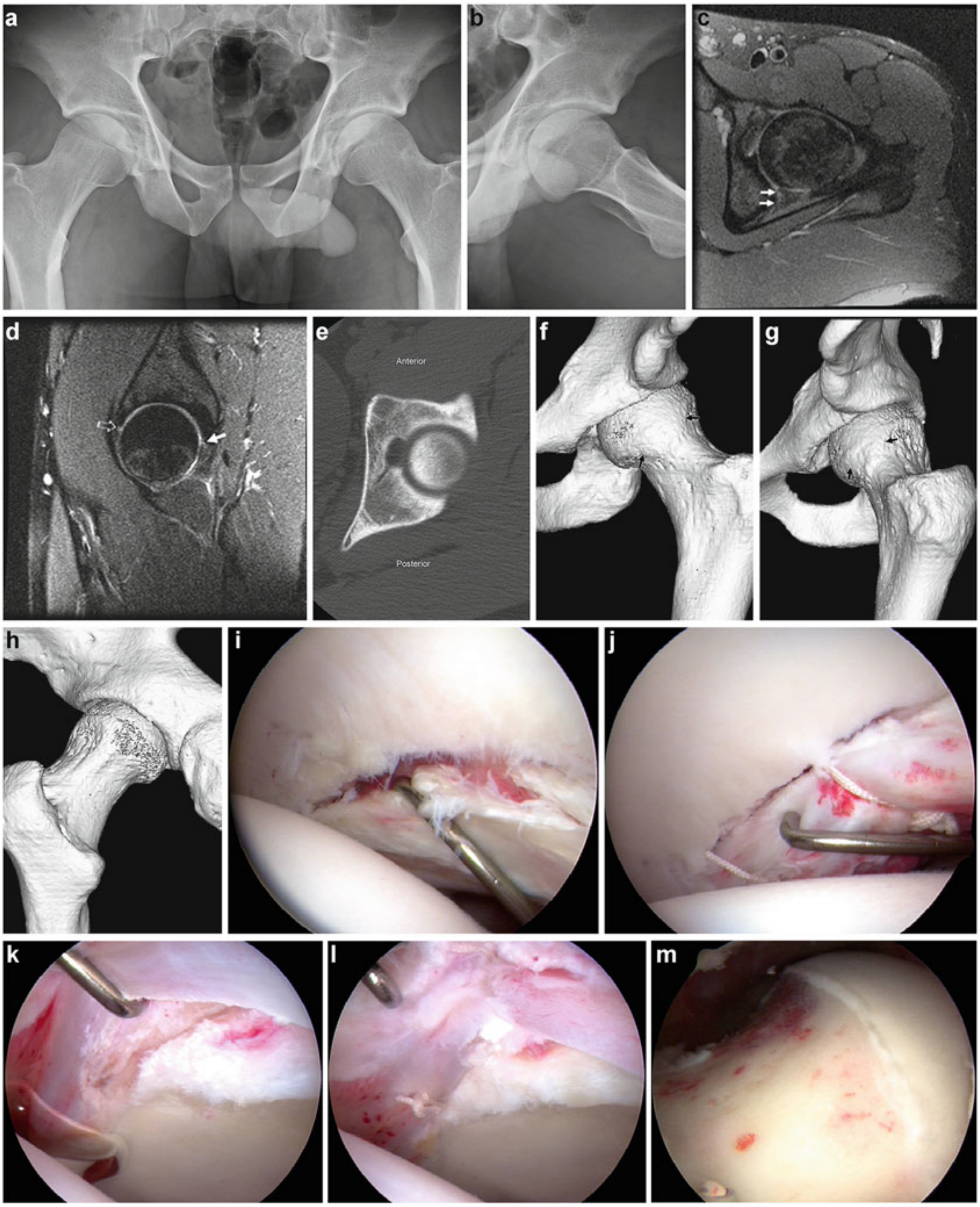


Fig. 6 A 21-year-old collegiate linebacker had sustained a previous traumatic posterior subluxation episode of his left hip. He recovered uneventfully and returned to play and then a year later sustained a second subluxation episode with a noncontact mechanism. (**a**) AP radiograph is indicative of significant acetabular retroversion characterized by positive crossover, posterior wall, and ischial spine signs. (**b**) Lateral radiograph reveals substantial cam morphology. (**c**) Axial MRI image reveals a posterior Bankart-type labral tear (*arrows*). (**d**) Sagittal MRI image partly reflects posterior labral tear (*white arrow*) as well as increased signal in the anterior labrum (*open arrow*). (**e**) Axial CT scan further illustrates acetabular retroversion. (**f**, **g**, **h**) 3-D reconstructions further illustrate the cam lesion (*arrows*) and acetabular retroversion. Arthroscopy was recommended, and views of the central compartment are

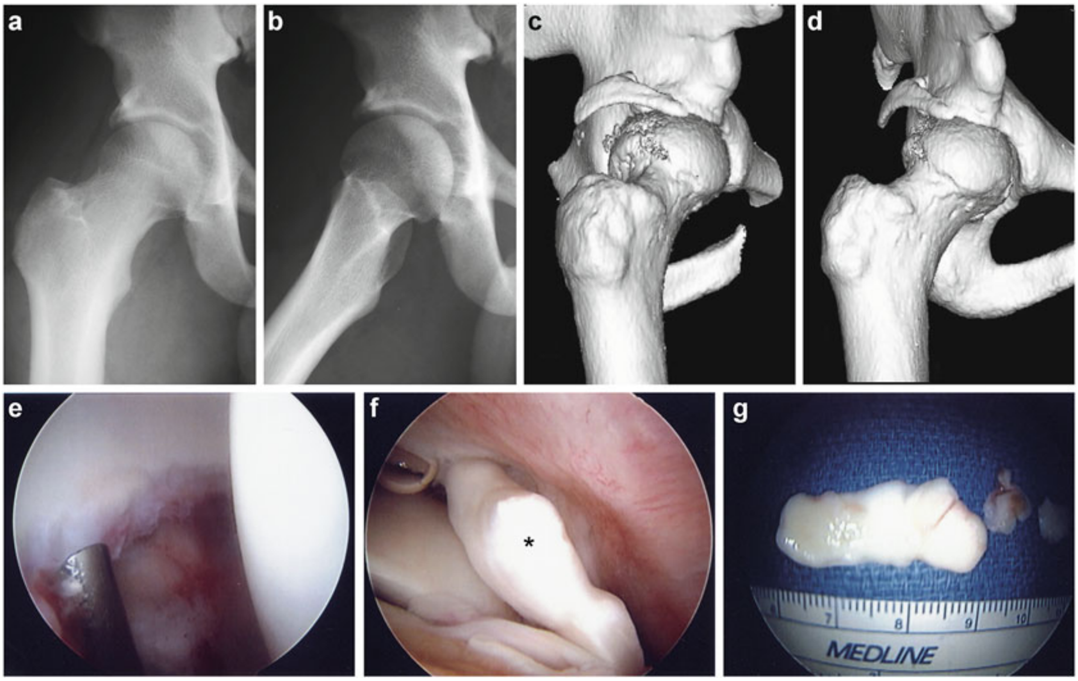


Fig. 7 A 25-year-old NFL fullback sustained a posterior fracture dislocation of the right hip that was reduced on the field. Five months later, he was continuing to have symptoms of pain and catching but no instability. (**a**, **b**). AP and lateral radiographs revealed a concentric reduction. (**c**, **d**). 3-D reconstructions revealed significant residual displacement of a malunited posterior rim fracture but did not seem to explain his mechanical symptoms in the absence of instability. (**e**). Viewing from the anterolateral portal, the posterior defect is identified but, again, did not explain his mechanical symptoms. (**f**). Viewing in the peripheral compartment, a large loose articular fragment was identified (*asterisk*). (**g**). The fragment was removed with alleviation of his mechanical symptoms. He subsequently returned to play professional football (©2014 J. W. Thomas Byrd, MD)

stabilizers, including dynamic muscle strength and the static contribution of the labrum, capsule, and, possibly, even the ligamentum teres. No strong scientific data or clinical outcomes exist regarding this approach. The capsule can be repaired, imbricated, or possibly even grafted, although some may be better served by an open approach [22]. Loss of the labrum can be substituted with autograft or allograft reconstruction, and some investigative work has been done in reconstruction of the ligamentum teres [23, 24]. These are challenging cases that may best be referred to tertiary hip preservation centers.

Summary

Sports-related traumatic instability of the hip is not uncommon. The symptoms may not be incapacitating, and the initial diagnosis may not be obvious. Thus, a sense of awareness must be maintained. Appropriate studies will confirm the nature of the injury. There is a high correlation

Fig. 6 (continued) from the anterolateral portal. (**i**) Viewing posteriorly, the posterior Bankart-type labral detachment is probed from the posterolateral portal. (**j**) The posterior labrum has been repaired with two suture anchors using a loop suture to restore the labrum and create a bolster. (**k**) Viewing anteriorly, a separate anterior labral tear is probed. (**l**). This tear was repaired. (**m**) Viewing in the periphery, the cam lesion was corrected to eliminate its levering effect against the anterior rim. The athlete recovered and returned uneventfully to intercollegiate football (©2014 J. W. Thomas Byrd, MD) with the presence of underlying FAI, which may predispose the joint to subluxation. Most cases of traumatic instability can be treated nonsurgically with a high likelihood of returning to athletic endeavors. When surgery is necessary, most can be managed arthroscopically with a high likelihood of successful outcomes. The eventual outcome is probably most determined by the magnitude of damage at the moment of injury. Most will do well, but some will not. Thus, a thoughtful, watchful approach is necessary in all cases to determine how the joint recovers.

References

1. Moorman III CT, Warren RF, Hershman EB, et al. Traumatic posterior hip subluxation in American football. J Bone Joint Surg. 2003;85-A(7):1190–6.
2. Foulk DM. Mullis BH: hip dislocation; evaluation and management. J Am Acad Orthop Surg. 2010;18(4): 199–209.
3. Philippon MJ, Kuppersmith DA, Wolff AB, Briggs KK. Arthroscopic findings following traumatic hip dislocation in 14 professional athletes. Arthroscopy. 2009;25(2):169–74. doi:10.1016/j.arthro.2008.09.013.
4. Krych AJ, Thompson M, Larson CM, Byrd JWT, Kelly BT. Is posterior hip instability associated with cam and pincer deformity? Clin Orthop Relat Res. 2012;470:3390–7. doi:10.1007/s11999-012-2468-3.
5. Larson CM, Sikka RS, Sardelli MC, Byrd JWT, et al. Increasing alpha angle is predictive of athletic-related “hip” and “groin” pain in collegiate National Football League prospects. Arthroscopy. 2013;29(3):405–10.
6. Kapron AL, Anderson AE, Aoki SK, Phillips LG, et al. Radiographic prevalence of femoroacetabular impingement in collegiate football players. J Bone Joint Surg Am. 2011;93(19):e111(1–10). doi:10.2106/JBJS.K.00544.
7. McKibbin B. Anatomical factors in the stability of the hip joint in the newborn. J Bone Joint Surg Br. 1970;52(1):148–59.
8. Martin RR, Palmer I, Martin HD. Ligamentum teres: a functional description and potential clinical relevance. Knee Surg Sports Traumatol Arthrosc. 2012;201:1209–14.
9. Shindle MK, Ranawat AS, Kelly BT. Diagnosis and management of traumatic and atraumatic hip instability in the athletic patient. Clin Sports Med. 2006;25(2): 309–26, ix-x.
10. Bowman Jr KF, Fox J, Sekiya JK. A clinically relevant review of hip biomechanics. Arthroscopy. 2010;26(8): 1118–29. doi:10.1016/j.arthro.2010.01.027. Review.
11. Safran MR, Lopomo N, Zaffagnini S, Signorelli C, et al. In vitro analysis of peri-articular soft tissues passive constraining effect on hip kinematics and joint stability. Knee Surg Sports Traumatol Arthrosc. 2013;21(7):1655–63. doi:10.1007/s00167-012-2091-6. Epub 2012 Jun 30.
12. Martin HD, Savage A, Braly BA, Palmer IJ, et al. The function of the hip capsular ligaments: a quantitative report. Arthroscopy. 2008;24(2):188–95. doi:10.1016/j.arthro.2007.08.024. Epub 2007 Nov 26.
13. Crawford MJ, Dy CJ, Alexander JW, Thompson M, et al. The 2007 Frank Stinchfield Award. The biomechanics of the hip labrum and the stability of the hip. Clin Orthop Relat Res. 2007;465:16–22.
14. Ferguson SJ, Bryant JT, Ganz R, et al. The acetabular labrum seal: a poroelastic finite element model. Clin Biomech. 2000;15(6):463–8.
15. Ferguson SJ, Bryant JT, Ganz R, et al. An in vitro investigation of the acetabular labral seal in hip joint mechanics. J Biomech. 2003;36(2):171–8.
16. Ferguson SJ. Biomechanics of the acetabular labrum. Ph.D. Thesis, Queen’s University, Kingston. ISBN 3-905363-00-3.
17. Shu B, Safran MR. Hip instability: anatomic and clinical considerations of traumatic and atraumatic instability. Clin Sports Med. 2011;30:349–67.
18. Coplen EM, Voight ML. Rehabilitation of the hip. In: Byrd JWT, editor. Operative hip arthroscopy. 3rd ed. New York: Springer; 2012. p. 411–40.
19. Byrd JWT. Hip luxation in the NFL. In: Proceedings of the NFL physicians society annual meeting, Indianapolis, 22 Feb 2008.
20. Bartlett CS, DiFelice GS, Buly RL, Quinn TJ, et al. Cardiac arrest as a result of intraabdominal extravasation of fluid during arthroscopic removal of a loose body from the hip joint of a patient with an acetabular fracture. J Ortho Trauma. 1998;12(4): 294–300.
21. Byrd JWT, Jones KS. Traumatic rupture of the ligamentum teres as a source of hip pain. Arthroscopy. 2004;20(4):385–91.
22. Domb BG, Philippon MJ, Giordano BD. Arthroscopic capsulotomy, capsular repair, and capsular plication of the hip: relation to atraumatic instability. Arthroscopy. 2013;29(1):162–73. doi:10.1016/j.arthro.2012.04.057. Epub 2012 Aug 15.
23. Geyer MR, Philippon MJ, Fagrelius THIS, Briggs KK. Acetabular labral reconstruction with an iliotibial band autograft outcome and survivorship analysis at minimum 3-year follow-up. Am J Sports Med. 2013;41(8):46–50.
24. Philippon MJ, Pennock A, Gaskill TR. Arthroscopic reconstruction of the ligamentum teres: technique and early outcomes. J Bone Joint Surg Br. 2012;94(11):1494–8. doi:10.1302/0301-620X.94B11.28576.

Surgical Technique: Arthroscopic Reduction and Internal Fixation of Femoral Head and Acetabulum Fractures

77

Dean K. Matsuda

Contents

Abstract

Beyond foreign body or fracture fragment removal, the use of hip arthroscopy has expanded into a minimally invasive treatment for the osteosynthesis of select femoral head and acetabular fractures. This section focuses on the arthroscopic surgical techniques enabling arthroscopic reduction and internal fixation of the acute and the malunited femoral head fracture as well as the acetabular rim and anterior column fracture.

Introduction

Arthroscopic and endoscopic osteosynthesis is increasingly utilized for certain intra-articular fracture types due to the minimally invasive nature of the procedures and high accuracy [1]. In general, advantages of arthroscopic fracture fixation over open methods are the less invasive and magnified visualization of the intra-articular space and chondral surfaces, enabling precise osteoarticular fracture reduction while facilitating concomitant treatment of associated intra-articular pathology and accelerated rehabilitation with earlier return to work and sports [1–3]. Further advantages include improved cosmesis and potential cost savings of outpatient surgery [1, 2]. However, sometimes lengthy, technically demanding procedures with a prolonged learning curve and limited fixation alternatives are disadvantages of arthroscopic osteosynthesis

D.K. Matsuda
Department of Orthopedic Surgery, Kaiser West Los Angeles Medical Center, Los Angeles, CA, USA
e-mail: dean.k.matsuda@kp.org

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_85

[1]. This chapter introduces the indications, utility, key concepts, and surgical techniques for arthroscopic osteosynthesis of select femoral head and acetabular fractures.

Femoral Head Fractures

Femoral head fractures are relatively uncommon injuries typically associated with hip dislocations. These tend to be high-energy injuries with historically poor outcomes (e.g., posttraumatic osteoarthrosis and/or osteonecrosis) despite treatment with nonsurgical or open surgical means [4]. The more common posterior hip dislocation may cause an infrafoveal fracture (Pipkin 1) which is nonweight bearing and may tolerate resection. Anterior dislocations tend to be associated with more critical weight-bearing suprafoveal fractures of the femoral head. The first reported case of arthroscopy-assisted osteosynthesis was a small infrafoveal fracture that was reduced by hip positional manipulation followed by fixation with an absorbable percutaneous pin [5]. More recently, arthroscopic osteosynthesis has been performed with encouraging short-term outcomes on acute suprafoveal femoral head fractures [6, 7] (Fig. 1) and even a femoral head malunion [8].

Preoperative Planning

Experience

Arthroscopic osteosynthesis of femoral head and acetabular fractures is technically challenging. One should assess his/her personal and surgical team's experience and ability to perform these procedures in a safe manner.

Game Plan/Contingencies

Fixate critical fragments that contribute to weight-bearing articular congruency (Fig. 2) and/or structural integrity but consider removal of others. If one decides to perform arthroscopic osteosynthesis, a contingency plan is recommended in case the procedure does not proceed as planned. It is better to convert to an open osteosynthesis than perform an inadequate arthroscopic reduction and/or fixation. Keep in mind the general principle of anatomic reduction with secure internal fixation permitting early joint motion. Resection, even arthroscopic, of a critical weight-bearing or structural fracture fragment is the last option if all reasonable attempts at arthroscopic or open osteosynthesis fail.

FAI Considerations

Acetabular overcoverage from pincer FAI may prevent an acceptable angle of approach for

Fig. 1 Shows a left femoral head fracture with displaced suprafoveal weight-bearing osteochondral fragment (With permission from Orthopedics Today)

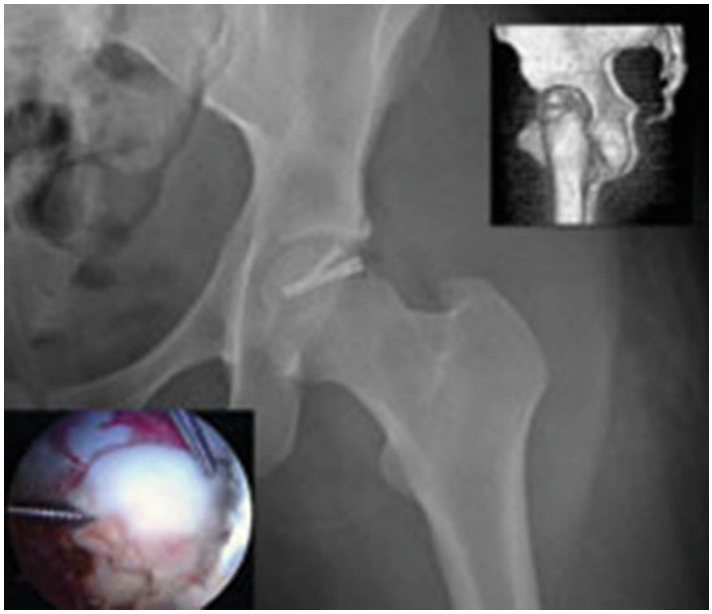


Fig. 2 Shows the same fracture during arthroscopic reduction using the chopstick maneuver (*lower left*) and postoperative imaging (With permission from Orthopedics Today)

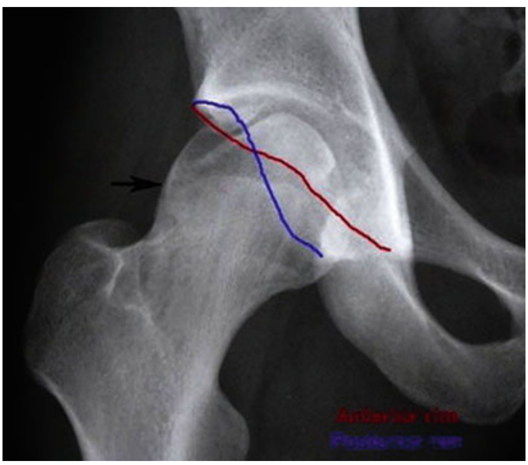


Fig. 3 Is a detail of a preoperative AP pelvis radiograph showing a double-density shadow of a clamshell suprafoveal femoral head fracture seen after emergent closed reduction of anterior dislocation (Brumback type 4B). Note cam (*arrow*) and pincer FAI with focal acetabular overcoverage and crossover sign (With permission from Elsevier Arthroscopy Journal)

screw fixation of the fracture fragment(s). In such instances, adjunctive arthroscopic acetabuloplasty of the overcovered femoral head may enable successful arthroscopic fixation with headless screws [7] (Fig. 3, 4, 5, and 6). Note that one must not cause iatrogenic dysplasia by overzealous rim trimming and the labrum should be preserved, typically with refixation. Cam FAI, even if previously asymptomatic, may be addressed with arthroscopic femoroplasty without traction as a potentially prophylactic measure. Finally, if a fracture fragment involves a region of cam morphology, resection of that fragment may improve structural offset in that region.

Consent

Consent should include arthroscopic and open osteosynthesis plus possible resection of fracture fragment(s). Arthroscopic acetabuloplasty, femoroplasty, and chondrolabral surgery with possible labral debridement, refixation, and reconstruction should be included as appropriate.

Equipment

Ensure that the fracture table and/or portable hip distractor provides sufficient freedom of hip motion for hip positioning and dynamic testing and does not obstruct fluoroscopic visualization of the operative hip on anterior–posterior and lateral projections. Even if one does not routinely use

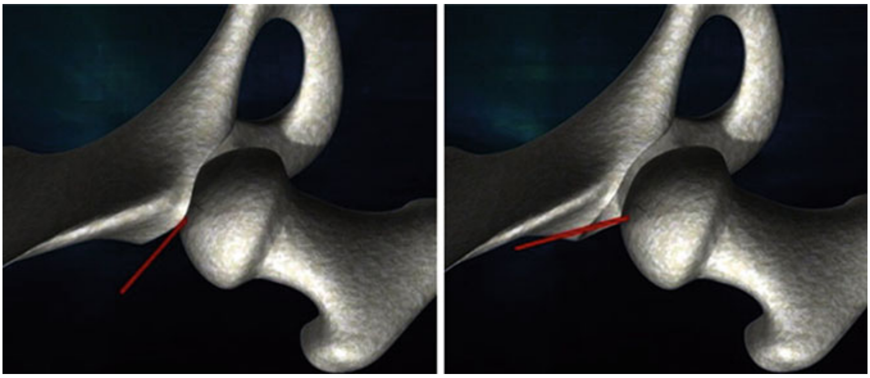


Fig. 4 Depicts screw path (*red line*) before (*left*) and after (*right*) arthroscopic acetabuloplasty. A more perpendicular trajectory for screw fixation is achieved (With permission from Elsevier Arthroscopy Journal)

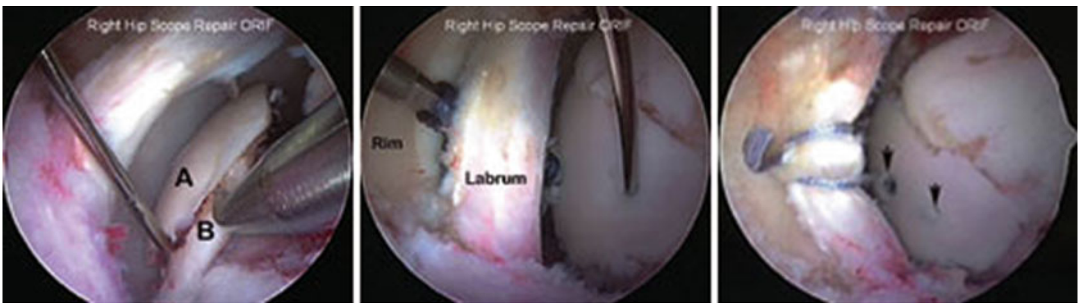


Fig. 5 Shows the clamshell fracture with folded osteochondral fragment with superior (**a**) and inferior (**b**) fragments being pried apart with microfracture awl (*left*). The reduced fracture is being fixated with cannulated headless compression screws (*middle*). The acetabular rim has been trimmed and the labrum preserved with a screw being inserted between the structures to optimize screw fixation path. Arthroscopic labral refixation is then completed (*right*). *Arrows* indicate buried screw heads below chondral surface (With permission from Orthopedics Today)

Fig. 6 Shows postoperative healed femoral head fracture. *Arrow* indicates superolateral acetabuloplasty (With permission from Elsevier Arthroscopy Journal)

fluoroscopic guidance for hip arthroscopy, a C-arm image intensifier is strongly recommended and is very helpful, especially when using metallic (radiopaque) screws (see below). A cannulated headless screw system is needed. These systems vary in their specific instructions so it behooves one to become familiar with the chosen system.

Setup

Although lateral position hip arthroscopy is an option, supine hip arthroscopy will be described. Position the image intensifier between the abducted legs enabling AP, lateral, and dynamic fluoroscopy. The operative hip is positioned in

10° flexion, 20° abduction, and 30+ degrees of internal rotation.

Consider using the fluoroscopic templating technique [9] especially in cases where one anticipates possible acetabular rim trimming. Moreover, pelvic positioning is standardized by aligning the pelvis to the vertical beam of the C-arm in frontal and sagittal planes prior to commencement of surgery.

Traction

A detailed preoperative assessment of sciatic function is important as there is a relatively high incidence of sciatic injury associated with trauma from hip dislocations.

Remain vigilant of traction time *AND* force [10]. Limit the amount of hip distraction to 10 mm of actual space between the acetabular and femoral head chondral surfaces during central compartment arthroscopy. Rather than over-distract, consider hip adduction along with adjustments in hip rotation to permit acceptable screw trajectory. The use of a traction “time-out” for every hour of applied traction is prudent. Concurrent procedures requiring no traction (e.g., femoroplasty) may be performed during this time.

Portals

The anterolateral portal (ALP) and the modified midanterior portal (mMAP) [21] are used. The latter is typically 3 cm anterior and 4–5 cm distal to the ALP and is made in the aforementioned internal rotated hip position. Typically, a 70° arthroscope permits sufficient visualization from the ALP and the mMAP is the working portal, although occasionally interportal exchange is needed.

A vertical line passing through the anterior superior iliac spine (ASIS) along the operative thigh is a landmark beyond which one should not stray medial to minimize risk of inadvertent neurovascular damage. Percutaneous passage of guide pins and cannulated screws should remain lateral to this landmark whenever possible.

Fluid Pressure

Minimizing arthroscopic fluid pressure minimizes the risk of inadvertent abdominal compartment syndrome. With hypotensive anesthesia, fluid pressure of 50 mmHg is often sufficient for adequate arthroscopic visualization. In some cases, even “dry” arthroscopy may be considered. Intermittent palpation of the draped abdomen, monitoring of hemodynamics and core body temperature, and, if indicated, iliopsoas release at the conclusion of surgery are prudent precautionary measures [11]. Intentional removal of intra-articular debris should be performed in hopes of minimizing third body wear; a suction-shaver and high-flow (but low pressure) arthroscopic irrigation may expedite this step and should be repeated at the conclusion of the surgery.

Arthroscopic Reduction

Although closed reduction techniques can be used by manipulating operative hip position for minimally displaced fractures, significantly displaced fractures typically require arthroscopic reduction. A switching stick or probe may enable gross translation of the osteochondral fragment to the fracture site. If fragment derotation is necessary, one may use a toggle stick method; however, this violates the articular surface. The chopstick technique [6] (Fig. 2) uses two percutaneous guide pins with two points of chondral contact to aid in fragment derotation for arthroscopic reduction.

Arthroscopic Internal Fixation

Once fracture reduction is achieved, the guide pins used to enable arthroscopic reduction can then be used to provide transient fracture fixation. The percutaneous entry sites for these guide pins should enable an acceptable trajectory for cannulated headless screw fixation. Hip adduction in traction may facilitate arthroscopic screw fixation by exposing some femoral head fractures out from under the obstructive coverage of the acetabulum. Perfectly perpendicular screw fixation is not mandatory for successful fracture union; however, a relatively perpendicular position is desirable as it permits optimal engagement of the subchondral bone while seating the headless screw below the chondral surface. Current headless compression screw design compresses the fracture site with antegrade advancement. Typically two or three screws (Acutrak mini, Acumed, Hillsboro, Oregon) are required although this will vary with the size and thickness of the osteochondral fragment. Avoid overzealous antegrade screw advancement as this may lose subchondral purchase and compromise compression if one then decides to partially back the screw out in a retrograde manner.

Although radiolucent bioabsorbable implants may be used, an advantage of metallic headless screws is that their position may be monitored to detect even subtle joint encroachment via intraoperative fluoroscopy and postoperative radiographs [6]. If detected, one may perform arthroscopic screw removal or antegrade screw advancement. A relative disadvantage of metallic implants is unwanted scatter from computed tomographic and magnetic resonance imaging, although some scanners have metal-subtraction technology.

Dynamic Arthroscopic and Fluoroscopic Testing

Dynamic arthroscopic and fluoroscopic examinations confirm safe positioning of headless screws and also confirm absence or eradication of coexistent FAI. Static biplane fluoroscopy may not detect a proud screw violating the joint. Rotating the C-arm around the femoral head and moving the hip within the static C-arm are both acceptable methods, although the latter may permit more range of motion including internal and external rotation and may be quicker with less radiation exposure.

Postoperative Considerations

Early range of hip motion while protecting against excessive weight-bearing stress is desired. Typically 6–8 weeks of protected weight bearing of the operative hip with two crutches or a walker is sufficient. Exercise bicycling is permitted after 1 week and swimming (freestyle stroke) and jogging in a pool begin when portals are healed. Return to impact activity is individualized to the patient and his/her fracture, but even in the best case scenario, running is not begun until 3 months. Key pearls and pitfalls of arthroscopic femoral head osteosynthesis are provided in Table 1.

Table 1 Pearls for arthroscopic reduction and internal fixation of femoral head fractures

Perform accurate preoperative fracture assessment
Perform accurate preoperative self-assessment of surgical experience and arthroscopic skills
Be willing to perform possible open reduction–internal fixation (rather than arthroscopic fragment removal) if arthroscopic method fails
Consider fluoroscopic templating technique to standardize pelvic position under hip distraction
Hip adduction may improve path for screw fixation
If pincer FAI, acetabuloplasty of overcoverage may improve path of screw fixation
N.B. Do not cause iatrogenic dysplasia
Pay careful attention to safe portal placement (may require several accessory portals), capsulotomies, intra-articular fluid pressure, and distraction amount and time
Mobilize, translate, and reduce fracture fragment(s); consider use of chopstick technique where indicated
Consider arthroscopic fixation using radiopaque screw(s) or pin(s) visible under intermittent fluoroscopic guidance
Consider removal of osteochondral bone not essential to weight bearing or structural integrity of fracture construct
Confirm accurate reduction and stable fixation by arthroscopic and fluoroscopic dynamic testing
Allow early mobilization of hip and protected weight-bearing commensurate with assessed fracture fixation
Perform interval postoperative radiographic assessment with special attention to joint space narrowing and/or hardware migration/violation of hip joint

Femoral Head Malunions

Femoral head malunions (Fig. 7) present a degree of technical complexity beyond acute femoral head fracture fixation. After removal of any surgical hardware, one must locate the malunion site via arthroscopic and fluoroscopic visualization. Although percutaneous screws used in femoral neck fracture fixation are typically readily removed, smaller screws of the femoral head may be buried. As long as the malunion site is identifiable, the malunion may be mobilized using ¼ inch straight and/or angled osteotomes (Fig. 8) via percutaneous placement in safe areas lateral to the vertical line passing through the ASIS. Then arthroscopic reduction may be performed using aforementioned techniques. Moreover, any retained bent screw may actually aid reduction; hitting it with a mallet and small osteotome, the screw may straighten, indicating improved reduction, while distributing impact forces across a larger surface area [8].

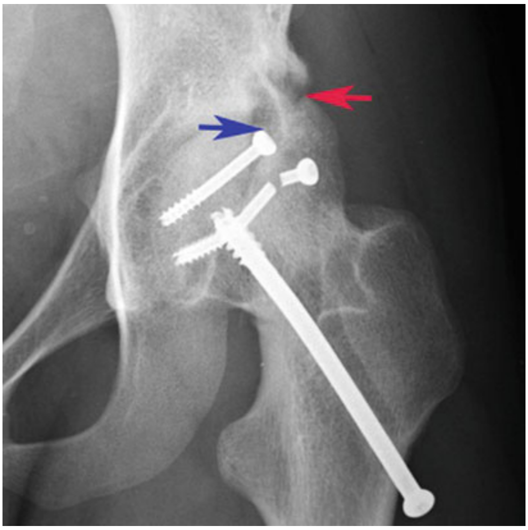


Fig. 7 Shows femoral head malunion after initial ORIF failed with premature ambulation. Note the lateral column of femoral head fracture (*red arrow*) is causing laterocephalic acetabular impingement against superolateral rim. The inferior malunion is >1 cm displaced (*blue arrow*). The long screw was removed prior to takedown of the malunion (With permission from Orthopedics Today)

Once the femoral head malunion is mobilized and reduced, arthroscopic bone grafting may be performed by passing graft material via a cannula. Osteoinductive bone graft putty can be "muzzle-loaded" into an arthroscopic cannula. The "loaded" cannula can then be positioned through the mMAP and positioned so that the graft substance can be inserted into the malunion site (Fig. 8) in a controlled manner using a matching blunt stylet as a plunger under transient "dry" arthroscopic visualization [8, 12]. Upon completion, arthroscopic fixation of the previously malunited fracture may be performed using aforementioned arthroscopic headless screw fixation techniques or, if the femoral head fragment is sufficiently large, outside-in percutaneous fixation using 7 mm cannulated screws under fluoroscopic guidance (Fig. 9).

Acetabular Fractures

Although there are several articles published on percutaneous screw fixation of acetabular fractures under fluoroscopic guidance, there is less

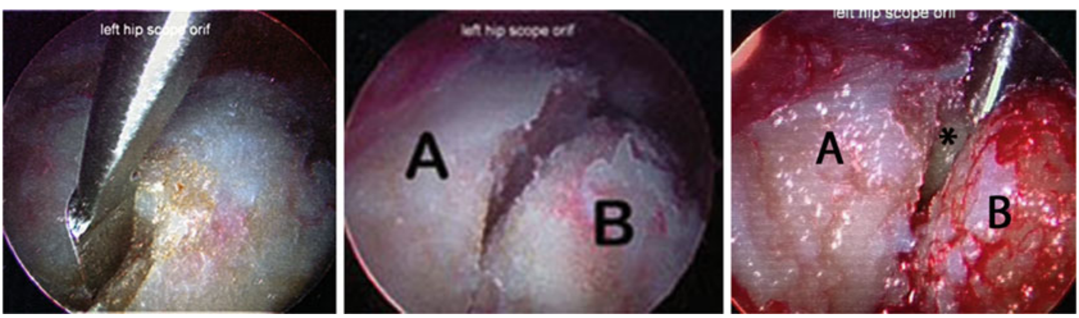


Fig. 8 Supine arthroscopic images during arthroscopic takedown with ¼ inch angled osteotome (*left*), after arthroscopic reduction (*middle*) (With permission from Elsevier Arthroscopy Journal), and during arthroscopic insertion of bone graft putty via cannula under dry arthroscopic visualization (*right*) prior to compression of fracture site with percutaneous lag screws. *A* medial femoral head fragment, *B* intact lateral column, *bone graft putty (With permission from Elsevier Arthroscopy Journal)

Fig. 9 Shows postoperative radiograph with reduced and fixated malunion. Note straightened inferior femoral head screw and eradication of laterocephalic acetabular impingement (With permission from Orthopedics Today)

Table 2 Pearls and pitfalls for arthroscopic osteosynthesis of rim fractures (With permission from Elsevier Arthroscopy Journal)

Pearls
CE angles should be measured with and without associated rim fractures/os acetabuli
When rim fractures contribute to pincer-type FAI and CE angles without the fragments are >20°–25° (LCE angle) and >20° (ACE angle), the fragments can be completely excised
When CE angles without these associated rim fragments result in an LCE angle <20°–25° and ACE angle <20°, these fragments should be either maintained or partially resected with or without internal fixation as part of a joint preservation procedure
Drilling across the fibrocartilaginous junction and internal fixation with cannulated screws may help to promote healing of these fragments in the previously mentioned situations
Direct arthroscopic and fluoroscopic visualization and placement of portals further distal during screw fixation can facilitate safe screw placement and avoid the potential for intra-articular acetabular penetration

written about arthroscopic osteosynthesis. Acetabular fractures may be differentiated into rim and non-rim fractures.

Rim Fractures

Acetabular rim fractures or os acetabuli typically occur in the anterosuperior and superolateral regions and may be treated via arthroscopic resection, screw fixation, or a combination of both. The key concept is to adapt the treatment so that neither hip instability nor impingement results. Guidelines are given in Table 2. Arthroscopic resection (either en bloc or via burring) may be performed in many cases, followed by labral refixation or reconstruction. But if rim fragment resection will result in iatrogenic dysplasia, consider arthroscopic osteosynthesis with one or two 3.5 mm cannulated lag screws. Successful union has been reported with arthroscopic screw fixation after resection of interfragmentary fibrocartilaginous tissue [13] or, in cases where the acetabular chondral remains intact, in situ fixation without “freshening” the fracture site [14]. Following transient fixation with a percutaneously placed guide pin (staying lateral to the ASIS sagittal line), lag screw fixation under arthroscopic and fluoroscopic guidance may be performed. If a sizable rim fracture fragment would result in hip instability if removed but pincer impingement if retained, partial osteoplasty of the fragment with an arthroscopic burr may precede screw fixation (Fig. 10).

Anterior Column Fractures

Fluoroscopic-guided percutaneous screw fixation of the anterior acetabular column (Fig. 11) can provide stable interfragmentary compression and less morbidity than open surgery [15–17], but inadvertent medial wall penetration with intra-articular screw placement and significant fluoroscopic radiation exposure are concerns. Arthroscopic visualization of the central compartment permits diagnostic assessment of intra-articular injury, particularly to the chondral surfaces, removal of fracture debris, concomitant

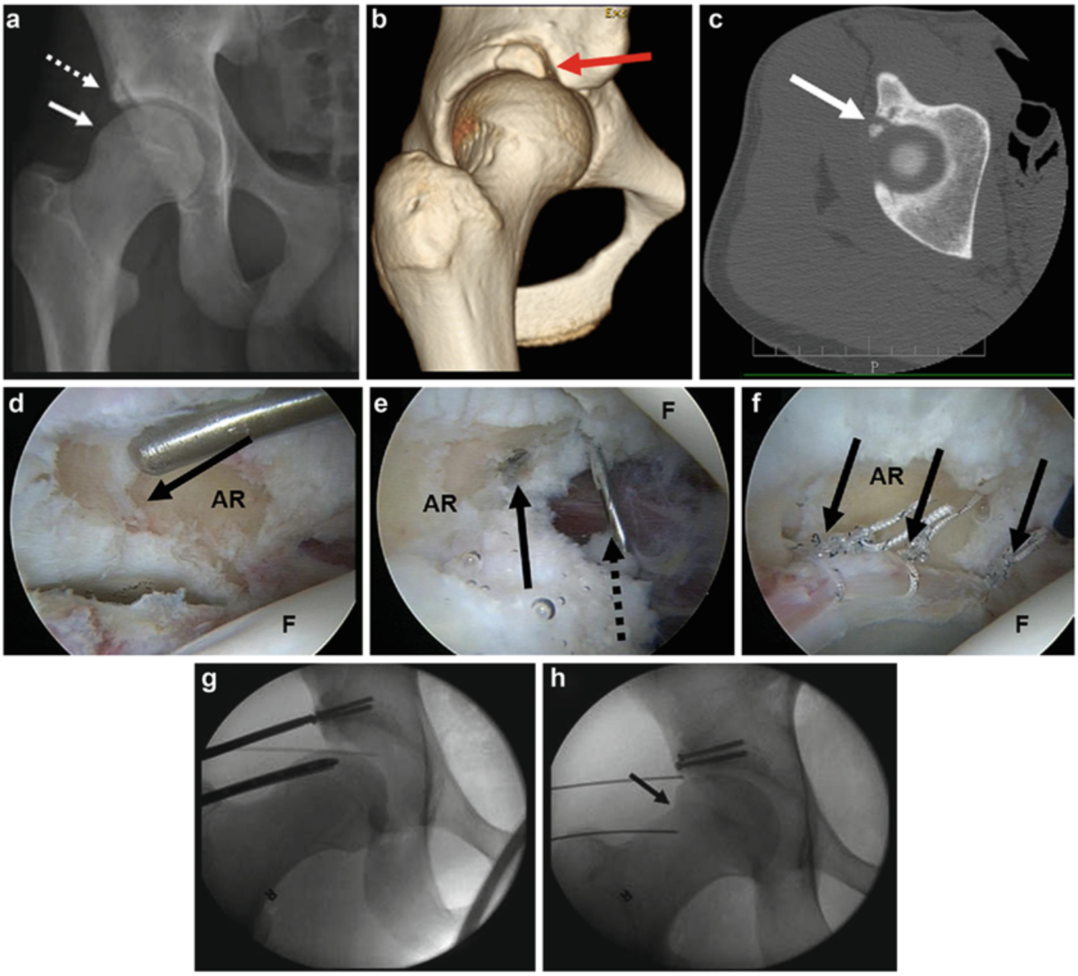


Fig. 10 (**a**) Preoperative AP radiograph shows cam-type impingement (*solid arrow*) and a rim fracture superiorly (*dashed arrow*) with an LCE angle of 35° with the fragment and 18° without the fragment. Computed tomography showed (**b**) a large superior rim fracture (*arrow*) on 3-dimensional reconstructions and (**c**) superior acetabular retroversion on axial images. (**d**) Arthroscopy after rim resection showed the fibrocartilaginous junction between the native acetabulum and the rim fracture (*arrow*), (**e**) placement of a screw (arrow) and drilling for a second screw (*dashed arrow*) in the superior rim, and (**f**) final image after rim resection, partial resection and internal fixation of the rim fracture, and labral refixation with suture anchors (*arrows*). Intraoperative fluoroscopy showed (**g**) placement of cannulated screws to secure the rim fracture after rim resection and (**h**) a final image after internal fixation of the rim fracture and femoral osteochondroplasty (*arrow*) (*F* femoral head, *AR* acetabular rim) (With permission from Elsevier Arthroscopy Journal)

chondrolabral surgery, assessment of fracture reduction and interfragmentary compression, detection of possible lag screw violation, and reduction in radiation exposure [18]. Controlled hip distraction may facilitate fracture reduction. The fracture site is arthroscopically visualized and lavage and debridement are performed. Arthroscopic manual reduction may be aided with an arthroscopic probe or switching stick. Guidewires are positioned with protective drill sleeves. Antegrade or retrograde percutaneous cannulated screw fixation using one or more 6.5 mm partial threaded screw(s) is performed under arthroscopic visualization (Figs. 12 and 13) with fluoroscopic spot imaging. Yang et al. [18] described a modified percutaneous entry site that avoids the

Fig. 11 Right hip anteroposterior view of transverse fracture with displaced anterior column (With permission from Elsevier Arthroscopy Journal)

thinnest region below the teardrop or near the psoas (Fig. 14). Familiarity with percutaneous screw fixation and periacetabular skeletal geometry [19] as well as the basic principles of acetabular fracture fixation should precede attempts at arthroscopic osteosynthesis of displaced anterior column fractures.

Intra-abdominal fluid extravasation has occurred in this setting [20] so one must be vigilant of this potentially life-threatening complication and use the aforementioned precautions described for femoral head fracture treatment. "Dry" arthroscopy with intermittent lavage and suction is an alternative to using fluid pressures in the 50–60 mmHg range and may safely permit arthroscopic acetabular osteosynthesis. The optimal timing of arthroscopic acetabular fracture osteosynthesis has not been established, but waiting more than

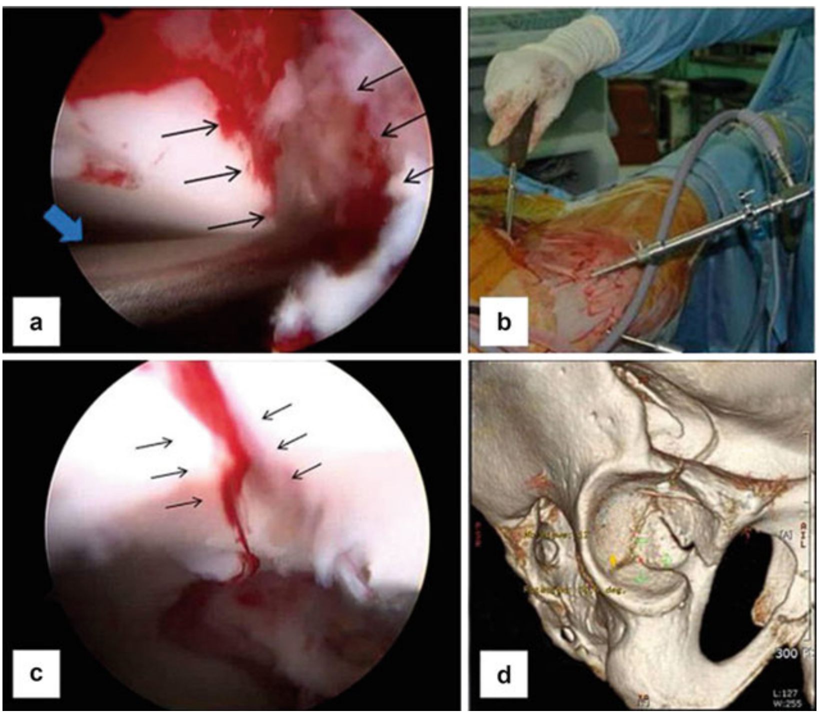


Fig. 12 (**a**) Arthroscope through anterior portal viewing fracture site (*black arrows*) after debridement of fracture margins. An arthroscopic hook (*blue arrow*) through the anterolateral portal was used as a reduction tool during compression of the fracture gap. (**b**) Arthroscopy of hip. It should be noted that the anterolateral portal was used for viewing and the posterolateral portal for outflow. (**c**) Reduction of intra-articular fracture site (*arrows*) was confirmed intraoperatively under direct visualization by hip arthroscopy. (**d**) Postoperative computed tomographic imaging showing anatomic reduction of articular fracture site (With permission from Elsevier Arthroscopy Journal)

Fig. 13 Three-year follow-up radiographs after arthroscopy-assisted screw fixation of anterior column fracture of the right hip showing consolidation of fracture (With permission from Elsevier Arthroscopy Journal)

Fig. 14 Guidewire placement with two different directions on bone model, with wire A showing screw direction with lesser angle to sagittal plane and more central convergence compared with wire B. Guidewire A has less risk of inadvertent intra-articular penetration (With permission from Elsevier Arthroscopy Journal)

Table 3 Tips for arthroscopy-assisted acetabular fracture reduction (With permission from Elsevier Arthroscopy Journal)

Use general anesthesia for full relaxation of the pelvis musculature
Use a fracture table and prepare the C-arm
Prepare the uninjured contralateral lower limb in the lithotomy position on the fracture table. This position will allow viewing of C-arm images and arthroscopic visualization simultaneously
Distract the injured limb for hip joint arthroscopic instrument introduction
Use 3 portals: anterior, anterolateral, and posterolateral (posterolateral portal for outflow)
Be cautious about extravasation of arthroscopic irrigation fluid
Use pump pressure of approximately 60–70 mmHg
Debride and remove any interposed soft tissue at the fracture gap or loose bodies before reduction

7–10 days may make fracture reduction more challenging. Key pearls and pitfalls of arthroscopic acetabular osteosynthesis are provided in Table 3.

Summary

Arthroscopic osteosynthesis of select femoral head and acetabular fractures have been performed with encouraging outcomes. It is conceivable that hip arthroscopy will play an expanding role in the less invasive future treatment of select acute and even malunited fractures.

References

1. Atesok K, Doral MN, Whipple T. Arthroscopy-assisted fracture fixation. Knee Surg Sports Traumatol Arthrosc. 2011;19:320–9.
2. Matsuda DK. Arthroscopic osteosynthesis of femoral head and acetabular fractures. Symposium: arthroscopic treatment of fractures. ISAKOS 2013, 14 May, Toronto; 2013.
3. Matsuda DK. Hip arthroscopy for trauma: Innovative techniques for a new frontier. Orthop Today. 2010. http://www.healio.com/orthopedics/arthroscopy/news/print/orthopedics-today/%7B4bfbbb94-26db-42bf-

9bca-1754d7515dcf%7D/hip-arthroscopy-for-trauma-innovative-techniques-for-a-new-frontier
4. Epstein HC, Wiss DA, Cozen L. Posterior fracture dislocation of the hip with fractures of the femoral head. Clin Orthop Relat Res. 1985;201:9–17.
5. Yamamoto Y, Ide T, Ono T, Hamada Y. Usefulness of arthroscopic surgery in hip trauma cases. Arthroscopy. 2003;19(3):269–73.
6. Matsuda DK. A rare fracture, an even rarer treatment: the arthroscopic reduction and internal fixation of an isolated femoral head fracture. Arthroscopy. 2009; 25(4):408–12.
7. Matsuda DK, Hamani D. The clamshell fracture and adjunctive acetabuloplasty in the arthroscopic osteosynthesis of femoral head fractures with femoroacetabular impingement. Arthrosc Tech. 2012;1(1):e5–10.
8. Matsuda DK. Arthroscopic osteosynthesis of a femoral head malunion. Arthrosc Tech. 2013;3(1):e31–4.
9. Matsuda DK. Fluoroscopic templating technique for precision arthroscopic rim trimming. Arthroscopy. 2009;25(10):1175–82.
10. Telleria JJ, Safran MR, Harris AH, Gardi JN, Glick JM. Risk of sciatic nerve traction injury during hip arthroscopy – is it the amount or duration? An intraoperative nerve monitoring study. J Bone Joint Surg Am. 2012;94(22):2025–32.
11. Kocher MS, Frank JS, Nasreddine AY, Safran MR, Philippon MJ, Sekiya JK, Kelly BT, Byrd JW, Guanche CA, Martin HD, Clohisy JC, Mohtadi NG, Griffin DR, Sampson TG, Leunig M, Larson CM, Ilizaliturri Jr VM, McCarthy JC, Gambacorta PG. Intra-abdominal fluid extravasation during hip arthroscopy: a survey of the MAHORN group. Arthroscopy. 2012;28(11):1654–60.
12. Jamali AA et al. Minimally invasive bone grafting of cysts of the femoral head and acetabulum in femoroacetabular impingement: arthroscopic technique and case presentation. Arthroscopy. 2010;26(2): 279–85.
13. Epstein NJ, Safran MR. Stress fracture of the acetabular rim: arthroscopic reduction and internal fixation. J Bone Joint Surg Am. 2009;91:1480–6.
14. Larson CM, Stone RM. The rarely encountered rim fracture that contributes to both femoroacetabular impingement and hip stability: a report of 2 cases of arthroscopic partial excision and internal fixation. Arthroscopy. 2011;27(7):1018–22.
15. Starr AJ, Jones AL, Reinert CM, Borer DS. Preliminary results and complications following limited open reduction and percutaneous screw fixation of displaced fractures of the acetabulum. Injury. 2001;32 Suppl 1: SA45–50.
16. Hong G, Cong-Feng L, Cheng-Fang H, Chang-Qing Z, Bing-Fang Z. Percutaneous screw fixation of acetabular fractures with 2D fluoroscopy-based computerized navigation. Arch Orthop Trauma Surg. 2010;130: 1177–83.
17. Xian-Quan W, Jin-Fang C, Xue-Cheng C, et al. A quantitative anatomic study of plate-screw fixation of the acetabular anterior column through an anterior approach. Arch Orthop Trauma Surg. 2010;130: 257–62.
18. Yang JH, Chouhan DK, Oh KJ. Percutaneous screw fixation of acetabular fractures: applicability of hip arthroscopy. Arthroscopy. 2010;26(11):1556–61.
19. Shahulhameed A, Roberts CS, Pomeroy CL, Acland RD, Giannoudis PV. Mapping the columns of the acetabulum – implications for percutaneous fixation. Injury. 2010;41:339–42.
20. Bartlett CS, DiFelice GS, Buly RL, Quinn TJ, Green DS, Helfet DL. Cardiac arrest as a result of intraabdominal extravasation of fluid during arthroscopic removal of a loose body from the hip joint of a patient with an acetabular fracture. J Orthop Trauma. 1998;12:294–9.
21. Matsuda DK, Villamor A. The modified midanterior portal for hip arthroscopy. Arthroscopy Tech. 2014. In Press.

Femoral Head Fractures

78

Jaimo Ahn and Mara L. Schenker

Contents

Abstract

Fractures of the femoral head are rare high-energy injuries that are commonly associated with posterior dislocations of the hip. Controversy exists regarding many aspects of treatment of these fractures, but following emergent reduction of associated hip dislocations, anatomic reduction of large fracture fragments, removal of loose intra-articular fragments, and restoration of hip joint stability and congruity are recommended. Reported outcomes on these fractures continue to be relatively poor, despite advances in approaches and implant design. High rates of posttraumatic arthritis, avascular necrosis, and heterotopic ossification are common. The purpose of this chapter is to summarize the available literature on femoral head injury mechanisms and epidemiology, classification systems, and treatment options (operative vs. nonoperative and associated outcomes) and to propose potential mechanisms for improving outcomes in these rare, high-energy injuries.

Introduction

Dr. John Birkett described the first reported fracture of the femoral head in 1869 in a 35-year-old woman who fell from a second-story window in London, sustaining a fractured skull and a "lacerated brain," and on further examination, she was noted to have "a portion of the head of the femur

J. Ahn (✉) • M.L. Schenker
Orthopaedic Trauma and Fracture Service, Department of Orthopaedic Surgery, University of Pennsylvania, Philadelphia, PA, USA
e-mail: Jaimo.ahn@uphs.upenn.edu; jaimo_ahn@yahoo.com; mara.schenker@uphs.upenn.edu

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_86

had been broken off. This fragment, to which the greater part of the ligamentum teres was still attached, remained in the acetabulum." [1] Nearly 150 years later, outcomes of these injuries remain generally poor, with high rates of posttraumatic arthritis, avascular necrosis, and heterotopic ossification, and optimized treatment algorithms are lacking. The purpose of this chapter is to summarize the available literature on femoral head injury mechanisms and epidemiology, classification systems, and treatment options (operative vs. nonoperative and associated outcomes) and to propose potential mechanisms for improving outcomes in these rare, high-energy injuries.

The Normal Hip Joint: An Inherently Stable Articulation

The articulation between the proximal femur and the acetabulum is inherently stable, due to the deep bony constraints, thick surrounding fibrous capsule, and ligamentous support, consisting of the iliofemoral, pubofemoral, and ischiofemoral ligaments. Numerous methods have been described to map the complex distribution of stresses in the hip joint during everyday activities, including finite element modeling [2, 3], pressure-sensitive film [4], and implanted prosthetic sensor-equipped models [5]. Over 70 % of the femoral head has been shown to be a "weight-bearing" surface [4]. The thickest cartilage in the femoral head is in the anterosuperior quadrant, corresponding to the area of large stress distribution seen during standing up and lifting, with the hip joint flexed [6]. Small femoral and acetabular cartilage incongruencies have been shown to yield large effects on overall contact stresses, explaining the high rates of posttraumatic arthritis in femoral head fractures and arguing for the need for anatomic reduction of these fractures [3].

The blood supply to the femoral head has been well described previously by Ganz and colleagues [7]. The primary blood supply to the femoral head consists of the terminal branches of the deep branch of the medial circumflex femoral artery, which originates from the profunda femoris artery and travels along the posterosuperior femoral neck as the superior retinacular artery, and enters the bone just beyond the articular margin. Further anatomic study has revealed the inferior vincular artery as an important source of blood flow to the femoral head with extensive intraosseous anastamoses between the superior retinacular artery, inferior vincular artery, and the subfoveal plexus [8]. Understanding the vascular anatomy of the femoral head is essential to avoid iatrogenic injury during surgical repair of femoral head fractures.

Femoral Head Fractures: Epidemiology and Clinical Evaluation

Femoral head fractures are rare. A recent systematic review reported the incidence to be 11.7 % in patients with hip dislocations [9]. Most studies evaluating femoral head fractures as a result are small case series, and hence, the development of optimized treatment algorithms has been limited.

Femoral head fractures result from an axial force transmitted along the long axis of the femur, as most commonly seen in a "dashboard injury." The position of the hip at the time of the load transmission, in terms of flexion, abduction, and direction of force applied, determines the injury pattern and severity. If the hip is flexed and adducted, a simple dislocation often results; however, if the hip is extended and abducted, the axial compression is directed more into the hip joint, creating a shear injury, and subsequent fracture, between the femoral head and the posterior wall [10]. In addition, a more anteverted femoral neck may predispose simple dislocation over associated fracture injuries [11].

A systematic review reporting mechanisms of 453 femoral head fractures demonstrated that motor vehicle collisions were the most common mechanism (84 %), followed by motorcycle collisions (5 %) and falls (4 %) [9]. They are commonly associated with hip dislocations, specifically posterior hip dislocations in 82–94% of femoral head fractures. They are often associated with posterior wall fractures of the acetabulum. Less commonly, femoral head fractures are associated with anterior hip dislocations; however, the pattern of damage to the femoral head differs from the patterns seen in posterior dislocations and is often an "indention" or transchondral-type fracture [12]. In addition, femoral head fractures are associated with fractures of the femoral neck and other "dashboard injuries" (including patella fractures and other knee injuries) [13]. As femoral head fractures are often the result of high-energy impact, concomitant injuries to the skeletal and other systems are common and necessitate a thorough secondary and tertiary examination.

A complete imaging workup is necessary to guide treatment of femoral head fractures. A standard trauma series anterior-posterior (AP) pelvis radiograph is used to assess associated dislocation patterns, often demonstrating a noncongruent hip joint, with the femoral head displaced superiorly, overlapping the acetabulum, with disruption of Shenton's line (Fig. 1). In addition, the AP radiograph should be closely inspected for associated fractures of the femoral neck before any closed manipulation of the hip joint is attempted. Judet radiographs are helpful in diagnosing any associated acetabular fractures. Computed tomography (CT) should be obtained routinely after successful closed reduction and before open reduction of an irreducible femoral head. CT should be inspected for femoral head fracture patterns (location and size), the presence of incarcerated intra-articular fragments, joint congruence, and associated fractures (posterior wall, femoral neck).

Classification

Although several classification systems have been described for femoral head fractures, the system proposed by Pipkin in 1957 is the most commonly used [14] (Fig. 2). It describes femoral head fractures based on the location relative to the fovea capitis, as well as associated bony injuries to the hip articulation.

Another system proposed by Brumback in 1987 is more comprehensive than the Pipkin classification and includes five types, including central and anterior hip dislocations, as well as A and B subtypes [15] (Fig. 3).

The Arbeitsgemeinschaft für Osteosynthesefragen (AO) classification of femoral head fractures includes 31-C1 (split), 31-C2 (split/depression), and 31-C3 (associated femoral neck fractures) [16] (Fig. 4).

Management

Initial Management

A fracture-dislocation of the hip is considered to be an orthopedic emergency. Provided that there is no contraindication to closed reduction (e.g., femoral neck fracture, clear intra-articular blockage to

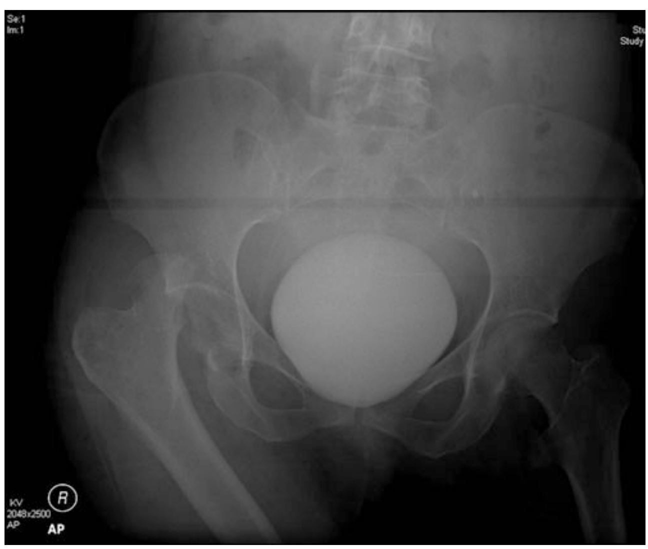


Fig. 1 Anteroposterior radiograph demonstrating right hip dislocation, with superior displacement of the proximal femur, disruption of Shenton's line, and the femoral head fragment of a Pipkin Type I injury remaining in the acetabulum (Figure from Kloen et al. [16])

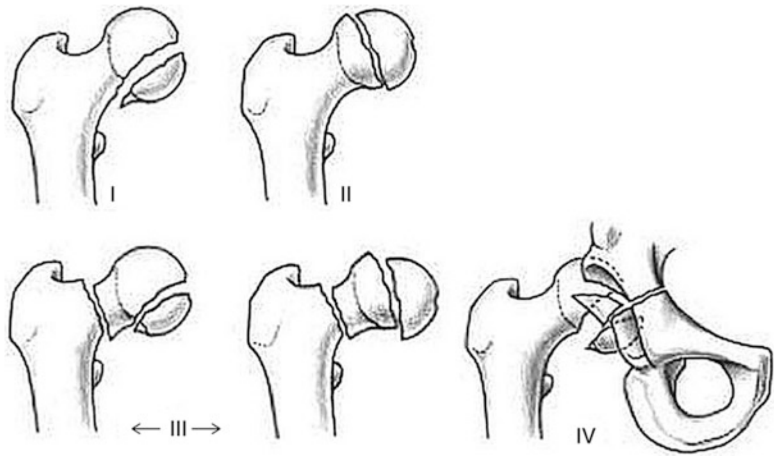


Fig. 2 The Pipkin classification: Type I (fracture of the head caudal to the fovea), Type II (fracture of the head cephalad to the fovea), Type III (Type I or II fracture with femoral neck fracture), Type IV (Type I or II fracture with acetabular fracture) (Figure from Kloen et al. [16])

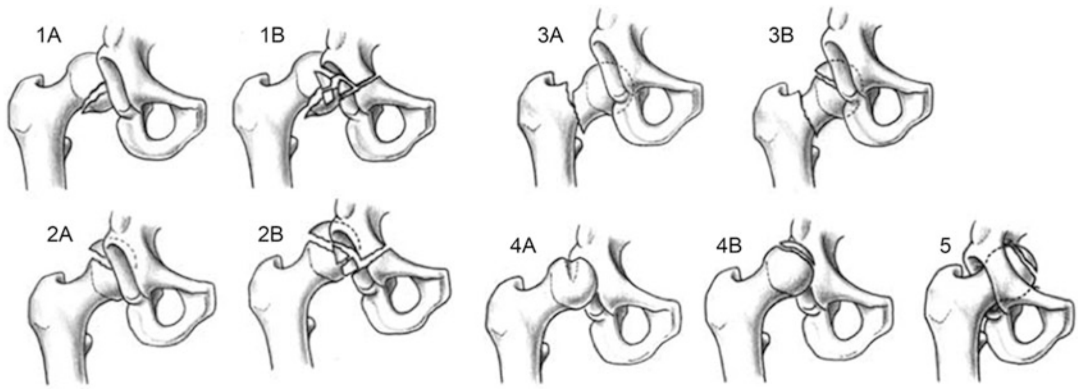


Fig. 3 The Brumback classification: Type I, posterior hip dislocation with femoral head fracture involving the inferomedial portion of the femoral head (IA, minimal or no fracture of the acetabular rim and stable hip joint after reduction; IB, significant acetabular rim fracture and hip joint instability); Type 2, posterior hip dislocation with femoral head fracture involving the superomedial, non-weight-bearing portion of the femoral head (IIA, minimal or no fracture of the acetabular rim and stable hip joint after reduction; IIB, significant acetabular rim fracture and hip joint instability); Type 3, dislocation of the hip with associated femoral neck fracture (IIIA, no associated femoral head fracture; IIIB, associated femoral head fracture); Type 4, anterior dislocation of the hip with fracture of the femoral head (IVA, indentation type; IVB, transchondral shear); Type 5, central fracture-dislocation of the hip with fracture of the femoral head (Figure from Kloen et al. [16])

reduction), immediate closed reduction should be attempted. A recent systematic review showed that 84 % of femoral head fracture-dislocations are reducible in the closed setting [9]. Lower rates of avascular necrosis have been reported when the hip is reduced within 6 h [17]. It has been recommended that multiple vigorous attempts at closed reduction be avoided due to risk of further trauma, including fracture to the femoral neck [9]. Further, a case series of irreducible superior femoral head fracture-dislocations has been presented in which the femoral head was found to be buttonholed through the capsulolabral junction with common findings of superior dislocation on x-ray and a clinical constellation of hip position in slight and fixed flexion and absent hip rotation, with leg length discrepancy [18]. The authors recommended that closed reduction should not be attempted in patients with this presentation, due to risk of iatrogenic femoral neck

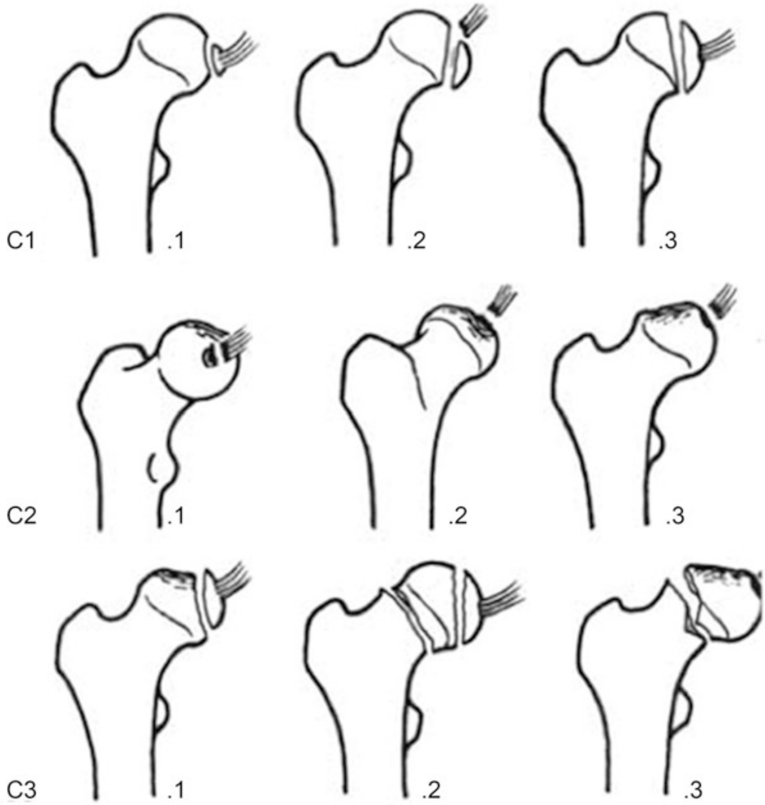


Fig. 4 The AO classification: 31-C1 (split), 31-C2 (split/depression), 31-C3 (associated femoral neck fractures) (Figure from Kloen et al. [16])

fracture. If closed reduction is unsuccessful or deemed to be too risky to be attempted, emergent open reduction should be performed.

Following closed reduction, repeat AP pelvis radiographs and a CT should be obtained to further evaluate the femoral head fracture pattern (location and size), presence of incarcerated intra-articular fragments, and joint congruence and better define any associated acetabular fractures to help guide further management.

Definitive Management

The overall goals of definitive management are to create a stable, concentric hip joint to maximize function and decrease the risk of future posttraumatic arthritis. Numerous methods have been proposed to this end, ranging from nonoperative management to surgical excision to open reduction and internal fixation to arthroplasty. Due to the rarity of these injuries, an optimized treatment algorithm has not been well defined, and small case series and personal experiences have guided the operative indications, surgical approach, and fixation strategies listed in this section.

Nonoperative Treatment

Historically, most femoral head fractures were treated with prolonged bed rest, immobilization, and traction [14]. For numerous reasons, this has largely been abandoned. However, it has been suggested that nonoperative indications still remain for small Pipkin I/II fractures, in which the femoral head is anatomically or near-anatomically (within 2 mm) reduced and the hip joint is stable and congruent, without interposed articular fragments. However, it has been shown in one study that only 1 in 12 femoral head fractures met those criteria for acceptable closed reduction [10], indicating a modern shift towards operative management of most femoral head fractures. In a small, randomized control trial comparing closed treatment to surgical excision of Pipkin I fractures, functional outcome scores of operatively treated patients were superior to those managed nonoperatively [19]. Hence, recent data suggests that even Pipkin Type I and II fractures may be better treated operatively; however, the exact criteria for acceptable fracture size, fracture location, joint stability, and joint concentricity are not well defined.

If nonoperative treatment is selected, the patient is restricted to partial weight bearing for approximately 3 months, and serial radiographs should be obtained frequently to ensure maintenance of fracture and joint reduction.

Operative Treatment

Most femoral head fractures are now managed operatively. Operative indications include all irreducible femoral head fracture-dislocations, Pipkin III injuries, Pipkin IV injuries (inherently unstable), and Pipkin I/II fractures with large, displaced fragments or any fracture-dislocation combination in which intra-articular fragments create a nonconcentric joint articulation. The goals of operative management are to create an anatomic reduction and stable articulation and to remove loose fragments. There has been significant debate as to the optimal approach (arthroscopic vs. open, various open approaches), treatment option (excision vs. ORIF vs. arthroplasty), and fixation strategy for these injuries.

Surgical Approach: Arthroscopy

A recent report suggested that incidence of hip arthroscopy performed for all indications has increased by over 600 % from 2006 to 2010 [20]. Consistent with this increase, recent studies have evaluated the utility of this technique in treating fractures of the femoral head and acetabulum [21–23], including removal of loose articular fragments and in arthroscopic-assisted open reduction and internal fixation of femoral head fragments.

Mullis et al. performed a diagnostic arthroscopy of 36 patients who sustained simple hip dislocations or fracture-dislocations that did not meet their indications for open surgical management [22]. They found loose bodies in 33 of 36 patients, including 7 of 9 patients who were not found to have fragments on standard postreduction x-ray and CT scans. They were able to successfully remove the loose bodies from the joint and concluded that arthroscopy is a good tool for removing loose fragments in hips that otherwise do not require open treatment. Further, Matsuda reported the successful arthroscopic treatment of a suprafoveal femoral head fracture, fixed with two Herbert screws [23].

Proposed advantages of the arthroscopic approach to femoral head fracture treatment include improved access for removal of loose bodies, less disruption to the capsuloligamentous structures, minimal blood loss, and faster recovery time [24]. Proposed disadvantages include technical difficulty, as well as other standard hip arthroscopy risks (traction neuropraxia, injury to lateral femoral cutaneous nerve, avascular necrosis, intraperitoneal fluid extravasation, retroperitoneal fluid extravasation) [24].

Surgical Approach: Open Techniques

Numerous open surgical approaches to the hip joint have been described for use in femoral head fractures, including medial (Ludloff), direct lateral (modified Hardinge), posterior (Kocher-Langenbeck), anterior (Smith-Peterson, modified Hueter), anterolateral (Watson-Jones), and, more recently, the trochanteric flip (digastric) osteotomy [13]. Each of these approaches has advantages and disadvantages, and ultimately, the approach should be decided based upon the location of the femoral head fracture, the treatment plan (excision vs. fixation), associated injuries (acetabular, femoral neck), and the direction of the dislocation.

The anterior approaches to femoral head fractures include the Smith-Peterson approach and modified Hueter approach. Historically, the anterior approaches were discouraged due to the belief that they would further compromise the femoral head blood supply, increasing the risk of avascular necrosis, by surgical ligation of the ascending branch of the lateral circumflex femoral artery. However, recent cadaveric studies have disproven this by demonstrating that the medial circumflex femoral artery is the dominant blood supply to the femoral head, with almost no contribution from the lateral circumflex femoral artery [7].

Advantages to anterior approaches include improved visualization of anterior-based femoral head fractures for reduction and fixation, decreased operative time, and lower rates of AVN when compared to posterior approaches [24–26]. Disadvantages include higher rates of heterotopic ossification (HO) compared to posterior approaches, decreased visualization of associated posterior wall fractures, and decreased access posteriorly, especially critical in the presence of irreducible incarcerated fracture-dislocations [24–26].

To address some of the downsides in visualization of the femoral head with the anterior approach, a modified Smith-Peterson approach has been described, in which anterior dislocation of the femoral head is possible for improved circumferential access of the femoral head [24]. The surgical technique is described in detail in the next chapter.

The posterior approach to femoral head fractures involves the standard Kocher-Langenbeck approach. This was historically favored for the treatment of femoral head fractures, as it was postulated that blood supply to the femoral head would be damaged after posterior hip dislocations and that further damage to the blood supply during an anterior surgical procedure was not advisable. As mentioned above, this has been disproven with recent understanding of the vascular anatomy of the femoral head [7]. The posterior approach, however, is advantageous for irreducible, incarcerated femoral head fracture-dislocations, in which posterior soft tissues, including the piriformis tendon and sciatic nerve, can block reduction of the femoral head. In addition, it is a favorable approach in femoral head injuries with associated acetabular fractures. A reported disadvantage of the standard posterior approach for femoral head fractures is an increased rate of AVN, recently reported in a meta-analysis to be 3.67 times higher compared to anterior approaches and 2.24 times higher compared to the trochanteric flip approach [9]. The main disadvantage to the posterior approach is that femoral head access is limited and reduction and fixation of anterior femoral head fractures are often difficult.

The trochanteric flip osteotomy, or digastric osteotomy, was originally described by Ganz and colleagues [27]. In this technique, a posterior approach to the hip joint is modified by the addition of a digastric trochanteric osteotomy to allow for anterior dislocation of the hip joint and preservation of the posterior blood supply (medial femoral circumflex artery). This approach, popularized for use in treatment of femoroacetabular impingement, has been applied for the treatment of femoral head fractures [28]. The surgical technique is described in detail in the next chapter.

Advantages of the digastric osteotomy in the treatment of femoral head fractures include complete access to the femoral head and acetabulum for reduction and fixation, with good outcomes reported for Pipkin IV injuries [29]. Disadvantages to the digastric osteotomy include inherent risk of injury to the femoral head blood supply and trochanteric nonunion.

In summary, the surgical approach selected for treatment of femoral head injuries should be made based on the location of the femoral head fracture, the treatment plan for the femoral head fracture (excision vs. fixation), the treatment plan for associated injuries (acetabular, femoral neck), and the need to reduce any irreducible dislocations (Table 1).

Surgical Treatment: Excision Versus ORIF Versus Arthroplasty

Although open reduction has become popularized in the treatment of femoral head fracture-dislocations, controversy remains as to whether the fragment should be excised or internally fixed and when arthroplasty should be performed.

Historically, femoral head fractures were treated with simple fragment excision, if the fragment comprised less than 1/3 the surface area of the femoral head. This was supported by a biomechanical study, which showed no changes in load and mean and peak pressures when excision was

Table 1 Surgical approaches to femoral head fractures

Approach	Advantages	Disadvantages
Anterior (Smith-Peterson, modified Smith-Peterson, modified Hueter)	Excellent access to femoral head fracture for reduction and fixation	Increased HO rate (42.1 %, 16 of 38) [26]
	Decreased operative time	Poor access to acetabulum for associated fractures
	Decreased AVN rate [26] (7.9 %, 3 of 38)	Poor access posteriorly for irreducible dislocations
Anterolateral (Watson-Jones)	Good access to femoral head fracture for reduction and fixation	Increased HO rate (60 %, 3 of 5) [26]
	Decreased AVN rate (0 %, 0 of 5) [26]	Increased risk of injury to superior gluteal nerve
		Poor access to acetabulum for associated fractures
		Poor access posteriorly for irreducible dislocations
Direct lateral (Hardinge)	Good access to femoral head fracture for reduction and fixation	Requires disruption of abductors
	Decreased AVN rate (1 of 9) [26]	Poor access to acetabulum for associated fractures
	Decreased HO rate (0 of 9) [26]	Poor access posteriorly for irreducible dislocations
Posterior (Kocher-Langenbeck)	Excellent access to posterior wall of acetabulum	Poor access anteriorly for visualization and fixation of femoral head fractures
	Excellent access posteriorly for irreducible dislocations	Increased AVN rate (16.4 %, 11 of 67) [26], 3.67 times higher than anterior approach [9], 2.24 times higher than trochanteric flip approach [9]
	Decreased HO rate (24.8 %, 24 of 67) [26]	
Trochanteric flip (digastric osteotomy)	Complete visualization of femoral head, acetabulum	Intermediate AVN rate (12.5 %, 3 of 24) [26]
		Intermediate HO rate (33 %, 8 of 24) [26]

performed in the infrafoveal region. Conversely, excision of suprafoveal or large fragments was found to significantly alter the loading pattern in the hip joint [30]. A recent systematic review showed that there was a statistical trend towards better outcomes in Pipkin I fractures treated with excision, compared to open reduction and internal fixation. The authors concluded that this may be the result of a Type II statistical error; however, if operative treatment is considered for these injuries, excision of small fragments may be an adequate intervention [9]. With the conflicting available data, the authors' preference is that fragments large enough to fix anatomically should be fixed, and any remaining fragments should be excised.

For open reduction and internal fixation of femoral head fragments, after anatomic reduction, temporary fixation may be obtained with Kirschner wires. Definitive fixation is then performed with interfragmentary positional or lag screws. Numerous implants have been successfully used, including countersunk 2.7 mm or 3.5 mm screws, headless compression screws, and bioabsorbable pins [31]. When a large portion of the femoral head is involved and reconstruction of small, comminuted segments is not feasible, osteochondral allograft is a reasonable option in younger patients [32] and arthroplasty (hemiarthroplasty or total hip arthroplasty) in older patients.

For Pipkin III injuries, with associated femoral neck fractures, emergent anatomic surgical reduction and fixation of the femoral neck fracture should be performed first. Once adequate reduction and fixation is obtained, the femoral head fracture may be addressed through the same approach (anterior or anterolateral approaches) or through a separate exposure if needed.

For Pipkin IV injuries, with associated acetabular fractures, the type of acetabular fracture dictates the approach to the femoral head. For the typical associated posterior wall fracture, the posterior wall can be accessed through a traditional Kocher-Langenbeck approach and the femoral head with the addition of a digastric osteotomy described above [28]. For acetabular fractures involving the anterior column, the ilioinguinal or Stoppa approach may be utilized with a Smith-Peterson extension to address the femoral head fracture [13].

Outcomes of Femoral Head Fractures

The overall outcome of femoral head fractures is poor, with high rates of posttraumatic arthritis, avascular necrosis, heterotopic ossification, and poor functional scores. The various case series reporting outcomes of these injuries are limited by small sample sizes, variety of included injury severities and treatments, and the lack of long-term follow-up.

In a recent systematic review, Giannoudis et al. reported the outcomes on 405 femoral head fractures. Irrespective of treatment or fracture type, they found 14.3 % excellent, 39.8 % good, 19.3 % fair, and 26.5 % poor results. Results were worse for Pipkin III and IV injuries, compared to Pipkin I and II injuries, with the former reporting 33.3 % poor results and the latter reporting 11.8 % poor results [9].

In terms of complications of these injuries, Giannoudis et al. reported early complications of infection (3.2 %) and nerve injury (pure dislocation 1 %, femoral head fracture 1 %, associated acetabular fracture 24 %). Late complications were also common: posttraumatic arthritis (20 %), heterotopic ossification (16.8 %), and avascular necrosis (11.8 %).

A more recent long-term study reported outcomes on 21 patients with femoral head fractures at an average follow-up of 81 months (range, 12–210 months). Their series included Pipkin 1 fractures (4), Pipkin 2 fractures (9), and Pipkin 4 fractures (8). The authors concluded that nearly all of the patients had some evidence of posttraumatic arthritis on radiographs (95 %), which were mild (47.6 %), moderate (33.3 %), and severe (14.2 %). Although degenerative changes were found with all treatment types and injury severities, the most notable severe changes occurred in the patients treated with surgical excision of fragments.

Summary

Fractures of the femoral head are rare high-energy injuries that are commonly associated with posterior dislocations of the hip. Thorough physical examination and radiographic evaluation are necessary to adequately characterize the hip injury but also to recognize other associated injuries in these frequently polytraumatized patients. Treatment principles include emergent reduction of associated hip dislocations, anatomic reduction of the fracture, removal of loose intra-articular fragments, and restoration of hip joint stability and congruity. Reported outcomes on these fractures continue to be relatively poor, despite advances in approaches and implant design. High rates of posttraumatic arthritis, avascular necrosis, and heterotopic ossification are common. Future well-designed studies are needed to optimize treatment algorithms.

References

1. Birkett J. Description of a dislocation of the head of the femur, complicated with its fracture; with remarks. Med Chir Trans. 1869;52:133–8.
2. Chen GX, Yang L, Li K, et al. A three-dimensional finite element model for biomechanical analysis of the hip. Cell Biochem Biophys. 2013;67(2):803–8.
3. Harris MD, Anderson AE, Henak CR, Ellis BJ, Peters CL, Weiss JA. Finite element prediction of cartilage contact stresses in normal human hips. J Orthop Res. 2012;30(7):1133–9.
4. Greenwald AS, O'Connor JJ. The transmission of load through the human hip joint. J Biomech. 1971;4(6): 507–28.
5. Rushfeldt PD, Mann RW, Harris WH. Improved techniques for measuring in vitro the geometry and

pressure distribution in the human acetabulum. II Instrumented endoprosthesis measurement of articular surface pressure distribution. J Biomech. 1981;14(5): 315–23.

6. Armstrong CG, Gardner DL. Thickness and distribution of human femoral head articular cartilage. Changes with age. Ann Rheum Dis. 1977;36(5): 407–12.
7. Gautier E, Ganz K, Krugel N, Gill T, Ganz R. Anatomy of the medial femoral circumflex artery and its surgical implications. J Bone Joint Surg Br. 2000;82(5): 679–83.
8. Grose AW, Gardner MJ, Sussmann PS, Helfet DL, Lorich DG. The surgical anatomy of the blood supply to the femoral head: description of the anastomosis between the medial femoral circumflex and inferior gluteal arteries at the hip. J Bone Joint Surg Br. 2008;90(10):1298–303.
9. Giannoudis PV, Kontakis G, Christoforakis Z, Akula M, Tosounidis T, Koutras C. Management, complications and clinical results of femoral head fractures. Injury. 2009;40(12):1245–51.
10. Henle P, Kloen P, Siebenrock KA. Femoral head injuries: which treatment strategy can be recommended? Injury. 2007;38(4):478–88.
11. Upadhyay SS, Moulton A, Burwell RG. Biological factors predisposing to traumatic posterior dislocation of the hip. A selection process in the mechanism of injury. J Bone Joint Surg Br. 1985;67(2): 232–6.
12. DeLee JC, Evans JA, Thomas J. Anterior dislocation of the hip and associated femoral-head fractures. J Bone Joint Surg Am. 1980;62(6):960–4.
13. Droll KP, Broekhuyse H, O'Brien P. Fracture of the femoral head. J Am Acad Orthop Surg. 2007;15(12): 716–27.
14. Pipkin G. Treatment of grade IV fracture-dislocation of the hip. J Bone Joint Surg Am. 1957;39-A(5):1027–42 passim.
15. Brumback RJ, Kenzora JE, Levitt LE, Burgess AR, Poka A. Fractures of the femoral head. Hip. 1987;1987:181–206.
16. Kloen P, Siebenrock KA, Raaymaker E, Marti RK, Ganz R. Femoral head fractures revisited. Eur J Trauma. 2002;4:221–33.
17. Hougaard K, Thomsen PB. Coxarthrosis following traumatic posterior dislocation of the hip. J Bone Joint Surg Am. 1987;69(5):679–83.
18. Mehta S, Routt Jr ML. Irreducible fracture-dislocations of the femoral head without posterior wall acetabular fractures. J Orthop Trauma. 2008;22(10):686–92.
19. Chen ZW, Lin B, Zhai WL, et al. Conservative versus surgical management of Pipkin type I fractures associated with posterior dislocation of the hip: a randomised controlled trial. Int Orthop. 2011;35(7):1077–81.
20. Bozic KJ, Chan V, Valone III FH, Feeley BT, Vail TP. Trends in hip arthroscopy utilization in the United States. J Arthroplasty. 2013;28(8 Suppl):140–3.
21. Yamamoto Y, Ide T, Ono T, Hamada Y. Usefulness of arthroscopic surgery in hip trauma cases. Arthroscopy. 2003;19(3):269–73.
22. Mullis BH, Dahners LE. Hip arthroscopy to remove loose bodies after traumatic dislocation. J Orthop Trauma. 2006;20(1):22–6.
23. Matsuda DK. A rare fracture, an even rarer treatment: the arthroscopic reduction and internal fixation of an isolated femoral head fracture. Arthroscopy. 2009; 25(4):408–12.
24. Ross JR, Gardner MJ. Femoral head fractures. Curr Rev Musculoskelet Med. 2012;5(3):199–205.
25. Swiontkowski MF, Thorpe M, Seiler JG, Hansen ST. Operative management of displaced femoral head fractures: case-matched comparison of anterior versus posterior approaches for Pipkin I and Pipkin II fractures. J Orthop Trauma. 1992;6(4):437–42.
26. Guo JJ, Tang N, Yang HL, Qin L, Leung KS. Impact of surgical approach on postoperative heterotopic ossification and avascular necrosis in femoral head fractures: a systematic review. Int Orthop. 2010;34(3):319–22.
27. Ganz R, Gill TJ, Gautier E, Ganz K, Krugel N, Berlemann U. Surgical dislocation of the adult hip a technique with full access to the femoral head and acetabulum without the risk of avascular necrosis. J Bone Joint Surg Br. 2001;83(8):1119–24.
28. Gardner MJ, Suk M, Pearle A, Buly RL, Helfet DL, Lorich DG. Surgical dislocation of the hip for fractures of the femoral head. J Orthop Trauma. 2005;19(5): 334–42.
29. Solberg BD, Moon CN, Franco DP. Use of a trochanteric flip osteotomy improves outcomes in Pipkin IV fractures. Clin Orthop Relat Res. 2009;467(4):929–33.
30. Holmes WJ, Solberg BD, Bay BK, Laubach JE, Olson SA. Biomechanical consequences of excision of displaced Pipkin femoral head fractures. J Orthop Trauma. 2000;14(2):149–50.
31. Prokop A, Helling HJ, Hahn U, Udomkaewkanjana C, Rehm KE. Biodegradable implants for Pipkin fractures. Clin Orthop Relat Res Mar. 2005;432:226–33.
32. Nousiainen MT, Sen MK, Mintz DN, et al. The use osteochondral allograft in the treatment of a severe femoral head fracture. J Orthop Trauma. 2010;24(2): 120–4.

79

Surgical Technique: Open Hip Dislocation, Open Reduction, and Internal Fixation

Mara L. Schenker, Samir Mehta, and Jaimo Ahn

Contents

Abstract

Femoral head fractures are high-energy injuries, often associated with posterior hip dislocations. Outcomes from these injuries, irrespective of treatment modality, have historically been very poor, with high reported rates of posttraumatic arthritis, avascular necrosis (AVN), and heterotopic ossification. Much controversy remains as to the best treatment algorithm for femoral head fractures and femoral head fracture-dislocations, including the optimal surgical approach. This review highlights two described techniques of surgical dislocation: digastric (trochanteric flip) osteotomy and modified Smith-Petersen. Surgical dislocation of the hip provides significant advantages over traditional approaches, including complete visualization of the femoral head for fracture reduction and fixation, access to the joint for debridement, and access to the acetabulum for fixation of associated injuries.

Introduction

Femoral head fractures are high-energy injuries, often associated with posterior hip dislocations. Outcomes from these injuries, irrespective of treatment modality, have historically been very poor, with high reported rates of posttraumatic arthritis, avascular necrosis (AVN), and heterotopic ossification. The previous chapter highlighted the injury patterns frequently

M.L. Schenker (✉) • J. Ahn (✉)
Orthopaedic Trauma and Fracture Service, Department of Orthopaedic Surgery, University of Pennsylvania, Philadelphia, PA, USA
e-mail: mara.schenker@uphs.upenn.edu; Jaimo.ahn@uphs.upenn.edu

S. Mehta
Hospital of the University of Pennsylvania, Orthopaedic Surgery, Penn Orthopaedics, Philadelphia, PA, USA
e-mail: samir.mehta@uphs.upenn.edu

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_87

observed and the current treatment algorithm for femoral head fractures. The purpose of this review is to describe the technique for surgical dislocation of the hip to address femoral head fractures and associated injuries, specifically the relevant vascular anatomy, and the techniques of digastric trochanteric flip osteotomy and modified Smith-Petersen dislocation.

Vascular Anatomy

Understanding the vascular anatomy of the proximal femur and femoral head is of paramount importance to minimize iatrogenic injury during surgical exposure and treatment of femoral head fractures. The incidence of AVN after femoral head injury and hip dislocations is highly variable in the reported case series; however, a recent systematic review showed the overall rate to be 12 % following treatment of femoral head fractures [1]. The precise etiology of post-injury femoral head necrosis is not known, but proposed mechanisms include acute vessel disruption; compression, or kinking; and also delayed thrombosis and fibrosis. Changes in femoral head perfusion have been noted in both short duration (1 h) hip dislocations and prolonged dislocations (24 h) [2], indicating that a prompt reduction is likely not enough to diminish the risk of subsequent AVN. The overall risk of AVN following femoral head fracture injury and treatment is likely multifactorial, including injury characteristics (e.g., associated injuries, time period between dislocation and reduction), host characteristics (e.g., coagulopathies, smoking history), as well as iatrogenic injury of the complex vascular anastomoses during surgical exposure.

The precarious nature of the blood supply to the femoral head has been described as far back as 1822, when Astley Cooper noted that the predominant supply to the femoral head passed along the femoral neck and also through a small subsidiary vessel in the ligamentum teres [3]. Tucker and Trueta performed early cadaveric studies defining the intraosseous anatomy of the femoral head, noting that the predominant supply to the epiphysis came from the lateral epiphyseal branches of the superior retinacular artery. A lesser portion of the epiphysis, particularly the perifoveal region, was found to be supplied by the medial epiphyseal vessels of the inferior retinacular artery [3, 4]. In the last two decades, Ganz and colleagues have performed several anatomic studies and have stimulated further interest in other groups in defining the extracapsular vascular anatomy of the proximal femur, with the goal to define safe zones for surgical approaches to the hip joint which minimize iatrogenic risk of AVN.

The medial femoral circumflex artery (MFCA) is the primary blood supply to the femoral head. There are five described branches of the MFCA, including the acetabular branch, which yields the medial epiphyseal and inferior retinacular vessels, and the deep branch of the MFCA, which yields the superior retinacular and lateral epiphyseal vessels (Table 1). All together, the deep branch of the MFCA and its branches have been shown to be the predominant vascular supply to the femoral head. Gautier et al. defined the anatomy of the deep branch of the MFCA [5], and Ganz later described the digastric trochanteric osteotomy highlighted below as a safe way to approach the entirety of the hip joint, without injuring this artery and without increasing the risk of AVN [6]. It is important to recognize that several studies have shown the importance of an anastomosis with the between the inferior gluteal artery and the MFCA, at the level of the piriformis [5, 7, 8]. This anastomosis may be capable of compensating after an injury to the deep MFCA and needs to be preserved during surgical approach.

Table 1 The five branches of the medial circumflex femoral artery

Branch	Path
Superficial	Courses between pectineus and adductor longus
Ascending	To adductor brevis, adductor magnus, and obturator externus
Acetabular	Gives off foveolar artery (medial epiphyseal artery)
Descending	Courses between quadratus femoris and adductor magnus
Deep	To the head of the femur

Surgical Technique: Digastric (Trochanteric Flip) Osteotomy

The patient is positioned in the lateral decubitus position using a radiolucent beanbag or hip positioners on a standard flat-top radiolucent table [6, 9]. A Kocher-Langenbeck (gluteus maximus split) or modified Gibson exposure (anterior to gluteus maximus) can be utilized to expose and access the proximal femur and femoroacetabular joint. The hip is then internally rotated, and electrocautery is used to identify the posterior margin of the gluteus medius and vastus lateralis insertions on the proximal femur. The planned osteotomy is pre-drilled to accept at least three 3.5 mm screws in an effort to minimize difficulty with reduction and fixation of the osteotomy at the completion of the case. A trochanteric osteotomy is performed using an oscillating saw and broad osteotome, creating a wafer of bone approximately 1.5 cm in thickness. At its proximal limit, the osteotomy should exit just anterior to the most posterior fibers of the gluteus medius; this preserves and protects the deep branch of the MFCA, which becomes intracapsular at the level of the superior gemellus. The remaining gluteus medius fibers are then released, as are the posterior fibers of the vastus lateralis, to the level of the gluteus maximus insertion on the femur. By performing these releases, the trochanteric wafer is then mobilized anteriorly via the interval between the piriformis and the gluteus medius, exposing the anterior capsule. The capsule is then incised in a z-shaped fashion along the anterolateral femoral neck, with the medial limb directed posteriorly, parallel to, and just lateral to the labrum. The lateral limb must remain anterior to the lesser trochanter in order to protect the MFCA, which lies just superior and posterior to it. The hip is then dislocated anteriorly with gentle flexion and external rotation. The ligamentum teres may need to be incised, allowing the femoral head to dislocate, offering a complete view of the femoral head and acetabular socket. Once exposed, any damage to the labrum or acetabulum can be visualized and treated and loose intra-articular bodies may be removed.

Small comminuted fragments of the femoral head may then be excised, particularly fractures that are caudal to the fovea. Care should be taken excising fragments in patients with ipsilateral posterior wall fractures. For fragments large enough to fix, it is the authors' opinion and practice to anatomically fix them. Temporary fixation may be obtained with Kirschner wires. Definitive fixation is then performed with interfragmentary positional or lag screws. Numerous implants have been successfully used, including countersunk 2.7 mm or 2.4 mm screws (flat-headed screws), headless compression screws, and bioabsorbable pins [10]. Regardless of device selected, it is paramount to recess the implant to allow a smooth articulation following fixation. Once the fracture is stabilized, the hip is reduced with traction, extension, and internal rotation. The hip is cycled through a full physiologic range of motion to ensure a concentric reduction and smooth articulation. The capsule may be repaired, but not tightened, since this may create tension on the retinacular vessels and a decreased femoral head perfusion [11]. The trochanter is then reattached using three 3.5 mm cortical screws, pre-drilled previously, directed towards the lesser trochanter.

Postoperatively, the patient is mobilized immediately. The patient is maintained with toe-touch or touch-down weightbearing with crutches (or walker) and on hip precautions for the first 6 weeks if posterior dislocation was present. Low molecular weight heparin is given for thromboprophylaxis during hospitalization until the patient is mobile, and aspirin is continued for up to 6 weeks postoperatively. The authors do not currently use prophylaxis for heterotopic ossification. Radiographs are obtained to evaluate trochanteric and femoral head healing at 6 weeks and every 6 weeks until union is achieved.

Surgical Technique: Modified Smith-Petersen Exposure with Dislocation

The patient is placed in the supine position on a standard flat-top radiolucent table. An incision is made from the anterior superior iliac spine distally towards the lateral border of the patella for 12–16 cm [12]. The lateral femoral cutaneous nerve is identified and protected, and the interval between the tensor fascia lata (retracted laterally) and the sartorius (retracted medially) is bluntly divided, exposing the deep interval. The gluteus medius is identified and retracted laterally, while the common tendon of the rectus femoris is divided proximally and tagged for later repair. The branches of the lateral femoral circumflex artery are identified and can be ligated, if needed to extend the exposure. The hip is then flexed to approximately 30–45°, and the iliocapsularis muscle is dissected off of the capsule and retracted medially. The capsule is then incised in a T shape, along the anterior neck, and extending superiorly and inferiorly along the acetabular rim, just lateral to the labral insertion. Retraction stitches are placed in the corners of the incision to aid with retraction and later repair. At this point, if the femoral head fracture is adequately exposed, fixation may be performed as described in the previous section. If exposure is inadequate, the hip may be dislocated. Pharmacologic relaxation is critical, and the hip is dislocated anteriorly with gentle traction and external rotation and sharp division of the ligamentum teres. At this point, near complete access to the femoral head is achieved for assessment of associated acetabular injuries, removal of loose bodies, and reduction and fixation of the femoral head fractures as described above. Once fixation is achieved, the hip may be reduced with traction and internal rotation. The hip is then cycled through a full range of motion to ensure concentric reduction and smooth motion. The capsule is repaired, but not tightened, since this may create tension on the retinacular vessels and decrease in femoral head perfusion [11]. The iliocapsularis muscle is then debrided and the rectus femoris tendon is repaired.

Postoperatively, the patient is mobilized immediately. The patient is maintained toe-touch weightbearing with crutches for 6 weeks with hip precautions if dislocation was posterior in direction. Low molecular weight heparin is given for thromboprophylaxis during hospitalization until the patient is mobile, and aspirin is continued up to 6 weeks postoperatively. The authors do not currently use prophylaxis for heterotopic ossification. Radiographs are obtained to evaluate femoral head healing at 6 weeks, and every 6 weeks until union is achieved.

Case 1

A 36-year-old female was transferred from an outside hospital following a motor vehicle collision. She presented with a shortened, externally rotated right hip, as well as multiple other injuries, including splenic laceration, pulmonary contusions, rib fractures, and aortic dissection. She was stabilized at the original trauma center, including emergent intubation, and splenic embolization. Vascular consultation recommended no need for aortic intervention. Upon transfer, orthopedics was urgently consulted, and a thorough neurologic examination of the limb could not be obtained due to her intubated and sedated status.

Initial radiographs of the right hip are shown in Fig. 1a, which demonstrated a Pipkin IV fracture-dislocation, with a small posterior wall acetabular fragment and a femoral head fragment contained within the joint (Fig. 1b). The remaining femoral head/neck/shaft was dislocated posteriorly. A single attempt at a closed reduction was performed and was unsuccessful. The patient was then urgently taken to the operating room for open reduction. A digastric trochanteric flip osteotomy approach was used, and the femoral head fracture was anatomically fixed with countersunk flat-headed 2.4 mm cortical screws (Fig. 1c). Given the stability of the femoral head in the acetabulum and the size of the posterior wall fragment, the acetabular fracture was not surgically repaired. The trochanteric fragment was stabilized with two 3.5 mm cortical screws directed towards the lesser trochanter (Fig. 1d).

Postoperatively, the patient maintained toe-touch weightbearing with crutches and hip precautions for 6 weeks. She received thromboprophylaxis with low molecular weight heparin while hospitalized and then a total of 6 weeks of aspirin following discharge. At 6 months postoperatively (her last visit), she had

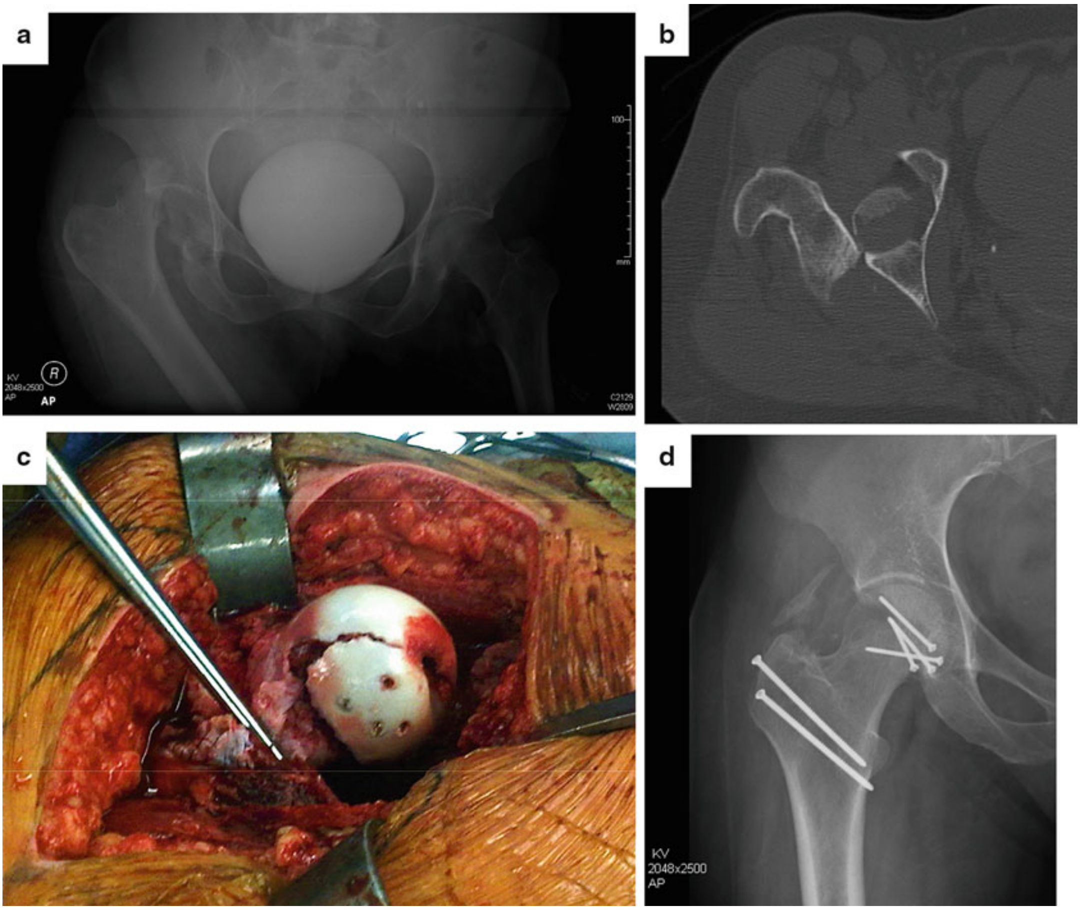


Fig 1 A 36-year-old female presented with a traumatic *right* hip fracture-dislocation, with a large, displaced intra-articular femoral head fracture (**a**). CT scan demonstrated the complex fracture pattern (**b**). Using a digastric trochanteric flip osteotomy, the articular surface was visualized and the large fragment anatomically repaired using countersunk 2.4 mm cortex screws (**c**). Postoperative radiographs at 6 months reveal an anatomic fracture reduction with minimal heterotopic ossification and no evidence of radiographic posttraumatic arthritis or avascular necrosis (**d**)

no pain in the hip, had full range of motion, and was back to full activities of daily living. Her radiographs demonstrated a healed fracture and osteotomy, mild heterotopic ossification (Brooker class III), and no evidence of posttraumatic arthritis or AVN.

Case 2

A 45-year-old female presented to the trauma bay following an unrestrained rollover motor vehicle collision with an isolated shortened left lower extremity without apparent rotational deformity. Initial radiographs are shown in Fig. 2a, which demonstrate a Pipkin IV fracture-dislocation of the left hip, with a posterior wall acetabular fracture and an infra-foveal femoral head fracture.

A closed reduction attempt was successful in the trauma bay following intubation and paralysis. A postreduction CT scan was obtained (Fig. 2b, c), and preoperatively, it was decided to proceed with surgical fixation of the femoral head fracture, without fixation of the posterior wall acetabular fracture.

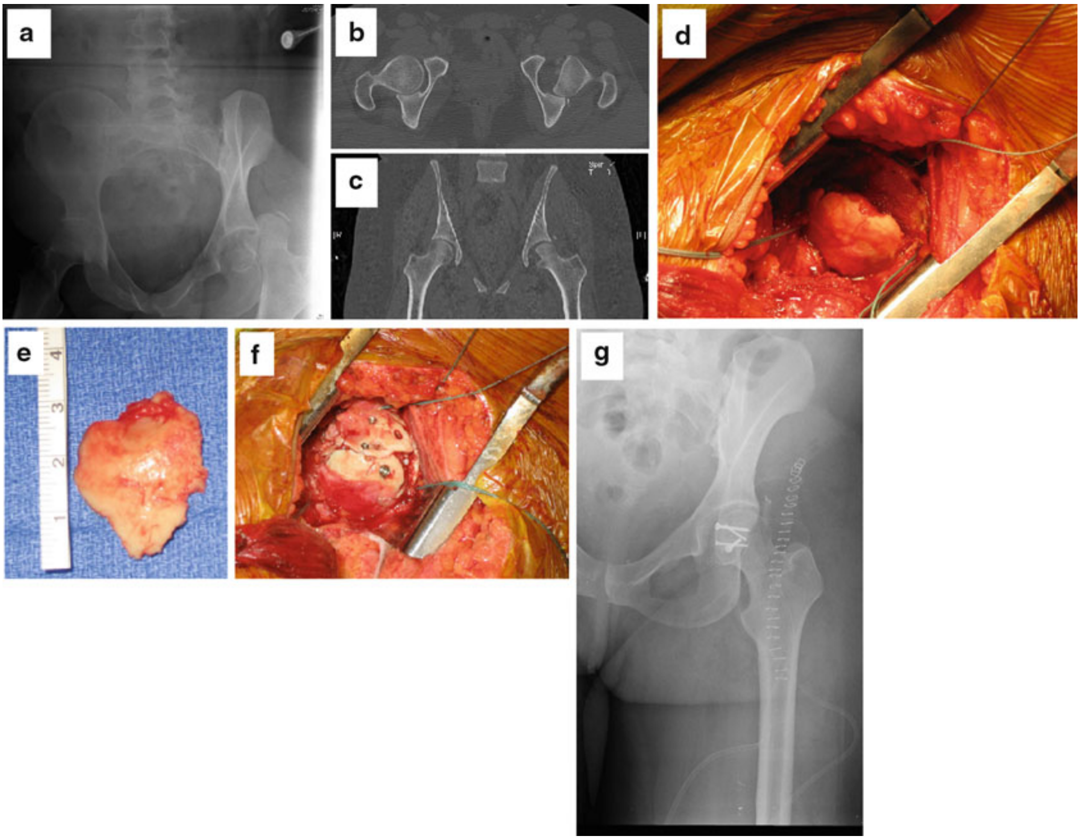


Fig 2 A 45-year-old female presented to the trauma bay with a traumatic *left* hip fracture-dislocation with a large, displaced femoral head fracture (**a**). CT scan demonstrated the complex fracture pattern (**b**, **c**). Using a Smith-Petersen dislocation approach, the articular surface was visualized and the large fragment anatomically repaired using countersunk flat-headed 2.0 mm and 2.7 mm cortical screws (**d**–**f**). Postoperative radiographs reveal an anatomic fracture reduction (**g**)

The patient was then semi-urgently taken to the operating room for open reduction and internal fixation. A Smith-Petersen exposure with dislocation was used (Fig. 2d–f), and the femoral head fracture was anatomically fixed with countersunk flat-headed 2.0 mm and 2.7 mm cortical screws (Fig. 2g).

Postoperatively, the patient maintained toe-touch weightbearing with crutches and hip precautions for 6 weeks. She received thromboprophylaxis with low molecular weight heparin while hospitalized and then a total of 6 weeks of aspirin following discharge. Three years postoperatively, she has no hip pain and full range of motion of her hip.

Summary

Much controversy remains as to the best treatment algorithm for femoral head fractures and femoral head fracture-dislocations, including the optimal surgical approach. Surgical dislocation of the hip provides significant advantages over traditional approaches, including complete visualization of the femoral head for fracture reduction and fixation, access to the joint for debridement, and access to the acetabulum for fixation of associated injuries. The techniques described above for hip dislocation via the Smith-Petersen and digastric trochanteric flip osteotomy have been shown to be safe and effective at treating these injuries. Outcomes studies are necessary to evaluate long-term postoperative function, as well as the development of any subsequent complications, including AVN, arthritis, and heterotopic ossification.

References

1. Giannoudis PV, Kontakis G, Christoforakis Z, Akula M, Tosounidis T, Koutras C. Management, complications and clinical results of femoral head fractures. Injury. 2009;40(12):1245–51.
2. Yue JJ, Sontich JK, Miron SD, et al. Blood flow changes to the femoral head after acetabular fracture or dislocation in the acute injury and perioperative periods. J Orthop Trauma. 2001;15(3):170–6.
3. Tucker FR. Arterial supply to the femoral head and its clinical importance. J Bone Joint Surg Br. 1949; 31B(1):82–93.
4. Trueta J, Harrison MH. The normal vascular anatomy of the femoral head in adult man. J Bone Joint Surg Br. 1953;35-B(3):442–61.
5. Gautier E, Ganz K, Krugel N, Gill T, Ganz R. Anatomy of the medial femoral circumflex artery and its surgical implications. J Bone Joint Surg Br. 2000;82(5): 679–83.
6. Ganz R, Gill TJ, Gautier E, Ganz K, Krugel N, Berlemann U. Surgical dislocation of the adult hip a technique with full access to the femoral head and acetabulum without the risk of avascular necrosis. J Bone Joint Surg Br. 2001;83(8):1119–24.
7. Kalhor M, Beck M, Huff TW, Ganz R. Capsular and pericapsular contributions to acetabular and femoral head perfusion. J Bone Joint Surg Am. 2009; 91(2):409–18.
8. Grose AW, Gardner MJ, Sussmann PS, Helfet DL, Lorich DG. The surgical anatomy of the blood supply to the femoral head: description of the anastomosis between the medial femoral circumflex and inferior gluteal arteries at the hip. J Bone Joint Surg Br. 2008;90(10):1298–303.
9. Gardner MJ, Suk M, Pearle A, Buly RL, Helfet DL, Lorich DG. Surgical dislocation of the hip for fractures of the femoral head. J Orthop Trauma. 2005; 19(5):334–42.
10. Prokop A, Helling HJ, Hahn U, Udomkaewkanjana C, Rehm KE. Biodegradable implants for Pipkin fractures. Clin Orthop Relat Res. 2005;432:226–33.
11. Notzli HP, Siebenrock KA, Hempfing A, Ramseier LE, Ganz R. Perfusion of the femoral head during surgical dislocation of the hip. Monitoring by laser Doppler flowmetry. J Bone Joint Surg Br. 2002; 84(2):300–4.
12. Ross JR, Gardner MJ. Femoral head fractures. Curr Rev Musculoskelet Med. 2012;5(3):199–205.

Atraumatic Instability and Surgical Technique

80

Joshua D. Harris, William Slikker III , Geoffrey D. Abrams, and Shane J. Nho

Contents

J.D. Harris (✉)
Houston Methodist Center for Orthopaedics & Sports Medicine, Houston, TX, USA

Rush University Medical Center, Chicago, IL, USA
e-mail: joshuaharrismd@gmail.com

W. Slikker III
Midwest Orthopaedics at Rush, Chicago, IL, USA
e-mail: wslikker@gmail.com

G.D. Abrams
Department of Orthopaedic Surgery, Stanford University, Palo Alto, CA, USA
e-mail: gabrams@gmail.com

S.J. Nho
Department of Orthopaedic Surgery, Rush University Medical Center, Chicago, IL, USA

Midwest Orthopaedics at Rush, Hip Preservation Center, Rush University Medical Center, Chicago, IL, USA
e-mail: shane.nho@rushortho.com

Abstract

The normal hip has a natural tendency to stability due to its depth, congruency, and surrounding contractile and inert tissues. Hip instability may occur either with or without trauma. Hip microinstability may also occur with or without trauma. However, microinstability is a concept that is currently unproven, but sound anatomically, biomechanically, and radiographically, and with limited in vivo clinical studies. In the absence of other clear sources for persistent hip symptoms despite treatment, the astute clinician may diagnose microinstability. However, microinstability may also be the cause or the effect of other concomitant hip pathologies. If prior surgery has been performed, the operative report, photographs, and videos should be scrutinized in detail, especially in regard to osseous, chondrolabral, and capsuloligamentous management. Patients should be assessed for generalized hypermobility. Certain subjects (such as young female gymnasts, ballet, dance, yoga) may be at particular risk. Impingement-induced instability may also be an underlying contributor, especially in males with cam deformities. The examiner must assess the difference between laxity (asymptomatic) and instability (symptomatic). The true location of pain and tenderness, motion, and strength should be evaluated. Both radiographic and advanced imaging may be indicated. Initial management of microinstability

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_88

Table 1 Concept of hip microinstability; FAI (femoroacetabular impingement)

Study author(s)	Year	Journal title	Definition
Cerezal et al.	2012	Eur J Radiol	Inability to keep femoral head centered within acetabular fossa, without complete luxation or marked subluxation of the joint
			Hip laxity is not equivalent to microinstability (presence of symptoms = microinstability)
			Overuse and repetitive motion is most common cause of microinstability
			Microinstability may lead to concomitant intra- and extra-articular injury
Domb et al.	2013	Arthroscopy	Microinstability may lead to ligamentum teres tears, large labral tears, and advanced acetabular chondral damage
Amenabar et al.	2012	Arthroscopy	Idiopathic instability that occurs in absence of trauma, dysplasia, overuse, or connective tissue disorder
			Ligamentum teres has role as hip stabilizer and should be included in the evaluation of stability
Guanche et al.	2005	Arthroscopy	Eight elite runners (mean age 36 years) underwent hip arthroscopy for labral tear
			Six subjects had Outerbridge Grade III acetabular articular cartilage injury
			Three subjects had complete avulsions of the ligamentum teres
			Theorize repetitive hyperextension during stride leads to “subtle instability” or “recurrent subluxation” and attritional stress at chondrolabral junction and ligamentum teres
Kolo et al.	2013	Skeletal Radiol	Professional ballet dancers
			MRI-documented joint subluxation in all hips in the “splits” position with only 1/59 hips demonstrating cam or pincer FAI
			In comparison to control, ballet dancers had significantly more acetabular chondral lesions, labral tears, and herniation pits

should be nonoperative. In patients that have failed conservative treatment, arthroscopic evaluation for diagnostic and therapeutic purposes may be undertaken. Without femoroacetabular impingement or labral pathology, examination under anesthesia may better reveal subtle loss or excessive motion asymmetries. Capsulorrhaphy (plication) with or without ligamentum teres management may provide successful clinical outcome. Short-term results are successful. However, mid- and long-term outcomes do not yet exist.

Introduction

The normal hip has a natural tendency to stability due to its depth, congruency, and surrounding contractile and inert tissues. Hip instability may occur either with or without trauma. Hip microinstability may also occur with or without trauma. However, microinstability is a concept that is currently unproven, but sound anatomically, biomechanically, and radiographically, and with limited in vivo clinical studies (Table 1). In the absence of other clear sources for persistent hip symptoms despite treatment, the astute clinician may diagnose microinstability. However, microinstability may also be the cause or the effect of other concomitant hip pathologies. Patients should be assessed for generalized hypermobility. There is a significant difference between laxity (asymptomatic) and instability (symptomatic), and this must be established in active discussion with the patient.

Relevant Anatomy

Capsulotomy is necessary to navigate the joint for both diagnostic and therapeutic purposes. However, many authors now contend that iatrogenic

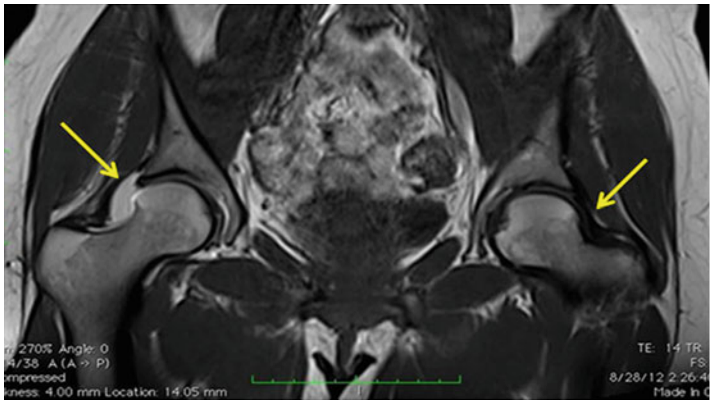


Fig. 1 T1-weighted coronal series right magnetic resonance hip arthrogram. In comparison to the normal left hip, the post-arthroscopic right hip (with capsulotomy left open) demonstrates capsular opening from the acetabular rim with dye extrusion (Modified from McCormick et al. [37] with kind permission from Springer Science and Business Media)

instability may result if the capsulotomy is not closed (Fig. 1). As the number of hip arthroscopies has increased dramatically (up to 18-fold) over the past decade [1], warranted concern has been emphasized in the size and type of capsulotomy performed and its routine closure [2]. Capsular preservation (minimal capsulotomy, routine closure) is encouraged for the retention of normal anatomy and optimizing outcomes. The iliofemoral ligament is the strongest of four discrete hip capsular ligaments (in addition to ischiofemoral, pubofemoral, zona orbicularis), and its primary purpose is to restrain anterior translation, with hip extension and external rotation. This static soft tissue stabilizer of the hip joint is disrupted during interportal (anterolateral to mid-anterior; Fig. 2) and “T” (Fig. 3) capsulotomies. Three recent cadaveric biomechanical studies (Table 2) have illustrated the importance of the iliofemoral ligament to the structural integrity of normal hip joint mechanics: Iliofemoral sectioning results in increased external rotation, extension, and anterior translation with no difference between the intact and repaired state [3–5].

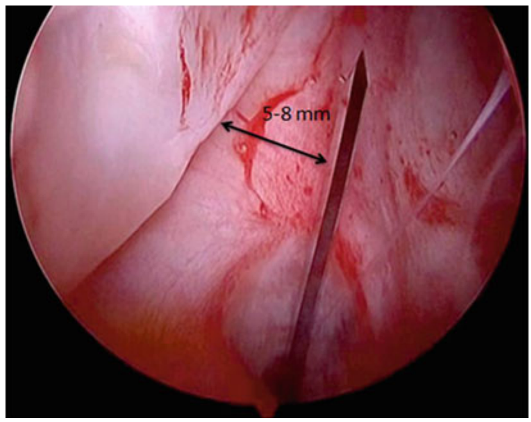


Fig. 2 Interportal capsulotomy. This incision is made in the right hip anterior capsule with the patient supine, in traction, using an arthroscopic scalpel, while viewing from anterolateral portal. It is important to leave a sufficient cuff of tissue on the acetabular side to repair at the conclusion of the case (Reprinted from Harris et al. [2] with permission from Elsevier)

The ligamentum teres is a strong hip joint stabilizer (Table 3). It becomes taut with hip flexion, adduction, and external rotation. Despite its name “teres” (Latin for “round”), the ligament is actually triangular to flattened in shape for part of its intra-articular course (approximately 30–35 mm) [6]. Its diameter is highly variable and circumferentially covered with synovium. In the normal hip, the synovium is not highly vascular to the naked eye or with the arthroscope. However, in a hip with Legg-Calve-Perthes disease, the synovium and the ligament itself may be thicker with significant inflammation [7]. In a developmentally dysplastic hip, the ligament may be abnormally hypertrophic (thicker, longer) [7]. In traumatic ligamentum teres ruptures, the acetabular side tears before the femoral side and intrasubstance tears are rare [8].

The ligamentum teres originates from the transverse acetabular ligament and spans the bony junction of the ischium-pubis from the 5 to 7 o’clock position on the acetabular clock face. It

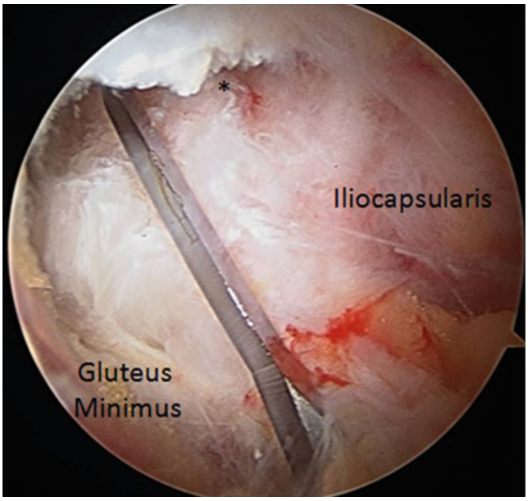


Fig. 3 "T" capsulotomy. This incision is made in the right hip anterior capsule over the anterosuperior femoral neck with the patient supine, in traction, using an arthroscopic scalpel, while viewing from anterolateral portal. The iliocapsularis (medial) and gluteus minimus (lateral) are the anatomic landmarks used to make the "T" in the capsule, affording >180° visualization of the peripheral compartment for femoral head/neck cam osteochondroplasty (Reprinted from Harris et al. [2] with permission from Elsevier)

inserts on the femoral side at the anterosuperior aspect of the fovea capitis, which is a bare area without articular cartilage, located posteroinferiorly on the femoral head. Within the ligament is the artery of the ligamentum teres, a branch usually derived from the posterior division of the obturator artery and less commonly from the acetabular branch of the medial femoral circumflex artery (medial epiphyseal artery). Regardless of its source, the amount of vascularity supplied to the femoral epiphysis is variable and minimal.

Table 2 Role of hip capsule in microinstability; IFL (iliofemoral ligament)

Study	Study design	Role
Myers et al. [3]	Cadaveric biomechanical	Increased external rotation with IFL sectioning (increased 12.9°) ($p <$ .0001)
	Fluoroscopy	Increased anterior translation with IFL sectioning (increased 1.8 mm) ($p <$ .001)
		No difference in external rotation or anterior translation between intact/repaired state
Martin et al. [4]	Cadaveric biomechanical	Release of medial, lateral arms IFL gave greatest increase of external rotation
	Motion tracking	Lateral arm release provides more motion in flexion and neutral
		Lateral arm release also provides more internal rotation, primarily in extension
Hewitt et al. [5]	Cadaveric biomechanical	IFL much stronger than the ischiofemoral ligaments
	Load to failure	IFL greater stiffness than ischiofemoral ligaments
		IFL greater tensile load to failure than ischiofemoral ligaments

Relevant Pathophysiology

The presence of microinstability does not preclude other concomitant intra- and extra-articular disorders. In fact, subtle instability may actually increase the stress response and potential damage to local structures including labrum, articular cartilage and underlying subchondral bone, ligamentum teres, iliopsoas tendon, iliotibial band, hip abductors, and the remaining normal capsule [9]. Further, FAI symptoms may be exacerbated due to the excessive motion [10]. Conversely, impingement may actually induce instability in the following four ways: (1) Excessive acetabular anteversion can result in anterior hip instability and posterior acetabular rim impingement; (2) excessive acetabular retroversion can result in anterior impingement and posterior instability; (3) excessive femoral anteversion can result in anterior hip instability and posterior acetabular rim impingement; (4) excessive femoral retroversion can result in anterior impingement and posterior instability. In

Table 3 Role of ligamentum teres in instability; CEA (center-edge angle)

Study author(s)	Year	Journal title	Role
Domb et al.	2013	Arthroscopy	Ligamentum teres tear risk factors assessed in 463 hips undergoing arthroscopy
			Hips with low lateral coverage index (lateral CEA – acetabular inclination) 1.74 times more likely to have ligamentum tear than those with high lateral coverage index
			Significantly greater number of ligamentum tears when labral tear extended posterior to 11 o'clock or anterior to 4 o'clock
			Significantly more advanced acetabular chondral damage seen in ligamentum tears
			Ligamentum teres tears speculated to be a sign of microinstability
Cerezal et al.	2012	Eur J Radiol	Stabilizer in adduction, flexion, external rotation
			Ligamentum teres tear may lead to microinstability
			Microinstability may lead to ligamentum teres tear
Haviv et al.	2011	Knee Surg Sports Traumatol Arthrosc	Arthroscopic debridement of 29 hips with isolated partial ligamentum teres tear provides significant pain relief and improvement in function
			High risk of recurrence associated with ligamentous laxity
			Partial ruptures are due to repetitive stretching and may be seen in isolation – mostly in young female gymnasts, dancers, and with calisthenics
Amenabar et al.	2012	Arthroscopy	Case report of young female with persistent pain after ligamentum debridement, anterior capsulorrhaphy. Underwent ligamentum reconstruction using semitendinosus autograft
			Successful 12-month outcome, but new lateral pain over suture knot at 15 months
			Repeat arthroscopy showed reconstruction sutures intact, but graft resorption

a study that examined the association between arthroscopic iliopsoas tenotomy and femoral version, significantly worse outcomes were observed in subjects with greater degrees of femoral anteversion (>25°) due to the loss of the anterior dynamic stabilizing effect performed by the iliopsoas [11].

Cam impingement prohibits true ball-and-socket mechanics, thus causing anterior levering over the rim (fulcrum) with subsequent posterior instability [12]. With traumatic posterior hip dislocation, there is a greater prevalence of anterior cams and femoral retroversion than in normal hips [13]. In an exclusively athletic population with low-energy subluxation or dislocation and posterior acetabular rim fracture, cam and/or pincer impingement has been identified in 64–82 % of subjects [14–16]. In athletes with larger degrees of motion (such as ballet, dancing, gymnasts), there may be impingement-induced instability without abnormal cam or pincer deformities. In a cohort of 59 professional ballet dancers, only one hip had evidence of a cam deformity, while several other abnormalities were identified on magnetic resonance imaging due to a dynamic "pincer" mechanism from the extreme motion involved with their sport [17]. Further, while in the "splits" position, all hips subluxated (mean 2.1 mm). In comparison to a control group, the abnormalities in ballet included significantly more acetabular cartilage lesions (mostly superior), more labral tears (mostly posterosuperior to anterosuperior), and more superior herniation pits. Despite the latter study's prevalence of imaging abnormalities, less

Table 4 Advantages and disadvantages of capsular closure during hip arthroscopy

Complete capsular repair or plication	Leaving capsulotomy open
Advantages	*Advantages*
Retention of hip stability	May be therapeutic (pre-op tight anterior capsule)
External rotation, anterior translation	Reduced surgical time
Prevention of iatrogenic subluxation, dislocation	No risk of overtightening anterior capsule and subsequent loss of motion
Prevention of edge loading of repaired labrum	
No loss of motion	
Disadvantages	*Disadvantages*
Technically demanding, may damage articular surfaces	May lead to iatrogenic instability, especially in ligamentously lax hypermobile individuals and in sport-specific athletes
Multiple nonabsorbable sutures in anterior capsule	Exacerbated during excessive femoral osseous resection
Increased surgical time	

than two-thirds of subjects were symptomatic at the time imaging was performed in a follow-up investigation [18]. Further, when examining certain ballet positions, the mean translation was up to 4.6 mm (6.4 mm in one subject) [19]. Whether or not the microinstability observed in these studies (with the associated intra-articular pathologies) will lead to early osteoarthritis is yet to be determined.

Discrete frank hip dislocation is at the end of the microinstability spectrum. In the postoperative setting, instability may occur along this spectrum to variable degrees. Nine cases of post-arthroscopic iatrogenic hip dislocation have been presented in the literature [10, 20–25]. Due to publication bias, this is likely a significant underestimate of the true incidence of instability following hip arthroscopy. Thus, many authors have subsequently recommended routine capsular closure during arthroscopy, which has both potential advantages and disadvantages (Table 4) [2]. The following patient-, hip-, and surgical technique-specific factors have been associated with this uncommon, but serious, complication: capsulotomy or capsulectomy without repair, labral resection (versus repair or refixation), aggressive acetabuloplasty rim trimming in dysplastic configurations, and overall capsular laxity [26]. In this situation, revision surgery (either arthroscopic or open) for capsulorrhaphy may be indicated.

Table 5 Ligamentum teres tear classification systems

Gray and Villar classification [27]	Botser classification [28]
I – Complete rupture	0 – no tear
II – Partial rupture	1 – <50 % tear
III – Degenerative ligament tear	2 – >50 % tear
	3–100 % tear

In addition to the capsule, other structures may be pathologically involved in microinstability. Ligamentum teres ruptures may be a sign of microinstability, or they may cause microinstability. Two classification systems exist to categorize these injuries (Table 5) [27, 28]. The more recently defined classification, a descriptive classification, was published in a level IV evidence retrospective case series of 616 arthroscopies in which 51 % had ligamentum teres ruptures [28]. It was observed that patients with tears had significantly greater range of motion, less lateral acetabular coverage, larger labral tears, and more advanced acetabular articular cartilage damage [28, 29]. Despite the possible role that the ligamentum teres may play in hip stability, counter to this argument is that it is commonplace for the ligament to be resected during open surgical dislocation femoral osteochondroplasty without residual instability following surgery. However, close history and examination does reveal that these patients do have subtle instability, similar to that of a partial (Gray and Villar Type II) ligamentum rupture, rather than a complete (Type I) rupture [30]. In addition to the capsuloligamentous structures, the musculotendinous dynamic structures that cross the joint may be weak, potentially altering the axial/appendicular skeleton mechanics and leaving the joint less stable.

Diagnosis

The diagnosis of hip microinstability is based upon a thorough history and physical examination. The presence of other concomitant diagnoses such as labral tear or impingement does not preclude the diagnosis of microinstability. It may occur in patients with generalized ligamentous laxity (congenital or acquired). Patients with Ehlers-Danlos, Marfan, Down, and/or undiagnosed ligamentous laxity syndromes may be hypermobile and at risk for microinstability [31]. In addition, patients with developmental hip dysplasia may be at risk for microinstability due to the lack of femoral head coverage and inherent congruency-dependent joint stability. A history of other dislocated joints may be elicited (shoulder or patella). Acquired causes of microinstability may be either traumatic or atraumatic. Following hip dislocation or subluxation, a resultant elongated position of the capsuloligamentous structures may predispose to microinstability. Without traumatic injury or definitive hypermobility syndrome, the diagnosis is often elusive and may be made only after exclusion of other sources of pain [31]. Patients with microinstability may complain of their leg "giving out," snapping, or buckling in provocative positions, during everyday activities of daily living, and with sports [9, 31].

In patients that have already undergone a hip arthroscopy, a full review of the surgeon's history, physical examination, imaging, operative report, and operative photographs and/or videos should be done. Attention should be paid to the indications for surgery, especially the patient's complaints of pain location, duration, exacerbating, and relieving factors. Further, the patient's response to a preoperative injection should be known. A new detailed physical examination should elicit residual impingement, iliopsoas tendonitis, or residual anterior capsular defect (capsulotomy performed but not closed) with resultant microinstability. In this setting, microinstability may be a diagnosis of exclusion. If the prior surgeon performed a capsulotomy, one should see the type and size and whether or not it was closed. On the contrary, it is also possible that too much plication was performed and anterior capsular tightness with adhesion formation is a cause of symptoms. A comparison of pre- and postoperative imaging should identify the presence of residual cam and/or pincer impingement.

Physical examination of the hip should assess for all sources of hip, lumbosacral spine, abdominopelvic, and lower extremity causes. A single sine qua non examination technique for the diagnosis of microinstability does not exist. A 9-point Beighton score may be measured (Table 6) [32]. Further history and examination may assess for the Brighton criteria [32] to make a diagnosis of Benign Joint Hypermobility Syndrome (BJHS) [33]. Core, pelvic, hip, and lower extremity strength should be assessed to evaluate if weakness is contributing to instability. While supine, loss of logroll recoil when the hip is allowed to drift into normal external rotation is observed in subjects with attenuated anterior capsuloligamentous structures [34]. Gait assessment should evaluate the presence or absence of an abductor lurch or Trendelenburg gait (and Trendelenburg sign). If microinstability exists, proximate structures may be affected (Table 7).

Additional objective evidence in the setting of hip pain with suspected microinstability should include radiographs with or without magnetic resonance imaging (+/− arthrography) and/or three-dimensional computed tomography scanning (if indicated for bony morphology). Plain radiographs should include standing anteroposterior (AP) pelvis view, standing false profile view, and supine Dunn (45° and/or 90°) or frog-leg lateral radiographs. Advanced imaging should assess for labral abnormalities, subchondral edema, paralabral cyst, tenosynovitis, bursitis, effusion, loose bodies, and stress fracture.

Surgical Technique

Indications

Symptomatic patients should initially undergo conservative, nonsurgical management for suspected microinstability. Physical therapy with core, pelvic, hip abductor, gluteus maximus, and quadriceps/

Table 6 Beighton score [32] and Brighton criteria [33]; MCP (metacarpophalangeal joint)

Beighton score (0–9)		Brighton criteria	
Palms flat on floor while bending over at hips with knees fully extended		2 major	4 minor
		1 major + 2 minor	2 minor + 1 affected sibling
Left elbow hyperextends beyond 10°	Right elbow hyperextends beyond 10°	Major criteria	Minor criteria
		Beighton score ≥4	Beighton score 1–3
Left knee hyperextends beyond 10°	Right elbow hyperextends beyond 10°	Arthralgia for >3 months in ≥4 joints	Arthralgia in 1–3 joints or back pain
Passive flexion of left thumb to volar forearm	Passive flexion of right thumb to volar forearm		Dislocation in >1 joint or 1 joint >1 time
Passive extension of left small finger MCP beyond 90°	Passive extension of right small finger MCP beyond 90°		≥3 soft tissue lesions (bursitis, tenosynovitis)
			Marfanoid habitus
			Skin striae, thin, hyperextensibility
			Drooping eyelids, myopia, antimongoloid slant
			Varicose veins, hernia, rectal or uterine prolapse

Table 7 Secondary physical examination findings in setting of hip microinstability

Iliotibial band	*Labral tear*
Snapping over greater trochanter with flexion/extension with leg neutral/adducted in lateral decubitus position	Impingement signs
	Flexion, adduction, internal rotation
	Loss of passive internal rotation with flexed hip
Tenderness over greater trochanteric bursa or abductor tendon insertion	FABER/Patrick test (localized anteriorly to groin versus posteriorly over sacroiliac joint)
Positive Ober test	Positive painful circumduction arc
Trendelenburg gait, weak abductors	Positive McCarthy's test
Iliopsoas tendon	Positive logroll
Snapping over iliopectineal eminence or femoral head	DEXRIT test (dynamic external rotatory impingement)
When hip flexed 90°, tendon lateral to eminence; hip extension then displaces/snaps the tendon medially over the eminence	DIRI test (dynamic internal rotatory impingement)
Reproducible with arising from seat or stairs	Posterior rim impingement test (posterior labral/acetabular rim)
Pain with resisted hip flexion or active straight leg raise	
Thomas test (tight iliopsoas versus rectus femoris)	
"Fan" test – voluntary hip circumduction	

hamstrings strengthening should be initiated if muscular weakness is a component of instability. Musculotendinous units (e.g., iliotibial band, hamstrings, rectus femoris) should be stretched if abnormally tight. Oral anti-inflammatory medications may be liberally used if inflammation may be inciting pain. A trial of rest and activity modification should also be attempted. In patients that have failed these measures, response to an intra-articular injection of local anesthetic (with or without corticosteroid) may help differentiate an intra- and extra-articular disorder.

If nonoperative treatment is unsuccessful, then diagnostic arthroscopy with possible capsular plication to address capsular redundancy may be undertaken [35]. Preoperative imaging work-up should have already elucidated the presence or absence of common findings such as cam or pincer FAI, labral tear, osteoarthritis, capsular defect (if prior surgery; Fig. 1), effusion, bursitis, or tenosynovitis.

Technique

Surgeon preference may dictate arthroscopic setup (supine versus lateral position) and portal placement. Prior to sterile preparation and draping, examination under anesthesia should evaluate for bilateral hip range of motion and loss of recoil. The surgeon should also be cognizant of the force required for distraction, as there may be less force necessary in patients with subtle instability. Joint entry may be either via only interportal capsulotomy or both interportal and "T" capsulotomy. This permits central and peripheral compartment visualization and instrumentation. In patients with microinstability, double-loaded suture anchor labral refixation (Fig. 4) may allow for both labral refixation and capsular repair to the anatomic location on the acetabulum [36]. Upon the conclusion of central and peripheral compartment work (labral refixation, articular cartilage treatment, ligamentum teres management, acetabular rim trimming, and femoral osteochondroplasty [Fig. 5]), the surgeon is faced with the

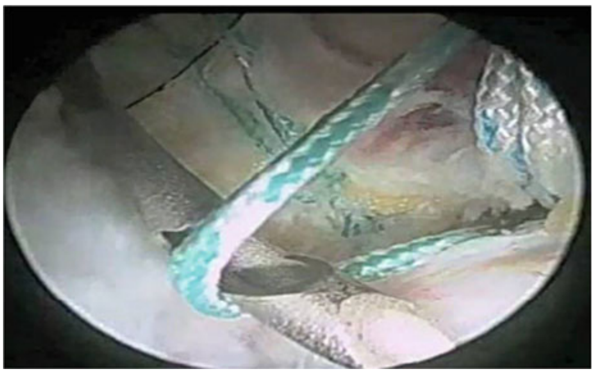


Fig. 4 Double-loaded suture anchor placed over acetabular labrum. One suture is utilized for secure labral fixation with one limb through the chondrolabral junction and the other limb through the base of the labrum to achieve a mattress configuration. The other suture in the anchor is utilized for capsular reattachment during closure of the interportal capsulotomy

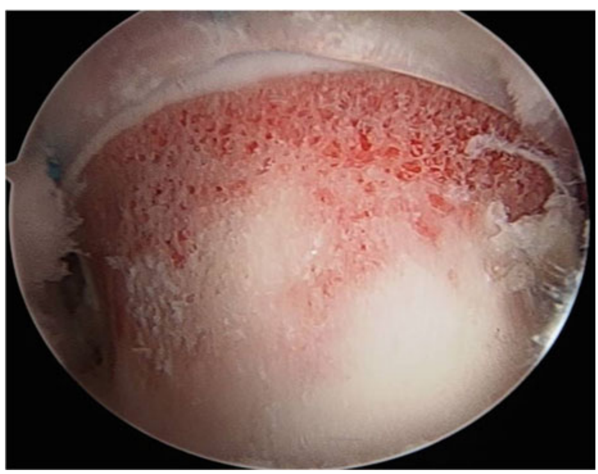


Fig. 5 Completed femoral cam osteochondroplasty demonstrating smooth articulation with the acetabular labrum without impingement

Fig. 6 SutureLasso (Arthrex, Inc., Naples, FL, USA) passing through capsular edge with nitinol wire for eventual suture passage

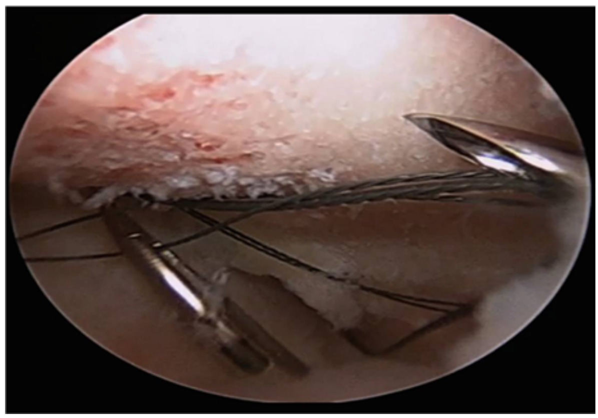


Fig. 7 Tissue penetrator BirdBeak (Arthex, Inc., Naples, FL, USA) passing through contralateral capsular edge to retrieve nitinol wire for suture passage

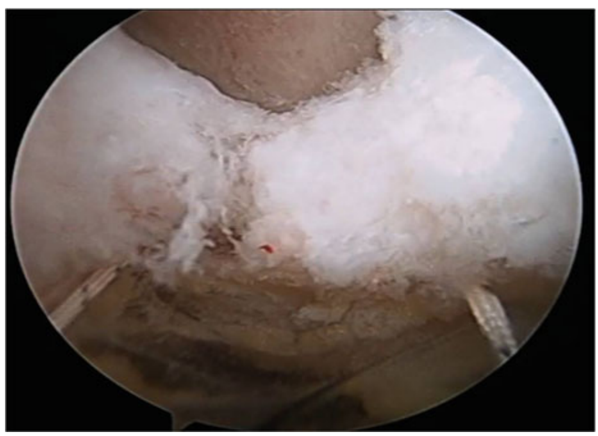


Fig. 8 High-strength nonabsorbable suture shuttled, ready to be tied

decision of capsular closure. Clearly, larger capsulotomies require more sutures for repair/plication. Thus, make the capsulotomy at the start of the case as large as necessary to address the pathology, but no larger. Avoid aggressive capsulectomy when exposing the acetabular rim for acetabuloplasty, as this may leave insufficient tissue for repair later. Further, if excessive tension is required to repair the capsule, this may predispose the patient to postoperative stiffness. However, in patients with generalized ligamentous laxity, greater degrees of plication may be warranted. The degree of repair or plication may be customized with the “size of the bite” taken from each capsular edge. Closure should begin at the most distal end of the “T” capsulotomy (near the intertrochanteric line) and proceed proximally toward the interportal capsulotomy. Visualization is reduced with each successive suture as advancing. It may be easier to tie each knot as they progress, rather than tie all sutures after they have all been passed.

Depending on the surgical indications and type and size of capsulotomy performed, there are three ways to close the capsule. Many hip arthroscopists agree that this part of the case is difficult and warrants precision in suture placement. Suture-shuttling systems (Arthrex SutureLasso, Naples, FL, USA; ConMed Linvatec Spectrum, Largo, FL, USA) are familiar to most shoulder arthroscopic surgeons, as the same principles may be applied to the hip. The suture shuttle instrument pierces one limb (Fig. 6) of the capsule and the shuttle bridges the capsular gap, while a tissue penetrator-passer pierces the other limb and retrieves the shuttle (Fig. 7). The shuttle then passes the suture (Fig. 8). The authors prefer high-strength, braided, nonabsorbable suture for closure, using standard arthroscopic knot-tying fundamentals (Fig. 9).

Automated suture-passing devices are also commonly utilized in capsular closure (InJector II, Pivot Medical, Sunnyvale, CA, USA). These instruments pierce the capsule, pass, and retrieve the suture through one capsular limb in one step (Fig. 10). When closing the interportal capsulotomy, pierce and pass suture on the acetabular side before the capsular side. While these devices appose the tissue edge to edge, other newer devices allow for greater degrees of single-step capsular plication. One device (CapsulePass, Pivot Medical, Sunnyvale, CA,

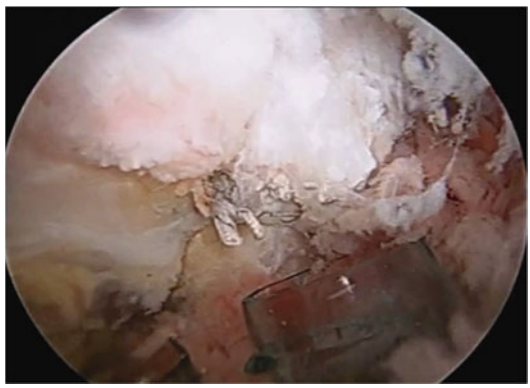


Fig. 9 Appearance of tied arthroscopic suture knot via reversing half hitches on an alternating post configuration

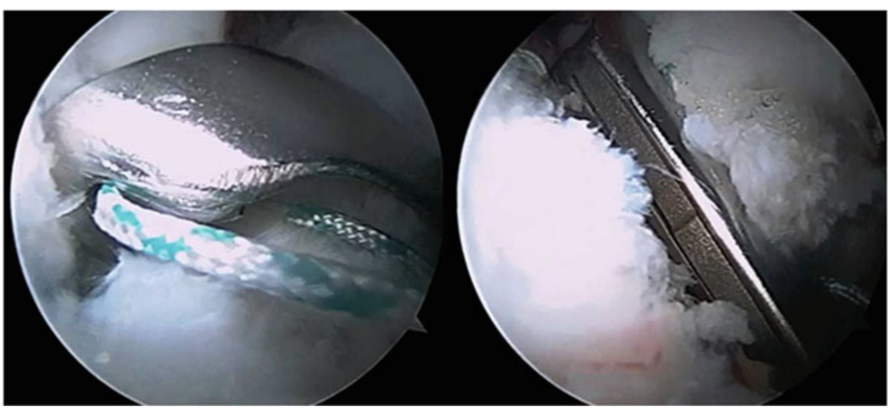


Fig. 10 Automated capsular closure device (InJector II) grasps acetabular side first (*left*) and then capsular side second (*right*)

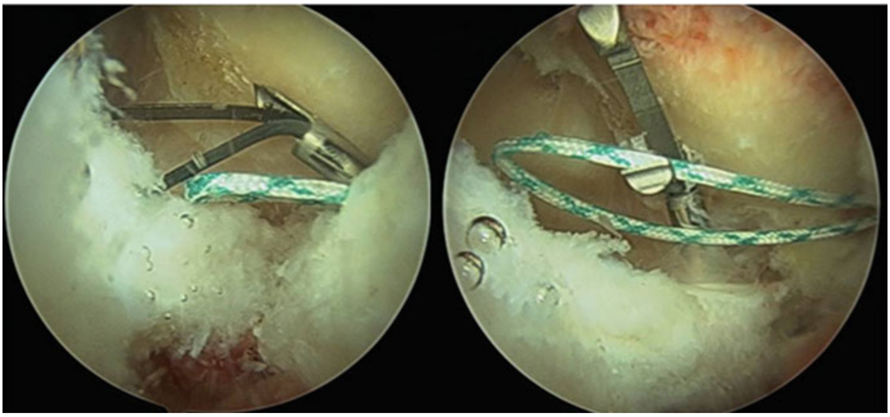


Fig. 11 Single-step suture passer and retriever (CapsulePass) pierces one capsular limb (*left*), passes the suture, and, then via same cannula, retrieves it through the other capsular limb (*right*)

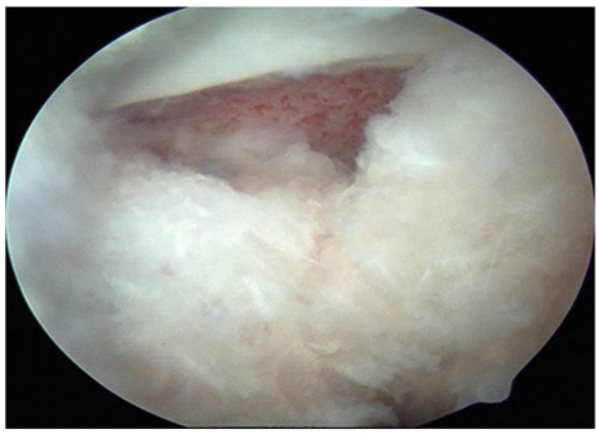


Fig. 12 Distal "T" capsulotomy appearance once closed with three sutures

USA) that can accomplish the latter pierces one capsular limb, passes the suture, then pierces the other capsular edge, and retrieves the suture all through the same portal (mid-anterior; Fig. 11). It is easiest to begin capsular closure distally and close the "T" first (usually 3–4 sutures; Fig. 12) and then the interportal capsulotomy with sutures from the double-loaded anchors already placed (usually 2–4 sutures; Fig. 13). Even if double-loaded anchors were not used, as with the automated suture-passing devices, it is easiest to pierce and pass suture on the acetabular side prior to the capsular side. Verification of complete capsular closure is ensured when no femoral head or acetabular labrum is visible (Fig. 14).

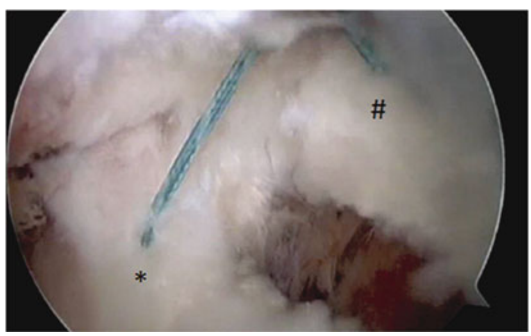


Fig. 13 Appearance of closed "T" capsulotomy and second suture from double-loaded suture anchor in acetabulum. The iliofemoral ligament proximal edge (#) and distal edge (*) are reattached to the acetabulum via double-loaded suture anchor, and a secure arthroscopic knot is tied via reversing half hitches on an alternating post configuration

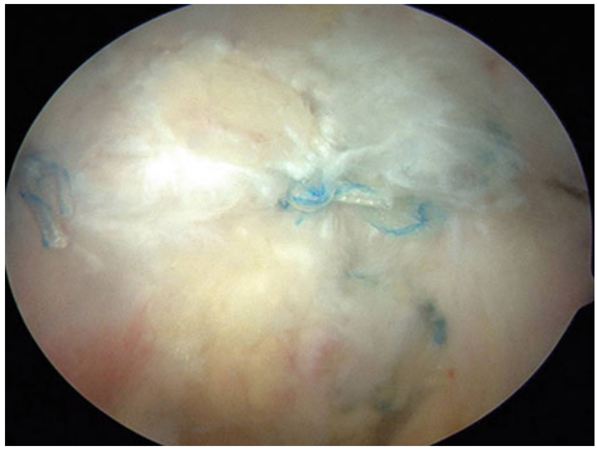


Fig. 14 Final appearance of complete capsular closure. The interportal (horizontal) and "T" (vertical) capsulotomies are securely closed, so that no portion of the joint is visible

Postoperative Rehabilitation

A postoperative hip orthosis allows motion from 0° to 90° of flexion, with crutch-assisted gait with 20 lb flat-foot weight bearing. An abduction pillow or anti-external rotation roots prevent hip external rotation at night while sleeping. External rotation and/or extension may disrupt any capsulolabral repair performed. Continuous passive motion and/or stationary bicycling may be utilized for the first postoperative month to help reduce adhesion formation. Brace and crutch use is weaned after 3–4 weeks. Avoidance of iliopsoas tendonitis is prudent with slow muscle strengthening progression. Treadmill running may commence at 3–4 months and sport-specific training at 3–6 months.

Summary

Hip microinstability is a challenging clinical entity. The clinician must be aware of its existence in order to make its diagnosis. Recognition of microinstability as a source of symptoms requires thorough history, physical examination, and imaging review. Microinstability may be the cause or the effect of other concomitant intra- or extra-articular diagnoses. All underlying problems must be addressed. Initial management should be nonoperative. Short-term surgical outcomes are successful in those that have failed nonsurgical treatment.

References

1. Colvin AC, Harrast J, Harner C. Trends in hip arthroscopy. J Bone Joint Surg Am. 2012;94(4):e23.
2. Harris J, Slikker W, Gupta A, Abrams G, Nho S. Routine complete capsular closure during Hip arthroscopy. Arthrosc Tech. 2013;2(2):e89–94.
3. Myers CA, Register BC, Lertwanich P, Ejnisman L, Pennington WW, Giphart JE, et al. Role of the acetabular labrum and the iliofemoral ligament in hip stability: an in vitro biplane fluoroscopy study. Am J Sports Med. 2011;39(Suppl):85S–91.
4. Martin HD, Savage A, Braly BA, Palmer IJ, Beall DP, Kelly B. The function of the hip capsular ligaments: a quantitative report. Arthroscopy. 2008; 24(2):188–95.
5. Hewitt JD, Glisson RR, Guilak F, Vail TP. The mechanical properties of the human hip capsule ligaments. J Arthroplasty. 2002;17(1):82–9.
6. Bardakos NV, Villar RN. The ligamentum teres of the adult hip. J Bone Joint Surg Br. 2009;91(1):8–15.
7. Kawaguchi AT, Otsuka NY, Delgado ED, Genant HK, Lang P. Magnetic resonance arthrography in children with developmental hip dysplasia. Clin Orthop Relat Res. 2000;374:235–46.
8. Wenger D, Miyanji F, Mahar A, Oka R. The mechanical properties of the ligamentum teres: a pilot study to assess its potential for improving stability in children's hip surgery. J Pediatr Orthop. 2007;27(4):408–10.
9. Cerezal L, Arnaiz J, Canga A, Piedra T, Altonaga JR, Munafo R, et al. Emerging topics on the hip: ligamentum teres and hip microinstability. Eur J Radiol. 2012;81(12):3745–54.
10. Ranawat AS, McClincy M, Sekiya JK. Anterior dislocation of the hip after arthroscopy in a patient with capsular laxity of the hip. A case report. J Bone Joint Surg Am. 2009;91(1):192–7.
11. Fabricant PD, Bedi A, De La Torre K, Kelly BT. Clinical outcomes after arthroscopic psoas

lengthening: the effect of femoral version. Arthroscopy. 2012;28(7):965–71.
12. Leunig M. CORR insights: femoroacetabular impingement predisposes to traumatic posterior hip dislocation. Clin Orthop Relat Res. 2013;471(6):1944–5.
13. Steppacher SD, Albers CE, Siebenrock KA, Tannast M, Ganz R. Femoroacetabular impingement predisposes to traumatic posterior hip dislocation. Clin Orthop Relat Res. 2013;471(6):1937–43.
14. Manner HM, Mast NH, Ganz R, Leunig M. Potential contribution of femoroacetabular impingement to recurrent traumatic hip dislocation. J Pediatr Orthop B. 2012;21(6):574–8.
15. Philippon MJ, Kuppersmith DA, Wolff AB, Briggs KK. Arthroscopic findings following traumatic hip dislocation in 14 professional athletes. Arthroscopy. 2009;25(2):169–74.
16. Krych AJ, Thompson M, Larson CM, Byrd JW, Kelly BT. Is posterior hip instability associated with cam and pincer deformity? Clin Orthop Relat Res. 2012;470(12):3390–7.
17. Kolo FC, Charbonnier C, Pfirrmann CW, et al. Extreme hip motion in professional ballet dancers: dynamic and morphological evaluation based on magnetic resonance imaging. Skeletal Radiol. May 2013;42(5):689–698.
18. Duthon VB, Charbonnier C, Kolo FC, Magnenat-Thalmann N, Becker CD, Bouvet C, et al. Correlation of clinical and magnetic resonance imaging findings in hips of elite female ballet dancers. Arthroscopy. 2013.
19. Charbonnier C, Kolo FC, Duthon VB, Magnenat-Thalmann N, Becker CD, Hoffmeyer P, et al. Assessment of congruence and impingement of the hip joint in professional ballet dancers: a motion capture study. Am J Sports Med. 2011;39(3):557–66.
20. Benali Y, Katthagen BD. Hip subluxation as a complication of arthroscopic debridement. Arthroscopy. 2009;25(4):405–7.
21. Ilizaliturri Jr VM. Complications of arthroscopic femoroacetabular impingement treatment: a review. Clin Orthop Relat Res. 2009;467(3):760–8.
22. Matsuda DK. Acute iatrogenic dislocation following hip impingement arthroscopic surgery. Arthroscopy. 2009;25(4):400–4.
23. Mei-Dan O, McConkey MO, Brick M. Catastrophic failure of hip arthroscopy due to iatrogenic instability: can partial division of the ligamentum teres and iliofemoral ligament cause subluxation? Arthroscopy. 2012;28(3):440–5.
24. Sansone M, Ahlden M, Jonasson P, Sward L, Eriksson T, Karlsson J. Total dislocation of the hip joint after arthroscopy and ileopsoas tenotomy. Knee Surg Sports Traumatol Arthrosc. 2013;21(2):420–3.
25. Souza BG, Dani WS, Honda EK, Ricioli Jr W, Guimaraes RP, Ono NK, et al. Do complications in hip arthroscopy change with experience? Arthroscopy. 2010;26(8):1053–7.
26. Harris JD, McCormick FM, Abrams GD, Gupta AK, Ellis TJ, Bach Jr BR, et al. Complications and reoperations during and after hip arthroscopy: a systematic review of 92 studies and more than 6,000 patients. Arthroscopy. 2013;29(3):589–95.
27. Gray AJ, Villar RN. The ligamentum teres of the hip: an arthroscopic classification of its pathology. Arthroscopy. 1997;13(5):575–8.
28. Botser IB, Martin DE, Stout CE, Domb BG. Tears of the ligamentum teres: prevalence in hip arthroscopy using 2 classification systems. Am J Sports Med. 2011;39(Suppl):117S–25S.
29. Domb BG, Martin DE, Botser IB. Risk factors for ligamentum teres tears. Arthroscopy. 2013;29(1):64–73.
30. Phillips AR, Bartlett G, Norton M, Fern D. Hip stability after ligamentum teres resection during surgical dislocation for cam impingement. Hip Int. 2012;22(3):329–34.
31. Bellabarba C, Sheinkop MB, Kuo KN. Idiopathic hip instability. An unrecognized cause of coxa saltans in the adult. Clin Orthop Relat Res. 1998;355:261–71.
32. Beighton P, Horan F. Orthopaedic aspects of the Ehlers-Danlos syndrome. J Bone Joint Surg Br. 1969;51(3):444–53.
33. Grahame R, Bird HA, Child A. The revised (Brighton 1998) criteria for the diagnosis of benign joint hypermobility syndrome (BJHS). J Rheumatol. 2000;27(7):1777–9.
34. Blakey CM, Field MH, Singh PJ, Tayar R, Field RE. Secondary capsular laxity of the hip. Hip Int. 2010;20(4):497–504.
35. Philippon MJ. The role of arthroscopic thermal capsulorrhaphy in the hip. Clin Sports Med. 2001;20(4):817–29.
36. Slikker 3rd W, Van Thiel GS, Chahal J, Nho SJ. The use of double-loaded suture anchors for labral repair and capsular repair during hip arthroscopy. Arthrosc Tech. 2012;1(2):e213–7.
37. McCormick F, Slikker W, 3rd, Harris JD, et al. Evidence of capsular defect following hip arthroscopy. Knee Surg Sports Traumatol Arthrosc. Apr 2014;22(4):902–905.

Stress Fractures of the Hip and Pelvis

81

Ronald Tsao and Kathleen Weber

Contents

R. Tsao (✉)
Rush University Medical Center, Chicago, IL, USA
e-mail: ronald_tsao@rush.edu

K. Weber
Midwest Orthopaedics at Rush, Rush University Medical Center, Chicago, IL, USA
e-mail: kweber@rushortho.com

Abstract

Stress fractures of the hip and pelvis are relatively rare events. They have been well described in female athletes and military recruits. There are two types of stress fractures: fatigue and insufficiency fractures. Fatigue fractures occur as a result of repetitive forces on normal bone, whereas insufficiency fractures occur in structurally abnormal bone. There are many risk factors that have been implicated in contributing to the development of a stress fracture which include gender differences, biomechanical, anatomical, baseline fitness, and nutritional factors. Magnetic resonance imaging is the most sensitive and specific imaging modality available for imaging stress fractures. Most hip and pelvic stress fractures can be managed with a conservative approach of adequate pain control, relative rest through partial or non-weight-bearing status, and a progressive plan to return to normal activity.

Introduction

Stress fractures are common injuries and have been reported to account for up to 20 % of all injuries seen in a sports medicine clinic [1]. Stress fractures are common in the metatarsals, tibia, and fibula. However, hip and pelvic stress fractures are fairly rare and often present with nonspecific signs and symptoms such as groin or lower back pain.

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_91

Pain is usually insidious in onset and an inciting traumatic event is usually absent. These factors make identifying hip and pelvic stress fractures a diagnostic challenge. A delay in diagnosis or failure to identify a stress fracture can lead to significant morbidity such as prolonged time to normal physical activity or return to training, chronic pain, delayed union or nonunion, avascular necrosis, or even catastrophic fracture displacement. Therefore, it is imperative for the clinician to be aware of the possibility of these conditions in the differential and to keep a high level of suspicion. The goal of this chapter is to bring awareness by providing a better understanding of the presentation, diagnostic modalities, and strategies in treatment and rehabilitation for hip and pelvic stress fractures.

Etiology

Under normal circumstances there is equilibrium between osteoclastic and osteoblastic activity. Repeated stress on bone can push the balance toward osteoclastic activity and result in overall bone resorption which can predispose it to microfractures, with maximal osteoclastic activity occurring at about 3 weeks [2]. Stress fractures occur when normal repetitive forces, forces in and of themselves not capable of causing a fracture, result in microfractures. Over time, these microfractures accumulate due to the inability of osteoblastic activity to keep up with the bone remodeling needed for bone repair. This causes a disruption in the integrity of the bone which can lead to the development of a fracture in the cortical bone.

Stress fractures are generally categorized into two groups: fatigue and insufficiency fractures. Fatigue fractures occur when repetitive forces cause a fracture in structurally normal bone. On the other hand, insufficiency fractures occur when these normal forces cause a fracture in bone that is structurally deficient. Whereas fatigue fractures often occur in highly active younger adults, insufficiency fractures are often seen in older adults with a predisposition for fractures such as those with osteopenia or osteoporosis, rheumatologic diseases, chronic corticosteroid use, endocrinopathies, or history of external beam radiation.

Risk Factors

Gender Differences and the Female Athlete Triad

Females are at higher risk for developing a stress fracture. This has been demonstrated in many research studies involving athletes and military recruits [3]. Special mention should be made to the female athlete triad: eating disorder, amenorrhea, and osteoporosis. The combination of decreased caloric intake coupled with increased caloric expenditure from exercise leads to a negative energy balance or low energy state. This can result in amenorrhea, estrogen deficiency, and deficiency of other hormones that play a role in overall bone health [4]. It is well known that amenorrhea and menstrual irregularities have been found to be a risk factor for stress fractures [5, 6]. In a survey of 1,630 females in the US Army, those with a history of amenorrhea lasting more than 6 months were more likely to suffer from one or more stress fractures during their training [7]. This may be in part due to a hypoestrogenic state. Estrogen has the effect of protecting bone from resorption. Interestingly, estrogen receptors are found on osteoblasts and can stimulate their activity [8]. Also, a low energy state can hinder repair of microfractures. This coupled with increased bone resorption from repetitive stresses can also increase fracture risk. Other than anatomical and biomechanical differences, the female athlete triad is likely to contribute to the higher incidence of stress fractures in females over males.

Muscle Fatigue

It has been speculated that muscle fatigue might play a role in subjecting bone to higher forces [9, 10]. During exercise, muscles can act to absorb, counteract, and redirect forces experienced by bone. For example, when a bone is subjected to a bending force, one side experiences a compressive force and the opposite side a tensile force. Eccentric contraction of a muscle on the tensile side of bone can lessen the tensile force experienced by the bone. With muscle fatigue, this protective mechanism is dampened, and as a result the bone is subjected to higher repetitive forces leading to bone fatigue. Another example of this is in activities that involve running. Muscle fatigue can lead to a change in gait which in turn can cause an increase in shear force experience in a section of bone that is not accustomed to this higher level of force subjecting it to increased microdamage [9].

Anatomy

Anatomical differences can also account for increased risk of sustaining stress fractures. Several studies have shown leg length discrepancies to be a predisposing factor [10, 11]. A prospective military study showed that pes planus (flatfoot) predisposed recruits to metatarsal stress fractures and those with pes cavus (high arch) were more prone to tibial and femoral stress fractures [12]. Interestingly, Giladi et al. showed that out of 289 military recruits who were followed over 14 weeks of basic training, those with a narrower tibial width sustained more tibial, femoral, and total stress fractures [13]. Further studies have demonstrated that more specifically it is decreased bone cross-sectional area that increases the risk for developing stress fractures [14, 15].

Baseline Fitness and Other Extrinsic Factors

A sudden increase in the amount of stress to bone can also increase one's risk in developing a stress fracture. Changing footwear, running surfaces, and sudden change or increase in intensity of training have been associated with increased risk of stress fractures [16–18]. Shaffer et al. [6] suggested that baseline fitness was the strongest predictor for stress fractures. In this study, 3,249 female Marine Corps recruits were followed through basic training and those in the slowest quartile of timed runs at the beginning of their training were three to four times more likely to suffer from a pelvic or femoral stress fracture when compared to women in the fastest quartile. This data suggests that preconditioning or a graduated exercise program prior to participating in repetitive strenuous activity might help to prevent stress fractures.

Calcium and Vitamin D

The need for calcium and vitamin D and their impact on overall bone health is undisputed. However, the optimal amount of calcium and vitamin D remain a topic of debate. At the time this chapter was written, recommendations per the Institute of Medicine on daily amounts of calcium and vitamin D in otherwise healthy adults aged 18–70 are 1,000 mg of calcium and 600 IU of vitamin D, for adolescents 1,300 mg of calcium and 600 IU of vitamin D, and for adults aged 71 and older 1,200 mg of calcium and 800 IU of vitamin D daily [19]. Studies on calcium and vitamin D in preventing stress fractures have been mixed. In a prospective study of track and field athletes, Bennell et al. [5] did not show any significant difference in calcium intake between individuals that incurred a stress fracture and those that did not. More recent studies suggest calcium supplementation to be preventative [20, 21]. Studies on the role of vitamin D supplementation in prevention of stress fractures have also been conflicting [22, 23]. Using data from the Growing Up Today Study, which is an ongoing prospective cohort study of adolescents girls in the United States, Sonneville et al. [24] showed that vitamin D from dietary sources and supplements were protective against stress fractures. This was especially true for those in the highest quintile of vitamin D intake who showed a 50 % lower risk of stress fractures compared to those in the lowest quintile (HR = 0.49, 95 % CI = 0.24–1.01; ptrend = 0.07). About 90 % of stress fractures occurred in girls who participated in ≥1 h per day of high-impact activity. Among this highly active group, girls in the highest quintile for vitamin D intake had a 52 % lower risk of stress fracture compared to the lowest quintile suggesting that vitamin D intake was protective for stress fractures in highly active girls. In this same study, calcium intake from dietary sources and supplements did not show any protective effects on preventing stress fractures. Limitations to this study included its lack of ethnic diversity, those in the lower socioeconomic status were likely underrepresented as participants were offspring from mothers that were nurses, and participants had a high level of activity which likely does not represent the true population of adolescents across the United States.

Imaging Modalities

Radiography

Due to their low cost and availability, standard plain film radiographs are usually the first imaging study obtained. Plain radiographs have been reported to have a sensitivity around 10 % in identifying stress fractures [1] and reach nearly 0 % in detecting sacral stress fractures [25]. Due to their low sensitivity, they have limited utility in detecting stress fractures especially if obtained in the first week of onset of pain. Radiographic findings suggesting the presence of a stress fracture include periosteal elevation, endosteal elevation, cortical sclerosis, or callous formation. A sclerotic fracture line might take 3 weeks to 3 months from the onset of pain to show up on a radiograph [26]. Although not usually useful for initial diagnosis, it can be used to monitor healing through serial x-rays to look for callous formation and bone healing.

Computed Tomography

Some consider CT scans to be the gold standard in diagnosing stress fractures. Specifically multidetector CT has shown to be useful due to its ability to reconstruct 3D images, high resolution, and ability to detect subtle fracture lines. However, due to its high ionizing radiation, CT has a limited utility in the diagnosis of stress fractures especially in young individuals. It has shown to be very specific but less sensitive than bone scintigraphy and MRI [27]. Findings on CT include periosteal reaction, endosteal reaction, and presence of a fracture line. It can be a useful adjunctive diagnostic tool in cases when MR or bone scintigraphy is inconclusive.

Bone Scintigraphy

Radionuclide bone scanning can show abnormalities seen as areas of increased uptake as early as 6–72 h after an injury [10]. Its sensitivity has been reported to reach nearly 100 % [28]. Infection, tumors, trauma, avascular necrosis, and metabolic bone disease can also cause increased uptake of the radioisotope on a bone scan, and as such it has a low specificity. The need to subject a patient to the radiation load is also an area of concern.

Magnetic Resonance Imaging

MR imaging is both highly sensitive and specific in detecting stress fractures and is achieved without subjecting the patient to ionizing radiation. It has been reported to be 87–100 % sensitive and nearly 100 % specific in detecting stress fractures [10, 27–29]. It is also superior to CT at imaging injuries to soft tissue structures such as ligaments, muscles, and tendons that may be the source of a patient's complaints. It also requires less time to perform than scintigraphy. For these reasons, it should be considered the gold standard in detecting stress fractures. Bone marrow edema is usually the first abnormality detectable in MR imaging. These areas show up on T1-weighted images as areas of low signal intensity and on T2-weighted images as areas of high signal intensity. T2-weighted short tau inversion recovery (STIR) images are even more sensitive than T1- or T2-weighted images [17]. Its disadvantages include its higher cost, lack of availability compared to other imaging modalities, and contraindication of its use with certain metals.

An MRI grading system to describe the severity of stress injuries has been proposed by Fredericson and colleagues [17]. A normal MRI is a grade 0. Grade 1 shows mild to moderate periosteal edema without marrow changes on STIR or T2-weighted images. Grade 2 has moderate to severe periosteal edema with marrow changes on STIR or T2-weighted images. Grade 3 demonstrates moderate to severe edema of both the periosteum and marrow on T1-weighted and STIR images. And finally a low-intensity fracture line is seen in Grade 4 injuries. It would be intuitive to assume that a higher grade injury would correlate to a longer recovery time. However, several authors have not shown this assumption to be true [30, 31].

Stress Fractures of the Hip

Femoral Neck

Femoral neck fractures account for 5 % of all stress fractures [32] and seem to be more common in females with a high level of activity such as long distance runners and military recruits [33, 34]. Patients usually present with an insidious onset of groin pain but can also present with pain in the anterior thigh or hip. Pain initially presents near the end of an activity and is usually relieved by rest. Pain can be worse during loading of the affected leg. If left untreated, the stress injury can progress to a complete fracture requiring surgical pinning. Further complications of an untreated femoral neck fracture can lead to delayed union, nonunion, varus deformity, displacement, and avascular necrosis.

There have been three proposed classification systems to describe femoral neck fractures. Devas categorized femoral neck fractures into two groups: compression and tension fractures (Fig. 1) [35]. Compression femoral neck fractures involve the inferior side of the femoral neck where compression forces predominate. Tension femoral neck fractures occur at the superior part of the femoral neck where tensile forces predominate. Blickenstaff and Morris classified femoral neck fractures into three types: type I fractures have callus formation without a visible fracture line, type II fractures have a fracture line through the neck of the femur without displacement, and type III is a displaced fracture [36]. Fullerton and Snowdy also categorized femoral neck fractures into three groups: nondisplaced tension-sided fractures, nondisplaced compression-sided fractures, and displaced fractures [37].

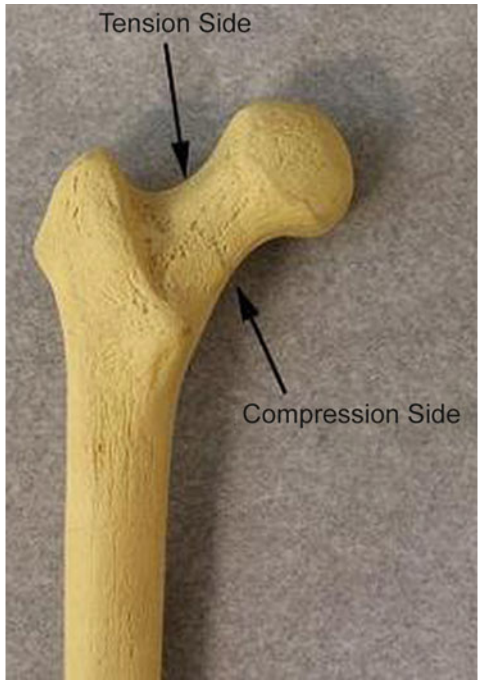


Fig. 1 Model of femur demonstrating where a tension or compression fracture of the femoral neck would occur

Compression-sided femoral neck fractures are considered stable and can potentially be treated conservatively with partial or non-weight-bearing status (Fig. 2). Tension-sided fractures are considered potentially unstable and are at higher risk for displacement. Their treatment remains controversial. Some argue tension-sided femoral neck fractures may need prophylactic internal fixation to avoid potential displacement. Others have demonstrated successful treatment with strict bed rest [35, 36]. This option may be too burdensome for some and can result in deconditioning, decreased bone density from prolonged bed rest, decubitus ulcers, pneumonia, or venous thromboembolisms. One case series demonstrated successful

Fig. 2 Compression side femoral neck stress reaction (arrow) is noted on coronal proton density fat-saturation MRI image

nonoperative management using a non-weight bearing strategy in three patients with of a tensile-sided femoral neck stress fracture [38]. The authors do note that the tensile-sided femoral stress fractures were more superiorly placed, away from the midportion of the femoral neck which is the point of maximum stress. As such, each case should be carefully considered and a treatment plan developed accordingly.

A detailed history including activity level and any recent changes in training or exercise regimen should be elicited. In females it is helpful to obtain a menstrual history. On physical exam, a patient may have an antalgic gait. Swelling and erythema are usually absent. Clinical assessment should also include leg length discrepancies, leg alignment, or presence of pes planus or cavus. Due to the deep structure, it may be difficult to elicit bony tenderness with palpation. There may be pain with flexion and internal rotation of the hip. Special tests may also be positive such as hopping on the affected side or fulcrum test. Obtaining plain radiographs prior to more dynamic tests may avoid potential worsening or displacement of the fracture at the time of testing.

Once the diagnosis of a femoral neck stress fracture is determined, serial radiographs can be used to monitor for displacement and progression of healing. If displacement is seen, the patient should be taken for emergent surgical stabilization. Avascular necrosis has been reported to occur in 25–30 % of patients with displaced femoral neck fractures treated with internal fixation [39]. Overall prognosis of nondisplaced femoral neck fractures seems to be very good. In a Finnish study of military recruits, 66 patients with 70 femoral neck fractures were followed for a mean of 18.3 years. None of these military recruits had any signs of osteoarthritis or avascular necrosis at follow-up [33].

Stress Fractures of the Pelvis

Pelvic stress fractures have been well described in long distance runners and military recruits. However, it is not uncommon to see older patients with pelvic insufficiency fractures. It has been estimated that pelvic stress fractures account for 1–7 % of all stress fractures [40]. The most common pelvic bone to be fractured is the inferior pubic ramus [41]. Having a concomitant pelvic fracture such as an acetabular or pubic ramus fracture has been seen in up to 25–80 % of pelvic stress fracture cases [42].

Sacrum

Stress fractures of the sacrum should be considered in individuals with insidious onset of asymmetric low back pain or buttock pain. Patients most commonly present with lumbar back or gluteal pain, although hip, groin, and radicular type pain have also been reported. Symptoms are usually worsened with weight-bearing activities and relieved with rest. Sacral stress fractures are often misdiagnosed as the history and physical exam can be suggestive of more common etiologies such as spondylolysis, spondylolisthesis, sacroiliac joint dysfunction, piriformis syndrome, lumbago, vertebral compression fractures, or spinal stenosis.

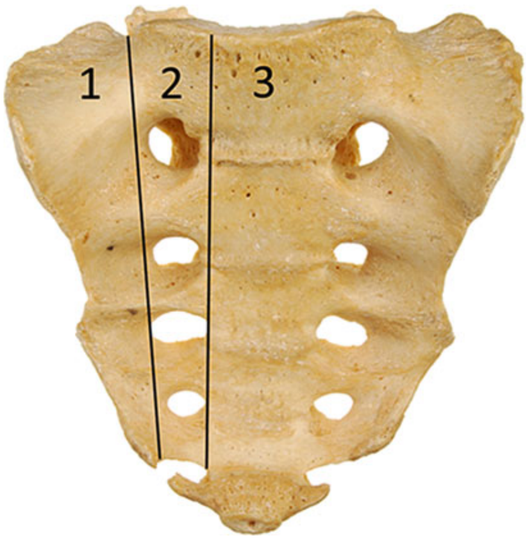


Fig. 3 Sacral fracture zones defined by Denis et al. [43]

Denis et al. classified sacral fractures into three zones (Fig. 3) [43]. Zone 1 involves the sacral ala or wing and is the most common part of the sacrum to sustain a stress fracture. Zone 2 extends from the sacral foramina to the sacral body. Fractures in zone 2 can result in unilateral lumbosacral radiculopathies. Fractures in zone 3 involve the sacral body or canal of the sacrum. Fractures in zone 3 can cause bilateral neurological symptoms, saddle anesthesia, and sphincter tone dysfunction.

Physical exam may reveal sacral tenderness with palpation which is a nonspecific finding. The patient may have pain with the flexion-abduction-external rotation (FABER) test or Gaenslen's test. The FABER test is performed by flexing the hip of the affected side to 90°. The examiner then places the hip in external rotation while pushing the knee on the affected side toward the exam table. The Gaenslen's test is done with the patient lying supine with the knee on the affected side fully flexed with the contralateral leg dangling off the exam table. The examiner then simultaneously applies flexion to the affected hip while hyperextending the contralateral side. The test is positive if pain is elicited. Having the patient hop on the leg of the affected side might also elicit pain. About 70 % of patients may complain of neurologic symptoms that suggest a radiculopathy or myelopathy; objective neurological findings may only be found in 2–14 % of cases [42]. In the rare case where the fracture involves the sacral body, neurologic symptoms such as sphincter tone dysfunction or limb paresthesias can be found. Careful attention should also be put into examining the pubic rami. As discussed above, there is an association of concomitant pelvic stress fractures. One study found that 78 % of patients with a sacral stress fracture also had a coexisting pubic ramus fracture [44]. Disruption of the pelvic ring can cause increased stress in other areas and may be the reason why concomitant pelvic stress fractures occur.

Fractures are usually found in the sacral ala (Fig. 4) and run parallel to the sacroiliac joint [42]. Radiographs are of limited utility as stress fractures in this area may be obscured by the presence of stool, bowel gas pattern, and calcified vessels. Bilateral sacral fractures may be seen and in some cases can be connected by a horizontal fracture through the sacral body creating the so-called H sign which can be seen on bone scintigraphy. The presence of the "H sign" has been reported to be highly specific for an insufficiency fracture [45].

Pubic Ramus

Pubic ramus fractures account for 1.25 % of all stress fractures [46]. Most pubic ramus stress fractures occur at the medial portion of the pubic ramus or at the junction between the inferior pubic ramus and the ischial ramus (Fig. 5) [47]. The adductor magnus muscle originates at this junction, and its repetitive contraction has been attributed to contribute to the development of a pubic ramus stress fracture [46]. Patients often complain of insidious onset of groin pain but can also present with perineal, anterior thigh, or buttock pain. On physical exam there may be direct tenderness to palpation over the pubic ramus. There may also be an antalgic gait, pain with abduction, or pain with resisted adduction and external rotation of the hip.

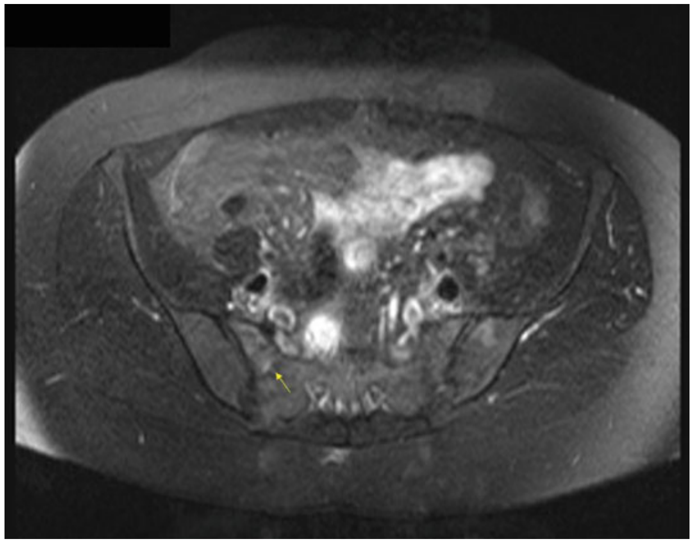


Fig. 4 Right sacral ala insufficiency fracture. T2 axial fat-saturation MRI reveals abnormal bone marrow edema (*arrow*)

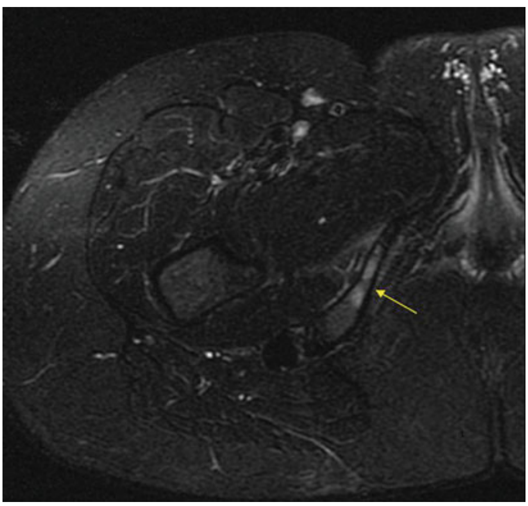


Fig. 5 MRI T2-weighted sequence of a right inferior pubic ramus stress fracture

General Diagnostic and Treatment Guidelines

In most cases, radiography should be the first diagnostic imaging obtained. If the radiographs do not demonstrate a stress fracture and the diagnosis is still suspected, an MRI can be obtained as it is highly sensitive in detecting stress fractures, does not subject the patient to ionizing radiation, and can give valuable information on surrounding soft tissue structures. Alternatively, another acceptable option is to obtain serial radiographs as findings are often not seen initially and may become apparent a few weeks after the onset of pain. Serial radiographs may also be useful in monitoring healing once the diagnosis has been established. Although not typically needed, repeating an MRI can be done to assure that the stress fracture is healing or has healed. A clinical example where repeating an MRI may be useful is in a patient with persistent pain despite treatment. The presence of stool, bowel gas, and calcifications of vessels on radiographs can make it difficult to see pelvic stress fractures. In this case, a repeat MRI can be done to monitor for healing of a sacral stress fracture.

The first step to treating a stress fracture should involve pain control. Medications, activity modification, and immobilization can help control pain. A variety of medications can be considered based on the patient's reported pain level. These include acetaminophen, nonsteroidal anti-inflammatory drugs (NSAIDs), and narcotics which are typically not needed. Some studies suggest that the use of NSAIDs should be avoided in management of fractures due to risk of delayed union or nonunion [48, 49]. However, a meta-analysis in 2010 did not find any significant risk of nonunion with NSAID use [50]. When considering the use of NSAIDs for pain control in stress fracture management, the treating physician should carefully consider the current scientific literature regarding its use in fracture healing.

Treatment plans should be tailored to each patient. In general relative rest and partial or full non-weight-bearing status should be implemented if significant amount of pain is present. The patient should avoid any aggravating activities and gradually increase activity as directed by the treating clinician when pain-free. In some cases low impact exercise such as stationary cycling or swimming may be appropriate. Physical therapy should be initiated when the patient is without significant pain. Once the patient is pain-free with normal daily activities, a graduated progressive plan back to sports can be initiated. If bed rest is chosen, deep vein thrombosis prevention and early mobilization, when appropriate, should be the goal. In general, total rehabilitation time can take 4–8 weeks.

Summary

Stress fractures are a common occurrence especially in female athletes. However, stress fractures of the hip and pelvis are relatively rare events and can be difficult to diagnose. The cause of these injuries is multifactorial and includes intrinsic and extrinsic factors such as the female athlete triad and biomechanical, anatomical, training, and nutritional factors. The clinician should maintain a high level of suspicion especially in young female athletes that display even part of the female athlete triad to prevent delay in diagnosis. Most hip and pelvic stress fractures can successfully be treated conservatively. Early rehabilitation should be implemented to return patients to their prior level of activity as soon as medically possible. It is important to have a good knowledge base about the causes of stress fractures as this can aid in its prevention and other overuse injuries.

References

1. Fredericson M, Jennings F, Beaulieu C, Matheson GO. Stress fractures in athletes. Top Magn Reson Imaging. 2006;17:309–25.
2. Frost HM. Some ABC's of skeletal pathophysiology. 5. Microdamage physiology. Calcif Tissue Int. 1991;49:229–31.
3. Wentz L, Liu PT, Haymes E, Illich JZ. Females have a greater incidence of stress fractures than males in both military and athletic populations: a systematic review. Mil Med. 2011;176:420–30.
4. Nattiv A, Loucks AB, Manore MM, Sanborn CF, Sundgot-Borgen J, Warren MP. American college of sports medicine position stand: the female athletic triad. Med Sci Sports Exerc. 2007;39:1867–82.
5. Bennell KL, Malcolm SA, Thomas SA, Reid SJ, Brukner PD, Ebeling PR, Wark JD. Risk factors for stress fractures in track and field athletes. A twelve-month prospective study. Am J Sports Med. 1996;24:810–8.
6. Shaffer RA, Rauh MJ, Brodine SK, Trone DW, Macera CA. Predictors of stress fracture susceptibility in young female recruits. Am J Sports Med. 2006;34(1):108–15.
7. Friedl KE, Nuovo JA, Patience TH, Dettori JR. Factors associated with stress fracture in young Army women: indications for further research. Mil Med. 1992;157: 334–8.
8. Eriksen EF, Colvard DS, Berg NJ, Graham ML, Mann KG, Spelsberg TC, Riggs BL. Evidence of estrogen receptors in normal human osteoblast-like cells. Science. 1988;241:84–6.
9. Friberg O. Leg length asymmetry in stress fractures: clinical and radiological study. J Sports Med Phys Fitness. 1982;22(4):485–8.
10. Yoshikawa T, Mori S, Santiesteban AJ, Sun TC, Hafstad E, Chen J, Burr DB. The effects of muscle fatigue on bone strain. J Exp Biol. 1994;188:217–33.
11. Korpelainen R, Orava S, Karpakka J, Siira P, Hulkko A. Risk factors for recurrent stress fractures in athletes. Am J Sports Med. 2001;29(3):304–10.
12. Simkin A, Leichter I, Giladi M, Stein M, Milgrom C. Combined effect of foot arch structure and an orthotic device on stress fractures. Foot Ankle. 1989;10(1):25–9.
13. Giladi M, Milgrom C, Simkin A, Danon Y. Stress fractures identifiable risk factors. Am J Sports Med. 1991;19(6):647–52.
14. Beck TJ, Ruff CB, Mourtada FA, Shaffer RA, Maxwell-Williams K, Kao GL, Sartoris DJ, Brodine S. Dual energy x-ray absorptiometry derived structural geometry for stress fracture prediction in male U.S. Marine Corps recruits. J Bone Miner Res. 1996;11(5):645–53.
15. Beck TJ, Ruff CB, Shaffer RA, Betsinger K, Trone DW, Brodine SK. Stress fracture in military recruits: gender differences in muscle and bone susceptibility factors. Bone. 2000;27(3):437–44.
16. Campbell SE, Fajardo RS. Imaging of stress injuries of the pelvis. Semin Musculoskelet Radiol. 2008;12(1): 62–71.

17. Fredericson M, Bergman AG, Hoffman KL, Dillingham MS. Tibial stress reaction in runners: correlation of clinical symptoms and scintigraphy with a new magnetic imaging grading system. Am J Sports Med. 1995;23:472–81.
18. Goldberg B, Pecora C. Stress fractures: a risk of increased training in freshman. Phys Sports Med. 1994;22:68–78.
19. Institute of Medicine of the National Academies [Internet]. Washington, DC: National Academies Press; 2010 [updated 2013 Sept 5; cited 2013 Sept 5]. http://www.iom.edu/Reports/2010/Dietary-Reference-Intakes-for-Calcium-and-Vitamin-D.aspx
20. Lappe J, Cullen D, Haynatzki G, Recker R, Ahlf R, Thompson K. Calcium and vitamin d supplementation decreases incidence of stress fracture in female navy recruits. J Bone Miner Res. 2008;23:741–9.
21. Nieves JW, Melsop K, Curtis M, Kelsey JL, Bachrach LK, Greendale G, Sowers MR, Sainani KL. Nutritional factors that influence change in bone density and stress fracture risk among young female cross-country runners. PM R. 2010;2(8):740–50.
22. Ruohola JP, Laaksi I, Ylikomi T, Haataja R, Mattila VM, Sahi T, Tuohimaa P, Pihlajamäk H. Association between serum 25(OH)D concentrations and bone stress fractures in Finnish young men. J Bone Miner Res. 2006;21(9):1483–8.
23. Valimaki VV, Alfthan H, Lehmuskallio E, Loyttyniemi E, Sahi T, Suominen H, Välimäki MJ. Risk factors for clinical stress fractures in male military recruits: a prospective cohort study. Bone. 2005;37:267–73.
24. Sonneville KR, Gordon CM, Kocher MS, Pierce LM, Ramappa A, Field A. Vitamin d, calcium, and dairy intakes and stress fractures among female adolescents. Arch Pediatr Adolesc Med. 2012;166(7):595–600.
25. Kiuru MJ, Pihlajamaki HK, Ahovuo JA. Fatigue stress injuries of the pelvic bones and proximal femur: evaluation with MR imaging. Eur Radiol. 2003;13(3):605–11.
26. Knapp TP, Garrett Jr WE. Stress fractures, general concepts. Clin Sports Med. 1997;16:339–56.
27. Gaeta M, Minutoli F, Scribano E, Ascenti G, Vinci S, Bruschetta D, Magaudda L, Blandino A. CT and MR imaging findings in athletes with early tibial stress injuries: comparison with bone scintigraphy findings and emphasis on cortical abnormalities. Radiology. 2005;235(2):553–61.
28. Ganiyusufoglu AK, Onat L, Karatoprak O, Enercan M, Hamzaoglu A. Diagnostic accuracy of magnetic resonance imaging versus computed tomography in stress fractures of the lumbar spine. Clin Radiol. 2010;65(11):902–7.
29. Murray SR, Reeder MT, Udermann BE, Pettitt RW. High-risk stress fractures: pathogenesis, evaluation, and treatment. Compr Ther. 2006;32(1):20–5.
30. Beck BR, Bergman AG, Miner M, Arendt EA, Klevansky AB, Matheson GO, Norling TL, Marcus R. Tibial stress injury: relationship of radiographic, nuclear medicine bone scanning, MR imaging, and CT severity grades to clinical severity and time to healing. Radiology. 2012;263(3):811–8.
31. Slocum KA, Gorman JD, Puckett ML, Jones SB. Resolution of abnormal MR signal intensity in patients with stress fractures of the femoral neck. AJR Am J Roentgenol. 1997;168(5):1295–9.
32. Fullerton Jr LR. Femoral neck stress fractures. Sports Med. 1990;9(3):192–7.
33. Pihlajamäk HK, Ruohola JP, Weckström M, Kiuru MJ, Visuri TI. Long-term outcome of undisplaced fatigue fractures of the femoral neck in young male adults. J Bone Joint Surg. 2006;88(12):1574–9.
34. Talbot JC, Cox G, Townsend M, Langham M, Parker PJ. Femoral neck stress fractures in military personnel—a case series. J R Army Med Corps. 2008;154(1):47–50.
35. Devas MB. Stress fractures of the femoral neck. J Bone Joint Surg. 1965;47(4):728–38.
36. Blickenstaff LD, Morris JM. Fatigue fracture of the femoral neck. J Bone Joint Surg. 1966;48(6):1031–47.
37. Fullerton Jr LR, Snowdy HA. Femoral neck stress fractures. Am J Sports Med. 1988;16(4):365–77.
38. Provencher MT, Baldwin AJ, Gorman JD, Gould MT, Shin AY. Atypical tensile-sided femoral neck stress fractures: the value of magnetic resonance imaging. Am J Sports Med. 2004;32(6):1528–34.
39. Min BW, Kim SJ. Avascular necrosis of the femoral head after osteosynthesis of femoral neck fracture. Orthopedics. 2011;34(5):349. doi: 10.3928/01477447-20110317-13.
40. Snyder RA, Koester MC, Dunn WR. Epidemiology of stress fractures. Clin Sports Med. 2006;25(1):37–52.
41. Kiuru MJ, Pihlajamaki HK, Perkio JP, Ahovuo JA. Dynamic contrast-enhanced MR imaging in symptomatic bone stress of the pelvis and the lower extremity. Acta Radiol. 2001;42:277–85.
42. Schindler OS, Watura R, Cobby M. Sacral insufficiency fractures. J Orthop Surg. 2007;15(3):339–46.
43. Denis F, Davis S, Comfort T. Sacral fractures: an important problem. Retrospective analysis of 236 cases. Clin Orthop Relat Res. 1988;277:67–81.
44. Aretxabala I, Fraiz E, Pérez-Ruiz F, Ríos G, Calabozo M, Alonso-Ruiz A. Sacral insufficiency fractures. High association with pubic rami fractures. Clin Rheumatol. 2000;19(5):399–401.
45. Schneider R, Yacovone J, Ghelman B. Unsuspected sacral fractures: detection by radionuclide bone scanning. AJR Am J Roentgenol. 1985;144:337–41.
46. Kim SM, Park CH, Gartland JJ. Stress fracture of the pubic ramus in a swimmer. Clin Nucl Med. 1987;12:118–9.

47. Pavlov H, Nelson TL, Warren RF, Torg JS, Burstein AH. Stress fractures of the pubic ramus. A report of twelve cases. J Bone Joint Surg. 1982;64:1020–5.
48. Dimmen S, Nordsletten L, Engebretsen L, Steen H, Madsen JE. Negative effect of parecoxib on bone mineral during fracture healing in rats. Acta Orthop. 2008;79(3):438–44.
49. Ziltener JL, Leal S, Fournier PE. Non-steroidal anti-inflammatory drugs for athletes: an update. Ann Phys Rehabil Med. 2010;53:278–82.
50. Dodwell ER, Latorre JG, Parisini E, Zwettler E, Chandra D, Mulpuri K, Snyder B. NSAID exposure and risk of nonunion: a meta-analysis of case-control and cohort studies. Calcif Tissue Int. 2010;87(3):193–202.

Surgical Technique: Arthroscopic Removal of Loose or Foreign Body

82

Geoffrey S. Van Thiel

Contents

G.S. Van Thiel
Orthopedics and Orthopedic Surgery, Rockford Orthopedic Associates, Rockford, IL, USA

Rush University Medical Center, Chicago, IL, USA
e-mail: gvanthiel@rockfordortho.com

Abstract

Intra-articular loose bodies in the hip can pose a significant treatment dilemma to the treating surgeon. Open approaches carry a significant morbidity and risk of complications. Therefore, arthroscopy provides an excellent alternative. Loose bodies can be the result of a multitude of different pathologies, and each pathology carries its own inherent challenges. Synovial chondromatosis creates many free bodies, whereas a hip dislocation can result in a single free large osteochondral fragment. Bullets may remain lodged beneath the cartilage, and loose implants can cause significant joint destruction. Regardless of the cause, arthroscopic removal of loose bodies from the hip joint is as much about planning as it is about technique. The correct instruments can make the case easy, and lack of the appropriate equipment can render the arthroscopic surgery impossible. The following chapter highlights some of the causes of loose bodies and explores some technical tips.

Introduction

Intra-articular loose bodies can result from a multitude of pathologies. Regardless of the cause, each case can pose a significant treatment challenge for the treating surgeon. There is often a balance between surgical morbidity and postoperative benefit. Prior to the introduction of

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_119

arthroscopic techniques, open surgery was the only option. Although open techniques are an excellent option, significant complications are possible. These include everything from postoperative stiffness to nonunion and infection.

Perhaps a case example best illustrates the value of arthroscopic techniques. A 21-year-old college student on a ski trip sustains a posterior hip dislocation that spontaneously reduces after a serious fall. Post-injury imaging reveals a small loose piece of the posterior acetabular wall in the intra-articular joint. His options for removal include an open removal with a trochanteric osteotomy and hip dislocation or an arthroscopic approach with hip arthroscopy. The arthroscopic approach results in a quicker recovery and the potential for a significant decrease in postoperative morbidity.

However, each case is different and requires different techniques to address the variety of problems. Lee et al. [1] described the use of hip arthroscopy to treat synovial chondromatosis. The authors reported good results and described the use of a medial portal to remove loose bodies in the medial anteroinferior and posteroinferior joint space. Probably the most common use of hip arthroscopy for removal of loose bodies is in the setting of traumatic dislocations. Mullis et al. [2] described the removal of traumatic loose bodies including head and posterior wall fragments in 36 patients. Interestingly, the authors found that 78 % of patient with a concentric reduction and no loose fragments on x-ray or CT actually had significant free cartilaginous pieces in the joint.

Multiple authors have also provided case reports for removal of bullets from the hip joint with good success [3–8]. Teloken et al. [8] described the use of an unusual inferomedial portal to assist with bullet removal that was difficult using standard techniques. Occasionally, surgical complications can result in loose bodies. Two authors have provided different techniques for the removal of a broken guidewire during the arthroscopic procedure [9, 10].

Lastly, germane to the topic of intra-articular free fragments is the concept of fixing some of these fragments. Matsuda describes a technique for arthroscopic fixation of a femoral head fracture that occurred during a traumatic dislocation. Regardless of the cause, hip arthroscopy has proven to be an effective tool in the treatment of intra-articular loose bodies.

Arthroscopic technique is important in the removal of loose bodies. However, equally important is a thorough understanding of the anatomy and the different portal options that are available. Furthermore, planning the procedure and having the appropriate equipment is essential. For example, the standard grasper in an arthroscopic hip set will not be large enough to grasp a bullet. Thus, the surgeon with inadequate instruments may have excellent technique but be unsuccessful in removing the offending object. Planning, preparation, and technique are all required for a successful and smooth arthroscopic procedure.

Surgical Technique

Prior to the arthroscopy, there are a couple things to plan for and essential points to understand:

1. With supine arthroscopy, most loose bodies will sit in the posterior aspect of the joint regardless of where they appear on preoperative imaging.
2. In the setting of trauma (hip dislocation), visualization can be significantly impaired secondary to excess tissue or clotted blood in the joint.
3. Understand all available portal options.
4. Be prepared! In many cases, a standard hip arthroscopy set will not have the appropriate equipment. Some instruments that may provide assistance:
   (a) A large grasper.
      (i) Arthrex AR-16400 is excellent for grasping bullets and other round hard objects.
      (ii) Smith & Nephew large “pitbull” grasper is good for grabbing large objects.
   (b) Punches and scissors can be used to make large fragments smaller.
   (c) Large cannulas can aid in the outflow of smaller loose bodies.

As described previously, standard arthroscopic technique is used to initiate the hip arthroscopy. The senior author prefers a supine arthroscopy set up with either a distractor attachment to a standard operating room bed or a fracture table. One to 1.5 cm of distraction is created across the hip joint. Depending on the size of the loose fragment, additional distraction may be required during removal. An anterolateral portal is established under spinal needle localization and fluoroscopic guidance. The next portal established is the anterior portal. However, the senior author will move the anterior portal more proximal than the standard anterior portal. The standard anterior portal is approximately 2–3 cm distal to a line extended from the top of the trochanter. However, during loose body removal, it is more important to have a direct path through the joint than a good angle on the acetabular rim. The anterior portal during loose body removal is typically in line with the tip of the greater trochanter. Spinal needle localization is used to confirm the appropriate trajectory into the joint.

Once the first two portals have been established, the fun begins. As noted above, every loose body is different and requires different techniques. The descriptions provided below will address some common issues facing a variety of pathologies.

The Gunshot

These can be very rewarding cases due to the fact that there is a defined discreet foreign object that can be removed with a dramatic improvement in patient symptoms. Preoperative CT scans should be obtained to both define the exact location of the fragment and identify other relevant pathology. There is a concern that a bullet traversing the intrapelvic region and ending in the hip joint may leave a tract to the intra-abdominal/pelvic cavity. This has not been reported but could potentially provide an outflow for arthroscopic fluid. In these cases, arthroscopic pressure and the amount of fluid being used should be monitored. An excessive amount of fluid use can signify extravasation. On CT scan and x-ray, the bullet may appear to be in the subchondral region (Fig. 1). The senior author has found this to be rarely the case; more commonly, the bullet is resting in the fovea and appears to be subchondral (Figs. 2 and 3). Essentially, the bullet may not appear accessible by arthroscopy, but in reality, it often is.

Once the location and pathology have been defined, appropriate equipment is necessary. A standard arthroscopic grasper will not be able to retrieve a bullet. The two graspers mentioned above are excellent options (Fig. 4). Additional

Fig. 1 X-ray of a patient that was shot in the buttock region. A previous surgeon attempted a bullet removal from an open posterior approach. This was unsuccessful. The bullet appears to be through the articular surface

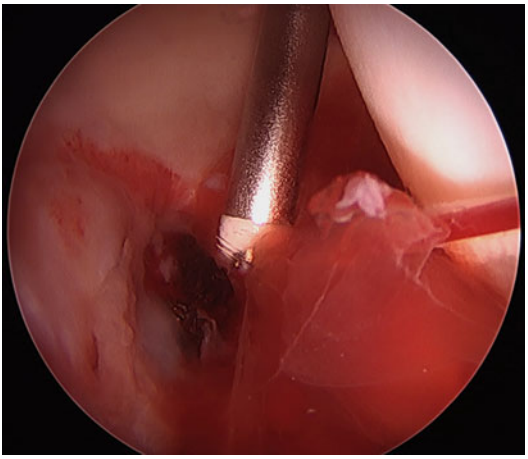


Fig. 2 Arthroscopic images of the same bullet in Fig. 1. The anatomy of the hip often obscures the true location of the fragment. This bullet was removed arthroscopically

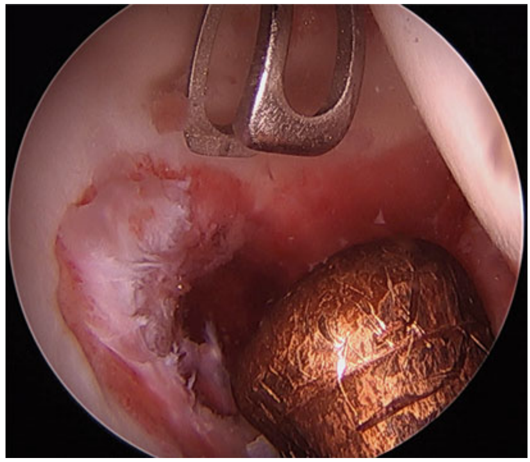


Fig. 4 Arthrex AR-16400 grasper used to remove the bullet from the hip joint

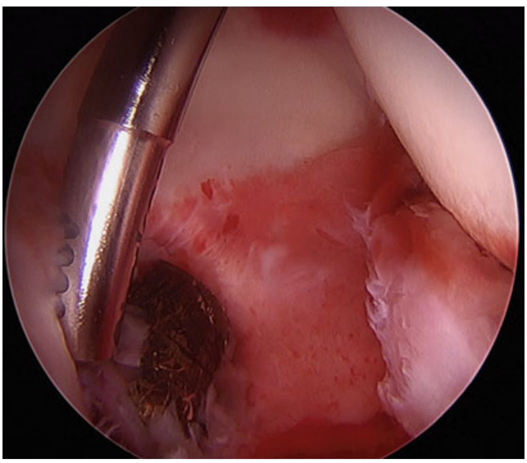


Fig. 3 A shaver is used to free the bullet from its resting place in the acetabular cartilage

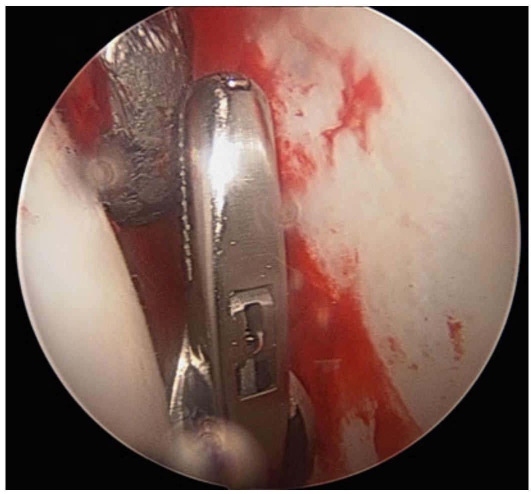


Fig. 5 A posterolateral portal is established and a second grasper is used to bring the bullet fragment more anterior and accessible

equipment that may be useful include threaded Steinmann pins that as described by Lee et al. [4] can be used to drive the threaded end into the soft bullet for control.

During supine hip arthroscopy, the bullet will most frequently remain in the posteroinferior aspect of the joint. This can make access from the anterior portal difficult. In these cases, a posterolateral portal should be established. This portal is created under spinal needle localization at the posterosuperior aspect of the greater trochanter. A switching stick or second grasper can then be used to "lever" the bullet out from the posterior aspect of the joint (Figs. 5 and 6) and support it while the grasper is used from the anterior portal to remove the fragment (Fig. 7). The anterior capsulotomy and portal will need to be large enough to support the bullet, and traction can be increased to allow the bullet to pass through the joint (Figs. 8 and 9).

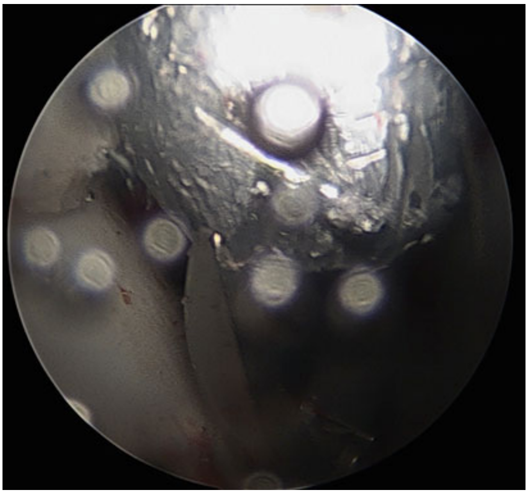


Fig. 6 The bullet is then brought into the anterior aspect of the joint where it is more accessible for removal

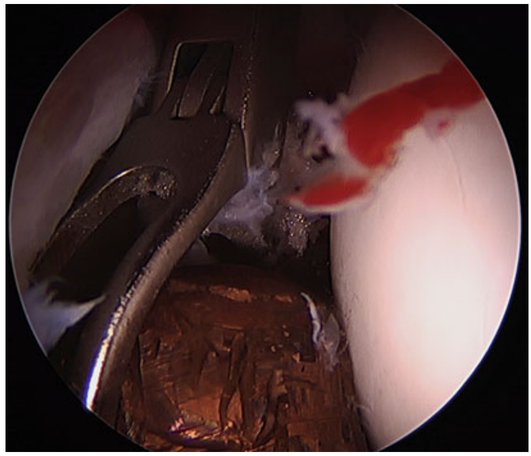


Fig. 8 Distraction is increased across the hip joint in order to accommodate the size of the bullet

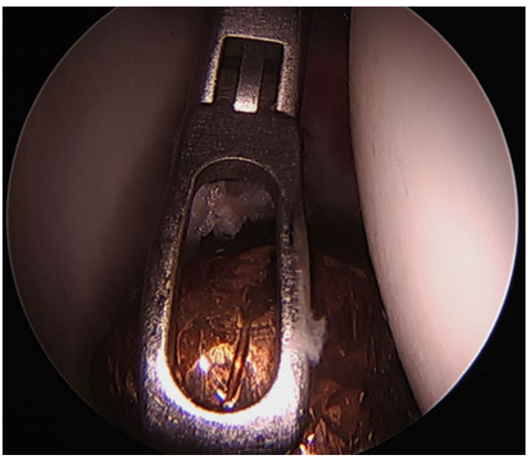


Fig. 7 A large grasper is used through the anterior portal to remove the bullet

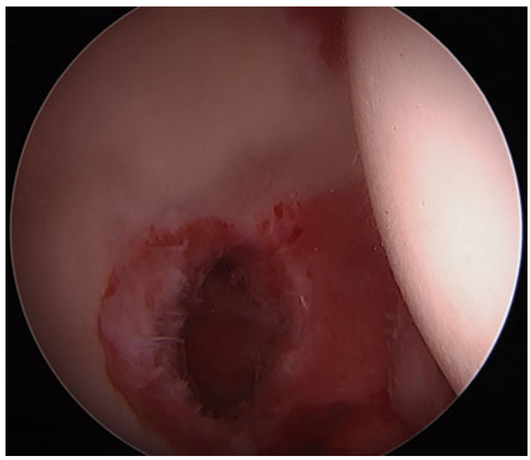


Fig. 9 Cavity after bullet removal from the patient in Fig. 1

The Posterior Wall or Femoral Head Fragment After a Hip Dislocation

These are the most common and reported case of loose body removal. Various authors have reported a high percentage of retained fragments after these injuries. The technique required is dependent on the fragment size. Matsuda has even fixed a femoral head fracture arthroscopically with screws [11]. However, more commonly, only fragment removal is required. One of the more difficult aspects to these cases includes the establishment of appropriate visualization. An acute injury often disrupts the hip capsule and brings additional tissue into the joint. This can make maintenance of pressure difficult and visualization tricky. An early outflow portal can provide assistance, but often the tissue is thick and additional measures are required to establish a view. The senior author typically uses a curved shaver for hip arthroscopy, but in these

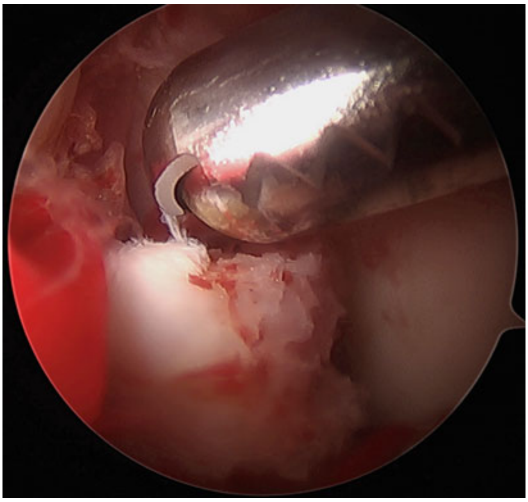


Fig. 10 A bone-cutting shaver is used to reduce the size of a large posterior wall acetabular fragment after a hip dislocation

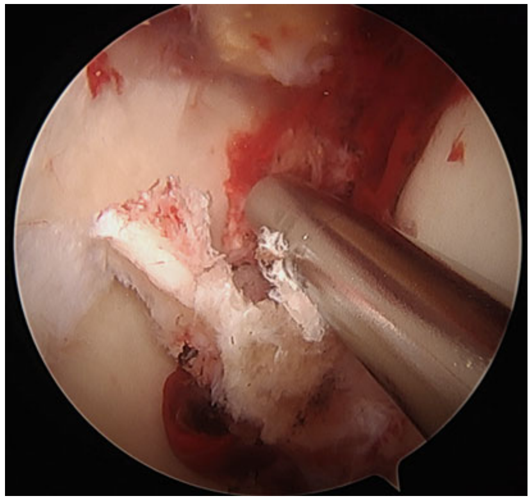


Fig. 11 Reduction in size of the posterior wall fragment

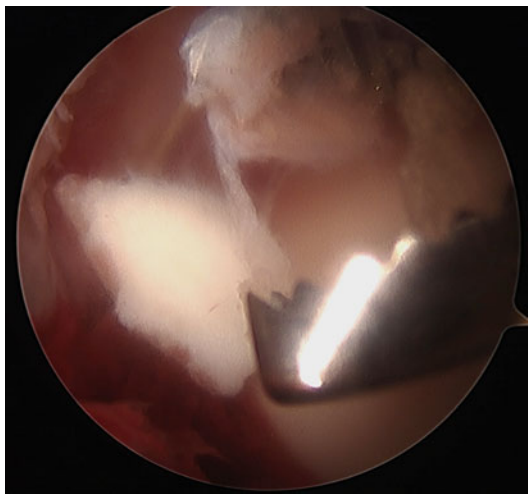


Fig. 12 Removal of the remaining posterior wall fragment with a grasper

cases a straight shaver can be very beneficial. An outflow spinal needle can be established blindly and confirmed by the outflow of fluid. A nitinol wire is then used to establish a full outflow cannula. This too is occasionally necessary to do blindly. Lastly, the straight shaver can be inserted through the cannula, directed away from the labrum and used on oscillate to remove this tissue. In the rare circumstance, the shaver can also be used blindly in the joint.

Once visualization is established, the fragment can be identified and removed with the appropriate technique. Small fragments can be removed with the use of a grasper or a bone-cutting shaver if necessary. The bone-cutting shaver is also an excellent tool to reduce the size of large fragments (Fig. 10). These fragments can be difficult to "bite" with a grasper or biter due to the fact that any attempt can push the fragment into a more inaccessible location. The shaver uses suction to pull the fragment to the shaver. These large fragments are fairly simple to reduce in size and remove with standard means (Figs. 11 and 12). Again, it should be noted that fluid pressure and quantity should be monitored in the cases.

The Broken Guidewire

The best way to treat this problem is to avoid it. However, it does happen. In the case of a small fragment that exists in the intra-articular space, a grasper can be used. If the piece cannot be reached with standard arthroscopic techniques, there is a piece of equipment that can be very very useful, a magnetic suction device. Many companies have them available, but these are not readily available. Talk to your representative about having one around your OR. These are effectively suction units with a magnetic tip. They are excellent for retrieving hard to reach metallic fragments and may save the day at some point in your arthroscopic career. For the broken guidewire that resides in the soft tissues and the joint, the portal may need to be extended and fluoroscopy can be used to locate the tip. This can then be removed.

Synovial Chondromatosis

Lastly, synovial chondromatosis can create multiple loose bodies in both the central and the peripheral compartment. A large open cannula can be used to remove the majority of these small fragments. A standard arthroscopy is initiated including a posterolateral portal. The large cannula is then rotated between the anterior, anterolateral, and posterolateral portals as the fluid source is also changed. The remaining fragments can be removed using a shaver or grasper as needed. After evacuation of the central compartment, traction should be removed and a thorough irrigation/evaluation of the peripheral compartment completed with the hip in approximately 30 degrees of flexion. Occasionally, a T-capsulotomy is required to access all loose fragments in the peripheral compartment. This can be completed by establishing a mid-anterior or accessory anterolateral portal in line with the femoral neck. A blade can then be used through this portal to create the capsulotomy.

Summary

Arthroscopic loose body removal in the hip joint can be a rewarding experience for both the patient and the surgeon. The patient avoids the significant morbidity of an open approach and is able to return to activity much quicker. That being said, the concept of removal is simple, but the reality of extraction can be more complicated. The arthroscopic surgeon must appropriately plan and prepare for the case. This includes the identification of the fragment location and any associated pathologies that may affect the case, selection and attainment of appropriate equipment, and a thorough understanding of the various portals available during hip arthroscopy. These are not easy cases and need to be respected.

References

1. Lee JB, Kang C, Lee CH, Kim PS, Hwang DS. Arthroscopic treatment of synovial chondromatosis of the hip. Am J Sports Med. 2012;40(6):1412–8.
2. Mullis BH, Dahners LE. Hip arthroscopy to remove loose bodies after traumatic dislocation. J Orthop Trauma. 2006;20(1):22–6.
3. Al-Asiri J, Wong I. Arthroscopic bullet removal from the acetabulum (hip joint). J Surg Tech Case Rep. 2012;4(2):121–5.
4. Lee GH, Virkus WW, Kapotas JS. Arthroscopically assisted minimally invasive intraarticular bullet extraction: technique, indications, and results. J Trauma. 2008;64(2):512–6.
5. Mineo RC, Gittins ME. Arthroscopic removal of a bullet embedded in the acetabulum. Arthroscopy. 2003;19(9):E121–4.
6. Singleton SB, Joshi A, Schwartz MA, Collinge CA. Arthroscopic bullet removal from the acetabulum. Arthroscopy. 2005;21(3):360–4.
7. Sozen YV, Polat G, Kadioglu B, Dikici F, Ozkan K, Unay K. Arthroscopic bullet extraction from the hip in the lateral decubitus position. Hip Int J Clin Exp Res Hip Pathol Ther. 2010;20(2):265–8.
8. Teloken MA, Schmietd I, Tomlinson DP. Hip arthroscopy: a unique inferomedial approach to bullet removal. Arthroscopy. 2002;18(4):E21.
9. Ilizaliturri Jr VM, Zarate-Kalfopulos B, Martinez-Escalante FA, Cuevas-Olivo R, Camacho-Galindo J. Arthroscopic retrieval of a broken guidewire fragment from the hip joint after cannulated screw fixation of slipped capital femoral epiphysis. Arthroscopy. 2007;23(2):227e1–4.
10. Salamon PB. Broken guide pin: a method of retrieval from within the hip joint. Orthopedics. 2002;25(3):299–301.
11. Matsuda DK. A rare fracture, an even rarer treatment: the arthroscopic reduction and internal fixation of an isolated femoral head fracture. Arthroscopy. 2009;25(4):408–12.

Part VII

Avascular Necrosis

Hip Avascular Necrosis: Overview

83

Matthew T. Houdek, John R. Martin, and Rafael J. Sierra

Contents

M.T. Houdek • J.R. Martin • R.J. Sierra (✉)
Department of Orthopedic Surgery, Mayo Clinic, Rochester, MN, USA
e-mail: houdek.matthew@mayo.edu; martin.john@mayo.edu; sierra.rafael@mayo.edu

Abstract

Avascular necrosis (AVN) of the femoral head is a progressive disease that predominantly affects younger patients. The disease is characterized by a vascular insult to the femoral head blood supply, which can lead to collapse of the femoral head and subsequent degenerative changes. The exact pathophysiology underlying AVN has yet to be elucidated, although a number of risk factors have been determined. The diagnosis of AVN is commonly made based on clinical and radiological findings. Radiographs are initially performed; however, MRI has become the gold standard for diagnosis and has proven beneficial in staging patients. Most surgeons attempt to diagnose and treat AVN in the early stages, prior to collapse of the femoral head. There are a number of nonoperative and operative treatments that have been investigated for early stage AVN. Nonoperative modalities include statins, stanozolol, and bisphosphonates. Operative treatments include core decompression alone, core decompression with injection of bone marrow aspirate, or placement of some form of bone substitute and osteotomies. Nonsurgical options are ineffective after femoral head collapse, and treatment options become more invasive. Bone-preserving techniques utilized for post-collapse AVN include vascularized fibula grafts and osteotomies, but their results are unpredictable. The ultimate salvage options include hip resurfacing, bipolar

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_92

arthroplasty, and total hip arthroplasty. While the reconstructive options offer excellent pain relief, there are many operative and clinical considerations given to the young age of this patient population.

Introduction

Background

Avascular necrosis (AVN) of the femoral head occurs when the osteocytes of the trabecular bone spontaneously die [1]. In the United States, this process affects up to 20,000 new patients every year, predominantly in patients younger than 40 years of age [2–4]. Currently the pathogenesis of the disease process is thought to be multifactorial and has not been fully explained. Although it is not currently known what causes the death of the osteocytes, in a majority of patients, collapse of the femoral head is likely to occur [5]. Once collapse has occurred, these patients often necessitate a total hip arthroplasty (THA) for pain relief and improvement in daily function [5–8].

Table 1 Risk factors for AVN of the femoral head

Traumatic/direct injury
Femoral neck/head fracture
Hip dislocation
Slipped capital femoral epiphysis
Nontraumatic
Corticosteroid use
Alcohol abuse
Idiopathic
Sickle cell disease
Caissons disease
Systemic lupus erythematosus
Cushing disease
Organ transplantation
Prior radiation therapy
Smoking
Pregnancy
Chronic pancreatitis
Coagulopathy
Chronic renal failure
Gaucher disease
Arteritis
Disseminated intravascular coagulation
Lipid disorders

Etiology

Excluding traumatic or direct injury to the vascular supply, the pathogenesis of AVN of the femoral head has yet to be elucidated. Typically, patients have a history of a "risk factor" which has been associated with AVN. A multitude of different factors are thought to be risks, most commonly alcohol abuse and corticosteroid use [9, 10]; however, HIV, radiation exposure, smoking, pregnancy, autoimmune conditions, and coagulopathies, among others, have also been implicated in the pathogenesis of the disease process (Table 1). Even with these known associations, in a quarter of patients, there is no associated risk factor for the development of AVN and is termed "idiopathic AVN." Although the exact pathogenesis is not fully understood, the common end point is necrosis of the trabecular bone and bone marrow of the femoral head [2].

Today, AVN is thought of as a multifactorial disease process including the risk factors previously listed and also in some patients a genetic predispositions to the development of disease. In a coagulation study, it was noted that 82 % of patients with AVN had at least one coagulation abnormality [11, 12]. Patients with AVN have also been found to have genetic polymorphisms in the genes for endothelial nitric oxide synthase (eNOS) which is involved in regulating the tone of blood vessels [13–16]. Genetic polymorphisms in the genes involved with collagen production, as well as alcohol and steroid metabolism, have been identified in patients with AVN [17–20]. Investigating for these rare associations is important, as treatment success for AVN can potentially be predicated upon the presence or absence of these risk factors.

Presentation

Patients with AVN typically are less than 40 years of age and present with a complaint of groin pain; however, the intensity of pain varies depending on the stage of AVN. They will typically deny any traumatic injury to the hip, and many will present with a history of a known risk factor. Pain in early stages is secondary to synovitis and an increased pressure in the femoral head as described by Ficat, as a result of venous congestion within the femoral head [1]. Patients without collapse of the femoral head typically complain of an insidious onset of a dull, deep ache; however, once subchondral fracturing and collapse occurs, the pain can become acutely severe [1]. In the early stages of disease, pre-collapse, patients will frequently have a normal range of motion; however, this will become limited secondary to pain with advanced stages of disease, especially with forced internal rotation of the hip. In advanced stages of AVN, after collapse of the femoral head has led to degenerative changes, patients will have findings consistent with end-stage osteoarthritis.

Imaging

Radiographic analysis of the hip should begin with anteroposterior (AP) and frog-lateral radiographs (Fig. 1). Since a majority of patients with AVN will have bilateral involvement, it is important to also evaluate the contralateral side [2]. The frog-leg lateral view is important to identify subtle changes in the subchondral bone of the femoral head which can be missed on AP and cross-table lateral views and is important in staging of the disease [2]. Plain radiographs have also been historically used to determine the proportion of the femoral head involved by measuring the arc of involvement on the AP and lateral radiographs and adding these together [21].

Currently, there is no modality that is 100 % sensitive and specific to diagnose AVN of the femoral head. If a patient presents with a history and physical exam consistent with a diagnosis of AVN, an MRI can be ordered to diagnose and also evaluate the extent of AVN [2, 22–24]. The use of MRI is very effective in detecting AVN at the earliest stages and is 98 % sensitive and 98 % specific. Initially edema appears in the femoral head as a hypointense signal on T1-imaging and hyperintensity on T2 [22, 23].

Staging

In order to formulate a treatment plan, accurate staging of the disease is imperative. Staging of the disease also allows the physician to be able to counsel the patient on the disease course and treatment options. Historically, the classification system by Ficat (Table 2) is most commonly used [1]. This system uses plain radiographs to evaluate the femoral head and separates early from late-stage AVN by the presence of a "crescent sign," signifying a fracture in the subchondral bone [1]. Similar to the Ficat classification, the Steinberg classification (Table 2) also uses the "crescent sign" to separate early from late-stage AVN [23].

With the advancement of MRI, it has become easier to detect the subtle changes of early AVN [22, 23]. Since physicians are able to detect AVN at an earlier stage, MRI is used to determine characteristics which aid in predicting outcomes in certain patients [24–26]. The most important prognostic factor is the percent involvement of the weight-bearing portion of the femoral head.

The Association Internationale de Recherche sur la Circulation Osseuse (ARCO) developed a classification system that is the most complete [27]. The ARCO classification combines plain film radiographs, MRI, and bone scan findings to determine different stages of disease (Table 3); however, due to the multiple stages which a patient can fall into, it is difficult to use and apply in clinical practice.

Treatment Options

Treatment modalities for AVN can be divided into two main categories: pre-collapse and post-collapse. Many of the treatments that are utilized in the pre-collapse phase of AVN are not effective once the femoral head has collapsed. If the disease process is halted in the pre-collapse phase, patients can avoid THA and other salvage-type

Fig. 1 Corresponding radiographs (AP and frog leg lateral) and MRIs (coronal T1 and T2) for patients with various stages in the Steinberg classification [23]. *Stage 1* (**a**–**d**) shows no radiographic features of AVN (**a** and **b**), however, features readily apparent on MRI (**c** and **d**). *Stage 2* (**e**–**h**) shows subtle increase sclerosis in the femoral head on radiographs (**e** and **f**) and features of AVN on MRI (**g** and **h**). *Stage 3* (**i**–**l**) shows collapse of the articular surface with a "crescent sign" on radiographs (**i** and **j**), along with corresponding MRI images (**k** and **l**). *Stage 4* (**m**–**p**) radiographs show flattening of the femoral head without joint space narrowing (**m** and **n**), along with corresponding MRI images (**o** and **p**). *Stage 5* (**q**–**t**) radiographs show joint space narrowing, with minimal involvement of the femoral head (**q** and **r**), along with corresponding MRI images showing AVN (**s** and **t**). *Stage 6* (**u**–**x**) radiographs show advanced degenerative changes (**u** and **v**), along with corresponding MRI images showing AVN (**w** and **x**)

Table 2 Radiographic stages for AVN of the femoral head

Stage	Ficat classification [1]	Steinberg classification [23]
0	No symptoms	No symptoms
	Normal X-rays	Normal X-rays
	MRI nondiagnostic	MRI nondiagnostic
1	Mild pain in the affected hip	Mild pain in the affected hip
	Pain with internal rotation	Pain with internal rotation
	Normal X-ray	Normal X-ray
	MRI diagnostic	MRI diagnostic
2	Worsening or persistent pain	Worsening or persistent pain
	Increased sclerosis or cysts in the femoral head	Increased sclerosis or cysts in the femoral head
3	Subchondral collapse producing a crescent sign	Subchondral collapse producing a crescent sign
	Flattening of the femoral head	
	Normal joint space	
4	Collapse of head	Flattening of the femoral head
	Flattened head	Normal joint space
	Decreased joint space	
5		Joint space narrowing with/without femoral head involvement
6		Advanced degenerative changes

Table 3 ARCO classification for AVN of the femoral head [27]

Stage	Findings	Femoral head involvement
0	Negative plain radiographs, CT, bone scan and MRI	None
I	MRI or bone scan positive	I-A: <15 %[a]
		I-B: 15–30 %[a]
		I-C: >30 %[a]
II	Plain radiographs show osteosclerosis, cyst formation, and osteopenia	II-A: <15 %[a]
	No crescent sign	II-B: 15–30 %[a]
		I1-C: >30 %[a]
III	Presence of crescent sign	III-A: <15 % crescent sign or <2 mm depression of femoral head[b]
		III-B: 15–30 % crescent sign or 2–4 mm depression of femoral head[b]
		III-C: >30 % crescent sign or 4 mm depression of femoral head[b]
IV	Joint space narrowing with acetabular degenerative changes (sclerosis, cyst formation and osteophytes)	

[a]Based on MRI findings
[b]The crescent sign percentage is the amount of the femoral head that the crescent sign forms on the AP and lateral radiographs

procedures. This group encompasses stages 0–2 of the Steinberg classification.

Pre-collapse AVN

Nonoperative Modalities

Statins

Hyperlipidemia is commonly seen in patient with AVN. One study noted that 12 out of 19 patients who were receiving corticosteroids and developed AVN had elevated lipid levels, with many patients requiring a lipid lowering agent [28]. Interest has grown for the use of statins both in prevention and treatment of early-stage AVN. Pritchett reviewed a series of 284 patients who received steroids for a variety of reasons [29]. All patients received at least one statin during this time, some patients received two or more. Only 1 % of patients developed AVN in the statin treatment group. It is believed that etiologies such as fat emboli and metabolic abnormalities contribute to the development of AVN in this patient population [29]. The exact mechanism for the effectiveness of statins has yet to be fully understood. Experimentally, statins have been found to lower the intramedullary pressure and increase femoral bone density in femoral head AVN [30, 31].

Stanozolol

A hypercoagulable state has been hypothesized to contribute to the pathogenesis of AVN. One study investigated the relationships between certain hypercoagulable states and the development of AVN. It was found that a Factor V Leiden mutation as well as homocysteinemia was more common in patients with multifocal AVN than in the control groups [13]. Medications that can reverse their hypercoagulable states are currently under investigation for the treatment of early-stage AVN. Stanozolol, a synthetic steroid, which is commonly taken orally for anemia and hereditary angioedema is one of these agents. Glueck studied a group of five patients with AVN; four of these patients had hypofibrinolysis. All patients were treated with Stanozolol. Three of the patients had normalization of hypofibrinolysis and resolution of their hip symptoms, although one progressed radiographically [14].

Bisphosphonates

Bisphosphonates work by inhibiting osteoclasts which prevents bone resorption which leads to net bone formation and are commonly used to treat osteoporosis. These properties are appealing in the setting of AVN, since it is thought that collapse of the femoral head is due to osteoclasts removing necrotic subchondral bone. To this end, alendronate has been studied in the treatment of AVN. The results indicate that patients had significantly less pain, increased their walking time and also hip range of motion. Radiographic progression was typically minimal; however, 10 % of patients required surgery during the study [32, 33]. The use of bisphosphonates for the treatment of AVN has also been shown to be efficacious in randomized clinical control trials. Alendronate or placebo was given to patients with Steinberg stage IIC or IIIC nontraumatic AVN of the femoral head [34]. At 24-months of follow-up, in the patients who received alendronate, only two patients (7 %) went onto collapse, while 19 (76 %) in the placebo group collapsed [34]. However, in a recent study by Chen, similar patients with Steinberg stage IIC or IIIC AVN were again randomized to receive alendronate or placebo to determine the effect of the medication on the progression of AVN and subsequent need for total hip arthroplasty [35]. The results of this study showed no difference between the alendronate and placebo group with 13 % of the alendronate group and 15 % of the placebo group progressing to total hip arthroplasty [35].

Operative Modalities

Core Decompression

Increased femoral head intramedullary pressure is thought to be part of the pathophysiology of AVN. The increase in pressure decreases blood flow through the retinacular vessels within the femoral neck which provide perfusion of the femoral head. With decreased perfusion of the terminal aspect of the femoral head, necrosis subsequently develops. Core decompression has been proposed as a surgical option to reduce the intramedullary pressure within the femoral head [36]. Core decompression is a relatively minor outpatient surgery that is typically only used in early AVN before femoral head collapse has occurred. The procedure is performed supine on a radiolucent table. Fluoroscopic imaging is required to localize the areas of AVN of the femoral head. Through a lateral incision over the greater trochanter, a guide wire can be inserted through the femoral neck and into the femoral head under fluoroscopy. Cores are then removed from the femoral head and neck to decrease the intramedullary pressure. Caution should be utilized when approaching areas of necrosis as this can dislodge the necrotic lesion from the femoral head.

Hungerford and Ficat were among the first to describe the utilization of core decompression for Ficat stages 0, 1, and 2 AVN [37]; however, core decompression was initially described by Graber-Duvernay for osteoarthritis in 1932. Core decompression, in the early setting, has been found to delay progression in approximately 60 % of patients. Postoperatively patients are usually made protected weight bearing for 1–2 weeks depending on the size of core that was taken intraoperatively, with most patients able to wean from crutches within 1 month of surgery. Complications associated with the procedure include surgical site infection, postoperative femoral head collapse, and subtrochanteric fracture. The risk of subtrochanteric fracture can be minimized by ensuring that the lateral cortex of the femur is entered proximal to the level of the lesser trochanter.

Autologous Bone Marrow Injection

Core decompression was one of the first surgical interventions for AVN in the early stages of disease progression. It is an outpatient procedure, and most patients make a rapid recovery. The decompression immediately decreases the intramedullary pressure, which can lead to instantaneous resolution of symptoms. However, it was believed that core decompression alone was not enough to promote healing of the AVN site. Hernigou described a technique where bone marrow was aspirated from the iliac crests [38]. The bone marrow aspirate was then concentrated and then injected into the femoral head in the areas of avascular necrosis using fluoroscopy to accurately guide the injections [38].

The introduction of bone marrow aspirate into the areas of AVN has several potential benefits. Bone marrow aspirate contains mesenchymal stem cells, which are pluripotent cells, possessing the ability to differentiate into various mesenchymal cell lines, including osteoblasts. Additionally bone marrow has osteoinductive properties, containing bone morphogenic proteins (BMPs). Since the areas of AVN have no living osteoblasts/osteocytes and no blood flow to reconstitute these deficiencies, injecting the concentrated bone marrow allows the introduction of osteogenic and osteoinductive agents directly to the sites of necrosis. Multiple randomized prospective trials have compared core decompression to core decompression with bone marrow and the results have continually favored the use of the latter [39–41]. Core decompression with bone marrow has been shown to decrease pain and rates of radiographic progression, improve function, and prevent the need for the further surgical intervention [42–44]. Likewise with the addition of the bone marrow, studies have shown evidence of healing based on MRI, compared to core decompression alone in pre-collapse AVN [42–44]. The addition of platelet-rich plasma to the bone marrow concentrate has been recently described. The platelet-rich plasma adds additional growth factors such as VEGF, PDGF, and FGF which may increase the rate of healing of AVN lesions [44].

Operative Modalities After Collapse

Femoral head collapse signifies the end of preventative measures in the treatment of AVN. Medical treatment and the previously described surgical options are ineffective in this stage. Surgical intervention changes from attempting to reverse the early effects of AVN and moves toward salvage operations. Some surgeons favor the use of a vascularized fibula graft, while others use osteotomies to move the area of femoral head collapse from the weight-bearing regions of the hip joint. These options are often attempted initially in young AVN patients. Avoiding arthroplasty options initially may prevent patients from undergoing multiple revision operations if they are able to preserve their native hip joints. If the femoral head collapse is significant, patients can develop severe, uncontrollable pain, along with severe degenerative changes very rapidly that necessitates hip arthroplasty. Several surgical options have been utilized in this patient population including hip resurfacing, hemiarthroplasty, and total hip arthroplasty.

Vascularized Fibula Grafts

The use of vascularized fibular graft has been utilized for this since the 1980s and is advocated since it allows for the maintenance of the patient's own bone stock. Vascularized fibula grafts can be utilized in the pre- and post-collapse populations. Aldridge et al. [45] studied a series of 224 hips in patients with post-collapse AVN. They noted an overall survival rate of 67.4 % at a minimum of 2-year follow-up, with a significantly improved Harris Hip Scores.

Performing a vascularized fibula graft requires microvascular surgery, a skilled operating room staff, and often two separate surgical teams to perform this complex procedure. The two separate procedures, harvesting the fibula and preparation of the femur, required for this procedure can be performed simultaneously if the appropriate surgical staff is available. One surgical team harvests a 15-cm fibula autograft along with the peroneal vessels from either side. Meanwhile, the other surgical team exposes the ascending branch of the lateral circumflex femoral artery and decompresses the femur. The necrotic segment of the femur can be resected under fluoroscopic guidance from a lateral femoral entry point similar to a core decompression. An attempt is made at impacting the collapsed segment of femoral head by placing bone graft into defect. After the femoral head collapse has been restored and the femur is prepared, the fibula autograft is impacted into the femoral head, confirming the location by fluoroscopy. The graft is then anastamosed to the previously prepared anterior branch of the lateral circumflex femoral vessels [46].

Vascularized fibula graft has been a very promising surgical option for those with post-collapse AVN. The survival rate appears to be stable at 2- and 5-year follow-up, with overall survival of 67.4 % and 64.5 %, respectively [45]. Adequate decompression of the femoral head likely decreases the intramedullary pressure and theoretically increased blood flow to the femoral head; along with bone graft being introduced in previous areas of AVN, this introduces an osteoconductive and osteoinductive environment for healing. After impacting the area of femoral head collapse back to the pre-collapse site, the fibula graft is positioned to support this area in attempt to buttress it from collapse, improving and accelerating healing. Although many benefits have been realized by this procedure, patients with larger femoral head defects have not had as much success [23]. Additionally, due to the increased complexity of the surgery, there are many potential associated complications. Graft failure, clawing of the big toe, infection of both the donor site and the femur, fracture of the tibia, nerve injury, and donor site pain have all been previously described complications [47].

Osteotomies

The use of femoral osteotomies has been described for several pathologies including trauma, severe coxa vara and valga, slipped capital femoral epiphysis (SCFE), etc. Femoral osteotomies allow the surgeon to reorient the center of the femoral head within the acetabulum. The same osteotomy can also be used to position the pathologic portion of the femoral head away from the weight-bearing portion of the joint. In AVN the anterosuperior aspect of the femoral head is most commonly necrotic, leaving the posteroinferior femoral head intact. Through osteotomies, the necrotic segment of the femoral head can be moved out of the weight-bearing segment of the acetabulum. Two osteotomies, rotational and angular intertrochanteric, have been used to accomplish this goal. Similar to vascularized free-fibula grafts, osteotomies can also be performed in the pre-collapse as well as the post-collapse patient population.

Rotational Transtrochanteric Osteotomy

Rotational transtrochanteric osteotomy was initially described by Sugioka [48]. Three osteotomies are made during this surgery: a greater trochanteric osteotomy, a superomedial to interolateral intertrochanteric osteotomy, and one between the proximal flare of the lesser trochanter and the femoral shaft [49]. The femoral head is typically rotated anteriorly 80–90°, shifting the intact femoral cartilage of the posteroinferior femoral head into the position previously occupied by the necrotic femoral head. This osteotomy is used in patients with necrotic lesions involving the weight-bearing region of the femoral head, an uninvolved posterior femoral head, and lack of arthritis [48]. This osteotomy is technically very challenging, and complications include necrosis of the entire femoral head, joint incongruity, nonunion, and significant degenerative changes. In select patient populations, the rotational transtrochanteric osteotomy can be a great surgical option. In a study of 295 patients treated in this manner, 79 % of patients were found to have good to excellent results [48].

Angular Intertrochanteric Osteotomy

Varus and valgus intertrochanteric osteotomies have both been used in patients with AVN. Prior to this procedure, it is important to assess hip range of motion to ensure that patients will have adequate postoperative range of motion after each type of osteotomy is performed. Excessive angulation produced by the osteotomy may predispose patients to contractures and gait abnormalities. In patients with anterolateral necrotic lesions, a valgus flexion osteotomy is most commonly used. Similar to the rotational osteotomy, patients must have small lesions and the osteotomy should reposition the necrotic segment outside of the weight-bearing area. In a series of 43 patients treated with a valgus flexion osteotomy, 87 % had good to excellent results at 65 months follow-up [50]. In patients with medial lesions that have intact lateral femoral heads, varus osteotomies are typically utilized. Similar results have been described for varus osteotomies with good or excellent results in 74 % of patients at 11 year follow-up [51]. Varus osteotomies are often combined with flexion or extension depending if anterior or posterior lesions are involved, respectively.

Joint Reconstruction

There are multiple surgical options in the setting of femoral head collapse. Joint reconstruction with the use of a prosthesis remains the ultimate salvage operation in patients with end-stage AVN. All of the previously described surgical options for joint preservation are contraindicated in the setting of arthritis. Reconstruction offers immediate pain relief and drastic improvements in function in this patient population. Most surgeons prefer to postpone the use of reconstruction techniques in this young group as long as possible; however, in patients that have failed other salvage techniques, those with arthritis, and those with intractable symptoms are best managed by hip reconstruction.

Bipolar

Bipolar hemiarthroplasty is most commonly used in the treatment of displaced femoral neck fractures in elderly patients. The procedure requires no preparation of the acetabulum and therefore can be performed faster than a total hip arthroplasty (THA). Because the acetabulum is not resurfaced, articular cartilage loss in the acetabulum can be a source of pain and results may not be as consistent as THA. Replacing the femoral head with a bipolar completely removes the necrotic segments of the femoral head, effectively removing the pathology. The use of a bipolar hemiarthroplasty for patients with AVN has been disappointing. One study examined a series of 31 hips treated with a bipolar; 16 hips were found to have fair or poor results at an average follow-up of 4.6 years. Radiographic changes, including narrowing of the femoral prosthesis and acetabulum, were noted in 14 patients [52]. These poor results are likely multifactorial; however, most surgeons have abandoned bipolar hemiarthroplasty for other reconstruction methods [53].

Hip Resurfacing

Hip resurfacing is typically reserved for young active male patients with osteoarthritis, who would like to continue with their active lifestyles. Femoral head resurfacing maintains adequate bone stock in the femoral neck, and therefore, revision surgery should be easier than a standard revision total hip arthroplasty. Resurfacing of the femoral head removes the areas of necrosis and replaces the femoral head with metal bearing surface. Hip resurfacing is more stable than a standard total hip arthroplasty because of the large femoral head. In a study of 29 hips that underwent femoral head resurfacing for AVN, Harris Hip Scores and patient function were found to significantly improve. The overall survivorship of this patient population at 3-years was 75.9 % [53]. Three postoperative complications were noted: one femoral neck fracture, one persistently draining incision site, and one hip dislocation. In the patient with the femoral neck fracture, it was converted to a THA, the persistently draining incision site eventually quit draining with no further management, and the patient with the dislocation eventually required a THA for continued postoperative pain. Hemiresurfacing has been advocated as a predictable alternative to hemiarthroplasty and avoids the use of a polyethylene bearing surface [53].

Total Hip Arthroplasty

Total hip arthroplasty is the final salvage operation for all patients with AVN which have not been adequately treated by the other methods previously described. Total hip arthroplasty has been shown to reliably eliminate pain and improve function in patients with post-collapse AVN; however, some believe that THA for AVN has been less successful in the long term than for other etiologies [54]. Younger patients, along with increased activity have significant effect on the overall outcome on these patients treated with THA. Ritter and Meding reviewed a cohort of matched patients with 105 hips with AVN and 891 hips with osteoarthritis. The AVN group was younger on average than the osteoarthritis group, but the groups had similar complication and revision rates [54]. These results suggested that THA for AVN is a reliable and durable treatment option. However, the authors noted that longer follow-up and increased patient populations would be required to determine absolute recommendations [54].

Total hip arthroplasty in patients with AVN has several special surgical considerations that will likely increase the longevity of the implants. Femoral fixation with cement has improved over the last few decades [55]. While cementing techniques have improved, many surgeons have moved toward biologic fixation with bone ingrowth stems. In a study of 53 bone ingrowth stems, 100 % of stems had stable ingrowth at a minimum of 5-year follow-up [56]. Instability has also been found to be twice as common as regards dislocation rate when comparing patient with AVN with those with osteoarthritis [57]. Additionally, polyethylene wear and osteolysis are concerns in this young patient population. The wear characteristic of highly cross-linked polyethylene has been well documented and will likely lead to an improved wear characteristics in patients with AVN as well. Likewise alternative bearing surfaces including ceramic-on-ceramic and ceramic-on-polyethylene have also been shown to have improved wear characteristics [58].

Summary

Avascular necrosis of the femoral head is a complex and intriguing medical condition. Currently, the exact pathophysiology behind AVN is unknown, but many theories and risk factors to explain this clinic entity exist. AVN commonly effects younger patient populations than osteoarthritis, typically with an insidious onset of groin pain. However, when the femoral head collapses, a sudden and severe presentation of pain is most common. Physical examination findings are often specific for hip pathology, but not necessarily AVN.

Imaging of AVN often begins with plain radiographs, which may reveal subtle signs of sclerosis of the femoral head. More advanced stages may show the classic crescent sign, which indicates subchondral collapse. MRI is the most sensitive and specific imaging modality for AVN and femoral head involvement. MRI is very useful in selecting treatment options for the patient and may be useful in screening patients at risk for developing the problem after exposure to a risk factor, with larger lesions more likely to be treated operatively.

Treatment options for AVN are divided into pre-collapse and post-collapse options. Pre-collapse AVN treatment modalities include both surgical and nonsurgical options. Commonly used nonsurgical options for pre-collapse AVN include statins, stanozolol, and bisphosphonates with varying degrees of effectiveness. Surgical options for this group include core decompression and core decompression with bone marrow. Vascularized fibula bone graft and femoral intertrochanteric osteotomies can be used before and after collapse has occurred. These options are commonly advocated in post-collapse AVN prior to joint reconstruction because of the young patient population and the uncertainty of the longevity of THA implants. Reconstruction techniques are the ultimate salvage option and are commonly reserved for patients with degenerative changes or those that have failed a different surgical option. Reconstruction options include bipolar hemiarthroplasty, resurfacing, and THA. Advances in cement techniques, bone ingrowth surfaces, and bearing surfaces should improve the long-term survival of these implants.

References

1. Ficat RP. Idiopathic bone necrosis of the femoral head. Early diagnosis and treatment. J Bone Joint Surg Br. 1985;67(1):3–9.

2. Mont MA, Hungerford DS. Non-traumatic avascular necrosis of the femoral head. J Bone Joint Surg Am. 1995;77(3):459–74.
3. Mont MA et al. Osteonecrosis of the femoral head. Potential treatment with growth and differentiation factors. Clin Orthop Relat Res. 1998;(355 Suppl): S314–35.
4. Lavernia CJ, Sierra RJ, Grieco FR. Osteonecrosis of the femoral head. J Am Acad Orthop Surg. 1999;7 (4):250–61.
5. Mont MA, et al. The natural history of untreated asymptomatic osteonecrosis of the femoral head: a systematic literature review. J Bone Joint Surg Am. 2010;92(12):2165–70.
6. Bozic KJ, Zurakowski D, Thornhill TS. Survivorship analysis of hips treated with core decompression for nontraumatic osteonecrosis of the femoral head. J Bone Joint Surg Am. 1999;81(2):200–9.
7. Ito H, et al. Mid-term prognosis of non-traumatic osteonecrosis of the femoral head. J Bone Joint Surg Br. 2003;85(6):796–801.
8. Hernigou P, Bachir D, Galacteros F. The natural history of symptomatic osteonecrosis in adults with sickle-cell disease. J Bone Joint Surg Am. 2003;85-A(3):500–4.
9. Hungerford DS, Zizic TM. Alcoholism associated ischemic necrosis of the femoral head. Early diagnosis and treatment. Clin Orthop Relat Res. 1978;130:144–53.
10. Cruess RL. Steroid-induced osteonecrosis: a review. Can J Surg (J canadien de chirurgie). 1981;24(6):567–71.
11. Jones LC, Hungerford DS. Osteonecrosis: etiology, diagnosis, and treatment. Curr Opin Rheumatol. 2004;16(4):443–9.
12. Jones LC, et al. Procoagulants and osteonecrosis. J Rheumatol. 2003;30(4):783–91.
13. Glueck CJ, et al. Thrombophilia, hypofibrinolysis, the eNOS T-786C polymorphism, and multifocal osteonecrosis. J Bone Joint Surg Am. 2008;90(10): 2220–9.
14. Glueck CJ, et al. Idiopathic osteonecrosis, hypofibrinolysis, high plasminogen activator inhibitor, high lipoprotein(a), and therapy with Stanozolol. Am J Hematol. 1995;48(4):213–20.
15. Glueck CJ, et al. Enoxaparin prevents progression of stages I and II osteonecrosis of the hip. Clin Orthop Relat Res. 2005;435:164–70.
16. Koo KH, et al. Endothelial nitric oxide synthase gene polymorphisms in patients with nontraumatic femoral head osteonecrosis. J Orthop Res. 2006;24(8):1722–8.
17. Liu YF, et al. Type II collagen gene variants and inherited osteonecrosis of the femoral head. N Engl J Med. 2005;352(22):2294–301.
18. Chao YC, et al. Investigation of alcohol metabolizing enzyme genes in Chinese alcoholics with avascular necrosis of hip joint, pancreatitis and cirrhosis of the liver. Alcohol Alcohol. 2003;38(5):431–6.
19. Kaneshiro Y, et al. Low hepatic cytochrome P450 3A activity is a risk for corticosteroid-induced osteonecrosis. Clin Pharmacol Ther. 2006;80(4):396–402.
20. Tokuhara Y, et al. Low levels of steroid-metabolizing hepatic enzyme (cytochrome P450 3A) activity may elevate responsiveness to steroids and may increase risk of steroid-induced osteonecrosis even with low glucocorticoid dose. J Orthop Sci. 2009;14(6): 794–800.
21. Kerboul M, et al. The conservative surgical treatment of idiopathic aseptic necrosis of the femoral head. J Bone Joint Surg Br. 1974;56(2):291–6.
22. Sakamoto M, et al. Osteonecrosis of the femoral head: a prospective study with MRI. J Bone Joint Surg Br. 1997;79(2):213–9.
23. Steinberg ME, Hayken GD, Steinberg DR. A quantitative system for staging avascular necrosis. J Bone Joint Surg Br. 1995;77(1):34–41.
24. Lafforgue P, et al. Early-stage avascular necrosis of the femoral head: MR imaging for prognosis in 31 cases with at least 2 years of follow-up. Radiology. 1993;187(1):199–204.
25. Beltran J, et al. Core decompression for avascular necrosis of the femoral head: correlation between long-term results and preoperative MR staging. Radiology. 1990;175(2):533–6.
26. Ha YC, et al. Prediction of collapse in femoral head osteonecrosis: a modified Kerboul method with use of magnetic resonance images. J Bone Joint Surg Am. 2006;88 Suppl 3:35–40.
27. Gardeniers J. A new international classification of osteonecrosis of the ARCO Committee on terminology and classification. J Jpn Orthop Assoc. 1992;66: 18–20.
28. Fisher DE. The role of fat embolism in the etiology of corticosteroid-induced avascular necrosis: clinical and experimental results. Clin Orthop Relat Res. 1978;130:68–80.
29. Pritchett JW. Statin therapy decreases the risk of osteonecrosis in patients receiving steroids. Clinical Orthop Relat Res. 2001;386:173–8.
30. Yang J, et al. An experimental study on treatment of steroid-associated femoral head necrosis with simvastatin and BMSCs transplantation. Zhongguo xiu fu chong jian wai ke za zhi. 2008;22(3):290–4.
31. Bowers JR, et al. Drug therapy increases bone density in osteonecrosis of the femoral head in canines. J Surg Orthop Adv. 2004;13(4):210–6.
32. Agarwala S, et al. Alendronate in the treatment of avascular necrosis of the hip. Rheumatology. 2002;41(3):346–7.
33. Agarwala S, et al. Efficacy of alendronate, a bisphosphonate, in the treatment of AVN of the hip. A prospective open-label study. Rheumatology. 2005;44(3):352–9.
34. Lai KA, et al. The use of alendronate to prevent early collapse of the femoral head in patients with nontraumatic osteonecrosis. A randomized clinical study. J Bone Joint Surg Am. 2005;87(10):2155–9.
35. Chen CH, et al. Alendronate in the prevention of collapse of the femoral head in nontraumatic osteonecrosis: a two-year multicenter, prospective,

randomized, double-blind, placebo-controlled study. Arthritis Rheum. 2012;64(5):1572–8.

36. Ficat P, et al. Therapeutic results of drill biopsy in primary osteonecrosis of the femoral head (100 cases). Revue du rhumatisme et des maladies osteo-articulaires. 1971;38(4):269–76.
37. Hungerford DS. Pathogenesis of ischemic necrosis of the femoral head. Instr Course Lect. 1983;32:252–60.
38. Hernigou P, Beaujean F. Treatment of osteonecrosis with autologous bone marrow grafting. Clin Orthop Relat Res. 2002;405:14–23.
39. Gangji V, et al. Treatment of osteonecrosis of the femoral head with implantation of autologous bone-marrow cells. A pilot study. J Bone Joint Surg Am. 2004;86-A(6): 1153–60.
40. Gangji V, Hauzeur JP. Treating osteonecrosis with autologous bone marrow cells. Skeletal Radiol. 2010; 39(3):209–11.
41. Gangji V, De Maertelaer V, Hauzeur JP. Autologous bone marrow cell implantation in the treatment of non-traumatic osteonecrosis of the femoral head: five year follow-up of a prospective controlled study. Bone. 2011;49(5):1005–9.
42. Sen RK, et al. Early results of core decompression and autologous bone marrow mononuclear cells instillation in femoral head osteonecrosis: a randomized control study. J Arthroplasty. 2012;27(5):679–86.
43. Zhao D, et al. Treatment of early stage osteonecrosis of the femoral head with autologous implantation of bone marrow-derived and cultured mesenchymal stem cells. Bone. 2012;50(1):325–30.
44. Martin JR, Houdek MT, Sierra RJ. Use of concentrated bone marrow aspirate and platelet rich plasma during minimally invasive decompression of the femoral head in the treatment of osteonecrosis. Croat Med J. 2013;54(3):219–24.
45. Aldridge 3rd JM, et al. Free vascularized fibular grafting for the treatment of postcollapse osteonecrosis of the femoral head. Surgical technique. J Bone Joint Surg Am. 2004;86-A Suppl 1:87–101.
46. Korompilias AV, et al. Vascularised fibular graft in the management of femoral head osteonecrosis: twenty years later. J Bone Joint Surg Br. 2009;91(3):287–93.
47. Yoo MC, et al. Long-term followup of vascularized fibular grafting for femoral head necrosis. Clin Orthop Relat Res. 2008;466(5):1133–40.
48. Sugioka Y. Transtrochanteric rotational osteotomy in the treatment of idiopathic and steroid-induced femoral head necrosis, Perthes' disease, slipped capital femoral epiphysis, and osteoarthritis of the hip. Indications and results. Clin Orthop Relat Res. 1984;184: 12–23.
49. Shannon BD, Trousdale RT. Femoral osteotomies for avascular necrosis of the femoral head. Clin Orthop Relat Res. 2004;418:34–40.
50. Scher MA, Jakim I. Intertrochanteric osteotomy and autogenous bone-grafting for avascular necrosis of the femoral head. J Bone Joint Surg Am. 1993;75(8): 1119–33.
51. Mont MA, et al. Corrective osteotomy for osteonecrosis of the femoral head. J Bone Joint Surg Am. 1996;78(7):1032–8.
52. Lachiewicz PF, Desman SM. The bipolar endoprosthesis in avascular necrosis of the femoral head. J Arthroplasty. 1988;3(2):131–8.
53. Adili A, Trousdale RT. Femoral head resurfacing for the treatment of osteonecrosis in the young patient. Clin Orthop Relat Res. 2003;417:93–101.
54. Ritter MA, Meding JB. A comparison of osteonecrosis and osteoarthritis patients following total hip arthroplasty. A long-term follow-up study. Clin Orthop Relat Res. 1986;206:139–46.
55. Garino JP, Steinberg ME. Total hip arthroplasty in patients with avascular necrosis of the femoral head: a 2- to 10-year follow-up. Clin Orthop Relat Res. 1997;334:108–15.
56. D'Antonio JA, et al. Hydroxyapatite coated implants. Total hip arthroplasty in the young patient and patients with avascular necrosis. Clin Orthop Relat Res. 1997;344:124–38.
57. Berry DJ, et al. The cumulative long-term risk of dislocation after primary Charnley total hip arthroplasty. J Bone Joint Surg Am. 2004;86-A(1):9–14.
58. Patel D, Parvizi J, Sharkey PF. Alternative bearing surface options for revision total hip arthroplasty. Instr Course Lect. 2011;60:257–67.

84 Surgical Technique: Arthroscopic Core Decompression

Rachel M. Frank, Anil Gupta, Michael D. Hellman, and Shane J. Nho

Contents

R.M. Frank (✉) • A. Gupta • M.D. Hellman
Department of Orthopaedic Surgery, Rush University Medical Center, Chicago, IL, USA
e-mail: rmfrank3@gmail.com; anilgupta07@hotmail.com

S.J. Nho
Department of Orthopaedic Surgery, Rush University Medical Center, Chicago, IL, USA

Midwest Orthopaedics at Rush, Hip Preservation Center, Rush University Medical Center, Chicago, IL, USA
e-mail: shane.nho@rushortho.com

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_93

Abstract

Osteonecrosis of the femoral head remains a challenging problem in the young patient population. The pathophysiology of this condition is poorly understood, and often, the underlying etiology is unknown. Several risk factors have been identified, including steroid use, alcohol use, trauma, and a history of hypercoagulable disorders. The management of osteonecrosis is dependent on the stage of the disease, which is typically divided into pre-collapse and post-collapse stages. The management of pre-collapse osteonecrosis of the femoral head is controversial, and both medical and surgical options have been described. The most commonly used surgical options include core decompression as well as bone grafting (vascularized or non-vascularized). Core decompression is a technique that theoretically decreases intraosseous pressure of the femoral head, resulting in a local vascularized healing response, and recently, arthroscopic techniques have been reported.

Introduction

Etiology

Osteonecrosis, also referred to as avascular necrosis, is a condition in which subchondral bone loses its viability, resulting in sclerosis, weakening of the surrounding bone, subchondral collapse, articular incongruity, and, ultimately, resultant osteoarthritis [1, 2]. While a majority of cases are idiopathic without a known cause, several risk factors for the development of osteonecrosis of the femoral head have been identified. These risk factors can be broken down into two categories, including traumatic and atraumatic. Traumatic etiologies include fracture as well as a history of hip subluxation or dislocation. Atraumatic etiologies are more common [3] and include corticosteroid use, alcohol use, sickle cell disease, and hypercoagulable conditions [4], including thrombophilia, protein S deficiency, and protein C deficiency. Other, less common causes are listed in Table 1. In the pediatric patient population, diagnoses including Legg–Calve–Perthes (LCP) disease [8] and slipped capital femoral epiphysis (SCFE) [9, 10] are more common .

Table 1 Less common causes of osteonecrosis of the femoral head

Bone marrow replacing disease processes, such as Gaucher's disease [5]
Dysbaric disorders, such as decompression sickness (Caisson's disease, aka the "bends")
Systemic lupus erythematosus [6]
Inflammatory bowel disease
History of undergoing organ or tissue transplant [7]
Chronic renal failure
Pancreatitis
Pregnancy

Pathophysiology

The pathophysiology is poorly understood, making it difficult to prevent this condition. The pathogenesis can be multifactorial, with a combination of metabolic factors and local/host factors affecting blood supply to the femoral head. The disease process is thought to involve an interruption to the vascular supply of the femoral head, which causes adjacent hyperemia, demineralization of the bone, and trabecular thinning, leading to subchondral collapse. Specifically, coagulation of intraosseous microcirculation occurs, followed by resultant venous thrombosis and retrograde arterial occlusion, which increases intraosseous pressure, leading to decreased blood supply to the femoral head. This ultimately leads to death of osteocytes and osteoprogenitor cells, resulting in subchondral fracture and collapse. In cases with underlying traumatic etiologies, typically injury to the medial femoral circumflex artery is responsible. As such, osteonecrosis is most common in cases of displaced femoral head fractures, followed by basicervical femoral neck fractures; the highest risk being combined intrafoveal femoral head and displaced femoral neck fractures (Pipkin III). Hip dislocations are also associated with the development of osteonecrosis; however, if the joint is reduced within 6–8 h of injury, the rate of osteonecrosis is decreased [11, 12].

Clinical Presentation

History and Physical Examination

Patients with femoral head osteonecrosis can be asymptomatic and the diagnosis can be made from imaging performed for another indication. The vast majority of symptomatic patients complain of progressive pain localized to the groin and general hip region. While pain localized to the groin is most specific for intra-articular pathology, pain in the thigh and buttock is also commonly seen. These symptoms can be exacerbated with weight bearing and with combined flexion and internal rotation. Patients should be asked about prior history of trauma, surgery, corticosteroid use, ethanol use, and personal or family history of blood disorders. Sudden inability to bear weight associated with fevers, chills, weight loss, and/or night sweats warrant further workup for infectious, inflammatory, and or malignant underlying etiologies. Physical examination findings can often be nonspecific. Patients may walk with a mild antalgic gait favoring the affected side, but otherwise the majority of examination findings are normal during early stages of the condition. Any neurovascular deficits or isolated muscular weakness should be further worked up for other underlying etiologies.

Imaging Studies

All patients with suspected osteonecrosis of the femoral head should undergo a complete radiographic workup including an anterior–posterior (AP) view of the pelvis, an AP view of the affected hip, and a cross-table or frog-leg lateral view of the affected hip. Often, it is the lateral view that best demonstrates subchondral collapse and/or collapse. The most commonly utilized classification systems for osteonecrosis are based on radiographic findings and historically include the Ficat classification [13], and more recently the Steinberg classification (Table 2) [14]. Of note, patients who are diagnosed with AVN in another area of the body (i.e., humeral head) should always get hip radiographs as there is an increased risk of having concomitant (but asymptomatic) femoral head AVN [15]. Radiographs can remain completely normal for months after symptoms begin (Fig. 1). Early radiographic findings of osteonecrosis will show mild density changes within the femoral heads due to micro-infarcts with corresponding calcification. In both of these systems, stage III will show a crescent sign radiographically, indicating subchondral collapse (Fig. 2). Typically, advanced imaging with magnetic resonance imaging (MRI) is also employed if there is a high index of suspicion and/or the patient is at risk (i.e., alcoholic) despite normal radiographs. This modality can reveal changes due to osteonecrosis at earlier stages than plain radiographs are capable of showing. MRI has been shown to have excellent specificity and sensitivity with regard

Table 2 Steinberg classification system

0	Normal XR, normal MR
I	Normal XR, abnormal MR (and/or bone scan)
II	Cystic/sclerosis, abnormal MR (and/or bone scan)
III	Crescent sign (subchondral collapse), abnormal MR (and/or bone scan)
IV	Femoral head flattening, abnormal MR (and/or bone scan)
V	Narrowing of joint, abnormal MR (and/or bone scan)
VI	Advanced degenerative changes, abnormal MR (and/or bone scan)

Fig. 1 AP radiograph of the pelvis demonstrating well-preserved joint space without evidence of collapse, cam, pincer, or dysplasia of the bilateral hips

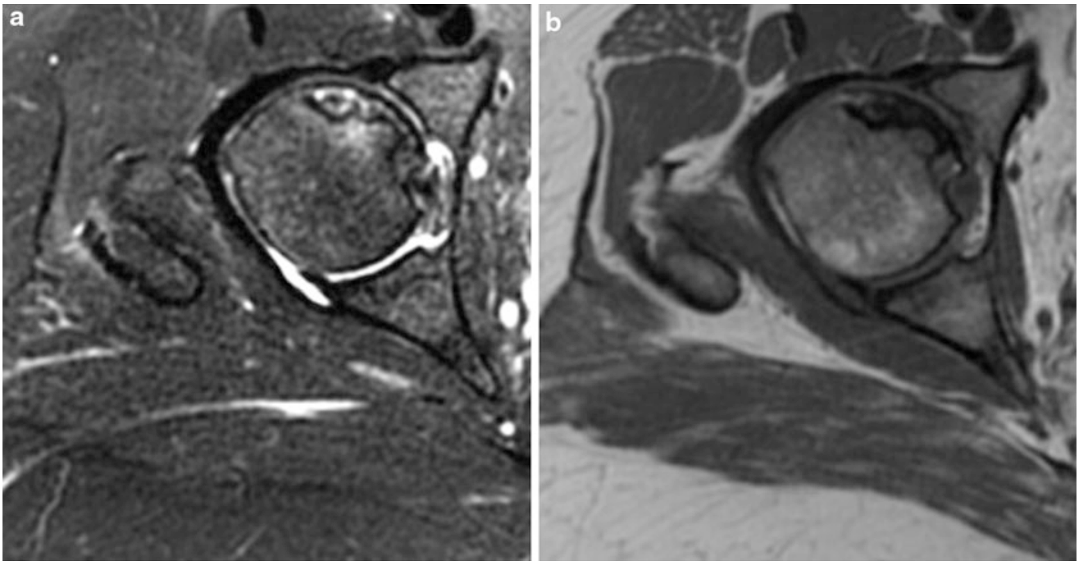


Fig. 2 Axial cuts from MRI demonstrating osteonecrosis (*ON*) of the right anterior superior femoral head (**a**: T2-weighted image, **b**: T1-weighted image)

to osteonecrosis and will typically appear bright on T2-weighted images with corresponding dark findings on T1-weighted images. Bone scan is also another modality that can be used to help diagnose and stage osteonecrosis of the femoral head, but is not as specific as MRI [16, 17].

Management Options

Based on symptoms and imaging findings, osteonecrosis of the femoral head is typically divided into early, or pre-collapse, versus late, or post-collapse, stages. Treatment options vary depending on the stage, with late stage presentations the most difficult to manage (from a non-arthroplasty perspective). Without any intervention, the natural history of osteonecrosis is that of ultimate disease progression, with most patients ultimately progressing to femoral head collapse and end-stage arthritis [18, 19]. Asymptomatic patients typically present in the pre-collapse stages and can be managed nonoperatively with close clinical follow-up. These patients must be watched closely, and the development of pain should prompt a repeat examination with repeat imaging studies.

Table 3 Surgical options for osteonecrosis of the femoral head

Core decompression
Rotational osteotomy
Vascularized graft transfer (free fibula)
Non-vascularized graft transfer
Total hip arthroplasty
Unipolar or bipolar hemiarthroplasty
Hip resurfacing

Patients who are symptomatic and in the pre-collapse stages will most often require intervention. While some medical therapies including marrow/stem cells [20–24] and bisphosphonate [25] use have been advocated, current results are inconsistent, and the majority of patients will undergo surgical intervention. Surgical options for these pre-collapse patients are outlined in Table 3. Non-arthroplasty options include core decompression, vascularized bone graft [26–30], non-vascularized bone graft, and osteotomies. Treatment options for post-collapse patients, however, are more limited, given the advanced stage of the disease. While most post-collapse osteonecrosis patients can be successfully managed with total hip arthroplasty [31], joint replacement may not be the best option in young, active

Table 4 Surgical steps

Establish anterolateral and anterior portals
Evaluate femoral head for subchondral collapse
Evaluate integrity of articular cartilage
Evaluate labrum and bony margin of acetabulum
Interportal or T-capsulotomy to address concomitant labral and/or femoral head–neck pathology
Maintain arthroscope in the joint to assist in avoiding subchondral penetration
Introduce drill and reamer under X-ray guidance, lesion typically in anterior superior femoral head
Introduce arthroscope into socket to confirm integrity of subchondral bone at lesion site
Implant ceramic putty to provide compressive strength
Perform dynamic arthroscopy of femoral head

patients, given the obvious concern for early polyethylene wear with subsequent osteolysis, component loosening, osteolysis, and the potential need for early revision.

Core decompression is a surgical technique in which the necrotic lesion is reamed or drilled to decrease local intraosseous pressure and stimulate a vascularized healing response. Multiple authors have demonstrated effective results with this treatment strategy for pre-collapse osteonecrosis, including arthroscopic-assisted decompression [32–34]. Given recent advances in techniques and instrumentation, hip arthroscopy is now the gold standard for the diagnosis of intra-articular hip pathology [35, 36]. Substantial improvements in hip-specific diagnostic modalities have improved the understanding of bony and soft tissue pathology in patients with intra-articular hip disorders. The association of concomitant soft tissue and/or bony pathology with osteonecrosis of the femoral head is currently unknown. Further, the ability of imaging studies to predict collapse and/or associated hip pathology in addition to osteonecrosis at the time of surgery is also unknown. Thus, arthroscopy at the time of decompression provides an accurate means to confirm the presence or absence of femoral head subchondral collapse, chondral delamination, and associated labral, capsular, and synovial pathology [37–39]. If present, such concomitant pathology can be addressed at the time of decompression and obviate the need for a subsequent surgery. Further, arthroscopy allows for verification and guidance during drilling and/or reaming to avoid penetration of the articular surface. The following section describes the surgical technique [40] for arthroscopic-assisted core decompression of the femoral head (Table 4).

Arthroscopic-Assisted Core Decompression: Surgical Technique

Patient Positioning and Surgical Setup

The patient is positioned supine on a traction table with a well-padded perineal post placed in the groin between the legs (Smith and Nephew hip traction system, Smith and Nephew, Andover, MA). Traction is applied with the leg in neutral extension, axially distraction, and adduction to provide a cantilever moment to the operative hip.

Landmarks

The greater trochanter and anterior superior iliac spine borders are palpable landmarks used to identify appropriate portal placement. These are marked on the skin.

Portals

Under fluoroscopic visualization, a standard anterolateral (AL) portal is created 1 cm proximal and 1 cm anterior to the AL aspect of the greater trochanter. The 70° arthroscope is inserted over a guidewire. While viewing from the AL portal, needle localization is used to establish an anterior portal, penetrating the capsule at the 2 o'clock position. The anterior portal is approximately 1 cm lateral to the line drawn vertically from the anterior superior iliac spine (ASIS) and a line drawn horizontally from the AL portal.

Diagnostic Arthroscopy

Diagnostic arthroscopy begins with the arthroscope in the AL portal (Dyonics Arthroscopy

Fig. 3 Femoral head articular surface, visualization through the anterolateral (*AL*) portal

System, Smith and Nephew, Inc., Andover, MA) (Fig. 3). The posterior-superior and posterior-inferior labrum, superior and lateral femoral head, and acetabular articular surface are evaluated for a labral tear or chondral lesion. Any unstable tissue or cartilage is probed through the anterior portal to determine instability. The arthroscope is then switched to the anterior portal and the remainder of the labrum, acetabulum, and femoral head are visualized. The anterior portal is best for visualizing the anterior superior femoral head, the most common site of osteonecrosis. It provides a direct "bird's-eye" view. The site, however, can also be visualized from the AL portal with the 70° arthroscope lens. Interportal capsulotomy is reserved for circumstances where labral or articular cartilage work must be performed such that increased arthroscope and instrumentation mobility is warranted.

Throughout each of the steps (guide pin placement, reaming, curettage, and putty placement), the arthroscope is maintained in the AL portal, focused on the articular side of the area of necrosis, to ensure that the subchondral bone and articular cartilage are not violated. The 70° arthroscope enables visualization of the articular side of the lesion so that combined fluoroscopic imaging can be performed to evaluate debridement and filling of the socket without the radiopaque arthroscope obscuring fluoroscopic visualization if maintained in the anterior portal.

Fluoroscopic Confirmation of the Lesion

Fluoroscopic assistance confirms the location of the osteonecrotic lesion. The articular surface is evaluated and probed for any depression, softening, and/or delamination. If softening is present, a probe is used to measure the size, stability, and depth of the lesion. This is typically performed through the anterior portal while visualizing through the AL portal. The arthroscope is then switched back to the AL portal, while any instrumentation in the anterior portal is removed to allow for adequate visualization of the femoral neck on AP and frog-leg lateral fluoroscopic views. A spinal needle is placed into the anterior portal to allow for outflow and the pressure of the inflow is reduced to 40 mmHg.

Core Decompression

Core decompression is then performed through a separate 3 cm incision distal and posterior to the AL portal. This incision is created with fluoroscopic guidance. Blunt dissection is subsequently carried down to the tensor fascia lata, which is incised longitudinally. The vastus lateralis muscle belly is elevated anteriorly to expose the proximal intertrochanteric portion of the femur.

Guide Pin Placement

The starting point of the guide pin is biased posterior to the midpoint of the proximal femur in the sagittal plane and proximal to the lesser trochanter. With arthroscopic visualization,

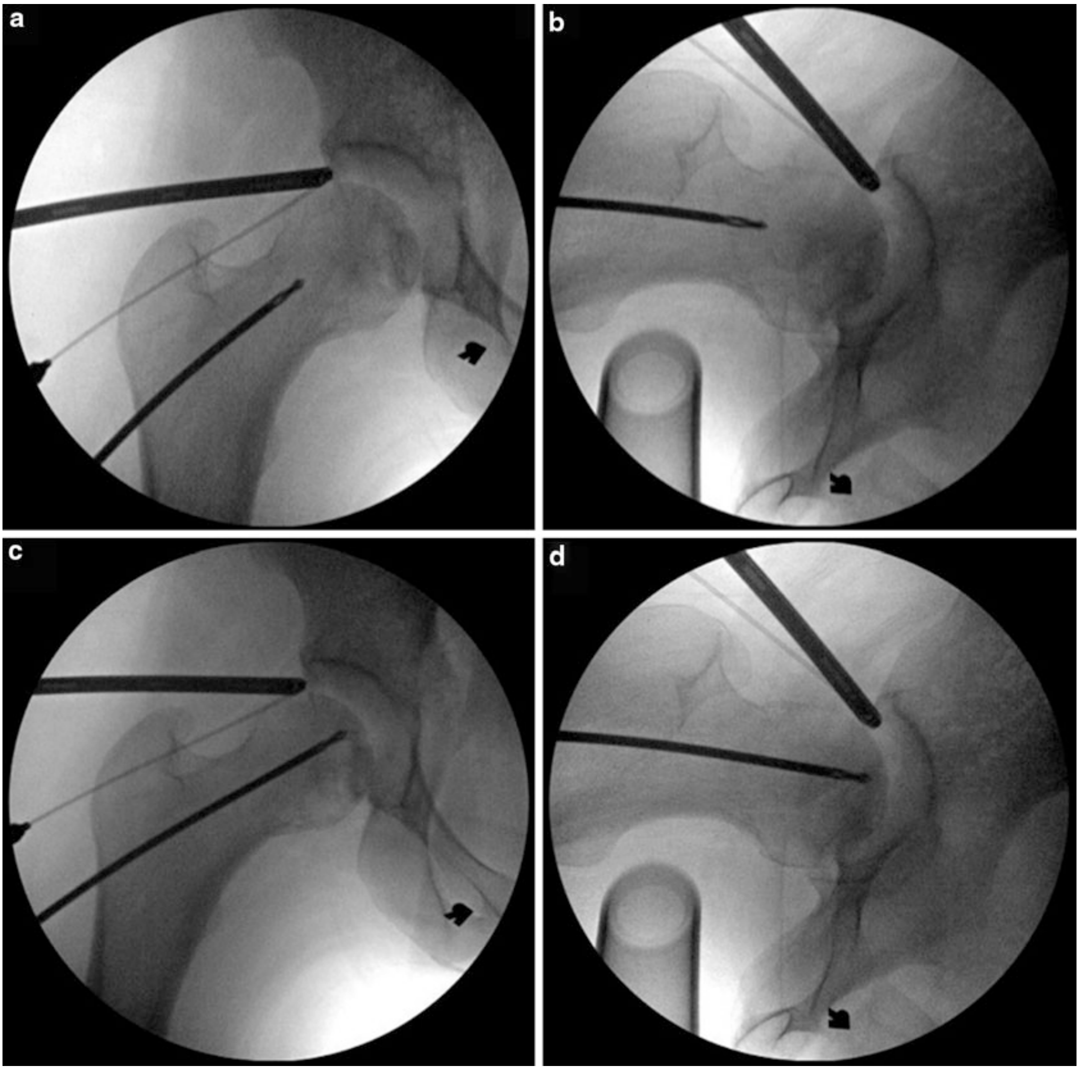


Fig. 4 Intraoperative fluoroscopic imaging demonstrating guide pin placement into the center of lesion (**a** and **b**, AP and lateral, **c** and **d**, AP and lateral after pin advancement)

a probe is positioned over the area of cartilage softening, and the guide pin is directed toward the area of osteonecrosis. The trajectory is posterolateral to anteromedial to penetrate the necrotic lesion. The guide pin advanced to 1–2 mm deep to the subchondral bone (Fig. 3a). Appropriate positioning in the femoral head is confirmed with anterior–posterior (AP) and cross-table lateral radiographs (Fig. 4).

Introduction of Reamer

A soft tissue guide is placed over the guide pin and a reamer is advanced to the same depth (Advanced Core Decompression System, Wright Medical Technology Inc., Arlington, TN). Careful attention is paid not to allow the guide pin or reamer to advance beyond the subchondral bone. Confirmation that the reamer has penetrated the necrotic

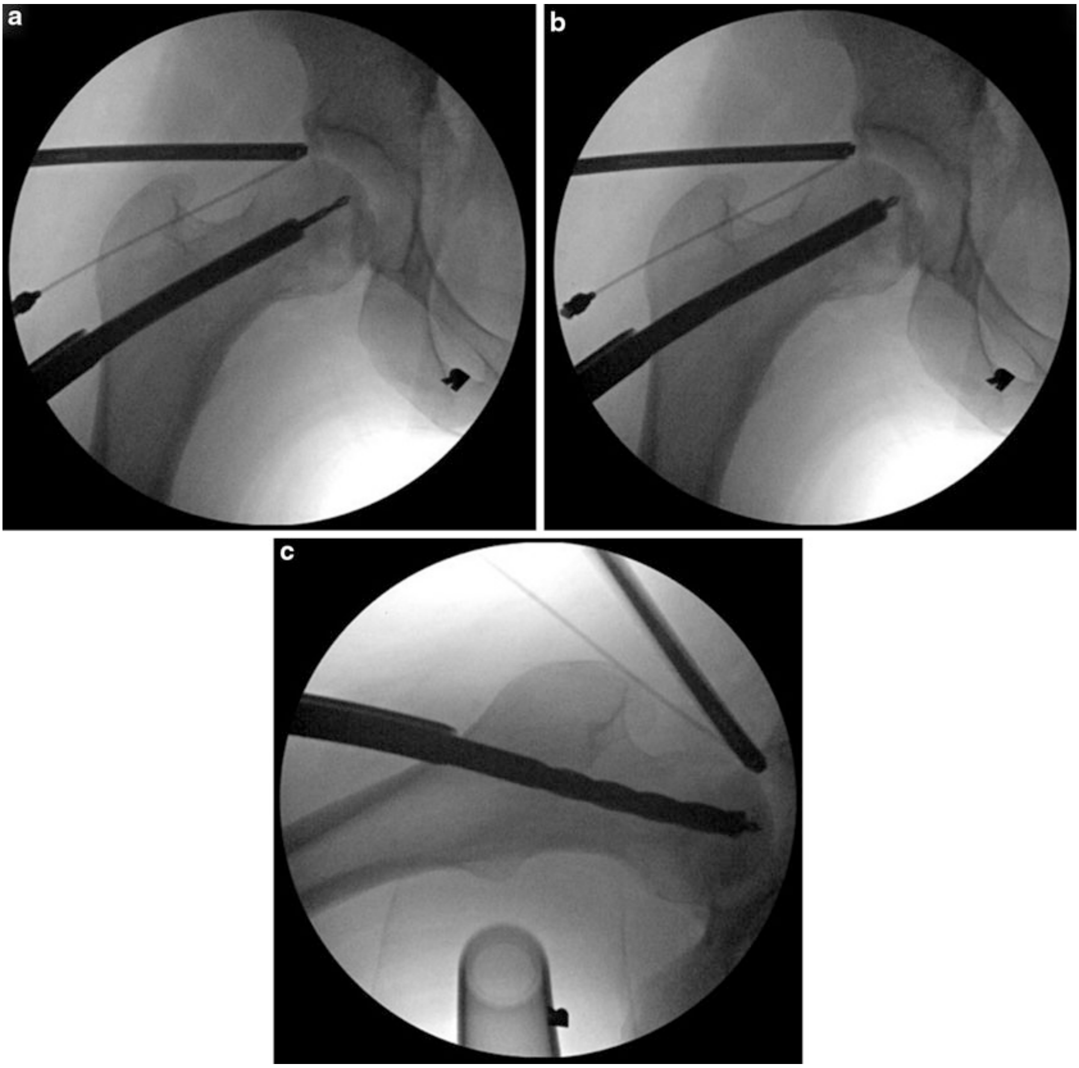


Fig. 5 Intraoperative fluoroscopic imaging demonstrating introduction and advancement of reamer over pin

lesion is obtained fluoroscopically as well as by tactile sensation – the surgeon will feel resistance while reaming the sclerotic bone (Fig. 5).

Introduction of 30° Curved Curette

The pin/reamer is removed and a 30° curved curette is advanced up the socket and the residual necrotic bone is removed (Fig. 6).

Insertion of Syringe into Socket

A syringe is inserted into the socket and combination calcium phosphate (CaPO4)/calcium sulfate (CaSO4) synthetic putty (ProDense®, Wright Medical Technology Inc., Arlington, TN) is injected into the socket [41]. It is injected in a retrograde fashion, allowing the pressure of the putty to guide the syringe (Fig. 7). Intra-articular arthroscopic visualization confirms no

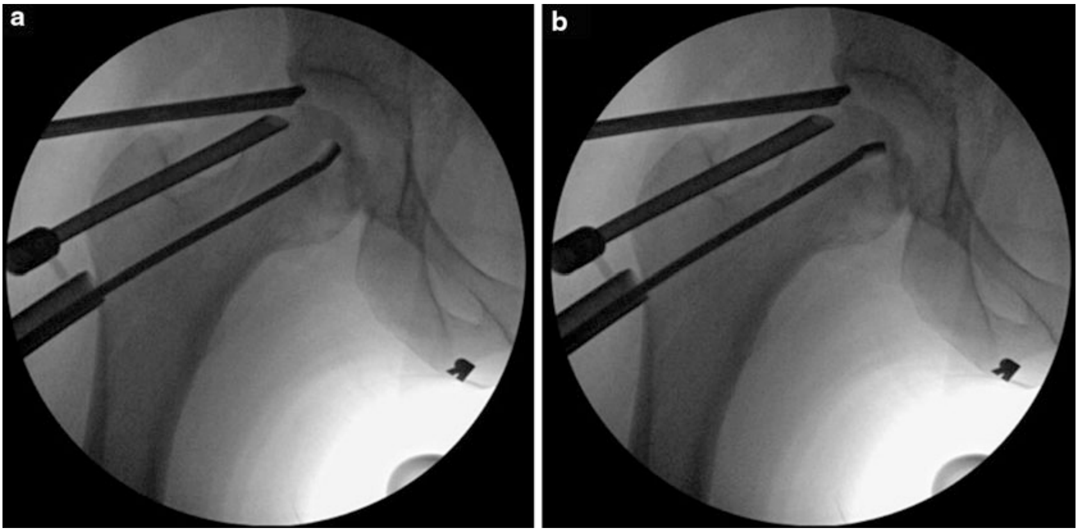


Fig. 6 Intraoperative fluoroscopic imaging demonstrating curettage of lesion

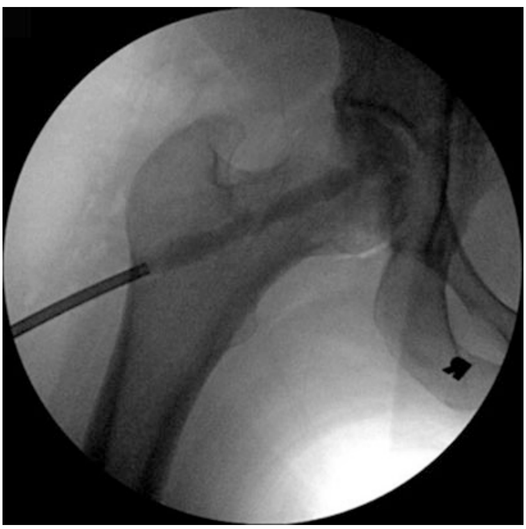


Fig. 7 Intraoperative fluoroscopic imaging demonstrating filling of socket with calcium sulfate and calcium phosphate cement mixture

intra-articular penetration of the putty. Final AP and lateral fluoroscopic views are obtained verifying fill of the socket (Fig. 8). Once the putty is cured (as evidenced on the side table), instruments are removed from the hip, and traction is released. Dynamic fluoroscopy is performed verifying joint reduction.

Closure

The tensor fascia lata is closed with 0 absorbable braided suture, the dermis with 2-0 braided absorbable suture, and the skin (along with portals) with 3-0 monofilament suture.

Postoperative Course

Postoperatively the patient is maintained touchdown weight bearing with crutches for 6 weeks. Immediate passive range of motion is begun the evening of surgery with continuous passive motion. At 6 weeks the patient is progressed to weight bearing as tolerated. Return to athletic activity is delayed for a minimum of 3 months.

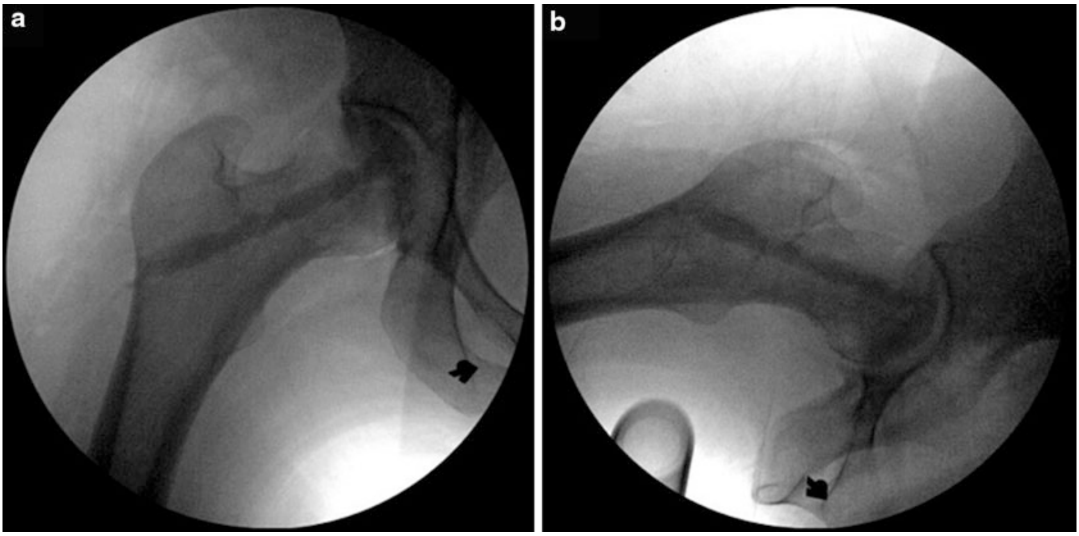


Fig. 8 Intraoperative fluoroscopic imaging verifying fill of the socket

Summary

Core decompression is a technique that theoretically decreases intraosseous pressure of the femoral head, resulting in a local vascularized healing response. Arthroscopic-assisted core decompression of the femoral head is an effective treatment option for pre-collapse osteonecrosis of the femoral head. Arthroscopy at the time of decompression provides the added advantage of intra-articular visualization to confirm the diagnosis, allow for treatment of associated bony and soft tissue pathology, and avoid the risk of joint penetration in carefully selected patients.

References

1. Kaushik AP, Das A, Cui Q. Osteonecrosis of the femoral head: an update in year 2012. World J Orthop. 2012;3(5):49–57.
2. Lavernia CJ, Sierra RJ, Grieco FR. Osteonecrosis of the femoral head. J Am Acad Orthop Surg. 1999;7 (4):250–61.
3. Mirzai R, Chang C, Greenspan A, Gershwin ME. Avascular necrosis. Compr Ther. 1998;24(5):251–5.
4. Rueda JC, Duque MA, Mantilla RD, Iglesias-Gamarra A. Osteonecrosis and antiphospholipid syndrome. J Clin Rheumatol Pract Rep Rheum Musculoskelet Dis. 2009;15(3):130–2.
5. Khan A, Hangartner T, Weinreb NJ, Taylor JS, Mistry PK. Risk factors for fractures and avascular osteonecrosis in type 1 Gaucher disease: a study from the International Collaborative Gaucher Group (ICGG) Gaucher Registry. J Bone Miner Res Off J Am Soc Bone Miner Res. 2012;27(8):1839–48.
6. Sayarlioglu M, Yuzbasioglu N, Inanc M, et al. Risk factors for avascular bone necrosis in patients with systemic lupus erythematosus. Rheumatol Int. 2012;32(1):177–82.
7. McClune B, Majhail NS, Flowers ME. Bone loss and avascular necrosis of bone after hematopoietic cell transplantation. Semin Hematol. 2012;49 (1):59–65.
8. Nguyen NA, Klein G, Dogbey G, McCourt JB, Mehlman CT. Operative versus nonoperative treatments for Legg-Calve-Perthes disease: a meta-analysis. J Pediatr Orthop. 2012;32(7):697–705.
9. Loder RT. What is the cause of avascular necrosis in unstable slipped capital femoral epiphysis and what can be done to lower the rate? J Pediatr Orthop. 2013;33 Suppl 1:S88–91.
10. Jarrett DY, Matheney T, Kleinman PK. Imaging SCFE: diagnosis, treatment and complications. Pediatr Radiol. 2013;43 Suppl 1:S71–82.
11. Hougaard K, Thomsen PB. Coxarthrosis following traumatic posterior dislocation of the hip. J Bone Jt Surg Am Vol. 1987;69(5):679–83.
12. Nerubay J. Traumatic anterior dislocation of hip joint with vascular damage. Clin Orthop Relat Res. 1976;116:129–32.

13. Beaule PE, Amstutz HC. Management of Ficat stage III and IV osteonecrosis of the hip. J Am Acad Orthop Surg. 2004;12(2):96–105.
14. Steinberg ME, Hayken GD, Steinberg DR. A quantitative system for staging avascular necrosis. J Bone Jt Surg Br Vol. 1995;77(1):34–41.
15. LaPorte DM, Mont MA, Mohan V, Jones LC, Hungerford DS. Multifocal osteonecrosis. J Rheumatol. 1998;25(10):1968–74.
16. Sedonja I, Jevtic V, Milcinski M. Bone scintigraphy as a prognostic indicator for bone collapse in the early phases of femoral head osteonecrosis. Ann Nucl Med. 2007;21(3):167–73.
17. Malizos KN, Karantanas AH, Varitimidis SE, Dailiana ZH, Bargiotas K, Maris T. Osteonecrosis of the femoral head: etiology, imaging and treatment. Eur J Radiol. 2007;63(1):16–28.
18. Mont MA, Zywiel MG, Marker DR, McGrath MS, Delanois RE. The natural history of untreated asymptomatic osteonecrosis of the femoral head: a systematic literature review. J Bone Jt Surg Am Vol. 2010;92 (12):2165–70.
19. Lieberman JR, Engstrom SM, Meneghini RM, SooHoo NF. Which factors influence preservation of the osteonecrotic femoral head? Clin Orthop Relat Res. 2012;470(2):525–34.
20. Kang JS, Moon KH, Kim BS, et al. Clinical results of auto-iliac cancellous bone grafts combined with implantation of autologous bone marrow cells for osteonecrosis of the femoral head: a minimum 5-year follow-up. Yonsei Med J. 2013;54(2):510–5.
21. Rackwitz L, Eden L, Reppenhagen S, et al. Stem cell- and growth factor-based regenerative therapies for avascular necrosis of the femoral head. Stem Cell Res Ther. 2012;3(1):7.
22. Gangji V, De Maertelaer V, Hauzeur JP. Autologous bone marrow cell implantation in the treatment of non-traumatic osteonecrosis of the femoral head: five year follow-up of a prospective controlled study. Bone. 2011;49(5):1005–9.
23. Cui Q, Botchwey EA. Emerging ideas: treatment of precollapse osteonecrosis using stem cells and growth factors. Clin Orthop Relat Res. 2011;469(9):2665–9.
24. Zhao D, Cui D, Wang B, et al. Treatment of early stage osteonecrosis of the femoral head with autologous implantation of bone marrow-derived and cultured mesenchymal stem cells. Bone. 2012;50(1):325–30.
25. Agarwala S, Shah SB. Ten-year follow-up of avascular necrosis of femoral head treated with alendronate for 3 years. J Arthroplast. 2011;26(7):1128–34.
26. Fang T, Zhang EW, Sailes FC, McGuire RA, Lineaweaver WC, Zhang F. Vascularized fibular grafts in patients with avascular necrosis of femoral head: a systematic review and meta-analysis. Arch Orthop Trauma Surg. 2013;133(1):1–10.
27. Garrigues GE, Aldridge 3rd JM, Friend JK, Urbaniak JR. Free vascularized fibular grafting for treatment of osteonecrosis of the femoral head secondary to hip dislocation. Microsurgery. 2009;29(5):342–5.
28. Zhang C, Zeng B, Xu Z, et al. Treatment of femoral head necrosis with free vascularized fibula grafting: a preliminary report. Microsurgery. 2005;25(4):305–9.
29. Le Nen D, Genestet M, Dubrana F, Stindel E, Lacroix J, Lefevre C. Vascularized fibular transplant for avascular necrosis of the femoral head: 16 cases. Rev Chir Orthop Reparatrice Appar Mot. 2004;90(8):722–31.
30. Gaskill TR, Urbaniak JR, Aldridge 3rd JM. Free vascularized fibular transfer for femoral head osteonecrosis: donor and graft site morbidity. J Bone Jt Surg Am Vol. 2009;91(8):1861–7.
31. Johnson AJ, Mont MA, Tsao AK, Jones LC. Treatment of femoral head osteonecrosis in the United States: 16-year analysis of the Nationwide Inpatient Sample. Clin Orthop Relat Res. 2014;472(2):617–23.
32. Guadilla J, Fiz N, Andia I, Sanchez M. Arthroscopic management and platelet-rich plasma therapy for avascular necrosis of the hip. Knee Surg Sports Traumatol Arthrosc Off J ESSKA. 2012;20(2):393–8.
33. Dines JS, Strauss EJ, Fealy S, Craig EV. Arthroscopic-assisted core decompression of the humeral head. Arthrosc J Arthrosc Relat Surg Off Publ Arthrosc Assoc N Am Int Arthrosc Assoc. 2007;23(1):103. e101–4.
34. Zhuo N, Wan Y, Lu X, Zhang Z, Tan M, Chen G. Comprehensive management of early stage avascular necrosis of femoral head by arthroscopic minimally invasive surgery. Zhongguo xiu fu chong jian wai ke za zhi = Zhongguo xiufu chongjian waike zazhi = Chin J Repar Reconstr Surg. 2012;26(9):1041–4.
35. Kelly BT, Williams 3rd RJ, Philippon MJ. Hip arthroscopy: current indications, treatment options, and management issues. Am J Sports Med. 2003;31(6):1020–37.
36. Clarke MT, Arora A, Villar RN. Hip arthroscopy: complications in 1054 cases. Clin Orthop Relat Res. 2003;406:84–8.
37. McCarthy J, Puri L, Barsoum W, Lee JA, Laker M, Cooke P. Articular cartilage changes in avascular necrosis: an arthroscopic evaluation. Clin Orthop Relat Res. 2003;406:64–70.
38. Ruch DS, Sekiya J, Dickson Schaefer W, Koman LA, Pope TL, Poehling GG. The role of hip arthroscopy in the evaluation of avascular necrosis. Orthopedics. 2001;24(4):339–43.
39. Sekiya JK, Ruch DS, Hunter DM, et al. Hip arthroscopy in staging avascular necrosis of the femoral head. J South Orthop Assoc. 2000;9(4):254–61.
40. Gupta AK, Frank RM, Harris JD, McCormick F, Mather RC, Nho SJ. Arthroscopic-assisted core decompression for osteonecrosis of the femoral head. Arthrosc Tech. 2013;3(1):e7–e11.
41. Civinini R, De Biase P, Carulli C, et al. The use of an injectable calcium sulphate/calcium phosphate bioceramic in the treatment of osteonecrosis of the femoral head. Int Orthop. 2012;36(8):1583–8.

Surgical Technique: Free Vascularized Fibula Graft for Avascular Necrosis

85

Richard C. Mather III, Andrew E. Federer, and David S. Ruch

Contents

R.C. Mather III (✉)
Department of Orthopaedic Surgery, Duke University School of Medicine, Durham, NC, USA
e-mail: chad.mather@gmail.com

A.E. Federer
Midwest Orthopaedics at Rush, Hip Preservation Center, Rush University Medical Center, Chicago, IL, USA
e-mail: afederer34@gmail.com

D.S. Ruch
Duke University Health System, Durham, NC, USA
e-mail: d.ruch@duke.edu

Abstract

Avascular necrosis (AVN) or osteonecrosis of the femoral head (ONFH) is a common cause of hip pain and disability in a young patient demographic with an average age of mid-30s. If left untreated, approximately 80 % of cases of ONFH will result in femoral head collapse and hip degeneration necessitating total hip arthroplasty (THA) in a mean of 2 years. The relatively poor outcomes and requisite lifestyle changes associated with THA in this patient population have challenged surgeons to seek alternative treatment options. Vascularized bone grafts have been shown to have better radiographical and clinical results compared to nonvascularized bone grafts. Of these options, free vascularized fibular graft (FVFG) is the treatment of choice for its structural and vascular support within the femoral head. Improved technique has led to complication rates, including donor-site morbidity, statistically similar to that of nonvascular fibular grafts. When used in young patients with precollapse ONFH, FVFG can greatly improve patient outcome, with lower rates of conversion to THA than other treatment modalities.

Introduction

Avascular necrosis (AVN) or osteonecrosis (ON) of the femoral head is a common cause of hip pain and disability. As many as 18 % of total hip

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_94

arthroplasties (THA) are performed for osteonecrosis and this remains an excellent treatment option for older patients [1, 2]. However, the majority of patients with osteonecrosis of the femoral head (ONFH) are young, with an average age of mid-30s [3, 4]. If left untreated, approximately 80 % of cases of ONFH will result in femoral head collapse and hip degeneration necessitating THA in a mean of 2 years [3, 5, 6]. The relatively poor outcomes and requisite lifestyle changes associated with THA in this patient population have challenged surgeons to seek alternative treatment options.

Presentation and Imaging

Early stages of the disease may present without pain, but ultimately the physical exam will show painful and limited range of motion that will eventually include restricted passive range of motion [3]. Plain radiographs are necessary for the diagnosis of ONFH; however, findings are often limited or absent in the early stages of the disease. Plain radiographs only pick up bone density changes associated with remodeling or disuse and therefore may only detect changes months or even years after the disease starts [7]. The Ficat-Arlet (FA) staging system is the most common and stages hips based on the four parameters of collapse, size, femoral head depression, and acetabular involvement. In cases of suspected ONFH without signs on plain radiographs, magnetic resonance imaging (MRI) should be considered. MRI is the most sensitive imaging study for the diagnosis of ONFH, especially for asymptomatic or early-stage cases [7–9].

Management

For young patients with symptomatic disease, there appears to be very little benefit to nonoperative management. Evidence from a meta-analysis including over 800 hips shows that 76 % of hips treated solely with restrictive weight-bearing required an arthroplasty or salvage procedure [10]. The results from this and similar studies show universal agreement that restrictive weight-bearing as sole treatment is inappropriate [11]. Operative management is largely determined by the stage of the lesion, with femoral head collapse being the most important factor in management. Larger and more advanced lesions become progressively more difficult to treat. For small, precollapse lesions, core decompression has been widely used. First coined by Hungerford, Ficat and Arlet, core decompression is thought to relieve pain by lowering interosseus pressure and stimulating neovascularization of the femoral head and neck [12, 13]. This procedure is favored for its technical ease, short operative time, and allowance for any number of various subsequent surgical options. The efficacy of core decompression in more advanced ON lesions has not been demonstrated.

Larger and more advanced lesions have historically been treated by a variety of surgical options to attempt hip preservation in younger patients. Osteotomies have been used but concern remains over the potential for disruption of the blood supply to the femoral head and the difficulty in converting these hips to an arthroplasty as a salvage procedure. Conventional bone grafting has been reported either through a trapdoor [14] in the articular cartilage or through a cortical window in the femoral neck [15, 16]. Both of these procedures are performed with intracapsular dissection and risk the vascularity to the femoral head. In the late 1970s, several potential vascularized bone graft techniques were used in hopes of aiding the biology of bone healing within the femoral head. Vascularized bone grafting and its subtypes share the goal of lesion decompression via the removal of necrotic bone and the placement of bone graft with a vascular source to provide osteoinductive progenitor cells for healing. Local pedicled bone grafts, most commonly a muscle-pedicle-bone graft from either the quadratus femoris or the tensor fascia lata, have been reported with superior results as compared to conventional nonvascularized bone grafts [17–22]. This technique is dependent upon muscular metaplasia to be successful. This, combined with the lack of structural bone graft provided by these grafts, led some authors to consider other sources of vascularized bone graft.

More distant donor sites that provided structural support for ON lesions and did not depend upon muscular metaplasia became possible with the development of the operating microscope and improved microsurgical techniques for free vascularized bone grafts. In the late 1970s, Urbaniak [23], Brunelli and Brunelli [24], and Judet and Gilbert [25] began using the free vascularized fibular graft (FVFG) to treat ON of the femoral head. The fibula provides a large corticocancellous bone graft that can be placed from an extracapsular entry point. It has a reliable vascular pedicle and when performed correctly has a low donor-site morbidity. Free vascularized fibular grafts have been compared to nonvascularized fibular grafts and demonstrate superior results in larger and more advanced ON lesions [26]. Several studies have cited vascularized fibular bone grafts as being radiographically and clinically superior to nonvascularized bone grafts in terms of progression to the next stage of disease [6, 27]. Harris hip scores (HHS) improved in 70 % of hips using fibular vascularized and 36 % using nonvascularized grafts. The rate of survival for stage I and II precollapse lesions at 7 years was 86 % for FVFG and 30 % for nonvascularized fibular grafts (NVFG) [6, 27].

Often cited drawbacks from using vascularized bone grafts include increased surgical difficulty of vascular grafting as well as donor-site morbidity [11]. However, donor-site morbidity is debated, with previous research showing anywhere from nearly no morbidity to 10 % [15, 28]. A recent meta-analysis vascularized fibular grafts (VFG) was compared to other treatment methods including core decompression, NVFG, and vascularized iliac grafts and found that 122/740 (16 %) of VFGs while 104/244 (42.6 %) of other methods resulted in failure (i.e., THA) [29]. A weighted test of the complication rate between VFG and other treatment methods showed no statistically significant difference. However, there has yet to be a prospective randomized control trial comparing VFG to other methods.

As a whole, hips treated with free vascularized fibular grafts demonstrated less radiographic progression, less femoral head collapse, and improved HHS. The authors have used this technique successfully for the management of this challenging clinical problem in young patients.

The free vascularized fibular graft remains the transfer of choice for osteonecrosis of the femoral head. The FVFG provides a large stock of corticocancellous bone graft for biological and structural support with a reliable vascular pedicle in the peroneal artery and veins. The graft can be harvested with a skin paddle for flap surveillance; however, there is a lack of consensus regarding the necessity of this [30].

Patient Population

The ideal patient for free vascularized fibular grafting for ON of the femoral head is a young, active patient with a symptomatic precollapse lesion (FA Stage I and II). Age, activity level, comorbidities, etiology, stage of disease, and radiographic findings are all considered when determining the appropriate management of each patient with ON of the femoral head.

There is no age limit for treatment of ON with FVFG; however, with increasing age there is an increased likelihood that arthroplasty will provide a successful management option for the patient. In general, patients older than 50 years are best treated by total hip arthroplasty. Because arthroplasty is associated with activity restrictions, patients younger than 50 years of age are evaluated on an individual basis for activity level and lifestyle so that an appropriate treatment option can be selected. Younger patients with an active lifestyle are typically best managed with FVFG.

Comorbidities and etiology are also considered when evaluating a patient's candidacy for FVFG. Alcohol and corticosteroid use are two common associated factors in the development of ON of the femoral head. While some centers consider current corticosteroid use as a contraindication to FVFG, the authors routinely offer FVFG to these patients with noted success. Active alcohol abuse should be considered a relative contraindication as both the pathophysiology of alcohol abuse and the potential noncompliance of postoperative weight-bearing restrictions can compromise outcomes.

Fig. 1 The patient is positioned as distal on the table as allowable to facilitate two teams, one approaching and preparing the hip and one harvesting the fibula

The stage of the ON lesion is given great weight when choosing the appropriate treatment. Asymptomatic hips are not treated with FVFG as some of these early-stage lesions will remain asymptomatic. Knowledge of the progression of ON in silent hips is limited to the evaluation of patients with contralateral disease. When ON is diagnosed on one side, Hungerford and Zizic found that 67 % of patients will develop collapse of the femoral head on the opposite side [31]. In the authors' experience, only 6 % of patients without evidence of ON in the silent hip progressed to symptomatic disease, but 72 % of patients with asymptomatic lesions present progressed [32].

Patients with precollapse lesions with maintenance of a spherical femoral head have the most to gain by an attempt at biological preservation of the femoral head. ON lesions that demonstrate some mild collapse on radiographs are considered for FVFG in younger patients with good mobility of the hip and an active lifestyle. For these patients, the questionable long-term survival of an arthroplasty procedure makes the FVFG a more attractive option.

Surgical Approach and Technique

The patient is positioned in a lateral decubitus position on a pegboard with the lower leg on a padded Mayo stand. Because of the duration of the procedure, all bony prominences should be well padded and an axillary roll should be placed to avoid positioning complications. Before prepping and draping the patient, confirm that the patient is positioned as distal on the operating table as allowable. With the patient's foot at the distal end of the table, the surgeon harvesting the fibula will minimize reach and strain while operating from the foot of the bed (Fig. 1). Furthermore, confirm that the pegs placed for patient positioning are out of the anticipated radiographic field of the femoral head.

The hip is approached through a curvilinear incision over the lateral aspect of the affected hip. The incision is approximately 10–15 cm in length and is directed convex anterior. This incision is placed such that the lateral femoral circumflex vessels are centered proximal to distal. These donor vessels are located approximately 10 cm distal to the anterior superior iliac spine (Fig. 2). With respect to anteroposterior placement of the incision, it must be positioned such that the microvascular work (anterior) and the hip reaming (posterior) can be performed through the same incision. This requires surface identification of the anterior and posterior borders of the proximal femur and the vastus ridge. The anterior margin of the incision is placed approximately 2 cm anterior to the anterior border of the femur with one-third of the incision superior to the vastus ridge and two-thirds inferior. This incision can be easily

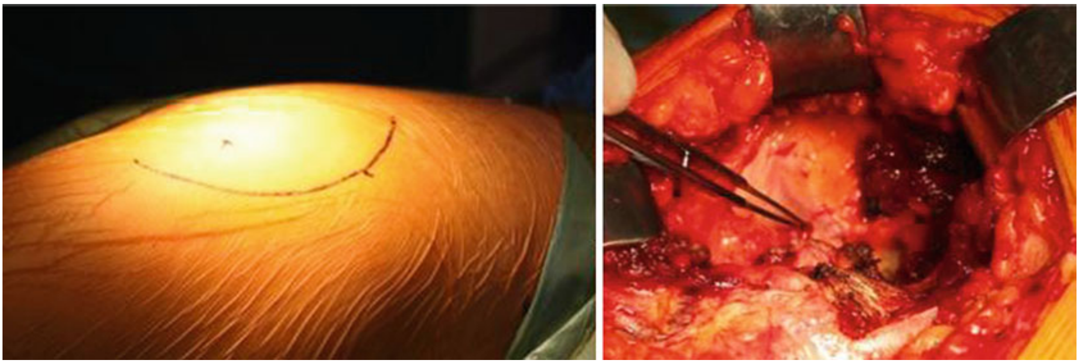


Fig. 2 The hip is approached through a 10–15 cm convex anterior curvilinear incision over the lateral aspect of the affected hip. The lateral femoral circumflex donor vessels are located approximately 10 cm distal to the anterior superior iliac spine

drawn by the operating surgeon placing his long finger on the vastus ridge with the tip of the finger 2 cm anterior to the anterior margin of the femur and the index finger superior. With the fingers maximally abducted, a line drawn connecting the fingertips corresponds with the desired skin incision [33].

After the skin incision, a variation of the Watson-Jones approach to the hip is used with dissection continuing in the interval between the tensor fascia latae and the gluteus medius. The fascia is incised in a curvilinear fashion directed convex posterior. A four-quadrant retractor is placed to assist with visualization of the deep structures. The anterior and posterior limbs are placed over the cut edges of the fascia, the superior limb is placed around the substance of gluteus medius, and the inferior limb is placed on the skin. The vastus lateralis and vastus intermedius are identified originating from the vastus ridge. The donor vessels can now be visualized anterior to the vastus intermedius in the interval between vastus intermedius and rectus femoris. The ascending branch of the lateral femoral circumflex artery and its two veins typically run obliquely cephalad at a 45° angle at a distance of 10 cm distal to the anterior superior iliac spine (Fig. 2).

Once the donor vessels are identified and can be appropriately protected, the vastus lateralis is sharply elevated to expose the lateral aspect of the proximal femur. An L-shaped incision is used with a longitudinal limb posteriorly and a transverse limb at the level of the vastus ridge. As the vastus intermedius is encountered anteriorly, a right-angle clamp and Metzenbaum scissors are used to safely release its attachments from the proximal femur and to avoid injury to the donor vessels. The complete reflection of the vastus intermedius provides a trough for the donor vessels during the later microvascular anastomosis. The vastus lateralis and vastus intermedius muscles are then secured to the anterior skin edge and placed deep to the inferior limb of the four-quadrant retractor.

The donor vessels can now be clearly seen lying within the aponeurotic falx of the rectus femoris anteriorly. The ascending branch of the lateral femoral circumflex artery and its two veins are carefully dissected out under loupe magnification. The smaller side branches of the vessels are ligated with small vascular clips such that a pedicle of at least 4 cm is obtained for later anastomosis. Typically, there is sufficient pedicle length if the vessels reach halfway up to the anticipated entry site of the fibula. The hip wound is copiously irrigated and the four-quadrant retractor is removed for preparation of the femoral head.

Preparation of the femoral head is done with the aid of c-arm fluoroscopy. The c-arm fluoroscope is draped sterilely and positioned over the patient like an arch. It is canted cephalad slightly to allow the surgeon a greater working space. The ability to obtain true anteroposterior (AP) and frog-leg lateral views is confirmed prior to

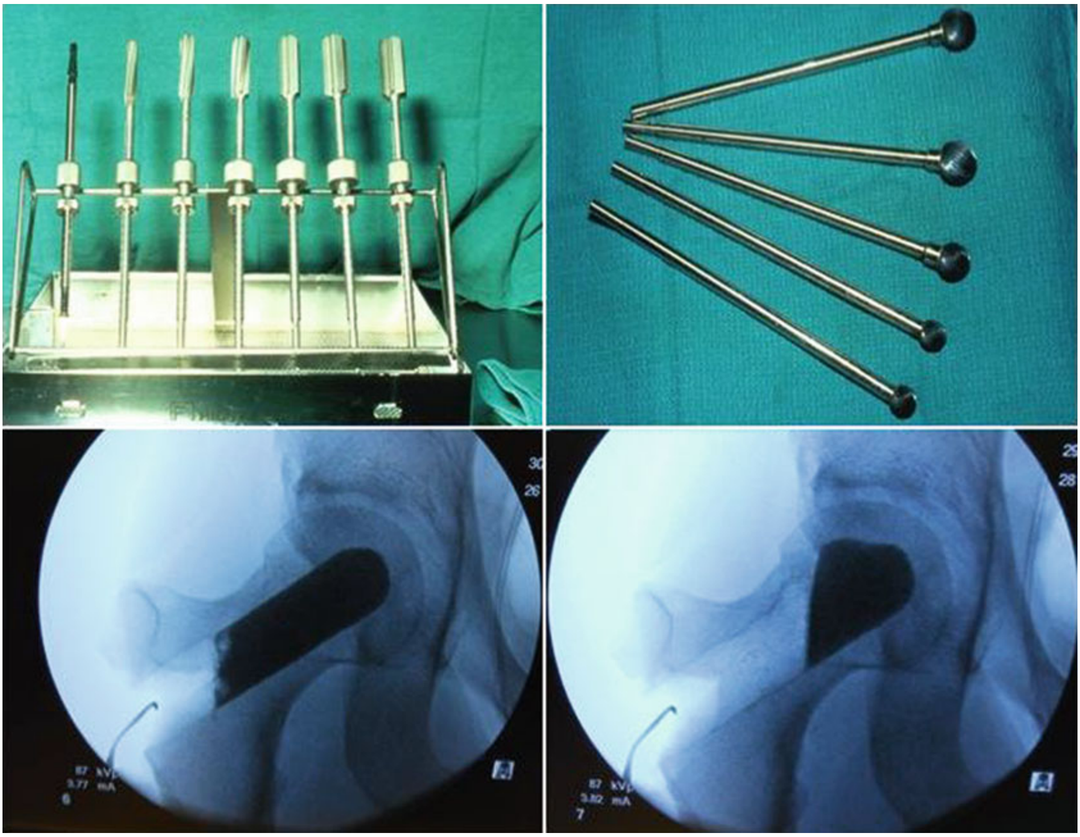


Fig. 3 Straight and ball-tipped reamers are used to remove necrotic bone. Radiographic contrast is used to evaluate the adequacy of the excavation of the osteonecrotic lesion

beginning preparation of the femoral head. With fluoroscopic guidance, a 3 mm sharp-tipped guide pin is placed into the center of the ON lesion. The starting point for this pin is approximately 2 cm distal to the vastus ridge at the junction of the middle and posterior thirds of the proximal femur. Care is taken to avoid making the entry point too distal as encroachment on the high-stress subtrochanteric region will increase the risk of fracture.

Once correct pin placement is confirmed on AP and lateral fluoroscopic views, the guide pin is overdrilled with a cannulated 8 mm drill bit. The sharp-tipped guide pin is exchanged for a blunt-tipped guide pin that is sequentially over-reamed with a series of specialized straight reamers beginning at 10 mm in diameter. These reamers are available in 13, 16, 17.5, 19, and 21 mm and they are sequentially used up to the size of the harvested fibula. The reaming is performed up to 4 mm from the subchondral plate using live fluoroscopy. The healthy bone that is collected in the flutes of the reamer is saved for later bone grafting while obviously necrotic bone is discarded. Similarly, a filtered suction device is used to collect bone fragments generated by the reaming from the core. The scrub nurse fashions this collected bone into "bullets" on the back table for later grafting.

After the final size-matched straight reamer is used, the guide pin is removed and a ball-tipped reamer is placed to remove the remaining area of necrotic bone. These reamers allow the creation of a bulbous cavity and are best used with the assistance of fluoroscopy. Radiographic contrast is used to evaluate the adequacy of the excavation of the ON lesion (Fig. 3).

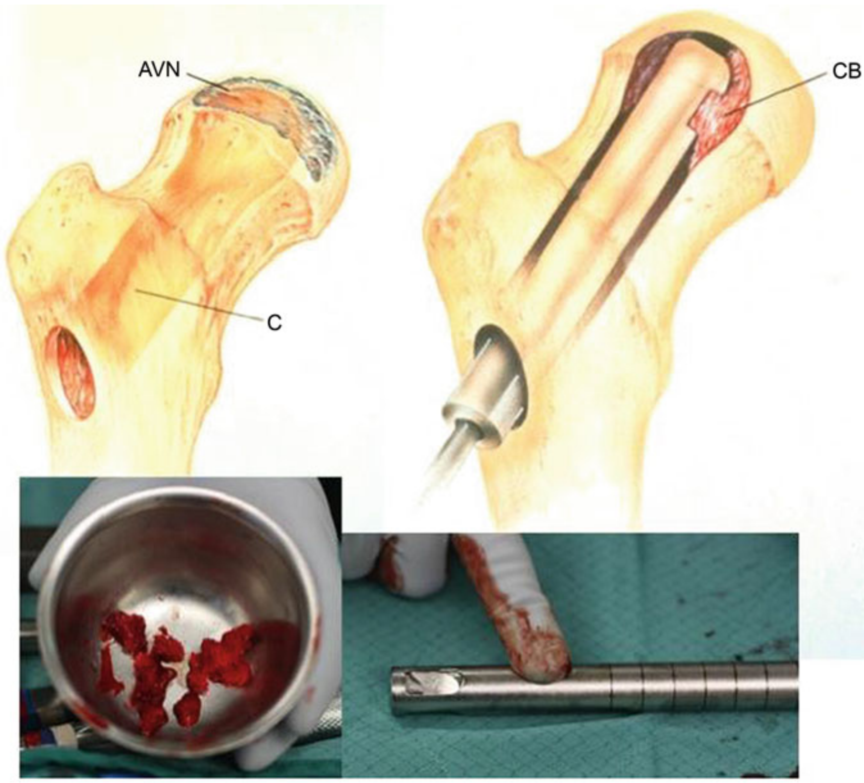


Fig. 4 Cancellous bone graft and *bullets* are placed within the core of the custom-made bone impactor, which disperses the graft within the cavity [34]

Cancellous bone is harvested from the greater trochanter through the lateral entry point using a large curet. This bone is kept separate from the bone graft obtained from the reamings. Long DeBakey forceps are used to place this bone graft into the cavity created within the femoral head. The Urbanipactor, a specialized impaction instrument designed for this procedure, is used to impact the bone graft. After placing the cancellous bone graft within the cavity, the Urbanipactor is fully inserted and its position is verified with fluoroscopy. The remaining cancellous bone graft and the bullets are placed within the core of the Urbanipactor and the specialized drill bit is used to disperse the graft within the cavity (Fig. 4). Graduated circumferential markings on the outside of the Urbanipactor are utilized to measure the depth of the core to determine the length of the fibular graft. The Urbanipactor is then removed and radiographic contrast medium is reinjected to confirm adequate bone grafting of the cavitary defect (Fig. 5).

The fibular graft is harvested at the same time as the hip preparation. The lower extremity is exsanguinated with an Esmarch bandage and a thigh tourniquet is inflated to 350 mmHg. A line is marked on the skin connecting the fibular head with the lateral malleolus. The proximal and distal 10 cm of fibula are marked and care is taken to preserve both the proximal and distal tibiofibular joints over this distance. Typically, this leaves approximately 15 cm of the central portion of the fibula available for harvest. The skin is incised over the sulcus between the lateral and posterior compartments of the leg with maintenance of full-thickness skin flaps. The fascia of the lateral compartment is incised longitudinally in line with the peroneal tendon with care to protect the superficial peroneal nerve. The peroneal muscles are

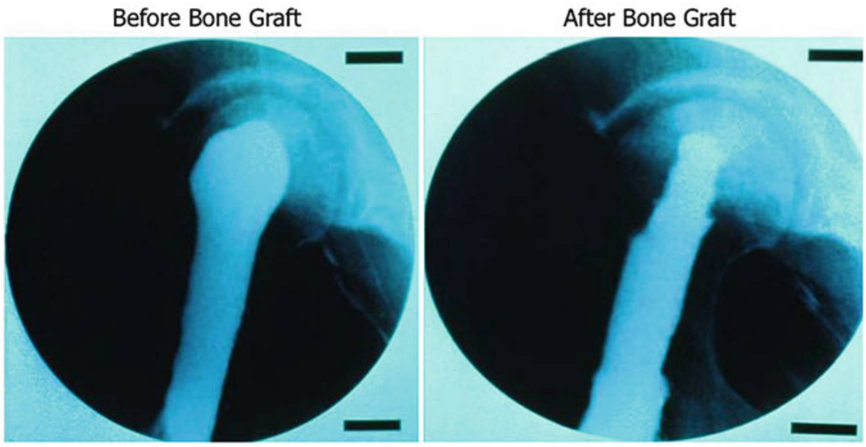


Fig. 5 Radiographic contrast medium is reinjected to confirm adequate bone grafting of the cavitary defect

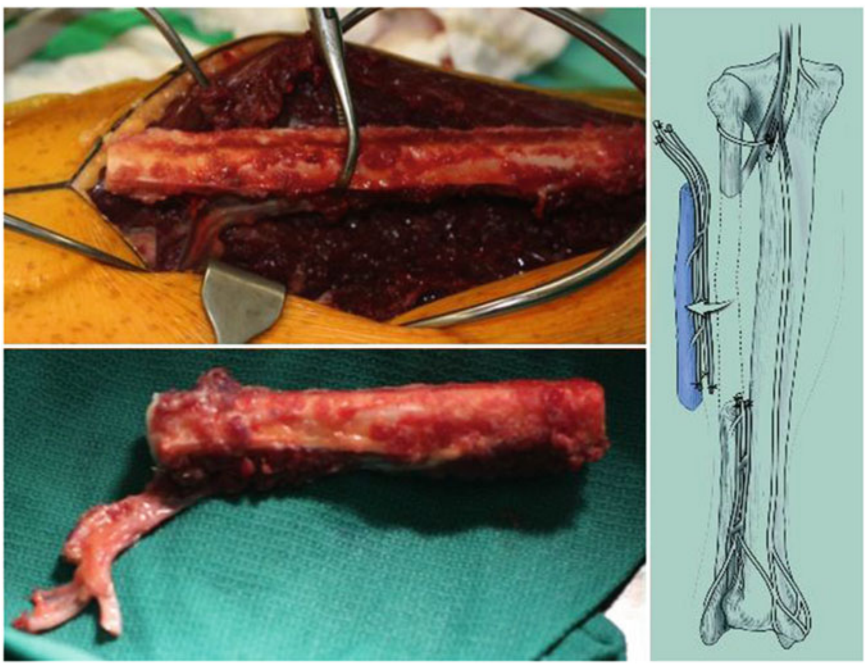


Fig. 6 Harvested fibular graft with peroneal vascular pedicle. During isolation, the neurovascular bundle is protected using a right-angle Beaver blade [34]

reflected anteriorly off the posterior intermuscular septum. The lateral aspect of the fibula is palpable deep to the peroneal muscles. Two large Gelpi retractors are placed to demonstrate this interval and maintain tension of the peroneal muscles as they are sharply reflected off of the fibula. A thin 1–2 mm layer or muscle is left with the fibula ("marble-izing") in order to preserve the periosteal blood supply to the fibula. Dissection is continued from posterior to anterior until the anterior intermuscular septum is encountered.

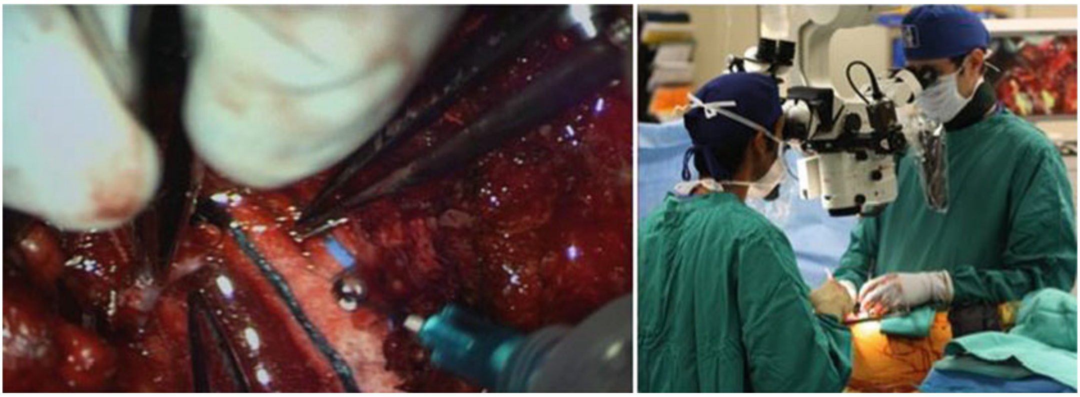


Fig. 7 The peroneal vessels and the ascending branch of the lateral femoral circumflex vessels are reliably present for anastomosis. Their orientation makes performing the microvascular anastomosis easiest from patient's posterior side

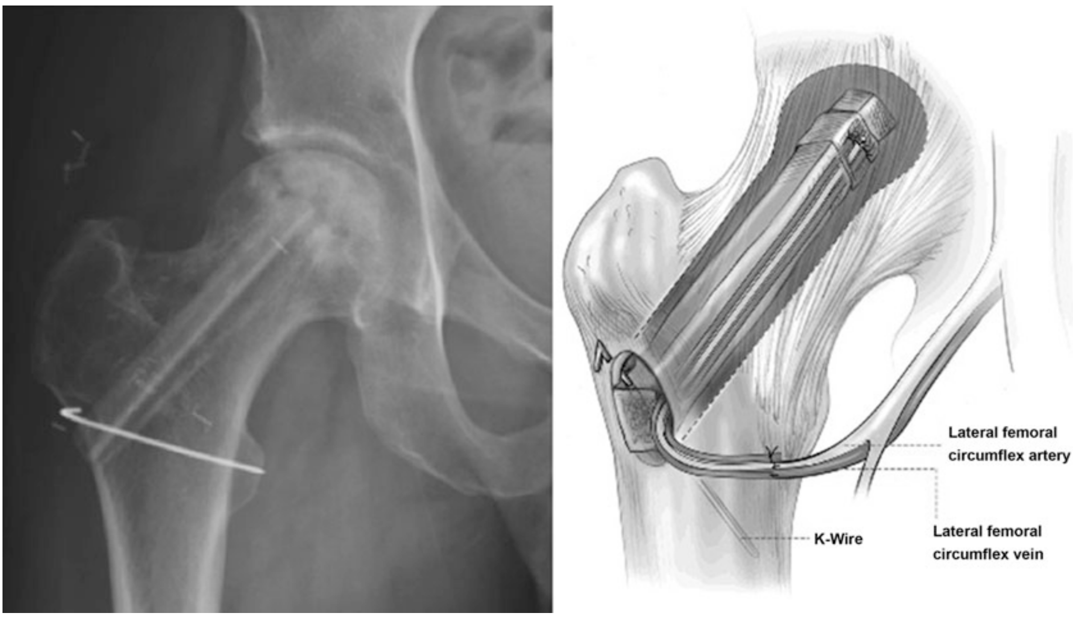


Fig. 8 A final schematic and 1-year anteroposterior postoperative radiograph [34]

A #15 scalpel is used to divide the anterior intermuscular septum approximately 1–2 mm off of the fibula. The anterior compartment musculature is then encountered and bluntly dissected off of the fibula with Metzenbaum scissors and DeBakey forceps. The interosseous membrane is then easily visualized as are the anterior tibial artery and the deep peroneal nerve. The neurovascular structures are protected while the interosseous membrane is released from the fibula with the use of a right-angle Beaver blade (Fig. 6).

The peroneal artery and the ascending branch of the lateral femoral circumflex artery are typically 1.5–2 mm in size and are reliably present. The concomitant veins are typically 2.5–4 mm in diameter. The vessels are oriented in an anteroposterior direction, which usually places the microsurgical field on a slight incline. For this reason, it is easiest for the surgeon performing the microvascular anastomosis to be standing on the patient's posterior side (Fig. 7). A final schematic and 1-year postoperative radiograph is seen in Fig. 8.

Summary

ONFH is a common cause of hip pain that can result in significant morbidity in a young adult patient demographic. Vascularized bone grafts have been shown to have better radiographical and clinical results compared to nonvascularized bone grafts. Of these options, free vascularized fibular graft is the treatment of choice for its structural and vascular support within the femoral head. Improved technique has led to complication rates, including donor-site morbidity, statistically similar to that of nonvascular fibular grafts. When used in young patients with precollapse ONFH, FVFG can greatly improve patient outcome, with lower rates of conversion to THA than other treatment modalities [29]. At 10-year follow-up, 75 % (49/65 hips) of FVFG were surviving. In those that were converted, the average conversion was at 8.3 years postoperatively [35]. As judged by Harris hip scores and SF-12 physical component summary scores, the function of THA and FVFG is similar; however, SF-12 mental component summary scores are higher in patients surviving FVFG [35]. Moreover, these patients also have a higher capacity to compete in athletic events, including impact sports, than their THA counterparts.

References

1. Jacobs B. Epidemiology of traumatic and nontraumatic osteonecrosis. Clin Orthop Relat Res. 1978;130:51–67.
2. Coventry MB, Beckenbaugh RD, Nolan DR, Ilstrup DM. 2,012 total hip arthroplasties. A study of postoperative course and early complications. J Bone Joint Surg Am. 1974;56(2):273–84.
3. Mont MA, Hungerford DS. Non-traumatic avascular necrosis of the femoral head. J Bone Joint Surg Am. 1995;77(3):459–74.
4. Mankin HJ. Nontraumatic necrosis of bone (osteonecrosis). N Engl J Med. 1992;326(22):1473–9.
5. Forgon M, Montsko P. Muscle-pedicle grafts or muscle-pedicle-bone grafts in the treatment of avascular necrotic femoral heads. Arch Orthop Trauma Surg. 1996;115(3–4):223–6.
6. Tetik C, Basar H, Bezer M, Erol B, Agir I, Esemenli T. Comparison of early results of vascularized and non-vascularized fibular grafting in the treatment of osteonecrosis of the femoral head. Acta Orthop Traumatol Turc. 2011;45(5):326–34.
7. Etienne G, Mont MA, Ragland PS. The diagnosis and treatment of nontraumatic osteonecrosis of the femoral head. Instr Course Lect. 2004;53:67–85.
8. Steinberg M. Diagnostic imaging and role of stage and lesion size in determining outcome in osteonecrosis of the femoral head. Tech Orthop. 2001;16:6–15.
9. Lang P, Jergesen HE, Moseley ME, Block JE, Chafetz NI, Genant HK. Avascular necrosis of the femoral head: high-field-strength MR imaging with histologic correlation. Radiology. 1988;169(2):517–24.
10. Mont MA, Carbone JJ, Fairbank AC. Core decompression versus nonoperative management for osteonecrosis of the hip. Clin Orthop Relat Res. 1996;324:169–78.
11. Sen RK. Management of avascular necrosis of femoral head at pre-collapse stage. Indian J Orthop. 2009;43 (1):6–16.
12. Hungerford D Bone marrow pressure, venography, and core decompression in ischemic necrosis of the femoral head. The Hip: Proceedings of the seventh open scientific meeting of the hip society. 1979;218.
13. Ficat R, Arlet J. Functional investigation of bone under normal conditions. In: Ficat R, Arlet J, Hungerford D, editors. Ischemia and necrosis of bone. Baltimore: Williams & Wilkins; 1980. p. 29–52.
14. Mont MA, Einhorn TA, Sponseller PD, Hungerford DS. The trapdoor procedure using autogenous cortical and cancellous bone grafts for osteonecrosis of the femoral head. J Bone Joint Surg (Br). 1998;80 (1):56–62.
15. Ganz R, Buchler U. Overview of attempts to revitalize the dead head in aseptic necrosis of the femoral head – osteotomy and revascularization. Hip. 1983;296–305.
16. Buckley PD, Gearen PF, Petty RW. Structural bone-grafting for early atraumatic avascular necrosis of the femoral head. J Bone Joint Surg Am. 1991;73 (9):1357–64.
17. Chacha PB. Vascularised pedicular bone grafts. Int Orthop. 1984;8(2):117–38.
18. Meyers MH. The treatment of osteonecrosis of the hip with fresh osteochondral allografts and with the muscle pedicle graft technique. Clin Orthop Relat Res. 1978;130:202–9.
19. Lee CK, Rehmatullah N. Muscle-pedicle bone graft and cancellous bone graft for the “silent hip” of idiopathic ischemic necrosis of the femoral head in adults. Clin Orthop Relat Res. 1981;158:185–94.
20. Baksi DP. Treatment of osteonecrosis of the femoral head by drilling and muscle-pedicle bone grafting. J Bone Joint Surg (Br). 1991;73(2):241–5.
21. Iwata H, Torii S, Hasegawa Y, et al. Indications and results of vascularized pedicle iliac bone graft in avascular necrosis of the femoral head. Clin Orthop Relat Res. 1993;295:281–8.
22. Stein H, Volpin G, Horer D. Vascularized muscle pedicle flap for osteonecrosis of the femoral head. Orthopedics. 2002;25(5):485–8.

23. Urbaniak JR, Coogan PG, Gunneson EB, Nunley JA. Treatment of osteonecrosis of the femoral head with free vascularized fibular grafting. A long-term follow-up study of one hundred and three hips. J Bone Joint Surg Am. 1995;77(5):681–94.
24. Brunelli G, Brunelli G. Free microvascular fibular transfer for idiopathic femoral head necrosis: long-term follow-up. J Reconstr Microsurg. 1991;7(4):285–95.
25. Judet H, Judet J, Gilbert A. Vascular microsurgery in orthopaedics. Int Orthop. 1981;5(2):61–8.
26. Kim SY, Kim YG, Kim PT, Ihn JC, Cho BC, Koo KH. Vascularized compared with nonvascularized fibular grafts for large osteonecrotic lesions of the femoral head. J Bone Joint Surg Am. 2005;87(9):2012–8.
27. Plakseychuk AY, Kim SY, Park BC, Varitimidis SE, Rubash HE, Sotereanos DG. Vascularized compared with nonvascularized fibular grafting for the treatment of osteonecrosis of the femoral head. J Bone Joint Surg Am. 2003;85-A(4):589–96.
28. Tang CL, Mahoney JL, McKee MD, Richards RR, Waddell JP, Louie B. Donor site morbidity following vascularized fibular grafting. Microsurgery. 1998;18 (6):383–6.
29. Fang T, Zhang EW, Sailes FC, McGuire RA, Lineaweaver WC, Zhang F. Vascularized fibular grafts in patients with avascular necrosis of femoral head: a systematic review and meta-analysis. Arch Orthop Trauma Surg. 2013;133(1):1–10.
30. Yoshimura M, Shimamura K, Iwai Y, Yamauchi S, Ueno T. Free vascularized fibular transplant. A new method for monitoring circulation of the grafted fibula. J Bone Joint Surg Am. 1983;65(9):1295–301.
31. Hungerford DS, Zizic TM. Alcoholism associated ischemic necrosis of the femoral head. Early diagnosis and treatment. Clin Orthop Relat Res. 1978;130:144–53.
32. Urbaniak JR, Harvey EJ. Revascularization of the femoral head in osteonecrosis. J Am Acad Orthop Surg. 1998;6(1):44–54.
33. Marchant Jr MH, Zura RD, Urbaniak JR, Aldridge 3rd JM. Hip incision planning for free vascularized fibular grafting of the proximal femur: a handy tip. J Surg Orthop Adv. 2007;16(4):204–6.
34. Aldridge 3rd JM, Berend KR, Gunneson EE, Urbaniak JR. Free vascularized fibular grafting for the treatment of postcollapse osteonecrosis of the femoral head. Surgical technique. J Bone Joint Surg Am. 2004;86-A Suppl 1:87–101.
35. Eward WC, Rineer CA, Urbaniak JR, Richard MJ, Ruch DS. The vascularized fibular graft in precollapse osteonecrosis: is long-term hip preservation possible? Clin Orthop Relat Res. 2012;470(10):2819–26.

Surgical Technique: Bone Graft for Avascular Necrosis of the Hip

86

Jonathan M. Frank, Andrew E. Federer, Richard C. Mather III, and David S. Ruch

Contents

Abstract

Avascular necrosis of the hip is a debilitating disease process leading to end-stage hip degeneration and eventual total hip arthroplasty. Early recognition allows for salvage treatment options that may delay joint degeneration. Core decompression with bone grafting is an effective treatment strategy.

Introduction

Avascular necrosis (AVN) of the hip results from a disruption in blood supply to the trabecular bone of the femoral head. This condition affects a predominantly young demographic, average age mid-30s, amounting to 10,000–20,000 cases every year [1, 2]. This can be due to trauma, thromboemboli, prolonged steroid therapy, alcoholism, Caisson's disease, or increased intramedullary pressure within the bone as seen in Legg-Calvé-Perthes or Gaucher's disease [3]. If left untreated, ~80 % of cases of osteonecrosis of the femoral head (ONFH) will result in femoral head collapse and hip degeneration [1, 4]. This natural history of disease, in conjunction with a young demographic, has led to the search for a treatment that can restore structural integrity of the femoral head and delay or prevent the eventual need for total hip arthroplasty (THA).

J.M. Frank (✉)
Department of Orthopaedic Surgery, Rush University Medical Center, Chicago, IL, USA
e-mail: jon.m.frank@gmail.com

A.E. Federer
Midwest Orthopaedics at Rush, Hip Preservation Center, Rush University Medical Center, Chicago, IL, USA
e-mail: afederer34@gmail.com

R.C. Mather III
Department of Orthopaedic Surgery, Duke University School of Medicine, Durham, NC, USA
e-mail: chad.mather@gmail.com

D.S. Ruch
Duke University Health System, Durham, NC, USA
e-mail: david.ruch@duke.edu

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_110

Clinical Exam, Imaging, and Staging

Early stages of the disease may present without pain, but ultimately the physical exam will show painful and limited range of motion (ROM) [1]. The Harris hip score (HHS), which takes into account factors such as pain, function, and range of motion, is one of the most common tools for evaluating hip status [5].

Plain radiographs are necessary for the diagnosis of ONFH; however, findings are often limited or absent in the early stages of the disease. Plain radiographs will only show bone density changes associated with remodeling or disuse and therefore may only detect changes months or even years after the disease starts [3]. The anteroposterior view is used to detect the majority of AVN; however, since the anterior and posterior acetabulum may overlap with and skew images of the femoral head, frog-leg lateral views may be particularly useful for more detailed assessment, such as detecting a crescent sign [5, 6]. In cases of suspected ONFH without signs on plain radiographs, magnetic resonance imaging (MRI) should be considered.

MRI is the most sensitive imaging study for the diagnosis of ONFH, especially for asymptomatic or early stage cases [3, 6, 7]. The presence of a low-intensity band on T1-weighted images is an early sign of osteonecrosis, and T2-weighted images nearly always show focal increases in signal intensity [8]. For at-risk patients, sensitivity and specificity can be as high as 100 % and 98 %, respectively [3, 9–12]. In evaluating nonfat-suppressed T2 SE and turbo spin echo (TSE), a “double line” sign is virtually diagnostic [5, 13]. Fat-suppressed PD/T2-weighted and cartilage-specific sequences, especially axial oblique views for the anterosuperior femoral head, can be used to determine femoral head marrow abnormalities [13]. According to reports [14–16], high fat content and bone marrow edema in the proximal femur is predictive of osteonecrosis [14–17].

There are four major classification systems used in diagnosing ONFH based on radiographic staging: Ficat and Arlet, Steinberg, ARCO, and Japanese Orthopaedic Association [18]. In all these classification systems, loss of the spherical contour of the femoral head is principal [19]. The Ficat-Arlet staging system is the most common and stages hips based on the four parameters of collapse, size, femoral head depression, and acetabular involvement. Stage I shows no radiographic changes to the femoral head. Stage II represents precollapse of the femoral head with radiographic findings of subchondral sclerosis, most commonly at the anterior and superior femoral head. Stage III shows the crescent sign, which represents subchondral fracture best seen on frog-leg view, and subchondral collapse. Stage IV shows acetabular involvement and gross joint deformity.

Treatment

Nonoperative interventions are considered initially, especially in asymptomatic individuals. Depending upon the specific etiology, lipid-lowering drugs, anticoagulants, vasodilators, and bisphosphonates may be offered [5]. Though a non-weight-bearing status is often used in conjunction with other methods, evidence from a meta-analysis including over 800 hips shows that 76 % of hips treated solely with restrictive weight bearing required an arthroplasty or salvage procedure [20]. The results from this and similar studies show universal agreement that restrictive weight bearing as sole treatment is inappropriate [5]. Nonoperative treatment is not recommended in symptomatic patients [20, 21]. The status of the femoral head is the most important management factor in determining which procedure to use.

Surgical options include core decompression, with and without vascularized or nonvascularized bone graft, osteotomy (transtrochanteric or intertrochanteric), as well as bone reshaping, hemi- resurfacing, total resurfacing, and THA. Bone grafting is often used in conjunction with core decompression and can consist of autogenous or allograft cancellous bone of the iliac crest, tibia, or fibula with augmentation with calcium phosphate cement [5].

Core decompression, first coined by Hungerford, Ficat, and Arlet, is thought to work by lowering interosseus pressure and stimulating neovascularization of the femoral head and neck.

However, it is sometimes considered incomplete due to inadequate creeping substitution and bone remodeling [22]. Bone grafting is performed with core decompression as it provides mechanical support for the articular surface while encouraging bone healing with vascularization of the subchondral bone [3, 5]. Broad categories of bone grafting for ONFH are vascularized, nonvascularized, bone morphogenetic proteins and bone marrow mesenchymal grafting.

Nonvascularized bone grafts have excellent short- to midterm follow-up and work best in cases of precollapse. The most commonly employed nonvascularized techniques are the core tract [23], chondral window [24], and "light bulb" [25] procedures. The core tract technique, described by Phemister et al [23], involves creating a core tract through the femoral neck, removing the necrotic bone and inserting a cortical strut allograft. This method, like most core decompression-bone graft techniques, is aimed at precollapse femoral heads [26]. Buckley et al [27] used this technique with autogenous and allograft tibial and fibular nonvascularized bone graft in 20 patients with Stage I and II AVN, resulting in 90 % satisfaction at a mean follow-up of 8 years. Despite these promising short-term results, several long-term studies have shown a decrease in satisfaction. Smith et al [28] report a poor clinical result in the surviving patients at a mean 14-year follow-up with 25 of 38 patients having undergone revision at an average of 9 years. Keizel et al [29] conducted a study with 80 hips of varying stages looking at long-term survival of both nonvascularized fibula autografts and tibial allografts. They saw 59 % survival at 5-year follow-up with 44 % revision surgery at a mean of 4 years. Tibial allograft showed a statistically significant benefit using both clinical and radiographical endpoints compared to fibular autograft.

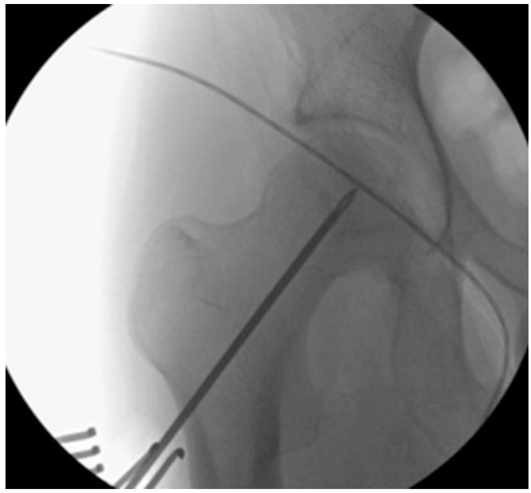


Fig. 1 A guide wire is passed through the lateral femoral cortex into the necrotic area of the femoral head. The starting point should not be distal to the lesser trochanter as this may increase the risk of subtrochanteric fracture

Surgical Technique

The patient is placed in the lateral decubitus position on a radiolucent table. The greater trochanter is outlined and a 5-cm longitudinal incision is made just distal to the trochanteric ridge along the lateral aspect of the femur. Dissection is carried down to and through the iliotibial band using electrocautery, in line with the skin incision. The vastus lateralis is then incised, again in line with the skin incision up to the trochanteric ridge. Fluoroscopy is used to introduce a 2.3 mm guide pin (Fig. 1) from a starting point of just distal to the intertrochanteric ridge, up into the femoral head lesion. A cannulated drill is then passed over the guide wire (Fig. 2), decompressing the necrotic lesion but being careful to not penetrate into the joint. Using the same starting point, multiple trajectories are taken with the guide wire advanced to different portions of the necrotic lesion. With the necrotic lesion adequately decompressed, curettes are then used to harvest autologous cancellous bone graft from the greater trochanter. This bone graft is formed into pellets that can be impacted into the necrotic cavity in the femoral head. With the lesion packed with cancellous autograft, calcium phosphate bone void filler (Norian SRS, Synthes Inc, West Chester, PA) is then used to fill the entry to the core tunnel (Fig. 3). The wound is then thoroughly irrigated with saline. The iliotibial band, subcutaneous layer, and skin are closed, and a sterile dressing is applied.

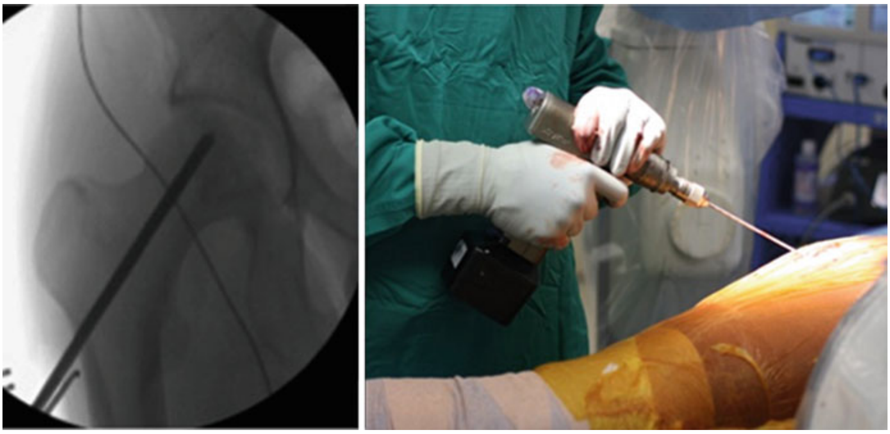


Fig. 2 A cannulated drill is introduced over the guide wire into the subchondral bone. This is repeated in several directions to fully decompress the necrotic lesion

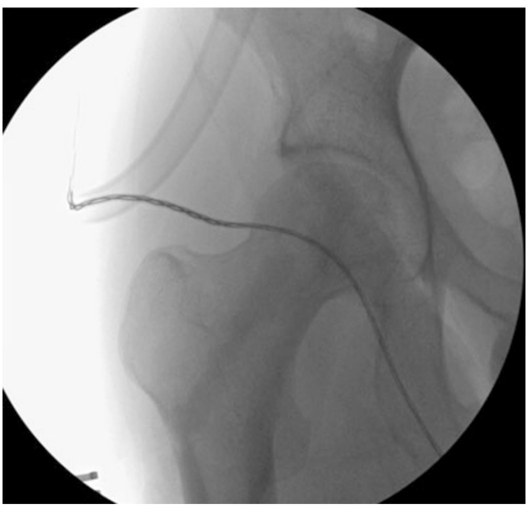


Fig. 3 Anteroposterior intraoperative view of the hip after the core has been filled with autologous bone graft followed by calcium phosphate bone void filler (Norian)

The rehabilitation protocol includes non-weight bearing with crutches for the first 6 weeks to minimize the risk of femoral neck fracture. The patient will begin supervised physical therapy immediately postoperatively to maintain hip range of motion. At 6 weeks after surgery, the patient is allowed to progressively weight bear until full weight bearing is achieved.

Summary

AVN often afflicts a younger demographic, making the prospect of undergoing THA at this age a difficult decision. Hip prostheses in this demographic often undergo greater wear than in older patients, which is important as the average survivorship of a total hip implant is inversely proportional to activity level [30], setting the patient up for several revision surgeries over his lifespan. Moreover, in a more athletic demographic, hip resurfacing and THA often signify the end of a competitive athletic career; therefore these procedures are typically deferred to as latter options [31]. In appropriately selected patients, core decompression with bone graft is a viable and reliable option to delay the need for arthroplasty.

References

1. Mont MA, Hungerford DS. Non-traumatic avascular necrosis of the femoral head. J Bone Joint Surg Am. 1995;77(3):459–74.
2. Mankin HJ. Nontraumatic necrosis of bone (osteonecrosis). N Engl J Med. 1992;326(22):1473–9.
3. Etienne G, Mont MA, Ragland PS. The diagnosis and treatment of nontraumatic osteonecrosis of the femoral head. Instr Course Lect. 2004;53:67–85.

4. Forgon M, Montsko P. Muscle-pedicle grafts or muscle-pedicle-bone grafts in the treatment of avascular necrotic femoral heads. Arch Orthop Trauma Surg. 1996;115(3–4):223–6.
5. Sen RK. Management of avascular necrosis of femoral head at pre-collapse stage. Indian J Orthop. 2009;43 (1):6–16.
6. Steinberg M. Diagnostic imaging and role of stage and lesion size in determining outcome in osteonecrosis of the femoral head. Tech Orthop. 2001;16:6–15.
7. Lang P, Jergesen HE, Moseley ME, Block JE, Chafetz NI, Genant HK. Avascular necrosis of the femoral head: high-field-strength MR imaging with histologic correlation. Radiology. 1988;169(2):517–24.
8. Jergesen HE, Heller M, Genant HK. Signal variability in magnetic resonance imaging of femoral head osteonecrosis. Clin Orthop Relat Res. 1990;253:137–49.
9. Fink B, Degenhardt S, Paselk C, Schneider T, Modder U, Ruther W. Early detection of avascular necrosis of the femoral head following renal transplantation. Arch Orthop Trauma Surg. 1997;116(3):151–6.
10. van Vugt RM, Sijbrandij ES, Bijlsma JW. Magnetic resonance imaging of the femoral head to detect avascular necrosis in active rheumatoid arthritis treated with methylprednisolone pulse therapy. Scand J Rheumatol. 1996;25(2):74–6.
11. Sugano N, Masuhara K, Nakamura N, Ochi T, Hirooka A, Hayami Y. MRI of early osteonecrosis of the femoral head after transcervical fracture. J Bone Joint Surg Br. 1996;78(2):253–7.
12. Sakamoto M, Shimizu K, Iida S, Akita T, Moriya H, Nawata Y. Osteonecrosis of the femoral head: a prospective study with MRI. J Bone Joint Surg Br. 1997;79(2):213–9.
13. Karantanas AH, Drakonaki EE. The role of MR imaging in avascular necrosis of the femoral head. Semin Musculoskelet Radiol. 2011;15(3):281–300.
14. Cui Q, Wang Y, Saleh KJ, Wang GJ, Balian G. Alcohol-induced adipogenesis in a cloned bone-marrow stem cell. J Bone Joint Surg Am. 2006;88 Suppl 3:148–54.
15. Lee JS, Lee JS, Roh HL, Kim CH, Jung JS, Suh KT. Alterations in the differentiation ability of mesenchymal stem cells in patients with nontraumatic osteonecrosis of the femoral head: comparative analysis according to the risk factor. J Orthop Res. 2006;24(4):604–9.
16. Suh KT, Kim SW, Roh HL, Youn MS, Jung JS. Decreased osteogenic differentiation of mesenchymal stem cells in alcohol-induced osteonecrosis. Clin Orthop Relat Res. 2005;431:220–5.
17. Petrigliano FA, Lieberman JR. Osteonecrosis of the hip: novel approaches to evaluation and treatment. Clin Orthop Relat Res. 2007;465:53–62.
18. Mont MA, Marulanda GA, Jones LC, et al. Systematic analysis of classification systems for osteonecrosis of the femoral head. J Bone Joint Surg Am. 2006;88 Suppl 3:16–26.
19. Karantanas AH. Accuracy and limitations of diagnostic methods for avascular necrosis of the hip. Expert Opin Med Diagn. 2013;7(2):179–87.
20. Mont MA, Carbone JJ, Fairbank AC. Core decompression versus nonoperative management for osteonecrosis of the hip. Clin Orthop Relat Res. 1996;324:169–78.
21. Musso ES, Mitchell SN, Schink-Ascani M, Bassett CA. Results of conservative management of osteonecrosis of the femoral head. A retrospective review. Clin Orthop Relat Res. 1986;207:209–15.
22. Ficat RP. Idiopathic bone necrosis of the femoral head. early diagnosis and treatment. J Bone Joint Surg Br. 1985;67(1):3–9.
23. Phemister DB. Treatment of the necrotic head of the femur in adults. J Bone Joint Surg Am. 1949;31A (1):55–66.
24. Mont MA, Einhorn TA, Sponseller PD, Hungerford DS. The trapdoor procedure using autogenous cortical and cancellous bone grafts for osteonecrosis of the femoral head. J Bone Joint Surg Br. 1998;80(1):56–62.
25. Rosenwasser MP, Garino JP, Kiernan HA, Michelsen CB. Long term followup of thorough debridement and cancellous bone grafting of the femoral head for avascular necrosis. Clin Orthop Relat Res. 1994;306:17–27.
26. Nelson LM, Clark CR. Efficacy of phemister bone grafting in nontraumatic aseptic necrosis of the femoral head. J Arthroplasty. 1993;8(3):253–8.
27. Buckley PD, Gearen PF, Petty RW. Structural bone-grafting for early atraumatic avascular necrosis of the femoral head. J Bone Joint Surg Am. 1991;73 (9):1357–64.
28. Smith KR, Bonfiglio M, Montgomery WJ. Non-traumatic necrosis of the femoral head treated with tibial bone-grafting. A follow-up note. J Bone Joint Surg Am. 1980;62(5):845–7.
29. Keizer SB, Kock NB, Dijkstra PD, Taminiau AH, Nelissen RG. Treatment of avascular necrosis of the hip by a non-vascularised cortical graft. J Bone Joint Surg Br. 2006;88(4):460–6.
30. Aldridge III JM, Urbaniak JR. Vascularized fibular grafting for osteonecrosis of the femoral head with unusual indications. Clin Orthop Relat Res. 2008;466 (5):1117–24.
31. Aldridge III JM, Urbaniak JR. Avascular necrosis of the femoral head: role of vascularized bone grafts. Orthop Clin North Am. 2007;38(1):13–22.

Part VIII

Hip Cartilage Restoration

Hip Cartilage Restoration: Overview

87

Lisa M. Tibor and Jeffrey A. Weiss

Contents

L.M. Tibor (✉)
Kaiser Permanente Medical Center, South San Francisco, CA, USA
e-mail: lisa.tibor@gmail.com

J.A. Weiss
Departments of Bioengineering and Orthopaedics, and Scientific Computing and Imaging Institute, University of Utah, Salt Lake City, UT, USA
e-mail: jeff.weiss@utah.edu

Abstract

Articular cartilage must resist mechanical loading modes that include compression, tension, and shear. The material characteristics of articular cartilage are optimized for these roles and are intricately related to structure and composition. When full-thickness chondral and osteochondral defects occur in the hip, they are painful, causing mechanical symptoms and inflammation as a result of cartilage breakdown. These defects often progress over time. The treatment goals for cartilage restoration surgery are the resolution of symptoms, return to activity, and prevention of progressive damage. To achieve this, a preoperative plan is essential, and the nature of the lesion including size, location, underlying etiology, or associated structural pathoanatomy must be known. Indications for treating a focal chondral defect in the hip include acute trauma with an unstable fragment, continued pain and symptoms despite conservative management, a visible chondral defect on preoperative imaging with a positive response to a diagnostic intra-articular injection, and intra-articular loose bodies. The objective of this chapter is to describe the current state of the art for restoration of focal articular cartilage defects in the hip. To support this objective, we review the basic structure and function of articular cartilage as well as the biomechanics of the hip and of focal defects. Subsequently, the clinical presentation, diagnosis, and suspected underlying causes of damage

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_96

to articular surfaces in the hip are reviewed. Finally, we discuss the available treatment options, their relative indications, and their published outcomes to date.

Introduction

Full-thickness chondral and osteochondral defects in the hip are painful, causing mechanical symptoms and inflammation as a result of cartilage breakdown. In addition, these defects progress over time from increased mechanical stress on the surrounding cartilage and from the increased intra-articular inflammation. Thus, the treatment goals for cartilage restoration surgery in the hip are to improve pain and symptoms and to slow the progression to osteoarthritis. Accordingly, this chapter focuses on treatments for focal full-thickness chondral or osteochondral defects.

Cartilage damage negatively influences the outcomes of open and arthroscopic management of femoroacetabular impingement (FAI) as well as the outcome of acetabular reorientation for dysplasia [1, 2]. Conversely, untreated FAI or dysplasia causes continued mechanical stress on an area of repaired cartilage, which negatively influences the outcomes of cartilage surgery. Thus, identification of bony pathoanatomy is an important part of the preoperative planning for cartilage restoration surgery. In addition to FAI and dysplasia, there are several traumatic and nontraumatic causes of focal cartilage damage. Osteochondral lesions occur in patients who had Perthes' disease in childhood [3] and in those with femoral head avascular necrosis or osteochondritis dissecans lesions. Patients with femoroacetabular impingement (FAI) frequently have acetabular rim lesions and, less commonly, parafoveal defects [4]. Instability occurring as a result of dysplasia causes chondral damage at the superior aspect of the acetabulum and femoral head (Fig. 1) [5], whereas an episode of traumatic instability in conjunction with FAI usually causes a posterior rim lesion and/or a parafoveal femoral head defect (Fig. 2) [6]. Finally, a fall on the greater trochanter can cause a lateral impact event, transmitting force to the central femoral head and/or acetabulum and causing cartilage damage [7].

When compared to what is known about cartilage restoration in the knee, there is relatively little information specifically about cartilage restoration in the hip. Currently, most cartilage repair strategies for the hip are based on basic science and techniques that were developed for the knee. In the past decade, however, there has been a dramatic improvement in magnetic resonance imaging (MRI), arthroscopy, and open surgery for younger patients with hip pain. These improvements should also lead to hip-specific cartilage science and restoration techniques. The objective of this chapter is to describe the current state of the art for treating focal articular cartilage defects in the hip. To support this objective, we review the basic structure and function of articular cartilage as well as the biomechanics of the hip and focal articular defects. Subsequently, the clinical presentation, diagnosis, and suspected underlying causes of damage to articular surfaces in the hip are reviewed. Finally, we discuss the available treatment options, their relative indications, and their published outcomes to date.

Cartilage Basic Science: General

Articular cartilage must resist mechanical loading modes that include compression, tension, and shear. The material characteristics of articular cartilage are optimized for these roles and are intricately related to structure and material composition. This section provides a brief review of these topics.

Structure and Composition

Articular cartilage is composed primarily of water, collagen, and large proteoglycans (Fig. 3, left panel). By wet weight, cartilage is 68–85 % water, 10–20 % collagen, 5–10 % proteoglycan, and <5 % other matrix molecules [8]. The interstitial fluid contains dissolved electrolytes, predominantly Na^{+}, Ca^{2+}, Cl^{-}, and K^{+}. Chondrocytes account for less than 10 % of the total tissue volume [9] and are responsible for the metabolic activity of cartilage. The primary collagen in articular cartilage is fibril-forming type II collagen with variable orientation of

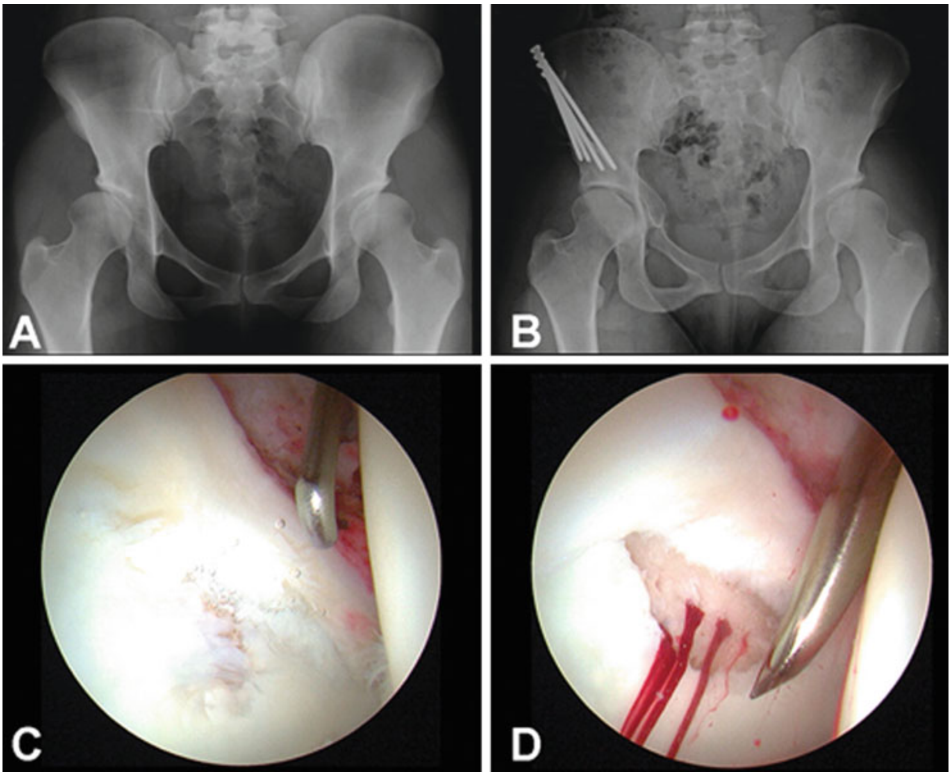


Fig. 1 Images from a patient with dysplasia (**a**) who underwent arthroscopy and periacetabular osteotomy (**b**). At the time of arthroscopy, she had full-thickness chondral damage at the acetabular rim (**c**) and underwent microfracture of the lesion (**d**) (Reprinted from [5], Copyright © 2011 by American Orthopaedic Society for Sports Medicine, by permission of SAGE Publications)

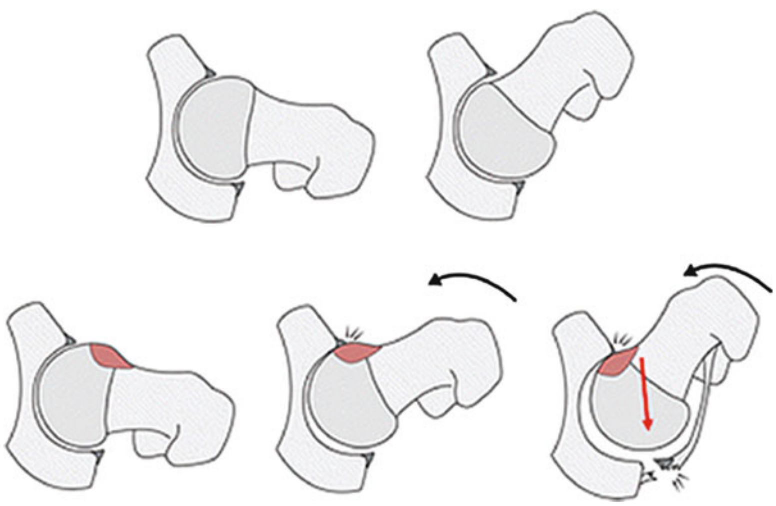


Fig. 2 Episodes of traumatic hip posterior hip dislocation occur with the hip in flexion and internal rotation, with a posteriorly directed force on the femur. If there is a cam lesion present, the femoral head and neck can lever on the anterior acetabular rim. As the femoral head dislocates posteriorly, chondrolabral damage can occur at the posterior labral rim, analogous to a bony Bankart lesion in the shoulder (Reprinted with permission from Springer Science+Business Media, from [6]. Copyright © 2012, The Association of Bone and Joint Surgeons®)

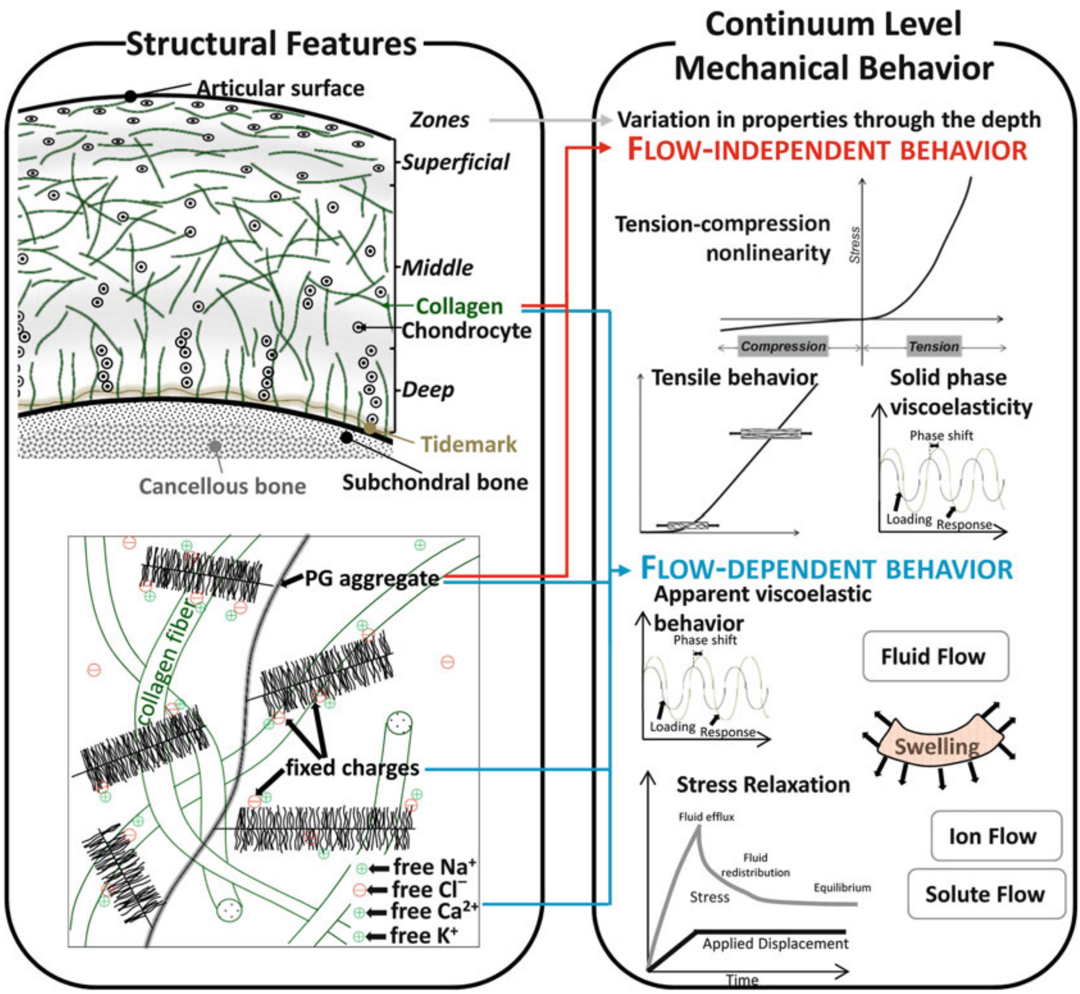


Fig. 3 Cartilage structural features and their relationship to continuum level mechanical behavior. *Left panel* – the structure and orientation of collagen and proteoglycan aggregates determine the continuum level mechanical behavior. *Right panel* – key features of continuum mechanical behavior include tension–compression nonlinearity, anisotropy, viscoelastic material behavior, and swelling

collagen fibers depending on the depth of the tissue. In the superficial zone (top 10–20 %), fibers are oriented parallel to the articular surface; in the middle zone (middle 40–60 %), fibers are oriented randomly; and in the deep zone (bottom ~30 %), the fibers are oriented perpendicularly to the subchondral bone. Aggrecan accounts for 80–90 % of all proteoglycan in cartilage. Chondroitin sulfate, keratin sulfate, and hyaluronan are the primary glycosaminoglycan (GAG) side chains in cartilage. Chondroitin sulfate and keratin sulfate have negatively charged sulfate and carboxyl groups that make the chains anionic overall. Hyaluronan is not sulfated and interacts with aggrecan and link proteins to form large aggregates that are immobilized in the extracellular matrix and which consequently stabilize the extracellular matrix. Since these charged proteoglycans are immobilized within the extracellular matrix, the resulting charge is referred to as the “fixed charge density.” The proteoglycan distribution, and therefore the distribution of fixed charge density, varies through the cartilage depth. Aggrecan level is the lowest in the superficial zone and increases with depth [8].

Material Properties

The structure of articular cartilage produces complex material behavior (Fig. 3, right panel). Because cartilage structure and composition vary with depth, material properties also vary with depth [10–13]. The material behavior of cartilage varies between species within the same joint, between joints within species, and spatially within each joint within each species [14–16]. Cartilage exhibits nonlinear material behavior in both tension and compression. Under uniaxial tensile stress, cartilage material behavior is primarily determined by the collagen fibrils. The stress–strain curve in tension exhibits a toe region followed by an approximately linear region due to the uncrimping of collagen fibers followed by loading of straightened fibers [8]. Under uniaxial compressive stress, the material response of cartilage is governed by the proteoglycan matrix and fluid flow. The modulus of cartilage in tension is approximately one to two orders of magnitude lower than in compression, a discrepancy known as tension–compression nonlinearity [16]. This characteristic is important for most modes of cartilage deformation that are relevant to whole-joint mechanics.

Fluid–solid interactions, the intrinsic viscoelasticity of the solid phase, and fixed charge density causes flow-dependent viscoelasticity of the cartilage and swelling [17–19]. The cartilage swelling is caused by the fixed charge density of the tissue and charge–charge repulsion between closely packed GAGs attracting interstitial fluid counterions [8]. Collagen primarily bears tensile stress, and thus the fibrillar collagen resists expansion of the solid matrix during swelling [20].

Solutes, including nutrients and metabolic by-products, move through cartilage via diffusion. Solute diffusivity in cartilage is smaller than in aqueous solution [8], and diffusivity decreases as the tissue is compressed [21, 22]. The size of the solute also influences solute diffusivity. For large solutes, cyclic loading can enhance diffusion but it has no effect on small solutes [23].

Mechanics of the Hip

The kinematics (motion) of the hip are primarily determined by the congruency of the articular surfaces, their shape and curvature, the limits of motion imposed by bony contact between the femur and acetabulum or labrum, and the joint reaction forces related to musculature and body weight. The stress at the articular cartilage layers and the cartilage contact area during articulation are determined by joint congruency, curvature, chondral thickness and material properties, and joint load. Local joint congruency, defined as the congruency of the two articulating surfaces in the region of a particular point of contact, is in turn a function of joint kinematics.

The magnitude and orientation of the abductor muscle force as well as the distance from the joint center counteracts the force from body weight, so that the overall joint reaction forces across the hip are determined by the sum of these two oppositely directed forces. Typical joint reaction forces during single-leg stance are approximately three times the body weight but vary as a function of activity. The study by Bergman et al. is the most widely quoted report on in vivo hip joint reaction forces [24]. These investigators measured hip joint reaction forces during activities of daily living in patients who had received a total hip replacement with a telemeterized load cell. Joint reaction forces ranged from 2.4 times body weight during level walking to 2.6 times body weight when walking downstairs. Independent of other factors, joint reaction forces are decreased by medializing the center of rotation. For example, this can occur nonsurgically by the use of a cane in the contralateral hand, or surgically via lateralization of greater trochanter, which moves the relative point of application of the abductor muscles farther from the joint center.

Although the normal hip is generally considered to be a highly congruent joint, the local congruency varies with changes in joint orientation, such as flexion–extension or abduction–adduction. Further, congruency is often altered in joints with pathomorphologies such as acetabular dysplasia and FAI. For a given joint reaction force, higher

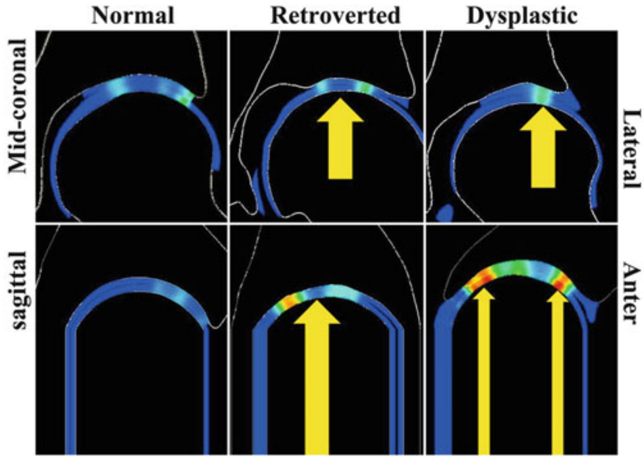


Fig. 4 Cartilage contact pressure in coronal (*top*) and sagittal (*bottom*) slices of a representative normal hip (*left*), retroverted hip (*middle*), and dysplastic hip (*right*). Normal hips exhibited centered, distributed loading (*left column*). Contact in retroverted hips was moved medially and superiorly with respect to normal hips (*middle column*). In dysplastic hips, loading was shifted laterally, and in comparison to normal hips, a significantly higher percentage of load was supported by the acetabular labrum [25, 30]

congruency generally implies a larger contact area and thus lower contact stresses. If the global congruency is defined as, for instance, the congruency of a pair of spheres to the articular surfaces, dysplastic hips have a lower global level of congruency than normal hips. However, this does not necessarily translate into higher local contact stresses since local congruency is not significantly different between normal and dysplastic hips [25].

Contact Patterns

Areas of contact on the articular surfaces vary somewhat with activity and motion during the activity in both normal and pathomorphologic hips. In the normal hips, the direction of loading and the location of chondral contact change from predominantly superior–posterior during ascending stairs, to more superior during walking, to superior–anterior during descending stairs [26]. Predicted peak stress in normal hips ranges from 7.52 ± 2.11 MPa for heel-strike during walking (2.3 times the body weight) to 8.66 ± 3.01 MPa for heel-strike during descending stairs (2.6 times the body weight). Across all activities of daily living, the contact area of the femur on the acetabular cartilage occupies about a third of the total surface area. Even in the normal hips, the distribution of contact stress is highly nonuniform due to local incongruities between the femoral and acetabular cartilage. In addition, contact stress varies more between different subjects for a single activity than between different activities for a single subject.

In the hips with acetabular dysplasia, the acetabulum is shallow. Dysplasia is typically diagnosed radiographically by an anterior and/or lateral center edge angle (CEA) less than 20–25° and an acetabular index greater than 10°. These measures are indicative of a shallow acetabulum and an upwardly sloping sourcil, respectively. Patient-specific finite element (FE) predictions of contact area show that only the superior region of the acetabulum exhibits significantly different labral contact areas when comparing normal and dysplastic subjects [25] (Fig. 4). This suggests that during activities of daily living, the superior labrum in the dysplastic hip is loaded more than other portions of the acetabular labrum. Predictions of labral load support corroborate this finding – the labrum in dysplastic hips supports loads that are 2.8–4.0 times larger than that of normal hips. These results are consistent with clinical observations of labral hypertrophy, as well as the superior or anterosuperior location of labral tears in the dysplastic hip [27–29].

In the hips with acetabular retroversion, the acetabulum opens more posterolaterally than normal. This is recognized on anteroposterior radiographs by the presence of a crossover sign, which indicates a prominent anterior acetabular wall, a deficient posterior acetabular wall, or both. Cartilage contact in retroverted hips is focused in the superomedial acetabulum, whereas in normal hips, contact patterns are distributed over more of the entire acetabulum (Fig. 4) [30]. Additionally, retroverted subjects tend to have patches of more medial contact.

Mechanical Causes of Articular Cartilage Defects

Focal cartilage defects often result from cartilage delamination, typically due to shear loading that causes failure at the osteochondral interface. In vitro, both articular surface contact stress and maximum shear stress have been shown to predict cartilage fissuring under impact loads [31, 32]. The maximum shear stress during activity for the normal hip tends to occur at the interface of the acetabular cartilage and subchondral bone, near the junction of the cartilage with the labrum [33].

For a specific joint, chondral defects greater than a certain size tend to increase in size, generate an inflammatory response in the joint, and ultimately lead to OA, while other smaller defects will remain stable and not progress to disease [34]. Furthermore, a number of geometric factors contribute to the variability in clinical success treating osteochondral defects, as these factors often produce unfavorable conditions for the formation of new cartilage or the survival of implanted plugs, scaffolds, or cells [35]. The major geometric factors are defect size, joint curvature, joint congruence, cartilage thickness, and status of the rim of the defect. Increased joint curvature generally causes the rim of the defect to experience higher stresses and strains during joint loading and articulation. This is further exacerbated if the joint surfaces are incongruent. Thinner regions of articular cartilage have less ability to increase the local congruency during deformation since deformation is limited by the reduced thickness. And, finally, if the rim of the defect is well defined and intact, the defect will be more likely to remain stable and not progress in the joint, whether it is repaired or not.

As illustrated in the upper panels of Fig. 5 for a defect in the femoral condyle of the knee, increasing defect size exposes the rim of the defect to higher stresses and strain. Furthermore, in larger defects, healing neocartilage or implanted plugs or cells are more exposed to deformation during healing, resulting in a progressively worse outcome independent of other factors. Because the congruency of diarthrodial joints can vary as a function of joint kinematics, a defect may be subjected to different degrees of loading as a function of joint articulation. This is illustrated in the lower panels of Fig. 5 for the femoral condyle of the knee as a function of knee flexion.

Clinical Characteristics of Hip Cartilage Defects

There is no single history or physical examination finding that is pathognomonic for cartilage damage or chondral defects in the hip. There are, however, some predictable patterns of injury. Byrd reported a series of athletes who had chondral damage after lateral impact events, sustaining a blow to the greater trochanter during a fall [7]. Because younger patients typically have little soft tissue to absorb the force of an impact, the force is transmitted to the central joint surface. The cartilage subsequently fails at the medial aspect of the femoral head. There may also be a corresponding lesion in the weight-bearing portion of the acetabulum just above the fossa, which may result from chondrocyte compression [7]. Parafoveal chondral defects have been described in athletes with FAI that participate in sports that require rapid lower extremity flexion, torsion, and force. The lesion seems to occur from impingement-induced instability and translation, with incongruent hip motion causing high shear stresses and cartilage deformation [4]. Similarly, athletes who have a distinct hip subluxation or dislocation can have chondral injuries and ligamentum teres tears around the fovea, as well

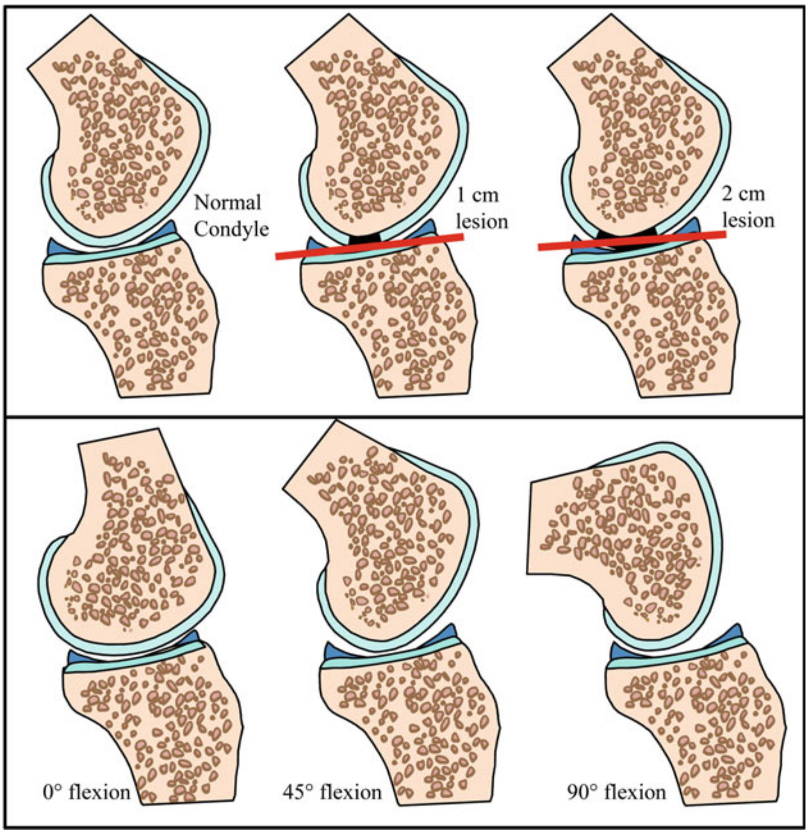


Fig. 5 Effects of defect size and joint curvature on focal cartilage defects, shown for the femoral condyle of the knee joint. *Top panels* – as defect size increases, the rim of the defect and healing tissue within the defect is subjected to increasingly high strains during normal joint loading. *Bottom panels* – joint congruency and curvature often vary as a function of joint orientation. These changes have implications for the effective strains that a defect will experience. As knee flexion increases, congruency of the articular surfaces and, thus contact area, decreases while contact stresses and strains increase

as posterior labrum and cartilage injury [6]. Finally, about 25 % of patients with a history of Perthes' disease in childhood have full-thickness chondral defects [3]. Regardless of the proposed etiology, patients report some combination of sharp groin or buttock pain, stiffness, clicking, popping, or catching [7, 36]. These symptoms are also characteristic of labral tears, and patients with chondral defects frequently have a coexisting labral tear [36].

No single exam maneuver is specific for chondral pathology. The location of the lesion may influence the presence and nature of pain with weight-bearing or with specific exam maneuvers. Pain with hip "logrolling" usually indicates synovitis and/or a hip joint effusion. A full hip examination including stance, gait, range of motion, strength, location of specific sites of tenderness, and provocative maneuvers including apprehension and impingement testing should be performed. It is important to evaluate for FAI or dysplasia, labral tears, compensatory tendinopathies, and compensatory gait patterns. Both FAI and dysplasia can cause or exacerbate chondral damage and should be addressed if surgery is planned.

All patients should have radiographs of the hip to evaluate for FAI, dysplasia, joint incongruity, and early arthritic changes. A CT scan with version analysis is helpful for further evaluation of FAI or dysplasia, but does not image the cartilage directly. Cartilage defects are best seen with a high quality MRI or MR arthrogram of the hip. The MRI should be performed with a 1.5T or 3T magnet and small-field-of-view coil. A combination of coronal, sagittal, axial, and radial images best characterizes the location, size, and nature of the defect. Cartilage imaging is, however, notoriously difficult. MRA is more effective for detecting labral tears than cartilage damage, with sensitivity for chondral defects ranging from 47 % to 81 %, specificity ranging from 66 % to 89 %, and accuracy of 67 % [36, 37]. Bony edema adjacent to the defect can indicate recent trauma [7], or local overload [36].

Treatment Options

The treatment goals for cartilage restoration surgery are the resolution of symptoms, return to activity, and prevention of progressive damage. To achieve this, a preoperative plan is essential, and the nature of the lesion including size, location, underlying etiology, or associated structural pathoanatomy must be known. Indications for treating a focal chondral defect in the hip include acute trauma with an unstable fragment, continued pain and symptoms despite conservative management, a visible chondral defect on preoperative imaging with a positive response to a diagnostic intra-articular injection, and intra-articular loose bodies. The timing of surgery depends on the type of lesion, age of the patient, and any previous interventions. Of note, patients who have loose bodies or unstable fragments should undergo surgery in a more urgent fashion because of the potential for severe cartilage damage from a loose fragment within the joint.

Treatment options for cartilage defects include conservative measures like activity modification, weight management, and viscosupplementation as well as surgical techniques like microfracture, second-generation ACI techniques, and osteochondral allografts (Figs. 6, 7, and 8) [38, 39]. Specific technique chapters follow for each type of cartilage restoration surgery; the discussion of each type of intervention in this chapter will focus on the basic science rationale for each technique and any published outcomes for the hip.

Nonoperative Management

For many patients with chondral defects, a trial of nonoperative measurement is appropriate. This often consists of activity modification or weight management to decrease the mechanical load across the damaged area of cartilage. Physical therapy focusing on hip and core strengthening may also help improve the muscular dynamics around the hip, particularly if there is a component of instability [40, 41]. Patients frequently ask about nutritional supplements. The most well known of these is a combination of glucosamine and chondroitin. Individual trials have shown mixed results, and in meta-analysis, there was no clinically relevant effect on joint pain or joint space narrowing [42].

Intra-articular cortisone injections may be used to reduce symptoms from inflammation. However, each injection has some risk of infection, soft tissue weakening, and possibly a microscopic effect on the cartilage [43]. Multiple cortisone injections should not be the definitive management strategy for young patients who are potential candidates for cartilage restoration procedures. Platelet-rich plasma (PRP) has also been touted as a possible treatment for cartilage defects or osteoarthritis. Advocates for intra-articular PRP injections promote it as a topical application of biological factors that promote healing of the defect. However, preparations of PRP vary and although the effective agent has been proposed, it is not definitively known [44]. There are two studies of PRP injection for hip arthritis; other studies of intra-articular PRP are for the knee or for the talus. Most patients have some initial improvement with gradual worsening over time [45]. Hyaluronic acid or viscosupplementation improves symptoms by a combination of antiinflammatory effects, restoration of the viscosity of synovial fluid, and normalization of synovial cell hyaluronate synthesis [46]. It appears to be safe, without potential adverse effects [46]. The results of hyaluronic acid injections are mixed. In the hip, 45–60 % of people have pain relief 6 months after injection [46].

Primary Repair Versus Arthroscopic Debridement

Primary repair can be considered for an osteochondral fragment especially in the setting of an acute trauma where the fragment should undergo fracture-type healing. If there is a purely chondral flap, the healing is less predictable. Case reports of direct repair have been published. These

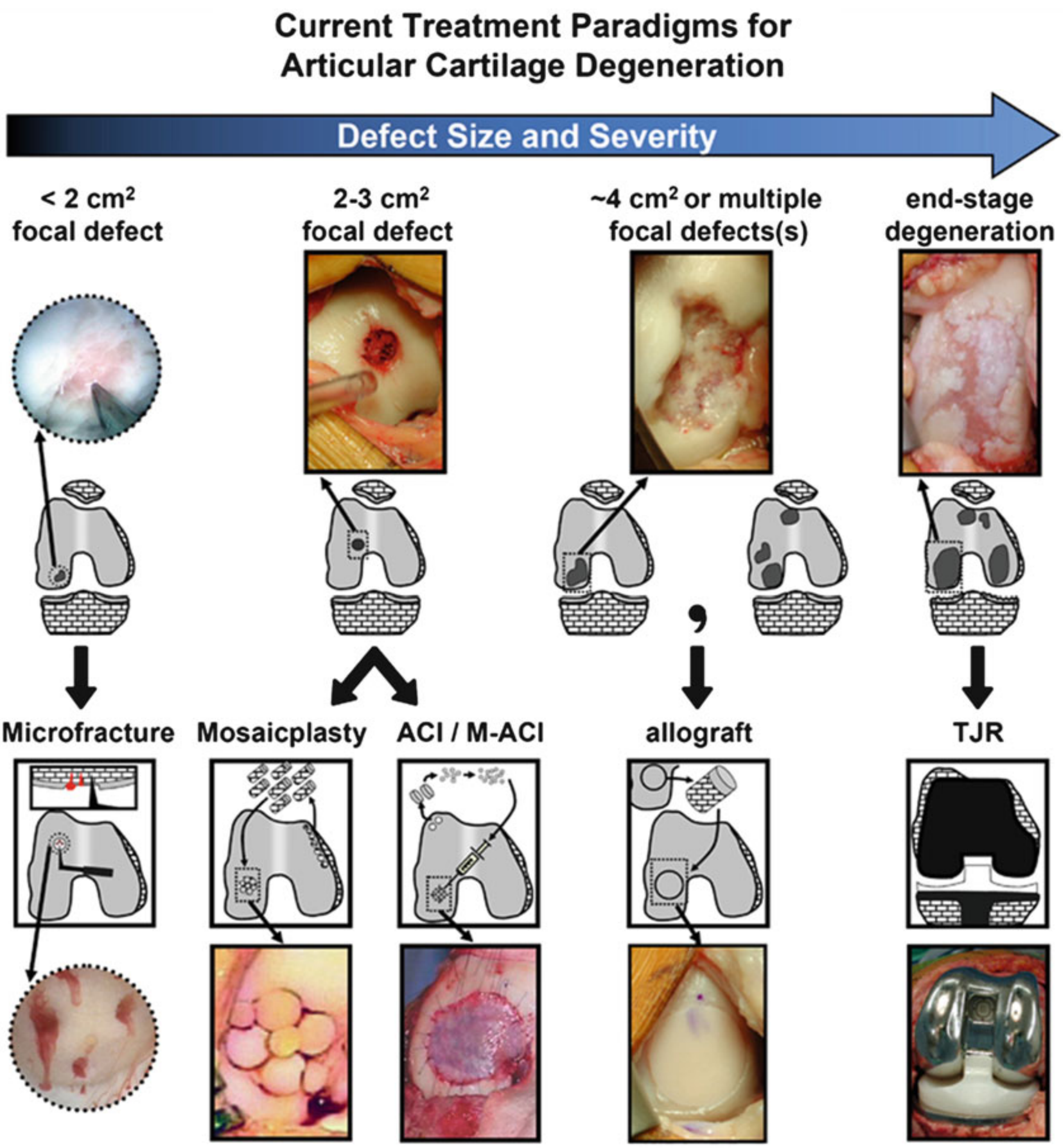


Fig. 6 Currently, treatment decisions for cartilage restoration are based on the size and type of lesion (here, depicted for the knee). Small focal defects (<2 cm^2) are often treated with microfracture. Larger defects (2–3 cm^2) can be treated with osteochondral autograft or cell-based therapies like ACI, MACI, AMIC, or allogenic chondrocytes. Even larger defects (~4 cm^2) are treated with allografts (Reprinted from [39] with permission from Elsevier)

are limited, but the results are reported to be good in the short-term follow-up [47]. Debridement of the flap removes a mechanical irritant and prevents the formation of loose bodies. This may allow symptoms to resolve and permit return to activity or sports [7]. Arthroscopy is definitive for diagnosis of an unstable flap if the preoperative imaging was inconclusive and arthroscopic debridement is often the definitive therapy. Occasionally, however, the lesion is larger than anticipated and a second open cartilage restoration procedure is indicated. Arthroscopic chondral debridement is often used as the comparison procedure for other cartilage

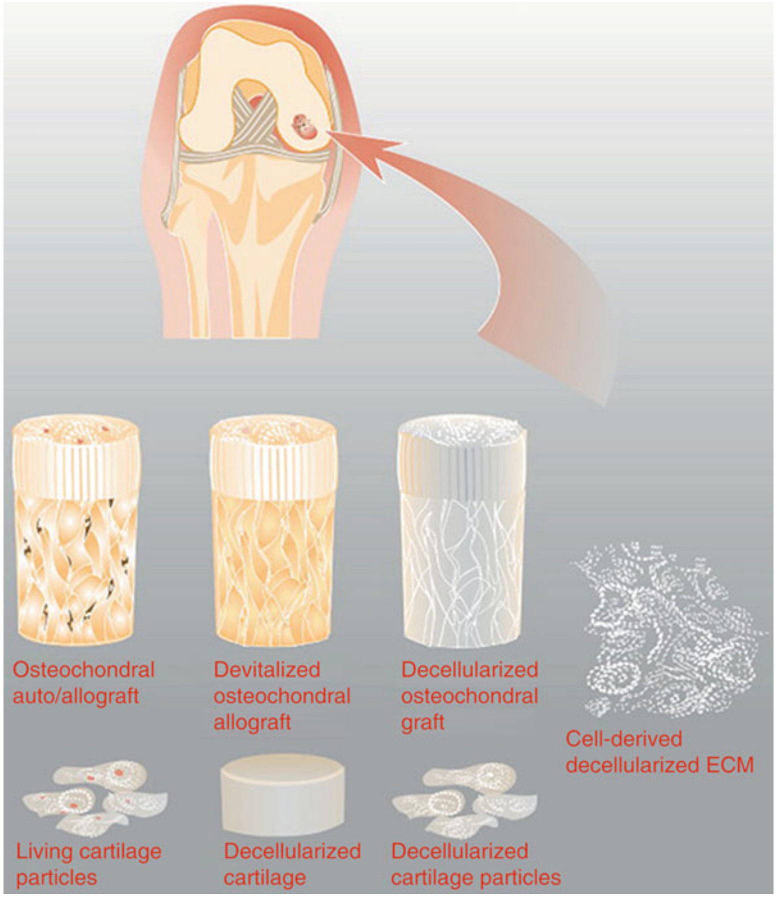


Fig. 7 The treatment strategy for a chondral defect can also be considered in the context of the matrix and scaffold being applied. Currently used grafts may consist of cells and matrix, as in the case of an osteochondral allografts and autografts, and juvenile cartilage particles, or just matrix as is the case with devitalized grafts. In the future, cell-derived decellularized ECM produced in vitro may also be a treatment option (Reprinted from [38] with permission from Elsevier)

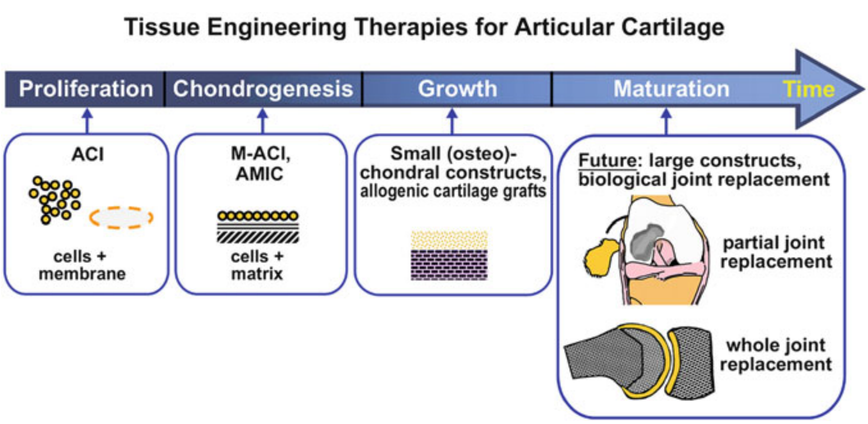


Fig. 8 Developmental progression of tissue-engineering therapies for articular cartilage repair. In general, cartilage repair therapies are based on a strategy of reproducing normal cartilage growth and development. Early strategies like ACI rely on chondrocyte proliferation. Second-generation therapies like MACI or AMIC involve cells implanted in a matrix and covered with a membrane with the goal of producing in situ cartilage. More recently developed therapies include implantation of juvenile cartilage, with the goal of expansion and development into mature adult cartilage tissue. Future directions include development of larger constructs and potentially a biological joint replacement (Reprinted from [39] with permission from Elsevier)

restoration techniques. In the hip, debridement has worse outcomes when compared with autologous chondrocyte implantation (ACI) [48] or microfracture [49].

Microfracture

Microfracture consists of using a surgical awl or drill to penetrate the subchondral bone at the base of a cartilage lesion. The holes in the subchondral plate promote bleeding into the defect, with migration of stem cells and formation of a "superclot," ultimately resulting in the production of reparative fibrocartilage within the defect [50]. For microfracture to successfully produce fibrocartilage, the defect needs to have vertical walls of stable, normal cartilage, creating a "well-shouldered" lesion. This decreases shear and compression forces and protects the clot during healing, which is important because the stability of the clot contributes to the success of the procedure [51]. The advantages of microfracture are that it is technically straightforward, can be performed arthroscopically, and is low-cost. The disadvantage of microfracture compared to other cartilage repair techniques is that it produces less Type II cartilage and has different biomechanical properties than hyaline cartilage, which may make the repair less durable than other techniques. In addition, the overall concentration of mesenchymal cells in the bone marrow is low and their chondrogenic potential declines with age [52].

Microfracture is indicated for full-thickness lesions in patients undergoing concomitant treatment of FAI or dysplasia. Most commonly, these are acetabular rim lesions but, when technically feasible, microfracture can also be used for small femoral head lesions (Fig. 6). It is contraindicated for lesions over 2 cm^2, for patients not willing to endure postoperative non-weight-bearing or rehabilitation, and for bipolar lesions. The results of microfracture for lesions in the hip have generally been reported in combination with treatment for FAI [49]. One study reported better results with microfracture than with debridement, with the greatest improvement seen by 8 weeks with maintenance of the result for up to 12 months [49]. On second-look arthroscopy, patients had fibrocartilage fill of most or all of the defect [49]. Patients with more extensive lesions, however, still progress to arthroplasty and it seems that the technique is limited by the size and extent of the lesion.

Autologous Chondrocyte Implantation (ACI) and Matrix-Associated Chondrocyte Implantation (MACI)

Both ACI and MACI involve harvesting autologous chondrocytes, growing them in culture, and subsequently implanting them [53] (Figs. 6 and 8). MACI and matrix-assisted chondrocyte transplantation (MACT) are second-generation techniques that utilize absorbable scaffolds to support the implanted chondrocytes during healing. Theoretically, ACI and MACI should restore hyaline cartilage at the defect. Unfortunately, both ACI and MACI are two-stage procedures, with a technically demanding second stage performed via a surgical hip dislocation approach. In the original technique, a periosteal patch is used to cover the implanted chondrocytes. Complications related to the periosteum are not infrequent, although most surgeons who perform ACI regularly are now using a synthetic collagen membrane.

ACI and MACI are indicated for symptomatic, unipolar, well-contained defects that are between 2 and 10 cm^2 and with less than 6–8 mm of bone loss. There has only been one outcomes study published for ACI or MACI in the hip. In this series, MACI was better than debridement at mid-term follow-up. It should be noted, however, that all lesions were acetabular [48]. In addition, results tend to be worse when ACI is performed for failed microfracture [54]. When compared to microfracture, the results of ACI tend to be stable or show a trend toward continued improvement. When compared with autograft transplant, the results are similar, but the clinical response tends to be slower [55].

Autologous Matrix-Induced Chondrogenesis (AMIC)

Essentially, AMIC is a second-generation microfracture technique. After the microfracture is performed, the fibrin gel is placed in the defect and a porcine collagen I/III matrix is sewn over the lesion. This protects the clot and allows the mesenchymal stem cells to differentiate [51]. When compared to MACI, AMIC is less expensive and is a single-stage surgery. Furthermore, there is no autograft donor site morbidity. Because the technique involves sewing a membrane over the stabilized superclot, it does require a surgical hip dislocation approach. A modified all-arthroscopic version of the technique has also been published [48].

AMIC is indicated for symptomatic full-thickness chondral and osteochondral lesions in weight-bearing regions. The maximum recommended size is as yet unknown, but a case series of patients who underwent AMIC for femoral head and acetabular lesions measuring more than 2 cm^2 has been published [56]. This series consisted of six patients with a minimum of 1-year follow-up, all of whom had improved short-term function. In the knee, the published results are stable at 1–2 years, with improvements in both outcomes and activity scores [57]. Some patients have developed intralesional osteophytes after AMIC and some patients in each series still progressed to arthroplasty [57].

Osteochondral Autograft

Osteochondral autograft techniques involve transplanting healthy, mature cartilage from a non-weight-bearing part of the hip or knee to the site of a chondral defect (Fig. 7). The transplanted plug undergoes osseous integration with the subchondral bone, and the transplanted cartilage integrates with the adjacent host cartilage via fibrocartilage [58]. The advantage of osteochondral autografting is that it places new mature hyaline cartilage into the defect in a single-stage procedure. Nonetheless, it is limited by donor site morbidity, graft availability, and the potential for dead space between the graft plugs [58].

Osteochondral autografts are indicated for small- to medium-sized focal lesions on the femoral head and acetabulum (2.5–4.0 cm^2) (Fig. 6) [58]. Autografts are contraindicated for patients with avascular necrosis and advanced osteoarthritis and for patients older than 50 [59, 60]. In the hip, there have been several case reports and case series of osteochondral autografts performed for varying indications. These generally have good results in short-term follow-up [61]. Within a larger series of patients with Perthes' disease, four patients underwent autografting, with anecdotally good results [62]. The exception to this trend was a series of patients who underwent autografting for lesions caused by avascular necrosis, who had uniformly poor results [60].

Osteochondral Allograft

Like osteochondral autografts, osteochondral allograft involves a cartilage transplant of intact viable cartilage and underlying subchondral bone into a defect (Fig. 7) [63]. Cartilage is relatively immunoprivileged and has an avascular matrix, which means that the host immune reaction is limited [63]. As part of the healing process, allograft bone becomes necrotic and is reabsorbed via creeping substitution. This provides a scaffold and supports the articular surface during gradual incorporation [64].

Osteochondral allografts are indicated for treatment of larger lesions or for lesions with substantial bone loss (Fig. 6). It has the advantage of precisely restoring the chondral surface architecture in a single-stage procedure with viable hyaline cartilage. It can also be used for large defects with no donor site morbidity [63]. Graft availability can, however, be limited and the grafts can be expensive. There is some risk of rejection, incomplete incorporation, or disease transmission as well. Finally, it can be technically demanding to match or size the allograft intraoperatively. There are only limited clinical reports available for osteochondral allografting in the hip. Patients receiving fresh allografts performed via a surgical dislocation approach for acetabular or femoral head lesions had an average 25-point improvement in their Harris Hip Score at a minimum of 2-year follow-up [65].

Allogenic Cartilage Graft

Allogenic cartilage grafting is the most recently developed cartilage repair technique. With this technique, the allogenic cartilage is a cell carrier and not a structural graft (Figs. 7 and 8). There are two types of allogenic cartilage grafting: morcellized cartilage allograft and allogenic chondrocyte implants. In the former, morcellized juvenile chondrocytes are placed into a defect. The chondrocytes then migrate out of the cartilage cubes and produce extracellular matrix to fill the defect [66]. With allogenic chondrocyte implants, the cartilage is harvested and subsequently enzymatically digested to release and isolate the chondrocytes. The cells are then mixed with alginate to form beads for implantation [67].

The indications for allogenic cartilage grafting are similar to those for ACI: symptomatic unipolar well-contained defects measuring 2–10 cm^2 with less than 6–8 mm of bone loss. Both types of chondrocyte grafting can be performed as a single-stage procedure and the chondrocytes themselves appear to be immunoprivileged via a lack of surface allo-reactivity proteins [67]. In addition, if genetic rather than mechanical factors contributed to the formation of the defect, allogenic cartilage replaces the patient's potentially compromised cells [67]. The disadvantages of chondrocyte grafting are that the complications are similar to those for ACI, there is some risk of disease transmission because it is a donor tissue, and clinical results are limited. There are no clinical reports of allogenic cartilage grafting for the hip. In the knee, there is one case series that reported significant and stable clinical improvement at a 5-year follow-up, but with a 19 % (4/21) failure rate [68].

Summary

There are many different techniques available for cartilage repair or restoration in the hip. When compared with the knee, the published results are limited. There are several reasons for this; however, the awareness of young adult hip disease is increasing and the hip preservation field as a whole is continuing to develop. This is likely to increase the use of cartilage repair techniques for the hip and concomitantly increase the number of published clinical results.

References

1. Matheney T, Kim YJ, Zurakowski D, Matero C, Millis M. Intermediate to long-term results following the bernese periacetabular osteotomy and predictors of clinical outcome: surgical technique. J Bone Joint Surg Am. 2010;92(Suppl 1 Pt 2):115–29.
2. Ng VY, Arora N, Best TM, Pan X, Ellis TJ. Efficacy of surgery for femoroacetabular impingement: a systematic review. Am J Sports Med. 2010;38(11):2337–45.
3. Ross JR, Nepple JJ, Baca G, Schoenecker PL, Clohisy JC. Intraarticular abnormalities in residual Perthes and Perthes-like hip deformities. Clin Orthop Relat Res. 2012;470(11):2968–77.
4. Zaltz I, Leunig M. Parafoveal chondral defects associated with femoroacetabular impingement. Clin Orthop Relat Res. 2012;470(12):3383–9.
5. Ross JR, Zaltz I, Nepple JJ, Schoenecker PL, Clohisy JC. Arthroscopic disease classification and interventions as an adjunct in the treatment of acetabular dysplasia. Am J Sports Med. 2011;39 Suppl:72S–8S.
6. Krych AJ, Thompson M, Larson CM, Byrd JW, Kelly BT. Is posterior hip instability associated with cam and pincer deformity? Clin Orthop Relat Res. 2012;470 (12):3390–7.
7. Byrd JW. Lateral impact injury. A source of occult hip pathology. Clin Sports Med. 2001;20(4):801–15.
8. Mow VC, Gu WY, Chen FH. Structure and function of articular cartilage and meniscus. In: Mow VC, Huiskes R, editors. Basic orthopaedic biomechanics and mechano-biology. 3rd ed. Philadelphia: Lippincott; 2005. p. 181–258.
9. Guilak F, Mow VC. The mechanical environment of the chondrocyte: a biphasic finite element model of cell-matrix interactions in articular cartilage. J Biomech. 2000;33(12):1663–73.
10. Buckley MR, Gleghorn JP, Bonassar LJ, Cohen I. Mapping the depth dependence of shear properties in articular cartilage. J Biomech. 2008;41(11):2430–7.

11. Chen AC, Bae WC, Schinagl RM, Sah RL. Depth- and strain-dependent mechanical and electromechanical properties of full-thickness bovine articular cartilage in confined compression. J Biomech. 2001;34(1):1–12.
12. Schinagl RM, Gurskis D, Chen AC, Sah RL. Depth-dependent confined compression modulus of full-thickness bovine articular cartilage. J Orthop Res Off Publ Orthop Res Soc. 1997;15(4):499–506.
13. Setton LA, Zhu W, Mow VC. The biphasic poroviscoelastic behavior of articular cartilage: role of the surface zone in governing the compressive behavior. J Biomech. 1993;26(4–5):581–92.
14. Athanasiou KA, Agarwal A, Dzida FJ. Comparative study of the intrinsic mechanical properties of the human acetabular and femoral head cartilage. J Orthop Res Off Publ Orthop Res Soc. 1994;12(3):340–9.
15. Demarteau O, Pillet L, Inaebnit A, Borens O, Quinn TM. Biomechanical characterization and in vitro mechanical injury of elderly human femoral head cartilage: comparison to adult bovine humeral head cartilage. Osteoarthr Cartil/OARS Osteoarthr Res Soc. 2006;14(6):589–96.
16. Huang CY, Stankiewicz A, Ateshian GA, Mow VC. Anisotropy, inhomogeneity, and tension-compression nonlinearity of human glenohumeral cartilage in finite deformation. J Biomech. 2005;38(4):799–809.
17. Huang CY, Soltz MA, Kopacz M, Mow VC, Ateshian GA. Experimental verification of the roles of intrinsic matrix viscoelasticity and tension-compression nonlinearity in the biphasic response of cartilage. J Biomech Eng. 2003;125(1):84–93.
18. Mak AF. The apparent viscoelastic behavior of articular cartilage–the contributions from the intrinsic matrix viscoelasticity and interstitial fluid flows. J Biomech Eng. 1986;108(2):123–30.
19. Mow VC, Kuei SC, Lai WM, Armstrong CG. Biphasic creep and stress relaxation of articular cartilage in compression? Theory and experiments. J Biomech Eng. 1980;102(1):73–84.
20. Setton LA, Tohyama H, Mow VC. Swelling and curling behaviors of articular cartilage. J Biomech Eng. 1998;120(3):355–61.
21. Burstein D, Gray ML, Hartman AL, Gipe R, Foy BD. Diffusion of small solutes in cartilage as measured by nuclear magnetic resonance (NMR) spectroscopy and imaging. J Orthop Res Off Publ Orthop Res Soc. 1993;11(4):465–78.
22. Evans RC, Quinn TM. Solute diffusivity correlates with mechanical properties and matrix density of compressed articular cartilage. Arch Biochem Biophys. 2005;442(1):1–10.
23. Mauck RL, Hung CT, Ateshian GA. Modeling of neutral solute transport in a dynamically loaded porous permeable gel: implications for articular cartilage biosynthesis and tissue engineering. J Biomech Eng. 2003;125(5):602–14.
24. Bergmann G, Deuretzbacher G, Heller M, Graichen F, Rohlmann A, Strauss J, et al. Hip contact forces and gait patterns from routine activities. J Biomech. 2001;34(7):859–71.
25. Henak CR, Abraham CL, Anderson AE, Maas SA, Ellis BJ, Peters CL, et al. Patient-specific analysis of cartilage and labrum mechanics in human hips with acetabular dysplasia. J Osteoarthritis Cartilage 2014; 22(2):210–217.
26. Harris MD, Anderson AE, Henak CR, Ellis BJ, Peters CL, Weiss JA. Finite element prediction of cartilage contact stresses in normal human hips. J Orthop Res 2012;30(7):1133–1139.
27. Peelle MW, Della Rocca GJ, Maloney WJ, Curry MC, Clohisy JC. Acetabular and femoral radiographic abnormalities associated with labral tears. Clin Orthop Relat Res. 2005;441:327–33.
28. Fujii M, Nakashima Y, Jingushi S, Yamamoto T, Noguchi Y, Suenaga E, et al. Intraarticular findings in symptomatic developmental dysplasia of the hip. J Pediatr Orthop. 2009;29(1):9–13.
29. Leunig M, Podeszwa D, Beck M, Werlen S, Ganz R. Magnetic resonance arthrography of labral disorders in hips with dysplasia and impingement. Clin Orthop Relat Res. 2004;418:74–80.
30. Henak CR, Carruth ED, Anderson AE, Harris MD, Ellis BJ, Peters CL, et al. Finite element predictions of cartilage contact mechanics in hips with retroverted acetabula. Osteoarthr Cartil/OARS Osteoarthr Res Soc. 2013;21(10):1522–9.
31. Atkinson TS, Haut RC, Altiero NJ. Impact-induced fissuring of articular cartilage: an investigation of failure criteria. J Biomech Eng. 1998;120(2):181–7.
32. Haut RC, Ide TM, De Camp CE. Mechanical responses of the rabbit patello-femoral joint to blunt impact. J Biomech Eng. 1995;117(4):402–8.
33. Henak CR, Ateshian GA, Weiss JA. Finite element prediction of transchondral stress and strain in the human hip. J Biomech Eng. 2014;136(2):021021
34. Schinhan M, Gruber M, Vavken P, Dorotka R, Samouh L, Chiari C, et al. Critical-size defect induces unicompartmental osteoarthritis in a stable ovine knee. J Orthop Res. 2012;30(2):214–20.
35. Kock L, van Donkelaar CC, Ito K. Tissue engineering of functional articular cartilage: the current status. Cell Tissue Res. 2012;347(3):613–27.
36. Neumann G, Mendicuti AD, Zou KH, Minas T, Coblyn J, Winalski CS, et al. Prevalence of labral tears and cartilage loss in patients with mechanical symptoms of the hip: evaluation using MR arthrography. Osteoarthr Cartil/OARS Osteoarthr Res Soc. 2007;15(8):909–17.
37. Keeney JA, Peelle MW, Jackson J, Rubin D, Maloney WJ, Clohisy JC. Magnetic resonance arthrography versus arthroscopy in the evaluation of articular hip pathology. Clin Orthop Relat Res. 2004;429:163–9.
38. Benders KE, van Weeren PR, Badylak SF, Saris DB, Dhert WJ, Malda J. Extracellular matrix scaffolds for cartilage and bone regeneration. Trends Biotechnol. 2013;31(3):169–76.

39. Williams GM, Chan EF, Temple-Wong MM, Bae WC, Masuda K, Bugbee WD, et al. Shape, loading, and motion in the bioengineering design, fabrication, and testing of personalized synovial joints. J Biomech. 2010;43(1):156–65.
40. Leite DX, Vieira JMM, Carvalhais VOC, Araújo VL, Silva PLP, Fonseca ST. Relationship between joint passive stiffness and hip lateral rotator concentric torque. Rev Bras Fisioter. 2012;16:414–21.
41. Retchford TH, Crossley KM, Grimaldi A, Kemp JL, Cowan SM. Can local muscles augment stability in the hip? A narrative literature review. J Musculoskelet Neuronal Interact. 2013;13:1–12.
42. Wandel S, Juni P, Tendal B, Nuesch E, Villiger PM, Welton NJ, et al. Effects of glucosamine, chondroitin, or placebo in patients with osteoarthritis of hip or knee: network meta-analysis. BMJ. 2010;341:c4675.
43. Nichols AW. Complications associated with the use of corticosteroids in the treatment of athletic injuries. Clin J Sport Med. 2005;15(5):370–5.
44. Kon E, Filardo G, Matteo BD, Marcacci M. PRP for the treatment of cartilage pathology. Open Orthop J. 2013;7:120–8.
45. Sanchez M, Guadilla J, Fiz N, Andia I. Ultrasound-guided platelet-rich plasma injections for the treatment of osteoarthritis of the hip. Rheumatology (Oxford). 2012;51(1):144–50.
46. van den Bekerom MP, Lamme B, Sermon A, Mulier M. What is the evidence for viscosupplementation in the treatment of patients with hip osteoarthritis? Systematic review of the literature. Arch Orthop Trauma Surg. 2008;128(8):815–23.
47. Tzaveas AP, Villar RN. Arthroscopic repair of acetabular chondral delamination with fibrin adhesive. Hip Int. 2010;20(1):115–9.
48. Fontana A. A novel technique for treating cartilage defects in the hip: a fully arthroscopic approach to using autologous matrix-induced chondrogenesis. Arthrosc Tech. 2012;1(1):e63–8.
49. Haviv B, Singh PJ, Takla A, O'Donnell J. Arthroscopic femoral osteochondroplasty for cam lesions with isolated acetabular chondral damage. J Bone Joint Surg (Br). 2010;92(5):629–33.
50. Steadman JR, Briggs KK, Rodrigo JJ, Kocher MS, Gill TJ, Rodkey WG. Outcomes of microfracture for traumatic chondral defects of the knee: average 11-year follow-up. Arthroscopy. 2003;19(5):477–84.
51. Erggelet C, Endres M, Neumann K, Morawietz L, Ringe J, Haberstroh K, et al. Formation of cartilage repair tissue in articular cartilage defects pretreated with microfracture and covered with cell-free polymer-based implants. J Orthop Res Off Publ Orthop Res Soc. 2009;27(10):1353–60.
52. Payne KA, Didiano DM, Chu CR. Donor sex and age influence the chondrogenic potential of human femoral bone marrow stem cells. Osteoarthr Cartil/OARS Osteoarthr Res Soc. 2010;18(5):705–13.
53. Brittberg M, Lindahl A, Nilsson A, Ohlsson C, Isaksson O, Peterson L. Treatment of deep cartilage defects in the knee with autologous chondrocyte transplantation. N Engl J Med. 1994;331(14):889–95.
54. Pestka JM, Bode G, Salzmann G, Sudkamp NP, Niemeyer P. Clinical outcome of autologous chondrocyte implantation for failed microfracture treatment of full-thickness cartilage defects of the knee joint. Am J Sports Med. 2012;40(2):325–31.
55. Harris JD, Siston RA, Pan X, Flanigan DC. Autologous chondrocyte implantation: a systematic review. J Bone Joint Surg Am. 2010;92(12):2220–33.
56. Leunig M, Tibor LM, Naal FD, Ganz R, Steinwachs MR. Surgical technique: second-generation bone marrow stimulation via surgical dislocation to treat hip cartilage lesions. Clin Orthop Relat Res. 2012;470(12):3421–31.
57. Gille J, Schuseil E, Wimmer J, Gellissen J, Schulz AP, Behrens P. Mid-term results of Autologous Matrix-Induced Chondrogenesis for treatment of focal cartilage defects in the knee. Knee Surg Sports Traumatol Arthrosc Off J ESSKA. 2010;18(11):1456–64.
58. Lane JG, Healey RM, Chen AC, Sah RL, Amiel D. Can osteochondral grafting be augmented with microfracture in an extended-size lesion of articular cartilage? Am J Sports Med. 2010;38(7):1316–23.
59. Gomoll AH, Filardo G, de Girolamo L, Espregueira-Mendes J, Marcacci M, Rodkey WG, et al. Surgical treatment for early osteoarthritis. Part I: cartilage repair procedures. Knee Surg Sports Traumatol Arthrosc Off J ESSKA. 2012;20(3):450–66.
60. Rittmeister M, Hochmuth K, Kriener S, Richolt J. Five-year results following autogenous osteochondral transplantation to the femoral head. Orthopade. 2005;34(4):320, 322–6.
61. Nam D, Shindle MK, Buly RL, Kelly BT, Lorich DG. Traumatic osteochondral injury of the femoral head treated by mosaicplasty: a report of two cases. HSS J. 2010;6(2):228–34.
62. Anderson LA, Erickson JA, Severson EP, Peters CL. Sequelae of Perthes disease: treatment with surgical hip dislocation and relative femoral neck lengthening. J Pediatr Orthop. 2010;30(8):758–66.
63. Bugbee WD, Convery FR. Osteochondral allograft transplantation. Clin Sports Med. 1999;18(1):67–75.
64. Williams SK, Amiel D, Ball ST, Allen RT, Tontz Jr WL, Emmerson BC, et al. Analysis of cartilage tissue on a cellular level in fresh osteochondral allograft retrievals. Am J Sports Med. 2007;35(12):2022–32.
65. Kosashvili Y, Raz G, Backstein D, Lulu OB, Gross AE, Safir O. Fresh-stored osteochondral allografts for the treatment of femoral head defects: surgical technique and preliminary results. Int Orthop. 2013;37(6):1001–6.

66. Almqvist KF, Wang L, Wang J, Baeten D, Cornelissen M, Verdonk R, et al. Culture of chondrocytes in alginate surrounded by fibrin gel: characteristics of the cells over a period of eight weeks. Ann Rheum Dis. 2001;60(8):781–90.
67. Gomoll AH, Filardo G, Almqvist FK, Bugbee WD, Jelic M, Monllau JC, et al. Surgical treatment for early osteoarthritis. Part II: allografts and concurrent procedures. Knee Surg Sports Traumatol Arthrosc Off J ESSKA. 2012;20(3):468–86.
68. Dhollander AA, Verdonk PC, Lambrecht S, Verdonk R, Elewaut D, Verbruggen G, et al. Midterm results of the treatment of cartilage defects in the knee using alginate beads containing human mature allogenic chondrocytes. Am J Sports Med. 2012;40(1):75–82.

88 Surgical Technique: Arthroscopic Microfracture of Acetabular Articular Cartilage Lesions

James E. Voos, Struan H. Coleman, Richard W. Kang, and Nicholas C. Barnthouse

Contents

J.E. Voos (✉)
Division of Sports Medicine, Department of Orthopaedic Surgery, University Hospitals Case Medical Center, Cleveland, OH, USA
e-mail: jvoos@osmckc.com

S.H. Coleman
Sports Medicine, Hospital for Special Surgery, New York, NY, USA
e-mail: ColemanS@HSS.edu

R.W. Kang
Sports Medicine and Shoulder Surgery, Hospital for Special Surgery, New York, NY, USA
e-mail: RWKang.MD@gmail.com

N.C. Barnthouse
School of Medicine, University of Kansas City, Kansas City, KS, USA
e-mail: NBarnthouse@gmail.com

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_97

Abstract

Acetabular articular cartilage lesions can occur as a result of femoroacetabular impingement, traumatic subluxation or dislocation, labral tears, loose bodies, osteonecrosis, osteochondritis dessicans, slipped capital femoral epiphysis, hip dysplasia, or early degenerative joint disease. Cartilage lesions cause significant morbidity, particularly in athletes and active patients. Microfracture techniques in the setting of hip arthroscopy have been developed recently. The ideal patient is a young (<50–60 years old), active patient willing to follow the postoperative rehabilitation protocol. The ideal lesion to treat with microfracture is well contained, focal (<2–4 cm), Outerbridge grade IV, and with intact subchondral bone. Surgical outcomes in small case series have shown promising clinical outcomes.

Introduction

The use of hip arthroscopy to treat both intra-articular and extra-articular pathology of the hip has become increasingly more common with the advent of improved arthroscopic equipment and techniques [1]. Many causes of hip pain in younger patients and athletes have been identified and include femoroacetabular impingement (FAI), labral pathology, ligamentum teres injury, iliopsoas and iliotibial band bursitis, osteochondritis dissecans, and chondral lesions of both the femoral and acetabular components of the hip joint [2]. Articular cartilage abnormalities are the second most common pathology encountered in hip arthroscopy [1]. Acetabular articular cartilage lesions can cause significant morbidity, particularly in athletes and active patients. The goals associated with the use of hip arthroscopy are to alleviate pain and reduce joint instability, but also to potentially prevent further articular cartilage damage and progression to end-stage osteoarthritis later in life [3, 4]. Chondral lesions of the hip can be classified as acute, chronic, or degenerative and can be caused by a variety of factors, including femoroacetabular impingement (cam-, pincer-, or mixed-type FAI), traumatic subluxation or dislocation, labral tears, loose bodies, osteonecrosis, osteochondritis dissecans, slipped capital femoral epiphysis, and hip dysplasia as well as degenerative joint disease resulting in more diffuse cartilage damage [1–5].

Further Descriptions of Mechanism of Injury: FAI

Femoroacetabular impingement (FAI) is one of the most common causes of acetabular articular cartilage injury in athletes. FAI can be divided into cam- and pincer-type FAI, each having its own characteristic injury pattern. Cam impingement is characterized by pathologic contact between an abnormally shaped femoral head and neck with a normal acetabulum, while pincer impingement results from contact between an abnormal acetabulum (commonly retroverted or protruded) and a normal femoral head and neck [4]. In cam impingement, the acetabular chondral rim is particularly at risk because as the hip flexes and internally rotates, the abnormally shaped femoral head-neck junction contacts the anterosuperior aspect of the acetabulum and the resultant compressive and shear forces cause a focal articular cartilage defect [4–6]. The labrum can be torn in severe cases of cam impingement, although labral pathology is more commonly observed in pincer impingement [4]. Acetabular cartilage damage in pincer impingement tends to be shallower and more generalized about the superior aspect of the rim [4]. The posteroinferior acetabulum can be injured in pincer impingement through a "contrecoup" mechanism in which repeated impingement of the anterior femoral head on the abnormally shaped acetabulum creates a moment arm acting on the posteroinferior aspect of the acetabulum [7].

Necessity of Microfracture Technique

Although commonly caused by concomitant pathology, such as femoroacetabular impingement, chondral lesions of the acetabulum will often not improve once the underlying pathologic mechanism has been corrected, as evidenced by second-look arthroscopy following the index procedure [3]. This is thought to be due to the limited healing capacity of articular cartilage regardless of the etiology of injury [7]. This concept points out the need for joint-preserving treatments that specifically address the chondral lesions of the hip, not just the causative pathology. Similar to surgical techniques employed to restore damaged articular cartilage of the knee, several treatments exist for chondral lesions of the hip. These procedures include microfracture, chondral resurfacing, osteochondral drilling, autologous chondrocyte implantation (ACI), and osteochondral allograft and autograft transplantations, among others [3, 7, 8].

Indications

The indications for microfracture of the hip are similar to those employed in the knee. Patient selection is a critical factor in the procedure to assure a successful result [5]. The ideal patient is a young (<50–60 years old), active patient willing to follow the postoperative rehabilitation protocol required once the procedure is complete [1, 4, 5, 7]. The ideal lesion is well contained, focal (<2–4 cm), Outerbridge grade IV, and with intact subchondral bone [1, 5]. Patients with focal areas of chondral loss as a result of degenerative joint disease may be considered. Patients with diffuse degenerative changes, inflammatory arthritis, and bipolar lesions are contraindicated.

Microfracture Concept and Brief Description of Procedure

The microfracture technique has been used with success in the treatment of full-thickness articular cartilage defects of the knee, and its application in the treatment of chondral lesions about the hip relies on the same basic principle. Microfracture is a marrow-stimulating procedure that enables the migration of undifferentiated stem cells into the chondral defect [4, 5]. This technique involves the creation of microfracture perforations in the subchondral bone underlying the calcified cartilage (deep) zone of articular cartilage and the subsequent formation of a marrow clot at the location of the chondral defect [9]. In addition to the potential source of nutrients and growth factors supplied by the subchondral defect, the marrow clot is packed with mesenchymal stem cells and pluripotent marrow cells, which differentiate and promote fibrocartilage tissue formation at the site of injury [4, 5, 9]. This technique relies on an intact cartilage margin surrounding the lesion in order for adequate pooling and adherence of the marrow clot. Therefore, microfracture is intended for the treatment of small, focal chondral lesions, and many surgeons apply stringent limits on the size of defects in which to employ microfracture [9].

Initial Descriptions of Hip Microfracture (Open and Arthroscopic Procedures)

Microfracture of the hip has been described using an open or mini-open technique, in which a small 4-cm Smith-Petersen approach is used to gain access to the anterior hip joint. However, hip arthroscopy has generally been adopted as a less invasive alternative to the open technique, which is now reserved for more extensive procedures such as osteochondral allograft transplantation or autologous chondrocyte implantation [3, 4]. After proper patient positioning and portal placement, the arthroscopic microfracture technique begins with a complete diagnostic examination of the hip joint, with the goals of identifying and classifying the chondral lesion based on location and extent of involvement [4]. The Outerbridge classification system is a commonly used system to classify articular cartilage lesions about the knee and has been revised to apply to chondral defects of the hip [6]. Chondral lesions are classified intraoperatively based on the depth of cartilage erosion in relation to the underlying subchondral bone. The Outerbridge classification is defined as follows: grade 0, normal cartilage; grade I, cartilage with softening and swelling; grade II, partial-thickness defect with fissures on the surface that do not reach subchondral bone; grade III, full-thickness defect with a maximum width <30 % of the distance from the acetabular edge to the fovea; and grade IV, full-thickness defect with a maximum width >30 % of the aforementioned distance [6, 10]. Microfracture is generally warranted for Outerbridge grade IV lesions with a surface area <400 mm^2 in patients who are candidates for arthroscopy [6, 7, 9]. If a full-thickness lesion is identified on diagnostic arthroscopy, loose cartilage flaps are debrided with an arthroscopic shaver or curette to create a stable peripheral margin [7]. The size of the lesion is then measured and a curette is used to remove the calcified cartilage layer at the base of the defect. Upon exposure of subchondral bone, an awl is used to create microfracture holes that will eventually lead to the formation of a marrow clot in the debrided footprint of the chondral defect [9]. Microfracture through the subchondral bone is confirmed based on the visualization of bleeding and fat droplets escaping from the marrow cavity [5]. For partial-thickness lesions, which have adequate articular cartilage beneath the defect, chondroplasty is preferred [4].

Results

Until recently, microfracture of the hip had not been studied extensively; however, with the increased use of arthroscopy to treat hip pathology, there are now several studies that have investigated the clinical efficacy of microfracture. Byrd et al. [11] were one of the first to validate microfracture in the treatment of chondral lesions of the acetabulum. In a series of 220 hip arthroscopy cases, nine patients were observed to have grade IV chondral lesions of their acetabulum. Of these nine patients, three patients were treated with microfracture for an isolated chondral defect. At a final follow-up of 2 years, the three patients who underwent microfracture were the only patients who returned to an active lifestyle. In a subsequent study conducted by Haviv et al. [6], 29 of 135 patients with full-thickness acetabular chondral lesions underwent microfracture. At a mean follow-up of 22 months, the 29 patients treated with microfracture had significantly higher non-arthritic hip scores (NAHS) than those treated with debridement alone. This study is potentially biased; however, as microfracture was only implemented with lesions <300 mm^2 and no defect size parameters were given for patients receiving debridement only. In a study by Philippon et al. [5], second-look arthroscopy was used to evaluate fibrocartilage formation within the chondral defect after microfracture. In a series of nine patients, with an average full-thickness lesion of 163 mm^2, undergoing revision arthroscopy for a variety of reasons, the mean percent fill of the chondral defect was 91 % at a mean follow-up of 20 months after the index microfracture procedure. In addition to having a full-thickness chondral defect, one of the nine patients in this study had diffuse osteoarthritis at the time of the index procedure, but opted to undergo microfracture to treat the chondral defect in an attempt to return to baseball. At the time of revision arthroscopy 10 months after the index procedure, this patient had a percent fill of only 25 % with progression of diffuse osteoarthritis.

In a study evaluating the efficacy of microfracture in elite athletes, McDonald et al. [2] used return to play as a parameter to compare athletes undergoing microfracture to those undergoing hip arthroscopy with debridement alone. Thirty-nine elite male athletes with Outerbridge grade IV chondral defects underwent hip arthroscopy with microfracture, while 81 elite male athletes underwent hip arthroscopy without microfracture for conditions other than a grade IV chondral defect. In the microfracture group, 30 athletes (77 %) underwent microfracture of the acetabulum, 5 athletes (13 %) underwent microfracture of the femoral head, and 4 athletes (10 %) underwent microfracture of both the acetabulum and femoral head. The average size of the chondral defect in the microfracture group was 162 mm^2. Seventy-seven percent of patients undergoing microfracture returned to play, while 84 % of the patients undergoing hip arthroscopy without microfracture returned to play after

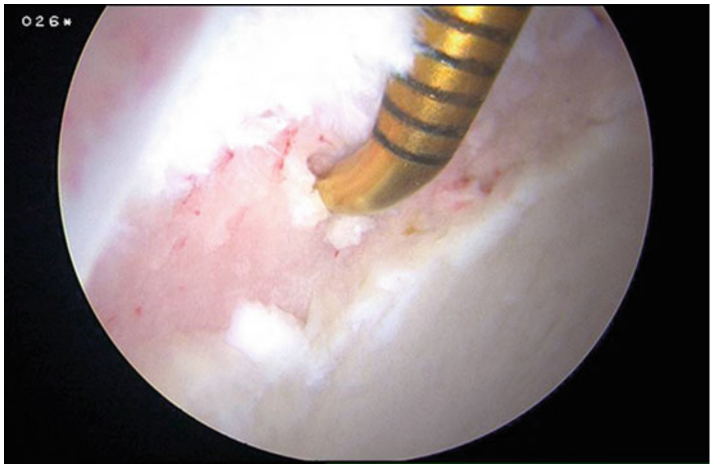


Fig. 1 Microfracture hole in the subchondral bone. A microfracture depth of 2–4 mm is made to access the marrow elements and provide a pathway for release of the underlying mesenchymal stem cells

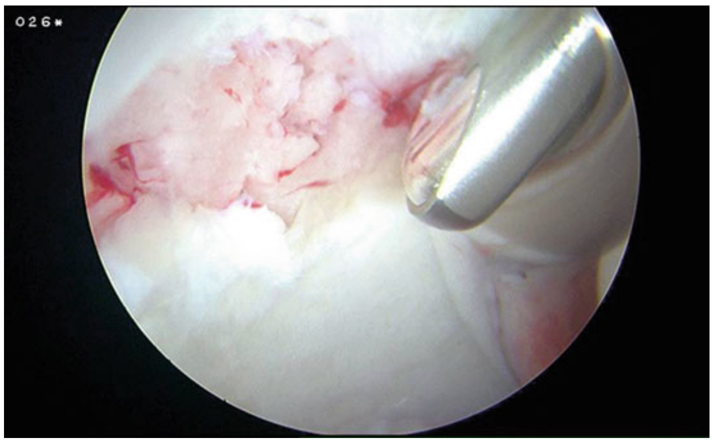


Fig. 2 The pump pressure is decreased to observe the release of blood and fat droplets from the microfracture holes

Fig. 3 Stryker snap caps (*left*) and microfracture guides (*right*). The guides have angled necks including 0°, 45°, 70°, and 90°. The working lengths of the guides are 3.5 and 7 in. in length. Snap caps can be used with the guides and are available in 4-, 5-, 6-, and 7-mm options to allow surgeons to choose an appropriate depth of microfracture (Image courtesy of Stryker, Mahwah, NJ)

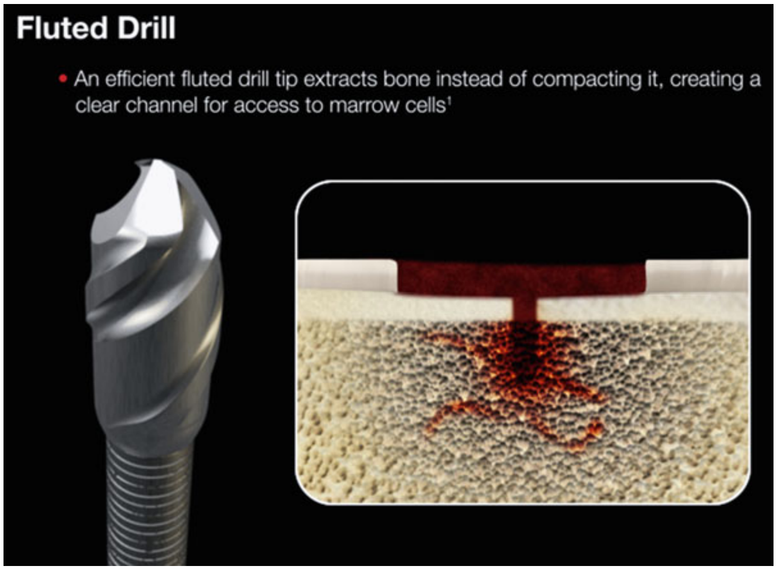


Fig. 4 Stryker drill has a 2-mm tip with flutes to allow for bone extraction rather than compaction. Extracting bone leaves clear channels for the efflux of marrow cells (Image courtesy of Stryker, Mahwah, NJ)

surgery. Horisberger et al. [12] alternatively looked at the use of microfracture in a slightly older patient population with more diffuse acetabular osteoarthritis. In this study, 20 patients with an average age of 47 years old with Outerbridge grade II or higher chondral lesions and associated signs of diffuse acetabular osteoarthritis underwent hip arthroscopy. A subset of these patients with grade IV cartilage defects also underwent microfracture. At a mean follow-up of 3 years, 50 % of the microfracture patients required total hip arthroplasty (THA). The authors recommended that patients with Tonnis grade III osteoarthritis in addition to a focal chondral lesion of the acetabulum should not undergo hip arthroscopy and would most likely require THA at some point.

Fig. 5 Stryker microfracture guide has a mouth that has teeth to reduce the risk of skiving (Image courtesy of Stryker, Mahwah, NJ)

Surgical Technique

The authors perform hip arthroscopy supine on a hip positioning system table (Smith & Nephew, Andover, MA). Once traction is placed on the operative limb, a C-arm is used to confirm that an adequate amount of joint distraction is achieved. Anterior and anterolateral portals are then established. In most cases, these are the only two portals that will be needed for this procedure. The procedure can also be performed in the lateral position.

A diagnostic examination is performed to identify the chondral defect. Microfracture of the chondral defect in the hip follows the same principles as in the knee [9]. Using a combination of a ring curette and mechanical shaver, loose flaps of damaged cartilage are removed to prepare a stable and smooth rim of cartilage around the defect. While maintaining the subchondral plate, a mechanical shaver is used to remove the calcified

Fig. 6 Smith & Nephew XL Microfracture Pick. Note the mid-shaft strike point, which allows for the 90° angle pick to be impacted with a perpendicular force into the subchondral bone (Image courtesy of Smith & Nephew, Andover, MA)

Fig. 7 Close-up of Smith & Nephew XL Microfracture Pick with 90° angle (Image courtesy of Smith & Nephew, Andover, MA)

Fig. 8 Close-up of Smith & Nephew XL Microfracture Pick mid-shaft strike point (Image courtesy of Smith & Nephew, Andover, MA)

cartilage layer. Arthroscopic awls with an angle sufficient enough to be perpendicular to the subchondral surface are then used to create multiple microfracture holes in the subchondral bone. The holes are created with about 3–4 mm of distance from the other holes. A microfracture depth of 2–4 mm is made to access the marrow elements and provide a pathway for release of the underlying mesenchymal stem cells (Fig. 1). Once all the holes have been made, the pump pressure is decreased to observe the release of blood and fat droplets from the holes (Fig. 2). This observation confirms that the holes have been made to a sufficient depth.

Working in the hip presents a few technical challenges that are different than those found in the knee. One of the more difficult obstacles is the ability to access different parts of the hip with an instrument and create microfracture holes that are perpendicular to the cartilage surface. Various companies are vested in the development of devices that can help surgeons overcome these obstacles.

Stryker (Mahwah, NJ) has developed a MicroFX^{TM} OCD system with multiple curette and microfracture guide options to facilitate access to different parts of the hip. Their curettes are ring, pear, and reverse triangle shaped to help create vertical shoulders that are mechanically stable. The guides have angled necks including 0°, 45°, 70°, and 90°. The working lengths of the curettes and microfracture guides are 3.5 and 7 in. in length. Snap caps can be used with the guides and are available in 4-, 5-, 6-, and 7-mm options to allow surgeons to choose an appropriate depth of microfracture (Fig. 3). The drill has a 2-mm tip with flutes to allow for bone extraction rather than compaction. Extracting bone leaves clear channels for the efflux of marrow cells [13] (Fig. 4). The microfracture guide has a mouth that has teeth to reduce the risk of skiving (Fig. 5).

Smith & Nephew (Andover, MA) has developed an XL Microfracture Pick that is added to its hip instruments tray (Fig. 6). The pick has an angle of 90° (Fig. 7). The pick has a mid-shaft strike point allowing for the 90° angle pick to be impacted with a perpendicular force into the subchondral bone (Fig. 8). This mid-shaft strike point also helps minimize the chances of skiving. The shaft is tapered and has a working length of 15.2 in., thus allowing it to be used with a slotted cannula in a standard arthroscopy portal.

Rehabilitation

As in the surgical technique, the postoperative rehabilitation protocol is also adopted from our understanding in the knee [9]. The initial part of the rehab will focus on protecting the marrow clot and achieving range of motion. Cryotherapy is used throughout the initial postoperative phase to facilitate pain relief and mitigate the inflammatory response to surgery. Stationary bicycle exercises without resistance are started in the immediate postoperative phase. Crutch-assisted toe-touch weight bearing is recommended for 6–8 weeks with concomitant use of a continuous passive motion (CPM) machine. Advancement to full weight bearing is then achieved by 8–10 weeks. Once range of motion, strength, and agility have returned, the patient may then return to impact sports, which is typically around 4–6 months postoperatively.

Summary

Microfracture techniques have been developed to treat acetabular articular cartilage lesions. The ideal lesion is grade IV, focal in size, well contained, and with intact subchondral bone. With appropriate patient selection, surgical technique, and careful rehabilitation, good clinical outcomes have been reported.

References

1. Karthikeyan S, Roberts S, Griffin D. Microfracture for acetabular chondral defects in patients with femoroacetabular impingement: results at second-look arthroscopic surgery. Am J Sports Med. 2012;40(12):2725–30.
2. McDonald JE, Herzog MM, Philippon MJ. Return to play after hip arthroscopy with microfracture in elite athletes. Arthroscopy. 2013;29(2):330–5.
3. Jordan MA, et al. Operative treatment of chondral defects in the hip joint: a systematic review. Curr Rev Musculoskel Med. 2012;5(3):244–53.
4. Yen YM, Kocher MS. Chondral lesions of the hip: microfracture and chondroplasty. Sports Med Arthrosc. 2010;18(2):83–9.
5. Philippon MJ, et al. Can microfracture produce repair tissue in acetabular chondral defects? Arthroscopy. 2008;24(1):46–50.
6. Haviv B, et al. Arthroscopic femoral osteochondroplasty for cam lesions with isolated acetabular chondral damage. J Bone Joint Surg Br. 2010;92(5):629–33.
7. Crawford K, et al. Microfracture of the hip in athletes. Clin Sports Med. 2006;25(2):327–35. x.
8. Egerton T, et al. Intraoperative cartilage degeneration predicts outcome 12 months after hip arthroscopy. Clin Orthop Relat Res. 2013;471(2):593–9.
9. Mithoefer K, et al. Chondral resurfacing of articular cartilage defects in the knee with the microfracture technique. Surgical technique. J Bone Joint Surg Am. 2006;88(Suppl 1 Pt 2):294–304.
10. Cameron ML, Briggs KK, Steadman JR. Reproducibility and reliability of the outerbridge classification for grading chondral lesions of the knee arthroscopically. Am J Sports Med. 2003;31(1):83–6.
11. Byrd JW, Jones KS. Osteoarthritis caused by an inverted acetabular labrum: radiographic diagnosis and arthroscopic treatment. Arthroscopy. 2002;18(7):741–7.
12. Horisberger M, Brunner A, Herzog RF. Arthroscopic treatment of femoral acetabular impingement in patients with preoperative generalized degenerative changes. Arthroscopy. 2010;26(5):623–9.
13. Chen H, Sun J, Hoemann CD, Lascau-Coman V, Ouyang W, McKee MD, Shive MS, Buschmann MD. Drilling and microfracture lead to different bone structure and necrosis during bone-marrow stimulation for cartilage repair. J Orthop Res. 2009;27(11):1432–8.

Surgical Technique: Microfracture and Fibrin Glue for Cartilage Delamination

89

Edward D.R. Bray, Samuel J. Barke, Giles H. Stafford, and Richard N. Villar

Contents

Abstract

Injury to articular cartilage can result from femoroacetabular impingement (FAI). Early damage may manifest as peripheral cartilage delamination from subchondral bone. This is demonstrated during hip arthroscopy by the "wave" sign. Studies have shown that within this damaged cartilage there are viable chondrocytes. It is therefore reasonable to consider surgical methods, which preserve rather than excise these areas of chondral damage.

Since the early twentieth century, fibrin has been used in surgery because of its hemostatic and adhesive properties. Consequently, a technique has been developed which uses a combination of the microfracture principals with fibrin glue to adhere delaminated cartilage back to subchondral bone. Fibrin has been shown not only to adhere to cartilage but also to encourage migration and proliferation of chondrocytes to areas of damaged cartilage. This encourages a repopulation of chondrocytes and healthy hyaline cartilage. Studies have shown this method to be at least comparable, if not superior, to traditional cartilage sacrificing techniques such as chondroplasty and microfracture alone.

Introduction

Acetabular cartilage delamination is a frequent finding during open and arthroscopic hip surgeries for femoroacetabular impingement (FAI) [1].

E.D.R. Bray (✉) • S.J. Barke • G.H. Stafford
The EOC, Epsom General Hospital, Epsom, Surrey, England
e-mail: edward.bray@eoc.nhs.uk; sambarke1@doctors.org.uk; giles.stafford@eoc.nhs.uk

R.N. Villar
The Richard Villar Practice, Spire Cambridge Lea Hospital, Impington, Cambridge, UK
e-mail: RNV1000@aol.com

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_98

Delaminated cartilage is macroscopically normal with active live chondrocytes but has been detached from its subchondral bone. In the early stages, the chondrolabral junction remains intact, but with further progression, this may break down and unstable chondral flaps may form, the precursor to fulminant osteoarthritis [2]. During hip arthroscopy, delaminated cartilage is indicated by the "wave" or "carpet" sign, a phenomenon that occurs when applying pressure to the adjacent labral area [3].

Minimally invasive management of articular cartilage injuries takes its origin in arthroscopy of the knee, with a transfer of techniques to hip arthroscopy. There are a number of methods that an arthroscopic hip surgeon can employ [4–6]. A surgeon may wish to manage the cartilage delamination conservatively and just treat the cause, usually FAI [1].

Microfracture is a technique that was first described by Steadman in the knee and then latterly applied to the hip by Philippon. Damaged hyaline cartilage is excised to a stable edge and the layer of calcified cartilage also removed before the microfracture is performed to the exposed subchondral bone in an attempt to stimulate formation of a layer of fibrocartilage. The encouraging results and the ease with which it is performed have resulted in this becoming a popular technique [7, 8].

The microfracture technique does show good results. However, as it requires the removal of living tissue, a novel cartilage preservation technique has been developed. Delaminated cartilage has been shown histologically to contain viable chondrocytes that gain their nutrients from synovial fluid rather than the subchondral bone; it is argued that excision of this valuable tissue matrix is unnecessary. Rather, using fibrin glue to adhere this macroscopically normal cartilage back to the subchondral bone results in conservation of the hyaline cartilage. Furthermore, mesenchymal stem cells that mature into chondrocytes infiltrate the previously delaminated cartilage promoting the development of healthy hyaline cartilage [1, 3]. It is hypothesized that this may lead to a more durable repair.

Fibrin

Fibrin glue or sealant is a unique biological material, which is used topically in surgery for both hemostasis and as an adhesive. It utilizes the final stage of the clotting cascade in which fibrinogen is catalyzed by thrombin to fibrin.

In Germany, Bergel et al. first described the use of fibrin in 1909 as a hemostatic agent [9]. Approximately 30 years later, Young and Medawar used fibrinogen for peripheral nerve attachment [10]. This technique evolved to mix fibrinogen with thrombin to secure skin grafts [11]. Development of Cohn fractionation allowed production of concentrated fibrinogen in the 1970s resulting in more efficacious adhesion and making fibrin glues a more popular among surgeons [12, 13].

In Europe fibrin glues have been used extensively for the last 15 years, but only as before 1998, the United States Food and Drug Administration would not license a fibrinogen-based product because of the risk of blood-borne viruses associated with blood products in the 1980s [14].

There are now many commercially available preparations of fibrin glue. These use two syringes containing fibrinogen and thrombin dissolved in calcium chloride, which are mixed on application creating a fibrin clot. Apoprotein and tranexamic acid act to inhibit fibrinolysis and are added to the mixture in varying concentrations to stabilize the clot and control fibrinolysis. The mixture can either be dripped or sprayed on to the desired area [15–18].

In addition to hemostatic and adhesive effects of fibrin glue, the fibrin matrix has been shown to influence cellular activity resulting in increased proliferation, migration, and survival of chondrocytes. Studies have shown that migration of osteoprogenitor cells and fibroblasts is mediated through the actions of thrombin and fibrinogen on cell surface receptors. Furthermore recruitment of inflammatory cells and chondrocyte precursors, through fibrin and thrombin's actions on cell surface receptors, increases the rate at which damaged tissues heal [1, 3].

Surgical Uses

Fibrin glue is widely used in surgical practice for its hemostatic and adhesive properties. Commercially available preparations include Tisseel (Baxter, UK), Beriplast (Farma-tek, Turkey), and Evicel (Ethicon, USA); these are stored in freezers and must be thawed completely prior to use.

Electrocautery is often utilized to aid hemostasis in surgery; however, there are situations in surgery where this is not appropriate because there may be vital structures including nerves and vasculature near the hemorrhagic area. Fibrin glue is useful as hemostatic adjunct when applied topically to slowly bleeding foci, diffuse parenchymal oozing, and bleeding from needle puncture [19, 20]. Its unique hemostatic properties are utilized in cardiac surgery where there is a propensity to bleed because cardiopulmonary bypass may cause fibrinolysis and platelet destruction [21].

In both peripheral and cardiac vascular surgeries, fibrin glue has been shown to be a superior method in sealing vascular anastomoses and has been shown to stop hemorrhage from the site more effectively than pressure alone [22]. Furthermore, fibrin glue has been used as an adjunct to artificial grafts to attach endothelial cells with the aim of creating a new endothelium. Care must be taken not to inject fibrin glue intravascularly as it will cause thrombosis [23]. Similar to its use to seal vascular anastomoses, fibrin sealant is used in gastrointestinal surgery to reduce rates of bowel anastomotic leaks and in thoracic surgery to seal potential air leaks following lung resection [24].

Hepatobiliary surgery may result in raw areas of liver and spleen that often ooze blood and this can be managed with the application of fibrin glue. Furthermore, in traumatic injuries to the liver and spleen where organs previously had not been salvageable, the use of fibrin glue has allowed the organ to be saved [25, 26].

Its adhesive properties are used both in oncological breast surgery and in plastic surgery. In breast surgery the fibrin glue is used to close potential dead space reducing the risk of painful seroma formation and also reduces the risk of infection [27]. In plastic surgery it has been applied in a number of situations, including as a hemostat following burn debridement and as an adjuvant to attach skin grafts and to reduce dead space following the creation of a muscle flaps [28].

Possible adverse events associated with the use of fibrin glue include surgical site infection, as the fibrin clot acts as a nidus for infection. Anaphylaxis has been associated particularly in bovine derived products. Care should be taken in patients who have previously had anaphylaxis related to plasma products or those with IgA deficiency [29]. As previously discussed vascular thrombosis is a side effect if injected directly into a vessel, though its appropriate use is not associated with an increased risk of vascular thrombosis. As it is a blood product, there is an inherent risk of transmission of blood-borne infection, though this is now minimal.

Uses in Trauma and Orthopaedics

The use of fibrin glue as a hemostat is limited in orthopaedics; it may be used to gain hemostasis during a knee replacement or after periacetabular osteotomy. In orthopaedics it is most commonly used for its adhesive effects or as a scaffold for cell growth. Intra-articular fractures may result in damage to the articular cartilage. Fibrin glue has been used to reattach chondral and osteochondral fragments with the advantage that it causes no further chondral damage [30, 31], and there is no requirement for the removal of metalwork. However, this is thought to be less reliable than internal fixation, and the patient is required to be in plaster for 3 weeks, with which there are increased risks of thrombosis and muscular atrophy [32].

Fibrin glue is used in both autologous chondrocyte implantation (ACI) and matrix-induced autologous chondrocyte implantation (MACI) to secure chondrocyte implantation. ACI is a two-stage process used in patients with isolated areas of chondral damage. Chondrocytes are first harvested and propagated. These chondrocytes are then reimplanted at a second procedure and can be attached to the area of chondral damage using fibrin adhesive [33].

In both ACI and MACI, the primary function of the fibrin glue is as an adhesive, but evidence shows that there is also increased migration and proliferation of chondrocytes. Consequently, fibrin glue has been used to suspend chondrocytes and as a result these can be directly applied to areas of chondral damage; this is simpler than ACI and can be used on areas which are less amenable for traditional ACI [34].

Fibrin glue has been used in meniscal and ligament repair in the knee. However, current direct methods of meniscal repair and the use of hamstring grafts in anterior cruciate ligament repair are very successful without the use of fibrin adhesive. It has therefore been made redundant in these procedures [35].

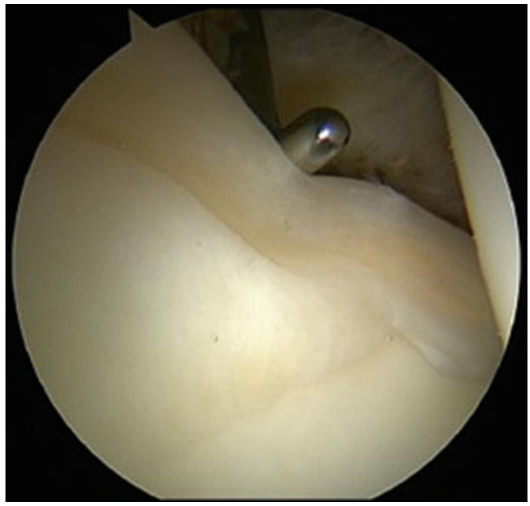


Fig. 1 The wave or carpet sign (Source: Mr. Giles Stafford)

Use in Hip Arthroscopy

Within damaged hyaline cartilage there are viable chondrocytes present [36, 37]. It is possible to secure delaminated cartilage to the subchondral bone below using fibrin glue with good effect [30, 38]. This can therefore be reasonably applied in hip arthroscopy.

Furthermore, fibrin glue has been shown to promote chondrocyte proliferation and migration [39]. Prior to application of the fibrin glue, the senior author described a technique that uses microfracture to the subchondral bone. This allows release of marrow containing mesenchymal stem cells and growth factors into the chondral defect. A combination of the above factors results in a repopulation and proliferation of chondrocytes to a normal level seen in healthy cartilage. It is also hypothesized that microfracture holes also allow a more mechanically stable fibrin clot.

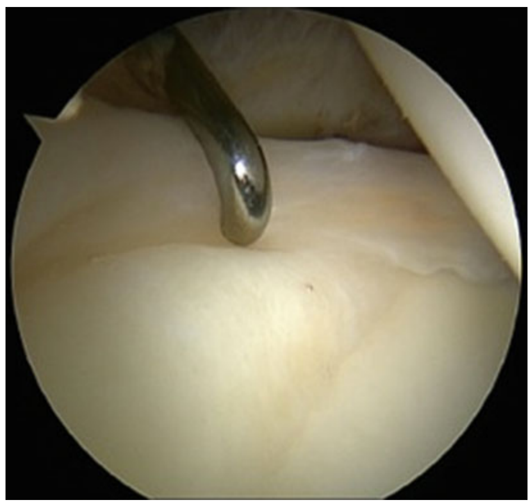


Fig. 2 Stable labrum (Source: Mr. Giles Stafford)

Technique

Areas of delamination with an intact chondrolabral junction are demonstrated by applying peripheral pressure to the labrum, which will lift the adjacent cartilage resulting in the “wave” or “carpet” sign (Fig. 1). After evaluating the stability of the adjacent labrum (Fig. 2), the radiofrequency ablation probe or arthroscopic shaver may be used to develop the perilabral sulcus gaining adequate exposure to the labral base. An arthroscopic knife is used under the labrum to access the area of delamination, termed the “pocket” (Fig. 3).

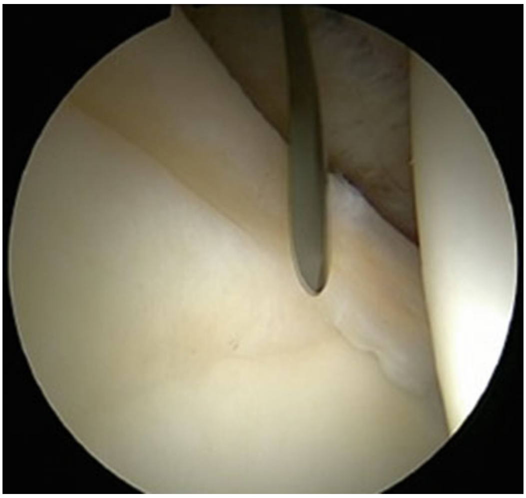


Fig. 3 Arthroscopic knife gaining access to delaminated cartilage (Source: Mr. Giles Stafford)

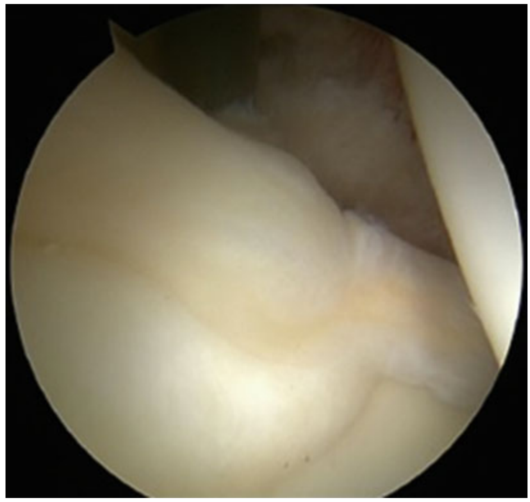


Fig. 5 Needle to access “the pocket” (Source: Mr. Giles Stafford)

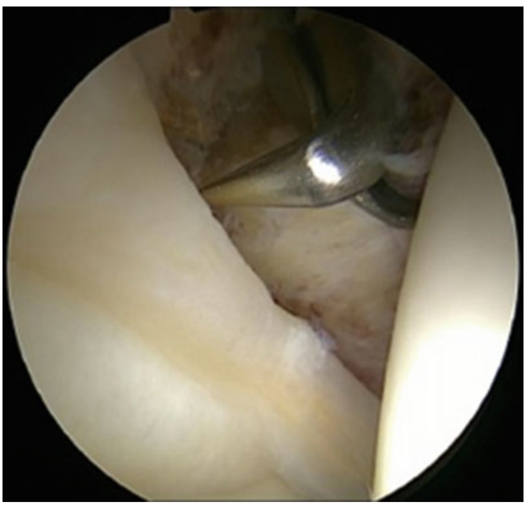


Fig. 4 Microfracture to subchondral bone (Source: Mr. Giles Stafford)

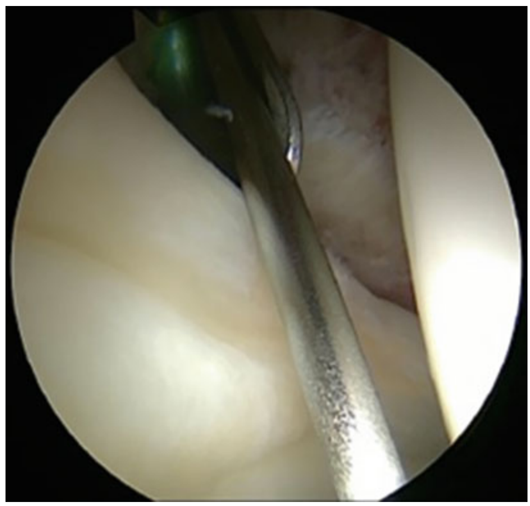


Fig. 6 Use of needle to aspirate joint fluid, located in the cotyloid fossa (Source: Mr. Giles Stafford)

The microfracture awl is passed under the labrum into the pocket and the microfracture technique is performed on the underlying subchondral bone (Fig. 4). Care must be taken to ensure that no further damage occurs to the cartilage.

The application of the fibrin glue requires two arthroscopy needles. The first is into the pocket behind the delaminated cartilage for application of the fibrin (Fig. 5). Another needle is inserted through the anterolateral portal into the base of the central compartment or cotyloid fossa for aspiration of the joint to dryness (Fig. 6). Once the irrigation fluid is removed (Fig. 7), the fibrin glue may be applied using the first needle (Fig. 8). Immediately following this, an angled arthroscopic punch is used to press firmly on the delaminated cartilage to ensure it securely adheres to subchondral bone while the fibrin glue sets

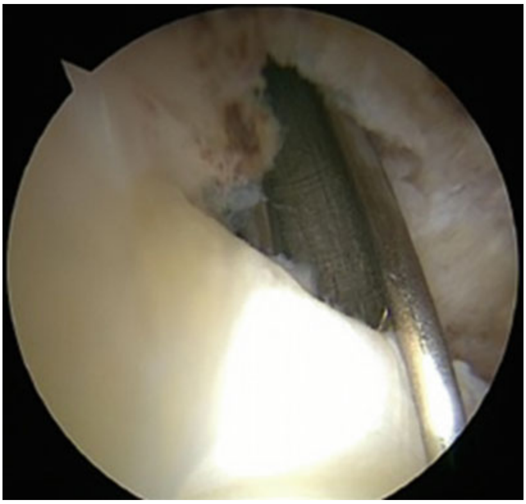


Fig. 7 Needle insertion into the pocket behind the labrum (Source: Mr. Giles Stafford)

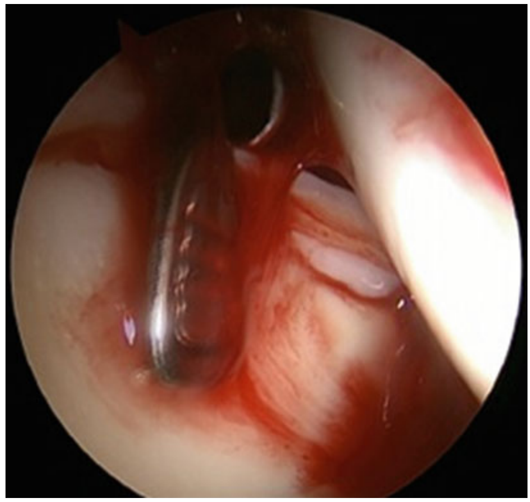


Fig. 9 Arthroscopic punch used to apply pressure to the cartilage to ensure adhesion between the cartilage and subchondral bone (Source: Mr. Giles Stafford)

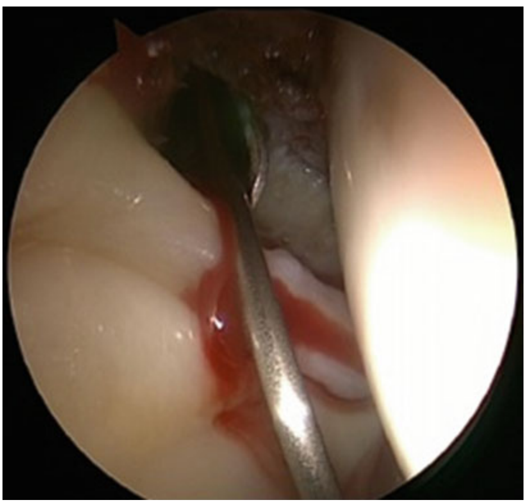


Fig. 8 Application of fibrin glue using the needle in "the pocket" (Source: Mr. Giles Stafford)

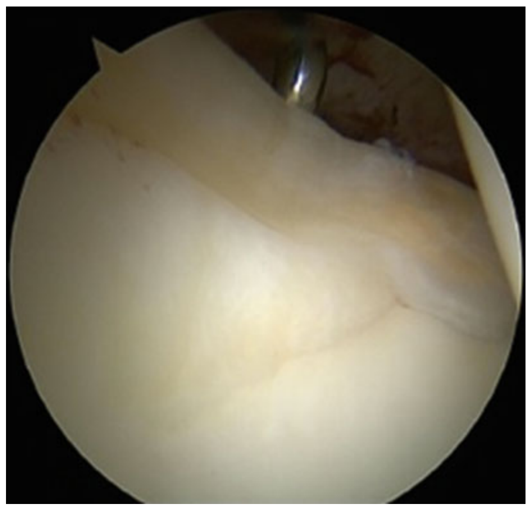


Fig. 10 Successful repair following fibrin glue adhesion (Source: Mr. Giles Stafford)

(Fig. 9). The process of bonding the cartilage to the bone takes 2 min, resulting in a successful repair (Fig. 10). Any excess adhesive that spills into the central compartment can be removed using arthroscopic graspers.

Reduction of the femoral head into the acetabulum results in further compression of the cartilage onto the subchondral bone. Postoperatively the patients are discharged after physiotherapy review and implementation of a rehabilitation regime with adequate analgesia.

Results in the Literature

Results are limited to a case series of 43 patients followed up at 1 and 3 years. Patients were clinically assessed pre- and postoperatively using the

Modified Harris Hip Score, and these results are statistically analyzed.

At 28 months there was a statistically significant improvement in pain from a mean of 21.8 (95 % CI 19–24.7) pre-operatively to 35.8 (95 % CI 32.6–38.9). A more modest statistically significant improvement in function was also demonstrated [3].

Three patients underwent revision hip arthroscopy surgery for iliopsoas pathology. The repaired articular cartilage in these patients was inspected and appeared to be in good condition, suggesting that this method is effective in the treatment of delaminated cartilage [3].

The Future

A small case number and no reported histological analysis of the repaired cartilage limit the above study. Therefore, to further validate this as an effective method of repair, a double-blinded randomized controlled trial would be required. Biopsy of the macroscopically healthy cartilage may be viewed as ethically complicated. Therefore, a less invasive method of assessing the health of the cartilage using image novel MRI techniques include dGEMRIC (delayed gadolinium-enhanced MRI of cartilage) and T2 cartilage mapping, both of which are currently being validated with regard to the measurement of cartilage quality [40, 41, 42].

There are developments in fibrin glue, which may improve the efficacy of the healing process. In addition adhesive and hemostatic properties studies have shown fibrin glue is also a useful vehicle for the delivery of mesenchymal stem cells which results in earlier healing of meniscal injuries in animal models and improved healing in other areas of medicine [43–47].

Overall, fibrin glue as an adjunct to microfracture has been shown to be safe and effective in the management of cartilage delamination with equivalent and possibly superior results to traditional methods involving excision of cartilage with microfracture alone.

Summary

In summary this chapter outlines the history of fibrin and its properties, both hemostatic and adhesive. Furthermore, the technique whereby delaminated cartilage is adhered back to its subchondral bone using fibrin glue is described. The fibrin also has effects at a cellular level to stimulate the repopulation of the cartilage with chondrocytes and therefore results in healthy cartilage. This technique has been shown to be effective in improving Harris Hip Scores and halt progression of hyaline cartilage damage.

References

1. Tzaveas AP, Villar RN. Arthroscopic repair of acetabular chondral delamination with fibrin adhesive. Hip Int. 2010;20(1):115–9.
2. Beck M, Leunig M, Parvizi J, Boutier V, Wyss D, Ganz R. Anterior femoroacetabular impingement: part II. Midterm results of surgical treatment. Clin Orthop Relat Res. 2004;418:67–73.
3. Stafford GH, Bunn JR, Villar RN. Arthroscopic repair of delaminated acetabular articular cartilage using fibrin adhesive. Results at one to three years. Hip Int. 2011;21(6):744–50.
4. Hubbard MJ. Arthroscopic surgery for chondral flaps in the knee. J Bone Joint Surg. 1987;69:794–6.
5. Miller BS, Steadman JR, Briggs KK, Rodrigo JJ, Rodkey WG. Patient satisfaction and outcome after microfracture of the degenerative knee. J Knee Surg. 2004;17(1):13–7.
6. Brittberg M, Lindahl A, Nilsson A, Ohlsson C, Isaksson O, Peterson L. Treatment of deep cartilage defects in the knee with autologous chondrocyte transplantation. N Engl J Med. 1994;331:889–95.
7. Steadman JR, Rodkey WG, Simgleton SB, Briggs KK. Microfracture technique for full-thickness chondral defects: technique and clinical results. Oper Tech Orthop. 1997;7(4):300–4.
8. Philippon MJ, Schenker ML, Briggs KK, Maxwell RB. Can microfracture produce repair tissue in acetabular chondral defects? Arthroscopy. 2008;24(1):46–50.
9. Bergel S. Uber Wirkungen des Fibrins. Dtsch Med Wochenschr. 1909;35:633.
10. Young JZ, Medawar PB. Fibrin suture of peripheral nerves. Lancet. 1940;275:126–32.
11. Cronkite EP, Lozner EL, Deaver J. Use of thrombin and fibrinogen in skin grafting. JAMA. 1944;124:976–8.
12. Siedentop KH, Harris DM, Sanchez B. Autologous fibrin tissue adhesive. Laryngoscope. 1985;95:1074–6.

13. Spotnitz WD, Mintz PD, Avery N, et al. Fibrin glue from stored human plasma: an inexpensive and efficient method for local blood bank preparation. Am Surg. 1987;53:460–4.
14. Bove JR. Fibrinogen–is the benefit worth the risk? Transfusion. 1978;18:129.
15. Mannucci PM. Hemostatic drugs. N Engl J Med. 1998;339:245.
16. Kram HB, Nathan RC, Mackabee JR, et al. Clinical use of nonautologous fibrin glue. Am Surg. 1988;54:570.
17. Harris DM, Siedentop KH, Ham KR, Sanchez B. Autologous fibrin tissue adhesive biodegration and systemic effects. Laryngoscope. 1987;97:1141.
18. Spotnitz WD, Burks S. Hemostats, sealants, and adhesives: components of the surgical toolbox. Transfusion. 2008;48:1502.
19. Gibble JW, Ness PM. Fibrin glue: the perfect operative sealant? Transfusion. 1990;30:741.
20. Radosevich M, Goubran HI, Burnouf T. Fibrin sealant: scientific rationale, production methods, properties, and current clinical use. Vox Sang. 1997;72:133.
21. Schenk W, Burks S, Gagne P, et al. Fibrin sealant improves hemostasis in peripheral vascular surgery: a randomized prospective trial. Ann Surg. 2003; 237:871–6.
22. Saha SP, Muluk S, Schenk 3rd W, et al. A prospective randomized study comparing fibrin sealant to manual compression for the treatment of anastomotic suture-hole bleeding in expanded polytetrafluoroethylene grafts. J Vasc Surg. 2012;56:134.
23. Geissler HP, Gosselin C, Ren D, et al. Biointeractive polymers and tissue engineered blood vessels. Biomaterials. 1996;17:329–36.
24. Thistlethwaite PA, Luketich JD, Ferson PF, et al. Ablation of persistent air leaks after thoracic procedures with fibrin sealant. Ann Thorac Surg. 1999; 67:575–7.
25. Noun R. Fibrin glue effectiveness and tolerance after elective liver resection: a randomized trial. Hepatogastroenterol. 1996;43:221–4.
26. Uranues S, Mischinger HJ, Pfeifer J, et al. Hemostatic methods for the management of spleen and liver injuries. World J Surg. 1996;20:1107–12.
27. Langer S, Guenther JM, DiFronzo LA. Does fibrin sealant reduce drain output and allow earlier removal of drainage catheters in women undergoing operation for breast cancer? Am Surg. 2003;69:77–81.
28. Spotnitz WD, Burks SG, Prabhu R. Fibrin-based adhesives and hemostatic agents. In: Quinn JV, editor. Tissue adhesives in clinical medicine. Hamilton: BC Decker; 2005. p. 77–112.
29. Singer AJ, Hollander JE, Valentine SM, et al. Prospective, randomized, controlled trial of tissue adhesive (2-octylcyanoacrylate) vs standard wound closure techniques for laceration repair. Stony Brook Octylcyanoacrylate Study Group. Acad Emerg Med. 1998;5:94.
30. Schlag G, Redil H. Fibrin sealant in orthopaedic surgery. Orthop Relat Res. 1988;227:269–85.
31. Kaplonyi G, Zimmerman I, Frenyo AD, Farkas T, Nemes G. The use of fibrin adhesive in the repair of chondral and osteochondral injuries. Injury. 1988; 19(4):267–72.
32. Plaga BR, Royster RM, Donigian AM, Wright GB, Caskey PM. Fixation of osteochondral fractures in rabbit knees. A comparison of Kirschner wires, fibrin sealant, and polydioxanone pins. J Bone Joint Surg Br. 1992;74(2):292–6.
33. Patel S, Rodrigues-Merchan EC, Haddad FS. The use of fibrin glue in surgery of the knee. J Bone Joint Surg Br. 2010;92-B:1325–31.
34. Kim MK, Choi SW, Kim SR, Oh IS, Won MH. Autologous chondrocyte implantation in the knee using fibrin. Knee Surg Sports Traumatol Arthrosc. 2010; 18:528–34.
35. Aubart F, Roger B, Zeine G. Repair by glueing under arthroscopic control of ruptures of the anterior cruciate ligament: preliminary study of 10 cases controlled by magnetic resonance imaging. Rev Chir Orthop Reparatrice Appar Mot. 1991;77:267–72 (in French).
36. Hembree WC, Ward BD, Furman BD, et al. Viability and apoptosis of human chondrocytes in osteochondral fragments following joint trauma. J Bone Joint Surg Br. 2007;89:1388–95.
37. Ball ST, Jadin K, Allen RT, Schwartz AK, Sah RL, Brage ME. Chondrocyte viability after intra-articular calcaneal fractures in humans. Foot Ankle Int. 2007;28:665–8.
38. Kirilak, et al. Fibrin sealant promotes migration and proliferation of human articular chondrocytes: possible involvement of thrombin and protease-activated receptors. Int J Mol Med. 2006;17:551–8.
39. Steadman JR, Rodkey WG, Rodrigo JJ. Microfracture: surgical technique and rehabilitation to treat chondral defects. Clin Orthop Relat Res. 2001;(391 Suppl): S362–9.
40. van Tiel J, Oei EH. Quantitative measurement of articular cartilage quality using MRI Ned Tijdschr Geneeskd. J Magn Reson Imaging. 2013;157(36): A6340. doi:10.1002/jmri.24096. [Epub ahead of print].
41. Zilkens C, Miese F, Kim YJ, Jäger M, Mamisch TC, Hosalkar H, Antoch G, Krauspe R, Bittersohl B. Direct comparison of intra-articular versus intravenous delayed gadolinium-enhanced MRI of hip joint. University of Dusseldorf, Medical Faculty, Department of Orthopedic Surgery, Dusseldorf.
42. Ishimura M, Ohgushi H, Habata T, Tamai S, Fujisawa Y. Arthroscopic meniscal repair using fibrin glue. Part I: experimental study. Arthroscopy. 1997; 13(5):551–7.
43. Izuta Y, Ochi M, Adachi N, et al. Meniscal repair using bone marrow-derived mesenchymal stem cells: experimental study using green fluorescent protein transgenic rats. Knee. 2005;12:217–23.
44. Wing K, et al. Fibrin glue with autogenic bone marrow mesenchymal stem cells for urethral injury

repair in rabbit model. Tissue Eng Part A. 2012; 18(23–24):2507–17.
45. Yamada Y, et al. Bone regeneration following injection of mesenchymal stem cells and fibrin glue with a biodegradable scaffold. J Craniomaxillofac Surg. 2003; 31(1):27–33.
46. Lim JK, Hui J, Li L, et al. Enhancement of tendon graft osteointegration using mesenchymal stem cells in a rabbit model of anterior cruciate ligament reconstruction. Arthroscopy. 2004;20:899–910.
47. Falanga V, et al. Autologous bone marrow-derived cultured mesenchymal stem cells delivered in a fibrin spray accelerate healing in murine and human cutaneous wounds. Tissue Eng. 2007;13(6):1299–312.
48. Haleem AL, et al. The clinical use of human culture-expanded autologous bone marrow mesenchymal stem cells transplanted on platelet-rich fibrin glue in the treatment of articular cartilage defects: a pilot study and preliminary results. Cartilage. 2010; 1(4):253–61.

Stem Cell Therapy for Hip Cartilage Lesions: Clinical Applications

90

Rodrigo Mardones and Catalina Larrain

Contents

Abstract

Hip cartilage lesions represent a diagnostic challenge and can be an elusive source of pain. This chapter will focus on new technologies to enhance the standard techniques. These new technologies are based on stem cell therapies: as intra-articular injections of expanded mesenchymal stem cells, mononuclear concentrate in a platelet-rich plasma matrix, and expanded mesenchymal stem cells seeded in a collagen membrane. This review will discuss the bases, techniques, and preliminary results obtained with the use of stem cells for the treatment of hip cartilage lesions.

Introduction

As it was mentioned in ► Chap. 91, "Stem Cell Therapy for Hip Cartilage Lesions: General Concepts and Basic Science," the fibrocartilage newly formed at the microfractured area is a low quality tissue; therefore we will describe some techniques involving stem cell therapy that may lead to a better hyaline-like cartilage. The use of these novel technologies has demonstrated promising results in animal and clinical studies.

R. Mardones (✉) • C. Larrain
Orthopedics Department, Clínica Las Condes, Santiago, Chile
e-mail: rmardones@clc.cl; clarrain@clinicalascondes.cl

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_120

Mononuclear Concentrate in a Platelet-Rich Plasma Matrix

Human platelet-rich plasma (PRP) studies include overuse pathologies (epicondylitis [1], patellar [2], and Achilles [3] tendinopathies), sports medicine (anterior cruciate ligament reconstruction [4–8] and rotator cuff repair [9–12]), chondral pathology (osteoarthritis and focal chondral lesions [13–17]), spine [18, 19]), trauma (fractures and pseudoarthrosis [20, 21]), and management of skin lesions (acute and chronic [22, 23]). The results of the clinical studies are conflicting, demonstrating positive effects and no changes in the same pathologies; however, the utility of the PRP has been proven in epicondylitis, patellar tendinopathy, pseudoarthrosis, and chronic wound management. It is likely that interpatient variability and diversity of commercial kits, preparation, implementation, and applied concentrations may play an important role in product efficacy, thus influencing the results [24, 25]. Despite this, the studies agree on the anti-inflammatory and procoagulant effect of the PRP.

Hip chondral lesions traditionally have been handled equal to other joints, with similar results of that obtained at the knee, but the spherical form of the hip, the composition and anatomy of the cartilage, and the unique types of chondral lesions (delamination) make the techniques and the results obtained at other joints not replicable at the hip. Hip chondral lesion clinical studies are limited to treatment with microfractures and fibrin clot (fibrin glue®) for delamination [26–28]. These, though they are small clinical series, showed some promising results in localized chondral lesions. Milano et al., in a study conducted in sheeps, demonstrated that the use of a PRP clot associated with microfractures achieved a complete filling of the chondral lesion with macroscopic, biomechanic, and microscopic characteristics similar to normal hyaline cartilage [26].

Currently, the PRP clot is used as a carrier or membrane carrier of mesenchymal stem cells, a technique that will be explained later.

In summary, the use of a PRP clot in hip chondral lesions has little published evidence; however, the minimal cost and risks of the procedure associated with the promising results obtained in both animal and preliminary clinical studies support its use in the clinical practice of arthroscopic hip surgery.

Treatment of Choice

The treatment of choice for hip chondral lesions is the use of a platelet-rich plasma clot and MCC over the microfractured area (Fig. 1). The surgical technique is described below.

Excellent results have been obtained with this technique, confirming the restoration of glycosaminoglycan concentration by MRI metabolic-type dGEMRIC (delayed gadolinium-enhanced magnetic resonance imaging of cartilage) (Fig. 1f, g).

Surgical Technique

After rim trimming and labrum refixation, cartilage assessment is made. If chondral lesion exists, the harvesting of 15 cc of bone marrow is made and centrifugated. obtaining 2–4 cc of autologous bone marrow – mesenchymal stem cell concentrate (average 14 millions of nucleated cells/cc^3). At the same time, 50 cc of peripheral blood is taken and centrifugated twice, in order to obtain 4 cc of PRP (6–9×), ready to be activated with autologous thrombin. Treatment of chondral lesion is made as described by Steadman in the knee, with debridement of all remaining unstable cartilage, followed by the removal of the calcified plate. After preparation of the bed, multiple holes in the exposed subchondral bone plate are made, leaving about 3–4 mm between each. Once microfracture is complete, traction is released and femoral osteoplasty is completed, obtaining a free range of motion with no abnormal contact between acetabular rim and femoral neck-head junction. At the end of the procedure, traction is reinstalled and the final part of the procedure is performed. After activation of platelet-rich plasma and clot formation, a slotted cannula is inserted via the anterior portal. Platelet-rich plasma clot is inserted through the cannula and positioned over the microfractured area. A 21-gauge trocar is then inserted passing through

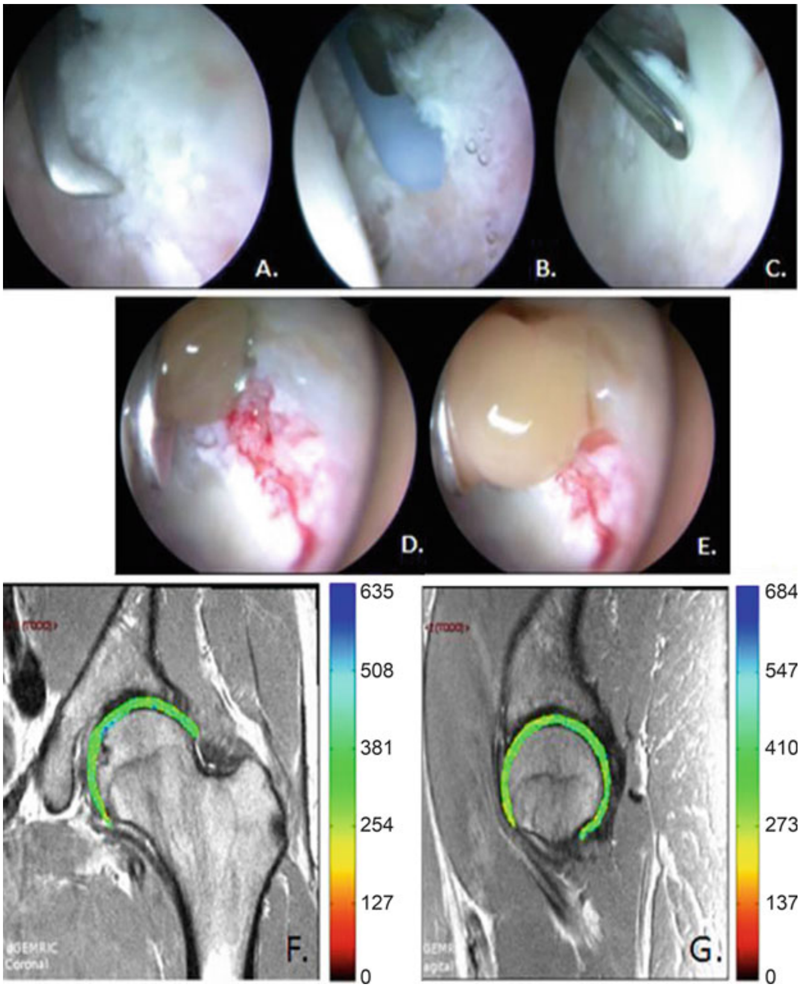


Fig. 1 Hip chondral lesions: surgical alternatives and novel technique with platelet-rich plasma and mononuclear cells concentrate. (**a**–**c**) Standard alternatives for hip chondral lesions. (**a**) Microfractures. (**b**) Thermal chondroplasty. (**c**) Chondral flaps resection. (**d**) and (**e**) Novel surgical technique. The PRP clot is positioned over the microfractured area and mononuclear cells concentrate is instilled under it. (**f**) and (**g**) dGEMRIC images at 6 months postop of the same patient, in which a homogenous captation of gadolinium is observed, meaning the restoration of glycosaminoglican content

previously located clot and autologous bone marrow – mesenchymal stem cell concentrate is instilled under PRP clot. Traction is then released and the procedure is finished.

Rehabilitation protocol: Passive motion device is maintained for 8 h. Two crutches with partial weight bearing are indicated for 6–8 weeks. Progressive physical activities are allowed.

Preliminary Results

At the time, 13 patients with chondral lesion of the hip had been treated with microfractures and autologous bone marrow – mesenchymal stem cell concentrate transplanted on a platelet-rich plasma clot. All patients' symptoms improved over the follow-up period of 8 months (4–12 months). Average Hip Outcome, Vail Hip, and Modified Harris Hip scores for all patients showed significant improvement at 3 and 6 months. dGEMRIC of 4 patients at 6 months postoperatively revealed complete defect fill and complete surface congruity with native cartilage.

Intra-articular Injections of Expanded Mesenchymal Stem Cells

Osteoarthritis (OA) is the most common type of arthritis and the leading cause of disability in the United States [29]. Several systemic treatments, mostly symptom-modifying rather than disease-modifying agents, are available for OA

[30]. However, there is a real need for effective and safe disease-modifying OA therapies that can not only effectively treat those with established OA but also possibly delay or prevent progression in those with early OA [31]. As it was mentioned before in focal chondral lesions, mesenchymal stem cells represent a valid alternative for treatment, but multiple chondral lesions or established osteoarthritis are not suitable for focal treatment.

Adult mesenchymal stem cells were originally believed to only differentiate into tissue-specific cells. However, these cells have two major properties that could explain some of the results seen with the intra-articular injections of expanded mesenchymal stem cells, and these are *homing* and *response to specific signals*. Homing is a particular property of these cells, meaning that they respond to systemic stimuli and "travel to the place that needs repair." The homing effect has been demonstrated in several animal studies, using labeled mesenchymal stem cells administered via systemic intravascular route or by direct local implantation, showing the presence of the marker at the injury site [32]. Mesenchymal stem cells have the ability to differentiate into a different tissue in response to specific signals released by the injury site, such as chondrogenic lineage in an osteoarthritic joint [33].

Hip extensive damage or mild OA is usually treated with local infiltrations, symptom-modifying treatments, pain killers, and finally a total hip replacement, but an increasing number of active patients seek for a non-arthroplasty treatment, and stem cells may present as an alternative to this group of patients. Intra-articular injections of expanded mesenchymal stem cells have not been described in the hip joint; however, there are some animal and clinical studies in other joints. Mokbel et al. labeled autologous adult stem cells suspended in hyaluronic acid were injected intra-articularly into carpal joints in an experimental arthritis induced by intra-articular (IA) amphotericin-B in donkeys [33]. Significant improvement was noted in clinical and radiographic OA, and significantly lesser histopathological changes of OA were seen in carpal joints that received stem cells compared to control contralateral joints. Importantly, injected stem cells were incorporated into the articular cartilage of the injected joint, as evident by their integration in the surface of the cartilage and also the interior of the cartilage [33]. Emadedin et al. injected expanded mesenchymal stem cells in six female patients with OA that required joint replacement. At 12 months follow-up, there was a significant decrease in mean pain, as well as improvements in joint functioning, walking distance, time to gelling, patellar crepitus, and knee flexion. Magnetic resonance images (MRI) obtained at 6 months after treatment showed an increase in cartilage thickness and extension of the repair tissue over the subchondral bone in half of the patients, in addition to a decrease in subchondral bone edema [34]. McIlwraith et al. evaluated intra-articular injection of bone marrow-derived mesenchymal stem cells to augment healing with microfractures in horses. At 6 months, arthroscopic and gross evaluation confirmed a significant increase in repair tissue firmness and a trend for better overall repair tissue quality in treated joints compared to microfractures alone. Immunohistochemical analysis showed significantly greater levels of aggrecan in repair tissue treated with stem cell injection [35].

In summary, the use of intra-articular injections of expanded mesenchymal stem cells in OA has little published evidence; however, in a young active patient, it seems to be a promising non-arthroplasty treatment.

Treatment of Choice

The treatment of choice for hip diffuse chondral damage and mild osteoarthritis in an active patient seeking for a non-arthroplasty treatment is intra-articular injections of expanded mesenchymal stem cells. The surgical technique is described below.

Excellent results have been obtained with this technique, with an increase in hip functional scores (Vail-10 hip score and Harris Hip Score) and a decrease in mean pain values.

Surgical Technique

Patients were placed on an operating table in the prone position under general anesthesia. The harvesting of 15 cc of bone marrow is made and

Fig. 2 Autologous mesenchymal stem cells concentrate. (**a**) Autologous bone marrow autograft (**b**) Centrifugation process with a single spin. (**c**) Layer separation by a density filter and identification of mononuclear cells layer. (**d**) Autologous mononuclear cells concentrate – final view

Fig. 3 Intra-articular injection of expanded mesenchymal stem cells. Fluoroscopic image

centrifugated, obtaining 2–4 cc of autologous bone marrow – mesenchymal stem cell concentrate (average 14 millions of nucleated cells/cc^3) (Fig. 2). Bone marrow concentrate is then processed in a GMP laboratory, and over a 1-month period, mesenchymal stem cells are expanded to 20×10^6 cells and taken to the hospital in a portable incubator. Under fluoroscopy, cells were injected into the patients' hips (Fig. 3).

Preliminary Results

At the time, 7 patients with mild OA of the hip had been treated with intra-articular injections of expanded mesenchymal stem cells. All patients' symptoms improved over the follow-up period of 10 months (8–14 months). Average Vail-10 and Modified Harris Hip scores for all patients showed significant improvement at 3 and 6 months. None of the patients has required a total hip replacement at the time.

Expanded Mesenchymal Stem Cells Seeded in a Collagen Membrane

As mentioned in ▶ Chap. 91, "Stem Cell Therapy for Hip Cartilage Lesions: General Concepts and Basic Science," hip chondral lesions can be an elusive source of pain, and their treatment is limited to chondroplasty and debridement in partial defects and microfractures for full-thickness chondral lesions. Microfracture involves penetration of the subchondral bone to release blood and bone marrow into the defect, initiating cartilage repair. This technique has produced good clinical results in defects <2 cm^2. For larger lesions, bone marrow concentrate in a PRP clot seems to be a good alternative. Other treatment options include autologous chondrocyte implantation (ACI), matrix-induced ACI (MACI), autologous matrix-induced chondrogenesis (AMIC), and membrane seeded with expanded mesenchymal stem cells.

ACI has been used increasingly for the repair of larger chondral lesions in the knee. For hip chondral lesion management, only two reports were found. Fontana et al. described a case control study in 30 patients with hip chondral lesions, 15 treated with ACI and 15 with debridement alone. At 74 months follow-up, Harris Hip Score was significantly better in ACI group compared to the debridement group [36]. Akimau et al. described a case of severe chondrolysis and osteonecrosis of the femoral head after a severe fracture dislocation in a 31-year-old man. Twenty-one months after the injury, they performed a MACI technique. At 1-year follow-up, the subjective hip score and range of motion had increased compared to preoperative values. At 15 months follow-up, biopsy demonstrated a 2 mm thickness cartilage, well populated with viable cells and integrated with the underlying bone [37].

Fontana described a fully arthroscopic technique for the hip for AMIC. This is a low-cost, single procedure and arthroscopic technique in which the author used a collagen matrix (Chondro-gide, Geistlich Pharma AG, Wolhusen, Switzerland) over the microfractured area, containing the blood and bone marrow for a better quality reparative tissue [38]. There is no published clinical data available.

The use of membranes seeded with expanded mesenchymal stem cells has risen in response of some problems observed with the use of MACI, such as the morbidity of the donor site and insufficient coverage of the defect area due to some shrinkage effect. This technique has shown good results in other joints, but it has not been described in the hip joint.

Summary

Hip chondral lesions are a frequent finding in hip arthroscopy for FAI. Treatment is often difficult and insufficient. Novel strategies based on cell regenerative therapy represent a promising treatment alternative. The novel alternatives for hip chondral lesions treatment and its preliminary results were reviewed.

References

1. Gosens T, Peerbooms JC, van Laar W, den Oudsten BL. Ongoing positive effect of platelet-rich plasma versus corticosteroid injection in lateral epicondylitis: a double-blind randomized controlled trial with 2-year follow-up. Am J Sports Med. 2011;39(6):1200–8.
2. Filardo G, Kon E, Della Villa S, Vincentelli F, Fornasari PM, Marcacci M. Use of platelet-rich plasma for the treatment of refractory jumper's knee. Int Orthop. 2010;34(6):909–15.
3. Sanchez M, Anitua E, Azofra J, Andia I, Padilla S, Mujika I. Comparison of surgically repaired Achilles tendon tears using platelet-rich fibrin matrices. Am J Sports Med. 2007;35:245–51.
4. Murray MM, Spindler KP, Ballard P, Welch TP, Zurakowski D, Nanney LB. Enhanced histologic repair in a central wound in the anterior cruciate ligament with a collagen-platelet-rich plasma scaffold. J Orthop Res. 2007;25:1007–17.
5. Radice F, Gutierrez V, Ibarra A, Verdugo A. Arthroscopic, histologic and MRI correlation in the maturation process of the graft in ACL reconstruction in humans. Arthroscopy. 1998;14 Suppl 1:S20.
6. Ekdahl M, Wang JH, Ronga M, Fu FH. Graft healing in anterior cruciate ligament reconstruction. Knee Surg Sports Traumatol Arthrosc. 2008;16:935–47.
7. Orrego M, Orrego M, Larrain C, Rosales J, Valenzuela L, Matas J, Durruty J, Sudy H, Mardones R. Effects of platelet concentrate and a bone plug on the healing of hamstring tendons in a bone tunnel. Arthroscopy. 2008;24(12):1373–80.
8. Figueroa D, Melean P, Calvo R, Vaisman A, Zilleruelo N, Figueroa F, Villalón I. Magnetic resonance imaging evaluation of the integration and maturation of semitendinosus-gracilis graft in anterior cruciate ligament reconstruction using autologous platelet concentrate. Arthroscopy. 2010;26(10): 1318–25.
9. Barber FA, Hrnack SA, Snyder SJ, Hapa O. Rotator cuff repair healing influenced by platelet-rich plasma construct augmentation. Arthroscopy. 2011;27(8): 1029–35.
10. Gamradt SC. Short-term positive effects of platelet-rich plasma in arthroscopic rotator cuff repair. J Bone Joint Surg Am. 2011;93(13):1–3.
11. Rodeo SA, Delos D, Williams RJ, Adler RS, Pearle A, Warren RF. The effect of platelet-rich fibrin matrix on rotator cuff tendon healing: a prospective, randomized clinical study. Am J Sports Med. 2012;40(6):1234–41. Article in press.
12. Jo CH, Kim JE, Yoon KS, Shin S. Platelet-rich plasma stimulates cell proliferation and enhances matrix gene expression and synthesis in tenocytes from human rotator cuff tendons with degenerative tears. Am J Sports Med. 2012;40(5):1035–45.
13. Kon E, Mandelbaum B, Buda R, Filardo G, Delcogliano M, Timoncini A, Fornasari PM, Giannini S, Marcacci M. Platelet-rich plasma intra-articular injection versus hyaluronic acid viscosupplementation as treatments for cartilage pathology: from

early degeneration to osteoarthritis. Arthroscopy. 2011;27(11):1490–501.
14. Sanchez M, Anitua E, Azofra J, Aguirre JJ, Andia I. Intra-articular injection of an autologous preparation rich in growth factors for the treatment of knee OA: a retrospective cohort study. Clin Exp Rheumatol. 2008;26:910–3.
15. Klatt BA, Lopez HH, Segal NA, Chimes GP. Treatment options in knee osteoarthritis: total knee arthroplasty versus platelet-rich plasma. PM R. 2011;3(4):377–86.
16. Sampson S, Reed M, Silvers H, Meng M, Mandelbaum B. Injection of platelet-rich plasma in patients with primary and secondary knee osteoarthritis: a pilot study. Am J Phys Med Rehabil. 2010;89(12):961–9.
17. Dhillon M, Patel S, Bali K. Platelet-rich plasma intra-articular knee injections for the treatment of degenerative cartilage lesions and osteoarthritis. Knee Surg Sports Traumatol Arthrosc. 2011;19(5):863–4.
18. Hartmann EK, Heintel T, Morrison RH, Weckbach A. Influence of platelet-rich plasma on the anterior fusion in spinal injuries: a qualitative and quantitative analysis using computer tomography. Arch Orthop Trauma Surg. 2010;130(7):909–14.
19. Landi A, Tarantino R, Marotta N, Ruggeri AG, Domenicucci M, Giudice L, Martini S, Rastelli M, Ferrazza G, De Luca N, Tomei G, Delfini R. The use of platelet gel in postero-lateral fusion: preliminary results in a series of 14 cases. Eur Spine J. 2011;20 Suppl 1:S61–7.
20. Galasso O, Mariconda M, Romano G, Capuano N, Romano L, Iannò B, Milano C. Expandable intramedullary nailing and platelet rich plasma to treat long bone non-unions. J Orthop Traumatol. 2008;9(3): 129–34.
21. Mariconda M, Cozzolino F, Cozzolino A, D'Agostino E, Bove A, Milano C. Platelet gel supplementation in long bone nonunions treated by external fixation. J Orthop Trauma. 2008;22(5):342–5.
22. De Leon JM, Driver VR, Fylling CP, Carter MJ, Anderson C, Wilson J, Dougherty RM, Fuston D, Trigilia D, Valenski V, Rappl LM. The clinical relevance of treating chronic wounds with an enhanced near-physiological concentration of platelet-rich plasma gel. Adv Skin Wound Care. 2011;24(8):357–68.
23. Dougherty EJ. An evidence-based model comparing the cost-effectiveness of platelet-rich plasma gel to alternative therapies for patients with nonhealing diabetic foot ulcers. Adv Skin Wound Care. 2008;21(12): 568–75.
24. Mazzocca AD, McCarthy MB, Chowaniec DM, Cote MP, Romeo AA, Bradley JP, Arciero RA, Beitzel K. Platelet-rich plasma differs according to preparation method and human variability. J Bone Joint Surg Am. 2012;94(4):308–16.
25. Tschon M, Fini M, Giardino R, Filardo G, Dallari D, Torricelli P, Martini L, Giavaresi G, Kon E, Maltarello MC, Nicolini A, Carpi A. Lights and shadows concerning platelet products for musculoskeletal regeneration. Front Biosci (Elite Ed). 2011;1(3): 96–107.
26. Milano G, Sanna Passino E, Deriu L, Careddu G, Manunta L, Manunta A, Saccomanno MF, Fabbriciani C. The effect of platelet rich plasma combined with microfractures on the treatment of chondral defects: an experimental study in a sheep model. Osteoarthritis Cartilage. 2010;18(7):971–80.
27. Philippon MJ, Schenker ML, Briggs KK, Maxwell RB. Can microfracture produce repair tissue in acetabular chondral defects? Arthroscopy. 2008; 24(1):46–50.
28. Tzaveas AP, Villar RN. Arthroscopic repair of acetabular chondral delamination with fibrin adhesive. Hip Int. 2010;20(1):115–9.
29. Centers for Disease Control and Prevention. Prevalence and most common causes of disability among adults – United States, 2005. MMWR Morb Mortal Wkly Rep. 2009;58:421–6.
30. Sinusas K. Osteoarthritis: diagnosis and treatment. Am Fam Physician. 2012;85:49–56.
31. Hunter DJ. Osteoarthritis. Best Pract Res Clin Rheumatol. 2011;25:801–14.
32. Chen A, Siow B, Blamire A, et al. Transplantation of magnetically labeled mesenchymal stem cells in a model of perinatal injury. Stem Cell Res. 2010; 5(3):255–66.
33. Mokbel AN, El Tookhy OS, Shamaa AA, et al. Homing and reparative effect of intra-articular injection of autologous mesenchymal stem cells in osteoarthritic animal model. BMC Musculoskelet Disord. 2011;12:259.
34. Emadedin M, Aghdami N, Taghiyar L, et al. Intra-articular injection of autologous mesenchymal stem cells in six patients with knee osteoarthritis. Arch Iran Med. 2012;15(7):422–8.
35. McIlwraith CW, Frisbie DD, Rodkey WG, Kisiday JD, Werpy NM, Kawcak CE, Steadman JR. Evaluation of intra-articular mesenchymal stem cells to augment healing of microfractured chondral defects. Arthroscopy. 2011;27(11):1552–61.
36. Fontana A, Bistolfi A, Crova M, et al. Arthroscopic treatment of hip chondral defects: autologous chondrocyte transplantation versus simple debridement – a pilot study. Artroscopy. 2012;28(3):322–9.
37. Akimau P, Bhosale A, Harrison P, et al. Autologous chondrocyte implantation with bone grafting for osteochondral defect due to posttraumatic osteonecrosis of the hip – a case report. Acta Orthop. 2006;77(2):333–6.
38. Fontana A. A novel technique for treating cartilage defects in the hip: a fully arthroscopic approach to using autologous matrix – induced chondrogenesis. Artrhosc Techn. 2012;1(1):e63–8.

Stem Cell Therapy for Hip Cartilage Lesions: General Concepts and Basic Science

91

Rodrigo Mardones and Catalina Larrain

Contents

Abstract

Hip cartilage lesions represent a diagnostic challenge and can be an elusive source of pain. Treatment may present difficulties due to localization and spherical form of the joint and is most commonly limited to excision, debridement, thermal chondroplasty, and microfractures. This chapter will focus on new technologies to enhance the standard techniques. These new technologies are based on stem cell therapies: as intra-articular injections of expanded mesenchymal stem cells, mononuclear concentrate in a platelet-rich plasma matrix, and expanded mesenchymal stem cells seeded in a collagen membrane. This review will discuss the bases, techniques, and preliminary results obtained with the use of stem cells for the treatment of hip cartilage lesions.

Hip Chondral Lesions

Femoroacetabular impingement is frequently associated with chondral damage. The abnormal contact between the femoral neck and the acetabular rim results in labral detachment and acetabular chondral damage [1, 2].

In the hip, the types of chondral lesion differ from other joints due to the spherical form of the hip and the mechanisms of damage; the cam-type femoroacetabular impingement is frequently associated with chondrolabral junction damage

R. Mardones (✉) • C. Larrain
Orthopedics Department, Clínica Las Condes, Santiago, Chile
e-mail: rmardones@clc.cl; clarrain@clinicalascondes.cl

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_100

with the subsequent acetabular cartilage detachment, and the pincer type with the contrecoup injury of the posteroinferior acetabulum [3].

Delamination is a characteristic chondral lesion of the hip (wave sign), in which the cartilage detaches from the subchondral bone leading to a "bag lesion" and chondral flaps. Outerbridge is the most used chondral lesion classification system; although delamination was not originally described, it could be considered as a type III. Konan et al. recently described a new classification system for hip chondral lesions, including the wave sign, delamination, and chondrolabral lesions considering extension and location [4].

The frequency of chondral lesions in hip arthroscopy for femoroacetabular impingement is high, up to 67.3 % of the patients, as described by Nepple et al. Risk factors for the presence of a chondral lesion are male, tonnis 1 or 2, and alpha angle over 50° [5].

Standard Treatment for Chondral Lesions

The arthroscopic treatment of chondral lesions of the hip is limited to excision (rim trimming and femoral neck osteoplasty), debridement, chondroplasty, and microfractures. Rim trimming and femoral neck osteoplasty could lead to the complete excision of the chondral lesion if located in the overcoverage area. When the chondral damage extends beyond the resection area, the treatment of choice will be chosen according to Outerbridge or Konan classifications as follows:

– *Type I or II*

  The treatment of choice in this type of lesions is thermal chondroplasty. It has shown to be a safe technique for closed chondral lesions leading to morphological changes with better structural characteristics than mechanical debridement [1, 6–8].

– *Delamination*

  Delamination represents a treatment challenge among chondral lesions. Excising such an area of chondral instability seems an unnecessary surgical maneuver, particularly if the articular cartilage itself may contain a significant number of viable chondrocytes [9]. The main objective is the reattachment of the cartilage to the underlying subchondral bone. This could be achieved with transchondral microfractures, forming an adherent retrolabral clot or with the use of an adhesive such as fibrin glue. Tzaveas and Villar report on a series of 19 patients treated with fibrin adhesive showing improvement in pain and function at 6 months and one year after surgery [10].

– *Type III or IV (full-thickness chondral lesion)*

  The indications for microfracture of the hip are similar to the knee and include focal and contained lesions, typically less than 2–4 cm^2 in size (Outerbridge III or IV), including delamination. Microfracture is a marrow-stimulating procedure that brings undifferentiated stem cells from a subchondral perforation into the chondral defect [11]. A clot formed in the microfractured area provides an environment for both pluripotent marrow cells and mesenchymal stem cells to differentiate into stable fibrocartilaginous tissue [12, 13]. Several studies had shown good midterm results with this technique; however, we know that this fibrocartilaginous tissue does not have the required mechanical properties and eventually will fail, leading to advanced chondral damage and osteoarthritis [13, 14].

Novel Treatments for Chondral Lesions

As we mentioned before, the fibrocartilage newly formed at the microfractured area is a low quality tissue; therefore we will describe some techniques, based on stem cell therapy, that may lead to a better quality hyaline-like cartilage. The use of these novel technologies has demonstrated promising results in animal and clinical studies [15–18].

Stem Cells: Background

There are some important concepts regarding stem cells that we think could be useful to discuss

Fig. 1 Autologous mesenchymal stem cell concentrate: (**a**) autologous bone marrow autograft, (**b**) centrifugation process with a single spin, and (**c**) layer separation by a density filter and identification of mononuclear cells layer. (**d**) Autologous mononuclear cells concentrate – final view

prior to clinical application, as their origin, differentiation potential, and possible adverse effects may vary according to each of the subtypes. Stem cells have the ability to divide indefinitely in culture and the potential to give rise to mature tissue. These can be classified according to their origin and potentiality. Thus we have, by origin, *embryonic stem cells* that can be divided by its potentiality in **totipotent** (originating from the zygote and early blastomers of an embryo) and **pluripotent** (derived from the blastocyst inner cell mass) and *somatic or adult stem cells*, which can be found in a fully developed organism. These may be *mesenchymal* or *hematopoietic* according to the tissue that may give rise and are referred as **multipotent** [19].

Embryonic stem cells have greater potential for differentiation, response, and growth than somatic stem cells and, in the past years, have been widely studied in animals [20]. However, this vast potential could generate an excessive response to local stimuli at implantation site, giving rise to teratomas [21, 22].

Hematopoietic stem cells can be found mainly in the bone marrow of adult specimens, in a proportion of 1 in 10,000 nucleated cells. Its clinical application has been extensively studied in the management of hematological diseases.

Mesenchymal stem cells (MSC's) are also found in the bone marrow of adult specimens, in a proportion of 1 in 100.000 nucleated cells, decreasing with aging [23]. These cells have differentiation potential into bone, fat, chondral, tendinous, ligamentous, neural, and muscular tissue, according to the growth factors released by the injury site.

MSC's from adult specimens can be obtained from fat tissue, bone marrow, and other tissues; however, bone marrow as a donor site has several advantages compared to other locations because of the ease of procurement, purity of the sample, and greater cell growth potential.

The cells are obtained through a trocar from the iliac crest (2 cm cephalic to the anterior superior iliac spine), extracting a bone marrow sample. The latter is poured into a tube equipped with a filter and centrifuged in order to obtain layers by density gradients. The mononuclear cell layer is then identified and aspirated, obtaining a mononuclear cell concentrate (MCC) in which MSCs are found. This concentrate can be applied directly to the receptor site or may be processed in a specialized laboratory for mesenchymal stem cell expansion and differentiation (Fig. 1).

Summary

Hip chondral lesions are a frequent finding in hip arthroscopy for FAI. Treatment is often difficult and insufficient. Novel strategies based on cell regenerative therapy represent an interesting treatment alternative. We reviewed the general concepts of hip chondral lesions treatment and the stem cell background.

In the next part of the chapter, we will discuss different treatment options for hip chondral lesions with the use of mesenchymal stem cells.

References

1. Yen YM, Kocher MS. Chondral lesions of the hip: microfracture and chondroplasty. Sports Med Arthrosc. 2010;18(2):83–9.
2. Bedi A, Lynch EB, Sibilsky Enselman ER, et al. Elevation in circulating biomarkers of cartilage damage and inflammation in athletes with femoroacetabular impingement. Am J Sports Med. 2013;41:2585–90.
3. Johnston TL, Schenker ML, Briggs KK, Philippon MJ. Relationship between offset angle alpha and hip chondral injury in femoroacetabular impingement. Arthroscopy. 2008;24(6):669–75.
4. Konan S, Rayan F, Meermans G, Witt J, Haddad FS. Validation of the classification system for acetabular chondral lesions identified at arthroscopy in patients with femoroacetabular impingement. J Bone Joint Surg Br. 2011;93(3):332–6.
5. Nepple JJ, Carlisle JC, Nunley RM, Clohisy JC. Clinical and radiographic predictors of intra-articular hip disease in arthroscopy. Am J Sports Med. 2011;39(2):296–303.
6. Kaplan LD, Chu CR, Bradley JP, Fu FH, Studer RK. Recovery of chondrocyte metabolic activity after thermal exposure. Am J Sports Med. 2003;31(3):392–8.
7. Lotto ML, Wright EJ, Appleby D, Zelicof SB, Lemos MJ, Lubowitz JH. Ex vivo comparison of mechanical versus thermal chondroplasty: assessment of tissue effect at the surgical endpoint. Arthroscopy. 2008;24(4):410–5.
8. Edwards RB, Lu Y, Cole BJ, Muir P, Markel MD. Comparison of radiofrequency treatment and mechanical debridement of fibrillated cartilage in an equine model. Vet Comp Orthop Traumatol. 2008;21(1):41–8.
9. Voss JR, Lu Y, Edwards RB, Bogdanske JJ, Markel MD. Effects of thermal energy on chondrocyte viability. Am J Vet Res. 2006;67(10):1708–12.
10. Tzaveas AP, Villar RN. Arthroscopic repair of acetabular chondral delamination with fibrin adhesive. Hip Int. 2010;20(1):115–9.
11. Frisbie DD, Oxford JT, Southwood L, Trotter GW, Rodkey WG, Steadman JR, Goodnight JL, McIlwraith CW. Early events in cartilage repair after subchondral bone microfracture. Clin Orthop Relat Res. 2003;407:215–27.
12. Steadman JR, Rodkey WG, Rodrigo JJ (2001) Microfracture: surgical technique and rehabilitation to treat chondral defects. Clin Orthop Relat Res 391 (Suppl):S362–9
13. Steadman JR, Rodkey WG, Briggs KK. Microfracture to treat full-thickness chondral defects: surgical technique, rehabilitation, and outcomes. J Knee Surg. 2002;15(3):170–6.
14. Philippon MJ, Schenker ML, Briggs KK, Maxwell RB. Can microfracture produce repair tissue in acetabular chondral defects? Arthroscopy. 2008;24(1):46–50.
15. McIlwraith CW, Frisbie DD, Rodkey WG, Kisiday JD, Werpy NM, Kawcak CE, Steadman JR. Evaluation of intra-articular mesenchymal stem cells to augment healing of microfractured chondral defects. Arthroscopy. 2011;27(11):1552–61.
16. Gobbi A, Karnatzikos G, Scotti C, Mahajan V, Mazzucco L, Grigolo B. One-step cartilage repair with bone marrow aspirate concentrated cells and collagen matrix in full-thickness knee cartilage lesions: results at 2-years follow-up. Cartilage. 2011;2(3):286–99.
17. De Girolamo L, Bertolini G, Cervellin M, Sozzi G, Volpi P. Treatment of chondral defects of the knee with one step matrix-assisted technique enhanced by autologous concentrated bone marrow: in vitro characterization of mesenchymal stem cells from iliac crest and subchondral bone. Injury. 2010;41(11):1172–7.
18. Haleem A, El Singergy A, Sabry D, Atta H, Rashed L, Chu C, El Shewy M, Azzam A, Abdel Aziz M. The clinical use of human culture–expanded autologous bone marrow mesenchymal stem cells transplanted on platelet-rich fibrin glue in the treatment of articular cartilage defects: a pilot study and preliminary results. Cartilage. 2010;1(4):253–61.
19. Broxmeyer HE. Insights into the biology of cord blood stem/progenitor cells. Cell Prolif. 2011;44 Suppl 1:55–9.
20. Leeb C, Jurga M, McGuckin C, Forraz N, Thallinger C, Moriggl R, Kenner L. New perspectives in stem cell research: beyond embryonic stem cells. Cell Prolif. 2011;44 Suppl 1:9–14.
21. Kimura T, Nakano T. Induction of pluripotency in primordial germ cells. Histol Histopathol. 2011;26(5):643–50.
22. Fong CY, Gauthaman K, Bongso A. Teratomas from pluripotent stem cells: a clinical hurdle. J Cell Biochem. 2010;111(4):769–81.
23. Caplan AI. Adult mesenchymal stem cells for tissue engineering versus regenerative medicine. J Cell Physiol. 2007;213(2):341–7.

Surgical Technique: Osteochondral Autograft Transfer and Osteochondral Allograft Transplant for Preservation of the Femoral Head and Acetabulum

92

Luke Spencer-Gardner, Aaron Krych, and Bryan T. Kelly

Contents

Abstract

Few surgical options exist to treat large chondral and osteochondral defects involving the articular surface of the hip. This challenging problem is often encountered in young, active patients whom may be best served with biologic joint preservation. Although restorative procedures for chondral and osteochondral defects in other joints such as the knee and ankle have demonstrated excellent outcomes in the literature, similar procedures for the articular surface of the hip have lagged behind. Therefore, restorative procedures in the hip present a tremendous area of opportunity for progress in the realm of hip preservation surgery. Recent advancements in the field, including the development of a safe anatomic and reliable surgical approach to the hip, have allowed the application of restorative procedures for chondral defects of the hip. This chapter outlines the indications and the surgical technique of two surgical options for hip preservation in the setting of large chondral or osteochondral defects of the femoral head and acetabulum: osteochondral autograft transfer and osteochondral allograft transplant. The current body of literature as it pertains to the use of these techniques in the hip is also reviewed.

L. Spencer-Gardner (✉) • A. Krych
Department of Orthopedic Surgery, Mayo Clinic, Rochester, MN, USA
e-mail: spencergardner.luke@mayo.edu; krych.aaron@mayo.edu

B.T. Kelly
Department of Orthopaedic Surgery, Center for Hip Preservation Surgery, Hospital for Special Surgery, New York, NY, USA

Weill Medical College of Cornell University, New York, NY, USA
e-mail: kellyb2847@hotmail.com

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_101

Introduction

Chondral and osteochondral defects of the hip are increasingly recognized as an intra-articular source of hip pain. While purely chondral lesions are frequently found on MRI and encountered at the time of arthroscopy, osteochondral defects are rare. Posttraumatic incongruity as sequelae of femoral head fractures, acetabular fractures, and fracture dislocations of the hip can result in damage to the articular surface. The outcome is often pain and early secondary degenerative changes, with limited surgical options for salvage, short of total hip arthroplasty [1–3]. Additionally, nontraumatic damage to the articular surface of the hip can occur secondary to tumors, osteochondritis dissecans, avascular necrosis of the femoral head, Legg-Calve-Perthes disease, slipped capital femoral epiphysis, hip dysplasia, and femoroacetabular impingement. Management options for chondral lesions of the hip include chondroplasty, microfracture, osteochondral autograft transfer (OAT), osteochondral allograft transplant (OCA), and autologous chondrocyte implantation (ACI) [4, 5]. This chapter will focus on the implementation of OAT and OCA as treatment options for osteochondral defects of the femoral head and acetabulum. OAT involves the harvest of a healthy osteochondral plug from the periphery of a joint, in an area of low contact stress and transferring the plug to the zone of chondral or osteochondral injury. When multiple plugs are utilized, the technique is known as mosaicplasty. OCA utilizes fresh-stored cadaveric donor bone and living articular cartilage for subsequent transplant into larger defects.

To date, there are only a handful of case reports and small case series on OAT and OCA as treatment options for the hip. When indicated, OAT and OCA provide the hip preservation surgeon with techniques for joint salvage in younger patients that might otherwise have few surgical options short of total hip arthroplasty.

Indications

Injury to the articular surface of the hip occurs along a spectrum, with defects ranging from focal chondral delamination often encountered with cam-type femoroacetabular impingement to large post-traumatic osteochondral defects [6, 7]. Indications for the selective use of cartilage preserving techniques in hip preservation surgery have been extrapolated from data regarding their use in other large joints such as the knee. Factors including patient age, activity level, and size of the defect should all be weighed when selecting the appropriate treatment option.

Focal partial-thickness tears of the cartilage are best managed with chondroplasty. Full-thickness chondral defects measuring less than 2–4 cm^2 with intact subchondral bone are accepted indications for microfracture [4]. Indications for microfracture may be extended for larger defects in a low-demand, older patient without diffuse degenerative changes. OAT may also be used for defects measuring 1–4 cm^2 with intact subchondral bone in the younger, high-demand patient [8]. Data published on OAT for larger defects in the knee up to 8–9 cm^2 resulted in increased donor site morbidity [9], and in one series where larger defects of the femoral head were treated with mosaicplasty, the results were comparatively poor [10].

ACI has been reported as a treatment option to address an osteochondral defect of the femoral head [11]. It is typically reserved for larger defects measuring greater than 2–4 cm^2 with chondral defects or osteochondral defects with minimal bone loss.

Chondral defects without intact subchondral bone (bone loss) necessitate the use of osteochondral grafts. Smaller osteochondral defects measuring less than 1–4 cm^2 may be managed with OAT. The requirement of an autologous donor site limits the application of this technique to smaller defects [12]. As outlined in the knee literature, larger chondral defects greater than 2.5 cm^2 and defects with substantial bone loss should be managed with OCA [13]. One advantage of this approach over OAT is the lack of donor site morbidity. However, with the use of allograft bone, there are the disadvantages of graft availability, surgical scheduling, and potential risk of disease transmission.

Patient Evaluation and Preoperative Planning

Patients presenting with activity-related groin pain require a thorough evaluation, beginning with a detailed history and physical examination. Radiographs are scrutinized for structural abnormalities that may need to be addressed at the time of restorative procedures. Gadolinium-enhanced magnetic resonance imaging of the hip should be obtained with special attention paid to chondral defects and CT of the hip should be considered in the setting of osteochondral defects (Fig. 1). Intra-articular injection can be diagnostic and therapeutic and should be considered as a component of initial nonoperative management.

Preoperative planning is paramount when OAT and OCA are considered options for hip preservation surgery. When OAT is indicated, consideration must be given to the planned donor site. Reported donor sites for treatment of articular defects of the hip include low-weight-bearing areas such as the inferior aspect of the femoral head and the femoral head and neck junction in instances where a concomitant cam lesion exists [5, 8, 14–16]. Alternatively, the lateral margin of the femoral trochlea in the knee may be used [8, 16, 17]. The advantage of harvesting osteochondral plugs from a local donor site in the hip are implicit in that there is no requirement for a separate incision, limiting donor site morbidity. When a large osteochondral lesion necessitates OCA, arrangements must be made for a fresh allograft that should ideally be stored at 4 ºC for no longer than 28 days, requiring some flexibility in scheduling surgery [18]. Allograft options reported in the literature for the acetabular defects include the medial tibial plateau and the acetabulum [19]; however, the availability of fresh acetabulum allograft can be particularly difficult. For defects of the femoral head, the use of allograft femoral head has been described in a number of studies [20–23].

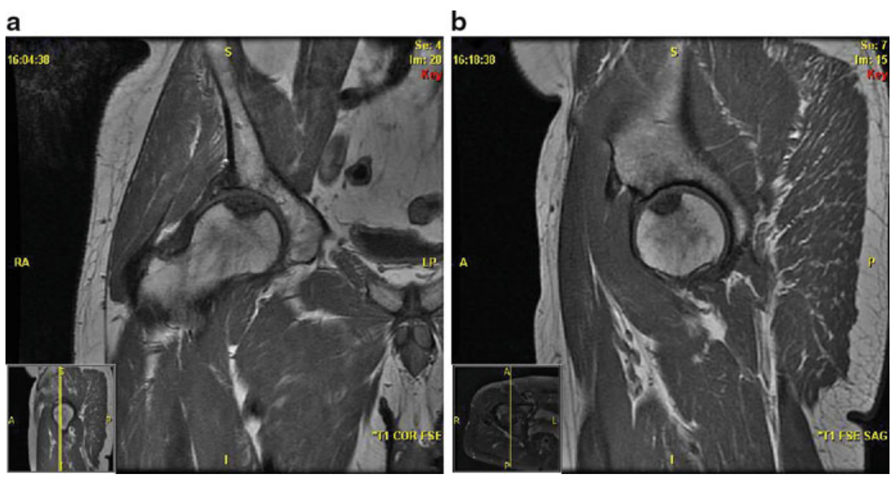


Fig. 1 MRI of the right hip. T_1-weighted coronal (**a**) and sagittal (**b**) images demonstrating an osteochondral defect in the weight-bearing region of the right femoral head

Surgical Technique

Articular defects amenable to OCA or OAT require an open approach to the hip. Although various approaches have been described in the literature for restorative cartilage procedures, the surgical hip dislocation, as described by Ganz, offers full access to the femoral head and acetabulum while preserving blood flow to the femoral head and is therefore the preferred approach [24, 25]. After safely exposing the femoral head and acetabulum, the typical sequence of events is as follows: preparation of the recipient site, graft harvest from the donor site, and finally, placement of graft into the defect. Any one of the commercially available systems may be used to complete the steps as outlined below for OAT and OCA.

OAT and Mosaicplasty

Preparation of the recipient site initially involves debridement of the defect to stable margins of surrounding bone and cartilage. The defect is then sized to determine the appropriate dimensions and configuration of the donor plug, or plugs that may be required. When mosaicplasty is required, a 2–3 mm bone bridge should be preserved between donor plug areas whenever possible, to avoid graft instability and convergence (Fig. 2). Finalization of the recipient site by drilling to the appropriate size and depth is then completed. The recipient site may be underdrilled by 1 mm in some systems to allow for press-fit impaction of the graft. Donor plug harvest sites for the hip include local and remote options as previously outlined. When harvesting the graft, care should be taken to ensure the harvester remains perpendicular to the articular surface as this increases the likelihood of obtaining a congruent surface at the time of graft impaction. The harvester is advanced with gentle blows of a mallet to the appropriate depth, and the graft is then removed. Graft insertion is accomplished according to the system used. Typically there is a plunger that assists with the introduction of the graft into the defect, with a final impactor for fine-tuning. Throughout the introduction, care should be taken to avoid forcefully impacting the graft as this can lead to chondrocyte damage. Finally, back filling of the donor site can be considered at this time with either osteochondral allograft or a synthetic bone void filler [8]. When transferring multiple plugs, it is preferred to harvest and insert each plug fully before proceeding with the next plug.

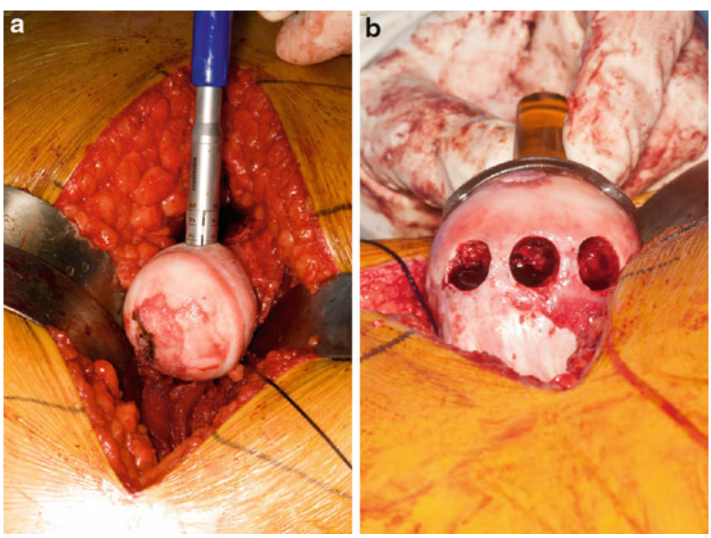


Fig. 2 Intraoperative photographs demonstrating the use of mosaicplasty for an osteochondral defect of the femoral head. (**a**) Osteochondral plug harvest from a non-weight-bearing region of the proximal femur. (**b**) Care should be taken to preserve a bone bridge of at least 2–3 mm between harvest sites

OCA

Preparation of the recipient site for OCA begins as outlined for OAT. Sizing for OCA is accomplished with a cannulated cylindrical sizer. A guide pin is placed into the bone through the sizer and the recipient site is reamed over the guide pin to the specified depth based on the system used. The recipient site should be marked with a reference point, and the depth of the defect after reaming should be measured in four quadrants. Harvest from the donor site begins with placement of the cylindrical sizer and guidewire into the portion of the allograft that will provide the best fit at the recipient site. When using allograft of the same bone (i.e., femoral head allograft for femoral head defect or acetabular allograft for the acetabulum), reference points may be used to match the location of donor and recipient sites. When alternative options are used (i.e., tibial plateau for acetabulum), the best fit should be sought based on surface characteristics The allograft is harvested with a cylindrical drill and the depth is fine-tuned based on the measurements of the four quadrants measured at the recipient site. The deep edges of the graft may be tapered with a rongeur prior to insertion. The allograft is then press fit, taking care to orient the graft ensuring the depth of the graft matches the depth of the recipient site in all four quadrants as measured (Figs. 3 and 4).

Postoperative Care

The postoperative course begins with the implementation of continuous passive motion (CPM) immediately after surgery. Throughout the first 8 weeks after surgery, CPM is advised for

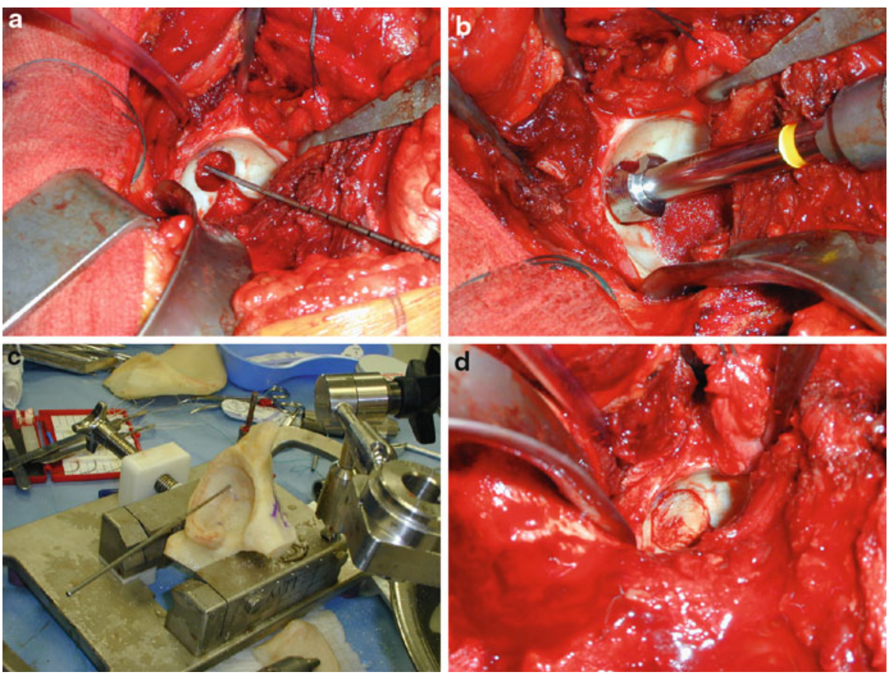


Fig. 3 Intraoperative photograph demonstrating the use of an acetabular osteochondral allograft for an acetabular articular surface defect. (**a**) A cylindrical drill is used over a guidewire to prepare the recipient site. (**b**) Recipient site after preparation. (**c**) Graft table setup shows donor acetabulum with guidewire in place. Note the reference points on the donor acetabulum used to match the location of the donor and recipient sites. (**d**) Press-fit graft placement, reconstituting the articular surface of the acetabulum (Reproduced from Krych AJ, Lorich DG, Kelly BT. Treatment of focal osteochondral defects of the acetabulum with osteochondral allograft transplantation. *Orthopedics*. 2011 Jul;34(7):e307–11., with permission from SLACK, Inc)

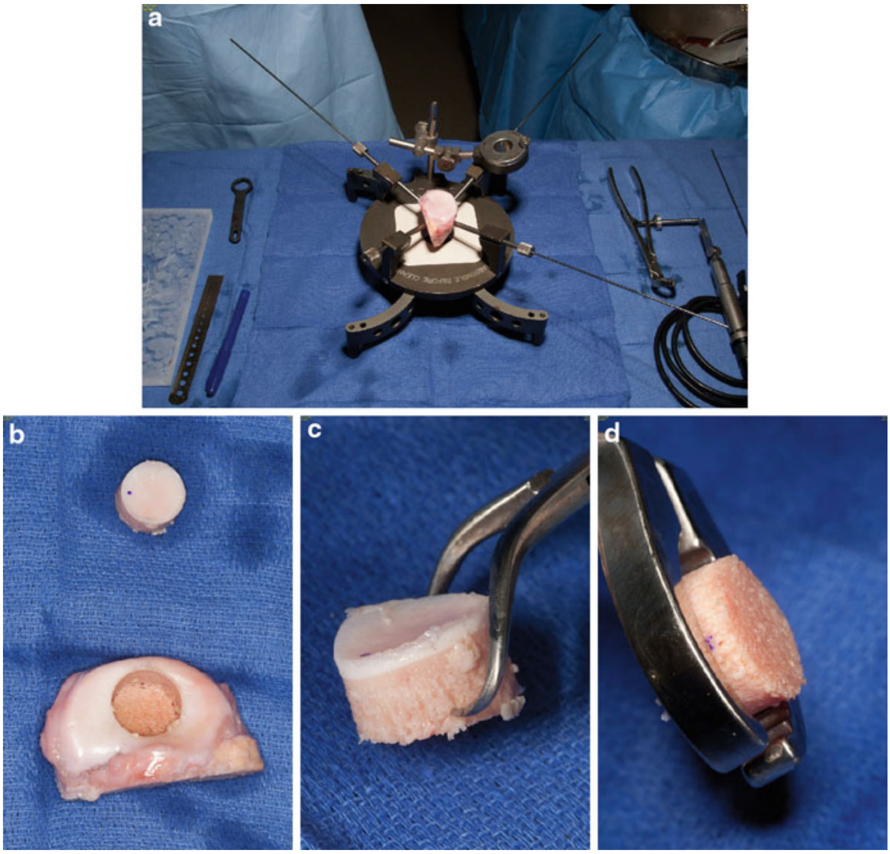


Fig. 4 Intraoperative photographs demonstrating the use of a medial tibial plateau to fill an osteochondral defect. (**a**) Graft table setup. (**b**) Osteochondral dowel after harvest. (**c**) Osteochondral dowel profile demonstrating best fit contour to match the recipient site. (**d**) Fine-tuning the depth of the osteochondral dowel to ensure the donor graft matches the recipient site

8–10 h per day. Weight bearing is restricted to toe touch for the first 8 weeks postoperatively with a gradual advancement to weight bearing as tolerated allowed from that point onwards. After 6 months (OAT) to 12 months (OCA), MRI of the hip is obtained, and if graft incorporation is demonstrated, impact activities including running may be resumed.

Outcomes

Limited clinical data exists regarding the use of OAT and OCA in the treatment of chondral defects of the hip. These data are summarized in Table 1. While preliminary data is promising in some of the more recent series, further studies are needed to verify the long-term viability of these techniques for hip preservation surgery.

Summary

Osteochondral defects of the hip present a unique set of challenges to the hip preservation surgeon. At early follow-up, OAT and OCA show promise in improving pain and function and provide an alternative to total hip arthroplasty for the young, active patient with osteochondral lesions of the femur and acetabulum. Although the current body of literature is limited, these data can be used to help guide the appropriate application of OAT and

Table 1 Literature review

Authors/year	LOE	Defect location	Etiology	Patient characteristics	Procedure	Approach	Outcome measures	Follow-up	Results/conclusion
Meyers et al. [22]	4	Femoral head	AVN	21 hips in 20 patients	Fresh OCA	Gibson	d'Aubigne and Postel hip scores	Min.18 months	71 % success rate overall (15/21)[a]
				Age: 18–44 years	Grafts composed of osteochondral shell with underlying iliac crest bone chips				50 % success rate with steroid-induced AVN (4/8)
					Donor: allograft femoral head				85 % success rate in nonsteroid group (11/13)
Meyers [23]	4	Femoral head	AVN, femoral head fracture	25 hips in 21 patients	As above	Gibson	None reported	9–63 months	Follow-up study
				Age: not reported					68 % success rate overall (17/25)[b]
									80 % success rate in nonsteroid group (12/15)
Meyers [26]	4	Femoral head	AVN, post-traumatic	75 hips in 72 patients	As above	Gibson	None reported	Min. 1 year	Success not defined
				Age: 16–46 years					80.5 % successful at 1 year
									13 failed and had THA within 1 year
Hart et al. [17]	4	Femoral head	Intra-articular screw placement for acetabular fracture fixation	1 patient	Mosaicplasty	Kocher-Langenbeck	HHS	6 months	HHS
				Age: 28 years	Defect size: 14 mm diameter				Preoperative 69
					Donor site: lateral condyle of femur				Postoperative 100
					Number of plugs: 4				

(*continued*)

Table 1 (continued)

Authors/year	LOE	Defect location	Etiology	Patient characteristics	Procedure	Approach	Outcome measures	Follow-up	Results/conclusion
Rittmeister et al. [10]	4	Femoral head	AVN	5 Patients	Mosaicplasty	Smith-Peterson	THA as endpoint	Mean 57 months	4/5 cases failed with THA performed at a mean of 49 months
				Age: 34–51	Defect size: 1.4–9.0 cm^2				1/5 cases successful. 36-year-old with smallest defect: 1.2 × 1.2 cm
					Donor site: ipsilateral femoral head, ipsilateral knee				HHS
					Number of plugs: 1–3				Postoperatively 100
Sotereanos et al. [14]	4	Femoral head	AVN (steroid induced)	1 patient	Mosaicplasty	Anterolateral approach	HHS, SMFA	66 months	HHS
				Age: 36 years	Defect size: 1.5 cm diameter				Preoperative 45
					Donor site: inferior femoral head				Postoperative 96
					Number of plugs: 3				SMFA
									0 (higher scores = more dysfunction)
Evans et al. [20]	4	Femoral head	OCD	1 patient	Fresh OCA	SHD	HHS	12 months	HHS
				Age: 32 years	Defect size: 23 mm diameter				Preoperative 66
					Donor: allograft femoral head				Postoperative 94
									X-ray: near-complete incorporation of graft, maintenance of joint space

Stilli et al. [21]	4	Femoral head	Chondroblastoma	1 patient	Fresh OCA	Smith-Peterson	Clinical data	7.5 years	No tumor recurrence
				Age: 14 years	Graft fixation with a 4.5 Herbert screw				No pain
					Lesion treated with curettage and phenol				X-ray: no femoral head collapse, complete allograft incorporation, early degenerative changes seen in the acetabulum
					Defect size: not recorded				
					Donor: allograft femoral head				
Bastian et al. [15]	4	Femoral head	Femoral head impaction fracture	1 patient	Osteochondral shell autograft	SHD	HHS	2 years	HHS 100
				Age: 48 years	Defect size: 10 × 15 × 5 mm				MRI: no signs of AVN Ulcerations in transplanted cartilage and adhesions between donor site and capsule were present
					Donor site: anterior head-neck junction of femur				
					Fixation with countersunk 2 mm cortical screws				
Nousiainen et al. [7]	4	Femoral head	Femoral head fracture (Pipkin IV)	1 patient	Frozen OCA	SHD	HHS, SF-36	46 months	HHS 100
				Age: 18 years	Fixation of fracture fragments and OCA with absorbable screws				SF-36 81
					Donor: frozen allograft femoral head				MRI: no AVN. Subchondral cyst and sclerosis was present with diffuse partial- to full-thickness cartilage loss

(*continued*)

Table 1 (continued)

Authors/year	LOE	Defect location	Etiology	Patient characteristics	Procedure	Approach	Outcome measures	Follow-up	Results/conclusion
Nam et al. [16]	4	Femoral head	Femoral head fracture (Pipkin II)	2 patients	Mosaicplasty combined with fracture fixation	SHD	Clinical data	1 year	No pain, return to pre-injury activities in each case
				Age: 15, 21 years	Defect sizes:				MRI: incorporation of grafts
					2 cm × .8 cm, 1 cm diameter				
					Donor sites: inferior femoral head, lateral condyle of femur				
					Number of plugs: 3, 1				
Girard et al. [27]	4	Femoral head	LCPD, SEP, EP	10 patients	Mosaicplasty	SHD	HHS, Merle d'Aubigne, clinical data	Mean: 29 months	Mean Merle d'Aubigne
				Age: 15–21 years	Defect size: mean 4.8 cm^2				Preoperative 10.5
					Donor site: inferior femoral head				Postoperative 15.5
					Number of plugs: 2–4				Mean HHS
									Preoperative 52.8
									Postoperative 79.5
									CT arthrography 6 months postoperative demonstrated incorporation in all cases
Sen et al. [28]	4	Femoral head	Giant cell tumor	1 patient	Frozen OCA	Smith-Peterson	HHS	3.5 years	HHS
				Age: 22 years	En bloc marginal resection and OCA				Preoperative 54
					Tumor size:				Postoperative 100
					5.2 × 3.3 cm				

Krych et al. [19]	4	Acetabulum	Periacetabular cyst, fibrous dysplasia s/p curettage and cementing	2 patients	Fresh OCA	SHD	HHS	Min. 2 years (24, 42 months)	HHS
				Age: 24, 32 years	Defect size:		HOS		Preoperative 75, 79
					18 × 18 mm				Postoperative 97, 100
					12 × 10 mm				HOS for ADLs and sports
					Donor site: allograft acetabulum, medial tibial plateau				Postoperative: 100, 100
									MRI: incorporation of allograft with maintenance of joint congruity
Emre et al. [29]	4	Femoral head	LCP	1 Patient	Mosaicplasty	Smith-Peterson	HHS	24 weeks	HHS
				Age: 22 years	Defect size: not reported				Preoperative 49
					Donor site: ipsilateral knee				Postoperative 96
					Number of plugs: 3				
Krych et al. [8]	4	Femoral head	Posterior hip dislocation	2 patients	Mosaicplasty	SHD	HHS	Mean: 4.3 years	HHS
				Age: 15, 29 years	Osteochondral fracture fixation with absorbable screws in both cases followed by mosaicplasty		HOS		Preoperative Not reported
					Defect size:				Postoperative 96, 100
					2 cm × 5–8 mm				HOS for ADLs and sports
					1 cm × 2 cm				Postoperative 100, 100
					Donor site: ipsilateral knee				MRI: incorporation of autograft at 6 months
					Number of plugs: 3, 2				

AVN avascular necrosis, *OCA* osteochondral allograft transplant, *HHS* Harris hip score, *SMFA* short musculoskeletal function assessment, *OCD* osteochondritis dissecans, *SHD* surgical hip dislocation, *MVC* motor vehicle collision, *LCP* Legg-Calve-Perthes disease, *SEP* spondyloepiphyseal dysplasia, *EP* epiphyseal dysplasia, *HOS* hip outcomes score

[a]Success defined as scores >3 in function (walks long distances with a cane, limited without a cane) and >4 in pain (pain with activity, not at rest)

[b]Success defined as “patient functioning with minimal pain, without external support, and with use of nonarcotic analgesics occasionally for relief of discomfort”

OCA in hip joint preservation moving forward. OAT and OCA appear to demonstrate better results in young patients with smaller osteochondral defects, without generalized degenerative changes. When possible, autogenous osteochondral grafts may be taken from either the ipsilateral hip or knee with little donor site morbidity. When defect size and bone loss dictate, use of fresh osteochondral allograft is recommended. By maintaining these principles and following the indications as described, good short-term outcomes can be expected.

References

1. Ranawat A, Zelken J, Helfet D, Buly R. Total hip arthroplasty for posttraumatic arthritis after acetabular fracture. J Arthroplasty. 2009;24(5):759–67.
2. Tonetti J, Ruatti S, Lafontan V, Loubignac F, Chiron P, Sari-Ali H, et al. Is femoral head fracture-dislocation management improvable: a retrospective study in 110 cases. Orthop Traumatol Surg Res. 2010;96(6):623–31.
3. Mears DC, Velyvis JH, Chang CP. Displaced acetabular fractures managed operatively: indicators of outcome. Clin Orthop Relat Res. 2003;407:173–86.
4. Yen YM, Kocher MS. Chondral lesions of the hip: microfracture and chondroplasty. Sports Med Arthrosc. 2010;18(2):83–9.
5. Petit CJ, Philippon MJ. Arthroscopic management of osteochondral defects of the hip. Op Tech Sports Med. 2008;16(4):194–200.
6. Beck M, Kalhor M, Leunig M, Ganz R. Hip morphology influences the pattern of damage to the acetabular cartilage: femoroacetabular impingement as a cause of early osteoarthritis of the hip. J Bone Joint Surg Br. 2005;87(7):1012–8.
7. Nousiainen MT, Sen MK, Mintz DN, Lorich D, Paul O, Buly RL, et al. The use osteochondral allograft in the treatment of a severe femoral head fracture. J Orthop Trauma. 2010;24(2):120–4.
8. Krych AJ, Lorich DG, Kelly BT. Osteochondral autograft transfer for a posttraumatic osteochondral defect of the femoral head. Am J Orthop. 2012;41(10): 472–476.
9. Hangody L, Fules P. Autologous osteochondral mosaicplasty for the treatment of full-thickness defects of weight-bearing joints: ten years of experimental and clinical experience. J Bone Joint Surg Am. 2003;85-A Suppl 2:25–32.
10. Rittmeister M, Hochmuth K, Kriener S, Richolt J. Five-year results following autogenous osteochondral transplantation to the femoral head. Orthopade. 2005; 34(4):320, 2–6.
11. Akimau P, Bhosale A, Harrison PE, Roberts S, McCall IW, Richardson JB, et al. Autologous chondrocyte implantation with bone grafting for osteochondral defect due to posttraumatic osteonecrosis of the hip–a case report. Acta Orthop. 2006;77(2):333–6.
12. Jakob RP, Franz T, Gautier E, Mainil-Varlet P. Autologous osteochondral grafting in the knee: indication, results, and reflections. Clin Orthop Relat Res. 2002;401:170–84.
13. McCulloch PC, Kang RW, Sobhy MH, Hayden JK, Cole BJ. Prospective evaluation of prolonged fresh osteochondral allograft transplantation of the femoral condyle: minimum 2-year follow-up. Am J Sports Med. 2007;35(3):411–20.
14. Sotereanos NG, DeMeo PJ, Hughes TB, Bargiotas K, Wohlrab D. Autogenous osteochondral transfer in the femoral head after osteonecrosis. Orthopedics. 2008;31(2):177.
15. Bastian JD, Buchler L, Meyer DC, Siebenrock KA, Keel MJ. Surgical hip dislocation for osteochondral transplantation as a salvage procedure for a femoral head impaction fracture. J Orthop Trauma. 2010; 24(12):e113–8.
16. Nam D, Shindle MK, Buly RL, Kelly BT, Lorich DG. Traumatic osteochondral injury of the femoral head treated by mosaicplasty: a report of two cases. HSS J. 2010;6(2):228–34.
17. Hart R, Janecek M, Visna P, Bucek P, Kocis J. Mosaicplasty for the treatment of femoral head defect after incorrect resorbable screw insertion. Arthroscopy. 2003;19(10):E1–5.
18. LaPrade RF, Botker J, Herzog M, Agel J. Refrigerated osteoarticular allografts to treat articular cartilage defects of the femoral condyles. A prospective outcomes study. J Bone Joint Surg Am. 2009; 91(4):805–11.
19. Krych AJ, Lorich DG, Kelly BT. Treatment of focal osteochondral defects of the acetabulum with osteochondral allograft transplantation. Orthopedics. 2011;34(7):e307–11.
20. Evans KN, Providence BC. Case report: fresh-stored osteochondral allograft for treatment of osteochondritis dissecans the femoral head. Clin Orthop Relat Res. 2010;468(2):613–8.
21. Stilli S, Marchesini Reggiani L, Boriani L, Donzelli O. Osteochondral allograft in a femoral head chondroblastoma: a case report. Hip Int. 2010; 20(2):280–3.
22. Meyers MH, Jones RE, Bucholz RW, Wenger DR. Fresh autogenous grafts and osteochondral allografts for the treatment of segmental collapse in osteonecrosis of the hip. Clin Orthop Relat Res. 1983;174:107–12.
23. Meyers MH. Resurfacing of the femoral head with fresh osteochondral allografts. Long-term results. Clin Orthop Relat Res. 1985(197):111–4.
24. Ganz R, Gill TJ, Gautier E, Ganz K, Krugel N, Berlemann U. Surgical dislocation of the adult hip a technique with full access to the femoral head and acetabulum without the risk of avascular necrosis. J Bone Joint Surg Br. 2001;83(8):1119–24.

25. Gautier E, Ganz K, Krugel N, Gill T, Ganz R. Anatomy of the medial femoral circumflex artery and its surgical implications. J Bone Joint Surg Br. 2000;82(5):679–83.
26. Meyers MH. The surgical treatment of osteonecrosis of the femoral head with an osteochondral allograft. Acta Orthop Belg. 1999;65 Suppl 1:66–7.
27. Girard J, Roumazeille T, Sakr M, Migaud H. Osteochondral mosaicplasty of the femoral head. Hip Int. 2011;21(5):542–8.
28. Sen RK, Tripathy SK, Meena DS, Gupta N. Osteochondral allograft transplantation after excision of femoral head giant cell tumour: a case report. Acta Orthop Belg. 2011;77(4):555–60.
29. Emre TY, Cift H, Seyhan B, Ceyhan E, Uzun M. Mosaicplasty for the treatment of the osteochondral lesion in the femoral head. Bull NYU Hosp Jt Dis. 2012;70(4):288–90.

Labral Deficiency

93

Scott C. Faucett and Marc J. Philippon

Contents

Abstract

Damage to the labrum has been recognized as a source of pain and morbidity surrounding the hip. For many patients the initial treatment was labral debridement. This loss of labral tissue subsequently can lead to a dysfunctional deficient labrum. Labral dysfunction can lead to overloading of the articular cartilage of the hip and may be a precursor of osteoarthritis. Labral deficiency can also be related to adhesions, hip instability, and dysplasia. For patients with loss of joint space, injections made provide relief while waiting for total hip replacement. In patients with maintained joint space, the deficient can be reconstructed with iliotibial autograft or other graft sources. Midterm results have shown excellent outcome following labral reconstruction. In conclusion, labral deficiency is seen more commonly due to previous debridements and other pathologies. Treating the deficiency with labral reconstruction provides an excellent choice for the patient.

Introduction

As advances in the arthroscopic treatment of hip joint pathology are made, lesions of the acetabular labrum are being increasingly recognized as a source of intra-articular pain and hip morbidity. For the past several years, standard treatment for labral tears has been debridement and/or

S.C. Faucett • M.J. Philippon (✉)
Department of Hip Arthroscopy, Center for Outcomes-based Orthopaedic Research, Steadman Philippon Research Institute, Vail, CO, USA
e-mail: scott.c.faucett@gmail.com; drphilippon@sprivail.org

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_102

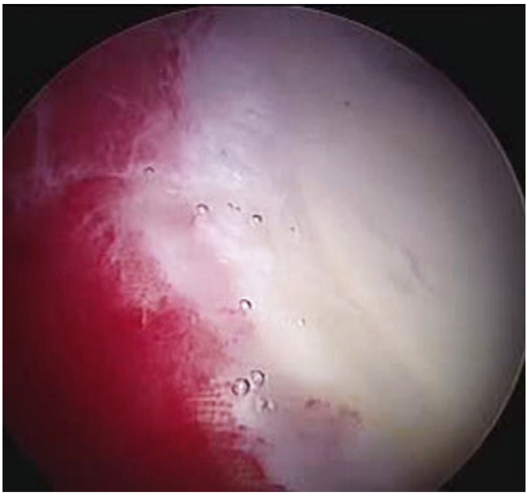


Fig. 1 Arthroscopic view of a deficient labrum in a hip that has undergone previous labral debridements (Courtesy Steadman Philippon Research Institute)

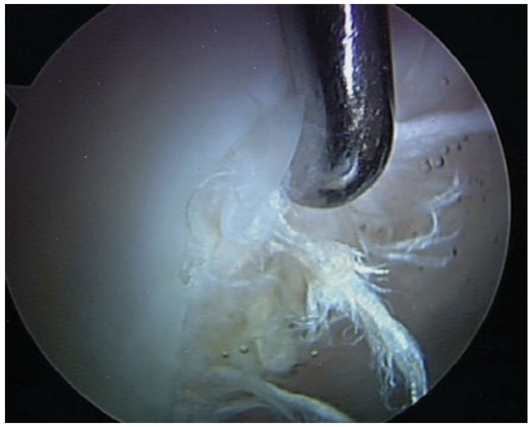


Fig. 2 Arthroscopic view of a labrum with a complex tear with complete disruption of longitudinal fibers (Courtesy Steadman Philippon Research Institute)

repair [2–6]. These techniques have been shown to produce symptomatic relief and return in function in most patients [2–6]. In patients who have multiple injuries or require repeated treatment of labral pathology for continued symptoms, additional labral tissue is removed. At some point, the labrum becomes deficient and is no longer functional (Fig. 1). Labral deficiency has also been identified in patients who have not had previous surgery. This chapter will discuss labral deficiency and the current treatment alternatives and outcomes for this condition.

Labral Function

The acetabular labrum attaches to the outer rim of the acetabulum and plays an important role in the normal mechanical function of the hip joint [7, 8]. The intact labrum deepens the socket which limits femoral head translation. In addition, the labrum provides a fluid seal to maintain the hydrostatic fluid pressure within the joint [9, 10]. This fluid seal protects the cartilage with the diffusion of nutrients to chondrocytes and also reduces cartilage consolidation by helping to distribute forces throughout the joint [11]. Labral dysfunction can lead to overloading of the articular cartilage of the hip and may be a precursor of osteoarthritis [12].

Etiology

While labral tears are associated with damage caused by femoroacetabular impingement (FAI), labral deficiency has multiple etiologies. This can be seen in primary or revision arthroscopies. Labral tears can be complex in nature, with complete disruption of the longitudinal fibers (Fig. 2). These tears are often seen in traumatic hip dislocations The labrum no longer functions and there is not enough healthy tissue to repair. As with the meniscus of the knee, labral tissue needs to be preserved for the health of the joint. Removing the complex tear would result in a deficient labrum. In addition, hypotrophic labrums are also seen in hips with no prior surgeries. In this chapter, a deficient labrum is defined as one that is 3 mm or less in width (Fig. 3). In primary arthroscopies, a hypotrophic labrum can be an anatomic variation or from repeated damage due to impingement.

Labral deficiency has multiple etiologies in the revision arthroscopy case including debridement, arthrofibrosis, and complex tears [1, 2, 13, 14]. After the pathophysiology of femoral acetabular impingement was popularized by Ganz and others, the most popular treatment to manage the resultant labral injuries was labral debridement and resection [2]. However, multiple studies have shown higher reoperation rates and lower modified Harris Hip Scores with labral debridement versus labral repair [3, 5, 6].

Arthrofibrosis can frequently form after injury or as a sequel of hip surgery [12, 13]. Adhesions are commonly seen in the hip at the site of the femoral neck osteoplasty and between the labrum and capsule (Fig. 4a, b). Occasionally the hip capsule can adhere to the labrum effectively elevating the labrum and disrupting the contact between the labrum and femoral head. This results in an area of deficiency in the biomechanics of the labrum. Despite careful separation of these adhesions, the remaining tissue is either of insufficient volume or has poor quality thereby creating a labral deficiency.

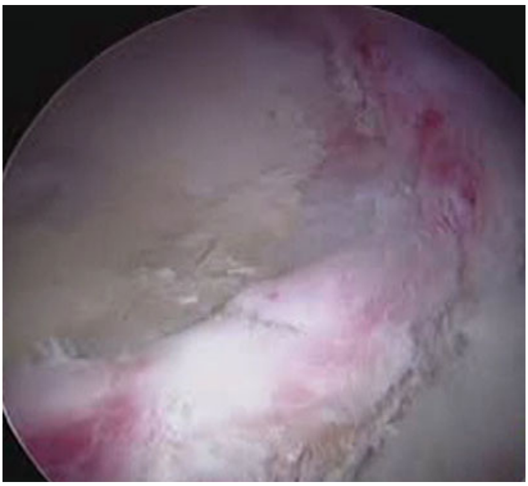


Fig. 3 Deficient labrum as seen at arthroscopy (Courtesy Steadman Philippon Research Institute)

Hip Stability/Labral Deficiency

Sporting activities which require repetitive twisting and pivoting of the hip put athletes at risk for labral tears and hip instability. The labrum is a secondary stabilizer of the hip and increases the articular contact area and volume of the acetabulum by 22 % and 33 %, respectively [8]. Ferguson et al. showed that after labral debridement the force to dislocate the hip is reduced [9]. Crawford et al. demonstrated that loss of the labrum leads to decreased femoral stability relative to the acetabulum during extremes of range of motion. In particular, 60 % less force is required to distract the femur 3 mm after the labrum has been removed [17]. Benali et al. reported gross instability resulting in hip subluxation after debridement of the acetabular labrum [13]. Meyers, et al. showed that while sectioning of iliofemoral ligament alone resulted in increased translation and external rotation, if the labrum was also sectioned, these values significantly increased [18]. If the iliofemoral ligament and the labrum were

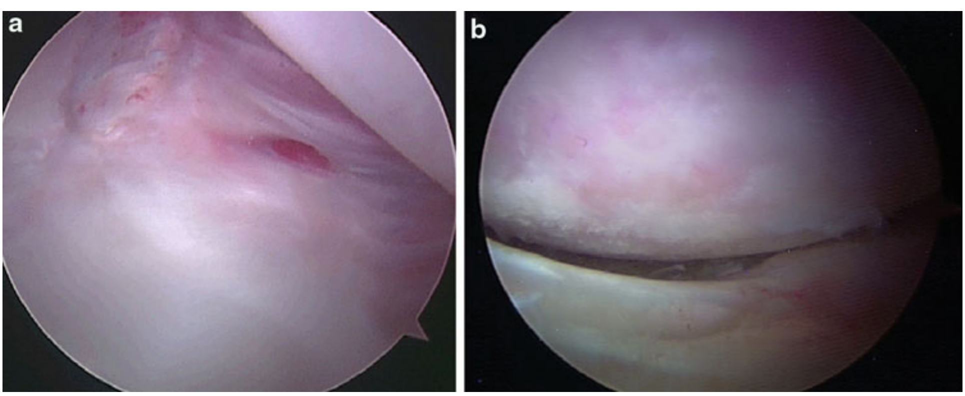


Fig. 4 (**a**) Adhesions between the capsule and the labrum seen during revision arthroscopy (Courtesy Steadman Philippon Research Institute). (**b**) Adhesions causing the labrum not to seal with the femoral head (Courtesy Steadman Philippon Research Institute)

repaired, the translation and external rotation significantly decreased [18].

When the labrum is deficient, the amount of strain on the remaining labrum also puts the hip at risk for instability. Smith and Sekiya also have demonstrated that labral strain increases as the circumferential tear is enlarged particularly after 2 cm of deficiency is obtained [19]. Frequently these patients will have pain and sensations of the hip slipping during extremes of motion.

Ferguson and Ganz also demonstrated that after labral, resection fluid pressurization in the central compartment was markedly lowered and cartilage consolidation was greater, in the labral deficient group than the intact group [18]. A recent study has shown that a fluid efflux was increased in hips with a labral tear compared to those with an intact labrum and that the efflux specifically occurred at the tear location [20]. Labral debridement was also associated with a higher fluid efflux compared to normal hips [20].

Dysplasia

The deficient labrum is even a greater problem in a patient with shallower hips. Henak et al. showed that the labrum has an even more critical role in dysplastic hips as it has a significant role in load transfer and hip stability than in hips with normal acetabular geometry [21]. The study showed in a normal model the labrum supports 1–2 % of the load, while in a dysplastic model, the labrum supports 4–11 % of the load. In the dysplastic model, the femoral head was in equilibrium at the lateral edge of the acetabulum instead of centered in the acetabulum. In addition, the study showed that cartilage contact area was lower in the dysplastic model during walking and going up and down stairs. The study concluded that the labrum may play a larger role in joint stability in the dysplastic hip. This reinforces the need for treatment of labral deficiency in the dysplastic hip [21]. In symptomatic dysplasia, labral tears have been seen in up to 90 % of cases [22]. Therefore, most authors recommend treatment of labral tears with repair primarily or in the setting of a deficient labrum to consider labral reconstruction in the dysplastic hip. Furthermore labral reconstruction and repair also plays a critical role in patients requiring bony correction of their dysplasia. The timing of when to perform labral repair and reconstruction in the context of a patient needing a bony correction surgery is still debated.

Cartilage Effects of Labral Deficiency

The acetabular labrum provides the suction seal to the central compartment. The function of this seal is to aid in the fluid dynamics of the hip joint [10, 11]. Through the seal, distraction and shear forces are reduced as described by Ferguson et al. [10, 11]. In addition cartilage consolidation is much greater in a labral deficient hip. Many authors have demonstrated that tears and partial resection of the labrum can dramatically reduce the functional seal. Furthermore others have shown a resultant increase in cartilage load, concentration, and potentially shear forces, predisposing the articulation to early degenerative changes after a labral tear or labral resection [11, 20]. Song has also demonstrated that the resistance to rotation is increased once the acetabular labral seal is lost such as in the setting of focal or complete labral resection [23]. Greaves et al. measured articular cartilage strain in cadaveric hips under a compressive load using 7 T MRI. The author found no significant effect of a labral tear compared to the intact state but did find a 4–6 % decrease in cartilage strain associated with labral repair compared to labral resection [24, 25]. Thus the labrum plays a vital role in the preservation of the labral seal of the central compartment and has potential cartilage protective effects by reducing cartilage strain and shear forces.

Treatment Alternatives

Non-operative Management

If a patient has minimal pain with his or her current activity level, non-operative treatment is an alternative. Furthermore if the patient demonstrates findings consistent with osteoarthritis and/or a joint space less than 2 mm, then management should consist of conservative management with injections until they are a candidate for arthroplasty [5, 6, 26].

Reconstruction

Several authors have described methods to reconstruct the labrum [1, 27–29]. Tissue such as the iliotibial band (allograft and allograft), gracilis, and ligamentum teres have all been described to reconstruct the labrum [1, 27–29]. The goals of reconstruction are to maintain the acetabular seal to improve the fluid mechanics in the central compartment and reduce shear forces on the acetabular cartilage [1, 20, 27–29]. The senior author uses iliotibial band autograft tissue (Fig. 5) in most cases; however, in cases of very large labral deficiencies, an allograft is used.

Outcomes

Patients who have undergone labral resection have demonstrated poorer outcomes and higher reoperation rates than those whom have retained their labrum. Kalore and Jiranek also reported on a group of 106 patients with 28 % reoperation rate for patients whom underwent debridement compared to 11 patients whom underwent labral repair [22]. Larson and Philippon have also separately reported on improved outcomes of patients whom have undergone labral repair versus patients undergoing labral debridement [3, 4, 5]. A recent randomized control trial also showed better outcomes in the repair group [31].

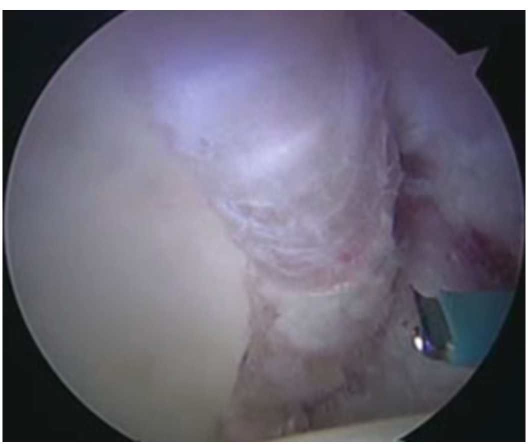


Fig. 5 Iliotibial band autograft labral reconstruction for the treatment of labral deficiency (Courtesy Steadman Philippon Research Institute)

While repairs have been shown to have superior outcomes, if the labrum is deficient, or not amenable to repair, a labral reconstruction is performed. Results have been encouraging following labral reconstruction with an iliotibial band autograft [27–29, 32, 33]. At an average of 18 months following reconstruction, patients showed an improvement in modified Harris Hip Score from 62 preoperatively to 85 at follow-up [28]. The median satisfaction was 8 on a 1–10 scale with 10 being very satisfied. This early follow-up showed a conversion to total hip arthroplasty in 8 % of hips with older patients more likely to undergo arthroplasty. The only independent predictor of patient satisfaction that was identified was age younger than 30 years [28].

Recently, 4-year follow-up was published on the same technique. Mean survivor (survivor = not requiring hip arthroplasty) time was 4.9 years [32]. In patients who did not have total hip arthroplasty, follow-up was obtained at an average of 49 months following arthroscopy. Patients' modified Harris Hip Scores improved from 60 to 83 at follow-up, and the median patient satisfaction was 8. A hip joint space of 2 mm or less was shown to be a contraindication for acetabular labral reconstruction [32]. Another study compared the results of labral reconstruction with a gracilis autograft with labral fixation [27]. The authors showed that even though the reconstruction group had a significantly lower preoperative score, more improvement was seen in the reconstruction group. The authors concluded that labral reconstruction is safe and effective and that reconstruction may not have inferior outcomes to repair [27].

As discussed earlier, labral deficiency is common in athletes. A recent publication reported on the results of labral reconstruction in the elite athlete [33]. The study followed 21 elite athletes whose sports included soccer, hockey, American football, skiing, baseball, basketball, and ice skating. Two patients required total hip arthroplasty; however, they were older than the average athletes. Eighty-six percent of the athletes return to professional sports and 81 % returned to their previous level of play. These athletes also showed improved outcome scores and high patient satisfaction with outcome. The study showed that iliotibial band autograft labral reconstruction provided high level athletes, including contact athletes with labral deficiency, the opportunity to return to professional sports [33].

Summary

With the increase use of hip arthroscopy to treat hip disorders, resultant labral deficiency is becoming more common. This condition is most frequently identified in patients who have had previous surgery but has also been seen in patients with impingement, instability, dysplasia, and adhesions. Labral deficiency results in increased compression and damage to the articular cartilage of the acetabulum. Labral reconstructions have been shown to reestablish the seal and reduce fluid efflux. The senior author's graft choice for labral reconstruction is iliotibial band autograft; however, multiple other techniques have been reported. The clinical outcomes 4 years after surgery have shown patients return to excellent function and even high level competitive sports following labral reconstruction.

References

1. Ejnisman L, Philippon MJ, Lertwanich P. Acetabular labral tears: diagnosis, repair, and a method for labral reconstruction. Clin Sports Med. 2011;30:317–29.
2. Santori N, Villar RN. Acetabular labral tears: result of arthroscopic partial limbectomy. Arthroscopy. 2000; 16:11–5.
3. Larson CM, Giveans MR. Arthroscopic debridement versus refixation of the acetabular labrum associated with femoroacetabular impingement. Arthroscopy. 2009;25:369–76.
4. Larson CM, Giveans MR, et al. Arthroscopic debridement versus refixation of the acetabular labrum associated with femoroacetabular impingement: mean 3.5-year follow-up. Am J Sports Med. 2012;40: 1015–21.
5. Philippon MJ, Briggs KK, et al. Outcomes following hip arthroscopy for femoroacetabular impingement with associated chondrolabral dysfunction: minimum two-year follow-up. J Bone Joint Surg (Br). 2009;91: 16–23.
6. Philippon MJ, Schroder BG, et al. Hip arthroscopy for femoroacetabular impingement in patients aged 50 years or older. Arthroscopy. 2012;28:59–65.
7. Safran MR. The acetabular labrum: anatomic and functional characteristics and rationale for surgical intervention. J Am Acad Orthop Surg. 2010;18: 338–45.
8. Tan V, Seldes RM, et al. Contribution of acetabular labrum to articulating surface area and femoral head coverage in adult hip joints: an anatomic study in cadaver. Am J Orthop. 2001;30(11):809–12.
9. Ferguson SJ, Bryant JT, et al. An in vitro investigation of the acetabular labral seal in hip joint mechanics. J Biomech. 2003;36:171–8.
10. Ferguson SJ, Bryant JT, et al. The acetabular labrum seal: a poroelastic finite element model. Clin Biomech. 2000;15:463–8.
11. Ferguson SJ, Bryant JT, et al. The influence of the acetabular labrum on hip joint cartilage consolidation: a poroelastic finite element model. J Biomech. 2000;33:953–60.
12. McCarthy JC, Noble PC, Schuck MR, Wright J, Lee J. The role of labral lesions to development of early degenerative hip disease. Clin Orthop Relat Res. 2001;393:25–37.
13. Benali Y, Katthagen BD. Hip subluxation as a complication of arthroscopic debridement. Arthroscopy. 2009;25:405–7.
14. Freehill MT, Safran MR. The labrum of the hip: diagnosis and rationale for surgical correction. Clin Sports Med. 2011;30:293–315.
15. Philippon MJ, Schenker ML, et al. Revision hip arthroscopy. Am J Sports Med. 2007;35:1918–21.
16. Ward JP, Rogers P, et al. Failed hip arthroscopy: causes and treatment options. Orthopedics. 2012;35:612–7.
17. Crawford MJ, Dy CJ, et al. The 2007 Frank Stinchfield Award. The biomechanics of the hip labrum and the stability of the hip. Clin Orthop Relat Res. 2077;465: 16–22.
18. Myers CA, Register BC, Lertwanich P, Ejnisman L, Pennington WW, Giphart JE, LaPrade RF, Philippon MJ. Role of the acetabular labrum and the iliofemoral ligament in hip stability: an in vitro biplane fluoroscopy study. Am J Sports Med. 2011;39(Suppl): 85S–91.
19. Smith MV, Panchal HB, et al. Effect of acetabular labrum tears on hip stability and labral strain in a joint compression model. Am J Sports Med. 2011;39 Suppl:103S–10S.
20. Cadet ER, Chan AK, et al. Investigation of the preservation of the fluid seal effect in the repaired, partially resected, and reconstructed acetabular labrum in a cadaveric hip model. Am J Sports Med. 2012;40: 2218–23.

21. Henak CR, Ellis BJ, et al. Role of the acetabular labrum in load support across the hip joint. J Biomech. 2011;44:2201–6.
22. Kalore NV, Jiranek WA. Save the torn labrum in hips with borderline acetabular coverage. Clin Orthop Relat Res. 2012;470:3406–13.
23. Song Y, Ito H, et al. Articular cartilage friction increases in hip joints after the removal of acetabular labrum. J Biomech. 2012;45:524–30.
24. Greaves LL, Gilbart MK, et al. Deformation and recovery of cartilage in the intact hip under physiological loads using 7 T MRI. J Biomech. 2009;42:349–54.
25. Greaves LL, Gilbart MK, et al. Effect of acetabular labral tears, repair and resection on hip cartilage strain: a 7 T MR study. J Biomech. 2010;43:858–63.
26. Philippon MJ, Briggs KK, et al. Joint space predicts THA after hip arthroscopy in patients 50 years and older. Clin Orthop Relat Res. 2013;471:2492–6.
27. Matsuda DK, Burchette RJ. Arthroscopic hip labral reconstruction with a gracilis autograft versus labral refixation: 2-year minimum outcomes. Am J Sports Med. 2013;41:980–7.
28. Philippon MJ, Briggs KK, et al. Arthroscopic labral reconstruction in the hip using iliotibial band autograft: technique and early outcomes. Arthroscopy. 2010;26:750–6.
29. Sierra RJ, Trousdale RT. Labral reconstruction using the ligamentum teres capitis: report of a new technique. Clin Orthop Relat Res. 2009;467:753–9.
30. Walker JA, Pagnotto M, et al. Preliminary pain and function after labral reconstruction during femoroacetabular impingement surgery. Clin Orthop Relat Res. 2012;470:3414–20.
31. Krych AJ, Thompson M, Knutson Z, Scoon J, Coleman SH. Arthroscopic labral repair versus selective labral debridement in female patients with femoroacetabular impingement: a prospective randomized study. Arthroscopy. 2013;29:46–53.
32. Geyer MR, Philippon MJ, Fagrelius TS, Briggs KK. Acetabular labral reconstruction with an iliotibial band autograft: outcome and survivorship analysis at minimum 3-year follow-up. Am J Sports Med. 2013;41:1750–6.
33. Boykin RE, Patterson D, Briggs KK, Dee A, Philippon MJ. Results of arthroscopic labral reconstruction of the hip in elite athletes. Am J Sports Med. 2013;41: 2296–301.

Surgical Technique: Arthroscopic Rectus Autograft

94

Thomas G. Sampson

Contents

Abstract

The importance of the acetabular labrum has been well documented for the health and function of the hip joint. Absence of the labrum through degeneration or injury and excision in an open or arthroscopic procedure has shown to cause continued pain as well as progressive destruction of the joint. The iliotibial band, harvested as a free graft, has most commonly been used to substitute for damaged or diseased labri; however, it requires a separate incision and may leave a defect in the fascia lata which may be bothersome to some patients. Fascia lata free grafts also require back table preparation and placement of sutures to create a tube graft. An alternative technique using the indirect head of the rectus femoris has been used on over 30 patients for intercalary labral grafts and provides an alternate technique in which the graph is harvested and repaired to the acetabular rim through the same arthroscopic portals. It is present in 100% of index hip surgeries, and sacrifice does not cause any significant dysfunction to the hip. An additional advantage is that it remains vascularized if left attached distally.

T.G. Sampson
Medical Director of Hip Arthroscopy, Post Street Surgery Center, San Francisco, CA, USA

Post Street Orthopaedics and Sports Medicine, San Francisco, CA, USA
e-mail: tgsampsonmd@gmail.com

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_103

Introduction

Since the inception of modern hip arthroscopy in the early 1980s, damage and disease of the labrum has been well recognized and until this decade has been treated with partial or total excision. With preservation techniques in recent years, the function of the labrum has been well recognized and therefore preserved whenever possible.

The acetabular labrum functions as a shock absorber and allowing proper joint lubrication and pressure distribution to protect and hydrate articular cartilage. In the absence of the labrum, hydrostatic pressurization and joint lubrication are impaired [1].

Philippon et al. showed that patients who have labral deficiency or advanced labral degeneration had good outcomes and high patient satisfaction after arthroscopic acetabular labral reconstruction using free iliotibial band graft [2] Nepple et al. using eight cadavers found labral reconstruction with an iliotibial band graft significantly improved pressurization similar to levels of the intact state and stability to distractive force, when compared to a partial labral resection [3].

Matsuda described an alternate technique using the gracilis tendon with good outcomes; however, a separate incision at the knee was necessary [4].

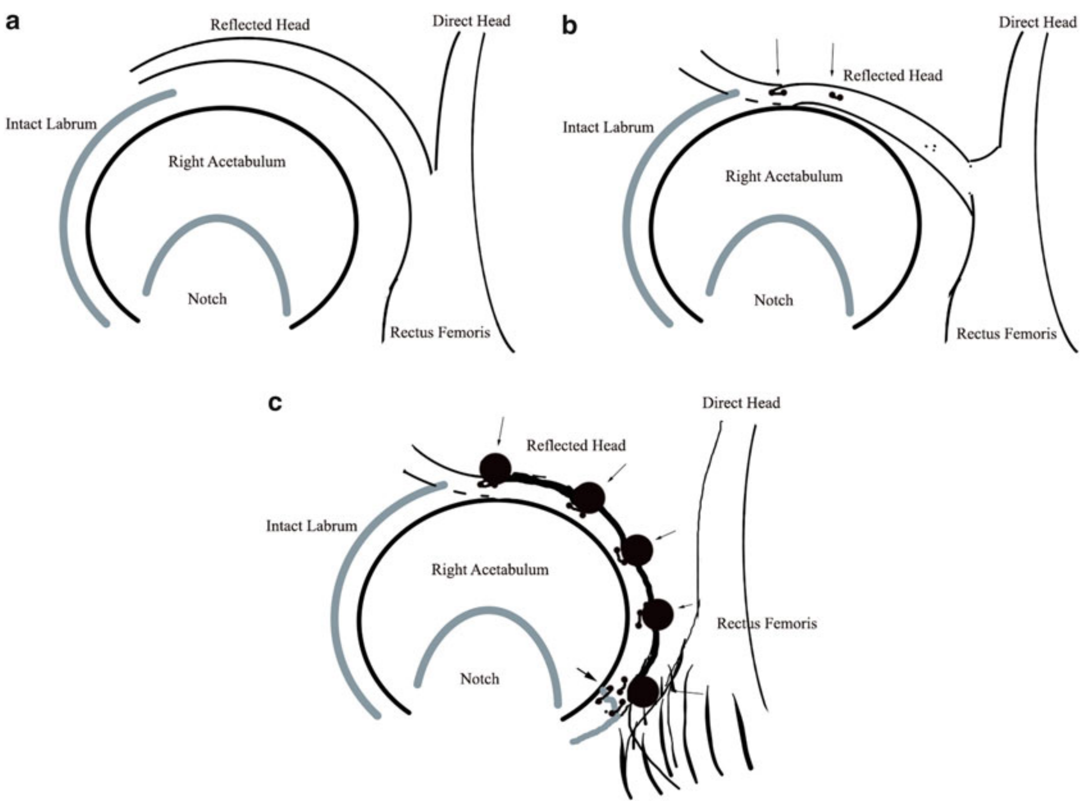


Fig. 1 (**a**–**c**) (**a**) Line drawing showing the reflected (indirect) head of the rectus femoris spanning over the defect of the acetabulum. Note its "Y" attachment to the direct head of the rectus femoris, (**b**) the indirect head has been partially detached from its insertion on the ileum leaving the distal attachment intact and an interim attachment to the muscular portion of the direct head, (**c**) the indirect head attached to the rim of the acetabulum with suture anchors; note the distal attachment remains intact and the direct head attachment has been released. Note the side-to-side anastomosis (*arrow*)

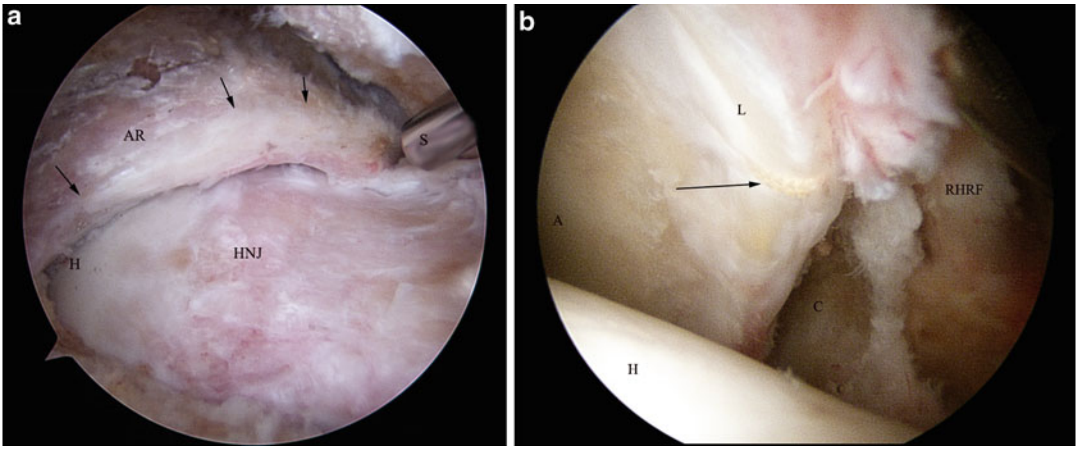


Fig. 2 (**a**–**b**) (a) View of a left hip assessing the labral quality; note the degeneration and poor tissue (*arrows*), acetabular rim (*AR*), femoral head (*H*), and head neck junction (*HNJ*); (**b**) remaining loop suture (*arrow*) around a stiff nonfunctional labrum (*L*), acetabulum (*A*)

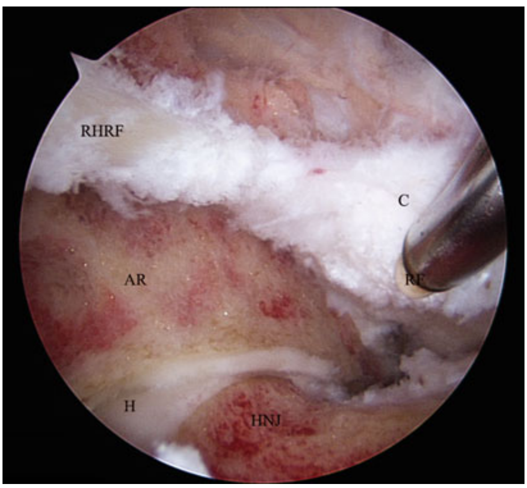


Fig. 3 View of a left hip through the anterolateral portal showing the area to be grafted after rim trimming of acetabulum; acetabular rim (*AR*), reflected head rectus femoris (*RHRF*), capsule (*C*)

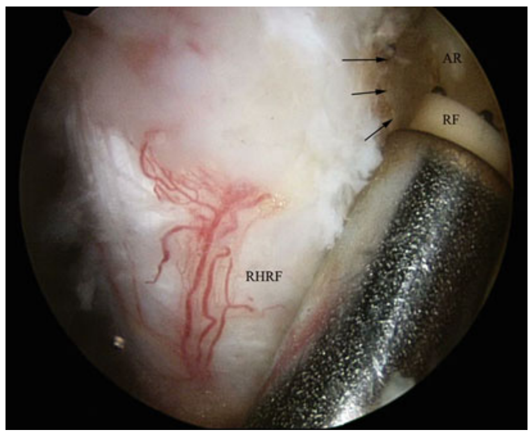


Fig. 4 View of a right reflected head of the rectus femoris prior to detachment from the acetabular rim (*arrows*) by an *RF* wand (*RF*)

Indications

The indications for labral graft reconstruction include hips in which the labrum has been destroyed by injury or surgery. Additionally global pincer over-coverage or profunda, ossified labri, and large rim fractures or os acetabuli may have insufficient labral function. Most revision hip preservation surgery should be preoperatively prepared for a labral reconstruction.

Technique

The concept is to use the indirect head of the rectus femoris between the muscular attachment with the direct head and the lateral insertion leaving at least the distal insertion intact for a blood

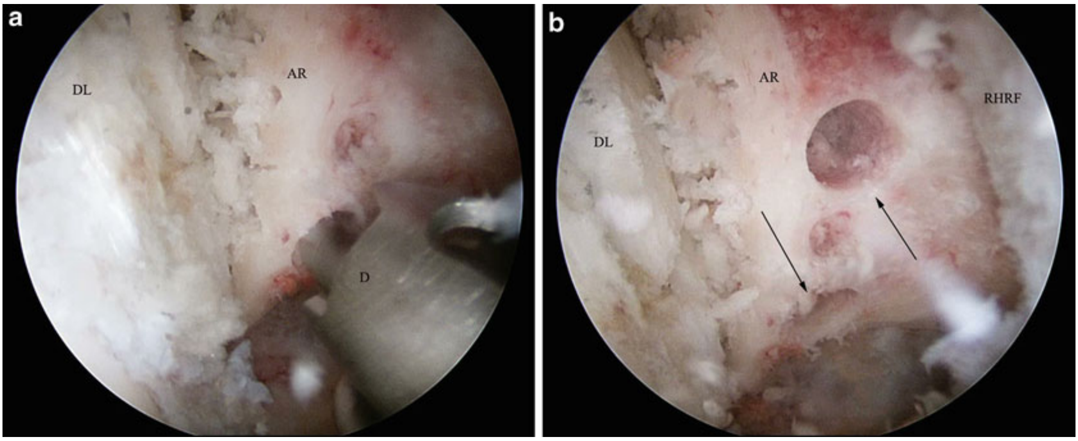


Fig. 5 (**a**–**b**) (**a**) The acetabular rim has been predrilled to accept anchors prior to grafting. Degenerated labrum (*DL*), drill (*D*), (**b**) drill holes (*arrows*)

supply. In some cases the muscular attachment may be partially be preserved (Fig. 1a–c). After standard arthroscopic techniques with either supine or a lateral approach and the acetabular rim has been trimmed to the appropriate level, the remaining labrum is assessed for presence, quality, and functionality (Fig. 2). Most cases involve placing an intercalary graft in acetabular zones 1–3 and with some revisions as far as zone 5 [5]. Most indirect heads of the rectus are long enough to span from zone 4 to 1; however, they are probably too short for the entire circumference from zones 1 to 5, and one of the other choices should be used.

Through an extensive capsulotomy, expose the rim of the acetabulum and the area to be grafted (Fig. 3). Identify the indirect head of the rectus femoris and release it from its superior attachment to the ilium, leaving the lateral insertion and the muscular attachments intact (Fig. 4). The idea is to create a “bucket handle” which will be sequentially attached to the acetabular rim from lateral to anterior while leaving the peripheral attachments to tension it while laying onto the rim.

The anchor holes are predrilled usually one per zone (Fig. 5). Using any suture technique, horizontal mattress sutures are places with high-strength no-absorbable suture. Knotted or knotless anchors may be employed. As the graft is laid down and the suture is tightened forcing the graft into position, the muscular attachment may need to be partially released in sequence while still tensioning the graft (Fig. 6). Once the graft has covered the desired area, the muscular attachment is completely released to do a side-to-side anastomosis with the remaining labrum if desired.

Results

From June 2009 to October 2013, 31 hips have been treated with a rectus graft. There were 16 males and 15 females. All but one were revisions, and of the revisions, 3 were from prior open surgical dislocations and 27 were from previous hip arthroscopy. The average preop mHHS was 65.3 and the average post-op score was 83.5 with an improvement of 18.2. It was common for the maximum benefit to be perceived after one year, but initial satisfaction was felt as early as 6 weeks. All patients were allowed to weight bare as tolerated and get off crutches when they felt stable and the

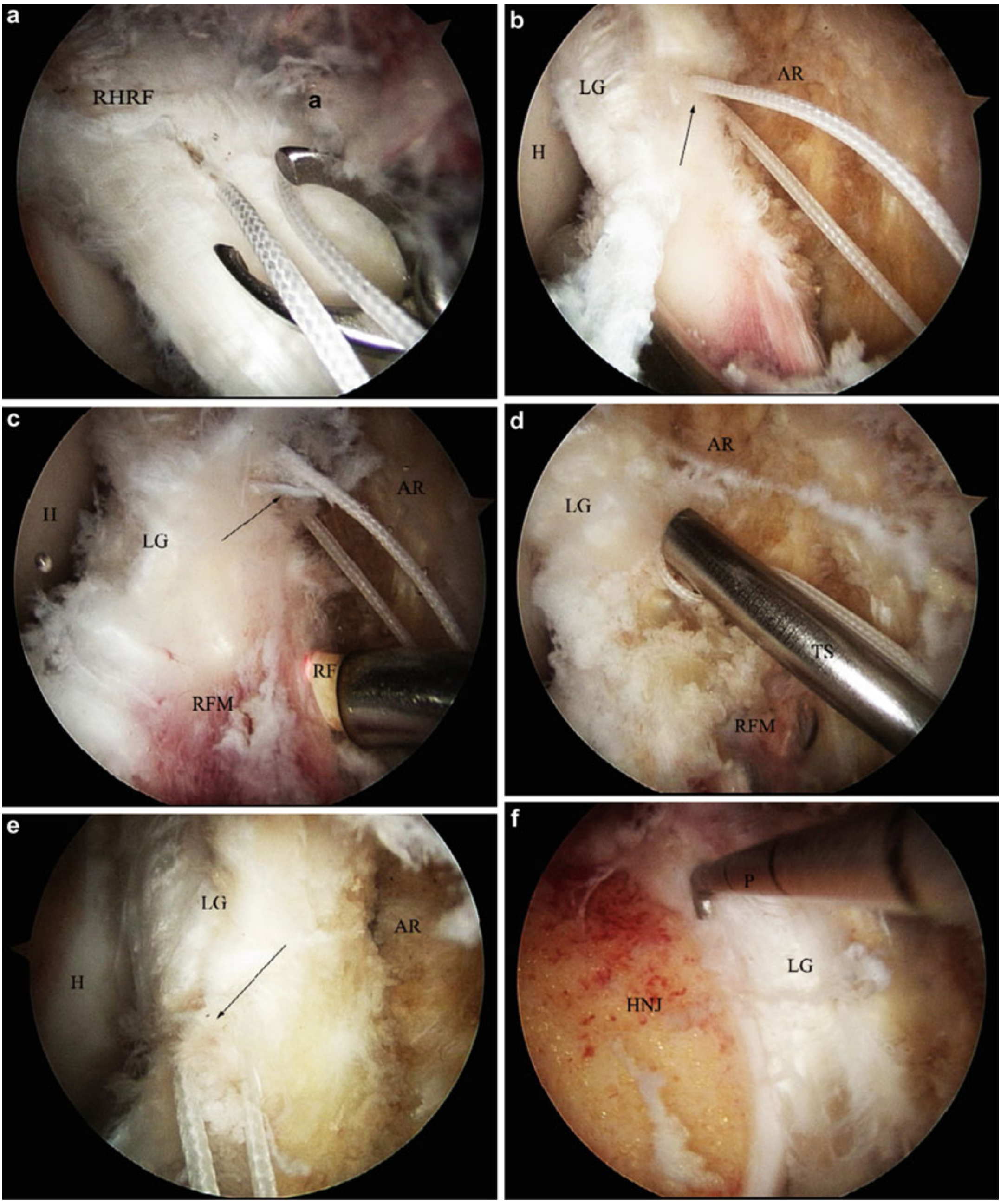


Fig. 6 (continued)

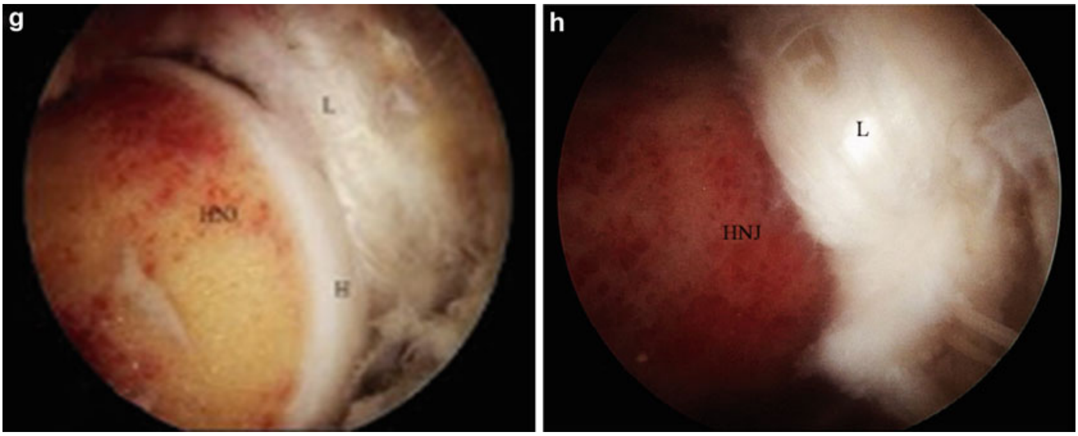


Fig. 6 (**a**–**h**) The sequence of grafting a right hip; (**a**) the partially detached reflected head rectus femoris (*RHRF*) has been partially fixed using a horizontal mattress suture to the acetabular rim (*AR*); note the muscular attachment remains intact to tension the labral graft (*LG*) and the tied suture (*arrow*), (**c**) the RF probe (*RF*) is detaching the muscular portion of the rectus femoris (*RFM*) while tightening the suture (*arrow*), (**d**) pushing the knot with the tying stick (*TS*) and labral graft (*LG*) to the acetabular rim (*AR*), (**e**) final knot (*arrow*), (**f**) checking for the labral seal with the prone (*p*), (**g**) and distracting to check the suction seal, (**h**) followed by a motion study to check that there is no remaining impingement

pain was diminished. The earliest of external walking aids was 3 days and the longest was 9 months.

Summary

An alternate to iliotibial band grafts for labral deficiency is the reflected head of the rectus femoris. It is available in all index hips and offers a graft that may be obtained through the same arthroscopic portals and may be partially vascularized if the distal attachment is left intact. No "back table work" is necessary to prepare the graft, and the results are comparable to the IT band graft.

References

1. Bedi A, Allen AA. Superior labral lesions anterior to posterior-evaluation and arthroscopic management. Clin Sports Med. 2008;27(4):607–30.
2. Philippon MJ, et al. Arthroscopic labral reconstruction in the hip using iliotibial band autograft: technique and early outcomes. Arthroscopy. 2010;26(6):750–6.
3. Nepple J, et al. The effect of an acetabular labral tear, repair, resection, and reconstruction on the Hip fluid seal. Arthrosc J Arthrosc Relat Surg Off Publ Arthrosc Assoc North Am Int Arthrosc Assoc. 2013;29(12):e208.
4. Matsuda DK. Arthroscopic labral reconstruction with gracilis autograft. Arthrosc Tech. 2012;1(1):e15–21.
5. Ilizaliturri Jr VM, et al. A geographic zone method to describe intra-articular pathology in hip arthroscopy: cadaveric study and preliminary report. Arthroscopy. 2008;24(5):534–9.

95 Surgical Technique: Arthroscopic Labral Reconstruction Using Iliotibial Band Autograft or Allograft

Marc J. Philippon and Dominic Carreira

Contents

Electronic supplementary material: The online version of this chapter (doi:10.1007/978-1-4614-6965-0_104) contains supplementary material, which is available to authorized users.

M.J. Philippon (✉)
Department of Hip Arthroscopy, Center for Outcomes-based Orthopaedic Research, Steadman Philippon Research Institute, Vail, CO, USA
e-mail: drphilippon@sprivail.org

D. Carreira
Broward Health Imperial Point, Orthopedic Surgery – Foot and Ankle, Broward Health Medical Center, Fort Lauderdale, FL, USA
e-mail: dcarreira@gmail.com

Abstract

Labral tears cannot always be repaired. When the labrum tear is complex or previous debridement has left limited function tissue, labral reconstruction is indicated. Labral reconstruction of the hip has been demonstrated to be a safe and effective technique, with high patient satisfaction rates, improved pain, and improved patient outcome scores. Tissue for the labral reconstruction can be either autografts or allografts. This chapter described labral reconstructions using iliotibial band autograft and allograft.

Introduction

The treatment of labral tears of the hip has evolved rapidly as a better understanding of the outcomes of various procedures has been gained. During the earlier stages of the evolution of hip arthroscopy, labral debridements demonstrated good to excellent outcomes in 62 % of cases at mean follow-up of 8.4 years [1]. More recently, several studies have demonstrated improved outcomes in patients with repairs when compared to debridements [2–5]. However, similar to the meniscus of the knee, when the labrum tear is complex and cannot be repaired or previous debridement has left limited function tissue, debridement or repair is not an option. Labral reconstruction of the hip has been demonstrated to be a safe and effective technique, with high patient satisfaction rates,

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_104

improved pain, and improved patient outcome scores [6–9]. In this chapter, two all-arthroscopic techniques for reconstruction of the labrum are presented, utilizing autograft or allograft fascia lata.

Indications

The type and extent of tearing contribute to the ability to repair the tissue, with more complex and radial tears considered a relative indication. A greater extent of degeneration, typically demonstrated by yellowish discoloration and friability of the tissue, also is a relative indication for reconstruction. Other factors include the extent of bruising, the extent of instability, and the size of the tissue before (hypoplasia) and after debridement to stable tissue. Commonly, reconstruction is performed in the setting of previous hip arthroscopy and labral debridement.

Graft Choice

The advantages and disadvantages of allograft versus autograft tissue are similar to other reconstructive procedures in sports medicine. In the case of the hip, the labrum does not serve as a ligament resisting stress, but increases the surface area of articulation on the side of the acetabulum and creates a suction seal for improved joint mechanics. Advantages of autograft tissue compared to allograft include cost and the avoidance of the risk of disease transmission. Other potential advantages include speed of graft incorporation and the avoidance of sterile reactions to allograft tissue. Potential disadvantages of autograft tissue include donor site morbidity (pain and infection) and time in surgery.

Arthroscopic Technique

Regional anesthesia in the form of lumbar plexus block or epidural block may be administered for perioperative analgesia. Patients are placed on a fracture table in a supine position, and traction is placed with the operative hip flexed 10° and neutral abduction/adduction and with the contralateral leg placed in 40° abduction. Paralysis is maintained while the patient is in traction. Standard arthroscopic portals are established atraumatically: the anterolateral portal first and the midanterior portal under direct visualization with needle guidance.

A diagnostic arthroscopy is performed and a capsulotomy is routinely performed. Associated pathologies are identified and treated, which include rim trimming and/or femoral osteoplasty for FAI, debridement of ligamentum teres tears, removal of synovitis, removal of loose bodies, and articular cartilage debridement or microfracture. The size of cartilage lesion at the chondrolabral junction can be reduced when performing the rim trimming (Fig. 1). Using a probe, the quality and stability of the labral tissue is examined as well as the extent of damage. Reconstruction of the labral tissue may extend up to the 3 (right) or 9 (left) o'clock position anteromedially and to the 8 (right) or 4 (left) o'clock position posterolaterally. Once native, healthy tissue has been established at the anteromedial and posterolateral extents, the damaged segment in between may be removed using a beaver blade and shavers. A border of native labral tissue is necessary to help create a suction seal once the reconstructed labrum is fixated (Fig. 2). It is not always necessary to suture between the native and reconstructed labrum in order to create a suction seal; however, this may be determined during a dynamic exam after the graft is in place. After removal of the labral tissue, rim trimming (Fig. 3) and treatment of the articular cartilage during labral reconstruction are facilitated with improved visualization.

Autograft Technique

Upon completion of the diagnostic arthroscopy and confirmation that the labrum is not repairable, the remnant labrum is resected, the acetabular rim is prepared with a mechanical burr, and the defect site is measured. The graft is harvested with the leg in extension through a longitudinal incision

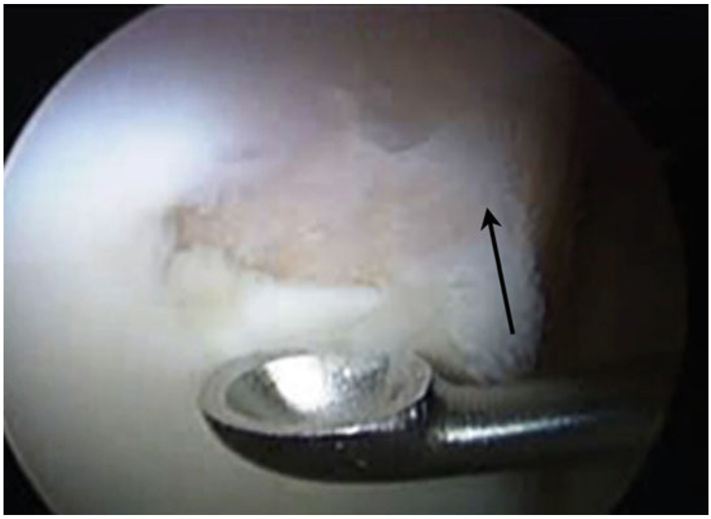


Fig. 1 An articular cartilage lesion at the chondral labral junction (*arrow*). The overall size of the lesion was decreased by the rim trimming

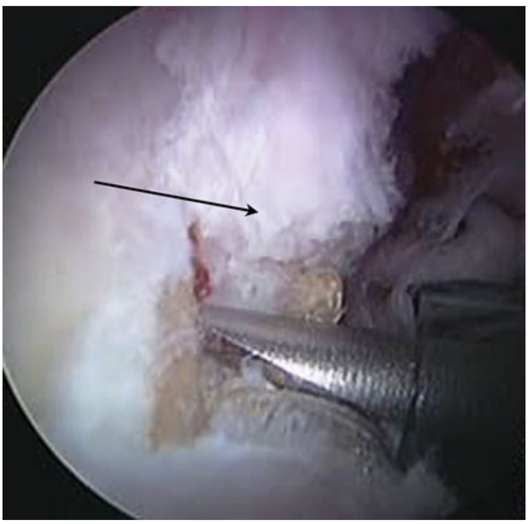


Fig. 2 After debridement of deficient labrum, a border of healthy native labrum (*arrow*) is the starting point for anchoring the graft

centered over the greater trochanter. A graft, measuring approximately 60 mm by 15 mm, is harvested from the central and posterior third of the iliotibial band. The length of the graft is determined by the size of the defect. Unless tension is seen in the iliotibial band, the defect is typically closed. A suture anchor is placed at the anteriormost aspect of the defect. The graft is tubularized and advanced into the joint through the midanterior portal (Fig. 4). A suture anchor is then placed at the posterior aspect of the defect and the graft is secured. Suture anchors are then place along the graft to ensure stability and apposition of the graft (Fig. 5). Sutures can be looped around the graft or passed through the graft. Sutures looped through the graft tend to evert the labrum and sutures passed through the labrum tend to invert it. Using a combination of these sutures to manage the position of the graft results in better restoration of the suction seal. Traction is released and a dynamic exam is performed to ensure the suction seal has been restored. The dynamic exam should include moving the hip through full range of motion to ensure adequate seal (Fig. 6). If the graft appears unstable, additional suture anchors can be placed.

Allograft Technique (Carreira Shuttle Technique)

For this allograft technique, an accessory distal portal is created for the shuttling of allograft tissue and for anteromedial anchor placement. The fascia lata is tubularized on a back table using a baseball stitch with 2-0 Vicryl (Fig. 7).

Key steps:

1. A percutaneous anchor is placed through the accessory distal portal (ADP) at the anteromedial extent of the labrum reconstruction. This anchor is placed with a striped suture

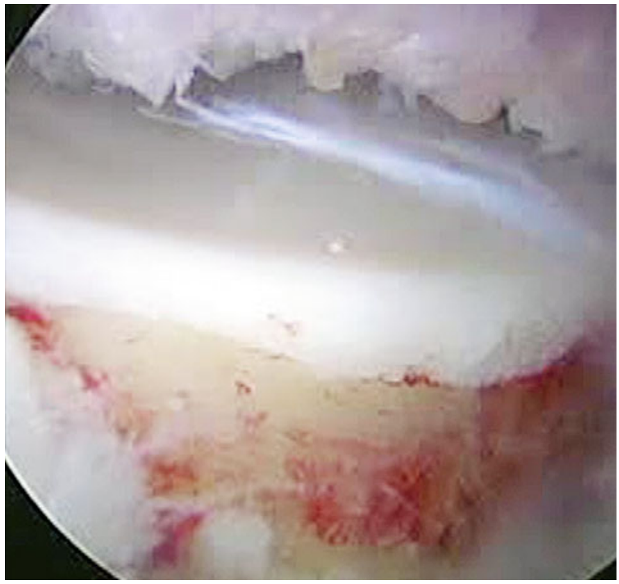


Fig. 3 Prepared rim for labral reconstruction. With no labrum the rim trimming can be done with increased precision

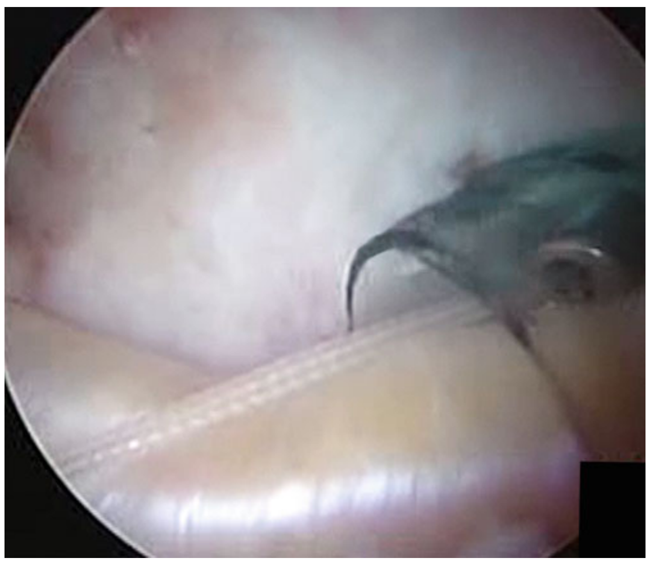


Fig. 4 The tubularized autograft coming into the joint through the midanterior portal

to allow for measuring of the length of the defect once the second anchor has been placed.

2. While visualizing from the midanterior portal (MAP), a second anchor is placed through a labral repair cannula at the anterolateral portal (ALP) at the posterolateral extent of the reconstruction. This suture is clamped with a hemostat to provide tension on the suture to prevent suture crossing.

One of the sutures from the anteromedial anchor is passed through the ALP. Using a knot pusher, the limb from the anteromedial anchor located in the ALP is used to measure the number of crossing lines between the two anchors. The overall length can then be calculated. For example, if the length between the stripes on the suture is 3 mm and the 10 stripes are counted between anchors, the chord length is 30 mm.

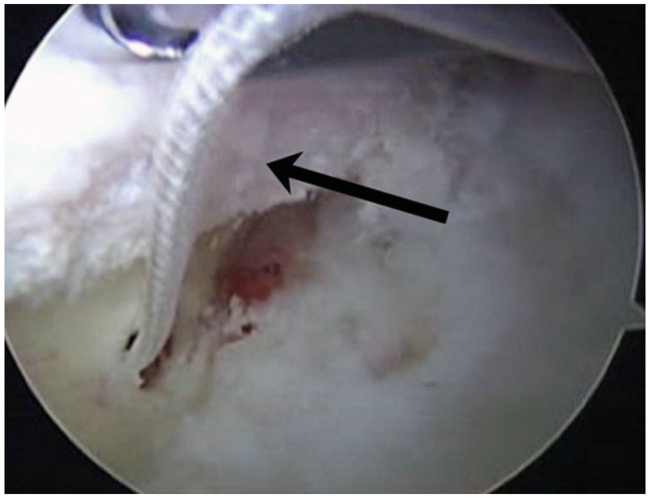


Fig. 5 Suture anchors placed along the acetabular rim to secure graft in place

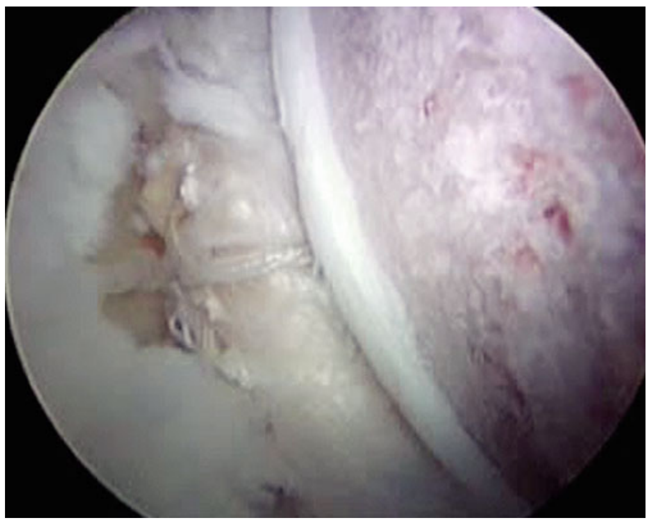


Fig. 6 Arthroscopic view of the dynamic exam. The labral autograft recreates the seal with the femoral head

The chord length is then multiplied by 1.3 to determine the arc length, i.e., the graft length.

3. The camera is then placed in the ADP portal; a second labral repair cannula is placed at the MAP. The limb of suture from the percutaneous anchor placement is then passed into the labral repair cannula in the MAP. A free needle is used to pass the suture material through the graft outside of the joint. One limb from each suture anchor passing through the ALP is tied securely to the graft, allowing enough length of suture material to allow for suture tying.
4. The limb from the MAP portal is pulled and the graft is introduced halfway into the joint. Prior to fully seating the anteromedial extent of the graft, sutures are visualized to make sure they are not crossed. The second limb from the anteromedial anchor is then passed into the MAP. The ends of the labrum reconstruction are tied using a standard knot-tying technique.
5. Similar to a standard labral repair, the segment in between is secured with suture anchors.

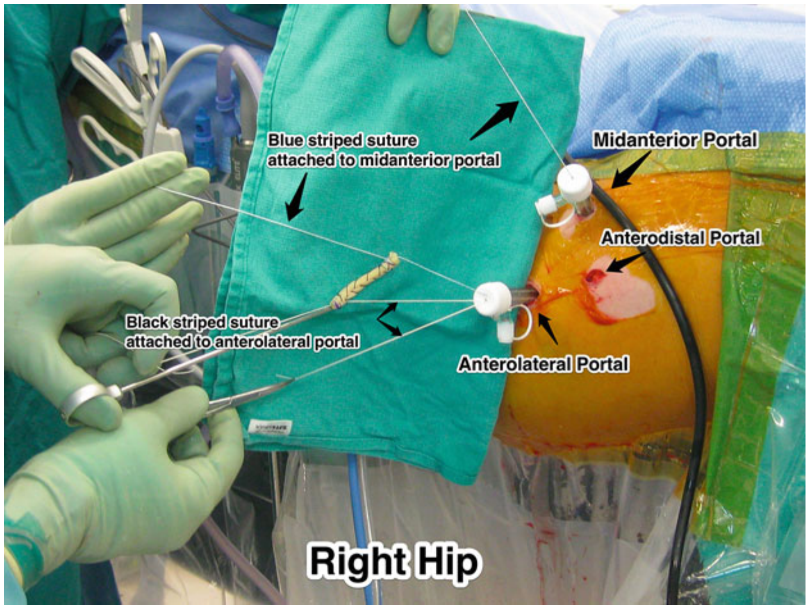


Fig. 7 Setup for the insertion of the labral allograft

Video 1 Labrum Allograft Reconstruction Technique (Carriera Shuttle Technique)

A video of the labrum allograft reconstruction technique (Carriera Shuttle Technique) is provided online (Video 1).

Summary

Labral reconstruction is indicated when the type and extent of tearing do not allow the labrum to be repaired, as with more complex and radial tears. The advantages and disadvantages of allograft versus autograft tissue are similar to other reconstructive procedures in sports medicine. The remnant labrum is resected, the acetabular rim is prepared with a burr, and the size of the graft needed is measured. The graft is harvested, prepared, and sutured into the defect. A dynamic exam is performed to show the graft is stable. Labral reconstruction is a technical procedure which has shown excellent midterm follow-up. Although there are no current published outcomes using an allograft, it is a viable option in many cases.

References

1. Meftah M, Rodriguez JA, Panagopoulos G, Alexiades MM. Long-term results of arthroscopic labral debridement: predictors of outcomes. Orthopedics. 2011;34 (10):e588–92.
2. Krych AJ, Thompson M, Knutson Z, Scoon J, Coleman SH. Arthroscopic labral repair versus selective labral debridement in female patients with femoroacetabular impingement: a prospective randomized study. Arthroscopy. 2013;29:46–53.
3. Larson CM, Giveans RM, Stone RM. Arthroscopic debridement versus refixation of the acetabular labrum associated with femoroacetabular impingement: mean 3.5-year follow-up. Am J Sports Med. 2012;40:1015–21.
4. Philippon MJ, Weiss DR, Kuppersmith DA, Briggs KK, Hay CJ. Arthroscopic labral repair and treatment of femoroacetabular impingement in professional hockey players. Am J Sports Med. 2010;38:99–104.
5. Philippon MJ, Briggs KK, Yen YM, Kuppersmith DA. Outcomes following hip arthroscopy for femoroacetabular impingement with associated chondrolabral dysfunction: minimum two-year follow-up. J Bone Joint Surg Br. 2009;91:16–23.
6. Geyer MR, Philippon MJ, Fagrelius TS, Briggs KK. Acetabular labral reconstruction with an iliotibial band autograft: outcome and survivorship analysis at minimum 3-year follow-up. Am J Sports Med. 2013;41:1750–6.
7. Matsuda DK, Burchette RJ. Arthroscopic hip labral reconstruction with a gracilis autograft versus labral refixation: 2-year minimum outcomes. Am J Sports Med. 2013;41:980–7.
8. Philippon MJ, Briggs KK, et al. Arthroscopic labral reconstruction in the hip using iliotibial band autograft: technique and early outcomes. Arthroscopy. 2010;26:750–6.
9. Boykin RE, Patterson D, Briggs KK, Dee A, Philippon MJ. Results of arthroscopic labral reconstruction of the hip in elite athletes. Am J Sports Med. 2013;41:2296–301.

Labral Reconstruction Using Hamstring Autograft/Allograft

96

Michael J. Salata and Richard C. Mather III

Contents

Abstract

Arthroscopic procedures of the femoral acetabular joint have become much more common in recent years as advances in surgical instrumentation and diagnostics have become available. Indications for various surgical procedures continue to evolve, and new problems have become recognized as potential etiologies that lead to pain and disability in certain patient populations. As the arthroscopic treatment of labral pathology has evolved, the need for advanced reconstructive techniques for certain labral conditions has also been developed. As with all surgical procedures, appropriate patient indications are paramount, and a reliable and reproducible surgical technique to address these challenging clinical issues is required. This chapter will address a method for reconstruction of the acetabular labrum.

Introduction

Labral pathology can exist in the setting of FAI. There are several studies that suggest the labrum may play an important role in normal hip joint kinematics. It has been shown that preservation of the labrum can lead to improved surgical outcomes when compared to labral debridement. In the setting of a deficient or irreparable labrum, reconstruction may allow the joint to be restored to a more normal

M.J. Salata (✉)
Department of Orthopaedic Surgery, Joint Preservation and Cartilage Restoration Center, University Hospitals Case Medical Center, Cleveland, OH, USA
e-mail: michael.salata@uhhospitals.org

R.C. Mather III
Department of Orthopaedic Surgery, Duke University School of Medicine, Durham, NC, USA
e-mail: richard.mather@duke.edu; chad.mather@gmail.com

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_105

state. Several techniques can be used to reconstruct the labrum; this chapter will discuss the use of hamstring tendon as a graft source for this procedure.

Patient History and Evaluation

The evaluation of a potential patient for a reconstructive labral procedure begins in the same manner as all patients presenting with hip pain. A thorough history of the patient's symptoms and mechanism of injury are very important. Any previous surgical procedures that have been performed on the hip are important to document. Any previous operative reports should be obtained and critically evaluated in the case of revision surgery. Note should be made of whether a labral repair, debridement, or resection was performed at the index or subsequent surgeries. The treating surgeon should try to determine the type (biocomposite or PEEK) and number of surgical implants previously used, the location and size of the tear that was treated, and the condition of the patient's labrum at the time of the previous operations. Also age and general medical history should be obtained.

Often patients will have previously undergone a labral repair or debridement in association with other surgical procedures such as a rim recession for pincer lesions, a femoral osteochondroplasty, or microfracture of a focal acetabular or femoral chondral defect. Patients may complain of persistent groin pain as the most common symptom and duration and aggravating activities should be documented.

A thorough exam should include a neurovascular examination to assess for any signs of chronic regional pain syndrome, an abdominal exam to assess for possible concomitant conditions such as athletic pubalgia, and a detailed and extensive examination of the affected hip. Hip range of motion which should be compared to the contralateral side should be documented as should specific diagnostics tests such as pain provocative maneuvers like the FADIR test.

Imaging

Standard hip radiographs should be the initial imaging modality. A well-done AP pelvis radiograph supplemented with a 45° Dunn lateral view and a false profile view of the affected hip are standard for an initial evaluation.

More advanced imaging in the form of a high-resolution MRI either with or without the addition of an arthrogram can be performed to evaluate for evidence of labral retear, labral deficiency, chondral pathology, capsular deficiency, and capsulolabral adhesions.

A three-dimensional CT scan is a very useful tool to evaluate the adequacy of previous rim recessions or femoral osteochondroplasty and can also be utilized to evaluate for over-resection of the femoral head which can be a source of persistent pain or mechanical symptoms.

Indications

Currently there are no definitive indications for labral reconstruction. Some surgeons will utilize this procedure for the augmentation of hypoplastic labra or for labral tissue that is too damaged to repair at the time of an index operation.

The current most definitive indication for labral reconstruction is in patients who have previously undergone a total or subtotal labrectomy who have persistent pain that is unresponsive to nonoperative treatment. Rarely does this condition exist in isolation with residual CAM deformity being the most common concomitant additional pathology identified.

Procedure

The patient is identified in the holding area and informed consent is reviewed. Risks and benefits of the procedures are discussed and documented. If allograft tissue is to be used, it is clearly stated on the consent form, and a discussion of the risks of allograft use including disease transmission is defined.

Once the patient is consented, regional anesthesia may be utilized as an adjunct as deemed necessary. A well-done fascia iliacus block or a lumbar plexus block has been utilized with good success and can be administered based on the comfort level and experience of the anesthesiologist. Once the patient is in the operating room, a commercially available table attachment used for hip arthroscopy through a fracture table setup can also be utilized depending on surgeon preference. Well-padded boots and a very well-padded and offset perineal post are placed, and the patient, once anesthetized, is placed into the distraction setup. Intravenous antibiotics are administered prior to skin incision, and a sequential compression device on the nonoperative limb for DVT prophylaxis is utilized. Neuromuscular blockade can be used to assist with the distraction of the hip joint, but this is felt by some to be less important.

A C-arm image intensifier is then centered over the patient's operative hip, and under spot fluoroscopic imaging, the hip is distracted gently and gradually to allow adequate access to the hip joint. A goal of 8–10 mm is required for safe and atraumatic access to the joint. If distraction is difficult to obtain, the hip can be prepped and a spinal trocar needle may be introduced from the position of the standard anterolateral (AL) portal to vent the hip and release the vacuum suction seal. Once the surgeon is confident that the needle is in the acetabulum, an air arthrogram can be safely performed that allows excellent delineation of the labrum and articular cartilage of the hip. The needle can be repositioned as necessary if it has pierced the labrum on this view. Once vented the hip can often be more easily distracted with less force necessary. If the hip is not distractible, an outside-in approach should be utilized to gain safe entry into the joint. Once adequate distraction is obtained, the hip should be placed into a slightly flexed position and internally rotated. The limb is then prepped and draped in the standard fashion. It is important to prep the limb distal to the pes anserine insertion if planning to harvest an ipsilateral hamstring graft.

Once prepped and draped, a standard AL portal is created via a nitinol wire-guided technique and a 4.5 mm cannula is introduced. A modified mid-anterior portal (MMA) is then created under direct visualization and exchanged for a 5.5 mm cannula. The 70° arthroscope is then moved to the MMA portal and look back on the initial AL portal to ensure it is appropriately positioned. An interportal capsulotomy is then created once a diagnostic scope has been performed that connects the two portals. This can be extended posteriorly or anteriorly as required to gain adequate access and maneuverability in the joint. In general, the capsule should not be opened any further anteriorly than necessary, most often stopping before the level of the psoas U.

Fig. 1 The labral defect is identified and defined. A stump of native labrum is *left* to either side of the defect if possible to allow for anastomosis of the graft to the native labrum

The features of the diagnostic arthroscopy include the condition of the chondral surfaces of the acetabulum and the femoral head, the condition of the labrum, the integrity of the ligamentum teres, and the psoas tendon if previous work was done in that area. Also the condition and integrity of the hip capsule is documented.

The labrum is probed and if deficient or severely damaged is debrided back to a stable stump to either side of the defect (Fig. 1). Most labral tears occur from the 12 o'clock superior position and extend to a variable degree anteriorly. Often once a sufficient debridement is performed, the defect will often extend from 11 o'clock to the 3 or 9 o'clock position depending on if the hip is a

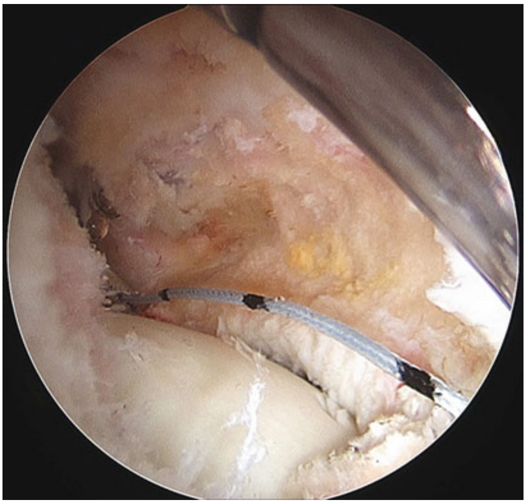


Fig. 2 The labral defect is measured with a high-tensile suture marked at 5 mm increments. Oversizing the graft by 5–7 mm is helpful in most cases

right or a left. It is helpful to leave the stump of the native labrum intact in these positions whenever possible to serve as an anastomosis site for the labral graft.

Once the defect is defined, a motorized burr is utilized to prepare the acetabular rim. Recession for pincer lesion is performed as deemed necessary from the preoperative planning, and it is also common to decorticate the rim in the location of the defect to have a bleeding surface for the labral graft to adhere to during the healing process.

A distal anterolateral accessory (DALA) portal can be utilized for many parts of this procedure. It is useful for the anchor drilling sequence as it provides a more ideal trajectory to access the rim and also is useful for suture and graft management. It is created in line with the standard AL portal 3–4 cm distally and identified with a spinal needle. If the labrum is deficient in a more posterior position, it is useful to often establish a standard posterolateral portal which can be used to effectively deliver anchors to this location.

The defect is then measured prior to graft preparation or harvest. It is useful to mark a high-tensile suture at 5 mm increments with a surgical marking pen (Fig. 2). This marked suture is then introduced into the joint, and use two graspers to place it into the labral defect along the acetabular rim. The distance is then recorded and the graft is oversized by 5–7.5 mm to ensure the graft is of adequate length. Many labral defects will measure between 4 and 5 cm in length.

Once the defect size is known, the graft harvest is performed if using an autograft. If allograft is selected, a semitendinosus allograft (STA) or tibialis anterior tendon allograft (TAA) can be utilized depending on the size of the patient's native labrum, and attempts are made to match this as close as possible with the graft selected. If autograft is preferred, an incision centered over the ipsilateral pes anserinus is made. The soft tissue above the sartorius fascia and bursal tissue are cleared, and the upper border of the gracilis tendon is identified. An L-shaped flap can be created to identify the tendons on the back side of the flap, or a split in the sartorius fascia can be made and the ST tendon identified. It is freed from its insertion of the tibia and the adhesions (usually at least two main connections to the gastrocnemius) are released. The tendon is then stripped with the use of a standard tendon-stripping instrument. On the back table, the muscle is stripped from the tendon.

The remainder of the procedure is identical for either allo- or autograft. On the back table, the most robust portion of the tendon is identified, and the graft is cut to the desired length as determined previously. In the case of a more robust native labrum, the tendon can be folded over and whipstitched to make a larger graft if desired. Often the native labrum is very close in size to the ST, and this doubling procedure is unnecessary.

Each end of the tendon is then whipstitched with a high-tensile suture. Leaving the stitches long will allow parachuting the graft into place, and by bringing the stitches out the posterior aspect of the graft a few millimeters shy of the absolute end, it facilitates anastomosis to the native labrum. A mattress type of suture is then placed in the center of the graft, so there are three total sutures at the end of the preparation. This graft is then placed on tension on the back table and protected while the pilot holes for knotless anchors are prepared.

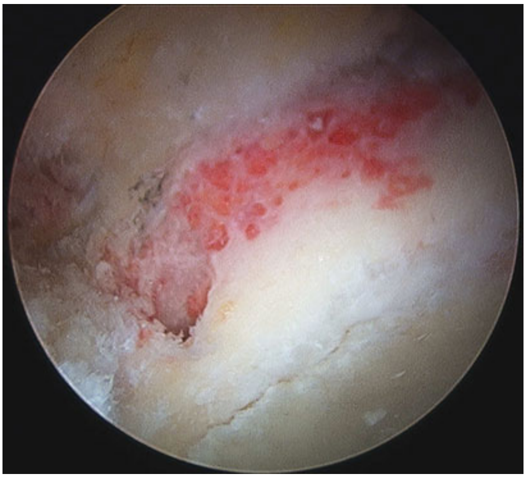


Fig. 3 A pilot hole is shown having been drilled to accept the knotless anchors

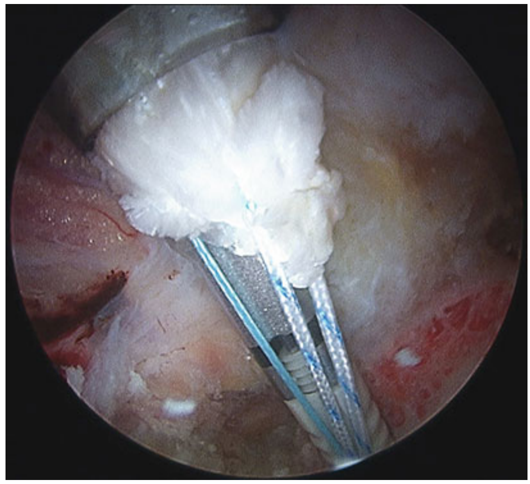


Fig. 4 The graft which has been prepared on the *back* table is parachuted into the joint at the location of the most superior anchor

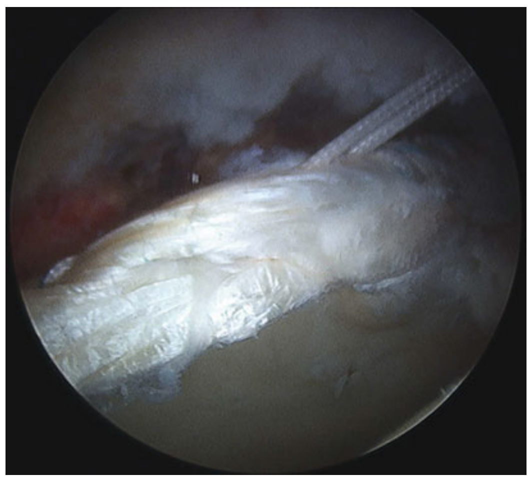


Fig. 5 The central and final knotless anchor is placed and the graft oriented to recreate the suction seal effect of the native labrum

While many anchor choices exist, this technique can be aided with PEEK knotless anchors. Once the graft is prepped, the first pilot hole is placed on the rim just behind the remaining native labrum at the superior aspect of the defect (Fig. 3). It is critically important to be able to visualize this hole later when shuttling the graft into place so a shaver or RF device can be used to clear soft tissue from the whole and define the pilot hole. This location can be accessed often from the DALA portal while looking from either the AL or MMA portal, the AL portal, or the PL portal if it is in a more posterior position. A second pilot hole is then drilled at the center of the labral defect, and a final hole is drilled just behind the anterior remnant. Often the majority of the time, all of these pilot holes can be drilled through the DALA portal with limited to no tension on the drill guide.

An 8.5 × 110 mm plastic cannula is then placed into the DALA portal and the camera into the AL portal. The sutures from one of the ends of the graft are then placed into a knotless PEEK anchor. It can be helpful to utilize an anchor where the tension can be adjusted after the anchor is placed to avoid over- or under-delivering the graft to the proper position. The graft is then parachuted into the joint through this cannula and the anchor malleted into place and then tensioned and locked (Fig. 4). These sutures are then clipped at the anchor. The second anchor placed is the most anterior anchor and is passed in a similar fashion. It is helpful to use a grasper from the MMA portal to help guide the graft into place during the part of the procedure. Once the anchor is locked and tensioned, this stitch is also clipped at the anchor. Finally the central anchor is placed. It is critical to orient the graft properly during this step to be sure it sits on the rim of the acetabulum and not in an everted position (Fig. 5). Once the main three securing anchors are placed,

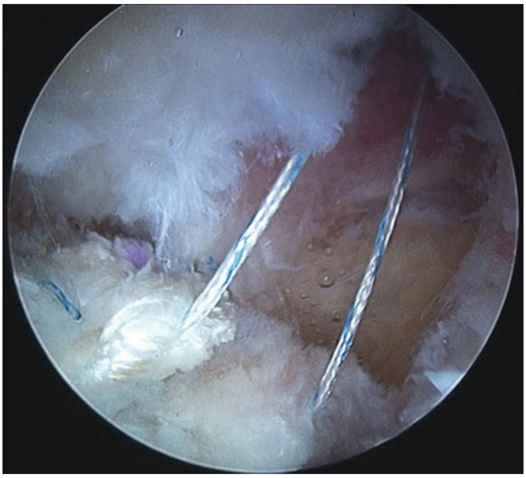


Fig. 6 The graft ends are secured to the native labrum at each end with use of another anchors. It is typical to use 5–7 anchors for this procedure

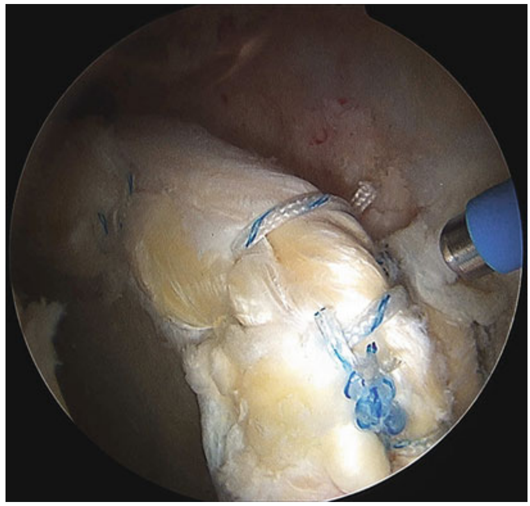


Fig. 7 The appearance of the final construct in place

we will then place a small solid PEEK anchor at the junction of the native labral stump and the transplanted graft. A suture-passing device is then used to pass one suture limb of the anchor through the native labrum and the other through the reserved several millimeters of the graft end (Fig. 6). These are then tied with alternating half hitches, and an effort is made to keep the knot stack away from the joint. This suture is then clipped at the knot once secure. This process is repeated at the other site of anastomosis (Fig. 7). The traction is then released and an assessment of a suction seal is performed (Fig. 8a, b). The femoral head is rotated and the hip flexed and extended to ensure the graft is secure and stable.

Other procedures such as microfracture, revision femoral osteochondroplasty, capsular repair, or reconstruction can be performed as necessary.

Postoperative Management

Postoperatively the patient is placed into a commercially available brace for 3 weeks and kept heel touch weight bearing for a similar amount of time. A standard hip physical therapy program focusing on early range of motion and deep and soft tissue releases with progressive strengthening beginning on postoperative day 1 is initiated and continued 2x/week for 3 months. Aquatic activities are allowed at 6 weeks post-op with straight ahead running allowed at 3 months. Return to pivoting and higher-level activity is allowed between 4.5 and 6 months from surgery once range of motion, strength, and pain levels are felt to be at appropriate levels by the treating surgeon.

Results

While several studies report outcomes after labral reconstruction, most of these utilize iliotibial band autograft. Only one study reports the outcomes of hamstring graft, in particular, gracilis autograft [1]. This study, by Matusda and Burchette, compared labral reconstruction to primary repair. They performed a level III cohort study, retrospectively comparing a similar group of patients with similar treatment (treatment of bony morphology), labral repair or reconstruction being the primary difference. The labral repair cohort had 46 patients compared to eight in the reconstruction group. The primary outcome measure was the non-arthritic hip score (NAHS), and the mean follow-up was 2 years. The mean postoperative NAHS was higher in the reconstruction group (78 vs. 92) as was the total mean points gained (22 vs. 50). Further statistical modeling to account for asymmetry in group size found the outcomes

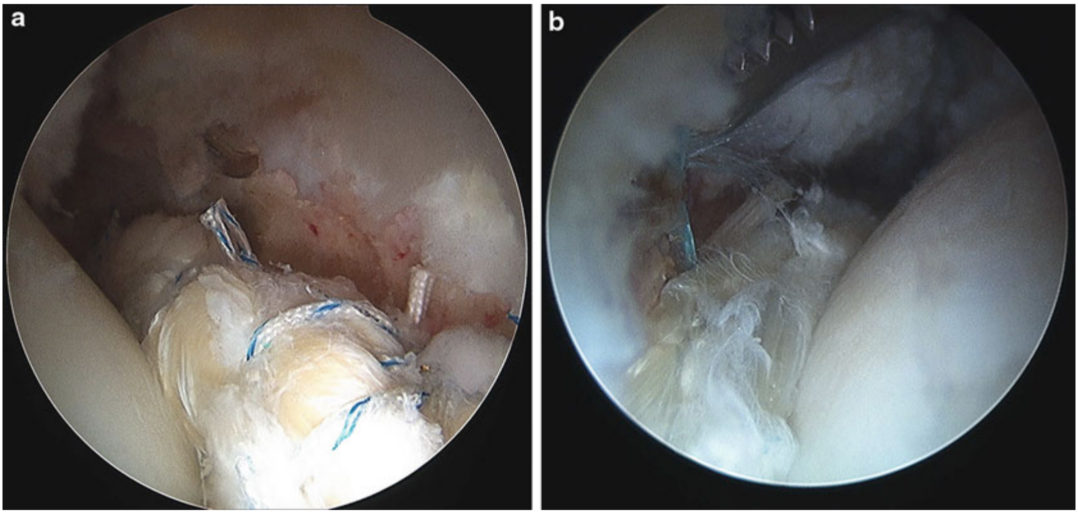


Fig. 8 (**a**, **b**) These images show the recreation of the suction seal by the reconstructed labrum

for each group were essentially equal. This study demonstrates that labral reconstruction with hamstring autograft is a safe and reliable procedure with outcomes as good as primary repair. The technique for this chapter was also published [2].

Summary

The evidence continues to grow supporting the efficacy of labral reconstruction. The indications range from failed labrectomy to primary reconstruction when the existing labrum cannot be repaired to recreate normal anatomy. The technique for either graft is similar but hamstring auto- or allograft may simply be the procedure. Allograft reduces the length of the procedure, and hamstring autograft harvest is familiar to many surgeons. Since labral reconstruction is infrequent even for high-volume hip arthroscopists, the use of hamstring graft may simplify this complex procedure.

References

1. Matsuda DK, Burchette RJ. Arthroscopic hip labral reconstruction with a gracilis autograft versus labral refixation: 2-year minimum outcomes. Am J Sports Med. 2013;41(5):980–7.
2. Matsuda DK. Arthroscopic labral reconstruction with gracilis autograft. Arthrosc Tech. 2012;1(1):e15–21.

Surgical Technique: Open Hip HS Allograft

97

K. Corten, K. Carpentier, and Michael Leunig

Contents

Abstract

Femoroacetabular impingement is a well-known cause of hip pain. When impingement is caused by a relative over-coverage of the femoral head, it is called pincer impingement. A possible treatment option consists of rim trimming. Pincer impingement is often associated with labral thinning and degeneration. If the labrum is not repairable, a labral reconstruction is needed to restore labral function.

The preferred surgical technique for a circumferential labral reconstruction is an open reconstruction using a hamstring allograft. A surgical dislocation is performed according to the technique described by Ganz et al. After performing the rim trimming, a reconstruction of the labrum using a semitendinosus allograft is performed. This graft is prepared and then fixed to the rim using bone anchors from anterior to posterior. Both ends of the graft are also firmly fixed to the transverse acetabular ligament. This reconstruction ensures a 360° repair of the sealing function of the labrum.

Introduction

Femoroacetabular impingement is characterized by abutment of the femoral neck against the acetabular rim and is a well-recognized cause of hip pain and degenerative changes of the hip joint. This impingement is either caused by a deformation of the proximal femur (cam deformity), by an

K. Corten (✉)
Department of Orthopaedic Surgery, Ziekenhuis Oost-Limburg, Genk, Belgium
e-mail: kristoffcorten@me.com

K. Carpentier
AZ St Blasius, Dendermonde, Heverlee, Belgium
e-mail: karel.carpentier@gmail.com

M. Leunig
Department of Orthopaedic Surgery, Schulthess Clinic, Zürich, Switzerland
e-mail: michael.leunig@kws.ch

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_106

Fig. 1 This 45-year-old female presented with groin pain at the right hip. She has got global pincer impingement caused by coxa profunda. The teardrop is located medial to the ilio-ischial line. The center-edge angle is 45°

over-coverage of the acetabulum (pincer impingement), or by a combination of both. This abutment might cause labral and cartilage damage and will eventually induce progressive degeneration of the hip joint.

Pincer impingement is associated with acetabular-sided abnormalities such as acetabular retroversion, coxa profunda, or protrusio acetabuli. The latter two represent a typical morphology of generalized over-coverage of the femoral head by the acetabular rim (Fig. 1). As a result, the femoral neck will abut against the acetabular rim. It is hypothesized that this can cause periosteal irritation followed by bone apposition and overgrowth of the labrum, which in turn will become stretched [1]. The resultant labral thinning will jeopardize the sealing function especially when the acetabular rim had been surgically removed and the remnants of the labrum are fixed to the acetabular bone [2–4]. Because of the generalized aspect of the acetabular over-coverage, the impingement needs to be treated with an anterior-to-posterior and sometimes even circumferential rim trimming. As the posterior rim is difficult to be reached by hip arthroscopy, this can only adequately be conducted by means of an open surgical dislocation procedure. Since the labrum is quite thin, it is frequently not sufficient to be used for refixation. To reconstruct the deficient labrum, a circumferential allograft can be used. A circumferential labral reconstruction also allows recreating ring tension, which is not possible when segmental reconstructions are performed.

Fig. 2 The recess sign is a radiographic indentation sign of the lateral acetabular rim indicating an overgrowth of the labrum by the acetabular bone [1]. This indicates that the labrum is stretched and will be hypoplastic. This labrum probably needs reconstruction after rim trimming

This chapter describes a surgical reconstruction technique that involves anterior-to-posterior rim trimming followed by a circumferential reconstruction of the labrum with a semitendinosus allograft to restore the labral sealing function.

Surgical Technique

Preop planning. A reconstruction of the labrum is anticipated in case of radiographic signs of generalized impingement in accordance to specific signs for labral stretching such as the recess sign (Fig. 2). On a preoperative x-ray of the pelvis, the lateral center-edge angle of Wiberg is defined [5, 6]. This angle should be $\leq 39°$ and a rim

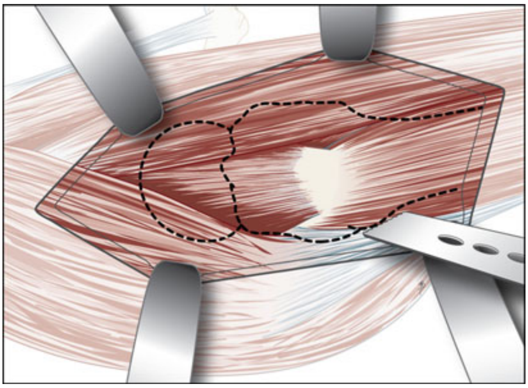


Fig. 3 The Gibson approach opens the interval between the gluteus maximus and the tensor fascia lata. The greater trochanter is visualized and the sliding or Z-shaped osteotomy is prepared

trimming is planned with a resection of 1 mm for every degree of over-coverage [7]. The patient should be informed about the potential need for allograft tissue.

Procedure. The surgical dislocation is performed according to the technique described by Ganz et al. [8]. In brief, the patient is positioned in lateral decubitus. The Gibson approach is conducted and the tip of the greater trochanter is identified in 30° of internal rotation (Fig. 3). The piriformis and gluteus minimus muscles are identified. The trochanteric branch of the medial circumflex artery is identified and a Z-shaped step-cut osteotomy of the trochanter is made. Approximately 3 mm of the tip of the trochanter are left in place. Care is taken to leave the anterior cortex intact in order to fracture the cortex, which will ensure anterior stability when the osteotomized fragment is put back in place. The gluteus minimus muscle is then carefully released from the underlying capsule, which will be incised in a Z-shape to avoid damaging the medial femoral circumflex artery at the level of the femoral neck (Fig. 4). The femoral head is then dislocated out of the acetabulum with an external rotation-flexion maneuver. The ligamentum teres is cut with a curved scissors, which then allows for posterior dislocation of the head beyond the posterior rim. Anterior, superior, and posterior retractor placement provides full visualization of the hip joint and the acetabular rim (Fig. 5).

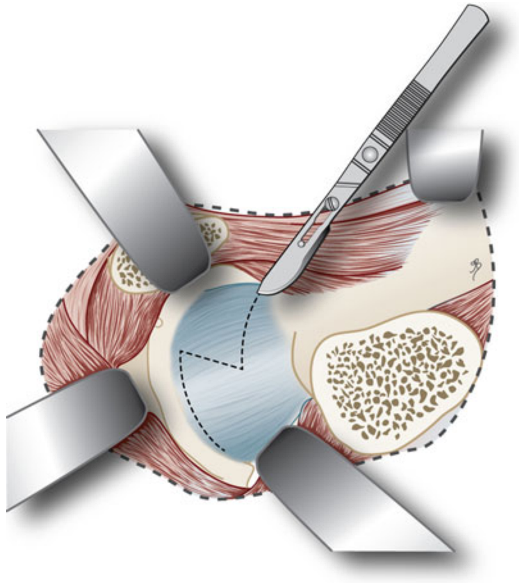


Fig. 4 The capsule is incised in a Z-fashion in order to protect the retinacular vessels that penetrate the femoral head a distance of 4–5 mm from the postero-superior head neck junction

The hip joint is inspected and the labrum is then circumferentially detached from the acetabular rim. Most often the labrum is thinned and will not provide sufficient coverage of the head to ensure an adequate sealing function (Fig. 6). A semitendinosus allograft is used because it is long enough (8–15 cm), will be thick enough to provide adequate coverage, and is not associated with comorbidities of the graft harvesting. Therefore, the semitendinosus allograft is preferred to reconstruct the labrum. The graft is thawed and prepared during rim trimming. The length of the graft can be measured with the debrided labrum or with a pliable caliper. Both tendon ends are secured to an ACL-preparation kit with an absorbable suture (Vicryl 1) using a Krackow stitch. A tubular double loop is created using a baseball stitch with absorbable sutures (Vicryl 2/0). Both tendon strands are sewn to each other over the full length of the graft (Fig. 7).

The rim is appropriately trimmed after the labrum had been removed. It is crucial to keep the transverse acetabular ligament intact because the graft will be fixed to the ligament in order to create a circumferential labrum reconstruction.

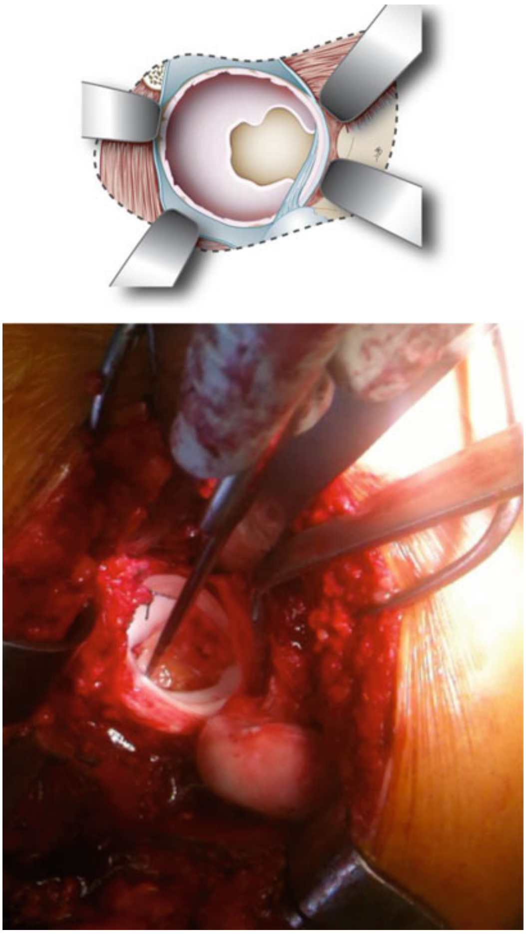


Fig. 5 The femoral head is dislocated and the view on the acetabulum is excellent from anterior to posterior. The transverse acetabular ligament is seen and is used as a landmark to fix the graft circumferentially. The labrum is hypoplastic and very thin. Therefore, the sealing function is expected to be insufficient

Fig. 6 Remnants of a hypoplastic labrum

Bone anchors are placed at a distance of 3–4 mm from the rim with an angle of 45° to the rim and parallel to the acetabular articular surface. The distance between the anchors is approximately 8–10 mm starting from the anterior insertion of the transverse ligament in an anterior-to-posterior direction (Fig. 8). Access to the posterior rim can be facilitated by slightly increasing the external rotation and increasing the abduction to move the femoral head in the posterior direction away from the posterior rim. The sutures of the first (anterior) anchor are pulled through the graft at a distance of 2 cm from the end of the graft. The graft is provisionally fixed to the rim and brought under tension. Next the following suture is pulled to the graft and fixed to the rim. Finally both free ends of the graft are firmly fixed to the transverse ligament using the Vicryl 2/0 sutures. This final step ensures a 360° sealing function of the reconstructed labrum (Fig. 9). Before reducing the femoral head, several absorbable sutures are placed in the graft and keep the graft under tension during reduction of the femoral head. This will prevent entrapment of the graft during reduction of the head into the acetabulum. In case of combined impingement, the femoral neck is reshaped by means of cam resection with a curved chisel. A template is used to ensure proper reshaping of the femoral neck.

After reduction, the hip is assessed for impingement during a full range of motion. The capsule is closed with Vicryl 1 sutures and the trochanter osteotomy is closed and fixed with two cortical screws. Postoperatively, partial weight bearing (50 % of body weight) is allowed for 6 weeks with two crutches. Physiotherapy is started after 4–6 weeks.

Fig. 7 A prepared allograft that provides sufficient coverage and restores the sealing function

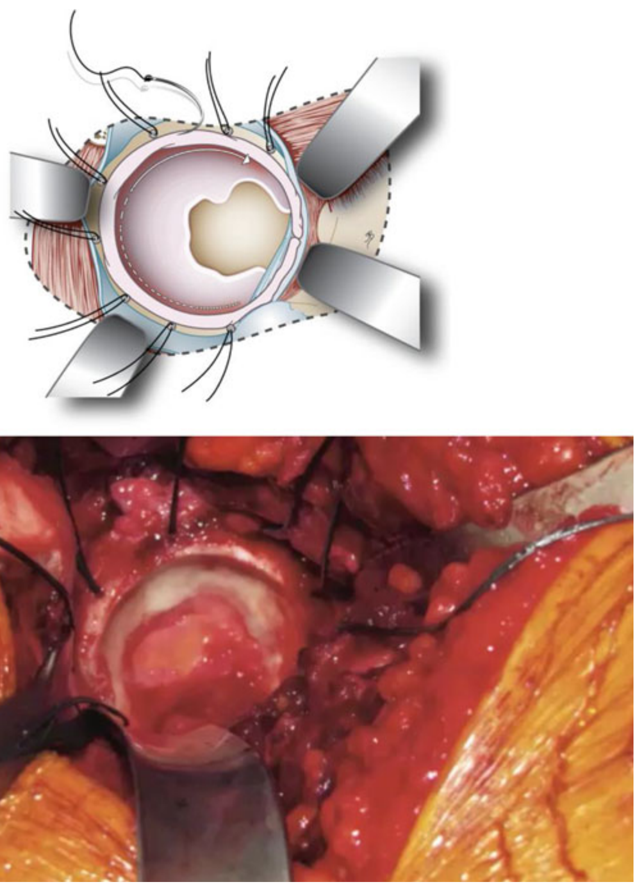


Fig. 8 The anchors are inserted at the acetabular rim after removal of the labrum. The graft (indicated in *pink*) must be long enough so it can be inserted circumferentially starting from the anterior rim at the anterior insertion of the transverse acetabular ligament

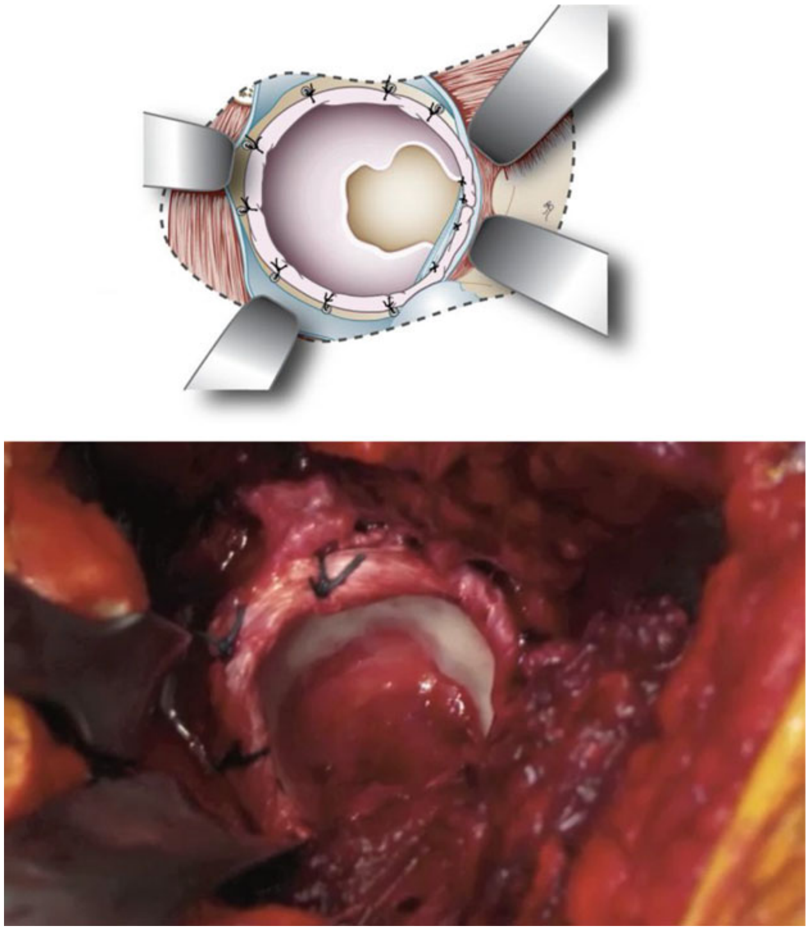


Fig. 9 The graft is fixed to the acetabular rim and sutured to the transverse acetabular ligament

Summary

Pincer impingement is often associated with labral thinning and degeneration. Treatment includes not only rim trimming but also restoring the labral function. The labrum is often to degenerative to allow a repair and a labral reconstruction is needed. To restore a 360° sealing function, an open reconstruction with a semitendinosus allograft is the preferred technique. To allow a good visualization of the acetabulum, an open dislocation of the hip is performed, and a semitendinosus graft is fixed to the acetabular rim with bone anchors. Additionally both ends of the graft are sewn to the transverse ligament. This technique restores the function of the labrum and allows for other femoral or acetabular deformities causing impingement to be treated during the same procedure.

References

1. Corten K, Ganz R, Chosa E, Leunig M. Bone apposition of the acetabular rim in deep hips: a distinct finding of global pincer impingement. J Bone Joint Surg Am. 2011;93 Suppl 2:10–6. Epub 2011/05/13.
2. Adeeb SM, Sayed Ahmed EY, Matyas J, Hart DA, Frank CB, Shrive NG. Congruency effects on load bearing in diarthrodial joints. Comput Methods Biomech Biomed Engin. 2004;7(3):147–57. Epub 2004/10/30.
3. Ferguson SJ, Bryant JT, Ganz R, Ito K. An in vitro investigation of the acetabular labral seal in hip joint mechanics. J Biomech. 2003;36(2):171–8. Epub 2003/01/28.
4. Hlavacek M. The influence of the acetabular labrum seal, intact articular superficial zone and synovial fluid thixotropy on squeeze-film lubrication of a spherical synovial joint. J Biomech. 2002;35(10):1325–35. Epub 2002/09/17.
5. Fredensborg N. The CE, angle of normal hips. Acta Orthop Scand. 1976;47(4):403–5. Epub 1976/08/01.

6. Wiberg G. Studies on dysplastic acetabula and congenital subluxation of the hip joint. Acta Chir Scand. 1939;83:58.
7. Philippon MJ, Wolff AB, Briggs KK, Zehms CT, Kuppersmith DA. Acetabular rim reduction for the treatment of femoroacetabular impingement correlates with preoperative and postoperative center-edge angle. Arthroscopy. 2010;26(6):757–61. Epub 2010/06/01.
8. Ganz R, Gill TJ, Gautier E, Ganz K, Krugel N, Berlemann U. Surgical dislocation of the adult hip a technique with full access to the femoral head and acetabulum without the risk of avascular necrosis. J Bone Joint Surg. 2001;83(8):1119–24. Epub 2002/01/05.

Part IX

Synovial Disorders

Synovial Disorders of the Hip

98

Brian McGrath and Joseph Kuechle

Contents

Abstract

The hip is a synovial joint which links the axial skeleton to that of the lower extremity by the articulation of the femur and acetabulum. The tissue of the synovium which lines the joint has the important role of regulation of synovial fluid production but also can be a source of significant but rare pathology. These disorders include synovial chondromatosis and pigmented villonodular synovitis (PVNS). Synovial chondromatosis will typically present with monoarticular pain and stiffness with mechanical complaints of locking and catching during the third to fifth decade of life due to the generation of loose bodies. Pigmented villonodular synovitis is another benign proliferative disorder of the synovium. While the lesion is benign, it can be locally aggressive and cause the destruction of both intra- and extra-articular structures in the third and fourth decades of life. Both disorders are treated with arthroscopic or open debridement with synovectomy. Synovial disorders of the hip are fortunately very rare but can be devastating. Surgical treatment, when undertaken in a timely fashion, can be effective at preserving hip function.

Introduction

The hip joint is a synovial joint which links the axial skeleton to that of the lower extremity by the articulation of the round head of the femur

B. McGrath (✉) • J. Kuechle
Department of Orthopedics, State University of New York, Buffalo, NY, USA
e-mail: bmcg112960@aol.com; jkuechle@gmail.com

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_107

and the cup-like acetabulum. The tissue of the synovium of the hip is a thin surface that lines the capsule surrounding the joint. This tissue has the important role of regulation of the production of synovial fluid which lubricates the joint. It can be affected in inflammatory processes such as osteoarthritis, rheumatoid arthritis, and transient synovitis but also can be the source of significant but more rare pathology in the hip. These disorders include synovial chondromatosis and pigmented villonodular synovitis (PVNS). This chapter will review the clinical presentation, pathophysiology, and treatment of these later more rare disorders which affect the hip.

It is important to understand the normal form and function of the synovium first in order to understand its pathophysiology. The layer lining the capsule of the joint is called the intimal layer and is typically from one to three cell layers thick. There are two cell types in this layer: a macrophage-like cell type (type A) and fibroblastic (type B) [1]. The type A cells, which are monocyte derived, are involved in phagocytosis of debris and waste in the joint cavity as well as antigen-presenting cells. The type B cells, which are very metabolically active and contain significant amount of rough endoplasmic reticulum, are active in the production of hyaluronan, collagens, and fibronectin. Underlying this layer is a fragmented and limited basement membrane. The cells making up the intima do contain junctional apparatus such as desmosome and gap junctions but not as tight as is seen in other epithelia [2]. The subintima is the layer of tissue below this rudimentary membrane which interfaces with surrounding tissue and typically consists of a loose areolar tissue made up of mostly fibroblastic cells but can be fibrous or fatty depending on its location in the joint and what joint is involved. This layer typically has an extensive capillary network which provides the joint fluid. Below this layer is the stratum fibrosum, a dense connective tissue that makes up the joint capsule.

Synovial Chondromatosis

Pathophysiology

Synovial chondromatosis is one of these rare synovial disorders that are highlighted here. It is a benign monoarticular process. It involves the metaplastic transformation of sub-synovial membrane tissue which results in the generation of multiple cartilaginous nodules in joints, tendon sheaths, or bursae [3]. The loose bodies that are generated range in size from 1 mm to 3 cm. These bodies are often created at the synovium-cartilage junction. The loose bodies can continue to grow in diameter by metaplasia of the synovial layer while the central zones calcify. This pathological process in the synovium is nonaggressive in that the tissue itself does not invade or erode the neighboring tissue. While the tissue itself is not directly destructive, the mechanical presence of loose bodies themselves within the joint can cause pain, debilitation, and ultimately destruction of intra-articular cartilage and lead to arthritis. The pathogenesis of synovial chondromatosis is currently unknown. Many consider it to be a reactive process that is self-limiting [4]. However, there has been some cytogenetic analysis that suggests that at least some of the cases do possess a clonal neoplastic origin with consistent chromosomal rearrangement patterns as well as aneuploidy [5, 6].

Two forms of synovial chondromatosis have been described. These forms include a primary and a secondary form, both of which involve metaplastic transformation of the sub-synovial layers [7]. The primary form is a more diffuse form involving the entirety of the synovium. It results in small, more numerous, uniform loose bodies. This form has a higher incidence of recurrence. The secondary form is more focal and results in large, fewer loose bodies and likely is caused by metaplasia induced by chondral fragments from intra-articular pathology such as osteoarthritis, inflammation, infection, or traumatic osteochondral fracture.

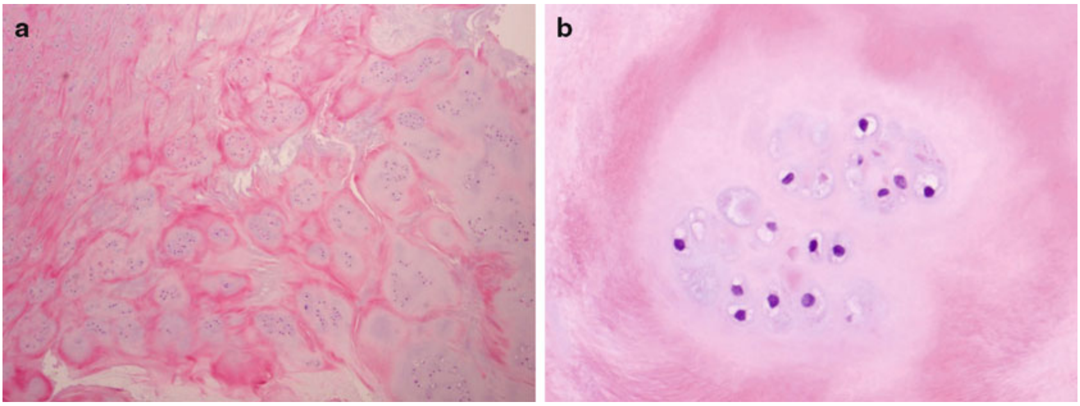


Fig. 1 (**a**) 4× and (**b**) 40× H + E stain from loose bodies after surgical removal demonstrating discrete hyaline cartilage nodules with chondrocytes with mild atypia (Courtesy of University at Buffalo Orthopedic Oncology Database)

For both types, the natural history that Milgram had described is still thought to be applicable. This includes the disease process progressing through three stages [8]. The first stage is active intrasynovial disease without loose bodies, and the second stage involves active synovial proliferation with loose bodies. In the third stage, there are multiple loose bodies without intrasynovial disease. It is felt that by the third stage, the process that had generated the pathology has since "burned itself out."

On histology, the removed tissue will appear as focal islands of disorganized hyaline cartilage in the synovium. This will include the loose bodies as well as the synovia lining the joint. The cartilage can appear aggressive given the hypercellularity and enlarged chondrocytes and may have significant pleomorphism of the nuclei (Fig. 1).

Clinical Features and Diagnosis

The clinical presentation for synovial chondromatosis is that of a gradual onset of monoarticular pain and stiffness with mechanical complaints of locking and catching. In the hip specifically clinical signs are more obscured since it is more difficult to detect loose bodies or an effusion on exam. It most commonly involves the knees (50–65 %) of cases, followed by elbows (20–25 %) while hips account for only 10 % of cases. Males are twice as likely to be affected as females. It typically presents during the third to fifth decades of life [9].

X-rays are normal in greater than 50 % of cases because the cartilage in the loose bodies is not well ossified and can result in delayed diagnosis. By the time loose bodies are apparent, the disease can have progressed significantly, and multiple radiopaque round or oval lesions are seen as well as a joint effusion, degenerative arthritis, osteophytes, and subchondral sclerosis (Fig. 2). CT scan can be useful to visualize loose bodies that are not seen on X-ray and will demonstrate distension of the capsule. MRI is also often useful if there is an unclear diagnosis from radiographs. Kramer et al. described three different types of MRI imaging [10]. Pattern A (12 %) is described as a lobulated homogeneous intra-articular signal isointense to muscle on T1-weighted images and hyperintense on T2-weighted images. Pattern B (80 %) incorporates the features of pattern A with addition of signal void foci on all pulse sequences. Pattern C (8 %) has features of patterns A and B plus foci of lesions with peripheral low signal surrounding a central fatlike signal. The significance of the various imaging in prognosis and pathogenesis is unclear at this point. Magnetic

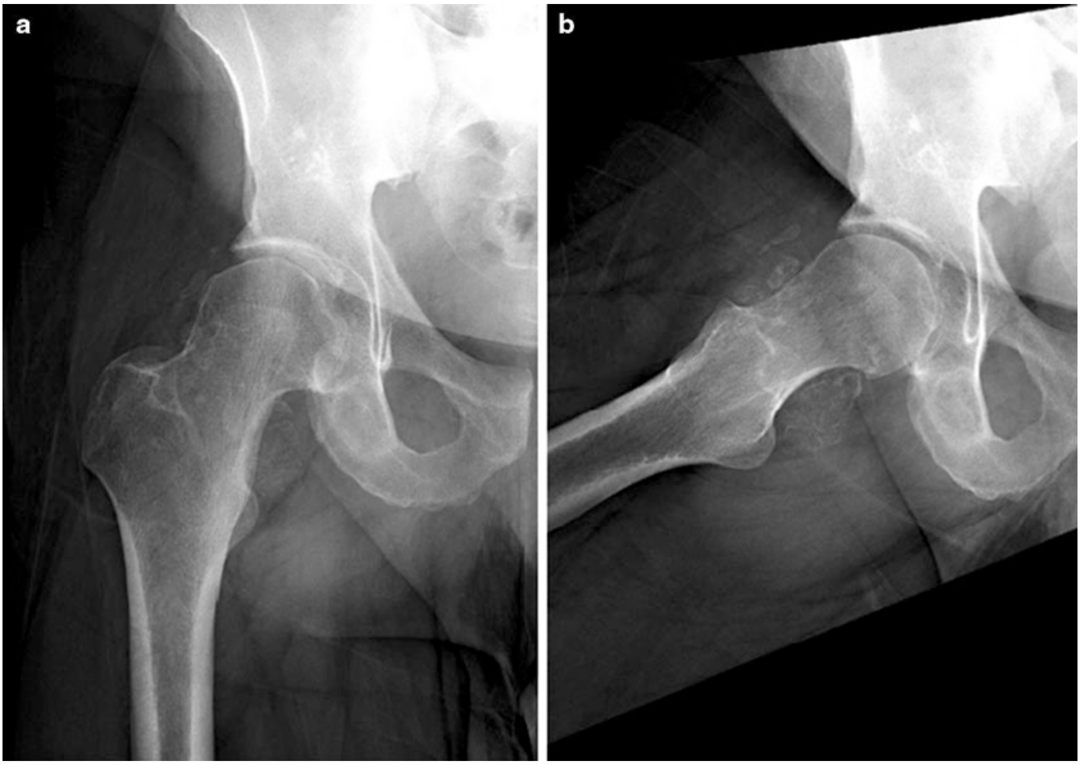


Fig. 2 (**a**) AP and (**b**) frog leg lateral radiographs of a 42-year-old male with right hip pain associated with synovial chondromatosis. Note the loose bodies seen throughout the hip joint and arthritic changes seen in the femoral head (Courtesy of University at Buffalo Orthopedic Oncology Database)

Fig. 3 MRI arthrogram coronal (**a**) T1 and (**b**) STIR sequences from a pelvis of a 42-year-old male with right hip pain associated with synovial chondromatosis. Again loose bodies are seen within the hip joint (Courtesy of University at Buffalo Orthopedic Oncology Database)

resonance arthrography with gadolinium contrast will show multiple filling defects in the joint (Fig. 3). Bone scan may show increased uptake around calcified loose bodies.

Other causes of loose bodies and intra-articular pathology need to be considered when considering a diagnosis of synovial chondromatosis. The differential includes tumors, synovial sarcoma, fragmented osteophytes in OA, or osteochondral dissecans. However, in these conditions, loose bodies will typically be smaller in both number and size. Intra-articular calcifications can also be seen with rare hemangiomas, villonodular synovitis, or post-septic arthritis.

Treatment

The treatment of synovial chondromatosis includes surgical removal of loose bodies with partial or complete synovectomy. While it has been agreed that it is necessary to remove the loose bodies in order to relieve the symptoms of pain, stiffness, and locking that the patients often present with, the role of synovectomy to prevent further damage is currently still debated. In Milgram stage 3 and secondary disease, synovectomy may not be as important since there is less expectation of the generation of further loose bodies. However, in diffuse primary disease, evidence has accumulated that synovectomy is very important in preventing recurrence in both the knee and the hip [11].

In the hip, the standard treatment involves a surgical dislocation in order to facilitate loose body excision and synovectomy. This surgical dislocation can be performed through a Watson-Jones approach or through an anterior Smith-Peterson approach with Ganz dislocation of the hip [12–14]. Some authors have described using the trochanteric osteotomy to improve visualization, allow dislocation, and assure complete synovectomy. Occasionally, a small posterior capsulotomy has been performed to facilitate removal of loose bodies [15]. Successful arthroscopic management of synovial chondromatosis has also been described. However, this involves selective patients with limited focal disease and increases the risk of recurrence [16–19]. Recurrence rates with synovectomy have been described between 0 % and 23 %.

If synovial chondromatosis is not diagnosed early and treated promptly, the loose bodies themselves can cause significant damage to the labrum and cartilage generating secondary osteoarthritis. Late complications such as secondary hip subluxation [20] and femoral neck fractures have been described [21]. Also, rare but described has been degeneration into chondrosarcoma [22]. The outcome for many of these patients with significant articular damage may be total joint arthroplasty or resurfacing [23, 24]. Even in the presence of an arthroplasty, there is the possibility of recurrence of the disease.

Pigmented Villonodular Synovitis

Pathophysiology

Pigmented villonodular synovitis is another benign proliferative disorder of the synovium in the joint, tendon sheath, and bursa. While the lesion is benign, it can be locally aggressive and cause the destruction of both intra- and extra-articular structures. The true pathophysiology of this process is still incompletely understood. Jaffee et al. considered the disease to be a reactive inflammatory response [25]. In a recent review, 56 % of patients with PVNS were associated with trauma to the joint [26]. There is reason to believe that it has neoplastic characteristics as evidenced by its monoclonality, metastatic potential [27], presence of chromosomal abnormalities [28], and aneuploidy [29]. PVNS is characterized by a proliferation of synovial-like mononuclear cells, with multinucleated giant cells, lipid- or hemosiderin-laden macrophages, and inflammatory cells. The hypertrophic, hypervascular synovial tissue will have villous, nodular, or a combination of proliferative shapes. Hemosiderin is deposited in the macrophages within the synovium. The inflammatory cells secrete cytokines which stimulate osteoclasts to act and resorb both periarticular bone and damage cartilage.

There are two types of PVNS described including a diffuse and a localized type. It most often affects the knee rather than the hip, but any synovia can be affected. The diffuse type can be more aggressive and recur more frequently. It has high cell proliferation rate and cell atypia. The focal or localized forms often occur as discrete nodular lesions involving tendon sheaths and are often known as giant cell tumors of the tendon sheath.

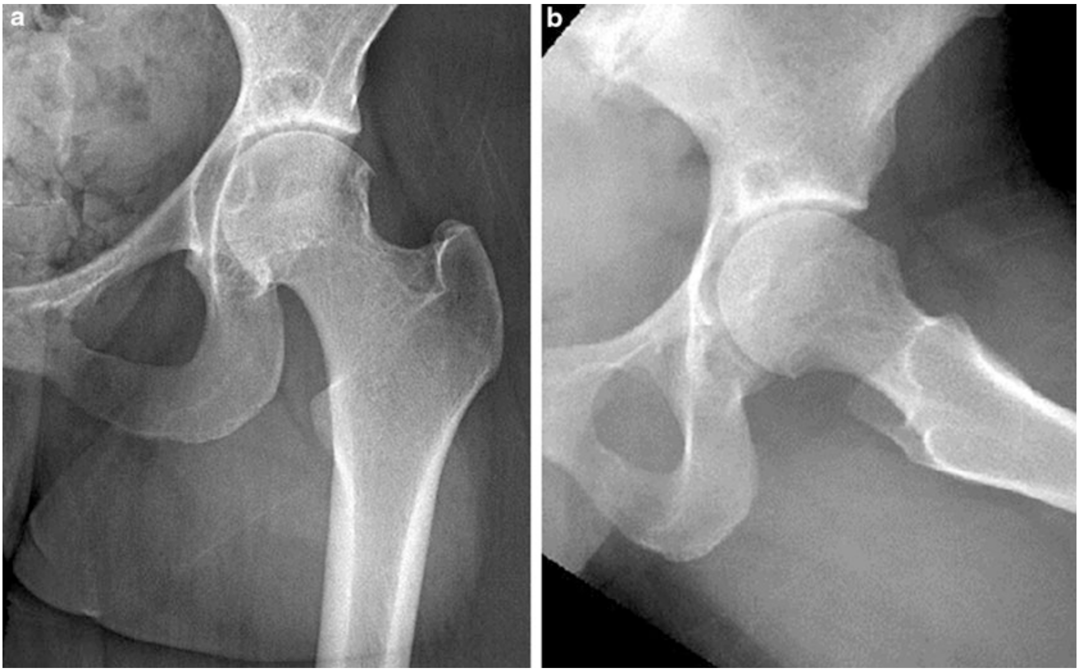


Fig. 4 (**a**) AP and (**b**) frog leg lateral of a 28-year-old female with left hip pain due to PVNS. Early erosive changes are demonstrated (Courtesy of University at Buffalo Orthopedic Oncology Database)

These occur more often in the hands and feet but can be present in the hip. These lesions are often well circumscribed and have a lower recurrence rate.

Clinical Features and Diagnosis

The presentation of PVNS in the hip involves slowly progressive monoarticular pain, stiffness, locking, and decreased range of motion. Patients may report a slow-growing mass that is palpated in the local form. In the diffuse articular form, there will be pain with extremes of range of motion of the hip. It presents in slightly higher frequency in women than men and is most frequently encountered in the third and fourth decades [30].

Radiographs of the hip will commonly show bone erosion or cysts. Joint space is typically preserved and there may be an effusion [31, 32] (Fig. 4). In early disease, radiographs are commonly normal. Hemosiderin will be detected on CT scan, and this form of imaging can be useful to determine the extent of the synovial involvement and bone erosion or cysts. Diffuse thickening of the tissue around the joint will be demonstrated.

On MRI "bloom" artifacts will be created by the hemosiderin deposits and appear as low or absent petal-shaped areas on both T1- and T2-weighted images which distort the surrounding image (Fig. 5). MR arthrogram will show extensive synovial thickening with villous or nodular projections. It is not expected to see multiple filling defects like synovial chondromatosis [33]. PET imaging has been used to document treatment when following recurrent disease [34] but not for primary diagnosis.

On gross examination of the tissue, the diffuse form of PVNS is a tan mass of villi and folds of synovium. There can be both sessile and pedunculated nodules present. The local form of PVNS is usually a well-circumscribed, pedunculated, firm nodule. PVNS on histological examination will show synovial cell hyperplasia both on the surface of the synovium and deeper into the involved tissue. Giant cells,

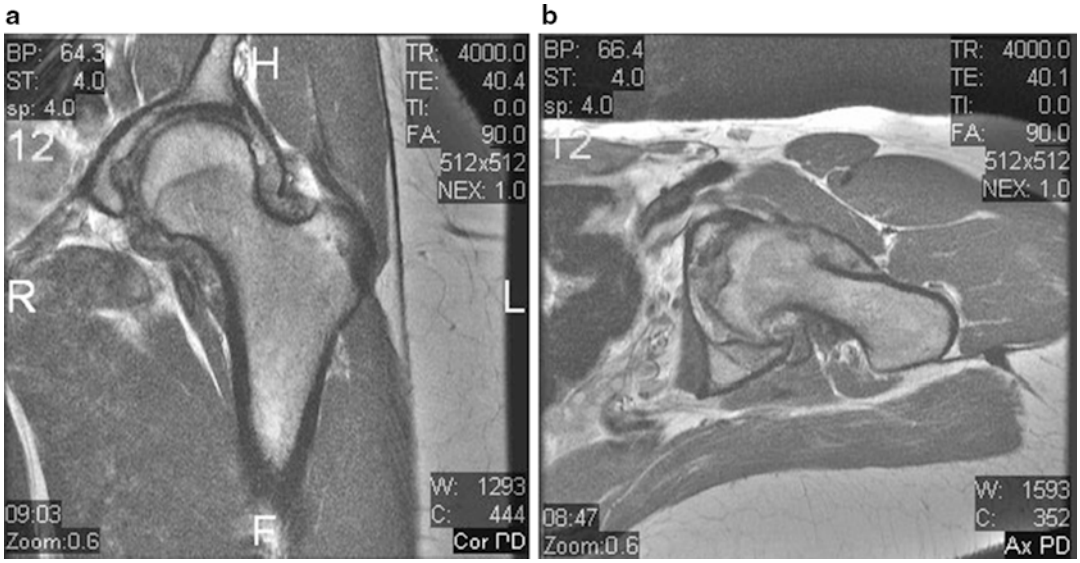


Fig. 5 (**a**) Coronal and (**b**) axial cuts from T1 MRI from a 28-year-old female with *left* hip pain associated with PVNS. Present is thickened villous synovium and bloom artifact (Courtesy of University at Buffalo Orthopedic Oncology Database)

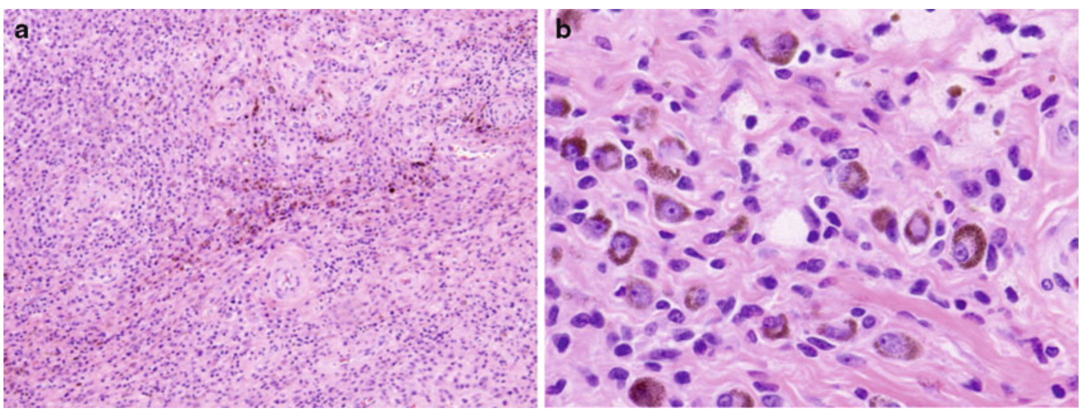


Fig. 6 10× (**a**) and 40× (**b**) magnification of H + E staining of pathology from the surgical excision of hip PVNS from a 28-year-old female. Present are hemosiderin-laden macrophages and multinucleated giant cells in a cellular stroma (Courtesy of University at Buffalo Orthopedic Oncology Database)

hemosiderin, and foam cells will be scattered throughout (Fig. 6).

Other disease processes to consider in the differential diagnosis of this lesion are hemosiderotic synovitis, rheumatoid arthritis, and synovial chondromatosis. The unique features of PVNS which allow diagnosis are the significant amount of bony erosion seen with PVNS and the characteristic bloom artifacts on MRI which are not seen in these other disease processes.

Treatment

The standard treatment for PVNS both focal and diffuse is surgical synovectomy. While local disease can be treated with arthroscopy techniques, the challenge of complete synovectomy often requires an open approach. Vastel et al. used an anterior dislocation through direct lateral approach and had no symptomatic recurrences in a mean of 16.7 years [35]. Treatment with the goal of saving the hip in PVNS of the hip has a high failure rate partly because of periarticular acetabular and femoral head destruction in the enclosed hip capsule, as well as because surgical resection can be difficult.

There does not appear to be sufficient evidence for dysprosium or yttrium intra-articular injection as recurrence rate is high at 18 % [36, 37]. Radiation may have some benefit in recalcitrant cases in both the knee and the hip, and low- to moderate-dose (16–25 Gy) external beam radiation has been used in combination with surgical synovectomy [38]. As of yet, no known chemotherapeutic agents that are useful for control of the disorder. Overall it appears that the thoroughness of the synovectomy is the most important factor in preventing recurrence.

When extensive bony destruction has taken place, treatment often involves total hip replacement [39] or hip resurfacing [40]. The rate of recurrence has been shown to be lower with arthroplasty given the wider dissection but is still present in low percentages. Therefore, all patients should be monitored for recurrence even if they have had a hip replacement. Malignant transformation has been described in the literature; however, it is not clear if this is directly related to PVNS or is a different entity as it is such a rare pathology.

Summary

Synovial disorders of the hip are fortunately very rare but should be in consideration for the differential diagnosis whenever the histories, physical, and imaging are not consistent with more common hip pathologies. Surgical treatment when undertaken in a timely fashion can be effective for these forms of pathological disorders.

References

1. Iwanaga T, et al. Morphology and functional roles of synoviocytes in the joint. Arch Histol Cytol. 2000;63(1):17–31.
2. Barland P, Novikoff AB, Hamerman D. Electron microscopy of the human synovial membrane. J Cell Biol. 1962;14:207–20.
3. Maurice H, Crone M, Watt I. Synovial chondromatosis. J Bone Joint Surg Br. 1988;70(5):807–11.
4. Apte SS, Athanasou NA. An immunohistological study of cartilage and synovium in primary synovial chondromatosis. J Pathol. 1992;166(3):277–81.
5. Buddingh EP, et al. Chromosome 6 abnormalities are recurrent in synovial chondromatosis. Cancer Genet Cytogenet. 2003;140(1):18–22.
6. Davis RI, et al. Cell proliferation studies in primary synovial chondromatosis. J Pathol. 1998;184(1):18–23.
7. Villacin AB, Brigham LN, Bullough PG. Primary and secondary synovial chondrometaplasia: histopathologic and clinicoradiologic differences. Hum Pathol. 1979;10(4):439–51.
8. Milgram JW. Synovial osteochondromatosis: a histopathological study of thirty cases. J Bone Joint Surg Am. 1977;59(6):792–801.
9. Murphy FD, Dahlin DC, Sullivan CR. Articular synovial chondromatosis. J Bone Joint Surg. 1962;44(1):77–86.
10. Kramer J, et al. MR appearance of idiopathic synovial osteochondromatosis. J Comput Assist Tomogr. 1993;17(5):772–6.
11. Ogilvie-Harris DJ, Saleh K. Generalized synovial chondromatosis of the knee: a comparison of removal of the loose bodies alone with arthroscopic synovectomy. Arthroscopy. 1994;10(2):166–70.
12. Schoeniger R, et al. Modified complete synovectomy prevents recurrence in synovial chondromatosis of the hip. Clin Orthop Relat Res. 2006;451:195–200.
13. Gilbert SR, Lachiewicz PF. Primary synovial osteochondromatosis of the hip: report of two cases with long-term follow-up after synovectomy and a review of the literature. Am J Orthop (Belle Mead NJ). 1997;26(8):555–60.
14. Coles MJ, Tara Jr HH. Synovial chondromatosis: a case study and brief review. Am J Orthop (Belle Mead NJ). 1997;26(1):37–40.
15. Lim SJ, et al. Operative treatment of primary synovial osteochondromatosis of the hip. J Bone Joint Surg Am. 2006;88(11):2456–64.
16. Boyer T, Dorfmann H. Arthroscopy in primary synovial chondromatosis of the hip: description and outcome of treatment. J Bone Joint Surg Br. 2008;90(3):314–8.
17. Witwity T, Uhlmann RD, Fischer J. Arthroscopic management of chondromatosis of the hip joint. Arthroscopy. 1988;4(1):55–6.
18. Krebs VE. The role of hip arthroscopy in the treatment of synovial disorders and loose bodies. Clin Orthop Relat Res. 2003;406:48–59.
19. Lee JB, et al. Arthroscopic treatment of synovial chondromatosis of the hip. Am J Sports Med. 2012;40(6):1412–8.
20. Hardacker J, Mindell ER. Synovial chondromatosis with secondary subluxation of the hip. A case report. J Bone Joint Surg Am. 1991;73(9):1405–7.
21. Sakellariou A, Johnstone DJ, Pringle JA. Synovial chondromatosis presenting as a pathological fracture

of the femoral neck in an 89-year-old woman–a case report. Acta Orthop Scand. 1998;69(6):640–1.
22. Campanacci DA, et al. Synovial chondrosarcoma of the hip: report of two cases and literature review. Chir Organi Mov. 2008;92(3):139–44.
23. Ackerman D, et al. Results of total hip and total knee arthroplasties in patients with synovial chondromatosis. J Arthroplasty. 2008;23(3):395–400.
24. Ligato A, Nelson S, Bengs BC. Hip resurfacing as treatment for synovial chondromatosis. Orthopedics. 2010;33(3).
25. Jaffe HL, Lichtenstein L, Sutro CJ. Pigmented villonodular synovitis, bursitis and tenosynovitis: a discussion of the synovial and bursal equivalents of the tenosynovial lesion commonly denoted as xanthoma, xanthogranuloma, giant cell tumor, or myeloplaxoma of the tendon sheath, with some considerations of this tendon sheath lesion itself. Arch Pathol. 1941;31:731–65.
26. Ma X, et al. Pigmented villonodular synovitis: a retrospective study of seventy five cases (eighty one joints). Int Orthop. 2013;37(6):1165–70.
27. Choong PF, et al. Pigmented villonodular synovitis. Monoclonality and metastasis–a case for neoplastic origin? Acta Orthop Scand. 1995;66(1):64–8.
28. Sciot R, et al. Analysis of 35 cases of localized and diffuse tenosynovial giant cell tumor: a report from the Chromosomes and Morphology (CHAMP) study group. Mod Pathol. 1999;12(6):576–9.
29. Abdul-Karim FW, et al. Diffuse and localized tenosynovial giant cell tumor and pigmented villonodular synovitis: a clinicopathologic and flow cytometric DNA analysis. Hum Pathol. 1992;23(7): 729–35.
30. Mankin H, Trahan C, Hornicek F. Pigmented villonodular synovitis of joints. J Surg Oncol. 2011;103(5):386–9.
31. Masih S, Antebi A. Imaging of pigmented villonodular synovitis. Semin Musculoskelet Radiol. 2003;7(3): 205–16.
32. Dorwart RH, et al. Pigmented villonodular synovitis of synovial joints: clinical, pathologic, and radiologic features. AJR Am J Roentgenol. 1984;143(4):877–85.
33. Steinbach LS, et al. MRI of the knee in diffuse pigmented villonodular synovitis. Clin Imaging. 1989;13(4):305–16.
34. Amber IB, Clark BJ, Greene GS. Pigmented villonodular synovitis: dedicated PET imaging findings. BMJ Case Rep. 2013
35. Vastel L, et al. Surgical treatment of pigmented villonodular synovitis of the hip. J Bone Joint Surg Am. 2005;87(5):1019–24.
36. Chin KR, et al. Treatment of advanced primary and recurrent diffuse pigmented villonodular synovitis of the knee. J Bone Joint Surg Am. 2002;84-A(12): 2192–202.
37. Shabat S, et al. The use of surgery and yttrium 90 in the management of extensive and diffuse pigmented villonodular synovitis of large joints. Rheumatology (Oxford). 2002;41(10):1113–8.
38. O'Sullivan B, et al. Outcome following radiation treatment for high-risk pigmented villonodular synovitis. Int J Radiat Oncol Biol Phys. 1995;32(3):777–86.
39. Gonzalez Della Valle A, et al. Pigmented villonodular synovitis of the hip: 2- to 23-year followup study. Clin Orthop Relat Res. 2001;388:187–99.
40. Hoberg M, Amstutz HC. Metal-on-metal hip resurfacing in patients with pigmented villonodular synovitis: a report of two cases. Orthopedics. 2010;33(1):50.

Surgical Technique: Arthroscopic Treatment of Synovial Chondromatosis

99

Anthony Marchie, Philip J. Glassner, Ian Panuncialman, and Joseph C. McCarthy

Contents

Abstract

Synovial chondromatosis of the hip is often underdiagnosed. A high index of suspicion and good-quality imaging studies are therefore essential. A wide array of long and curved instrumentation is needed at surgery. Efficient use of time and an organized surgical approach are critical to decreasing joint distraction time and potential complications. A successful outcome, with pain relief, return of joint function and range of motion are predicated on incremental follow-up.

Introduction

Arthroscopic intervention of the hip has been reported for loose bodies, synovial plicae, synovial chondromatosis, pigmented villonodular synovitis, as well as rheumatoid and septic arthritis [1–6]. Unlike standard radiologic imaging, hip arthroscopy allows the surgeon to inspect, biopsy, and treat within one procedure. And unlike arthrotomy, hip arthroscopy avoids the risks of an extensive surgical exposure with prolonged rehabilitation and risk of osteonecrosis.

Synovial chondromatosis is a rare condition in which foci of cartilage develop in the synovial membrane of joints, bursae, or tendon sheaths as a result of metaplasia of the subsynovial connective tissue (Fig. 1). Extracapsular involvement may occur when the loose bodies penetrate out of the joint to adjacent structures, such as tendons

A. Marchie
Mackenzie Health, Richmond Hill, ON, Canada
e-mail: anthony.marchie@mackenziehealth.ca; anthony.marchie@gmail.com

P.J. Glassner
Hunterdon Orthopedic Institute, Flemington, NJ, USA
e-mail: philip.glassner@gmail.com

I. Panuncialman
Harris Orthopaedic Laboratory, Massachusetts General Hospital, Boston, MA, USA
e-mail: ipanuncialman@yahoo.com

J.C. McCarthy (✉)
Harris Orthopaedic Laboratory, Massachusetts General Hospital, Boston, MA, USA

Joint Reconstruction Center, Newton–Wellesley Hospital, Boston, MA, USA
e-mail: jcmccarthy1@partners.org

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0_108

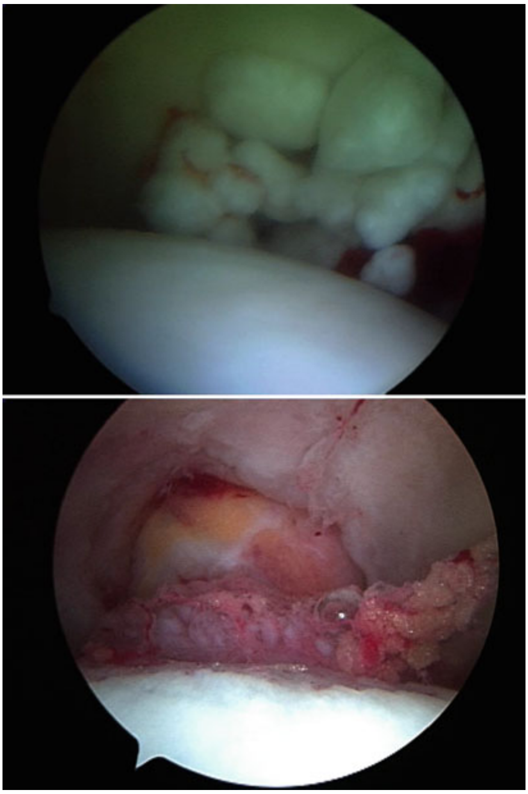


Fig. 1 Synovial chondromatosis, a condition of synovial metaplasia with four or more osteochondral loose bodies

Fig. 2 Radiographic and magnetic resonance imaging showing loose bodies in the hip joint

and bursa [7]. Subsequently, calcification and ossification are commonly seen, and multiple cartilaginous loose bodies are found when the metaplastic foci become pedunculated and detached [8].

These loose bodies, which are either ossified or non-ossified, can become trapped in the acetabular fossa and gutter and cause pain (Fig. 2). The number within each joint may range from a handful to hundreds, and their removal may consequently be a challenge. Nevertheless, an organized and efficient approach that optimizes access, visualization, adequate joint distraction, and appropriate instrumentation for addressing central and peripheral compartment lesions will minimize potential difficulties. Recent improvements in technique and instrumentation have made hip arthroscopy an effective way to diagnose and treat synovial chondromatosis [9] and a variety of other intra-articular problems. Many hip disorders that are now managed arthroscopically previously were undetected and therefore untreated.

Technical Pearls

Arthroscopy can be done supine or lateral; and the latter is preferred by the senior author. With the patient in the lateral decubitus position, a dedicated hip distractor (Innomed Corp, Savannah, GA) is applied to the well-padded leg and ankle (Fig. 3). A regular fluoroscopic table is necessary. The patient must be carefully padded at the perineum to avoid neuropraxic injury.

Fig. 3 A dedicated hip distractor with the patient in the lateral decubitus position (Innomed, Savannah, GA)

Position and Joint Distraction

The femur is placed in slight flexion (approximately 10–20°), with the foot in neutral or slight external rotation. Positioning the hip greater than 20° can translate the sciatic nerve anteriorly, bringing it into danger with the inserting trocar. Excessive external rotation of the femur will also move the greater trochanter posteriorly, making it more likely to deflect the inserting trocar toward the sciatic nerve. The well-padded perineal post is perpendicular to the long axis of the thigh, 10–15 cm distal to the ischial tuberosity. This distal placement of the post avoids pressure on the pudendal nerve and allows a cantilever effect on the proximal femur when traction is applied to lift the femoral head up from the medial wall of the acetabulum and over the transverse acetabular ligament [10]. After skeletal muscle relaxation but prior to skin preparation, fluoroscopy is used to determine the degree of distraction required. Axial distraction is applied with the leg abducted, usually between 0° and 20°, depending on the patient's neck-shaft angle and the depth of the acetabulum. Adequate visualization and cannula insertion require the femoral head to be distracted at least 7–10 mm between the articular surfaces. If distraction is suboptimal, visualizing and maneuvering of instruments within the joint will be challenging, and unexpected loss of traction can further lead to articular damage and instrument breakage. Traction should be less than 1 h at a time for the central compartment. Peripheral compartment surgery is performed without distraction. The distractor is subsequently relaxed after adequate distraction has been confirmed via fluoroscopy. Standard skin preparation and draping would follow.

Once the spinal needles are inserted into the joint, and joint fluid is aspirated to confirm proper location. A blunt, conically tipped trocar is then inserted for controlled penetration of the hip capsule to create an arthroscopic portal. Portal placement requires palpation, identification, and marking of the anatomic landmarks, especially the femoral neurovascular bundle. The procedure may require two or more portals: direct anterior, anterior paratrochanteric or anterolateral, proximal trochanteric, superior paratrochanteric or posterolateral, and direct posterior have all been well described [11]. The superior paratrochanteric portals pass through fewer muscle planes and minimize chance of injury to the lateral femoral cutaneous nerve; these portals also allow the trocars to puncture the superior hip capsule which is thinner. The senior author prefers the anterior and superior paratrochanteric portals initially [12], as they allow visualization of the entire articular

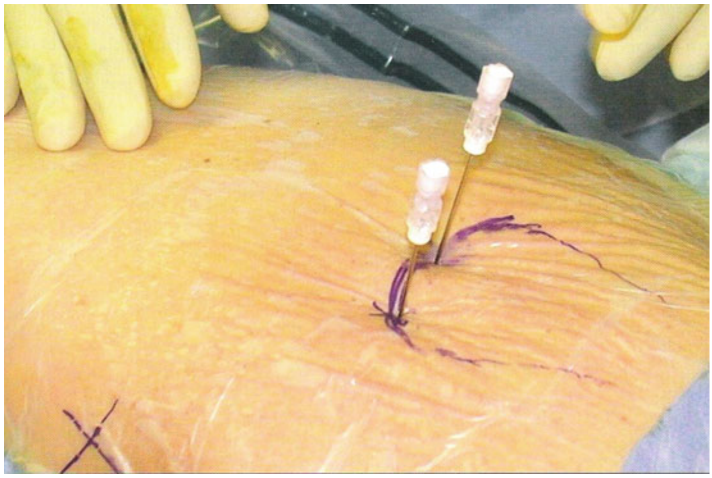


Fig. 4 The senior author's (JCM) preferred arthroscopic portals: anterior superior paratrochanteric portal, which gives excellent visualization of the femoral head, anterior neck, anterior labrum, and synovial tissues beneath the zona orbicularis, and the posterior superior paratrochanteric portal, used for viewing the posterior capsule, posterior labrum, and posterior femoral head

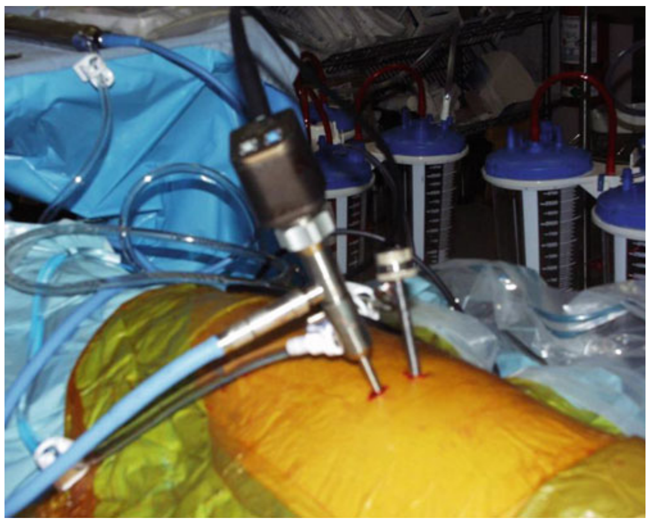


Fig. 5 The senior author's (JCM) preferred arthroscopic portal sites with the patient in the lateral decubitus position

portion of the joint in 95 % of cases (Figs. 4 and 5). After the portals are established, the hip is distended with normal saline to overcome the joint's native intra-articular negative pressure. Pressure-sensitive high-volume lavage is required for optimal visualization.

The Hip Joint

The hip joint has both intra-articular (or central) and peripheral compartments. Hip pathology is often found within the intra-articular region. The peripheral compartment, which is intracapsular but extra-articular, may also contain lesions which are overlooked with traction alone because synovial disease frequently involves the capsule, and loose bodies may therefore sequester in the peripheral recesses.

Central Compartment

Loose bodies tend to aggregate in the fossa of the central compartment (Fig. 6) and may be encased within the pulvinar soft tissue. The femoral head's

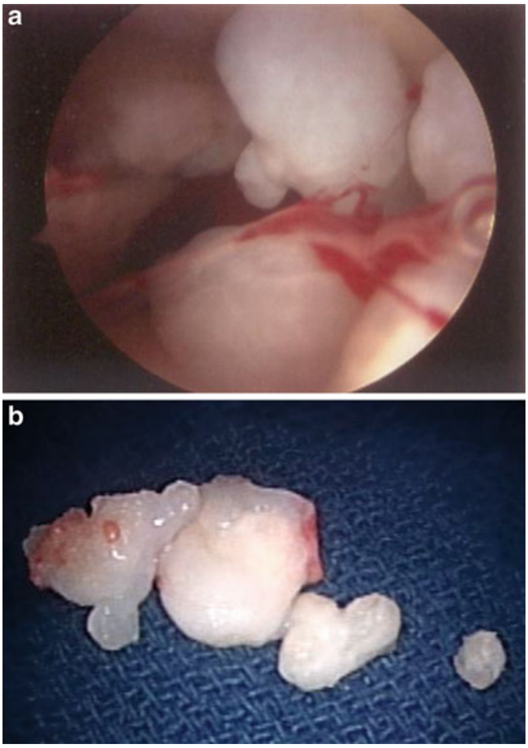


Fig. 6 (**a**) An intraoperative view of the loose bodies prior to removal; (**b**) post-loose body removal

arc of curvature also presents a challenge to access of the joint, particularly with straight tools. Adequate distraction and curved instrumentation, inserted via half-pipe and telescoping cannulas (Arthrex®, Naples FL), are therefore essential for successful removal of loose bodies. Loose bodies may also be trapped in the gutters, within the labro-capsular recesses. Although most of the joint can be seen with a standard 30° arthroscope, the 70° arthroscope and an accessory portal are useful in such cases. An accompanying synovectomy with an arthroscopic shaver and/or electrothermal device would also be critical to help minimize the chance of recurrence.

Peripheral Compartment

Once the central compartment has been addressed, then peripheral compartment lesions can be accessed by releasing traction and flexing the hip from 20 to 40°. The 70° arthroscope is essential for visualization here, and an additional third portal to improve access and permit a partial capsulectomy with the electrothermal device or "banana" knife may be needed. Large, well-fixed bodies may be left alone provided they do not interfere with joint articulation.

The Difficult Loose Body Removal

The depth of the hip joint requires specially designed and lengthened arthroscopic instruments that can pass through the cannulae, protecting the soft tissue structures surrounding the hip. Extra-long, curved shaver blades with either convex or concave surfaces allow operative arthroscopy around the femoral head. Long suction punches and hand tools, such as alligator graspers, both straight and curved, are needed for resection and aspiration of soft tissue and loose bodies. Large loose bodies may require morsellization, displacement, and telescoping cannulae for their removal. A half-pipe cannula may sometimes be needed to introduce curved instruments and improve ease of passage as large loose bodies are extracted out of the portal. A partial synovectomy can be carried out with either straight or curved extra-long shavers. Flexible electrothermal devices which have precise control of temperature and coagulation are useful for debriding torn labral and chondral flaps or inflamed synovial tissue folds. White zone labral and chondral lesions should be judiciously debrided or resected back to a stable base and healthy tissue while preserving the capsular labrum. Extra-articular loose bodies that are found within the psoas bursa, for example, may require combined arthroscopic and mini-open approaches for access and removal.

Conclusion

Hip arthroscopy plays an important role in the early detection and treatment of synovial chondromatosis, a condition which can be challenging to diagnose via conventional methods. The presence of loose bodies found in synovial chondromatosis can cause and accelerate irreversible damage to the chondral surface and labrum.

In the senior author's experience with 30 cases thus far, representing the largest North American series, arthroscopic treatment permitted a definitive diagnosis, removal of 5 to 300 loose bodies (particularly those clustered within the fossa), treatment of chondral damage, and synovectomy. Though there was a disease recurrence rate of about 10 % despite intervention, arthroscopy can be repeated and was beneficial to patients with mild to moderate degenerative changes.

Complications have been previously reported to occur in 0.5–5 % of patients and are most often related to the distraction of the joint. Transient neuropraxia is the most common injury [13, 14]. Damage to the labrum on entry into the joint or scuffing of the femoral head can also be avoided by using an image intensifier to confirm adequate distraction.

As there is a steep learning curve with the technique, in addition to the challenge of maneuvering in a deep joint, proper patient positioning and adequate hip distraction are important for a successful procedure. The use of adequate portals, saline fluid dynamics, and tapered telescoping cannulae helps to additionally avoid instrument breakage or scuffing of articular surfaces. Electrothermal devices, long curved shavers, and long graspers are necessary to reach formerly inaccessible areas. And generous padding of the perineum, leg, and ankle, combined with minimal distraction time, will help prevent neurovascular complication.

Specific long-term prospective outcome data for arthroscopic hip surgery are still needed to refine its role in orthopedic practice. A validated, self-administered questionnaire assessing non-arthritic hip pain in patients with high activity demands and expectations can be used prior to intervention and after treatment [15].

Summary

- Synovial chondromatosis of the hip is often underdiagnosed. A high index of suspicion and good-quality imaging studies are therefore essential.
- A wide array of long and curved instrumentation is needed at surgery.
- Efficient use of time and an organized surgical approach are critical to decreasing joint distraction time and potential complications.
- A successful outcome, with pain relief and return of joint function and range of motion, is predicated on incremental follow-up.

References

1. Dkhissi M, Aboutaieb R, Hermas M, Zryouil B. Villonodular synovitis of the large joints. Acta Orthop Belg. 1993;59(1):81–6.
2. Gitelis S, Heligman D, Morton T. The treatment of pigmented villonodular synovitis of the hip. A case report and literature review. Clin Orthop Relat Res. 1989;239:154–60.
3. Holgersson S, Brattström H, Mogensen B, Lidgren L. Arthroscopy of the hip in juvenile chronic arthritis. J Pediatr Orthop. 1981;1(3):273–8.
4. Janssens X, Van Meirhaeghe J, Verdonk R, Verjans P, Cuvelier C, Veys EM. Diagnostic arthroscopy of the hip joint in pigmented villonodular synovitis. Arthroscopy. 1987;3(4):283–7.
5. Ochi T, Iwase R, Kimura T, Hirooka A, Masada K, Owaki H, Wakitani S, Murata N, Ono K. Effect of early synovectomy on the course of rheumatoid arthritis. J Rheumatol. 1991;18(12):1794–8.
6. Sim FH. Synovial proliferative disorders: role of synovectomy. Arthroscopy. 1985;1(3):198–204.
7. Hardacker J, Mindell ER. Synovial chondromatosis with secondary subluxation of the hip. A case report. J Bone Joint Surg Am. 1991;73(9):1405–7.
8. Maurice H, Crone M, Watt I. Synovial chondromatosis. J Bone Joint Surg (Br). 1988;70(5):807–11.
9. Boyer T, Dorfmann H. Arthroscopy in primary synovial chondromatosis of the hip: description and outcome of treatment. J Bone Joint Surg (Br). 2008;90(3):314–8.
10. Mason JB, McCarthy JC, O'Donnell J, Barsoum W, Mayor MB, Busconi BD, Krebs VE, Owens BD. Hip arthroscopy: surgical approach, positioning, and distraction. Clin Orthop Relat Res. 2003;406:29–37.
11. McCarthy JC. Hip arthroscopy: applications and technique. J Am Acad Orthop Surg. 1995;3(3):115–22.
12. Glick JM. Hip arthroscopy by the lateral approach. Instr Course Lect. 2006;55:317–23.
13. Clarke MT, Arora A, Villar RN. Hip arthroscopy: complications in 1054 cases. Clin Orthop Relat Res. 2003;406:84–8.
14. Griffin DR, Villar RN. Complications of arthroscopy of the hip. J Bone Joint Surg (Br). 1999;81(4):604–6.
15. Christensen CP, Althausen PL, Mittleman MA, Lee JA, McCarthy JC. The nonarthritic hip score: reliable and validated. Clin Orthop Relat Res. 2003;406:75–83.

Index

S.J. Nho et al. (eds.), *Hip Arthroscopy and Hip Joint Preservation Surgery*,
DOI 10.1007/978-1-4614-6965-0