

Vicente Sanchis-Alfonso
Editor

Atlas of the Patellofemoral Joint

 Springer

Atlas of the Patellofemoral Joint

Vicente Sanchis-Alfonso
Editor

Atlas of the Patellofemoral Joint

 Springer

Editor

Vicente Sanchis-Alfonso
Depto. Cirugía Ortopédica
Hospital Arnau de Vilanova
Valencia
Spain

ISBN 978-1-4471-4494-6 ISBN 978-1-4471-4495-3 (eBook)

DOI 10.1007/978-1-4471-4495-3

Springer London Heidelberg New York Dordrecht

Library of Congress Control Number: 2012955058

Originally published as part of Anterior Knee Pain and Patellar Instability, 2nd Ed (ISBN: 978-0-85729-506-4) in 2011

© Springer-Verlag London 2013

This work is subject to copyright. All rights are reserved by the Publisher, whether the whole or part of the material is concerned, specifically the rights of translation, reprinting, reuse of illustrations, recitation, broadcasting, reproduction on microfilms or in any other physical way, and transmission or information storage and retrieval, electronic adaptation, computer software, or by similar or dissimilar methodology now known or hereafter developed. Exempted from this legal reservation are brief excerpts in connection with reviews or scholarly analysis or material supplied specifically for the purpose of being entered and executed on a computer system, for exclusive use by the purchaser of the work. Duplication of this publication or parts thereof is permitted only under the provisions of the Copyright Law of the Publisher's location, in its current version, and permission for use must always be obtained from Springer. Permissions for use may be obtained through RightsLink at the Copyright Clearance Center. Violations are liable to prosecution under the respective Copyright Law.

The use of general descriptive names, registered names, trademarks, service marks, etc. in this publication does not imply, even in the absence of a specific statement, that such names are exempt from the relevant protective laws and regulations and therefore free for general use.

While the advice and information in this book are believed to be true and accurate at the date of publication, neither the authors nor the editors nor the publisher can accept any legal responsibility for any errors or omissions that may be made. The publisher makes no warranty, express or implied, with respect to the material contained herein.

Printed on acid-free paper

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

Preface

A picture is worth a thousand words, and the goal of this atlas is to better understand patellofemoral conditions through pictures. This is the reason we have decided to publish this book as a spin-off of the original book *Anterior Knee Pain and Patellar Instability*. All the images and summaries are compiled in order to convey a simple understanding of this complex problem. It is designed to be a detailed visual guide, presenting the reader with a wealth of images that will form a crucial aid when faced with surgery of the knee and particularly that of the patella. It is a very practical book, aimed at the general orthopedic surgeon but will also be very useful to those that already specialize in surgery of the knee.

Contents

Part I Etiopathogenic Bases and Therapeutic Implications

1 Background: Patellofemoral Malalignment Versus Tissue Homeostasis	3
Vicente Sanchis-Alfonso	
2 In Search of the Etiology of Anterior Knee Pain	9
Jan Näslund	
3 Biological Causes of Anterior Knee Pain	11
Vicente Sanchis-Alfonso, Esther Roselló-Sastre, Juan Saus-Mas, and Fernando Revert-Ros	
4 Pathogenesis of Anterior Knee Pain in the Active Young: Is There a Relation Between the Presence of Patellofemoral Malalignment and Pain?	21
Vicente Sanchis-Alfonso, Fermín Ordoño, Alfredo Subías-López, and Carmen Monserrat	
5 Unilateral Anterior Knee Pain as a Sign of Knee Asymmetry	25
K. Donald Shelbourne and Emily E. Krodel	
6 Biomechanical Bases for Anterior Knee Pain and Patellar Instability	29
Vicente Sanchis-Alfonso, Carolina Ávila-Carrasco, Jaime M. Prat-Pastor, Carlos M. Atienza, and Enrique Cuñat	
7 Anatomy of Patellar Dislocation	41
Najeeb Khan, Donald C. Fithian, and Eiki Nomura	
8 Evaluation of the Patient with Anterior Knee Pain and Patellar Instability	47
Vicente Sanchis-Alfonso, Erik Montesinos-Berry, Agustín Serrano, and Vicente Martínez-Sanjuan	
9 Influence of Psychological Factors on Pain and Disability in Anterior Knee Pain Patients	63
Julio Doménech, Vicente Sanchis-Alfonso, and Begoña Espejo	

10	Uncommon Causes of Anterior Knee Pain	65
	Vicente Sanchis-Alfonso, Erik Montesinos-Berry, Francisco Aparisi-Rodriguez, and Vicente Belloch-Ugarte	
11	Risk Factors and Prevention of Anterior Knee Pain	83
	Erik Witvrouw, Damien Van Tiggelen, and Tine Willems	
12	Non-operative Treatment of Athletes with Anterior Knee Pain: Science, Classical, and New Ideas	87
	Suzanne Werner	
13	Conservative Management of Anterior Knee Pain: The McConnell Program	97
	Jenny McConnell and Kim Bennell	
14	Skeletal Malalignment and Anterior Knee Pain: Rationale, Diagnosis, and Management	103
	Robert A. Teitge and Roger Torga-Spak	
15	Biochemical Causes of Patellar Tendinopathy?	109
	Patrik Danielson and Alexander Scott	
16	Prevention of Anterior Knee Pain After Anterior Cruciate Ligament Reconstruction	113
	K. Donald Shelbourne, Scott E. Urch, and Heather Freeman	
17	Anterior Knee Pain with Special Emphasis on the Clinical, Radiographic, Histological, Ultrastructural, and Biochemical Aspects After Anterior Cruciate Ligament Reconstruction Using Autografts	115
	Jüri Kartus, Lars Ejerhed, and Tomas Movin	
Part II Emerging Technologies for Investigating Patellofemoral Joint. Clinical Relevance		
18	Imaging and Musculoskeletal Modeling to Investigate the Mechanical Etiology of Patellofemoral Pain	125
	Thor F. Besier, Christine Draper, Saikat Pal, Michael Fredericson, Garry Gold, Scott Delp, and Gary Beaupré	
19	Computational Models for Investigating the Patellofemoral Joint: Clinical Relevance	135
	John J. Elias and Andrew J. Cosgarea	
20	Kinetic Analysis: A Sensitive Outcome Objective Measurement Method in Evaluating Lateral Patellar Instability	139
	Vicente Sanchis-Alfonso, José María Baydal-Bertomeu, Erik Montesinos-Berry, Andrea Castelli, and José David Garrido-Jaén	

21 Kinetic and Kinematic Analysis in Evaluating Patients with Anterior Knee Pain	149
Vicente Sanchis-Alfonso, Susana Marín-Roca, Erik Montesinos-Berry, José María Baydal-Bertomeu, and María Francisca Peydro-De Moya	
Part III Clinical Cases Commented	
22 Failure of Patellofemoral Surgery: Analysis of Clinical Cases	161
Robert A. Teitge and Roger Torga-Spak	
23 Neuromatous Knee Pain: Evaluation and Management	167
Maurice Y. Nahabedian	
24 Kinetic and Kinematic Analysis of Iatrogenic Medial Patellar Instability: Clinical Relevance	171
Vicente Sanchis-Alfonso, Erik Montesinos-Berry, Andrea Castelli, Susana Marín-Roca, and Alex Cortes	
Part IV Surgical Techniques “How I Do It”	
25 Ultrasound and Doppler-Guided Arthroscopic Shaving for the Treatment of Patellar Tendinopathy/Jumper’s Knee: Biological Background and Description of Method	181
Håkan Alfredson, Lotta Willberg, Lars Öhberg, and Sture Forsgren	
26 Arthroscopic Patellar Denervation for Anterior Knee Pain	183
Jordi Vega, Pau Golanó, and Vicente Sanchis-Alfonso	
27 Reconstruction of the Medial Patellofemoral Ligament: How I Do It	187
Eric W. Edmonds and Donald C. Fithian	
28 Reconstruction of the Medial Patellofemoral Ligament: How I Do It	191
Robert A. Teitge and Roger Torga-Spak	
29 MPFL Reconstruction: Principles and Complications	193
Pieter J. Erasmus and Mathieu Thauinat	
30 Reconstruction of the Lateral Patellofemoral Ligament: How I Do It	197
Jack T. Andrish	
31 Reconstruction of the Lateral Patellofemoral Ligament: How I Do It	201
Robert A. Teitge and Roger Torga-Spak	

32 Osteotomies Around the Patellofemoral Joint	203
Roland M. Biedert and Philippe M. Tscholl	
33 Sulcus Deepening Trochleoplasty	211
David Dejour and Paulo Renato F. Saggin	
34 Rotational Tibial Osteotomy	217
Robert A. Teitge and Roger Torga-Spak	
35 Rotational Femoral Osteotomy	221
Robert A. Teitge and Roger Torga-Spak	
36 Patella Infera and Patella Alta	223
Paulo Renato F. Saggin and David Dejour	
37 Anteromedial Tibial Tubercle Osteotomy (Fulkerson Osteotomy)	229
Jack Farr, Brian J. Cole, James Kercher, Lachlan Batty, and Sarvottam Bajaj	
38 The Patella Thinning Osteotomy: A New Technique for Patellofemoral Arthritis	233
Javier Vaquero and José Antonio Calvo	
39 Cartilage Restoration in the Patellofemoral Joint	239
Jack Farr, Brian J. Cole, Michael J. Salata, Marco Collarile, and Sarvottam Bajaj	
40 Autologous Osteochondral Mosaicplasty	241
László Hangody and Eszter Baló	
41 Patellofemoral Allografts	245
Robert A. Teitge and Roger Torga-Spak	
42 Patellofemoral Arthroplasty: Pearls and Pitfalls	247
Ronald P. Grelsamer and Jason Gould	
43 The Patellofemoral Arthroplasty: The Great Solution, the Great Problem	249
Vicente Sanchis-Alfonso	
44 A Philosophy of the Patellofemoral Joint: A Logical Clinical Approach	259
Alan C. Merchant	
Index	263

Contributors

Håkan Alfredson M.D., Ph.D. Sports Medicine Unit, Department of Surgical and Perioperative Sciences, Umeå University, Umeå, Sweden

Jack T. Andrish, M.D. Department of Orthopaedic Surgery, Center of Sports Health, Cleveland Clinic, Cleveland, OH, USA

Francisco Aparisi, M.D., Ph.D. Department of Radiology, Hospital Universitario La Fe, Valencia, Spain

Carlos M. Atienza, Mech. Eng., Ph.D. Instituto de Biomecánica de Valencia (IBV), Universidad Politécnica de Valencia, Grupo de Tecnología Sanitaria (GTS-IBV), CIBER-BBN, Valencia, Spain

Carolina Ávila-Carrasco, Mech. Eng. Instituto de Biomecánica de Valencia (IBV), Universidad Politécnica de Valencia, Grupo de Tecnología Sanitaria (GTS-IBV), CIBER-BBN, Valencia, Spain

Sarvottam Bajaj, B.E. Division of Sports Medicine, Rush University Medical Center, Chicago, IL, USA

Eszter Baló, M.D. Orthopaedic and Trauma Department, Uzsoki Hospital, Budapest, Hungary

Lachlan Batty, M.B.B.S., B.MedSc. The Alfred Hospital, Melbourne, VIC, Australia

José María Baydal-Bertomeu, Mech. Eng. Instituto de Biomecánica de Valencia (IBV), Universidad Politécnica de Valencia, Valencia, Spain

Gary Beaupré, Ph.D. Research Career Scientist, VA Rehabilitation Research and Development Center, Palo Alto, CA, USA

Vicente Belloch-Ugarte Clinica ERESA, Hospital Universitario La Fe, Valencia, Spain

Kim Bennell, Ph.D. Centre for Health, Exercise and Sports Medicine Physiotherapy, School of Health Sciences, The University of Melbourne, Melbourne, VIC, Australia

Thor F. Besier, Ph.D. Department of Orthopaedics, Stanford University, Stanford, CA, USA

Roland M. Biedert, M.D. University of Basel, Biel, Switzerland

Sportclinic Villa Linde, Swiss Olympic Medical Center Magglingen-Biel,
Biel, Switzerland

José Antonio Calvo, M.D., Ph.D. Hospital General Universitario Gregorio
Marañón, Madrid, Spain

Andrea Castelli, Biomed. Eng. Instituto de Biomecánica de Valencia
(IBV), Universidad Politécnica de Valencia, Valencia, Spain

Brian J. Cole, M.D., M.B.A. Division of Sports Medicine, Departments of
Orthopaedics & Anatomy and Cell Biology, Cartilage Restoration Center at
Rush, Rush University Medical Center, Chicago, IL, USA

Marco Collarile, M.D. Orthopaedic Department and Knee Surgery Center,
Sacro Cuore Don Calabria Hospital, Negrar, Verona, Italy

Alex Cortés, M.D., Ph.D. Instituto de Biomecánica de Valencia (IBV),
Universidad Politécnica de Valencia, Valencia, Spain

Andrew J. Cosgarea, M.D. Department of Orthopaedic Surgery, Johns
Hopkins University, Baltimore, MD, USA

Enrique Cuñat E. Cuñat Physiotherapy, Valencia, Spain

Patrik Danielson, M.D., Ph.D. Section for Anatomy, Department of
Integrative Medical Biology, Faculty of Medicine, Umeå University, Umeå,
Sweden

David Dejour, M.D. Lyon Ortho Clinic, Lyon, France

Scott Delp, Ph.D. Department of Bioengineering, Stanford University,
Stanford, CA, USA

Julio Domenech, M.D., Ph.D. Associate Professor of Orthopaedic Surgery,
Facultad de Ciencias de la Salud, Universidad Cardenal Herrera – CEU,
Valencia, Spain

Christine Draper, Ph.D. Department of Bioengineering,
Stanford University, Stanford, CA, USA

Eric W. Edmonds, M.D. Department of Orthopaedic Surgery,
University of California, San Diego, San Diego, CA, USA

Sports Medicine Program, Pediatric Orthopaedic and Scoliosis Center,
Rady Children's Hospital San Diego, San Diego, CA, USA

John J. Elias, Ph.D. Akron General Medical Center, Calhoun Research
Lab, Akron, OH, USA

Lars Ejerhed, M.D., Ph.D. Orthopaedic Department, University of
Gothenburg, NU-Hospital Organization, Trollhättan/Uddevalla, Sweden

Pieter J. Erasmus, M.D. University of Stellenbosch, Mediclinic,
Die Boord, Stellenbosch, South Africa

Begoña Espejo Department of Methodology of the Behavioural Sciences, University of Valencia, Valencia, Spain

Jack Farr, M.D. Voluntary Clinical, Orthopaedic Surgery, IU School of Medicine, OrthoIndy Knee Care Institute, Cartilage Restoration Center of Indiana, Indianapolis, IN, USA

Donald C. Fithian, M.D. Southern California Permanente Medical Group, El Cajon, CA, USA

Sture Forsgren, M.D., Ph.D. Department of Integrative Medical Biology, Anatomy, Umeå University, Umeå, Sweden

Michael Fredericson, M.D. Department of Orthopaedics, Stanford University, Stanford, CA, USA

Heather Freeman, PT, DHS Shelbourne Knee Center, Methodist Hospital, Indianapolis, IN, USA

José David Garrido-Jaén, Mech. Eng. Instituto de Biomecánica de Valencia (IBV), Universidad Politécnica de Valencia, Valencia, Spain

Pau Golanó Laboratory of Arthroscopic and Surgical Anatomy, Department of Pathology and Experimental Therapeutics, Human Anatomy Unit, Faculty of Medicine, University of Barcelona, Barcelona, Spain

Garry Gold, M.S., M.D. Department of Radiology, Stanford University, Stanford, CA, USA

Jason Gould, M.D. Department of Orthopedic Surgery, Mount Sinai Medical Center, New York, NY, USA

Ronald P. Grelsamer, M.D. Patellofemoral Reconstruction, Mount Sinai Medical Center, New York, NY, USA

László Hangody, M.D., Ph.D., D.Sc. Department of Orthopaedics, Uzsoki Hospital, Budapest, Hungary

Jüri Kartus, M.D., Ph.D. Orthopaedic Department, University of Gothenburg, NU-Hospital Organization, Trollhättan/Uddevalla, Sweden

James Kercher, M.D. Division of Sports Medicine, Rush University Medical Center, Chicago, IL, USA

Najeeb Khan, M.D. Southern California Permanente Medical Group, San Marcos, CA, USA

Emily E. Krodel, M.D. Indiana University School of Medicine, Indianapolis, IN, USA

Susana Marín-Roca, Mech. Eng. Instituto de Biomecánica de Valencia (IBV), Universidad Politécnica de Valencia, Valencia, Spain

Vicente Martínez-Sanjuan, M.D., Ph.D. MR and CT Unit, ERESA-Hospital General Universitario, Valencia, Spain

Jenny McConnell, BAppSci (Phty), Grad Dip Man Ther, M Biomed Eng. McConnell & Clements Physiotherapy, Sydney, Australia

Alan C. Merchant, M.D. Department of Orthopedic Surgery, Stanford University School of Medicine, Palo Alto, CA, USA

Department of Orthopedic Surgery, El Camino Hospital, Mountain View, CA, USA

Carmen Monserrat Department of Radiology, Hospital Arnau de Vilanova, Valencia, Spain

Erik Montesinos-Berry, M.D. Hospital de Manises, Valencia, Spain

Tomas Movin, M.D., Ph.D. Emergency Department, Karolinska University Hospital, Karolinska Institutet, Stockholm, Sweden

Maurice Y. Nahabedian, M.D., FACS Department of Plastic Surgery, Georgetown University, Georgetown, Washington DC, USA

Jan Näslund, Ph.D. Department of Physiology and Pharmacology, Karolinska Institutet, Stockholm, Sweden

Eiki Nomura, M.D. Department of Orthopaedic Surgery, International Goodwill Hospital, Yokohama, Japan

Fermín Ordoño, M.D., Ph.D. Department of Neurophysiology, Hospital Arnau de Vilanova, Valencia, Spain

Lars Öhberg, M.D., Ph.D. Department of Radiation Sciences, Diagnostic Radiology, Umeå, Sweden

Saikat Pal, Ph.D. Department of Bioengineering, Stanford University, Stanford, CA, USA

María Francisca Peydro-De Moya, M.D., Ph.D. Instituto de Biomecánica de Valencia (IBV), Universidad Politécnica de Valencia, Valencia, Spain

Jaime M. Prat, M.D., Ph.D. RTD Area, Instituto de Biomecánica de Valencia (IBV), Universidad Politécnica de Valencia, Grupo de Tecnología Sanitaria (GTS-IBV), CIBER-BBN, Valencia, Spain

Fernando Revert-Ros Centro de Investigación Príncipe Felipe, Biomedicina, Valencia, Spain

Esther Roselló-Sastre, M.D., Ph.D. Department of Pathology, Hospital Universitario Dr. Peset, Valencia, Spain

Paulo Renato Saggin, M.D. Instituto de Ortopedia e Traumatologia (I.O.T.), Passo Fundo, Brazil

Michael J Salata, M.D. Division of Sports Medicine, Rush University Medical Center, Chicago, IL, USA

Vicente Sanchis-Alfonso, M.D., Ph.D. International Patellofemoral Study Group, ACL Study Group, Hospital 9 de Octubre, Hospital Arnau de Vilanova, School of Medicine, Valencia Catholic University, Valencia, Spain

Juan Saus-Mas Centro de Investigación Príncipe Felipe, Biomedicina, Valencia, Spain

Alexander Scott, B.Sc (PT), Ph.D. Department of Physical Therapy, Faculty of Medicine, University of British Columbia, Vancouver, BC, Canada

K. Donald Shelbourne, M.D. Shelbourne Knee Center at Methodist Hospital, Indiana University School of Medicine, Indianapolis, IN, USA

Agustín Serrano, M.D. Hospital de Manises, Valencia, Spain

Alfredo Subías-López, M.D. Hospital General de Almansa, Albacete, Spain

Robert A. Teitge, M.D. Department of Orthopaedics, Wayne State University School of Medicine, Detroit, MI, USA

Mathieu Thauinat, M.D. Department of Orthopedic Surgery, Hopital André Mignot, Le Chesnay, France

Roger Torga-Spak, M.D. Department of Surgery, Faculty of Orthopaedics and Traumatology, Instituto Universitario CEMIC, Buenos Aires, Argentina

Philippe M. Tscholl, M.D. Sportclinic Villa Linde, Swiss Olympic Medical Center Magglingen-Biel, Biel, Switzerland

Scott E. Urch, M.D. Shelbourne Knee Center at Methodist Hospital, Indiana University School of Medicine, Indianapolis, IN, USA

Damien Van Tiggelen, PT Department of Rehabilitation Sciences and Physical Therapy, Faculty of Medicine, Ghent University of Ghent, Ghent, Belgium

Department of Traumatology and Rehabilitation, Military Hospital Queen Astrid, Brussels, Belgium

Javier Vaquero, M.D., Ph.D. Universidad Complutense de Madrid, Hospital General Universitario Gregorio Marañón, Madrid, Spain

Jordi Vega Department of Orthopedic and Trauma Surgery, Hospital Asepeyo Sant Cugat, Sant Cugat del Vallés, Barcelona, Spain

Lotta Willberg, M.D. Capio Arthro Clinic, Karolinska Institute, Stockholm, Sweden

Suzanne Werner, Ph.D. Stockholm Sports Trauma Research Center, Karolinska institutet, Stockholm, Sweden
Capio Arthro Clinic, Stockholm, Sweden

Tine Willems Department of Rehabilitation Sciences and Physical Therapy, Faculty of Medicine, University of Ghent, Ghent, Belgium

Erik Witvrouw, Ph.D. Department of Rehabilitation Sciences and Physical Therapy, Faculty of Medicine, Ghent University of Ghent, Ghent, Belgium

Part I

**Etiopathogenic Bases
and Therapeutic Implications**

Background: Patellofemoral Malalignment Versus Tissue Homeostasis

1

Myths and Truths About Patellofemoral Disease

Vicente Sanchis-Alfonso

The pathology we discuss in the present monograph presents itself with a multifactorial etiology and a great pathogenic, diagnostic and therapeutic complexity.

The consideration of anterior knee pain to be a self-limited condition in patients with an underlying neurotic personality should be banished from the orthopaedic literature.

Our knowledge about anterior knee pain has evolved throughout the twentieth century. While until the end of the 1960s this pain was attributed to chondromalacia patellae, a concept born at the beginning of the century, after that period it came to be connected with abnormal patellofemoral alignment. More recently, the pain was put down to a wide range of physiopathological processes such as peripatellar synovitis, the incre-

ment in intraosseous pressure, and increased bone remodelling. We are now at a turning point. New information is produced at breakneck speed. Nowadays, medicine in its entirety is being reassessed at subcellular level, and this is precisely the line of thought we are following in the approach to anterior knee pain syndrome. Still to be seen are the implications that this change of mentality will have in the treatment of anterior knee pain syndrome in the future, but I am sure that these new currents of thought will open the doors for us to new and exciting perspectives that could potentially revolutionize the management of this troublesome condition in the new millennium we have just entered. Clearly, we are only at the beginning of the road that will lead to understanding where anterior knee pain comes from.

V. Sanchis-Alfonso, M.D., Ph.D.
International Patellofemoral Study Group,
ACL Study Group, Hospital 9 de Octubre,
Hospital Arnau de Vilanova, School of Medicine,
Valencia Catholic University, Valencia, Spain
e-mail: vicente.sanchis.alfonso@gmail.com

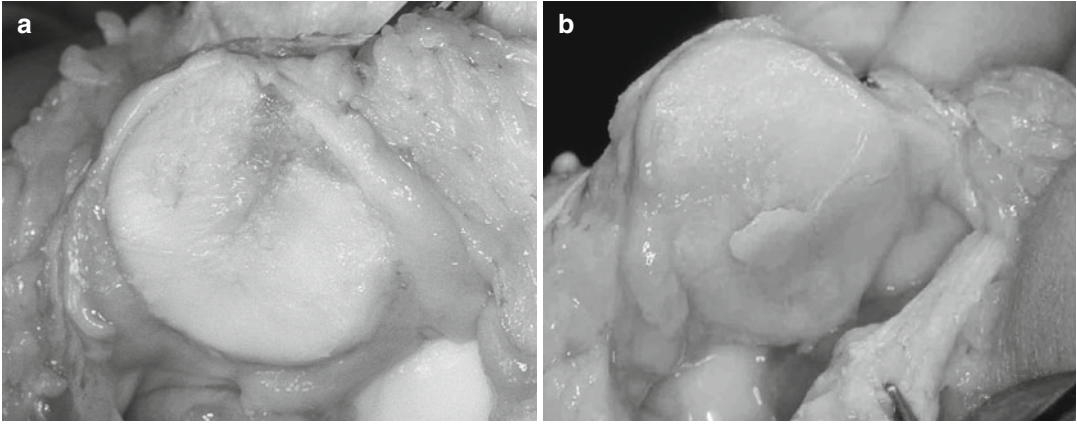


Fig. 1.1 The intensity of preoperative pain is not related to the seriousness or the extension of the chondromalacia patellae found during surgery. The most serious cases of chondromalacia arise in patients with a recurrent patellar

dislocation, who feel little or no pain between their dislocation episodes (a). Chondral lesion of the patella with fragmentation and fissuring of the cartilage in a patient with PFM that consulted for anterior knee pain (b)

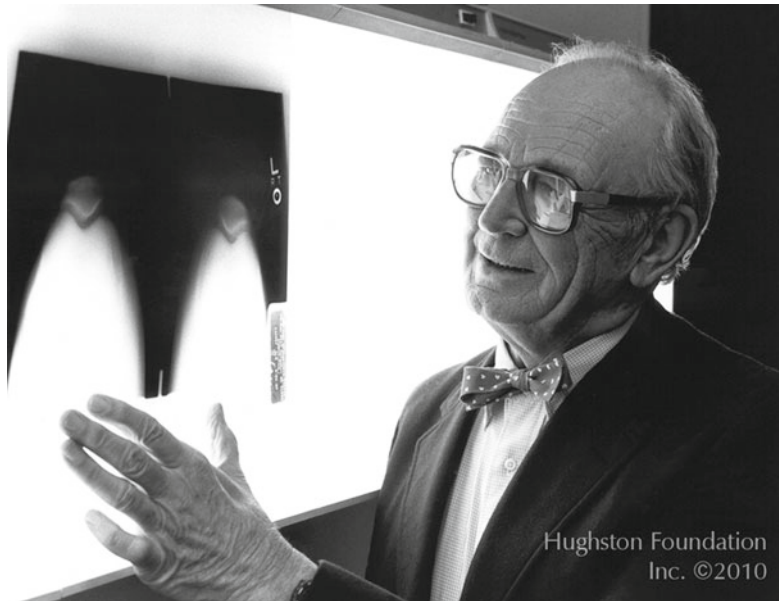


Fig. 1.2 Jack C. Hughston, MD (1917–2004). One of the founding fathers of Sports Medicine (© The Hughston Foundation, Inc)

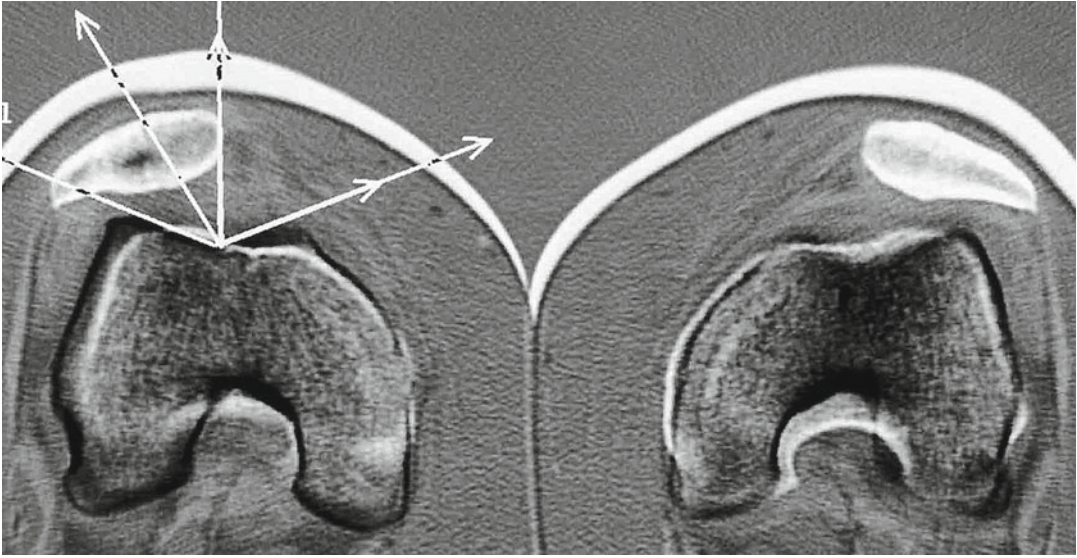


Fig. 1.3 CT at 0° from a patient with anterior knee pain and functional patellofemoral instability in the right knee, however the left knee is completely asymptomatic. In both knees, the PFM is symmetric

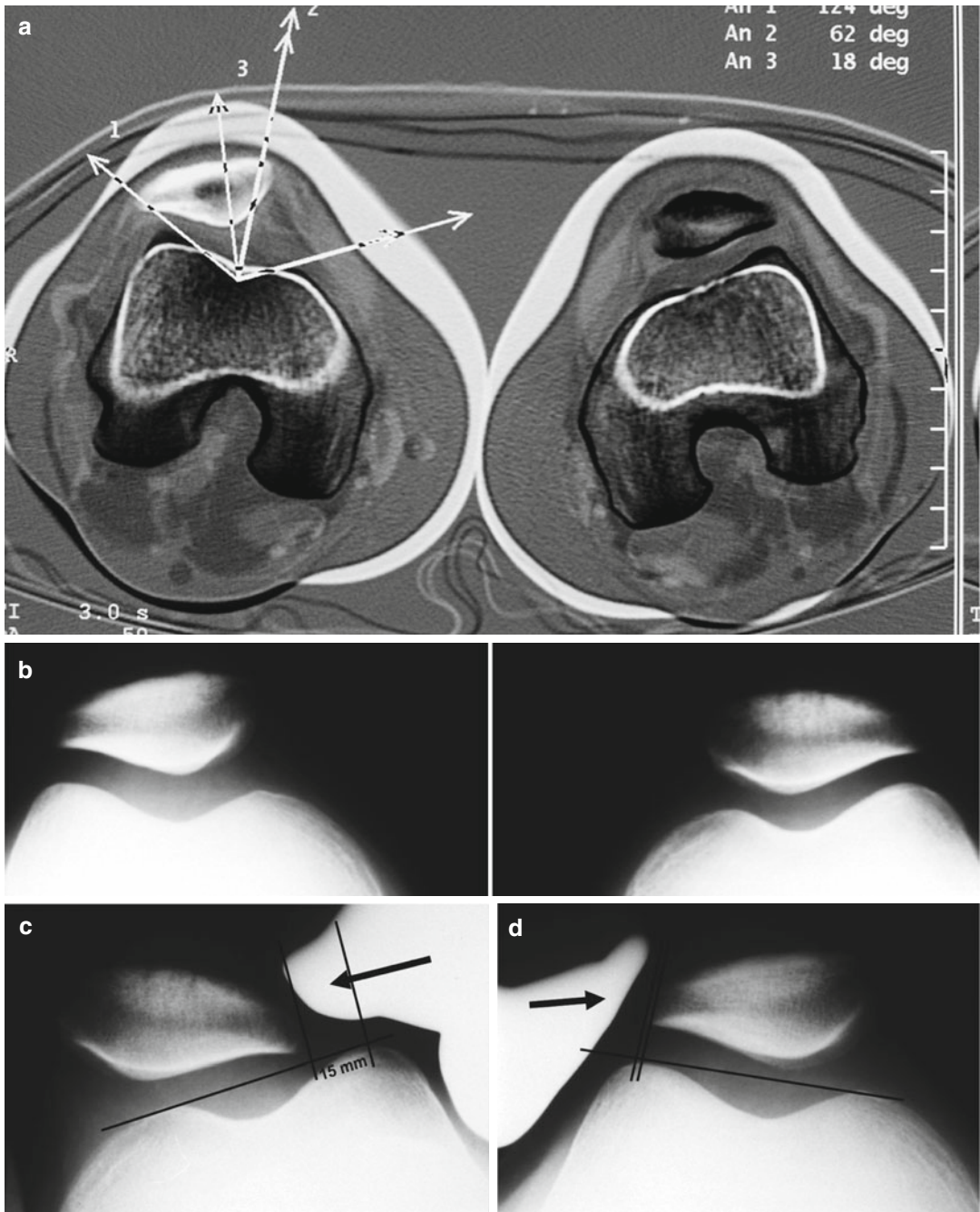


Fig. 1.4 CT at 0° from a patient with severe anterior knee pain and patellofemoral instability in the left knee (a). This knee, which was operated on 2 years ago, performing an Insall's proximal realignment, was very symptomatic in spite of the correct patellofemoral congruence. Fulkerson test for medial subluxation was positive. Nevertheless, the right knee was asymptomatic despite the PFM. Conventional radiographs were normal and the

patella was seen well centered in the axial view of Merchant (b). Axial stress radiograph of the left knee (c) allowed us to detect an iatrogenic medial subluxation of the patella (medial displacement of 15 mm). Note axial stress radiograph of the right knee (d). The symptomatology disappeared after surgical correction of medial subluxation of the patella using iliotibial tract of and patellar tendon for repairing the lateral stabilizers of the patella

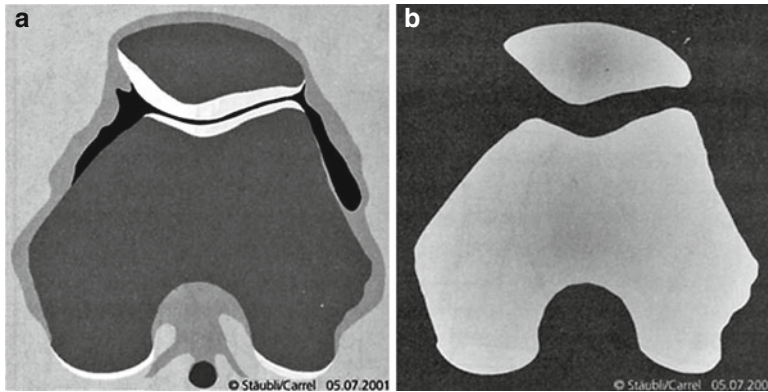


Fig. 1.5 Scheme of gadolinium-enhanced MR arthro-tomogram of the left knee in the axial plane. Note perfect patellofemoral congruence (a). Note patellofemoral incongruence of the osseous contours (b) (Reprinted from

Staeubli HU, Bosshard C, Porcellini P, et al. Magnetic resonance imaging for articular cartilage: cartilage-bone mismatch. Clin Sports Med. 2002;21:417–433. With permission from Elsevier)

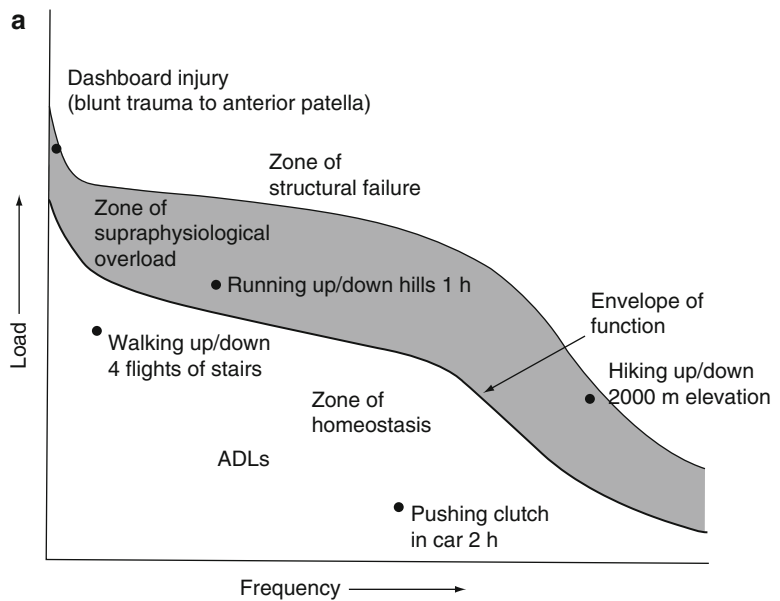
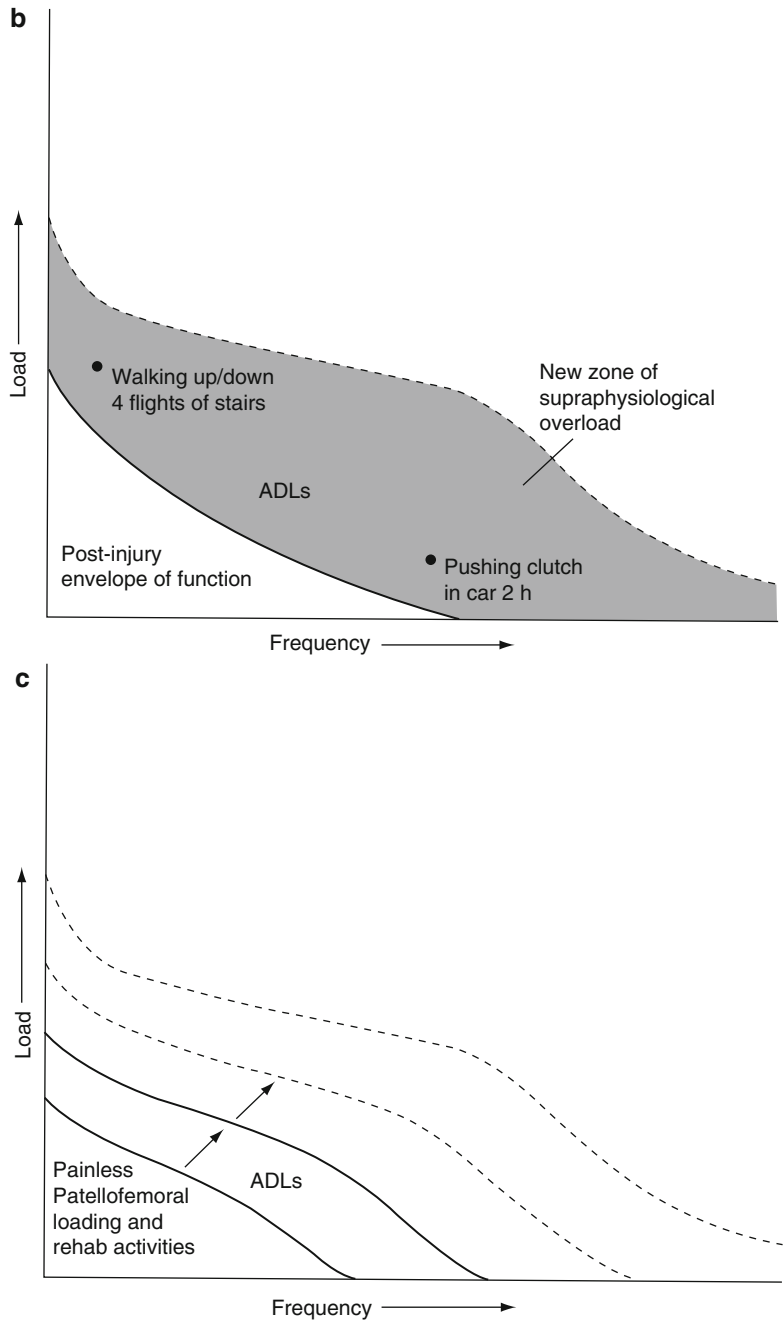


Fig. 1.6 The Dye envelope of function theory. (a) Zone of homeostasis, envelope of function and zone of structural failure. (b) Post-injury envelope of function. (c) Decreasing loading to within the new envelope of function allows normal healing processes

Fig. 1.6 (continued)



In Search of the Etiology of Anterior Knee Pain

2

Jan Näslund

In any long-term pain condition, a causal explanation is important to seek. One is that a mild disturbance of homeostasis, so mild that it is clinically undetectable, could signal pain. Pain mechanisms differ depending on onset stimulus, immune reactions, and type of inflammation, so distinguishing possible pain mechanisms is valu-

able. Analyzing inflammatory mediators and immune cells may reveal meaningful information in the search for a diffuse AKP etiology. Retrospective analyses of prior treatment modalities may also yield important clues. This chapter has argued that hypoxia suits well as an etiological factor in AKP.

J. Näslund, Ph.D.
Department of Physiology and Pharmacology,
Karolinska Institute,
Stockholm, Sweden
e-mail: jan.e.naslund@ki.se

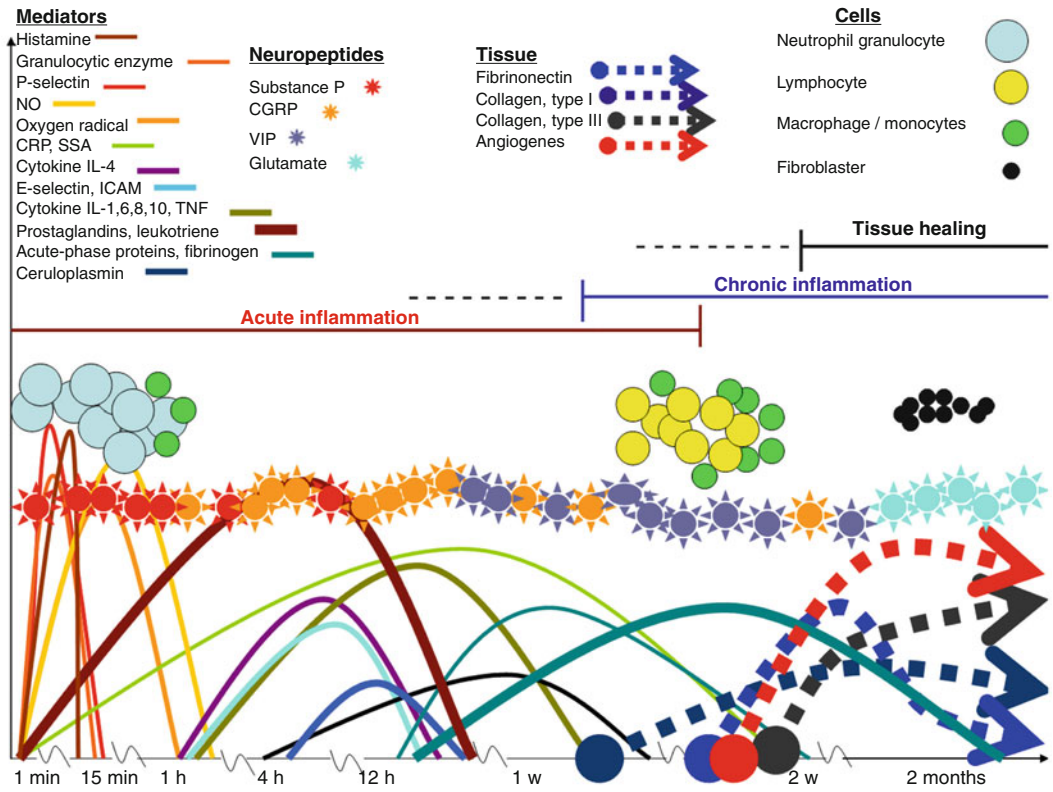


Fig. 2.1 Cells, neuropeptides, and mediators involved in inflammatory processes

Biological Causes of Anterior Knee Pain

3

Vicente Sanchis-Alfonso, Esther Roselló-Sastre,
Juan Saus-Mas, and Fernando Revert-Ros

We review the pathophysiology of anterior knee pain in the young patient. Emphasis is placed on newer findings. We have developed what we call the “Neural Model” as an explanation for the genesis of anterior knee pain. We have demonstrated a neuroanatomical basis for PFPS in the young patient and the clinical observation that the lateral retinaculum may have a key role in the origin of this pain. According to our studies we hypothesize that periodic short episodes of ischemia in the lateral retinaculum could be implicated in the pathogenesis of anterior knee pain, at least in a subgroup of anterior knee pain patients, by triggering neural proliferation of nociceptive axons (substance P positive nerves), mainly in a perivascular location.

Our findings are compatible with the tissue homeostasis theory widely accepted currently to explain the genesis of anterior knee pain. If the “neural model” of anterior knee pain proves to have a certain validity, it would lead in many cases to therapeutic recommendations to alleviate pain more effectively and safer than the attempts to correct “malalignment.” Moreover, we believe that instability in patients with PFPS can be explained, at least in part, because of the damage of nerves of the lateral retinaculum which can be related with proprioception. Our findings, however, do not preclude the possibility of pain arising in other anatomical structures such as infrapatellar fat pad, synovium and subchondral bone.

V. Sanchis-Alfonso, M.D., Ph.D. (✉)
International Patellofemoral Study Group,
ACL Study Group, Hospital 9 de Octubre,
Hospital Arnau de Vilanova, School of Medicine,
Valencia Catholic University,
Valencia, Spain
e-mail: vicente.sanchis.alfonso@gmail.com

E. Roselló-Sastre, M.D., Ph.D.
Department of Pathology,
Hospital Universitario Dr. Peset,
Valencia, Spain

J. Saus-Mas • F. Revert-Ros
Centro de Investigación Príncipe Felipe, Biomedicina,
Valencia, Spain

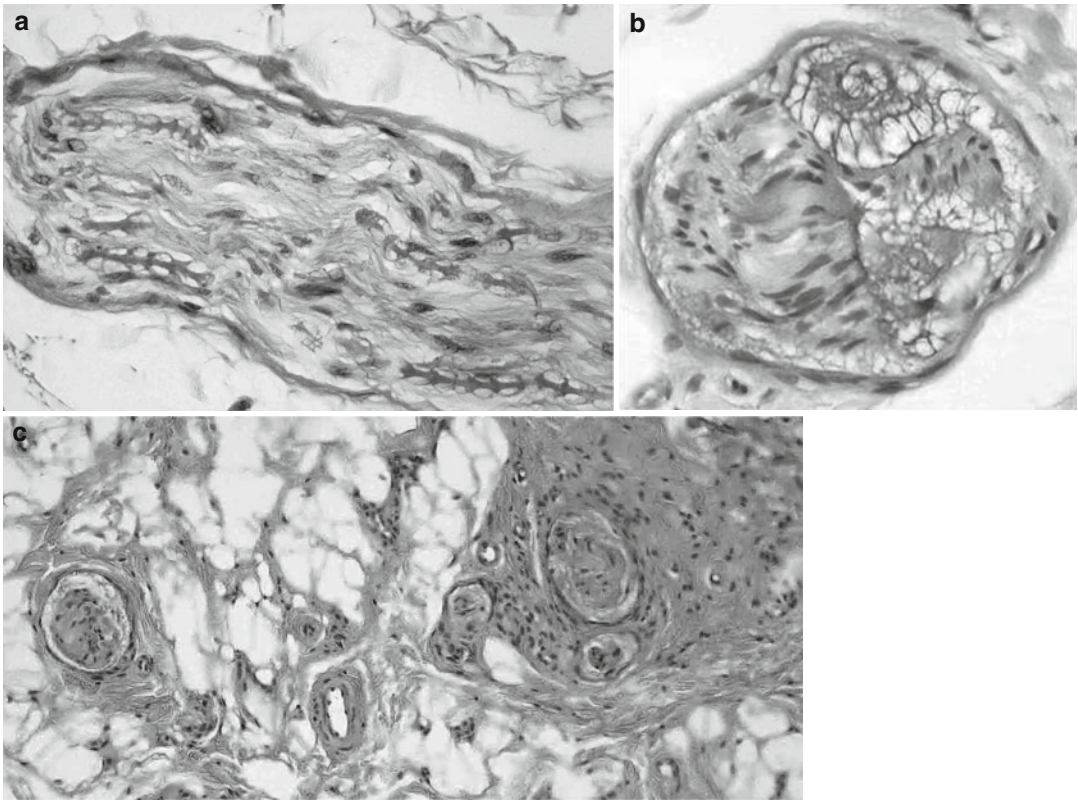


Fig. 3.1 Histologic features of a normal nerve (**a**), a nerve with neural myxoid degeneration (**b**), and a tissular neuroma (**c**) in the lateral retinaculum (Hematoxylin-Eosin stain) (**b** – Reproduced from Sanchis-Alfonso V, Roselló-Sastre E, Monteagudo-Castro C, et al. Quantitative analysis of nerve changes in the lateral retinaculum in patients with isolated symptomatic patellofemoral malalignment. A preliminary study. *Am J Sports Med.* 1998;26:703–9. Copyright © 1998, Reprinted by permission of SAGE Publications)

1998;26:703–9. Reprinted by permission from Theime, c – Reproduced from Sanchis-Alfonso V, Roselló-Sastre E, Monteagudo-Castro C, et al. Quantitative analysis of nerve changes in the lateral retinaculum in patients with isolated symptomatic patellofemoral malalignment. A preliminary study. *Am J Sports Med.* 1998;26:703–9. Copyright © 1998, Reprinted by permission of SAGE Publications)

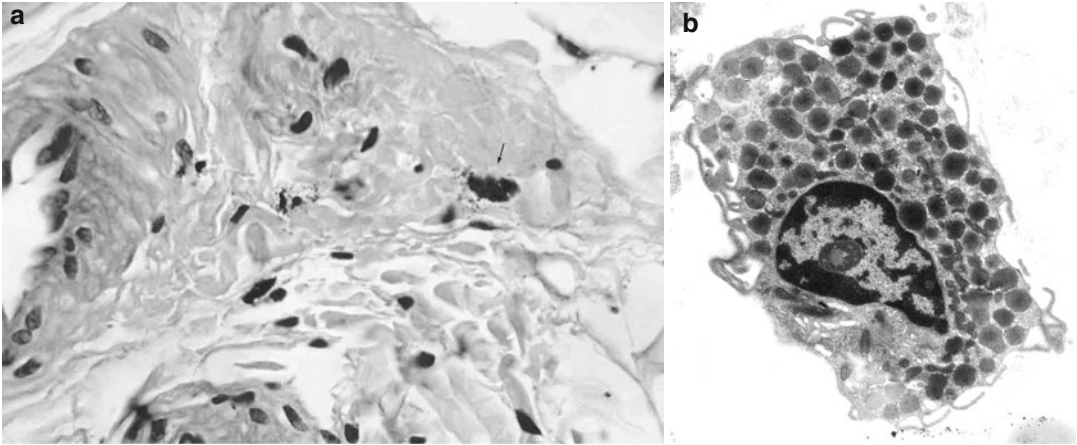


Fig. 3.2 Mast cells are abundant in the stroma (*arrow*), mainly in a perivascular disposition. Some of them show a degranulation process (activated mast cells) (**a**) (Giemsa stain). Ultrastructural image of a mast cell of the lateral retinaculum with its cytoplasm full of chemotactic granules, (TEM) (**b**) (**a** – Reproduced from Sanchis-Alfonso V, Roselló-Sastre E. Immunohistochemical analysis for

neural markers of the lateral retinaculum in patients with isolated symptomatic patellofemoral malalignment. A neuroanatomic basis for anterior knee pain in the active young patient. *Am J Sports Med.* 2000;28:725–31. Copyright © 2000, Reprinted by permission of SAGE Publications)

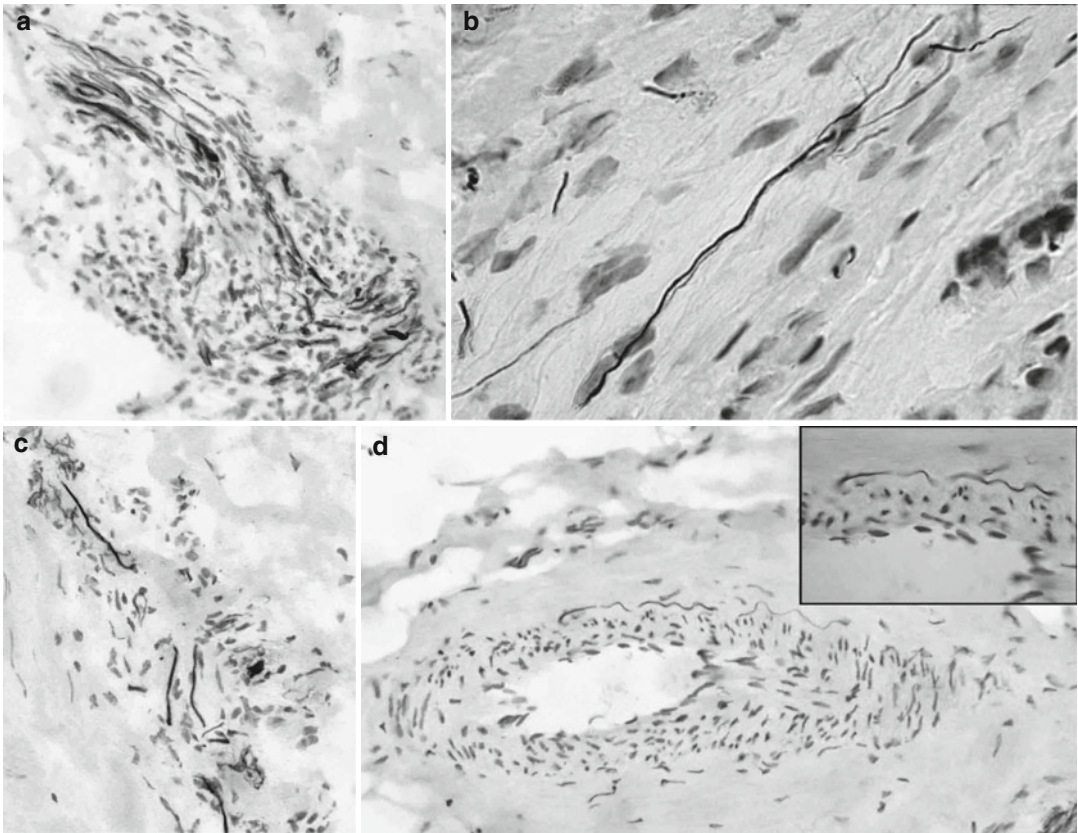


Fig. 3.3 An increased innervation is evident in the connective tissue, showing microneuromas (a) and free nerve endings immersed in the stroma (b), or next to small vessels (c). Vascular innervation is also increased with tiny axons arranged like a necklace in the adventitia (d) (a–c – Reproduced from Sanchis-Alfonso V, Roselló-Sastre E.

Immunohistochemical analysis for neural markers of the lateral retinaculum in patients with isolated symptomatic patellofemoral malalignment. A neuroanatomic basis for anterior knee pain in the active young patient. *Am J Sports Med.* 2000;28:725–31. Copyright © 2000, Reprinted by permission of SAGE Publications)

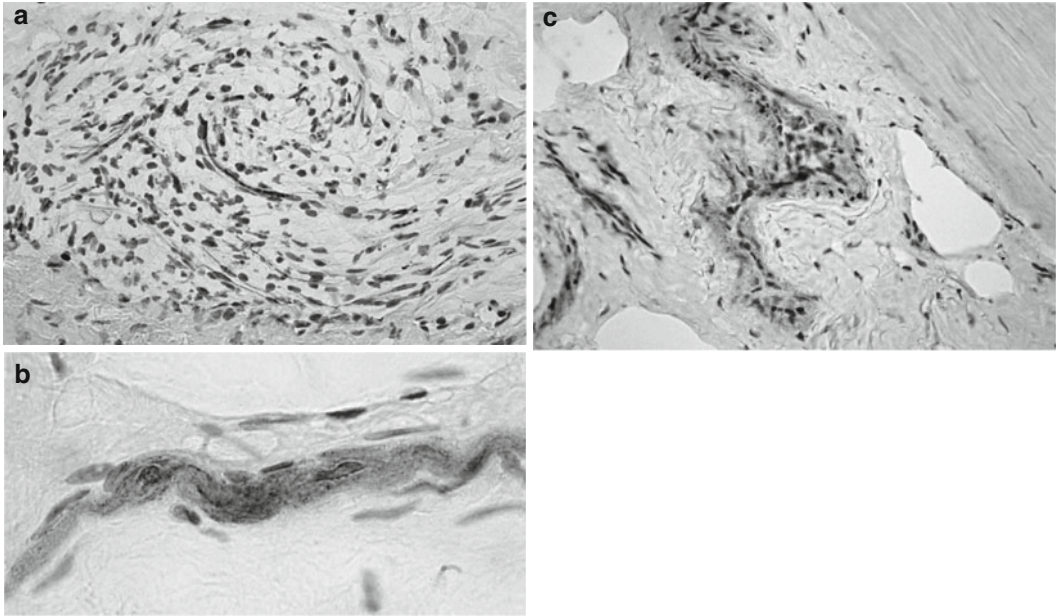


Fig. 3.4 Neuromas are rich in nociceptive axons, as can be demonstrated studying substance P (**a**). Substance P is present in the axons of the nerves and in the free nerve endings with a granular pattern (**b**), and can be observed in the vessel walls in some patients with painful symptoms (**c**) (Immunohistochemistry for Substance P. Frozen sections) (**a**, **b** – Reproduced from Sanchis-Alfonso V,

Roselló-Sastre E. Immunohistochemical analysis for neural markers of the lateral retinaculum in patients with isolated symptomatic patellofemoral malalignment. A neuroanatomic basis for anterior knee pain in the active young patient. *Am J Sports Med.* 2000;28:725–31. Copyright © 2000, Reprinted by permission of SAGE Publications)

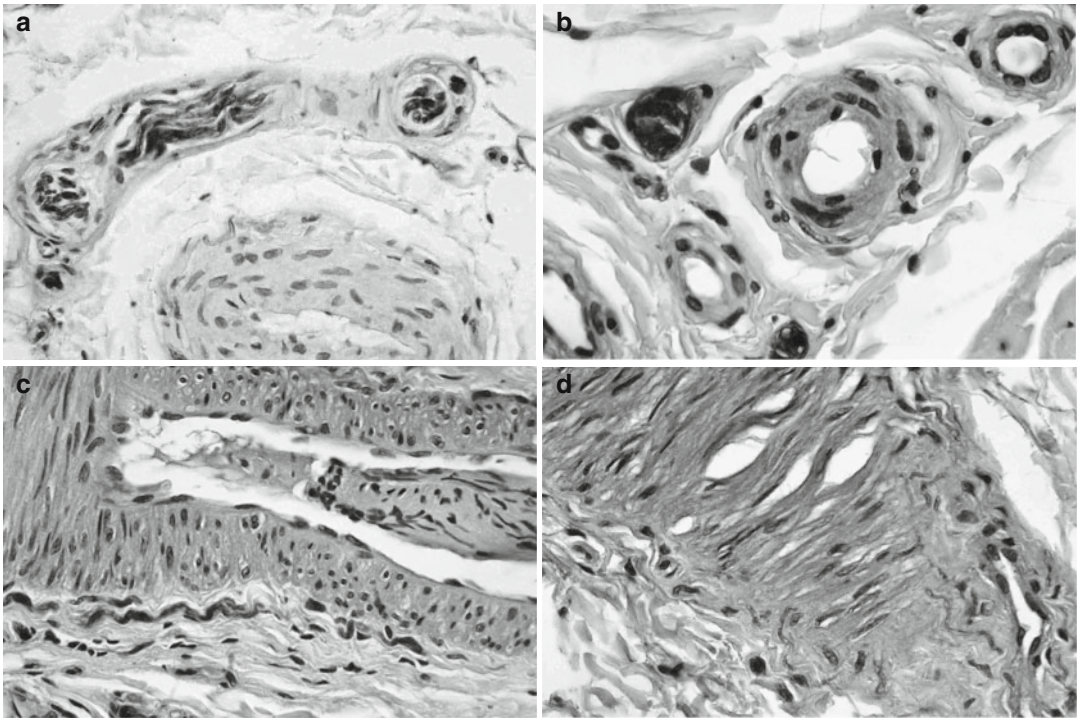


Fig. 3.5 An increase in periadventitial innervation is detectable in our patients expressed as a rich vascular network made up of tiny myelinated fibers that, from the arterial adventitia, enter into the outer muscular layer, conforming a necklace (**a**, **b**). Transversal section (**c**) and tangential section (**d**) (Immunohistochemistry for protein S-100) (Reproduced from Sanchis-Alfonso V, Roselló-

Sastre E, Monteagudo-Castro C, et al. Quantitative analysis of nerve changes in the lateral retinaculum in patients with isolated symptomatic patellofemoral malalignment. A preliminary study. *Am J Sports Med.* 1998;26:703–9. Copyright © 1998, Reprinted by permission of SAGE Publications)

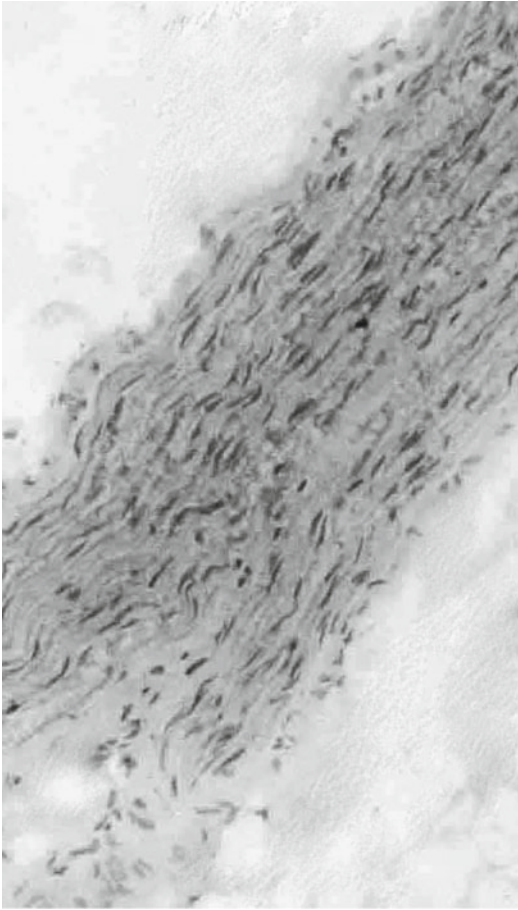


Fig. 3.6 NGF is present in thick nerves in the axons in a granular distribution and in the cytoplasm of the Schwann cells

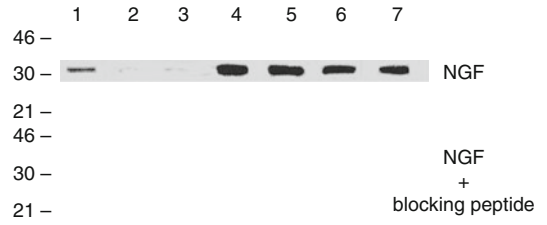


Fig. 3.7 Immunoblotting detection of NGF, showing a thick band located at the level of NGF precursor in patients with pain (cases 4,5,6,7) and absence or a very thin band in the patients with instability as the main symptom (cases 1,2,3). The numbers at the *left* indicate molecular mass in kDa

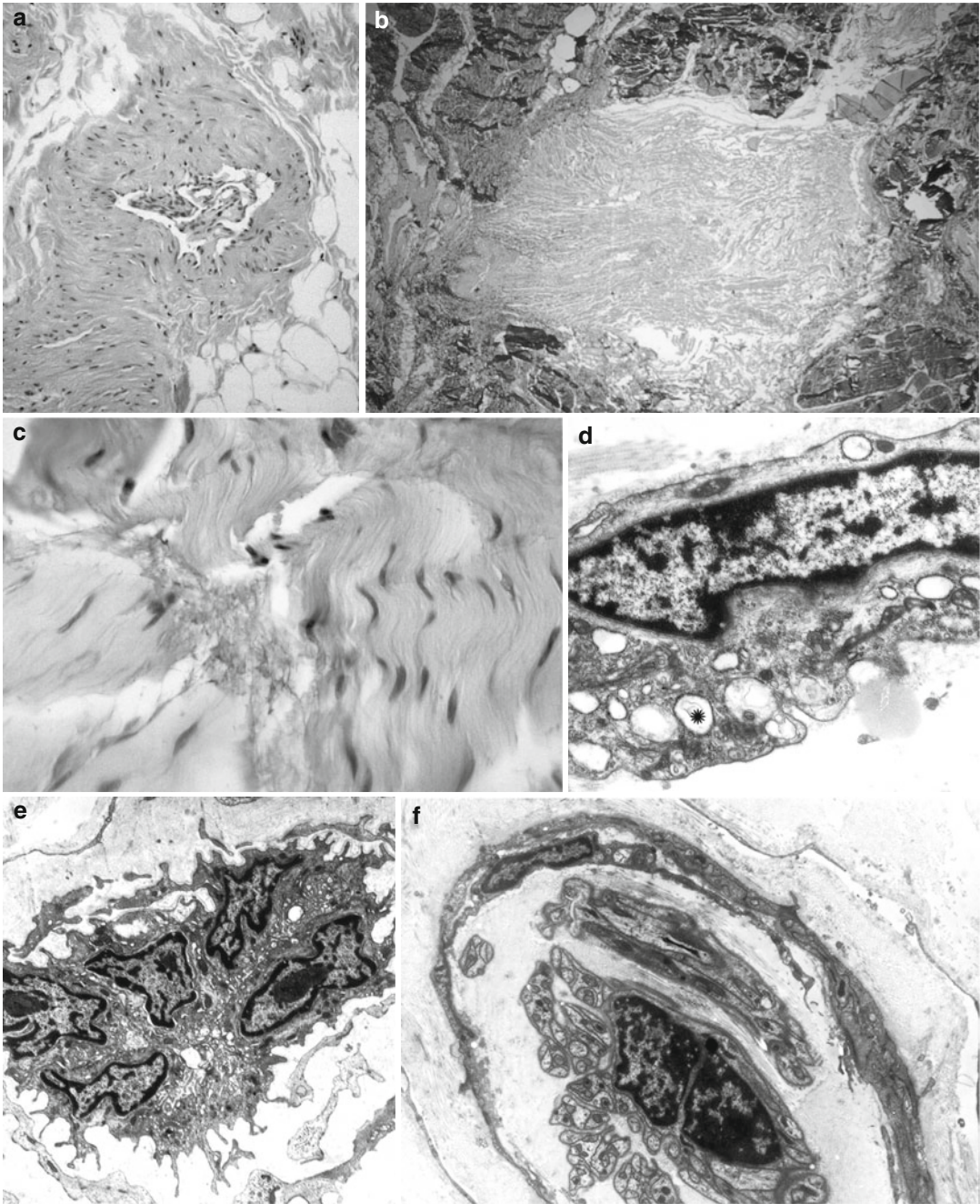


Fig. 3.8 (a) Arterial vessel in the reticular tissue can show a prominent and irregular endothelium and thick muscular walls or even an irregular reduction of the vascular lumen (Hematoxylin-Eosin stain). (b) InfRACTED foci in the connective tissue showing a degenerative pattern of the collagen fibers, with loss of the fibrillar component and accumulation of myxoid material in the interstitium, (Masson's Trichrome stain). (c) Myxoid stromal degeneration in the middle of the fibrous reticular tissue (Hematoxylin-Eosin

stain). (d) Degenerative changes in fibroblasts (increased autophagic vacuoles – *asterisk* –) secondary to hypoxia (TEM). (e) Young vessels with endothelial cells containing active nuclei and conspicuous nucleoli. (f) Neural sprouting is detected ultrastructurally as a bunch of tiny axons immersed in the Schwann cell cytoplasm. (g) Neural sprouting detail (b – Reproduced with permission from Sanchis-Alfonso V, Roselló-Sastre E. Proliferación neural e isquemia. Rev Patol Rodilla. 1998;3:60–3)



Fig. 3.8 (continued)

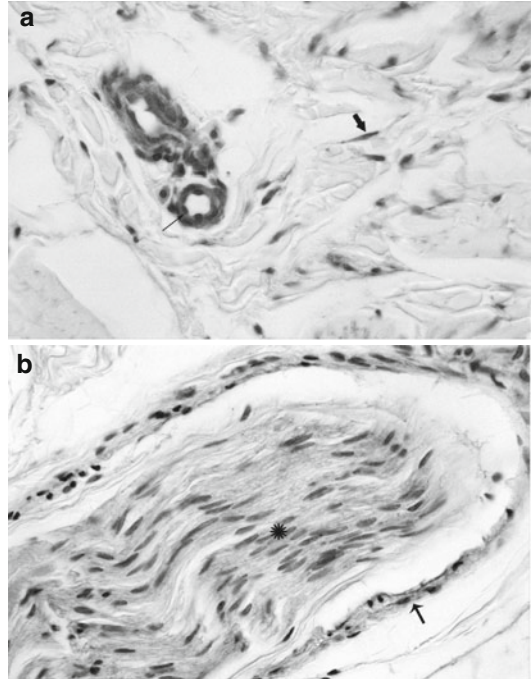


Fig. 3.9 VEGF is present in small vessels (wall and endothelium) (*thin arrow*) and in perivascular fibroblasts (*thick arrow*) in patients with moderate to severe pain (**a**). Some cases have VEGF expression even in the perineural sheath (*thin arrow*) and inside the axons (*asterisk*) (**b**) (Immunohistochemistry for VEGF)

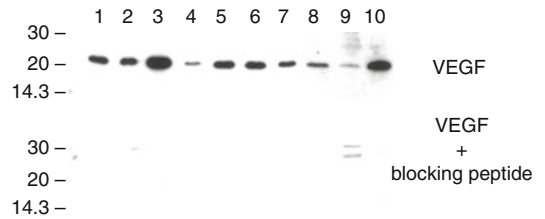


Fig. 3.10 Immunoblotting detection of VEGF, showing a thicker band in cases with severe pain, whereas it is hardly expressed in two patients in whom instability and not pain was the main problem (Severe pain: cases 2,3,10; moderate pain: cases 1,5,8; and light pain: cases 4,6,7,9)

Fig. 3.11 Osteoporosis associated to anterior knee pain syndrome (left knee)

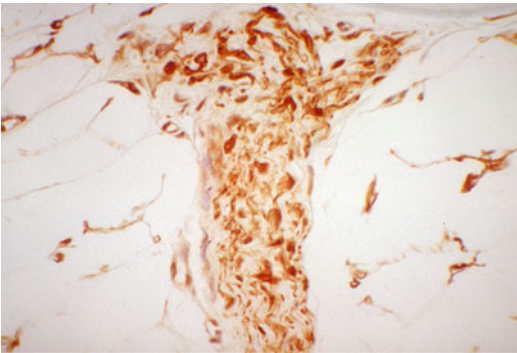
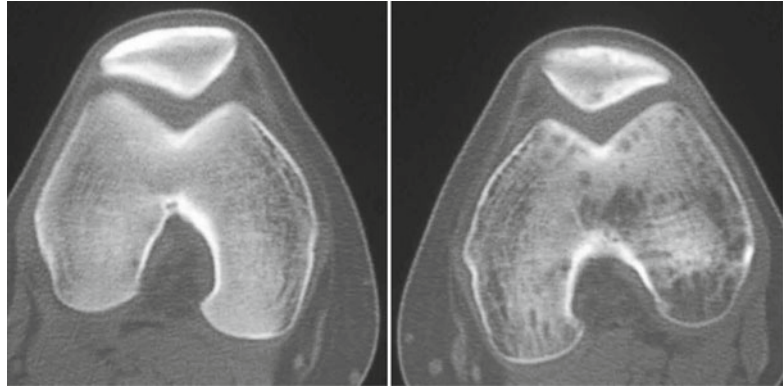


Fig. 3.12 Medium-size nerves near the osteotendinous insertion suffer a “nerve sprouting” process, losing their perineurium sheath and liberating their tiny nerve branches (anti-S 100 protein)

Pathogenesis of Anterior Knee Pain in the Active Young: Is There a Relation Between the Presence of Patellofemoral Malalignment and Pain?

What Have We Learned from Realignment Surgery?

Vicente Sanchis-Alfonso, Fermín Ordoño, Alfredo Subías-López, and Carmen Monserrat

This chapter is not intended to advocate for a particular surgical technique, but it does provide insight into improving our understanding of the pathophysiology of anterior knee pain syndrome. Our objectives were: to identify a relationship, or lack of one, between the presence of PFM and the presence of anterior knee pain; to analyze the long-term response of VMO muscle fibers to increased resting length; and to determine the

incidence of patellofemoral osteoarthritis after IPR surgery. Our findings indicate (1) that not all PFM knees show symptoms; that is, PFM is not a sufficient condition for the onset of symptoms, at least in postoperative patients; (2) that the advancement of VMO has no deleterious effects on VMO; and (3) that IPR does not predispose to retropatellar osteoarthritis.

V. Sanchis-Alfonso, M.D., Ph.D. (✉)
International Patellofemoral Study Group,
ACL Study Group, Hospital 9 de Octubre,
Hospital Arnau de Vilanova, School of Medicine,
Valencia Catholic University, Valencia, Spain
e-mail: vicente.sanchis.alfonso@gmail.com

F. Ordoño, M.D., Ph.D.
Department of Neurophysiology,
Hospital Arnau de Vilanova, Valencia, Spain

A. Subías-López, M.D.
Orthopaedic Surgeon,
Hospital General de Almansa,
Albacete, Spain

C. Monserrat
Department of Radiology,
Hospital Arnau de Vilanova,
Valencia, Spain

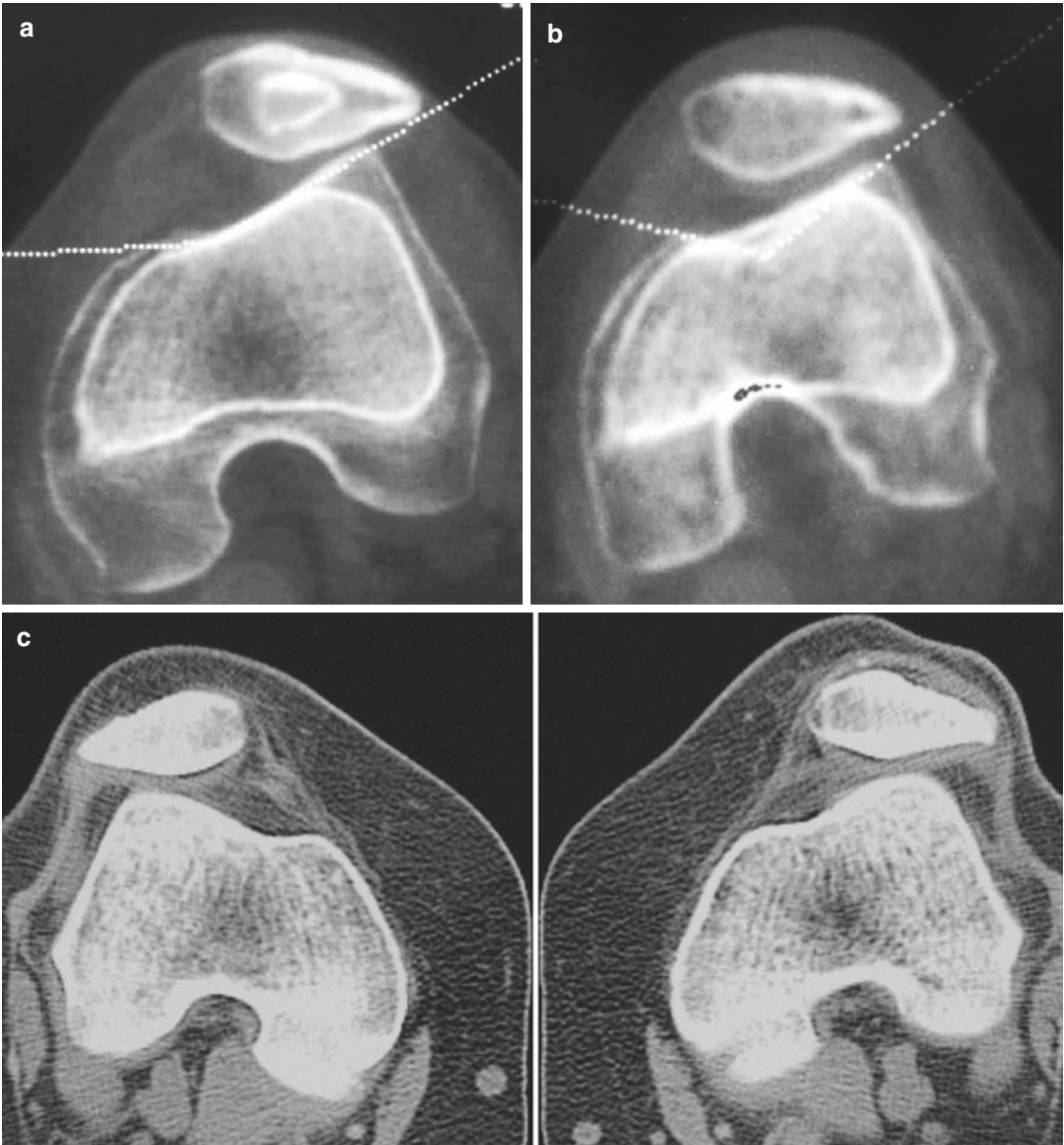


Fig. 4.1 CT at 0° of knee flexion. (a) Preoperative CT: PFM. (b) At short-term follow-up after IPR, there is a correct patellofemoral congruence. (c) At long-term follow-up (13 years after IPR), we can observe a bilateral asymptomatic PFM (a—Reproduced from Sanchis-Alfonso

V, Roselló-Sastre E, Martínez-SanJuan V. Pathogenesis of anterior knee pain syndrome and functional patellofemoral instability in the active young. A review. *Am J Knee Surg.* 1999;12:29–40. Reprinted by permission from Thieme)

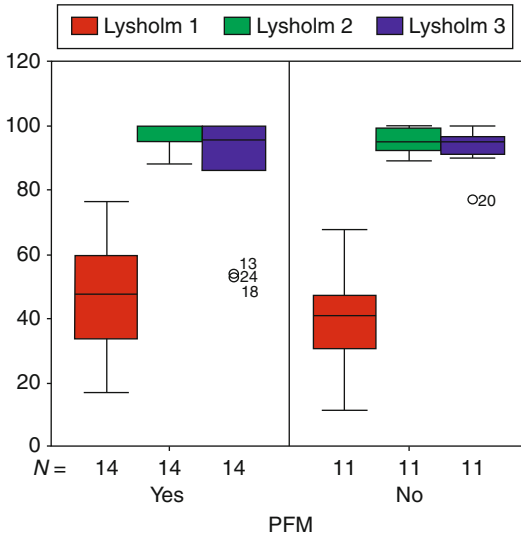


Fig. 4.2 Lysholm scores of the patients with and without PFM. Lysholm 1 = Preoperative Lysholm score; Lysholm 2 = Lysholm score at medium-term follow-up; Lysholm 3 = Lysholm score at long-term follow-up

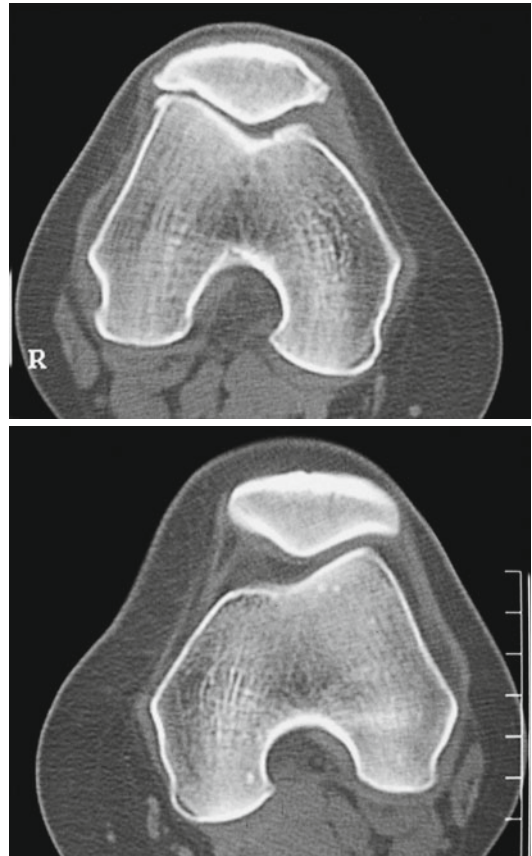


Fig. 4.3 CT images at 0° of knee flexion from a female 36 years old operated on 12 years ago of the right knee with an Insall's proximal realignment. We can see osteophytes on the patella and femoral condyles with a visible narrowing of the patellofemoral joint gap (right knee). However, clinical result at 12 years follow-up was good. The left knee is asymptomatic despite the PFM

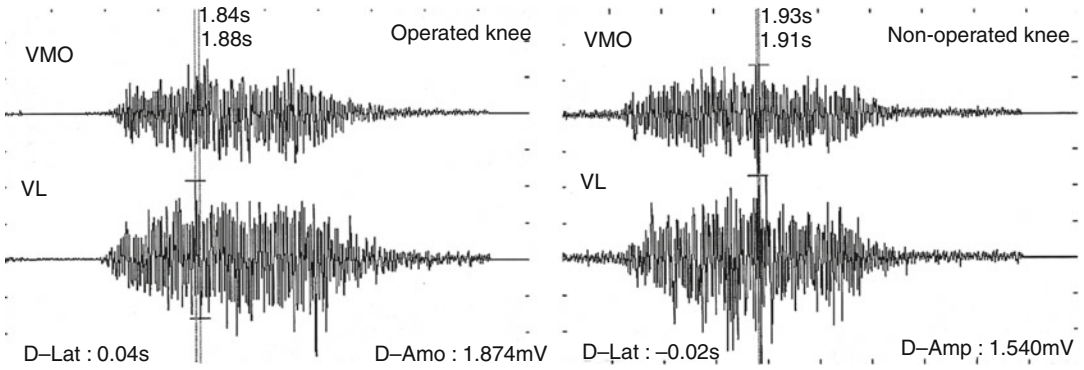


Fig. 4.4 SEMG activity of the VMO of the operated knee and VMO of the contralateral asymptomatic nonoperated knee. SEMG activity of the VL of the operated knee and VL of the contralateral asymptomatic nonoperated knee

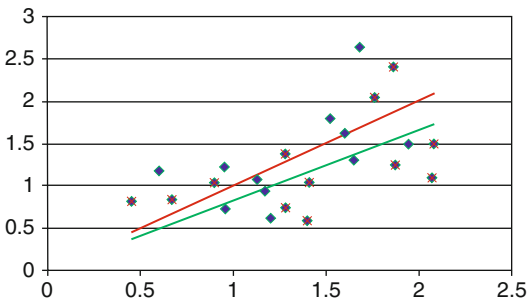


Fig. 4.5 VMO: VL ratio of the operated knee (green line) vs. nonoperated knee (red line). Asterisk = nonoperated knee. Rhombus = operated knee

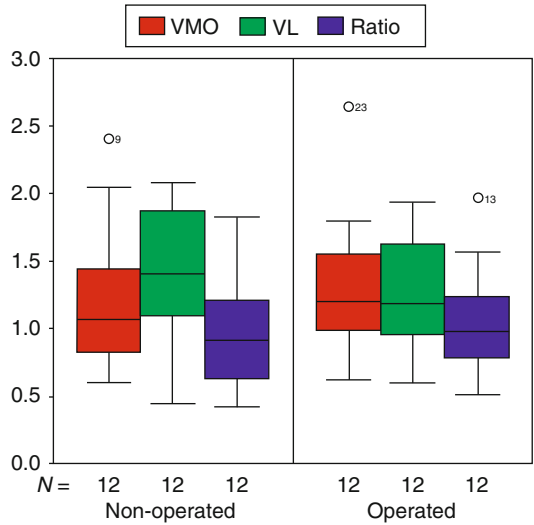


Fig. 4.6 Amplitude of the VMO and VL of the operated knee and the contralateral asymptomatic nonoperated knee. VMO: VL ratio of the operated knee vs. nonoperated knee

Unilateral Anterior Knee Pain as a Sign of Knee Asymmetry

5

K. Donald Shelbourne and Emily E. Krodel

Anterior knee pain is a common complaint in the orthopaedic community. It is nonspecific in nature and can be frustrating to treat. Symmetry between the knees is necessary for people to perform everyday activities comfortably and for athletes to function at a high level. Through observation and research on thousands of patients, we have found that unilateral anterior knee pain can largely be attributed to asymmetry between knees, in particular the loss of full knee extension. Loss of full knee extension can be caused by many factors. Regardless of the specific etiology, patients favor one knee by standing solely on the normal knee rather than using both legs equally. Patients subconsciously stop using the

involved leg and a flexion contracture can develop when they avoid weight bearing on the leg and they keep the knee bent. As the leg becomes weaker, the patient favors it even more and the knee can become “deconditioned.” Even small degrees of extension loss can cause anterior knee pain. Our experience has shown that the most important objective to keep in mind when dealing with anterior knee pain is the restoration and maintenance of symmetry between knees. This is primarily accomplished by treating any deficits in knee range of motion first followed by improving leg strength with appropriate rehabilitative exercises and instructions for correct symmetrical use of the legs with daily activities.

K.D. Shelbourne, M.D. (✉)
Shelbourne Knee Center at Methodist Hospital,
Indianapolis, IN, USA

Indiana University School of Medicine,
Indianapolis, IN, USA
e-mail: acldoc@aclmd.com

E.E. Krodel, M.D.
Indiana University School of Medicine,
Indianapolis, IN, USA

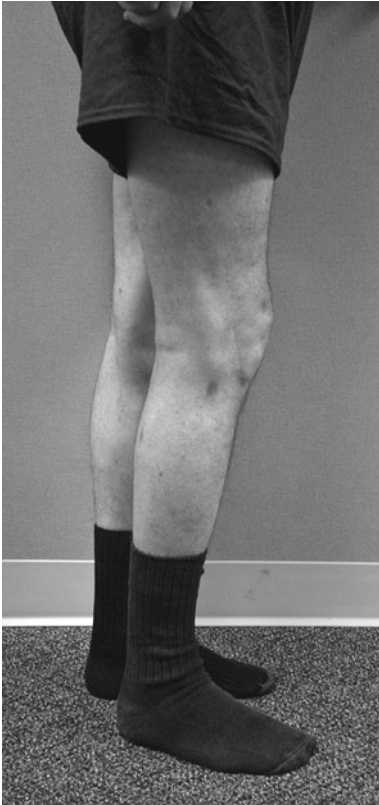


Fig. 5.1 Patients with knee pain typically favor one leg by standing on the normal knee and leaving the painful knee bent



Fig. 5.2 As part of the physical examination, we have the patient lie supine on the examination table and observe the position of the legs. Patients who favor a knee by keeping it bent can be identified easily because they will have one leg and foot rotated externally

Fig. 5.3 Lack of normal knee extension can be observed by placing an object over the top of the knees. The bent knee will be the higher side



Fig. 5.4 To critically evaluate small degrees of extension loss, the examiner can place his or her palm behind the patient's knee and evaluate whether the patient can actively extend the knee so that the back of the knee touches the palm



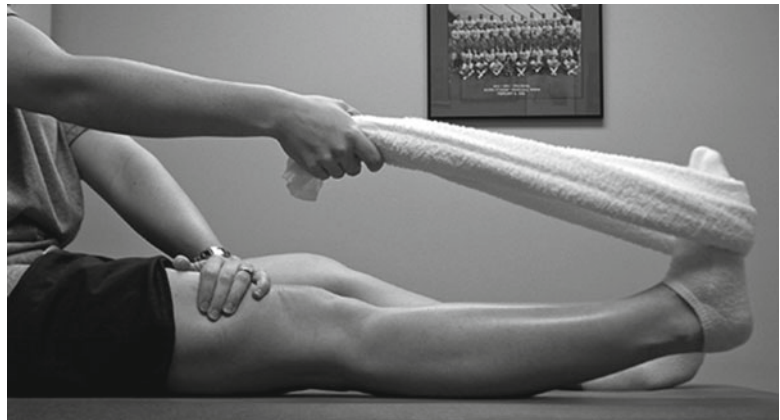
Fig. 5.5 To evaluate for knee hyperextension, the examiner places one hand above the knee to hold down the thigh and places the other hand on the foot to raise the foot passively to bring the knee into hyperextension. Not only can you evaluate the degree of hyperextension but also how easily the knee moves into hyperextension



Fig. 5.6 Heel prop exercise is used to increase knee extension. This exercise is also used to measure for knee hyperextension



Fig. 5.7 Towel extension exercise is used to increase knee extension. The patient holds onto the ends of a towel and loops the middle of the towel around the foot. While holding down the thigh, the patient can pull on the ends of the towel to bring the knee into hyperextension



Biomechanical Bases for Anterior Knee Pain and Patellar Instability

6

Vicente Sanchis-Alfonso, Carolina Ávila-Carrasco,
Jaime M. Prat-Pastor, Carlos M. Atienza,
and Enrique Cuñat

The anterior knee pain syndrome and functional patellar instability in the active young person is one of the most complex knee disorders, with a multiple factor and highly variable pathogenesis, with intermingling mechanical and neurological factors. Probably the neural factor is the cause of the well established symptoms in patients with certain mechanical anomalies and a knee overuse.

The word “*overuse*” is closely linked to sport, which is one of the most popular activities nowadays. In addition to favouring personal relationship, sport is a source of physical and mental health. It is amusing, relaxing, it encourages a sense of discipline, fellowship, team spirit, and will to excel. Therefore we ought to encourage it and support those who practise it. But sport can be the cause of lesions, and it is the orthopae-

dic surgeon’s duty not only to diagnose and heal them, but also to play an active roll in the education of the patient to prevent them. This prevention implies detecting persons and risk situations and taking an active part in the education of the sportsplayer by means of teaching healthy habits (e.g., training of the proprioception). It could be said that the sport lesions are not accidental ones, as many of them can be prevented. If the doctor, the physiotherapist, the physical trainer, and the administration do not cooperate in this prevention, the practice of sport should not be encouraged.

Taking into account that overuse, training errors, and specific patterns of mobility in each sport can be important factors in the appearance of symptoms, it is then easy to understand that reeducating the patient is necessary for the success of treatment and the prevention of relapses. To achieve this it is necessary to analyse the gait and to video-analyze how the patient practises the sport. Any treatment programme overlooking reeducation (training of brain«software», altering the expectations and the life style) will fail in the long run. In addition to that, the surgeon, the patient, and his family should judge whether it is convenient for the patient himself to continue practicing the same sport at the same level as before the onset of the symptoms. One has to be realistic when counselling the patient’s return to sport. We have to keep in mind that not everyone is fit to practice a sport, for instance, people who show important biomechanical alterations in the alignment of the lower limbs.

V. Sanchis-Alfonso, M.D., Ph.D. (✉)
International Patellofemoral Study Group,
ACL Study Group, Hospital 9 de Octubre,
Hospital Arnau de Vilanova, School of Medicine,
Valencia Catholic University,
Valencia, Spain
e-mail: vicente.sanchis.alfonso@gmail.com

C. Ávila-Carrasco, Mech. Eng.
J.M. Prat-Pastor, M.D., Ph.D.
C.M. Atienza, Mech. Eng., Ph.D.
Instituto de Biomecánica de Valencia (IBV),
Universidad Politécnica de Valencia,
Grupo de Tecnología Sanitaria (GTS-IBV),
CIBER-BBN, Valencia, Spain

E. Cuñat
E. Cuñat Physiotherapy,
Valencia, Spain

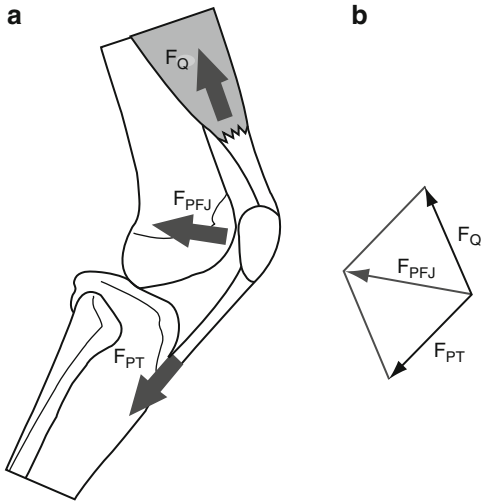


Fig. 6.1 (a) Simplified scheme of the forces acting on the patellofemoral joint. (b) Graphic calculation of the patellofemoral reaction force. F_Q force upon quadriceps, F_{PT} force transmitted to the patellar tendon, F_{PFJ} reaction force upon the patellofemoral joint

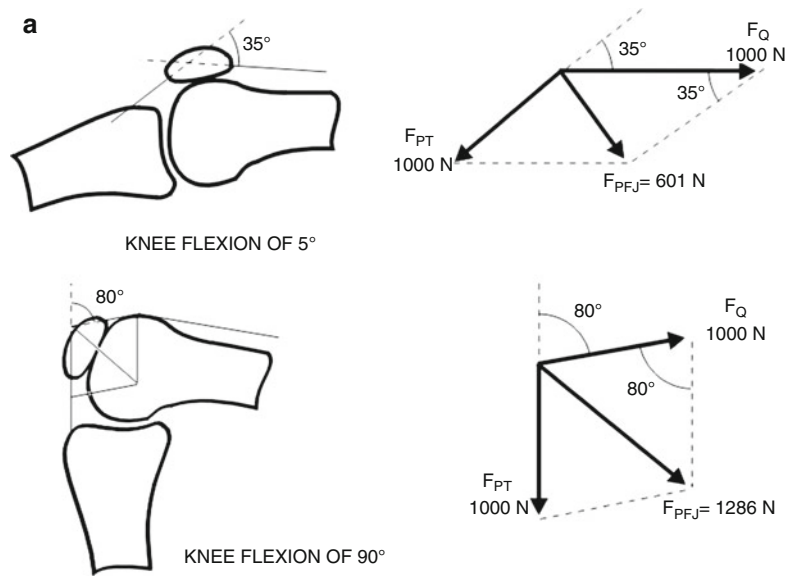


Fig. 6.2 (a) Patellofemoral reaction force determined for a knee flexion of 5° and 90° with a quadriceps muscle force of 1,000 N. (b) The reaction force increases as the knee flexion increases. Positions of maintained knee flexion are frequent in sports (b – Reproduced with permission of Promo Sport)



Fig. 6.2 (continued)

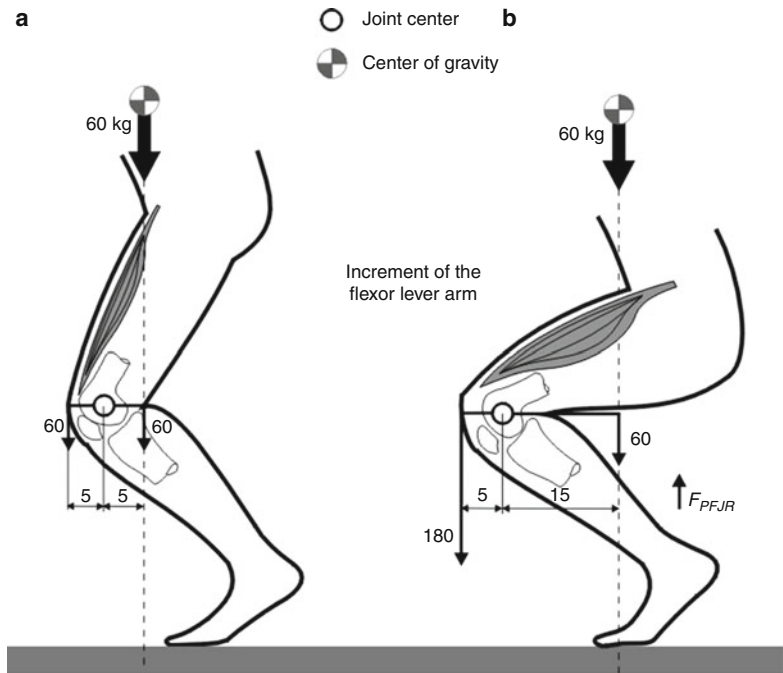


Fig. 6.3 (a, b) Body weight line of action and quadriceps extension force applied to different positions of knee flexion. Effect of the increment of the flexor lever arm upon the reaction force in the patellofemoral joint (F_{PFJR}) (Units of force kg, and distance cm)

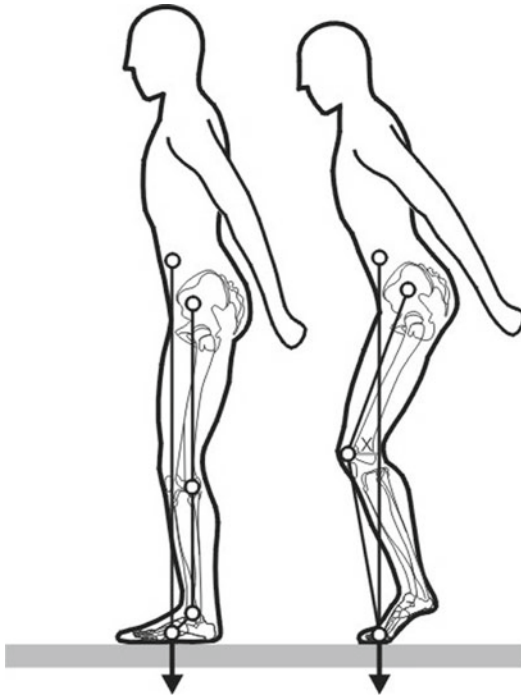


Fig. 6.4 Effect of the hip flexion on the reaction force in the patellofemoral joint

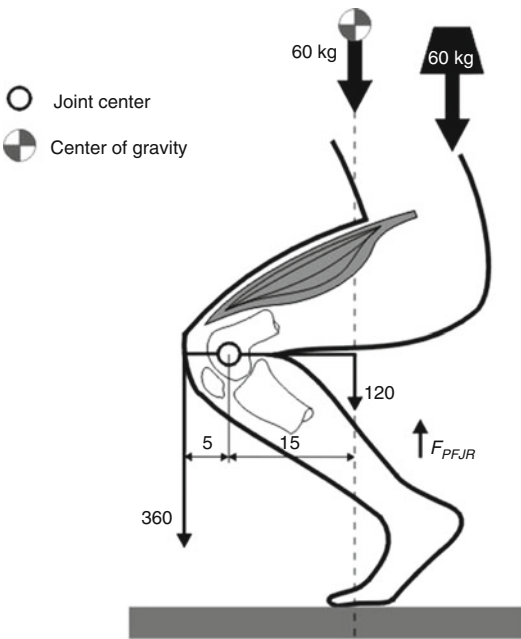


Fig. 6.5 Effect of the complementary weights (60 kg) upon the patellofemoral joint reaction force (F_{PFJR}) (Units of force kg, and distance cm)

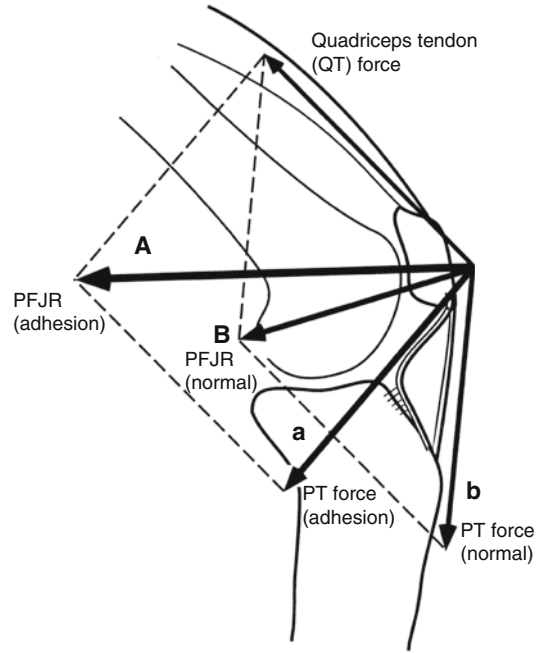
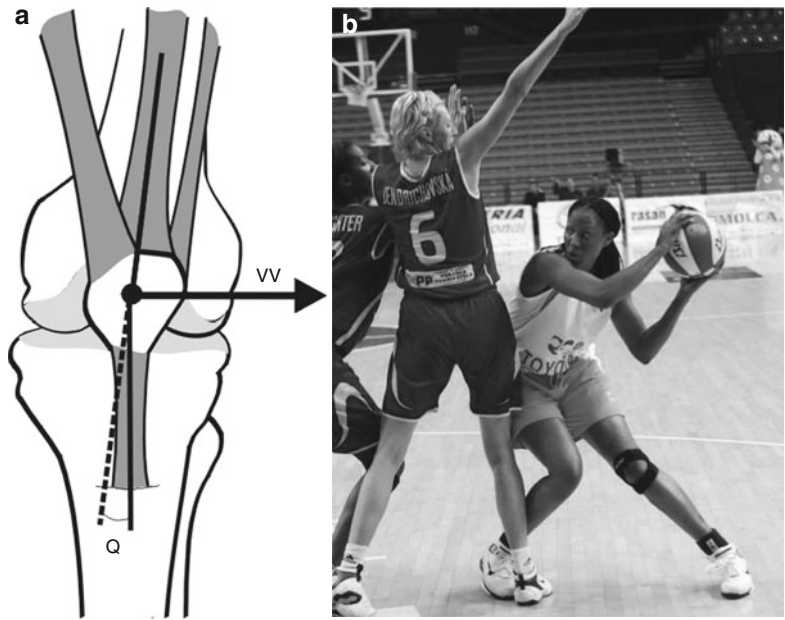


Fig. 6.6 Patellofemoral joint reaction force (F_{PFJR}) in a knee with patellar tendon adhesions to the proximal tibial surface (Reproduced from Ahmad CS, Kwak SD, Ateshian GA, et al. Effects of patellar tendon adhesion to the anterior tibia on knee mechanics. Am J Sports Med. 1998;26:715–24.. Reprinted by permission of SAGE Publications)

Fig. 6.7 (a) Q angle and valgus vector (VV). The Q angle imposes a valgus vector in the last degrees of extension. (b) In many sport positions, knee valgus is strained, which increases the Q angle and the valgus vector (Fig. b is reproduced with permission from ROS CASARES/JACOBO PAYA)



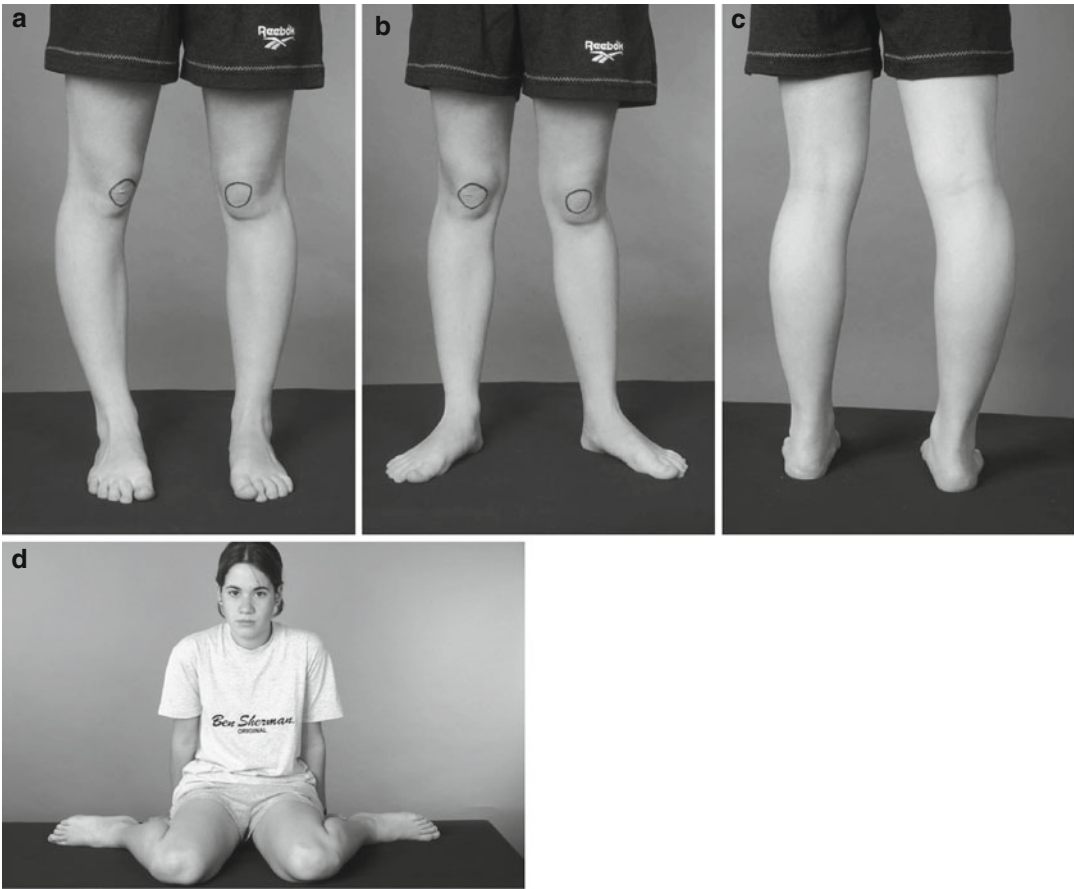


Fig. 6.8 “Miserable malalignment syndrome” is characterized by inward-looking patellae (a), external tibial torsion (b), pronated right foot with positive Helbing’s sign (medial bowing of the Achilles tendon) (c), and femoral neck anteversion (d) (Reproduced from Sanchis-Alfonso

V, Roselló-Sastre E, Martínez-SanJuan V. Pathogenesis of anterior knee pain syndrome and functional patellofemoral instability in the active young. *Am J Knee Surg.* 1999;12:29–40. Reprinted by permission from Thieme)

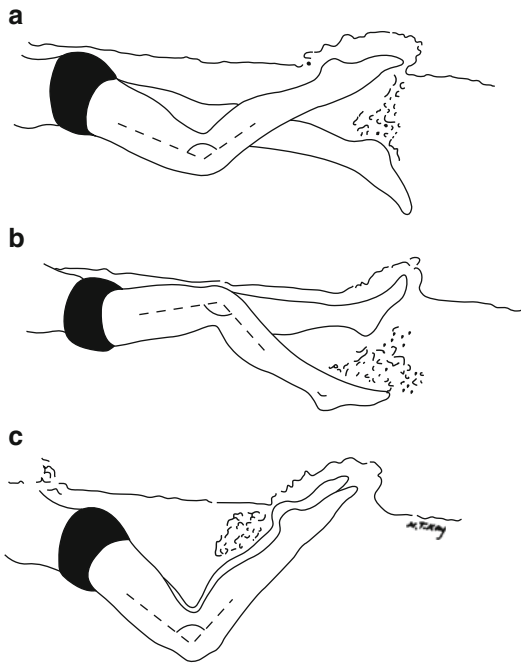


Fig. 6.9 Flexion of the knee in freestyle (a), backstroke (b), and butterfly (c). Degree of flexion associated with each impulse (Reprinted from Rodeo SA. Knee pain in competitive swimming. Clin Sports Med. 1999;18:379–87. With permission from Elsevier)

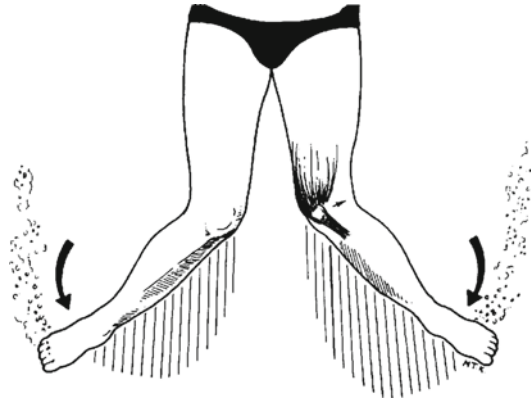


Fig. 6.10 Position of the lower limbs in breast stroke (Reprinted Rodeo SA. Knee pain in competitive swimming. Clin Sports Med. 1999;18:379–87. With permission from Elsevier)

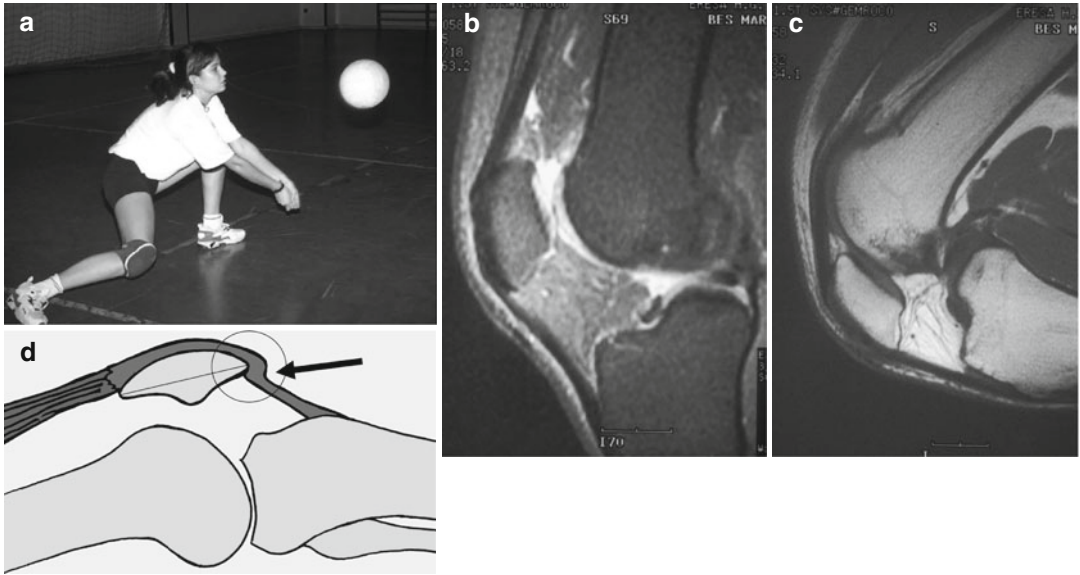


Fig. 6.11 (a) Volleyball player with her left knee in a maintained hyperflexion position (increase of the patellofemoral joint reaction force). The right knee is forced into excessive valgus and, eventually, will sustain an indirect or direct traumatism. (b, c) Functional study of the knee by magnetic resonance in a patient with right jumper's knee and a highly positive Puddu's maneuver. With

flexion of the knee, the inferior patellar pole impinges on the patellar tendon posterior aspect in its proximal end. The patient referred severe pain in the knee after any position with maintained hyperflexion. He had problems with everyday activities like driving a car. (d) Scheme to show Puddu's maneuver producing impingement of the patellar inferior pole upon the patellar tendon



Fig. 6.12 Generalized ligamentous laxity criteria: elbow hyperextension $>10^\circ$ (a), fifth finger passive hyperextension $>90^\circ$ (b), passive thumb to forearm contact (c), knee hyperextension $>10^\circ$ (d), palms in contact with the ground with knees extended (e). Ligamentous laxity exists when the patient can do three or more of these tests

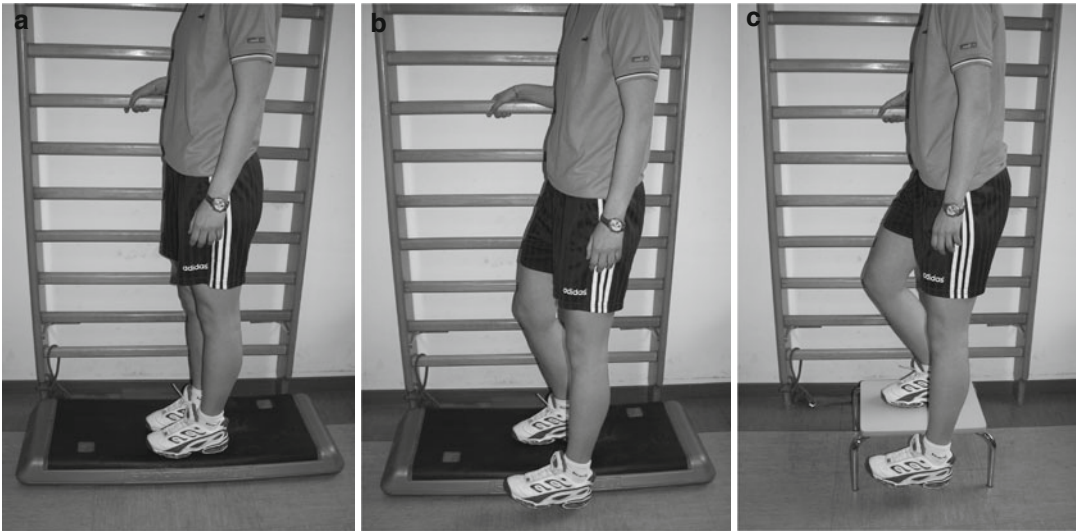


Fig. 6.13 Exercises for quadriceps (of the right leg) in closed kinetic chain (lateral step) with eccentric work. (a) Starting position. (b) Strengthening position. (c) Strengthening position with a higher step

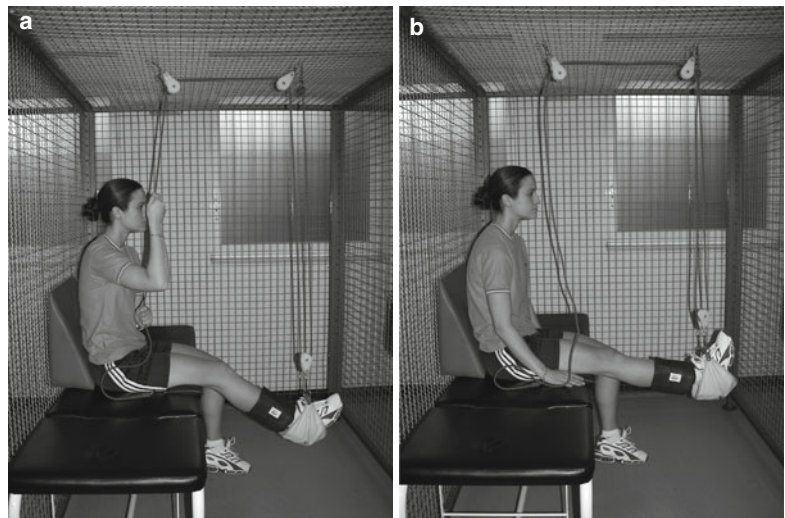


Fig. 6.14 Strengthening of quadriceps in open kinetic chain with eccentric work. Patient sitting down (in Rocher cage). (a) With the help of pulleys, the patient extends the limb. (b) After that she flexes the knee, exercising the quadriceps in eccentric phase

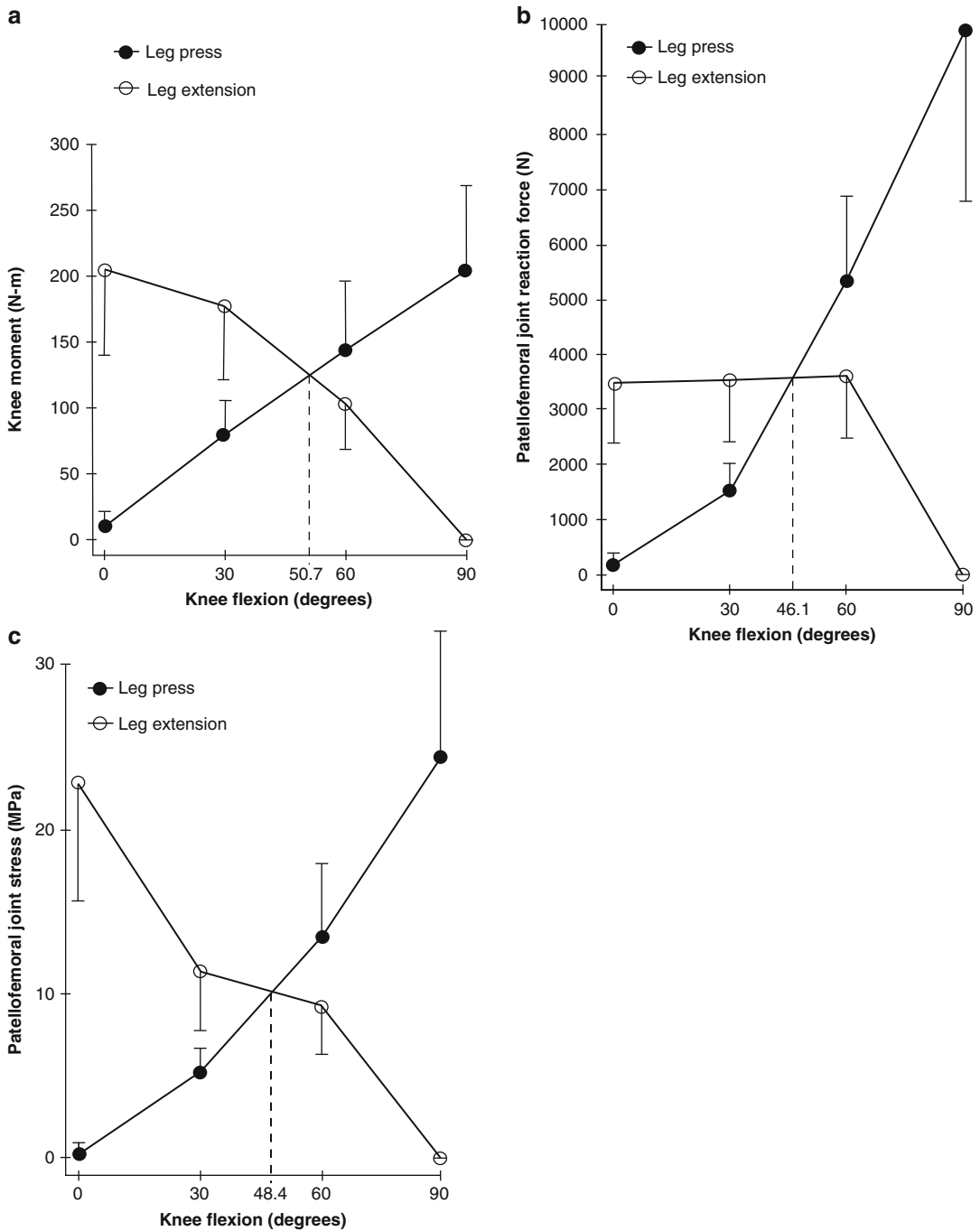


Fig. 6.15 (a) Comparative values of the articular moment at 0°, 30°, 60°, and 90° in open and closed kinetic chains. (b) Comparative values of the reaction force at 0°, 30°, 60°, and 90° in open and closed kinetic chains. (c) Comparative values of the pressures at 0°, 30°, 60°, and

90° in open and closed kinetic chains (Reproduced from Steinkamp LA, Dillingham MF, Markel MD, et al. Biomechanical considerations in patellofemoral joint rehabilitation. *Am J Sports Med.* 1993;21:438–44. Reprinted by permission of SAGE Publications)

Najeeb Khan, Donald C. Fithian, and Eiki Nomura

Acute patellar dislocation is a common injury that can lead to disabling knee pain and/or recurrent instability. Recent research focuses on patellofemoral anatomy, on the injuries associated with acute patellar dislocation, and the specific contributions the injured structures make to patellar stability in intact knees. The implication is that injury to specific structures may have

important consequences in converting a previously asymptomatic, though perhaps abnormal, patellofemoral joint into one that is painful and/or unstable. These studies have been intended to improve the precision of surgical treatment for patellar instability, and their results are driving refinements in our surgical indications as well as technique.

N. Khan, M.D. (✉)
Southern California Permanente Medical Group,
San Marcos, CA, USA
e-mail: najeebkhan@mac.com

D.C. Fithian, M.D.
Southern California Permanente Medical Group,
El Cajon, CA, USA

E. Nomura, M.D.
Department of Orthopaedic Surgery,
International Goodwill Hospital,
Yokohama, Japan

Fig. 7.1 Macroscopic observation of the MPFL. When the VMO is reflected, the (a) MPFL can be seen. (b) With VMO resected, the full course of the MPFL is seen. Two pins are placed at the femoral attachment (From Nomura E, Fujikawa T, Takeda T, et al. Anatomical study of the medial patellofemoral ligament. Orthop Surg Suppl. 1992;22:2-5)

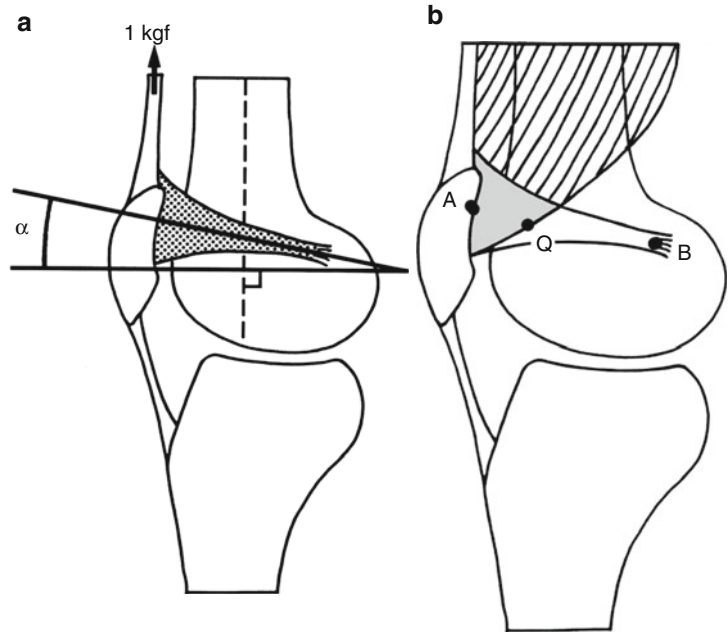
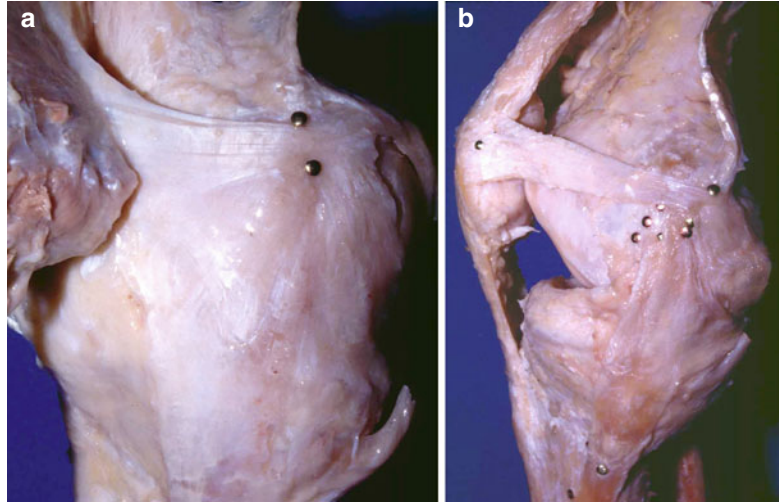


Fig. 7.2 (a) The axis of the MPFL deviates proximally from a line drawn perpendicular to the femoral axis. (b) The VMO tendon becomes confluent with the MPFL in the region from Q to A (From Nomura E, Fujikawa T, Takeda T, et al. Anatomical study of the medial patellofemoral ligament. Orthop Surg Suppl. 1992;22:2-5)

Fig. 7.3 The MPFL and VMO as seen from the perspective of the femur (From Nomura E, Fujikawa T, Takeda T, et al. Anatomical study of the medial patellofemoral ligament. *Orthop Surg Suppl.* 1992;22:2–5)



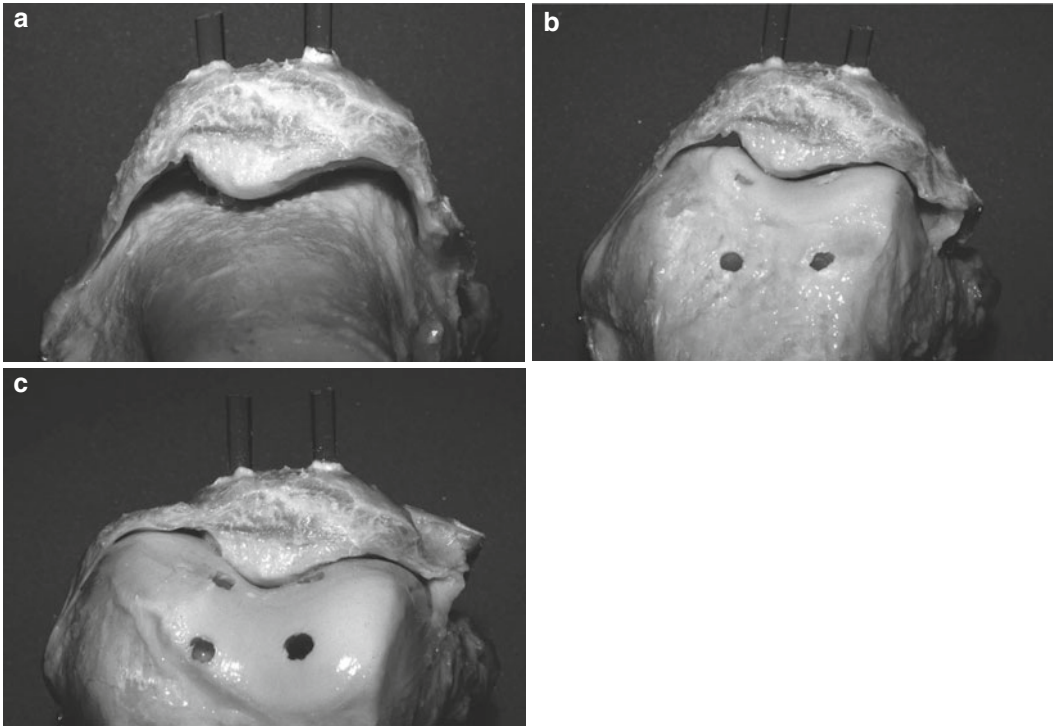


Fig. 7.4 Axial view of the patellofemoral articulation at (a) 0°, (b) 60°, and (c) 120° flexion with a 1-kg load applied to the quadriceps (From Nomura E, Horiuchi Y,

Kihara M. Medial patellofemoral ligament restraint in lateral patellar translation and reconstruction. *Knee*. 2000;7:121–7)

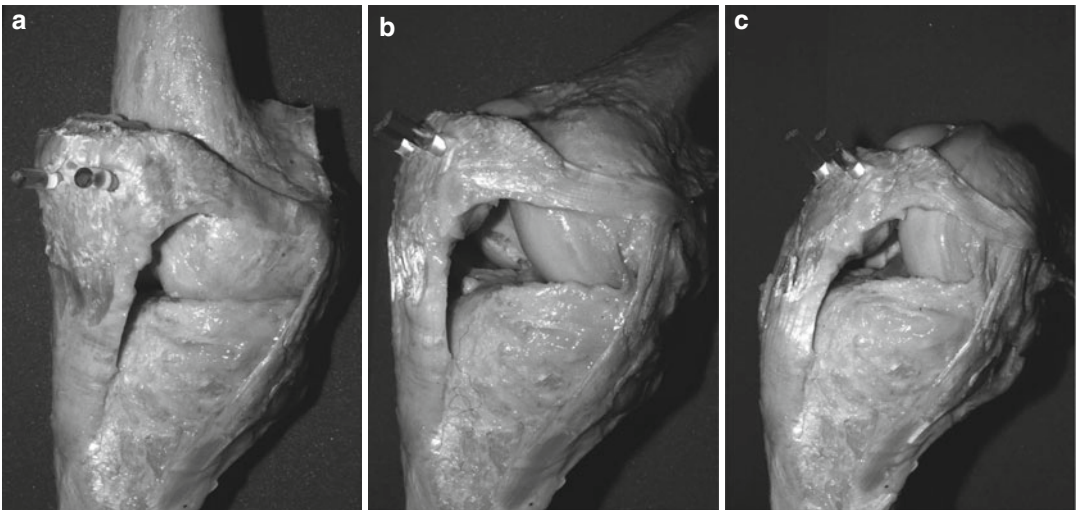


Fig. 7.5 Anterolateral view of the patellofemoral articulation at (a) 0°, (b) 60°, and (c) 120° flexion with a 1-kg load applied to the quadriceps (From Nomura E, Fujikawa

T, Takeda T, et al. Anatomical study of the medial patellofemoral ligament. *Orthop Surg Suppl*. 1992;22:2–5)

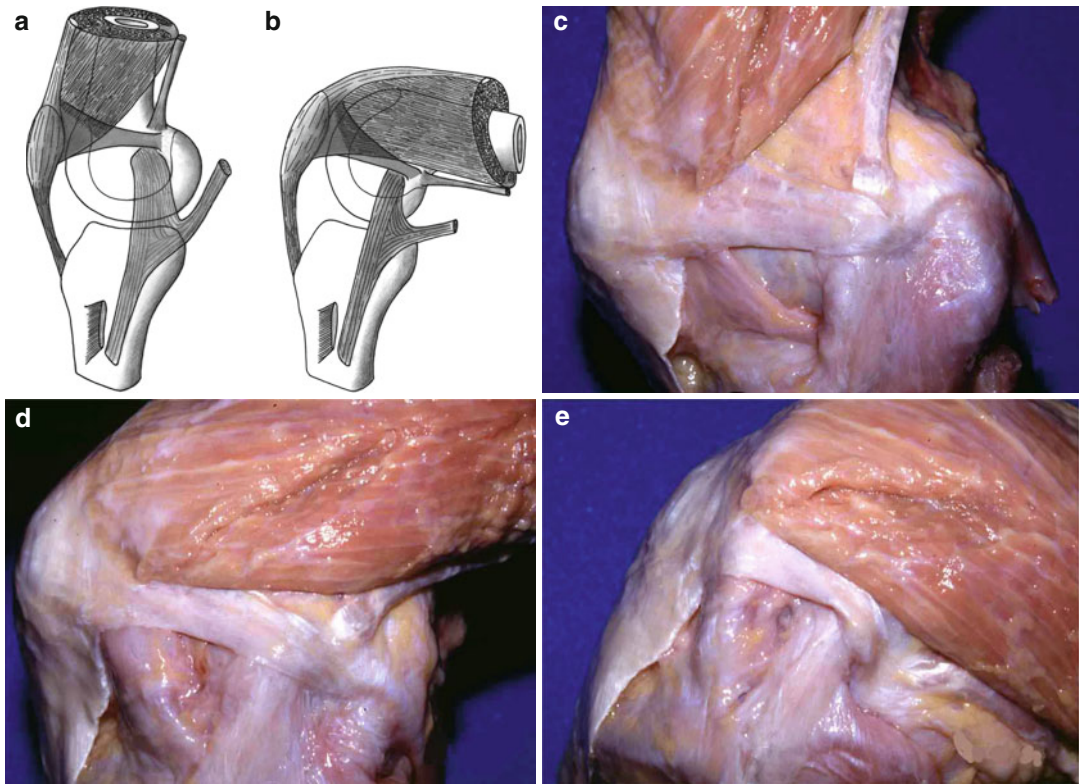


Fig. 7.6 (a) Schematic drawing of the relationships between the VMO and the MPFL. (b) The vastus medialis overlies the distal one-third of the MPFL (From Nomura E, Fujikawa T, Takeda T, et al. Anatomical study of the medial patellofemoral ligament. *Orthop Surg Suppl.*

1992;22:2-5). Its angle of pull relative to the MPFL fibers changes dramatically as the knee is flexed. (c) 0°, (d) 60°, (e) 120° (From Nomura E, Horiuchi Y, Kihara M. Medial patellofemoral ligament restraint in lateral patellar translation and reconstruction. *Knee.* 2000;7:121-7)



Fig. 7.7 Lateral radiograph taken after reduction of a lateral patellar dislocation. Note that even at the moderate degree of flexion shown, the inferior pole of the patella (as marked by the *arrow*) has barely entered the trochlear groove

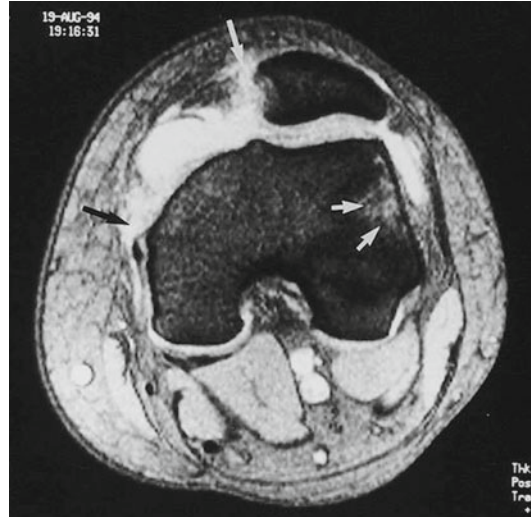


Fig. 7.8 Axial MR image following lateral patellar dislocation. Note complete discontinuity of signal in the area of the MPFL (indicative of complete rupture) both at the medial femoral epicondyle (*black arrow*) and at the medial border of the patella (*long white arrow*). Lateral condylar marrow edema is also seen (*short white arrows*)

Evaluation of the Patient with Anterior Knee Pain and Patellar Instability

8

Vicente Sanchis-Alfonso, Erik Montesinos-Berry,
Agustin Serrano, and Vicente Martínez-Sanjuan

There is no substitute for a thorough history and a complete and careful physical examination. The history and physical examination still remain the first step for making an accurate diagnosis of anterior knee pain and patellar instability above any technique of diagnostic image. Imaging studies are a second step and can never replace the

former. Surgical indications should not be based only on methods of image diagnosis as there is a poor correlation between clinical and image data. Finally, arthroscopy should be used judiciously and no realignment surgery should be based solely on the arthroscopic analysis of the patellofemoral congruence.

V. Sanchis-Alfonso, M.D., Ph.D. (✉)
International Patellofemoral Study Group,
ACL Study Group, Hospital 9 de Octubre,
Hospital Arnau de Vilanova, School of Medicine,
Valencia Catholic University, Valencia, Spain
e-mail: vicente.sanchis.alfonso@gmail.com

E. Montesinos-Berry, M.D. • A. Serrano, M.D.
Hospital de Manises, Valencia, Spain

V. Martínez-Sanjuan, M.D., Ph.D.
MR and CT Unit,
ERESA-Hospital General Universitario,
Valencia, Spain



Fig. 8.1 The most important diagnostic tool is the finger. The location of pain is extremely helpful to make the diagnosis and to plan the treatment. Lateral X-ray of a patient who underwent a Maquet procedure that shows an asymptomatic patellofemoral osteoarthritis. He suffered from highly localized pain on the lytic lesion he had of the proximal tibia (*arrow*). Curettage and bone grafting of the lesion solved his anterior knee pain

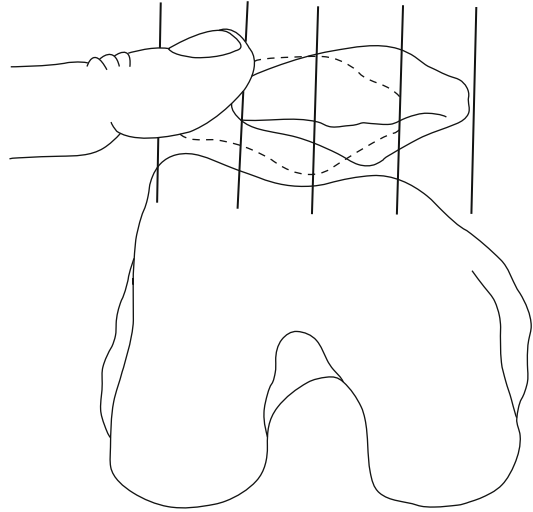


Fig. 8.2 Patellar glide test. The patellofemoral joint is mentally divided into quadrants and patellar mobility is assessed in both directions

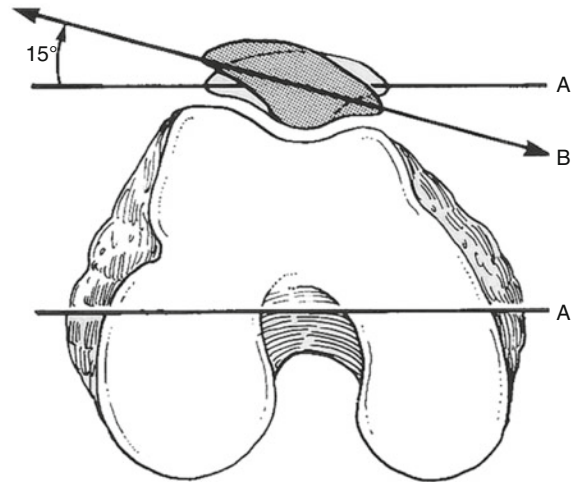
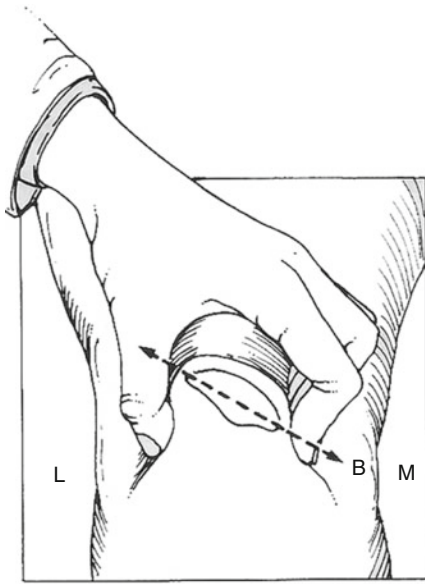


Fig. 8.3 Patellar tilt test (Reprinted from Scuderi GR, ed. *The Patella*. New York: Springer; 1995, p. 79. With permission from Springer-Verlag)



Fig. 8.4 Axial compression patellar test. We compress the patella against the trochlea with the palm of the hand (black arrow)



Fig. 8.5 Palpation on the distal pole of the patella and the proximal patellar tendon



Fig. 8.6 Patellar apprehension test

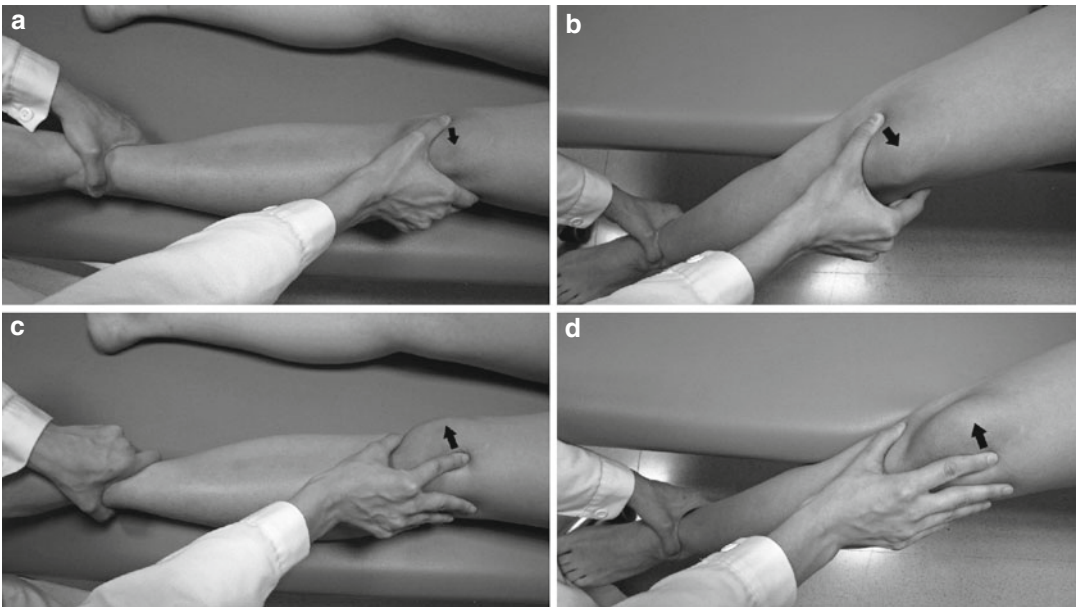


Fig. 8.7 The moving patellar apprehension test begins with the knee held in full extension and the patella is manually translated laterally (*black arrow*) with the thumb (**a**). The knee is then flexed to 90° and then brought back to full extension while the lateral force on the patella (*black arrow*) is maintained (**b**). In the second part of the test, the knee is started in full extension (**c**), brought to 90° of

flexion (**d**), and then back to full extension while the index finger is used to translate the patella medially (*black arrow*). For a positive test, in the first part, the patient expresses apprehension and may activate his or her quadriceps in response to apprehension. However, in the second part of the test, the patient experiences no apprehension and allows free flexion and extension of the knee



Fig. 8.8 Patellar glide test in a patient with multidirectional instability. Pathologic lateral displacement of the patella (a). Contralateral asymptomatic knee (b). We have seen an image (a) similar to the sulcus sign observed in patients with multidirectional instability of the shoulder (c)

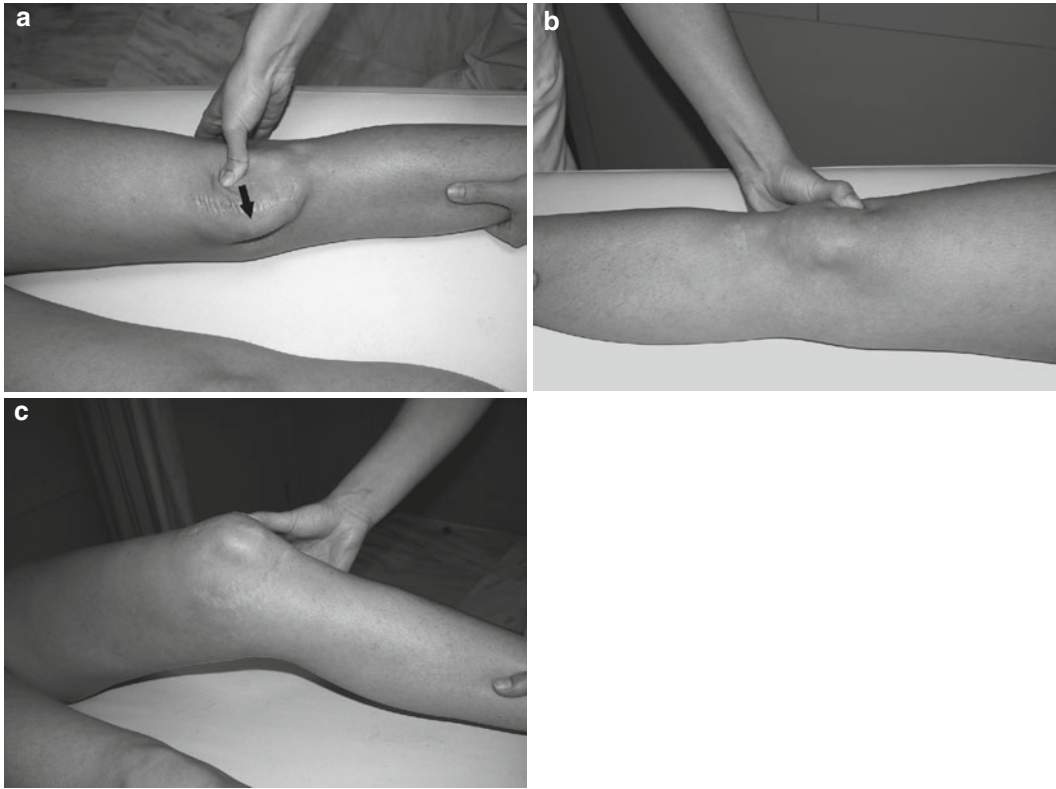


Fig. 8.9 Fulkerson's relocation test. We hold the patella slightly in a medial direction (*black arrow*) with the knee extended (**a**). Contralateral asymptomatic knee (**b**). Then, we flex the knee while letting go the patella, which causes the patella to go into the femoral trochlea (**c**)



Fig. 8.10 Evaluation of quadriceps flexibility



Fig. 8.12 Evaluation of gastrocnemius flexibility



Fig. 8.11 Evaluation of hamstrings flexibility

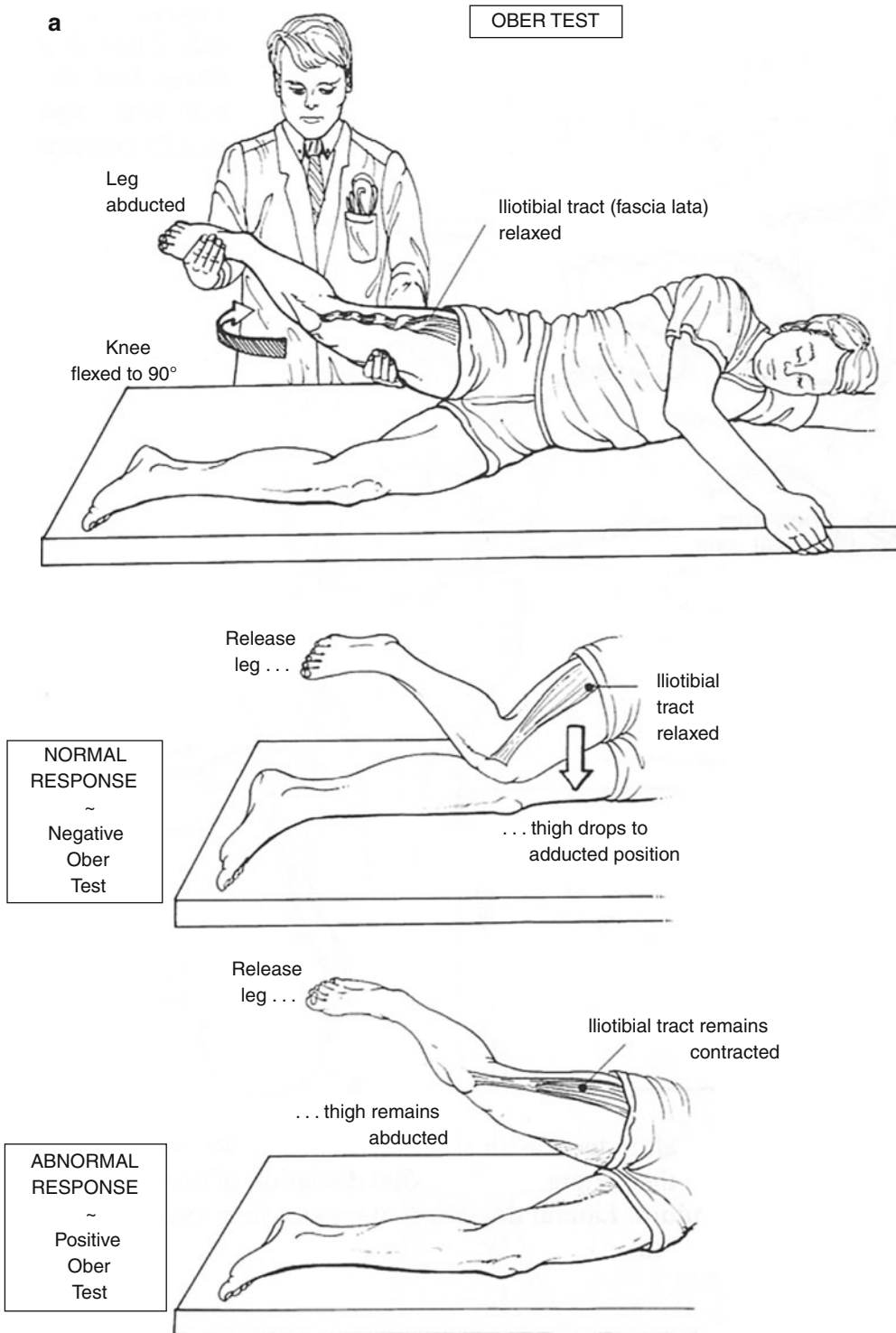


Fig. 8.13 (a) Ober's test (Reprinted from Scuderi GR, ed. The Patella. New York: Springer; 1995, p. 80. With permission from Springer-Verlag). (b, c) Positive test

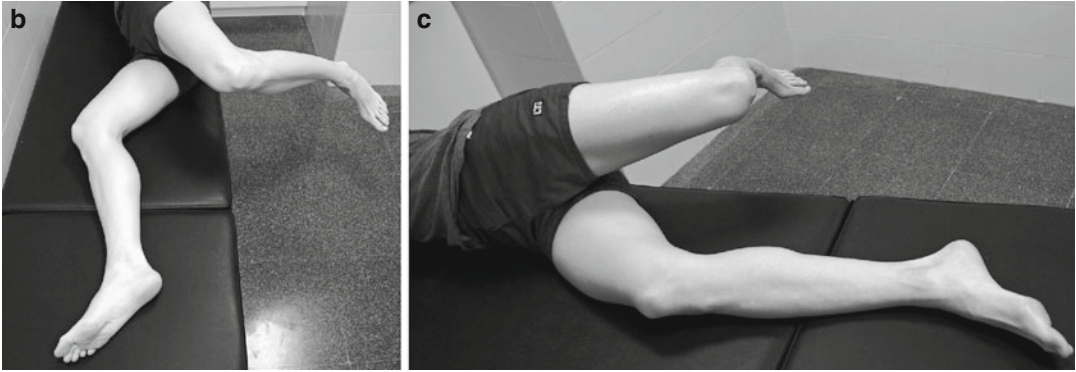


Fig.8.13 (continued)



Fig. 8.14 Skin laxity in Ehlers–Danlos syndrome

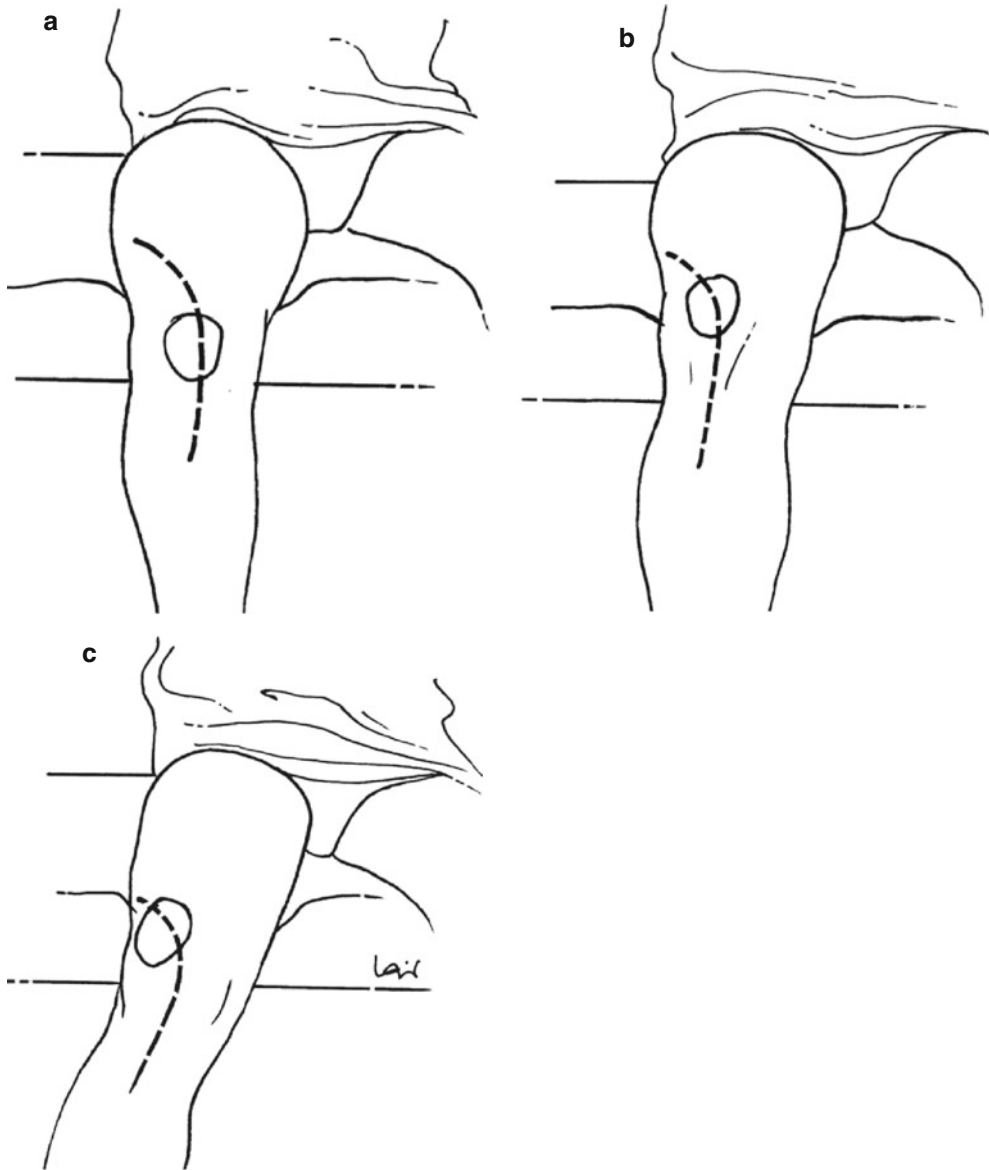


Fig. 8.15 The “J” sign. When the knee is extended from 90° (a) to 0° (c) the patella describes an inverted J-shaped course. Intermediate position between 90° and 0° (b)



Fig. 8.16 Pronated foot



Fig. 8.17 Increased femoral anteversion. (a) Patient sitting down. (b) Patient in supine position (c) Patient in prone position



Fig. 8.18 Evaluation of hip flexion contracture by flexing the contralateral hip completely. If the ipsilateral hip cannot lie flat on the table, hip flexion contracture is present

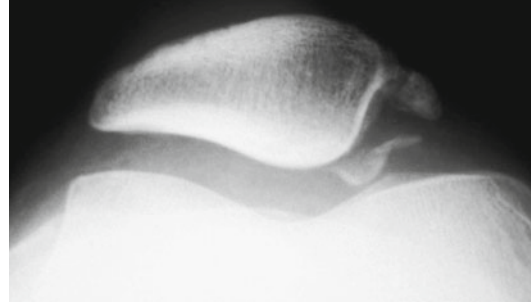


Fig. 8.20 Merchant axial view, where two bony fragments are seen at the patellar medial border, sequelae from former dislocation episodes



Fig. 8.19 True lateral radiograph

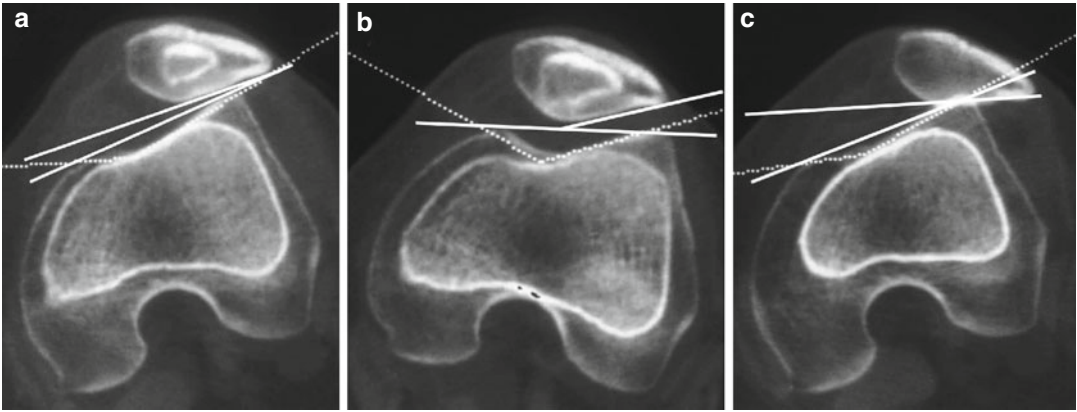


Fig. 8.21 An 18-year-old woman, referred for anterior knee pain and patellar instability of her left knee with repeated hemarthrosis and severe giving-way with falling to the ground with activities of daily living. Conventional radiographs were normal and the patella was seen well-centered in the axial view of Merchant. CT shows PFM type 2 (a) with the patella relocated into the femoral tro-

chlea at 30° (b). With the contraction of the quadriceps subluxation and tilt increases (c) (Reproduced from Sanchis-Alfonso V, Roselló-Sastre E, Martínez-SanJuan V. Pathogenesis of anterior knee pain syndrome and functional patellofemoral instability in the active young. A review. *Am J Knee Surg.* 1999;12:29–40. Reprinted by permission from Thieme)

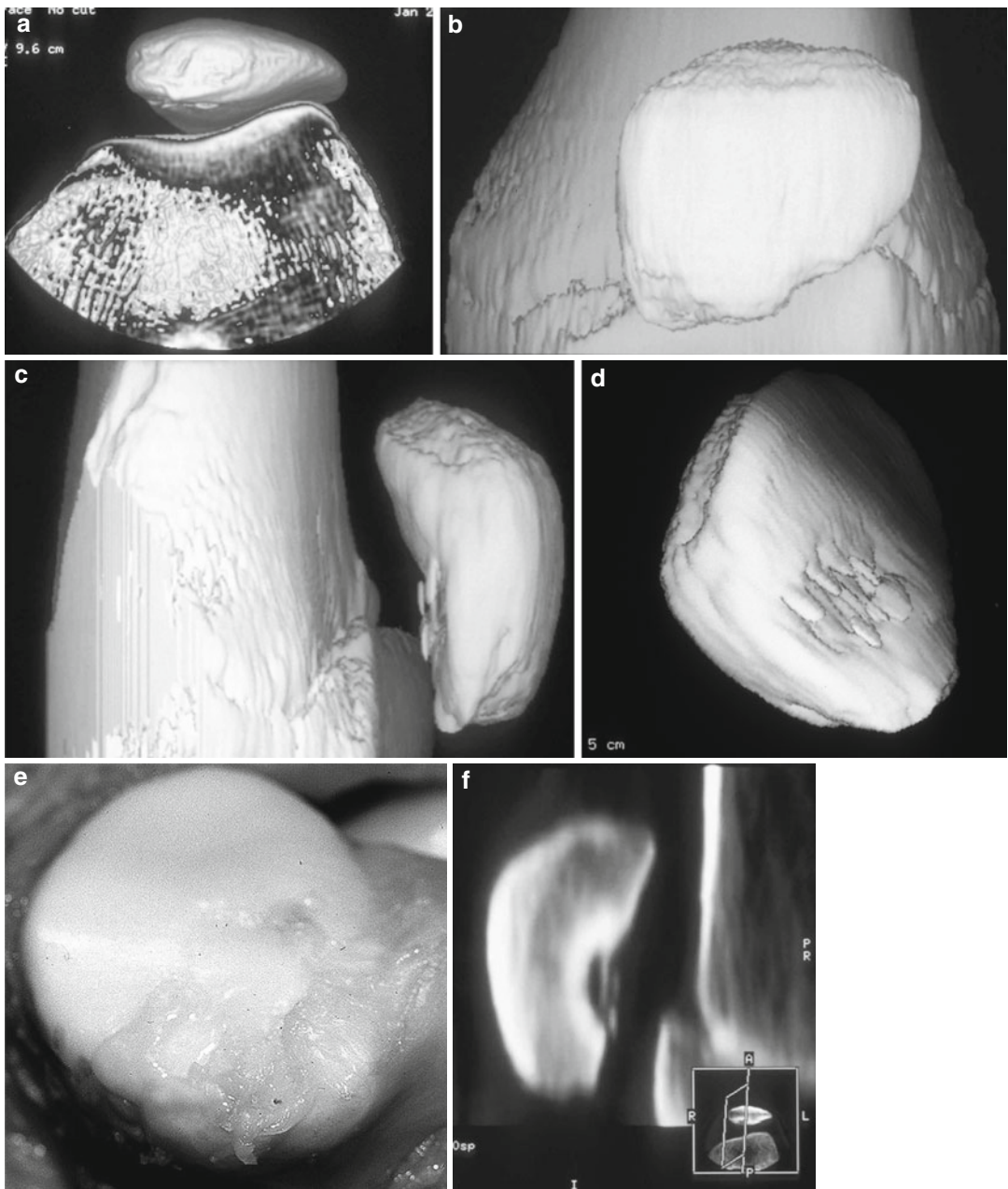


Fig. 8.22 3D-CT reconstruction of the patellofemoral joint. Axial plane showing degenerative changes of the articular cartilage of the medial patellar facet (a), frontal plane (b), and sagittal plane (c). 3D-CT shows great fidelity of the surface anatomy including size and location

of the chondral lesion (d, e), although it is unable to show undersurface detail, which is clearly shown by conventional CT scans (f) or by MRI (g) (Sagittal SE T1W MR image)

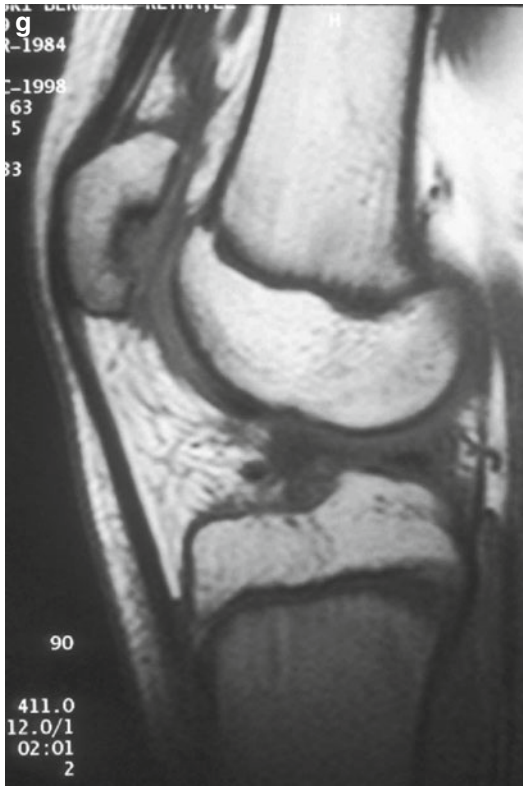


Fig. 8.22 (continued)

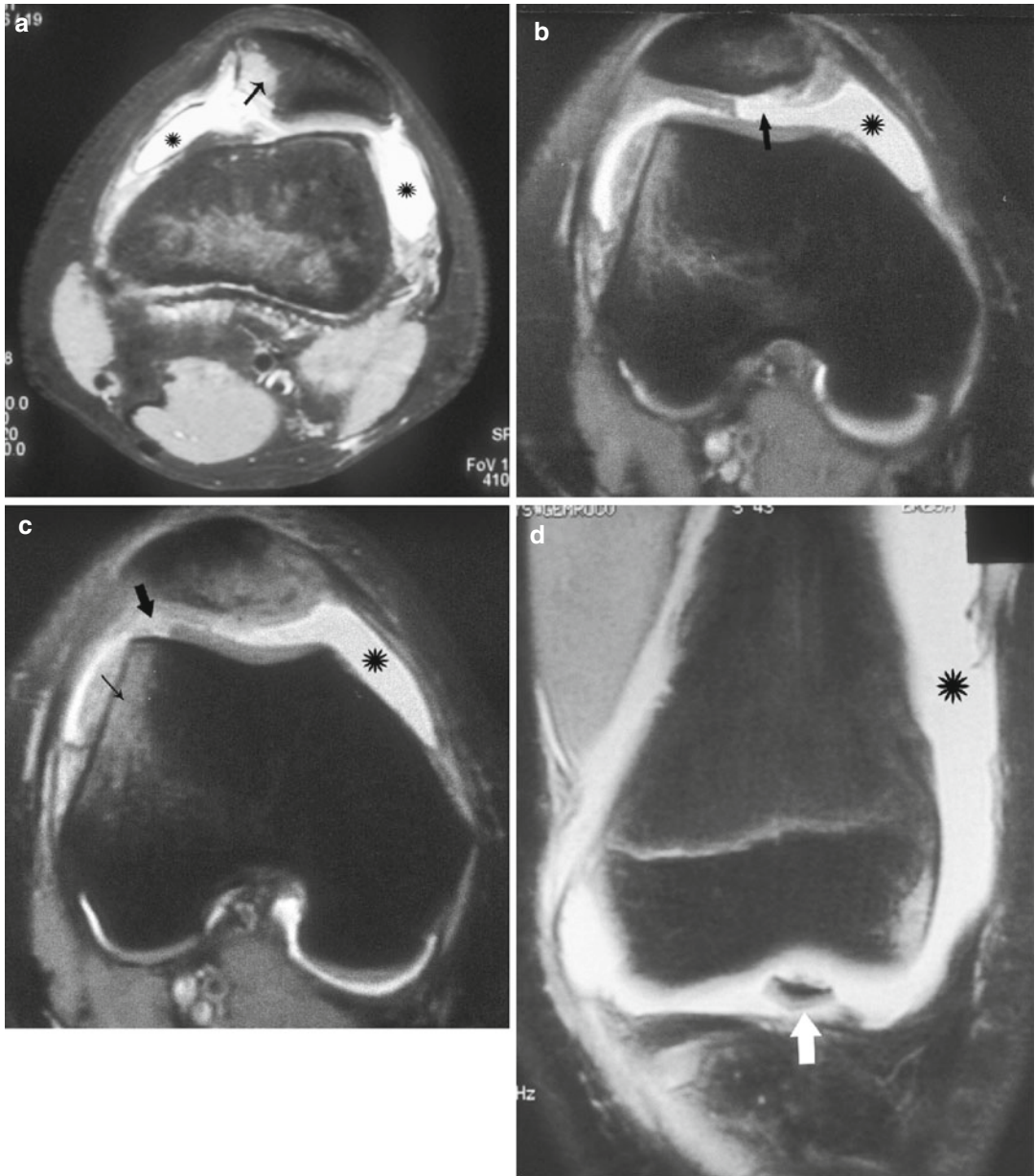


Fig. 8.23 MRI signs of acute lateral patellar dislocation: contusions of the anterior portion of the lateral femoral condyle and of the medial patellae (*black thin arrow*), osteochondral defects (*black thick arrow*), intra-articular bodies (*white thick arrow*), and joint effusions (*asterisk*).

(a) Axial FSE PDW Fat Sat MR image. (b, c) Axial FSE PDW Fat Sat MR images. (d) Coronal FSE PDW Fat Sat MR image

Influence of Psychological Factors on Pain and Disability in Anterior Knee Pain Patients

9

Julio Doménech, Vicente Sanchis-Alfonso,
and Begoña Espejo

AKP patients express chronic pain but also disability. However, the correlation between pain and disability is not complete and linear. Some patients with a lot of pain show mild disability while others with much less pain also show great disability. The disability is profoundly influenced by other emotional and cognitive factors that are associated with the perception of pain. Therefore, the clinical efforts do not have to be focused only

on treating the pain as a feeling but on identifying and modifying these factors.

Can AKP have a psychological cause as it has been classically believed? We think not. There is a structural lesion that causes pain and disability although sometimes it may not be found. The psychological factors modulate the course of the disease but are not the cause. Even so, psychological factors are of utmost importance, which is why it is essential that physicians be aware of them.

Even if the importance of emotion in pain and disability in AKP patients seems clear, disorders in negative emotions (anxiety and depression) are also sources of suffering in these patients and deserve to be treated independently from other measures taken in the course of the condition.

In many AKP patients the classic biomedical approach has failed to provide adequate treatment despite decades of research. Given that the biopsychosocial model provides a better understanding of articular pain and has contributed to the improved treatment of other musculoskeletal conditions, it seems reasonable to think that a biopsychosocial approach would provide a useful tool for the conventional medical treatment of AKP. It is therefore interesting to contemplate cognitive-behavioral treatment models as another therapeutic option to help these patients.

J. Doménech, M.D., Ph.D. (✉)
Department of Orthopaedic Surgery,
School of Medicine, Catholic University of Valencia,
Valencia, Spain

Facultad de Ciencias de la Salud,
Universidad Cardenal Herrera – CEU,
Valencia, Spain
e-mail: julio.domenech@ucv.es

V. Sanchis-Alfonso, M.D., Ph.D.
International Patellofemoral Study Group,
ACL Study Group, Hospital 9 de Octubre,
Hospital Arnau de Vilanova,
School of Medicine, Valencia Catholic University,
Valencia, Spain

B. Espejo
Department of Methodology of the Behavioural
Sciences, University of Valencia,
Valencia, Spain

Fig. 9.1 The Biopsychosocial model of chronic pain and disability. ICF International Classification of Functioning Disability and Health, WHO World Health Organization (Modified from Waddell G. The back pain revolution. 2nd ed. London: Churchill-Livingston; 2004)

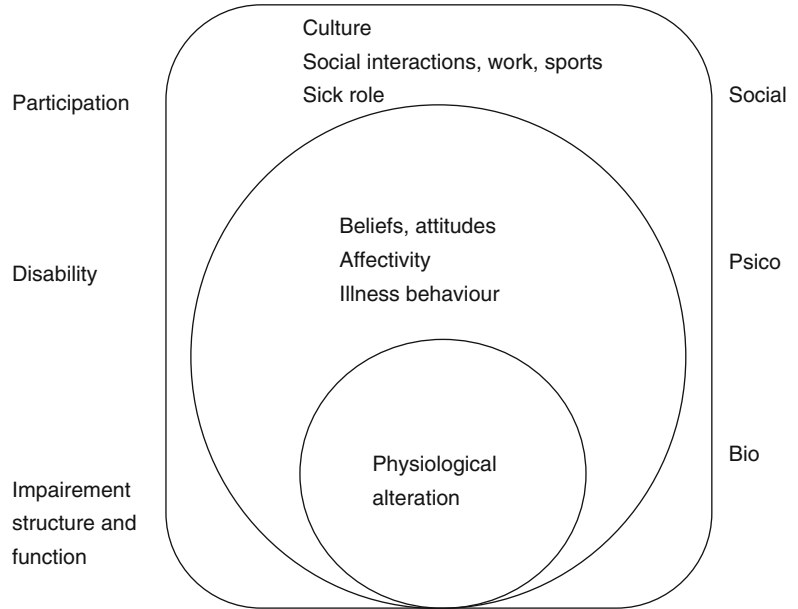
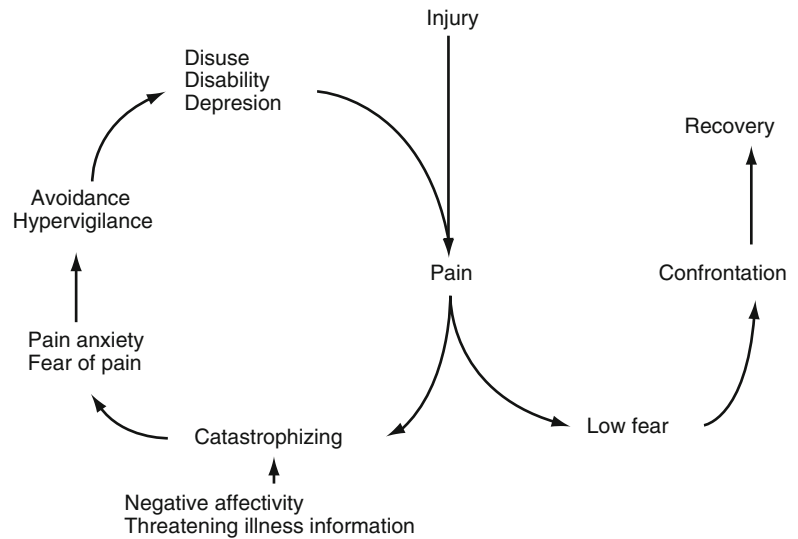


Fig. 9.2 The fear-avoidance model of chronic pain based on the fear-avoidance model of Vlaeyen and Linton (Vlaeyen JWS, Linton SJ. Fear avoidance and its consequences in chronic musculoskeletal pain: a state of the art. Pain. 2000;85:317–32) and the fear-anxiety-avoidance model of Asmundson et al. (Asmundson GJ, Norton PJ, Vlaeyen JWS. Fear avoidance models of chronic pain: an overview. In: Asmundson GJ, Vlaeyen JWS, Crombez G, editors. Understanding and treating fear of pain. Oxford: Oxford University Press; 2004:3–24)



Vicente Sanchis-Alfonso, Erik Montesinos-Berry,
Francisco Aparisi-Rodriguez,
and Vicente Belloch-Ugarte

Anterior knee pain is a common symptom, which may have a large variety of causes. Although, patellofemoral malalignment (PFM) is a potential cause of anterior knee pain in young patients, not all malalignments are symptomatic. To think of anterior knee pain as somehow being necessarily tied to PFM is an oversimplification that has positively stultified progress towards better diagnosis and treatment of patients with anterior knee pain syndrome. PFM could be the single culprit for the pain but it is also possible that it bears no relation whatsoever with the patient's complaint or that it is only partly to blame for the problem. PFM can exist without anterior knee pain,

and anterior knee pain can exist without PFM. There are many causes of anterior knee pain, some of them related to PFM and many more not related to PFM. Likewise, we should bear in mind that there are teenage patients with anterior knee pain who lack evidence of organic pathology (i.e., their condition is of a psychosomatic nature [21]) and also patients who suffer from the "malingering syndrome". In this chapter we analyze uncommon causes of anterior knee pain, emphasizing the fact that not all malalignments are symptomatic.

The question to be addressed is, therefore, what factor is responsible for the patient's symptoms? As with any other pathology, it is necessary to make an accurate diagnosis before embarking on a specific treatment plan. An incorrect diagnosis may lead to inappropriate or unnecessary surgical procedures, which can cause morbidity and unnecessary expenses. Moreover, an unsuitable treatment, resulting from an incorrect diagnosis, may worsen the situation. The final result could be disastrous since it may add to an already serious condition a reflex sympathetic dystrophy or an iatrogenic medial dislocation of the patella.

The goal of an orthopedic surgeon treating patients with anterior knee pain is to precisely determine the etiology of the pain since this is the only way to come up with a "tailored treatment".

V. Sanchis-Alfonso, M.D., Ph.D. (✉)
International Patellofemoral Study Group,
ACL Study Group, Hospital 9 de Octubre,
Hospital Arnau de Vilanova, School of Medicine,
Valencia Catholic University,
Valencia, Spain
e-mail: vicente.sanchis.alfonso@gmail.com

E. Montesinos-Berry, M.D.
Hospital de Manises,
Valencia, Spain

F. Aparisi-Rodriguez, M.D., Ph.D.
Department of Radiology,
Hospital Universitario La Fe,
Valencia, Spain

V. Belloch-Ugarte
Radiologist Clinica Eresa,
Hospital Universitario La Fe,
Valencia, Spain



Fig. 10.1 Osteochondritis dissecans of the patellofemoral groove in a patient with symptomatic PFM (a–c). The MRI shows the chondral lesion healed a year and a half after realignment surgery (d, e) (b–e – GrE T2* MR images)

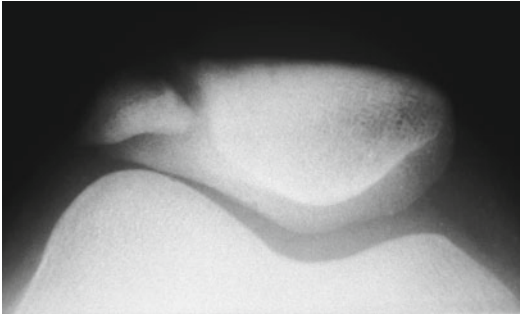


Fig. 10.2 Bipartite patella of a volleyball player with excessive lateral pressure syndrome

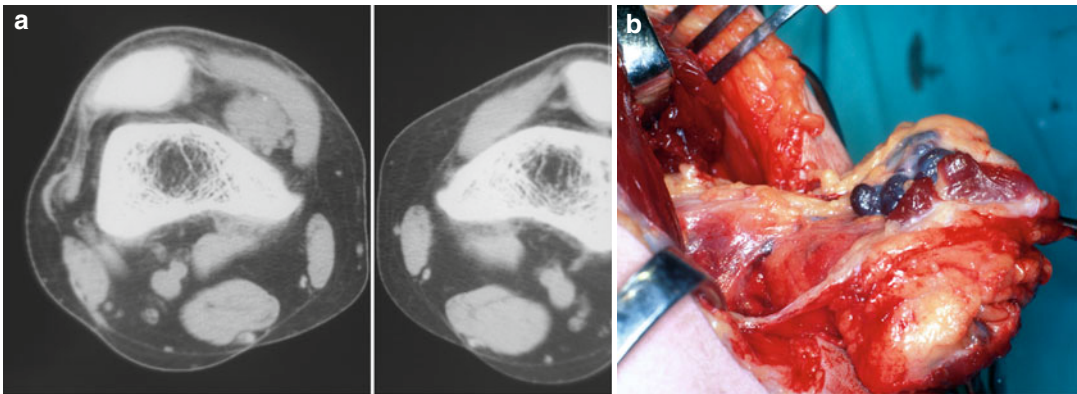


Fig. 10.3 Intramuscular hemangioma of the vastus medialis obliquus muscle (a) CT image (b) Macroscopic appearance (Reproduced from Sanchis-Alfonso V,

Fernandez CI, Sanchez C, et al. Hemangioma intramuscular (Aportación de 6 casos y revisión de la literatura). *Rev Esp de Cir Ost.* 1990;25:367–78. With permission)

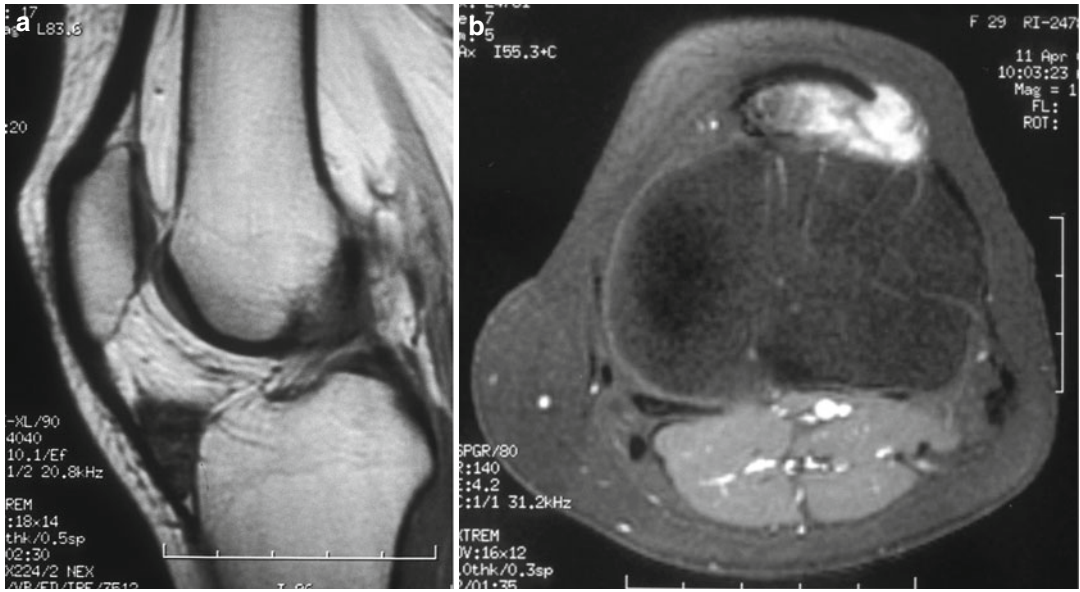


Fig. 10.4 Localized pigmented villonodular synovitis of the Hoffa's fat pad. (a) Sagittal FSE T1W MR image. Hypointense lesion in the Hoffa's Fat Pad. (b) Axial GrE T1W + gd-DTPA. Heterogeneous enhancement lesion in the Hoffa's Fat Pad

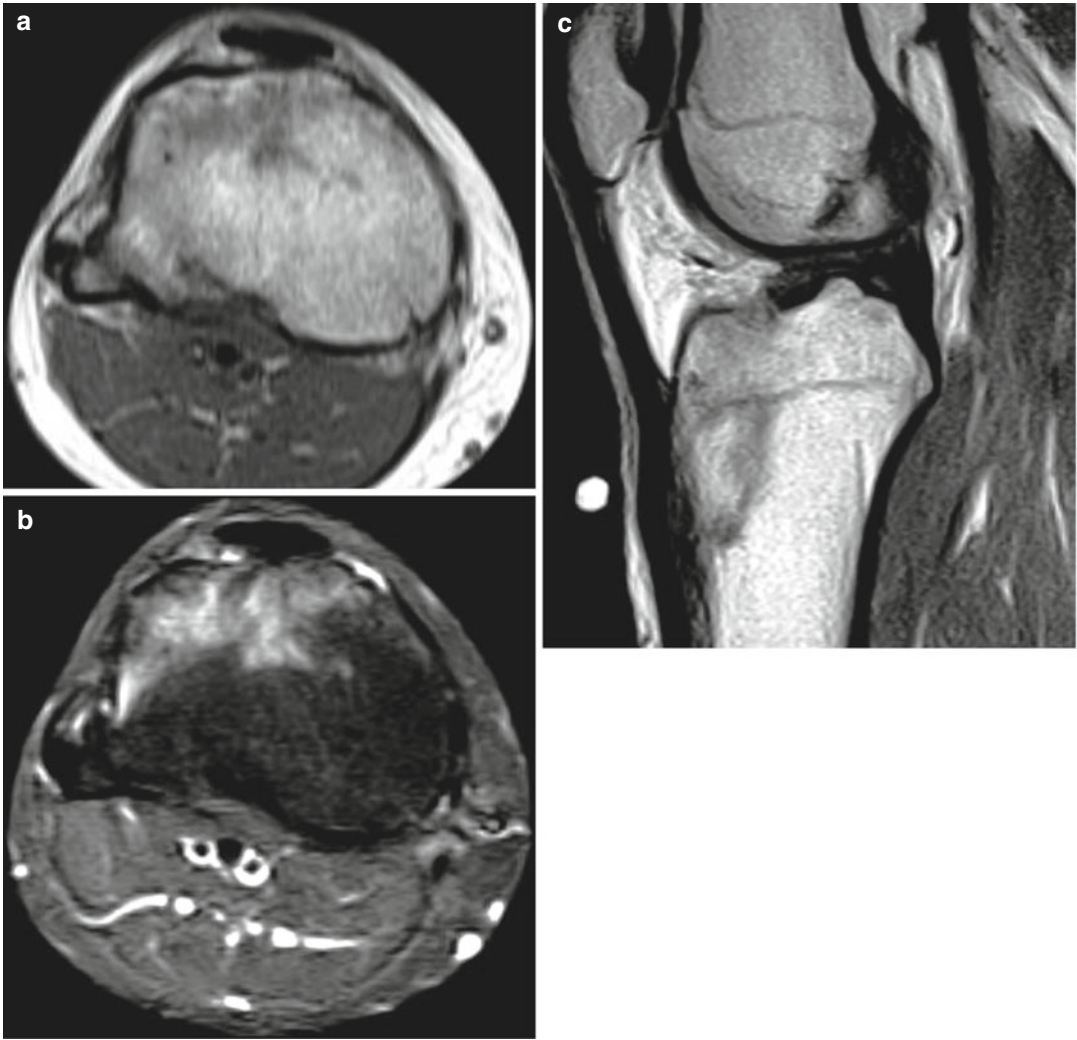


Fig. 10.5 A subperiosteal osteoid osteoma on the anterior aspect of the proximal end of the tibia is an extremely rare cause of anterior knee pain. Conventional x-rays were negative. Axial T1-weighted MR image (a). Axial

T2-weighted MR image (with fat suppression) (b). Note a well-defined edematous area without significant extraosseous involvement. Sagittal T1-weighted MR image (c)

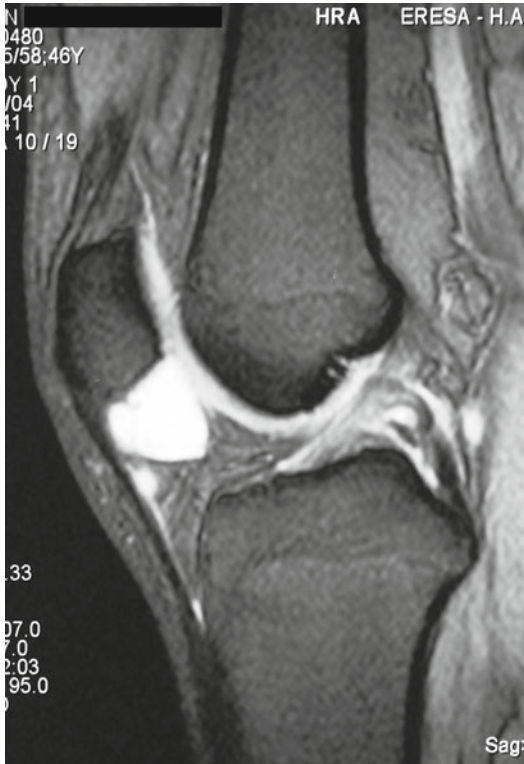


Fig. 10.6 Sagittal FSE PDW Fat Sat MRI showing an intra-articular ganglion cyst in the Hoffa's fat pad

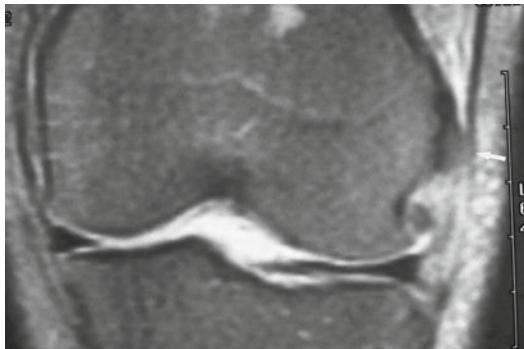


Fig. 10.7 Coronal FSE PDW Fat Sat MRI. Iliotibial friction band syndrome in a female surfer. Note the bone exostosis of the lateral femoral condyle (*arrow*), which leads to an impingement on the iliotibial tract

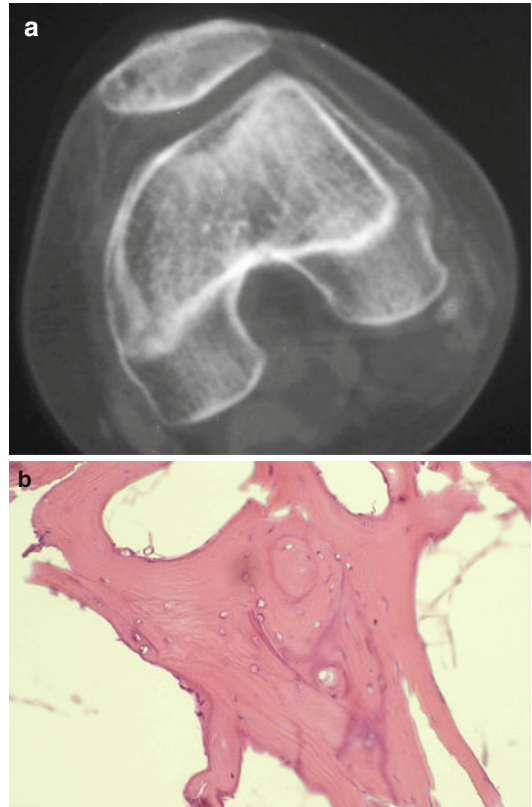


Fig. 10.8 Localized osteonecrosis of the patella (a) CT image. (b) microscopic appearance (Reproduced from Sanchis-Alfonso V, Roselló-Sastre E, Martínez-SanJuan V, et al. Occult localized osteonecrosis of the patella. Case report. Am J Knee Surg. 1997;10:166–70. Reprinted by permission from Thieme)

Fig. 10.9 Stress fracture in the proximal tibia in a patient who consulted for anterior knee pain without traumatism
(a) Anteroposterior view.
(b) Lateral view

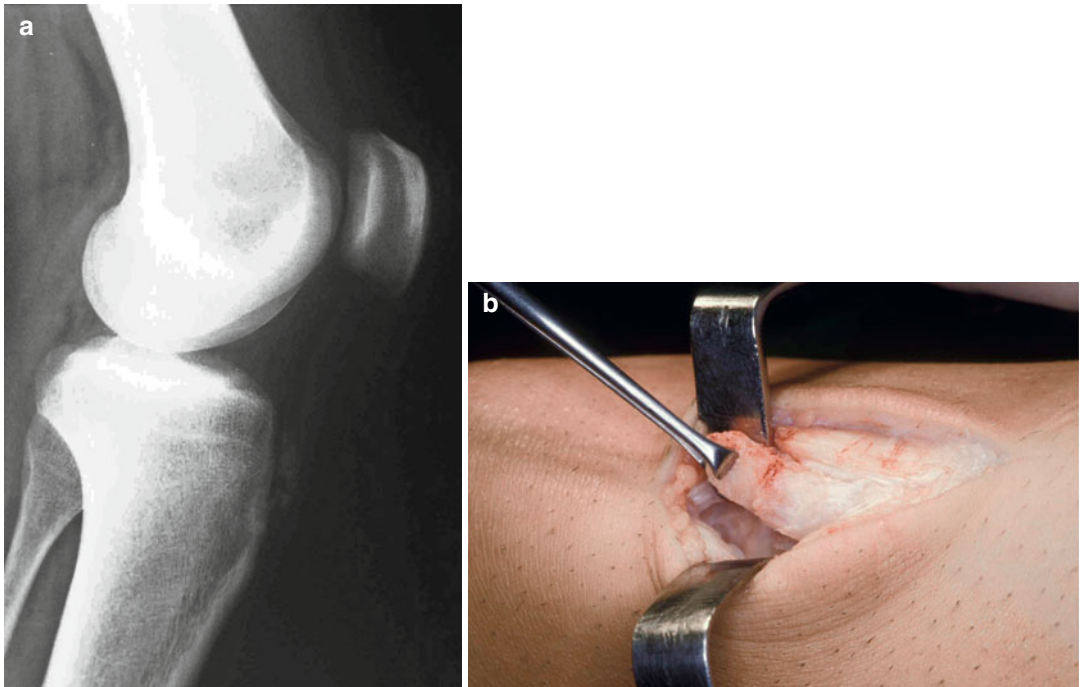
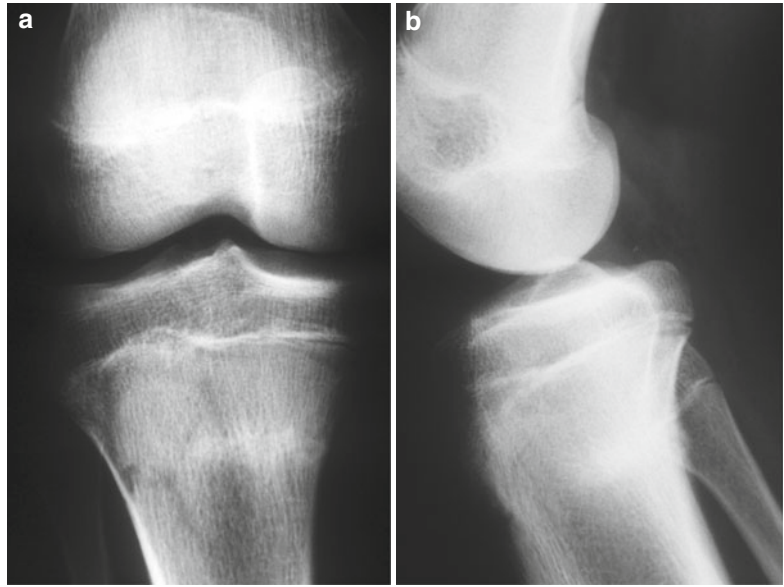


Fig. 10.10 This is a patient who presented with swelling and pain in the anterior tibial tubercle. Lateral x-ray showing ossicles in the anterior tibial tubercle (a). Excision of the ossicles via a transtendinous approach (b)

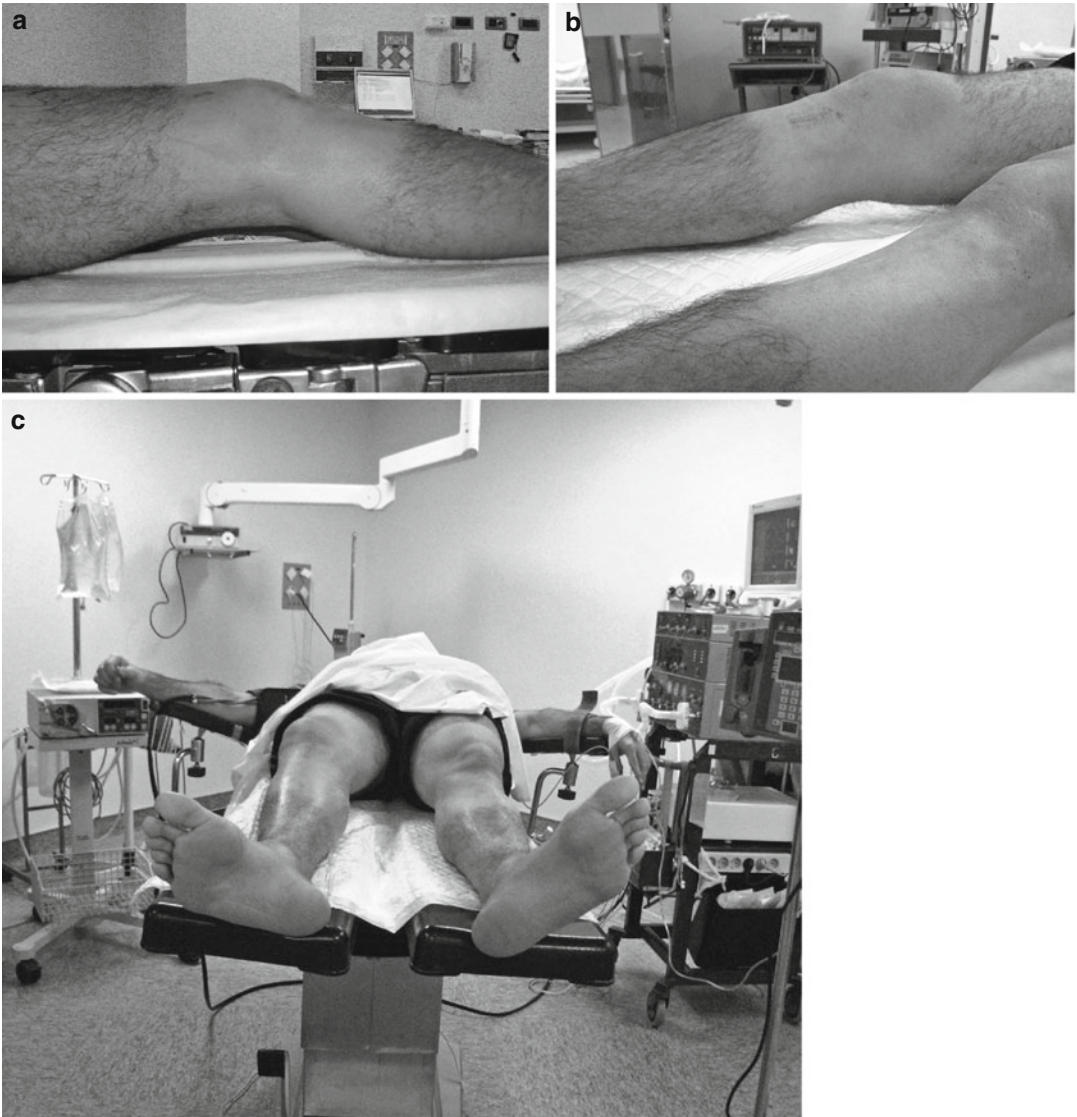


Fig. 10.11 Anterior knee pain after ACL reconstruction with autogenous hamstrings tendons 11 months ago. (a, b) Lack of normal knee extension can be observed (bent knee). Patients who favor a knee by keeping it bent can be

identified easily because they will have one leg and foot rotated externally (c). Chondral lesion of the trochlea (d). Cyclops syndrome (e, f)

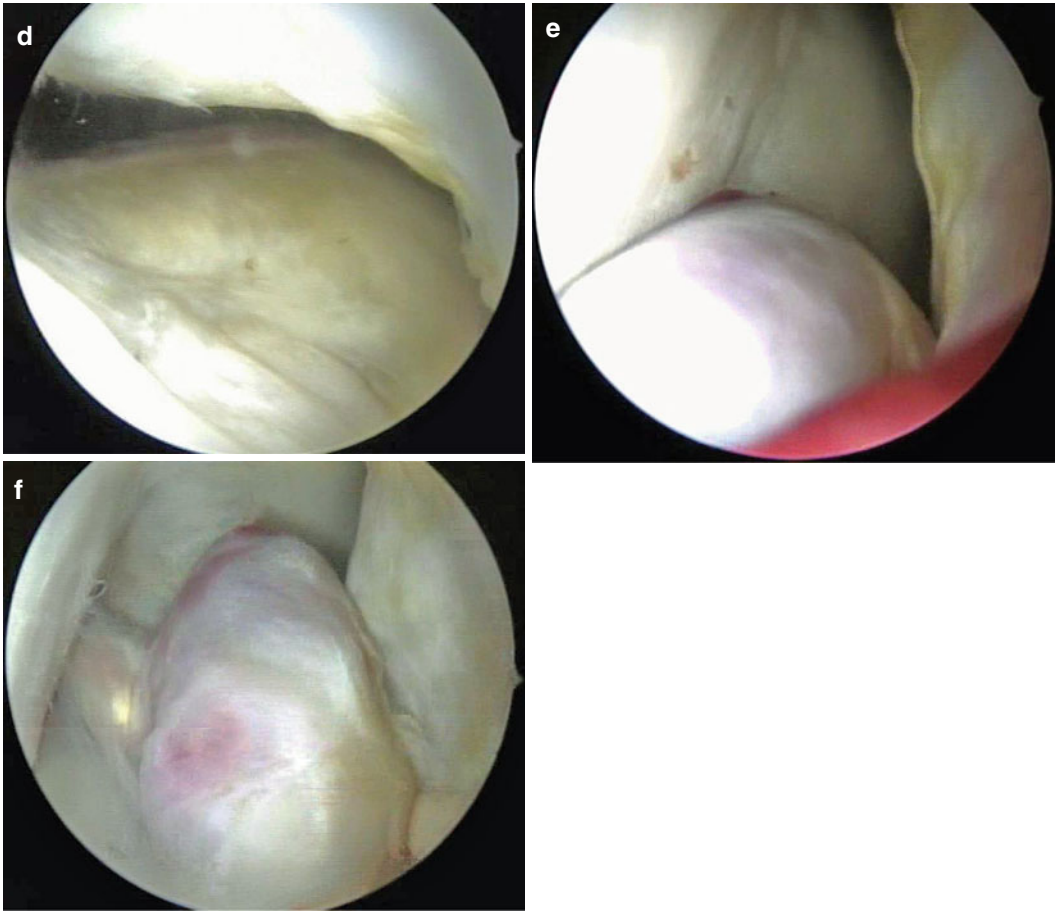


Fig. 10.11 (continued)

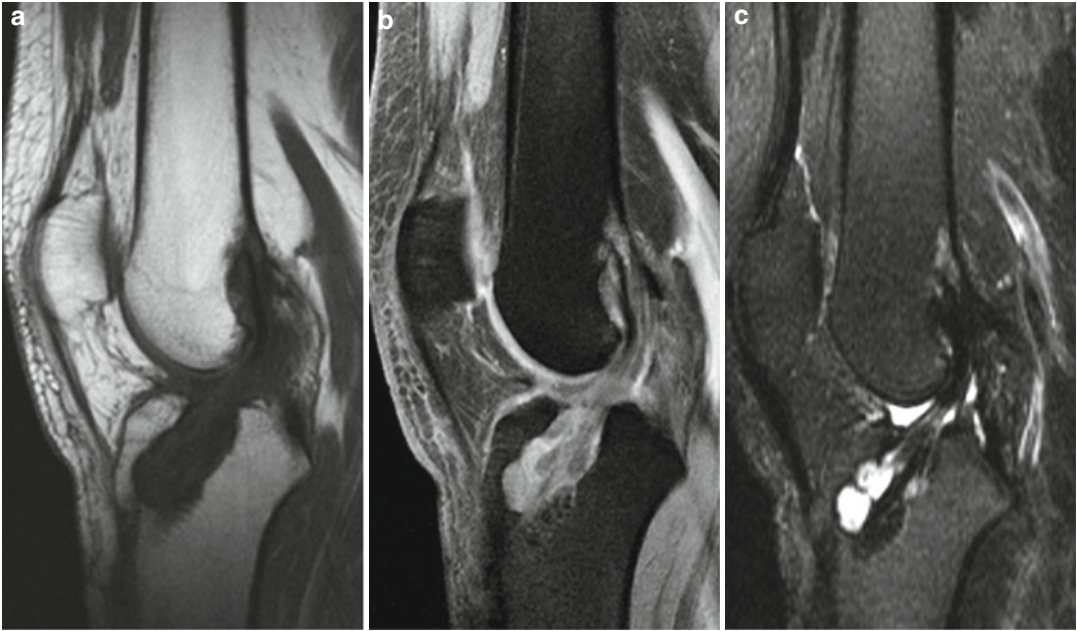


Fig. 10.12 A 38-year-old male presented with a history of continuous chronic anterior left knee pain with daily living activities refractory to conservative treatment. The patient underwent an endoscopic ACL reconstruction 5 years before using autogenous hamstrings tendons fixed with bioabsorbable polylactic acid interference screws. An MRI showing an osteolytic tibial cyst in the tibial tunnel. **(a)** Sagittal FSE T1 weighted. A hypointense

lesion is seen at the tibial tunnel. **(b)** Sagittal fat sat FSE T1 weighted following administration of paramagnetic contrast medium. There is an irregular enhancement at the wall related to the fibrous and inflammatory component of the cavity. The cyst shows no enhancement and corresponds to the central part of the cavity. **(c)** Sagittal fat sat FSE T2 weighted. The cyst is shown as a hyperintense image. The graft shows no abnormalities

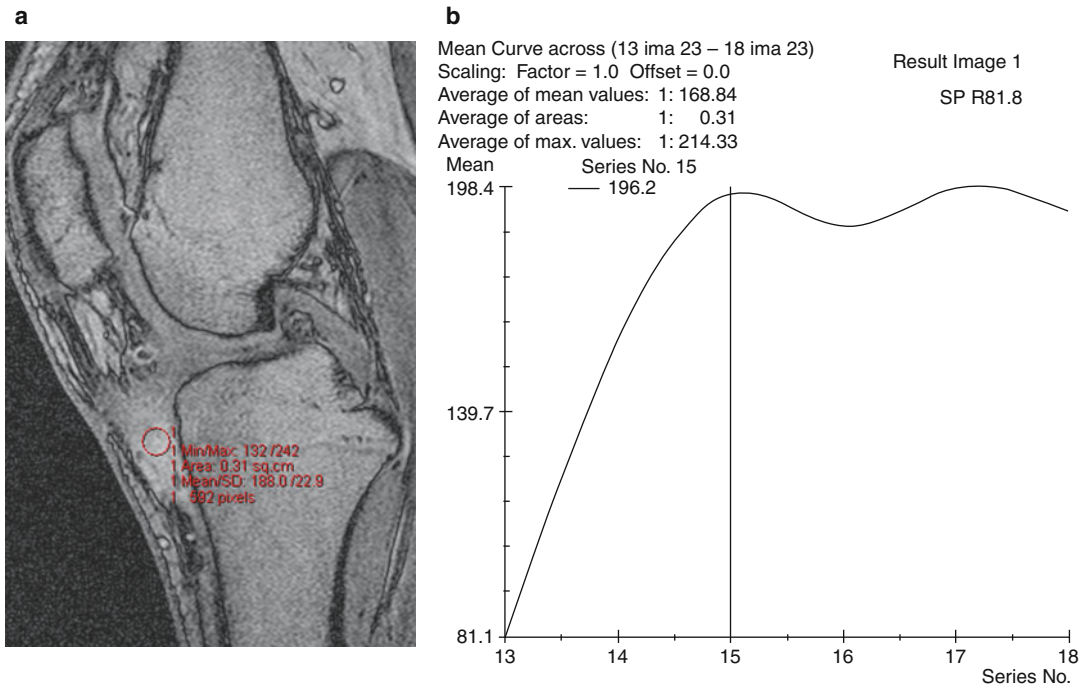


Fig. 10.13 Extraskelatal osteosarcoma of the infrapatellar Hoffa’s fat pad. (a) Dynamic study after in bolus paramagnetic contrast administration. 3D sagittal T1-weighted echo sequence, 6 acquisitions, 20 s duration for each one. Contrast uptaking soft tissue tumor adjacent to the anterior tibial surface. The ROI is marked (red circle) for contrast uptake curve analysis. (b) Graphic representation of the paramagnetic contrast uptake curve timing. The uptake

intensity is represented on the vertical axis, and the series, equivalent to time units (20 s per series) is represented on the horizontal axis. The curve indicates the biological aggressive behavior of the tumor: it has a high growth slope in the initial series (13, 14, and 15), corresponding to the first minutes, reaching stable intensity values (“plateau”) in the intermediate and last series (16, 17, and 18)

Fig. 10.14 Nonspecific chronic synovitis of the popliteal aspect of the right knee. Axial CT scan at 0° of knee flexion (a). Sagittal GrE T2* MR images (b, c)

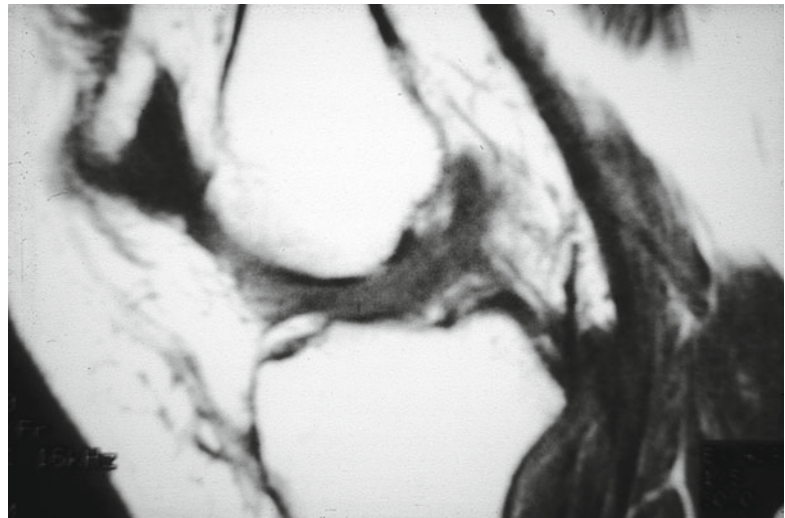
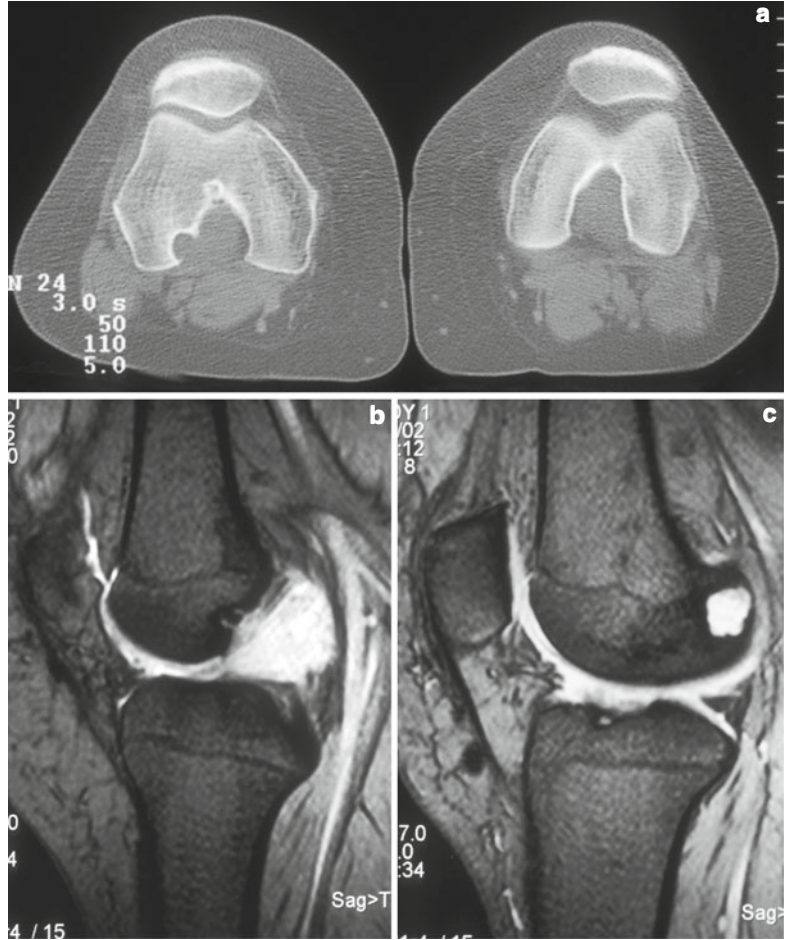
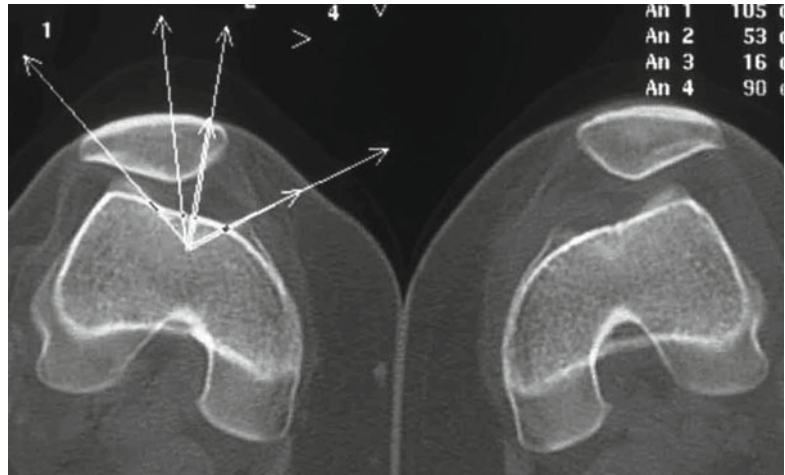


Fig. 10.15 Sagittal SE T1W MR image. False positive detection of an ACL tear

Fig. 10.16 Asymptomatic bilateral PFM. CT scan with the knees at 0° of flexion with a relaxed quadriceps. The patient's actual problem was a chronic rupture of the ACL and a bucket-handle tear of the medial meniscus. The result of the physical examination of the extensor mechanism was negative for both knees. Two years after the CT scan was performed, the results of the physical examination of the extensor mechanism were still negative. The importance of a physical examination cannot be underestimated



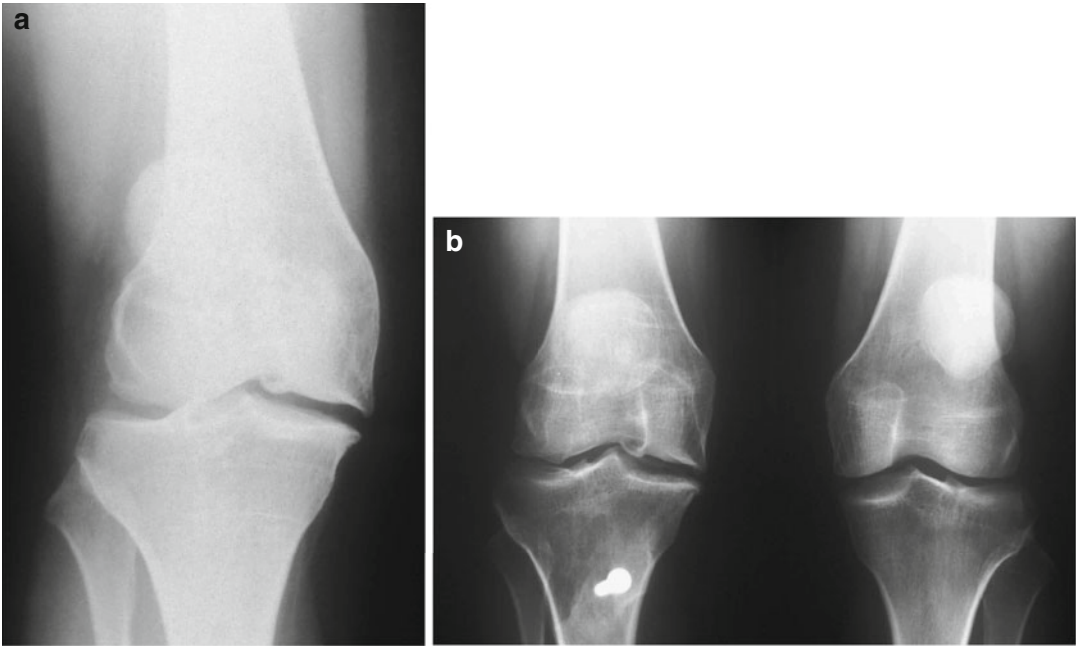


Fig. 10.17 Post-menisectomy osteoarthritis in a patient who had been mistakenly diagnosed with a rupture of the medial meniscus owing to a confusion between patello-femoral and meniscal pathologies (a). An extensor mechanism realignment surgery did away with the symptoms, which led to the first operation (b)

Fig. 10.18 Magic angle phenomenon (**a, b**). Sagittal views, which show sequences with T1 (**a**) and T2 (GE) (**b**) – weighted images. Signal variations can be observed in the patellar tendon suggesting a structural alteration. If one looks at the image more closely, one notices that the signal variation follows the tendon’s axis and that, in addition, there is no change whatsoever in its profile. This alteration corresponds to an imaging artifact arising from the magic angle phenomenon. This term covers the signal variations shown by certain structures when they are not aligned with the direction of the magnetic field (50°). This phenomenon is seen more often when the gradient echo (GE) technique is used. This is therefore an example of a false positive. Typical MR image of a patellar tendinopathy, T2-weighted FSE image, sagittal plane (**c**)

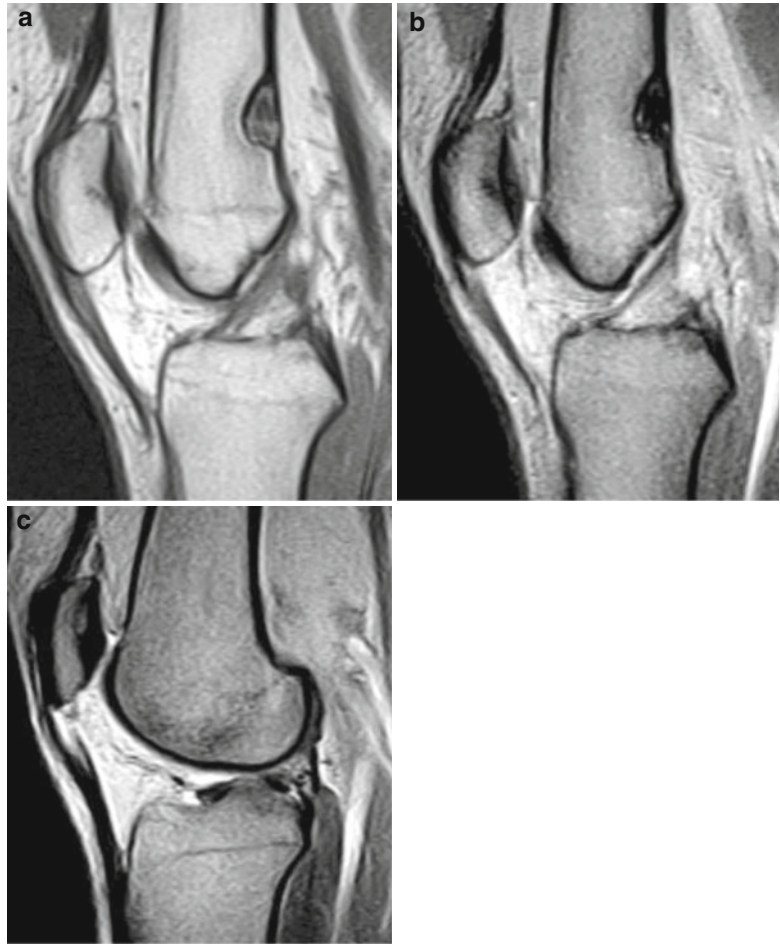


Fig. 10.19 This patient had been suffering from anterior knee pain for several months caused by trauma due to a car accident. Conventional x-rays did not show any pathological finding. However, MRI did. (a) Sagittal SE T1W MR image. (b) Axial FSE PDW Fat Sat MR image. (c) Sagittal FSE T2W MR image

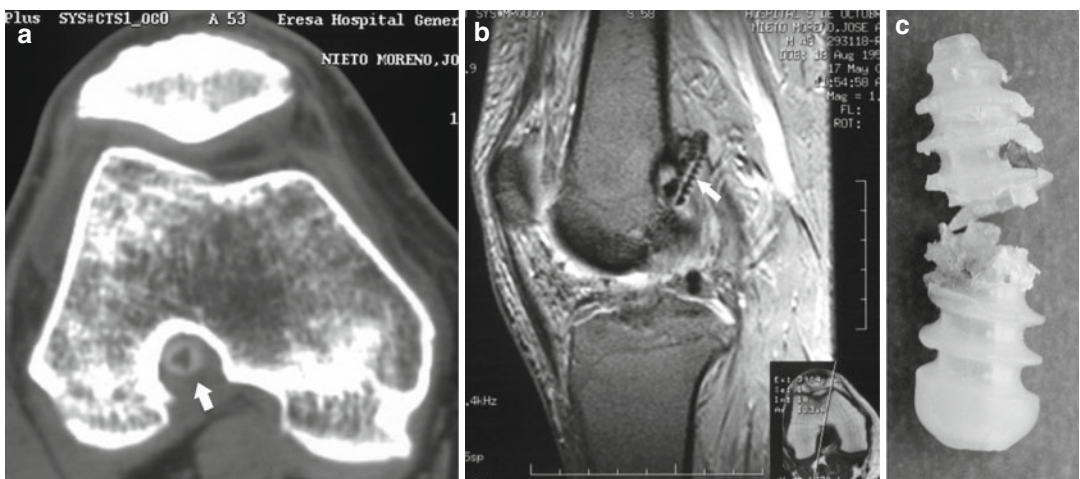
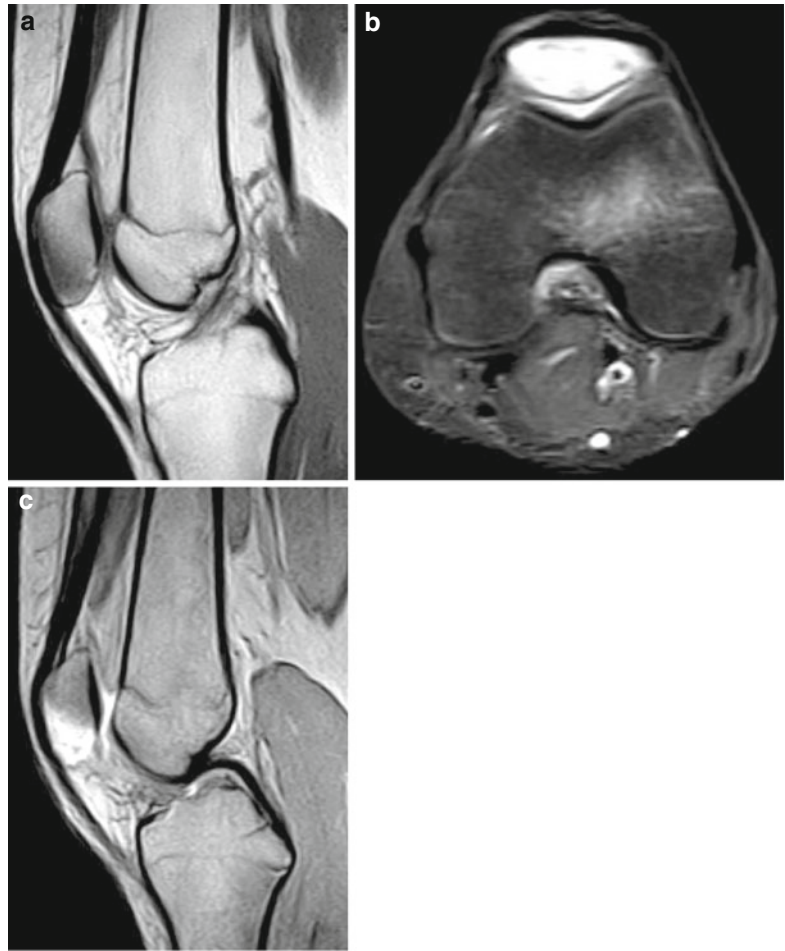
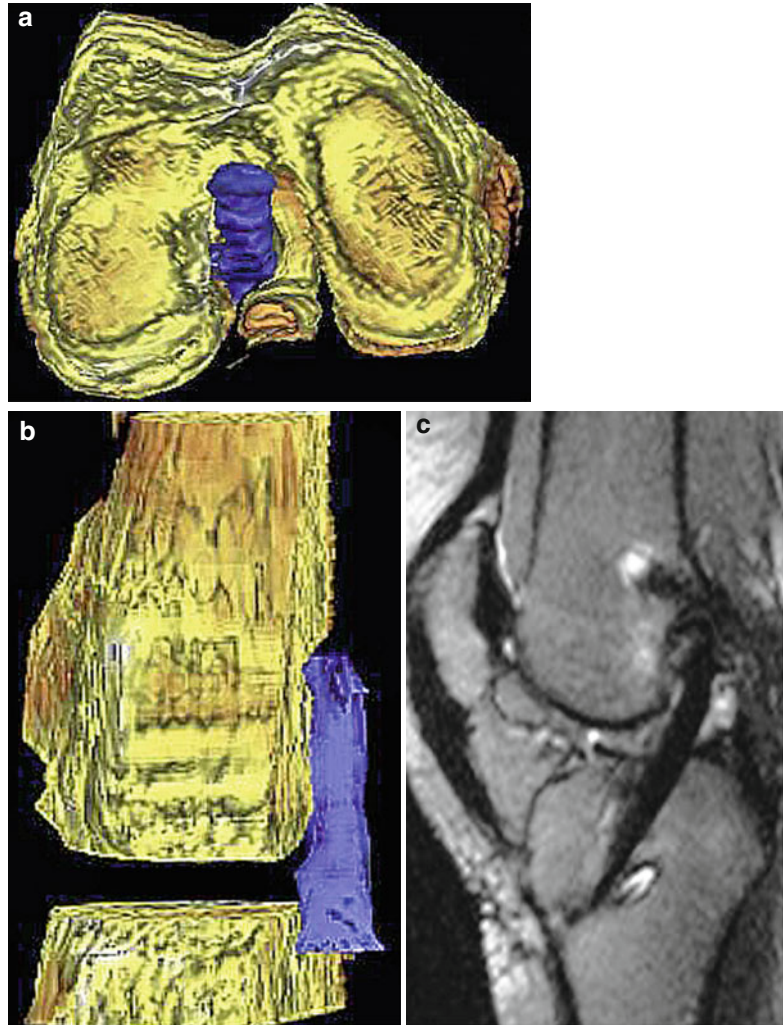


Fig. 10.20 (a) Axial CT scan at 0° of knee flexion demonstrating a lateral subluxation of the patella. Femoral screw (*arrow*). (b) Sagittal GrE T2* MRI demonstrating a severe femoral screw/tunnel divergence. Moreover, you can note that the screw is broken (*arrow*). (c) Broken femoral

oral interference screw (Reprinted from Sanchis-Alfonso V, Tintó-Pedrerol M. Femoral interfere screw divergente after anterior cruciate ligament reconstruction provoking severe anterior knee pain. *Arthroscopy*. 2004;20:528–31. With permission from Elsevier)

Fig. 10.21 Anterior knee pain after ACL reconstruction in a patient with a correct patellofemoral alignment. MRI 3D reconstruction in the knee extended position demonstrated that the PCL is displaced medially and indented by a vertical ACL-graft in the coronal plane. Our patient was pain-free after an anatomical ACL-graft replacement. (**a, b**) T2-weighted 3D echo sequence in transverse acquisition with 3D rendering with surface algorithm with Barco Voxar 3D software. Bone structures (tibia, femur, patella) are segmented according to signal intensities. The segmentation is done by manually delineating the ACL-graft in each of the transverse planes. Bone structures are in *yellow*, and the ACL-graft is in *blue*. We can see the verticalization of the ACL-graft. (**c**) Sagittal FSE T2 2D sequence where we can see the ACL as a markedly hypointense structure



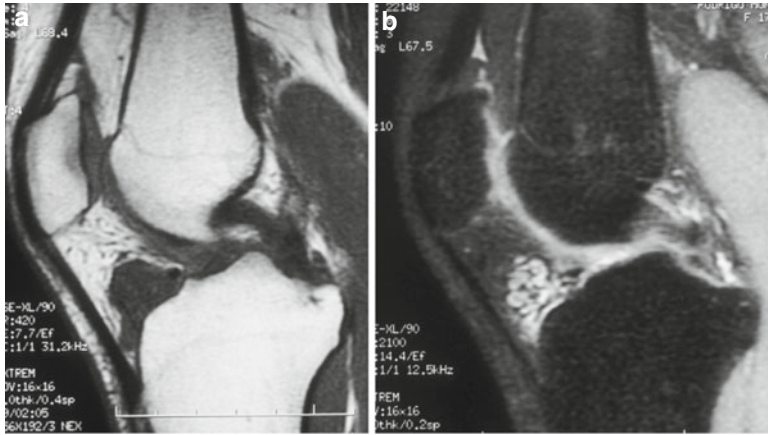


Fig. 10.22 MRI. Sagittal plane FSE T1W, showing a rounded lesion involving the infrapatellar Hoffa's fat pad, isointense with skeletal muscle, displacing the intermeniscal ligament but without affecting either bone or the patellar tendon (a). Oblique sagittal plane FSE PDW with Fat

Sat, showing the same lesion as in the Fig. a (b). The lesion appears hyperintense, of polycyclic appearance, and with hemosiderin and/or ferritin within the interior and at periphery. Likewise, no bone or tendon involvement is noted in this image

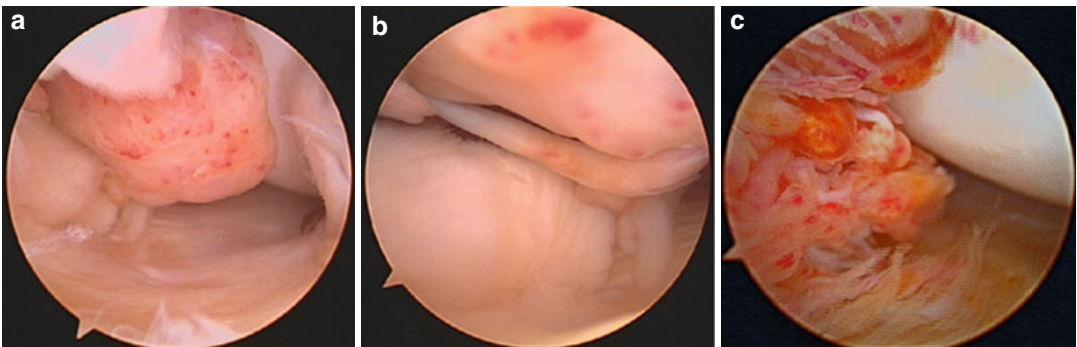


Fig. 10.23 Arthroscopic view of the tumor. Tumor-like mass, well encapsulated, in the anteromedial aspect of the knee (a). Long pedicle attaching the mass to adjacent syn-

ovium (b). Involvement of the surrounding synovium with hypertrophic villous-like projections with brownish pigmentation (c)

Risk Factors and Prevention of Anterior Knee Pain

11

Erik Witvrouw, Damien Van Tiggelen,
and Tine Willems

The etiopathogenic basis of anterior knee pain must be considered as multifactorial. Consequently, before any prevention program can be constructed, knowledge of the intrinsic and extrinsic risk factors of anterior knee pain is needed. The association between clinical overload of the patellofemoral joint (extrinsic risk factors) and anterior knee pain is well known. However, since only very few prospective studies are performed, the importance and identification of the different intrinsic risk factors of anterior knee pain remains enigmatic.

However, determination of these intrinsic risk factors of anterior knee pain is the first step in the sequence of injury prevention. Trying to interpret the few existing prospective and follow-up studies it seems clear that the quadriceps muscle can be considered as an important characteristic in the genesis of anterior knee pain. Consequently, training of this muscle seems the cornerstone in

the prevention of anterior knee pain. Data on the quadriceps muscle seem to show that the flexibility and the functional strength are important. Accordingly, it can be postulated that stretching of the hamstring and quadriceps should be considered as an important aspect of a preventive (and conservative) treatment program in anterior knee pain patients, and should preferably be incorporated in these treatment programs.

Concerning the muscle strength of the quadriceps, it seems that especially a lack of “functional” quadriceps strength is an important aspect in the development of anterior knee pain. This leads us to conclude that the use of functional strength training (closed kinetic chain exercises) as a preventive measure should be advised.

Striking, and not in agreement with common practice, these very few prospective data seem to indicate that clinically measured lower leg alignment characteristics such as leg length difference, height, weight, Q angle, genu varum/valgum and recurvatum seem not to be that important in the development of anterior knee pain.

Although some studies have shown the beneficial effect of the use of orthotics in the treatment of anterior knee pain, no prospective studies on the use of orthoses as preventive measure for AKP are yet performed. Therefore, today no substantial evidence exists on the preventive use of foot orthoses for anterior knee pain.

On the other hand, two prospective studies have shown that patellofemoral bracing does help to prevent anterior knee pain in subjects undergoing a strenuous training program. The exact

E. Witvrouw, Ph.D. (✉) • T. Willems
Department of Rehabilitation Sciences
and Physical Therapy, Faculty of Medicine,
Ghent University of Gent,
Ghent, Belgium
e-mail: erik.witvrouw@ugent.be

D. Van Tiggelen, PT
Department of Rehabilitation Sciences
and Physical Therapy, Faculty of Medicine,
Ghent University of Gent,
Ghent, Belgium

Department of Traumatology and Rehabilitation,
Military Hospital Queen Astrid,
Brussels, Belgium

underlying mechanism remains obscure, but one study showed that bracing is able to facilitate quadriceps strength.

However, it must be remembered that conclusions drawn in this chapter are based on the results

of relatively few prospective data and should therefore always be warranted. Accordingly, it seems obvious that a lot of research is still needed before a scientific prevention program for anterior knee pain can be composed.

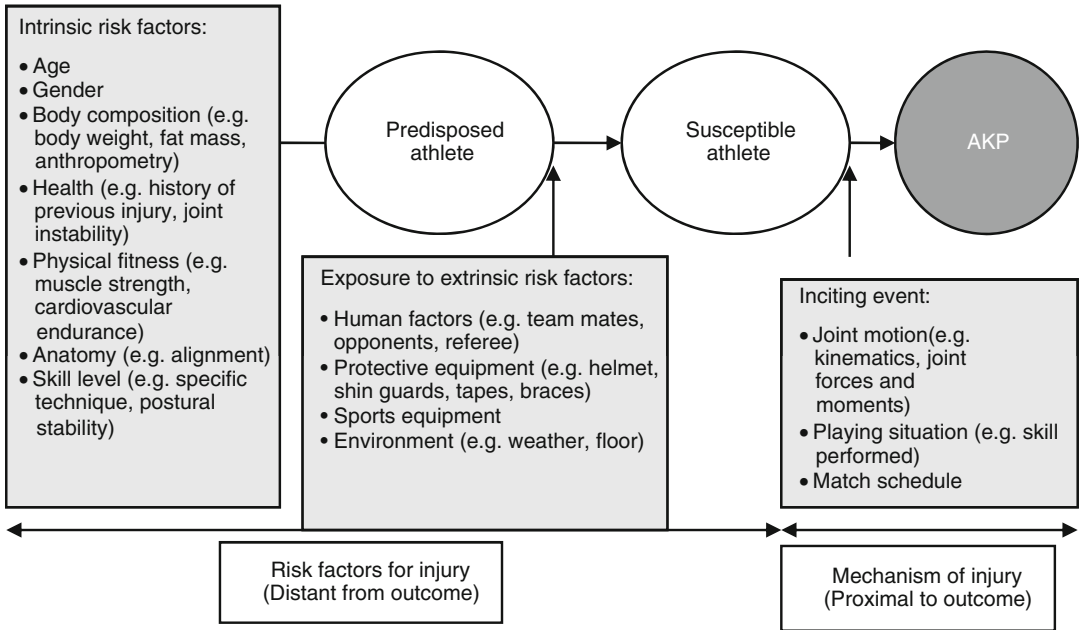


Fig. 11.1 A dynamic, multifactorial model of sports injury etiology (Adapted from Meeuwisse WH. Assessing causation in sport injury: a multifactorial model. Clin J Sport Med. 1994;4:166–70)

Fig. 11.2 The sequence of prevention of sports injuries (Adapted from Van Mechelen W, Hlobil H, Kemper HC, et al. Incidence, aetiology and prevention of sports injuries: a review of concepts. Sports Med. 1992;14:82–9. With permission from Adis International, Wolters Kluwer Health)

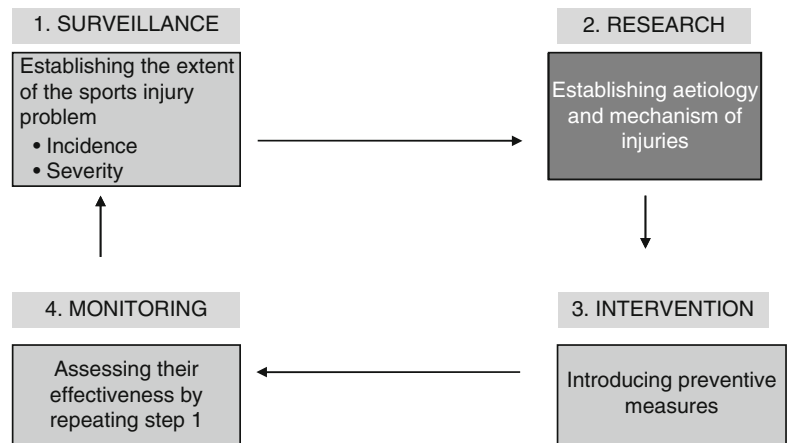




Fig. 11.3 Performance of the single leg hop test. The arms stay at the back during the entire test

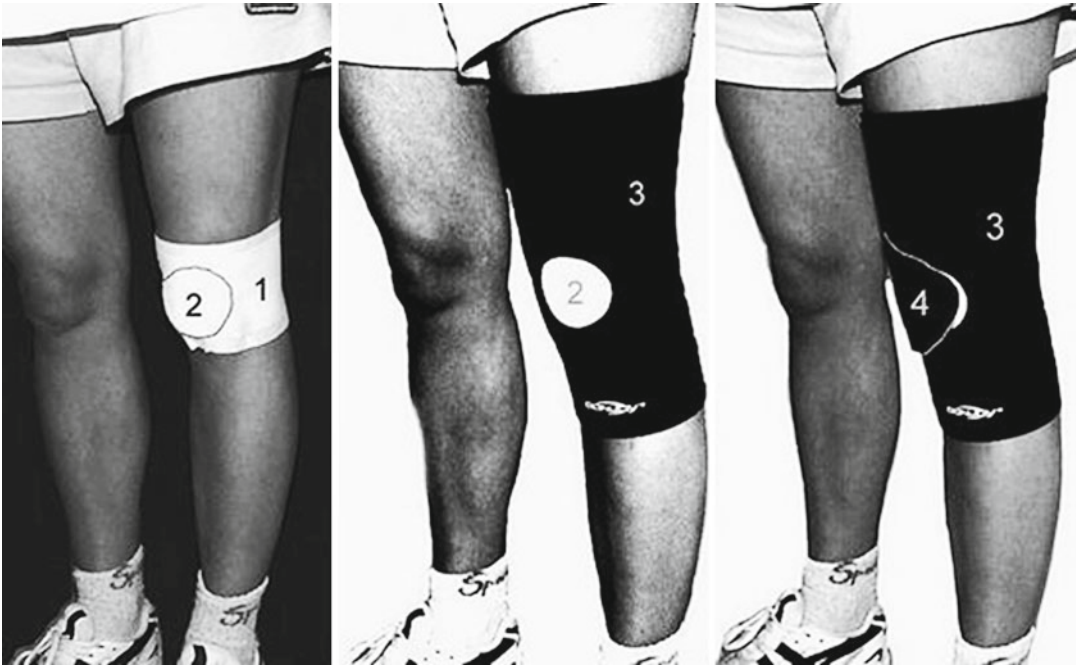


Fig. 11.4 The On-Track brace (Donjoy) in three parts. A self-adhesive patch (1) is applied on the knee with the loop circle (2) on the patella. The neoprene cuff (3) is pulled onto the leg so the loop circle shows through the opening of the cuff. The hook circle (4) is attached to the loop circle on the patch (Reproduced from Van Tiggelen

D, Witvrouw E, Roget Ph, et al. Effect of bracing on the prevention of anterior knee pain: a prospective randomized study. *Knee Surg Sports Traumatol Arthrosc.* 2004;12:434–9. With kind permission of Springer Science + Business Media)

Non-operative Treatment of Athletes with Anterior Knee Pain: Science, Classical, and New Ideas

12

Suzanne Werner

12.1 A Step by Step Treatment Protocol for Akp Patients

Phase 1

Goals:

Reduce pain and swelling, improve VMO: VL balance and thereby patellar tracking, improve flexibility, restore normal gait, and decrease loading of the patellofemoral joint.

Treatment:

- Cryotherapy – after the physical therapy exercise and daily activities, that exacerbate symptoms to reduce pain and edema.
- Transcutaneous electrical stimulation of VMO to restore the function of VMO and improve VMO: VL balance (Fig. 12.1). This could be done according to our specific protocol (Table 12.3).
- Flexibility training. Stretching of tight muscle structures, usually the tensor fascia lata and the iliotibial band (Fig. 12.2), the quadriceps, in particular rectus femoris (Fig. 12.3), the hamstrings (Fig. 12.4), and the gastrocnemius. Tight lateral retinaculum can, except for stretching, be treated

with medial patellar glide, friction and massage.

- If gait has been altered, the patient should be instructed in proper gait mechanics, which preferably could be done in front of a mirror.
- Instruct the patient to change postural habits such as e.g. standing in genu recurvatum.
- If patellar hypermobility exists, it is recommended to either tape the patella or to use a patellar stabilizing brace during the physical therapy exercises. However, patellar supporting devices should only be used temporarily until exercises and functional activities can be performed without knee pain.
- If increased pronation of the subtalar joint exists, treat the patient with foot orthotics or arch taping. Foot orthotics can be used temporarily or may be needed indefinitely to improve patellar tracking and alignment of the lower extremity.
- Check the patient's shoe wear, in particular sport shoes, and if needed suggest shock absorptive shoes.
- Modify daily activity level to temporarily reduce the load on the patellofemoral joint.

Phase 2

Goals:

Improve balance of the lower extremity, increase quadriceps strength, and restore good knee function.

Treatment, add:

- Balance and coordination training with gradual increase of difficulty and loading

S. Werner, Ph.D.
Stockholm Sports Trauma Research Center,
Karolinska institutet,
Stockholm, Sweden

Capio Artro Clinic,
Stockholm, Sweden
e-mail: suzanne.werner@ki.se

on the patellofemoral joint. In order to try to mainly train the knee joint stabilizers I suggest that these exercises should be performed in a standing position with a slightly flexed knee joint. Balance training on a balance board can initially be performed standing on one leg with addition of electrical stimulation of VMO to facilitate a proper balance between VMO and VL (Fig. 12.5). When good muscle control is achieved the patient can continue the balance exercise standing on one leg without electrical muscle stimulation (Fig. 12.6) or standing on both legs on two balance boards (Fig. 12.7).

- Stationary bicycle training with a high seat aimed to reduce a big knee flexion angle and thereby compression forces within the patellofemoral joint (Fig. 12.8). This type of exercise might improve both physical conditioning and thigh muscle strength.
- Functional knee exercises. Start with shallow squats, and proceed with deeper ones. Squatting can initially be performed with addition of electrical stimulation of VMO to improve the VMO: VL balance (Fig. 12.9). Stepping- down can also be started with addition of electrical muscle stimulation (Fig. 12.10), and gradually be performed without (Fig. 12.11).
- Quadriceps strengthening is recommended to be started when a good balance between VMO and VL exists. Closed kinetic chain

exercises should be performed during terminal knee extension, approximately between 30° – 0° of knee flexion, and open kinetic chain between approximately 90 – 40° of knee flexion. Isokinetic training should preferably be performed at $120^{\circ}/s$ or higher during concentric actions and at $90^{\circ}/s$ or lower during eccentric actions (Fig. 12.12).

Phase 3

Goal:

Return to previous physical activity level.

Treatment, add:

- Functional training with a gradual increase of knee loading activities can begin after improved quadriceps strength. Walking, jogging and different types of jumping exercises are recommended during this phase. However, proceeding to a higher knee loading activity or exercise should only be allowed, if there is no knee pain and no swelling.
- Sport specific exercises with a gradual increase of intensity can start as soon as the athletic patient is painfree, has a good muscle function and a proper movement pattern during functional knee exercises.
- It is recommended to give the patient individual guidelines for physical activity and exercises regarding e.g. number of repetitions, duration, intensity and frequency.
- Patient education is also recommended in order to try to prevent recurrence of knee symptoms.

Fig. 12.1 Transcutaneous electrical stimulation of VMO

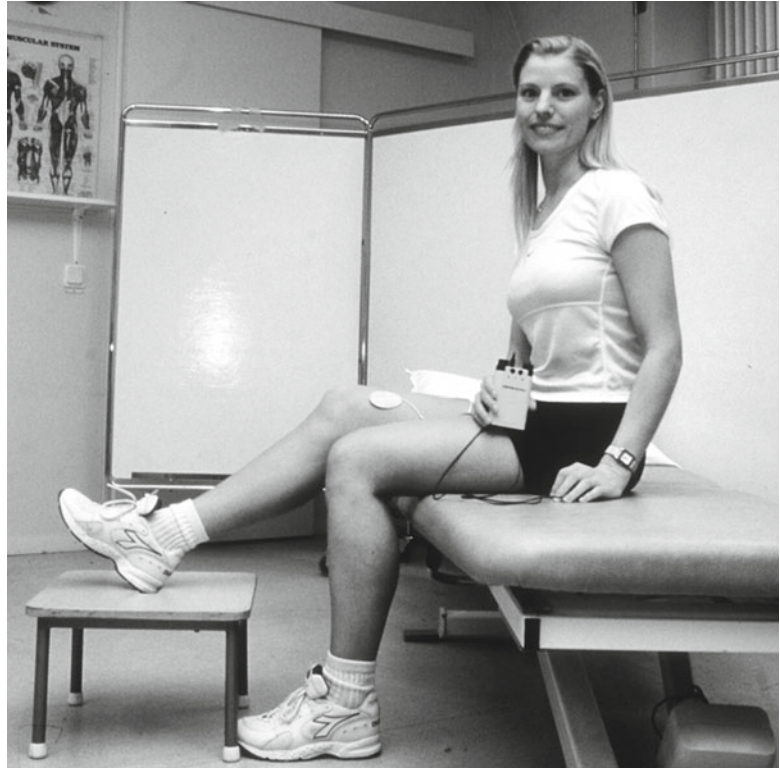




Fig. 12.2 Stretching of lateral muscle structures, the tensor fascia lata, and the iliotibial band

Fig. 12.3 Stretching of rectus femoris



Fig. 12.4 Stretching of hamstrings



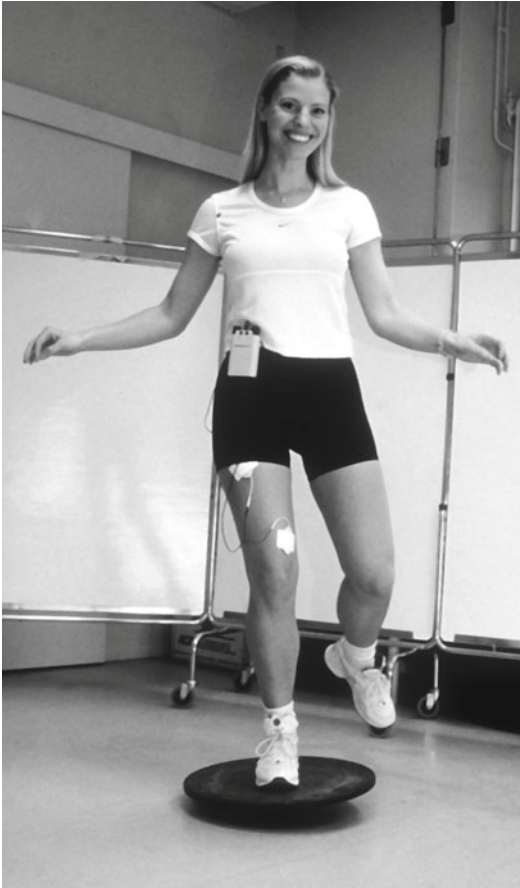


Fig. 12.5 Single-leg standing balance board training with addition of electrical stimulation of VMO



Fig. 12.6 Single-leg standing balance board training

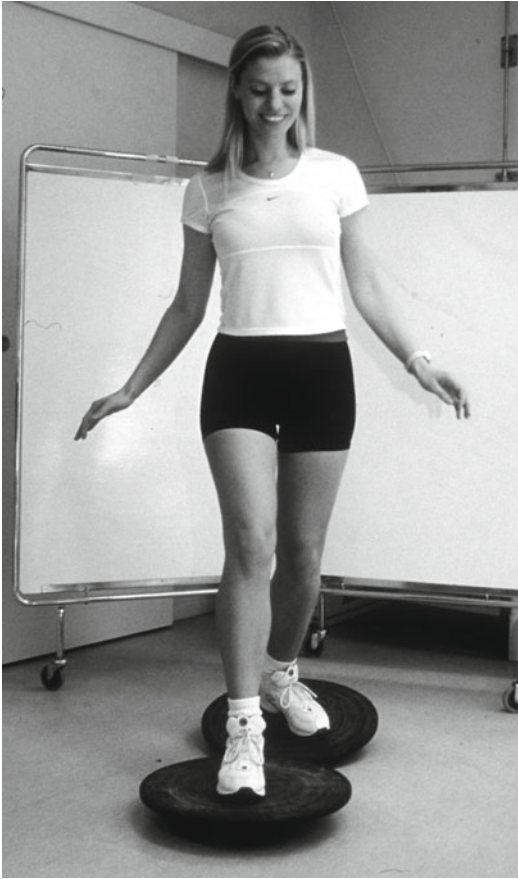


Fig. 12.7 Balance board training standing on two legs on two balance boards

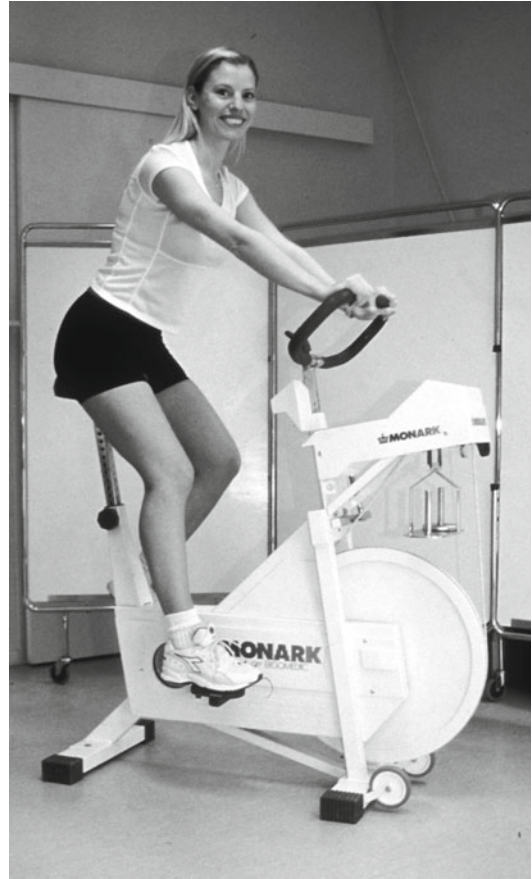


Fig. 12.8 Stationary bicycle training with a high seat



Fig. 12.9 Squatting with addition of electrical stimulation of VMO

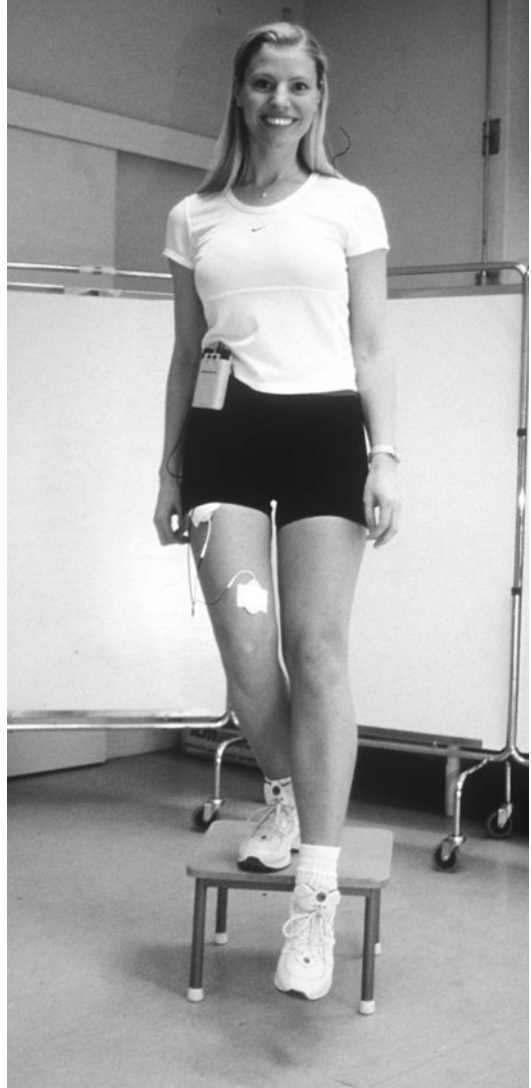


Fig. 12.10 Stepping down with addition of electrical stimulation of VMO



Fig. 12.11 Stepping down

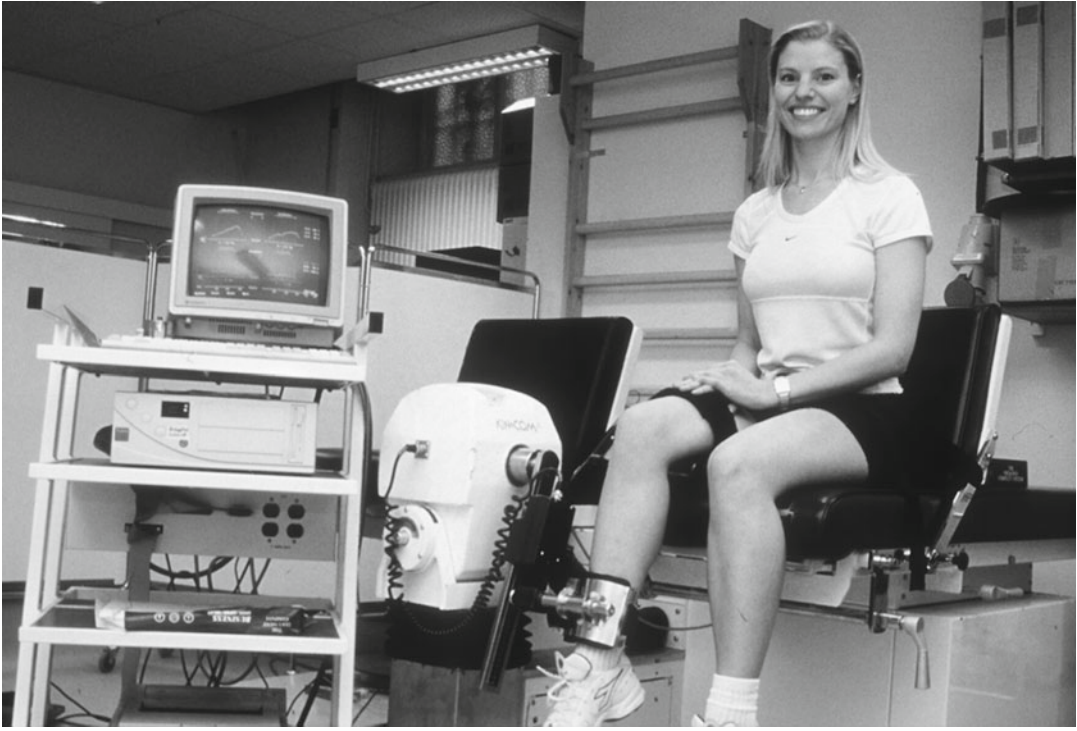


Fig. 12.12 Isokinetic quadriceps training

Conservative Management of Anterior Knee Pain: The McConnell Program

13

Jenny McConnell and Kim Bennell

Management of patellofemoral pain is no longer a conundrum if the therapist can determine the underlying causative factors and address those factors in treatment. It is imperative that the patient's symptoms are significantly reduced. This is often achieved by taping the patella, which not only decreases the pain, but also promotes an

earlier activation of the VMO and increases quadriceps torque. Management will need to include specific VMO training, gluteal control work, stretching tight hip and lateral patellar structures and appropriate advice regarding the foot, be it orthotics, training or taping.

J. McConnell, BAppSci (Phy), Grad Dip Man Ther,
M Biomed Eng.
McConnell & Clements Physiotherapy,
Sydney, Australia

K. Bennell, Ph.D. (✉)
Centre for Health, Exercise and Sports Medicine,
Physiotherapy, School of Health Sciences,
The University of Melbourne,
Victoria, VIC, Australia
e-mail: k.bennell@unimelb.edu.au

Fig. 13.1 Common biomechanical presentation – internal rotation of the femurs

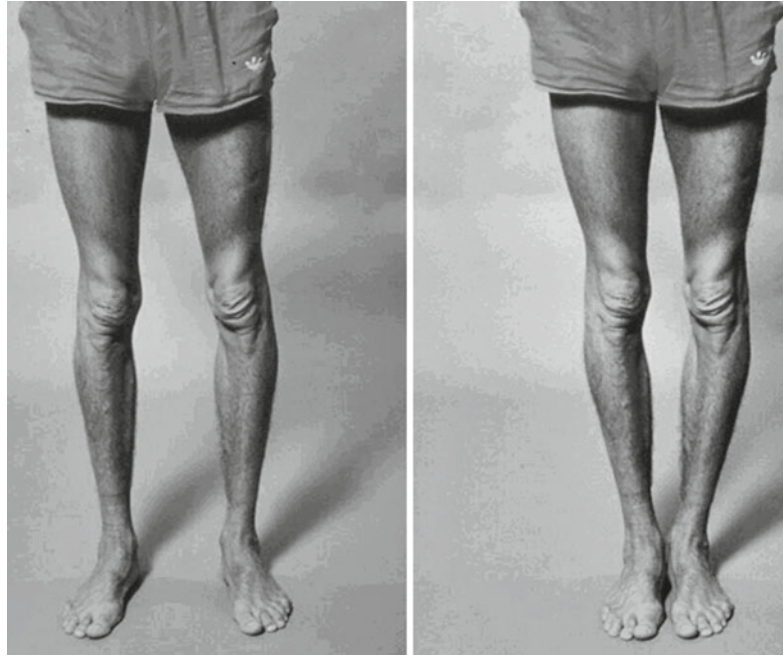


Fig. 13.2 Assessment of patellar glide



Fig. 13.3 Assessment of posterior tilt of the inferior pole of the patella

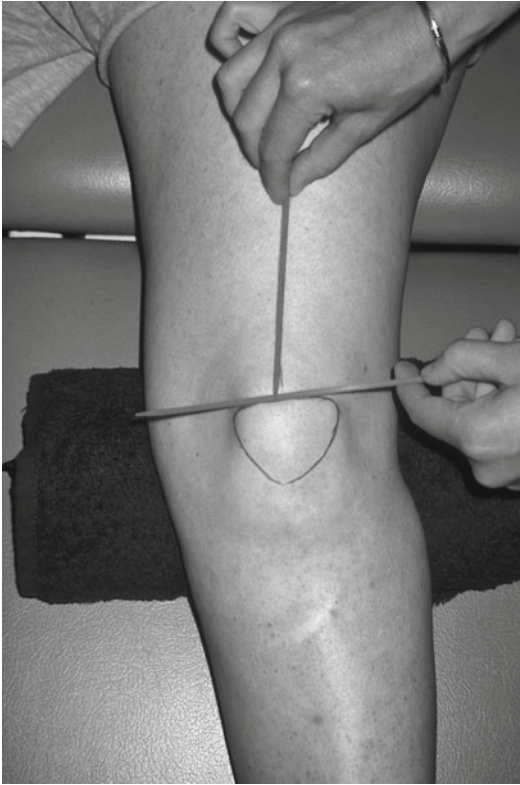


Fig. 13.4 Assessment of rotation of the patella



Fig. 13.5 Assessment of the flexibility of the anterior hip structures

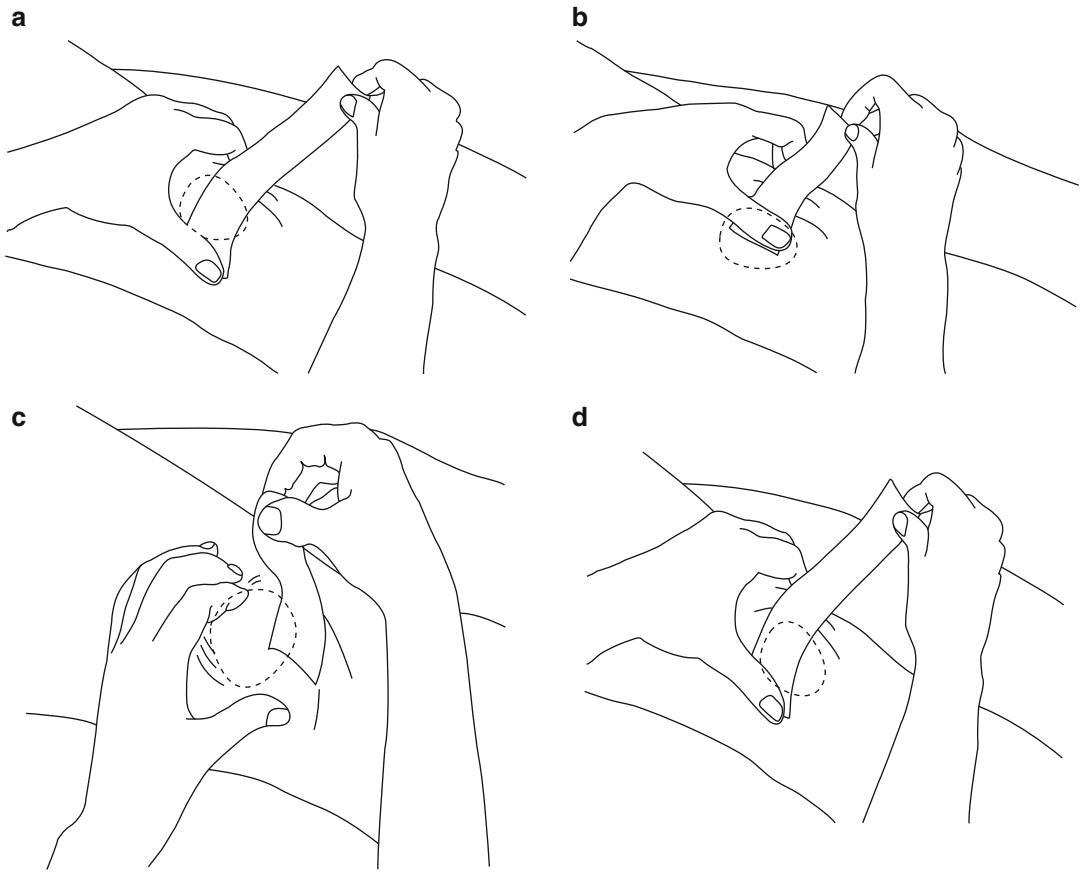


Fig. 13.6 Taping components: (a) medial glide, (b) Medial tilt, (c) Internal rotation, (d) Anterior tilt

Fig. 13.7 (a) Tape to correct lateral glide. (b) Tuck or fold in the skin. (c) Tape in internal rotation to correct external rotation of the patella. (d, e) Unloading fat pad with tape, lift the soft tissue towards the patella

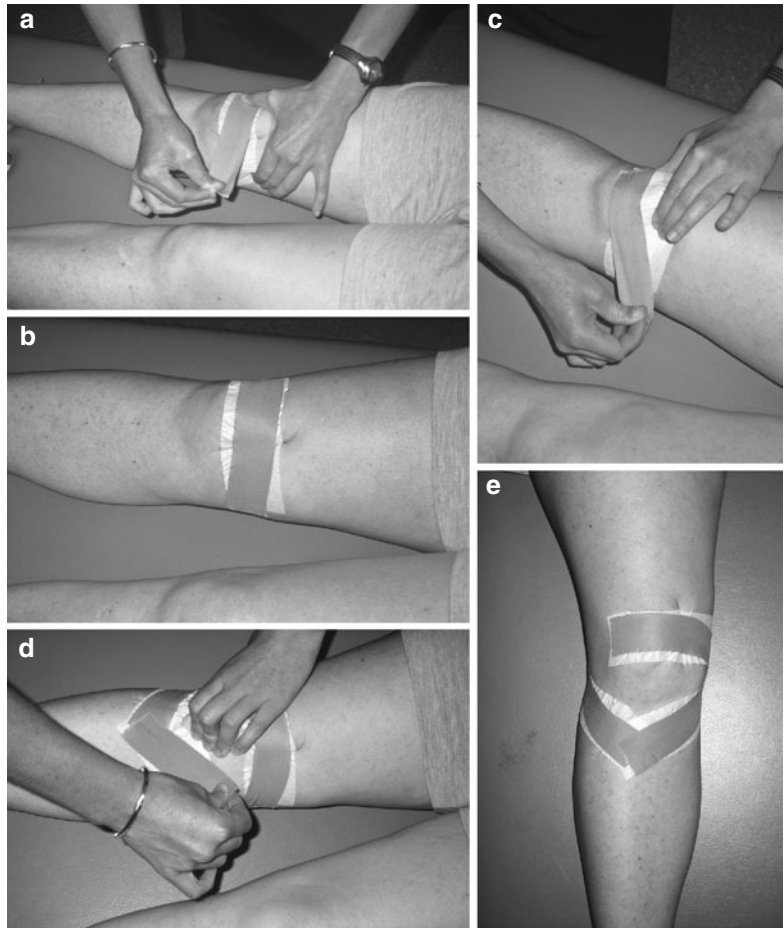
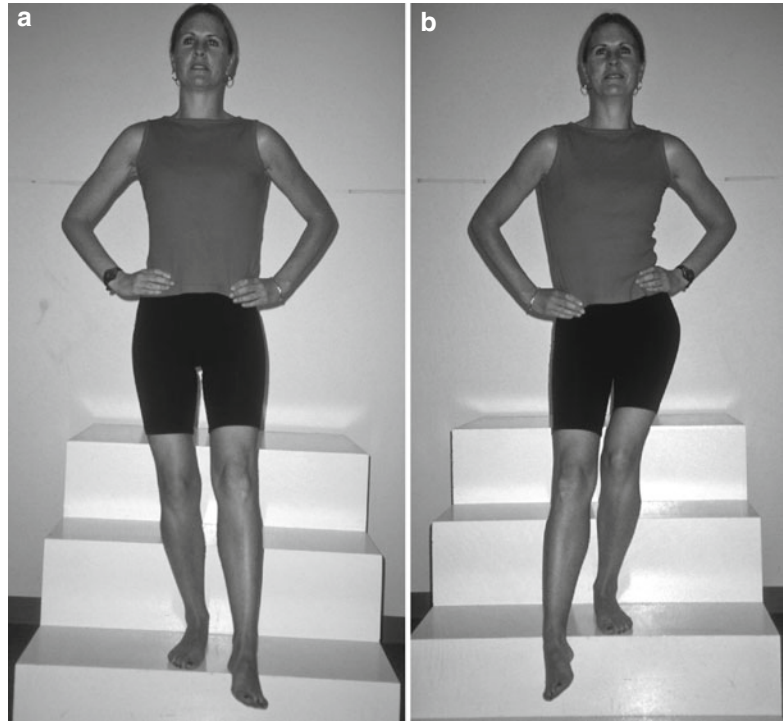


Fig. 13.8 (a) Stepping down with correct limb alignment.
(b) Stepping down with incorrect limb alignment



Skeletal Malalignment and Anterior Knee Pain: Rationale, Diagnosis, and Management

14

Robert A. Teitge and Roger Torga-Spak

Any variation from optimal skeletal alignment may increase the vector forces acting on the patellofemoral joint causing either ligament failure with subsequent subluxation or cartilage failure as in chondromalacia or arthrosis or both ligament and cartilage failure (Fig. 14.1). Anterior knee pain may result from these abnormal forces or their consequences.

The mechanical disadvantage provided by a skeleton with a geometrical or architectural flaw distributes abnormal stresses to both the ligaments and the joints of the misaligned limb. Ligament overload and subsequently failure (insufficiency) may occur with a single traumatic episode as well as repetitive episodes of minor trauma or

chronic overload. Skeletal malalignment may cause chondromalacia patella and subsequently osteoarthritis by creating an increased mechanical leverage on the patellofemoral joint which can exceed the load capacity of the articular cartilage. A reduction in contact surface area such as a small patella or a high patella or a subluxed patella may also increase the unit area loading beyond the load capacity of the articular cartilage leading to cartilage failure (osteoarthritis).

Anterior knee pain in association with bony malalignment may be the result of the abnormal tension or compression placed on the capsule, ligaments, synovium or subchondral bone.

R.A. Teitge, M.D.
Department of Orthopaedics,
Wayne State University School of Medicine,
Detroit, MI, USA

R. Torga-Spak, M.D. (✉)
Department of Surgery, Faculty of Orthopaedics
and Traumatology, Instituto Universitario CEMIC,
Buenos Aires, Argentina
e-mail: rtorgaspak@gmail.com

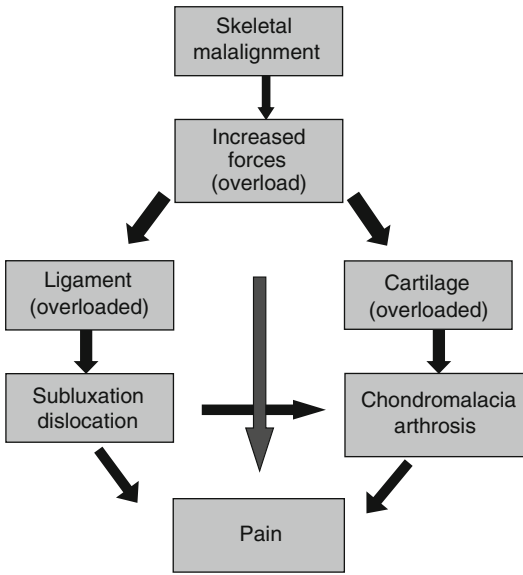


Fig. 14.1 Pathogenesis of anterior knee pain



Fig. 14.2 Whole limb standing radiograph with mechanical axis added showing varu

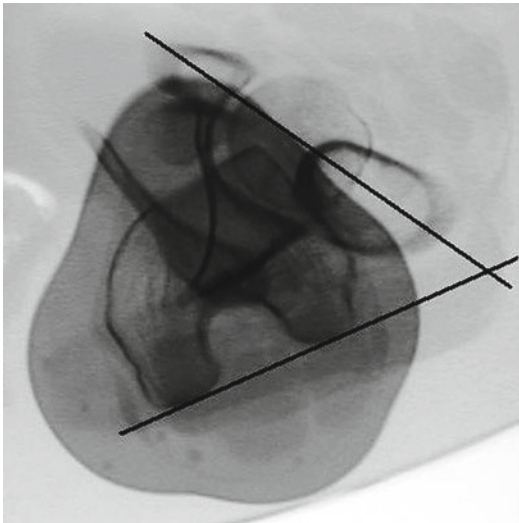


Fig. 14.3 Femoral torsion can be seen with an overlay of proximal and distal femur

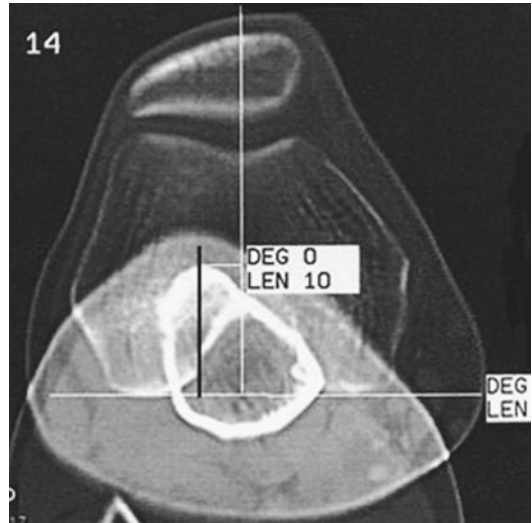


Fig. 14.5 CT scan shows measurement of the distance TT-TG. Tibial Tubercle- Trochlear Groove is seen with overlay of distal femur and tibia at the level of tibial tuberosity. Also note trochlear shape, patellar shift, patellar tilt, patellar and trochlear subchondral bone sclerosis

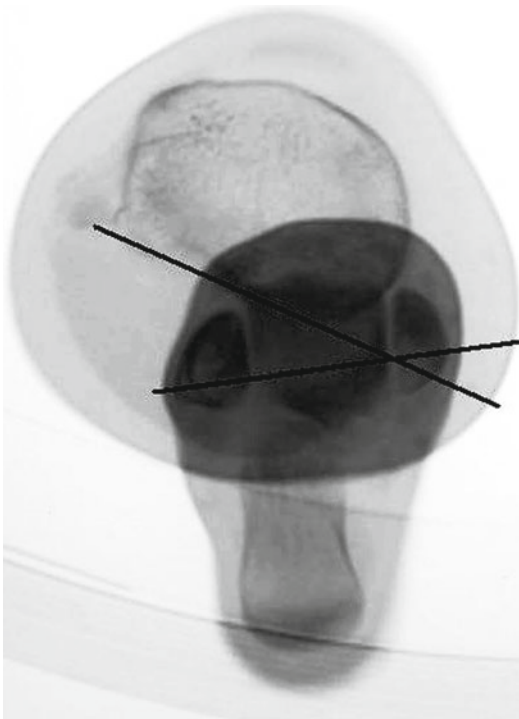


Fig. 14.4 Tibial torsion can be seen with an overlay of the proximal tibia and distal tibia

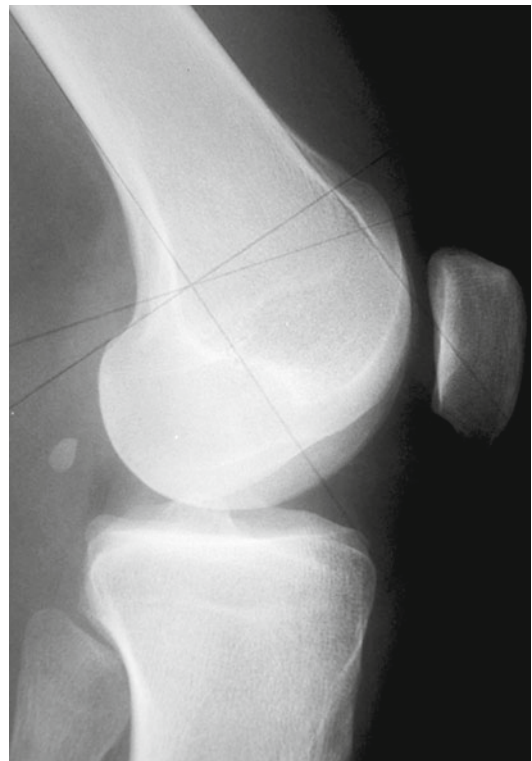


Fig. 14.6 True lateral view shows trochlear dysplasia. The trochlear line crosses the contour of the condyles (*crossing sign*) and a trochlear boss or prominence is present

Fig. 14.7 Lateral facet pressure change with femoral rotational osteotomy

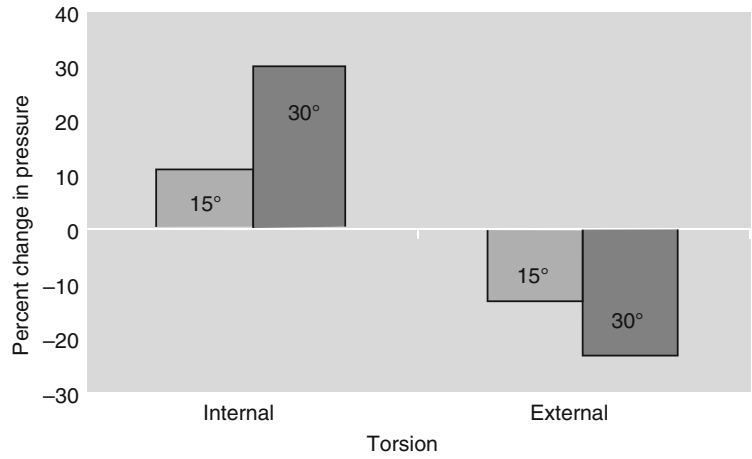
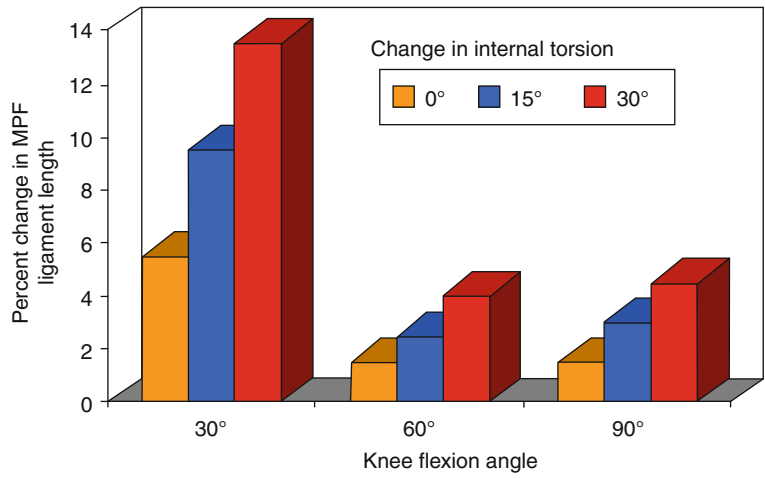


Fig. 14.8 Change in length of the medial patellofemoral ligament with increased femoral torsion



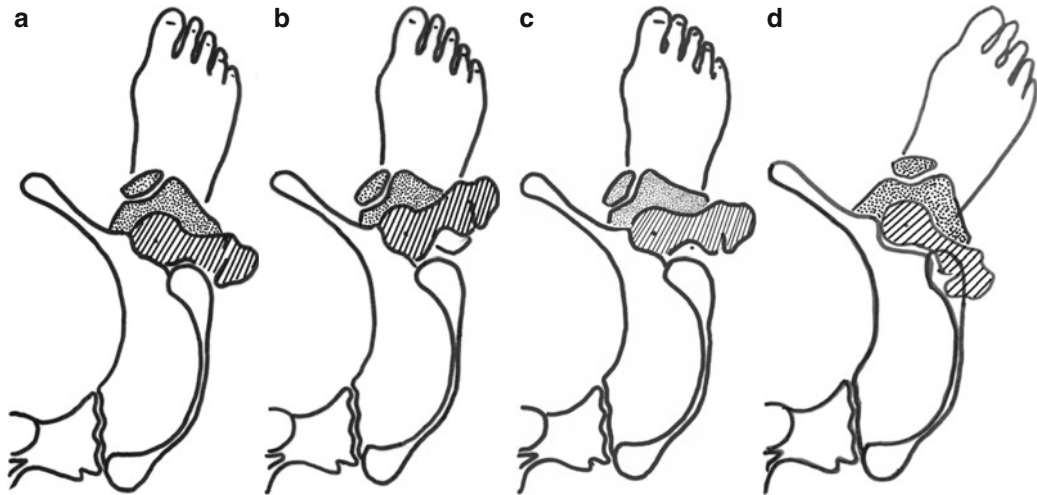
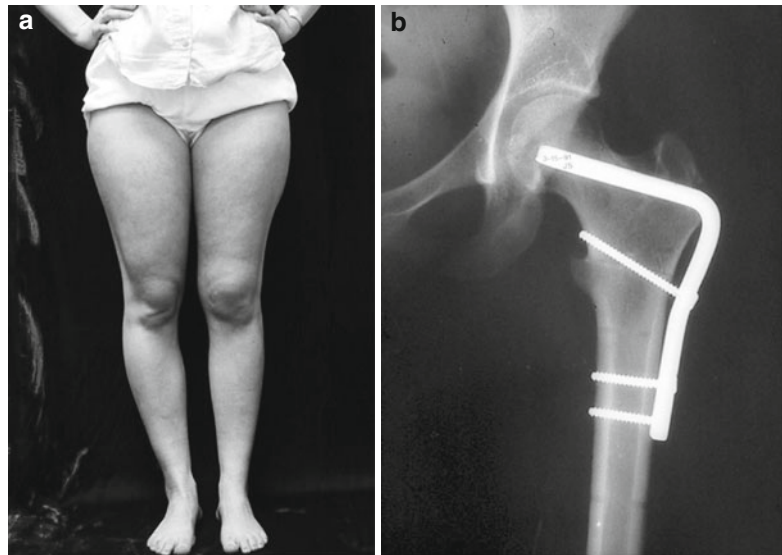


Fig. 14.9 (a) Drawing shows 20° excess femoral anteversion. With the foot forward, the knee joint points inward. (b) Drawing shows 20° excess tibial external torsion. With the foot forward, the knee joint points inward, but the hip is also excessively internally rotated. (c) Combined excess external tibial torsion (20°) and excess femoral anteversion (20°) with the foot forward. The

inward pointing of the knee is the sum of the increase in femoral anteversion plus the excess of external tibial torsion. The hip in this position gains abduction leverage. (d) Combined 20° excess external tibial torsion and 20° excess femoral anteversion. With the knee joint pointing forward, the foot points outward and the hip is in a position of abductor weakness

Fig. 14.10 (a) Picture shows a patient with excess femoral anteversion. On the left side, a proximal intertrochanteric femoral derotational osteotomy was performed, the right lower extremity had no surgery. Observe the difference between right and left in the alignment of the extremity. On the right, the patella points inward, the calf muscles are more prominent given a pseudovarus appearance and the foot is more pronated. (b) Postoperative x-rays after proximal femur derotational osteotomy



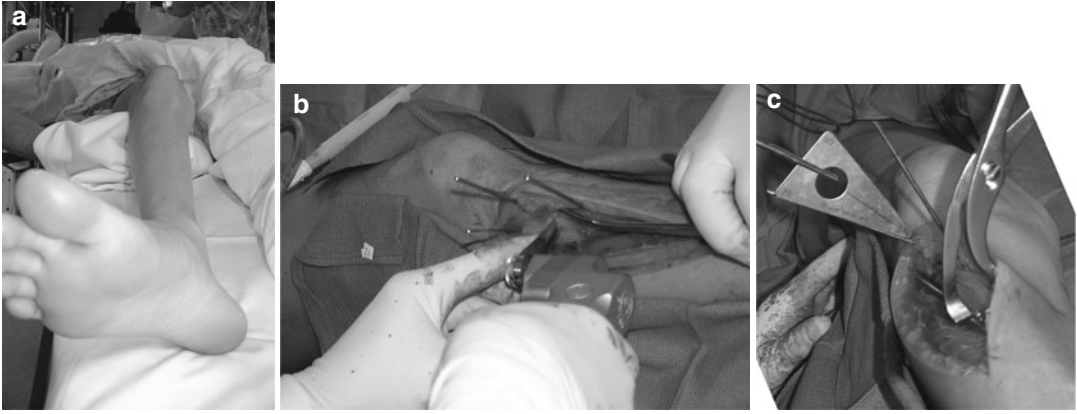
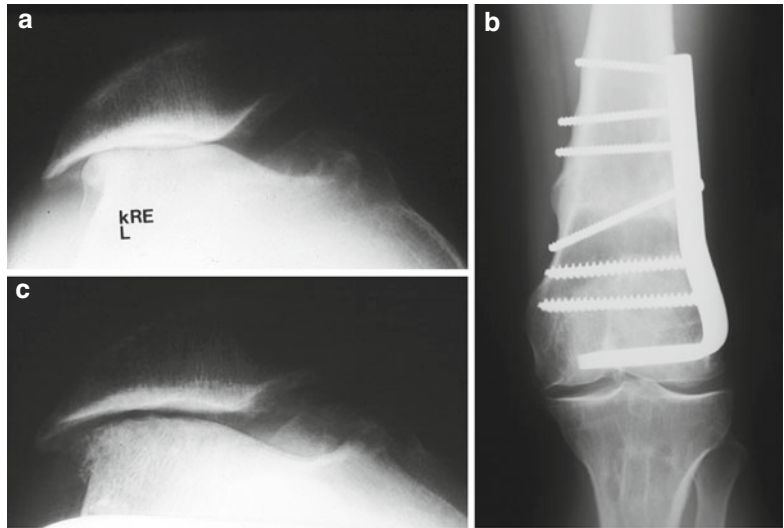


Fig. 14.11 (a) Patient with excess tibial external torsion (55°) and normal TT-TG, the foot points outward while the patella points to the front. (b) A proximal tibial internal rotation osteotomy is performed below the tibial tubercle. (c) K-wires show a 30° correction. A blade plate is used for fixation

Fig. 14.12 (a) Preoperative axial view of a 28-year-old female with history of pain and instability shows collapse of the lateral patellofemoral joint. The patient had valgus and increased femoral anteversion (43°). (b) AP Postoperative x-rays after distal femoral varus and external rotation osteotomy. (c) Axial view taken 5 years postoperative shows widening of the lateral patellofemoral space



Patrik Danielson and Alexander Scott

In conclusion, evidence points in a new direction: that biochemical mediators, deriving from nerves in or around the patellar tendon or from the tendon tissue itself, may profoundly influence the nerves, blood vessels, and tenocytes in patellar tendon tissue. The findings furthermore suggest that these phenomena arise or increase in response to patellar tendinopathy, or even precede/elicit the condition, as they are only rarely or very moderately seen in normal patellar tendons. Thus, the ramifications of neuronal or non-neuronal biochemical mediators in patellar tendinopathy include effects on tendon tissue (tendinosis changes), vascular regulation, and/or pain signaling. Clinical use of this knowledge in the future might be of potentially high impact. If the model of biochemical pathogenesis/pathology in patellar tendinopathy proves to have some validity, it would mean that clinical management would aim

to modify the biochemical milieu, rather than just focusing on collagen repair. Eccentric training regimens and surgery would probably still have their uses, but researchers would be encouraged to pursue a pharmaceutical approach focused on reducing the irritant biochemical compounds in or around the tendon, if proven to be a causative factor in tendinopathy. This would actually mean that treatments might challenge the cause, rather than only the symptoms or consequences, of tendinopathy development.

However, first experimental studies must follow, to bring clarity to the actual role of the biochemical mediators produced in tendinosis tissue. Which substances inflict or enhance pain and tissue degeneration, and which substances promote tissue healing? Animal and cell culture models are currently being used to capture the dynamic events of tendinosis and answer these questions.

P. Danielson, M.D., Ph.D. (✉)

Section for Anatomy, Department of Integrative Medical Biology, Faculty of Medicine, Umeå University, Umeå, Sweden
e-mail: patrik.danielson@anatomy.umu.se

A. Scott, B.Sc. (PT), Ph.D.

Department of Physical Therapy, Faculty of Medicine, University of British Columbia, Vancouver, BC, Canada

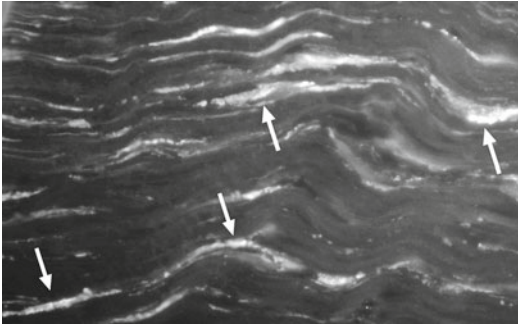


Fig. 15.1 Evidence of intratendinous ACh production. Tenocytes of patellar tendon tissue have been shown to harbor enzymes related to acetylcholine (ACh) production in tendinopathy patients. The ACh synthesizing enzyme choline acetyltransferase (ChAT), as well as its mRNA, have been found at intracellular locations. Furthermore, vesicular acetylcholine transporter (VACHT) – an enzyme that shuffles ACh from an intracellular site of synthesis into vesicles – has also been detected inside tenocytes as shown by this picture. Immunohistochemical staining (immunofluorescence method, TRITC) show specific immunoreactions inside the tenocytes, some indicated with *arrows*

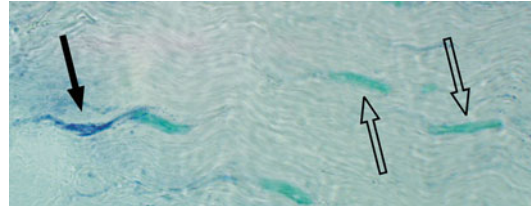


Fig. 15.2 Evidence of intratendinous catecholamine production. In-situ hybridization method shows reactions for tyrosine hydroxylase (TH) mRNA within some tenocytes (*filled arrow*) in patellar tendon tissue from a patient with tendinopathy. Other tenocytes (*unfilled arrows*) are negative in this regard. TH is the rate-limiting enzyme in the synthesis of catecholamines

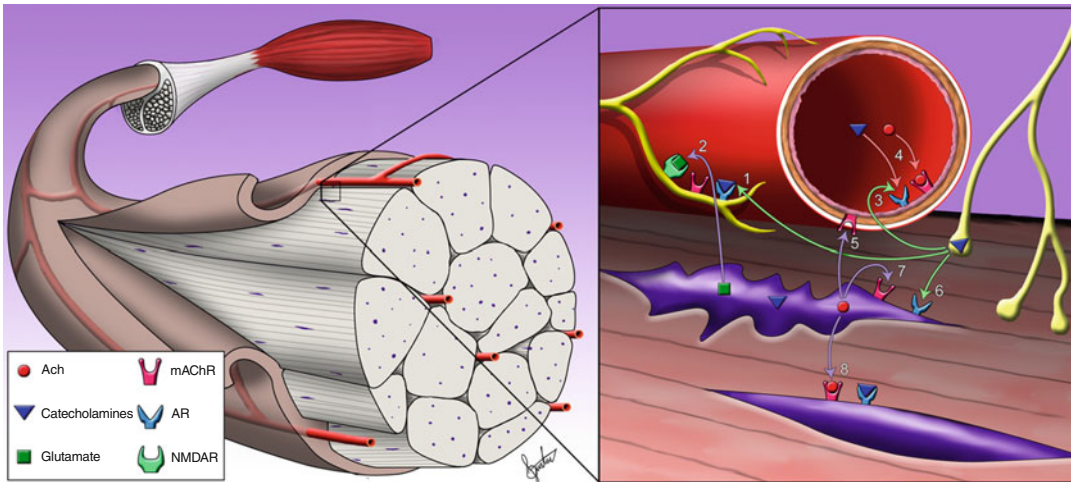


Fig. 15.3 The biochemical model for patellar tendinopathy. Schematic figure of patellar tendon tissue, showing the possible roles of biochemical mediators. The microscopic milieu is depicted in the frame to the right. Afferent sensory nerve fibers, here seen to the left in close association with a blood vessel, express muscarinic acetylcholine receptors (mAChR), *N*-methyl-d-aspartate receptors (NMDA R), and adrenergic receptors (AR). The sensory nerves are hereby susceptible to stimulation by the neurotransmitters acetylcholine (ACh) and glutamate, as well as by catecholamines. All these substances might thus theoretically affect pain signalling from the tendon. The adrenergic receptors might be influenced by catecholamines produced by neighboring efferent sympathetic nerves (1) “sympathetically maintained pain”). However, the mAChRs, the NMDA Rs, and the adrenergic receptors on the sensory nerves might also be stimulated by acetylcholine (ACh), glutamate, and catecholamines, respectively, which are produced by the tenocytes themselves (2) since these principal tendon cells have been shown to express biosynthetic enzymes for the substances in question when tendinopathy occurs. This phenomenon has been noted for the morphologically disfigured tenocytes that are frequently seen in tendinosis tissue (upper tenocyte in picture). Such tenocytes lack the slender, spindle-

shaped, appearance of normal tenocytes (lower tenocyte in picture). The efferent sympathetic nerves are furthermore likely to affect blood vessel regulation, via stimulation of adrenergic receptors in the blood vessel walls (3). Such receptors, alongside mAChRs in the blood vessel walls, are moreover expected to be stimulated by circulating catecholamines and ACh, respectively (4). A third possible source of catecholamines and ACh affecting blood vessel regulation is the tenocytes of the tendon tissue (5). The tenocytes, in addition to producing the signal substances in question, express adrenergic receptors and mAChRs, making them receptive to catecholaminergic and cholinergic effects (proliferation, changes in collagen production, and/or degeneration/apoptosis). The receptors on the tenocytes might, in the case of adrenergic receptors, be influenced by signal substances (catecholamines) produced by efferent nerves (6), or by signal substances (ACh and catecholamines) produced by the tenocytes themselves. In the latter case, autocrine (7) as well as paracrine (8) loops are suggested to occur. In summary, receptors on sensory nerves, blood vessels, and tenocytes in patellar tendons, might be affected by substances from efferent nerves (green arrows), the blood circulation (red arrows), and/or the tendon tissue itself (purple arrows) Copyright with artist: Gustav Andersson

Prevention of Anterior Knee Pain After Anterior Cruciate Ligament Reconstruction

16

K. Donald Shelbourne, Scott E. Urch,
and Heather Freeman

Anterior knee pain following reconstruction of the ACL is a problem that has plagued many patients. After extensively studying patients with this problem and comparing them to those that do not suffer from this entity, we have concluded that loss of full hyperextension is the most frequent cause of anterior knee pain after ACL reconstruction. This can be prevented with proper preoperative, intraoperative, and

postoperative management. Prevention should be the number-one concern. If anterior knee pain after ACL reconstruction does occur, the symptoms can usually be alleviated through nonoperative means. Occasionally, surgical intervention may become necessary. Other causes for this pain syndrome are rare, but with proper evaluation can easily be differentiated and treated.

K.D. Shelbourne, M.D. (✉) • S.E. Urch, M.D.
Shelbourne Knee Center at Methodist Hospital,
Indianapolis, IN, USA

Indiana University School of Medicine,
Indianapolis, IN, USA
e-mail: acldoc@aclmd.com

H. Freeman, PT, DHS
Shelbourne Knee Center at Methodist Hospital,
Indianapolis, IN, USA



Fig. 16.1 Most patients' knees have some degree of hyperextension. To evaluate the amount of hyperextension, place one hand above the knee to hold the thigh and place the other hand on the patient's foot to lift the heel off of the examination table



Fig. 16.2 Immediately after surgery, patients should be able to obtain full hyperextension in the ACL-reconstructed knee equal to the normal knee. The heel prop exercise shown in this figure is an easy method for achieving full hyperextension

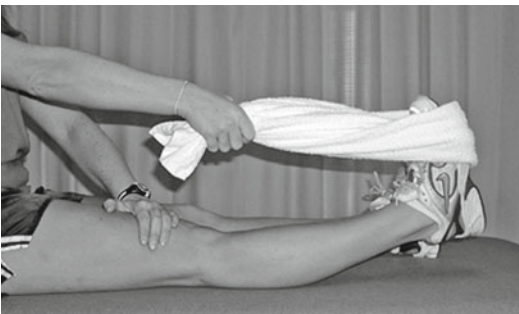


Fig. 16.3 Towel stretch extension exercise: A towel is looped around the arch of the foot and the patient holds onto both ends of the towel with one hand. The other hand pushes down on the top of the thigh while using the towel to pull up on the foot. The maneuver allows the patient to bring the knee into hyperextension passively



Fig. 16.4 Patients can use a hyperextension device for 10 min several times a day to regain the natural hyperextension in their knee. The patient controls the amount of stretch applied to the knee



Fig. 16.5 Active heel lift: The patient actively lifts the heel off of the table by simultaneously contracting the quadriceps muscle and dorsiflexing the ankle

Anterior Knee Pain with Special Emphasis on the Clinical, Radiographic, Histological, Ultrastructural, and Biochemical Aspects After Anterior Cruciate Ligament Reconstruction Using Autografts

Jüri Kartus, Lars Ejerhed, and Tomas Movin

Postoperative donor-site morbidity and anterior knee pain following ACL surgery may result in substantial impairment for the patient. The selection of graft, surgical technique, rehabilitation programme as well as concomitant injuries can affect the occurrence of undesirable pain conditions.

The loss or disturbance of anterior sensitivity caused by intraoperative injury to the infrapatellar nerve(s) in conjunction with patellar tendon harvest is correlated with donor-site discomfort and an inability to kneel and knee walk.

The patellar tendon at the donor site displays significant clinical, radiographical, histological and ultrastructural abnormalities several years after harvesting its central third. The donor-site discomfort correlates poorly with radiographical and histological findings after the use of patellar tendon autografts. The use of hamstring tendon autografts

causes less postoperative donor-site morbidity and anterior knee problems than the use of patellar tendon autografts. There also appears to be a regrowth of the hamstring tendons within 2 years after the harvesting procedure as seen in radiographical, histological as well as dissection studies. There is a lack of knowledge in terms of the course of the donor site after harvesting fascia lata autografts. Harvesting quadriceps tendon autografts appears to cause low donor-site morbidity.

Efforts should be made to spare the infrapatellar nerve(s) during ACL reconstruction using patellar tendon autografts and hamstring autografts. Reharvesting the patellar tendon cannot be recommended due to significant clinical, radiographical, histological and ultrastructural abnormalities several years after harvesting and also after re-harvesting its central third. Patients who regain full range of motion and strength after the use of any type of autograft are less likely to have anterior knee pain problems.

Since randomised controlled trials have shown that the laxity measurements and clinical results following ACL reconstruction using hamstring tendon autografts are similar to those of patellar tendon autografts, we recommend the use of hamstring tendon autografts due to fewer donor-site problems.

J. Kartus, M.D., Ph.D. (✉) • L. Ejerhed, M.D., Ph.D.
Orthopaedic Department,
University of Gothenburg, NU-Hospital Organization,
Trollhättan/Uddevalla, Sweden
e-mail: juri.kartus@vregion.se

T. Movin, M.D., Ph.D.
Emergency Department,
Karolinska University Hospital, Karolinska Institutet,
Stockholm, Sweden



Fig. 17.1 The infrapatellar nerve splits into two branches, right in the center of a central anterior 8 cm incision. The towel clamps indicate the paratenon. The patellar tendon autograft in this specimen was harvested using the two-incision technique with the aim of sparing the infrapatellar nerve(s) and the paratenon. In this specimen, the two incisions have subsequently been conjoined in order to examine the result of the harvesting procedure (Kartus J. Donor site morbidity after anterior cruciate ligament reconstruction. Clinical, anatomical, radiographic and histological investigations with special reference to the use of central-third patellar tendon autografts. Thesis, Göteborg, Sweden: Göteborg University; 1999) (Copyright Elsevier)

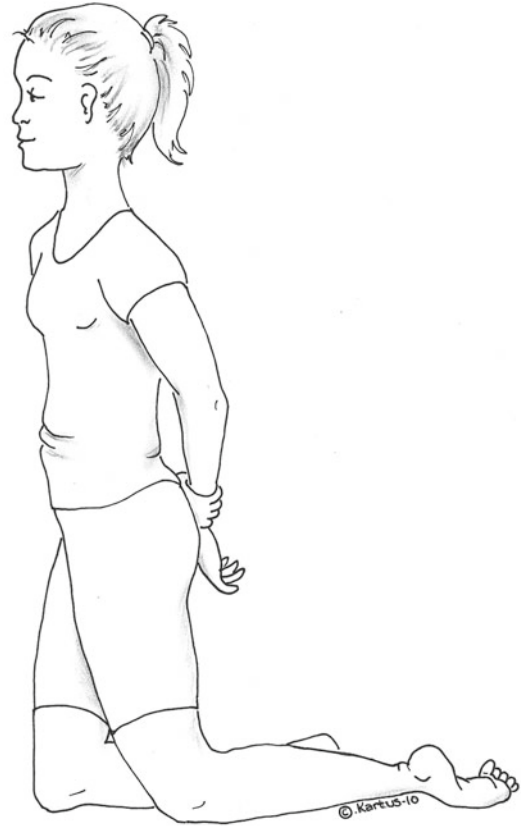


Fig. 17.2 The knee-walking test can be used to determine the discomfort in the anterior knee region after ACL reconstruction (Kartus J, Ejerhed L, Sernert N, et al. Comparison of traditional and subcutaneous patellar tendon harvest. A prospective study of donor site-related problems after anterior cruciate ligament reconstruction using different graft harvesting techniques. *Am J Sports Med.* 2000;28:328–35) (Copyright Catarina Kartus)

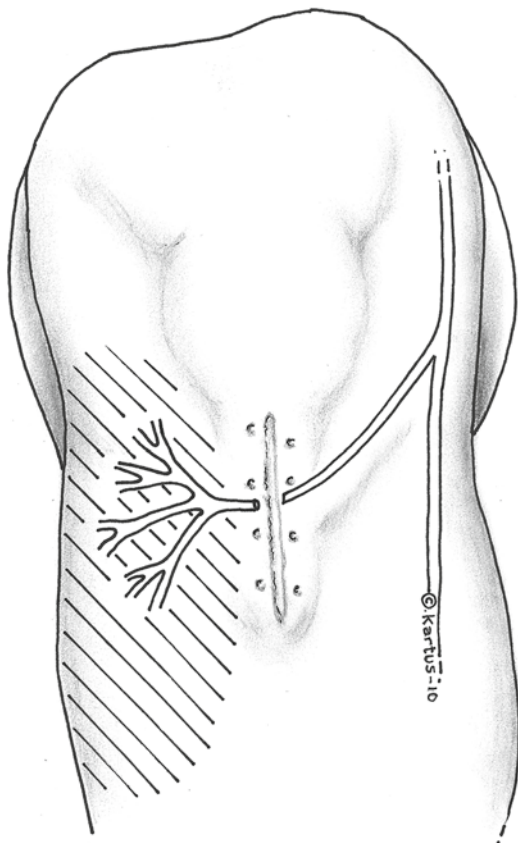


Fig. 17.3 After the use of a central one-incision technique to harvest a patellar tendon autograft, the discomfort during the knee-walking test correlated with the area of disturbed sensitivity in the anterior knee region (Kartus J, Ejerhed L, Sernert N, et al. Comparison of traditional and subcutaneous patellar tendon harvest. A prospective study of donor site-related problems after anterior cruciate ligament reconstruction using different graft harvesting techniques. *Am J Sports Med.* 2000;28:328–35; Kartus J, Magnusson L, Stener S, et al. Complications following arthroscopic anterior cruciate ligament reconstruction. A 2-5-year follow-up of 604 patients with special emphasis on anterior knee pain. *Knee Surg Sports Traumatol Arthrosc.* 1999;7:2–8) (Copyright Catarina Kartus)



Fig. 17.4 The use of the two-incision technique to harvest a patellar tendon autograft resulted in less discomfort during the knee-walking test than the use of the central one-incision technique (Copyright Elsevier Publication)



Fig. 17.5 A persistent donor site gap is displayed on this MRI examination in the axial dimension obtained 26 months after harvesting a central third patellar tendon autograft (Copyright Jüri Kartus)

Fig. 17.6 A persistent donor site gap is displayed on this ultrasonography examination obtained 25 months after harvesting a central third patellar tendon autograft (Copyright Jüri Kartus)

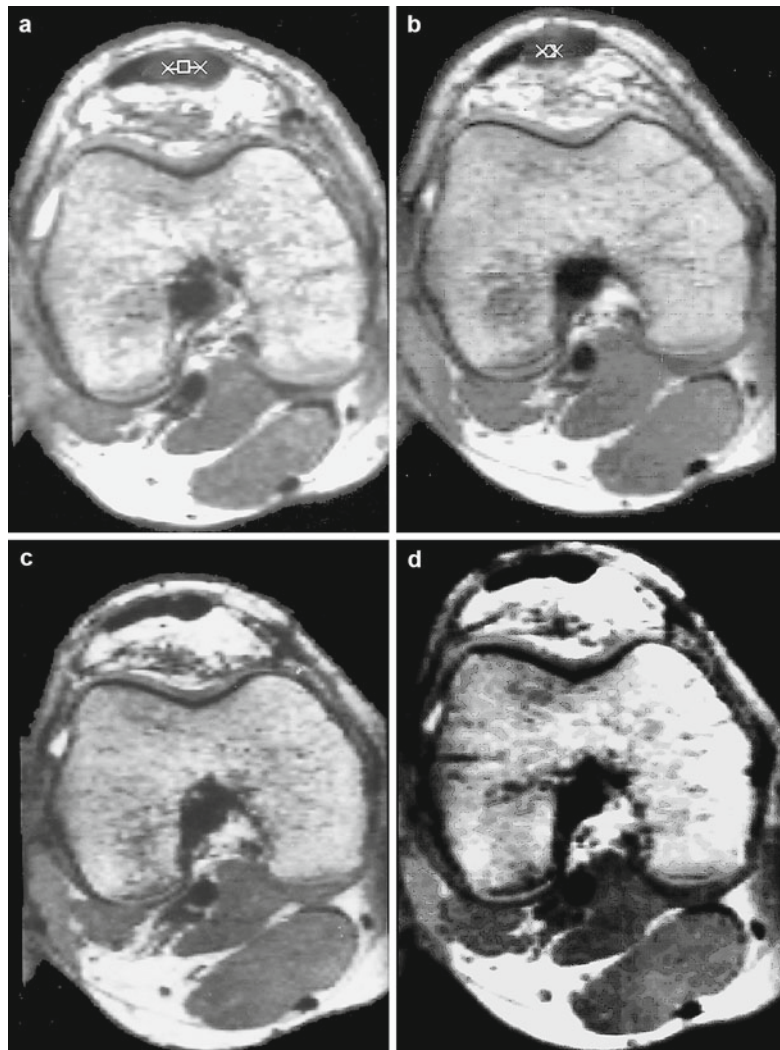
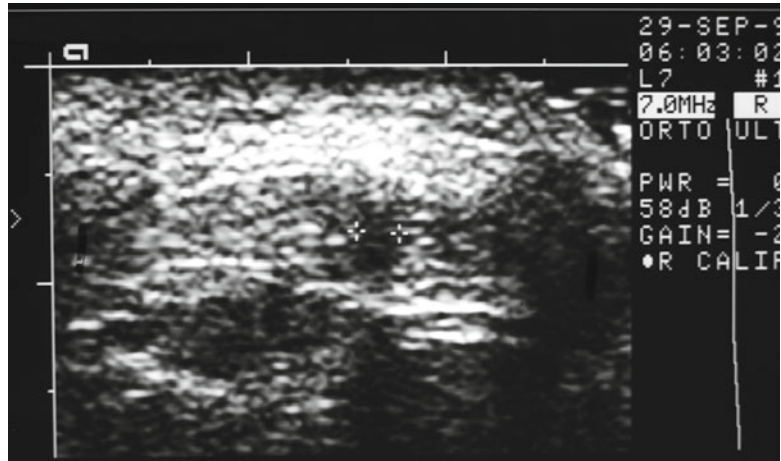


Fig. 17.7 The serial MRI examinations demonstrate that the donor site gap was 7 mm at 6 weeks (a), 2 mm at 6 months (b) and completely healed at 27 months and 6 years (c) and (d). Furthermore, the thickness of the patellar tendon decreased over time. This is a male patient who, at the time of the index operation, was 18 years old (Copyright Jüri Kartus)

Fig. 17.8 A high-power view of a biopsy obtained from the central part of the patellar tendon 6 years after the harvesting procedure showing increased cellularity, vascularity and non-parallel fibers (Kartus J, Movin T, Papadogiannakis N, et al. A radiographic and histologic evaluation of the patellar tendon after harvesting its central third. *Am J Sports Med.* 2000;28:218–26) (Hematoxylin and eosin staining; original magnification, $\times 200$) (Copyright Springer-Verlag)

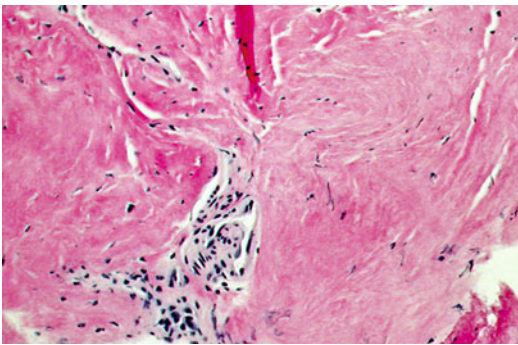
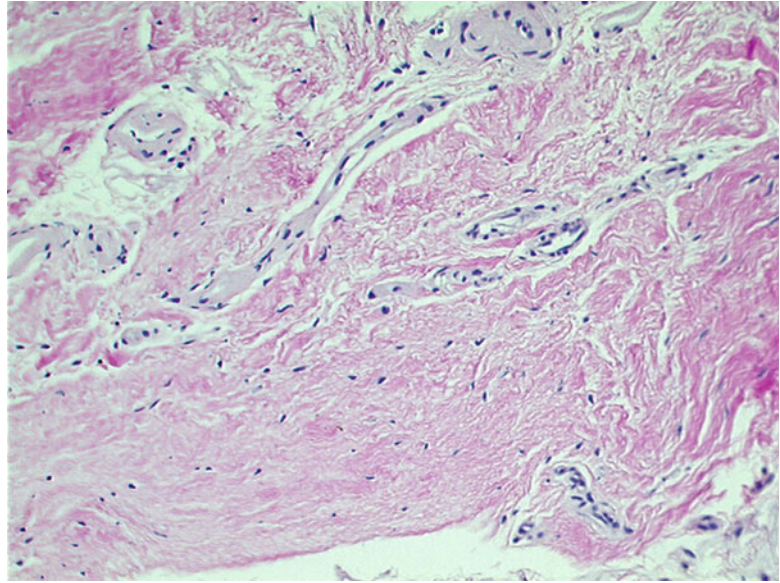


Fig. 17.9 A high-power view of a biopsy obtained from the peripheral part of the patellar tendon 6 years after the harvesting procedure showing increased cellularity, vascularity and non-parallel fibers (Kartus J, Movin T, Papadogiannakis N, et al. A radiographic and histologic evaluation of the patellar tendon after harvesting its central third. *Am J Sports Med.* 2000;28:218–26) (Hematoxylin and eosin staining; original magnification, $\times 200$) (Copyright Springer-Verlag)

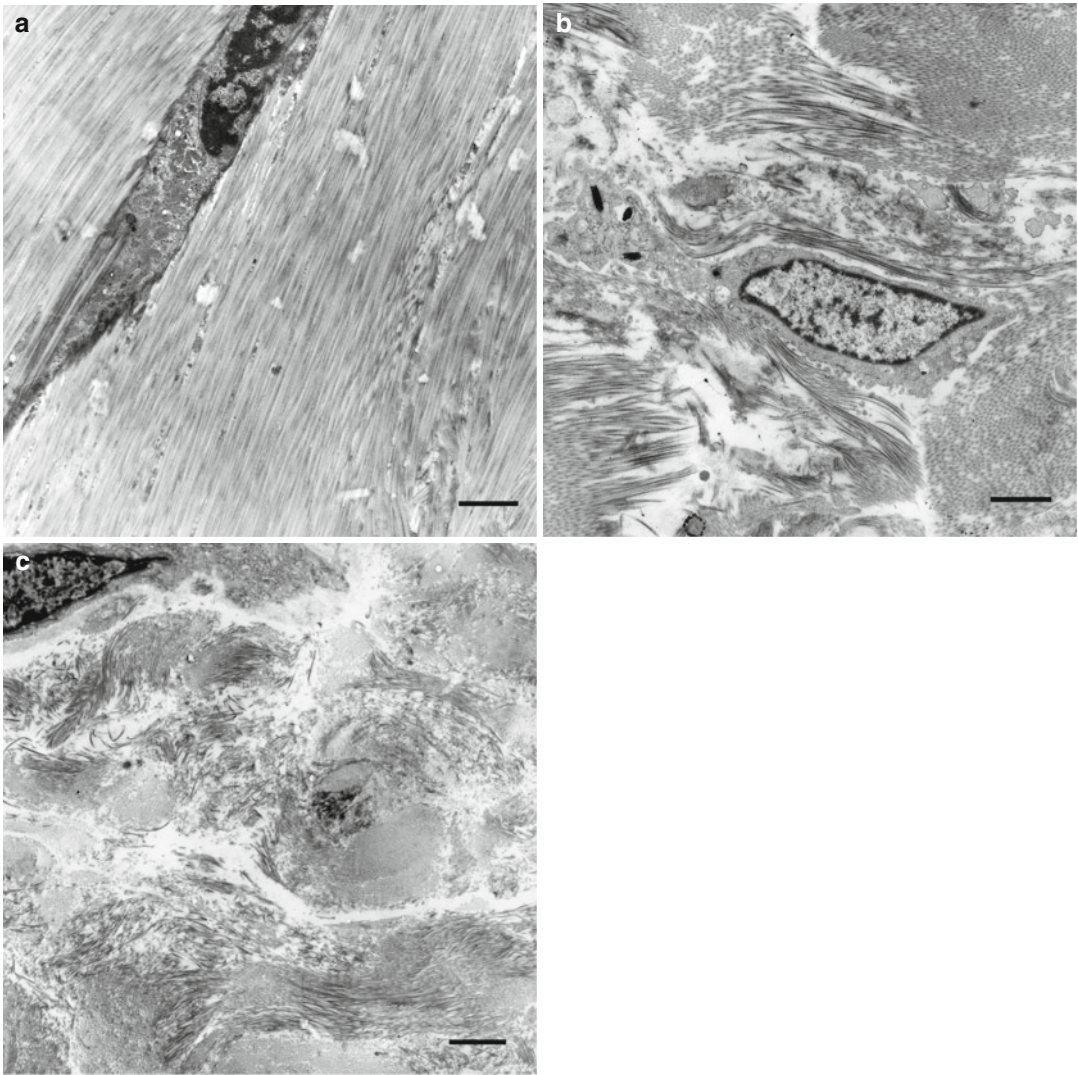


Fig. 17.10 Transmission electron micrographs from control tendons (a), lateral parts (b), and central parts (c) of the tendons in the study group 6 years after the harvesting procedure. The fibrils were less regularly orientated in

both the central and lateral part of the harvested tendon compared with normal tendon (Bar = 2 mm, original magnification, $\times 3,000$) (Copyright Jüri Kartus)

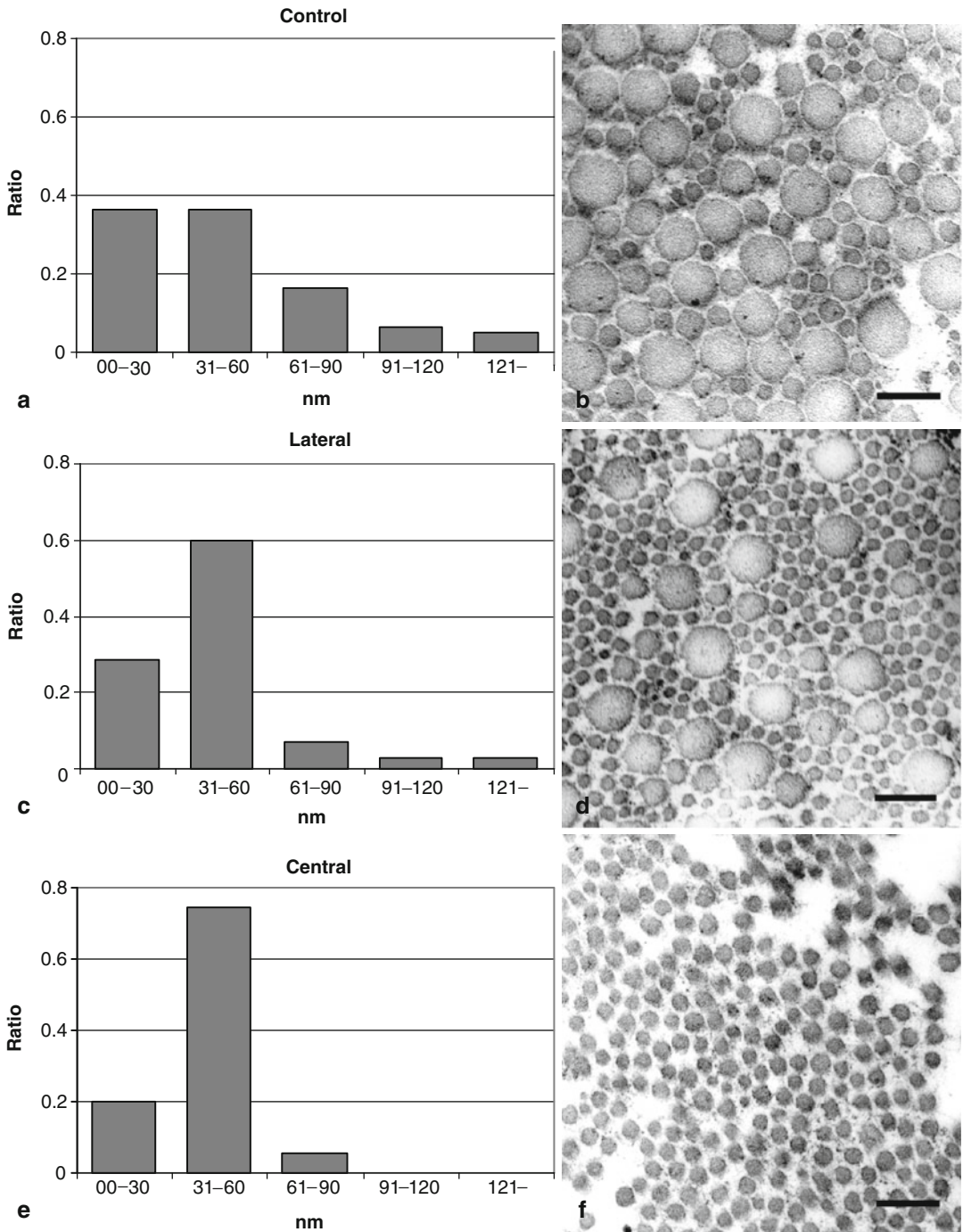


Fig. 17.11 Relative distribution and transmission electron micrographs of the fibril diameter size in human patellar tendon from controls (a, b), lateral (c, d) and central parts (e, f) 6 years after the harvesting procedure (Bar = 200 nm, original magnification, $\times 101,000$). These figures show that there was a significant difference in fibril

size distribution between the groups (Svensson M, Movin T, Rostgard-Christensen L, et al. Ultrastructural collagen fibril alterations in the patellar tendon 6 years after harvesting its central third. *Am J Sports Med.* 2007;35:301-6) (Copyright Jüri Kartus)

Part II

Emerging Technologies for Investigating Patellofemoral Joint. Clinical Relevance

Imaging and Musculoskeletal Modeling to Investigate the Mechanical Etiology of Patellofemoral Pain

18

Thor F. Besier, Christine Draper, Saikat Pal,
Michael Fredericson, Garry Gold, Scott Delp,
and Gary Beaupré

The combination of advanced medical imaging and musculoskeletal modelling presented here provides us with a unique set of tools to investigate the complex form and function of the PF joint. In particular, the ability to

estimate patient-specific stresses throughout various tissues of the PF joint enables us to test the fundamental hypothesis that the onset and development of PF pain has an underlying mechanical aetiology.

T.F. Besier, Ph.D. (✉)
Auckland Bioengineering Institute,
The University of Auckland,
Auckland, New Zealand

Department of Orthopaedics, Stanford University,
Stanford, CA, USA
e-mail: t.besier@auckland.ac.nz

C. Draper, Ph.D. • S. Pal, Ph.D. • S. Delp, Ph.D.
Department of Bioengineering, Stanford University,
Stanford, CA, USA

M. Fredericson, M.D.
Department of Orthopaedics, Stanford University,
Stanford, CA, USA

G. Gold, M.S., M.D.
Department of Radiology, Stanford University,
Stanford, CA, USA

G. Beaupré, Ph.D.
VA Rehabilitation Research and Development Center,
Palo Alto, CA, USA

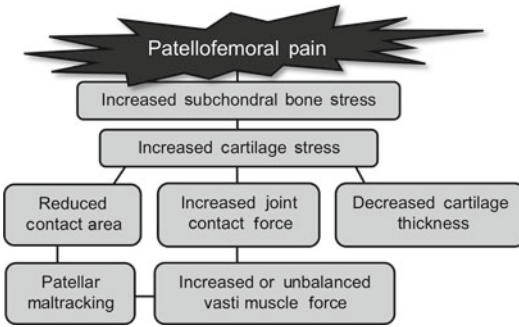


Fig. 18.1 Factors that may contribute to increased cartilage and subchondral bone stress and patellofemoral pain

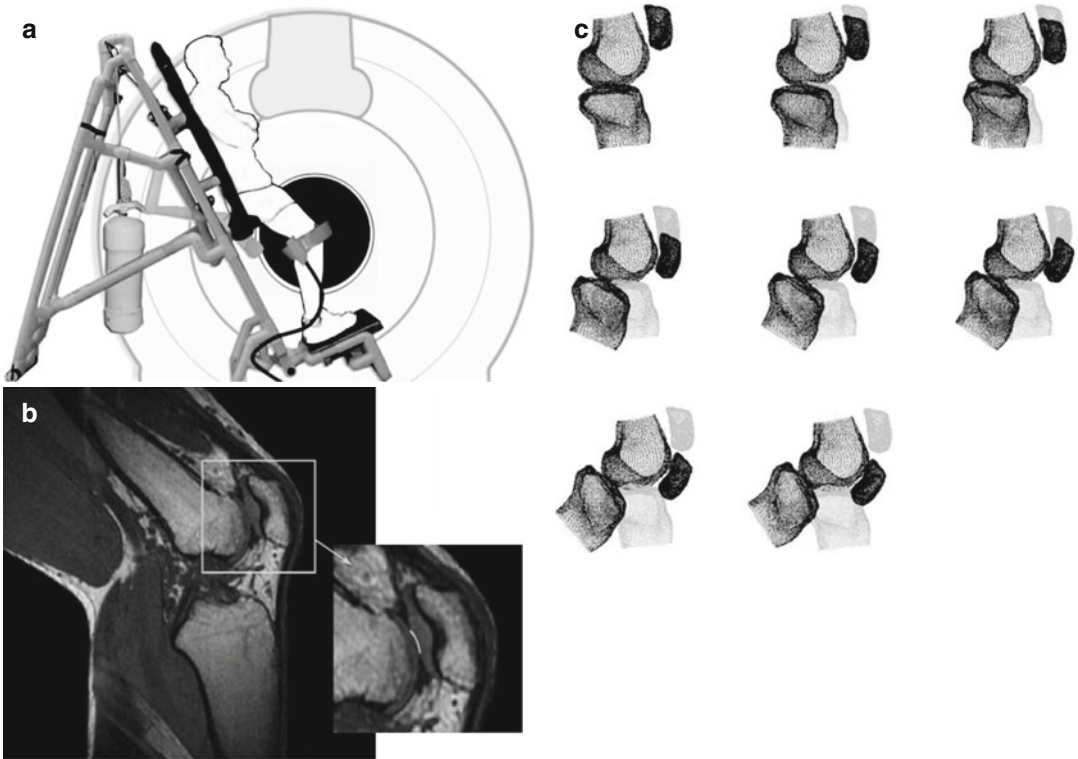


Fig. 18.2 Upright weight-bearing imaging in the 0.5T GE Signa MRI scanner. The custom backrest (a) enables subjects to remain still during the scan, while supporting ~90% of their body weight. The backrest can be locked into place and a small seat can be engaged from behind to enable images to be taken under minimal load and no

quadriceps activity (~0.15 body weight). Volumetric images of the knee can then be used to determine contact areas (b) and the three-dimensional orientation of the patellofemoral joint through different amounts of knee flexion (c)

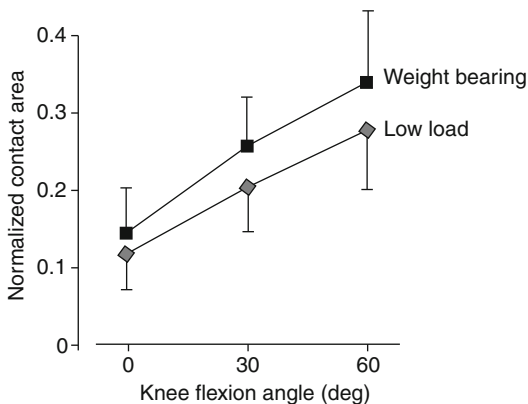


Fig. 18.3 Normalized patellofemoral joint contact areas under loaded and unloaded conditions (Adapted from Besier T, Delp S, Gold G, et al. Patellofemoral pain subjects display greater femoral cartilage stresses than pain-free controls. In: Patellofemoral pain syndrome: international research retreat. Baltimore: Elsevier; 2009)

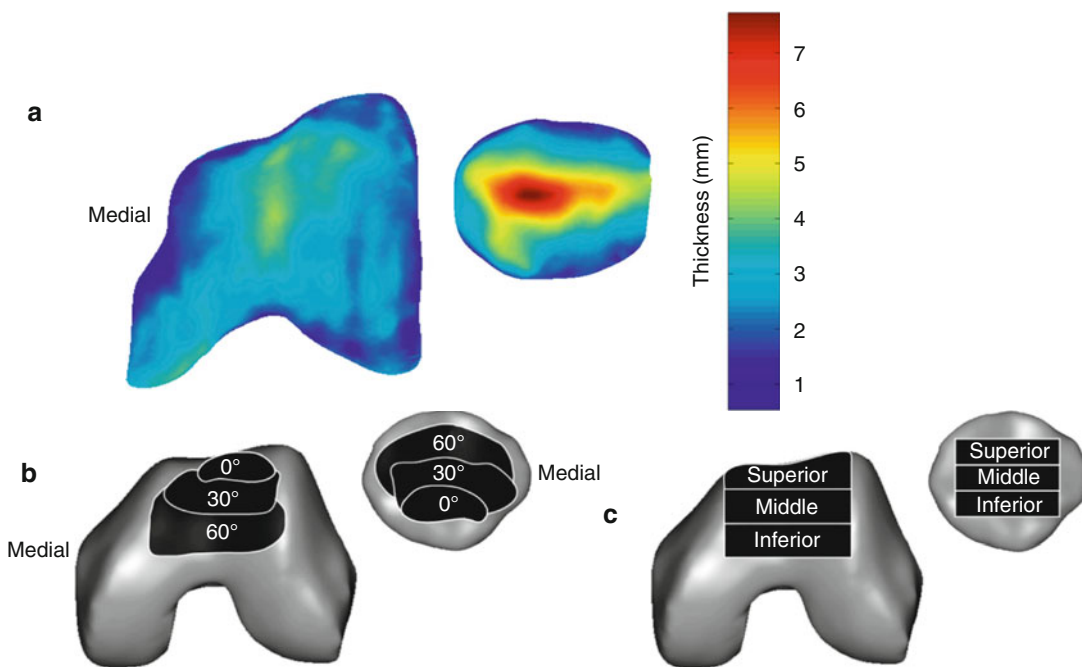


Fig. 18.4 (a) Cartilage thickness map of the anterior femur and patella. (b) Contact areas corresponding to 0°, 30°, and 60° of knee flexion. (c) Discretized regions where cartilage thicknesses were examined (Adapted from

Draper CE, Besier TF, Gold GE, et al. Is cartilage thickness different in young subjects with and without patellofemoral pain? *Osteoarthritis Cartilage*. 2006;14:931–7)

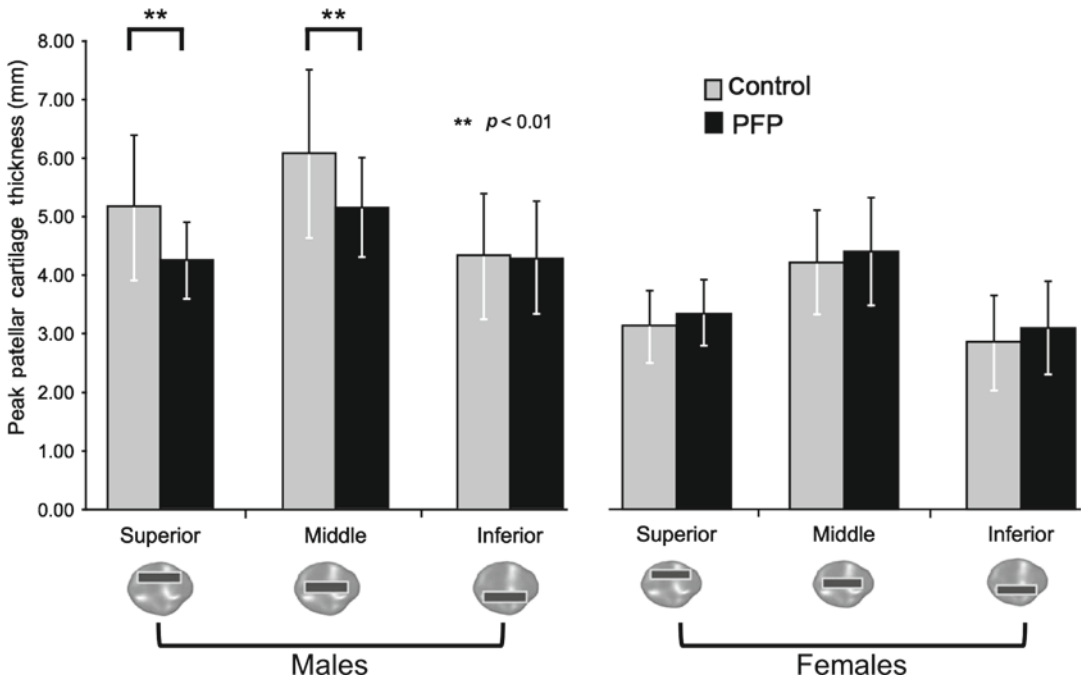


Fig. 18.5 Peak patellar cartilage thickness of males and females with patellofemoral pain (PFP) and a group of pain-free controls. Males with patellofemoral pain had thinner cartilage compared to controls, which might lead

to increased cartilage stresses, particularly at deeper angles of knee flexion when the contact is superior and middle

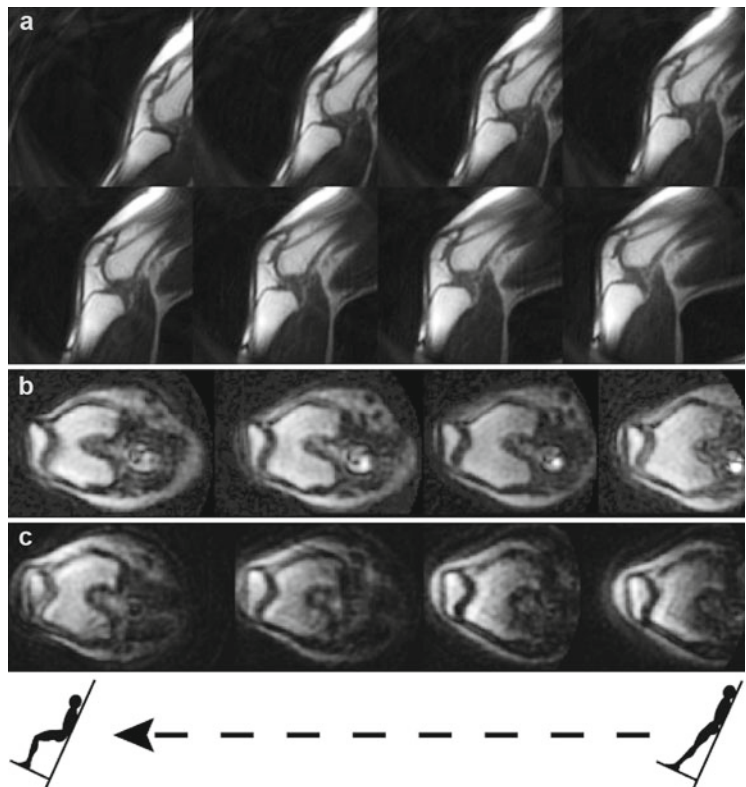


Fig. 18.6 (a) Sample sagittal plane real-time MR images of patellofemoral joint during weight-bearing knee flexion. (b) Axial images from healthy, pain-free control and (c) axial images from a subject with patellofemoral pain

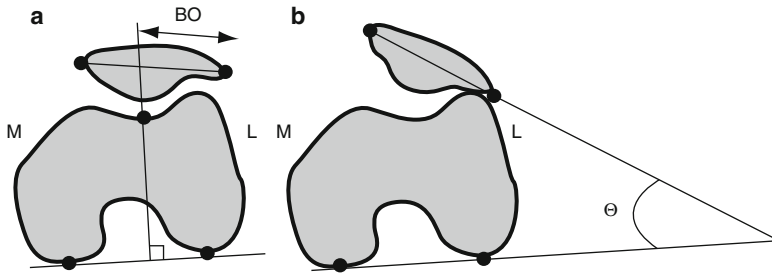


Fig. 18.7 Axial-plane PF joint kinematics illustrating: (a) the Bisect Offset index (BO) – a measure of the percentage of the patellar width lateral to the midline of the femur, and (b) patellar tilt (theta) – the angle formed by lines joining the posterior femoral condyles and the maxi-

mum width of the patella (Adapted from Draper CE, Besier TF, Gold GE, et al. Is cartilage thickness different in young subjects with and without patellofemoral pain? *Osteoarthritis Cartilage*. 2006;14:931–7)

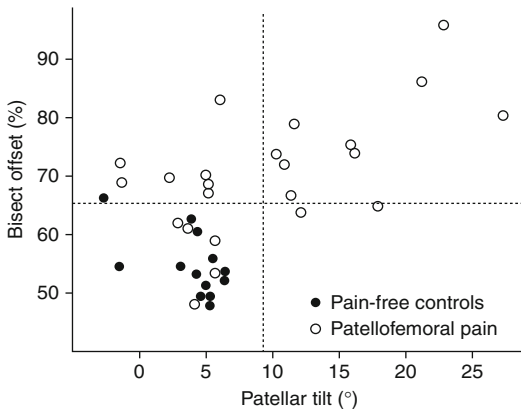


Fig. 18.8 Relationship between bisect offset and patellar tilt at full extension in pain-free controls (*solid circles*) and patellofemoral pain subjects (*hollow circles*). The *dashed lines* represent two standard deviations above the average bisect offset and tilt of the pain-free subjects and were used as thresholds to identify subjects with abnormal patellofemoral joint kinematics (Adapted from Draper CE, Besier TF, Gold GE, et al. Is cartilage thickness different in young subjects with and without patellofemoral pain? *Osteoarthritis Cartilage*. 2006;14:931–7)

Fig. 18.9 Co-registered axial PET/CT image of a unilateral chronic PF pain patient (Male, age 32, characterized with abnormal weight-bearing bisect offset index at full extension). The superimposed CT image enables accurate localization of the PET hotspot, in this case within the apex of the left patella, which was consistent with the area of pain

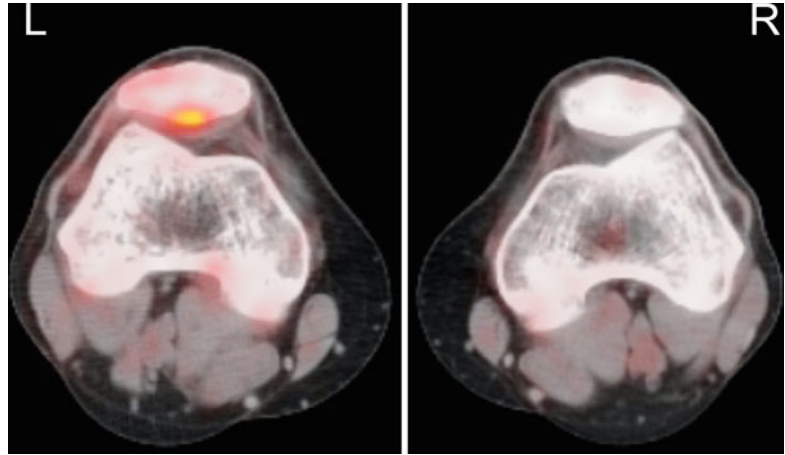


Fig. 18.10 Axial MRI of chronic PF pain patient (a), showing no abnormalities within bone or cartilage of the PF joint. Corresponding PET hotspot in the same subject (b), indicating areas of high metabolic activity

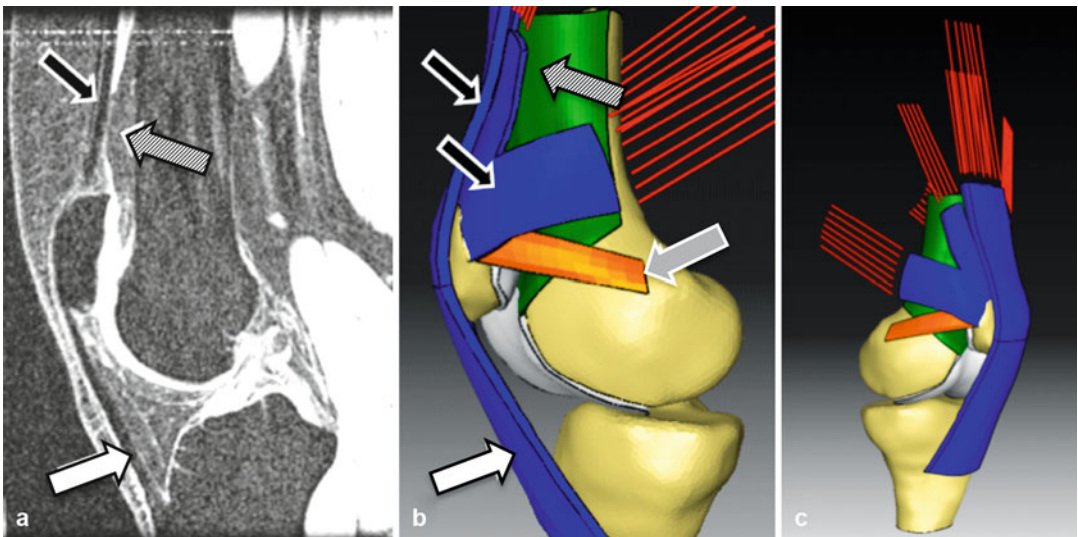
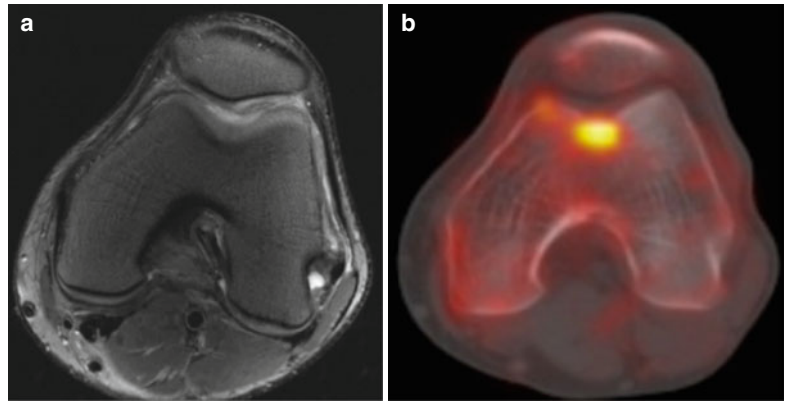


Fig. 18.11 Sagittal MR image (a) and corresponding finite element model (b, c) of the patellofemoral joint. The patellar ligament (white arrows) and quadriceps tendon (black arrows) were represented as nonlinear fiber-reinforced

solid elements, while the medial patellofemoral ligament (gray arrow) was modeled using 2D composite elements. Articular cartilage and supra-trochlear fat pad (striped arrows) were modeled as linear elastic solids

Fig. 18.12 Axial CT image of chronic PF pain patient illustrating variation in bone mineral density. Color coding on right shows most dense bone (*red*) in the anterior aspect of the patella as well as the lateral facet of the patella

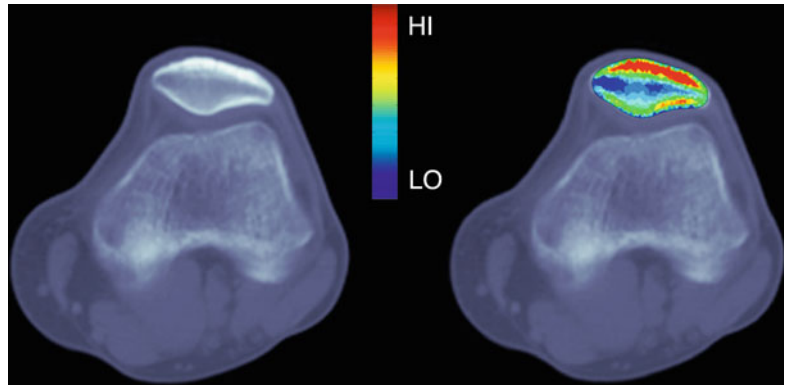
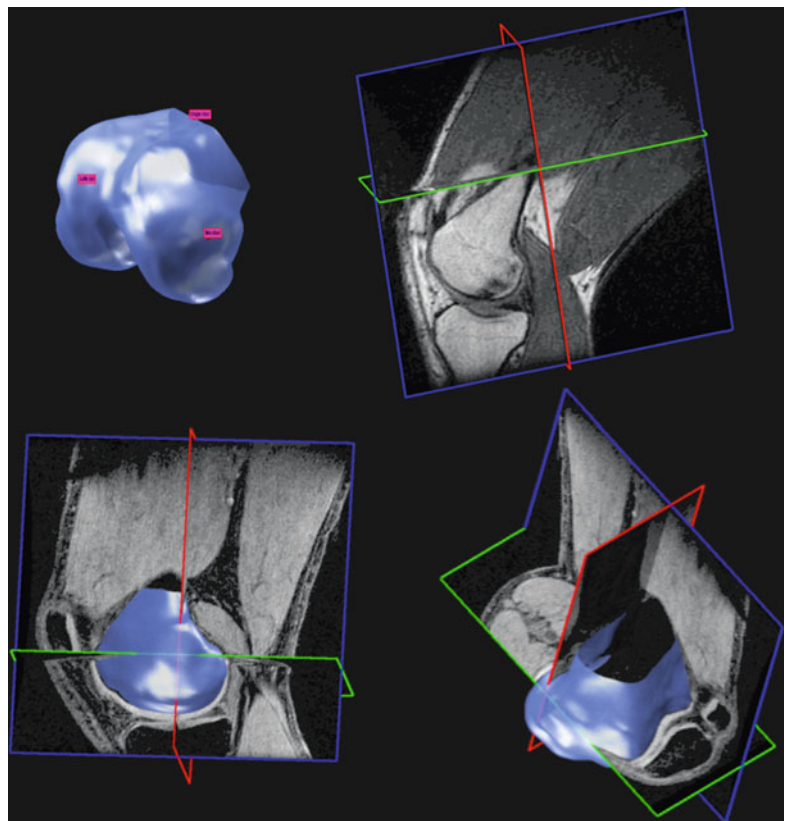


Fig. 18.13 Registration of femur finite element mesh (*upper left*) into upright weight-bearing MR imaging volume (*upper right*). Selecting edges of the bone within the imaging data set ensures a close match between the model and MR images (*lower images*)



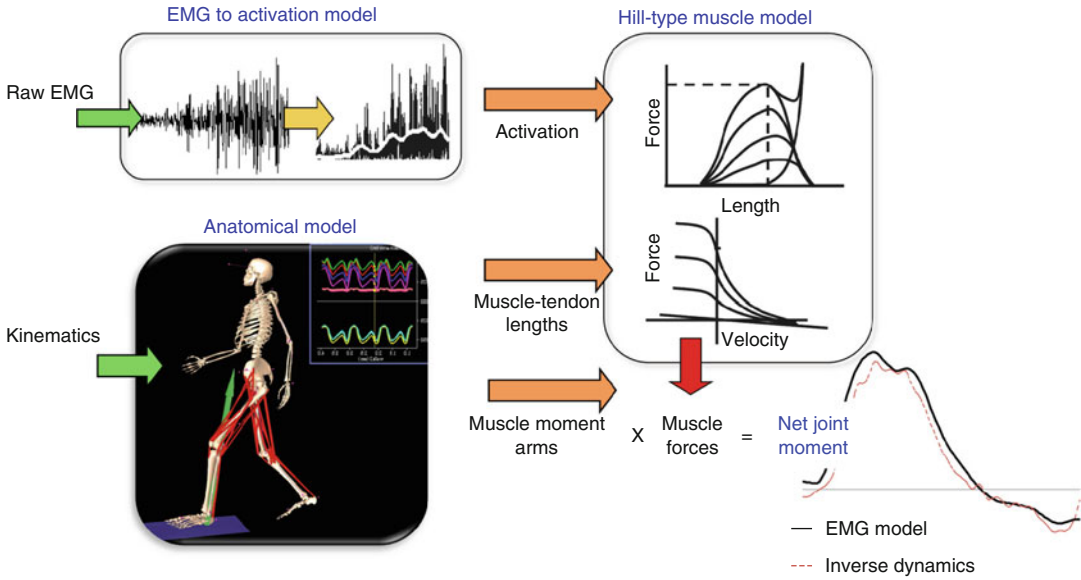


Fig. 18.14 EMG-driven musculoskeletal model overview. Raw EMG and joint kinematics are used to estimate activation and muscle tendon lengths, which are input into a Hill-type muscle model to estimate muscle force. Muscle moment arms calculated from the anatomical model

(OpenSIM) are multiplied by the muscle forces to obtain the net joint moment. The net joint moment from the model is compared to the moment calculated from inverse dynamics in a calibration/validation procedure

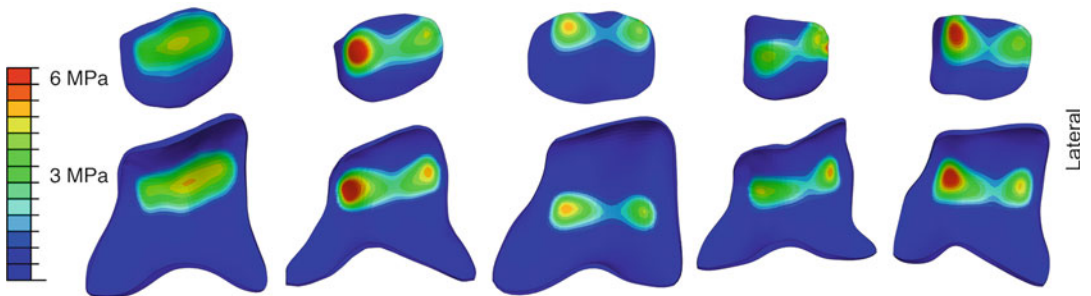


Fig. 18.15 Example hydrostatic stresses in the layer of patellar and femoral cartilage closest to the subchondral bone in five patients with PF pain during a static squat at 60° of knee flexion. Note the varied distribution and mag-

nitudes of peak hydrostatic pressure across this small sample. The lateral aspect of the joint is toward the right on each example. Stress “hot spots” are common on the medial aspect of the PF joint cartilage

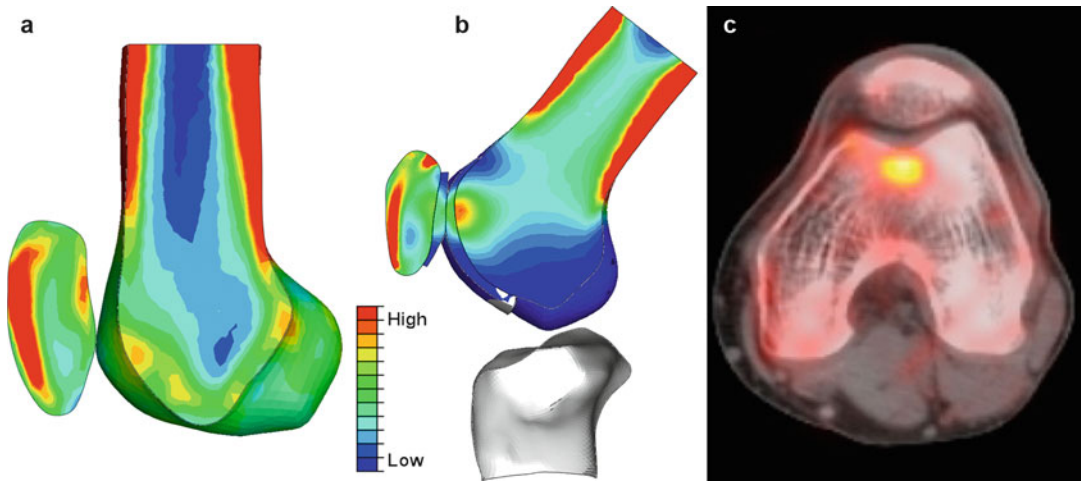


Fig. 18.16 Bone density assignment from CT-based Hounsfield Units (a), and predicted bone-cartilage stress from finite element modeling (b) during a 60° static squat. Peak subchondral bone stresses in the trochlea of the femur correspond to the hot spot from PET scanning on the same subject (c)

Computational Models for Investigating the Patellofemoral Joint: Clinical Relevance

19

John J. Elias and Andrew J. Cosgarea

Computational models of the patellofemoral joint are commonly utilized to characterize patellofemoral kinematics, areas of contact, and pressure applied to patellofemoral cartilage. Computational models can provide enhanced display of patellofemoral kinematics by graphically recreating motions performed by subjects. The reconstructed motions are typically highly accurate, but studies can be limited by the imaging requirements and the recruitment of subjects. Graphical models of

joint anatomy can also be combined with mathematical representations of the soft tissues surrounding the joint to predict the desired output. These models provide more flexibility for parametric studies focused on the influence of surgical and nonsurgical treatment methods on patellofemoral biomechanics, but the mathematical approximations raise concerns about accuracy. Thorough validation is required for these models.

J.J. Elias, Ph.D. (✉)
Calhoun Research Lab,
Akron General Medical Center,
Akron, OH, USA
e-mail: john.elias@akrongeneral.org

A.J. Cosgarea, M.D.
Department of Orthopaedic Surgery,
Johns Hopkins University,
Baltimore, MD, USA

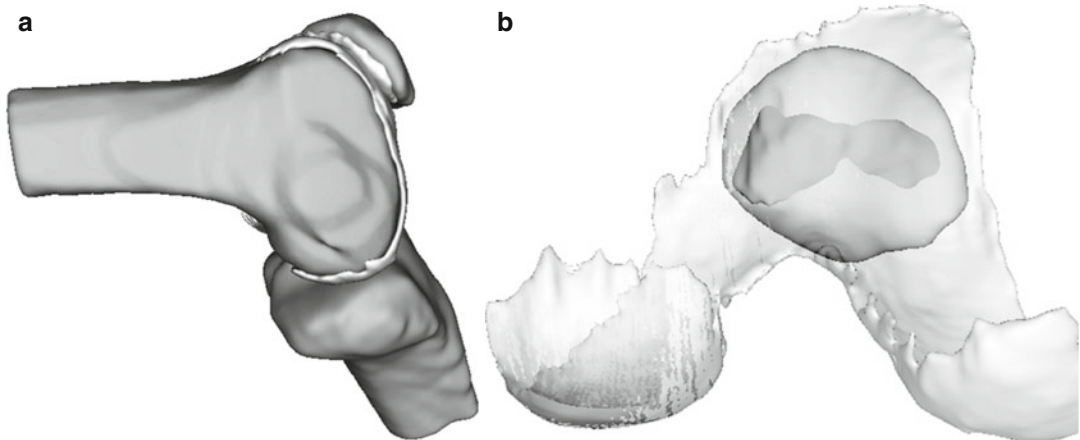


Fig. 19.1 Graphical representation of a flexed knee, showing the femur, patella, tibia, and the cartilage on the femur and patella (a). Manipulating the model to remove

the femur and make the cartilage on the femur transparent allows visualization of the area of patellofemoral contact (b)

Fig. 19.2 Representation of a subject in a MRI scanner for a study focused on enhanced visualization of patellofemoral kinematics. Muscle loading is induced by applying a force to the foot plate, which is connected to the weight outside of the scanner

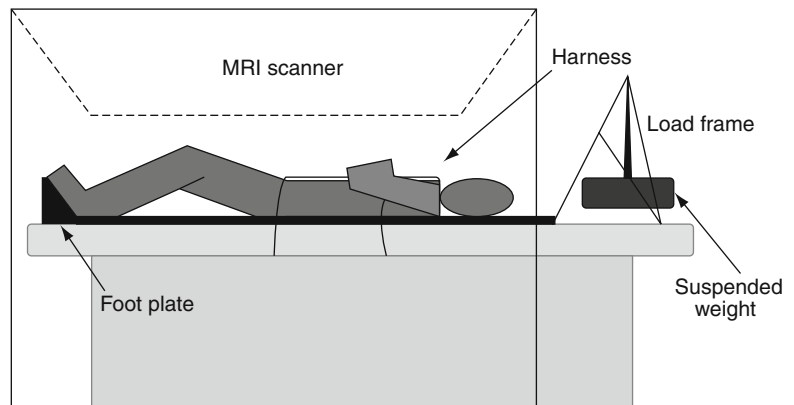


Fig. 19.3 For the dual-orthogonal fluoroscopic image system used for enhanced visualization of patellofemoral kinematics, a virtual system is developed to align computational models of knees constructed from MRI data with the images acquired with the fluoroscopy units (From Nha KW, Papannagari R, Gill TJ, et al. In vivo patellar tracking: clinical motions and patellofemoral indices. *J Orthop Res.* 2008;26:1067–74. With permission)

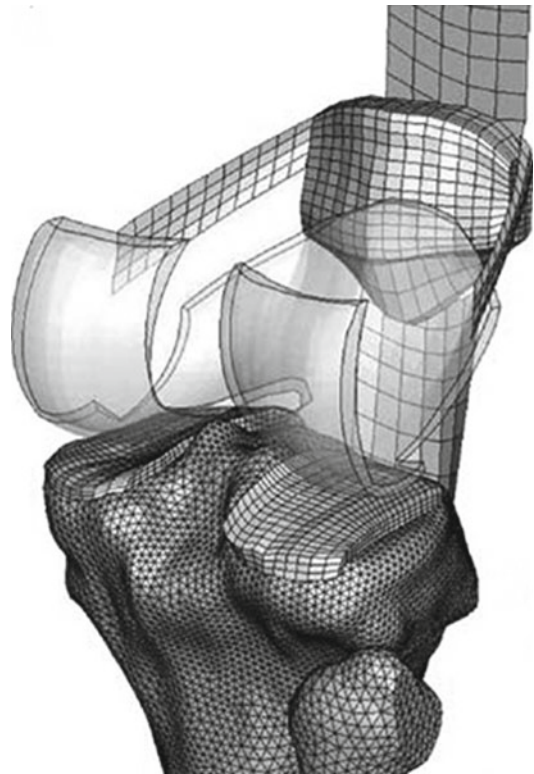
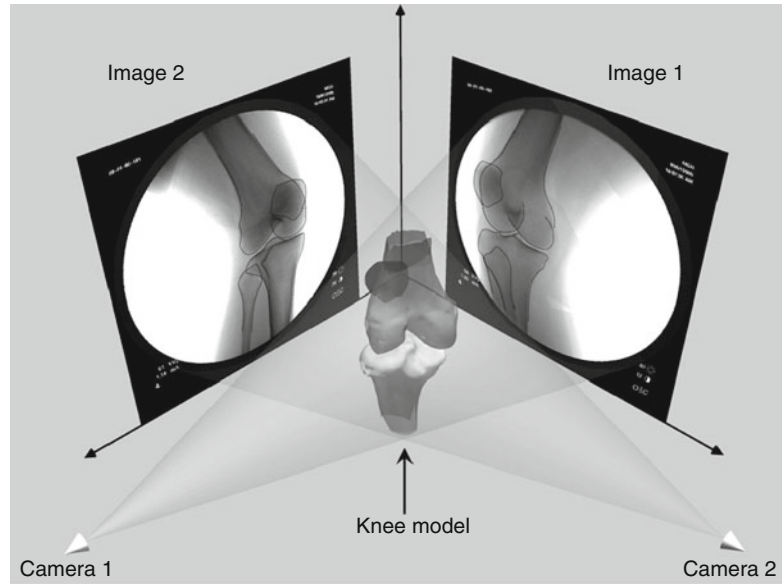


Fig. 19.4 Representation of a finite element model of the patellofemoral joint, showing the mesh assigned to the tibia and patella, as well as the cartilage on the tibia and patella. Meshes are also shown for the representation of the quadriceps tendon, the patella tendon, and the medial and lateral patellofemoral ligaments. The femur and mesh on the femoral cartilage have been removed for clarity (Reprinted from Baldwin MA, Clary C, Maletsky LP, et al. Verification of predicted specimen-specific natural and implanted patellofemoral kinematics during simulated deep knee bend. *J Biomech.* 2009;42:2341–8. With permission from Elsevier)

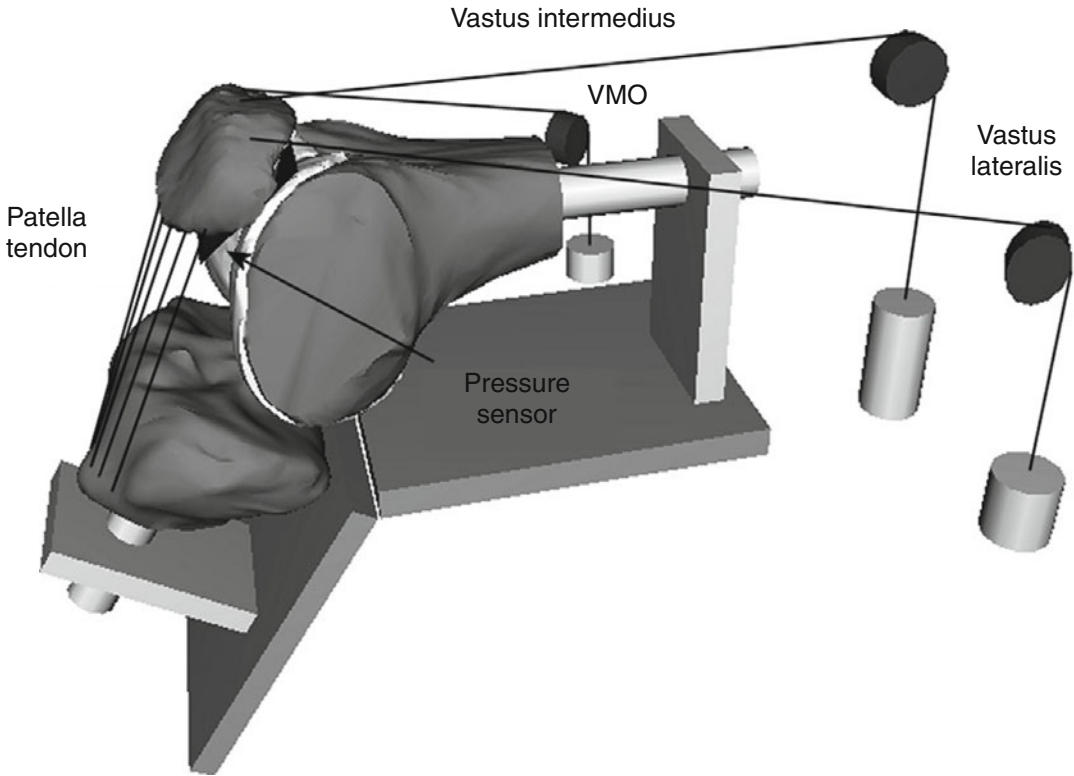


Fig. 19.5 Computational model of a flexed knee, embedded in a graphical representation of an in vitro testing frame used to validate the computational model. The loading cables used to apply forces through the quadriceps muscles are represented, along with representations of the pulleys and weights. The line segments representing

the patella tendon are also shown, along with the position of the pressure sensor used to experimentally measure patellofemoral pressures (Adapted from Elias JJ, Cech JA, Weinstein DM, et al. Reducing the lateral force acting on the patella does not consistently decrease patellofemoral pressures. *Am J Sports Med.* 2004;32:1202–8)

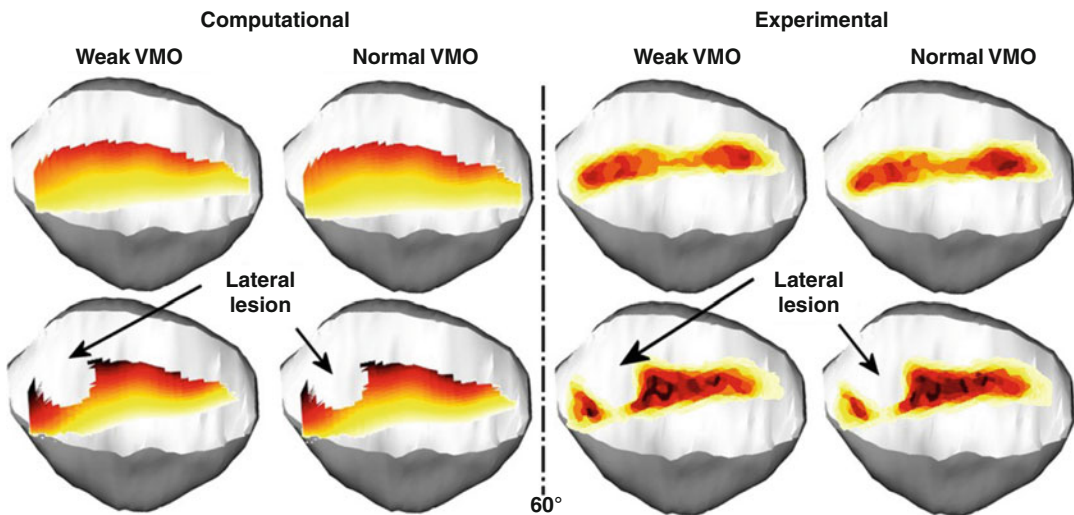


Fig. 19.6 Computationally determined and experimentally determined pressure patterns superimposed over a graphical representation of the patella for one knee at 60° of flexion. The pressure distribution is shown for a weak VMO and a normal VMO, for intact cartilage and cartilage with a

lateral lesion. Decreasing the force applied by the VMO shifts pressure laterally on the patella (Reprinted from Elias JJ, Cech JA, Weinstein DM, et al. Reducing the lateral force acting on the patella does not consistently decrease patellofemoral pressures. *Am J Sports Med.* 2004;32:1202–8)

Kinetic Analysis: A Sensitive Outcome Objective Measurement Method in Evaluating Lateral Patellar Instability

A Preliminary Study

Vicente Sanchis-Alfonso, José María Baydal-Bertomeu, Erik Montesinos-Berry, Andrea Castelli, and José David Garrido-Jaén

Precise, repeatable, and objective outcome tools are necessary to refine surgical procedures so that short-term and long-term knee function and patient satisfaction are improved. Our study supports the fact that kinetic analysis using dynamometric platforms could be a useful tool in the objective evaluation of patients with lateral patellar instability under realistic loading conditions (evaluation of functional instability). With this

task we duplicate: (1) muscle forces, (2) weight-bearing conditions, and (3) rotational loads caused by sports gestures (higher than the load applied to the knee during clinical examination).

Kinetic analysis has some limitations: (1) not useful in acute injuries, (2) in the presence of marked muscle wasting, and (3) when both knees are involved. However, our data are only a “snapshot” analysis from an ongoing study.

V. Sanchis-Alfonso, M.D., Ph.D. (✉)
International Patellofemoral Study Group,
ACL Study Group, Hospital 9 de Octubre,
Hospital Arnau de Vilanova, School of Medicine,
Valencia Catholic University,
Valencia, Spain
e-mail: vicente.sanchis.alfonso@gmail.com

J.M. Baydal-Bertomeu, Mech. Eng. • A. Castelli,
Biomed. Eng. • J. D. Garrido-Jaén, Mech. Eng.
Instituto de Biomecánica de Valencia (IBV),
Universidad Politécnica de Valencia,
Valencia, Spain

E. Montesinos-Berry, M.D.
Hospital de Manises,
Valencia, Spain

Fig. 20.1 A moment is a force couple that produces a rotator effect

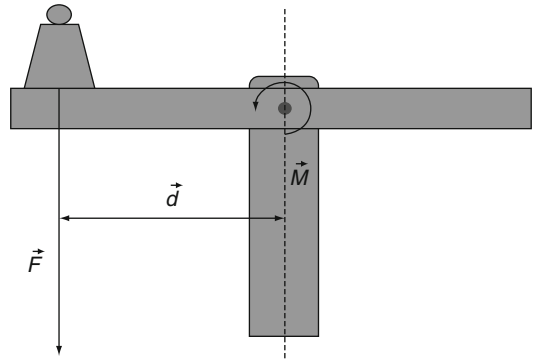


Fig. 20.2 Mechanism of lateral patellar dislocation



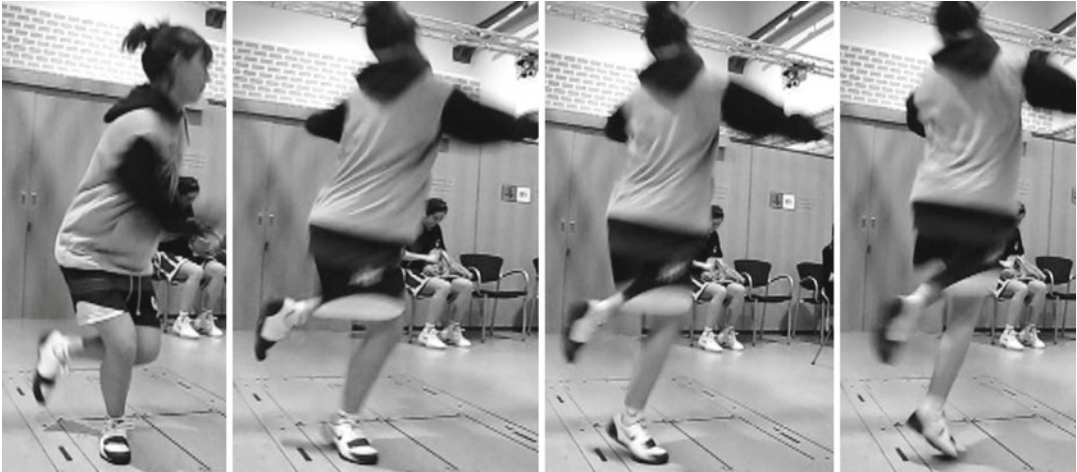


Fig. 20.3 Image sequence of the monopodal jumping with pivoting with external tibial rotation test

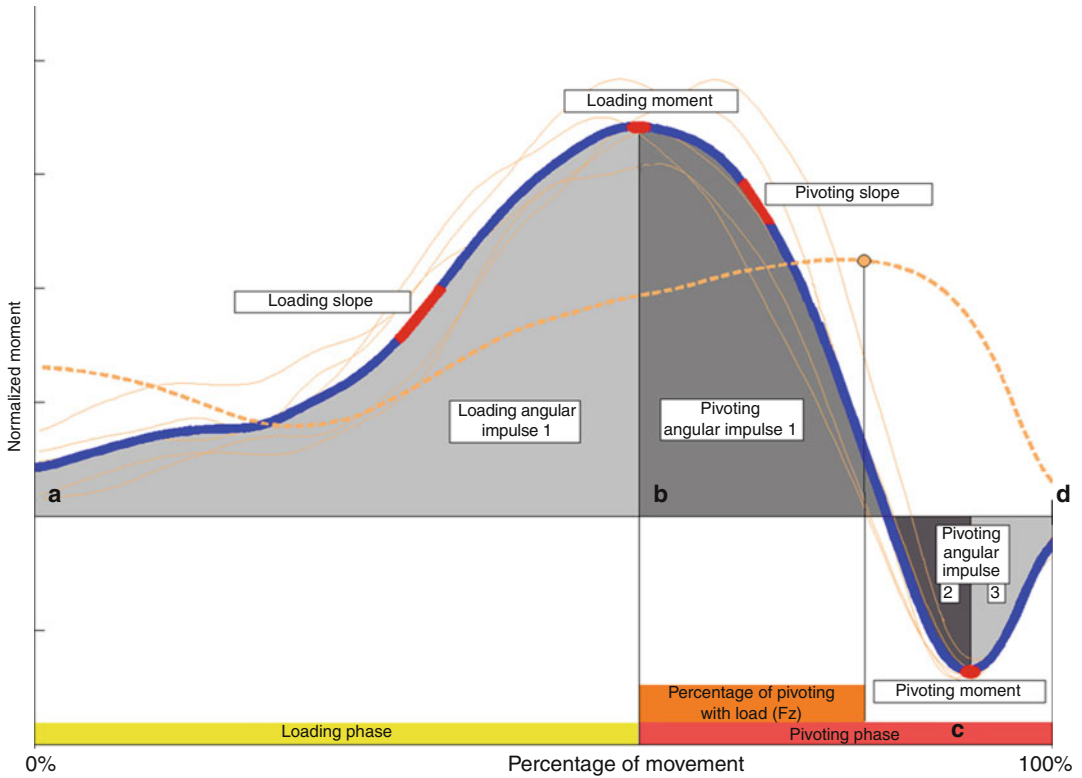
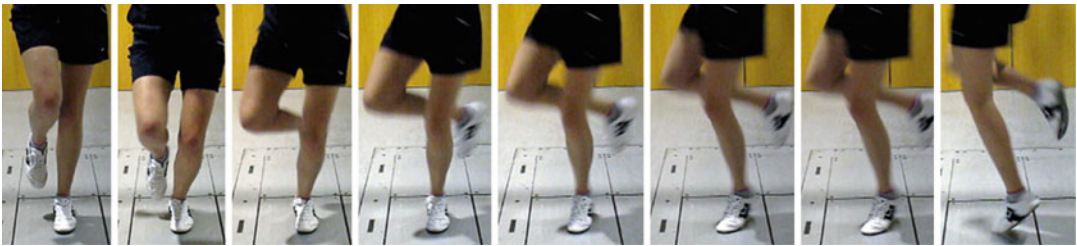


Fig. 20.4 Curve representing the normalized moments and the vertical reaction force (F_z) registered during the jumping with pivoting with external tibial rotation test



Fig. 20.4 (continued)

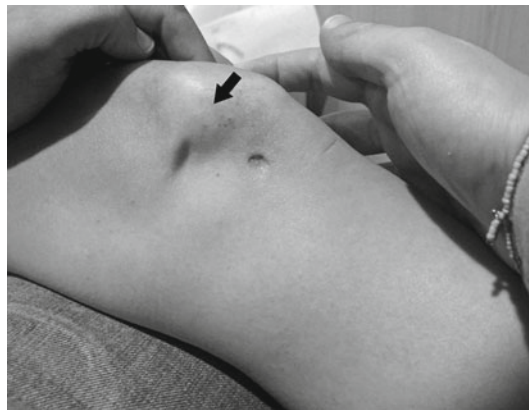
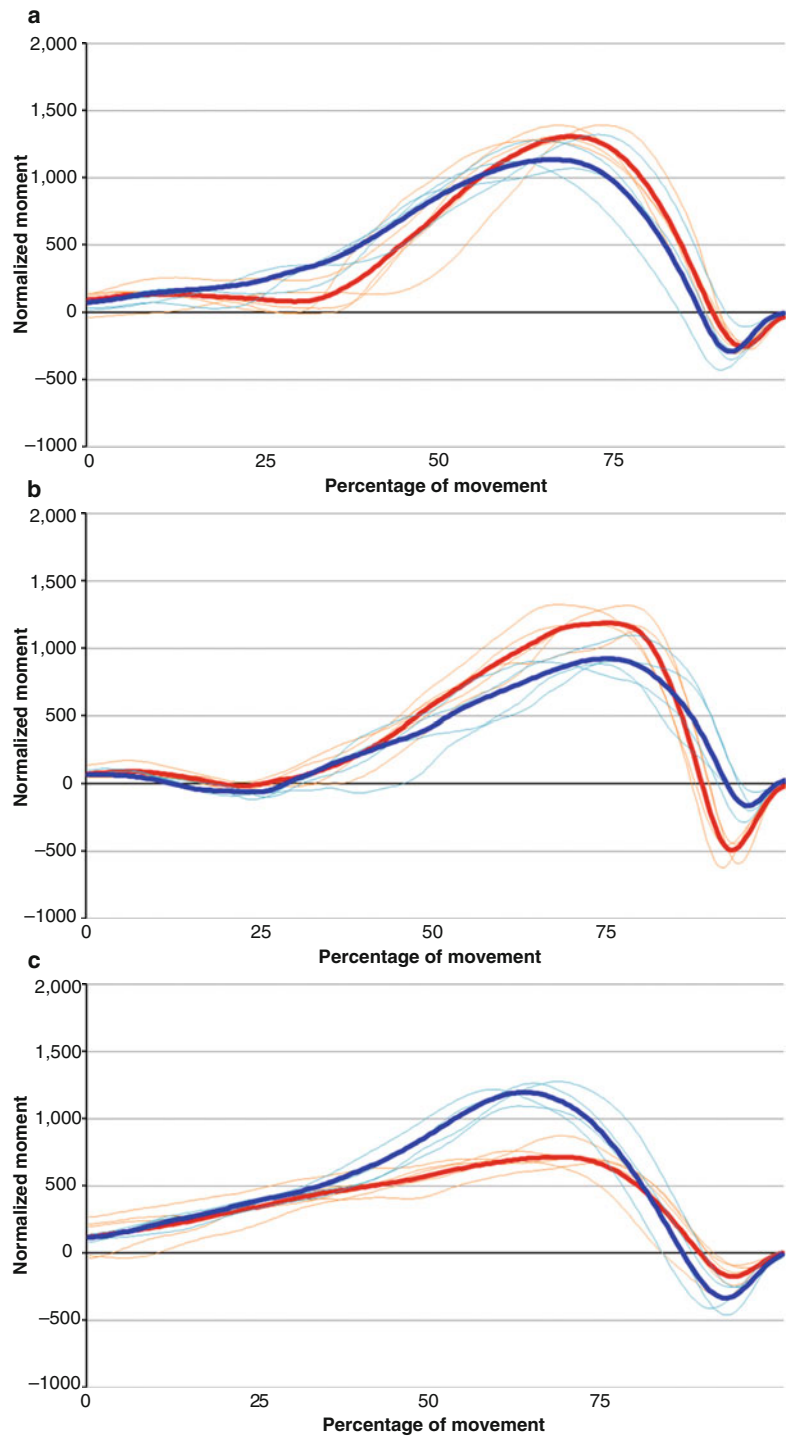


Fig. 20.5 MPFL is a restraint to medial patellar displacement

Fig. 20.6 Normalized moments registered during the monopodal jumping with pivoting with external tibial rotation test. **(a)** Normal knees. **(b)** Isolated ACL tear of the left knee (*blue line*). **(c)** Isolated lateral patellar instability of the right knee (*red line*)



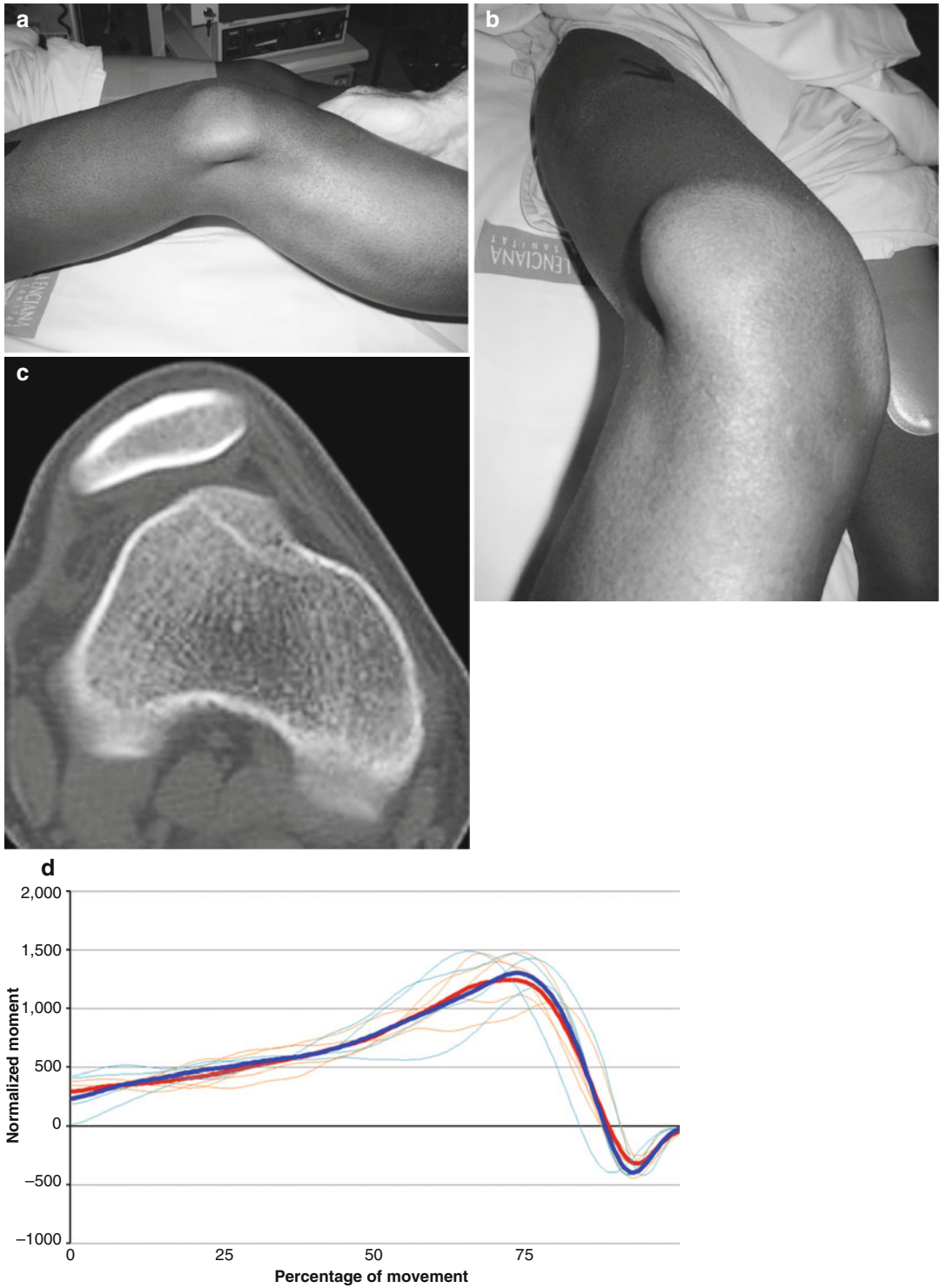
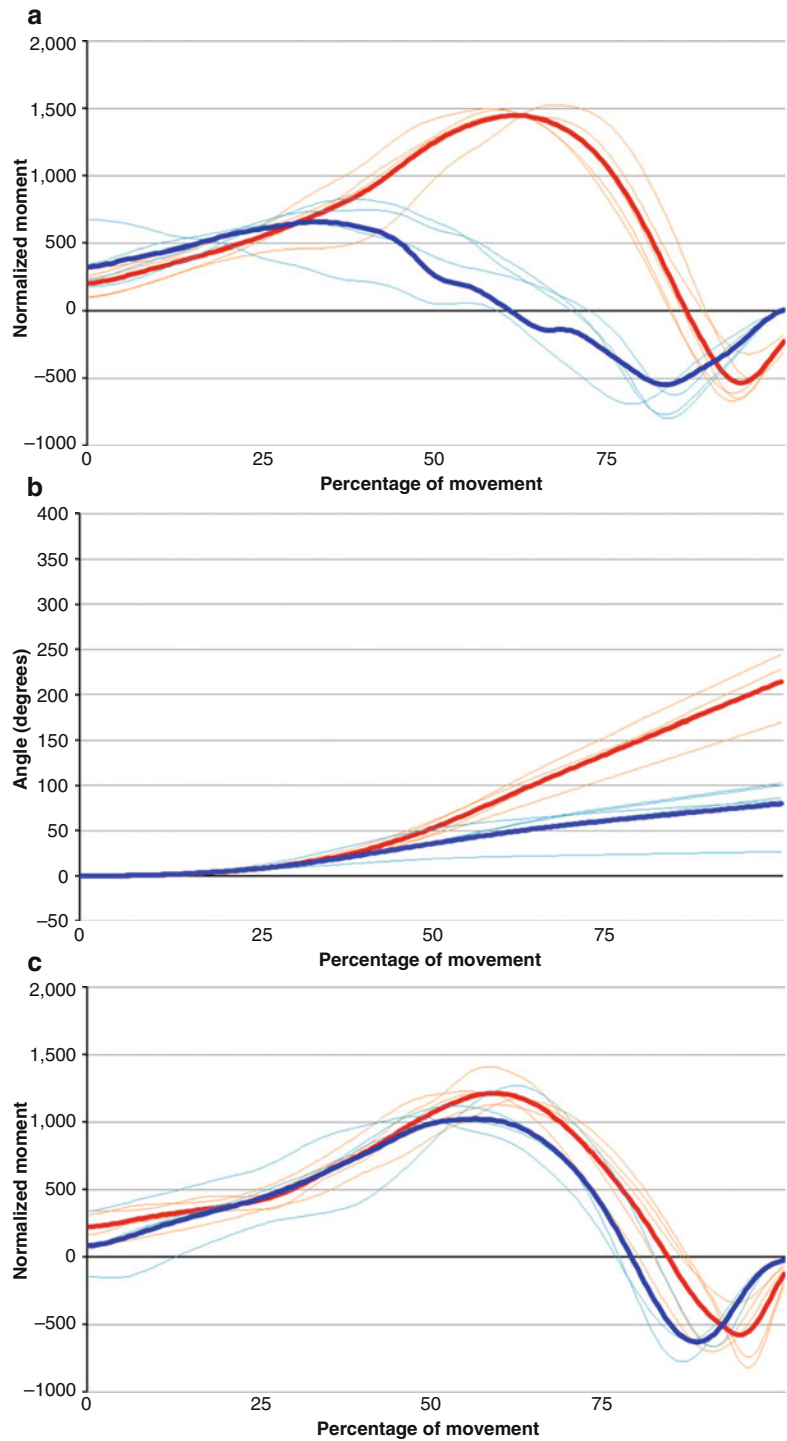


Fig. 20.7 Case # 4. (a, b) Right lateral patellar dislocation. (c) CT showing trochlear dysplasia type D. (d) Preoperative normalized moments registered during the monopodal jumping with pivoting with external tibial rotation test

Fig. 20.8 Case # 5. (a) Preoperative normalized moments registered during the monopodal jumping with pivoting with external tibial rotation test. (b) Preoperative body twist angle. (c) Postoperative normalized moments registered during the monopodal jumping with pivoting with external tibial rotation test. (d) Postoperative body twist angle. (e) Preoperative MRI. (f) Postoperative CT. TT-TG distance



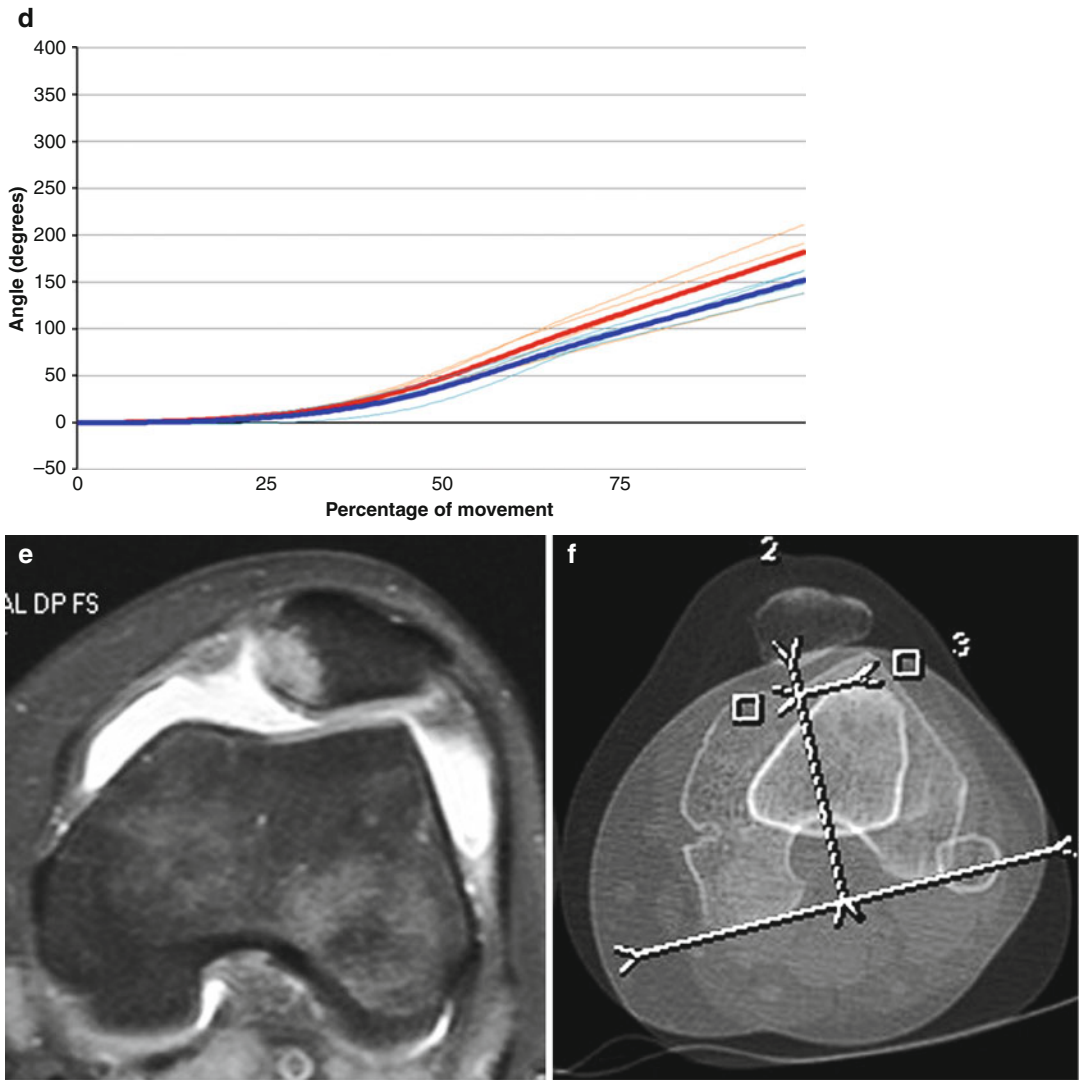


Fig. 20.8 (continued)

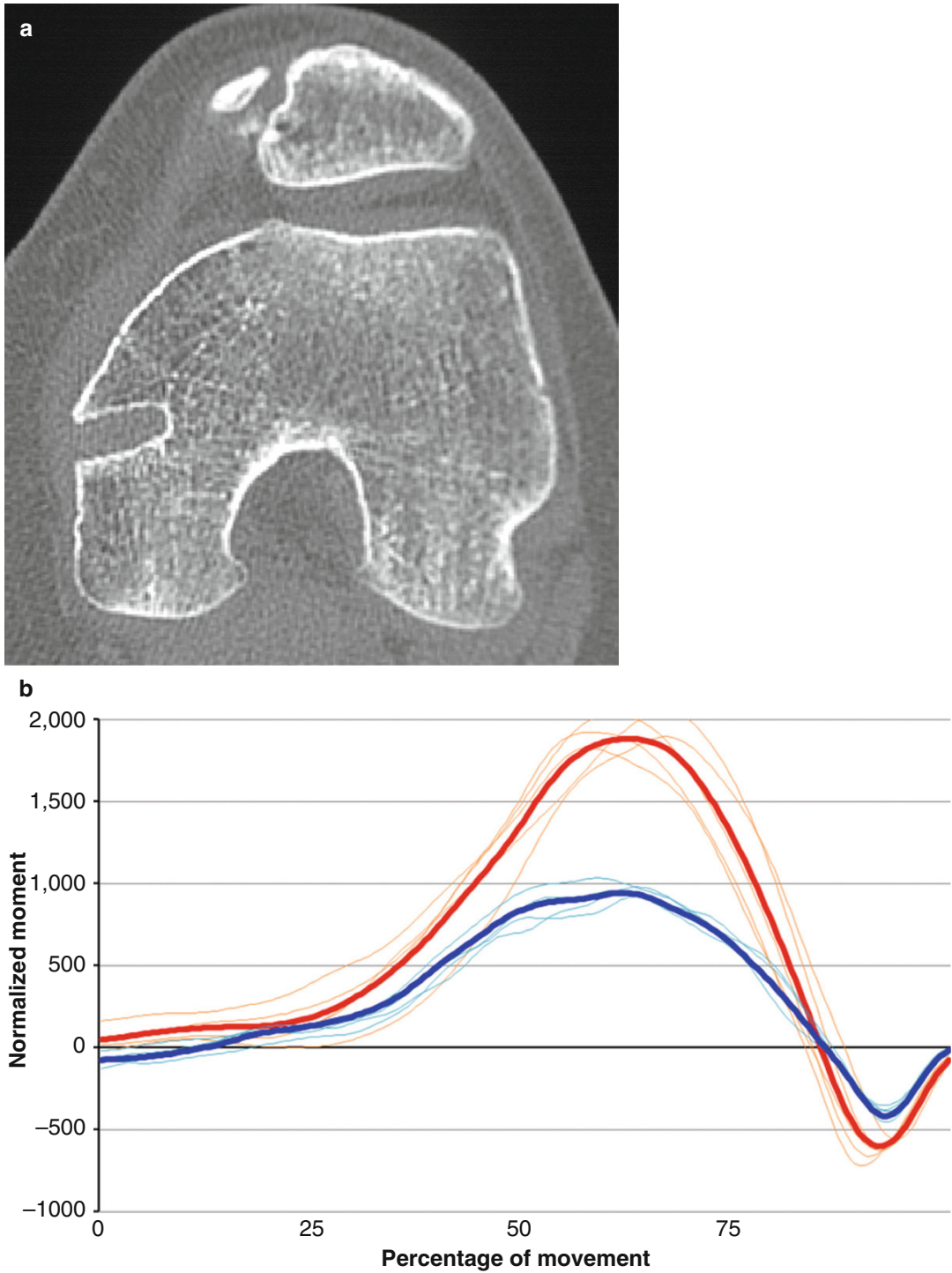


Fig. 20.9 Case # 6. (a) Postoperative CT showing a flat trochlea. (b) Postoperative normalized moments registered during the monopodal jumping with pivoting with external tibial rotation test

Kinetic and Kinematic Analysis in Evaluating Patients with Anterior Knee Pain

21

Vicente Sanchis-Alfonso, Susana Marín-Roca,
Erik Montesinos-Berry, José María Baydal-Bertomeu,
and María Francisca Peydro-De Moya

Most assessments of anterior knee pain treatment progression are made using subjective measurements. Kinetic and kinematic analyses would be appropriate to provide the physician with an objective dynamic measurement of treatment progression. However, we must insist that the kinetic and kinematic analysis of stair descent is not a diagnostic tool.

Our findings suggest that anterior knee pain patients use strategies to decrease PFJ loading

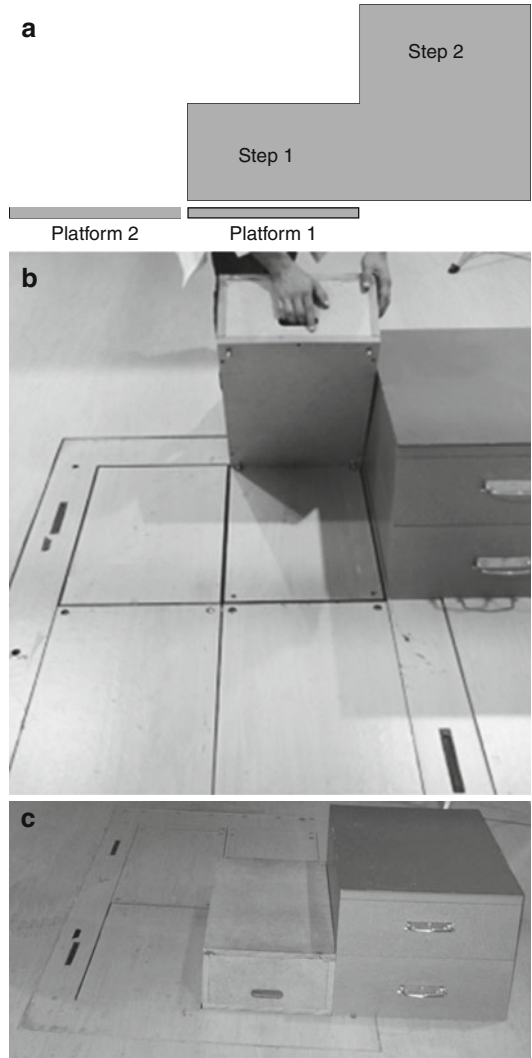
while going down stairs compared to a pain-free control group. The problem is that compensatory strategies require some time to develop and may remain even when pain disappears, which weakens the usefulness of this measurement technique as a treatment progression evaluation method. On the contrary, this technique helps us understand some of the mechanisms behind the development of knee osteoarthritis.

V. Sanchis-Alfonso, M.D., Ph.D. (✉)
International Patellofemoral Study Group,
ACL Study Group, Hospital 9 de Octubre,
Hospital Arnau de Vilanova, School of Medicine,
Valencia Catholic University,
Valencia, Spain
e-mail: vicente.sanchis.alfonso@gmail.com

S. Marín-Roca, Mech. Eng. • J.M. Baydal-Bertomeu,
Mech. Eng. • M.F. Peydro-De Moya, M.D., Ph.D.
Instituto de Biomecánica de Valencia (IBV),
Universidad Politécnica de Valencia,
Valencia, Spain

E. Montesinos-Berry, M.D.
Hospital de Manises,
Valencia, Spain

Fig. 21.1 (a) Dynamometric platform disposition. (b) Step adaptation to the dynamometric platform. (c) Step disposition



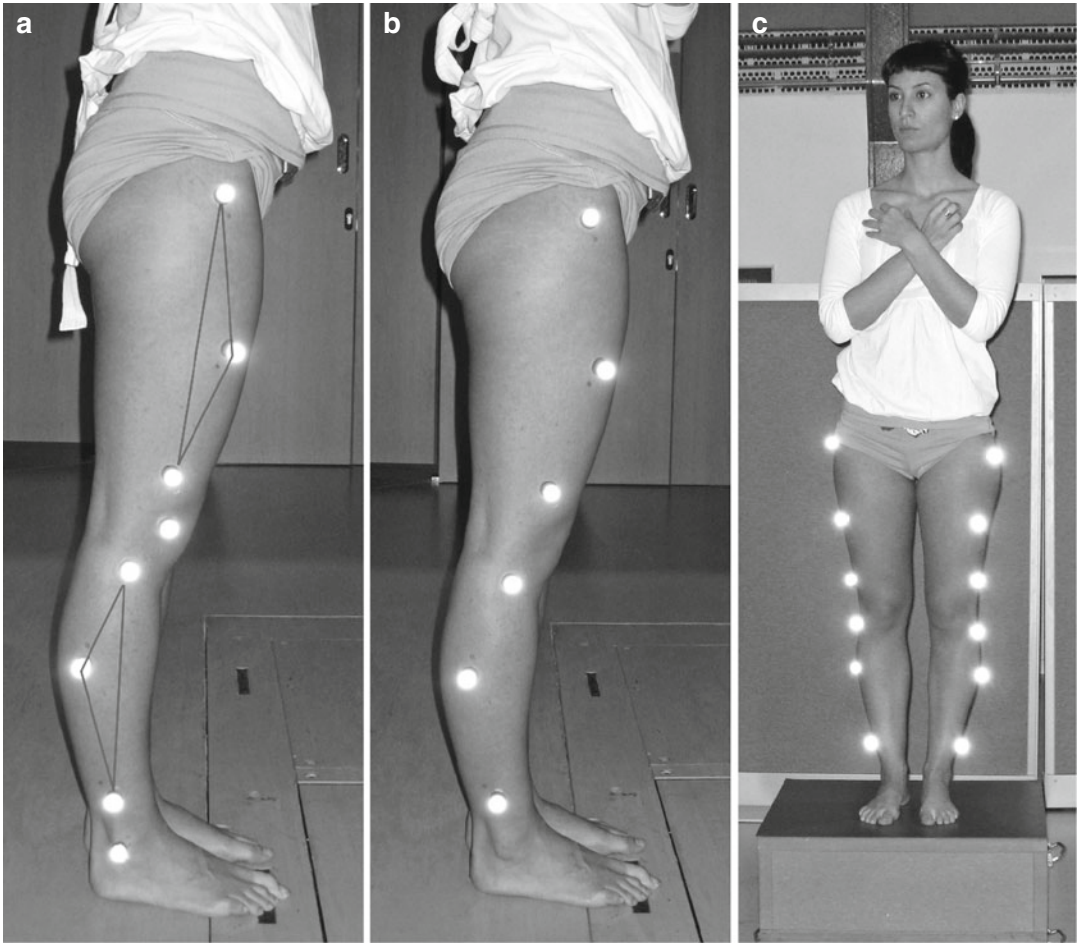
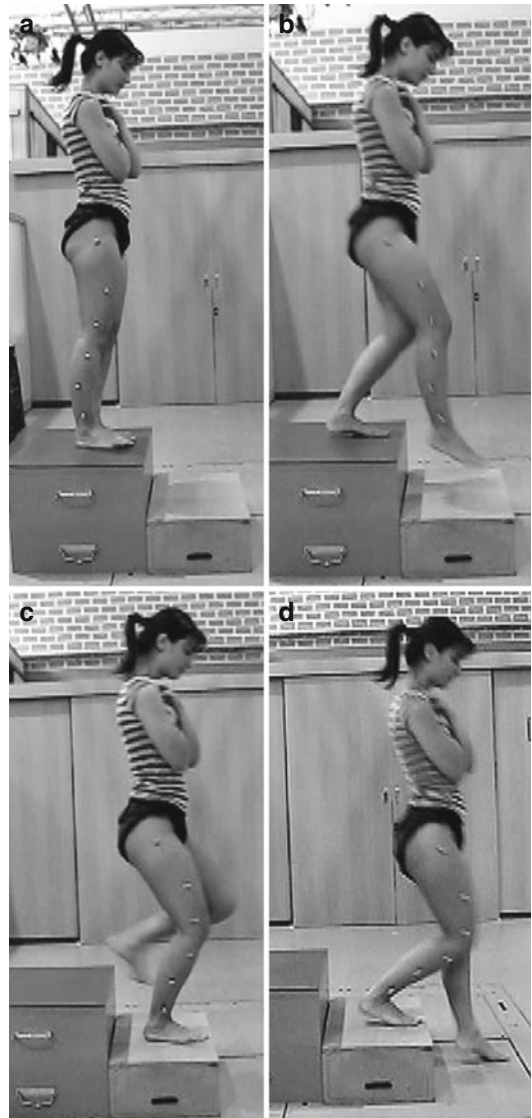


Fig. 21.2 Marker disposition. (a) With calibration markers. (b, c) Without calibration markers

Fig. 21.3 Photographic sequence of the stair descent test



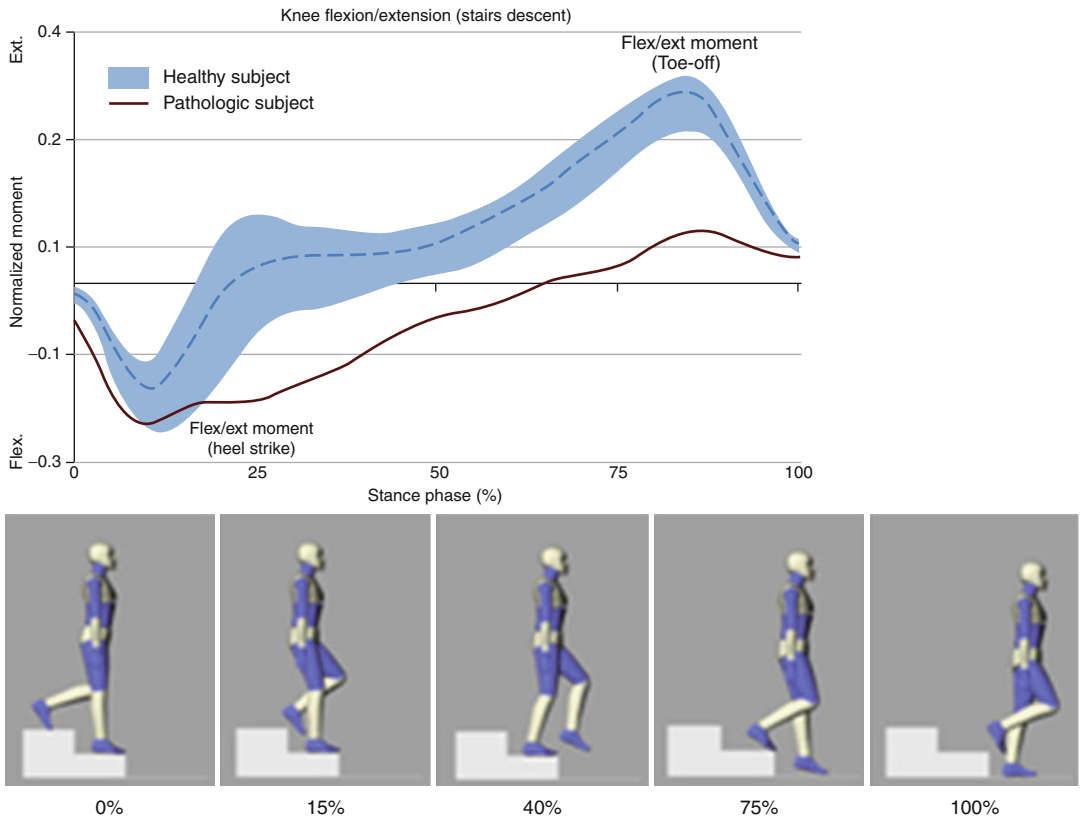


Fig. 21.4 Knee extension moment

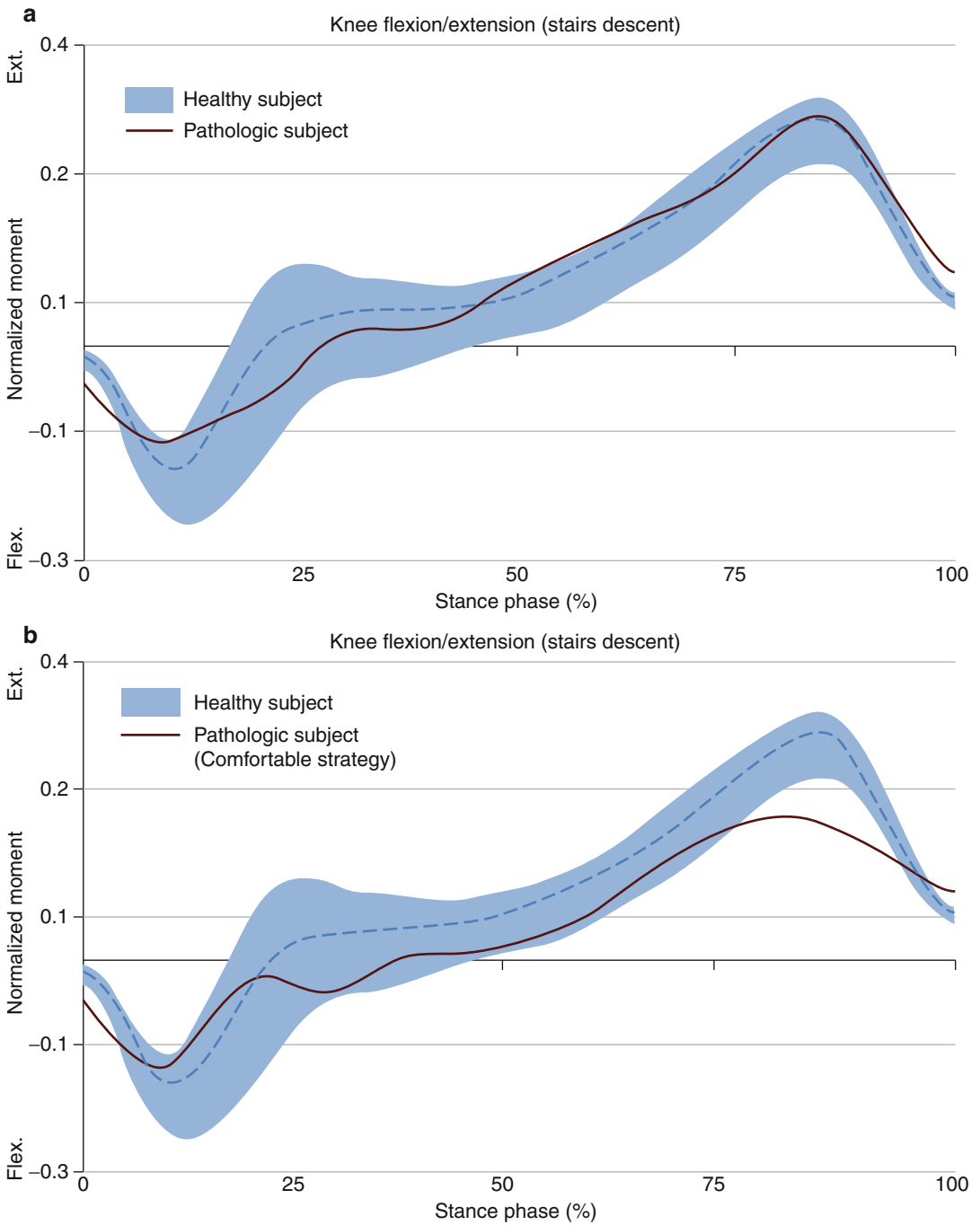


Fig. 21.5 Knee extension moment. (a) Standard stair descending test. (b) Stair descending test following the comfort strategy

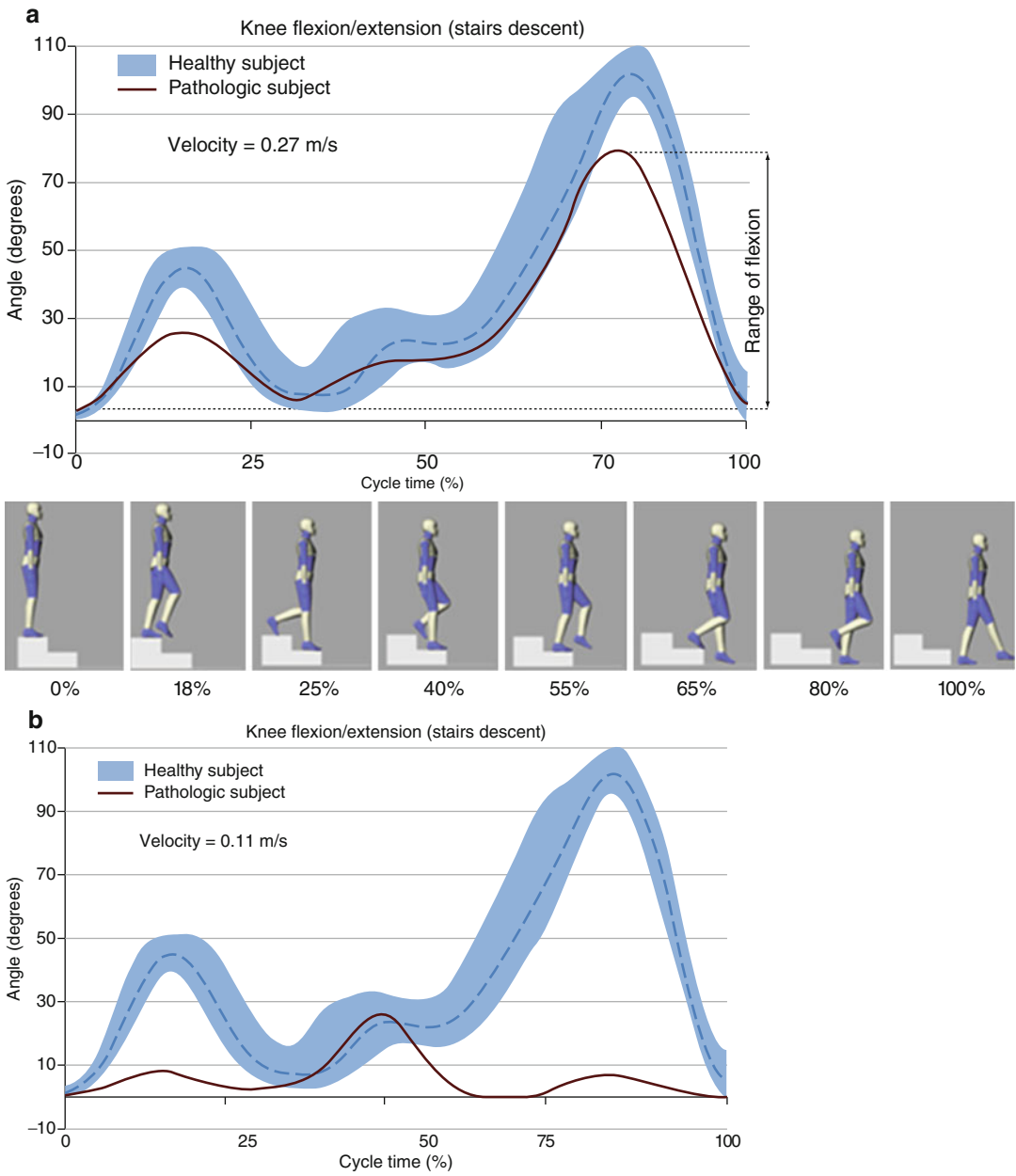


Fig. 21.6 Knee flexion during stair descending test (a). (b) Stair descending with a severe knee extension pattern

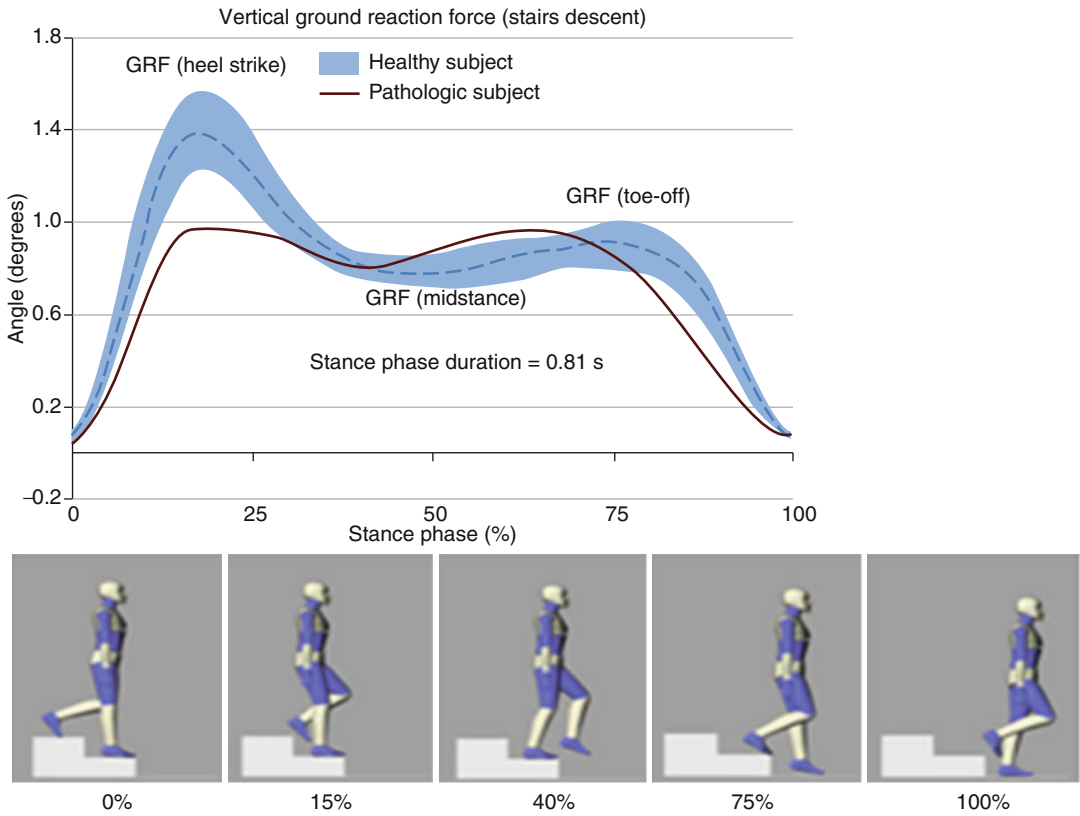


Fig. 21.7 Vertical ground reaction force during stair descending test

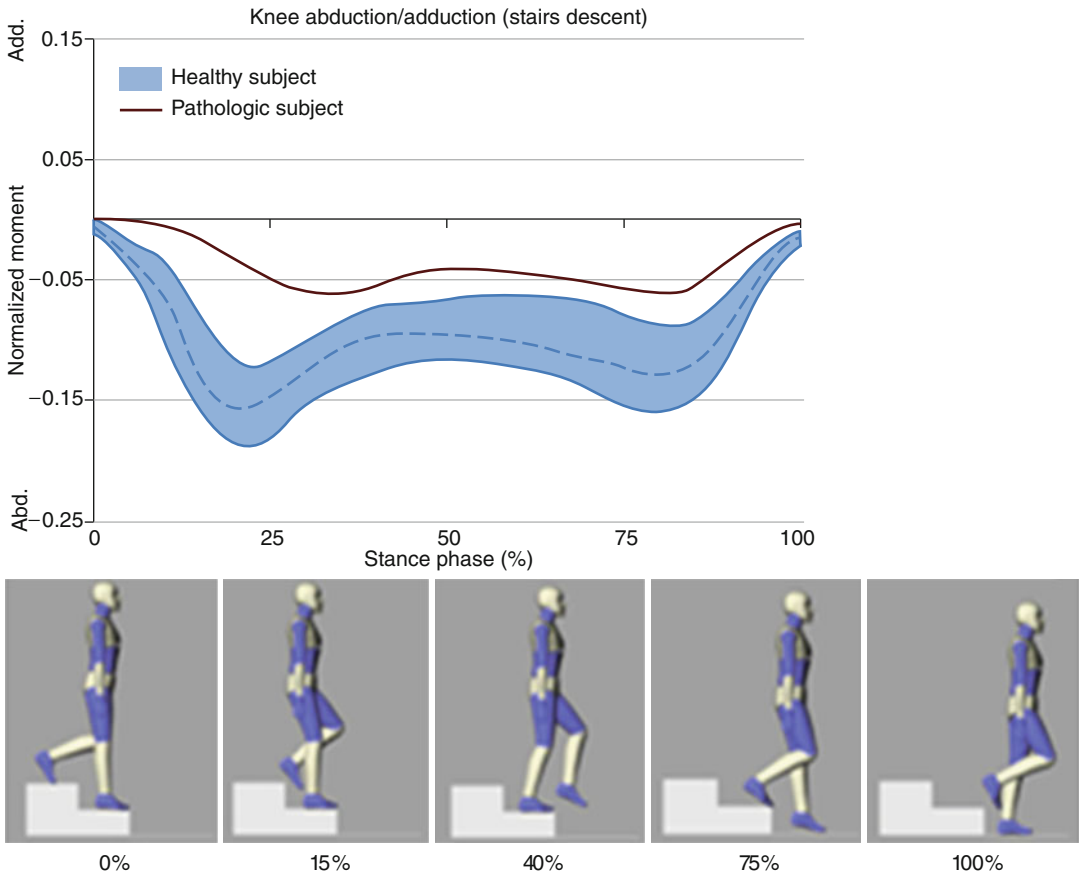


Fig. 21.8 Knee abduction moment during stair descending test

Part III

Clinical Cases Commented

Failure of Patellofemoral Surgery: Analysis of Clinical Cases

22

Robert A. Teitge and Roger Torga-Spak

Malfunction of the patellofemoral joint is the result from a failure of any one or a combination of three factors: Alignment, Stability, Articular Cartilage.

An analysis of patellofemoral dysfunction best proceeds with an independent analysis of each of these elements. A more clear understanding of the clinical syndrome can be made if one first looks at each factor independently and then attempts to relate the factors sequentially and causally. As yet there exists no formula, which can quantitatively determine the relative contributions of each of

these components in such a way as to define the mechanics, the pathology, and the clinical picture. These three factors are not the same but are often related. It is important to think of the disease process as resulting from a combination of contributions of abnormality from each of these three components. There may be a failure of any one area individually, simultaneously or sequentially. If the pathomechanics can be determined, then a revision surgery that first reverses the previous surgeries and then corrects the primary mechanical etiology has the best chances of success.

R.A. Teitge, M.D.
Department of Orthopaedics,
Wayne State University School of Medicine,
Detroit, MI, USA

R. Torga-Spak, M.D. (✉)
Department of Surgery, Faculty of Orthopaedics and
Traumatology, Instituto Universitario CEMIC,
Buenos Aires, Argentina
e-mail: rtorgaspak@gmail.com

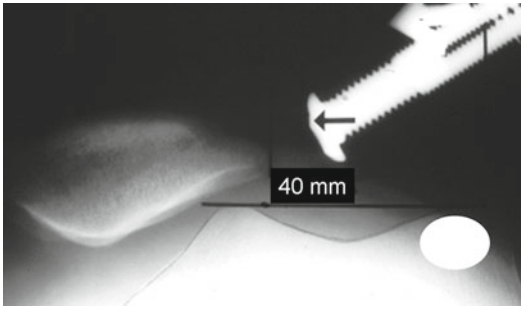


Fig. 22.1 Medial dislocation of the patella after arthroscopic lateral patellar release

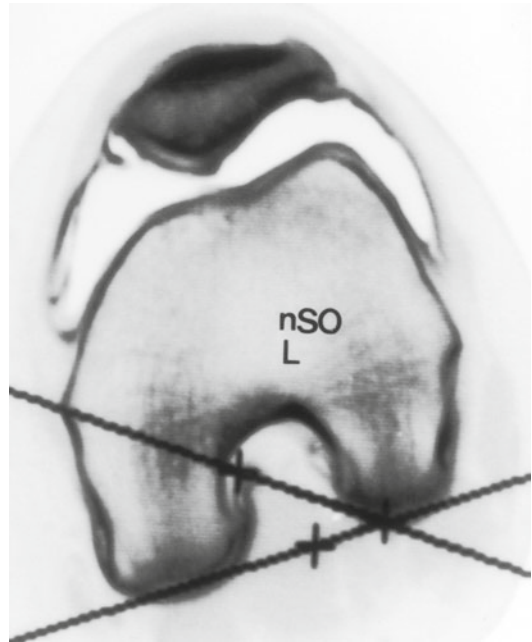


Fig. 22.3 CT arthrogram. Loss lateral articular cartilage, shallow trochlea, post lateral release and arthroscopic chondroplasty



Fig. 22.2 Example of medial dislocatable patella post realignment

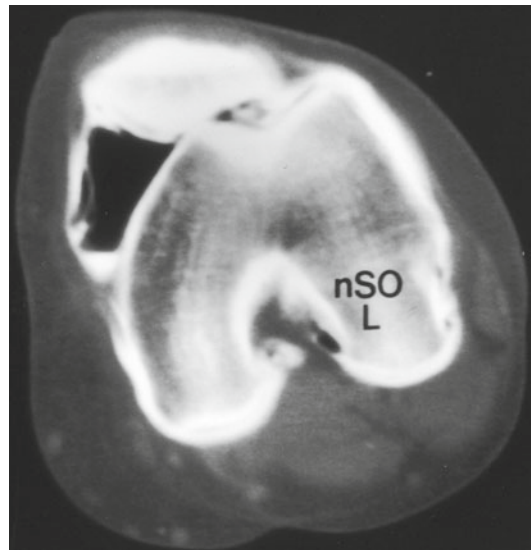


Fig. 22.4 Medial dislocation patella post lateral release showing why medial dislocation causes lateral facet damage

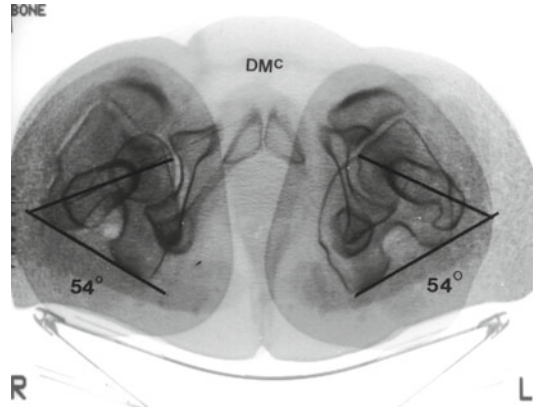


Fig. 22.6 Preop CT rotational scan shows bilateral 54° femoral anteversion

Fig. 22.5 A 34-year-old-patient post right Maquet osteotomy with inpointing left patella. She is post-op right intertrochanteric 40° external rotation osteotomy

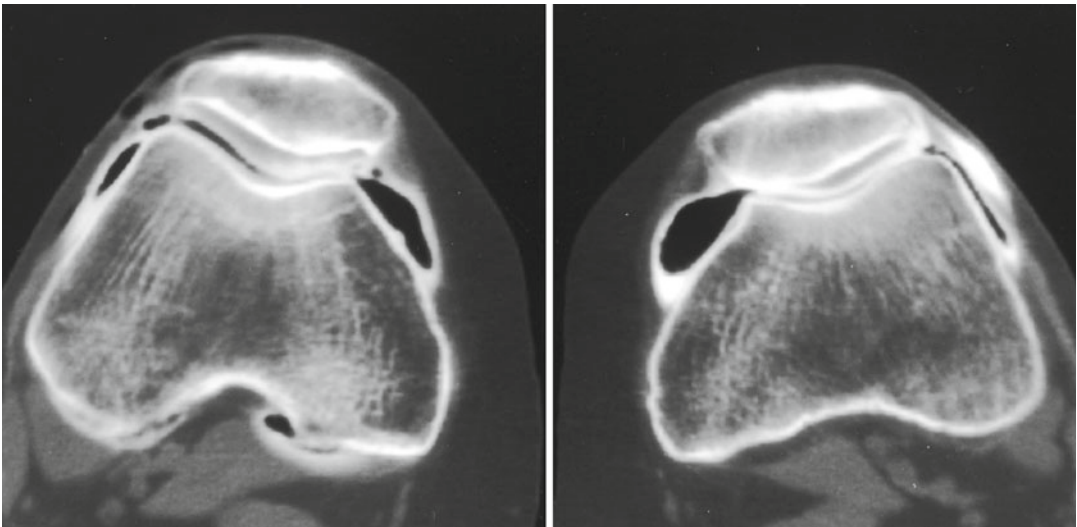


Fig. 22.7 Double contrast CT arthrogram showing loss of medial patellofemoral cartilage bilaterally after medial tibial tubercle transfer

Fig. 22.8 CT rotational study showing bilateral femoral anteversion. Treatment was lateral tibial tubercle transfer to unload medial facet and intertrochanteric external rotational osteotomy to treat rotational alignment

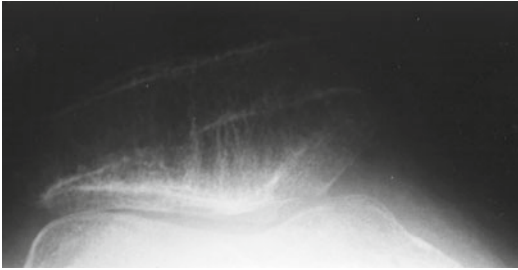
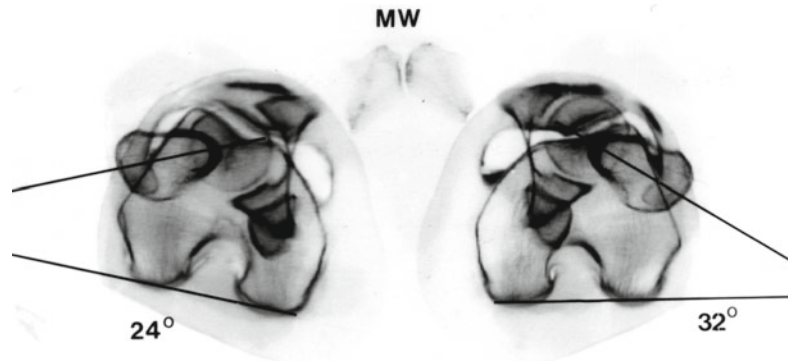


Fig. 22.9 Post lateral PF ligament reconstruction development arthrosis PF joint



Fig. 22.10 CT scan showing arthrosis

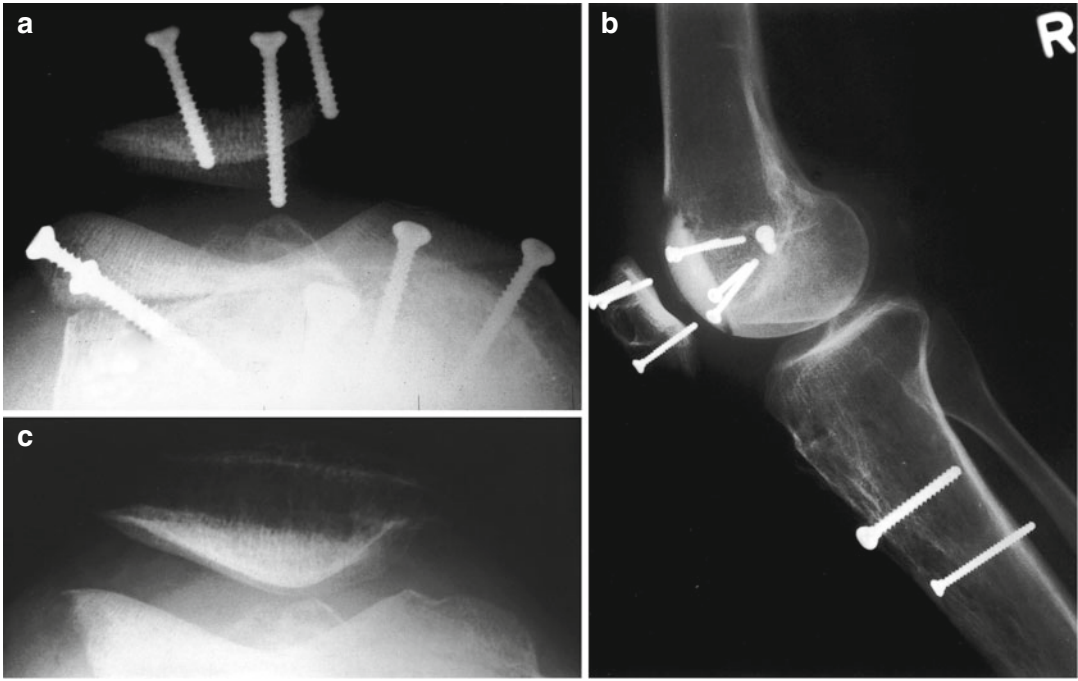


Fig. 22.11 (a) Axial x-ray 2 weeks post PF fresh allograft. (b) Lateral x-ray 2 weeks post PF fresh allograft. (c) Axial x-ray 10 months post PF fresh allograft. (d) Lateral x-ray 10 months post PF fresh allograft. (e) Axial x-ray 5 years post PF fresh allograft. (f) CT 9 years post PF fresh allograft. Compare with Fig. 22.10

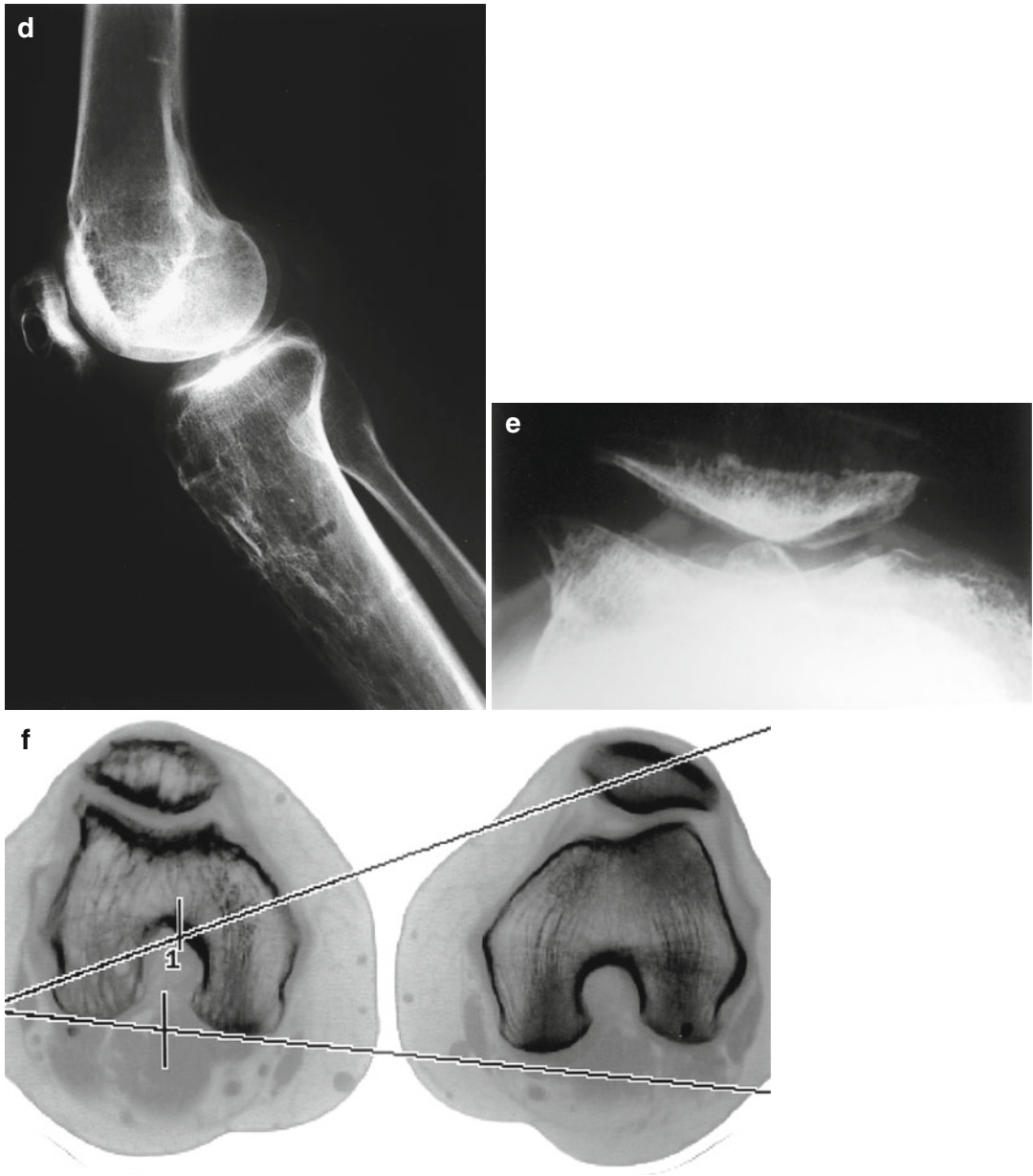


Fig. 22.11 (continued)

Neuromatous Knee Pain: Evaluation and Management

23

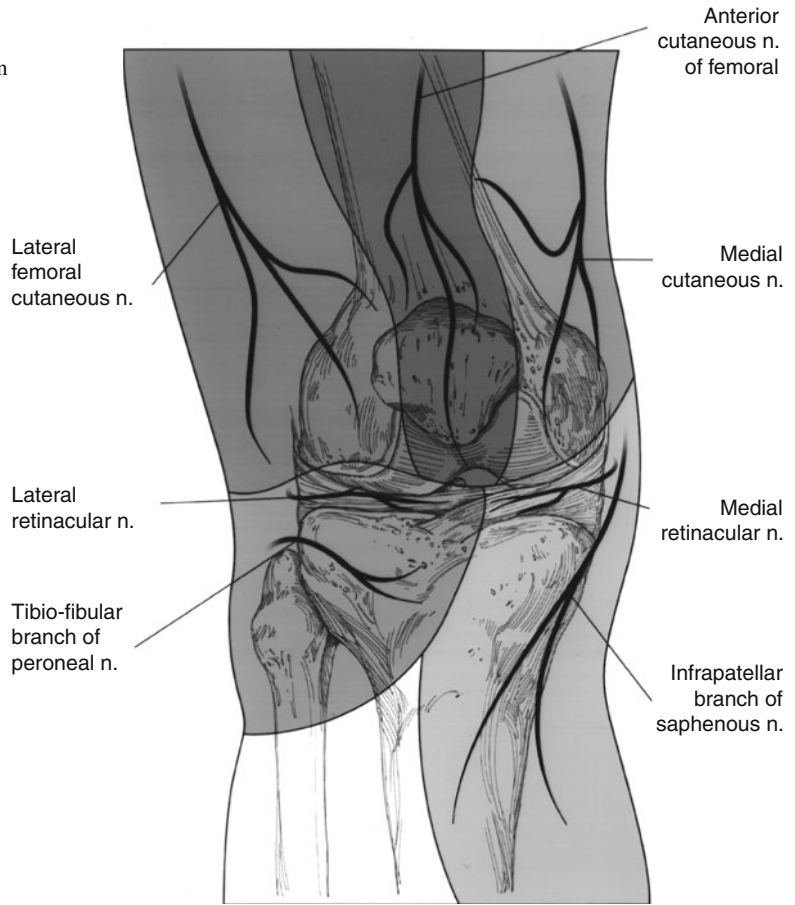
Maurice Y. Nahabedian

Selective denervation for neuromatous pain about the knee joint can be a beneficial procedure. Proper patient selection is a critical component that impacts the success of the operation. The salient components include pain of at least 1-year duration unrelied by conservative measures, the presence of a

Tinels sign in the painful territory, and at least a 5-point reduction in the visual analog score following nerve blockade with 1 % lidocaine. This procedure is not recommended for pain on nonneuromatous origin, pain that is less than 1-year duration, and for diffuse knee pain without a Tinels sign.

M.Y. Nahabedian, M.D., FACS
Department of Plastic Surgery, Georgetown University,
Washington, DC, USA
e-mail: dmahabedian@aol.com

Fig. 23.1 An illustration demonstrating the course and cutaneous territories of the seven surgically identifiable nerves about the knee



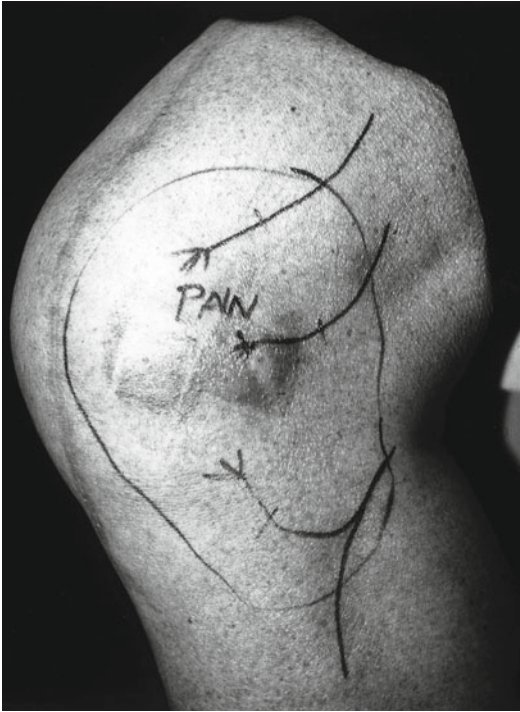


Fig. 23.2 A photograph demonstrating the delineated territory of pain secondary to a neuroma of the infrapatellar branch of the saphenous nerve



Fig. 23.4 Radiograph demonstrating proximal tibial screws that resulted in a neuroma



Fig. 23.3 A photograph demonstrating the Tinels point and the paths of radiation



Fig. 23.5 The incision site for exposure of the common peroneal nerve is delineated

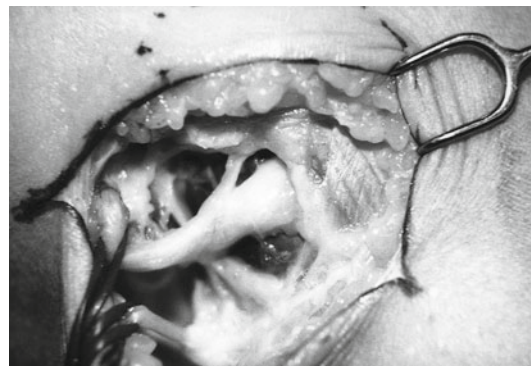


Fig. 23.6 The common peroneal nerve is isolated and the tibiofibular branch is isolated as it traverses under the peroneus longus muscle

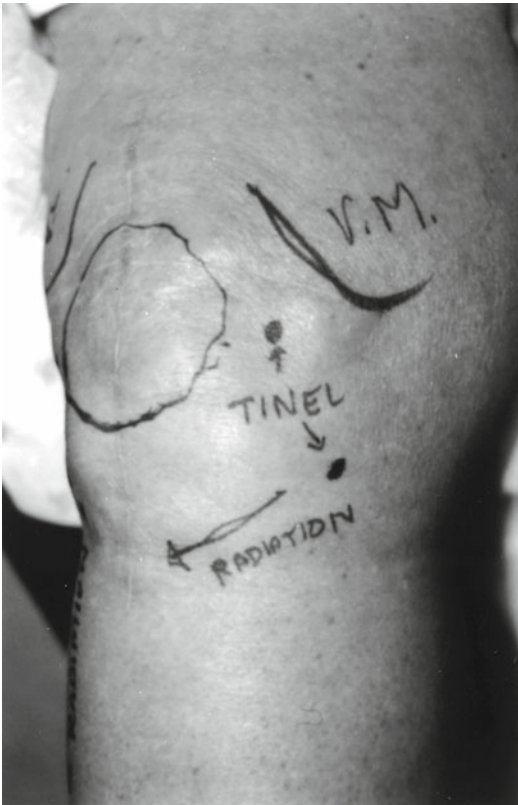


Fig. 23.7 The medial aspect of the knee is depicted demonstrating the patella and vastus medialis muscle (VM). The Tinel points and path of radiation are outlined



Fig. 23.8 The lateral aspect of the knee is depicted demonstrating the fibular head and the vastus lateralis muscle (VL). The Tinel points are marked

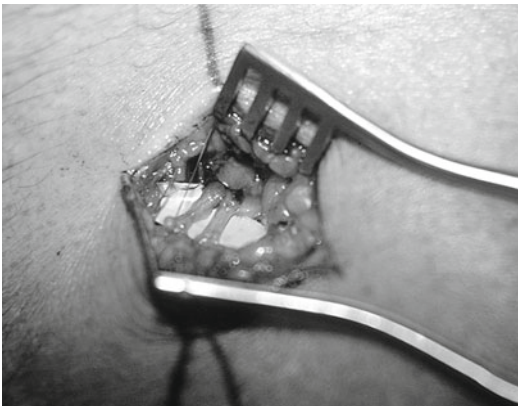


Fig. 23.9 Two branches of the infrapatellar branch of the saphenous nerve are illustrated. These branches were excised and the proximal stumps buried in adjacent muscle

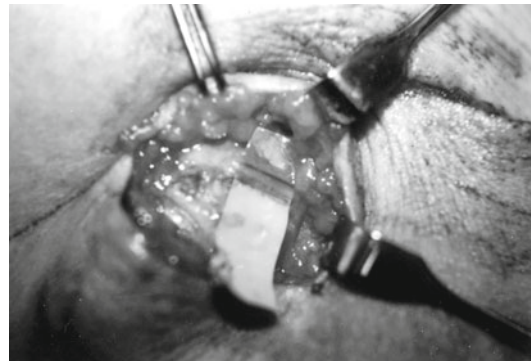


Fig. 23.10 The medial cutaneous nerve of the knee is demonstrated

Kinetic and Kinematic Analysis of Iatrogenic Medial Patellar Instability: Clinical Relevance

24

Vicente Sanchis-Alfonso, Erik Montesinos-Berry,
Andrea Castelli, Susana Marín-Roca, and Alex Cortes

We speculate that chronic recurrent soft tissue fatigue or overload due to patellofemoral imbalance could cause anterior knee pain. However, many chondropathies can be asymptomatic due to the biomechanic defence strategies used by the patients. Moreover, we emphasize the importance of the passive restraining structures in patellar stability, in contrast to the role of muscle function advocated by some authors. This could explain why an exercise rehabilitation program could be unsuccessful in improving patellar instability, as

it occurred in our cases. Finally, these cases highlight the need for surgeons to be more judicious in their surgical selection for patellofemoral instability and serve as a warning for the generalized use of lateral retinacular release. The question we ask ourselves is: Is there a place for the isolated lateral retinacular release? In this sense, it would be interesting to consider the lengthening of the lateral retinaculum described by Roland Biedert as an alternative to lateral retinacular release.

V. Sanchis-Alfonso, M.D., Ph.D. (✉)
International Patellofemoral Study Group,
ACL Study Group, Hospital 9 de Octubre, Hospital
Arnao de Vilanova, School of Medicine,
Valencia Catholic University, Valencia, Spain
e-mail: vicente.sanchis.alfonso@gmail.com

E. Montesinos-Berry, M.D.
Department of Orthopaedic Surgery,
Hospital de Manises, Valencia, Spain

A. Castelli, Biomed. Eng. • S. Marín-Roca, Mech. Eng.
A. Cortes, M.D., Ph.D.
Instituto de Biomecánica de Valencia (IBV),
Universidad Politécnica de Valencia,
Valencia, Spain

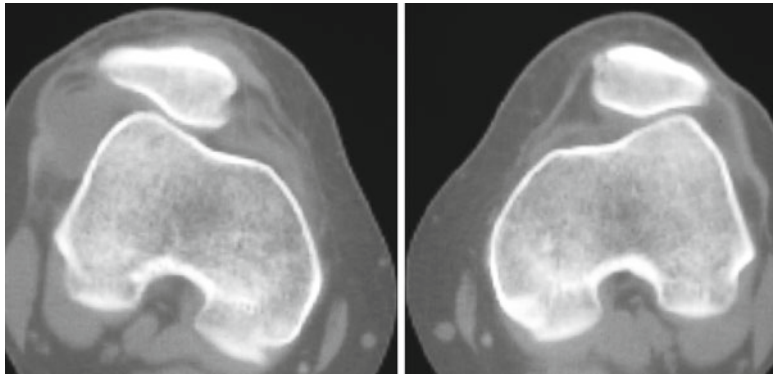


Fig. 24.1 Computed tomography (CT) examination in 0° extension and quadriceps contraction shows lateralization of the patella (Reprinted from Sanchis-Alfonso V, Torga-Spak R, Cortes A. Gait pattern normalization after lateral

retinaculum reconstruction for iatrogenic medial patellar instability. *Knee*. 2007;14:484–8. With permission from Elsevier)

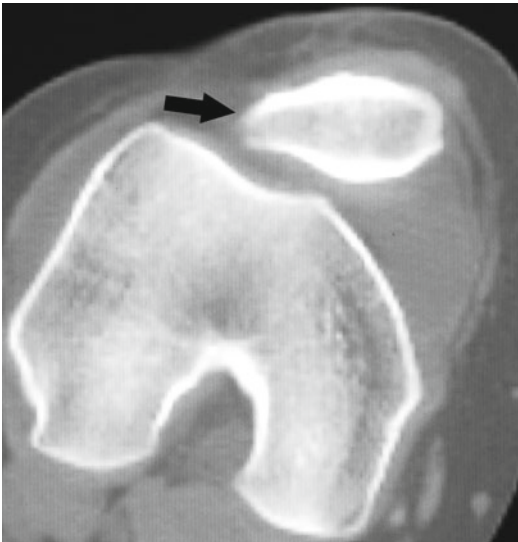
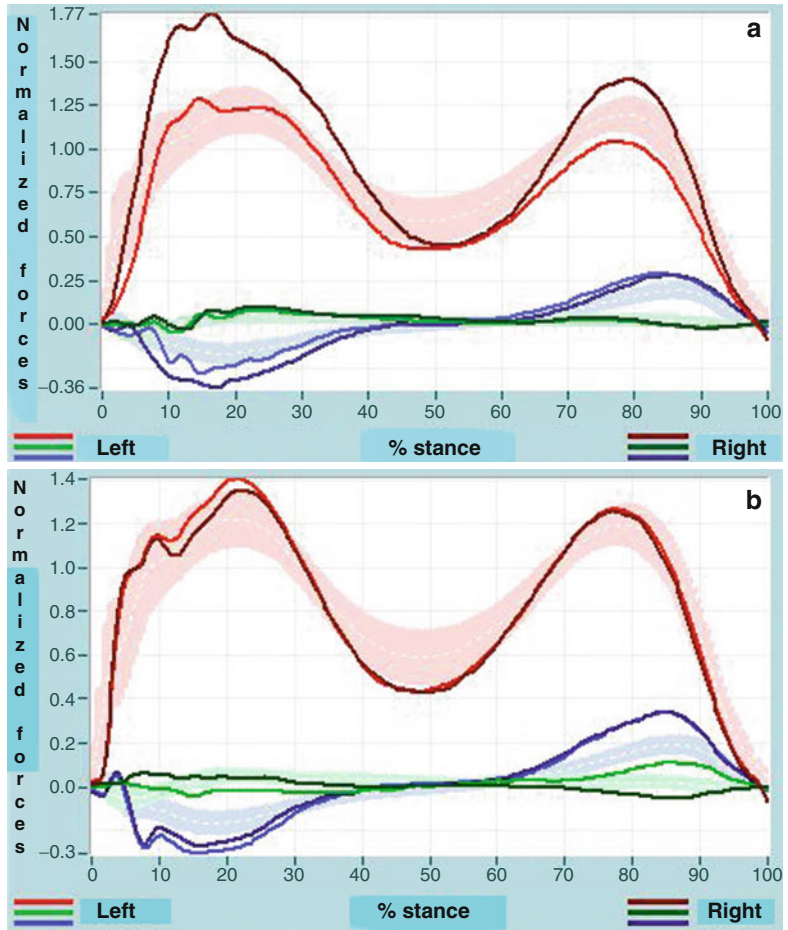


Fig. 24.2 Documenting of medial patellar instability using manual pressure (finger on the lateral aspect of the patella *black arrow*) (Reprinted from Sanchis-Alfonso V, Torga-Spak R, Cortes A. Gait pattern normalization after lateral retinaculum reconstruction for iatrogenic medial patellar instability. *Knee*. 2007;14:484–8. With permission from Elsevier)

Fig. 24.3 Gait analysis.
 (a) Preoperative.
 (b) Postoperative (Reprinted from Sanchis-Alfonso V, Torga-Spak R, Cortes A. Gait pattern normalization after lateral retinaculum reconstruction for iatrogenic medial patellar instability. *Knee*. 2007;14:484–8. With permission from Elsevier)



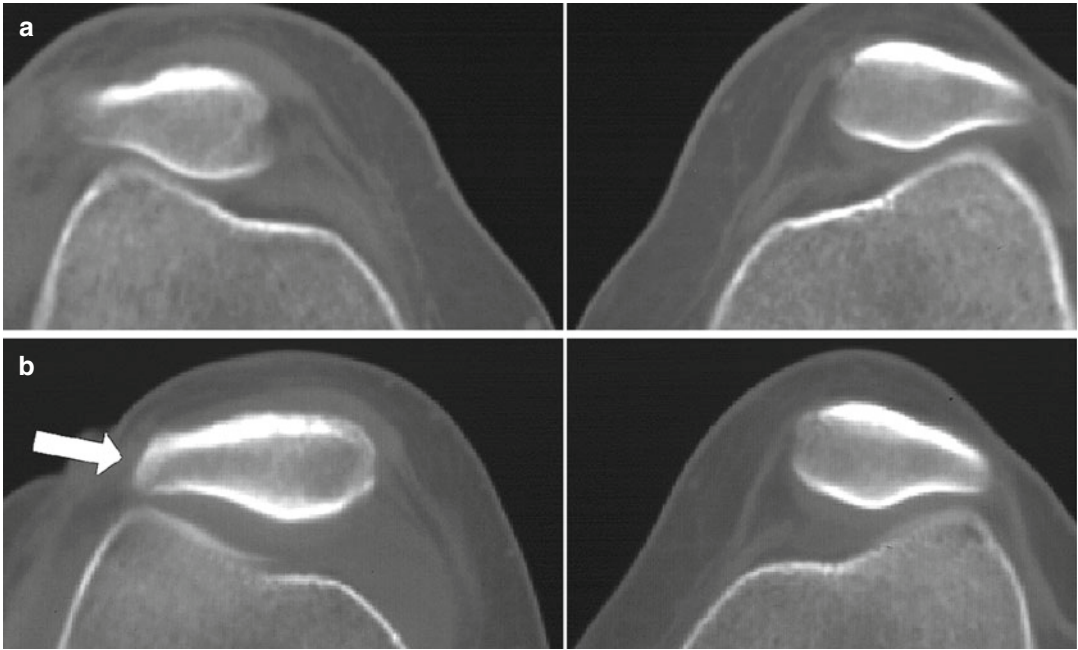


Fig. 24.4 Follow-up CT scan at 0° extension with quadriceps contraction demonstrates similar lateral displacement of the patella in both knees (a), and stress CT revealed medial patellar stability (finger on the lateral aspect of the patella *white arrow*) (b) (Reprinted from

Sanchis-Alfonso V, Torga-Spak R, Cortes A. Gait pattern normalization after lateral retinaculum reconstruction for iatrogenic medial patellar instability. *Knee*. 2007;14:484–8. With permission from Elsevier)



Fig. 24.5 Conventional radiographs showing isolated patellofemoral osteoarthritis. (a) anteroposterior view, (b) lateral view (c) axial view

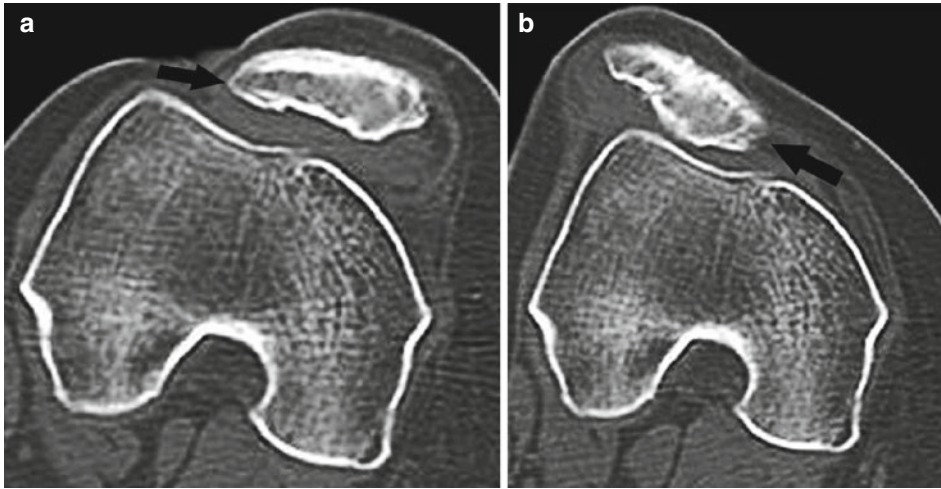


Fig. 24.6 (a) Medial stress CT at 0° extension (finger on the lateral aspect of the patella *black arrow*). (b) Lateral stress CT at 0° extension (finger on the medial aspect of the patella *black arrow*). Note the presence of a shallow trochlea

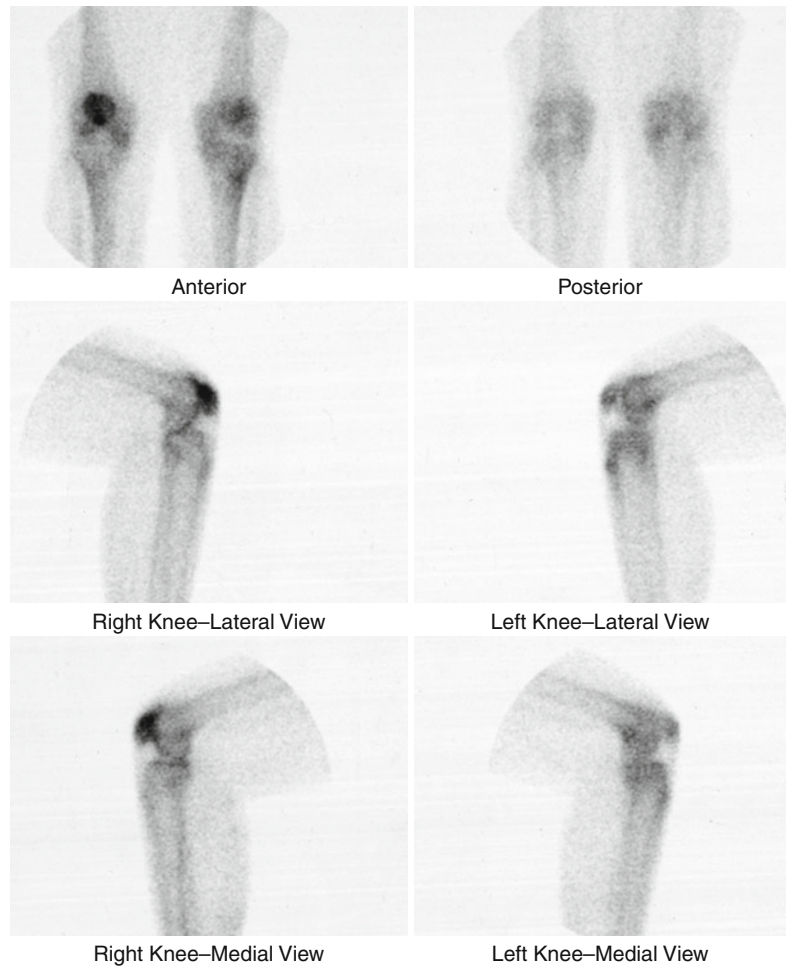


Fig. 24.7 Standard technetium 99 methylene diphosphonate bone scan showing increased osseous metabolic activity of the patella

Fig. 24.8 Knee kinetics and kinematics during stair descent in case #2. **(a)** Knee joint angle during stair descent. **(b)** Ground force reactions during stair descent. **(c)** Flexion–extension knee moments during stair descent. **(d)** Abduction–Adduction knee moments during stair descent

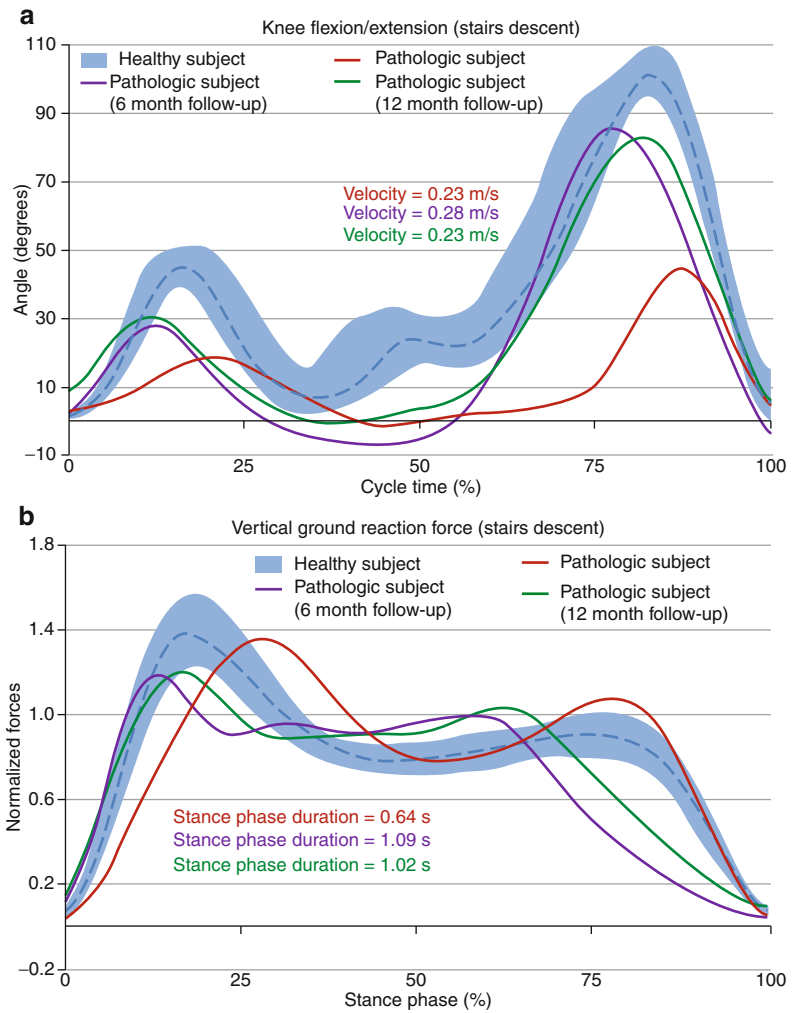
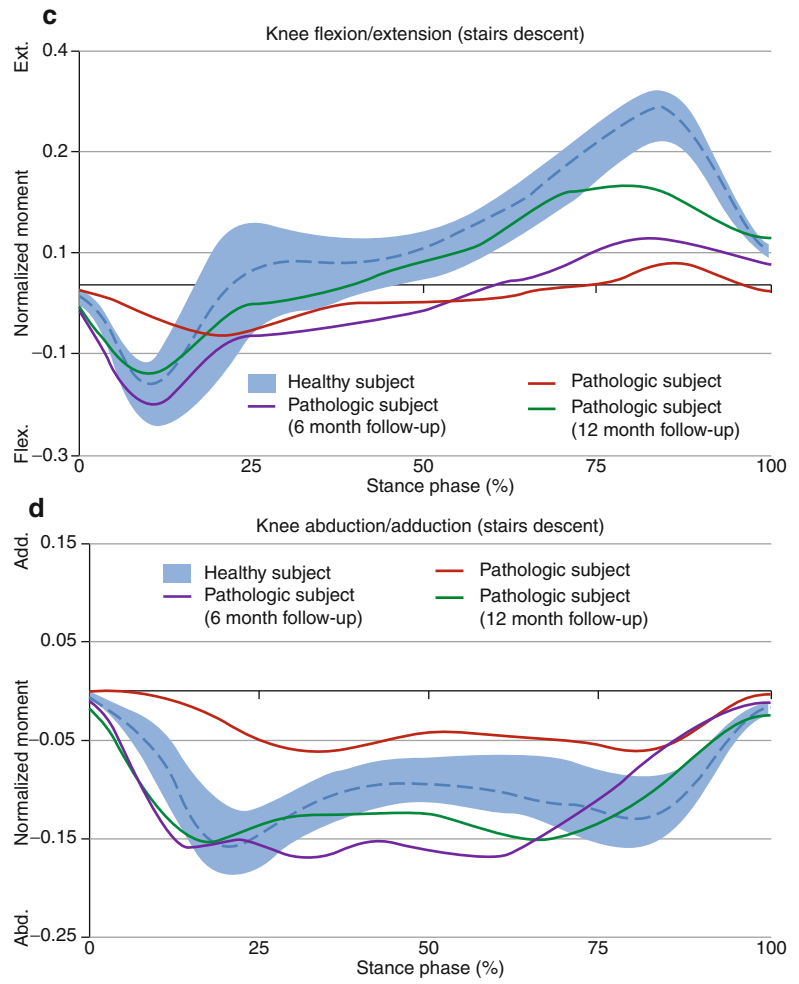


Fig. 24.8 (continued)



Part IV

Surgical Techniques “How I Do It”

Ultrasound and Doppler-Guided Arthroscopic Shaving for the Treatment of Patellar Tendinopathy/Jumper's Knee: Biological Background and Description of Method

Håkan Alfredson, Lotta Willberg, Lars Öhberg,
and Sture Forsgren

Treatment with ultrasound and Doppler-guided arthroscopic shaving of the region with vessels and nerves outside the dorsal tendon has shown promising clinical results in patients with proximal patellar tendinopathy/Jumper's knee. The results concerning only a limited patient material has been published in a scientific

paper. Results on larger materials are under evaluation for later publication. Proper understanding of the ultrasound and Doppler findings, to enable for a precise and minimal arthroscopic shaving procedure on the dorsal side of the tendon, are cornerstones using this new type of treatment.

H. Alfredson, M.D., Ph.D. (✉)
Sports Medicine Unit, Department of Surgical and
Perioperative Sciences, Umeå University,
Umeå, Sweden
e-mail: hakan.alfredson@idrott.umu.se

L. Willberg, M.D.
Capio Artro Clinic, Karolinska Institute,
Stockholm, Sweden

L. Öhberg, M.D., Ph.D.
Department of Radiation Sciences,
Diagnostic Radiology,
Umeå, Sweden

S. Forsgren, M.D., Ph.D.
Department of Integrative Medical Biology, Anatomy,
Umeå University,
Umeå, Sweden

Fig. 25.1 Gray-scale ultrasound and color Doppler examination of a patient with patellar tendinopathy/Jumper's knee, showing thickening of the proximal tendon, irregular tendon structure and hypoechoicity, and high blood flow (*color*) inside the tendon and outside the dorsal part of the tendon

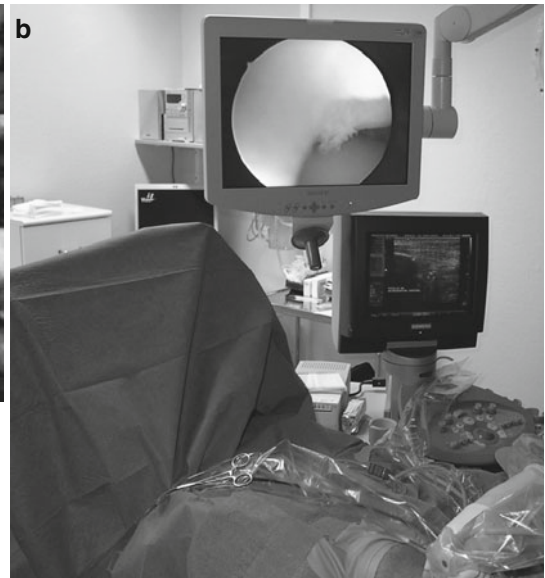
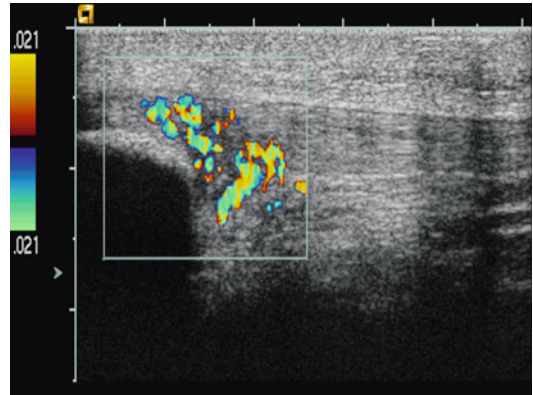


Fig. 25.2 For treatment of the patellar tendinopathy/Jumper's knee changes, ultrasound and arthroscopy monitors are used simultaneously. An assistant is managing the

ultrasound probe (draped sterile), while the surgeon is using the ultrasound and Doppler findings as a guide for the arthroscopic procedure

Jordi Vega, Pau Golanó, and Vicente Sanchis-Alfonso

Arthroscopic patellar denervation has shown promising clinical results in the treatment of young patients with AKP, with or without minimal malalignment recalcitrant to conservative treatment. The substantial benefits, the considerable comfort for the patient, minimal risk and low rates of morbidity, made this technique a comfortable procedure for the patient. Therefore, arthroscopic patellar denervation might be a reasonable first surgical step in patients with AKP resistant to conservative therapy. Moreover, arthroscopic patellar denervation might become a beneficial addition to other surgical methods for treating AKP.

Moreover, the knee joint can be explored during arthroscopic procedure in order to evaluate the presence of possible causes of AKP not observed in previous complementary studies (plicae formations and cartilage lesions in the femoropatellar joint). A further advantage of the arthroscopic patellar denervation is the fact that it does not interfere with the knee kinetics and allows future surgical techniques to be performed if necessary. Finally, arthroscopic patellar denervation is a simple procedure that could be performed by surgeons with little experience in knee arthroscopy. However, before recommending this treatment method for general use, the results from randomized studies with more patients are needed.

J. Vega (✉)
Department of Orthopedic and Trauma Surgery,
Hospital Asepeyo Sant Cugat,
Sant Cugat del Vallés, Barcelona, Spain
e-mail: jordivega@hotmail.com

P. Golanó
Laboratory of Arthroscopic and Surgical Anatomy,
Department of Pathology and Experimental
Therapeutics, Human Anatomy Unit,
Faculty of Medicine, University of Barcelona,
Barcelona, Spain

V. Sanchis-Alfonso, M.D., Ph.D.
International Patellofemoral Study Group,
ACL Study Group, Hospital 9 de Octubre, Hospital
Arnau de Vilanova, School of Medicine,
Valencia Catholic University,
Valencia, Spain

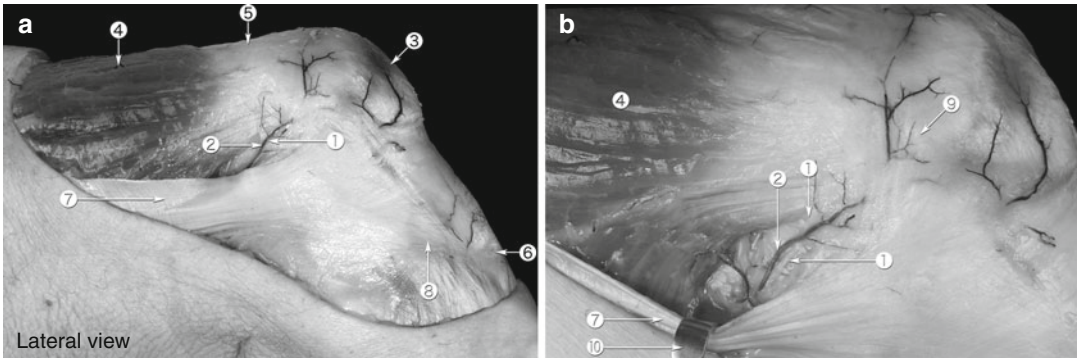


Fig. 26.1 (a) Anatomic dissection of the lateral side of the right knee. (b) Macrophotography of the lateral patellar innervation. (1) Lateral retinacular nerve. (2) Superior lateral genicular artery. (3) Patella. (4) Vastus lateralis. (5) Quadriceps femoralis tendon. (6) Patellar tendon insertion (tibial tuberosity). (7) Iliotibial tract (8) Gerdy's tubercle. (9) Superior lateral angle of the patella. (10) Sean-Miller retracting the iliotibial tract

Fig. 26.2 Anatomic dissection of the medial side of the right knee. Macrophotography. (1) Medial retinacular nerve and branches. (2) Superior medial genicular artery. (3) Patella (superior medial angle of the patella). (4) Vastus medialis. (5) Quadriceps femoralis tendon. (6) Medial patelofemoral ligament. (7) Fascia (rejected). (8) Medial epicondyle

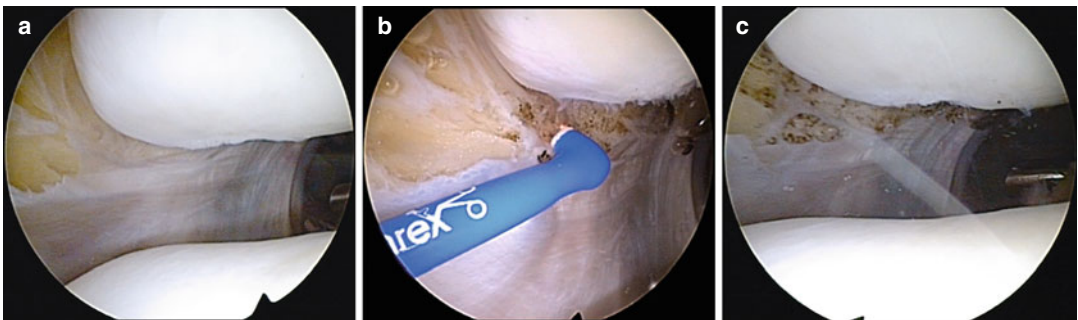
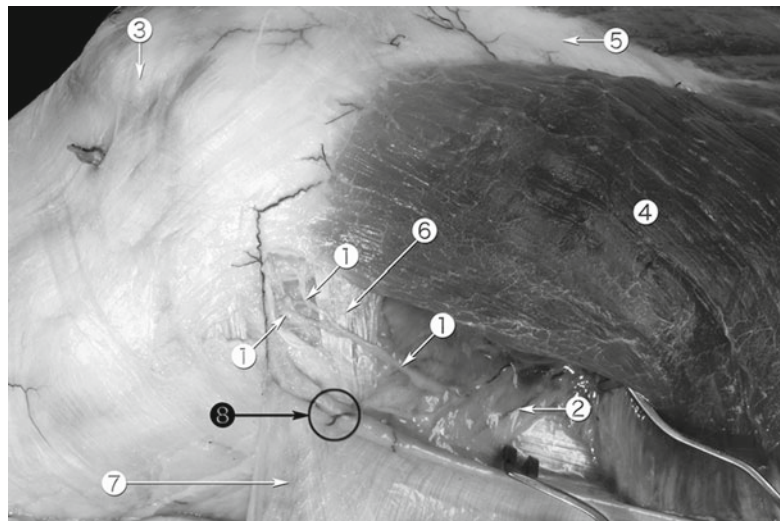


Fig. 26.3 Arthroscopic image of thermal lesion to the peripatellar synovial tissue produced by the electrocoagulator. (a) Medial region of peripatellar soft tissue before denervation. (b) Simple thermal lesion to the peripatellar soft tissue in the region closest to the patella. (c) Arthroscopic view of the lesion produced by the electrocoagulator in the medial region of the peripatellar soft tissue in order to eliminate a considerable number of pain receptors

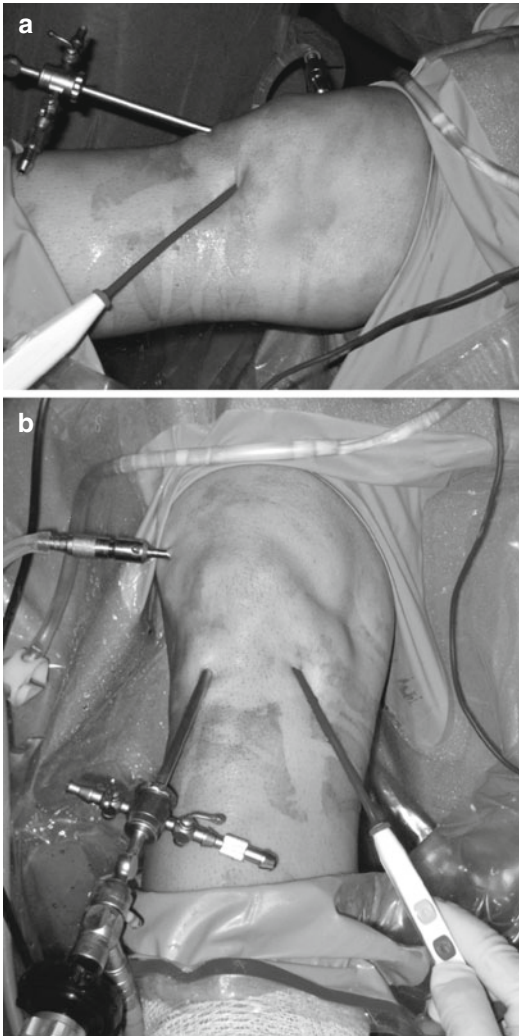


Fig. 26.4 Maximum extension of the knee and the insufflation of fluid to distend the joint will separate the patella and the peripatellar region from the usual portals in knee arthroscopy (anteromedial and anterolateral portal). The patella and the anatomic protrusion of the distal knee region limit the electrocoagulator movement and make the procedure difficult. **(a)** Medial view of the knee with electrocoagulator inserted through the anteromedial portal, and pointed to the anterior knee area. **(b)** Anterior view of the knee with electrocoagulator inserted through the anteromedial portal, and pointed to the anterior and medial knee area

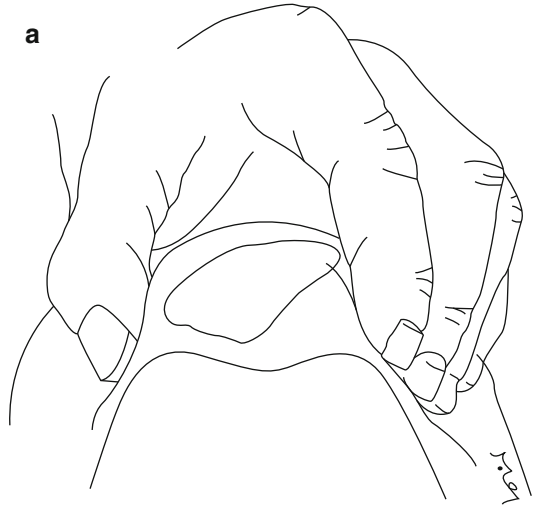


Fig. 26.5 A selective pressure with fingers on the patella or on the peripatellar tissue will bring these structures to the instruments: Schematic view **(a)**. Surgical view with the electrocoagulator inserted through the anteromedial portal **(b)**

Reconstruction of the Medial Patellofemoral Ligament: How I Do It

27

Eric W. Edmonds and Donald C. Fithian

Reconstruction of the medial patellofemoral ligament (MPFL) has been described utilizing a myriad of fixation techniques. We have elected to utilize a technique that secures the graft to the femur with

an interference screw and secures the graft to the patella by suturing it to itself after threading it through bone tunnels. This allows for easy tensioning the graft whilst not sacrificing fixation.

E.W. Edmonds, M.D. (✉)
Department of Orthopaedic Surgery,
University of California,
San Diego, CA, USA

Pediatric Orthopaedic and Scoliosis Center, Rady
Children's, Hospital San Diego,
San Diego, CA, USA
e-mail: ericwedmonds@yahoo.com

D.C. Fithian, M.D.
Southern California Permanente Medical Group,
El Cajon, CA, USA

Fig. 27.1 Intraoperative photograph demonstrating standard incisions for MPFL reconstruction, including: two anterior para-patellar arthroscopy portals, tibial incision for hamstring autograft harvest, medial patella incision, and medial distal femur incision

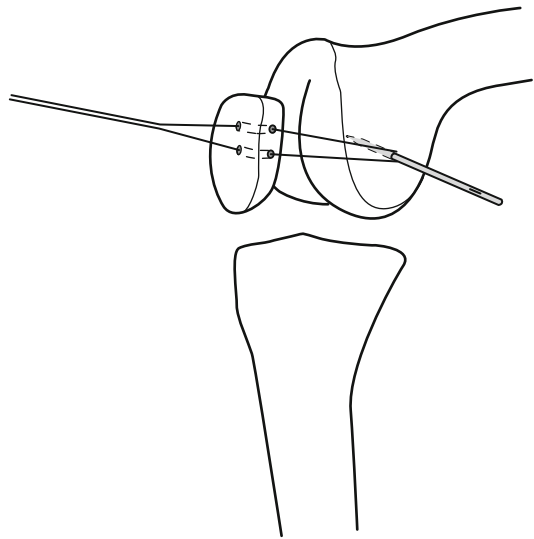


Fig. 27.2 Line drawing demonstrating assessment of isometric point on femur utilizing Bieth pin and suture passed through patella tunnels

Fig. 27.3 Line drawing demonstrating placement of graft into femoral tunnel, secured using interference screw

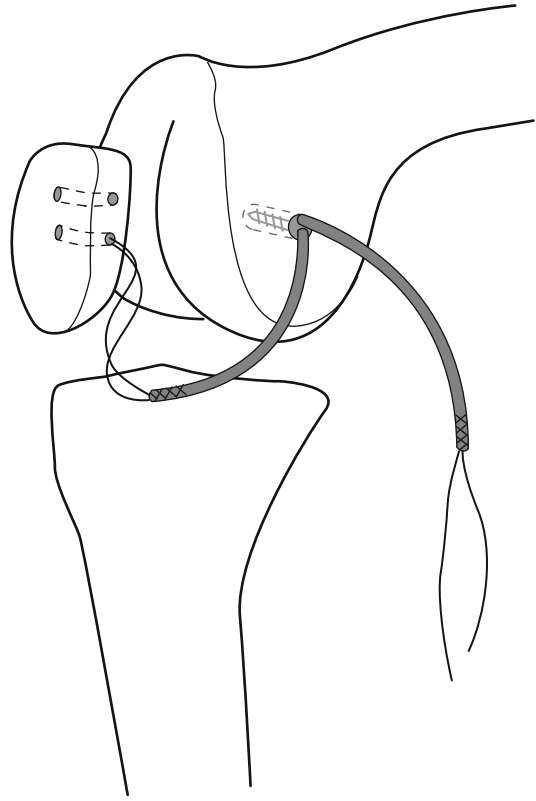


Fig. 27.4 Intraoperative photograph demonstrating graft in place after passage between two working incisions ready for placement through patellar tunnels and tensioning

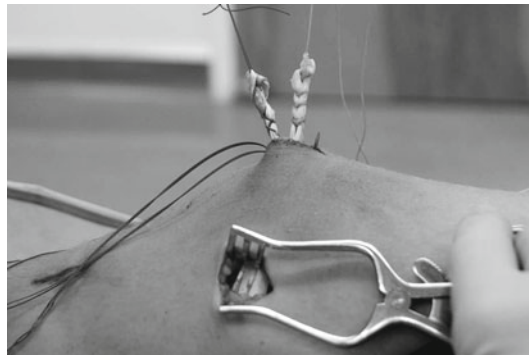
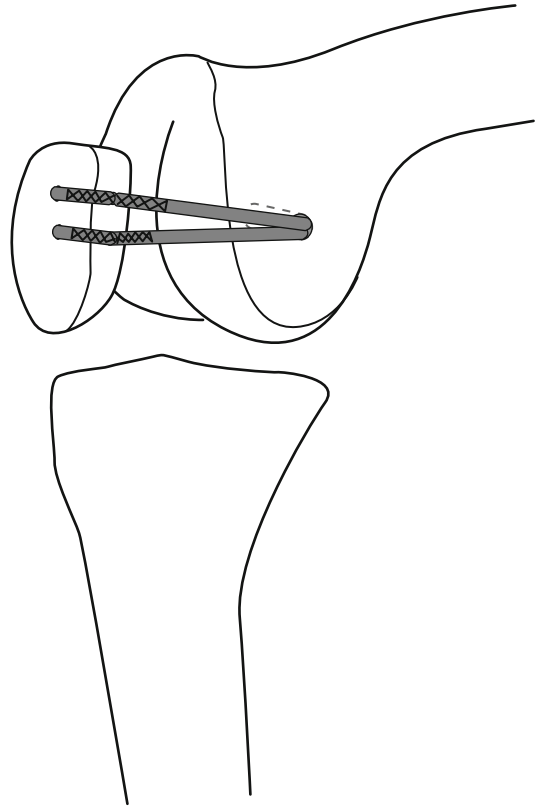


Fig. 27.5 Line drawing demonstrating secured graft, sutured onto itself, post-tensioning, at the completion of the MPFL reconstruction



Reconstruction of the Medial Patellofemoral Ligament: How I Do It

28

Robert A. Teitge and Roger Torga-Spak

The medial patellofemoral ligament (MPFL) has been recognized as the primary stabilizer against lateral patellar dislocation or subluxation. Consequently if there is lateral patellar instability there is also an insufficient MPFL. Various reconstruction procedures of the MPFL using adductor magnus, quadriceps tendon, semitendinosus, gracilis, and synthetic tissue have been recently developed.

The technique we postulate follows the same basic principles of all ligament reconstruction: (1)

selection of a sufficiently strong and stiff graft, (2) isometric graft placement, (3) correct tension, (4) adequate fixation, and (5) no condylar rubbing or impingement. Adductor longus tendon was used in most of our reconstructions, whereas quadriceps tendon autograft or bone patellar tendon allograft was preferred in cases with trochlear dysplasia based on the concept that a stronger structure is needed to compensate for inadequate support provided by a flat trochlea.

R.A. Teitge, M.D.
Department of Orthopaedics,
Wayne State University School of Medicine,
Detroit, MI, USA

R. Torga-Spak, M.D. (✉)
Department of Surgery, Faculty of Orthopaedics and
Traumatology, Instituto Universitario CEMIC,
Buenos Aires, Argentina
e-mail: rtorgaspak@gmail.com

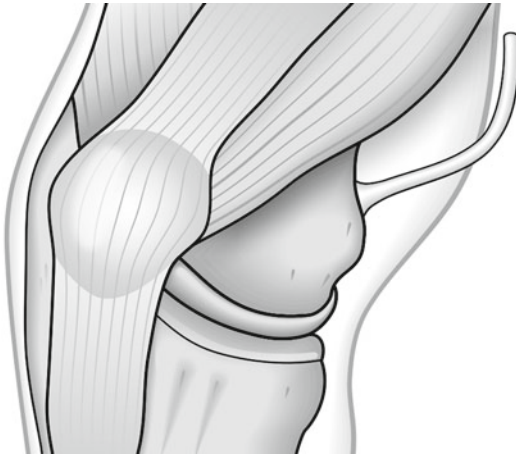


Fig. 28.1 Harvesting of the adductor magnus tendon

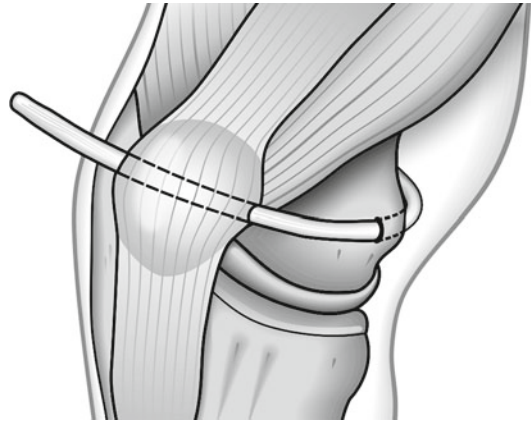


Fig. 28.3 Graft passing through the tunnel in the medial epicondyle and through the patellar tunnel

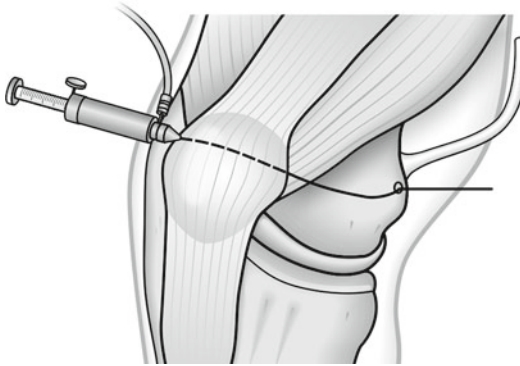


Fig. 28.2 Location of the isometric point on the medial epicondyle

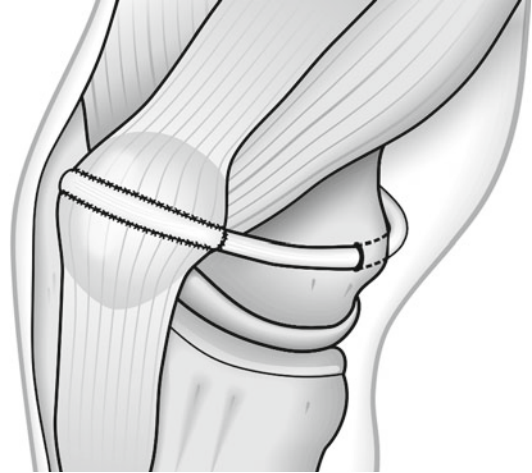


Fig. 28.4 Graft sutured to the quadriceps expansion on the anterior aspect of the patella

Pieter J. Erasmus and Mathieu Thaunat

Medial patellofemoral ligament (MPFL) reconstructions have good results with few complications notwithstanding varied techniques used. Biomechanical and technical principles should be adhered to in preventing complications. The reconstructed MPFL should be tight in extension and lax in flexion. In cases of severe patella alta, a distalization of the tibial tubercle should be considered. With maximum quadriceps contraction the tension in the patellar tendon should be

more than the tension in the reconstructed ligament. Drill holes in the patella should be through the medial rim preferably not exceeding 3.5 mm. Prominence of the reconstructed graft or fixation material over the medial condyle will lead to localized tenderness and is easily avoided by using non prominent fixation devices. There seems to be no progression in patellofemoral degeneration after MPFL reconstructions, in follow up periods of 7–12 years.

P.J. Erasmus, M.D. (✉)
University of Stellenbosch, Mediclinic, Die Boord,
Stellenbosch, South Africa
e-mail: pieter@orthoclinic.co.za

M. Thaunat, M.D.
Department of Orthopedic Surgery,
Hopital André Mignot,
Le Chesnay, France

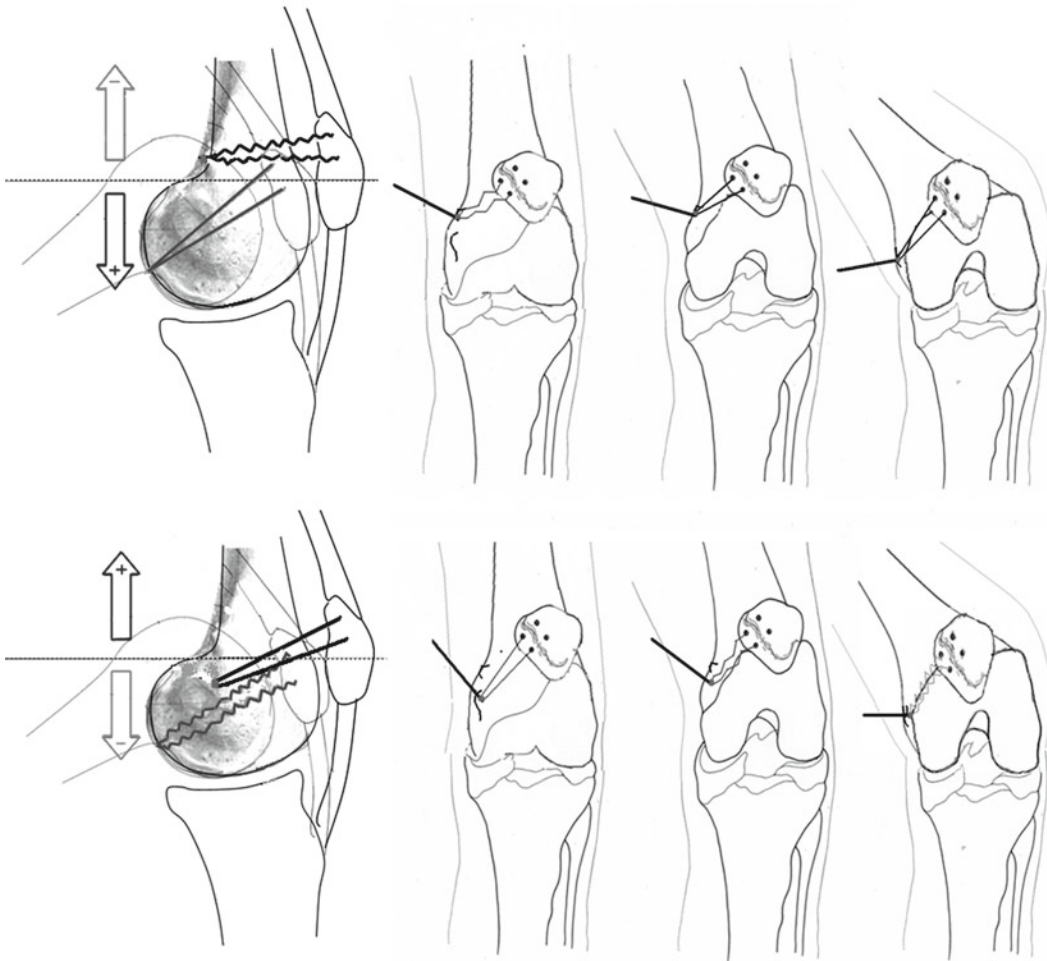


Fig. 29.1 A proximal position on the femur will result in a graft that is loose in extension and tight in flexion. Conversely a distal femoral position will result in a graft that is tight in extension and loose in flexion

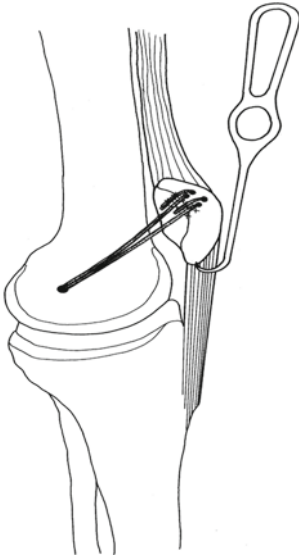


Fig. 29.2 Tensioning the MPFL graft in full extension ensuring that the tension in the reconstruction is less than in the patellar tendon

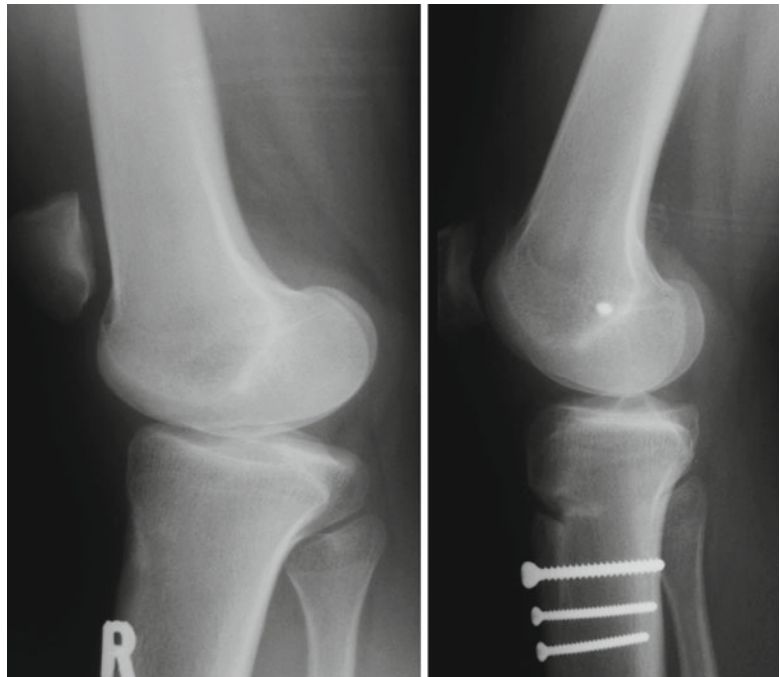


Fig. 29.3 Distalization of the tibial tubercle combined with a MPFL reconstruction

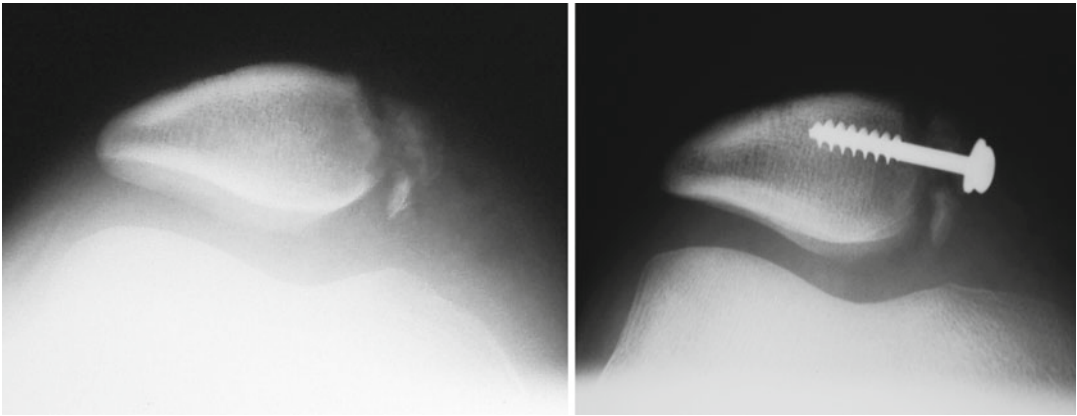


Fig. 29.4 Redislocation after MPFL reconstruction with a fracture of the medial rim reattached with a screw and washer



Fig. 29.5 Central patella fracture secondary to drill hole that exit too centrally on the patella



Fig. 29.6 Transverse patella fracture secondary to transverse drill hole for MPFL reconstruction

Reconstruction of the Lateral Patellofemoral Ligament: How I Do It

30

Jack T. Andrish

Reconstruction of the lateral patellofemoral ligament is a valuable tool for the treatment of symptomatic medial instability of the patella. However, remembering that the lateral retinaculum is a restraint to lateral translation of the patella as well as medial, its clinical usefulness is also useful during revision surgery for recurrent lateral

instability. This chapter describes, step by step, one method of reconstructing the deep transverse retinaculum using a transfer of the iliotibial band to be used for the treatment of iatrogenic medial patellar instability as well as a possible component of revision surgery for lateral patellar instability.

J.T. Andrish, M.D.
Department of Orthopaedic Surgery,
Center of Sports Health, Cleveland Clinic,
Cleveland, OH, USA
e-mail: andrisj@ccf.org

Fig. 30.1 The lateral retinaculum is exposed to include the iliotibial band (a). The anterior half of the iliotibial band is detached from Gurdy's tubercle and then reflected proximally, beyond the lateral femoral epicondyle (b) (Copyright The Cleveland Clinic Foundation)

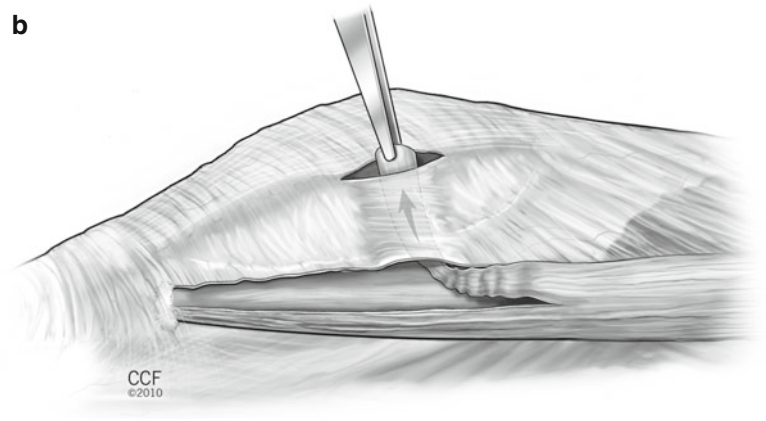
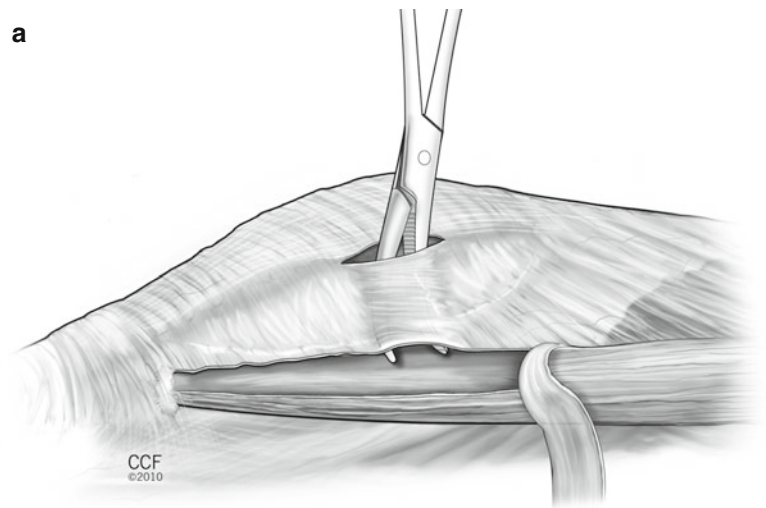
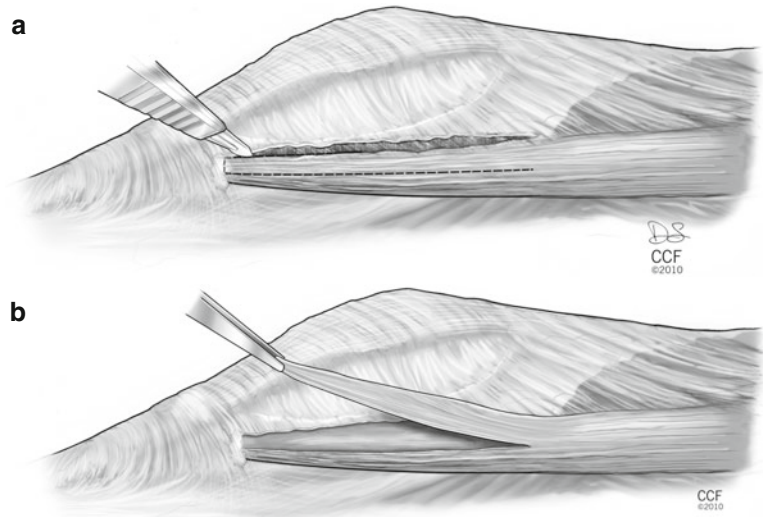


Fig. 30.2 If there is some manner of attenuated lateral retinaculum remaining, an interval is developed between the lateral capsule and the retinaculum (a). The strip of iliotibial band is then brought through this interval to be attached to the lateral border of the junction of the middle and proximal third of the patella (b) (Copyright The Cleveland Clinic Foundation)

Fig. 30.3 The transferred tendon is then attached by suture to the remaining peripatellar retinacular tissue, or by suture anchor. It is not necessary to attempt attachment by a drill hole (Copyright The Cleveland Clinic Foundation)

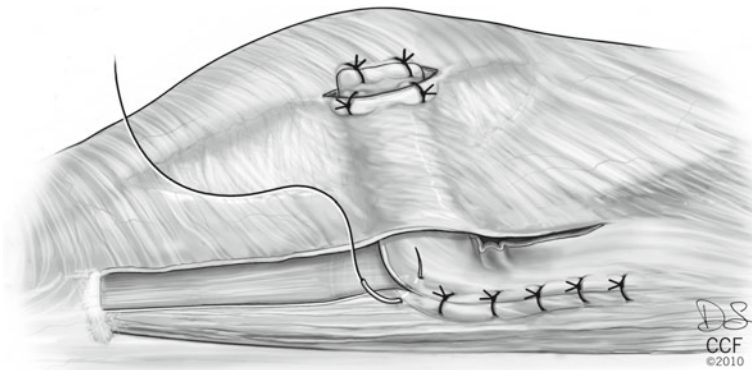
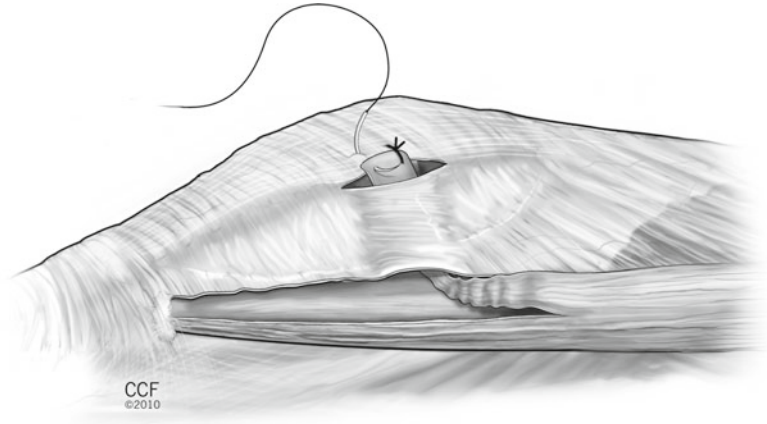


Fig. 30.4 Our goal is to make the transferred tendon transversely oriented and “attached” to the remaining intact iliotibial band at the level of the lateral femoral epicondyle. In order to do this, and to adjust and establish tension, a series of sutures are placed reattaching the posterior border of the transferred tendon to the anterior

border of the remaining intact iliotibial band. This begins at the proximal location of the isolation and works distally until the desired orientation and tension of the transfer has been achieved (Copyright The Cleveland Clinic Foundation)

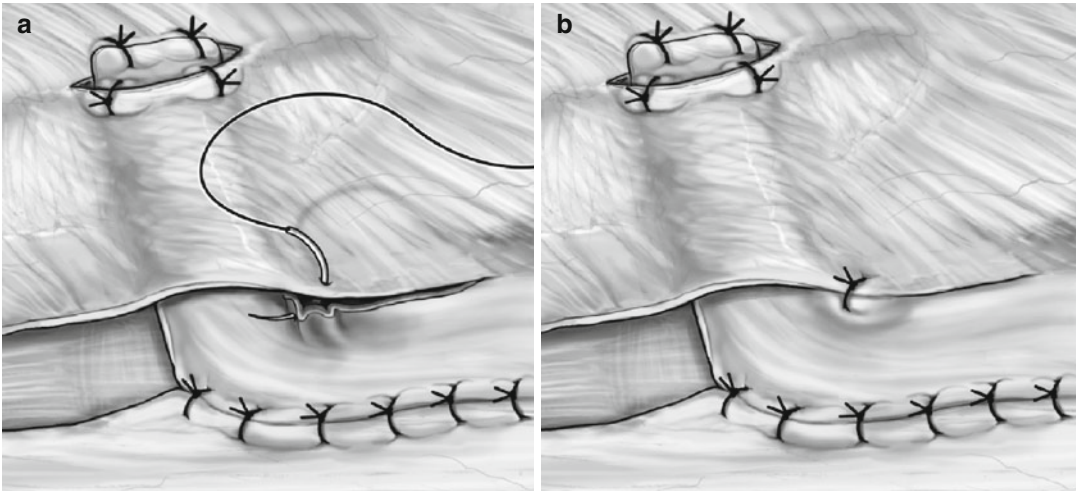


Fig. 30.5 Often, there will be a kink at the bend of the transfer. A simple suture from the corner of the iliotibial band to remaining retinacular tissue improves the alignment (a). All sutures in place and properly “tensioned,” the deep transverse lateral retinaculum has been reconstructed (b) (Copyright The Cleveland Clinic Foundation)

Reconstruction of the Lateral Patellofemoral Ligament: How I Do It

31

Robert A. Teitge and Roger Torga-Spak

Medial dislocation or subluxation of the patella is a disabling condition that can occur after an isolated lateral release, or after lateral release in combination with tibial tubercle transfer or medial soft tissue imbrications.

Techniques to repair the lateral retinaculum can be found in the literature, as well as descriptions of reconstruction with local soft-tissue augmentation (fascia lata, patellar tendon). In our experience with lateral retinacular repair and imbrication, a noticeable increase in medial

excursion usually would reappear after the first postoperative year. This led us to develop a technique for lateral patellofemoral ligament (LPFL) reconstruction following the same principles of the medial patellofemoral ligament reconstruction previously described: (1) selection of a sufficiently strong and stiff graft, (2) isometric graft placement, (3) adequate fixation, (4) correct tension, and (5) no condylar rubbing or impingement.

R.A. Teitge, M.D.
Department of Orthopaedics, Wayne State University
School of Medicine,
Detroit, MI, USA

R. Torga-Spak, M.D. (✉)
Department of Surgery, Faculty of Orthopaedics
and Traumatology, Instituto Universitario CEMIC,
Buenos Aires, Argentina
e-mail: rogers@traumatologiapenta.com.ar

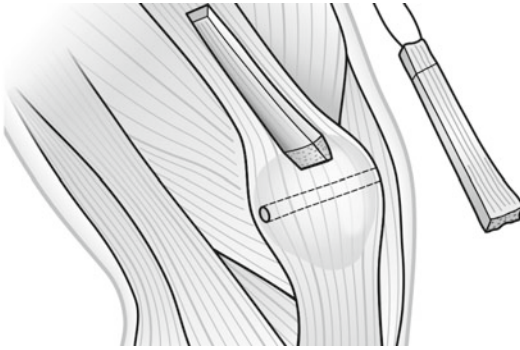


Fig. 31.1 Harvesting of the quadriceps graft. A transverse tunnel is performed through the patella

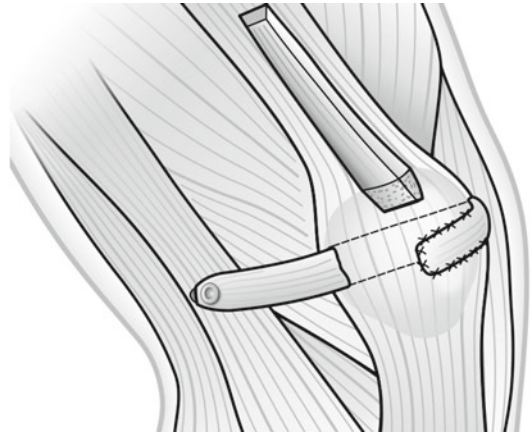


Fig. 31.3 Quadriceps graft fixed to the lateral condyle, passed through the patellar tunnel, and sutured on the anterior aspect of the patella

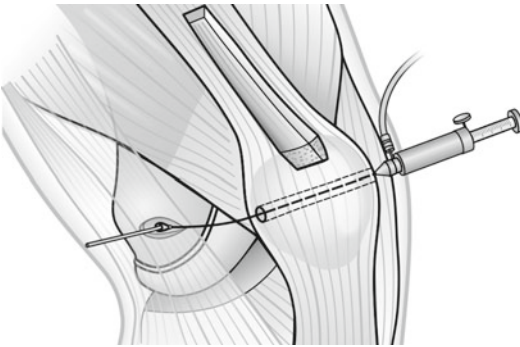


Fig. 31.2 Isometric location of the insertion point in the lateral condyle

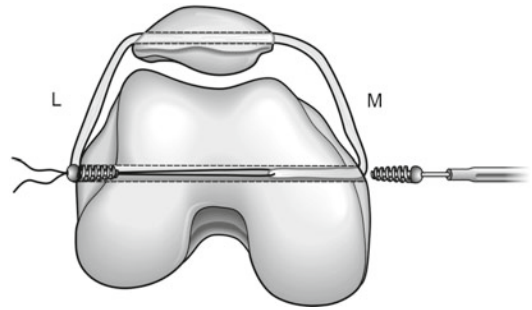


Fig. 31.4 Combined MPFL and LPFL reconstruction passing a free graft through a transverse patellar and epicondylar tunnel

Roland M. Biedert and Philippe M. Tscholl

A normal patellofemoral gliding mechanism with perfect stability is guaranteed by the complex interaction of skeletal geometry, soft tissues, and neuromuscular control. During knee flexion, the patella moves from a medial to a lateral tilted position as knee flexion approaches 90°. Abnormal skeletal geometry – such as increased femoral anteversion, trochlear dysplasia, patella alta or infera, increased tibial external torsion, increased

tibial tubercle lateralization, and variations of combined deformities – may lead to patellofemoral complaints. Altered vectors and forces acting on the patellofemoral joint (PFJ) can cause cartilage failure with secondary osteoarthritis instability, and musculotendinous insufficiency. Osteotomy with soft tissue balancing might be the best treatment, depending on the underlying pathology. Surgery aims to eliminate the present pathomorphology.

R.M. Biedert, M.D. (✉)

University of Basel,
Biel, Switzerland

Sportclinic Villa Linde,
Swiss Olympic Medical Center Magglingen-Biel,
Biel, Switzerland
e-mail: r.biedert@bluewin.ch

P.M. Tscholl, M.D.
Sportclinic Villa Linde, Swiss Olympic Medical Center
Magglingen-Biel,
Biel, Switzerland

Fig. 32.1 (a) Internally rotated patellae in comfortable standing position. (b) Active correction of the patella to normal causes increased external rotation of the feet

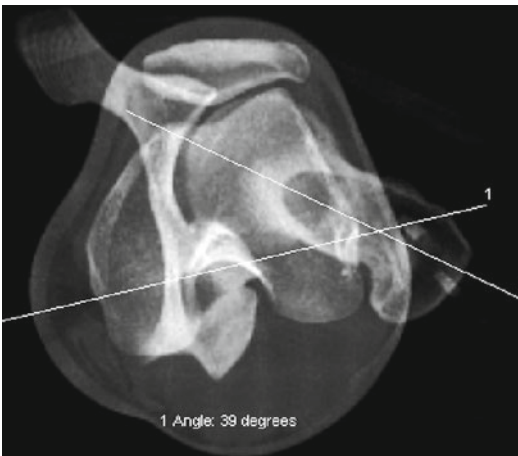


Fig. 32.2 Axial CT evaluation of the femoral anteversion. The femoral anteversion is 39°

Fig. 32.3 Two Kirschner wires indicate the angle of correction

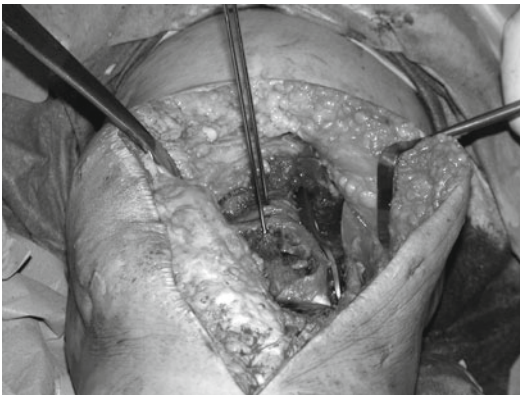
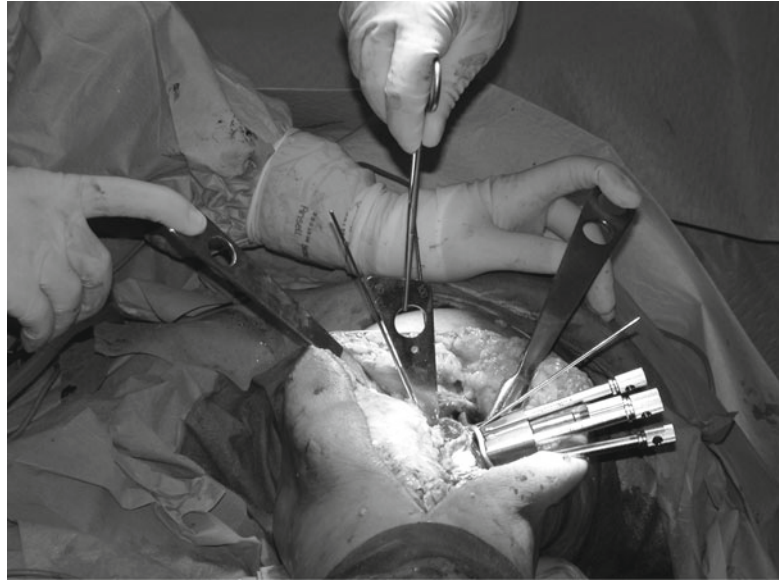


Fig. 32.4 Fixation of the supracondylar rotation osteotomy

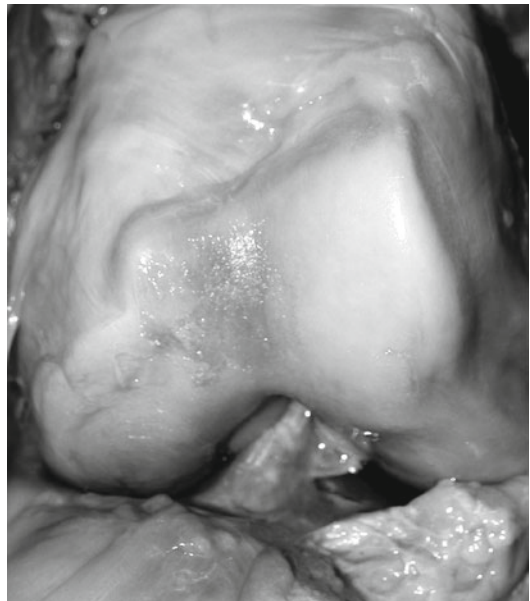


Fig. 32.5 Shape of a normal trochlea (cadaver study)

Fig. 32.6 (a) Well-centered patella. (b) Muscle contraction causes dynamic superolateral patellar subluxation

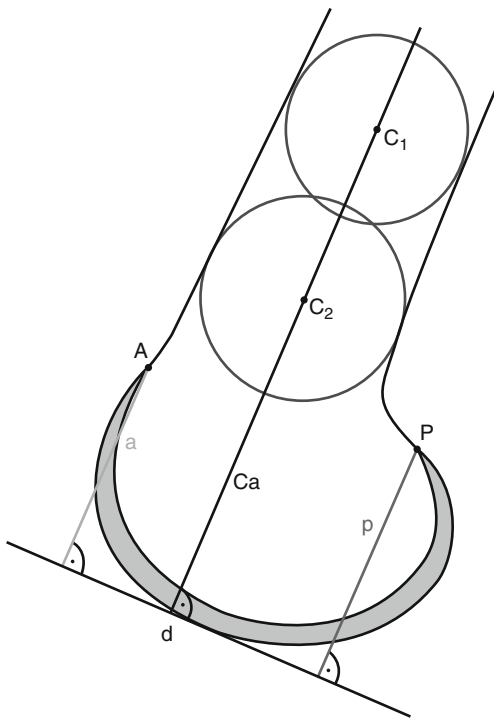
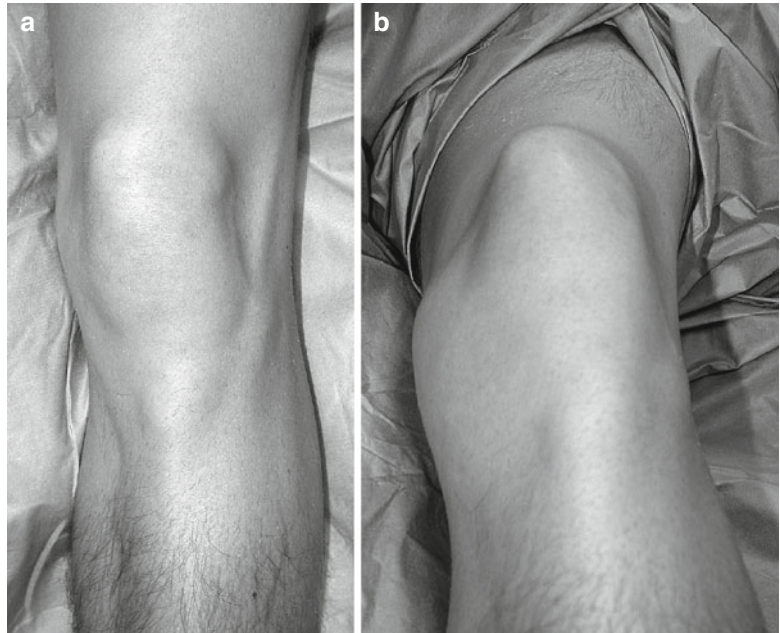


Fig. 32.7 MR measurements of the lateral condyle index



Fig. 32.8 MR measurement shows a too short lateral condyle index of 75.2 %

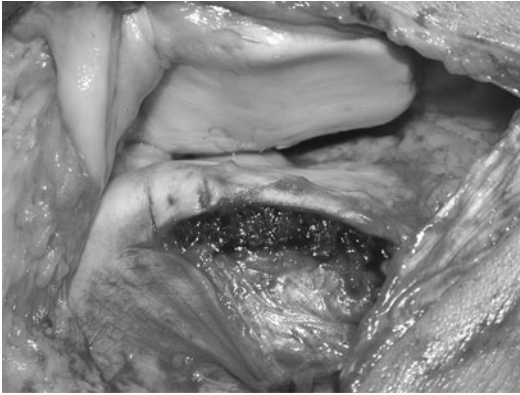


Fig. 32.9 Lengthening osteotomy

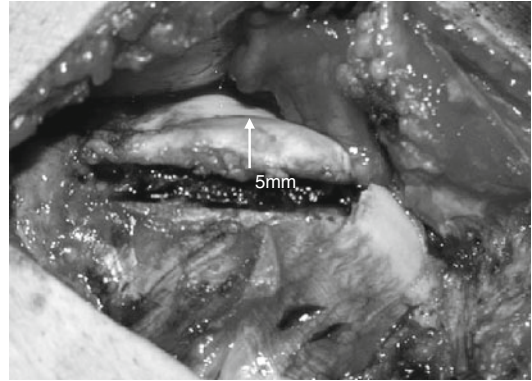


Fig. 32.11 Intraoperative view after elevation of a flat trochlea

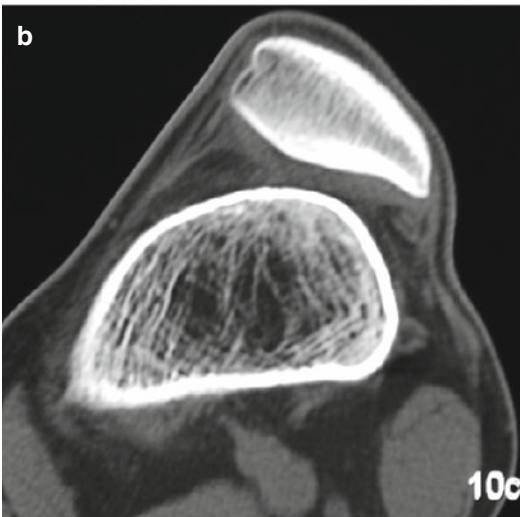


Fig. 32.10 (a) Axial CT scans with flat trochlea and lateralization of the patella (*in extension*). (b) Increased lateral subluxation of the patella with muscle contraction (same patient as in Fig. 32.10a)

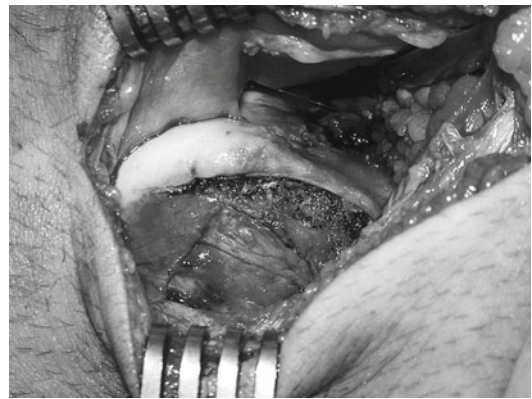


Fig. 32.12 Lengthening and elevation of a flat and too short trochlea (*right knee*)

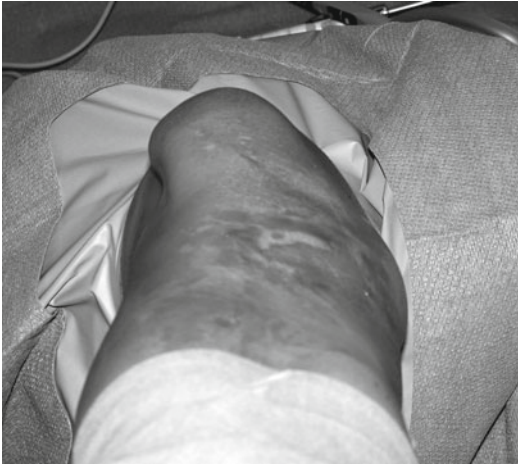


Fig. 32.13 Subluxation of the patella (*in extension, relaxed*)

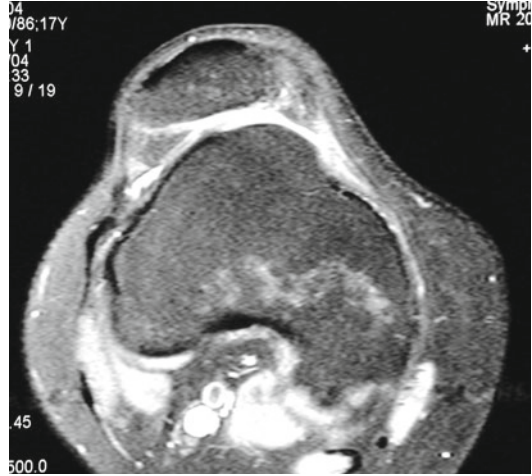


Fig. 32.15 Central bump with severe patellar subluxation (*axial MRI*)



Fig. 32.14 Crossing sign (*arrow*)

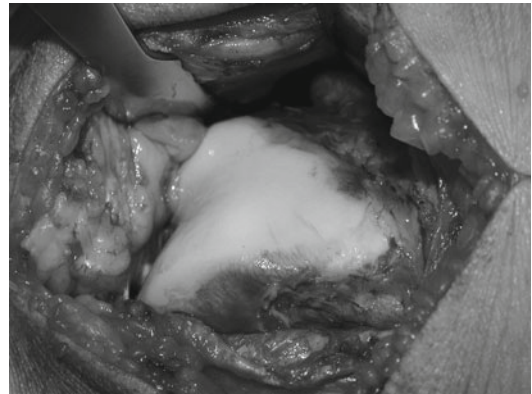


Fig. 32.16 Convex proximal trochlea with central bump

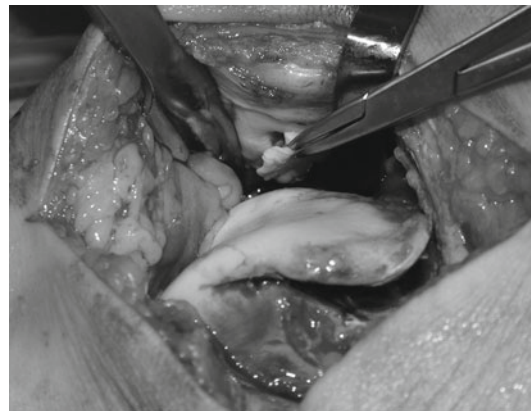


Fig. 32.17 Osteochondral flap

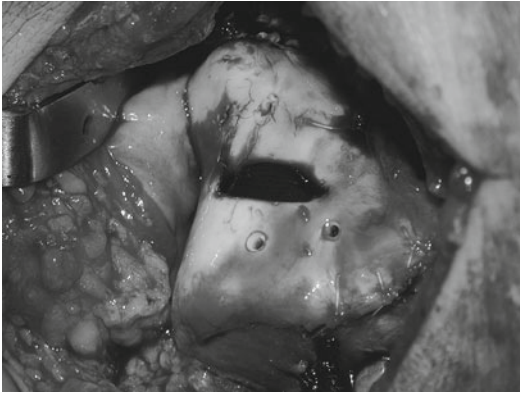


Fig. 32.18 Deepened trochlea with refixation of the osteochondral flap

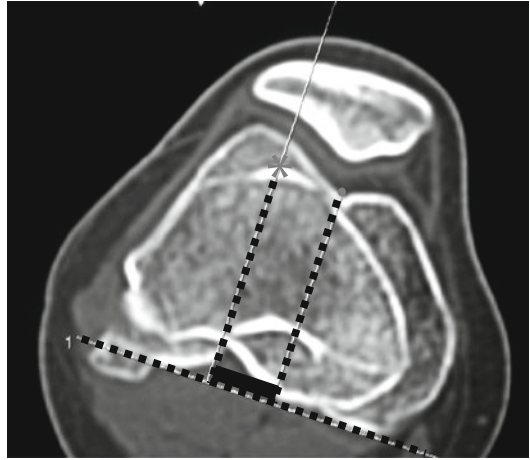


Fig. 32.20 TT-TG distance measurements on axial CT scans

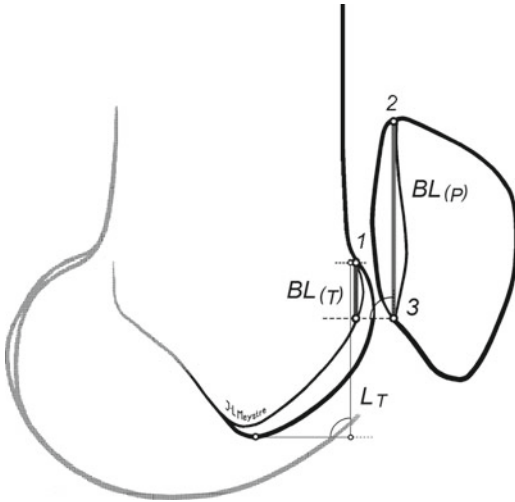


Fig. 32.19 MR measurement for the patello-trochlear index. 1 Most anterior aspect of the trochlear cartilage, 2 Most superior aspect of the cartilage of the patella, 3 Most inferior aspect of the cartilage of the patella, *BLP* Baseline Patella, *BLT* Baseline trochlea

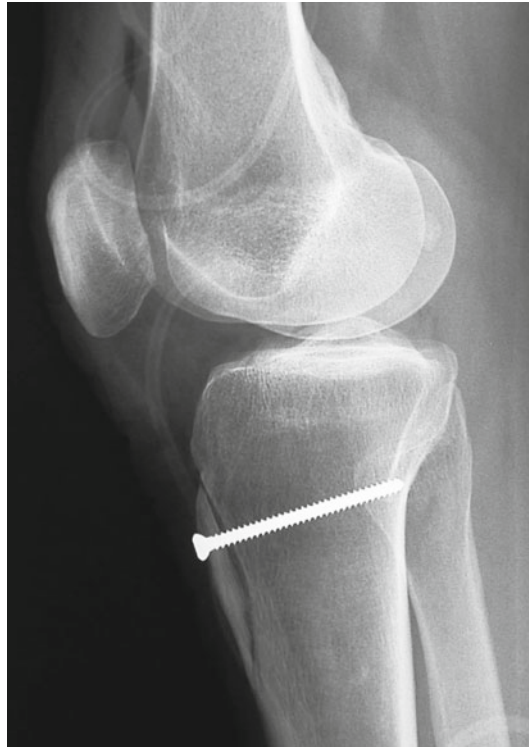


Fig. 32.21 Screw fixation after medialization of the tibial tubercle

David Dejour and Paulo Renato F. Saggin

Deepening trochleoplasty is a very rare procedure in the objective patellar dislocation group. It concerns only patients with high grade trochlear dysplasia types B or D and abnormal patellar tracking. It is part of the “menu à la

carte” which leads to the correction of the anatomical abnormalities, one by one. The technical procedure is highly demanding and prone to complications. It is, however, very effective in providing stability.

D. Dejour, M.D.
Lyon Ortho Clinic,
Lyon, France

P.R.F. Saggin, M.D. (✉)
Instituto de Ortopedia e Traumatologia (I.O.T.),
Passo Fundo, RS, Brazil
e-mail: paulosaggin@yahoo.com.br



Fig. 33.1 High-grade trochlear dysplasia (anterior view of a right knee). There is no sulcus, and in the lateral aspect (left) a big bump can be observed

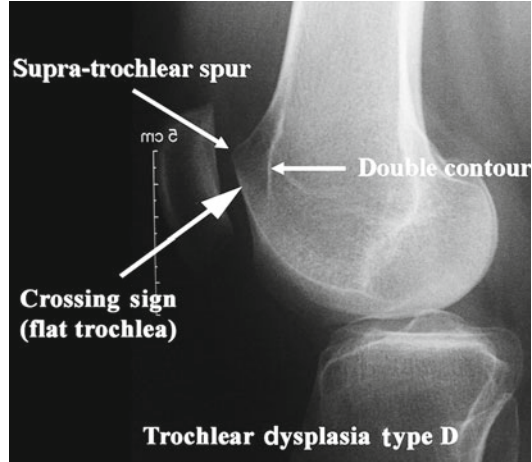


Fig. 33.3 To analyze the trochlear dysplasia a true profile is needed with a perfect superimposition of the posterior femoral condyles. The three trochlear dysplasia signs are: (1) the crossing sign; (2) the supratrochlear spur; and (3) the double-contour which goes below the crossing sign

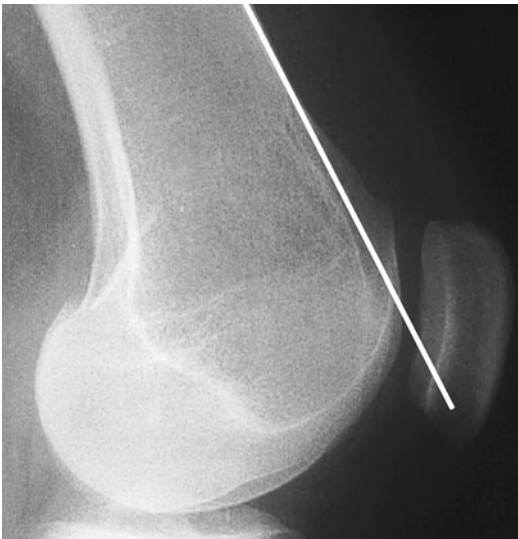


Fig. 33.2 The trochlear bump is calculated as the amount of trochlea which is in front of a line parallel to the anterior femoral cortex. Alternatively, the sulcus floor position can also be calculated from this line

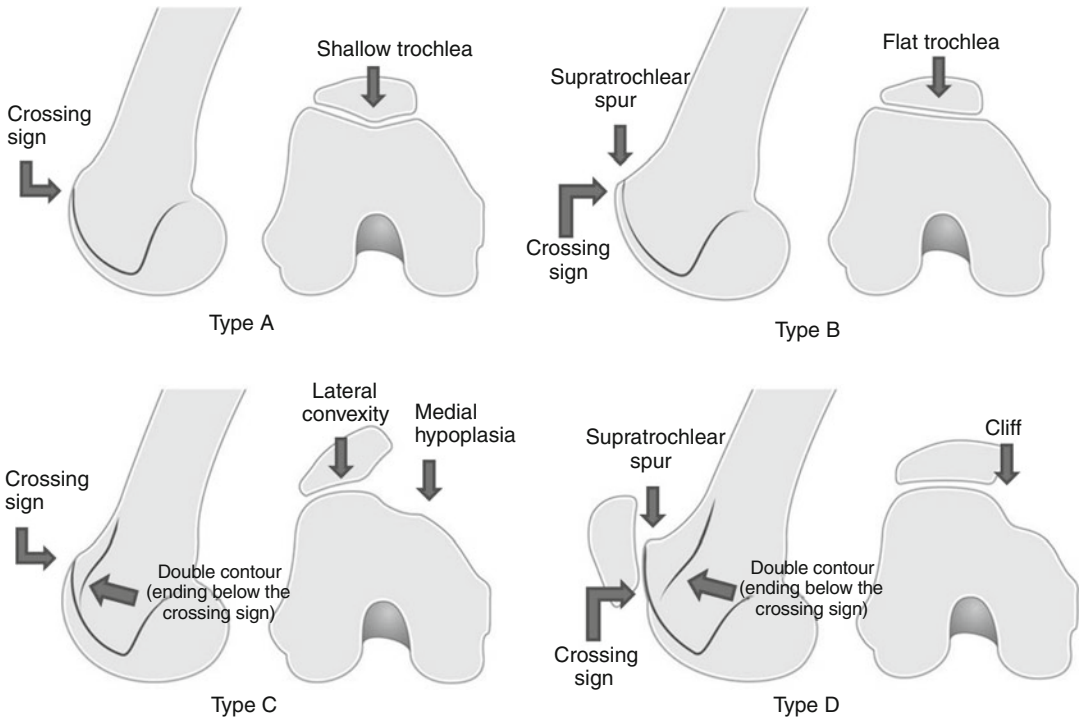


Fig. 33.4 Trochlear dysplasia classification according to David Dejour

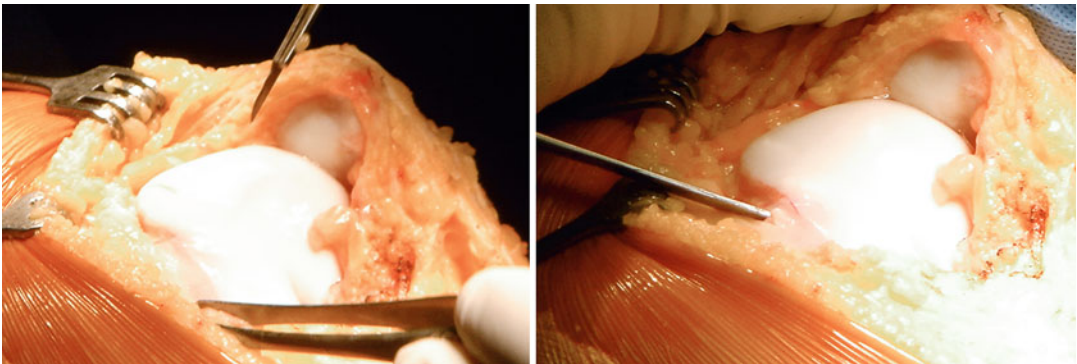


Fig. 33.5 Surgical exposure. The periosteum is incised along the osteochondral edge and reflected away from the trochlear margin. The anterior femoral cortex should be visible to guide the bone resection

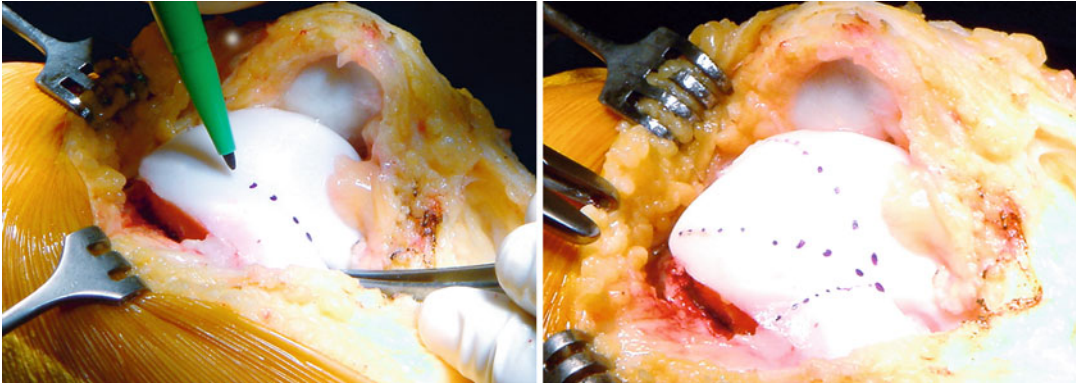


Fig. 33.6 After the surgical exposure, the new trochlea is drawn. From the intercondylar notch, the bottom of the sulcus and the facets are planned

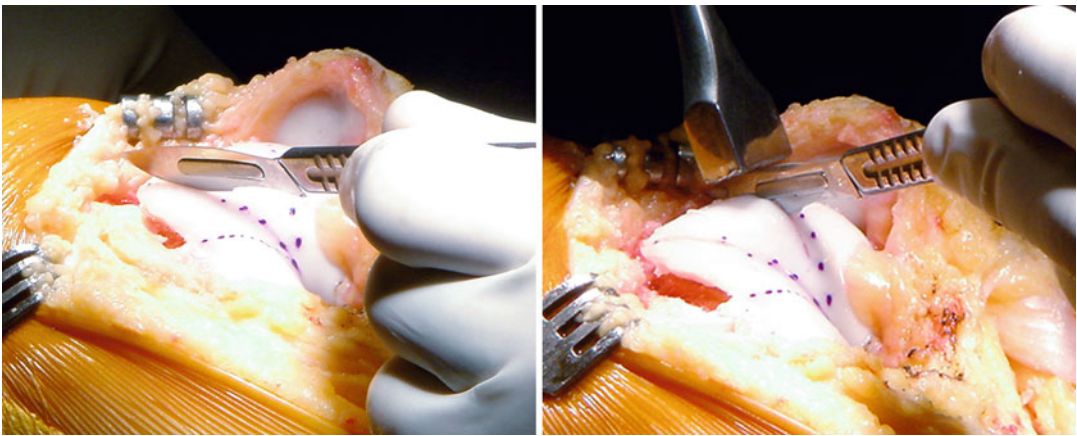


Fig. 33.7 In order to allow further modeling to the underlying bone bed, the osteochondral flaps may be cut in the sulcus and facets lines

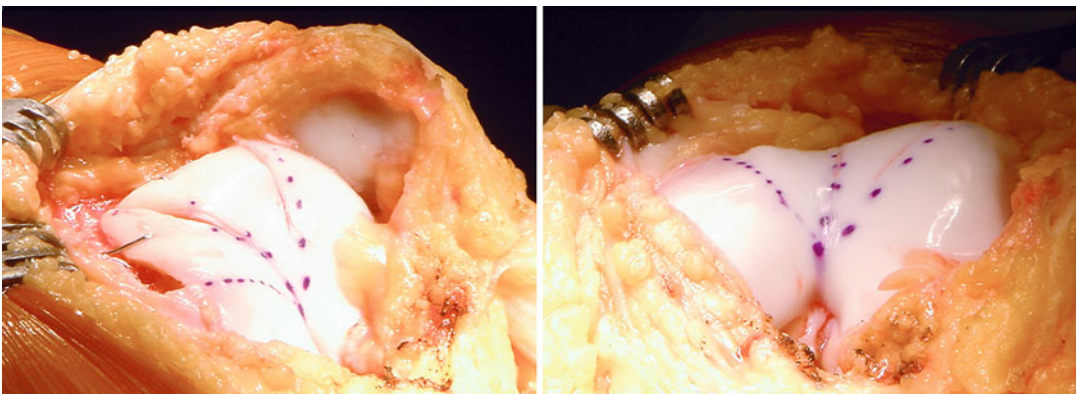


Fig. 33.8 Lateral and anterior views of dysplastic trochlea after trochleoplasty. Notice that the sulcus and facets relationship resembles a “normal trochlea”

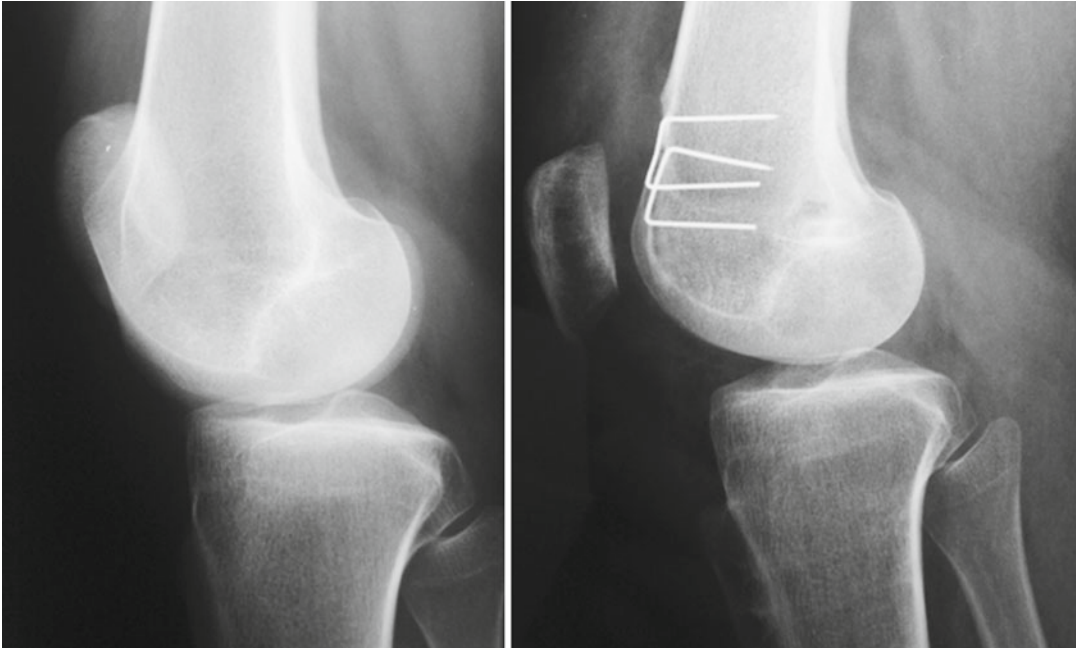


Fig. 33.9 Pre- and postoperative lateral x-rays showing the resection of the supratrochlear bump and trochlear prominence correction. Additionally, patellar tilt is clearly improved

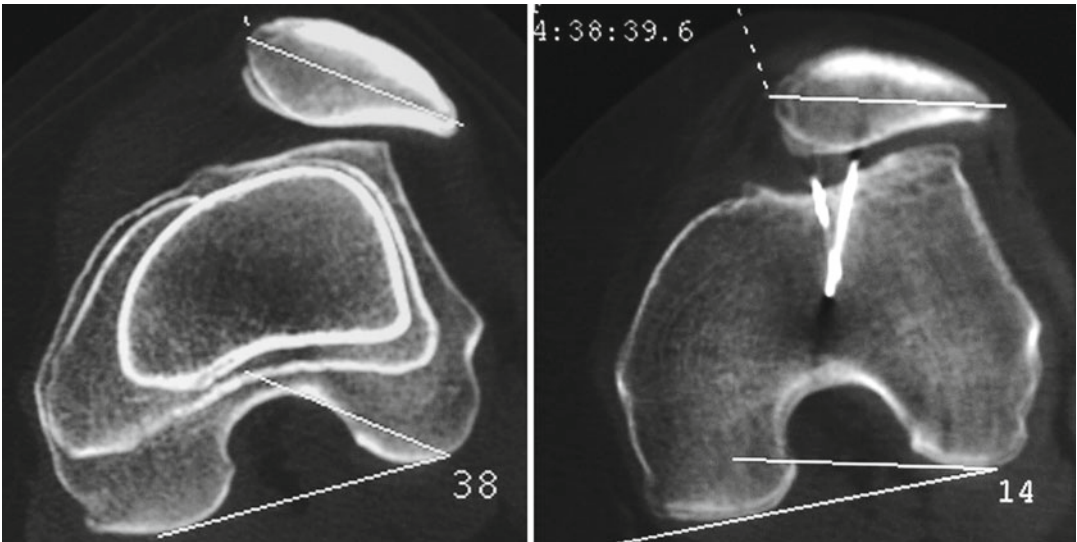


Fig. 33.10 CT scan axial views before and after trochleoplasty. The trochlear sulcus is restored and patellar tilt is corrected. Patellar subluxation is also improved

Robert A. Teitge and Roger Torga-Spak

1. Miserable Malalignment as described by James exists and must be recognized in the patient with PF pain, instability or arthrosis.
2. Torsional Limb Malalignment may present with only pain or may coexist with Patellar Instability and/or Patellar Chondrosis and it may be a biomechanical contributor to the etiology of both instability and arthrosis, but all three conditions (alignment, instability and cartilage injury) must be evaluated independently.
3. Restoration of normal limb alignment in all three planes is thought to restore the normal direction of the force vectors which cross the knee joint.
4. The Transverse (Horizontal) plane is frequently overlooked and is best evaluated with a CT scan which allows the axis of the hip, knee and ankle to be measured relative to each other.
5. Realignment of the femur and tibia by rotational osteotomy may not only be appropriate but necessary. It is the only reasonable surgical treatment for PF symptoms which are due to horizontal skeletal malalignment.
6. Surgery for instability or for cartilage restoration is more likely to fail in the mechanical environment of persistent lower limb maldorsion.
7. Surgical morbidity must not be underestimated.

R.A. Teitge, M.D.
Department of Orthopaedics, Wayne State University
School of Medicine,
Detroit, MI, USA

R. Torga-Spak, M.D. (✉)
Department of Surgery, Faculty of Orthopaedics and
Traumatology, Instituto Universitario CEMIC,
Buenos Aires, Argentina
e-mail: rtorgaspak@gmail.com

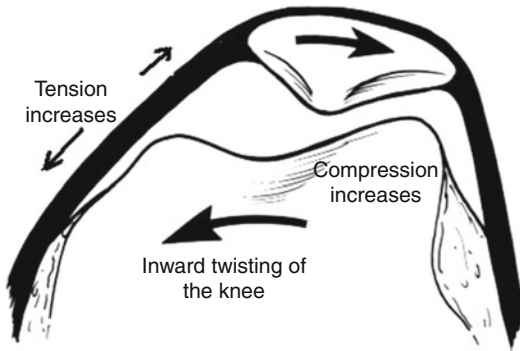


Fig. 34.1 When the knee joint is twisted medially and the body is moving forward, there is an increase in tension on the medial PF ligaments, while the lateral PF joint is compressed

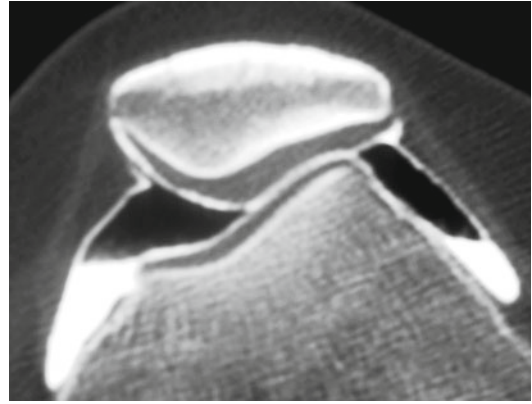


Fig. 34.2 Increased sclerosis in the lateral patellar facet subchondral bone is indicative of a localized chronic increase in pressure. This double contrast CT arthrogram demonstrates intact articular cartilage on both the patella and the trochlea with an increased compression of cartilage at the lateral facet

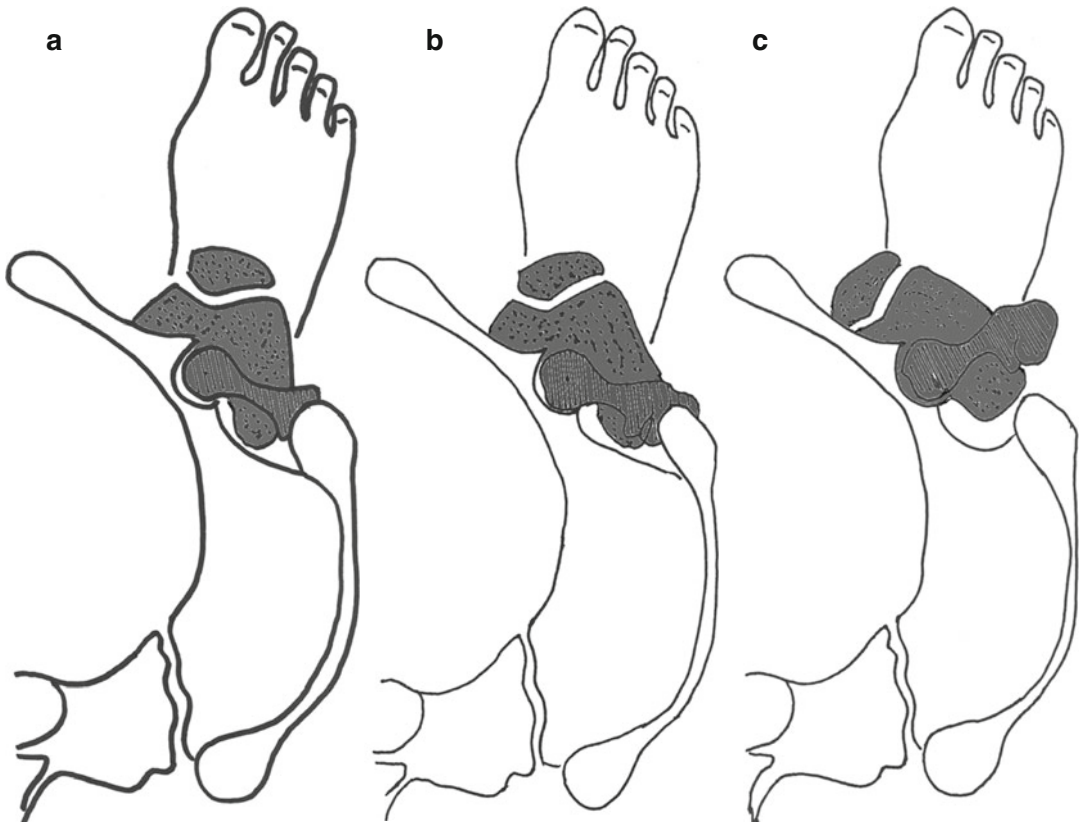


Fig. 34.3 (a) Normal male with femoral anteversion of 13° and external tibial torsion of 21° . Note that with a foot progression angle of 13° , the knee joint faces slightly outward. (b) Normal female: 13° femoral anteversion, 27° tibial torsion (external). Note that knee joint is pointing slightly inward, and the greater trochanter slightly more anterior than the normal male. (c) Female with 30°

increase in tibial torsion. To keep the foot progression angle normal the knee joint axis points inward nearly 30° causing increased strain on the medial knee and increased lateral PF pressures. The hip appears markedly internally rotated with the greater trochanter pointing somewhat anteriorly

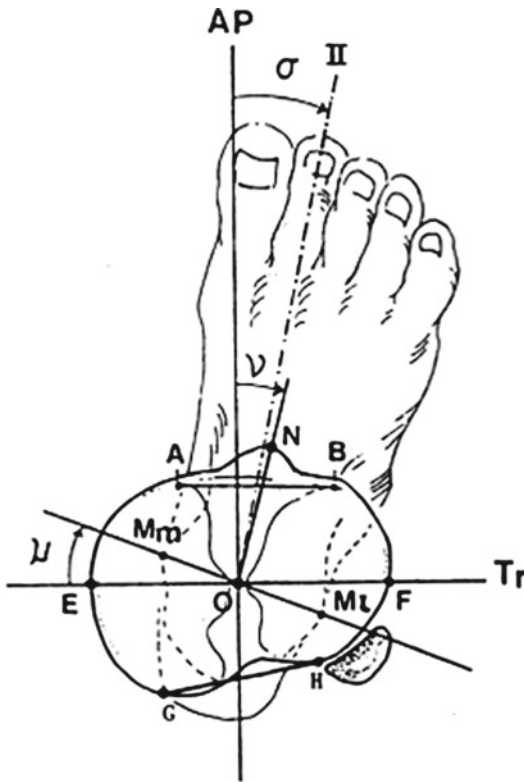


Fig. 34.4 The measure of tibial torsion is the angle m measuring the angle between the transverse axis of the proximal tibia (Tr) and the ankle joint axis Mm-ML (From Yoshioka Y, Siu DW, Scudamore RA, et al. Tibial anatomy and functional axes. J Orthop Res. 1989;7:132-7)

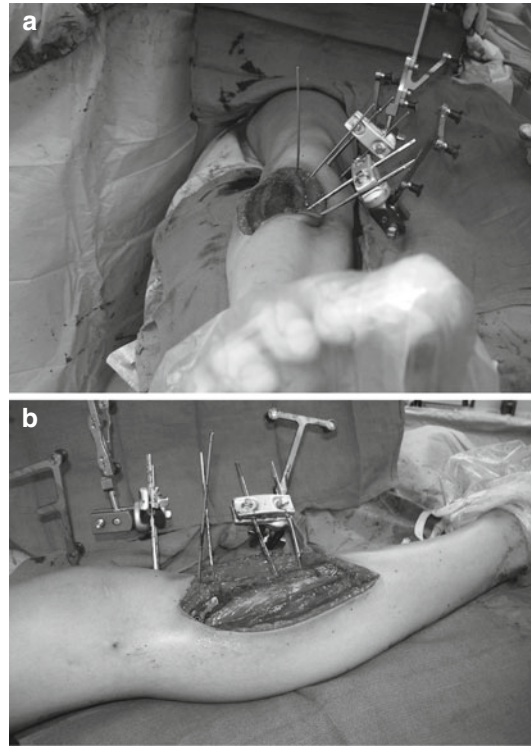


Fig. 34.5 Internal rotational osteotomy of the tibia for correction of 30° excess external tibial torsion. (a) Note that K-wires which were set parallel and located above and below the osteotomy are now separated by the 30° internal rotation of the distal fragment. (b) Navigation reflectors (trackers) are attached to the distal fragment below the osteotomy, the proximal fragment above the osteotomy and the femur

Robert A. Teitge and Roger Torga-Spak

Skeletal alignment in all three planes has a great influence on patellar tracking and loading. The source of patellofemoral loading is extraarticular; this is the reason that operations limited to the knee joint frequently fail if this is not recognized. The treatment of skeletal malalignment requires the correct bony operation. If there is genu valgum because of a short lateral femoral condyle, a femoral varus osteotomy is indicated. If the genu valgum is the result of a valgus bow to the tibia then a varus osteotomy of the tibia

near the deformity is indicated. Genu varum with medial trochlear degeneration should be treated with tibial valgus osteotomy. Inward pointing knees with secondary lateral subluxation should be treated with external rotation femoral osteotomy if it is caused by increased femoral anteversion; internal rotation tibial osteotomy if it is caused by increased external tibial torsion. Combined deformities are not uncommon and the type of osteotomy and location depends on the deformity.

R.A. Teitge, M.D.
Department of Orthopaedics,
Wayne State University School of Medicine,
Detroit, MI, USA

R. Torga-Spak, M.D. (✉)
Department of Surgery, Faculty of Orthopaedics and
Traumatology, Instituto Universitario CEMIC,
Buenos Aires, Argentina
e-mail: rtorgaspak@gmail.com

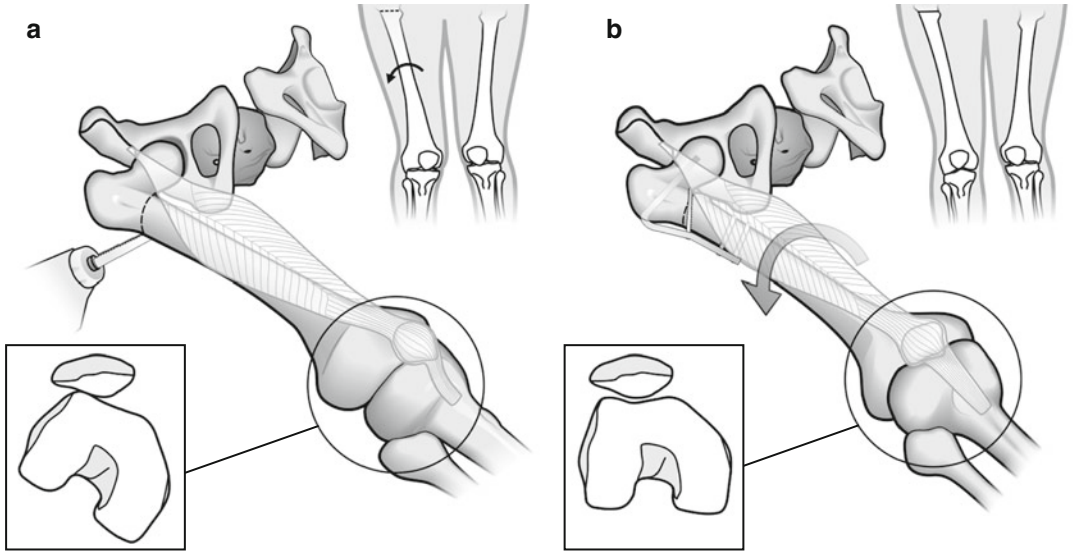


Fig. 35.1 (a) The transverse osteotomy is performed at the level of the top of the lesser trochanter. (b) The distal fragment is rotated externally until the desired rotation is obtained

Paulo Renato F. Saggin and David Dejour

Patella infera represents the low position of the patella in relation to the femur and the tibia. Two terminologies are used to qualify this low position of the patella: “patella baja” or “patella infera”. As the latin form is the common way to describe the high position of the patella (patella alta), it is coherent to define patella infera as the appropriate term. Its real importance lies in the biomechanical abnormality caused by the inferior situation of the patella in relation to the knee. While normal patellae are not engaged in the trochlea in extension, in cases of patella infera the patella is always in contact with the trochlea. It is most of the time a complication of a traumatic

event or a previous surgery on the knee. Rarely, it is a constitutional position of the patella and it is not pathological.

Patella alta is one of the four anatomical factors leading to patellar instability (the others are trochlear dysplasia, excessive TT-TG and patellar tilt). The patella engages the femoral trochlea late in flexion, which predisposes to instability. As in patella infera, the diagnosis is established on the lateral view, where appropriate measurements are performed.

Patella alta correction is performed through distal transfer of the tibial tubercle. Associated abnormalities should be corrected simultaneously to achieve patellar stability.

P.R.F. Saggin, M.D. (✉)
Instituto de Ortopedia e Traumatologia (I.O.T.),
Passo Fundo, RS, Brazil
e-mail: paulosaggin@yahoo.com.br

D. Dejour, M.D.
Lyon Ortho Clinic,
Lyon, France

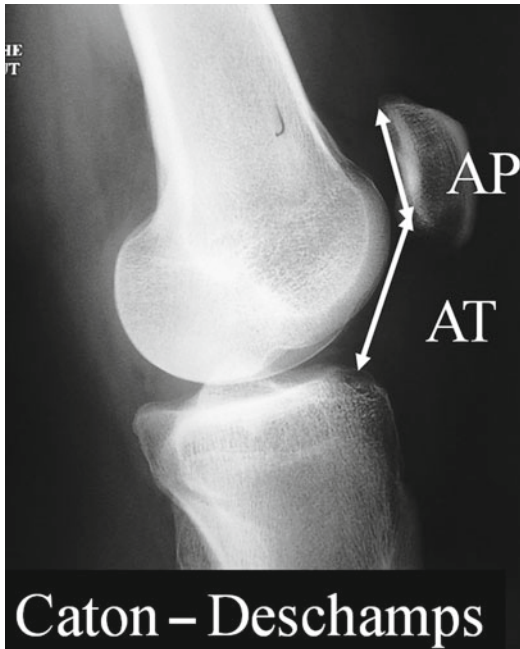


Fig. 36.1 The Caton–Deschamps index (AT/AP) is the ratio between the distance from the lower edge of the patella's articular surface to the anterosuperior angle of the tibia outline (AT), and the length of the articular surface of the patella (AP)

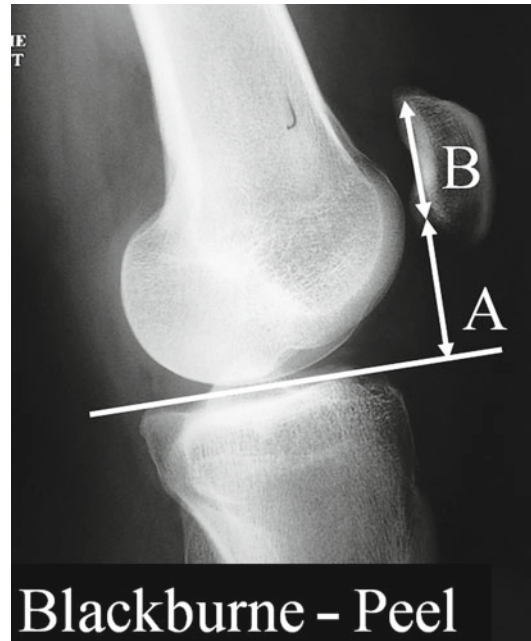


Fig. 36.3 The Blackburne–Peel index (A/B) is the ratio between the length of the perpendicular line drawn from the tangent to the tibial plateau until the inferior part of the articular surface of the patella (A) and the length of the articular surface of the patella (B)

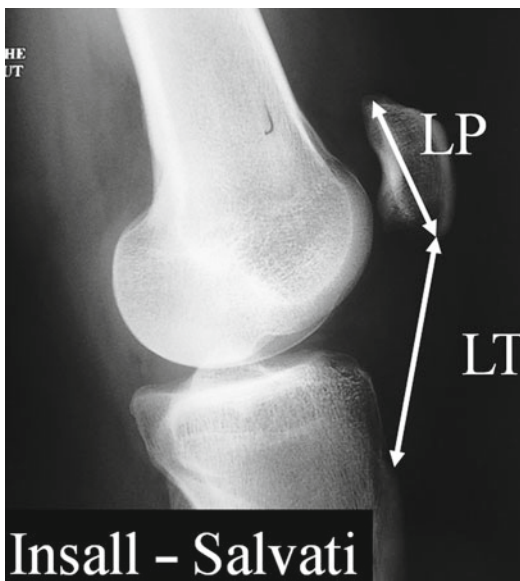


Fig. 36.2 The Insall–Salvati index (LT/LP) is the ratio between the length of the patellar tendon (LT) and the longest sagittal diameter of the patella (LP)

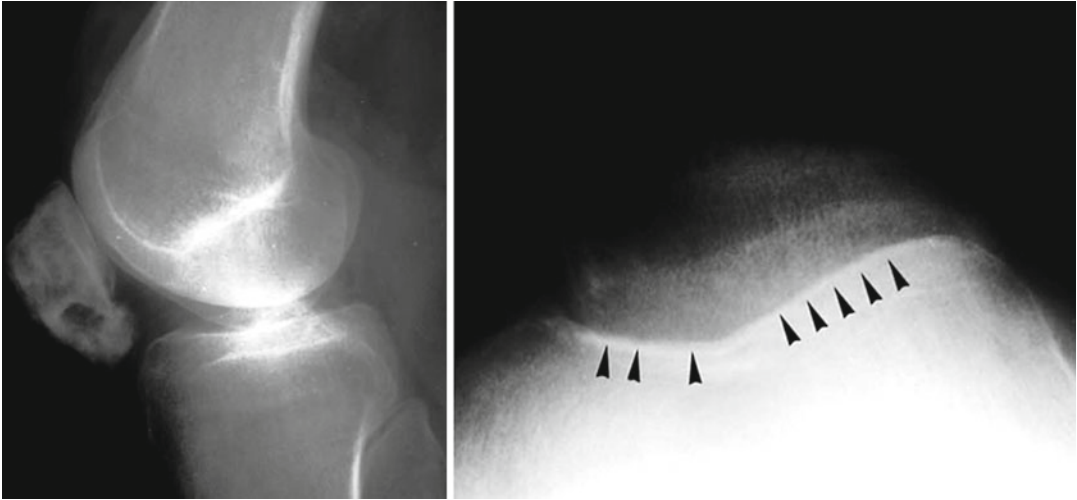


Fig. 36.4 Patella infera. On the lateral view, the distance from the lower edge of the patella’s articular surface to the anterosuperior angle of the tibia outline is almost zero. On

the axial view, the “sunset pattern” is shown as the articular space cannot be seen

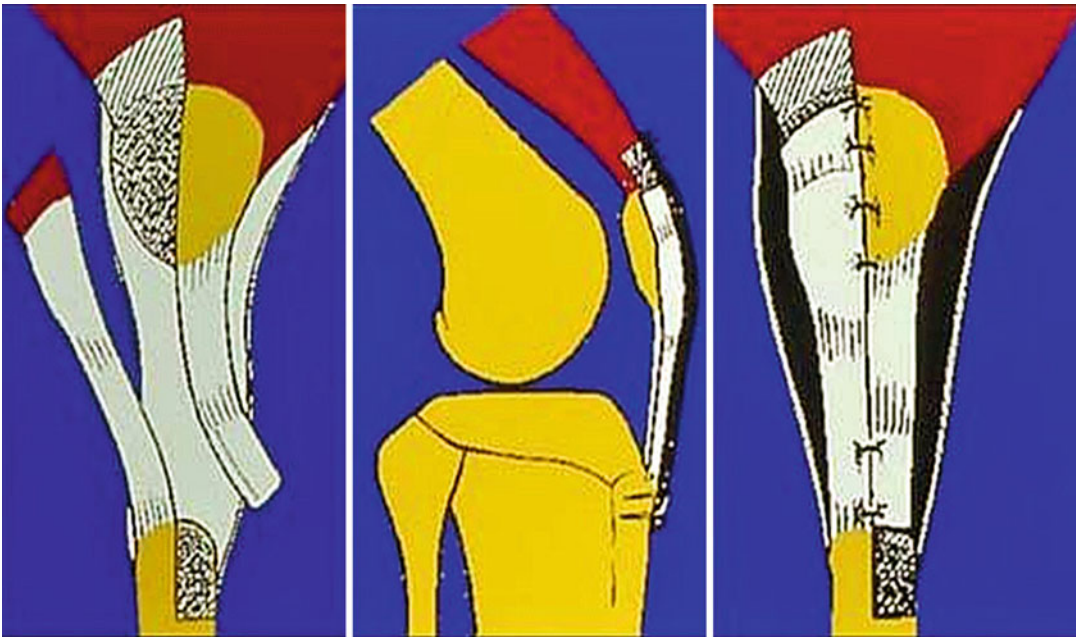


Fig. 36.5 Patellar tendon Lengthening (DeJour).The three steps: (1) Z plasty of the patellar tendon; (2) periop-

erative x-ray evaluation of the patellar height; (3) fixation with stitches protected with a metallic wire for 6 months

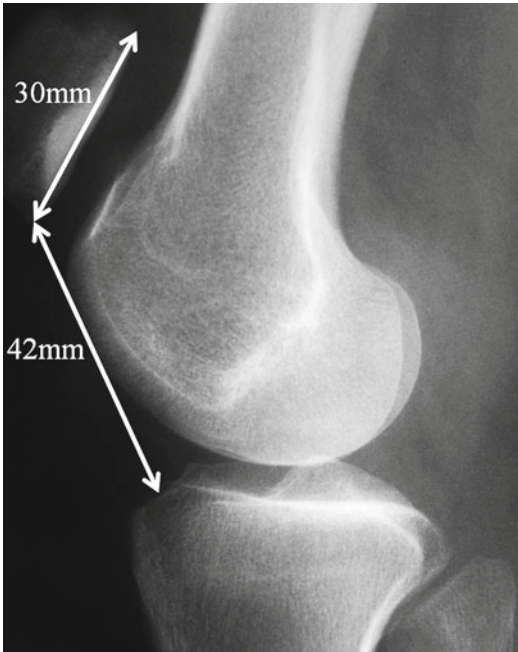


Fig. 36.6 Patella alta. The Caton–Deschamps index is 1.4

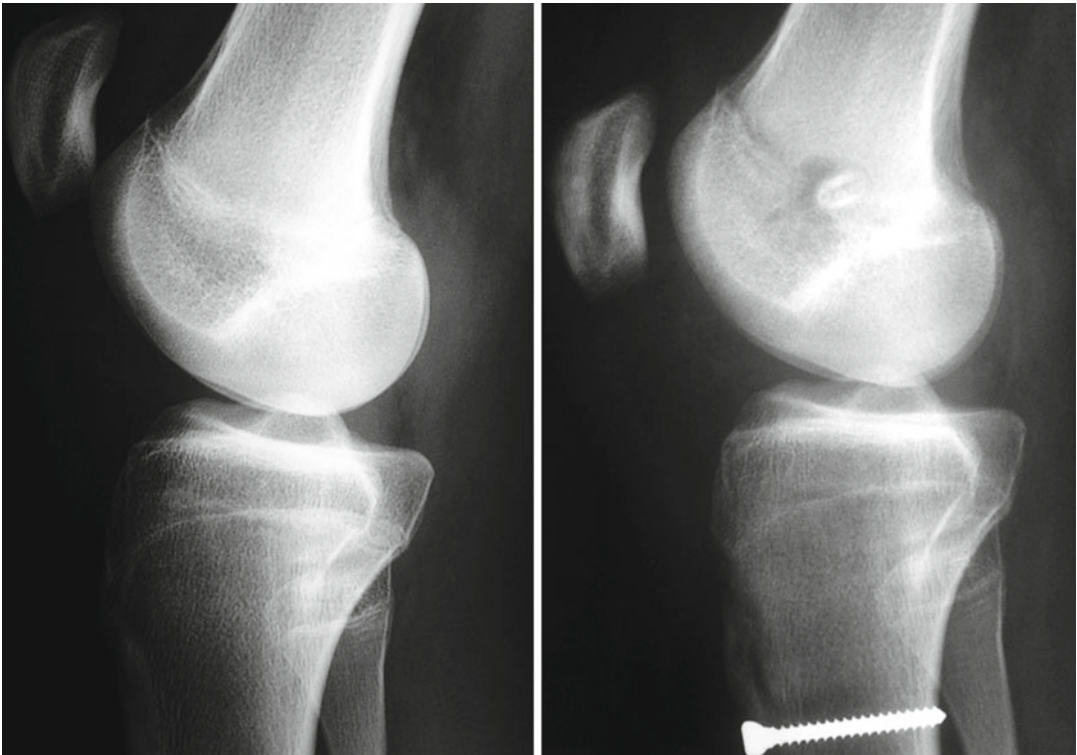


Fig. 36.7 Pre and postoperative lateral views of patella alta. After the distal tibial tubercle transfer, the patellar height is corrected (1.5 preoperatively to 1 postoperatively)

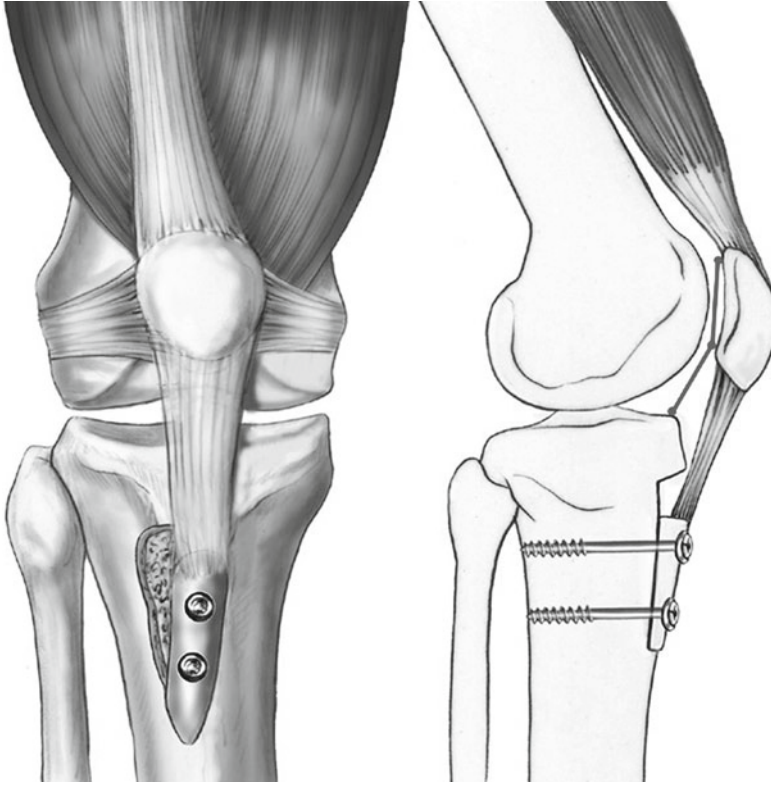


Fig. 36.8 Medial and distal tibial tubercle transfer. Two screws are used. This procedure is used to correct increased TT-TG distance and patella alta. Caution should be taken to not overmedialize the tubercle, since the

distalization procedure alone induces automatic medialization. Screws must be perpendicular to the tibial shaft to allow good compression and avoid proximal displacement during lagging

Anteromedial Tibial Tubercle Osteotomy (Fulkerson Osteotomy)

37

Jack Farr, Brian J. Cole, James Kercher,
Lachlan Batty, and Sarvottam Bajaj

Multiple case series have reported outcomes of the AMZ procedures. Despite the heterogeneity in outcome measurements, results demonstrate high percentages of excellent and good results and improvements in objective, subjective and functional measures. Attention to details related to surgical planning and properly

managing patient expectations is most likely to lead to good or excellent results. Newer techniques (i.e., the T3 system) allow the surgeon to objectively determine the inclination of the osteotomy to properly restore patellofemoral mechanics based upon the preoperative planning.

J. Farr, M.D. (✉)
Voluntary Clinical, Orthopaedic Surgery,
IU School of Medicine, OrthoIndy Knee Care Institute,
Cartilage Restoration Center of Indiana,
Indianapolis, IN, USA
e-mail: indyknee@hotmail.com

B.J. Cole, M.D., M.B.A.
Division of Sports Medicine,
Departments of Orthopaedics & Anatomy and
Cell Biology, Cartilage Restoration Center at Rush,
Rush University Medical Center,
Chicago, IL, USA

J. Kercher, M.D. • S. Bajaj, B.E.
Division of Sports Medicine,
Rush University Medical Center,
Chicago, IL, USA

L. Batty, M.B.B.S., B.MedSc.
The Alfred Hospital,
Melbourne, VIC, Australia



Fig. 37.1 Anterior compartment musculature is elevated from the lateral wall of the tibia with retractor protecting neurovascular structures posteriorly

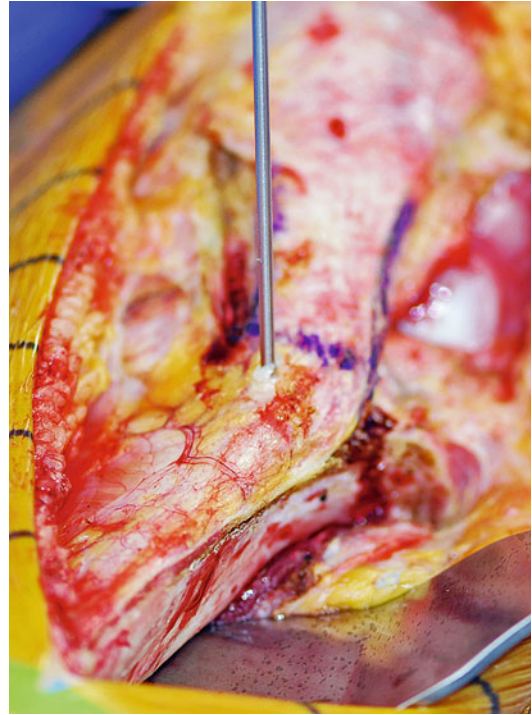


Fig. 37.3 Reference pin is inserted through the guide just distal to Gird's tubercle

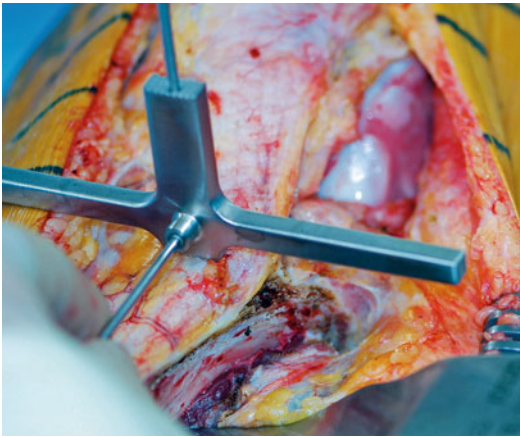


Fig. 37.2 The reference pin guide is orientated so it is perpendicular to the posterior cortex of the tibia

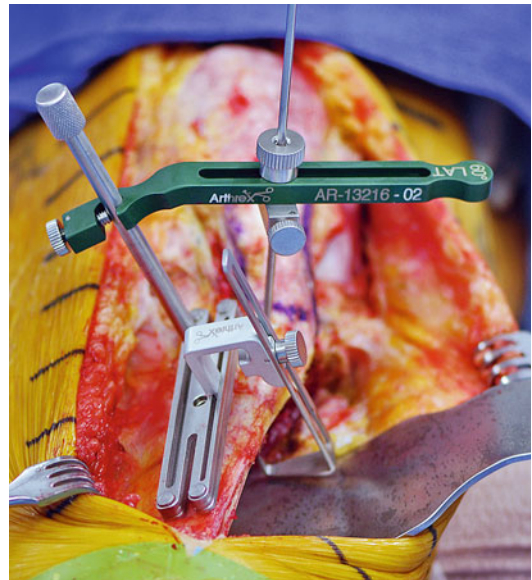


Fig. 37.4 The cutting guide is placed over the reference pin and the cutting block is positioned medial to the patella tendon

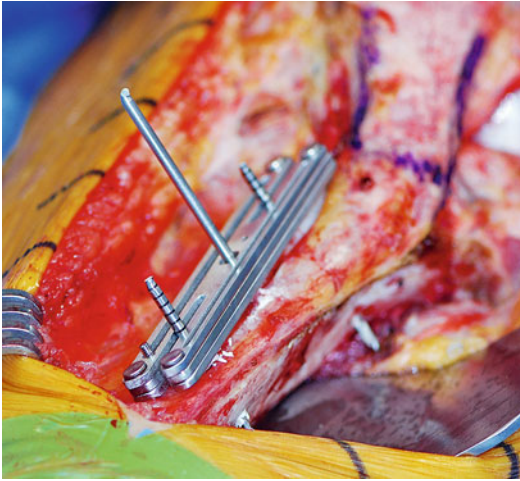


Fig. 37.5 Break-away pins secure the cutting block after positioning is confirmed



Fig. 37.6 Oscillating saw cooled with saline creates the initial sloped osteotomy, exiting on the protective retractor

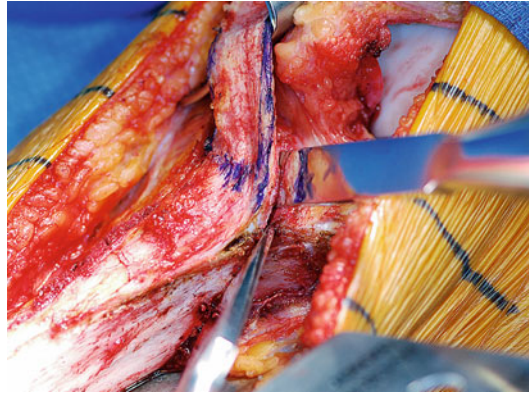


Fig. 37.7 Proximal cuts are completed with small osteotome

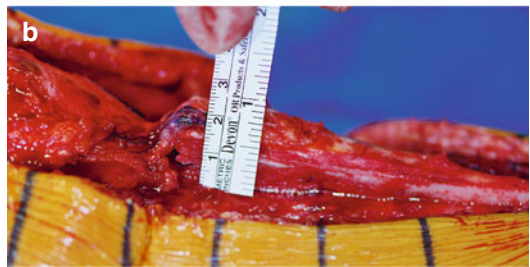
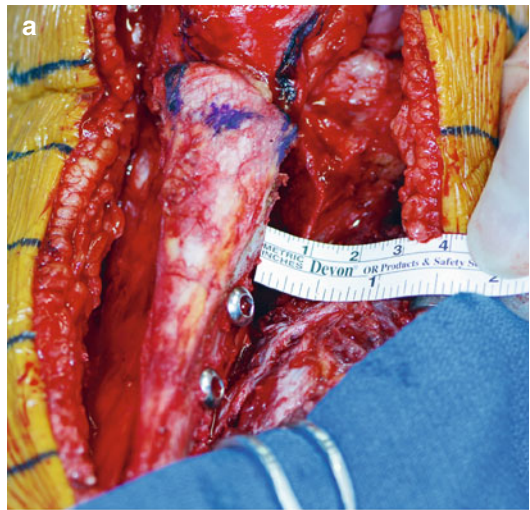


Fig. 37.8 The distance of medialization (a) and anteriorization (b) is measured directly and the pedicle is secured with 2–4.5 mm screws

The Patella Thinning Osteotomy: A New Technique for Patellofemoral Arthritis

38

Javier Vaquero and José Antonio Calvo

Patella thinning osteotomy is a conservative technique with good clinical and radiological results in our patients at a mean of 9 years, and a new option

for the treatment of isolated patellofemoral osteoarthritis. This osteotomy is a less aggressive alternative for dealing with a difficult clinical problem.

J. Vaquero, M.D., Ph.D. (✉)
Department of Orthopaedic Surgery,
Universidad Complutense de Madrid, Hospital General
Universitario Gregorio Marañón,
Madrid, Spain
e-mail: vaqueroct@aartroscopia.com

J.A. Calvo, M.D., Ph.D.
Department of Orthopaedic Surgery,
Hospital General Universitario Gregorio Marañón,
Madrid, Spain



Fig. 38.1 A 5 mm side cutting high-speed burr used to remove the central patellar bone

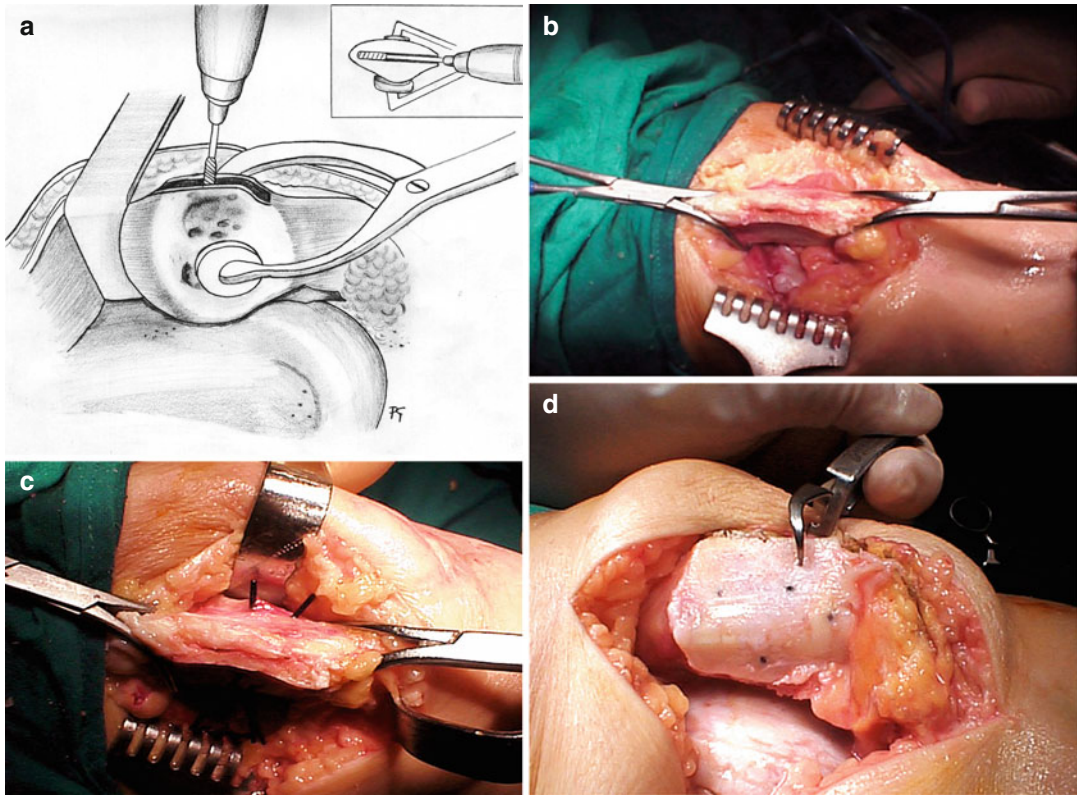


Fig. 38.2 (a) Patella thinning osteotomy: making the through in the lateral rim of the patella. Drawing of the technique that shows the direction of the burr strictly parallel to the anterior face of the patella. (b) Patella thinning osteotomy: collapse of the central portion obtaining a thinner patella. (c) Patella thinning osteotomy: fixation of fragments with biodegradable pins. Note the osteotomy line. (d) Patella thinning osteotomy: final result with a

congruent joint surface and preservation of the soft tissues attachments in the proximal and distal poles of the articular fragment (Reproduced and adapted with permission and copyright of the British Editorial Society of Bone and Joint Surgery from Vaquero J, Calvo JA, Chana F, Perez-Mañanes R. The patellar thinning osteotomy in patellofemoral arthritis: four to 18 years follow-up. *J Bone Joint Surg Br.* 2010;92-B:1385–91)

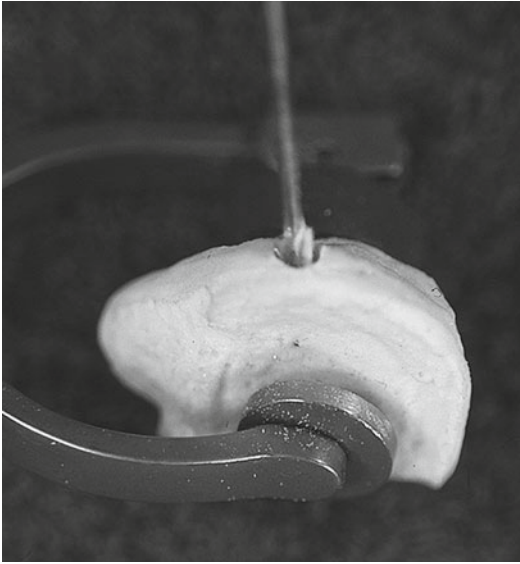


Fig. 38.3 Pilot hole in saw-bone that determines the correct plane for the osteotomy

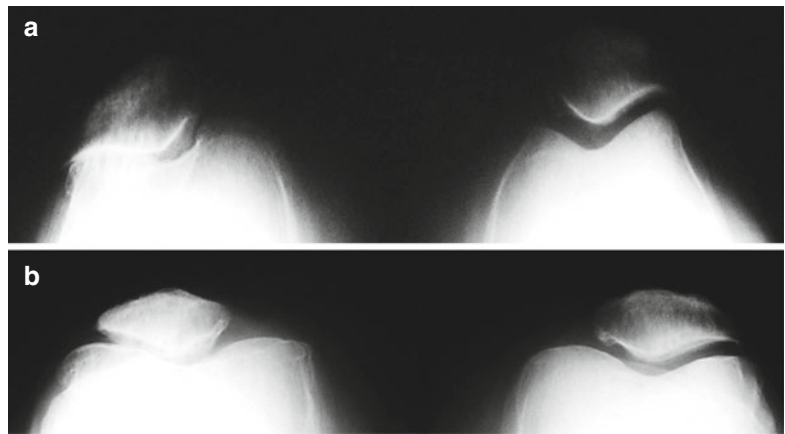


Fig. 38.4 Skyline view preoperative (a) and 5 years (b) after a thinning osteotomy on the right side. Note the preservation of the joint line over time



Fig. 38.5 Total knee arthroplasty implanted without technical complications due to femorotibial osteoarthritis progression 5 years after a patella thinning osteotomy. Lateral view (Reproduced and adapted with permission and copyright of the British Editorial Society of Bone and Joint Surgery from Vaquero J, Calvo JA, Chana F, Perez-Mañanes R. The patellar thinning osteotomy in patellofemoral arthritis: four to 18 years follow-up. *J Bone Joint Surg Br.* 2010;92-B:1385–91)

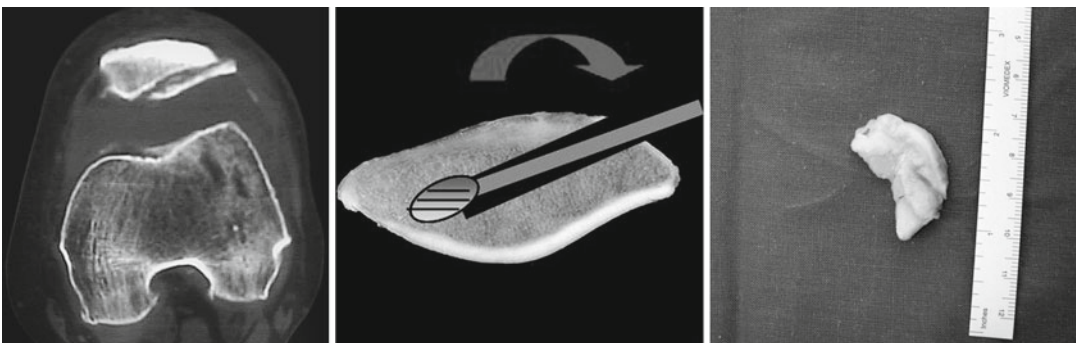


Fig. 38.6 Skyline view and drawing that shows an oblique osteotomy too close to the articular surface. The necrotic fragment had to be removed arthroscopically 1 year later

Cartilage Restoration in the Patellofemoral Joint

39

Jack Farr, Brian J. Cole, Michael J. Salata,
Marco Collarile, and Sarvottam Bajaj

Management of patients with a PF joint that presents with pain and dysfunction associated with chondral pathology remains a difficult clinical problem. Attention to the entire PF system including issues pertaining to alignment is paramount in achieving a successful outcome. Similar to the

tibiofemoral joint, all comorbidities must be addressed. Multiple options exist to manage the chondral pathology and are chosen based upon defect size and location. Outcomes support good and excellent results similar to that seen in with the management of the tibiofemoral joint.

J. Farr, M.D. (✉)

Voluntary Clinical, Orthopaedic Surgery, IU School of
Medicine, OrthoIndy Knee Care Institute,
Cartilage Restoration Center of Indiana,
Indianapolis, IN, USA
e-mail: indyknee@hotmail.com

B.J. Cole, M.D., M.B.A.

Division of Sports Medicine, Departments of
Orthopaedics & Anatomy and Cell Biology,
Cartilage Restoration Center at Rush,
Rush University Medical Center,
Chicago, IL, USA

M.J. Salata, M.D. • S. Bajaj, B.E.

Division of Sports Medicine,
Rush University Medical Center,
Chicago, IL, USA

M. Collarile, M.D.

Orthopaedic Department and Knee Surgery Center,
Sacro Cuore Don Calabria Hospital,
Negrar, Verona, Italy

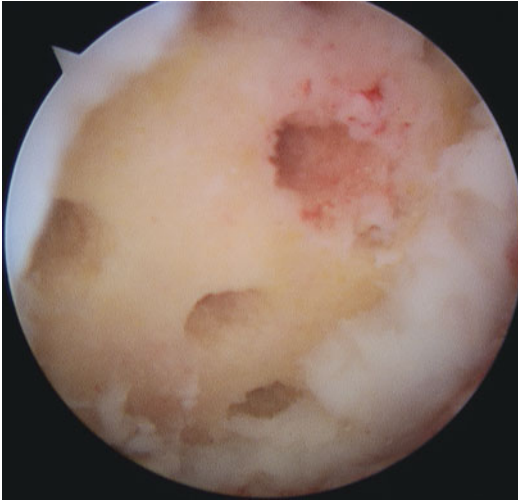


Fig. 39.1 Marrow stimulation of trochlea

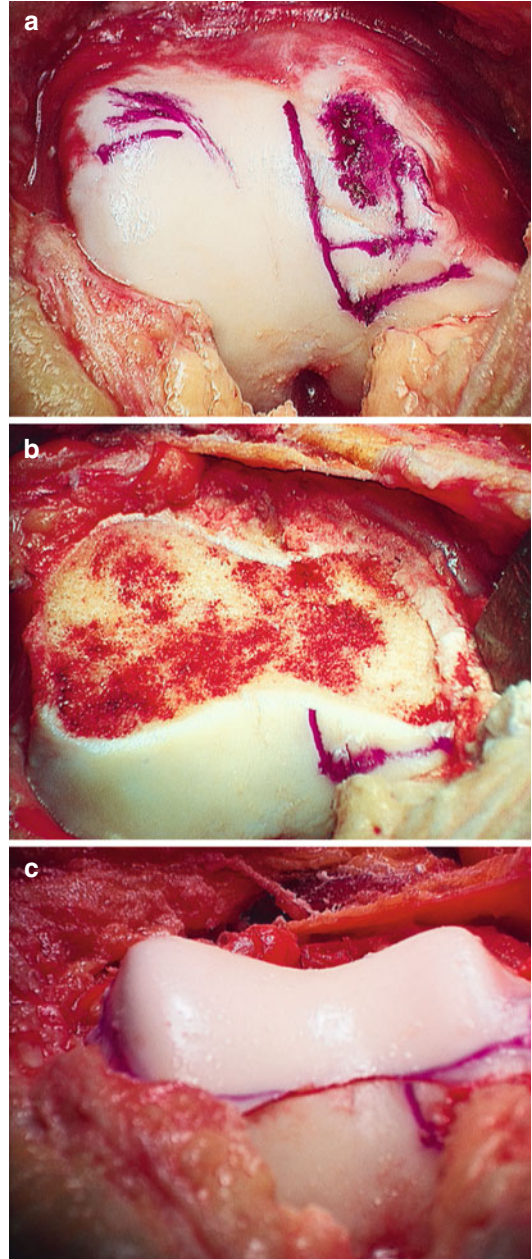


Fig. 39.3 The area of chondrosis is identified (a). The bony cuts are made with a cooled oscillating saw (b). The donor graft is shaped to fit the defect and secured (c)

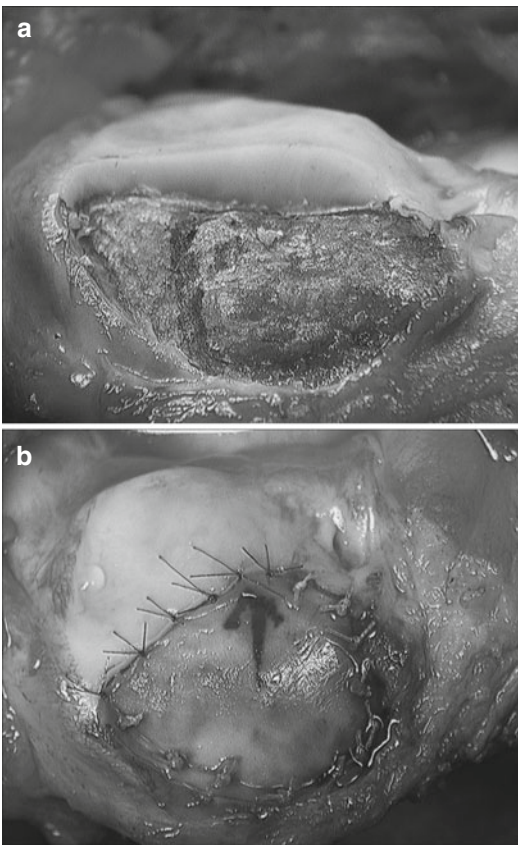


Fig. 39.2 (a) The defect is cleared without penetrating the subchondral bone. Walls are vertical. (b) The periosteal patch or collagen patch is secured with suture and sealed with fibrin glue. The suspension of cultured chondrocytes is injected deep to the patch

László Hangody and Eszter Baló

Nowadays one of the most frequently used methods to treat focal full thickness chondral or osteochondral defects on the weight bearing area is autologous osteochondral mosaicplasty. The main point of this method is to harvest small, different sized osteochondral grafts from the less weight bearing area of the knee and to transplant them to the defect of the weight bearing surface. Later gaps between transplanted plugs are filled with fibrocartilage. The new composite cartilage surface is consisted of about 80–90 % transplanted hyaline cartilage and 10–20 % regenerative fibrocartilage

with same biomechanical characteristics than the healthy hyaline surface has. This chapter reports about indications and contraindications of mosaicplasty particularly among patellofemoral diseases. Surgical procedure step by step and postoperative management are also detailed. Complications and their treatments are described. The author's clinical experiences with mosaicplasty are also summarized during 17 years follow up.

Some pearls and pitfalls are also presented that the author met in his clinical practice using mosaicplasty.

L. Hangody, M.D., Ph.D., DSc (✉)
Department of Orthopaedics, Uzsoki Hospital,
Budapest, Hungary
e-mail: hangody@t-online.hu

E. Baló, M.D.
Orthopaedic & Trauma Department, Uzsoki Hospital,
Budapest, Hungary



Fig. 40.1 Open mosaicplasty on the patella – filling by ten grafts of same size



Fig. 40.3 Open mosaicplasty on the trochlea – graft harvest from the periphery and filling of a central defect



Fig. 40.2 Open mosaicplasty on the patella – filling by nine grafts of different size

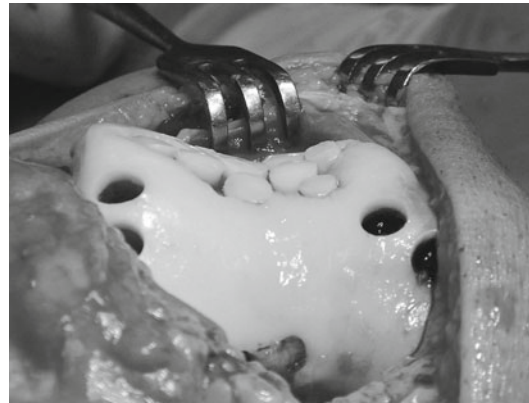


Fig. 40.4 Open mosaicplasty on the trochlea – perfect congruency according to axial view

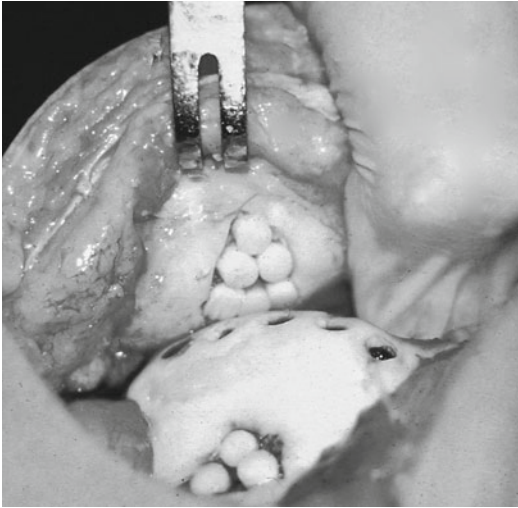


Fig. 40.5 Mosaicplasty for kissing lesions of patellofemoral joint – exceptional indication



Fig. 40.8 Magnetic resonance image of mosaicplasty donor sites – minimum 3 mm should be kept between two donor tunnels

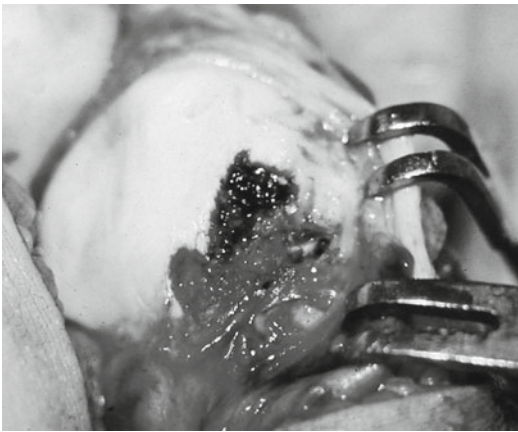


Fig. 40.6 Fresh traumatic osteochondral lesion of the patella

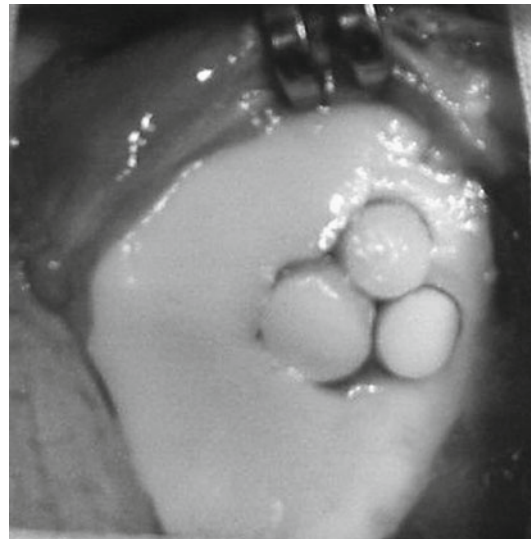


Fig. 40.9 Mosaicplasty by three grafts in the centre of the patella

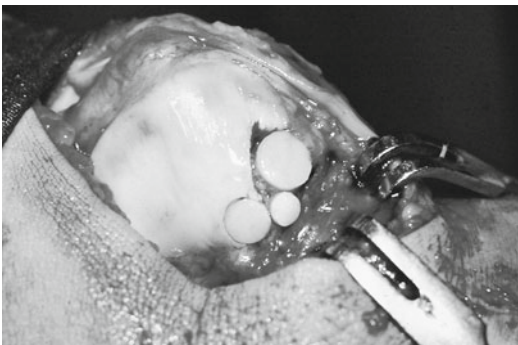


Fig. 40.7 Filling of the previous defect by three grafts

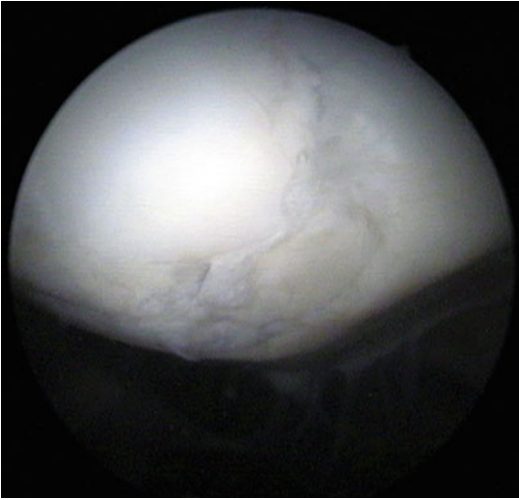


Fig. 40.10 Two-year-old control arthroscopy of the previous implantation – congruent hyaline-like coverage of the defect

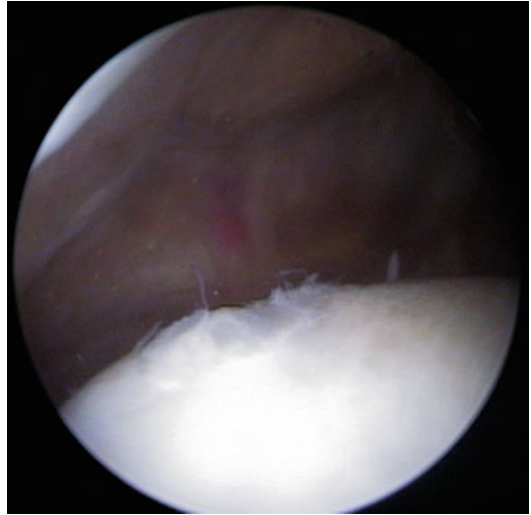


Fig. 40.11 Two-year-old control arthroscopy of a mosaicplasty donor site – congruent fibrocartilage coverage of the donor tunnel

Robert A. Teitge and Roger Torga-Spak

If the articular cartilage has been lost and osteoarthritis develops, two alternatives are available: (1) restoration of the normal extra-articular anatomy and stability, and (2) replacement of the articular cartilage. Options for articular cartilage replacement are biologic or prosthetic.

Osteochondral allografts have a long clinical history and have demonstrated >75 % clinical success in the treatment of knee joint defects. The procedure consists of transplanting a shell of subchondral bone and mature articular cartilage into the damaged area of the joint. The transplant can be either unipolar

(only one surface is transplanted) or bipolar (if two reciprocal articulating surfaces are transplanted). Chondrocyte viability is vital to the success of cartilage transplantation. Because chondrocyte survival is diminished after freezing, and as time elapses, fresh allografts improve the prospects of preserving chondrocyte viability. Rejection is less of a concern for articular cartilage transplantation than for other tissues because the chondrocyte surface cell antigens are isolated from the immunologic cells of the host by the complex high-molecular-weight matrix that surrounds the lacunae.

R.A. Teitge, M.D.
Department of Orthopaedics, Wayne State University
School of Medicine,
Detroit, MI, USA

R. Torga-Spak, M.D. (✉)
Department of Surgery, Faculty of Orthopaedics and
Traumatology, Instituto Universitario CEMIC,
Buenos Aires, Argentina
e-mail: rtorgaspak@gmail.com

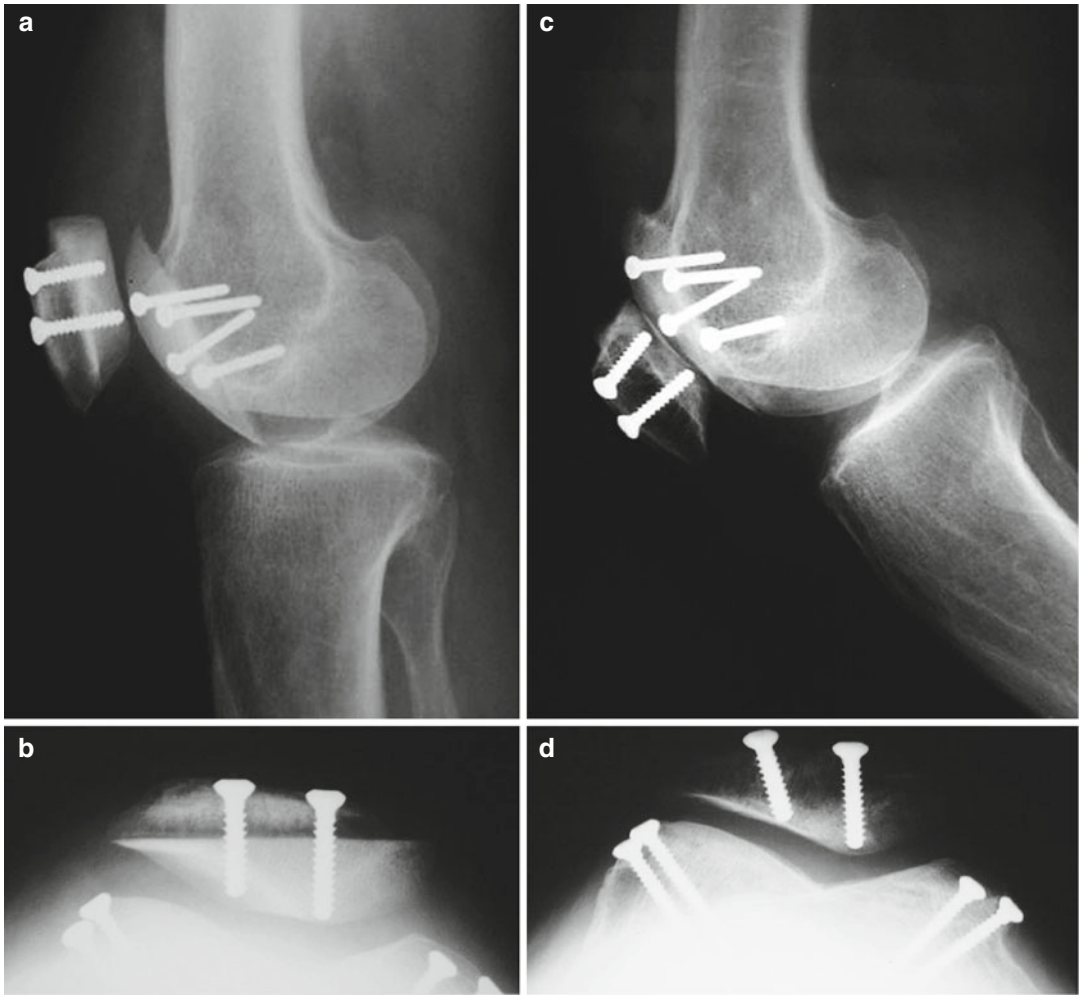


Fig. 41.1 (a) Immediate postoperative lateral view after bipolar transplant. (b) Immediate postoperative axial view after bipolar transplant. (c) Lateral view after 5 years of bipolar patellofemoral transplant. (d) Axial view after 5 years of bipolar patellofemoral transplant

Patellofemoral Arthroplasty: Pearls and Pitfalls

42

Ronald P. Grelsamer and Jason Gould

Patellofemoral Replacement surgery, popular in Europe for the last 25 years, is gaining popularity in the United States. This is reflected in the proliferation of patellofemoral implants.

Nevertheless, it remains a niche procedure that is best indicated for patients who are unlikely to develop arthritis in their femoro-tibial compartments. The least satisfying results have been obtained in patients whose arthritis is of unknown etiology and who are young enough to develop femoro-tibial arthritis.

On the other hand, the procedure requires far less surgical dissection than a standard total knee replacement and leads to far less blood loss. It can appeal to patients who would otherwise refuse knee replacement surgery.

The key technical points are: not placing the trochlear component too far distally, not leaving either the proximal or distal portion of the trochlea proud, and not leaving the patella unstable.

R.P. Grelsamer, M.D. (✉)
Patellofemoral Reconstruction,
Mount Sinai Medical Center,
New York, NY, USA
e-mail: ronald.grelsamer@mountsinai.org

J. Gould, M.D.
Department of Orthopedic Surgery,
Mount Sinai Medical Center,
New York, NY, USA

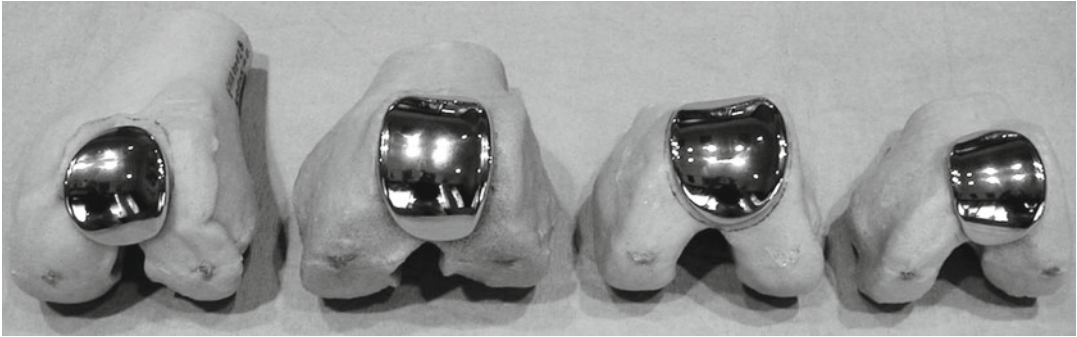


Fig. 42.1 There exist dramatic variations in trochlear anatomy. This is particularly true in candidates for patellofemoral replacement surgery (Reprinted with permission from Kinamed, California USA)

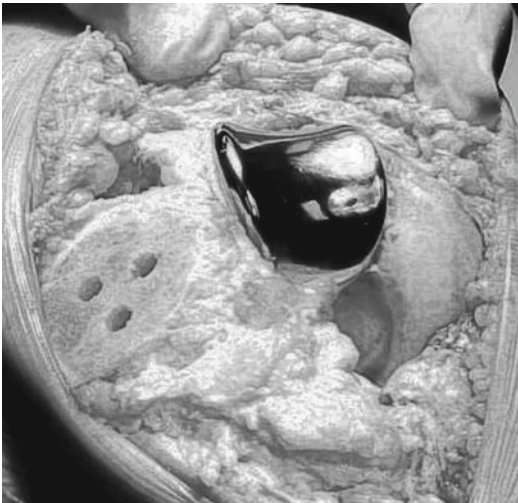


Fig. 42.2 Custom trochlear component manufactured from a CT scan of the patient's trochlea (Reprinted with permission from Kinamed, California USA)

The Patellofemoral Arthroplasty: The Great Solution, the Great Problem

43

Vicente Sanchis-Alfonso

Unfortunately, many orthopedic surgeons believe that the definite solution for eliminating anterior knee pain in a young patient with multiple operations is the patellofemoral prosthesis (“The Great Solution”). But, they fail to ask themselves the

most important question; what is causing the pain? When a patellofemoral prosthesis is implanted in these young patients without finding out the cause of the pain, a more serious problem could be created with a worse solution (“The Great Problem”).

V. Sanchis-Alfonso, M.D., Ph.D.
International Patellofemoral Study Group,
ACL Study Group, Hospital 9 de Octubre, Hospital
Arnau de Vilanova, School of Medicine,
Valencia Catholic University,
Valencia, Spain
e-mail: vicente.sanchis.alfonso@gmail.com



Fig. 43.1 Physical examination shows a patella alta

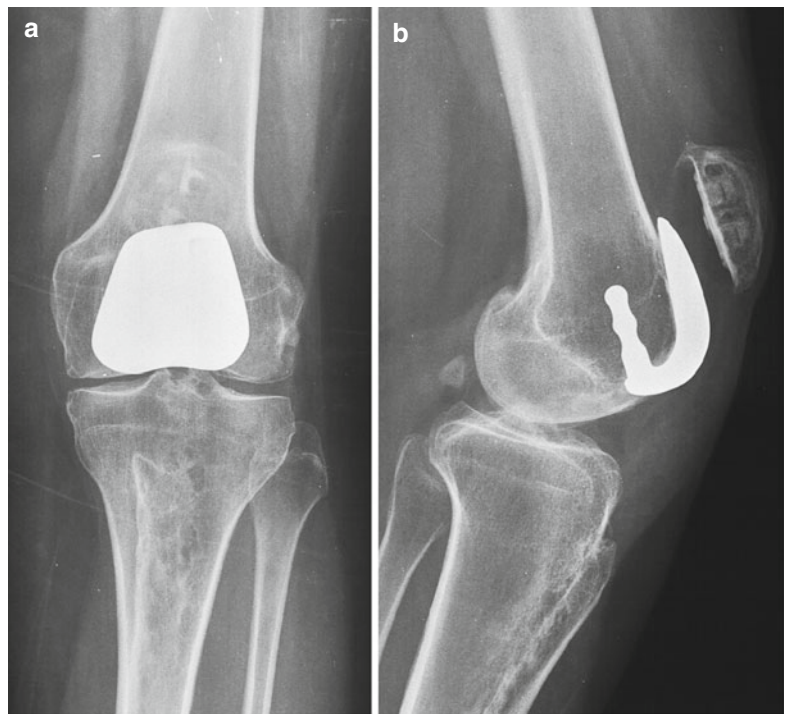


Fig. 43.2 Anteroposterior (a) and lateral (b) radiographs of the left knee showing a patella alta and a patellofemoral arthroplasty

Fig. 43.3 Standard technetium 99 methylene diphosphonate bone scan showing increased osseous metabolic activity in femoral condyles

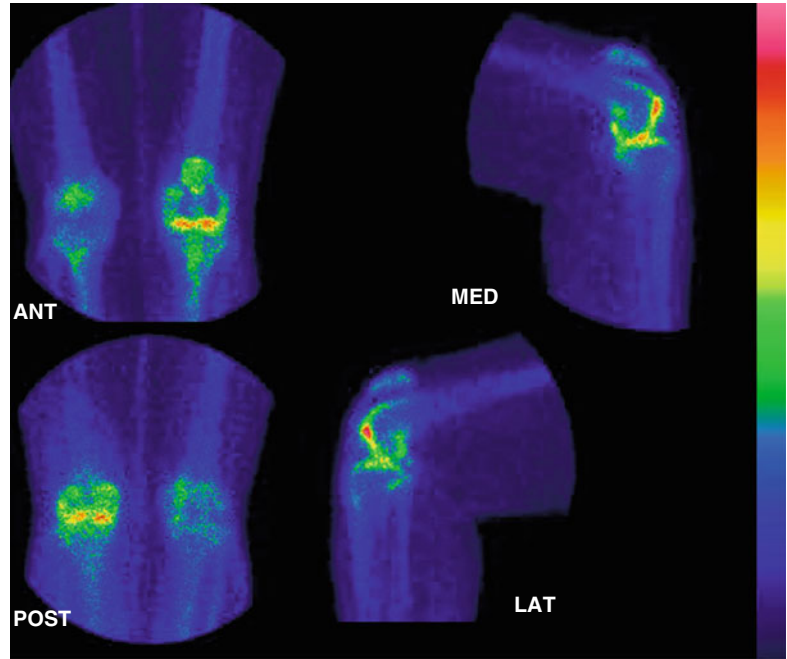
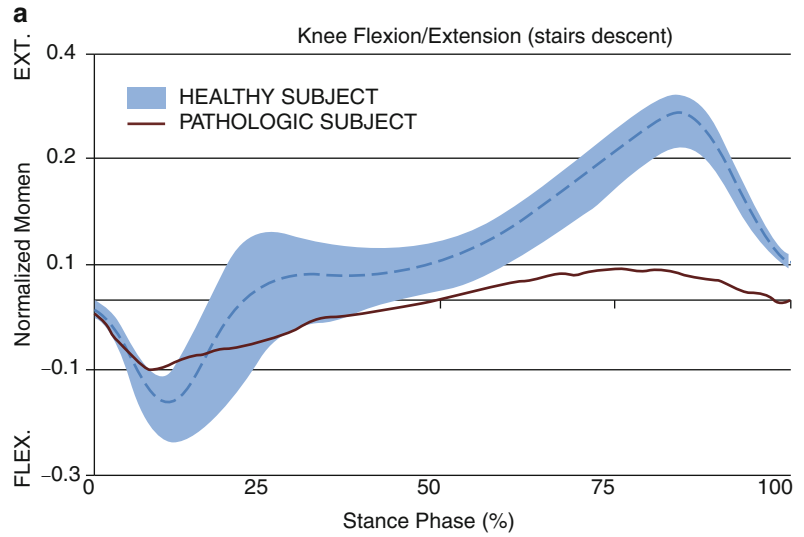


Fig. 43.4 (a) Knee flexion–extension moment during stair descending test. (b) Vertical ground reaction force during stair descending test. (c) Knee abduction moment during stair descending test



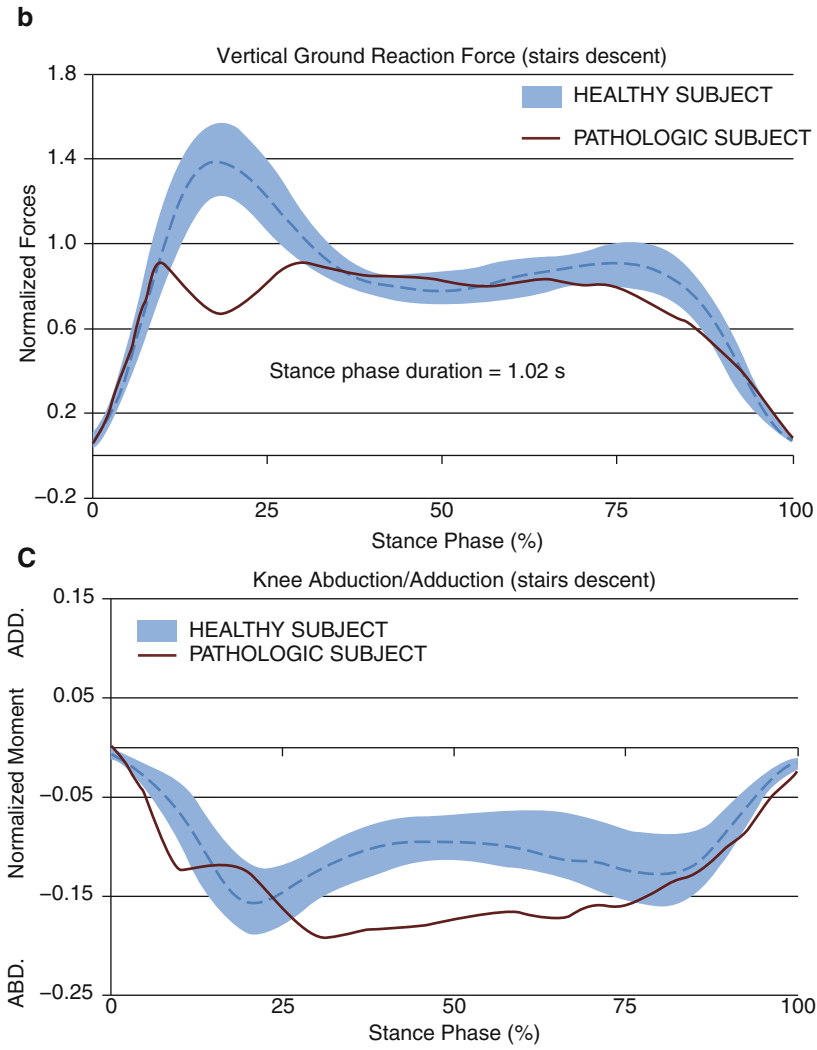


Fig. 43.4 (continued)

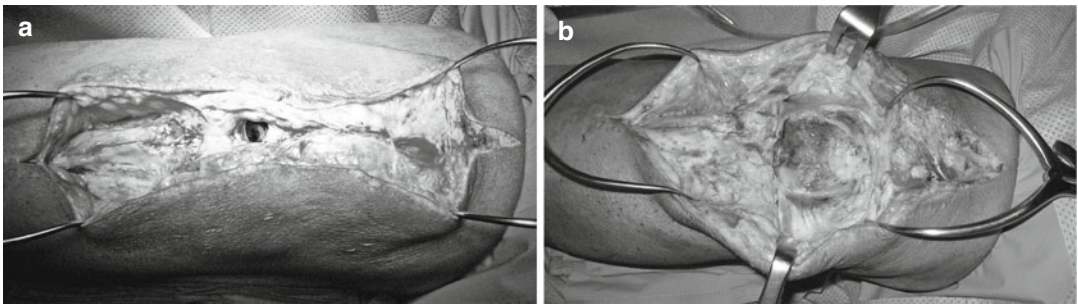


Fig. 43.5 (a) Intraoperative photograph showing a complete absence of the patellar tendon. (b) Femoral trochlea after removing the femoral shield of the patellofemoral prosthesis

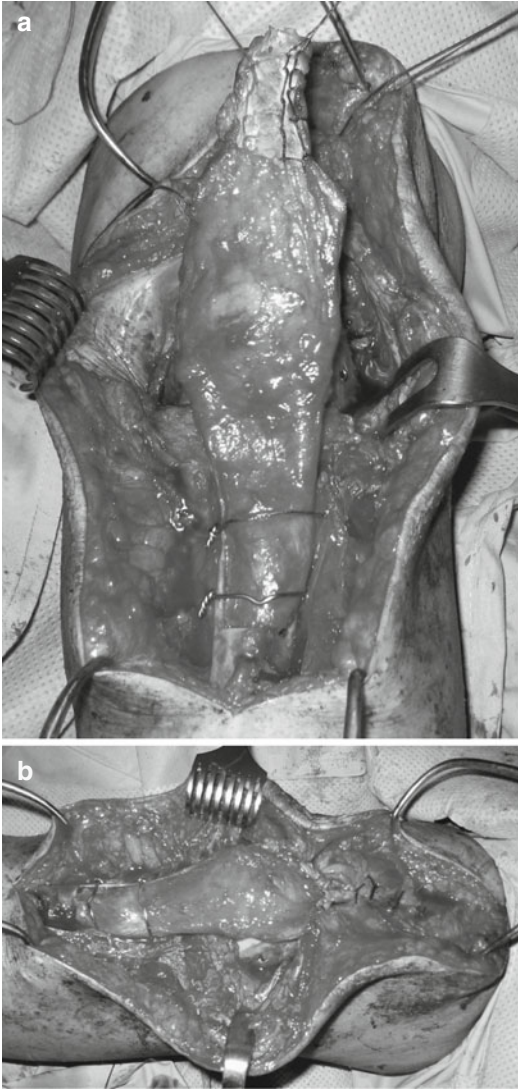


Fig. 43.6 (a) Intraoperative photograph of the allograft extensor mechanism in situ. Fixation of the tibial allograft with stainless steel wires. Two sutures placed in the allograft quadriceps allow the allograft to be tensioned proximally. (b) Good coverage of the allograft with autologous soft tissue

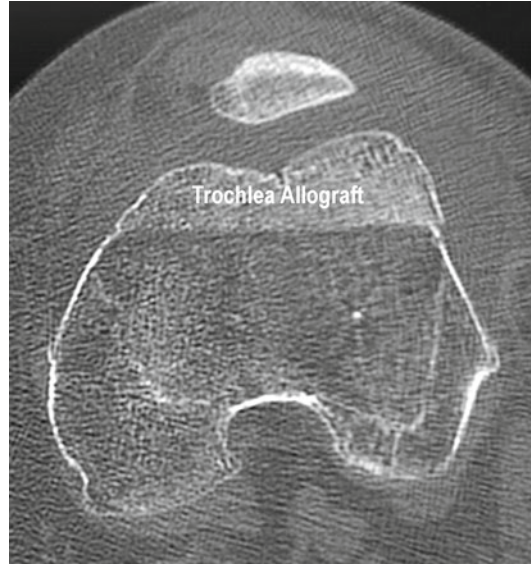


Fig. 43.7 Postoperative CT at 3 months follow-up. Femoral trochlea allograft without degenerative changes or a trochlear dysplasia (FTA)



Fig. 43.8 Extensive posttraumatic ossification of the patellar tendon

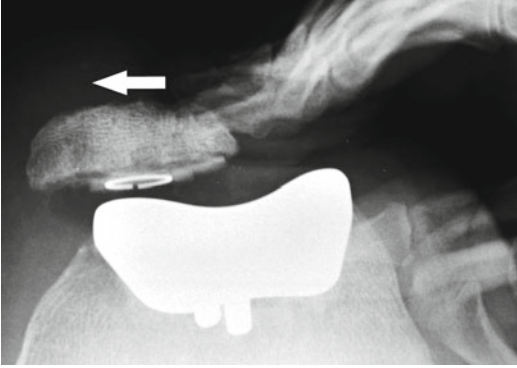


Fig. 43.9 Axial stress radiograph of the left knee showing an iatrogenic medial subluxation of the patella

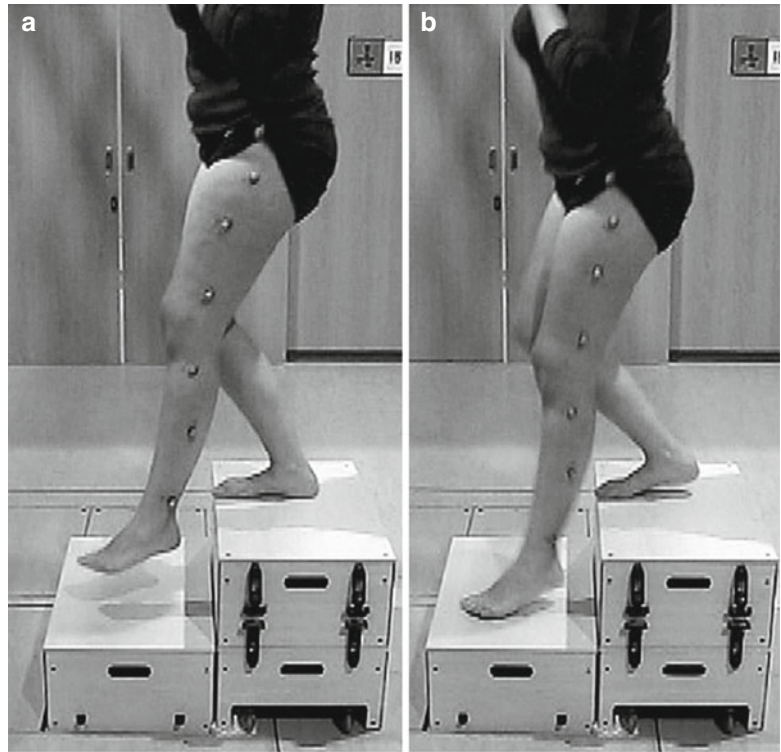


Fig. 43.10 Knee joint angle during stair descent (Knee extension pattern). One factor that could contribute to the knee extensor moment reduction is the decrease of knee flexion angle during the stance phase of stair ambulation. It would be a strategy to reduce the extensor moment and therefore pain during stair descent. We speculate that the knee extension pattern during stair descent is a strategy to avoid instability and therefore pain. Due to this knee extension pattern, the posterior muscles work in a chronic manner in an elongated eccentric condition; this situation could be responsible for the posterior knee pain in our patient

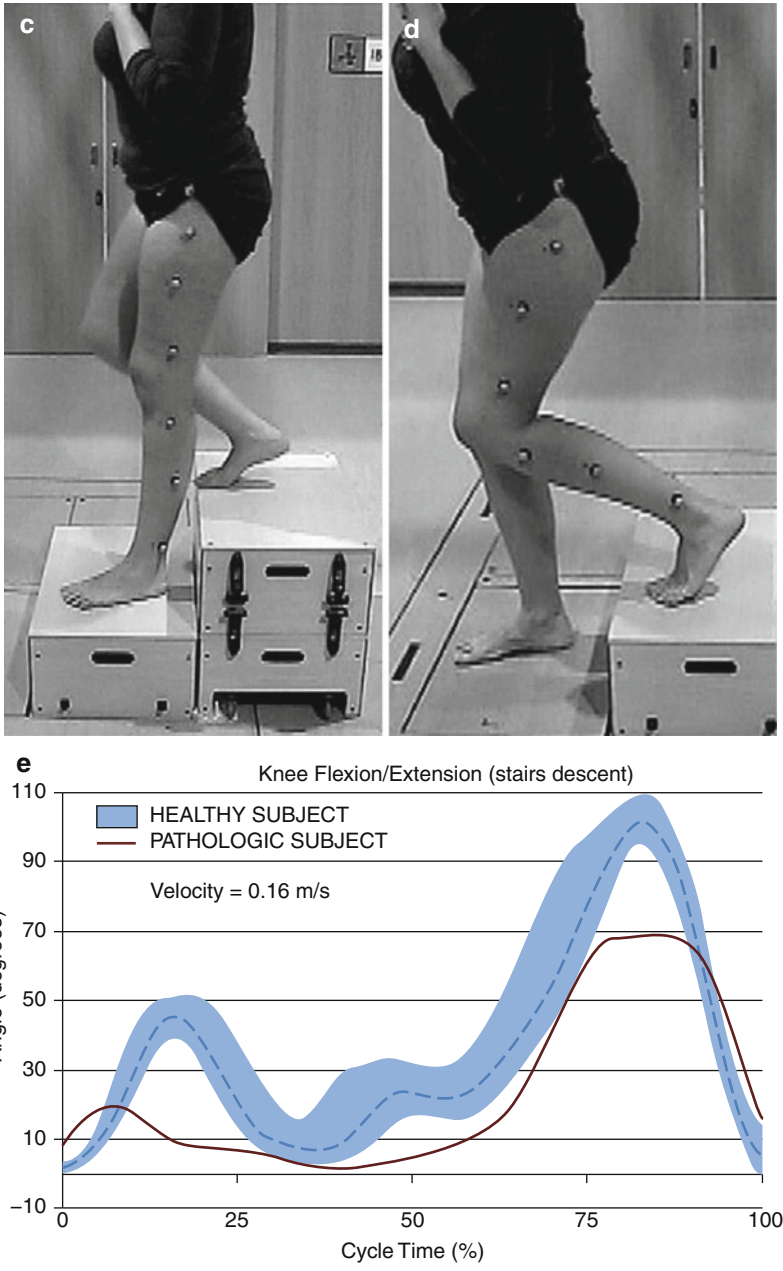


Fig. 43.10 (continued)

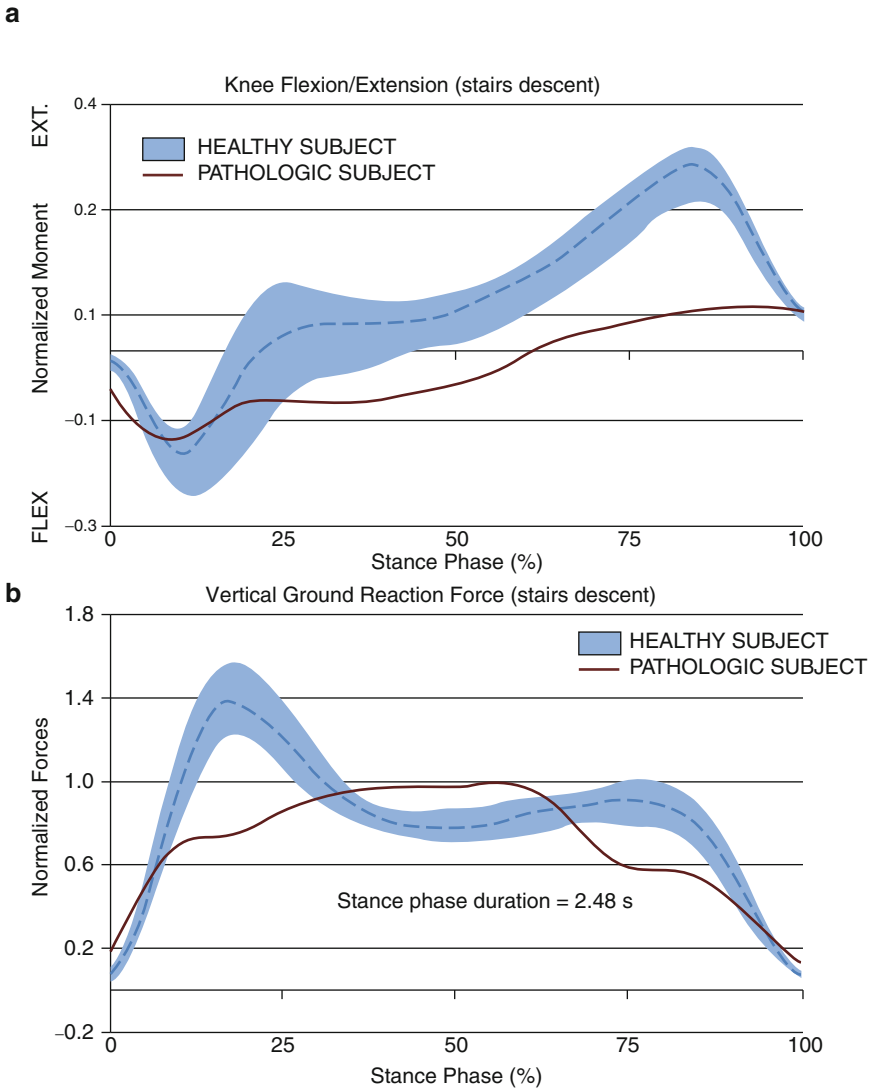
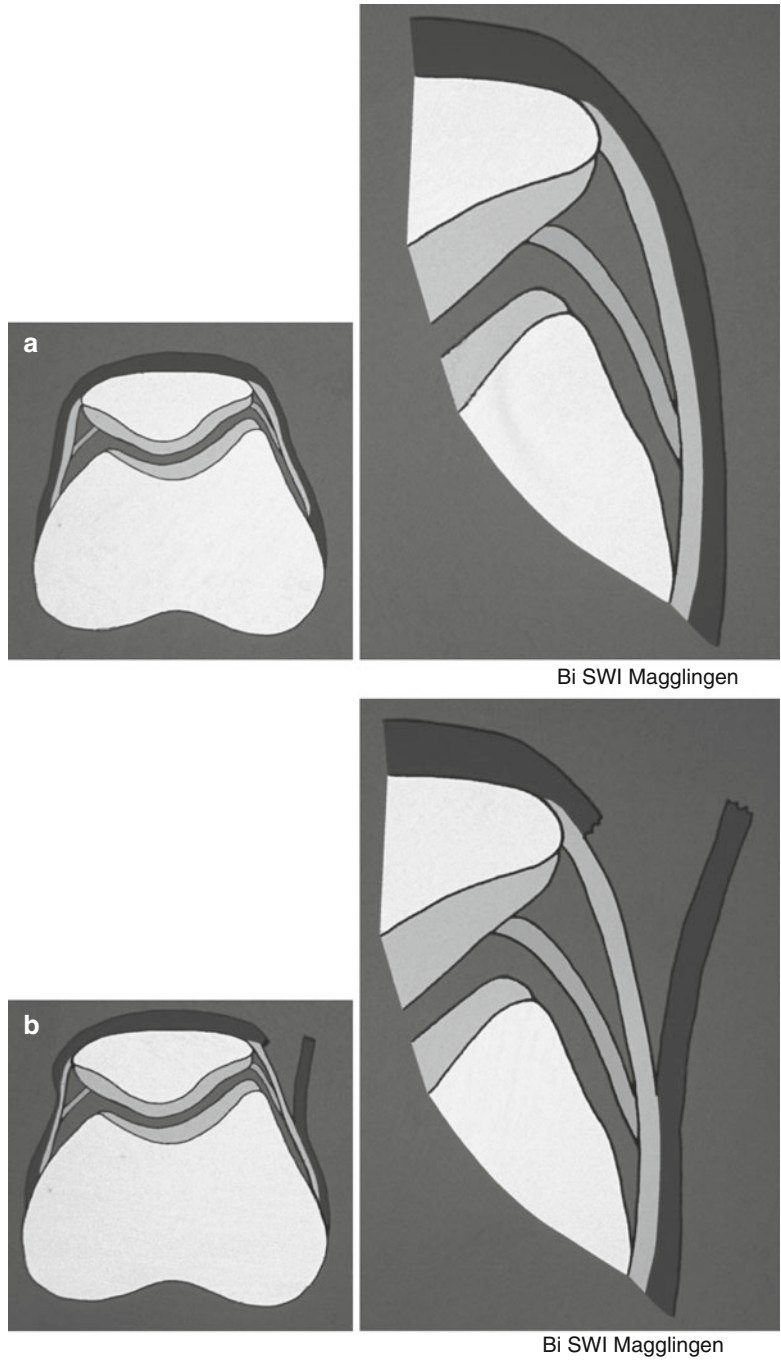
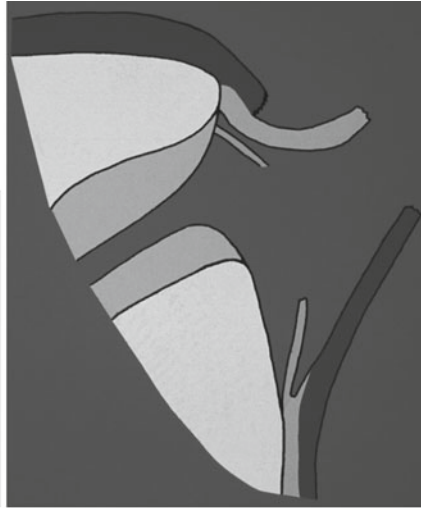
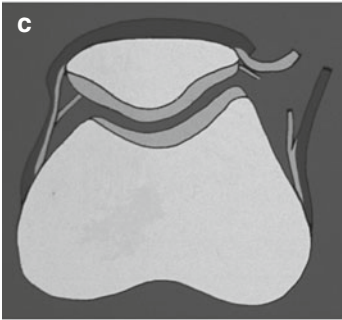


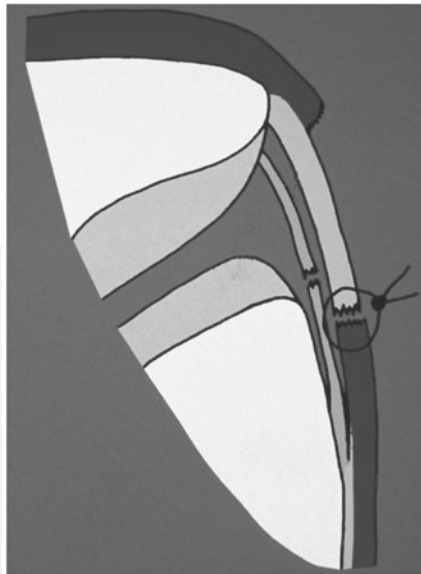
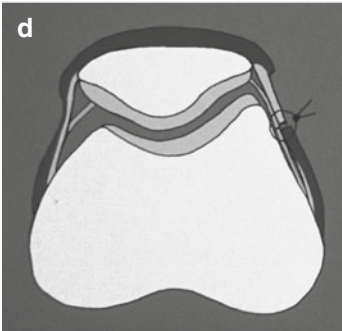
Fig. 43.11 (a) Flexion–extension knee moments during stair descent. (b) Ground force reactions during stair descent

Fig. 43.12 Schematic diagram showing the lengthening of the lateral retinaculum (Technical note according to Roland M. Biedert)





Bi SWI Magglingen



Bi SWI Magglingen

Fig. 43.12 (continued)

A Philosophy of the Patellofemoral Joint: A Logical Clinical Approach

44

Alan C. Merchant

A philosophy or pathomechanical and pathophysiological theory of the patellofemoral joint is described which leads to a logical etiology-based clinical classification, provides a framework for measurement and analysis of the six anatomic abnormalities that affect the normal function of the patellofemoral joint, and presents a simple

and logical approach to treatment, both operative and non-operative. This theory, based on the careful clinical measurement of these six potential abnormalities, is also able to explain the two major symptoms of patellofemoral disorders, anterior knee pain and patellar instability, using available scientific data.

A.C. Merchant, M.D.
Department of Orthopedic Surgery,
Stanford University School of Medicine,
Stanford, CA, USA

Department of Orthopedic Surgery,
El Camino Hospital,
Mountain View, CA, USA
e-mail: kneemd@sbcglobal.net

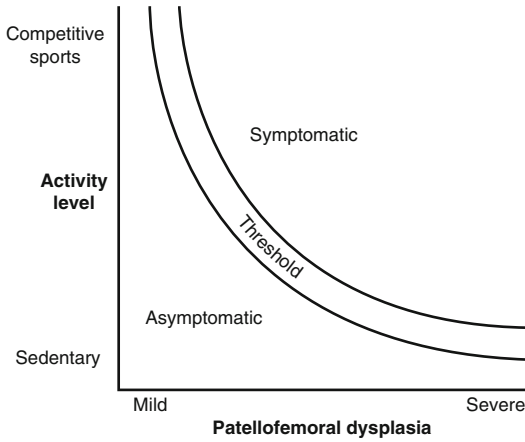


Fig. 44.1 This diagram shows the inverse relationship between severity of patellofemoral dysplasia and activity level, which determines when any given patient will cross the symptomatic threshold (From Merchant AC. Patellofemoral disorders: biomechanics, diagnosis, and non-operative treatment. In: McGinty JB, Casperi RB, Jackson RW, Poehling GG, editors. Operative arthroscopy. New York: Raven; 1991. With permission)

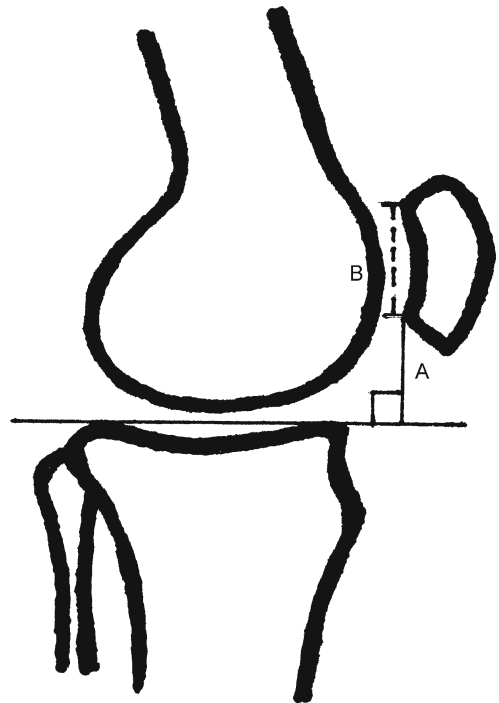


Fig. 44.3 Blackburn–Peel technique for measuring patellar height ratio. *A* vertical distance above the tibial plateau. *B* patellar articular height. *A/B* patellar height ratio. Normal mean ratio = 1:1, ± 0.2



Fig. 44.2 With the quadriceps contracted holding the knee at 30° flexion and with the foot unsupported, a deficient vastus medialis obliquus will leave a characteristic hollow on the medial aspect of the knee

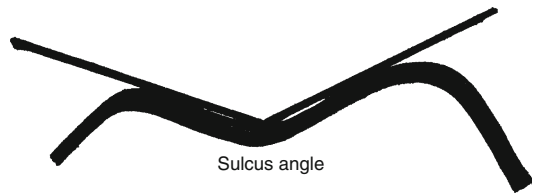


Fig. 44.4 Sulcus angle measurement from an accurate axial view radiograph with the knee flexed 45°. In 100 normal subjects, the sulcus angle mean = 138°, range = 126–150°

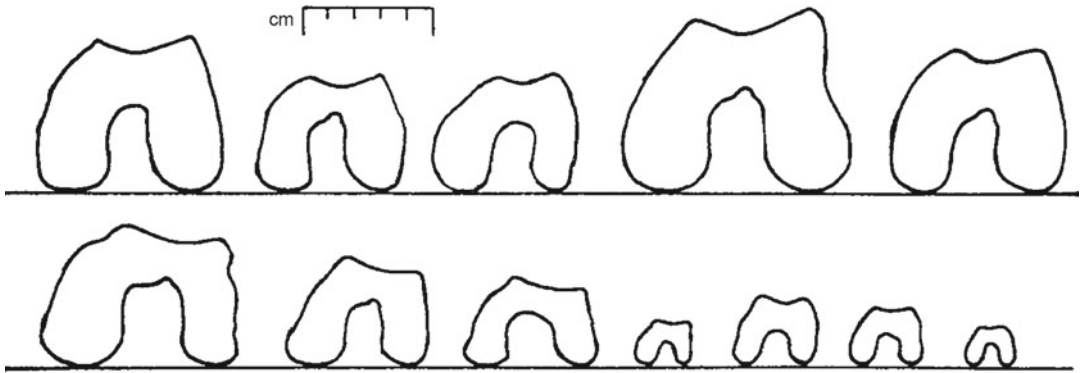


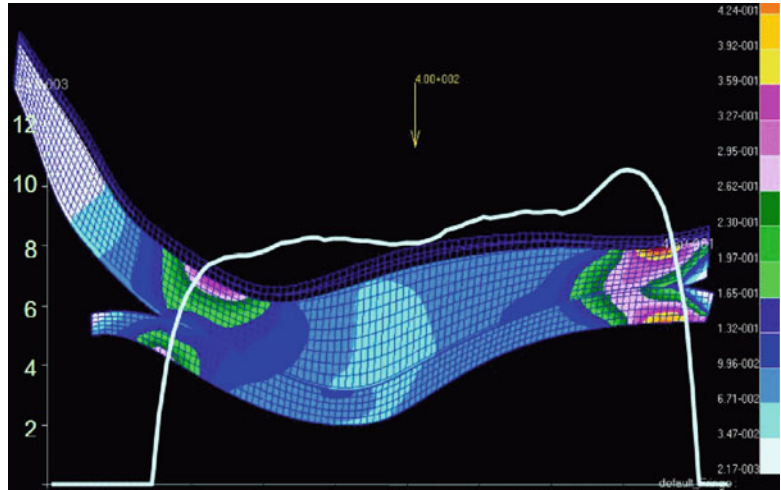
Fig. 44.5 These drawings are traced outlines from scale photographs of distal femurs arranged (or reversed) so that the lateral condyle is to the right. Primates with erect bipedal gait are on the top row, and

primates with quadrupedal gait are shown on the bottom (Redrawn from Heiple KG, Lovejoy CO. The distal femoral anatomy of Australopithecus. *Am J Phys Anthropol.* 1971;35:75-84)

Fig. 44.6 This drawing depicts safe straight leg weight lifting (or isometric progressive resistive quadriceps) exercises (From Merchant AC. Patellofemoral disorders: biomechanics, diagnosis, and non-operative treatment. In: McGinty JB, Casperi RB, Jackson RW, Poehling GG, editors. *Operative arthroscopy.* New York: Raven; 1991. With permission)



Fig. 44.7 Finite element analysis showing cartilage stress distributions during a static squat with the knee at 60° of flexion from an MRI image. Note stress concentrations adjacent to the subchondral bone (Image courtesy of Besier TF, Gold GE, Beaupre GS, et al. A modeling framework to estimate patellofemoral joint cartilage stress in vivo. *Med Sci Sports Exerc.* 2005;37:1924–30)



Index

A

- Acetylcholine (ACh)
 - intratendinous production, 110
 - synthesizing enzyme, 110
 - VACHT, 110
- ACh. *See* Acetylcholine (ACh)
- AKP. *See* Anterior knee pain (AKP)
- Anatomic dissection, right knee
 - lateral side, 184
 - medial side, 184
- Anatomy, patellar dislocation
 - axial MR image, 46
 - injuries, 41
 - lateral radiograph, 46
 - MPFL (*see* Medial patellofemoral ligament (MPFL))
 - patellofemoral articulation
 - anterolateral, 44
 - axial, 44
 - relationships, VMO and the MPFL, 45
- Anterior cruciate ligament (ACL) reconstruction
 - active heel lift, 114
 - hyperextension, 114
 - problem, 113
 - towel stretch extension exercise, 114
- Anterior knee pain (AKP)
 - arthroscopic patellar denervation (*see* Arthroscopic patellar denervation, AKP)
 - athletes (*see* Non-operative treatment, athletes with AKP)
 - autografts (*see* Autografts, anterior cruciate ligament reconstruction)
 - biological causes (*see* Biological causes, AKP)
 - etiology, 9–10
 - and functional patellofemoral instability, CT, 5–6
 - pain and disability (*see* Pain and disability, AKP)
 - and patellar instability (*see* Biomechanical bases, AKP and patellar instability)
 - pathogenesis (*see* Pathogenesis, AKP)
 - patients evaluation (*see* Kinetic and kinematic analysis, AKP)
 - prevention (*see* Anterior cruciate ligament (ACL) reconstruction)
 - risk factors and prevention
 - beneficial effect, 83
 - characteristics, 83
 - dynamic, multifactorial model, sports injury etiology, 84
 - hamstring and quadriceps, 83
 - intrinsic risk factors, 83
 - on-track brace, 85
 - patellofemoral bracing, 83–84
 - sequence, prevention, 84
 - single leg hop test, 85
 - uncommon causes (*see* Uncommon causes, AKP)
 - unilateral (*see* Unilateral AKP, knee asymmetry)
- Anteromedial tibial tubercle osteotomy
 - AMZ procedures, 229
 - anterior compartment musculature, 230
 - break-away pins, 231
 - cutting guide and block, 230
 - Gird's tubercle, 230
 - medialization, distance, 231
 - oscillating saw cooled, saline, 231
 - proximal cuts, 231
 - reference pin guide, 230
 - T3 system, 229
- Arthroscopic patellar denervation, AKP
 - anatomic dissection, 184
 - anterior view, knee and electrocoagulator insertion, 185
 - image, 184
 - knee joint, 183
 - macrophotography, 184
 - medial view, knee and electrocoagulator insertion, 185
 - procedure, 183
 - surgical view, knee and electrocoagulator insertion, 185
 - treatment, young patients, 183
- Autografts, anterior cruciate ligament reconstruction
 - central one-incision technique, 117
 - high-power view, biopsy, 119
 - infrapatellar nerve splits, 116
 - knee-walking test, 116
 - persistent donor site gap, 117, 118
 - postoperative donor-site morbidity, 115
 - reharvesting, 115
 - TEM in controls, lateral and central tendons, 120, 121
 - two-incision technique, 117

Autologous osteochondral mosaicplasty
 kissing lesions, PF, 243
 MRI, 243
 patella-graft flling, 242, 243
 pearls and pitfalls, 241
 transplanted plugs, 241
 traumatic osteochondral lesion, 243
 trochlea-graft filling, 242
 two-year-old control arthroscopy, 244

B

Biochemical causes, patellar tendinopathy
 clinical use, 109
 eccentric training regimens and surgery, 109
 experimental studies, 109
 intratendinous ACh production, 110
 intratendinous catecholamine production, 110
 mAChR, AR and NMDA R, 111
 neuronal/non-neuronal biochemical mediators, 109

Biological causes, AKP
 degenerative changes, 18
 infracted foci, 18
 mast cells, 13
 microneuromas, 14
 myxoid stromal degeneration, 18
 “nerve sprouting” process, 20
 “neural model”, 11
 neural sprouting, 18–19
 neuromas, 15
 NGF, 17
 normal nerve, 12
 osteoporosis association, 20
 periadventitial innervation, 16
 PFPS, 11
 retinacular tissue, 18
 VEGF, 19
 young vessels, 18

Biomechanical bases, AKP and patellar instability
 comparative values
 articular moment, 39
 pressures, 39
 reaction force, 39
 neural factor, 29
 “overuse”, 29
 patellofemoral joint
 action and quadriceps extension force, 31
 breast stroke, 35
 complementary weights, 32
 forces action, 30
 freestyle, 35
 graphic calculation, 30
 hip flexion, 32
 hyperflexion position, volleyball player, 36
 knee flexion, 30
 ligamentous laxity criteria, 37
 “miserable malalignment syndrome”, 34

promo sport, 30–31
 Q angle and valgus vector, 33
 quadriceps in closed/open kinetic chain,
 eccentric work, 38
 tendon adhesions, 32
 sport, 29
 treatment programme, 29

C

Cartilage restoration. *See* Patellofemoral joint (PFJ)

E

Ehlers–Danlos syndrome, 55
 Etiology, AKP
 inflammatory processes, 10
 pain mechanisms, 9
 retrospective analyses, 9

F

Femoral osteotomy
 distal fragmentation, 222
 Genu varum, 221
 skeletal alignment, 221
 transverse osteotomy, 222

Fulkerson osteotomy. *See* Anteromedial tibial tubercle osteotomy

Fulkerson’s relocation test
 contralateral asymptomatic knee, 52
 femoral trochlea, 52
 patella, 52

G

Gait analysis
 postoperative, 173
 preoperative, 173

I

Iatrogenic medial patellar instability
 conventional radiographs, 175
 extension and quadriceps contraction, 172, 174
 Gait analysis, 173
 knee kinetics and kinematics, 177–178
 lateral stress CT, 176
 medial patellar instability, 172
 medial stress CT, 176
 passive restraining structures, 171
 standard technetium 99 methylene diphosphonate bone scan, 176

Imaging and musculoskeletal models, PF pain
 axial CT image, 131
 axial MR image, 130
 axial-plane PF joint kinematics illustration, 129

- bisect offset and patellar tilt, relationship, 129
 - bone density assignment, CT-based Hounsfield Units, 133
 - cartilage and subchondral bone stress, 126
 - co-registered axial PET/CT image, 130
 - description, 125
 - EMG-driven musculoskeletal model, 132
 - femur finite element mesh, 131
 - hydrostatic stresses, 132
 - normalized contact, loaded and unloaded conditions, 127
 - peak patellar cartilage thickness, males and females, 128
 - sagittal MR image, 130
 - sagittal plane real-time MR images, 128
 - upright weight-bearing imaging, 126
- K**
- Kinetic and kinematic analysis, AKP
 - dynamometric platform disposition, 150
 - knee abduction moment during stair descending test, 157
 - knee extension moment, 153–154
 - knee flexion during stair descending test, 155
 - marker disposition, 151
 - stair descent
 - abduction-adduction, 177
 - flexion-extension, 177
 - ground force reactions, 177
 - knee joint angle, 177
 - strategies, 149
 - treatment progression, 149
 - vertical ground reaction force during stair descending test, 156
- L**
- Lateral patellar instability, evaluation
 - cases, 143–146
 - duplicate, 139
 - dynamometric platforms, 139
 - limitations, 139
 - mechanism, lateral patellar dislocation, 140
 - MPFL, 142
 - normalized moments and vertical reaction force, 141–142
 - rotator effect, 140
 - Lateral patellofemoral ligament (LPFL)
 - reconstruction
 - deep transverse retinaculum, 197
 - description, 197
 - isometric location, 202
 - lateral capsule and retinaculum, development, 198
 - lateral retinacular repair and imbrication, 201
 - lateral retinaculum, iliotibial band, 198
 - medial dislocation/subluxation, patella, 201
 - and MPFL reconstruction, 202
 - quadriceps graft, 202
 - tendon transformation, 199
 - LPFL reconstruction. *See* Lateral patellofemoral ligament (LPFL) reconstruction
- M**
- mAChR. *See* Muscarinic acetylcholine receptors (mAChR)
 - McConnell Program
 - assessment
 - flexibility, 99
 - patellar glide, 98
 - posterior tilt, 98
 - rotation, 99
 - common biomechanical presentation-internal rotation, 98
 - correct and incorrect limb alignment, 102
 - taping components, 100
 - therapist, 97
 - unloading fat pad, tape, 101
 - Medial patellofemoral ligament (MPFL)
 - axis, 42
 - macroscopic observation, 42
 - reconstruction (*see* MPFL reconstruction) and VMO, 43
 - Miserable malalignment syndrome
 - active young, 34
 - external tibial torsion, 34
 - femoral neck anteversion, 34
 - positive Helbing's sign, 34
 - MPFL. *See* Medial patellofemoral ligament (MPFL)
 - MPFL reconstruction
 - adductor magnus tendon, 192
 - biomechanical and technical principles, 193
 - central patella fracture secondary to drill hole, 196
 - description, 187
 - distalization, 195
 - drill holes, patella, 193
 - intraoperative photograph, 188–190
 - isometric point, medial epicondyle, 192
 - lateral patellar instability, 191
 - principles, 191
 - proximal position, femur, 194
 - quadriceps expansion, 192
 - redislocation, 196
 - tension, 195
 - transverse patella fracture secondary to transverse drill hole, 196
 - Muscarinic acetylcholine receptors (mAChR), 111
- N**
- Nerve growth factor (NGF)
 - immunoblotting detection, 17
 - Schwann cells, 17

- Neuromatous knee pain
 course and cutaneous territories, 168
 delineated territory, 169
 incision site, 169
 lateral aspect, 170
 medial aspect, 170
 medial cutaneous nerve, 170
 patella and vastus medialis muscle, 170
 procedure, 167
 proximal tibial screws, 169
 Tinels point and the paths, radiation, 169
- NGF. *See* Nerve growth factor (NGF)
- Non-operative treatment, athletes with AKP
 balance and coordination training, 87–88
 balance board training, 93
 cryotherapy, 87
 daily activity level, 87
 flexibility training, 87
 functional knee exercises, 88
 functional training, 88
 isokinetic quadriceps training, 96
 orthotics, 87
 patellar hypermobility exists, 87
 patient education, 88
 quadriceps strengthening, 88
 single-leg standing balance board training, 92
 squatting, 94
 stationary bicycle training, 88, 93
 stepping, 94, 95
 stretching, 90, 91
 transcutaneous electrical stimulation,
 VMO, 87, 89
- O**
- Osteotomy, PFJ
 axial CT scans, flat trochlea and lateralization, 207
 cause, 203
 convex proximal trochlea, 208
 dynamic superolateral patellar subluxation, 206
 femoral anteversion, axial CT evaluation, 204
 fixation, supracondylar rotation, 205
 internal and external rotated patellae, 204
 intraoperative view, flat trochlea, 207
 Kirschner wires indication, 205
 knee flexion, 203
 lengthening, 207
 MR measurement, 206, 209
 normal trochlea, 205
 osteochondral flap, 208
 patellar subluxation, 208
 refixation, osteochondral flap, 209
 TT-TG distance measurements, 209
- P**
- Pain and disability, AKP
 biopsychosocial model, 64
 classic biomedical approach, 63
 description, 63
 fear-avoidance model, 64
 psychological cause, 63
- Patella alta
 Caton–Deschamps index, 226
 description, 223
 height, correction, 226
 medial and distal tibial tubercle transfer, 227
 TT-TG distance, 227
- Patella infera
 and alta (*see* Patella alta)
 articular surface, 225
 Blackburne–Peel index, 224
 Caton–Deschamps index, 224
 definition, 223
 description, 223
 Insall–Salvati index, 224
 patella baja, 223
 tendon lengthening, 225
- Patellar dislocation. *See* Anatomy, patellar dislocation
- Patellar glide test
 contralateral asymptomatic knee, 51
 multidirectional instability, shoulder, 51
 PF joint, 48
 sulcus sign, 51
- Patellar instability and AKP
 apprehension test, 50
 arthroscopy, 47
 axial compression patellar test, 49
 CT scan, 59
 3D-CT reconstruction, patellofemoral
 joint, 60–61
 diagnostic tool, 48
 femoral anteversion, 57
 Fulkerson’s relocation test, 52
 full extension and patella, 50
 gastrocnemius flexibility, 53
 glide test, 48, 51
 hamstrings flexibility, 53
 hip flexion contracture, 58
 “J” sign, 56
 Merchant axial view, 58
 MRI signs, acute lateral patellar dislocation, 61
 Ober’s test, 54–55
 pathologic lateral displacement, 51
 physical examination, 47
 pronated foot, 57
 quadriceps flexibility, 53
 skin laxity, Ehlers–Danlos syndrome, 55
 tilt test, 49
 true lateral radiograph, 58
- Patellar tendinopathy/Jumper’s knee. *See* Ultrasound
 and Doppler-guided arthroscopic shaving
- Patellar tilt test, 49
- Patella thinning osteotomy
 articular surface, 237
 collapse, central portion, 235
 description, 233
 fixation, fragments with biodegradable pins, 235
 lateral rim, 235
 pilot hole, saw-bone, 236

- preservation, joint line, 236
 - removing, central patellar bone, 234
 - soft tissues attachments, proximal and distal poles, 235
 - total knee arthroplasty, 237
 - Patellofemoral (PF)
 - allografts, 245–246
 - arthritis (*see* Patella thinning osteotomy)
 - arthroplasty (*see* Patellofemoral (PF) arthroplasty)
 - pain (*see* Imaging and musculoskeletal models, PF pain)
 - Patellofemoral (PF) arthroplasty
 - anteroposterior, 250
 - axial stress radiograph, 254
 - extensive posttraumatic ossification, 253
 - femoral trochlea, 252
 - femoro-tibial compartments, 247
 - flexion–extension knee (stair descent), 256
 - intraoperative photograph, 252–253
 - knee abduction/adduction, 251, 252
 - knee flexion/extension, 251
 - knee joint angle, 254–255
 - lengthening, lateral retinaculum, 257–258
 - osseous metabolic activity, femoral condyles, 251
 - physical examination, patella alta, 250
 - postoperative CT, 253
 - prosthesis, 249
 - proximal/distal portion, 247
 - trochlear anatomy and component, 248
 - vertical ground reaction force, 251, 252
 - Patellofemoral joint (PFJ)
 - Blackburn–Peel technique, 260
 - cartilage stress distributions, 262
 - chondral pathology, 239
 - chondrosis, area, 240
 - computational models
 - description, 135
 - dualorthogonal fluoroscopic image system, 137
 - enhanced display, 135, 136
 - experimental determination, 138
 - femur and patella, 136
 - finite element model, 137
 - flexed knee, 138
 - graphical model, 135
 - MRI scanner, 136
 - inverse relationship, dysplasia and activity level, 260
 - marrow stimulation, trochlea, 240
 - osteotomy (*see* Osteotomy, PFJ)
 - pathomechanical and pathophysiological theory, 259
 - periosteal/collagen patch, 240
 - quadriceps, 260
 - safe straight leg weight lifting exercises, 261
 - scale photographs, distal femurs arranged, 261
 - subchondral bone, 240
 - Sulcus angle measurement, 260
 - Patellofemoral pain syndrome (PFPS), 11
 - Patellofemoral (PF) surgery, failure
 - axial and lateral x-ray, 166–167
 - clinical syndrome, 161
 - CT arthrogram, 162
 - CT rotational study, 164
 - double contrast CT arthrogram, 163
 - ligament reconstruction development
 - arthrosis, 165
 - medial dislocatable patella post realignment, 162
 - medial dislocation, 162
 - pathomechanics, 161
 - post right Maquet osteotomy, 163
 - preop CT rotational scan, 163
 - Pathogenesis, AKP
 - CT images at 0° of knee flexion, 22, 23
 - IPR surgery, 21
 - lysholm scores, 23
 - PFM and VMO, 21
 - SEMG activity, 24
 - VMO and VL, 24
 - PF. *See* Patellofemoral (PF)
 - PF allografts
 - articular cartilage, 245
 - bipolar transplant, 246
 - chondrocyte viability, 245
 - osteochondral allografts, 245
 - PFJ. *See* Patellofemoral joint (PFJ)
 - PF malalignment *vs.* tissue homeostasis
 - AKP and functional patellofemoral instability, CT, 5–6
 - dye envelope, function theory, 7–8
 - gadolinium-enhanced MR arthrotomogram, 7
 - physiopathological processes, 3
 - preoperative pain, 4
 - PFPS. *See* Patellofemoral pain syndrome (PFPS)
 - Post PF fresh allograft
 - axial x-ray, 165
 - lateral x-ray, 165
- S**
- Skeletal malalignment and AKP
 - AP postoperative x-rays, 108
 - bony, 103
 - CT scan, 105
 - excess femoral anteversion, 107
 - excess tibial external torsion, 108
 - femoral torsion, 105
 - K-wires, 108
 - lateral facet pressure change, 106
 - limb standing radiograph, mechanical axis, 104
 - MPF change, 106
 - pathogenesis, 103, 104
 - preoperative axial view, 108
 - tibial internal rotation osteotomy, 108
 - tibial torsion, 105
 - true lateral view, trochlear dysplasia, 105
 - Sports injury
 - dynamic and multifactorial model, 84
 - sequence, prevention, 84
 - Standard technetium 99 methylene diphosphonate
 - bone scan
 - osseous metabolic activity, femoral condyles, 251
 - osseous metabolic activity, patella, 176

Sulcus deepening trochleoplasty
 after surgical exposure, 214
 CT scan axial views before and after
 trochleoplasty, 215
 high-grade trochlear dysplasia, 212
 lateral and anterior views, dysplastic trochlea after
 trochleoplasty, 214
 osteochondral flaps, 214
 periosteum, 213
 pre- and postoperative lateral x-rays, 215
 surgical exposure, 213
 technical procedure, 211
 trochlear bump, calculation, 212
 trochlear dysplasia classification, 213

T

TH. *See* Tyrosine hydroxylase (TH)

Tibial osteotomy

cartilage restoration, 217
 femoral anteversion, 219
 increased sclerosis, 218
 internal and external, 220
 knee joint, 218
 limb alignment, 217
 measurement, torsion, 220
 miserable malalignment, 217
 realignment, femur, 217
 torsional limb malalignment, 217
 transverse (horizontal) plane, 217

Tyrosine hydroxylase (TH), 110

U

Ultrasound and Doppler-guided arthroscopic shaving
 dorsal tendon, 181
 gray-scale ultrasound and color Doppler
 examination, 182
 patellar tendinopathy/Jumper's knee changes, 182
 Uncommon causes, AKP
 after ACL reconstruction, 72–73, 81
 arthroscopic view, 82
 asymptomatic bilateral PFM, 77
 axial CT scan at 0° of knee flexion, 80

bipartite patella, volleyball player, 67
 continuous chronic anterior left knee pain, 74
 Coronal FSE PDW Fat Sat MRI, 70
 extraskeletal osteosarcoma, 75
 Hoffa's fat pad, 68
 intramuscular hemangioma, vastus medialis
 obliquus muscle, 67
 localized osteonecrosis, patella, 70
 magic angle phenomenon, 79
 nonspecific chronic synovitis, 76
 orthopedic surgery, 65
 osteochondritis dissecans, 66
 patient's symptoms, 65
 PFM, 65
 post-menisectomy osteoarthritis, 78
 sagittal FSE PDW Fat Sat MRI, 70
 sagittal GrE T2* MRI, 80
 sagittal plane FSE T1W MRI, 82
 sagittal SE T1W MR image, 76
 stress fracture, 71
 subperiosteal osteoid osteoma, 69
 swelling and pain, anterior tibial tubercle, 71
 trauma, car accident, 80
 Unilateral AKP, knee asymmetry
 extension loss, 27
 full knee extension, 25
 heel prop exercise, 28
 knee hyperextension, 27
 normal knee extension, 27
 physical examination, 26
 symmetry, 25
 towel extension exercise, 28

V

VAcHT *see* Vesicular acetylcholine transporter (VAcHT)
 Vastus medialis obliquus (VMO)
 macroscopic observation, MPFL, 42, 43, 45
 SEMG activity, 24
 tendon, 42
 transcutaneous electrical stimulation, 87, 89, 92, 94
 VL ratio, 24
 Vesicular acetylcholine transporter (VAcHT), 110
 VMO. *See* Vastus medialis obliquus (VMO)