Piero Volpi
Editor

Arthroscopy and Sport Injuries

Applications in High-level Athletes

Società Italiana di Artroscopia

Springer

Arthroscopy and Sport Injuries

Piero Volpi
Editor

Arthroscopy and Sport Injuries

Applications in High-level Athletes

Springer

Editor
Piero Volpi
Knee Orthopedics and Sport Traumatology Unit
Humanitas Clinical Institute IRCCS
Milan
Italy

Head of Medical Staff FC Internazionale Milan
Milan
Italy

The educational contribution offered by Smith & Nephew is gratefully acknowledged

ISBN 978-3-319-14814-4 ISBN 978-3-319-14815-1 (eBook)
DOI 10.1007/978-3-319-14815-1

Library of Congress Control Number: 2015937616

Springer Cham Heidelberg New York Dordrecht London

Printed on acid-free paper

Springer International Publishing AG Switzerland is part of Springer Science+Business Media (www.springer.com)

Foreword I

I am particularly happy to be a part of this major project as Chairman of the Italian Society of Arthroscopy (SIA), as an orthopedic surgeon, and as a personal friend of Professor Piero Volpi.

As Chairman of the SIA, I am especially honored since this is the first time that our members have been called upon to contribute to a project of such high scientific value, a task that they all approached with great dedication and enthusiasm.

As an orthopedic surgeon I am grateful because this undertaking has allowed me to add another important experience to my professional career, which has been enriched through the opportunity of working together with leading Italian and European colleagues.

Finally, I am happy to participate in this project as a friend of Piero Volpi, with whom I share not only a deep commitment to work, mutual respect and friendship but also a strong passion for football, which, like our work, is a source of energy and motivation.

Gianezio Paribelli
President SIA,
Italian Society of Arthroscopy,
Ravenna, Italy

Foreword II

This is the first time that I have been called upon to write the foreword to a book, and the invitation probably means that I am getting old! However, as President of ESSKA this is one of my duties, and in this particular case it is also a great pleasure.

I feel that I have known Piero Volpi forever, and over the past 15 years I have shared my professional life with him. Throughout this time we have worked very closely together on the basis of a friendly and loyal relationship.

Piero Volpi was a professional footballer in his youth, and football has never ceased to be at the center of his life. After his retirement as a player, he went on to put his medical training to use as an orthopedic surgeon. As one of the pioneers of Italian arthroscopy and a sports trauma specialist, he has been able to combine his two great passions: Sport and Orthopedics.

Piero Volpi has devoted much time and effort to practical scientific research with the aim of enhancing clinical outcomes and thereby benefiting the patients and athletes for whose care he is responsible.

I believe that this book perfectly mirrors this marriage of experience and research and is a worthy testament to Piero Volpi's inexhaustible desire to gain knowledge and expand his scientific experience.

This work will be very useful for both trainees and experienced practitioners in the field of orthopedic sports traumatology and will be invaluable for all who would like to practice arthroscopic surgery.

Matteo Denti
President ESSKA,
European Society of Sports Traumatology, Knee Surgery and Arthroscopy,
Milan, Italy

Preface I

Sport has long been a necessity for a large part of the population. During recent years, traditionally popular activities have been supplemented by the emergence of various new types of sport that have broadened the choice available to participants. Furthermore, the creation of new or the restructuring of existing facilities, gymnasiums, and natural places (jogging trails, ski resorts, golf courses, etc.) is progressively increasing the chances for everyone to become regularly involved in their favorite sport. This increase in opportunities for physical exercise and access to sports has in turn given rise to the need for many more researchers and specialists who have expertise in the classification, etiology, diagnosis and treatment of the diverse traumatic diseases that may arise during sporting activities. Greater attention has been devoted to the study of athletic skills, the development of better training methods, a sound understanding of biomechanics, and knowledge of the rules and materials applicable in each sport. In the medical literature, studies have focused on the epidemiology of trauma, on various aspects of injury prevention, and on the diagnostic, evaluative, therapeutic and rehabilitative options relevant to specific sports populations. The extensive background research in sports traumatology that has been conducted with the professional athlete in mind has also resulted in practical applications for the world of amateur sports and the general patient population. In an era in which sports performance, especially for high-level athletes, is reaching previously unimaginable limits, it is obvious that joints and musculotendinous structures may be subjected to repetitive stress and increasingly damaging insults. A higher incidence of trauma in professional sports is also being fostered by the longevity of sports careers, the intensity of workouts, the frequency of matches and competitive events, and the inadequate recovery periods, often dictated by economic interests.

In this context, over the past 20 years, as numerous arthroscopic techniques have emerged and become established, surgeons have made strenuous efforts to improve the reparative and reconstructive surgical techniques for the most frequent injuries, especially trauma to the meniscus and ligaments and osteocartilaginous lesions. Advancements in surgery and, in equal measure, a better understanding of anatomy and biomechanics have highlighted the importance of aspects such as the optimal choice of surgical technique and usage of surgical materials. The increasing use of biotechnology and biomaterials is also impacting significantly on the field. The pressing needs of the elite athlete, the type of sport, the time point during the season at which the injury occurs, the phase of the athlete's career and the athlete's age are among the parameters that determine the options open to the sport traumatologist.

All of the above aspects are addressed in this book, which describes the current applications of arthroscopy across a very wide range of sports injuries, explaining the mechanisms of injury for each condition and describing the role of arthroscopy in diagnosis and treatment.

Piero Volpi
Humanitas Research-IRCCS
Milan, Italy

Preface II

Arthroscopic surgery has been a fundamental part of the treatment of certain sports-related disorders for a number of years. Against this background, I wish to thank Dr. Piero Volpi for accepting the responsibility for developing a text, aimed at young orthopedists and others, in which arthroscopy and sports traumatology are perfectly intertwined.

Within Italy, the specialty of sports traumatology that we know today originated during the 1960s, when, under the patronage of the FMSI (Italian Federation of Sport Medicine), the first Italian Congresses of Sports Traumatology were organized by Profs. La Cava and Venerando. The major breakthrough, however, occurred in 1967 during the conference organized by the Orthopedic Institute G. Pini, University of Milan: it was then that the first Centre of Sports Traumatology was created at the university, under the direction of Prof. Albino Lanzetta. As a result of this initiative, other universities introduced courses on sports traumatology and started to undertake studies and produce scientific papers on the subject. After an enlightened publisher recognized the value of such work, the bilingual publication *Italian Journal of Sports Traumatology* was founded, with Prof. Mario Boni and Prof. Lamberto Perugia as editors.

Simultaneously, the Italian Society of Sports Traumatology (SI.Tra.S) was formed. Many distinguished orthopedists have served as the chairman of this society, including Prof. D. Tagliabue, Prof. P.G. Marchetti, Prof. A. Lanzetta, Prof. G. Cerulli and Prof. C. Velluti. Each year, the Congress has focused on a single theme (rugby, basketball, artistic gymnastics, volleyball, winter sports and, most recently, golf). In 2004, SI.Tra.S merged with the newly established Italian Society of Arthroscopy Knee Cartilage and Sport (SIGASCOT).

The early Italian experiences of arthroscopy were all attributable to a group of young surgeons who, in the early 1980s, offered this technique despite the various difficulties and the reticence that they encountered from academic circles. This group founded the Italian Arthroscopy Group (GIA), which later became the Italian Arthroscopy Society (SIA). Over the years, the leaders of this group became colleagues. They included Drs. Aglietti, Mariani, Pellacci, Benazzi, Bianchi, Branca, Frizziero, Gandolfi, Coari, Zini, Pederzini, Viola, Priano, Minola and others, who slowly but persistently persuaded the orthopedic world of the value of the arthroscopic technique. Towards the mid-1990s, S.I.Tra.S and SIA began to collaborate closely, and the publication of S.I.Tra.S became the official journal of the SIA. This close collaboration has led many lovers of sports traumatology to obtain more experience of the arthroscopic technique and, conversely, many arthroscopists to deepen their knowledge of sports traumatology. The consequence has been great benefits for the treatment of many pathological conditions related to sports. These benefits have not only been aesthetic; for example, the recovery times from sports injuries have also been reduced. I believe that this fruitful collaboration has stimulated my friend Dr. Piero Volpi, editor of this volume, to present the different conditions related to sports injuries in which the technique of arthroscopy plays a significant role in treatment and outcome. I wish all readers, in particular young colleagues who are encountering sports traumatology more and more frequently, an enriching reading experience, remembering that we never stop learning and that the challenges never cease.

Alberto Branca
Past President, SIA, Italian Society of Arthroscopy, Bologna, Italy

About the Editor

Dr. Piero Volpi is an Orthopedic Surgeon and Sports Medicine Specialist and Director of the Knee Surgery and Sport Traumatology Unit at Humanitas Clinical Institute IRCCS, Milan. He played professional football from the beginning of the 1970s until 1985 while maintaining his medical studies. He is now Head of Medical Staff FC Internazionale Milan, Italy, and is also Responsible Physician of the Italian Association of Football Players (AIC). Dr. Volpi is Teaching Professor at the Speciality School of Orthopedics and Traumatology at the University of Milan. He is also Vice-President of Italian Arthroscopy Society (SIA) and member of several National and International Scientific Societies: SIOT, SIGASCOT, FMSI, ISAKOS, ESSKA, and EKA. He is the author of more than 300 publications and over 600 contributions, including book chapters, articles and presentations to scientific National and International meetings. He is Editor in Chief of the *Journal of Sports Traumatology* and member of Editorial Board of *Medicina dello Sport Journal*.

Contents

Future in Arthroscopy and Sports Medicine

1

Fabio V. Arilla, Benjamin B. Rothrauff, Claudia Guglielmino, Christopher D. Murawski, and Freddie H. Fu

1.1 Introduction

During the 41st Congress of the German Society of Surgeons in 1912, Severin Nordentoft, an unknown Danish surgeon, presented his self-built "trocar-endoscope" and described how to use it for cystoscopy, laparoscopy, and endoscopy of the knee joint. In his report, he used the term "arthroscopy" for the first time in a medical meeting. Despite this, his technology received little attention, as most physicians at that time were more interested in the pervasive problems of fracture, sepsis, and tuberculosis [1, 2]. Now, more than 100 years after Dr. Nordentoft's presentation, arthroscopic surgery has become the gold standard for treatment of many joint diseases, especially those involving the knee and shoulder. Arthroscopy continues to evolve, with ongoing research seeking to improve arthroscopic techniques and to expand the technology to procedures that are presently done via open approach. These efforts will be bolstered by advances in computing technology and biologics, offering the prospect of minimally invasive anatomical reconstruction with subsequent recapitulation of native tissue structure and function [3, 4].

1.2 Noninvasive Devices for Improved Diagnosis and Prognosis

A correct diagnosis is key for successful treatment in any medical field. In this regard advances in technology, especially in imaging, aid in the diagnostic process. However, the physical examination remains the most cost-effective and accurate diagnostic tool for the vast majority of injuries. In the near future, it may be possible to enhance the diagnostic power of the physical exam by quantifying exam parameters through the implementation of emerging technologies. For instance, several devices are being developed to quantify the pivot-shift test (PST) of the knee joint. The PST detects rotatory knee laxity, the magnitude of which correlates with anterior cruciate ligament (ACL) insufficiency [5]. Additionally, a positive PST following ACL reconstruction correlates with poor subjective and objective outcomes, even when the graft remains intact [5, 6]. However, subjective grading and variability in technique among examiners prevents valid comparison across studies. In an attempt to objectify the PST, some authors have employed invasive devices such as navigation systems and electromagnetic devices [6, 7]. Despite reliable parameters obtained with these methods, there are several limitations, including cost, complexity, and the need to be used in the operating room [8]. To address these issues, noninvasive devices are being developed. As an example, the triaxial accelerometer [9] is a novel device where the accelerometer is attached to the lateral aspect of the tibia and is wirelessly connected to a laptop computer. The accelerometer records the triaxial acceleration during the PST [9, 10]. Another promising device is the PIVOT® software [5]. It can be downloaded to a regular tablet computing device, and it is able to quantify the anterior tibial translation of the lateral tibial compartment during the PST by tracking stickers placed on predetermined bony landmarks: the femoral lateral epicondyle, fibula head, and Gerdy's tubercle (Fig. 1.1).

It is anticipated that these emerging technologies will allow objective quantification of previously subjective clinical grades, reducing interobserver discrepancies and improving diagnostic accuracy. Nevertheless, additional tools are needed to provide diagnostic and prognostic information, especially in the absence of overt signs and symptoms, a scenario often encountered in chronic degeneration of musculoskeletal tissues. To that end, advanced imaging techniques offer the possibility of early identification of

F.V. Arilla, MD • B.B. Rothrauff • C. Guglielmino, MD
C.D. Murawski • F.H. Fu, MD, DSc (Hon), DPs (Hon) (✉)
Department of Orthopaedic Surgery, University of Pittsburgh, 3471 Fifth Avenue, Pittsburgh, PA 15213, USA
e-mail: fabioarilla@hotmail.com; ben.rothrauff@gmail.com; claudiagugli@hotmail.com; cdmurawski@gmail.com; ffu@upmc.edu

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_1,

Fig. 1.1 (**a**) A surgeon performs the PST while an assistant holds the tablet. The PIVOT® software tracks the stickers placed on the bony landmarks. (**b**) The software analyzes the magnitude of the reduction phase of the PST. The chart shows anterior tibial translation in the lateral compartment of 4.7 mm

tissue damage before it is appreciable with conventional imaging modalities (i.e., plain films, MRI, CT) or arthroscopic inspection. For instance, quantitative MRI UTE-T2* mapping revealed deep cartilage tissue changes following ACL injury and reconstruction [11]. Likewise, the identification of biological markers (biomarkers) indicative of acute or chronic musculoskeletal pathology is an area of extensive investigation [12, 13]. Both approaches offer the prospect of earlier diagnosis, the enhanced prognostic power, and the ability to monitor the efficacy of therapeutic interventions. However, it should be recognized that much of this research is still in development. Validation in preclinical and clinical studies must be achieved prior to widespread adoption in the general patient population. Cost is also an important aspect to these modalities and must be considered.

1.3 Injury Prevention

Diagnostic tools that provide earlier and more accurate characterization of sports injuries offer the opportunity for the introduction of therapies that stave off further tissue damage or promote healing. But even the earliest interventions following injury are costlier and likely to yield less satisfactory results than prevention of the injury in the first place. There is an increasing appreciation for the role that fatigue and impaired movement patterns play in injury. In particular, neuromuscular fatigue can alter joint biomechanics and diminish postural control, as shown following exercise of the lower and upper extremities [14, 15]. As a result, monitoring of fatigue during competition is essential to reduce injury risk, as are training programs designed to improve neuromuscular control and improve muscular endurance. As an example, a multitude of neuromuscular and educational programs have been shown to reduce the mean incidence of ACL rupture by 50 %, but the estimated effect differs largely between studies [16]. In particular, neuromuscular training programs may be most effective in reducing injury in at-risk populations (e.g., female basketball and soccer athletes), as the efficacy of prevention programs in male athletes was shown to be limited and equivocal [17]. An improved understanding of the mechanisms of sports injuries and confirmation of the efficacy of strategies aimed at minimizing their occurrence will be essential in promoting safe participation in sport, extending from the recreational athlete to the elite sportsmen.

1.4 Individualized Treatment

When injury does occur, and conservative treatments are unable to restore adequate musculoskeletal function, surgical intervention may be indicated. Despite great progress in arthroscopic surgery in the past few decades, allowing faster recovery and improved outcomes, no surgical approach is universally successful. One possible factor contributing to surgical failures may be the standardization of surgical techniques that ignore anatomic variation between individuals. It is now accepted that surgical reconstruction that restores native anatomy yields better outcomes than nonanatomic approaches [5]. As an example, ACL reconstruction has evolved from positioning bone tunnels by a clock face technique to direct visualization (or even computer-assisted localization) of the insertion sites. These improvements in surgical techniques were predicated upon improved understanding of joint anatomy.

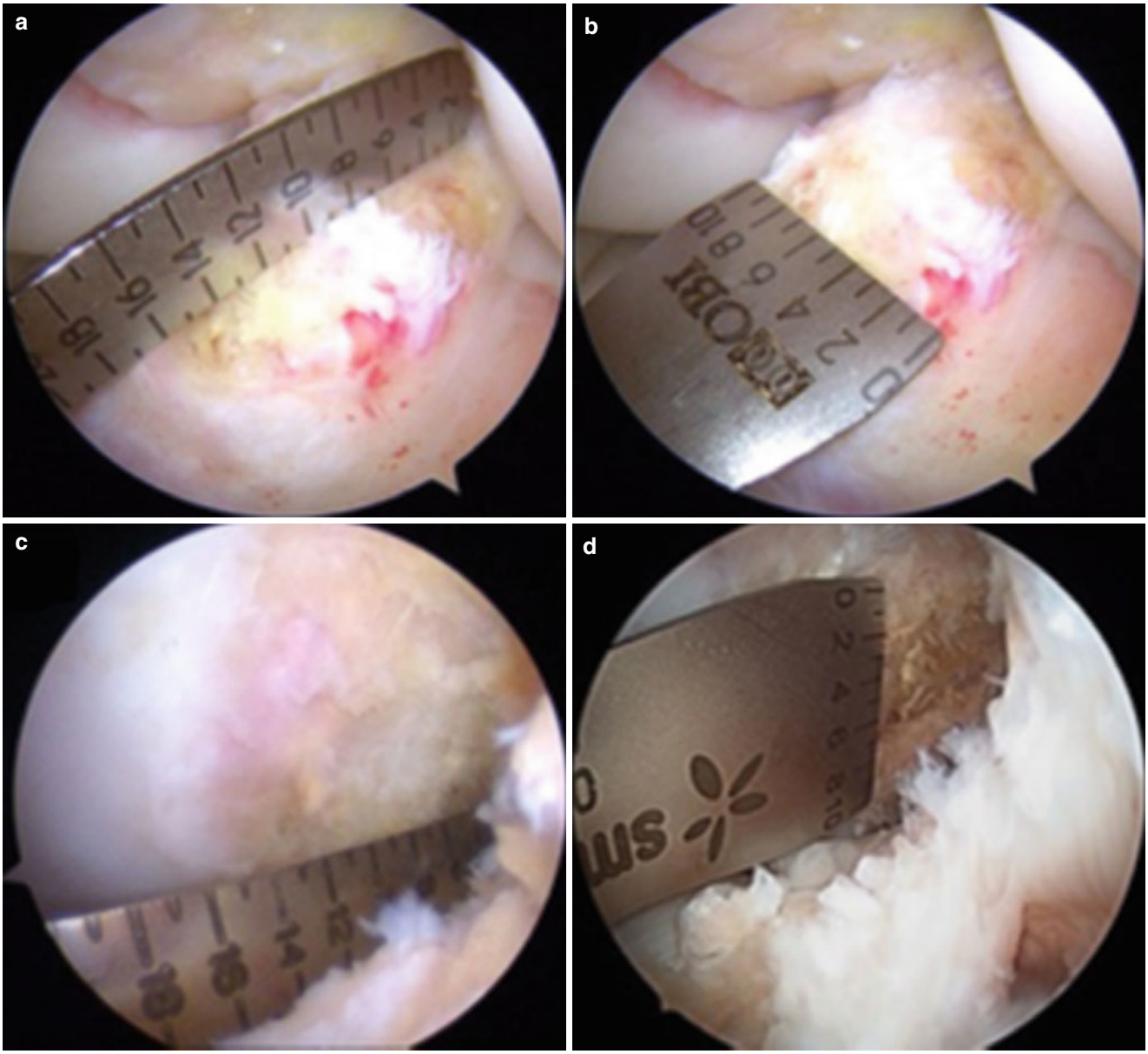


Fig. 1.2 Arthroscopic measurements of the tibial insertion site (**a**) length and (**b**) width; femoral insertion site (**c**) length and (**d**) height (From Hofbauer et al. [19] with permission)

Likewise, the introduction of double-bundle reconstruction led to more accurate restoration of native ACL anatomy. Nevertheless, there was room for improvement; further individualization of treatment was necessary. In particular, Ferretti et al. [18] found that the ACL tibial footprint length ranged from 13.7 to 22.1 mm, demonstrating the variability in anatomy among patients. As a result, measurements of the ACL insertion sites and bony structures of the knee joint are crucial for a successful individualized ACL reconstruction. That is, double-bundle reconstruction may not always be indicated. Tibial insertion site length of <14 mm is more suitable for a single-bundle technique, while insertion sites >18 mm may be better served with a double-bundle reconstruction given that two tunnels would cover a larger percentage of the native ACL insertion site [19]. The intercondylar notch is another anatomical reference that can be used when deciding between single- and double-bundle reconstruction. A notch width of <12 mm may be too small for placement of two tunnels. Similarly, a shallow notch (<12 mm) may lead to impingement of the graft [5]. See Fig. 1.2 for an example of intraoperative measurements used in individualized ACL reconstruction as it is performed at the University of Pittsburgh.

It is important to understand that individualized anatomical ACL reconstruction is a concept rather than a technique. It can be described as four basic principles: (1) to

restore the functional bundles of the ACL, (2) to restore the anatomical footprints, (3) to tension each bundle according to native tensioning pattern, and (4) to customize the surgery for each individual patient. Customizing the surgery comprises more than the decision to utilize either single- or double-bundle technique. Additional characteristics including graft choice, patients' preferences, lifestyle, and activity level must be taken into account when planning for individualized anatomical ACL reconstruction. Beyond this, the surgeon must take into account if there is a partial or complete ACL tear and the utility of pre- and postoperative rehabilitation for each individual patient [19].

1.5 Computer-Assisted Navigation

Computer-assisted surgery (CAS) was developed with the intention to facilitate more accurate and precise results while reducing the learning curve in knee arthroplasty, trauma, and spine surgery. Efforts are now being made to incorporate computer-assisted solutions to arthroscopic surgery [20]. CAS in ACL reconstruction has now reached more than 20 years of research [21]. The main goal of navigated procedures has been to improve the graft tunnel positioning [22], as it has been shown that incorrect tunnel position is the main reason for graft failure and persistent joint instability after ACL reconstruction [23]. Several reports have shown some improvement in the accuracy of tunnel placement with computer assistance during ACL reconstruction (Fig. 1.3) [20, 25, 26]. Furthermore, CAS may facilitate learning for junior surgeons and help experienced surgeons in complicated cases [27]. It also has been a valuable tool in research due to its precision in intraoperative evaluation of joint anatomy and laxity. For these reasons, it is probable that CAS will play an increasing role in all surgical fields, including arthroscopy for sports injuries.

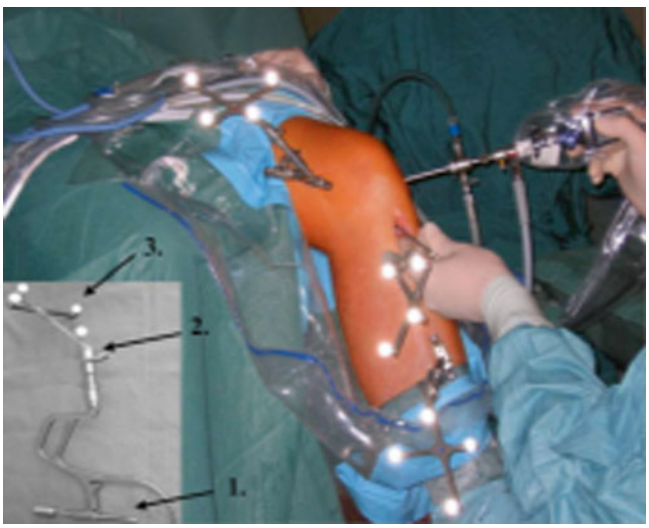


Fig. 1.3 Photograph showing arthroscopy of the left knee with computer assistance. K-wires (*arrow 1*) with rigid bodies (*arrow 2*) and reflective markers (*arrow 3*) are placed at the distal femur and proximal tibia to provide a connection to the OrthoPilot® navigation system (From Hofbauer et al. [24] with permission)

1.6 Biologics in Sports Medicine

Now in the third decade after the seminal publication of Langer and Vacanti [28] describing tissue engineering – the application of cells, scaffolds, and bioactive agents to replace or repair damaged tissue – there is rapidly expanding interest in using biologics to enhance healing of sports injuries [29]. While ex vivo engineering of whole organs derived from an autologous cell source may provide a novel, and desperately needed, alternative for organ transplantation, successful application of tissue engineering in sports medicine will likely require recapitulation of native tissue structure and function in focal defects (e.g., osteochondral lesions) or gap defects (e.g., nonunion fractures or tendon ruptures). Several technologies have shown promise clinically, including autologous chondrocyte implantation (ACI) [30] for focal cartilage lesions and the administration of bone morphogenetic protein-2 (BMP-2) for nonunion fractures and spinal fusion [31]. However, these early-generation biologics are not without limitations. In particular, first-generation ACI, which utilized periosteal membranes in conjunction with ex vivo expanded autologous chondrocytes, was associated with hypertrophy [32]. Additionally, the preponderance of fibrocartilage formed in the repair site, the expense of cell expansion in accordance with good manufacturing practice (GMP), and the uncertain clinical superiority over microfracture have prevented broad adoption of this biological approach. Likewise, the broader implementation of BMP-2 to improve bone healing has been slowed by questions of cost-effectiveness [31] and potential complications coupled with financial conflicts of interest that mired early reports on its efficacy [33]. While newer generations of these products may obviate several of these potential limitations, economics will continue to influence clinical care, necessitating the use of biologics in a one-step procedure. The biologics will likely be procured from the patient's own tissues, available as off-the-shelf, time-efficient products, or some combination thereof [34] (Fig. 1.4).

Equally important to the success of biologics in promoting healing is the growing understanding of the role of musculoskeletal loading in tissue remodeling. Recent studies on postoperative rehabilitation of musculoskeletal tissues, such as the rotator cuff [36, 37] and flexor [38] and Achilles tendons [39], point to the need for controlled mobilization [40]. However, the relationship between physical therapy and biologics remains largely unknown [41]. Interestingly, several

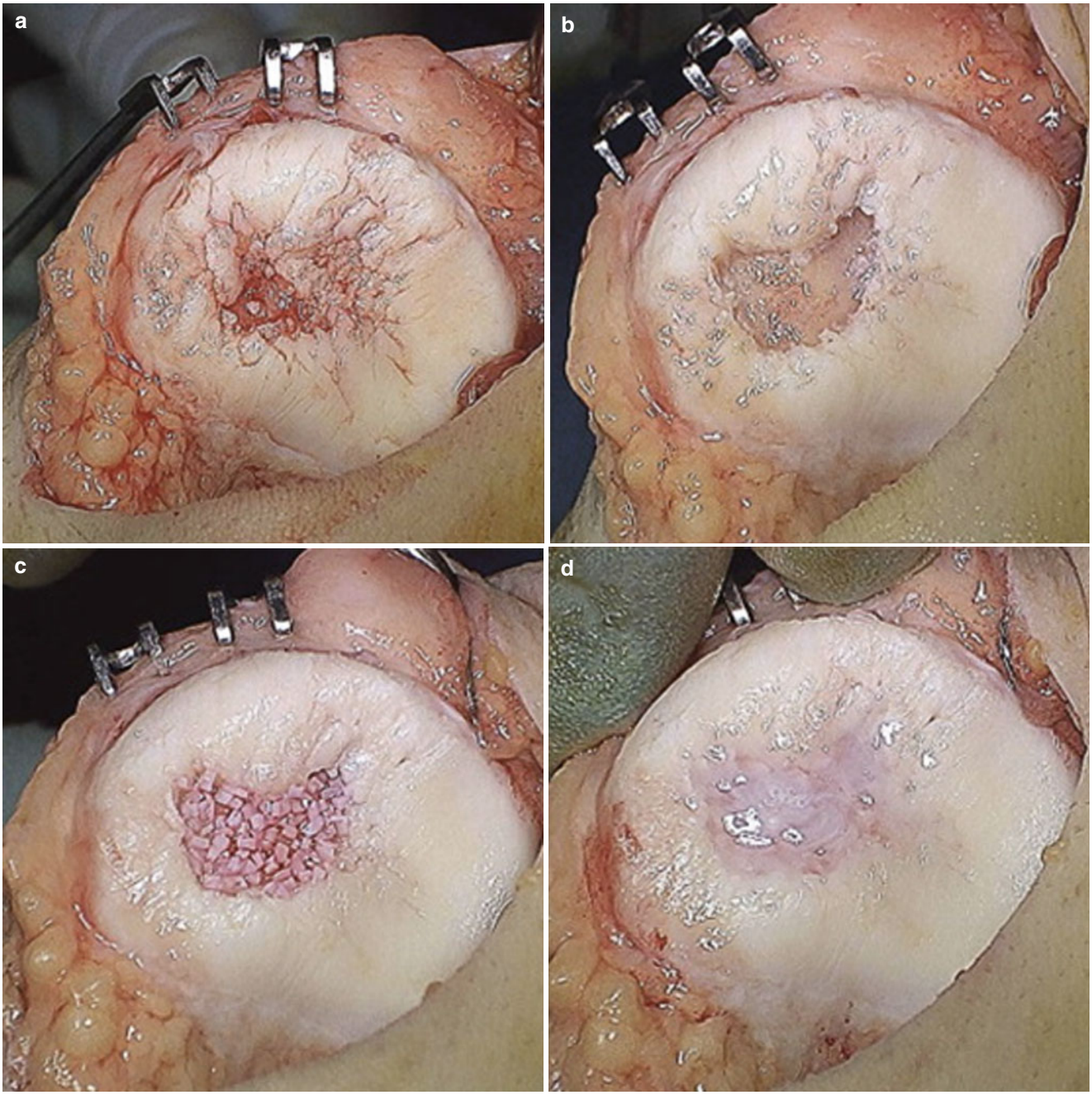


Fig. 1.4 Single-stage cartilage restoration technique utilizing particulated juvenile articular cartilage pieces placed in the chondral defect of the patella. The patella chondral defect (**a**) is prepared for allograft placement (**b**). Juvenile articular cartilage pieces are localized to the defect by fibrin glue (**c**). Additional fibrin glue seals the allograft in the defect (**d**). Similar single-step biological approaches are being explored in an effort to improve articular cartilage regeneration (Reproduced with permission from Tompkins et al. [35])

preclinical models suggest a synergistic effect between biologics and mechanical loading. Hodde et al. [42] demonstrated that early motion of an Achilles defect augmented with a small intestine submucosa scaffold produced superior tissue remodeling when compared against immobilized joints. Likewise, Virchenko and Aspenberg [43] found that platelet-rich plasma interacted synergistically with mechanical loading to improve structural properties of a healing Achilles tendon in a rat model, but this effect was most pronounced when loading began after the inflammatory phase of healing (~5 days). In a similar study, Ambrosio et al. [44] showed that the engraftment of mesenchymal stem cells in damaged muscle tissue was enhanced by functional muscle stimulation. Clearly, future investigations to elucidate the complex mechanical and biochemical microenvironment of healing musculoskeletal tissues, coupled with a

growing understanding of the mechanisms by which biologics exert favorable effects, will be essential to fulfill the promise of biologics in sports medicine.

Conclusion

Emerging technologies and continual refinement of surgical techniques will further expand the utility of arthroscopy in treating sports injuries. While a growing understanding of joint biomechanics and neuromuscular control may help prevent injury, enhancements in diagnostics and anatomical reconstruction are likely to improve outcomes when damage does occur. Biologics also offer the possibility of restoring native structure and function as closely as possible to musculoskeletal tissues that possess a poor innate healing capacity. In all, these emerging technologies hold tremendous promise for the future of arthroscopy and sports medicine. However, meticulous research and prudent implementation of these devices is arguably necessary to assure the maximum benefit to the patients we serve, whether it is an elite sports athlete or a recreational player.

References

1. Kieser CW, Jackson RW (2001) Severin Nordentoft: the first arthroscopist. Arthroscopy 17(5):532–535
2. Jackson RW (2010) A history of arthroscopy. Arthroscopy 26(1): 91–103
3. Iqbal S et al (2013) A history of shoulder surgery. Open Orthop J 7:305–309
4. Glick JM, Valone F 3rd, Safran MR (2014) Hip arthroscopy: from the beginning to the future–an innovator's perspective. Knee Surg Sports Traumatol Arthrosc 22(4):714–721
5. Araujo PH et al (2014) Individualized ACL reconstruction. Knee Surg Sports Traumatol Arthrosc 22(9):1966–1975
6. Ishibashi Y et al (2009) Navigation evaluation of the pivot-shift phenomenon during double-bundle anterior cruciate ligament reconstruction: is the posterolateral bundle more important? Arthroscopy 25(5):488–495
7. Kitamura N et al (2013) Biomechanical characteristics of 3 pivot-shift maneuvers for the anterior cruciate ligament-deficient knee: in vivo evaluation with an electromagnetic sensor system. Am J Sports Med 41(11):2500–2506
8. Araujo PH et al (2012) Comparison of three non-invasive quantitative measurement systems for the pivot shift test. Knee Surg Sports Traumatol Arthrosc 20(4):692–697
9. Lopomo N et al (2012) An original clinical methodology for non-invasive assessment of pivot-shift test. Comput Methods Biomech Biomed Engin 15(12):1323–1328
10. Maeyama A et al (2011) Evaluation of rotational instability in the anterior cruciate ligament deficient knee using triaxial accelerometer: a biomechanical model in porcine knees. Knee Surg Sports Traumatol Arthrosc 19(8):1233–1238
11. Chu CR et al (2014) Quantitative magnetic resonance imaging UTE-T2* mapping of cartilage and meniscus healing after anatomic anterior cruciate ligament reconstruction. Am J Sports Med 42(8):1847–1856
12. Lotz M et al (2013) Value of biomarkers in osteoarthritis: current status and perspectives. Ann Rheum Dis 72(11):1756–1763
13. Jayabalan P, Sowa GA (2014) The development of biomarkers for degenerative musculoskeletal conditions. Discov Med 92: 59–66
14. Frank BS et al (2014) Neuromuscular fatigue alters postural control and sagittal plane hip biomechanics in active females with anterior cruciate ligament reconstruction. Sports Health 6(4):301–308
15. Gandhi J et al (2012) Voluntary activation deficits of the infraspinatus present as a consequence of pitching-induced fatigue. J Shoulder Elbow Surg 21(5):625–630
16. Gagnier JJ, Morgenstern H, Chess L (2013) Interventions designed to prevent anterior cruciate ligament injuries in adolescents and adults a systematic review and meta-analysis. Am J Sports Med 41(8):1952–1962
17. Alentorn-Geli E et al (2014) Prevention of non-contact anterior cruciate ligament injuries in sports. Part II: systematic review of the effectiveness of prevention programmes in male athletes. Knee Surg Sports Traumatol Arthrosc 22(1):16–25
18. Ferretti M et al (2012) Bony and soft tissue landmarks of the ACL tibial insertion site: an anatomical study. Knee Surg Sports Traumatol Arthrosc 20(1):62–68
19. Hofbauer M et al (2014) The concept of individualized anatomic anterior cruciate ligament (ACL) reconstruction. Knee Surg Sports Traumatol Arthrosc 22(5):979–986
20. Nawabi DH et al (2013) Hip arthroscopy: the use of computer assistance. HSS J 9(1):70–78
21. Dessenne V et al (1995) Computer-assisted knee anterior cruciate ligament reconstruction: first clinical tests. J Image Guid Surg 1(1):59–64
22. Zaffagnini S, Klos TV, Bignozzi S (2010) Computer-assisted anterior cruciate ligament reconstruction: an evidence-based approach of the first 15 years. Arthroscopy 26(4):546–554
23. Meredick RB et al (2008) Outcome of single-bundle versus double-bundle reconstruction of the anterior cruciate ligament: a meta-analysis. Am J Sports Med 36(7):1414–1421
24. Hofbauer M et al (2010) Rotational and translational laxity after computer-navigated single- and double-bundle anterior cruciate ligament reconstruction. Knee Surg Sports Traumatol Arthrosc 18(9):1201–1207
25. Hart R et al (2008) Outcomes after conventional versus computer-navigated anterior cruciate ligament reconstruction. Arthroscopy 24(5):569–578
26. Plaweski S et al (2006) Anterior cruciate ligament reconstruction using navigation: a comparative study on 60 patients. Am J Sports Med 34(4):542–552
27. Plaweski S et al (2012) Evaluation of a computer-assisted navigation system for anterior cruciate ligament reconstruction: prospective non-randomized cohort study versus conventional surgery. Orthop Traumatol Surg Res 98(6 Suppl):S91–S97
28. Langer R, Vacanti JP (1993) Tissue engineering. Science 260(5110):920–926
29. Tuan RS (2013) The coming of age of musculoskeletal tissue engineering. Nat Rev Rheumatol 9(2):74–76
30. Brittberg M et al (1994) Treatment of deep cartilage defects in the knee with autologous chondrocyte transplantation. N Engl J Med 331(14):889–895
31. Garrison KR et al (2007) Clinical effectiveness and cost-effectiveness of bone morphogenetic proteins in the non-healing of fractures and spinal fusion: a systematic review. Health Technol Assess 11(30):1–150
32. Harris JD et al (2011) Failures, re-operations, and complications after autologous chondrocyte implantation – a systematic review. Osteoarthritis Cartilage 19(7):779–791
33. Carragee EJ et al (2012) A biologic without guidelines: the YODA project and the future of bone morphogenetic protein-2 research. Spine J 12(10):877–880

34. Evans CH et al (2007) Facilitated endogenous repair: making tissue engineering simple, practical, and economical. Tissue Eng 13(8):1987–1993
35. Tompkins M et al (2013) Preliminary results of a novel single-stage cartilage restoration technique: particulated juvenile articular cartilage allograft for chondral defects of the patella. Arthroscopy 29(10):1661–1670
36. Kim YS et al (2012) Is early passive motion exercise necessary after arthroscopic rotator cuff repair? Am J Sports Med 40(4):815–821
37. Parsons BO et al (2010) Does slower rehabilitation after arthroscopic rotator cuff repair lead to long-term stiffness? J Shoulder Elbow Surg 19(7):1034–1039
38. Griffin M et al (2012) An overview of the management of flexor tendon injuries. Open Orthop J 6(suppl1:M3):28–35
39. Schepull T, Aspenberg P (2013) Early controlled tension improves the material properties of healing human achilles tendons after ruptures: a randomized trial. Am J Sports Med 41(11):2550–2557
40. Killian ML et al (2012) The role of mechanobiology in tendon healing. J Shoulder Elbow Surg 21(2):228–237
41. Ambrosio F et al (2010) The emerging relationship between regenerative medicine and physical therapeutics. Phys Ther 90(12): 1807–1814
42. Hodde JP, Badylak SF, Shelbourne KD (1997) The effect of range of motion on remodeling of small intestinal submucosa (SIS) when used as an achilles tendon repair material in the rabbit. Tissue Eng 3(1):27–37
43. Virchenko O, Aspenberg P (2006) How can one platelet injection after tendon injury lead to a stronger tendon after 4 weeks?: interplay between early regeneration and mechanical stimulation. Acta Orthop 77(5):806–812
44. Ambrosio F et al (2010) The synergistic effect of treadmill running on stem-cell transplantation to heal injured skeletal muscle. Tissue Eng Part A 16(3):839–849

Injury Risk in the Olympic Games

2

Lars Engebretsen, Kathrin Steffen, and Torbjørn Soligard

2.1 Introduction

With more than 11,000 athletes from 204 countries, the XXX London 2012 Olympic Games were the largest sports event ever. The Olympic Winter Games are traditionally smaller, but more than 3,000 athletes participated in the XXI Winter Olympic Games held in Vancouver in 2010. The Games are spectacular media events, not just for the audience but probably even more for the participating countries and the athletes themselves. Protection of an athlete's health is an important task for the International Olympic Committee (IOC) [1]. Systematic injury and illness surveillance monitors trends over long periods of time, and the identification of high-risk sports, including their most common and severe injuries and illnesses, provides valuable knowledge to reduce the risk of occurrence [2–4]. Following the four-stage model of van Mechelen et al. [5], analysing the extent of a problem, such as high injury and/or illness risk in a specific population, is the first step in the development of effective prevention strategies.

As early as in 1998, the Fédération Internationale de Football Association (FIFA) started to survey all injuries incurring during their competitions [6–12], and other major sports federations followed the role model of FIFA's Medical Assessment and Research Centre (F-MARC) [13–17]. In 2004, an injury surveillance system was applied for all team sports during the Summer Olympic Games in Athens [9]. Based on these experiences, a group of experts, gathered by the IOC, developed an injury surveillance for multi-sport events [18], and the IOC performed, for the first time, an injury surveillance during the 2008 Beijing Olympic Games [3], and in the 2010 Vancouver Olympics a full injury and illness surveillance [2]. The surveillance study was further developed in the 2012 London [4] and 2014 Sochi Olympic Games.

For Olympic winter sports compared to summer sports, much less knowledge on injury risk exists. Furthermore, sports such as snowboard and freestyle skiing are relatively recent additions to the traditional Olympic winter sports. In 2006, the International Ski Federation (FIS) introduced an injury surveillance system for world-class skiing athletes in an attempt to record injuries in all FIS sports disciplines throughout a whole World Cup season and thereby monitor injury trends over time [19]. As the second step in the development of injury preventive strategies is to map the causes of injuries, new projects have been conducted to identify intrinsic and extrinsic risk factors and injury mechanisms [20–23], but many questions on how to protect high-risk athletes earlier in their careers still remain unanswered. Similarly, there is only a limited number of papers available aimed at investigating illnesses during single [7, 15, 16] or multi-sport events [2, 4].

Continuous injury and illness surveillance during these major sport events will build a foundation for providing evidence for health development in sports and for the development of injury prevention programmes [1]. The aim of the present chapter is to summarise the occurrence of injuries and illnesses in the three Olympic Games from 2008 to

The injury and illness surveillance during the XXIX 2008, XXX 2012 Summer, and the XXI 2010 Winter Olympic Games

L. Engebretsen (✉)
Department of Sports Medicine, Oslo Sports Trauma Research Center, Norwegian School of Sport Sciences, Pb 4014 Ullevål Stadion, 0806 Oslo, Norway

Medical & Scientific Department, The International Olympic Committee, Lausanne, Switzerland

Department of Orthopedic Surgery, Faculty of Medicine, Oslo University, Oslo, Norway
e-mail: lars.engebretsen@medisin.uio.no

K. Steffen
Department of Sports Medicine, Oslo Sports Trauma Research Center, Norwegian School of Sport Sciences, Pb 4014 Ullevål Stadion, 0806 Oslo, Norway

Medical & Scientific Department, The International Olympic Committee, Lausanne, Switzerland

T. Soligard
Medical & Scientific Department, The International Olympic Committee, Lausanne, Switzerland

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_2,

2012 [2–4] to enable the national team physicians to be better prepared for the coming Olympic Games. Practical implications and suggestions for further research to protect the athletes' health will be given.

2.2 Methods

The IOC injury surveillance system for multi-sport events was developed in 2008 [18]. The injury definition and data collection procedures were successfully implemented during the Olympic Games 2008 in Beijing [3]. Based on the experiences from athletics [15] and aquatic sports [16], surveillance was expanded to also include the registration of illnesses occurring during the Olympic Winter Games 2010 [2].

All National Olympic Committees' (NOC) head physicians were asked to participate in the Olympic surveillance studies and to report daily the occurrence (or nonoccurrence) of newly sustained injuries and illnesses (injuries only in Beijing) on a standardised reporting form (Medical Encounter Form is attached/supplemented). In addition, information on all athletes treated for injuries and illnesses by the local organising committee (OCOG) medical services was retrieved from the available medical centres located at major venues.

2.2.1 Implementation of Data Collection

Six months before the Games, the NOCs were informed about the study by the IOC. The medical representatives of all participating countries received a booklet with detailed information about the study, including the injury and illness forms to be filled out. Two days before the opening of the Games, NOCs physicians, physiotherapists, and the medical representatives of the Summer and Winter Olympic International Sports Federations were invited to a meeting covering the details of the studies. All NOC head team physicians were asked to submit a daily injury and illness form. In addition, athletes seen for an injury or illness in the venue medical stations or the central clinics were reported through the central clinic database. To encourage compliance with the reporting procedures during the Games, members of the study group were in frequent personal contact with the NOCs [2–4].

2.2.2 Injury and Illness Report Form

The report form required documentation of the following information: athlete's accreditation number, sport discipline/event, date and time of occurrence, time, competition/training, injured body part, injury type, causes, and estimated time loss. The illness part of the report form was located directly below the injury part on the same page and followed a similar design. The illness documentation included the diagnosis, affected system, main symptom(s), and cause of illness, as well as an estimate of time loss. Detailed instructions on how to fill out the form correctly were given in the booklet with example for injuries and illnesses. Daily injury information was also received from the polyclinic in the Olympic Village. Injury and illness report forms were distributed to all NOCs in the following languages of choice: Chinese, English, French, German, Russian, Spanish, and Arabic [2–4].

2.2.3 Definition of Injury and Illness

An athlete was defined as injured or ill if he/she received medical attention regardless of the consequences with respect to absence from competition or training. Following the IOC injury surveillance system, an injury should be reported if it fulfilled the following criteria: (1) musculoskeletal complaint or concussion, (2) newly incurred (pre-existing, not fully rehabilitated should not be reported) or reinjuries (if the athlete has returned to full participation after the previous injury), (3) incurred in competition or training, and (4) incurred during the period of the Olympic Games. An illness was defined as any physical complaint (not related to injury) newly incurred during the Games that received medical attention regardless of the consequences with respect to absence from competition or training [2–4].

All information was treated strictly confidentially, and the injury reports were made anonymous after the Olympic Games. Ethical approval was obtained by the Regional Committee for Medical Research Ethics, Region Øst-Norge, Norway.

2.3 Results

2.3.1 Response Rate and Coverage of the Athletes

All NOCs with more than 50, 30, or 10 (Beijing, London, and Vancouver, respectively) registered athletes were included in the analysis of response rate, and these countries represented more than 89 % of all participating athletes. In Beijing, the head physicians of all the participating NOCs returned a total 1,050 injury report forms (72 %). In addition, 264 injury report forms were received from medical stations at the different Olympic venues and through daily reports from the polyclinic in the Olympic Village [3]. Throughout the 17 days of the Vancouver Olympics, the 33 participating NOCs (with more than 10 athletes) returned a total of 461 out of a maximum of 561 forms to the project group (mean 82 %, range 77–89 %) [2]. In London [4], the response was better. Throughout the 17 days of the London Games, the 74 NOCs submitted a total of 1,204 of a maximum of 1,258 forms (mean 96 %, range 12–100 %) (Table 2.1).

Table 2.1 Response rates, injuries, and illnesses in NOCs of different sizes (measured by number of athletes)

	<30	30–99	100–200	>200	All
NOC (athletes)	132 (1,114)	37 (1,890)	20 (2,510)	15 (5,050)	204 (10,564[a])
Injuries (%)	256 (23)	308 (16)	380 (15)	416 (8)	1,360[b] (13)
Illnesses (%)	108 (10)	187 (10)	182 (7)	280 (6)	757[c] (7)
Report forms submitted[d] (%)	–	624 (97)	332 (93)	248 (98)	1,204 (96)

[a]Independent Olympic Athletes excluded
[b]NOC is missing for one injury
[c]NOC is missing for one illness
[d]Countries with less than 30 athletes were excluded from the response rate analysis

2.3.2 Incidence of Injuries

In Beijing, a total of 1,055 injuries were reported among 10,977 athletes, equivalent to an incidence of 9.6 injuries per 100 registered athletes [3]. Among the 2,567 registered athletes (1,045 females, 1,522 males) in Vancouver, a total of 287 injuries were reported resulting in an injury rate of 11.3 injuries per 100 registered athletes [2]. On average 10–11 % of the registered athletes sustained at least one injury. In total, 10,568 athletes took part in the London Olympic Games [4]. Of these, 4,676 were female (44 %) and 5,892 male (56 %). Among these athletes, we recorded a total of 1,361 injuries, equalling an overall injury rate of 12.9 injuries per 100 registered athletes. On average, 11 % of the athletes sustained at least one injury ($n=1,190$). There were 114, 18, and 7 athletes with 2, 3, and 4 injuries, respectively (Table 2.2).

2.3.3 Injury Risk in Different Sports

The incidence of injuries varied substantially among the different sports in all three Olympic Games. In relation to the number of registered summer sport athletes, the risk of sustaining an injury was highest for football, taekwondo, field hockey, handball, weightlifting, and boxing in Beijing (all ≥15 % of the athletes) [3]. In comparison, the relative risk of an athlete to be injured in the London Games was highest in taekwondo, football, BMX, handball, mountain bike, hockey, weightlifting, athletics, and badminton (15–39 % of registered athletes were affected in each sport) [4]. The relative injury risk was lowest for archery, canoe slalom and sprint, track cycling, rowing, shooting, and equestrian (less than 5 % of the athletes were injured) (Table 2.2). The injury risk in female (13.3 injuries per 100 athletes [95 % CI 12.2–14.3]) and male (12.1 [11.2–13.0], RR = 1.10 [0.97–1.22], $P=0.11$) athletes was similar. Nonetheless, male athletes were at significantly higher risk of injury in taekwondo (RR = 1.9; 1.1–3.5, $P=0.03$), whereas females were at higher risk of injury in football (RR = 1.7; 1.2–2.2, $P<0.001$).

In the Winter Games in Vancouver, injury risk was highest for bobsleigh, ice hockey, short track, alpine, and freestyle and snowboard cross (15–35 % of registered athletes were affected in each sport) [2]. Every fifth female athlete was injured in bobsleigh, ice hockey, snowboard cross, and freestyle cross and aerials, while the highest-risk sports for male winter sport athletes were short track (28 % of registered male athletes), bobsleigh (17 %), and ice hockey (16 %).

2.3.4 Injury Location and Type

In Beijing, the distribution of injuries was as followed: about half of the diagnoses ($n=600$; 54 %) affected the lower extremity, 20 % were related to the upper extremity ($n=218$), 13 % to the trunk ($n=149$), and 12 % to the head/neck ($n=133$). The thigh (13 %) and knee (12 %) were most commonly injured, followed by the lower leg, ankle, and head injuries (9 %), mainly diagnosed as skin lesions or contusions [3]. Similar results were found in the London Games (Table 2.3) [4].

In Vancouver, for both genders, the face, head, and cervical spine (female 20 %, male 21 %) and knee (female 16 %, male 11 %) were the most prominent injury locations, followed for females by wrist (8 %) and for male athletes by thigh (10 %). Contusions (female 32 %, male 26 %), ligament sprains (female 20 %, male 11 %), and muscular strains (female 8 %, male 16 %) were the most common injury types. In alpine, freestyle, and snowboarding, 22 out of 102 injuries (22 %) affected the head/cervical spine and one fourth of all injuries the knee (24 %). Twenty concussions were reported, affecting 7 % of the registered athletes. These athletes participated in the snowboard (boarder cross and half pipe) and freestyle disciplines (ski cross and aerials), in bobsleigh, in short track, in alpine skiing, and in ice hockey. A catastrophic injury with death as outcome occurred in luge [2].

2.3.5 Injury Mechanism and Circumstance

In Beijing, one third of the injuries ($n=282$; 33 %) were caused by contact with another athlete. Noncontact trauma ($n=172$; 20 %) and overuse either with gradual ($n=78$; 9 %) or sudden onset ($n=110$; 13 %) were also frequent causes of injury [3]. Similar results were found in the London Games [4]. In Vancouver, the three most common reported injury mechanisms were a noncontact trauma ($n=57$, 23 %),

Table 2.2 Injury and illness distribution (percentage of participating athletes) for selected sports on the programme for the 2008 Beijing, 2010 Vancouver, and 2012 London Olympic Games

	Injuries			Illnesses	
Olympic sports	Beijing 2008	Vancouver 2010	London 2012	Vancouver 2010	London 2012
Alpine skiing		14.9		4.2	
Aquatics					
Diving	2.1		8.1		5.1
Swimming	3.4		5.4		7.3
Synchronised swimming	1.9		13.5		12.5
Water polo	9.7		13.1		
Archery	7.0		1.6		7.8
Athletics	11.3		17.7		10.5
Badminton	4.7		15.9		3.0
Baseball	11.1		–		
Basketball	13.2		11.1		3.1
Beach volleyball	8.3		12.5		18.8
Biathlon		1.5		11.4	
Bobsleigh		20.0		4.4	
Boxing	14.9		9.2		6.4
Canoeing/kayaking	1.2		5.2		10.4
Cross-country skiing		3.1		6.8	
Curling		4.0		10.0	
Cycling	5.8				
BMX			31.3		4.2
MTB			21.1		6.6
Road			9.0		3.3
Track			3.0		19.6
Equestrian	5.2		4.5		5.5
Fencing	2.4		9.3		5.3
Field hockey	20.4		17.0		7.5
Figure skating		14.3	12.3		
Football	31.5		35.2		12.2
Freestyle					
Aerials		19.1		2.1	
Cross		19.0		2.9	
Moguls		1.8		0	
Gymnastics	7.5				
Artistic			7.7		2.6
Rhythmic			7.3		1.0
Trampoline			6.3		3.1
Handball	17.4		21.8		4.9
Ice hockey		18.5		5.6	
Judo	11.2		12.3		4.2
Luge		1.9		6.5	
Modern pentathlon	5.6		8.3		1.4
Nordic combined		1.9		7.8	
Rowing	1.8		3.3		7.3
Sailing	0.8		14.7		10.0
Shooting	7.8		3.8		4.4
Short track		9.0		9.2	
Skeleton		6.4		10.6	
Ski jumping		4.5		1.5	
Snowboard					
Cross		35.0		10.5	
Half pipe		13.0		5.8	

Table 2.2 (continued)

	Injuries			Illnesses	
Olympic sports	Beijing 2008	Vancouver 2010	London 2012	Vancouver 2010	London 2012
Slalom		6.8		6.8	
Softball	13.4		–		–
Speed skating		2.8		12.5	
Table tennis	5.2		6.3		6.9
Taekwondo	27.0		39.1		10.9
Tennis	5.9		11.4		2.2
Triathlon	9.2		14.5		6.4
Volleyball	8.0		6.9		2.8
Weightlifting	16.9		17.5		4.0
Wrestling	9.4		12.0		4.7
Total	9.6	11.2	12.9	7.2	7.

Table 2.3 Comparison between injuries sustained by athletes participating in Summer (Beijing 2008, London 2012) and Winter Olympic Games (Vancouver 2010)

	Beijing 2008	London 2012	Vancouver 2010
Participating athletes	10,977	10,568	2,567
Injuries (per 1,000 athletes)	1,055 (96.1)	1,361 (128.8)	287 (111.8)
Most common diagnosis	Ankle sprains (7 %), thigh strains (7 %)	Most severe injuries: shoulder, elbow, and knee dislocations, muscle strains and ruptures, fractures/stress fractures, ligament sprains and ruptures, incl. ACL, tendon ruptures	Concussions (7 %)
Most affected locations	Trunk (13 %), thigh (13 %), head/neck (12 %), knee (12 %)		Head/neck (16 %), knee (14 %), thigh (7 %)
Most common mechanisms	Noncontact (20 %)	Contact with another athlete (14 %)	Contact with another athlete (15 %)
	Overuse (22 %)	Noncontact (20 %)	Contact with a stationary object (22 %)
	Contact with another athlete (33 %)	Overuse (25 %)	Noncontact (23 %)
Expected time-loss injuries	50 %	35 %	23 %[a]
Competition – training injuries	73–27 %	55–45 %	46–54 %
High-risk sports (injuries per 100 athletes)	Football, taekwondo, field hockey, handball, weightlifting	Football, taekwondo, BMX, handball, MTB, athletics	Snowboard cross, freestyle aerials and cross, bobsleigh, ice hockey
Low-risk sports (injuries per 100 athletes)	Canoeing/kayaking, diving, rowing, sailing, synchronised swimming, fencing	Archery, canoe slalom and sprint, track cycling, rowing, shooting, equestrian	Nordic skiing disciplines, curling, speed skating

[a]This figure may be underestimating the number of time-loss injuries as the response rate to this information was low and many of the injuries were of severe outcome, without estimated time loss registered (more details in the Vancouver paper) (Engebretsen et al. [2])

contact with a stagnant object ($n=54$, 22 %), and contact with another athlete ($n=36$, 15 %) [2].

While 73 % of the injuries in Beijing occurred in the competition [3], injuries in Vancouver were evenly distributed between official training (54 %) and competition (46 %) ($P=.18$) [2], similar to the 2012 London Games [4]. However, in Vancouver, a specifically high proportion of training injuries was found for the three snowboard disciplines, freestyle cross skiing, short track, figure skating, skeleton, and biathlon. In these sports, three out of four injuries occurred outside of the competition (Table 2.3) [2].

2.3.6 Injury Severity

In Beijing, about half of the injuries were expected to prevent the athletes from further training or competition ($n=419$; 50 %). Physicians estimated that one third of the injuries would result in an absence from sport with up to 1 week [3]. In Vancouver, of the 287 injuries, 65 (23 %) were expected to result in a time-loss situation for the athlete. Of those with expected time loss, 11 injuries (17 %) had an estimated absence from training or competition of more than 1 week [2].

In London, while two thirds of the injuries were estimated to not result in any time loss from sport ($n=878$, 65 %), a total of 482 (35 %) injuries were expected to prevent the athlete from participating in competition or training. It was estimated that 246 (18 %) injuries would result in an absence from sports from 1 to 3 days, 62 (5 %) in an absence from 4 to 7 days, 105 (8 %) in an absence from 8 to 28 days, and 69 (5 %) in an absence for more than 28 days. Information on severity was missing on one injury (Table 2.4) [4].

A total of 174 injuries (13 %) entailed an estimated absence from training or competition of more than 1 week. These injuries were 10 shoulder, elbow, and knee dislocations (in hockey, football, judo, BMX, and weightlifting); 38 muscle strains, of which 24 were thigh strains (mostly in athletics); 24 fractures (mostly in team sports; all body locations) and six stress fractures (in running sports); eight Achilles, knee, and shoulder tendon ruptures (in athletics, badminton, handball, and basketball); 47 ligament sprains (across all joints and sports); and 15 knee sprains, including six ACL and one PCL ruptures (in fencing, handball, judo, wrestling, badminton, table tennis, tennis, and football). None of the six reported concussions (three in football, one each in water polo, athletics, and BMX) were classified as severe.

2.3.7 Incidence and Distribution of Illnesses (Registered in Vancouver and London Only)

Among 173 out of 2,567 athletes (7 %) in Vancouver, a total of 185 illnesses were reported, resulting in an incidence of 7.2 illnesses per 100 athletes [2]. Illnesses were reported from a variety of sports. In skeleton, figure and speed skating, curling, snowboard cross, and biathlon, every tenth athlete suffered from at least one illness. The majority of the illnesses ($n=113$, 63 %) affected the respiratory system, mostly observed in the ice skating and Nordic skiing disciplines (Table 2.2). As a consequence, the illness cause was most often classified as an infection ($n=111$, 64 %), affecting athletes in mainly the same sports as mentioned above. The most frequent diagnosis was upper respiratory tract infection (pharyngitis, sinusitis, tonsillitis) ($n=61$, 54 %).

In London, among the 10,568 athletes, a total of 758 illnesses were reported, resulting in an incidence of 7.2 illnesses per 100 athletes (Table 2.4). The incidence of illnesses was significantly higher in female compared to male athletes (8.6 versus 5.3 illnesses per 100 athletes, RR = 1.6, 95 % CI 1.4–1.9, $P<0.001$) [4].

Illnesses were reported from a variety of sports. In athletics, beach volleyball, football, sailing, synchronised swimming and taekwondo, more than every tenth athlete suffered from at least one illness. The peak in relative illness rates occurred in beach volleyball, where 19 % of the players suffered from an illness during the Games.

2.3.8 Affected System, Causes, Symptoms, and Severity of Illness

A total of 310 illnesses (41 %) in London affected the respiratory system, and these were most frequently observed in athletics, beach volleyball, football, swimming, and water polo (3–5 % of the athletes). In beach volleyball, 61 % of the illnesses were reported as respiratory infections. Concomitantly, infection was the most common cause of illness ($n=209$, 67 %), affecting athletes in mainly the same sports as mentioned above. Symptoms of pain were present in 209 (16 %) of the illnesses. However, for a third (248, 33 %) of all illnesses, symptoms were not reported. About one out of five illnesses ($n=145$, 19 %) were expected to result in absence from training or competition. Of these, nine illnesses were expected to result in an estimated time loss of more than 7 days (seven cases of stress, fatigue, and exhaustion, one upper respiratory tract infection, and one instance of abdominal pain) [4].

In the Vancouver Winter Games, a total of 113 illnesses (62.8 %) affected the respiratory system, mostly observed in the ice skating and Nordic skiing disciplines (Table 2.2). As a consequence, the illness cause was most often classified as an infection ($n=111$, 63.8 %), affecting athletes in mainly the same sports as mentioned above. The most commonly reported symptoms were pain ($n=50$, 27.9 %) and dyspnoea/cough ($n=38$, 21.2 %). The most frequent diagnosis was upper respiratory tract infection (pharyngitis, sinusitis, tonsillitis) ($n=61$, 54.0 %). A total of 24 (13.8 %) illnesses were caused by exercise-induced or environmental factors.

About a third of the illnesses (65 of 185; 35.1 %) were expected to result in absence from further training or competition. Of those, three illnesses were expected to result in an estimated time loss of 8–10 days (one endocrinological problem, two respiratory/dyspnoea–acute sinusitis and tonsillitis) [2].

2.4 Discussion

This chapter summarises the first three IOC surveillance projects on the injury and illness occurrence of athletes during the 2008 Beijing and 2012 London Summer and 2010 Vancouver Winter Olympic Games with all sports of the Games included. The principal findings were that at least 10–12 % of the athletes incurred an injury during the Olympic Games and 7 % of the athletes an illness. Although, variations in injury risk have been detected, it can be concluded that specifically some team sports (such as soccer, ice hockey, field hockey, handball, and basketball), martial art or

Table 2.4 Risk of injuries overall, injuries leading to time loss (≥1 day or >7 days of estimated absence), competition and training injuries, and illnesses overall in the Olympic sports during the 2012 London Games

					Injuries		
Olympic sport	Athletes (*n*)	All	≥1 day	>7 days	Competition	Training	All illnesses
Archery	128	2 (1.6)	0 (0.0)	0 (0.0)	0 (0.0)	1 (100.0)	10 (7.8)
Athletics	2,079	368 (17.7)	145 (7.0)	59 (2.8)	133 (39.5)	204 (60.5)	219 (10.5)
Aquatics							
Diving	136	11 (8.1)	5 (3.7)	2 (1.5)	2 (25.0)	8 (75.0)	7 (5.1)
Swimming	931	50 (5.4)	7 (0.8)	1 (0.1)	13 (31.0)	29 (67.0)	68 (7.3)
Synchronised swimming	104	14 (13.5)	4 (3.8)	0 (0.0)	2 (20.0)	8 (80.0)	13 (12.5)
Water polo	260	34 (13.1)	13 (5.0)	0 (0.0)	26 (78.8)	7 (21.2)	21 (8.1)
Badminton	164	26 (15.9)	7 (4.3)	4 (2.4)	11 (47.8)	12 (52.3)	5 (3.0)
Basketball	287	32 (11.1)	10 (3.5)	7 (2.4)	21 (75.0)	7 (25.0)	9 (3.1)
Beach volleyball	96	12 (12.5)	2 (2.1)	0 (0.0)	6 (54.5)	5 (45.5)	18 (18.8)
Boxing	283	26 (9.2)	9 (3.2)	1 (0.4)	16 (72.7)	6 (27.3)	18 (6.4)
Canoe slalom	83	2 (2.4)	1 (1.2)	0 (0.0)	0 (0.0)	2 (100.0)	4 (4.8)
Canoe sprint	249	7 (2.8)	1 (0.4)	0 (0.0)	3 (50.0)	3 (50.0)	14 (5.6)
Cycling							
BMX	48	15 (31.3)	5 (10.4)	2 (4.2)	11 (73.3)	4 (26.7)	2 (4.2)
MTB	76	16 (21.1)	8 (10.5)	2 (2.6)	5 (31.3)	11 (68.7)	5 (6.6)
Road	210	19 (9.0)	7 (3.3)	2 (0.9)	14 (73.7)	5 (26.3)	7 (3.3)
Track	167	5 (3.0)	3 (1.8)	0 (0.0)	1 (20.0)	4 (80.0)	16 (9.6)
Equestrian	199	9 (4.5)	4 (2.0)	2 (1.0)	6 (100.0)	0 (0.0)	11 (5.5)
Fencing	246	23 (9.3)	10 (4.1)	2 (0.8)	10 (45.5)	12 (54.5)	13 (5.3)
Football	509	179 (35.2)	67 (13.2)	11 (2.2)	132 (74.2)	46 (25.8)	62 (12.2)
Gymnastics							
Artistic	195	15 (7.7)	8 (4.1)	4 (2.1)	8 (66.7)	4 (33.3)	5 (2.6)
Rhythmic	96	8 (8.3)	1 (1.0)	0 (0.0)	1 (16.7)	5 (83.3)	1 (1.0)
Trampoline	32	2 (6.3)	0 (0.0)	0 (0.0)	0 (0.0)	2 (100.0)	1 (3.1)
Handball	349	77 (22.1)	32 (9.2)	16 (4.6)	55 (75.3)	18 (24.7)	17 (4.9)
Hockey	388	66 (17.0)	25 (6.4)	10 (2.6)	44 (71.0)	18 (29.0)	29 (7.5)
Judo	383	47 (12.3)	22 (5.7)	12 (3.1)	26 (68.4)	12 (31.6)	16 (4.2)
Modern pentathlon	72	6 (8.3)	2 (2.8)	0 (0.0)	3 (60.0)	2 (40.0)	1 (1.4)
Rowing	549	18 (3.3)	2 (0.4)	0 (0.0)	7 (53.8)	6 (46.2)	40 (7.3)
Sailing	380	55 (14.5)	3 (0.8)	1 (0.3)	30 (62.5)	18 (37.5)	38 (10.0)
Shooting	390	15 (3.8)	4 (1.0)	0 (0.0)	1 (7.1)	13 (92.9)	17 (4.4)
Table tennis	174	11 (6.3)	7 (4.0)	2 (1.1)	7 (70.0)	3 (30.0)	12 (6.9)
Taekwondo	128	50 (39.1)	16 (12.5)	7 (5.5)	16 (33.3)	32 (66.7)	14 (10.9)
Tennis	184	21 (11.4)	7 (3.8)	4 (2.2)	14 (66.7)	7 (33.3)	4 (2.2)
Triathlon	110	16 (14.5)	8 (7.3)	3 (2.7)	11 (73.3)	4 (26.7)	7 (6.4)
Volleyball	288	20 (6.9)	7 (2.4)	3 (1.0)	11 (55.0)	9 (45.0)	8 (2.8)
Weightlifting	252	44 (17.5)	19 (7.5)	11 (4.4)	18 (45.0)	22 (55.0)	10 (4.0)
Wrestling	343	41 (12.0)	11 (3.2)	6 (1.7)	20 (62.5)	12 (37.5)	16 (4.7)
Total	10,568	1,361 (12.9)	482 (4.6)	174 (1.6)	684[a] (54.9)	561[a] (45.1)	758 (7.2)

Values are numbers (percentages) of injured or ill athletes, unless otherwise indicated
[a]Information on training/competition is missing in 116 injuries

weight class sports (such as taekwondo, boxing, and weightlifting), and speed sports (such as bob and the alpine skiing and snowboard disciplines) have a relatively high injury risk. Upper respiratory tract infections, the major cause of reported illnesses in Vancouver, were specifically suffered by Nordic skiing and skating athletes.

2.4.1 Incidence and Distribution, Type, and Cause of Injuries

In Vancouver, the injury incidence was with 111.8 injuries per 1,000 athletes slightly higher than reported from the Summer Olympics in Beijing 2008 (96.1 injuries per 1,000 athletes) [2].

In London, we recorded a total of 1,361 injuries, equalling an overall injury rate of 12.9 injuries per 100 registered athletes [4]. This observed difference is most likely due to the differences in the sports themselves, since both IOC surveillance projects were conducted by the same research group using the same methodology to obtain data [18].

As illustrated for winter sports by Torjussen and Bahr [24] and for summer sports by Junge et al. [18], choosing the appropriate method to report the risk of injury in sports is a challenge if the aim is to compare the risk between different sports or disciplines where exposure may differ considerably. During major sport events, such as the Olympics, athletes can have, for example, 15 jumps in a high jump competition; 8 throws in a javelin competition; 1 ski run in a 50 km cross-country race; 5 runs in snowboard cross; several matches in ice hockey, soccer, or basketball; and only 1–4 starts in a 100 m sprint competition. Thus, as an alternative to relative injury risk where the risk is expressed as rate corrected for exposure, e.g. injuries per run/matches, using the absolute injury risk is highly relevant for the present summary, where injuries and illnesses are expressed as the total number of injuries/illnesses per registered athletes for each sport/discipline.

Having this in mind, athletics, soccer, and ice hockey caused the greatest portion of injuries in the 2008 and 2012 Summer [3, 4] and 2010 Winter Olympics [2]. Out of all injuries registered during both Games, 18 % of injuries occurred in athletics, 12 % in soccer, and 6 % in ice hockey. However, this does not mean that athletes in these sports are at the greatest risk of injury. The explanation is that these sports have a great number of competing participants.

The picture was slightly different when the number of injuries was calculated in relation to the number of participating athletes. In Beijing and London, the risk of sustaining an injury was highest for soccer, taekwondo, field hockey, BMX, mountain bike, handball, weightlifting, boxing, triathlon, hockey, badminton, and athletics [3, 4]. These findings are in accordance with the literature [12, 25, 26] where systematic surveillance data are available. In Vancouver, freestyle and snowboard cross, bobsleigh, ice hockey, short track, and alpine skiing were the sports with the highest injury risk [2].

Compared to high-risk summer sports, where in, for instance, team sports many of the injuries are expected to result from player-to-player contact, many winter sports are characterised by a high speed. Injury risk has also been documented to be high in freestyle and snowboard cross. To position themselves in the front through their heats while competing against three other skiers/riders, athletes had to pass several challenges, e.g. turns, jumps, and waves [20]. Combined with the speed component, competing in heats may promote a risk-taking attitude for the athletes. In addition, body contact within the rules of the sport occurs and may force the athlete to unanticipated reaction, loss of control, and probably higher-risk situations [20, 22].

On the other hand, the lowest injury risk during the Beijing and London Olympics was observed for water sports such as sailing, archery, canoeing/kayaking, rowing, synchronised swimming, diving, and swimming [3, 4]. The low injury risk for athletes competing in the Nordic skiing disciplines compared to alpine, freestyle, and snowboard athletes is not surprising as they are not exposed to high speed on icy surfaces with minimal protection [27].

In line with a previous report [19], the knee and the head were the most frequent body parts injured among alpine and freestyle skiers and snowboarders. The same picture was seen among summer sport athletes, too, where thigh, knee, lower leg, ankle, and head injuries were most commonly injured [3, 4]. Also, a concern also should be that every fifth registered injury in the Winter Olympic Games affected the head, neck, and cervical spine, mainly diagnosed as abrasion, skin lesion, contusion, fracture, or concussion. In many cases, head and knee injuries result in long absence from training and competition, and the prevention of concussions and severe knee ligament sprains, including anterior cruciate ligament ruptures, is of significant importance. In Vancouver, a total of 20 concussions, constituting 7 % of all participating athletes, were diagnosed [2]. These figures are twice as high as reported from the Summer Olympic Games [3, 4]. In a national cohort, Emery et al. [28] found a high rate of concussions among young elite ice hockey players. To sum up, except for skiing [21, 22, 27], snowboarding and freestyle skiing [20, 23], and ice hockey [28, 29], there is little data available on winter sports regarding elite athletes' injury risk.

2.4.2 Incidence and Distribution, Type, and Cause of Illnesses (Registered in London and Vancouver Only)

The illness incidence in Vancouver and London was 7 illnesses per 100 athletes [2, 4]. The high incidence of respiratory infections mirrors data from other elite sport events, and these findings are consistent with data from athletics (7 %) [15], aquatics (7 %) [16], and football (12 %) [7]. Almost two thirds of the illnesses affected the respiratory system (62 %) caused by infections (64 %), which is a higher rate than reported in swimming (respiratory system 50 %, infection 49 %) [30].

Predominant risk factors are mechanical and dehydration stresses generated within the airways and the level of airborne pollutants, irritants, and allergens inhaled by the athlete under high ventilatory exercise conditions [31]. It has earlier been reported that airway hyperresponsiveness/asthma is the most common chronic medical condition experienced by both summer and winter Olympic athletes [30].

Improving the education of athletes and their entourage on infectious disease prevention strategies as well as the provision of more hand sanitisation stations at training and competitive venues should lead to a decrease in this problem.

2.5 Practical Implications and Further Research

Before preventive measures can be suggested, risk factors and mechanisms need to be characterised [5]. For example, whether an injury in, for example, freestyle cross occurs in a landing after a jump, resembling the boot-induced anterior drawer mechanism with deep knee flexion, or is due to collisions with other skiers or the skier coming out of balance by fighting for a better positioning in the course needs to be investigated [32]. In addition, slope, snow and weather conditions, the athlete's speed, and equipment may play an active role in the inciting event of an injury [21, 22]. A recent report describing situations leading to ACL injuries in World Cup alpine skiing revealed that individual technical errors and inappropriate tactical choices were the dominant risk factors [22]. In another systematic video analysis of 19 injury cases among elite snowboard riders, jumping appeared to be the most challenging situation in snowboard cross, where a technical error at takeoff was the primary cause of the injuries [20]. These studies indicate that close evaluations are necessary on course design and setting, race conditions, visibility and speed, and other technically difficult obstacles, such as height and distance between jumps [20, 22]. Future research should also aim to explore the physiological and psychological requirements for these athletes [23]. Also, by using video analysis and a model-based image-matching technique, detailed information on joint kinematics can be obtained from uncalibrated injury video recordings [33]. This approach will help to better understand injury mechanisms. The IOC research group has analysed the most serious injuries in Vancouver in an effort to improve the knowledge on injury risk factors and mechanisms in high-risk sports.

As the cause of injury varied substantially between sports, successful preventive strategies need to be tailored to the respective sport and athlete at risk [2, 34]. Based on the experiences from the Vancouver Olympics, where more than half of the injuries in the bobsleigh run and skiing/snowboarding slopes incurred as a result of contact with a stationary object, preventive measures need to address the importance of creating safe sports arenas (optimal preparation of the skating ring, bobsleigh run, freestyle and snowboard courses/pipes). In addition, the high proportion of training injuries in the skiing and snowboarding speed disciplines may suggest additional training runs and optimising training facilities. The effect of these potential measures to reduce injury risk has to be monitored in the upcoming Games.

The IOC and other major international sports federations, such as the International Football Association (FIFA) [7], the International Aquatic Federation (FINA) [16], and the International Association of Athletics Federations (IAAF) [13, 15], have extended their injury surveillance in a second step to also include illness monitoring. In addition, the IOC is developing a periodic health exam (PHE) system, which may be offered to the NOCs prior to future Olympic Games. This should improve pre-Game knowledge both on injuries and illnesses and will help NOCs to maximise the health protection of their elite athletes [1].

In London 2012, the IOC with the NOC and IF continued running the injury and illness surveillance system. As in Beijing and Vancouver, the monitoring of the athletes' health enables researchers and clinicians to follow the injury and disease trends in the various sports and continue the premier goal of the IOC: to protect the health of the athlete. The message from this and other long-term projects initiated by the IOC and the IFs is that we need to monitor the development of injury and illness rates over several years to identify potential risk factor and mechanisms for injury and illnesses in disciplines and sports. By acquiring new knowledge on injury trends, we can optimise and target future research on injury risk factors, mechanisms, and, finally, prevention. The key to a meaningful study of epidemiology lies in a well-organised procedure for data collection with coordinated efforts from sports medicine professionals, coaches, and athletes, combined with systematic subsequent analyses.

Conclusion

The present data collection procedures were accepted by the medical staff of the National Olympic Committees as demonstrated by the high response rates of returned injury and illness forms at the various events. At least 10–12 % of the athletes incurred an injury during the 2008 and 2012 Summer or 2010 Winter Games, and 7 % of the winter sport athletes suffered at least from one illness occurrence. The incidence of injuries and illnesses varied substantially between sports. In the future, risk factor and video analyses of injury mechanisms in high-risk Olympic sports are essential to better direct injury prevention strategies. Pre-Game monitoring will be an essential part of an athlete's medical support.

Acknowledgements The research group wishes to acknowledge sincere gratefulness to the President of the IOC Medical Commission, Professor Arne Ljungqvist, and to NOCs and IFs for their strong support for these studies. We gratefully acknowledge the International Olympic Committee (IOC) for funding these studies. We also like to give credit to F-MARC for initiating the systematic assessment of the injuries from 1998.

References

1. Ljungqvist A, Jenoure P, Engebretsen L, Alonso JM, Bahr R, Clough A et al (2009) The International Olympic Committee (IOC) Consensus Statement on periodic health evaluation of elite athletes March 2009. Br J Sports Med 43(9):631–643
2. Engebretsen L, Steffen K, Alonso JM, Aubry M, Dvorak J, Junge A et al (2010) Sports injuries and illnesses during the Winter Olympic Games 2010. Br J Sports Med 44(11):772–780
3. Junge A, Engebretsen L, Mountjoy ML, Alonso JM, Renstrom PA, Aubry MJ et al (2009) Sports injuries during the Summer Olympic Games 2008. Am J Sports Med 37(11):2165–2172
4. Engebretsen L, Soligard T, Steffen K, Alonso JM, Aubry M, Budgett R et al (2013) Sports injuries and illnesses during the London Summer Olympic Games 2012. Br J Sports Med 47(7): 407–414
5. van Mechelen W, Hlobil H, Kemper HC (1992) Incidence, severity, aetiology and prevention of sports injuries. A review of concepts. Sports Med 14(2):82–99
6. Dvorak J, Junge A, Grimm K, Kirkendall DT (2007) Medical report from the 2006 FIFA World Cup GermanyTM. Br J Sports Med 41(9):578–581
7. Dvorak J, Junge A, Derman W, Schwellnus M (2011) Injuries and illnesses of football players during the 2010 FIFA World Cup. Br J Sports Med 45(8):526–530
8. Junge A, Dvorak J, Graf-Baumann T, Peterson L (2004) Football injuries during FIFA Tournaments and the Olympic Games, 1998–2001: development and implementation of an injury-reporting system. Am J Sports Med 32(1 Suppl):80S–89S
9. Junge A, Langevoort G, Pipe A, Peytavin A, Wong F, Mountjoy M et al (2006) Injuries in team sport tournaments during the 2004 Olympic Games. Am J Sports Med 34(4):565–576
10. Junge A, Dvorak J (2010) Injury risk of playing football in Futsal World Cups. Br J Sports Med 44(15):1089–1092
11. Junge A, Dvorak J, Graf-Baumann T (2004) Football injuries during the World Cup 2002. Am J Sports Med 32(1 Suppl):23S–27S
12. Junge A, Dvorak J (2013) Injury surveillance in the World Football Tournaments 1998–2012. Br J Sports Med 47(12):782–788
13. Alonso JM, Tscholl PM, Engebretsen L, Mountjoy M, Dvorak J, Junge A (2010) Occurrence of injuries and illnesses during the 2009 IAAF World Athletics Championships. Br J Sports Med 44(15):1100–1105
14. Alonso JM, Junge A, Renstrom P, Engebretsen L, Mountjoy M, Dvorak J (2009) Sports injuries surveillance during the 2007 IAAF World Athletics Championships. Clin J Sport Med 19(1):26–32
15. Alonso JM, Edouard P, Fischetto G, Adams B, Depiesse F, Mountjoy M (2012) Determination of future prevention strategies in elite track and field: analysis of Daegu 2011 IAAF Championships injuries and illnesses surveillance. Br J Sports Med 46(7):505–514
16. Mountjoy M, Junge A, Alonso JM, Engebretsen L, Dragan I, Gerrard D et al (2010) Sports injuries and illnesses in the 2009 FINA World Championships (Aquatics). Br J Sports Med 44(7): 522–527
17. Bahr R, Reeser JC (2003) Injuries among world-class professional beach volleyball players. The Federation Internationale de Volleyball beach volleyball injury study. Am J Sports Med 31(1):119–125
18. Junge A, Engebretsen L, Alonso JM, Renström P, Mountjoy M, Aubry M et al (2008) Injury surveillance in multi-sport events: the International Olympic Committee approach. Br J Sports Med 42(6):413–421
19. Flørenes TW, Nordsletten L, Heir S, Bahr R (2011) Recording injuries among World Cup skiers and snowboarders: a methodological study. Scand J Med Sci Sports 21(2):196–205
20. Bakken A, Bere T, Bahr R, Kristianslund E, Nordsletten L (2011) Mechanisms of injuries in World Cup Snowboard Cross: a systematic video analysis of 19 cases. Br J Sports Med 45(16):1315–1322
21. Bere T, Flørenes TW, Krosshaug T, Koga H, Nordsletten L, Irving C et al (2011) Mechanisms of anterior cruciate ligament injury in World Cup alpine skiing: a systematic video analysis of 20 cases. Am J Sports Med 39(7):1421–1429
22. Bere T, Flørenes TW, Krosshaug T, Nordsletten L, Bahr R (2011) Events leading to anterior cruciate ligament injury in World Cup Alpine Skiing: a systematic video analysis of 20 cases. Br J Sports Med 45(16):1294–1302
23. Steenstrup SE, Bere T, Flørenes TW, Bahr R, Nordsletten L (2011) Injury incidence in qualification runs versus final runs in FIS World Cup snowboard cross and ski cross. Br J Sports Med 45(16):1310–1314
24. Torjussen J, Bahr R (2006) Injuries among elite snowboarders (FIS Snowboard World Cup). Br J Sports Med 40(3):230–234
25. Feddermann-Demont N, Junge A, Edouard P, Branco P, Alonso JM (2014) Injuries in 13 international Athletics championships between 2007–2012. Br J Sports Med 48(7):513–522
26. Møller M, Attermann J, Myklebust G, Wedderkopp N (2012) Injury risk in Danish youth and senior elite handball using a new SMS text messages approach. Br J Sports Med 46(7):531–537
27. Flørenes TW, Bere T, Nordsletten L, Heir S, Bahr R (2009) Injuries among male and female World Cup alpine skiers. Br J Sports Med 43(13):973–978
28. Emery CA, Kang J, Shrier I, Goulet C, Hagel BE, Benson BW et al (2010) Risk of injury associated with body checking among youth ice hockey players. JAMA 303(22):2265–2272
29. McKay CD, Tufts RJ, Shaffer B, Meeuwisse WH (2014) The epidemiology of professional ice hockey injuries: a prospective report of six NHL seasons. Br J Sports Med 48(1):57–62
30. Fitch KD (2012) An overview of asthma and airway hyper-responsiveness in Olympic athletes. Br J Sports Med 46(6):413–416
31. Fitch K, Anderson S (2012) Intense exercise and airway hyperresponsiveness/asthma: importance of environmental factors. Br J Sports Med 46(6):379–380
32. Randjelovic S, Heir S, Nordsletten L, Bere T, Bahr R (2014) Injury situations in Freestyle Ski Cross (SX): a video analysis of 33 cases. Br J Sports Med 48(1):29–35
33. Krosshaug T, Slauterbeck JR, Engebretsen L, Bahr R (2007) Biomechanical analysis of anterior cruciate ligament injury mechanisms: three-dimensional motion reconstruction from video sequences. Scand J Med Sci Sports 17(5):508–519
34. Steffen K, Andersen TE, Krosshaug T, van Mechelen W, Myklebust G, Verhagen E et al (2010) ECSS position statement 2009: prevention of acute sports injuries. Eur J Sport Sci 10(4):223–236

Tennis: Epidemiology and Injury Mechanism

3

Giovanni Di Giacomo, Nicola de Gasperis, and Alberto Costantini

3.1 Introduction

Tennis injuries have been reported throughout all regions of the body with more common areas being the spine, the ankle and the shoulder (Fig 3.1). Tennis injuries can occur as a consequence of a trauma (acute injuries, more common in the lower extremity), but most of the injuries in tennis can be defined as overuse injuries (chronic injuries, more common in the upper extremity and trunk) coming from the repetitive microtrauma inherent in the sport [1, 2]. Upper extremity injuries were most frequently located in the elbow and the shoulder regions, with tendon injuries of the shoulder and tennis elbow (humeral epicondylitis) as most frequent injuries. Lower extremity injuries were most frequently located in the ankle and the knee regions, with ankle sprain and patellar tendinosis as most frequent injuries. Usually upper extremity injuries are associated with kinetic chain dysfunction, scapular dyskinesis and GIRD [3]. The repetitive stressors and loading sequences in tennis create muscular imbalances specific to the sport that requires preventative interventions believed to lower injury risk. This chapter will show an overview of the epidemiology and the mechanism of the most common injuries in tennis players.

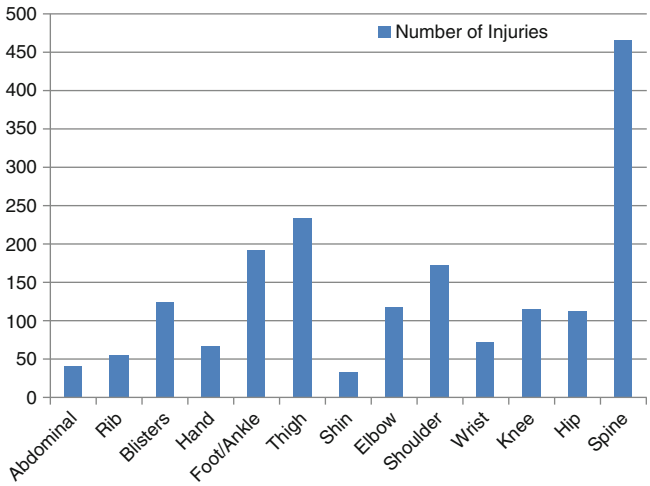


Fig 3.1 ATP World Tour 2013 injuries

3.2 Epidemiology and Mechanism of Upper Limb Injuries in Tennis Players

3.2.1 The Shoulder

The upper extremity is one of the most frequently injured regions in tennis players. In the 2013 ATP World Tour, shoulder injuries (10 % of all injuries) were in fourth position after spine (26 %), thigh muscles (13 %) and foot/ankle (11 %) injuries. Sallis et al. found that there is no significant difference in injury rate between men and women [4]. The most common diagnosis in tennis players with shoulder pain are 'impingement' and 'rotator cuff and long head of the biceps tendonitis', frequently due to 'overuse syndrome' (chronic injuries). This syndrome could result from kinetic chain dysfunction, scapular dyskinesis and glenohumeral internal rotation deficit (GIRD). In case of kinetic chain dysfunction and scapular dyskinesis, the shoulder joint becomes the victim and not the culprit of a dysfunction, eventually resulting in an anatomical injury with clinical findings affecting the *shoulder girdle*. This kind of approach might explain the failure of some surgical ligament and tendon repair techniques, too often attributed to failed materials (anchors, suture, bio- and not reabsorbable material) or local biological factors (vascularization, fatty infiltration, etc.) forgetting that these anatomical lesions are often the end point of a failure in the kinetic chain that, if not correct, may inevitably reproduce the lesion over time, even after surgery. The dynamic scapulothoracic stability and the importance of the *core stability* give the ideal hint to understand those mechanisms that when

G. Di Giacomo, MD (✉) • N. de Gasperis, MD
A. Costantini, MD
Orthopaedic Department, Concordia Hospital for Special Surgery, Via Delle Sette Chiese 90, Rome 00145, Italy
e-mail: concordia@iol.it

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_3,

altered can lead to *shoulder dysfunction*. The shoulder is a complex mechanical structure containing several joints connecting the humerus, the scapula, the clavicle and the sternum. The scapula slides over the dorsal part of the thorax; it can glide over the so-called scapulothoracic gliding plane. It is a closed chain mechanism. The relation between the rotations of the humerus and scapula is commonly referred to as the *scapulohumeral rhythm*. The scapular motion strongly affects the *mechanical energy* delivered by the muscles and the *metabolic cost* required to obtain the desired force. At the same time the scapula has different roles being a functional part of the glenohumeral joint, retracting and protracting along the thoracic wall and elevating the acromion. It is a site for muscle attachment and a link in the proximal to distal sequencing of velocity, energy and force that allows the most appropriate shoulder function [5]. The *core* is where the centre of gravity is located and where movement begins. An efficient core allows for maintenance of the physiological length–tension relationship of functional agonists and antagonists and for normal force couple in the lumbo-pelvic hip complex. The musculoskeletal core of the body includes the spine, the hips, the pelvis, the proximal lower limb and abdominal structures; muscles of the trunk and pelvis are responsible for the maintenance of stability of the spine and are critical for the transfer of energy from large to small body parts during many work/sports activities. The roof of the core muscle structures is the diaphragm. At the opposite end of the trunk component of the core muscles are the pelvic floor muscles. Core muscles have large cross-sectional areas and generate a great amount of force and power for athletic activities. The thoracolumbar fascia is an important structure that connects the lower limbs (via the gluteus maximus) to the upper limbs (via the latissimus dorsi). Function, the end result of the kinetic chain, can be defined as *optimal anatomy acted upon by physiological muscle activations to produce optimal biomechanical forces and motions.* Core stability is essential for the maximum efficiency of the shoulder function. A functional definition of '*core stability*' *is the ability to control the trunk over the pelvis to allow the coordinated sequenced activation of body part to produce, to transfer and to control force and motion to the terminal segments in integrated body activities, to obtain the desired work/athletic task* [6]. This definition implies patterned sequences for force generation and transfer, proximal stability for distal mobility and control in three dimensions. Core muscle activation is used to generate rotational torques around the spine and provides stiffness to the entire central mass, making a rigid cylinder that confers a long lever arm around which rotation can occur and against which muscles can be stabilized as they contract [7]. One of the most important abnormalities in scapular biomechanics is actually the loss of the 'link function' in the kinetic chain; if the scapula does become deficient in motion or position, transmission of the large generated forces from the lower extremity to the upper extremity is impaired. This creates a deficiency in resultant maximum force that can be delivered to the hand or creates a situation of 'catch up' in which the more distal links have to work more actively to compensate for the loss of the proximally generated force. This can impair the function of the distal links because they do not have the size, the muscle cross section area or the time to efficiently develop these larger forces. Kibler calculations have shown that a 20 % decrease in kinetic energy delivered from the hip and trunk to the arm necessitates an 80 % increase in mass or a 34 % increase in rotational velocity at the shoulder to deliver the same amount of resultant force to the hand [5]. This required adaptation can cause overload problems with repeated use. In condition of sport-related stress, regulatory imbalance might result both in typical reaction patterns and individual response specificity; this can explain the anatomo-pathological difference of several lesions (extension, site, degrees of retraction, etc.), and it justifies those clinical pictures that even if triggered by similar lesions appear at different times and with different clinical features.

Repetitive concentric and eccentric demands on the rotator cuff and hypermobility and excessive laxity of the glenohumeral joint could lead to scapulothoracic muscular fatigue altering the normal shoulder biomechanics. Fatigue affects sensation of joint movement, decreases athletic performance and increases fatigue-related shoulder dysfunction. The muscular imbalance during the deceleration phase transfers distraction forces to the posterior capsule that becomes tight and leads to an internal rotation reduction (glenohumeral internal rotation deficit or GIRD). Posterior capsule tightness may be forcing the humeral head forward, causing mechanical impingement and a loss of range of motion as a result of the avoidance of painful movements. Although the factors contributing to secondary shoulder impingement are multiple, the posterior capsule tightness is thought to alter shoulder kinematics, with superior translation of the humeral head during flexion such that the rotator cuff is compromised by the overlying coracoacromial arch. Glenohumeral joint tightness can also create abnormal biomechanics of the scapula. Posterior shoulder inflexibility, due to capsular or muscular tightness (infraspinatus thixotropy), affects the smooth motion of the glenohumeral joint and creates a *wind-up* effect so that the glenoid and scapula actually get pulled in a forward and inferior direction by the moving and rotating arm [8]. This can create an excessive amount of protraction of the scapula on the thorax as the arm continues into the horizontally adducted position in *follow through*. Because of the geometry of the upper aspect of the thorax, the more the scapula is protracted in *follow through*, the farther it and its acromion move anteriorly and inferiorly around the thorax. In all cases with suspected impingement, a careful examination of both passive and active motion of the shoulders in all

planes is needed. In those patients with limited internal rotation and flexion, a therapy programme should be directed at improving these motion planes.

The abnormal scapular biomechanics, occurring as a result of dysfunction, create an abnormal scapular position that decreases normal shoulder function and exposes the shoulder to injury if prevention strategies are not applied. The most common shoulder lesion in tennis players are 'superior labral tears (SLAP)' and 'rotator cuff tears'. Posterior or posterior superior shoulder pain is felt without mechanical symptoms usually described as occurring during the late cocking and early acceleration phases of the throwing cycle. This is due to posterior superior glenohumeral instability directed by the posterior inferior capsular contracture as the shoulder abducts and rotates. The posterior superior shift of the glenohumeral contact and rotational point creates strain on the posterior superior labral glenoid interface as well as allows for increased external humeral rotation which brings the undersurface of the posterior superior rotator cuff in contact with the posterior superior glenoid margin resulting in the early symptoms of *internal impingement*. The SLAP event occurs when the posterior superior labrum and biceps anchor fail in tension from their glenoid attachments secondary to the capsular contracture mediated by posterior superior glenohumeral instability. Once the SLAP event has occurred, the thrower promptly develops mechanical symptoms in late cocking and early acceleration phases. Once mechanical symptoms appear, the problem becomes surgical and will not be improved or solved conservatively. Conversely, prior to the *slap event*, the symptomatic throwing shoulder can usually be successfully treated by a series of focused posterior inferior capsular stretches to eliminate the contracture and strength exercises to rehab any concomitant rotator cuff- and scapular stabilizer-deconditioned musculature. After the development of mechanical symptoms of the SLAP lesion, if the thrower continues to throw, subacromial and rotator cuff symptoms ensue due to contracture-mediated increasing posterior superior glenohumeral instability with secondary subacromial space restriction and increasing *internal impingement* events. This can lead the athlete, in the course of time, to a rotator cuff tear that have to be fixed arthroscopically.

3.2.2 The Elbow

Injuries to the elbow region in elite tennis players primarily involve repetitive overuse and centre on the tendonous structures inserting at the medial and lateral humeral epicondyle [9]. The reported injury rates for tennis elbow are quite high, with percentages ranging from 37 to 57 % in elite and recreational players. Nirschl and Sobel also show higher rates of incidence in elite players on the medial side of the elbow from overload on the serve and forehand strokes compared with higher rates of lateral humeral epicondylitis in lower-level recreational players from the overload on the backhand ground stroke [10]. The most frequent injuries in professional tennis player occur on the medial side of the elbow such as medial epicondylitis and medial instability due to ulnar collateral ligament lesion (UCL lesion), a result of chronic overuse. When acute injuries do occur, they are the result of a traumatic event. With increased training, players may experience muscle fatigue. Due to this reason changes in mechanics placing could increase strain on the UCL. Muscular fatigue may lead to a decreased shoulder abduction angle that has been shown to place greater force on the medial elbow [11]. Chronic overuse leads to microtrauma and attenuation of the ulnar collateral ligament. Traction spurs and calcification on the UCL are common radiographic signs of repeated injury on the medial side of the elbow. Progressive attenuation of the ligament ultimately results in an incompetent ligament and forms part of the continuum leading to valgus extension overload that ends with a medial instability. Players typically do not experience limitations until trying to serve beyond 75 % of the full potential. Pain is most commonly reported during the late cocking and early acceleration phases of serving. Players with UCL insufficiency could have symptoms involving the ulnar nerve, resulting of traction, compression or irritation from the surrounding inflammation in the setting of an incompetent UCL. The exercises recommended for prevention of elbow injury focus on increasing the strength and particularly the muscular endurance of the wrist and forearm musculature. It is important to note that contrary to common beliefs among coaches, players and even medical professionals, power generation does not come from the wrist and forearm in properly executed tennis strokes. Instead, the summation of forces from the entire body or kinetic chain produces the power that is transferred through the wrist, forearm and ultimately to the racquet head to generate power [12, 13]. Reliance on the forearm musculature for power generation is a common clinical hypothesis for the origin of elbow pathology in tennis players due to nonoptimal contributions from other segments of the kinetic chain and poor overall stroke biomechanics and whole body fitness [14].

3.3 The Spine

3.3.1 The Cervical Spine

Although often neglected by sports therapists, the cervical spine is one of the most complicated joint segments of the whole human body. The cervical joints help to sustain the head and ensure a large range of motion. Professional tennis players are frequently affected by cervical spine dysfunction

caused by the intense stresses applied to this area during play, especially during the serve. During the *initial wind-up* and *early cocking phase* of the serve, the risk of trauma is statistically less frequent because of the slow controlled movements entailed. On the other hand, during the *late cocking phase*, the tennis player's neck is subject to torsional stresses when in extension. When a right-handed player is in a position of cervical extension and rotation towards the right, the diameter of the intervertebral foramen, through which nerve roots pass, will be narrowed. The rotational stresses on this area predispose to trauma of the cervical facet joints, the surrounding nerves and soft tissues (cervical facet syndrome). This situation could compromise effective shoulder movements on the side of the dominant limb. Pre-existing cervical spine disorders would be exacerbated by the typical movements of the serve and predispose to secondary trauma. The compression and torsion exerted on the cervical and upper thoracic spine during the *late cocking* phase continues into the subsequent '*acceleration*' phase. During the final phase of the serve, the so-called follow through, energy is absorbed and the athlete decelerates the arm–racquet complex. Finally it is not uncommon to observe that impaired scapular girdle function is due to joint, muscle or neurological damage of the cervical spine [15].

3.3.2 The Lower Back

The motions required in tennis include repeated flexion, extension, lateral flexion and rotation of the spine, and intense tennis play is generally held to be a risk factor for low back pain [16]. One of the motions that can particularly stress the spine in the elite player is the combined movements of extension, lateral flexion and rotation that are inherent in the cocking or loading phase of the tennis serve. These combined repetitive motions have been shown to stress the lumbar spine and are believed to be a causative factor for spondylolysis (fracture of a specific region of the vertebrae termed the pars interarticularis) and spondylolisthesis (pars fracture with graded anterior migration of the vertebral body) identified in many athletes in sports with repetitive extension-based movement requirements [17–19]. Tennis is no exception. Alyas et al. studied the spine of 33 asymptomatic, elite, adolescent tennis players (mean age 17.3 ± 1.7 years) [17]. Five players (15.2 %) had a normal magnetic resonance imaging examination, and 28 (84.8 %) had an abnormal examination. Nine players showed 10 pars lesions (3 complete fractures), and 23 patients showed signs of early facet arthropathy. This study shows the impact of repetitive loading on the adolescent spine even in asymptomatic elite-level players. Tennis players can suffer from lumbar disc disease, sciatica and facet syndromes secondary to the excessive repetitive loading as well [16]. To combat the effects of this loading, preventative conditioning strategies for tennis players include extensive core stability training. Similar to research on the shoulder, isokinetic profiling studies of elite tennis players show characteristic muscle development likely induced from the sport-specific demands imparted to the tennis players' body [20, 21]. Roetert et al. tested elite junior players and found the trunk extension-to-flexion ratio to be <100, indicating greater actual strength in the abdominals and trunk flexors compared with the back extensors in these elite players [21]. Research on normal populations (nonathletes and non-tennis players) typically produces ratios >100 in the extension-to-flexion ratio whereby the low back extensor strength exceeds trunk flexor strength [22]. Ellenbecker and Roetert tested elite junior players and found symmetrical torso rotation strength using an isokinetic dynamometer, indicating that healthy uninjured players should have symmetrical strength development in the directions of both left and right rotations [23]. These data provide insight into the training strategy for core stabilization in tennis players. Emphasis on both the flexors and extensors must be given to ensure that balanced extensor and flexor muscular development occurs as well as an emphasis on rotational exercise due to the predominance of trunk rotation inherent in all tennis strokes.

3.4 Epidemiology and Mechanism of Lower Limb Injuries in Tennis Players

3.4.1 The Ankle

A sprained or twisted ankle is one of the most common injuries in tennis players, and it usually occurs as a consequence of a trauma (acute injuries) [1, 2]. The players have to run from one side of the court to the other, changing direction rapidly to strike the ball with their racquets. These sudden sideways movements that are required during the play can cause the ankle to twist, particularly if the surface is slippery or the player is fatigued. An inversion sprain is the most common sprained ankle injury and happens when the sole of the foot twists inwards, compressing the ligaments on the inside, such as the medial (deltoid) ligament, pulling or overextending the outer ligaments of the ankle. A twisted ankle can then cause damage to ligaments and other soft tissues around the ankle. The ligament most involved in ankle sprain is the anterior talofibular ligament. Depending on the severity of the injury, the ligaments may be overstretched or torn, resulting in instability of the ankle. The symptoms are pain and swelling around the ankle, later followed by discoloration of the skin. Once an ankle sprain occurred, the ligaments in the ankle are weaker and reoccurrence of the injury is highly probable.

3.4.2 The Knee

Knee injuries can occur as a consequence of a trauma (acute injuries) or can be due to overuse (chronic injuries) [1, 2]. Acute injuries can occur after a knee twist that can lead to meniscus or ligament lesions. On the other hand, the most frequent chronic injury of the knee is the patellar tendinopathy (jumper's knee) that is an overuse injury of the patellar tendon that results from repetitive overloading of the extensor mechanism of the knee. It is a common complaint in tennis players due to the explosive muscle contractions needed for the sprinting, jumping and quick changes of directions during tennis. Poor flexibility of the quadriceps (thigh muscles) and hamstrings and variations in leg and foot type (knock knees, bow legs, flat feet, etc.) can contribute to extra load on the tendon and development of jumper's knee. Patellar tendonitis begins as inflammation of the patellar tendon where it attaches to the patella. It can also progress by tearing or degeneration of the tendon. Players with jumper's knee have pain in the area of the patellar tendon, usually near its attachment to the patella, which is present during jumping, sprinting, serving and change of direction after running wide to reach a ball. It typically starts as a dull ache but can gradually increase over a period of time. Initially, the soreness is usually felt following a game or workout, but as the condition worsens, the player may feel stiffness, grinding and swelling in the knee.

3.4.3 The Hip

Historically, injuries to the hip region were thought to focus on the powerful muscles that spanned not only the hip joint but also the knee joint (rectus femoris and hamstrings). An increased understanding of the evaluation and diagnosis of the hip has led to the identification of other forms of hip pathology in tennis due to the impact loading and multidirectional movement patterns and abrupt stopping, starting, cutting and twisting that occur in the lower extremities during tennis [24]. Injuries to the hip including femoroacetabular impingement and labral tears can occur in elite tennis players and require surgical treatment in some cases [25]. In addition to ensuring that proper flexibility exists around the hip and pelvic girdle, exercises to provide greater stabilization to the hip joint and core are recommended to potentially decrease hip injury risk.

References

1. Kibler WB (2002) Pathophysiology of tennis injuries: an overview. In: Renstrom P (ed) Tennis. Tennis Blackwell Publishing Company, Oxford, pp 147–154
2. Pluim BM, Staal JB (2009) Tennis injuries in olympic sports. In: Caine D, Harmer P, Schiff M (eds) Encyclopedia of sports medicine. Wiley-Blackwell, Hoboken
3. Van der Hoeven H, Kibler WB (2006) Shoulder injuries in tennis players. Br J Sports Med 40(5):435–440; discussion 440. Review
4. Sallis RE, Jones K, Sunshine S, Smith G, Simon L (2001) Comparing sports injuries in men and women. Int J Sports Med 22(6):420–423
5. Kibler WB (1995) Biomechanical analysis of the shoulder during tennis activities. Clin Sports Med 14:79–85
6. Kibler WB (2000) Evaluation and diagnosis of scapulothoracic problems in the athlete. Sports Med Arthrosc Rev 8:192–202
7. Kibler WB, Livingston BP (2001) Closed chain rehabilitation for upper and lower extremities. J Am Acad Orthop Surg 9:412–421
8. Harryman DT II, Sidles JA, Clark JM et al (1990) Translation of the humeral head on the glenoid with the passive glenohumeral motion. J Bone Joint Surg 72A:1334–1343
9. Nirschl RP, Ashman ES (2004) Tennis elbow tendinosis (epicondylitis). Instr Course Lect 53:587–598
10. Nirschl RP, Sobel J (1981) Conservative treatment of tennis elbow. Phys Sports Med 9:43–54
11. Safran MR (1995) Elbow injuries in athletes – a review. Clin Orthop Relat Res 310:257–277
12. Elliott B, Fleisig G, Nicholis R, Escamilla R (2003) Technique effects on upper limb loading in the tennis serve. J Sci Med Sport 6:76–87
13. Kibler WB (1994) Clinical biomechanics of the elbow in tennis. Implications for evaluation and diagnosis. Med Sci Sports Exerc 26:1203–1206
14. Ellenbecker TS (1995) Rehabilitation of shoulder and elbow injuries in tennis players. Clin Sports Med 14:87–110
15. Lee HW (1995) Mechanism of neck and shoulder injuries in tennis players. J Orthop Sports Phys Ther 21(1):28–37
16. Hainline B (1995) Low back injury. Clin Sports Med 14:241–266
17. Alyas F, Turner M, Connell D (2007) MRI findings in the lumbar spines of asymptomatic adolescent elite tennis players. Br J Sports Med 41:836–841
18. Green TP, Allvey JC, Adams MA (1994) Spondylosis. Bending of the inferior articular processes of lumbar vertebrae during simulated spinal movements. Spine 19:2683–2691
19. McNeely ML, Torrance G, Magee DJ (2003) A systematic review of physiotherapy for spondylolysis and spondylolisthesis. Man Ther 8(2):80–91
20. Renkawitz T, Linhardt O, Grifka J (2008) Electric efficiency of the erector spinae in high performance amateur tennis players. J Sports Med Phys Fitness 48:409–416
21. Roetert EP, McCormick TJ, Brown SW, Ellenbecker TS (1996) Relationship between isokinetic and functional trunk strength in elite junior tennis players. Isokinet Exerc Sci 6:15–20
22. Timm KE (1995) Clinical applications of a normative database for the Cybex TEF and TORSO spinal isokinetic dynamometers. Isokinet Exerc Sci 5:43–49
23. Ellenbecker TS, Roetert EP (2004) An isokinetic profile of trunk rotation strength in elite tennis players. Med Sci Sports Exerc 36:1959–1963
24. Kovacs M (2009) Movement for tennis: the importance of lateral training. Strength Cond J 31(4):77–85
25. Byrd JW (2006) The role of hip arthroscopy in the athletic hip. Clin Sports Med 25:255–278

Rugby Injuries: Epidemiology and Mechanism

4

Vittorio Calvisi, Remo Goderecci, and Stefano Necozione

4.1 Introduction

Rugby is one of the most popular sports in the world and is played in more than 150 countries [1]. This sport is very popular in the UK, Ireland, Australia, France, South Africa, New Zealand and several Pacific Island nations including Fiji; it is played at junior, amateur, semi-professional and professional levels of participation, and also for media the Rugby World Cup must be considered the third sport event after Soccer World Cup and the Olympic Games. Worldwide there are now flourishing professional leagues in many countries, and after a long absence, rugby union will be returning to the Olympic Games in 2016.

Specifically rugby union is played with 15 players, 8 forwards and 7 backs. The game consists of three basic phases in which most injuries result: *scrum* occurs when opposing 8 forwards (the pack) bind together (engagement) in a pyramid formation, the ball is put in play by the scrum half, and each team attempts to drive their opponents off the ball; *tackle* occurs when a player, ball carrier, is held by an opponent or brought to the ground; and *ruck* (with the ball on the turf) or *maul* (with the ball in the hands of a player) is usually formed when a player is tackled. *Line-outs* occur when the ball goes out of field and usually a ruck or maul is formed once possession is gained by one of the jumpers. The attack forms a platform to protect the ball whilst the defence attempts to crash through the platform and either gain possession.

By spirit of the game, it is an intermittent collision sport characterised by large numbers of physical collisions and tackles, short repeated sprints, rapid acceleration, deceleration and changes of direction and an ability to produce high levels of muscular force extremely rapidly [2].

V. Calvisi (✉) • R. Goderecci • S. Necozione
Dipartimento MESVA, Università degli Studi dell'Aquila, p.le S. Tommasi, L'Aquila 67100, Italy
e-mail: vittorio.calvisi@univaq.it; remogoderecci@hotmail.it; stefano.necozione@cc.univaq.it

As such there is a risk of musculoskeletal injury occurring from the match and training environments due to the number of voluntary physical collisions and tackles that occur [3].

4.2 Definition and Characteristics of Injuries

Definition of injury in rugby is the first aspect to consider: the various authors consider the injury as "Any pain or disability that occurs during participation in rugby league match or training activities that is sustained by a player, irrespective of the need for match or training time loss or for first aid or medical attention; an injury that results in a player requiring first aid or medical attention is referred to as a 'medical attention injury' and an injury that results in the player being unable to partake in full part of future training and/or match activities is referred to a 'time loss' injury" [4, 5].

In 2005 Brooks et al. published a large-scale epidemiological study (546 players from English Premiership during 2002–2003 and 2003–2004 seasons took part in the study), where they defined in detail injury profile of match injuries in elite rugby union. According to the severity, an injury can be classified as mild (left the field or missed a match or both), moderate (missed from two to three matches) or severe (missed more than three matches) [6]. Best et al. in their "Rugby World Cup 2003 injury surveillance project" reported that 70 % of injuries were classified as mild, 14 % as moderate and 16 % as severe. As expected, fractures and dislocations along with sprains caused the longest absence from the game. The most frequently injured body regions were the head, neck and face (33.7 injuries per 1,000 player game hours), followed by the ankle and foot (14 injuries per 1,000 player game hours), the knee and the thigh [7].

Brooks et al. in 2005 obtained that the incidence of new injuries (82 %) was significantly higher than that of recurrent injuries (18 %), and the severity of recurrent injuries was significantly higher than that of new injuries for forwards and backs. They also obtained that the two most common

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_4, © Springer International Publishing Switzerland 2016

pathologies were muscle/tendon and joint (non-bone)/ligament injuries [6]. In line with this data, Schneiders et al. found that the most common site of injury was the face (16 %), followed by the knee and the shoulder (both 14 %); however, the majority of facial injuries were classified as slight, consisting of lacerations, bruises and epistaxis, compared to knee injuries where more than half were classified as either moderate, severe or season ending [8].

About injury severity, it was described that "concussion and cervical nerve root injuries were the most common injuries to tacklers, whereas shoulder injuries resulted in the greatest loss of participation". Specifically, as described by Headey et al., shoulder injuries, anterior glenohumeral dislocation and instability, caused the greatest proportion of days absent from sport than any other part of the body except the knee in professional rugby players [9]; in fact "Medial collateral ligament (MCL) injury of the knee and lateral ankles sprains were the most common sprains; however, anterior cruciate ligament (ACL) injuries account for the highest number of days missed from Sport" [10].

4.3 Incidence and Severity of Injuries

Specifically analysing the incidence of these lesions according to the different roles of the players, Brooks reported that no significant differences were found in the incidence of injury for forwards and backs, whereas other studies at club level have reported higher proportions or incidences of injury for forwards compared with backs in rugby union (65 and 59 %) and rugby league (forwards, 139 injuries/1,000 player-hours; backs, 93 injuries/1,000 player-hours) [6, 11, 12].

Greater contact and collision demands placed on forwards compared with backs and the significantly greater body mass of forwards, which allows them to develop greater momentum, have been suggested as possible explanations for the higher incidence of injury in forwards. Conversely, at the international level, the incidence of injury was reported to be lower for forwards than for backs, although the differences were not significant [13]. The incidences of most neck and spinal injuries were significantly higher for forwards than for backs, and this may result from scrummaging and the higher exposures to contact activities, such as tackling, rucking and mauling, experienced by forwards compared with backs. ACL, MCL and knee meniscal/articular cartilage injuries were particularly severe for both forwards and backs, a finding that was similar to previous studies. Specifically front-row players are subjected to the absorption and transmission of greater forces in scrummaging than other forwards, and this may contribute to the higher risk of absence from shoulder, cervical nerve root, knee meniscal/articular cartilage, calf muscle and Achilles tendon injuries experienced by hookers. Midfield backs (centres) tend to experience more collisions than the other back line players, and this may explain the higher level of absence from shoulder dislocation/instability, wrist and hand fractures, cervical nerve root injuries and thigh haematomas experienced by outside centres. In the Brooks study, the hooker and the fly half were the most commonly injured players, and the right lock and open-side flanker received the most severe injuries [6].

On the contrary the overall incidence of non-contact injuries was significantly higher for backs than forwards. As reported in a 2013 study by Roberts, hamstring strains were the most common non-contact injury type. The higher incidence in backs compared with forwards could be due to the greater sprint and high-intensity running loads (and therefore higher deceleration loads) undertaken by this positional group. Most hamstring injuries were sustained in the first match quarter, indicating that an appropriate prematch warm up may be particularly important. Strategies to reduce hamstring injuries should be given particular consideration as these injuries are likely to be influenced by intrinsic factors, which are potentially modifiable with appropriate training interventions and match warm-up exercises [14] (Fig. 4.1).

Epidemiological studies about rugby injuries include the analysis of incidence and severity of injuries both during the single match and during the season. From the Brooks results, during the match, incidence during the final quarter was higher for players starting a match (114) than for replacement players (87). The higher severity of injuries observed for backs in the third quarter was mainly because 75 % of all ACL injuries sustained by backs (average severity 204 days) occurred during this period; during the season, although there were no significant differences in the monthly incidences of in-season injury, there was a significantly lower incidence of injury during preseason (August) matches compared with the average incidence of injury for in-season matches: it probably reflected the lower competitive nature of the matches (friendlies) played at this time of the season. Also in line with literature, the incidence was higher during the major club competitions than during second team matches. One reason suggested for the higher incidences of injury observed at higher standards of play is the more efficient injury-reporting regimens available at elite clubs because of the superior standard of their medical support [6]. As suggested by Bird and Quarrie studies of 1998 and 2001, respectively, the greater body mass of players at higher playing standards can also contribute to explain these differences. Other factors that may explain the differences include body composition, levels of player fitness and strength, ball in play times and the more competitive nature of matches at higher standards. Bird obtained a detailed analysis: injury events that occurred in club competition games were 87 %, in preseason club games were 8 % and in representative games were 5 % [15, 16] (Fig. 4.2).

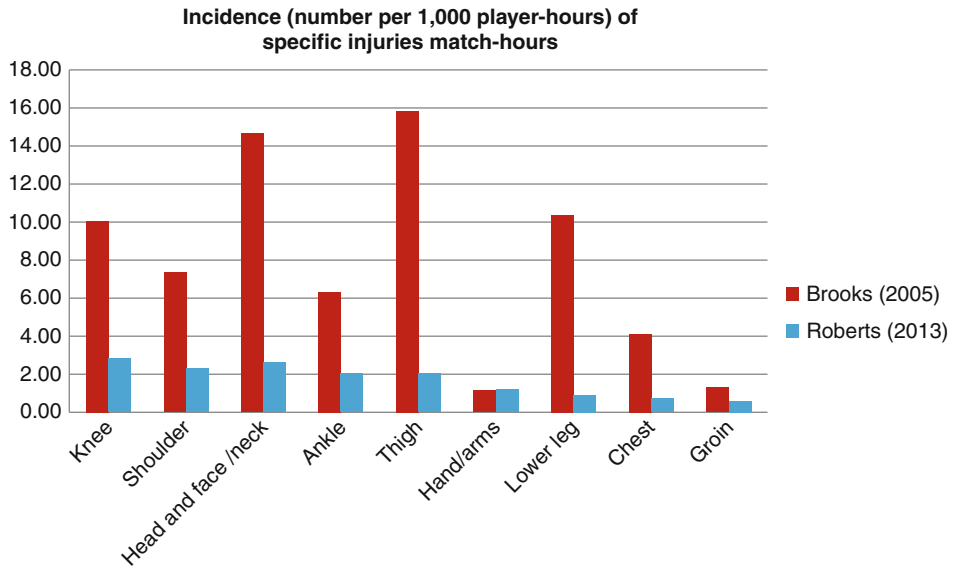


Fig. 4.1 Its reported comparison in the incidence of injuries in England regrouped for top 10 injuries site by Brooks (2002/03 and 2003/04 seasons) and Roberts (2009/10, 2010/11 and 2011/12 seasons) studies. Brooks considered only professional players, whilst Roberts three different levels of the game (semiprofessional, amateur and recreational) [6, 14]

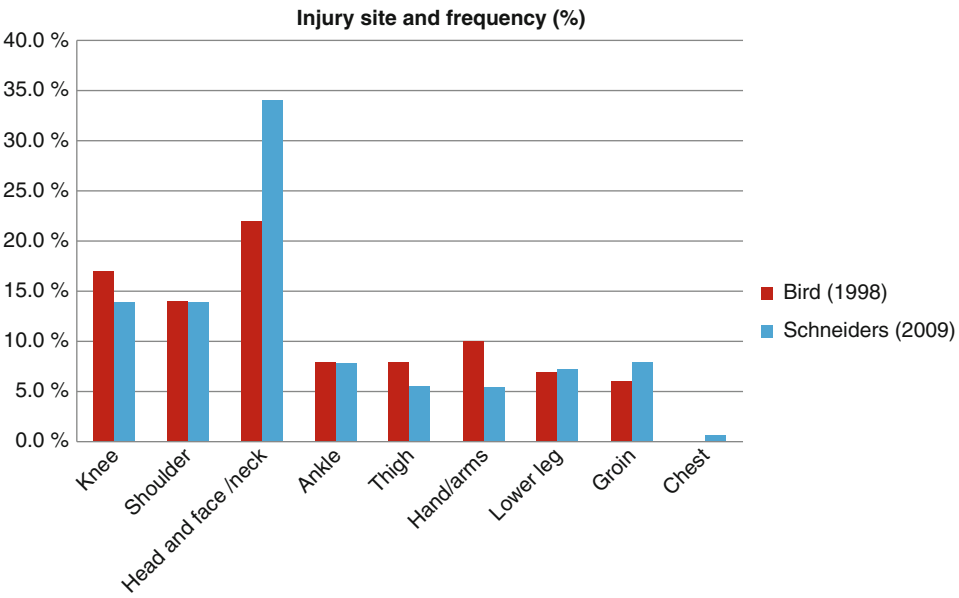


Fig. 4.2 Its reported comparison in the incidence of injuries recorded in New Zealand by Bird (season 1993) and Schneiders (season 2002) studies. Bird's results consider only injuries resulting from tackle injury events including women and men players and included different level of player [8, 15]

4.4 Mechanism of Specific Site Injuries

Many of the lesions of the musculoskeletal system that we find in the game of rugby are absolutely similar to those that we can find in other contact sports, so we wanted to deal with more specificity three skeletal segments that, in our opinion, have a pathophysiology and a peculiar incidence in rugby players: the shoulder, cervical spine and knee.

4.4.1 Shoulder

During a collision sport like rugby, forces applied to the shoulder can either be indirect with the player falling onto his outstretched hand and forearm or directly through a point of impact on the shoulder. Most common shoulder injuries include contusion, acromioclavicular injuries, glenohumeral dislocations, clavicle fractures, rotator cuff tears and muscle sprains, axillary nerve injuries and traumatic myositis ossificans [17].

A number of mechanisms have been identified for injuries to the shoulder and upper extremities. During a rugby match, tackling of an opponent can result in a number of impacts and forces to the shoulder and upper extremities [18] (Fig. 4.3).

McIntosh et al. identified six different tackling methods, all resulting in impact or leveraging forces upon the shoulder [19].

The most common method for tackling is performed from a crouched position with the arms abducted in attempt to reach

Fig. 4.3 During the tackle, a posteriorly directed force can result, in horizontal abduction of the arms and leveraging forces over the glenohumeral joint

around the opponent's trunk. This usually results in an impact directed to the anterior-superior surface of the shoulder and arms. This mechanism can result in a posteriorly directed force resulting in horizontal abduction of the arms and leveraging forces over the glenohumeral joint. Situations where direct impacts and the application of lever forces occur can lead to acromioclavicular joint separations, labral tears, glenohumeral dislocations and clavicle and scapular fractures. The other common tackling method is described as "arm tackling" where the players attempt to tackle the opponent by diving and reaching outwards to grab the ankle for a tripping tackle or grabbing the trunk to slow down or change the direction of the ball carrier [19–21]. Another method of tackling is described as "smothering" where the tackle is attempted from an upright position, wrapping the arms around ball carrier's trunk and arm to trap the ball so that the ball carrier is unable to pass the ball during the tackling manoeuvre. Additional risk factors can be mentioned for shoulder injury when a tackle occurs, for example, the relative size and speed of the opponent, the direction of movement of the tackler as they approach their opponent and the forces delivered by other team members who are also attempting a tackle. Fuller et al. and King et al. have identified greater risks for injuries when two tacklers are involved and when approaching the ball carrier from the side [22, 23].

4.4.2 Cervical Spine

Cervical spinal injuries have always caused greater interest and concern, and more attention has been directed to the aetiology of these injuries and their preventive measures. Injuries to the cervical column may include musculo-ligamentous strains, cervical vertebral injuries with or without neurological deficit (disc injuries or herniations, cervical fractions, subluxations, dislocations), brachial plexus injuries and central cord damage [17].

Due to the large number of collisions commonly occurring at the scrum, ruck and maul, as well as in tackles [19, 22] and fatiguing nature of the game, cervical spine injuries are common at all levels of the game [24–26]. Cervical spine injuries in the scrum could be due to forces greater than two-thirds of a tonne being spread across the front row [27], and these forces may contribute to the neck being forced into a hyperflexed or hyperextended position [28, 29]. At the ruck and maul, cervical spine injuries may be caused by the neck being forcefully positioned into flexion whilst the player is in a vulnerable position such as being on the ground [28]. Finally, with reference to tackling, both the ball carrier and tackler are at risk of cervical spine injury. Under normal circumstances, the forces transmitted to the cervical spine are dissipated by energy capabilities of muscles, discs and, to a lesser extent, ligaments. When a force applied to the cervical spine exceeds the elastic capabilities of involved structures, injuries occur. Mechanical input to the head and neck can either be slow (static loading) occurring gradually or, more commonly, rapidly (dynamic loading) during impact on the head. With neck flexion the normal lordosis is obliterated. As the spine becomes a straight column, forces are transmitted directly to the vertical discs and ligaments rather than to the muscles. When maximum vertebrae compression deformation is reached, the structure becomes unstable and buckles. Compressive fractures develop and discs and ligaments fail subsequently. Hyperflexion, developing in rugby-playing facets like the scrum must still be regarded as an important mechanism of injury. Hyperflexion precedes cervical subluxation and facet dislocation [17] (Fig. 4.4).

Shelly et al. in their study about spinal injuries in rugby reported that "Interestingly, there are national differences in the proportion of injuries occurring in the different phases of play. In South Africa and Ireland there are fewer injuries in the scrum and more in the tackle, whereas in Argentina and Australia, scrums account for the majority of injuries. The explanations for these geographical differences are not clear, but this discrepancy does indicate that different preventive measures need to be emphasised in different countries" [29].

Different approaches may be considered to reduce the incidence of cervical spine injuries in rugby union: rule changes [25, 30] and correct tackling technique [19, 31] have been suggested to reduce the risk of cervical spine injury [32].

Since the modification of these rules, there has not been a serious scrum-related cervical spine injury in Australian rugby. In a New Zealand study, Burry and Gowland [33] also showed a threefold decrease in the incidence of cervical

Fig. 4.4 In rugby scrum, the cervical spine hyperflexion must still be regarded as an important mechanism of injury with subsequent facet subluxation and/or dislocation

spine injuries when similar rules were adopted to slow the pace of scrum engagement [34].

Another approach that has been suggested to decrease these injuries is strengthening the neck musculature [6, 24, 35]. It may be postulated that increased muscle strength may help to dampen the deceleration of the neck into the end-range positions that cause damage to soft tissues [32].

4.4.3 Knee

As reported by Dallalana et al. in their study published in 2007, "the knee was not the most common injury location… but it caused the largest proportion of total days absent (21 %) from playing and training compared with injuries sustained to other body parts". The incidence of knee injuries sustained during match play (11.0 injuries per 1,000 player-hours) was significantly higher than the incidence during training (0.16 injuries per 1,000 player-hours). The most common knee injuries were MCL injuries and minor injuries, predominately soft tissue hematomas or a loosely defined "sprain or jar" of the knee.

The incidence of ACL injuries in matches and training was lower than all other knee injury categories; however they had the greatest average severity and consequently caused the highest proportion of days absent from playing and training due to knee injuries. MCL injuries and chondral/meniscal injuries also caused a high proportion of the days of absence due to knee injuries. A higher proportion of match knee injuries were sustained in contact (72 %) than in training (48 %); ACL, MCL and other minor injuries occurred more often in contact than the other injury categories for both match play and training. The most common match injury mechanism was being tackled, for all injury categories except patellofemoral/extensor mechanism injuries. Angular forces across the knee incurred during a tackle or collapsed maul are high and sufficient to cause significant collateral ligament injury, although none of the injuries required surgical repair or reconstruction of the MCL. A greater proportion of the ACL injuries to professional rugby union players in the mentioned study (86 %) were sustained during contact with other players. This disparity may result from the fact that a larger proportion of actual playing time is spent in some form of contact or in collision with other players than in other "non-contact" sports such as soccer and handball; it therefore follows that more injuries of all types, including ACL injury, will occur during contact. Furthermore, they can involve acts of cutting or pivoting at speed, often whilst being thrown off balance. However, the exact mechanisms of these high-risk injuries need to be examined in greater detail using video analysis and three-dimensional modelling. ACL injuries (89 %; one partial tear was not reconstructed) and chondral/meniscal injuries (49 %) required operation most frequently, resulting in a significantly greater absence from playing and training compared with injuries that were not operated on.

Despite the severe stress from sagittal trauma from anterior to posterior during a tackle, isolated posterior cruciate ligament (PCL) injuries are lesions infrequently found [36]. This type of injury is commonly misdiagnosed, often being classified as a minor sprain/jar or remaining undiagnosed. The nature of rugby produces frequent episodes of landing heavily on the flexed knee and forced hyperextension. The low-severity scores for these injuries and the lack of surgical involvement is consistent with the general belief that athletes either cope well with a minor degree of chronic PCL or posterolateral corner injury or that the injury heals satisfactorily. Torn menisci or chondral injury accounted for 20 % of all days lost, and a high proportion of these injuries were recurrent (33 %) compared with all knee injuries (17 %). There was, perhaps not surprisingly, a trend for the incidence of chondral/meniscal injuries to be higher in older players. Many older rugby players continue active competition with knees showing high-grade degenerative changes, injury to articular surfaces and menisci and possibly a degree of PCL laxity often being the precursors. These players seem to have the ability to "play through" the condition, which would in other sports be career limiting [10].

Conclusion

Several studies have tried to define incidence, nature, severity and causes of rugby injury sustained by professional players, and also the senior authors in the past have dealt with the epidemiology of rugby, but in the later years, in order to make the game more spectacular and continuous, rugby rules are changed and consequently tactics, strategies and tips: defensive phase must be aggressive in the

aim to claw back the ball, making immediately a counter-blast during a vulnerable phase of the opponent team, and the tackle is made for stopping the antagonist but better made on the ball so that he cannot pass the ball. During the attack phase, the player ball carrier must gain ground, but when he is tackled, as they fall over, flip up (offload) the ball to a player in support in a space to go through (interval space), making a powerful tool to attack behind the defensive line in so-called continuous attacking, avoiding ruck and maul which however results centuplicate. More recently we observed that all players, although with evident anthropometrical differences, must be able to do a lot of technical movements with the same efficiency (i.e. rolling tackles, offload) in attack phases and in defensive one: nowadays rugby players are athletes which could play in more than one role, and so in the future there may be no significant differences in injury frequency and severity as function of playing position (Mascioletti M, 2014, personal communication).

In the Brooks study, incidence of injury was lower than that reported previously for rugby union, whilst it was higher than that reported previously in professional ice hockey, soccer and cricket.

Mainly, because of the respect for opponents and the rules of the game the referee enforces strictly and without difficulty, only 6 % of all injuries were caused by foul play and most injuries (72 %) were sustained during contact with another player [6].

Taking this into account, we could say that the incidence of musculoskeletal injury in rugby is lower than we could aspect, having in mind that there are continuous high-energy contacts occurring from the match and training environments due to the number of voluntary physical collisions and tackles that occur (Fig. 4.5).

Fig. 4.5 One of game phases that in spite of the high energy contacts do not cause injuries to players who contend the oval

Bibliography

1. International Rugby Board Web site. Dublin: International Rugby Board (2008) [cited 2 Jan 2010]. Available from: www.irb.com/mm/document/home/0/2008distributionreportinccountries-asat110208_4414.pdf
2. Gabbett TJ (2010) The development and application of an injury prediction model for noncontact, soft-tissue injuries in elite collision sport athletes. J Strength Cond Res 24(10):2593–2603
3. King DA, Hume PA, Milburn P, Gianotti S (2009) Rugby league injuries in New Zealand: a review of 8 years of Accident Compensation Corporation injury entitlement claims and costs. Br J Sports Med 43:595–602
4. Salvia A, Ieracitano VM, Bottiglia Amici Grossi F, Calvisi V et al (2008) Approccio metodologico per lo studio dell'incidenza dei traumi nello sport: l'esempio del rugby. Med Sport 61: 247–257
5. King DA, Gabbett TJ, Gissane C, Hodgson L (2009) Epidemiological studies of injuries in rugby league: suggestions for definitions, data collection and reporting methods. J Sci Med Sport 12(1): 12–19
6. Brooks JHM, Fuller CW, Kemp SPT, Reddin DB (2005) Epidemiology of injuries in English professional rugby union: part 1 match injuries. Br J Sports Med 39:757–766
7. Best JP, McIntosh AS, Savage TN (2005) Rugby World Cup 2003 injury surveillance project. Br J Sports Med 39:812–817
8. Schneiders AG, Takemura M, Wassinger CA (2009) A prospective epidemiological study of injuries to New Zealand premier club rugby union players. Phys Ther Sport 10:85–90
9. Headey J, Brooks JHM, Kemp SPT (2007) The Epidemiology of shoulder injuries in English professional rugby union. Am J Sports Med 35:1537–1543
10. Dallalana RJ, Brooks JH, Kemp SPT, Williams AM (2007) The epidemiology of knee injuries in English professional rugby union. Am J Sports Med 35:818–830
11. Targett SGR (1998) Injuries in professional rugby union. Clin J Sport Med 8:280–285
12. Bathgate A, Best JP, Craig G et al (2002) A prospective study of injuries to elite Australian rugby union players. Br J Sports Med 36:265–269
13. Brooks JHM, Fuller CW, Kemp SPT et al (2005) A prospective study of injuries and training amongst the England 2003 Rugby World Cup squad. Br J Sports Med 39:288–293
14. Roberts SP, Trewartha G, England M et al (2013) Epidemiology of time-loss injuries in English community-level rugby union. BMJ Open 3:e003998
15. Bird YN, Waller AE, Marshall SW et al (1998) The New Zealand rugby injury and performance project. V. Epidemiology of a season of rugby injury. Br J Sports Med 32:319–325
16. Quarrie KL, Alsop JC, Waller AE et al (2001) The New Zealand rugby injury and performance project. VI. A prospective cohort study of risk factors for injury in rugby union football. Br J Sports Med 35:157–166
17. Hugo EP (1992) Epidemiology of shoulder and spinal injuries in rugby. "Traumatologia della colonna cervicale e della spalla nel rugby" di Romanini, Calvisi, Albanese Ginammi, pp 13–21 (CIC edizioni internazionali)
18. Helgeson K, Stoneman P (2014) Shoulder injuries in rugby players: mechanisms, examination, and rehabilitation. Phys Ther Sport 15(4):218–227, http://dx.doi.org/10.1016/j.ptsp.2014.06.001
19. McIntosh AS, Savage TN, McCrory P, Frechede BO, Wolfe R (2010) Tackle characteristics and injury in a cross section of rugby union football. Med Sci Sports Exerc 42(5):977–984
20. Badge R, Tambe A, Funk L (2009) Arthroscopic isolated posterior labral repair in rugby players. Int J Shoulder Sur 3(1):4–7

21. Crichton J, Jones DR, Funk L (2012) Mechanisms of traumatic shoulder injury in elite rugby players. Br J Sports Med 46(7):538–542
22. Fuller CW, Ashton T, Brooks JHM, Cancea RJ, Hall J, Kemp SPT (2010) Injury risks associated with tackling in rugby union. Br J Sports Med 44(3):159–167
23. King D, Hume PA, Clark T (2012) Nature of tackles that result in injury in professional rugby league. Res Sports Med 20(2):86–104
24. Swain MS, Lystad RP, Pollard H, Bonello R (2011) Incidence and severity of neck injury in rugby union: a systematic review. J Sci Med Sport 14:383–389
25. Quarrie KL, Gianotti SM, Hopkins WG, Hume PA (2007) Effects of a nationwide injury prevention programme on serious spinal injuries in New Zealand rugby union: ecological study. Br Med J 334:1150–1153
26. Scher AT (1998) Rugby injuries to the cervical spine and spinal cord: a 10-year review. Clin J Sports Med 17:195–206
27. Milburn PD (1993) Biomechanics of rugby union scrummaging: technical and safety issues. Sports Med 16:168–179
28. Fuller CW, Brooks JH, Kemp SP (2007) Spinal injuries in professional rugby union: a prospective cohort study. Clin J Sport Med 17:10–16
29. Shelly MJ et al (2006) Spinal injuries in Irish rugby. A ten-year review. J Bone Joint Surg Br 88-B:771–775
30. Bohu Y, Julia M, Bagate C, Peyrin J-C, Colonna J-P, Thoreux P, Pascal-Moussellard H (2009) Declining incidence of catastrophic cervical spine injuries in French rugby: 1996–2006. Am J Sports Med 37:319–323
31. Hendricks S, Lambert M (2010) Tackling in rugby: coaching strategies for effective technique and injury prevention. Int J Sports Sci Coach 5:117–135
32. Naish R et al (2013) Can a specific neck strengthening program decrease cervical spine injuries in a men's professional rugby union team? A retrospective analysis. J Sports Sci Med 12:542–550
33. Burry HC, Gowland H (1981) Cervical injury in rugby football-A New Zealand survey. Br J Sports Med 15:56–59
34. Wetzler MJ, Akpata T, Albert T, Foster TE, Levy AS (1996) A retrospective study of cervical spine injury in American rugby, 1970 to 1994. Am J Sports Med 24:454–458
35. Peek K, Gatherer D (2005) The rehabilitation of a professional rugby union player following a C7/T1 posterior microdiscectomy. Phys Ther Sport 6:195–200
36. Romanini L, Santilli G, Calvisi V, Dragoni S (1988) Epidemiologia delle lesioni nel rugby. I J Sports Trauma 10(1):33–39

Basketball: Epidemiology and Injury Mechanism

5

Marco Bigoni, Diego Gaddi, and Massimiliano Piatti

A first and important step in sports injury prevention is an accurate identification and description of the *epidemiology* of this kind of injuries; it is fundamental also to determine their etiology and *traumatic mechanisms* [1]. Basketball is popular not only in the United States but worldwide; it is one of the most practiced sports in Italy, and its diffusion has led to a greater focus on associated injuries and how to prevent/treat them [2–4]. *Basketball* was created in 1891 as a noncontact sport. Evolution of the game has progressed significantly and modified its characteristics, from low post play to high-flying dunks. The physical nature of the game has become extremely aggressive, and the seasons even longer. Nowadays basketball-correlated injuries are frequent on all levels, high school competition, collegiate level, adult professional players, and amateur players; injuries mainly occur during contact between players [5–10]. There are small differences between the American NBA (National Basketball Association) rule book and the FIBA Europe (Fédération Internationale de Basketball Association) one; probably these differences could change the characteristics of the game, but it is not proved that this influences the injury risk or pattern [3]. During the game the players are jumping, landing, pivoting, cutting, running forwards and backwards, changing directions many times, and having contacts with each other; all of them could lead to an injury (Fig. 5.1) [11, 12]. McKay et al. described three important injury risk factors: a history of ankle injury, players wearing shoes with air insoles, and players who didn't do stretching before the game [13].

Also playground, surfaces, and sports equipment influence the pattern of injury [14]. Competitions have a bigger injury ratio than practice, both in professional and amateur basketball categories [5, 15]. Related to training time, different authors showed that those who train longer have more injuries compared with those who train less. Probably the increased exposure may be related to an increased risk of injury due to repetitive and cumulative trauma [7, 16, 17]. Different data analysis observed that most basketball injuries resulted in at least 10 consecutive days of restricted or total loss of participation [6].

Position played by an athlete and anthropometric characteristics are other potential factors that influence the risk of injury. A center is most of the time under the basket in a high–player density area and is thus more at risk for contact injury than a player at the periphery, such as a point guard or forward [18, 19]. An analysis on Brazilian professional basketball players showed that the highest number of injuries concerned center players (44.1 %), followed by forwards (35.3 %) and guards (20.6 %). Center players suffered hand, chest, and abdomen trauma and sprained ankle more than others, mostly after he moves in the free-throw lane and has more physical contact to catch rebounds or for short shots. On the other hand, they have less nontraumatic injuries, probably because their movements are not as intense as forwards and guards, who presented a high rate of nontraumatic injuries [20].

In basketball lower extremity injuries predominate; the ankle is the most frequently injured anatomic site and specifically ankle sprains representing the most common injury (Fig. 5.2) [6, 8].

Injuries to the distal lower extremity have a multifactorial etiology; there is an interaction of psychological, physiological, biomechanical, and anthropometric factors. It was observed that the tissue composition of the leg, specifically the ratio of fat mass to bone mineral content, is related to distal lower extremity injury. The bone mineral content and stiffness of the lower extremity are also inversely correlated to the playground surface with no clear consequences in shock experience [21–23].

Knee and back injuries are also relatively prevalent, whereas injuries at the hip and groin occur less frequently [8]. In the upper extremity, hand and wrist injuries are most commonly encountered [5, 6].

M. Bigoni (✉) • D. Gaddi • M. Piatti
Orthopedic Department, San Gerardo Hospital,
Milano-Bicocca University, Monza 20900, Italy
e-mail: marco.bigoni@unimib.it;
diegogaddi@virgilio.it; massimiliano_piatti@hotmail.com

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_5,

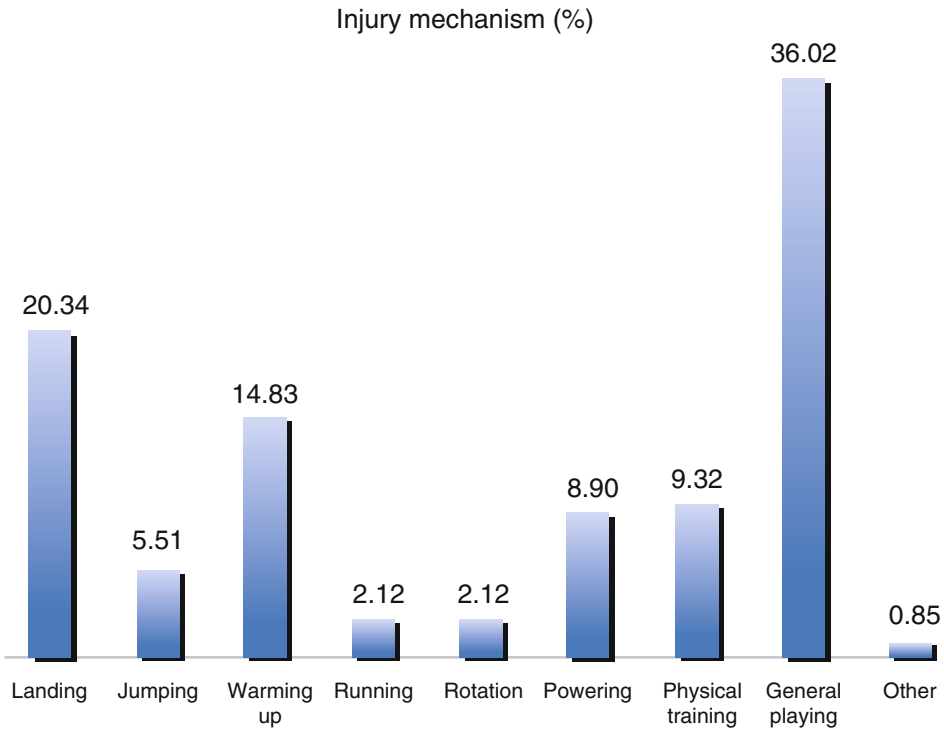


Fig. 5.1 Injury rate by traumatic mechanism (Data from a 3-year observational study on an Italian first league basketball academy)

Both in male and female, young and adult players, there are commonalities and differences in injury patterns [2, 3, 24, 25]. Some studies state that female athletes have increased risk for injury; however in literature there is no unambiguous consent regarding this statement [26–29].

Characteristics of children basketball players' musculoskeletal system make them vulnerable to injuries unseen in adults. Open physis trauma could result in fractures, while in an adult we see strains and sprains because tendons, ligaments, and capsules are stronger than physeal cartilage. The physis is particularly vulnerable during times of rapid growth. The pull of a strong tendon near a growth center can result in repetitive traction injury, as occurs in the Osgood–Schlatter lesion. In a rapidly growing child, there could be differences in growth rates of bones and soft tissues which can result in a loss of flexibility and coordination and muscle imbalances [30–33]. Following gender comparisons of young basketball players, boys are more predisposed to lacerations, fractures, and dislocations, whereas girls suffer mainly traumatic brain injuries, sprains, strains, and soft tissue injuries, and frequently knees and upper extremities are involved. Randazzo et al. described in their data how upper extremity injuries (specifically to the finger) and traumatic brain injuries are more common in younger children (5–10 years of age), whereas lower extremity injuries (specifically to the ankle), sprains and strains, and lacerations are more frequent in older children and adolescents (15–19 years of age) [34].

5.1 Acute Injuries

As *acute injury* we define any basketball accident requiring minimum care and causing the absence of the injured player for at least one training or game session [35].

5.1.1 Ankle Sprains

Ankle sprains are the most common acute injuries in adult as well as young basketball players, both in professional players and amateur players. The risk of ankle sprains is higher during games than during training and more likely in offense than defense [3, 6, 8, 24, 36–38]. Lateral ankle sprains predominate over medial sprains, and some researchers refer to ankle sprain as exclusively an injury of the lateral ligament complex [39–41]. The most frequent traumatic mechanism in lateral sprains is an inversion injury with the foot in slight plantar flexion. This happens mostly when one player steps on the foot of another player rolling the ankle inwards or when the player lands awkwardly. Cutting, turning, and pushing off awkwardly are other common causes for ankle sprains. The anterior talofibular ligament is most commonly injured followed by the calcaneofibular ligament. Eversion injury is much less common, occurring secondary to either dorsiflexion with eversion or external rotation of the foot. Nevertheless, eversion injuries may be relatively serious because the deltoid ligament, anterior tibiofibular ligament, and interosseous membrane may be

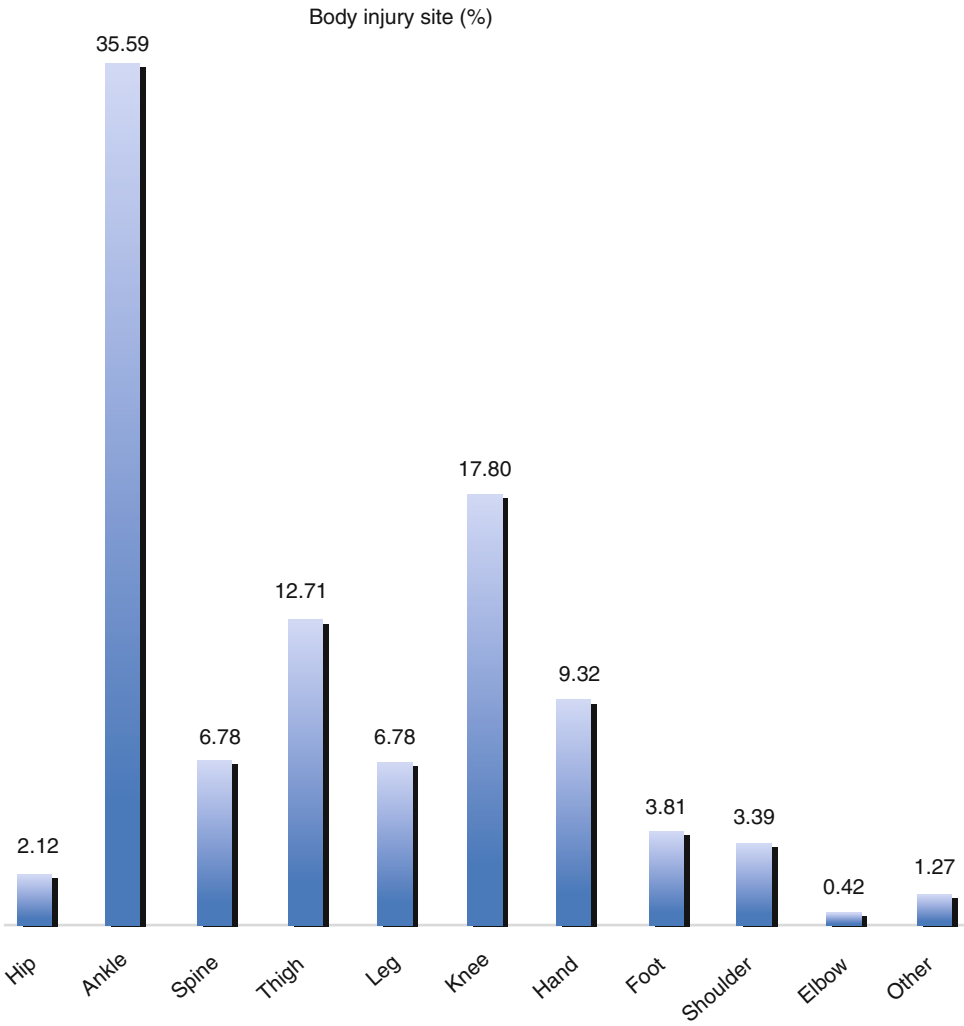


Fig. 5.2 Injury rate by body site (Data from a 3-year observational study on an Italian first league basketball academy)

involved with the disruption of the ankle mortise [3, 39, 40]. Magnetic resonance imaging (MRI) is useful to confirm ligament deficiency and to detect injuries that are radiographically occult, including chondral or osteochondral injuries of the talar dome [40, 42–44].

Ankle taping and bracing, high-top shoes, and balance training show in different studies a protective effect on the rate of ankle sprains in basketball; they are particularly effective in players with prior ankle injuries. Thacker et al. found semirigid ankle braces to be effective in preventing ankle sprain and that braces do not adversely affect performance [13, 45–49]. A lot of ankle sprains sustained during basketball become recurrent, and a large part of these players perceived mechanical instability and persisting symptoms [50, 51].

It's very important to evaluate ankle injuries and fractures in child athletes accurately due to the presence of open growth physes and make sure to exclude a Salter I fracture of the distal fibula, which may only be suggested on radiographs by soft tissue swelling adjacent to the injured growth plate [52].

A lot of acute foot injuries are associated to an ankle sprain. These injuries include avulsion fractures of the navicular, avulsion fractures of the base of the fifth-ray metatarsal (secondary to inversion stress), avulsion at the origin of the extensor digitorum brevis muscle from the lateral calcaneus, fractures of the anterolateral process of the calcaneus (typically occur with the foot abducted and plantar flexed), fractures of the anterolateral process of the calcaneus, os trigonum fractures, and tear of the superior peroneal retinaculum (SPR) with peroneal tendon subluxation (it may occur during acute dorsiflexion with strong contraction of the peroneal muscles to prevent further dorsiflexion) [42].

5.1.2 Knee Ligament Acute Injuries

The mechanism of injury to the *anterior cruciate ligament* (ACL) in basketball is more commonly noncontact, deceleration and sudden change in direction that may cause abnormal rotation of the tibia, resulting in ACL injury.

Furthermore valgus *knee* collapse occurs more frequently in women [53, 54].

Different studies show a strong female predilection in ACL injury in basketball players; different meta-analysis of the incidence of ACL injury as a function of gender and sport reveal a female to male ratio of 3.5:1 [7, 25, 55, 56]. The following risk factors can explain this increased risk of ACL injury in female adult and young athletes: a heavy risk in the preovulatory phase of the menstrual cycle, decreased intercondylar notch width on radiographs (this factor is not confirmed in professional male basketball players), and a predisposition to increased knee abduction on landing in female athletes [33, 57, 58].

A common pattern of ACL injury for a skeletally immature athlete is an avulsion fracture of the tibia, or less commonly the femur, at the site of ACL attachment, as the chondro-osseous junction is the weakest part of the ACL complex [33].

ACL tear is a serious injury with significant loss of playing time and long rehabilitation after surgical repair. Although many competitive basketball players return to action after ACL reconstruction, Busfield et al. showed that 22 % of NBA players didn't return to compete and 44 % of those who returned experienced a lower player efficiency rating [59].

Injuries to the posterior cruciate ligament (PCL) are extremely rare in basketball; the mechanism in an athlete typically involves a fall on the shin or a hyperextension injury [60].

Avulsion of the tibial tubercle in a young athlete has been well described among basketball players and may occur bilaterally. The mechanism involves violent knee flexion against a tightly contracted quadriceps muscle or violent quadriceps contraction with a fixed foot [33].

5.1.3 Hip and Pelvis Acute Injuries

Compared to ankle and knee injuries, injuries to the pelvis, hip, and upper thigh are moderately common in basketball players. According to the National Collegiate Athletic Association (NCAA) and NBA, injuries to the pelvis, hip, and upper leg accounted for approximately 10 % of game-related injuries and 11 % of injuries sustained during practice. In all data thigh injuries are more prevalent, and the specific injuries identified were musculotendinous strains and contusions [2, 6, 41]. Although there is a heightened understanding of intra-articular hip pathology, most athletic-related injuries to the hip are extra-articular [10]. Most injuries occurring about the pelvis, hips, and upper thighs were composed of musculotendinous strains and contusions, which included adductor and rectus abdominis strains, hamstring injuries, and thigh muscle contusions. Hamstring and adductor tears have been shown to be predominately proximal and constitute injury about the hip rather than the knee [61, 62]. The quadriceps is the most commonly injured (contusion/strain) structure and has significant game-related injury rate compared with other structures. The hamstring muscle group was the most frequently strained as result from dynamic overload/eccentric contractions. Jackson et al. demonstrated in their statistical analyses that strains were most frequent in the first month of the season (the preseason) and the cumulative risk is related with length of the player career and of each season [10].

5.1.4 Upper Extremity Acute Injuries

Upper extremity injuries, overall, accounted for 12–13 % of injuries sustained at both the high school and professional levels of play [5, 8]. Hand and arm injuries predominate over injuries to the shoulder or elbow [63]. The fingers and thumb represent the most likely site of acute fracture in basketball players, and the proximal interphalangeal joints (PIP) are the most frequently injured sites [7]. "Dunk lacerations" have also been described in basketball players. These injuries occur secondary to the impact of the player's hand with sharp edges of the rim or with the flange connecting the rim to the backboard [64]. A mallet finger, also known as a hammer finger, is a very common basketball injury that disrupts the extensor mechanism at the distal interphalangeal joint. The mechanism is typically an axial load on a partially flexed finger, usually a ball striking the tip of the finger. In a young athlete, an avulsion fracture of the epiphysis is more likely than a rupture of the extensor tendon. Other hand/finger injuries may also include tears of the volar plate (sometimes associated with an avulsion fracture), metacarpophalangeal joint injuries (thumb's ulnar collateral ligament tear is very common), and carpometacarpal joint injuries [65, 66]. It's an accepted opinion that for young children, age-appropriate basketballs should be used, which may decrease the rates of concussions and finger-related injuries, and rough play should be discouraged, to minimize collisions [34].

Shoulder injuries sustained during basketball are uncommon. In the NBA, the most frequently identified shoulder injuries were glenohumeral sprain, acromioclavicular joint sprain, and rotator cuff inflammation. In a review of shoulder injuries in high school athletes between 2005 and 2007, the incidence was 0.47 injuries per 10,000 exposures for boys and 0.45 injuries per 10,000 exposures for girls. Injuries were much more commonly sustained during competition than during practice, with most injuries occurring during defending and rebounding [8, 67].

5.1.5 Wrist Acute Injuries

Wrist injuries are most typically the consequence of falling on an outstretched hand. The location of a fracture following this type of fall depends on the angle of the wrist upon hitting

the ground as well as the age of the patient at the time of injury. If the wrist is more flexed, the athlete is more likely to sustain a scaphoid fracture. If the wrist is more extended, he or she is more likely to suffer a distal radial or ulnar fracture. Also, the risk of scaphoid fracture in children increases as the bone matures [68, 69].

Ligamentous wrist injuries can include injuries to the triangular fibrocartilage complex (TFCC). This type of injury may occur due to acute trauma or repetitive injury. When acutely injured, the mechanism is often related to axial load bearing with rotational stress, often during a fall on an outstretched hand [68].

5.1.6 Back Acute Injuries

Back injuries in basketball players accounted for 6.8 % of all injuries sustained by NBA players over a 10-year period but represented 11 % of all days missed. Back muscle strain was the most common presentation; disk rupture/herniation was far less prevalent. Cervical spine injuries were significantly less common than lumbar injuries, accounting for 1.3 % of injuries overall. Sacral injuries amounted to 0.6 % of the total and thoracic spine injuries 0.5 % of the total [8].

5.2 Overuse Injuries

As *overuse injuries* we define those causing physical discomfort with an insidious onset; they cause pain and/or stiffness and can potentially affect the player during and/or after the basketball activity [3].

5.2.1 Ankle, Foot, and Lower Leg Overuse Injuries

MRI is considered the gold standard for early diagnosis of stress injury. Osseous stress fractures occurring below the knee in the basketball player frequently involve the tibia or the distal fibula, the latter most common approximately 5 cm proximal to the tip of the lateral malleolus. Stress fractures may also be observed, albeit less commonly, in the tarsal navicular, calcaneus, metatarsals, and cuneiforms [70].

Tibial *stress fractures* most commonly involve the posteromedial cortex, a pattern of injury most commonly seen in athletes participating in running sports. Athletes involved in jumping sports such as basketball can develop a more specific stress fracture involving the anterior tibial cortex. For stress fractures of the posteromedial surface, the prognosis is generally good with conservative management. The prognosis is worse if the fracture involves the anterior tibia, as these have a higher rate of nonunion or progression to complete fracture [71, 72]. Potentially modifiable risk factors for stress fractures in female basketball players include low cardiorespiratory fitness, lack of resistance training, poor nutrition (e.g., low calcium intake, negative energy balance), menstrual dysfunction, shoes, and less time to recover with hard off-season workouts [73–75].

One severe foot injury in basketball players, which may result from overuse, is the Jones fracture. The Jones fracture is located at the diaphyseal-metaphyseal junction of the proximal fifth metatarsal and results from the abnormal loading of the lateral foot when the heel is elevated and the metatarsophalangeal (MTP) joints are hyperextended [76]. Jones fractures may be slow to heal, and they have a high rate of nonunion when treated nonoperatively [77].

Several soft tissue conditions in the lower leg and foot of basketball players may result from overuse. Achilles tendinosis may be both insertional and non-insertional in location. At the ankle and hindfoot, plantar fasciitis is not uncommon, and anterior ankle impingement may be a sequel of chronic lateral ankle sprains with ligament injury.

At the forefoot, sesamoid and MTP joint injuries, synovitis, and adventitial bursa formation may be observed [42, 76]. Sports-related Achilles tendon rupture peaks in the fourth decade. Degenerative change is usually present in complete rupture, and most ruptures occur 2–6 cm above the calcaneus, where the blood supply is lowest, and flow decreases with age [78–80]. Achilles tendon injuries occur when significant forces are translated through a malaligned tendon with a dorsiflexed foot and an extended knee. Of the 18 players identified by Amin et al. over 23 NBA seasons, only 44 % were able to return to play for longer than 1 season after their surgical repair. Those who did return to play did not perform as well as their control-matched peers [81].

5.2.2 Knee Overuse Injuries

Overuse injuries at the knee predominate at the extensor apparatus. Some investigators consider the entity jumper's knee to apply to tendinosis occurring anywhere along the *extensor mechanism* from quadriceps tendon to the tibial tubercle and not exclusively to that involving the proximal patellar tendon [82]. In a 10-year prospective study of injury and illness in the NBA, patellofemoral inflammation accounted for 8.1 % of orthopedic injuries [8].

Eccentric muscle contraction inherent in sudden decelerations and jumping can result in extensor mechanism injuries, including the patellofemoral joint. These mechanisms may lead to microscopic tendon tear, especially in the proximal patellar tendon. Similarly, excessive force generated across the patellofemoral articulation may result in overuse injury, especially in people with underlying joint malalignment. Heavy and long-standing abnormal stress may even lead to

patellar or quadriceps tendon rupture or patellar stress fractures [82]. Early patellofemoral arthrosis in basketball players may be asymptomatic. Whether or not symptoms are present, patellofemoral chondromalacia may manifest as cartilage signal alteration, fissuring, or fibrillation. Focal cartilage defects along patella and trochlea may be observed, often with underlying subchondral cysts or bone marrow edema [83].

A repetitively strained attachment of tendon to bone can develop traction apophysitis. Sinding-Larsen-Johansson lesion occurs at the junction of the inferior patella and the patellar tendon and most commonly occurs in athletes between the ages of 10 and 14 years. The Sinding-Larsen-Johansson lesion (chronic injury) must be differentiated from an inferior patellar sleeve avulsion injury (acute injury). The Osgood-Schlatter lesion occurs at the insertion of the patella tendon to the tibial tuberosity. This typically concerns boys between 11 and 15 years and sports participation during a rapid growth spurt [33].

5.2.3 Hip and Pelvis Overuse Injuries

Hip stress fracture is exceedingly rare in the basketball sport. In addition, very few players presented symptoms of internal derangement at the hip. Awareness of intra-articular disorders such as femoroacetabular impingement and labral tears has increased substantially over the last decade. Some authors have suggested that adductor strain with rectus strain can occur in the setting of labral tears in what has been called the "sports hip triad," and we know from different epidemiological study that adductor strains in basketball are very common [10, 84].

5.2.4 Back Overuse Injuries

Careful evaluation of the posterior spinal elements, particularly at L5, is critical on cross-sectional imaging studies, even in the absence of spondylolisthesis. Pars interarticularis defects are an important cause of back pain in young athletes, including basketball players [85].

References

1. Van Mechelen W et al (1992) Incidence, severity, aetiology and prevention of sports injuries: a review of concepts. Sports Med 14(2):82–99
2. Zelisko JA et al (1982) A comparison of men's and women's professional basketball injuries. Am J Sports Med 10:297–299
3. Cumps E et al (2007) Prospective epidemiological study of basketball injuries during one competitive season: ankle sprains and overuse knee injuries. J Sports Sci Med 6:204–211
4. Stone WJ et al (1993) Year-round conditioning for basketball. Clin Sports Med 12:173–191
5. Borowski LA et al (2008) The epidemiology of US high school basketball injuries, 2005–2007. Am J Sports Med 36:2328–2335
6. Dick R et al (2007) Descriptive epidemiology of collegiate men's basketball injuries: National Collegiate Association Injury Surveillance System, 1988–1989 through 2003–2004. J Athl Train 42:194–201
7. Agel J et al (2007) Descriptive epidemiology of collegiate women's basketball injuries: National Collegiate Association Injury Surveillance System, 1988–1989 through 2003–2004. J Athl Train 42:202–210
8. Starkey C et al (2000) Injuries and illnesses in the National Basketball Association: a 10-year perspective. J Athl Train 35:161–167
9. Guyette RF et al (1993) Facial injuries in basketball players. Clin Sports Med 12:247–264
10. Jackson TJ et al (2013) Epidemiology of hip injuries in the National Basketball Association a 24-year overview. Orthop J Sports Med 1:3
11. Jackson MD et al (1996) Essentials of sport medicine. Mosby-Year Book, St Louis, pp 558–570
12. Burnham BR et al (2010) Mechanisms of basketball injuries reported to the HQ Air Force Safety Center a 10-year descriptive study, 1993–2002. Am J Prev Med 38(1S):S134–S140
13. McKay GD et al (2001) Ankle injuries in basketball: injury rate and risk factors. Br J Sports Med 35(2):103–108
14. Bassett FH et al (1994) Sports injuries: mechanisms, prevention and treatment. Williams & Wilkins, Baltimore, pp 209–222
15. Stergioulas A et al (2007) Amateur basketball injuries. A prospective study among male and female athletes. Biol Exerc 3:35–46
16. Vanderlei FM et al (2013) Sports injuries among adolescent basketball players according to position on the court. Int Arch Med 6:5
17. Gianoudis J et al (2008) Volume of physical activity and injury occurrence in young basketball players. J Sports Sci Med 7(1):139–143
18. Meeuwisse WH et al (2003) Rates and risks of injury during intercollegiate basketball. Am J Sports Med 31:379–385
19. Neto Paiva A et al (2005) Body composition assessment in male basketball players in Brazilian National Basketball League 2003. Rev Bras Cine Des Hum 7(1):35–44
20. Moreira P et al (2003) Prevalence of injuries of Brazilian Basketball National Team during 2002 season. Rev Bras Med Esporte 9(5):258–262
21. Burkhart TA et al (2013) Tissue mass ratios and the reporting of distal lower extremity injuries in varsity athletes at a Canadian University. J Sports Sci 31(6):684–687
22. Hardin EC et al (2004) Kinematic adaptations during running: effects of footwear, surface, and duration. Med Sci Sport Exerc 36:838–844
23. Holmes JD et al (2005) Predicting in-vivo soft tissue masses of the lower extremity using segment anthropometric measurements and DXA. J Appl Biomech 21:371–382
24. Deitch JR et al (2006) Injury risk in professional basketball players: a comparison of Women's National Basketball Association and National Basketball Association Athletes. Am J Sports Med 34:1077–1083
25. Moul JL et al (1998) Differences in selected predictors of anterior cruciate ligament tears between male and female NCAA division I collegiate basketball players. J Athl Train 33:118–121
26. De Loes M et al (1995) Epidemiology of sports injuries in the Swiss organization Ÿouth and Sports 1987–1989. Injuries, exposure and risks of main diagnoses. Int J Sports Med 16:134–138
27. De Loes M et al (2000) A 7-year study on risks and costs of knee injuries in male and female youth participants in 12 sports. Scand J Med Sci Sports 10:90–97
28. Colliander E et al (1986) Injuries in Swedish elite basketball. Orthopedics 9:225–227
29. Messina DF et al (1999) The incidence of injury in Texas high school basketball. A prospective study among male and female athletes. Am J Sports Med 27:294–299

30. Maffulli N et al (1995) Common skeletal injuries in young athletes. Sports Med 19:137–149
31. Van Mechelen W et al (1997) The severity of sports injuries. Sports Med 24:176–180
32. Outerbridge AR et al (1995) Overuse injuries in the young athlete. Clin Sports Med 14:503–516
33. Gaca AM et al (2009) Basketball injuries in children. Pediatr Radiol 39:1275–1285
34. Randazzo C et al (2010) Basketball-related injuries in school-aged children and adolescents in 1997–2007. Pediatrics 126:727
35. Van Mechelen W et al (2004) A one season prospective cohort study of volleyball injuries. Br J Sports Med 38:477–481
36. Pappas E et al (2011) The epidemiology of pediatric basketball injuries presenting to US Emergency Departments: 2000–2006. Sports Health 3(4):331–335
37. Kofotolis N et al (2007) Ankle sprain injuries: a 2-year prospective cohort study in female Greek basketball players. J Athl Train 42:388–394
38. Nelson AJ et al (2007) Ankle injuries among United States high school athletes, 2005–2006. J Athl Train 42:381–387
39. Sickles RT et al (1993) The adolescent basketball player. Clin Sports Med 12:207–219
40. Johnson KA et al (1993) Sprained ankles as they relate to the basketball player. Clin Sports Med 12:363–371
41. Drakos MC et al (2010) Injury in the National Basketball Association: a 17-year overview. Sports Health 2:284–290
42. McDermott EP et al (1993) Basketball injuries of the foot and ankle. Clin Sports Med 12:373–393
43. Perrich KD et al (2009) Ankle ligaments on MRI: appearance of normal and injured ligaments. Am J Roentgenol 193:687–695
44. O'Loughlin PF et al (2010) Current concepts in the diagnosis and treatment of osteochondral lesions of the ankle. Am J Sports Med 38:392–404
45. Thacker SB et al (1999) The prevention of ankle sprains in sports: a systematic review of the literature. Am J Sports Med 27:753–760
46. Garrick JG et al (1973) Role of external support in the prevention of ankle sprains. Med Sci Sports 5:200–203
47. McGuine TA et al (2006) The effect of a balance training program on the risk of ankle sprains in high school athletes. Am J Sports Med 34:1103–1111
48. Van Mechelen W et al (2000) The effect of preventive measures on the incidence of ankle sprains. Clin J Sport Med 10:291–296
49. Van Mechelen W et al (2004) The effect of a proprioceptive balance board training program for the prevention of ankle sprains. A prospective controlled trial. Am J Sports Med 32:1385–1393
50. Barrett JR et al (1993) High- versus low-top shoes for the prevention of ankle sprains in basketball players: a prospective randomized study. Am J Sports Med 21(4):582–585
51. Leanderson J et al (1993) Ankle injuries in basketball players. Knee Surg Sports Traumatol Arthrosc 1(3–4):200–202
52. Omey ML et al (1999) Foot and ankle problems in the young athlete. Med Sci Sports Exerc 31:S470–S486
53. Emerson RJ et al (1993) Basketball knee injuries and the anterior cruciate ligament. Clin Sports Med 12:317–328
54. Krosshaug T et al (2007) Mechanisms of anterior cruciate ligament injury in basketball: video analysis of 39 cases. Am J Sports Med 35:359–367
55. Agel J et al (2005) Anterior cruciate ligament injury in National Collegiate Association Basketball and Soccer: a 13-year review. Am J Sports Med 33:524–531
56. Prodromos CC et al (2007) A meta-analysis of the incidence of anterior cruciate ligament tears as a function of gender, sport, and a knee injury reduction regimen. Arthroscopy 23:1320–1325
57. Renstrom P et al (2008) Non-contact ACL injuries in female athletes: an International Olympic Committee current concepts statement. Br J Sports Med 42:394–412
58. Lombardo S et al (2005) Intercondylar notch stenosis is not a risk factor for anterior cruciate ligament tears in professional male basketball players: an 11-year prospective study. Am J Sports Med 33:29–34
59. Busfield BT et al (2009) Performance outcomes of anterior cruciate ligament reconstruction in the National Basketball Association. Arthroscopy 25:825–830
60. Moyer RA et al (1993) Injuries of the posterior cruciate ligament. Clin Sports Med 12:307–315
61. Karlsson J et al (1994) Chronic groin injuries in athletes. Recommendations for treatment and rehabilitation. Sports Med 17:141–148
62. Koulouris G et al (2003) Evaluation of the hamstring muscle complex following acute injury. Skeletal Radiol 32:582–589
63. Sonzogni JJ et al (1993) Assessment and treatment of basketball injuries. Clin Sports Med 12:221–237
64. Kirk AA et al (1979) Dunk lacerations and unusual injuries to the hands in basketball players. JAMA 242:415
65. Mastey RD et al (1997) Primary care of hand and wrist athletic injuries. Clin Sports Med 16:705–724
66. Major NM (2006) Role of MRI in prevention of metatarsal stress fractures in collegiate basketball players. AJR Am J Roentgenol 186:255–258
67. Bonza JE et al (2009) Shoulder injuries among United States high school athletes during the 2005–2006 and 2006–2007 school years. J Athl Train 44:76–83
68. Rettig AC et al (2003) Athletic injuries of the wrist and hand. Part I: traumatic injuries of the wrist. Am J Sports Med 31:1038–1048
69. Nafie SA et al (1987) Fractures of the carpal bones in children. Injury 18:117–119
70. Palmer WE et al (1999) MR imaging of myotendinous strain. AJR Am J Roentgenol 173:703–709
71. Baublitz SD et al (2004) Acute fracture through an intramedullary stabilized chronic tibial stress fracture in a basketball player: a case report and literature review. Am J Sports Med 32:1968–1972
72. Boden BP et al (2000) High-risk stress fractures: evaluation and treatment. J Am Acad Orthop Surg 8:344–353
73. Rauh MJ et al (2006) Epidemiology of stress fracture and lower-extremity overuse injury in female recruits. Med Sci Sports Exerc 38:1571–1577
74. Shaffer RA et al (2006) Predictors of stress fracture susceptibility in young female recruits. Am J Sports Med 34:108–115
75. Joy EA et al (2005) Stress fractures in the female athlete. Curr Sports Med Rep 4:323–328
76. Meyer SA et al (1993) Stress fractures of the foot and leg. Clin Sports Med 12:395–413
77. Fernandez FM et al (1999) Fractures of the fifth metatarsal in basketball players. Knee Surg Sports Traumatol Arthrosc 7:373–377
78. Flood L et al (2009) Epidemiology of basketball and netball injuries that resulted in hospital admission in Australia, 2000–2004. Med J Aust 190(2):87–90
79. Jozsa L (1989) The role of recreational sport activity in Achilles-tendon rupture – a clinical, pathoanatomical, and sociological study of 292 cases. Am J Sports Med 17:338–343
80. Carr AJ et al (1989) The blood-supply of the calcaneal tendon. J Bone Joint Surg Br 71:100–101
81. Amin NH et al (2013) Performance outcomes after repair of complete achilles tendon ruptures in national basketball association players. Am J Sports Med 41(8):1864–1868
82. Molnar TJ et al (1993) Overuse injuries of the knee in basketball. Clin Sports Med 12:349–362
83. Major NM et al (2002) MR imaging of the knee: findings in asymptomatic collegiate basketball players. Am J Roentgenol 179:641–644
84. Feeley BT et al (2008) Hip injuries and labral tears in the National Football League. Am J Sports Med 36:2187–2195
85. Herskowitz A et al (1993) Back injuries in basketball players. Clin Sports Med 12:293–306

Football: Epidemiology and Injury Mechanism

6

Piero Volpi, Cristiano Eirale, and Gian Nicola Bisciotti

6.1 Introduction

Football is the most popular sport in the world; according to data provided by the Federation of International Football Associations (FIFA), it counts 250 million members across the five continents. In this huge movement, the numerical contribution of young players is relevant, as well as the increasing number of women who are dedicated to this sport.

This massive participation has stimulated research and scientific activity on football injuries. Most of the players have to suspend periodically their activities in order to undergo medical treatment, surgery and rehabilitation following an injury, and this time out of the field may cause to the player a significant physical and psychological discomfort. Therefore, research on football injuries has significantly increased in the last decades. Initially, most of the efforts have focused on the study of injury treatment; however, despite a better comprehension of the physiopathology of sport injury, new scientific findings have a limited impact on the return-to-play time and the recurrence rate. Therefore, it has become clear that preventing injuries may be the main way to decrease the absence of the athlete from the competitions.

Many accidents are unpredictable; they are a natural part of the characteristics of a sport that requires speed, strength, explosiveness, but also physical contacts with the opponent. However, many injuries can be avoided; conducting injury surveillance studies is the fundamental first step in order to implement adequate programmes of prevention. A scientifically validated knowledge of the injury incidence and patterns will lead to a correct evaluation of their risk factors and therefore to the implementation of adequate prevention programmes.

It is necessary to underline how in football, even at professional level, while many efforts are focused on continuous improvement of the physical, technical and tactical performance, less is done in terms of injury prevention. This is at least curious, considering the impact of injuries on players' health and performance and therefore on club finances. Recent researches have emphasised this, proving how injuries impact the team success: the higher the number of injuries in a team, the lower the results of that team during that season [1, 2].

6.2 Football Epidemiology

Epidemiology is the science studying the incidence and the patterns of injuries that occur during sport activities. Knowing injury incidence and patterns is the fundamental first step in order to implement an adequate programme of prevention. However, sometimes it is difficult to compare researches as they are based on a different methodology.

Epidemiological surveys on football injuries have been implemented in Europe since the end of the seventies, in particular in the Northern countries [3, 4]. These researches described football injuries showing for the first time the possibility of reducing their incidence [5, 6]. In the last three decades, professionalism in football has increased, and injury surveys have been performed at all levels of football: amateur [7], semiprofessional [8], professional [9] and elite [10].

P. Volpi (✉)
Knee Orthopedics and Sport Traumatology Unit,
Humanitas Clinical Institute IRCCS, Milan, Italy

Head of Medical Staff FC Internazionale Milan, Milan, Italy
e-mail: volpi.piero@libero.it

C. Eirale
Aspetar Hospital, Sport City, Doha 29222, Qatar
e-mail: Cristiano.eirale@aspetar.com

G.N. Bisciotti
Qatar Orthopaedic and Sport, Medicine Hospital,
FIFA Center of Excellence, Doha 29222, Qatar

Kinemove Centers, Pontremoli, La Spezia, Italy
e-mail: bisciotti@libero.it

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_6,

Moreover, they have been performed also at youth level [11] and in women football [12] and implemented in many extra-European countries [13].

6.3 Methodological Considerations

Unfortunately, researches on football epidemiology have often employed different methods of data collection. These differences make difficult to interpret the findings, which can also be very different, in a comparable manner. In the last few years, experts have provided guidelines on injury definitions and collecting procedures [14] allowing comparisons of the results from more recent research, permitting a better definition of typical patterns of different settings.

Another important factor is the reliability of data. Football epidemiological studies often require the involvement of multiple clubs and therefore of different clinicians. This can be a source of bias. First, it has to be verified that all participants utilise the same medical approach in diagnosis and prognosis. In this view, it is better that all clinicians involved in the data collection are part of the same professional category, for example, sport medicine physicians, orthopaedic surgeons or physiotherapists. Sometimes, this is not enough because they may be part of different medical schools; some studies involve team from all over the world [15] with an undeniably different approach to sport medicine. Therefore, scientists have to keep in mind these considerations, in order to avoid bias. Another important issue is not only the quality of the data but also their completeness. Prospective studies may last an entire season or even more. Constant supervision of the people in charge of the data collection is fundamental in order to ensure complete collection. New technologies such as mobile phones, table PC and specific software have been implemented with this aim. In addition, recent literature shows the need for a revision of the best definition of injury to be utilised, in order to capture at the best kinds of injuries and complaints [16]. First reports utilised data from emergency departments or insurance companies; however, it is evident how minor injuries were overlooked in this way. The most utilised injury definitions are the medical attention and time-loss injuries. Medical attention injuries are all the injuries for which the footballers require the help of a member of the medical staff. In this way, a high number of injuries can be collected; however, it is more difficult to identify the categories of lesions that have an impact on players' health and football performance. Time-loss injuries are those injuries for which the player has to stop football activity. This definition allows a good identification of the injuries impacting the footballer's activity; however, sometimes players continue to play and train despite injuries that may decrease their performance or may lead to more severe or different injuries [17], and such injuries, for the abovementioned consequences, must not be overlooked.

6.4 Epidemiological Researches in Professional Football

The highest expression of club level football in the world is arguably represented by the UEFA Champions League. Since 2001, UEFA has implemented an injury analysis among some clubs participating to this competition, with the aim of reducing injuries which, at this level, has a high economical impact. Actually, this study involves more than 20 European top level football clubs from different countries. Each day, injury data, together with information on the football activity exposure of each player, are collected by the medical staff of these clubs [18]. The results of this survey have been extensively published in the scientific literature [10, 19] and helped to identify the most common injuries at that level of football and their characteristics. Moreover, feedback on the club's injury profile is sent periodically by the study group to the clubs, in order to help them to implement specific prevention programmes.

Similarly, FIFA is also conducting surveys in all its main competitions, in male, female and young players [15, 20, 21]. Many national associations, such as in Norway [22], Qatar [13], Sweden [23, 24] and Denmark [23, 25], have implemented prospective epidemiological studies in football, and numerous clubs have developed independent injury registries for their teams [26].

Some of these data are available in the scientific literature and allowed a better delineation of the football injury incidence and patterns in professional footballers. However, due to the many intrinsic and extrinsic risk factors related to football, further research is warranted.

6.5 Injury Incidence in Football

Professional football is an extremely high–injury risk profession. It has been estimated that its overall risk of injury is about 1,000 times higher than for typical industrial occupations generally regarded as high risk [27].

Fortunately, while in the last three decades the training and match volume has remarkably increased due to the improvement in professionalism, the risk of injury has not augmented. This is perhaps due to the development of athlete healthcare and to the implementation of prevention programmes. According to the UEFA Champions League injury study, a professional football team can expect about 50 injuries that cause time loss from play each season, which equates to two injuries per player per season and, on average,

12 % of the squad unavailable due to injury at any point during the season [28]. Most injuries occur during matches, where there is five times higher risk of injury compared with training. This is possibly due to the high intensity of the match play and also to the higher number and energy of duels and impacts. It has to be also considered how there is no possibility, during matches, of seeking medical care if not for few minutes, while during training injuries may be evaluated more carefully by the medical staff and preventive measures can be taken. In particular, the last 15 min of each half are the periods in which players are most vulnerable to injuries, and fatigue plays a major role in this.

In accordance with their aetiology, injuries are classified into overuse and traumatic. For overuse injuries a specific moment in which the injury occurred cannot be identified [29]. While there is evidence that overuse football injuries have a higher incidence during the preseason, different trends of seasonality are linked to different regions and their specific environmental conditions.

In epidemiological studies taking into consideration time-loss injuries, the most common injury location is the thigh, followed by the knee, groin and ankle, while the most common type of injury is strain, followed by contusion and sprain. When considering medical attention injuries, contusions are the most common type of injuries. However, a contusion seldom obliges a player to stop his sport activity; therefore, they are less recorded in studies utilising a time-loss definition. This example highlights the importance of choosing an appropriate injury definition and the implication of a correct interpretation of the results in accordance with the chosen definition.

Hamstring strains are the most common subtype of injuries, and a typical 25-player team can expect about seven hamstring strains per season [19, 28]. It is therefore evident how, like in many other sports, many efforts are focused on the prevention and treatment of this injury.

Despite their relative low incidence (<1 % of all injuries), anterior cruciate ligament (ACL) ruptures are the most attention-drawing pathology in football. Nowadays, in football, ACL lesions are generally treated surgically, and the average time to return to play is between 6 and 7 months. While some decades ago it was a career-ending injury, progress in athletes' healthcare has led to a return-to-football outcome at the same level as before the injury in 90 % of the patients. However, on return to play there is an increased risk of new knee pathologies, especially overuse in aetiology [30]. This may indicate knee abuse due to the absolute necessity of professional footballers of returning to play [31].

The hip and groin are normally the third and fourth most common injury location in prospective epidemiological studies based on a time-loss injury definition [13, 28]. It seems that the real magnitude of the problem has been underestimated so far, due to the relatively difficult of diagnosis of the pathologies located in that region, not always allowing their clear identification and classification. Moreover, groin injuries are often overuse in aetiology and may be underestimated in researches based on a time-loss injury definition [16]. In the champions league, an average of seven groin injuries per team per season has been estimated [32], but, for the above-mentioned reasons, this may be only the tip of the iceberg. In fact, it is well known among clinicians working with footballers that the groin pain syndrome often requires long treatments, with periods of exacerbation of symptoms and recurrences, and an appropriate management of training and match loading by the medical staff.

The ankle is the most common injury location in many sports [33]. While first football epidemiological researches performed in the 1980s were reporting that the ankle was the most common location of injury (around 30 % of total number of injuries), more recent researches show a much inferior incidence rate [34]. This decreasing trend can be the result of prevention strategies (e.g. neuromuscular training, bracing, taping) which have proved to be effective in reducing the incidence of ankle sprain, or this may be due to changes of the rules of the game which have been applied, such as a red card for a tackle from behind. In any case, this finding has to be taken into account as this may be an example of the possibility of reducing injuries in football.

Women's football has gained more and more prominence in the last years and with the number of participants quickly increasing together with the discipline's professionalism. While males have a general higher risk of injury compared with female players, the risk of sustaining a moderate to severe injury (>1 week absence) does not vary between men and women. Injury patterns are also basically comparable, but women have relatively more knee injuries, while men more groin pathologies [35]. Female players are particularly more vulnerable to ACL injuries than men. It has been estimated that the risk is two to three time higher than their male counterparts. Females also tend to sustain their ACL injury at a younger age and have a higher risk of injury especially during match play, although no significant gender-related dissimilarity has been described during training sessions [35]. These data have provided the basis for an extensive scientific research on the prevention for ACL injury in female footballers.

Young footballers' injuries are more overuse in aetiology compared with adults. Of vital importance seems to be a strict training load monitoring. The huge differences in growth between subjects and the unpreparedness to too intense training loads may be a serious risk factor [36–38].

In general, a reinjury is defined as an injury at the same side and location of a previous lesion. When it occurs within 2 months from the return to play, it is usually defined as early reinjury and is often seen as a failure of the treatment.

Therefore, reinjury rate is sometimes considered as an instrument to measure the skills of medical staff. In fact,

while injury frequency may be mostly affected by technical and performance activities, reinjuries, as determined by an early return to play and incomplete rehabilitation, are often associated with medical staff performance. In more recent football studies, a reinjury rate between 12 and 30 % has been reported [10, 24, 39]. Also, reinjuries are generally associated with higher severity (return-to-play time) compared with first injuries. This relatively high incidence and severity underline the importance of correct guidelines in the return-to-play decision. There is a need for new football-specific medical and physical tests to assess athletes before they return to play. Objective data could help the clinician avoid an attitude that is too aggressive (which could increase the risk of reinjury) or too conservative (which would have a negative impact on the layoff time of the athletes).

These data highlight the importance of the medical staff, whose role in many clubs and federations is still undervalued. Often, investments on medical personnel and equipment are still overlooked in professional clubs. There is debate on the methods of medical staff recruitment and its effective impact on the economy of the clubs [40]. Undeniably, however, epidemiological data suggest that, due to the impact of injuries, professional football managers should at least carefully evaluate the possibility of providing their athletes with the best medical care, with the aim to preserve their health but also to increase their availability and performance.

6.6 Risk Factors

In football, injury risk is multifactorial [27]. Several intrinsic and extrinsic risk factors have to be taken into consideration.

Among the intrinsic risk factors, age, career duration and previous injury have been shown having a negative effect on the athlete injury risk. Players with positive injury history have been shown to be two to three times more likely to suffer an identical injury in the subsequent season [24]. Mechanical instability in ankles or knees, joint laxity or functional instability also seems to predispose players to injuries, in particular of the hamstrings, groin and knee [41–45]. Other potential intrinsic risk factors, like ethnicity and mental characteristics, may play a role, but the scientific literature is still inconclusive and further research is warranted.

The analysis of intrinsic risk factor may allow clinicians to prepare individual programmes of prevention at the beginning of the season.

The influence of extrinsic risk factors needs to be analysed as well.

Physical and psychosocial stress appears to increase the injury risk. However, the role of testing the level of stress of the footballers is still generally underestimated. In order to prevent injuries, a close training load monitoring seems at least necessary.

As stated initially by Ekstrand et al. [4] and confirmed by successive researchers [46–49], a high training/match ratio is a protective factor in football. This is probably due to the fact that physically well-trained and mentally prepared players can bear the physical stress during the game with a reduced risk of injury.

Also, the relative shortness of the period of physiological pre-season preparation can be considered a potential contributing factor to the occurrence of accidents [49].

The "economic value" of the game is today definitely higher than previously. Also, the constant pressure of the media is contributing to increase the stress on footballers and therefore may put them at further risk. In addition to this, also the increased performance during the game, the different training methodology and technical and tactical innovations are aspects to consider in modern football. Indeed, the widespread use of tactical solutions, such as the pressing, the offside trap and the double marking, made at maximum intensity, is a potential risk factor for injury.

Moreover, modern football involves tactical situation in which many players are present in a limited area of the field, increasing the possibility of contacts. In addition to this, the exasperated physical preparation may predispose the players to high-force contacts which can result in acute (ligament injuries of the knee, in particular ACL lesions, tendon ruptures, etc.) and chronic (tendonitis, enthesitis, chondropathies, etc.) pathologies.

There is evidence that the professional players are nowadays heavier and taller than some decades ago [49]. It can be hypothesised that these parameters may increase the energy of the contacts and therefore the injury risk. However, this thesis has not been confirmed by scientific data, so far. Several researches showed also a greater muscle strength in the lower limbs of the current professional player [43, 50]. Also in this case, it can be speculated that this may have increased the injury risk; on the other hand, however, this can be also considered a protective factor.

As previously discussed, a congested calendar and relative limited time for training, recovery and pre-season preparation are potential contributing factors to the occurrence of accidents. However, it seems that a period of 3 days between matches and a correct rotation of the players may help to avoid an increase of the injury risk [51–54]. Despite there are no studies confirming this, also the pressure of the media is contributing to increase the pressure on footballers, especially of elite level, and may affect the injury risk.

The association between the playing position and injury risk is still undefined. While the majority of the studies showed no relation [55–58], there is some evidence of a higher incidence of injuries in defenders [59], halfbacks [9] and midfielders [27, 60]. As a confounder factor, modern football requires defending actions in forwards and attaching phases of the game in defenders. Moreover, players may

change role during the season or even during a game. These may be reasons of an indeterminate link between injury risk and playing position.

Football pitches have certainly to be taken into consideration as a potential extrinsic risk factor. Despite the results of the scientific research are still controversial [11, 61, 62], it seems that there is not an increased risk of injury associated with playing on last-generation artificial turfs. However, ankle sprain incidence is increased, while quadriceps strains decreased on this surface [22, 62, 63]. This may suggest the use of specific preventive measures for teams playing or training regularly on this surface.

Moreover, weather and pitch conditions seem to affect injuries, creating regional differences in match injury incidence. Teams from northern Europe have a higher general risk of injury than the teams from southern Europe, possibly due to poorer climate and surface conditions. Conversely, there is a trend towards an increased risk of ACL injury in Mediterranean countries, where it has been hypothesised that the pitches, with higher rotational and traction forces, may be the most important risk factor [64]. These data seems to be confirmed by some researches on Australian football [65, 66] and underline the importance of developing regional epidemiological studies in order to define and characterise the particular injuries for each country.

In this view, attention must be also paid on the choice of footwear and cleats, in order to avoid excessive ground-shoes rotational forces that may increase the number of injuries, in particular located to the knee.

With the 2012 Olympic Games and the 2014 FIFA World Cup both being organised during the period of Ramadan (Islamic holy month of fasting), there was a debate about the possible effects of fasting on physical performance and injury.

A study of injury epidemiology in Qatari footballers [67] has allowed the investigation of the influence of Ramadan on football injury incidence and patterns. In a population with a majority of Muslim footballers competing in a Muslim country, no significant difference of injury incidence, characteristics and patterns has been shown during Ramadan compared to the rest of the football season. On the contrary, there is some evidence that non-Muslim footballers competing in a Muslim country suffer more during this time as they struggle to cope with the changes in social and sport life during this period such as training time modifications.

In modern football, despite this has never been explored in scientific researches, the frequent changes of coaches and technical staff can be considered a possible risk factor. Another aspect to take onto account is the high number of players for each professional team: if this may ensure an appropriate turnover allowing a better recovery of tired and injured players and therefore can be seen as a protective factor, on the other hand, the pressure and the competitiveness between the players may increase, leading to a higher risk of injury. While some of these risk factors are not easy to be corrected, the player's lifestyle represents a central risk factor that can be quickly modified or corrected. For example, smoking is an element of absolute toxicity, and alcohol, even when taken occasionally, can be counterproductive for the performance [49].

Also an excessive use of drugs in order to continue sport activity despite an injury or to speed up the recovery time should be considered as an important risk factor to the footballers' health [49].

On top of these considerations, fair play among players, coaches and managers as well as the central role of the referee in the protection of the players are key risk factors that the national and international federation should continuously take into consideration, in order to perform the adapted adjustments.

References

1. Hagglund M, Walden M, Magnusson H et al (2013) Injuries affect team performance negatively in professional football: an 11-year follow-up of the UEFA Champions League injury study. Br J Sports Med 47(12):738–742
2. Eirale C, Tol JL, Farooq A et al (2013) Low injury rate strongly correlates with team success in Qatari professional football. Br J Sports Med 47(12):807–808
3. Ekstrand J, Gillquist J (1982) The frequency of muscle tightness and injuries in soccer players. Am J Sports Med 10:75–78
4. Ekstrand J, Gillquist J, Moller M et al (1983) Incidence of soccer injuries and their relation to training and team success. Am J Sports Med 11:63–67
5. Ekstrand J, Tropp H (1990) The incidence of ankle sprains in soccer. Foot Ankle 11:41–44
6. Ekstrand J, Gillquist J (1983) The avoidability of soccer injuries. Int J Sports Med 4:124–128
7. Herrero H, Salinero JJ, Del Coso J (2014) Injuries among Spanish male amateur soccer players: a retrospective population study. Am J Sports Med 42(1):78–85
8. Hawkins RD, Fuller CW (1998) An examination of the frequency and severity of injuries and incidents at three levels of professional football. Br J Sports Med 32:326–332
9. McGregor JC, Rae A (1995) A review of injuries to professional footballers in a premier football team (1990–93). Scott Med J 40:16–18
10. Walden M, Hagglund M, Ekstrand J (2005) UEFA Champions League study: a prospective study of injuries in professional football during the 2001–2002 season. Br J Sports Med 39:542–546
11. Soligard T, Bahr R, Andersen TE (2012) Injury risk on artificial turf and grass in youth tournament football. Scand J Med Sci Sports 22:356–361
12. Junge A, Dvorak J (2007) Injuries in female football players in top-level international tournaments. Br J Sports Med 41(Suppl 1):i3–i7
13. Eirale C, Farooq A, Smiley FA et al (2013) Epidemiology of football injuries in Asia: a prospective study in Qatar. J Sci Med Sport 16:113–117
14. Fuller CW, Ekstrand J, Junge A et al (2006) Consensus statement on injury definitions and data collection procedures in studies of football (soccer) injuries. Clin J Sport Med 16:97–106
15. Junge A, Dvorak J, Graf-Baumann T, Peterson L (2004) Football injuries during FIFA tournaments and the Olympic Games, 1998–2001: development and implementation of an injury-reporting system. Am J Sports Med 32:80S–89S

16. Bahr R (2009) No injuries, but plenty of pain? On the methodology for recording overuse symptoms in sports. Br J Sports Med 43: 966–972
17. Clarsen B, Myklebust G, Bahr R (2013) Development and validation of a new method for the registration of overuse injuries in sports injury epidemiology: the Oslo Sports Trauma Research Centre (OSTRC) overuse injury questionnaire. Br J Sports Med 47: 495–502
18. Hagglund M, Walden M, Bahr R, Ekstrand J (2005) Methods for epidemiological study of injuries to professional football players: developing the UEFA model. Br J Sports Med 39:340–346
19. Ekstrand J, Hagglund M, Walden M (2011) Epidemiology of muscle injuries in professional football (soccer). Am J Sports Med 39:1226–1232
20. Junge A, Dvorak J, Graf-Baumann T (2004) Football injuries during the World Cup 2002. Am J Sports Med 32:23S–27S
21. Dvorak J, Junge A, Grimm K, Kirkendall D (2007) Medical report from the 2006 FIFA World Cup Germany. Br J Sports Med 41: 578–581; discussion 581
22. Bjorneboe J, Bahr R, Andersen TE (2010) Risk of injury on third-generation artificial turf in Norwegian professional football. Br J Sports Med 44:794–798
23. Hagglund M, Walden M, Ekstrand J (2005) Injury incidence and distribution in elite football–a prospective study of the Danish and the Swedish top divisions. Scand J Med Sci Sports 15:21–28
24. Hagglund M, Walden M, Ekstrand J (2006) Previous injury as a risk factor for injury in elite football: a prospective study over two consecutive seasons. Br J Sports Med 40:767–772
25. Holmich P, Thorborg K, Dehlendorff C et al (2014) Incidence and clinical presentation of groin injuries in sub-elite male soccer. Br J Sports Med 48:1245–1250
26. Hassabi M, Mohammad-Javad Mortazavi S, Giti MR et al (2010) Injury profile of a professional soccer team in the premier league of Iran. Asian J Sports Med 1:201–208
27. Fuller CW, Junge A, Dvorak J (2012) Risk management: FIFA's approach for protecting the health of football players. Br J Sports Med 46:11–17
28. Ekstrand J, Hagglund M, Walden M (2011) Injury incidence and injury patterns in professional football: the UEFA injury study. Br J Sports Med 45:553–558
29. Orava S (1980) Exertion injuries due to sports and physical exercise. A clinical and statistical study of nontraumatic overuse injuries of the musculoskeletal system of athletes and keep-fit athletes, In edition. University of Oulu, Oulu
30. Walden M, Hagglund M, Ekstrand J (2006) High risk of new knee injury in elite footballers with previous anterior cruciate ligament injury. Br J Sports Med 40:158–162; discussion 158–162
31. Ekstrand J (2011) A 94 % return to elite level football after ACL surgery: a proof of possibilities with optimal caretaking or a sign of knee abuse? Knee Surg Sports Traumatol Arthrosc 19:1–2
32. Werner J, Hagglund M, Walden M, Ekstrand J (2009) UEFA injury study: a prospective study of hip and groin injuries in professional football over seven consecutive seasons. Br J Sports Med 43: 1036–1040
33. Fong DT, Hong Y, Chan LK et al (2007) A systematic review on ankle injury and ankle sprain in sports. Sports Med 37:73–94
34. Ekstrand J, Hagglund M, Kristenson K et al (2013) Fewer ligament injuries but no preventive effect on muscle injuries and severe injuries: an 11-year follow-up of the UEFA Champions League injury study. Br J Sports Med 47:732–737
35. Walden M, Hagglund M, Werner J, Ekstrand J (2011) The epidemiology of anterior cruciate ligament injury in football (soccer): a review of the literature from a gender-related perspective. Knee Surg Sports Traumatol Arthrosc 19:3–10
36. Bergeron MF, McKeag DB, Casa DJ et al (2005) Youth football: heat stress and injury risk. Med Sci Sports Exerc 37:1421–1430
37. Brito J, Rebelo A, Soares JM et al (2011) Injuries in youth soccer during the preseason. Clin J Sport Med 21:259–260
38. Carter CW, Micheli LJ (2011) Training the child athlete: physical fitness, health and injury. Br J Sports Med 45:880–885
39. Eirale C, Hamilton B, Bisciotti G et al (2012) Injury epidemiology in a national football team of the Middle East. Scand J Med Sci Sports 22:323–329
40. Orchard JW (2009) On the value of team medical staff: can the "Moneyball" approach be applied to injuries in professional football? Br J Sports Med 43:963–965
41. Hagglund M, Walden M, Ekstrand J (2013) Risk factors for lower extremity muscle injury in professional soccer: the UEFA Injury Study. Am J Sports Med 41:327–335
42. Engebretsen AH, Myklebust G, Holme I et al (2011) Intrinsic risk factors for acute knee injuries among male football players: a prospective cohort study. Scand J Med Sci Sports 21:645–652
43. Fousekis K, Tsepis E, Poulmedis P et al (2011) Intrinsic risk factors of non-contact quadriceps and hamstring strains in soccer: a prospective study of 100 professional players. Br J Sports Med 45:709–714
44. Engebretsen AH, Myklebust G, Holme I et al (2010) Intrinsic risk factors for groin injuries among male soccer players: a prospective cohort study. Am J Sports Med 38:2051–2057
45. Witvrouw E, Danneels L, Asselman P et al (2003) Muscle flexibility as a risk factor for developing muscle injuries in male professional soccer players. A prospective study. Am J Sports Med 31: 41–46
46. Ostenberg A, Roos H (2000) Injury risk factors in female European football. A prospective study of 123 players during one season. Scand J Med Sci Sports 10:279–285
47. Arnason A, Sigurdsson SB, Gudmundsson A et al (2004) Risk factors for injuries in football. Am J Sports Med 32:5S–16S
48. Hagglund M, Walden M, Ekstrand J (2003) Exposure and injury risk in Swedish elite football: a comparison between seasons 1982 and 2001. Scand J Med Sci Sports 13:364–370
49. Volpi P, Taioli E (2012) The health profile of professional soccer players: future opportunities for injury prevention. J Strength Cond Res 26:3473–3479
50. Requena B, Gonzalez-Badillo JJ, de Villareal ES et al (2009) Functional performance, maximal strength, and power characteristics in isometric and dynamic actions of lower extremities in soccer players. J Strength Cond Res 23:1391–1401
51. Carling C, Le Gall F, Dupont G (2012) Are physical performance and injury risk in a professional soccer team in match-play affected over a prolonged period of fixture congestion? Int J Sports Med 33:36–42
52. Dellal A, Lago-Penas C, Rey E et al (2013) The effects of a congested fixture period on physical performance, technical activity and injury rate during matches in a professional soccer team. Br J Sports Med. [Epub ahead of print]
53. Dupont G, Nedelec M, McCall A et al (2010) Effect of 2 soccer matches in a week on physical performance and injury rate. Am J Sports Med 38:1752–1758
54. Ekstrand J, Walden M, Hagglund M (2004) A congested football calendar and the wellbeing of players: correlation between match exposure of European footballers before the World Cup 2002 and their injuries and performances during that World Cup. Br J Sports Med 38:493–497
55. Engstrom B, Forssblad M, Johansson C, Tornkvist H (1990) Does a major knee injury definitely sideline an elite soccer player? Am J Sports Med 18:101–105
56. Hawkins RD, Fuller CW (1999) A prospective epidemiological study of injuries in four English professional football clubs. Br J Sports Med 33:196–203
57. Ekstrand J, Gillquist J (1983) Soccer injuries and their mechanisms: a prospective study. Med Sci Sports Exerc 15:267–270

58. Ekstrand J, Gillquist J, Liljedahl SO (1983) Prevention of soccer injuries. Supervision by doctor and physiotherapist. Am J Sports Med 11:116–120
59. Peterson L, Junge A, Chomiak J et al (2000) Incidence of football injuries and complaints in different age groups and skill-level groups. Am J Sports Med 28:S51–S57
60. Arnason A, Sigurdsson SB, Gudmundsson A et al (2004) Physical fitness, injuries, and team performance in soccer. Med Sci Sports Exerc 36:278–285
61. Kristenson K, Bjorneboe J, Walden M et al (2013) The Nordic Football Injury Audit: higher injury rates for professional football clubs with third-generation artificial turf at their home venue. Br J Sports Med 47:775–781
62. Sousa P, Rebelo A, Brito J (2012) Injuries in amateur soccer playerson artificial turf: a one-season prospective study. Phys Ther Sport 14(3):146–151
63. Ekstrand J, Hagglund M, Fuller CW (2011) Comparison of injuries sustained on artificial turf and grass by male and female elite football players. Scand J Med Sci Sports 21:824–832
64. Walden M, Hagglund M, Orchard J et al (2013) Regional differences in injury incidence in European professional football. Scand J Med Sci Sports 23(4):424–430
65. Orchard J, Rodas G, Til L et al (2008) A hypothesis: could portable natural grass be a risk factor for knee injuries? J Sports Sci Med 7:184–190
66. Orchard J, Seward H, McGivern J, Hood S (1999) Rainfall, evaporation and the risk of non-contact anterior cruciate ligament injury in the Australian Football League. Med J Aust 170:304–306
67. Eirale C, Tol JL, Smiley F et al (2013) Does Ramadan affect the risk of injury in professional football? Clin J Sport Med 23(4): 261–266

Return to Sport (General Aspects)

7

Ryan R. Sullivan, Antony Hazel, Sarunas Skadas, and Pietro M. Tonino

7.1 Introduction

As the population has become more active, anterior cruciate ligament (ACL) injuries have become more common. More and more individuals are choosing ACL reconstruction in order to attempt to return to their previous functional level. Much of the previous research regarding ACL reconstruction has revolved around optimizing surgical variables. A new focus has arisen attempting to determine when patients are ready to return to full activity. ACL grafts continue to mature after one year which is well past the time when most athletes are cleared to return [1, 2]. A balance exists in trying to allow for one's biology to recover and heal from surgery while minimizing the time off. The following chapter explores the multiple factors that contribute to one's ability to return to sport and the current tools available that can assist in making an accurate determination of an athlete's readiness.

7.2 Physical Recovery

Successful recovery from ACL reconstruction begins with prehabilitation. Prior to surgery, the goals are to reduce swelling, inflammation, and pain which can limit knee range of motion (ROM) and inhibit muscle activation. Knee ROM is optimized with emphasis on full knee extension; and muscle strength maximized to help prevent atrophy [3, 4]. This is important for minimizing postoperative complications such as arthrofibrosis and persistent muscle weakness [3–6].

After surgery, the focus of rehabilitation is to regain strength, dynamic control, and functional stability on the injured extremity. Mechanical and dynamic knee stability is an important indicator of the ability to protect the healing graft. Before initiating specific sport training, certain milestones must be met to verify that the above have been achieved.

The patient's history and physical exam are important in assessing stability. Patients often report episodes of "giving way" in the affected extremity when laxity is present. The pivot shift test and Lachman's maneuver can also identify laxity within the grafts and thus patients who may be susceptible to reinjury [7, 8]. Increased laxity is important to recognize because it may be associated with altered contact loading of the articular surfaces and inferior structural properties of the graft [4, 9].

There are other more functional means to assess stability and a "knee at risk." The squat and hold is one such test, requiring the athlete to hold a one-legged stance position with the knee in flexion to accentuate any residual strength deficits in the injured limb. Balance tests on an unstable platform can be used to assess overall postural stability and neuromuscular control deficits.

After sufficient knee stability has been demonstrated, patients progress to more advanced assessments that are designed to evaluate strength, coordination, functional stability, and comparison between the injured and uninjured limbs. These factors are indicators of neuromuscular control, which along with biomechanical asymmetry is a main contributor to primary and secondary ACL injury risk when deficient [7, 10–12].

Test results are often reported using a limb symmetry index (LSI), which is a ratio comparing the injured limb to the uninjured limb. Normative data on healthy subjects has shown an LSI of 85 % or greater to be consistent with normal strength and function, and most researchers report an LSI result of 85–90 % for each individual test as satisfactory to allow return to sport [5, 10, 13].

The assessment of strength is a key component when making the decision for return to sport [7, 10, 14–16]. Adequate quadriceps strength is necessary for force generation and attenuation about the knee as well as for

R.R. Sullivan, MD • A. Hazel, MD • S. Skadas, PT, DPT
P.M. Tonino, MD (✉)
Department of Orthopaedic Surgery and Rehabilitation,
Loyola University Medical Center,
2160 S. First Ave, Maguire Building Suite 1700,
Maywood, IL 60153, USA
e-mail: rysullivan@lumc.edu; ahazel@lumc.edu;
sskadas@lumc.edu; ptonino@lumc.edu

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_7,

function and gait restoration [10, 17]. Hamstring strength is critical for knee stabilization and landing mechanics [10, 15, 16, 18–20]. Strength can be measured with isokinetic knee flexion and extension tests, squats, and leg press. The ratio of hamstring to quadriceps (H:Q) strength within the injured limb is an important indicator of the hamstrings' ability to decrease anterior tibial displacement and shear generated by the anterior pull of the quadriceps, subsequently reducing ACL strain and injury risk [10, 15, 18–20]. The H:Q ratio is a main determinant for primary ACL injury risk and also correlates to self-reported knee function [10, 21].

Hip abduction strength can also be assessed as it influences initial contact and knee valgus angles during drop-landing tasks. Altered hip kinematics and excessive knee valgus angles predict secondary ACL injury after reconstruction [12]. Power measurements can also be employed as a more specific gauge of the use of strength in sports, as the rapid production of force is important for performance and injury protection [22].

The restoration of neuromuscular control after ACL reconstruction depends not only on muscle strength but also on dynamic knee stability and limb performance symmetry, which aid in attaining successful functional, subjective, and return-to-sport outcomes [8, 10, 14, 23–25].

Hop tests are frequently instituted as a reliable measure of dynamic stability, reflecting integration of neuromuscular control, strength and power, endurance, and confidence in the limb [26]. Single-leg hop tests correlate with quadriceps strength, limb stability, subjective outcomes, and return-to-sport outcomes [14, 23, 24, 27–31]. Athletes who attain hop test results greater than 85 % of the contralateral extremity are significantly more likely to return to pre-injury level of sports at 1 year [32]. Frequently used hop tests include single-limb hop for distance, single-limb crossover hop for distance, single-limb triple hop for distance, single-limb timed hop for distance, and single-limb vertical power hop.

Additional studies have found that hop testing under fatigued conditions affords a more sensitive way to detect persistent functional limitations. In one study, only two-thirds of athletes performed satisfactorily under fatigued conditions at 1 year following ACL reconstruction despite demonstrating greater than 90 % hop capacity when not fatigued [33]. A single-leg hop test at 11 months postoperatively preceded by pre-exhaustion exercise has been shown to increase the sensitivity of the single-leg hop. One study reported a greater than 90 % success rate under non-fatigued conditions which declined to 68 % demonstrating abnormal hop symmetry following a pre-exhaustion exercise protocol [34]. Testing after the lower extremity is fatigued to failure may be more pertinent measure of endurance, replicating the events during sports participation.

A battery of tests possesses a greater ability than any single test alone to discriminate between injured and non-injured sides [35–37]. A prospective cohort study [35] found that at 24 months postoperatively, rates of attaining a 90 % LSI for strength and hop test batteries were 48 and 44 %, respectively, compared to more than 90 % when the tests were evaluated individually. When combining the strength and hop test batteries, success rates were only 22 %. The combination of single-leg hop for distance, vertical jump, and side hop was found to possess a sensitivity of 91 % and accuracy of 88 % for detecting abnormal limb symmetry in patients who underwent ACL reconstruction 6 months previously [37]. In another study, evaluating a strength test battery of knee extension, knee flexion, and leg press was found to be more sensitive compared with any of the three tests individually. Eighty-six percent of patients had abnormal limb symmetry in knee extension at 6 months postoperatively, along with 42 % of patients in knee flexion and 61 % in leg press [36]. When combined, 95 % of patients did not meet the requirements of greater than 90 % LSI for all three tests. Another group found that test sensitivity improved from 52 to 62 % by using two hop tests rather than one [38].

In addition to objective measures, subjective self-evaluation of knee function is an important determinant of an athlete's ability to return to sport. Self-reporting of knee function, instability, and knee symptoms such as pain and swelling are often the factors most strongly associated with return-to-sport status [23, 39, 40]. There are numerous instruments used to assess subjective outcomes in patients with knee injuries, such as the commonly used scales International Knee Documentation Committee (IKDC) Subjective Knee Form, Lysholm knee scale, and the Cincinnati knee score. These scales are reliable and valid for evaluation of postoperative symptoms, function, and sports activity after an ACL reconstruction [7, 41–43]. The IKDC is effective and responsive to changes in perceived function over time [7, 44, 45].

7.3 Psychological Recovery

Many athletes who achieve normal knee function following reconstruction are unable to return to pre-injury levels because of psychological factors [46]. Up to 30 % of athletes who do not return report fear of reinjury as the primary reason [40, 46, 47]. Psychological readiness to return to sport may lag behind physical readiness. Return prior to psychological readiness can lead to worsening psychological state as well as decreased performance and subsequent reinjury [48–50]. Recognition of maladaptive psychological processes that may negatively affect an athlete's rehabilitation is therefore essential.

There are a number of tools for assessing psychological recovery after ACL reconstruction. One of the most widely used postsurgical psychological assessments is the Tampa Scale of Kinesiophobia (TSK), which measures fear of movement and

reinjury [47, 51]. The TSK and its shortened version more specific to ACL-reconstructed athletes is a survey that demonstrates prognostic value in ACL-reconstructed athletes, predicting return to sport within 12 months when measured at 4 months postoperatively [48, 51]. Another scale, the 12-item ACL-Return to Sport after Injury (ACL-RSI), measures frustration, fear, and confidence. The value of ACL-RSI is that it can be used preoperatively as well as at 4–6 months postoperatively as a predictor of return to sport within 12 months [48, 50, 52]. Other assessment scales measure additional psychological aspects such as the emotional response to injury, an athlete's self-efficacy, confidence, motivation, perception of control, and pain-catastrophizing behavior. These are important determinants of rehabilitation effort, perceived and actual rehabilitation outcomes, objective and subjective knee function, and return to pre-injury sports activity level [51, 53, 54].

Surgeons need to recognize these psychological issues in order to understand the postoperative recovery process and counsel the athlete as needed as they attempt a return to sport. Screening for maladaptive psychological responses to identify at-risk athletes should be performed early on so that psychological recovery can parallel physical rehabilitation. Interventions that may positively influence psychological recovery include peer modeling, relaxation, guided imagery, goal setting, provision of clear postoperative and rehabilitation instructions, and improvement of coping skills and self-efficacy beliefs [55]. Future research into improving psychological readiness should include further development of interventions specific to ACL reconstruction rehabilitation to provide standardized instruments for improving rehabilitation and return to sport.

7.4 Discussion

Historically, return-to-sport clearance has been based on time from surgery. Many athletes received clearance around the 6-month time point to allow for postoperative healing while progressing through rehabilitation [10, 25]. However, athletes recover and progress through rehabilitation at variable rates. When assessed after sports participation, many athletes demonstrate functional deficits that are independent of time from surgery [56]. Therefore, the return-to-sport decision should be based on the individual athlete's recovery and monitored using specified objective and subjective criteria. Objective data should be used to ensure that the athlete possesses adequate knee strength and stability to protect the healing graft and safely return to sport. These measures help guide the athlete to provide a goal-oriented rehabilitation. These criteria include achievement of symmetrical joint motion, strength, and functional performance between the injured and uninjured limbs, as well as subjective and psychological scores within the norm, prior to return to sport.

Different centers have studied the benchmarks required for an athlete to return to sport. One group advocates return to sport when full ROM is achieved, hop and strength test results are at least 85 % of the uninjured extremity, the hamstring/quadriceps strength ratio is less than 15 % difference compared to the uninjured side, and the patient tolerates sport-specific activities with no increase in pain or swelling [57]. Another proposes criteria including a LSI of 90 % for strength and functional testing as well as a score of 90 % on two self-reported outcome measures. They saw 40 % of ACL-reconstructed athletes cleared for return to practice at 6 months and 73 % cleared at 12 months [5]. Those who did not meet criteria continued rehabilitation, focusing on areas in which they were deficient. It is important to note that although correlations exist between strength and stability testing with subjective and functional outcomes, there is a lack of data directly associating success in these areas with reduced risk of reinjury upon return to sport. Tests that accurately define adequate neuromuscular control are not clear [7, 10]. It is conceivable that normal strength and function promote neuromuscular control and graft protection leading to lower reinjury rate; therefore current testing methods evaluate these factors. However, future outcome research is needed to correlate successful strength and functional indices with lower risk of reinjury.

The ability to return to sport following ACL reconstruction requires the restoration of physical function and psychological readiness in addition to specific patient, sport, injury, and rehabilitation factors. Premature return to sport may predispose the patient to further injury, with incidence of reinjury to the graft or rupture of the contralateral ACL ranging from 6 to 32 % [10]. For athletes who successfully return to activity, one in four will go on to a second knee injury [10]. Of the many risk factors for graft injury or contralateral ACL injury, history of ACL reconstruction and return to high-level competitive pivoting/cutting/jumping sports are among the greatest contributors [10, 11, 58]. Primarily implicated in this increased risk is the asymmetric and impaired neuromuscular control and lower extremity biomechanics that both lead to and result from the initial ACL injury [10–12]. Injury prevention programs emphasizing neuromuscular training to reduce these modifiable impairments can potentially reduce the rate of ACL injuries by up to 50 % [59]. Plyometric testing such as drop vertical jumps and tuck jumps can be used to assess abnormalities in neuromuscular control, balance, symmetrical limb loading, force contribution, and force attenuation [5, 7, 10]. This allows observation of asymmetries in lower extremity positioning and movement as well as detrimental biomechanics such as decreased knee flexion and valgus knee angles upon landing that increase ACL shear and strain forces and risk of ACL injury [11, 12, 20, 60]. The evaluator can then direct the correction of these deficiencies and improvement of technique and limb

symmetry [5, 7, 12]. Incorporating serial functional and strength testing is essential to decrease the risk of secondary ACL injury and upon reintegration into sport.

7.5 Summary

ACL reconstruction is performed for many athletes with ACL rupture to prevent instability and further injury. Deficits in strength and dynamic stability may persist following reconstruction and must be corrected through postoperative rehabilitation. Whereas in the past, the decision to return to sport following ACL reconstruction was often based on time from surgery, current literature leads the surgeon toward using a criteria-based progression through rehabilitation. Requiring the athlete to meet specified subjective and objective goals prior to sport clearance allows for a more individualized postoperative rehabilitation, which is necessary since individual athletes may not progress at the same rate. The "timeframe" for an athlete's rehabilitation should be determined by these criteria, which typically rate the injured against the uninjured extremity. Using a criteria-based progression through rehabilitation and return to sport may be a more effective method of assuring a safe and successful return to sport. In addition, neuromuscular deficits and psychological hindrances must be recognized and addressed in order to ensure an athlete's complete readiness for return to sport.

References

1. Abe S, Kurosaka M, Iguchi T, Yoshiya S, Hirohata K (1993) Light and electron microscopic study of remodeling and maturation process in autogenous graft for anterior cruciate ligament reconstruction. Arthroscopy 9(4):394–405
2. Rougraff B, Shelbourne KD, Gerth PK, Warner J (1993) Arthroscopic and histologic analysis of human patellar tendon autografts used for anterior cruciate ligament reconstruction. Am J Sports Med 21(2):277–284
3. Wilk KE, Macrina LC, Cain EL, Dugas JR, Andrews JR (2012) Recent advances in the rehabilitation of anterior cruciate ligament injuries. J Orthop Sports Phys Ther 42(3):153–171
4. Cascio BM, Culp L, Cosgarea AJ (2004) Return to play after anterior cruciate ligament reconstruction. Clin Sports Med 23(3):395–408, ix
5. Adams D, Logerstedt DS, Hunter-Giordano A, Axe MJ, Snyder-Mackler L (2012) Current concepts for anterior cruciate ligament reconstruction: a criterion-based rehabilitation progression. J Orthop Sports Phys Ther 42(7):601–614
6. Eitzen I, Holm I, Risberg MA (2009) Preoperative quadriceps strength is a significant predictor of knee function two years after anterior cruciate ligament reconstruction. Br J Sports Med 43(5):371–376
7. Myer GD, Paterno MV, Ford KR, Quatman CE, Hewett TE (2006) Rehabilitation after anterior cruciate ligament reconstruction: criteria-based progression through the return-to-sport phase. J Orthop Sports Phys Ther 36(6):385–402
8. Kocher MS, Steadman JR, Briggs KK, Sterett WI, Hawkins RJ (2004) Relationships between objective assessment of ligament stability and subjective assessment of symptoms and function after anterior cruciate ligament reconstruction. Am J Sports Med 32(3):629–634
9. Beynnon BD, Johnson RJ, Toyama H, Renstrom PA, Arms SW, Fischer RA (1994) The relationship between anterior-posterior knee laxity and the structural properties of the patellar tendon graft. A study in canines. Am J Sports Med 22(6):812–820
10. Hewett TE, Di Stasi SL, Myer GD (2013) Current concepts for injury prevention in athletes after anterior cruciate ligament reconstruction. Am J Sports Med 41(1):216–224
11. Sward P, Kostogiannis I, Roos H (2010) Risk factors for a contralateral anterior cruciate ligament injury. Knee Surg Sports Traumatol Arthrosc 18(3):277–291
12. Paterno MV, Schmitt LC, Ford KR, Rauh MJ, Myer GD, Huang B et al (2010) Biomechanical measures during landing and postural stability predict second anterior cruciate ligament injury after anterior cruciate ligament reconstruction and return to sport. Am J Sports Med 38(10):1968–1978
13. Barber SD, Noyes FR, Mangine RE, McCloskey JW, Hartman W (1990) Quantitative assessment of functional limitations in normal and anterior cruciate ligament-deficient knees. Clin Orthop Relat Res 255:204–214
14. Sekiya I, Muneta T, Ogiuchi T, Yagishita K, Yamamoto H (1998) Significance of the single-legged hop test to the anterior cruciate ligament-reconstructed knee in relation to muscle strength and anterior laxity. Am J Sports Med 26(3):384–388
15. Li RC, Maffulli N, Hsu YC, Chan KM (1996) Isokinetic strength of the quadriceps and hamstrings and functional ability of anterior cruciate deficient knees in recreational athletes. Br J Sports Med 30(2):161–164
16. Tsepis E, Vagenas G, Giakas G, Georgoulis A (2004) Hamstring weakness as an indicator of poor knee function in ACL-deficient patients. Knee Surg Sports Traumatol Arthrosc 12(1):22–29
17. Lewek M, Rudolph K, Axe M, Snyder-Mackler L (2002) The effect of insufficient quadriceps strength on gait after anterior cruciate ligament reconstruction. Clin Biomech (Bristol, Avon) 17(1):56–63
18. Draganich LF, Vahey JW (1990) An in vitro study of anterior cruciate ligament strain induced by quadriceps and hamstrings forces. J Orthop Res 8(1):57–63
19. Withrow TJ, Huston LJ, Wojtys EM, Ashton-Miller JA (2008) Effect of varying hamstring tension on anterior cruciate ligament strain during in vitro impulsive knee flexion and compression loading. J Bone Joint Surg Am 90(4):815–823
20. Fujiya H, Kousa P, Fleming BC, Churchill DL, Beynnon BD (2011) Effect of muscle loads and torque applied to the tibia on the strain behavior of the anterior cruciate ligament: an in vitro investigation. Clin Biomech (Bristol, Avon) 26(10):1005–1011
21. Myer GD, Ford KR, Khoury J, Succop P, Hewett TE (2010) Development and validation of a clinic-based prediction tool to identify female athletes at high risk for anterior cruciate ligament injury. Am J Sports Med 38(10):2025–2033
22. Kraemer WJ, Adams K, Cafarelli E, Dudley GA, Dooly C, Feigenbaum MS et al (2002) American College of Sports Medicine position stand. Progression models in resistance training for healthy adults. Med Sci Sports Exerc 34(2):364–380
23. Sernert N, Kartus J, Kohler K, Stener S, Larsson J, Eriksson BI et al (1999) Analysis of subjective, objective and functional examination tests after anterior cruciate ligament reconstruction. A follow-up of 527 patients. Knee Surg Sports Traumatol Arthrosc 7(3):160–165
24. Wilk KE, Romaniello WT, Soscia SM, Arrigo CA, Andrews JR (1994) The relationship between subjective knee scores, isokinetic testing, and functional testing in the ACL-reconstructed knee. J Orthop Sports Phys Ther 20(2):60–73

25. Kvist J (2004) Rehabilitation following anterior cruciate ligament injury: current recommendations for sports participation. Sports Med 34(4):269–280
26. Reid A, Birmingham TB, Stratford PW, Alcock GK, Giffin JR (2007) Hop testing provides a reliable and valid outcome measure during rehabilitation after anterior cruciate ligament reconstruction. Phys Ther 87(3):337–349
27. Fitzgerald GK, Lephart SM, Hwang JH, Wainner RS (2001) Hop tests as predictors of dynamic knee stability. J Orthop Sports Phys Ther 31(10):588–597
28. Keays SL, Bullock-Saxton JE, Newcombe P, Keays AC (2003) The relationship between knee strength and functional stability before and after anterior cruciate ligament reconstruction. J Orthop Res 21(2):231–237
29. Petschnig R, Baron R, Albrecht M (1998) The relationship between isokinetic quadriceps strength test and hop tests for distance and one-legged vertical jump test following anterior cruciate ligament reconstruction. J Orthop Sports Phys Ther 28(1):23–31
30. Fitzgerald GK, Axe MJ, Snyder-Mackler L (2000) A decision-making scheme for returning patients to high-level activity with nonoperative treatment after anterior cruciate ligament rupture. Knee Surg Sports Traumatol Arthrosc 8(2):76–82
31. Logerstedt D, Grindem H, Lynch A, Eitzen I, Engebretsen L, Risberg MA et al (2012) Single-legged hop tests as predictors of self-reported knee function after anterior cruciate ligament reconstruction: the Delaware-Oslo ACL cohort study. Am J Sports Med 40(10):2348–2356
32. Ardern CL, Webster KE, Taylor NF, Feller JA (2011) Return to the preinjury level of competitive sport after anterior cruciate ligament reconstruction surgery: two-thirds of patients have not returned by 12 months after surgery. Am J Sports Med 39(3):538–543
33. Augustsson J, Thomee R, Linden C, Folkesson M, Tranberg R, Karlsson J (2006) Single-leg hop testing following fatiguing exercise: reliability and biomechanical analysis. Scand J Med Sci Sports 16(2):111–120
34. Augustsson J, Thomee R, Karlsson J (2004) Ability of a new hop test to determine functional deficits after anterior cruciate ligament reconstruction. Knee Surg Sports Traumatol Arthrosc 12(5): 350–356
35. Thomee R, Neeter C, Gustavsson A, Thomee P, Augustsson J, Eriksson B et al (2012) Variability in leg muscle power and hop performance after anterior cruciate ligament reconstruction. Knee Surg Sports Traumatol Arthrosc 20(6):1143–1151
36. Neeter C, Gustavsson A, Thomee P, Augustsson J, Thomee R, Karlsson J (2006) Development of a strength test battery for evaluating leg muscle power after anterior cruciate ligament injury and reconstruction. Knee Surg Sports Traumatol Arthrosc 14(6): 571–580
37. Gustavsson A, Neeter C, Thomee P, Silbernagel KG, Augustsson J, Thomee R et al (2006) A test battery for evaluating hop performance in patients with an ACL injury and patients who have undergone ACL reconstruction. Knee Surg Sports Traumatol Arthrosc 14(8):778–788
38. Noyes FR, Barber SD, Mangine RE (1991) Abnormal lower limb symmetry determined by function hop tests after anterior cruciate ligament rupture. Am J Sports Med 19(5):513–518
39. Lentz TA, Zeppieri G Jr, Tillman SM, Indelicato PA, Moser MW, George SZ et al (2012) Return to preinjury sports participation following anterior cruciate ligament reconstruction: contributions of demographic, knee impairment, and self-report measures. J Orthop Sports Phys Ther 42(11):893–901
40. Lee DY, Karim SA, Chang HC (2008) Return to sports after anterior cruciate ligament reconstruction - a review of patients with minimum 5-year follow-up. Ann Acad Med Singapore 37(4):273–278
41. Tegner Y, Lysholm J (1985) Rating systems in the evaluation of knee ligament injuries. Clin Orthop Relat Res 198:43–49
42. Barber-Westin SD, Noyes FR, McCloskey JW (1999) Rigorous statistical reliability, validity, and responsiveness testing of the Cincinnati knee rating system in 350 subjects with uninjured, injured, or anterior cruciate ligament-reconstructed knees. Am J Sports Med 27(4):402–416
43. Irrgang JJ, Anderson AF, Boland AL, Harner CD, Kurosaka M, Neyret P et al (2001) Development and validation of the international knee documentation committee subjective knee form. Am J Sports Med 29(5):600–613
44. Irrgang JJ, Anderson AF, Boland AL, Harner CD, Neyret P, Richmond JC et al (2006) Responsiveness of the International Knee Documentation Committee Subjective Knee Form. Am J Sports Med 34(10):1567–1573
45. Anderson AF, Irrgang JJ, Kocher MS, Mann BJ, Harrast JJ (2006) The International Knee Documentation Committee Subjective Knee Evaluation Form: normative data. Am J Sports Med 34(1): 128–135
46. Ardern CL, Webster KE, Taylor NF, Feller JA (2011) Return to sport following anterior cruciate ligament reconstruction surgery: a systematic review and meta-analysis of the state of play. Br J Sports Med 45(7):596–606
47. Kvist J, Ek A, Sporrstedt K, Good L (2005) Fear of re-injury: a hindrance for returning to sports after anterior cruciate ligament reconstruction. Knee Surg Sports Traumatol Arthrosc 13(5): 393–397
48. Ardern CL, Taylor NF, Feller JA, Whitehead TS, Webster KE (2013) Psychological responses matter in returning to preinjury level of sport after anterior cruciate ligament reconstruction surgery. Am J Sports Med 41(7):1549–1558
49. Chmielewski TL, Jones D, Day T, Tillman SM, Lentz TA, George SZ (2008) The association of pain and fear of movement/reinjury with function during anterior cruciate ligament reconstruction rehabilitation. J Orthop Sports Phys Ther 38(12):746–753
50. Langford JL, Webster KE, Feller JA (2009) A prospective longitudinal study to assess psychological changes following anterior cruciate ligament reconstruction surgery. Br J Sports Med 43(5): 377–381
51. George SZ, Lentz TA, Zeppieri G, Lee D, Chmielewski TL (2012) Analysis of shortened versions of the tampa scale for kinesiophobia and pain catastrophizing scale for patients after anterior cruciate ligament reconstruction. Clin J Pain 28(1):73–80
52. Webster KE, Feller JA, Lambros C (2008) Development and preliminary validation of a scale to measure the psychological impact of returning to sport following anterior cruciate ligament reconstruction surgery. Phys Ther Sport 9(1):9–15
53. Thomee P, Wahrborg P, Borjesson M, Thomee R, Eriksson BI, Karlsson J (2008) Self-efficacy of knee function as a pre-operative predictor of outcome 1 year after anterior cruciate ligament reconstruction. Knee Surg Sports Traumatol Arthrosc 16(2):118–127
54. Morrey MA, Stuart MJ, Smith AM, Wiese-Bjornstal DM (1999) A longitudinal examination of athletes' emotional and cognitive responses to anterior cruciate ligament injury. Clin J Sport Med 9(2):63–69
55. Tripp DA, Stanish W, Ebel-Lam A, Brewer BW, Birchard J (2007) Fear of reinjury, negative affect, and catastrophizing predicting return to sport in recreational athletes with anterior cruciate ligament injuries at 1 year postsurgery. Rehabil Psychol 52(1):74–81
56. Myer GD, Martin L Jr, Ford KR, Paterno MV, Schmitt LC, Heidt RS Jr et al (2012) No association of time from surgery with functional deficits in athletes after anterior cruciate ligament reconstruction: evidence for objective return-to-sport criteria. Am J Sports Med 40(10):2256–2263
57. van Grinsven S, van Cingel RE, Holla CJ, van Loon CJ (2010) Evidence-based rehabilitation following anterior cruciate ligament reconstruction. Knee Surg Sports Traumatol Arthrosc 18(8): 1128–1144

58. Salmon L, Russell V, Musgrove T, Pinczewski L, Refshauge K (2005) Incidence and risk factors for graft rupture and contralateral rupture after anterior cruciate ligament reconstruction. Arthroscopy 21(8):948–957
59. Gagnier JJ, Morgenstern H, Chess L (2013) Interventions designed to prevent anterior cruciate ligament injuries in adolescents and adults: a systematic review and meta-analysis. Am J Sports Med 41(8):1952–1962
60. Withrow TJ, Huston LJ, Wojtys EM, Ashton-Miller JA (2006) The relationship between quadriceps muscle force, knee flexion, and anterior cruciate ligament strain in an in vitro simulated jump landing. Am J Sports Med 34(2):269–274

The Groin Pain Syndrome

8

Gian Nicola Bisciotti

8.1 Introduction

Groin pain represents a widespread problem in sport in both amateur and professional areas. However, the term "groin pain" should describe only the symptoms or a symptom, the pain in the groin area, a medical problem with still unclear pathophysiology. One of the reasons for this could be the anatomical complexity of the pubic area and the frequent overlapping of different pathology [1]. The term groin pain or pubalgia is according to some authors ambiguous or at least simplistic and not suitable for the complexity of the medical issue in question. It is better defined as groin pain syndrome [2]. Conversely to this lack of clarity, the groin pain syndrome has spread from a typical pathology of high-profile athletes into all levels of athletes. It currently affects mostly intermediate-level athletes, as their fitness levels for sport activity are often not suitable for its prevention, while the athletic load is high enough to favor its onset [3]. The diagnosis of groin pain syndrome has been reported by Spinelli more than 70 years ago as a medical problem affecting fencers [4], and since then, controversy and different conceptual interpretations started [5, 6].

Sport activities most at risk are represented in Europe by football and then, with less impact, by hockey, rugby, and distance running [7–16]. However, none of these publications relate the incidence of the injury to the number of licensed athletes into the various activities in question, and most of these studies would be rejected if we follow the minimum criteria of a meta-analysis [17].

G.N. Bisciotti, MSc, PhD
Qatar Orthopaedic and Sport Medicine Hospital,
FIFA Center of Excellence Doha, Doha, Qatar

Kinemove Rehabilitation Centers,
Via Veterani dello Sport N° 1,
Pontremoli, La Spezia, Italy 54027, Italy
e-mail: bisciotti@libero.it; giannicola.bisciotti@aspetar.com

8.2 Etiology and Clinical Classification

Different entities of groin pain are classified according to the type of pathologic lesion and to symptoms that are reported by the patient. Very often, an inaccurate diagnosis, leading to inadequate therapeutic interventions, can further lead to a very debilitating medical problem, sometimes forcing the athlete to long suspension of sport activity.

In our view, this discrepancy of clinical judgments is mainly generated by the excessive overlapping of possible clinical entities. For example, some authors [16, 18] identify from 15 to 72 cases of groin pain including mainly muscle and tendon pathologies (insertional tendinopathy, ectopic calcification, avulsions, hernia) but also bone and joint diseases such as stress fractures, osteochondrosis or osteonecrosis, infections, cancer, bursitis, nerve entrapment, and pain of the visceral source.

Considering the importance of a correct diagnosis, the first step in this direction seems to adopt a correct and rational nosological framework. One of the most systematic, practical references derives from Brunet's [19] and from Durey's and Rodineau's studies [9]. According to the experience of these authors, the groin pain in athletes refers to three different anatomo-clinical entities often associated as follows:

1. Parieto-abdominal pathology, affecting the lower part of the anterior abdominal muscles (external and internal oblique muscles and transverse muscle), fascia transversalis, conjoint tendon, and inguinal ligament
2. Adductor muscle pathology mainly affecting the adductor longus and pectineus muscle
3. Pubic symphysis pathology

Bouvard's theory [1] is also interesting and worthy to note. These authors have proposed a revision of Brunet, Durey, and Rondineau's [9] classification and suggest a single disease presenting in four different clinical forms:

1. The pubic osteoarthropathy affecting the pubic symphysis joint and the adjacent bone branches due to microtraumatic etiology. This needs to be differentiated

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_8, © Springer International Publishing Switzerland 2016

from the rare infectious pubic osteoarthritis [10, 20, 21]. Sometimes bone modifications could be evident appearing in the form of erosion, or as real "nail shots" sometimes with bone fragments. Occasionally, erosions may occur in such marked and conspicuous manner to include in the differential diagnosis of neoplastic erosive osteopathy [22].

2. The inguinal canal pathology with diagnosis initially formulated by Nesovic [23], arbitrarily named "sports hernia" since in this case, a real hernia is not present [9, 14, 24]. Many authors report a high percentage (36–84 %) of non-palpable hernias but with similar symptoms in the groin [25–30]. All painful symptoms caused by inguinal canal posterior wall anatomical defects are included in this category, that is, localized weakness of fascia transversalis, an area where striated muscles are absent [14]. Pathology of the inguinal canal posterior wall can be confirmed by ultrasonography [31, 32]; herniography has only historical significance since it is very invasive [11, 26, 27]. Moreover, anterior wall inguinal canal lesions such as conjoined tendon or external oblique muscle tear should be considered [33] as they may occasionally lead to ilioinguinal and iliohypogastric nerve entrapment [6, 14, 17, 29, 31]. This group also includes external oblique muscle aponeurosis lesion and inguinal ligament and fascia transversalis lesions [14, 31, 34–37].
3. Rectus abdominis insertional tendinopathy [9, 12, 38–40].
4. Bone–tendon junction and muscle–tendon junction tendinopathy of the hip adductor muscles possibly complicated by obturator nerve entrapment [29, 41, 42].

Benazzo et al. [43] proposed a similar clinical classification, especially in terms of nosological rationality, and subdivided the possible clinical cases into three groups:

1. Adductor and/or abdominal muscle insertional tendinopathies, occasionally associated with pubic osteoarthropathy, likely due to microtraumatic repetitive stress. The basic anatomical lesion is represented by an adductor muscle–tendinous unit sprain affecting in most cases the adductor longus, with a potential rectus abdominis involvement at the level of its distal insertion. In this context, it may also be associated with a secondary bone alteration at the pubic symphysis. According to the authors, this type of injury would be the most prevalent in football.
2. Abdominal wall lesions, especially the inguinal canal lesion as hernia, structural weakness of the posterior wall, and the conjoint tendon abnormalities.
3. The less common causes of groin pain, not directly linked to abdominal wall pathologies. These clinical situations defined by the authors with the term "pseudopubalgia" include iliopsoas, quadratus femoris and obturator internus muscle strains or tears, nerve compression syndromes (especially affecting the obturator, ilioinguinal, femoral cutaneous, femoral, pudendal, iliohypogastric, and genitofemoral nerve), abdominal muscles perforating branches compression, and spinal nerves anterior roots pathologies. A condition included in this group, and relatively frequent in football, is the obturator nerve entrapment syndrome, with pathogenesis that, although not yet clearly defined, seems to be due to a fascia inflammatory process which could cause an obturator nerve (anterior branch) involvement of its part over the adductor brevis muscle. Furthermore, in this group, there are bone lesions, such as the osteitis pubis, femoral head stress fractures, pubic symphysis stress lesions, diastasis, osteochondritis dissecans, osteomyelitis, and tumors.

However, besides the proposed three clinical classifications, we can still find many authors that consider pubalgia as a "unique" clinical entity which is summarized in inguinal canal pathology [8, 14, 36, 44], adductor muscle insertional tendinopathy [17, 45], or pubic osteoarthropathy [46]. As it has been pointed out in some studies [36, 47], it is very important to distinguish the so-called true pubic lesions, directly affecting the pubic skeletal structure, and the false pubic lesions represented by insertional tendinopathy, hernia, sport hernia, and nerve entrapment. In addition, it should be noted that some authors [48] do not agree with the inguinal canal diagnosis and consider that it is only associated with a more general groin pain framework. Inguinal forms relate almost exclusively to the male population, affecting football players in 70 % of the cases, followed by hockey players, rugby players, and long-distance runners [2, 14, 49]. However, other authors consider that the term groin pain or pubalgia should be used only for the parietal lesions and that all other forms should have a different and very specific nomenclature.

According to these authors [2, 48, 50], all "no parietal forms" include the following:

1. Rectus abdominis tendinopathy
2. Adductor longus m., pectineus m., and gracilis m. tendon damages and adductor muscle belly lesions
3. Iliopsoas muscle lesions
4. Pubic osteoarthropathy
5. Pubic stress fracture
6. Coxofemoral pathologies
7. Maigne's intervertebral syndrome, though with rare incidence

Other authors also agree in some way to this clinical approach. According to Gilmore [14], in case of symptoms that he described with the term "groin pain disruption," it is possible to find simultaneously a conjoined tendon lesion, and its avulsion from the pubic tubercle, an external oblique muscle aponeurosis injury, or a dehiscence between the conjoined tendon and the inguinal ligament. In addition, in 40 % of the cases, there is an adductor muscle weakness.

According to Albers [51], in 90 % of the surgically treated groin pain cases, we can find a focal fascial protrusion called "bulging." In particular, there is often an abnormally high conjoined tendon insertion pointed out. For these reasons, the author underlines the fact that groin pain is caused by a myofascial pubic–abdominal abnormality (pubalgic abdominal myofascial abnormality, PAMA). According to the theory that the term "pubalgia" is only used in cases of parietal disease, it is possible to find in bibliography a widespread consensus on the dominant factors in the pubalgia framework (i.e., inguinal canal widening, inguinal canal posterior wall weakness, groin pain disruption, and PAMA).

In any case, given the "key concept" that the term groin pain, or pubalgia, represents only the description of a symptom or a cohort of symptoms and is not a diagnosis, speaking of "pseudo-groin pain" and/or "pseudo-pubalgia" represents a conceptual error. For this reason, currently, the more rational clinical classification is, in our opinion, the one proposed by Omar et al. [52]. It suggests a differential diagnosis of groin pain syndrome based on 37 major diseases, subdivided in 10 different categories (Table 8.1).

Table 8.1 The differential diagnosis of groin pain in athletes proposed by Omar et al. [52] (modified)

Category 1: Visceral causes
Inguinal hernia
Other abdominal hernias
Testicular torsion
Category 2: Hip-associated causes
Acetabular labral tear and femoroacetabular impingement
Osteoarthritis
Snapping hip syndrome and iliopsoas tendinopathy
Avascular necrosis
Iliotibial band syndrome
Category 3: Pubic symphyseal causes
Rectus abdominis tear
Adductor muscle–tendon dysfunction
Rectus abdominis–adductor longus aponeurosis tear
Osteitis pubis
Category 4: Infectious causes
Septic arthritis
Osteomyelitis
Category 5: Pelvic inflammatory disease
Prostatitis
Epididymitis and orchitis
Herpes infection
Category 6: Inflammatory causes
Endometriosis
Inflammatory bowel disease
Pelvic inflammatory disease
Category 7: Traumatic causes
Stress fracture
Tendon avulsion
Muscle contusion
Baseball pitcher–hockey goalie syndrome
Category 8: Developmental causes
Apophysitis
Growth plate stress injury or fracture
Legg–Calvé–Perthes disease
Developmental dysplasia
Slipped capital femoral epiphysis
Category 9: Neurologic causes
Nerve entrapment syndromes (e.g., ilioinguinal nerve)
Referred pain
Sacroiliitis
Sciatic entrapment (piriformis syndrome)
Hamstring strain
Knee pain
Category 10: Neoplastic causes
Testicular carcinoma
Osteoid osteoma

8.3 Injury Mechanisms and Predisposing Factors

Intrinsic and extrinsic factors may predispose the athlete to the groin pain syndrome. Among the intrinsic factors, those receiving the greater consensus in literature [1, 10, 32, 53–59] are as follows:

1. Hip and/or sacrum–iliac joint diseases
2. Lower limbs asymmetry
3. Lumbar hyperlordosis
4. Functional imbalance between abdominal and adductor muscles, with a weakness of the abdominal muscles compared to the adductors leading to their excessive stiffness or a weakness of both muscular groups, leading to a reactive contracture of adductor muscles
5. Excessive hamstring stiffness
6. Adductor weakness
7. Previous injury

It is important to remember that some authors [60] proposed as intrinsic cause a core muscular weakness or a delayed onset of transversus abdominal muscle recruitment.

Furthermore, there is an ongoing debate in literature regarding the age and/or sport experience as risk factors for groin injury [60–62].

The extrinsic factors [19, 23, 40, 63–65] are as follows:

1. Inadequacy of sport equipment: a typical example in football is the use of cleats, too long on dry surfaces or too short on soft ground [3].
2. Inadequate pitch surfaces [40, 63].
3. Errors in training planification [65].

Regarding the inadequacy of pitch surfaces, we must make some important clarifications. A parameter which we must carefully assess is represented by the interaction, in terms of mechanical constraint, between the pitch and the shoe. An interesting data in this regard comes from the

American National Football League (NFL), which shows that abductor tendinopathy would increase by 27 % on the artificial turf pitches when compared to natural turf pitches [66], although these data do not find further confirmation in the literature [67, 68]. Also some natural grass surfaces may be a risk factor for the onset of abductor tendinopathy. The association of hot climates and some types of grass having a particularly strong and deep root system creates an excessive constraint between the shoe and the ground. Conversely, other types of grass with an insufficient radical apparatus, if used in cold climates, would not be able to create a sufficient mechanical constraint between the foot and the playing surface. Both situations could represent a risk factor for onset of adductor tendinopathy especially in athletes with pelvic instability [66].

One of the sports where groin pain is most frequent is football [69]. Many technical movements in football may favor the onset of the injury: jumps, dribbling, cutting movements in general, and tackles performed sliding with abducted leg and adductor muscle contracted. These are factors that cause high stress on the pubic symphysis, triggering a synergic mechanism between adductors and abdominal muscles [43]. Moreover, shooting and running performed on irregular surfaces represent other intense and abnormal functional stress factors [70].

In this context, it is important to consider the Maigne theory [71], based on the functional imbalance of the football players' column biomechanics. Specifically, this theory argues that football players are playing in a constant hyperlordotic gait which creates a conflict at the dorsal-lumbar spine level between the vertebral joints and genito-abdominal nerves, responsible for the groin region sensitive innervations. This theory could justify the high incidence of groin pain in football reported by different authors [72, 73].

There is no strong evidence in the literature supporting a causal association for any extrinsic or intrinsic risk factors and groin pain syndrome onset. In effect, the majority of the studies are based on conjecture, expert opinion, or case series.

Athletes affected by groin pain syndrome would most likely be subjected to a combination of excessive muscular contractions by abdominal and adductor muscles. Torsion and impact causing bone stress can occur during running, violent movement performed with poor muscle control (such as sprint, shoots, tackles, change of direction), and mechanical constraints especially of torsion type at the pubic symphysis level [12, 32, 63, 65, 74]. The majority of authors agree that during normal activity, the abdominal and adductor muscles have an antagonistic but biomechanically balanced function. In the case of groin pain, there is no more muscle balance between the adductors and abdominals, with the adductor muscles being too powerful and the abdominals too weak or with adductors being extremely stiff thus producing an abnormal tension in the pelvis with a negative impact on the pubis [19, 23, 36, 44, 57, 75, 76]. Finally, quadriceps muscle hypertonia would further aggravate this functional imbalance [76].

It is important to underline the rectus abdominis and adductor longus origin from a common aponeurosis insertion at the periosteum of the anterior aspect of the pubic body and their antagonist function during rotation and extension [77].

Moreover, we must remember that also a force ratio less than 80 % between the adductor and abductor muscles has been identified as a potential groin pain risk factor [45]. Other authors found that the same deficit between the extensor and the flexor trunk muscle force ratio could induce groin pain [16]. Finally, other studies [1] include poor proprioception among the predisposing factors. However, our therapeutic experience does not allow us to share this hypothesis; in effect, both static proprioception management and dynamic proprioception management reflect an extremely multifactorial control mode which makes it difficult to provide evidence in this specific field.

It is important to remember that six of the seven adductor muscles[1] are innervated by the obturator nerve and that their origin is in close proximity to the pubis. This allows them biomechanically to act in open kinetic chain as hip adductors and have an important stabilizing role in the closed kinetic chain. Not surprisingly, athletes affected by groin pain syndrome generally have significant concentric muscle strength in the lower limb muscles while simultaneously presenting with a deficit of postural muscle strength [1, 45].

8.4 Clinical and Diagnostic Examination

Symptoms of groin pain syndrome are bilateral in 12 % of cases, affecting the adductor region in 40 % of the cases and the perineal area only in 6 % of the cases [14]. The onset of reported groin pain syndrome symptoms is insidious in 2/3 of the patients and acute in 1/3 [14]. The groin pain clinical framework is characterized by subjective and objective symptomatology.

Subjective symptoms are mainly identified in pain and functional deficit [78, 79]. The intensity of pain has highly significant variability and can range from a mere annoyance to acute pain. The intensity of which can even affect the patient's normal daily life activity, such as walking, dressing, and getting out of bed or car, and sometimes even preventing

[1] There are seven adductor muscles; the closest to the surface is the pectineus. The adductor longus, the gracilis, and the adductor brevis are located within the second layer. The adductor magnus is in the deep muscular layer. The pectineus muscle is innerved by the femoral nerve and the obturator nerve. The adductor magnus is innervated by the obturator nerve or by the ischiatic nerve and the tibial nerve. The adductor longus and the adductor brevis are innervated only by the obturator nerve. In the gluteal region, the muscles performing adductor functions are the obturator externus muscle innervated by the obturator nerve, the quadratus femoris muscle innervated by the ischiatic nerve, and the quadratus femoris muscle nerve.

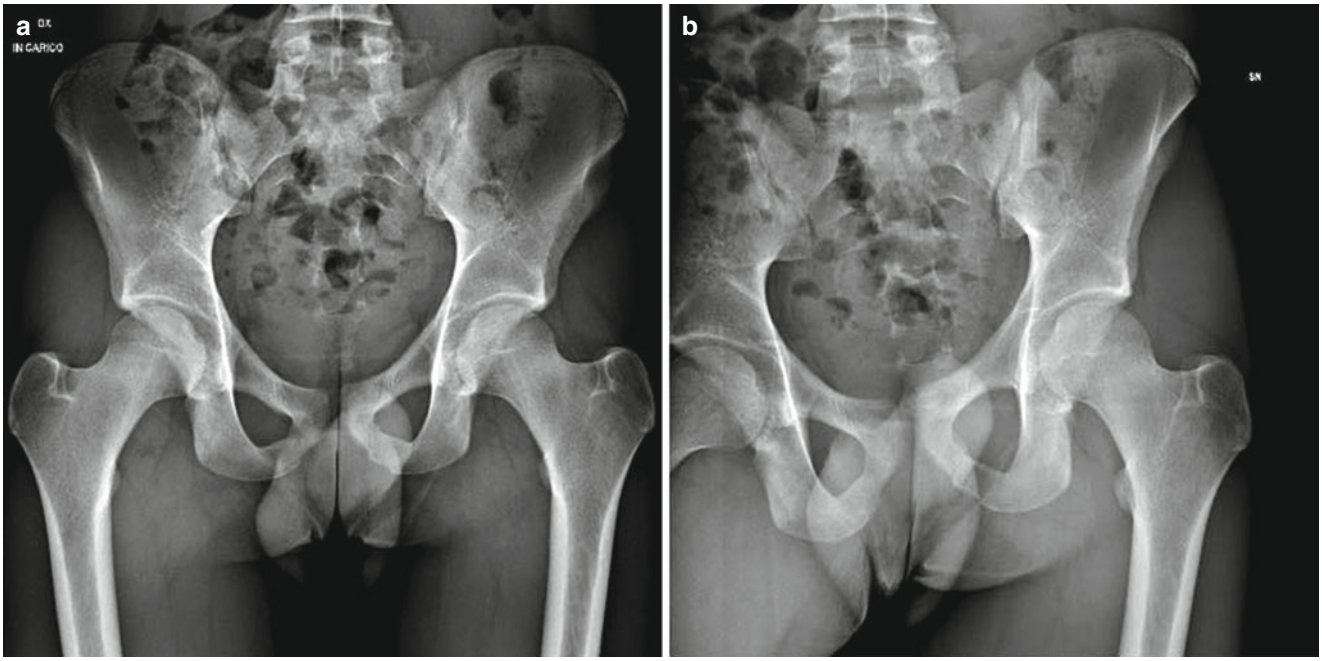


Fig. 8.1 A double-stance X-ray (**a**) compared to a dynamic flamingo view X-ray (**b**) made in alternating single-stance support (in this case in single right stance). The subject, a 25-year-old professional football player, shows a vertical offset greater than 3 mm between the pubic horizontal branch that allows us to make the diagnosis of symphysis instability

sleep. The painful event can occur during competition and/or training. It can already be present prior to exercise and disappear during warm-up, reappearing later during activity or appearing after the exercise, while cooling down, or even the morning after. In extreme cases, symptoms can effectively preclude performance. Pain may radiate outward and extend along the adductor and/or abdominal muscles in the direction of the perineum and the genitals. This generates possible diagnostic errors [79]. The functional deficit is obviously correlated with pain intensity.

From an objective point of view, the patient can complain of pain at palpation, resisted contraction, and during stretching. In addition, clinical examination is based on several muscle tests based both on active contractions and on passive and active muscle stretching [80–83]. Moreover, in this context, it is important to observe how the patient moves, walks, and undresses [84].

8.4.1 Imaging

Radiological investigations can help in groin pain syndrome diagnosis. Pelvic X-rays highlighting the pubic symphysis are always advisable to rule out possible bone erosion, pubic branch dissymmetry, osteoarthritis (also frequent in young subjects), hip joint pathology, and especially tumors or avulsion fractures [85–87]. It is important to emphasize how through a dynamic X-ray made in alternating monopodalic support, the so-called flamingo views (Fig. 8.1), when a vertical offset greater than 3 mm between the pubic horizontal branches is found, we can make the diagnosis of symphysis instability [44, 88, 89]. Musculoskeletal ultrasound (US) finds its indication in inguinal hernia suspicion. It can highlight edema areas, hematomas (in the case of muscle–tendon tears), myxoid degeneration areas, chondral metaplasia or metaplastic calcification, and fibrosis [30, 90] with the advantage of having the possibility of being carried out in dynamic conditions. This highlights musculofascial movements and, in particular, inguinal bulging (inguinal canal posterior wall weakness). However, US currently falls short in the identification of inflammatory and degenerative bone processes.

Nuclear bone scan is a highly sensitive but nonspecific tool. Every type of symphysis bone lesion of traumatic, tumoral, or infectious etiology would lead to an increased uptake activity at the symphysis level [30, 91, 92]. However, a previous uptake that normalizes after conservative treatment is an important factor which may play a role in making a decision for possible return to sports activity [91, 93, 94].

Magnetic resonance imaging (MRI) is considered the gold standard examination providing detailed information concerning both bone and insertion structures [8, 30, 86, 89]. An MRI groin pain-specific protocol should include sequences covering the entire bony pelvis as well as higher-resolution sequences dedicated to the pubic symphysis region. A relatively outperformed model like a 1.5 T MRI unit is an adequate instrument to generate high-quality images of the pelvis, while a 3 T scanner can

offer indubitable advantages in signal and resolution but is also prone to generate more imaging artifacts [95]. Images must be acquired in standard coronal, sagittal, and axial planes; however, it is important to underline that coronal oblique imaging plane performed along the anterior margin of the iliac crest is a very important sequence for optimal assessment of the rectus abdominis/adductor longus common aponeurosis at the pubic level [96]. Some authors proposed the use of intravenous contrast, but its use generally adds little in the identification of lesions, and a non-contrast protocol at 1.5 T can be considered standard [52].

One of the most important advantages in the use of MRI for the assessment of patients affected by groin pain is its high sensitivity for a wide array of both musculoskeletal and visceral lesions that may concur to the symptomatology. In effect, it is not uncommon to discover an unsuspected lesion with pelvic MRI. For these reasons, it is important to include in MRI protocol several large field of view sequences covering the entire bony and visceral pelvis even if there is a strong suspicion for a simple pubic symphysis lesion. In fact, it is not uncommon that the groin pain is caused by bursitis, benign and malignant soft tissue tumors in various locations around the pelvis, visceral pelvis sources such endometriosis and inflammatory bowel disease, osseous injuries such stress fracture, primary osseous tumor such as osteoid osteoma, or scarring and fibrosis related to prior herniorrhaphy. With a deep MRI evaluation protocol, the majority of these lesions should be observed or at least suspected [97].

8.5 Rehabilitation and Treatment Strategy

8.5.1 Type of Exercise and the Progression of Work Plan

Concerning the type of exercise, the study with the strongest evidence considers strengthening exercise as the main component of the work plan [80, 98, 99]. Target muscles involved are the adductor, abductor, hip flexor, and deep and superficial abdomen muscles. The progression begins with isometric contractions and continues with concentric and eccentric exercises, reaching the functional standing position. This is to be as similar as to those required by the athlete's specific sport activity during the last stage of the rehabilitation protocol. Isokinetic exercises should also be present throughout the protocol. Holmich et al. [80] used a predetermined graduated exercise protocol, while many researches adopt the following criteria for exercise progression:

1. Absence of pain during exercise
2. Full acquisition of functional control
3. Ability of performing functional exercise or a predetermined number of repetitions

The available evidence suggests that strengthening exercise represents an important component in an effective work plan. However, variability between the different protocols in terms of the muscle concerned does not allow for a conclusion to be reached on the specific target muscle group [80, 98, 99]. Conversely, research shows a uniformity of exercise progression from the isometric modality to be completed by sport-specific functional standing positions.

8.5.2 The Intensity, the Frequency, and the Duration of Exercise

To the best of our knowledge, only one reliable study may be found in the available literature providing enough detail concerning intervention frequency and duration of exercise [80]. This study suggests a work plan of 90 min of strengthening exercises for the hip and abdominal muscles to be performed three times per week for an overall duration of 8–12 weeks. According to this research, the outcome is good, allowing the athlete to return to sport activities without groin pain.

The duration of conservative treatment is between a minimum of 2–3 weeks [14] and a maximum of 6 months generally [100]. The majority of authors agree on a duration of around 6 months [23, 92, 94, 101–104]. In summary, it is clear that the variation in duration of rehabilitation work plans used reflects the variation in the severity and multifactorial characteristics of groin pain.

8.5.3 Therapeutic Interventions

In essence, the majority of studies report the use of one or more co-interventions, from manipulation techniques and massages [92, 102–104] to anti-inflammatory [18, 98, 100, 101, 105] and corticosteroid medication [58, 106, 107]. Some studies included jogging, running, and cycling as co-interventions [56, 98, 99, 104]. Furthermore, some studies underline the importance of physiotherapist-supervised exercise programs [56, 99, 102].

8.5.4 Surgical Treatment

As previously discussed, groin pain syndrome may be caused by several pathologies responding to conservative therapy. However, if conservative therapy fails, then a surgical option must be considered. In this final section, we will briefly describe the most common diseases requiring such treatment.

8.5.5 Inguinal Hernia

Athletes are susceptible to inguinal (direct and indirect) hernias like the general population and sometimes even more, especially in sports like weightlifting. However, in athletes, direct hernias are more frequent [108]. Real-time dynamic US during a provocative maneuver, such as Valsalva, may help visualize a subtle hernia possibly causing symptoms only during sport activity and otherwise difficult to detect. The risk of complications such as bowel incarceration and strangulation is not an issue in this case; it is impossible to participate in sports due to pain. This is why in most cases posterior wall weakness of the inguinal canal are surgically repaired [109].

Even though surgical treatment is successful in the large majority of cases, one should bear in mind the possibility of surgical complications and, in some cases, the inability to achieve prior levels of athletic performance [52]. It has been proposed that this variability in surgical repair outcome is occasionally due to the increasing stabilization of the pubic region because of progressive fibrosis [52]. However, patients with inguinal hernia have little chance of success with conservative treatment [52, 110]. After herniorrhaphy, an average of 87 % of the athletes have a positive outcome and are able to return to full and unrestricted athletic activity in 4 weeks or less [29, 110, 111].

8.5.6 Sports Hernia

Sports hernia, also known as sportsman's hernia, athletic hernia, and incipient hernia, represents a difficult clinical problem [112].

The diagnosis of sports hernia is formulated when no inguinal hernia is found but there is persistent inguinal pain during sports activity. The symptoms resemble an hernia and are present only during sport. We must also point out that some authors underline that sports hernia is often associated with femoroacetabular dysplasia and/or femoroacetabular impingement [113].

There also is no hernia present on physical examination and ultrasound, hence the term sports hernia (Fig. 8.2). Sports hernias rarely improve without surgery [11, 114–118], and surgical repair should be considered when conservative treatment over a period of 6–8 weeks has failed. Careful examination has to additionally exclude other potential pain sources [112, 119].

Some authors propose laparoscopic repair with prosthetic mesh [120, 121]. This "tension-free" technique involves placing prosthetic material suitably shaped, nonabsorbable, and biocompatible. This acts as mechanical reinforcement of the abdominal wall [120, 121]. However, the mesh has no elasticity and creates more scar tissue, and

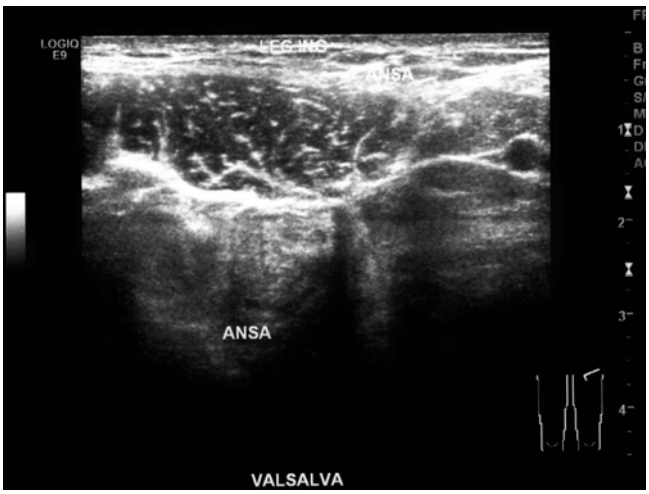


Fig. 8.2 Left inguinal ultrasound in a 27-year-old professional footballer that shows a modest pre-hernial area with about 8 mm of intestinal loop in correspondence to the weak zone. This situation is pathognomonic for sports hernia

mesh-related complications can occur years after surgery. Another laparoscopic method used for the treatment of sport hernias is inguinal release procedure [122]. After laparoscopic repair, the recovery before full return to competition is generally between 2 and 8 weeks [110, 115, 119, 123–128].

Some authors prefer open surgical inguinal repair: Shouldice repair, Maloney darn, or Bassini with or without adductor longus tenotomy or only the "minimal repair" of the weak area of transversalis fascia [14, 129, 130]. In a meta-analysis study [119], the authors found that the period of time to return to sport is on average 17.7 weeks for patients who underwent open approaches and 6.1 weeks for laparoscopic repairs. Several authors underline mesh-related complications such as infections with chronic groin infection and fistula formation. These complications sometimes require mesh removal [131] or cause mesh migration and penetration into the bladder or bowel [132, 133]. In addition, a foreign body reaction with decrease of arterial perfusion and testicular temperature [134] accompanied by secondary azoospermia may occur [134, 135].

It is interesting to note that Muschaweck et al. [112, 130] after previously utilizing the Shouldice repair under local anesthesia for years, in 2000 developed a new surgical technique called the "minimal repair technique." The aim of this surgical intervention was to stabilize the posterior wall by a tension-free suture without the use of a prosthetic mesh and by repairing only the weak spot of the transversalis fascia. The authors chose to avoid the use of a prosthetic mesh to allow the athlete's full elasticity and muscle sliding between the abdominal muscles after surgery [112]. According to some authors, opinions regarding this technique apart from avoiding prosthetic mesh insertion have several advantages.

These include not requiring general anesthesia, less traumatization, and a lower risk of severe complications. The authors underline a quicker resumption of sports activity following this surgical technique compared to the laparoscopic or open surgery with mesh insertion. They report that on average their patients resumed moderate training after 7 days and felt complete relief of pain after 14 days. Return to full activity was achieved after 18.5 days [112].

8.5.7 Adductor Tendinopathy

With the increase of knowledge of the pubic symphysis' complex anatomy, the incidence of isolated adductor tension lesion has seemingly decreased [96]. In any case, adductor tendinopathy is one of the most common causes of groin pain syndrome in athletes in athletes and is most often associated with either rectus abdominis/adductor longus aponeurosis lesions or midline pubic plate lesions (i.e., lesions originate at the midline of the pubis and propagate either unilaterally or bilaterally, also called "midline core muscle injuries"). One of the main causes of groin pain syndrome is the imbalance between the abdominal and hip adductor muscles, with the abdominals too weak or the adductors too strong [5]. Adductor tendinopathy is frequently related to an adductor longus overuse or to its aponeurotic injury [136]. A vast majority of patients respond positively to conservative treatment, both in the case of overuse tendinopathy or in muscle–tendon injury. There are not many scientific papers on failed conservative treatment on chronic adductor-related groin pain [137]. Adductor tenotomy is proposed for cases nonresponsive to conservative treatment [5, 136–139]. The criteria for surgery is a history of long-standing (ranging from 3 to 48 months according to various authors) and of distinct pain at the origin of the adductor longus muscle, refractory to conservative treatment. The operation is performed by releasing the anterior ligamentous fibers of the adductor longus while keeping the fleshy part of the muscle intact on the deep aspect, thus minimizing the loss of adductor strength after surgery and constituting a template for future regrowth of the tendon. In the patients undergoing tenotomy, there is an average of 10 % postoperative strength reduction which does not result in any obvious functional or speed limitation because other muscles in the adductor group, namely, adductor brevis, adductor magnus, and pectineus, take over adductor longus function [140]. In the reported studies [129, 136, 137], the subjects returned to competitive sport after 19.8 weeks (range 27–14 weeks). The cited studies report that following surgery, 70.6 % of the subjects (range 90–62 %) performed sport activities at the same level, 24 % (range 32–9 %) performed sports activities at a reduced level, and 5 % had to stop sport activities altogether. It is interesting to note that some authors associate the adductor tendon release to a pelvic floor repair [45, 141].

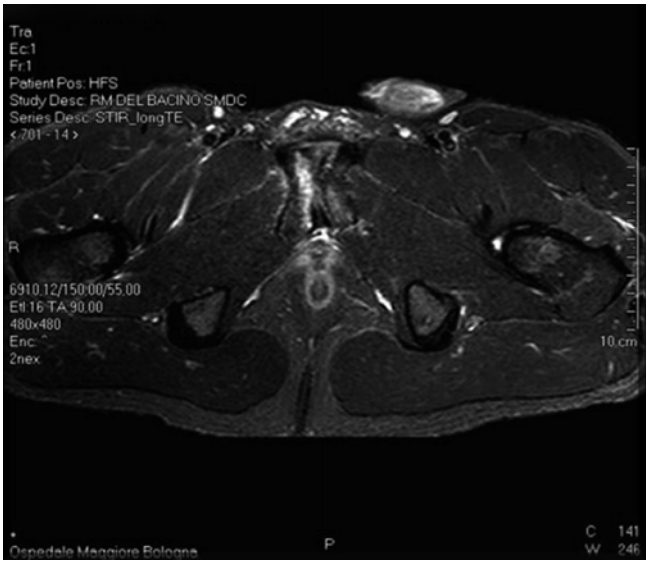


Fig. 8.3 MRI axial STIR showing bone marrow edema extending to the whole surface of the right pubic branch in a 27-year-old professional football player. This bone marrow edema extending into the anteroposterior totality of pubic rami is pathognomonic for osteitis pubis and should be distinguished from sub-entheseal marrow edema at the pubic tubercle level sometimes present in a rectus abdominis and/or adductor longus tendinopathy without osteitis pubis at the symphysis

Surgically treated adductor acute tears are rarely described in scientific literature. We could find only one study [138] reporting three cases of acute proximal adductor longus insertional tear repaired with anchor sutures and followed by postoperative rehabilitation. The patients followed in this study resumed their full sport ability after 5, 6, and 7 months, respectively.

8.5.8 Osteitis Pubis

Osteitis pubis is a common medical problem in soccer players, long-distance runners, and hockey players. In terms of etiology, the main risk factor is believed to be pubic symphysis instability [52]. This causes a chronic, repetitive shear and an imbalanced tensile stress of the muscles inserted on the pubic symphysis. This biomechanical alteration can cause an inflammatory response with osteitis and periostitis.

Normally, from a radiological point of view, into the MRI pubic symphysis evaluation, any subchondral bone marrow edema, bony sclerosis, or cystic or osteophytic formation is termed osteitis pubis. This type of assessment is not entirely correct. In effect, a true active osteitis pubis should include at least an element of subchondral bone marrow edema (often asymmetric) spanning the pubis joint anterior to posterior on axial fat-suppressed sequence (Fig. 8.3). This bone marrow edema extending into the anteroposterior totality of pubic rami should be distinguished from

sub-entheseal marrow edema at pubic tubercle level sometimes present in a rectus abdominis and/or adductor longus tendinopathy without osteitis pubis at the symphysis [96, 142]. It is in any case important to note that osteitis pubis is strongly associated with rectus abdominis and/or adductor longus tendinopathy [96]. Osteitis pubis is normally a "self-limiting" disease and requires a lengthy treatment of 12-month duration on average [105]. The management is initially conservative with physical rehabilitation, NSAID, and/or steroid injections. The historical surgical treatment options were symphysis curettage and arthrodesis and are now abandoned by the majority of surgeons. This is due to the lack of results and frequent side effects. In most cases, adductor tenotomy/surgical abdominal strengthening is reserved for the subjects with symptoms nonresponsive to conservative treatment [52, 107].

In any case, it is important to note that for some authors [143], "osteitis pubis" is a vaguely defined diagnosis. Osteitis pubis is a term originally used to describe an infection at the pubic bone at the symphysis joint level. In effect, the osteitis pubis characteristic radiologic findings, that is, widening of the symphysis, bone resorption, and sclerosis along the pubis rami, can often be found also in athletes without groin pain. This could be explained by the fact that groin-straining sport activity, for example, football or ice hockey, increases the shearing forces at the symphysis joint level. The high stress level in the symphysis might thus lead to these radiological signs, therefore indicating an increased mechanical load at the joint level rather than pathology. Hölmich [143] compares this situation with a knee joint effusion: this is in itself not an injury but a result of an overused or injured knee and therefore is not a diagnosis in itself. Especially concerning the bone marrow, edema is possible that this one in athletes represents a normal sign of bone remodeling, which may become symptomatic once loading exceeds a certain threshold. According to the authors, for these reasons, the term "osteitis pubis" should not be used as a specific diagnosis in the case of groin pain syndrome, unless an infection is present in the pubic bone. In effect, the term should be used to describe "osteitis pubis-like" radiological changes at the symphysis joint level.

8.5.9 Hockey Goalie–Baseball Pitcher Syndrome

This unusual syndrome is caused by an epimysial or myofascial herniation of the adductor longus muscle belly. It occurs several centimeters away from the site of its pubic attachment [141]. The etiology of myofascial herniations in hockey goalie–baseball pitcher syndrome has not been established. However, several authors suggest a relationship with chronic repetitive stress at the level of neurovascular penetration [144]. The treatment for chronic pain is surgical epimysiotomy and debridement [145].

8.5.10 Acetabular Labral Tear

Generally, hip pathology may cause groin pain due to synovitis, osteoarthritis, intra-articular loose bodies, and tears of the ligament teres. The most common problems are acetabular labral tears [146]. The anterior–superior part of the labrum is poorly vascularized, and for this reason, it is susceptible to injuries, particularly during hyperextension and external rotation [146, 147]. Dance, golf, hockey, and soccer are sports associated with a higher incidence of hip injuries [148].

Labral tears are initially managed conservatively with rest and NSAID therapy. Subjects with persistent symptoms often require labrum surgical debridement. During the operation, the surgeon might decide to also correct other morphologic abnormalities of the acetabulum or the proximal femur predisposing the patient to femoroacetabular impingement. This will prevent progressive cartilage loss and osteoarthritis [149, 150].

Hip arthroscopy is both a diagnostic (gold standard) and therapeutic tool, although it is technically more difficult than arthroscopy of other joints such as the knee or shoulder. During this procedure, to access the hip joint, it is necessary to distract the hip for approximately 10–15 mm. This traction may cause several complications such as neuropraxias [149]. In a number of other case series, arthroscopy has shown to provide benefit in recent traumatic labral injury [151–153]. It is also important to note that the often disappointing chronic hip pain is probably due to degenerative change and chondral lesions of the acetabulum [154, 155].

8.5.11 Internal Snapping Hip

The internal snapping hip or coxa saltans may be an occasional cause of pain in the anterior part of the hip and in the inguinal region. This pathology is characterized by a typical snapping sensation frequently accompanied by a snapping sound that the patient adverts when the tendons near the hip joint pass over an osseous protuberance. The internal snapping hip may be of extra-articular or intra-articular source. The internal snapping hip is defined as extra-articular when it is caused by the snap of the iliopsoas tendon over the iliopectineal eminence, at the level of the anterior region of the hip. The slippage and the resulting "snap" of the iliopsoas tendon occur usually when the subject passes from a position of flexed, abducted, and externally rotated hip to an extended adducted and internally rotated position. When this situation is chronically repeated, it can give rise to iliopsoas

tendonitis and bursitis [156]. Conversely, the intra-articular internal snapping hip is caused by acetabular labrum lesions or articular cartilage lesions that may be interposed between the surface of the femoral head and the surface of the acetabulum during hip motion. A further cause of intra-articular internal snapping hip can be represented by the presence of loose bodies within the joint such as cartilaginous fragments and/or calcifications [156]. The conservative treatment consists of pain control with NSAID therapy and/or corticosteroid injections in cases of bursitis; iliopsoas muscle stretching is also recommended [156, 157]. Surgical lengthening of the iliopsoas tendon (in extra-articular internal snapping hip) or cartilage repair and/or the removal of loose bodies (in intra-articular internal snapping) occasionally is necessary in patients that do not respond to conservative treatment [147].

8.5.12 Osteoid Osteoma

Osteoid osteoma is a benign bone tumor usually observed in subjects between the ages of 5 and 30 years. Usually it is most common in the long bones, especially in the femur and tibia. It can also involve the pubic bones where it may cause groin pain [52]. Total removal of the osteoid osteoma generally results in a complete resolution of symptoms, while its partial removal may lead to recurrent symptoms [158, 159].

8.5.13 Nerve Entrapment

The groin and upper thighs and sensory and motor innervations are provided by several nerves including the obturator, femoral, iliohypogastric, genitofemoral, ilioinguinal, and lateral femoral cutaneous nerves. An entrapment of any of these structures may cause groin pain [48, 58, 155]. For example, obturator nerve entrapment may be caused from a fascial thickening of the adductor compartment or a "mass effect" caused by an obturator hernia, a pelvic fracture, or an acetabular paralabral cyst [41, 52, 160]. Femoral nerve entrapment may be caused by some surgical procedures such as hip arthroplasty, herniorrhaphy, or abdominal hysterectomy [142]. Ilioinguinal and genitofemoral nerve entrapment can be observed after abdominal surgery in blunt trauma or in muscle hypertrophy [147]. If the nerve entrapment is suspected, elimination of symptoms by local anesthetic infiltration and nerve conduction studies can be considered.

The treatment of nerve entrapment syndromes often requires a surgical solution normally consisting in debridement of the perineural scar tissue or division of constricting fascia [147].

8.6 Return to Play

At the beginning of this chapter, we pointed out that the "key concept" in the diagnosis, and therefore the treatment, of groin pain is that the term "groin pain" does not represent a diagnosis but only a symptom or better a cohort of symptoms. For this reason, it is clear that it is not possible to generalize regarding the time to return to sports after conservative or surgical treatment. Besides the fact that every sporting activity must be assessed according to the specific imposed functional demands, the recovery times and the therapeutic program are obviously dependent from the groin pain etiopathogenesis. Furthermore, it is clear that, independently from the groin pain etiopathogenesis, it is extremely important to adopt a strategy which allows to reduce to a minimum the risk of recurrence. In general, we can say that a correct balance of muscle forces acting on the pelvis in addition to an adequate strength of the core muscles may represent the principal strategy to adopt. However, to date, literatures are still lacking good evidence studies that may indicate both the effectiveness of a preventive strategy or the means most indicated to its development.

Conclusions

The groin pain syndrome is an interesting and controversial subject of discussion, especially regarding therapeutic management, either conservative or surgical.

It is very important to underline the enormous importance in this field for proper and early diagnosis. Only after having diagnosed precisely, the etiology is that it is possible to refer the patient to the most appropriate type of treatment. For this reason, clinical examination should be supported by appropriate imaging studies which help the treating specialist in reaching a diagnosis. Conservative treatment, where it is recommended, should follow clearly defined intervention criteria in relation with the patient's functional progress and in full respect of the pain reported by the subject.

References

1. Bouvard M, Dorochenko P, Lanusse P, Duraffour H (2004) La pubalgie du sportif- stratégie thérapeutique. J Traumatol Sport 21:146–163
2. Vidalin H, Neouze G, Petit J, Brunet-Guedi E (2004) Prise en charge chirurgicale des pubalgies du sportif. J Traumatol Sport 21:166–173
3. Puig PL, Trouve P, Savalli L (2004) Pubalgia: from diagnosis to return to the sport field. Ann Readapt Med Phys 47(6):356–364
4. Spinelli A (1932) Una nuova malattia sportiva: la pubalgia degli schermitori. Orthop Trauma App Mot 4:111
5. Cugat R (1997) Instructional course lecture no. 105: groin pain in soccer players. ISAKOS Congress, 11–16 May, Buenos Aires
6. Irschad K, Feldman LS, Lavoi C, Lacroix VJ, Mudler DS, Brown RA (2001) Operative management of "hockey groin syndrome": 12

years of experience in National Hockey League Players. Surgey 130:759–766
7. Arezky N, Zerguini Y, Mekhaldi A, Zerdani S, Massen R, Bouras R (1991) La maladie pubienne chez le sportif. Priorité au traitement médical. J Traumatol Sport 8:91–97
8. Berger A (2000) Approches diagnostiques et thérapeutique de la pubalgie du sportif. Thèse Med 10157. Genève
9. Durey A, Rodineau J (1976) Les lésions pubiennes des sportif. Ann Med Phys 9:282–291
10. Durey A (1987) Modifications radiologiques microtraumatiques du pubis. Micro-traumatologie du sport. Masson 15:185–192
11. Ekstrand J, Ringborg S (2001) Surgery versus conservative treatment in soccer players with chronic groin pain: a prospective randomised study in soccer players. Eur J Sports Traumatol Relat Res 23(4):141–145
12. Gibbon WW (1999) Groin pain in professional soccer players: a comparison of England and the rest of Western Europe. Br J Sports Med 33:435
13. Gibbon WW (1999) Groin pain in athletes. Lancet 353:1444–1445
14. Gilmore J (1998) Groin pain in the soccer athlete: fact, fiction and treatment. Clin Sports Med 17:787–793
15. Le Gall F (1993) La pubalgie du sportif. A propos de 214 cas. Thèse en médicine Université de Rennes
16. Gal C (2000) La pubalgia. Prevenzione e trattamento. Società Stampa Sportiva (Ed), Rome
17. Orchard J, Read JW, Verral GM, Slavotinek JP (2000) Pathophysiology of chronic groin pain in the athlete. Intern Sports Med J 1(1):134–147
18. Jarvinen M, Orava S, Kuyala M (1997) Groin pain (Adductor syndrome). Oper Techn Sport Med 5(3):133–137
19. Brunet B (1983) La pubalgie, un syndrome "fourre tout". Thèse Med. Université de Lyon
20. Baril L, Caumes E, Bricaire F (1998) Pubic pain after marathon. Lancet 351(9103):642
21. Ross JJ, Hu LT (2003) Septic arthritis of the pubic symphysis: a review of 100 cases. Medicine (Baltimore) 82:340–345
22. Ferrario A, Monti GB, Jelmoni GP (2000) Lesioni da sport del bacino e dell'anca. Pelvi, articolazione sacro-iliaca, anca. Edi Ermes, Milan
23. Brunet B, Brunet-Guedj E, Genety J, Comptet JJ (1984) A propos du traitement des pubalgies. J Traumatol Sport 1:51–55
24. Renström P, Peterson L (1980) Groin injuries in athletes. Br J Sports Med 14:30–36
25. Ekberg O (1981) Inguinal herniography in adults. Radiology 138:31–36
26. Smedberg SG, Broome AE, Gullmo A, Roos H (1985) Herniography in athletes with groin pain. Am J Surg 149:378–382
27. Fon LJ, Spencer AJ (2000) Sportman's hernia. Br J Surg 87:545–552
28. Srinivasan A, Schuricht A (2002) Long-term follow-up of laparoscopic preperitoneal hernia repair in professional athletes. J Laparoendosc Adv Surg Tech A 12(2):101–106
29. Zoga AC, Kavanagh EC, Omar IM, Morrison WB, Koulouris G, Lopez H, Chaabra A, Domesek J, Meyers WC (2008) Athletic pubalgia and the "sports hernia": MR imaging findings. Radiology 247(3):797–807
30. Davies AG, Clarke AW, Gilmore J, Wotherspoon M, Connell DA (2009) Review: imaging of groin pain in the athlete. Skeletal Radiol 39(7):629–644
31. Orchard JW, Read JW, Neophyton J, Garlick D (1999) Groin pain associated with ultrasound finding of inguinal canal posterior wall deficiency in Australian Rules footballers. Br J Sports Med 32(2):134–139
32. Bradley M, Morgan D, Pentlow B, Roe A (2003) The groin hernia – an ultrasound diagnosis? Ann R Coll Surg Engl 85:178–180
33. Irshad K, Feldman LS, Lavoie C, Lacroix VJ, Mulder DS, Brown RA (2001) Operative management of "hockey groin syndrome": 12 years of experience in National Hockey League players. Surgery 130(4):759–764
34. Ziprin P, Williams P, Foster ME (1999) External oblique aponeurosis nerve entrapment as a cause of groin pain in athlete. Br J Surg 86:566–568
35. Combelles F (1993) Le repos est la phase essentielle de traitement de la pubalgie. Le Quotidien du Médicin 18:5246
36. Christel P, Djian P, Roger B, Witvoet J, Demarais Y (1996) Apport de l'IRM dans la stratégie du traitement chirurgical des pubalgies. J Traumatol Sport 13:95–101
37. Lynch SA, Renström PA (1999) Groin injuries in sport-treatment strategies. Sports Med 28:137–144
38. Martens MA, Hansen L, Mulier JC (1987) Adductor tendinitis and musculus rectus abdominis tendinopathy. Am J Sports Med 15:353–356
39. Ghebontni L, Roger B, Christel P, Rodineau J, Grenier P (1996) La pubalgie du sportif: intérêt de l'IRM dans le démembrement des lésions. J Traumatol Sport 13:86–93
40. Volpi P (1992) La pubalgie: notre experience. J Traumatol Sport 9:53–55
41. Bradshaw C, McCrory P, Bell S, Brukner P (1997) Obturator nerve entrapment: a cause of groin pain in athletes. Am J Sports Med 25:402–408
42. Bruckner P, Bradshaw C, Mac Crory P (1999) Obturator nerve entrapment: a cause of groin pain in athletes. Physician Sports Med 27:62–64
43. Benazzo F, Mosconi M, Zanon G, Bertani B (1999) Groin pain. J Sport Traumatol Rel Res 21(1):30–40
44. Christel P, Djian P, Wittvoet J (1993) La pubalgie, un syndrome du sportif correspondant à une pathologie loco-régionale. Rev Prat 43(6):729–732
45. Nicholas SJ, Tyler TF (2002) Adductor muscle strains in sport. Sports Med 5:339–344
46. Chanussot JC, Gholzane L (2003) Pathologie de la paroi abdominale et du carrefour pubien du sportif. Kinésithérapie Scientifique 439:59–60
47. Djian P (1997) La pubalgie- traitement médical et chirurgical. Médicins du Sport 9:11–23
48. Fredberg U, Kissmeyer-Nielsen P (1996) The sports-man's hernia-fact or fiction? Scand J Med Sci Sports 6:201–204
49. Smedberg S, Roos H (2002) Hockey groin syndrome. Surgery 132(5):906–907
50. Delavierre D, Rigaud J, Sibert L, Labat JJ (2010) Symptomatic approach to referred chronic pelvic and perineal pain and posterior ramus syndrome. Prog Urol 20(12):990–994
51. Albers SL (2001) Findings in athletes with pubalgia. Skeletal Radiol 30:270–277
52. Omar IM, Zoga AC, Kavanagh EC, Koulouris G, Bergin D, Gopez AG, Morrison WB, Meyers WC (2008) Athletic pubalgia and "sports hernia": optimal MR imaging technique and findings. RadioGraphics 28:1415–1438
53. Durey A (1984) Aspects cliniques de la pubalgie du sportif. J Traumatol Sport 1:46–50
54. Joliat G (1986) Les déséquilibres fonctionelles pelvi-rachidiens et les souffrances du carrefour pubien du footballeur. Med et Hyg 44:1973–1977
55. Rochcongar P, Durey A (1987) Biomécanique de la symphyse pubienne et des articulations sacro-iliaques. Micro-traumatologie du Sport Mass Ed 4:62–67
56. Morelli V, Smith V (2001) Groin injuries in athletes. Am Fam Physician 64:1405–1414
57. Robertson BA, Barker PJ, Fahrer M, Schache AG (2009) The anatomy of the pubic region revisited: implications for the pathogenesis

and clinical management of chronic groin pain in athletes. Sports Med 39(3):225–234
58. Mardones RR, Barrientos CV, Nemtala UF, Tomic A, Salineros UM (2010) Femoroacetabular impingement as a cause of inguinal pain. Rev Med Chil 138(1):102–108
59. Engebretsen AH, Myklebust G, Holme I, Engebretsen L, Bahr R (2010) Intrinsic risk factors for groin injuries among male soccer players: a prospective cohort study. Am J Sports Med 38(10): 2051–2057
60. Maffey L, Emery C (2007) What are the risk factors for groin strain injury in sport? A systematic review of the literature. Sports Med 37(10):881–894
61. Emery CA, Meeuwisse WH, Powell JW (1999) Groin and abdominal strain injuries in the National Hockey League. Clin J Sport Med 9(3):151–156
62. Aleman KB, Meyers MC (2010) Mountain biking injuries in children and adolescents. Sports Med 40(1):77–90
63. Braun P, Jensen S (2007) Hip pain – a focus on the sporting population. Aust Fam Physician 36(6):406-8–410-3
64. Hölmich P, Larsen K, Krogsgaard K, Gluud C (2010) Exercise program for prevention of groin pain in football players: a cluster-randomized trial. Scand J Med Sci Sports 20(6):814–821
65. Paajanen H, Ristolainen L, Turunen H, Kujala UM (2011) Prevalence and etiological factors of sport-related groin injuries in top-level soccer compared to non-contact sports. Arch Orthop Trauma Surg 131(2):261–266
66. Kinchington M (2013) Groin pain a view from below. The impact of lower extremity, function and podiatric intervention. ASPETAR Sport Med J 2(3):360–366
67. Fuller CW, Dick RW, Corlette J, Schmalz R (2007) Comparison of the incidence, nature and cause of injuries sustained on grass and new generation artificial turf by male and female football players. Part 1: match injuries. Br J Sports Med 41(Suppl I):i20–i26
68. Fuller CW, Dick RW, Corlette J, Schmalz R (2007) Comparison of the incidence, nature and cause of injuries sustained on grass and new generation artificial turf by male and female football players. Part 2: training injuries. Br J Sports Med 41(Suppl I): i27–i32
69. Hölmich P, Thorborg K, Dehlendorff C, Krogsgaard K, Gluud C. Incidence and clinical presentation of groin injuries in sub-elite male soccer. Br J Sports Med. 2014;48(16):1245–50
70. Scott AL, Renström FH (1999) Groin injuries in sport. Sports Med 28(2):137–144
71. Maigne R (1981) Le syndrome de la charnière dorso-lombaire. Lombalgie basse, douleurs pseudo-viscérales, pseudo-douleur de hanche, pseudotendinite des adducteurs. Sem Hop Paris 57(11–12): 545–554
72. Smodlaka VN (1980) Groin pain in soccer players. Phys Sports Med 8:57–61
73. Koulouris G (2008) Imaging review of groin pain in elite athletes: an anatomic approach to imaging findings. AJR Am J Roentgenol 191(4):962–972
74. Wodecki P, Djian P, Christel P, Witvoet J (1998) La pubalgie. Rev Rhum 65:109–117
75. Anderson K (1989) Hip and groin injuries in athletes. Thése Méd. Université Lyon 1
76. Kremer Demuth G (1998) La pubalgie du footballeur. Thèse Méd. Université de Strasbourg 1
77. Robinson P, Salehi F, Grainger A (2007) Cadaveric and MRI study of the musculotendinous contributions to the capsule of the symphysis pubis. AJR Am J Roentgenol 188:W440–W445
78. Hureibi KA, McLatchie GR (2010) Groin pain in athletes. Scott Med J 55(2):8–11
79. Garvey JF, Read JW, Turner A (2010) Sportsman hernia: what can we do? Hernia 14(1):17–25
80. Holmich P, Uhrskou P, Kanstrup IL (1999) Effectiveness of active physical training as treatment for long-standing adductor-related groin pain in athletes: randomised trial. Lancet 353:439–443
81. Unverzagt CA, Schuemann T, Mathisen J (2008) Differential diagnosis of a sports hernia in a high-school athlete. J Orthop Sports Phys Ther 38(2):63–70
82. Brown RA, Mascia A, Kinnear DG, Lacroix V, Feldman L, Mulder DS (2008) An 18-year review of sports groin injuries in the elite hockey player: clinical presentation, new diagnostic imaging, treatment, and results. Clin J Sport Med 18(3):221–226
83. Campanelli G (2010) Pubic inguinal pain syndrome: the so-called sports hernia. Hernia 14(1):1–4
84. Kehlet H (2010) Groin pain. Ugeskr Laeger 172(49):3393
85. Ilaslan H, Arslan A, Koç ON, Dalkiliç T, Naderi S (2010) Sacroiliac joint dysfunction. Turk Neurosurg 20(3):398–401
86. Zoga AC, Mullens FE, Meyers WC (2010) The spectrum of MR imaging in athletic pubalgia. Radiol Clin North Am 48(6): 1179–1197
87. Thorborg K, Serner A, Petersen J, Madsen TM, Magnusson P, Hölmich P (2011) Hip adduction and abduction strength profiles in elite soccer players: implications for clinical evaluation of hip adductor muscle recovery after injury. Am J Sports Med 39(1): 121–126
88. Death AB, Kirby L, Mc Millan L (1982) Pelvic ring mobility: assessment by stress radiograph. Arch Phys Med Rehabil 63:204–206
89. Ghebontni L, Roger B, El-khoury J, Brasseur JL, Grenier PA (2000) MR arthrography of the hip: normal intra-articular structures and common disorders. Eur Radiol 10(1):83–88
90. Lorenzini C, Sofia L, Pergolizzi FP, Trovato M (2008) The value of diagnostic ultrasound for detecting occult inguinal hernia in patients with groin pain. Chir Ital 60(6):813–817
91. Jansen JA, Mens JM, Backx FJ, Stam HJ (2008) Diagnostics in athletes with long-standing groin pain. Scand J Med Sci Sports 18(6):679–690
92. Jansen JA, Mens MA, Backx N et al (2008) Treatment of long-standing groin pain in athletes; a systematic review. Scand J Med Sci Sports 18:263–274
93. Lejeune JJ, Rochcongar P, Vazelle F, Bernard AM, Herry JY, Ramée A (1984) Pubic pain syndrome in sportsmen: Comparison of radiographic and scintigraphic findings. Eur J Nucl Med 9:250–253
94. Zeitoun Z, Frot B, Sterin P, Tubiana JM (1995) Pubalgie du sportif. Ann Radiol 38(5):244–254
95. Palisch A, Zoga AC, Meyers WC (2013) Imaging of athletic pubalgia and core muscle injuries: clinical and therapeutic correlation. Clin Sports Med 32(3):427–447
96. Zoga C (2014) Imaging athletic pubalgia and core muscle injury. Relevance for sports medicine. ASPETAR Sports Med J 1:58–65
97. Slavotinek JP, Verral GM, Fon GT, Sage MR (2005) Groin pain in footballers: the association between preseason clinical and pubic bone magnetic resonance imaging findings and athlete outcome. Am J Sports Med 33:894–899
98. Rodriguez C, Miguel A, Lima H (2001) Osteitis pubis syndrome in the professional soccer athlete: a case report. J Athl Train 36:437–440
99. Wollin M, Lovell G (2006) Osteitis pubis in four young football players: a case series demonstrating successful rehabilitation. Phys Ther Sport 7:53–60
100. Holt MA, Keene JS, Graf BK, Helwig DC (1995) Treatment of osteitis pubis in athletes; results of corticoid injections. Am J Sports Med 23(5):601–606
101. Fournier JY, Richon CA (1992) Revue critique de 25 patients traités pour pubalgie par myorraphie inguinale (opération de Nesovic). Helv Chir Acta 59:775–778

102. Machotka Z, Kumar S, Perraton LG (2009) A systematic review of the literature on the effectiveness of exercise therapy for groin pain in athletes. Sports Med Arthrosc Rehabil Ther Technol 1(1):5
103. Jansen JA, van de Port IG, Van de Sande HB, Tol JL, Backx FJ (2010) Manual or exercise therapy for long-standing adductor-related groin pain: a randomised controlled clinical trial. Man Ther 15:72–76
104. Verrall GM, Slavotinek JP, Fon GT (2007) Outcome of conservative management of athletic chronic groin injury diagnosed as pubic stress injury. Am J Sports Med 35:467–474
105. Baquie P (2000) Groin pain. Aust Fam Physician 29:158–160
106. Batt ME, Mc Shane JM, Dillingham MF (1995) Osteitis pubis in collegiate football players. Med Sci Sports Exerc 27(5):629–633
107. Anderson K, Strickland SM, Warren R (2001) Hip and groin injuries in athletes. Am J Sports Med 29:521–533
108. Gullmo A (1980) Herniography: the diagnosis of hernia in the groin and incompetence of the pouch of Douglas and pelvic floor. Acta Radiol Suppl 361:1–76
109. Bax T, Sheppard BC, Crass RA (1999) Surgical options in the management of groin hernias. Am Fam Physician 59:893–906
110. Ahumada LA, Ashruf S, Espinosa-de-los-Monteros A, Long JN, de la Torre JI, Garth WP, Vasconez LO (2005) Athletic pubalgia: definition and surgical treatment. Ann Plast Surg 55(4):393–396
111. Kachingwe AF, Grech S (2008) Proposed algorithm for the management of athletes with athletic pubalgia (sports hernia): a case series. J Orthop Sports Phys Ther 38(12):768–781
112. Muschaweck U, Berger L (2010) Minimal repair technique of sportsmen's groin: an innovative open-suture repair to treat chronic inguinal pain. Hernia 14:27–33
113. Nicholson J, Scott M (2012) Conjoint tendon disruption: redefining and recognizing "Gilmore's groin "a review of 1200 cases. Hernia 16:143–240
114. Polglase AL, Frydman GM, Farmer KC (1991) Inguinal surgery for debilitating chronic groin pain in athletes. Med J Aust 155(10): 674–677
115. Hackney RG (1993) The sports hernia: a cause of chronic groin pain. Br J Sports Med 27(1):58–62
116. Ingoldby CJ (1997) Laparoscopic and conventional repair of groin disruption in sportsmen. Br J Surg 84(2):213–215
117. Farber AJ, Wilckens JH (2007) Sports hernia: diagnosis and therapeutic approach. J Am Acad Orthop Surg 15(8):507–514
118. Moeller JL (2007) Sportsman's hernia. Curr Sports Med Rep 6(2):111–114
119. Caudill P, Nyland J, Smith C, Yerasimides J, Lach J (2008) Sports hernias: a systematic literature review. Br J Sports Med 42(12): 954–964
120. Susmallian S, Ezri T, Elis M, Warters R, Charuzi I, Muggia-Sullam M (2004) Laparoscopic repair of "sportman's hernia" in soccer players as treatment of chronic inguinal pain. Med Sci Monit 10(2):52–54
121. Peeters E, Spiessens C, Oyen R, De Wever L, Vanderscheren D, Pennickx F, Miserz M (2010) Laparoscopic inguinal hernia repair in man with lightweight meshes may significantly impair sperm motility: a randomized control trial. Ann Surg 252(2): 240–246
122. Mann CD, Sutton CD, Garcea G, Lloyd DM (2009) The inguinal release procedure for groin pain: initial experience in 73 sportsmen/women. Br J Sports Med 43:579–583
123. Azurin DJ, Go LS, Schuricht A, McShane J, Bartolozzi A (1997) Endoscopic preperitoneal herniorrhaphy in professional athletes with groin pain. J Laparoendosc Adv Surg Tech A 7(1):7–12
124. Kumar A, Doran J, Batt ME, Nguyen-Van-Tam JS, Beckingham IJ (2002) Results of inguinal canal repair in athletes with sports hernia. J R Coll Surg Edinb 47(3):561–565
125. Kluin J, Den Hoed PT, van Linschoten R, IJzerman JC, Van Steensel CJ (2004) Endoscopic evaluation and treatment of groin pain in the athlete. Am J Sports Med 32(4):944–949
126. Genitsaris M, Goulimaris I, Sikas N (2004) Laparoscopic repair of groin pain in athletes. Am J Sports Med 32(5):1238–1242
127. Edelman DS, Selesnick H (2006) "Sports" hernia: treatment with biologic mesh (Surgisis): a preliminary study. Surg Endosc 20(6):971–973
128. Van Veen RN, De Baat P, Heijboer MP, Kazemier G, Punt BJ, Dwarkasing RS, Bonjer HJ, van Eijck CH (2007) Successful endoscopic treatment of chronic groin pain in athletes. Surg Endosc 21(2):189–193
129. Van Der Donckt K, Steenbrugge F, Van Den Abbeele K, Verdonk R, Verhelst M (2003) Bassini's hernia repair and adductor longus tenotomy in the treatment of chronic groin pain in athletes. Acta Orthop Belg 69(1):35–41
130. Muschaweck U (2003) Umbilical and epigastric hernia repair. Surg Clin North Am 83(5):1207–1221
131. Avtan L, Avci C, Bulut T, Fourtanier G (1997) Mesh infections after laparoscopic inguinal hernia repair. Surg Laparosc Endosc 7(3):192–195
132. Bodenbach M, Bschleipfer T, Stoschek M, Beckert R, Sparwasser C (2002) Intravesical migration of a polypropylene mesh implant 3 years after laparoscopic transperitoneal hernioplasty. Urologe 41(4):366–368
133. Lange B, Langer C, Markus PM, Becker H (2003) Mesh penetration of the sigmoid colon following a transabdominal preperitoneal hernia repair. Surg Endosc 17(1):157
134. Peiper C, Junge K, Klinge U, Strehlau E, Ottinger A, Schumpelick V (2006) Is there a risk of infertility after inguinal mesh repair? Experimental studies in the pig and the rabbit. Hernia 10(1):7–12
135. Shin D, Lipshultz LI, Goldstein M, Barmé GA, Fuchs EF, Nagler HM, McCallum SW, Niederberger CS, Schoor RA, Brugh VM, Honig SC (2005) Herniorrhaphy with polypropylene mesh causing inguinal vasal obstruction: a preventable cause of obstructive azoospermia. Ann Surg 241(4):553–558
136. Akermark C, Johansson C (1992) Tenotomy of the adductor longus tendon in the treatment of chronic groin pain in athletes. Am J Sports Med 20:640–643
137. Atkinson HD, Johal P, Falworth MS, Ranawat VS, Dala-Ali B, Martin DK (2010) Adductor tenotomy: its role in the management of sports-related chronic groin pain. Arch Orthop Trauma Surg 130(8):965–970
138. Lohrer H, Nauck T (2007) Proximal adductor longus tendon tear in high level athletes. A report of three cases. Sportverletz Sportschaden 21(4):190–194
139. Robertson IJ, Curran C, McCaffrey N, Shields CJ, McEntee GP (2011) Adductor tenotomy in the management of groin pain in athletes. Int J Sports Med 32(1):45–48
140. Garvey JF (2012) Chronic athletic groin pain. One surgeon's approach. ASPETAR Sport Med J 1:24–27
141. Meyers WC, Lanfranco A, Castellanos A (2002) Surgical management of chronic lower abdominal and groin pain in high-performance athletes. Curr Sports Med Rep 1:301–305
142. Kunduracioglu B, Yilmaz C, Yorubulut M, Kudas S (2007) Magnetic resonance findings of osteitis pubis. J Magn Reson Imaging 25:535–539
143. Hölmich P (2013) Groin pain in football players. A systematic diagnostic approach. ASPETAR Sport Med J 2:192–196
144. Gokhale S (2007) Three-dimensional sonography of muscle hernias. J Ultrasound Med 26:239–242
145. Mellado JM, Perez del Palomar L (1999) Muscle hernias of the lower leg: MRI findings. Skeletal Radiol 28:465–469
146. Overdeck KH, Palmer WE (2004) Imaging of hip and groin injuries in athletes. Semin Musculoskelet Radiol 8:41–55

147. Morelli V, Weaver V (2005) Groin injuries and groin pain in athletes: part 2. Prim Care 32:185–200
148. Mason JB (2001) Acetabular labral tears in the athlete. Clin Sports Med 20:779–790
149. Huffman GR, Safran M (2002) Tears of the acetabular labrum in athletes: diagnosis and treatment. Sports Med Arthrosc Rev 10:141–150
150. Philippon MJ (2006) New frontiers in hip arthroscopy: the role of arthroscopic hip labral repair and capsulorrhaphy in the treatment of hip disorders. Instr Course Lect 55:309–316
151. Dorfmann H, Boyer T (1999) Arthroscopy of the hip: 12 years of experience. Arthroscopy 15:67–72
152. Byrd JWT, Jones KS (2000) Prospective analysis of hip arthroscopy with 2-year follow-up. Arthroscopy 16:578–587
153. O'Leary JA, Berend K, Vail TP (2001) The relationship between diagnosis and outcome in arthroscopy of the hip. Arthroscopy 17:181–188
154. McCarthy JC, Busconi B (1995) The role of hip arthroscopy in the diagnosis and treatment of hip disease. Can J Surg 38(Suppl 1):S13–S17
155. Brukner PD, Crossley KM, Morris H, Bartold SJ, Elliott B (2006) Recent advances in sports medicine. Med J Aust 184(4):188–193
156. Brittenden J, Robinson P (2005) Imaging of pelvic injuries in athletes. Br J Radiol 78:457–468
157. Blankenbaker DG, Tuite MJ (2006) The painful hip: new concepts. Skeletal Radiol 35(6):352–370
158. Cantwell CP, Obyrne J, Eustace S (2004) Current trends in treatment of osteoid osteoma with an emphasis on radiofrequency ablation. Eur Radiol 14:607–617
159. Ghanem I (2006) The management of osteoid osteoma: updates and controversies. Curr Opin Pediatr 18:36–41
160. Harvey G, Bell S (1999) Obturator neuropathy: an anatomic perspective. Clin Orthop Relat Res 363:203–211

Achilles Tendinopathies

9

Francesco Benazzo, Matteo Marullo, Cristian Indino, and Giacomo Zanon

Achilles tendinopathies are typical overuse injuries characterized by a combination of pain and swelling in and around the Achilles tendon, accompanied by impaired performance [1, 2]. In the last decades, terminology for impaired Achilles tendon disorders has changed constantly, so the lack of a common terminology led to semantic confusion.

In 1976, Perugia et al. were the first to propose a terminology based on histological findings [3]. They differentiated inflammatory disorders in peritendinitis, with inflammation of the peritendinous sheaths; peritendinitis with tendinosis, when tendon degeneration is associated with involvement of its sheaths; and pure tendinosis, characterized by degenerative phenomena exclusively. Tendinosis implied tendon degeneration, without any clinical or histological signs of intratendinous inflammation.

In 1998, Maffulli suggested to use the broadly used terms "tendinosis," "paratendinitis," and "tendinitis" only if histopathologically proven, not in clinical practice when discussing overuse tendon injuries [1]. In everyday clinical practice, he suggested the term tendinopathy to name the clinical syndrome of pain, swelling, and impaired performance.

The first classification according to location of pain was in 1992, when Clain and Baxter divided the definitions of Achilles "tendinitis" in insertional and non-insertional [4].

Actually, the definitions for Achilles tendon disorders comprise anatomic location, symptoms, clinical findings, and histopathology [5].

Midportion Achilles tendinopathy defines a pathology located 2–7 cm from the calcaneal insertion and involved degeneration of the tendon proper (tendinosis). Paratendinopathy is an acute or chronic inflammation of the tendon sheath, which causes pain during exercise and local swelling.

F. Benazzo • M. Marullo (✉) • C. Indino • G. Zanon
Clinica Ortopedica e Traumatologica, Università degli Studi di Pavia, IRCCS Fondazione Policlinico S. Matteo IRCCS, Vial Golgi, Pavia 27100, Italy
e-mail: f.benazzo@unipv.it; matteomarullo@hotmail.it; Zanon.g@libero.it

Insertional Achilles tendinopathy is located at the insertion of the Achilles tendon onto the calcaneus, possibly with the formation of bone spurs and calcifications in the tendon proper at the insertion site [5].

Midportion Achilles tendinopathy accounts for approximately 66 % of Achilles tendon pathology; disorders of the Achilles insertion account for another 20–25 % [6].

9.1 Etiology

The basic etiology of Achilles tendon overuse injuries is multifactorial. Several intrinsic and extrinsic factors predispose to these problems.

The most frequent intrinsic factor is malalignment of the lower extremity. Kwist found some kind of lower limb malalignment in 60 % of patients with Achilles tendon disorders [6]. The most common malalignment is foot hyperpronation. Other predisposing factors are excessive motion of the hindfoot in the frontal plane (a lateral heel strike with compensatory pronation), limited range of motion of the ankle joint, and forefoot varus [7, 8]. Leg length discrepancy is another contributing factor. A discrepancy of more than 5–6 mm in elite athletes may be symptomatic, and a shoe insert is recommended for more than 10 mm of difference [8]. Muscle weakness and imbalance can predispose to Achilles tendinopathy; muscular strength, endurance, and flexibility are important in the prevention of tendon injuries [8, 9].

Ankle joint laxity (e.g., secondary to a ligament injury) is another cause of Achilles tendinopathy [10]. Chronic Achilles tendon disorders, in particular insertional tendinopathy, are more common in older athletes than in younger (<20 years) ones [11, 12].

Less common intrinsic factors are inflammatory arthropathies, diabetes, hypertension, obesity, gout, hyperostotic conditions, and lipidemias [13].

The most common extrinsic factors are related to sport participation. They are associated with strenuous physical

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_9, © Springer International Publishing Switzerland 2016

activities such as running and jumping. The annual incidence of Achilles disorders in top-level runners is between 7 and 9 % [6].

Excessive loading of the lower extremities and training errors were found in 60–80 % of patients with Achilles tendinopathy [6, 13]. Inadequate footwear, irregular training surfaces, poor technique, or changes in training pattern can contribute to onset of the disease [14]. Even some drugs predispose to Achilles tendon pathology, like corticosteroids (local and systemic), fluoroquinolones, anabolic steroids, or narcotics; they inhibit tenocyte proliferation and reduce collagen and extracellular matrix synthesis.

9.2 Injury Mechanism

Overuse Achilles injuries are consequences of repetitive tendon strains. When tendon has been repeatedly strained to 5–8 % strain, microscopic disruption of the tendon fibers occurs. The high frequency of this process outpaces the intrinsic healing capacity of the tendon. To be homogeneous and stiff, the tendon tissue is poorly vascularized, so its anabolism is slow. Vascularization is particularly reduced in the central third of the tendon, a region 2–6 cm far from the calcaneal insertion.

The impaired healing response led to intrinsic tendon degeneration (tendinosis), which is histologically recognized as haphazard proliferation of tenocytes, cellular degeneration, and disruption of collagen fibers with increase in noncollagenous matrix [1, 15, 16].

Peritendinitis is an acute inflammation of the tendon sheath caused by excessive forces on the tendon. Acute peritendinitis is characterized by edema and hyperemia of the paratenon with infiltration of inflammatory cells and production of a fibrinous exuding chronic peritendinitis; the exudate rich in fibrin led to paratenon thickening and forms adhesion between tendon, paratenon, and crural fascia [17].

At the calcaneal insertion, Achilles tendinopathy affects more frequently the anterior aspect of the tendon. As the posterior aspect undergoes a higher strain on dorsiflexion, it has been thought that stress shielding and potential underuse phenomenon play a role in the etiology of insertional tendinopathy [18]. Insertional tendinopathy is also associated with a posterosuperior calcaneal prominence, also called Haglund's disease; this is an insertional spur secondary to an adaptive process to tendon microtears or inflammatory changes rather than their cause. Moreover, the retrocalcaneal bursa is squeezed between the tendon and the calcaneal prominence during dorsiflexion; this repetitive process could lead to inflammation, synovial fold hypertrophy, and bursitis.

9.3 Clinical and Diagnostic Examination

A careful history collection and a detailed clinical examination are mandatory for a correct diagnosis. Diagnostic imaging is required to verify a clinical suspicion or to exclude other musculoskeletal disorders, such as os trigonum syndrome, pathologies of the peroneal tendons or the plantar flexors, and neuroma of the sural nerve [14].

Pain is the main symptom of Achilles tendinopathy. Generally, it occurs at the beginning and at the end of a training session, with a period of decreased discomfort in between. In severe cases, it can oblige the athlete to stop it and also interfere with activities of daily living. A report of morning stiffness is also indicative.

Clinical examination should be performed with the patient standing and then prone.

The foot and the heel should be examined for any malalignment, deformity, Haglund's heel, asymmetry in the tendons widths, and localized thickening.

The first clinical test to perform is the calf squeeze test, to exclude a rupture of the Achilles tendon. Then tendon excursion should be estimated to determine any tightness or painful arc of movement. Any tendon swelling should be evaluated also during active ankle dorsiflexion and plantar flexion. Moreover, a careful palpation of the entire length of the tendon permits to detect any swelling or nodule and any tender point. The tendon should be gently squeezed between the thumb and the index finger (Fig. 9.1). A localized area of tendon swelling 2–6 cm proximal to the calcaneal insertion with a painful nodule that moves with the tendon during ankle movements is indicative of midportion

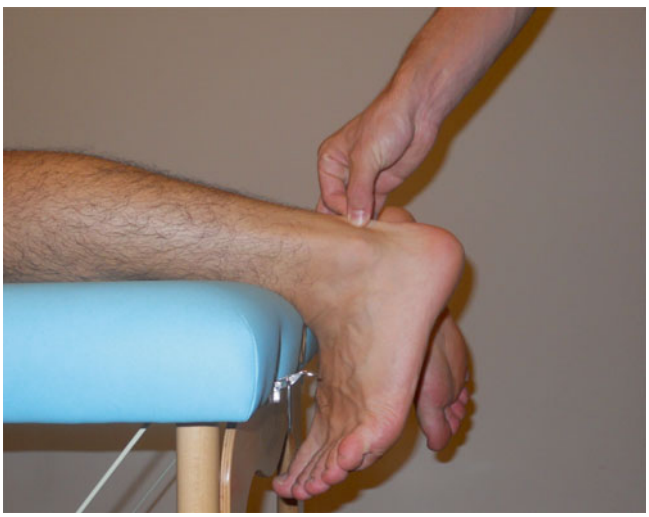


Fig. 9.1 Palpation of a left Achilles tendon. The tendon should be squeezed between the thumb and the index fingers; this procedure should be performed for all the length of the tendon to appreciate any swelling or nodule

Achilles tendinopathy. If the localized tenderness and swelling remains fixed during dorsiflexion and plantar flexion, it is indicative of paratendinopathy. In acute paratendinopathy, fibrin precipitation around the tendon can cause palpable crepitation when the skin is glided over the tendon; when paratendinopathy moves to chronic, crepitation reduces. A warm tendon is another indicative sign of paratendinopathy.

Other tests are performed with the patient standing. Tendon stretching during passive dorsiflexion of the ankle is performed placing the affected leg backward and leaning forward; the heel must not lift off the ground. The test is positive if the patient complains of pain during the exercise. Other tests are the single-leg heel raise, performed by the patient rising up onto tiptoes and lowering back to the floor on the affected leg, and the hop test, performed by the patient hopping forward over a line marked on the floor. Weakness and/or pain during these exercises are suspected for Achilles tendinopathy.

Anyway, in evaluating reproducibility and accuracy of all these clinical test for Achilles tendinopathy, Hutchison et al. demonstrate that "palpation" and "self-reported pain" are the most valid ones [19].

Imaging studies are important to confirm the diagnosis and to evaluate the entity of the disease and its evolution. They comprise magnetic resonance imaging, ultrasonography, color and power Doppler, and sonoelastography.

Magnetic resonance imaging (MRI) provides extensive information about the internal and external morphology of tendon. It is useful to evaluate the stages of chronic degeneration and to differentiate between paratendinopathy and tendinopathy. Mucoid degeneration (altered collagen fiber structure and increased extracellular matrix) is visible as increased signal intensity areas on T1- and T2-weighted images (Fig. 9.2). Any peritendinous fluid or inflammation is clearly visible as hypointense in T1 and hyperintense in T2 [20].

Ultrasonography is more widespread and easier to obtain than MRI, even if it is operator dependent. Tendon thickening and peritendinous swelling or edema are easily detectable with US. Intratendinous hypoechoic areas are signs of Achilles tendinosis. One of the main advantages of US in comparison with other imaging modalities is the interactive facility, which helps in reproducing symptoms by transducer compression and concentrate on the pathologic area [21].

Color and power Doppler improve standard ultrasound tendon imaging demonstrating entity and pattern of the blood flow in the tissue. In normal Achilles tendon, blood flow is not detectable; in Achilles tendon disorders, blood flow increases and is linked with greater pain scores, poorer function, and longer symptoms in the Achilles tendon [22].

Sonoelastography is a novel US technique that can assess the elastic properties of tissues [23]. After a mechanical compression (performed by the examiner with the probe),

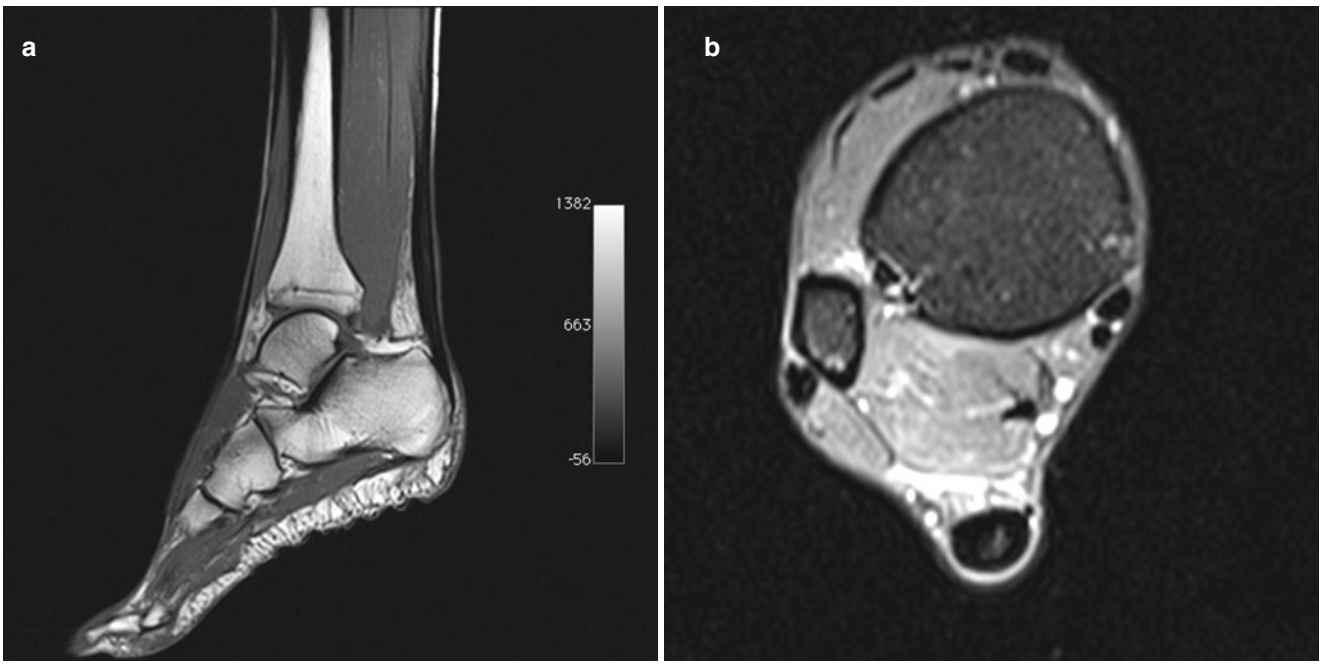


Fig. 9.2 Magnetic resonance imaging of a 38-year-old female runner with left midportion Achilles tendinopathy. (**a**) T1 sagittal view shows midportion tendon thickening with an area of increased signal. (**b**) Proton density axial view shows a hyperintense area into the tendon, sign of intratendinous mucoid degeneration

any tissue is deformed with a specific spatial and temporal pattern. The elastic properties of normal Achilles tendon are altered under pathological conditions, and a distinct intratendinous softening can be detected by sonoelastography. This new promising technique could be useful in detecting early-stage tendinopathy and in monitoring its evolution during treatments.

9.4 Treatment Strategy

Treatment differs according to the site of the disorder: insertional or non-insertional.

9.4.1 Non-insertional Achilles Tendinopathy

9.4.1.1 Nonoperative Treatment

Conservative treatment is recommended as the initial strategy by most authors [24].

Identification and correction of possible etiological factors combined with a symptom-related approach permit to return to previous activities in most patients; an 8-year follow-up study showed a failure rate of only 29 % with conservative treatment [25].

Initial rest (complete or modified activity), modification of training regimes, specific exercises, and correction of underlying lower limb malalignment with orthoses (heel lift, change of shoes, corrections of malalignments) are the mainstay of the treatment [26, 27].

The use of nonsteroidal anti-inflammatory drugs (NSAIDs) is debatable because there is no evidence of their effects on symptoms; moreover, the histologic absence of inflammatory cells in the tendinopathic tissue led its use questionable [28, 29]. NSAIDs should even have detrimental effects; an in vitro study demonstrated that celecoxib inhibits tendon cell migration and proliferation [30].

Corticosteroid injections are reported to reduce pain and swelling. These early benefits are counterbalanced by reports of adverse effects in up to 82 % of corticosteroid trials [31], in particular tendon rupture [32, 33] and decreased tendon strength [34, 35].

Eccentric exercises are beneficial in the early treatment of non-insertional Achilles tendinopathy; they lead to normalization of tendon structure and reduction in neovascularization, even if the exact mechanism by which they work is poorly understood [36, 37].

Alfredson et al. reported return to normal activities at 12 weeks in 82 % of cases in the group treated with eccentric exercises, compared with 36 % of patients treated with concentric exercises used concentric exercises; improvement was still present at 12 months [38, 39]. Anyway, the results of eccentric exercises observed from other study groups out from Scandinavia are less convincing, with only a 50–60 % of good outcome [40, 41].

The results of extracorporeal shock wave therapy in treating midportion Achilles tendinopathy are conflicting. Low-energy shock wave therapy and eccentric training produced comparable results, superior to the wait-and-see policy [41]. The combination of these two treatment strategies led to significant improvement and better outcome than eccentric exercises alone [42].

Platelet-rich plasma (PRP) injection is going to become widely used in several orthopedic areas. It demonstrated improved tendon healing compared with control in vitro, but a randomized double-blind placebo-controlled trial showed no difference in improvement in pain and activity at 6 months [43, 44].

Sclerosing therapy with polidocanol injections has the objective of destroying neovessels and the consequent neonerves in the paratenon to reduce pain and even leading intratendinous remodeling. The first authors introducing this treatment strategy reported good clinical results with 38 of 42 patients satisfied at 2-year follow-up [45]. However, newer contrasting results have been reported. In a recent study, van Sterkenburg et al. reported that only 44 % of 53 tendons were painless or minimally painful at 6 weeks after treatment with 3 sessions of ethoxysclerol injections [46]. Moreover, at 2.7–5.1-year follow-up, 53 % of tendons had undergone additional (nonoperative or surgical) treatment.

Peritendinous high-volume injections of anesthetic (10 ml 0.5 % bupivacaine) and normal saline solution (40 ml) seem effective in reducing pain and improving function; anyway, these studies are limited case series and with limited follow-up [47, 48].

Intratendinous hyperosmolar dextrose injections (prolotherapy) are another promising treatment in vitro but wide clinical studies with mid- and long-term follow-up are lacking [49].

9.4.1.2 Surgical Treatment

Conservative treatment for midportion Achilles tendinopathy fails to resolve symptoms and to allow sports resumption in 24–45.5 % of cases [50].

Traditional surgical treatment consists in open release of adhesions with or without resection of the paratenon. Multiple tenotomies are performed to initiate vascular ingrowth from the surrounding tissue, and intratendinous areas of tendinopathy are excised [51–53]. If debridement is superior to 50 % or the tendon width, augmentation should be performed with plantaris tendon or turndown flaps for small defects or tendon transfer (peroneus brevis, flexor digitorum longus, flexor hallucis longus) for larger defects [54, 55].

Recently, the transfer of a soleus pedicle graft into a central tenotomy was proposed, with the aim of increasing tendon blood supply and promoting a faster tendon healing (Fig. 9.3) [56]. When compared with longitudinal tenotomies, despite similar outcomes in postoperative functional scores at the 4-year

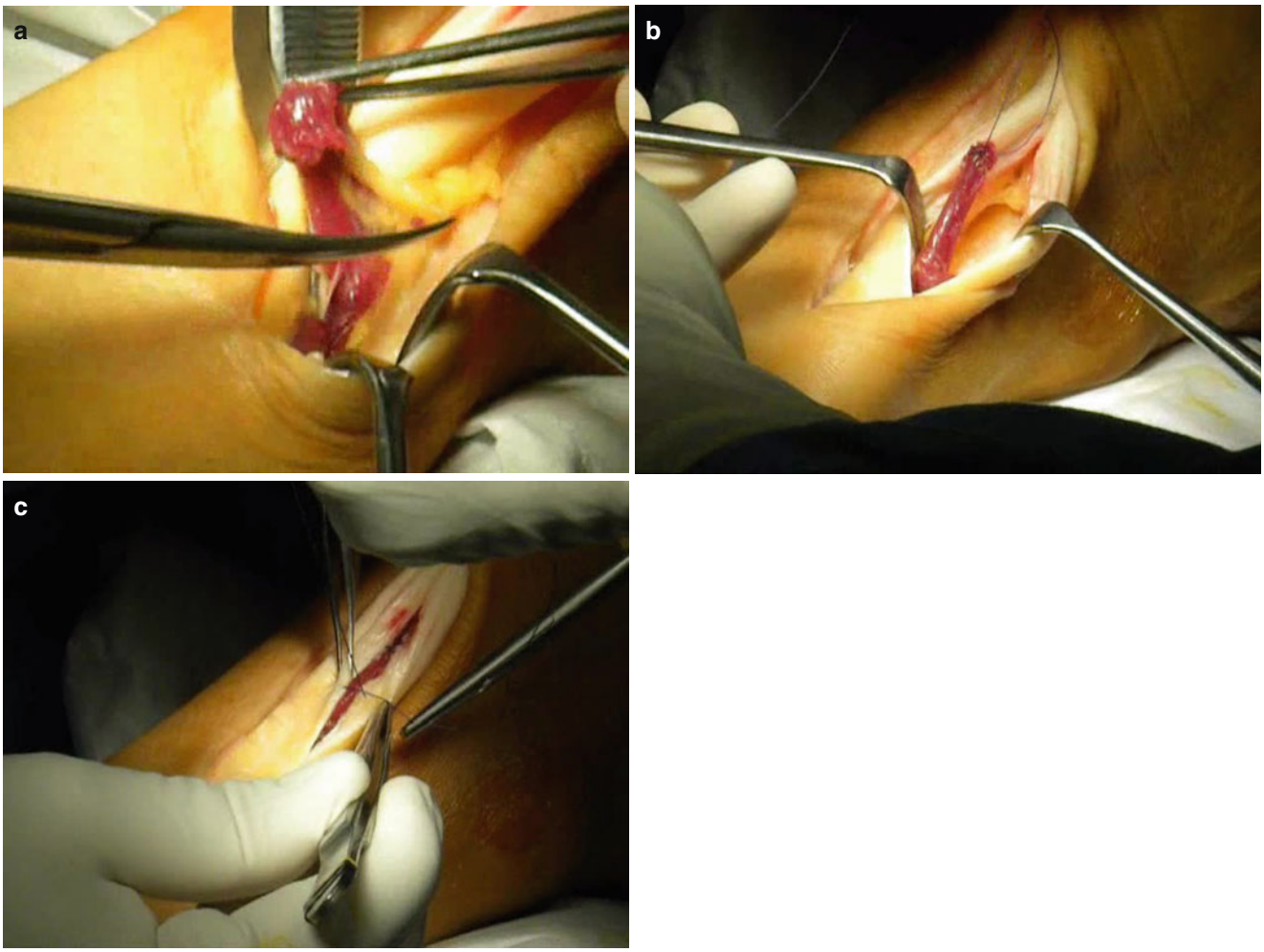


Fig. 9.3 Intraoperatory view of the soleus fiber transfer technique performed in a left Achilles tendon. (**a**) After a lateral paratendon incision, a cylindrical bundle of the inferolateral portion of the soleus muscle is isolated by blunt dissection. (**b**) The muscle bundle, left attached at its distal end, is flipped 180° and placed inside a full-thickness tenotomy in a "sandwich" way. (**c**) The bundle is secured with stitches, and the tenotomy is closed

follow-up, soleus transfer allows a faster recovery and return to run but has a higher incidence of tendon thickening [56].

The main concern about open surgery for midportion Achilles tendinopathy is the non-negligible risk of wound healing complications [53, 55]. Other complications are nerve and soft tissues damage. Paavola et al., in a large series of 432 consecutive patients, reported an overall complication rate of 11 % at 5 months of follow-up [55]; the most frequent ones were wound edge necrosis (3 %), superficial infection (2.5 %), and sural nerve irritation (1 %).

Stripping of the paratenon follows the same principle of sclerosing injection: destroying the neovessel and the neonerves around the Achilles tendon in order to reduce pain. This technique reported good results in 75–100 % of cases [57, 58], even if they were case series with a limited number of patients treated. Moreover, Naidu et al. reported a complication rate of 7 %, mainly due to delayed wound healing [58]. Endoscopic tendon debridement seems promising, but studies with a consistent number of patients are lacking [59, 60]. Maquirriain performed 27 endoscopic paratenon debridement and longitudinal tenotomies in 24 patients with midportion Achilles tendinopathy [59]. All patients had an improved clinical outcome at a mean final follow-up of 7.7 years (range 5–14). 96 % of patients obtained a complete resolution of symptoms. Pearce et al. added plantaris tendon release to tenoscopy in 11 patients with midportion Achilles tendinopathy [60]. After a minimum follow-up of 2 years, the mean AOFAS scores improved from 68 preoperatively to 92 postoperatively ($p = 0.0002$). Eight patients were satisfied.

9.4.2 Insertional Achilles Tendinopathy

9.4.2.1 Nonoperative Treatment

The first therapeutic strategy is to reduce load-bearing activities and modification of training regimes. Immobilization

should be avoided for its detrimental effect on tendon structure and strength [61].

Heel lift permits to reduce pressure on the retrocalcaneal bursa by plantar flexing the heel, potentially favoring healing of the tendon insertion. Orthoses to correct any malalignment are helpful to improve symptoms [62].

The same eccentric exercise program already applied in midportion Achilles tendinopathy was also evaluated in insertional diseases. The good results obtained for non-insertional disease were not replicated for insertional pathology; it showed success rates of 28–32 % [62, 63]. Eccentric exercises with ankle dorsiflexion were supposed to be detrimental in insertional pathology. Jonsson et al. proposed eccentric training using floor-level exercises only, founding improved outcomes in 67 % of cases [64].

Regular stretching may increase the working length of the muscle–tendon unit; indeed, it increases ankle dorsiflexion by only 1° [65].

Shock wave treatment seems effective in treating insertional tendinopathy; Rompe et al. reported good or excellent results after 4 months in almost 83 % of patients compared to 39 % in conventionally treated patients [66].

Corticosteroids are rarely used because of their historic risk of partial or complete tendon rupture [67]. Injections of hyperosmolar dextrose solution, sclerosing agents, or platelet-rich plasma seemed promising in treating insertional pathologies, but only one study for each treatment is reported [68–70].

9.4.2.2 Operative Treatment

Endoscopic calcaneoplasty and bursa debridement showed good results in 75–95 % of patients treated, with small scars, minimal debridement, and rapid recovery [62, 71].

Open surgery is more invasive and restricted for recalcitrant disease; it consists of debridement of the tendon insertion, removal of bursal tissue and of bony prominence, reattachment of the insertion as required (with bone anchors/screws or trans-osseous sutures), and/or augmentation of the tendon with a tendon transfer/graft [62]. Even the 50 % of the tendon insertion can be debrided with minimal risk or rupture [72].

The best graft used for augmentation of the Achilles tendon is the flexor hallucis longus (FHL). It is in proximity, is synergist, and has good vascularity [73]. Peroneus brevis should also be employed, but subsequent ankle instability and foot inversion can occur [74]. Even if weaker than the FHL and more difficult to harvest, the flexor digitorum longus should also be used [75]. Other sources of tendon tissue are autograft as quadriceps, patellar tendon, or hamstring. Also the V–Y advancement of the musculotendinous junction of the gastrocnemius should be performed in repairing insertional defects longer than 2 cm.

9.5 Rehabilitation and Return to Play

Achilles tendinopathies represent a big chapter in sport traumatology and, as previously shown, a broad spectrum of treatments are possible. The choice depends from the type of pathology, its location and degree, and the preference and the level of expertise of the clinician. The number of different treatments reflects the difficulty in treating these pathologies and their often unpredictable outcome.

Eccentric exercises are historically proposed as a 12-week program; it led to satisfactory outcome in 50–60 % of midportion Achilles tendinopathies [40, 41] and in 28–32 % of insertional tendinopathies [62, 63].

In 20–30 % of cases, conservative treatment fails in improving symptoms and allowing sports resumption [75]. In these cases, surgery is the second step of treatment. Minimally invasive surgery permits a lower incidence of complications, in particular wound infection or delayed healing, and also a faster recovery. All procedures that address only peritendinous structures showed a recovery time of 6 weeks to 6 months. After these techniques, complete mobilization of the ankle and partial weight bearing is permitted till the first day after surgery. On the contrary, surgical procedures that address the tendon itself (debridement, incisions, augmentations) temporarily weaken it. Therefore, recovery time can be extended up to 3–18 months. In these cases, the ankle should be immobilized in a dorsal splint or in a cast up to 3 weeks with the ankle in slight plantar flexion. Partial weight bearing will be allowed after cast removal. Mobilization will be performed passively, then actively in open kinetic chain. Once there is complete recovery of the range of motion, closed kinetic chain exercises start. Cycling and walking in the water play an important role in recovery range of motion and strength of calf muscles. When the patient is able to walk without limping, gentle stretching and light jogging are allowed. Exercises for static and dynamic proprioception are central in the rehabilitation process.

Sport-specific exercises can be introduced when the calf muscle strength is close to normality (close to the contralateral side). A close monitoring of the healing process by imaging is suggested; ultrasonography is a fast and easy way to do it, even if it is operator dependent. Magnetic resonance imaging permits a finest evaluation, even if it is not often readily available.

Return to play is permitted when the patient passes specific functional tests as the single-leg hop test, the triple-leg hop test, the shuttle run test, the side jump test, and the carioca test or with the isokinetic evaluation.

References

1. Maffulli N, Khan KM, Puddu G (1998) Overuse tendon conditions: time to change a confusing terminology. Arthroscopy 14: 840–843
2. Khan KM, Cook JL, Kannus P, Maffulli N, Bonar SF (2002) Time to abandon the "tendinitis" myth. BMJ 324:626–627
3. Perugia L, Ippolito E, Postacchini F (1976) A new approach to the pathology, clinical features and treatment of stress tendinopathy of the Achilles tendon. Ital J Orthop Traumatol 2:5–21
4. Clain MR, Baxter DE (1992) Achilles tendinitis. Foot Ankle 13:482–487
5. van Dijk CN, van Sterkenburg MN, Wiegerinck JI, Karlsson J, Maffulli N (2011) Terminology for Achilles tendon related disorders. Knee Surg Sports Traumatol Arthrosc 19(5):835–841
6. Kvist M (1994) Achilles tendon injuries in athletes. Sports Med 18:173–201
7. Leppilahti J, Orava S, Karpakka J, Takala T (1991) Overuse injuries of the Achilles tendon. Ann Chir Gynaecol 80:202–207
8. Kannus P (1997) Etiology and pathophysiology of chronic tendon disorders in sports. Scand J Med Sci Sport 7(2):78–85
9. Lysholm J, Wiklander J (1987) Injuries in runners. Am J Sports Med 115:168–171
10. Holmes GB, Lin J (2006) Etiologic factors associated with symptomatic Achilles tendinopathy. Foot Ankle Int 27:952–959
11. Kannus P, Niittymki S, Jarvinen M, Lehto M (1989) Sports injuries in elderly athlete: a three-year prospective, controlled study. Age Ageing 18:263–270
12. de Jonge S, van den Berg C, de Vos RJ, van der Heide HJ, Weir A, Verhaar JA, Bierma-Zeinstra SM, Tol JL (2011) Incidence of midportion Achilles tendinopathy in the general population. Br J Sports Med 45(13):1026–1028
13. Paavola M, Kannus P, Järvinen TA, Lehto M (2002) Achilles tendinopathy. J Bone Joint Surg Am 84-A:2062–2076
14. Longo UG, Ronga M, Maffulli N (2009) Achilles tendinopathy. Sports Med Arthrosc 17(2):112–126
15. Movin T, Gad A, Reinholt FP, Rolf C (1997) Tendon pathology in long-standing achillodynia. Biopsy findings in 40 patients. Acta Orthop Scand 68:170–175
16. Jarvinen TAH, Kannus P, Maffulli N, Khan KM (2005) Achilles tendon disorders: etiology and epidemiology. Foot Ankle Clin 10:255–266
17. Paavola M, Jarvinen TA (2005) Paratendinopathy. Foot Ankle Clin 10:279–292
18. Lyman J, Weinhold PS, Almekinders LC (2004) Strain behavior of the distal Achilles tendon: implications for insertional Achilles tendinopathy. Am J Sports Med 32:457–461
19. Hutchison AM, Evans R, Bodger O, Pallister I, Topliss C, Williams P, Vannet N, Morris V, Beard D (2013) What is the best clinical test for Achilles tendinopathy? Foot Ankle Surg 19:112–117
20. Movin T, Kristoffersen-Wiberg M, Shalabi A, Gad A, Aspelin P, Rolf C (1998) Intratendinous alterations as imaged by ultrasound and contrast medium-enhanced magnetic resonance in chronic achillodynia. Foot Ankle Int 19:311–317
21. Gibbon WW (1996) Musculoskeletal ultrasound. Baillieres Clin Rheumatol 10:561–588
22. Peers K, Brys P, Lysens R (2003) Correlation between power Doppler ultrasonography and clinical severity in Achilles tendinopathy. Int Orthop 27:180–183
23. Klauser AS, Faschingbauer R, Jaschke WR (2010) Is sonoelastography of value in assessing tendons? Semin Musculoskelet Radiol 14:323–333
24. Kader D, Saxena A, Movin T, Maffulli N (2002) Achilles tendinopathy: some aspects of basic science and clinical management. Br J Sports Med 36:239–249
25. Paavola M, Kannus P, Paakkala T, Pasanen M, Järvinen M (2000) Long-term prognosis of patients with Achilles tendinopathy: an observational 8-year follow-up study. Am J Sports Med 28:634–642
26. Alfredson H, Cook J (2007) A treatment algorithm for managing Achilles tendinopathy: new treatment options. Br J Sports Med 41:211–216
27. Gross ML, Davlin LB, Evanski PM (1991) Effectiveness of orthotic shoe inserts in the long-distance runner. Am J Sports Med 19:409–412
28. Aström M, Westlin N (1992) No effect of piroxicam on Achilles tendinopathy: a randomized study of 70 patients. Acta Orthop Scand 63:631–634
29. Fredberg U, Stengaard-Pedersen K (2008) Chronic tendinopathy tissue pathology, pain mechanisms, and etiology with a special focus on inflammation. Scand J Med Sci Sports 18:3–15
30. Tsai WC, Hsu CC, Chou SW, Chung CY, Chen J, Pang JH (2007) Effects of celecoxib on migration, proliferation and collagen expression of tendon cells. Connect Tissue Res 48:46–51
31. Hart L (2011) Corticosteroid and other injections in the management of tendinopathies: a review. Clin J Sport Med 21:540–554
32. Smith AG, Kosygan K, Williams H, Newman RJ (1999) Common extensor tendon rupture following corticosteroid injection for lateral tendinosis of the elbow. Br J Sports Med 33:423–424
33. Gottlieb NL, Riskin WG (1980) Complications of local corticosteroid injections. JAMA 243:1547–1548
34. Hugate R, Pennypacker J, Saunders M, Juliano P (2004) The effects of intratendinous and retrocalcaneal intrabursal injections of corticosteroid on the biomechanical properties of rabbit Achilles tendons. J Bone Joint Surg Am 86-A:794–801
35. Haraldsson BT, Langberg H, Aagaard P, Zuurmond AM, van El B, Degroot J, Kjaer M, Magnusson SP (2006) Corticosteroids reduce the tensile strength of isolated collagen fascicles. Am J Sports Med 34:1992–1997
36. Mafi N, Lorentzon R, Alfredson H (2001) Superior short-term results with eccentric calf muscle training compared to concentric training in a randomized prospective multicenter study on patients with chronic Achilles tendinosis. Knee Surg Sports Traumatol Arthrosc 9:42–47
37. Öhberg L, Alfredson H (2004) Effects on neovascularisation behind the good results with eccentric training in chronic mid-portion Achilles tendinosis? Knee Surg Sports Traumatol Arthrosc 12:465–470
38. Alfredson H, Pietilä T, Jonsson P, Lorentzon R (1998) Heavy-load eccentric calf muscle training for the treatment of chronic Achilles tendinosis. Am J Sports Med 26:360–366
39. Silbernagel KG, Thomeé R, Eriksson BI, Karlsson J (2007) Continued sports activity, using a pain-monitoring model, during rehabilitation in patients with Achilles tendinopathy: a randomized controlled study. Am J Sports Med 35:897–906
40. Sayana MK, Maffulli N (2007) Eccentric calf muscle training in non-athletic patients with Achilles tendinopathy. J Sci Med Sport 10:52–58
41. Rompe JD, Nafe B, Furia JP, Maffulli N (2007) Eccentric loading, shock- wave treatment, or a wait-and-see policy for tendinopathy of the main body of tendo Achillis: a randomized controlled trial. Am J Sports Med 35:374–383
42. Rompe JD, Furia J, Maffulli N (2009) Eccentric loading versus eccentric loading plus shock-wave treatment for midportion

Achilles tendinopathy: a randomized controlled trial. Am J Sports Med 37:463–470
43. de Mos M, van der Windt AE, Jahr H, van Schie HT, Weinans H, Verhaar JA, van Osch GJ (2008) Can platelet-rich plasma enhance tendon repair? A cell culture study. Am J Sports Med 36:1171–1178
44. de Jonge S, de Vos RJ, Weir A, van Schie HT, Bierma-Zeinstra SM, Verhaar JA, Weinans H, Tol JL (2011) One-year follow-up of platelet-rich plasma treatment in chronic Achilles tendinopathy: a double-blind randomized placebo-controlled trial. Am J Sports Med 39:1623–1629
45. Lind B, Ohberg L, Alfredson H (2006) Sclerosing polidocanol injections in mid-portion Achilles tendinosis: remaining good clinical results and decreased tendon thickness at 2-year follow-up. Knee Surg Sports Traumatol Arthrosc 14(12):1327–1332
46. van Sterkenburg MN, de Jonge MC, Sierevelt IN, van Dijk CN (2010) Less promising results with sclerosing Ethoxysclerol injections for midportion Achilles tendinopathy: a retrospective study. Am J Sports Med 38(11):2226–2232
47. Chan O, O'Dowd D, Padhiar N, Morrissey D, King J, Jalan R, Maffulli N, Crisp T (2008) High volume image guided injections in chronic Achilles tendinopathy. Disabil Rehabil 30:1697–1708
48. Humphrey J, Chan O, Crisp T, Padhiar N, Morrissey D, Twycross-Lewis R, King J, Maffulli N (2010) The short-term effects of high volume image guided injections in resistant non-insertional Achilles tendinopathy. J Sci Med Sport 13:295–298
49. Freeman JW, Empson YM, Ekwueme EC, Paynter DM, Brolinson PG (2011) Effect of prolotherapy on cellular proliferation and collagen deposition in MC3T3-E1 and patellar tendon fibroblast populations. Transl Res 158:132–139
50. Maffulli N, Kader D (2002) Tendinopathy of tendo achillis. J Bone Joint Surg Br 84(1):1–8
51. Rolf C, Movin T (1997) Etiology, histopathology, and outcome of surgery in achillodynia. Foot Ankle Int 18:565–569
52. Nelen G, Martens M, Burssens A (1989) Surgical treatment of chronic Achilles tendinitis. Am J Sports Med 17:754–759
53. Tallon C, Coleman BD, Khan KM, Maffulli N (2001) Outcome of surgery for chronic Achilles tendinopathy: a critical review. Am J Sports Med 29:315–320
54. Paavola M, Kannus P, Orava S, Pasanen M, Järvinen M (2002) Surgical treatment for chronic Achilles tendinopathy: a prospective seven month follow up study. Br J Sports Med 36:178–182
55. Paavola M, Orava S, Leppilahti J, Kannus P, Järvinen M (2000) Chronic Achilles tendon overuse injury: complications after surgical treatment: an analysis of 432 consecutive patients. Am J Sports Med 28:77–82
56. Benazzo F, Zanon G, Klersy C, Marullo M (2014) Open surgical treatment for chronic midportion achilles tendinopathy: faster recovery with the soleus fibres transfer technique. Knee Surg Sports Traumatol Arthrosc. doi:10.1007/s00167-014-3232-x
57. Longo UG, Ramamurthy C, Denaro V, Maffulli N (2008) Minimally invasive stripping for chronic Achilles tendinopathy. Disabil Rehabil 30:1709–1713
58. Naidu V, Abbassian A, Nielsen D, Uppalapati R, Shetty A (2009) Minimally invasive paratenon release for non-insertional Achilles tendinopathy. Foot Ankle Int 30(7):680–685
59. Maquirriain J (2013) Surgical treatment of chronic Achilles tendinopathy: long-term results of the endoscopic technique. J Foot Ankle Surg 52(4):451–455
60. Pearce CJ, Carmichael J, Calder JD (2012) Achilles tendinoscopy and plantaris tendon release and division in the treatment of non-insertional Achilles tendinopathy. Foot Ankle Surg 18(2): 124–127
61. Johnson MD, Alvarez RG (2012) Nonoperative management of retrocalcaneal pain with AFO and stretching regimen. Foot Ankle Int 33:571–581
62. Wiegerinck JI, Kerkhoffs GM, van Sterkenburg MN, Sierevelt IN, van Dijk CN (2012) Treatment for insertional Achilles tendinopathy: a systematic review. Knee Surg Sports Traumatol Arthrosc 21:1345–1355
63. Fahlström M, Jonsson P, Lorentzon R, Alfredson H (2003) Chronic Achilles tendon pain treated with eccentric calf-muscle training. Knee Surg Sports Traumatol Arthrosc 11:327–333
64. Jonsson P, Alfredson H, Sunding K, Fahlström M, Cook J (2008) New regimen for eccentric calf-muscle training in patients with chronic insertional Achilles tendinopathy: results of a pilot study. Br J Sports Med 42:746–749
65. Rosenbaum D, Hennig EM (1995) The influence of stretching and warm-up exercises on Achilles tendon reflex activity. J Sports Sci 13:481–490
66. Rompe JD, Furia J, Maffulli N (2008) Eccentric loading compared with shock wave treatment for chronic insertional Achilles tendinopathy: a randomized, controlled trial. J Bone Joint Surg Am 90-A:52–61
67. Mahler F, Fritschy D (1992) Partial and complete ruptures of the Achilles tendon and local corticosteroid injections. Br J Sports Med 26:7–14
68. Ryan M, Wong A, Taunton J (2010) Favorable outcomes after sonographically guided intratendinous injection of hyperosmolar dextrose for chronic insertional and midportion Achilles tendinosis. Am J Roentgenol 194:1047–1053
69. Ohberg L, Alfredson H (2003) Sclerosing therapy in chronic Achilles tendon insertional pain: results of a pilot study. Knee Surg Sports Traumatol Arthrosc 11:339–343
70. Monto RR (2012) Platelet rich plasma treatment for chronic Achilles tendinosis. Foot Ankle Int 33:379–385
71. Jerosch J, Schunck J, Sokkar SH (2007) Endoscopic calcaneoplasty (ECP) as a surgical treatment of Haglund's syndrome. Knee Surg Sports Traumatol Arthrosc 15:927–934
72. Wagner E, Gould JS, Kneidel M, Fleisig GS, Fowler R (2006) Technique and results of Achilles tendon detachment and reconstruction for insertional Achilles tendinosis. Foot Ankle Int 27:677–684
73. Panchbhavi VK (2007) Chronic achilles tendon repair with flexor hallucis longus tendon harvested using a minimally invasive technique. Tech Foot Ankle Surg 6:123–129
74. White RK, Kraynick BM (1959) Surgical uses of the peroneus brevis tendon. Surg Gynecol Obstet 108:117–121
75. Roche AJ, Calder JDF (2013) Achilles tendinopathy. Bone Joint J 95-B:1299–1307

Achilles Tendon Rupture

10

Nicola Maffulli, Alessio Giai Via, and Francesco Oliva

10.1 Introduction

The Achilles tendon (AT) is the thickest and the strongest tendon in the human body with a tensile strength in the order of 50–100 N/mm [1]. About 15 cm long, it originates in the midcalf and extends distally to insert into the posterior surface of the calcaneus. It is formed from the joining of the two tendons of soleus (dorsally) and gastrocnemius (ventrally). Despite its strength, it is one of the tendons most commonly affected by spontaneous rupture. Most acute ruptures (75 %) occur during recreational activities in men between 30 and 40 years old, in particular in soccer, basketball, tennis, and squash, but 25 % of ruptures may occur in sedentary patients [2]. The incidence rate ranges from 6 to 18 per 100,000 people per year, and it has been steadily increasing during the past few decades [2].

AT rupture can present acutely or as chronic tears (>6 weeks). Management of acute ruptures is still controversial. Recent well-conducted randomized controlled trials showed that conservative treatment with accelerated functional rehabilitation and open surgery management produce, in an unselected population, similar functional results [3, 4]. However, a relatively higher re-rupture rate is still reported in patients treated conservatively [4, 5], and healing in a lengthened position may determine loss of calf muscle strength. These results are not acceptable in young patients and athletes. In these patients, operative management provides lower re-rupture rate, early functional treatment, less calf atrophy, and better functional performance than nonsurgical treatment.

10.2 Etiopathogenesis and Injury Mechanism

Acute AT rupture is a serious injury for high-level athletes. Tendon fibers begin to disrupt after a length increase of 3–4 % and rupture after an increase of 8 %. Ruptures usually occur between 2 and 6 cm of its insertion into the superior surface of the calcaneus. The tendon is at the greatest risk of rupture when it is obliquely loaded, the muscle is contracting maximally, and the tendon length is short [6]. The most common mechanism of injury is pushing off with the weight-bearing forefoot while extending the knee. Sudden unexpected dorsiflexion of the ankle or violent dorsiflexion of a plantar flexed foot may also result in ruptures [6]. Men are more frequently affected than women, in particular in their fourth decade [6].

Even though the rupture seemingly occurs as consequence of a traumatic insult on a nevertheless healthy tendon, in reality it is the end result of a single eccentric contraction on a tendon in which a tendinopathic process had been present, albeit not symptomatic [7]. The etiopathogenesis of AT rupture is still unknown, but histological evidence of failed healing response is relatively common. Both tendinopathic and ruptured tendons have a greater degree of histological evidence of tendinopathy compared with normal tendons, and the degree of degeneration in the ruptured group is statistically greater than in the tendinopathic group [7]. Corticosteroids, fluoroquinolone use, rheumatoid arthritis, and renal transplantation have been associated with AT rupture [6, 8]. More recent researches show that metabolic disease and endocrine disorders such as diabetes mellitus, hypothyroidism, hypercholesterolemia, and obesity could predispose to tendinopathies and tendon tears [9–12].

N. Maffulli, MD, PhD, FRCP, FRCS (✉)
Department of Musculoskeletal Disorders, School of Medicine and Surgery, University of Salerno, Salerno, Italy

Centre for Sports and Exercise Medicine, Mile End Hospital, Barts and the London School of Medicine and Dentistry, Mary University of London, 275 Bancroft Road, London E1 4DG, England, UK
e-mail: n.maffulli@qmul.ac.uk

A. Giai Via, MD • F. Oliva, MD, PhD
Department of Orthopaedic and Traumatology, School of Medicine, University of Rome "Tor Vergata", Viale Oxford 81, Rome 00133, Italy
e-mail: alessiogiaivia@hotmail.it; olivafrancesco@hotmail.com

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_10, © Springer International Publishing Switzerland 2016

10.3 Clinical and Diagnostic Examination

Acute AT rupture is usually a clinical diagnosis based on a careful history and detailed clinical examination. Patients often give a history of feeling a blow to the posterior aspect of the leg and may describe an audible snap followed by pain and inability to walk. A gap in the Achilles tendon is usually palpable. With increased time after the tear, the gap may be obliterated by edema, which makes palpation unreliable, while in the early stages, edema and bruising may not be apparent. Active plantar flexion of the foot is usually preserved given the action of the tibialis posterior, the long toe flexors, and the peroneus muscles. Numerous clinical tests have been described to aid in the diagnosis of Achilles tendon tears, and palpation, calf squeeze test, Matles test, O'Brien test, and Copeland test have been used [13]. All the tests described in the literature may be used to correctly diagnose a subcutaneous Achilles tendon tear with a high degree of certainty.

A retrospective study showed that if two or more of these tests are positive, the diagnosis of an AT tear is certain [13]. As the Copeland and the O'Brien tests may cause discomfort, the diagnosis of a subcutaneous tear of the Achilles tendon may be reliably made on the basis of the calf squeeze and Matles tests. The calf squeeze test, first described by Simmonds in 1957 [14] but often credited to Thompson, is performed with the patient prone and the ankles clear of the table. The examiner squeezes the fleshy part of the calf, causing deformation of the soleus and resulting in plantar flexion of the foot if the Achilles tendon is intact. The affected leg should be compared to the contralateral leg. The Matles test or knee flexion test is performed with the patient prone and the ankles clear of the table [15]. The patient is asked to actively flex the knee to 90°. During this movement, the foot on the affected side falls into neutral or dorsiflexion, and a rupture of the Achilles tendon can be diagnosed (Fig. 10.1).

Sometimes diagnostic imaging may be required to verify a clinical suspicion or for chronic injuries. Plain lateral radiographs may reveal an irregular configuration of the fat-filled triangular space anterior to the Achilles tendon and between the posterior aspect of the tibia and the superior aspect of the calcaneus. It is also helpful to exclude bone injuries in case of acute trauma. Ultrasound (US) and magnetic resonance imaging (MRI) are widely used, even if there is no clear evidence that they improve the rate of correct diagnosis. According to the AAOS guidelines for acute AT rupture, there is not enough evidence to recommend for or against the routine use of US and MRI to confirm the diagnosis [16]. A recent study showed that physical examination, including an abnormal calf squeeze test, a palpable defect, and decreased resting tension, is more sensitive in diagnosing an acute complete AT rupture than MRI (sensitivity 100 % vs 90.9 %) [17]. Moreover MRI is time consuming and expensive and can lead to a delay in treatment (mean time to surgery 5.6 days vs 12.4 day in MRI group). The authors concluded recommending careful evaluation and judicious use of advanced imaging as needed.

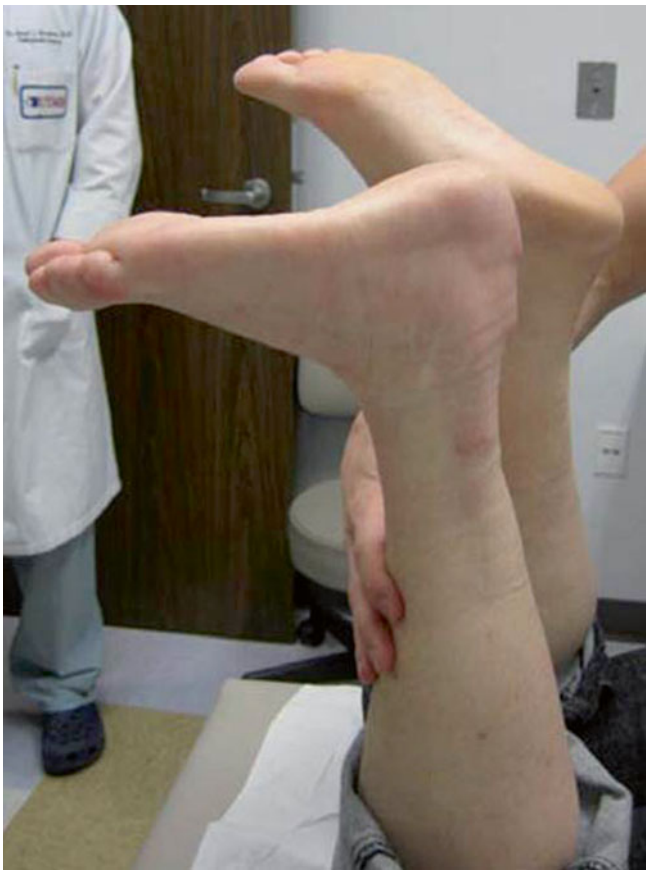


Fig. 10.1 The Matles test or the knee flexion test: the foot on the affected side falls into neutral or dorsiflexion

10.4 Treatment Strategy

The consensus for athletes is surgery [2], as it provides early functional treatment, less calf atrophy, and the best functional performance with a lower re-rupture rate. Open, percutaneous, or minimally invasive procedures have been successfully used. Open surgery provides good strength to the repair, low re-rupture rates, and reliably good endurance and power to the gastrocnemius-Achilles tendon complex. However, open surgical approaches resulted in high risk of infection and morbidity. Review articles and meta-analysis showed high costs and a 20-fold higher rate of complications in open procedures than conservative treatment [13]. Therefore, minimally invasive procedures have been successfully used to avoid these complications. Minimally invasive AT repair provides many advantages. Major advantages are less iatrogenic damage to normal tissues, lower postoperative pain, accurate opposition of the tendon ends

minimizing surgical incisions, and improved cosmetics. A recent systematic review reported a rate of superficial infections of 0.5 and 4.3 % after minimally invasive and open surgery, respectively [18]. Shorter hospitalization time and average time to return to working activities was also showed. Functional outcomes were not significantly different between minimally invasive and open surgery. Although sural nerve injury has been reported as a potential complication of this kind of surgery, new techniques have minimized the risk of sural nerve damage [18].

10.5 Percutaneous Achilles Tendon Repair: Surgical Technique

A 1 cm transverse incision is made over the defect using a size 11 blade. Four longitudinal stab incisions are made lateral and medial to the tendon 6 cm proximal to the palpable defect. Two further longitudinal incisions on either side of the tendon are made 4–6 cm distal to the palpable defect. Forceps are then used to mobilize the tendon from beneath the subcutaneous tissues. A 9 cm Mayo needle is threaded with two double loops of Number 1 Maxon, and this is passed transversely between the proximal stab incisions through the bulk of the tendon (Fig. 10.2). The bulk of the tendon is surprisingly superficial. The loose ends are held with a clip. In turn, each of the ends is then passed distally from just proximal to the transverse Maxon passage through the bulk of the tendon to pass out of the diagonally opposing stab incision. A subsequent diagonal pass is then made to the transverse incision over the ruptured tendon. To prevent entanglement, both ends of the Maxon are held in separate clips. This suture is then tested for security by pulling with both ends of the Maxon distally. Another double loop of Maxon is then passed between the distal stab incisions through the tendon (Fig. 10.3) and in turn through the tendon and out of the transverse incision starting distal to the transverse passage (Fig. 10.4). The ankle is held in full plantar flexion, and in turn the opposing ends of the Maxon thread are tied together with a double-throw knot, and then three further throws before being buried using the forceps (Fig. 10.5). A clip is used to hold the first throw of the lateral side to maintain the tension of the suture. We use 3-0 Vicryl suture to close the transverse incision and Steri-Strips close the stab incisions. A nonadherent dressing is applied. A full plaster cast is applied in the operating room with the ankle in physiologic equinus. The cast is split on both medial and lateral sides to allow for swelling. The patient is discharged on the same day of the operation.

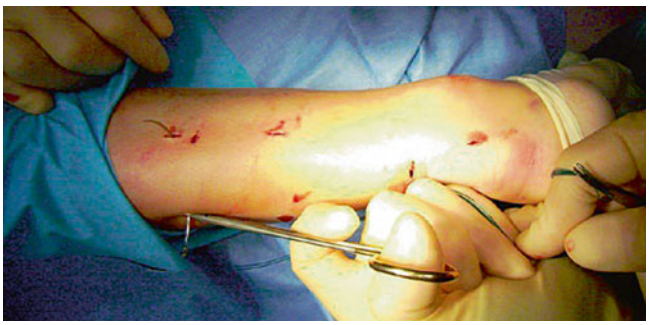


Fig. 10.2 A 9 cm Mayo is threaded with two double loops of Number 1 Maxon, and this is passed transversely between the proximal stab incisions through the bulk of the tendon

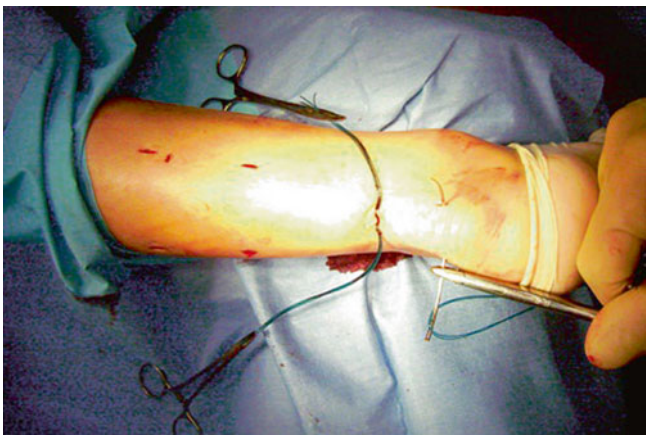


Fig. 10.3 Another double loop of Maxon is then passed between the distal stab incisions through the tendon

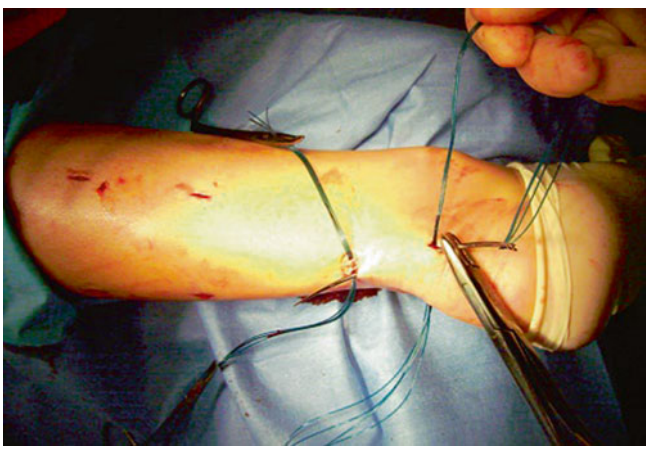


Fig. 10.4 The double loop of Maxon is passed in turn through the tendon and out of the transverse incision starting distal to the transverse passage

10.6 Rehabilitation and Return to Play

Following percutaneous repair, patients are encouraged to bear weight on the operated limb as soon as possible as tolerated. The cast is removed at 2 weeks postoperatively, and a boot with the ankle in a plantigrade position is used. Removal of the boot under supervision of a physiotherapist allows the ankle to be plantar flexed fully but not dorsiflexed. These

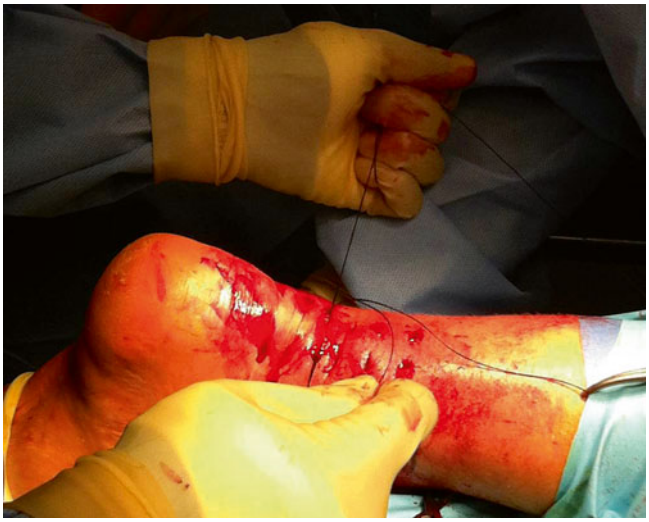


Fig. 10.5 The two tendon stumps are sutured together with the ankle in full plantar flexion

exercises are performed against manual resistance. At 6 weeks postoperatively, the boot is removed, and the patient referred to physiotherapy for active mobilization. At 12 weeks postoperatively, patients are assessed as to whether they were able to undertake more vigorous physiotherapy and are encouraged to gradually return to their normal activities. Progressive activities are incorporated as strength allows, with the aim to return to unrestricted activities by 6 months following surgery. Patients are reviewed at 3-month intervals and discharged at 9 or 12 months after the operation once they are able to perform at least five toe raises unaided on the operated leg and after they returned to their normal activities.

10.7 Discussion

AT ruptures are common in athletes. Surgical repair provides good results in young and active people. Open, percutaneous, or minimally invasive procedures have been successfully used. Open surgery provides good strength to the repair, low re-rupture rates, and reliably good endurance and power to the gastrocnemius-Achilles tendon complex. However, open surgical repair may result in high risk of infection and morbidity. Review articles and meta-analysis showed high costs and a 20-fold higher rate of complications in open procedures than conservative treatment [6]. Therefore, minimally invasive procedures have been successfully used to avoid these complications. Minimally invasive Achilles tendon repair provides many advantages. Major advantages are less iatrogenic damage to normal tissues, lower postoperative pain, accurate opposition of the tendon ends minimizing surgical incisions, shorter hospitalization time, lower rate of postsurgical infections, and improved cosmesis [18]. Excellent results have been reported in 17 elite athletes after percutaneous surgical repair of Achilles tendon ruptures [19]. All patients returned to high-level competition, with an average time to return to full sport participation of 4.8 months (range 3.2–6.5).

Table 10.1 Parameters used in assessing the time frame of patients undergoing Achilles tendon surgery return to activity (RTA)

Parameters predicting the ability to RTA	
Concentric strengthening	Ability to perform 5 sets of 25 single-legged heel raises
Muscle girth	Calf circumference: 5 mm or less difference measured 10 cm distal to the tibial tuberosity of the operative limb as compared with the nonoperative limb
Range of motion (ROM)	Ankle dorsiflexion and plantar flexion ROM within 5° of the nonoperative limb

Rehabilitation of the Achilles tendon is complex and often nonstandardized. Detailed postoperative physical therapy programs for the AT often vary. Return to activity (RTA) can be defined as the time in which patients initiate their desired activity or sport that was limiting them. Evidence-based study of physical therapy regimens with regard to the foot and ankle is very limited. Modalities that have been rigorously studied have shown little benefit, including ultrasound, massage, and injections [20, 21]. Both eccentric exercises and extracorporeal shockwave therapy (ESWT) have been studied with a wide range of results. When patients can return to sports reducing the risk of further injuries is a big question, in particular for athletes because physicians are often faced with the pressing requirements of the athlete himself, the coach, and the team. Recurrence of AT tendinopathy and reinjury risk has been reported to be higher after short recovery periods [22]. Saxena et al. reported that simple parameters such as single-legged concentric strength, range of motion, and muscle girth can predict the ability to RTA [21]. If patients meet all 3 of these criteria, they are allowed to return to sport (Table 10.1), and the mean time to RTA after AT surgical repair was 21.8 ± 4.0 weeks. Females were more likely to have a delay in RTA.

Restoring the normal structure and function of injured tendons is a great challenge, so several strategies have been proposed to enhance tendon healing. Recently research focused on regenerative therapies such as growth factors (GFs) and plasma-rich platelet (PRP), but this is still controversial. The use of PRPs has expanded to meet multiple medical problems where current treatment options were judged suboptimal. It is currently a common treatment for the tendon injuries because of the autologous source, safety profile, and minimal manipulation [23]. In vitro studies showed that the addition of PRP to human tenocytes resulted in cell proliferation, collagen deposition, well-ordered angiogenesis, and improved gene expression for matrix-degrading enzymes

and endogenous growth factors [24, 25]. More recently, two studies demonstrated that PRP induced in vitro tendon mesenchymal stem cell (T-MSC) differentiation into active tenocytes and that PRP has an anti-inflammatory function by suppressing the levels of prostaglandin (PGE) biosynthetic pathway components (COX-1, COX-2, and mPGES-1 expression) and PGE2 production [26]. These results have important clinical implications because high levels of PGE2 cause pain, decrease cell proliferation and collagen production, and induce degenerative changes in rabbit tendons [27]. The same authors also reported that even though PRP is able to induce the differentiation of T-MSCs into tenocytes under regular culture conditions, PRP injection in routine clinical practice may not be able to effectively reverse the degenerative conditions of late-stage tendinopathy [28]. Currently, many studies are published in literature with conflicting results. In fact, although a recent study suggests that vascular endothelial growth factor-111 (VEGF-111) could have a potential positive effect on the healing of AT lesions in rats [29], another animal study shows that a single injection of PRP did not influence tendon healing [30]. Well-conducted clinical studies do not report any substantial benefit using PRP, and its routine use is thus not recommended [31].

Conclusion

AT rupture is a serious injury not only in high-level athletes. The management should take into account the age, occupation, and level of sporting activity of the patient. Open surgery is frequently associated with higher risk of superficial skin breakdown and wound problems, which can be prevented by performing percutaneous repair. Percutaneous repair followed by early functional rehabilitation is becoming increasingly common and may be considered in athletes.

References

1. Viidik A (1962) Tensile strength properties of Achilles tendon systems in trained and untrained rabbits. Acta Orthop Scand 10:261–272
2. Longo UG, Petrillo S, Maffulli N et al (2013) Acute Achilles tendon rupture in athletes. Foot Ankle Clin 18:319–338
3. Nilsson-Helander K, Silbernagel KG, Thomee R et al (2010) Acute Achilles tendon rupture: a randomized, controlled study comparing surgical and nonsurgical treatments using validated outcome measures. Am J Sports Med 38:2186–2193
4. Willits K, Amendola A, Bryant D et al (2010) Operative versus nonoperative treatment of acute Achilles tendon ruptures: a multicenter randomized trial using accelerated functional rehabilitation. J Bone Joint Surg Am 92:2767–2775
5. Guillo S, Del Buono A, Dias M et al (2013) Percutaneous repair of acute ruptures of the tendo Achillis. Surgeon 11:14–19
6. Maffulli N (1999) Rupture of the Achilles tendon. J Bone Joint Surg Am 81A:1019–1036
7. Tallon C, Maffulli N, Ewen SW (2001) Ruptured Achilles tendons are significantly more degenerated than tendinopathic tendons. Med Sci Sports Exerc 33:1983–1990
8. Stephenson AL, Wu W, Cortes D, Rochon PA. Tendon Injury and Fluoroquinolone Use: A Systematic Review. Drug Saf. 2013. [Epub ahead of print]
9. Bourne JW, Lippell JM, Torzilli PA (2014) Glycation cross-linking induced mechanical-enzymatic cleavage of microscale tendon fibers. Matrix Biol 34:179–184
10. Oliva F, Berardi AC, Misiti S, Maffulli N (2013) Thyroid hormones and tendon: current views and future perspectives. Concise review. Muscles Ligaments Tendons J 3:201–203
11. Beason DP, Abboud JA, Kuntz AF, Bassora R, Soslowsky LJ (2011) Cumulative effects of hypercholesterolemia on tendon biomechanics in a mouse model. J Orthop Res 29:380–383
12. Abate M, Oliva F, Schiavone C, Salini V (2012) Achilles tendinopathy in amateur runners: role of adiposity (Tendinopathies and obesity). Muscles Ligaments Tendons J 2:44–48
13. Maffulli N (1998) The clinical diagnosis of subcutaneous tear of the Achilles tendon. A prospective study in 174 patients. Am J Sports Med 26(2):266–70
14. Simmonds FA (1957) The diagnosis of the ruptured Achilles tendon. Practitioner 179:56–58
15. Matles AL (1975) Rupture of the tendon Achilles. Another diagnostic sign. Bull Hosp Joint Dis 36:48–51
16. Kou J (2010) AAOS clinical practice guideline: acute Achilles tendon rupture. J Am Acad Orthop Surg 18:511–513
17. Garras DN, Raikin SM, Bhat SB, Taweel N, Karanjia H (2012) MRI is unnecessary for diagnosing acute Achilles tendon ruptures: clinical diagnostic criteria. Clin Orthop Relat Res 470:2268–2273
18. Del Buono A, Volpin A, Maffulli N (2014) Minimally invasive versus open surgery for acute Achilles tendon rupture: a systematic review. Br Med Bull 109:45–54
19. Maffulli N, Longo UG, Maffulli GD et al (2011) Achilles tendon ruptures in elite athletes. Foot Ankle Int 32:9–15
20. Park D, Chou L (2006) Stretching for prevention of Achilles tendon injuries: a review of the literature. Foot Ankle Int 27:1086–1095
21. Saxena A, Ewen B, Maffulli N (2011) Rehabilitation of the operated Achilles tendon: parameters for predicting return to activity. J Foot Ankle Surg 50(1):37–40
22. Gajhede-Knudsen M, Ekstrand J, Magnusson H, Maffulli N (2013) Recurrence of Achilles tendon injuries in elite male football players is more common after early return to play: an 11-year follow-up of the UEFA Champions League injury study. Br J Sports Med 47:763–768
23. Andia I (2014) Platelet rich plasma therapies: a great potential to be harnessed. Muscles Ligaments Tendons J 4:1–2
24. Oliva F, Via AG, Maffulli N (2011) Role of growth factors in rotator cuff healing. Sports Med Arthrosc 19:218–226
25. Yuan T, Zhang CQ, Wang JH (2013) Augmenting tendon and ligament repair with platelet-rich plasma (PRP). Muscles Ligaments Tendons J 3:139–149
26. Wang JHC (2014) Can PRP, effectively treat injured tendons? Muscles, Ligaments and Tendons J 4:35–37
27. Khan MH, Li Z, Wang JH (2005) Repeated exposure of tendon to prostaglandin-E2 leads to localized tendon degeneration. Clin J Sport Med 15:27–33
28. Zhang J, Wang JHC (2014) PRP treatment effects on degenerative tendinopathy – an in vitro model study. Muscles, Ligament s and Tendons J 4:10–17
29. Kaux JF, Janssen L, Drion P et al (2014) Vascular Endothelial Growth Factor-111 (VEGF-111) and tendon healing: preliminary results in a rat model of tendon injury. Muscles Ligaments Tendons J 4:24–28
30. Parafioriti A, Armiraglio E, Del Bianco S, Tibalt E, Oliva F, Berardi AC (2011) Single injection of platelet-rich plasma in a rat Achilles tendon tear model. Muscles Ligaments Tend ons J 1:41–47
31. Maffulli N (2013) Autologous blood products in musculoskeletal medicine. BMJ 346:f2979

Patellar and Quadriceps Tendinopathy

11

Andrea Ferretti and Antonio Pasquale Vadala

11.1 Introduction

Quadriceps and patellar tendinopathies are known as "jumper's knee," similarly following a tradition of reference to the etiology of a pathology, such as "tennis elbow" or "thrower's shoulder."

Even though patellar tendinopathy had already been described in Italy [1], the term "jumper's knee" was due to a study published in 1973 by Blazina et al. [2], one of the coworkers of the famous sports physician Frank Jobe, Chief of the Sports Medicine Center in Inglewood (California, USA), located near the sports center where the prestigious Los Angeles Lakers team now plays. It was certainly to the basketball players that Blazina was referring when he wisely described the painful syndrome "jumper's knee."

Successively, many other studies have focused on this so far unclear pathology which deserves to be known by sports physicians who, considering the frequency of this pathology in some types of players, will have to face this pathology during their career.

In this chapter, we will discuss the different aspects of patellar and quadriceps pathologies according to the most recent experiences and the newest types of treatments.

A. Ferretti (✉)
Orthopaedic and Traumatology Department, Faculty of Medicine, "Kirk Kilgour" Sports Traumatology Center, S. Andrea Hospital, University "La Sapienza", Grottarossa St. 1037, Rome 00189, Italy

Sports Traumatology Center, S. Andrea Hospital, University of Rome "La Sapienza", Via Grottarossa 1035, Rome 00100, Italy
e-mail: aferretti51@tiscali.it

A.P. Vadala
Sports Traumatology Center, S. Andrea Hospital, University of Rome "La Sapienza", Via Grottarossa 1035, Rome 00100, Italy
e-mail: anto.vado@libero.it

11.2 Epidemiology and Predisposing Factors

As cited above, patellar and quadriceps tendinopathies are very common among athletes involved in jumping activities or whose activity is based on the use of the lower limb's extensor apparatus (such as weight lifters). It has been estimated that up to 40 % of volleyball players suffer or have suffered from this pathology during their career [3].

In the sport of soccer, jumper's knee syndrome was unknown until the 1980s, when coaches started putting more attention on basic training with weight-lifting exercises, jumping (plyometric), eccentric strengthening, and so forth. Nowadays patellar tendinopathy affects about 2.5 % of soccer athletes, data which is still lower compared to athletes of other sports who comprise the highest percentage of incidence (volleyball, basketball) [4, 5].

Predisposing factors to the onset of such a pathology are classified as "intrinsic" and "extrinsic"; intrinsic factors mean those related to the athlete, and extrinsic are those due to environmental stresses.

Besides sex incidence, which is higher in male patients, many epidemiologic studies have shown no correlation between morphologic characteristics of patients and jumper's knee syndrome. In particular, it has never been shown that varus or valgus malalignment of the knee, a malalignment of the extensor apparatus, a patellar hypermobility, or a high or low patella syndrome might be predisposing factors, and aside from the knee area, nor have valgus hindfoot deformity, pronated foot valgus, cavus foot, or even others. Researchers' interest therefore focused more on peculiar characteristics of the patellar tendon tissue and on its collagen composition.

However, the results about extrinsic factors were different. The first data, which is also the most obvious, concerns the correlation between the number of training sessions and matches per week and the incidence of jumper's knee

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_11,

syndrome. This strict positive correlation clearly confirms the importance of overuse in the onset of such a type of tendinopathy. Conversely less significant was the type of training (if carried out with weights, therabands, or jumps) [6].

Training fields have been widely studied with the result that the harder the field, the higher the incidence. Regarding soccer games however, there do not seem to be significant differences between players who usually train on natural fields and those who train on artificial fields [7].

Other factors predisposing patellar tendinopathy, which might be considered both intrinsic and extrinsic, regard jumping capability with a higher incidence among athletes with higher explosive strength [8].

11.3 Symptomatology and Classification

The main symptom of jumper's knee is pain, with varying intensity, but always, localized in one of the typical places which represent the fulcrum of the extensor apparatus. The most frequent localization is the origin of the patellar tendon at the lower site of the patella (70 % of the cases), followed by the insertion of the quadriceps tendon at the superior site of the patella (20 %) and by the insertion of the patellar tendon on the anterior tibial tuberosity (10 %). The most recent classifications are based on the intensity of the pain with jumper's knee which, compared to the first classification proposed by Blazina, focus more attention on evaluating the effect of the pain on sports performance.

11.3.1 Jumper's Knee Classification According to Symptoms

Ferretti and coworkers [9]
STADIUM 0 – No pain
STADIUM I – Rare pain with no sports restriction
STADIUM II – Moderate pain during sports activity with no restriction on sports performance (normal performance)
STADIUM III – Pain with slight qualitative and quantitative restriction on performance (reduced number of training sessions or minor intensity)
STADIUM IV – Pain with severe restriction of sports performance
STADIUM V – Pain during daily activity; sports activity impossible

As we can see, the classification above described does not take into account rupture of the patellar tendon (catastrophic jumper's knee) [10] which cannot be considered the evolution of the insertional patellar tendinopathy but an acute event as a consequence of a chronic tendinous degeneration, which is totally different from an anatomo-pathology point of view. Indeed the discrepancy between pain (severe) and tendinous damage (moderate) seen in jumper's knee forces the athlete to suspend the activity much earlier before the pathology leads to rupture; the exact opposite happens with tendinosic degeneration where the tendinous damage is severe and progressive with a painful symptomatology usually moderate or even absent. By an objective point of view, local digital pain represents the only important factor. Locally, in some cases, we can observe a mild swelling of the soft tissue but never an articular effusion. Knee articular semeiotic, as well as the one concerning the extensor apparatus, is negative.

11.4 Radiological Findings

Radiological evaluation consists of X-rays, ultrasound evaluations, and magnetic resonance imaging. Standard X-rays might show insertional calcifications (spurs) which show the exact localization of the pathology. Ultrasound may show a thickening of the patellar tendon close to the insertion and even a loss of the normal fibrillar pattern. Recently ultrasound has also been used to study patellar tendon vascularization (eco color Doppler): this methodology might show cases with hypervascularization as well as cases of normal or reduced vascularization, even though these patterns are not adequately understood [11]. Magnetic resonance allows better definition of the tendon morphology which often shows a tendon modification at the insertional site. However, MRI rarely ever changes therapeutic protocol.

11.5 Anatomo-pathology

With regard to anatomo-pathologic classification of overuse tendinopathy as proposed by Perugia et al. [12], jumper's knee might be included in the insertional tendinopathies.

On the other hand, an anatomo-pathologic pattern of subcutaneous ruptures usually represents the consequence of a chronic degenerative process (tendinosis) which is often totally asymptomatic and which usually occurs, abruptly, with an acute rupture. In these cases tendinous tissue shows wide areas of degeneration with lack of the normal fibrillar pattern and an important reduction in the cellular component.

With regard to jumper's knee, peculiar data which surgeons detected in the 1970s was the discrepancy between painful symptomatology, which was often very restricting in daily activities, and the lack of tendinous damage. Research of the "nidus" of tendon degeneration, as proposed by Basset, was often useless in a surgical approach, with the consequence that many surgical procedures were performed with the aim of treating different pathologies (meniscus, patellar,

chondral damages). In a previous histological study, we showed the presence of microscopic insertional alteration consisting of a disruption of the *blue line* along with a microcystic area full of granulation tissue, suggesting microinsertional detachments with an attempt of reparation, which nowadays represents one of the most plausible pathogenetic hypotheses of this pathology [13]. Obviously, spots of degenerative tendinous tissue can come together even though their role in the painful symptomatology is not yet clear.

11.6 Treatment

Patellar tendinopathy treatment is basically conservative and consists of medical and physical therapy.

Medical therapy is based on the use of NSAIDs. Their use is not justified though since at the level of the tendinous degeneration, a phlogistic process has never been shown. For this reason the success of such a type of therapy, widespread even though there is a lack of scientific studies supporting their use, is perhaps dependent more on their analgesic effect than their antiphlogistic effect.

What is certainly efficient is the use of local infiltration of corticosteroids, even though there is a big debate about their use. While it was once considered the gold standard, it was progressively abandoned and afterward criminalized because of its potential effect on the tendinous tissue leading to rupture [14]. Actually in specific and well-selected cases and especially during some period of the athletic season and of the career of an athlete, corticosteroid infiltration may be a valid therapeutic option for the sports physician, in particular in those cases in which radiological exams do not show a significant degenerative process. Beneficial effects of corticosteroid injections are usually immediate even though temporary; this pain-free period should be used in adjunct to other physical therapies for better long-time follow-up results. Recently corticosteroid injections have been duplicated in more diluted preparations with satisfactory preliminary results even with restrictions similar to the traditional steroidal infiltration [15].

Other infiltrative drugs have recently been proposed but major literature about them is still missing [16].

A promising but not yet adequately understood type of treatment for tendinopathies is represented by the infiltration of autologous PRP. It consists of the use of growth factors present within platelets, potentially able to facilitate the cellular differentiation toward a fibroblastic line with the aim of promoting tissue healing [17]. The methodology for using PRP was introduced about 10 years ago, and it reached a high popularity after its use on professional athletes with very satisfactory results documented in scientific studies; this apparent success has created a big economic interest around its use with sometimes criticizable implications. However it is a matter of fact that its use has a solid biological base, it seems without related risks, and even without picturing this type of treatment as a miraculous option, it may be a valid alternative for sports physicians. In our experience, the use of PRP for patellar tendinopathy consisting of three injections, one each week, provided better medium- and long-term results compared to the use of ESWT [18]. Besides radial and focalized ESWT [19], many other types of conservative treatments (ultrasound, laser, TENS, magnetotherapy, TECAR) have been used for the treatment of patellar tendinopathy, but always with uncertain and sometimes unsatisfactory results.

Kinesitherapy, whose main aim would consist of improving the mechanical properties of the tendon through selective training, nowadays represents the gold standard in the treatment of jumper's knee. It is based on the practice of different types of muscular exercises: isotonic, isometric, isokinetic, and eccentric; in particular eccentric exercises are recommended in any phase of the pathology, both alone and with all other types of exercises [20]. The knowledge of kinesitherapy as a valid option for the cure and prevention of jumper's knee is so widespread among athletes, volleyball players in particular, that they usually practice it themselves as soon as they feel the onset of the symptomatology.

The use of elastic bandages or braces at the level of the patellar tendon has been practiced by many athletes with the aim of reducing painful symptomatology during sports practice. Despite the fact that their use has been documented to reduce insertional stresses, their real efficacy has never been totally demonstrated [21].

Surgical treatment is indicated in a very small number of cases (less than 10 %) [22], usually in the advanced phases of the pathology, when all other conservative options have failed, and at the end of the agonistic season. Surgery has the aim of promoting a valid healing at the level of tendon insertion and consists of different steps: deepest tendon fiber disinsertion, removal of the damaged tissue (*Basset's nidus*) when present, plastic of the lowest part of the patella or apicoectomy, patella drilling, or tendinous scarifications [9].

Postoperative protocol consists of a short period of immobilization (2–4 weeks) followed by a progressive recovery of range of motion and then of muscular trophism and usually requires about 4–6 months before the return to specific training and professional matches. Results of surgical procedures, even though usually satisfactory, are not always brilliant, especially in athletes involved in high performance jumps with percentages of total lack of pain not higher than 65 % and with a little higher percentage in those athletes affected by quadriceps rather than patellar insertional pathology. The above mentioned surgical steps may also be carried out arthroscopically but with more difficulties, with a longer learning curve, and, most of all, with no advantage in terms of final results and recovery time [23, 24].

11.7 Patellar Tendon Rupture

Patellar tendon rupture will be presented here in a different chart because it is usually a consequence of a different tendinous pathology which involves the middle part of the tendon and not the insertional end. Patellar tendon rupture, which usually occurs abruptly, involves a dramatic clinical feeling of a tear in the anterior part of the knee, immediate fall, and complete lack of capability of weight-bearing, walking, or actively extending the knee followed by severe swelling, hemarthrosis, and wide ecchymosis; it is hardly ever preceded by the typical jumper's knee symptoms. It is commonly the final event of a progressive degenerative process involving the tendon itself which leads to a progressive weakening of the tendon until the final rupture, similarly to what happens with the Achilles tendon. The pattern of severe tendinous damage detected in the surgery room is significantly different than the one observed in cases of insertional tendinopathy where the tendon itself looks macroscopically normal, despite an intense clinical symptomatology with severe restrictions. Diagnosis of patellar tendon rupture is easy and is based on the anamnesis and the evidence of a gap below the apex of the patella which also appears higher than the contralateral knee. Quadriceps tendon rupture has a similar clinical onset even though the gap in these cases is localized above the patella. X-ray exam shows a high patella (or low in cases of quadriceps rupture), while ultrasound and MRI exams may give some more details in regard to the tendinous pathology even though they do not change the type of therapy needed which is surgery. Surgical procedure consists of suturing the tendon and in some cases reinforcement with transosseous sutures. As is well known, since tendon biology is prosperous and has a high capability of healing, it is hardly ever necessary to use autologous, homologous, or synthetic tissue reinforcement; on the contrary, we recommend to only and always use reabsorbable suture. Postoperative follow-up consists of knee immobilization in full extension for 5 weeks, with the first two with no weight-bearing, followed by a physical rehabilitation aimed at recovering range of motion first and strengthening the quadriceps muscle later. Return to sports is not allowed before 6 months. Postoperative rehabilitation is usually easier and faster in quadriceps suture. Not common but even more dramatic are the cases of bilateral simultaneous rupture of the tendons, the so-called catastrophic jumper's knee, which obviously requires the same type of surgical treatment but with a less favorable prognosis in regard to return to sports activity.

References

1. Maurizio E (1963) la tendinite rotulea nei giocatori di pallavolo. Arch Soc Tosco Umbra Chir 24:443–452
2. Blazina ME, Kerlan RK, Jobe FW, Carter VS, Carlson GJ (1973) Jumper's knee. Orthop Clin North Am 4(3):665–678
3. Ferretti A (1986) Epidemiology of jumper's knee. Sports Med 3(4): 289–295
4. Zwerver J, Bredeweg SW, van den Akker-Scheek I (2011) Prevalence of Jumper's knee among nonelite athletes from different sports: a cross-sectional survey. Am J Sports Med 39(9):1984–1988. doi:10.1177/0363546511413370. Epub 2011 Jul 7
5. Lian OB, Engebretsen L, Bahr R (2005) Prevalence of jumper's knee among elite athletes from different sports: a cross-sectional study. Am J Sports Med 33(4):561–567. Epub 2005 Feb 8
6. Ferretti AI et al (1984) Jumper's Knee: an epidemiological study in volleyball players. Phys Sportsmed 12:97–103
7. Hägglund M, Zwerver J, Ekstrand J (2011) Epidemiology of patellar tendinopathy in elite male soccer players. Am J Sports Med 39(9):1906–1911
8. Visnes H, Aandahl H, Bahr R (2013) Jumping ability and change of jumping ability as risk factors for developing jumper's knee. Br J Sports Med 47(9):e2. doi:10.1136/bjsports-2013-092459.22
9. Ferretti A, Conteduca F, Camerucci E, Morelli F (2002) Patellar tendinosis: a follow-up study of surgical treatment. J Bone Joint Surg Am 84-A(12):2179–2185
10. Tarsney FF (1981) Catastrophic jumper's knee. A case report. Am J Sports Med 9(1):60–61
11. Hoksrud A, Ohberg L, Alfredson H, Bahr R (2008) Color Doppler ultrasound findings in patellar tendinopathy (jumper's knee). Am J Sports Med 36(9):1813–1820
12. Perugia L, Ippolito E, Postacchini F (1981) I tendini. Masson Ed, Milan
13. Ferretti A, Ippolito E, Mariani P, Puddu G (1983) Jumper's knee. Am J Sports Med 11(2):58–62
14. Meissner A, Tiedtke R (1985) Tendon rupture of the extensor muscles of the knee. Aktuelle Traumatol 15(4):170–174
15. Crisp T, Khan F, Padhiar N, Morrissey D, King J, Jalan R, Maffulli N, Frcr OC (2008) High volume ultrasound guided injections at the interface between the patellar tendon and Hoffa's body are effective in chronic patellar tendinopathy: a pilot study. Disabil Rehabil 30(20–22):1625–1634
16. van Ark M, Zwerver J, van den Akker-Scheek I (2011) Injection treatments for patellar tendinopathy. Br J Sports Med 45(13):1068–1076
17. Ferretti A (2011) PRP treatment in football players. UEFA Direct 107(4):8–13
18. Vetrano M, Castorina A, Vulpiani MC, Baldini R, Pavan A, Ferretti A (2013) Platelet-rich plasma versus focused shock waves in the treatment of jumper's knee in athletes. Am J Sports Med 41(4):795–803. doi:10.1177/0363546513475345. Epub 2013 Feb 13
19. Furia JP, Rompe JD, Cacchio A, Del Buono A, Maffulli N (2013) A single application of low-energy radial extracorporeal shock wave therapy is effective for the management of chronic patellar tendinopathy. Knee Surg Sports Traumatol Arthrosc 21(2):346–350. doi:10.1007/s00167-012-2057-8
20. Visnes H, Bahr R (2007) The evolution of eccentric training as treatment for patellar tendinopathy (jumper's knee): a critical review of exercise programmes. Br J Sports Med 41(4):217–223
21. Lavagnino M, Arnoczky SP, Dodds J, Elvin N (2011) Infrapatellar straps decrease patellar tendon strain at the site of the jumper's knee lesion: a computational analysis based on radiographic measurements. Sports Health 3(3):296–302
22. Ferretti A, Puddu G, Mariani PP, Neri M (1985) The natural history of jumper's knee. Patellar or quadriceps tendonitis. Int Orthop 8(4):239–242
23. Pascarella A, Alam M, Pascarella F, Latte C, Di Salvatore MG, Maffulli N (2011) Arthroscopic management of chronic patellar tendinopathy. Am J Sports Med 39(9):1975–1983
24. Marcheggiani Muccioli GM, Zaffagnini S, Tsapralis K, Alessandrini E, Bonanzinga T, Grassi A, Bragonzoni L, Della Villa S, Marcacci M (2013) Open versus arthroscopic surgical treatment of chronic proximal patellar tendinopathy. A systematic review. Knee Surg Sports Traumatol Arthrosc 21(2):351–357. doi:10.1007/s00167-012-2100-9. Epub 2012 Jun 20

Patellar Tendon Rupture

12

Federica Rosso, Umberto Cottino,
Davide Edoardo Bonasia, Matteo Bruzzone,
Federico Dettoni, and Roberto Rossi

12.1 Introduction

Extensor apparatus injuries have an incidence of 0.5–6 %, comprehensive of both patellar tendon and quadriceps tendon lesions. These are rare but serious injuries because the extensor mechanism is essential for normal human gait. The patellar tendon connects the patella to the anterior tibial tuberosity, and it is part of this structure. Its rupture is the third in frequency after patellar fracture and quadriceps tendon rupture, and it is a rare injury. However, patellar tendon injuries can lead to great disability that may last for several months, even if the treatment is correct [1].

Patellar tendon ruptures are more frequently caused by direct or indirect trauma in young people [2]. Besides they can occur because of overuse trauma or microtraumatism. Both tendon healing and rupture are closely dependent on tendon anatomy and histological characteristics. The cellular population is constituted by tenoblasts at 90–95 %; the remaining 5–10 % consists of chondrocytes (at bone attachment), synovial cells, and vascular cells [3]. Metabolic activity in the patellar tendon is very low, in fact it has been estimated to be 7.5 times lower than in skeletal muscles [4]. This low metabolic activity and the tendon vascularization are perfect to grant a greater resistance to continuous loads and protect from avascular necrosis [1]. In the patellar tendon, the vascularization depends on different sources: an intrinsic system at the myotendinous and osteotendinous junctions and an extrinsic system through the paratenon or synovial sheath [5, 6].

12.2 Etiology

In literature, there are few data regarding patellar tendon ruptures in active people, because they are more frequent after a total prosthesis. There are a lot of case reports with really small numbers, and the focus is placed on different repairing techniques despite of etiology or diagnostic process. The real challenge is how to manage a rare but complex lesion with an unclear etiology but potential catastrophic consequences. Typically, patella tendon tears occur in active young people (under 40 years old), and it is usually caused by an indirect trauma to the knee: a sudden contraction of the quadriceps with the knee in slight flexion (sudden impulsion, sprint, avoiding a fall, etc.) [7]. Traumatic lesions of the patellar tendon are also observed during rapid dynamic loading conditions occurring in many sports situations; on the other hand, injuries in maximum tensile stress during static test conditions are less common. This more common mechanism of injury was firstly described in 1977 by Zernicke et al.: they reported on the case of a 29-year-old weight lifter of the 1975 US National Weightlifting Championship; he broke his patellar tendon lifting 142.5 kg (1.73 times his body weight) in a fast movement [8]. Some authors described with biomechanical test the minimum force required to cause a patellar tendon rupture, and it turns out equal to 17.5 times the body weight [8]. This is an impressive amount of force considering that climbing stairs generates 3.2 times body weight only [9].

F. Rosso (✉) • U. Cottino • M. Bruzzone • F. Dettoni • R. Rossi
Department of Orthopedics and Traumatology,
AO Mauriziano Umberto I, Largo Turati 62, Torino 10128, Italy
e-mail: federica.rosso@yahoo.it; umberto.cottino@gmail.com;
matteobruzzone@yahoo.it; federicodettoni@tin.it;
rossir@fastwebnet.it

D.E. Bonasia
Department of Orthopedics and Traumatology,
AO Città della Salute e della Scienza,
Via Zuretti 29, Torino 10126, Italy
e-mail: davidebonasia@virgilio.it

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_12,

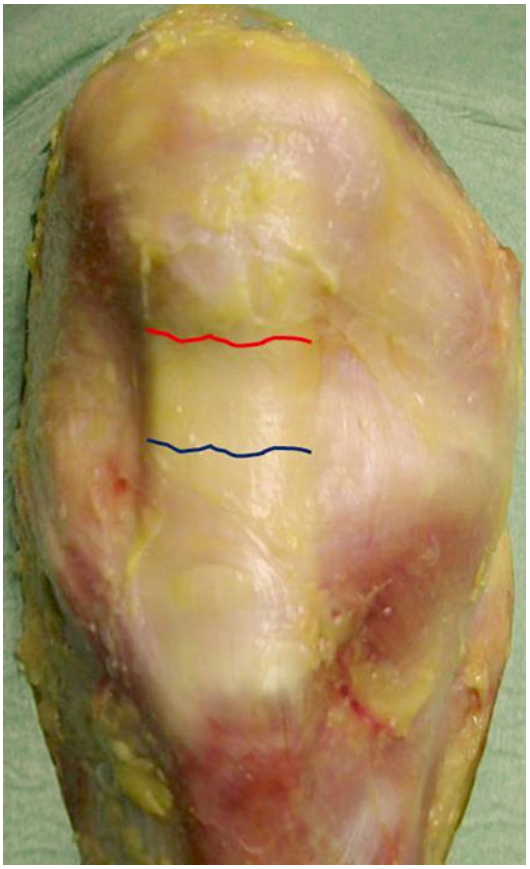


Fig. 12.1 Most common types of patellar tendon lesion: injury at the osteotendinous insertion (*red line*) and mid-substance injury (*blue line*)

Strain forces on the patellar tendon are greater at the osteotendinous insertion than in the mid-substance: for this reason, mid-substance lesions are much more rare than avulsions in normal tendons. Figure 12.1 shows the most common types of patellar tendon lesion. Because of the great forces needed to break a healthy tendon, in the international literature, it is commonly agreed that ruptures usually involve a degenerated tendon, overall if a mid-substance lesion occurs [10]. Tendon quality variations correlated to increased risk of rupture are to be referred to three different patterns of degeneration: mucoid, tendolipomatosis, and calcific [11]. Besides, different authors described other health conditions predisposing for patellar tendon rupture as listed below:

- Corticosteroids local injections [12]
- Diabetes [13, 14]
- Microtrauma [15]
- Rheumatic diseases [16–18]
- Metabolic disorders [19, 20]
- Iatrogenic lesions (ACL reconstruction) [21]
- Fluoroquinolones systemic use [22, 23]

12.3 Clinical and Diagnostic Examination

The first step in the diagnosis of a patellar tendon rupture is a good anamnesis. Typically, the patient refers an unexpected pain and subsequent knee impotence that can be sometimes associated to a fall, during a fast movement. The surgeon should ask for previous episodes of pain, in order to find a history of patellar tendinosis. Besides the attention should focus on other comorbidities or risk factors for patellar tendon ruptures (diabetes, rheumatoid arthritis, corticosteroid injection, etc.). At clinical inspection, the knee is usually swollen and, frequently, the patella cannot be recognized because of the edema. Palpating the knee, it is possible to evidence a defect in the patellar tendon and a patella alta, especially in complete breakings. Intraarticular hematoma is usually considerable and can be evacuated in emergency. The limit of the clinical inspection is the patients' discomfort and pain during knee examination. In cases of partial patellar tendon ruptures, active extension may be possible with pain, but, in cases of complete tears, the surgeon should search for an extension leg sign. Besides, both passive and active and under opposition forces, extension strength should be evaluated.

In an emergency, it is important to exclude first a bone fracture and to analyze anatomical landmarks of the knee. A plain radiograph in two projections (anteroposterior and latero-lateral) has always to be done. A patellar avulsion, a patella alta or a tibial tuberosity fracture can be suggestive for extensor mechanism rupture. When there is suspicion of a patellar tendon rupture, it can be useful to perform an ultrasound scan (US) that can show a hypoechoic area within the proximal patellar tendon. The advantage of US is the possibility of a dynamic study of the lesion while flexing and extending the knee. The limitations of this diagnostic test are the subjectivity rendered by the physician performing the exam and the lack of reproducibility.

The gold standard for tendon rupture still remains MRI but it is considered to be more expensive and less available [24]. MRI sensibility is higher and has to be performed when the diagnosis is not fully clear; this happens more frequently in obese and very muscular patients. CT scan can be useful when suspecting a patellar avulsion to better visualize the amount of bone attached to the tendon, but it is not useful if a mid-substance lesion occurs.

In conclusion, ultrasonography may not be a reliable method in establishing the diagnosis of acute injuries to the extensor mechanism of the knee, particularly the quadriceps tendon ruptures in the obese and the very muscular patients.

12.4 Treatment Strategy

12.4.1 Acute Ruptures

Incomplete ruptures of the patellar tendon with intact function of the extensor apparatus may be treated nonoperatively. The period of immobilization in full extension has to be 2–3 weeks, followed by progressive range of motion with active flexion and extension for 4 weeks; strengthening has to be started after 6 weeks [11]. If the incomplete lesion is associated to functional deficits, or in cases of complete patellar tendon rupture, the surgical treatment is indicated regardless of age and physical activity. It should be performed as soon as possible: acute repair has the best chance to achieve a tension-free tendon apposition [7]. When treating acute patellar tendon injuries, isolated suture repair is generally not recommended because of the high rate of re-rupture. Most of lesions occur closed to the osteotendinous junction along the inferior pole of the patella: this localization facilitates transosseous sutures [25–27]. The surgical approach normally suggested for a patellar tendon repair is the anterior one with a midline incision. After a careful dissection, the peritenon is longitudinally split, the retinacular tears are dissected out, and the tendon ends are debrided. Then, the inferior pole of the patella, the most common site of injury, is decorticated with a curette or a burr to improve bleeding. This is made in order to improve the healing process. A pair of continuous, locking heavy (No. 2 or 5) nonabsorbable sutures is then placed in the tendon using a Krackow or similar suture technique. Three parallel bone tunnels are drilled with a 2.5 mm drill from distal to proximal in the patella; a suture passer is used to pass the free suture ends in the bone tunnels, with the central one containing two sutures.

Some authors proposed to create the tunnels using an ACL tibial guide [28]. The suture ends are temporarily fixed to check patellar tracking and to avoid a patella infera. Once the correct position and tracking of the patella have been checked, the strands are tensioned with the knee fully extended to approximate the tendon to the inferior pole of the patella. The retinacula are now closed with an absorbable suture, and the knee is flexed at 90° to test the repair [11]. As well as for quadriceps tendon ruptures, suture anchors can be used for patellar tendon acute repair [29]. Some authors described a technique using three suture anchors incorporated into a six-stranded Krackow technique. Some authors concluded that this technique could result into a low-profile construct, with better recreation of the tendon's insertion into the inferior pole of the patella. This minimizes the possibilities of loosening through bone tunnels and abnormal stress forces [30]. Other authors demonstrated that with suture anchors, compared to trans-patellar sutures, there were less gap formations after cyclic loading [31, 32]. If the rupture occurs in the mid-substance of the tendon and there is enough proximal and distal substance, it can be repaired with continuous locking stitches; a Krackow or similar repair can be used [33]. In these cases, bone tunnels can be used in the patella to hold the proximal fragment [9]. Alternatively, a neutralization nonabsorbable wire or heavy sutures can be placed transversely through the patella and through another bone tunnel in the proximal tibia; this technique have been used to provide additional stability to the primary repair [33, 34]. When the primary repair is tenuous, it can be augmented using different surgical techniques. McLaughlin et al. described a technique of primary repair augmented with a tension wire anchored to the patella [35]. Kasten et al. describe their results comparing an augmentation with a metal wire or a strenuous PDS one, reporting no differences in terms of clinical results [36]. Other authors described an augmentation using semitendinosus autograft, nonabsorbable vascular graft, or nonabsorbable tape [37–40]. The augmentation using semitendinosus or gracilis autograft is one of the most commonly described in literature. The tendon is isolated and stripped, maintaining its tibial insertion. A tibial tunnel is normally drilled from medial to lateral to the anterior tibial tuberosity, and the tendon is passed through it. A second tunnel is then drilled in the patella, and the tendon is passed through it from lateral to medial. At this point, the end of the tendon is sutured back to its tibial insertion with nonabsorbable stitches [41]. Figure 12.2 shows some steps of this technique. However, the need for an augmentation in primary patellar tendon repair is still debated in literature. Some authors demonstrated better biomechanical resistance in augmented repair; other clinical studies showed similar results in patients with or without augmentation during the primary repair [42–44].

12.4.2 Chronic Ruptures

In the chronic setting, primary repair is difficult because of tendon retraction, tendon quality, and adhesion formation [11]. Some authors described a primary repair with fascia lata or hamstrings augmentation; sometimes, reconstruction is the only surgical option [45, 46]. In chronic patellar tendon rupture or re-rupture, the hamstrings can be used with techniques similar to the one described for augmentation. The tendons are normally left inserted, the tibial tunnel is then drilled, and the tendons are passed all around the patella in a circular or figure-of-eight fashion and then sutured back to their insertion [47–50]. Some authors also described a percutaneous technique with good clinical results, but in a small

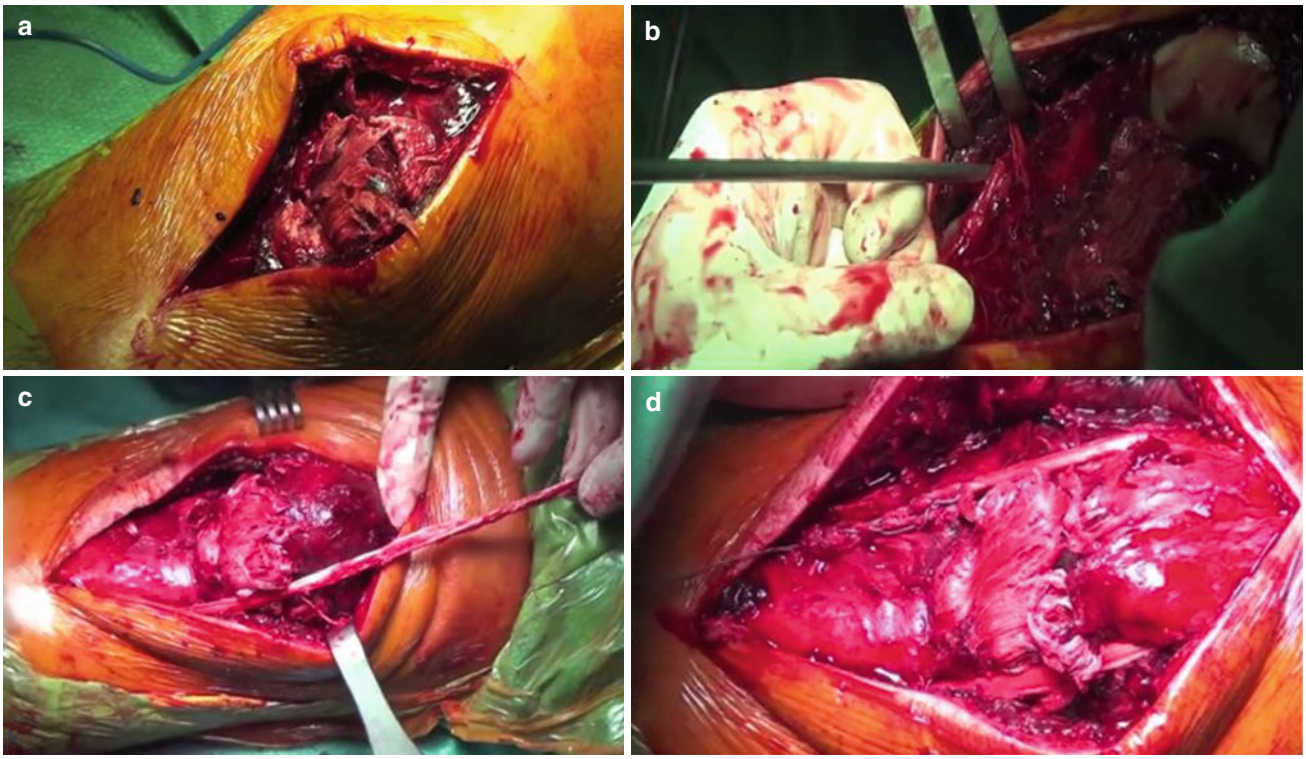


Fig. 12.2 Augmentation of patellar tendon rupture with semitendinosus tendon. (**a**) The osteotendinous insertion lesion. (**b**) Identification of the semitendinosus tendon and stripping, leaving the tibial insertion intact. (**c**) A tibial tunnel is drilled in from medial to lateral to the anterior tibial tuberosity, and the tendon is passed through it. Another tunnel is then drilled into the patella. (**d**) The tendon is passed through the second tunnel and then sutured back to the tibia with nonabsorbable stitches

case series [51]. Other authors described a technique using hamstrings passed through a longitudinal bone tunnel and then fixed to the superior pole of the patella with an Endobutton® (Smith & Nephew) [52]. However, in cases of chronic rupture, the augmentation is always suggested, even in cases in which a primary repair is possible.

Some authors suggest the use of the contralateral central third of the patellar tendon with proximal and distal bone plugs for the reconstruction of chronic patellar tendon rupture [7, 53]. Wiegand et al. proposed their technique using a Y-shaped flap folded back from the vastus lateralis fascia with good clinical and functional results [54]. Recently, different surgical techniques have been proposed using allograft tissue in patellar tendon reconstruction. Probably, the most commonly used allograft is the Achilles tendon [55]. The bone plugs of the allograft are normally fixed to the tibial tubercle after a squared trench is performed. The tendon is split into three parts: the central one is passed through a longitudinal patellar tunnel and sutured to the superior pole of the patella, and the other two parts are sutured to the lateral and medial retinaculum with nonabsorbable stitches.

In chronic settings, also synthetic grafts have been used. Naim et al. in a case report described a surgical technique using LARS ligament for the reconstruction of a patellar chronic tendon rupture in a low-demand patient [56]. However, because of the low biological profile of synthetic grafts, they are normally indicated in low-demanding patients.

12.5 Outcome

Outcomes following patellar tendon repair or reconstruction are favorable. Siwek and Rao in 1981 reported good results in 80 % of patients treated with acute patellar tendon rupture repair using sutures augmented with a Steinmann [45].

Dejour et al. were the first to report on using contralateral patellar ligament for chronic patellar tendon reconstruction, with good results on 13 patients at 8.6 months of follow-up [57]. Hsu et al. examined 35 patients treated with primary repair associated to a neutralization wire. Using the same criteria proposed by Siwek and Rao, there were 57 % of excellent outcomes, without any re-rupture [33]. Marder and Timmerman reported on 14 patients treated with primary repair without augmentation with 86 % of excellent results allowing them to resume their previous activity [44]. Kasten et al. compared two different augmentations after a direct suture of the tendon: the first using a wire cerclage and the

second with PDS. They concluded that both the techniques were reliable and demonstrated good intermediate to long-term results [36].

Bhargava et al. reported good results with an average range of motion (ROM) of 1,378° in patients treated with repair and cerclage augmentation [58]. Ramseier et al. showed no differences in terms of muscle strength between injured and uninjured leg in a series of 19 cases of acute patellar tendon ruptures [26]. Bushnell et al. described their technique using suture anchors in 14 cases to repair acute patellar tendon lesion, with good or excellent results in 11 patients [27]. West et al. reported on 30 patellar tendons in which they perform a primary repair augmented with a non-absorbable suture and early postoperative mobilization, with the patients reaching their pre-injury activity level in 6 months [59]. Massoud et al. described a new reinforcement suture called "suture line tension-regulating suture," with the aim to reduce forces on the primary suture using an absorbable device. They reported good results using this technique, with good ROM and Insall-Salvati ratio [60]. In 2013, Wiegand et al. described a new technique for chronic patellar tendon rupture using a Y-shaped flap folded back from the vastus lateralis fascia. The authors reported good physical and ultrasound healing, with increasing in the mean Knee Society pain and function scores as well as the average ROM [54]. Maffulli et al. in 2013 described their surgical technique in which they performed an autologous ipsilateral hamstring tendon reconstruction for the management of chronic patellar tendon rupture. The authors concluded that all patients returned to their pre-activity level, and 14 out of 16 patients were very satisfied with the procedure [48].

Recently, Jain et al. described a percutaneous technique using semitendinosus tendon for reconstruction of chronic patellar tendon rupture, with all patients showing quadriceps strength 80 % or more compared to the contralateral leg.

In conclusion, in literature small case series of different techniques for both acute and chronic patellar tendon rupture are reported; in most of the cases, the authors described good results, without significant differences between the techniques.

Table 12.1 briefly describes the case series reported on literature.

12.6 Rehabilitation and Return to Play

In literature, different postoperative protocols have been described. The aim of the surgery should be to perform a repair or reconstruction as much stable as possible in order to allow early mobilization and to reduce the risk of arthrofibrosis and loss of ROM.

West et al. suggested to begin early ROM in the first week after surgery limiting motion from 0° to 45° with active flexion and passive extension. After the first week, they normally increase the ROM of 15° each week. Besides, the authors suggested beginning of isometric quadriceps and hamstring exercises from the day after the surgery, and the patients were in full weight bearing with the knee locked in full extension for 6 weeks. With this protocol, the patients demonstrate good quadriceps strength in 12–20 weeks [11, 59]. Other authors suggested a more prudent postoperative protocol with knee immobilization in a walking cast for at least 1 month, followed by a movable splint until rehabilitation begins. In the cases in which this protocol has been used the patients required 6 months to obtain full knee function recovery [7]. However, complications after knee immobilization include also decreased patellar mobility, persistent pain, muscle weakness, and patella baja. West advocated the utility of using a protective "relaxing suture" strong enough to allow early ROM. Postoperatively, a hinged knee brace locked in extension during ambulation was used for 6 weeks and removed daily to allow active ROM exercises from 0° to 55°, in association to full weight bearing. With this postoperative protocol, they obtain 120° of flexion and brace-free ambulation at a mean of 7.7 weeks, with all patients reaching their previous activity level in 6 months [59]. Other authors reported similar postoperative protocols with a hinged knee brace locked in extension for 6 weeks allowing immediate ROM exercises from 0° to 45° with good clinical results [44]. For what concerns strengthening exercise, some authors allowed straight-leg raising without resistance at 6–8 weeks, stationary cycling at 8 weeks, and progressive quadriceps muscle exercises at 12 weeks. Running was normally permitted at 16–20 weeks, and contact sports or jumping was permitted at a minimum of 6 months after repair [44]. Braghava et al. described a third type of postoperative protocol in association to an augmented repair in which no immobilization was used; the patients started continuous passive motion from 0° to 60° immediately, and they were allowed to mobilize with crutches up to 65 weeks after surgery [58].

There is no randomized control trial comparing early ROM to cast immobilization after patellar tendon repair or reconstruction. Generally speaking, if a strenuous primary repair is performed, an early ROM between 0° and 45–60° can be allowed, overall if an augmentation is performed. On contrary, in cases of tenuous repair, chronic lesions, or re-rupture, a more prudent postoperative protocol is suggested, but the risk of loss of ROM increased. Some authors stated that cast immobilization may be better compared to hinged knee brace because it better maintains the position of the patella and patellar tendon, but it should be used for no more than 2 weeks to prevent stiffness and anterior knee pain [48].

Most of the authors reported good clinical outcomes using both early mobilization and cast immobilization, with return of the pre-injury activity level in most of the patients. However, there are few studies reporting on return-to-sport

Table 12.1 Brief description of the case series reported on literature with their results

Author	Year	Number	Median age (range)	Median follow-up	Technique	Results
Siwek [45]	1981	31 patients (25 acute/6 chronic)	<40 years	2–11 years	Acute: Bunnell pull-out wire augmented with a Steinman	Acute repair: 20 excellent results, 4 good, and 1 re-rupture
					Chronic: initially treated with traction	Chronic repair: 2 excellent results, 3 good, and 1 unsatisfactory result
Dejour [57]	1992	13 (chronic)	Not reported	8.6 months	Contralateral bone-patellar-bone tendon	Correction of the extensor lag in all the cases and mean postoperative flexion of 91°
Hsu [33]	1994	35 patients (all acute)	Not reported	Not reported	Primary repair with a neutralization wire	57 % of outcomes excellent; 28.6 % good; and 14.2 % unsatisfactory. No re-rupture of the tendon
Marder [44]	1999	14 patients (acute)	33 years	2.6 years	Primary repair with Krackow and patella tunnels	86 % excellent results with patients resuming their previous activity level
Kasten [36]	2001	32 patients	Not reported	8.1 years (1–18)	End-to-end suture augmented with wire cerclage or PDS	2 of 22 patients had an extension lag in group A (wire cerclage) compared with no extension lag in group B (PDS cerclage). Average flexion in group
						A was 130° (SD 29°) compared with 137° (SD 12°) in group B. The average Hospital for Special Surgery Knee Score was 92 (SD 17) in group A and 96 (SD 12) in group B. Three patients were dissatisfied to determine whether this phenomenon can be reproduced
Bhargava [58]	2004	11 patients (acute)	42 years	26 months (14–38)	Suture repair of the patella tendon and retinacula protection by a cerclage wire	The mean Lysholm knee score at review was 97 (range 92–100). The mean Insall-Salvati measurement was 1 (range 0.95–1.1). No patients had fixed flexion deformities or demonstrated an extensor lag, and the average range of motion was 0–137°. Mean loss of power of 6 % (range 2–11) when measuring concentric extension and a mean loss of 7 % power in concentric flexion
Ramseier [26][a]	2006	19	46.1 years	57 months	Suture repair associated to McLaughlin cerclage	No difference between the range of motion and muscle strength when the injured leg was compared to the non-injured leg. Multiple injured patients had a significant reduction in muscle strength ($p=0.017$) and circumference ($p=0.019$); however, patient satisfaction did not differ from the non-multiple injured patient group

Bushnell [27]	2008	14	34 years	29 months	Suture anchors	No intraoperative complication, 3 failures (21 %), mean extension gap 1°
West [59]	2008	30	44 years	4 years (1–12)[a]	Trans-patellar repair with nonabsorbable wire augmentation	No complication. No evidence of shortening or lengthening of the tendon. At 6 months, 40 had full extension, and 10 lacked 3–10° of active extension (not specified if quadriceps or patellar tendon)
Massoud [60]	2010	12	32 years (18–42)	45 months (24–48)	Trans-patellar suture reinforced with a "suture line tension-regulating suture"	The patients resumed their pre-injury activities at an average of 6.1 months. The active knee movement averaged 0–154.6° compared to 0–156.7° in the contralateral knee
Wiegand [54]	2013	16	68 years (37–81)	28 months (16–36)	Y-shaped flap folded back from the vastus lateralis fascia	Both the physical and ultrasound monitoring showed that all 16 tendons healed properly. The mean Knee Society pain and function scores as well as the average range of motion of the knee increased significantly after the operations
Maffulli [48]	2013	19	Not reported	5.8 years (4–7.8)	Ipsilateral hamstring tendon graft reconstruction	The mean modified Cincinnati and Kujala scores were notably improved. All patients had returned to ordinary daily activities. 14 patients were very satisfied, 3 were satisfied, 3 were moderately satisfied, and 1 was unsatisfied
Jain [51]	2014	9	31.5 years	4.5 years	Percutaneous semitendinosus reconstruction	Lysholm score and Siwek and Rao grading were good to excellent in all patients, and at 12 months, all patients showed quadriceps strength 80 % or more of opposite quadriceps

[a]Age and months referred to the entire group (quadriceps and patellar tendon repair)

rate after acute or chronic patellar ruptures, with most of them reporting on moderate rates.

Conclusion

Patellar tendon rupture is a rare but complex pathology. When occurring in young people, it can be a consequence of a serious trauma or a systemic pathology. In every case, considering the low vascularization and number of cells, the repair and the following healing are a challenge. For low-energy lesions, considering systemic pathologies causing tendinopathy, it can be necessary to treat systemic pathology in a multidisciplinary approach. The history told by the patient can be really suggestive but has to be confirmed by instrumental exams in order to locate and measure the lesion.

Different surgical techniques have been described both for acute and chronic patellar tendon ruptures.

In the acute setting, most of the authors described a repair using trans-patellar tunnels associated or not to an augmentation. Recently, other authors reported on patellar tendon repair using suture anchors, with some biomechanical studies demonstrating less gap formation after cyclic loading compared to trans-patellar sutures. The role of the augmentation in acute patellar tendon rupture is still debated. The advantage in using augmentation, with both metallic and nonabsorbable wires, is that repair is more theoretically strenuous allowing an early mobilization. On the other hand, augmentation with metallic wire required a second surgery to remove it. However, there are no studies confirming better outcomes in patients in which an augmentation was performed compared to suture repair alone.

In the chronic setting, a primary repair is often not suitable because of tendon retraction. In these cases, a reconstruction is suggested, and different autologous, allogenic, or synthetic grafts have been used, with the semitendinosus the most described. There are no studies describing the superiority of a graft or of a surgical technique on the others. In chronic patellar tendon rupture, postoperative protocols are normally more cautious, with a longer period of knee immobilization.

There is no consensus in literature on the most proper postoperative and rehabilitation protocol. Good results have been described both for more cautious and aggressive protocols, and most of the literature concluded that the rehabilitation should be set on the strength of the repair/reconstruction.

In conclusion, patellar tendon ruptures are relatively rare but often require surgery. Neglected or chronic patellar tendon rupture is exceptional. If surgery is performed in the acute setting (within 7–14 days), primary suture repair can be performed, and functional results are normally satisfactory. In case of tendon retraction or defect, however, the surgical technique can be more demanding and functional outcomes less satisfactory.

References

1. Sharma P, Maffulli N (2005) Tendon injury and tendinopathy: healing and repair. J Bone Joint Surg Am 87(1):187–202. doi:10.2106/JBJS.D.01850
2. Raschke D, Schuttrumpf JP, Tezval M, Sturmer KM, Balcarek P (2014) Extensor-mechanism-reconstruction of the knee joint after traumatic loss of the entire extensor apparatus. Knee 21(3):793–796. doi:10.1016/j.knee.2014.02.003
3. Kannus P, Jozsa L, Jarvinnen M (2000) Basic science of tendons. In: Garrett WE Jr, Speer K Jr, Kirkendall DT (eds) Principles and practice of orthopaedic sports medicine. Lippincott Williams & Wilkins, Philadelphia, pp 21–37
4. Vailas AC, Tipton CM, Laughlin HL, Tcheng TK, Matthes RD (1978) Physical activity and hypophysectomy on the aerobic capacity of ligaments and tendons. J Appl Physiol Respir Environ Exerc Physiol 44(4):542–546
5. Carr AJ, Norris SH (1989) The blood supply of the calcaneal tendon. J Bone Joint Surg Br 71(1):100–101
6. Kvist M, Hurme T, Kannus P, Jarvinen T, Maunu VM, Jozsa L et al (1995) Vascular density at the myotendinous junction of the rat gastrocnemius muscle after immobilization and remobilization. Am J Sports Med 23(3):359–364
7. Saragaglia D, Pison A, Rubens-Duval B (2013) Acute and old ruptures of the extensor apparatus of the knee in adults (excluding knee replacement). Orthop Traumatol Surg Res 99(1 Suppl):S67–S76. doi:10.1016/j.otsr.2012.12.002
8. Zernicke RF, Garhammer J, Jobe FW (1977) Human patellar-tendon rupture. J Bone Joint Surg Am 59(2):179–183
9. Matava MJ (1996) Patellar tendon ruptures. J Am Acad Orthop Surg 4(6):287–296
10. Kelly DW, Carter VS, Jobe FW, Kerlan RK (1984) Patellar and quadriceps tendon ruptures – jumper's knee. Am J Sports Med 12(5):375–380
11. Lee D, Stinner D, Mir H (2013) Quadriceps and patellar tendon ruptures. J Knee Surg 26(5):301–308. doi:10.1055/s-0033-1353989
12. Clark SC, Jones MW, Choudhury RR, Smith E (1995) Bilateral patellar tendon rupture secondary to repeated local steroid injections. J Accid Emerg Med 12(4):300–301
13. Peters KM, Bucheler D, Westerdorf G (2000) [Bilateral rupture of the patellar ligament in diabetes mellitus]. Unfallchirurg 103(2):164–167
14. Ramirez-Castillo HD, Carbajal-Contreras R, Gonzalez-Morales DD (2010) Acute bilateral lesion of the patellar tendon associated to diabetes mellitus. Case report. Acta Ortop Mex 24(1):23–27
15. Blazina ME, Kerlan RK, Jobe FW, Carter VS, Carlson GJ (1973) Jumper's knee. Orthop Clin North Am 4(3):665–678
16. Alves EM, Macieira JC, Borba E, Chiuchetta FA, Santiago MB (2010) Spontaneous tendon rupture in systemic lupus erythematosus: association with Jaccoud's arthropathy. Lupus 19(3):247–254. doi:10.1177/0961203309351729
17. Oda R, Fujiwara H, Tokunaga D, Kishida A, Taniguchi D, Seno T et al (2014) Spontaneous flexor tendon rupture in systemic lupus erythematosus: a case report. Mod Rheumatol:1–4. doi:10.3109/14397595.2014.924193
18. Lu M, Johar S, Veenema K, Goldblatt J (2012) Patellar tendon rupture with underlying systemic lupus erythematosus: a case report. J Emerg Med 43(1):e35–e38. doi:10.1016/j.jemermed.2009.08.054
19. Hughes GN, Harder JA (1979) Bilateral patellar tendon rupture associated with chronic glomerulonephritis. Can J Surg 22(4):389

20. Sullivan RL (1986) Traumatic bilateral patellar tendon rupture with chronic renal disease. Wis Med J 85(2):12–13
21. Kartus J, Movin T, Karlsson J (2001) Donor-site morbidity and anterior knee problems after anterior cruciate ligament reconstruction using autografts. Arthroscopy 17(9):971–980. doi:10.1053/jars.2001.28979
22. Maffulli N, Wong J (2003) Rupture of the Achilles and patellar tendons. Clin Sports Med 22(4):761–776
23. Chen CH, Niu CC, Yang WE, Chen WJ, Shih CH (1999) Spontaneous bilateral patellar tendon rupture in primary hyperparathyroidism. Orthopedics 22(12):1177–1179
24. Heyde CE, Mahlfeld K, Stahel PF, Kayser R (2005) Ultrasonography as a reliable diagnostic tool in old quadriceps tendon ruptures: a prospective multicentre study. Knee Surg Sports Traumatol Arthrosc 13(7):564–568. doi:10.1007/s00167-004-0576-7
25. Benner RW, Shelbourne KD, Urch SE, Lazarus D (2012) Tear patterns, surgical repair, and clinical outcomes of patellar tendon ruptures after anterior cruciate ligament reconstruction with a bone-patellar tendon-bone autograft. Am J Sports Med 40(8):1834–1841. doi:10.1177/0363546512449815
26. Ramseier LE, Werner CM, Heinzelmann M (2006) Quadriceps and patellar tendon rupture. Injury 37(6):516–519. doi:10.1016/j.injury.2005.12.014
27. Bushnell BD, Tennant JN, Rubright JH, Creighton RA (2008) Repair of patellar tendon rupture using suture anchors. J Knee Surg 21(2):122–129
28. Ong BC, Sherman O (2000) Acute patellar tendon rupture: a new surgical technique. Arthroscopy 16(8):869–870
29. Ho HM, Lee WK (2003) Traumatic bilateral concurrent patellar tendon rupture: an alternative fixation method. Knee Surg Sports Traumatol Arthrosc 11(2):105–111. doi:10.1007/s00167-002-0332-9
30. Capiola D, Re L (2007) Repair of patellar tendon rupture with suture anchors. Arthroscopy 23(8):e1–e4
31. Bushnell BD, Byram I, Weinhold PS, Creighton RA (2006) The use of suture anchors in repair of the ruptured patellar tendon: a biomechanical study. Am J Sports Med 39(4):1492–1499
32. Ettinger M, Dratzidis A, Hurschler C, Brand S, Calliess T, Krettek C et al (2013) Biomechanical properties of suture anchor repair compared with transosseous sutures in patellar tendon ruptures: a cadaveric study. Am J Sports Med 41(11):2540–2544. doi:10.1177/0363546513500633
33. Hsu KY, Hsu WW, Ho WP, Wang KC (1994) Traumatic patellar tendon ruptures – a follow-up study of primary repair and neutralization wire. Changgeng Yi Xue Za Zhi 17(1):39–43
34. Giles SN, Morgan-Jones R, Brown MF (1999) The use of hinged Kirschner wires for fixation of patellar tendon rupture. Injury 30(8):539–540
35. Mc LH (1947) Repair of major tendon ruptures by buried removable suture. Am J Surg 74(5):758–764
36. Kasten P, Schewe B, Maurer F, Gosling T, Krettek C, Weise K (2001) Rupture of the patellar tendon: a review of 68 cases and a retrospective study of 29 ruptures comparing two methods of augmentation. Arch Orthop Trauma Surg 121(10):578–582
37. Fujikawa K, Ohtani T, Matsumoto H, Seedhom BB (1994) Reconstruction of the extensor apparatus of the knee with the Leeds-Keio ligament. J Bone Joint Surg Br 76(2):200–203
38. Levy M, Goldstein J, Rosner M (1987) A method of repair for quadriceps tendon or patellar ligament (tendon) ruptures without cast immobilization. Preliminary report. Clin Orthop Relat Res 218:297–301
39. Miskew DB, Pearson RL, Pankovich AM (1980) Mersilene strip suture in repair of disruptions of the quadriceps and patellar tendons. J Trauma 20(10):867–872
40. Kinmont JC, Walter E, Curtis MJ (2002) Augmentation of patellar tendon repair with poly-p-dioxannone cord. Injury 33(3):263–264
41. Larson RV, Simonian PT (1995) Semitendinosus augmentation of acute patellar tendon repair with immediate mobilization. Am J Sports Med 23(1):82–86
42. Mihalko WM, Vance M, Fineberg MJ (2010) Patellar tendon repair with hamstring autograft: a cadaveric analysis. Clin Biomech (Bristol, Avon) 25(4):348–351. doi:10.1016/j.clinbiomech.2010.01.003
43. Ravalin RV, Mazzoca AD, Grady-Benson JC, Nissen CW, Adams DJ (2002) Biomechanical comparison of patellar tendon repairs in a cadaver model: an evaluation of gap formation at the repair site with cyclic loading. Am J Sports Med 30(4):469–473
44. Marder RA, Timmerman LA (1999) Primary repair of patellar tendon rupture without augmentation. Am J Sports Med 27(3):304–307
45. Siwek CW, Rao JP (1981) Ruptures of the extensor mechanism of the knee joint. J Bone Joint Surg Am 63(6):932–937
46. Ecker ML, Lotke PA, Glazer RM (1979) Late reconstruction of the patellar tendon. J Bone Joint Surg Am 61(6A):884–886
47. Nguene-Nyemb AG, Huten D, Ropars M (2011) Chronic patellar tendon rupture reconstruction with a semitendinosus autograft. Orthop Traumatol Surg Res 97(4):447–450
48. Maffulli N, Del Buono A, Loppini M, Denaro V (2013) Ipsilateral hamstring tendon graft reconstruction for chronic patellar tendon ruptures: average 5.8-year follow-up. J Bone Joint Surg Am 95(17):e1231–e1236. doi:10.2106/JBJS.L.01462
49. Harris JD, Fazalare JJ, Phieffer LS, Flanigan DC (2013) Patellar tendon reconstruction with semitendinosus-gracilis autograft. J Knee Surg 26(Suppl 1):S19–S24. doi:10.1055/s-0031-1280972
50. Chen B, Li R, Zhang S (2012) Reconstruction and restoration of neglected ruptured patellar tendon using semitendinosus and gracilis tendons with preserved distal insertions: two case reports. Knee 19(4):508–512. doi:10.1016/j.knee.2011.07.007
51. Jain JK, Vidyasagar JV, Chabra R (2014) Percutaneous reconstruction of patellar tendon using semitendinosus tendon in chronic patellar tendon injury – case series and outcome. Knee 21(3):726–730. doi:10.1016/j.knee.2014.02.002
52. Gokce A, Ekici H, Erdogan F (2008) Arthroscopic reconstruction of a ruptured patellar tendon: a technical note. Knee Surg Sports Traumatol Arthrosc 16(6):581–584. doi:10.1007/s00167-008-0511-4
53. Milankov MZ, Miljkovic N, Stankovic M (2007) Reconstruction of chronic patellar tendon rupture with contralateral BTB autograft: a case report. Knee Surg Sports Traumatol Arthrosc 15(12):1445–1448. doi:10.1007/s00167-007-0365-1
54. Wiegand N, Naumov I, Vamhidy L, Warta V, Than P (2013) Reconstruction of the patellar tendon using a Y-shaped flap folded back from the vastus lateralis fascia. Knee 20(2):139–143. doi:10.1016/j.knee.2012.05.008
55. Burks RT, Edelson RH (1994) Allograft reconstruction of the patellar ligament. A case report J Bone Joint Surg Am 76(7):1077–1079
56. Naim S, Gougoulias N, Griffiths D (2011) Patellar tendon reconstruction using LARS ligament: surgical technique and case report. Strategies Trauma Limb Reconstr 6(1):39–41. doi:10.1007/s11751-010-0101-0
57. Dejour H, Denjean S, Neyret P (1992) [Treatment of old or recurrent ruptures of the patellar ligament by contralateral autograft]. Rev Chir Orthop Reparatrice Appar Mot 78(1):58–62
58. Bhargava SP, Hynes MC, Dowell JK (2004) Traumatic patella tendon rupture: early mobilisation following surgical repair. Injury 35(1):76–79
59. West JL, Keene JS, Kaplan LD (2008) Early motion after quadriceps and patellar tendon repairs: outcomes with single-suture augmentation. Am J Sports Med 36(2):316–323. doi:10.1177/0363546507308192
60. Massoud EI (2010) Repair of fresh patellar tendon rupture: tension regulation at the suture line. Int Orthop 34(8):1153–1158. doi:10.1007/s00264-009-0879-x

Hamstring Injuries

13

Gianni Nanni, Francesco Della Villa, Margherita Ricci, Diego Rizzo, and Stefano Della Villa

13.1 Introduction

Muscle injuries represent the most common type of injury in football, and hamstring injuries are the most common subtype, representing 12 % of all injuries. In professional football, muscle injuries represent 31 % of all injuries, causing 25 % of layoff time from training and competition. A recent study showed that a team of 25 players can expect 4–6 hamstring injuries per season [1]. Referring to hamstrings, 83 % of injuries affect the biceps femoris muscle, 11 % the semimembranosus muscle, while only 5 % the semitendinosus [2]. Even if the influence of this kind of injury has been very widely studied in professional sports, especially in football, this issue is also impacting on competitive (lower divisions) and recreational athletes, both in football and other sports, especially rugby, dancing and track and field [3]. One of the most challenging aspects of hamstring injuries is the high rate of recurrence (12–43 % reported in recent literature), which probably indicates inadequate rehabilitation programme and/or a premature return to sport.

Despite their clinical relevance, the treatment of these injuries is still controversial and often empirical. We think that a proper approach should be based on a clear understanding of the injury mechanism, a quick and correct diagnosis, a well-planned recovery programme and a safe return to sport strategy.

The aim of this chapter is to go through all the aspects of hamstring disorders, from the injury to return to play and reinjury prevention.

G. Nanni • D. Rizzo
Isokinetic Medical Group – FIFA Medical Centre of Excellence, Via Casteldebole 8/10, Bologna, Italy 40132, Italy
e-mail: g.nanni@isokinetic.com; d.rizzo@isokinetic.com

F.D. Villa • M. Ricci • S.D. Villa (✉)
Isokinetic Medical Group – FIFA Medical Centre of Excellence, Via Casteldebole 8/4, Bologna, Italy 40132, Italy
e-mail: f.dellavilla@isokinetic.com; m.ricci@isokinetic.com; s.dellavilla@isokinetic.com

13.2 Injury Mechanism

In order to discuss the topic of *hamstring* injuries, it is important to comprehend what the main risk factors are (RF) and how these injuries happen.

Two recent systematic reviews have been performed in order to establish the evidence about the RF for hamstring injuries [4, 5]. Foreman et al. [4] and Prior et al. [5] identified hamstring muscle weakness, thigh muscle imbalance, limited muscle flexibility and previous hamstring injury as potential risk factors. Among demographic characteristics, age and black ethnicity also seem to play a role.

The strongest risk factor seems to be a history of previous hamstring strain [5], with a reported 6.3 times greater relative risk at 8 weeks from return to activity [95 % confidence interval (CI), 5.21–7.70] [6]. This result was also confirmed by Freckleton et al. [7] in another recent review.

Hamstring injuries can occur with both *noncontact* (NC) or *contact* (C) traumatic events. NC patterns are the most common mechanisms involved, and they can occur in a variety of situations, while C injuries are due to the direct application of external force. We will mainly focus on the first category.

Regarding NC injuries, there are at least two different types of acute injury mechanisms. The first one occurs during high-speed running and sprinting (*type I or high-speed running type*). The other occurs during movements leading to extensive lengthening of the hamstrings, such as high kicking and sliding tackles (*type II or stretching type*) [8]. Askling et al. [8] argued that this distinction may play an important role in the recovery process.

Type I injuries usually involve the long head of the biceps femoris and may be located at both the proximal and distal muscle-tendon junctions. In this case, the main mechanism of injury, as mentioned above, is sprinting. They normally occur during the terminal swing phase of running, when the hamstrings work eccentrically, to decelerate the knee extension. Type I injuries are more common in sports like football, rugby and track and field.

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_13,

Type II injuries usually involve the proximal hamstrings near the ischial tuberosity and can be typically located at the proximal free tendon of the semimembranosus. The mechanism of injury is often due to an excessive hamstring stretch in hip flexion. Type II injuries are more common in sports like dance and gymnastics. This type of injury is often due to a longer recovery time when compared to type I [8].

As we mentioned before, a deep comprehension of the injury mechanism is a crucial part of the recovery process.

13.3 Diagnosis

It is well established that an immediate and correct diagnosis is the key to plan an appropriate rehabilitation programme [9]. The diagnosis process comprehends an accurate *clinical history,* a careful *physical examination* and the appropriate *imaging investigations.*

During the *clinical history* there has to be a proper identification of patient's general characteristics, including the type of sport, sport level, training history and patient's general health. In addition, focusing on the actual problem, the clinician has to investigate the exact circumstances of the injury and the symptoms' onset and features. Plus it is mandatory to gather information about any previous injury, obviously focusing on previous hamstring disorders, being aware of the fact that a recurrence differs from a primary injury.

The pain history is a crucial aspect in hamstring injuries. There are at least two modalities of pain onset. The first scenario is defined by a sudden onset of a well-localised and sharp pain, evocated by a specific movement and preceded by a "pop" or "snap" feeling. This is the typical onset modality of *structural injuries*, according to the recent Munich consensus [10]. The second one may be characterised by a progressive (during activity) or delayed (typically post activity) onset of a poorly localised muscle soreness. These are the typical onset modalities of *functional (nonstructural) injuries* that represent the vast majority of hamstring disorders.

Another feature of *structural injuries* is the immediate functional impairment, frequently reported by the patient as a sudden fall to the ground after the injury. In this case, most of the time, the athlete cannot continue to perform and has to stop the activity.

The *physical examination* has to be carefully performed with a step-by-step approach. It consists of a posture and gait inspection, inspection and palpation of the muscle bellies, flexibility tests, strength evaluation and functional tests [11]. It is recommended to perform any evaluation or test bilaterally, to compare the injured limb to the contralateral.

In the literature, the first clinical examination is often carried out within 12 h to 2 days post-injury [12].

During the gait inspection the patient may limp, due to the knee extension deficit and pain. Plus you can evaluate the dynamic muscle morphology comparing it to the contralateral side; there may be discontinuity in the muscle shape. This is especially true if we are dealing with a severe injury and in the acute phase after trauma.

The inspection and palpation of muscle bellies are clinical milestones of the diagnostic process. Through the inspection, swelling and ecchymosis of the posterior thigh may be detected; these skin changes usually arise a few days after the injury.

The palpation, craniocaudally performed, is useful for identifying the right site of the injury through pain provocation and the presence/absence of a palpable defect. The palpation findings are different in *functional* and *structural* injuries.

In *functional* injuries a muscle bundle stiffness is frequently present, while other findings may be missing. On the other hand, *structural* injuries are characterised by a well-localised evocated pain during palpation.

Depending on the severity and size of the lesion, a structural defect may be found. This finding is often missing in *minor partial tear* [10] (defect size less than a muscle fascicle/bundle) because of the limited number of disrupted fibres, while it is often detected in *moderate partial tear* [10] (defect size greater than a muscle fascicle/bundle) and always present in *subtotal muscle tear and tendinous avulsion* [10].

Flexibility tests are usually performed to assess the degree of impairment. Both the hip and knee ranges of motions (ROM) are decreased in the acute phase [9]. The flexibility tests, like the "the sit and reach test" or the "hip flexibility test", are often positive.

A manual against resistance strength evaluation may be performed. For example, you can evaluate the knee flexor strength with the patient in a prone position, with extended hip and applying resistance at the heel [9]. The two main findings may be a strength deficit and an evocated pain during the muscle contraction. They are often both present in severe *structural injuries* and often missing in *functional injuries*. The two previously mentioned signs (strength deficit and evocated pain) are equally clinically important.

As part of the differential diagnosis, the process is crucial not to miss a referred pain to the posterior thigh that can be due to lumbar spine problems. The distinction between an L5–S1 disc prolapse and a real hamstring injury may be based on the clinical history and imaging [11].

The clinical suspect is usually confirmed with a proper use of *imaging investigations*. Ultrasonography (US) and magnetic resonance imaging (MRI) are the most suitable techniques for depicting hamstring lesions [13]. In current clinical practise US is frequently used because of its

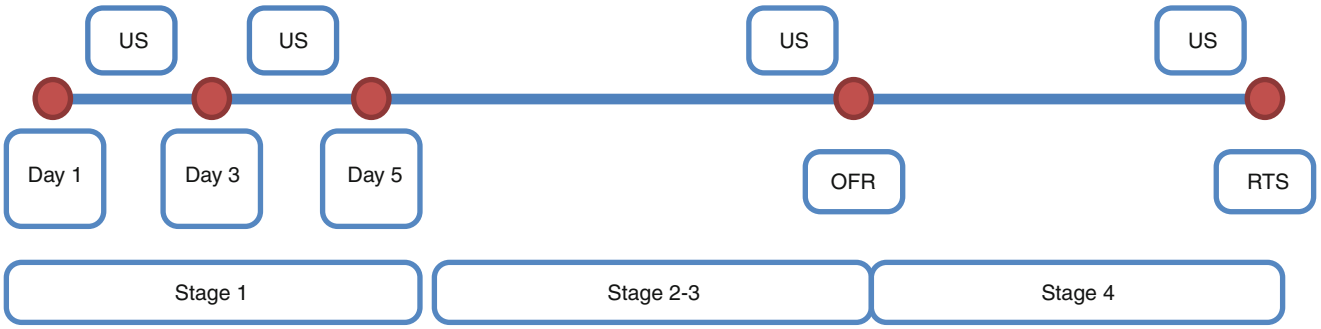


Fig. 13.1 Ultrasound monitoring scheme. *US* ultrasound, *OFR* on-field rehabilitation, *RTS* return to sport

advantages, such as the low cost, a wide availability, the relatively easy use and the possibility to perform a dynamic assessment [11]. On the other hand this modality is highly user dependent, requires long training, is poorly reproducible and is inadequate for deep structure evaluation. MRI is more sensitive for identifying low-grade injuries and deeper muscle lesions [14].

The time of imaging is still controversial, even if 24–48 h after trauma seems to be the best moment [11]. Follow-up imaging is also frequently performed to assess the progression of rehabilitation. The ideal moment of follow-up differs in every single case; however, we developed an ideal scheme of imaging (US) monitoring that we adopt in our daily activity, especially with professional athletes (Fig. 13.1).

13.4 Treatment Strategy

The conservative treatment is the recommended approach in most of the cases; surgery is required only in rare ones, such as a total proximal hamstring rupture with tendinous avulsion [15]. We will focus the discussion on the conservative treatment.

First of all, in our vision a correct strategy also means having a proper team, proper facilities and a proper method.

The team is formed by the *medical doctor,* the *physiotherapists* and the *athletic trainer.* The team acts together to coordinate the process around the patient, with a constant sharing of information. For proper facilities we mean a *rehab gym, pool and field*. The patient will be treated with sessions in each single environment. Regarding the method, we adopt a *criteria-based rehabilitation protocol* that represents the current concept for hamstring injuries.

Our protocol is divided into four different stages, with specific treatment goals and progression criteria for stage advancement and return to sport (Table 13.1).

The primary goal of rehabilitation is to return the patient to his/her pre-injury activity level as safely and quickly as possible while minimising the risk of reinjury.

Table 13.1 Criteria-based return to sport protocol for hamstring injuries

Stage 1	P.O.L.I.C.E. approach Lumbo-pelvic exercises Cold physical modalities Early mobilisation in the pool
	Walk normally without any complaints
Stage 2	Stretching/flexibility exercises (static/dynamic) Isometric strength exercises Isotonic strength exercises Core stability Aerobic threshold workout (stationary bike, tapis-roulant)
	Running (low-intensity) forwards/backwards without any complaints Pain-free hamstring strength exercises/test
Stage 3	Stretching exercises (continued) Eccentric strength exercises Proprioception exercises and core stability Aerobic and lactacid anaerobic workout Sport-specific drills (low-demand activities) FIFA 11+
	Running 20–30 m at maximal speed without any complaints Pain-free posterior chain flexibility test/stretching Complete recovery of hamstring strength
Stage 4	Eccentric strength exercises Anaerobic workout (especially alactacid, very high intensity) Sport-specific drills (high-demand activities) Sport-specific situations FIFA 11+
	*RTS criteria* No US and MRI signs of muscle lesion "Muscle feeling" as the contralateral Complete recovery of ROM and posterior chain flexibility Complete recovery of hamstring strength (isokinetic test) Recovery of aerobic/anaerobic fitness (metabolic tests) Complete the OFR programme

Each stage should be initiated only when the criteria (traffic lights) are satisfied

13.5 Rehabilitation and Return to Sport

13.5.1 Stage 1: Protection and Optimal Loading

The main goals of this stage are *reducing inflammation, protecting the injured muscle and maintaining neuromuscular activity.*

In the immediate post trauma (first 24–48 h), the muscle has to be protected from movements that could potentially influence and slow down the healing process. The PRICE (Protection, Rest, Ice, Compression and Elevation) concept [16] is extensively applied. The aim of this approach is to reduce the haematoma, prevent the fibre retraction and the overall quantity of fibrous tissue neoformation [17]. Lately the rest concept has been widely discussed. It is accepted that an optimal loading concept rather than a complete rest has to be preferred. So it is suggested to move to the new acronym POLICE which represents protection, optimal loading, ice compression and elevation [18]. This is not only a new algorithm but a stimulus to think differently and customise the rehabilitation strategies to the injury severity and location. Following this idea, it is not recommended to extend the immobilisation over 3–5 days. A careful and progressive loading enhances positive effects both histologically and biomechanically [17]. So we recommend starting the first low-intensity, ROM-protected, pain-free exercises involving the entire lower extremity and lumbo-pelvic region to develop neuromuscular control. Plus, the use of physical modality such as low-level laser therapy (LLLT) and lymph draining massage is introduced to reduce haematoma and inflammation. Moreover, an early mobilisation in the pool, using high-level water, may be planned to give progressive stimuli to the healing tissues (Fig. 13.2).

Fig. 13.2 Early mobilisation in the pool. Low-intensity exercises in the pool, with optimal loading progression, may be helpful for severe injuries

The criterion to move to the subsequent stage is *walking normally without any complaints*.

13.5.2 Stage 2: Aerobic Threshold Workout and Running Recovery

The main goals of this stage are *an initial aerobic reconditioning and the initial recovery of flexibility and strength.*

In this stage there is a progressive introduction of pain-free flexibility and strength exercises, with attention to the whole kinetic chain. It is mandatory to work properly on muscle flexibility throughout different types of exercises. Both active/passive and static/dynamic pain-free stretching exercises may be used. Isometric exercises and subsequently isotonic exercises are introduced to initially restore thigh and lower limb muscle strength (Fig. 13.3). It is important to keep applying a load progression principle; moreover, core stability and lumbo-pelvic exercises are part of the protocol. An aerobic threshold low-intensity workout is initiated as soon as possible, through the activation of the uninjured muscles. During this stage the main location of the sessions is the rehabilitation gym.

The criteria to move to the next stage are *running without pain/complaints and performing pain-free against resistance hamstring strength tests.*

13.5.3 Stage 3: Anaerobic Lactacid and Eccentric Strength Recovery

The main goals of this stage are *the complete recovery of flexibility and strength, an initial anaerobic threshold reconditioning and starting on-field exercises.*

Fig. 13.3 Proximal strengthening exercises. There are a lot of different exercises for posterior chain strengthening to be performed in the gym. It is mandatory to focus on the lesion area. In this case the patient is performing a proximal strengthening exercise

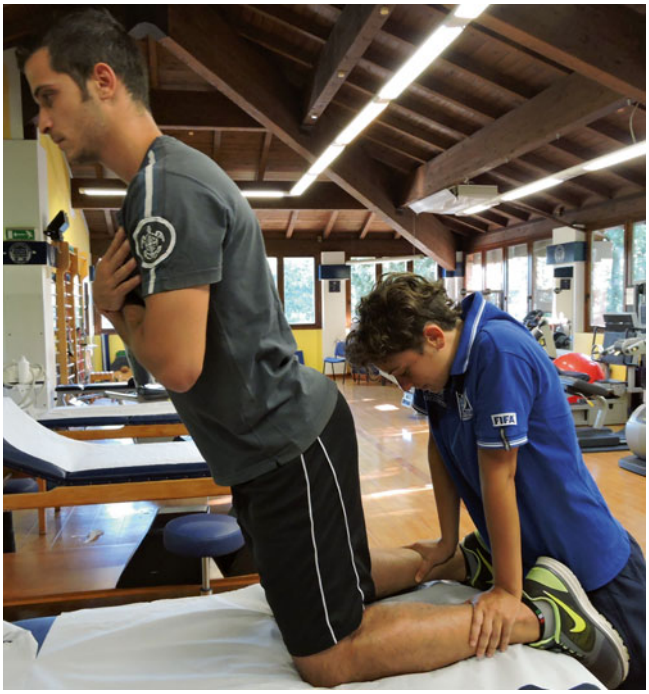


Fig. 13.4 Nordic hamstring eccentric exercise. An eccentric strengthening programme is very important both during rehabilitation and for prevention

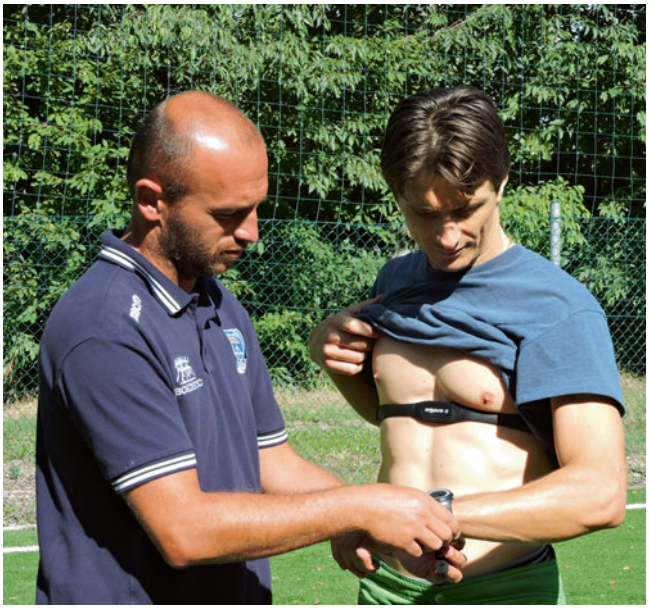


Fig. 13.5 Heart rate monitoring on the field. The work intensity progression has to be well monitored through the metabolic threshold test and the subsequent heart rate monitoring during the sessions

Stretching/flexibility and strength recovery workout are continued and intensified during this stage. Eccentric strengthening exercises are part of the programme, with the same principle of load progression (Fig. 13.4). Due to the hamstring biarticular nature, the exercises have to be properly thought out to act on the proximal, medium or distal part of the muscle group, depending on the lesion site. Proprioception exercises are introduced in the programme, with progressive increasing of complexity and duration. Subsequently they are combined with core stability, continuously proposed in the protocol. Aerobic workout is intensified and anaerobic threshold exercises are progressively introduced in the programme. During this stage the patient works both in the gym and on the field to better restore the musculoskeletal function. Very first sport-specific movements are introduced.

The criteria to move to the final stage are *running at maximal speed for 20–30 m drills without any complaints, pain-free posterior chain flexibility test/stretching and complete recovery of muscle strength.*

13.5.4 Stage 4: Return to the Team and Prevention Pro-education

The main goal of this stage is *a safe return to sport at the same activity level*, with *minimal risk of reinjury*.

The final stage of recovery, as usual, is the most challenging. During this last period, there is a progressive introduction of more complex sport-specific drills. Jumping, sprinting and cutting manoeuvres are part of the programme; the athlete should check his ability in these drills before returning to unrestricted sport. The work on the specific injury pattern has to be stressed widely; the action that caused the original injury must be repeated many times. The use of sport-specific tools is suggested to better restore the final gestures, so activities with balls have to be introduced properly. Aerobic and especially anaerobic workouts are continued and intensified compared to previous stages. We always suggest the use of a heart rate monitor (HRM) to better control the type of activity the patient is performing (Fig. 13.5).

Regarding prevention we adopt a continuous prevention strategy, from the very beginning of the programme to the last session. Specific interventions with well-known result in reducing reinjury rate, such as the Nordic eccentric hamstring exercise, are early introduced in the programme [19]. Moreover we introduce the FIFA 11+ programme during the on-field rehabilitation (OFR) sessions. Patients perform the programme at the beginning of each session. The pro-education of the patient on this very important topic is crucial to obtain a long-lived result. A three times a week maintenance programme works brilliantly in reducing musculoskeletal injuries in football, with a reduction of 29 and 37 %, respectively, in matches and training [20].

Defining *objective criteria to return to sport* (RTS) is still challenging, but not to be overlooked. The athlete should be allowed to return to the team/unrestricted individual sporting activity only when meeting certain criteria *(this is the result both of our experience and the current evidence in literature).*

The criteria to RTS are *no US or MRI signs* of muscle lesion, *feeling the injured muscle like the contralateral* during or after any activity (OFR included), *complete recovery of ROM* and *posterior chain flexibility, complete recovery of hamstring strength* (isokinetic test should be performed with the goal of complete symmetry among the limbs), *recovery of the aerobic and anaerobic fitness* (metabolic test should be performed) and *complete the OFR programme* (functional abilities to be checked).

Conclusions

Hamstring disorders are a common problem in athletes. When facing these pathologies, sports medicine clinician has to deal with a lot of different aspects, from the biological healing to the complete functional restoration. Posterior chain flexibility and strength recovery, especially eccentric, are milestones in treating these patients; however, we have to consider the metabolic aspects and a careful prevention programme as part of the recovery process.

We proposed a criteria-based strategy because we firmly believe that this approach allows the patient to return to pre-injury activity reducing the reinjury rate.

References

1. Ekstrand J, Hägglund M, Waldén M (2011) Epidemiology of muscle injuries in professional football (soccer). Am J Sports Med 39(6):1226–1232
2. Hallén A, Ekstrand J (2014) Return to play following muscle injuries in professional footballers. J Sports Sci 32(13):1229–1236
3. Askling CM et al (2008) Proximal hamstring strains of stretching type in different sports: injury situations, clinical and magnetic resonance imaging characteristics, and return to sport. Am J Sports Med 36(9):1799–1804
4. Foreman TK et al (2006) Prospective studies into the causation of hamstring injuries in sport: a systematic review. Phys Ther Sport 7:101–109
5. Prior M, Guerin M, Grimmer K (2009) An evidence-based approach to hamstring strain injury: a systematic review of the literature. Sports Health 1(2):154–164
6. Orchard JW (2001) Intrinsic and extrinsic risk factors for muscle strains in Australian football. Am J Sports Med 29(3):300–303
7. Freckleton G, Pizzari T (2013) Risk factors for hamstring muscle strain injury in sport: a systematic review and meta-analysis. Br J Sports Med 47(6):351–358
8. Askling CM, Malliaropoulos N, Karlsson J (2012) High-speed running type or stretching-type of hamstring injuries makes a difference to treatment and prognosis. Br J Sports Med 46(2):86–87
9. Heiderscheit BC et al (2010) Hamstring strain injuries: recommendations for diagnosis, rehabilitation, and injury prevention. J Orthop Sports Phys Ther 40(2):67–81
10. Mueller-Wohlfahrt HW et al (2013) Terminology and classification of muscle injuries in sport: the Munich consensus statement. Br J Sports Med 47(6):342–350
11. Kerkhoffs GM et al (2013) Diagnosis and prognosis of acute hamstring injuries in athletes. Knee Surg Sports Traumatol Arthrosc 21(2):500–509
12. Warren P et al (2010) Clinical predictors of time to return to competition and of recurrence following hamstring strain in elite Australian footballers. Br J Sports Med 44(6):415–419
13. Koulouris G, Connell D (2006) Imaging of hamstring injuries: therapeutic implications. Eur Radiol 16(7):1478–1487
14. Connell DA et al (2004) Longitudinal study comparing sonographic and MRI assessments of acute and healing hamstring injuries. AJR Am J Roentgenol 183(4):975–984
15. Askling CM et al (2013) Total proximal hamstring ruptures: clinical and MRI aspects including guidelines for postoperative rehabilitation. Knee Surg Sports Traumatol Arthrosc 21(3):515–533
16. Orchard JW et al (2008) The early management of muscle strains in the elite athlete: best practice in a world with a limited evidence basis. Br J Sports Med 42(3):158–159
17. Järvinen TA et al (2005) Muscle injuries: biology and treatment. Am J Sports Med 33(5):745–764
18. Bleakley CM, Glasgow P, MacAuley DC (2012) PRICE needs updating, should we call the POLICE? Br J Sports Med 46(4):220–221
19. Arnason A et al (2008) Prevention of hamstring strains in elite soccer: an intervention study. Scand J Med Sci Sports 18(1):40–48
20. Soligard T et al (2008) Comprehensive warm-up programme to prevent injuries in young female footballers: cluster randomised controlled trial. BMJ 337:a2469

Return to Sport After Muscle Tendon Lesions

14

Gian Luigi Canata and Valentina Casale

14.1 Introduction

Musculotendon units are responsible for force production and energy storage. Musculoskeletal movements are a result of synergistic action of skeletal muscles, tendons, and ligaments [1].

In the last 20 years, sport activities have become even more important, and even more attention has been paid to high-level athletes in competitive sports. Unfortunately this has increased the risk of traumatic and overuse injuries, because training starts even sooner and it has recently become highly intensive for longer periods of time.

Many factors contribute to the return to play, such as pain threshold, motivation, timing of the season, financial factors, and, of course, severity of injury. In view of these considerations, it can be said that return to play is a subjective result [2].

Tendinopathies and muscular disorders are a leading cause of long-term pain and physical disability worldwide.

Muscle and tendon injuries are responsible for a large portion of time lost from competition, and a quick return to training and competition is certainly a priority for all professional athletes [3]. However, it is also important not to return to sport prematurely, when the risk of reinjury is still high.

As muscles and tendons are generally subjected to large mechanical loads, they can frequently be injured. As a result, injuries cannot only cause impairment of mobility or abnormal kinematics but also undermine tissues adjacent to the joint, such as cartilage [4].

14.1.1 Pathological Processes

While appropriate mechanical loads produce anabolic effects on muscles and tendons, mechanical "overloading" can alter structural and mechanical properties of these tissues. An abnormal mechanical loading deviates from a normal one by changes in magnitude, frequency, duration, and/or direction [5].

A good example is the production of high levels of prostaglandin (PGE2), an inflammatory mediator which is present in injured tissues. Among its many properties, PGE2 can stimulate the tendon cell differentiation in non-tenocytes, potentially causing the development of non-tendinous or non-muscle tissue in tendon and muscle structures. This process alters the homogeneity of musculotendon properties, impairing tissue integrity and leading to high risks of tears and ruptures.

Other specific modifications include disrupted collagen with fibers thinner than normal, increased ground substance with high levels of glycosaminoglycans and proteoglycans, changes in cellularity, an increase in apoptosis most likely caused by oxidative stress, and, most of all, neovascularization [6].

On the other hand, insufficient mechanical loads can negatively affect muscles and tendons. Disuse or prolonged immobilization leads to changes in cell shape, cell number, collagen fiber alignment, and possible tissue degeneration. Without a constant mechanical load applied on muscles and tendons, they can become atrophied, losing weight and stiffness [4].

Musculotendinous strains and tears can be caused by a single, traumatic event due to an excessive stretching (as for high-speed runners), eccentric contractions (as for football players), or an excessive range over sequential joints (as for dancers).

Otherwise even a microlesion provoked by an eccentric exercise can sometimes develop to major tears: this happens more frequently in sports such as track and field, football, and rugby [3].

Once muscle injury has occurred, healing progresses through three distinct phases. The first one is commonly known as the *destruction inflammatory* phase, covering the first week after injury: myofibers are broken, hematoma develops, and the inflammatory cascade starts. The second and third phases consist in the *repair* and the *remodeling* stages: repair usually lasts from 2 to 6 weeks since injury

G.L. Canata, MD (✉) • V. Casale, MD
Centre of Sports Traumatology, Koelliker Hospital,
Corso Duca degli Abruzzi, 30, Turin 10129, Italy
e-mail: studio@ortosport.it, canata@ortosport.it;
casalevalentina0@gmail.com

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_14,

occurs, while remodeling begins about 7 weeks after injuring, and it can last for several months. During this time, myofibers start regeneration, and a connective tissue scar develops [7].

14.2 Evaluation

Clinical assessment of the muscle injury severity is paramount for planning the rehabilitation protocols and predicting recovery time, especially in elite athletes.

The mechanism of injury and the subsequent tissue injury have an important prognostic value in estimating the rehabilitation time needed. Injuries involving an intramuscular tendon or aponeurosis and adjacent muscle fibers typically require a shorter healing period than those involving a proximal free tendon [8].

Measuring strength, range of motion (ROM), and pain can be fundamental tools to evaluate the rehabilitation duration of lesions involving intramuscular tendon and adjacent muscle fibers.

Musculotendinous lesions are clinically characterized by a gradual development of stiffness in the tendon, activity-related pain, decreased function, and sometimes localized swelling.

Usually, clinical examination consists in evaluating pain and function by means of stretching, isometric contractions, and palpation of the pathological area.

While clinical evaluation is the basis for the diagnosis, imaging techniques are frequently used on a professional level: they can enhance the diagnosis quality in order to prognosticate healing time and duration of layoff from sport [9].

Magnetic resonance imaging (MRI) and ultrasound (US) are currently considered the first choice in the assessment of musculotendinous injuries.

Ultrasound is inexpensive and widely available, and someone prefers it to MRI for the initial assessment of injury. It allows dynamic imaging diagnosis, and whenever a large hematoma has to be drained, it can be used as a guide during the procedure.

When acute traumatic injury is present, a US image usually shows alterations in water content of the affected units. Other sonographic signs of muscle tears include avulsion and proximal retraction of the fibrofatty septa; in low-grade injuries, the space between the retracted tissue and the aponeurosis is filled with a hyperechoic area, reflecting the presence of clots and blood collection. On the other hand, worse injuries are characterized by a more copious extravasation of blood, easily detectable from 1 to 2 days after injury [10].

Power Doppler US is also useful for evaluating blood flow in the skeletal muscle, identifying hyperemia or neovascularization and then allowing the detection of even tiny lesions.

However, US sensitivity for detecting muscle healing is not as accurate as MRI. US underestimates the degree of injury; it cannot identify areas of subtle edema as well as it cannot distinguish between old and new lesions. Moreover, the use of US during follow-up can reveal some problems because it is almost impossible to reproduce the exact imaging position at the following visits [11].

Musculotendinous strains can occur even without a muscle tear. In such cases, muscle functions can be preserved. MRI shows interstitial edema and hemorrhage both at the musculotendinous junction and up to the adjacent muscle fascicles, developing hyperintensity on fluid-sensitive sequences.

When a partial tear of fibers without retraction is present, there can be a mild loss of muscle function; in this case, on MR images, hematoma at the junction and perifascial fluid accumulation can be added to interstitial edema and hemorrhage.

A complete musculotendinous rupture instead is commonly associated with a clinically visible hematoma, while MR images can be helpful for evaluating the extent of retraction [3].

14.3 Managing Healing Process

The decision whether an athlete can safely return to sport is challenging. In fact it has been reported that 59 % of reinjuries occur within the first month after return to play [12].

First and foremost, a distinction should be made between a tendon injury and lesions that involve muscle fibers, epimysial fascia, or the musculotendinous junction. Indeed, tendon tissue recovers more slowly than the others: for example, tendons defined as “free” have prolonged healing times, such as the supraspinatus, patella, quadriceps and Achilles tendon [13].

Imaging plays a significant role in determining if an athlete is ready or not to return to competition (Fig. 14.1).

US and MRI are equally sensitive in assessing muscle or tendon injury. However, MRI offers a more detailed analysis of the lesion, and it is not user dependent (Fig. 14.2).

Despite all these features, there is no clinical classification system able to predict the return to play on the basis of the mere extent of the lesion seen on MRI [2].

Nevertheless, several studies have demonstrated that whenever a structural damage is identified on MRI, return to play will take longer than those traumas without any structural defect [14].

Some authors have reported MRI findings at return to sport, such as still increased signal intensity on fluid-sensitive sequences (correlated with edema persistence) or decreased signal intensity (correlated with scar tissue formation) [15, 16].

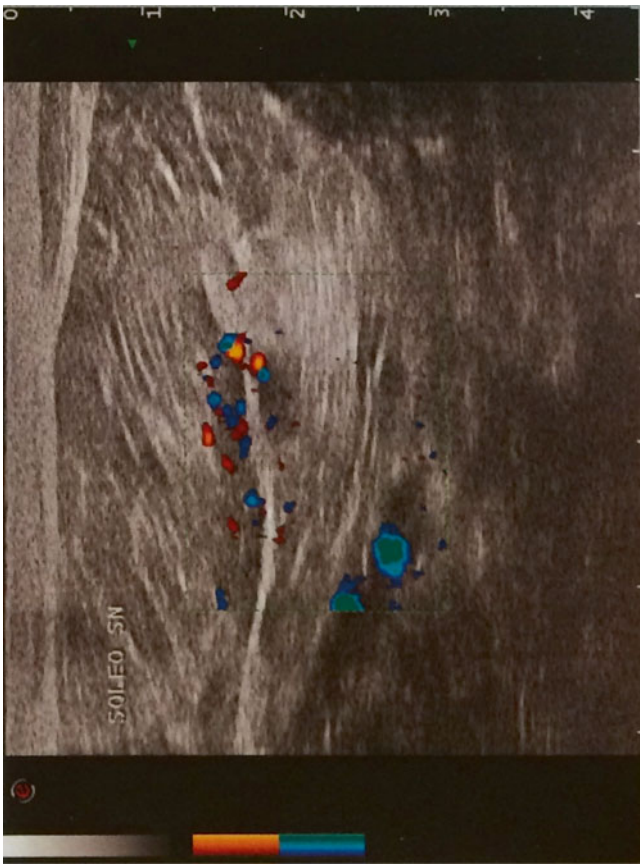


Fig. 14.1 Echographic image of mild focal soleus muscle overstretching

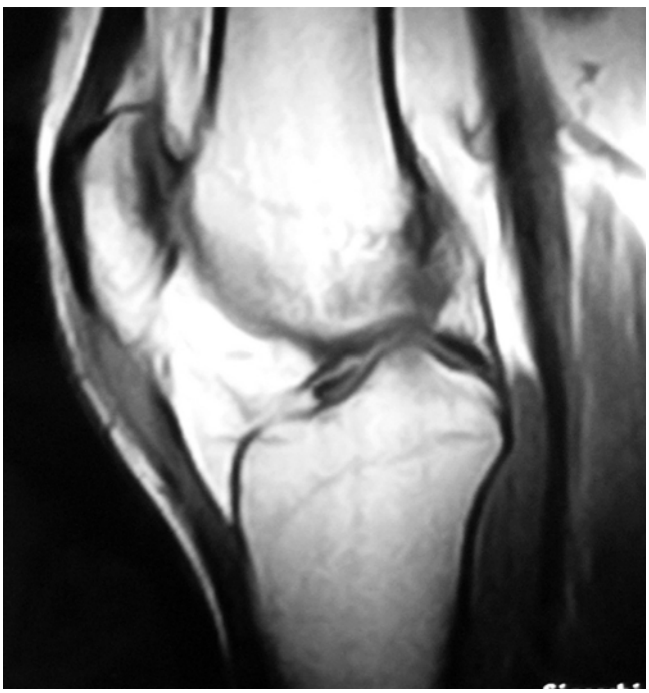


Fig. 14.2 MR image of insertional patellar tendinopathy

However, a high intensity signal in fluid-sensitive sequences in MRI sometimes does not indicate the specific phase of injury. There are indeed clinically recovered athletes in which the amount of increased signal intensity in fluid-sensitive sequences at return to sport exceeds that of other athletes at the time of initial injury. This seems to suggest that the extent of this kind of sequences does not differentiate an injured from a recovered muscle [17, 13, 12].

Nevertheless changes of the hematoma over time can successfully guide treatment decisions and managing [10].

Tendon Doppler flow among active athletes can show adaptive response to mechanical loading. A neovascularity is considered one of the hallmark features of tendinopathy. Despite this, it has not been demonstrated whether tendon Doppler flow is related to prolonged activity, although it is well known that Doppler flow increases after an acute improvement of mechanical loading [18].

Some authors have established a relationship between Doppler flow and tendon structure abnormalities, in particular the tendon was characterized by a widening of the structure and the presence of an hypoechoic region [19]. Conversely, other authors have demonstrated that there was no association between Doppler flow and hypoechogenicity, but between Doppler flow and heterogeneous echogenicity, which may explain the lack of correlation between Doppler flow and pain [18].

A unique correlation between Doppler flow imaging and pathological disorders does not exist yet, because it depends on multiple factors including athlete's activity level, the amount of mechanical loading, and operator's ability.

Taking together several studies, it can be stated that Doppler flow is not necessarily a sign of current tendon pain, but it can be associated with tendon stiffness or pathologic abnormalities (Fig. 14.3).

Therefore, Doppler flow can be correlated to asymptomatic changes in tendon morphologic characteristics in response to mechanical load among active athletes [18].

Generally speaking, several researches have affirmed that tendons showing US signs of tendinopathy can be pain-free, and at the same time, US alterations are not necessarily associated with bad prognosis [20].

It can also be said that sensory nerves probably contribute to produce pain in the site of increased vascularity. Even biochemical substances like substance P or glutamate, correlated with neurovascular ingrowth, can be involved in the development of tendon pain [21].

It seems plausible that tendons undergo changes during the asymptomatic phase before the pain threshold is reached and the alterations become symptomatic [22].

However, recent studies point out that intratendinous flow can be increased during exercise both in normal healthy asymptomatic patients and in symptomatic athletes.

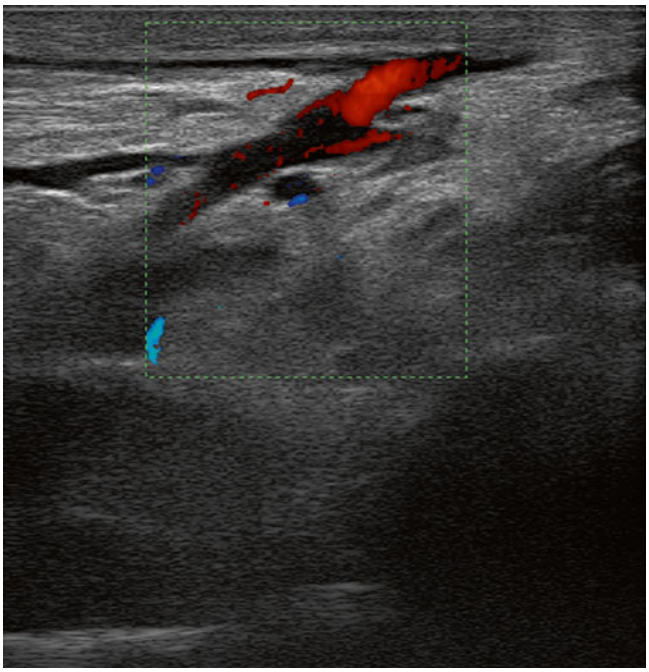


Fig. 14.3 Distal biceps tendon detachment. Echographic image

This suggests that intratendinous flow is not necessarily a sign of overuse injury, but it could be just a normal physiological response to loading [23].

Therefore, it should be underlined that US and color Doppler must be associated to other diagnostic tools, such as MRI and clinical tests, to provide a complete evaluation.

Most muscle injuries successfully respond to conservative management.

A short period of total inactivity is fundamental for a rapid and complete recovery. However, immobilization should not last more than 1 week, so that the adverse effects of immobilization can be limited. The ideal resting period should last 4–6 days after injury, preventing excessive scar formation and reinjuries at the lesion site [24].

This initial rest must be followed by active rehabilitation, as an early mobilization intensifies the regeneration phase as well as it induces angiogenesis. Exercise also facilitates the regenerating myofibers to arrange themselves in the proper orientation [24].

Caution is fundamental when the repair site remains completely or even partially unloaded for a while.

Whereas some kind of structures show benefits from immobilization, like rotator cuff tendons, other tissues like flexor tendons of the hand or patellar tendon can develop fibrous adhesions and a reduction in repair strength, probably due to increased collagen turnover rather than atrophy.

A complete and prolonged removal of load has been demonstrated to limit the healing process of muscles or tendons, largely due to a decreased production of extracellular matrix.

These concepts can be also applied when passive motion is required. Some tendons need long excursions to restore the proper functioning, because they are encased in synovial sheaths. Therefore, in order to maintain the proper tissue sliding, adhesions between the tendon surface and its sheath must be prevented.

At any rate, whatever the tendon-specific function is, continuous passive motion after immobilization or/and after surgery can only be helpful for a complete tendon or muscle healing [25].

Unfortunately, we cannot rely on specific measures for predicting exactly the downtime of the athlete after a serious muscle injury.

It has been supposed by some authors that four simple measures performed during the clinical examination 3–5 days after injury could predict a recovery longer than 4 weeks; these parameters take account of the presence of bruising or hematoma, tenderness to palpation, lack of complete ROM, and pain during isometric limb lengthening [26].

It has been widely demonstrated indeed that athletes who take more than 1 day to walk painless after injury will need more than 3 weeks to return to competition. At the same time, a reduction of ROM worse than 30° determines a prolonged layoff period after injury [24].

Another important healing goal is to take account of all those modifiable risk factors which can induce injury recurrence. These are muscle weakness, fatigue and lack of flexibility, with a strength disparity between eccentric and concentric muscles. As a result, a good rehabilitation program must address all these factors [8].

Rehabilitation protocols vary according to the clinical case, depending on all the information about the event, such as the site, quality, and severity of the injury.

However, some authors have established a protocol valid for track and field activities which is divided into four phases. The first one is the *acute* phase, during which immobilization is required to normalize the gait. The second one is the *range of motion* phase, to regain a painless ROM, starting with concentric and progressing to eccentric training. The third, *functional* one establishes limb loading and returning to running activities, agility drills, and plyometric training. The last stage is the *return-to-play* phase: it is achieved after a variable time, which depends on the severity of the injury as well as the athlete's response to the whole protocol [10].

In conclusion, to simplify the rehabilitation protocols, it can be said that the appropriate recovery time for patients who perform sport-specific training can be based on: (1) the ability to stretch the injured muscle as much as the healthy contralateral muscle and (2) the painless use of the injured muscle in simple movements [24].

References

1. Sikdar S, Wei Q, Cortes N (2014) Dynamic ultrasound imaging applications to quantify musculoskeletal function. Exerc Sport Sci Rev 42(3):126–135
2. Cohen SB, Towers JD, Zoga A et al (2011) Hamstring injuries in professional football players: magnetic resonance imaging correlation with return to play. Sports Health 3(5):423–430
3. Hayashi D, Hamilton B, Guermazi A et al (2012) Traumatic injuries of thigh and calf muscles in athletes: role and clinical relevance of MR imaging and ultrasound. Insights Imaging 3(6):591–601
4. Wang JH, Guo Q, Li B (2012) Tendon biomechanics and mechanobiology – a mini-review of basic concepts and recent advancements. J Hand Ther 25(2):133–140
5. Thornton GM, Hart DA (2011) The interface of mechanical loading and biological variables as they pertain to the development of tendinosis. J Musculoskelet Neuronal Interact 11(2):94–105
6. Weinreb JH, Sheth C, Apostolakos J et al (2014) Tendon structure, disease, and imaging. Muscles Ligaments Tendons J 4(1):66–73
7. Delos D, Maak TG, Rodeo SA (2013) Muscle injuries in athletes: enhancing recovery through scientific understanding and novel therapies. Sports Health 5(4):346–352
8. Heiderscheit BC, Sherry MA, Silder A et al (2010) Hamstring strain injuries: recommendations for diagnosis, rehabilitation and injury prevention. J Orthop Sports Phys Ther 40(2):67–81
9. Hallén A, Ekstrand J (2014) Return to play following muscle injuries in professional footballers. J Sports Sci 32(13):1229–1236
10. Malliaropoulos N, Papacostas E, Kiritsi O et al (2010) Posterior thigh muscle injuries in elite track and field athletes. Am J Sports Med 38(9):1813–1819
11. Connell DA, Schneider-Kolsky ME, Hoving JL et al (2004) Longitudinal study comparing sonographic and MRI assessments of acute and healing hamstring injuries. AJR Am J Roentgenol 183(4):975–984
12. Reurink G, Goudswaard GJ, Tol JL et al (2013) MRI observations at return to play of clinically recovered hamstring injuries. Br J Sports Med. doi:10.1136/bjsports-2013-092450
13. Comin J, Malliaras P, Baquje P et al (2013) Return to competitive play after hamstring injuries involving disruption of the central tendon. Am J Sports Med 41(1):111–115
14. Ekstrand J, Askling C, Magnusson H et al (2013) Return to play after thigh muscle injury in elite football players: implementation and validation of the Munich muscle injury classification. Br J Sports Med 47(12):769–774
15. Sanfilippo JL, Silder A, Sherry MA et al (2013) Hamstring strength and morphology progression after return to sport from injury. Med Sci Sports Exerc 45(3):448–454
16. Silder A, Sherry MA, Sanfilippo J et al (2013) Clinical and morphological changes following 2 rehabilitation programs for acute hamstring strain injuries: a randomized clinical trial. J Orthop Sports Phys Ther 43(5):284–299
17. Kerkhoffs GM, van Es N, Wieldraaijer T et al (2013) Diagnosis and prognosis of acute hamstring injuries in athletes. Knee Surg Sports Traumatol Arthrosc 21(2):500–509
18. Malliaras P, Richards PJ, Garau G et al (2008) Achilles tendon Doppler flow may be associated with mechanical loading among active athletes. Am J Sports Med 36(11):2210–2215
19. Ohberg L, Lorentzon R, Alfredson H (2001) Neovascularisation in Achilles tendons with painful tendinosis but not in normal tendons: an ultrasonographic investigation. Knee Surg Sports Traumatol Arthrosc 9(4):233–238
20. Giombini A, Dragoni S, DI Cesare A et al (2013) Asymptomatic Achilles, patellar, and quadriceps tendinopathy: a longitudinal clinical and ultrasonographic study in elite fencers. Scand J Med Sci Sports 23(3):311–316
21. Bjur D, Alfredson H, Forsgren S (2005) The innervation pattern of the human Achilles tendon: studies of the normal and tendinosis tendon with markers for general and sensory innervation. Cell Tissue Res 320(1):201–206
22. Fredberg U, Bolvig L, Andersen NT (2008) Prophylactic training in asymptomatic soccer players with ultrasonographic abnormalities in Achilles and patellar tendons: the Danish Super League Study. Am J Sports Med 36(3):451–460
23. Boesen AP, Boesen MI, Torp-Pedersen S et al (2012) Associations between abnormal ultrasound color Doppler measures and tendon pain symptoms in badminton players during a season. Am J Sports Med 40(3):548–555
24. Järvinen TA, Järvinen M, Halimo H (2014) Regeneration of injured skeletal muscle after the injury. Muscles Ligaments Tendons J 3(4):337–345
25. Killian ML, Cavinatto L, Galatz LM et al (2012) The role of mechanobiology in tendon healing. J Shoulder Elbow Surg 21(2):228–237
26. Guillodo Y, Bouttier R, Saraux A (2011) Value of sonography combined with clinical assessment to evaluate muscle injury severity in athletes. J Athl Train 46(5):500–504

Shoulder Imaging Evaluation

15

Francesc Soler and Jan C. Fallone

15.1 Introduction

Shoulder pain is a common cause of consultation in primary care medicine. Plain radiography is often a first choice evaluation approach as it identifies direct or indirect signs associated with certain pathologies.

Imaging of the patient with a shoulder trauma begins with a complete radiographic trauma series, consisting of the scapular anteroposterior, scapular lateral, and axillary views. The combination of these images provides important information about possible fractures and position of the humeral head with respect to the glenoid.

CT scans are also helpful to the evaluation of bone structure and accurate quantification of bone lesion with three-dimensional rendered images.

Dynamic shoulder ultrasonography is a low-cost, noninvasive, excellent way of evaluating soft tissue injury and allows a diagnosis in most cases (rotator cuff injury, tendinopathy of the long head of the biceps, bursitis, effusion, calcifications). Convenience and lack of risk make dynamic ultrasonography an excellent imaging tool for evaluating shoulder injuries.

MRI plays a critical role in all orthopedic practices. Recent advances in anatomic and functional imaging highlight the great potential of MRI for musculoskeletal evaluation. MRI is indicated to evaluate rotator cuff tears, instabilities, and tumoral pathologies. MR arthrography, the intra-articular injection of dilute gadolinium before MR imaging, improves sensitivity in the detection of shoulder pathology [1]. Other imaging studies depending on the pathology suspected may be carried out.

F. Soler (✉) • J.C. Fallone
Orthopaedic Surgery Department, EGARSAT,
Garcia Humet, 40, Terrassa, Barcelona 08222, Spain
e-mail: fsoler@egarsat.es; jcfallone@egarsat.es

15.2 Impingement and Rotator Cuff Tears

15.2.1 Subacromial Impingement Syndrome (SIS)

SIS is a painful compression of the supraspinatus tendon, the subacromial-subdeltoid bursa, and the long head of the biceps tendon between the humeral head and the anterior portion of the acromion occurring during abduction and forward elevation of the internally rotated arm [2].

It is a common cause of shoulder pain [3]. Possible etiologies of shoulder pain related to SIS include a spectrum ranging from subacromial bursitis and rotator cuff tendinopathy to partial- and full-thickness rotator cuff tears.

The etiology of rotator cuff disease has long been debated, and the cause is likely multifactorial [4, 5]. Combinations of intrinsic and extrinsic factors play an important role in rotator cuff injury development. Secondary causes of impingement include tuberosity fracture nonunion or malunion, a mobile os acromiale, calcific tendinitis, instability including superior labrum anterior posterior (SLAP), muscle imbalance with increased forces centering the humeral head under the coracoacromial arch, and supraspinatus hypertrophy due to occupation or sports activities and iatrogenic factors.

Coracoid impingement is a potential cause of anterior shoulder pain, particularly with movements requiring forward flexion, internal rotation, and horizontal adduction of the humerus [6]. Subcoracoid pain can occur as a result of compression of the subscapularis tendon or biceps tendon between the bony structures of the lesser tuberosity and the coracoid process [7, 8].

Accurate diagnosis and effective treatment require a thoughtful and thorough history and physical examination as well as appropriate imaging.

15.2.1.1 Imaging of Impingement Syndrome: Imaging Modalities

Imaging techniques to evaluate impingement syndrome and rotator cuff pathologies include radiographs, ultrasound

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_15, © Springer International Publishing Switzerland 2016

(US), arthrography, computed tomography (CT), magnetic resonance imaging (MRI), and magnetic resonance arthrography (MRA). Ultrasound is used frequently to get an overview of the rotator cuff pathology. Arthrography is only rarely used as a single method. When available, a combination of MRI following the distension of the joint capsule with fluid is a widely used practice.

Radiography

Imaging of the shoulder should always start with radiographs. Three projections are routinely obtained: A "true AP" view is needed with 30° rotation of the opposite shoulder away from the screen holder and 10–15° caudal angulation of the tube. This view provides a tangent projection of the glenoid.

The AP view provides information regarding the relationship of the humeral head to the glenoid and to the inferior part of the acromion. The subacromial space, measured by the acromiohumeral interval (AHI), closely reflects the thickness of the rotator cuff tendons. The normal AHI measures 9–10 mm with a range from 8 to 12 mm. An AHI of less than 7 mm indicates pathologic thinning of the supraspinatus tendon due to degeneration, or partial- or small full-thickness tear.

A lateral view of the acromion for evaluation of the acromial shape is possible with an "outlet view," a modified transscapular lateral view at a 5–10° caudal angle of the central beam to compensate for the down slope of the acromion from medial to lateral. The "outlet view" demonstrates the bone appositions at the undersurface of the acromion, the AP extension of those appositions, and the congruity of the relation of the coracoacromial arch and the humeral head.

The "Rockwood" view is an anteroposterior projection at a 30° caudal angle of the X-ray beam [9]. The anterior extension of bone appositions at the anterior acromion is best seen on this view. This projection outlines the down-bowing curvature of the lateral clavicle and the acromioclavicular joint from an anterosuperior point of view. A tangent line is drawn caudally along the cortex of the clavicle from medial to lateral. The normal acromion does not override the lateral extension of this line.

Ultrasound

Ultrasound (US) is a helpful imaging tool in the evaluation of the musculoskeletal system. The US scan is a valuable diagnostic technique for rotator cuff complete or incomplete ruptures [10]. It is widely used for the evaluation of the rotator cuff but is largely evaluator dependent. US technique for shoulder examination depends on patient positioning, scanning protocol for every tendon and anatomic part, and dynamic imaging. The examination is performed with the patient seated. Imaging begins with the patient's arm adducted and in the neutral position and continues as the arm is internally rotated and placed behind the back. Transducers with a frequency range of 9–13 MHz are used, providing an in plane resolution of 200–400 μm and a section thickness of 0.5–1.0 mm.

Major diagnostic criteria for rotator cuff tears are a well-defined discontinuity within the normal echogenic cuff substance, which is usually visible as a hypoechoic focus within the cuff, absence or nonvisualization of the cuff, indicating a large tear, and an echogenic focus within the cuff [11].

Rotator cuff ultrasound was reported to be less reliable than MRI and to have a limited role in the evaluation of rotator cuff pathologies [12].

Magnetic Resonance Imaging (MRI)

Typical sequences for routine shoulder imaging include axial, oblique coronal, and oblique sagittal fat-suppressed PD-weighted sequences, oblique coronal T2-weighted FSE sequences, and on sequences in which fat saturation can be suboptimal with low field strength magnets, oblique coronal and oblique sagittal STIR sequences. Fluid-sensitive sequences are used to identify marrow abnormalities, to confirm and further define tendon abnormalities visible on PD-weighted magnetic resonance images, and to visualize bursal distension and joint effusion. The fibrocartilaginous labrum is evaluated on PD-weighted sequences, and the cuff is best evaluated on PD- and T2-weighted STIR sequences. Muscle atrophy is assessed on PD-weighted magnetic resonance images, whereas muscle edema is best seen on T2-weighted and STIR sequences.

MR Arthrography

MR arthrography enhances the accuracy of MRI in the evaluation of rotator cuff tendons [13]. It is the most sensitive and specific technique for diagnosing both full- and partial-thickness rotator cuff tears. Ultrasound and MRI are comparable in both sensitivity and specificity [14]. Conventional MRI of the shoulder is limited in depicting intra-articular structures when insufficient fluid is present to outline their structure. Fat-suppressed sequences further improve the diagnostic performance of MR arthrography, especially in the differentiation of partial- from full-thickness cuff tears and in the detection of small partial tears of the inferior tendon surface [15].

A routine protocol uses fat-suppressed T1-weighted images at a 3-mm slice thickness in all three planes (coronal oblique, sagittal oblique, and axial) postinjection, if gadolinium is added to the injected fluid. If only saline is injected, proton density–weighted images with fat suppression are acquired. Three-dimensional (3D) gradient echo acquisitions with T2 contrast and fat suppression have also proved to be very useful.

The abduction external rotation position (ABER) in which the patient's arm is abducted and externally rotated is

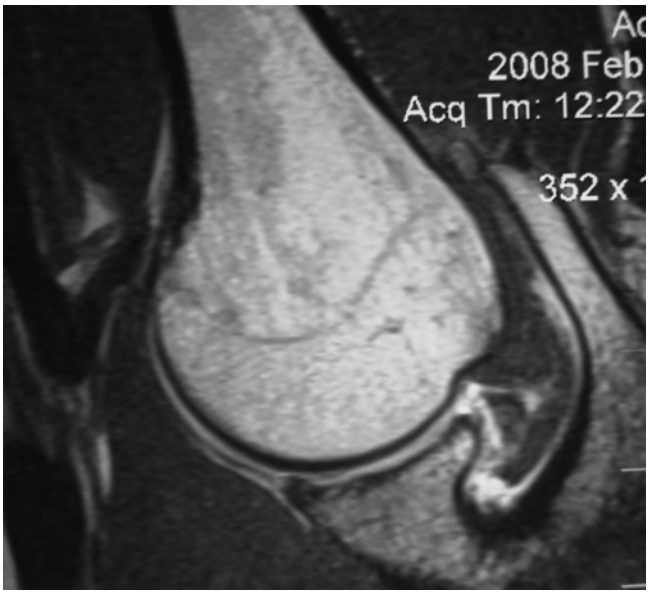


Fig. 15.1 MRI in ABER position showing partial articular rotator cuff tear

reported to enhance the detectability of partial-thickness tears of the undersurface of the rotator cuff, especially in the infraspinatus tendon during MR arthrography [16]. The ABER position and external rotation alone have been shown to optimize the visualization of the biceps labral complex and glenohumeral ligaments [17]. The ABER position should be included in the imaging protocol in athletes suffering shoulder pain from throwing [18] (Fig. 15.1).

A study suggests that CT and MR arthrography have similar diagnostic performance for the evaluation of rotator cuff tendon tears. There was no statistically significant difference in sensitivity and specificity between CT arthrography and MR arthrography in depiction of rotator cuff lesions [19].

15.2.1.2 Imaging Findings in Impingement Syndrome and Rotator Cuff Tears

Radiographs should be obtained to evaluate possible bony abnormalities of the coracoacromial arch. Routine radiographs include AP and Grashey views (AP radiograph of the shoulder in the plane of the scapula) as well as outlet and axillary views. The outlet view provides visualization of acromial morphology, and the axillary view best demonstrates evidence of os acromiale, which may lead to secondary impingement.

Acromioclavicular joint osteoarthritis with inferior osteophyte formation, acromial enthesophytes or sclerosis, and cystic changes of the humeral head are the more common radiographic findings related to impingement. However, all of these findings may be present in asymptomatic subjects, making the relationship of such findings to the diagnosis of impingement controversial.

MRI provides a detail of potential sites of subacromial impingement through the supraspinatus outlet. Ossification of the coracoacromial ligament (CAL) or the presence of a subacromial spur can be best identified in the sagittal oblique plane; however, differentiation of a pathologic spur and the normal CAL can be difficult. MRI also may demonstrate findings of subacromial-subdeltoid bursitis. Findings that indicate this condition include bursal thickness >3 mm, the presence of fluid medial to the acromioclavicular joint, and the presence of fluid in the anterior aspect of the bursa [20].

Subacromial Bursitis-Bursal Effusion

Fluid distending the subacromial-subdeltoid bursa is a nonspecific finding, as it may be encountered in association with subacromial impingement, partial or complete rotator cuff tears, and calcifying tendinitis.

During impingement of the supraspinatus tendon, the subacromial-subdeltoid bursa becomes compressed between the greater tuberosity of the humeral head and the anterior portion of the acromion. This chronic compression can result in an inflammatory reaction of the bursal synovium and secretion of fluid into the bursa. Fluid is not detected in the normal bursa [21]. Fluid in the bursa is recognized on T2-weighted images as the increased signal intensity of the subacromial-subdeltoid bursa indicates local bursal inflammation [22].

Tendinosis

A normal supraspinatus tendon should exhibit low signal intensity on all pulse sequences. There is general agreement that a focal area of increased signal intensity on a T1-weighted image without increased signal intensity on a T2-weighted image and without thickening or thinning of the tendon is due to the magic angle artifact or is without clinical relevance.

A tendon with focal or diffuse increased signal intensity on proton density-weighted images without further increase of signal intensity on T2-weighted images and an indistinct margin at the articular side of the supraspinatus tendon corresponds to eosinophilic, fibrillar, and mucoid degeneration and scarring. Tendons with areas of increased signal intensity on T2-weighted images are associated with severe degeneration and disruption of the supraspinatus tendon [23] and have increased signal intensity also on STIR and T1-weighted images. No focal or linear area of water-equivalent signal intensity is seen.

Tendinosis in many cases is a result of impingement syndrome. Fluid collections can be present in the subacromial-subdeltoid bursa, findings similar to that of inflammation. This early stage of rotator cuff disease is frequently called tendinitis.

Partial-Thickness Tears

Partial-thickness tears of the rotator cuff can be articular-sided, bursal-sided, intratendinous, or a combination thereof. Partial-thickness tears are subdivided based on location and thickness of tear.

Throwing athletes may show a predilection for articular surface tears [24]. Most tears affect the supraspinatus tendon. The infraspinatus, subscapularis, and teres minor tendons are much less commonly involved [25, 26].

Isolated intratendinous tearing of the supraspinatus tendon is rare, and most cases are associated with bursal or joint side cuff tears [10]. If fluid is present, water-equivalent signal intensity is seen in the area of the defect (Fig. 15.2). Partial-thickness tears are usually circumscribed and rarely exceed 10 mm, measured in the AP or sagittal direction. They can be missed by MRI and arthrography [27].

Consistent differentiation of tendinosis, partial-thickness tears, and full-thickness tears of the rotator cuff tendons is more difficult on non-enhanced MR images when no fluid is present in the joint cavity or in the bursa [28].

Partial-thickness tears or a horizontal splitting of the tendon beginning from the joint surface or the bursal surface is only detectable if there is fluid signal present in the defect or between the tendon layers. Frequently this fluid is absent, and sensitivity for partial-thickness tears of non-enhanced MR imaging of the shoulder is low.

Unfortunately partial-thickness tears originating from the bursal surface of the tendon are still missed, as long as there is no communication of the joint cavity with the bursa. MR arthrography can solve the problem of partial-thickness tears originating from the joint surface of the tendon.

Rotator Cuff Tears

Small full-thickness tears will enlarge over time. When the tear is only a few millimeters, the humeral head remains in place and keeps its relationship to the glenoid. In tears greater than 10 mm in diameter, the humeral head is slowly moved upward by those shoulder muscles, centering the humeral head underneath the coracoacromial arch.

In cases of massive tears, which exceed 20 mm in diameter, the infraspinatus tendon in addition to the supraspinatus tendon and, in rare cases, the subscapularis tendon become torn and insufficient. The humeral head migrates upward and finally articulates with the undersurface of the acromion.

Goutallier et al. [29] introduced the concept of fatty degeneration of the rotator cuff in 1989. They devised a staging system and noted that degeneration of the rotator cuff muscles was associated with rotator cuff tears.

In the initial iteration of the Goutallier staging system, axial CT was used to evaluate the supraspinatus, subscapularis, and infraspinatus muscles [30, 31]. The supraspinatus was evaluated on the axial image with the most muscle surface area (approximately 5 mm above the humeral head). The subscapularis and infraspinatus were evaluated superiorly at the level of the tip of the coracoid and inferiorly at the level of the lower glenohumeral joint. The muscles were then assigned a stage: Stage 0 is normal muscle. Stage 1 some fatty streaks. Stage 2 is less than 50 % fatty muscle atrophy. Stage 3 is 50 % fatty muscle atrophy. Stage 4 the fatty muscle atrophy is greater than 50 % (Fig. 15.3).

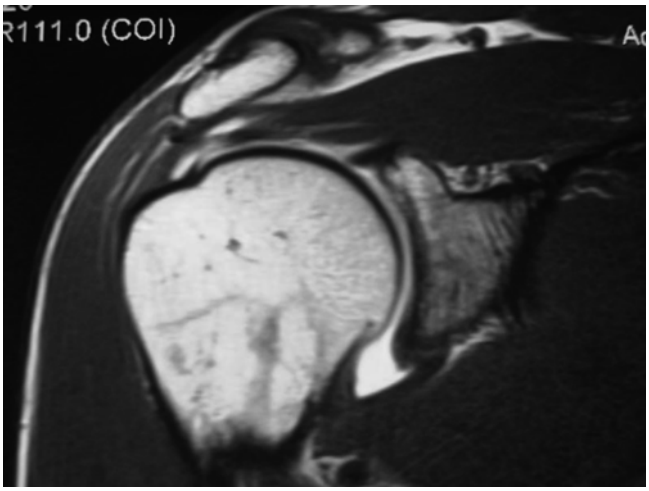


Fig. 15.2 Partial intra-articular rotator cuff tear. An MRI image

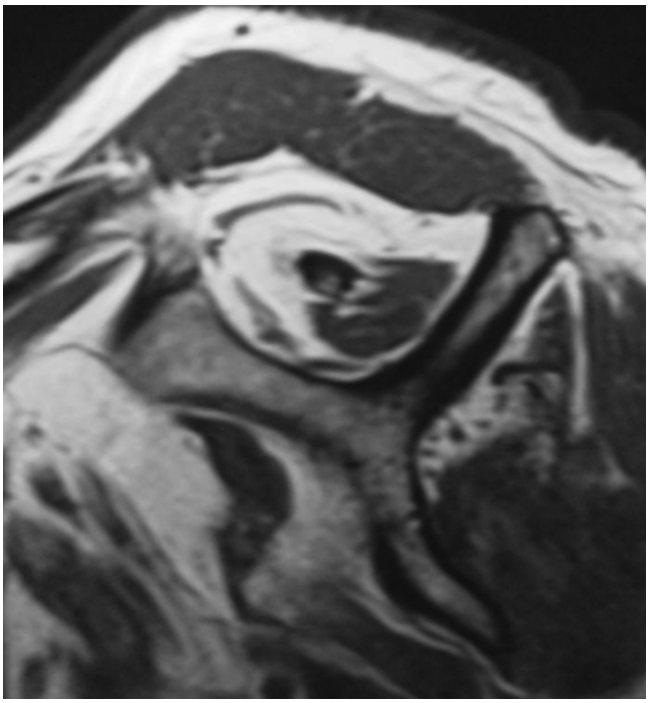


Fig. 15.3 Goutallier stage 4

Ultrasonography can be used as the primary diagnostic imaging modality for fatty changes in rotator cuff muscles.

Rotator cuff atrophy has generally been evaluated using either an occupation ratio, as described by Thomazeau et al. [32], or the tangent sign introduced by Zanetti et al. [33]. The occupation ratio was defined as the surface area supraspinatus muscle/surface area supraspinatus fossa [28]. The tangent sign is negative if the supraspinatus crosses a line

between the superior aspect of the coracoid and the superior border of the scapular spine. MRI evaluation is performed using the most lateral image where the scapular spine is in contact with the body of the scapula [34].

Subscapularis Tears and Biceps Tendon Lesions

Lafosse et al. [35] proposed a subscapularis tear classification system based on CT findings and intraoperative assessment. Four types of subscapularis tendon lesions are described: Type I involves partial lesion of superior one-third of the subscapularis tendon. Type II involves complete lesion of superior one-third of the tendon. Type III tears represent complete lesion of superior two-thirds of the tendon rupture. Type IV are those with complete lesion of the tendon but head centered and fatty degeneration classified as less than or equal to Goutallier stage III.

Only 2 % of rotator cuff tears predominantly or exclusively involve the subscapularis tendon [36].

Imaging studies are also useful for evaluating subscapularis tears and concomitant shoulder pathology. Typically, routine radiographic evaluation is normal in the setting of a subscapularis tear. However, AP, outlet, and axillary lateral radiographs should be carefully assessed for anterior humeral head subluxation, proximal humeral head migration, and/or the presence of glenohumeral degenerative changes, all of which could indicate more chronic rotator cuff tear pathology.

Ultrasonography offers the ability for dynamic, noninvasive, real-time, and even bilateral comparison of the rotator cuff tendons. It is also less expensive than MRI. Ultrasound sensitivity increases with the size of the rotator cuff tear, reaching 100 % for massive full-thickness tears that involve the subscapularis.

CT arthrography, magnetic resonance arthrography, and routine MRI are commonly used to evaluate the subscapularis. On CT arthrography, extravasation of intra-articular contrast onto the lesser tuberosity is seen in the setting of an isolated subscapularis tear [37]. Also, it will show communication with air reaching from the glenohumeral joint to the bicipital groove. A medial dislocation of the biceps tendon is indicative of a complete rupture of the subscapularis tendon. Along with the coracohumeral and transverse humeral ligaments, which are parts of the rotator cuff interval, the subscapularis tendon is a major stabilizer of the long biceps tendon. Degeneration or disruption of the subscapularis tendon has been reported to be a common predisposing factor to medial dislocation of the biceps tendon [38, 39].

On MRI, in the presence of a tear of the subscapularis tendon, the contours of the tendon are poorly defined and of abnormally high signal intensity on T2-weighted images. Discontinuity and frank retraction is seen in 78 % of patients. Subscapularis tears are well detected in the sagittal oblique plane with the deltoid muscle seen in direct apposition to the anterior humerus and the lesser tuberosity [29].

Subscapularis tears are often missed on MRI. Complete and appropriate image sequencing, including T2-weighted axial images, parasagittal images, and the use of magnetic resonance arthrography, allows for improved detection of subscapularis tears [40–43].

Incomplete tears of the subscapularis tendon occur in conjunction with small- or medium-sized tears of the supraspinatus tendon. This combination is common in older patients, and most subscapularis lesions are incomplete tears on the articular side [44].

15.3 Instability

The glenohumeral joint is the most frequently dislocated major joint, and most cases involve an anterior dislocation. Glenohumeral instability is common in young athletes [45]. Athletes competing in contact sports are at especially high risk of recurrent instability [46, 47]. Almost half of all anterior shoulder dislocations occur in persons aged 15–29 years, and the incidence is nearly three times higher in males than in females [48]. This diagnosis encompasses a large spectrum of injury, from microinstability to glenohumeral dislocation. The imaging evaluation of an athlete with glenohumeral instability includes diverse modalities: radiography, CT, and MRI. Many missed dislocations result to an incomplete radiographic evaluation [25, 49, 50].

15.3.1 Shoulder Imaging Techniques

15.3.1.1 Radiography

X-ray evaluation is always recommended. Standard imaging of the shoulder should include a scapular AP view with the humerus in internal and external rotation as well as an axillary view of the shoulder. The contour of the anteroinferior glenoid should be readily visible on both the AP and the axillary views; a blurry or poorly defined contour suggests bone loss, especially in the setting of recurrent instability. Radiographs will also identify an associated Hill-Sachs lesion of the humeral head as well as greater tuberosity fracture or other associated fractures. Additional views that are particularly useful in assessing the unstable shoulder include the West Point axillary view, the Stryker notch view, and the Bernageau view. When X-rays are negative, more precise exploratory methods such as CT arthrography, MRI, or MR arthrography are usually indicated.

15.3.1.2 CT and CT Arthrography

Axial CT scans are helpful in determining the extent of glenoid bone loss and humeral head impaction. Accurate quantification of bone loss is often difficult to achieve. However, its efficiency seems to be improved by such technical

refinements as helical acquisition, which enables high-quality multiplanar and three-dimensional (3D) reconstructions. Three-dimensional rendered images are preferred for defining the orientation and degree of glenoid bone loss and the extent of a Hill-Sachs lesion [26, 51, 52].

15.3.1.3 MRI and MR Arthrography

The value of MRI in shoulder disorders is continuously changing frequently due to the constant improvements of equipment and pulse sequences. MRI is very sensitive for confirming the clinical suspicion of a labral tear. MRI also identifies associated injuries to the rotator cuff and cartilage.

The image of the different anatomical structures of the shoulder varies according to the different pulse sequences used. The best results are usually obtained with the fast spin-echo moderately T2-weighted sequence associated with a fat suppression signal (Fat Sat). The echo train length must not be set too high in order to limit blurring artifacts.

Intra-articular gadolinium injection is used because of its usefulness in defining the extent of labral tears. This technique improves the analysis of the labrum, ligaments, and capsular structures as it increases intra-articular contrast and distends the capsule.

Two different techniques have been adopted [53]. Firstly, T1-weighted sequences are obtained after intra-articular gadolinium injection, diluted either with physiological serum or iodinated contrast media. This technique is widely used due to its excellent ability to detect articular abnormalities. In the second procedure, iodinated contrast media (or physiological serum) is injected solely and fast spin-echo T2-weighted sequences are obtained. The last method offers substantial advantages as it combines two widely used methods (arthrography and MRI) and does not require any complex manipulation of contrast media. It does not involve intra-articular gadolinium injection. Moreover, examinations are of high quality, and images are rapidly available. Finally, T2-weighted sequences used in this technique constitute the basis of shoulder exploration [54].

To put stress on the anteroinferior labrocapsular structures and therefore to obtain a higher definition of their lesions, acquisition with abduction and external rotation of the arm (ABER) or in the apprehension test position can be performed [54, 55]. ABER positioning is also recommended for exploring the painful athlete's shoulder [18].

15.3.2 Imaging Findings in Instability

15.3.2.1 Anterior Instability

Unidirectional anterior shoulder instability usually begins with a discrete traumatic event. Imaging is fundamental in exploring instabilities.

In anterior instability, a standard X-ray evaluation is usually sufficient. The three anteroposterior views of the glenohumeral joint (external, neutral, and internal rotation), apical oblique view, and the Bernageau view (glenoid profile) are standard procedures.

In order to identify bony lesions, CT scan may eventually be used. CT arthrography or MRI gives more specific details as to the severity of the lesions, particularly soft tissue alterations.

In subtle forms of instability, diagnosis or instability direction is not clearly assessed clinically, and standard X-ray evaluation is usually normal. In this case, further imaging with CT arthrography, MRI, or MR arthrography are recommended to confirm the diagnosis of instability and to evaluate its direction. The technique of choice is undoubtedly MR arthrography; however, the imaging technique used depends mostly on several factors, principally quality, performance, and access to the machines.

15.3.2.2 Bony Lesions: Humeral Head Defect

Bony lesions associated with anterior instability are mainly represented by the Hill-Sachs lesion. Depending upon the direction of the displacement, the impaction may be observed higher or lower on the humeral head.

Reverse Hill-Sachs lesions are associated with posterior shoulder dislocation.

Humeral head defect is usually outlined by the anteroposterior (AP) view of the shoulder with the arm in internal rotation or by the apical oblique view [56]. Other less applied methods have been described such as the "Stryker notch view."

CT scan or CT arthrographies are more efficient than the abovementioned methods to diagnose these abnormalities, as they are more efficient in detecting small bony defects. Finally, MRI is a valuable method to detect humeral head fractures, especially if performed shortly after the accident. These cases show bone marrow edema as well as the bony compression fracture at the posterolateral aspect of the humeral head. However, when bone marrow edema is no longer visible, small defects are less detectable with MRI than with CT scan.

15.3.2.3 Anteroinferior Lesions of the Glenoid Rim

The most common lesion is an anteroinferior capsulolabral avulsion from the glenoid rim [57, 58], typically with associated capsular attenuation. In addition, acute fracture and/or attritional glenoid bone loss may contribute to recurrent instability by altering the glenohumeral contact area and the function of the static glenohumeral restraints [59, 60].

In general, plain radiographs are moderately accurate at demonstrating glenoid bone loss [61]. A bony shadow or displaced bony Bankart fragment may be visualized on a standard AP view or in other projections parallel to the

glenoid face, such as the axillary or glenoid profile view [62]. The highest yield projections, however, are angled relative to the glenoid face, such as the apical oblique [18]. The Stryker notch view and AP view with the humerus in internal rotation should also be obtained, given their utility in visualizing potential Hill-Sachs lesions on the humeral side [63].

Beyond standard radiography, MRI or magnetic resonance arthrography (MRA) studies may suggest the degree of bone loss in the most lateral glenoid cut on the sagittal oblique series. However, the current standard imaging modality for quantifying glenoid bone loss is CT.

Standard CT scans can be used to estimate bone loss and detect rim fracture fragments; Three-dimensionally reconstructed scans is the most reliable modality for predicting glenoid bone loss [64]. CT scan can also be performed with digital subtraction of the humeral head. By use of this modality, glenoid osseous deficiency is quantified as a percentage of the normal inferior glenoid surface area [65]. A best-fit circle is drawn on the inferior two-thirds of the glenoid image, which has been shown to be a consistent anatomic configuration [66]. The amount of bone missing from the circle, as a percentage of the total surface area of the inferior circle, is then determined with digital measurements.

15.3.2.4 Capsulolabral Detachments

The ALPSA (anterior labroligamentous periosteal sleeve avulsion) lesion is an avulsion of the anteroinferior glenoid labrum with an intact scapular periosteum. The ALPSA lesion differs from the Bankart lesion in that the ALPSA lesion has an intact anterior scapular periosteum allowing the labroligamentous structures to displace medially and rotate inferiorly on the scapular neck. In the Bankart lesion, the anterior scapular periosteum rupture results in displacement of the labrum and attached ligaments anterior to the glenoid rim.

CT arthrography, MRI, or MR arthrography can show different pathological patterns whose relationship with arthroscopic data can be difficult to establish. A fibrous scar may mimic an intact labrum on CT arthrography, and a wear and tear of the anteroinferior glenoid rim can be observed with a pseudo-intact labrum. In this case, MRI with intravenous gadolinium injection can show a tissue enhancement at the base of the pseudolabrum indicating the presence of fibrous tissues.

An irregular or severed anteroinferior recess is due to a fibrous scar and is usually well defined when the joint is filled with contrast media. Therefore, it can be demonstrated with CT arthrography or MR arthrography but is barely visible with MRI. A slight periosteal reaction is due to an extension of the tearing of the capsule at its insertion together with a periosteal tearing. This process is better observed with CT or CT arthrography.

The value of the different imaging methods in the diagnosis of labral tears remains controversial. The data obtained from different studies supports the use of indirect MRA as standard practice in patients with shoulder instability due to suspected labral pathology where further investigative imaging is indicated [67–69].

It is difficult to assess whether standard MRI is more efficient than CT arthrography, when their sensitivity and specificity vary, respectively, from 73 to 93 % and from 73 to 80 % [70]. Taking into account these large variations in accuracy, many authors advocate the use of MR arthrography as the most reliable method with a 95 % accuracy to evaluate labrocapsular structures and to detect labral tears [71].

Traumatic shoulder dislocation or subluxation may result in avulsion of the IGHL from its humeral attachment (HAGL lesion). Although both traumatic lesions occur with the arm in hyperabduction, HAGL lesions are more likely to occur with hyperabduction and external rotation [72]. HAGL lesions result in incompetence of the IGHL complex, which leads to glenohumeral instability, particularly in abduction and external rotation.

True AP radiographs with the shoulder in neutral and internal rotation are obtained along with scapular Y and axillary views. These images are used to evaluate for concentric glenohumeral alignment, impaction fracture of the humeral head (Hill-Sachs lesion), glenoid rim fracture, and glenoid hypoplasia. Scalloping of the medial aspect of the humeral neck on the AP view may be associated with an HAGL lesion [73]. The Garth view of the glenohumeral joint can be used to visualize the bony fragment [74].

MRI is the imaging modality of choice for the assessment of a suspected HAGL lesion (Fig. 15.4). The IGHL complex is

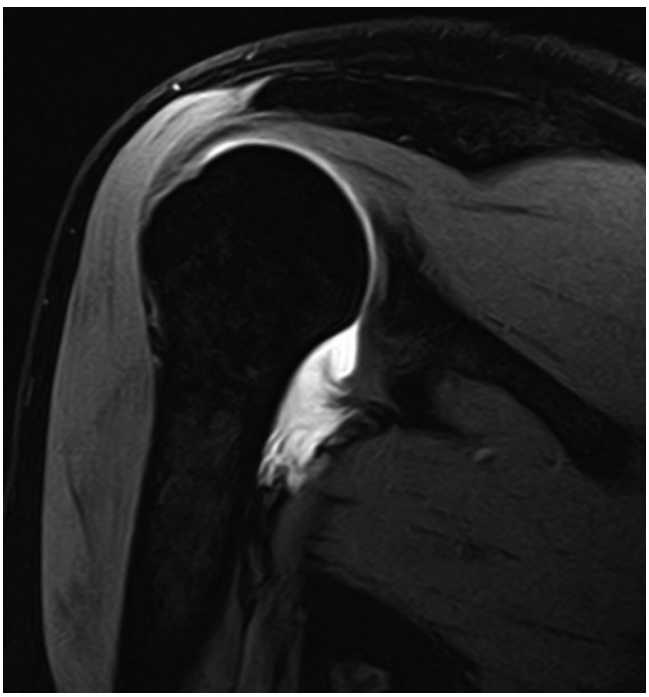


Fig. 15.4 HAGL lesion in MRI arthrography

best evaluated on coronal oblique or sagittal oblique T2-weighted fat-suppressed magnetic resonance images [65]. Normal IGHL anatomy appears as a U-shaped structure due to fluid distension of the axillary pouch. The presence of an HAGL lesion can cause the normally U-shaped axillary pouch to appear J-shaped as a result of extravasation of contrast or joint fluid across the torn capsule and ligament [65, 75]. Detection of an HAGL lesion is facilitated by the presence of glenohumeral joint effusion or by injection of intra-articular contrast dye. Chronic HAGL lesions may be difficult to visualize on magnetic resonance images because the torn edge on the humeral side may scar down to the capsule. No study has determined the accuracy of standard MRI or magnetic resonance arthrography in detecting the presence of HAGL lesion.

15.3.2.5 Posterior Instabilities

Radiographs

Plain radiographs of the shoulder should include true anteroposterior views in neutral, internal, and external rotation, a transscapular view or Y view, and an axillary view. These views are needed to ensure that the joint is correctly located to evaluate the posterior glenoid rim and to look for impaction fractures of the humeral head. Diagnosis can be suggested on the AP view of the glenohumeral joint when the humeral head seems to be fixed in internal rotation with a pseudo-widening of the articular space. On the contrary, posterior dislocation is suggested when the articular space cannot be visualized without bony overlapping.

In addition to humeral head position, these studies demonstrate glenoid rim morphology (hypoplasia, excessive retroversion, and/or fracture of the posterior glenoid rim). However, most individuals with recurrent posterior instability do not have bony abnormalities. For those with a volitional component, dynamic radiographs can confirm the diagnosis.

Multiplanar Imaging

Computed tomography (CT) or magnetic resonance imaging (MRI) is essential to assess the version and morphology of the glenoid. These tests also help detect subtle anterior humeral head defects and glenoid fractures. Contrast can enhance the ability to evaluate the posterior labrum and capsule, particularly with injuries such as capsulolabral disruptions or lateral capsular injuries [76]. Contrast also enhances assessment of the superior labrum.

Although the gadolinium-enhanced magnetic resonance arthrogram provides excellent soft tissue detail, we think that a CT scan with intra-articular contrast provides the best information with regard to bony anatomy and articular orientation. CT is superior in its ability to determine the glenoid morphology as well as the degree of glenoid retroversion. Glenoid retroversion is best measured on axial CT scan images through the midglenoid.

15.3.2.6 Superior Labral Lesions

Injuries to the superior labrum and biceps tendon origin were further characterized and classified by Snyder et al. in 1990 [77] and remains the most widely recognized [78]. He described four major variants [79].

Imaging

Radiographic evaluation includes standard views of the shoulder (AP, scapular AP, axillary, outlet). These views are helpful primarily in identifying other potential sources of shoulder pain.

MRI is the preferred imaging technique for patients with suspected SLAP tears. Multiplanar images are obtained in the coronal, parasagittal, and axial planes. Spinoglenoid cysts are readily apparent on MRI scans. Distinguishing SLAP tears from normal anatomy is difficult because of the variable anatomy of the anterosuperior labrum and the medial insertion of the superior labrum. The accuracy of MRI may be improved with the shoulder positioned in abduction and external rotation. Findings suggestive of a type II SLAP tear include high signal intensity and intra-articular contrast extension under the superior labrum/biceps root on coronal images. Other type II-specific findings include laterally curved high signal intensity in the superior labrum, concomitant anterosuperior labral tear, and anteroposterior extension of high signal intensity at the superior labrum/biceps root on axial images [80, 81]. A study suggests that SLAP tears are often incorrectly diagnosed based on MRI evaluation, with MRI providing a high level of sensitivity and low level of specificity. On the basis of the results of this study, conventional MRI is not a suitable test to accurately evaluate the biceps labral complex for the presence of a SLAP tear [82].

Despite advances in MRI techniques, the accuracy of detecting SLAP tears remains variable.

References

1. Ito Y, Sakai T, TomNakao Y, Inui K, Koike T, Nakatsuchi T, Takaoka KOH (2005) Computerized assessment of Bankart lesions under tension with magnetic resonance arthrography. J Shoulder Elbow Surg 14(3):247–251
2. Neer CS Jr (1972) Anterior acromioplasty for the chronic impingement syndrome in the shoulder: a preliminary report. J Bone Joint Surg Am 54:41–50
3. Michener LA, McClure PW, Karduna AR (2003) Anatomical and biomechanical mechanisms of subacromial impingement syndrome. Clin Biomech (Bristol, Avon) 18(5):369–379
4. Fukuda H, Hamada K, Nakajima T, Tomonaga A (1994) Pathology and pathogenesis of the intratendinous tearing of the rotator cuff viewed from en bloc histologic sections. Clin Orthop Relat Res 304:60–67
5. Meislin RJ, Sperling JW, Stitik TP (2005) Persistent shoulder pain: epidemiology, pathophysiology, and diagnosis. Am J Orthop (Belle Mead NJ) 34(12 Suppl):5–9

6. Ferrick MR (2000) Coracoid impingement: a case report and review of the literature. Am J Sports Med 28(1):117–119
7. Dumontier C, Sautet A, Gagey O, Apoil A (1999) Rotator interval lesions and their relation to coracoid impingement syndrome. J Shoulder Elbow Surg 8(2):130–135
8. Garofalo R, Conti M, Massazza G, Cesari E, Vinci E, Castagna A (2011) Subcoracoid impingement syndrome: a painful shoulder condition related to different pathologic factors. Musculoskelet Surg 95(Suppl 1):S25–S29. doi:10.1007/s12306-011-0142-7
9. Rockwood CA Jr, Lyons FR (1993) Shoulder impingement syndrome: diagnosis, radiographic evaluation and treatment with a modified Neer acromioplasty. J Bone Joint Surg Am 75:409–424
10. Pavic R, Margetic P, Bensic M, Brnadic RL (2013) Diagnostic value of US, MR and MR arthrography in shoulder instability. Injury 44(Suppl 3):S26–S32
11. Soble MG, Kaye AD, Guay RC (1989) Rotator cuff tear: clinical experience with sonographic detection. Radiology 173:319–321
12. Nelson MC, Leather GP, Nirschl RP, Pettrone FA, Freedman MT (1991) Evaluation of the painful shoulder. A prospective comparison of magnetic resonance imaging, computerized tomographic arthrography, ultrasonography, and operative findings. J Bone Joint Surg Am 73:707–716
13. Hodler J, Kursunoglu-Brahme S, Snyder SJ, Cervilla V, Karzel RP, Schweitzer ME, Flannigan BD, Resnick D (1992) Rotator cuff disease: assessment with MR arthrography versus standard MR imaging in 36 patients with arthroscopic confirmation. Radiology 182:431–436
14. de Jesus JO, Parker L, Frangos AJ, Nazarian LN (2009) Accuracy of MRI, MR arthrography, and ultrasound in the diagnosis of rotator cuff tears: a meta-analysis. AJR Am J Roentgenol 192(6):1701–1707
15. Palmer WE, Brown JH, Rosenthal DI (1993) Rotator cuff: evaluation with fat-suppressed MR arthrography. Radiology 188:683–687
16. Tirman PF, Bost FW, Steinbach LS, Mall JC, Peterfy CG, Sampson TG, Sheehan WE, Forbes JR, Genant HK (1994) MR arthrographic depiction of tears of the rotator cuff: benefit of abduction and external rotation of the arm. Radiology 192:851–856
17. Kwak SM, Brown RR, Trudell D, Resnick D (1998) Glenohumeral joint: comparison of shoulder positions at MR arthrography. Radiology 208:375–380
18. Roger B, Skaf A, Hooper AW, Lektrakul N, Yeh L, Resnick D (1999) Imaging findings in the dominant shoulder of throwing athletes: comparison of radiography, arthrography, CT arthrography, and MR arthrography with arthroscopic correlation. AJR Am J Roentgenol 172:1371–1380
19. Omoumi P, Bafort AC, Dubuc JE, Malghem J, Vande Berg BC, Lecouvet FE (2012) Evaluation of rotator cuff tendon tears: comparison of multidetector CT arthrography and 1.5-T MR arthrography. Radiology 264(3):812–822
20. White EA, Schweitzer ME, Haims AH (2006) Range of normal and abnormal subacromial/subdeltoid bursa fluid. J Comput Assist Tomogr 30(2):316–320
21. Bureau NJ, Dussault RG, Keats TE (1996) Imaging of bursae around the shoulder joint. Skeletal Radiol 25:513–517
22. Schraner AB, Major NM (1999) MR imaging of the subcoracoid bursa. AJR Am J Roentgenol 172:1567–1571
23. Kjellin I, Ho CP, Cervilla V, Haghighi P, Kerr R, Vangness CT, Friedman RJ, Trudell D, Resnick D (1991) Alterations in the supraspinatus tendon at MR imaging: correlation with histopathologic findings in cadavers. Radiology 181:837–841
24. Andrews JR, Broussard TS, Carson WG (1985) Arthroscopy of the shoulder in the management of partial tears of the rotator cuff: a preliminary report. Arthroscopy 1:117–122
25. Tasu JP, Miquel A, Rocher L, Molina V, Gagey O, Blery M (2001) MR evaluation of factors predicting the development of rotator cuff tears. J Comput Assist Tomogr 25:159–163
26. Gartsman GM, Milne JC (1995) Articular surface partial thickness rotator cuff tears. J Shoulder Elbow Surg 4(6):409–415
27. Fukuda H, Hamada K, Nakajima T, Yamada N, Tomonaga A, Goto M (1996) Partial-thickness tears of the rotator cuff. A clinicopathological review based on 66 surgically verified cases. Int Orthop 20:257–265
28. Robertson PL, Schweitzer ME, Mitchell DG, Schlesinger F, Epstein RE, Frieman BG, Fenlin JM (1995) Rotator cuff disorders: interobserver and intraobserver variation in diagnosis with MR imaging. Radiology 194:831–835
29. Goutallier D, Bernageau J, Patte D (1989) Assessment of the trophicity of the muscles of the ruptured rotator cuff by CT scan. Rev Chir Orthop Reparatrice Appar Mot 75:126–127
30. Weber SC (1997) Arthroscopic debridement and acromioplasty versus mini-open repair in the management of significant partial-thickness tears of the rotator cuff. Orthop Clin North Am 28:79–82
31. Goutallier D, Postel JM, Bernageau J, Lavau L, Voisin MC (1994) Fatty muscle degeneration in cuff ruptures: pre- and postoperative evaluation by CT scan. Clin Orthop Relat Res 304:78–83
32. Thomazeau H, Boukobza E, Morcet N, Chaperon J, Langlais F (1997) Prediction of rotator cuff repair results by magnetic resonance imaging. Clin Orthop Relat Res 344:275–283
33. Zanetti M, Gerber C, Hodler J (1998) Quantitative assessment of the muscles of the rotator cuff with magnetic resonance imaging. Invest Radiol 33(3):163–170
34. Iannotti JP, Zlatkin MB, Esterhai JL, Kressel HY, Dalinka MK, Spindler KP (1991) Magnetic resonance imaging of the shoulder. Sensitivity, specificity, and predictive value. J Bone Joint Surg Am 73:1729
35. Lafosse L, Jost B, Reiland Y, Audebert S, Toussaint B, Gobezie R (2007) Structural integrity and clinical outcomes after arthroscopic repair of isolated subscapularis tears. J Bone Joint Surg Am 89(6):1184–1193
36. Li XX, Schweitzer ME, Bifano JA, Lerman J, Manton GL, El-Noueam KI (1999) MR evaluation of subscapularis tears. J Comput Assist Tomogr 23:713–717
37. Walch G, Nove-Josserand L, Levigne C, Renaud E (1994) Tears of the supraspinatus tendon associated with "hidden" lesions of the rotator interval. J Shoulder Elbow Surg 3(6):353–360
38. Chan TW, Dalinka MK, Kneeland JB, Chervrot A (1991) Biceps tendon dislocation: evaluation with MR imaging. Radiology 179:649–652
39. Patten RM (1994) Tears of the anterior portion of the rotator cuff (the subscapularis tendon): MR imaging findings. AJR Am J Roentgenol 162:351–354
40. Wall LB, Teefey SA, Middleton WD, Dahiya N, Steger-May K, Kim HM, Wessell D, Yamaguchi K (2012) Diagnostic performance and reliability of ultrasonography for fatty degeneration of the rotator cuff muscles. J Bone Joint Surg Am 94(12):e83
41. Hamada K, Fukuda H, Mikasa M, Kobayashi Y (1990) Roentgenographic findings in massive rotator cuff tears. A long-term observation. Clin Orthop 254:92–96
42. Lyons RP, Green A (2005) Subscapularis tendon tears. J Am Acad Orthop Surg 13(5):353–363
43. Zanetti M, Weishaupt D, Gerber C, Hodler J (1998) Tendinopathy and rupture of the tendon of the long head of the biceps brachii muscle: evaluation with MR arthrography. AJR Am J Roentgenol 170:1557–1561
44. Sakurai G, Ozaki J, Tomita Y, Kondo T, Tamai S (1998) Incomplete tears of the subscapularis tendon associated with tears of the supraspinatus tendon: cadaveric and clinical studies. J Shoulder Elbow Surg 7:510–515
45. Owens BD, Agel J, Mountcastle SB, Cameron KL, Nelson BJ (2009) Incidence of glenohumeral instability in collegiate athletics. Am J Sports Med 37(9):1750–1754

46. Hovelius L, Eriksson K, Fredin H et al (1983) Recurrences after initial dislocation of the shoulder: results of a prospective study of treatment. J Bone Joint Surg Am 65(3):343–349
47. Sachs RA, Lin D, Stone ML, Paxton E, Kuney M (2007) Can the need for future surgery for acute traumatic anterior shoulder dislocation be predicted? J Bone Joint Surg Am 89(8):1665–1674
48. Zacchilli MA, Owens BD (2010) Epidemiology of shoulder dislocations presenting to emergency departments in the United States. J Bone Joint Surg Am 92(3):542–549
49. Hawkins RJ, Neer CS, Pianta RM, Mendoza FX (1987) Locked posterior dislocation of the shoulder. J Bone Joint Surg Am 69:9–18
50. Wilson J, McKeever F (1949) Traumatic posterior (retroglenoid) dislocation of the humerus. J Bone Joint Surg Am 31:160–172
51. Kodali P, Jones MH, Polster J, Miniaci A, Fening SD (2011) Accuracy of measurement of Hill-Sachs lesions with computed tomography. J Shoulder Elbow Surg 20(8):1328–1334
52. Chuang TY, Adams CR, Burkhart SS (2008) Use of preoperative three-dimensional computed tomography to quantify glenoid bone loss in shoulder instability. Arthroscopy 24(4):376–382
53. Zanetti M, Hodler J (1997) Contrast media in MR arthrography of the glenohumeral joint: intra-articular gadopentetate vs saline: preliminary results. Eur Radiol 7:498–502
54. Wintzell G, Haglund-Akerlind Y, Larsson H, Zyto K, Larsson S (1999) Open MR imaging of the unstable shoulder in the apprehension test position: description and evaluation of an alternative MR examination position. Eur Radiol 9:1789–1795
55. Cvitanic O, Tirman PF, Feller JF, Bost FW, Minter J, Carroll KW (1997) Using abduction and external rotation of the shoulder to increase the sensitivity of MR arthrography in revealing tears of the anterior glenoid labrum. AJR Am J Roentgenol 169:837–844
56. Garth WP, Slappey CE, Ochs CW (1984) Roentgenographic demonstration of instability of the shoulder: the apical oblique projection. J Bone Joint Surg Am 66:1450–1453
57. Rowe CR, Patel D, Southmayd WW (1978) The Bankart procedure: a long-term end result study. J Bone Joint Surg Am 60:1–16
58. Hintermann B, Gächter A (1995) Arthroscopic findings after shoulder dislocation. Am J Sports Med 23:545–551
59. Itoi E, Lee SB, Berglund LJ, Berge LL, An KN (2000) The effect of a glenoid defect on anteroinferior stability of the shoulder after Bankart repair: a cadaveric study. J Bone Joint Surg Am 82:35–46
60. Sugaya H, Moriishi J, Dohi M, Kon Y, Tsuchiya A (2003) Glenoid rim morphology in recurrent anterior glenohumeral instability. J Bone Joint Surg Am 85:878–884
61. Calandra JJ, Baker CL, Uribe J (1989) The incidence of Hill-Sachs lesions in initial anterior shoulder dislocations. Arthroscopy 5(4):254–257
62. Edwards TB, Boulahia A, Walch G (2003) Radiographic analysis of bone defects in chronic anterior shoulder instability. Arthroscopy 19:732–739
63. Pavlov H, Warren RF, Weiss CB Jr, Dines DM (1985) The roentgenographic evaluation of anterior shoulder instability. Clin Orthop Relat Res 194:153–158
64. Bishop JY, Jones GL, Rerko MA, Donaldson C (2013) 3-D CT is the most reliable imaging modality when quantifying glenoid bone loss. Clin Orthop Relat Res 471(4):1251–1256
65. Burkhart SS, De Beer JF (2000) Traumatic glenohumeral bone defects and their relationship to failure of arthroscopic Bankart repairs: significance of the inverted-pear glenoid and the humeral engaging Hill-Sachs lesion. Arthroscopy 16(7):677–694
66. Huysmans PE, Haen PS, Kidd M, Dhert WJ, Willems JW (2006) The shape of the inferior part of the glenoid: a cadaveric study. J Shoulder Elbow Surg 15:759–763
67. Magee T (2009) 3-T MRI of the shoulder: is MR arthrography necessary? AJR Am J Roentgenol 192(1):86–92
68. Balich SM, Sheley RC, Brown TR, Sausor D, Quinn SF (1996) MR Imaging evaluation of the glenoid labrum. Radiology 201:430
69. Fallahi F, Green N, Gadde S, Jeavons L, Armstrong P, Jonker L (2013) Indirect magnetic resonance arthrography of the shoulder; a reliable diagnostic tool for investigation of suspected labral pathology. Skeletal Radiol 42(9):1225–1233
70. Jahnke AJ, Petersen SA, Neumann C, Steinbach L, Morgan FA (1992) A prospective comparison of computerized arthrotomography and magnetic resonance imaging of the glenohumeral joint. Am J Sports Med 20:695–700
71. Fotiadou A, Drevelegas A, Nasuto M, Guglielmi G (2013) Diagnostic performance of magnetic resonance arthrography of the shoulder in the evaluation of anteroinferior labrum abnormalities: a prospective study. Insights Imaging 4(2):157–162
72. Lo IK, Parten PM, Burkhart SS (2004) The inverted pear glenoid: an indicator of significant glenoid bone loss. Arthroscopy 20:169–174
73. Parameswaran AD, Provencher MT, Bach BR Jr, Verma N, Romeo AA (2008) Humeral avulsion of the glenohumeral ligament: injury pattern and arthroscopic repair techniques. Orthopedics 31(8):773–779
74. Bui Mansfield LT, Taylor DC, Uhorchak JM, Tenuta JJ (2002) Humeral avulsions of the glenohumeral ligament: imaging features and a review of the literature. AJR Am J Roentgenol 179(3):649–655
75. Bui Mansfield LT, Banks KP, Taylor DC (2007) Humeral avulsion of the glenohumeral ligaments: the HAGL lesion. Am J Sports Med 35(11):1960–1966
76. Oh CH, Schweitzer ME, Spettell CM (1999) Internal derangements of the shoulder: decision tree and cost-effectiveness analysis of conventional arthrography, conventional MRI, and MR arthrography. Skeletal Radiol 28:670–678
77. Snyder SJ, Karzel RP, Del Pizzo W, Ferkel RD, Friedman MJ (1990) SLAP lesions of the shoulder. Arthroscopy 6:274–279
78. Nicola T (1942) Anterior dislocation of the shoulder: the role of the articular capsule. J Bone Joint Surg Am 25:614–616
79. Mileski RA, Snyder SJ (1998) Superior labral lesions in the shoulder: pathoanatomy and surgical management. J Am Acad Orthop Surg 6:121–131
80. Jin W, Ryu KN, Kwon SH, Rhee YG, Yang DM (2006) MR arthrography in the differential diagnosis of type II superior labral anteroposterior lesion and sublabral recess. AJR Am J Roentgenol 187:887–893
81. Tuite MJ, Cirillo RL, De Smet AA, Orwin JF (2000) Superior labrum anterior-posterior (SLAP) tears: evaluation of three MR signs on T2-weighted images. Radiology 215:841–845
82. Phillips JC, Cook C, Beaty S, Kissenberth MJ, Siffri P, Hawkins RJ (2013) Validity of noncontrast magnetic resonance imaging in diagnosing superior labrum anterior-posterior tears. J Shoulder Elbow Surg 22(1):3–8

Shoulder Management of the First Time Anterior Shoulder Dislocation

16

Stefano Boschi, Gianezio Paribelli, Mario Perretti, and Alfonso Massimo Cassarino

16.1 Aetiology

Anterior shoulder dislocation is a very common injury among the general population but especially among athletes [1–3]. It has a reported recurrence rate of 20–50 % in the general population [1, 3, 4], which rises to 47–100 % among younger patients. The recurrence rate is reported to be even higher, reaching 80–94 % among young athletes [1, 2, 4–8]. Previous studies [4, 8] have identified an association between the type of sports engaged in, the number of participants and the rate of recurrence after a first-time dislocation in young athletes. They found that not all sports correlated with recurrence of dislocation and that athletes taking part in contact sports had greater rates of instability and surgical treatment than those involved in noncontact sports [4]. Among young athletes engaged in overhead or contact sports, 75 % experience recurrent dislocation if they are permitted an early return to play after a first-time dislocation, since the ligaments are not given the time to heal properly.

The first parameter to be considered is the natural history of shoulder dislocations in young athletes, which has been well studied and shows that recurrences of dislocation and instability follow nonsurgical treatment in 50–100 % of cases [3, 4, 6–10]. The priority of physicians is to be familiar with the fundamentals of instability, including conservative treatments of acute dislocations, and be aware that the results achieved with early arthroscopic stabilisation have dramatically changed the natural history of this major problem [3, 4, 7, 8, 10, 11]. The fate of a dislocation following a first episode is closely related to the patient's age – the second parameter. Recurrence rates are 100 % among children under the age of 13 years and decrease in old age, and with high percentage values from 79 % to 94 % being recorded at the time of peak sports performance, that is, between the ages of 16 and 30 years [2, 6, 7]. Several recent papers [3, 7, 8, 10, 11] report recurrence rates of 60–75 % for athletes treated conservatively and 11–20 % for those treated surgically. Surgical treatment apparently also prevents further secondary damage to the joint structures. The third parameter to be considered is the type of sport: sports are traditionally divided into the two broad categories of contact-collision sports (soccer, rugby, American football, *basketball*, wrestling, boxing, fencing, *water polo*, *handball*, Graeco-Roman wrestling, judo and martial arts in general, skiing, cycling, motorcycling) and noncontact sports (*volleyball*, *tennis*, *swimming*, *throwing sports*, *pitching in baseball and softball*, golf, body building, bowling). However, the underlined sports in the above list are also overhead sports, which increase decision-making problems [12]. In these sports in fact typical lesions may precede the dislocation episode, such as SLAP lesions and long-head-of-the-biceps lesions, rotator cuff lesions, capsular ligamentous lesions, chondral lesions and lesions of the rotator interval. In over-head sports, we need to consider that a complete (or as complete as possible) range of motion is the priority in order to ensure a high level of activity, whereas in contact-collision sports it is more important to have a solid joint stability. The fourth parameter is the type of lesion. It should be remembered that a first-time anterior shoulder dislocation is a multifactorial condition [13] and that no single injury is responsible for the instability, but there may be several simultaneous lesions affecting different soft tissues as well as both humeral and glenoid bony lesions. After a first-time dislocation, the following lesions may be present – singly or in combination depending on the kinetics and energy of the trauma – a

S. Boschi (✉) • M. Perretti
Ospedale Accreditato Privato Prof. Fogliani,
Via Lana 1, Modena 41124, Italy

Ospedale Privato Accreditato Prof. Fogliani,
Via Lana 1, Modena 41124, Italy
e-mail: s.boschi@iol.it; Mario.perretti@libero.it

G. Paribelli
Ospedale Privato Accreditato Domus Nova, Ravenna, Italy

Domus Nova, Via P.Pavirani, 44, Ravenna 48121, Italy
e-mail: geparibelli@libero.it

A.M. Cassarino
Ospedale Privato Accreditato Domus Nova, Ravenna, Italy

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_16,

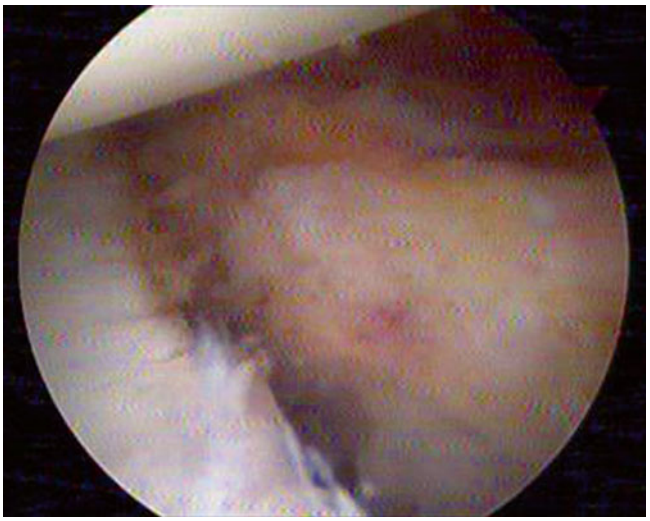


Fig. 16.1 Arthroscopic image of the labral capsular ligamentous complex after a first traumatic episode of antero-inferior shoulder dislocation in a basketball player

typical Bankart lesion (Fig. 16.1), labral tears extending also posteriorly, Kim's lesions, more or less extensive rotator cuff lesions with possible subscapularis detachment, SLAP lesions with both anterior and posterior extension, rotator interval lesions, capsular lesions with glenohumeral ligament injuries such as humeral avulsion of the anterior glenohumeral ligament (HAGL) or lesions of the humeral insertion of the posterior inferior humeral ligaments (reversed HAGL or RHAGL), and sometimes nerve injuries which may be partial or complete injuries of the brachial plexus but more commonly involving only the axillary nerve. However, the most important and most widely debated issue in decisions regarding the timing and type of surgical procedure concerns isolated or combined bony defects of the glenoid bone (typical bony Bankart lesion) and of the humeral bone (typical Hill-Sachs lesion) of any size; in addition, especially as regards humeral lesions, the site is extremely important since, depending on location, these lesions are capable of completely altering the glenoid track [14]. Finally, the last parameter is that of the timing of dislocation in relation to the competitive season. We can hypothesise three scenarios [15]: (1) attempt to complete the season with or without a brace and defer surgery until they have a recurrence of instability, (2) attempt to complete the season with or without a brace and defer surgery until the off season and (3) immediate surgery and forfeit the current season. Each scenario has its own obvious advantages and disadvantages. Even in this case the athlete's age, type of sport, importance of the sporting event, beginning, middle and end of the season may affect the decision, including clearly the possibility of returning to play without undergoing a surgical procedure [3, 4, 7, 8, 15], since every athlete with a shoulder dislocation, despite a high risk, will not have a recurrence. Any choice should be carefully considered and discussed first with the athlete and his or her family, if a minor, and then with the coaches, rehabilitators and trainers so as to ensure, once the decision has been made, that the timeframe and methods for functional recovery are respected.

16.2 Diagnosis

The mechanism underlying anterior, or better antero-inferior, shoulder dislocation is a traumatic injury with or without contact during abduction and external rotation. The diagnosis of anterior dislocation is established on the basis of a physical examination which shows a change in the anatomical profile of the shoulder, more evident in less muscular, leaner athletes, with loss of subdeltoid contour and often the humeral head in the antero-inferior portion of the axilla. The patient reports anterolateral shoulder pain, described as internal, which normally subsides when the dislocation is reduced. During this phase the physician needs to carefully assess the possible presence of associated nerve lesions. Confirmation is provided by a radiographic series (true AP and true lateral and axillary views) to assess both the presence of the dislocation and possible associated humeral or glenoid bony lesions. A thorough history is also crucial, during which the patient should be asked whether he/she had previous episodes of partial dislocation or sensation of instability or whether he/she remembers the manner in which the trauma causing the dislocation occurred.

The dislocation should be reduced as soon as possible and possibly at the first attempt so as to avoid adding further tissue injuries due to failed attempts at reduction. For this reason and because athletes often have a particularly well-developed musculature, it is best to perform the reduction following anaesthesia or deep sedation and possibly under imaging guidance. Once the dislocation has been reduced, the resulting shoulder stability should be tested with gentle movements and clearly without forcing with specific tests against resistance; the patient should be assessed contralaterally and from a general point of view in order to identify any congenital laxity that might complicate the decision-making process. A post-reduction radiogram is obtained, followed by other imaging investigations that will help decide on the most appropriate treatment strategies. These will include an unenhanced magnetic resonance (MR) examination to assess the soft tissues, supplemented by contrast-enhanced scans in the event of doubts in the interpretation of capsular ligamentous structures, and a computed tomography (CT) study with 3D reconstructions to assess bony defects of the glenoid and humeral head in terms of both size and position. At this point the physician has all the information necessary to decide on which treatment to perform – whether conservative or surgical and, if surgical, the best procedure. We should always

remember the characteristics of the natural history of shoulder dislocations, which include a high rate of recurrences, progressive damage to the stabilising structures and the fact that early arthroscopic treatment is beneficial to young athletes under the age of 25 whether involved in noncontact or contact-collision sports [7, 8, 10, 11, 16]. We can in fact identify and treat all lesions, understand the quality of the tissue and its healing capacity, using a noninvasive method associated with a low complication rate.

16.3 Treatment

Our treatment algorithm for a first-time anterior shoulder dislocation in an athlete is to perform an arthroscopic stabilisation procedure as soon as possible, within a week at the most. When necessary, we prefer to use small-diameter bioabsorbable anchors and high-resistance threads in order to avoid problems related to metal and its possible mobilisation. Recently we have started to use soft anchors with great satisfaction, as these have the advantages described above with the added benefit of an excellent pull-out strength and major sparing of bone tissue. Arthroscopy enables the complete study of both the articular space and the subacromial and subcoracoid spaces [17] and thus allows us to treat any associated lesions in the same session. Early repair allows us to exploit the natural biological tendency to heal without interfering with the surgical procedure. Problems may arise due to bony lesions and especially simultaneous humeral and glenoid lesions and anterior HAGL lesions. In the case of bony Bankart lesions [18], treatment is arthroscopic, with a possibility for open stabilisation in the event of extensive glenoid fractures. Depending on position, Hill-Sachs lesions are sometimes treated with an arthroscopic remplissage procedure [19]. When the two bony lesions are combined, with evident loss of the glenoid track, the choice is between an open or arthroscopic Latarjet procedure in the case of contact-collision sports; however, we are always doubtful in athletes younger than 20 years of age engaged in noncontact, non-collision sports or overhead sports since the Latarjet procedure completely disrupts shoulder anatomy, and it is a replacement rather than a repair procedure [20, 21]. Some authors [22] have suggested adopting a preoperative score to assess the severity of the instability and help choose the most appropriate procedure. However, in our opinion, this score is too unbalanced, and young athletes easily attain scores indicating a need for the more aggressive Latarjet stabilisation procedure. Moreover, we agree with other authors [16] that this score is only a part of a puzzle that requires validation by prospective studies. HAGL lesions are treated arthroscopically or with open surgery depending on whether they are associated with major subscapularis lesions. Following the surgical procedure, the patient is placed in an ultrasling shoulder brace in neutral position for 4 weeks; several times a day, gentle wrist, elbow and shoulder mobilisation movements of the pendulum type are prescribed – but not external rotations and abductions.

The conservative approach is reserved for lower-level athletes over the age of 30 engaged in noncontact-collision sports and no overhead sports and who do not have bony lesions, HAGL lesions or rotator cuff lesions. Even in this case postoperative care consists of immobilisation with an ultrasling shoulder brace in neutral position for 4 weeks.

As stated above, we clearly take into account the level of the athlete and the timing of the dislocation in relation to the competitive season. A temporary conservative treatment allowing the athlete to complete the season consists of the ultrasling shoulder brace for 2/3 weeks followed by functional recovery on land and in water to restore range of motion; only after this has been restored can the athlete start exercises to strengthen the muscles and restore specific athletic movement. Very important are early exercises for the scapulothoracic joint aiming at mobilisation and progressive strengthening of its stabilisers. Return to play in these cases is estimated at around 6/8 weeks, but may take longer in the case of overhead athletes.

In the event of arthroscopic surgical stabilisation, functional recovery after removal of the shoulder brace takes place in a similar fashion, with recovery of first passive and then active motion – always within the pain threshold – strengthening exercises after 2/3 months, sports-specific exercises after 3/4 months and finally return to play after 5/6 months. In the event of a Latarjet stabilisation procedure, after 4 weeks of ultrasling shoulder brace in neutral position and follow-up radiography, functional recovery takes place using the same methods but with a shorter timeframe, and the athlete can return to play ca. 3/4 months after radiography or 3D computed tomography has confirmed that the graft has consolided. Unfortunately, however, in many cases due to the needs of the athlete or his or her family and pressure from the coaches and media, return to play is permitted before complete healing of the tissues, and this exposes the athlete to recurrences and the surgeon and rehabilitators to having to justify a treatment failure.

Conclusions

Young athletes are at high risk of recurrence after a first-time anterior shoulder dislocation, and immobilisation and strengthening exercises are unable to influence the recurrence rate. The disorder is multifactorial, and no single lesion is responsible for the instability but a summation of pathoanatomical lesions in part dependent on the type of trauma sustained and the type of sports played. We should not forget that also predisposing conditions exist such as congenital laxity which further complicate the clinical picture and treatment decisions. A high recurrence rate among

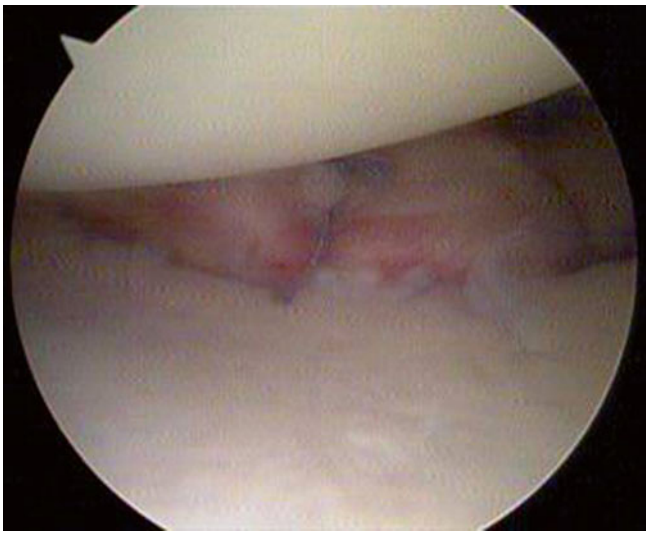


Fig. 16.2 Arthroscopic image of stabilisation of the lesion of the labral capsular ligamentous complex after a first traumatic episode of antero-inferior shoulder dislocation in a basketball player

athletes has also been correlated to bony defects and especially to combined bony defects that alter the glenoid track [14]. Early arthroscopic treatment has shown a high success rate [7, 8, 10, 11] and has contributed to changing the natural history of dislocations. We also need to consider the different priorities of different types of sports, with contact-collision sports requiring joint stability and overhead sports requiring the greatest possible range of motion. Finally, athletes differ in terms of age, needs and prospects, with some having long seasons of athletic activity and others shorter seasons.

However, consistent with previous authors [8, 11, 16], we recommend to fix first-time anterior shoulder dislocations in young athletes as soon as possible and using a prevalently arthroscopic-assisted surgical stabilisation procedure (Fig. 16.2), bearing in mind that each case is different and that the indications and prognosis should be discussed with the athlete and his or her parents.

References

1. Rhee YG, Cho NS, Cho SH (2009) Traumatic anterior dislocation of the shoulder: factor affecting the progress of the anterior dislocation. Clin Orthop Surg 1(14):188–193. doi: 10.4055/cios.2009.1.4.188
2. Blanie TA, Park MC, Levine WN (2002) Shoulder dislocation in young athletes: current concepts in management. Phys Sportsmed 30(12):41–48
3. Johannsen HV, Jacobsen BW, Suder P, Sojbjerg JO (2007) Primary repair vs Conservative treatment of first time traumatic anterior dislocation of the shoulder: a randomized study with 10-year follow up. Arthroscopy 23(2):118–123
4. Bottoni CR, Wilckens JH, DE Berardino TM et al (2002) A prospective randomized evaluation of arthroscopic stabilization versus non operative treatment in patients with acute traumatic first time shoulder dislocations. Am J Sports Med 30(4):576–580
5. Hovelius L, Augustini BG, Fredin H et al (1996) Primary anterior dislocation of the shoulder in young patients: a ten-year prospective study. J Bone Joint Surg Am 78(11):1677–1684
6. Hovelius L (1999) The natural history of primary anterior dislocation of the shoulder in the young. J Orthop Sci 4(4):307–317
7. Brophy RH, Marx RG (2009) The treatment of traumatic anterior instability of the shoulder: nonoperative and surgical treatment. Arthroscopy 25(3):298–304
8. Berkovitch Y, Shaphira J, Haddad M, Keren Y, Rosemberg N (2013) Current clinical trends in first traumatic shoulder dislocation. Merit Res J Med Med Sci 1(1):7–13
9. Henry JH, Genius JA (1982) Natural history of glenohumeral dislocation-revisited. Am J Sports Med 10(3):135–137
10. Boone JL, Arciero RA (2010) First-time anterior shoulder dislocations: has the standard changed? Br J Sports Med 44:355–360
11. Arciero RA, Taylor DC, Snyder RJ et al (1994) Arthroscopic Bankart repair versus non operative treatment for acute initial shoulder dislocation. Am J Sports Med 22(5):589–594
12. Castagna A, Delle Rose G, Borroni M et al (2012) Arthroscopic stabilization of the shoulder in adolescent athlete participating in overhead or contact sports. Arthroscopy 28(3):309–315
13. Boschi S, Paribelli G (2005) Come tratto una lesione di Bankart. Artroscopia 6(1):41–49
14. Yamamoto N, Itoi E, Abe H, Minagawa H, Seki N, Shimada Y, Okada K (2007) Contact between glenoid and the humeral head in abduction, external rotation, and horizontal extension: a new concept of glenoid track. J Shoulder Elbow Surg 16(5):649–656
15. Burris MW, Mair SD, Johnson DL (2007) Management of in-season anterior shoulder dislocation in the amateur athlete. Orthopedics 30(5):362–364
16. Randelli P, Taverna E (2009) Primary anterior shoulder dislocation in young patients: fix them. Knne Surg Sports Traumatol Arthrosc 17:1404–1405
17. Paribelli G, Boschi S (2005) Complete subscapularis tendon visualization and axillary nerve identification by arthroscopy technique. Arthroscopy 21(8):1016–1021
18. Porcellini G, Campi F, Paladini P (2002) Arthroscopic approach to acute bony Bankart lesion. Arthroscopy 18:764–769
19. Wolf EM, Pollack M, Smalley C (2008) Hill-Sachs remplissage: an arthroscopic solution for the engaging Hill-Sachs lesion. Arthroscopy 24(6):723–726
20. Bottoni CR, Smith EL, Berkovitz MJ, Towle RB, Moore JH (2006) Arthroscopic versus open shoulder stabilization for recurrent anterior instability a prospective randomized clinical trial. Am J Sports Med 34:1730–1737
21. Fabbriciani C, Milano G, DE Montis A, Fadda S (2004) Arthroscopic versus open shoulder stabilization for recurrent anterior instability: a prospective randomized study. Arthroscopy 20:456–462
22. Belg F, Boileau P (2007) The instability severity index score. A simple pre-operative score to select patients for arthroscopy or open shoulder stabilization. J Bone Joint Surg Br 89:1470–1477

Shoulder Superior Labral Repair

17

S. De Giorgi, R. Garofalo, G. Delle Rose, and A. Castagna

17.1 The Superior Labrum: Anatomy, Role, and Pathology

The upper part of the glenoid labrum has a clear morphological diversity if we compare it with the lower part. It evokes the meniscal morphology, with a roughly triangular section, the margin thin free towards the center of the joint and an insertion at the glenoid rim relatively vulnerable as constituted by a connective end that can be stretched easily since the lack of inelastic fibers. By light microscopy, there is a transition zone between the fibrocartilaginous hyaline cartilage of the glenoid labrum and the labral tissue. The study by electron microscopy [29] shows that in this transitional area, fine fibrils in the labrum fit the glenoid rim with Sharpey's fibers in radial course. By contrast, the fibrils of the capsule of the glenohumeral ligament, the biceps tendon, and labrum are parallel to the bone.

Some authors [18] identify in this complex system of fibers a true functional unit: the periarticular fibrillar system (PAFS). So the glenoid labrum appears to be a narrow band of fibrocartilage located between the junction of the articular capsule and the margin of the glenoid cavity. It consists of three layers of collagen fibrils that with their spatial orientation make it suitable to withstand mechanical stress, tensile and compressive, functionally resembling to a bumper or a bearing. This feature, however, is conditioned by the morphology and mode of insertion on the glenoid that varies according to the topographical site. The upper portion of the labrum due to its triangular morphology with free margin is more exposed and vulnerable to tensile stresses. In addition, the superior labrum interacts with the insertion of the tendon of the long head of the biceps (LHB). Authoritative anatomical studies at the end of the last century reported to have placed the proximal insertion of the tendon of the long head of the biceps only at the supraglenoid tubercle. One cadaveric study [42] of the insertion of the long head of the biceps of 100 specimens aged between 30 and 90 years recorded the percentage of fibers that were part of the tubercle and the anterior and the posterior labrum. Only in 30 % of cases, they were demonstrated to originate exclusively from the supraglenoid tubercle; in other cases, the origin appears to be divided between the tubercle and glenoid labrum. The same authors have therefore distinct modes of insertion of the fibers in relation to the bicipital superior labrum into four types:

I. All fibers arose from the posterior lip of the front without any contribution (22 %).
II. Most of the posterior lip with a small contribution from the front lip (33 %).
III. Equal contribution from the front lip and rear (37 %).
IV. The majority of the front lip with a small contribution from the posterior lip (8 %)

As a conclusion, the superior labrum is a vulnerable anatomical structure functionally interacting with other joint elements and with a variable anatomy.

A lesion of this complex system may lead to significant dysfunction especially in young thrower athletes.

17.2 Etiology

Superior glenoid lesions or SLAP (superior labrum from anterior to posterior) lesions are infrequent but important causes of shoulder pain and disability [25, 39].

These lesions have been classified by Snyder et al. [39] into four kinds:

1. SLAP I: Degeneration or fraying of the labrum without a tear of the biceps tendon.

S. De Giorgi (✉)
Department of Basic Medical Sciences,
Neurosciences and Sensory Organs – University of Bari,
Bari, Italy
e-mail: silvana.degiorgi@uniba.it

R. Garofalo
Shoulder Service, Miulli Hospital,
Acquaviva delle Fonti, Bari, Italy

G. Delle Rose • A. Castagna
Shoulder and Elbow Unit, IRCCS Humanitas Institute,
Rozzano, Milan, Italy

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_17,

2. SLAP II: Complete detachment of the superior labrum and biceps anchor from the superior glenoid tubercle, besides a certain fraying of the edge of the labrum.
3. SLAP III: Presence of a bucket haft of the superior labrum with a normal biceps tendon.
4. SLAP IV: It is a SLAP III lesion with an associated partial longitudinal lesion of the biceps tendon which is dislocated into the joint.

Later on, some authors extended the classification of SLAP lesions to nine kinds [24]. In his original paper, Snyder reports a total incidence of SLAP lesions of 4.8 %, but later on (1995) he reported an incidence of 6.9 % [40]. SLAP II lesions are the commonest SLAP lesions. They are generally associated to other shoulder pathologies such as Bankart lesions, Rotator cuff tears (SLAC: superior labrum anterior cuff) [36], and elongation of the MGHL [9]. Kim et al. reported 90 % association with other lesions, i.e., rotator cuff tear and Bankart lesion [21]. Rotator cuff lesions were more common in patients older than 40 years old, whereas Bankart lesions were more commonly seen in patients aged less than 40 years old [11].

17.3 Injury Mechanism

A SLAP lesion is often described in overhead athletes, such as swimmers and throwers who can present an internal impingement (posterosuperior) with a contact during the throwing between the articular side of the supraspinatus tendon and the posterosuperior glenoid labrum [14, 19]. A SLAP lesion can be associated to an AIOS (acquired instability in overstressed shoulder surgery) which can be observed as minor instabilities in throwers or overhead workers. It is related to repetitive microtrauma that can produce a degeneration or a dysfunction of the static stabilizing systems of the shoulder and a minor instability. The anterior translation of the humeral head can produce a SLAP lesion for a mechanism of traction on the biceps anchor [9]. The progressive detachment of the superior labrum known as "peel back" of the posterosuperior labrum from the glenoid is a consequence of repetitive throwing microtraumas and can lead to a prevalent posterior SLAP II lesion [8].

17.3.1 Biceps Anchor Anatomy

The anterosuperior labrum extending from the biceps anchor to the midglenoid notch (the 3 o'clock position on the glenoid rim) is one of the most variable and confusing areas of glenohumeral anatomy [13]. A small recess or synovial reflection just below the biceps insertion or the supraglenoid tubercle is usually present. The supraglenoid labrum is loosely attached to the glenoid, and its configuration resembles that of the meniscus in the knee. The glenoid labrum is triangular in cross section. The superior and middle glenohumeral ligaments usually attach to the anterosuperior labrum. The biceps anchor inserts for 40–60 % of its fibers on the supraglenoid tubercle of the scapula and 5 mm medially to the joint rim and to the rest on the glenoid labrum [42]. Its morphology can be normal or meniscoid, which is present in 15 % of the cases [39] with a large free part in the joint. A sublabral foramen is distinguished from a slap tear by virtue of its smooth borders and its medial extension between the superior labrum and the bony glenoid, while a sublabral tear would extend laterally or superiorly into the labrum [43]. In SLAP II lesions, the biceps anchor peels off from the supraglenoid tubercle with the associated detachment of the labrum extending for a variable distance anteriorly and posteriorly; the biceps anchor can be displaced medially towards the glenoid neck on probing. A "peel back" of the anchor can be demonstrated by abduction and external rotation of the arm [27].

It can be assumed that the superior labrum with its attached capsuloligamentous structures has a role in glenohumeral joint instability. Generally the instability produced by a SLAP lesion is proportionate to the extension of the tear in the anteroposterior direction and to the involvement of the biceps anchor [25]. Nevertheless the length of the tear may not always quantify the level of translation of the humeral head compared to the glenoid, and other pathological findings can be involved to explain shoulder instability and pain.

17.4 Clinical and Diagnostic Examination

Extremely variable presentations with nonspecific clinical and radiographic findings make preoperative diagnosis of superior glenoid lesions difficult [13, 20, 31]. Generally, SLAP lesions present with shoulder pain and mechanical symptoms such as clicking, catching, or popping. Clinical symptoms of SLAP lesions are often sneaky and variable. The patient can present a painful shoulder during throwing activities, and a loss of passive internal rotation often occurs. Some patients can present a "locked shoulder" or a "dead arm" feeling. The speed test is positive in 50 % of cases. The O'Brien test seems to be the more specific test for a superior labrum lesion [30, 36].

The Neer and Hawkins tests can be positive if a secondary impingement is present [30]. The Jobe or Whipple test can be positive if an SLAC lesion occurs [36]. In minor instabilities with a deficiency of the MGHL, the Castagna test is positive [9] when there is a painful and apprehensive shoulder with the arm at 45–60° of abduction in external rotation. In the case of an associated Bankart lesion, the apprehension and relocation tests are positive too.

Also the standard imaging can be confusing. Standard X-rays, echography, and CT can only exclude other concomitant shoulder pathologies. The arthro-MRI is the most sensitive radiological exam, but it can often be positive without any symptoms related to the superior labrum. The convergence of anamnestic history, clinical symptoms, and arthro-MRI can direct the clinician towards the diagnosis of a SLAP lesion, but its confirmation can only be made by arthroscopy.

17.5 Treatment Strategy

Generally the first treatment of a SLAP lesion is conservative, trying to recover a good internal rotation which is often lost in overhead athletes. The use of analgesics or FANS drugs can only produce a transient pain relief.

In case of a failure of the conservative treatment, a surgical arthroscopic treatment is used. Thus the diagnosis can be confirmed and the optimal treatment performed.

During arthroscopy, a careful assessment of the SLAP complex should be done and a Buford complex, Sublabral hole, and cord-like MGHL should be diagnosed if eventually present. The treatment of SLAP lesions varies on the basis of the kind of lesion and age of the patient [1, 5, 15, 22, 23, 38].

- *SLAP I lesions*: The superior labrum is debrided with a shaver to avoid possible mechanical blocks.
- *SLAP II lesions*: The lesion is abraded and can be sutured with bioresorbable anchors (age <40 years), or a tenotomy/tenodesis can be performed [6] (age >40 years). Brockmeier et al. [7] recently showed that using an arthroscopic repair for an isolated and posttraumatic SLAP lesion, very good outcomes can be achieved with a full recovery of the previous performed sports. Arthroscopic repair of SLAP II lesion and Bankart lesion can be a cause of postsurgical shoulder stiffness [41]. Repair of the SLAP II lesions has been shown to be a successful procedure in the young overhead athlete; nevertheless, recent literature reported that the ability to return to preinjury level of sports remains a concern [26, 28]. Several techniques of SLAP II lesion repair are described, differing in the portals and numbers of anchors [32], but they are all related to the labral stability without understanding the anatomy we are aiming to restore. As a matter of fact, overtensioning the biceps anchor and the superior labrum may lead to residual stiffness and clinical symptoms. No statistical differences in the function and strength of the arm following tenotomy or tenotomy and tenodesis of the LHB have been reported in the literature [6]; thus the choice between these two techniques is only based on an esthetic reason to avoid the Popeye sign.
- *SLAP III lesions*: They present a bucket haft of the superior labrum. Therefore they should be carefully analyzed to assure a stable anchorage of the biceps anchor and residual labrum. The torn fragment of the labrum must be removed.
- *SLAP IV lesions*: The treatment can be different according to the extension of the biceps tendon lesion and to the age of the subject. If less than 30 % of the tendon is torn, the detached labrum and the torn part of the LHB can be removed. If more than 30 % of the tendon is torn, in an old patient a tenotomy can be performed, while in a young patient a SLAP repair is recommended [3].

The standard arthroscopic repair of a SLAP II lesion may result in a residual pain and stiffness of the shoulder in overhead athletes [6, 7, 16, 37], and the return to elite throwing sports particularly at the same preoperative level remains challenging [28]. The restoration of the real anatomy can probably improve clinical outcomes and sport performances, even if the reported literature about this topic is lacking. Therefore, during surgery for SLAP repair, the mobility of the labrum must be preserved, because if it is firmly fixed to the glenoid, the risk of severe loss of rotation could be high. Nevertheless, the stability of the LHB anchor must be restored, thus stabilizing also the insertions of the SGHL and MGHL [34], to reestablish the stabilizing effect of the biceps tendon for the shoulder joint. Traditional techniques of SLAP lesions repair often strangle the LHB giving a high rigidity to the labrum which loses its mobility. Thus, a certain stiffness of the shoulder often occurs. We thus describe our personal technique for treating SLAP II lesions. It aims to accurately reproduce the normal anatomy of the superior labrum. In our daily practice, an arthroscopic surgery under interscalene block anesthesia with the patients in the lateral decubitus position is usually performed. A standard posterior viewing portal and an accessory anterosuperior working portal, high in the rotator interval region, using the inside-out technique were created. An evaluation of the rest of the intra-articular structures of the shoulder (cuff, biceps tendon, articular surface), apart from the superior labrum, was carried out in order to confirm the presence of concurrent pathologies. Then, using a probe, the SLAP II lesion was confirmed (Fig. 17.1) according to the existence of a complete detachment of the biceps anchor from the superior glenoid tubercle, besides certain fraying of the edge of the labrum [39]. A 4.5 mm shaver was used in order to regularize the edge of the superior labrum when necessary and debride the bleeding bone of the superior part of the glenoid neck (Fig. 17.2). Using the anterior portal, a bioabsorbable anchor loaded with two sutures (Lupine, Depuy Mitek) was inserted into a predrilled hole in the glenoid rim just below the biceps anchor, 2–3 mm medial to the articular surface (12 o'clock position) (Fig. 17.3). With the help of a suture passer, a free non-reabsorbable monofilament suture was passed through the posteromedial aspect of the biceps anchor from superior and medial to inferior and lateral, emerging just under the

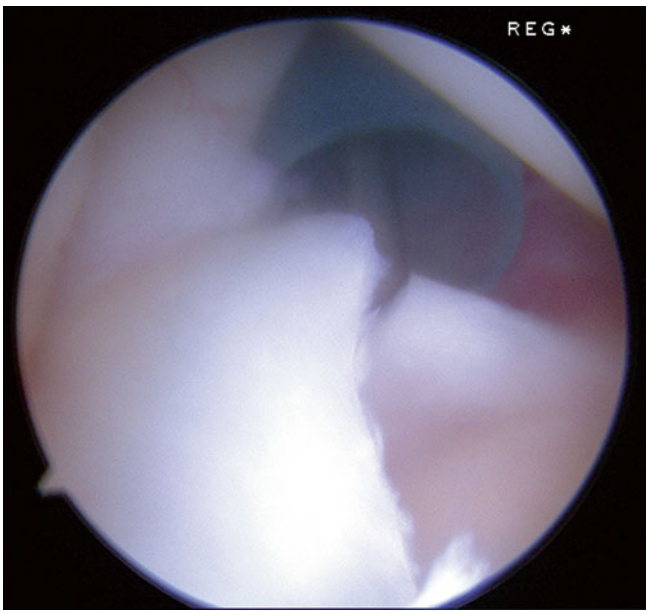


Fig. 17.1 Using a probe, the SLAP type II lesion was confirmed according to the existence of a complete detachment of the biceps anchor from the superior glenoid tubercle, besides certain fraying of the edge of the labrum

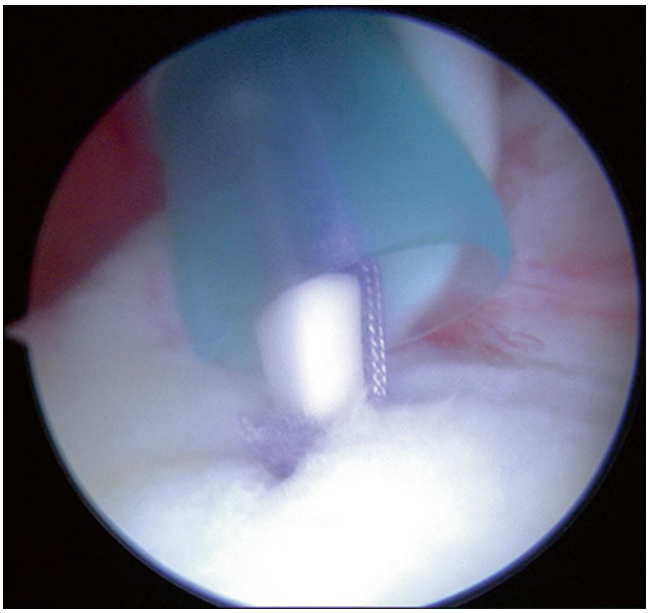


Fig. 17.3 Using the anterior portal, a bioabsorbable anchor loaded with two sutures (Lupine Depuy Mitek) was inserted into a predrilled hole in the glenoid rim just below the biceps anchor, 2–3 mm medial to the articular surface (12 o'clock position)

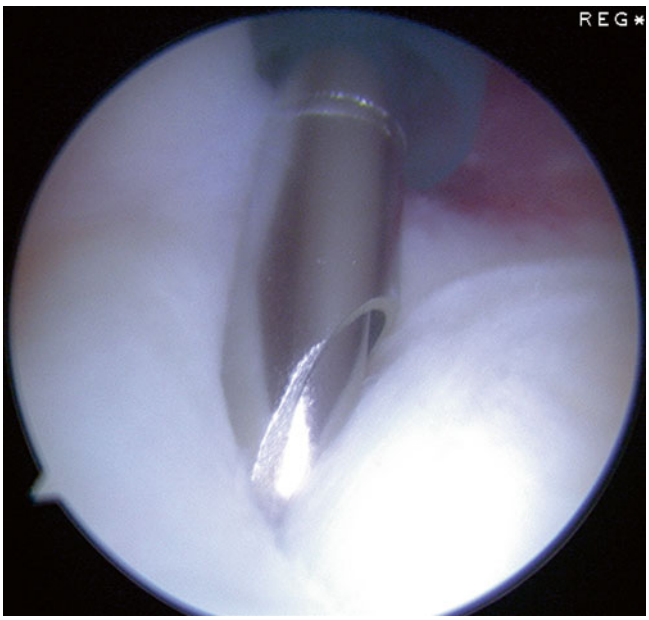


Fig. 17.2 A 4.5 mm shaver was used in order to regularize the edge of the superior labrum when necessary and debride to bleeding bone the superior part of the glenoid neck

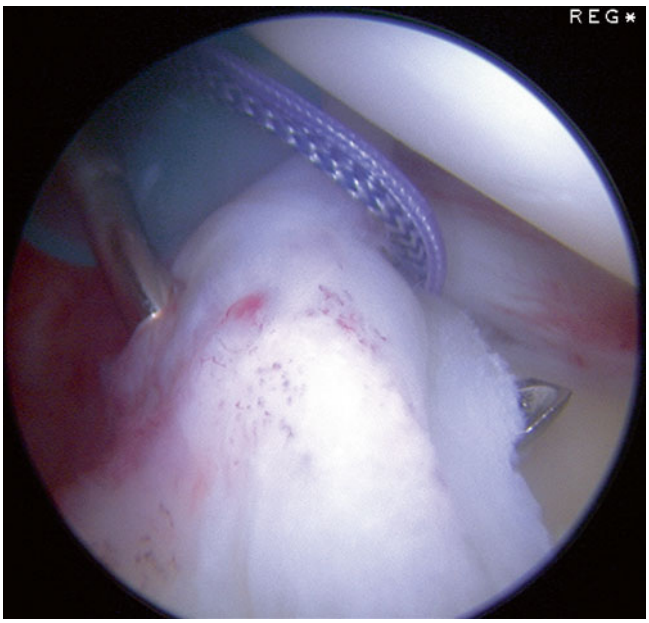


Fig. 17.4 With the help of a suture passer, a free non-resorbable monofilament suture is passed through the posteromedial aspect of the biceps anchor from superior and medial to inferior and lateral, emerging just under the posterior aspect of the superior labrum

posterior aspect of the superior labrum (Fig. 17.4). The intra-articular end of this monofilament suture was retrieved through the anterosuperior portal in order to shuttle one of the limbs of the sutures in the anchor through the labrum (Fig. 17.5). The other limb was passed through the labrum adjacent to the biceps, a few millimeters anterior to the first one, thus creating a mattress stitch (Fig. 17.6). The horizontal mattress suture should not cross over point "A" (anterior edge of LHB on the labrum) because no LHB fiber was sent anterior to the anterior edge of the supraglenoid tubercle [2]. Using the same suture passer technique, one of the limbs of the second suture of the anchor was passed through the superior labrum at the level of the insertion of the MGHL and the SGHL (Fig. 17.7), creating a simple stitch (Fig. 17.8).

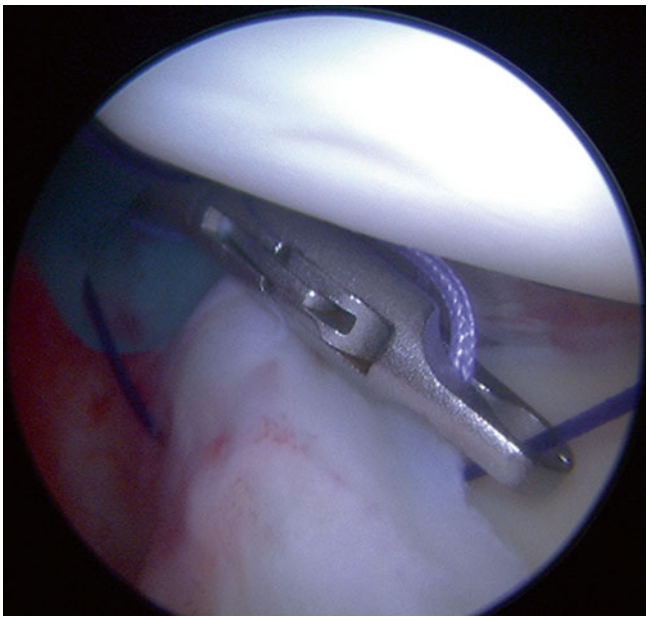


Fig. 17.5 The intrarticular end of this monofilament suture is retrieved through the antero-superior portal in order to shuttle one of the limbs of the sutures in the anchor through the labrum

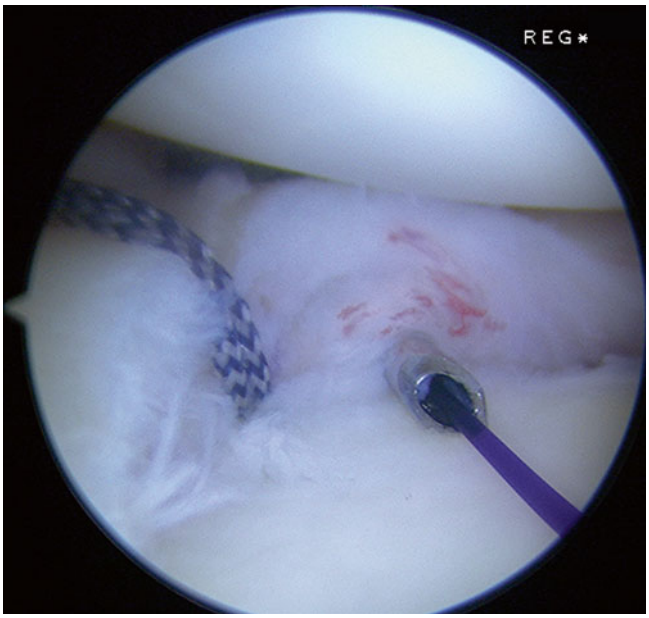


Fig. 17.7 The horizontal matress suture should not cross over point "A" (anterior edge of LHB on the labrum)

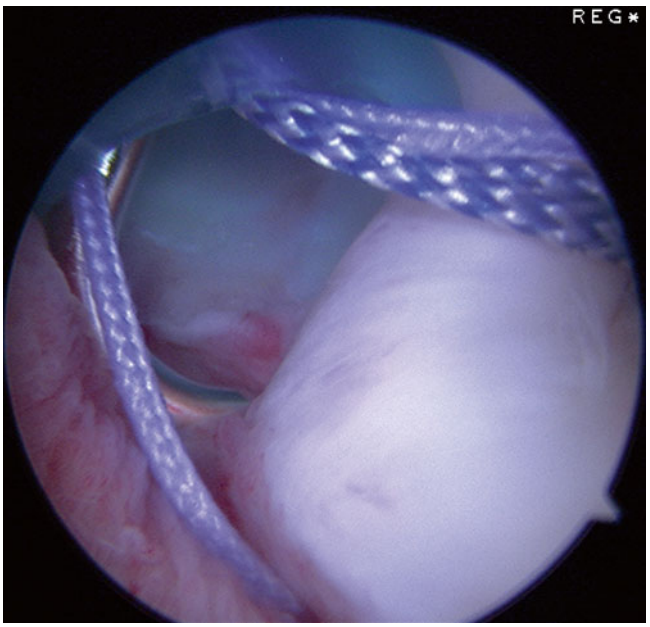


Fig. 17.6 The other limb is passed through the labrum adjacent to the biceps, a few millimeters anterior to the first one, thus creating a mattress stitch

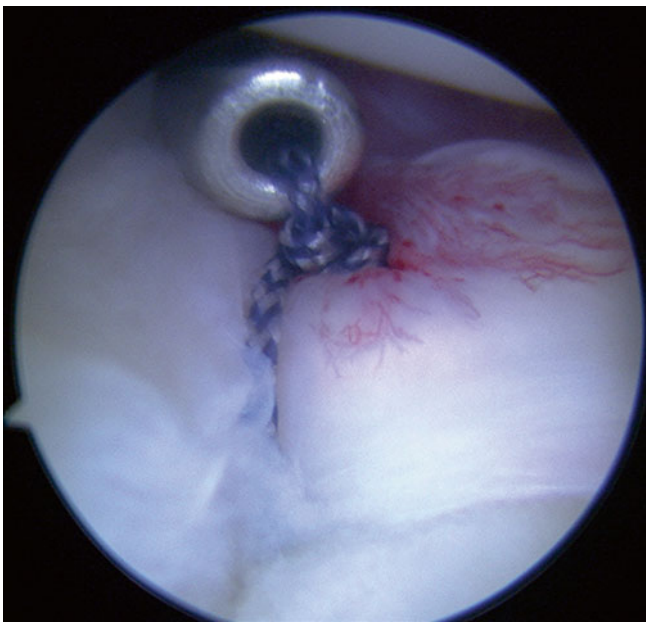


Fig. 17.8 Using the same suture passer technique, one of the limbs of the second suture of the anchor is passed through the superior labrum at the level of the insertion of the MGHL and the SGHL, creating a simple stitch

Finally, the biceps and labral stability were tested with a probe (Fig. 17.9).

We reported our above-described technique for isolated SLAP II lesion repair with a short-term follow-up [10]. Nine males (64.3 %) and five females aged 28.4 ± 6.6 years were included in this study. The mean follow-up was 12.7 ± 7.4 months (range, 5–24). The dominant arm was involved in 10 cases (71.4 %), while the right arm was involved in 78.6 % of the cases. Every single patient was assessed before and after surgery by the passive ROM and using the Constant score [12], the ASES score [33], the UCLA score, and the ROWE score. The O'Brien and the painful apprehension tests were used for clinical diagnosis [17]. All the patients underwent an arthro-MRI for imaging confirmation before surgery. For each enrolled patient, a

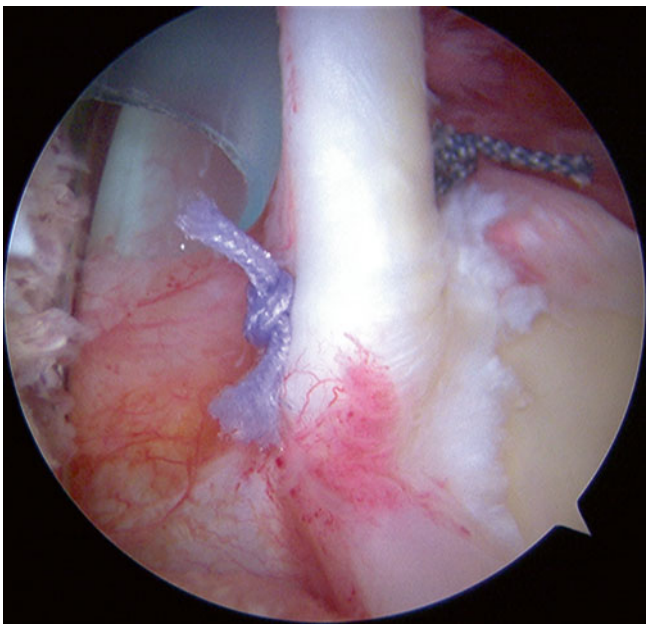


Fig. 17.9 Finally, the biceps and labral stability is tested with a probe

standardized form reporting demographic data (age, sex), time of follow-up, dominant arm (right/left), affected shoulder, passive ROM, Constant score [12], ASES score, UCLA score, ROWE score, O'Brien test, the time of the surgery (time zero), and the time of follow-up (6 months and final follow-up) was completed. For the comparison of the means at different times, a Student t-test for paired samples was used. A multiple logistic regression model to evaluate a relationship with age, sex, and sport of the examined parameters was performed. The level of significance was set at $p < 0.05$.

Of the 14 patients operated with this technique from 2011 to 2012, all the patients had isolated SLAP II lesions. The number and proportion of patients with positive O'Brien sign was 12 (85.7 %) at time zero and 1 (7.1 %) at 6 months and at final follow-up (chi-square = 15.2; $p < 0.0001$) (Table 17.1). Means of Constant [12], ASES [33], ROWE, and SST scores and VAS (visual analog scale for pain evaluation) statistically improved from time zero to 6 months and from 6 months to final follow-up (Table 17.2). The number

Table 17.1 Detailed list of the enrolled patients including sports, function, and symptoms before surgery

Enrolled patients	Elite throwing sport performed	Mean Constant score preop. ± SD	O'Brien sign	Apprehension and relocation test
14	4	64.6 ± 13.9	12	8

Table 17.2 Means values and standard deviations with statistical analysis of Constant, ASES, ROWE Scores; VAS; and SST at time zero, 6 months' follow-up, and at final follow-up

Scores	Time zero	Follow-up 6 months ± SD	Time zero versus 6 months	Final follow-up ± SD	6 months versus 12 months
Constant	64.6 ± 13.9	80.7 ± 25.1	$t = 1.9$	92.6 ± 11.8	$t = 6.5$
			$p = 0.04$		$p < 0.0001$
ASES	76.9 ± 22.4	100.6 ± 7.5	$t = 3.8$	108.3 ± 8.5	$t = 5.0$
			$p = 0.001$		$p = 0.001$
ROWE	53.6 ± 20.6	88.6 ± 10.1	$t = 7.4$	96.5 ± 7.2	$t = 7.8$
			$p < 0.0001$		$p < 0.0001$
VAS	5.7 ± 3.4	2.1 ± 1.5	$t = 6.4$	0.57 ± 0.93	$t = 5.7$
			$p < 0.0001$		$p < 0.0001$
SST	6.9 ± 2.1	8.5 ± 0.6	$t = 2.5$	9.1 ± 0.9	$t = 3.1$
			$p = 0.013$		$p = 0.0039$

Table 17.3 Means values and standard deviations (SD) with statistical analysis of ABD, AF, ER1, ER2, and IR at time zero, 6 months' follow-up, and at final follow-up

	Time zero	Follow-up 6 months	Time zero to 6 months	Final follow-up	6 months versus 12 months
ABD	90.7 ± 6.2	84.6 ± 12.8	n.s.	85.4 ± 12.8	n.s.
AF	167.9 ± 11.2	162.8 ± 26.1	n.s.	165.7 ± 27.1	$t = 2.3$
					$p = 0.02$
ER1	71.8 ± 17.7	58.9 ± 23.9	n.s.	60.3 ± 24.5	n.s.
ER2	85.7 ± 14	80.4 ± 14.2	n.s.	81.8 ± 14.6	n.s.
IR	66.4 ± 14.5	56.4 ± 19.8	$t = 1.9$	61.4 ± 22.1	$t = 2.9$
			$p = 0.04$		$p = 0.0065$

and proportion of patients who referred to practice sports was 12 (85.7 %) at time zero, 10 (71.4 %) at 6 months follow-up, and 10 (71.4 %) at final follow-up (chi-square = 0.6; n.s). Of the four patients who had participated in overhead agonistic athletics preoperatively (volleyball and tennis), all the four were able to return to their preinjury level. The mean value of passive abduction (ABD), external rotation with arm at side (ER1), and external rotation with 90° of abduction of the arm (ER2) did not differ in the three evaluations (n.s.). Anterior flexion of the arm (AF) and internal rotation of the arm (IR) were increased from 6 months to final follow-up ($t = 2.9$; $p = 0.0065$) (Table 17.3). The apprehension and relocation tests were positive in eight patients before surgery and negative in all the cases after surgery (chi-square = 13.3; $p = 0.003$). Clinical scores were better in male patients ($t = 2.7$ $p = 0.028$), if the dominant arm was involved ($t = 3.4$, $p = 0.010$) and in older patients (low significance) ($t = 2.3$ $p = 0.046$). Passive ROM was not reduced by surgery (Table 17.3). The main difference with a standard repair is that in the reported technique, a mattress stitch to reinsert the medial supratubercle origin of the biceps fibers, at the medial side of the biceps anchor, and a simple stitch anteriorly, through the superior labrum at the level of the insertion of the MGHL and the SGHL, are applied, thus stabilizing those ligaments. In the standard repair, two simple stitches are applied anteriorly and posteriorly to the biceps, and this can strangle the LHB and can create pain with loss of joint mobility [4]. In the reported technique, the anatomy is respected: the articular aspect of the superior labrum is loose and the medial side reinforced.

17.6 Rehabilitation and Return to Play

Repair of the SLAP II lesions has been shown to be a successful procedure in the young overhead athlete; nevertheless, recent literature reported that the ability to return to preinjury level of sports remains a concern [25, 28].

Postoperatively to our technique of SLAP II repair, the patients are protected in a sling in neutral rotation and 20° of abduction for 3 weeks. They are limited to early pendular shoulder exercises with a gradual progression of forward flexion from 90° to 150° over 6 weeks. Strengthening exercises begin 6 weeks after the operation in a progressive manner. A proprioceptive program of the scapulothoracic joint with a strengthening of the rotator cuff muscles must be associated. Patients are advised to avoid vigorous sports activities for 6 months after the operation. After the fourth month, stretching and strengthening exercises are improved and a progressive return to the throwing action can begin. Overhead athletes can return to their throwing activities 6 months after surgery. This kind of program can give good medium-/long-term outcomes [35].

References

1. Abbot AE, Li X, Busconi BD (2009) Arthroscopic treatment of concomitant superior labral anterior posterior (SLAP) lesions and rotator cuff tears in patients over the age of 45 years. Am J Sports Med 37:1358–1362
2. Arai R, Kobayashi M, Harada H, Tsukiyama H, Saji T, Toda Y, Hagiwara Y, Miura T, Matsuda S (2014) Anatomical study for SLAP lesion repair. Knee Surg Sports Traumatol Arthrosc 22:435–441
3. Baker CL 3rd, Romeo AA (2009) Combined arthroscopic repair of a type IV SLAP tear and Bankart lesion. Arthroscopy 25: 1045–1050
4. Baldini T, Snyder RL, Peacher G, Bach J, Mc Carty E (2009) Strength of single versus double anchor repair of type II SLAP lesions: a cadaveric study. Arthroscopy 25(11):1257–1260
5. Bedi A, Allen AA (2008) Superior labral lesions anterior to posterior-evaluation and arthroscopic management. Clin Sports Med 27:607–630
6. Boileau P, Parratte S, Chuinard C et al (2009) Arthroscopic treatment of isolated type II SLAP lesions. Am J Sports Med 37:929–936
7. Brockmeier SF, Voos JE, Williams RJ III, Altchek DW, Cordasco FA, Allen AA (2009) Outcomes after arthroscopic repair of type II SLAP lesions. J Bone Joint Surg Am 91(7):1595–1603
8. Burkhart SS, Morgan CD (1998) The peel-back mechanism: its role in producing and extending posterior type II SLAP lesions and its effect on SLAP repair rehabilitation. Arthroscopy 14(6):637–640
9. Castagna A, Nordenson U, Garofalo R, Karlsson J (2007) Minor shoulder instability. Arthroscopy 23(2):211–215
10. Castagna A, De Giorgi S, Garofalo R, Tafuri S, Conti M, Moretti B (2014) A new anatomic technique for type II SLAP lesions repair. Knee Surg Sports Traumatol Arthrosc Nov 21 [Epub ahead of print]
11. Cho HL, Lee CK, Hwang TH, Suh KT, Park JW (2010) Arthroscopic repair of combined Bankart and SLAP lesions: operative techniques and clinical results. Clin Orthop Surg 2:39–46
12. Constant CR, Murley AH (1987) A clinical method of functional assessment of the shoulder. Clin Orthop 214:160–164
13. Dierickx C, Ceccarelli E, Conti M, Vanlommel J, Castagna A (2009) Variations of the intra-articular portion of the long head of the biceps tendon: a classification of embryologically explained variations. J Shoulder Elbow Surg 18(4):556–565
14. El-Khoury GY, Kathol MH, Chandler JB, Albright JP (1986) Shoulder instability: impact of glenohumeral arthrotomography on treatment. Radiology 160:669–673
15. Franceschi F, Longo UG, Ruzzini L et al (2008) No advantages in repairing a type II superior labrum anterior and posterior (SLAP) lesion when associated with rotator cuff repair in patients over age 50. Am J Sports Med 36:247–253
16. Gorantia K, Gill C, Wright RW (2010) The outcome of type II SLAP repair: a systematic review. Arthroscopy 26(4):537–545
17. Hegedus EJ, Goode A, Campbell S, Morin A, Tamaddoni M, Moorman CT III, Cook C (2008) Physical examination tests of the shoulder: a systematic review with meta-analysis of individual tests. Br J Sports Med 42:80–92
18. Huber W, Putz R (1997) Periarticular fiber system of the shoulder joint. Arthroscopy 13(6):680–691
19. Jobe FW, Kvitne RS (1989) Shoulder pain in the overhand or throwing athlete. Orthop Rev 18:963–975
20. Karlsson J (2010) Physical examination tests are not valid for diagnosing SLAP tears: a review. Clin J Sport Med 20:134–135
21. Kim TK, Queale WS, Cosegarea AJ, McFarland EG (2003) Clinical features of the different types of SLAP lesions: an analysis of one hundred and thirty-nine cases superior labrum anterior posterior. J Bone Joint Surg Am 85A:66–71

22. Kim DS, Park HK, Park JH, Yoon WS (2012) Ganglion cyst of the spinoglenoid notch: comparison between SLAP repair alone and SLAP repair with cyst decompression. J Shoulder Elbow Surg 21:1456–1463
23. Li X, Lin TJ, Jager M, Price MD, Deangelis NA, Busconi BD, Brown MA (2010) Management of type II superior labrum anterior posterior lesions: a review of the literature. Orthop Rev 2:e 6. 2
24. Maffet MW, Gartsman GM, Moseley B (1995) Superior labrum-biceps tendon complex lesions of the shoulder. Am J Sports Med 23:93–98
25. Malal J, Khan Y, Farrar G, Waseem M (2013) Superior labral anterior and posterior lesions of the shoulder. Open Orthop J 6(7):356–360
26. Mc Cormick F, Bhatia S, Chalmers P, Gupta A, Verna N, Romeo AA (2014) The management of type II superior labral anterior to posterior injuries. Orthop Clin North Am 45:121–128
27. Morgan CD, Burkhart SS, Palmieri M, Gillespie M (1998) Type II SLAP lesions- three subtypes and their relationships to superior instability and rotator cuff tears. Arthroscopy 14:553–565
28. Neuman BJ, Boisvert CB, Reiter B, Lawson K, Ciccotti MG, Cohen SB (2011) Results of arthroscopic repair of type II superior labral anterior posterior lesions in overhead athletes: assessment of return to preinjury playing level and satisfaction. Am J Sports Med 39(9):1883–1888
29. Nishida K, Hashizume H, Toda K, Inoue H (1996) Histologic and scanning electron microscopic study of the glenoid labrum. J Shoulder Elbow Surg 5:132–138
30. Nordenson U, Garofalo R, Conti M, Linger E, Classon J, Karlsson J, Castagna A (2011) Minor or occult shoulder instability: an intra-articular pathology presenting with extra-articular subacromial impingement symptoms. Knee Surg Sports Traumatol Arthrosc 19(9):1570–1575
31. O'Brien SJ, Pagnani MJ, McGlynn SR, Fealy S, Wilson JB (1996) A new and effective test for diagnosing labral tears and acromioclavicular joint pathology. Presented at: 63rd annual meeting of the American Academy of Orthopaedic Surgeons, Atlanta, Feb 22
32. Ok JH, Kim YS, Kim JM, Yoon KS (2012) A new technique of arthroscopic fixation using double anchors for SLAP lesions. Knee Surg Sports Traumatol Arthrosc 20:1939–1946
33. Padua R, Padua L, Ceccarelli E, Bondi R, Alviti F, Castagna A (2010) Italian version of ASES questionnaire for shoulder assessment: cross-cultural adaptation and validation. Musculoskelet Surg 94(S1):S85–S90
34. Pouliart N, Somers K, Eid S, Gagey O (2007) Variations in the superior capsuloligamentous complex and description of a new ligament. J Shoulder Elbow Surg 16(7):821–836
35. Rose T, Hepp P, Korner C, Lill H (2002) Anterosuperior labrum lesions of the shoulder joint: pathogenesis, arthroscopic treatment and results. Knee Surg Sports Traumatol Arthrosc 10(5): 316–320
36. Savoie FH 3rd, Field LD, Atchinson S (2001) Anterior superior instability with rotator cuff tearing: SLAC lesion. Orthop Clin North Am 32(3):457–461
37. Schroder CP, Skare O, Stiris M, Gjengedal E, Uppheim G, Brox JI (2008) Treatment of labral tears with associated spinoglenoid cyst without cyst decompression. J Bone Joint Surg Am 90: 523–530
38. Skare Ø, Schrøder CP, Reikerås O, Mowinckel P, Brox JI (2010) Efficacy of labral repair, biceps tenodesis, and diagnostic arthroscopy for SLAP Lesions of the shoulder: a randomised controlled trial. BMC Musculoskelet Disord 11:228
39. Snyder SJ, Karzel RP, Del Pizzo W, Ferkel RD, Friedman MJ (1990) SLAP lesions of the shoulder. Arthroscopy 6:274–279
40. Snyder SJ, Banas MP, Karzel RP (1995) An analysis of 140 injuries to the superior glenoid labrum. J Shoulder Elbow Surg 4: 243–248
41. Takase K (2009) Risk of motion loss with combined Bankart and SLAP repairs. Orthopedics 32(8). doi:10.3928/01477447-20090624-05
42. Vangsness CT Jr, Jorgenson SS, Watson T, Johnson DL (1994) The origin of the long head of the biceps from the scapula and the glenoid labrum. An anatomical study of 100 shoulders. J Bone Joint Surg Br 76:951–954
43. Waldt S, Metz S, Burkhart A et al (2006) Variants of the superior labrum and labrobicipital complex: a comparative study of shoulder specimens using MR arthrography multislice CT arthrography and anatomical dissection. Eur Radiol 16:451–458

Shoulder Anterior Instability

18

Francesco Franceschi, Sebastiano Vasta,
Edoardo Franceschetti, Rocco Papalia,
and Vincenzo Denaro

18.1 Epidemiology

Anterior glenohumeral instability (GHI) is a common injury among athletes, with a higher incidence compared to the general population.

In the USA, the incidence rate of shoulder dislocation is 23.9 per 100,000 person-years in the general population presenting to the emergency departments [73], while athletes sustain 0.12 episodes of glenohumeral dislocation per 1,000 athlete exposures (meant as one athlete's participation in 1 practice or competition session without reference to duration of activity) [45]. A higher number of dislocations occur among contact athletes, being American football, ice hockey, and rugby the most injurious [45]. There are no differences in the overall dislocation occurrence with regard to gender when sports participation was analyzed per sexes [45]. In the general population, 46.8 % of all dislocations were in patients between 15 and 29 years of age [73].

18.2 Joint

Static and dynamic stabilizers guarantee the balance between shoulder mobility and stability. Proximal humeral shaft, humeral head and glenoid shape, labrum, negative intra-articular pressure, and capsular ligaments are static restraints. Cadaveric and in vivo studies showed the contribution that different elements of the capsule-ligamentous complex give to stability. When the arm is abducted and externally rotated, in the position of anterior apprehension, the primary restraint to anterior dislocation is the anterior band of the inferior glenohumeral ligament. On the contrary, the posterior band is essential in preventing posterior instability [44, 62, 66]. The rotator interval complex, especially the medial part represented mainly by the coracohumeral ligament, has a primary role in limiting inferior translation [30]. When the arm is at 45° of abduction and external rotation, the medial glenohumeral is a static restraint to both anterior and posterior translation, while the superior glenohumeral ligament is mainly involved in limiting inferior and anterior translation with the arm at the side [9]. The labrum acts by deepening the glenoid by 50 % [22], increasing therefore the anteroposterior stability by 20 % [36]. The glenoid has a pear shape, being the upper part 20 % narrower than the lower one. The humeral head has the shape of a sphere; therefore, just the 25–30 % of the glenohumeral surfaces are in contact [4]. Rotator cuff muscles, long head of the biceps, deltoid, and other periscapular muscles are dynamic stabilizers. They act compressing the humeral head to the glenoid therefore maintaining a concentric joint [18].

18.3 Injury Mechanism

Anterior dislocations account for about 95 % of instances, while posterior and inferior represent together less than 5 %. Typically, in case of an anterior shoulder dislocation in the athletes, the arm is abducted and externally rotated against an externally rotating force [23]. Less commonly, a forward fall creates a hyperflexion/external rotation glenohumeral position. Posterior dislocations usually come from a direct humeral head force while the arm is in adduction and internal rotation or in a fall with an extended and internally rotated arm [40]. In an epidemiologic study over a 4-year period on 131 subjects whom sustained a first-time shoulder dislocation, 86 % were contact or collision athletes [53].

Football is a sport in which there is a higher risk for shoulder dislocation: most of the injuries occur during running plays, and in all phases of play, injuries most frequently occur when the athlete is tackling, being tackled, and

F. Franceschi (✉) • S. Vasta • E. Franceschetti • R. Papalia
V. Denaro
Department of Orthopaedics and Trauma Surgery,
Campus Bio Medico di Roma,
Via Alvaro Del Portillo 200, Rome 00128, Italy
e-mail: f.franceschi@unicampus.it;
s.vasta@unicampus.it; e.franceschetti@unicampus.it;
r.papalia@unicampus.it

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_18,

blocking. For soccer and wrestling, the most common mechanism of dislocation is contact with another player or contact with the playing surface, with takedown in wrestling being responsible for about 50 % of shoulder dislocation. In basketball, the most common mechanism is contact with the playing apparatus for males, whereas contact with another player for females [32]. Rugby also exposes a high risk of shoulder dislocation, with an overall incidence rate (IR) of primary anterior shoulder dislocation in matches up to 3.2 per 1,000 layer-hours. The players who compose the front row have a significantly higher IR than those in other positions, and tackling is by far (67.6 %) the most frequent mechanism of all shoulder injuries [31]. In ice hockey, the IR of shoulder dislocation ranges between 8.6 and 21.9 % [56] and even worse among junior players (22 %) [59]. In a study on 24 professional hockey players presenting at the author's institution after a shoulder injury, 75 % had an anterior labral lesion/Bankart lesion [14].

18.4 Pathoanatomy

The most common lesion following an anterior traumatic dislocation is the Bankart lesion (Fig. 18.1), in which the anteroinferior capsulolabral complex is torn away from the glenoid rim.

This injury can also come with a bony fragment from the anteroinferior edge of the glenoid [61]. The detached labrum fails its deepening effect, therefore destabilizing the glenohumeral joint. This mechanism is increased when a bony lesion is also present (bony Bankart) (Fig. 18.2), since it contributes to decrease the glenoid surface. During a trauma in which the humeral head, while in abduction and external rotation, is forced anteriorly, a compression fracture can occur as the superior posterolateral aspect of the head hits the anterior glenoid rim (Fig. 18.3). This occurrence was identified by Hill and Sachs in 1940 [20], and it is present in 40–90 % of all anterior shoulder dislocation [61, 72]. When the defect on the superior posterolateral aspect of the humeral head engages the anterior glenoid rim, the lesion becomes an engaging Hill-Sachs (Fig. 18.4). Typically it happens with 90° of abduction and a various degree of external rotation [10, 47].

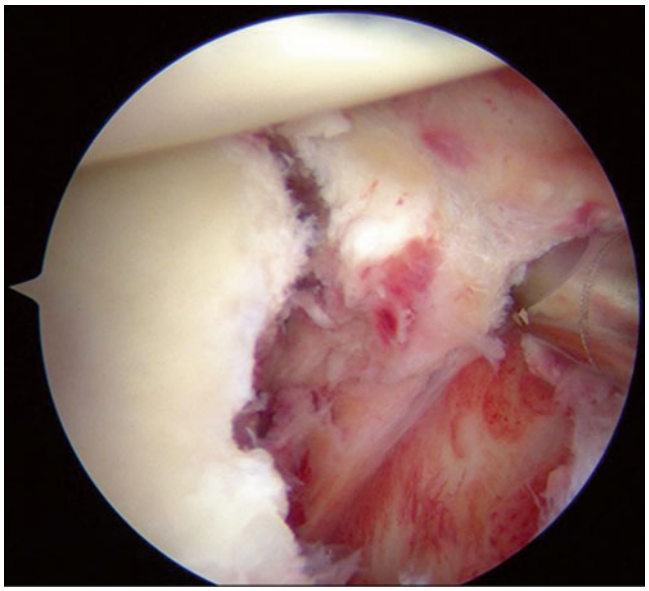


Fig. 18.2 Bony Bankart lesion

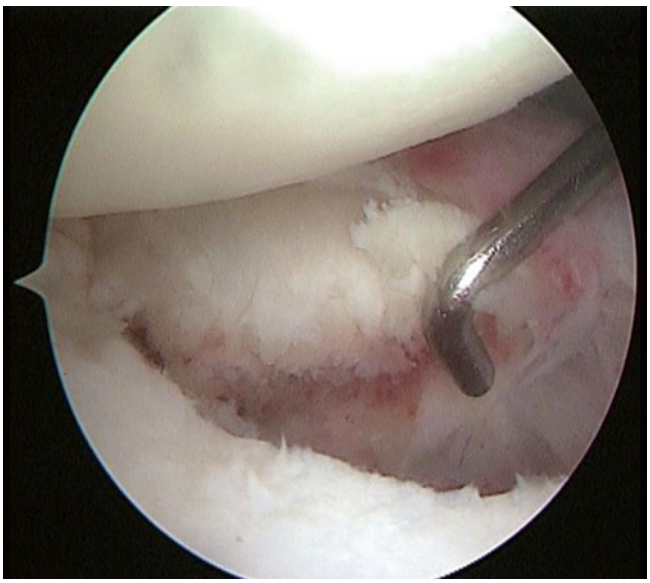


Fig. 18.1 Bankart lesion

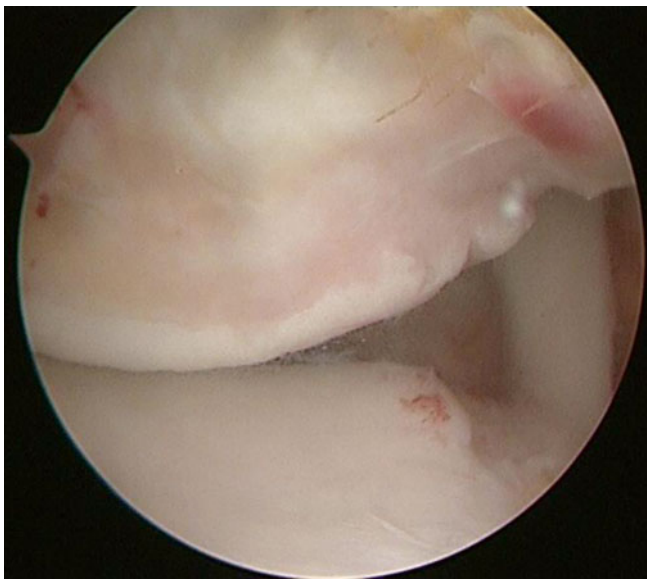


Fig. 18.3 Hill-Sachs lesion

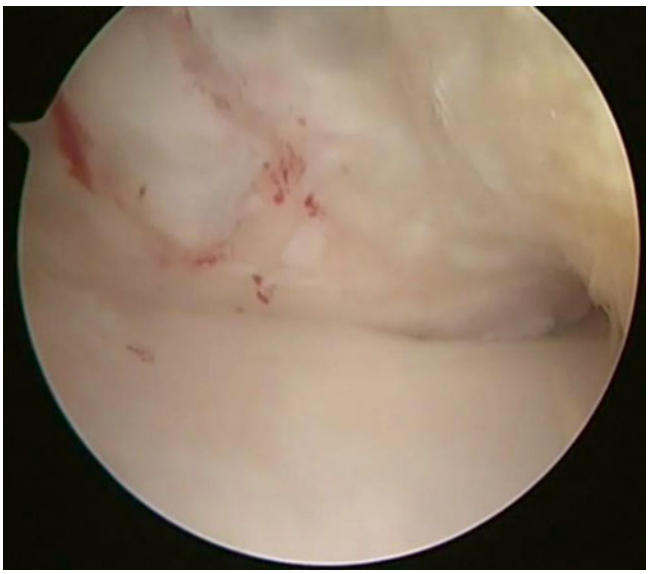


Fig. 18.4 Engaging Hill-Sachs lesion

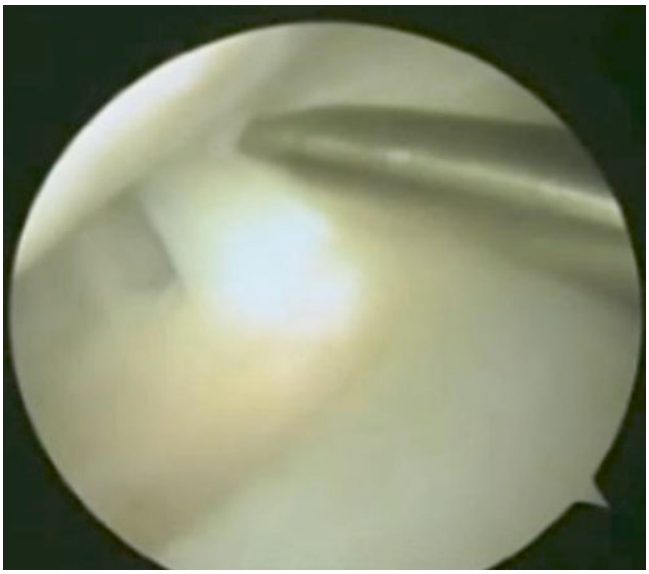


Fig. 18.6 Humeral avulsion of the glenohumeral ligament (HAGL)

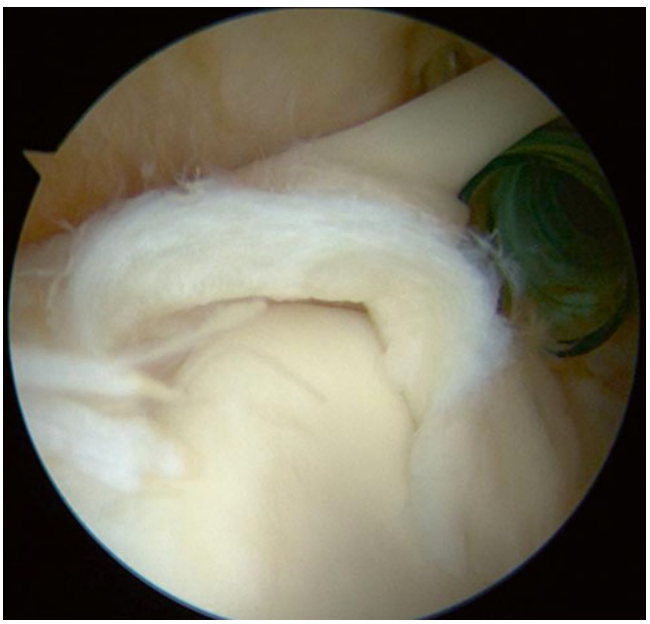


Fig. 18.5 Superior Labrum Anterior to Posterior Lesion (SLAP)

Although less common, other structures can be injured following an anterior shoulder dislocation. Specially in overhead/throwing athletes, the superior labrum can detach at the biceps tendon/labrum complex as described by Andrews et al. in 1985 [1], and successively, Snyder named this injury Superior Anterior to Posterior Lesion (SLAP) (Fig. 18.5) and classified it into four types [58].

Humeral avulsion of the glenohumeral ligament (HAGL) (Fig. 18.6) is another well-described cause of post-traumatic anterior shoulder instability, accounting up to 9 % of the cases [64, 70]. It has been also described as an association between anterior shoulder instability and a tear of the rotator interval complex, as described by Rowe and Zarins [52].

18.5 Clinical and Diagnostic Examination

Examination starts with a careful history. Even in the acute setting, a detailed history on the trauma pattern can help in diagnosing the underlying injury. Moreover, a complete neurovascular assessment is mandatory in the acute dislocation, since an axillary nerve injury occurs in 5–35 % of first injury [49], and a gross physical examination should also evaluate the presence of obvious deformity, open wounds, and limited range of motion. In the subacute setting, the examiner should focus on age at the first dislocation, mechanism of injury, type and level of played sport, any further dislocation and shoulder positions that lead to dislocation, and the total number of episodes. Muscle status should be assessed, together with the presence of deformities and sensory-motor impairment. Examination should also encompass generalized ligamentous laxity test. The main essential differential diagnosis should focus on traumatic versus atraumatic shoulder instability. The first one follows a traumatic injury, usually is unidirectional, and frequently needs surgery to be fixed (TUBS), and the second one is the result of a generalized ligament laxity, shows a multidirectional instability, and is mainly addressed by physical therapy (AMBRI) [63].

The apprehension test is considered positive when elicits apprehension while the patient is supine and the arm is passively abducted and externally rotated both to 90°. Sometimes patients experience pain, although the pain alone has a lesser predictive value [52]. If the patient complains for discomfort

and apprehension while the shoulder is at 90° of abduction and external rotation, the examiner can push posteriorly the humeral head. If this maneuver lowers the apprehension, the so-called relocation test is considered positive, as described by Jobe et al. [29]. When the examiner removes suddenly his hand after the relocation test, the patient could experience the symptoms. This is defined as a positive surprise test [57]. A study evaluating the three tests [37] showed that the surprise test was the single most accurate test (sensitivity = 63.89 %; specificity = 98.91 %) followed by the apprehension test (sensitivity = 52.78 %; specificity = 98.91 %), the relocation adds few value to the examination, being the less accurate (sensitivity = 45.83 %; specificity = 54.35 %).

The anterior and posterior drawer tests [16] and the load and shift tests [41] are used to assess the stability of the glenohumeral joint. The drawer tests are considered positive if the humeral head has an increased translation compared to the contralateral shoulder. The load and shift test is considered positive when the humeral head could be shifted anteriorly off the glenoid.

Radiological evaluation should include routine X-rays in three views, a true antero-posterior view, the West Point Axillary view, and the Apical Oblique view. This set of view allows to evaluate the anteroinferior glenoid rim. When a bone loss is suspected, routine X-rays should be supplied with CT-scan that allows for a quantification of the glenoid bone loss. To this purpose, different methods have been described [24, 54, 60]. MRI should be considered to evaluate soft tissue injuries, including labrum, rotator cuff, and glenohumeral ligaments, and for these latter, a better view could be achieved by using the ABER position [55]. The intra-articular injection of gadolinium could be used to enhance the diagnostic power (arthro-MRI) once the post-traumatic hemarthrosis in the acute setting has been solved. Arthro-MRI identifies labral tears with a sensitivity of 88–96 % and a specificity of 91–98 % [69].

18.6 Treatment Strategy

The treatment of anterior shoulder instability is a heavily debated issue. A lot of factors enter in the decision-making process: age and level of activity of the patient, the kind of sport and the role of the athlete (overhead/thrower vs non-overhead/nonthrower), the type of lesion (soft tissue or soft tissue and bony lesion), the number of dislocations, and the timing with respect to sport season.

The athlete incurring in a shoulder dislocation in the mid-season, who wish to finish the season, has no alternatives than nonoperative management. But no all the athletes are amenable to be treated nonsurgically. Although evidences are little, the following criteria have been proposed: (1) little or no pain, (2) patient subjectivity, (3) near normal range of motion, (4) near normal strength, (5) normal functional ability, and (6) normal sports-specific skills. When the strength is 80–90 % of normal and mild apprehension, the athlete could return to play with an abduction and external rotation limiting brace that can be removed once the symptoms are gone [40].

18.6.1 Nonoperative Management

Immobilization, physical therapy, and bracing, with a delayed return to activity, are the basis of nonoperative management. The duration and position of the immobilization are controversial. Itoi et al. used the MRI to assess if there were differences in immobilizing the arm internally or externally rotated in patients affected by a Bankart lesion. They found that immobilization in internal rotation displaces the labrum and immobilization in external rotation better approximates the Bankart lesion [26]. In a later prospective study, comparing patients randomly assigned to immobilization in either internal or external rotation, Itoi and colleagues found a significantly lower recurrence rate in the external rotation group than in the internal rotation group (26 % vs 42 %), with a relative risk reduction of 38.2 %. In the subgroup of patients who were 30 years of age or younger, the relative risk reduction was even more substantial (46.1 %) [25]. However, in a more recent prospective randomized study on 188 patients with a primary anterior traumatic dislocation of the shoulder randomly assigned to immobilization in either internal rotation or external rotation for 3 weeks [35], the authors showed that the recurrence rate was 24.7 % in the internal rotation group and 30.8 % in the external rotation group ($p = 0.36$).

The duration of immobilization is even more controversial than position. The duration ranges from 3 to 6 weeks and the recurrence rate varies from 10 to 100 % [21, 39].

18.6.2 Operative Management

Although on the basis of weak evidences, there is general agreement among surgeons that failure of nonoperative management, recurrent dislocation, large or engaging Hill-Sachs lesions, and glenoid bone loss greater than 20 % are factors pushing towards surgery [67]. Outcomes from both nonoperative [39, 51] and operative [53] treatments showed that age is a primary risk factor for recurrence. In the acute setting, indications to surgery are the presence of a proximal humerus fracture requiring surgery, irreducible dislocation or interposed tissue or nonconcentric reduction, and instability during sports-specific drills [46].

Studies comparing nonoperative to operative treatment of initial traumatic anterior dislocation showed that the recurrence rate is higher for the nonoperative option [7, 28, 34].

Early surgical stabilization compared to nonoperative treatment leads to better outcomes and smaller recurrence rate, especially for younger and more active subjects. Both at short-term follow-up (7 % vs 46 %) and at longer-term follow-up (10 % vs 58 %) operative management is associated with a significantly lower rate of recurrent instability for primary anterior shoulder dislocation [8].

Nearly 90 % of shoulder stabilization surgeries are arthroscopically performed, while a significant decline in the incidence of open Bankart repair has been observed in the USA [74]. In 2010, a meta-analysis on 6 studies, for a total number of patients of 501, 234 suture anchors and 267 open, concluded that arthroscopic repair using suture anchors results in similar recurrence and reoperation rate compared to open Bankart repair [50]. A recent systematic review of level I to IV studies showed that arthroscopic suture anchor and open Bankart techniques for anterior shoulder instability yield comparable results in terms of recurrence rate (arthroscopic suture anchor 8.5 %, open Bankart repair 8 %), clinical outcome scores, return to play (arthroscopic suture anchor 87 %, open Bankart repair 89 %), and incidence of postoperative osteoarthritis [19].

A recently published systematic review has shown that there is no significant difference in recurrence rate of instability among open Bankart procedures (5.5 %), Bristow-Latarjet procedures (14.3 %), and arthroscopic Bankart procedures (14.7 %). The open Bankart repair yielded to the lowest recurrence rate, clustering the shoulders by the amount of bone loss or the presence of an engaging Hill-Sachs, showed that for glenoid bone loss lesser than 30 % or non-engaging/non-severe Hill-Sachs lesions, open Bankart repair was successful in 100 % of patients without recurrences. Conversely for greater than 30 % glenoid bone loss or engaging or severe Hill-Sachs, open Bakart repair yielded 57.1 % of recurrences, while for the same conditions, the Bristow-Latarjet achieved a 14.3 % rate of recurrent instability [15]. Balg and Boileau assessed that bone loss (both at the glenoid or at the humeral side), patient age under 20 years at the time of surgery, contact sports or overhead activity, and shoulder hyperlaxity are clear risk factors for recurrence after arthroscopic Bankart repair [3, 6]. Therefore, they successively developed the "instability severity index score," a scoring system that takes into account the previously named risk factors and determines which patient would be better treated by an open procedure [3]. Failed shoulder stabilization procedures in an athlete are always a tough issue for the orthopedic surgeon. Causes of failure can be relative to a new trauma or an underestimated lesion at the time of the primary treatment. Several studies support the arthroscopic Bankart repair as a feasible option even in the revision setting [2, 5, 12, 33, 42, 43, 48]. But revision Bankart repair generally yields to lower results compared to primary repair in terms of recurrence rate, return to play, and clinical outcomes [17]. With regard to bony defects, there is general agreement that glenoid bone loss greater than 25 % should be addressed by bony procedures to widen the glenohumeral articular surface [38]. But less agreement there is about bipolar bone loss lesions when the glenoid lesion is lesser than 25 % also because of the lack of a shared quantification method for humeral-sided bone loss. Itoi and colleagues in 2007 introduced a new concept, the glenoid track [71]. When the arm is abducted, the contact zone between humeral head and glenoid shifts from the inferomedial to the superolateral portion of the posterior aspect of the humeral head; that area is named glenoid track. This concept is consistent to the one by Burkhart and De Beer [10] about engaging versus non-engaging Hill-Sachs lesions. Combining these concepts, Di Giacomo, Itoi, and Burkhart recently proposed to divide the lesions into "on track" and "off track" [13]. In the presence of a Hill-Sachs, if the medial margin of the lesion falls inside the glenoid track, it will be on track and therefore will not engage. On the contrary, if the medial edge of the lesion falls medially to the margin of the glenoid track, the Hill-Sachs will be off track and an engaging lesion. The authors proposed that for off-track lesions, regardless of the amount of the glenoid defect, a procedure addressing the humeral head should be considered [13] (Fig. 18.7). An increasing number of studies support the concept of the glenoid track as a powerful method in the diagnostic and treatment approach [65].

18.7 Rehabilitation and Return to Play

Rehabilitation after surgical treatment is tailored for each athlete according to the type of surgery he underwent and the role he plays. However, the early phases are shared despite the nonoperative or surgical treatment and the sporting activity. A sling during the first 2 weeks allows for soft tissue protection, reduction of inflammation, and healing. Limited passive and gentle ranges of motion (ROM) are useful during that period to avoid the risk of shoulder stiffness. Usually from the third/fourth week, ROM are gradually increased, and isotonic, closed-chain exercises for rotator cuff muscles and other periscapular stabilizers are started, in order to favor joint stability. After four postoperative weeks, the sling is discontinued, and the patient can sleep with a pillow to keep the arm at 45° of abduction. Successively, the goals of the rehabilitation are to recover full ROM and increase the muscular strength and the joint neuromuscular control. The final phase is targeted to recover specific sports drills. Progression to advanced phases of the rehabilitation is based on pain, ROM gain, strength of the stabilizing periscapular muscles, and joint control. On average, a 4-month period is necessary for return to sport [27]. Usually athletes are allowed to return to sport if they show a comparable motion, strength, and stability to the contralateral side during specific sports drills [11, 68].

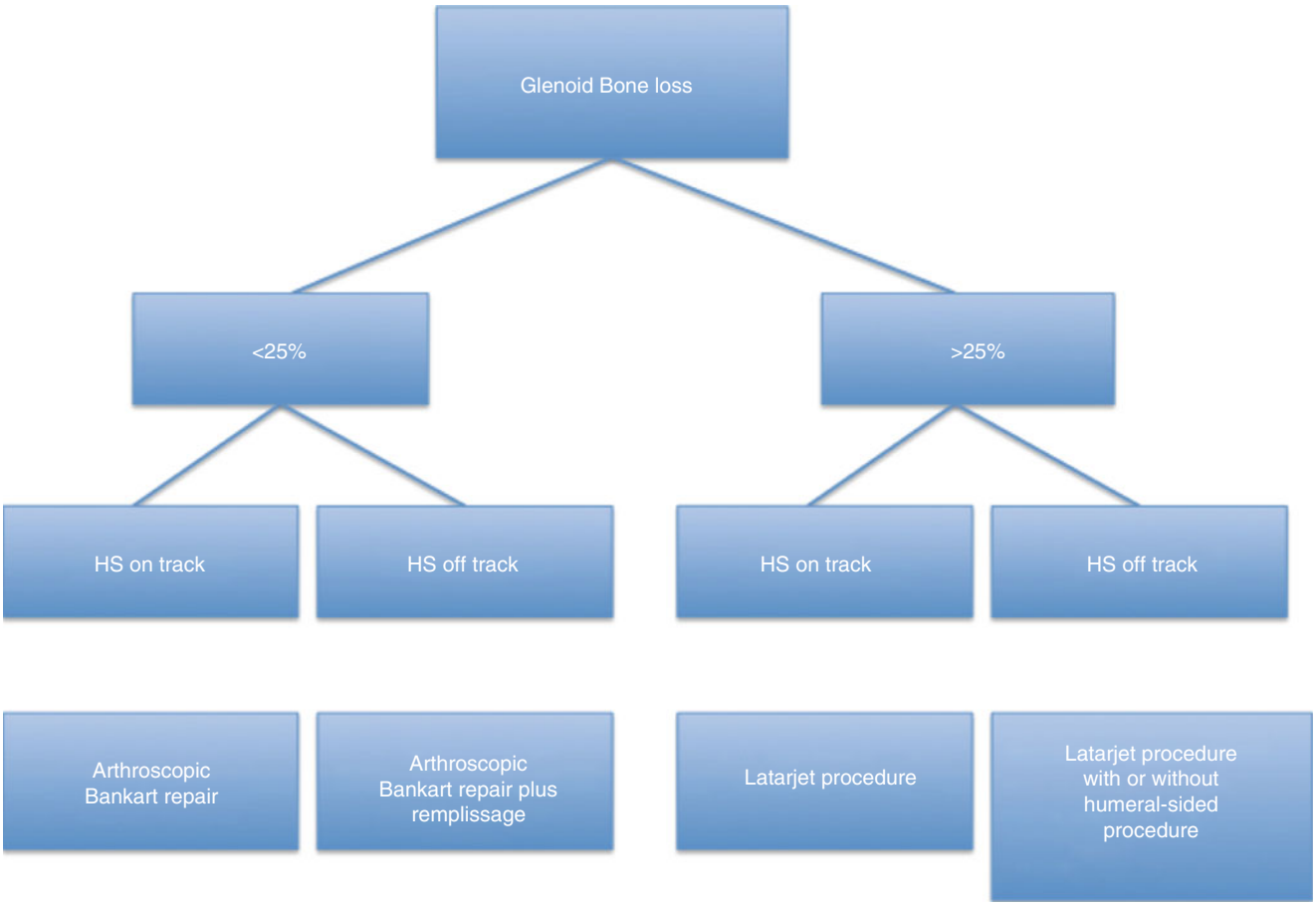


Fig. 18.7 Algorithm of treatment for bipolar bone loss lesions. HS Hill-Sachs [13]

References

1. Andrews JR, Carson WG Jr, McLeod WD (1985) Glenoid labrum tears related to the long head of the biceps. Am J Sports Med 13(5):337–341
2. Arce G, Arcuri F, Ferro D, Pereira E (2012) Is selective arthroscopic revision beneficial for treating recurrent anterior shoulder instability? Clin Orthop Relat Res 470(4):965–971. doi:10.1007/s11999-011-2001-0
3. Balg F, Boileau P (2007) The instability severity index score. A simple pre-operative score to select patients for arthroscopic or open shoulder stabilisation. J Bone Joint Surg Br 89(11):1470–1477. doi:10.1302/0301-620X.89B11.18962
4. Bigliani LU, Kelkar R, Flatow EL, Pollock RG, Mow VC (1996) Glenohumeral stability. Biomechanical properties of passive and active stabilizers. Clin Orthop Relat Res 330:13–30
5. Boileau P, Richou J, Lisai A, Chuinard C, Bicknell RT (2009) The role of arthroscopy in revision of failed open anterior stabilization of the shoulder. Arthroscopy J Arthroscopic Relat Surg Off Pub Arthroscopy Assoc North Am Int Arthroscopy Assoc 25(10):1075–1084. doi:10.1016/j.arthro.2009.04.073
6. Boileau P, Villalba M, Hery JY, Balg F, Ahrens P, Neyton L (2006) Risk factors for recurrence of shoulder instability after arthroscopic Bankart repair. J Bone Joint Surg Am 88(8):1755–1763. doi:10.2106/JBJS.E.00817
7. Bottoni CR, Wilckens JH, DeBerardino TM, D'Alleyrand JC, Rooney RC, Harpstrite JK, Arciero RA (2002) A prospective, randomized evaluation of arthroscopic stabilization versus nonoperative treatment in patients with acute, traumatic, first-time shoulder dislocations. Am J Sports Med 30(4):576–580
8. Brophy RH, Marx RG (2009) The treatment of traumatic anterior instability of the shoulder: nonoperative and surgical treatment. Arthroscopy J Arthroscopic Relat Surg Off Pub Arthroscopy Assoc North Am Int Arthroscopy Assoc 25(3):298–304. doi:10.1016/j.arthro.2008.12.007
9. Burkart AC, Debski RE (2002) Anatomy and function of the glenohumeral ligaments in anterior shoulder instability. Clin Orthop Relat Res 400:32–39
10. Burkhart SS, De Beer JF (2000) Traumatic glenohumeral bone defects and their relationship to failure of arthroscopic Bankart repairs: significance of the inverted-pear glenoid and the humeral engaging Hill-Sachs lesion. Arthroscopy J Arthroscopic Relat Surg Off Pub Arthroscopy Assoc North Am Int Arthroscopy Assoc 16(7):677–694
11. Burns TC, Owens BD (2010) Management of shoulder instability in in-season athletes. Phys Sports Med 38(3):55–60. doi:10.3810/psm.2010.10.1808
12. Creighton RA, Romeo AA, Brown FM Jr, Hayden JK, Verma NN (2007) Revision arthroscopic shoulder instability repair. Arthroscopy J Arthroscopic Relat Surg Off Pub Arthroscopy Assoc North Am Int Arthroscopy Assoc 23(7):703–709. doi:10.1016/j.arthro.2007.01.021
13. Di Giacomo G, Itoi E, Burkhart SS (2014) Evolving concept of bipolar bone loss and the Hill-Sachs lesion: from "engaging/non-engaging" lesion to "on-track/off-track" lesion. Arthroscopy J

Arthroscopic Relat Surg Off Pub Arthroscopy Assoc North Am Int Arthroscopy Assoc 30(1):90–98. doi:10.1016/j.arthro.2013.10.004
14. Dwyer T, Petrera M, Bleakney R, Theodoropoulos JS (2013) Shoulder instability in ice hockey players: incidence, mechanism, and MRI findings. Clin Sports Med 32(4):803–813. doi:10.1016/j.csm.2013.07.013
15. Friedman LG, Griesser MJ, Miniaci AA, Jones MH (2014) Recurrent instability after revision anterior shoulder stabilization surgery. Arthroscopy J Arthroscopic Relat Surg Off Pub Arthroscopy Assoc North Am Int Arthroscopy Assoc 30(3):372–381. doi:10.1016/j.arthro.2013.11.019
16. Gerber C, Ganz R (1984) Clinical assessment of instability of the shoulder. With special reference to anterior and posterior drawer tests. J Bone Joint Surg Br 66(4):551–556
17. Gwathmey FW Jr, Warner JJ (2013) Management of the athlete with a failed shoulder instability procedure. Clin Sports Med 32(4):833–863. doi:10.1016/j.csm.2013.07.016
18. Halder AM, Kuhl SG, Zobitz ME, Larson D, An KN (2001) Effects of the glenoid labrum and glenohumeral abduction on stability of the shoulder joint through concavity-compression: an in vitro study. J Bone Joint Surg Am 83-A(7):1062–1069
19. Harris JD, Gupta AK, Mall NA, Abrams GD, McCormick FM, Cole BJ, Bach BR Jr, Romeo AA, Verma NN (2013) Long-term outcomes after Bankart shoulder stabilization. Arthroscopy J Arthroscopic Relat Surg Off Pub Arthroscopy Assoc North Am Int Arthroscopy Assoc 29(5):920–933. doi:10.1016/j.arthro.2012.11.010
20. Hill HA, Sachs M (1940) The grooved defect of the humeral head: a frequently unrecognized complication of dislocations of the shoulder joint. Radiology 35:690–700
21. Hovelius L, Eriksson K, Fredin H, Hagberg G, Hussenius A, Lind B, Thorling J, Weckstrom J (1983) Recurrences after initial dislocation of the shoulder. Results of a prospective study of treatment. J Bone Joint Surg Am 65(3):343–349
22. Howell SM, Galinat BJ (1989) The glenoid-labral socket. A constrained articular surface. Clin Orthop Relat Res 243:122–125
23. Hudson VJ (2010) Evaluation, diagnosis, and treatment of shoulder injuries in athletes. Clin Sports Med 29(1):19–32. doi:10.1016/j.csm.2009.09.003, table of contents
24. Huysmans PE, Haen PS, Kidd M, Dhert WJ, Willems JW (2006) The shape of the inferior part of the glenoid: a cadaveric study. J Shoulder Elbow Surg Am Shoulder Elbow Surg [et al] 15(6):759–763. doi:10.1016/j.jse.2005.09.001
25. Itoi E, Hatakeyama Y, Sato T, Kido T, Minagawa H, Yamamoto N, Wakabayashi I, Nozaka K (2007) Immobilization in external rotation after shoulder dislocation reduces the risk of recurrence. A randomized controlled trial. J Bone Joint Surg Am 89(10):2124–2131. doi:10.2106/JBJS.F.00654
26. Itoi E, Sashi R, Minagawa H, Shimizu T, Wakabayashi I, Sato K (2001) Position of immobilization after dislocation of the glenohumeral joint. A study with use of magnetic resonance imaging. J Bone Joint Surg Am 83-A(5):661–667
27. Jaggi A, Lambert S (2010) Rehabilitation for shoulder instability. Br J Sports Med 44(5):333–340. doi:10.1136/bjsm.2009.059311
28. Jakobsen BW, Johannsen HV, Suder P, Sojbjerg JO (2007) Primary repair versus conservative treatment of first-time traumatic anterior dislocation of the shoulder: a randomized study with 10-year follow-up. Arthroscopy J Arthroscopic Relat Surg Off Pub Arthroscopy Assoc North Am Int Arthroscopy Assoc 23(2):118–123. doi:10.1016/j.arthro.2006.11.004
29. Jobe FW, Kvitne RS, Giangarra CE (1989) Shoulder pain in the overhand or throwing athlete. The relationship of anterior instability and rotator cuff impingement. Orthop Rev 18(9):963–975
30. Jost B, Koch PP, Gerber C (2000) Anatomy and functional aspects of the rotator interval. J Shoulder Elbow Surg Am Shoulder Elbow Surg [et al] 9(4):336–341. doi:10.1067/mse.2000.106746
31. Kawasaki T, Ota C, Urayama S, Maki N, Nagayama M, Kaketa T, Takazawa Y, Kaneko K (2014) Incidence of and risk factors for traumatic anterior shoulder dislocation: an epidemiologic study in high-school rugby players. J Shoulder Elbow Surg Am Shoulder Elbow Surg [et al]. doi:10.1016/j.jse.2014.05.007
32. Kerr ZY, Collins CL, Pommering TL, Fields SK, Comstock RD (2011) Dislocation/separation injuries among US high school athletes in 9 selected sports: 2005–2009. Clin J Sport Med Off J Canadian AcadSportMed21(2):101–108.doi:10.1097/JSM.0b013e31820bd1b6
33. Kim SH, Ha KI, Kim YM (2002) Arthroscopic revision Bankart repair: a prospective outcome study. Arthroscopy J Arthroscopic Relat Surg Off Pub Arthroscopy Assoc North Am Int Arthroscopy Assoc 18(5):469–482. doi:10.1053/jars.2002.32230
34. Kirkley A, Werstine R, Ratjek A, Griffin S (2005) Prospective randomized clinical trial comparing the effectiveness of immediate arthroscopic stabilization versus immobilization and rehabilitation in first traumatic anterior dislocations of the shoulder: long-term evaluation. Arthroscopy J Arthroscopic Relat Surg Off Pub Arthroscopy Assoc North Am Int Arthroscopy Assoc 21(1):55–63. doi:10.1016/j.arthro.2004.09.018
35. Liavaag S, Brox JI, Pripp AH, Enger M, Soldal LA, Svenningsen S (2011) Immobilization in external rotation after primary shoulder dislocation did not reduce the risk of recurrence: a randomized controlled trial. J Bone Joint Surg Am 93(10):897–904. doi:10.2106/JBJS.J.00416
36. Lippitt SB, Vanderhooft JE, Harris SL, Sidles JA, Harryman DT 2nd, Matsen FA 3rd (1993) Glenohumeral stability from concavity-compression: a quantitative analysis. J Shoulder Elbow Surg Am Shoulder Elbow Surg [et al] 2(1):27–35. doi:10.1016/S1058-2746(09)80134-1
37. Lo IK, Nonweiler B, Woolfrey M, Litchfield R, Kirkley A (2004) An evaluation of the apprehension, relocation, and surprise tests for anterior shoulder instability. Am J Sports Med 32(2):301–307
38. Lynch JR, Clinton JM, Dewing CB, Warme WJ, Matsen FA 3rd (2009) Treatment of osseous defects associated with anterior shoulder instability. J Shoulder Elbow Surg Am Shoulder Elbow Surg [et al] 18(2):317–328. doi:10.1016/j.jse.2008.10.013
39. Marans HJ, Angel KR, Schemitsch EH, Wedge JH (1992) The fate of traumatic anterior dislocation of the shoulder in children. J Bone Joint Surg Am 74(8):1242–1244
40. McCarty EC, Ritchie P, Gill HS, McFarland EG (2004) Shoulder instability: return to play. Clin Sports Med 23(3):335–351, vii–viii. doi:10.1016/j.csm.2004.02.004
41. McFarland EG, Torpey BM, Curl LA (1996) Evaluation of shoulder laxity. Sports Med 22(4):264–272
42. Millar NL, Murrell GA (2008) The effectiveness of arthroscopic stabilisation for failed open shoulder instability surgery. J Bone Joint Surg Br 90(6):745–750. doi:10.1302/0301-620X.90B6.20018
43. Neri BR, Tuckman DV, Bravman JT, Yim D, Sahajpal DT, Rokito AS (2007) Arthroscopic revision of Bankart repair. J Shoulder Elbow Surg Am Shoulder Elbow Surg [et al] 16(4):419–424. doi:10.1016/j.jse.2006.05.016
44. Ovesen J, Nielsen S (1985) Stability of the shoulder joint. Cadaver study of stabilizing structures. Acta Orthop Scand 56(2):149–151
45. Owens BD, Agel J, Mountcastle SB, Cameron KL, Nelson BJ (2009) Incidence of glenohumeral instability in collegiate athletics. AmJSportsMed37(9):1750–1754.doi:10.1177/0363546509334591
46. Owens BD, Dickens JF, Kilcoyne KG, Rue JP (2012) Management of mid-season traumatic anterior shoulder instability in athletes. J Am Acad Orthop Surg 20(8):518–526. doi:10.5435/JAAOS-20-08-518
47. Palmer I, Widen A (1948) The bone block method for recurrent dislocation of the shoulder joint. J Bone Joint Surg Br 30B(1):53–58
48. Patel RV, Apostle K, Leith JM, Regan WD (2008) Revision arthroscopic capsulolabral reconstruction for recurrent instability of the shoulder. J Bone Joint Surg Br 90(11):1462–1467. doi:10.1302/0301-620X.90B11.21072

49. Perlmutter GS, Apruzzese W (1998) Axillary nerve injuries in contact sports: recommendations for treatment and rehabilitation. Sports Med 26(5):351–361
50. Petrera M, Patella V, Patella S, Theodoropoulos J (2010) A meta-analysis of open versus arthroscopic Bankart repair using suture anchors. Knee Surg Sports Traumatol Arthrosc Off J ESSKA 18(12):1742–1747. doi:10.1007/s00167-010-1093-5
51. Rowe CR (1980) Acute and recurrent anterior dislocations of the shoulder. Orthop Clin North Am 11(2):253–270
52. Rowe CR, Zarins B (1981) Recurrent transient subluxation of the shoulder. J Bone Joint Surg Am 63(6):863–872
53. Sachs RA, Lin D, Stone ML, Paxton E, Kuney M (2007) Can the need for future surgery for acute traumatic anterior shoulder dislocation be predicted? J Bone Joint Surg Am 89(8):1665–1674. doi:10.2106/JBJS.F.00261
54. Saito H, Itoi E, Sugaya H, Minagawa H, Yamamoto N, Tuoheti Y (2005) Location of the glenoid defect in shoulders with recurrent anterior dislocation. Am J Sports Med 33(6):889–893. doi:10.1177/0363546504271521
55. Sanders TG, Zlatkin M, Montgomery J (2010) Imaging of glenohumeral instability. Semin Roentgenol 45(3):160–179. doi:10.1053/j.ro.2009.12.008
56. Sim FH, Simonet WT, Melton LJ 3rd, Lehn TA (1988) Ice hockey injuries. Am J Sports Med 16(Suppl 1):S86–S96
57. Slliman JK, Hawkins R (1994) Clinical examination of the shoulder complex. In: Andrews JR (ed) The Athlete's shoulder. Churchill Livingstone, New York, pp 45–58
58. Snyder SJ, Karzel RP, Del Pizzo W, Ferkel RD, Friedman MJ (1990) SLAP lesions of the shoulder. Arthroscopy J Arthroscopic Relat Surg Off Pub Arthroscopy Assoc North Am Int Arthroscopy Assoc 6(4):274–279
59. Stuart MJ, Smith A (1995) Injuries in junior a ice hockey. A three-year prospective study. Am J Sports Med 23(4):458–461
60. Sugaya H, Moriishi J, Dohi M, Kon Y, Tsuchiya A (2003) Glenoid rim morphology in recurrent anterior glenohumeral instability. J Bone Joint Surg Am 85-A(5):878–884
61. Taylor DC, Arciero RA (1997) Pathologic changes associated with shoulder dislocations. Arthroscopic and physical examination findings in first-time, traumatic anterior dislocations. Am J Sports Med 25(3):306–311
62. Terry GC, Hammon D, France P, Norwood LA (1991) The stabilizing function of passive shoulder restraints. Am J Sports Med 19(1):26–34
63. Thomas SC, Matsen FA 3rd (1989) An approach to the repair of avulsion of the glenohumeral ligaments in the management of traumatic anterior glenohumeral instability. J Bone Joint Surg Am 71(4):506–513
64. Tirman PF, Steinbach LS, Feller JF, Stauffer AE (1996) Humeral avulsion of the anterior shoulder stabilizing structures after anterior shoulder dislocation: demonstration by MRI and MR arthrography. Skeletal Radiol 25(8):743–748
65. Trivedi S, Pomerantz ML, Gross D, Golijanan P, Provencher MT (2014) Shoulder instability in the setting of bipolar (glenoid and humeral head) bone loss: the glenoid track concept. Clin Orthop Relat Res 472(8):2352–2362. doi:10.1007/s11999-014-3589-7
66. Turkel SJ, Panio MW, Marshall JL, Girgis FG (1981) Stabilizing mechanisms preventing anterior dislocation of the glenohumeral joint. J Bone Joint Surg Am 63(8):1208–1217
67. Ward JP, Bradley JP (2013) Decision making in the in-season athlete with shoulder instability. Clin Sports Med 32(4):685–696. doi:10.1016/j.csm.2013.07.005
68. Wilk KE, Macrina LC (2013) Nonoperative and postoperative rehabilitation for glenohumeral instability. Clin Sports Med 32(4):865–914. doi:10.1016/j.csm.2013.07.017
69. Woertler K, Waldt S (2006) MR imaging in sports-related glenohumeral instability. Eur Radiol 16(12):2622–2636. doi:10.1007/s00330-006-0258-6
70. Wolf EM, Cheng JC, Dickson K (1995) Humeral avulsion of glenohumeral ligaments as a cause of anterior shoulder instability. Arthroscopy J Arthroscopic Relat Surg Off Pub Arthroscopy Assoc North Am Int Arthroscopy Assoc 11(5):600–607
71. Yamamoto N, Itoi E, Abe H, Minagawa H, Seki N, Shimada Y, Okada K (2007) Contact between the glenoid and the humeral head in abduction, external rotation, and horizontal extension: a new concept of glenoid track. J Shoulder Elbow Surg Am Shoulder Elbow Surg [et al] 16(5):649–656. doi:10.1016/j.jse.2006.12.012
72. Yiannakopoulos CK, Mataragas E, Antonogiannakis E (2007) A comparison of the spectrum of intra-articular lesions in acute and chronic anterior shoulder instability. Arthroscopy J Arthroscopic Relat Surg Off Pub Arthroscopy Assoc North Am Int Arthroscopy Assoc 23(9):985–990. doi:10.1016/j.arthro.2007.05.009
73. Zacchilli MA, Owens BD (2010) Epidemiology of shoulder dislocations presenting to emergency departments in the United States. J Bone Joint Surg Am 92(3):542–549. doi:10.2106/JBJS.I.00450
74. Zhang AL, Montgomery SR, Ngo SS, Hame SL, Wang JC, Gamradt SC (2014) Arthroscopic versus open shoulder stabilization: current practice patterns in the United States. Arthroscopy J Arthroscopic Relat Surg Off Pub Arthroscopy Assoc North Am Int Arthroscopy Assoc 30(4):436–443. doi:10.1016/j.arthro.2013.12.013

Shoulder Instability with Bony Lesions

19

Enrico Gervasi and Alessandro Spicuzza

19.1 Introduction

Shoulder instability with bony defects, either anterior or posterior, is commonly caused by recurrent episodes of dislocation [1]. Most of these defects are "small-sized ones" or not big enough which influence the instability after surgical soft tissue repair. In the last 15 years, authors focused on bony lesions as the main risk factor causing postsurgical recurrence of anterior instability [2]. Differences can exist among anterior and posterior instability, whose bone component (Fig. 19.1) has not been deeply studied as the anterior one [3]. Risk factors are associated with the presence, type, size, and location of the bone defects, either in the humeral or in the glenoid side.

Burkhart and De Beer [4] analyzed a cohort of 194 arthroscopic Bankart procedures performed on athletes in order to identify specific factors related to the recurrent instability. Bony lesion on the glenoid side, getting the glenoid to the "inverted pear" shape, and the ones at the humeral side were identified as the main factors affecting the postsurgical recurrences after the arthroscopic Bankart treatment. They assumed that the Hill-Sachs lesion [5] is at risk when its orientation matches the glenoid rim at 90° of abduction in maximal external rotation. This pattern was named "engaging Hill-Sachs lesion." Although this observation represented a starting point, it was not definitively exhaustive.

Athletes' needs are peculiar compared to those of the standard population; several factors, indeed, affect the sport season and the career. As a consequence, the treatment has to be related to these needs. The elite athlete is exposed to specific risks at an early age. The onset of the instability is mainly due to trauma, either in a normal shoulder or in a constitutional ligament laxity.

E. Gervasi (✉) • A. Spicuzza (✉)
Surgical Department, Latisana Hospital,
Via Sabbionera, 45, Latisana 33056, Italy
e-mail: gervasi.enrico@libero.it; A_spicuzza@tiscali.it

According to the literature [6], patients with less than 15 % of glenoid bone loss can be successfully treated by soft tissue procedures. If the deficit exceeds the 25 % of the surface, these are not sufficient. Bony procedures are indicated for these cases. In between these values, several factors influence the decision-making [7].

Bone deficiency can be due to a single dislocating episode as to recurrent ones; it also can be a consequence of attritional mechanism in overhead athletes due to the anterior capsule being stretched allowing the humeral head repetitively riding the anterior glenoid edge. Each of these sports-related mechanisms and specific instability patterns lead to different treatments and surgical approaches. The literature review shows significant differences among authors' treatment choices for the same anatomic lesions: depending on the surgeons' country of origin or their confidence with a procedure over another. Since objective data are the bases for supporting a treatment over another, imaging techniques have a great value when choosing bone procedures, when these can be challenged by soft tissue procedures and when soft tissue procedures are the best choice.

Another aspect, more crucial for athletes than in other population, is the choice, surgical or conservative, at the first dislocation episode.

19.2 Mechanical and Clinical Correlations

The bone loss deficit is due to the energy transmitted by the dislocating humeral head against the glenoid. High-energy dislocations, typical of collision sports (football, rugby), produce glenoid rim fractures in an acute traumatic episode. The dislocation can either occur with the arm in elevation abduction or, specific of collision sports, in mild abduction, forced extension and external rotation while the arm is caught by an antagonist player. This can lead to different orientations of the humeral bony lesion.

Low-energy mechanism of dislocation causes smoothing of the glenoid, getting its deficiency over the time. The

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_19,

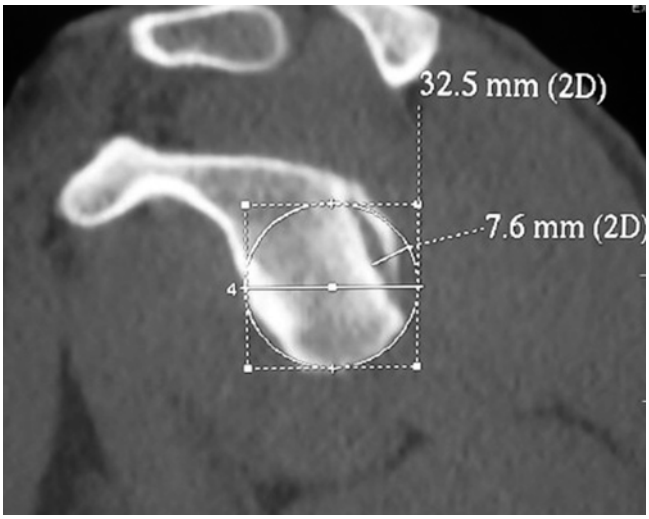


Fig. 19.1 Posterior glenoid deficiency evaluated by TC 2D "Pico technique"

last, affecting lax morphotype patients, is typically the one of overhead sportsmen (javelin throwers, baseball players), sometimes with no evidence of dislocations (the so-called minor instability) [8]; it is associated with stretched anterior capsule, superficial and peripheral Hill-Sachs lesions, partial articular side cuff lesions [9], and posterior capsular stiffness. The onset of the problem in these cases lacks a major trauma, where it is the rule into determining the glenoid fractures. In between these two opposite trauma patterns, the most frequent one is traumatic dislocation due to overstretched arm in external rotation and abduction, causing the avulsion from the glenoid of the labrum and its attached capsule-ligament complex (Bankart lesion) with or without a bony fragment (bony Bankart). At the time of injury, the high-energy dislocations need external maneuvers to accomplish the joint reduction, often requiring anesthesia, whereas the low-energy ones of constitutional lax shoulders do not. The time elapsed from dislocation to the joint reduction results in deepening of the Hill-Sachs lesion, since the cancellous bone of the greater tuberosity is soft. Furthermore, the rotator cuff units, stretched in the dislocating position of the humerus, act vigorously in the healthy shoulder of an athlete, getting the humeral head to be compressed against the glenoid edge: the posterior bone defect deepens and the reduction gets more difficult over the hours.

In order to give the best treatment to the athlete, we have to properly understand the natural history of the instability. The Bankart labral lesion [10] occurs in about 97 % of dislocations. Glenoid bone insufficiency stands at 20 % of patients at first dislocation, rising fast in the recurrences, to reach about 90 % in the post anterior stabilization recurrences; this is due either to the mechanical impact during the dislocation or to the loss of bone substance caused by the surgical implants.

Bony defect, either humeral or glenoid sided, impairs one of the mechanisms of shoulder stabilization. Others are the glenohumeral static stabilizers (capsule and ligaments) and the dynamic ones (the rotator cuff units and the scapulathoracic muscles). The concavity compression depends on several factors: bone morphology, cartilage thickness, and height of the labrum. The thickness of the cartilage at the glenoid periphery gives a huge contribution to the concavity of the glenoid fossa and thus to stability; its loss is missed by CT and x-ray investigations. As these show bone damage, even initially, we can assume that half or more of the glenoid deepness is lost.

The amount of bone insufficiency to get the shoulder unstable is not well defined.

19.3 Diagnosis

19.3.1 History

The suspicion of significant bone lesions should arise from instances of a high-energy trauma, arm abducted over 70° and extended, and when the first traumatic episode is followed by subluxations for negligible traumas or by apprehension and/or instability with the arm externally rotated at less than 60° of abduction (mid-range post-traumatic instability). Similar findings of mid-range instability but without a major trauma are typical either in constitutional multidirectional instability or in unstable shoulders due to massive cuff tears (the latter much more common in older patients).

19.3.2 Physical Examination

Patient examination starts by the inspection of both shoulders, mainly the backside, focusing on muscle atrophy and scapular diskinesia. The specific tests for anterior instability are assessed: anterior load and shift test, anterior drawer test, anterior apprehension test, Jobe relocation test (fulcrum test) [9], throwing test, and bony apprehension test. The last one, similar to the standard apprehension test, is performed with the arm at a 45/60° of abduction; if positive, it means that the bony lesion is the cause of instability. The dynamic anterior jerk test is usually performed under anesthesia.

19.3.3 Imaging

The West Point radiologic view, tangential to the anterior-inferior glenoid margin, is very effective to detect its bony defect. Early after the dislocation, the patient's compliance can be inadequate to maintain the position with arm abducted at 90°; in this case, the Garth (apical oblique) can be an

alternative [11]. The patient is seated with the arm adducted and internally rotated; the x-ray beam is tilted 45° caudally and from the plane of the scapula. Hill-Sachs lesions as subluxations are thus detected. The Stryker notch view can assess the Hill-Sachs lesion and its size and orientation. The patient's hand is placed on top of the head; the x-ray beam is tilted 10° superiorly and centered to the coracoid process, going through the humerus and aiming the cassette placed against the posterior shoulder.

The bony lesions were classified by Bigliani into three types: displaced avulsion of the glenoid rim along with the capsule attachment (type 1), medially displaced fragment (type 2), and glenoid rim erosion (type 3).

As the presence of bone insufficiency is defined and the surgical procedure has to be set, further imaging exams should be carried out in order to properly assess the bony lesions at the glenoid and the humeral sides. The arthro-MRI is useful to study soft tissue lesions and cartilage damages; its role is dominant in the diagnosis of GLAD lesions, Bankart or SLAP lesions, HAGL and RHAGL lesions, and the articular-sided rotator cuff lesions. Its two-dimensional images and the intercut distance, often too big, may underestimate the degree of bone loss, losing its significance in the evaluation of bone insufficiency. The computed tomography is the most affordable exam to evaluate bone defects. The two-dimensional CT accuracy can be impaired by variations in the glenoid version, since the beam should be exactly parallel to the glenoid surface. The goal could be impossible in individuals with glenoid having different versions among the upper and lower glenoid surface, since these come from different ossification centers. Few years ago, in order to study bone defects, several authors developed original 2D CT scan techniques [12], since it was more widely available than the 3D. All these started from Huysmans' observation [13] that the inferior aspect of the glenoid has the shape of a true circle. It gets possible to quantify the glenoid lesions. The percentage of bone loss is determined by the difference between the radius of the best-fit circle of the intact glenoid surface and the one at level of the defect. PICO is the method we are still using today, having been using it since its release; we now complete the CT study by 3D images.

The 3D CT exam reconstruction, indeed, completed by images with subtraction of the humeral head, today is the gold standard in obtaining information about the extent and the pattern of bone injury or loss. Imaging methods quantify both the glenoid circle radius deficit and the deficiency in superior-to-inferior length.

The arthroscopic measurement of the glenoid deficit, as suggested by Burkhart, based on the bare spot can be imprecise. Kralinger et al. verified by CT that the bare spot is about 1.4 mm anterior to the true center of the best-fit circle, getting the deficit to be overestimated. Similarly, Huysmans demonstrated that the bare spot is not identifiable in all the glenoid. The arthroscopic view, on the other hand, gives nicely detailed information about the pattern of the insufficient glenoid and the extent of the defect. The most common glenoid shape is pear-like (88 %); facing the oval-shape pattern (12 %), the surgeon could overestimate the bone loss. Furthermore, he can miss proper information about the osseous fragment, hidden by scar tissue and the attached capsule labrum itself. The anterior-to-posterior glenoid diameter measures on average 24–26 mm. As beyond 25 % of bone loss, the soft tissue procedures fail; it means that 6–8 mm is the borderline to shift to an osseous augmentation procedure.

19.4 Instability with Bone Deficiency: Treatment

There is consensus about the need of surgical treatment for patients with bony instability over the nonoperative one, with limited exception of some true glenoid fractures: these last, indeed, can heal with no consequences to those structures put at the head of the stability. In the past were used just the open techniques as bone augmentation procedures; later on, new arthroscopic or combined techniques have been described for this purpose [14, 15]. These increase the armamentarium for the treatment of "instability with bone deficiency." The actual issue is that albeit the arthroscopic procedures could allow a detailed examination of all articular structures, giving evidence of their eventual pathology, they are evolving and not yet standardized. Conversely, the open techniques allow treatment of just the bone defect and the anterior capsule lesions, not the accessory ones.

The standard open approach for anterior shoulder instability procedures is the deltopectoral approach. It is, ideal to give access to the anterior structures, not optimal to treat the posterior Hill-Sachs lesion. To approach them, the takedown of the long biceps and forced maneuvers of internal rotation and extension are necessary. This event, when complete detachment of the subscapularis is met, can result to muscle changes with some postoperative insufficiency, crucial in case of revision surgery [16]. Other lesions such as the posterior and superior labrum tears are hidden to the anterior operative field.

19.4.1 Hill-Sachs Defects

The defect on the humeral side is one of the two bony predisposing factors (the other being the glenoid deficiency) for recurrence of dislocation after Bankart repair in athletes.

There is no evidence that the size of the defect is significant for stability. Most authors state it is between 20 and 40 %. Sekya reported that a defect as small as 12 % can

somehow be mechanically significant to affect stability [17]. Yamamoto studied the relationship of humeral and glenoid contact introducing the concept of "glenoid track" [18]; then recently, Di Giacomo et al. [19] published the concept of "in track" and "off track" of the Hill-Sachs lesion, defining the latter as the one at risk for dislocation.

Nonanatomic procedures are mainly focused on the external rotation limitation. The subscapularis and the capsule are shortened by doubling them (Putti-Platt) or are detached and transferred laterally (Magnuson-Stack). All these can lead to osteoarthritis and are not indicated for athletes, since they badly affect the external rotation.

The humeral osteotomy, as proposed by Weber, is practically abandoned: performed at the superior margin of the pectoralis major attachment, which puts the humeral defect somehow away, far from the anterior glenoid rim.

Several anatomic techniques have been proposed; their aim is to give back convexity to the humeral head. The use of a fluoroscopic-guided humeral tamp, inserted through a cortical window just lateral to the biceps groove, to elevate the depressed humeral head has been described as other similar techniques are described. Several authors suggest to fill the humeral defect by bone graft through a posterior approach, limited or not, or an anterior extended one. The graft is fixed by headless screws or contoured and inserted press-fit. Graft failure and resorption are possible complications. The described mosaicplasty could be inadequate since the plugs are unstable.

The attempt of reduction under anesthesia of fixed posterior dislocations with reverse Hill-Sachs lesions limited to less than 25 % of the articular surface is possible within 3 weeks from trauma. In the pattern of fixed posterior dislocation, the reverse Hill-Sachs lesions, anterior, are well addressed by the standard deltopectoral approach: in these cases, the humeral filling by graft is often the procedure of choice [20], even in athletes. The large defects, involving half humeral head, can be managed by humeral head replacement, partial (hemi-cap) or not. This should be avoided in young patients. The significant defects can be assessed by several procedures: the MacLaughlin (the subscapularis inserted into the defect) or its modification suggested by Neer (the lesser tuberosity is transferred). Subscapularis weakness can be the effect of its medialization.

The most actual procedure as treatment of the Hill-Sachs lesion in the anterior shoulder instability, named "remplissage," has been proposed by Purchase in 2008: the posterior humeral side defect is filled by capsule and infraspinatus tendon. This refers to the procedure originally proposed by Connolly in 1972.

The remplissage technique consists of a few steps: freshening the defect's surface by peeling the cartilage islands to the subchondral bone, getting a dense, bleeding surface; going to the subacromial space shaving the soft tissues, thus obtaining space for the suture tightening; going back inside to insert anchors; passing the anchors' sutures through the infraspinatus and capsule; and tightening the sutures. We skip the subacromial step, boring, accepting some few deltoid fibers could be involved within the suture limbs.

Several authors support the efficacy of filling the Hill-Sachs lesion by the infraspinatus tendon as effective to gain stability, but few articles describe its related effects on movement restriction or side symptoms. These aspects are crucial when regarding an overhead sportsman, whose performance is strictly dependent on the amplitude of the external rotation of the abducted arm. Just one level 2 prospective study (Nourissat) [21] compares two groups of patients with humeral side defect, without glenoid bone deficiency: one treated by the added remplissage, the other by just the Bankart repair. He found limitation of movement at 1 year after the remplissage procedure. The mean value of this are: ER1 13°, ER2 18.5, forward elevation 14°, and IR 2 vertebra levels. Although not statistically significant, there is a slight difference with the Bankart procedure alone. One third of patients in the remplissage group had residual pain after 2 years. The author suggests the incomplete tendinous healing its cause; conversely we mean that this can be explained by the early impingement of the articular side cuff, medialized, and the posterior glenoid rim (internal impingement). This irritative mechanism is likely to affect overhead elite athletes reducing the indication of the infraspinatus tenodesis versus the competitor Latarjet procedure.

19.4.2 Glenoid Deficiency

Most authors state as "significant" the glenoid bone insufficiency if exceeding the threshold of 20–25 %, hence moving from soft tissue procedures to bone augment ones. Off-track Hill-Sachs lesions as part of bipolar defect stretch the indication for glenoid bone augmentation.

A "significant" bony fragment produced by an acute traumatic dislocation has potentialities of healing in the first week post trauma; these lessen by the time. I mean that, at least in athletes, at any age, this evidence should lead the surgeon to an early repair, reaching a close-to-normal anatomy. These lesions can be repaired even though the width is more than 5–6 mm, equivalent to a glenoid deficit over 20–25 % [22]. In these first dislocation episode cases with acute fresh fracture, often the capsule has minor lesions: the fragment is separated from the glenoid fossa along with the labrum and its attached ligaments that are not too much elongated. The anterior glenoid fracture frequently comes with the pattern of an osseous flap, having some continuity with the joint surface by the inferior labrum that, conversely, is torn at the proximal level. This allows the fragment to spin (Fig. 19.2). The stability is achieved by getting back the bone continuity, thus the glenoid width. Sutures should incorporate and then fix back the bone fragment.

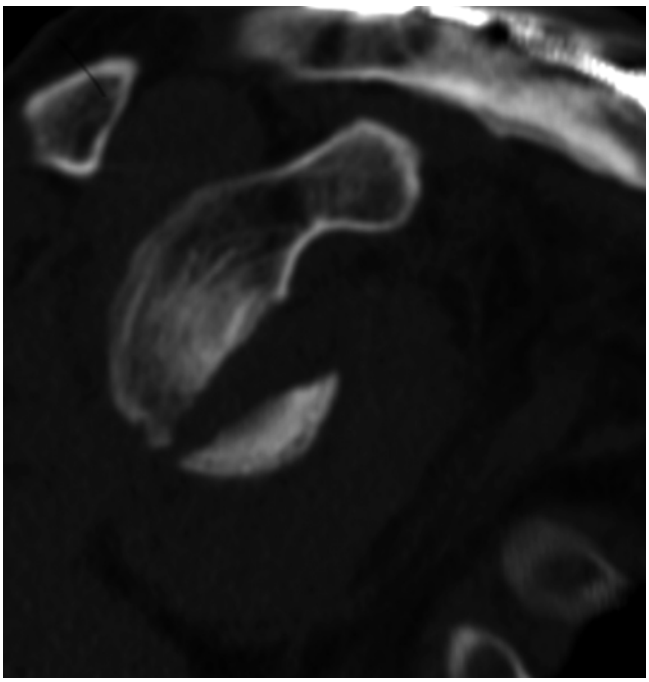


Fig. 19.2 Bone fragment spinning in anterior-inferior glenoid fracture

The unrepaired bony fragment has some resorption, even not complete, over time. Anyway, the bone fragment should be looked for, even if hidden by scar tissue, in order to incorporate it into the repair. It is mandatory to obtain stability [14, 22].

The procedures addressing the glenoid contribution to the stability can be set in the following two groups – (a) coracoid transfer: Bristow or Latarjet, without or with the coracoacromial ligament (Patte), and (b) bone grafts (iliac crest, allografts). We later discuss about salvage procedures.

The coracoid transfer (Latarjet [23], Bristow) allows widening of the glenoid width and, at once, contributes to prevent the humeral head from dislocating by the conjoined tendon dynamic "sling effect." The conjoined tendon, indeed, gets the subscapularis acting as capsular reinforcement of the externally rotated, abducted arm. The coracoid osteotomy is made at its knee: the level of coracoclavicular ligaments (not included). The joint access is made by the subscapularis split, done at the lower third or at the middle of it; this last is preferred in case of very poor capsule in constitutionally lax patients. The scapula neck and the coracoid surface are then freshened. The coracoid is fixed to the scapula neck by two screws. Care should be taken in order to place the graft at the level of the articular surface or just 1–2 mm below: when lateral to the joint line, it leads to arthritic humeral changes; conversely, when too medial, the stability is impaired. The medial edge of the capsule can be repaired at the glenoid margin (optional) to get the graft extra-articular. The Patte variant provides for fixing the stump of the coracoacromial ligament to the capsule, reinforcing it.

The original procedure is made open, by a deltopectoral approach (we suggest slightly medialized). Lafosse published the arthroscopic technique [24]; according to the author, the advantages should be the better cosmesis, reduced postoperative pain, and earlier rehabilitation. Boileau described the combined arthroscopic Bristow procedure (tip of the coracoid transfer) along with the Bankart repair. Latarjet procedure is challenging if compared to traditional stabilization ones. Shah et al. [25] recently reported a 25 % complication rate: neurologic injuries, infections, and coracoid healing failure. The arthroscopic version is even more challenging, with increased risk of complications during the learning curve.

Glenoid bone grafting with tricortical iliac crest (Eden-Hybinette) can be either assessed arthroscopically [26, 27] or open. The iliac crest grafting [28] can be complicated by lateral femoral cutaneous nerve palsy (transient or not), donor site pain, and hematoma. It allows treating large glenoid defects. Alternative autologous donor sites have been described.

Fresh or frozen allografts are alternative sources of bone, with no donor site morbidity.

Both glenoid and the peroneal sides of the distal tibia plateau can provide viable cartilage (fresh allografts) and can match perfectly the articular congruency (Provencher), either in anterior or posterior (Millett) glenoid bone deficit. There is potential risk of transmitted disease; the bone healing/integration is less predictable than for autologous grafts. Actually no peer-reviewed articles are available.

19.5 Postoperative Care and Return to Sports

High-level athletes have specific needs compared to recreational ones. Discussing the treatment and the related rest, they can choose to postpone the treatment to the off-season period. The discussion is carried out with the athlete's coach, trainer, and physiotherapist, sometimes involving the patient's relatives. The use of sport harness or taping during sport activities and physical therapy can be a reasonable choice. After surgery, the arm is supported by a holder at 30° of external rotation for 4 weeks. The scapula-thoracic muscles are trained even in this period of rest.

19.6 Discussion

In case of attritional bone loss, the pathology is the combined bone deficit along with capsule elongation and, often, labrum absence. In this scenario, where the capsule itself is flimsy, no one soft tissue procedure can give guarantees of stability. The surgeon should move to a Latarjet one. If the bone deficit is predominant over the ligaments elongation, other "bone augment" surgeries should be preferred over the coracoid (and

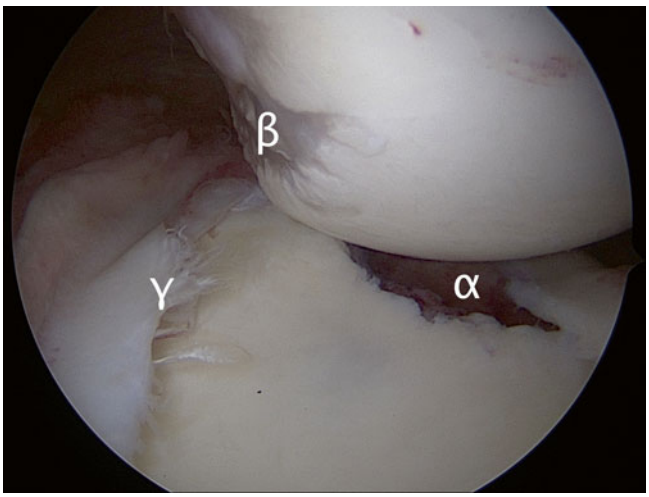


Fig. 19.3 *Complex pattern of instability*: (α) severe anterior glenoid defect; (β) Hill-Sachs lesion; (γ) SLAP lesion extended at the distal posterior labrum

conjoint tendon) transfer [29, 30]. The meaning is to go back to the anatomy if possible, whereas other treatments (Latarjet over all) should be considered when this is not possible.

Instability episodes produce lesions at opposite sides of the "circle": the main ones in the direction of dislocation, due either to push (capsule-ligament, labrum) or impact (glenoid), the others at the rear side due to pull (posterior capsule, labrum). The treatment of lesions of just one side gets to miss the ones of the opposite side. Since open surgery does not approach properly so far surgical fields, anterior and posterior, we figure out that arthroscopy would be the ideal technique for managing the complex patterns of instability (Fig. 19.3). It needs evolving to become more affordable and less risky in the bone deficit management.

Each instability episode adds damages to the tissues, causing them structural changes; thus, after several dislocations, it can be impossible to restore both gross and structural anatomy. Although there are athletes that after the first dislocation will not redislocate, we are with those surgeons that would operate all of them at the first episode: the detached, not yet elongated or stretched ligaments, and the labrum could be fixed in their original position, thus getting back to the normal anatomy. The dislocation, the main problem of instability, is not the only likely symptomatic one. Other problems, aside from pain or impaired proprioception, are not well known till today but can be consistent, affecting the high-demand athlete performance.

"Significant glenoid deficiency" refers to bone and cartilage defect. It comes within different scenarios. The following descriptions address the main anatomic, thus functional, categories:

(a) *Glenoid fracture with minor ligament damage*. We recommend the arthroscopic treatment, freshening the scapula neck along with the bone fragment and including it into the repair.

(b) *Humeral engaging, off track Hill-Sachs lesion*. The choice is the arthroscopic tenodesis of the infraspinatus tendon into the defect.

(c) "*Significant*" *bone defect with minor ligament damage*. It includes both the glenoid deficiency alone and the combination of it with the humeral one. Bone and cartilage are both lost at the glenoid side. The glenoid track is impaired. The glenoid side treatment is crucial, often sufficient to obtain shoulder stability. The choice is glenoid grafting [31]; the ideal graft should recover bone and viable cartilage. Fresh allograft, glenoid or the peroneal half of the distal tibia [32], would fit perfectly, but to date, there are concerns about potential disease transmission, availability, and cost. Reported results support the evidence of bone integration, but further investigations are needed. Frozen grafts cannot guarantee viable cartilage. The bone resorption can cause the fixation screws exposure then the risk of humeral head subluxation and damage. Alternative to them is the tricortical iliac crest autograft. The potential complications of the donor site are pain, hematoma, and femoral cutaneous lateralis nerve palsy (transient or permanent). Other aspects lessen the surgeon's compliance around the iliac crest graft, as the need of two surgical fields and related patient positioning. Another suggested autologous graft is the lateral clavicle.

(d) *Significant bone defect associated to severe ligament damage*. The procedure of choice is the coracoid transfer with its attached conjoined tendon. It gives dynamic stability by forcing the distal subscapularis unit against the humeral head when the arm is externally rotated and abducted. According to the capsule fixation, at the glenoid rim or outside, the coracoid can be inside the joint or external. This is a nonanatomic procedure [33]. Its great value is the low rate of dislocation recurrences. Collision and contact in young male athlete population has the maximum benefit from it. The open, anterior approach to the shoulder misses the side lesions (posterior and superior labrum and biceps, articular-sided cuff lesions) (Fig. 19.4). The weak aspects of the arthroscopic technique, challenging even for experienced surgeons, are two. The minor one is the difficulty to retract the subscapularis tendon, the superior and inferior half, in order to have adequate access to the joint. The major problem is the difficulty to fix the screws, and therefore the graft, parallel to the glenoid surface. This is due to the scapula joint line and neck orientation, as they are rotated anteriorly around the chest wall; it prevents the optimal screwing direction from an anterior approach. If these problems are overcome, the arthroscopic technique will be of choice, allowing the treatment of the multiple lesions of the unstable shoulder. The assumption

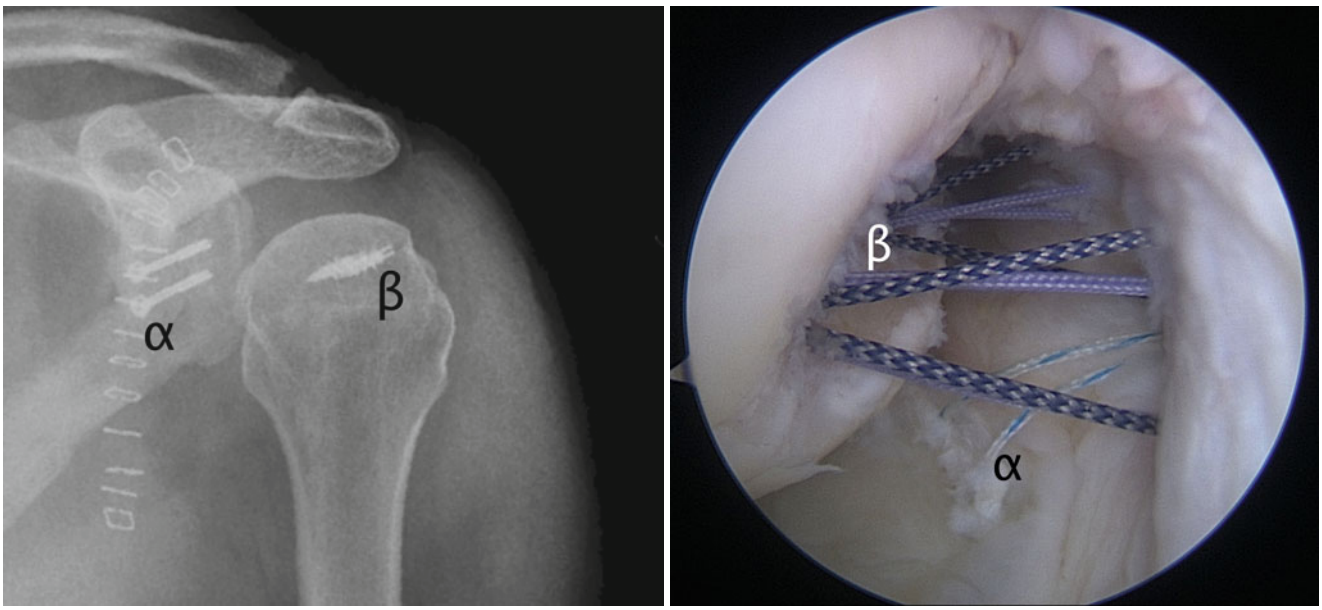


Fig. 19.4 (**a**) Arthroscopic management of posterior lesions combined to Latarjet procedure: (α) labrum fixation (white/light blue sutures) and (β) Hill-Sachs remplissage. (**b**) Radiographic postoperative control*:* (α) coracoid graft and (β) remplissage screws

that the arthroscopic technique gives better cosmesis, reduced postoperative pain, and earlier rehabilitation is not supported by evidences.

19.6.1 Indication to the Latarjet as Procedure of Choice According to the Severity Index Score Should be Regarded with Extreme Caution

The thesis supporting this score validity stands on the incorrect assumption that the arthroscopic Bankart technique, the Latarjet one, is compared to the arthroscopic gold standard. The Boileau study was carried out between 1999 and 2002. The anchors used were Panalok, loaded each one by a single PDS 2 suture: it means that resorbable anchors were loaded by resorbable sutures! High-resistance sutures were not available in those years. No one surgeon today would support the thesis that this specific arthroscopic technique, challenging the Latarjet technique, is the actual arthroscopic gold standard. This bias undermines the reliability of his study.

(e) *Revision cases*. The old techniques of external rotation restriction (Magnuson-Stack, Putti-Platt, Delitala) maintain a role, very seldom, in selected revision cases of Latarjet procedure, but they are affected by an unacceptable rate of arthritic changes. The iliac crest grafting best treats most of the bony instability failures [34]. Lenny Johnson suggested the upper subscapularis tenodesis [35] as beneficial for stability; recently Maiotti and Massoni [36] supports this technique even for primary surgeries. We do not recommend it for the likely arthritic evolution.

The aspect of instability with bony lesions is a manifold problem. Patient expectations often exceed the realistic possibilities of surgery and rehabilitation. A variable amount of mobility is lost. This can affect or not the athlete performance, according to the sport. The specific sport activities are restricted for months, up to 1 year, after surgery.

The best surgery is the one addressing the multiple lesions in that specific individual. The shoulder surgeon should be trained to properly manage various surgical techniques in order to adapt himself to the patient needs, not vice versa.

References

1. Tauber M, Resch H, Forstner R, Raffl M, Schauer J (2004) Reasons for failure after surgical repair of anterior shoulder instability. J Shoulder Elbow Surg 13(3):279–285
2. Milano G, Grasso A, Russo A, Magarelli N, Santagada DA, Deriu L, Baudi P, Bonomo L, Fabbriciani C (2011) Analysis of risk factors for glenoid bone defect in anterior shoulder instability. Am J Sports Med 39(9):1870–1876
3. Paul J, Buchmann S, Beitzel K, Solovyova O, Imhoff AB (2011) Posterior shoulder dislocation: systematic review and treatment algorithm. Arthroscopy 27(11):1562–1572
4. Burkhart SS, De Beer JF (2000) Traumatic glenohumeral bone defects and their relationship to failure of arthroscopic Bankart repairs: significance of the inverted-pear glenoid and the humeral engaging Hill-Sachs lesion. Arthroscopy 16(7):677–694
5. Hill HA, Sachs MD (1940) The groove defect of the humeral head. A frequently unrecognized complication of dislocations of the shoulder joint. Radiology 35:690–700
6. Tokish J, Shaha J et al (2014) Critical bone loss in shoulder instability reconsidered: subcritical bone loss correlates to worse outcome in arthroscopic Bankart repair. Arthroscopy 30 (6 Supplement):e4

7. Pagnani MJ (2008) Open capsular repair without bone block for recurrent anterior shoulder instability in patients with and without bony defects of the glenoid and/or humeral head. Am J Sports Med 36:1805–1812
8. Castagna A, Nordenson U, Garofalo R, Karlsson J (2007) Minor shoulder instability. Arthroscopy 23(2):211–215
9. Jobe FW, Kvitne RS, Giangarra CE (1989) Shoulder pain in the overhand or throwing athlete. The relationship of anterior instability and rotator cuff impingement. Orthop Rev 18:963–975
10. Bankart ASB (1923) Recurrent or habitual dislocation of the shoulder joint. Br Med J 2:1132
11. Garth WP Jr, Slappey CE, Ochs CW (1984) Roentgenographic demonstration of instability of the shoulder: the apical oblique projection. A technical note. J Bone Joint Surg Am 66:1450–1453
12. Baudi P, Righi P, Bolognesi D, Rivetta S, Rossi Urtoler E, Guicciardi N, Carrara M (2005) How to identify and calculate glenoid bone deficit. Chir Organi Mov 90:145–152
13. Huysmans PE, Haen PS, Kidd M, Dhert WJ, Willems JW (2006) The shape of the inferior part of the glenoid: a cadaveric study. J Shoulder Elbow Surg 15:759–763
14. Sugaya H, Kon Y, Tsuchiya A (2005) Arthroscopic repair of glenoid fractures using suture anchors. Arthroscopy 21:635
15. Porcellini G, Campi F, Paladini P (2002) Arthroscopic approach to acute bony Bankart lesion. Arthroscopy 18:764–769
16. Bushnell BD, Creighton RA, Herring MM (2008) Bony instability of the shoulder. Arthroscopy 24(9):1061–1073
17. Skendzel JG, Sekiya JK (2012) Diagnosis and management of humeral head bone loss in shoulder instability. Am J Sports Med 40:2633
18. Yamamoto N, Itoi E, Abe H, Minagawa H, Seki N, Shimada Y, Okada K (2007) Contact between the glenoid and the humeral head in abduction, external rotation, and horizontal extension: a new concept of glenoid track. J Shoulder Elbow Surg 16(5):649–656
19. Di Giacomo G, Itoi E, Burkhart SS (2014) Evolving concept of bipolar bone loss and the Hill-Sachs lesion: from "engaging/non-engaging" lesion to "on-track/off-track" lesion. Arthroscopy 30(1):90–98
20. Gerber C, Lambert SM (1996) Allograft reconstruction of segmental defects of the humeral head for the treatment of chronic locked posterior dislocation of the shoulder. J Bone Joint Surg Am 78:376–382
21. Nourissat G, Kilinc AS, Werther JR, Doursounian L (2011) A prospective, comparative, radiological, and clinical study of the influence of the "remplissage" procedure on shoulder range of motion after stabilization by arthroscopic Bankart repair. Am J Sports Med 39(10):2147–2152
22. Sugaya H, Moriishi J, Kanisawa I, Tsuchiya A (2005) Arthroscopic osseous Bankart repair for chronic recurrent traumatic anterior glenohumeral instability. J Bone Joint Surg Am 87:1752–1760
23. Helfet AJ (1958) Coracoid transplantation for recurring dislocation of the shoulder. J Bone Joint Surg Br 40:198–202
24. Lafosse L, Lejeune E, Bouchard A, Kakuda C, Gobezie R, Kochhar T (2007) The arthroscopic Latarjet procedure for the treatment of anterior shoulder instability. Arthroscopy 23(11):1242.e1–1242.e5
25. Shah AA, Butler RB, Romanowski J, Goel D, Karadagli D, Warner JJ (2012) Short-term complications of the Latarjet procedure. J Bone Joint Surg Am 94(6):495–501
26. Taverna E, Golanò P, Pascale V, Battistella F (2008) An arthroscopic bone graft procedure for treating anterior-inferior glenohumeral instability. Knee Surg Sports Traumatol Arthrosc 16(9):872–875
27. Scheibel M, Kraus N, Diederichs G, Haas NP (2008) Arthroscopic reconstruction of chronic anteroinferior glenoid defect using an autologous tricortical iliac crest bone grafting technique. Arch Orthop Trauma Surg 128(11):1295–1300
28. Warner JJ, Gill TJ, O'hollerhan JD, Pathare N, Millett PJ (2006) Anatomical glenoid reconstruction for recurrent anterior glenohumeral instability with glenoid deficiency using an autogenous tricortical iliac crest bone graft. Am J Sports Med 34:205–212
29. Griesser MJ, Harris JD, McCoy BW, Hussain WM, Jones MH, Bishop JY, Miniaci A (2013) Complications and re-operations after Bristow-Latarjet shoulder stabilization: a systematic review. J Shoulder Elbow Surg 22(2):286–292
30. Montgomery WH Jr, Wahl M, Hettrich C, Itoi E, Lippitt SB, Matsen FA 3rd (2005) Anteroinferior bone-grafting can restore stability in osseous glenoid defects. J Bone Joint Surg Am 87(9):1972–1977
31. Weng PW, Shen HC, Lee HH, Wu SS, Lee CH (2009) Open reconstruction of large bony glenoid erosion with allogeneic bone graft for recurrent anterior shoulder dislocation. Am J Sports Med 37:1792–1797
32. Provencher MT, Ghodadra N, LeClere L, Solomon DJ, Romeo AA (2009) Anatomic osteochondral glenoid reconstruction for recurrent glenohumeral instability with glenoid deficiency using a distal tibia allograft. Arthroscopy 25:446–452
33. Hovelius L, Sandström B, Saebö M (2006) One hundred eighteen Bristow-Latarjet repairs for recurrent anterior dislocation of the shoulder prospectively followed for fifteen years: study: the evolution of dislocation arthropathy. J Shoulder Elbow Surg 15(3): 279–289
34. Lunn JV, Castellano-Rosa J, Walch G (2008) Recurrent anterior dislocation after the Latarjet procedure: outcome after revision using a modified Eden-Hybinette operation. J Shoulder Elbow Surg 17:744–750
35. Johnson LL (1986) Arthroscopic surgery: principle and practice. Mosby, St. Louis, pp 1420–1424. Chapter 15
36. Maiotti M, Massoni C (2013) Arthroscopic augmentation with subscapularis tendon in anterior shoulder instability with capsulolabral deficiency. Arthrosc Tech 2(3):e303–e310

Shoulder Microtraumatic and Atraumatic Instability

20

Gianezio Paribelli, S. Boschi, A.M. Cassarino, and F. Leonardi

20.1 Introduction

Instability is a pathological condition characterised by discomfort or pain associated with excessive translation of the humeral head over the glenoid cavity during active mobilisation of the shoulder.

Classified on the basis of pathogenic mechanisms and pathoanatomical, clinical and therapeutic features, instability is distinguished into the three following groups:

- *TUBS* (traumatic, unilateral, Bankart, surgery)
- *AMBRI* (atraumatic, multidirectional, bilateral, rehabilitation, inferior capsular shift) and *AMSI* (atraumatic, minor, shoulder, instability)
- *AIOS* (acquired, instability, overstressed, surgery)

AMBRI, AIOS and AMSI are defined as *minor instabilities* and are characterised by a smaller translation, often not perceived by the patient.

In most cases the diagnosis is complex because the symptoms are so severe that they interfere with the clinical tests, and imaging may be negative.

G. Paribelli (✉) • A.M. Cassarino
Struttura accreditata S.S.N. Maria Cecilia Hospital,
Via Corriera, 1, Cotignola (RA) 48010, Italy
e-mail: geparibelli@libero.it

S. Boschi
Ospedale Privato Accreditato Prof. Fogliani
Modena, Modena, Italy

F. Leonardi
Struttura accreditata S.S.N. Maria Cecilia Hospital,
Via Corriera, 1, Cotignola (RA) 48010, Italy

Ospedale Privato Accreditato Prof. Fogliani
Modena, Modena, Italy

20.2 Mechanism of Injury: Overhead Movement

Overhead movement (throwing in baseball, spiking in volleyball, arm stroke in swimming) is a forced action in abduction and external rotation, with features that vary depending on the sport.

It is the result of a fine organisation of numerous muscular districts that coordinate with one another in kinetic chains.

An overhead movement that is characteristic of a certain sports role and performed repetitively by the player can progressively damage, through repeated microtraumas and in the presence of other concomitant conditions, the structures involved in glenohumeral stabilisation, thus becoming the main cause of microinstability in the athlete.

The pathogenesis of microinstability is related to the force and frequency of the movement.

Professional swimmers cover an average distance of 10 km per day, equal to 4,000 strokes/day [1].

Spiking is the most explosive movement made by a volleyball player. The ball can reach a speed of 28 m/s. Professional players train for 16–20 h per week, and spikers can repeat the movement even more than 40,000 times in a season [2].

Angular velocity during baseball pitching is 7,000° per second, probably the fastest recorded human movement [3].

A baseball pitcher's, or a quarterback's, throw expresses forces equal to 1/1.5 times the player's body weight [4].

20.3 Pathogenesis

The various lesions may occur isolated or in combination, according to an aetiopathogenic basis that is still being investigated:

1. Most probably, the first abnormality that triggers the cascade of events is a tendinopathy due to fatigue of the external rotator cuff muscles, caused by repetitive eccentric microtraumatic contractions during the deceleration phase of the overhead movement [5].

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_20,

2. This fatigue places strain on the underlying capsule, causing a posterior [4] or posteroinferior [6] capsular contracture and consequent decrease in internal rotation.

Consequences

The two aspects discussed above are biomechanically very similar, but they occur on different tissue substrates: muscle and capsule.

24 h after an overhead sporting event, there is a reversible decrease in internal rotation [4] probably of a muscular nature.

12 weeks of an overhead sport [5], such as tennis and baseball, are sufficient to cause a persisting deficit in internal rotation, probably of a capsular nature.

3. Posterior stiffness and the resultant internal rotation deficit gradually lead to a posterosuperior translation of the humeral head, known as hyperangulation [7].

Consequences

In this new position, the humeral head is capable of greater external rotation.

This could be a compensatory mechanism of the shoulder to counterbalance the internal rotation deficit and preserve total range of motion. The result is a counterclockwise shift of range of motion for the right shoulder and clockwise for the left shoulder.

Studies have reported an increase in external rotation in professional tennis players 4–6 months after the beginning of the season [8] and a 5° increase in external rotation in baseball players at the end of the season [4].

4. Hyperangulation on the one hand entails a greater loading phase of the throw with evident increase in force and, on the other, a delicate balance:
   (a) The stabilisation complex is, in fact, repeatedly taxed by numerous attempts to withstand also greater tensile stresses in external rotation, predisposing the capsule to anterior laxity (Fig. 20.1) [9].
   (b) The movement, which is violent, repeated over time and now capable of increased external rotation in abduction, causes a greater torsion of the biceps anchor with more strain being placed on the anchor itself and on the posterior glenoid labrum [10].

This mechanism, known as "peel back", may lead to a potential detachment of the posterior biceps anchor (posterior SLAP II lesion) or the extension of a previous injury.

This SLAP lesion may also occur during the deceleration phase of the throw [11].

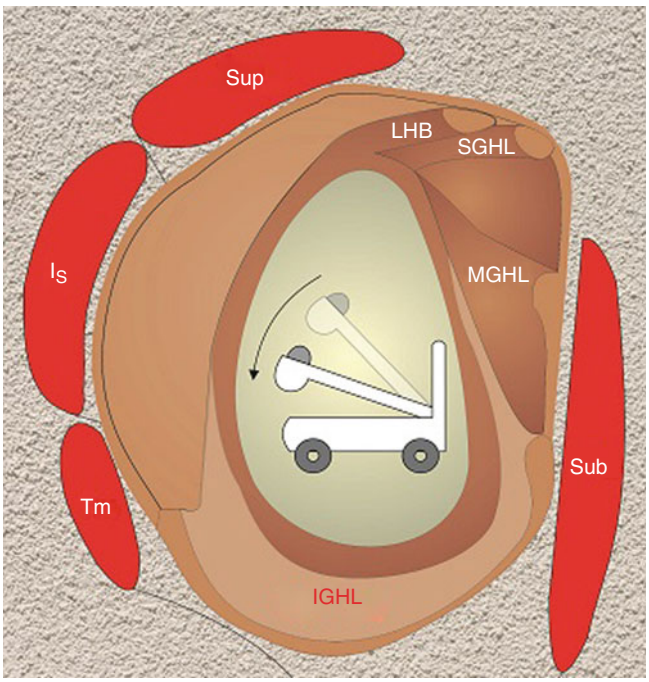


Fig. 20.1 Hyperangulation entails a greater loading phase of the throw with evident increase in force (like a catapult) but also a delicate balance: the stabilisation complex is, in fact, repeatedly taxed by numerous attempts to withstand also greater tensile stresses in external rotation, predisposing the capsule to anterior laxity. *Tm* teres minor, *Is* infraspinatus, *Sup* supraspinatus, *Sub* subscapularis, *LHB* long head biceps, *SGHL* superior gleno humeral ligament, *MGHL* medium gleno humeral ligament, *IGHL* inferior gleno humeral ligament

Consequences

Cadaver studies have reported lengthening of the anterior band of the inferior glenohumeral ligament [12] and a consequent increase in anteroinferior translation capability [13] as well as (further) external rotation capability, with a decline in performance and the onset of pain.

A SLAP lesion also leads to a posterosuperior instability which, combined with repetitive movement in abduction-external rotation, results in a progressive posterior insertional stress of the supraspinatus and its progressive surrendering.

The greater external rotation and the more posterior position of the humeral head increase the likelihood of the overhead motion causing pinching of the cuff between the posterior glenoid rim and the greater tuberosity, which is located posteriorly during external rotation plus abduction, resulting in internal impingement.

Even though there is no consensus in the literature in this respect, the internal impingement could cause an intra-articular lesion of the posterior cuff and corresponding rim, with worsening of the SLAP lesion.

A cadaver study has demonstrated the importance of the rotator interval in anterior, as well as posterior

and inferior, instability, suggesting that the mechanism of injury is ascribable to an anterosuperior sliding of the head, resulting from an insufficient interval [14].

In overhead athletes, the pain related to instability is due to contact between the articular surface of the cuff and the anterior-superior labrum.

The combination of lesions affecting these two structures is known as a SLAC (superior labrum anterior cuff) lesion (Fig. 20.2).

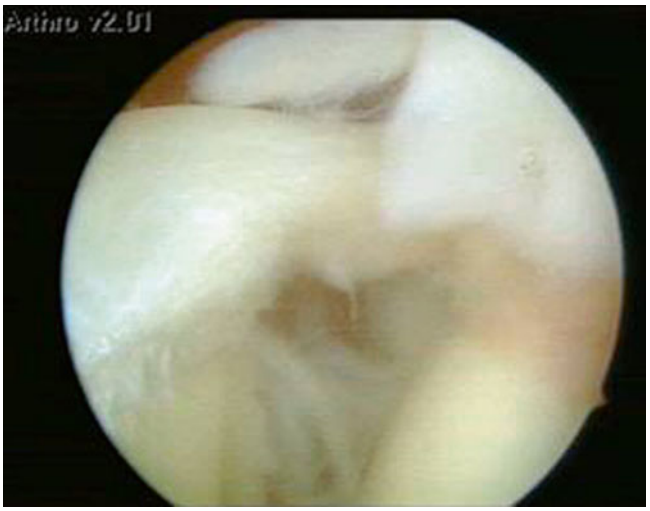


Fig. 20.2 Arthroscopic image of a typical associated lesion in the overhead athlete: SLAP lesion and injury to the articular surface of the rotator cuff

20.4 Role of the Scapulothoracic Joint

A positive correlation has been demonstrated between positional abnormalities of the scapula and overhead sports [4, 15].

Several studies, analysing the influence of pain and fatigue on the shoulder girdle muscles [16, 17], found a decrease in posterior tilt and an increase in clavicular retraction on anterior elevation of the arm.

Conversely, conflicting results have been reached with regard to lateral scapular rotation and protraction.

Several authors believe that the former increases from pre- to post-fatigue [16].

Protraction, on the other hand, is thought to increase post-fatigue (wind up) [15, 18].

Considering muscular activity, studies have demonstrated a delayed and decreased activation of the middle and lower trapezius and serratus anterior, an increased activity of the upper trapezius [19] and increased activity of the infraspinatus during the descending, hence eccentric, phase as a probable compensatory mechanism.

The upper and lower trapezii constitute a major force couple: A decrease in the activity of the lower trapezius coupled with increased activity of the upper trapezius can cause cranial translation of the horizontal scapular axis, with an increase in the likelihood of impingement [20].

This may contribute to the decrease in posterior tilt and external rotation, reducing the subacromial space and further increasing the risk of impingement.

It is likely that a change in the mutual position of the scapula and humerus [21], by causing a misalignment of tendon structures, affects their length/force ratio.

Further studies are required to demonstrate a causal link between dyskinesia and microinstability, possibly through an assessment of athletes pre- and postseason [15].

It is not clear whether the compensatory scapular positioning and hyperangulation can increase or reduce the risk of subacromial impingement.

20.5 The Proprioceptive System

The proprioceptive system is also involved in this cascade of events, in both the short and long term, decreasing reversibly during a single sporting event and returning to pre-fatigue values after 10 min [22].

20.6 Pathological Anatomy

The pathological anatomy of microtraumatic instabilities may affect the entire glenohumeral labral-ligamentous-tendinous complex.

The Head

The humeral head often assumes a more anterior position with respect to the horizontal axis of the body [23].

An intense overhead activity between the ages of 13 and 16 years may block the physiological process of humeral head anteversion, by enhancing its retroversion [24].

The Glenoid

Changes in glenohumeral kinematics reflect on the density of glenoid subchondral bone. In overhead athletes, this density is apparently increased in the anteroinferior, posterosuperior and inferior segments [25].

SLAP

The upper portion of the labrum may show a variety of lesions, often specific to type of sport played. Recognition of these lesions could have important therapeutic and prognostic implications [26].

Others

It is easy to hypothesise also other alterations affecting the spine, the appendicular and trunk muscles and the soft tissues.

20.7 Clinical Presentation

The symptoms reported by overhead athletes are related to the cascade of events described by Morgan [10].

Initially, the athletes may have subjective and objective perception of reduced throwing speed (without pain) and resulting decrease in performance: dead arm syndrome.

They may then develop vague pain and discomfort not otherwise specified.

This may be followed by pain due to internal impingement.

In the final phase of the cascade, mechanical symptoms may develop as a result of the development of posterior or posterosuperior labral lesions and rotator cuff dysfunction [27].

Anterior coracoid pain [3] and bursitis secondary to kinematic alterations of the scapulothoracic joint have also been reported.

Involvement of the long head of the biceps, although rare, may occur during preparation for the sports season, when the limb is not yet ready for major stresses [4].

Finally, apprehension and a sensation of subluxation and/or secondary deficits affecting the arm and forearm muscles may develop as further symptoms of instability.

20.8 Diagnosis

The approach to shoulder disorders is very complex, with only diagnostic imaging having provided the solution to many problems.

After history taking, inspection may reveal postural and/or spinal abnormalities.

A common finding is hypertrophy of the dominant shoulder or dropped scapula [18] caused by lengthening of the static and dynamic stabilisers that are intensely used in athletes.

Palpation, whenever possible, should include structures such as the posterior glenohumeral joint line, the subacromial space, the greater tuberosity, the acromioclavicular inter-joint space and the groove.

Laxity is often a general pre-existing condition in overhead athletes which predisposes to or exacerbates the symptoms and should always be assessed.

The usual tests are considered valid (thumb, fifth finger, elbow) and laxity may also be quantified according to the Brighton scale.

Shoulder ROM is then assessed, for which the mean values for the dominant arm of an asymptomatic athlete are as follows:

External rotation at 90° abduction 129–137° (7–9° > of non-dominant arm)

Internal rotation at 90° of abduction 54–61° (7–9° < of non-dominant arm)

TROM (total ROM) 183–198° (equal to nondominant arm)

The relationship between GIRD (glenohumeral internal rotation deficit) and TROM is fairly controversial.

Two types of GIRD have been suggested [28]:

1. Diminished internal rotation, unchanged or slightly increased external rotation and symmetrical TROM
2. Diminished TROM (pathological)

The literature suggests treating only pathological GIRD and, in particular, those cases characterised by TROM of dominant arm less than 5° when compared to the nondominant arm [29], since these are more likely to be affected by lesions or progression of the cascade: Treatment of non-pathological forms could further reduce shoulder stability.

It is important to remember that, even though symmetrical TROM suggests a non-pathological GIRD, it should nonetheless be considered a non-physiological condition indicating a neglected shoulder, and for this reason, it deserves continuous monitoring.

Assessment of the scapulothoracic joint follows, and scapular behaviour is tested statically and dynamically (we recommend having the patient repeat the movement several times and assessing both the concentric and eccentric phase) in all its phases.

Evaluation of strength is very difficult because the tests are often influenced by pain.

The bursae are more easily assessed: The patient lies prone on the examining table and is invited to place the back of the hand behind the back. This position will cause raising of the scapular medial margin facilitating the direct approach to the bursae for palpation and possible infiltration.

Stability testing: Many authors believe that the stability of a shoulder should be assessed in three directions, so that at least three tests are required (sulcus test, drawer test, Lachman test, fulcrum test).

The Lachman and fulcrum tests are the most important for evaluating stability in that they simulate the conditions of the throw [4].

The apprehension test is not really useful in overhead athletes, in that it assesses evident gross instabilities, while the subluxation/relocation test is better for assessing more subtle forms [30].

When performing tests for anterior and posterior instability, it is crucial to take into account the inclination of the glenoid plane, so the tests should be performed at 30° of flexion.

In addition, the test for anterior stability should be performed at different degrees of abduction, so as to evaluate

the different stabilisation structures: at ca. 30° for the superior glenohumeral ligament, at ca. 60° for the middle ligament and at ca. 90° for the inferior ligament.

As regards posterior instability, the only structure that needs to be assessed is the posterior band of the inferior glenohumeral ligament, so the test should be performed at 90° of abduction.

Tests for the rotator cuff tendons and internal impingement are then performed (e.g. the Kibler retraction test).

A clinical diagnosis of SLAP lesion is not easy.

The literature agrees that conceptually valid tests should reproduce the supposed mechanism of injury, so in the case of overhead athletes, the peel back (biceps load and modified biceps load, pain provocation and resisted supination) [27].

20.9 Imaging Investigations

Plain radiography can reveal typical findings, such as radiographic alterations of the greater tuberosity (cysts and/or sclerosis), osteochondral defects located close to the point of insertion of the supraspinatus (posterior humeral geode) or Bennett lesions (extra-articular ossifications of the posterior capsule probably due to protracted stresses on the capsule itself) [27].

The diagnosis is completed with contrast-enhanced magnetic resonance (MR) imaging, which enables visualisation of cuff tears and SLAP lesions.

The differential diagnosis includes pathological conditions of nervous and vascular aetiology.

20.10 Therapy and Return to Competitive Activity

We recommend to monitor the athlete throughout the competitive season:

- *Prevention*: There is agreement in the literature that athletes who perform stretching exercises during the season do not develop ROM changes throughout a 4-month season [22]. However, some believe that stretching alone is not sufficient and should be associated with attention to preparation techniques [24].
- *Early diagnosis*: This enables early detection of the signs of microinstability, both motor and proprioceptive, so that progression of the cascade can be prevented [5].

  ROM changes: Only retracted capsulomuscular structures should be treated. Treatment of non-retracted structures might worsen the instability [31]: sleeper and cross-body stretches for the capsule and posterior muscles, corner stretches, sitting stretches and manual stretching for the lesser pectoral [13], with assessment at 4 weeks [27].

  Onset of pain: it is recommended to interrupt all athletic activity and provide pharmacological pain relief, cryotherapy and physical therapy (ionophoresis, laser therapy, ultrasound, TECAR therapy, magnetotherapy).

  Once the pain has subsided, kinematics can be reorganised by physical therapy.

  Dyskinesias: The treatment of dyskinesias is perhaps the greatest challenge for the physician, as it is not easy to relate the dyskinesia to the appearance of pain.

  Continuous monitoring of the patient and the finding of a recent-onset dyskinesia assist in treatment decisions.

Treatment is initiated after having stabilised the kinetic chains, core, posture, head position [32] as well as all the "non-shoulder" joints involved in the overhead motion [33].

Once the various districts have been appropriately strengthened, exercises simulating the overhead movement should be planned [32].

Impingements: we believe that physical therapy targeted at correcting kinematics can also benefit any possible impingement (subacromial and/or posterior), as these are purely functional [17].

Cuff: the choice of treatment for rotator cuff tendons is related to the degree of injury.

Long head of the biceps: this is treated by suspension of athletic activity and stabilisation of the scapulothoracic joint.

Bursitis: good results have been achieved with infiltrations [3].

Instability: the current literature is not very exhaustive on the subject. Shoulder stabilisation requires the synchronous functioning of the static and dynamic stabilisers, and when this balance is missing, instability results. Treating the instability means increasing strength and resistance of the static and dynamic stabilisers and neuromuscular control [27].

Should surgical treatment fail, an arthroscopic capsular shift followed by a rehabilitation protocol is necessary.

SLAP Lesion: in the overhead athlete, this is a severe event carrying a negative prognosis.

Treatment choices are related to the supposed lesion type:

SLAP I lesions are best treated conservatively.

SLAP II lesions require arthroscopic stabilisation of the biceps anchor and tendon, whereas stabilisation of the anchor in SLAP III lesions depends on its involvement.

With regard to postoperative rehabilitation, the authors believe it is fundamental to be aware of the mechanism of injury: Where the SLAP lesion was caused by compression, all exercises involving compression should be avoided; for lesions caused by traction, eccentric contractures should be avoided; in peel-back lesions, all exercises in abduction and external rotation should be avoided [4].

Finally, correct posture, kinematics, proprioception and eventually the athletic movement should all be restored.

References

1. Tate A, Turner GN, Knab SE, Jorgensen C, Michener LA (2012) Risk factors associated with shoulder pain and disability across the lifespan of competitive swimmers. J Athl Train 47(2):149–158
2. Reeser JC, Fleisig GS, Bolt B, Ruan M (2010) Upper limb biomechanics during the volleyball serve and spike. Sports Health 2(5):368–374
3. Seroyer ST, Nho SJ, Bach BR Jr, Bush-Joseph CA, Nicholson GP, Romeo AA (2009) Shoulder pain in the overhead throwing athlete. Sports Health 1(2):108–120
4. Reinold MM, Gill TJ (2010) Current concepts in the evaluation and treatment of the shoulder in overhead-throwing athletes, part 1. Sports Health 2(1):39–50
5. Thomas SJ, Swanik KA, Swanik C, Huxel KC (2009) Glenohumeral rotation and scapular position adaptations after a single high school female sports season. J Athl Train 44(3):230–237
6. Marcondes FB, de Jesus JF, Bryk FF, de Vasconcelos RA, Fukuda TY (2013) Posterior shoulder tightness and rotator cuff strength assessments in painful shoulders of amateur tennis players. Braz J Phys Ther 17(2):185–194
7. Aldridge R, Guffey J, Whitehead MT, Head P (2012) The effects of a daily stretching protocol on passive glenohumeral internal rotation in overhead throwing collegiate athletes. Int J Sports Phys Ther 7(4):365–371
8. Dwelly PM, Tripp BL, Tripp PA, Eberman LE, Gorin S (2009) Glenohumeral rotational range of motion in collegiate overhead-throwing athletes during an athletic season. J Athl Train 44(6):611–616
9. Meister K, Day T, Horodyski M, Kaminski TW, Wasik MP, Tillman S (2005) Rotational motion changes in the glenohumeral joint of the adolescent/little league baseball player. Am J Sports Med 33(5):693–698
10. Burkhart SS, Morgan CD (1998) The peel-back mechanism: its role in producing and extending posterior type II SLAP lesions and its effect on SLAP repair rehabilitation. Arthroscopy 14(6):637–640
11. Kim TK, Queale WS, Cosgarea AJ, McFarland EG (2003) Clinical features of the different types of SLAP lesions, an analysis of one hundred and thirty-nine cases. J Bone Joint Surg Am 85(1):66–71
12. Mihata T, Lee Y, McGarry MH, Abe M, Lee TQ (2004) Excessive humeral external rotation results in increased shoulder laxity. Am J Sports Med 32(5):1278–1285
13. Kirchhoff C, Imhoff AB (2010) Posterosuperior and anterosuperior impingement of the shoulder in overhead athletes—evolving concepts. Int Orthop 34:1049–1058
14. Harryman DT et al (2000) The role of the rotator interval capsule in the passive motion and stability of the shoulder. J Shoulder Elbow Surg 9(1):36–46
15. Ozunlu N, Tekeli H, Baltaci G (2011) Lateral scapular slide test and scapular mobility in volleyball players. J Athl Train 46(4): 438–444
16. Joshi M, Thigpen CA, Bunn K, Karas SG, Padua DA (2011) Shoulder external rotation fatigue and scapular muscle activation and kinematics in overhead athletes. J Athl Train 46(4):349–357
17. Ludewig PM, Reynolds JF (2009) The association of scapular kinematics and glenohumeral joint pathologies. J Orthop Sports Phys Ther 39(2):90–104
18. Burkhart SS, Morgan CD, Kibler WB (2003) The disabled throwing shoulder: spectrum of pathology part I: pathoanatomy and biomechanics. Arthroscopy 19(4):404–420
19. Jiu-jenq L, Hanten WP, Olson SL, Roddey TS, Soto-quijano DA, Lim HK, Sherwood AM (2005) Functional activity characteristics of individuals with shoulder dysfunctions. J Electromyogr Kinesiol 15(6):576–586
20. Page P (2011) Shoulder muscle imbalance and subacromial impingement syndrome in overhead athletes. Int J Sports PhysTher 6(1):51–58
21. Hong J, Barnes MJ, Leddon CE, Van Ryssegem G, Alamar B (2011) Reliability of the sitting hand press-up test for identifying and quantifying the level of scapular medial border posterior displacement in overhead athletes. Int J Sports Phys Ther 6(4):306–311
22. Nodehi-Moghadam A, Nasrin N, Kharazmi A, Eskandari Z (2013) A comparative study on shoulder rotational strength, range of motion and proprioception between the throwing athletes and non-athletic persons. Asian J Sports Med 4(1):34–40
23. Cole AK, McGrath ML, Harrington SE, Padua DA, Rucinski TJ, Prentice WE (2013) Scapular bracing and alteration of posture and muscle activity in overhead athletes with poor posture. J Athl Train 48(1):12–24
24. Freehill MT, Ebel BG, Archer KR, Bancells RL, Wilckens JH, McFarland EG, Cosgarea AJ (2011) Glenohumeral range of motion in major league pitchers: changes over the playing season. Sports Health 3(1):97–104
25. Shimizu T, Iwasaki N, Nishida K, Minami A, Funakoshi T (2012) Glenoid stress distribution in baseball players using computed tomography osteoabsorptiometry: a pilot study. Clin Orthop Relat Res 470(6):1534–1539
26. Dewan AK, Garzon-Muvdi J, Petersen SA, Jia X, McFarland EG (2012) Intraarticular abnormalities in overhead athletes are variable. Clin Orthop Relat Res 470(6):1552–1557
27. Manske RC, Grant-Nierman M, Lucas B (2013) Shoulder posterior internal impingement in the overhead athlete. Int J Sports Phys Ther 8(2):194–204
28. Burkhart SS, Morgan CD, Kibler WB (2000) Shoulder injuries in overhead athletes. The “dead arm” revisited. Clin Sports Med 19(1):125–158
29. Ruotolo C, Price E, Panchal A (2006) Loss of total arc of motion in collegiate baseball players. J Shoulder Elbow Surg 15(1):67–71
30. Pagnani MJ, Warren RF (1994) Stabilizers of the glenohumeral joint. J Shoulder Elbow Surg 3(3):173–190
31. Reinold MM, Curtis AS (2013) Microinstability of the shoulder in the overhead athlete. Int J Sports Phys Ther 8(5):601–616
32. Brumitt J, Dale RB (2009) Integrating shoulder and core exercises when rehabilitating athletes performing overhead activities. N Am J Sports Phys Ther 4(3):132–138
33. Scher S, Anderson K, Weber N, Bajorek J, Rand K, Bey MJ (2010) Associations among hip and shoulder range of motion and shoulder injury in professional baseball players. J Athl Train 45(2):191–197

Posterior Shoulder Instability

21

Giovanni Merolla, Carlo Alberto Augusti, Paolo Paladini,
Elisa De Santis, and Giuseppe Porcellini

21.1 Etiology

Posterior instability of the shoulder is a rare clinical condition with a prevalence of 1.1 in 100,000 [1] and an incidence ranging from 2 to 10 % in the general population [2–4]. Higher incidence rates are found in football and rugby players, weight lifters, paddling sports athletes, wrestlers, climbers, and young athletes in general.

Recurrent posterior instability is best viewed as a syndrome whose pathological processes are incompletely understood and where multiple predisposing factors may coexist in the same patient [5]. Factors that may play a role separately or in combination can be divided into bony and soft tissue abnormalities, which in turn may be traumatic or congenital.

G. Merolla, MD (✉)
Unit of Shoulder and Elbow Surgery, D. Cervesi Hospital,
AUSL della Romagna, Ambito Territoriale di Rimini,
Via L.V Beethoven 5, 47841 Cattolica (RN), Italy

Biomechanics Laboratory "Marco Simoncelli",
D. Cervesi Hospital, AUSL della Romagna, Ambito Territoriale
di Rimini, Via L.V Beethoven 5, 47841 Cattolica (RN), Italy
e-mail: giovannimerolla@hotmail.com; giovanni.merolla@auslrn.net

C.A. Augusti
Unit of Orthopedics and Traumatology,
Papa Giovanni XXIII Hospital, Bergamo – ASL di Bergamo,
Piazza OMS 1, Bergamo 24127, Italy
e-mail: carlo.alberto.augusti@gmail.com

P. Paladini • G. Porcellini
Unit of Shoulder and Elbow Surgery, D. Cervesi Hospital,
AUSL della Romagna, Ambito Territoriale di Rimini,
Via L.V Beethoven 5, 47841 Cattolica (RN), Italy
e-mail: palpaolo@tin.it; gporcellini@tin.it

E. De Santis
Biomechanics Laboratory "Marco Simoncelli",
D. Cervesi Hospital, AUSL della Romagna, Ambito Territoriale
di Rimini, Via L.V Beethoven 5, 47841 Cattolica (RN), Italy

Shoulder Team Rehabilitative Office, Via A. Costa 33, Forlì, Italy
e-mail: ely.desantis@gmail.com

21.1.1 Bony Abnormalities of the Glenoid

- Glenoid hypoplasia. Glenoid hypoplasia, or dysplasia of the scapular neck, is usually a bilateral, symmetric finding. It may be associated with hypoplasia of the humeral head, an elongated glenoid cavity, and/or an altered coracoid or clavicle shape [6–8].
- Posterior glenoid rim deficiency. This abnormality is defined as localized hypoplasia of the posterior glenoid, whose shape is round (J) or triangular (delta). It has been postulated as a cause of glenohumeral (GH) instability [9, 10]. A bony deficiency of the posterior-inferior glenoid rim with a craniocaudal length exceeding 12 mm may determine recurrent (atraumatic) posterior instability [11].
- Glenoid retroversion. Patients with posterior shoulder instability may show evidence of glenoid retroversion. Retroversion is excessive if the angle is greater than −7° in the sagittal plane [12]. It has been calculated that every additional 1° of retroversion increases the risk of posterior instability by 17 % [13].
- Reverse bony Bankart lesion. A fracture of the posterior-inferior glenoid rim may occur after a posterior GH dislocation. Similar to the anterior lesion, malunion with a medialized posterior fragment can cause post-traumatic posterior instability.

21.1.2 Bony Abnormalities of the Humeral Head

- Reverse Hill-Sachs fracture. It is an anteromedial humeral head compression fracture after posterior humeral dislocation.

It is unclear whether glenoid bony changes such as retroversion precede posterior instability or whether instability itself induces the bone changes [14].

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_21,

Table 21.1 Kim classification system for posterior labral tear [27]

Type	Description
1.	Incomplete detachment
2.	Incomplete and concealed avulsion (Kim lesion)
3.	Chondrolabral erosion
4.	Flap tear

21.1.3 Soft Tissue Abnormalities

- Labrum. Isolated labral tears can be classified into four types according to Kim (Table 21.1). Labral tears are frequently accompanied by stretching of the posterior-inferior aspect of the capsule. A paraglenoid labral cyst may be found in association with muscle denervation due to compressive neuropathy.
- Posterior labrocapsular periosteal sleeve avulsion (POLPSA). This lesion is characterized by stripping of the posterior labrum and the intact posterior scapular periosteum from the glenoid; the avulsion creates a redundant recess that communicates with the joint space [15, 16].
- Bennett lesion. This is an extra-articular curvilinear calcification along the posterior-inferior glenoid near the attachment of the posterior band of the inferior GH ligament (IGHL) [17]. POLPSA lesions are suspected to be the acute stage of a Bennett lesion [16].
- Posterior humeral avulsion of the GH ligament (PHAGL). A PHAGL lesion is one where the posterior IGHL has been avulsed from its humeral attachment.
- Floating PHAGL. This very rare condition is a combination of a PHAGL and a reverse Bankart lesion.
- Stretched posterior capsule. Recurrent subluxations or dislocations can stretch the capsule, giving rise to a pathological posterior-inferior capsular pouch. The excessive capsular laxity and the capsular recess may be the primary lesion in patients with atraumatic instability [18].
- Accompanying lesions including SLAP tears, superior GH ligament tear and medial GH ligament, and an enlarged axillary pouch are found in patients with posterior shoulder instability [19].

21.2 Mechanism of Injury

The causes of posterior instability range from acute traumatic injury to repetitive microtrauma and a nontraumatic etiology [5, 20, 21]. Treatment requires correct identification of pathogenesis.

Repeated microtrauma to the posterior structures of the shoulder induced by repetitive bench press lifting, overhead weight lifting, rowing, swimming, playing as blocking lineman in football, and all sport activities involving a load borne in front of the body are the most frequent causes of posterior instability. In overhead athletes loading of the shoulder in flexion and IR causes stretch and injury of the posterior IGHL and posterior-inferior joint structures. This induce a progressive posterior laxity. Patients seldom remember a particular movement or situation that caused the symptoms.

Acute traumatic posterior dislocation commonly involves a direct load on the flexed and internally rotated shoulder. It is typically related to a high-energy trauma (e.g., motorcycle accident), electrocution, seizure, or high-weight bench press exercises. A nontraumatic pathogenesis is rare. Patients with generalized ligamentous laxity are predisposed to posterior instability [14].

21.3 Clinical Examination

Patients with posterior instability usually report general shoulder pain or pain deep in the posterior aspect of the shoulder [22, 23]. The pain can be associated with loss of strength and decreased athletic performances, such as reduced bench press capacity or inability to do the same number of push-ups [5, 24, 25]. In these nonspecific conditions, it is important to avoid confusing posterior instability with outlet impingement, biceps injuries, or myofascial disorders.

A common finding in patients with posterior instability is impaired scapulothoracic mechanics, due to rotator cuff imbalance which is related especially to the subscapularis.

Some patients can subluxate their shoulder voluntarily. This is a complex therapeutic situation where it is crucial to differentiate voluntary positional instability from voluntary muscular instability [23]. The former condition consists of subluxation in a provocative position; these subjects are not gratified by their ability to subluxate the shoulder, unlike those with willful voluntary instability, who have learned to subluxate their shoulder and do it habitually. Voluntary muscular instability is non-position-dependent and suggests ligamentous laxity or muscle imbalance and should not be considered as a form of true posterior instability.

Clinical examination plays a crucial role in the diagnosis of posterior instability. The patient usually reports nonspecific symptoms and the condition is often misdiagnosed. As in all instances both shoulders need to be evaluated for appearance, motion, muscle tropism, muscle strength, scapular tracking, and neurovascular status, including the axillary nerve, to elicit symptoms of posterior instability.

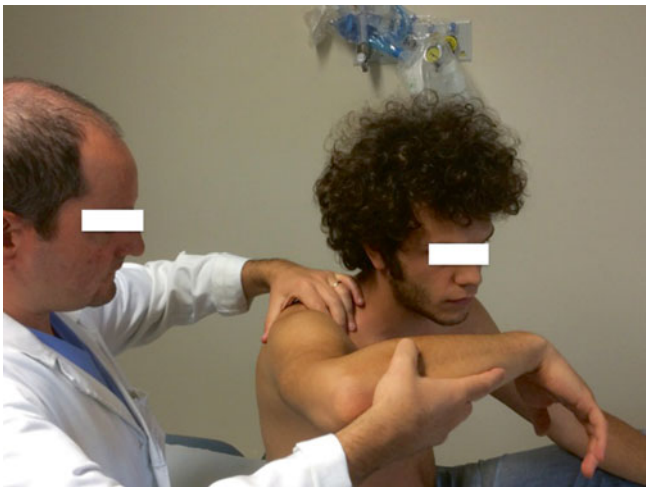


Fig. 21.1 Jerk test: the patient is standing, the arm is flexed and internally rotated, and the examiner pushes the flexed elbow posteriorly and the shoulder girdle anteriorly. The test is positive if a sudden jerk associated with pain occurs

Range of motion (ROM) is often normal and symmetric [21]. Some patients may present increased external rotation (ER) and a slight loss of internal rotation (IR).

A number of specialized tests are especially useful for a correct diagnosis:

- Apprehension test: an axial load is applied to the flexed, adducted, and internally rotated shoulder [21]. The test is positive if the patient experiences pain and a feeling of instability.
- Jerk test: the examiner standing next to the affected shoulder holds the elbow in one hand and the distal clavicle and scapular spine in the other. The arm is flexed and internally rotated. The examiner pushes the flexed elbow posteriorly and the shoulder girdle anteriorly (Fig. 21.1). The test is positive if a sudden jerk associated with pain occurs as the subluxated humeral head relocates in the glenoid fossa [26].
- Kim test: it is performed with the patient seated and the arm in 90° of abduction. An axial load is applied and the arm is slowly brought in forward elevation (Fig. 21.2). The test is positive if the patient experiences pain and a posterior subluxation [27].
- A combination of positive Kim and jerk tests has 97 % sensitivity for posterior instability [27].
- Posterior stress test: with the patient seated, the examiner stabilizes the medial border of the scapula and applies a posterior force with the arm in 90° forward flexion, adduction, and IR. The test is positive if the shoulder subluxates or dislocates with pain and/or apprehension.
- Load and shift test: with the patient supine and the arm in forward flexion and abduction, the humeral head is loaded while anterior and posterior stresses are applied (Fig. 21.3a, b). Excessive inferior translation of the humerus on the glenoid is often associated with posterior subluxation [22].
- Sulcus test: a downward traction is applied with the patient seated and the arm at the side in neutral position. The test is positive if a sulcus (depressed area) appears lateral or inferior to the acromion. A positive test may suggest inferior GH instability [2, 22, 28].

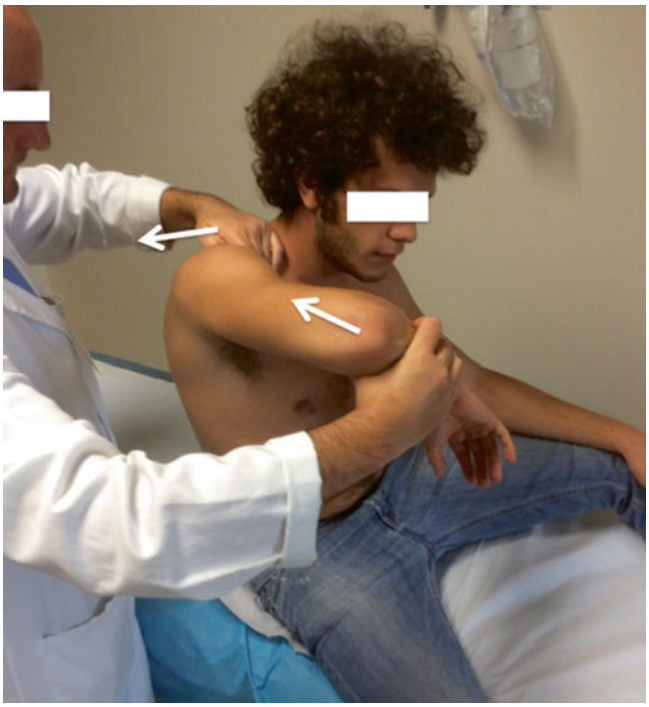


Fig. 21.2 Kim test: An axial load is applied on the arm in 90° of abduction and slowly brought in forward elevation (*white arrows*). The test is positive if the patient experiences pain and a posterior subluxation

21.4 Diagnostic Imaging

Diagnostic imaging is crucial to detect direct and indirect signs of posterior instability.

Plain radiographs including an anterior-posterior view, a true anterior-posterior view (Grashey view), a scapular (Y) lateral view, and, especially, an axillary lateral view provide essential information about glenoid and humeral head morphology and the presence of any bone defects.

CT scans are useful to evaluate bony morphology, particularly small bony defects or fractures such as reverse bony Bankart lesions. Three-dimensional reconstructions are very useful for preoperative planning. CT is endowed with 86.7 % sensitivity and 83.3 % specificity for posterior glenoid bone loss [29].

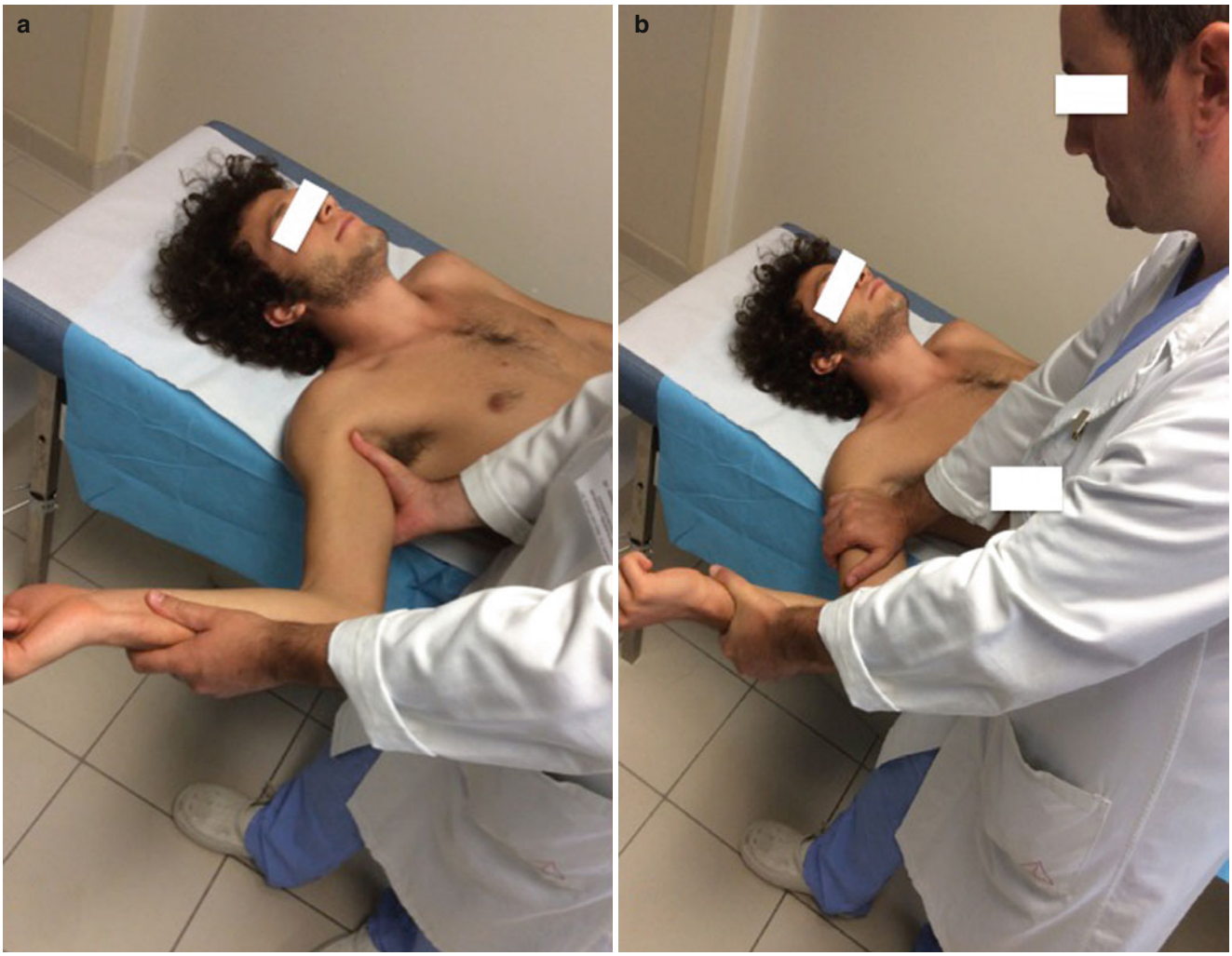


Fig. 21.3 (**a, b**) Load and shift test: the arm is placed in slight flexion and abduction (20°); the humeral head is loaded while anterior and posterior stresses are applied

Magnetic resonance imaging (MRI) and arthrography (MRA) accurately detect soft tissue injuries, in particular capsule lesions and partial or complete labral tears (Fig. 21.4). MRA is highly sensitive and specific for labral tears (91.9 and 96 %, respectively), SLAP lesions (89 and 93.8 %, respectively), and Hill-Sachs fractures (93.3 % accuracy) [30].

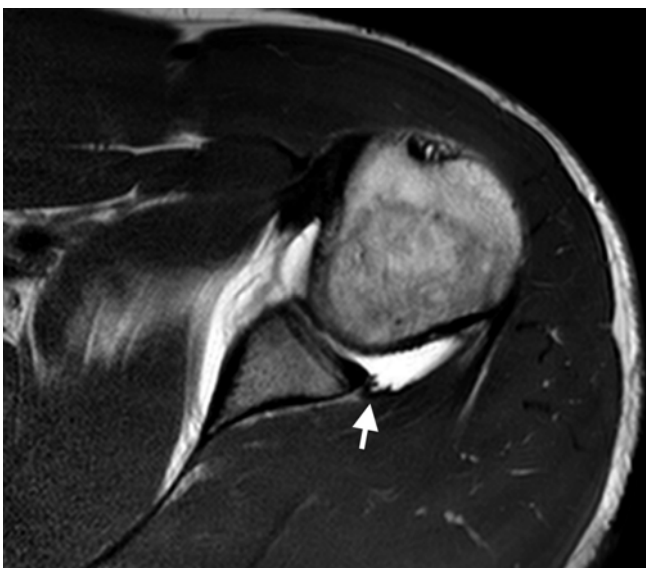


Fig. 21.4 Contrast-enhanced magnetic resonance (MR) of the left shoulder depicting a posterior labral detachment (*white arrow*) in a professional volleyball player

21.5 Treatment Strategy

The treatment of posterior instability in athletes depends on symptom severity, including pain intensity, degree of impairment of athletic performance, and pathological features of the joint lesion. All athletes should be encouraged to undertake conservative treatment including physical therapy and rehabilitation as an initial approach [20, 31]. An appropriate program of strengthening and proprioception exercises can reduce pain and improve stability in most cases of posterior and multidirectional instability [20, 31]. Subjects with

ligamentous laxity and repetitive microtrauma may benefit from physical therapy [21], whereas conservative treatment is less successful in patients with a history of shoulder joint trauma [20, 21, 31]. To our knowledge there is no evidence in the literature for the ability of braces or orthoses to prevent recurrence during games/matches.

Patients with clear symptomatic posterior labral injury rarely respond to conservative treatment and are therefore candidates for surgical management [14, 32]. If nonoperative treatment fails to restore joint stability and reduce pain during sport activities, surgery followed by an appropriate postoperative rehabilitation program is recommended. Voluntary posterior dislocation is a contraindication for surgical stabilization [33], even though arthroscopic stabilization seems to afford satisfactory outcomes [34]. Voluntary or posterior instability is not uncommon in professional swimmers and gymnasts, who have multi-joint capsule and ligament laxity [35]. Some of these patients may move from posterior positional to voluntary instability; in positional instability the humeral head subluxates posteriorly when the arm is abducted over 90° and relocates spontaneously when the arm returns along the side [33].

In our view management of posterior instability includes nonoperative treatment for at least 6 months for in-season athletes who complain of pain and a moderate feeling of instability but can tolerate training and competition. Considering that most of these athletes complain of pain and subluxation rather than complete dislocation, we recommend postponing surgery after the season, when postoperative rehabilitation and shoulder training programs are easier to follow. This strategy is consistent with methods described by other colleagues [36] and is the result of a 20-year experience in shoulder and elbow surgery.

21.6 Operative Technique

Although open surgery has been the procedure of choice for many years (deltoid split, release of the interval between infraspinatus and teres minor to incise the capsule and expose the posterior labrum) [37], most surgical procedures are now performed arthroscopically with similar outcomes [38–40].

Arthroscopy is performed under general anesthesia with an interscalene block of the brachial plexus. Examination under anesthesia shows a positive posterior drawer sign that confirms the diagnosis of posterior instability (Fig. 21.5a, b). The patient lies in lateral decubitus position with the shoulder in approximately 30° of abduction and 15° of forward flexion and a traction of 5 kg applied to the arm. After establishing a posterior portal about 2 cm inferior to the posterior acromial angle [41], the anterior-superior portal is created by the inside-out technique using a Wissinger rod [42]; finally, an accessory superolateral portal is created with the in-out technique [38]. The diagnosis of posterior instability is confirmed when a reverse Bankart lesion [39] is detected from the anterior portal (Fig. 21.6). The posterior-inferior capsular redundancy is evaluated first with the aforementioned intraoperative drawer sign, to determine the posterior subluxation of the humeral head, and next with a probe to test the elasticity of the posterior capsular tissue. One or two absorbable suture anchors are placed on the posterior glenoid to repair the reverse Bankart lesion (Fig. 21.7a, b). One or two posterior capsular plications are generally required to tighten the capsule; the anterior capsule may also be plicated if redundant. The capsule is shifted superiorly using curved suture hooks (Linvatec, Largo, FL, USA) and sutured to the posterior labrum with sliding knots. Our group and other colleagues [36] consider large, engaging reverse Hill-Sachs lesion, large reverse bony Bankart lesion, and reverse HAGHL as contraindications for arthroscopic treatment of posterior shoulder instability. In patients with previous failed surgery, we prefer arthroscopy-assisted posterior shoulder stabilization using an iliac bone graft [43].

21.7 Results of Surgical Management

Arthroscopic posterior shoulder stabilization provides 89–93 % good-excellent results in athletes [24, 25, 38, 44]. In a retrospective review of 27 patients, Kim et al. [25] described overall satisfactory outcomes with only one recurrence and an average loss of one vertebral level of IR. Wolf and Eakin [38] and Bradley et al. [24] reported a 90 % rate of return to play, whereas Radkowski et al. [44] reported that 89 % of throwing athletes and 93 % of non-throwing athletes had good or excellent results, although only 55 % of throwing athletes returned to their preinjury level compared with 71 % of non-throwers.

21.8 Rehabilitation and Return to Play

An appropriate shoulder rehabilitation program is essential for balanced muscle activity and to prevent worsening of pain and shoulder dysfunction. As described above, repeated overhead movements in some sports may induce posterior capsule weakness and sometimes tears of the glenoid labrum, which predispose the shoulder joint to subluxation [4]. These conditions are found especially during the follow-through phase of pitching in baseball, the backhand stroke of racquet sports, and the pull-through phase of swimming, where the arm is in forward flexion and is being actively adducted and internally rotated [45, 46]. Fatigue of the dynamic stabilizers is also a contributing factor to shoulder joint instability [47]. Rehabilitation of athletes with traumatic instability should

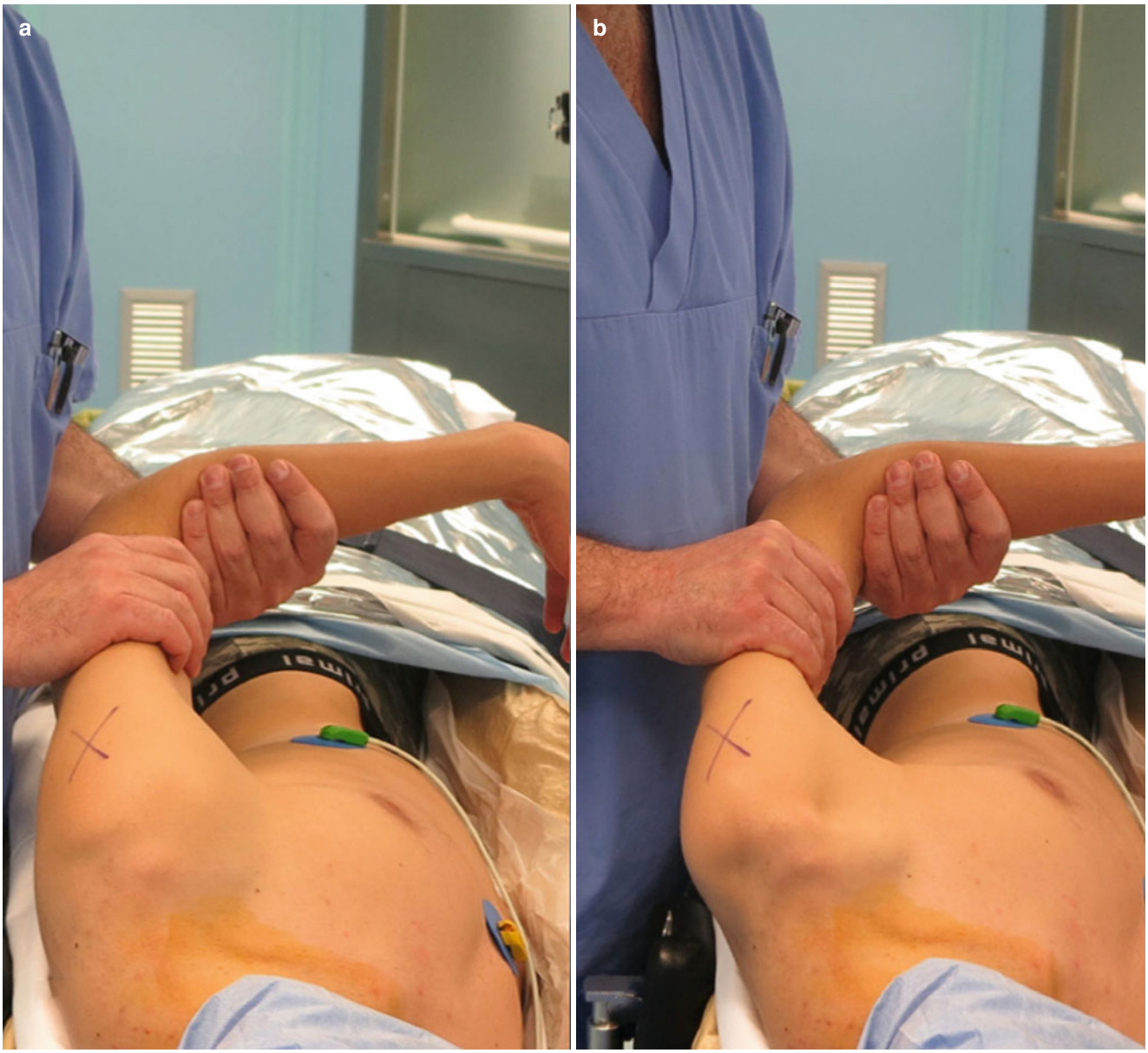


Fig. 21.5 (**a, b**) Positive drawer sign with the patient under anesthesia (examination under anesthesia, EUA). The humeral head is posteriorly dislocated when an anterior to posterior axial load is applied

include four phases: (1) acute, (2) intermediate, (3) advanced, and (4) return to activities.

In the acute phase motion is restricted to avoid tissue attenuation. A short period (1–2 weeks) of immobilization may be required to promote stability. Ice, laser therapy, and transcutaneous electrical nerve stimulation (TENS) may be applied to control pain and inflammation. Next, passive ROM (PROM) in a restricted arc of motion and active assisted "pain-free" exercises using the L-bar may be begun. Pain during rehabilitation indicates that the exercise is not being done correctly or is not appropriate for the current phase of recovery. Closed kinetic chain exercises, enhancing muscle coactivation, and scapular stability without shear forces across the shoulder joint should be performed using a fixed support. These exercises facilitate the recovery of function of the rotator cuff, enhancing joint stability and stimulating muscle coactivation and proprioception.

The main goal of the intermediate phase of recovery is to restore shoulder mobility while strengthening exercises of the scapular stabilizers and rotator cuff progress.

During rehabilitation the focus must be on scapular position and control, with tightened abdominal muscles holding the spine in neutral position and correct postural alignment, because proximal stability must be acquired before distal mobility.

Closed chain exercises can now be extended to weight-bearing on unstable surfaces such as a Swiss ball, to enhance

neuromuscular control at the reflex level; isotonic strengthening exercises including ER and IR with tubing are also initiated.

The advanced strengthening phase focuses on continued strength and endurance, dynamic stability, and neuromuscular control near the end range through progressive exercises such as plyometric exercises to promote return to their sports.

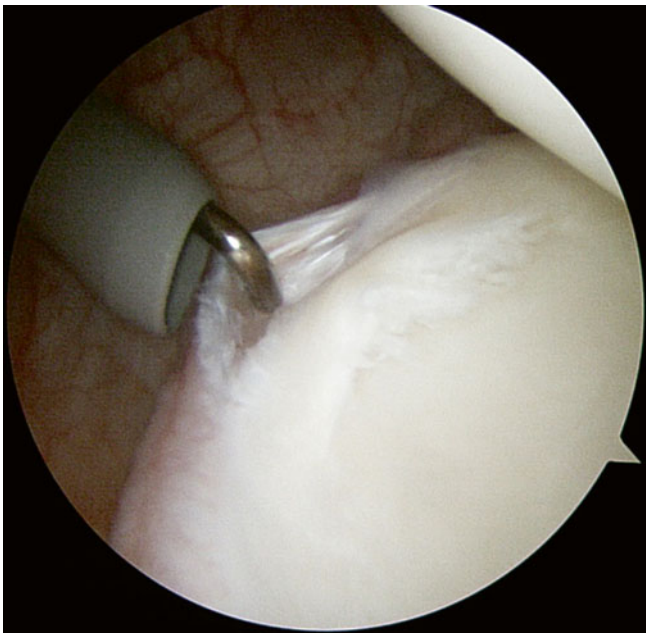


Fig. 21.6 Reversed posterior Bankart lesion in the left shoulder seen from the anterior portal

A maintenance program should continue to be followed after the return to activities. Whenever possible the specific technique of their sport should be analyzed with the coaches, to determine whether it has been the predisposing cause of the instability [47, 48].

Candidates for surgical intervention should follow a preoperative strengthening program for the scapular stabilizers, to assist in postoperative rehabilitation. Subscapularis muscle strength should be specially targeted, because it is the most important dynamic posterior stabilizer of the GH joint [14].

Rehabilitation following posterior shoulder stabilization and/or labrum repair to reduce the risk of postoperative recurrence is slightly more conservative than after anterior shoulder stabilization. The shoulder is immobilized in a sling (15° abduction and neutral or slight ER to minimize the tension on the posterior capsule) for 3 weeks. PROM exercises are generally allowed after sling removal; nevertheless, early restricted exercises may be performed immediately after surgery with the shoulder abducted to 45° and neutral IR and ER to approximately 20–25°. Over the first 8 weeks of recovery, the patient's PROM is gradually increased, taking care that this does not induce excessive posterior shoulder laxity and enhancing strength and dynamic stabilization. By the 10–12th week, the patient should have regained an almost full PROM. In this phase strengthening exercises for external rotators and the scapular musculature help to improve neuromuscular control and dynamic stabilization [48]. In the weeks from the 12th to the 26th, strengthening and dynamic stabilization exercises are increased and associated with isotonic strengthening exercises.

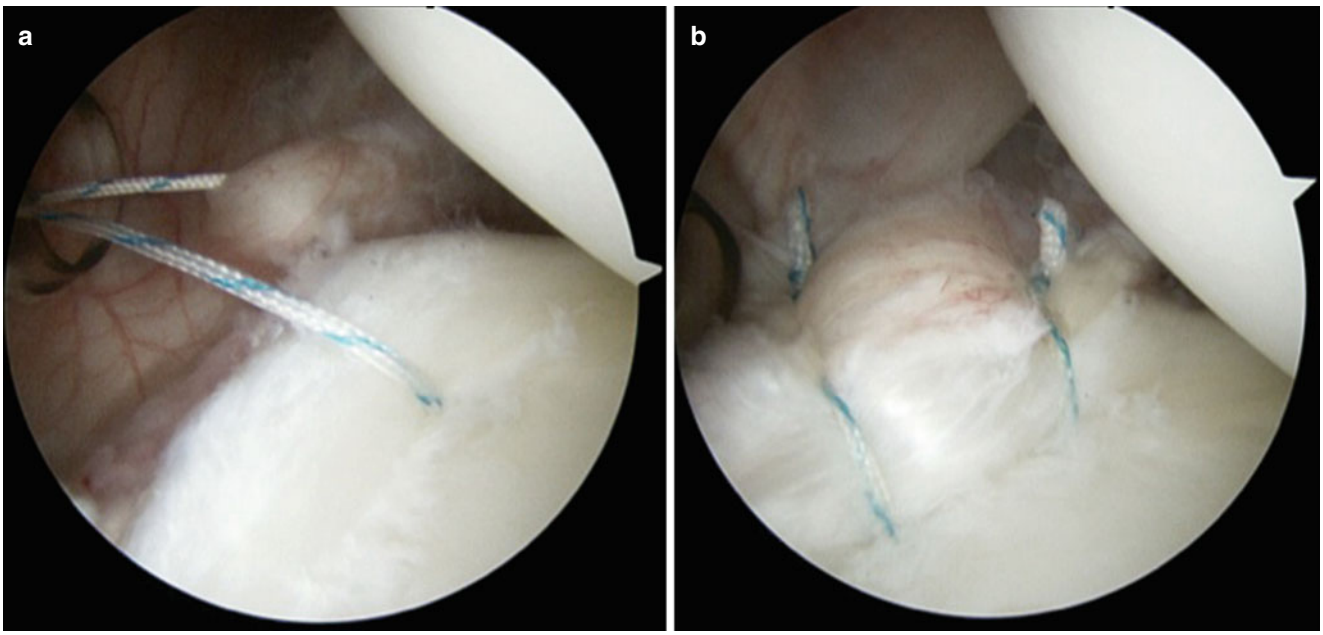


Fig. 21.7 (**a, b**) Posterior labrum repair with suture anchor. (**a**) The suture is passed through the posterior band of the inferior glenohumeral ligament (IGHL), (**b**) labrum reattachment with two suture anchors using sliding knots

Return to sport-specific drills depends on the sport and position they are returning to. For overhead throwers, especially baseball pitchers, a gradual return to sport is suggested at 6–7 months with a return to the actual game at approximately 9 months. For non-throwing athletes and those involved in contact sports, the return to competition is recommended at 7–9 months depending on their role [33, 48].

References

1. Robinson CM, Seah M, Akhtar MA (2011) The epidemiology, risk of recurrence, and functional outcome after an acute traumatic posterior dislocation of the shoulder. J Bone Joint Surg Am 93(17):1605–1613 [cited 2014 Jun 14]
2. Antoniou J, Duckworth DT, Harryman DT (2000) Capsulolabral augmentation for the management of posteroinferior instability of the shoulder. J Bone Joint Surg Am 82(9):1220–1230 [cited 2014 Jun 13]
3. Boyd HB, Sisk TD (1972) Recurrent posterior dislocation of the shoulder. J Bone Joint Surg Am 54(4):779–786 [cited 2014 Jun 13]
4. McLaughlin HL (1952) Posterior dislocation of the shoulder. J Bone Joint Surg Am 24-A-3:584–590 [cited 2014 Jun 13]
5. Robinson CM, Aderinto J (2005) Recurrent posterior shoulder instability. J Bone Joint Surg Am 87(4):883–892 [cited 2014 May 27]
6. Shah N, Tung GA (2009) Imaging signs of posterior glenohumeral instability. AJR Am J Roentgenol 192(3):730–735 [cited 2014 Jun 13]
7. Trout TE, Resnick D (1996) Glenoid hypoplasia and its relationship to instability. Skeletal Radiol 25(1):37–40 [cited 2014 Jun 13]
8. Wirth MA, Lyons FR, Rockwood CA (1993) Hypoplasia of the glenoid. A review of sixteen patients. J Bone Joint Surg Am 75(8):1175–1184 [cited 2014 Jun 13]
9. Fuchs B, Jost B, Gerber C (2000) Posterior-inferior capsular shift for the treatment of recurrent, voluntary posterior subluxation of the shoulder. J Bone Joint Surg Am 82(1):16–25 [cited 2014 Jun 13]
10. Edelson JG (1995) Localized glenoid hypoplasia. An anatomic variation of possible clinical significance. Clin Orthop Relat Res 321:189–195 [cited 2014 Jun 13]
11. Weishaupt D, Zanetti M, Nyffeler RW, Gerber C, Hodler J (2000) Posterior glenoid rim deficiency in recurrent (atraumatic) posterior shoulder instability. Skeletal Radiol 29(4):204–210 [cited 2014 Jun 13]
12. Brewer BJ, Wubben RC, Carrera GF (1986) Excessive retroversion of the glenoid cavity. A cause of non-traumatic posterior instability of the shoulder. J Bone Joint Surg Am 68(5):724–731 [cited 2014 Jun 13]
13. Owens BD, Campbell SE, Cameron KL (2013) Risk factors for posterior shoulder instability in young athletes. Am J Sports Med 41(11):2645–2649 [cited 2014 Jun 13]
14. Provencher MT, LeClere LE, King S, McDonald LS, Frank RM, Mologne TS et al (2011) Posterior instability of the shoulder: diagnosis and management. Am J Sports Med 39(4):874–886 [cited 2014 Jun 13]
15. Simons P, Joekes E, Nelissen RG, Bloem JL (1998) Posterior labrocapsular periosteal sleeve avulsion complicating locked posterior shoulder dislocation. Skeletal Radiol 27(10):588–590 [cited 2014 Jun 13]
16. Yu JS, Ashman CJ, Jones G (2002) The POLPSA lesion: MR imaging findings with arthroscopic correlation in patients with posterior instability. Skeletal Radiol 31(7):396–399 [cited 2014 Jun 13]
17. Wright RW, Paletta GA (2004) Prevalence of the Bennett lesion of the shoulder in major league pitchers. Am J Sports Med 32(1):121–124 [cited 2014 Jun 13]
18. Dewing CB, McCormick F, Bell SJ, Solomon DJ, Stanley M, Rooney TB et al (2008) An analysis of capsular area in patients with anterior, posterior, and multidirectional shoulder instability. Am J Sports Med 36(3):515–522 [cited 2014 Jun 13]
19. Savoie FH, Holt MS, Field LD, Ramsey JR (2008) Arthroscopic management of posterior instability: evolution of technique and results. Arthroscopy 24(4):389–396 [cited 2014 Jun 13]
20. Fronek J, Warren RF, Bowen M (1989) Posterior subluxation of the glenohumeral joint. J Bone Joint Surg Am 71(2):205–216 [cited 2014 Jun 14]
21. Schwartz E, Warren RF, O'Brien SJ, Fronek J (1987) Posterior shoulder instability. Orthop Clin North Am 18(3):409–419 [cited 2014 Jun 14]
22. Hawkins RJ, Koppert G, Johnston G (1984) Recurrent posterior instability (subluxation) of the shoulder. J Bone Joint Surg Am 66(2):169–174 [cited 2014 Jun 14]
23. Millett PJ, Clavert P, Hatch GFR, Warner JJP (2006) Recurrent posterior shoulder instability. J Am Acad Orthop Surg 14(8):464–476 [cited 2014 Jun 14]
24. Bradley JP, Baker CL, Kline AJ, Armfield DR, Chhabra A (2006) Arthroscopic capsulolabral reconstruction for posterior instability of the shoulder: a prospective study of 100 shoulders. Am J Sports Med 34(7):1061–1071 [cited 2014 Jun 5]
25. Kim S-H, Ha K-I, Park J-H, Kim Y-M, Lee Y-S, Lee J-Y et al (2003) Arthroscopic posterior labral repair and capsular shift for traumatic unidirectional recurrent posterior subluxation of the shoulder. J Bone Joint Surg Am 85-A:1479–1487
26. Blasier RB, Soslowsky LJ, Malicky DM, Palmer ML (1997) Posterior glenohumeral subluxation: active and passive stabilization in a biomechanical model. J Bone Joint Surg Am 79(3):433–440 [cited 2014 Jun 14]
27. Kim S-H, Park J-S, Jeong W-K, Shin S-K (2005) The Kim test: a novel test for posteroinferior labral lesion of the shoulder–a comparison to the jerk test. Am J Sports Med 33(8):1188–1192 [cited 2014 May 26]
28. Antoniou J, Harryman DT (2001) Posterior instability. Orthop Clin North Am 32(3):463–473, ix [cited 2014 Jun 14]
29. Chalmers PN, Hammond J, Juhan T, Romeo AA (2013) Revision posterior shoulder stabilization. J Shoulder Elbow Surg 22(9):1209–1220 [cited 2014 Jun 13]
30. Probyn LJ, White LM, Salonen DC, Tomlinson G, Boynton EL (2007) Recurrent symptoms after shoulder instability repair: direct MR arthrographic assessment–correlation with second-look surgical evaluation. Radiology 245(3):814–823 [cited 2014 Jun 16]
31. Burkhead WZ, Rockwood CA (1992) Treatment of instability of the shoulder with an exercise program. J Bone Joint Surg Am 74:890–896
32. Provencher MT, Bell SJ, Menzel KA, Mologne TS (2005) Arthroscopic treatment of posterior shoulder instability: results in 33 patients. Am J Sports Med 33:1463–1471
33. Merolla G, De Santis E, Cools AMJ, Porcellini G (2015) Functional outcome and quality of life after rehabilitation for voluntary posterior shoulder dislocation: a prospective blinded cohort study. Eur J Orthop Surg Traumatol 25:263–272
34. Greiwe RM, Galano G, Grantham J, Ahmad CS (2012) Arthroscopic stabilization for voluntary shoulder instability. J Pediatr Orthop 32:781–786
35. Hiley MJ, Yeadon MR (2012) Achieving consistent performance in a complex whole body movement: the Tkatchev on high bar. Hum Mov Sci 31:834–843
36. Ward JP, Bradley JP (2013) Decision making in the in-season athlete with shoulder instability. Clin Sports Med 32:685–696
37. Hawkins RJ, Janda DH (1996) Posterior instability of the glenohumeral joint. A technique of repair. Am J Sports Med 24(3):275–278 [cited 2014 Jul 27]

38. Wolf EM, Eakin CL (1998) Arthroscopic capsular plication for posterior shoulder instability. Arthroscopy 14:153–163
39. Mair SD, Zarzour RH, Speer KP (1998) Posterior labral injury in contact athletes. Am J Sports Med 26(6):753–758 [cited 2014 Jul 27]
40. Bottoni CR, Franks BR, Moore JH, DeBerardino TM, Taylor DC, Arciero RA (2005) Operative stabilization of posterior shoulder instability. Am J Sports Med 33(7):996–1002 [cited 2014 Jun 14]
41. Snyder SJ (2003) Shoulder Arthroscopy, Lippincott Williams & Wilkins, New York, p. 22–28
42. Matthews LS, Zarins B, Michael RH, Helfet DL (1985) Anterior portal selection for shoulder arthroscopy. Arthroscopy 1:33–39
43. Schwartz DG, Goebel S, Piper K, Kordasiewicz B, Boyle S, Lafosse L (2013) Arthroscopic posterior bone block augmentation in posterior shoulder instability. J Shoulder Elbow Surg 22:1092–1101
44. Radkowski CA, Chhabra A, Baker CL, Tejwani SG, Bradley JP (2008) Arthroscopic capsulolabral repair for posterior shoulder instability in throwing athletes compared with nonthrowing athletes. Am J Sports Med 36(4):693–699 [cited 2014 Jul 27]
45. Tibone JE, Bradley JP (1993) The treatment of posterior subluxation in athletes. Clin Orthop Relat Res 291:124–37
46. Tannenbaum EP, Sekiya JK (2013) Posterior shoulder instability in the contact athlete. Clin Sports Med 32:781–796
47. Jaggi A, Lambert S (2010) Rehabilitation for shoulder instability. Br J Sports Med 44:333–340
48. Wilk KE, Macrina LC (2013) Nonoperative and postoperative rehabilitation for glenohumeral instability. Clin Sports Med 32: 865–914

Shoulder Rotator Cuff Repair

22

Araldo Causero, Alessandro Beltrame,
and Paolo Di Benedetto

22.1 Introduction

Rotator cuff represents the most important dynamic stabilizer of the shoulder and it is often involved in sports injuries. Sporting activities as throwing, swimming, and tennis require an extreme use of overhead movements; during these movements impingement and instability syndrome may occur, becoming the main cause of pain and dysfunction in elite athlete's shoulder.

Usually professional athletes do not suffer from a single isolated traumatic event and shoulder pain is gradual and progressive. If a single event is reported, normally the patients may describe a preexisting shoulder pain of variable duration or previous episodes of resolved pain.

Successful management of these patients requires a deep knowledge of the pathology, as well as an accurate and complete diagnosis [1].

22.2 Etiology and Injury Mechanism

Cuff rotator tears can be secondary to various etiologies: first of all impingement syndrome and then instability (in particular in overhead athletes) or trauma (in particular in contact sports).

There are four types of impingement syndrome: primary impingement, secondary impingement, internal impingement, and coracoid impingement.

Primary impingement is due to the friction of supraspinatus tendon and subacromial bursa under the acromion, which often has an abnormal shape t. This is the first and the original impingement form described by Neer [2].

A. Causero (✉) • A. Beltrame • P. Di Benedetto
Clinica Ortopedica – Azienda Ospedaliero-Universitaria di Udine,
Pl. Santa Maria della Misericordia 15, Udine 33100, Italy
e-mail: causero.araldo@aoud.sanita.fvg.it; beltrame.alessandro@aoud.sanita.fvg.it; dibenedetto.paolo@aoud.sanita.fvg.it

Secondary impingement is due to a dynamic glenohumeral instability, which generally is anterior. Involved structures may be subscapularis tendon, supraspinatus tendon, and anterior capsule. SLAP (superior labral anteroposterior) lesion may contribute to articular symptoms.

Internal impingement is described as a conflict between posterior glenoid rim and postero-superior rotator cuff insertion into the greater tuberosity. This condition is the most frequent finding in throwing athletes. Jobe in 1995 indicated five risk areas in this kind of impingement: postero-superior labrum, articular side of the rotator cuff, bone of the postero-superior glenoid, greater tuberosity, and anterior capsulo-labral complex [3]. Coracoid syndrome arises during forward flexion and internal rotation of the shoulder. Etiology seems to be linked to a decreased distance between the coracoid and the anterior humeral structures [4].

Rotator cuff damage may be variable: fraying of the articular or bursal side of the cuff, PASTA (partial articular supraspinatus tendon avulsion from its insertion) lesions, PAINT (partial articular tears with intratendinous extension) lesion, intratendinous tears, and complete tears of the rotator cuff. Labral abnormalities can be also found in throwing athletes: postero-superior region is the most commonly involved, and SLAP lesions can often occur [5–10].

Recurring microtrauma, fatigue, and dyssynchrony of the dynamic stabilizers of the shoulder and scapula may also contribute to anterior translation of the humeral head during throwing motion. This can be considered as an anterior subluxation of the humeral head: impingement syndrome between rotator cuff and posterior glenoid rim can occur during abduction and maximal intrarotation of the shoulder. This may be the reason why it is frequent to find a posterior impingement syndrome associated with an anterior instability [7].

Other factors involved in shoulder injuries, especially in throwing athletes, may be tensile failure of the rotator cuff muscle (supraspinatus, infraspinatus, and teres minor come under significant stress during throwing); poor scapular mechanics (as an imbalance between glenohumeral and

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_22,

scapulo-thoracic rotation); rotator cuff imbalance, between internal and external rotators; anterior capsular laxity, in particular of the anterior band of inferior glenohumeral ligament (AB-IGHL) (which is the most important static stabilizer avoiding anterior translation of the humeral head, especially when the arm is abducted, extended, and externally rotated); and posterior capsular retraction with a resulting decrease in internal rotation, frequently found on the inferior part of the capsule in overhead athletes.

22.3 Clinical and Diagnostic Examination

22.3.1 Clinical Examination

Clinical examination begins with inspection. Obtaining a good view of both shoulders and thorax is mandatory. Shoulders must be explored for potential asymmetry between them, in particular checking the infraspinatus and supraspinatus fossae for muscular atrophy, which may be caused by chronic rotator cuff tear or suprascapular neuropathy. Scars from previous surgeries must be investigated.

Shoulder range of motion (ROM) in all directions must be assessed, taking note of potential discrepancies between active and passive motions. ROM of a normal shoulder is 180° flexion, 50° extension, 180° abduction, 80° external rotation (both at 0° and 90° abduction), and 100° internal rotation. Glenohumeral joint is responsible of 2/3 of the total ROM of the shoulder, while 1/3 is due to the scapular movement over the ribs.

Usually shoulders with rotator cuff tear show a reduction in active ROM (both for the pain for partial or small tears and for the complete tear of the tendon), while passive ROM remains normal or slightly limited.

During the motion tests, it is important to observe also the scapular rhythm, which represents the combined movement between the glenohumeral joint and the scapula. Usually this has a 2:1 ratio, confirming the different roles played in the ROM by glenohumeral and scapulo-thoracic joints. Scapular "dysrhythmia" may be caused by intra-articular adhesions (that lead to a reduction of the glenohumeral movement, keeping intact the contribution of the scapula) or by scapular winging (to explore the scapular stabilizers, it is useful to ask the patient to push against the wall).

The second phase of shoulder examination is palpation. Acromioclavicular and sterno-clavicular joints must be explored for instability and pain during digital pressure. The long head of biceps brachii (LHBB) tendon runs in its groove, situated medial to the greater tuberosity, and it may be painful upon palpation if tendinitis or tendon instability is present.

Muscular strength in athletes with rotator cuff injuries may be altered because of torn tendon, suprascapular neuropathy, or pain. Therefore, it is important to evaluate independently the muscular strength of every single muscle. This is evaluated with a 5-point scale and compared to the contralateral shoulder (5 = symmetric strength, 4 = decrease in strength against resistance, 3 = resistance to gravity only, 2 = resistance to gravity with assistance, 1 = visible contraction of the muscle, 0 = no muscular contraction) [11].

Specific tests are used to assess muscular strength and integrity. If pain is produced during the tests, they are considered as positive. The most used in the clinical practice are the following:

- Jobe's relocation test (or empty-can test) (supraspinatus): the arm is brought to 90° abduction and then it is flexed forward to 30° and internally rotated (so the thumbs point to the ground). The patient must hold this position while a downward force is applied by the examiner.
- Patte's test (infraspinatus): arms are placed at patient's sides, with elbow flexed to 90°. The patient must hold this position while the examiner applies a force in internal rotation.
- Lift-off test [12] (subscapularis): the dorsum of the ipsilateral hand is placed against the back of the patient (in maximal internal rotation of the shoulder). Then the patient is asked to lift the hand off from the back. The test is considered positive if the patient is unable to perform it.
- Bear Hug test [13]: the palm of the hand of the involved side is placed on the opposite shoulder with the fingers kept extended and the elbow just anterior to the trunk. The test is considered positive if the patient is not able to hold the starting position while the examiner tries to pull away the hand from the shoulder applying an external rotating force perpendicularly to the forearm.

As discussed above, different kinds of impingement syndrome may be present and even cause or worsen a rotator cuff tear, so they have to be correctly assessed with the following tests:

- Neer's sign: the examiner lifts up the patient's arm while stabilizing the scapula with the other hand. If pain is reproduced, the test is considered positive [14].
- Hawkins' test: the arm is placed at 90° forward flexion and 15° adduction while an internal rotating force is applied. The test is positive if pain is reproduced [15].
- Yocum's test: the patient is asked to lift up the elbow keeping the hand placed on the unaffected shoulder. The test is positive if pain is reproduced [16].

The clinical assessment of shoulder laxity and instability is explained in another chapter.

22.3.2 Radiography

Plain X-rays should always be the first imaging examination performed in a patient with a painful shoulder.

Although X-rays are generally not helpful in making the diagnosis of a rotator cuff tear, in some case, they may be useful, at least to exclude other pathologies. Anteroposterior, axillary, and outlet views should be always performed. X-rays are the best option to assess the presence of calcifications, which sometimes may be the source of pain. Humeral head position must be checked because its superior migration may be an indirect sign of massive tears. The Hill-Sachs defect is suggestive of recurrent anterior dislocation. X-rays are also useful to evaluate the presence of os acromiale and to assess the width of the subacromial space and the acromion's morphology. Acromion can be described as flat, curved, or hooked, according to Bigliani's classification. It was demonstrated that the latter two morphologies are associated with a higher prevalence of impingement syndrome and rotator cuff tears [17].

22.3.3 Magnetic Resonance Imaging

Magnetic resonance imaging is considered the gold standard to study rotator cuff lesions. This exam consents to distinguish between partial-thickness and full-thickness rotator cuff tears, also measuring the gap dimension. In chronic lesions it allows to assess tendon retraction and the grade of muscular atrophy.

Partial-thickness cuff tears can be found both on the articular and bursal sides and they are usually classified according to Ellman's classification in grade 1 if thinner than 3 mm, grade 2 if included between 3 and 6 mm, and grade 3 if thicker than 6 mm.

Full-thickness tears are described depending on lesion's shape according to Ellmann and Gartsman's classification (type 1 is crescent shaped, type 2 is reverse L shaped, type 3 is L shaped, type 4 is trapezoidal, while type 5 includes massive tears, considered as full-thickness lesions of at least two tendons).

Retraction is classified in early if tendon is between its normal insertion and the glenoid, late if tendon is at the level of the glenoid, and very late if tendon is retracted beyond the glenoid. Repair of the latter two grades may be difficult or impossible. Usually retraction tends to aggravate with time. Chronic lesions are usually associated to progressive muscular atrophy. The extent of atrophy is classified according to the Goutallier index, based on fatty infiltration of the muscle. In grade 0 there is no fatty infiltration, in grade 1 some fat is present among muscular fibers, grade 2 is characterized by a fatty infiltration inferior than 50 %, in grade 3 muscular tissue is as represented as fat, and in grade 4 fatty infiltration is superior than 50 %. An indirectly proportional relationship has already been demonstrated between muscular atrophy and functional outcome after surgical repair [18].

A contrast medium like gadolinium may be used to enhance the visualization of the labrum and of the rotator cuff articular surface, highlighting partial-thickness articular cuff tears [19] (Fig. 22.1, 22.2, 22.3, 22.4, and 22.5).

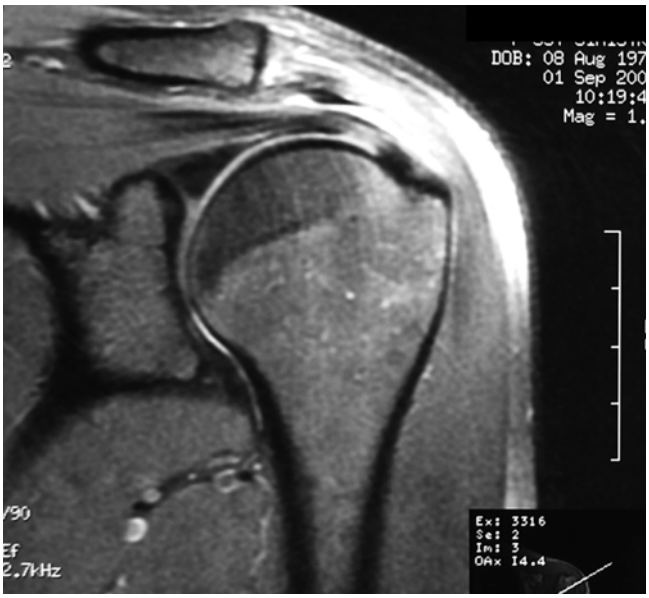


Fig. 22.1 MR – partial-thickness cuff tear on the articular side

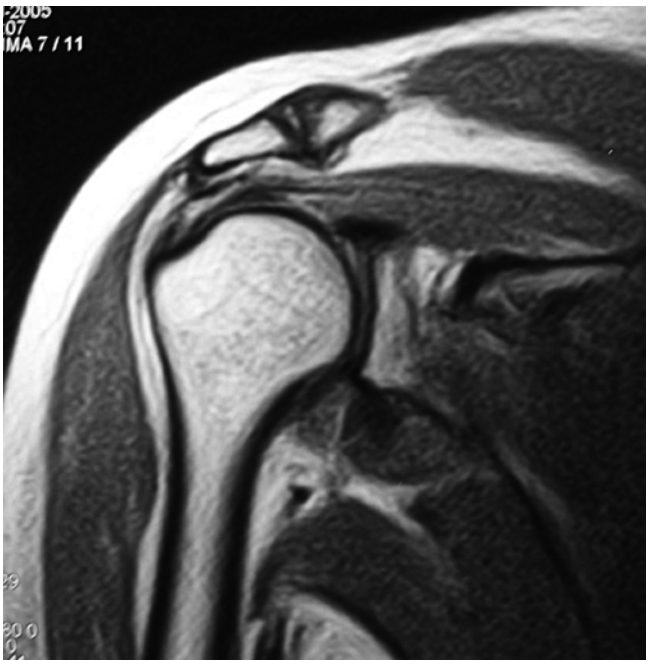


Fig. 22.2 MR – partial-thickness cuff tear on the bursal side

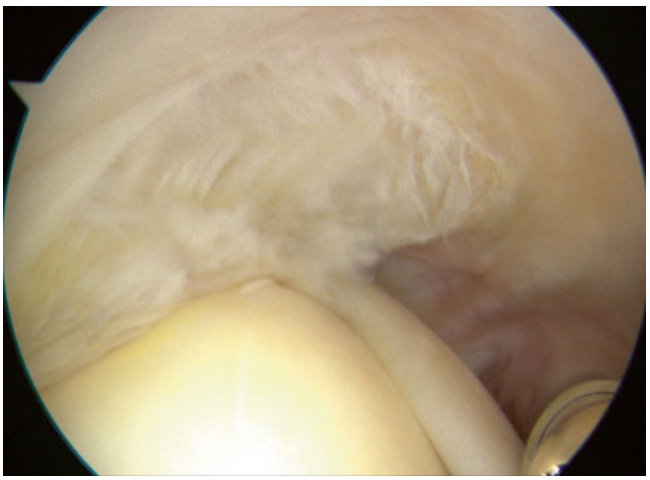


Fig. 22.3 Arthroscopy – partial-thickness cuff tear on the articular side (Ellman grade 1)

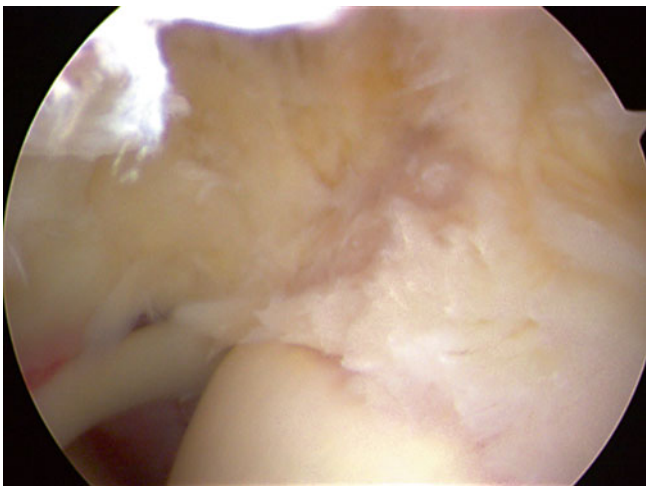


Fig. 22.4 Arthroscopy – partial-thickness cuff tear on the articular side (Ellman grade 3)

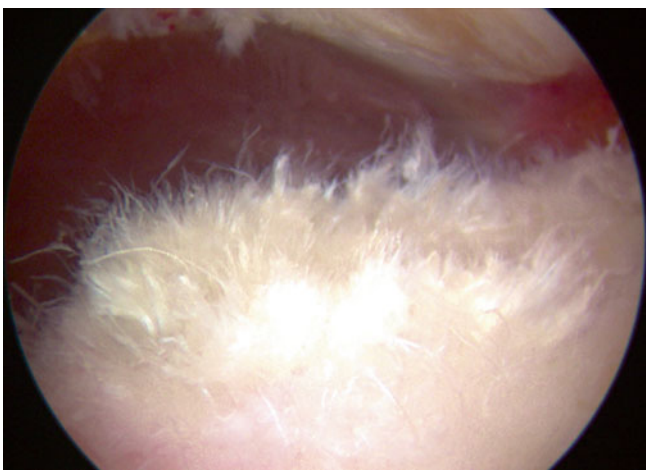


Fig. 22.5 Arthroscopy – partial-thickness cuff tear on the bursal side

22.4 Treatment Strategy

A complete diagnostic evaluation of the glenohumeral joint is necessary before choosing for a nonoperative or an operative treatment. In particular, in the overhead athletes, lesions of the superior labrum and of the capsulolabral structures should not be underestimated [20].

It's also important to understand that the needed outcome of operative treatment is different between a professional and a recreational athlete: high-level athletes have a strong inferior prognosis to come back at the pre-injury level in opposite with middle-age amateur athletes, which normally are satisfied [21].

22.4.1 Nonoperative Management

Generic guidelines exist, but nonoperative treatment must be specific for each patient, considering the grade of his or her pathology and his or her individual sport-specific demands. This treatment is divided in four main phases [1]:

Phase 1: it consists in rest from sport and irritating activities, in association with anti-inflammatory strategies such as phonophoresis, iontophoresis, ice massage, or ice pack application. Passive or active-assisted range-of-motion exercises are started in pain-free ranges (in these range-of-motion exercises, collagen tissue tension stimulates collagen healing). Rotational exercise is performed at 45° scapular plane abduction (below the impingement zone) and then increased with the symptom improvement. Pain-free submaximal exercise can be started. Strengthening can be started with scapulo-thoracic muscles and then with glenohumeral dynamic stabilizers. Aerobic activity as bicycling should continue.

Phase 2: it starts when pain and inflammation are resolved. Strengthening can increase and approach the end range, with particular attention to the stretching of the posterior capsule. An elastic band can be used to help to strengthen every single muscle. Movements that cause pain are avoided. Strengthening of the scapulo-thoracic system should continue, as aerobic activity. When the athlete has achieved a good scapular and glenohumeral system, sport-specific movements can start.

Phase 3: normally, the athlete should have a pain-free range of motion. Eccentric activity is suggested, especially for posterior cuff and medial scapular muscle. The strengthening program can be progressed to full range and maximal resistance. Isokinetics exercise can be started for glenohumeral rotators, elevator muscles, and scapular muscles.

Phase 4: the athlete can return gradually to sport activity. Each sport has its specific activity: for example, pitchers perform an interval throwing program, swimmers perform

low-intensity interval training, and tennis players start with forehands practice and then with backhands. The intensity is increased, until reaching pre-injury level, which is the target of this phase.

Outline exercise to preserve strength and motion should continue when the athlete comes back to activity.

22.4.2 Surgical Treatment

In patient with subacromial impingement, intact rotator cuff surgical treatment is indicated after several months of rehabilitation and activity modification failure. Generally, these criteria may be applied also to athletes. The surgeon must be sure that the cause of the shoulder pain is the mechanical supraspinatus outlet narrowing, because glenohumeral instability and labral pathology are more common, especially in shoulder pain of throwing athlete [1].

In partial rotator cuff tear, surgical indication is similar to those with an intact rotator cuff, but if MRI shows a lesion involving more than 50 % of the tendon thickness, nonoperative treatment may be shorted and the surgery proposed before 9 months [22–24]. Bursal-side tears may be more symptomatic then articular-side tears, and in this case surgical approach may be more aggressive. During diagnostic arthroscopy surgeon should check both the depth of the lesion and the quality of remaining tendon: it is necessary to check the rotator cuff insertion from both the glenohumeral joint side and the subacromial bursal side. Diagnostic arthroscopy is followed by arthroscopic acromioplasty if the surgeon finds bony supraspinatus outlet impingement. Debridement is suggested in articular-side partial tears less than 50 % and in bursal-side lesion less than 25 %. In other cases, partial rotator cuff tears are repaired [25].

In complete rotator cuff tears, if a 6- to 12-week trial of nonoperative treatment and activity modification fails, surgical approach is suggested, especially in young throwing athletes to permit a return to competition [1]. In overhead athletes most of lesions are small (1–2 cm or one tendon) and minimally retracted: surgical technique is often mini-open or arthroscopic repair. Instead rotator cuff injury in contact athletes is usually larger than 2 cm or one tendon and retracted: in this case some authors suggest open repair [21]. Our position is that both open and arthroscopic surgical techniques may be practiced, but arthroscopic treatment is preferred for its decreased scarring and minor deltoid morbidity, which may influence the postoperative rehabilitation and return to pre-injury level.

22.5 Rehabilitation and Return to Play

Return to sport activity, possibly at the same level before the injury, is the athletic rehabilitation target.

Rehabilitation after arthroscopic surgery without rotator cuff repair is the same of nonoperative treatment. In case of rotator cuff repair, postoperative rehabilitation is divided into four phases [1].

The goals of phase 1 (0–6 weeks) are patient education, pain and inflammation control, tendon healing, and maintenance of subacromial gliding if an acromioplasty was associated with the cuff repair. Patient is educated to place a support under the arm to obtain abduction and neutral rotation and to repeat active exercise at the elbow, wrist, and hand. To maintain subacromial gliding and capsulo-ligamentous complex, patient should perform immediate passive range-of-motion exercises: normally, exercises must be performed with 20 repetitions, three to six times a day. After about 10–15 days, patients come back to the surgeon to remove sutures and to assess the shoulder. If patient has again pain or excessive tightness, indications are cold therapy or hot therapy (or both), joint mobilization, and passive stretching. If there aren't problems, patient continues with home exercise program. At 4 weeks, active range-of-motion and isometrics exercises should be started (rotation, extension), with an external rotation range from 70 to 90°. If rotator cuff tear was larger than 1 cm, active exercises should be started at 6 weeks.

Second-phase duration is from 6 to 10 weeks after surgery. The goals are a complete range of motion and improvement of neuromuscular control and strength. Patient should continue with the same exercises of phase 1 and add stretching, if not already started, to improve range of motion. Most important is stretching exercises for external rotation in multiple positions (and for posterior capsule). Stretches should be maintained for 10–20 s, for 10–20 times a day. Strengthening continues, with the introduction of forward flexion and abduction. Multiangle exercises should be started at 45° in the scapular plane using manual resistance; depending the patient reaction to resistive exercises, the position of resistance is progressed toward 90° in the scapular plane. If rotator cuff tear was large or massive, return of a complete range of motion is gradual and slow: a continue discussion between surgeon and rehabilitation specialist about any decision of aggressive stretching is important.

From 10 to16 weeks is the length of third phase, whose goals are a complete range of motion, optimization of neuromuscular control, improvement of endurance, and return to functional activity. Normally in this phase, patients are pain-free and active elevation should pass the shoulder (be careful if patient has an abnormal scapulo-humeral rhythm). Strengthening and stretching should continue, especially for elevation. Sport-specific movements should be started. An upper extremity strengthening and flexibility program should be introduced.

In phase 4 (16 weeks to 6 months), athlete comes back gradually to training and then to competition, with the target of pre-injury level. Strengthening, stretching, upper extremity

and flexibility program, sport interval program, and sport-specific exercises should be continued both for training and prevention [1].

References

1. Williams GR Jr, Martin Kelley PT (2000) Management of rotator cuff and impingement Injuries in the athlete. J Athl Train 35(3):300–315
2. Neer CS II (1972) Anterior acromioplasty for the chronic impingement syndrome in the shoulder: a preliminary report. J Bone Joint Surg Am 54:41–50
3. Jobe CM (1995) Posterior superior glenoid impingement: expanded spectrum. Arthroscopy 11:530–536
4. Jobe CM, Coen MJ, Pat Screnar PT (2000) Evaluation of Impingement Syndromes in the overhead-throwing athlete. J Athl Train 35(3):293–299
5. Burkhart SS (2006) Internal impingement of the shoulder. Instr Course Lect 55:29–34
6. Conway JE (2001) Arthroscopic repair of partial-thickness rotator cuff tears and SLAP lesions in professional baseball players. Orthop Clin North Am 32:443–456
7. Paley KJ, Jobe FW, Pink MM, Kvitne RS, ElAttrache NS (2000) Arthroscopic findings in the overhand throwing athlete: evidence for posterior internal impingement of the rotator cuff. Arthroscopy 16:35–40
8. Siskosky MJ, ElAttrache NS (2007) Management of internal impingement and partial rotator cuff tears in the throwing athlete. Oper Tech Sports Med 15:132–143
9. Matava MJ, Purcell DB, Rudzki JR (2005) Partial-thickness rotator cuff tears. Am J Sports Med 33(9):1405–1417
10. Millstein ES, Snyder SJ (2003) Arthroscopic management of partial, full thickness, and complex rotator cuff tears: indications, techniques, and complications. Arthroscopy 19(Suppl 1):189–199
11. Baker CL et al (2000) Clinical evaluation of the athlete's shoulder. J Athl Train 35(3):256–260
12. Gerber C, Krushell RJ (1991) Isolated rupture of the tendon of the subscapularis muscle: clinical features in 16 cases. J Bone Joint Surg Br 83:389–394
13. Johannes RH, Barth MD et al (2006) The bear-hug test: a new and sensitive test for diagnosing a subscapularis tear. Arthroscopy 22(10):1076–1084
14. Neer CS II (1990) Shoulder reconstruction. WB Saunders, Philadelphia
15. Hawkins RJ, Bokor DJ (1998) Clinical evaluation of shoulder problems. In: Rockwood CA Jr, Matsen FA III (eds) The shoulder, 2nd edn. WB Saunders, Philadelphia, pp 149–176
16. Yocum LA (1983) Assessing the shoulder. History, physical examination, differential diagnosis, and special tests used. Clin Sports Med 2(2):281–289
17. Balke M et al (2013) Correlation of acromial morphology with impingement syndrome and rotator cuff tears. Acta Orthop 84(2):178–183
18. Goutallier D et al (2003) Influence of cuff muscle fatty degeneration on anatomic and functional outcomes after simple suture of full-thickness tears. J Shoulder Elbow Surg 12(6):550–554
19. Dodson CC, Brockmeier SF, Altchek DW et al (2007) Partial-thickness rotator cuff tears in throwing athletes. Oper Tech Sports Med 15:124–131
20. Stephen F, Brockmeier MD et al (2008) Arthroscopic intratendinous repair of the delaminated partial-thickness rotator cuff tear in overhead athletes. Arthroscopy J Arthroscopic Relat Surg 24(8):961–965
21. Dennis Liem MD et al (2008) Arthroscopic rotator cuff repair in overhead-throwing athletes. Am J Sports Med 36(7):1317–1322
22. Payne LZ, Altchek DW, Craig EV, Warren RF (1997) Arthroscopic treatment of partial rotator cuff tears in young athletes: a preliminary report. Am J Sports Med 25:299–305
23. Ellman H (1990) Diagnosis and treatment of incomplete rotator cuff tears. Clin Orthop 254:64–74
24. Gartsman GM, Milne JC (1995) Articular surface partial-thickness rotator cuff tears. J Shoulder Elbow Surg 4:409–415
25. Andrews JR, Broussard TS, Carson WG (1985) Arthroscopy of the shoulder in the management of partial tears of the rotator cuff: a preliminary report. Arthroscopy 1:117–122

Treatment of Acute Traumatic Acromioclavicular Dislocations

23

Ettore Taverna, Vincenzo Guarrella, and Riccardo D'Ambrosi

23.1 Etiology

Injuries to the acromioclavicular joint (ACJ) account for approximately 9–12 % of those to the shoulder girdle seen in clinical practice [1]. This is likely to be an underestimate of their true prevalence, since patients with minor sprains may not seek medical attention. Overall, AC dislocations represent 12 % of all dislocations of the shoulder girdle and 8 % of all joint dislocations in the body [2, 3, 7–9]. Most patients were younger than 35 years with a gender distribution of 8:1 in favor of men [3–9]. Sports activities are a common cause of ACJ injuries, especially alpine skiing, ice hockey, football, and rugby (32–40 % of shoulder injuries) [3, 4]. AC joint dislocation is also often diagnosed after road traffic accidents and fall on the side of the body.

23.2 Injury Mechanism

The most frequent injury usually involves a direct blow to the lateral aspect of the shoulder with the arm in an adducted position, leading to downward displacement of the scapula opposed by impaction of the clavicle onto the first rib [5]. The inherent strength and stability of the sternoclavicular joint transfers energy to both the acromioclavicular (AC) and coracoclavicular (CC) ligaments [10] (Fig. 23.1). The force initially injures the acromioclavicular ligaments. The CC ligament is one of the strongest ligaments in the body. As the force perpetuates, further energy is transmitted to the coracoclavicular ligaments, resulting in greater displacement of the clavicle with reference to the acromion. The acromioclavicular capsule ligamentous structures are first to fail with consecutive loading of the CC ligaments. Complete ACJ dislocations are defined as combined AC and CC ligament disruption leaving the deltoid and trapezoid muscle attachments as last restraints against the applied forces. A major injury will lead to further transmission of force and disruption of the deltoid and trapezius muscles, as the lateral end of the clavicle herniates through it [11], leading to high-degree ACJ instabilities.

Indirect mechanisms of ACJ injuries are rare. A fall on to the adducted arm leads to a pushing force of the humeral head upward against the inferior aspect of the acromion. The resulting forces create a wide spectrum of ACJ injuries including inferior displacement of the clavicle beneath the coracoid process (type VI dislocation according to the classification of Rockwood [12]).

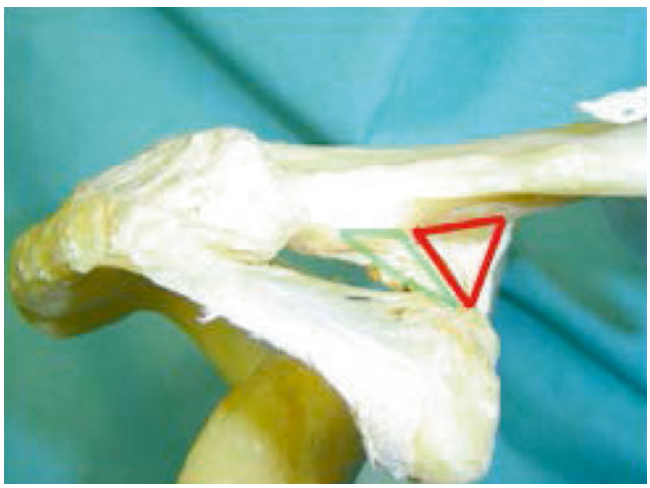


Fig. 23.1 Coracoclavicular ligaments: conoid ligament – located medial, coned or *triangular in shape*. Runs from the posterior medial aspect of the coracoid process to the posterior conoid tubercle in the clavicle. Responsible for restraining superior–inferior displacements. Trapezoid ligament – located lateral, *quadrilateral in shape*. Runs from the coracoid process shaft oblique and superior–lateral to the anterior–lateral clavicle trapezoid ridge. Responsible for resisting compressive forces and lateral displacement of the clavicle

E. Taverna • V. Guarrella • R. D'Ambrosi (✉)
U.O. Chirurgia della Spalla II, Istituto Ortopedico Galeazzi, Via R. Galeazzi 4, Milan 20161, Italy
e-mail: taverna@shoulder.it; vguarrella@hotmail.com; riccardo.dambrosi@hotmail.it

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_23,

23.3 Clinical and Diagnostic Examination

A detailed history including the mechanism of injury, location and duration of pain, and associated symptoms is vital to diagnosing an AC joint injury.

An accurate clinical evaluation may be difficult and painful in the acute setting. It is important to examine the patient in a sitting or standing position, allowing the weight of the injured arm to exaggerate any deformity. Pain may be variable in nature, given the AC joints' dual innervation from the suprascapular nerve and the lateral pectoral nerve [13]; however, the diagnosis is often clinched with a visible or palpable defect noted at the AC joint. Tenderness directly at the ACJ is the main symptom with visually evident step formation between the acromion and the distal clavicle end in complete ACJ dislocations. A comparison to the unaffected contralateral ACJ should be drawn due to a sometimes physiological prominent distal clavicle end on both shoulders. A key part of the clinical assessment represents the testing of horizontal instability. Hereby, the distal clavicle is shifted posteriorly with the acromion fixed by the other hand. An increased posterior translation in comparison to the unaffected side indicates a horizontal component of ACJ instability. Discomfort is often exacerbated with range of motion of the shoulder and with loading of the joint with the crossarm adduction test, which is performed by forward elevating the arm to 90° with arm adduction. Assessment of the AC joint for stability after an acute injury may be difficult secondary to guarding; however, for subacute and chronic injuries, this should be attempted. The Paxinos test (thumb pressure at the posterior AC joint) combined with a positive bone scan has been found to predict AC joint pathology with a high degree of confidence. Pain localized to the acromioclavicular joint or "on top" is diagnostic of acromioclavicular joint abnormality, whereas pain or painful clicking described as "inside" the shoulder is considered indicative of labral abnormality. However, the sensitivity of this test for AC pathology is only 41 %, with a specificity of 94 % [14]. A simple shoulder shrug may be helpful in determining if the deltotrapezial fascia has been separated from the clavicle [5]. Reduction of the AC joint can also be tested by stabilizing the clavicle in one hand and with the other hand placing an upward force on the ipsilateral elbow and assessing the joint for visible or palpable incongruity. Additionally, a thorough neurovascular assessment of the upper extremity including the cervical spine should be performed. Suspicion for other associated injuries, such as clavicle, coracoid, and rib fractures, should be raised with higher injury mechanisms [15].

They were initially graded I through III based on radiographic displacement and the degree of ligamentous damage [16, 17]. Rockwood later added types IV through VI to the classification system. The rising type correlates with greater displacement and higher levels of ligamentous injury [5].

Type I: This typically low-energy injury involves a sprain to the AC ligaments only. The CC ligaments are spared by the absorption of the impact by the AC ligaments. With the AC and CC ligaments intact, radiographic imaging appears normal.

Type II: As the energy imparted to the shoulder is increased, the AC joint capsule and ligaments are ruptured, and the distal clavicle is thereby rendered unstable in the horizontal plane. The CC ligaments remain intact, and there may be slight elevation of the clavicle on radiographs; however, the displacement is less than 100 % of the diameter of the distal clavicle, and the radiographic CC distance is increased by less than 20 %.

Type III: This higher-energy injury represents a complete disruption of both the AC and CC ligaments, which leads to complete dislocation of the AC joint. The insertion of the deltotrapezial fascia remains intact. Radiographs demonstrate displacement of the clavicle greater than 100 % of the diameter of the distal clavicle, and the radiographic CC distance is increased by 20–100 % [5].

Type IV: This injury involves a complete rupture of the AC and CC ligaments with posterior displacement of the distal clavicle into the trapezius fascia. It is important in this setting to evaluate the SC joint as concomitant anterior dislocation can occur.

Type V: This higher-energy variant of a type III injury represents a complete disruption of both the AC and CC ligaments, which leads to complete dislocation of the AC joint. The deltotrapezial fascia is stripped from its attachment to the clavicle. Radiographs demonstrate displacement of the clavicle greater than 300 % of the diameter of the distal clavicle, and the radiographic CC distance is increased by 100–300 %.

Type VI: This rare injury involves inferior displacement of the clavicle either subacromial or subcoracoid behind the conjoined tendon. The mechanism involves severe hyperabduction and external rotation of the arm combined with scapular retraction. It results from high-energy trauma, and neurovascular impairment is often present prior to reduction [15].

Confirmation of AC joint injury involves a complete radiographic shoulder series, which is essential in the analysis and classification of these injuries. Anteroposterior, scapular Y, and axillary views are obtained. These radiographs serve to provide information regarding the nearby glenohumeral joint and can rule out coexisting pathology. As standard anteroposterior (a.p.) projection, the Zanca view [18] (Fig. 23.4) has been established with the x-ray beam tilted for 10° in a caudocranial direction completed by a panorama stress view with a weight of 10 kg hanging on both wrists. Different methods have been employed for weight bearing. Detection of an increased AC and/or CC distance is indicative of ACJ dislocation. The normal ACJ

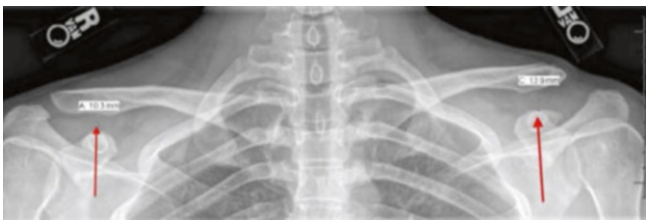


Fig. 23.2 Zanca view is the most accurate view to visualize the AC joint. This view is achieved by tilting the x-ray beam 10–15° cephalad and using one-half of the standard penetrance. Because of the significant variation in AC joint anatomy from one side to another, a bilateral Zanca view is recommended to visualize both AC joints on a single x-ray cassette while maintaining the same orientation of the x-ray beam. By visualizing both AC joints on the same cassette, the CC distance can be compared from side to side, pre- and postoperatively. The two *arrows* show the two acromion-clavicular joints. On the right is possible to observe a healthy clavicle. On the left an acromioclavicular dislocation

width in the frontal plane (Zanca view) measures 1–3 mm and decreases with age. An ACJ width >7 mm in men and 6 mm in women is found to be pathologic (Fig. 23.2). The introduction of Rockwood's classification necessitated a second plane to detect posterior dislocation of the distal clavicle. Routinely, at most radiologic departments, an axillary view with the patient in a sitting, supine, or standing position is performed [19].

Magnetic resonance (MR) imaging represents a sensitive diagnostic tool in evaluation of ACJ disorders. Assessment of the stabilizing soft tissue structures involving the AC ligaments, CC ligaments, and delto-trapezoidal fascia is possible in a reliable manner, and its results can change the clinical grading of dislocation [20, 21].

MR imaging is also useful if surgery is considered to identify accompanying pathologies and again to identify underestimated injuries.

T1-weighted imaging best demonstrates the CC ligaments, and fat-suppressed proton density-weighted or T2-weighted imaging best demonstrates the ligamentous disruption, when surrounded by blood or fluid [20].

In the author's clinical practice, a panorama stress view and axillary dynamic radiological evaluation represent the basic imaging tools, on which a therapeutic decision can be made in almost all cases. MRI is used only in selected cases, where associated glenohumeral soft tissue injuries are assumed.

23.4 Treatment Strategy

The goals of treatment for AC injuries are achieving painless range of motion of the shoulder, obtaining full strength, and exhibiting no limitation in activity. The treatment strategy varies according to the classification of the lesion.

Rockwood Type I

Sprains or partial tears at the ACJ are beyond all doubt treated nonoperatively [3, 5, 7, 17, 22]. Joint stability is maintained and ligament healing will occur in virtually all cases. Conservative therapy in terms of "skilful neglect" seems to be appropriate and sufficient. Occasionally, symptoms may appear between 6 months and 5 years, the 90 % are insignificant, reasonably well tolerated [38] and resolve within 12 months [23].

Rockwood Type II

General treatment recommendations are nonoperative for type II injuries [33, 5, 7, 22–24]. Similar to type I injuries, in most cases, symptoms disappear within 12 months [23]. Reasons for persistent complaints are residual instability, tearing of the intraarticular disk, articular cartilage injuries, residual joint incongruity, osteolysis of the lateral clavicle, and weakness [3, 5]. In case of type II injuries it's possible to observe an increased anteroposterior translation in terms of horizontal stability. This may be a further explanation for persistent symptoms due to mis-/underdiagnosis of the initial injury degree.

Rockwood Type III

Operative treatment of grade III injuries results in a better cosmetic outcome but greater duration of sick leave compared to nonoperative management. No difference regarding strength, pain, throwing ability, and incidence of acromioclavicular joint osteoarthritis has been observed between the treatments. Current treatment recommendations favor surgical treatment in young patients with physically demanding occupations or sporting activities. The current scientific evidence seems to show rather a cosmetic advantage of surgical treatment than a functional one [3, 5, 6].

Rockwood Type IV

There is consensus in the literature that the treatment of type IV injuries should be surgical [3, 5]. The argument to treat even the most inactive patients, was the extremely high pain level, considering only closed reduction as sufficient therapeutic measure [7]. When considering the complete disruption of the AC ligaments and the detachment of the delto-trapezoidal insertion, closed reduction alone is not deemed to be sufficient, requiring surgical stabilization. Surgical treatment should focus on ACJ reduction, AC ligament fixation, and reconstruction of the delto-trapezoidal fascia. Obviously if the CC ligament complex is involved, its pathology has to be addressed as well.

Rockwood Type V

All stabilizing anatomical structures, including the CC and AC ligaments and the delto-trapezoidal fascia, are disrupted. The treatment should be operative [7] with reconstruction of

the stabilizing structures including the delto-trapezoidal fascia. Thus, open procedures may be of advantage as compared to arthroscopic techniques, which usually fail to address adequately reconstruction of the delto-trapezoidal fascia.

Rockwood Type VI

This type of ACJ dislocation is quite rare and has been reported only in case reports [3, 15, 24, 25]. The treatment is always operative with reduction of the distal clavicle end and ACJ stabilization. Closed reduction may be difficult due to entrapment of the distal clavicle posterior to the conjoint tendon.

23.4.1 Treatment Modalities

23.4.1.1 Nonoperative Treatment

A common pitfall of these treatment attempts was skin and soft tissue related. Skin breakdown may be a potential complication of using external immobilization straps that apply continuous pressure over the lateral clavicle. A further prerequisite for successful nonoperative treatment is a maximum of compliance of the patient which often lacks in young and active patients.

Recognizing the difficulties to maintain sufficient reduction of the ACJ, several authors have recommended only the use of a simple shoulder sling [6]. In the author's opinion, nonoperative therapy in terms of "skillful neglect" represents a sufficient therapy consisting in immobilization of the respective arm until subsidence of the acute pain, ice application, analgesics according to the patient's needs and accompanying physical therapy to gain free range of motion and full muscle strength. A key pillar of physical rehabilitation programs represents the strengthening of the spino-scapulohumeral function chain. The main focus should be kept on the periscapular muscles, including the rhomboidei, levator scapulae, trapezius, and latissimus dorsi muscles to stabilize the scapula actively due to the lack of passive ligamentous suspension to the clavicle. This musculoskeletal pathology explains the resulting scapular dyskinesis in many patients suffering from chronic ACJ instability. Thus, nonoperative treatment should be symptomatic in the acute phase and functional in the subacute/chronic phase. Usually, freedom from pain and free range of motion should be present 3–4 weeks after the injury.

23.4.1.2 Surgical Treatment

Open Techniques

Bosworth Screw

For several decades, it represented an established method to treat acute ACJ dislocations. Until today, some orthopedic and trauma surgeons use this simple technique, which can be performed percutaneously and grants good to excellent long-term outcomes [3, 5, 6]. Possible malpositioning, screw breakage, damage of the CC repair, and necessity of screw removal represent disadvantages rendering this implant unpopular nowadays [26].

Hook Plate

It is still a widely used implant providing high primary stability but requiring a second surgery for implant removal.

Overall [27, 28], the hook plate provides high rates of successful functional restoration offering a high primary stability. Possible complications (loss of reduction, redislocation, and acromion osteolysis or fracture) have to be taken into consideration, as well as the need for plate removal after 3 months.

Pinning and Tension Banding

Percutaneous pinning and tension banding using two AC transarticular K-wires and a cerclage represent simple and cost-effective procedures [29, 30]. Good results can be achieved in 96 % of cases and reduction can be retained in 80 %. Only 5 % complained intermittent pain with an average visual analog score of 4 at follow-up. The overall complication rate is 15 % including K-wire migration in 4 % and ACJ redislocation in 11 %.

PDS Sling

It is not a universally accepted technique because it requires a large exposure resulting in soft tissue damage and a high rate of redislocation reported by a few authors [31, 32].

Extra-anatomical Procedures

Transfer of the coracoacromial ligament from the acromion into the resected distal clavicle was suggested already in 1917 by Cadenet and reported in 1972 by Weaver and Dunn [33] for both acute and chronic cases. However, the coracoacromial ligament transfer should not be indicated for acute cases when the CC and AC ligaments are likely to heal spontaneously after repair. For chronic cases, biomechanical [34, 35] and clinical [3, 5] data proved anatomic CC ligament reconstruction using autologous semitendinosus tendon to be superior to the Weaver–Dunn procedure.

Arthroscopic Techniques

Following the development of arthroscopic techniques in joint surgery, suitable implants have been searched allowing for a minimally invasive, arthroscopically assisted procedure. In 2001, Wolf and Pennington described for the first time the arthroscopic CC stabilization using polyethylene wire cerclages reporting on 81 % good and excellent results. Rolla et al. [36] described the arthroscopically assisted use of a cannulated Bosworth screw with excellent early results. Elser et al. used suture anchors for arthroscopic CC stabiliza-

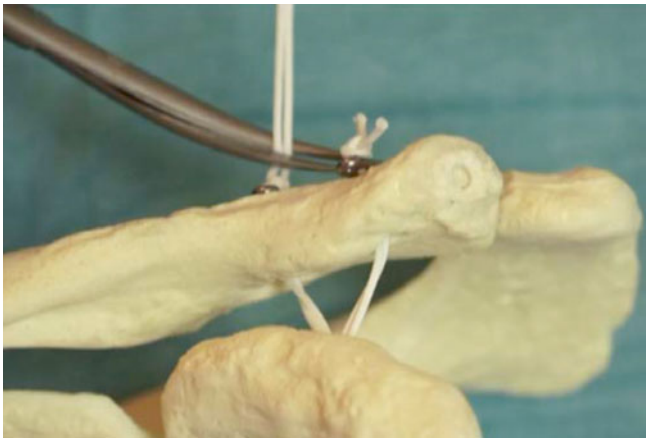


Fig. 23.3 Reduction after dislocation of the clavicle with a TightRope™ technique

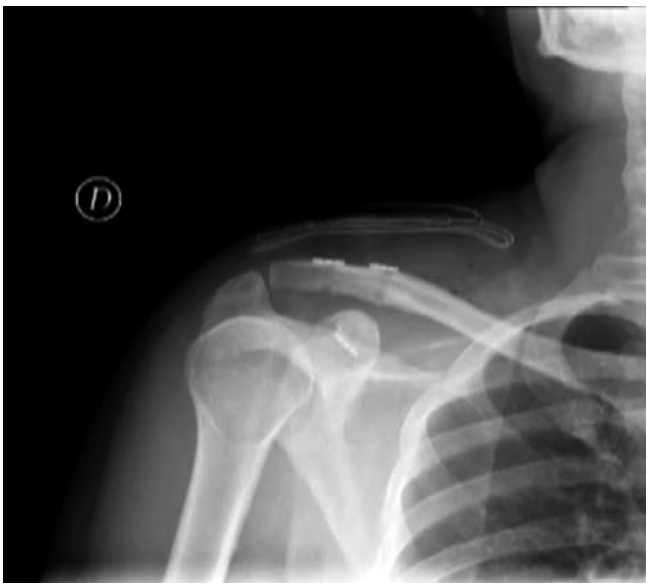


Fig. 23.4 Rx: anteroposterior view after TightRope™ reconstruction (right acromioclavicular joint)

tion without image intensifier with good results. The TightRope™ system (Arthrex, Naples, FL, USA) or similar can be used to repair ACJ with excellent results [37]. Arthroscopic techniques allow for contemporaneous treatment of the associated intraarticular lesion and ACJ stabilization (Figs. 23.3 and 23.4).

The principle of the TightRope™ stabilization can be performed in a mini-open technique as well.

23.5 Rehabilitation and Return to Play

The role of the postoperative treatment for ACJ reconstruction is very important. In contrast to ligamentous injuries of other joints, the gravity creates a continuous stress to the CC and AC ligaments preventing ligament stump contact and healing. Therefore, exceptional protection of the ACJ repair has to be guaranteed in the immediate postoperative period which contributes significantly to the success of the surgery and minimizes the risk for redislocation. In the literature, a broad spectrum regarding postoperative rehabilitation protocols has been reported ranging from early unrestricted rehabilitation over early active mobilization to 90° after 2–3 days to only passive motion up to 90° twice a week for 4 weeks with immobilization using a sling. Care should be taken in regard to the model of sling immobilization. It is of crucial importance to provide a sufficient support to the forearm and elbow to neutralize CC gravity distraction forces. In addition, a high compliance of the patient is required to follow the physician's advices of strictly limited postoperative activity.

The postoperative protocol typically involves the use of a sling or shoulder immobilizer for a period of 4–6 weeks to allow the reconstruction to heal. This provides support for the involved upper extremity when in an upright position [38]. Limited supine passive and active assisted range of motion is initiated as early as 7–10 days postoperatively, while strengthening and upright range of motion is typically restricted until 6 weeks.

Shoulder must be immobilized, and no range of motion is allowed for 4–6 weeks, necessary time for biological healing. The sling or shoulder immobilizer can be removed at this time with active range of motion and strengthening of the scapular stabilizers encouraged. Shoulder range of motion is initially limited to 90° of forward elevation, 90° of abduction, 30° of external rotation, and internal rotation to the chest wall. Weight training is initiated at 8–12 weeks, followed by return to noncontact athletic activity at 3–6 months. Peak strength is often obtained by 9 months, whereby patients can return to contact activities. It has been suggested that use of various types of tendon grafts may allow a more accelerated rehabilitation program [39].

References

1. Emery R (1997) Acromioclavicular and sternoclavicular joints. In: Copeland S (ed) Shoulder surgery. WB Saunders, London
2. Riand N, Sadowski C, Hoffmeyer P (1999) Acute acromioclavicular dislocations. Acta Orthop Belg 65(4):393–403
3. Tauber M (2013) Management of acute acromioclavicular joint dislocations: current concepts. Arch Orthop Trauma Surg 133: 985–995
4. Webb J, Bannister G (1992) Acromioclavicular disruption in first class rugby players. Br J Sports Med 26(4):247–248
5. Epstein D, Day M, Rokito A (2012) Current concepts in the surgical management of acromioclavicular joint injuries. Bull NYU Hosp Jt Dis 70(1):11–24
6. Babhulkar A, Pawaskar A (2014) Acromioclavicular joint dislocations. Curr Rev Musculoskelet Med 7:33–39

7. Collins DN (2009) Disorders of the acromioclavicular joint. In: Rockwood CA, Matsen FA, Wirth MA, Lippitt SA (eds) The shoulder, 4th edn. Saunders, Philadelphia
8. Flik K, Lyman S, Marx RG (2005) American collegiate men's ice hockey: an analysis of injuries. Am J Sports Med 33(2):183–187
9. Headey J, Brooks JH, Kemp SP (2007) The epidemiology of shoulder injuries in English professional rugby union. Am J Sports Med 35(9):1537–1543
10. Mazzocca AD, Sellards R, Romeo A (2002) Acromioclavicular joint injuries: pediatric and adult. In: DeLee JC, Drez D (eds) Orthopedic sports medicine. WB Saunders, Philadelphia
11. Lemos MJ (1998) The evaluation and treatment of the injured acromioclavicular joint in athletes. Am J Sports Med 1:137–144
12. Rockwood CA (1984) Injuries to the acromioclavicular joint. In: Rockwood CA, Green DP (eds) Fractures in adults, 2nd edn. JB Lippincott, Philadelphia, pp 860–910
13. Gerber C, Galantay RV, Hersche O (1998) The pattern of pain produced by irritation of the acromioclavicular joint and the subacromial space. J Shoulder Elbow Surg 7(4):352–355
14. O'Brien SJ, Pagnani MJ, Fealy S et al (1998) The active compression test: a new and effective test for diagnosing labral tears and acromioclavicular joint abnormality. Am J Sports Med 26(5):610–613
15. Gerber C, Rockwood CA Jr (1987) Subcoracoid dislocation of the lateral end of the clavicle. A report of three cases. J Bone Joint Surg Am 69(6):924–927
16. Tossy JD, Mead NC, Sigmond HM (1963) Acromioclavicular separations: useful and practical classification for treatment. Clin Orthop Relat Res 28:111–119
17. Allman FL Jr (1967) Fractures and ligamentous injuries of the clavicle and its articulation. J Bone Joint Surg Am 49(4):774–784
18. Zanca P (1971) Shoulder pain: involvement of the acromioclavicular joint. (Analysis of 1,000 cases). Am J Roentgenol Radium Ther Nucl Med 112(3):493–506
19. Tauber M, Koller H, Hitzl W, Resch H (2010) Dynamic radiologic evaluation of horizontal instability in acute acromioclavicular joint dislocations. Am J Sports Med 38(6):1188–1195
20. Antonio GE, Cho JH, Chung CB, Trudell DJ, Resnick D (2003) Pictorial essay. MR imaging appearance and classification of acromioclavicular joint injury. AJR Am J Roentgenol 180(4): 1103–1110
21. Barnes CJ, Higgins LD, Major NM, Basamania CJ (2004) Magnetic resonance imaging of the coracoclavicular ligaments: its role in defining pathoanatomy at the acromioclavicular joint. J Surg Orthop Adv 13(2):69–75
22. Mouhsine E, Garofalo R, Crevoisier X, Farron A (2003) Grade I and II acromioclavicular dislocations: results of conservative treatment. J Shoulder Elbow Surg 12(6):599–602
23. Shaw MB, McInerney JJ, Dias JJ, Evans PA (2003) Acromioclavicular joint sprains: the post-injury recovery interval. Injury 34(6): 438–442
24. Beitzel K et al (2013) Current concepts in the treatment of acromioclavicular joint dislocations. Arthroscopy 29(2):387–397
25. Torrens C, Mestre C, Perez P, Marin M (1998) Subcoracoid dislocation of the distal end of the clavicle. A case report. Clin Orthop Relat Res 348:121–123
26. Assaghir YM (2011) Outcome of exact anatomic repair and coracoclavicular cortical lag screw in acute acromioclavicular dislocations. J Trauma 71(3):E50–E54
27. Gstettner C, Tauber M, Hitzl W, Resch H (2008) Rockwood type III acromioclavicular dislocation: surgical versus conservative treatment. J Shoulder Elbow Surg 17(2):220–225
28. Salem KH, Schmelz A (2009) Treatment of Tossy III acromioclavicular joint injuries using hook plates and ligament suture. J Orthop Trauma 23(8):565–569
29. Modi CS, Beazley J, Zywiel MG, Lawrence TM, Veillette CJ (2013) Controversies relating to the management of acromioclavicular joint dislocations. Bone Joint J 95-B(12):1595–1602
30. Kazda S, Pasa L, Pokorny V (2011) Clinical outcomes of surgical management of acromioclavicular dislocation with and without ligament suturing. Rozhl Chir 90(10):561–564
31. Eschler A, Gradl G, Gierer P, Mittlmeier T, Beck M (2012) Hook plate fixation for acromioclavicular joint separations restores coracoclavicular distance more accurately than PDS augmentation, however presents with a high rate of acromial osteolysis. Arch Orthop Trauma Surg 132(1):33–39
32. Greiner S, Braunsdorf J, Perka C, Herrmann S, Scheffler S (2009) Mid to long-term results of open acromioclavicular-joint reconstruction using polydioxansulfate cerclage augmentation. Arch Orthop Trauma Surg 129(6):735–740
33. Weaver JK, Dunn HK (1972) Treatment of acromioclavicular injuries, especially complete acromioclavicular separation. J Bone Joint Surg Am 54(6):1187–1194
34. Mazzocca AD, Santangelo SA, Johnson ST, Rios CG, Dumonski ML, Arciero RA (2006) A biomechanical evaluation of an anatomical coracoclavicular ligament reconstruction. Am J Sports Med 34(2):236–246
35. Costic RS, Labriola JE, Rodosky MW, Debski RE (2004) Biomechanical rationale for development of anatomical reconstructions of coracoclavicular ligaments after complete acromioclavicular joint dislocations. Am J Sports Med 32(8):1929–1936
36. Rolla PR, Surace MF, Murena L (2004) Arthroscopic treatment of acute acromioclavicular joint dislocation. Arthroscopy 20(6):662–668
37. Scheibel M, Droschel S, Gerhardt C, Kraus N (2011) Arthroscopically assisted stabilization of acute high-grade acromioclavicular joint separations. Am J Sports Med 39(7):1507–1516
38. White B, Epstein D, Sanders S, Rokito A (2008) Acute acromioclavicular injuries in adults. Orthopedics 31(12)
39. Lee SJ, Nicholas SJ, Akizuki KH et al (2003) Reconstruction of the coracoclavicular ligaments with tendon grafts: a comparative biomechanical study. Am J Sports Med 31(5):648–655

Return to Sport After Shoulder Arthroscopy

24

Roberto Vianello, Antonello Pannone, Marco Conca, and Roberto Corezzola

Shoulder arthroscopy is one of the most common procedures in orthopedic surgery especially for the treatment of a large number of shoulder joint pathologies in the field of sport.

We don't forget how arthroscopy of the shoulder is not always accompanied by the fastest recovery times without any complications [1]. This point is very important when we are speaking about athletes.

Return to sport after shoulder arthroscopy therefore is not so quick and depends on many factors: type of pathology, type of tissue, type of sport (contact, not contact, overhead, etc.), or fitness of the single athlete. But many other factors play a very important role to return to sport after shoulder arthroscopic surgery and these are more relevant if the athlete is a high-level competitor. We are speaking about sponsors, media, managers, team, and supporters. We also can't forget the psychological aspect of athletes and his fear for a new injury: sometimes, the athlete appears to be ready to return to sport at the judgment of the surgeon, physiotherapist, and trainer too but he often says "I am not ok" because he doesn't feel himself ready 100 %. The "time of return to sport" scenario so may vary greatly from few months or even more than a year.

The type and the site of lesion, the time of surgery, and the biological time of repair are all key points.

The most common shoulder pathologies in the athletes are the instability, the minor instability, the SLAP lesion, and more rarely the rotator cuff tears.

R. Vianello, MD (✉) • R. Corezzola
Casa di Cura "Giovanni XXIII", Monastier (TV), Italy
e-mail: robyvianello1@gmail.com

A. Pannone
Casa di Cura "Città di Udine, Udine, Italy

M. Conca
Clinica "San Carlo", Paderno Dugnano (MI), Italy

24.1 Instability

24.1.1 Anatomy of the Shoulder

The shoulder is relatively unconstrained, allowing an extreme range of motion. Joint stability is provided by both static and dynamic elements [2–7], which allows the joint to maintain a large degree of freedom while remaining concentric. The bony anatomy of the glenoid is such that it only covers approximately 25–30 % of the humeral head. Additionally, the bony surface of the glenoid is almost flat and its depth has been found to average only 2.5 mm in transverse plane and 9.0 mm in the caudal–cranial plane.

24.1.2 Dislocation Mechanism/Incidence

Dislocation of glenohumeral joint occurs during all types of athletic endeavors. Anterior dislocations account for approximately 95 % of all shoulder dislocations.

In a series of 57 patients, Baker et al. found that 76 % of the anterior dislocations occurred in an athletic activity [8]. Posterior dislocations are uncommon and an incidence of 4 % of all dislocations has been reported [9]. Posterior subluxation can frequently occur, however, in athletic events. Even rarer is "luxatio erecta" or inferior dislocation. The last variation of shoulder instability is voluntary instability. We don't forget another type of instability that is very common in the sport that is minor instability. Overhead athletes who develop pain and inflammation in their shoulders without overt instability episodes have been postulated to have increased laxity or occult instability. This theory was first promulgated by F. Jobe [10, 11]. Due to repetitive nature of the sport, the AIGH ligament stretches over time due to minor trauma. This creates increased laxity of the shoulder with subsequent increased contact of the humeral head to the acromion or to the posterior-superior glenoid. This can determine a partial thickness rotator cuff tears, superior labrum pathology, or SLAP lesion. When the contact leads to

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_24, © Springer International Publishing Switzerland 2016

posterior-superior labrum tears, this is called "internal impingement" by Walch et al. [12] and by others [13]. It is important to distinguish this type of instability from traumatic instability, because the recovery and return to sport are different for these groups.

24.1.3 Surgery

If a decision for operative management is made in the athletes with shoulder instability, we must decide whether an arthroscopic or open stabilization procedure will be performed. Traditionally, open stabilization of the shoulder has provided excellent overall results in patients with recurrent anterior instability. In the last years, arthroscopic stabilization has produced better results than previously experienced and very similar to open stabilization.

Contraindications for arthroscopic stabilization in the athletes include HAGL lesions, large glenoid defect (>20 %), large Hill-Sachs lesions (>30–40 %), and poor quality of the capsular tissue. As techniques and surgeons' skills have improved, arthroscopic stabilization in the contact sports has become acceptable. O'Neill [14] prospectively studied arthroscopic anterior stabilization of first-time dislocation in a group of 41 athletes, including 17 football players. He demonstrated a 98 % return to preoperative sport; 95 % had no additional dislocation and 90 % had good to excellent results. Mair et al. [15] reported on their experience of arthroscopic posterior stabilization of posterior labral detachments in contact athletes. All 9 athletes were able to make a successful return to their sport, football and lacrosse.

24.1.4 Return to Sport After Arthroscopic Surgery for Instability

It is clear that there is a large amount of anatomical pathology encompassed in shoulder instability and dislocations. As can be expected, there are a myriad of protocols and guidelines that have been recommended after these various shoulder surgeries to allow a timely return by the athlete to sport. Despite the differences in surgical techniques, there are some general principles to consider in the rehabilitation process. Many of the same criteria evaluated for return to sports after a nonoperative treatment apply to athletes trying to return to their sport after surgical stabilization. We suggest also an MR imaging 6 months post-op to verify any repaired instability (Fig. 24.1).

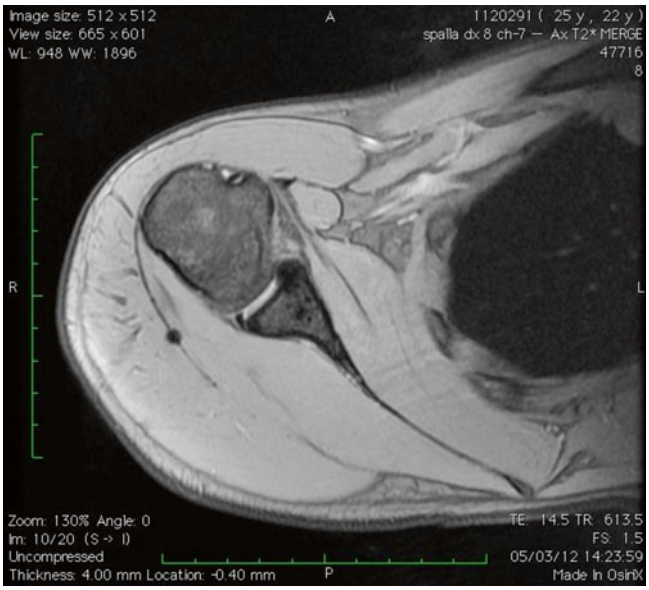


Fig. 24.1 MR of an instability repair after 6 months

Ideal Criteria for Return to Play

- No pain
- Patient subjectivity
- Normal ROM
- Near normal strength
- Normal functional ability
- Normal sport-specific skills

Whether the stabilization is performed by arthroscopy (or open), the athlete likely will need a minimum 14–16 weeks before being ready to return. Typically, the time range for return is from 4 to 6 months and it may take as long 9–12 months: we must distinguish that return to sport is sometimes different than return to competition or game.

Protocols vary for sling use after surgical stabilization, but most use a time frame of about 6 weeks. During this time, the athletes begin on range of motion and some strengthening exercises; again, this is dependent on surgeon preference. As soon as the athlete is out of the sling, more aggressive rehabilitation begins, and neuromuscular training is instituted.

Kim et al. [16] randomized 66 patients who had received arthroscopic Bankart repair into two different therapy protocols. Group 1 had pillow sling immobilization for 3 weeks, followed by pendulum and forward elevation using a pulley. At 4 weeks, external rotation strengthening started, but ER was prohibited. At 6 weeks, ER strength started. At 9 weeks, they allowed more vigorous exercise. Group 2 had sling in sleep only for 2 weeks. Forward elevation was limited to 90° for 2 weeks, and at 4 weeks, full range of motion was allowed but extreme external rotation should be avoided. There was no recurrence in either group but two patients in each group had positive apprehension. This article supports the concept that immobilization need not be extended in many cases after arthroscopic stabilization.

The rehabilitation process must be individualized, because each patient will progress at different level due to quality of

tissue health, speed of healing response, adequacy of fixation, number of dislocations, bone defect if present, and type of procedure performed.

It is very important to analyze the patient, his history and motivations, his psychology, and if he plays a professional sport.

Generally, the postoperative rehabilitation can be divided into four phases (see Table 24.1, from 1 to 3).

From the fourth to the sixth month (shoulder sport return rehabilitation phase), the full range of motion including stressless ER without loading is allowed. Return to play exercises for the integration of sensorimotor system (proprioception, kinesthesia) and strengthening of synergetic force couple are performed for the functional sport. From the sixth month, stressful exercises for the anterior-inferior capsular labrum complex during rehabilitation and practice are performed for return to play transition. After an adequate return to play transition, the resumption of previous shoulder-dependent sport is allowed (shoulder sport phase).

Ultimately, a careful clinical exam of the patient by the physician is essential before giving clearance for return to sport. It is crucial for the staff to determine how the athlete has achieved appropriate isokinetic strength as well as how he learned any mental skills.

The importance of appropriate rehabilitation cannot be overemphasized.

As the athlete progresses in rehabilitation, goals for return to play are full pain-free functional range of motion, normal strength and endurance, and no apprehension. Once an athlete demonstrates that he is physically and mentally ready, the return may take place.

Murray and Mc Birnie [17] report that surgery achieved successful stabilization in 88 % of patients but only 51 % returned to full sport activity. Patients aged 30 years or under were twice as likely to return to their pre-injury level of sport than those aged over 30 years. Half of those patients who felt unable to return to sport had no subjective symptoms of instability or other physical symptoms related to their shoulder but were inhibited by fear of further injury. In order to increase the number of athletes returning to sport following primary arthroscopic stabilization of the shoulder, those patients with no restrictive symptoms should be encouraged to return to full activity.

Solomon and Provencher [18] report that after instability surgery, the results are generally excellent with appropriate rehabilitation gated toward functional- and sport-specific activities accomplished prior to return. The range for this therapy is about 3–20 months, with most able to return by 6–8 months depending on several factors, such as sport or position played and specific shoulder structures injured and repaired. Often, it can be difficult to define "safe to return" and maximal medical improvement. The decision process for how best to treat an athlete after shoulder dislocation must include a discussion of limitations, anticipated activities, and goals with athlete, parents, trainer, therapist, coach, and surgeon, sometimes also with the manager.

24.2 Multidirectional Shoulder Instability

Historically, open capsular shift techniques have been the standard in the operative treatment of patients with MDI. More recently, however, a number of arthroscopic techniques have been described for treatment of MDI. These techniques include thermal or radiofrequency capsular repair/shrinkage, as well as other arthroscopic approaches using suture techniques and/or suture anchors and the rotator interval closure.

The postoperative rehabilitation and return to sport in MDI after arthroscopic stabilization depend on many factors: first, quality of the tissue and capsula and, second, the type of sport.

Immediately after surgery, the rehabilitation protocol employed the use of an ultra sling brace (DonJoy, Carlsbad, California) that immobilized the shoulder in approximately 30° of abduction and protect the shoulder joint. On the first postoperative day, the patient begins active wrist and elbow flexion and extension exercises as well as gentle pendulums and passive scaption exercises. Patients are immobilized for 4–6 weeks depending on the amount of postoperative stiffness seen at follow-up. After sling immobilization is discontinued, gentle passive ROM exercises are advanced and active assisted ROM exercises are initiated. Range of motion is progressed to full passive and active ROM by 2–3 months postoperatively. At this time, capsular stretching exercises are started and isotonic strengthening continued, with emphasis on rotator cuff. At 4 months postoperatively, patients are progressed into the functional phase of rehabilitation with plyometrics, more aggressive strengthening, and overhead lifting as tolerated. In general, athletes must have painless ROM, strength, and endurance comparable with that of the contralateral side before return to competition. Depending on the sport, most athletes are allowed to return to competition at or around 6–8 months postoperatively.

24.3 Acute Traumatic Posterior Shoulder Dislocation

First described in 1838 by Sir A. Cooper [19], traumatic posterior dislocation of the shoulder represents an unusual and challenging clinical problem. These injuries account for 2–5 % of all shoulder dislocations [19]. Seizures, high-energy trauma, and electrocution are associated with a much greater risk of posterior dislocation [19–21]; diagnosis is missed or delayed in up to 79 % of patients [20, 21]. Early identification of these dislocations reduces morbidity and facilitates treatment.

Table 24.1 Instability repair rehab program

Rehab protocol for unstable shoulder after reparative operation (page 1)

	GOALS	TUTOR	RANGE OF MOTION	THERAPEUTIC EXCERCISES
Phase 1 (1-4 weeks)	Respect of biological cicatrization times Check pain and inflammation	Yes, according to the surgeon's prescription	Only passive movements not exceeding 90° in bending and extra rotation Avoid abduction movements	Codman's pendulum exercise Active mobilization exercises for wrist and elbow Isometric stabilization exercises for shoulder blade
Phase 2 (5-8 weeks)	Initial stage of complete recovery of the movement Prevention of compensation movements (i.e. lifting shoulder blades)	Removed, at the discretion of the surgeon	Avoid combined abduction and extra rotation	Aided active mobilization in range of motion without pain Strengthening stabilizer muscles of shoulder and thorax Strengthening the rotator cuff at scapular level

Rehab protocol for unstable shoulder after reparative operation (page 2)

	GOALS	TUTOR	RANGE OF MOTION	THERAPEUTIC EXCERCISES
Phase 3 (9-12 weeks)	Complete recovery of articular movements	No	Progressively complete and pain-free	Active exercises and posterior capsule stretches in order to regain complete movement progressively Close kinetic chain proprioception exercises and open kinetic chain strengthening exercises
Phase 4 (13-14 weeks)	Complete recovery of muscular strength Recovery of neuromuscular control	No	Complete	Combined exercises for the neuromuscular control of the trunk and shoulder girdle Selective strengthening of the supraspinatus

Rehab protocol for unstable shoulder after reparative operation (page 3)

	GOALS	TUTOR	RANGE OF MOTION	THERAPEUTIC EXCERCISES
Phase 5 (15-20 weeks)	Complete recovery of sport-specific movements	No	Complete	Power and precision exercises Plyometric exercises Sport-specific exercises

The treatment can be open (McLaughlin procedure, modified McLaughlin procedure, anterior approach and bone grafting, posterior open Bankart procedure, arthroplasty) or arthroscopic.

In rehabilitation, regardless of management type, the shoulder is braced in 20° of external rotation and abduction for 4 weeks to aid healing of posterior capsule. Pendulum exercise and elbow range of motion three times per day are encouraged. At 4 weeks, unlimited progressive range of motion is initiated as well as isometric posterior rotator cuff strengthening. Noncontact sports are allowed 3 months after reduction or surgery, and contact sports are permitted 4–6 months postoperatively [22].

24.4 Posterior Instability of the Shoulder

Posterior shoulder instability has an incidence between 2 and 12 % in all cases of shoulder instability [23]. While posterior instability is relatively uncommon when compared with anterior instability, it has become increasingly more recognized as a challenging condition in athletic populations. Posterior shoulder instability includes a continuum of pathological changes ranging from a chronic locked posterior dislocation to the more common recurrent posterior subluxation. Athletes typically develop posterior shoulder instability secondary to repetitive, sport-specific motions, which inflict minor traumatic stress to the posterior capsular labrum complex.

Many operative procedures have been described for the treatment of posterior instability, but today, the arthroscopic capsular labrum reconstruction is an effective and reliable procedure (with or without anchors).

24.4.1 Postoperative Rehabilitation and Return to Sport

Immediately after surgery, the rehabilitation protocol employed the use of an ultra sling (DonJoy, Carlsbad, California) that immobilized the shoulder in approximately 30° of abduction while preventing internal rotation. The sling is used for 4–6 weeks depending on the amount of capsular laxity found at time of the surgery. On the first postoperative day, the patient begins active wrist and elbow flexion and extension exercises as well as gentle pendulums and passive scaption exercises. After the sling immobilization is discontinued at 4–6 weeks postoperatively, gentle passive ROM exercises are advanced, and gentle, pain-free internal rotation is allowed. Active assisted ROM exercises and isometric internal and external rotation exercises are also initiated at this time. By 2–3 months postoperatively, the patient progress to full passive and active ROMs. At this time, capsular strengthening exercises are also instituted and isotonic strengthening continued, with the emphasis on the rotator cuff and the posterior deltoid. At approximately 6 months postoperatively, isokinetic testing is performed. Once the patient is able to achieve 80 % strength and endurance compared with the contralateral side, a sport-specific rehabilitation protocol can be initiated. In general, athletes are required to achieve full ROM without pain, full strength, and endurance comparable with the contralateral side before return to competition. Most patients can return to competition by 6 months postoperatively. Throwing athletes deserve special consideration and accordingly are placed on a specific protocol in which their throwing distance and speed are closely monitored and slowly advanced over 2–3 months. Once the throwing athlete is able to perform full-speed throwing for 2 consecutive weeks without symptoms, return to full competition is permitted.

24.5 SLAP Lesion

Tears of superior labrum were initially identified and characterized by Andrews et al. [24] in 1985 during the early years of shoulder arthroscopy. A few years later, Snyder et al. [25] coined the phrase "SLAP" tear to identify the anatomic location and orientation of the lesion: "superior labrum anterior to posterior" (Figs. 24.2 and 24.3). It was thought that these lesions were the result of significant traction on the long head of the biceps tendon during the deceleration phase of throwing [26]. More recently, it has been suggested that forces during the late cocking and acceleration phases of throwing may create a "peel-back" phenomena that leads to SLAP [24, 25].

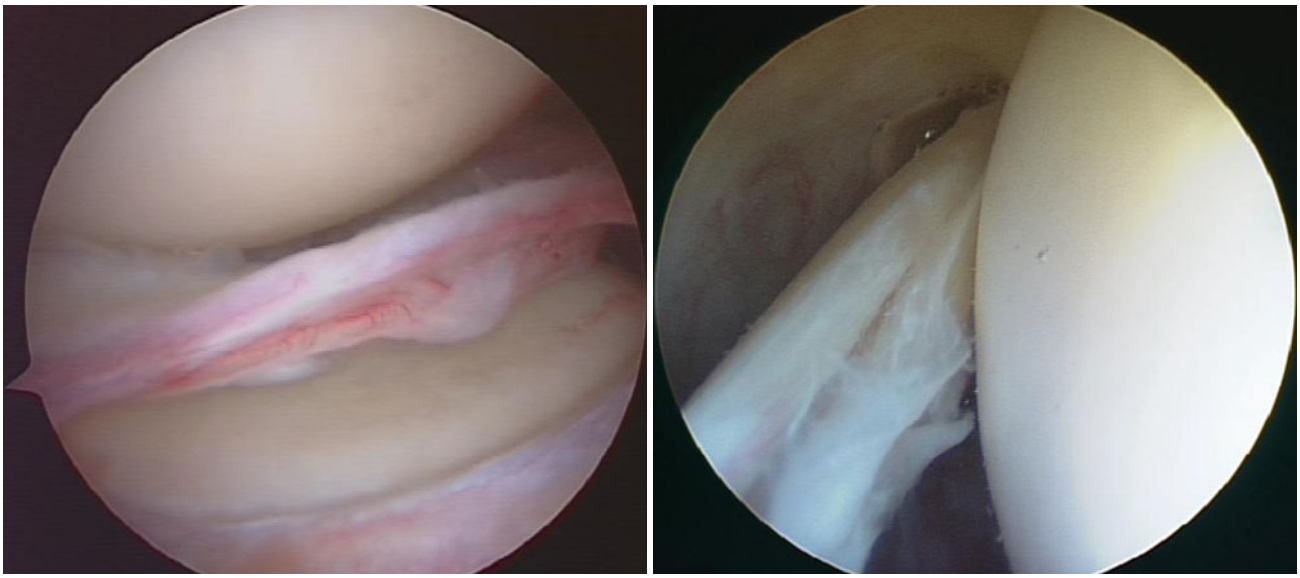
While treatment typically begins with a structured therapy regimen, surgery may be necessary in elite and recreational level athletes to allow them to continue in their sport.

After surgery, it is extremely important to protect the biceps/labral complex for 6 weeks postoperatively to allow appropriate healing. Many protocols have been divided into phases. Each phase may vary slightly based on the individual patient and special circumstances.

The *overall goals* of the surgical procedure and rehabilitation are to:

- Control pain and inflammation
- Regain normal upper extremity strength and endurance
- Regain normal shoulder range of motion
- Achieve the level of function based on the orthopedic and patient goals

Early passive range of motion with noted limitations is highly beneficial to enhance circulation within the joint to promote healing. The physical therapy should be initiated within the first week following surgery. The supervised rehabilitation program is to be supplemented by a home fitness



Figs. 24.2 and 24.3 Example of SLAP lesion (type 3 and 4)

program where the patient performs the given exercises at home or at a gym facility.

Important postoperative signs to monitor include:

- Swelling of the shoulder and surrounding soft tissue
- Abnormal pain response, hypersensitivity—an increase in night pain
- Severe range of motion limitations
- Weakness in the upper extremity musculature

Return to activity requires both time and clinical evaluation. At 6 months postoperatively, pitchers are generally able to begin throwing from a mound and progressed accordingly, initially throwing fastballs with increasing effort and followed then by off-speed pitches.

To safely and most efficiently return to normal or high-level functional activity, the patient requires adequate strength, flexibility, and endurance. Functional evaluation including strength and range of motion testing is one method of evaluating a patient's readiness to return to activity. Return to intense activities following a SLAP repair requires both a strenuous strengthening and range of motion program along with a period of time to allow for tissue healing. Symptoms such as pain, swelling, or instability should be closely monitored by the patient.

In recent years, there have been several reports regarding the outcomes of arthroscopic repair of SLAP lesion. However, clinical experience with elite throwing athletes has indicated that is not, in fact, always the case [27].

High-demand patients, particularly overhead elite athletes, have greater challenges in returning to their pre-injury level of play, despite good or excellent results on outcomes questionnaires and routine function.

Neuman et al. [28] support the hypothesis that SLAP repair leads to improved shoulder function during routine daily activities but that consistent return to elite throwing sports, particularly at same preoperative level, remains challenging.

Recently, several authors have demonstrated that returning throwing and overhead athletes to pre-injury level may be more difficult than previously reported [29].

Arthroscopic SLAP repair provides a clinical and statistically significant improvement in shoulder outcomes. However, a reliable return to the previous activity level is limited and within this population, 37 % of patients failed with 28 % revision rate. Age greater than 36 years is associated with a higher chance of failure [30]. Additional work is necessary to determine the optimal diagnosis, indications, and surgical management for those with SLAP injuries.

24.6 Rotator Cuff

The rotator cuff is an important structure for overhead-throwing athletes because it functions as the primary dynamic stabilizer of the shoulder during throwing and other overhead activity. Rupture of the rotator cuff can happen for trauma on the shoulder during sport, but the rotator cuff injury in overhead-throwing athletes is focused on young professional athletes, mostly baseball players in the USA or volley player in Europe, for example. They have a repetitive throwing motion that leads to posterior-superior impingement of the rotator cuff against the glenoid, which in turn can lead to articular-sided partial tears of the rotator cuff (Fig. 24.4) with accompanying lesions of the biceps such as SLAP or pulley lesions.

The coupled force vectors of the subscapularis and teres minor muscles contribute to depression of the humeral head

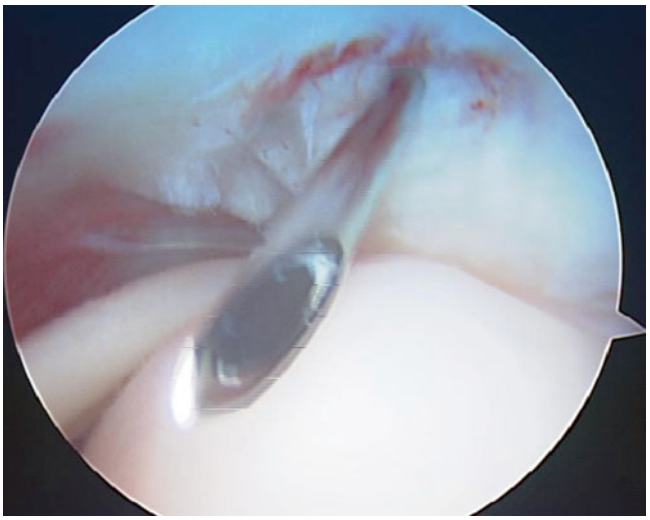


Fig. 24.4 Supraspinatus articular partial tear

in the glenoid cavity. This provides dynamic stability to the glenohumeral joint and prevents impingement of the humeral head with the acromion during deltoid activation. They also function to prevent superior translation of the humeral head after rotator cuff tear. The scapula also plays an important role in glenohumeral function by providing a stable base for muscle activation and load transfer within the kinetic chain. Alteration in normal scapular position or kinematics has been termed scapular dyskinesia and can affect rotator cuff function. Scapular dyskinesia has also been described following rotator cuff tears and is thought to represent a compensatory mechanism for glenohumeral motion deficits [31]. The tendon healing is the really important point: healing of ruptured rotator cuff tendon only occurs when the tendon is surgically repaired back to its footprint on the proximal humerus. Histological studies suggest that three phases of rotator cuff healing occur after surgical repair. These include an inflammatory phase, a proliferative or repair phase, and remodeling phase [32]. A firm understanding of the timing of these phases is important to safely individualize rehabilitation protocols after rotator cuff repair. Following surgical tendon-to-bone fixation, inflammatory cells followed by platelets and fibroblasts migrate into the repair site over the first week and begin to proliferate over the next 2–3 weeks. The cellular proliferation and matrix deposition of this phase are thought to be regulated by several growth factors and initially yield primarily type III collagen. Approximately 3–4 weeks following repair, the remodeling phase begins and scar tissue organizes through extracellular matrix turnover. The initial type III collagen deposition is slowly replaced by type I collagen, continuing until mature scar tissue is formed. Remodeling repair tissue does not reach maximal tensile strength for a minimum of 12–16 weeks post repair. The normal tendon-to-bone transition including unmineralized and mineralized fibrocartilage is not recreated during the remodeling phase. Rather, repairing the rotator cuff to its anatomic location facilitates the formation of scar tissue in a manner that secures the torn tendon to the greater tuberosity in a more anatomic location. This, in turn, allows the rotator cuff to function at an anatomic length and tension, which is important if normal function is to be restored.

The post-op rehabilitation protocol is normally the same for all athletes. The patients are immobilized in an abduction pillow holding the arm in approximately 20° of abduction and 30° of internal rotation for 3–4 weeks. The pillow is gradually discontinued after 3 weeks. During the first 6 weeks, physiotherapy consisted of passive range of motion exercises for the shoulder. Range of motion limits are continuously increased from 60° of abduction, 60° of flexion, and 10° of external rotation in week 1 to 90° of abduction, 145° of flexion, and 45° of external rotation in week 6. Free passive range of motion is allowed at week 7 and isometric strengthening exercises are increased to eccentric strengthening and weight training in week 12. Sport-specific exercises are begun after week 12, whereas return to overhead-throwing sport is advised no sooner than week 21.

The arthroscopic rotator cuff repair allows patients to return to their pre-injury level in every kind of sport in very high percent [27].

24.7 Discussion

"***Hey Doc, when can I play again after this shoulder surgery?" How many times have you been asked this question***?

Athletes, sports associations, sports agents, and coaches all need to know how long recovery times will be before returning to competitive sports and these times should possibly be as short as possible.

Answers should be different, depending on pathology, technique used, etc., *but the most important information required is the exact biological recovery time of the tissues treated. For a safe recovery of athletic movements, an evaluation of the full functional and muscle recovery is always required. Far too often, an early return to competitive sports in poor athletic condition or with a wrong posture, during a moment of supposed tissue recovery, implies a higher risk of traumatic recurrence or the reappearance of pain with or without a limitation of articular functions.*

The joint capsule of the shoulder often reacts to reparative surgery through various forms of adhesive capsulitis, also in localized occurrences that need to be identified timely and treated accordingly. A recovery of the complete passive articular ROM should always be a prerequisite, before proceeding to performing any athletic movement with the upper limbs.

A return to throwing events after therapeutic shoulder arthroscopy can be therefore permitted after accurate medical,

physiotherapeutic, and athletic evaluation. For example, an indication of 6 months as the average recovery time before returning to competitive sports after arthroscopic capsular plastic is only a purely indicative statistical datum reported in literature.

This rule cannot and shall not be applied automatically, without precise biological, clinical, and biomechanical evaluations. Isolated strength recovery can lead to misevaluate the complete recovery of an operated shoulder, but cannot guarantee articular stability.

The evaluation of tendon integrity in rotator cuff reparative surgery can be studied and assessed through NMR, ultrasonography, and/or medical tests, but doubts regarding tissue elasticity, mechanical resistance, and final biology of the repaired tendon will not be dispelled [31].

American authors have in fact, unfortunately, often observed that a full athletic recovery, that is, a recovery achieving the technical, competitive, and professional level prior to the onset of the injury, is not accomplished easily at the follow-up, particularly in athletes affected by a SLAP lesion of the long head of the biceps, even when correctly repaired, and despite following rigorous rehab protocols (*see annex*).

Which rehab protocols shall be applied? The following tables show the experience of an Italian Scientific Society (*SIGASCOT*; *see* Table 24.2 *from 1 to 2*) *with the sole purpose of indicating scientific trends in rehabilitation protocols rather than guidelines. In professional and amateur sports, as well as in work activities requiring physical effort, the focus is on athletes or workers, rather than in their illness or on a different surgical act. Before passing from a phase of the rehab protocol to the subsequent, the target set for the previous phase has to be reached.*

Accelerating functional and strength recovery times are pointless if, for example, the shoulder has not reached a complete degree of passive articular ROM and pain has not disappeared. A faster or slower return to physical activity also depends on the different sporting disciplines (e.g., *in contact sports, a slight residual stiffness can be tolerated, but this is a serious problem in throwing events*). *An Italian Consensus Conference like SICS&G* (*Italian Society Shoulder & Elbow Surgery/ISS&ES*) *speaks, for example, about 180 days at least for a reasonable time to sport return after a surgical treatment for instability pathology* [33].

Table 24.2 Rotator cuff repair rehab program

Rehab protocol after rotator cuff reparative operation (page 1)

	GOALS	TUTOR	RANGE OF MOTION	THERAPEUTIC EXCERCISES
Phase 1 (1-4 weeks)	Respect of biological cicatrization times Reduction of pain Reduction of articular stiffness	Recommended for 4-6 weeks, also during sleeping. It can be taken off only when doing passive mobilization exercises	Progressive passive bending up to 90° Progressive passive abduction up to 90° Passive extra rotation only when arm is abducted	Codman's pendulum exercise Active mobilization exercises for wrist and elbow Only passive mobilization of shoulder
Phase 2 (5-8 weeks)	Improvement of articular mobility Intial recovery of muscular strength	Tutor removal	Progressive passive bending up to 160° Progressive passive abduction up to 90° Passive extra rotation up to 60°	Capsular stretching Self-aided mobilization exercises (pulleys) Close kinetic chain isometric exercises Strengthening shoulder-blade stabilizers Aquatic rehabilitation

Rehab protocol after rotator cuff reparative operation (page 1)

	GOALS	TUTOR	RANGE OF MOTION	THERAPEUTIC EXCERCISES
Phase 1 (1-4 weeks)	Respect of biological cicatrization times Reduction of pain Reduction of articular stiffness	Recommended for 4-6 weeks, also during sleeping. It can be taken off only when doing passive mobilization exercises	Progressive passive bending up to 90° Progressive passive abduction up to 90° Passive extra rotation only when arm is abducted	Codman's pendulum exercise Active mobilization exercises for wrist and elbow Only passive mobilization of shoulder
Phase 2 (5-8 weeks)	Improvement of articular mobility Intial recovery of muscular strength	Tutor removal	Progressive passive bending up to 160° Progressive passive abduction up to 90° Passive extra rotation up to 60°	Capsular stretching Self-aided mobilization exercises (pulleys) Close kinetic chain isometric exercises Strengthening shoulder-blade stabilizers Aquatic rehabilitation

Rehab protocol after rotator cuff reparative operation (page 2)

	GOALS	TUTOR	RANGE OF MOTION	THERAPEUTIC EXCERCISES
Phase 3 (9-16 weeks)	Recovery of muscular strength Recovery of neuromuscular control Recovery of	No	Progressive complete recovery of articular mobility at all levels	Strengthening exercises with elastic bands and later, open kinetic chain exercises with light dumbbels Plyometric exercises with therapy balls Neuromotorial re-education with Kabat method of exercise Stretching exercises
Phase 4 (17-24 weeks)	Recovery of sports movement	No	At this stage, it has to be complete	Continue with strengthening exercises with increasing load Sport-specific exercises and recovery of athletic condition Stretching exercises

References

1. Martin CT, Gao Y, Pugely AJ, Wolf BR (2013) 30-day morbidity and mortality after elective shoulder arthroscopy: a review of 9410 cases. J Shoulder Elbow Surg 22:1667–1675
2. Tjoumakaris FP, Bradley JP (2008) Posterior shoulder instability. In: Galaz LM (ed) Orthopaedic knowledge update: shoulder and elbow, 3rd edn. American Academy of Orthopedic Surgeons, Rosemont, pp 313–320
3. Matsen FA III (2002) Letter: the biomechanics of glenohumeral stability. J Bone Joint Surg Am 84(3):495–496
4. Halder AM, Kuhl SG, Zobitz ME, Larson D, An KN (2001) Effects of glenoid labrum and glenohumeral abduction on stability of the shoulder joint through concavity-compression: an in vivo study. J Bone Joint Surg Am 83(7):1062–1069
5. Levine WN, Flatow EL (2000) The pathophysiology of the shoulder instability. Am J Sports Med 28(6):20–28
6. Lippitt S, Matsen F (1993) Mechanisms of glenohumeral joint stability. Clin Orthop Relat Res 291:20–28
7. Curl LA, Warren RF (1996) Glenohumeral joint stability: selective cutting studies on the static capsular restraints. Clin Orthop Relat Res 330:54–65
8. Baker CL, Uribe JW, Witman C (1990) Arthroscopic evaluation of acute initial anterior shoulder dislocation. Am J Sports Med 18(1):25–28
9. Mc Laughlin HL (1952) Posterior dislocation of the shoulder. J Bone Joint Surg Am 34:584–590
10. Jobe FW, Kvitne RS, Giangarra CE (1989) Shoulder pain in the overhand or throwing athlete. The relationship of anterior instability and rotator cuff impingement. Orthop Rev 18(9):963–975
11. Jobe CM (1995) Posterior superior glenoid impingement expanded spectrum. Arthroscopy 11(5):530–536
12. Walch G, Boileau P, Noel E (1992) Impingement of the deep surface of the supraspinatus tendon on the posterosuperior glenoid rim: an arthroscopic study. J Shoulder Elbow Surg 1:238–245
13. Tibone JE (1996) Glenohumeral instability in overhead athletes. In: Bigliani L (ed) The instable shoulder. American Academy of Orthopaedic Surgeons, Rosemont, pp 91–98
14. O'Neill DB (1999) Arthroscopic Bankart repair of anterior detachments of the glenoid labrum. A prospective study. J Bone Joint Surg Am 81(10):1357–1366
15. Mair SD, Zarzour RH, Speer KP (1998) Posterior labrum injury in contact athletes. Am J Sports Med 26(7):753–758
16. Kim SH, Ha KI, Jung MW, Lim MS, Kim YM, Park JH (2003) Accelerated rehabilitation after arthroscopic Bankart repair for selected cases: a prospective randomized clinical study. Arthroscopy 19(7):722–731
17. Murray IR, Mc Birnie J (2009) Return to sport following arthroscopic shoulder stabilization. British Elbow and Shoulder Society. Shoulder Elbow 1:114–118
18. Solomon DJ, Provencher MT (2008) Shoulder instability and surgical stabilization: return to sports and activities. Sports Med Update 1:2–7
19. Kowalsky MS, Levine WN (2008) Traumatic posterior glenohumeral dislocation: classification, pathoanatomy, diagnosis and treatment. Orthop Clin North Am 4:519–533
20. Hawkins RJ (1985) Unrecognized dislocations of the shoulder. Instr Course Lect 34:258–263
21. Rowe CR, Zarins B (1982) Chronic unreduced dislocation of the shoulder. J Bone Joint Surg Am 64(4):494–505
22. Roleau DM, Davies JH, Robinson MC (2014) Acute traumatic posterior shoulder dislocation. J Am Acad Orthop Surg 22(3): 145–152
23. Antoniou J, Harryman DT II (2001) Posterior instability. Orthop Clin North Am 32:463–473
24. Andrews JR, Carson WG Jr, Mc Leod WD (1985) Glenoid labrum tears related to the long head of the biceps. Am J Sports Med 13:337–341
25. Snyder SJ, Karzel RP, Del Pizzo W, Ferkel RD, Friedman MJ (1990) SLAP lesions of the shoulder. Arthroscopy 6:274–279
26. Shepard MF, Dugas JR, Zeng N, Andrews JR (2004) Differences in the ultimate strength of the biceps anchor and the generation of the type II superior labral anterior posterior lesion in a cadaveric study. Am J Sports Med 32(5):1197–1201
27. Cohen SB, Sheridan S, Ciccotti MG (2011) Return to sports for professional baseball players after surgery of the shoulder and elbow. Sports Health 3(1):105–111
28. Neuman BJ, Boisvert CB, Reiter B, Lawson K, Ciccotti MG, Cohen SB (2011) Results of arthroscopic repair of type II superior labral anterior posterior lesion in overhead athletes: assessment of return to preinjury playing level and satisfaction. Am J Sports Med 39:1883–1889
29. Knesek M, Skendzel JG, Dines JS, Altechek DW, Allen AA, Bedi A (2013) Diagnosis and management of superior labral anterior posterior tears in throwing athletes. Am J Sports Med 41:444–460
30. Provencher MT, McCormick F, Dewing F, McIntire S, Solomon D (2013) A prospective analysis of 179 type 2 superior labrum anterior and posterior repairs: outcomes a factors associated with success and failure. Am J Sports Med 41:880–886
31. Economopoulos KJ, Brockmeier SF (2012) Rotator cuff tears in overhead athletes review article. Clin Sports Med 31(4): 675–692
32. van der Meijden OA, Westgard P, Chandler Z, Gaskill TR, Kokmeyer D, Millett PJ (2012) Rehabilitation after arthroscopic rotator cuff repair: current concepts review and evidence-based guidelines. Int J Sports Phys Ther 7(2):197–218
33. McCarty EC, Ritchie P, Gill HS, McFarland EG (2004) Shoulder instability: return to play. Clin Sports Med 23:335–351

Thrower's Elbow

25

Christopher S. Warrell, Daryl C. Osbahr,
and James R. Andrews

25.1 Introduction

With the growing popularity of overhead throwing sports, and the increasing involvement in year-round competition, overuse injuries to the elbow have become an epidemic among throwing athletes. As more attention has been directed toward this population, a better understanding of throwing biomechanics and the associated pathology has been obtained, and numerous unique injury patterns have been identified.

As the arm passes through the late cocking and early acceleration phases of the throwing motion, tensile stress is placed upon the medial soft tissues, while the lateral and posterior compartments of the elbow experience compressive and medially directed sheer forces, respectively. These abnormal forces can result in a multitude of distinct injuries relating to the thrower's elbow, including ulnar collateral ligament (UCL) tears, flexor-pronator mass injuries, ulnar neuritis, posteromedial impingement, olecranon stress fractures, osteochondritis dissecans (OCD) of the capitellum, and medial epicondyle apophyseal injuries. Accurate diagnosis and proper care of elbow injuries in the throwing athlete require a thorough understanding of elbow anatomy and function, and numerous unique injury patterns have been identified.

C.S. Warrell, MD (✉) • D.C. Osbahr, MD
Division of Sports Medicine, Orlando Health Orthopedic Institute,
Orlando Health, 1222 S. Orange Avenue, 5th Floor,
Orlando, FL 32806, USA
e-mail: cswarrell@gmail.com; osbahrd@gmail.com

J.R. Andrews, MD
Division of Sports Medicine, The Andrews Institute,
1040 Gulf Breeze Parkway, Suite 203,
Gulf Breeze, Florida, 32561, USA

25.2 Functional Anatomy

The elbow is a ginglymus, or hinged, joint, which is comprised of three separate articulations: the ulnohumeral, radiocapitellar, and proximal radioulnar joints. The ulnohumeral joint provides the primary bony support of the elbow via a constrained articulation between the distal humeral trochlea and the sigmoid notch of the ulna. In particular, it is the largest contributor to elbow stability at less than 20° of flexion and greater than 120° of flexion, when the coronoid process and olecranon engage their respective fossae on the distal humerus. The radiocapitellar joint provides a lesser degree of valgus stability through resistance of compressive forces at the lateral elbow. Between 20° and 120° of flexion, the majority of the static and dynamic stability of the elbow is provided by surrounding soft tissue structures.

The ulnar collateral ligament (UCL) is the most important static soft tissue contributor to elbow stability in the throwing athlete. The UCL is composed of three distinct components: an anterior bundle, a posterior bundle, and a transverse ligament. The anterior bundle originates on the anteromedial edge of the medial epicondyle of the humerus and inserts on the sublime tubercle of the ulna. The posterior bundle is a fan-shaped fascicle that originates on the posteroinferior medial epicondyle and attaches on the medial aspect of the ulna. The transverse ligament, or Cooper's ligament, extends from the olecranon to the base of the coronoid process. Previous studies have shown that the anterior bundle of the UCL serves as the primary restraint to valgus force throughout the functional range of motion, between 20° and 120° of flexion [1–3]. The posterior bundle is thinner and weaker, and it provides secondary elbow stability at greater degrees of flexion, while the transverse ligament serves to expand the greater sigmoid notch as a thickening of the joint capsule.

Important dynamic stabilizers of the elbow include the flexor-pronator mass (FPM). This muscular group shares a common origin on the medial epicondyle of the humerus and, from proximal to distal, consists of the pronator teres,

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_25, © Springer International Publishing Switzerland 2016

flexor carpi radialis (FCR), palmaris longus (when present), flexor digitorum superficialis (FDS), and flexor carpi ulnaris (FCU). Altogether, the flexor-pronator mass assists the UCL in creating the varus torque required to counteract the valgus force generated in the throwing motion. However, biomechanical data has shown that the FCU serves as the primary dynamic restraint to valgus force, as a result of its position in line with the UCL [4].

In throwing athletes, the ulnar nerve can be prone to symptomatology and must be evaluated. The ulnar nerve runs along the medial upper arm and crosses from the anterior to the posterior compartment at the arcade of Struthers before passing through the cubital tunnel at the elbow. The nerve then exits the cubital tunnel between the humeral and ulnar heads of the FCU and runs superficial to the flexor digitorum profundus (FDP) in the forearm.

25.3 Pathophysiology and Biomechanics

The throwing motion has been divided into six phases: windup, early cocking, late cocking, acceleration, deceleration, and follow-through [5–9]. The late cocking/early acceleration and deceleration phases have been identified as being particularly injurious to the thrower's elbow, as the joint moves from approximately 110 to 20° of flexion with extension velocities approaching 3,000°/s [10]. This violent motion creates significant valgus and extension moments at the elbow, which produce tensile stress on the medial stabilizing structures and compression and medially directed shear forces in the lateral and posterior compartments of the elbow, respectively. This combination of forces may subsequently result in overload of the valgus stabilizers of the elbow resulting in pathologic changes in the medial aspect of the elbow, and it serves as the basic underlying etiology for most pathologic conditions in the thrower's elbow [11–14].

Tensile stress at the medial elbow is primarily resisted by the anterior bundle of the UCL. Previous studies have shown that the UCL produces approximately 54 % of the varus torque necessary to counteract valgus force on the elbow at 90° of flexion [1]. The maximum valgus torque generated in the overhead throw can exceed 64 N-m, which is roughly twice the ultimate tensile strength of the UCL (32.1 ± 9.6 N-m) [10]. Repetition of these near-tensile failure loads during the overhead throwing motion in the presence of flexor-pronator fatigue can lead to attritional injury or acute rupture of the UCL. Depending upon the throwing athlete's age, increased stress to the static stabilizers of the medial aspect of the elbow can result in UCL injuries, flexor-pronator injuries, olecranon stress fractures, medial epicondylitis, medial epicondyle apophysitis, and medial epicondyle avulsion injuries [12, 13, 15].

The ulnar nerve is also susceptible to injury secondary to its position at the medial elbow. Even in healthy arms, the overhead throwing motion has been shown to cause increases in ulnar nerve intraneural pressure and cubital tunnel pressure [16–18]. In addition, the throwing motion places upon the nerve a significant amount of strain, which approaches the elastic and circulatory limits of the nerve [19]. This suggests that repetition of the throwing motion can lead to intraneural injury and ischemia, and the nerve could be particularly vulnerable in the presence of concomitant valgus instability. In addition, the presence of osteophytes, flexor-pronator mass hypertrophy, and/or thickening of medial elbow soft tissues can lead to direct compression of the nerve, as well as to restriction of the nerve's mobility [20].

On the lateral side of the elbow, compressive forces measuring upwards of 500 N have been observed at the radiocapitellar joint during the late cocking and early acceleration phases of throwing [10]. It has been theorized that such pathologic overloading of the lateral elbow compartment leads to changes in the subchondral blood supply, which may result in radiocapitellar chondromalacia, cartilage degeneration, and formation of osteochondral fractures and loose bodies [21].

At the posterior elbow, the combined valgus and extension forces result in a "windshield wiper" effect, where the tip of the olecranon translates medially on the humeral trochlea. During normal kinematics in a healthy elbow, the FPM and UCL function to prevent excessive medial translation and consequential impingement between the olecranon and olecranon fossa. However, in the setting of valgus instability, further impingement may occur at the posteromedial elbow, possibly leading to chondromalacia and osteophyte formation. Classically, this phenomenon has been described during the deceleration phase of the throwing motion at low elbow flexion angles [11, 12, 22–25]. However, recent biomechanical data from Osbahr et al. has confirmed that there are increased contact forces in the posteromedial UCL-deficient elbow at 90° of flexion during the acceleration phase of throwing [13]. This study introduced the concept of ulnohumeral chondral and ligamentous overload (UCLO), which suggests that there is a continuum of abnormal contact forces and resultant posteromedial ulnohumeral impingement throughout the entire arc of the throwing motion in the setting of UCL insufficiency.

25.4 History and Physical Examination

Evaluation of the throwing athlete with elbow pain begins with a thorough history and physical examination. When obtaining the history, it is important to note details regarding sport participation, previous injuries, recent changes in training regimen, and aggravating factors. For pitchers, particular attention should be paid to pitch count, innings pitched, and

types of pitches thrown. Any changes in velocity, accuracy, strength, or stamina should also be documented, as these could suggest a chronic UCL injury. If possible, it is also important to determine the phase of throwing during which pain is experienced, as the majority of athletes with valgus instability will report experiencing pain during the late cocking/early acceleration and deceleration phases of the throwing motion [10]. Mechanical symptoms may suggest the presence of intra-articular loose bodies, radiocapitellar osteochondral lesions, posteromedial chondromalacia, or posteromedial olecranon osteophytes.

Inquiry should also be made regarding vascular or neurologic complaints. Numbness or tingling in the hand or fingers, cold intolerance, subjective loss of grip strength, and frequent dropping of objects may indicate an ulnar neuropathy [17, 26]. Ulnar nerve symptoms can present in the setting of nerve subluxation or chronic overuse, or they may provide a clue to additional underlying pathology, such as valgus instability.

The physical examination involves a standardized approach to evaluation of the entire kinetic chain, including the shoulder and elbow, with an intent to truly comprehend how to improve the pathological process relating to the injured throwing athlete. Both upper extremities are first inspected to detect any asymmetry in the forearm musculature, the resting position of the elbow, and the elbow carrying angle. In particular, a more valgus carrying angle may be due to adaptive changes to repetitive abnormal valgus stress placed upon the elbow [27]. While the normal carrying angle is 11° of valgus in men and 13° of valgus in women, King et al. noted that the throwing athlete is commonly found to have a carrying angle >15° [27].

It is important to assess both passive and active range of motion of the elbow compared to the contralateral upper extremity. The normal flexion/extension arc should range from 0° to 140°, while normal pronation and supination should both range approximately 80–90° from neutral. As pointed out by Cain et al., particular attention should be paid to the "end-feel" with the flexion/extension motion arc [12]. The normal "end-feel" of flexion should be that of the soft tissue of the upper arm contacting the soft tissue of the forearm, while extension should conclude with the firm sensation of the olecranon engaging the olecranon fossa. A soft end-feel at terminal extension may indicate a flexion contracture, which is present in approximately half of professional pitchers and not necessarily indicative of an injury [27–29]. Conversely, a firm end-feel that interrupts terminal flexion is generally a pathologic finding, related to osteophytic changes or loose bodies [12].

Palpation of the elbow should employ a systematic approach and investigate both bony landmarks and soft tissue structures. Important bony landmarks include the olecranon, the medial epicondyle, and the radial head. Pain with palpation of the olecranon may indicate an olecranon stress fracture or inflammation due to underlying posteromedial impingement [30, 31]. Tenderness at the medial epicondyle may indicate apophysitis or, in the immature athlete, an avulsion fracture or growth plate injury. The radial head is palpated while the forearm is passively rotated, and the presence of pain may provide a diagnostic clue to an underlying fracture or OCD lesion [21].

Palpation of the soft tissue begins with the UCL. This is performed with the elbow in approximately 50–70° of flexion to displace the FPM anterior to the ligament [12]. The UCL should be palpated from its origin on the medial epicondyle to its insertion on the sublime tubercle of the ulna. Pain with palpation has high sensitivity (81–94 %) but poor specificity (22 %) for ligamentous injury [32, 33]. Tenderness with palpation of the FPM can indicate a muscular strain, which may be an isolated injury or an indication of increased stress due to underlying valgus instability.

The ulnar nerve should be palpated along its course at the medial elbow. Any pain with palpation or paresthesias with percussion at the cubital tunnel should alert the examiner to the possibility of ulnar neuropathy. The nerve should also be examined for subluxation at the medial epicondyle, which can be a source of pain and paresthesias [26, 30, 34].

Provocative maneuvers are an important part of the physical examination of throwing athletes. Maneuvers designed to test for medial instability include the valgus stress test, the moving valgus stress test, and the "milking maneuver." With the valgus stress test, the patient's arm is stabilized, the elbow is flexed to approximately 30°, and a valgus stress is applied. In this position, most of the stress is placed on the anterior band of the UCL [15]. The test is positive if there is loss of a firm end point and increased medial sided joint opening when compared to the contralateral upper extremity. The test produces pain in approximately 50 % of patients with a torn UCL, and it has a sensitivity and specificity of 66 and 60 %, respectively [32, 35]. The moving valgus stress test was initially described by O'Driscoll and colleagues and was designed to simulate the valgus force experienced during the overhead throwing motion [36]. The maneuver begins with the patient's elbow placed in full flexion, and the examiner maintains a constant valgus torque on the elbow while the elbow is quickly extended to approximately 30°. The test is positive if it reproduces the patient's medial elbow pain between 70° and 120° of flexion. The original study describing the technique reported high sensitivity (100 %) and specificity (75 %) for UCL insufficiency [36]. The "milking maneuver," however, is performed by pulling on the patient's thumb with the forearm fully supinated, the shoulder flexed forward, and the elbow flexed beyond 90°. This exam places valgus torque on the elbow in a higher degree

of flexion and better assesses the integrity of the posterior band of the UCL. A positive test reveals medial sided elbow pain and instability [37, 38]. For the purpose of valgus stress testing, a cadaveric study by Safran et al. showed that neutral rotation is the best forearm position to reveal valgus laxity [39].

The valgus extension overload test is similar to the moving valgus stress test, but is performed at lower degrees of elbow flexion. The elbow is placed in approximately 20–30° of flexion, a constant valgus force is applied, and the elbow is repeatedly forced into terminal extension [11, 40]. This test attempts to recreate the impingement of the posteromedial olecranon in the olecranon fossa, and the test is positive if it reproduces the pain that the patient experiences during throwing. The active radiocapitellar compression test is used to detect OCD lesions of the radiocapitellar joint. The test is performed by applying an axial load on the fully extended arm, while the patient actively pronates and supinates the forearm. The test is positive if there is pain or crepitus at the lateral compartment of the elbow [21, 40].

25.5 Diagnostic Imaging

Diagnostic imaging should begin with standard anteroposterior and lateral radiographs of the injured elbow, which may reveal osteochondral lesions in the radiocapitellar joint, loose bodies, or changes consistent with chronic instability, such as calcification of the UCL. One may also consider including internal and external oblique views to obtain a full "thrower's series" of the elbow, as well as an oblique axial view with the elbow in 110° of flexion to visualize posteromedial olecranon osteophytes [11]. Anteroposterior valgus stress views can reveal excessive medial joint line opening. An opening greater than 3 mm has been considered diagnostic of valgus instability [32, 41], while a difference of >0.5 mm compared to the non-injured upper extremity has been shown to be consistent with a UCL tear [42].

Bone scintigraphy and computed tomography (CT) are useful in the assessment of bony pathology, including stress fractures and avulsion fractures. The soft tissue structures around the elbow may be assessed with the use of ultrasound or CT arthrography. In particular, a study by Timmerman et al. showed that CT arthrogram has a high sensitivity (86 %) and specificity (91 %) for UCL injury [35]. However, MRI is largely considered the gold standard for evaluation of soft tissue injuries about the elbow, including ligamentous injury and tendinopathy, as well as injury to the articular cartilage. Standard MRI without contrast has been reported to have a sensitivity of 57–79 % and a specificity of 100 % for diagnosing UCL tears [35, 43]. Enhancement of the MRI with intra-articular saline or gadolinium increases the sensitivity to greater than 90 % and improves diagnosis of partial tears, but it comes with the limitations of being a more invasive and costly test [44, 45]. Standard and enhanced MRI also has an important role in the classification and management of capitellar OCD lesions, as these studies can help identify unstable and high-grade lesions, which may respond poorly to nonoperative treatment [46, 47].

25.6 Prevention

With the growing popularity of overhead throwing sports, and the increasing involvement in year-round competition, overuse injuries to the elbow have become an epidemic among throwing athletes. Coincident with the rise in such injuries has been an increased interest in their prevention. To this end, the USA Baseball Medical & Safety Advisory Committee was created to provide scientifically based information and recommendations to help reduce injury. In 2004, this committee established guidelines for youth baseball players in an effort to reduce the incidence of such injuries [48]. These guidelines, which were partly based on research by the American Sports Medicine Institute, included recommendations for pitch counts, pitch types, pitching mechanics, multiple appearances, multiple leagues, year-round baseball, and physical conditioning [49, 50] (Table 25.1). Adherence to these guidelines has been shown to correlate with the incidence of pitching-related arm pain and pitching-

Table 25.1 USA Baseball Youth Baseball Pitching Recommendations [48]

Arm pain	Remove from game immediately; if >4 days of arm pain, seek medical attention			
Pitch counts	Game	Week	Season	Year
9–10 years old	50	75	1,000	2,000
11–12 years old	75	100	1,000	3,000
13–14 years old	75	125	1,000	3,000
Pitch types	No breaking pitches until bones have matured around puberty (~13 years old)			
Multiple appearances	Once removed from the mound, do not return to pitching in the same game			
Showcases	De-emphasize and/or avoid, if necessary; give adequate time to prepare with no overthrowing			
Multiple leagues	Pitch for only one team at a time, with no overlapping seasons			
Year-round baseball	Baseball pitchers should compete in <9 months of baseball each year			

related injuries in multiple studies [50–54]. In particular, a follow-up study by Olsen et al. noted that athletes pitching more than 8 months per year were five times more likely to require shoulder or elbow surgery, and athletes pitching more than 80 pitches per game were four times more likely to require surgery [50]. Additionally, those who only occasionally pitched with a fatigued arm were four times more likely to undergo surgery, while those who regularly pitched with a fatigued arm were 36 times more likely to have an injury that required surgery [50].

Despite the literature supporting the US baseball injury prevention guidelines and the implementation of injury prevention programs, additional research shows that further work must be done to raise public awareness of high-risk throwing activities. A recent study by Ahmad et al. investigated the public perception of UCL reconstruction and found that 31 % of coaches, 28 % of players, and 25 % of parents did not believe that the number of pitches thrown was a risk factor for injury [55]. In addition, 51 % of high school athletes, 37 % of parents, 30 % of coaches, and 26 % of collegiate athletes thought that UCL reconstruction should be performed on players without elbow injury in order to enhance performance. These studies highlight the need for continued endeavors to better educate players, parents, and coaches regarding prevention of overuse throwing injuries [55].

25.7 Valgus Instability/Ulnar Collateral Ligament Injuries

Injury to the UCL was first described in javelin throwers by Waris in 1946 [56]. Since that time, UCL injuries have been reported in increasing frequency among other overhead athletes, particularly baseball pitchers. The UCL is the primary restraint to valgus stress throughout the functional range of motion, between 20° and 120° of flexion, and it is subjected to enormous valgus forces during the throwing motion. These forces approach the ultimate tensile strength of the UCL, and repetition of the overhead throwing motion can lead to attritional injury and/or acute rupture. The most well-studied treatment options for UCL injury include nonoperative management with formal rehabilitation, direct ligament repair, and ligament reconstruction.

Nonoperative management of an isolated UCL injury begins with short-term immobilization to control pain and inflammation, as well as to limit valgus stress on the elbow. This is followed by a comprehensive rehabilitation program as described by Wilk et al., which consists of functional exercises and plyometrics and focuses on pitching mechanics, shoulder kinematics, and motion deficits, as well as strengthening of the core, upper extremities, and lower extremities [57–60]. Once the throwing athlete is pain-free and kinetic chain deficits have been addressed, they may transition to an interval throwing program. This conservative approach is generally indicated in non-throwing athletes and similarly low-demand individuals, and it may also be considered in the immature throwing athlete with a partial tear of the ligament [61]. Skeletally mature, UCL-deficient athletes involved in high-demand throwing sports may not respond well to nonoperative treatment [62, 63]. A study by Rettig reported that 42 % of throwing athletes were able to return to their sport at or above their pre-injury level of play following nonoperative management with appropriate rehabilitation [64]. An injection of platelet-rich plasma (PRP) may be considered, although the data is limited for use in UCL tears. Podesta et al. treated 34 overhead athletes (including 27 professional baseball players) with partial UCL tears with injections of platelet-rich plasma (PRP) and rehabilitation [65]. They reported 88 % excellent results with return to their previous level of competition or higher.

Direct repair of the UCL was initially the treatment of choice for UCL injuries, as early data showed better clinical outcomes when compared to nonoperative treatment [66]. However, additional studies that compared UCL repair to reconstruction found that overhead athletes are more likely to achieve better outcomes and return to their previous level of competition with reconstruction of the ligament [15, 67, 68]. A recent study by Savoie et al. showed that a good indication for UCL repair may be the young athlete with a proximal or distal UCL tear with a good quality ligament. In their retrospective case series of 60 young amateur athletes (mean age, 17.2 years), they reported 93 % good or excellent outcomes following direct repair of proximal or distal UCL tears using suture anchors or suture plication with repair to bone drill holes [69].

Reconstruction of the UCL is often indicated in the high-level overhead throwing athlete who sustains a complete tear of the UCL and wishes to return to throwing sports. Ligament reconstruction is also considered in the throwing athlete who sustains a partial tear of the UCL and continues to have pain and/or instability despite an appropriate course of nonoperative treatment, including a comprehensive rehabilitation program as noted above. Jobe et al. described the first reconstruction technique that afforded players a successful return to competition, utilizing a free-tendon graft placed through bone tunnels in the ulna and medial epicondyle of the humerus in a figure-of-eight fashion [5]. The flexor-pronator origin was detached for the surgical approach, and submuscular transposition of the ulnar nerve was performed. Ten of 16 (63 %) throwing athletes were able to return to their previous level of competition; however, roughly one half of the patients had complications, including five ulnar neuropraxias and one flexor-pronator mass rupture [41].

Since the original figure-of-eight technique was described, multiple modifications have been made in an effort to facilitate anatomic reconstruction, obtain strength similar to the

native UCL, and expedite secure graft fixation, all while decreasing morbidity associated with disruption of the flexor-pronator mass and transposition of the ulnar nerve [32, 70–74]. To this end, most modifications have addressed the surgical approach and/or the method of graft fixation on both the ulnar and humeral sides. With regard to the surgical approach, Jobe himself transitioned to a flexor-pronator muscle splitting approach, as described by Smith and Altchek [75], and abandoned obligatory transposition of the ulnar nerve. This modified Jobe technique exhibited improved results with a greater proportion of patients returning to their previous level of play (82 %), as well as a decreased complication rate (12 %) [32].

In 1995, Andrews and Timmerman introduced the American Sports Medicine Institute (ASMI) modification, which utilizes a posterior approach between the two heads of the flexor carpi ulnaris, with elevation of the flexor-pronator mass and obligatory subcutaneous ulnar nerve transposition [67]. This approach leaves the flexor-pronator origin intact and avoids morbidity associated with takedown and repair of the flexor-pronator mass. Cain et al. evaluated the clinical outcome of the ASMI modification in 1,281 throwing athletes and found that 83 % of athletes were able to return to their pre-injury level of competition [76]. Complications occurred in approximately 20 % of the patients, but most (96 %) of these were considered minor, including transient ulnar nerve symptoms.

Modifications for graft fixation have included the docking technique [70], interference screw fixation [71], suture anchor fixation [72], and cortical suspensory fixation [73]. The DANE TJ technique (named in acknowledgement of Drs. David Altchek and Neal ElAttrache, as well as the first professional baseball player to undergo UCL reconstruction and successfully return to competition, Tommy John) is one modification which employs two modern fixation techniques, utilizing interference screw fixation on the ulnar side and the docking technique on the humeral side [74]. To date, the figure-of-eight and docking techniques remain the most well-studied reconstruction techniques with reported long-term outcomes [23, 24, 76–79]. However, regardless of the fixation used, most modern techniques have similar outcomes, with 80–90 % of athletes returning to their previous level of play. An overall complication rate has been reported of between 15 and 20 %, with most consisting of transient ulnar neuropathy and superficial wound infection at either the graft harvest site or the elbow [23, 24, 76–79].

Following surgery, the patient should engage in a four-phase rehabilitation program as described by Wilk et al. and noted above [57–60]. The first phase begins immediately after surgery and continues for 3 weeks. Following the UCL reconstruction, the patient's arm is placed in a posterior splint to immobilize the elbow at 90° of flexion. The splint is kept in place for 1 week to allow for initial wound healing, and the patient is permitted to perform wrist and hand range of motion and hand grasping exercises during this time. After 1 week, a hinged brace is applied and adjusted to allow motion from 30° to 100° of elbow flexion. The elbow motion is increased in a stepwise fashion until the patient achieves full range of motion by the end of the fifth to sixth week after surgery. The hinged elbow brace is discontinued at the end of the 8th week. During phase II (weeks 4–10) and phase III (weeks 10–16), the patient works on progressive strengthening and continued stretching and flexibility exercises. By week 12 the patient is permitted to begin an isotonic lifting program, including bench press, latissimus dorsi pull downs, seated rows, triceps push downs, and biceps curls. Week 12 also marks the time when the throwing athlete may begin a plyometric throwing program. The first 2 weeks of the plyometric program consist of two-hand throws, such as chest passes, soccer throws, and side throws. During the following 2 weeks, the patient is allowed to transition to one-hand throws. Phase IV (weeks 16 and beyond), the return to activity phase, consists of a formal interval throwing program. Throwing athletes are permitted to begin throwing from the mound approximately 6–8 weeks after initiation of the interval throwing program, and return to competitive throwing can be expected 9–12 months after surgery [57–60].

25.8 Ulnar Neuritis

Ulnar nerve neuritis can also occur in overhead throwing athletes secondary to the nerve's position at the medial elbow, where it is susceptible to compression and traction as well as to inflammation of nearby stabilizing structures. On presentation, athletes typically complain of pain at the medial elbow and sensory disturbance in the ulnar aspect of the hand as well as the ring and small fingers. Overt motor weakness is rare in the thrower, but it can instead present as loss of ball control or difficulty with performance of complex hand tasks. On exam, the physician should determine if there is subluxation or dislocation of the nerve with palpation or elbow range of motion (Fig. 25.1). Patients may also exhibit a positive Tinel sign at the cubital tunnel, as well as a positive elbow flexion test, which reproduces pain, numbness, and tingling in the ulnar nerve distribution with maintained maximum elbow flexion and wrist extension for at least 1 min [80].

In addition to standard radiographic imaging, electrodiagnostic studies including electromyography (EMG) and nerve conduction velocities (NCV) may be obtained as part of the diagnostic work-up in cases with equivocal findings on the physical examination. However, results of such studies must be interpreted with caution, as negative test results do not rule out the diagnosis of ulnar neuritis and symptoms of dynamic compression or traction. Rather, positive findings are typically seen only with chronic or advanced nerve entrapment [18, 26, 33]. Symptoms of ulnar nerve inflammation or compression should alert the physician to possible

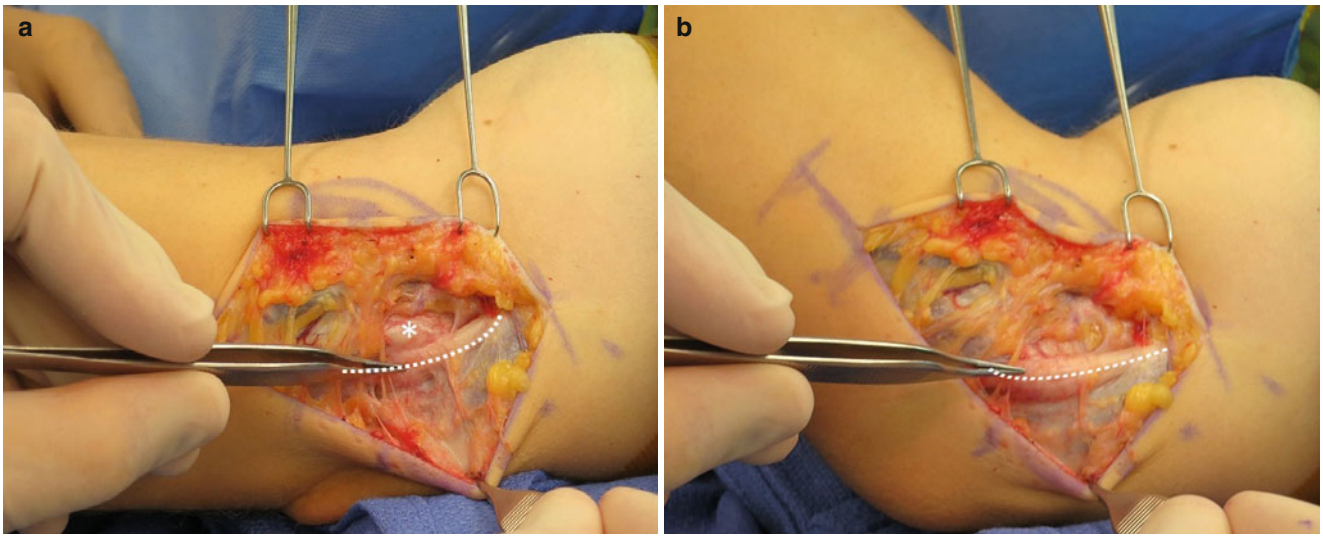


Fig. 25.1 (**a**) Intraoperative examination of the ulnar nerve (*dashed line*) with the arm in extension identifies the nerve located in its anatomic position behind the medial epicondyle (*). (**b**) In the setting of ulnar nerve instability, flexion of the arm at the elbow results in dislocation of the nerve (*dashed line*) anterior to the medial epicondyle (Copyright Daryl C. Osbahr)

underlying elbow instability. In a systematic review of athletes undergoing UCL reconstruction, approximately 30 % endorsed concomitant ulnar neuropathy [81]. Similarly, ulnar nerve symptoms are reported in as many as 60 % of throwing athletes with medial epicondylitis. Treatment options for ulnar neuritis include nonoperative management, decompression, medial epicondylectomy, and anterior submuscular or subcutaneous transposition.

Treatment of isolated ulnar neuritis should begin with nonoperative management, including cessation of sports activities, rest, ice, and nonsteroidal anti-inflammatory drugs (NSAIDs). In the presence of nerve subluxation or dislocation, a 2-week trial of immobilization may be indicated, as well. Once the patient is asymptomatic, a stretching routine may be established for the elbow, forearm, and wrist, followed by a progressive isometric strengthening program and gradual return to sport-specific functions [33]. Greater duration and severity of symptoms, as well as presence of concomitant valgus instability, may predict decreased success with nonoperative treatment [82, 83].

Surgical intervention may be considered when nonoperative management fails or when the patient presents with advanced symptoms, such as motor weakness or muscular wasting. There is limited data on use of simple decompression or medial epicondylectomy to treat ulnar neuritis in throwing athletes. However, in the throwing athlete, decompression alone is generally not recommended, as it does not eliminate traction force on the ulnar nerve, and medial epicondylectomy may destabilize the UCL or FPM as well as predispose to ulnar nerve subluxation or dislocation [37, 83, 84]. Most of the available literature focuses on anterior submuscular or subcutaneous transposition of the nerve. Historically, some authors have recommended submuscular transposition for the potential advantage of better protection of the ulnar nerve from direct and indirect trauma [17, 18, 20, 33, 34, 41]. More recently, there has been increasing support for subcutaneous transposition in throwing athletes, as this avoids morbidity associated with disruption of the flexor-pronator mass, especially in overhead athletes [23, 67, 68, 85, 86].

Regardless of the method of surgically addressing the ulnar nerve, the nerve must be adequately released and mobilized to ensure that there is no tethering or compression of the nerve along its entire course. Particular attention should be made to free the nerve proximally from the arcade of Struthers and distally from the fascia between the two heads of the FCU, as these areas have been identified as common causes of incomplete release and recurrent ulnar nerve symptoms [87–89].

We prefer subcutaneous transposition for the aforementioned reasons. The surgical approach is similar to that used for UCL reconstruction, and it begins with a 4–5 cm incision centered over the medial epicondyle. The medial antebrachial cutaneous nerve is identified and protected, and the ulnar nerve is released from the cubital tunnel, as well as its proximal and distal restraints, as noted above. A fascial sling is created from a strip of the medial intermuscular septum. The ulnar nerve is transposed anterior to the medial epicondyle, and the fascial sling is laid loosely over the nerve and sutured to the fascia of the FPM. The elbow is then taken through a gentle range of motion to ensure that the ulnar nerve is able to move freely without compression or tethering. The cubital tunnel and fascia of the FCU are both closed. Meticulous hemostasis is obtained using electrocautery, and a drain is placed with plans for removal before discharge home the same day. The wound is closed in two layers, including a sub-

cuticular closure reinforced with Steri-Strips (3 M, St. Paul, Minnesota). The elbow is splinted at 90° of flexion for 1 week. Following splint removal, the patient is permitted to begin progressive range of motion exercises and rehabilitation.

25.9 Flexor-Pronator Injuries

The flexor-pronator musculature provides dynamic stability to the medial elbow, and it assists the UCL in creating the varus torque necessary to counteract the valgus forces created during the overhead throwing motion. Repetition of the throwing motion can lead to muscular fatigue, chronic tendinosis, attritional injury, and acute rupture of the FPM [12, 90]. On presentation, the throwing athlete will typically describe pain during the late cocking and acceleration phases of the throwing motion. Examination usually reveals tenderness just distal to the FPM origin on the medial epicondyle, and resisted wrist flexion and forearm pronation exacerbate pain. The most significant differential diagnosis which must be ruled out is concomitant injury to the UCL. Studies have shown that FPM injuries accompany approximately 4.3 % of UCL injuries, and the risk of combined FPM and UCL injuries among baseball players increases after 30 years of age [15, 91].

The vast majority of FPM injuries respond well to conservative treatment, including rest, ice, and a course of anti-inflammatory medication, followed by physical therapy and a gradual return to throwing. Surgery is considered in throwers with chronic tendinosis, which does not respond to at least 3–6 months of nonoperative treatment, and in the rare case of complete rupture with associated valgus instability.

The literature on operative treatment and outcomes of FPM injuries is limited. A study by Vangsness showed that approximately 90 % of patients with isolated chronic tendinosis have a good or excellent result, and >95 % of athletes are able to return to sports activities, following detachment of the FPM origin, excision of abnormal tissue, and reattachment of FPM [92]. A more recent case series by Osbahr et al. identified a population of baseball players undergoing UCL reconstruction who sustained concomitant flexor-pronator injuries [91] (Fig. 25.2). Compared to baseball players with isolated UCL injuries, baseball players with combined flexor-pronator and UCL injuries were found to be significantly older (33.4 years versus 20.1 years) and had a significantly lower rate of return to prior level of play (12.5 %) [91].

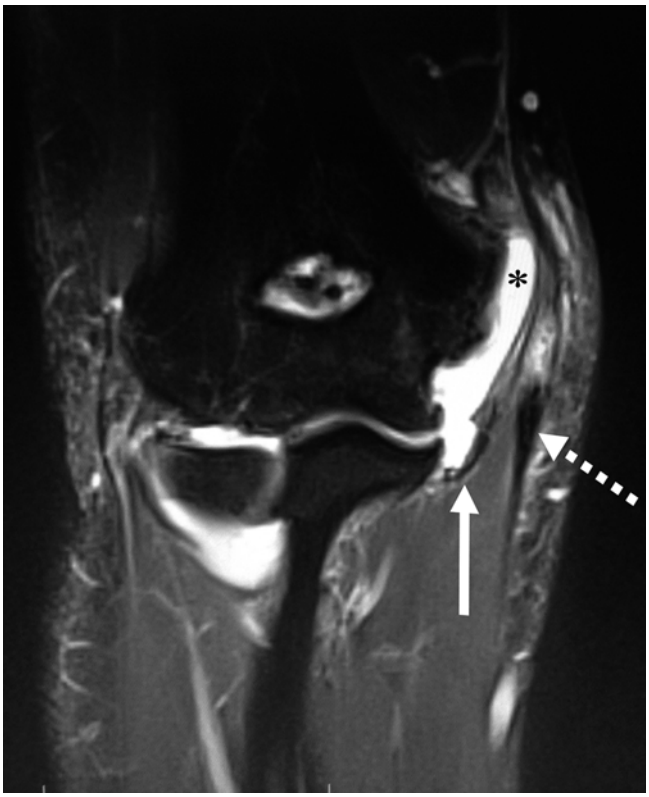


Fig. 25.2 Coronal T2-weighted MR arthrogram of the elbow demonstrating combined injury to the ulnar collateral ligament (*solid arrow*) and flexor-pronator mass (*dashed arrow*). Note the abnormal proximal extension of intra-articular contrast (*) to the level of the medial epicondyle (Copyright Daryl C. Osbahr)

25.10 Medial Epicondyle Apophyseal Injuries

Recent decades have seen an increase in elbow injuries in youth baseball pitchers [53]. This has been attributed to high pitch counts and increased sport participation, including year-round league play, involvement in concurrent leagues, and travel team play [51, 52]. While adolescent athletes are susceptible to UCL injuries, the substantial valgus forces created by the overhead throw more typically affect the relatively weak medial epicondyle apophyseal plate, resulting in medial epicondyle apophysitis and avulsion injuries [12].

Classically, medial epicondyle apophyseal injuries have been thought to result from repetitive microtrauma over a prolonged time period. Early studies by Bennett and Brogdon introduced the concept of "Little Leaguer's elbow" to describe the clinical and radiographic findings discovered in the throwing arms of youth baseball players [93, 94]. Their patients were noted to present with a prior history of pain, swelling, and tenderness at the medial elbow, and radiographs revealed fragmentation and physeal widening at the medial epicondyle. More recent studies have corroborated their initial reports; however, the aforementioned chronic radiographic findings have since been seen among asymptomatic adolescent baseball athletes, including both pitchers and position players [95–100]. This has created some controversy regarding the overall significance of these findings, as well as their exact incidence among asymptomatic youth

throwers. Depending on the study population, the incidence of radiographic widening and fragmentation of the medial epicondyle has been reported to range from 4 to 50 % [95, 97–100].

While medial epicondyle apophysitis generally presents with chronic complaints and findings, medial epicondyle avulsion fractures may occur with a characteristic acute presentation while throwing. A case series by Osbahr et al. reported on eight previously asymptomatic youth baseball players who experienced a sudden acute avulsion fracture during the act of throwing [95]. Patients typically reported a sudden pain or "pop" while throwing and presented with acute pain, swelling, and tenderness, as well as decreased range of motion [95]. Plain radiographs are usually sufficient for diagnosis and most often reveal a Salter-Harris type I fracture, but fragmentation of the epicondyle may be observed. A CT scan may be considered to determine total fracture displacement, as well as to assist with treatment decision making.

As previously noted, prevention is key in the management of elbow injuries in youth athletes, and adherence to the USA Baseball Medical & Safety Advisory Committee guidelines has been shown to correlate with the incidence of youth pitching-related arm pain and pitching-related injuries [48–54]. Beyond prevention, the management of medial epicondyle apophysitis is generally straightforward, and good results have been achieved with rest, ice, and activity modification with occasional bracing or splinting [97]. However, there is still much debate within the literature about the optimal treatment of medial epicondyle fractures [12, 91, 101–104]. Many authors agree that non-displaced fractures may be adequately treated with a brief period of immobilization in a long-arm splint or cast with the elbow flexed to 90°, yet there [12, 103]. Considerable controversy regarding treatment of minimally displaced (2–5 mm) medial epicondyle fractures in throwing athletes, as based upon the notion that minimal degrees of valgus instability may be less tolerable in this population. Surgical decision making is further complicated by the fact that the magnitude of fracture displacement may be underestimated by standard radiographs, and the fact that there is low interobserver and intraobserver agreement as based upon standard radiograph measurements [105, 106]. A CT scan may be obtained if there is uncertainty regarding fracture displacement and optimal treatment, but it comes with the risk of increased radiation exposure. Absolute surgical indications typically include open fractures, gross elbow instability, incarceration of the fracture fragment, or entrapment of the ulnar nerve [103, 107].

When operative treatment is indicated, most authors support open reduction and internal fixation with a single screw, with or without a washer [95, 103, 104]. Postoperatively, the elbow is immobilized at 70–90° of flexion with the forearm in neutral rotation for a maximum of 3 weeks. Patients are then placed in a hinged elbow brace to resist valgus forces. Rehabilitation begins at 3 weeks with physical and occupational therapy to work on range of motion, followed by progressive strengthening and gradual return to physical activity. A throwing program may begin once there is radiographic evidence of fracture union, good upper extremity strength, and pain-free range of motion [95].

25.11 Osteochondritis Dissecans of the Capitellum

Osteochondritis dissecans of the capitellum is another condition that is seen primarily in the adolescent overhead athlete. The exact etiology of this disorder remains controversial, but it is believed to be multifactorial and strongly associated with repeated microtrauma to the poorly vascularized immature capitellum [21, 108, 109]. Vascular studies have shown that the capitellum is primarily supplied by posterior end arteries that traverse the articular cartilage, and there is an absence of significant metaphyseal collateral blood flow [21–111]. The overhead throwing motion produces significant compression forces in the lateral compartment of the elbow, which are believed to cause injury to the aforementioned subchondral end arteries, resulting in ischemia, osteonecrosis, and formation of loose bodies [12, 21, 109, 112].

Historically, the management of capitellar OCD has been based upon multiple factors, including the grade and size of the lesion, as well as the state of the capitellar physis [108, 109, 112–114]. Multiple grading systems have been established and are based upon the appearance of the OCD lesion on plain radiographs, CT, MRI, and arthroscopy [112, 115–117]. In general, each of these systems grades the lesion as stable, unstable but attached, or detached and loose. Nonoperative management is typically reserved for patients with stable lesions and an open capitellar physis, and it includes activity modification, use of nonsteroidal anti-inflammatory drugs (NSAIDs), and cessation of sports participation for 3–6 months. Recent data has shown that approximately 90 % of such patients can expect spontaneous healing with nonoperative management [112, 113].

Operative management is indicated in patients with stable lesions that have failed 6 months of nonoperative management and in patients who present with unstable lesions, articular loose bodies, or mechanical symptoms. The goals of surgery are stimulation of a healing response, removal of loose bodies, and resolution of mechanical symptoms. Surgical treatment options include arthroscopic versus open removal of loose bodies, capitellum debridement, abrasion chondroplasty, fragment excision, fragment fixation, micro-

fracture, humeral osteotomy, or osteochondral autograft transplantation surgery (OATS procedure). Most of the outcome data for surgical treatment of capitellar OCD comes from retrospective case series, which had small sample sizes and/or did not utilize modern arthroscopic techniques [118–126]. This makes it difficult to draw significant conclusions or recommend one procedure over another. A systematic review by de Graaff et al. reported on the findings of nine such studies with 219 total patients undergoing arthroscopic treatment for OCD of the capitellum [127]. This included 41 patients who underwent osteochondral autografting and 178 patients who underwent debridement, drilling, microfracture, and/or fragment fixation depending on the grade of their osteochondral lesion. Those patients undergoing osteochondral autografting had a return to sport rate ranging from 77–90 %, and 94 % were pain-free, while all other patients had a return to sport ranging from 80–100 %, and 84–100 % were pain-free [127].

Ruchelsman et al. provided a useful algorithm on capitellar OCD treatment [21]. They recommend retrograde drilling for lesions with intact overlying cartilage and arthroscopic debridement with marrow stimulation (microfracture) for lesions with unstable cartilage caps or loose bodies. Open osteochondral autograft and allograft procedures are reserved for large defects that involve more than 50 % of the width of the articular surface or that engage the radial head [21]. When an open approach is desired, either a direct lateral or posterolateral approach to the elbow may be used, depending on the location of the lesion.

25.12 Posteromedial Impingement

Impingement of the posteromedial bony and soft tissue structures may occur with the repetitive elbow extension and valgus forces created by the overhead throwing motion, particularly in the setting of UCL insufficiency. Such impingement can result in soft tissue swelling, osteophyte formation, chondromalacia, and the development of intra-articular loose bodies. Athletes may complain of pain at the posterior elbow, swelling, crepitus, locking, and/or loss of terminal extension, and they are likely to present with a positive valgus extension overload test [14, 128–130]. Plain radiographs, especially axial and oblique views, can help identify posterior elbow osteophytic changes, and MRI with intra-articular contrast can be performed to detect loose bodies and inflammation of soft tissue structures.

Treatment of posteromedial impingement begins with prevention, including the early recognition and prompt treatment of UCL insufficiency. Nonoperative management typically consists of NSAIDs and active rest, followed by rehabilitation focusing on the entire kinetic chain, including the lower extremities, core, scapular shoulder, and elbow. Elbow rehabilitation should focus on range of motion, flexibility, and flexor-pronator strengthening. As symptoms resolve, the athlete may be permitted to begin a throwing mechanics program followed by a progressive interval throwing program and a gradual return to competition. If the patient does not obtain relief despite adequate rehabilitation, they may be considered a candidate for arthroscopic or open

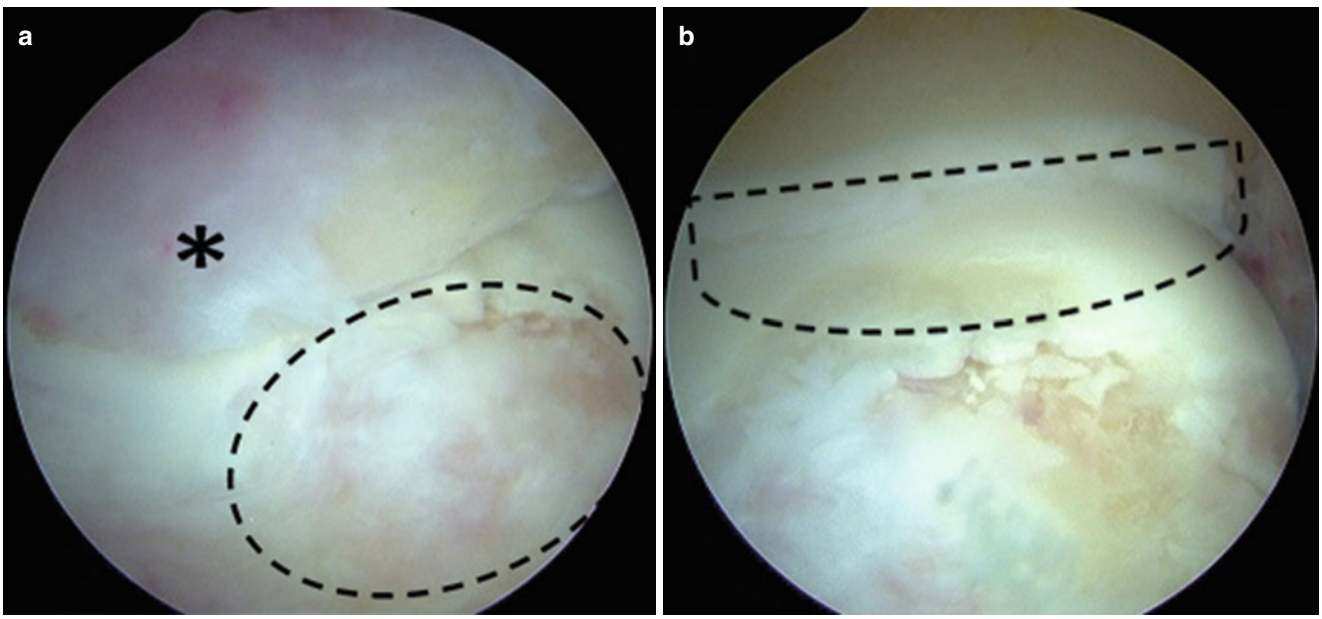


Fig. 25.3 (**a**) Arthroscopic examination of an athlete with posteromedial impingement reveals a large posteromedial olecranon osteophyte (*) and associated chondromalacia of the humeral trochlea (*circle*). (**b**) Removal of the olecranon osteophyte (*dashed line*) reveals more extensive cartilage damage and allows further evaluation and treatment of the posterior humeral trochlea (Copyright Daryl C. Osbahr)

debridement of the elbow with focus on osteophyte excision, treatment of chondromalacia, and removal of loose bodies.

Arthroscopic debridement has become the treatment of choice, as it allows excision of loose bodies, direct visualization of articular surfaces, drilling of osteochondral defects, and evaluation of the UCL for undersurface tears (Fig. 25.3). To date, there have been very few studies which have specifically investigated the outcomes of arthroscopic debridement for the treatment of posteromedial impingement. Rahusen et al. reported on 16 athletes with isolated posterior impingement who underwent arthroscopic debridement of the olecranon and posterior fossa [131]. There was no comparison group, but their cohort had statistically significant improvement in the modified Andrews elbow scoring system (69/100 preoperatively versus 93/100 postoperatively) and the visual analog scale for pain, both at rest (3/10 versus 0/10) and with activity (7/10 versus 2/10) [131]. Outcome data from other studies has shown that arthroscopic treatment with debridement, olecranon osteophyte excision, and loose body removal has permitted 72–85 % athletes to return to play at their previous level of competition [31, 132, 133]. Additionally, the American Sports Medicine Institute's 2-year follow-up data on UCL reconstruction showed that athletes had an equivalent or higher return to play rate (86 % versus 82 %) when olecranon osteophyte excision was performed at the same time as their UCL reconstruction, compared to performing UCL reconstruction alone [76]. In the same study, arthroscopic debridement of an olecranon osteophyte was the most common reason for additional surgery, and reoperation for olecranon osteophyte excision after UCL reconstruction carried a worse prognosis for return to play at the same level of competition or higher (71 %) [76].

When planning to perform arthroscopic excision of olecranon osteophytes, it is important to discuss with the athlete the risk of unmasking or creating valgus instability. A study by Andrews and Timmerman noted that approximately 25 % of professional baseball players who had previously undergone a posteromedial olecranon osteophyte excision required a subsequent UCL reconstruction [67]. This phenomenon may be due to an unmasking of preexisting subclinical valgus instability caused by an insufficient UCL, such as that seen in the setting of ulnohumeral chondral and ligamentous overload, and highlights the importance of early recognition of UCL insufficiency. Similarly, when osteophyte excision is performed, care must be taken to limit the removal of bone to the osteophytic overgrowth, only, as recent studies have shown that overzealous excision involving the native olecranon may result in medial elbow instability [134–137]. A biomechanical study by Kamineni et al. found that resections of the posteromedial aspect of the olecranon >3 mm may jeopardize the function of the anterior bundle of the UCL because it places increased strain upon the ligament [136].

Table 25.2 Olecranon stress injuries

Type 1	Adolescent apophyseal line olecranon stress injury
Type 2	Metaphyseal olecranon stress reaction
Type 3	Mid-proximal oblique olecranon stress fracture
Type 4	Proximal transverse olecranon stress fracture
Type 5	Olecranon tip stress fracture

25.13 Olecranon Stress Fractures

Olecranon stress fractures in overhead throwing athletes are thought to be related to the interaction of the osseous and soft tissue restraints of the elbow as they respond to the repetitive, enormous valgus, and extension loads, which are generated during the throwing motion [10, 15]. This includes impingement of the posteromedial olecranon, as well as excessive tensile stress of the triceps tendon and an intact UCL [10, 30]. Patients will typically present with tenderness upon palpation of the posterior olecranon, particularly the posteromedial aspect [30]. Conventional radiographs are an important first step in imaging for stress fractures; however, radiographic findings may not be apparent early in the disease process resulting in a delay in diagnosis [138–143]. For this reason, advanced imaging including bone scintigraphy, MRI, or CT may be necessary to establish a diagnosis.

To further guide diagnosis and treatment, Osbahr et al. established a classification system, which expanded upon previous work by Nakaji et al., to include both skeletally immature and skeletally mature injury patterns [144, 145] (Table 25.2). In general, olecranon stress injuries without a discrete fracture line on imaging will heal well with conservative management, including throwing cessation and active rest with or without use of a bone stimulator, followed by a three-phase rehabilitation program focusing on progressive range of motion, strengthening, and a throwing program [30, 144, 146, 147]. However, in throwing athletes, olecranon stress injuries with a fracture line demonstrated on conventional radiographs may require operative treatment to ensure successful healing and return to play [144, 145, 148–150]. When operative treatment is indicated, many authors support open reduction and internal fixation with the use of a single, cannulated cancellous screw (6.5–7.3 mm) for transverse fracture patterns, while more proximal oblique fracture patterns may require two 4.0 mm screws placed perpendicular to the fracture line [144, 150–152]. Figure-of-eight tension band wiring with either high strength suture or metal wire may be used to supplement the fixation, but may increase the likelihood of reoperation for painful retained hardware [152–154]. Elbow arthroscopy may be employed to aid in fracture reduction, as well as to address other intra-articular pathologies, including loose bodies, osteophytes, and chon-

dromalacia. Additionally, olecranon tip fractures can be treated via arthroscopic debridement with isolated olecranon tip excision [12, 144].

In the largest case series to date, Paci et al. reported on the outcomes of 18 baseball players with olecranon stress fractures, which were treated by open reduction and internal fixation with a single, titanium, cannulated compression screw [152]. All patients went on to successful union, and 17 of the 18 patients (94 %) were able to return to baseball at or above their prior level of play. However, six (33 %) patients required hardware removal, including two for infection. Their series also highlighted the incidence of concomitant and future throwing injuries, including UCL tears (11 %), and the value of using titanium screws to reduce MRI interference should future MRI be warranted [152].

25.14 Summary

The overhead throwing motion creates significant valgus and extension moments at the elbow, which can result in unique injury patterns seen in the throwing athlete. These abnormal forces can result in a multitude of distinct injuries, including ulnar collateral ligament tears and sprains, flexor-pronator mass tears and strains, ulnar neuritis, posteromedial impingement, olecranon stress fractures, osteochondritis dissecans (OCD) of the capitellum, and medial epicondyle apophyseal injuries. Accurate diagnosis and proper treatment of elbow injuries in the throwing athlete requires a thorough understanding of elbow function and anatomy, as well as an understanding of throwing mechanics and pathomechanics. The preoperative evaluation should focus on a thorough history and physical examination, followed by use of specific diagnostic imaging modalities. Recent advances in both nonoperative and operative treatment, including arthroscopic techniques, have afforded the athlete a successful return to competition.

Resources

1. Morrey BF, An KN (1983) Articular and ligamentous contributions to the stability of the elbow joint. Am J Sports Med 11:315–319
2. Schwab GH, Bennett JB, Woods GW, Tullos HS (1980) Biomechanics of elbow instability: the role of the medial collateral ligament. Clin Orthop 146:42–52
3. Morrey BF, Tanaka S, An KN (1991) Valgus stability of the elbow: a definition of primary and secondary constraints. Clin Orthop 265:187–195
4. Park MC, Ahmad CS (2004) Dynamic contributions of the flexor-pronator mass to elbow valgus stability. J Bone Joint Surg Am 86:2268–2274
5. Jobe FW, Moynes DR, Tibone JE, Perry J (1984) An EMG analysis of the shoulder in pitching: a second report. Am J Sports Med 12:218–220
6. Sisto DJ, Jobe FW, Moynes DR, Antoneli DJ (1987) An electromyographic analysis of the elbow in pitching. Am J Sports Med 15:260–263
7. Glousman RE, Barron J, Jobe FW, Perry J, Pink M (1992) An electromyographic analysis of the elbow in normal injured pitchers with medial collateral ligament insufficiency. Am J Sports Med 20:311–317
8. DiGiovine NM, Jobe FW, Pink M, Perry J (1992) An electromyographic analysis of the upper extremity in pitching. J Shoulder Elbow Surg 1:15–25
9. Hamilton CD, Glousman RE, Jobe FW, Brault J, Pink M, Perry J (1996) Dynamic stability of the elbow: electromyographic analysis of the flexor pronator group and the extensor group in pitchers with valgus instability. J Shoulder Elbow Surg 5:347–354
10. Fleisig GS, Andrews JR, Dillman CJ, Escamilla RF (1995) Kinetics of baseball pitching with implications about injury mechanisms. Am J Sports Med 23:233–239
11. Wilson FD, Andrews JR, Blackburn TA, McCluskey G (1983) Valgus extension overload in the pitching elbow. Am J Sports Med 11:83–88
12. Cain EL Jr, Dugas JR, Wolf RS, Andrews JR (2003) Elbow injuries in throwing athletes: a current concepts review. Am J Sports Med 31(4):621–635
13. Osbahr DC, Dines JS, Breazeale NM, Deng XH, Altchek DW (2010) Ulnohumeral chondral and ligamentous overload: biomechanical correlation for posteromedial chondromalacia of the elbow in throwing athletes. Am J Sports Med 38:2535–2541
14. Osbahr DC, Dines JS, Rosenbaum AJ, Nguyen JT, Altchek DW (2012) Does posteromedial chondromalacia reduce rate of return to play after ulnar collateral ligament reconstruction. Clin Orthop Relat Res 470:1558–1564
15. Conway JE, Jobe FW, Glousman RE, Pink M (1992) Medial instability of the elbow in throwing athletes. Treatment by repair or reconstruction of the ulnar collateral ligament. J Bone Joint Surg Am 74:67–83
16. Pechan J, Julis I (1975) The pressure measurement in the ulnar nerve: a contribution to the pathophysiology of the cubital tunnel syndrome. J Biomech 8:75–79
17. Glousman RE (1990) Ulnar nerve problems in the athlete's elbow. Clin Sports Med 9:365–377
18. Rokito AS, McMahon PJ, Jobe FW (1996) Cubital tunnel syndrome. Oper Tech Sports Med 4:15–20
19. Aoki M, Takasaki H, Muraki T, Uchiyama E, Murakami G, Yamashita T (2005) Strain on the ulnar nerve at the elbow and wrist during throwing motion. J Bone Joint Surg Am 87:2508–2514
20. Boatright JR, D'Alessandro DF (1996) Nerve entrapment syndromes at the elbow. In: Jobe FW, Pink MM, Glousman RE, Kvitne RE, Zemel MP (eds) Operative techniques in upper extremity sports injuries. Mosby-Year Book, St Louis, pp 518–537
21. Ruchelsman DE, Hall MP, Youm T (2010) Osteochondritis dissecans of the capitellum: current concepts. J Am Acad Orthop Surg 18:557–567
22. Hyman J, Breazeale NM, Altchek DW (2001) Valgus instability of the elbow in athletes. Clin Sports Med 20:25–45
23. Dodson CC, Thomas A, Dines JS, Nho SJ, Williams RJ 3rd, Altchek DW (2006) Medial ulnar collateral ligament reconstruction of the elbow in throwing athletes. Am J Sports Med 34:1926–1932
24. Vitale MA, Ahmad CS (2008) The outcome of elbow ulnar collateral ligament reconstruction in overhead athletes: a systematic review. Am J Sports Med 36:1193–1205
25. Marshall KW, Marshall DL, Busch MT, Williams JP (2009) Osteochondral lesions of the humeral trochlea in the young athlete. Skeletal Radiol 38:479–491
26. Del Pizzo W, Jobe FW, Norwood L (1977) Ulnar nerve entrapment syndrome in baseball players. Am J Sports Med 5:182
27. King JW, Brelsford HJ, Tullos HS (1969) Analysis of the pitching arm of the professional baseball player. Clin Orthop 67:116–123
28. Brown LP, Niehues SL, Harrah A, Yavorsky P, Hirshman HP (1988) Upper extremity range of motion and isokinetic strength of the internal and external shoulder rotators in major league baseball players. Am J Sports Med 16:577–585

29. Wright RW, Steger-May K, Wasserlauf BL, O'Neal ME, Weinberg BW, Paletta GA (2006) Elbow range of motion in professional baseball pitchers. Am J Sports Med 34:190–193
30. Schickendantz MS, Ho CP, Koh J (2002) Stress injury of the proximal ulna in professional baseball players. Am J Sports Med 30(5):737–741
31. Fideler BM, Kvitne RS, Jordan S (1997) Posterior impingement of the elbow in professional baseball players. J Shoulder Elbow Surg 6:169–170
32. Thompson WH, Jobe FW, Yocum LA, Pink MM (2001) Ulnar collateral ligament reconstruction in athletes: muscle-splitting approach without transposition of the ulnar nerve. J Shoulder Elbow Surg 10:152–157
33. Hariri S, Safran MR (2010) Ulnar collateral ligament injury in the overhead athlete. Clin Sports Med 29:619–644
34. Childress HM (1975) Recurrent ulnar nerve dislocation at the elbow. Clin Orthop 108:168–173
35. Timmerman LA, Schwartz ML, Andrews JR (1994) Preoperative evaluation of the ulnar collateral ligament by magnetic resonance imaging and computed tomography arthrography. Evaluation in 25 baseball players with surgical confirmation. Am J Sports Med 22:26–32
36. O'Driscoll SW, Lawton RL, Smith AM (2005) The "moving valgus stress test" for medial collateral ligament tears of the elbow. Am J Sports Med 33(2):231–239
37. Chen FS, Rokito AS, Jobe FW (2001) Medial elbow problems in the overhead throwing athlete. J Am Acad Orthop Surg 9:99–113
38. Jones KJ, Osbahr DO, Schrumpf MA, Dines JS, Altcheck DW (2012) Ulnar collateral ligament reconstruction in throwing athletes: a review of current concepts. J Bone Joint Surg Am 94:e49(1–12)
39. Safran MR, McTarry MH, Shin S, Han S, Lee TQ (2005) Effects of elbow flexion and forearm rotation on valgus laxity of the elbow. J Bone Joint Surg Am 87(9):2065–2074
40. Dugas JR (2010) Valgus extension overload: diagnosis and treatment. Clin Sports Med 29(4):645–654
41. Jobe FW, Stark H, Lombardo SJ (1986) Reconstruction of the ulnar collateral ligament in athletes. J Bone Joint Surg Am 68(8):1158–1163
42. Rijke AM, Goltz HT, McCue FC, Andrews JR, Berr SS (1994) Stress radiography of the medial elbow ligaments. Radiology 191:213–216
43. Ouellette H, Bredella M, Labis J, Palmer WE, Torriani M (2008) MR imaging of the elbow in baseball pitchers. Skeletal Radiol 37:115–121
44. Schwartz ML, Al-Zahrani S, Morwessel RM, Andrews JR (1995) Ulnar collateral ligament injury in the throwing athlete: evaluation with saline-enhanced MR arthrography. Radiology 197:297–299
45. Hill NB Jr, Bucchieri JS, Shon F, Miller TT, Rosenwasser MP (2000) Magnetic resonance imaging of injury to the medial collateral ligament of the elbow: a cadaver model. J Shoulder Elbow Surg 9:418–422
46. Kijowski R, De Smet AA (2005) MRI findings of osteochondritis dissecans of the capitellum with surgical correlation. Am J Roentgenol 185(6):1453–1459
47. Takahara M, Mura N, Sasaki J, Harada M, Ogino T (2007) Classification, treatment, and outcome of osteochondritis dissecans of the humeral capitellum. J Bone Joint Surg Am 89(6):1205–1214
48. USA Baseball Medical & Safety Advisory Committee. Youth baseball pitching injuries. 30 Nov 2008. http://web.usabaseball.com. Accessed 10 June 2014
49. Lyman S, Fleisig GS, Andrews JR, Osinski ED (2002) Effect of pitch type, pitch count, and pitching mechanics on risk of elbow and shoulder pain in youth baseball pitchers. Am J Sports Med 30:463–468
50. Olsen SJ 2nd, Fleisig GS, Dun S, Loftice J, Andrews JR (2006) Risk factors for shoulder and elbow injuries in adolescent baseball pitchers. Am J Sports Med 34:905–912
51. Register-Mihalik JK, Oyama S, Marshall SW, Mueller FO (2012) Pitching practices and self-reported injuries among youth baseball pitchers: a descriptive study. Athl Train Sports Health Care 4:11–20
52. Fleisig GS, Andrews JR, Cutter GR et al (2011) Risk of serious injury for young baseball pitchers: a 10-year prospective study. Am J Sports Med 39:253–257
53. Flesig GS, Andrews JR (2012) Prevention of elbow injuries in youth baseball pitchers. Sports Health 4(5):419–424
54. Yang J, Mann BJ, Guettler JH, Dugas JR, Irrgang JJ, Fleisig GS, Albright JP (2014) Risk-prone pitching activities and injuries in youth baseball: findings from a national sample. Am J Sports Med 42:1456–1463
55. Ahmad CS, Grantham WJ, Greiwe RM (2012) Public perceptions of Tommy John surgery. Phys Sportsmed 40(2):64–72
56. Waris W (1946) Elbow injuries of javelin-throwers. Acta Chir Scand 93:563–575
57. Wilk KE, Arrigo CA, Andrews JR (1993) Rehabilitation of the elbow in the throwing athlete. J Orthop Sports Phys Ther 17:305–317
58. Wilk KE, Arrigo CA, Andrews JR et al (1996) Rehabilitation following elbow surgery in the throwing athlete. Oper Tech Sports Med 4:114–132
59. Wilk KE, Arrigo CA, Andrews JR et al (1996) Preventative and rehabilitation exercises for the shoulder and elbow, 4th edn. American Sports Medicine Institute, Birmingham
60. Wilk KE, Azar FM, Andrews JR (1995) Conservative and operative rehabilitation of the elbow in sports. Sports Med Arthrosc Rev 3:237–258
61. Bruce JR, Andrews JR (2014) Ulnar collateral ligament injuries in the throwing athlete. J Am Acad Orthop Surg 22:315–325
62. Arendt EA (ed) (1999) Orthopaedic knowledge update: sports medicine. American Academy of Orthopaedic Surgeons, Rosemont, pp 225–235
63. Kenter K, Behr CT, Warren RF, O'Brien SJ, Barnes R (2000) Acute elbow injuries in the national football league. J Shoulder Elbow Surg 9:1–5
64. Rettig AC, Sherrill C, Snead DS, Mendler JC, Mieling P (2001) Nonoperative treatment of ulnar collateral ligament injuries in throwing athletes. Am J Sports Med 29:15–17
65. Podesta L, Crow SA, Volkmer D, Bert T, Yocum LA (2013) Treatment of partial ulnar collateral ligament tears in the elbow with platelet-rich plasma. Am J Sports Med 41(7):1689–1694
66. Barnes DA, Tullos HS (1978) An analysis of 100 symptomatic baseball players. Am J Sports Med 6:62–67
67. Andrews JR, Timmerman LA (1995) Outcome of elbow surgery in professional baseball players. Am J Sports Med 23:407–413
68. Azar FM, Andrews JR, Wilk KE, Groh D (2000) Operative treatment of ulnar collateral ligament injuries of the elbow in athletes. Am J Sports Med 28:16–23
69. Savoie FH 3rd, Trenhaile SW, Roberts J, Field LD, Ramsey JR (2008) Primary repair of ulnar collateral ligament injuries of the elbow in young athletes: a case series injuries to the proximal and distal ends of the ligament. Am J Sports Med 36:1066–1072
70. Rohrbough JT, Altchek DW, Hyman J, Williams RJ 3rd, Botts JD (2002) Medial collateral ligament reconstruction of the elbow using the docking technique. Am J Sports Med 30(4):541–548
71. Ahmad CS, Lee TQ, ElAttrache NS (2003) Biomechanical evaluation of a new ulnar collateral ligament reconstruction technique with interference screw fixation. Am J Sports Med 31(3):332–337
72. Hechtman KS, Zvijac JE, Wells ME, Botto-van Bemden A (2011) Long-term results of ulnar collateral ligament reconstruction in throwing athletes based on a hybrid technique. Am J Sports Med 39(2):342–347
73. Jackson TJ, Adamson GJ, Peterson A, Patton J, McGarry MH, Lee TQ (2013) Ulnar collateral ligament reconstruction using bisuspensory fixation: a biomechanical comparison with the docking technique. Am J Sports Med 41(5):1158–1164

74. Dines JS, ElAttrache NS, Conway JE, Smith W, Ahmad CS (2007) Clinical outcomes of the DANE TJ technique to treat ulnar collateral ligament insufficiency of the elbow. Am J Sports Med 35(12):2039–2044
75. Smith GR, Altchek DW, Pagnani MJ, Keeley JR (1996) A muscle-splitting approach to the ulnar collateral ligament of the elbow. Neuroanatomy and operative technique. Am J Sports Med 24:575–580
76. Cain EL Jr, Andrews JR, Dugas JR, Wilk KE, McMichael CS, Walter JC, Riley RS, Arthur ST (2010) Outcome of ulnar collateral ligament reconstruction of the elbow in 1281 athletes: results in 743 athletes with minimum 2-year follow-up. Am J Sports Med 38:2426–2434
77. Koh JL, Schafer MF, Keuter G, Hsu JE (2006) Ulnar collateral ligament reconstruction in elite throwing athletes. Arthroscopy 22:1187–1191
78. Paletta GA Jr, Wright RW (2006) The modified docking procedure for elbow ulnar collateral ligament reconstruction: 2-year follow-up in elite throwers. Am J Sports Med 34:1594–1598
79. Bowers AL, Dines JS, Dines DM, Altchek DW (2010) Elbow medial ulnar collateral ligament reconstruction: clinical relevance and the docking technique. J Shoulder Elbow Surg 19(2 Suppl): 110–117
80. Buehler MJ, Thayer DT (1988) The elbow flexion test: a clinical test for the cubital tunnel syndrome. Clin Orthop 233:213–216
81. Watson JN, McQueen P, Hutchinson MR (2014) A systematic review of ulnar collateral ligament reconstruction techniques. Am J Sports Med 42:2510–2516
82. Grana W (2001) Medial epicondylitis and cubital tunnel syndrome in the throwing athlete. Clin Sports Med 20:541–548
83. Cummins CA, Schneider DS (2009) Peripheral nerve injuries in baseball players. Phys Med Rehabil Clin N Am 20:175–193
84. Aldridge JW, Bruno RJ, Strauch RJ, Rosenwasser MP (2001) Nerve entrapment in athletes. Clin Sports Med 20(1):95–122
85. Rettig AC, Ebben JR (1993) Anterior subcutaneous transfer of the ulnar nerve in the athlete. Am J Sports Med 21:836–840
86. Petty DH, Andrews JR, Fleisig GS, Cain EL (2004) Ulnar collateral ligament reconstruction in high school baseball players: clinical results and injury risk factors. Am J Sports Med 32:1158–1164
87. Gabel GT, Amadio PC (1990) Reoperation for failed decompression of the ulnar nerve in the region of the elbow. J Bone Joint Surg 72A:213–219
88. Kleinman WB (1994) Revision ulnar neuroplasty. Hand Clin 10:461–477
89. Mackinnon SE, Novak CB (2007) Operative findings in reoperation of patients with cubital tunnel syndrome. Hand (N Y) 2:137–143
90. Norwood LA, Shook JA, Andrews JR (1981) Acute medial elbow ruptures. Am J Sports Med 9:16–19
91. Osbahr DC, Swaminathan SS, Allen AA, Dines JS, Coleman SH, Altchek DW (2010) Combined flexor-pronator mass and ulnar collateral ligament injuries in the elbows of older baseball players. Am J Sports Med 38:733–739
92. Vangsness CT, Jobe FW (1991) Surgical treatment of medial epicondylitis. Results in 35 elbows. J Bone Joint Surg Br 73:409–411
93. Brogdon BG, Crow NE (1960) Little Leaguer's elbow. Am J Roentgenol Radium Ther Nucl Med 83:671–675
94. Bennett GE (1959) Elbow and shoulder lesions of baseball players. Am J Surg 98:484–492
95. Osbahr DC, Chalmers PN, Frank JS, Williams RJ III, Widmann RF, Green DW (2010) Acute, avulsion fractures of the medial epicondyle while throwing in youth baseball players: a variant of Little League elbow. J Shoulder Elbow Surg 19:951–957
96. Adams JE (1973) Little league elbow. Calif Med 118:34–35
97. Torg JS, Pollack H, Sweterlitsch P (1972) The effect of competitive pitching on shoulders and elbows of preadolescent baseball players. Pediatrics 49:267–272
98. Gugenheim JJ Jr, Stanley RF, Woods GW, Tullos HS (1976) Little League survey: the Houston study. Am J Sports Med 4:189–200
99. Hang DW, Chao CM, Hang YS (2004) A clinical and roentgenographic study of Little League elbow. Am J Sports Med 32:79–84
100. Larson RL, Singer KM, Bergstrom R, Thomas S (1976) Little League survey: the Eugene study. Am J Sports Med 4:201–209
101. Farsetti P, Potenza V, Caterini R, Ippolito E (2001) Long-term results of treatment of fractures of the medial humeral epicondyle in children. J Bone Joint Surg Am 83:1299–1305
102. Lee HH, Shen HC, Chang JH, Lee CH, Wu SS (2005) Operative treatment of medial epicondyle fractures in children and adolescents. J Shoulder Elbow Surg 14:178–185
103. Gottschalk HP, Eisner E, Hosalkar HS (2012) Medial epicondyle fractures in the pediatric population. J Am Acad Orthop Surg 20:223–232
104. Lawrence JTR, Neeraj PM, Macknin J, Flynn JM, Cameron D, Wolfgruber JC, Ganley TJ (2013) Return to competitive sports after medial epicondyle fractures in adolescent athletes: results of operative and nonoperative treatment. Am J Sports Med 41:1152–1157
105. Patel NM, Ganley TJ (2012) Medial epicondyle fractures of the humerus: how to evaluate and when to operate. J Pediatr Orthop 32(Suppl 1):S10–S13
106. Edmonds EW (2010) How displaced are "nondisplaced" fractures of the medial humeral epicondyle in children? Results of a three-dimensional computed tomography analysis. J Bone Joint Surg Am 92(17):2785–2791
107. Pappas N, Lawrence JT, Donegan D, Ganley T, Flynn JM (2010) Intraobserver and interobserver agreement in the measurement of displaced humeral medial epicondyle fractures in children. J Bone Joint Surg Am 92(2):322–327
108. Schenck RC Jr, Goodnight JM (1996) Osteochondritis dissecans. J Bone Joint Surg Am 78(3):439–456
109. Bradley JP, Petrie RS (2001) Osteochondritis dissecans of the humeral capitellum: diagnosis and treatment. Clin Sports Med 20(3):565–590
110. Yadao MA, Field LD, Savoie FH III (2004) Osteochondritis dissecans of the elbow. Instr Course Lect 53:599–606
111. Yamaguchi K, Sweet FA, Bindra R, Morrey BF, Gelberman RH (1997) The extraosseous and intraosseous arterial anatomy of the adult elbow. J Bone Joint Surg Am 79(11):1653–1662
112. Takahara M, Ogino T, Takagi M, Tsuchida H, Orui H, Nambu T (2000) Natural progression of osteochondritis dissecans of the humeral capitellum: initial observations. Radiology 216(1):207–212
113. Mihara K, Tsutsui H, Nishinaka N, Yamaguchi K (2009) Nonoperative treatment for osteochondritis dissecans of the capitellum. Am J Sports Med 37(2):298–304
114. Ahmad C, El Attrache N (2006) Treatment of capitellar osteochondritis dissecans. Tech Should Elbow Surg 7(4):169–174
115. Minami M, Nakashita K, Ishii S et al (1979) Twenty-five cases of osteochondritis dissecans of the elbow. Rinsho Seikei Geka 14:805–810
116. Nelson DW, DiPaola J, Colville M, Schmidgall J (1990) Osteochondritis dissecans of the talus and knee: prospective comparison of MR and arthroscopic classifications. J Comput Assist Tomogr 14(5):804–808
117. Baumgarten TE, Andrews JR, Satterwhite YE (1998) The arthroscopic classification and treatment of osteochondritis dissecans of the capitellum. Am J Sports Med 26(4):520–523
118. Micheli LJ, Luke AC, Mintzer CM, Waters PM (2001) Elbow arthroscopy in the pediatric and adolescent population. Arthroscopy 17:694–699
119. Takeda H, Watarai K, Matsushita T, Saito T, Terashima Y (2002) A surgical treatment for unstable osteochondritis lesions of the humeral capitellum in adolescent baseball players. Am J Sports Med 30:713–717

120. Rahusen FT, Brinkman JM, Eygendaal D (2006) Results of arthroscopic debridement for osteochondritis dissecans of the elbow. Br J Sports Med 40:966–969
121. Brownlow HC, O'Connor-Read LM, Perko M (2006) Arthroscopic treatment of osteochondritis dissecans of the capitellum. Knee Surg Sports Traumatol Arthrosc 14:198–202
122. Yamamoto Y, Ishibashi Y, Tsuda E, Sato H, Toh S (2006) Osteochondral autograft transplantation for osteochondritis dissecans of the elbow in juvenile baseball players: minimum 2-year follow-up. Am J Sports Med 34:714–720
123. Nobuta S, Ogawa K, Sato K, Nakagawa T, Hatori M, Itoi E (2008) Clinical outcome of fragment fixation for osteochondritis dissecans of the elbow. Ups J Med Sci 113:201–208
124. Iwasaki N, Kato H, Ishikawa J, Masuko T, Funakoshi T, Minami A (2009) Autologous osteochondral mosaicplasty for osteochondritis dissecans of the elbow in teenage athletes. J Bone Joint Surg Am 91:2359–2366
125. Mihara K, Suzuki K, Makiuchi D, Mishinaka N, Yamaguchi K, Tsutsui H (2010) Surgical treatment for osteochondritis dissecans of the humeral capitellum. J Shoulder Elbow Surg 19:31–37
126. Jones KJ, Wiesel BB, Sankar WN, Ganley TJ (2010) Arthroscopic management of osteochondritis dissecans of the capitellum: mid-term results in adolescent athletes. J Pediatr Orthop 30:8–13
127. de Graaff F, Krijnen MR, Poolman RW, Willems WJ (2011) Arthroscopic surgery in athletes with osteochondritis dissecans of the elbow. Arthroscopy 27(7):986–993
128. Andrews JR, Wilk KE, Satterwhite YE, Tedder JI (1993) Physical examination of the thrower's elbow. J Orthop Sports Phys Ther 17(6):296–304
129. Miller CD, Savoie FH 3rd (1994) Valgus extension injuries of the elbow in the throwing athlete. J Am Acad Orthop Surg 2: 261–269
130. Moskal MJ (2001) Arthroscopic treatment of posterior impingement of the elbow in athletes. Clin Sports Med 20:11–24
131. Rahusen FT, Brinkman JM, Eygendaal D (2009) Arthroscopic treatment of posterior impingement of the elbow in athletes: a medium-term follow-up in 16 cases. J Shoulder Elbow Surg 18(2):279–282
132. Eygendaal D, Safran MR (2006) Postero-medial elbow problems in the adult athlete. Br J Sports Med 40(5):430–434
133. Reddy AS, Kvitne RS, Yocum LA, ElAttrache NS, Glousman RE, Jobe FW (2000) Arthroscopy of the elbow: a long-term clinical review. Arthroscopy 16(6):588–594
134. Andrews JR, Heggland EJH, Fleisig GS, Zheng N (2001) Relationship of ulnar collateral ligament strain to amount of medial olecranon osteotomy. Am J Sports Med 29(6):716–721
135. Kamineni S, Hirahara H, Pomianowski S, Neale PG, O'Driscoll SW, ElAttrache N, An KN, Morrey BF (2003) Partial posteromedial olecranon resection: a kinematic study. J Bone Joint Surg Am 85-A(6):1005–1011
136. Kamineni S, ElAttrache NS, O'Driscoll SW, Ahmad CS, Hirohara H, Neale PG, An KN, Morrey BF (2004) Medial collateral ligament strain with partial posteromedial olecranon resection. A biomechanical study. J Bone Joint Surg Am 86-A(11):2424–2430
137. Inagaki K (2013) Current concepts of elbow-joint disorders and their treatment. J Orthop Sci 18:1–7
138. Greaney RB, Gerber FH, Laughlin RL, Kmet JP, Metz CD, Kilcheski TS, Rao BR, Silverman ED (1983) Distribution and natural history of stress fractures in U.S. Marine recruits. Radiology 146(2):339–346
139. Stafford SA, Rosenthal DI, Gebhardt MC, Brady TJ, Scott JA (1986) MRI in stress fracture. AJR Am J Roentgenol 147(3): 553–556
140. Daffner RH, Pavlov H (1992) Stress fractures: current concepts. AJR Am J Roentgenol 159(2):245–252
141. Anderson MW, Greenspan A (1996) Stress fractures. Radiology 199(1):1–12
142. Spitz DJ, Newberg AH (2002) Imaging of stress fractures in the athlete. Radiol Clin North Am 40(2):313–331
143. Sofka CM (2006) Imaging of stress fractures. Clin Sports Med 25(1):53–62
144. Osbahr DC, Bedi A, Conway JE (2012) Olecranon stress fractures. In: Dines JS, ElAttrache NS, Altchek DW, Andrews JR, Yocum LA, Wilk KE (eds) Sports medicine in baseball. Lippincott Williams and Wilkins, Philadelphia
145. Nakaji N, Fujioka H, Tanaka J, Sugimoto K, Yoshiya S, Fujita K, Kurosaka M (2006) Stress fracture of the olecranon in an adult baseball player. Knee Surg Sports Traumatol Arthrosc 14(4): 390–393
146. Mamanee P, Neira C, Martire JR, McFarland EG (2000) Stress lesion of the proximal medial ulna in a throwing athlete. A case report. Am J Sports Med 28(2):261–263
147. Blake JJ, Block JJ, Hannah GA, Kan JH (2008) Unusual stress fracture in an adolescent baseball pitcher affecting the trochlear groove of the olecranon. Pediatr Radiol 38(7):788–790
148. Nuber GW, Diment MT (1992) Olecranon stress fractures in throwers. A report of two cases and a review of the literature. Clin Orthop Relat Res 278:58–61
149. Suzuki K, Minami A, Suenaga N, Kondoh M (1997) Oblique stress fracture of the olecranon in baseball pitchers. J Shoulder Elbow Surg 6(5):491–494
150. Rettig AC, Wurth TR, Mieling P (2006) Nonunion of olecranon stress fractures in adolescent baseball pitchers: a case series of 5 athletes. Am J Sports Med 34(4):653–656
151. Hutchinson DT, Horwitz DS, Ha G, Thomas CW, Bachus KN (2003) Cyclic loading of olecranon fracture fixation constructs. J Bone Joint Surg Am 85-A(5):831–837
152. Paci JM, Dugas JR, Guy JA, Cain EL, Fleisig GS, Hurst C, Wilk KE, Andrews JR (2012) Cannulated screw fixation of refractory olecranon stress fractures with and without associated injuries allows a return to baseball. Am J Sports Med 41(2):306–312
153. Carofino BC, Santangelo SA, Kabadi M, Mazzocca AD, Browner BD (2007) Olecranon fractures repaired with FiberWire or metal wire tension banding: a biomechanical comparison. Arthroscopy 23(9):964–970
154. Sahajpal D, Wright TW (2009) Proximal ulna fractures. J Hand Surg Am 34(2):357–362

Biceps and Triceps Tendon Lesions

26

Enrico Guerra, Daniele Fabbri, Graziono Bettelli, Alessandro Marinelli, Michele Cavaciocchi, and Roberto Rotini

26.1 Distal Biceps Lesions

The treatment of lesions of the distal tendon of the humeral biceps still represents an open chapter of orthopaedic surgery, with questions related to the anatomy of the native tendon and to the epidemiology, diagnosis and therapeutic options.

In the face of a complete lesion of the distal biceps tendon, surgical reconstruction is advised, especially in athletes; however, the best surgical approach, fixation technique and rehabilitation protocol are still matters of debate in the literature.

26.1.1 Epidemiology

Breakage of the distal biceps tendon is a rare occurrence that causes an evident loss of strength of the limb. Its incidence in the general population is about 0.9–1.8 new cases per 100,000 persons every year [1], representing about 3 % of all biceps lesions. It is more frequent in men (>95 % of cases) aged between 30 and 60 years. The dominant limb is most frequently involved (86 % vs. 14 %). In smokers and in subjects taking steroids the relative risk is increased about 7.5 times. People typically incurring this type of lesion are bodybuilders, sportsmen or hard manual labourers [1].

26.1.2 Etiology

The pathogenic mechanisms of distal biceps lesions are not completely clear. As for all tendon lesions, advancing age and increased functional demands contribute to normal time-related degeneration. In theory, there may be a basic vascular impairment, which is secondarily affected by mechanical factors. Seiler et al. [2], in a cadaveric study, have observed a less vascularised zone about 2.14 cm long between the musculotendinous portion and the bone insertion. Branches of the radial artery supply the proximal portion, and electronic microscopy has shown focal degenerative zones in the hypovascularised area, which may act as a *locus minoris resistentiae*, favouring failure. In the same study, serial tomographies at different degrees of pronosupination have also shown that during maximum pronation the distance between the lateral edge of the ulna and the radial tuberosity is 48 %, lower than during maximum supination. Moreover, during pronation of the wrist the tendon fills about 85 % of the radioulnar space at the level of the tuberosity; therefore, repetitive pronosupination movements can cause a mechanical impingement (Fig. 26.1). Although the real causes of distal biceps tendon lesions have not yet been defined, the conclusions drawn from this study confirm the validity of the vascular and of the mechanical theory [2]. The limited space available for the tendon can be restricted even more by chronic bursitis or by hypertrophy of the radial tuberosity [3, 4]. In the same way, during work or sport activity, the proximal migration of the forearm muscle bellies in the course of a "power grip" increases the tension at the lacertus fibrosus. This in turn modifies the distal biceps geometry, distorting it in the ulnar direction and increasing the forces placed on the tendon [5].

26.1.3 Clinical Presentation

Usually, the patients refer to the tendon rupturing during sudden extension of the flexed elbow. This movement produces an eccentric muscular contraction followed by pain in the antecubital fossa. Also, an audible pop is often felt before the pain sensation and the appearance of an ecchymosis. After the acute pain has decreased, a feeling of discomfort or pain of minor intensity is felt during elbow flexion and more severely during active supination. At the clinical observation

E. Guerra (✉) • D. Fabbri • G. Bettelli • A. Marinelli
M. Cavaciocchi • R. Rotini
SC Shoulder and Elbow Unit, Rizzoli Orthopaedic Institute, Bologna, Italy
e-mail: enrico.guerra@ior.it

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_26,

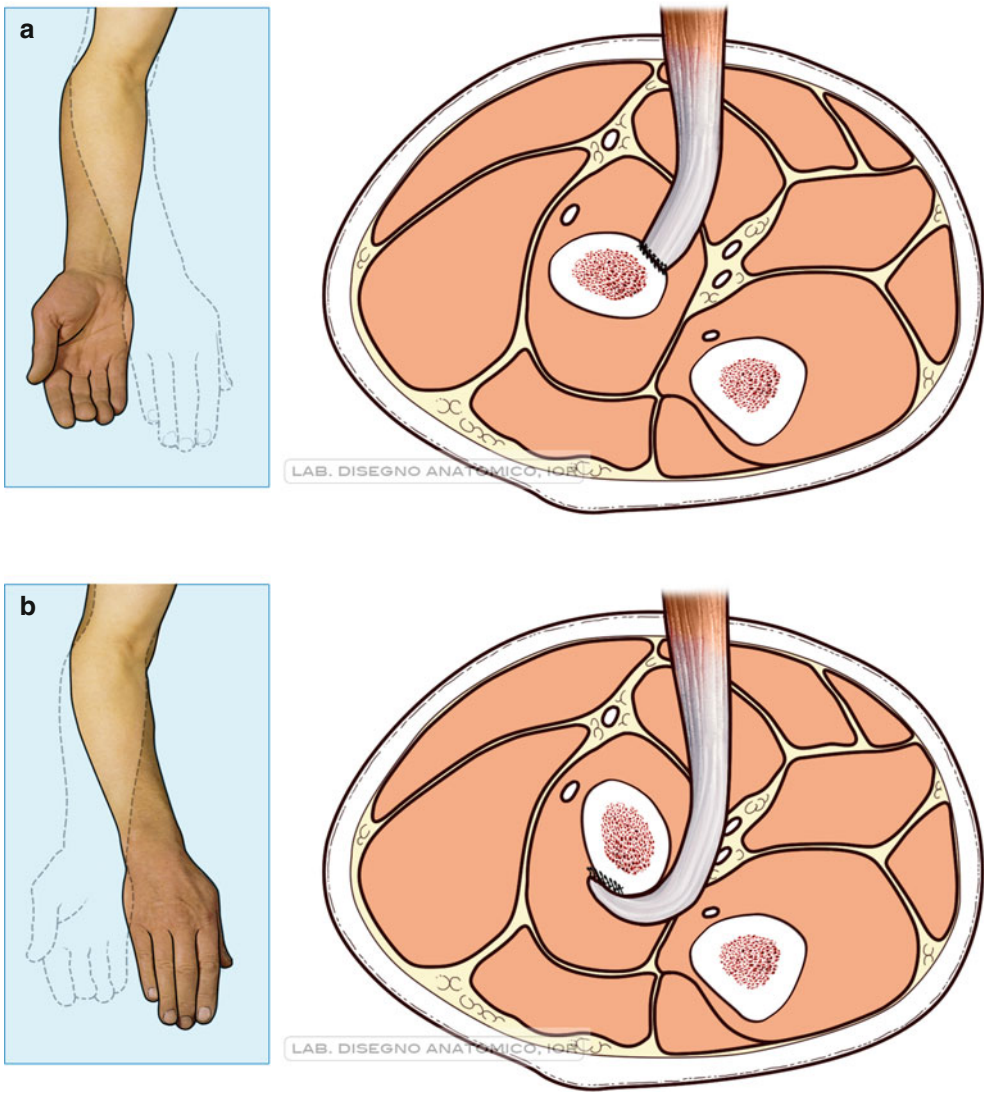


Fig. 26.1 Anatomical relations of the distal biceps tendon with the radius and ulna in supination (**a**) and pronation (**b**) (School of Anatomical Drawing, University of Bologna, Rizzoli Orthopaedic Institute)

the normal biceps contour disappears and an odd profile appears, called "Popeye deformity". This type of deformity is more evident in patients with a well-developed muscular mass. At the acute stage patients do not complain about restrictions of flexion, extension, pronation and supination. An evaluation of strength proves difficult, as pain affects the clinical results. At the subacute stage the patient refers to easy fatigability during consecutive movements of supination together with elbow flexion. Although this is the typical clinical history, tendon rupture can also go unrecognised, especially when the lacertus fibrosus remains intact. If the lesion is misdiagnosed and not adequately treated, pain gradually decreases over time. About 21 days after trauma most of the patients feel no pain or functional limitation, which is revealed only when resuming working or the recreational activities formerly performed. A chronic lesion is more difficult to treat and the results are worse [6, 7]; therefore, early clinical diagnosis is important. Several authors have made the effort to define a clinical test with good sensitivity and specificity for distal biceps lesions. Ruland et al. [6] have suggested a test similar to the Thomson test for Achille's tendon lesions, with "squeezing" of the biceps muscle belly while observing whether or not spontaneous forearm supination takes place. The test is positive when the movement is absent. Thomson [7] have described the "hook test". The examiner tries to "hook" the tendon in the antecubital fossa with the elbow during 90° flexion. When this is not possible there is a high probability that the tendon is broken. The results of their study indicate sensitivity and specificity of about 100 %, compared with 92 % sensitivity and 85 % specificity for MRI in the same patients. The authors stress the importance of grasping the lateral part of the tendon to avoid the medially placed lacertus fibrosus provoking misdiagnosis of the lesion.

In our experience, the clinical examination has proved essential. The history of the traumatic event may be evident,

while sometimes it suggests more subtle lesions with sequential two-stage ruptures, without any evident sprain and with a history of recurrent or chronic pain. An accurate evaluation looking for a popeye sign is important; however, a thick subcutaneous fat layer can hide it. Care also has to be taken in palpating the muscle mass of the retracted biceps. As time since the traumatic event passes, the deformity becomes more evident and the muscle tone improves. A less retracted but evidently flaccid muscle indicates an acute lesion. The hook test remains the best, to be performed several times, with the elbow during flexion, during different degrees of pronation and supination. In any case, only a comparison with the opposite limb allows clinical examination to indicate the diagnosis.

A differential diagnosis with other possible causes of pain in the antecubital fossa is always necessary; namely, biceps tendon tendinitis, a partial tendon lesion, bursitis, posterior interosseous nerve syndrome and entrapment of the lateral cutaneous nerve of the forearm.

26.1.4 Diagnostic Examinations

26.1.4.1 X-Ray

Plain radiograms have no diagnostic value in distal biceps tendon lesions, but they are useful for a general assessment and to rule out possible concomitant bone lesions. No cases have been reported with radiographically visible bony avulsions. When present, calcific degeneration of the biceps insertion may be demonstrated; however, this does not imply a tendon lesion.

26.1.4.2 MRI

Magnetic resonance imaging is the gold standard examination for identifying these lesions. Usually, it is performed with the arm in extension and in the axial plane, owing to the longitudinal course of the tendon in the antecubital fossa. This type of view allows easier diagnosis of partial tears that are otherwise difficult to identify [8, 9].

Giuffre and Moss [9] described a technique for optimal patient positioning, allowing examination of the whole length of the tendon from the musculotendinous junction to the insertion on the radial tuberosity in at least one if not two sections. The patient is placed inside the scanner in prone position with the arm to be examined elevated overhead (shoulder in 180° abduction with the arm beside the head, the elbow in 90° flexion, the forearm in supination and the thumb pointing upwards). This position is known under the acronym FABS (flexion, abduction, supination). The optimal equipment includes a shoulder coil to be placed around the elbow, so that it lies in the middle of the magnets. The FABS sequence is added to the conventional positions of the biceps.

26.1.4.3 Echography

Ultrasound imaging has been extensively used for the diagnosis of distal biceps rupture, as it has a lower cost and greater availability than MRI. This technique allows the continuity of the distal tendon or changes in its size to be assessed [9]. Dynamic ultrasound examination can help in distinguishing between partial and complete rupture. Unfortunately, this is an operator-dependent diagnostic technique, less reproducible than MRI, and it loses both sensitivity and specificity in obese patients, or those with highly developed muscular masses, or those with previous surgical procedures in the same area. Echography, therefore, requires an operator with very good musculoskeletal pathology training. Bird [10] developed the use of the pronator teres acoustic window, which can provide better visualisation of the bicipital insertion.

26.1.5 Treatment

26.1.5.1 Conservative

The first studies on these lesions suggested that conservative treatment might be the best therapeutic option, allowing excellent functional recovery as early as 4 weeks after the event [10–12]. In 1985, Baker and Bierwagen [13] were the first to examine the changes in strength in conservatively treated patients compared with those who had received surgical treatment. The results reported in their study show a 40 % loss of strength during supination, a 79 % loss in supination cycles, and a 30 % loss of strength during flexion, both absolute and cyclic, for non-surgical patients compared with those undergoing surgery. Morrey et al. [14] reported similar conclusions, with a 40 % loss during supination and a 30 % loss during flexion. These studies have stressed how the bicipital action occurs primarily during supination and only secondarily during elbow flexion. More recently, two studies conducted by Chillemi et al. and by Hetsroni et al. [15, 16] have shown that even from the view point of clinical scoring systems (European Society for Surgery of the Shoulder and Elbow Score) and of subjective and objective perceptions (isokinetic tests) the results are superior after surgical treatment to those after conservative treatment. The most recent trial, with the wider pool of patients and the longest follow-up, has been conducted by Freeman's group [17]. They showed after conservative treatment supination strength equal to 74 % and flexion strength equal to 88 % compared with the contralateral healthy side, and the results were better when the lesion had occurred in the non-dominant arm (83 % vs 60 % for supination strength). In the same study, it was once again demonstrated that the main loss of strength after conservative treatment concerned supination—74 % vs 101 % of surgical patients—and it was confirmed that the gap increased in the case of lesions of the dominant arm. Disabilities of the arm, shoulder and hand (DASH) scores,

however, were inferior (lower impairment) in conservatively treated patients than in those who had received surgery. The authors' conclusion is that the results in subjective outcomes should be attributed to the absence of surgery and to the post-operative protocol.

Nowadays, we therefore restrict conservative treatment to sedentary patients who have no need for full absolute strength and who do not perform repetitive movements, mainly during pronosupination, patients who cannot be scheduled for surgery and those who voluntarily elect to accept loss of muscular strength and endurance rather than facing the risks of surgery and the long recovery time. Conservative treatment includes temporary immobilisation, the use of drugs for pain control and then rehabilitation cycles. Resuming previous manual activities is allowed as soon as the patient feels ready, usually no longer than 1 month after the tendon rupture. Even though cases are reported in which there is persistent discomfort or pain in the antecubital fossa, the results are generally satisfactory.

26.1.5.2 Anatomical or Non-anatomical Repair

When the advantages of the surgical treatment as the best therapeutic option for the lesions of the distal biceps were understood, the question arose of whether an anatomical or a non-anatomical repair might be better. The first option involves the reinsertion of the tendon at the radial tuberosity, whereas the second one is aimed at suturing the tendon to the brachialis muscle. The literature review by Ratanen and Orava about 147 patients [18] describes a higher level of clinical satisfaction in the patients operated on using an anatomical procedure than in those subjected to non-anatomical surgery (60 %) or conservative treatment (14 %). More recently, Klonz et al. [19] evaluated using an isokinetic test the real functional recovery of patients operated on using both techniques, detecting similar values in flexion strength recovery, but a definitely lower supination strength in patients with a non-anatomical reconstruction. On the other hand, patients subjected to an anatomical repair had a higher rate of heterotopic ossifications.

Nowadays, when the indication for surgery is selected, the anatomical reinsertion of the tendon is preferred. Suturing the tendon to the brachialis muscle is a salvage option for the few cases in which taking the biceps tendon back to the radial tuberosity proves impossible at surgery.

26.1.5.3 Partial Ruptures

Partial rupture of the biceps tendon is a rare occurrence and few cases are described in the literature. The diagnosis in these cases is more difficult, as patients complain about vague symptoms and also clinical signs are less clear. Patients often describe a clinical history of vague pain in the antecubital fossa and when a loss of strength during supination and flexion is present, it is less evident than in patients with a total lesion. In these cases, performing an MRI is more useful, as it allows the extension of the lesion to be defined and in many cases the collateral aspects of tendonitis, or tenosynovitis, or bursitis to be shown.

The first treatment in these cases is the control of pain using anti-inflammatory drugs, which can be associated with a physiotherapy protocol aimed at stretching the anterior structures of the elbow. In the case of persistent pain and functional limitations, surgical treatment is warranted. The authors who described this type of lesion suggest transforming the partial lesion into a complete lesion and to reinsert the tendon at its anatomical site [19–23].

26.1.5.4 Chronic Ruptures

When the lesion dates back to 6–8 weeks or more, then it should be defined as chronic. These lesions are the most difficult to treat, mainly because of the muscular retraction. An intact lacertus fibrosus provides the conditions for the possibility of reinserting the biceps tendon into the radius, because if it breaks, proximal tendon retraction, soft-tissue adhesions, tendon shortening and resorption preclude reconstruction with the native tendon. The first suggested options were conservative treatment [11–24] and suture to the brachialis muscle [25–29], with results similar to those formerly described; namely, a partial loss of flexion strength and a more consistent loss of supination strength. Sotereanos et al. [30] tried to repair chronic lesions in one surgical step, separating the tendon from soft-tissue adhesions as the first step, then sectioning the lacertus fibrosus and lengthening the tendon by making radial micro-incisions when traction alone was ineffective. Unfortunately, it was not possible to take the tendon back to the radial tuberosity in a number of cases and in these patients a graft was employed. This extensively studied technique can include the use of fascia lata, or the ligament advanced reinforcement system (LARS), or semitendinosus, or homologous tendons, and none of the types of graft proved mechanically superior to the others.

26.1.5.5 Surgical Technique

The choice of the surgical approach for a lesion of the distal tendon of the biceps brachii is still a source of controversy among surgeons, because there is no scientific evidence of the superiority of one technique over the other. The single incision with anterior approach is historically associated with a higher chance of neurological lesions, probably the consequence of a larger dissection and the need for wider retractors to visualise the tendon and to reinsert it on its anatomical footprint. With the aim of avoiding these complications, Boyd and Anderson developed the two-incision technique, limiting the anterior tissue dissection. However, its safety was initially impaired by an increased chance of radio-ulnar synostoses and injuries of the posterior interosseous nerve.

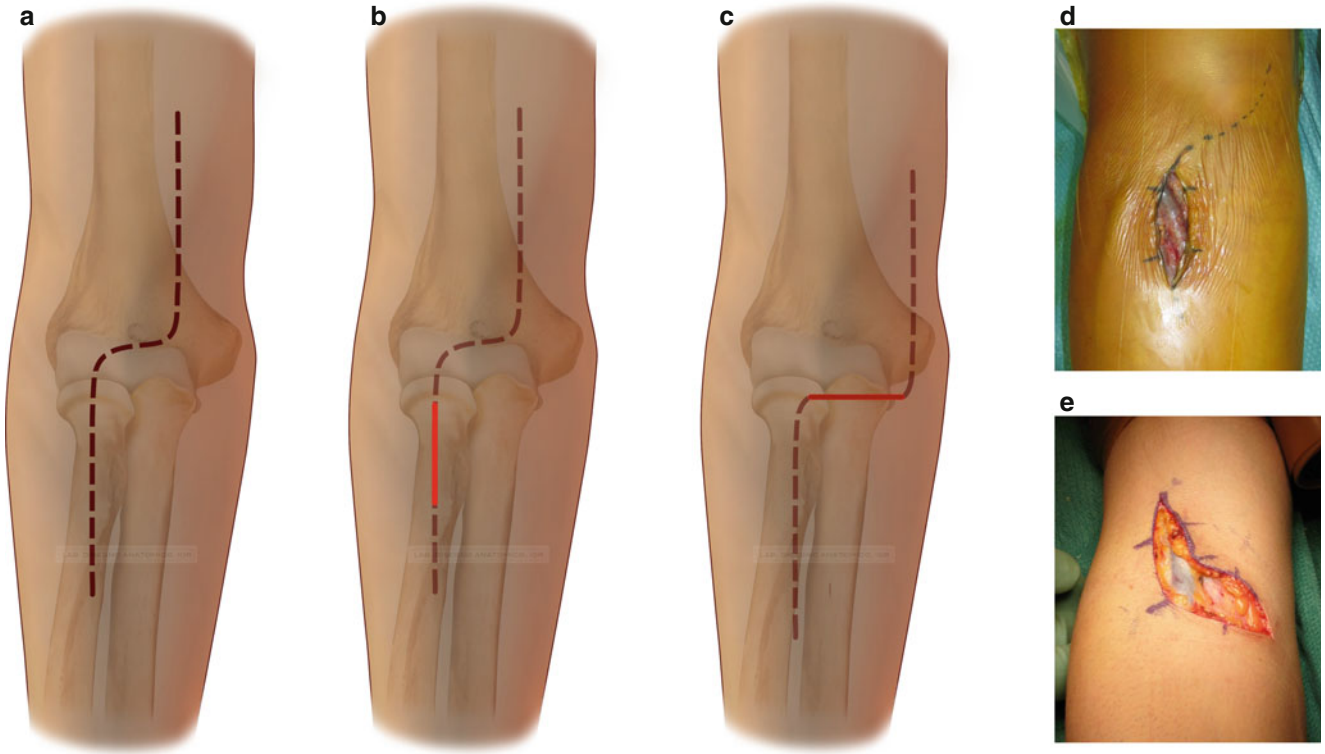


Fig. 26.2 Surgical approaches. Extended italic S incision (**a**) and less invasive incisions, longitudinal (**b**) and transverse (**c**). If needed, both of these incisions can be extended proximally or distally (**d**, **e**) (School of anatomic drawing, University of Bologna, Rizzoli Orthopaedic Institute)

Over the years these two techniques have been improved. Morrey et al. [31] refined the two-incision approach, improving the posterior muscle splitting to avoid elevating the ulnar periosteum and therefore the risk of radio-ulnar synostoses. The improvements in the one-incision approach have been related to the introduction of fixation systems (anchors, endobutton, interference screws), which allowed the anterior exposure to be limited.

Regional anaesthesia is generally preferred, unless the anaesthesiologist or the patient prefers otherwise. The patient is placed supine on the surgical table with the upper limb in about 90° abduction over a hand surgery stand in supination. The surgeon and the assistant are on the opposite sides of the limb, while the scrub nurse is in front of the stand on the same side of the body as the surgeons. A tourniquet is placed at the upper arm (it is advisable to exsanguinate the limb only after identifying and isolating the biceps tendon). After preparing the skin the surgical field is set up.

Tendon Identification and Evaluation of the Lesion

The first incision described was shaped like an italic S centered on the cubital crease, with isolation of the main vascular and nervous trunks and anatomical dissection down to the radial tuberosity where the biceps tendon had to be reinserted. Over the years, the need for more limited surgical aggressiveness has led to the development of less invasive techniques.

The anterior incision only needs to be a few centimetres long (Fig. 26.2) The anterior cubital aspect has several transverse skin folds that become evident with elbow flexion. If a transverse incision is selected, following one of these folds will allow a better cosmetic result. This corresponds to the central part of the italic S incision. The choice of the level of the incision is important. If it is more proximal it allows better exposure of the biceps at the myotendinous junction, but it impairs exposure of the radial tuberosity. It is therefore preferable to make a more distal incision, about 2 cm from the main cubital crease. The biceps tendon can be identified by elbow flexion and subcutaneous tissue dissection; at the same time, by elbow extension, it is possible to expose the proximal radius without excessive soft tissue traction by the retractors. It is always possible, if needed, to enlarge the approach with a proximal or distal longitudinal extension of the incision.

In our experience we have changed the line of incision, from the formerly described transverse line to a 3- to 4-cm longitudinal incision centred over the radial head. Elbow flexion and the use of retractors allows the same view as permitted by the transverse incision for tendon identification, while the anatomical dissection for exposure of the radial tuberosity proves more simple and safe, although it is more difficult in muscular and tall patients. It is possible, in any case, to enlarge the exposure by a proximal extension of the incision following the line of the italic S (Fig. 26.2).

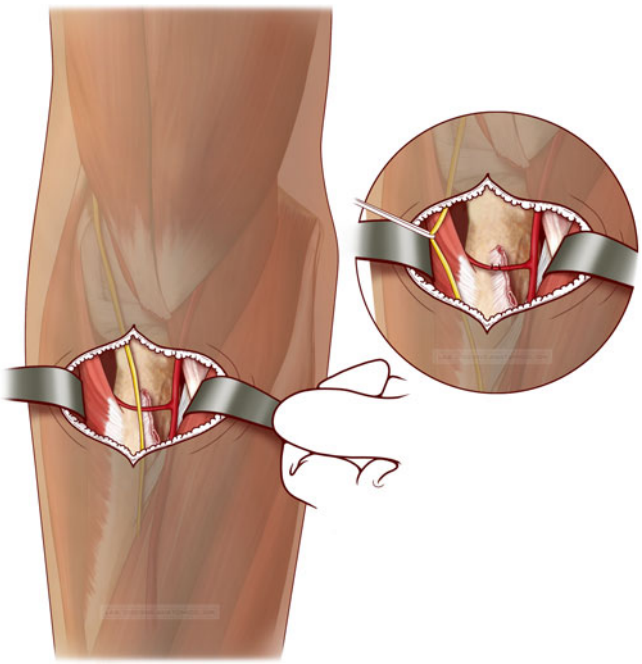


Fig. 26.3 Exposure of the proximal radius. The cephalic vein is moved laterally; the superficial branch of the radial nerve is identified, protected and moved laterally. The recurrent vessels have to be isolated and ligated before approaching the radial tuberosity in the depth of the field (School of Anatomical Drawing, University of Bologna, Rizzoli Orthopaedic Institute)

In the subcutaneous layer the superficial cubital veins are situated. Whenever possible, these veins are isolated and respected; however, if they are an obstacle for surgical exposure they have to be ligated, as their size does not allow simple cauterisation. The cephalic vein usually lies over the lateral cutaneous nerve of the forearm. It is advisable to identify this nerve, isolating it using a vessel loop (Fig. 26.3) and placing it in the lateral part of the surgical field. It is now possible to safely pay attention to the search for and exploration of the biceps tendon.

Multiple conditions may occur. The tendon may only be partially detached from the tuberosity. In this case the medial lacertus fibrosus is intact and the tendon lies in its anatomical site inside its sheath, although it has lost its tension. It is easy to follow it distally and to test its resistance to traction using a hook. In most cases a mild traction makes the lesion complete and the tendon comes out. If on the contrary it resists traction, the option to abort surgery can be considered (after careful examination of the myotendinous junction to rule out a proximal rupture). However, if the tendon is loose, it is better to expose the tuberosity and transform the partial lesion into a total lesion by detaching the few residual fibres.

When the lesion is complete, the sheath is empty, and inside there is serous haematic fluid, which is more abundant if the injury is recent. The tendon stump can usually be found proximally, underneath the biceps muscle belly. When the lacertus fibrosus is also broken, the tendon can be found folded in itself and migrated proximally by several centimetres. In these cases, especially if surgery is performed 15–20 days after the injury, it is wise to extend the incision proximally and perform a very careful dissection of the tendon from the surrounding scar tissue, which can show the same colour and consistency as the tendon. Once the tendon is isolated, its distal portion is taken out of the surgical wound and the degenerated fibres at the extremity are removed.

Tendon Repair at the Radial Tuberosity

The surgical techniques can be separated into two main groups, the single anterior approach and the double anterior and posterior approach.

Single Anterior Approach Technique

The radial tuberosity should be carefully exposed. The literature reports bony anchors, or endobutton, or interference screws as fixation devices alternative to the transosseous suture. A transosseous repair with a bony trough, as described for the double incision technique, is not easy to perform with an anterior single approach and is less strong in cadaver tests. The mechanically stronger methods are the endobutton, which unfortunately is associated with a risk of damage to the posterior interosseous nerve, and fixation by an interference screw, which carries the risk of late bony resorption of the proximal radius. Apart from evaluation of which is the mechanically stronger method, none of the techniques has shown clinical superiority over the others. We therefore selected the anterior single approach technique to use bony anchors, as we believe that they represent the best compromise among mechanical strength, surgical reproducibility and risk of complications.

After the lateral cutaneous nerve of the forearm has been identified and moved laterally, the deep dissection is carried out. The superficial veins are ligated or cauterised and the brachioradialis and round pronator muscle bellies are retracted laterally and medially. The recurrent vessels are so exposed that they correspond to the level of the radial tuberosity and have to be carefully dissected and ligated to gain access to the bone (Fig. 26.3).

Forceful forearm supination allows access to the tuberosity, from which the tendon fibre remnants and scar tissue need to be cleaned. The working field is deep; a lever can be used on the medial side, while attention should be paid to the lateral side, where the posterior interosseous nerve lies inside the supinator muscle close to the bone. It is therefore prudent to only place a retractor on this side, which applies less pressure than a lever (Fig. 26.4).

The bony surface is roughened to promote tendon-to-bone healing. The holes for the anchors have to be carefully prepared so that they are inserted correctly and they are guaranteed to hold. It is our practice to drill

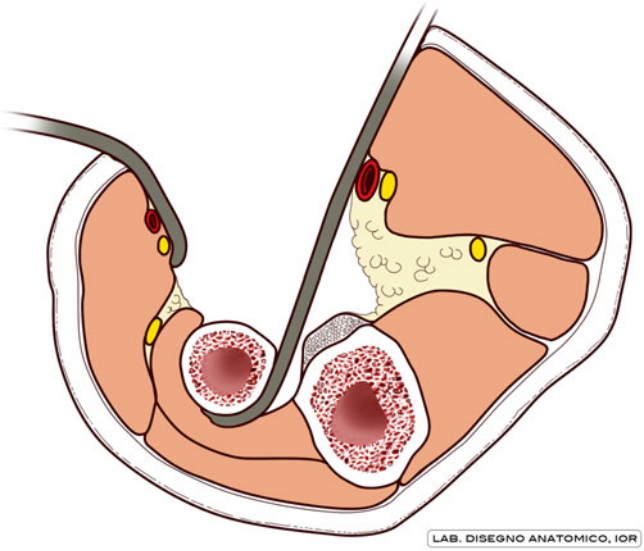


Fig. 26.4 Exposure of the proximal radius, axial section (School of Anatomical Drawing, University of Bologna, Rizzoli Orthopaedic Institute)

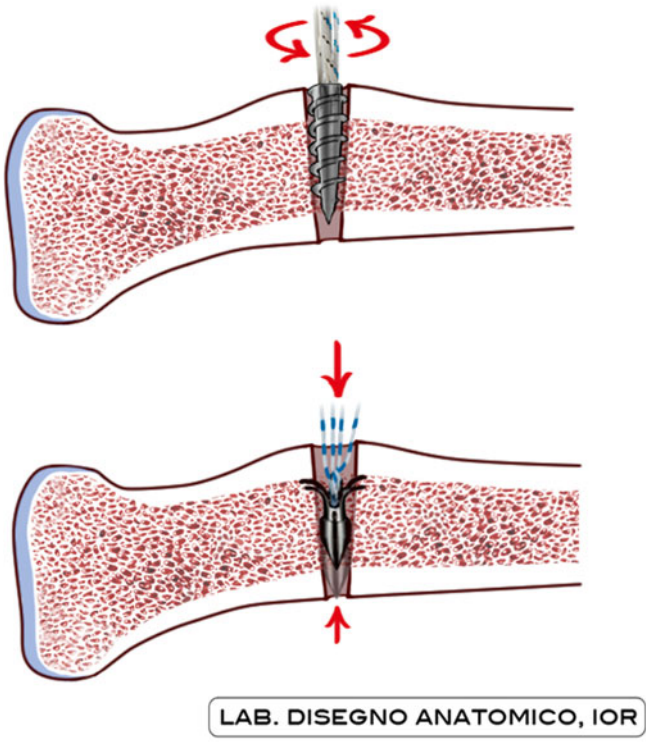


Fig. 26.5 Surgical technique of anchor introduction. Drilling both cortices is useful to allow the correct introduction of the tip of the anchor. The proximal hole should be enlarged to prevent cortical fractures, which may compromise the pull-out strength of the anchors (School of Anatomical Drawing, University of Bologna, Rizzoli Orthopaedic Institute)

both cortices using a 1.5-mm drill; the proximal hole is then enlarged by a 2.5-mm drill to insert a 5-mm anchor (Fig. 26.5). To reduce the risk of cortical fracture, a 10 cm gap is left between the first and the second anchor. Our preference is to use threaded anchors, as they can be easily removed if needed. Careful lavage is performed after each step to remove as much of the bony debris as possible, and with the same intent we advise using drill bits rather than smooth bits.

The tendon is grasped by the sutures of the anchors (Fig. 26.6). The proximal anchor is connected to the medial part and the distal anchor to the lateral part of the tendon. Each anchor bears two sutures. By one head, a Krackow-type suture is placed over a 4-cm length of the tendon, while both heads of the second suture are passed in a mattress configuration into the distal head of the tendon. With this method the first suture allows a stronger hold on the tendon, while the second suture increases the contact surface between the tendon and the bony surface.

We have no experience of repair using the endobutton (Fig. 26.7), except on cadavers. This technique has proved to be biomechanically stronger, but in surgical practice it has been reported to be more difficult and dangerous for the posterior interosseous nerve.

We used interference screws (Fig. 26.7) in only a few cases. The literature reports cases of osteolysis around resorbable screws and for this reason we chose polyetheretherketone (PEEK) screws. Even in this case the surgical technique must be meticulous. The hole for the screw has to be large enough, but if the grip is loosened, it becomes difficult to switch to another fixation technique.

Double Anterior and Posterior Approach Technique

This is a reliable and low-cost technique that has not been modified since its original description by the Sotereanos et al. [30].

The suggested anterior incision is transverse and proximal. Once the tendon is retrieved as formerly described, it has to be cleaned and then grasped by two high-resistance sutures with Krackow-type stitches over a 4-cm length. An extensive anterior dissection is not necessary; the peritoneum should be smoothly followed until the radial tuberosity. A curved haemostat should be passed very closely to the radius while the forearm is placed in flexion and complete pronation. The haemostat is advanced until it reaches the subcutaneous layer posteriorly (Fig. 26.8). The author draws attention to the tool direction, which should follow the posterior radial cortex to limit the possible muscular injury, predisposing to heterotopic ossifications.

A short incision is made over the tip of the haemostat. The radial tuberosity is exposed through a smooth split of the extensor and supinator muscles. Placing the elbow in flexion moves the tip of the haemostat proximally, pointing at the proximal end of the muscular split, which should therefore be opened distal to the tip of the surgical tool. It is now possible

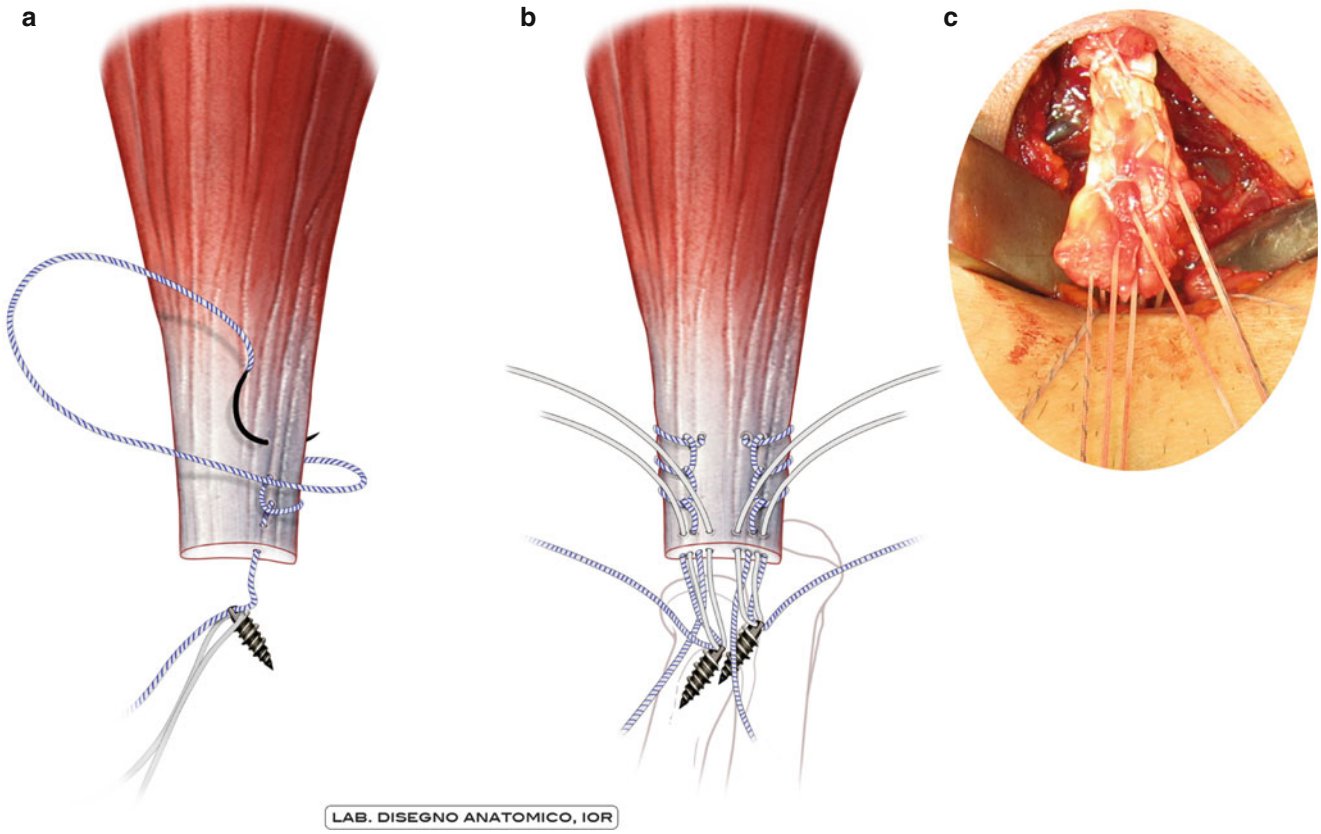


Fig. 26.6 Drawings (**a**, **b**) and surgical detail (**c**) of the passage of the anchor sutures inside the tendon (School of Anatomical Drawing, University of Bologna, Rizzoli Orthopaedic Institute)

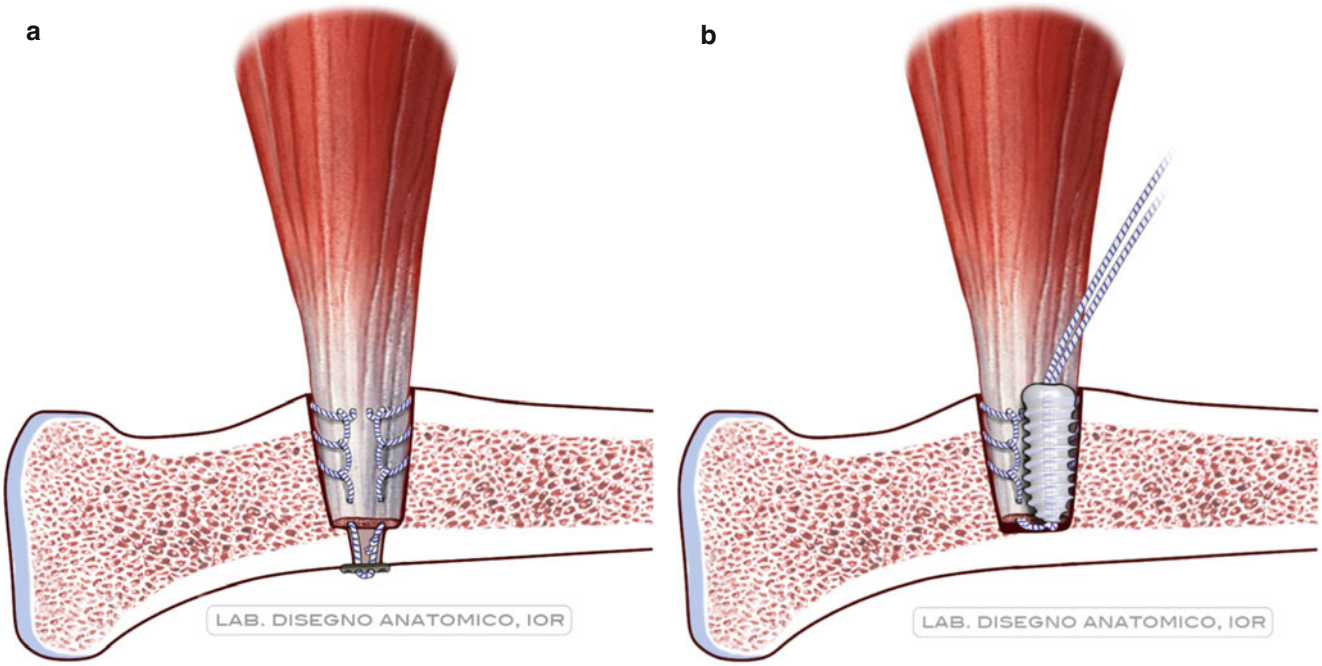


Fig. 26.7 Tendon repair using the endobutton system (**a**) and by the interference screw (**b**) (School of Anatomical Drawing, University of Bologna, Rizzoli Orthopaedic Institute)

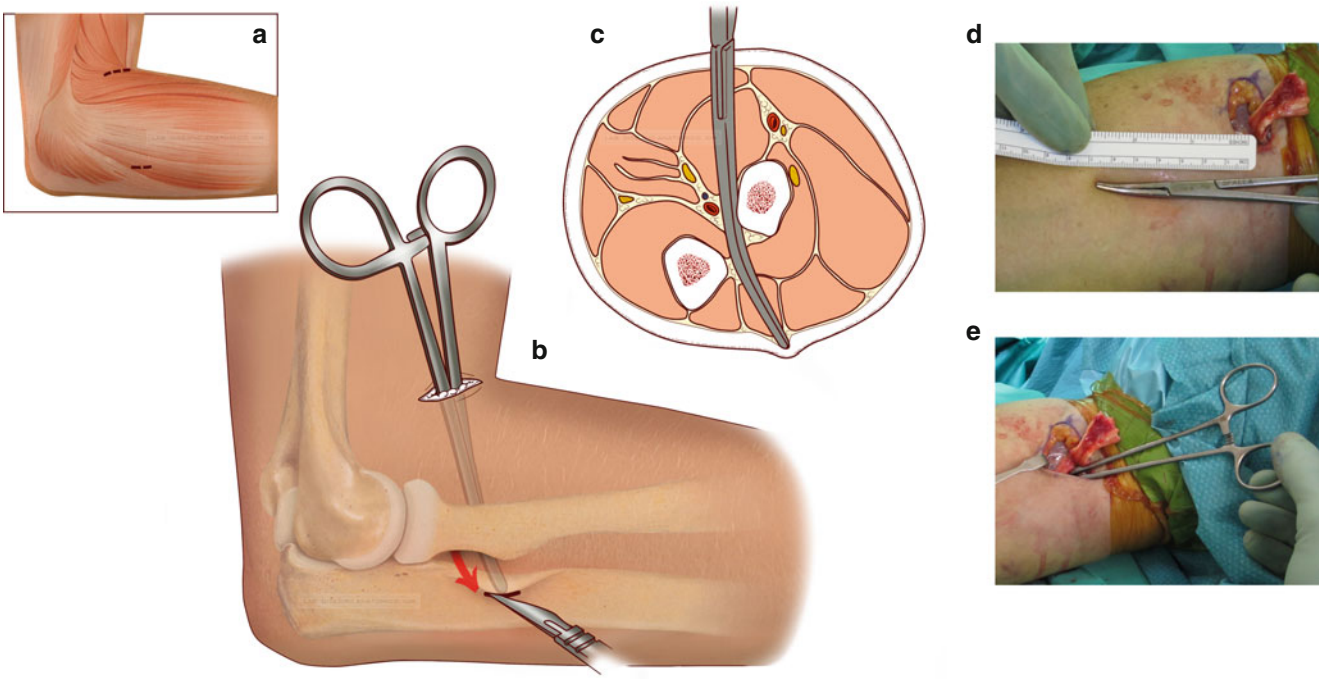


Fig. 26.8 Drawings (**a**–**c**) and surgical details (**d**, **e**) of the blind passage of the curved haemostat (The drawings reproduce the author's [30]) original pictures, by the School of Anatomical Drawing, University of Bologna, Rizzoli Orthopaedic Institute)

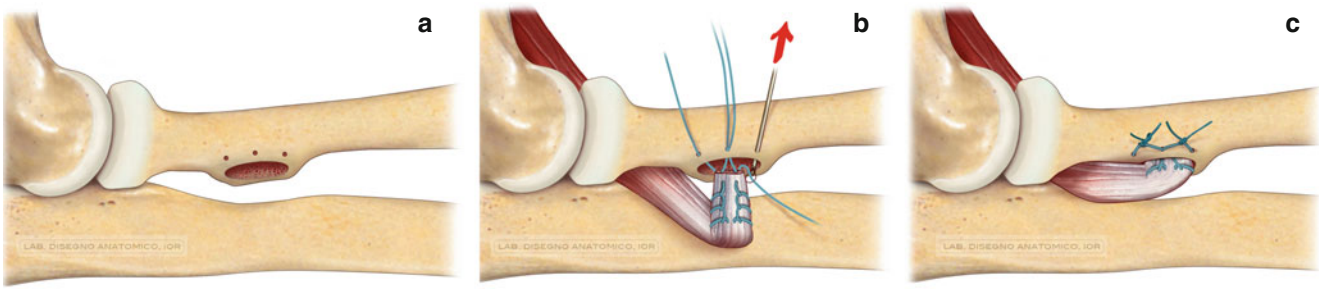


Fig. 26.9 Tendon fixation using the transosseous technique. The trough should be as large as the tendon end and the distance between the holes behind the osseous ridge should not be less than 7 mm, with convergent directions (**a**). Passing the sutures is easier if done by a needle and a shuttle suture (**b**). Tightening the stitches should pull and stabilise the tendon inside the trough (**c**) (The drawings reproduce the Author's [30] original pictures, by the School of Anatomical Drawing, University of Bologna, Rizzoli Orthopaedic Institute)

to prepare the bony trough inside, which the accommodates the tendon end and the three holes for the passage of the sutures (Fig. 26.9). To dig the trough we prefer to make multiple drill holes and then remove the small bony bridges using a rongeur, while the use of a high speed burr should be restricted to the final smoothing of the edges. This precaution, together with frequent lavage, is aimed at removing as much of the bony debris as possible, which may a contributing cause of heterotopic ossifications. The Sotereanos et al. [30] recommends a 7- to 8-mm gap between the holes to leave the bony bridges strong enough. We drill the three holes using a 1.6-mm K-wire, allowing passage of an 18G needle. The holes should have convergent directions towards the trough; this trick proves useful for the passage and sliding of the sutures.

To bring the tendon to its site, the use of a ligament passer (designed for anterior cruciate ligament repair using the "over the top" technique) allows an easier and less traumatic passage (Fig. 26.10), again with the goal of limiting the risk of soft tissue injuries.

Now, the four heads of the two sutures have to be passed from inside the bony trough to the outside, through the three holes drilled behind the tuberosity. This can be easily carried out using a needle technique, with a simple straight syringe 18G needle and a shuttle monofilament 0 size suture, as described for meniscal suture repair (Fig. 26.9). Tightening the sutures pulls the tendon inside the bony trough, which therefore has to be wide enough in relation to the tendon size.

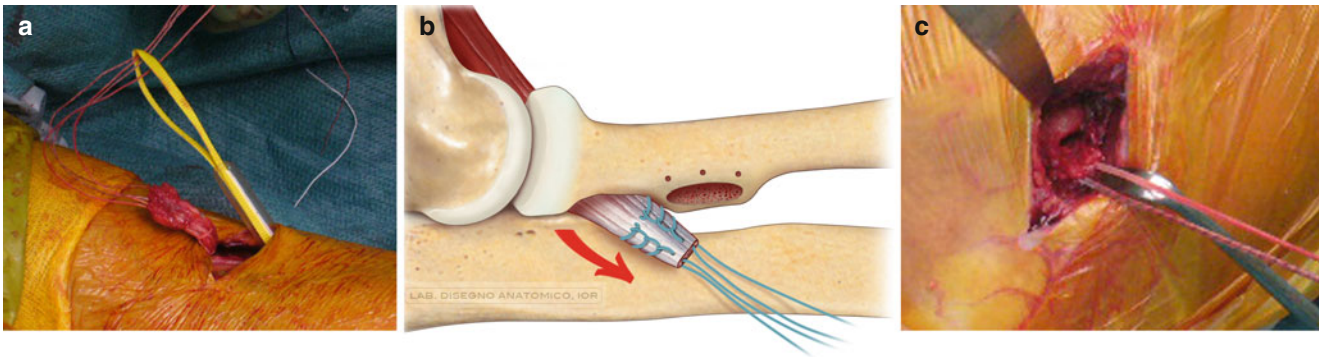


Fig. 26.10 Surgical detail and graphic rendering of the tendon transfer by the ligament passer (**a**) between the radius and ulna (**b**), up to the osseous trough and over (**c**) (School of Anatomical Drawing, University of Bologna, Rizzoli Orthopaedic Institute)

In our Unit both of the techniques described above are performed. As a general rule, in acute cases, when the tendon canal is easily identifiable, the double incision technique is simple and has the advantage of avoiding extensive anterior dissection, which is more aggressive in muscular and tall patients.

On the contrary, when the organising haematoma precludes easy and smooth identification of the path to the radial tuberosity, we select the single approach technique. It is our opinion that careful anatomical dissection is more prudent than a blind and forceful approach.

26.1.5.6 Rehabilitation Treatment

Conservative Treatment of the Acute Lesion

The arm is immobilised in a brace in adduction with the elbow in 90° flexion for 7–10 days. As soon as allowed by pain, the patient begins passive and active assisted rehabilitation to recover the normal range of motion (ROM), which is generally not compromised by the injury. At about 30 days, exercises for strength in supination and flexion can be started, in addition to a gradual resumption of normal activities.

Post-surgical Treatment After Tendon Repair

Rehabilitation is definitely more complex after surgical repair, as no evidence is available in the literature showing whether or not a more aggressive protocol is more or less effective than a prudent one [31].

We therefore report the data from our experience, leading us to a slight reduction of the time of immobilisation and prognosis. The following is the approximate schedule that we give to our patients.

- Brace with the elbow in 90° flexion and a pronosupination block (in slight supination) for 30 days
- Start mobilisation exercises on the 7th day
  - Active extension (up to 60° in the first few days, then gradually increase) and passive flexion (unlimited)
  - Passive pronosupination with the elbow in 90° of flexion
  - The target is a complete passive ROM at about 1 month after surgery
- Thirty days after surgery, the brace is progressively unlocked (20° per week) and the pronosupination block is removed. Active and passive mobilisation exercises in flexion–extension and in pronation–supination are started (no load, best if in brace).
- Sixty days after surgery, the brace is definitively removed and active progressive rehabilitation started
- One hundred and twenty days after surgery, working activities are resumed

Together with this protocol, which is only approximate and suggested, hydrokinesis therapy is advised at every step. This therapy, when available, provides favourable conditions for the recovery of patients.

26.1.6 Results

A number of authors have reported their experiences. In each paper data are available on the functional recovery and the incidence of complications. These data are considerably different in the various articles, which precludes the possibility of drawing any conclusions based on scientific evidence. Summarising data from the literature it is possible to state that:

- The best functional recovery relative to strength is obtained with surgical treatment, even though the results can be affected by the possible complications, which in some authors' experience can be frequent, in up to 31 % of cases [15]
- Conservative treatment has the advantages of fast recovery and of the absence of complications. The loss of strength is sometimes surprisingly well tolerated by the patients. The loss of strength and endurance during supination is certainly less well tolerated when it is specifically necessary because of the type of work or sports activities.

- The incidence of complications is dramatically lower if the patients are operated on during the first 15 days after injury.
- In general, the most frequent complication is heterotopic ossification; a preventive therapy with indometacin is therefore advisable [32], irrespective of the type of surgical technique.
- The most probable complications are heterotopic ossifications for the double incision repair and neurological lesions (lateral cutaneous nerve of the forearm and posterior interosseous nerve) for the single incision repair.
- The recurrence of tendon breakage is extremely rare and is the consequence of new traumatic events.

The most effective recent study is that by Grewal et al. [33], who compared in a prospective randomised trial 47 patients treated using the single incision technique and 44 using the double incision repair. The authors concluded that the clinical and functional results of these two techniques are equivalent and the incidence of complications is comparable. What make the difference in choosing the type of surgical approach are therefore the experience and the personal preference of the surgeon.

26.2 Triceps Lesions

Lesions of the triceps tendon are very rare, with few cases reported in the literature. A higher incidence has been recorded in male patients (2:1), with an age distribution from 13 to 72 years [34]. Anzel et al. reviewed 1,014 tendon lesions treated at the Mayo Clinic. Eighty-five percent of the injuries involved the upper limb, but only 8 (about 0.8 % of cases) involved the triceps, and 4 were open injuries [35]. In a paper reporting lesions occurring in the National Football League, only 21 cases of triceps rupture were recorded over a 6 year period [36]. On this basis, it is difficult to assess the true incidence of distal triceps injuries; it may be underestimated in light of the possible increase in the number of these events due to an increase in the practice of contact sports and an increase in steroid consumption in professional athletes [34, 36]. Vidal et al. first and then Kibuule and Fehringer reported how these lesions can occur in the paediatric age, in patients with recently closed or partially closed growth cartilage [37, 38].

26.2.1 Mechanism of Lesion

The main cause of triceps tendon injury is a fall on the elbow during extension [37–42]. Other mechanisms reported in literature include a posterior blow and lifting a weight [40, 42–45], the use of a baseball bat [46], road accidents [67], throwing sports [48–50, 65], volleyball, boxing and shotput [51, 52]. With the exception of direct trauma, in the other conditions the biomechanics of the lesion is always similar; namely, a triceps contraction with the elbow in flexion, together with a sudden deceleration and with eccentric overload of the tendon [37, 38, 41–44, 46, 47, 53, 54]. A number of factors can modify the structural integrity of the tendon and lower the maximum tolerable load: diabetes mellitus, osteogenesis imperfecta, rheumatoid arthritis and systemic lupus erythematosus, corticosteroids (both systemic or in local injections), anabolic steroids, secondary hyperparathyroidism in chronic renal insufficiency, and ciprofloxacin administration [43, 45, 46, 53, 55–57, 66]. A possible familial predisposition has also been hypothesised [53]. However, most of the cases of triceps rupture take place in a healthy tendon [39]. Triceps tendon rupture usually happens at the olecranon insertion [38, 43, 46, 54, 58, 59], in many cases with an osseous avulsion [60]. More rarely, the lesion involves the musculotendinous junction and/or the muscle belly [37, 47]. Associated lesions have been reported; namely, radial head fracture [61], rupture of the ulnar collateral ligament [43], ulnar nerve compression due to haematoma [62] and, in the more severe cases, a compartmental syndrome with radial nerve compression [46, 47] and fracture of the distal humerus [63].

26.2.2 Clinical Picture

Patients with a triceps tendon lesion often complain about a sudden and sharp pain in the region of the lesion, with impaired flexion and extension of the elbow and a palpable, painful gap at the olecranon insertion of the triceps. When the clinical aspects are evident the diagnosis is easy and reliable. The loss of strength can be compensated for by the anconeus action if the patient is asked to extend his or her elbow and keep it below the level of the shoulder. It is therefore appropriate during the clinical examination to test triceps strength with the elbow above the head, first against gravity and then against resistance. This test is usually positive and reliable except in the case of partial lesions [48]. An area of ecchymosis may be present around the olecranon. A modified Thompson test has also been described, performed with the patient in prone position with the elbow in flexion and the forearm hanging down the table. Squeezing the triceps muscle belly should cause passive elbow extension if the tendon is intact [68, 69].

26.2.3 Diagnosis

Standard X-rays are the first diagnostic procedure. They can disclose the “fleck sign”, that is, a small bony fragment of the triceps insertion dislocated at the level of the distal humerus

[64, 67, 70], in addition to any possible associated fractures. Ultrasound examination is highly reliable in an expert's hands; however, the gold standard in diagnosis is MRI [35, 70].

26.2.4 Anatomy

The triceps originates from the dorsal aspect of the arm and is composed of the lateral, long and medial heads. A variant fourth head, the dorso-epitrochlear, consists of a muscle between the triceps and latissimus dorsi. The triceps tendon footprint is dome-shaped and covers 78 % of the width of the olecranon. The tendon and a band of muscle fibres extend laterally, distally (over the anconeus) and medially (over the medial epicondyle) making a superficial layer that overlays the entire olecranon [58].

26.2.5 Treatment

Partial triceps tendon ruptures have historically been treated non-operatively, although this is not a consensus opinion. The non-operative treatment consisting of posterior splinting in 30° flexion for 15–21 days generally resulted in full ROM and subjective strength recovery within 3–9 months Special attention should be paid to the type of sports gesture to lessen the incidence of tendonitis proximal and distal to the elbow. Failure of healing leads to delayed surgical intervention, making complications higher and results worse. This is the reason why many authors suggest surgery when the lesion involves more than 50–75 % of the tendon [43, 48].

When the tear is complete, surgery is necessary except for a few selected cases (e.g. elderly patients with few functional demands). Several repair methods have been described: transosseous sutures, interference screws, bony anchors with simple or complex configuration (bridge-equivalent sutures) with the aim of reproducing an adequate footprint. The paucity of cases reported in the literature and the lack of prospective trials preclude the possibility of stating which technique is superior to the others. Simple transosseous suture is therefore the preferred option as it is simple and reproducible, certainly the only treatment in skeletally immature patients.

26.2.6 Surgical Technique

A posterior curved incision avoiding the tip of the olecranon satisfactorily exposes the tendon insertion. If the haematoma is abundant, ulnar nerve isolation may be advisable to lessen the chances of late neuropathy. The lesion often involves the tendon below the superficial aponeurotic layer, which has to be incised. The tendon end is refreshed and using two or more high-resistance sutures is inserted into the olecranon through simple or crossed bony tunnels [35], using the same technique of needle and shuttle suture as that described above. The repair is performed with the elbow in about 30° flexion and its strength is tested by placing the elbow in flexion before suturing the superficial layers. It is also important to retrieve the superficial tendon tissue to completely cover the olecranon (Fig. 26.11).

When a bony fragment is present (flake sign), if it is of adequate size, fixation using a cannulated 3.5-mm screw and washer may be added to the transosseous suture. Shortening by 2 cm causes a 40 % loss of strength and it is therefore mandatory to avoid this.

In chronic cases, the loss of tissue needs compensation. Sanchez-Sotelo and Morrey and Van Riet et al. [43, 46] described an anconeus rotation flap technique. The proximal anconeus–lateral triceps flap is lifted from the ulna, mobilised over the olecranon, and reattached to the extensor mechanism when the anconeus is of good quality and continuous with the lateral triceps. Possible alternatives can be autologous or homologous tendon grafts (palmaris longus, gracilis, fascia lata, homologous Achilles tendon, etc.). The results of these salvage techniques are intuitively worse. For the patients who were treated in our Unit we chose the Achilles tendon with a bony strut to be fixed to the olecranon [71]; in our opinion, this is the best compromise available nowadays.

26.2.7 Results

Strength recovery is slow but effective in all the reported series and ranges from 63 to 100 %, depending mainly on the time span from injury to surgery, with an average restriction of passive ROM of 10° extension. Almost all of the patients have been able to resume their former activities.

26.2.8 Rehabilitation

As for the biceps lesion, in the face of a lack of literature evidence we report our rehabilitation protocol.

- Immobilisation for 2 weeks at 30° elbow flexion
- Progressive flexion block at 30°, 45°, 60° and 90° by week 5. Full flexion by week 6
- Passive rehabilitation 2 weeks after surgery
- Active extension by week 6
- Extension strengthening by 12 weeks
- Unrestricted activity at 5–6 months

Functional recovery can certainly last longer, with strength increasing up to 1 year after surgery.

Never make promises…

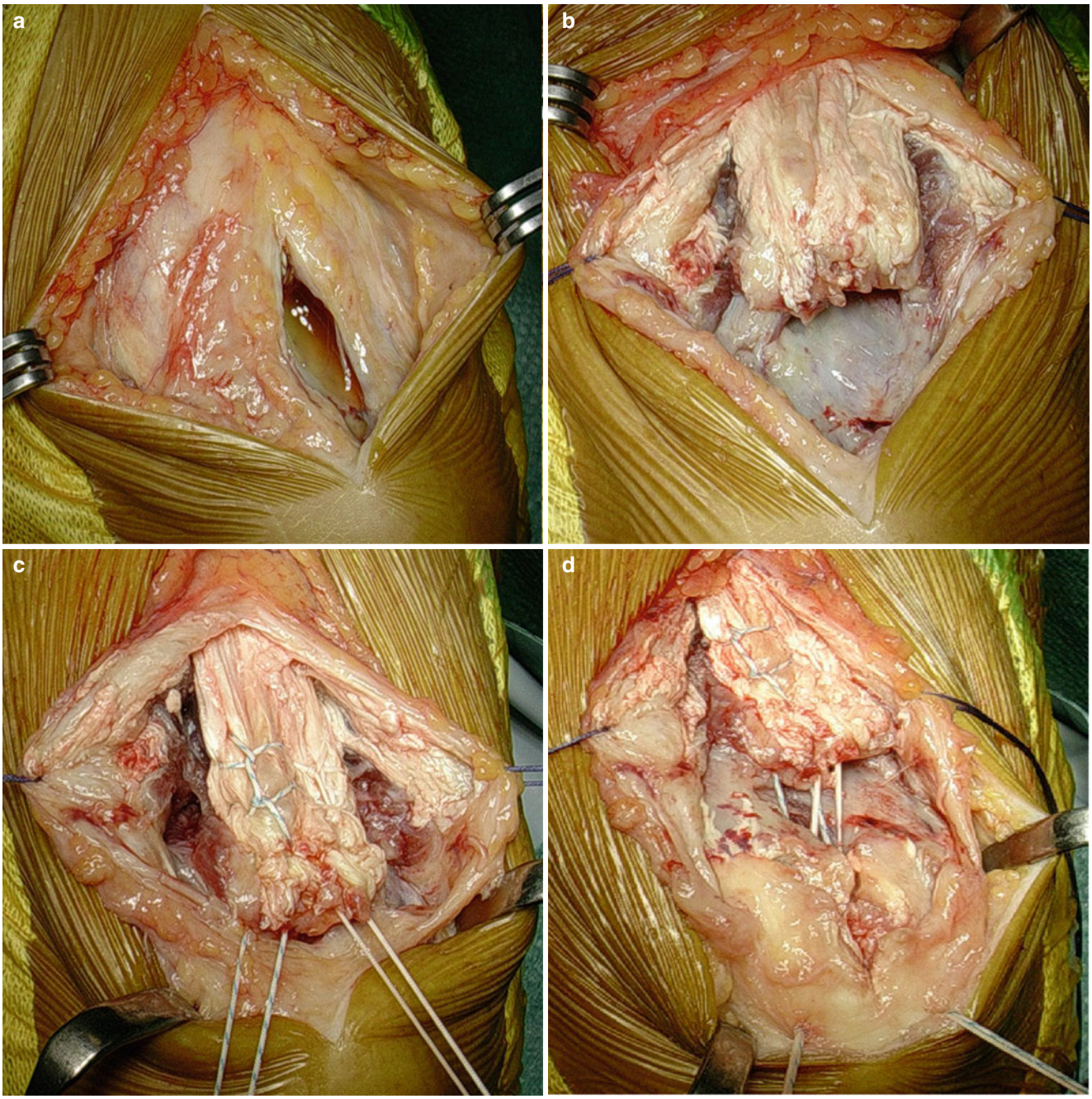


Fig. 26.11 Surgical steps (**a**–**e**) for transosseous triceps reinsertion: opening of the superficial layer (**a**); finding the deeper and stronger triceps tendon (**b**); grasping the tendon using reinforced sutures (**c**); passing the sutures through holes into the olecranon (**d**); closing of the osteosutures and multiple stitches accessories to reinforce the repair and recreate the footprint (**e**); closing the superficial triceps layers (**f**)

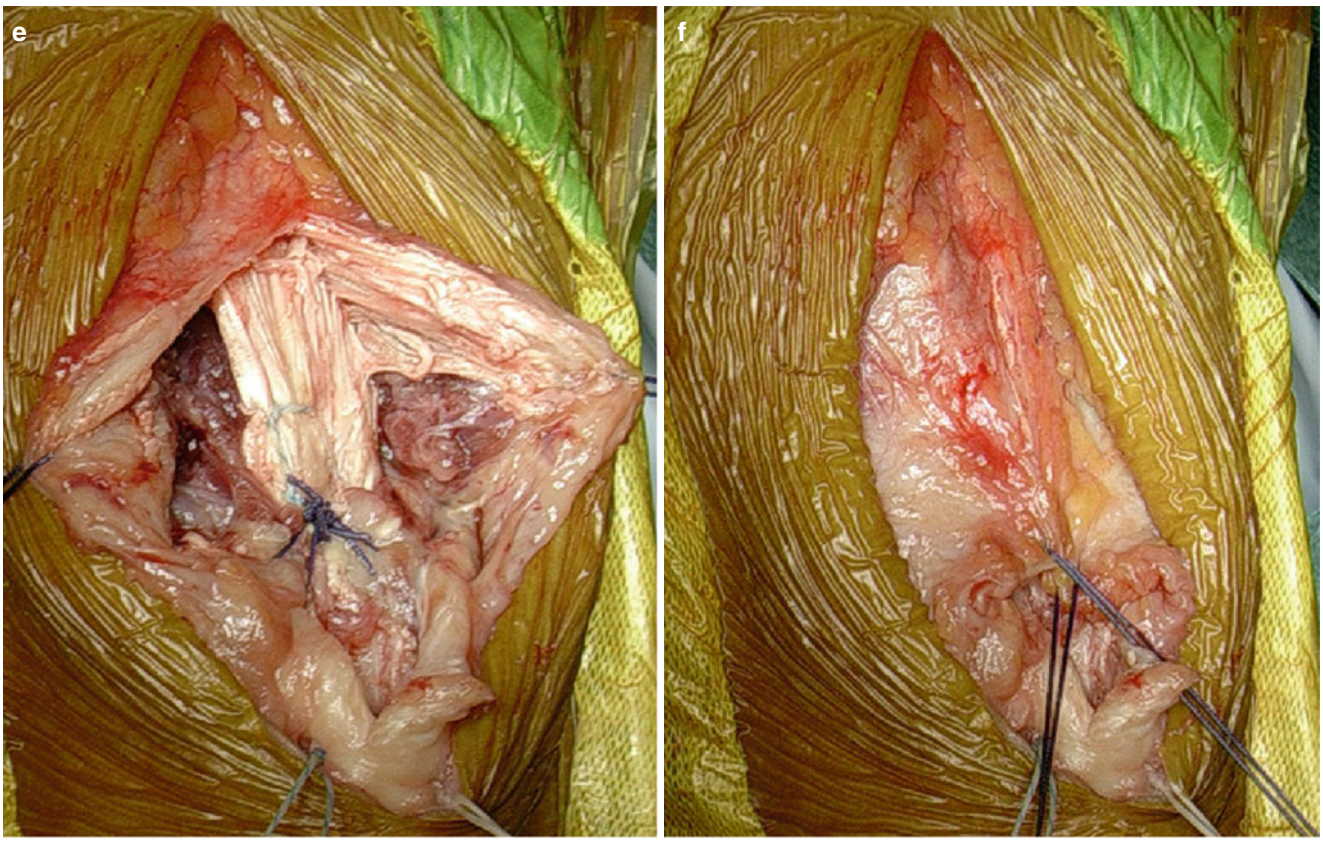


Fig. 26.11 (continued)

References

1. Safran MR, Graham SM (2002) Distal biceps tendon ruptures: incidence, demographics, and the effect of smoking. Clin Orthop Relat Res 404:275–283
2. Seiler JG 3rd, Parker LM, Chamberland PD, Sherbourne GM, Carpenter WA (1995) The distal biceps tendon. Two potential mechanisms involved in its rupture: arterial supply and mechanical impingement. J Shoulder Elbow Surg 4:149–156
3. Johnson DC, Allen AA (2001) Biceps and triceps tendon injury. In: Altchek DW, Andrews JR (eds) The athlete's elbow. Lippincott Williams & Wilkins, Hagerstown, pp 105–120
4. Davis WM, Yassine Z (1956) An etiological factor in tear of the distal tendon of the biceps brachii; report of two cases. J Bone Joint Surg Am 38-A(6):1365–1368
5. Bain GI, Johnson LJ, Turner PC (2008) Treatment of partial distal biceps tendon tears. Sports Med Arthrosc 16(3):154–161
6. Ruland RT, Dunbar RP, Bowen JD (2005) The biceps squeeze test for diagnosis of distal biceps tendon ruptures. Clin Orthop Relat Res 437:128–131
7. Thompson TC (1962) A test for rupture of the tendo achillis. Acta Orthop Scand 32:461–465
8. Chew ML, Giuffre BM (2005) Disorders of the distal biceps brachii tendon. Radiographics 25(5):1227–1237
9. Giuffre BM, Moss MJ (2004) Optimal positioning for MRI of the distal biceps brachii tendon: flexed abducted supinated view. AJR Am J Roentgenol 182(4):944–946
10. Bird S (2006) A new method for ultrasound evaluation of the distal biceps Brachii tendon. Ultrasound Med Biol 32(5S):294 [Official proceedings of the 11th congress of the World Federation for Ultrasound in Medicine and Biology, May 2006]
11. Dobbie RP (1941) Avulsion of the lower biceps brachii tendon: analysis of fifty-one previously unreported cases. Am J Surg 51:662–683
12. Carroll RE, Hamilton LR (1967) Rupture of the biceps brachii tendon: conservative methods of treatment. J Bone Joint Surg 49:1016
13. Baker BE, Bierwagen D (1985) Rupture of the distal tendon of the biceps brachii. Operative versus non-operative treatment. J Bone Joint Surg Am 67:414–417
14. Morrey BF, Askew LJ, An KN, Dobyns JH (1985) Rupture of the distal tendon of the biceps brachii. A biomechanical study. J Bone Joint Surg Am 67:418–421
15. Chillemi C, Marinelli M, De Cupis V (2007) Rupture of the distal biceps brachii tendon: conservative treatment versus anatomic reinsertion—clinical and radiological evaluation after 2 years. Arch Orthop Trauma Surg 127:705–708
16. Hetsroni I, Pilz-Burstein R, Nyska M, Back Z, Barchilon V, Mann G (2008) Avulsion of the distal biceps brachii tendon in middle-aged population: is surgical repair advisable? A comparative study of 22 patients treated with either nonoperative management or early anatomical repair. Injury 39:753–760
17. Freeman CR, McCormick KR, Mahoney D, Baratz M, Lubahn JD (2009) Nonoperative treatment of distal biceps tendon ruptures compared with a historical control group. J Bone Joint Surg Am 91:2329–2334
18. Rantanen J, Orava S (1999) Rupture of the distal biceps tendon. A report of 19 patients treated with anatomic reinsertion, and a meta-analysis of 147 cases found in the literature. Am J Sports Med 27:128–132
19. Klonz A, Loitz D, Wöhler P, Reilmann H (2003) Rupture of the distal biceps brachii tendon: isokinetic power analysis and complications after anatomic reinsertion compared with fixation to the brachialis muscle. J Shoulder Elbow Surg 12:607–611

20. Bourne MH, Morrey BF (1991) Partial rupture of the distal biceps tendon. Clin Orthop Relat Res 271:143–148
21. Nielsen K (1987) Partial rupture of the distal biceps brachii tendon. A case report. Acta Orthop Scand 58(3):287–288
22. Rokito AS, McLaughlin JA, Gallagher MA et al (1996) Partial rupture of the distal biceps tendon. J Shoulder Elbow Surg 5(1):73–75
23. Vardakas DG, Musgrave DS, Varitimidis SE et al (2001) Partial rupture of the distal biceps tendon. J Shoulder Elbow Surg 10(4):377–379
24. Kelly EW, Steinmann S, O'Driscoll SW (2003) Surgical treatment of partial distal biceps tendon ruptures through a single posterior incision. J Shoulder Elbow Surg 12(5):456–461
25. Klonz A, Eggers C, Reilmann H (1998) Proximal and distal biceps tendon rupture – an indication for surgery. Unfallchirurg 101:735–739 [German]
26. Friedmann E (1963) Rupture of the distal biceps brachii tendon: report of 13 cases. JAMA 184:60–63
27. Kron S, Satinsky B (1954) Avulsion of the distal biceps brachii tendon. Am J Surg 88:657–659
28. Postacchini F, Puddu G (1975) Subcutaneous rupture of the distal biceps brachii tendon: a report on seven cases. J Sports Med Phys Fitness 15:81–90
29. Bell RH, Wiley WB, Noble JS, Kuczynski DJ (2000) Repair of distal biceps brachii tendon ruptures. J Shoulder Elbow Surg 9:223–226
30. Sotereanos DG, Pierce TD, Varitimidis SE (2000) A simplified method for repair of distal biceps tendon ruptures. J Shoulder Elbow Surg 9(3):227–233
31. Morrey BF (2000) The elbow and its disorders, 3rd edn. Saunders Company, Philadelphia
32. Spencer EE, Tisdale A, Kostka K, Ivy RE (2008) Is therapy necessary after distal biceps tendon repair? Hand 3(4):316–319
33. Grewal R, Athwal GS, MacDermid JC, Faber KJ, Dros-dowech DS, El-Hawary R, King GJ (2012) Single versus double- incision technique for the repair of acute distal biceps tendon ruptures: a randomized clinical trial. J Bone Joint Surg Am 94:1166–1174
34. Anzel SH, Covey KW, Weiner AD et al (1959) Disruption of muscles and tendons; an analysis of 1,014 cases. Surgery 45(3): 406–414
35. Yeh PC, Dodds SD, Smart LR et al (2010) Distal triceps rupture. J Am Acad Orthop Surg 18(1):31–40
36. Mair SD, Isbell WM, Gill TJ et al (2004) Triceps tendon ruptures in professional football players. Am J Sports Med 32(2):431–434
37. Vidal AF, Drakos MC, Allen AA (2004) Biceps tendon and triceps tendon injuries. Clin Sports Med 23(4):707–722, xi
38. Kibuule LK, Fehringer EV (2007) Distal triceps tendon rupture and repair in an otherwise healthy pediatric patient: a case report and review of the literature. J Shoulder Elbow Surg 16:e1–e3
39. Weistroffer JK, Mills WJ, Shin AY (2003) Recurrent rupture of the triceps tendon repaired with hamstring tendon autograft augmentation: a case report and repair technique. J Shoulder Elbow Surg 12:193–196
40. Duchow J, Kelm J, Kohn D (2000) Acute ulnar nerve compression syndrome in a powerlifter with triceps tendon rupture: a case report. Int J Sports Med 21:308–310
41. Singh RK, Pooley J (2002) Complete rupture of the triceps brachii muscle. Br J Sports Med 36:467–469
42. Weng PW, Wang SJ, Wu SS (2006) Misdiagnosed avulsion fracture of the triceps tendon from the olecranon insertion: case report. Clin J Sport Med 16:364–365
43. van Riet RP, Morrey BF, Ho E et al (2003) Surgical treatment of distal triceps ruptures. J Bone Joint Surg Am 85(10):1961–1967
44. Daglar B, Delialioglu OM, Ceyhan E et al (2009) Combined surgical treatment for missed rupture of triceps tendon associated with avulsion of the ulnar collateral ligament and flexor-pronator muscle mass. Strategies Trauma Limb Reconstr 4:35–39
45. Harris PC, Atkinson D, Moorehead JD (2004) Bilateral partial rupture of triceps tendon: case report and quantitative assessment of recovery. Am J Sports Med 32:787–792
46. Sanchez-Sotelo J, Morrey BF (2002) Surgical techniques for reconstruction of chronic insufficiency of the triceps: rotation flap using anconeus and tendoachillis allograft. J Bone Joint Br 84: 1116–1120
47. Brumback RJ (1987) Compartment syndrome complicating avulsion of the origin of the triceps muscle: a case report. J Bone Joint Surg Am 69:1445–1447
48. Farrar EL III, Lippert FG III (1981) Avulsion of the triceps tendon. Clin Orthop Relat Res 161:242–246
49. Searfoss R, Tripi J, Bowers W (1976) Triceps brachii rupture: case report. J Trauma 16:244–246
50. Gilcreest EL (1925) Rupture of muscles and tendons. JAMA 84:1819–1822
51. Haldeman KO, Soto-Hall R (1935) Injuries to muscles and tendons. JAMA 104:2319–2324
52. Newmark H III, Olken SM, Halls J (1985) Ruptured triceps tendon diagnosed radiographically. Australas Radiol 29:60–63
53. McCulloch PC, Spellman J, Bach BR Jr (2008) Familial triceps tendon ruptures. Orthopedics 31:600–602
54. Rettig AC (2002) Traumatic elbow injuries in the athlete. Orthop Clin North Am 33:509–522
55. Clayton ML, Thirupathi RG (1984) Rupture of the triceps tendon with olecranon bursitis: a case report with a new method of repair. Clin Orthop Relat Res 184:183–185
56. Tsourvakas S, Gouvalas K, Gimtsas C et al (2004) Bilateral and simultaneous rupture of the triceps tendons in chronic renal failure and secondary hyperparathyroidism. Arch Orthop Trauma Surg 124:278–280
57. Irby R, Hume DM (1968) Joint changes observed following renal transplants. Clin Orthop Relat Res 57:101–114
58. Blackmore SM, Jander RM, Culp RW (2006) Management of distal biceps and triceps ruptures. J Hand Ther 19:154–168
59. Thornton R, Riley GM, Steinbach LS (2003) Magnetic resonance imaging of sports injuries of the elbow. Top Magn Reson Imaging 14:69–86
60. Wagner JR, Cooney WP (1997) Rupture of the triceps muscle at the musculotendinous junction: a case report. J Hand Surg Am 22: 341–343
61. Levy M, Goldberg I, Meir I (1982) Fracture of the head of the radius with a tear or avulsion of the triceps tendon. J Bone Joint Surg Br 64:70–72
62. Tatebe M, Horii E, Nakamura R (2007) Chronically ruptured triceps tendon with avulsion of the medial collateral ligament: a report of 2 cases. J Shoulder Elbow Surg 16:e5–e7
63. Gupta RR, Murthi AM (2010) Distal humeral fracture with associated triceps tendon avulsion in a renal transplant recipient. Orthopedics 10:204–207
64. Bach BR Jr, Warren RF, Wickiewicz TL (1987) Triceps rupture. A case report and literature review. Am J Sports Med 15(3): 285–289
65. Sherman OH, Snyder SJ, Fox JM (1984) Triceps tendon avulsion in a professional bodybuilder. A case report. Am J Sports Med 12(4):328–329
66. Lambert MI, St Clair GA, Noakes TD (1995) Rupture of the triceps tendon associated with steroid injections. Am J Sports Med 23(6):778
67. Bos CF, Nelissen RG, Bloem JL (1994) Incomplete rupture of the tendon of triceps brachii. A case report. Int Orthop 18(5):273–275
68. Viegas SF (1990) Avulsion of the triceps tendon. Orthop Rev 19(6):533–536
69. Pina A, Garcia I, Sabater M (2002) Traumaticavulsion of the tricepsbrachii. J Orthop Trauma 16:273–276
70. Kijowski R, Tuite M, Sanford M (2005) Magnetic resonance imaging of the elbow. Part II: abnormalities of the ligaments, tendons, and nerves. Skeletal Radiol 34(1):1–18
71. Wolf JM, McCarty EC, Ritchie PD (2008) Triceps reconstruction using hamstring graft for triceps insufficiency or recurrent rupture. Tech Hand Up Extrem Surg 12:174–179

Medial Epicondylitis

27

José Luis Avila Lafuente, Miguel A. Ruiz Iban,
Miguel García Navlet, Sergi Sastre Solsona,
Ricardo Cuellar Gutiérrez, and Eduardo Sánchez Alepuz

27.1 Introduction

Lateral and medial epicondylitis are two of the more common diagnoses in elbow pain and often occur as a result of occupational activities and sports. Histologically, it involves tendon degeneration rather than inflammatory changes. The term tendinosis is more appropriate [1].

Medial epicondylitis is a tendinopathy of the common flexor-pronator origin (CFPO). The muscles involved are the *pronator teres*, *flexor carpi radialis*, *palmaris longus*, and *flexor carpi ulnaris*; these are localized from superior to inferior in the CFPO. It is the most common reason of medial elbow pain, but four to seven times less frequent than lateral epicondylitis [2]. It affects the dominant extremity twice as often as the nondominant.

J.L.A. Lafuente (✉)
Servicio de Traumatología y C. Ortopédica, Hospital MAZ,
Avda. Academia General Militar 74, Zaragoza 50015, Spain
e-mail: jlavila@maz.es

M.A.R. Iban
Servicio de Traumatología y C. Ortopédica, Hospital Ramon y Cajal, Crtra. De Colmenar Viejo km 9,100, Madrid 28034, Spain
e-mail: drmri@hotmail.com

M.G. Navlet
Servicio de Traumatología y C. Ortopédica, Hospital de Asepeyo, Calle Joaquin Cardenas 2, Coslada, Madrid 28820, Spain
e-mail: miguelgnavlet@gmail.com

S.S. Solsona
Servicio de Traumatología y C. Ortopédica, Hospital Clinic, Calle Villarroel 170, Barcelona 08036, Spain
e-mail: sergi.sastre@gmail.com

R.C. Gutiérrez
Servicio de Traumatología y C. Ortopédica, Hospital Universitario Donostia,
Paseo Dr. Beguiristain s/n, San Sebastian, Guipuzcua 20014, Spain
e-mail: rcuellarg@gmail.com

E.S. Alepuz
Servicio de Traumatología y C. Ortopédica, Union De Mutuas, Avda. Del Lledó 69, Castellón 12004, Spain
e-mail: esancheza@telefonica.net

Although the natural history of medial epicondylitis has not been well investigated, one prospective study has suggested that most cases of acute or subacute medial epicondylitis heal spontaneously [3]. Surgical treatment is required when this condition fails to respond to nonoperative methods [4]. If medial epicondylitis treatments are unsuccessful, associated ulnar neuropathy should be considered. In athletes participating in sports that involve overhead throwing with valgus overload of the elbow, such as baseball pitchers, a chronic ulnar collateral ligament (UCL) insufficiency should also be considered.

27.2 Etiology and Injury Mechanism

The CFPO is a bone-to-tendon interface and is therefore subject to high stress and frequent injuries because of the differing physical properties of the bone and tendon [5]. Medial epicondylitis develops as a result of medial stress overload on the flexor muscles at the elbow. The cause of the disorder seems to be repetitive eccentric or concentric overloading of the flexor and pronator muscle masses (*pronator teres*, *common flexor*, and *flexor carpi ulnaris*). Most cases are related to occupational exposure in older patients and cause subacute or chronic symptoms. Only 10–20 % of patients, usually young athletes, have an acute injury.

In athletes, medial epicondylitis can affect golfers (golfer's elbow) or throwing athletes, particularly baseball pitchers and javelin throwers. Medial epicondylitis can also present in a tennis player. In this case, the pain can be reproduced on the forehand, serve, or overhead stroke [2]. Other athletes less commonly affected are softball players, archers, sport rock climbers, wrestlers, gymnasts, and weight lifters.

27.3 Clinical and Diagnostic Examination

Patients have pain and tenderness over the affected tendinous insertions in anterior medial epicondyle, which are accentuated with specific movements as active resisted pronation of

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_27,

Table 27.1 The Gabel-Morrey classification of medial epicondylitis

Type	Ulnar nerve neuropathy
I-A	None
I-B	Mild
II	Moderate or severe

the forearm and with resisted wrist flexion with the forearm supinated. A decrease in grip strength is observed too. Sometimes, a loss of range of movement of the elbow is associated [6].

It is necessary to evaluate carefully the ulnar nerve for signs of neuritis as more than 50 % of patients with medial epicondylitis have an associated ulnar nerve entrapment. Gabel and Morrey classified medial epicondylitis according to the level of the associated ulnar neuropathy (Table 27.1). They showed that patients with more advanced ulnar nerve affection had poorer results after operative treatment [7].

In throwing athletes, assessing the integrity of the ulnar ligament is imperative. When applying valgus force to the elbow slightly flexed with the forearm in pronation and wrist in flexion, opening of the joint and pain appear in patients with ligament insufficiency.

Medial epicondylitis presents with cervical radiculopathy in slightly more than half the patients. Weakening of the *flexor carpi radialis* and *pronator teres* and imbalance of the flexor and extensor muscles from the C6 and C7 radiculopathy allow for easy onset of medial epicondylitis. Patients with medial epicondylitis should be examined for C6 and C7 radiculopathy to ensure proper treatment. Physicians dealing with golfers, pitchers, or other patients with medial epicondylitis should be aware of the association between these two diagnoses to optimize care [8].

Quality anteroposterior, oblique, oblique axial in flexion, posteroanterior axial, and cubital tunnel radiographic views should be assessed for the presence of calcification or a bony exostosis at the medial epicondyle [9]. When instability is suspected, elbow stress radiographs can be of help in identifying ulnar ligament tears.

Musculoskeletal ultrasonography allows for an inexpensive dynamic evaluation of commonly injured structures [10]. Medial epicondylitis is ultrasonographically diagnosed when a focal hypoechoic (degeneration) or anechoic (tear) region with increased vascularity in color Doppler imaging is present at the medial epicondylar muscles with or without calcification [11].

Magnetic resonance (MR) imaging is the preferred imaging modality for chronic elbow pain [12, 13]. MR imaging findings of patients with clinically diagnosed medial epicondylitis include thickening and increased T1 and T2 signal intensity of the common flexor tendon and soft tissue edema around the tendon [14]. The use of MR arthrography at some imaging centers has been reported to have great sensitivity in identifying UCL tears [15].

When ulnar nerve injury or cervical radiculopathy are suspected, the use of electromyographic studies is necessary.

27.4 Treatment Strategy

27.4.1 Non-operative Treatment

Once the diagnosis is established in acute or subacute cases, a period of conservative management is mandatory as a high percentage of patients are known to heal spontaneously without significant intervention. Stahl et al. [3] compared prospectively the effect of local injection of lidocaine and methylprednisolone versus saline solution, associated with physical therapy and nonsteroidal anti-inflammatory drugs (NSAIDS) to treat medial epicondylitis and showed that after 3 and 12 months, patients reported significant improvement in both groups without significant differences. Furthermore, non-operative treatment has been shown to be effective in up to 90 % of cases [3].

There are many options regarding conservative management, but first line should include a short 2–4 week course of NSAIDS associated with rest. NSAIDS are useful in dealing with the concomitant synovitis, and refraining from the injuring activity is the key in avoiding further damage. A detailed review of the specific sport technique and equipment might help as subtle modifications in these can reduce the stress to the CFPO and rapidly improve symptoms.

Full immobilization of the elbow is rarely necessary and reserved for patients with acute symptoms and significant pain and disability, but counterforce bracing during sport practice may be helpful, although it is less effective than in lateral epicondylitis cases.

Different physical therapy modalities might be of use: a combination of ice and flexibility and strengthening training is effective in many tendinosis. The short-term analgesic effect of manipulation techniques may allow more vigorous stretching and strengthening exercises resulting in a better and faster recovery [16]. Forearm Kinesio Taping may enhance absolute force sense and improve pain condition for athletes suffering from medial epicondylitis as baseball players [17].

Instrumental electrophysical modalities – ultrasound, extracorporeal shock wave therapy (ESWT), transcutaneous electrical nerve stimulation (TENS), laser therapy, iontophoresis, phonophoresis, and others – can provide benefit, mainly with ultrasound and laser [18, 19].

Local corticosteroid injections seem to be effective in the short term but not in the long term [3, 19]. Their usefulness is thus controversial. Subcutaneous deposition of the injection must be avoided to prevent complications like hypopigmentation and fat atrophy.

Other types of injections and acupuncture: Suresh et al. [20] suggested that a combination of dry needling and autologous blood injection under ultrasound guidance might be an effective treatment for refractory medial epicondylitis, but all these types of techniques continue to be controversial.

Platelet-rich plasma (PRP): reviewing current literature regarding the biology of PRP and the efficacy of using PRP to augment healing of tendon, ligament, and muscle injuries, as well as early osteoarthritis, the most compelling evidence to support the efficacy of PRP is for its application to tendon damage associated with lateral and medial epicondylitis. PRP therapies have shown to be safe, but a number of questions remain unanswered, including the optimal concentration of platelets, what cell types should be present, the ideal frequency of application, or the optimal rehabilitation regimen for tissue repair and return to full function [21].

27.4.2 Surgical Treatment

It is indicated if conservative treatment fails, after a not well defined period. Patient compliance with activity modification and therapy exercises is mandatory before deciding to perform a surgical procedure. Typically a period of at least 6 months of conservative treatment is required to define failure, but in athletes about 2–4 months of ineffective conservative treatment might be enough to proceed to surgery. This can be accelerated if structural injury (such as tearing or detachment of the CFPO) is identified or if associated progressive ulnar nerve damage is detected [6].

The patients should know that operative treatment usually improves patient function significantly with increase in grip strength and improvement of clinical scores like DASH or Mayo postoperatively [22, 23]. But, 1 year after surgery, 5–30 % of patients will still experience moderate to severe tenderness at the medial epicondyle [24]. No major complications are usually observed.

Little has been published regarding operative techniques for medial epicondylitis. It is generally agreed that the same surgical principles used on the lateral side apply to the medial side: exposing the affected tendon, excising the pathologic portion, repairing the resultant defect, and sometimes reattaching any elevated tendon origin back to the epicondyle [6].

General or regional anesthesia is used. The patient is placed supine with the affected arm in a hand table, and a tourniquet is applied. A 6 cm incision is made centered just anterior to the medial epicondyle. Subcutaneous tissue is dissected. In this point, the medial antebrachial cutaneous nerve is placed at risk of injury. The CFPO is identified. Ulnar nerve and ulnar collateral ligament must be protected, but it is not necessary to expose them if they are not affected. The *pronator teres* and *flexor carpi radialis* interval is incised. The degenerated tissue is excised. Creating a bleeding surface in the medial epicondyle by removing fibrous tissue and performing multiple small drill holes might be effective. The remaining tendon is reattached to its origin if possible. At the end, the interval, the subcutaneous tissue, and the skin are closed after placing a drain. A posterior plaster splint is placed with the elbow at 90° and forearm and wrist in neutral position [7]. Vangsness and Jobe [4] reported good to excellent results in more than 95 % of patients with this technique, 86 % of the patients referred no limitation in elbow use a mean of 4 years after the surgical procedure.

The FETOR technique (fascial elevation and tendon origin resection) which facilitates the complete visualization and resection of the CFPO with limited soft tissue dissection is an effective and safe method described for the treatment of chronic recalcitrant medial epicondylitis [24]. It also minimizes the risk of incomplete removal of the degenerated tendon and allows a rapid rehabilitation. The most important step in this technique is elevating the intact fascia before CPFO resection and a carefully closing of the fascia at the end (Figs. 27.1 and 27.2). Kwon et al. [24] have recently presented the two-year results of this technique, with good or excellent results in 90 % of cases.

There are some less invasive surgical options described in the literature but without clearly superior results. A percutaneous release technique has been reported [25], but there is an increased risk of medial antebrachial cutaneous nerve and ulnar nerve damage. Other authors have proposed using a mini-open muscle resection procedure under local anesthesia [26] or the use of radiofrequency probe to create healing tissue in affected region by a limited exposure. A safety arthroscopic technique has been described in a cadaveric study [27], but there are not any reports in humans.

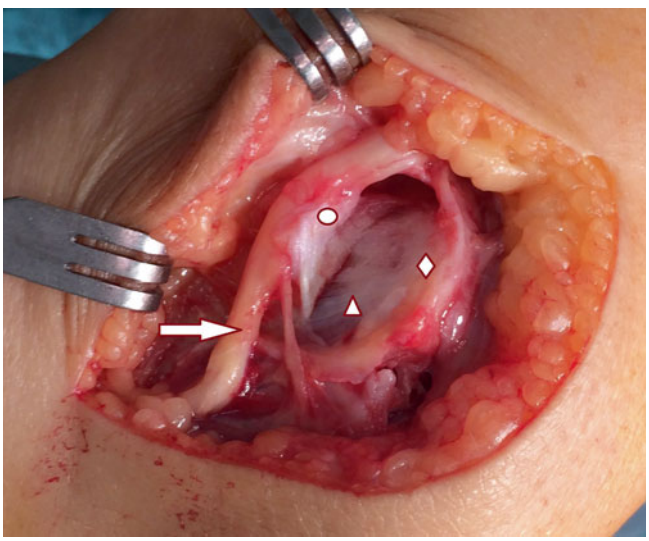


Fig. 27.1 Intraoperative image where it is exposed the medial epicondyle (*rhombus*), the elevated fascia (*circle*), the common flexor-pronator origin (*arrowhead*), and the released and transposed ulnar nerve (*arrow*)

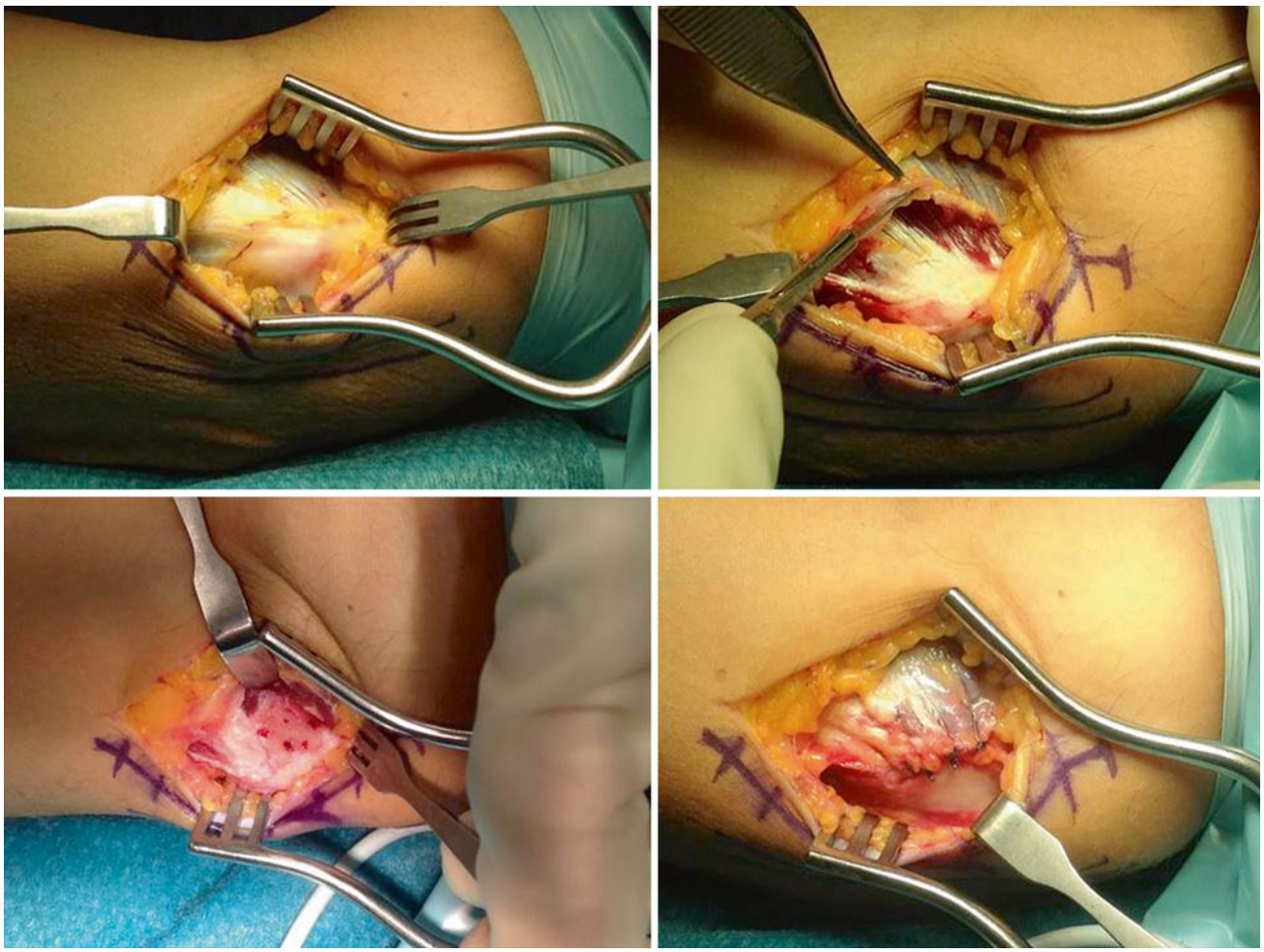


Fig. 27.2 FETOR technique. Intraoperative images where it is exposed the medial epicondyle approach (*up-left*), the elevated fascia and degenerative tissue (*up-right*), after removing tendon and medial epicondyle drilling (*down-left*) and fascial closing (*down-right*)

Concurrent ulnar nerve and/or ulnar collateral ligament pathology should be addressed and managed appropriately if symptoms are present.

If ulnar nerve entrapment is associated, cubital tunnel (mainly the Osborne ligament) and ulnar nerve release should be performed along the CPFO resection. Sometimes, an anterior nerve transposition, usually in subcutaneous and less commonly in intermuscular or submuscular position, is added. If moderate or severe neuropathy is present, the patient should be warned as the worst surgical results have been reported in this specific group [7].

If a medial collateral ligament insufficiency is present, a ligament reconstruction should be added to the procedure. There are many different techniques available but a reconstruction performed with an autograft (*palmaris*, *plantaris*, or Achilles tendon) in a figure of eight configurations [28] or a docking technique [29] usually solves the problem.

27.5 Rehabilitation and Return to Play

The plaster splint is removed 7–10 days after surgery. After that, a progressive passive and active range of motion of the elbow, forearm, wrist, and hand is initiated and prolonged during weeks 2–4. In this time, resisted wrist flexion and forearm pronation are avoided. After 4 weeks, light resisted isometric exercises should be started. Then, at 6 weeks from surgery, it is possible to start progressive strengthening, including isotonic exercises. Shoulder exercises and body conditioning are also carried out in this moment. Equipment and technique enhancements should be considered as well.

A sport simulation can be performed at 10–12 weeks in order to evaluate the path to return-to-sport timing, but full return to sports is not recommended until 4–6 months postoperatively, when full strength has returned to the extremity [30].

If the ulnar nerve and ligament are also treated, this protocol must be more conservative.

References

1. Kahn KM, Cook JL, Kannus P, Maffully N, Bonar SF (2002) Time to abandon the "tendinitis" mith, Painful, overuse tendon conditions have a non-inflammatory pathology. BMJ 324(7338):626
2. Leach RE, Miller JK (1987) Lateral and medial epicondylitis of the elbow. Clin Sports Med 6:259–272
3. Stahl S, Kaufman T (1997) The efficacy of an injection of steroids for medial epicondylitis: a prospective study of sixty elbows. J Bone Joint Surg Am 79(11):1648–1652
4. Vangsness CT Jr, Jobe FW (1991) Surgical treatment of medial epicondylitis. Results in 35 elbows. J Bone Joint Surg 73B:409–411
5. Shaw HM, Benjamin M (2007) Structure-function relationships of enthuses in relation to mechanical load and exercise. Scan J Med Sci Sports 17(4):303–315
6. Ollivierre CO, Nirschl RP, Pettrone FA (1995) Resection and repair for medial tennis elbow: a prospective analysis. Am J Sports Med 23(2):214–221
7. Gabel GT, Morrey BF (1995) Operative treatment of medial epicondylitis. J Bone Joint Surg 77A(7):1065–1069
8. Lee AT, Lee-Robinson AL (2010) The prevalence of medial epicondylitis among patients with c6 and c7 radiculopathy. Sports Health 2(4):334–336
9. Wright RW, Steger-May K, Klein SE (2007) Radiographic findings in the shoulder and elbow of Major League Baseball pitchers. Am J Sports Med 35:1839–1843
10. Miller TT, Adler RS, Friedman L (2004) Sonography of injury of the ulnar collateral ligament of the elbow-initial experience. Skeletal Radiol 33:386–391
11. Gy P, Lee SM, Lee MY (2008) Diagnostic value of ultrasonography for clinical medial epicondylitis. Arch Phys Med Rehabil 89(4):738–742
12. Dewan AK, Chhabra AB, Khanna AJ, Anderson MW, Brunton LM (2013) MRI of the elbow: techniques and spectrum of disease: AAOS exhibit selection. J Bone Joint Surg Am 95(14):1–13
13. Wenzke DR (2013) MR imaging of the elbow in the injured athlete. Radiol Clin North Am 51(2):195–213
14. Kijowski R, De Smet AA (2005) Magnetic resonance imaging findings in patients with medial epicondylitis. Skeletal Radiol 34(4):196–202
15. Hill NB Jr, Bucchieri JS, Shon F, Miller TT, Rosenwasser MP (2000) Magnetic resonance imaging of injury to the medial collateral ligament of the elbow: a cadaver model. J Shoulder Elbow Surg 9:418–422
16. Hoogvliet P, Randsdorp MS, Dingemanse R, Koes BW, Huisstede BM (2013) Does effectiveness of exercise therapy and mobilisation techniques offer guidance for the treatment of lateral and medial epicondylitis? A systematic review. Br J Sports Med 47(17):1112–1119
17. Chang HY, Wang CH, Chou KY, Cheng SC (2012) Could forearm Kinesio Taping improve strength, force sense, and pain in baseball pitchers with medial epicondylitis? Clin J Sport Med 22(4):327–333
18. Dingemanse R, Randsdorp M, Koes BW, Huisstede BM (2014) Evidence for the effectiveness of electrophysical modalities for treatment of medial and lateral epicondylitis: a systematic review. Br J Sports Med 48(12):57–65
19. Lee SS, Kang S, Park NK, Lee CW, Song HS, Sohn MK, Cho KH, Kim JH (2012) Effectiveness of initial extracorporeal shock wave therapy on the newly diagnosed lateral or medial epicondylitis. Ann Rehabil Med 36(5):681–687
20. Suresh SP, Ali KE, Jones H, Connell DA (2006) Medial epicondylitis: is ultrasound guided autologous blood injection an effective treatment? Br J Sports Med 40(11):935–939
21. Halpern BC, Chaudhury S, Rodeo SA (2012) The role of platelet-rich plasma in inducing musculoskeletal tissue healing. HSS J 8(2):137–145
22. Shahid M, Wu F, Deshmukh SC (2013) Operative treatment improves patient function in recalcitrant medial epicondylitis. Ann R Coll Surg Engl 95(7):486–488
23. Kurvers H, Verhaar J (1995) The results of operative treatment of medial epicondylitis. J Bone Joint Surg Am 77(9):1374–1379
24. Kwon BC, Kwon YS, Bae KJ (2014) The fascial elevation and tendon origin resection technique for the treatment of chronic recalcitrant medial epicondylitis. Am J Sports Med 42(7):1731–1737
25. Baumgard SH, Schwartz DR (1982) Percutaneous release of the epicondylar muscles for humeral epicondylitis. Am J Sports Med 10(4):233–236
26. Cho BK, Kim YM, Kim DS, Choi ES, Shon HC, Park KJ, Lee EM (2009) Mini-open muscle resection procedure under local anesthesia for lateral and medial epicondylitis. Clin Orthop Surg 1(3):123–127
27. Zonno A, Manuel J, Merrell G, Ramos P, Akelman E, DaSilva MF (2010) Arthroscopic technique for medial epicondylitis: technique and safety analysis. Arthroscopy 26(5):610–616
28. Conway JE, Jobe FW, Glousman RE, Pink M (1992) Medial instability of the elbow in throwing athletes. Treatment by repair or reconstruction of the ulnar collateral ligament. J Bone Joint Surg Am 74:67–83
29. Rohrbough JT, Altchek DW, Hyman J, Williams RJ 3rd, Botts JD (2002) Medial collateral ligament reconstruction of the elbow using the docking technique. Am J Sports Med 30:541–548
30. Ciccotti MG, Charlton WPH (2001) Epicondylitis in the athlete. Clin Sports Med 20(1):77–93

Lateral Epicondylitis of the Elbow

28

Paolo R. Rolla, Dario Pitino, and Giacomo Delle Rose

Lateral epicondylitis is commonly defined "tennis elbow."

The abuse from physicians and patients of the term of epicondylitis is very frequent in the clinical practice.

The term of epicondylitis defines a frequent myotendinosis occurring more specifically at the common extensor tendon that originates from the lateral epicondyle.

The tendon damage is related to repetitive strain injury caused by repetitive overuse of the extensor muscles of the wrist.

28.1 Etiology

The typical age at onset of epicondylitis is between 35 and 50 years, with a median of 41 years [1]. The disease is most common in not professional athletes in the third, fourth, and fifth decade, but this tendinopathy also occurs in patients such as teenagers and in the elderly amateur athletes over seventy.

No gender predominance was found in the reports.

The dominant arm is affected more frequently in athletes.

In the tennis epicondylitis is very common, but also in other athletic activities is quite frequent, as baseball, fencing, and swimming.

28.2 Injury Mechanism

The primary cause of tendinosis is overuse: excessive intensity and duration of arm use.

Inadequate or wrong training compromises the integrity of the tendon structure.

In some cases the tendon degeneration occurs after a direct trauma.

Lateral epicondylitis is directly related to activities that increase tension loads of the wrist and finger extensors and the supinator muscles. The extensor carpi radialis brevis is active in the elbow flexion and extension, but also in the varus and valgus stress.

The primary overload abuse in tendinosis is caused by intrinsic eccentric and concentric muscular contraction. The overload causes micro tears within the tendon starting the tendon degeneration.

28.3 Pathology

The primary anatomical structure involved is the extensor carpi radialis brevis tendon origin. In many cases also the anterior portion of the extensor digitorum communis tendon is involved.

Epicondylitis was historically believed to be an inflammatory disease at the insertion of the tendon, but in 1979, it was described as an "angiofibroblastic hyperplasia." The tendon lesion is characterized by the alteration of normal collagen structure with fibroblast proliferation in association with an immature vascular reparative cellular response.

The degree of angiofibroblastic infiltration appears to correlate generally with the duration of symptoms.

On gross examination, the tendon with tendinosis reveals gray, dull, sometimes edematous and friable, tissue very similar to mature granulation tissue [2].

28.4 Clinical and Diagnostic Examination

Epicondylitis typically causes lateral elbow pain and functional impairment in the use of the arm in daily activities, nocturnal pain, and limitation or impossibility to perform the athletic activity. The pain increases with wrist extension, and grip strength is diminished.

The palpation of the lateral epicondyle and, overall, of the extensor aponeurosis is painful.

The area of maximal tenderness typically lies 5 mm distal and anterior to the tip of the lateral epicondyle.

P.R. Rolla, MD (✉) • D. Pitino, MD • G.D. Rose, MD
Shoulder and Elbow Surgery, Humanitas Research Hospital,
Via Manzoni 56, Rozzano (MI) 20089, Italy

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_28,

Provocative stress testing consists of resisted wrist and finger extension with the elbow in flexion. Increased pain is common when the elbow is brought into extension.

The elbow has commonly a full range of motion

Radiographic examination is necessary to exclude bony epiphyseal disease. In a small percentage of patients plain X-rays reveal an irregular profile of the tip of the lateral epicondyle. Small calcific deposits in the substance of the tendon can rarely occur. Magnetic resonance imaging (MRI) in T2 and fat sat scans clearly confirm the pathology in the tendons with proximal insertion at the lateral epicondyle.

The patients with epicondylitis can be divided in three different categories:

Category I

Acute pathology with reversible inflammation without angiofibroblastic invasion.

Minor pain is reported, usually after heavy or specific activities.

Anti-inflammatory drugs and physiotherapy, followed by rehabilitative exercise and specific limitation to avoid overuse of the arm, achieve a quick response to the treatment of this pathology.

Category II

Partial angiofibroblastic invasion of the tendon characterizes the pathology.

The tissue modification is permanent, but some healing response may occur.

Patients have intense pain with activity and moderate pain at rest. After periods of rest, however, most daily activities can be accomplished without significant discomfort.

Magnetic resonance imaging in coronal view usually shows a pathologic signal in the ERB tendon.

If less than half of the tendon diameter is involved, treatment concepts that promote healing gradually bring about resolution, and this process can be managed nonoperatively. Occasionally, however, these patients require surgery for a more complete resolution of symptoms.

Category III

Extensive angiofibroblastic degeneration, frequently associated at partial or complete tear of the tendon, is found in these patients.

The patients are exacerbated for significant functional impairment in daily activities and pain. Pain at rest in the night and during the day.

T2 and FS coronal sections of MRI reveal major signal changes.

The patients in this condition often require surgery for pain relief, considering the frequent failure of the nonoperative measures.

28.5 Differential Diagnosis

Differential diagnosis has to consider:

- Carpal tunnel syndrome, more frequent in women; Tinel test and Phalen test are positive; to confirm the suspect, EMG is necessary.
- Entrapment of the motor branch of the radial nerve (posterior interosseus nerve) at the Frohse arcade, with more anterior pain and EMG positive.
- Distal biceps brachii tendinitis: pain in the flexion and supination of the elbow against resistance and pain localized in the anterior cubital fossa.
- Cervicobrachial pain related to nerve root compression, with pain evocated by neck mobilization.
- Osteoid osteoma of the proximal radius: the pain increases with alcohol assumption and is controlled by acetylsalicylic acid; TC scan, MRI, and bone scan are necessary for the diagnosis.
- Necrosis of the humeral lateral condyle (capitulum humeri), osteochondritis dissecans of the capitulum humeri and Panner's disease are evident with the elbow MRI.
- Focal synovitis: suspected in the MRI, but confirmed with the elbow arthroscopy.
- Synovial plica of the humeral-radial joint: tenderness at the palpation of the posterolateral soft spot.

The diagnosis is commonly made through clinical history and physical examination; however, in the athletes it is imperative to perform plain x-rays and MRI of the elbow, to confirm the diagnosis of epicondylitis, but overall to prevent mistakes in the so wide spectrum of the differential diagnosis, as above reported.

28.6 Treatment

Most patients improve with nonoperative measures, such as activity modification, physical therapy and injections. A small percentage of patients will require surgical release of the extensor carpi radialis brevis tendon, performed via percutaneous, arthroscopic, or open approaches.

28.6.1 Nonsurgical Treatment

The main problem of the patient with elbow tendinosis is pain, but the pain control does not necessarily imply enhancement of healing. Relative rest (not absence of activity but abstinence from abuse) and application of cold are appropriate. Activity that aggravates the condition should be eliminated. The use of nonsteroidal anti-inflammatory medications seems to be helpful in some patients. The physical therapy

has been helpful in relieving pain at the beginning of the disease.

If the patient does not respond to an appropriate pain control program, a cortisone local injection may be indicated [3]. The author uses 1.5 mL of 0.2 % lidocaine (Xylocaine) mixed with 40 mg of 40 mg methylprednisolone hemisuccinate, instilled with a 25 or 27 G needle under the tendon of the extensor brevis just anterior and slightly distal to the lateral epicondyle. After the injection, the local anesthesia permits to perform scarifications by the needle of the area of tendinosis. If the injection is too superficial or is done on a repetitive basis, subdermal atrophy may occur.

The repeated use of cortisone injections is related to tenocyte cellular death and weakening of the surrounding normal tissues.

28.6.2 Promotion of Healing

A biologic healing response includes infiltration of healthy neovascular and fibroblastic elements, collagen production, and collagen maturation at the cellular level in addition to the restoration of strength and endurance.

The healing process may be hastened by three general measures: relative rest, physical therapy, and most importantly rehabilitation exercises.

The injured part may be rested through the use of immobilizing devices to avoid abuse, modification of inappropriate activity, counterforce bracing and selection of proper sport equipment.

Partial immobilization by wrist extension splints has the limited value of casting except short-term use in the early, fully reversible inflammatory phase of category I injuries.

A progressive activity program for the injured part, as well as an aggressive activity program for the adjacent normal, undamaged tissues, should be accentuated.

As a result of the studies on compression braces, it has been demonstrated that there is no proven advantage and it may have a theoretical disadvantage, if balanced forearm muscle strength is disturbed.

As figured previously, on one hand rigid types of immobilization at the elbow or wrist relieve pain, but on the other hand they cause atrophy and immobility, making this procedure not recommended.

Rehabilitation should include not only the cardinal forearm exercises but also those for the upper back and shoulder [4–6]. Once the probable initial adjacent inflammatory response and the pain have been controlled, an orderly progression of the graduated strength and endurance exercise is started [5, 6]. The patient is protected until about 60 % of the rehabilitation exercise goals [2, 6, 7].

After the patient has reached the early rehabilitative strength and flexibility goals, by doing graduated exercise and full-strength training, these functions are monitored by objective testing until strength, endurance, and flexibility have returned to nearly normal levels.

Continuation of the strength and endurance exercise program beyond the preinjury level includes either isokinetic or isotonic, interspaced with isoflex exercises. Before a final return to a sport or an occupational activity, the patient should be capable of anaerobic sprint repetitions to fatigue without major activity pain [6].

Recent reports have suggested the existence of other approaches through which we may achieve pain control and the possible biologic alteration of tendinosis tissue. These include shock wave, various forms of prolotherapy injections (autologous blood, platelets, and dilute sugar), nitric oxide patches, magnetic, cold laser, acupuncture, and high-intensity infrared heat. Of this group, shock wave and autologous injections have the most research support, although success has been inconsistent and further research is indicated.

Platelet-rich plasma (PRP), autologous blood, and prolotherapy are aimed at inducing inflammation rather than suppressing it. PRP is used for chronic tendinitis and is defined as a volume of the plasma fraction of autologous blood having a platelet concentration above baseline. Both PRP and autologous blood contain platelets, and these platelets have strong growth factors and granules that have critical role in the healing process of chronic injuries. Due to higher concentration of platelets in PRP than whole blood, it was shown to have greater effect in the repair process in the treatment of chronic epicondylitis.

Extracorporeal shock wave therapy had been commonly used to treat urolithiasis or choledocholithiasis, and the therapy was introduced to treat musculoskeletal conditions such as calcific tendinitis of the shoulder joint, lateral epicondylitis of the elbow joint, and plantar fasciitis in Germany in the 1990s. In addition, the therapy was also introduced to treat plantar fasciitis and chronic epicondylitis of the elbow joint in the United States in the 2000s. Nevertheless, it is hard to find research about the therapy as an initial treatment option because research on the effects of extracorporeal shock wave therapy for lateral or medial epicondylitis of the elbow has been primarily conducted with patients who failed to respond to conservative management after the acute phase.

Lee in 2012 has shown that initial extracorporeal shock wave therapy was effective for the patients newly diagnosed as lateral or medial epicondylitis, although the effectiveness on completion of the treatments was inferior to local steroid injection [8]. Therefore, extracorporeal shock wave therapy can be another option when local steroid injection is contraindicated.

28.6.3 Surgical Treatment

The surgery choice for epicondylitis treatment makes reference to two techniques, arthroscopic surgery of recent use and open surgery. The choice of surgical treatment must still be reserved to patients who have failed conservative treatment and in which the painful symptoms impair athletic performance.

28.6.4 Arthroscopy

The patient is in lateral position, with all bony prominences well padded. We favor regional anesthesia. Bony landmarks are drawn out including the path of the ulnar nerve.

Next, a standard anteromedial portal is established. This is started several centimeters proximal and anterior to the medial epicondyle and well anterior to the palpable intermuscular septum. The medial portal allows one to view the lateral joint including the radial head, capitulum humeri and lateral capsule. By tensioning the capsule anteriorly, improved visualization of the lateral capsule and soft tissues can be achieved. *The arthroscopic inspection of the lateral capsule often reveals a full-thickness tear.*

A modified anterolateral portal is established using an out-in technique. This is started 2 cm above and anterior to the lateral epicondyle. This allows instrumentation down to the tendon origin rather than entering the joint through the ECRB tendon itself. The capsule is next released. Occasionally in epicondylitis, one can find a disruption of the underlying capsule from the humerus. It easier to release the lateral soft tissues in layers using a monopolar thermal device. In this way, the capsule is first incised or released from the humerus. When it retracts distally, one can value the ECRB tendon posteriorly and the ECRL, which is principally muscular, more anterior. As noted earlier, the ECRB tendon spans from the top of the capitulum humeri to the midline of the radiocapitellar joint [9].

Once the capsule is adequately resected, the ECRB origin is released from the epicondyle. This is started at the top of the capitellum and carried posteriorly. The lateral collateral ligament is not at risk if the release is kept anterior to the midline of the radiocapitellar joint. On average, adequate resection of the ECRB must include approximately 13 mm of tendon origin from anterior to posterior [10]. Care is taken to drive the scope adequately to view the release down to the midline of the radiocapitellar joint. Typically, the entire ECRB retracts distally from the humerus.

Care is taken not to release the extensor aponeurosis, which lies behind the ECRB tendon. This can be visualized as a stripped background of transversely oriented tendon and muscular fibers much less distinct than the ECRB. It is located posterior to the ECRL, which again is principally muscular in origin. The date of literature report variable results in using various arthroscopic techniques and may be related to increased difficulty in identifying the ECRB origin through the arthroscope. Tseng reported satisfactory results in nine of eleven patients [11]. However, he also had a 33 % complication rate. Stapleton and Baker compared five patients treated arthroscopically with ten patients treated by open debridement [12]. They reported similar results and complication rates between the two groups. Later, Baker et al. [13] reported on 39 elbows treated arthroscopically, with 37 reporting being "better" or "much better" at follow-up. Peart et al. [14] reported on 33 arthroscopic procedures for lateral epicondylitis, with 28 % of patients failing to achieve good or excellent outcomes.

The tendon is extra-articular, and capsular release is required to visualize its origin.

Arthroscopic release of the ECRB appears to be an effective option for the surgical treatment of chronic lateral epicondylitis unresponsive to conservative modalities. Knowledge of the anatomy, including the extensor tendon origins as visualized from an intra-articular perspective, is essential for effective surgical release.

28.6.5 Open Surgery

Identification of pathology and excision of all pathologic tendinosis tissue generally include most of, if not the entire, origin of the extensor carpi radialis brevis. In addition (approximately 50 %), excision of pathology also includes the anteromedial aspects of the extensor digitorum communis aponeurosis and, rarely, the removal of pathologic tissue from the underside of the extensor longus. When the extensor brevis origin is excised, the intimate and firm attachments between the fascia of the extensor brevis and the annular ligament and insertion into the distal aponeurosis eliminate any distal extensor brevis retraction beyond 1–2 mm. Once the pathologic tissue has been removed, a tissue defect in the triangular recess is present in varying degrees. It is appropriate to attempt to enhance the blood supply to this area by drilling one or two small holes through the cortical bone to cancellous depth in the triangular recess just distal and anteromedial to the epicondyle. Do not however drill into the epicondyle itself because this will increase postoperative pain. Drilling in the appropriate area encourages hematoma formation with ingrowth of vascular and fibrotendinous healthy replacement tissue [15].

Restoration of the normal extensor anatomic position, by sewing the posterolateral edge of the extensor longus to the anterior edge of the extensor aponeurosis, has been successful without causing loss of motion and is the repair technique of choice.

Because all the incisions are made longitudinally and, in most cases, do not disturb the extensor aponeurosis attachment to the lateral epicondyle, the surgery provides a firm anchoring point for quick initiation of the postoperative rehabilitative exercises with rapid return to full motion.

The popular literature concerning the surgical treatment of tennis elbow is considered to have begun in 1927 with Hohmann [16], who described the release of the extensor aponeurosis at the level of the lateral epicondyle. The technique, now commonly referred to as a muscle slide or release procedure, does not identify the offending pathology.

More recent versions of this approach have included percutaneous release. The reports to date are short-term follow-ups but do suggest that this approach can be effective in as many as 90 % or more of patients and has a relatively low complication rate.

Several surgical options are available besides release of the extensor aponeurosis from the lateral epicondyle. Kaplan reported three cases of resection of the radial nerve branches to the lateral epicondyle and lateral articular areas, with no attempt to identify or remove pathologic tendon tissues. He noted excellent pain relief, but denervation of a motor branch to the extensor brevis probably occurred with this technique. Interestingly, his three patients were hospitalized postoperatively for an average of 7 days. Roles and Maudsley described 33 patients who responded to surgical decompression of the posterior interosseus nerve and release of the brevis tendon. Posterior interosseous nerve compression continues to be recognized as an entity, with recent reports emphasizing the association of compression of the nerve at the arcade of Frohse [17].

More recently, Tasto et al. [18] have introduced radiofrequency ablation Testo tendinosis pathologic tissue, and Baker and Cummings [7, 13] introduced an arthroscopic release approach as a surgical option for lateral tennis elbow.

Arthroscopy results in increased instrument costs, setup time, and operative time. Arthroscopy also risks neurovascular harm and intra-articular scuffing and results in some flexion contracture via intra-articular transgression for a problem that is extra-articular. For those cases with associated intra-articular pathology, it is better to prefer the arthroscopic surgical procedure [2].

A basic principle of any orthopedic surgery is that a clear definition of the pathology and its location is essential for a well-conceived surgical procedure. Because the extensor brevis origin is largely covered by the muscle of the extensor carpi radialis longus, extensor digitorum communis (EDC) aponeurosis release operations do not visualize the extensor carpi radialis brevis tendinosis pathology. Release of the common extensor origin, however, may alter the attachment of the brevis, because a significant segment of its origin is derived from the extensor aponeurosis. This anatomy helps to explain the instances of success of release techniques, including percutaneous procedures.

Overall, the extensor carpi radialis brevis is involved by tendinosis in 100 % of cases, with additional involvement of the anteromedial aspects of the extensor digitorum communis tendon also in approximately 50 %.

28.7 Rehabilitation and Return to Play

In the postoperative rehabilitation, the elbow is maintained in 90° of flexion, with the wrist and hand free in an easily removable elbow immobilizer for approximately 6 days [4]. Limbering activities are undertaken, however, on days 2–3, generally by working the arm actively, followed by a gradual return to strength training exercises (usually daily) without resistance for the first 3 weeks postoperatively. Starting at 3 weeks postoperatively, gradual strengthening isotonic and isoflex resistances are implemented. Postoperative counterforce bracing usually is not used in our patients. For recreational tennis, it is usual to start easy strokes about 10–12 weeks from the time of surgery. For the return to competitive athletics or occupational activities, the increase in intensity should be gradual and gentle, until full strength has returned to the extremity. A return to full-strength use of the arm in competitive athletics averages 6–8 months for lateral elbow tendinosis.

References

1. Nirschl RP (1973) Tennis elbow. Orthop Clin North Am 4:787
2. Nirschl RP, Ashman E (2003) Elbow tendinopathy: tennis elbow. Clin Sports Med 22:813–836
3. Clarke AK, Woodland J (1975) Comparison of two steroid preparations used to treat tennis elbow, using the hypospray. Rheumatol Rehabil 14:47
4. Nirschl RP (1996) Arm care. Medical Sports Publishing, Arlington
5. Nirschl RP (1983) Isoflex exercise system. Medical Sports Publishing, Arlington
6. Ellenbecker TS, Nirschl R, Renstrom P (2013) Current Concepts in Examination and Treatment of Elbow Tendon Injury Sports Health 5(2):186–194
7. Baker C, Cummings P (1998) Arthroscopic management of miscellaneous elbow disorders. Operative Tecniques. Sportsmed 6:16
8. Lee SS, Kang S, Park NK, Lee CW, Song HS, Sohn MK, Cho KH, Kim JH (2012) Effectiveness of Initial Extracorporeal Shock Wave Therapy on the Newly Diagnosed Lateral or Medial Epicondylitis. Ann Rehabil Med 36(5):681–687
9. Smith AM, Castle JA, Ruch DS (2003) Arthroscopic resection of the common extensor origin: anatomic considerations. J Shoulder Elbow Surg 12:375
10. Cohen MS, Romeo AA, Hennigan SP, Gordon M (2008) Lateral epicondylitis: anatomic relationships of the extensor tendon origins and implications for arthroscopic treatment. J Shoulder Elbow Surg 17(6):954–960

11. Tseng V (1994) Arthroscopic lateral release for treatment of tennis elbow. Arthroscopy 10:335
12. Stapleton TR, Baker CL (1996) Arthroscopic treatment of lateral epicondylitis. Arthroscopy 10:335
13. Baker CL, Murphy KP, Gottlob CA, Curd DT (2000) Arthroscopic classification and treatment of lateral epicondylitis: two-year clinical results. J Shoulder Elbow Surg 9:475
14. Peart RE, Strickler SS, Schweitzer KM Jr (2004) Lateral epicondylitis: a comparative study of open and arthroscopic lateral release. Am J Orthop 33:565
15. Nirschl RP (1994) Lateral and medial epicondylitis. In: Morrey B (ed) Master techniques in orthopedic surgery: the elbow. Raven Press, New York, pp 129–148
16. Hohmann G (1933) Das Wesen und die Behandlung des Sogenannten tennissellenbogens. Munch Med Wochenschr 80:250
17. Jalovaara P, Lindholm RV (1989) Decompression of the posterior interosseous nerve for tennis elbow. Arch Orthop Trauma Surg 108:243
18. Tasto J, Cummings J, Medlock J, Harwood F, Hardesty R, Amiel D (2003) The tendon treatment center: new horizons in the treatment of tendinosis. Arthroscopy 19(Suppl 1):213

Elbow Instability

29

Luigi Adriano Pederzini and Felice Di Palma

29.1 Introduction

The elbow is a trochleoginglymoid joint [1] consisting of three articulations: the humeroulnar, the humeroradial, and the proximal radioulnar joint.

This configuration makes the elbow a fairly constrained and one of the most congruous and stable joints of the body. The ulna and the radius are connected by the forearm interosseous membrane which highly contributes to the stability of the proximal and distal radioulnar joints.

The normal range of motion of the elbow is approximately 0° of extension and 140° of flexion. A functional range of motion for activities of daily living has been described to be of 30°–130°, and the functional arc of throwing ranges from 20° to 130°. The normal supination and pronation are both of approximately 80° [2].

Although it is not a weight-bearing joint, it can be subjected to high loads when practicing racket or throwing sports or in gymnastics. As a consequence of these continued sport activities, elbow stability, due to static and dynamic constraints, can be compromised.

The elbow is the second most commonly dislocated major joint [3], and 15–35 % of elbow dislocations can have residual instability [4, 5]. Elbow dislocations represent 11–28 % of all elbow injuries, with an annual incidence of 6–8 cases per 100,000 people [6].

The symptoms of the instability in athletes can occur following a single traumatic event or may be due to repetitive stress leading to chronic laxity such as in a throwing athlete.

The focus of this chapter will be on elbow instabilities connected to sport activities. The causes of instabilities can be divided into medial, lateral, and posterolateral rotatory (PLRI).

L.A. Pederzini (✉) • F. Di Palma
Orthopaedic and Arthroscopic Department,
New Sassuolo Hospital, via F. Ruini, 2,
Sassuolo, Modena 41049, Italy
e-mail: gigiped@hotmail.com; felice.dipalma@libero.it

29.2 Etiology

The elbow joint is one of the most useful joint of the body. Its stability is due to different structures that can be divided into primary and secondary stabilizers [7].

The elbow is a very congruous joint with two ligamentous complexes: MCL and LCL. They are involved in the pathoanatomy of throwing athletes or in elbow dislocations and instability.

The primary stabilizers are represented by:

- Ulnohumeral joint, a stable articulation in which the humeral trochlea articulates with the ulnar notch (or incisura semilunaris) of the proximal ulna, with greatest stability in full extension and flexion. This stability is augmented by compressive forces of muscles.
- Anterior bundle of medial collateral ligament that is the primary valgus stabilizer among the different components of the medial ligament complex [8–10]. The medial collateral ligament complex (MCL) consists of three bundles with different insertions forming a triangular shape: the anterior, posterior, and transverse. The anterior bundle (or anterior oblique ligament) is the most significant component of the MCL, being the main stabilizer to valgus stress of the elbow [8, 11–13]. The anterior bundle can be further divided into anterior and posterior bands [8, 11, 14]. Some authors have included a third deep middle band [15, 16] (Fig. 29.1).
- Lateral ligament complex, made up of lateral ulnar collateral, radial collateral, annular ligament, and the accessory collateral ligament described by Martin [17]. This complex is the primary restraint to posterolateral rotatory instability and varus forces. To create functional posterolateral instability are necessary combined injuries. In fact isolated lateral ulnar collateral or radial collateral ligament injuries do not result in instability [18, 19].

The secondary stabilizers are represented by:

- Radial head: an important secondary restraint [10] because 60 % of axial loads are imparted through radiocapitellar

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_29, © Springer International Publishing Switzerland 2016

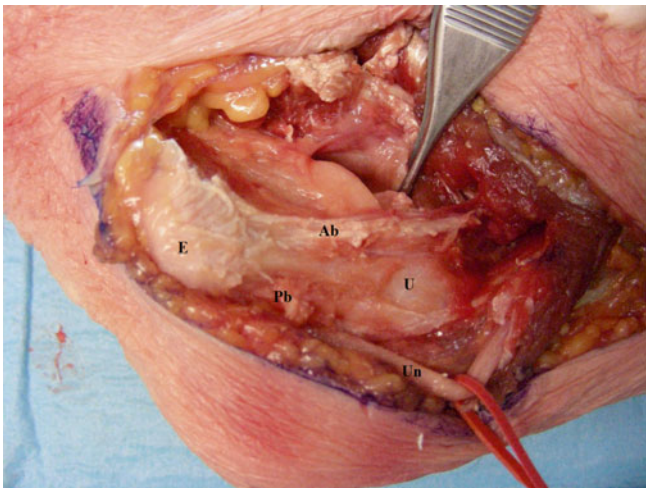


Fig. 29.1 Medial collateral ligament and ulnar nerve. *E* medial epicondyle, *Ab* anterior bundle, *Pb* posterior bundle, *U* ulna, *Un* ulnar nerve

joint [20]. Radial head injuries compromise lateral and medial stability by decreasing tension in the lateral ligament complex and because it is a secondary restraint to valgus load, respectively.

- Capsule: surrounding entirely the joint and gives better contribution in extension.
- Muscular support: anterior and posterior muscles that travel across the elbow and enable flexion and extension mobility. The elbow is also the site of origin for the flexor/pronator and extensor/supinator musculature of the forearm located medially and laterally, respectively. Anconeus muscle is anatomically oriented to provide restraint to posterolateral rotatory instability. The extensor/supinator musculature provides dynamic lateral stability; it is often avulsed with lateral ligaments. The flexor/pronator musculature provides dynamic valgus stability. These musculature groups have compression effect on ulnohumeral joint, augmenting bony stability [21].

29.3 Injury Mechanism

The mechanisms of injury are represented by subluxation and dislocation events.

The instabilities can be divided into simple and complex. The simple elbow instability indicates a dislocation with soft tissue lesions without associated fractures that can compromise joint stability [6, 22]. The most frequent form is a posterior dislocation produced by a posterolateral rotation mechanism (PLRI) as described by O'Driscoll [23].

The term complex elbow instability, which replaces the former "fracture–dislocation" and "transolecranon fracture," means on the other side the association of ligaments and bony lesions.

Simple dislocations without any secondary injuries to the bone occur more often than complex dislocation [6]. The latter account for 15–20 % of all elbow dislocations [24, 25].

Referring to direction and mechanism of dislocation, we can distinguish PLRI and valgus stress (which can be posttraumatic or due to overuse).

So dislocations can be simple (with only soft tissue injuries) and complex (with bone and soft tissue injuries).

Simple dislocations are classified as anterior, posterior (direct posterior, posterolateral, and posteromedial), and divergent (extremely rare in which the humerus is jammed between the radius and ulna and the interosseous membrane is destroyed) [26, 27].

Complex dislocations can be anterior and posterior.

A posterior dislocation is caused by a fall on the wrist while the elbow joint is extended and the wrist is pronated. The impact of the tip of the olecranon on the olecranon fossa has a leverage effect, while the coronoid process slips in a dorsal direction over the trochlea of humerus [23, 28].

A posterior dislocation is also caused by a fall on hand with a flexed elbow joint where the force, acting in a direct axial direction, makes the olecraon slip out [29, 30].

An anterior dislocation occurs through a combination between a flexed elbow and a force acting dorsally.

The most common mechanism is fall on outstretched hand generating axial load through the elbow.

29.3.1 PLRI

In all posterior dislocations, there is a lateral ligament disruption generating the so-called posterolateral rotatory instability (PLRI) [31, 32].

PLRI consist in three stages of instability that correlates with the severity of soft tissue injury:

Stage 1 – injury to lateral ligaments and extensor origin so this causes posterolateral shift of ulnohumeral and radiocapitellar joints.

Stage 2 – injury propagates to the anterior and posterior capsules, so this causes posterolateral subluxation with perching of the coronoid under the trochlea.

Stage 3 – posterolateral dislocation:

- 3a: anterior bundle of MCL is intact, so there is pivoting around intact ligament.
- 3b: anterior bundle of MCL is disrupted with complete dislocation (the most common injury pattern).
- 3c: complete stripping of all soft tissue from the distal humeral. It is grossly unstable unless flexed >90°.

O'Driscoll et al. [23] described a circle strategy for the sequence of injuries to soft tissue regarding to a simple posterior dislocation. The force generated flows from lateral to medial. This leads to rupture of the LCL, then the anterior and posterior capsules, and lastly the MCL.

29.3.2 Valgus Injuries

These injuries can be determined by falling with severe valgus moment or combined with direct contact at lateral elbow, often seen in contact athletes.

Repetitive microtraumas represent a common mechanism for valgus injuries in throwers.

Most common clinical pictures are represented by medial ligament injury, avulsion of flexor/pronator mass, and radial head/neck compression fractures.

29.3.3 Varus Injuries: Posteromedial Dislocation

Frequently due to a fall, a lateral ligament injury is often associated with medial facet coronoid fractures.

29.3.4 Complex Injuries

The mechanism of injury is similar to simple dislocation. Loading pattern and arm position determine associated bony lesions.

The timing of the lesions can be acute (post-traumatic) or chronic (post-traumatic or due to overuse).

29.4 Clinical Evaluation

First of all, it is necessary to have a complete medical history of the patient to locate the seat of the pain and the mode of onset, acute or chronic, and the cause, traumatic or not.

The patient may report a lateral elbow pain; a feeling of popping, snapping, or shifting; or recurrent subluxation or dislocations.

Patient may have apprehension while performing activities requiring forced extension of the elbow. The patient who practices sport launch may experience pain in the medial elbow during the acceleration phase.

Atraumatic onset is uncommon with PLRI.

Throwers may have changed their training; they can report loss of velocity and control.

The examiner should assess any predisposing factors such as surgical procedures performed in the lateral region as aggressive tennis elbow release, radial head resection, multiple lateral elbow injection (due to the possible weakening of the ligaments) and prior injury as cubitus varus for pediatric supracondylar humerus fracture malunion.

Anatomical deformity of the profile can hide bone injury or dislocation of the joint.

Before any clinical maneuver, it is important to rule out bony, nervous, and vascular disorders [24].

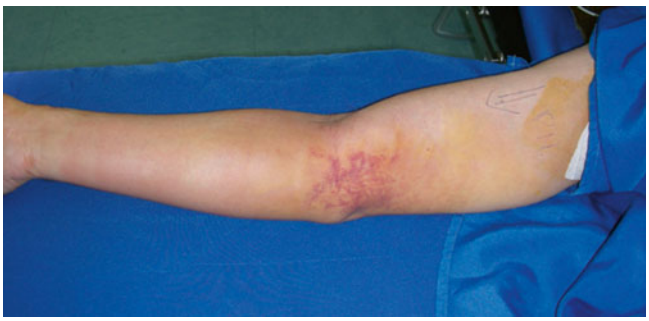


Fig. 29.2 MCL acute tear and elbow bruise

Clinical examination must include an accurate assessment of the ipsilateral shoulder, elbow, and wrist to exclude the presence of previous injuries or pathologies.

An evaluation of the ipsilateral distal radioulnar joint and the interosseous membrane for the presence of an Essex–Lopresti injury would also appear to be important [33, 34].

An injured elbow may be swollen due to the presence of periarticular edema or hematoma (Fig. 29.2). It is important to exclude the presence of a compartmental syndrome which rarely can develop from the beginning of the trauma. Therefore, clinical monitoring during the early hours is necessary [23].

Excluding bone and neurovascular lesions, stability tests must be performed.

Sometimes these maneuvers are performed after sedation (because of the pain) and under radiological control for better assessment.

Some patients arrive in the emergency department with joints which have already been spontaneously reduced.

In such cases, the diagnosis is derived from the case history and any possible instability, which may be present.

Several clinical maneuvers have been described to highlight different types of elbow instability.

29.4.1 Valgus Instability

Patients with this kind of instability usually report medial elbow pain and decreased strength during overhead activity; there may be symptoms of ulnar neuropathy from either acute or chronic UCL injury caused by edema/hemorrhage of the medial elbow or excessive traction on the nerve.

Patients with isolated UCL injury often have point tenderness 2 cm distal to the medial epicondyle, slightly posterior to the common flexor origin. The UCL stability can be assessed with specific physical exam tests. The "milking maneuver" involves having the patient apply a valgus torque to the elbow by pulling down on the thumb of the injured extremity with the contralateral limb providing stability [35]. With the modified milking maneuver, the examiner provides stability to the patient's elbow and pulls the thumb to create

a valgus stress on the UCL [36]. These tests result in pain and widening at the medial joint line if the UCL is insufficient. O'Driscoll and coworkers described the moving valgus stress test, in which the valgus torque is maintained constantly to the fully flexed elbow and then quickly extends the elbow [37]. This test is positive if medial elbow pain is elicited and has a 100 % sensitivity and 75 % specificity. The abduction valgus stress test is performed by stabilizing the patient's abducted and externally rotated arm with the examiner's axilla and applying a valgus force to the elbow at 30° of flexion. Testing with the forearm in neutral rotation has been shown to elicit the greatest valgus instability [38]. A positive test results in medial elbow pain and widening along the medial joint line. Even so, valgus laxity can be subtle on physical exam, and the range of preoperative detection is between 26 and 82 % of patients [39, 40]. Furthermore, Timmerman and colleagues found valgus stress testing to be only 66 % sensitive and 60 % specific for detecting abnormality of the anterior bundle of the UCL [41].

29.4.2 PLRI

PLRI is first described in 1991 by O'Driscoll and colleagues in a series of five patients [32].

The clinical mechanism of injury to the lateral stabilizers of the elbow that results in PLRI has been hypothesized to consist of supination of the forearm, combined with a valgus and axial load to the elbow [23]. The presentation is variable and can include lateral elbow pain; mechanical symptoms such as snapping, clicking, catching, or locking; and recurrent episodes of instability. Patients often report their elbow feels loose or like it is sliding out of place, especially when loading it in a slightly flexed position with a supinated forearm, as when pushing off an armrest while standing from a chair.

On physical exam, patients often have normal upper extremity strength and elbow range of motion and minimal to no tenderness around the LCL complex. Several provocative maneuvers have been developed to elicit instability symptoms. The posterolateral rotatory instability test is performed by supinating the forearm and applying valgus and axial forces to the elbow while flexing the elbow from full extension [32]. A positive test is demonstrated by reduction of a subluxated radial head when the patient is under general anesthesia or apprehension during testing when the patient is awake [32]. More recently, Regan and Lapner described two other apprehension tests, the chair sign and push-up sign [42]. The chair sign is performed by having the patient actively push off the armrests of a chair with the forearms supinated and the elbows at 90°. The test is considered positive with reluctance to fully extend the elbow during push off. The push-up sign is conducted by having the patient push off from the ground with the forearms supinated, elbows at 90°, and arms abducted to greater than shoulder width. A positive test results in apprehension and guarding as the elbow is terminally extended. These apprehension tests have been determined to be more sensitive than the posterolateral rotator instability test in awake patients. The table-top relocation test has been recently described by Arvind and Hargreaves [43]. The patient is asked to stand in front of a table. The hand of the symptomatic arm is placed over the lateral edge of the table. The test involves three parts. The patient is initially asked to perform a press-up with the elbow pointing laterally. This maintains the forearm in supination. Pressure is pushed down through the hand onto the table, as the elbow is allowed to flex (bringing the chest toward the table). In the presence of posterolateral rotatory instability (PLRI), positive apprehension and a reproduction of the patient's pain occur as the elbow reaches approximately 40° of flexion. The maneuver is then repeated, using the thumb of the examiner placed over the radial head, giving support and preventing posterior subluxation while the press-up is performed. Patients with posterolateral rotatory instability find that their symptoms of pain and instability are relieved by this second maneuver, which is similar to the relocation test of the shoulder. Finally, removal of the examiner's supporting thumb from the weight-bearing, partially flexed elbow reproduces the pain and apprehension. The relief and recurrence of pain during the second and third maneuvers helps to exclude articular pathology as the cause of pain and reinforces the diagnosis of instability.

29.5 Diagnostic Imaging

Conventional plain anteroposterior and lateral radiographs should be taken before any clinical maneuver. Oblique views may be necessary for a better assessment of the coronoid process and the radial head [6, 33]. Depending on the symptoms and pain experienced, radiographs should also be made of the adjacent joints. Similar to medial elbow instability, plain radiographs of the elbow are used to identify an avulsion fragments or associated fractures (e.g., coronoid, radial head) that can contribute to instability. Associated arthritic changes or loose bodies may also be seen. Widening of the ulnohumeral joint space after reduction of an acute dislocation, the so-called drop sign, has been associated with significant ligamentous injury and increased risk of recurrent instability [44].

Stress radiographs can be taken at the point of maximum rotatory subluxation during the pivot-shift test and may show widening of the ulnohumeral joint space on the lateral and anteroposterior views and posterior subluxation of the radial head on the lateral view.

CT scan is mandatory in course of complex dislocations [6, 24, 33].

MRI/arthroMRI is useful in case of chronic instabilities evidencing chondral associated lesions and possibly showing a leakage of contrast fluid in case of lateral or medial collateral lesions [45].

29.6 Treatment Strategy

The goal of the treatment is to achieve stable reduction of the elbow to begin as soon as possible a rehabilitation treatment to void stiffness [6, 46].

In the course of acute trauma with simple dislocation, the reduction maneuver generally does not require general anesthesia and can be performed with intravenous sedation.

Stability tests are carried out after the reduction, and in stable elbows, the rehabilitative protocols are begun in 10 days.

In case of instability, when the elbow joint remains reduced in a range from at least 60° of flexion to full flexion, the patients can start supervised active rehabilitation in stable arc of motion in hinged brace [6].

If there is not a stable arc of motion, open reduction with LCL or MCL repair or reconstruction is necessary.

29.7 Treatment of UCL Lesions

Initial treatment consists of rest, anti-inflammatory medications, icing, and bracing.

Literature reports 42 % success rate in returning to previous sport activities at 6 months' follow-up in conservative treatment [3, 47, 48].

These modest results lead to consider surgical treatment, particularly in high-level athletes as treatment of choice.

UCL repair is considered only in case of avulsion injuries in younger athlete [3, 47], performing surgery soon after injury and having MRI showing complete avulsion from the bone [47].

In adults, also in acute events, it is frequent to find an intrasubstance damage of the UCL, and the reconstruction must be considered.

Autografts or allografts can be used in performing UCL reconstruction.

The palmaris longus, if present, or the hamstring tendons are commonly utilized as graft source.

The original technique was performed in 1986 by Jobe, the "Tommy John procedure," from the name of the pitcher who was operated [49]. Several modifications to the original technique have been described in order to decrease morbidity, avoid ulnar nerve transposition, and obtain a better graft fixation.

A modification involving docking the two ends of the tendon graft into a single blind-ended humeral tunnel and tying the sutures over a humeral bone bridge or fixing graft with interference screws [50] was later described.

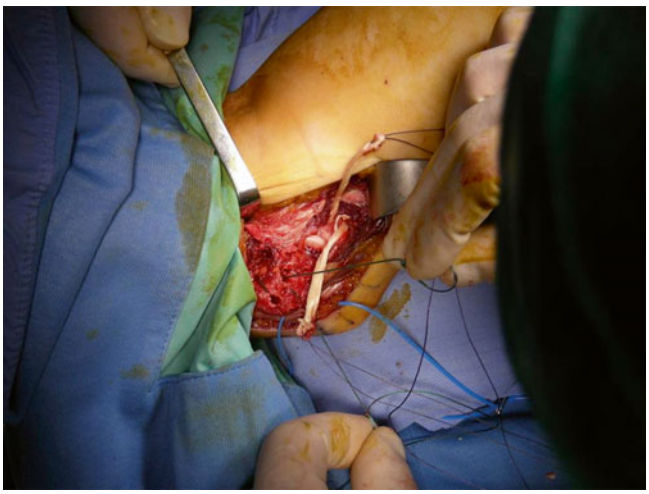


Fig. 29.3 Double-loop graft (semitendinosus) fixation with a 6 mm interference screw in a 7 mm bone tunnel at ulnar level

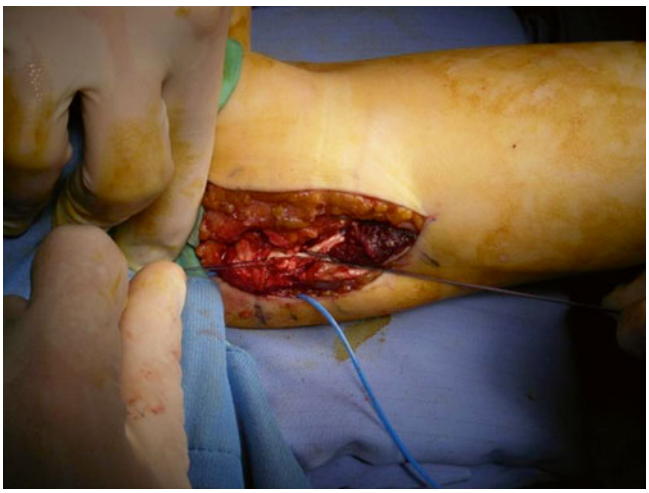


Fig. 29.4 Double-loop graft passed in the 7 mm humeral tunnel and in the 4.5 mm convergent tunnel and then sutured on itself

More recently, we presented a new double-bundle technique [51] in which, using gracilis from homolateral knee, we fix the distal insertion of the UCL using a bioabsorbable screw and the proximal insertion in a 7 mm (diameter) blinded tunnel connected to two 4.5 mm (diameter) divergent tunnels in which every single bundle is passed through (Fig. 29.3). The residual part of the tendon is sutured over itself at different degrees of flexion: anterior bundle at 30° and posterior bundle at 70° (Fig. 29.4).

This technique allows to reconstruct a new ligament tensed in all arc of motion and thick enough to reproduce the original UCL.

Postoperatively, the elbow is positioned in brace for 6 weeks, and rehabilitative protocols start in 2 weeks.

Sport activity progression is initiated at 3–4 months, and return to sport is allowed at 6–8 months post-op.

Reported outcomes of UCL surgery are generally favorable, and in the largest study to date, 83 % of 743 athletes were able to return to a previous or higher level of competition [52]. The most common complications are (often temporary) ulnar or medial antebrachial nerve dysfunction, stiffness, medial epicondyle fracture, and nonspecific elbow pain.

29.8 Treatment of LCL Lesions

After a simple elbow dislocation, most LCL injuries do not require surgical treatment. They are managed initially by splinting with the forearm in pronation to maximize stability. Graduated range of motion exercises are introduced over the first 6 weeks, and in most cases, the torn LUCL will heal and stability will be restored.

Recurrent PLRI after physiotherapy and bracing can require surgical reconstruction using autografts or allograft tendons.

Nestor et al. [53] described in 1992 LUCL reconstruction.

The LUCL is fixed at the origin of the LCL near the tip of the lateral epicondyle and attached to the supinator crest of the ulna.

Many methods of fixation have been described involving multiple osseous tunnels in the humerus and ulna. The grafts are tensioned with the forearm pronated at 30/40° of flexion.

Anterior and posterior lateral capsules can also be imbricated if further stability is required.

Savoie et al. have also described an arthroscopic plication of the posterolateral ligamentous complex with good results [54].

Postoperatively, protocols are based on brace, avoiding terminal 30° of extension for the first 6 weeks.

Sport activity is allowed 6 months after surgery [55].

A largest series of patients reported by Sanchez-Sotelo J et al. [56] showed 86 % of 44 patients had a satisfactory outcome after LUCL reconstruction or repair.

Better results were seen in patients with reconstruction using a tendon graft rather than repair.

Conclusions

Elbow instabilities in athletes comprise a large of spectrum pathologies from medial instabilities in throwing athlete to traumatic posterolateral rotatory instability.

A better comprehension of biomechanical factors and the development of radiological studies allow authors to increase the quality of the diagnosis and the result of surgical treatments.

The poor result of the conservative treatment indicates surgical approach as the treatment of choice in UCL and LCL lesions in athletes.

References

1. Prasad A, Robertson DD, Sharma GB et al (2003) Elbow: the trochleogingylomoid joint. Semin Musculoskelet Radiol 7(1):19–25
2. Morrey BF, Askew LJ, Chao EY (1981) A biomechanical study of normal functional elbow motion. J Bone Joint Surg Am 63(6):872–877
3. Safran MR, Baillargeon D (2005) Soft-tissue stabilizers of the elbow. J Shoulder Elbow Surg 14(1 Suppl S):179S–185S
4. Mehlhoff TL, Noble PC, Bennett JB, Tullos HS (1988) Simple dislocation of the elbow in the adult. Results after closed treatment. J Bone Joint Surg Am 70(2):244–249
5. Murthi AM, Keener JD, Armstrong AD, Getz CL (2011) The recurrent unstable elbow: diagnosis and treatment. Instr Course Lect 60:215–226
6. Hildebrand KA, Patterson SD, King GJ (1999) Acute elbow dislocations simple and complex. Orthop Clin North Am 30:63–79
7. Morrey BF, An KN (1983) Articular and ligamentous contributions to the stability of the elbow joint. Am J Sports Med 11(5):315–319
8. Callaway GH, Field LD, Deng XH, Torzilli PA, O'Brien SJ, Altchek DW, Warren RF (1997) Biomechanical evaluation of the medial collateral ligament of the elbow. J Bone Joint Surg Am 79(8):1223–1231
9. Eygendaal D, Olsen BS, Jensen SL, Seki A, Sojbjerg JO (1999) Kinematics of partial and total ruptures of the medial collateral ligament of the elbow. J Shoulder Elbow Surg 8(6):612–616
10. Morrey BF, Tanaka S, An KN (1991) Valgus stability of the elbow. A definition of primary and secondary constraints. Clin Orthop Relat Res 265:187–195
11. Morrey BF, An KN (1985) Functional anatomy of the ligaments of the elbow. Clin Orthop 201(Dec):84–90
12. Regan WD, Korinek SL, Morrey BF et al (1991) Biomechanical study of ligaments around the elbow joint. Clin Orthop 271:170–179
13. Miyake J, Moritomo H, Masatomi T et al (2012) In vivo and 3-dimensional functional anatomy of the anterior bundle of the medial collateral ligament of the elbow. J Shoulder Elbow Surg 21(8):1006–1012
14. Floris S, Olsen BS, Dalstra M et al (1998) The medial collateral ligament of the elbow joint: anatomy and kinematics. J Shoulder Elbow Surg 7(4):345–351
15. Fuss FK (1991) The ulnar collateral ligament of the human elbow joint: anatomy, function and biomechanics. J Anat 175(Apr): 203–212
16. Ochi N, Ogura T, Hashizume H et al (1999) Anatomic relation between the medial collateral ligament of the elbow and the humero-ulnar joint axis. J Shoulder Elbow Surg 8(1):6–10
17. Martin BF (1958) The annular ligament of the superior radio-ulnar joint. J Anat 92(3):473–482
18. Dunning CE, Zarzour ZD, Patterson SD, Johnson JA, King GJ (2001) Ligamentous stabilizers against posterolateral rotatory instability of the elbow. J Bone Joint Surg Am 83-A(12):1823–1828
19. McAdams TR, Masters GW, Srivastava S (2005) The effect of arthroscopic sectioning of the lateral ligament complex of the elbow on posterolateral rotator stability. J Shoulder Elbow Surg 14(3):298–301
20. Jensen SL, Olsen BS, Seki A, Ole Sojbjerg J, Sneppen O (2002) Radiohumeral stability to forced translation: an experimental analysis of the bony constraint. J Shoulder Elbow Surg 11(2):158–165
21. Park MC, Ahmad CS (2004) Dynamic contributions of the flexor-pronator mass to elbow valgus stability. J Bone Joint Surg Am 86-A(10):2268–2274
22. Morrey BF (1998) Complex instability of the elbow. Instr Course Lect 47:157–164
23. O'Driscoll SW, Morrey BF, Korinek S, An KN (1992) Elbow subluxation and dislocation. A spectrum of instability. Clin Orthop Relat Res 280:186–197

24. Sheps DM, Hildebrand KA, Boorman RS (2004) Simple dislocations of the elbow: evaluation and treatment. Hand Clin 20:389–404
25. Lill H, Korner J, Rose T et al (2001) Fractures-dislocations of the elbow joint: strategy for treatment and results. Arch Orthop Trauma Surg 121:31–37
26. Josefsson PO, Gentz CF, Johnell O et al (1987) Surgical versus non surgical treatment of ligamentous injuries following dislocation of the elbow joint: a prospective randomized study. J Bone Joint Surg Am 69:605–608
27. Josefsson PO, Gentz CF, Johnell O et al (1989) Dislocations of the elbow and intraarticular fractures. Clin Orthop Relat Res 246: 126–130
28. Sojbjerg JO, Helmig P, Kjaersgaard-Andersen P (1989) Dislocation of the elbow: an experimental study of the ligamentous injuries. Orthopedics 12:461–463
29. Deutch SR, Olsen BS, Jensen SL et al (2003) Ligamentous and capsular restraints to experimental posterior elbow joint dislocation. Scand J Med Sci Sports 13:311–316
30. Deutch SR, Jensen SL, Olsen BS et al (2003) Elbow joint stability in relation to forced external rotation: an experimental study of the osseous constraint. J Shoulder Elbow Surg 12:287–292
31. McKee MD, Schemitsch EH, Sala MJ, O'Driscoll SW (2003) The pathoanatomy of lateral ligamentous disruption in complex elbow instability. J Shoulder Elbow Surg 12(4):391–396
32. O'Driscoll SW, Bell DF, Morrey BF (1991) Posterolateral rotatory instability of the elbow. J Bone Joint Surg Am 73(3):440–446
33. Cohen MS, Hastings H II (1998) Acute elbow dislocation: evaluation and management. J Am Acad Orthop Surg 6:15–23
34. Jungbluth P, Frangen TM, Arens S et al (2006) The undiagnosed Essex-Lopresti injury. J Bone Joint Surg Br 88:1629–1633
35. Safran MR (2004) Ulnar collateral ligament injury in the overhead athlete: diagnosis and treatment. Clin Sports Med 23: 643–663
36. Safran MR, Caldwell GL, Fu FH (1996) Chronic instability of the elbow. In: Peimer CA (ed) Surgery of the hand and upper extremity. McGraw-Hill, New York, pp 467–490
37. O'Driscoll SWM, Lawton RL, Smith AM (2005) The moving valgus stress test for medial collateral ligament tears of the elbow. Am J Sports Med 33:231–239
38. Safran MR, Mcgarry MH, Shin S et al (2005) Effects of elbow flexion and forearm rotation on valgus laxity of the elbow. J Bone Joint Surg Am 87:2065–2074
39. Azar FM, Andrews JR, Wilk KE et al (2000) Operative treatment of ulnar collateral ligament injuries of the elbow in athletes. Am J Sports Med 28:16–23
40. Thompson W (2001) Ulnar collateral ligament reconstruction in athletes: muscle-splitting approach without transposition of the ulnar nerve. J Shoulder Elbow Surg 10:152–157
41. Timmerman LA, Schwartz ML, Andrews JR (1994) Preoperative evaluation of the ulnar collateral ligament by magnetic resonance imaging and computed tomography arthrography. Evaluation in 25 baseball players with surgical confirmation. Am J Sports Med 32:26–31
42. Regan W, Lapner PC (2006) Prospective evaluation of two diagnostic apprehension signs for posterolateral instability of the elbow. J Shoulder Elbow Surg 15:344–346
43. Arvind CHV, Hargreaves DG (2006) Tabletop relocation test: a new clinical test for posterolateral rotatory instability of the elbow. J Shoulder Elbow Surg 15:707–708
44. Mehta JA, Bain GI (2004) Posterolateral rotatory instability of the elbow. J Am Acad Orthop Surg 12(6):405–415
45. Potter HG, Weiland AJ, Schatz JA et al (1997) Posterolateral rotatory instability of the elbow: usefulness of MR imaging in diagnosis. Radiology 204:185–189
46. Deml C, Arora R, Oberladstatter J et al (2007) Functional therapy and the limitations for acute elbow dislocation. Unfallchirurg 110: 845–851
47. Freehill MT, Safran MR (2011) Diagnosis and management of ulnar collateral ligament injuries in throwers. Curr Sports Med Rep 10(5):271–278
48. Rettig AC, Sherrill C, Snead DS, Mendler JC, Mieling P (2001) Nonoperative treatment of ulnar collateral ligament injuries in throwing athletes. Am J Sports Med 29(1):15–17
49. Jobe FW, Stark H, Lombardo SJ (1986) Reconstruction of the ulnar collateral ligament in athletes. J Bone Joint Surg Am 68(8): 1158–1163
50. Ahmad CS, Lee TQ, ElAttrache NS (2003) Biomechanical evaluation of a new ulnar collateral ligament reconstruction technique with interference screw fixation. Am J Sports Med 31(3):332–337
51. Pederzini L, Prandini M, Tosi M, Nicoletta F (2012) The acute lesions of the medial collateral ligament of the elbow. GIOT 38(Suppl 2):14–18
52. Cain EL, Andrews JR, Dugas JR et al (2010) Outcome of ulnar collateral ligament reconstruction of the elbow in 1281 athletes: results in 743 athletes with minimum 2-year follow-up. Am J Sports Med 38(12):2426–2434
53. Nestor BJ, O'Driscoll SW, Morrey BF (1992) Ligamentous reconstruction for posterolateral rotator instability of the elbow. J Bone Joint Surg Am 74(8):1235–1241
54. Savoie FH, Holt MS, Field LD, Ramsey JR (2008) Arthroscopic management of posterior instability: evolution of technique and results. Arthroscopy 24(4):389–396
55. Cheung EV (2008) Chronic lateral elbow instability. Orthop Clin North Am 39(2):221–228, vi–vii
56. Sanchez-Sotelo J, Morrey BF, O'Driscoll SW (2005) Ligamentous repair and reconstruction for posterolateral rotatory instability of the elbow. J Bone Joint Surg Br 87(1):54–61

Wrist Ligament Injuries

30

Giulio Pezzella, Andrea Burini, and Marcello Nava

30.1 Introduction

Wrist trauma during sporting activities, whether direct or due to a force transmitted to the wrist, may result in several types of ligament injuries. The most frequently affected ligaments are the s*capholunate* (SL*)* or, more rarely, the *lunotriquetral* (LT) ligaments, which provide stability to the proximal carpal row, and the *triangular fibrocartilage complex* (TFCC), which stabilises the *distal radioulnar joint* (DRUJ). SL and LT tears may lead to *carpal instability*, whereas a TFCC tear may cause *instability of the distal radioulnar joint*.

30.2 Carpal Instabilities

30.2.1 Aetiology

30.2.1.1 Direct Mechanism

A very rare mechanism of injury in sport arises when the wrist is trapped under a forceful pressure as what occurs in a crushing injury [1]. In this situation, the carpal arch is crushed and the carpal bones may dissociate with consequent damage to the intrinsic ligaments (SL and LT).

30.2.1.2 Indirect Mechanism

The main cause of injury to the intrinsic ligaments is an indirect trauma to the wrist due to a distortion during sport activity. This may occur in sports directly involving the wrist and with a risk of distortion (e.g., contact sports) or be the result of an accidental fall in other types of sports.

G. Pezzella (✉) • A. Burini • M. Nava
Orthopaedic Equipe in Presidio Chirurgico Capitanio, Istituto Auxologico Italiano, Via Mercalli 30, Milano 20122, Italy
e-mail: giuliopezzella@libero.it; And.burini@libero.it; Mnava.md@gmail.com

30.2.2 Injury Mechanism

Often the wrist is subjected to strain during hyperextension as what happens in falls onto the palm of an outstretched hand. This type of strain most commonly results in a fracture of the scaphoid or distal radius, which may be associated with injury to the SL ligament [2, 3]. Isolated injuries to the SL and LT ligaments mainly occur in a violent trauma with ulnar deviation and radiocarpal supination [4]. Injuries to these ligaments fall into the broader concept of *progressive perilunate instability* [5, 6], which comprises four stages (Table 30.1-A).

Reagan et al. [7] suggested a second theory in which the wrist, following a distortion in hyperextension and radial deviation, is subjected to stress in the ulnar compartment, with resulting injury to the LT (*reverse progressive perilunate instability*). In this case the LT tear is stage I (Table 30.1-B)

However, the most common ligamentous injury in the wrist is an SL tear resulting in SL instability. If left untreated, this condition may give rise to several clinical forms [8] (Table 30.2).

30.2.3 Clinical and Diagnostic Examinations

30.2.3.1 Clinical Examination

Clinically, a proper diagnosis of SL ligament injury with possible dissociation is not easy to establish, especially if an associated lesion is present.

Table 30.1 The two different theories for the mechanism of injury in LP and LT tears

A. Progressive perilunate dislocation	B. Reverse progressive perilunate dislocation
Stage I. Tear of the SL/scaphoid fracture *Stage II*. Capitolunate dislocation *Stage III*. LT tear/triquetral bone fracture *Stage IV*. Lunate dislocation, in turn classified into three types (type 1, 2 or 3) based on extent	*Stage I*. Tear of the LT *Stage II*. Capitolunate dislocation *Stage III*. Tear of the SL

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_30,

Table 30.2 Different grades of an untreated SL dissociation

Predynamic SL dissociation	The SL ligament is only strained or partially torn
Dynamic SL dissociation	Complete destruction of the entire SL ligament which appears still repairable; the secondary scaphoid stabilisers (scaphotrapezial-trapezoid and radioscaphocapitate ligaments) are intact or only slightly damaged. The cartilage is intact
Static reducible SL dissociation	The ligament stumps are degenerated and not repairable; the secondary stabilisers are starting to collapse with resulting permanent (static instability), but reducible, instability. The cartilage is still intact
Static irreducible (fixed) SL dissociation	Fibrosis develops in the space between the scaphoid and surrounding bones. In these cases the instability cannot be reduced. The cartilage is still intact
Osteoarthritis secondary to SL dissociation (scapholunate advanced collapse, *SLAC wrist*)	The capitate migrates between the scaphoid and lunate as a result of the SL dissociation. The scaphoid, dislocated radially, creates an overload with the articular facet of the corresponding radius, with resulting damage to the cartilage

In the acute phase, the wrist presents with swelling and dorsal pain located 1 cm distal to Lister's tubercle on the SL ligament. The grip is painful and weakened and joint motion is painful, especially in hyperextension. Palpation of the anatomical snuff box may induce pain, because of the radio-scaphoid overload due to the altered congruity between the scaphoid and radius resulting from instability. A clinical test that proves helpful in the diagnosis is the Watson test (scaphoid shift test). This is done by holding the wrist in neutral position and pressing on the scaphoid tubercle while moving the wrist in the radial and ulnar direction: the test is positive if it causes pain and a clunking sensation [1, 8].

30.2.3.2 Imaging

Radiography

A well-performed radiographic evaluation may provide valuable assistance in the diagnosis of SL dissociation. Standard radiography is unable to provide direct visualisation of the intrinsic ligaments, but dislocation of the carpal bones due to dissociation may provide a clear diagnosis.

Several parameters exist that suggest scapholunate dissociation on wrist radiography. In our opinion, the most significant are (Fig. 30.1):

- *Increased SL space (Terry Thomas sign)*: seen on PA views when the space between the lunate and scaphoid is increased compared to the contralateral wrist. The space may increase on PA views obtained with clenched fist.
- *Scaphoid ring*: in the presence of scapholunate dissociation, the scaphoid flexes abnormally and the scaphoid tuberosity appears ring-shaped on PA views.
- *Increased scapholunate angle*: on the lateral view the angle formed by the lunate and scaphoid axes varies between 30° and 60°. When this exceeds 60°, a scapholunate dissociation should be suspected.

Magnetic Resonance Imaging (MRI)

The MRI permits direct visualisation of the SL ligament, but its specificity is limited, with values between 63 and 86 % [1–9].

Contrast-Enhanced Imaging (MRI Arthrography and CT Arthrography)

The intra-articular injection of contrast material considerably increases sensitivity and specificity as a result of the passage of contrast into the lesion itself. To avoid a 'valve effect' with failure of the contrast medium to pass through the lesion, the contrast should be injected into both the radiocarpal and midcarpal joints (two-compartment examination).

Diagnostic Wrist Arthroscopy

This is considered the gold standard examination for the diagnosis of intrinsic ligament injuries [1].

In addition to providing direct visualisation of the injury, it allows quantification and classification of the lesion. Geissler et al. [10] proposed an arthroscopic classification of SL ligament tears describing four grades of lesion (Table 30.2):

Grade 1 Partial tear of the SL ligament: the angles between the scaphoid and lunate are normal, the probe cannot penetrate the SL space.

Grade 2 Tear of the SL ligament: the angles between the scaphoid and lunate are modified, the tip of the probe passes into the SL space.

Grade 3 Tear of the SL ligament: there is malalignment between the scaphoid and lunate; the probe passes into and rotates in the SL space.

Grade 4 Massive tear of the SL ligament and malalignment between the scaphoid and lunate: the arthroscope passes through the SL space from the radiocarpal to the midcarpal joint and back.

30.2.4 Treatment Strategy

The surgical approach to SL ligament injuries depends on severity.

Whereas the more severe grades (*static irreducible SL dissociation and SLAC lesion*) require 'salvage' procedures,

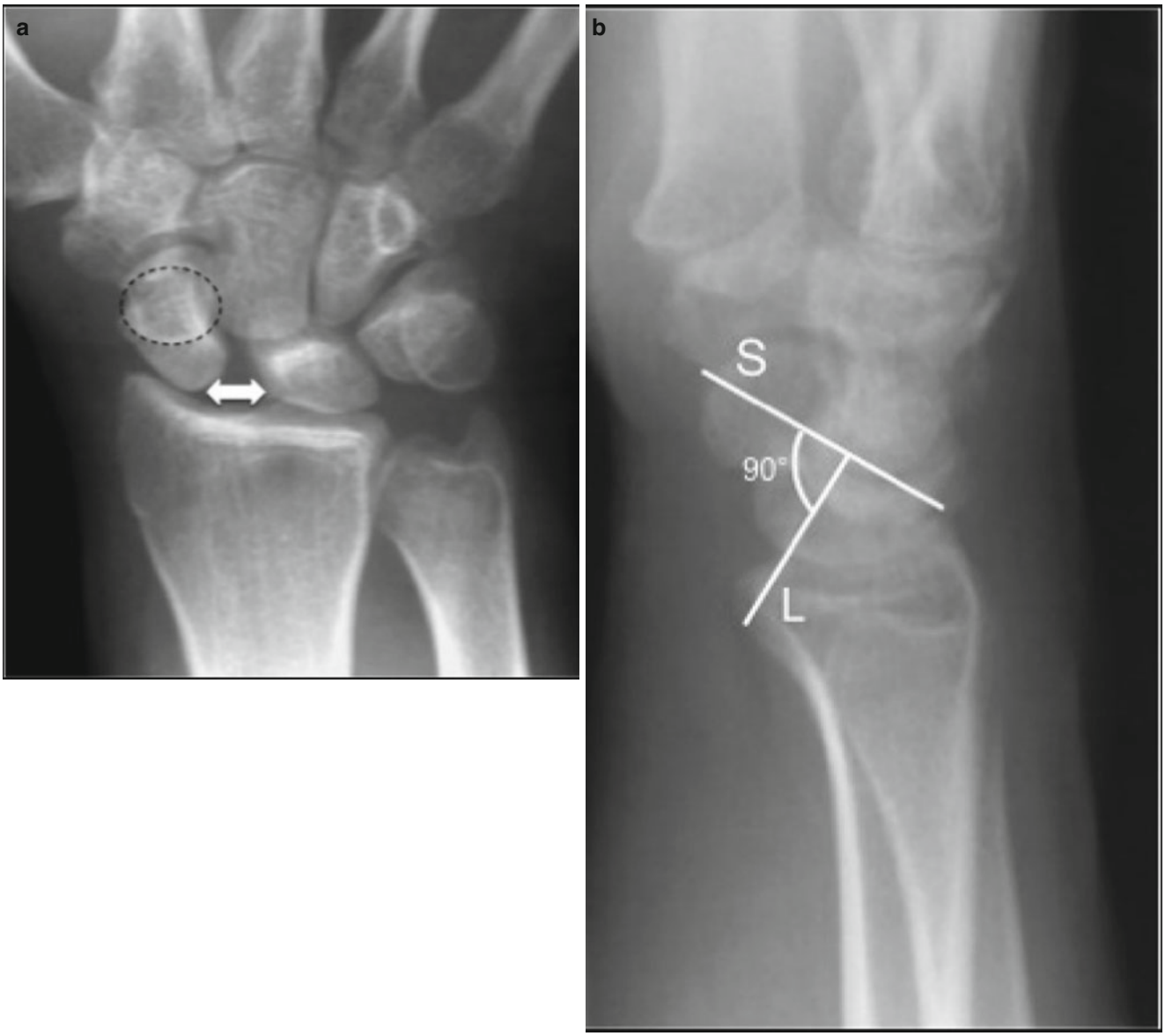


Fig. 30.1 X-ray signs of SL dissociation: scaphoid ring, SL distance and SL angle increase. The *arrow* means an increase of space between the scaphoide and lunate in the scapholunate dissociation. The *circle* is the *scaphoid ring*: appears in the presence of scapholunate dissociation, the scaphoid flexes abnormally and the scaphoid tuberosity appears ring-shaped on PA views

such as partial wrist arthrodesis or resection of the proximal carpal row, earlier grades (*predynamic, dynamic and static reducible SL dissociation*) can be treated with procedures aiming at repairing/rebuilding the SL ligament or restoring, as closely as possible, physiological carpal bone mechanics.

In the case of *predynamic SL dissociation*, up to Geissler's grade 2/3, the SL can be stabilised by using K wires (pinning) in a closed procedure performed under fluoroscopic control or as an arthroscopy-assisted procedure, with excellent results [11]. In these cases, arthroscopy offers the advantage of allowing removal of scar tissue and fibrosis of the ligament tear in order to enhance vascularisation and healing. The K wires are removed after 8–10 weeks. When the membranous portion of the SL is involved, a debridement with shaver or radiofrequency waves should be performed to remove the ligament flap – a possible cause of painful impingement – and relieve the pain [12].

In *dynamic SL dissociation,* if the lesion is recent and therefore has good healing capability, direct repair of the ligament using various techniques is indicated [1–8]. It should be noted that injured intrinsic ligaments tend to degenerate very rapidly, and the optimal timing for repair is within 2 weeks, after which the likelihood of recovery decreases dramatically (the repair procedure should not be

attempted after 4 weeks). After 2 weeks, capsulo-ligament reconstruction procedures can be carried out that aim either to avoid rotatory subluxation of the scaphoid [13] due to collapse of the secondary stabilisers or to strengthen the dorsal portion of the injured ligament [8]. Another proposed procedure is reconstruction of the dorsal portion of the SL ligament by using grafts fixed with bone-to-bone technique [14].

Wahegaonkar and Mathoulin [15] described an arthroscopic technique combining through suturing the dorsal portion of the scapholunate with capsulodesis with the *dorsal interosseous ligament* (DIL). The arthroscope is placed in the *ulnar midcarpal* (UMC) portal, and using a needle introduced through portal 3–4, a PDS 2/0 suture thread is passed through the DIL and lunate remnant of the torn SL and pulled out through the *radial midcarpal* (RMC) portal; the procedure is repeated for the scaphoid remnant of the SL. The two ends of the suture threads exiting from the RMC portal are tied into a knot and withdrawn within the midcarpal joint; finally, the proximal ends of the suture threads exiting from portal 3–4 are also tied, blocking the SL stumps with the DIL. According to the authors, this treatment should be reserved for cases with moderate diastasis, and the results appear encouraging. Del Pinal [16] proposed a similar technique, which uses a volar portal placed immediately ulnar to the *flexor carpi radialis* (FRC) *tendon*, to suture the volar portion of the SL ligament. Although these relatively recent techniques show encouraging preliminary results, they both require validation by studies carried out on larger series and with more adequate follow-up periods.

The techniques proposed for *static reducible SL dissociation* aim to treat rotatory subluxation of the scaphoid that has become stabilised in an attempt to restore the normal articular relations between the radius and scaphoid, altered by the SL dissociation. The most commonly used is the Brunelli technique [17] which has undergone numerous modifications over the years [8, 18]. In this technique, the scaphoid subluxation is reduced and stabilised in the correct position with the aid of a strip of the FRC tendon passed from palmar to dorsal through a hole in the distal pole of the scaphoid and secured dorsally first onto the scapholunate joint and then onto the radius. In static reducible SL dissociation, surgical arthroscopy has only limited efficacy: one option is to perform an arthroscopically assisted *reduction and association of the scaphoid and lunate* (RASL), described as an open procedure [19]. The technique involves stabilising the SL with a Herbert screw along the rotation axis with arthroscopic assistance. Arthroscopy offers the advantage of allowing debridement of fibrous tissue and thus greater chances of healing. However, the minimal error in centring the rotation axis or the prolonged permanence of the screw can cause serious damage at the articular level [20].

30.3 Instability of the Distal Radioulnar Joint

30.3.1 Aetiology

The *distal radioulnar joint* (DRUJ) is the distal joint of the articular system of the forearm, which allows pronation-supination. It is a joint with limited bony congruency, and it is stabilised by several anatomical structures [21, 22]: among them, the *triangular fibrocartilage complex* (TFCC) is considered the primary stabiliser. The complex structure of the TFCC comprises two ligaments that serve as true stabilisers for the DRUJ: the *palmar radioulnar ligament* (PRUL) and the *dorsal radioulnar ligament* (DRUL) (Fig. 30.2).

The TFCC inserts onto the ulnar aspect with two separate components [23]: a *proximal component* that inserts on the fovea ulnaris (pc-TFCC) and a *distal component* that inserts on the capsule and sheath of the extensor carpi ulnaris (dc-TFCC). Rupture of these two structures may lead to DRUJ instability and ulnar-sided wrist pain.

30.3.2 Injury Mechanism

A direct trauma to the ulnar side of the wrist rarely causes a TFCC injury. The mechanism leading to a TFCC tear is often indirect and related to a distortion with stress in hyperextension or abrupt rotation (forced pronation-supination). In the

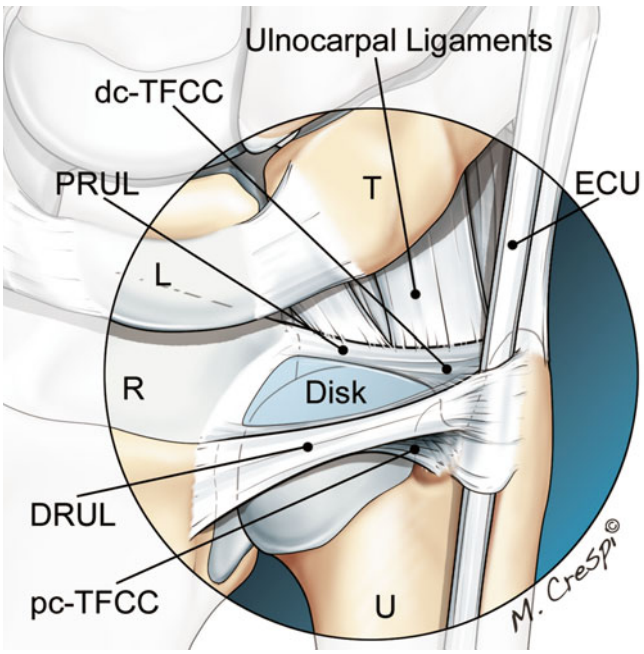


Fig. 30.2 Anatomy of the TFCC. Extensor carpi ulnaris (*ECU*) tendon, palmar radioulnar ligament (*PRUL*), and dorsal radioulnar ligament (*DRUL*). The TFCC inserts: proximal component (*pc-TFCC*), distal component (*dc-TFCC*), *T* Triquetrum, *L* Lunate, *R* Radius, *U* Ulna

former case, there may be an associated fracture of the distal epiphysis of the radius [3], whereas in the latter, the lesion tends to be isolated. Often, especially in sport activities that stress the wrist (tennis, golf, baseball, gymnastics, etc.), a TFCC injury may arise [24, 25] as a result of wrist overload during a rotational movement.

TFCC injuries have been classified by Palmer [26]. This classification makes a distinction between *traumatic* (type 1) and *degenerative* (type 2) lesions. *Traumatic lesions* are divided into:

1A central perforation of the disc

1B ulnar avulsion, divided into two subtypes:

- *Ligamentous*: a tear of the ulnar insertion of the TFCC, which may affect both the capsular and foveal insertions (dc-TFCC and pc-TFCC)
- *Bony:* avulsion fracture of the ulnar styloid

1C palmar avulsion: rare injury due to a direct trauma on the ulnar side of the wrist and affecting the ulnar-carpal ligaments

1D radial avulsion, divided into two subtypes:

- *Ligamentous*: disinsertion of the TFCC from the ulnar side of the radius
- *Bony:* detachment of a bony fragment of the ulnar portion of the radius

Although the most frequently used, this classification is exclusively based on a topographical description of the lesion, without considering the degree of DRUJ instability. For this reason, Atzei [27, 28] proposed a further classification based on clinical, radiological and arthroscopic findings. This classification subdivides type 1B injuries into six subtypes according to which TFCC component is involved (dc-TFCC, pc-TFCC) and thus to the degree of instability and provides a treatment algorithm for each type.

30.3.3 Clinical and Diagnostic Examination

30.3.3.1 Clinical Examination

Patients with a TFCC tear report intense pain on the ulnar side of the wrist, at the level of the ulnar head, which irradiates proximally towards the forearm and is exacerbated during rotation. Clinically, the pain is elicited by forced supination with hyperextended wrist and flexed elbow, as if the patient were carrying a tray (*waiter's test* or *tray test*). Another sign is tenderness on applying lateral pressure to the wrist, ulnar to the *extensor carpi ulnaris* (ECU) *tendon*, on the cutaneous projection of the fovea [29]. In cases of instability, there is dorsal subluxation of the ulnar head which decreases when pressure is applied (*piano key test*). Instability is also assessed with ballottement of the ulnar head relative to the radius in neutral position, during supination and pronation. Rarely, and in cases with severe instability, these manoeuvres can cause complete dislocation of the ulnar head, which may be palmar in the event of a DRUL tear and dorsal in the event of a PRUL tear.

30.3.3.2 Imaging

Radiography

Standard radiography is unable to provide direct visualisation of a TFCC tear, with the exception of an ulnar styloid fracture in a Palmer's type *1B bony lesion* or a fracture of the ulnar portion of the radius in a type *1D bony lesion*. However, in cases of instability, it is possible to detect an increase in the distance between the radius and ulna in the DRUJ on PA views and a subluxation of the ulna on lateral views.

Magnetic Resonance Imaging (MRI)

MRI is the most frequently used imaging modality for the diagnosis of TFCC lesions. Although the examination allows direct visualisation of anatomical structure, its use for a proper diagnosis, especially in ulnar avulsions, remains controversial, as it has low sensitivity and specificity compared with arthroscopy [30, 31]. This gap does, however, tend to shorten when high-resolution MRI is used [32].

Contrast-Enhanced Imaging (MRI Arthrography and CT Arthrography)

In TFCC tears, contrast material injected into the joint passes through the lesion and therefore provides indirect visualisation. This method, applied to MRI and CT *(MRI arthrography and CT arthrography),* has undoubtedly improved the results compared to non-contrast-enhanced studies, achieving both sensitivity and specificity values close to those of arthroscopy [33, 34].

30.3.4 Treatment Strategy

Arthroscopy is currently considered the gold standard for the diagnosis and correct classification of TFCC injuries. Also from a surgical standpoint, arthroscopy has proved to be a highly valuable technique. The treatment of TFCC injuries varies according to the type of lesion. Irrespective of the different classifications [26–28], a TFCC injury may involve the disc or the peripheral portion. The TFCC disc represents the central, avascular portion (hence with no possibility of healing), which serves as a spacer and 'shock absorber' for the axial load between the ulnar head and the carpal bones. In the case of rupture, the pain is caused by impingement of the flap that is produced between these bony segments, and the surgical procedure consists in removing the flap and smoothing the edge of the lesion, using wrist-specific basket, burr and radiofrequency device.

Peripheral TFCC lesions involve the purely ligamentous portion of the structure (DRUL and PRUL). The most common peripheral lesion is a tear of the ulnar insertion (Palmer's type 1B), with involvement of either one or both the TFCC components (dc-TFCC and pc-TFCC). On this basis, the resulting DRUJ instability may be more or less severe. If only

Fig. 30.3 Arthroscopic view of a double suture of the TFCC using all-inside technique

the distal portion is involved (dc-TFCC), it is possible to perform an arthroscopic suture of the TFCC with the capsule. The techniques described in the literature are numerous, but they may be divided into two categories: those involving tying the suture knot on the capsule externally and those involving an intra-articular suture knot on the TFCC. In the first case, we use the technique reported by Stanley [35], which involves a small ulnar incision and the isolation of the sensitive dorsal branch of the ulnar nerve. Through this incision, the suture is performed with a PDF 2/0 thread and the aid of two needles: one for passing a single thread and the other for passing a loop to catch the first. Suturing may be done with horizontal mattress sutures or vertical sutures over the tear and may involve one or several stitches, depending on the extent of the lesion. The disadvantage of this technique is pain on the ulnar portion produced by the knot. For this reason, in recent years other suturing techniques have been reported involving the creation of an external loop over the capsule and tying of the knot intra-articularly on the TFCC (all-inside technique) [36, 37]. Even in this case, the sutures may be mattress sutures or vertical sutures over the tear and may involve one or several stitches (Fig. 30.3).

In type 1B unstable ligamentous lesions, with involvement of both TFCC components (dc-TFCC and pc-TFCC) or with pseudoarthrosis of the ulnar styloid (chronic Palmer's type 1B bony lesion), it is recommended to reinsert the TFCC into the fovea. In the case of pseudoarthrosis, the styloid fragment is removed and the TFCC is reinserted arthroscopically with an anchor [38]. The technique involves inserting the anchor into the fovea and passing the threads in the TFC with monofilaments, used as carriers. Once passed through the TFC, the threads are grasped and pulled out of the joint through portal 6R. A gliding knot is then tied and a knot pusher is used to tighten it in the joint close to the TFCC. In the case of a ligamentous lesion with involvement of both insertions, the TFCC is reinserted into the fovea with an anchor using the same technique as described above.

If the type 1B bony lesion does not yet present pseudoarthrosis and the bony tissue has good healing capability, an open styloid synthesis procedure is recommended, after arthroscopic assessment of TFCC integrity.

30.3.5 Rehabilitation and Return to Play

The aim of rehabilitation is to obtain a functional range of wrist motion as close to normal as possible and to restore stability of the DRUJ, RCJ and MCJ, by acting above all on the neurophysiological and proprioceptive levels. The same concepts developed for other joints can also be applied to the wrist [39], and the following schedule can be helpful in achieving these goals [40]:

- Respect the biological healing time of repaired ligaments while trying to prevent the development of joint stiffness
- Restore functional range of motion taking care to respect joint stability
- Restore gross and fine grasp
- Restore functional proprioception and coordination of wrist movements

Irrespective of complete biological healing which takes 3–6 months in ligamentous lesions of the wrist, return to play is closely related to these principles, which concern above all restoring the physiological range of motion and re-establishing proprioception.

References

1. Luchetti R, Pegoli L, Papini Zorli I, Garcia Elias M (2007) Le instabilità del carpo. In: Trattato di chirurgia della mano. Verduci Editore, Rome, pp 117–157
2. Bell MJ (1983) Perilunar dislocation of the carpus and associated Colles fracture. Hand 15:262–266
3. Lindau T, Arner M, Hagberg L (1997) Intraarticular lesions in distal fractures of the radius in young adults. A descriptive arthroscopic study in 50 patients. J Hand Surg Br 22(5):638–643
4. Mayfield JK (1980) Mechanism of carpal injuries. Clin Orthop 149:45–54
5. Mayfield JK, Johnson RP, Kilcoyne RF (1976) The ligaments of human wrist and their significance. Anat Rec 186:417–426
6. Wagner CJ (1956) Perilunar dislocations. J Bone Joint Surg 38A:1198–1230

7. Reagan DS, Linscheid RL, Dobyns JH (1984) Lunotriquetral sprains. J Hand Surg Am 9A:502–514
8. Garcia-Elias M, Geissler WB (2004) Carpal instability. In: Green DP, Hotchkiss RN, Pederson WC, Wolfe SW (eds) Green's operative hand surgery, 5th edn. Elsevier, Philadelphia, pp 535–604
9. Schädel-Höpfner M, Iwinska-Zelder J, Braus T, Bohringer G, Klose KJ, Gotzen L (2001) MRI versus arthroscopy in the diagnosis of scapholunate ligament injury. J Hand Surg Br 26 B:17–21
10. Geissler WB, Freeland AE, Savoie FH, McIntyre LW, Whipple TL (1996) Intracarpal soft-tissue lesions associated with intra-articular fracture of the distal end of the radius. J Bone Joint Surg 78A:357–365
11. Linscheid RL (1984) Scapholunate ligamentous instabilities (dissociations, subdislocations, dislocations). Ann Chir Main 3:323–330
12. Weiss APC, Sachar K, Glowacki KA (1997) Arthroscopic debridement alone for intercarpal ligament tears. J Hand Surg Am 22A:344–349
13. Blatt G (1994) Dorsal capsulodesis for rotatory subluxation of the scaphoid. In: Gelberman RH (ed) Master techniques in orthopedic surgery: the wrist. Raven Press, New York, pp 147–166
14. Weiss APC (1998) Scapholunate ligament reconstruction using a bone-retinaculum-bone autograft. J Hand Surg Am 23A:205–215
15. Wahegaonkar AL, Mathoulin CL (2013) Arthroscopic dorsal capsulo-ligamentous repair in the treatment of chronic scapholunate ligament tears. J Wrist Surg 2(2):141–148
16. Del Piñal F (2013) Arthroscopic volar capsuloligamentous repair. J Wrist Surg 2(2):126–128
17. Brunelli GA, Brunelli GR (1995) A new surgical technique for carpal instability with scapho-lunar dislocation: Eleven cases. Ann Chir Main Memb Super 14:207–213
18. Van Den Abbeele KLS, Loh YC, Stanley JK, Trail IA (1998) Early results of a modified Brunelli procedure for scapholunate instability. J Hand Surg Br 23B:258–261
19. Rosenwasser MP, Strauch RJ, Miyasaka KC (1997) The RASL procedure: reduction and association of the scaphoid and lunate using the Herbert screw. Tech Hand Up Extrem Surg 1:263–272
20. Cognet JM, Levadoux M, Martinache X (2011) The use of screws in the treatment of scapholunate instability. J Hand Surg Eur Vol 36(8):690–693
21. Kihara H, Short WH, Werner FW, Fortino MD, Palmer AK (1995) The stabilizing mechanism of the distal radioulnar joint during pronation and supination. J Hand Surg Am 20A:930–936
22. Cozzolino R, Luchetti R, Atzei A (2013) Instabilità della radio ulnare distale: concetti generali. Riv Cir Mano Vol 50(2):214–218
23. Nakamura T, Yabe Y, Horiuchi Y (1996) Functional anatomy of the triangular fibrocartilage complex. J Hand Surg Br 21(5):581–586
24. Dwek JR, Cardoso F, Chung CB (2009) MR imaging of overuse injuries in the skeletally immature gymnast: spectrum of soft-tissue and osseous lesions in the hand and wrist. Pediatr Radiol 39(12):1310–1316
25. Baratz ME (2012) Central TFCC tears in baseball players. Hand Clin 28(3):339
26. Palmer AK (1989) Triangular fibrocartilage complex lesions: a classification. J Hand Surg Am 14(4):594–606
27. Atzei A (2009) New trends in arthroscopic management of type 1-B TFCC injuries with DRUJ instability. J Hand Surg Eur Vol 34(5):582–591
28. Atzei A, Luchetti R (2011) Foveal TFCC tear classification and treatment. Hand Clin 27(3):263–272
29. Tay SC, Tomita K, Berger RA (2007) The "ulnar fovea sign" for defining ulnar wrist pain: an analysis of sensitivity and specificity. J Hand Surg Am 32(4):438–444
30. Pederzini L, Luchetti R, Soragni O, Alfarano M, Montagna G, Cerofolini E, Roth JH (1992) Evaluation of the triangular fibrocartilage complex tears by arthroscopy, arthrography and MRI. Arthroscopy 8:191–197
31. Oneson SR, Timins ME, Scales LM, Erickson SJ, Chamoy L (1997) MR imaging diagnosis of triangular fibrocartilage pathology with arthroscopic correlation. AJR Am J Roentgenol 168(6): 1513–1518
32. Potter HG, Asnis-Ernberg L, Weiland AJ, Hotchkiss RN, Peterson MG, McCormack RR Jr (1997) The utility of high-resolution magnetic resonance imaging in the evaluation of the triangular fibrocartilage complex of the wrist. J Bone Joint Surg Am 79(11): 1675–1684
33. Meier R, Schmitt R, Krimmer H (2005) Wrist lesions in MRI arthrography compared with wrist arthroscopy. Handchir Mikrochir Plast Chir 37(2):85–89
34. De Filippo M, Pogliacomi F, Bertellini A, Araoz PA, Averna R, Sverzellati N, Ingegnoli A, Corradi M, Costantino C, Zompatori M (2010) MDCT arthrography of the wrist: diagnostic accuracy and indications. Eur J Radiol 74(1):221–225
35. Stanley J, Saffar P (1994) Wrist arthroscopy. Martin Dunitz, London
36. Pederzini LA, Tosi M, Prandini M, Botticella C (2007) All-inside suture technique for Palmer class 1B triangular fibrocartilage repair. Arthroscopy 23(10):1130.e1–1130.e4
37. Del Piñal F, García-Bernal FJ, Cagigal L, Studer A, Regalado J, Thams C (2010) A technique for arthroscopic all-inside suturing in the wrist. J Hand Surg Eur Vol 35(6):475–479
38. Pezzella G, Zucco P, Drocco L, Sartori A (2001) Ulnar styloid nonunion with DRUJ instability: arthroscopic suture of triangular fibrocartilage (TFC) with Mitek anchor. Artroscopia II(2):108–113
39. Lephart SM, Pincivero DM, Giraldo JL, Fu FH (1997) The role of proprioception in the management and rehabilitation of athletic injuries. Am J Sports Med 25(1):130–137
40. Fairplay T (2013) Instabilità radio-ulnare distale. Riabilitazione neuromuscolare e propriocettiva del polso e della mano. Riv Chir Mano Vol 50(2):227–233

Wrist Tendon Lesions

31

Antoni Salvador Albarracin

31.1 Introduction

The wrist is a complex joint that biomechanically transmits forces generated at the hand through to the forearm. The radial side of the wrist carries 80 % of the axial load and the ulnar side the remaining 20 % of the load. The incidence of wrist (and hand) injuries [1] in the sporting population is approximately 25 %.

The injuries are divided into four categories: overuse, nerve (and vascular), traumatic, and weight-bearing injuries. Overuse injuries are common in sports involving the hand and the wrist, such as racquet sports, golf, netball, basketball, and volleyball. Nerve injuries are more commonly compressive neuropathies and are seen with cyclists who may compress the ulnar nerve in Guyon's canal. Traumatic injuries are the most common and due to either a fall on to the wrist, a direct blow, or combination of a rotatory and torsional force. The weight-bearing injuries are more specific to gymnastics and result from repetitive excessive compressive and rotational forces across the wrist.

Golf and racquet sports are played throughout the world, both at a professional level and for health and recreation. With regard to golf, it was estimated in 2001 that 37 million individuals played 518 million rounds in the United States alone [2]. Major racquet sports around the world consist of tennis, table tennis, squash, and badminton. Tennis is the most common racquet sport; it has been estimated that tens of millions play tennis in the United States alone, and sports such as badminton are popular in Sweden, with 680,000 participating at least once per week during the winter season. With the large numbers of individuals participating in such golf and racquet sports activities, it is understandable that associated injuries are recognized.

A.S. Albarracin
Cirurgia Ortopèdica i Traumatologia,
Fundació Hospital De Sant Celoni,
Av. Hospital, 19, Sant Celoni 08470, Spain
e-mail: salvador2585@gmail.com

31.2 Etiology and Injury Mechanisms

Depending of the kind of sport in practice, we can find some different tendon lesion. A lot of studies have been done about, and the majority of them referred to golf and racquet sport.

31.2.1 Types of Injuries in Golf Players

The prevalence of golf injuries has ranged from 25 to 62 %, with individuals older than 50 years of age or with a low handicap (1–9) having a higher prevalence [2]. The prevalence of injuries of professional golfers has been reported to be as high as 88.5 %, probably related to the number of hours participating in the sport [2]. The etiology of golf injuries can include overuse, technical errors during swing, physical fitness deficiencies (aerobic, muscle strength, flexibility), no pregame warm-up, carelessness or lack of etiquette, and natural environmental conditions (uneven surface, thunderstorms). In amateur golfers, the three most common sites of injury are the thoracolumbar spine (27 %), elbow (26 %), and wrist and hand (16 %) [1]. For the professional golfer, the three most common sites are the wrist (27 %), lumbar spine (23 %), and shoulder (9 %) [1]. Lower extremity injuries are less common than spine or upper extremity injuries in either group [1]. Golf injuries can be considered as two groups: overuse (54.5 %) or acute trauma (45.5 %) [1].

The majority of wrist problems (29–67 % of all problems) involved the leading wrist (87 %). [3]

Symptoms can be described in a variety of anatomical location:

1. Ulnar-sided problems: A cause of pain comprises extensor carpi ulnaris (ECU) tendinosis, ECU subluxation, and ulnar pain after hitting a "fat" shot (where too much turf is taken while playing a shot). ECU subluxation can be acute as a result of an unexpected impact with a tree root or object during a full shot.
2. Radial-sided pain: de Quervain's disease can occur also in the leading wrist, and it has been described an

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_31,

intersection syndrome in a player at the beginning of the season [3].

3. Dorsal wrist pain: Some golfers have ganglia on the dorsum of the wrist, sometimes in the leading wrist and sometimes in the non-leading one. It has been also described [3] extensor tenosynovitis, dorsal rim impaction syndrome (a condition of hypertrophic radiolunate synovitis causing pain on loaded extension in the dorsal central area of the wrist).

The golf swing is traditionally divided into five phases (address, backswing, downswing, impact, and follow-through). The leading, non-dominant wrist (the left wrist for right-handed golfers) begins the golf swing in a position of ulnar deviation when addressing the ball. As the club is lifted away into the backswing, this wrist moves into radial deviation until it sits maximally radially deviated at the top of the backswing. At this point, the club changes direction to begin the downswing, and the leading wrist returns to ulnar deviation until impact. Several biomechanical works have studied the load transmission across the wrist and found an increased in distal ulnar loading with progressively increasing ulnar deviation [3].

31.2.2 Types of Injuries in Tennis Players

The biomechanics of a tennis swing have been extensively studied. In contrast, less has been described pertaining to the other major racquet sports of table tennis, squash, and badminton; however, certain aspects of the tennis swing can apply. The biomechanics of a tennis swing can be divided into three basic strokes: overhead or serve, forehand, and backhand. Significant forces are applied to the upper limb with hand speeds at ball impact approaching 47 miles per hour in world-class tennis players [2].

The tennis serve is composed of four stages: wind-up, cocking, acceleration, and deceleration and follow-through.

Forehand and backhand strokes consist of three stages: racquet preparation, acceleration, and follow-through. The wrist extensors are very active in every stroke. Of note, wrist extensors do not demonstrate increased activity with a single-handed backhand stroke compared with a double-handed backhand stroke.

In tennis players, injuries are typically from overuse or acute trauma.

Tendinitis and stenosing tenosynovitis have been described in association with almost all tendons in the wrist and hand. The mechanism is usually from chronic overstretching or recent initiations of an unaccustomed motion or activity. The symptoms are usually a vague pain radiating along the affected tendon into its insertion and occasionally affecting the joints over which tendons cross.

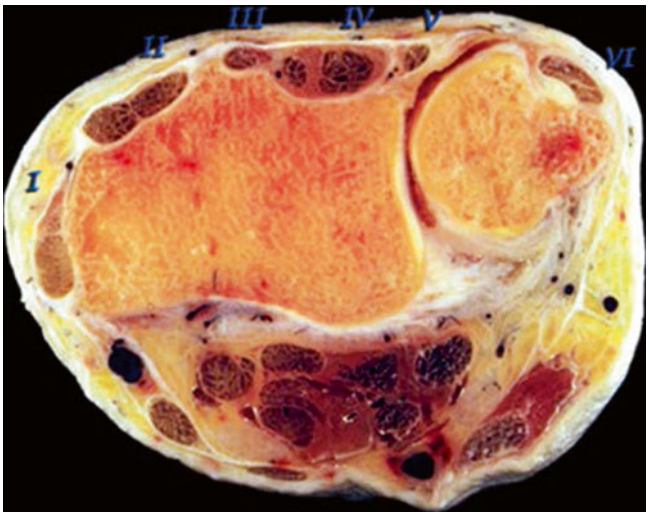


Fig. 31.1 The extensor compartments (with permission by Prof. Ivan Saenz Navarro. Hospital Clinic, Barcelona)

We subdivide wrist tendon lesion by its localization, dorsal or volar (Fig. 31.1):

- Dorsal:
  - De Quervain's tenosynovitis
  - Intersection syndrome
  - Extensor insertional tenosynovitis
  - ECU lesions
- Volar:
  - Flexor carpi radialis (FCR) tenosynovitis
  - Other tendons

31.3 Pathology

31.3.1 De Quervain's Tenosynovitis

The most common form of stenosing tenosynovitis reported in racquet sports is de Quervain's disease [4–7]. It consists of inflammation of the abductor pollicis longus (APL) and extensor pollicis brevis (EPB) as they pass through the first compartment at the level of the radial styloid. Often there are multiple slips of the APL. Repetitive ulnar deviation and grasp can inflame these tendons in this closed space resulting in pain with thumb use. Clinically there is swelling and tenderness along the radial aspect of the wrist. Filkenstein's test is positive, with pain reproduced by having the patient tuck his thumb into the palm and then ulnarly deviate the wrist. This lesion may be bilateral in 30 % of the cases, and it exist a septum between the tendons in approximately 70 %.

The differential diagnosis includes infectious tenosynovitis of the first extensor compartment, first carpometacarpal joint osteoarthritis, FCR tenosynovitis, intersection

syndrome, and isolated neuritis of the superficial radial nerve, also called "Wartenberg's syndrome."

MRI characteristics are fairly typical and include thickening and heterogeneity of the first compartment tendons and sheath, tenosynovial effusion and surrounding loss of fat planes due to soft tissue edema, and low-signal scar tissue (Figs. 31.2a, b).

The majority of the patients will respond of the conservative care. This includes nonsteroidal (NS) medications and immobilization. If this is not successful, then a steroid injection into the canal can be useful, taking care to avoid a dorsal radial sensory nerve's lesion. In recalcitrant cases, surgical release of the first compartment can be curative. Through a transverse skin incision, perform a longitudinal opening of the first compartment, realising the septum if it's present (Fig. 31.3).

Postoperatively, the patient is splinted for 3 weeks and started to use progressively his wrist. Racquet sport should be avoided for 8 weeks. Recently, Kang et al. [4] have published good results using an endoscopic release versus open release. They recommended surgical procedure 3 months after a non-successful conservative treatment including steroid injection, and they conclude that this technique seems to provide earlier improvement after surgery with fewer temporary symptoms of superficial radial nerve injury and greater scar satisfaction, without significant difference in pain score.

31.3.2 Intersection Syndrome

Wrist intersection syndrome or squeaker's wrist is a peritendinous inflammation that occurs between 4 and 8 cm dorsally and proximally to the de Quervain's lesion, exactly where the first extensor compartment tendons cross over the second one which contain extensor pollicis longus tendon (EPL). It's produced generally in sports like rowing, canoeing, playing racket sports [8], horseback riding, and skiing. The correct diagnosis can be difficult clinically, and MRI is very useful showing the presence of inflammation around the first and second extensor compartment [9]. Currently ultrasound can also help us in the diagnosis of this pathology. In chronicle situations, tendons can suffer a total rupture because of the maintained friction. Nonoperative treatment is also useful using NS medications and splint immobilization. Rarely a surgical solution with bursa's excision is needed.

31.3.3 Extensor Insertional Tenosynovitis

Insertional tendinitis can occur in any of the extensor tendons. Most commonly, we can find it affecting the second extensor compartment and the extensor carpi radialis longus (ECRL) and brevis (ECRB). This localization is not commonly

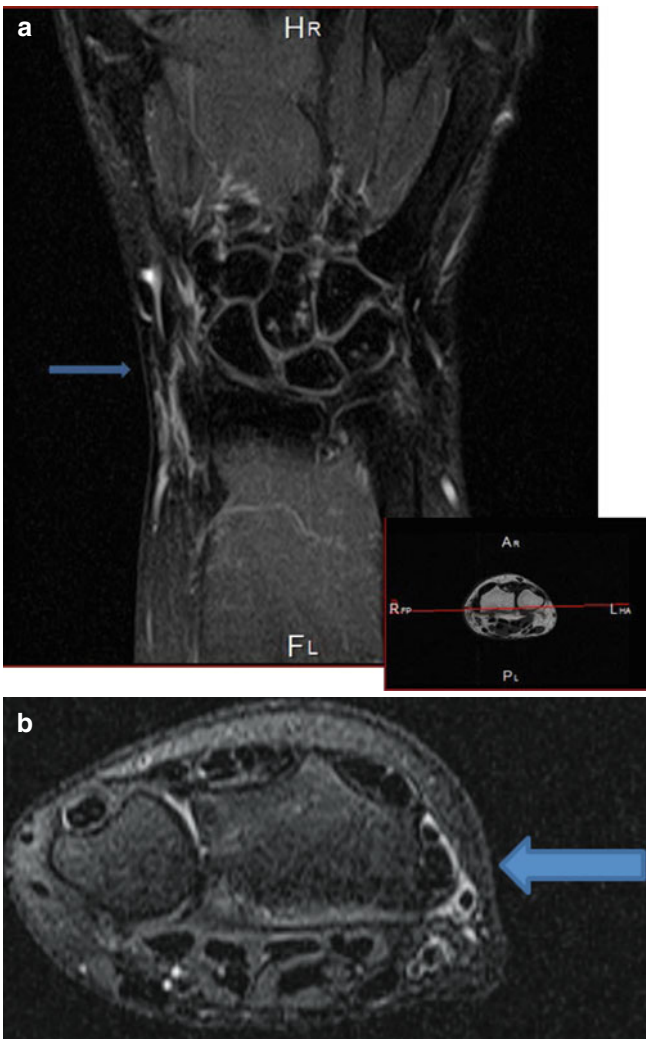


Fig. 31.2 (**a**) De Quervain's tenosynovitis. Coronal fat-suppressed T2-weighted image demonstrates tendon sheath effusion, peritendinous edema, and tendon thickening of the first extensor compartment (*arrow*). (**b**) Axial view. Peritendinous edema at the first extensor compartment (*arrow*)

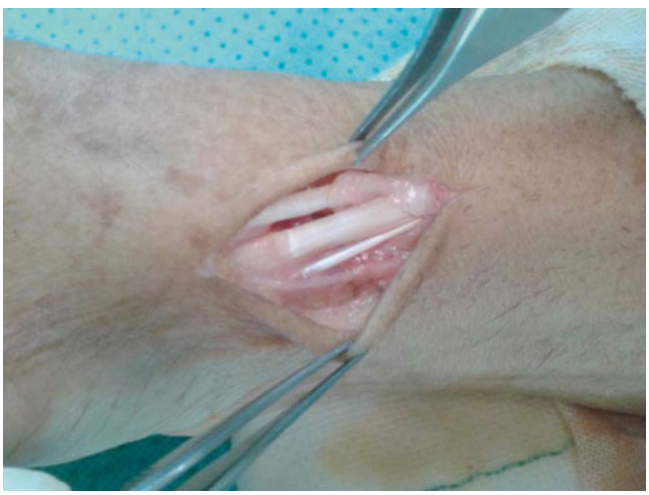


Fig. 31.3 First compartment opened surgically

reported, however, because of primary symptoms at the muscular origin. Classical lateral epicondylitis (tennis elbow) represents a proximal inflammation of the extensor carpal tendons and is more common in the amateur than in the professional tennis player. Elite players are more likely to incur inflammation in the medial epicondyle from flexor origin overload during the serve. Insertional tendinitis in the radial wrist extensors may often be associated with some bony thickening at their respective insertions at the base of the second and third metacarpals. This bony thickening called a "carpometacarpal boss" is occasionally mistaken for a ganglion, but the examiner should be alert to the different consistency of the swelling and its location over the junction of the carpometacarpal joints. Treatment generally follows the classic pattern of rest and nonsteroidal medication. In the digital extensors, tendinitis is most common in the extensor pollicis longus where it abuts Lister's tubercle or in the index and small extensor tendons because of their more oblique course [8].

31.3.4 Extensor Carpi Ulnaris (ECU) Lesions

ECU lesion is one of the pathology included in the so-called ulnar wrist syndrome, where we can find lesions such triangular fibrocartilage complex tears, lunotriquetral instability, distal radioulnar joint pathology, flexor carpi ulnaris tendinitis, and pisotriquetral arthritis.

The ECU tendon passes through the sixth extensor compartment and is held within the groove of the ulnar head by a deep retinaculum, referred as an ECU subsheath. ECU tendinitis or subluxation is seen in sports requiring repetitive wrist motion, such a rowing or racquet sports that require a snap of the wrist [10–12]

ECU tendinopathy and tendon tear can occur with repetitive wrist motion, usually in racquet sports. With 2-handed backhand, the non-dominant wrist will undergo exaggerated ulnar deviation, and the patient may complain of tenderness of the sixth extensor compartment adjacent to the ulnar styloid. The ECU tendon can also sublux if there is traumatic rupture of the ECU subsheath caused by forceful supination of the forearm, flexion, and ulnar deviation of the wrist during tennis, baseball, or other racquet sports. ECU tendon is in risk when the wrist is working in volar flexion, supination, and ulnar deviation because it's the more oblique position.

In golf players, the majority of wrist problems involve the leading wrist with ECU subluxation or ECU tendinosis [3].

Surgeons should differentiate between acute and chronic lesions. The first group corresponds to retinaculum evolving the tendon, and the second ones affect the tendon itself.

The MRI characteristics of tenosynovitis of the ECU tendon include thickening of the tendon sheath, tenosynovial effusion, and peritendinous inflammatory changes (Fig. 31.4).

When there is instability of the ECU tendon, patients usually complain of a clicking with forearm rotation, and the subluxation of the tendon can be visibly palpated and observed. It's possible to reproduce it by forced supination and palmar flexion.

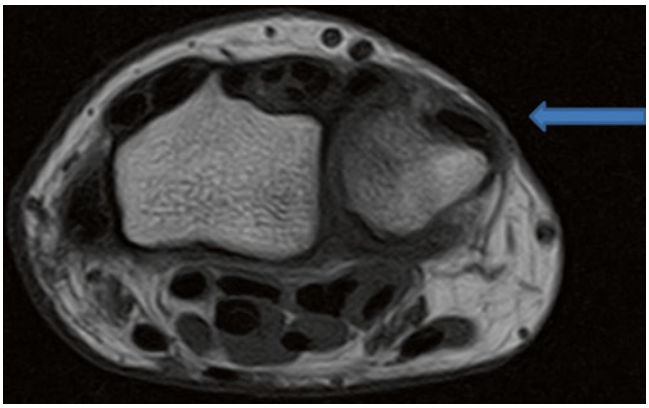


Fig. 31.4 This image shows intrasubstance longitudinal split of the ECU tendon (*arrow*) associated with peritendinous edema

Patterson et al. published [13] an acute traumatic ECU tendon subluxation treated conservatively in a case report. They placed the wrist in a short-arm cast in approximately 30° of extension, radial deviation, and pronation for 8 weeks with a good result.

In chronic cases, when immobilization is not effective, surgical procedures are needed. The majority of these procedures require the creation of a sling which is usually constructed from the extensor retinaculum.

31.3.5 Flexor Carpi Radialis (FCR) Tenosynovitis

Flexor tendon injuries are common in racquet sports and golf. With golf, the trailing hand normally undergoes a wider range or flexion and extension during the swing. In addition, unwanted fat shots and divots impart large forces onto the wrist. The spectrum of flexor carpi radialis tendon injuries ranges from tendinopathy (with tenderness over the insertion onto the second metacarpal base and pain with resisted wrist flexion) to rupture with inability of the patient to flex the wrist.

Clinically, there is pain and crepitus over the FCR tendon in the region just proximal to the flexor creases of the wrist. MRI is very useful in the diagnosis, and the findings include tendon sheath thickening. Inflammatory changes of the scaphotrapezial joint have also been described. Because of its proximity to the FCR, concomitant median nerve irritation may occur. MRI can be useful in approaching this entity, because the clinical diagnosis can be difficult, and it may be mistaken for scaphotrapezial joint disease, soft tissue ganglion, distal scaphoid fracture, distal radial fracture, and the Linburg's syndrome [10].

Treatment is always conservative.

31.3.6 Others Tendons

Other wrist tendons are also involved in overuse injuries. Flexor carpi ulnaris (FCU) tendinitis may be bilateral. Pain is felt with flexion and ulnar deviation and usually responds to conservative treatment. There is described a FCU tendinitis in pisotriquetral instability [14].

We can find anomalous tendon interconnections that are relatively uncommon.

Linburg's syndrome is a tendinous interconnection between the flexor pollicis longus and index profundus tendons, and this may produce pain in the forearm. The anomaly is present in 31 % of the population. If symptomatic, surgery restores the independent movement of the index finger and the thumb [1].

Another uncommon presentation of wrist pain is an extensor digitorum brevis manus, an anomaly muscle in the dorsum of the hand. It may be misdiagnosed as a ganglion and present as a painful lump following exercise. Kuschner et al. described a case in a weightlifter treated successfully by surgical excision [15].

References

1. Howse C (1994) Wrist injuries in sport. Sports Med 17(3): 163–175
2. Jacobson JA, Miller BS, Morag Y (2005) Golf and racquet sports injuries. Semin Musculoskelet Radiol 9(4):346–359
3. Hawkes R et al (2013) The prevalence, variety and impact of wrist problems in elite professional golfers on the European Tour. Br J Sports Med 47:1075–1079.
4. Kang HJ, Kho IH, Jang JW, Choi YR (2013) Endoscopic versus open release in patients with de Quervain's tenosynovitis. Bone Joint J 95-B:947–951
5. Guerini H, Drapé JL, Le Viet D, Thevenin F, Roulot E, Pessis E, Montalvan B, Feydy A, Chevrot A (2007) Imagerie du poignet du sportif. J Radiol 88:111–128
6. Heuck A, Bone H, Stäbler A, Schmitt R (1997) Imaging in sports medicine: hand and wrist. Eur J Radiol 26:2–15
7. Lemaire R, Hotermans JM (1983) Pathologie traumatique chronique en rapport avec la practique du tennis. Acta Orthop Belg 49:1–2
8. Osterman AL, Moskow L, Lowe DW (1988) Soft-tissues injuries of the hand and wrist in racquet sports. Clin Sports Med 7(2):329–348
9. Bencardino JT, Rosenberg ZS (2006) Sports-related injuries fo the wrist: an approach to MRI interpretation. Clin Sports Med 25:409–432
10. Aronowitz ER, Leddy JP (1998) Closed tendon injuries of the hand and wrist in athletes. Clin Sports Med 17(3):449–467
11. Yamabe E, Nakamura T, Pham P, Yoshioka H (2012) The athlete's wrist: ulnar-sided pain. Semin Musculoskelet Radiol 16:331–337
12. Buterbaugh GA, Brown TR, Horn PC (1998) Ulnar-sided wrist pain in athletes. Clin Sports Med 17(3):567–583
13. Patterson SM, Picconatto WJ, Alexander JA, Johnson RL (2011) Conservative treatment of an acute traumatic extensor carpi ulnaris tendon subluxation in a collegiate basketball player: a case report. J Athl Train 46(5):574–576
14. Carrol RE, Coyle MP Jr (1985) Dysfunction of the pisotriquetral joint. Treatment by excision of the pisiform. J Hand Surg Am 10:703–707
15. Kuschner SH, Gellman H, Bindinger A (1989) Extensot digitoris brevis manus – an unusual cause of exercise-induced wrist pain. Am J Sports Med 17:440–441

Distal Radius Fractures

32

Alberto Lazzerini

32.1 Introduction

Fractures of the distal epiphysis of the radius are rather common injuries, representing about 1.5 % of all emergency department cases [1]. These fractures are more common in children and in young male adults, due to sport injuries and traffic and work accidents, and in the elderly female population due to osteoporosis related to bone fragility [2, 3].

For many years, the treatment of these fractures has been mainly conservative, consisting in close reduction and cast immobilization. Conservative treatment may lead to poor results in unstable fractures where initial reduction may be lost due to muscle contracture and movement [4], or in fractures involving the radiocarpal or radioulnar joint surfaces where joint fragments are often not completely reducible by means of external manipulation.

In more recent years, due to a better knowledge of wrist joint function, more sophisticated diagnostic means, and less invasive and better tolerated implants, the surgical approach to distal radius fractures has been reconsidered and employed in an increasing number of cases [2]. The advantages of surgical treatment, consisting in better anatomical reconstruction and earlier mobilization, should be matched with higher costs of treatment and general surgical risk to identify the appropriate treatment approach for every single case.

32.2 Distal Radius Fractures and Sports

The wrist is exposed to trauma in several sports disciplines.

The most common mechanism of distal radius fracture is a fall with extended elbow and wrist as an attempt to protect the head [5, 6]. As the palm hits the ground, the carpus impacts the radial epiphysis transmitting axial, share, or combined forces that may induce different patterns of fractures. Such a situation can happen in most athletic activities, especially those where a fall may occur at high speed, like in biking or skiing or from a height, as in horse riding.

A less common injury to the distal radius is a direct stroke to the wrist or forearm as sometimes happens in contact sports. In such a situation, the ulna alone may be injured when the athlete wards off the hit with his forearm.

Occasionally, a strong rotation of the wrist or a hit holding a racket or a bat may generate forces capable of causing complex comminuted fractures to the distal radius.

In motor sports, the distal radius can suffer injury through a sudden violent deceleration with the hand grasping the steering wheel or gripping motorcycle handlebars.

Treatment of distal radius fractures in athletes is oriented toward a fast rehabilitation and return to the specific activity. Internal fixation is frequently preferred to conservative or less invasive surgical treatments allowing an earlier rehabilitation of wrist motion and reducing the risk of a secondary displacement that, although insignificant in normal daily activities, may limit top-level physical performances.

32.3 Pertinent Anatomy

The distal radius is a very complex bone segment.

It includes two joint surfaces, respectively, for the carpus at the radiocarpal joint and for the ulna at the distal radioulnar joint (DRUJ).

The radiocarpal joint surface is not perpendicular to the main axis of the radius, presenting a volar and ulnar tilt of approximately 15–20°. It is divided by a transverse ridge into two different joint facets, the radioscaphoid fossa facing the scaphoid and the lunate fossa facing the lunate in the proximal carpal row.

This arrangement of the joint surface has a fundamental role in the sophisticated biomechanics of the wrist and should always be preserved or restored when altered by trauma.

A. Lazzerini
Hand Surgery Unit, Humanitas Research Hospital,
Via Manzoni 56, Rozzano (MI) 20089, Italy
e-mail: alberto.lazzerini@humanitas.it

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_32,

The sigmoid notch of the radius is the articular surface facing the ulna in the distal radioulnar joint. The DRUJ is part of a more complex joint system which includes the proximal radioulnar joint and the interosseus membrane. It is frequently disregarded when involved in wrist trauma.

The triangular fibrocartilage (TFC), that with the radioulnar and ulnocarpal ligaments compose the triangular fibrocartilage complex (TFCC), arises from the ulnar margin of the articular surface of the radius and reaches the ulna at the fovea and the styloid in a complex arrangement. This ligament plays a fundamental role in radioulnar biomechanics and is frequently involved in distal radius fractures.

The dorsal aspect of the distal radius displays a series of bone ridges separating grooves, corresponding to the narrow retinacular canals. Their evenness is fundamental for the sliding motion of the extensor tendons as they reach the hand. The most prominent of these dorsal ridges is the Lister tubercle, around which the extensor pollicis longus tendon leaves the third retinacular canal and changes its direction heading toward the thumb. The tendon is therefore exposed to ruptures when a fracture involving the region heals with sharp or irregular edges of the tubercle.

The volar aspect of the distal radius is more regular; it is almost completely covered by the wide insertion of the pronator quadratus muscle, whose distal border corresponds to the watershed line, a transverse ridge lying just proximal to the joint edge, representing an important landmark in the placement of implants. A plate set distally to this line may interfere with the sliding motion of the flexor tendons.

Extrinsic wrist ligaments arise from the dorsal and volar edges of the distal radius and from the radial styloid, heading toward the carpus. The involvement and lesion of these ligaments in distal radius fractures are frequent and may generate chronic joint instability if not detected and treated.

32.4 Classification

Since the description made by Abraham Colles in the early nineteenth century [7], many physicians have defined distal radius fractures as "Colles fracture." As for all other fractures, a more accurate definition of the pattern and relations of fracture fragments is required to determine the most appropriate treatment.

There is no definitive classification of wrist fractures. Many factors must be considered:

- Fracture pattern
- Joint involvement
- Comminution
- Stability
- Soft tissue damage
- Associated lesions of ulna and carpal bones

Several classifications have been proposed in recent years. Among those most frequently considered, the following should be mentioned:

32.4.1 Frykman's Classification [8]

Proposed in 1967 is a very simple classification which considers the presence of articular involvement and ulnar fractures.

32.4.2 Mayo Clinic Classification [9]

Fractures are classified with regard to joint involvement, displacement, and reducibility.

32.4.3 Columnar Classification [10]

This classification is strongly oriented toward treatment choices; it divides the distal radioulnar complex into three columns with biomechanical significance.

32.4.4 Fernandez Classification [11]

This extensive classification is based on the injury mechanism determining the different fracture types and considers the associated lesions of soft tissues when suggesting the appropriate treatment.

32.4.5 AO Classification [12]

A widely adopted method that divides the fractures into three major groups and several subgroups depending on type of fracture, articular involvement, and metaphyseal comminution. This classification is rather complex but comprehensive of all possible fracture patterns and is easily accepted because this is comparable with the AO classification of all other district fractures.

Whichever classification is adopted, it is fundamental to refer to it for every case the surgeon has to deal with, with the intent to standardize treatment protocols and outcome evaluations.

32.5 Diagnosis

The diagnosis of distal radius fractures is based on clinical examination and x-ray assessment.

Deformity of the wrist is typical in many fractures with dorsal or palmar displacement; it may be less evident or absent

in fractures with minimal displacement of the carpus on the forearm, even in the presence of extensive comminution.

Swelling is an important sign present in all wrist fractures and is caused by early hematoma formation both in the joint and in the surrounding tissues. It is usually visible immediately after the injury since the wrist is a rather superficial joint, with no muscle cover.

Median nerve dysfunction occurs in a significant number of cases [13]. In many cases, it is present from the first observation due to compression in the carpal tunnel by displaced fracture fragments or hematoma and should always be considered when planning the best treatment option [14]. Pain is almost always present and is the main cause of early dysfunction and loss of strength and movement of the hand.

A high-energy trauma may cause skin tears due to the piercing by and subsequent exposure of sharp fractured bone tips, especially when the ulna is involved.

Standard x-rays include anteroposterior and lateral views. The lateral image should be obtained with the beam directed parallel to the articular surface, inclined approximately 15° distal to proximal (facet lateral radiograph) to avoid overlapping of the radius styloid. Additional x-rays must be performed when associated fractures of carpal bones are suspected.

CT scan is fundamental in the assessment of all articular fractures [15]. It gives important information on the number and displacement of fragments, comminution of articular surface and metaphyseal bone, and associated injuries of the carpus. It also suggests indirect evidence of ligament tears.

32.6 Treatment

32.6.1 Treatment Strategy

Several options are available for the treatment of distal radius fractures, and there is not just one method that can be applied to every fracture.

When determining the best treatment choice for a specific patient, many factors should be taken into account:
Fracture pattern (referring to the chosen classification)
Stability
Associated injuries
Nerve dysfunction
Patient specifications (age, general conditions, activity)

The AAOS guidelines for distal radius fractures [16] suggest operative fixation for fractures with postreduction radial shortening >3 mm, dorsal tilt >10°, or intra-articular displacement or step-off >2 mm as opposed to cast fixation. Many studies support these suggestions [4, 17].

Radial shortening and dorsal tilt, which play an important role in permanent disability after distal radial fractures, tend to recur after an appropriate reduction and cast immobilization in most cases.

Recent studies have focused on the importance of distal fragment ulnar residual translation that may determine radioulnar instability even without lesions of the radioulnar ligaments [18].

Tears of the radioulnar or intercarpal ligaments are often present in distal radius fractures [19, 20]. They frequently remain undetected because physicians tend to focus on fracture management, and a complete clinical examination is impossible for the presence of pain and deformity. An adequate treatment plan should always include the evaluation and early repair of these lesions.

Displaced fractures of the radial epiphysis may determine compression of the median nerve into the carpal tunnel [13]. An early reduction of the fracture can release the compression in most cases, while persisting nerve dysfunction may require surgical decompression.

Despite an incomplete reduction of the radial fracture, complaints regarding loss of function may be insignificant in patients with poor functional needs. In these cases, particularly when associated with contraindications to surgery, an incomplete reduction of the fracture and immobilization may represent an acceptable choice of treatment.

On the other hand, a surgical approach and stable internal fixation may be a better choice for relatively stable extra-articular or simple articular fractures in young active adults requiring the best possible functional long-term outcome and a faster recovery.

32.6.2 The Role of Conservative Treatment

Despite the great number of studies recommending a more gentle approach to displaced distal radius fractures [21, 22], it is not rare to see these injuries managed with aggressive reduction and immobilization in a long-arm cast with extremely flexed and ulnarized wrist position, in an attempt to stabilize a correct relation between the fracture fragments. The so-called Cotton-Loder position [23] is then protracted for several weeks, and the patient is told that the consequent pain and discomfort are unavoidable. This approach can generate severe complications and sequelae like median nerve compression, joint stiffness, regional sympathetic dystrophy, skin sores, and, in rare cases, Volkmann contracture [24]. Recovery requires a long rehabilitation program and is incomplete in many cases.

Adequate reduction of distal radius fractures treated with cast immobilization is lost in most cases at serial radiographic follow-up controls.

These considerations should not push physicians toward an excessive indication to surgery. A correct conservative approach to distal radius fractures is recommended in stable extra-articular or simple articular fractures, or in patients with low-functional needs or major contraindications to surgery.

An adequate conservative treatment should include the following steps:

Reduction of the fracture is obtained by gentle manipulation under local anesthesia, after a mild longitudinal traction prolonged for several minutes.

If the reduction is satisfactory and appears stable, a short-arm cast is applied. AAOS guidelines for distal radius fractures [16] proved unable to recommend immobilization of the elbow. Many authors [22, 25, 26] suggest the use of a double sugar tong splint for the first 3 weeks, allowing some flexion-extension of the elbow but impeding the rotation movement. The position of the wrist may vary from moderate flexion and ulnar deviation to extension, depending on the characteristics of the fracture, but extreme positioning should never be adopted.

Follow-up should include serial radiographs to ensure that the reduction is maintained.

Cast immobilization is prolonged for 5–6 weeks. After removal of the cast, an intensive rehabilitation program is started. Weightbearing is not permitted for a further 4–6 weeks.

32.6.3 The Role of Percutaneous Pin Fixation

Percutaneous pin fixation allows the stabilization of many unstable reducible fractures [27] (Fig. 32.1). Kirschner wires may be placed through the reduced fracture in different directions, based on the specific needs of fragment fixation. Pins piercing distal fragments may also be used to help reduce fractures difficult to manage by simple external manipulation.

Percutaneous pins alone cannot provide sufficient stability to prevent recurrent dislocations. Their use is always associated with the application of a splint or an external fixator, in the so-called augmented external fixation described later.

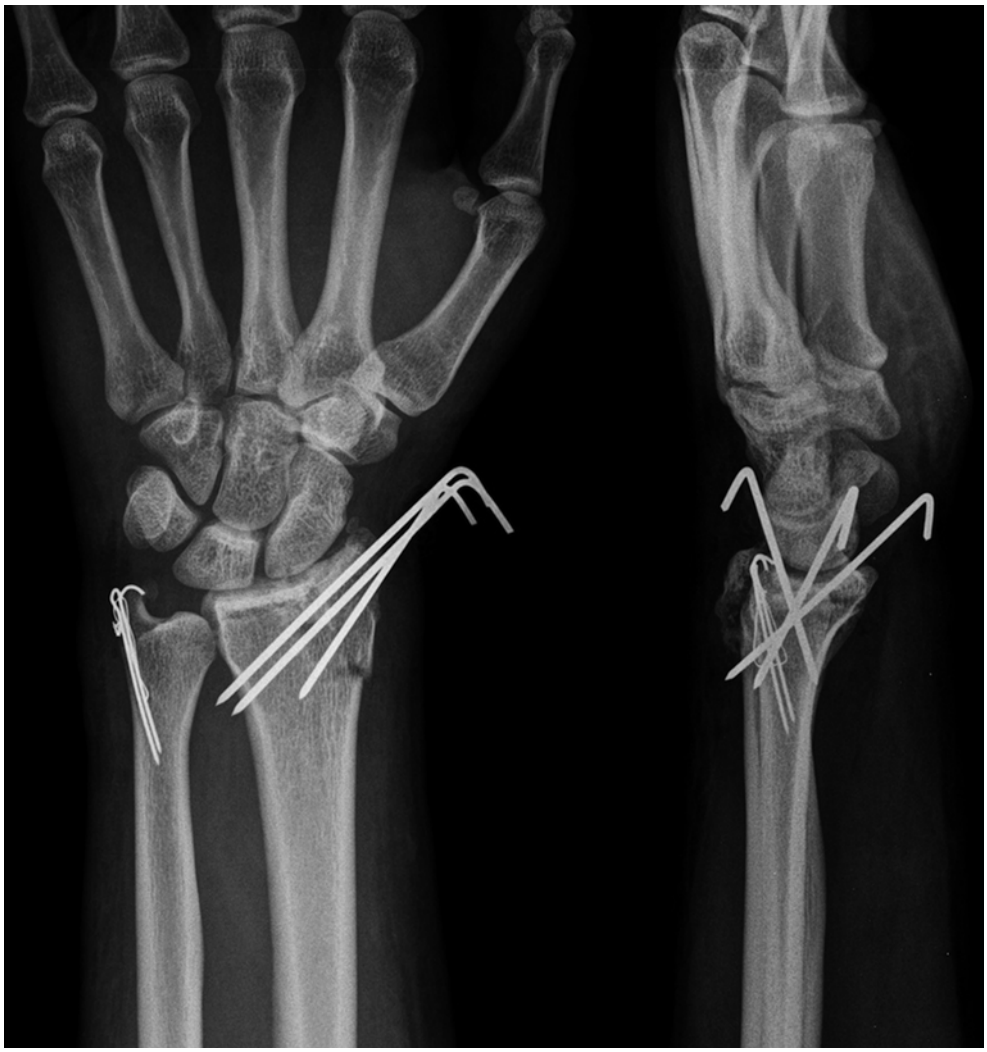


Fig. 32.1 A simple articular fracture of the radial and ulnar styloid, fixation with percutaneous Kirschner wires

Pin fixation is also the preferred surgical method for the treatment of fractures in children [28, 29] in cases where conservative treatment is unsuccessful.

32.6.4 The Role of External Fixation

External fixation has been considered an important means of treatment in the past years, as internal fixation devices were not as stable and versatile as they are today [30].

More recently, external fixation has retained a role in selected situations where extreme comminution, massive damage or loss of skin and soft tissue, critical survival conditions in trauma, and contamination of exposed bone discourage the use of internal fixation implants.

In many cases, external fixation is performed as a temporary stabilizer, and the treatment shifts to ORIF as adverse conditions are resolved.

In very comminuted fractures, where stability of internal fixation devices is not granted, external fixation may be associated in order to enhance stability in the early phases of bone healing [31].

The maintenance of reduction with external fixators is often based on distraction that may lead to adverse conditions such as ligament lesions, CRPS, and secondary hypercorrection [32].

The combination of external fixation devices with K-wires (augmented external fixation) enhances stability, prevents secondary displacement, and reduces the need for distraction and related complications [33] (Fig. 32.2).

32.6.5 The Role of Arthroscopy

Arthroscopy is considered an important means in the treatment of articular comminuted fractures of the wrist [34].

Arthroscopic assistance allows direct visualization of the ongoing reduction of articular fragments and a more accurate reconstruction of the joint surface, which greatly affect the outcome of medium- and long-term results.

Arthroscopy is also useful in the early evaluation and treatment of associated ligament lesions such as scapholunate or lunotriquetral ligaments or TFC [20]. These lesions may be underestimated during the assessment of the injury, as pain and deformity does not allow to adequately examine these articular structures, and stable or incomplete ligament tears often go undetected by standard x-rays and CT scan. Once detected, ligament tears associated to the fractures can be immediately repaired arthroscopically or by an open approach.

Wolfe [22] recommends postponing arthroscopic reduction for 3–7 days to avoid excessive bleeding, which may limit visibility, and risk development of a compartment syndrome due to extravasation of fluids into the soft tissues.

Arthroscopic reduction is followed by stabilization of the fracture by percutaneous pins or internal plate fixation.

32.6.6 The Role of Internal Fixation

For many years, internal fixation of distal radius fractures was affected by a high rate of complications due to the interference of implants with contiguous structures, in particular the extensor tendons, and to the rather poor stability of traditional fixation systems applied to osteoporotic or fragmented metaphyseal bone.

For these reasons, internal fixation used to be limited to those fractures considered untreatable by other means.

The development of a safe volar approach to the distal radial epiphysis [35] and the availability of fixed angle plates have dramatically changed surgical treatment of distal radius fractures. Metaphyseal comminution is a significant cause of instability. A great variety of devices has been developed for the treatment of distal radius fractures. Screws secured to the plate buttress the subchondral bone and prevent secondary displacement of the joint surface [36, 37]. The possibility to fix the screws to the plate at variable angles of last generation fixation devices has permitted a more reliable control of bone fragments in complex fractures and a better restoration of anatomy.

Low-profile and less invasive dorsal plates, designed for specific bone fragments, are used alone or in combination with volar plates when they are unable to maintain the obtained reduction of dorsal fragments alone [10].

32.6.6.1 Dorsal Approach

The dorsal epiphysis of the radius is approached by opening the third retinacular canal and elevating the retinaculum on both sides without exposing the extensors. Care must be taken in extensive comminution to preserve small bone fragments that tend to follow the elevated soft tissues.

Dorsal fixation of distal radius fractures has evolved during the last decades from plates applied to the whole dorsal surface of the distal epiphysis, to smaller low-profile plates designed for specific column fractures [10] (Fig. 32.3). Despite this evolution, interference with extensor tendons may still occur, and dorsal fixation is indicated only in those cases where a volar approach alone fails to restore anatomical conditions.

A dorsal approach can also be exploited to control "difficult" dorsal fragments that can be then fixed with a volar plate alone or in combination with dorsal screws or plates once reduced [38].

32.6.6.2 Volar Approach

The volar aspect of the distal radius is easily exposed with different approaches [35]. The pronator quadratus muscle is gently detached and elevated from the radial metaphysis. The

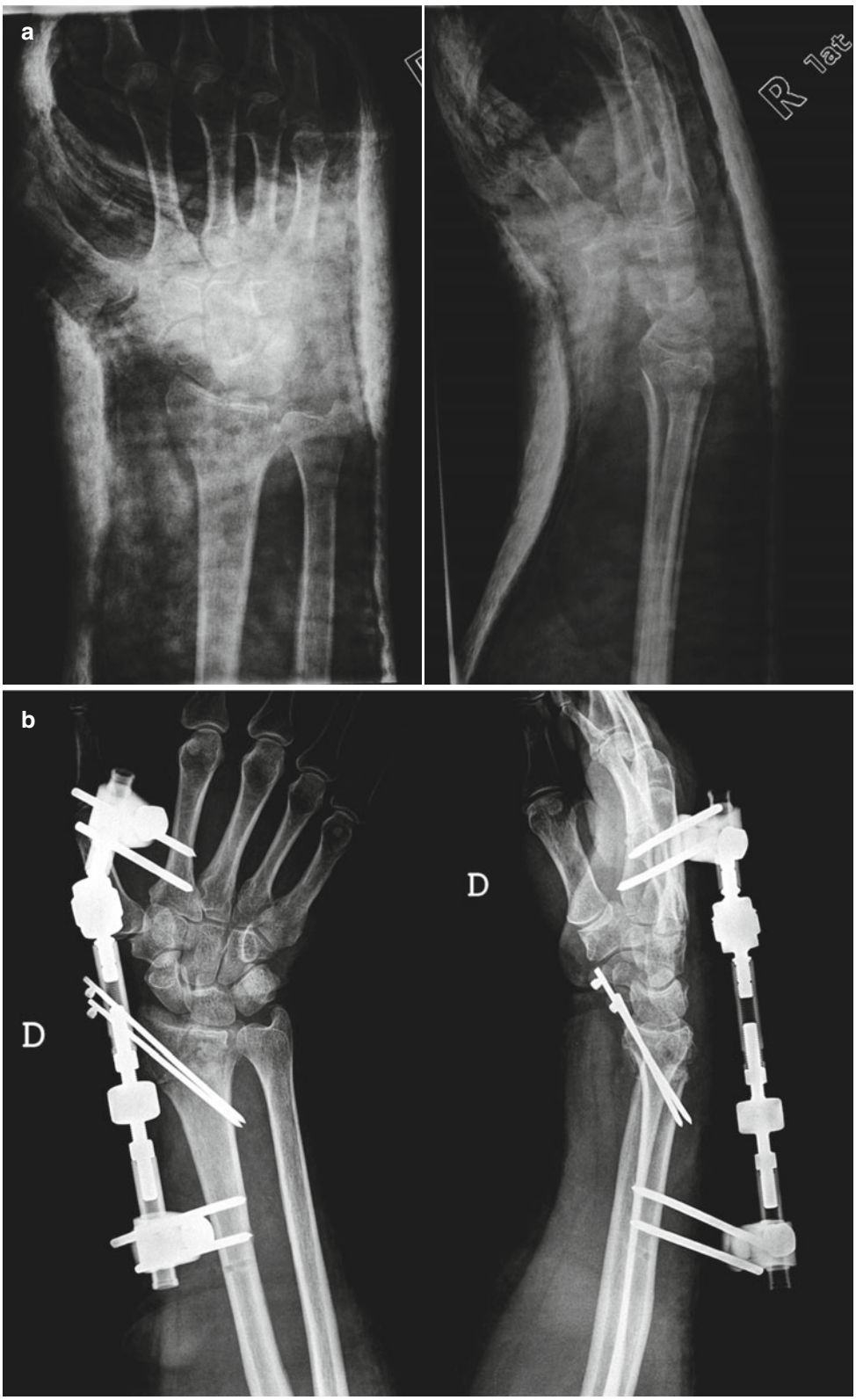


Fig. 32.2 Extra-articular fracture with radial translation of the distal epiphysis. (**a**) Cast immobilization fails in controlling the translation. (**b**) Reduction and stabilization with augmented external fixation

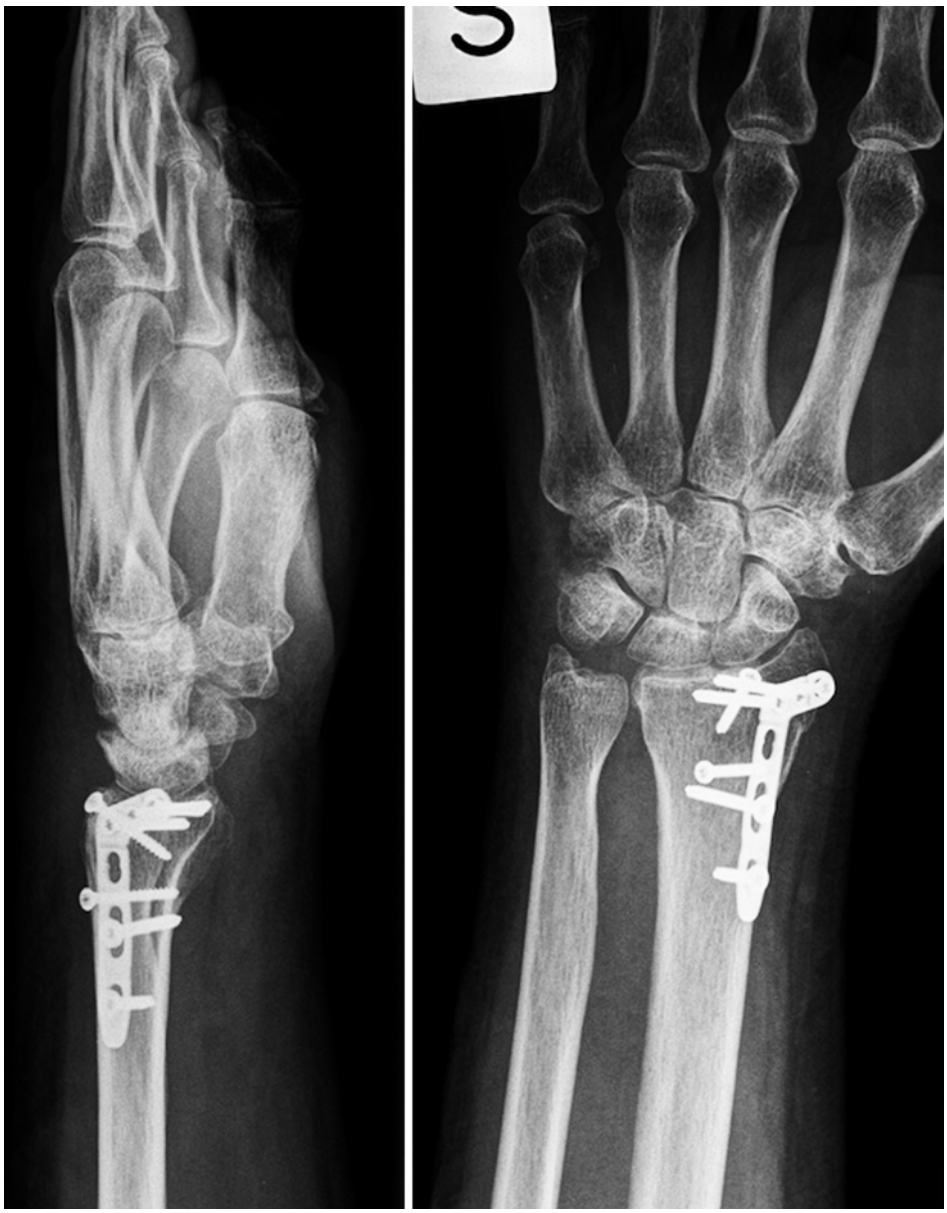


Fig. 32.3 Radial column fracture fixation with a dorsal specific fragment plate and screws

brachioradialis can be separated to facilitate the reduction of fractures involving the radial styloid. Articular fragments must be repositioned under direct view, arthroscopic, or fluoroscopic control. In cases with extensive metaphyseal comminution, bone grafts or bone substitutes can contribute to restore bone continuity and support subchondral bone and articular fragments. Once the fracture is reduced, a temporary stabilization with Kirschner wires can facilitate the positioning of a volar plate.

Most last generation plates have been designed to be placed proximally to the watershed line to prevent interference with flexor tendons. Locking and traditional screws can be chosen to achieve a better stabilization of every fracture, buttress subchondral bone, and pull dorsal fragments (Fig. 32.4). The repositioned pronator quadratus muscle covers the plate and protects the flexor tendons.

The postoperative regimen may vary depending on the stability of the implant.

In simple stable fractures, wrist mobilization can be started in the first days after the operation, with a protective splint to be removed for exercise.

In more comminuted fractures or osteoporotic bone, where stability is not granted, the joint is immobilized in a splint for 5 weeks. Elbow and finger movement is encouraged.

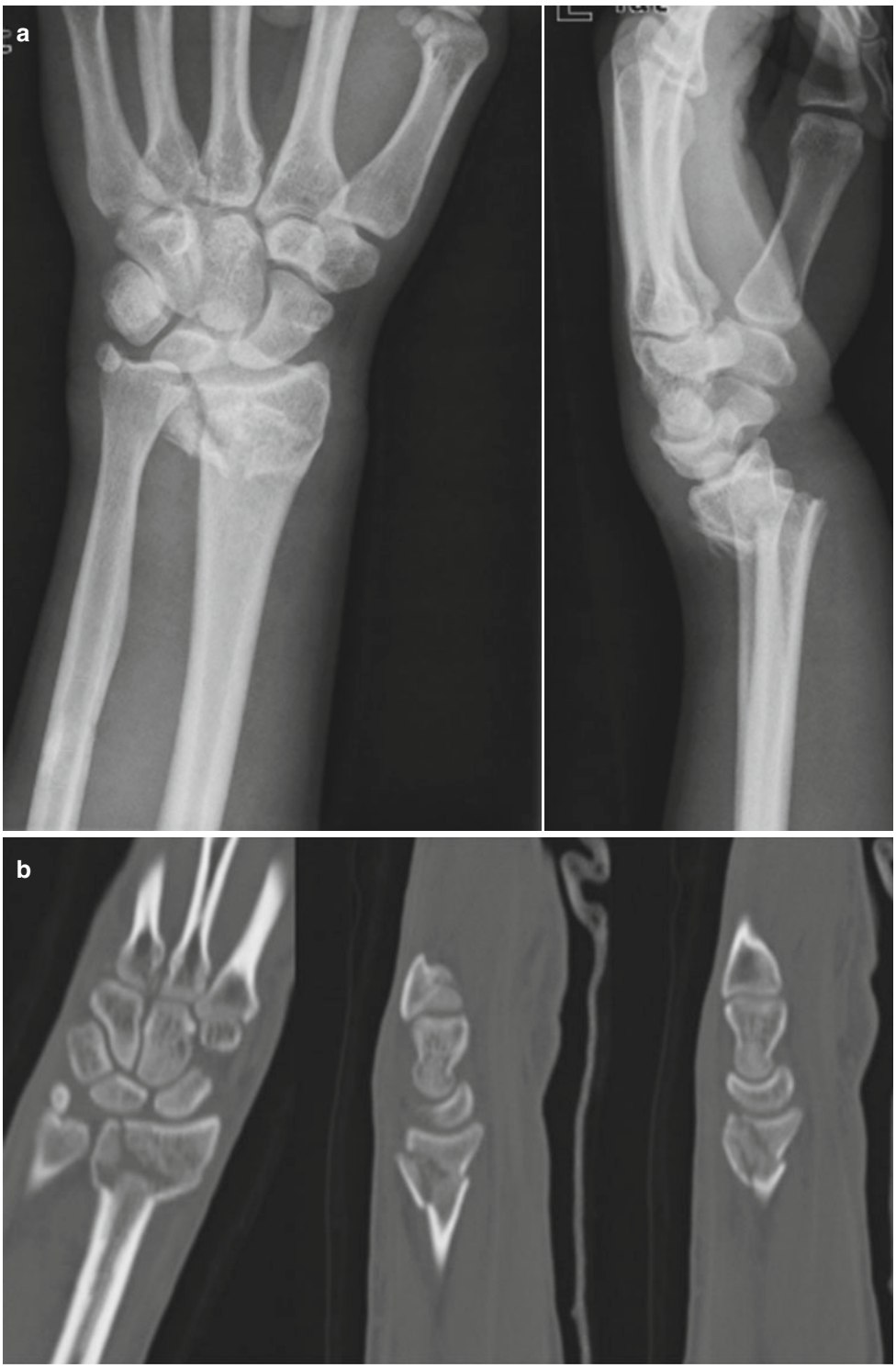
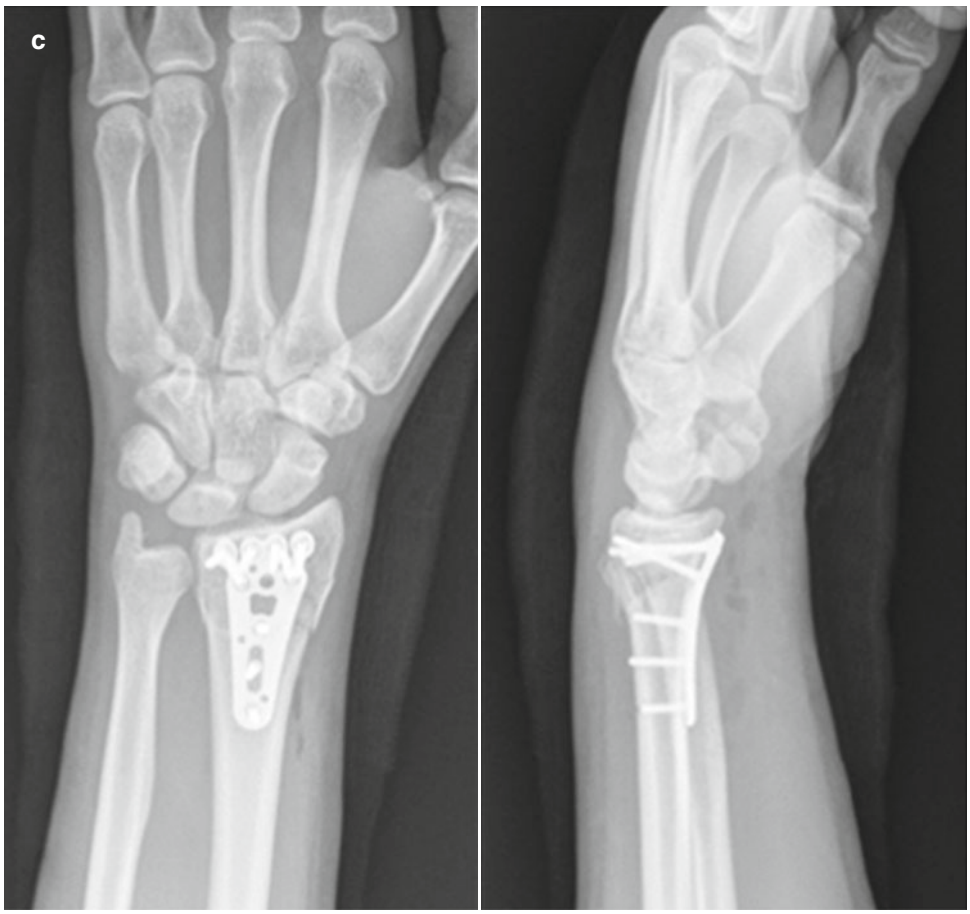


Fig. 32.4 Articular fracture of the distal radius. (**a**) Standard x-rays. (**b**) CT adds important information about the number and pattern of fragments. (**c**) Fixation by means of a volar locking plate positioned proximal to the watershed line

Fig. 32.4 (continued)



References

1. Chung KC, Spilson SV (2001) The frequency and epidemiology of hand and forearm fractures in the United States. J Hand Surg Am 26:908–915
2. Nellans KW, Kovalski E, Chung KC (2012) The epidemiology of distal radius fractures. Hand Clin 28(2):113–125
3. Koo KO, Tam DM, Chong AK (2013) Distal radius fractures: an epidemiological review. Orthop Surg 5(3):209–213
4. Gartland JJ, Werley CW (1951) Evaluation of healed Colles' fractures. J Bone Joint Surg Am 33:895–907
5. Bancroft LW (2013) Wrist injuries: a comparison between high- and low-impact sports. Radiol Clin North Am 51(2):299–311
6. Henn CM, Wolfe SW (2014) Distal radius fractures in athletes: approaches and treatment considerations. Sports Med Arthrosc 22(1):29–38
7. Colles A (1814) On the fracture of the carpal extremity of the radius. Edinb Med Surg J 10:182
8. Frykman GK (1967) Fracture of the distal radius including sequelae-shoulder-hand-finger syndrome, disturbance in the distal radioulnar joint and impairment of nerve function. A clinical and experimental study. Acta Orthop Scand 108:1–25
9. Cooney WP (1993) Fractures of the distal radius. A modern treatment based classification. Orthop Clin North Am 24(2): 211–216
10. Rikli DA, Regazzoni P (1996) Fractures of the distal end of the radius treated by internal fixation and early function. A preliminary report of 20 cases. J Bone Joint Surg Br 78:588–592
11. Fernandez DL (2001) Distal radius fractures: the rationale of classification. Chir Main 20(6):411–425
12. Mueller ME, Nazarian S, Koch P, Schatzker J (1990) The comprehensive classification of long bones. Springer, New York, pp 54–63
13. McKay SD, MacDermid JC, Roth JH, Richards RS (2001) Assessment of complications of distal radius fractures and development of a complication checklist. J Hand Surg Am 26A:916–922
14. Niver GE, Ilyas AM (2012) Carpal tunnel syndrome after distal radius fracture. Orthop Clin North Am 43(4):521–527
15. Harness NG, Ring D, Zurakowski D, Harris GJ, Jupiter JB (2006) The influence of three-dimensional computed tomography reconstructions on the characterization and treatment of distal radial fractures. J Bone Joint Surg Am 88:1315–1323
16. AAOS (2009) The treatment of distal radius fractures. Guideline and evidence report. A.A.O.S. v.1.0,12.05.09
17. Altissimi M, Antenucci R, Fiacca C, Mancini G (1986) Long term result of conservative treatment of fractures of the distal radius. Clin Orthop Relat Res 206:202–210
18. Ross M, Di Mascio L, Peters S, Cockfield A, Taylor F, Couzens G (2014) Defining residual radial translation of distal radius fractures: a potential cause of distal radioulnar joint instability. J Wrist Surg 3:22–29
19. Geissler WB, Freeland AE, Savoie FH, McIntyre LW, Whipple TL (1996) Intracarpal soft-tissue lesions associated with intra-articular fracture of the distal end of the radius. J Bone Joint Surg Am 78(3):356–365
20. Lindau T, Arner M, Hagberg L (1997) Intra articular lesions in distal fractures of the radius in young adults. A descriptive arthroscopic study in 50 patients. J Hand Surg Br 22B:638–643

21. Cooney WP (2010) Fractures of the distal radius, overview of diagnosis, classification and treatment considerations. In: Cooney WP (ed) The wrist, 2nd edn. Wolters Kluwer/Lippincott Williams & Wilkins, Philadelphia, pp 271–311
22. Wolfe SW (2011) Distal radius fractures. In: Wolfe SW, Hotchkiss RN, Pederson WC, Kozin SH (eds) Green's operative hand surgery, 6th edn. Elsevier Churchill Livingstone, Philadelphia, pp 561–638
23. Cotton FJ (1924) Dislocations and joint fractures. W.B. Saunders, Philadelphia, pp 348–358
24. Stern PJ, Derr RG (1993) Non-osseous complications following distal radius fractures. Iowa Orthop J 13:63–69
25. Grafstein E, Sternstrom R, Christenson J, Innes G, MacCormack R, Jackson C, Stothers K, Goetz T (2010) A prospective randomized controlled trial comparing circumferential casting and splinting in displaced Colles fractures. CJEM 12(3):192–200
26. Bong MR, Egol KA, Leibman M, Koval KJ (2006) A comparison of immediate postreduction splinting constructs for controlling initial displacement of fractures of the distal radius: a prospective randomized study of long-arm versus short-arm splinting. J Hand Surg Am 31(5):766–770
27. Handoll HH, Vaghela MV, Madhok R (2007) Percutaneous pinning for treating distal radial fractures in adults. Cochrane Database Syst Rev (3):CD006080
28. Abraham A, Handoll HH, Khan T (2008) Interventions for treating wrist fractures in children. Cochrane Database Syst Rev (16): CD004576
29. Mostafa MF, El-Adl G, Enan A (2009) Percutaneous Kirschner-wire fixation for displaced distal forearm fractures in children. Acta Orthop Belg 75(4):459–466
30. Cooney W (1983) External fixation of distal radius fractures. Clin Ortop 180:44–49
31. McAuliffe JA (2005) Combined internal and external fixation of distal radius fractures. Hand Clin 21(3):395–406
32. Kaempffe FA (1996) External fixation for distal radius fractures, adverse effects of excess distraction. Am J Orthop 25:205–209
33. Kaempffe FA, Walker KM (2000) External fixation for distal radius fractures: effect of distraction on outcome. Clin Orthop Relat Res 380:220–225
34. Doi K, Hattori Y, Otsuka K, Abe Y, Yamamoto H (1999) Intra-articular fractures of the distal aspect of the radius: arthroscopically assisted reduction compared with open reduction and internal fixation. J Bone Joint Surg Am 81(8):1093–1110
35. Ilyas AM (2011) Surgical approaches to the distal radius. Hand 6:8–17
36. Boswell S, McIff TE, Trease CA, Toby EB (2007) Mechanical characteristics of locking and compression plate constructs applied dorsally to distal radius fractures. J Hand Surg Am 32(5): 623–629
37. Orbay J (2005) Volar plate fixation of distal radius fractures. Hand Clin 21(3):347–354
38. Espen D (2003) Combined palmar and dorsal approach for complex distal radius fractures. Handchir Mikrochir Plast Chir 35:22–30

Hip Imaging Evaluation

33

Petr Zeman, Karel Koudela, and Jaroslav Zeman

Imaging methods play an important role in the evaluation of unexplained hip pain. The role of diagnostic imaging in the evaluation of the hip pain has been expanded recently by advances in computed tomography (CT) and magnetic resonance imaging (MRI) [1, 2]. Historically, MRI arthrography has shown success in detecting labral pathology, loose bodies, and also ligamentum teres abnormalities [3-5]. The addition of 3T MRI has increased our ability to visualize articular cartilage and other intra-articular and extra-articular abnormalities causing hip pain [6]. This chapter is intended for the arthroscopist and includes a brief review of various imaging modalities used in the evaluation of adult hip pain with emphasis on the diagnosis of intra-articular hip pathology.

33.1 Plain Radiography

Perhaps the most important tools in evaluating the hip are plain radiographs. Plain radiographs may demonstrate obvious causes of hip pain such as developmental dysplasia, stress fracture, avascular necrosis, degenerative joint disease, or tumor. Plain radiographs can also help us to clarify more subtle changes associated with mild cases of hip dysplasia or femoroacetabular impingement [7]. Just as is commonly seen in the other big joints, significant articular surface damage apparent at arthroscopy may be related to only subtle radiographic evidence of joint space narrowing on radiographs that could be superficially interpreted as normal.

Standard hip radiographic series include an anteroposterior (AP) view of the pelvis and frog-leg lateral views of the affected hip (Fig. 33.1). The frog-leg lateral radiograph provides accurate visualization of the femoral head-neck offset in patients being evaluated for femoroacetabular impingement [7]. This series may be completed with 45° and 90° Dunn views, false profile, and cross-table lateral views [7]. In traumatic cases, oblique or Judet views can help us to detect acetabular fractures.

Except the false profile view, all the remaining aforementioned projections are performed in lying supine position. In the AP view of the hip and pelvis, the patient's feet must be internally rotated 15° and the x-ray beam directed exactly in the AP plane [8]. The true AP pelvis view should have a neutral tilt and obliquity with the tip of coccyx 1.5–2 cm from symphysis. The AP pelvis film is centered low over the hips, and this is used rather than just an AP view of the affected hip for two reasons. First, it allows a radiographic examination of closely related areas, including the sacrum, sacroiliac joints, ischium, ilium, and pubis. Second, it allows us to compare subtle variations of the affected and contralateral hip.

The frog-leg lateral view is obtained with the hip abducted and the x-ray beam oriented in the AP direction [8]. The frog-leg lateral view provides a good lateral view of the femoral head and head-neck junction. The 45° and 90° Dunn views are taken with the hip flexed 45° or 90° and 20° abducted and the x-ray beam directed in the AP plane [7].

In recent years, much attention has been devoted to radiographic changes associated with FAI [7, 9]. The FAI syndrome could be divided into three types: pincer type, cam type, and mixed type (a combination of both). In the pincer type FAI, we could find excessive acetabular coverage, a crossover sign (COS) (Fig. 33.2), or posterior wall sign [9]. The crossover sign is associated with cranial acetabular retroversion but could also be often present on well-positioned AP pelvis radiographs in the absence of acetabular retroversion. Zaltz et al. reported that different morphologies and sizes of the anterior inferior iliac spine (AIIS) might contribute to the COS even in the absence of acetabular retroversion [10]. Findings supportive of the cam-type FAI include an aspherical femoral head with an abnormally increased alpha angle, focal prominence at the

P. Zeman, MD, PhD, MBA (✉) • K. Koudela, MD, PhD
J. Zeman, MD, PhD
Department of Orthopedics and Traumatology,
University Hospital, Pilsen, Alej Svobody, 80,
Pilsen 304 60, Czech Republic
e-mail: zempet@centrum.cz; k.koudela@seznam.cz;
zemanj@fnplzen.cz

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_33,

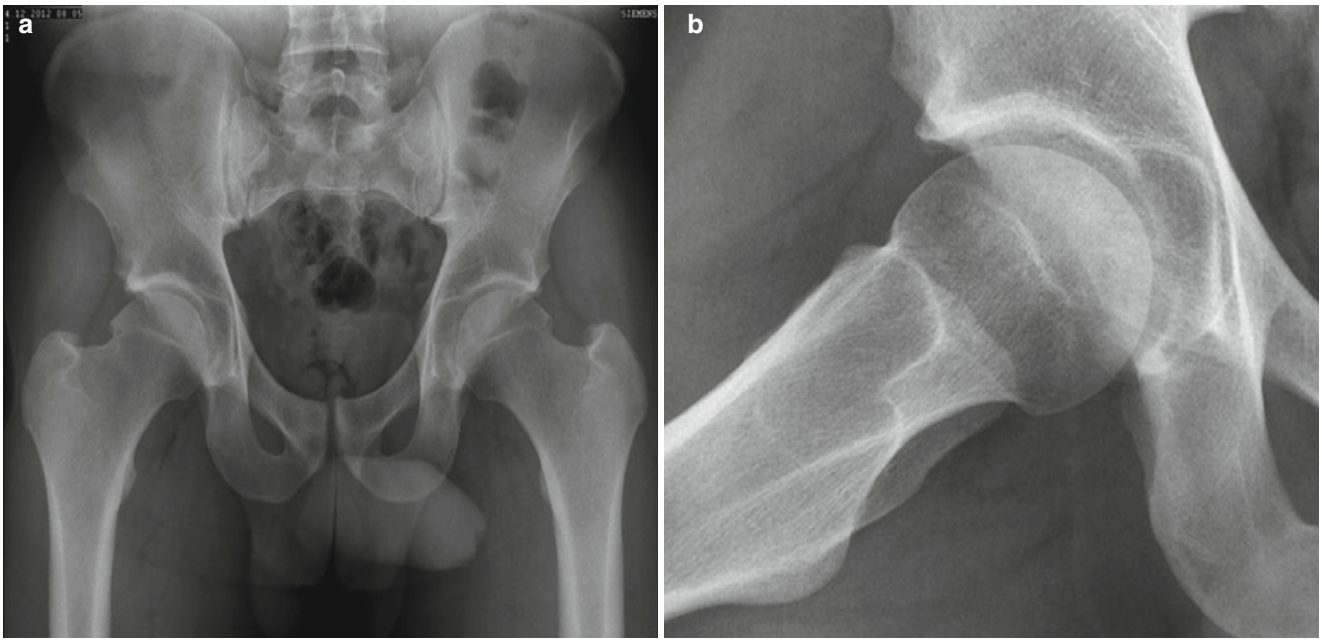


Fig. 33.1 Plain radiograph. (**a**) AP view of the pelvis with both hips with neutral tilt and obliquity. On the right hip, the FAI is present. (**b**) The frog-leg lateral radiograph provides accurate visualization of the femoral head-neck offset

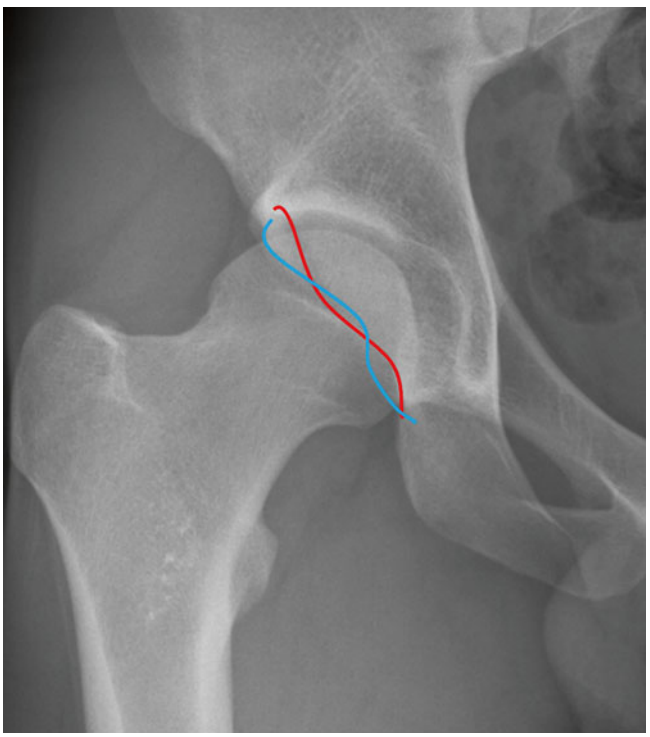


Fig. 33.2 The crossover sign in patient with Pincer type of FAI syndrome. Blue line indicates anterior wall and red line posterior wall of the acetabulum

anterior or lateral femoral head-neck junction (Fig. 33.3), and a typical pistol grip deformity of the proximal femur or cystic lesion [7, 9]. The alpha angle is defined as the angle subtended between the midline of the femoral neck and a line connecting the center of the femoral head to the point along the head-neck junction which first deviates from the sphericity of the femoral head (Fig. 33.3b). Notzli et al. reported significant increases in the alpha angle in FAI patients (72°) when compared with healthy controls (42°) [11].

Considerable variability in measurements could arise depending on pelvic position [7]. Plain radiographic findings in young patients with painful hips suggestive of any type of FAI should always be correlated with clinical evaluation.

33.2 Computed Tomography

CT is used to further characterize a bony detail of the hip not available on plain radiographs by providing cross-sectional information. CT of the hip should be done primarily in the setting of trauma or in suspicion to the neoplasm of the proximal femur or acetabulum. Occasionally, it is used to evaluate developmental hip dysplasia, to detect the small particle disease, and to evaluate for nonunion [12]. Multislice helical CT has markedly improved our ability to rapidly acquire high-resolution images with multiplanar 2D or 3D reconstruction with the benefits of a lower radiation dose than conventional CT, to improve resolution and to help us reduce the exam time. 3D CT reconstruction shows us a three-dimensional view of the acetabulum and femoral head and neck in FAI syndromes. The 3D CT helps us to clarify where exactly the cam or pincer lesion or other different abnormali-

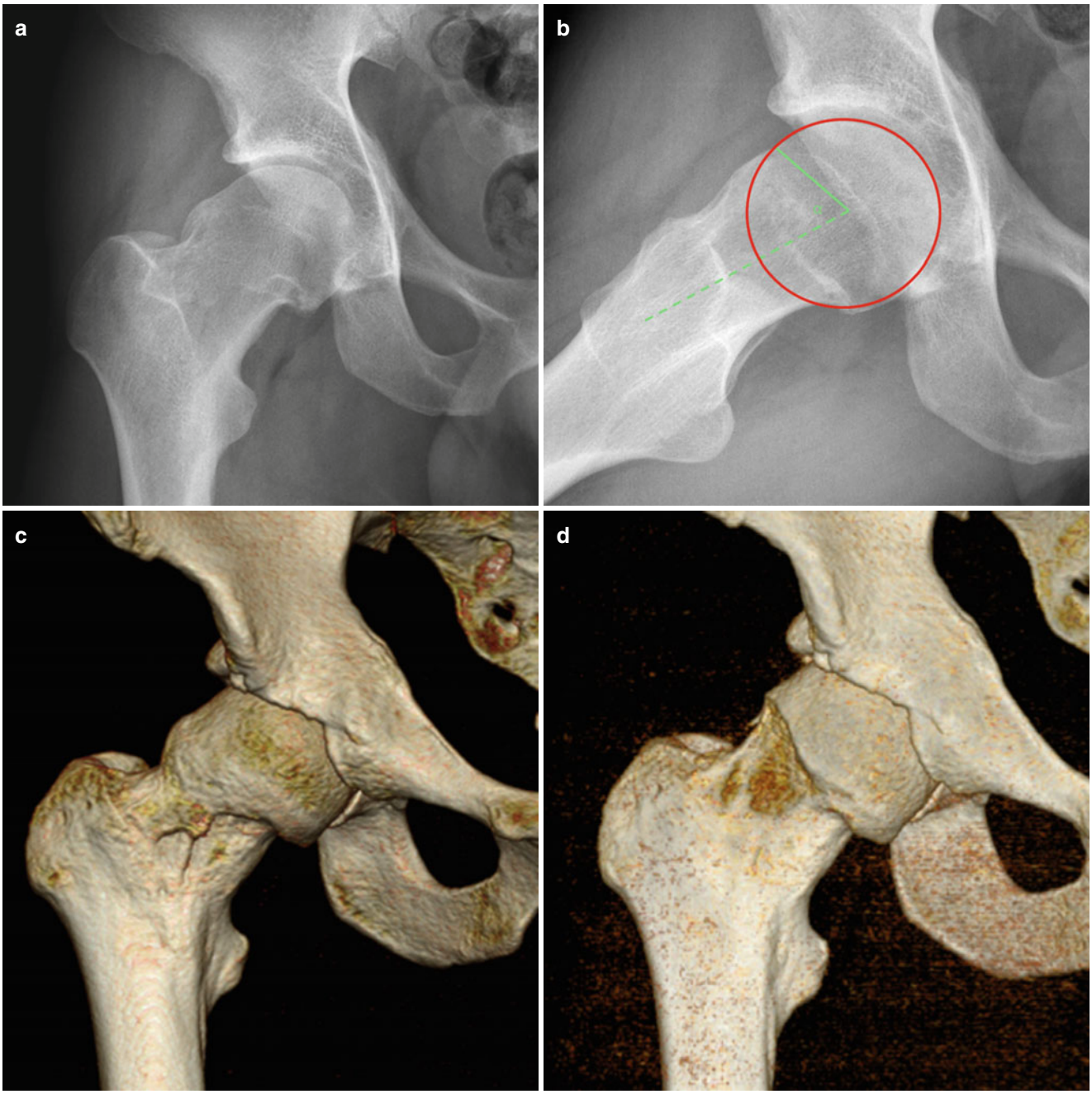


Fig. 33.3 A 28-year-old soccer player with painful right hip (**a**) AP view with pistol grip deformity on the lateral part of the head-neck junction typical for cam type of FAI syndrome. (**b**) The frog-leg view reveals anterior part of cam lesion. Measurement of the alpha angle is shown. (**c**) Anterior view of the right hip on 3D CT before and (**d**) after arthroscopic cam resection

ties are situated, and therefore, it is very helpful in preoperative planning (Figs. 33.3, 33.4, and 33.5). In our institution, 3D CT is performed in every patient before and also after arthroscopic management of the FAI syndrome to clarify whether the resection of the cam or pincer lesion has been sufficient.

In the setting of trauma, CT of the hip is used primarily to better characterize a fracture or fracture dislocation and to aid in the detection of articular surface fractures and intra-articular loose fragments [1]. CT can also yield information used in predicting hip instability in fractures of the posterior wall of the acetabulum.

CT offers a tool for preoperative measurement and planning for patients undergoing hip arthroplasty and can also be helpful in detecting and determining the extent of osteolysis around the hip prosthesis [13]. But in this case, a high artifact-free protocol is necessary.

CT can be used to help characterize the nature of the tumor matrix and demonstrate cortical thinning or destruction, in cases of neoplasm [14].

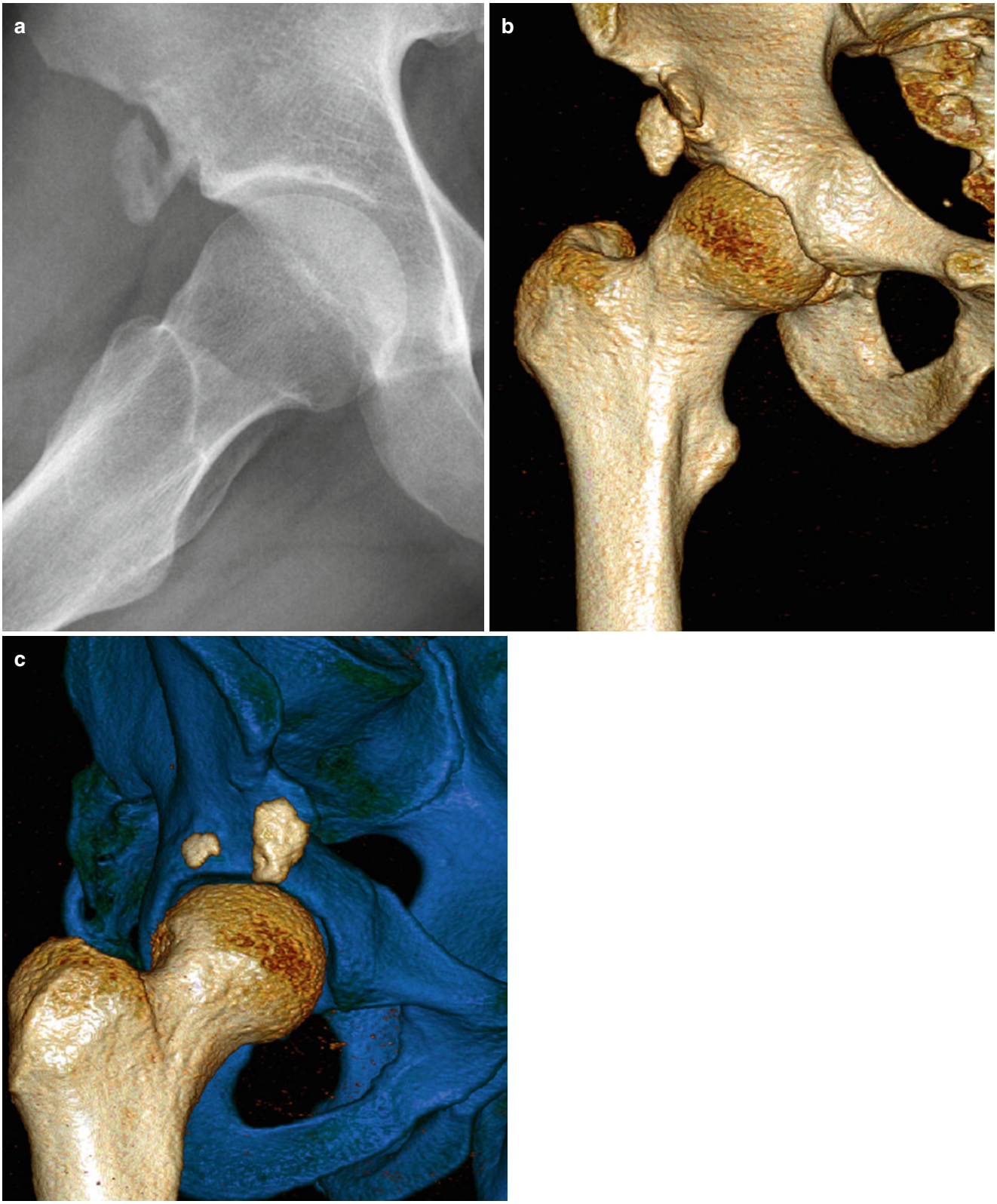


Fig. 33.4 A 24-year-old ice hockey player who suffered an avulsion fracture of the anterior inferior iliac spine 8 years ago. Subspine impingement syndrome is seen on the (**a**) frog-leg lateral view and (**b**) anterior and (**c**) anterolateral view of 3D CT

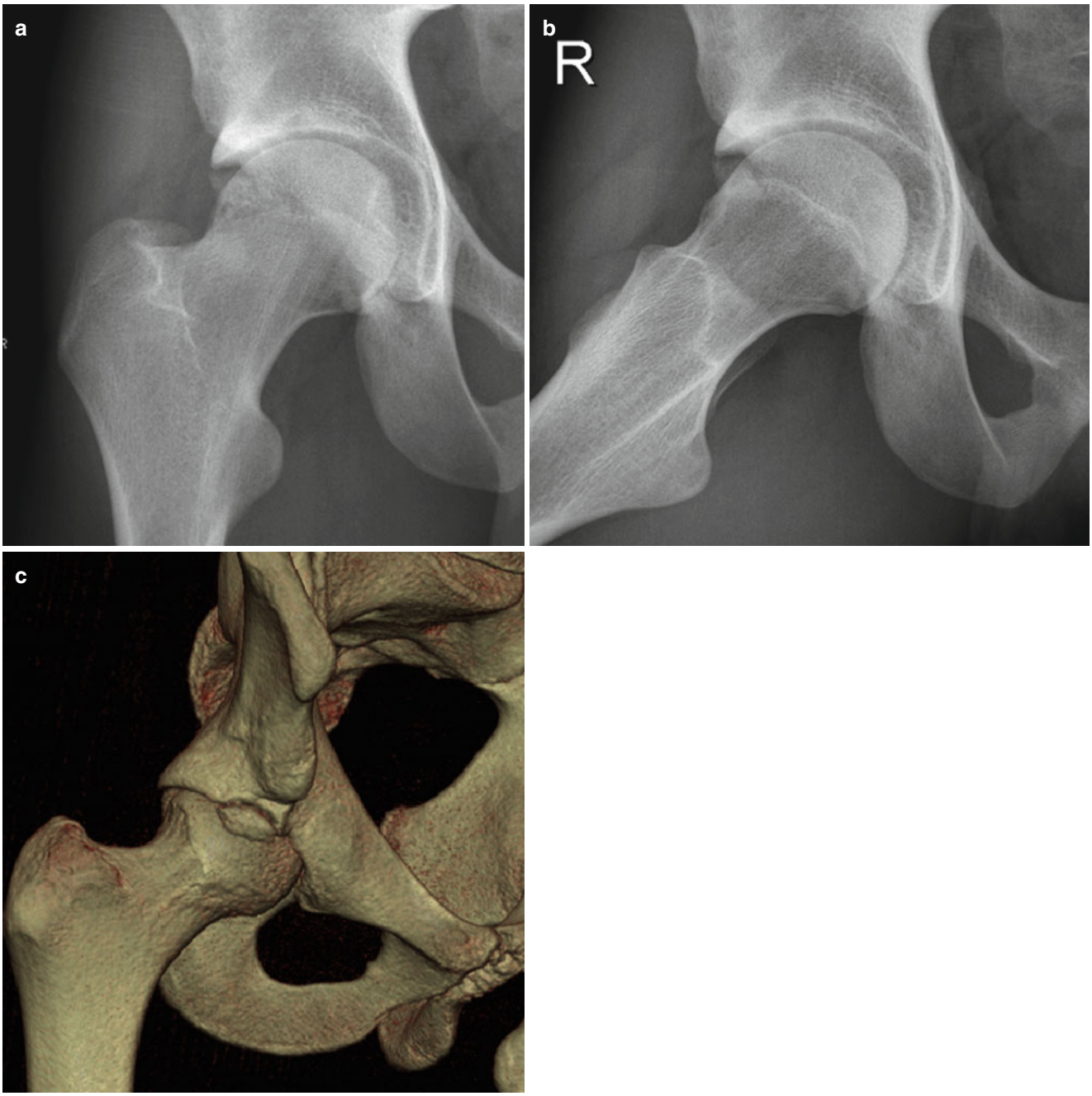


Fig. 33.5 A 34-year-old athlete with painful hip. (**a**) AP and (**b**) frog-leg lateral view with presence of os acetabuli and signs of mixed type of FAI syndrome. (**c**) An anterolateral view of 3D CT highlights os acetabuli and FAI syndrome better

33.3 CT Arthrography

CT arthrography of the hip is an excellent alternative to MR arthrography in patients with presence of loose metallic implants, presence of cardiostimulator, or with any other contraindications to MRI. CT arthrography can elucidate intra-articular abnormalities due to leakage of contrast into sites of labral, chondral, or ligamentous injury and can demonstrate loose bodies. Intra-articular injection before CT arthrography is preferably carried out using a combination of iodinated contrast with bupivacaine. In case of pain relief after local anesthetic application, it can help us to distinguish intra-articular from extra-articular sources of the hip pain [14].

33.4 Ultrasound

Ultrasound is a complementary diagnostics method to MRI in the evaluation of hip pain. Ultrasound is suitable especially for the evaluation of snapping hip syndrome and image-guided intervention [15, 16]. This method has many advantages over other imaging methods: It is noninvasive; it lacks ionizing radiation, allows concomitant evaluation of the contralateral hip, allows dynamic examination of the tendons and muscles, and can recognize pathologies of the tendons and soft tissue; and it is an inexpensive and quickly performed method and can be used as a guide for therapeutic injections and aspirations [17]. Ultrasound may provide a useful tool for early diagnosis of the cam-type FAI in daily practice [18].

Ultrasound is an excellent noninvasive tool for the diagnosis of different types of snapping hip syndrome in patients in case the snapping phenomenon may occur during a movement of the hip in the course of the examination [15].

Despite these advantages, ultrasound does not provide anatomic overview and demonstration of the bony structure, articular surfaces, bone marrow, and surrounding soft tissues in comparison with MRI [19].

Ultrasound is well suited for image-guided interventional procedures including injection of the joint, tendon sheaths, or bursa; drainage of para-articular fluid collections; and treatment of calcific tendinosis [17]. Continuous imaging during aspiration or injection helps us to verify a proper positioning of the needle tip. The problem is the impossibility to reevaluate the images of the hip later, and a very experience ultrasonographist must be present.

33.5 Conventional Magnetic Resonance Imaging

MRI is a secondary diagnostic method of choice in the evaluation of adult painful hip which provides excellent information about soft tissue and bone marrow abnormalities which are not seen on plain radiograph, CT, ultrasonography, or nuclear medicine exams. MRI is effective in demonstrating intra-articular and extra-articular pathology. Intra-articular disorders that are well demonstrated with MRI include osteonecrosis, stress fracture, occult fractures, joint effusions, osteoarthritis, and inflammatory arthropathies [20-23]. Different extra-articular pathologies such as pubic osteitis, sacroiliitis, bursitis, myotendinitis, and occult pelvis neoplasm may also be well recognized on MRI. In recent years, poor success has been demonstrated in the detection of labral tears and cartilage lesion on conventional MRI at 1,5 T, but this may improve with 3T MRI and with newer imaging techniques [3, 24]. Future improvements in MRI technology may lead to successful noninvasive evaluation of these structures. Studies in recent years have shown that 3T MR arthrography is much more beneficial in the detection of labral tears in comparison with conventional 3T MRI [6, 25]. Although MR arthrography is an excellent positive predictor in diagnosing acetabular labral tears and cartilage abnormalities, it still has limited sensitivity. A negative imaging study does not exclude the presence of intra-articular pathology that can be identified and treated arthroscopically [26].

Protocols for MRI of the hip vary among institutions and with the type of scanner used. The quality of the examination depends on the field strength of scanner, coil selection, technical parameters used, and whether or not dedicated small-field-of-view images of the hip are acquired. MRI protocols are adapted to the clinical presentation. For instance, at our institution, the typical exam for an adult with unexplained hip pain includes seven sequences and is tailored toward evaluating intra-articular and extra-articular pathology. The coronal T1-weighted images of both hips reveal anatomy and marrow-based pathologies such as occult fractures, tumors, or osteonecrosis. The T2-weighted fat-suppressed images of both hips demonstrate myotendinous injuries, intra-articular or extra-articular fluid collection such as bursitis and joint effusions, depict stress fracture and occult fracture, subchondral cyst, osteonecrosis, and tumor as marrow-based changes. For the detection of cartilaginous and labral lesions, it is essential to create small-field-of-view high-resolution imaging of the affected hip [12]. The oblique sagittal SPGR sequences are used to measure for the cam and pincer FAI and to assess for the characteristic osseous bump present with the cam FAI.

33.6 Magnetic Resonance Arthrography

Magnetic resonance arthrography (MRA) of the hip is a diagnostic method of choice of unexplained adult hip pain in the event that we suspect intra-articular pathologies. Several reports have documented the success of MRA of the hip in detecting cartilage and labral injuries [3, 5, 14, 27, 28]. MRA can also depict abnormalities of ligamentum teres and joint capsule and demonstrate the presence of loose bodies or proliferative changes of synovitis. In comparison with conventional MR, MR arthrography allows better visualization of intra-articular anatomy because the intra-articular application of the fluid displaces the joint capsule from the underlying bone and normal structures. Leakage of contrast into the labrum or cartilage is direct evidence of pathology of these structures [24]. MRA of the hip is thus helpful when conventional MR is noncontributory and when there is clinical suspicion of labral or other intra-articular pathologies.

MRA of the hip involves intra-articular injection of a solution of dilute gadolinium (1–2 mmol) under fluoroscopic guidance followed by multiplanar MRI of the hip (Fig. 33.6) At our institution, gadolinium is diluted approximately 1:200 with local anesthetic (1 % lidocaine HCl or 0.25 % bupivacaine HCl). Immediately after the injection, the patient is transferred to the MRI, and MR imaging should be performed within 45 min of the injection.

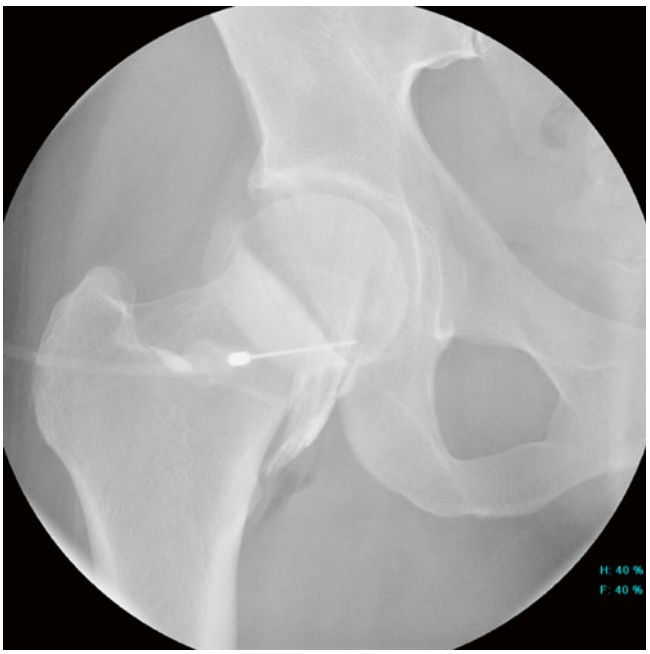


Fig. 33.6 Fluoroscopic guidance of intra-articular injection of a solution with gadolinium diluted in anesthetic before MR arthrography

Concomitant injection of the local anesthetic as a diluent adds the advantage of providing clinical information helpful in distinguishing intra-articular from extra-articular pathology. This technique of MRA provides us both anatomic and clinical information and is the preferred technique of the author. Symptomatic pain relief after MRA with anesthetic injection provides strong clinical evidence of intra-articular pathology, and MR images obtained can serve as a guide for the surgeon [29].

MRA of the hip at our institution is performed on a 3T MR scanner, focusing on the affected hip. For the detection of labral pathology, primarily coronal, sagittal, and axial T1 fat-suppressed images are used (Figs. 33.7, 33.8, and 33.9). T2 fat-suppressed images help us to identify extra-articular soft tissue pathologies such as bursitis or gluteus tendon tears and marrow-based findings (marrow edema, subchondral or labral cyst, etc.). Coronal proton density (PD) images are able to demonstrate cartilage and labral pathology. For measurement of acetabular overcoverage and the alpha angle associated with FAI oblique sagittal FSPGR images should be performed.

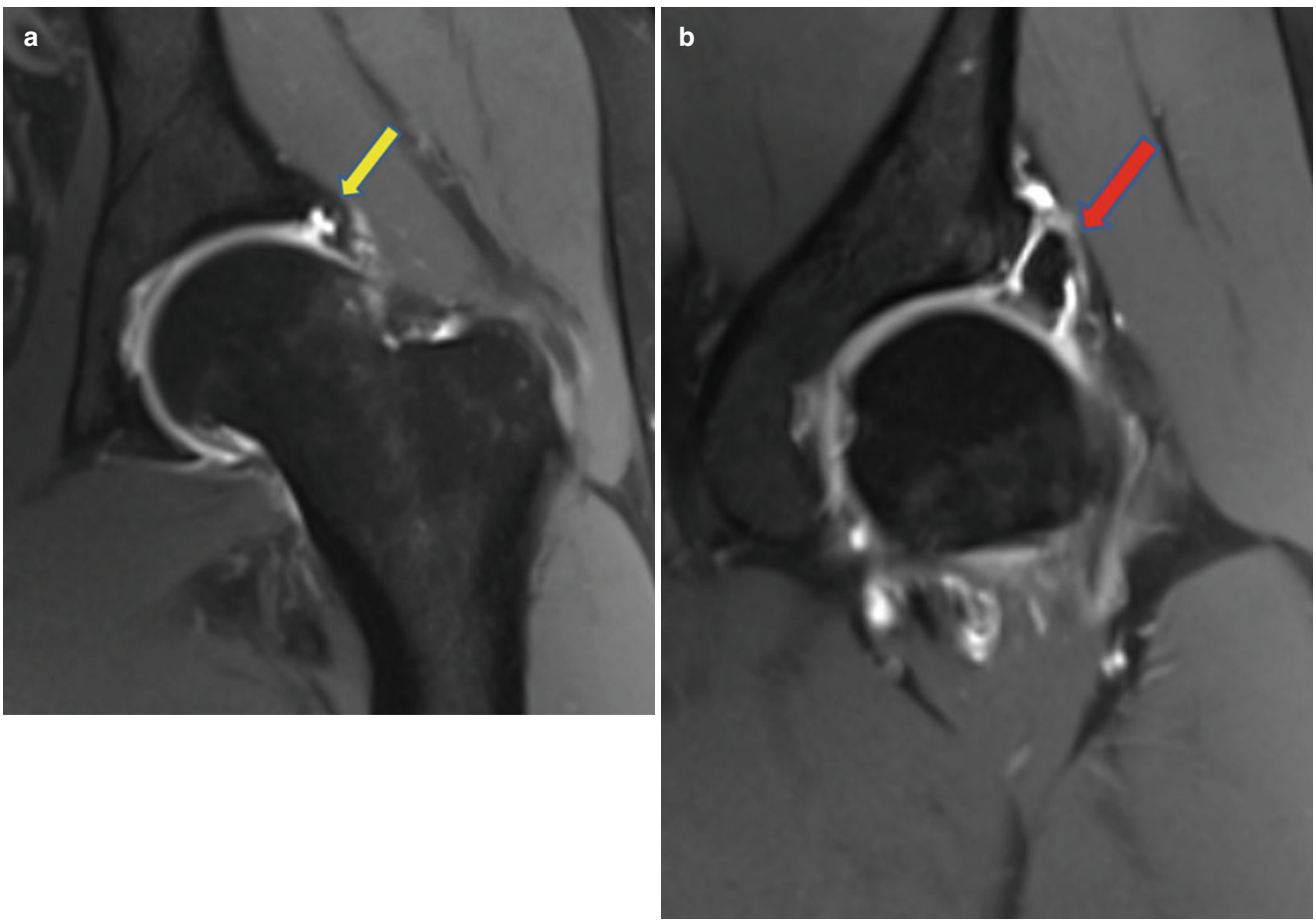


Fig. 33.7 MR arthrography: Coronal T1-weighted fat-suppressed images of the left hip demonstrate (**a**) chondrolabral lesion and cyst (*yellow arrow*) and (**b**) os acetabuli (*red arrow*) in a 24-year-old hand ball player with painful hip

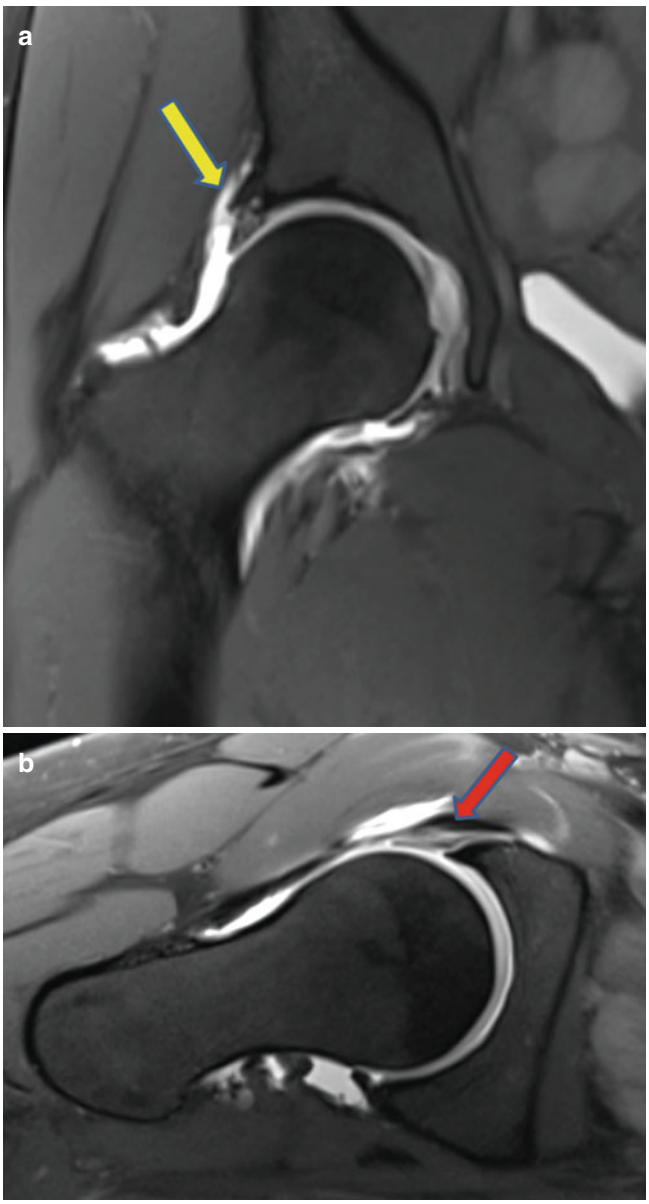


Fig. 33.8 MR arthrography of 36-year-old woman with painful internal snapping hip syndrome and psoas impingement. (**a**) Coronal T1-weighted fat-suppressed image and (**b**) sagittal T1-weighted fat-suppressed 3T image reveal anterior labrum degeneration (*yellow and red arrows*)

33.7 Nuclear Scintigraphy

Unlike the increasingly used MRI in recent years, nuclear scintigraphy is rarely used in the workup of unexplained pain in the native adult hip. The bone scan is the most common scintigraphic examination used in the evaluation of different skeletal pathologies. Bone scanning with a radionuclide shows us bone turnover and may elucidate changes in local blood flow and can help distinguish monostotic from polyostotic disease [30].

This method is increasingly used to assess for infection or loosening in patients with painful hips following arthroplasty. Increased activity can normally be seen around the hip arthroplasty components for up to 2 years after surgery. Persistent activity near the trochanters or around the tip of the femoral component must arouse in us a suspicion of loosening [30]. Increased intensity around the whole femoral component can indicate infection [30].

Some authors recommend the use of bone scan to diagnose the FAI syndrome of adult hip pain as a secondary examination [31, 32].

33.8 Hip Arthrography, Injection, Aspiration, and Bursography

Historically, conventional arthrography of the hip in adults was used in cases of suspected intra-articular loose bodies, hip infection, and synovial disorders and also to evaluate loosening or infection of a hip arthroplasty [33]. With the advent and development of MRI, most arthrographies of the hip are performed as an MR arthrography. Image guidance for arthrography of the hip could be with fluoroscopy or with sonographic guidance. At our institution, we typically use the anterior approach, when the needle is inserted into the peripheral compartment of the hip under fluoroscopic control. The position of the entry point of the needle on the skin must always be lateral to the neurovascular femoral structures in case of the native hip. The overlying skin is prepped with Betadine solution, and the skin and subcutaneous tissue are anesthetized with 1 % lidocaine HCl. In suspicion of infection (septic arthritis or infected hip prosthesis), first of all, diagnostic aspiration must be done. A small amount (1–2 ml) of iodinated contrast is injected to verify an intra-articular position of the needle.

Intra-articular diagnostic or therapeutic injection of corticosteroid or long-acting anesthetic is typically used to distinguish intra-articular from extra-articular source of pain and significantly help us to indicate the correct therapy. At our institution, we use 80 mg of methylprednisolone acetate (Depo-Medrol) and 0.25 % bupivacaine HCl. Aspiration is performed first to exclude evidence of infection and to remove joint fluid to allow space for the anesthetic and steroid. If the turbulent fluid is present, laboratory analysis of the fluid is to be performed, and we should avoid steroid application. The patient's pain level is monitored before and after the application of injection, and it is documented.

Diagnostic aspirations are preformed in both native hips and in patients with hip prosthesis when the suspicion of the infection is present. For the native hip, we use the same approach which was mentioned above. In cases of hip arthroplasty, the entry site is just targeted to the superior margin of the femoral neck which allows us better visualization of the needle and gives us a higher chance to harvest the joint.

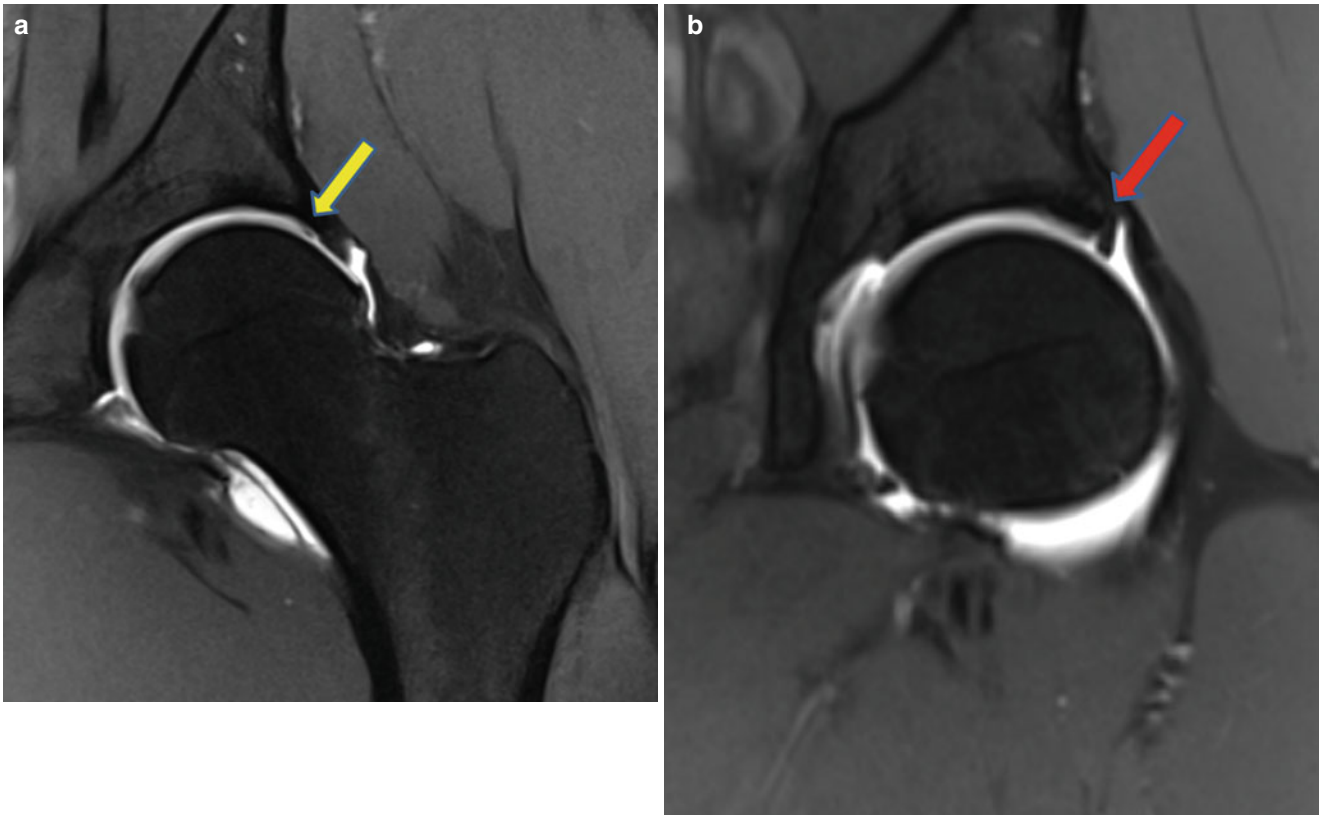


Fig. 33.9 MR arthrography with subtle abnormalities on the labrum and cartilage in a patient with FAI syndrome. (**a**) Coronal T1-weighted fat-suppressed 3T image with suspected chondral lesion of the superolateral acetabulum (*yellow arrow*). (**b**) Coronal T1-weighted fat-suppressed 3T image demonstrates lesion of the lateral labrum (*red arrow*)

Iliopsoas bursography under fluoroscopic guidance has been used for many years for determining the diagnosis of iliopsoas snapping hip syndrome [34]. Due to exposing the patient to ionizing radiation and because of the other disadvantages of the method, ultrasonography because of its advantages has completely replaced this technique. Ultrasonography is noninvasive and cheaper, and it allows us a dynamic examination of the hip in both the internal and external types of snapping hip syndrome [35]. Sonographic guidance also allows direct visualization of the needle tip and its position relative to the psoas tendon and bursa wall. In our institution, we prefer injection of iliopsoas bursa for pain relief in cases of painful bursitis under sonographic control. We inject methylprednisolone acetate diluted in 20 % bupivacaine into the bursa. The same approach on the skin for injection is used as was mentioned in arthrography.

33.9 Summary

Imaging methods together with the history and a precise clinical examination represent an essential element for a definitive diagnosis. In unexplained adult hip pain, correctly performed plain radiographs should always be the initial imaging exam. Plain radiographs of patients in whom we suspect FAI syndrome could often demonstrate subtle findings or may even be normal, and therefore, we should always compare these findings with clinical examination and MRI or MR arthrography. MRI is a secondary diagnostic method of choice in the evaluation of unexplained adult hip pain. In cases where we suspect lesion of labrum or cartilage, MR arthrography is a more sensitive and specific diagnostic tool compared to conventional MRI. Also on suspicion of ligamentum teres injuries and the presence of the other intra-articular pathologies, MR arthrography is an excellent diagnostic method. With the introduction of 3T MRI, a particular diagnosis of cartilage abnormalities associated with FAI is becoming increasingly more accurate. Ultrasonography plays an important role in therapeutic intervention, and dynamic ultrasonography is an excellent diagnostic tool in both types of snapping hip syndrome. The abovementioned diagnostic methods help the arthroscopist to choose the right treatment strategy and aid in surgical planning.

References

1. Conway WF, Totty WG, McEnery KW (1996) CT and MR imaging of the hip. Radiology 198:297–307

2. Gabriel H, Fitzgerald SW, Myers MT et al (1994) MR imaging of the hip disorders. Radiographics 14:763–781
3. Czerny C, Hofmann S, Urban M et al (1999) MR arthrography of the adult acetabular capsule-labral complex: correlation with surgery and anatomy. AJR Am J Roentgenol 173:345–349
4. Erb RE (2001) Current concepts in imaging the adult hip. Clin Sports Med 20:661–696
5. Petersilge CA (2001) MR arthrography for evaluation of the acetabular labrum. Skeletal Radiol 30:423–430
6. Magee T (2010) Comparison of 3 Tesla MR versus 3 Tesla MR arthrography of the hip for detection of acetabular labral tears in the same patient population. AJR Am J Roentgenol 194:A91–A95
7. Clohisy JC, Carlisle JC, Beaule PC et al (2008) A systematic approach to the plain radiographic evaluation of the young adult hip. J Bone Joint Surg Am 90:47–66
8. Sartoris DJ, Resnick D (1998) Plain film radiography: routine and specialized techniques and projections. In: Resnick D, Niwayana G (eds) Diagnosis of bone and joint disorders, vol 1, 2nd edn. Saunders, Philadelphia, p 38
9. Laborie LB, Lehmann TG, Engesaeter IO et al (2011) Prevalence of radiographic findings thought to be associated with femoroacetabular impingement in a population-based cohort of 2081 healthy young adults. Radiology 260:495–502
10. Zaltz I, Kelly BT, Hetsroni I, Bedi A (2013) The crossover sign overestimates acetabular retroversion. Clin Orthop Relat Res 471(8):2463–2470
11. Notzli HP, Wyss TF, Stoecklin CH, Schmid MR, Treiber K, Hodler J (2002) The contour of the femoral head-neck junction as a predictor for the risk of anterior impingement. J Bone Joint Surg Br 84(4):556–560
12. Erb RE (2005) Adult hip imaging. In: Byrd JWT (ed) Operative hip arthroscopy, 2nd edn. Springer, New York, p 51
13. Park JS, Ryu KN, Hong HP et al (2004) Focal osteolysis in total hip replacement: CT findings. Skeletal Radiol 33:632–640
14. Erb RE (2013) Adult hip imaging for the arthroscopist. In: Byrd JWT (ed) Operative hip arthroscopy, 3rd edn. Springer, New York, p 35
15. Lee KS, Rosas HG, Phancao JP (2013) Snapping hip: imaging and treatment. Semin Musculoskelet Radiol 17(3):286–294
16. Byrd JW, Potts EA, Allison RK, Jones KS (2014) Ultrasound-guided hip injections: a comparative study with fluoroscopy-guided injections. Arthroscopy 30(1):42–46
17. Joines MM, Motamedi K, Seeder LL et al (2007) Musculoskeletal interventional ultrasound. Semin Musculokelet Radiol 11:192–198
18. Lerch S, Kasperczyk A, Wernecke J, Berndt T, Ruhmann O (2013) Evaluation of Cam-type femoroacetabular impingement by ultrasound. Int Orthop 37(5):783–788
19. Friedman T, Miller TT (2013) MR imaging and ultrasound correlation of hip pathologic conditions. Magn Reson Imaging Clin N Am 21(1):183–194
20. Moss SG, Schweitzer ME, Jacobson JA et al (1998) Hip joint fluid: detection and distribution at MR imaging an US with cadaveric correlation. Radiology 208:43–48
21. Kunešová M, Koudela K Jr, Koudela K Sr, Koudelová J (2006) Magnetic resonance imaging for examination of proximal femoral fractures: its contribution to clinical medicine. Acta Chir Orthop Traumatol Cech 73(6):380–386
22. Pandey R, McNally E, Ali A et al (1998) The role of MRI in the diagnosis of occult hip fractures. Injury 29:61–63
23. Beltran J, Caudill JL, Herman LA et al (1987) Rheumatoid arthritis: MR imaging manifestations. Radiology 165:153–157
24. Toomayan GA, Holman WR, Major NM et al (2006) Sensitivity of MR arthrography in evaluation of acetabular tears. AJR Am J Roentgenol 186:449–453
25. Smith TO, Hilton G, Toms AP, Donell ST, Hing CB (2011) The diagnostic accuracy of acetabular labral tears using magnetic resonance imaging and magnetic resonance arthrography: a meta-analysis. Eur Radiol 21(4):863–874
26. Keeney JA, Peelle MW, Jackson J, Rubin D et al (2004) Magnetic resonance arthrography versus arthroscopy in the evaluation of articular hip pathology. Clin Orthop Relat Res 429:163–169
27. Filigenzi JM, Bredella MA (2008) MR imagine of femoroacetabular impingement. Appl Radiol 37:12–19
28. Pfirrmann CWA, Duc SR, Zaneti M et al (2008) MR arthrography of acetabular cartilage delamination in femoroacetabular cam impingement. Radiology 249:236–241
29. Byrd JWT, Jones KS (2004) Diagnostic accuracy of clinical assessment, magnetic resonance imaging, magnetic resonance arthrography, and intra-articular injection in the hip arthroscopy patients. Am J Sports Med 32:1668–1674
30. Mettler FA, Giuberteau MJ (2006) Skeletal system. In: Mettler FA, Giuberteau MJ (eds) Essentials of nuclear medicine imaging, 5th edn. Saunders, Philadelphia, pp 276–278
31. Matar WY, May O, Raymond F, Beaule PE (2009) Bone scintigraphy in femoroacetabular impingement: a preliminary report. Clin Orthop Relat Res 467(3):676–681
32. Banks KP, Song WS (2008) Acetabular impingement on planar and SPECT bone scintigraphy. Clin Nucl Med 33:916–919
33. Ghelman B, Freiberger RH (1979) The adult hip. In: Freiberger RH, Kaye JJ (eds) Arthrography. Appleton- century-Crofts, New York, p 189
34. Harper MC, Schaberg JE, Allen WC (1987) Primary iliopsoas bursography in the diagnosis of disorders of the hip. Clin Orthop Relat Res 221:238–241
35. Deslandes M, Guillin R, Cardinal E et al (2008) The snapping iliopsoas tendon: new mechanisms using dynamic sonography. AJR Am J Roentgenol 190:576–581

Femoroacetabular Impingement (FAI) and Sport

34

Raul Zini, Manlio Panascì, and Andrea Carraro

34.1 CAM and PINCER: What Are They?

FAI is a pathologic condition due to an abnormal contact between the articular rim of the acetabulum and the proximal femoral portion, at the junction between the neck and the head.

Specific radiologic features and clinical aspects characterize this condition, and although in past times it was considered as a consequence of the degenerative arthritic process of the hip, it is an important and frequent causal predisposition to arthritis itself [1].

Nowadays, a correct acknowledgment of this pathologic condition enlarges the therapeutic options in favor of a huge category of young and active patients, who can therefore greatly improve their prognosis "ad valetudinem" and posticipate an arthritic evolution of the hip.

Historically, the first clinical description of an abnormal contact between the femur and acetabulum belongs to Smith-Petersen in the 1930s; nevertheless, the author who has to be considered the real discoverer of FAI is undoubtedly R. Ganz, from Switzerland [2]. He described the peculiarities of this pathologic condition in the 1990s, and in 2008 he underlined that, in order to prevent hip arthritis, a correct therapeutic treatment of FAI has the same importance as a correct treatment of hip dysplasia [3].

An abnormal morphology of the femoral epiphysis or the acetabular cavity, or both of them, causes femoroacetabular impingement. CAM-type impingement is correlated to an abnormal conformation of the femoral head; PINCER type, on the contrary, is caused by an excessive coverage by the acetabular cavity toward the femoral head. In most cases, anyway, CAM and PINCER are contemporarily present in the same hip, creating a mixed FAI.

CAM impingement is sustained by an abnormal conformation of the head-neck junction at the proximal femoral epiphysis, with a loss of the femoral head sphericity and a mechanical limit to the normal range of motion. The loss of sphericity of the femoral head is determined by a bony prominence at the head-neck junction; this bony prominence is usually called "bump," and it causes a precocious contact between the femur and the acetabular edge (and, of course, the acetabular labrum) in the movement of flexion and internal rotation [4, 5].

An excess of acetabular surface covering the femoral epiphysis causes PINCER impingement. The excessive acetabular coverage can be localized, in case of acetabular retroversion, or global and generalized, in case of "coxa profunda" or "protrusio acetabuli" [6]. Although this abnormal conformation grants a major area of superficial contact between femoral head and acetabular cavity, it is easy to understand that it leads to a precocious contact between the acetabular edge and the femoral neck [7].

34.2 CAM and PINCER Physiopathology

As previously mentioned, CAM impingement is characterized by a loss of the femoral head sphericity, with a bony prominence at the head-neck junction which conflicts against the acetabular labrum determining a progressive damage of the labrum itself and finally a detachment of the latter.

Moreover, the continuous pressure that the bump has on the labrum gradually causes a delamination of the chondral acetabular surface, which is just adjacent to the labrum. This microtraumatic process is due to a tangential force vector created by the bump against the acetabular edge and finally leads to the formation of an unstable cartilage flap and an enlarging area of exposed subchondral bone at the peripheral board of the acetabulum: this is the beginning of an arthritic process in the anterosuperior portion of the hip.

R. Zini • M. Panascì (✉) • A. Carraro
Department of Orthopedic Surgery, GVM Care and Research,
Via Corriera 1, Cotignola 48010, Italy
e-mail: raulzini@gmail.com; manliopa@gmail.com;
Andr.carraro@libero.it

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_34,

CAM impingement is more frequent in young and active men, with clinical appearance usually before 30 years of age [8].

The very first symptom is pain, usually located at the groin and laterally, in the trochanteric area (C sign: the patient indicates the painful area using the first two fingers of his hand, thus forming a "C" that embraces the area across the great trochanter, from anterior to posterior). Pain becomes much more continuous and intense as time goes by and is higher in case of long sitting position and in the movement of flexion and internal rotation of the hip (the latter consisting in the "anterior impingement test"). At the objective examination, internal rotation is often very poor or completely absent, just as the combined movement of flexion, abduction, and external rotation (FAbEr test). At the beginning of the symptoms caused by CAM impingement, very often the acetabular cartilage damage, which is peculiar of this pathologic condition, is already present, as it is sensibly precocious although clinical symptoms may be very occasional [9]. On the contrary, in case of PINCER impingement, the compression of the acetabular labrum activates the proprioceptive fibers belonging to the labrum itself, so pain precociously appears and patients affected by PINCER impingement usually consult an orthopedic surgeon before a cartilage damage has occurred. Obviously, in case of PINCER impingement, the acetabular labrum is the very first component to be involved in the contact between the femoral neck and prominent acetabular edge, and this contusive trauma leads to labrum tear and degeneration. Moreover, PINCER impingement causes a recoil damage in the posteroinferior portion of the acetabulum, because the contact between the prominent acetabular edge and the femoral neck acts as a fulcrum that shifts the femoral head frontward, till hitting the inferior portion of the cavity: this pathologic mechanism leads to the creation of the so-called contrecoup lesion. PINCER impingement is more frequent in active women, and symptoms usually appear later than in case of CAM impingement, at about 40 years of age. Differently from CAM impingement, the articular movement which mostly evocates pain is extension and external rotation of the hip, thus creating the "posterior impingement test" [3, 7]. Pain can be localized either at the groin, trochanteric area, or, typically in case of PINCER-type FAI, gluteal area, specifically because of the contrecoup lesion previously mentioned above.

34.3 CAM and PINCER Anatomopathology

In order to comprehend correctly and deeply the anatomopathology of FAI, a complete and accurate radiologic evaluation is essential [10]. First of all, it is mandatory to obtain a correct AP x-ray evaluation: 120 cm between the origin of the x-ray and the patient, whose inferior limbs have to be internally rotated for 15°, in order to compensate the physiological femoral neck anteversion. Moreover, an axial projection is necessary, for a better evaluation of the anterior edge of the head-neck junction. Various radiologic techniques have been described to obtain a correct axial view of the hip. One of the most efficient and easily obtainable axial projection is the "modified Dunn view," which is an AP projection to the hip in slight abduction (20°) and flexion (45°); another common technique for the axial view is an AP projection in "frog leg position" of the patient's lower limbs [11]. In the normal hip, the anterior edge of the acetabulum remains in a medial position in comparison to the posterior edge. On the contrary, in case of acetabular retroversion or excessive coverage by the acetabular walls toward the femoral head (i.e., in the anatomic condition that leads to a PINCER impingement) in AP projections, the anterior edge of the acetabulum stands in a more external position than the posterior edge. Sometimes, a figure of "8" appears, designed by the lines of the two acetabular edges, the anterior and the posterior ones, crossing each other: such a radiographic finding is called "crossover sign" and is pathognomonic of an acetabular retroversion [12]. In case of CAM impingement, the presence of the "bump" at the head-neck junction appears as a prominence that, in axial x-ray projections, makes the femoral epiphysis similar to pistol grip. In axial projections, furthermore, an easy method to evidence the presence of the bump and, consequently, a CAM-type impingement is the measurement of the head-neck offset: in case of CAM impingement, the presence of the bump causes a reduction of the offset (being the imaginary line passing on the anterior edge of the femoral neck much closer to the imaginary line passing for the anterior limit of the femoral head than it should be in normal cases) [13].

Even more important is the measurement, again in axial x-ray projections, of the alpha angle. The higher this angle appears, the bigger is the bump determining the CAM impingement. In fact, the alpha angle is formed by two lines: one is the femoral neck axis and the other is the radius of the ideal circumference of the femoral head at the point where the profile of the femoral head no longer follows the ideal circumference, but continues into the neck profile. Therefore, in normal hips the alpha angle is always <50°, while in case of CAM impingement, the presence of the bump alters the profile of the neck-head junction, increasing the value of alpha angles always >50°, sometimes up to nearly 90° [14].

MRI gives a great help for the study of the articular anatomopathology in case of FAI. This exam has to be performed with contrasting solution (gadolinium) directly in the articulation (arthro-MRI), in order to evaluate the condition of the acetabular labrum; arthro-MRI proved to be very sensible and precise in detecting the status of the labrum and the gravity of its lesions, from the very precocious alterations of its structure till the complete detachment from the bony edge of the acetabulum [15–17].

3D CT static reconstructions offer beautiful and intuitive imagines of the anatomic structures, but they can offer no more information than a well-performed x-ray exam and an arthro-MRI.

On the opposite, new devices with 3D CT dynamic reconstruction could actually change the way we have been treating patients, by identifying areas and degree of hip impingement between the pelvis and femur, automating measurements, and providing adjustable ROM simulations that allow the development of a presurgical resection plan based on a patient's morphology [18].

34.4 Surgical Technique

Supine position is preferred in our routine. Traction is given with a dedicated system to obtain sufficient space and to perform arthroscopy without the risk for the intra-articular structures. We routinely use a 20 cm post pad in order to minimize risk of pudendal nerve palsy. Time of traction should always be no more than 2 h [19].

Standard anterolateral and mid-anterior portals are usually sufficient to perform the entire surgical procedure. We always start with a central compartment evaluation, and we routinely perform interportal capsulotomy, which allows a safer and easier instrument movement around the hip joint. Every structure is assessed to look for any pathologic sign, and treatment of every possible cause of pain is achieved. Diagnostic central compartment arthroscopy is performed with a 70° arthroscope to identify any labral or acetabular chondral lesions as well as the impingement: in case of a labrum tear, we usually reattach it with resorbable suture anchors; a debridement is a second choice only in case of degenerative lesions. Labral repair with a simple looped stitch, labral base stitch, or vertical mattress technique is chosen depending on labrum dimensions. It represents an evolution for orthopedic sports medicine, and although reports of labral debridement have been promising, restoring the normal chondrolabral junction with suture anchor repair techniques can potentially provide a more viable option for the healing potential of the labrum [20–22]. Chondral pathologies are treated with shaving, microfractures, or biomimetic scaffold positioning depending on the age of the patient and the size of the defect.

When decompression is performed, it is important not to neglect findings of impingement, but it is also important not to overtreat all abnormal radiographic changes.

PINCER-type impingement decompression is achieved under fluoroscopic control: after detaching the labrum from the bone with a beaver blade, trimming of the rim with a 5.5 mm round-tip burr is performed; if a subspine impingement is present, arthroscopic decompression is achieved (Fig. 34.1a, b). In patients who had showed relief from psoas injections, transcapsular evaluation of the iliopsoas tendon is executed just before removing the traction. A correlation between the psoas tendon and the status of the anterior labrum at approximately the 3 o'clock position is performed. In case of fraying or an erythematous, contusion-type lesion of the labrum, we performed a fractional release of the tendinous portion of the iliopsoas musculotendinous unit with a radiofrequency probe. A fractional lengthening of the psoas is also performed in case of impingement with the medial portion of the acetabular rim.

CAM-type impingement treatment is possible without traction and a 45° hip flexion. It is sometimes indicated to perform a longitudinal capsulotomy to better reach the head-neck junction. The scope is inserted from the anterolateral portal, while the instruments (shaver, burr) are inserted from the mid-anterior portal, but switching is common and very useful for a better three-dimensional assessment (Fig. 34.1c, d). Lateral-based lesions are challenging due to the intimate location of the retinacular vessels; thus, proper attention should be given to the vascular anatomy. Osteochondroplasty should include all pathologically appearing cartilage, but shall not go higher or more proximally to the epiphyseal scar, which can be confirmed fluoroscopically. It is also fundamental to perform a dynamic evaluation to ensure an adequate decompression, and this should be the last gesture before arthroscopy can be considered completed.

34.5 Technical Notes

Consider preoperative planning to evaluate the amount of acetabular trimming and femoroplasty (3D CT dynamic reconstruction)
Careful attention in portal placement when entry into the joint (labrum penetration, femoral head scuffing)
If rim trimming is not appreciated on intraoperative fluoroscopic imaging, direct arthroscopic visualization, dynamic testing, and preoperative x-rays should guide further resection
Divergent suture anchor placement orientation is recommended to prevent screw penetration of the acetabulum
"T" capsulotomy is useful in case of a wide femoral bump
Address lateral retinacular vessels before starting femoral decompression
Fluoroscopy and dynamic evaluation are mandatory to confirm the amount of bone resection

34.6 Rehabilitation

After hip arthroscopy, athletes wish to return to a fully active lifestyle and to practice their preferred sport as soon as possible. Currently, the best evidence for postoperative rehabilitation is based upon few scientific production; thus, communication with the specialist is vital to the treating

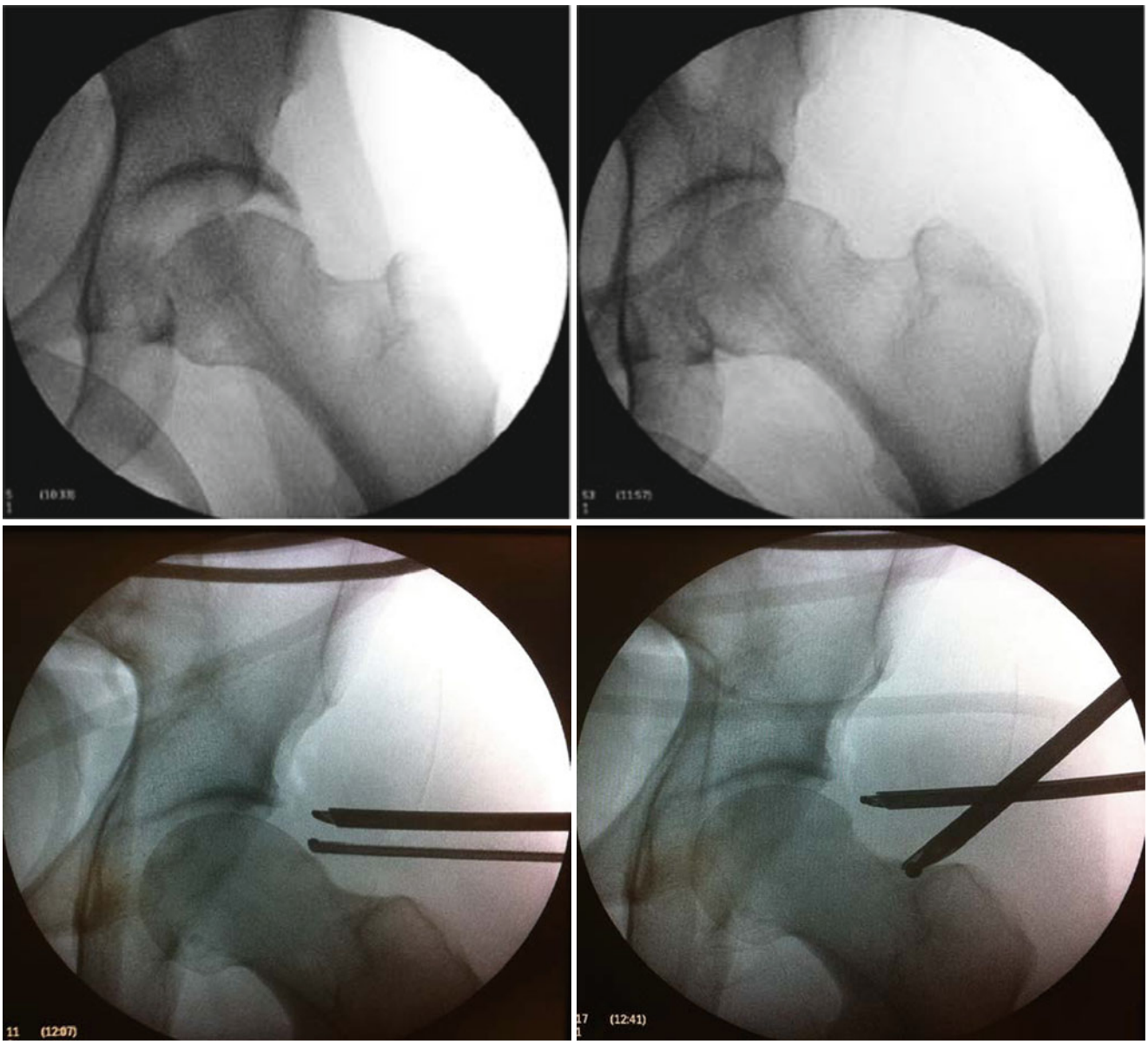


Fig. 34.1 PINCER fluoroscopy image (**a**), before and after rim trimming (**b**). Intraoperative fluoroscopic check before (**c**) and after femoroplasty (**d**), confirming complete CAM resection

physical therapist in order to give an individualized and evaluation-based program [23, 24].

Before starting rehabilitation, it is fundamental to know the exact procedure and operative findings, to plan a truly customized rehabilitation program both for simple and complex procedures (Table 34.1, Fig. 34.2).

Rehabilitation can be divided into four phases. The timeline for each phase is based on clinical findings. If clinical presentation meets the established criteria, the athlete may move to the next phase. Progression in terms of the type and intensity of the workout should be function based, not time based (Table 34.2).

Table 34.1 Classification based on the complexity of the surgical procedure

Simple	Diagnostic
	Removal loose body
	Labral debridement
	Ligamentum teres debridement
Intermediate	CAM decompression
	Iliotibial band release
	Iliopsoas release
Complex	Acetabular rim trimming + labral repair + CAM decompression
	Microfracture
Very complex	Acetabular rim trimming + labral repair + CAM decompression + capsular plication

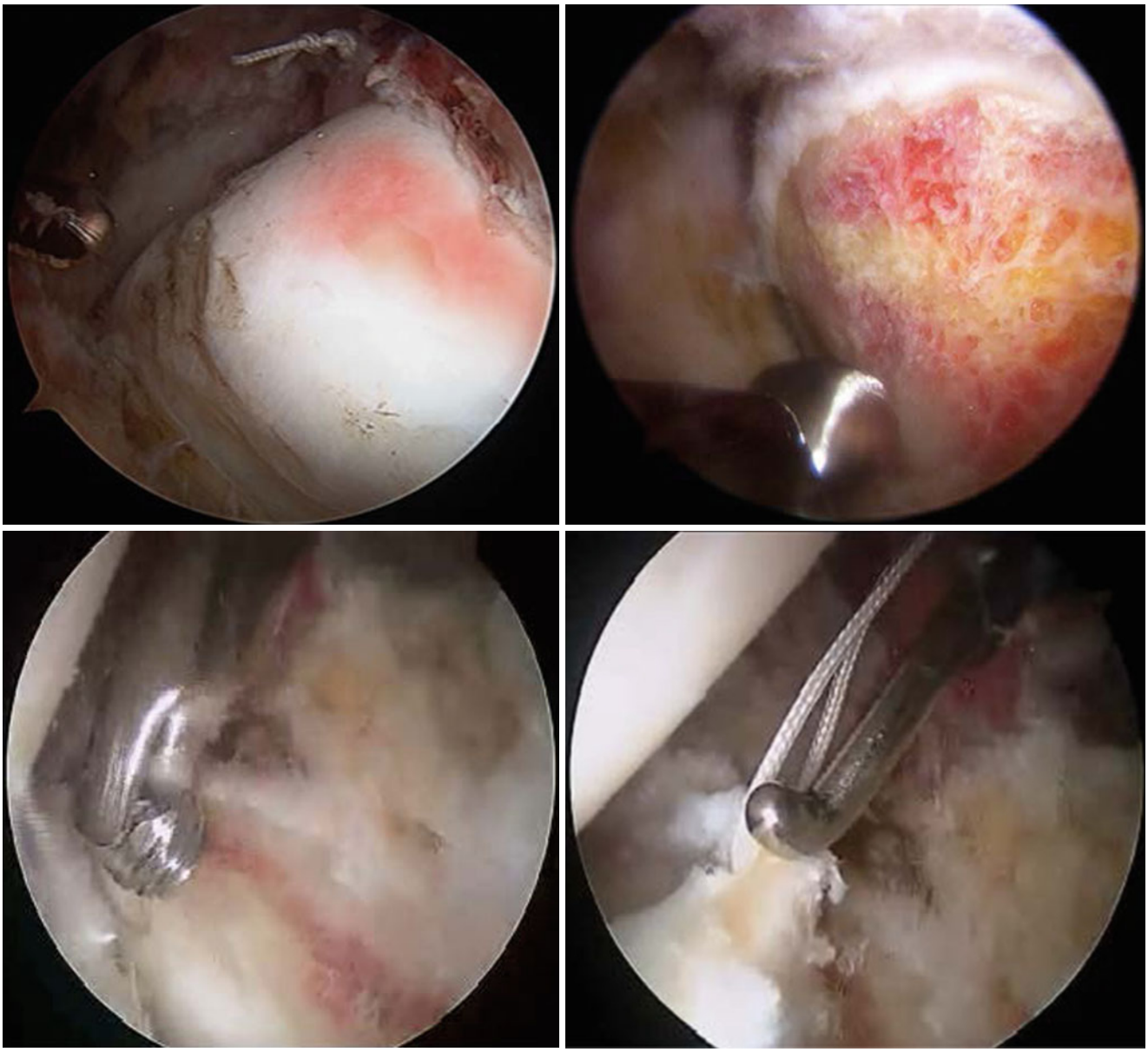


Fig. 34.2 Arthroscopic CAM image before (**a**) and after femoroplasty (**b**). Acetabuloplasty (**c**) and labrum refixation after rim trimming (**d**)

Table 34.2 Schedule of the rehabilitation program

Phase I	Start mobilization and isometric exercises; avoid swelling
Phase II	Continue recovery of range of motion and isometric exercises
Phase III	Recovery of full strength
Phase IV	Recovery of balance and neuromuscular control
Phase V	Functional recovery and return to sport

It would be helpful to see the player preoperatively to prepare the affected joint and explain process and timescales involved. It should also be mandatory to give written rehabilitation indications at discharge.

Time recovery for a full activity is usually 4 months, but it may last longer depending on operative findings or prolonged rehabilitation.

It is fundamental not to force recovery [25]. Possible risks of a premature return to sports activity are:

- Persistent pain
- Prolonged rehabilitation time
- Low performance
- Reinjury (new labral tear, articular cartilage lesion)
- New injuries

34.7 Outcome and Return to Play

Several published articles have been written on athletic patient population after hip arthroscopy.

Results of this studies show that athletes with FAI can return to high-level competitive sport following this procedure.

Philippon has published a cohort study of 28 professional hockey players who underwent hip arthroscopy for FAI. The return to sport was 3.8 months (range, 1–5 months) with MHHS of 95 at follow-up. Patients with symptoms lasting less than 1 year returned to sport at 3 months, but patients who delayed surgery over 1 year returned to sport at 4.1 months [26].

Brunner et al. in 2009 reported that values return to full sporting activity in 68.8 % of cases [27].

Byrd in 2009 reported its results with a mean follow-up of 27 months. In 90 % of professional and 85 % of college athletes, there was a return to full sports activity [28].

Another study by Nho et al. estimated the return to sport in patients undergoing hip arthroscopy up to 83 % [29].

A recent systematic review showed a high rate of return to pre-injury activity level in athletes treated for FAI. Results achieved a 92 % rate return to activity, observed in athletic populations across a variety of sports, with 88 % of athletes returning to pre-injury activity levels of participation [30].

Arthroscopic management among athletes is very favorable, but often performed when an important damage has occurred. Substantial secondary damage is frequently present that cannot be completely reversed. In fact FAI is very often not recognized, leading to a delay for a precise diagnosis. Early recognition and treatment have been demonstrated to have a tremendous impact on outcome; Philippon has showed that patients with symptoms lasting less than 1 year returned to sport at 3 months, but patients who delayed surgery over 1 year returned to sport at 4.1 months. Because early treatment is the only change for full recovery, athletes that decide to delay treatment should be aware of the risk [31].

Rehabilitation after hip arthroscopy is long and has its own peculiarity; thus, it is fundamental to follow the patient during the entire post-op protocol. Data suggest that professional athletes may show quicker return to sports than recreational athletes, but the hip scores and rate of return seem to be analogous. Villar recently published his result showing a quicker recovery in pros, with no statistical difference when comparing the overall return both in recreational and professional athletes [32].

In conclusion, hip arthroscopy is becoming the first choice for FAI treatment. Athletes have interest in early recovery and prompt return to play, and the less-invasive arthroscopic approaches are able to reach the desire for physical activity with good clinical outcomes and high satisfaction rate.

References

1. Burnett RS, Della Rocca GJ, Prather H, Curry M, Maloney WJ, Clo- hisy JC (2006) Clinical presentation of patients with tears of the acetabular labrum. J Bone Joint Surg Am 88:1448–1457
2. Ganz R, Parvizi J, Beck M et al (2003) Femoroacetabular impingement: a cause for osteoarthritis of the hip. Clin Orthop Relat Res 417:112–120
3. Ganz R, Leunig M, Leunig-Ganz K, Harris WH (2008) The etiology of osteoarthritis of the hip: an integrated mechanical concept. Clin Orthop Relat Res 466:264–272
4. Johnston TL, Scheker BS, Phillippon MJ et al (2008) Relationship between offset angle alpha and hip chondral injury in femoroacetabular impingement. Arthroscopy 24(6):669–675
5. Gosvig KK, Jacobsen S, Sonne-Holm S, Gebuhr P (2008) The prevalence of cam-type deformity of the hip joint: a survey of 4151 subjects of the Copenhagen Osteoarthritis Study. Acta Radiol 49:436–441
6. Steppacher SD, Tannast M, Werlen S, Siebenrock KA (2008) Femoral morphology differs between deficient and excessive acetabular coverage. Clin Orthop Relat Res 466:782–790
7. Tanzer M, Noiseux N (2004) Osseus abnormalities and early osteoarthritis: the role of hip impingement. Clin Orthop Relat Res 428:170–177
8. Hack K, Di Primio G, Rakhra K, Beaule PE (2010) Prevalence of cam-type femoroacetabular impingement morphology in asymptomatic volunteers. J Bone Joint Surg Am 92(14):2436–2444
9. Lorentzon R, Wedren J, Pietila T (1998) Incidence, nature and cause of ice hockey injuries: a three-year prospective study of a Swedish elite ice hockey team. Am J Sports Med 16:392–396
10. Dudda M, Albers C, Mamisch TC, Werlen S, Beck M (2009) Do normal radiographs exclude asphericity of femoral head-neck junction? Clin Orthop Relat Res 467(3):651–659
11. Tannast M, Siebenrock KA, Anderson SE (2007) Femoroacetabular impingement: radiographic diagnosis—what the radiologist should know. AJR Am J Roentgenol 188(6):1540–1552
12. Meyer DC, Beck M, Ellis T, Ganz R, Leunig M (2006) Comparison of six radiographic projections to assess femoral head/neck asphericity. Clin Orthop Relat Res 445:181–185
13. Notzli HP, Wyss TF, Stoecklin CH, Schmid MR, Treiber K, Hodler J (2002) The contour of the femoral head-neck junction as a predictor for the risk of anterior impingement. J Bone Joint Surg Br 84:556–560
14. Siebenrock KA, Wahab KH, Werlen S, Kalhor M, Leunig M, Ganz R (2004) Abnormal extension of the femoral head epiphysis as a cause of cam impingement. Clin Orthop Relat Res 418:54–60
15. Werlen S, Leunig M, Ganz R (2005) Magnetic resonance arthrography of the hip in femoroacetabular impingement: technique and findings. Oper Tech Orthop 15:191–203
16. Czerny C, Hofmann S, Neuhold A et al (1996) Lesions of the acetabular labrum: accuracy of MR imaging and MR arthrography in detection and staging. Radiology 200:225–230
17. Ito K, Minka MA, Leunig M et al (2001) Femoroacetabular impingement and the cam effect. A MRI-based quantitative anatomical study of the femoral head-neck offset. J Bone Joint Surg Br 83:171–176
18. Khan O, Witt J (2014) Evaluation of the magnitude and location of Cam deformity using three dimensional CT analysis. Bone Joint J 96-B(9):1167–1171. doi:10.1302/0301-620X.96B9.33555
19. Clarke MT, Villar RN (2003) Hip arthroscopy: complications in 1054 cases. Clin Orthop Relat Res 406:84–88
20. Philippon MJ, Arnoczky SP, Torrie A (2007) Arthroscopic repair of the acetabular labrum: a histologic assessment of healing in an ovine model. Arthroscopy 23:376–380

21. Kelly BT, Shapiro GS, Digiovanni CW, Buly RL, Potter HG, Hannafin JA (2008) Vascularity of the hip labrum: a cadaveric investigation. Arthroscopy 21:3–11
22. Bedi A, Chen N, Robertson W, Kelly BT (2008) The management of labral tears and femoroacetabular impingement of the hip in young, active patient. Arthroscopy 24:1135–1145
23. Cheatham SW, Kolber MJ (2012) Rehabilitation after hip arthroscopy and labral repair in a high school football athletes. Int J Sports Phys Ther 7(2):173–184
24. Bennell KL, O'Donnell JM, Takla A, Spiers LN, Hunter DJ, Staples M, Hinman RS (2014) Efficacy of a physiotherapy rehabilitation program for individuals undergoing arthroscopic management of femoroacetabular impingement – the FAIR trial: a randomised controlled trial protocol. BMC Musculoskelet Disord 26;15:58
25. Emery CA, Meeuwisse WH (2001) Risk factors for groin injuries in hockey. Med Sci Sports Exerc 33(9):1423–1433
26. Philippon MJ, Wiess DR, Kuppersmith DA, Briggs KK, Hay CJ (2010) Arthroscopic labral repair and treatment of femoroacetabular impingement in professional hockey players. Am J Sports Med 38(1):99–104
27. Brunner A, Horisberger M, Herzog RF (2009) Sports and recreation activity of patients with femoroacetabular impingement before and after arthroscopic osteoplasty. Am J Sports Med 37(5): 917–922
28. Byrd JW, Jones KS (2009) Arthroscopic femoroplasty in the management of cam-type femoroacetabular impingement. Clin Orthop Relat Res 467(3):739–746
29. Nho SJ et al (2011) Outcomes after the arthroscopic treatment of femoroacetabular impingement in a mixed group of high-level athletes. Am J Sports Med 39(Suppl):14S–19S
30. Alradwan H, Philippon MJ, Farrokhyar F et al (2012) Return to preinjury levels after surgical management of femoroacetabular impingement in athletes. Arthroscopy 28(10):1567–1576
31. Philippon M et al (2007) Femoroacetabular impingement in 45 professional athletes: associated pathologies and return to sport following arthroscopic decompression. Knee Surg Sports Traumatol Arthrosc 15(7):908–914
32. Malviya A, Paliobeis CP, Villar RN (2013) Do professional athletes perform better than recreational athletes after arthroscopy for femoroacetabular impingement? Clin Orthop Relat Res 471(8): 2477–2483

Hip Tears of the Acetabular Labrum

35

J.W. Thomas Byrd and Jason C. Phillips

35.1 Introduction and Etiology

Although several indications currently exist for arthroscopic evaluation of the hip, the most common indication remains tears and pathology involving the acetabular labrum. Byrd began to publish on labral tears as a source of pain in 1996 [1]; however, Ganz was the first to coin the term "femoroacetabular impingement" (FAI) as the main underlying cause of labral pathology [2]. It is paramount to understand that the majority of labral pathology occurs as a secondary phenomenon [3]. Labral tears can be a source of debilitating hip pain for athletes and can result in a significant decline in athletic performance. As such, understanding the etiology, diagnosis, and management of hip labral pathology is essential for any sports medicine physician.

The etiology of labral tears can either be traumatic or atraumatic in origin. Traumatic labral tears occur much less frequently than atraumatic tears. These tears, however, rarely occur without any underlying morphological abnormality of the hip. With traumatic tears, athletes will be able to recall a single event that triggered the onset of their pain. Traumatic tears can occur secondary to a subluxation-relocation event and a frank hip dislocation or from a tackle or fall with the hip in a position of extreme motion. With the hip in a position of flexion, adduction, and internal rotation, a posteriorly directed force can produce shearing of the posterior labrum resulting in a traumatic posterior labral tear. With the hip in a position of extension, abduction, and external rotation, a traumatic anterior labral tear can result. In the setting of a frank hip dislocation, intra-articular pathology is to be expected. Philippon performed arthroscopic evaluation of the hip in 14 professional athletes following a traumatic hip dislocation event, and all 14 had labral tears and chondral defects, and 11 of the 14 patients had tears of the ligamentum teres [4].

Atraumatic labral tears comprise the majority of labral tears. These occur secondary to some underlying phenomenon and are usually reported as a slow, gradual onset of hip pain, with a steady decline in athletic performance. Atraumatic labral tears can occur from acetabular deficiency (dysplasia), acetabular over-coverage or retroversion (pincer impingement), acetabular extra-articular impingement (subspine impingement) [5], loss of femoral head-neck offset (cam impingement), femoral retroversion [6], and, in some cases, possibly iliopsoas impingement [7]. Often multiple abnormalities lead to labral tearing, and failure to recognize the various contributing factors will compromise the results of any treatment strategy, both operative and nonoperative.

Acetabular dysplasia results from a poorly formed and underdeveloped acetabular socket. Radiographically, this is best characterized by a hip with a lateral center-edge angle (CEA) of Wiberg of less than 25° [8], an anterior center-edge angle of Lequesne of less than 25° [9], a hip migration index of Reimer of greater than 15–20 % [10], and a McKibbin index of greater than 50 [11]. The femoral neck will often show increased valgus. The loss of normal acetabular coverage, combined with increased femoral valgus, results in both static and dynamic overload of the anterosuperior and lateral labrum. Athletes involved in sports which require extremes of hip motion (i.e., gymnastics) may oftentimes have accompanying acetabular dysplasia. Recognition of hip dysplasia is important because isolated arthroscopic labral refixation is unlikely to result in a satisfactory outcome. Oftentimes, these athletes will require a periacetabular osteotomy, a femoral derotational osteotomy, or both in combination with either an open or arthroscopic labral refixation.

Labral tearing is mostly commonly associated with femoroacetabular impingement. This can occur from isolated acetabular impingement (pincer), isolated femoral impingement (cam),

J.W.T. Byrd, MD (✉)
Nashville Sports Medicine Foundation, Clinical Professor Orthopaedic Surgery and Rehabilitation, Vanderbilt University School of Medicine, 2011 Church St., Suite 100, Nashville, TN 37203, USA
e-mail: info@nsmfoundation.org

J.C. Phillips, MD
Phillips-Brayford Orthopaedics, 48 Tunnel Rd., Suite 203, Pottsville, PA 17901, USA

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_35,

or most commonly from combined cam and pincer impingement. Pincer impingement results from acetabular over-coverage which is defined as a lateral CEA greater than 40°, acetabular retroversion causing anterior impingement, or acetabular anteversion causing posterior impingement [12]. Cam impingement results from a loss of femoral head-neck offset as the large femoral head tapers in a narrower femoral neck. Because of the ball and socket nature of the hip joint, subtle joint incongruities between the proximal femur and acetabulum are poorly tolerated. As such, even a slight loss in femoral head-neck offset can result in cam impingement. Femoral version also plays a very important role in the magnitude of cam impingement [6]. Specifically, femoral retroversion increases the likelihood of cam impingement occurring, as the femoral neck is more exposed due to the retroverted nature of proximal femur. For example, a small loss of head-neck offset in a patient with a retroverted acetabulum will result in earlier engagement of the cam lesion in hip flexion and internal rotation, whereas that same lesion may be asymptomatic in a patient with a normal amount of femoral version, approximately 5–15° [6, 12, 13].

35.2 Clinical and Diagnostic Examination

The evaluation of the athlete with hip pain starts with a thorough history. Specifically, pain onset, duration, intensity, inciting events, alleviating factors, injection history, physical therapy protocols, prior operative treatments, as well as any imaging studies that have been performed should be discussed in great detail. If the pain started with a single event during sporting activity, oftentimes the event was video recorded, and the athlete should be asked to provide that as well. The team trainer is often an excellent source of information as trainers are the first line of treatment for most athletes.

The location of the pain will guide the physician as to the true pain generator. Oftentimes, the athlete will have developed compensatory pain due to attempts to play through the pain. Labral pathology will often present with groin-based pain that can radiate posteriorly into the buttocks as a result of the circumferential capsular inflammation and irritation that occur secondary to labral pathology. The author has described the "C-sign" which is often used by patients to describe the deep groin pain they are experiencing as a result of labral pathology (Fig. 35.1). Trochanteric-based pain is very common in athletes in the setting of labral pathology. Oftentimes, athletes will be told that they are experiencing trochanteric bursitis. It is the authors' experience that trochanteric bursitis is very rare in the young athletic population and that its existence should heighten the physician's suspicion that there exists a deeper underlying problem. Trochanteric-based pain usually represents a true gluteus medius and minimus tendinitis that has secondarily developed in an attempt to stabilize the hip. In the setting of labral pathology, hip microinstability has been postulated but, at the very least, the abductors are working hard to splint the hip, leading to abductor tendinitis. Internal snapping due to "popping" of the iliopsoas tendon may be present as a coincidental finding. Inflammation may accompany joint pathology, and the popping may become painful. Mechanical symptoms often accompany labral tears, occasionally to the point of creating intra-articular popping.

Fig. 35.1 (**a**, **b**) The C-sign. This term reflects the shape of the hand when a patient describes deep interior hip pain. The hand is cupped above the greater trochanter with the thumb posterior and the fingers gripping deep into the anterior groin (Reprinted with permission J. W. Thomas Byrd, MD)

Special attention should be paid to abdominal-based pain, as athletic pubalgia (AP) can often be present in the setting of hip pathology. Hammoud has shown that athletes with symptomatic FAI also have a high incidence of AP with 32 % of the patients in their series having undergone surgical treatment for both FAI and athletic pubalgia [14]. Patients with AP will often complain of pain near the pubic tubercle at the insertion of the conjoint tendon of the rectus abdominis musculature. Pain may also be present in the groin along the course of the adductor musculature, making an accurate distinction between the true causes of groin pain even more difficult. Patients with AP will often complain of pain with sudden activities such as sneezing, coughing, sprinting, kicking, and sit-ups, but rarely have pain at rest [15, 16].

Physical examination of the hip starts with an evaluation of the patient's gait pattern. Specifically, patients may oftentimes present with an antalgic gait pattern secondary to the inflammation and pain they are experiencing in their hip. With an antalgic gait pattern, the stance phase of gait is abnormally shortened relative to the swing phase. A Trendelenburg gait may also be present as a result of abductor weakness. During the stance phase, normal abductor strength is necessary to maintain a level pelvis. To compensate, the trunk lurches to the weakened side, reducing the forces required of the abductors.

The patient is then laid supine on the exam table in a pair of shorts, with their shoes and socks off. Leg lengths should also be measured from the anterior superior iliac spine down to the medial malleolus. Leg length discrepancies are not uncommon and can result in an altered gait pattern. The resting position of the feet can provide clues to the capsular status of the hip joint. Patients with hip pathology may develop a contracted hip capsule due to synovial inflammation or capsular laxity due to labral tearing and the loss of the suction-seal effect of the labrum which results in repeated capsular overload and stretching [17, 18]. This is best appreciated by comparing the resting position of the foot in the affected limb versus that of the unaffected limb. In the setting of a contracted hip capsule, the foot will sit internally rotated compared to the contralateral side. In a hip with capsular laxity, the opposite holds true as the foot sits externally rotated compared to the contralateral unaffected limb. The dial test can also be used to assess capsular laxity. This test is performed with the patient lying supine on the examination table with the limb in the neutral position. The limb is then internally rotated and then released, allowing passive external rotation. A positive test occurs when the patient's limb passively externally rotates beyond 45° and lacks a solid end point [19].

Range of motion testing is then performed with the patient in the supine position. Flexion, extension, abduction, adduction, internal rotation, and external rotation are all assessed. It is important, especially when testing hip flexion, to place a hand on the anterior superior iliac spine to allow the examiner to detect pelvic rotation when assessing range of motion. Once the pelvis starts to rotate or lift off the table, true isolated hip motion has ceased. This is the equivalent of assessing true glenohumeral motion in the shoulder by eliminating scapulothoracic motion. To determine true hip joint motion, compensatory pelvic motion must be eliminated to obtain an accurate measure. In the setting of FAI, hip flexion and internal rotation are often decreased due to a mechanical block to motion. A global loss of motion is not uncommon due to the synovial hypertrophy and inflammation that result from labral pathology and the impingement-mediated process. Excessive joint mobility and motion is often present in patients with hip dysplasia, as the normal bony constraints are no longer present, allowing for supraphysiologic motion to occur. Patients without dysplasia may develop capsular laxity as a result of the loss of the normal stabilizing suction-seal effect of the labrum. This results in capsular overload and eventual capsular stretching with plastic deformation. This most commonly involves the anterior capsular and iliofemoral ligament, resulting in an increase in hip extension and external rotation. Femoral version can be predicted from the sum total of internal and external range of motion. Increased internal rotation with a loss of external rotation is seen with increased femoral anteversion. Loss of internal rotation with an increase of external rotation is seen with femoral retroversion. One special note about adhesive capsulitis, occasionally encountered in middle-aged females: a characteristic feature is disproportionate painful restriction of external rotation compared to internal rotation [20].

Several examination maneuvers exist to detect labral pathology. The log roll test is simply a passive internal and external rotation test of the hip with the patient in the resting supine position, and a positive test is specific for intra-articular irritation of the hip joint. The flexion, adduction, internal rotation (FADDIR) test engages the femoral neck against the anterior rim of the acetabulum and recreates labral impingement [21]. With the patient in the supine position, the hip is maximally flexed, internally rotated, and adducted. Recreation of groin-based pain is considered a positive test for labral pathology. The flexion, abduction, external rotation (FABER) test is used to detect lateral labral pathology and also to stress the anterior capsulolabral complex. With the patient supine, the hip is brought into 90° of flexion, abduction, and external rotation so that the foot sits on the contralateral knee. The height of the knee from the examination table is then measured and compared to the contralateral side. This provides some insight into the integrity of the iliofemoral ligament and anterior capsular structures. This also provides the maximum amount of stress to the lateral labrum and can help detect labral pathology [22]. The Stinchfield test is used to detect iliopsoas irritation and possible iliopsoas impingement against the labrum [23]. With the patient lying supine, a straight leg raise is actively performed

to 45°, at which time active downward pressure is applied to the thigh, isolating the iliopsoas complex. Recreation of pain or weakness indicates a positive test. The posterior rim impingement test is performed by bringing the leg into extension and external rotation, allowing for engagement of the femoral neck against the posterior acetabular rim. This is often performed by bringing the patient's leg off the end or side of the table to allow for full hip extension to occur.

With the patient in the lateral position, the palpation of the trochanteric facets can be performed [24]. This allows for the detection of possible abductor pathology which, in the setting of labral pathology, is often a true gluteus medius or minimus tendinitis. Passive adduction tests are also performed in the lateral position to detect tightness of the iliotibial band, gluteus medius, and gluteus maximus. Each test is performed by allowing passive adduction of the limb. With the knee in full extension, contracture of the iliotibial band can be detected. With the knee flexed to 90°, contracture of the gluteus medius can be detected. With the knee in full extension and the hip forward flexed, gluteus maximus contracture can be detected [23].

Diagnostic studies to diagnose labral pathology include plain radiographs and MRI. Plain radiographs should always include a well-entered AP pelvis [25] and, variably, a frog lateral [26], a cross-table lateral, and a false-profile view [27]. An appropriate AP pelvis x-ray is indicated by the coccyx being centered over the pubic symphysis, with the tip of the coccyx 1–2 cm from the pubic symphysis [28]. This allows for standardization and correction of pelvic tilt and inclination which can alter the acetabular orientation and version [29]. The lateral center-edge angle of Wiberg, the Tonnis angle, and the acetabular version are measured from the AP pelvis radiograph. A crossover sign is indicative of acetabular retroversion and indicates possible pincer impingement. A normal lateral center-edge angle is between 25° and 40°, with less than 25° representing dysplasia and greater than 40° representing acetabular over-coverage [12]. The cross-table lateral and frog radiographs allow for evaluation of the head-neck offset and measurement of the alpha angle to determine if cam impingement exists. Normal head-neck offset measures approximately 10 mm, with anything less suggesting cam impingement [30]. A normal alpha angle measures approximately 50° or less, and anything greater than that may indicate cam impingement [31]. The false-profile view allows for assessment of the anterior center-edge angle and gives an assessment of anterior acetabular coverage. It also allows for visualization of the anterior-inferior iliac spine morphology, as this may serve as a source of extra-articular impingement [5, 32]. Lastly, in cases of suspected degenerative disease, the false profile can be very helpful to detect anterior or posterior joint space narrowing that may be missed in an AP projection.

If labral pathology is suspected, conventional magnetic resonance imaging (MRI) or MRI combined with arthrography (MRA) may be useful. Both demonstrate greater sensitivity at detecting labral lesions than accompanying articular damage [33]. MRA reports better sensitivity, but eliminates the ability to assess for the presence of an effusion and may obscure subchondral or soft tissue edema [34, 35]. A negative study should not preclude consideration of arthroscopic treatment and evaluation when there is a high clinical suspicion of a labral lesion, as even MRA has a poor negative-predictive value and cannot be used to rule out a labral lesion [34, 36]. X-rays are used as more of a screening tool, but once the diagnosis of FAI with labral pathology has been established and arthroscopy has been proposed, then a CT scan with 3D reconstructions can be very helpful in planning the surgical correction. This allows for a more accurate assessment of the osseous anatomy of the hip [37, 38]. Axial cuts through the distal femoral condyles are made to allow for accurate measurement of the overall femoral version, which is a vital piece of information needed for appropriate surgical treatment. The 3D reconstructions also allow for identification of cam lesions that may be missed on the multiple planes of the CT scan, as oftentimes the cam can be subtle and missed with standard CT-scan cuts. The 3D reconstructed views provide excellent visualization and detail of the AIIS. Overall, the 3D-reconstructed CT scan is the most vital imaging study used in formulating an arthroscopic plan, as it allows for an accurate identification of impingement lesions, bony resection planes, and age in overall capsular and soft tissue management intraoperatively.

35.3 Treatment Strategy

Treatment of acetabular labral tears normally starts with an effort to identify and modulate offending activities and may include a course of nonoperative trial of physical therapy, anti-inflammatory medications, and, oftentimes, an injection into the femoroacetabular joint. Joint injections are done using ultrasound guidance in the office but can also be done using fluoroscopic guidance [39]. Physical therapy should focus on core-muscle strengthening, correction of abnormal muscle firing patterns, and abductor strengthening to provide stability to the hip joint in cases of capsular laxity. Oftentimes, athletes will elect nonoperative treatment in an attempt to "play out their season" and elect for operative treatment in the off-season.

Operative treatment consists of arthroscopic or open correction of the impingement process with labral refixation or reconstruction. Several studies have shown that the results of labral repair are superior to simple labral debridement [40–42]. In the event that labral repair is not viable, labral reconstruction has been proposed. Currently, reconstruction is most clearly indicated for symptoms associated with a deficient labrum, often associated with previous surgical debridement [43]. Keep in mind that there may often be

multiple pain generators, and it can be difficult to ascribe symptoms solely to a labral deficiency. Although there were limitations of their methodology, Matsuda and Domb have reported results of labral reconstruction similar to those of labral repair and outperforming those of simple labral debridement [44, 45]. Open treatment consists of surgical dislocation of the hip, which was developed by Ganz and has been used by others to address labral pathology [42, 46–49]. Our current preferred method is to address labral pathology utilizing arthroscopic treatment in an outpatient setting with the patient in the supine position [50, 51].

35.3.1 Labral Debridement

Labrectomy is infrequently necessary. Even comminuted and degenerative tears can demonstrate successful outcomes with restoration. However, occasionally, simple debridement may be deemed the best option for some patients: elderly patients, patients with limited capacity to comply with necessary postoperative precautions, and some tears with limited healing capacity. Debridement alone does not necessarily result in uniformly poor results [52]. However, debridement must be carried out in a thoughtful fashion, as there is compelling evidence that excessive labral removal can result in accelerated degenerative changes [42]. The goal of labral debridement is to remove the damaged tissue, preserve as much healthy labrum as possible, and create a stable transition zone, lessening the likelihood of persistent symptoms or propagating further tearing (Fig. 35.2) [53].

35.3.2 Surgical Technique for Arthroscopic Repair/Refixation

The majority of labral tears are amenable to restoration and preservation. Mostly commonly, labral tears requiring treatment are seen in conjunction with pincer impingement and require concomitant acetabuloplasty. Thus, the labrum is mobilized as necessary with sharp dissection, shavers, and radio-frequency devices to expose the pincer lesion that must be resected. If the chondrolabral junction is intact, it is preferable to preserve its integrity, but adequate mobilization for proper correction of the pincer lesion takes precedent over the goal of preserving the chondrolabral junction.

Most labral tears begin anteriorly and extend laterally. Thus, most of the work is done with the arthroscope in the anterolateral position as a viewing portal and the anterior position as the working portal with a large-diameter disposable cannula (Fig. 35.3) [51, 53]. The anchors are placed on the capsular side of the labrum as close as possible to the articular rim but always being careful to avoid perforating the articular surface. If the anchors are placed too far away from the rim, it does not restore the labral anatomy, and it is unlikely that it is restoring its function. The anchors are placed and tied starting from medial to lateral.

Most anchors are placed from a percutaneous distally based site which easily allows placement from the 3 o'clock position anteriorly to well posterior to the 12 o'clock position laterally on a right hip [54]. Occasionally, a far medial anchor is placed from the anterior portal, giving a better direction to avoid the anchor perforating the medial pelvic cortex, possibly irritating the iliopsoas tendon.

When drilling, the articular surface of the acetabulum is carefully observed. Any signs of articular motion with the drilling process indicate that it could be violating the subchondral surface and necessitates redrilling further away. Anchors are spaced at 8–10 mm intervals for adequate security of the restored labrum.

Less commonly, for tears extending well posterior to the 12 o'clock position, the arthroscope can be switched to the anterior portal, and the anchors can be placed from the anterolateral portal. In this position, fluoroscopy helps to assure that, with drilling, there is adequate diversion from the articular surface. Fluoroscopy does not help for anteriorly based anchors because the direction of placement is more in the plane of the x-ray beam (Fig. 35.4).

Anchor placement is consistent from one case to the next, but suture management is variable and dictated by the size, morphology, and pattern of labral tearing. If the chondrolabral junction is intact, a simple suture can be used through the lateral margin of the labrum, rolling it up against the rim and recreating the labral structure (Fig. 35.5). For a large labrum, or one in which the chondrolabral junction has been disrupted, a modified single-limb mattress suture creating a labral base fixation can restore the chondrolabral junction and the normal anatomy of the labrum (Fig. 35.6) [55]. For a small labrum, or one in which the quality of the labral tissue is poor, a simple loop suture may be necessary in order to restore adequate labral tissue to the acetabular rim (Fig. 35.7).

35.3.3 Primary Labral Repair

Occasionally, a labral tear may be present without accompanying pincer impingement [56]. In this circumstance, no formal takedown of the labrum to correct the pincer lesion is necessary. It is just a matter of repairing the torn labrum. In this circumstance, a primary repair is often performed placing the anchor against the rim on the articular side of the labrum. The rim must still be first freshened with a burr or bonecutter blade, preparing a raw bony surface to potentiate healing. Both limbs of the suture can be passed through the labrum in a mattress fashion, keeping the knot on the capsular side and avoiding suture interposed between the labrum and the articular surface of the femoral head (Fig. 35.8). If the labrum is hypoplastic or the quality of the labral tissue marginal, a simple loop suture may still be necessary to fix

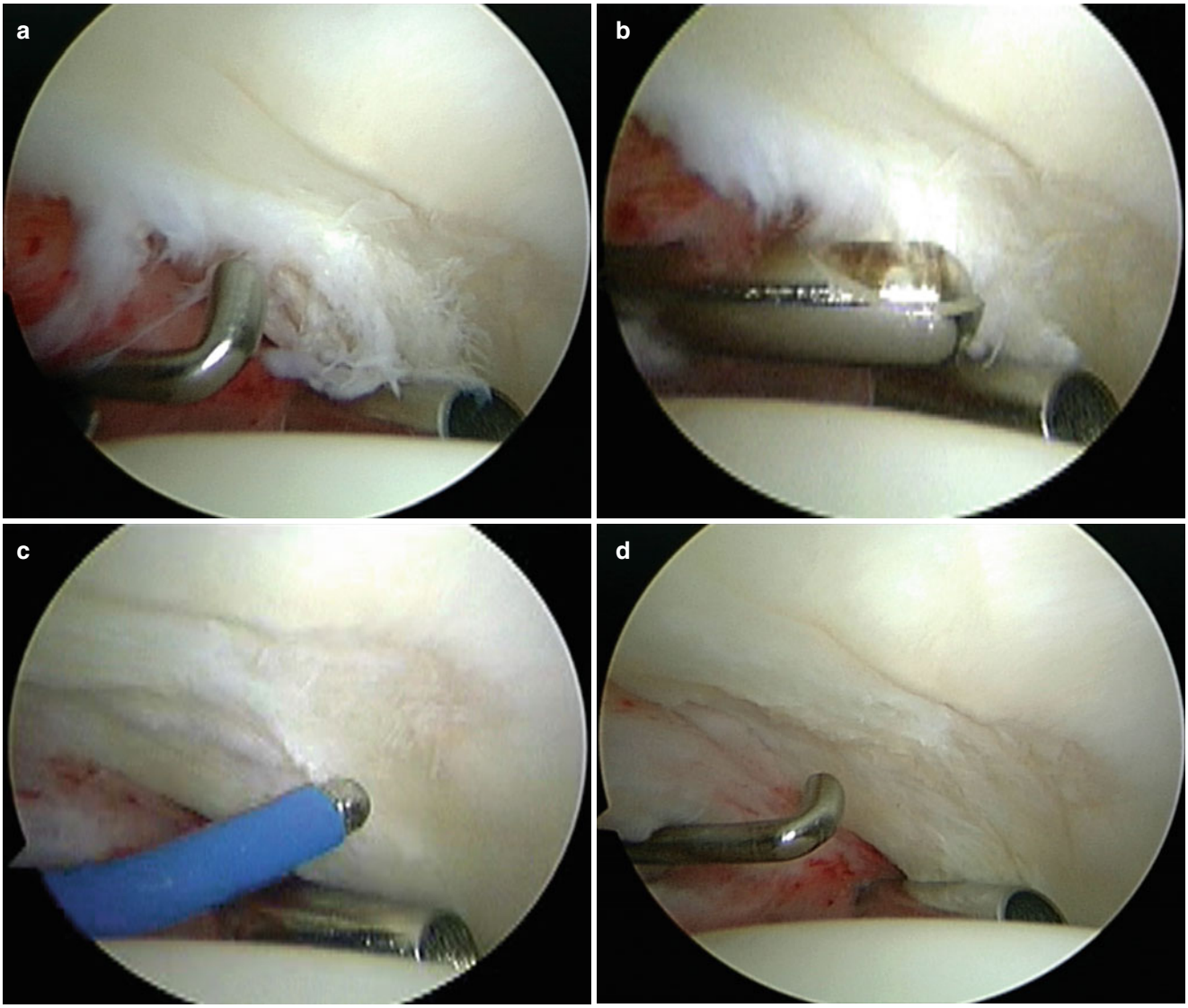


Fig. 35.2 Arthroscopic view of a right hip from the anterior portal. (**a**) A fragmented labral tear with degeneration within its substance is identified. (**b**) Debridement is initiated with the power shaver. (**c**) A portion of the comminuted labral tear is conservatively stabilized with a radiofrequency probe. (**d**) The damaged portion has been removed, preserving the healthy substance of the labrum (Reprinted with permission J. W. Thomas Byrd, MD)

the tissue (Fig. 35.9). A quest must always be made to try to ascertain and address the etiology of the labral tear.

35.3.4 Traumatic Posterior Bankart-Type Labral Tear

Posterior labral detachment may be associated with recurrent instability caused by traumatic posterior subluxation or dislocation [57]. These posterior labral tears are usually amenable to repair from the posterolateral portal. The portal position is well suited for anchor placement in the posterior rim, and simple loop sutures can restore the posterior labrum to the rim (Fig. 35.10). Sports-related traumatic posterior instability is often associated with underlying FAI that creates a fulcrum, predisposing the joint to posterior subluxation [58]. When evidence of damage due to FAI is also present, it may need to be corrected in the same setting.

35.4 Rehabilitation and Return to Play

Postoperatively, patients follow a specific rehabilitation protocol that maximizes joint mobilization and functional recovery while minimizing labral stress to allow for appropriate healing [59]. Philippon has shown that labral healing to the acetabular bone takes approximately 12 weeks [60]. Thus, any exercises that excessively stress that labrum should

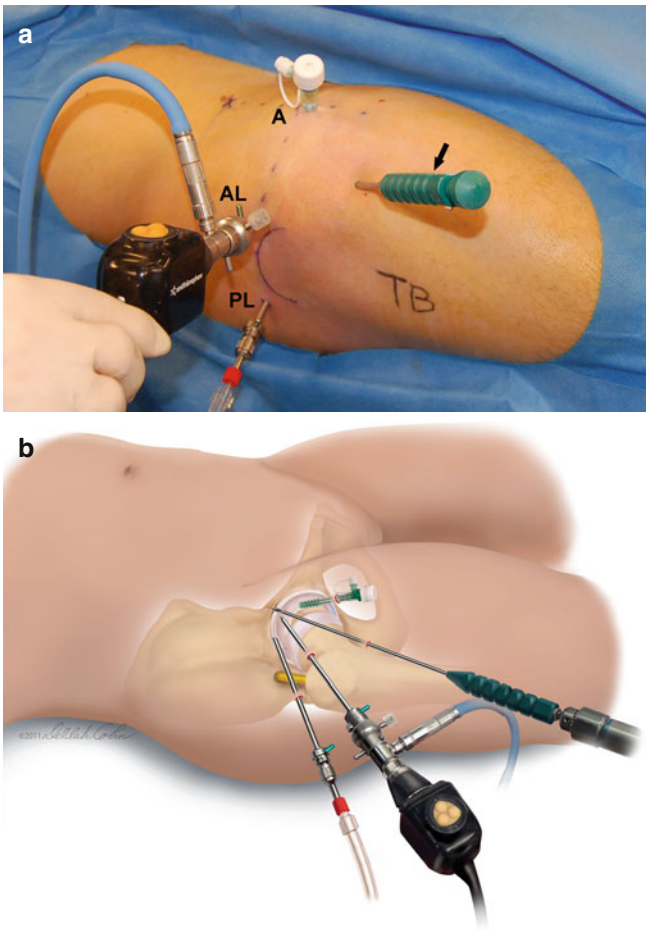


Fig. 35.3 (**a**) For this right hip, three standard portals are utilized for routine arthroscopy, including the anterior (*A*), anterolateral (*AL*), and posterolateral (*PL*). A large-diameter disposable cannula has been placed anteriorly for suture management. The anchor delivery system (*arrow*) has been placed distally midway between the anterior and anterolateral portals. (**b**) Schematic illustrates the drill sleeve placed against the acetabular rim (Reprinted with permission J. W. Thomas Byrd, MD)

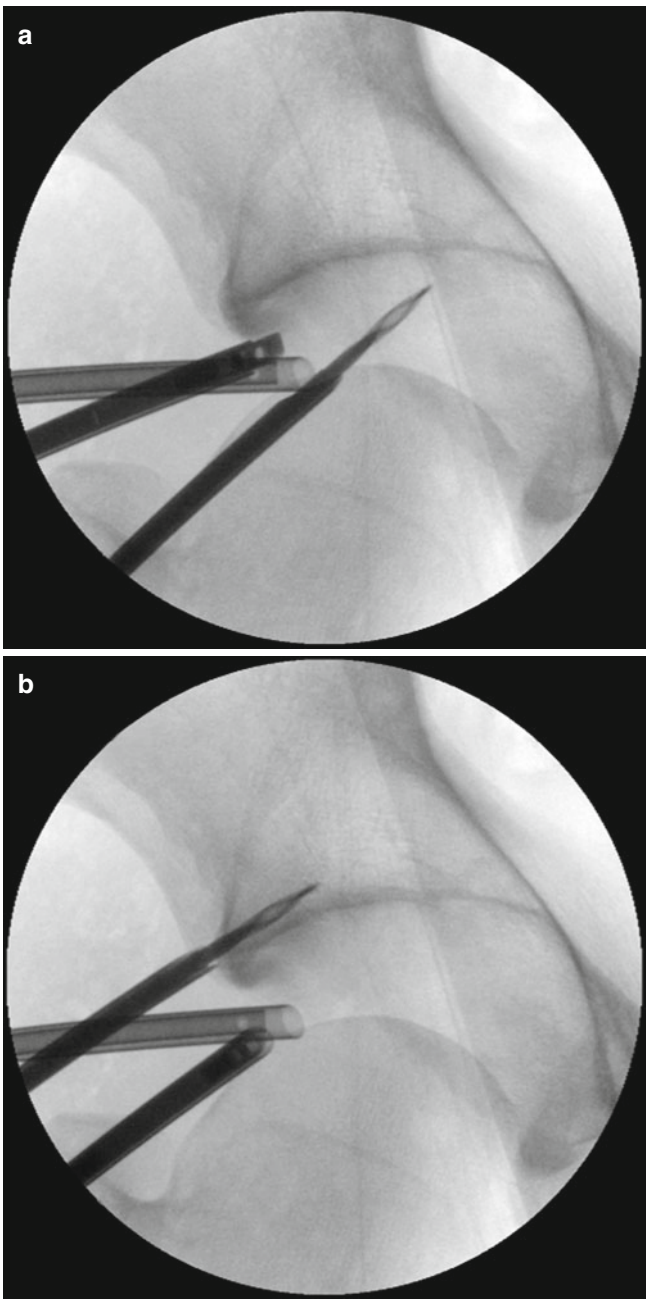


Fig. 35.4 (**a**) Fluoroscopic image of a right hip as drilling is performed for an anteriorly based anchor. Since the direction of placement is partly in the plane of the image, it is not helpful for assuring proper placement. (**b**) AP fluoroscopic image of a right hip with the anchor drill hole being placed laterally. Since this is perpendicular to the x-ray beam, the image is helpful in assuring that the anchor will diverge from the articular surface (Reprinted with permission J. W. Thomas Byrd, MD)

be avoided for the first 3 months postoperatively. Generally, patients are kept 50 % partial weight bearing for 4 weeks. During this period, external rotation and extremes of flexion are avoided to protect the labral repair. If a capsular closure is performed, extension beyond neutral is also avoided. In cases of microfracture, strict protected weight bearing is maintained for 8 weeks. Oscillatory joint mobilizations are combined with longitudinal traction to minimize labral stress and prevent joint adhesions. The gluteus medius muscle almost always experiences early postoperative inhibition as a result of the muscle penetration from portal placement. It is imperative to establish early return of abductor muscle function and normal firing patterns to allow for joint stabilization and restoration of a normal gait pattern. At approximately 6 weeks post-op, therapy begins to focus more on core strengthening, regaining proprioceptive control, and to progress range of motion and strengthening. By approximately 12 weeks post-op, the labrum should be healed, and sports-specific training and advanced plyometrics are begun. Aggressive aquatic training is begun as well. Ultimately, the decision to return an athlete is case dependent, but in general athletes must have full painless hip motion, hip strength

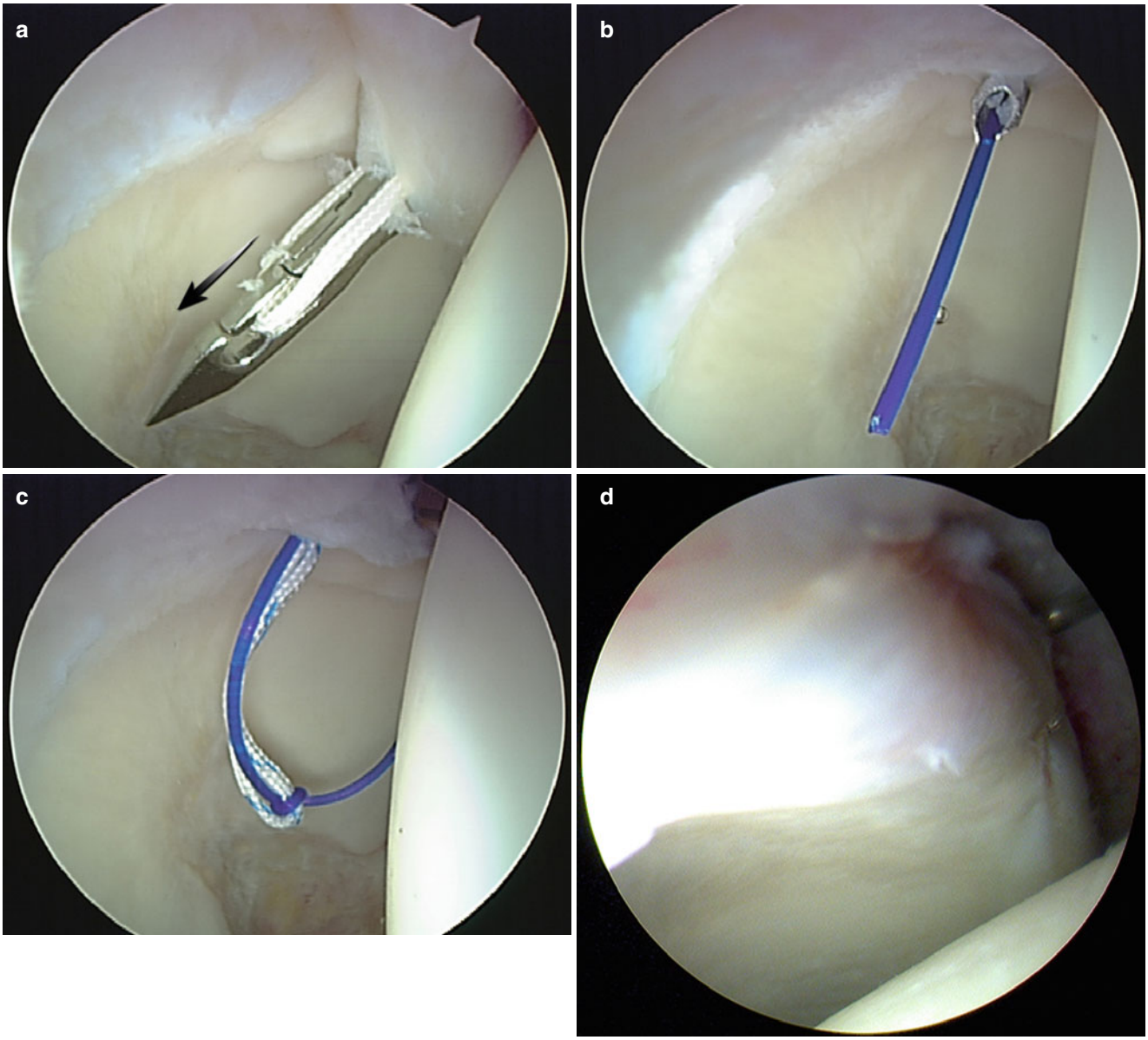


Fig. 35.5 Arthroscopic view of a right hip. Acetabuloplasty has been completed, and the anchor has been seated in the anterior acetabulum. The chondrolabral junction is preserved. (**a**) A soft tissue-penetrating device is used to push the suture limb through the labrum. (**b**) As an alternative method, a suture-passing device is placed to introduce a monofilament suture. (**c**) The braided anchor suture is then shuttled through the labrum, secured to the monofilament with a single half-hitch. (**d**) Three anchors have been placed with sutures tied, reapproximating the labrum to the rim of the acetabulum (Reprinted with permission J. W. Thomas Byrd, MD)

equal to the contralateral side, and the ability to perform sports-specific drills at full speed without pain. However, a complete guide to rehabilitation and return to play criteria is beyond the scope of this chapter. For the rare posterior labral repair associated with traumatic instability, a hip spica brace limiting flexion is used for 6 weeks with posteriorly directed forces avoided for 12 weeks.

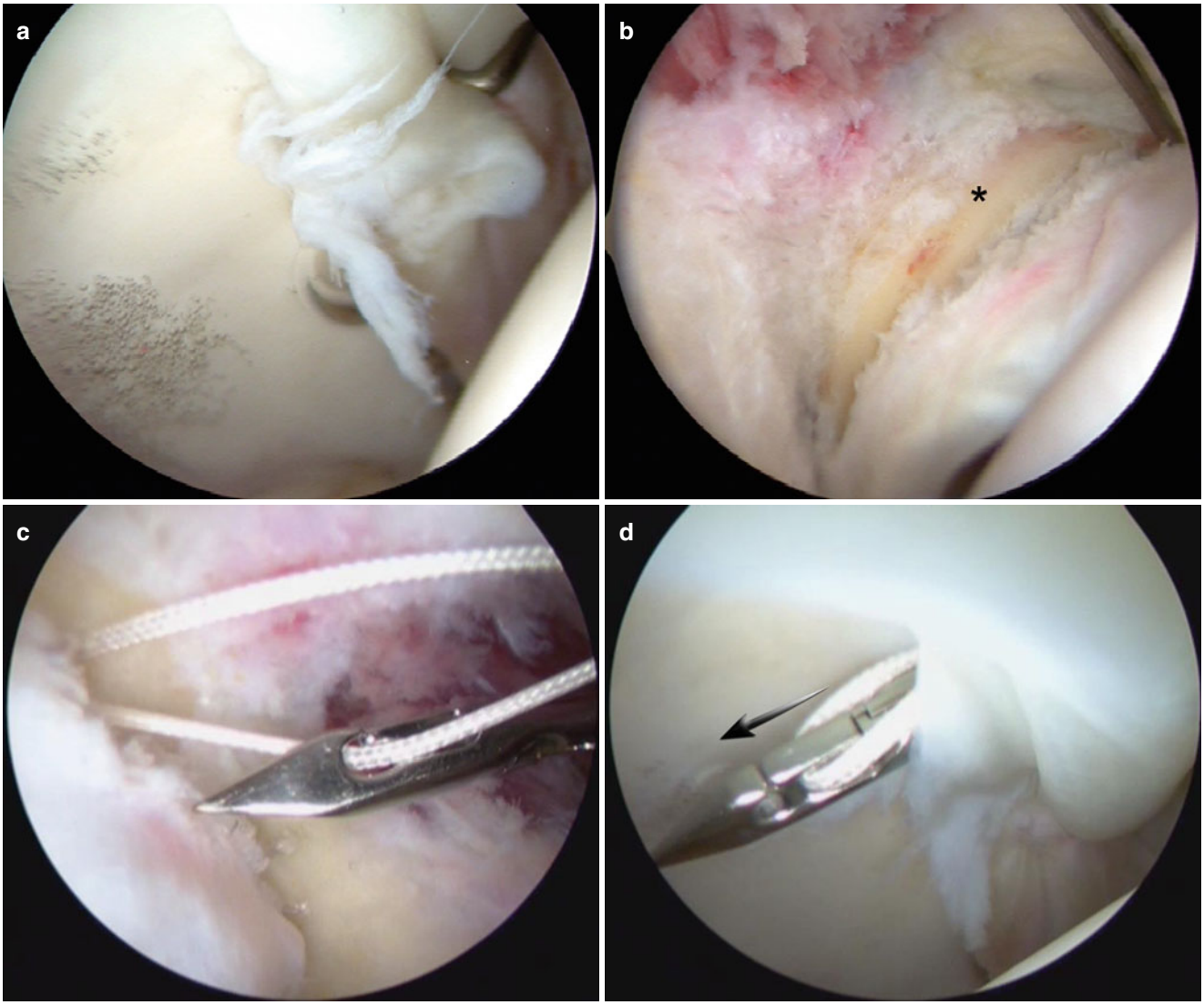


Fig. 35.6 Arthroscopic view of a right hip from the anterolateral portal. (**a**) The labrum is robust with disruption of the chondrolabral junction. (**b**) Viewing peripheral to the labrum, the acetabuloplasty (*asterisk*) has been completed. (**c**) A suture anchor has been seated in the bony rim, and one limb of the suture is grasped with a soft tissue-penetrating device. (**d**) With the penetrator, the suture has been passed into the joint at the chondrolabral junction. (**e**) The penetrator has been repositioned through the midsubstance of the labrum, preparing to grasp the suture limb. (**f**) The suture has been grasped and is withdrawn back out to the capsular rim. (**g**) Three anchors have been placed with sutures tied, restoring the labrum and the chondrolabral junction. (**h**) Labral restoration is further observed peripherally with reconstitution of the labral seal (Reprinted with permission J. W. Thomas Byrd, MD)

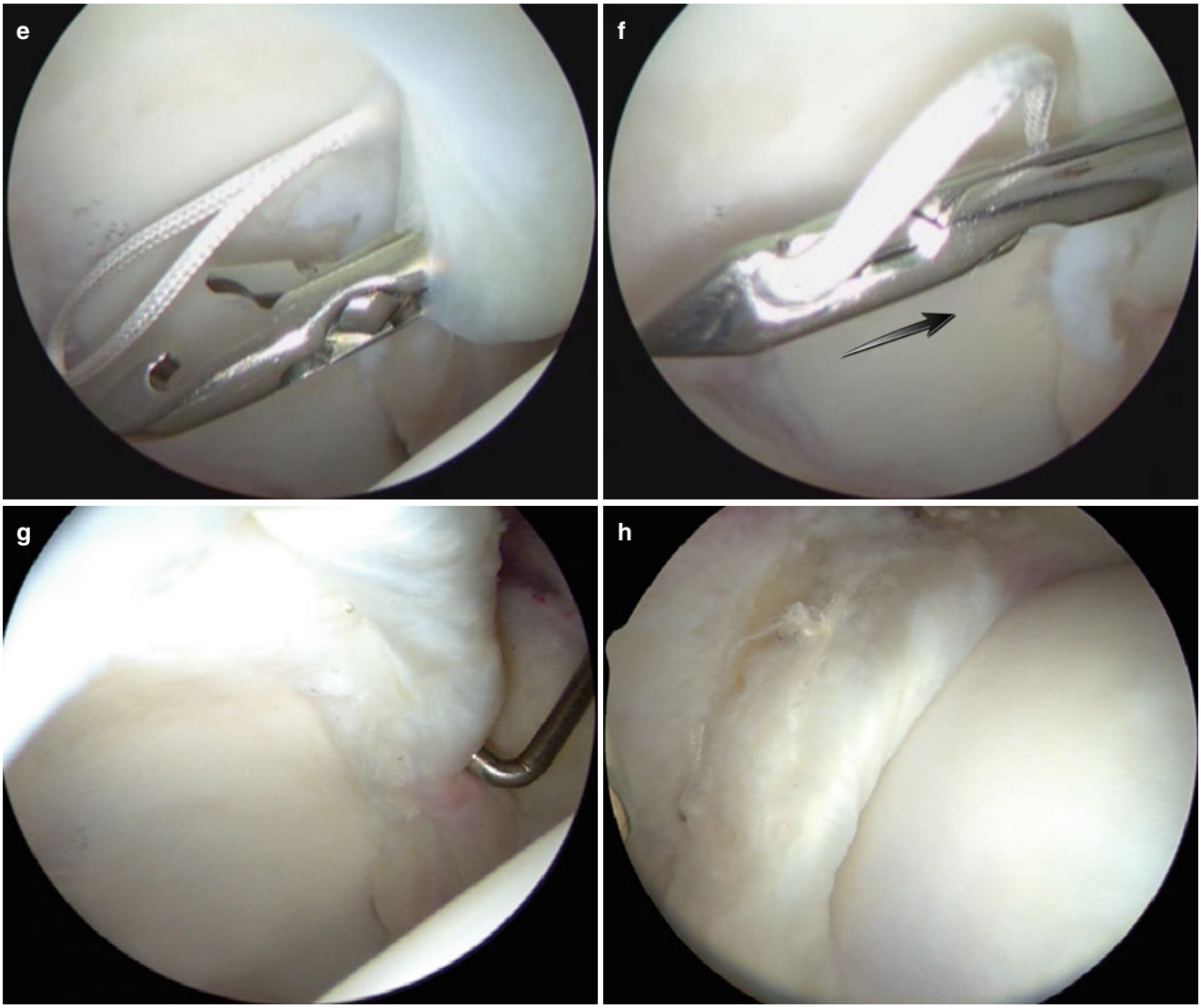


Fig. 35.6 (continued)

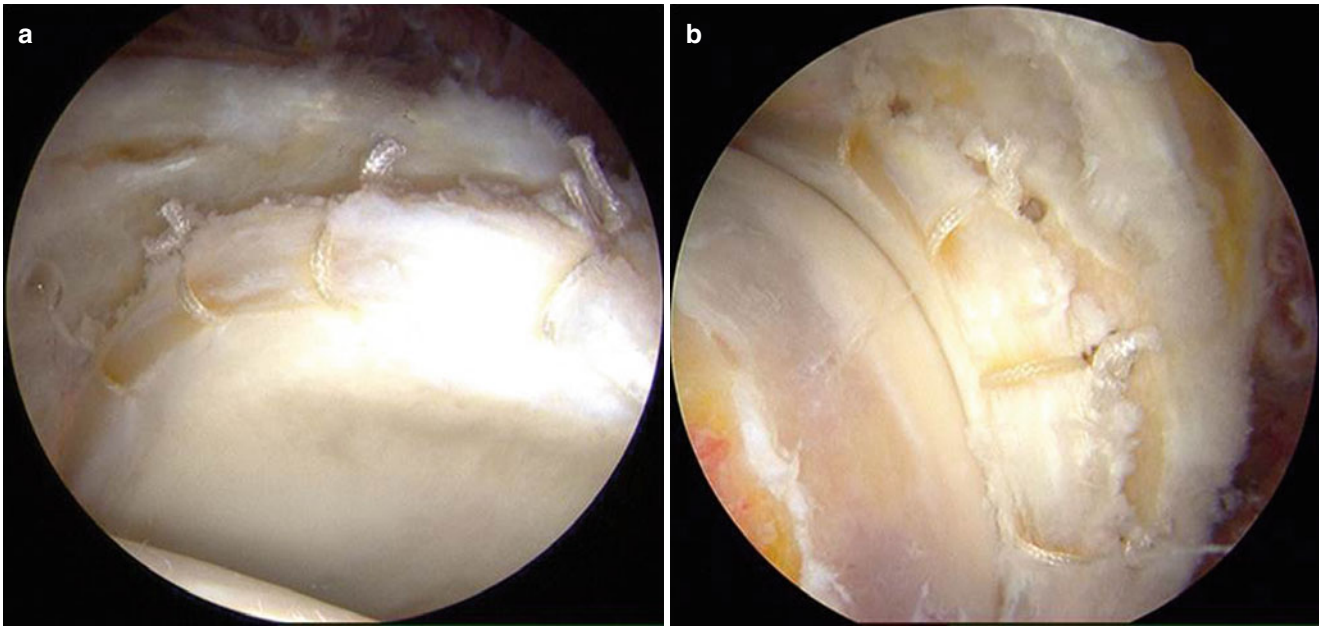


Fig. 35.7 Viewing a left hip from the anterolateral portal, an acetabuloplasty has been completed in the presence of this partially ossified labrum. (**a**) The diminutive remnant has been properly restored to the rim with multiple simple loop sutures. (**b**) With the joint reduced, the labrum is visualized in articulation with the femoral head (Reprinted with permission J. W. Thomas Byrd, MD)

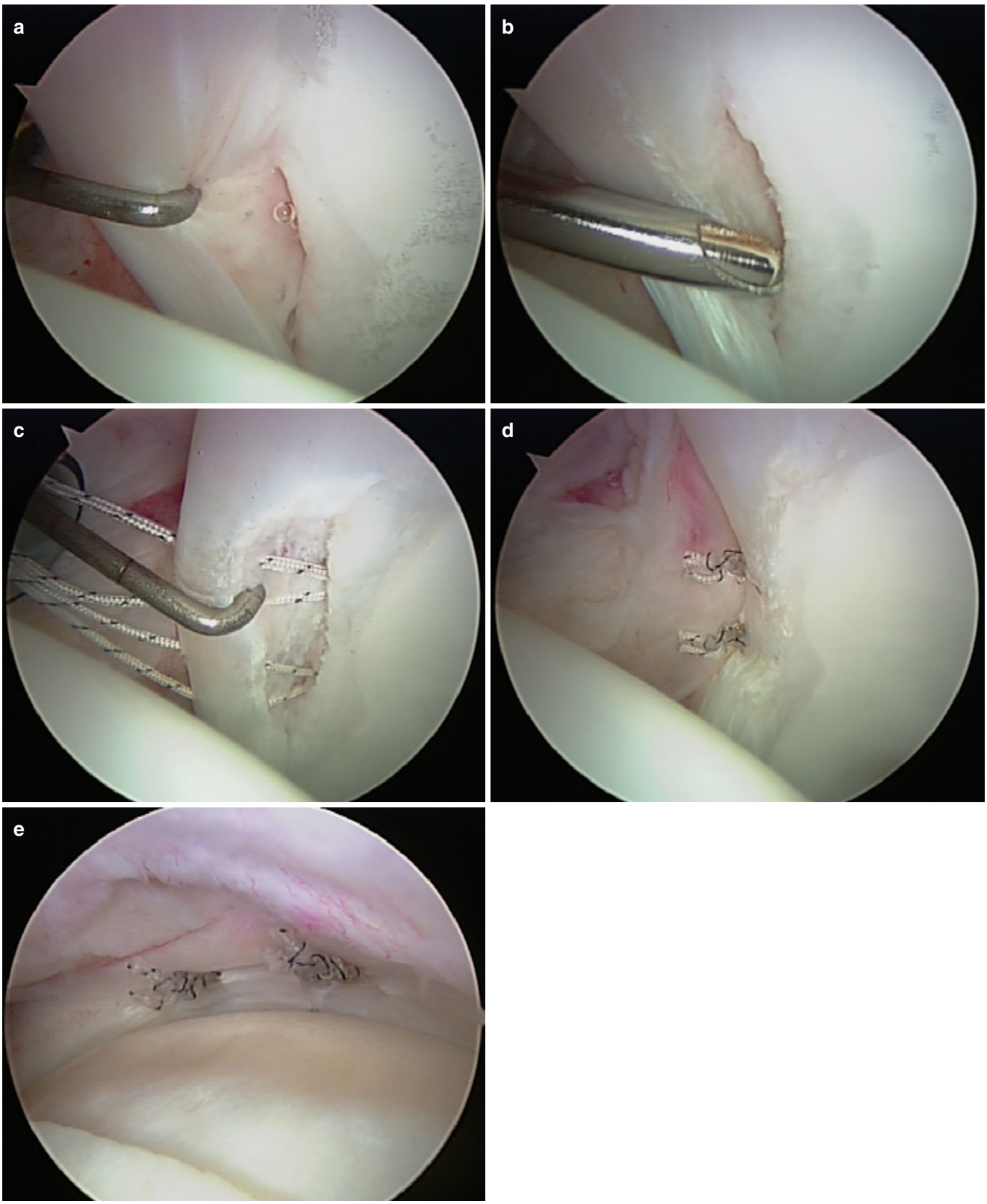


Fig. 35.8 An anterior labral tear of a right hip is being viewed from the anterolateral portal. (**a**) Pathological detachment of the labrum from the rim of the acetabulum is being probed. (**b**) Freshening the rim of the acetabulum, creating a bleeding bony surface aids in potentiating healing of the repair. (**c**) Two anchors have been placed in the rim of the acetabulum with the sutures passed through the labrum in a mattress fashion. (**d**) The sutures have been tied securely reapproximating the labrum to the rim of the acetabulum. (**e**) Now viewing from the peripheral compartment, the repair is inspected showing approximation of the labrum against the femoral head with the sutures well removed from the articular surface (Reprinted with permission J. W. Thomas Byrd, MD)

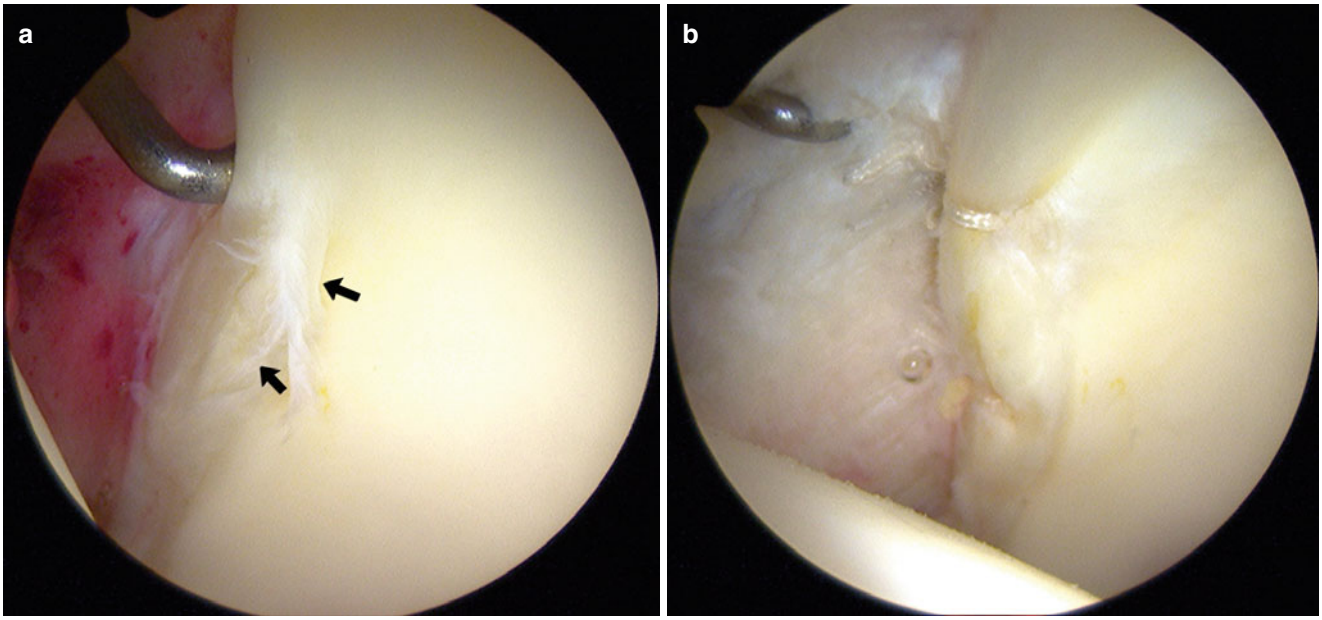


Fig. 35.9 A left hip viewing anteriorly from the anterolateral portal. (**a**) A diminutive tear of the anterior labrum (*arrows*) is identified. (**b**) A simple loop suture technique has been used to restore the labrum to the acetabular rim. This is necessary in order to have adequate tissue composition for the repair (Reprinted with permission J. W. Thomas Byrd, MD)

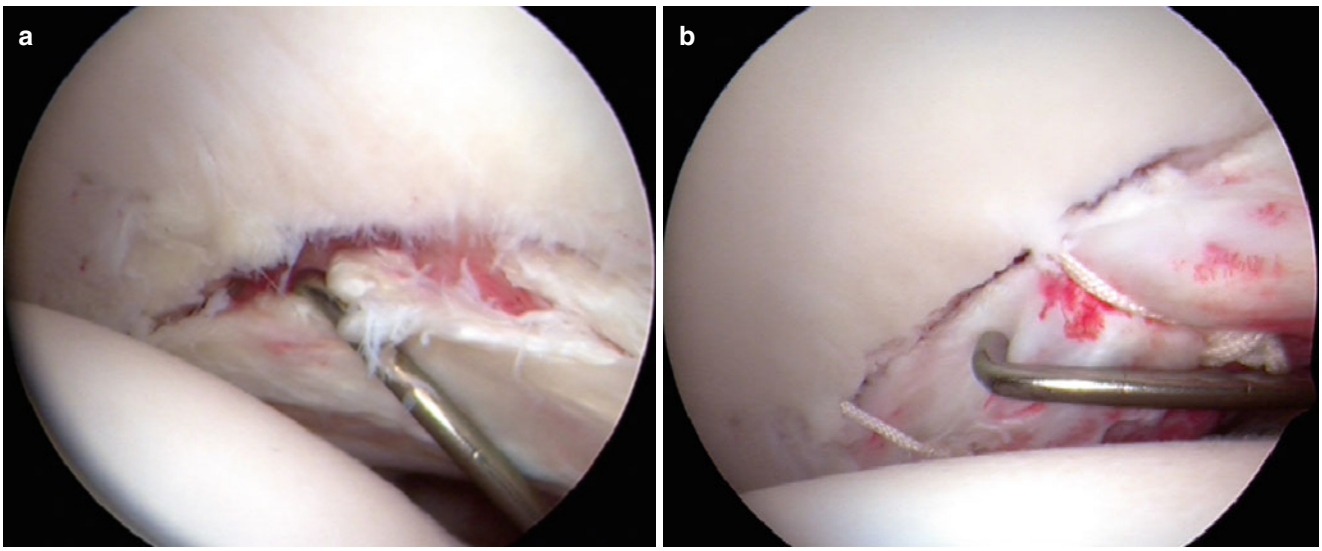


Fig. 35.10 Viewing posteriorly in this left hip in this athlete with recurrent posterior instability. (**a**) A traumatic detachment of the posterior labrum is being probed. (**b**) An arthroscopic Bankart-type repair has been performed with suture anchors (Reprinted with permission J. W. Thomas Byrd, MD)

References

1. Byrd JWT (1996) Labral lesions, an elusive source of hip pain: case reports and review of the literature. Arthroscopy 12(5):603–612
2. Ganz R, Parvizi J, Beck M et al (2003) Femoroacetabular impingement: a cause for osteoarthritis of the hip. Clin Orthop Relat Res 417:112–120
3. Wenger DE, Kendell KR, Miner MR et al (2004) Acetabular labral tears rarely occur in the absence of bony abnormalities. Clin Orthop Relat Res 426:145–150
4. Philippon MJ, Kuppersmith DA, Wolff AB, Briggs KK (2009) Arthroscopic findings following traumatic hip dislocation in 14 professional athletes. Arthroscopy 25(2):169–174
5. Larson CM, Kelly BT, Stone RM (2011) Making a case for anterior inferior iliac spine/subspine hip impingement: three representative case reports and proposed concept. Arthroscopy 27(12): 1732–1737
6. Tonnis D, Heinecke A (1999) Acetabular and femoral anteversion: relationship with osteoarthritis of the hip. J Bone Joint Surg Am 81(12):1747–1770
7. Domb BG, Shindle MK, MacArthur B et al (2011) Iliopsoas impingement: a newly identified cause of labral pathology of the hip. HSS J 7(2):145–150
8. Wiberg G (1939) Studies on dysplastic acetabula and congenital subluxation of the hip joint. Acta Chir Scand 83(Suppl 58):7
9. Lequesne MG, Laredo JD (1998) The faux profil (oblique view) of the hip in the standing position. Contribution to the evaluation of osteoarthritis of the adult hip. Ann Rheum Dis 57(11):676–681
10. Reimers J (1980) The stability of the hip in children. A radiological study of the results of muscle surgery in cerebral palsy. Acta Orthop Scand Suppl 184:1–100
11. McKibbin B (1970) Anatomical factors in the stability of the hip joint in the newborn. J Bone Joint Surg Br 52(1):148–159
12. Buller LT, Rosneck J, Monaco FM et al (2012) Relationship between proximal femoral and acetabular alignment in normal hip joints using 3D computed tomography. Am J Sports Med 40(2):367–375
13. Sutter R, Dietrich TJ, Zingg PO et al (2012) Femoral antetorsion: comparing asymptomatic volunteers and patients with femoroacetabular impingement. Radiology 263(2):475–483
14. Hammoud S, Bedi A, Magennis E et al (2012) High incidence of athletic pubalgia symptoms in professional athletes with symptomatic femoroacetabular impingement. Arthroscopy 28(10): 1388–1395
15. Taylor DC, Meyers WC, Moylan JA et al (1991) Abdominal musculature abnormalities as a cause of groin pain in athletes: inguinal hernias and pubalgia. Am J Sports Med 19:239–242
16. Meyers WC, Foley DP, Garrett WE et al (2000) Management of severe lower abdominal pain in high-performance athletes. Am J Sports Med 28(2):2–8
17. Ferguson SJ, Bryant JT, Ganz R et al (2000) The acetabular labrum seal: a poroelastic finite element model. Clin Biomech (Bristol, Avon) 15(6):463–468
18. Ferguson SJ, Bryant JT, Ganz R et al (2003) An in vitro investigation of the acetabular labral seal in hip joint. J Biomech 36(2):171–178
19. Philippon MJ, Zehms CT, Briggs KK et al (2007) Hip instability in the athlete. Oper Tech Sports Med 15:189–194
20. Byrd JWT (2006) Adhesive capsulitis of the hip. Arthroscopy 22(1):89–94
21. Klaue K, Durnin CW, Ganz R (1991) The acetabular rim syndrome. A clinical presentation of dysplasia of the hip. J Bone Joint Surg Br 73:423–429
22. Safran MR, Giordano G, Lindsey DP (2011) Strains across the acetabular labrum during hip motion: a cadaveric model. Am J Sports Med 39:92S–102S
23. Martin HD, Kelly BT, Leunig M et al (2010) The pattern and technique in the clinical evaluation of the adult hip: the common physical examination tests of hip specialists. Arthroscopy 26(2):161–172
24. Robertson WJ, Gardner MJ, Barker JU et al (2008) Anatomy and dimensions of the gluteus medius insertion. Arthroscopy 24(2): 130–136
25. Tannast M, Siebenrock KA, Anderson SE (2007) Femoroacetabular impingement: radiographic diagnosis. What the radiologist should know. Am J Roentgenol 188:1540–1552
26. Clohisy JC, Nunley RM, Otto RJ et al (2007) The frog-leg lateral radiograph accurately visualized hip cam abnormalities. Clin Orthop Relat Res 462:115–121
27. Lequesne M, De Seze S (1961) False profile of the pelvis: a new radiographic incidence for the study of the hip. Its use in dysplasias and different coxopathies. Rev Rhum Mal Osteoartic 12:643–651
28. Tannast M, Siebenbrock KA (2009) Conventional radiographs to assess femoroacetabular impingement. Instr Course Lect 58:203–212
29. Henebry A, Gaskill T (2013) The effect of pelvic tilt on radiographic markers of acetabular coverage. Am J Sports Med 41(11):2599–2603
30. Eijer H, Leunig M, Mahomed MN et al (2001) Cross table lateral radiograph for screening of anterior femoral head-neck offset in patients with femoro-acetabular impingement. Hip Int 11:37–41
31. Gosvig KK, Jacobsen S, Palm H et al (2007) A new radiological index for assessing asphericity of the femoral head in cam impingement. J Bone Joint Surg Br 89:1309–1316
32. Amar E, Druckmann I, Flusser G et al (2013) The anterior inferior iliac spine: size, position, location. An anthropometric and sex survey. Arthroscopy 29(5):874–881
33. Byrd JWT, Jones KS (2004) Diagnostic accuracy of clinical assessment, MRI, gadolinium MRI, and intraarticular injection in hip arthroscopy patients. Am J Sports Med 32(7):1668–1674
34. Tian CY, Wang JQ, Zheng ZZ et al (2014) 3.0 T conventional hip MR and hip MR arthrography for the acetabular labral tears confirmed by arthroscopy. Eur J Radiol 83:1822–1827
35. Byrd JWT (2013) Patient selection and physical examination. In: Byrd JWT (ed) Operative hip arthroscopy, 3rd edn. Springer, New York, pp 7–32
36. Reurnik G, Jansen SP, Bisselink JM et al (2012) Reliability and validity of diagnosing acetabular labral lesions with magnetic resonance arthrography. J Bone Joint Surg Am 94(18):1643–1648
37. Dolan MM, Heyworth BE, Bedi A et al (2011) CT reveals a high incidence of osseous abnormalities in hips with labral tears. Clin Orthop Relat Res 469:831–838
38. Zaltz I, Kelly BT, Hestroni I et al (2013) The crossover sign overestimates acetabular retroversion. Clin Orthop Relat Res 47(8):2463–2470
39. Byrd JW, Potts EA, Allison RK et al (2014) Ultrasound-guided hip injections: a comparative study with fluoroscopy guided injections. Arthroscopy 30(1):42–46
40. Larson CM, Giveans MR, Stone RM (2012) Arthroscopic debridement versus refixation of the acetabular labrum associated with femoroacetabular impingement: mean 3.5-year follow-up. Am J Sports Med 40(5):1015–1021
41. Krych AJ, Thompson M, Knutson Z et al (2013) Arthroscopic labral repair versus selective labral debridement in female patients with femoroacetabular impingement: a prospective randomized study. Arthroscopy 29(1):46–53
42. Espinosa N, Rothenfluh DA, Beck M et al (2006) Treatment of femoro-acetabular impingement: preliminary results of labral refixation. J Bone Joint Surg Am 88(5):925–935
43. Philippon MJ, Wolff AB, Briggs KK et al (2010) Acetabular rim reduction for the treatment of femoroacetabular impingement correlates with preoperative and postoperative center-edge angle. Arthroscopy 26(6):757–761

44. Matsuda D, Burchette RJ (2013) Arthroscopic hip labral reconstruction with a gracilis autograft versus labral refixation: 2-year minimum outcomes. Am J Sports Med 41(5):980–987
45. Domb BG, El Bitar YF, Stake CE et al (2014) Arthroscopic labral reconstruction is superior to segmental resection for irreparable labral tears in the hip: a matched-pair controlled study with minimum 2-year follow-up. Am J Sports Med 42(1):122–130
46. Peters CL, Erickson JA (2006) Treatment of femoro-acetabular impingement with surgical dislocation and débridement in young adults. J Bone Joint Surg Am 88(8):1735–1741
47. Ganz R, Gill TJ, Gautier E et al (2001) Surgical dislocation of the adult hip a technique with full access to the femoral head and acetabulum without the risk of avascular necrosis. J Bone Joint Surg Br 83(8):1119–1124
48. Naal FD, Schar M, Miozzari HH et al (2014) Sports and activity levels after open surgical treatment of femoroacetabular impingement. Am J Sports Med 42(7):1690–1695
49. Naal FD, Miozzari HH, Wyss TF et al (2011) Surgical hip dislocation for the treatment of femoroacetabular impingement in high-level athletes. Am J Sports Med 39(3):544–550
50. Byrd JW (1994) Hip arthroscopy in the supine position. Arthroscopy 10(3):275–280
51. Byrd JWT (2013) Routine arthroscopy and access: central and peripheral compartments, iliopsoas bursa, peritrochanteric, and subgluteal spaces. In: Byrd JWT (ed) Operative hip arthroscopy, 3rd edn. Springer, New York, pp 131–160
52. Byrd JWT, Jones KS (2009) Hip arthroscopy for labral pathology: prospective analysis with 10-year follow-up. Arthroscopy 25(4):365–368
53. Byrd JWT (2013) Labral management: an overview. In: Byrd JWT (ed) Operative hip arthroscopy, 3rd edn. Springer, New York, pp 171–184
54. Byrd JWT (2013) Modified anterior portal for hip arthroscopy. Arthrosc Tech doi:10.1016/j.eats.2013.05.006 http://dx.doi.org/10.1016/j.eats.2013.05.006
55. Fry R, Domb B (2010) Labral base refixation in the hip: rationale and technique for an anatomic approach to labral repair. Arthroscopy 26(9):S81–S89
56. Byrd JWT, Jones KS (2014) Primary repair of the acetabular labrum. Arthroscopy 30(5):588–592
57. Byrd JWT, Maiers GP (2014) Traumatic instability: acute and delayed management. In: Nho SJ, Leunig M, Larson CM, Bedi A, Kelly BT (eds) Hip arthroscopy and hip joint preservation surgery. Springer, New York, pp 961–970
58. Krych AJ, Thompson M, Larson CM, Byrd JW, Kelly BT (2012) Is posterior hip instability associated with cam and pincer deformity? Clin Orthop Relat Res 470(12):3390–3397
59. Coplen EM, Voight ML (2012) Rehabilitation of the hip. In: Byrd JWT (ed) Operative hip arthroscopy, 3rd edn. Springer, New York, pp 411–440
60. Philippon MJ, Arnoczky SP, Torrie A (2007) Arthroscopic repair of the acetabular labrum: a histologic assessment of healing in an ovine model. Arthroscopy 23(4):376–380

Return to Sport After Hip Arthroscopy

36

Stefano Respizzi, Lara Castagnetti,
Davide Pisoni, and Stefano Comi

36.1 Premise

The hip arthroscopy is a surgical technique in the rapidly changing and increasingly common treatment for many painful conditions of the hip in top-level athletes. There is growing evidence that demonstrates the utility of this technique in the treatment of numerous causes of coxalgia [1, 2], including femoro-acetabular impingement (FAI) and microfracture. This approach, certainly less invasive than traditional open surgery, was effective in facilitating a quick return to the sport by the athletes [3].

36.2 Directions of Rehabilitation

Acute traumatic events (subluxations, dislocations and also low-impact trauma at the level of the proximal femur and greater trochanter) as well as atraumatic femoral-acetabular conditions can lead to chondral damage in athletes [4].

While the prevalence of chondral defects of the knee appears to be higher among athletes than in the normal population, the prevalence of chondral defects at the hip has not yet been elucidated [5].

Pain syndromes of the hip susceptible to treatment with hip arthroscopy are not unique to athletes who practise contact sports (football, hockey, soccer) but also in athletes engaging in noncontact sports such as golf, baseball, running and tennis [5].

Return to competition is very high even in the presence of microfractures; elite athletes revert to the same level of competition as presurgery in variable percentages between 77 and 100 % depending on the studies [3–5].

S. Respizzi (✉) • L. Castagnetti
Department of Rehabilitation and Functional Recovery,
IRCCS Humanitas Research Hospital, Milan, Italy
e-mail: stefano.respizzi@humantias.it

D. Pisoni • S. Comi
Isokinetic Medical Group, Milan, Italy

In rare cases, when the integrity of the joint after surgery is uncertain, the return to the level of athletic performance prior to the injury may not be possible. The athlete will be forced to take up a different type of training/competition level or even to give up the activity practised. Establishing this early is important so that the actual goal reached in relation to the integrity of the joint can help minimise failure and frustration felt by the patient and the rehabilitation team [6].

The young age of the patient, combined with ambitions to return to competitive sports, raises the functional expectations and goals that the patient/athlete needs and wants to achieve. It is therefore essential that the protocol of a therapeutic programme starts immediately post-surgery and extends far beyond the design of a personalised rehabilitation project, aiming to achieve the highest functional level possible.

The return to the sport gives rise to an initial treatment setting which is based in the gym/pool for rehabilitation and should be started as early as possible. It's important to remember that the progression of rehabilitation has been divided into phases purely for educational purposes and are not considered as watertight compartments. The transition from one phase to another will not even take into account the deadlines but more so the satisfaction of functional criteria.

Keep in mind that the target of our rehabilitative intervention remains an elite athlete who is going through a period of inactivity which is difficult to endure both physically and mentally. Our project must therefore take into account this situation by proposing focus not only on the recovery of the hip but also the person as an athlete, bringing him/her to be committed both physically and emotionally, making it easier to return to the sport once the lesion has healed [7].

Once you have completed this preparatory programme and the patient has achieved good recovery of motion, muscular strength, flexibility and coordination, the athlete is ready to face the last period of rehabilitation that will allow him/her to return to sport and competition: the rehabilitation field.

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_36,

The setting will not be the gym but field "rehabilitation" that will have unique characteristics depending on the sport practised by the athlete (football field, tennis, etc.). Also in this phase, the athlete will be followed by the rehabilitation team, and the expertise of the physician and physiotherapist will be integrated with those of the trainer.

The times given in the literature for the beginning of this phase are variable since it is strongly influenced by the type of pathology underlying the arthroscopic procedure. In the case of microfractures, it is in fact necessary to have a period of no load or light weight for 4 to 6–8 weeks, which influences the progression of rehabilitation treatment. Even in the case of hip repair, a partial load tolerance for at least 2 weeks is recommended and a partial load of 3 weeks in the case of osteoplasty. The beginning of the rehabilitation on the field is generally between 12 and 16 weeks as a post-operative intervention in the most simple cases and from 20 to 28 in the more complex interventions [8]. Other authors begin, in the absence of complications, the rehabilitation of the field between 8 and 16 weeks post-surgery, by inserting a progressive manner in the activities of sport starting from 12 weeks [9]. In an experimental study, a sheep model has demonstrated healing of the glenoid labrum, repaired arthroscopically, 12 weeks after the formation of scar fibrovascular or new bone formation [10].

The extreme variability of the information makes it, therefore, essential to use functional criteria rather than time to decide on the beginning of rehabilitation treatment on the field and the return to competition.

The return to the pitch although a field of rehabilitation is important from the emotional point of view [11]; on one hand positive feeling for an imminent return to team and competition, but on the other hand the fear of a re-injury starts.

The rehabilitation field has the ultimate outcome and functional recovery of the athlete's return to sport. The rehabilitation field, such as that in the rehabilitation gym, should be guided by knowledge of the underlying injury but also by the objectives and physical skills necessary to the athlete.

The prevention of overload is a prerequisite for achieving the final objective, and the field is the place where you can more easily take that risk. This is because the patient is continuously subjected to exercise load.

Usually the occurrence of complications during rehabilitation on sports field depends on an erroneous adoption and progression of loads. The overload can affect the part of the body treated surgically but most often involves other areas.

Since the re-education on sports field coincides with the final phase of the rehabilitation programme, the prevention of complications start in the pool and gym, through the correct technique and full recovery of strength and proprioception, but especially after doing exercises in preparation for the race.

Even in the field, by adopting appropriate strategies, you can reduce the risk of overload. The first strategy is to make a proper warm up, including running mobility strength, and proprioception exercises. This is the first step to start session properly. You should also check the quantity, i.e. the proposed load.

Equally important is the recovery for neural plasticity and functionality of the proposed exercises. The type of footwear worn by the athlete is important as well as the type of turf on which the session is held.

But it is only in accordance with certain criteria that patients can begin rehabilitation on the sports field while minimising the risks:

- The subjects must have appropriate tone and tropism.
- A level of strength not less than 80 % of the operated limb compared to the healthy one.
- Ability to run for at least 20 min under aerobic conditions.

It is more difficult to recover control in the eccentric phase of movement, i.e. the action of deceleration. This ability is the first to be regained, through an introductory course, at the beginning of the rehabilitation project both in the water and in the gym. The same goes for the control of rotations. Being able to control vertical loads and rotations is important as they are present in most of the movements that occur in sports and especially in sports games. For these reasons, the rehabilitative physiotherapy field becomes crucial for the recovery of these capabilities.

36.3 Objectives to Be Achieved Through the Field Rehabilitation

36.3.1 Recovery of Functional Movement

The objective is to achieve and maintain good joint function of the patient's limb, safely and with confidence in the new environment and with new ways of working. The focus is also on developing the capacity of power, speed and agility. The patient begins to walk in a straight line, eventually bringing in large radius curves, which are then progressed and used in running exercises in the same way along with coordination exercises (using the rope during the race), decelerating exercises and exercises with progression to lateral sliding.

36.3.2 Recovery Sport-Specific Movements

The goal is to reintroduce sport-specific strength and to improve coordination; this phase also reintroduces sport-specific equipment (ball, bat, etc.). It's important to start with movements that are already known by the athlete and change one variable at a time. It is not advisable to experiment new

methods of running and unusual non-sport-specific distances. We can then use frequency, mode, intensity and sport-specific movements suitable for the athlete [7]. During the recovery of sport-specific movements, it is also important to focus on the movement that led to the injury; an athlete will never feel completely cured and able to return to competition until he/she manages to repeat the movement that caused the lesion several times, without pain [8].

36.3.3 Recovery of Fitness

The aerobic reconditioning is of utmost importance and is a prerequisite for the return of the athlete on the field. On the field, we continue the work started in the gym and gradually increase the intensity of aerobic activity [8]. It is a good idea to perform (or repeat) the test for identifying lactate aerobic (S2) and anaerobic (S4) threshold. Such metrics are useful for setting up a proper aerobic reconditioning programme and they represent a measure of verification of the results achieved [12]. The athlete may not resume training or compete if he/she is not completely reconditioned, in order to prevent another injury.

36.3.4 Prevention of Reinjury

A new injury is a dramatic event for the athlete returning back to competition. It's important that in addition to the recovery of strength and range of motion, the athlete has also to recover the correct proprioceptive and aerobic capacity used in critical moments during the match. Not surprisingly, the number of injuries increases at the end of the race times/match [13]. The programme should include exercises to work all planes of movement and all body segments, movements of static and dynamic posture and combined exercises with variable repetitions, in a controlled and uncontrolled environment [8]. In our opinion, the prevention of a new injury would be useful also to assess the kinematics of the sport-specific movements and the correction of any errors in the execution of the movements and misalignment that can facilitate the injury.

36.4 Return to Sport and Competition

It is not an easy decision to define a time for the athlete to return to his team sport. The timing of return to competition is variable but is usually between the 10–12 and 32 weeks [6, 14]. Few athletes who underwent a resection of the hip return to competition earlier than 8 weeks after surgery [6].

When can the athlete return to sport? When the athlete has achieved the highest functional level possible and he/she feels mentally/physically ready to return to competition.

36.5 Rehabilitation Field

Once the general objectives of re-education on the rehabilitation field are identified, how do you expect the programme to work?

First of all, it is good to point out that the recovery programme should follow the principle of progression, either for loading and for the difficulty of the exercises. In this regard, it is good that the same protocol is structured for progressive stages and that the next phase is dependent on the achievement of functional objectives provided for each phase.

36.5.1 First Phase

Functional Objectives
Restore the correct gait pattern and running, achieving a good range of motion.

In this phase, the focus is to improve running technique at different speeds and hip mobility using obstacles adjustable in heights (Figs. 36.1 and 36.2).

36.5.2 Second Phase

Functional Objectives
This phase include the cushioning ability of the vertical loads, achieving a good symmetry of the thrusts of the lower limbs in lateral movements and using sport-specific equipment with confidence.

Running with curvilinear trajectories (Fig. 36.3), skips and gaits (Fig. 36.4), supports mono- and bi-breech landing where care should be taken in the eccentric phase of the movement, lateral movements (Fig. 36.5) and progressions in line with the final deceleration and, above all, should be introduced for sport-specific skills.

36.5.3 Third Phase

Functional Objectives
Introduction of changes of direction (Fig. 36.6), achieving optimal confidence with sport-specific equipment and a perfect ability to cushion the load after a jump and rotation control.

We introduce jumps, if there are no contraindications by the surgeon, starting with bipedal stance and then moving on to monopodalic, starting with individual leaps and then progressing on to multi-jumps. With regard to the leaps, attention should be paid mainly on the landing stage, without worrying about the height or distance covered in that leap.

Fig. 36.1 Active mobilisation with obstacles

Fig. 36.2 Active mobilisation with obstacles: variant

Fig. 36.3 Running with curvilinear trajectories

Fig. 36.4 Skips and gaits

Fig. 36.5 Lateral movements

Fig. 36.6 Changes direction

36.5.4 Fourth Phase

Functional Objectives

Functional capacity to perform routes with changes of direction at maximum intensity and ability to perform sport-specific exercises with proper technique and high intensity.

The paths with changes of direction are made to the highest setting, and the mono- and bi-breech stance phases are finalised to pay maximum attention to the loading phase and the subsequent boost.

36.5.5 Fifth Phase

Functional Objectives

The functional ability to perform sport-specific movements and actions without fear.

We proposed phases of the game and sport-specific actions. Fuctional requirements should match sports requirements, such us for soccer players small sided games, game phases simulations and tackles. The stimulus provided must be comparable to those of the competition.

In this phase it is crucial to repeat the movement that caused the injury several times. This allows the athlete to fully regain self confidence and to be aware of having achieved a complete recovery, both functional and emotional.

Also from the point of view of metabolic stress, it would be appropriate to follow a progression of intensity, so, for example, in the first three phases, it should be solicited to aerobic metabolism through basic exercise working within the aerobic and anaerobic threshold. In the fourth phase, the goal is to stimulate anaerobic metabolism with lactate exercises, power and capacity, while the fifth phase calls for the anaerobic alactate metabolism through agility drills, skill and speed exercises.

Conclusions

The return to sports without restrictions after surgery in hip arthroscopy is possible.

To achieve the highest functional level possible, it becomes essential to carry out the last part of the rehabilitation on the sports field. On the field, in fact, you have the ability to provide functional and metabolic stimuli specific to the sport, which is useful to regain motor patterns, both basic and sport specific, while achieving an optimal state of mental and physical shape.

The revival of the movement that caused the injury is important from a psychological point of view because it gives the athlete the awareness of being fully healed. The progressive increase in the proposed stimulus, the proper adoption of the loads and the quality control of the movements are the criteria that allow you to work safely avoiding overloads.

In planning recovery activities on the rehabilitation field, it would be appropriate to establish the phases with specific functional goals, and once achieved, the athlete will be allowed to progress to the later stages.

In the next year the rehabilitation process and surgical techniques will be more efficient and individualised, with the goal to improve the quality of life for athletes with hip pathology.

References

1. Philippon MJ, Briggs KK (2007) Outcomes following hip arthroscopy with microfracture. Arthroscopy 23:211. doi:http://dx.doi.org/10.1016/j.arthro.2007.03.033
2. Bardakos NV, Vasconcelos JC, Villar RN (2008) Early outcome to hip arthroscopy for femoroacetabular impingement: the role of femoral osteoplasty in symptomatic improvement. J Bone Joint Surg Br 90:1570–1575
3. Singh PJ, O'Donnel JM (2009) The outcome of hip arthroscopy in Australian football league players: a review of 27 hips. Arthroscopy 26:743–749
4. Byrd JWT (2001) Lateral impact injury: a source of occult hip pathology. Clin Sports Med 20:801–815
5. McDonald JE, Herzog MM, Philippon MJ (2013) Return to play after hip arthroscopy with microfracture in elite athletes. Arthroscopy 29(2):330–335
6. Enseki KR, Martin RL, Draovitch P, Kelly BT, Philippon MJ, Schenker ML (2006) The hip joint: arthroscopy procedures and post-operative rehabilitation. J Orthop Sports Phys Ther 36(7):516–525
7. Wahoff M, Ryan M (2011) Rehabilitation after hip femoroacetabular impingement arthroscopy. Clin Sports Med 30:463–482
8. Edelstein J, Ranawat A, Enseki KR, Yun RJ, Draovitch P (2012) Post-operative guidelines following hip arthroscopy. Curr Rev Musculoskelet Med 5:15–23
9. Scott W, Cheatham PT (2012) Rehabilitation after hip arthroscopy and labral repair in a high school football athlete. Int J Sports Phys Ther 7(2):173–184
10. Philippoon MJ, Schroder e Souza BG, Briggs KK (2010) Labrum: resection, repair and reconstruction sports medicine and arthroscopy review. Sport Med Arthrosc 18(2):76–82
11. Tomaello L, Tencone F, Cilli P (2014) ilritorno allo sport dopo ricostruzione LCA. In: Manuale di riabilitazione ortopedica post chirurgica. CIC edizioni internazionali
12. Della Villa S, Tsapralis K, Salsi A (2010) Rehabilitation after hip arthroscopy. In: Zini R (ed) Hip arthroscopy. Argalia Editore, Urbino, pp 131–143
13. Ekstrand J, Hägglund M, Waldén M (2011) Injury incidence and injury patterns in professional football: the UEFA injury study. Br J Sports Med 45(7):553–558
14. Enseki KR, Martin R, Kelly BT (2010) Rehabilitation after arthroscopic decompression for femoroacetabular impingement. Clin Sports Med 29:247–255

Medial Meniscus

37

Ezio Adriani and Berardino Di Paola

37.1 Anatomy

In the axial plane, the medial meniscus has the shape of an open "C" with a larger surface at the posterior horn, whereas in the coronal plane, it is wedge shaped and becomes thinner towards the free edge [1]. Its average dimensions are as follows: circumference, 99.0 mm (range, 84–119; 9.3 SD); width of meniscal body, 9.3 mm (6.7–12.4; 1.3 SD); distance between anterior and posterior margin, 45.7 mm (30.1–56.1; 5.0 SD); and distance between the meniscal wall and medial margin of the tibial spines, 27.4 mm (23.3–32.7; 2.5 SD). The medial meniscus covers 64 % of the medial tibial plateau (range, 51–74 %) [2–4].

Knowledge of meniscal vascularity is crucial and will influence any assessment of the reparability of meniscal lesions. From early prenatal development until shortly after birth, the meniscus is completely vascular. At 10 years of age, vascularity is present in around 10–30 % of the meniscus, and in adults, blood vessels and nerves are present in only the peripheral areas (10–25 % of tissue) [2]. Three distinct regions can therefore be identified: an outer vascular/neural area (red-red zone), an intermediate area (red-white zone) and an inner avascular/aneural area (white-white zone) [5–7].

Similarly, innervation does not extend to the entire meniscus but follows the radial distribution of the vasculature and concentrates predominantly at the anterior and posterior horns and along the meniscal wall, reaching only the inner third of the body. The medial meniscus has been found to contain, in addition to nerve fibres with vasomotor functions, Pacinian corpuscles (proprioceptive function), slowly adapting Ruffini endings, and Golgi tendon organs (mechanoreceptors) whose presence suggests an important role for the menisci in transmitting information to the central nervous system [6, 8, 9].

From a structural standpoint, the collagen fibres making up the meniscus have circumferential orientation in the periphery (meniscal wall), whereas in the inner portion, they have a predominant radial arrangement running parallel to the joint surface. This organisation explains the different meniscal behaviour patterns: the central region is more suited to resisting compressive axial loads between the femur and tibia, whereas the peripheral region resists tensile forces [10]. The circumferential collagen fibres of the body continue with the anterior and posterior insertional ligaments (meniscal roots), by means of which the meniscus attaches to the subchondral bone of the tibia [7].

The medial meniscus has connections with the deep layer of the medial collateral ligament (MCL): the menisco-tibial and the menisco-femoral layer. Studies on knee laxity have shown that rupture of the MCL deep layer leads to increased external rotation of the tibia at 60–90° of knee flexion and that the ligament represents a secondary restraint to valgus. The MCL limits the movement of the medial meniscus: injury to the menisco-tibial layer will lead to increased meniscal mobility, with effects on the forces acting on the joint that have yet to be investigated in a satisfactory manner [11, 12].

37.2 Aetiology and Mechanism of Injury

The main distinction to be made in meniscal lesions is based on aetiology and divides lesions into traumatic and/or degenerative, which can be diagnosed on the basis of the patient's clinical history.

According to the American Academy of Orthopaedic Surgeons, the incidence of meniscal injuries in the United States is approximately 61/100,000 [13]. These injuries are particularly frequent in athletes participating in sports with high functional demands (soccer, basketball, baseball, skiing) [14–18].

E. Adriani, MD (✉) • B. Di Paola, MD
Sport Clinique, Mater Dei Clinic, via A. Bertoloni 34, Rome 00197, Italy
e-mail: ezioadriani@gmail.com; dipaoladino@yahoo.it

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_37,

Men are more prone to these injuries than women, with a male-to-female ratio between 2.5:1 and 4:1. As regards age, an analysis of arthroscopic knee procedures coded as simple or complex provides an incidence peak between 40 and 49 years and a second one between 50 and 59 years; if performed in conjunction with anterior cruciate ligament (ACL) reconstruction, the peak is seen in the 20–29-year age range [14, 17, 19, 20]. The mechanisms underlying meniscal lesions normally involve the development of a shear force secondary to twisting movements with the foot firmly planted and the knee in semiflexion. In other cases a meniscal tear is produced by hyperflexion or hyperextension movements as occurs, for example, when kicking the air [21].

The prevalence of meniscal injuries with associated ACL tear varies between 44 and 61 % [22–24]. In 1997, Bellabarba conducted a meta-analysis of models of meniscal injuries in knees with ACL tears. This demonstrated a predominance of lateral meniscal injuries (56 % versus 44 %), although the incidence of new medial meniscus injuries increased significantly with chronic ACL insufficiency (70 % medial versus 30 % lateral) [25]. Tandogan reported on a series of 764 patients with ACL tears (10.5 % women, mean age 27 years). The time from initial injury (TFI) was 19.8 months (range, 0.2–360 months). The study demonstrated that the TFI is the best predictor of medial meniscal injury: the likelihood of having a medial meniscal injury 2–5 years TFI was 2.2 times greater than the likelihood in the first year post-injury, and the likelihood at >5 years was 5.9 times greater [26].

A number of studies have also investigated the association between the type of meniscal injury and the state of the meniscus. Although the quality of the evidence is moderate, some authors have found that longitudinal, bucket-handle, or radial tears are more commonly associated with an acute traumatic event compared with horizontal or flap tears, which are more frequent in degenerative forms [27]. Poehling et al. drew the following conclusions from their study: complex meniscal tears without associated ACL injuries are more frequent between 40 and 50 years of age; horizontal tears manifest around 10 years before complex tears in the presence of an intact ACL; a flap tear is more frequent in the 30–40-year age group in men and in the 60–70-year group in women; peripheral longitudinal meniscocapsular or radial tears tend to be more common in young patients with an associated ACL tear who have therefore sustained a major traumatic injury [28]. In recent years, an increased incidence of meniscal lesions has been observed in skeletally immature children [29]. The main factor underlying this increase is the growing participation of children in physically demanding sports. In most cases (>71 %), the lesions are isolated [30–32]. In a proportion of cases, a predisposing factor is a discoid meniscus, which is, however, more frequent in the lateral compartment. This condition refers to a spectrum of morphological abnormalities, and the highly unstable forms present with the classical "snapping knee syndrome", while the stable variants may remain asymptomatic until a lesion develops [33]. The incidence of discoid meniscus ranges between 0.03 % and 0.12 % [2, 34]. The first medial discoid meniscus was reported by Cave and Staples in 1941 [35].

37.3 Classification of Meniscal Injuries

Meniscal injuries can be classified on the basis of several criteria: clinical history, lesion location relative to vasculature and "geometrical" features of the lesion:

1. As previously mentioned, meniscal lesions can be divided into traumatic or degenerative. "Traumatic" lesions are related to precise events, they predominantly affect young athletes, and they may be found in association with a cruciate ligament tear [36, 37]. "Degenerative" lesions tend to manifest as a result of chronic "wear and tear" of the meniscus in relation to the patient's age and/or joint overload due to multiple causes (obesity, malalignment, instability, overuse) [38].
2. Based on vascularity, the meniscus can be divided into three zones [5]: red-red, red-white and white-white. This classification allows an assessment of the reparability of meniscal tears since healing processes are highly likely in the first case, variable in the second and unlikely in the last case, for which resection is advisable.
3. The most commonly used classification is based on the pattern of the lesion and considers vertical (Figs. 37.1 and 37.2), horizontal and complex lesions. Vertical lesions include longitudinal and transverse/radial lesions [39]. Among them, two particularly important variants should be considered: a ramp lesion (Fig. 37.3), identified as a longitudinal lesion of the posterior horn, and a meniscal root lesion, a radial lesion of the meniscal horns or a bony avulsion of the tibial insertion [40]. Ramp lesions (Fig. 37.3) were first described in the late 1980s by Strobel [41]. In recent years, surgeons have paid increasing attention to this type of lesion, which is associated with an ACL tear in 9–17 % of cases [41, 42]. A ramp lesion affects the posteromedial corner of the medial meniscus and/or posterior menisco-tibial ligament, resulting in instability of the meniscus itself. The circumferential collagen fibres of the medial meniscal body continue posteriorly into the menisco-tibial ligament which attaches to the subchondral bone of the tibia in the most distal part of the joint space. This ligament represents a fibrocartilaginous transition zone, characterised by a gradual change in stiffness from ligament to bone: it guarantees anchoring of the meniscus and limits movement especially in the posterior direction [11, 12]. Ramp lesions are often unrecognised or misdiagnosed as partial tears, leading to loss of meniscal stability. A correct diagnosis of these lesions requires

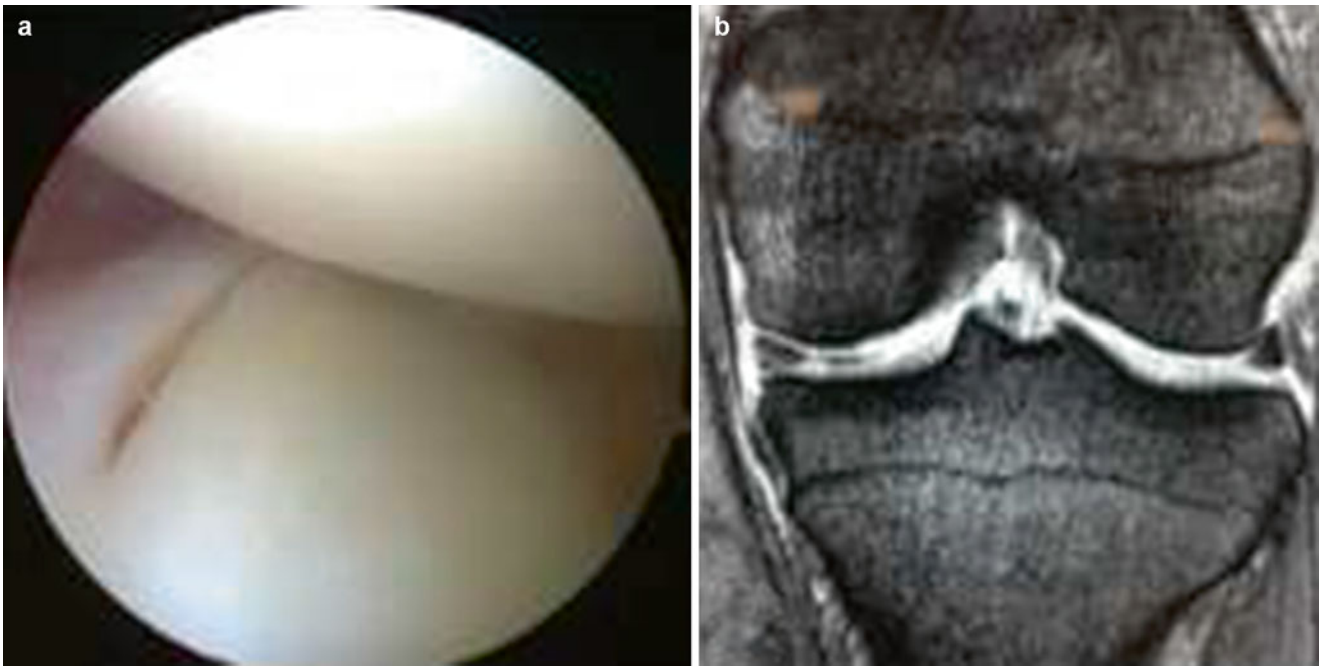


Fig. 37.1 Vertical or longitudinal tears. (**a**) Arthroscopic view. (**b**) Preoperative magnetic resonance imaging

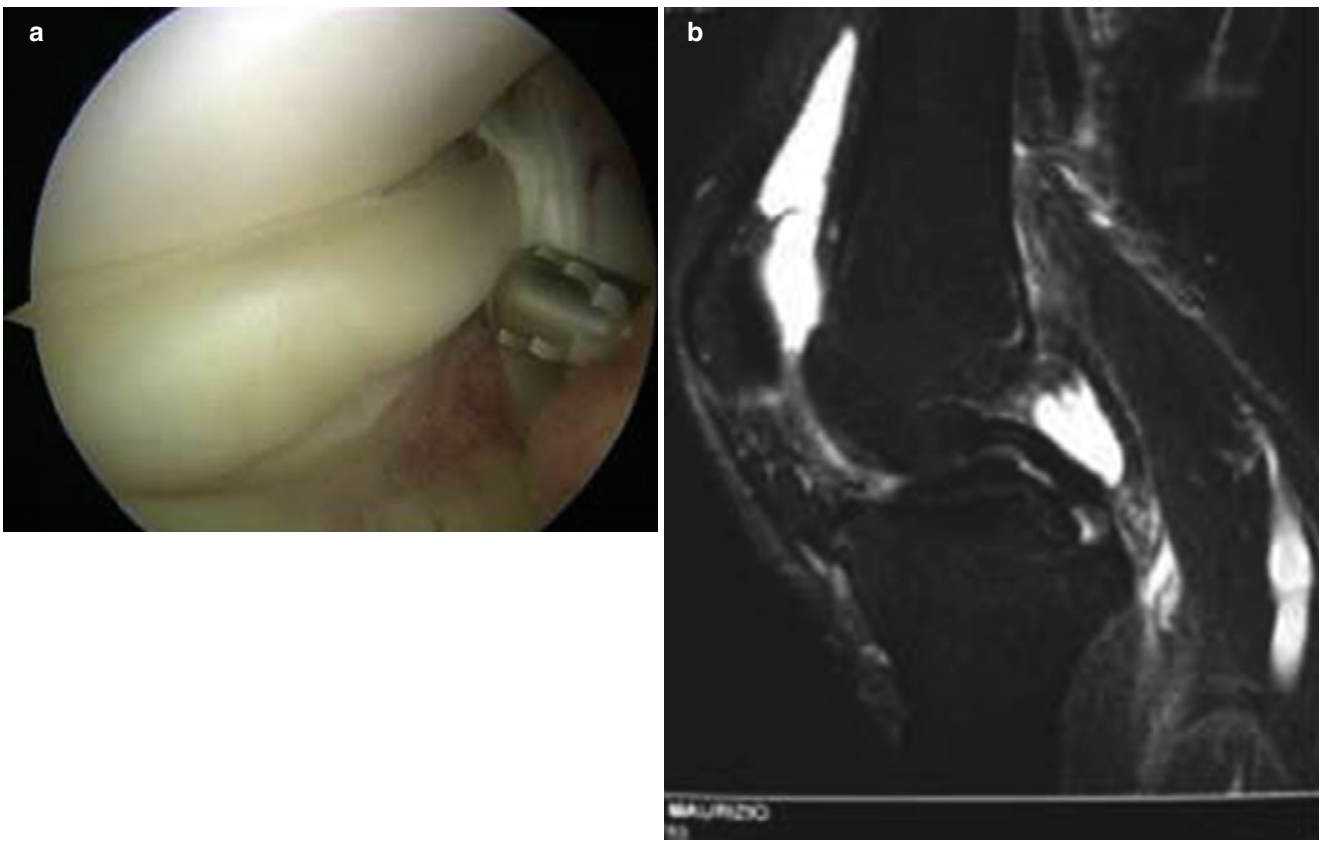


Fig. 37.2 Bucket-handle tears. (**a**) Arthroscopic view. (**b**) Preoperative magnetic resonance imaging

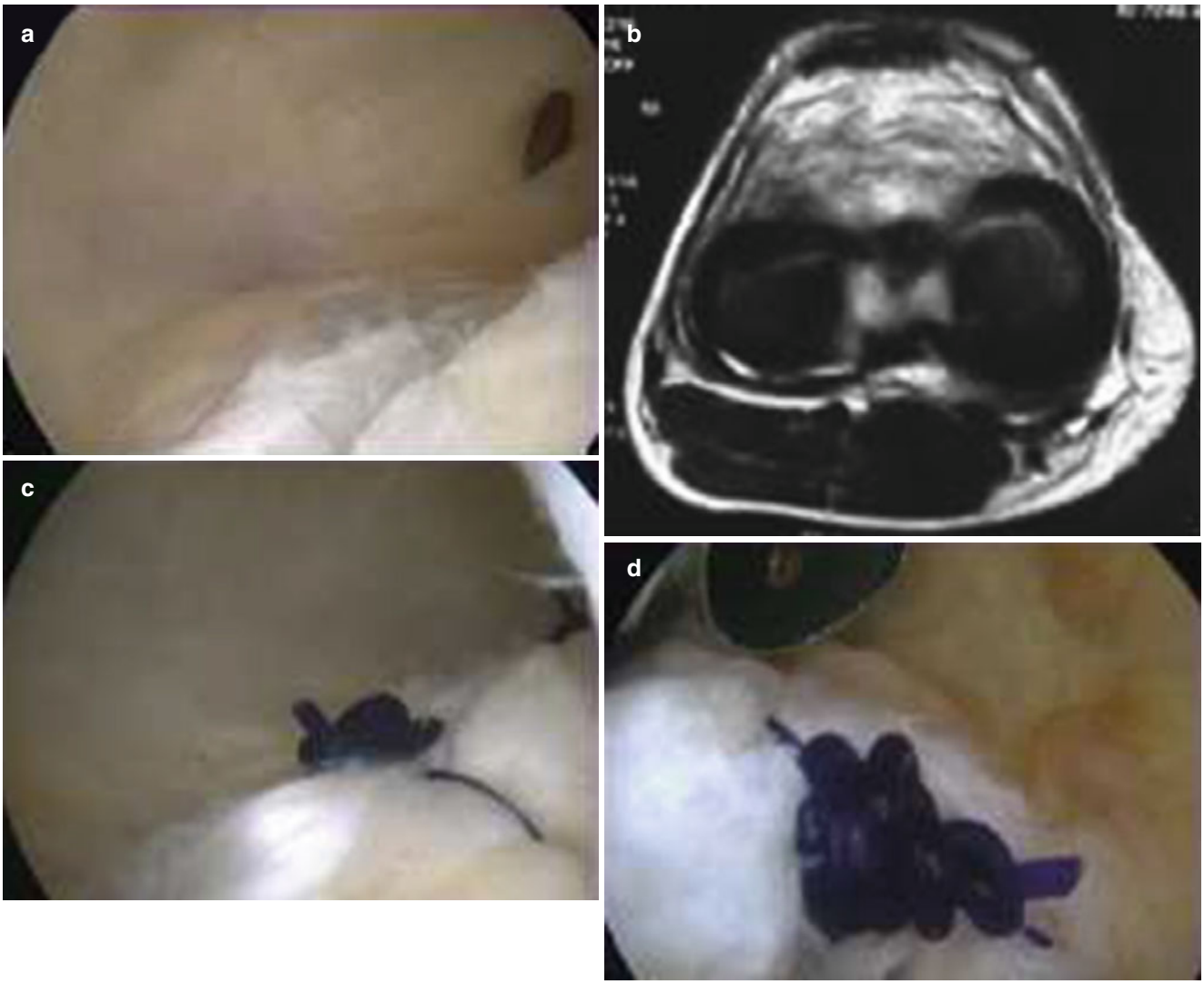


Fig. 37.3 "Ramp lesion" or "hidden lesion" of the posterior horn of the medial meniscus (MM). (**a**) Arthroscopic view. (**b**) Preoperative magnetic resonance imaging. (**c, d**) Final suture with a PDS suture. Suture through the posteromedial portal with a hook

visualisation through the posteromedial portal, and in some cases, the lesion can only be appreciated after minimal debridement of the soft tissues ("hidden lesions") [43, 44]. The incidence of meniscal root tears is greater than previously thought: tears of the medial posterior root are the most common, accounting for 10–20 % of surgeries on the medial meniscus, compared with 8 % of lateral posterior root tears (a difference explained by the greater mobility of the lateral meniscus), most of which are associated with an ACL tear. Conversely, it is not possible to establish the true incidence of anterior meniscal root lesions [40]. However, the values could be even higher if we consider that magnetic resonance imaging (MRI) may miss around one-third of radial lesions close to the posterior root of the medial meniscus. Older age, female gender, increased body mass index and lower levels of sporting activity have all been associated with a higher incidence of medial meniscus root tears [45–47]. The circumferential collagen fibres of the meniscus continue in the insertional ligaments, that is, the anterior and posterior meniscal roots, through which the meniscus attaches to the subchondral bone of the tibia. An axial load on the knee generates a compressive force on the meniscus which, with its wedge-shaped cross section, results in an outwardly directed radial vector. The circumferential fibres of the meniscus and the roots, tensed by these radial forces (hoop stress) [48, 49], allow dissipation into bone of the tensile forces generated by the load, they prevent meniscal extrusion (in combination with the peripheral connections), and they ensure preservation of articular congruity during knee movement [50–52]. A tear of the posterior root of the medial meniscus causes extrusion of the medial meniscus >3 mm [53] with similar consequences to a total meniscectomy [54].

37.4 Clinical and Diagnostic Examination

Meniscal tears may present with joint locking, limitation to extension, joint effusion and possible associated lesions of the collateral ligaments and/or ACL [41, 55, 56]. Prior to surgery, a presumptive diagnosis and a differential diagnosis can be formulated on the basis of a careful history, thorough clinical examination, and appropriate imaging investigations.

Generally, a meniscal tear will present with:

1. Acute joint pain localised to the medial joint line.
2. Joint effusion. If the tear involves the inner areas of the meniscus, the effusion generally develops gradually over a few hours. In peripheral lesions, the effusion develops much more rapidly (within minutes), and because the peripheral region is vascular, the effusion may contain blood.
3. Joint locking is a common symptom after a meniscal injury. Locking usually occurs at 20–45° of extension. It is due to a displaced meniscal fragment that becomes trapped between the femoral condyle and the tibial plateau [57].
4. A sensation of giving way may occur. In these circumstances, it is necessary to distinguish a meniscal lesion from joint instability due to other causes such as ACL and/or quadriceps femoris insufficiency. During the clinical examination, the uninvolved leg should be used as a reference for comparison of the qualitative and quantitative results of the involved leg. The examination should include inspection, palpation, range of motion (ROM), gait pattern, girth measurements and tests to assess the integrity of the menisci and other structures of the knee joint [58].

37.4.1 Tests

Several different specific tests can be applied to assess meniscal involvement. A positive result on any one test cannot by itself establish the presence of a meniscal lesion, but together with other objective findings, it can help in the differential diagnosis.

Diagnostic accuracy is improved by considering the results of three tests in combination. In general, all clinical tests tend to be less reliable in the presence of a concurrent ligamentous lesion and are less accurate in patients with degenerative lesions than in young patients with acute injuries [59]. Below are some tests that can be performed.

37.4.1.1 McMurray Test [60]

This test is positive in central tears or tears of the posterior horn. With the patient supine, the examiner holds the heel with one hand, and with the other, he/she supports the lower part of the knee, trying to extend the knee completely while rotating the tibia first internally and then externally. Pain during forced flexion suggests a lesion of the posterior meniscal horns (these meniscal portions slide back when the knee is flexed and are compressed between the proximal tibial surface and the femoral condyles); pain during forced extension suggests a lesion of the anterior meniscal horns (during extension, the menisci tend to slide forward and the anterior horns are compressed between the tibial and femoral condyles). Pain at 90° of flexion indicates a lesion of the meniscal body. Specificity is 81 %, and sensitivity is 44 %.

37.4.1.2 Apley Test [61]

The patient lies in the prone position, with knee flexed to 90° and thigh anchored to the table with the examiner's knee. The examiner distracts the patient's knee while rotating the tibia inwards and outwards. The presence of pain during this manoeuvre indicates involvement of the ligaments. The examiner then compresses the knee (grinding test) while rotating the tibia inwards (for the lateral meniscus) and outwards (for the medial meniscus). Pain during this manoeuvre indicates a meniscal tear. Specificity is 86 %, and sensitivity is 42 %.

37.4.1.3 Finocchietto Sign [62]

Sensation of a painful palpable click during the anterior drawer test is caused by entrapment of the meniscus between the femur and tibia during knee flexion and extension.

37.4.1.4 Childress Test [63]

The patient squats with knees completely flexed and attempts a "duckwalk". Limitation of movement indicates a possible meniscal lesion. However, pain in this position may indicate a meniscal lesion or involvement of the femoro-patellar articulation [63]. The Childress test has proved to be more accurate than other tests for the detection of meniscal lesions with associated ACL tears, with a sensitivity of 68 % and an accuracy of 66 %. More specific tests are the Steinmann I sign and the Apley test.

Other tests for meniscal lesions are the O'Donoghue test, the Payr sign and the Steinmann I sign.

37.4.2 Imaging Investigations

Anteroposterior (AP) and lateral knee radiography in young patients with acute trauma [64] may be combined with a Rosenberg projection [65] and a skyline view in subjects older than 40 years of age with suspected degenerative lesion. These imaging studies are required for detecting osteophytes or degenerative chondral changes through assessment of the joint space. They are also useful for detecting malalignment [66].

Magnetic resonance imaging (MRI) is the most powerful and accurate modality for the diagnosis of meniscal lesions.

It provides additional information about the state of the ligaments and cartilage [67]. It depicts many of the essential features of meniscal tears needed for making treatment decisions: location, shape, length and depth [68, 69]. As for diagnostic accuracy, it has 86–96 % sensitivity in the detection of medial meniscus tears, with a specificity of 84–94 % [70, 71]. In reality, if a pathological signal is detected on one section only, the accuracy of the diagnosis of a medial meniscal tear is 55 %. The accuracy increases if the pathological signal is seen in at least two consecutive images [71]. MRI is also accurate in the detection of repairable meniscal lesions, and it has good sensitivity for determining irreparable lesions. One study has shown that 89 % (103 of 116) of meniscal lesions were classified equally at MRI and subsequent arthroscopy with regard to reparability. However, the same study demonstrated that MRI had variable accuracy in predicting the configuration of the meniscal lesion later identified with arthroscopy [72]. MRI diagnosis is, however, associated with a risk of false-negative or false-positive results. False-negative results are more common with lesions located in the outer half of the posterior horn of the medial meniscus or in the inner third of the meniscal body. These are small lesions detected clinically by palpation of the joint line; they are stable and may be treated conservatively [73]. False-positive results are more frequent and concern the posterior horn of the medial meniscus. They are caused by chondrocalcinosis in the meniscus or healed meniscal lesions, which often continue to show high signal intensity. In the presence of chondrocalcinosis, MRI sensitivity in the medial meniscus is 89 %, specificity is 72 % and accuracy is 81 %. One of the causes of the diagnostic difficulties lies in the differences in descriptive terminology and interpretation used by orthopaedic surgeons and radiologists with regard to the lesion being examined [74, 75].

37.5 Treatment Strategy

Stable lesions <1 cm in length and not causing any significant mechanical symptoms can be treated with plain observation [76]. Surgery becomes necessary when the lesions give rise to mechanical symptoms. Lesions >1 cm can be distinguished into reparable and irreparable, depending on location. Only 20 % of meniscal tears are suitable for repair. The factors influencing success include the age of the lesion, the location, the type of lesion, the patient's age and the possible presence of associated lesions [77, 78].

37.5.1 Total Meniscectomy

In the past, meniscal lesions were treated with total meniscectomy. This occurred because the true functions of the meniscus were unknown, and in his 1940 paper "The Semilunar Cartilages", McMurray wrote that "The cause of failure of meniscal removal lies in the failure to remove the entire affected cartilage" [79]. In 1948, Fairbanks published what is now a classical paper, in which he described the characteristic radiographic changes produced by meniscectomy. Whereas today it is clear that these changes reflect the disruption of the tibio-femoral joint due to loss of the meniscal protective function, Fairbanks observed that "It seems likely that narrowing of the joint space will predispose to degenerative changes, but a connection between these appearances and later osteoarthritis is not yet established and is too indefinite to justify clinical deductions" [50]. During most of the twentieth century, therefore, orthopaedic surgeons carried out a large number of total meniscectomies with good short-term outcomes [80]. However, looking at the results over longer follow-up periods, the negative outcomes became increasingly apparent. For example, Tapper and Hoover from the Mayo Clinic reported on the results of a retrospective study of 213 patients followed up from 10 to 30 years after their meniscectomy, in which only 68 % had good or excellent results and only 38 % were asymptomatic. Moreover, they found that those with a bucket-handle tear but with intact peripheral meniscal rim had higher rates of satisfaction [81].

In the light of these results, there is an increased awareness of the importance of meniscal function [82], and the current motto of orthopaedic surgeons is "Save the meniscus". Recently, R. Verdonk stated that we have to "keep the meniscus… vestigial soft tissue structure in a self-maintaining transmission system" [83].

37.5.2 Partial Meniscectomy

The development of arthroscopic techniques and related surgical instruments has facilitated the performance of partial meniscectomy, which has become a pillar of the treatment of symptomatic lesions. The short-term results of partial meniscectomies are very satisfactory. Jareguito et al. have reported that in 90 % of cases, they obtained good or satisfactory results at 2-year follow-up and that 85 % of these patients returned to the desired activity level. However, at 8-year follow-up, the results remained good or excellent in 62 % of cases, and only 48 % maintained the pre-injury level of activity [84]. All in all, the differences in the functional scores between total and partial meniscectomy decrease over longer follow-up periods, on average after 7–8 years. Lee et al., in a biomechanical study, demonstrated that also partial meniscectomy influences the transmission of articular loads. They concluded that the goal is to preserve the greatest amount of meniscus possible [85]. Thus, we have biomechanical and clinical evidence suggesting the superiority of partial meniscectomy, despite its limitations of not being able to maintain

an adequate biomechanical behaviour and avoid degenerative changes.

Medial meniscectomy is performed with the patient under spinal anaesthesia or with local anaesthesia and sedation depending on the surgeon's preference and anaesthesiological concerns. Generally, a tourniquet is applied to the upper thigh, although some surgeons prefer to carry out the arthroscopy without it. The limb may be placed in a leg holder or on a flat table with a lateral post: both devices allow a valgus force to be applied in order to correctly visualise the internal compartment. The classical arthroscopic access points are used (antero-medial and antero-lateral); the use of the supero-medial portal for the outflow cannula is optional. Using an arthroscopic probe, the lesion is carefully assessed: a flap, a radial lesion, a dislocated bucket handle, and degenerate appearance of a complex lesion are all indications for partial meniscectomy. In the case of a flap tear, the probe is used to accurately visualise the site of attachment to then go on to resect it and remove the free meniscal fragment. Bucket-handle tears can be treated with a variety of techniques. The one we prefer consists in first resecting the anterior attachment base and then, after having caught the meniscal fragment with an appropriate grasper through an accessory antero-lateral portal (just below the standard portal), resecting the posterior attachment from the medial portal while applying traction and torsion. If visualisation of the area of the posterior attachment base proves difficult, a varus force with the semiflexed knee can be applied to facilitate the passage of the instrumentation: this will help to open up the space between the medial femoral condyle and the posterior cruciate ligament. Finally, the contour is smoothed with a shaver. If the procedure involves the posterior horn of the medial meniscus, a valgus force with extended and internally rotated knee may be required. In all cases, before ending the procedure, the stability of the meniscal remnant needs to be assessed. In patients with excessively tight MCL, it may be justified to release it to ensure correct visualisation of the medial compartment: this can be done by performing multiple needle punctures subcutaneously.

37.5.2.1 Complications

1. Infection. The infection rate in meniscectomies ranges from 0.04 to 0.42 % [86, 87].
2. Recurrent joint effusion. This is due to the conflict between the femoral condyle and tibial plateau in the absence of the meniscus.
3. Synovial fistula (take care to suture the posteromedial portal).
4. Embolic events. Data from phlebographic studies report an incidence of deep vein thrombosis (DVT) similar to that observed in other surgical procedures at moderate-to-high risk (18 % total DVT and 5 % proximal DVT) even though other, ultrasonography-based, studies have reported lower incidence rates. Application of the tourniquet seems to be an additional risk factor, but, on the other hand, it allows for shorter procedure times. In a study conducted on low-risk patients, reviparin prophylaxis (at a daily dose of 1,750 IU, suitable for moderate risk) for an average of 8 days was associated with a reduction of DVT from 4.1 to 0.85 % [88].
5. Iatrogenic damage to cartilage. A recent study found that treatment of lesions of the medial posterior horn caused mild damage to the femoral condyles in 28 % of cases and moderate-to-severe damage in 6 % of cases [89].
6. Ligamentous lesions. An uncommon complication: Small reported two cases of MCL stretching in a series of 1,184 arthroscopies [90].
7. Breakage of surgical instruments. This is a real, though uncommon, complication (0.3 %) which has decreased over the years with the introduction of new surgical instruments and growing surgeons' experience [91].
8. Vascular damage. Mostly pseudoaneurysms of the popliteal artery or arteriovenous fistulas, with an incidence of 0.05 % [92].
9. Nerve damage. Although definitely more common during meniscal repair, this complication has also been reported in meniscectomies, with a rate of 0.6 %. In most cases, the infrapatellar branch of the saphenous nerve is injured during creation of the arthroscopic portals [93].
10. Osteonecrosis. This complication was initially observed in cases of arthroscopic meniscectomy: osteonecrosis of the postoperative knee (ONPK). In fact, more recently it has also been observed following other arthroscopic procedures (chondroplasty, ACL reconstruction) [94–97]. It predominantly affects the medial femoral condyle (82 %), followed by the lateral femoral condyle, the lateral tibial plateau and finally the medial tibial plateau. In 65 % of cases, there is pre-existing chondral damage [95, 98, 99]. Possible causes are:
    - An aggressive postoperative rehabilitation may contribute to the development of this condition. Rapid return to weight-bearing activities and exercise (before the bone remodelling stimulated by the changes in load distribution) induces insufficiency fractures [100].
    - An increase in cartilage permeability to arthroscopic fluid (due to cartilage lesions or iatrogenic damage during the procedure) may lead to increased interosseous pressure, with oedema and necrosis [101].
    - Direct injury from thermal effects (laser) or photoacoustic trauma (gas bubbles = shock wave), during radiofrequency use. The inflammatory response leads to bone oedema, locally increased intraosseous pressure and osteonecrosis [102, 103].

- Even the presence of a medial meniscus lesion has been suggested as a potential aetiological factor. In particular, a tear of the posterior meniscal root has been implicated (incidence of 2.8 % among 1,500 knees examined). In 80 % of knees with this diagnosis, an osteonecrosis manifested clinically. The suggested pathogenic mechanism is a change in the loads transmitted to the femoral condyle due to loss of the dissipation function of the condyles themselves as a result of increased compartmental pressure and therefore osteolysis and osteonecrosis [104].

37.5.3 Meniscal Repair

Prompted by the studies of Arnozcky and Warren [5] on meniscal vascularity and healing potential, and also thanks to the introduction of new arthroscopic techniques, meniscal repair has become more widespread. The indications can be summarised as follows:

- Patient-related factors: instability, locking, effusion, grinding and pain
- Factors deriving from the physical examination: joint-line tenderness, effusion and limitation of motion
- Specific tests for meniscal tears
- MRI (tear of the red-red or red-white zone)
- Arthroscopy: reducible tear

The contraindications are degenerate or poorly vascular meniscus tissue, patient's age, patient's limited compliance with the postoperative physiotherapy protocol, untreated knee instability, osteoarthritis, chronic lesions, longitudinal tears shorter than 1 cm, or radial tears. Several different techniques are available for meniscal repair:

1. Scott and co-workers [105] and Cannon [106] developed the inside-out repair technique. This involves the use of double-lumen, zone-specific cannulae [107] through which needles preloaded with suture material are passed. In the inside-out repair of posterior horn tears, a counter-incision is made to prevent neurovascular complications. In the repair of medial meniscus tears, the incision must be posterior and parallel to the MCL and anterior to the medial gastrocnemius. The knee must be flexed to 5–15°. The technique can be used for tears of the posterior horn and meniscal body.
2. Warren [108] was the first to describe the outside-in repair technique, which is relatively inexpensive (use of an 18-gauge needle + monofilament) and simple. Its main indication is repair of the middle third of the meniscus and of the anterior horn. Possible complications are iatrogenic lesions to the meniscus due to multiple attempts at suture needle placement or neurovascular lesions.
3. Morgan introduced the all-inside technique utilising arthroscopic knots [109]. This is indicated in longitudinal tears of the posterior horn and ramp lesions. It is a difficult technique that requires creation of a posteromedial access through which a special hook or pigtail needle is passed to perform a vertical PDS suture of the posterior horn. This technique has seen the greatest efforts of manufacturers of orthopaedic materials to provide increasingly precise and easy-to-use devices to make the technique more accessible. Surgeons have at their disposal solid implants (polylactic acid anchors) [110] – in actual fact used increasingly less often because of the possible postoperative complications – or anchoring systems (partially bioabsorbable filaments mounted on bioabsorbable anchors) [90].

The characteristics and location of the tear and the operator's experience can influence the type of technique adopted, even though all-inside techniques are being increasingly used since they are relatively easy to apply and do not require accessory surgical accesses. However, several studies have raised the problem of the failure of these methods even though the results are all in all similar to those achieved with the inside-out technique. The possible complications of repair techniques for tears of the medial meniscus are:

1. Injury to the saphenous vein or nerve. In particular, a transient neuropraxia was found in 22 %, whereas permanent damage was seen in 0.4–1 % of cases [90, 111].
2. Involvement of the popliteal artery with the T-Fix system (Smith & Nephew Endoscopy) if no depth penetration limiter is utilised [112].
3. Risk of penetrating the sartorius tendon or deep fascia of MCL, which may slow functional recovery because of postoperative pain [113].
4. Failure and migration of the solid implants with cartilage lesions and/or synovitis [114]. Technique: debride the fibrous tissue with a rasp or shaver. In some cases, needling at the level of the meniscal joint line may be done to stimulate bleeding of the vascular plexus. Debridement of the posterior region of the medial meniscus may no doubt prove difficult, and a possible solution is to create an accessory posteromedial portal. Once the meniscus has been prepared, the various repair techniques can be performed:
   (a) In all-inside procedures, the sutures are generally inserted via the ipsilateral portal for posterior horn tears and via the contralateral portal for meniscal body tears.
   (b) In inside-out procedures, single- or double-bore cannulae are used. In this case the suture needles are retrieved from the posteromedial portal.
   (c) In outside-in procedures, PDS monofilaments are passed through 18G needles and are then tied over the capsule after making multiple small skin incisions.

Techniques for the repair of meniscal root tears are divided into two categories, both arthroscopy-assisted and with the possibility of creating accessory portals:

(a) Trans-osseous repair techniques [115, 116]: targeting devices for ligament reconstruction are used to create tunnels at the level of the root, and the meniscal horns are prepared with mattress sutures utilising shoulder forceps.
(b) Anchor insertion techniques [117].

The published results on meniscal repair are varied for several reasons. In the presence of concurrent ACL reconstruction, the failure rate is 10 %. In this situation, the healing process may be stimulated by the massive haemarthrosis. Clinically, patient satisfaction after repair procedures ranges from 87 to 98 %, but this does not imply that the lesion has definitely healed: recurrences can often be asymptomatic. In fact, second-look arthroscopy studies have shown a success rate of 75 % [118–120]. MR arthroscopy is a more useful imaging modality for detection of the healing process than conventional MRI. The intra-articular or intravenous (indirect form) injection of gadolinium increases the potential of MRI by boosting the signal of the synovial fluid and thereby enhancing the contrast of the anatomical structures in T1-weighted sequences [67].

37.5.4 Biological Repair

Biological repair techniques try to enhance and/or overcome the limitations of meniscal self-repair capabilities by promoting chemotaxis, matrix production and cell proliferation at the site of injury.

Among them are, first of all, mechanical techniques promote healing by creating vascular accesses and stimulating the cellular elements: synovial and meniscal abrasion and radial trephination of the meniscal wall. The concentration of major growth factors in the healing process as a result of these surgical actions has been demonstrated: in particular, the fibroblastic growth factor-2 (FGF-2) and the connective tissue growth factor (CTGF) proved to effectively enhance the repair process, whereas the vascular endothelial growth factor (VEGF) failed to show similar benefits.

Partly in view of these results, today there is a tendency to use minimally manipulated biological "materials" more frequently: platelet-rich plasma (PRP) and bone marrow aspirate concentrate (BMAC). In particular, PRP contains numerous growth factors (TGFß1, PDGF, VEGF) involved in cell differentiation, chemotaxis, angiogenesis and extracellular matrix production. However, further studies are required for the scientific validation of these products [121, 122].

37.6 Rehabilitation and Return to Play

The timing of rehabilitation should be dictated by patient- and surgical procedure-related considerations. In the case of a partial meniscectomy, the absence of symptoms could lead to a rapid return to activity. However, the joint cartilage may require some time to adjust to the changes in loading in the absence of the meniscus, and too early a return to play can cause an even greater risk of chondral degeneration at long-term follow-up. The athlete should therefore be advised to refrain from physical activity for a period of around 4–6 weeks.

In the case of meniscal repair, the main concern is to maintain and protect the healing processes. For this reason, full weight-bearing should be avoided for 4 weeks (so as not to expose the site to shear and compression forces likely to affect healing dynamics). In the following 2–3 weeks, progressive recovery of full weight-bearing and athletic motion can take place with return to play not before 4–6 months. Accelerated rehabilitation protocols are being studied that attempt to promote a faster recovery, but the major difficulties for return to play lie in the lack of reliable investigations to ascertain that the repaired meniscal tears have healed. These protocols are based on new knowledge regarding healing mechanics and biology: the action of mechanical forces could drive the formation of meniscal repair tissue as occurs with bony callus formation [120, 123].

References

1. Messner K, GAO J (1998) The menisci of the knee joint. Anatomical and functional characteristics and a rationale for clinical treatment. J Anat 193:161–178
2. Clark CR, Ogden JA (1983) Development of the menisci of the human knee joint. Morphological changes and their potential role in childhood meniscal injury. J Bone Joint Surg Am 65:538–547
3. Shaffer B, Kennedy S, Klimkiewicz J, Yao L (2000) Preoperative sizing of meniscal allografts in meniscus transplantation. Am J Sports Med 28:524–533
4. McDermott ID, Sharifi F, Bull AM, Gupte CM, Thomas RW, Amis AA (2004) An anatomical study of meniscal allograft sizing. Knee Surg Sports Traumatol Arthrosc 12:130–135
5. Arnoczky SP, Warren RF (1982) Microvasculature of the human meniscus. Am J Sports Med 10:90–95
6. Wilson AS, Legg PG, McNeur JC (1969) Studies on the innervation of the medial meniscus in the human knee joint. Anat Rec 165:485–491
7. Kohn D, Moreno B (1995) Meniscus insertion anatomy as basis for meniscus replacement: a morphological cadaveric study. Arthroscopy 11:96–113
8. Kennedy JC, Alexander IJ, Hayes KC (1982) Nerve supply of the human knee and its functional importance. Am J Sports Med 10:329–335
9. Zimmy ML, Albrigth DJ, Dabezies E (1984) Mechanoreceptors in the human medial meniscus. Acta Anat (Basel) 133:35–40
10. Beaupre A, Choukroun R, Guidouin R et al (1986) Knee menisci. Correlation between microstructure and biomechanics. Clin Orthop Relat Res 208:72–75

11. Robinson JR, Bull AMJ, Thomas RR (2006) The role of medial collateral ligament and posteromedial capsule in controlling knee laxity. Am J Sports Med 34:1815–1823
12. Mariani PP (2011) Posterior horn instability of the medial meniscus a sign of posterior meniscotibial ligament insufficiency. Knee Surg Sports Traumatol Arthrosc 19:1148–1153
13. Kessler MW, Sgaglione NA (2011). All-Arthroscopic meniscus repair of avascular and biologically at-risk meniscal tears. Instructional Course Lectures. 60:439–452
14. Baker BE, Peckham AC, Pupparo F, Sanborn JC (1985) Review of meniscal injury and associated sports. Am J Sports Med 13:1–4
15. Renstrom P, Johnson PJ (1990) Anatomy and biomechanics of the menisci. Clin Sports Med 9:523–538
16. Praemer A, Furner S, Rice DP (1999) Musculoskeletal condition in the United States. American Academy of Orthopaedic Surgeons, Rosemont
17. Steinbruck K (1999) Epidemiology of sports injuries–25-year-analysis of sports orthopedic-traumatologic ambulatory care. Sportverletz Sportschaden 13:38–52
18. Terzidis IP, Christodoulou A, Ploumis A, Givissis P, Natsis K, Koimtzis M (2006) Meniscal tear characteristics in young athletes with a stable knee arthroscopic evaluation. Am J Sports Med 34–7:1170–1175
19. Noble J, Hamblen DL (1975) The pathology of the degenerate meniscus lesion. J Bone Joint Surg Br 57:180–186
20. Salata MJ, Gibbs AE, Sekiya JK (2010) A systematic review of clinical outcomes in patients undergoing meniscectomy. Am J Sports Med 38:1907–1916
21. Greis PE, Bardana DD, Holmstrom MC, Burks RT (2002) Meniscal injury: I. Basic science and evaluation. J Am Acad Orthop Surg 10:168–176
22. Noyes FR, Chen RC, Barber-Westin SD, Potter HG (2011) Greater than 10- year results of red-white longitudinal meniscal repairs in patients 20 years of age or younger. Am J Sports Med 39:1008–1017
23. Ahlde'n M, Samuelsson K, Sernert N, Forssblad M, Karlsson J, Kartus J (2012) The Swedish National Anterior Cruciate Ligament Register: a report on baseline variables and outcomes of surgery for almost 18,000 patients. Am J Sports Med 40:2230–2235
24. Granan LP, Inacio MC, Maletis GB, Funahashi TT, Engebretsen L (2012) Intraoperative findings and procedures in culturally and geographically different patient and surgeon populations: an anterior cruciate ligament reconstruction registry comparison between Norway and the USA. Acta Orthop 83:577–582
25. Bellabarba C, Bush-Joseph CA, Bach BR Jr (1997) Patterns of meniscal injury in the anterior cruciate-deficient knee: a review of the literature. Am J Orthop (Chatham NJ) 26(1):18–23
26. Tandogan RN, Taser O, Kayaalp A, Taskiran E, Pinar H, Alparslan B et al (2004) Analysis of meniscal and chondral lesions accompanying anterior cruciate ligament tears: relationship with age, time from injury, and level of sport. Knee Surg Sports Traumatol Arthrosc 12(4):262–270
27. Englund M, Roos EM, Roos HP, Lohmander LS (2001) Patient-relevant outcomes fourteen years after meniscectomy: influence of type of meniscal tear and size of resection. Rheumatology 40(6):631–639
28. Poehling GG, Ruch DS, Chabon SJ (1990) The landscape of meniscal injuries. Clin Sports Med 9(3):539–549
29. Kocher MS, Micheli LJ (2001) The pediatric knee: evaluation and treatment. In: Surgery of the knee, 3rd edn. Churchill-Livingstone, New York, pp 1356–1397
30. Robert M, Gouault E, Moulies D, Alain JL (1986) Meniscal lesions in the child athlete. Acta Orthop Belg 52:72–80
31. Angel KR, Hall DJ (1989) The role of arthroscopy in children and adolescents. Arthroscopy 5:192–196
32. Maffulli N, Chan KM, Bundoc RC, Cheng JC (1997) Knee arthroscopy in Chinese children and adolescents: an eight-year prospective study. Arthroscopy 13:18–23
33. Kramer DE, Micheli LJ (2009) Meniscal tears and discoid meniscus in children: diagnosis and treatment. J Am Acad Orthop Surg 17:698–707
34. Schonholtz GJ, Koenig TM, Prince A (1993) Bilateral discoid medial menisci: a case report and literature review. Arthroscopy 9:315–317
35. Cave EF, Staples OS (1941) Congenital discoid meniscus, a cause of internal derangement of the knee. Am J Surg 54(Nov):371–376
36. Dickason JM, Del Pizzo W, Balzina ME et al (1982) A series of ten discoid medial menisci. Clin Orthop Relat Res 168:75–79
37. Englund M, Guermazi A, Lohmander SL (2009) The role of the meniscus in knee osteoarthritis: a cause or consequence? Radiol Clin North Am 47(4):703–712
38. Englund M (2008) The role of the meniscus in osteoarthritis genesis. Rheum Dis Clin North Am 34(3):573–579
39. Rosemberg TD, Kolowich PA (1990) Arthroscopic diagnosis and treatment of meniscal disorders. In: Scotts WN (ed) Arthroscopy of the knee. WB Saunders, Philadelphia, p 67
40. Bhatia S, LaPrade CM, Ellman MB, LaPrade R (2014) Meniscal root tears. Significance, diagnosis, and treatment. Am J Sports Med 42(12):3016–3016
41. Strobel MJ (1988) Menisci. In: Fett HM, Flechtner P (eds) Manual of arthroscopic surgery. Springer, New York, pp 171–178
42. Bollen SR (2010) Posteromedial meniscocapsular injury associated with rupture of the anterior cruciate ligament: a previously unrecognized association. J Bone Joint Surg Br 92:222–223
43. Liu X, Feng H, Zhang H, Hong L, Wang XS, Zhang J (2011) Arthroscopic prevalence of ramp lesion in 868 patients with anterior cruciate ligament injury. Am J Sports Med 39:832–837
44. Sonnery-Cottet B, Conteduca J, Thaunat M, Gunepin FX, Seil R (2014) Hidden lesions of the posterior horn of the medial meniscus. A systematic arthroscopic exploration of the concealed portion of the knee. Am J Sports Med 42:921–926
45. Bin SI, Kim JM, Shin SJ (2004) Radial tears of the posterior horn of the medial meniscus. Arthroscopy 20(4):373–378
46. Koenig JH, Ranawat AS, Umans HR, Difelice GS (2009) Meniscal root tears: diagnosis and treatment. Arthroscopy 25(9): 1025–1032
47. Hwang BY, Kim SJ, Lee SW et al (2012) Risk factors for medial meniscus posterior root tear. Am J Sports Med 40(7):1606–1610
48. Cox JS, Nye CE, Schaefer WW et al (1975) The degenerative effects of partial and total resection of the medial meniscus in dog's knees. Clin Orthop 109:178–183
49. Kamamura S, Kotito K, Rodeo SA (2003) Biomechanics and healing response of the meniscus. Oper Tech Sports Med 11:68–76
50. Fairbank TJ (1948) Knee joint changes after meniscectomy. J Bone Joint Surg Br 30:664–669
51. Pagnani MJ, Cooper DE, Warren RF (1991) Extrusion of the medial meniscus. Arthroscopy 7(3):297–300
52. Goertzen D, Gillquist J, Messner K (1996) Tensile strength of the tibial meniscal attachments in the rabbit. J Biomed Mater Res 30:125–128
53. Lerer DB, Umans HR, Hu MX, Jones MH (2004) The role of meniscal root pathology and radial meniscal tear in medial meniscal extrusion. Skeletal Radiol 33:569–574
54. Allaire R, Muriuki M, Gilbertson L, Harner CD (2008) Biomechanical consequences of a tear of the posterior root of the medial meniscus: similar to total meniscectomy. J Bone Joint Surg Am 90:1922–1931
55. Seedholm BB, Dawson D, Wright V (1974) Function of the menisci: a preliminary study. J Bone Joint Surg Br 56:381
56. Strobel MJ (2009) Manual of arthroscopic surgery, vol 1. Springer, Berlin, pp 99–200

57. Iobst CA, Stanitski CL (2000) Acute knee injuries. Clin Sports Med 19(4):621–635
58. Konan S, Rayan F, Haddad FS (2009) Do physical diagnostic tests accurately detect meniscal tears? Knee Surg Sports Traumatol Arthrosc 17(7):806–811
59. Terry GC, Tagert BE, Young MJ (1995) Reliability of the clinical assessment in predicting the cause of internal degeneraments of the knee. Arthroscopy 11:568–576
60. McMurray T (1942) The semilunar cartilages. Br J Surg 29:407–414
61. Apley A (1947) The diagnosis of meniscus injuries. J Bone Joint Surg 29:78–84
62. Finocchietto R (1956) The meniscus of the knee: leap sign. Prensa Med Argent 43(3):270
63. Tria AJ Jr (2001) Clinical examination of the knee. In: Insaii JN, Scott WN (eds) Surgery of the knee, 3rd edn. Churchill Livingstone, New York, pp 161–174
64. Wareluk P, Szopinski KT (2012) Value of modern sonography in the assessment of meniscal lesions. Eur J Radiol 81(9): 2366–2369
65. Rosenberg TD, Paulos LE, Parker RD et al (1988) The forty-five-degree posteroanterior flexion weight-bearing radiograph of the knee. J Bone Joint Surg Am 70:1479–1483
66. Railhac JJ, Fournie A, Gay R et al (1981) A radiologic study of the knee in an antero-posterior incidence with light flexion and standing up position. Its interest in the diagnosis of femoro-tibial osteoarthrosis. J Radiol 62:157–166
67. Vance K, Meredick R, Schweitzer ME, Lubowitz JH (2009) Magnetic resonance imaging of the postoperative meniscus. Arthroscopy 25(5):522–530
68. Rubin DA, Kettering JM, Towers JD (1998) MR imaging of knees having isolated and combined ligament injuries. AJR Am J Roentgenol 170(5):1207–1213
69. Rubin DA, Paletta GA Jr (2000) Current concepts and controversies in meniscal imaging. Magn Reson Imaging Clin N Am 8(2):243–270
70. Crues JV 3rd, Mink J, Levy TL, Lotysch M, Stoller DW (1987) Meniscal tears of the knee: accuracy of MR imaging. Radiology 164(2):445–448
71. De Smet AA, Asinger DA, Johnson RL (2001) Abnormal superior popliteomeniscal fascicle and posterior pericapsular edema: indirect MR imaging signs of a lateral meniscal tear. AJR Am J Roentgenol 176(1):63–66
72. Cotten A, Delfaut E, Demondion X (2000) MR imaging of the knee at 0.2 and 1.5T: correlation with surgery. AJR Am J Roentgenol 174(4):1093–1097
73. Justice WW, Quinn SF (1995) Error patterns in the MR imaging evaluation of menisci of the knee. Radiology 196(3):617–621
74. Kaushik S, Erickson JK, Palmer WE, Winalski CS, Kilpatrick SJ, Weissman BN (2001) Effect of chondrocalcinosis on the MR imaging of knee menisci. AJR Am J Roentgenol 177(4):905–909
75. De Smet AA, Nathan DH, Graf BK, Haaland BA, Fine JP (2008) Clinical and MRI findings associated with false-positive knee MR diagnoses of medial meniscal tears. AJR Am J Roentgenol 191(1):93–99
76. Weiss CV, Lundberg M, Hamberg P, DeHaven KE, Gillquist J (1989) Non-operative treatment of meniscal tears. J Bone Joint Surg Am 71:811–822
77. DeHaven KE (1981) Peripheral meniscal repair. An alternative to meniscectomy. J Bone Joint Surg Br 63:463
78. Warren RF (1985) Arthroscopic meniscal repair. Arthroscopy 1:170–172
79. McMurray TP (1940) The semilunar cartilages. Br J Surg 29:407–414
80. Perey O (1962) Follow-up results of meniscectomy with regard to working capacity. Acta Orthop Scand 32:457–460
81. Tapper EM, Hoover NW (1969) Late results after meniscectomy. J Bone Joint Surg Am 51:517–526
82. Masouros SD, McDermott ID, Bull AMJ, Amis AA (2010) Biomechanics. The Meniscus 1(4):29–37
83. Verdonk R (2010) Synthesis. In: The meniscus. Springer, Berlin/Heideberg. 4.7, p 155
84. Jareguito JW, Elliot JS, Leitner T et al (1995) The effects of arthroscopic partial lateral meniscectomy in an otherwise normal knee: a retrospective review of functional, clinical, and radiographic results. Arthroscopy 11:29–36
85. Lee SJ, Aadalen KJ, Malaviyan P et al (2006) Tibiofemoral contact mechanics after serial partial meniscectomies in the human cadaveric knee. Am J Sports Med 34:1334–1343
86. Armstrong RW, Bolding F, Joseph R (1992) Septic arthritis following arthroscopy: clinical syndromes and analysis of risk factors. Arthroscopy 8(2):213–223
87. Coudane H, Buisson P (2001) Symposium complications de l'arthoscopie. In: Perspectives en arthroscopie, vol 2. Springer, Berlin/Heidelberg, pp 120–137
88. Geerts WH et al; American College of Chest Physicians (2008) Prevention of venous thromboembolism. American College of Chest Physicians Evidence-Based Clinical Practice Guidelines (8th Edition). Chest 133:S381–S454
89. Lubowitz JH, Rossi MJ, Baker BS, Guttmann D (2004) Arthroscopic visualization of the posterior compartments of the knee. Arthroscopy 20(7):675–680
90. Small NC (1988) Complications in arthroscopic surgery performed by experienced arthroscopists. Arthroscopy 4(3):215–221
91. Kramer DE, Bahk MS, Cascio BM, Cosgarea AJ (2006) Posterior knee arthroscopy: anatomy, technique, application. J Bone Joint Surg Am 88(Suppl 4):110–121
92. Kim TK, Savino RM, McFarland EG, Cosgarea AJ (2002) Neurovascular complications of knee arthroscopy. Am J Sports Med 30(4):619–629
93. Sherman OH, Fox JM, Snyder SJ, Del Pizzo W, Friedman MJ, Ferkel RD, Lawley MJ (1986) Arthroscopy–"no-problem surgery". An analysis of complications in two thousand six hundred and forty cases. J Bone Joint Surg Am 68(2):256–265
94. Santori N, Condello V, Adriani E et al (1995) Osteonecrosis after arthroscopic medial meniscectomy. Arthroscopy 11:220–224
95. Faletti C, Robba T, DePetro P (2002) Postmeniscectomy osteonecrosis. Arthroscopy 18:91–94
96. Pape D, Seil R, Anagnostakos K, Kohn D (2007) Postarthroscopic osteonecrosis of the knee. Arthroscopy 23:428–438
97. Akgun RC, Tandogan NR, Karaman A, Akkaya T, Ozgur AF, Tuncay IC (2007) Development of osteonecrosis after arthroscopic meniscal and chondral knee surgery: a report of five cases. Acta Orthop Traumatol Turc 41:80–88
98. Johnson TC, Evans JA, Gilley JA, DeLee JC (2000) Osteonecrosis of the knee after arthroscopic surgery for meniscal tears and chondral lesions. Arthroscopy 16:254–261
99. DeFalco RA, Ricci AR, Balduini FC (2003) Osteonecrosis of the knee after arthroscopic meniscectomy and chondroplasty: a case report and literature review. Am J Sports Med 31(6):1013–1016, Review. No abstract available
100. Nakamura N, Horibe S, Nakamura S, Mitsuoka T (2002) Subchondral microfracture of the knee without osteonecrosis after arthroscopic medial meniscectomy. Arthroscopy 18(5): 538–541
101. Pruès-Latour V, Bonvin JC, Fritschy D (1998) Nine cases of osteonecrosis in elderly patients following arthroscopic meniscectomy. Knee Surg Sports Traumatol Arthrosc 6:142–147
102. Edwards RB III, Lu Y, Nho S et al (2002) Thermal chondroplasty of chondromalacic human cartilage. An ex vivo comparison of bipolar and monopolar radiofrequency devices. Am J Sports Med 30:90–97

103. Encalada I, Richmond JC (2004) Osteonecrosis after arthroscopic meniscectomy using radiofrequency. Arthroscopy 20(6): 632–636
104. Robertson DD, Armfield DR, Towers JD, Harner CD (2009) Meniscal root injury and spontaneous osteonecrosis of the knee. An observation. J Bone Joint Surg Br 91(2):190–195
105. Scott GA, Jolly BL, Henning CE (1986) Combined posterior incision and arthroscopic intra-articular repair of the meniscus. An examination of factors affecting healing. J Bone Joint Surg Am 68:847–861
106. Cannon WD Jr (1996) Arthroscopic meniscal repair. Inside-out technique and results. Am J Knee Surg 3:137–143
107. Cannon WD, Morgan CD (1994) Meniscal repair: arthroscopic repair techniques. J Bone Joint Surg Am 76:294
108. Warren RF (1995) Arthroscopic meniscus repair. Arthroscopy 1:170–172
109. Morgan CD (1991) The "all-inside" meniscus repair. Arthroscopy 7:120–125
110. Albrecht-Olsen P, Kristensen G, Tormala P (1993) Meniscus bucket-handle fixation with an absorbable Biofix tack: development of a new technique. Knee Surg Sports Traumatol Arthrosc 1:104
111. Small NC (1990) Complications in arthroscopic meniscal surgery. Clin Sports Med 9:609–617
112. Coen MJ, Caborn DN, Urban W, Nyland J, Johnson DL (1999) An anatomic evaluation of T-Fix suture device placement for arthroscopic all-inside meniscal repair. Arthroscopy 15:275–280
113. Espejo-Baena A, Golano P, Meschian S, Garcia-Herrera JM, Serrano Jiménez JM (2007) Complications in medial meniscus suture: a cadaveric study. Knee Surg Sports Traumatol Arthrosc 15:811–816
114. Seil R, Rupp S, Dienst M, Mueller B, Bonkhoff H, Kohn DM (2000) Chondral lesions after arthroscopic meniscus repair using meniscus arrows. Arthroscopy 16:E17
115. West RV, Kim JG, Armfield D, Harner CD (2004) Lateral meniscal root tears associated with anterior cruciate ligament injury: classification and management (SS-70). Arthroscopy 20:e32–e33 (Abstr)
116. Raustol OA, Poelstra KA, Chhabra A, Diduch DR (2006) The meniscal ossicle revisited: etiology and an arthroscopic technique for treatment. Arthroscopy 22:687.e1–687.e3
117. Engelsohn E, Umans H, DiFelice G (2007) Marginal fractures of the medial tibial plateau: possible association with medial meniscal root tear. Skeletal Radiol 36:73–76
118. Perdue PS, Hummer CD, Colosimo AJ (1996) Meniscal repair: outcomes and clinical follow-up. Arthroscopy 12:694
119. Stärke C, Kopf S, Petersen W, Becker R (2009) Meniscal repair. Arthroscopy 25(9):1033–1044
120. Benazzo F, Zanon G (2010) Meniscal sutures. Tech Knee Surg 9(3):159–164
121. Zhang ZN, Tu KY, Xu YK, Zhang WM, Liu ZT, Ou SH (1988) Treatment of longitudinal injuries in avascular area of meniscus in dogs by trephination. Arthroscopy 4:151–159
122. Zhang Z, Arnold JA (1996) Trephination and suturing of avascular meniscal tears: a clinical study of the trephination procedure. Arthroscopy 12:726–731
123. Barber FA (1994) Unrestricted rehabilitation of meniscus repairs. Arthroscopy 10:353

Lateral Meniscus

38

Giovanni Bonaspetti, Giovanni Battista Vinanti,
Flavio Azzola, Alessandro Corsini, and Massimo Petrera

38.1 Anatomy

The menisci, also known as semilunar cartilages, are wedge-shaped fibrocartilage structures. Both the medial (MM) and the lateral (LM) meniscus assume their shapes early in the prenatal development [1] and are identifiable around the seventh/eighth gestation week [2].

Adult LM is approximately 32.4–35.7 mm in length and 26.6–29.3 mm wide, while the MM is 40.5–45.5 mm long and 27 mm wide [3]. LM displays greater variability in size, shape, and thickness compared to the MM. Moreover, the LM has a more uniform width and a more circular shape than the MM, and it covers 70 % of the lateral tibial plateau while the MM about only 50 %.

The intermeniscal ligament connects the anterior horn of the LM and MM and can be noted on MR images in 44–58 % of individuals [4]. It has been described that some of the anterior cruciate ligament (ACL) fibers also extend into the anterior horn of the LM at the anterior root attachment. The posterior horn is connected to the posterior cruciate ligament (PCL) and the medial femoral condyle through the meniscofemoral ligament of Wrisberg (posteriorly) and Humphrey (anteriorly).

The LM is also linked to the popliteus tendon [5]. During flexion and internal rotation movements, the popliteal tendon retracts the posterior horn, reducing the risk of entrapment of the LM between the femur and the tibia. Overall, the LM results more mobile than the MM; the lack of attachment to the lateral collateral ligament also contributes to the increased mobility.

G. Bonaspetti • F. Azzola
2nd Orthopaedic division, Sant'Anna Clinical Institute,
Via del Franzone 31, Brescia 25127, Italy
e-mail: giovannibonaspetti@gmail.com; flavio.azzola@gmail.com

G.B. Vinanti • A. Corsini (✉) • M. Petrera
3rd Orthopaedic division, San Rocco Clinical Institute,
Via dei Sabbioni 24, Ome (BS) 25050, Italy
e-mail: gbvinanti@tin.it; dottor@alessandrocorsini.it;
massimopetrera@gmail.com

The blood supply of the LM originates from the lateral superior and inferior genicular arteries that reach the meniscus from the synovial membrane. Similarly to the MM, the blood supply is higher in the peripheral one third of the meniscus. Interestingly, beginning at birth, there is a progressive decrease of blood supply proceeding from the inner to the outer regions. At the age of 10, some vessels are still present in the inner zones, but in the adult meniscus, the inner two thirds are avascular [6].

The microanatomy of the LM is similar to the MM: a network of type I collagen fibers is arranged in a circumferential direction, while another network of radially oriented fibers, extending from the capsule, restrain the motion between the circumferential fibers, preventing splitting from longitudinally directed stress forces [7].

From a biomechanical standpoint, the menisci are dynamic structures, and their integrity is crucial for the kinematics of the knee joint. The fibrocartilage of the meniscus has the primary function of absorbing mechanical stress (about 50 % of the load is transmitted to the menisci) and contributes to the intrinsic stability of the knee. It has been reported that when the knee is flexed beyond 90°, about 90 % of the load is transmitted to the posterior horn of the LM and MM [8]. Lastly, it should be also highlighted that the fibrocartilage of the meniscus has an intrinsic proprioceptive function [9, 10].

38.1.1 Lateral Meniscal Variants: The Discoid Meniscus

The discoid meniscus is a morphologic abnormality first described by Young in 1889 [11] occurring almost exclusively on the lateral side. The prevalence among the patients undergoing knee arthroscopy is reported to be 0.4–20 %. Both genders are equally affected. Asian population has the highest reported rate, while the reported incidence rate in the American population ranges between 3 and 6 % [12].

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_38,

In 1978, Watanabe et al. [13] proposed a classification system that is still commonly used. The authors described three different types based on the shape and attachments of the LM:

- Complete discoid: disk-shaped meniscus with a thin central portion covering the tibial plateau entirely
- Incomplete discoid: semilunar-shaped meniscus with partial tibial plateau coverage
- Wrisberg type: hypermobile meniscus resulting from deficient posterior attachment and lack of the posterior tibio-meniscal ligaments (the only attachment posteriorly is the posterior meniscofemoral ligament)

However, although this classification system is widely utilized, recent studies [14] have shown significant variability in discoid meniscal size, shape, stability, and location of peripheral attachment. The prevalence of a "true" Wrisberg-type meniscus has been reported to range from 0 to 33 % [15]. Other studies [16, 17] reported of unstable discoid meniscal variations that cannot be included in the traditional classification.

Klingele et al. [18] have recently proposed a simplified classification scheme based on three factors: shape (complete or incomplete discoid), stability (stable or unstable due to lack of posterior attachments), and presence of a tear. This scheme seems to be helpful when planning the surgical treatment.

Usually, discoid LMs are thicker and have poorer vascularity compared to normal LM. In addition, they have a high prevalence of instability due to lack of peripheral attachments. Klingele et al. studied the prevalence and type of peripheral instability reporting peripheral instability in 28.1 % of discoid LM in children. The instability was more common in complete discoid menisci (38 %), and the anterior detachment was more commonly seen than middle or posterior detachment (47.2 % vs. 11.1 % and 38.9 %, respectively).

Papadopoulos et al. [19] recently demonstrated significant disorganization of the circumferential collagen network in the discoid meniscus and postulated that this disorganization might weaken the ultrastructure of the discoid meniscus.

The abovementioned factors seem to increase the risk of tearing as a result of mechanical and shear forces in the tibiofemoral joint [20, 21].

38.2 Injury Epidemiology

Sports injuries are twice more frequent than car accidents, and the 32.6 % of these injuries affect the knee [22].

Majewski et al. [23] have documented over 19,530 sports injuries during a period of 10 years; meniscal injuries were 14.5 % (MM 10.8 %, LM 3.7 %) and were the second cause of surgery with an incidence of 61/100.000.

Meniscal injuries have a higher incidence rate in soccer, volleyball, basketball, and baseball [24–26]. Yeh et al. [27], in a study on National Basketball Association (NBA) athletes including the injury reports of 21 seasons, reported that injuries of the LM had a stronger correlation with the number of games played compared to injuries of the MM. The authors also reported a significant correlation between age and injury rate: LM injuries were seen more frequently in younger athletes, while MM injuries were more common in older players.

Terzidis et al. [28], in a study group of 378 patients with an isolated LM meniscal tear who had undergone surgery, reported an incidence of 32.7 % of radial and 25.8 % of horizontal tears. The 91.4 % of tears were in the inner zone and more frequently in the body (61.2 %). Isolated tear of horns were less frequent, but the anterior horn of the LM was more frequently involved than the anterior horn of the MM (16 % vs. 2 %) [29].

One third of meniscal lesions are associated with an ACL injury. While MM tears seem to be correlated with chronic ACL deficiency, LM tear are more commonly seen in the setting of an acute trauma. Interestingly, Slauterbeck et al. [30], in a group of patients with an ACL-deficient knee, found 65 % of meniscal lesions: 52 % were isolated tears of the LM, 22 % were isolated tears of the MM, and 26 % were concomitant lesions of the MM and LM.

In recent years, there has been a growing interest toward meniscal root tears because of the concerns that this condition dramatically inhibits the normal meniscal function, leading to a condition biomechanically similar to a total meniscectomy. This condition includes pure meniscal root avulsion from the tibial plateau and a radial tear adjacent to the meniscal root. Meniscal root tears are generally infrequent, accounting for approximately 10 % of all arthroscopic meniscectomies [31]. In a recent case series of 388 ACL reconstruction surgeries, it was found that 27 cases (6.95 %) had a concomitant posterior LM root tear. Posterior LM root tears are classified based on arthroscopic findings in type I (oblique flap), type II (T shape), type III (longitudinal cleavage), and type IV (chronic inner loss) [32].

38.3 Clinical and Diagnostic Examination

38.3.1 Injury Mechanism in Meniscus Pathology

Meniscal injuries, particularly sports-related injuries, are usually caused by rotational forces. A hyperflexion of the knee (i.e., squatting) can sometimes be the cause of the tear. The most common traumatic mechanism combines a valgus or varus force applied to a flexed knee with the foot planted. In particular, a varus force applied on a flexed knee with the foot planted and the femur externally rotated may lead to an LM tear.

Sometimes a direct contact with another player might induce this abnormal motion on the player's knee, but most often an indirect trauma (twisting motion) is responsible of the tear.

On the other hand, degenerative meniscal lesions often occur in the absence of a traumatic event but are the result of a "wear and tear" process.

38.3.2 History

Obtaining an accurate and detailed history is the first critical step in patient's evaluation. History taking should include information about the onset of symptoms (acute vs. chronic), quality and severity of pain (dull vs. sharp, constant vs. intermittent, mild vs. severe), location (joint line, posterior), contributing factors (activity related, squatting), etiology and mechanism of injury, associated injuries, and previous treatments. Age, type of work, activity level, and sports participation should also be investigated.

In traumatic meniscal tears, patients typically recall an acute onset of pain following a twisting injury or deep flexion event. Joint line pain is commonly reported. A typical history in traumatic meniscal tears includes effusion developing immediately after the injury and subsequently being activity related. Limitation in sports and daily activities (squatting, doing stairs) is often reported. Presence of mechanical symptoms (locking, clicking, catching) should be investigated, with locking suggestive of a bucket-handle tear. Episodes of giving way are sometimes reported but are often related to associated knee disorders, such as instability or quadriceps weakness.

A longer history of pain, swelling, and mechanical symptoms is suggestive of degenerative meniscal pathology. These symptoms, most often in middle-aged patients, are usually intermittent and activity related.

Patients with a discoid meniscus have a highly variable presentation. In pediatric patients and adolescents, pain on the lateral joint line is the commonest complaint and is often associated to clicking over the lateral side. Recurrent effusions and episodes of locking may also be present. Other younger patients report of a painless knee clunk and lack of terminal knee extension. Discoid meniscus is usually asymptomatic in adults, unless a tear occurs.

38.3.3 Physical Examination

Patient's walking should be observed looking for limping or antalgic gait. Patients with a locked meniscal tear will present with a flexed knee. In standing position, the alignment (neutral, varus, or valgus) should also be evaluated.

With the patient supine, a careful examination of the knee should be performed looking for concomitant injuries as well. Signs suggestive of a meniscal tear are effusion, joint line tenderness, and pain on deep flexion. Often a painful clicking and loss of range of motion (ROM) are present. A reproducible clicking at 110° flexion is often noted in patients with a discoid meniscus. Lack of extension is typically seen in patients with a locked knee due to a displaced fragment in the joint (bucket-handle tear) or in patients with an unstable discoid meniscus.

Special tests (McMurray, Apley, and Thessaly) are helpful in confirming the clinical diagnosis. The McMurray test is performed with the patient supine. With the knee in maximal flexion, one hand palpates the joint line while the other hand holds the heel; the knee is slowly brought into extension while a rotation stress is applied (internal rotation stress tests the LM, external rotation tests the MM). The test is positive when pain in the appropriate joint line accompanied by a click is elicited.

The Apley's compression test is performed with the patient in prone position. The knee is flexed to 90° and axial force is applied through the heel along the tibia, while the tibia is internally and externally rotated. The test is considered positive when elicits pain.

The Thessaly test [33] is a dynamic test performed with the patient standing. With the examiner holding the patient's outstretched hands, the patient is asked to internally and externally rotate the knee, while keeping the knee in slight flexion (5°). The same maneuver is repeated with the knee flexed at 20°. Test is positive when patients report of medial or lateral joint line pain, with sometimes associated clicking or catching. The test should always be performed first on the normal knee so that the patient understands and performs correctly the procedure.

Finally, a complete knee stability evaluation (Lachman, anterior drawer, pivot shift, posterior drawer, sag sign, varus-valgus stress) should be performed to rule out associated ligament injuries.

38.3.4 Imaging

Although not useful for the diagnosis of meniscal tears, standard x-rays, including weight-bearing anteroposterior view, 45° flexion posteroanterior view, lateral view, and Merchant view, should be ordered to rule out concomitant pathologies, such as osteochondral lesions, arthritis, or fracture (i.e., Segond fracture) in the setting of a traumatic onset.

Magnetic resonance imaging (MRI) is a highly accurate imaging method in detecting meniscal tears. It allows determining meniscal morphology (normal and variants), the location of the tear (anterior horn, posterior horn, body), the tear pattern (horizontal, longitudinal, radial, complex,

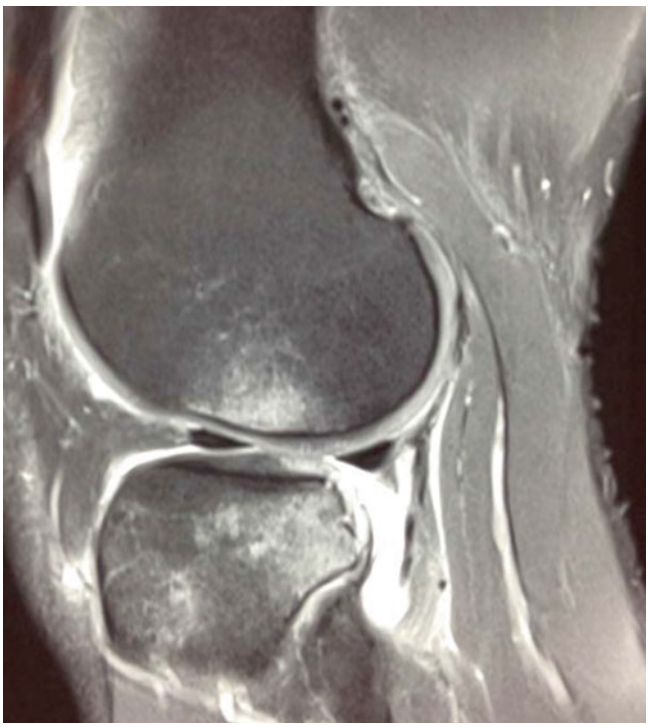


Fig. 38.1 Lateral meniscal root tear (MRI sagittal view)

Table 38.1 MRI classification of meniscal tear (Stoller et al. [35])

Grade	Pattern
Grade 0	Normal homogeneous low signal intensity
Grade 1	Nonarticular focal or globular intrasubstance increased signal
Grade 2	Linear intrasubstance increased signal, which does not involve articular meniscal surface
Grade 3	Increased signal intensity communicating at least with one articular meniscal surface

menisco-capsular separation), as well as concomitant chondral or ligamentous injuries.

The sensitivity of the MRI in detecting LM tears is 80 % versus 93 % for the MM. The sensitivity is even lower in detecting avulsions of the posterior root of the meniscus (root lesions) (Fig. 38.1), which most often are diagnosed at the time of arthroscopy. The percentage of false negative statistically increases when associated lesions, in particular ACL tears, are involved [34].

Stoller et al. [35] proposed an MRI classification of meniscal tears based on signal intensity and extension of the articular surface (see Table 38.1).

A complete discoid meniscus can be easily recognized on MR images because of its disk-like configuration. On the other hand, the recognition of an incomplete discoid meniscus may be more difficult. Typically, it involves only the posterior or anterior horn of the meniscus, and a trapezoidal appearance is seen.

A linear-increased signal communicating with the articular surface of a discoid meniscus is highly suggestive of meniscal tear.

38.4 Therapeutic Approach

The increased knowledge of the anatomical and functional importance of the menisci has radically modified the therapeutic approach to meniscal lesions.

Time has long passed since McMurray affirmed "a common error is the incomplete removal of the injured meniscus." This was based on the theory that the residual meniscus had a role in promoting the progression of knee arthropathy [36].

The modern approach to meniscal injuries in athletes and active patients is based on studies that have extensively demonstrated the negative impact that meniscectomy has on the articular cartilage, in particular in the lateral side of the knee. In a study with a 13-year follow-up, a 40 % reduction in the lateral joint line was seen after meniscectomy, while it was 28 % for the medial joint line [37]. This is related to the different anatomy and joint congruence between the lateral and the medial side of the knee.

Studies on biomechanical changes arising in the knees after a meniscectomy show that there is a 235 % increase of loads on the articular cartilage in the lateral side and 75 % in the medial side, with consequent biochemical and structural modifications in the cartilage tissue [38]. A published study reported that at a 30-year follow-up, 80 % of subjects who had undergone a total medial meniscectomy were satisfied with the outcomes of the procedure versus 47 % of subjects who had undergone a lateral meniscectomy [39].

Athletes are statistically more at risk to develop degenerative changes following meniscal lesions and consequent meniscectomy. The incidence of arthritis after meniscectomy is 15 % in professional soccer players versus 4 % in nonprofessional, while this rate is only 1.6 % in individuals who do not participate in sports. X-rays show signs of early arthritis already after 4.5 years. Arthritic changes are more evident after 14.5 years in 89 % of the athletes, with 46 % of them abandoning competitions due to pain [40–42]. Poor outcomes after meniscectomy are seen especially in professional volleyball players [43].

Therefore, based on the evidence available in the international literature, the surgical treatment of meniscal lesions in athletes has become less radical. Meniscal repair with the different suture techniques available is an option and is always taken into consideration in order to preserve meniscal function and prevent the degeneration of cartilage surfaces. However, when a meniscal repair is preferred, surgeons, athletes, and staff together should consider two fundamental aspects: the variable time frames in the rehabilitation pro-

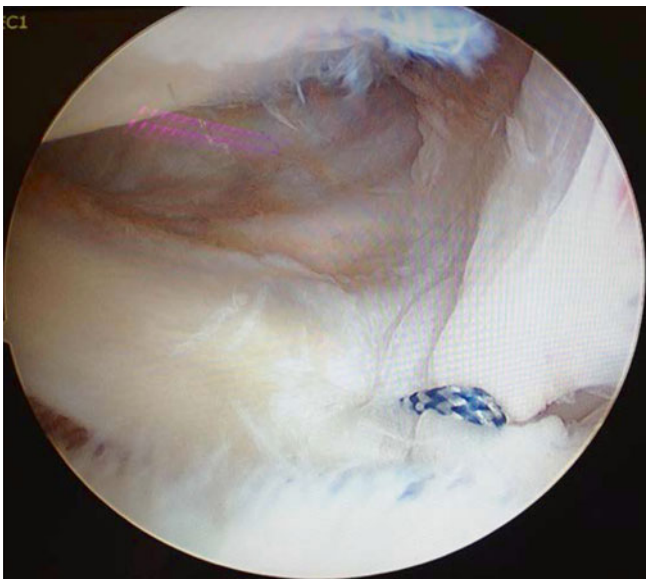


Fig. 38.2 Lateral meniscal root pull-out suture repair

gram/return to sport after the repair and the possibility of the failure of the repair with the risk of a second surgery.

The indication to treat a meniscal tear is based primarily on the patient history and physical examination. A torn meniscus that does not show a clinical pattern of pain and functional limitation may be treated conservatively. This decision is supported by the proven capacity of spontaneous healing in some types of meniscal lesions in the red-red zones (noncomplex longitudinal or vertical tears) [6, 44–46].

When dealing with a symptomatic traumatic LM tear in an athlete, the surgical treatment is indicated (Fig. 38.2). Acute LM lesions in the red-red or red-white zones may be treated with all-inside, inside-out, outside-in meniscal sutures, in relation to the morphology and site of the lesion. A meniscal suture is performed for a recent lesion in the red zone, in a stable knee, or when associated to an ACL reconstruction in a young and active individual. In the last decades, tissue-engineering approaches have been advocated to improve the reparative process. The use of growth factors seems in facts able to improve the meniscal healing [47, 48]; nevertheless, further studies are needed to evaluate the potential for clinical application.

Meniscal sutures, however, impose some postoperative restrictions that have to be accepted and respected by the athlete and the staff in order to reduce the risk of failure. In absence of a complete understanding and approval by the athlete and the team of the recovery time foreseen after surgery, the meniscal repair of the MM should not be taken into consideration; therefore, the only surgical option would be a meniscectomy.

In literature, the percentage of failures for meniscal repair ranges between 5 and 43 % (mean 15 %). Failures for MM repairs are statistically higher than those reported for the LM [49].

Several medium- and long-term follow-up studies analyzing the comparative results between partial meniscectomy and meniscal repair for traumatic lesions in athletes show that 96 % of the athletes who underwent a meniscal repair return to pre-injury activity level compared to 50 % of athletes who underwent meniscectomy [50].

In 2011, Paxton et al. published a literature review analyzing the long- and short-term outcomes of partial meniscectomy versus meniscal repair for acute meniscal tears. In the long term, 3.7 % of patients who had undergone partial meniscectomy had to undergo a second surgery; the incidence was greater for LM lesions compared to medial lesions. On the other side, the percentage of meniscal repair failures was 20.7 % with a higher rate reported for the MM compared to the LM. The long-term outcomes, in terms of functional recovery and x-ray findings, were better in individuals who underwent meniscal repair [51].

In selected cases, when athletes develop a "post-meniscectomy syndrome" secondary to a previous subtotal meniscectomy, a meniscal transplant with allograft (MAT: meniscus allograft transplantation) would be indicated with the aim to improve patients' symptoms and quality of life and prevent arthritis. A study by Cugat et al. analyzing the outcome of this procedure in 15 professional soccer players reported that 14 out of the 15 athletes were able to return to sports at a 36-month follow-up [52].

Conclusions

Based on the evidence from the available international literature, the treatment of meniscal lesions in athletes should be aimed to the preservation of the LM, favoring meniscal repair techniques. This would lead to a decreased risk of cartilage degeneration and progression toward osteoarthritis. On the other hand, it has also been shown that this approach ensures higher sport performance in the long term.

However, the athletes and the teams should be carefully informed and should understand that the meniscal repair is associated with a longer recovery time compared to a meniscectomy. Moreover, they should be aware of the possibility of a second surgery in the case of failure of the repair or if meniscal healing does not occur.

References

1. Clark CR, Ogden JA (1983) Development of the menisci of the human knee joint. Morphological changes and their potential role in childhood meniscal injury. J Bone Joint Surg Am 65: 538–547
2. Gardner E, O'Rahilly R (1968) The early development of the knee joint in staged human embryos. J Anat 102:289–299

3. Greis PE, Bardana DD, Holmstrom MC, Burks RT (2002) Meniscal injury: I. Basic science and evaluation. J Am Acad Orthop Surg 10:168–176
4. Aydingoz U, Kaya A, Atay OA et al (2002) MR imaging of the anterior intermeniscal ligament: classification according to insertion sites. Eur Radiol 12(4):824–829
5. Last RJ (1950) The popliteus muscle and the lateral meniscus. J Bone Joint Surg 32:93
6. Arnoczky SP, Warren RF (1982) Microvasculature of the human meniscus. Am J Sports Med 10:90–95
7. Rath E, Richmond JC (2000) The menisci: basic science and advances in treatment. Br J Sports Med 34(4):252–257
8. Maitra RS et al (1999) Meniscal reconstruction. Part I: indications, techniques, and graft consideration. Am J Orthop 28:213–218
9. Seedholm BB, Dowson D, Wright V (1974) Functions of the menisci: a preliminary study. J Bone Joint Surg 56-B:381–382
10. Jaureguito JW, Elliot JS, Lietner T et al (1995) The effects of arthroscopic partial lateral meniscectomy in an otherwise normal knee : a retrospective review of functional, clinical, and radiographic results. Arthroscopy 11:29–36
11. Young RB (1889) The external semilunar cartilage as a complete disc. In: Cleland J, Macke JY, Young RB (eds) Memoirs and memoranda in anatomy. Williams and Norgate, London, p 179
12. Carter C, Hoellwarth J, Weiis J (2012) Clinical outcomes as a function of meniscal stability in the discoid meniscus: a preliminary report. J Pediatr Orthop 32:9–14
13. Watanabe M, Takeda S, Ikeuchi H (1978) Atlas of arthroscopy. Igaku-Shoin, Tokyo, p 88
14. Jordan M (1996) Lateral meniscal variants: evaluation and treatment. J Am Acad Orthop Surg 2:239–253
15. Woods GW, Whelan JM (1990) Discoid meniscus. Clin Sports Med 9:695–706
16. Vandermeer RD, Cunningham FK (1989) Arthroscopic treatment of the discoid lateral meniscus: results of long term follow-up. Arthroscopy 5:101–109
17. Kobajashi A, Uezaki N, Mitsuyasu M (1975) Discoid meniscus of the knee joint. Clin Orthop Surg Japan 10:10–24
18. Klingele KE, Kocher MS, Hresko MT, Gerbino P, Micheli LJ (2004) Discoid lateral meniscus: prevalence of peripheral rim instability. J Pediatr Orthop 24:79–82
19. Papadopoulos A, Kirkos JM, Kapetanos GS (2009) Histomorphologic study of discoid meniscus. Arthroscopy 25:262–268
20. Jordan M, Duncan J, Bertrand S (1993) Discoid lateral meniscus: a review. South Orthop J 2:239–253
21. Pellacci F, Montanari G, Prosperi P, Galli G, Celli V (1992) Lateral discoid meniscus: treatment and results. Arthroscopy 8:526–530
22. Steinbruck K (1999) Epidemiology of sports injuries 25-year-analysis of sports orthopedic-traumatologic ambulatory care. Sportverletz Sportschaden 13:38–52
23. Majewski M, Habelt S, Klaus S (2006) Epidemiology of athletic knee injuries: a 10-year study. Knee 13(3):184–188
24. Muellner T, Weinstabl R, Schabus R et al (1997) The diagnosis of meniscal tears in athletes: a comparison of clinical and magnetic resonance imaging investigations. Am J Sports Med 25:7–12
25. Maffulli N, Binfield PM, King JB et al (1993) Acute haemarthrosis of the knee in athletes: a prospective study of 106 cases. J Bone Joint Surg Br 75:945–949
26. Binfield PM, Maffulli N, King JB (1993) Patterns of meniscal tears associated with anterior cruciate ligament lesions in athletes. Injury 24:557–561
27. Yeh PC, Starkey C, Lombardo S et al (2012) Epidemiology of isolated meniscal injury and its effect on performance in athletes from the National Basketball Association. Am J Sports Med 40: 589–594
28. Terzidis IP, Christodoulou A, Ploumis A et al (2006) Meniscal tear characteristics in young athletes with a stable knee: arthroscopic evaluation. Am J Sports Med 34:1170–1175
29. De Smet AA, Norris MA, Yandow DR et al (1993) MR diagnosis of meniscal tears of the knee: importance of high signal in the meniscus that extends to the surface. AJR Am J Roentgenol 161(1): 101–107
30. Slauterbeck JR, Kousa P, Clifton BC et al (2009) Geo- graphic mapping of meniscus and cartilage lesions associated with anterior cruciate ligament injuries. J Bone Joint Surg Am 91:2094–2103
31. Papalia R, Vasta S, Franceschi F, D'Adamio S, Maffulli N, Denaro V (2013) Meniscal root tears: from basic science to ultimate surgery. Br Med Bull 106:91–115
32. Ahn JH, Lee YS, Yoo JC, Chang MJ, Park SJ, Pae YR (2010) Results of arthroscopic all-inside repair for lateral meniscus root tear in patients undergoing concomitant anterior cruciate ligament reconstruction. Arthroscopy 26(1):67–75
33. Karachalios T, Hantes M, Zibis AH, Zachos V, Karantanas AH, Malizos KN (2005) Diagnostic accuracy of a new clinical test (the Thessaly test) for early detection of meniscal tears. J Bone Joint Surg Am 87(5):955–962
34. Laundre BJ, Collins MS, Bond JR, Dahm DL, Stuart MJ, Mandrekar JN (2009) MRI accuracy for tears of the posterior horn of the lateral meniscus in patients with acute anterior cruciate ligament injury and the clinical relevance of missed tears. AJR Am J Roentgenol 193:515–523
35. Stoller DW, Martin C, Crues JV 3rd, Kaplan L, Mink JH (1987) Meniscal tears: pathologic correlation with MR imaging. Radiology 163(3):731–735
36. McMurray TP (1942) The semilunar cartilages. Br J Surg 29:407–414
37. Beaufils P, Hardy P, Chambat P, Clavert P, Djian P, Frank A, Hulet C, Potel JF, Verdonk R, Société Française d'Arthroscopie (2006) Adult lateral meniscus. Rev Chir Orthop Reparatrice Appar Mot 92(5 Suppl):2S169–2S194
38. Baratz ME, Fu FH, Mengato R (1986) Meniscal tears: the effect of meniscectomy and of repair on intraarticular contact areas and stress in the human knee. Am J Sports Med 14:270–274
39. McNicholas MJ, Rowley DI, McGurty D et al (2000) Total meniscectomy in adolescence: a thirty-year follow-up. J Bone Joint Surg 82-B:217–221
40. McDermott ID, Amis AA (2006) The consequences of meniscectomy. J Bone Joint Surg 88-B:1549–1556
41. Roos H, Lindberg H, Gardsell P, Lohmander LS, Wingstrand H (1994) The prevalence of gonarthrosis and its relation to meniscectomy in former soccer players. Am J Sports Med 22:219–222
42. Jorgensen U, Sonne HS, Lauridsen F, Rosenklint A (1987) Long-term follow-up of meniscectomy in athletes: a prospective longitudinal study. J Bone Joint Surg 69-B:80–83
43. Hoshikawa Y, Kurosawa H, Fukubayashi T, Nakajima H, Watarai K (1983) The prognosis of meniscectomy in athletes: the simple meniscus lesions without ligamentous instabilities. Am J Sports Med 11:8–13
44. Dehaven KE, Arnoczky SP (1994) Meniscus repair. Part I: basic science, indications for repair, and open repair. J Bone Joint Surg Am 76-A:140–152
45. Weiss CB, Lundberg M, Hamberg P, Dehaven KE, Gillquist J (1989) Non-operative treatment of meniscal tears. J Bone Joint Surg Am 71-A:811–822
46. Goodfellow J (1980) He who hesitates is saved. J Bone Joint Surg 62-B:1–2
47. Scotti C, Pozzi A, Mangiavini L, Vitari F, Boschetti F, Domeneghini C, Fraschini G, Peretti GM (2009) Healing of meniscal tissue by cellular fibrin glue: an in vivo study. Knee Surg Sports Traumatol Arthrosc 17(6):645–651

48. Izuta Y, Ochi M, Adachi N, Deie M, Yamasaki T, Shinomiya R (2005) Meniscal repair using bone marrow-derived mesenchymal stem cells: experimental study using green fluorescent protein transgenic rats. Knee 12(3):217–223
49. Pujol N, Barbier O, Boisrenoult P, Beaufils P (2011) Amount of meniscal resection after failed meniscal repair. Am J Sports Med 39(8):1648–1652. Epub 2011 Apr 6
50. Stein T, Mehling AP, Welsch F, von Eisenhart-Rothe R, Jäger A (2010) Long-term outcome after arthroscopic meniscal repair versus arthroscopic partial meniscectomy for traumatic meniscal tears. Am J Sports Med 38(8):1542–1548
51. Paxton ES, Stock MV, Brophy RH (2011) Meniscal repair versus partial meniscectomy: a systematic review comparing reoperation rates and clinical outcomes. Arthroscopy 27(9):1275–1288
52. Alentorn-Geli E, Vázquez RS, Díaz PA, Cuscó X, Cugat R (2010) Arthroscopic meniscal transplants in soccer players: outcomes at 2- to 5-year follow-up. Clin J Sport Med 20(5): 340–343

Cartilage Injury Treatment

39

Mats Brittberg

39.1 Aetiology

Loss of cartilage function may lead to a painful joint with a decreased mobility. Several factors, i.e. epidemiological, biochemical and morphological, are associated with cartilage destruction; however, only trauma is known directly to cause osteoarthritis [1–5]. It is well known that once the cartilaginous tissue has been destroyed, the intrinsic reparative ability is poor; therefore, it is extremely important to increase knowledge about the cartilage, the tissue reaction to trauma and the intrinsic attempts to repair the defects as well as extrinsic methods.

Trauma to this viscoelastic unit can be either as:

- Direct contusion
- Rotational indirect shearing forces
- Gradual trauma on the tissues as seen after repeated microtrauma, instability and overweight

Meniscal injuries and unstable cruciate ligaments could induce pathological load on the surfaces influencing the joint homeostasis with cartilage loss as a result [2, 5].

39.1.1 Cartilage Layers and Injury Mechanism

Cartilage consists of four separate layers or zones [1]. The weakest part of the cartilage layer is the zone between the calcified layer and overlying cartilage. Due to such a weakness between the layers, the lesions often penetrate down to the calcified layer in adult joints making lesions in adults to partial-thickness lesions. In a juvenile joint, the calcified layer is poorly developed which results in deeper injuries often osteochondral injuries.

Most often the injuries are a combination of rotational and compressive forces.

It is important to differ between chondral and osteochondral injuries. The chondral injuries are more difficult to detect as they are not visible on normal x-rays, while osteochondral injuries may be noted due to its bony involvement.

39.2 Clinical and Diagnostic Examination

Articular cartilage injuries are common findings during arthroscopy and diagnostic imaging of the joints [6]. The examiner should interview the patient in order to collect the injury history. The patient's history is vital when to diagnose a knee injury.

Cartilage injuries give rise to symptoms with similarities of symptoms from meniscal tears, and differentiating between articular cartilage damage and meniscal tears can subsequently be difficult.

For the clinical examination, of importance are:

- Patient's trauma history or history of pain and disability development
- Occurrence of joint swelling
- Pain at rest + pain in motions with or without weight bearing
- Locking or catching of the joint
- Instability, "giving way" phenomena
- Earlier joint trauma
- Concomitant diseases

39.2.1 Imaging

39.2.1.1 Plain x-rays

Plain radiographs are useful for diagnosing severe cartilage injuries but are not of any help for detecting localised articular cartilage lesions of the knee. Attempts should be made to obtain all knee anteroposterior (AP) radiographs with the patient standing [7].

M. Brittberg, MD, PhD
Cartilage Research Unit (CRU), University of Gothenburg, Region Halland Orthopaedics (RHO), Kungsbacka Hospital, Kungsbacka S-434 80, Sweden
e-mail: Mats.brittberg@telia.com, Mats.brittberg@regionhalland.se

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_39,

The standard series includes a:

- Standing AP view.
- Lateral view with the knee flexed 35°.
- 45° patellar sunrise view.
- Lateral radiographs may indicate OCD or impaction fractures of the femoral condyles.
- If an area looks suspicious for OCD, an intercondylar notch (tunnel) view should also be obtained.

39.2.1.2 CT Arthrography

It is useful to study the extent of an osteochondral injury with the use of spiral CT 3D reconstructions. If one adds a CT arthrography, it is possible to evaluate the overlying cartilage layer and fissures and cracks on the surface [7].

39.2.1.3 MRI

MRI is a clinically useful, efficient and reliable technique for detecting cartilage defects and is also excellent for visualising menisci, ligaments, soft tissues and bones [8, 9].

MRI of articular cartilage lesions is best performed using high-magnetic-field-strength (1.5 or 3.0 Tesla) scanners. Higher field strength results in higher signal-to-noise ratio, enabling thinner slice and higher spatial resolution imaging, both of which are key factors for optimal articular cartilage visualisation.

Delayed gadolinium-enhanced MRI of cartilage (dGEMRIC) is an imaging technique used to evaluate articular joint cartilage glycosaminoglycan content and of less interest detecting cartilage damage after trauma [10]. Similar situation is with measurement of cartilage T2 (T2 mapping) which detects early signs of structural changes of the extracellular matrix and monitors these changes over time [10].

With magnetisation transfer (MT), it is possible to get quantitative and qualitative information on the collagen status of the damaged cartilage, and the technique may be applied for the routine monitoring of normal and abnormal articular cartilage (Fig. 39.1) [11].

39.2.1.4 Technetium Scintigraphy

In chronic medial knee pain, increased tracer uptake in bone scintigraphy is more sensitive for medial knee pain than bone marrow oedema pattern on MRI. Scintigraphic examination could be used when patients after trauma do not show any significant injury on MRI while still in considerable pain [12, 13].

Single-photon emission computed tomography (SPECT) [14] could be used to assess the physiology and homeostasis of subchondral bone adjacent to untreated and treated articular cartilage defects.

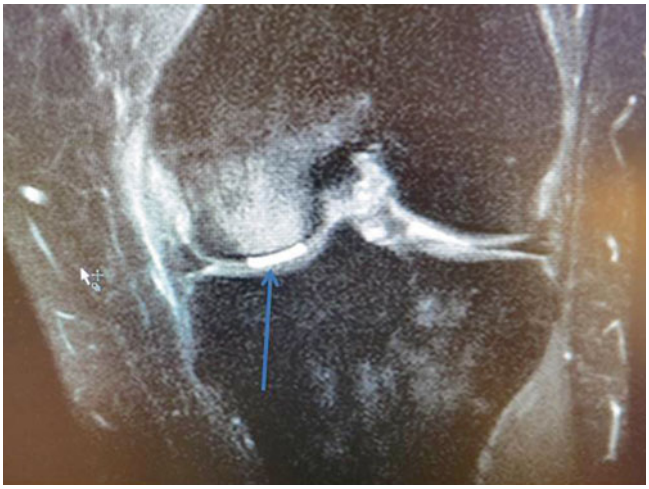


Fig. 39.1 An MRI on a left knee showing a cartilage lesion on the medial femoral condyle. See *arrow*

39.3 Operative Interventions

39.3.1 Arthroscopy

Despite significantly improved detection with MR, still arthroscopy evaluation is the gold standard.

However, even arthroscopy has its limitations [15].

When we have a patient with disturbed joint function and pain and is planning an arthroscopy, first of all, we need to classify the lesions to:

1. Location and grade (use the ICRS classification system [16])
2. Size
3. Morphology/character

Furthermore, the surgeon should try to find out the aetiology of the lesion. Is it a lesion due to trauma and is it acute or and elderly lesion, chronic lesion? The cartilage lesions should also be evaluated in relation to concomitant injuries such as ligament injuries, meniscal damage and sinusitis.

Use a standard arthroscopic probe to examine the joint surface to determine cartilage quality. The surgeon needs to carefully palpate fissures and tissue surrounding defect to determine integrity of surrounding cartilage. Using a graduated probe, measure the anterior-posterior and medial-lateral dimensions of each defect.

To decide what kind of treatment to choose, the surgeon needs to estimate:

1. Patients' age and activity level
2. The degree of pain and disability that the patients are experiencing

3. Location of cartilage lesions and the size and depth of cartilage lesions
4. Coexisting joint pathology such as loss of meniscus, ligament insufficiency, bone loss and malalignment
5. Other concomitant diseases

Furthermore, the following must be considered:

1. Body weight or body mass index (BMI).
2. Demand and functional need.
3. Expectation.
4. Ability to comply with rehabilitation.
5. An increased BMI (greater than 30) may have an adverse effect on some cartilage repair procedures [17].
6. Smoking may impair cartilage repair processes [18, 19].

Typically, the patients to treat are those with symptoms of pain, swelling and catching/locking and with the following appearances [16, 20]:

- Isolated cartilage defects ICRS grades 2–4 > 1 cm^2 on weight-bearing surfaces
- Cartilage defects with concentrated high uptake of technetium at the lesion size or similar with long-standing concentrated bone marrow oedema

Related to studies, early treatment of those lesions might be important for a more successful outcome.

- ICRS grade 1 lesions are superficial fissures and cracks and need no treatment.
- ICRS grade 2 lesions down to less than 50 % cartilage depth are often unstable, with partly detached fragments that need to be debrided to form stable lesions. The prognosis for ICRS-2 partial-thickness lesions seems good with diminished mechanical symptoms following a simple debridement that involves excision of the unstable cartilage fragments back to smooth edges and leaves the base intact [16, 20].
- In the literature, the deep to bare-bone lesions seem troublesome [21]. Lesions that extend through >50 % of the cartilage thickness are classified as ICRS-3 a–d. While debridement of unstable edges (as is suggested for ICRS-2 lesions) is suitable for ICRS-3 lesions, further treatment is recommended for these more extensive lesions [20].

Osteochondral ICRS-4 lesions can be treated in the same manner as described for ICRS-3 lesions, but a lesion with extensive extensions into the bone may require bone grafting [20, 22].

39.3.2 Cartilage Defects with Concomitant Joint Injuries

Cartilage lesions that are found together with other injuries have to be related to the severity of the other injury/injuries. If the cartilage lesions are suspected to be part of the symptomatology, they are treated the same way as the isolated lesions.

If instability is the major symptom from an ACL-deficient knee with small- to medium-size cartilage lesion without subchondral reaction, such a lesion may be left untreated.

If, however, the joint besides the ACl injury also has a major loss of the menisci and a cartilage lesion with instability and pain, the evaluation and decision is more difficult. Without damaged cartilage, such a knee joint might function well without ACL reconstruction and meniscal grafting. Instead, with all three areas destroyed, the joint is in danger to develop into OA. In such a situation, the cartilage lesion treatment may need to be supported by the meniscal and ACL grafting at the same time for a maximal protection of injured articular joint [23, 24].

39.4 Treatment Strategy

The prevalence of focal articular cartilage lesions among athletes is higher than in the general population [25]. Furthermore, the treatment goals differ considerably between the professional and recreational athletes. The high costs for the sports activities and involved professional clubs and the short duration of a professional career influence the treatment selection for the professional athlete with less influence in recreational sports.

Treatment goals also differ between recreational and professional sports players. Recreational players mostly hope for a relief of pain, return of functionality and, if possible, some sports participation, while professional players need a fast return to their previous, high-demanding, activity level without any delay. In the literature of reported results, ACI and osteochondral autografts seem to lead to a better structural tissue repair, and such a repair would be of interest to be able to perform at top again after trauma [26–28].

But in spite of such facts, ACI and OAT are not the treatments of first choice among professional athletes.

39.4.1 The Operative Alternatives

39.4.1.1 Microfracture and Other Bone Marrow Stimulation Techniques

Due to the long rehabilitation time after ACI, microfracture is most often considered the first treatment option among professional sportsmen. In studies, microfracture has shown a statistically significant improvement from baseline in functional outcome, pain scores and Tegner activity levels after at least 1-year follow-up among professional football (soccer)

players and other athletes [29]. Furthermore, the technique is fairly easy to use and cheap. Microfracture can also be used in combination with scaffold augmentations such as variants of AMIC (membrane protecting the microfractured area) [30] and scaffolds for cell ingrowth like Hyalofast [31] and blood clot augmentations (BST-CarGel) [32]. Different synthetic porous cylindrical grafts [33–35], biomimetic multilayer grafts [36] and BMAC (bone marrow aspirate concentrates) [37] are also possible but less useable for high-level sportsman.

39.4.1.2 Osteochondral Grafts

The osteochondral autograft transfer technique (OAT) showed superior clinical results compared to microfracture in a randomised study among both professional and recreational athletes [38]. Also, prospective case series show good clinical results up to 17-year follow-up in a mixed athletic population [39]. To use osteochondral grafts (OAT), having a shorter mean rehabilitation time compared to both microfracture and ACI could also be an alternative instead of microfracture when a short rehabilitation time is of interest. The technique is fairly cheap but may be difficult to use transarthroscopically as not all lesions reachable by microfracture could be treated by OAT.

Osteochondral allografts are normally not indicated for a sportsman to return to his/her earlier level. However, new technologies like DeNovo NT with allograft particulate cartilage transplantation [40] with similarities to below-mentioned CAIS [40, 41] might be of interest for the sportsmen.

39.4.1.3 ACI

ACI showed an improvement from baseline in functional outcome and pain scores and an even higher good to excellent treatment success in professional football (soccer) players and adolescent athletes compared to microfracture (66–83 % for microfracture vs. 72–95 % for ACI). Also, Tegner activity levels were significantly higher after ACI in football (soccer) players [42, 43]. The negative part for the athlete is the long rehab time needed.

If ACI is the treatment of first choice, a surgical debridement of injured cartilage area with an additional cartilage biopsy may be indicated to relieve symptoms during the ongoing series. The implantation may then be planned after the patient's wishes and related to the seasons (Fig. 39.2).

An interesting alternative is the fourth-generation ACI with fragmented cartilage implanted under a resorbable membrane (CAIS). The technique can be done in one stage, but the rehab time is equal compared to first- to third-generation ACIs [40, 41].

Mithoefer et al. showed that the average time to return to professional sports is highest after ACI (18 ± 4 months; range, 12–36 months) compared to microfracture (8 ± 1 months; range, 2–16 months) and OAT (7 ± 2 months; range, 4–11 months) [26].

39.4.2 Non-biological Local Repairs

Mini metal arthroplasties (MMA) with small custom-made metal implants [44, 45] treatment place in the sportsmen is also difficult to tell. It might be a solution for the recreational sportsmen, but more and longer follow-ups are needed to be able to give advice.

39.4.3 General Comments

For professional football (soccer) players and other high-level athletes with symptoms <12 months, Mithoefer et al. found a better clinical outcome and greater return to sports after microfracture as well as ACI [42, 43].

Also, when treated with OAT, the chronicity of the lesion seemed important on clinical outcome and return to sports.

From the sportsman's point of view, microfracture or variants of transarthroscopic bone marrow stimulation techniques are reasonable choices as they are safe and simple techniques and allow careers to continue for some more years. During the operative procedure, it is important to fast stabilise the fragmented cartilage defect. A cartilage biopsy may be taken for a future ACI.

The other alternative is an osteochondral graft related to the faster rehab time compared to microfracture. However, it is important to remember that with focal lesions >2 cm^2, microfracture and OAT showed significantly worse clinical outcomes and a lower return to high-level sports when compared to lesions <2 cm^2 [28].

In contrast, with ACI, no influence on lesion size and clinical outcome or return to sports participation has been found, which indicates that for larger lesions, ACI is the treatment option of first choice for both professional and recreational sports athletes [28].

39.5 Rehabilitation and Return to Play

A return to the preinjury sports activity level is much more important when dealing with high-level athletes compared to the recreational sportsmen. However, in today's community more and more people are active at higher and higher ages. The activities for the recreational people are also more demanding, meaning that also recreational sportsmen have high demands on their joint restoration.

Also it is important to remember that Mithoefer et al. showed a decline in sports performance and participation, after full rehabilitation, for microfracture-treated athletes in 47 %, while at least 87 % of ACI-treated professional football

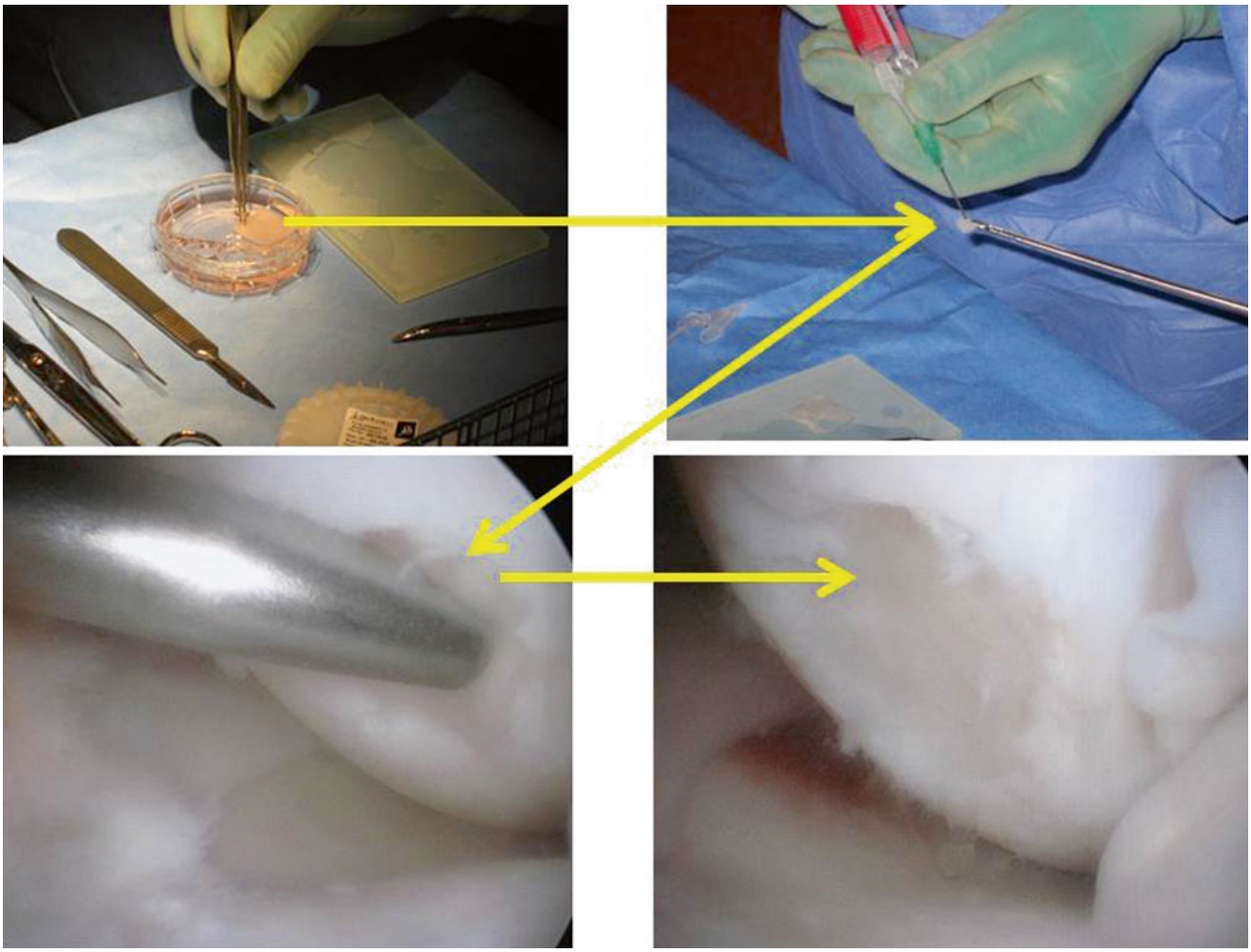


Fig. 39.2 The cartilage lesion in Fig. 39.1 is treated by transarthroscopic ACI with hyaluronan cell-seeded scaffold

(soccer) players remained at their previous sports level after rehabilitation for an average of 52 months [29, 43].

A longer rehab in time may then subsequently be worthwhile to sustain knowing that you will have a better chance to remain at your previous level.

39.5.1 The Rehabilitation Process

The concept of a slow gradual time course of healing is critical to understand for the rehabilitation following cartilage repair. If the intra-articular environment is protective, the maturation of this tissue, an ongoing process of remodelling of the tissue, will continue. However, if the repaired area is overloaded, failure can occur.

There is a degree of individual variation with the rehabilitation process, so the programme needs to be designed according to:

- The patient's status and needs
- The size and location of the lesion
- Any possible concomitant procedures performed

It is critical that there is regular contact between the physician, patient and therapist, especially during at least the first 24 months following a cartilage repair process.

In the early steps after the repair, it is important to avoid twisting rotational shearing forces. Introduced also in the beginning are:

- Continuous passive motion and a gradually increased weight bearing
- Isometric quadriceps training
- Straight leg raises
- Hamstring strengthening

These training components are progressively advanced to resistance exercises and return to greater degrees of functional activities.

From 3 weeks post-op:

- Start progressive closed-chain exercises with light resistance.

From around 8 weeks:

- Open-chain exercises can be initiated.

Running is not advised until the 8th or 9th month post ACI with high-level activities being initiated at 12th month. Such activities may be started at ca 4–6 months post microfracture treatment in small loaded defects <1 cm^2 but the same restriction for all repair techniques when lesions are larger than 2 cm^2.

Rehabilitation for patellar or trochlear lesions requires special considerations to still obtain early motions while protecting the forces across the repair tissue. Contact pressure of the patellofemoral articulation is maximised between 40° and 70° of knee flexion and thus should be avoided during active knee flexion until the graft is mature enough to withstand these shear stresses. Exercises to promote patella mobility should be initiated early to help prevent adhesions.

Passive motion with CPM or by using the contralateral leg to extend the involved leg is allowed and encouraged. The gradual progression of active extension exercises depends on the size and location of the defect as observed in the operating room; therefore, it is essential that the surgeon provide guidance and reassurance to the patient and the therapist. If the defects are large, one may consider using an unloader brace.

Most often the rehabilitation programmes try to follow the repair processes starting after the operation. Theoretically, there are four phases [46]:

- The proliferative phase
- The transitional phase
- The remodelling phase
- The maturation phase

The surgeon and the physiotherapist need to adjust the postoperative mobilisation after those phases in relation also to the size and location of the lesion, the patient's weight and type of surgery.

Traditionally, most surgeons are using non-weight bearing from start after surgery. The last years, however, the trend is towards more aggressive mobilisation with gradual fast partial weight bearing during ca 6 weeks.

My own rehab for all cartilage repairs is full weight bearing controlled by the level of pain and two crutches. A brace locked in extension is used for 2 weeks and for another 4 weeks an open side stabilisation brace just outdoors. Reinold et al. [47] have a very nice paper on current concepts for cartilage repair that can be read by those wanting a more deep knowledge.

References

1. Hall BK (2005) Bones and cartilage. Chapter 3. Cartilage. In: Hall BK (ed) Developmental and evolutionary skeletal biology. Elsevier Academic Press, San Diego, pp 33–47
2. Anderson DD, Chubinskaya S, Guilak F, Martin JA, Oegema TR, Olson SA, Buckwalter JA (2011) Post-traumatic osteoarthritis: improved understanding and opportunities for early intervention. J Orthop Res 29(6):802–809. doi:10.1002/jor.21359. Epub 2011 Feb 11. Review
3. Buckwalter JA, Mankin HJ, Grodzinsky AJ (2005) Articular cartilage and osteoarthritis. Instr Course Lect 54:465–480. Review
4. Moskowitz RW (1973) Osteoarthritis: a new look at an old disease. Geriatrics 28(6):121–128. Review
5. Schenker ML, Mauck RL, Ahn J, Mehta S (2014) Pathogenesis and prevention of posttraumatic osteoarthritis after intra-articular fracture. J Am Acad Orthop Surg 22(1):20–28. Review
6. Hjelle K, Solheim E, Strand T, Muri R, Brittberg M (2002) Articular cartilage defects in 1,000 knee arthroscopies. Arthroscopy 18(7): 730–734
7. Brittberg M (2012) Which lesions should be treated and why ? In: Brittberg M, Gobbi A, Imhoff A, Kon E, Madry H (eds) Cartilage repair. Clinical guidelines. DJO Publications, London, pp 3–14
8. Jazrawi LM, Alaia MJ, Chang G, Fitzgerald EF, Recht MP (2011) Advances in magnetic resonance imaging of articular cartilage. J Am Acad Orthop Surg 19(7):420–429. Review
9. de Windt TS, Welsch GH, Brittberg M, Vonk LA, Marlovits S, Trattnig S, Saris DB (2013) Is magnetic resonance imaging reliable in predicting clinical outcome after articular cartilage repair of the knee? A systematic review and meta-analysis. Am J Sports Med 41(7):1695–1702. Review
10. Jungmann PM, Baum T, Bauer JS, Karampinos DC, Erdle B, Link TM, Li X, Trattnig S, Rummeny EJ, Woertler K, Welsch GH. Cartilage repair surgery: outcome evaluation by using noninvasive cartilage biomarkers based on quantitative MRI techniques? Biomed Res Int 2014;840170. [Epub 2014 May 4]
11. Li W, Hong L, Hu L, Magin RL (2010) Magnetization transfer imaging provides a quantitative measure of chondrogenic differentiation and tissue development. Tissue Eng Part C Methods 16(6):1407–1415
12. Kock NB, van Tankeren E, Oyen WJ, Wymenga AB, van Susante JL (2010) Bone scintigraphy after osteochondral autograft transplantation in the knee: 13 patients followed for 4 years. Acta Orthop 81(2):206–210
13. Omoumi P, Mercier GA, Lecouvet F, Simoni P, Vande Berg BC (2009) CT arthrography, MR arthrography, PET, and scintigraphy in osteoarthritis. Radiol Clin North Am 47(4):595–615. Review
14. Leumann A, Valderrabano V, Plaass C, Rasch H, Studler U, Hintermann B, Pagenstert GI (2011) A novel imaging method for osteochondral lesions of the talus–comparison of SPECT-CT with MRI. Am J Sports Med 39(5):1095–1101
15. Oakley SP, Portek I, Szomor Z, Turnbull A, Murrell GA, Kirkham BW, Lassere MN (2003) Accuracy and reliability of arthroscopic estimates of cartilage lesion size in a plastic knee simulation model. Arthroscopy 19(3):282–289
16. Brittberg M, Winalski CS (2003) Evaluation of cartilage injuries and repair. J Bone Joint Surg Am 85-A(Suppl 2):58–69
17. Eskelinen AP, Visuri T, Larni HM, Ritsilä V (2004) Primary cartilage lesions of the knee joint in young male adults. Overweight as a predisposing factor. An arthroscopic study. Scand J Surg 93(3):229–233
18. Ciccotti MC, Kraeutler MJ, Austin LS, Rangavajjula A, Zmistowski B, Cohen SB, Ciccotti MG (2012) The prevalence of articular cartilage changes in the knee joint in patients undergoing arthroscopy for meniscal pathology. Arthroscopy 28(10):1437–1444
19. Jaiswal PK, Macmull S, Bentley G, Carrington RW, Skinner JA, Briggs TW (2009) Does smoking influence outcome after autologous chondrocyte implantation? A case-controlled study. J Bone Joint Surg Br 91(12):1575–1578
20. Brittberg M (2011) Debridement of cartilage. In: Brittberg M, Gersoff W (eds) Cartilage surgery; an operative manual. Elsevier-Saunders, Philadelphia, pp 19–25
21. Dzioba RB (1988) The classification and treatment of acute articular cartilage lesions. Arthroscopy 4(2):72–80

22. Head M, Richardson J (2011) Bone grafting around an articular joint. In: Brittberg M, Gersoff W (eds) Cartilage surgery; an operative manual. Elsevier-Saunders, Philadelphia, pp 263–271
23. Cox CL, Huston LJ, Dunn WR, Reinke EK, Nwosu SK, Parker RD, Wright RW, Kaeding CC, Marx RG, Amendola A, McCarty EC, Spindler KP (2014) Are articular cartilage lesions and meniscus tears predictive of IKDC, KOOS, and Marx activity level outcomes after anterior cruciate ligament reconstruction? A 6-year multicenter cohort study. Am J Sports Med 42(5):1058–1067
24. Røtterud JH, Sivertsen EA, Forssblad M, Engebretsen L, Arøen A (2013) Effect of meniscal and focal cartilage lesions on patient-reported outcome after anterior cruciate ligament reconstruction: a nationwide cohort study from Norway and Sweden of 8476 patients with 2-year follow-up. Am J Sports Med 41(3):535–543
25. Flanigan DC, Harris JD, Trinh TQ, Siston RA, Brophy RH (2010) Prevalence of chondral defects in athletes' knees: a systematic review. Med Sci Sports Exerc 42(10):1795–1801
26. Mithoefer K, Hambly K, Della Villa S, Silvers H, Mandelbaum BR (2009) Return to sports participation after articular cartilage repair in the knee: scientific evidence. Am J Sports Med 37(Suppl 1):167S–176S
27. Mithoefer K, Hambly K, Logerstedt D, Ricci M, Silvers H, Della VS (2012) Current concepts for rehabilitation and return to sport after knee articular cartilage repair in the athlete. J Orthop Sports Phys Ther 42(3):254–273
28. Bekkers JEJ, de Windt TS, Brittberg M, Saris DBF (2012) Cartilage repair in football (soccer) athletes: what evidence leads to which treatment? A Critical Review of the Literature. Cartilage 3:43S–49S
29. Mithoefer K, Williams RJ 3rd, Warren RF, Wickiewicz TL, Marx RG (2006) High-impact athletics after knee articular cartilage repair: a prospective evaluation of the microfracture technique. Am J Sports Med 34(9):1413–1418
30. Bark S, Riepenhof H, Gille J (2012) AMIC cartilage repair in a professional soccer player. Case Rep Orthop 2012:364342
31. Giannini S, Buda R, Vannini F, Cavallo M, Grigolo B (2009) One-step bone marrow-derived cell transplantation in talar osteochondral lesions. Clin Orthop Relat Res 467(12):3307–3320
32. Stanish WD, McCormack R, Forriol F, Mohtadi N, Pelet S, Desnoyers J, Restrepo A, Shive MS (2013) Novel scaffold-based BST-CarGel treatment results in superior cartilage repair compared with microfracture in a randomized controlled trial. J Bone Joint Surg Am 95(18):1640–1650
33. Brittberg M, Faxén E, Peterson L (1994) Carbon fiber scaffolds in the treatment of early knee osteoarthritis. A prospective 4-year followup of 37 patients. Clin Orthop Relat Res 307:155–164
34. Bekkers JE, Bartels LW, Vincken KL, Dhert WJ, Creemers LB, Saris DB (2013) Articular cartilage evaluation after TruFit plug implantation analyzed by delayed gadolinium-enhanced MRI of cartilage (dGEMRIC). Am J Sports Med 41(6):1290–1295
35. Joshi N, Reverte-Vinaixa M, Díaz-Ferreiro EW, Domínguez-Oronoz R (2012) Synthetic resorbable scaffolds for the treatment of isolated patellofemoral cartilage defects in young patients: magnetic resonance imaging and clinical evaluation. Am J Sports Med 40(6):1289–1295
36. Kon E, Delcogliano M, Filardo G, Busacca M, Di Martino A, Marcacci M (2011) Novel nano-composite multilayered biomaterial for osteochondral regeneration: a pilot clinical trial. Am J Sports Med 39(6):1180–1190
37. Gobbi A, Karnatzikos G, Sankineani SR (2014) One-step surgery with multipotent stem cells for the treatment of large full-thickness chondral defects of the knee. Am J Sports Med 42(3):648–657
38. Gudas R, Gudaite A, Pocius A, Gudiene A, Cekanauskas E, Monastyreckiene E, Basevicius A (2012) Ten-year follow-up of a prospective, randomized clinical study of mosaic osteochondral autologous transplantation versus microfracture for the treatment of osteochondral defects in the knee joint of athletes. Am J Sports Med 40(11):2499–2508
39. Gudas R, Kunigiskis G, Kalesinskas RJ (2002) Long-term follow-up of osteochondritis dissecans [Article in Lithuanian]. Medicina (Kaunas) 38(3):284–288
40. Farr J, Cole BJ, Sherman S, Karas V (2012) Particulated articular cartilage: CAIS and DeNovo NT. J Knee Surg 25(1):23–29
41. Cole BJ, Farr J, Winalski CS, Hosea T, Richmond J, Mandelbaum B, De Deyne PG (2011) Outcomes after a single-stage procedure for cell-based cartilage repair: a prospective clinical safety trial with 2-year follow-up. Am J Sports Med 39(6):1170–1179
42. Mithöfer K, Minas T, Peterson L, Yeon H, Micheli LJ (2005) Functional outcome of knee articular cartilage repair in adolescent athletes. Am J Sports Med 33(8):1147–1153
43. Mithöfer K, Peterson L, Mandelbaum BR, Minas T (2005) Articular cartilage repair in soccer players with autologous chondrocyte transplantation: functional outcome and return to competition. Am J Sports Med 33(11):1639–1646
44. Martinez-Carranza N, Berg HE, Lagerstedt AS, Nurmi-Sandh H, Schupbach P, Ryd L (2014) Fixation of a double-coated titanium-hydroxyapatite focal knee resurfacing implant: a 12-month study in sheep. Osteoarthritis Cartilage 22(6):836–844
45. Aslan H, Citak M, Bas EG, Duman E, Aydin E, Ates Y (2012) Early results of HemiCAP(®) resurfacing implant. Acta Orthop Traumatol Turc 46(1):17–21
46. Della Villa S, Kon E, Filardo G, Ricci M, Vincentelli F, Delcogliano M, Marcacci M (2010) Does intensive rehabilitation permit early return to sport without compromising the clinical outcome after arthroscopic autologous chondrocyte implantation in highly competitive athletes? Am J Sports Med 38(1):68–77
47. Reinold MM, Wilk KE, Macrina LC, Dugas JR, Cain EL (2006) Current concepts in the rehabilitation following articular cartilage repair procedures in the knee. J Orthop Sports Phys Ther 36(10):774–794. Review

Management of Collateral Ligament Injuries (Medial and Lateral) in Competitive Athletes

40

Massimo Berruto, Paolo Ferrua, Francesco Mattia Uboldi, Eva Usellini, Stefano Pasqualotto, and Marelli Bruno Michele

40.1 Medial Collateral Ligament and Posteromedial Corner

40.1.1 Introduction

Medial collateral ligament (*MCL*) is the principal passive restraint to valgus forces applied to the knee and, along with other anatomic structures such as hamstrings muscles, is responsible for the stability of the medial compartment of the knee on the coronal plane. In a sportsmen population, MCL is frequently ruptured alone or in association with menisci and cruciate ligaments. A high grade acute injury of the MCL can evolve in a *chronic valgus instability* that can seriously impair not only sports performance but also daily activities. Although this condition can be considered rare, a prompt treatment of an acute injury is mandatory in order to prevent it and allow the athlete to return at a level of activity comparable to the one before injury.

40.1.2 Epidemiology

MCL is one of the most frequently injured ligaments of the knee, involved in at least 42 % of all ligamentous injuries [1]. MCL injuries most occur in young, active, male population, and pivot-contact sports such as football, basketball, and rugby are considered at high risk [2]. Male athletes are indeed twice more likely to suffer for an isolated or associated medial compartment injury if compared to the same level female population [3]. The yearly incidence of medial knee injury of the knee is about 0.24 per 1,000 people, but analyzing a sportive population (United States Military Academy cadets), the incidence rises to 7.3 per 1,000 patients [4]. Sports-specific incidence is well studied in literature [5–10] (Table 40.1), and the *most represented injury mechanism* is a direct blow on the lateral side of the knee in flexion with the foot planted on the ground, which is quite frequent in contact sports such as rugby and American football. The second most frequent mechanism is an abnormal combined movement of valgus, flexion and external rotation, which frequently occurs in skiing and cutting/pivoting sports such as basketball and football. Lundblad et al. published in 2013 a prospective study about a cohort of 1,743 UEFA professional football players [10]: MCL injury rate was 0.33 per 1,000 h of activity, which means that a 25-player team experiences at least 2 MCL injuries per year. The most frequent mechanism was collision with an opponent (mostly associated with a foul), and injuries were significantly more frequent during the last 15 min of both the first and second half of the game, with a probable correlation to fatigue.

40.1.3 Clinical and Diagnostic Examination

First of all, it is mandatory to ask the patient or somebody who was present at the moment of the injury about its mechanism: an isolated MCL injury generally occurs with a valgus force applied to a flexed knee; if any rotational mechanism is present, one must suspect a multiple ligament injury, such as the frequent association of an ACL lesion. A careful inspection of the knee must be done looking for bruising, hemarthrosis, and localized swelling which are frequent in isolated MCL injuries. On the contrary, an early onset hemarthrosis must bring the suspect on an intra-articular pathology such as tibial plateau fracture or ACL/PCL lesion. Patients presenting an MCL injury normally refer pain to a well-defined area correspond-

M. Berruto (✉) • P. Ferrua • F.M. Uboldi
E. Usellini • S. Pasqualotto
SSD Chirurgia Articolare del Ginocchio, Istituto Ortopedico Gaetano Pini Milano, Piazza A. Cardinal Ferrari 1, Milan 20122, Italy
e-mail: massimo.berruto@fastwebnet.it; paolo.ferrua81@gmail.com; fraubo@gmail.com; usellini@gpini.it; stefano.pasqualotto20@gmail.com

M.B. Michele
Dipartimento Ortotraumatologia Generale e Chirurgie Ortopediche Specialistiche, Istituto Ortopedico Gaetano Pini, Piazza A.Cardinal Ferrari 1, Milan 20122, Italy
e-mail: prof.brunomarelli@gmail.com

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_40,

Table 40.1 Sports-specific incidence of MCL lesions

Sports practice	Incidence	Most common mechanism of injury
Rugby [5]	28 % of all knee injuries	38 % tackle
	3.1 per 1,000 h of play	25 % ruck and maul
American football [6]	36.1 % of all knee injuries	Mostly traumatic
	2.42 per 1,000 h of play	
Ice hockey [7, 8]	0.13 per 1,000 h of training	77 % collision with another player
	1.47 per 1,000 h of match	
Alpine skiing [9]	18 % of all injuries	Noncontact (valgus-external rotation)
	60 % of knee injuries	
Football [10]	Mean 0.33 per 1,000 h	70 % contact with an opponent
	1.31 per 1,000 h of match	

Table 40.2 Conservative treatment of isolated MCL injuries

Grade	Immobilization	Weight bearing	Restriction of ROM	Return to training
I	Only for pain prevention	Partial	Full-active ROM as soon as possible	10–15 days
II	Hinged short-arm knee brace	Partial	Full-active ROM as soon as possible	4 weeks
III[a]	Hinged long-arm then short-arm knee brace	Partial as soon as possible	Immobilization 30° for 3 weeks (with passive ROM recovery) then recovery of full ROM after 3 weeks	8 weeks

[a]In selected cases only [24, 25]

ing to the site of lesion, which should be identified through careful palpation. Assessing the medial joint line opening will help to grade the MCL lesion [11]: the valgus stress test must be applied at 0 and 30° of flexion and compared to the healthy knee. An opening between 0 and 5 mm defines a grade I lesion, between 5 and 10 mm a grade 2 lesion, and if the joint line opening is more than 10 mm, the lesion is graded 3. A positive valgus stress in extension is indicative of a posterior oblique ligament (POL) or posteromedial corner (PMC) lesion, and it's most commonly found in multiligamentous injuries, especially with involvement of the ACL [12]. Assessment of the combined rotational and central pivot instability can be very difficult in an acute setting and can possibly require examination under anesthesia to address diagnosis and give indications for successive treatments. Presence of an eventual ACL- and PCL-associated lesions has to be investigated carefully as they are very frequent. Assessment of the *associated rotational component* of the lesion can be obtained through different tests:

1. *Anterior drawer in external rotation*: the anterior drawer test is performed in a standard fashion at 90° of flexion and with the medial tibia held in external rotation [13].
2. *Swain test*: with the knee at 90° lying out of the bed, the tibia is forced in external rotation causing pain along the medial side of the joint [14].
3. *Combined valgus stress test*: the valgus test is performed at 30° of flexion with the foot held in external rotation.

In acute settings, a *standard radiographic exam* must be performed looking for potential fractures or epiphyseal cartilage damage and must include anterior-posterior, lateral, and patellar axial views. In chronic lesions, the exam must be completed with a full set of weight-bearing x-rays, including long-leg and Rosenberg-Schuss views in order to evaluate frequent concomitant osteoarthritic degeneration of the medial compartment and the presence of a calcification near to the femoral insertion, known as Pellegrini-Stieda lesion [15]. In the case of chronic laxity, many authors advocate the use of dynamic valgus stress x-rays in order to evaluate and measure differential laxity and plan the most appropriate treatment [16].

MRI is fundamental in the diagnosis of medial side injuries as it allows to evaluate not only ligament but also articular cartilage and meniscal lesions. In acute settings, a bone bruise of the medial compartment subchondral bone following a valgus-directed force can be frequently found. MRI grading of the lesion has a very good concordance with clinical grading and can be used as a complementary tool for addressing diagnosis and therapeutic indications [17].

40.1.4 Treatment Strategy

40.1.4.1 Conservative Treatment

The wide area of the lesion's surface and the relatively high blood supply of the MCL explain the very high rate of spontaneous healing without need of surgical reparation or reconstruction. In the case of a stage I to II lesion of the MCL, primary treatment must be conservative according to the principles shown in Table 40.2. The insulted knee must be immobilized in a brace to protect it from iterative injuries and decrease pain. Early passive motion and partial weight bearing should be allowed as soon as possible. Strengthening of the quadriceps and flexor muscles with

isometric exercises must start as soon as possible in order to be able to proceed to a fast rehabilitation program.

Return to play will be possible only at the resolution of pain and after having achieved a sufficient stability at the clinical exam. As shown by Indelicato [18] and Jones et al. [19], results of a well-conducted conservative treatment are positive in 90–95 % in a population of competitive athletes, also in the presence of a grade III lesion. There is no consensus about the effectiveness of using a protective brace during activity after a minor lesion of the medial compartment, the prescription must therefore be done only if clinically necessary and in selected cases such as contact sports or persistent subjective instability feeling [20]. A minor group of conservatively treated proximal s-MCL lesions can develop a localized pain at the femoral insertion scar which can require a delayed surgical treatment (debridement, scarring of the ligament, and microperforation of the insertion) with satisfactory results.

40.1.4.2 Surgical Treatment

Surgical reparation in athletes can find an indication in the presence of a bony avulsion, in multiligamentous injuries, and in the presence of an isolated complete tear involving both midsubstance and tibial insertion. A diagnostic arthroscopy should be routinely performed in order to check for eventual associated lesions of menisci, cruciate ligaments, and articular cartilage. A bony avulsion, more frequent at the femoral site of insertion (Stieda fracture), is normally reinserted using a metallic lag screw. In the presence of *distal or proximal avulsion*, reparation should be completed with use of suture anchors (Fig. 40.1) in order to obtain a stable and anatomic reinsertion. The distally avulsed MCL can lie on the medial side with the pes anserinus tendons interposed, defining a "Stener-like" lesion of the knee [14]. This specific lesion cannot repair without surgical intervention and has to be immediately recognized and addressed. A *midsubstance lesion* can be treated with a standard side-to-side reparation technique, but as the quality of the remnant tissue is frequently poor, often the association with an augmentation technique as the ones published by Kim et al. [21] and Stannard [22] is needed also in acute settings. In conclusion, a stepwise approach must be employed in the presence of an acute complete lesion of the MCL/PMC:

- Begin with the deep layer and reconstruct meniscotibial and meniscofemoral bundles of the deep MCL, stabilizing the medial meniscus.
- Progress and repair the eventually affected POL and the portion of the superficial MCL (s-MCL) interested by the lesion, taking care of providing a strong but anatomically correct reinsertion using suture anchors. In the presence of a midsubstance lesion, proceed to side-to-side reparation if the remnants are strong enough or switch to repair-augmentation or reconstruction if a correct stability all throughout the range of motion cannot be achieved.
- In the presence of a nonreparable midsubstance lesion, we prefer not to use autologous semitendinosus as a graft, as it weakens medial side residual stability, and we rely on an allogenic tubular graft or, in selected cases, biosynthetic graft.

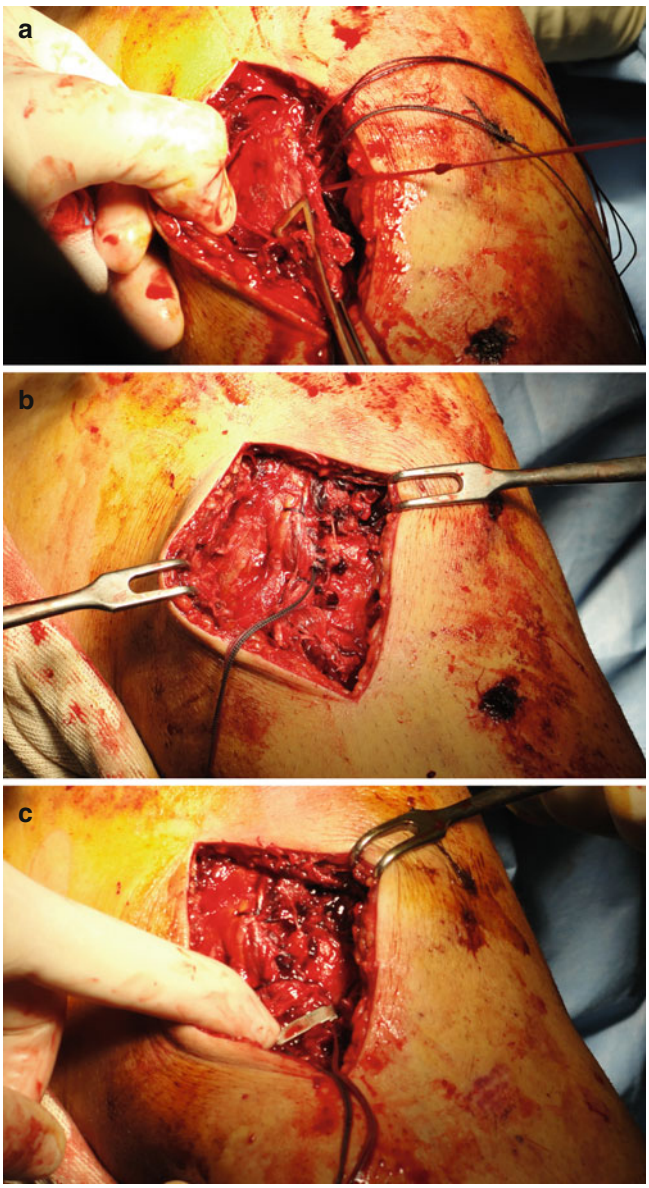


Fig. 40.1 Complete midsubstance and distal insertion lesion of the s-MCL: anatomic reinsertion is obtained using suture anchors (**a**), side-to-side Orthocord ® sutures (**b**) and completed positioning of a metallic staple on the proximal tibia (**c**)

Tibial/femoral avulsions recognize a better prognosis if compared to midsubstance lesions, and best results are achieved if the reparation is performed within 3 weeks from the moment of injury [14]. There is a lack of evidence about clinical outcomes of surgically treated acute isolated MCL lesions. Most of our knowledge is based on multiligamentous

injuries series including medial compartment reparation. Ibrahim et al. [23] reported about 18 patients undergoing a reparation of a complete acute MCL rupture: 89 % of patients were stable at a minimum follow-up of 39 months with a mean Lysholm score of 79.2, but mean Tegner score decreased to 4.7 from a pre-injury score of 7.6. Owens et al. [24] also reported good results on 11 complete MCL rupture at 48 months but with a relatively high ratio (27 %) of postoperative stiffness requiring secondary surgery (arthroscopic lysis).

40.1.4.3 Treatment of Chronic Lesions

Medial instability in the presence of an isolated chronic medial collateral ligament injury in competitive athletes is a very rare condition, but its association to ACL or ACL/PCL chronic lesions is far more common. In the case of a clinically relevant chronic medial instability, the surgical indication is mandatory. The results of reparative techniques are very poor, due to the chronicity of the lesion and the absence of healthy ligament tissue, necessary for achieving a good quality reparation, and this should not be performed in a chronic setting. *Reconstructive surgery* is commonly performed in these cases using different techniques and different grafts (autologous, heterologous, or synthetic). Most of the employable surgical interventions originate from the technique described by Bosworth in 1952 [25]. The semitendinosus tendon is stripped with an open stripper and left attached to its tibial insertion. The free end of the tendon is then whipstitched, and isometry is checked looping it on a K-wire positioned in the site of insertion of MCL on medial epicondyle. If isometry is confirmed, a half tunnel is performed, and the free part of the tendon is secured to the femur with a screw, having care of applying a varus moment to the tibia during fixation. If the semitendinosus tendon is not available as a graft (for instance associated ACL reconstruction), one can consider to use, with the same principles, a heterologous tubular graft (semitendinosus, peroneus longus, tibialis anterior or posterior). This technique has been heavily modified in the past few years, but it is still very useful in reconstruction of isolated chronic s-MCL lesion or as an augmentation in acute settings.

In the presence of a concomitant POL-PMC lesion/insufficiency, an *anteromedial rotatory instability* is defined, and the technique employed must include a stabilization of the PMC as an isolated reconstruction of the MCL could not be sufficient [21, 22, 26]. A fan-shaped graft as a heterologous fascia lata can be used in order to address also posteromedial component of the instability (Fig. 40.2): maintaining a single insertion on the femoral side (half tunnel and interference screw), the anterior part of the graft is inserted on the anteromedial side of the tibia using suture anchors and fixed a 30° of flexion. The rotational stability is then achieved fixing the posterior border of the graft on the posteromedial side of the tibia in full extension. In alternative, a number of techniques defined as "anatomic" have been described as

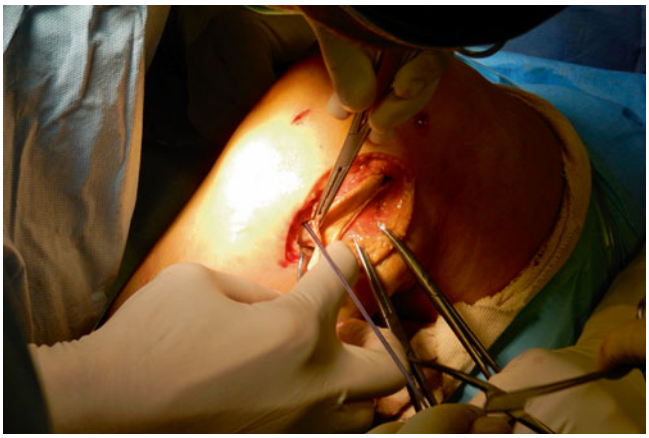


Fig. 40.2 Fan-shaped allogenic fascia lata can be used to reconstruct both superficial MCL and POL. The graft has already been fixed on the femur with an interference screw

the one described by Coobs et al. in 2010 [27]: two separate soft tissue grafts are used to reconstruct separately s-MCL and POL. All the grafts are secured on both sides with interference screws: first, the femoral insertions are fixed, then the distal part of the s-MCL is fixed at 30° of flexion and neutral rotation. The procedure ends with fixation of POL bundle in extension reproducing the "normal anatomy" of the medial side of the knee. This type of technique, although very promising on a biomechanical point of view, can be very complicated on the daily basis, as the increased number of tunnels and fixation devices can represent an issue, moreover, in the case of a surgically treated multiligamentous injury. Given this considerations, LaPrade and Wijdicks [28] have published very good results with this technique on 28 patients at a mean follow-up of 1.5 years: mean IKDC score improved from 43.5 to 76.2, and all patients noted a subjective improvement in terms of stability.

40.1.5 Postoperative Management and Rehabilitation

After reparative/reconstructive surgery of the medial compartment, the operated knee must be protected in a hinged knee brace during sleep and walking for the first 4–6 weeks. Partial weight bearing with crutches is immediately allowed, and recovery of passive ROM must start as soon as possible also by the means of continuous passive motion (CPM) in order to prevent postoperative stiffness. Strengthening of the quadriceps is obtained through early isometric exercises. After 6 weeks, if quadriceps control is obtained, the brace can be removed, and full weight bearing can be allowed. Return to training is possible at least after 3 months if the knee is painless and stable at the clinical examination.

40.2 Lateral Collateral Ligament

40.2.1 Introduction

Although isolated *lateral collateral ligament* (*LCL*) lesions are extremely rare, a *varus instability*-associated component is frequently noticed in the presence of a posterolateral instability or in multiligamentous injuries. Considering that the association of a nonidentified injury of the posterolateral corner (PLC) is a frequent cause of late failure of an ACL or PCL reconstruction [29, 30], these lesions must be identified and carefully ruled out both in acute and chronic cases. The most common causes of PLC lesions are motor vehicle accidents (mainly motorcycle) or high-energy traumas, and only 40 % of PLC lesions are considered related to sports activity [31]. Isolated PLC lesions are also relatively rare (less than 2 %) as more than 75 % of them are associated to ACL/PCL [32, 33].

40.2.2 Clinical and Diagnostic Examination

The *most frequent mechanism of injury* of the LCL is a direct blow to the medial aspect of the knee, but a hyperextension or noncontact varus stress is also common. Patients must be asked about paresthesia or motor impairment after trauma, as association of different degrees of peroneal nerve involvement is frequent in these types of injuries (15 %) [34]. Knee must be carefully inspected looking for ecchymosis and mostly hemarthrosis, which can be the signal of an associated significant intra-articular injury. In chronic settings, one should observe the patient's gait looking for a varus thrust phenomenon during deambulation, which is frequently noticed by the patient as one of the main symptoms. Specific evaluation of the LCL status is carried on performing varus stress test at 0° and 30° of flexion. The joint line opening and softness of the end point allows to grade the lesion similarly to the medial collateral ligament. The association with PLC lesions/deficiency can be investigated with specific tests:

1. *Dial test*: with the patient in prone decubitus, the external rotation of the tibia on the femur is evaluated at 30° and 90° of flexion and compared to the healthy contralateral side. If a side-to-side difference bigger than 10° of rotation is noticed at 30° of flexion, the test is considered positive and suggests a PLC lesion. If the test is positive also at 90° of flexion, a concomitant lesion of the PCL must be suspected.
2. *Hughston test*: in supine decubitus, the examiner lifts the leg by the great toe while stabilizing the distal femur. The degrees of hyperextension are recorded and compared to the contralateral side. If positive, a concomitant ACL/PCL and PLC lesion is presumed but with a very low degree of sensitivity.
3. *Posterolateral drawer*: patient is supine with the knee at 90° of flexion and foot stabilized. The examiner applies a posterior-directed force to the tibia kept in external rotation. A difference with contralateral can be suspicious for a PLC injury.
4. *Reverse pivot shift*: the knee is flexed at 90° and valgus and external rotation forces are applied to the tibia during extension. The test is positive if a reduction of the subluxated lateral tibial plateau is noticed during the extension movement in the injured knee.

In the presence of an *acute lateral knee trauma*, a complete x-ray exam is performed in order to rule out frequent associated fractures such as bony avulsion or Segond fractures of the tibial plateau. In chronic settings, the exam must be completed with long-arm weight-bearing x-rays (useful to quantify varus thrust sign) and Rosenberg-Schuss view which give additional information about eventual degeneration of the medial and lateral compartment. MRI is very useful, mostly in acute settings, to directly evaluate the LCL and PLC structures but also the presence of associated lesions of cruciate ligaments, menisci, and articular cartilage which are very common. A chronic varus instability should also be evaluated and assessed by the means of varus stress dynamic x-rays. During a*rthroscopic examination*, an LCL and PLC lesion can cause the so-called drive-through sign: a gap of more than 1 cm during varus stress at 30° of flexion (figure-of-four position) [35].

40.2.3 Treatment of Acute Lesions

Grade I and II lesions of the LCL are generally treated conservatively with full extension long-leg knee brace. Partial weight bearing is allowed as soon as possible, and recovery of flexion in prone decubitus can be performed in order to prevent stiffness during the immobilization. Return to sport (frequently with protective bracing) is allowed once pain is resolved and varus stability completely restored at the clinical examination and normally comes in the low-grade injuries at 6–8 weeks from injury.

In grade III injury of LCL, the risk of developing a chronic instability is very high, and the treatment of these lesions is mainly surgical. Even if direct reparation of lateral structures is associated with a high rate of complications such as recurrence of instability or postoperative extension lag, a reparative approach should be always preferred in acute settings. Akin to the medial compartment, we suggest a stepwise approach:

- After exposure of the lateral side of the knee, the *common peroneal nerve* must be identified and carefully protected all along the procedure.

- First of all, focus on the popliteal tendon which can be frequently avulsed from the femoral side and can be repaired with transfemoral sutures fixed on a button on the medial side. In alternative, if the lesion is midsubstance (frequently at the myotendinous junction), a tenodesis on the posterior aspect of the fibula can be performed, transforming it in a passive stabilizer [36].
- Once repaired the popliteus complex, the LCL lesion can be addressed: if the lesion is proximal, the reinsertion can be performed with transosseous drill holes, eventually in association to the ones used for popliteus reconstruction. If the lesion is distal, the repair can be obtained with transosseous sutures on the fibular head but some authors advocate the use of an interference screw [37].

40.2.4 Treatment of Chronic Lesions

Surgical reconstruction of LCL can be performed with many different techniques, depending on the presence of an associated lesion of the PLC. As a preliminary consideration, the proximity of the common peroneal nerve is a common feature in all these procedures, and it is again very important to identify and protect this structure during all the procedure.

- An isolated LCL chronic lesion can be treated as described by Latimer et al. [38] with a graft secured on the isometric point of the femur and on the proximal aspect of the fibula with two interference screws. The author promotes a bone-patellar tendon-bone graft, but the technique can be successfully applied also with autologous or heterologous soft tissue grafts (semitendinosus, for instance).
- If, as frequently happens, the varus instability comes in association with a mild posterolateral one, a reconstruction can be performed with a technique as described by Larson [39]: after exposure and isolation of common peroneal nerve, a tunnel is performed in the fibular head from anterior to posterior and from medial to lateral. The graft, commonly autologous semitendinosus, is then whipstitched, inserted into the peroneal tunnel, and fixed in a femoral half tunnel after checking for isometry (Fig. 40.3). As an alternative graft, a tubular heterologous graft or biosynthetic ligament can be used with comparable results and reducing donor site morbidity.
- In the presence of an *associated major PLC instability*, a complex reconstruction as the ones described by Laprade et al. [40] and Noyes et al. [41] can be indicated:
  - Noyes et al. described a femoral-fibular posterolateral reconstruction using an Achilles tendon allograft which is passed through a peroneal tunnel near to the site of insertion of LCL and femoral tunnels performed from posterior to anterior at its femoral insertion. The posterior portion of the graft is then sutured to the popliteus tendon to simulate the popliteofibular ligament, and posterolateral capsule is plicated in order to prevent hyperextension or varus recurvatum.
  - The La Prade's technique, performed using a splitted Achilles tendon allograft, allows the concomitant reconstruction of LCL, popliteus tendon, and popliteofibular ligament using four separate half tunnels (two on the femur, one on the fibula, and one on the proxi-

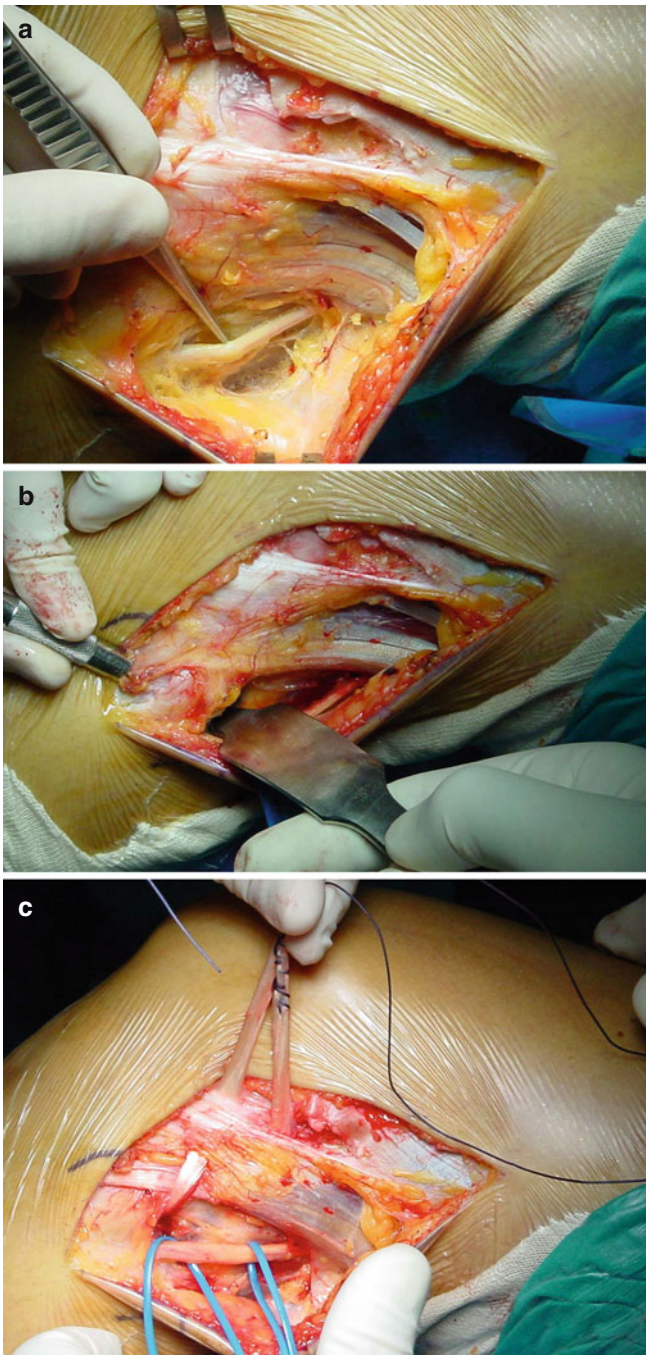


Fig. 40.3 PLC and LCL reconstruction according to Larson: identification and protection of the common peroneal nerve (**a**); drilling of the tunnel through proximal fibula (**b**); passage of the graft in the fibular tunnel and research of isometric fixation point on the femur (**c**)

mal tibia) and four interference screws. Recovery after this type of surgery must be very slow in competitive athletes, and return to sport should not be allowed before 9–12 months after surgery, moreover if it comes associated with a PCL or ACL reconstruction.

40.2.5 Postoperative Management and Rehabilitation

The operated knee must be placed in a hinged knee brace, and external rotation and varus stresses must be avoided in the first 6 weeks. Partial weight bearing is allowed as soon as possible, and passive ROM recovery exercises begin from postoperative day 1. Passive ROM is limited at 90° of flexion in the first 2 weeks, and the exercises are performed in prone decubitus in order to avoid posterior translation of the tibia and having care of not pushing the tibia in external rotation. Six weeks after surgery, the patient is normally allowed to walk without brace and progressively in full weight bearing. Return to training and competition is generally never allowed before 6–7 months after surgery and only if the knee is painful and stable at the clinical examination.

References

1. Bollen S (2000) Epidemiology of knee injuries, diagnosis and triage. Br J Sports Med 34(3):227–228
2. Phisitkul P, James SL, Wolf BR et al (2006) MCL injuries of the knee: current concepts review. Iowa Orthop J 26:77–90
3. Pedowitz RA, O'Connor JJ, Akeson WH (2003) Daniel's knee injuries: ligament and cartilage structure, function, injury, and repair, 2nd edn. Lippincott Williams & Wilkins, Philadelphia
4. Roach CJ, Haley CA, Cameron KL et al (2014) The epidemiology of medial collateral ligament sprains in young athletes. Am J Sports Med 42(5):1103–1109
5. Dallalana RJ, Brooks JH, Kemp SP et al (2007) The epidemiology of knee injuries in English professional rugby union. Am J Sports Med 35(5):818–830
6. Swenson D, Collins CL, Best T et al (2013) Epidemiology of knee injuries among us high school athletes, 2005/06-2010/11. Med Sci Sports Exerc 45(3):462–469
7. LaPrade RF, Wiljdicks CA, Griffith CJ (2009) Division I intercollegiate ice hockey team coverage. Br J Sports Med 43:1000–1005
8. Grant JA, Bedi A, Kurz J (2013) Incidence and injury characteristics of medial collateral ligament injuries in male collegiate ice hockey players. Sports Health 5(3):270–272
9. Warme WJ, Feagin JA, King P et al (1995) Ski injury statistics, 1982 to 1993, Jackson Hole Ski Resort. Am J Sports Med 23:597–600
10. Lundblad M, Walden M, Magnusson H et al (2013) The UEFA injury study: 11-year data concerning 346 MCL injuries and time to return to play. Br J Sports Med 47:759–762
11. Quarles JD, Hosey RG (2004) Medial and collateral ligament injuries: prognosis and treatment. Prim Care 31:957–975
12. Tibor LM, Marchant MH, Taylor Dean C et al (2011) Management of medial-sided knee injuries, part 2. Posteromedial corner. Am J Sports Med 39(6):1332–1340
13. Hughston JC (2003) Knee ligaments: injury and repair. The Hughston Sports Medicine Foundation, Columbus
14. Lonergan KT, Taylor DC (2002) Medial collateral ligament injuries of the knee: an evolution of surgical reconstruction. Tech Knee Surg 1:137–145
15. Altschuler EL, Bryce TN (2006) Images in clinical medicine. Pellegrini-Stieda syndrome. N Engl J Med 354(1):e1
16. LaPrade RF, Bernhardson AS, Griffith CJ et al (2010) Correlation of valgus stress radiographs with medial knee ligament injuries: an in vitro biomechanical study. Am J Sports Med 38:331–338
17. Yao L, Dungan D, Seeger LL (1994) MR imaging of tibial collateral ligament injury: comparison with clinical examination. Skeletal Radiol 23(7):521–524
18. Indelicato PA (1983) Non-operative treatment of complete tears of the medial collateral ligament of the knee. J Bone Joint Surg Am 65:323–329
19. Jones RE, Henley B, Francis P (1986) Non operative management of isolated grade III collateral ligament injury in high school football players. Clin Orthop Relat Res 213:137–140
20. LaPrade RF, Wijdicks CA (2012) The management of injuries to the medial side of the knee. J Orthop Sports Phys Ther 42(3):221–233
21. Kim SJ, Lee DH, Kim TE et al (2008) Concomitant reconstruction of the medial collateral and posterior oblique ligaments for medial instability of the knee. J Bone Joint Surg Br 90(10):1323–1327
22. Stannard JP (2010) Medial and posteromedial instability of the knee: evaluation, treatment and results. Sports Med Arthrosc 18(4):263–268
23. Ibrahim SA (1999) Primary repair of the cruciate and collateral ligaments after traumatic dislocation of the knee. J Bone Joint Surg Br 81(6):987–990
24. Owens BD, Neault M, Benson E et al (2007) Primary repair of knee dislocations: results in 25 patients (28 knees) at a mean follow up of four years. J Orthop Trauma 21(2):92–96
25. Bosworth DM (1952) Transplantation of the semitendinosus for repair of laceration of medial collateral ligament of the knee. J Bone Joint Surg Am 34-A(1):196–202
26. Lind M, Jakobsen BW, Lund B et al (2009) Anatomical reconstruction of the medial collateral ligament and posteromedial corner of the knee in patients with chronic medial collateral ligament instability. Am J Sports Med 37(6):1116–1122
27. Coobs BR, Wijdicks CA, Armitage BM et al (2010) An in vitro analysis of an anatomical medial knee reconstruction. Am J Sports Med 38:339–347
28. LaPrade RF, Wijdicks CA (2012) Surgical technique: development of an anatomic medial knee reconstruction. Clin Orthop Relat Res 470(3):806–814
29. O'Brien SJ, Warren RF, Pavlov H et al (1991) Reconstruction of the chronically insufficient anterior cruciate ligament with the central third of the patellar ligament. J Bone Joint Surg Am 73(2):278–286
30. Noyes FR, Barber-Westin SD, tRoberts CS (1994) Use of allografts after failed treatment of rupture of the anterior cruciate ligament. J Bone Joint Surg Am 76(7):1019–1031
31. Frank JB, Youm T, Meislim RJ et al (2007) Posterolateral corner injuries of the knee. Bull NYU Hosp Jt Dis 65(2):106–114
32. DeLee JC, Riley MB, Rockwood CA (1983) Acute posterolateral rotatory instability of the knee. Am J Sports Med 11(4):199–207
33. Fanelli GC, Orcutt DR, Edson CJ (2005) Current concepts: the multiple ligament injured knee. Arthroscopy 21:471–486
34. LaPrade RF, Terry GC (1997) Injuries to the posterolateral aspect of the knee. Association of anatomic injury patterns with clinical instability. Am J Sports Med 25:433–438

35. LaPrade RF (1997) Arthroscopic evaluation of the lateral compartment of the knees with grade 3 posterolateral knee complex injuries. Am J Sports Med 25:596–602
36. Murphy K, Helgeson M, Lehman R (2006) Surgical treatment of acute lateral collateral ligament and posterolateral corner injuries. Sports Med Arthrosc Rev 14(1):23–26
37. Cole BJ, Harner CD (1999) The multiple ligament injured knee. Clin Sports Med 18:241–262
38. Latimer HA, Tibone JE, ElAttrache NS et al (1998) Reconstruction of the lateral collateral ligament of the knee with patellar tendon allograft. Report of a new technique in combined ligament injuries. Am J Sports Med 26(5):656–662
39. Sidles JA, Larson RV, Garbini JL et al (1988) Ligament length relationships in the moving knee. J Orthop Res 6:593–610
40. LaPrade RF, Johansen S, Engebretsen L (2011) Outcomes of an anatomic posterolateral knee reconstruction: surgical technique. J Bone Joint Surg Am 93(Suppl 1):10–20
41. Noyes FR, Barber Westin SD (2011) Long term assessment of posterolateral ligament femoral-fibular reconstruction in chronic multiligament unstable knees. Am J Sports Med 39(3):497–505

Anterior Cruciate Ligament

41

Pier Paolo Mariani, Guglielmo Cerullo,
Germano Iannella, and Marco Giacobbe

41.1 Etiology

The *anterior cruciate ligament* (ACL) is one of the most commonly disrupted ligaments in the knee of athletes. Most of the injuries happen in a noncontact trauma, when an athlete self-generates great forces or moments at the knee, that applied excessive loading on the ACL. Numerous theories have been proposed to explain what predisposes an athlete to noncontact ACL injury. These theories are classified as anatomic, environmental, hormonal, and neuromuscular [1]. Anatomic factors include an A-shaped intercondylar notch instead of a reverse U-shaped notch that determines impingement of the ACL on the medial border of the lateral femoral condyle in valgus stress and on the roof of the notch in hyperextension. Varus or valgus lower limb alignment adds stress on ACL during sports activities. Muscular discrepancy between extensor and flexor of the knee and knee joint congenital laxity also predispose to ACL rupture. Environmental factors include shoe-surface interface that increases frictional force between foot and ground creating higher-energy forces in the knee during decelerations or in cutting actions. Weather conditions (too hot or cold or raining) may represent another factor of risk. Playing style has a genetic component, but it is influenced by coaching and training and represents a factor of risk. Hormonal factors: women sustain two to eight times more ACL injuries for the same sports than men do. Many studies have tried to demonstrate influence of estrogen, but females' differences from males in lower limbs alignment, muscle power, and neuromuscular factors interfere with this theory. Neuromuscular factors: the balance between quadriceps and hamstring muscles is crucial to functional knee stability as quadriceps antagonists and hamstring agonists of ACL. Any weakness, increased flexibility, or delayed recruit pattern of hamstrings increases susceptibility to ACL injury.

41.2 Injury Mechanism

ACL rupture may happen for a contact or a noncontact injury. In a contact injury, a direct blow is applied directly on the knee from an opponent player, and it is typical of contact sports such as soccer, rugby, basketball, and American football. In a noncontact ACL injury, the more frequent mechanism, athlete self-generates great forces or moments at the knee that applied excessive loading on the ACL during cutting, decelerations, and landing from a jump. Injury mechanisms are mostly three: valgus-external rotation (VRE), varus-internal rotation (VRI), and hyperextension and tend to occur when the center of gravity of the body is behind the knee and when ground contact with the entire foot occurs [2]. In a report of literature from 1950 through 2007, Shimokochi and Shultz demonstrated noncontact ACL injuries are likely to happen during deceleration and acceleration motions with excessive quadriceps contraction and reduced hamstrings co-contraction at or near full knee extension [3]. Higher ACL loading during the application of a quadriceps force when combined with a knee internal rotation moment compared with an external rotation moment was noted. The ACL loading was also higher when a valgus load was combined with internal rotation as compared with external rotation. However, because the combination of knee valgus and external rotation motions may lead to ACL impingement, these combined motions cannot be excluded from the noncontact ACL injury mechanisms. Further, excessive valgus knee loads applied during weight bearing, decelerating activities also increased ACL loading. Kobayashi et al. analyzed the

P.P. Mariani (✉)
Department of sports science and health,
Università degli Studi di Roma "Foro Italico",
Piazza L. De Bosis 15, Rome 00194, Italy
e-mail: ppmariani@virgilio.it; ppmariani@uniroma4.it

G. Cerullo • G. Iannella • M. Giacobbe
Villa Stuart Sport Clinic, FIFA Medical Center,
Via Trionfale 5952, Rome 00136, Italy
e-mail: g.cerullo@tin.it; dottiannella@yahoo.i;
ocramgiacobbe@yahoo.it

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_41,

data of more than 1,700 athletes (838 males and 880 females) with an ACL injury with the aim of confirming the relationship between the ACL injury occurrence and the dynamic alignment of the lower extremity at the time of the injury. The authors referred that at the time of the injury, the number of subjects who had the injury during "competitions" rather than training was the largest (846/1718), accounting for 49.2 % of all the subjects. Noncontact cases were the largest (417/809) than the number of collision cases. The number of the subjects with the alignment of "Knee-in and Toe-out" (VRE) was the largest (793/1,603), followed by "Knee-out and Toe-in" (VRI) and "hyperextension" in this order [4]. Most of the injuries are reported to occur with noncontact mechanisms, such as those involving landing from a jump and sudden deceleration of the body while running, with or without a change in direction. Anterior cruciate ligament injuries often happen when an individual attempts to decelerate the body from a jump or forward running while the knee is in a shallow flexion angle [5–7]. At the time of injury, combined motions such as knee valgus and knee internal-external rotation are often noted. The ACL has been widely known to be loaded with anterior tibial shear forces [8, 9]. Unopposed quadriceps muscle forces produce anterior shear forces, possibly damaging the ACL, especially near full extension [10, 11]. On the other hand, hamstrings co-contraction forces are protective to the ACL, increasing knee stability while the quadriceps are contracting. An ACL injury often occurs when the body is positioned with the weight back on the heel, which may increase the quadriceps contraction force and reduce the efficacy of the hamstrings [11]. Because a combination of knee external rotation and valgus motions may impinge the ACL against the femoral intercondylar notch and because these motions have been often observed during noncontact ACL injury, knee external rotation remains an important consideration for ACL injury [12]. Contact injuries of the ACL are mainly the result of a direct blow applied to the proximal tibia with anterior-posterior direction. Typical examples of this mechanism are front or side tackles in soccer.

The better understanding of etiology and injury mechanisms is at the base for developing a prevention program. Modern literature shows promising results in programs that involve proprioception, strength training, and improved jumping, stopping, and turning techniques.

41.3 Clinical and Diagnostic Examination

Evaluation of the patient starts with the history. An acute ACL tear history is very familiar to the knee surgeon and usually the athlete refers of a "pop" or "crack" with pain in a pivoting movement, often in noncontact type, while playing the sport and also describes the knee as "coming apart" [13]. Mild or marked effusion occurs within 6–12 h after trauma. However, some ACL tears happen with minor trauma, no internal sensation, no or minimal effusion and mild pain, and frequently in emergency room are diagnosed as minimal sprain and lead to chronic instability. An athlete with a chronic ACL history often refers of pain from a meniscal tear or cartilage damage, with minimal instability in pivoting activities and mild effusion after training or competition. However, today, it is very unusual that a top-level athlete could refer of a chronic instability as he is promptly evaluated and treated after trauma by his team physician. It can occur more often after ACL reconstruction if not a good stability is achieved with surgical operation.

Lachman test is considered the most reliable and reproducible method sign of an ACL tear, as it has in acute a sensitivity of 78–99 % [18]. In acute injuries of top-level athletes, with large muscular thigh or hamstrings spasm, the Lachman test is not simple to perform and often is unpredictable unless a firm end point is felt to exclude an ACL tear. We prefer to perform also the *prone Lachman test* [14] in which gravity assists the forward movement of the tibia, hip extension stabilizes the femur, and relaxation is enhanced by the contact of quadriceps with the table. The patient is prone, the knee is held 20° or 30° flexed, and the examiner's hands grasp the tibia; the fingers are positioned in the joint line. Anteroposterior tibiofemoral movement is attempted; its interpretation and the quality of end point are no different from that when the patient is supine.

Pivot shift is a specific but very insensitive test for acute ACL injury in the nonanesthetized patient [15, 16] and also subject to interobserver error, and we use it in operating room under anesthesia in deciding if to perform or not an additional lateral plasty in case of evident positivity.

Valgus, varus, and posterior laxity are also examined in the routine manner.

For *instrumented evaluation*, we routinely use arthrometer. We have used for many years KT-1000 or 2000 arthrometer, but since the last 3 years, we are using the GNRB (Genurob, Laval, France) that some studies have demonstrated superior intra- and interexaminer reproducibility over the KT-1000 and examiner independency [17]. With arthrometer, it is also possible to suspect an ACL partial tear [18].

Magnetic resonance imaging (MRI) is an invaluable test to the diagnosis of an ACL injury as it has a specificity of 95 % and a sensibility of 86 % with normal coronal, axial, and sagittal views [19]. We are routinely using the oblique coronal and oblique sagittal images for their improved accuracy [20]. Normal ACL is both distinctly seen and taut, while when acutely injured appears indistinct and lax. MRI is also useful in ruling out other internal derangements detect [21]. *Segond's fracture*, visible also in anteroposterior X-ray, reveals avulsion of the anterolateral ligament [22, 23] that in some case needs to be refixed or reconstructed.

41.4 Treatment Strategy

41.4.1 Indications

In sports activity that requires cutting, jumping, and pivoting stress, it is mandatory to carry out the reconstruction after an ACL tear. It is well documented in literature that early ligament reconstruction reduces the risk of subsequent meniscal injury, especially in athletic population. Sports activity predisposes early damage of all static structures. Therefore, in ACL tear, surgical treatment is our indication for those athletes who want to continue playing their sports. Natural history of *partial ACL tears* is quite good over the medium term in the patients that limit their sports activities but functional instability seems to progress with time, especially in athletic population [24]; therefore, also in the case of ACL partial tear in young athletes, our indication is ACL surgical reconstruction.

41.4.2 Timing

In the past, initial concern existed over early reconstruction of ACL injury because of the increased risk for arthrofibrosis, and the surgery was delayed until minimal swelling, good leg control, and full range of motion was achieved. Top-level athletes request early surgery in order to reduce the time of return to play. Moreover, in the presence of associated injuries such as meniscal or collateral ligaments tears, immediate suture repair gives better results than delayed repair. For these reasons, better postoperative pain control and a more aggressive rehabilitative protocol with continuous passive motion, and early muscles exercises, can yield results that are independent of the timing of surgery [25].

41.4.3 Graft

Graft selection is a topic of discussion. The central third of patella tendon (bone-patellar tendon-bone) (BPTB) and the four-strand hamstrings (HS) are the two most commonly used *autografts*. Some studies show similar results in terms of laxity and functional results between the two grafts, and others show better stability for BPTB but not correlated with functional outcome. *Allografts* are not as strong as autografts and are not recommended for top-level athletes except in some case of ACL revision surgery. BPTB is our first autograft choice in top-level athletes both in male and in female. The main reason of this choice is the biological fixation, bone to bone, that allows an accelerated protocol of rehabilitation when a prompt return to sport is required.

41.4.4 Surgical Procedure

Surgical treatment of ACL tears has evolved over the past century. Several techniques and methods of fixation have been described, and a detailed description of all procedures is impossible.

The two most commonly used techniques today are anatomical single-bundle ACL reconstruction with HS or BPTB and anatomical double-bundle ACL reconstruction with HS that restore both anteromedial and posterolateral ACL bundle. Double-bundle technique seems to give better restore on rotational stability but in high-level athletes are not rare tears of the reconstructed posterolateral bundle. Our preferred method of ACL reconstruction is a *single-bundle* reconstruction with *transtibial technique* and a lateral additional procedure to improve knee stability if needed. Following an initial arthroscopic examination that confirms ACL rupture, the meniscal lesions are searched and treated. When possible, a meniscal suture is always carried out. A vertical central skin incision is made from the center of the patella to the tibial tubercle. The deep fascia is incised and divided to expose the patella tendon that is harvested 10 mm in width with patella (20 mm in length) and tibial (30 mm in length) bone plugs. During the harvesting, the knee is held in flexion so that the tendon fibers are straight due to tension. An oscillating saw is used to make the bone cuts, and in the professional athletes, we perform an oblique cut in order to avoid abnormal stress and the potential risk of patellar fracture. The tibial tunnel is drilled with the knee in full extension using a Howell tibial guide (Arthrotek Inc., Warsaw, IN, USA). An impingement rod is used to avoid the femoral roof to impinge on the graft and notchplasty performed, if necessary, with an abrader. The femoral tunnel is drilled through the tibial tunnel with the knee flexed at 90° on a pin guide located in the center of the anatomical ACL insertion (at 10 o'clock for right knee and 2 o'clock for left knee, 7 mm anterior to the posterior margin of the lateral femoral condyle) [26]. Sometimes it is difficult to reach the anatomical point with the femoral transtibial guide. In this case, after a first pin has positioned with the guide, a second pin is positioned lower, in the right place, with the aid of a cannulated transtibial corrector. Another possibility is to drill the femoral tunnel through the anteromedial portal or by an "Out-In" technique. In this case, a mini lateral skin incision is requested. The graft, armed at the patellar plug with XTendobutton (Smith & Nephew) and at the tibial plug with two sutures, is passed through the tunnels and the button flipped over the lateral femoral cortex. The knee is repeatedly extended and flexed to allow stress relaxation of the graft with the knee flexed at 20° and the graft is tensioned at 80 N and fixed with an absorbable interference screw in the tibial tunnel. In the case of marked anterolateral rotatory instability (Jerk Test 2 or 3+), we are currently

using a lateral additional procedure to improve knee stability. Our preferred techniques are the iliotibial band (ITB) extra-articular tenodesis as described by Noyes [27] or the procedure described by Christel and Djian [28]. In acute cases, if the *anterolateral ligament* (ALL) is torn, it can be repaired or refixed if an interstitial rupture is present. The anterolateral ligament is an important restraint to internal rotation of the knee, thus preventing the pivot shift phenomenon. The origin of the ALL is situated at the prominence of the lateral femoral epicondyle, slightly anterior to the origin of the lateral collateral ligament, and has an oblique course to the anterolateral aspect of the proximal tibia, with firm attachments to the lateral meniscus, thus enveloping the inferior lateral geniculate artery and vein. Its insertion on the anterolateral tibia is grossly located midway between Gerdy's tubercle and the tip of the fibular head, definitely separated from the iliotibial band (ITB) [23]. For its reconstruction, we use an isolated strip of ITT (10×70 mm) that we fix on the anatomical site of femur through a blind tunnel and interference absorbable screw and with suture anchor or a screw washer on the tibia.

41.5 Rehabilitation After ACL Reconstruction

The importance of a rehabilitation program cannot be underestimated, and although there is no one rehabilitation program proven to be superior to others, the speed and safety with which an athlete returns to play is more dependent upon the rehabilitation program than whether the patient had arthroscopically assisted or two-incision technique, or what type of graft or fixation was used. A clear, logical, responsive, and appropriately *aggressive rehabilitation* program is the key to returning an athlete to play as quickly and safely as possible. Rehabilitation has undergone a relatively rapid and global evolution over the past years. Traditionally, rehabilitation is divided into three distinct phases based on experimental study performed by Amiel et al. [29] on biological process of graft implanted into a joint, process called *ligamentization*. For this reason, after surgery, many rehabilitative protocols have relied on protection of the reconstructed ligament by limiting knee extension and weight bearing. Current rehabilitative programs following ACLr are now more aggressive than those utilized in the past. Presently, we employ two different rehabilitation programs for isolated ACL-reconstructed patients. The accelerated protocol is utilized for professional athletes, whereas recreational patient would follow a slower program, referred to as the regular rehabilitative program. The main difference between the two programs is the rate of progression through the various phases of rehabilitation and the period of time necessary prior to running and return to sports.

Our current program emphasizes full-passive knee extension, immediate motion, immediate full weight bearing [30], and functional exercises. This approach is due to the documented improved outcomes with more aggressive rehabilitation. We recommend discharging as soon as possible the use of postoperative brace in full extension. The brace is used while ambulating and sleeping only during the first 2 weeks after surgery. Lower extremity muscle weakness represents an unresolved problem after ACLr. Persistent muscular weakness is in part caused by a failure of voluntary activation by *arthrogenic muscle inhibition*. As demonstrated by Zech et al. [31], it is during the first postoperative month that voluntary activation and quadriceps weakness show the most significant deficit. For this reason, we believe that a more aggressive rehabilitative program is important mainly during the first postoperative period in order to reduce the muscular weakness. Strengthening is performed during this phase using both *closed-chain exercises* (*CKC*) and selective use of *open-chain exercises* (*OKC*). CKC exercises have been justified because they (1) increase tibiofemoral compressive forces, (2) increase co-contraction of the hamstrings, (3) mimic functional activities more closely than OKC exercises, and (4) reduce the incidence of patellofemoral complications. Many protocols do not recommend the OKC exercises because they could produce harmful forces on healing ACL graft [32, 33]. On contrary, clinical evidence has shown that the addition of OKC quadriceps training after ACLr results in a significantly better improvement in quadriceps torque without reducing knee-joint stability at 6 months and also leads to a significantly higher number of athletes returning to their previous activity earlier and at the same level as before injury [34, 35]. In our protocol, we prescribe OKC and CKC exercises from the early phase of rehabilitation, and athletes are encouraged to progress by increasing resistance or duration as soon as they are able to perform exercises without demonstrating an extension lag or discomfort. *Neuromuscular control drills* are gradually advanced to include dynamic stabilization and controlled perturbation training. Once strength and neuromuscular control have been demonstrated, functional activities such as running may begin. The last phase of ACL rehabilitation involves the restoration of function through sports-specific training for athletes returning to competition. Many of drills, such as cone drills, lunges with sport cords, plyometric drills, and the running and agility progression, can be modified for the specific functional movement patterns associated with the patient's unique sport. Some sports-specific running and agility drills include side shuffling, cariocas, sudden starts and stops, zigzags, 45° cutting, and 90° cutting.

In order to ensure a safe progression, testing is performed throughout all rehabilitative courses by providing the needed objective criteria for advancement. Testing procedures also follow a progression, which begins with basic measures and

progresses to functional tests of increasing difficulty that include sports-specific testing before returning to field play. Graft selection has some impact on our rehabilitation program following ACLr. With soft tissue grafts such as the quadrupled hamstring/semitendinosus graft, the rehabilitation program is initially less aggressive. Therefore, the return to sports is slightly slower, and we do not allow isolated hamstrings strengthening for approximately 8 weeks, to allow appropriate graft site healing to occur.

41.6 Return to Play

A recent systematic review has shown that there is a lack in the literature of information regarding criteria for approving patients back to their previous athletic activity. In most studies, the time after surgery is the only criterion to decide if a patient can *RTP*, and the majority of surgeons consider postoperatively the time frame of 6 months necessary to allow patients to return to competitive sports. Postoperative time seems to be relevant regarding the remodeling process of autogenous tendon grafts in order to achieve vascular supply and cellular ingrowth. However, the remodeling process is not the only relevant factor why an athlete should be released to unrestricted motion and return to play. Even with modern anatomical surgical techniques or rehabilitation programs, there is strong evidence that deficits in proprioception, balance, strength, and neuromuscular control persist for several months postoperatively. Therefore, objective assessment methods are necessary before release to unrestricted sports activities after ACLr, and no one single outcome criterion has been shown to correlate with successful return to sports. Most clinicians use a combination of criteria, namely, functional, clinical, and subjective testing. We do not release players until muscle and functional objective measurements have been achieved regardless of the amount of time that has passed since surgery. In our practice, we use two different criteria: the first criterion should include full range of motion, absence of swelling or pain, and passive or active stability. A more specific criterion includes three different types of evaluations: muscular, neuromuscular, and athletic. The quadriceps and hamstring strength is evaluated with the *MVIC* (maximal voluntary isometric contraction) and isokinetic evaluation. Among the functional tests, the most common test is the one-leg single hop for distance, whereas less common are the one-leg triple hop for distance, the one-leg timed hop, and the one-leg cross-over hop for distance. The one-leg single hop is an indicator of power, whereas the other three tests indicate both power and endurance. The vertical drop jump test is performed in athletes involved in specific activity that requires jumps. Assessment of static and dynamic balance may be performed using computerized instrumentation on the *Biodex Balance System* that provides a high degree of statistical validity and reliability in determining postural sway differences between limbs. Star excursion balance or *Y balance test* (SEBT) has been shown to be reliable and valid in determining postural deficits and is used to evaluate dynamic balance [36, 37]. For the functional on-field tests, we prescribe the *modified agility test* (*MAT*) and the pro-shuttle test. Van Grinsven et al. [38] recently conducted a systematic review of the rehabilitation literature to develop an evidence-based postoperative ACL program that would allow a return to athletics within 6 months. They recommended the following for return-to-sports criteria: full ROM, 85 % or greater on strength (quadriceps and hamstrings), and single-leg hop tests when compared with the opposite leg, less than 15 % deficit on hamstring-quadriceps strength ratio, no pain or swelling with sports-specific activities, and a stable knee in active situations.

41.7 Outcome

Despite many reports of successful outcomes following ACLr, recent reports have suggested that the rates of return to sport may not match the success of this surgery. Restoration of mechanical restraints is only the first step in achieving knee functional recovery, but factors including the patient's motivation and willingness to complete the prescribed rehabilitation program may play a role in influencing outcome. The percentage of patients who return to some level of sport may range from 26 to 97 %. In a recent systematic review on RTP after ACLr, it has been reported that 82 % of the patients returned to some sports participation, but only 63 % were participating in their pre-injury sport at follow-up. When competitive sport was considered, only 44 % were participating at follow-up [39]. Seto et al. reported that athletes who participated in sports involving cutting and twisting motions were less successful in returning to pre-injury activity level after ACLr [40]. Others suggest that competitive athletes return to pre-injury level of activity and sports-related function more quickly and successfully than nonathletes [41] with a rate of 94 % [42]. Ekstrand argues that the access to the most experienced orthopedic surgeons and physiotherapists, a rapid time to diagnosis and treatment, as well as high financial incentives in elite players could be possible reasons for these differences [43]. In contrast, Brophy et al. reported that at a mean follow-up of seven years, only 36 % of soccer players were still playing compared to the 72 % that had resumed play at some time following their ACLr [44]. The type of sport plays an important role in the different series examined. Shah et al. [45] reported that only 63 % of American Football players returned to game play at an average of 10.8 months after surgery. Age at time of surgery, position, and the type and number of procedures were not significantly different between those who did and did not

return to play [45]. The RTP percentage in a series of NBA players was recently reported in one series to be 78 % [46]. The risk of suffering a new knee injury is significantly higher in the group of players with a history of ACL injury. The risk of new knee injury, especially overuse injury, is significantly increased on return to elite athlete after ACL injury. After ACL tears occurring during official matches in Australian Rules football, a significantly higher risk (10-fold) for graft re-rupture during the first year after ACL reconstruction is reported [47]. Several studies [48–50] have reported high re-injury rates in athletes on return to activity. The contralateral knee is at risk for ACL rupture as well, and some studies have documented even a higher rate of this injury than a tear of the ACL-reconstructed knee [49, 50]. These studies suggest that in many patients, severe neuromuscular deficits may persist after ACL reconstruction. Various factors have been associated with an increased risk of ACL graft or contralateral ACL rupture, but the evidence is conflicting with some authors finding few, if any, identifiable risk factors [51]. The young age is considered an important factor of risk for the re-rupture of the graft, whereas with regard to contralateral ACL rupture, female gender [50] have been identified as risk factors, although there are also studies that did not identify gender as a risk factor [51, 52].

References

1. Boden BP, Griffin LY, Garrett WE (2000) Etiology and prevention of noncontact ACL injury. Phys Sportsmed 28:53–60
2. Griffin LY, Agel J, Albohm MJ et al (2000) Noncontact anterior cruciate ligament injuries: risk factors and prevention strategies. J Am Acad Orthop Surg 8:141–150
3. Shimokochi Y, Shultz SJ (2008) Mechanism of noncontact anterior cruciate ligament injury. J Athl Train 43:396–408
4. Kobayashi H, Kanamura T, Koshida S et al (2010) Mechanisms of the anterior cruciate ligament injury in sports activities: a twenty-year clinical research of 1,700 athletes. J Sports Sci Med 9:669–675
5. Olsen OE, Myklebust G, Engerbretsen L et al (2003) Relationship between floor type and risk of ACL injury in team handball. Scand J Med Sci Sports 13:299–304
6. Burkhart B, Ford KR, Myer GD et al (2008) Anterior cruciate ligament tear in an athlete: does increased heel loading contribute to ACL rupture? N Am J Sports Phys Ther 3:141–144
7. Ferretti A, Papandrea P, Conteduca F et al (1992) Knee ligament injuries in volleyball players. Am J Sports Med 20:203–207
8. McNair PJ, Marshall RN, Matheson JA (1990) Important features associated with acute anterior cruciate ligament injury. N Z Med J 103:537–539
9. Livesay GA, Rudy TW, Woo SL et al (1997) Evaluation of the effect of joint constraints on the in situ force distribution in the anterior cruciate ligament. J Orthop Res 15:278–284
10. Draganich LF, Vahey JW (1990) An in vitro study of anterior cruciate ligament strain induced by quadriceps and hamstring forces. J Orthop Res 8:57–63
11. Markolf KL, O'Neil G, Jackson SR et al (2004) Effects of applied quadriceps and hamstrings muscle loads on forces in the anterior and posterior cruciate ligaments. Am J Sports Med 32:1144–1149
12. Fung DT, Zhang LQ (2003) Modelling of ACL impingement against the intercondylar notch. Clin Biomech (Bristol, Avon) 18:933–941
13. Torg JS, Conrad W, Kalen W (1976) Clinical diagnosis of anterior cruciate ligament instability in the athlete. Am J Sports Med 4:84–93
14. Feagin JA, Cooke TD (1989) Prone examination for anterior cruciate ligament insufficiency. J Bone Joint Surg Br 71:863
15. Benjamine A, Gokeler A, van der Schans CP (2006) Clinical diagnosis of an anterior cruciate ligament rupture: a meta-analysis. J Orthop Sports Phys Ther 36:267–268
16. van Eck CF, van den Bekerom MPJ, Fu FH et al (2013) Methods to diagnose acute anterior cruciate ligament rupture: a meta-analysis of physical examinations with and without anaesthesia. Knee Surg Sports Traumatol Arthrosc 21:1895–1903
17. Collette M, Courville J, Forton M et al (2012) Objective evaluation of anterior knee laxity; comparison of the KT-1000 and GNRB arthrometers. Knee Surg Sports Traumatol Arthrosc 20:2233–2238
18. Robert H, Nouveau S, Gageot S et al (2009) A new knee arthrometer, the GNRB: experience in ACL complete and partial tears. Orthop Traumatol Surg Res 95:171–176
19. Spindler KP, Wright RW (2008) Clinical practice. Anterior cruciate ligament tear. N Engl J Med 359:2135–2142
20. Kosaka M, Nakase J, Toratani T et al (2013) Oblique coronal and oblique sagittal MRI for diagnosis of anterior cruciate ligament tears and evaluation of anterior cruciate ligament remnant tissue. Knee 21:54–57
21. Dimond PM, Fadale PD, Hulstyn MJ et al (1998) A comparison of MRI findings in patients with acute and chronic ACL tears. Am J Knee Surg 11:153–159
22. Irvine GB, Dias JJ, Finlay DBL (1987) Segond fractures of the lateral tibial condyle: brief report. J Bone Joint Surg Br 69: 613–614
23. Vincent JP, Magnussen RA, Gezmez F et al (2012) The anterolateral ligament of the human knee: an anatomic and histologic study. Knee Surg Sports Traumatol Arthrosc 20:147–152
24. Pujol N, Colombet P, Cucurulo T et al (2012) Natural history of partial anterior cruciate ligament tears: a systematic literature review. Orthop Traumatol Surg Res 98(8 Suppl):160–164
25. Andernord D, Karlsson J, Musahl V et al (2013) Timing of surgery of the anterior cruciate ligament. Arthroscopy 29:1863–1871
26. Cain EL Jr, Clancy WG (2002) Anatomic endoscopic anterior cruciate ligament reconstruction with patella tendon autograft. Orthop Clin North Am 33:717–725
27. Noyes FR (2010) ITB extra-articular tenodesis. In: Noyes FR (ed) Noyes' knee disorders: surgery, rehabilitation, clinical outcomes. Saunders Elsevier, Philadelphia, pp 170–172
28. Christel P, Djian P (2002) Plastie extra articulaire antero latérale du genou utilisant une ténodèse courte au fascia-lata. Rev Chir Orthop Reparatrice Appar Mot 88:508–513
29. Amiel D, Kleiner JB, Roux RD et al (1986) The phenomenon of "ligamentization": anterior cruciate ligament reconstruction with autogenous patellar tendon. J Orthop Res 4:162–172
30. Tyler TF, McHugh MP, Gleim GW et al (1998) The effect of immediate weightbearing after anterior cruciate ligament reconstruction. Clin Orthop 357:141–148
31. Zech A, Awiswu F, Pfeifer K (2009) Longitudinal changes of neuromuscular quadriceps function after reconstruction of the anterior cruciate ligament. Curr Orthop Pract 20:276–280
32. Renström P, Arms SW, Stanwyck TS et al (1986) Strain within the anterior cruciate ligament during hamstring and quadriceps activity. Am J Sports Med 14:83–87
33. Grood ES, Suntay WJ, Noyes FR et al (1984) Biomechanics of the knee-extension exercises. Effects of cutting the anterior cruciate ligament. J Bone Joint Surg Am 66:725–734

34. Perry MC, Morrissey MC, King JB et al (2005) Effects of closed versus open kinetic chain knee extensor resistance training on knee laxity and leg function in patients during the 8- to 14-week post-operative period after anterior cruciate ligament reconstruction. Knee Surg Sports Traumatol Arthrosc 13:357–369
35. Mikkelsen C, Werner S, Eriksson E (2000) Closed kinetic chain alone compared to combined open and closed kinetic chain exercises for quadriceps strengthening after anterior cruciate ligament reconstruction with respect to return to sports: a prospective matched follow-up study. Knee Surg Sports Traumatol Arthrosc 8:337–342
36. Kinzey S, Armstrong C (1998) The reliability of the star-excursion test in assessing dynamic balance. J Orthop Sports Phys Ther 27:356–360
37. Plisky P, Rauh M, Kaminski TW (2006) Star excursion balance test as a predictor of lower extremity injury in high school basketball players. J Orthop Sports Phys Ther 36:911–919
38. van Grinsven S, van Cingel RE, Holla CJ et al (2010) Evidence-based rehabilitation following anterior cruciate ligament reconstruction. Knee Surg Sports Traumatol Arthrosc 18:1128–1144
39. Ardern CL, Webster KE, Taylor NF, Feller JA (2011) Return to sport following anterior cruciate ligament reconstruction surgery: a systematic review and meta-analysis of the state of play. Br J Sports Med 45:596–606
40. Seto JL, Orofino AS, Morrissey MC et al (1988) Assessment of quadriceps/hamstring strength, knee ligament stability, functional and sports activity levels five years after anterior cruciate ligament reconstruction. Am J Sports Med 16:170–180
41. Smith FW, Rosenlund EA, Aune AK et al (2004) Subjective functional assessments and the return to competitive sport after anterior cruciate ligament reconstruction. Br J Sports Med 38:279–284
42. Waldén M, Hägglund M, Magnusson H et al (2011) Anterior cruciate ligament injury in elite football: a prospective three-cohort study. Knee Surg Sports Traumatol Arthrosc 19:11–19
43. Ekstrand J (2011) A 94 % return to elite level football after ACL surgery: a proof of possibilities with optimal caretaking or a sign of knee abuse? Knee Surg Sports Traumatol Arthrosc 19:1–2
44. Brophy RH, Schmitz L, Wright RW et al (2012) Return to play and future ACL injury risk after ACL reconstruction in soccer athletes from the Multicenter Orthopaedic Outcomes Network (MOON) group. Am J Sports Med 40:2517–2522
45. Shah VM, Andrews JR, Fleisig GS et al (2010) Return to play after anterior cruciate ligament reconstruction in National Football league athletes. Am J Sports Med 38:2233–2239
46. Busfield BT, Kharrazi FD, Starkey C et al (2009) Performance outcomes of anterior cruciate ligament reconstruction in the National Basketball Association. Arthroscopy 25:825–830
47. Orchard J, Seward H, McGivern J et al (2001) Intrinsic and extrinsic risk factors for anterior cruciate ligament injury in Australian footballers. Am J Sports Med 29:196–200
48. Hui C, Salmon LJ, Kok A et al (2011) Fifteen-year outcome of endoscopic anterior cruciate ligament reconstruction with patellar tendon autograft for "isolated" anterior cruciate ligament tear. Am J Sports Med 39:89–98
49. Salmon LJ, Russell VJ, Refshauge K (2006) Long-term outcome of endoscopic anterior cruciate ligament reconstruction with patellar tendon autograft: minimum 13-year review. Am J Sports Med 34:721–732
50. Shelbourne KD, Gray T, Haro M (2009) Incidence of subsequent injury to either knee within 5 years after anterior cruciate ligament reconstruction with patellar tendon autograft. Am J Sports Med 37:246–251
51. Pinczewski LA, Lyman J, Salmon LJ et al (2007) A 10-year comparison of anterior cruciate ligament reconstructions with hamstring tendon and patellar tendon autograft: a controlled, prospective trial. Am J Sports Med 35:564–574
52. Wiger P, Brandsson S, Kartus J et al (1999) A comparison of results after arthroscopic anterior cruciate ligament reconstruction in female and male competitive athletes. A two- to five-year follow-up of 429 patients. Scand J Med Sci Sports 9:290–295

Revision Anterior Cruciate Ligament

42

Paolo Adravanti and Stefano Nicoletti

42.1 Introduction

The anterior cruciate ligament (ACL), of all ligaments in the human body, is the one most often ruptured, especially among the population practicing sports. The incidence of ACL rupture is 36.9–60.9 per 100,000 persons in the United States [1], where over 200,000 ACL ruptures are surgically reconstructed annually [2]. The rising number of primary ACL reconstructions, coupled with expected functional outcomes in high-functional-demand patients after ACL reconstruction, has led to an increase in revision ACL reconstructions. Key to correct anatomical reconstruction and functional outcome after primary repair is a thorough knowledge of ACL anatomy and biomechanics. High-functional-demand patients returning to sports with suboptimal knee joint function are at increased risk of recurrent ACL tear, chondral and meniscal damage, and progression of knee joint arthrosis [3, 4]. Though primary surgery does not always restore normal joint kinematics [5], improvement in joint stability and recovery of knee function [6] are seen in most patients, with good to excellent outcomes achieved in 80–90 % of cases [7–9]. Failure rates (recurrence of instability) range from 4 to 10 % [10–13], and results after revision ACL repair are decidedly lower, particularly in elite athletes [13–19]. Hence, the reasons for successful outcome after revision ACL reconstruction hinge on a full understanding of why the primary repair failed.

P. Adravanti, MD (✉) • S. Nicoletti, MD
Department of Orthopaedic, Clinic Città di Parma, Parma, Italy
e-mail: info@paoloadravanti.com; ste.nicoletti@libero.it

42.2 Complications and Failures in ACL Reconstruction

The complications ensuing from ACL reconstruction are multifactorial and can sometimes lead to graft failure and recurrence of instability. One of the most common complaints is postsurgical knee joint stiffness. Loss of motion after ACL repair occurs in 11–35 % of cases [20]. Reduced flexion may be due to arthrofibrosis, particularly when ACL repair is performed within 4 weeks after injury to the ligament or when knee joint inflammation with reduced motion is present before the operation [21, 22]; this latter study reported loss of extension after ACL repair in about 25 % of cases.

Also, a malpositioned tunnel can cause loss of knee joint motion. Excessively anterior tibial tunnel placement limits extension due to impingement with the roof of the intercondylar notch during extension, which may also give rise to localized anterior arthrofibrosis (cyclops lesion) [23, 24]. A tibial tunnel placed too posterior may lead to impingement of the implant with the posterior cruciate ligament, resulting in diminished flexion [24]. Furthermore, excessively anterior placement of the femoral tunnel may place excessive tension on the implant during flexion, with a consequent reduction in flexion, or elongation of the new ligament with a gradual loss in function [24]. In cases of postsurgical joint stiffness resulting from arthrofibrosis or implant malposition, arthroscopic arthrolysis may be required to remove the adherences and the new ligament, thus setting the stage for recurrence. Revision is performed following full recovery of joint motion.

Although septic arthritis following arthroscopic surgery is rare (incidence <1 %) [25, 26], it can cause graft failure and require its removal when early targeted antibiotic therapy and arthroscopic lavage are ineffective in resolving the infection.

Published failure rates vary from 3 to 52 %, depending on the criteria used to define failure [27]. Over 40 % of elite athletes undergoing ACL reconstruction do not return to their pre-operation level of athletic activity [13, 28]. According to a meta-analysis, although patients' subjective

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_42,

reports were optimal overall, residual instability was noted in 32 % of cases at the Lachman test and in 22 % at the pivot shift test [29]. Recurrent knee joint instability appears to be more common in males; a familial predisposition to ACL tears increases the risk of recurrence, irrespective of the type of graft implanted and the risk of ACL rupture in the contralateral ACL [30].

42.3 Recurrence of Knee Joint Instability

The main causes of recurrent instability after ACL reconstruction may be classified in three groups and they may occur in isolation or interact with one another: technical surgical error, new injury, and lack of graft incorporation into the surrounding tissue (biological failure) [31]. Failures may also be classified as early if they occur within 6 months of the operation or late if they occur any time thereafter [31]. Technical surgical errors are the most common cause of failure [24, 27, 32, 33], particularly in early recurrences occurring within the first year of athletic activity. However, new injury has been cited as the main cause of repeat rupture by numerous studies [13, 34–36], including the Danish ACL Reconstruction Registry, which reported new injury as being more frequent than surgical error (36.2 % vs. 21.5 %) in causing repeat ACL tears [37]. In addition, the data from the Multicenter ACL Revision Study (MARS) indicate that the cause of failure is multifactorial and that injury is more often the cause than surgical error: injury in 32 % of cases, surgical error in 24 %, biological incorporation failure in 7 %, and a combination of all of the above in 37 % [38].

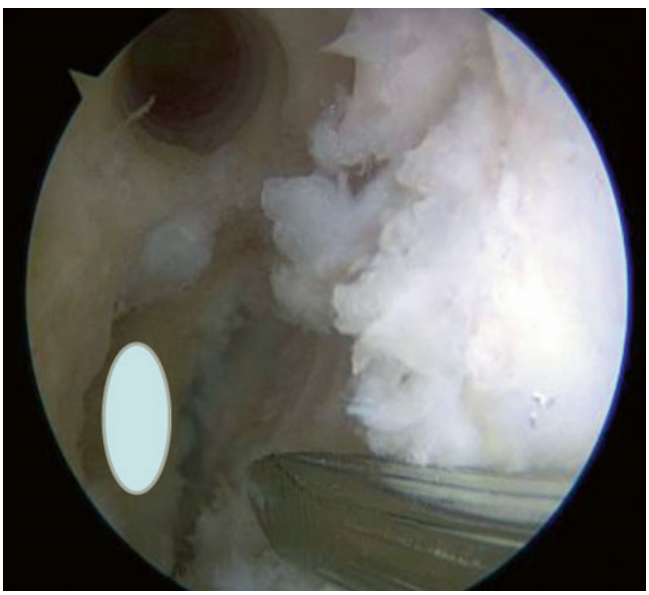


Fig. 42.1 Nonanatomical position of the femoral tunnel and the new correct position (*white circle*)

The most common technical error leading to ACL graft elongation is tunnel malposition, particularly nonanatomical position (Fig. 42.1) of the femoral tunnel [24, 38–42], which is three times more frequent than nonanatomical tibial tunnel placement [32]. Recurrence due to new injury is more common in people practicing sports; in the United States, the sports associated with greater risk are basketball, American football, and soccer [38].

42.4 Clinical and Instrumental Diagnosis

While clinical examination of recurrent anterior knee joint instability is identical to that for primary ACL rupture, a foremost concern is understanding what were the causes, besides injury, so that surgical treatment can be planned appropriately. Recurrence may be due to having missed peripheral ligament damage during primary reconstruction or because of axial malalignment with graft elongation over time. The incidence of such failures varies from 2.8 to 31 % [27, 36, 43]. The most commonly missed peripheral lesions are posteromedial and posterolateral injuries in particular [44]. If associated with previous total or partial medial meniscectomy, posterolateral injury can lead to varus angulation and progressive elongation of the new ACL, resulting in ligament deficiency. In such cases, revision should be combined with valgus-producing tibial osteotomy to avert repeat recurrence [45]. Another option that may be considered is meniscal graft placement when the varus deformity is related to the lack of the internal meniscus. Such approaches, however, will not satisfy the needs of professional athletes who want rapid return to sports activity.

Plain and weight-bearing radiographs are important for identifying metal implants that may interfere with the placement of a new tunnel, to evaluate tunnel position (Fig. 42.2) and any axial malalignment. Computed tomography is useful for quantifying bone tunnel enlargement, which may be over- or underestimated on plain radiographs [46, 47]. Examination will include magnetic resonance imaging (MRI) to evaluate the state of the tunnel, other ligaments, menisci, chondral tissue, and all joint compartments.

In addition to physical examination and imaging studies, preoperative work-up will include thorough history taking to collect information on the onset of symptoms, the time since primary surgery, the extent of new injury, the type of rehabilitation received, and the type of sport the patient engages in. Collectively, this information provides essential clues to understanding whether the recurrence is due to biological failure, injury, or surgical error. Also useful is having the patient's medical records at hand to review the type of graft implanted, the type of implantation technique, the

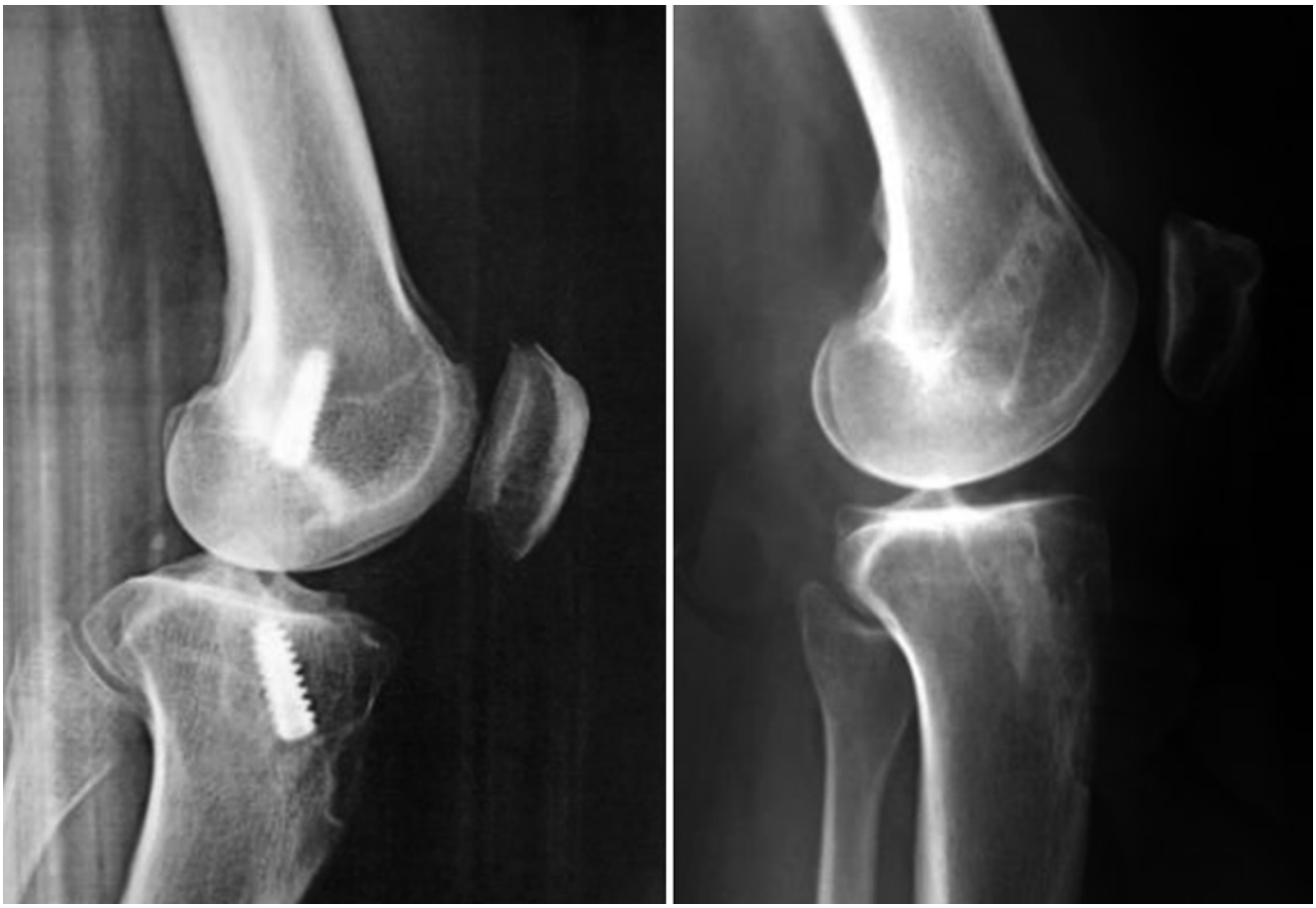


Fig. 42.2 X-ray standard to evaluate tunnel position (incorrect)

preoperative condition of the menisci and cartilage, and the type of fixation material used.

42.5 Graft Selection

Graft selection in revision ACL reconstruction depends on many factors, including the tendon used in the primary repair procedure, tunnel position and widening, peripheral laxity, and the surgeon's experience. Both autografts and allografts may be used. The most commonly used autografts are the patellar tendon, the gracilis and semitendinosus tendons, and the quadriceps tendon. The advantage of using the patellar tendon is that it can be harvested together with a large tibial wedge to fill tunnel enlargement. Though reuse of the patellar tendon from the primary surgery was once proposed [48], various authors have advised against employing this technique because of the tendon's diminished mechanical properties and the higher risk of complications such as patellar fracture and tendon rupture [49]. The quadriceps tendon has the same mechanical properties as the patellar tendon but is associated with a higher risk of patellar fracture when the patellar tendon has been used. The pes anserinus tendons offer the advantage that harvesting creates fewer problems at the harvest graft site; however, because they are relatively small, they cannot be used when a preexisting anatomic tunnel will be used and when the patellar tendon and a tunnel (diameter, 10–11 mm) were employed in the primary surgery. Again, because they are small, pes anserinus tendons cannot be used in tunnel enlargement; however, they do permit easier creation of a new tunnel when the old tunnel is malpositioned, and there is less space to create a new tunnel in a correct anatomical position. The literature reports no differences in results with the use of patellar or hamstring tendons, as for primary reconstructions.

Allografts have the distinct advantages that there is no harvest graft site morbidity and that they allow for multiple ligament reconstructions and, especially important, large bone gaps from tunnel enlargement can be filled. Though there do not appear to be any significant differences between the use of allografts and autografts [50], failure rates with soft tissue allografts were higher in young athletes [51]. Because biological incorporation of allografts is slower [52] and the failure rate is higher in elite athletes, we advise against their use as a first choice in graft selection. In the United States,

allografts are used in just over 50 % of revision ACL reconstructions and patellar grafts in 50 % [38]. Generally, we prefer using ipsi- or contralateral autografts, reserving allografts for repeat revision, associated ligament damage, and when large bone wedges are needed for tunnel enlargement.

42.6 Treatment Strategy

Revision ACL reconstruction can be performed as a two-stage or more often using a single-stage technique, the preferred of the two, because of the longer time needed for bone defect filling (3–5 months after bone grafting of the tunnel on average) and the higher risk of meniscal tears and chondral degeneration, with rapid progression of arthrosis [27, 53, 54]. The two-stage technique should be reserved for cases in which there is evidence on preoperative computed tomography or during surgery of excessive widening of ≥16–17 mm in diameter of one or both tunnels [55].

The less anatomic the tunnel position, the easier it will be to create an anatomically correct tunnel. New tunnels will have a vital spongious bone wall that will promote incorporation of the new graft (Fig. 42.3). We believe that, in order to create a new femoral tunnel, the surgeon will need to be familiar with the anteromedial and the outside-in techniques. Both portal techniques allow the tibial and femoral tunnels to be drilled independently for achieving an anatomically more correct tunnel position [56]. Particularly for the femoral bone, if a preexisting tunnel has been placed with the transtibial approach, a completely new tunnel can be created along its entire length. If the old femoral tunnel is malpositioned but close enough to the anatomical position, the outside-in technique is preferable since it is more likely to allow for the creation of a new tunnel with a correct exit point, which may be the same as the aperture of the old tunnel but having a completely different direction. The technique we apply entails reaming over the guide wire in both outside-in and anteromedial portal reconstructions at about

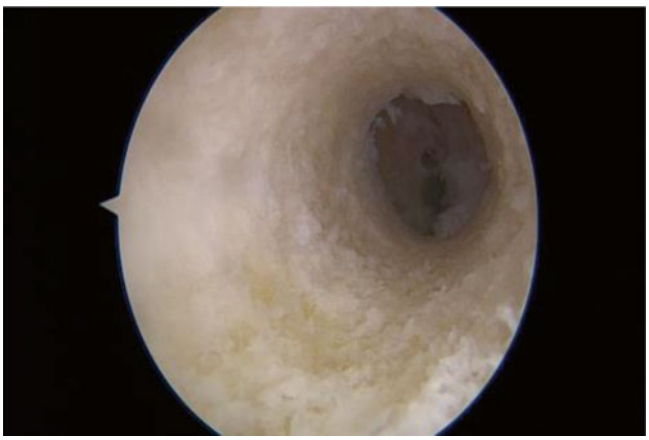


Fig. 42.3 New tunnels will have a vital spongious bone wall

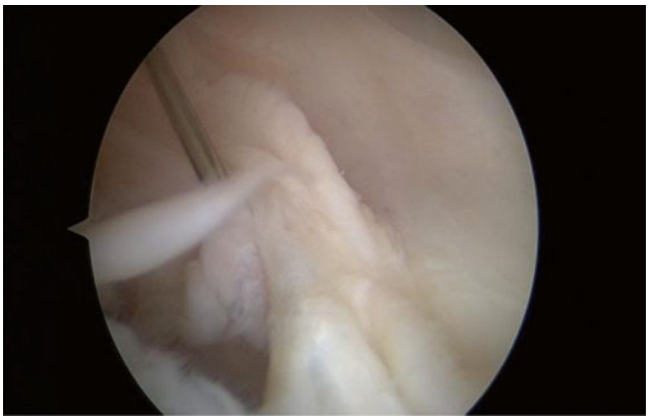


Fig. 42.4 Posteromedial meniscocapsular separation

6 mm in diameter less than the final diameter. The tunnel is then widened by free-hand reaming to the diameter of the previously prepared graft. Using increasingly larger diameter reamers permits approximation of the correct tunnel position until the definitive tunnel diameter has been created and allows for correcting tunnel placement, as determined by knee joint flexion-extension (anteromedial technique) and free-hand reamer direction without the guide wire [40]. Use of the medial port in both techniques permits complete visualization of the entire medial wall of the lateral condyle and precise positioning of the tunnel.

Assuming that anatomic placement of the femoral tunnel during primary reconstruction surgery will become increasingly precise, we will likely encounter anatomically placed tunnels in the future. In such cases, we will need to approach the old tunnel via the anteromedial or outside-in technique but drilling to a larger diameter to remove bone and obtain a tunnel wall with vital tissue that will promote integration of the new graft. For this reason, it will be better to use a patellar graft with a bone wedge that can be shaped to the new tunnel or to use an allograft.

One of the major problems is the tibial tunnel. Creating a completely new tibial tunnel is practically impossible because of overlap, at least at the exit point. A similar challenging situation is a preexisting tunnel aperture that is too posterior. Therefore, even if we have created a new, more anteriorly and anatomically correct tunnel, the exit points will overlap and position the new graft posteriorly, raising the risk of renewed failure. To overcome this problem, we can fill the posterior end of the tunnel with a bone dowel and perform a two-stage revision or revision with a double-bundle technique and tibial tunnel widening [55].

Synthesis materials should be removed if they induce hypersensitivity reactions or interfere with the new tunnel. If located near the new tunnel, they should be left in place to avert wall rupture and tunnel enlargement.

In recurrence of knee joint instability with moderate to severe ligament laxity (pivot shift test score 3+), external extra-articular plasty should be performed, for which we pre-

fer the Coker-Arnold procedure, or anterolateral ligament reconstruction considered.

An additional concern is deterioration of the secondary stabilizers in recurrences due to nontraumatic graft elongation. Posteromedial meniscocapsular separation (Fig. 42.4) must be repaired because the posterior horn of the medial meniscus and the tibial meniscal ligament serve as secondary stabilizers of anterior tibial translation when the ACL is absent. In chronic laxity, as in nonfunctional grafts, there is progressive detensioning, leading to rupture of the meniscocapsular insertion or of the posteromedial meniscotibial ligament [31]. Such injuries should always be repaired to prevent excessive strain on the graft, together with retensioning of the posteromedial corner, which can be easily performed using femoral or tibial suture anchors, depending on the type of lesion.

42.7 Rehabilitation and Return to Sports

Over 40 % of patients do not return to their pre-injury athletic activity level after repeat ACL reconstruction [13, 15, 36, 41]. Chondral and meniscal damage affects the capacity for recovery, and improvement after repeat ACL reconstruction is less favorable than after primary surgery. Except for a few case series, there are no studies involving only professional athletes [13]. In revisions with placement of a new tunnel, the rehabilitation protocol is the same as for primary reconstruction since we can rely on the primary fixation. If there are problems with tunnel enlargement, slow rehabilitation at least for the first month postsurgery is advisable to reduce the risk of further enlargement and biological failure. In peripheral reconstructions, the rehabilitation protocol should be adapted to the type of arthroplasty performed. We use the same rehabilitation protocol after external extra-articular plasty as after ACL reconstruction. Early rehabilitation after primary ACL surgery is important, whereas the use of braces affords no extra advantage. A slow rehabilitation program after revision ACL reconstruction is recommended; in many cases, bracing can aid in facilitating graft integration. The timing of return to sports should be discussed with the patient since it varies and depends on muscle recovery, psychological factors, neuromuscular control, and knee joint stability.

References

1. Giannotti SM, Marshall SW, Hume PA, Bunt L (2009) Incidence of anterior cruciate ligament injury and other knee ligament injuries: a national population-based study. J Sci Med Sport 12(6):622–627
2. Parkkari J, Pasanen K, Mattila VM, Kannus P, Rimpela A (2008) The risk for a cruciate ligament injury of the knee in adolescents and young adults: a population-based cohort study of 46 500 people with a 9 year follow-up. Br J Sports Med 42(6):422–426
3. Bach BR Jr (2003) Revision anterior cruciate ligament surgery. Arthroscopy 19(Suppl 1):14–29
4. Mullaji AB, Marawar SV, Luthra M (2008) Tibial articular cartilage wear in varus osteoarthritic knees: correlation with anterior cruciate ligament integrity and severity of deformity. J Arthroplasty 23(1):128–135
5. Biau DJ, Katsahian S, Kartus J, Harilainen A, Feller JA, Sajovic M, Ejerhed L, Zaffagnini S, Ropke M, Nizard R (2009) Patellar tendon versus hamstring tendon autografts for reconstructing the anterior cruciate ligament: a meta-analysis based on individual patient data. Am J Sports Med 37(12):2470–2478
6. Biau DJ, Tournoux C, Katsahian S et al (2007) ACL reconstruction: a meta-analysis of functional scores. Clin Orthop Relat Res 458: 180–187
7. Chouteau J, Benareau I, Testa R, Fessy MH, Lerat JL, Moyen B (2008) Comparative study of knee anterior cruciate ligament reconstruction with or without fluoroscopic assistance: a prospective study of 73 cases. Arch Orthop Trauma Surg 128(9):945–950
8. Corry IS, Webb JM, Clingeleffer AJ, Pinczewski LA (1999) Arthroscopic reconstruction of the anterior cruciate ligament: a comparison of patellar tendon autograft and four-strand hamstring tendon autograft. Am J Sports Med 27(4):444–454
9. Freedman KB, D'Amato MJ, Nedeff DD, Kaz A, Bach BR Jr (2003) Arthroscopic anterior cruciate ligament reconstruction: a meta-analysis comparing patellar tendon and hamstring tendon autografts. Am J Sports Med 31(1):2–11
10. Feller JA, Webster KE (2003) A randomized comparison of patellar tendon and hamstring tendon anterior cruciate ligament reconstruction. Am J Sports Med 31(4):564–573
11. Liden M, Ejerhed L, Sernert N, Naxdal G, Kartus J (2007) Patellar tendon or semitendinosus tendon autografts for anterior cruciate ligament reconstruction: a prospective, randomized study with a 7-year follow-up. Am J Sports Med 35(5):740–748
12. Carlisle JC, Parker RD, Matava MJ (2007) Technical considerations in revision anterior cruciate ligament surgery. J Knee Surg 20(4):312–322
13. Grossman MG, Elattrache NS, Shield CL, Glousman RE (2005) Revision anterior cruciate ligament reconstruction: three to nine year follow-up. Arthroscopy 21(4):418–423
14. Mayr R, Rosenberger R, Agraharam D, Smekal V, El Attal R (2012) Revision anterior cruciate ligament reconstruction: an update. Arch Orthop Trauma Surg 132:1299–1313
15. Denti M, Lo Vetere D, Bait C, Schonhuber H, Melegati G, Volpi P (2008) Revision anterior cruciate ligament reconstruction: causes of failure, surgical technique and clinical results. Am J Sports Med 36(10):1896–1902
16. Griffith TB, Allen BJ, Levy BA, Stuart MJ, Dahm DL (2013) Outcomes of repeat revision anterior cruciate ligament reconstruction. Am J Sports Med 41(6):1296–1301
17. Wright R, Spindler K, Huston L, Amendola A, Andrish J, Brophy R, Carey J, Cox C, Flanigan D, Jones M et al (2011) Revision ACL reconstruction outcomes: MOON cohort. J Knee Surg 24(4): 289–294
18. Wright RW, Gill CS, Chen L, Brophy RH, Matava MJ, Smith MV, Mall N (2012) Outcome of revision ACL reconstruction: a systematic review. J Bone Joint Surg Am 94:531–536
19. Gifstad T, Drogset JO, Viset A, Grontvedt T, Hortemo GS (2013) Inferior results after revision ACL reconstructions: a comparison with primary ACL reconstructions. Knee Surg Sports Traumatol Arthrosc 21:2011–2018
20. Harner CD, Irrgang JJ, Paul J, Dearwater S, Fu FH (1992) Loss of motion after anterior cruciate ligament reconstruction. Am J Sports Med 20(5):499–506
21. Mayr HO, Weig TG, Plitz W (2004) Arthrofibrosis following ACL reconstruction – reasons and outcome. Arch Orthop Trauma Surg 124(8):518–522
22. Mauro CS, Irrgang JJ, Williams BA, Harner CD (2008) Loss of extension following anterior cruciate ligament reconstruction: anal-

ysis of incidence and etiology using IKDC criteria. Arthroscopy 24(2):146–153
23. van Dijk RA, Saris DB, Willems JW, Fievez AW (2008) Additional surgery after anterior cruciate ligament reconstruction: can we improve technical aspects of the initial procedure? Arthroscopy 24(1):88–95
24. Carson EW, Anisko EM, Restrepo C, Panariello RA, O'Brien SJ, Warren RF (2004) Revision anterior cruciate ligament reconstruction: etiology of failure and clinical results. J Knee Surg 17(3):127–132
25. Wang C, Ao Y, Wang J, Hu Y, Cui G, Yu J (2009) Septic arthritis after arthroscopy ACL reconstruction: a retrospective analysis of incidence, presentation, treatment and cause. Arthroscopy 25(3): 243–249
26. Sonnery-Cottet B, Archbold P, Zayni R, Bortolletto J, Thaunat M, Prost T, Padua VB, Chambat P (2011) Prevalence of septic arthritis after ACL reconstruction among professional athletes. Am J Sports Med 39(11):2371–2376
27. Diamantopoulos AP, Lorbach O, Paessler HH et al (2008) Anterior cruciate ligament reconstruction: results in 107 patients. Am J Sports Med 36:851–860
28. Salmon LJ, Pinczewski LA, Russel VJ (2006) Revision anterior cruciate ligament reconstruction with hamstring tendon autograft: 5 to 9 year follow-up. Am J Sports Med 34:1604–1614
29. Biau DJ, Tournoux C, Katsahian S, Schranz PJ, Nizard RS (2006) Bone patellar tendon-bone autografts versus hamstring autografts for reconstruction of anterior cruciate ligament: meta-analysis. BMJ 332(7548):995–1001
30. Bourke HE, Salmon LJ, Waller A et al (2012) Survival of the anterior cruciate ligament graft and contralateral ACL at a minimum of 15 years. Am J Sports Med 40:1985–1992
31. Harner CD, Giffin JR, Dunteman RC, Annunziata CC, Friedman MJ (2001) Evaluation and treatment of recurrent instability after anterior cruciate ligament reconstruction. Instr Course Lect 50: 463–474
32. Trojani C, Sbihi A, Djian P, Potel JF, Hulet C, Jouve F, Bussiere C, Ehkirch FP, Burdin G, Dubrana F, Beaufils P, Franceschi JP, Chassaing V, Colombet P, Neyret P (2011) Causes of failure of ACL reconstruction and influence of meniscectomies after revision. Knee Surg Sports Traumatol Arthrosc 19(2):196–201
33. Johnson DL, Swenson TM, Irrgang JJ, Fu FH, Harner CD (1996) Revision anterior cruciate ligament surgery: experience from Pittsburgh. Clin Orthop Relat Res 325:100–109
34. O'Neill DB (2001) Arthroscopically assisted reconstruction of the anterior cruciate ligament. A follow-up report. J Bone Joint Surg Am 83-A(9):1329–1332
35. Shelbourne KD, O'Shea JJ (2002) Revision anterior cruciate ligament reconstruction using the contralateral bone-patellar tendon-bone graft. Instr Course Lect 51:343–346
36. Noyes FR, Barber-Westin SD (2001) Revision anterior cruciate surgery with use of bone-patellar tendon-bone autogenous grafts. J Bone Joint Surg Am 83-A(8):1131–1143
37. Lind M, Menhert F, Pedersen AB (2009) The first results from the Danish ACL reconstruction registry: epidemiologic and 2 year follow-up results from 5818 knee ligament reconstructions. Knee Surg Sports Traumatol Arthrosc 17(2):117–124
38. Wright RW, Huston LJ, Spindler KP, Dunn WR, Haas AK, Allen CR, Cooper DE, DeBerardino TM, Lantz BB, Mann BJ, Stuart MJ (2010) Descriptive epidemiology of the Multicenter ACL Revision Study (MARS) cohort. Am J Sports Med 38(10):1979–1986
39. Morgan JA, Dahm D, Levy B, Stuart MJ (2012) Femoral tunnel malposition in ACL revision reconstruction. J Knee Surg 25(5): 361–368
40. Yong MA, Ying-fang AO, Jia-kuo Y, Ling-hui DAI, Zhen-xing SHAO (2013) Failed anterior cruciate ligament reconstruction: analysis of factors leading to instability after primary surgery. Chin Med J 126(2):280–285
41. Garofalo R, Djahangiri A, Siegrist O (2006) Revision anterior cruciate ligament reconstruction with quadriceps tendon-patellar bone autograft. Arthroscopy 22(2):205–214
42. Taggart TF, Kumar A, Bickerstaff DR (2004) Revision anterior cruciate ligament reconstruction: a midterm patient assessment. Knee 11(1):29–36
43. Noyes FR, Barber-Westin SD (2006) Revision anterior cruciate surgery with using a 2 stage technique with bone grafting of the tibial tunnel. Am J Sports Med 34(4):678–680
44. Fanelli GC, Edson CJ, Maish DR (2001) Revision anterior cruciate ligament reconstruction: associated patholaxity, tibio-femoral malalignment, rehabilitation, and results. Am J Knee Surg 14(3):201–204
45. Noyes FR, Barber SD, Simon R (1993) High tibial osteotomy and ligament reconstruction in varus angulated, anterior cruciate ligament-deficient knees. A two to seven year follow-up study. Am J Sports Med 21(1):2–12
46. Merchant MH Jr, Willimon SC, Vinson E, Pietrobon R, Garrett WE, Higgins LD (2010) Comparison of plain radiography, computed tomography, and magnetic resonance imaging in the evaluation of bone tunnel widening after anterior cruciate ligament reconstruction. Knee Surg Sports Traumatol Arthrosc 18(8):1059–1064
47. Hoser C, Tecklenburg K, Kuenzel KH, Fink C (2005) Postoperative evaluation of femoral tunnel position in ACL reconstruction: plain radiography versus computed tomography. Knee Surg Sports Traumatol Arthrosc 13(4):256–262
48. Karns DJ, Heidt RS, Holladay BR, Colosimo AJ (1994) Case report: revision anterior cruciate ligament reconstruction. Arthroscopy 10:148–157
49. Kartus J, Stener S, Lindhal S, Eriksson BL, Karlsson J (1998) Ipsi- or contralateral patellar tendon graft in anterior cruciate ligament reconstruction. Am J Sports Med 26:499–504
50. Carey JL, Dunn WR, Dahm DL, Zeger SL, Spindler KB (2009) A systematic review of anterior cruciate ligament reconstruction with autograft compared with allograft. J Bone Joint Surg Am 91(9):2242–2250
51. Borchers JR, Pedroza A, Kaeding C (2009) Activity level and graft type as risk factors for anterior cruciate ligament graft failure: a case-control study. Am J Sports Med 37(12):2362–2367
52. Harner CD, Lo MY (2009) Future of allografts in sport medicine. Clin Sports Med 28(2):327–340
53. Thomas NP, Kankate R, Wandless F, Pandit H (2005) Revision anterior cruciate ligament reconstruction using a 2-stage technique with bone grafting of the tibial tunnel. Am J Sports Med 33(11): 1701–1709
54. Ohly NE, Murray IR, Keating JF (2007) Revision anterior cruciate ligament reconstruction: timing of surgery and the incidence of meniscal tears and degenerative change. J Bone Joint Surg Br 89(8):1051–1054
55. Cheatham SA, Johnson DL (2013) Anticipating problems unique to revision ACL surgery. Sports Med Arthrosc Rev 21(2):129–134
56. Silva A, Sampaio R, Pinto E (2012) ACL reconstruction: comparison between transtibial and anteromedial portal techniques. Knee Surg Sports Traumatol Arthrosc 20(5):896–903

Posterior Cruciate Ligament

43

Enrico Arnaldi, Andrea Bruno, and Jacopo Tamini

43.1 Anatomy

The *posterior cruciate ligament* (*PCL*) is an intra-articular structure, surrounded by synovial membrane, and it provides about 95 % of the total resistance to posterior translation of the tibia. Its tensile strength is nearly twice that of the ACL [1]. Different studies demonstrated that, just like the ACL, PCL is composed of two different bundles: anterolateral (AL) and posteromedial (PM), which have different functions during knee movement [2, 3]. PCL has been described to be from 32 to 38 mm long and to have a cross-sectional area of 11 mm [4]. Femoral insertion of the PCL is located on the lateral side of the medial femoral condyle, in an area which is much larger than the ligament's thickness: AL bundle insertion starts from the medial intercondylar ridge, 13 mm posterior to the medial articular cartilage-intercondylar wall interface and 13 mm inferior to the articular cartilage-intercondylar roof interface [5]. PM bundle insertion is located 8 mm posterior to the medial articular cartilage-intercondylar wall interface and 20 mm inferior to the articular cartilage-intercondylar roof interface [5]. The tibial insertion is located posteriorly to the posterior horn of the medial meniscus, 10–15 mm below the articular surface, around 7 mm anteriorly to the posterior tibial cortex [6, 7]. Just anteriorly to the PCL passes *Humphrey's ligament* (also called anterior meniscofemoral ligament), connecting the posterior horn of the lateral meniscus to the medial femoral condyle. The same structures are connected also by the posterior meniscofemoral ligament (*Wrisberg's ligament*), which is located posterior to the PCL. These two ligaments may serve as a secondary stabilizer in a PCL-deficient knee [8]. PCL is mainly vascularized by the middle geniculate artery and innervated by articular branches of the tibial nerve [9].

E. Arnaldi • A. Bruno (✉) • J. Tamini
Arthroscopic and Reconstructive Knee surgery,
Istituto Clinico Humanitas, Rozzano, Via Manzoni 56,
Rozzano (MI) 20089, Italy
e-mail: arnaenri@gmail.com; andrea.bruno@humanitas.it;
jacopotamini@gmail.com

43.2 Biomechanic

PCL biomechanic has been a hard challenge for years. Different studies in the past have shown the AL bundle to be extended during knee flexion and laxed during knee extension, while the PM bundle to be extended during knee extension and laxed during knee flexion [10, 11]. This led to the idea that the two bundles had an independent function, with the AL working in flexion and the PM working in extension. New studies changed this kind of vision, which nowadays has been substituted with a more synergical one. During flexion, actually, it is true that the AL bundle becomes tighter, but it becomes also more vertically oriented, losing its ability to resist posterior tibial translation. The PM bundle, instead, during flexion becomes more horizontally oriented, acquiring ability in posterior stabilization [10–12]. It is nowadays clear that none of the two PCL bundles acts independently in restraining posterior tibial translation at any degree of knee flexion.

43.3 Epidemiology

PCL injury is rarely an isolated lesion. It is more commonly associated with other lesions in patients with multiligamentous rupture.

The incidence of PCL rupture in literature goes from 1 to 44 % of all acute knee ligament injuries [13–17].

It was reported that up to 2 % of NFL professional players are found to have a PCL laxity, and they are usually unaware about this lesion [18].

The most frequent multiligamentous injuries involving the PCL are PCL-PLC (posterolateral corner), followed by PCL-ACL and PCL-PMC (posteromedial corner) [19].

43.4 Injury Mechanism

The most common injury causes are high-energy trauma like road traffic accidents (*dashboard* injury and motorcycle accidents), which are responsible for about 45 % of PCL

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_43,

tears. The second most frequent cause of lesion is sports injuries (40 %), which are usually responsible for low-energy traumatic ruptures [20]. Different mechanisms of injury are described:

- Posteriorly directed trauma on the proximal tibia (as dashboard injuries or the fall onto a flexed knee with foot in plantar flexion)
- Knee hyperflexion injuries
- Forced knee hyperextension (classic ACL + PCL injury)
- Rotational injury with varus or valgus stress (classic PCL + MCL/LCL)

43.5 Clinical and Diagnostic Examination

PCL tears are not always easy to discover, especially chronic lesions, because they can sometimes be asymptomatic.

Unlike ACL injuries, PCL isolated lesions usually cause a gradual and slow swelling over the first days, patients are usually able to bear weight on the injured knee, and especially for isolated tears, they rarely complain a sense of knee instability during daily life activities.

Patients with PCL injuries may complain generalized knee pain, not being able to identify the kind of problem and its location.

In patients with chronic PCL-deficient knees, pain with prolonged walking and descending stairs is the main issue and is localized to the medial side or patellofemoral joint [19].

A standard examination should be executed, with attention to eventual joint effusion, patient's gait pattern, and extremity alignment.

There are specific tests for investigating PCL tears:

- Posterior sag sign: patient supine with knees flexed 90°. In case of lesion, the tibia of the affected leg subluxates posteriorly (it may be negative in acute setting due to muscular contraction or swelling).
- Godfrey test: modification of posterior sag sign test. Both hip and knee flexed 90°.
- *Posterior drawer*: performed at 90° of knee flexion. In a stable knee, the anterior border of the tibial plateau is about 1 cm anterior to the femoral condyles.
  Results are graded basing on the tibia position and on its posterior translation during the test.
  Grade I: the tibia is located anteriorly to the medial femoral condyle, and it can be translated in a range of 0–0.5 cm posterior to the femoral condyle.
  Grade II: the tibia is located at the same level of the medial femoral condyle, and it can be translated in a range of 0.5–1 cm posterior to the femoral condyle.
  Grade III: the tibia is displaced posteriorly to the medial femoral condyle, and it can be translated more than 1 cm posterior to the femoral condyle [21]. Grade III is usually found in associated PCL-PLC lesions.

Posterior drawer test is 90 % sensitive and 99 % specific for the diagnosis of PCL injury [22].

- Quadriceps active test: knee flexed between 70 and 90°. In case of lesion, quadriceps contraction anteriorly translates tibia to its normal position.
- *Dial test*: the foot's angle is measured with an external rotation force applied with knees flexed 30° and 90°. If the angle on the affected side is more than 10–15° greater than the opposite side at 30° of flexion only, it suggests an isolated PLC injury; if the difference is found when the test is performed at 90° of flexion only, it suggests an isolated PCL injury; when the test is positive at both 30 and 90° of knee flexion, the diagnosis is a combined PCL and PLC injury [15, 23, 24].
- Reverse pivot shift: knee flexed and extrarotated is extended with a valgus stress. Positive if a sensation of reduction is appreciated.

It is always mandatory to clinically evaluate every other ligamentous structure of the knee when a PCL tear is found (ACL, PLC, MCL, LCL).

Imaging diagnostic exams should also be performed to complete the diagnosis:

- Radiographic evaluation: it is necessary for patients with an acute trauma to exclude bone lesions such as fractures of the tibial plateau, femoral condyles, or patella. It may also show a bone avulsion from tibial PCL insertion.
  When the clinical tests are not clear and the surgeon is not sure about the PCL integrity, a lateral radiography with tibial posterior stress (both with commercial stress systems and with maximum hamstring contraction) can demonstrate posterior tibial translation.
  In chronic lesion arthritic changes could develop.
- MRI: very accurate imaging exam (99 %), especially in the sagittal views [15]. It is indicated to have the confirmation of PCL lesion when there is a clinical suspect, but it is also useful for diagnosing associated lesions, even if bone bruise and meniscal tears are less commonly associated with PCL than they are with ACL [17]. Less sensitive in differentiating complete tears from partial tears [25]
- TC: has a minor role as for other ligamentous lesions but can be used to better evaluate every bone lesion such as tibial spine fractures or tibial plateau bone avulsion

43.6 Conservative Management

The natural history of untreated isolated PCL tears is still unclear. Patients may clinically tolerate an isolated PCL-deficient knee and may not appreciate any disability initially; that is why there was a general consensus that isolated tears do well with conservative treatment. Prospective studies with 2–5 years of follow-up showed that the majority of the patients treated conservatively had good subjective results; half were

able to return to their sport at the same or higher level [17, 26]. Nevertheless biomechanical studies showed that PCL deficiency results in altered loads and kinematics during functional activities [27–29]. Three specific phases have been described after PCL injuries. A survey of nonoperatively treated isolated PCL injuries at 15 and 25 years of follow-up shows a high incidence of arthrosis, especially in the medial and patellofemoral compartments as well as meniscal tear. At 15 years, 89 % had persistent pain and 50 % had chronic effusions [30]. At 25 years, almost all had degenerative changes [30]. The first is the adaptation stage which ranges from 3 to 18 months, the second is the functional tolerance stage that ranges from 15 to 20 years, and the final phase involves osteoarthritic degeneration and usually occurs after 25 years [30, 31]. The natural history of PCL-deficient knee is to develop overtime degenerative changes in the medial and patellofemoral compartments.

The goal of the rehabilitation program is to strengthen the musculature about the knee while minimizing forces across the patellofemoral and tibiofemoral compartments. In theory, quadriceps strengthening may compensate enough for loss of PCL [32]. Tibiofemoral compression forces are reduced with closed kinetic chain exercise; open kinetic chain quadriceps exercises exert an anterior pull on the tibia [33]. In our experience, acute grade I isolated PCL injuries are treated nonoperatively and could also be advocated for many grade II injuries. Individual treatment decision for an isolated grade II PCL injury is based on surgeon experience and patients factors and desires (usually high demanding athletes). Conservative management for grade I and II PCL injuries consist in pain relief, rest, ice, elevation regime, and immobilization of the knee with a brace locked in full extension 2–4 weeks, which will result in a decreased tension on the anterolateral bundle crutches and strengthening of the quadriceps muscle [34]. It is advisable to place within the brace a pad under the tibia to counter the force of gravity [35]. Conservative management of grade III PCL injuries could still be possible and is done by immobilizing the knee in full extension for 2–4 weeks. The patient is then allowed to progressive weight bearing with active, assisted range of motion exercises and quadriceps strengthening [20]. Return to sport is generally permitted when full quadriceps strength is regained, which usually takes 6–12 months. In spite of his rehabilitation program, progressive degeneration of the knee may be inevitable [30]. Nevertheless it is still not demonstrated whether the new advances in surgical reconstruction of PCL could change the natural history of this degenerative process.

43.7 Surgical Indications

Indications for surgical treatment of *acute PCL injuries* include insertion site avulsions with greater than grade I laxity, a decrease in tibial step of 8 mm or greater, and PCL tears combined with other structural injuries. Indications for surgical treatment of *chronic PCL injuries* are when an isolated PCL tear becomes symptomatic or when progressive functional instability develops. The symptoms are pain and swelling, but not instability as with the anterior cruciate-deficient knee. Initially the patient complains of patellofemoral pain, and then later on there is increased wear of the medial compartment.

43.8 Surgical Timing

The surgical timing should be considered in the context of the individual patient. Besides more than 50 % PCL injuries occur in multiple trauma patient [36], surgical timing is dependent upon vascular status, skin condition, systemic injuries, open versus closed knee injury, meniscus and cartilage, other orthopedic injuries, and the collateral/capsular ligaments involved. Multiple ligament knee injuries may require staged procedures (first acute capsular collateral structure repair and a delayed ACL/PCL reconstruction) [32].

Acute PCL reconstruction performed between 2 and 3 weeks post injury allows sealing of capsular tissues to permit an arthroscopic approach. Delayed PCL reconstruction may be indicated for patients below 60 years of age with persistent symptoms of pain and instability. These may help to rebalance pressure and shear forces within the knee, slowing progression of medial compartment degeneration, and the reduction of the posterior tibial translation restores the mechanical advantage of the quadriceps mechanism, thereby reducing patellofemoral contact forces as well [19]. Before performing a delayed PCL reconstruction, a thorough examination of the limb alignment and of the joint degeneration is mandatory. A long-standing weight-bearing x-ray AP (hip to ankle), lateral at 30° of knee flexion, and patellofemoral axial views are recommended. In case of a symptomatic varus malalignment a high tibial osteotomy should be performed first. In case of severe arthrosis a PCL reconstruction is not still indicated.

43.9 Graft Selection

Autograft from the same or the opposite side as well as allograft could be chosen. The autografts that could be used are quadriceps tendon, patellar tendon, and hamstrings. Allografts are Achilles tendon, patellar tendon, quadriceps tendon, hamstrings tendon, and tibialis tendon. The most popular are Achilles tendon allograft, which is the senior author's first choice, and quadriceps tendon autograft. These two grafts have a bone block at one end. Placing the bone block into the tibial tunnel eliminates the *killer tunnel angle* and looks like a posterior inlay graft [37].

43.10 Surgical Treatment

43.10.1 Avulsion Fragments

Fixation with a screw via a posterior approach is recommended for larger fragments; arthroscopic fixation with suture is also possible [38].

Whatever the surgical technique chosen, the goal of posterior cruciate ligament is reconstruction are to identify and treat all pathology (especially posterolateral and posteromedial instability), utilize strong graft material, accurately place tunnels in anatomic insertion sites, minimize graft bending, a proper graft tensioning, a strong fixation and employ appropriate postoperative rehabilitation program [20, 39].

The recommended surgical approaches are as follows:

1. All inside: transtibial single bundle and transtibial double bundle
2. Tibial inlay

43.10.2 The Transtibial Single-Bundle PCL Technique

The transtibial *single-bundle PCL* is the traditional and most common reconstruction and is the senior author's preferred procedure. The surgeon must clearly identify the PCL posterior tibial attachment site to place the tibial tunnel in an anatomic position and must avoid penetration into the posterior capsule and subsequent damage to the neurovascular structures. The goal is to place the guide pin in the distal central portion of the PCL fossa, 20–25 mm from the proximal entrance to leave 15 mm of tibial bone proximal to the tunnel to prevent anterior graft migration.

The following are safety procedures to avoid neurovascular injuries:

1. Clearly identify the whole PCL fossa.
2. Use a PCl guide protector and drill slowly with direct viewing of the guide pin (a fluoroscopy check can be used).
3. The ream of the final portion of the tunnel is done by hand.

Femoral tunnel placement is done in the anterior half of the femoral PCL footprint. The edge of the tunnel should be 2–3 mm from the articular cartilage, so the center of the tunnel, depending from the graft diameter, is placed 7–8 mm from the cartilage edge. The tunnel could be drilled from the AM portal or outside-in using the subvastus technique which is the senior author's choice. If the tunnel is placed too distal, the graft will be subject to high tensile forces in flexion resulting in a lack of flexion; otherwise a too proximal tunnel placement results in a slacken graft in flexion with tibial posterior subluxation. When pulling the graft out from the tibial tunnel into the femoral tunnel to avoid impingement of the graft on the superior edge of the tibial tunnel, a trocar is placed from the posteromedial portal between the graft and the tunnel superior edge to act as a pulley, pulling up and backward the graft while it is driven into the femoral tunnel.

The graft is fixed in the femoral tunnel with a nonmetal interference screw placed from outside-in. The graft is tensioned through several knee flexion-extension cycles. The tibial bone plug is fixed with an interference screw while the knee is flexed to 90°, and an anterior drawer is applied to the tibia.

The advantages of transtibial PCL reconstruction are: familiar technique of drilling the tibia, possibility to use soft tissue graft, and the use of spatula type tibial guides that reduces the risk of neurovascular injuries. The disadvantages of transtibial PCL reconstruction are as follows: risks for neurovascular injury and killer tunnel turn. This describes the sharp graft angulation that occurs when the graft winds around the posterior tibia. The killer turn could lead to graft elongation, fraying, and failure of the graft. To decrease soft tissue graft abrasion at the tibial tunnel, the bone portion of the graft is placed in a tibial tunnel directly adjacent to the tunnel exit. Also chamfering the tibial tunnel exit with a rasp results in decreasing the graft abrasion effect.

43.10.3 The Double-Bundle Technique

Biomechanical studies show that the *double-bundle* technique is able to restore normal knee laxity across the full range of flexion and may closely mimic the intact knee posterior tibial translation and PCL in situ forces with measurable benefits in rotational and posterior stability. The tibial tunnel is performed as the former technique. Two femoral tunnels are performed. The anterior tunnel is centered 6 mm deep to the articular cartilage and the posterior one 8 mm proximal to the articular cartilage edge. A 2–3 mm bone bridge is left between the two tunnels.

43.10.4 The Posterior Inlay Technique

The *posterior inlay* technique was felt to be an improvement as it avoided the "killer tunnel angle" at the posterior edge of the tibia. The tibial bone plug is secured at the PCL tibial footprint to avoid excessive graft abrasion. Cadaver lab study showed an attenuation and thinning of a transtibial graft as it passes around the back of the tibia [40]. The posterior inlay graft does not have this problem and should be able to survive the posterior force of gravity as well as the forces applied during the cyclic loading of the graft.

The advantages of the posterior inlay technique are the reduction of the killer tunnel turn and the reduction of the risk of neurovascular injury.

The disadvantages of the posterior inlay technique are that this procedure could be technically demanding, the

patient must be turned to expose the back of the tibia, and the large posterior arthrotomy may reduce the knee stability.

43.10.5 Innovations

An innovation is the split stacked Achilles tendon allograft that allows a longer bone plug and more collagen and results in a higher load to failure (40 % stronger 1,040 N vs 660 N) [41].

In order to reduce the "killer turn" at the back of the tibia, the tibial tunnel could be drilled from the lateral side of the tibia [42]; an augmentation of the PCL remnant (often the posteromedial bundle) through a transseptal approach seems to reduce attenuation of the graft around the back of the tibia [43].

A very interesting innovation is the *all-inside PCL reconstruction* or arthroscopic PCL inlay that could be an excellent compromise [44].

43.11 Postoperative Management

The *rehabilitation* approach is more conservative than the postoperative management after ACL reconstruction. The PCL is the primary static stabilizer of the knee, and allowing weight bearing during the immediate postoperative period could cause the failure of the graft [11].

The postoperative program is designed to progressively increase load and enhance the functional abilities with minimal exposure to injury risk positions [45]:

Weeks 0–5. Non-weight bearing and use of a long brace locked in full extension with a posterior tibial pad, cryotherapy, manual patella mobilization, ankle pumps, quad sets, and elevation.

Weeks 5–10. The knee brace is unlocked; partial weight bearing (20 % of body weight) with crutches, short-arc and long-arc knee extension, and passive knee flexion to target 90° of flexion by the end of the week 10.

Weeks 10–16. The brace is discontinued; closed chain exercises in a 0–60° range. Target 110° of flexion at month 4. Proprioception exercises.

Month 4–6. Straight-line jogging could be started if adequate strength and proprioception are obtained; aggressive quadriceps strengthening and a plyometric program could be slowly started at month 5. Isolated hamstring strengthening first against gravity.

Month 6–12. Continue with the above program.

43.12 Return to Sport

The return to sport is a multifactorial decision and needs a careful balance of the athletes' desire to return versus objective measure of their function and lingering impairments.

Usually it is considered post-op 6–7 months. The patient should achieve symmetrical strength and proprioception before returning to sport and of the uninvolved leg with all functional tests. It is important to include objective and measurable criteria like single-leg timed hop for distance and single-leg vertical jump for height.

A functional brace during sport could be helpful until the patient reaches 18 months post-op.

43.13 Clinical Outcomes

Clinical studies do not succeed in showing which reconstruction technique is the best. Surgical treatment provides improvement in subjective scores but does not fully restore the native biomechanics, and a residual laxity may result. Most patients will make a predictable return to recreational and athletic activity, although surgery does not restore normal stability and does not seem to prevent degenerative arthritis [46].

There is no difference in surgical outcomes between transtibial and inlay tibial fixation [39, 43, 47, 48].

The double-bundle technique may improve rotational stability, but there is no study demonstrating clinical superiority of a single-bundle or a double-bundle PCL reconstruction [39, 46, 49–52].

References

1. Kennedy JC, Hawkins RJ, Willis RB et al (1976) Tension studies of human knee ligaments: yield point, ultimate failure and disruption of the cruciate and tibial collateral ligaments. J Bone Joint Surg Am 58:350
2. Makris CA, Georgoulis AD, Papageorgiou CD, Moebius UG, Soucacos PN (2000) Posterior cruciate ligament architecture: evaluation under microsurgical dissection. Arthroscopy 16(6):627–632
3. Matava MJ, Ellis E, Gruber B (2009) Surgical treatment of posterior cruciate ligament tears: an evolving technique. J Am Acad Orthop Surg 17(7):435–446
4. Harner CD, Baek GH, Vogrin TM, Carlin GJ, Kashiwaguchi S, Woo SL (1999) Quantitative analysis of human cruciate ligament insertions. Arthroscopy 15(7):741–749
5. Morgan CD, Kalman VR, Grawl DM (1997) The anatomic origin of the posterior cruciate ligament: where is it?-reference landmarks for PCL reconstruction. Arthroscopy 13(3):325–331
6. Edwards A, Bull AM, Amis AA (2007) The attachments of the fiber bundles of the posterior cruciate ligament: an anatomic study. Arthroscopy 23(3):284–290
7. Moorman CT 3rd, Murphy Zane MS, Bansai S et al (2008) Tibial insertion of the posterior cruciate ligament: a sagittal plane analysis using gross, histologic, and radiographic methods. Arthroscopy 24(3):269–275
8. Clancy WG, Shelbourne KD, Zoellner GB et al (1983) Treatment of knee joint instability secondary to rupture of the posterior cruciate ligament. J Bone Joint Surg Am 65:310
9. Petersen W, Tillmann B (1999) Blood and lymph supply of the posterior cruciate ligament: a cadaver study. Knee Surg Sports Traumatol Arthrosc 7:42–50

10. Girgis FG, Marshall JL, Monajem A (1975) The cruciate ligaments of the knee joint: anatomical, functional and experimental analysis. Clin Orthop Relat Res 106:216–231
11. Van Dommelon BA, Fowler PJ (1989) Anatomy of the posterior cruciate ligament. A review. Am J Sports Med 17:24–29
12. Ahmad CS, Cohen ZA, Levine WN, Gardner TR, Ateshian GA, Mow VC (2003) Codominance of the individual posterior cruciate ligament bundles: an analysis of bundle lengths and orientation. Am J Sports Med 31(2):221–225
13. Fanelli GC (1993) Posterior cruciate ligament injuries in trauma patients. Arthroscopy 9:291–294
14. Fanelli GC, Edson CJ (1995) Posterior cruciate injuries in trauma patients. Part II. Arthroscopy 11:526–529
15. Harner CD, Hoher J (1998) Evaluation and treatment of posterior cruciate ligament injuries. Am J Sports Med 26:471–482
16. Lobenhoffer P, Lattermann C, Krettek C, Blauth M, Tscherne H (1996) Rupture of the posterior cruciate ligament: current status of treatment (in German). Unfallchirurg 99:382–399
17. Shelbourne KD, Davis TJ, Patel DV (1999) The natural history of acute isolated, nonoperatively treated posterior cruciate ligament injuries. A prospective study. Am J Sports Med 27:276–283
18. Andrews JR, Edwards JC, Satterwhite YE (1994) Isolated posterior cruciate ligament injuries. History, mechanism of injury, physical findings, an ancillary tests. Clin Sports Med 13:519–530
19. Lopez-Vidriero E, Simon DA, Johnson DH (2010) Initial evaluation of posterior cruciate ligament injuries: history, physical examination, imaging studies, surgical and nonsurgical indications. Sports Med Arthrosc Rev 18:230–237
20. Rigby JM, Porter KM (2010) Posterior cruciate ligament injuries. Trauma 12:175–281
21. Petrie RS, Harner CD (1999) Evaluation and management of the posterior cruciate ligament injured knee. Oper Tech Sports Med 7:93–103
22. Hughston JC, Norwood LA Jr (1980) The posterolateral drawer test and external rotational recurvatum test for posterolateral rotatory instability of the knee. Clin Orthop Relat Res 147:82–87
23. Wind WM Jr, Bergfeld JA, Parker RD (2004) Evaluation and treatment of posterior cruciate ligament injuries: revisited. Am J Sports Med 32:1765–1775
24. Lubowitz JH, Bernardini BJ, Reid JB 3rd (2008) Current concepts review: comprehensive physical examination for instability of the knee. Am J Sports Med 36:577–594
25. Patten RM, Richardson ML, Zink-Brody G et al (1994) Complete versus partial-thickness tears of the posterior cruciate ligament: MR findings. J Comput Assist Tomogr 18:793–799
26. Parolie JM, Bergfeld JA (1986) Long-term results of nonoperative treatment of isolated posterior cruciate ligament injuries in the athlete. Am J Sports Med 14:35–38
27. Kozanek M, Fu EC, Van de Elde SK, Gill TJ, Li G (2009) Posterolateral structures of the knee in posterior cruciate ligament deficiency. Am J Sport Med 37(3):534–541
28. Logan M, Williams A, Lavelle J, Gedroyc W, Freeman M (2004) The effect of posterior cruciate ligament deficiency on knee kinematics. Am J Sport Med 32(8):1915–1922
29. Van de Velde SK, Bingham JT, Gill TJ, Li G (2009) Analysis of tibiofemoral cartilage deformation in the posterior cruciate ligament-deficient knee. J Bone Joint Surg Am 91(1):24–29
30. Dejour H, Walch G, Peyrot J et al (1988) The natural history of rupture of the posterior cruciate ligament. Fr J Orthop Surg 2:112–120
31. Covey DC, Sapega AA, Sherman GM (1996) Testing for isometry during reconstruction of the posterior cruciate ligament. Anatomic and biomechanical considerations. Am J Sport Med 24:740–746
32. Fanelli GC, Beck JD, Craig j (2010) Current concepts review: the posterior cruciate ligament. J Knee Surg 23(2):61–72
33. Lutz GE, Palmitier RA, An KN, Chao EYS (1993) Comparison of tibiofemoral joint forces during open-kinetic-chain and closed-kinetic chain exercises. J Bone Joint Surg Am 75:732–739
34. Cosgarea AJ, Jay PR (2001) Posterior cruciate ligament injuries: evaluation and management. J Am Acad Orthop Surg 9:297–307
35. Johnson DH (2009) My approach to posterior cruciate ligament injuries. Oper Tech Sports Med 17:167–174
36. Shulz MS, Russe K, Weiler A, Eichhorn HJ, Strobel MJ (2003) Epidemiology of posterior cruciate ligament injuries. Arch Orthop Trauma Surg 123:186–191
37. Markolf K, Davies M, Zoric B, McAllister D (2003) Effects of bone block position and orientation within the tibial tunnel for posterior cruciate ligament graft reconstructions: a cyclic loading study of bone-patellar tendon-bone allografts. Am J Sports Med 31:673–679
38. Zhao J, He Y, Wang J (2006) Arthroscopic treatment of acute tibial avulsion fracture of the posterior cruciate ligament with suture fixation technique through Y-shaped bone tunnels. Arthroscopy 22(2):172–181
39. Kim SJ, Kim TE, Jo SB, Kung YP (2009) Comparison of the clinical results of three posterior cruciate ligament reconstruction techniques. J Bone Joint Surg Am 91(11):2543–2549
40. Bergfeld JA et al (2001) A biomechanical comparison of posterior cruciate ligament reconstruction techniques. Am J Sports Med 29(2):129–136
41. DeBerardino TM, Lonergan KT, Brooks DE (2008) Comparison of the split stacked versus the split achilles allograft for dual femoral tunnel posterior cruciate ligament reconstruction. Am J Sports Med 36(1):142–148
42. Ohkoshi YN, Yamamoto S, Shibata K, Ishida N, Hashimoto R, Yamane TS (2003) Description of a new endoscopic posterior cruciate ligament reconstruction and comparison with a 2-incision technique. Arthroscopy 19(8):825–832
43. Ahn JH, Yang HS, Jeong WK, Kohn KH (2006) Arthroscopic transtibial posterior cruciate ligament reconstruction with preservation of posterior cruciate ligament fibers: clinical results of minimum 2-year follow-up. Am J Sports Med 34(2):194–204
44. Salata MJ, Sekiya JK (2011) Arthroscopic posterior cruciate ligament tibial inlay reconstruction: a surgical technique that may influence rehabilitation. Sports Health 3:52–58
45. Edson CJ, Fanelli GC, Beck JD (2010) Postoperative rehabilitation of the posterior cruciate ligament. Sports Med Arthrosc Rev 18:275–289
46. Kim YM, Lee CA, Matava MJ (2011) Clinical results of arthroscopic single-bundle transtibial posterior cruciate ligament reconstruction: a systematic review. Am J Sports Med 39(2):425–434
47. MacGilvray JD, Stein BE, Park M, Allen AA, Wickiewicz TL, Warren RF (2006) Comparison of tibial inlay versus transtibial techniques for isolated posterior cruciate ligament reconstruction: minimum 2-year follow-up. Arthroscopy 22(3):320–328
48. Noyes FR, barber-Westin S (2002) Posterior cruciate ligament replacement with a two-strand quadriceps tendon-patellar bone autograft and a tibial inlay technique. J Bone Joint Surg Am 84(6):938–944
49. Houe T, Jorgensen U (2004) Arthroscopic posterior cruciate ligament reconstruction: one- vs two-tunnel technique. Scand J Med Sci Sports 14(2):107–111
50. Kohen RB, Sekiya JK (2009) Single-bundle versus double-bundle posterior cruciate ligament reconstruction. Arthroscopy 25(12): 1470–1477
51. Wang CJ, Chan YS, Weng LH, Yuan LJ, Chen HS (2004) Comparison of autogenous and allogenous posterior cruciate ligament reconstructions of the knee. Injury 35(12):1279–1285
52. Wang CJ, Weng LH, Hsu CC, Chan YS (2004) Arthroscopic single- versus double-bundle posterior cruciate ligament reconstruction using hamstring autograft. Injury 35(12):1293–1299

Posterolateral Instability

44

C. Zorzi, V. Condello, V. Madonna, G. Piovan, and D. Screpis

44.1 Introduction

Posterolateral corner complex (PLC) plays a critical role in maintaining knee stability. It acts synergistically with the posterior cruciate ligament [1, 2] providing posterior translation, varus, and external rotation stability.

Isolated injuries to the posterolateral corner are very uncommon, occurring in less than 2 % of all ligamentous injuries of the knee [3]. This kind of lesions are usually included within the context of complex knee injuries, especially in association with anterior cruciate ligament (ACL) or, more frequently, posterior cruciate ligament (PCL) injuries [4–8]. According to LaPrade et al., the incidence of high-grade posterolateral knee injuries in patients with an acute knee injury with a hemarthrosis is 9.1 % [9]. Lee and Jung demonstrated the presence of posterolateral complex lesions in 60–80 % of cases of injuries to the PCL [10].

In the last decades, the study of PLC structures has gained growing importance because of the complex instability generated by its injury, as well as the higher failure rates after ACL and PCL reconstruction surgery with concomitant PLC lesions. In fact, posterolateral rotatory instability (PLRI) is frequently unrecognized or underestimated, especially when associated with PCL injuries [11], also because of the difficult diagnosis both clinical and radiological. Some studies reported that undiagnosed or untreated PLC injuries are one of the most important factors in recurrence of instability after PCL reconstruction [12].

C. Zorzi (✉) • V. Condello • V. Madonna
Divisione Ortopedica, Ospedale Sacro Cuore Don Calabria, Negrar (VR), Italy
e-mail: claudio.zorzi@sacrocuore.it

G. Piovan
Clinica Ortopedica, University of Trieste, Trieste, Italy

D. Screpis
Clinica Ortopedica, University of Perugia, Perugia (PG), Italy

44.2 Anatomy and Biomechanics

Anatomically, the posterolateral corner is a complex arrangement of muscles, tendons, and ligaments [13].

Seebacher et al. in 1982 [14] described the knee lateral structures as being composed of three distinct layers; however, there does not seem to be standardization of the layers, due to the high anatomic variability in the PLC and competing nomenclature in the literature [13, 15, 16].

- Superficial Layer: lateral fascia, iliotibial tract, and biceps tendon
- Middle Layer: patellar retinaculum, patellofemoral, and patellomeniscal ligaments
- Deep Layer: fibular collateral ligament (FCL), lateral meniscotibial ligament, popliteus muscle and tendon, popliteofibular ligament (PFL), arcuate ligament, fabellofibular ligament, and lateral joint capsule with its attachment to the lateral meniscus edge

It is possible to classify each component of the PLC in passive stabilizers, such as capsular and noncapsular ligaments (FCL, PFL, posterolateral joint capsule, arcuate ligament complex, and fabellofibular ligament), and dynamic stabilizers, which are the musculotendinous units and their aponeuroses (popliteus complex, iliotibial band, lateral head of gastrocnemius, and biceps femoris tendons). The PLC provides stability against posterolateral rotation and varus displacement; it plays the greater role in restricting posterolateral rotation, whereas the FCL alone is the major restraint to varus displacement of the tibia.

44.3 Injury Mechanism

The majority of PLC injuries are primarily due to athletic activity participation, falls, or road traffic collisions [17]. Many specific mechanisms of injury are described in the literature [18, 19]:

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_44,

- Direct blow to the medial aspect of the proximal tibia in a fully extended knee, with the force directed in a posterolateral direction or external rotation
- Hyperextension injury
- Anterior rotatory dislocations: varus stress and hyperextension
- Posterior rotatory dislocation: varus stress, posteriorly directed blow to a proximal tibia in flexion (dashboard injury)
- Forceful deceleration while the distal leg is planted
- Abrupt external rotation of the extended knee

44.4 Clinical and Diagnostic Examination

Although various physical examination tests are described for the diagnosis of PLC lesions, in 72 % of cases, these lesions are not identified at their initial presentation, which shows the difficulty in both performing these tests and interpreting the results. The difficulty increases significantly when central pivot and medial collateral ligament injuries are associated [20].

In chronic lesions, a varus thrust gait due to a lateral laxity of the knee may be present, especially in patients with an underlying varus limb alignment. The varus thrust gait pattern is likely associated with a lift-off of the lateral compartment of the knee, which has been shown to increase medial compartment joint stresses [4, 21].

Several clinical tests have been developed to diagnose PLRI:

- Varus stress test at 0° and 30°: positivity at 0° of flexion reflects a serious posterolateral injury with a high probability of associated cruciate ligament lesion. Positivity at 30° is more suggestive of partial tears or complete tears of the posterolateral structures.
- Recurvatum and external rotation: lifting the leg by the great toe shows hyperextension associated with external rotation of the injured knee.
- Posterolateral drawer test: the knee is kept at 90° of flexion and the foot at 15° of external rotation. In this position, a force directed posteriorly is applied to the proximal tibia, causing greater posterior translation of the lateral compartment compared with the undamaged limb [22].
- Reverse pivot-shift test: the knee is placed in 70° of flexion, and the foot is externally rotated. This leads to posterior subluxation of the lateral compartment of the PLC-injured knee. The knee is then slowly extended to about 20° of flexion, at which point the force vector of the iliotibial band changes and the tibia is pulled forward, reducing the subluxation [23].
- Posterolateral rotation or dial test: while the patient is placed in ventral decubitus position with the knees at 30° of flexion, both ankles are dorsiflexed and externally rotated simultaneously. The test is positive for an increase in external rotation of 10–15°. If the test is positive both at 30° and 90° of knee flexion, an associated PCL lesion is present [24, 25].

Table 44.1 Grading of PLC lesion [28, 29]

Fanelli scale for PLC injury (location based)	
A	Injury to PFL and popliteus tendon
B	Injury to PFL, popliteus tendon, and FCL
C	Injury to PFL, popliteus tendon, FCL, lateral capsule avulsion, and cruciate ligament disruption
Hughston scale for collateral ligament injury (instability based)	
1+	Varus opening 0–5 mm
2+	Varus opening 5–10 mm
3+	Varus opening >10 mm

Plain anteroposterior (AP) and lateral radiographs can identify Segond fractures and fibular head avulsion fractures. Comparative varus stress AP radiographs, both at 0 and 30° of knee flexion, can be helpful to quantify the lateral joint space opening in both acute and chronic lesions [26, 27]: an increase in lateral compartment gapping of 2.7 mm indicates an isolated FLC tears, more than 4.0 mm indicates the presence of a high-grade injury [28]. All patients with chronic posterolateral knee injuries should be assessed for their limb alignment. MRI should always be performed in order to evaluate not only the posterolateral structures but also cruciate ligaments, medial collateral ligament, articular cartilage, and meniscal injuries. MRI technique was effective for identifying injuries to the FCL, popliteus tendon, popliteofibular ligament, and biceps femoris. Sensibility of MRI in identifying lesion of the FCL is 57.5 % and only 24.2 % for the popliteus muscle tendon. MRI, thus, cannot be the determining factor for surgical indication for reconstruction [29].

Arthroscopic evaluation can be a powerful diagnostic tool: a drive-through sign occurs when there is more than 1 cm of lateral joint opening when a varus stress is applied to the knee in "Figure four" position [27]. Arthroscopy is particularly helpful in identifying the location of injuries at the femoral attachment of the popliteus tendon, coronary ligament of the posterior horn of the lateral meniscus, and the popliteomeniscal fascicles.

Grading of PLC injuries can be performed as shown in Table 44.1.

44.5 Treatment Strategies

Conservative management of PLC injuries is not well documented in literature. This kind of lesions is in most cases combined with injuries to one or both the cruciate ligaments, with absolute surgical indication. In our experience, in grade I-II isolated PLC lesions, conservative treatment is appropriate. Grade III lesions, isolated or associated to other ligamentous injuries, always need surgical treatment.

In the acute setting, isolated PLC injuries can be treated with direct repair or repair with augmentation. Acute injuries treated within 3 weeks from the trauma results in better outcomes than chronic injuries. Damaged structures can be directly sutured or anchored back to their bony attachments. All major structures of the posterolateral complex should be macroscopically evaluated in order to provide a complete restoration of the anatomy. The repair should be performed within the first 2–3 weeks after surgery in order to avoid retraction and arthrofibrosis.

Chronic injuries to the PLC are better managed with PLC reconstruction techniques rather than direct repair. Patients with chronic combined posterolateral knee injuries and varus alignment will first require an opening-wedge osteotomy as part of a staged procedure. In varus knee, there is a high risk of failure secondary to the stretching out of the grafts. An axial correction with a proximal opening-wedge osteotomy causes a significant reduction of posterolateral laxity. Second stage PLC reconstruction is not necessary in approximately 40 % of patients who had a previous open-wedge HTO [30, 31].

Several surgical techniques for the treatment of posterolateral knee instability have been reported, but there is still no consensus on the best technique to use. These techniques can be classified into nonanatomical fibular-based reconstructions and anatomical tibial and fibular-based reconstructions. The most widely used nonanatomical reconstruction procedure was described by Larsen et al. [32]. They found the fibular head to be isometric to the lateral femoral epicondyle, so they recommended the use of a graft passed through the fibular head and inserting into the lateral epicondyle. Larsen's PLC reconstruction aims to restore the functions of the popliteofibular ligament and the lateral collateral ligament; it is nonanatomical because the femoral insertion lies on the lateral epicondyle and not at the anatomical insertion sites of these structures.

More recently, there has been an emphasis on restoring native knee anatomy with more anatomical reconstructions. Noyes and Barber-Westin [33] described a technique that uses two femoral tunnels to restore the anatomy of the PLC on the distal femur with a transfibular tunnel, and Arciero [34] described a similar fixation of the femur with an oblique fibular tunnel.

LaPrade et al. [35] proposed an anatomical tibial and fibular-based reconstruction technique that attaches ligaments to both the fibula and tibia to better restore the anatomy of the popliteus tendon.

The authors believe LaPrade procedure to be very accurate in restoring anatomy and biomechanics of the posterolateral complex. Given the high number of tunnel to be reamed, the authors prefer to perform LaPrade reconstruction in isolated PLC lesions because in multiligamentous reconstructions, it could be challenging to avoid femoral tunnel collision. In combined reconstructions, the authors' preferred technique is a modified Larsen procedure with a more anatomical placement of the fibular tunnel which is not anteroposterior as originally described but directed from anterior-lateral-inferior to posterior-medial-superior.

44.5.1 Surgical Technique

Both procedures are performed with a lateral vertical skin incision starting from just proximal to the lateral epicondyle followed distally to the fibular head. Further incision is performed down to the deep layer in line with the fibers of the iliotibial band centered on the lateral femoral epicondyle. For the exposure of the fibular attachments of the LCL and PFL, an incision is made just anterior to the long head of the biceps tendon so that the posterior aspect of the fibular head and the biceps bursa can be identified. Identification and isolation of the peroneal nerve is always performed to prevent iatrogenic lesions (Fig. 44.1).

44.5.1.1 The Modified Larson Technique

Fibular tunnel is drilled after identifying fibular insertion of FCL and PFL; FCL attachment site is on the anterolateral aspect of the fibular head; the region of attachment of the PFL should be identified on the posteromedial downslope on the fibular styloid. A spoon is placed posteriorly to the fibular head to prevent guide pin protrusion, and the guide pin is then drilled. The tunnel is overreamed with a 6 mm reamer (Fig. 44.2). The authors' preferred graft for this reconstruction is the tendon of the semitendinosus muscle, both autologous and allogenic.

The tendon graft is passed through the tunnel so that the middle part of the graft lies free in the fibular tunnel. The posterior end of the graft is passed through the popliteal hiatus, the anterior below the ITB. Both the anterior and the posterior limbs of the graft are held together, and the isometric point is located on the femur while the knee is flexed and extended from 0 to 90°. The entry point is usually slightly distal/anterior to the attachment of the collateral ligament. The femoral tunnel is drilled from the isometric point, and the anterior and posterior limbs of the graft are passed through the tunnel and tensioned together at 10° of flexion (Fig. 44.3).

44.5.1.2 The LaPrade Technique [35]

Fibular tunnel is drilled as described for the previous technique. Tibial tunnel is reamed from a flat spot just distal and medial to the Gerdy's tubercle to the musculotendinous junction of the popliteus muscle. Femoral tunnels are drilled after the identification of the femoral attachment sites of the popliteus tendon and the FCL. FCL attachment point is approximately 3.2 mm proximal and posterior to the lateral epicondyle. The popliteus tendon femoral attachment site is

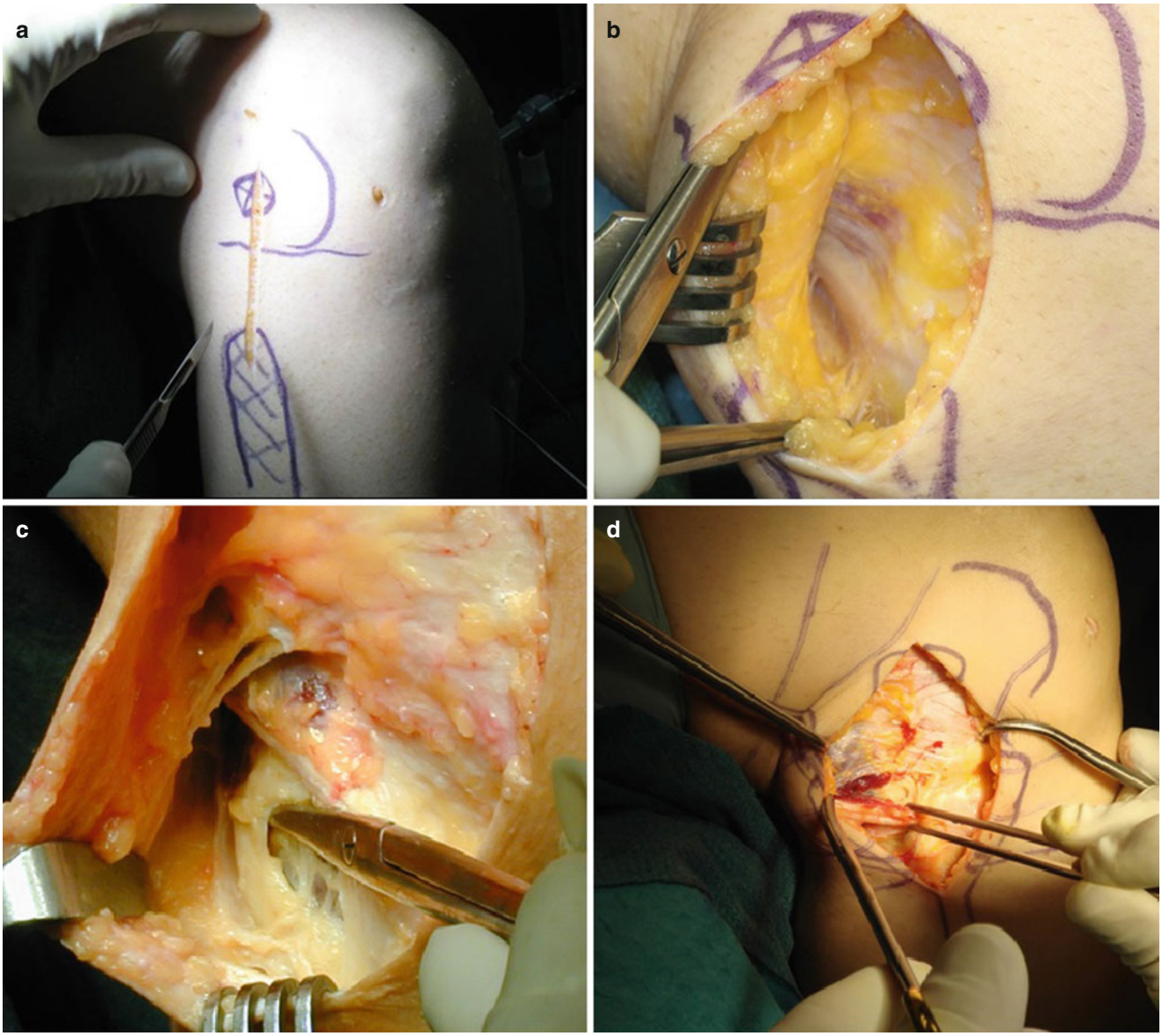


Fig. 44.1 Surgical approach. (**a**) Skin incision. (**b**) Deep fascia. (**c**) Identification of the peroneal nerve. (**d**) Biceps bursa incision

identified on the top fifth of the anterior aspect of the popliteal sulcus. Once the two grafts are prepared, they are inserted into each femoral tunnel and fixed with interference screws. The popliteus tendon graft is then passed through the popliteal hiatus then beyond the posterolateral aspect of the knee into the tibial tunnel. The FCL graft is passed distally under the superficial layer of the ITB and the lateral aponeurosis of the long head of the biceps and passed through the fibular head tunnel where it is fixed with a bioadsorbable screw while the knee is kept at 20° of flexion. The remaining end of the graft is passed from posterior to anterior into the tibial tunnel together with the popliteus tendon graft. Fixation is performed with the knee flexed at 60° in neutral rotation (Fig. 44.4).

In this kind of anatomical reconstruction, it is very important to complete the PLC reconstruction before the central pivot fixation. In PCL-PLC combined reconstruction, the fixation of the PCL before the PLC fixation would cause joint overconstraining and overload on the PCL graft [2].

If a combined ACL-PLC reconstruction is performed, the fixation of ACL should be performed after PLC fixation in order to avoid that the ACL tension could cause an abnormal external rotation of the tibia caused by the lack of posterolateral structures [36].

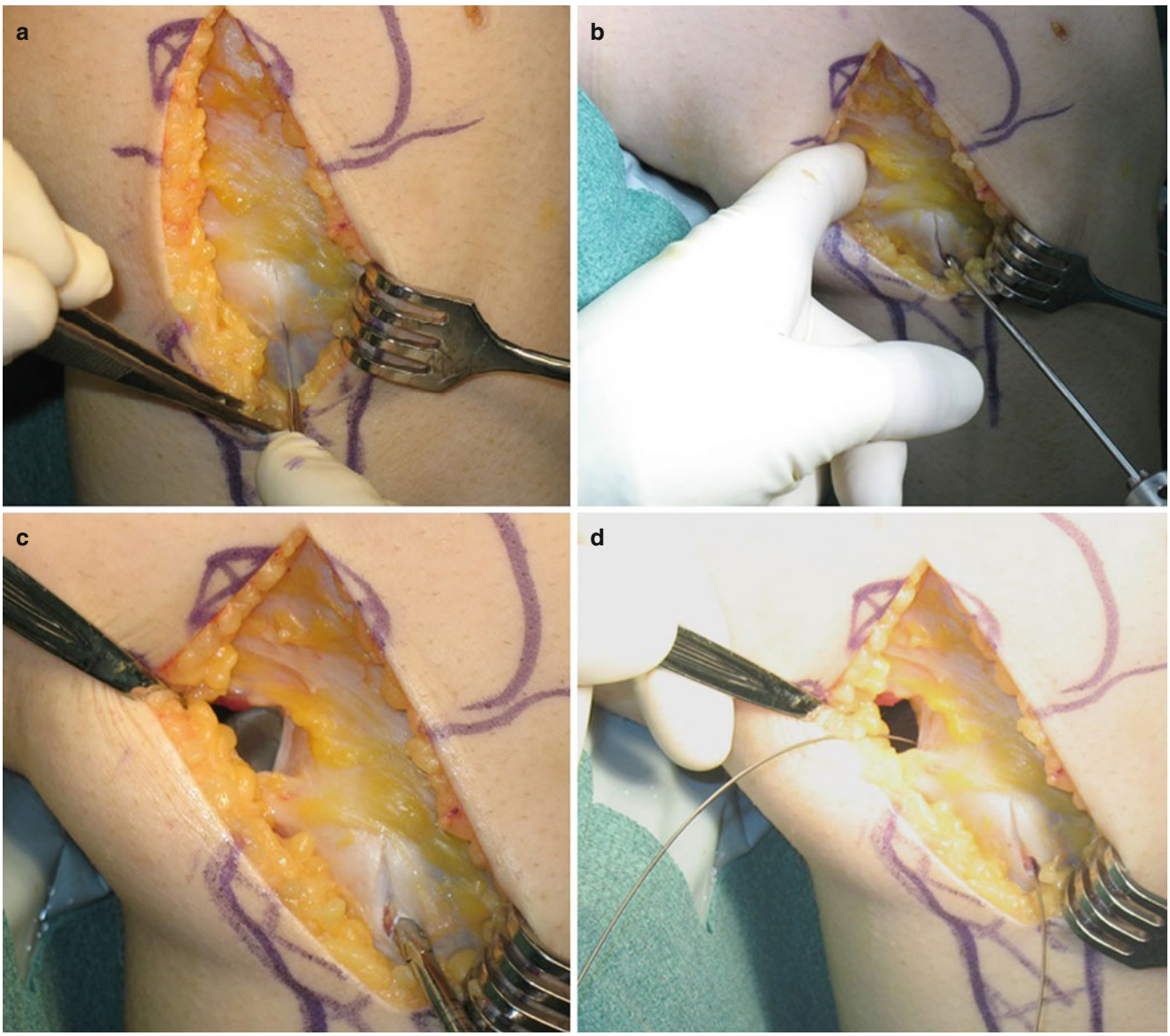


Fig. 44.2 Fibular tunnel preparation. (**a**) Exposition of the fibular head. (**b**) Guide pin insertion after identification of FCL and PFL insertion points. (**c**) Reaming. A spoon is placed beyond to prevent lesions to the peroneal nerve. (**d**) Nitinol guidewire

44.6 Rehabilitation and Return to Sports Activity

Postoperative rehabilitation is a critical part of the treatment for a PLC injury. The patient is placed in a knee brace locked in extension for 2 weeks; early quadriceps activation can be stimulated with isometric exercises, electrical stimulation, and taping. The range of motion is passively increased in the following 4 weeks, aiming to obtain full ROM within the sixth week. Weight bearing is allowed as tolerated from the fourth week; full weight bearing should be gained within the sixth week. Strength, core stability, proprioception, and endurance are developed between the second and the fourth postoperative month. For the first 4 months postoperatively, closed-kinetic chain exercises are limited to 70° or less knee flexion; tibial external rotation is avoided such as resistive and repetitive hamstring exercises with the knee in flexion. Light jogging should start after the fourth month postoperatively if the patient demonstrates a normal gait pattern, adequate lower extremity knee range, and absence of joint effusion with prolonged walking. Return to sports is usually after 6 months, if functional and clinical tests show a satisfactory graft healing.

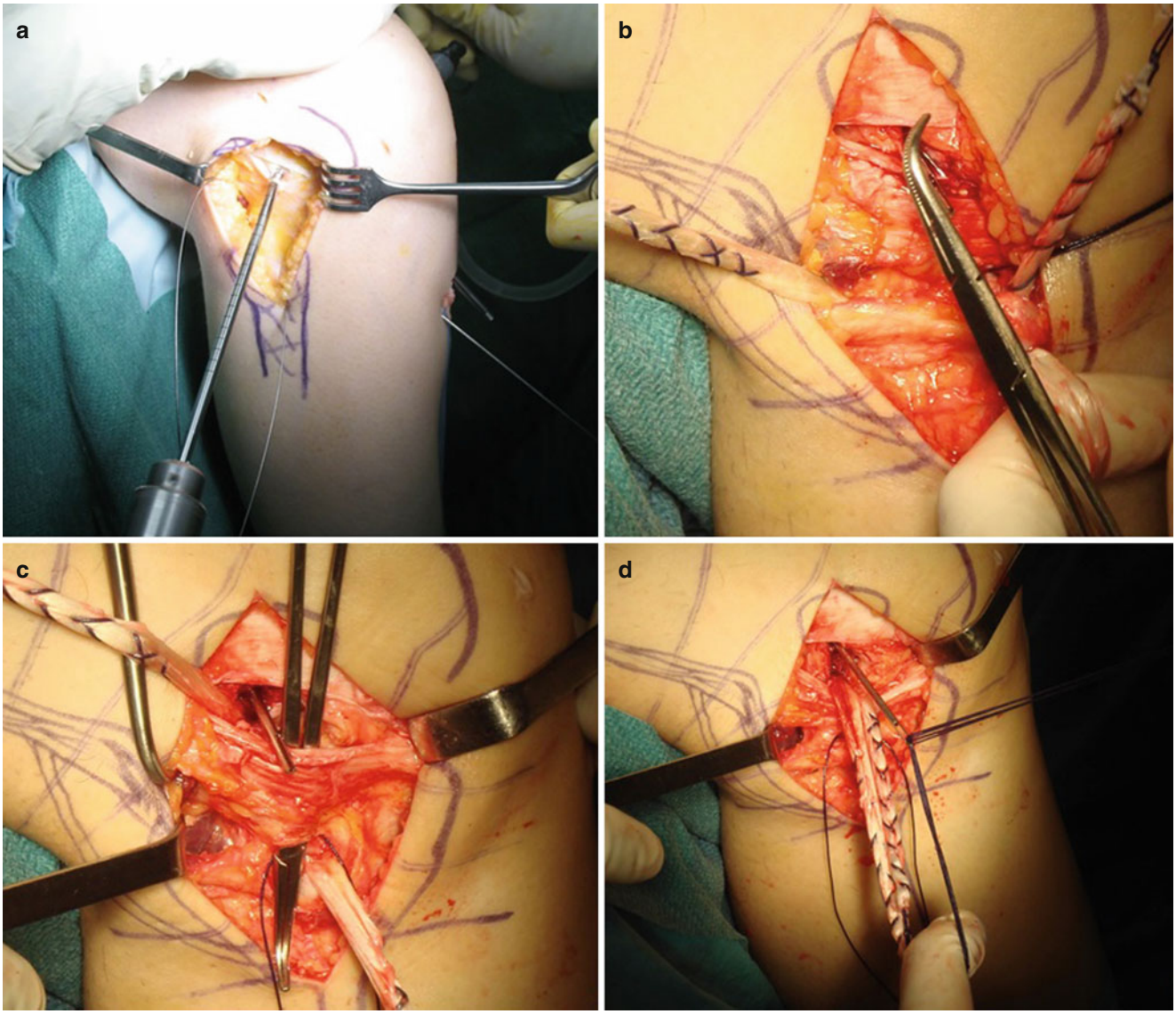


Fig. 44.3 Modified Larsen technique. (**a**) Femoral tunnel reaming after identification of the isometric point. (**b**) Graft passed through the fibular tunnel. (**c**) Posterior graft end passed through the popliteal hiatus. (**d**) Anterior graft end passed below ITB. Both ends of the graft are then inserted in the femoral tunnel

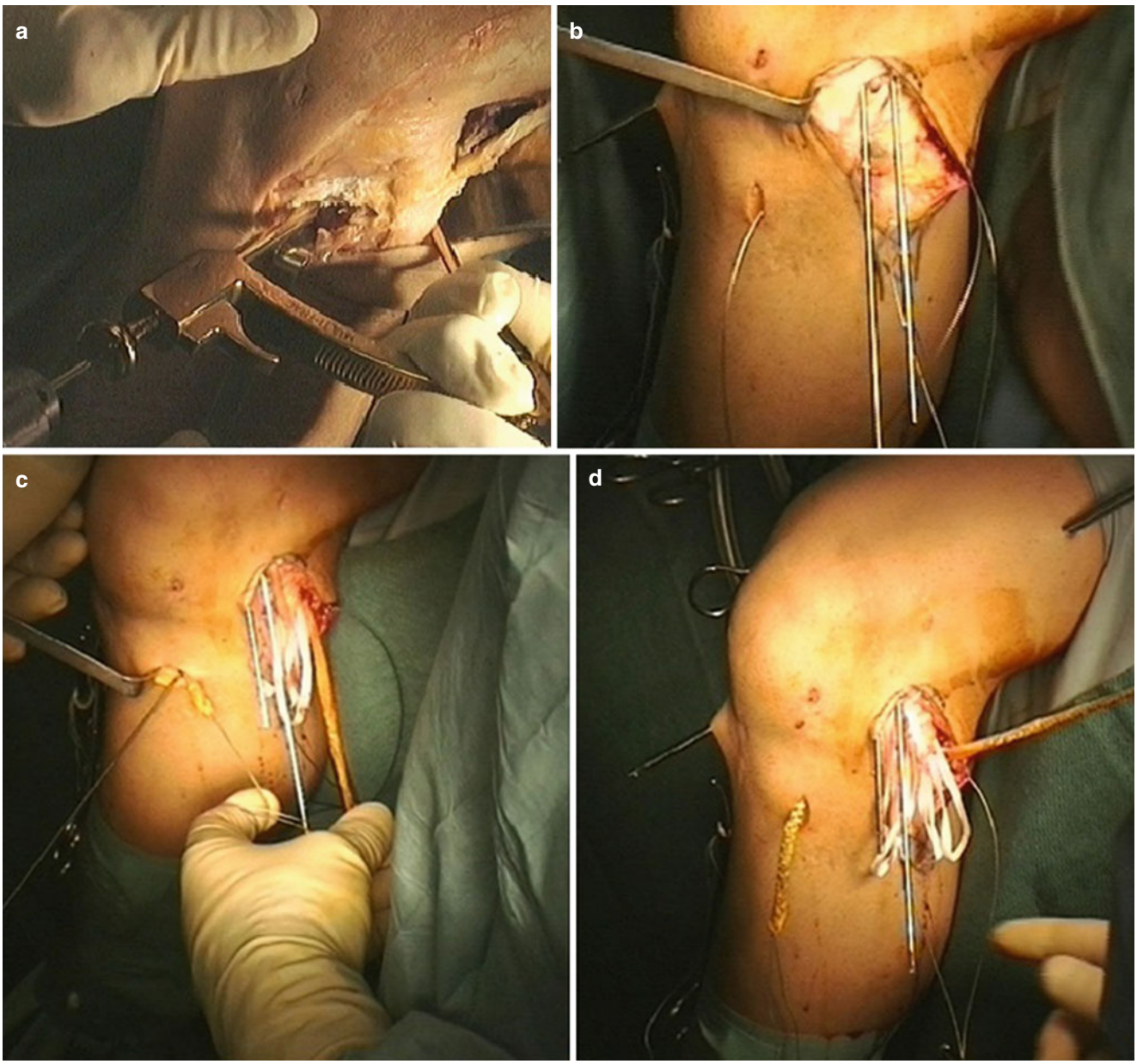


Fig. 44.4 LaPrade technique. (**a**) AP tibial tunnel drilling. (**b**) Femoral tunnel guide pins placement. (**c**, **d**) Grafts insertion and fixation

References

1. Harner CD, Vogrin TM, Hoher J, Ma CB, Woo SL (2000) Biomechanical analysis of a posterior cruciate ligament reconstruction. Deficiency of the posterolateral structures as a cause of graft failure. Am J Sports Med 28:32–39
2. LaPrade RF, Muench C, Wentorf F, Lewis JL (2002) The effect of injury to the posterolateral structures of the knee on force in a posterior cruciate ligament graft: a biomechanical study. Am J Sports Med 30:233–238
3. DeLee JC, Riley MB, Rockwood CA Jr (1983) Acute posterolateral rotatory instability of the knee. Am J Sports Med 11:199–207
4. LaPrade RF, Wentorf F (2002) Diagnosis and treatment of posterolateral knee injuries. Clin Orthop Relat Res 402:110–121
5. Hughston JC, Jacobson KE (1985) Chronic posterolateral rotatory instability of the knee. J Bone Joint Surg Am 67:351–359
6. Krukhaug Y, Molster A, Rodt A, Strand T (1998) Lateral ligament injuries of the knee. Knee Surg Sports Traumatol Arthrosc 6:21–25
7. Fanelli GC, Edson CJ (1995) Posterior cruciate ligament injuries in trauma patients: part II. Arthroscopy 11:526–529
8. Baker CL Jr, Norwood LA, Hughston JC (1983) Acute posterolateral rotatory instability of the knee. J Bone Joint Surg Am 65: 614–618
9. LaPrade RF, Wentorf FA, Hollis Fritts MS, Gundry C, Hightower CD (2007) A prospective magnetic resonance imaging study of the incidence of posterolateral and multiple ligament injuries in acute knee injuries presenting with a hemarthrosis. Arthroscopy 23(12): 1341–1347

10. Lee YS, Jung YB (2013) Posterior cruciate ligament: focus on conflicting issues. Clin Orthop Surg 5:256–262
11. Noyes FR, Barber-Westin SD (1996) Surgical restoration to treat chronic deficiency of the posterolateral complex and cruciate ligaments of the knee joint. Am J Sports Med 24:415–426
12. Lee SH, Jung YB, Lee HJ, Jung HJ, Kim SH (2012) Revision posterior cruciate ligament reconstruction using a modified tibial inlay double-bundle technique. J Bone Joint Surg Am 94:516–522
13. Geiger D, Chang E, Pathria M et al (2013) Posterolateral and posteromedial corner injuries of the knee. Radiol Clin North Am 51(3):413–432, 328–329
14. Seebacher JR, Inglis AE, Marshall JL et al (1982) The structure of the posterolateral aspect of the knee. J Bone Joint Surg Am 64:536–541
15. Davies H, Unwin A, Aichroth P (2004) The posterolateral corner of the knee. Anatomy, biomechanics and management of injuries. Injury 35(1):68–75, 317
16. Morelli V, Bright C, Fields A (2013) Ligamentous injuries of the knee. Prim Care 40(2):335–356, 356
17. DeLee JC, Drez D Jr, Miller MD (2009) DeLee & Drez's orthopaedic sports medicine, 3rd edn. Saunders Elsevier, Philadelphia
18. Green NE, Swiontkowski MF (2008) Skeletal trauma in children, vol 3, 4th edn. Saunders Elsevier, Philadelphia
19. Beall DP, Googer JD, Moss JT et al (2007) Magnetic resonance imaging of the collateral ligaments and the anatomic quadrants of the knee. Radiol Clin North Am 45(6):983–1002
20. Pacheco RJ, Ayre CA, Bollen SR (2011) Posterolateral corner injuries of the knee: a serious injury commonly missed. J Bone Joint Surg Br 93:194–197
21. Veltri DM, Warren RF (1994) Anatomy, biomechanics, and physical findings in posterolateral knee instability. Clin Sports Med 13:599–614
22. Hughston JC, Norwood LA Jr (1980) The posterolateral drawer test and external rotational recurvatum test for posterolateral rotatory instability of the knee. Clin Orthop Relat Res (147):82–87
23. Jakob RP, Hassler H, Staeubli HU (1981) Observations on rotatory instability of the lateral compartment of the knee. Experimental studies on the functional anatomy and the pathomechanism of the true and the reversed pivot shift sign. Acta Orthop Scand Suppl 191:1–32
24. Bahk MS, Cosgarea AJ (2006) Physical examination and imaging of the lateral collateral ligament and posterolateral corner of the knee. Sports Med Arthrosc Rev 14:12–19
25. Wascher DC, Veitch AJ (2004). Posterior cruciate ligament and posterolateral corner injuries. In: Andrew Cosgarea (Ed.), Hospital physician board review manual. Orthopaedic sports medicine board review manual, Vol. 1, Part 1; Turner-White Communications, Inc., Wayne, Pennsylvania, pp. 2–12
26. LaPrade RF, Terry GC (1997) Injuries to the posterolateral aspect of the knee: association of injuries with clinical instability. Am J Sports Med 25:433–438
27. LaPrade RF, Bollom TS, Gilbert TJ, Wentorf FA, Chaljub G (2000) The MRI appearance of individual structures of the posterolateral knee: a prospective study of normal and surgically verified grade 3 injuries. Am J Sports Med 28:191–199
28. LaPrade RF, Heikes C, Bakker AJ et al (2008) The reproducibility and repeatability of varus stress radiographs in the assessment of isolated fibular collateral ligament and grade-III posterolateral knee injuries. An in vitro biomechanical study. J Bone Joint Surg Am 90:2069–2076
29. Bonadio MD, Helito CP, Gury LA, Demange MK, Pécora JR, Angelini FJ (2014) Correlation between magnetic resonance imaging and physical exam in assessment of injuries to posterolateral corner of the knee. Acta Orthop Bras 22(3):124–126
30. LaPrade RF, Engebretsen L, Johansen S, Wentorf F, Kurtenbach C (2008) The effect of a proximal tibial medial opening wedge osteotomy on posterolateral knee instability. A biomechanical study. Am J Sports Med 36:956–960
31. Arthur A, LaPrade RF, Agel J (2007) Proximal tibial opening wedge osteotomy as the initial treatment for chronic posterolateral corner deficiency in the varus knee: a prospective clinical study. Am J Sports Med 35:1844–1850
32. Larsen RV, Sidles JA, Beals CT (1996) Isometry of lateral collateral and popliteofibular ligaments and a technique for reconstruction. University of Washington, Seattle, p 4244 (research report)
33. Noyes FR, Barber-Westin SD (1995) Surgical reconstruction of severe posterolateral corner injuries of the knee using allograft tissues. Am J Sports Med 23:2–12
34. Arciero RA (2005) Anatomic posterolateral corner knee reconstruction. Arthroscopy 21:1147
35. LaPrade RF, Johansen S, Wentorf FA, Engebretsen L, Esterberg JL, Tso A (2004) An analysis of an anatomical posterolateral knee reconstruction: An in vitro biomechanical study and development of a surgical technique. Am J Sports Med 32:1405–1414
36. Wentorf FA, LaPrade RF, Lewis JL, Resig S (2002) The influence of the integrity of posterolateral structures on tibiofemoral orientation when an anterior cruciate ligament graft is tensioned. Am J Sports Med 30:796–799, 38

Patellar Instability

45

Giancarlo C. Coari, Filippo Troiani, Alessandro Tripodo, and Francesco Miele

Patellofemoral disorder represented by 20–40 % of all knee problems can be one of the sports-related common injuries [1]. It can be cause of disability, especially in the female population, and in some cases, it can lead to degenerative arthritic changes in the knee joint. The patellofemoral joint is one of the most complex articulations of our body. The causes are a combination of anatomical bone component, capsule-ligament structures, and muscle forces, and that is why no single pathophysiology or therapeutic approach can solve patellofemoral instability.

45.1 Etiology

Disorders of the patellofemoral joint are numerous and are of great importance because they seriously limit patients' function. Treatment of these conditions is highly dependent on accurate diagnosis and determination of the correct cause. Patellar instability has been shown to be associated with four major bone abnormalities: (a) trochlear dysplasia, (b) pathologic TT-TG value, (c) patellar tilt, and (d) patella alta. Each of those factors is defined by measures in the lateral or axial view x-rays and with CT scan [2]. Soft tissue abnormalities such as hypoplasia of the vastus medialis and patellofemoral and patellotibial ligament disorders are direct part of the problem. Soft tissue structures that provide stability to the patellofemoral joint can be categorized as static and dynamic restraints. Static restraints: retinacula, capsule, and ligament. The medial patellofemoral ligament (MPFL) is the primary passive restraint to lateral displacement [3, 4]. Dynamic restrains: vastus medialis obliquus. Despite the enormous volume of literature on patellofemoral instability, more attention has been given to the soft tissue structures that are injured during patellar dislocation and the contribution these injured structures make in controlling patellar motion in the intact knee. The MPFL restraint to lateral translation only in early knee flexion, as the knee progress in flexion, trochlear geometry, patellofemoral congruence, and in particular the slope angle of the lateral trochlear wall, provides the major restraints to lateral patellar displacement. The MPFL has been recently more recognized as playing an important role in patellar biomechanics and its implication after dislocation [3–5].

Finally we have to consider other factors affecting the rotula outside the knee; excessive femoral anteversion with internal rotation of the femoral condyles or excessive external tibial rotation can cause higher stress forces on the rotula and instability [6].

D. Dejour defined three major groups of patellofemoral disorder: patellar dislocation, pain, and pain plus anatomical disorder. The first group includes different categories: subjective instability with anatomical abnormalities, traumatic, dislocation with anatomical abnormalities, and patellofemoral pain [7]. The rate of subsequent dislocation after the first episode varies from 15 to 44 % following conservative management; this rate is increased in those who have more than one episode. In a natural history study, Fithian and associates showed that only 17 % of first-time dislocators suffered a second dislocation within the next 2–5 years. In contrast, patients who presented with recurrent patellar instability were much more likely to have subsequent dislocation than those who had only one dislocation episode. The risk of an addiction dislocation within 2–5 years was around 50 % among patients with a history of prior patellar instability [8].

45.2 Injury Mechanism

The average annual incidence of a primary patellar dislocation is 5.8 per 100,000. This incidence increases to 29 per 100,000 in the 10–17-year age group. The most common mechanism of first time dislocation are sports (61 %) injuries

G.C. Coari (✉) • F. Troiani • A. Tripodo • F. Miele
Casa di Cura San Camillo,
Via padre ignazio da carrara 37,
Forte dei Marmi (Lucca) 55042, Italy
e-mail: gccoari@cdh.it; ftroiani76@yahoo.it

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_45,

[8]. Nietosvaara reported 55 % during sport activity [9]. The direction of the instability or subluxation is more frequently lateral. Direct patellar trauma occurs more often with the knee in greater degrees of flexion. Patellar dislocation is a dramatic, memorable event, and the patient will frequently describe having to extend the knee for the patella to reduce spontaneously or require manipulation. The clinical manifestation of a primary dislocation of the patella varies from a massive swollen knee to a nearly painless knee. More commonly, the patient will describe an indirect mechanism consisting of a strong eccentric quadriceps contraction and a flexed and valgus knee position. It may occur from a rapid, noncontact deceleration or change of direction maneuver.

The typical "morphotype" of the patellar dislocator has been characterized as an adolescent female with ligamentous laxity and multiple developmental anomalies including patella alta, trochlear dysplasia, and rotational angular bony malalignment. Trochlear dysplasia and patella alta, which reduce the "containment" of the patella within the femoral trochlea at any given flexion angle compared to the normal knee, contribute directly to the risk of recurrent patellar dislocation by reducing the relative height of the lateral trochlear wall.

45.3 Clinical and Diagnostic Examination

45.3.1 Clinical Examination

Typically, patients with patellar disorder have a painful anterior knee pain suited behind the patella, often even on the medial side. Pain may be in the soft tissue or in the bone. Swelling and hemarthrosis can be appreciated after acute patellar dislocation. Standing examination with the patient barefoot in front of the examiner. We can appreciate the varus/valgus alignment of the knee, the orientation of the patella, and the morphology of the forefeet. With the patient in supine position, all the anatomic structures of the knee are palped to find out local pain or patellar tendon insertion and medial and lateral retinaculum. Crepitus during flexion-extension movement can be appreciated. The Q angle has a normal value of 10–15° in men and 15–20° for women. Increased Q angle may increase the laterally directed force on the extensor mechanism (quadriceps vector) predisposing the patella to malpositioning and instability. The tubercle sulcus angle is an alternative physical exam for estimating excessive quadriceps vector with the knee flexed 90°. The normal value is 0°, and values greater than 10° are considered pathologic. In the apprehension test the patient should extend the knee, while the examiner pushes the patella laterally. The test is positive when the patient resists because he recognizes instability symptoms. Patellar tilt limited upward movement would indicate excessively tight lateral retinaculum. Excessive patellar glide suggests reduced restraint from the medial structures and tightness of the lateral retinaculum.

45.3.2 Diagnostic Examination

Standard x-rays are always required. In the sagittal view trochlea dysplasia can be observed. Usually the sulcus line follows the Blumenstat line. In a normal knee this line stays posteriorly to the condylar line, meaning that the trochlea is deep and congruent. Henrì Dejour described it like the "crossing sign" which characterized the trochlear dysplasia on the sagittal view. In case of a trochlear dysplasia, there will be a crossing sign between the sulcus line and lateral condyle meaning that the trochlea is flat. The crossing sign is found in the 96 % of the population with history of true dislocation and only 3 % of the healthy control population [10]. In 1996 D. Dejour and Le Coultre redefined the classification adding a fourth type. Other than the crossing sign, two new radiographic signs were introduced. The "supratrochlear spur" and the "double contour." The fourth type is the combination of the different signs. Type A is the most common [11] (Fig. 45.1).

The patellar height can be measured with the Insall-Salvati index or Caton-Deschamps index. The first is the ratio of the length of the patellar tendon to the longest sagittal diameter of the patella [12]. The second is the ratio of the distance from the lower edge of the articular surface of the patella to the anterosuperior angle of the tibia outline [13]. Two basic types of axial views (Merchant and Laurin) can be done with different types of measurement. The Merchant can measure the sulcus angle and the congruence angle [14]. The Laurin can measure the lateral patellofemoral angle and the patellofemoral index [15]. There is a great variability in the patellar shape, and Wiberg classified them into three types. The most frequent type of patella in patellar dislocation is Wiberg type II [16].

Use of CT scan imaging for exploration of the patellofemoral relationship has led to a better understanding of the dynamics of this joint in normal and pathologic knees, and the use of it is widely accepted. It allows the study of many keen parameters. The tibial tubercle-trochlear groove distance (TT-TG) is the instrumental measurement of the "Q" angle. First described in 1978 by Goutallier as radiologic measurement, then in 1987 H. Dejour adapted this measurement to the CT scan. We use two cuts to measure the distance between the central point of the tibial tubercle (TT) and the deepest point of the trochlear groove (TG). Normal value is around 12 mm and in the patellar dislocation population is greater than 20 mm [10]. This pathologic value can be

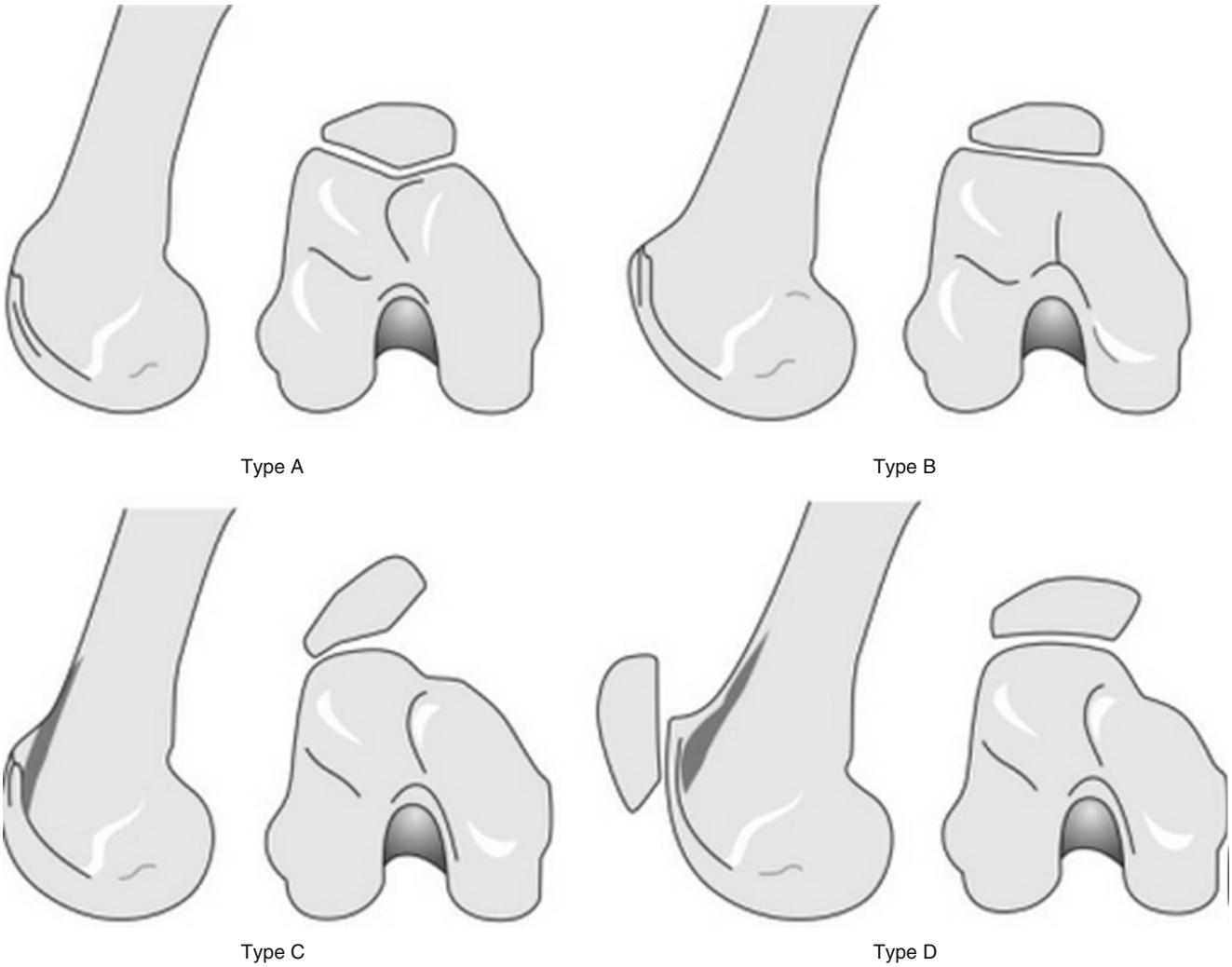


Fig. 45.1 Dejour's trochlear dysplasia classification. *Type A*, crossing sign, trochlear normal (>145°); *Type B*, crossing sign, supratrochlear spur, flat or convex trochlea; *Type C*. crossing sign, double contour; *Type D*, crossing sign, supratrochlear spur, double contour [11]

indicative for tibial tubercle medicalization. The patellar tilt is the angle between the transverse axis of the patella and the posterior femoral condyles. Eighty-three percent of the patellar dislocation population has a value greater than 20° compared to 3 % in the normal group [10]. We can obtain and even measure femoral anteversion and external tibial rotation. MRI has been considered more useful in defining cartilage status than measuring patellofemoral instability parameters. Some authors have compared, with the same reproducibility, TT-TG measurement with CT scan and MRI, but still need more study [17].

45.4 Treatment Strategy

Surgical treatment of patellar instability has two approaches. One is that anatomic alignment is the most important factor, and all "malalignment" factors must be correct. The other approach is softer because it recognized the importance of soft tissue restraint to lateral patellar translation: to stabilize or recreate, this restraint can be sufficient in many cases with bony defects uncorrected [18]. The first evaluation is about conservative versus surgical treatment. Surgical treatments contain soft tissue and bony procedures. Soft tissue procedures: lateral release, medial soft tissue reefing, proximal realignment, and MPFL reconstruction. Bone procedures are distal realignment, trochleoplasty, and distal femoral osteotomy. Substantial controversy exists about treatment strategy. From a logical standpoint, the procedures adopted should correct the observed root abnormalities, and it is more likely that a combination of procedures would correct those abnormalities one by one, rather than one standard procedure for every case. To remedy patellar instability, the surgeon will need to combine soft tissue and bony procedures to address all involved factors, each corrected individually.

For acute first-time dislocation, the classic treatment is conservative. The more important exception to this is the presence of an osteochondral fracture. Some authors propose acute repair in cases of substantial medial structure disruption and a laterally subluxated patella with a normally aligned opposite knee. The main goals of conservative treatment are swelling and pain remission, as well as restoration of range of motion. Quadriceps strengthening is another goal of the conservative management strategy, and good quadriceps strength seems to alleviate symptoms, but whether it prevents future dislocation is unclear. Immobilization for up to 6 weeks may help medial structure heeling, but stiffness is a problem. If recurrent dislocation occurs, they will put the patient in a different category from treatment purposes: the chronic dislocation group.

45.4.1 Lateral Release

We can find LR treatment isolated or associated procedures. Isolated LR has no role in treatment of acute or recurrent patellar instability because from a mechanical perspective, isolated LR cannot correct the causes of patellar instability. Isolated LR can be a successful procedure in patients with isolated lateral patellar tightness. Associated with medial reefing or to a proximal/distal realignment, we have a better result [19].

45.4.2 Medial Reefing

The indication for arthroscopic medial reefing after acute dislocation is the presence of persistent patellar dislocation or detachment of the medial retinaculum or MPFL from the patellar insertion (usually the lesion is on the femoral side) [3]. A relative contraindication can be trochlear dysplasia grade B or C according to H. Dejour or rupture of MPFL on the femoral insertion.

45.4.3 Medial Patellofemoral Ligament Reconstruction

The increased mobility after dislocation appears to be attributable to medial retinacular deficiency. On cadaver it has been shown that the MPFL is the most important structure resisting lateral patellar motion. According to literature MPFL injury may be present in approximately 65 % of cases [20]. Restoration of the MPFL has been described by primary repair alone, repair with augmentation, and reconstruction alone. MPFL repair has been approached in various ways. The repair may be done acutely or after a period of initial healing. Repair has been described at the patellar insertion of the MPFL. Our favorite technique for the reconstruction is with the semitendon fixed to the rotula with two soft anchors and half tunnel in femur with a screw. Previously we create a slot on the medial side of the rotula to allow integration, and we don't perform any tunnel in the rotula to avoid fractures, especially in small rotula, or cartilage damage. The key point is to choose the right point of insertion to restore proper kinematics. Non-anatomical femur insertion can lead to excessive medial pressure on the rotula cartilage [21]. We measured rotula and selected two-third proximal to implants the two soft anchors. Femur position is the most difficult step of the reconstruction. The MPFL originates from a ridge between the medial epicondyles and the adductor tubercle (Fig. 45.2).

Indications for MPFL reconstruction are laxity of the medial retinacula due to recurrent patellofemoral dislocation. MPFL is used alone if there are no bony defects to correct. Its goal is to restore the loss of the medial soft tissue patella stabilizer, which is injured or chronically lax due to recurrent lateral patellar dislocation (Table 45.1).

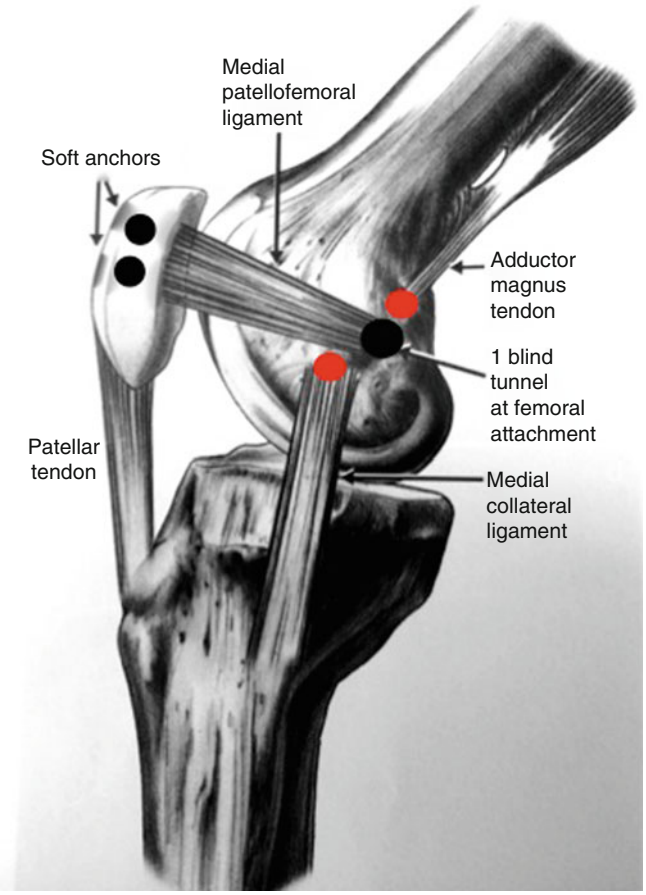
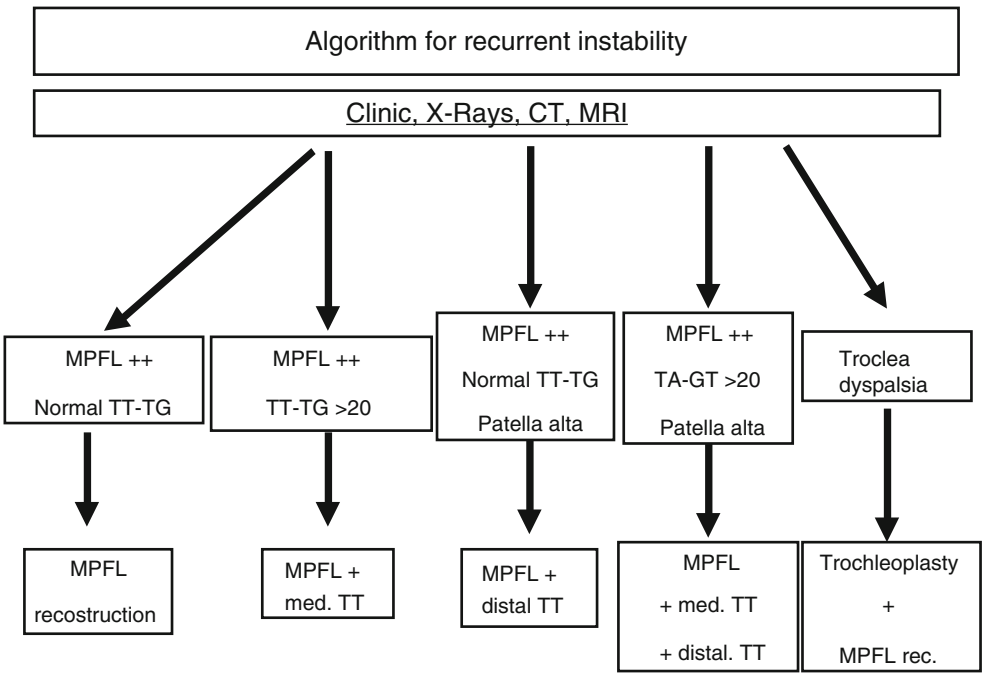


Fig. 45.2 Anteromedial view of the knee. Two-third of the medial patella side and the middle point in of the ridge between the adductor tubercle posteriorly and medial epicondyles anteriorly

Table 45.1 Algorithm for recurrent patellar instability



45.4.4 Distal Realignment

The goal of *medicalization of the tibial tuberosity (Elmslie-Trillat)* is to reduce TT-TG distance to value between 12 and 16 mm. It can be performed in association to lateral release and medial reefing. The indications change in last year, reducing to young patients with symptomatic patellar instability and TT-TG more than 20 mm measured on CT scan. CT scan is needed perioperatively and postoperatively (risk of over correction) [22].

45.4.5 Trochleoplasty

Trochleoplasty is indicated to correct severe trochlear dysplasia. It is a challenging surgery not widely performed. *Lateral facet-elevating trochleoplasty* is indicated in patients with flat or shallow trochlea, but without trochlear prominence. This procedure is certainly efficient in terms of stability but could lead to arthrosis changes. *Sulcus deepening trochleoplasty* is more anatomic, and it is indicated in severe dysplasia (type C or D) in which the trochlea is prominent and convex and the patella impinges on the trochlear bump during flexion knee. Frequently there are other abnormalities associated in this patient population so that this procedure is usually performed in combination to bony or soft tissue techniques [23].

45.5 Rehabilitation and Return to Play

Immobilization after surgery aims to allow better healing of tissue as well as preventing damage to the graft. Later release doesn't need immobilization. The other techniques required time of immobilization that is not defined for each procedure. If the surgical fixation is adequate, you can reduce the time for immobilization to prevent arthrofibrosis and stiffness. An individual plan for each patient considering the quality of surgery, tissue healing, and nature of the damage should be considered. In conservative treatment, a longer period of immobilization is recommended. Not much limitation in weight bearing. More attention for immobilization in tibial osteotomy. Most of the procedure changes stress contact area in the patella so that passive continuous mobilization (0–90°) can help to find a new balance between the structures and help cartilage nutrition. Restoration of quadriceps strength is one of the goals in rehabilitation. Open and closed kinetic chain quadriceps exercise should be granted with different timing in relation to the type of surgery. In the MPFL reconstruction, we prefer after 3 weeks. In distal osteotomy to avoid early stress on the screw, we prefer after 5 weeks and even in nonoperative treatment to help better tissue healing. Later release has no restriction.

References

1. Zaffagnini S et al (2010) Pathophysiology of lateral patellar dislocation. In: Zaffagnini S, Dejour D, Arendt EA (eds) Patellofemoral pain, instability, and arthritis. Springer, Berlin, pp 17–25
2. Dye SF (1996) The knee as a biologic transmission with an envelope of function- a theory. Clin Orthop 323:10–18
3. Conlan T, Garth WP, Lemons JE (1993) Evaluation of the medial soft-tissue restraints of the extensor mechanism of the knee. J Bone Joint Surg Am 75(5):682–693
4. Desio SM, Burks RT, Bachus KN (1998) Soft tissue restraints to lateral patellar translation in the human knee. Am J Sports Med 26(1):59–65
5. Hautamaa PV, Fithian DC, Pohlmeyer AM, Kaufman KR, Daniel DM (1998) The medial soft tissue restraints in lateral patellar instability and repair. Clin Orthop Relat Res 349:174–182
6. Eckhoff DG, Montgomery WK, Kilcoyne RF, Stamm ER (1994) Femoral morphometry and anterior knee pain. Clin Orthop Relat Res 302:64–68
7. Dejour D, Nove-Josserand L, Walch G (1998) Patellofemoral disorders-classification and an approach to operative treatment for instability. In: Controversies in orthopedic sports medicine. Williams & Wilkins, Hong Kong, pp 235–244
8. Fithian DC et al (2004) Epidemiology and natural history of acute patellar dislocation. Am J Sports Med 32(5):1114–1121
9. Nietosvaara Y, Al K, Kallio PE (1994) Acute patellar dislocation in children: incidence and associated osteochondral fractures. J Pediatr Orthop 14:513–515
10. Dejour H, Walch G, Neyret P et al (1990) Dysplasia of the femoral trochlea. Rev Chir Orthop Reparatrice Appar Mot 76:45–54
11. Dejour D, Reynaud P, Lecoulture B (1998) Douleurs et instabilitè rotulienne. Essai de Classification. Médecine et Hygiène 5:1466–1471
12. Insall J, Salvati E (1971) Patella position in the normal knee joint. Radiology 101:101–104
13. Canton J, Deschamps G, Chambat P, Lerat JL, Dejour H (1982) Les rotules basses: a propos de 128 observations. Rev Chir Orthop 68:317–325
14. Merchant AC (2001) Patellofemoral imaging. Clin Orthop Relat Res 389:15–21
15. Laurin CA, Dussault R, Levesque HP (1979) The tangential x-ray investigation of the patellofemoral joint: x-rays technique, diagnostic criteria and their interpretation. Clin Orthop Relat Res 1444:16–26
16. Wiberg G (1941) Roentgenographic and anatomic studies on the patellofemoral joint. Acta Orthop Scand 12:319–410
17. Schottle PB, Zanetti M, Seifert B, Pfirmann CW, Fucentese SF (2006) The tibial tuberosità trochlear groove distance. A comparative study between CT and MRI imaging. Knee 13:26–31
18. Ardent AE (2010) MPFL reconstruction: the adductor sling approach. In: Zaffagnini S, Dejour D, Arendt EA (eds) Patellofemoral pain, instability, and arthritis. Springer, Berlin, pp 175–179
19. Lattermann C, Toth J, Bach BR Jr (2007) The role of lateral retinacular release in the treatment of patellar instability. Sports Med Arthrosc 15(2):57–60
20. Tuxoe JI, Teir M, Winge S et al (2001) The medial patellofemoral ligament: a dissection study. Knee 10:138–140
21. Elias JJ, Cosgarea AJ (2006) Technical errors during medial patellofemoral ligament reconstruction could overload medial patellofemoral cartilage: a computational analysis. Am J Sports Med 34(9):1478–1485
22. Trillat A, Dejour H, Couette A (1964) Diagnostic et traitment des subluxations recidivantes de la rotule. Rev Chir Orthop 50:813–824
23. Dejour D, Byn P, Saggin PR (2010) Deepening trochleoplasty for patellar instability. In: Zaffagnini S, Dejour D, Arendt EA (eds) Patellofemoral pain, instability, and arthritis. Springer, Berlin, pp 225–232

Anterior Knee Pain

46

Alfredo Schiavone-Panni, Carlo Perisano, Chiara Del Regno, Katia Corona, Antonio D'Amelio, and Michele Vasso

Anterior knee pain (AKP) or *patellofemoral pain syndrome* (PFPS) is a prevalent musculoskeletal condition characterised by a mechanical pain perceived in the anterior region of the knee exacerbated by activity and relieved by rest. AKP often has a poor prognosis and can significantly impact daily activities as well as participation in physical activity [1–3].

The AKP is one of the most pathologic knee disorders defined by Stanley James as the "black hole of orthopaedics" [4]. AKP can refer to a number of symptoms. In the past the terms "chondromalacia patellae" and "patellofemoral pain" have been used loosely to describe such symptoms [5–7]. This is to be avoided, as while both conditions are recognised, they may not be the cause of the pain [5–7]. The term "anterior knee pain" may be used when no specific diagnosis has been made. Management of anterior knee pain involves consideration of each individual's presentation and the potential contribution of local, proximal and distal knee factors [5–7].

The incidence of "anterior knee pain" is high and is located at 22/1,000 persons per year. AKP has a high prevalence in the population (range 15–45 %) that is higher in childhood and adolescence (≅19 % with a range of 3–40 %) and in women (M/F = 2/1) and represents the 25 % of all knee pathologies and the 10 % of all orthopaedic examination [8, 9].

A. Schiavone-Panni • C. Del Regno, MD • K. Corona, MD
A. D'Amelio, MD • M. Vasso, MD
Orthopaedic Department, Molise University,
Science of Health, Campobasso 86100, Italy
e-mail: a.schiavone@iol.it; chiaradelregno@gmail.com; vassomichele@gmail.com

C. Perisano (✉)
Department of Geriatrics, Neurosciences
and Orthopaedic, Catholic University of Sacred Heart,
L.A. Gemelli 1, Rome 00168, Italy
e-mail: carloperisano@hotmail.it

46.1 Aetiology

The aetiopathogenesis for anterior knee pain is multifactorial and mainly affects people without any structural changes or significant pathological changes in articular cartilage [10]. The pathologies that cause AKP are multiple and change with age (Fig. 46.1) [11]. The two most common are the patellofemoral syndrome and the patellar tendinopathy [3, 10, 11]. A possible trigger for the patellofemoral pain syndrome may be overload of the patellofemoral joint (e.g. high-intensity training) due to multiple factors that may cause anterior knee pain bringing to a vicious cycle of pain [9]. Several risk factors have been proposed as possible parameters involved in the pathogenesis of PFPS:

- *Extrinsic risk factors*: related to factors outside human body, such as the type of sports activity (runners, jumpers, skiers, bicyclists and soccer players), the environmental conditions and the surface and equipment used [3, 11–14].
- *Intrinsic risk factors*: relate more to individual characteristics. Some of these intrinsic risk factors are modifiable (overheight, tightness or weakness of the lower limb muscles, patellar malalignment and hypermobility, postural problem), while others are not (lower limb dislocation fracture, joint laxity, young adult, women) [3, 11–14].

The pathogenesis of knee anterior pain is multiple:

- *Patella tracking*: although its role has long been controversial, recent studies show the key role. The study of Draper and Wilson demonstrated an increased lateralisation (maltracking), lateral tilt and spin of the patella in patients with a *PFPS* with consequently high stresses between the lateral facet of the patella and the lateral trochlea [15, 16].
- *Role of quadriceps*: an imbalance in the activation of the M. vastus medialis obliquus and M. vastus lateralis, with a delayed activation of the first and an earlier activation of the second, was shown in patients with PFPS. A quadriceps tightness may cause high patellofemoral stresses and quadriceps weakness, specifically VMO weakness (often atrophic) in comparison to the VL, can lead to lateral

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_46, © Springer International Publishing Switzerland 2016

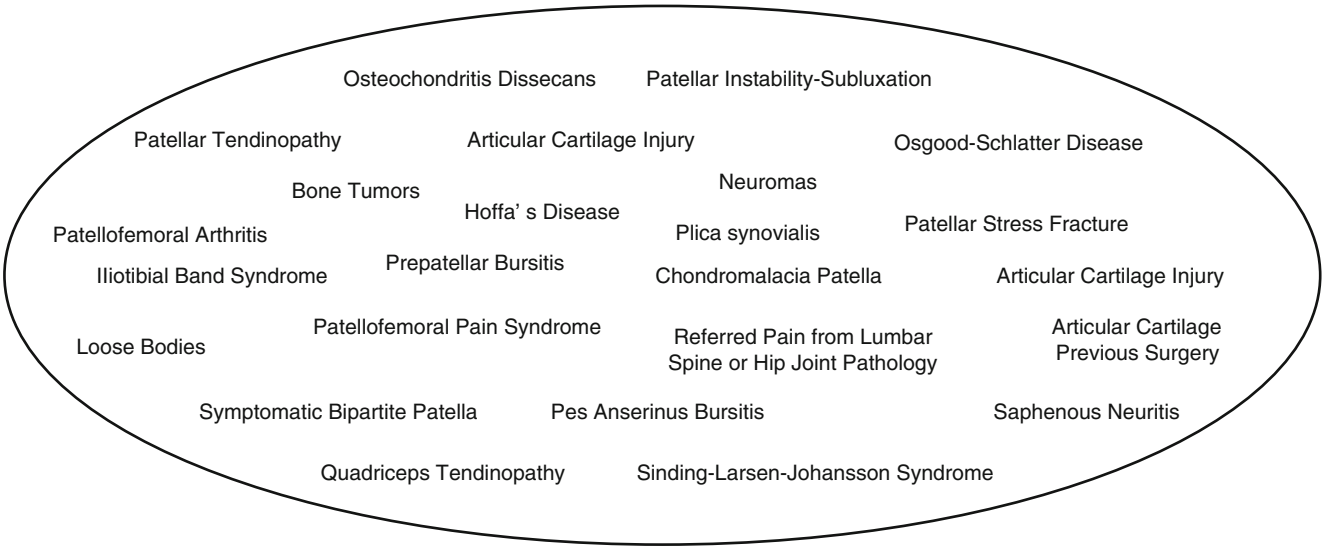


Fig. 46.1 Common pathologies that cause anterior knee pain

displacement of the patella causing the articulating pressure to be on the lateral facet.

- Despite these results, however, it is not clear whether the M. vastus lateralis or medialis imbalance is the primary cause for patellar maltracking [17, 18].
- *Quadriceps angle* (*Q angle*): its static role is controversial; in theory an increased Q angle is associated with increased lateral patellofemoral contact pressures and patellar dislocation, while decreasing the Q angle, we have major medial tibiofemoral contact with consequently higher incidence of *PFPS*. Rather a dynamic or functional malalignment is seen in these patients with an increased knee abduction moment that causes a dynamic valgus position of the knee joint that can influence patella tracking leading to lateralisation of the patella and have a role in the pathogenesis of PFPS [19, 20].
- *Role of the hip musculature*: Prins Mr et al. showed, in particular in women, a weakness of hip external rotators, abductors (M. gluteus medius and minimus) and iliopsoas causes an internal rotation of the femur, an increased Q angle and consequently a functional malalignment in the knee and increased PF joint stresses. In addition strong hip adductors serve as a stable origin for VMO contraction [21].
- *Iliotibial tract*: through anatomical correlations to the lateral retinaculum and patella, it will increase the lateral force vector on the patella during flexion to increase the lateral PF joint stresses [22].
- *Hamstring*: they can have a possible role; two studies showed a significant association between PFPS and hamstring tightness, underlining the imbalance in the lateral and medial hamstring contraction due to the increased joint contact force and joint stress, while another doesn't show it [23, 24].
- *Gastrocnemius and soleus*: the tightness of these muscles causes a tibial internal rotation which will cause femoral internal rotation to increase the Q angle and higher PF joint stresses [13].
- *Generalised ligamentous/joint laxity*: it should increase the patellar mobility which would alter patellar tracking and lead to symptoms [13].
- *Limb length discrepancy*: proposed as a probable risk factor for *patellofemoral pain* [25].
- *Genu varum*, *genu valgum*, *pes cavus and pes planus*: they have not been found to contribute to PFPS or other related conditions. Additional research is needed [13].
- *Disorders of the foot mechanics*: delayed timing of peak rear-foot eversion, increased rear-foot eversion at heel strike, reduced rear-foot eversion range, increased navicular drop, navicular drift and dorsiflexion and pronated foot type can be present in patients with PFPS [26].
- *Knee-spine syndrome*: Tsuji et al. examined the correlation between patellofemoral joint pain, lumbar lordosis and sacral inclination in elderly patients with anterior knee pain showing a significant difference in sacral inclination between subjects with and without anterior knee pain. For younger patients with *PFPS*, this mechanism has not been examined [27].
- *Psychological factors*: its role should not be underestimated. Studies suggested higher incidence of psychological and mental distress as depression, hostility, passive attitude and anxiety. In some cases, the knee problems can be triggered by secondary disease profit. This may play a role in young competitive athletes, who are no

longer capable of the increasing demands. The knee problems then may serve as an explanation for the stagnation or reduced performance [28].

- *Neurophysiological cause of pain*: the exact cause is unclear. A high expression of several neural markers such as neurofilament protein, S-100 protein, neural growth factor and substance P is observed in the lateral retinacula, Hoffa's fat pad and subchondral bone, so as an increased metabolic bone activity in patients with PFPS using F NaF PET/CT. A central mechanism may cause pain as a localised or distal hyperalgesia or an aberrant sensory function in PFPS patients. A possible role of cytokines as IL-1–6 or TNF-α has not yet been defined [6].
- *Neuromotor dysfunction*: EMG study showed a less activity of the vastus medialis, a difference in EMG onset, a faster VMO/VL reflex response time and an earlier fired VMO compared to the VL in PFPS patients [6, 13].

46.2 Clinical Presentation

Anterior knee pain is a dull, aching pain that is most often felt behind, below and on the sides of the kneecap (patella). One common symptom is a grating or grinding sensation when the knee is flexed, swelling and recurrent clicking. Symptoms may be more noticeable with the deep knee bends, going downstairs, running downhill, standing up after sitting for awhile, walking after prolonged sitting and pain at night or during activities that repeatedly bend the knee (i.e. jumping, squatting, running and other exercise, especially involving weightlifting) or related to a change in activity level or intensity, playing surface or equipment. The *anterior knee pain* may be the most common injury in sports and in athletes that often occurs after a change in training pattern, may present insidiously or may follow any injury to the knee, and it may terminate a promising athletic career [3, 6, 9].

46.3 Clinical Examination

Patient History

It is fundamental to make a diagnosis, because we have to investigate the multiple causes and the different ways of presentation of the *AKP* [6].

Physical Examination

A systematic and thorough physical examination, with the patient walking, standing, sitting, supine and prone, must be performed and include assessment of *static limb alignment* [femoral anteversion, knee position (varum, valgum, recurvatum), foot/ankle WB position, malalignment posture, Q angle, obesity, muscle atrophy, leg length discrepancy, torsional deformities], *dynamic limb alignment* (may exist during movement as a result of poor muscular control; can have patient step slowly up/down from stool or single-leg squats; presence of any abnormal movements of patella as it engages into trochlea, anybody sifting, trunk rotation and loss of hip control; excessive contralateral hip drop; hip adduction; knee abduction; tibial ER; hyperpronation), *range of motion*, *strength testing*, *ligamentous stability* and *neurovascular status*. The findings should be compared with the uninjured knee [2, 29, 30].

The prediagnostic examination for *PFPS* includes:

- J sign as result of lateral retinacular tightness or medial retinacular weakness.
- Ely test for the decreased quadriceps flexibility, specifically rectus femoris tightness.
- Ober test to value the decreased IT band flexibility.
- Thomas test to value the decreased hip flexor flexibility.
- Trendelenburg test to value weak hip abductors.
- Q angle measurement in excess of 20° may increase PFPS risk.
- Weak quadriceps or quadriceps atrophy.
- Altered VMO muscle reflex time compared to VL.
- Decreased vertical jump.
- Generalised ligamentous laxity [2, 29, 30].

46.4 Diagnostic Examination

X-Rays

Conventional radiography of the knee comprises a standard anteroposterior weight-bearing view, a lateral view and axial views with the quadriceps muscle relaxed and the knee flexed 45°. These let us value the vertical position of the patella, patellar subluxations, depth of the trochlea, bump sign, patellar height, Insall-Salvati index, Blackburne-Peel index, sulcus angle, lateral patellofemoral angle, congruence angle, patellofemoral index, patellar tilt, dysplastic condyles or crossing sign [4, 30–34].

CT Scan

CT scan is able to evaluate the PFJ relationships in different degrees of flexion to detect a malalignment and to assess the TT-TG distance [4, 30–34].

Magnetic Resonance Imaging (MRI)

Static MRI can visualise the components of the extensor mechanism and show lesions of articular cartilage and menisci, plicae, MPFL lesion, osteoarthritic change, muscle lesion, synovitis and joint effusion, with a limited radiation exposure to patients. *Kinematic MRI* can be used to assess the contribution of associated soft tissue structures to PFJ

function. And it is felt to be superior to CT imaging in the evaluation of patellofemoral tracking [4, 30–34].

Functional MRI has revealed that central activation of the brain mediates pain during OA and that nonsteroidal anti-inflammatory drug (NSAID) therapies may be partially acting via a central mechanism. fMRI, positron emission tomography (PET), single-photon emission computed tomography (SPECT) and magnetoencephalography (MEG) are novel imaging modalities that have helped unravel how central pain pathways in the perception of chronic pain function [4, 30–34].

46.5 Treatment

The literature provides evidence for a multimodal nonoperative therapy concept with short-term use of NSAIDs, short-term use of a medially directed tape and complex exercise programmes with the inclusion of the lower extremity and hip and trunk muscles. There is also low evidence for the use of patellar braces and foot orthosis. Recent studies have reemphasised that quadriceps muscle deficiency is a fundamental problem in patients with this condition. Treatment today should be individualised: every tight structure should be mobilised and the kinetic chain balanced appropriately for the individual patient. A specific bracing or taping programme and an aerobic low-impact conditioning programme suited to the patient should be designed. Nonoperative treatment is effective for most patients. The surgery is indicated only in selected cases [30, 35, 36].

46.6 Physical Rehabilitation

Physiotherapy is the most used therapy for *PFPS*. Many rehabilitation regimes exist for the treatment of *AKPS*. These include quadriceps strengthening, stretching, specific warm-up and warm-down sequences, core stability and hip strengthening. Muscle-specific electrostimulation has also been described. Theoretically all structures that can predispose to *AKP* must be treated [2, 6, 37].

Quadriceps strengthening encompasses a range of techniques. The exercises can involve concentric (muscle shortening), eccentric (muscle lengthening), isotonic (constant strain without change in muscle length), isometric (knee position is constant), isokinetic (constant contraction through a range of movement at constant velocity) and plyometric (explosive muscle contraction) activities [2, 6, 37].

Furthermore, exercises can be subdivided into closed or open chain. Generally eccentric exercises are closed chain involving cycles, step repetitions or squats. Open-chain exercises are generally isotonic or isometric, e.g. straight leg raises. Stretching exercises are an important component of physiotherapy for *AKPS* [2, 6, 37].

Literature shows a large number of studies that investigated the effects of physiotherapy on AKPS, but these are characterised by a high heterogeneity, different inclusion criteria and outcomes and short follow-up and often of low quality; in general they show a general efficacy of the different protocols [38–43].

Two meta-analyses have been published. In 2003, Heintjes et al. [44] published a Cochrane review about exercise for *PFPS*. This meta-analysis identified one high- and two low-quality studies comparing exercise with a control group without exercise. This meta-analysis reported the positive effects for pain reduction for patients treated with exercise. One of the low-quality studies even reported also functional improvement in the exercise groups.

In a more recent meta-analysis published in 2008, 10 prospective randomised studies could be analysed. All these studies showed a positive effect of exercise on pain reduction [45]. Positive results have been described in particular with active stretching exercises, squats, ergometer, static quadriceps exercises, active leg raises, leg press and raising and lowering climbing exercises. Four of the exercise programmes also included exercises to strengthen the hip abductors. In one study, trunk-stabilising exercises, including the rectus abdominis, were analysed. The most frequent duration of the exercise programmes was 6 weeks. The exercises were conducted two-to-four times daily with 10 repetitions. Closed- and open-chain exercises, balance training and proprioceptive exercises have been described. Eight studies also included stretching exercises. In 8 studies, additional interventions such as restriction of symptom-inducing activities, tape, braces and NSAIDs were allowed. In summary, there is strong evidence for exercise in the treatment of PFPS in the literature. These exercises should address the hip muscles, trunk stability, quadriceps, hamstrings and iliotibial tract [37–45].

46.7 Tape

The aim of classical taping is to modify *patella tracking* by applying adhesive tape stripes to the skin [46–48]. The tape should apply a medially directed force to counteract lateral patella maltracking. Literature showed as the medially directed taping is able to correct lateral patella maltracking and patellar tilt and to active earlier the vastus medialis obliquus (VMO) and have a positive effect on the symptoms of *PFPS*, but these are only short-term studies. One meta-analysis was published in 2002 and found that adhesive tape combined with exercise was significantly superior to an exercise programme alone regarding functional improvement and decrease in pain [46]. Another meta-analysis was published by Warden et al. in 2008 and showed that medially directed tape produces a clinically meaningful reduction in knee pain in patients with PFPS [47].

Therefore, the current evidence supports the use of tape as a temporary pain-relieving treatment of *anterior knee pain* in PFPS patients. The positive influence of the tape on pain and function probably explains the synergistic effect of tape and physiotherapy. The simultaneous application of a restraining tape and a physiotherapy exercise programme achieved a better role as the sole tape system [46–48].

46.8 Patella Braces

The role of patella braces is controversial, but it should be limited because some authors showed a positive effect on pain and function, while others didn't find these effects; so better-designed studies should be performed to evaluate the effect of braces on patients with PFPS [46, 47, 49].

46.9 Foot Orthosis

Literature analysis didn't show neither a role nor a synergist effect of foot orthosis in patients with PFPS. Foot orthosis might be a treatment option for patients with the combination of disorders of foot posture and PFPS; some predictors for the efficacy of foot orthoses in PFPS patients are a body height less than 165 cm, age older than 25, lower pain levels and midfoot abnormalities [50–52].

46.10 Pharmacological Therapy

On the use of drug therapy for *anterior knee pain*, there have been many trials assessing the effects of nonsteroidal anti-inflammatory medication (NSAIDs) and steroids usually in combination with physiotherapy, but none of these provide a definitive evidence on the efficacy. No significant differences were found when comparing aspirin to placebo and naproxen to placebo; the usefulness of glycosaminoglycan polysulphate (GAGPS) or steroid intra-articular injections is contradictory, while the use of botulinum toxin type A (Dysport) injections helped to achieve improved pain reduction and function in comparison to saline placebo in patients with chronic knee pain with quadriceps muscle imbalance [53].

46.11 Surgical Treatment

A surgical approach can be necessary if conservative treatment fails. A lot of procedures have been proposed and often it is needed to combine them [2]. The arthroscopy can have a diagnostic role or can allow to do specific procedure, but it's not recommended in patients with AKP. Kettunen et al. showed in a prospective randomised study with PFPS patients that an arthroscopy, in combination with physiotherapy, had no positive effect compared to physiotherapy alone; therefore, the treatment of PFPS is primarily nonoperative [54, 55].

In the past lateral retinacular release was well accepted as a primary surgical procedure for patients with resistant *patellofemoral pain* with ill-defined indications [2, 4, 56, 57]. Today we know that this approach is appropriate for patients with a tight lateral retinaculum associated with rotational (tilt) malalignment of the patella and an excessive lateral pressure [2, 4, 56, 57]. Lateral retinacular restraints are important in blocking excessive lateral displacement of the patella, as well as in limiting medial displacement, so an inappropriate or overzealous release can be a problem. In most patients, release to the level of the proximal patellar pole, maintaining some vastus lateralis obliquus muscle support on the lateral side, is all that is necessary [2, 4, 56, 57].

Balance of support structures around the front of the knee is important, as is proper *patella tracking*. Some patients require a proximal medial imbrication or a reconstruction of the medial patellofemoral ligament for a medial patellar instability or a proximal realignment or a reconstruction of patella-tibial and patella-meniscal ligaments [2]. The importance with the surgery is to avoid any overload of a painful or potentially painful articular lesion [2, 58].

Medial Patellofemoral Ligament Reconstruction. The reconstruction of primary passive restraint can be necessary if there's a lateral patellar dislocation, reducing the risk of redislocation and with a satisfaction rate of over 80 % [59].

Retinacular and Synovial Problems. Some patients have an isolated source of pain in the synovium, peripatellar retinaculum, patellar tendon or soft tissue around the front of the knee. A superficial neuroma related to previous surgery can cause intractable pain that can be treated simply by excision of the neuroma, once it is identified [2, 60].

ACL Reconstruction and Patellofemoral Pain. Many studies underline the high incidence of anterior knee pain after ACL reconstruction [2]. This can be due to changes in patellofemoral tightness or to the site of a bone-patellar tendon-bone graft or to an alteration of the patellar alignment or an inappropriate graft placement or a scarring behind the patellar tendon or a delay in the motion after the reconstruction [61]. Surgery in these patients should be specific for the clinical findings and may include infrapatellar contracture release, tibial tubercle anteriorisation, patellar tendon or harvest site debridement, lateral release, steep anteromedial tibial tubercle transfer, notchplasty, cyclops debridement, neuroma resection or a combination of these procedures [2, 61, 62].

Patellofemoral Salvage Surgery in Athletes. Unfortunately, there are some athletes and vigorous persons who become severely disabled by anterior knee pain and fail to improve or are made worse by surgery. Such patients require very careful attention. Often the treatment goal may be a return to pain-free

or even less painful daily activity instead of sports. Nonetheless, there are situations in which vigorous activity or return to full athletic activity may be possible after the correction of previous patellofemoral surgery failure. Selection of the proper salvage surgical procedure is not really much different from planning primary surgery except that the surgeon should look carefully for evidence of retinacular scar related to previous surgery, infrapatellar contracture, extensive cartilage damage, medial patella instability or a previously overlooked source of pain, sometimes in an area other than the patella-femoral joint. One must also recognise reflex sympathetic dystrophy, complicating psychiatric issues and secondary gain from legal or compensation issues. Most patients, however, including those with workers' compensation issues, have real pain and need help [2, 6, 55–62].

Conclusion

The *anterior knee pain* is a large chapter of the knee disorders, and the definition by Stanley James as the "black hole of orthopaedics" is still valid today. The aetiopathogenesis is multifactorial and the symptoms are typicals, therefore is necessary an accurate clinical and diagnostic examination. There are no guidelines about the treatment; the nonoperative treatment is of choice but it must be individualised and multimodal. The surgical treatment is necessary only in selected cases, and it is important to avoid unnecessary surgery because its consequences are often the cause of pain.

References

1. Post WR (2005) Patellofemoral pain: results of nonoperative treatment. Clin Orthop Relat Res 436:55–59
2. Fulkerson JP (2002) Diagnosis and treatment of patients with patellofemoral pain. Am J Sports Med 30(3):447–456
3. Llopis E, Padròn M (2007) Anterior knee pain. Eur J Radiol 62:27–43
4. Schutzer SF, Ramsby GR, Fulkerson JP (1986) The evaluation of patellofemoral pain using computerized tomography. A preliminary study. Clin Orthop Relat Res 204:286–293
5. Grelsamer RP (2005) Patellar nomenclature: the Tower of Babel revisited. Clin Orthop Relat Res 436:60–65
6. Sanchis-Alfonso V (2006) Anterior knee pain and patellar instability. Springer, London
7. Patellofemoral semantics: the Tower of Babel. The International Patellofemoral Study Group (1997) Am J Knee Surg 10(2):92–95
8. Boling M, Padua D, Marshall S et al (2010) Gender differences in the incidence and prevalence of patellofemoral pain syndrome. Scand J Med Sci Sports 20(5):725–730
9. Petersen W, Ellermann A, Gosele-Koppenburg A et al (2013) Patellofemoral pain syndrome. Knee Surg Sports Traumatol Arthrosc. doi:10.1007/s00167-013-2759-6
10. Dye SF (2005) The pathophysiology of patellofemoral pain: a tissue homeostasis perspective. Clin Orthop Relat Res 436:100–110
11. Waryasz GR, McDermott AY (2008) Patellofemoral pain syndrome (PFPS): a systematic review of anatomy and potential risk factors. Dyn Med. doi:10.1186/1476-5918-7-9
12. Dvorak J, Junge A, Chomiak J et al (2000) Risk factor analysis for injuries in football players. Possibilities for a prevention program. Am J Sports Med 28(5 Suppl):S69–S74
13. Witvrouw E, Lysens R, Bellemans J et al (2000) Intrinsic risk factors for the development of anterior knee pain in an athletic population. A two-year prospective study. Am J Sports Med 28(4):480–489
14. Lankhorst NE, Bierma-Zeinstra SM, van Middelkoop M (2013) Factors associated with patellofemoral pain syndrome: a systematic review. Br J Sports Med 47(4):193–206
15. Draper CE, Besier TF, Santos JM et al (2009) Using real-time MRI to quantify altered joint kinematics in subjects with patellofemoral pain and to evaluate the effects of a patellar brace or sleeve on joint motion. J Orthop Res 27(5):571–577
16. Wilson NA, Press JM, Koh JL et al (2009) In vivo noninvasive evaluation of abnormal patellar tracking during squatting in patients with patellofemoral pain. J Bone Joint Surg Am 91(3):558–566
17. Pal S, Draper CE, Fredericson M et al (2011) Patellar maltracking correlates with vastus medialis activation delay in patellofemoral pain patients. Am J Sports Med 39(3):590–598
18. Cowan SM, Bennell KL, Hodges PW et al (2001) Delayed onset of electromyographic activity of vastus medialis obliquus relative to vastus lateralis in subjects with patellofemoral pain syndrome. Arch Phys Med Rehabil 82(2):183–189
19. Park SK, Stefanyshyn DJ (2011) Greater Q angle may not be a risk factor of patellofemoral pain syndrome. Clin Biomech (Bristol, Avon) 26(4):392–396
20. Rauh MJ, Koepsell TD, Rivara FP et al (2007) Quadriceps angle and risk of injury among high school cross-country runners. J Orthop Sports Phys Ther 37:725–733
21. Prins MR, van der Wurff P (2009) Females with patellofemoral pain syndrome have weak hip muscles: a systematic review. Aust J Physiother 55(1):9–15
22. Wu CC, Shih CH (2004) The influence of iliotibial tract on patellar tracking. Orthopedics 27(2):199–203
23. Patil S, Dixon J, White LC et al (2011) An electromyographic exploratory study comparing the difference in the onset of hamstring and quadriceps contraction in patients with anterior knee pain. Knee 18(5):329–332
24. White LC, Dolphin P, Dixon J (2009) Hamstring length in patellofemoral pain syndrome. Physiotherapy 95(1):24–28
25. Brady RJ, Dean JB, Skinner TM et al (2003) Limb length inequality: clinical implications for assessment and intervention. J Orthop Sports Phys Ther 33:221–234
26. Barton CJ, Levinger P, Crossley KM et al (2012) The relationship between rearfoot, tibial and hip kinematics in individuals with patellofemoral pain syndrome. Clin Biomech (Bristol, Avon) 27(7):702–705
27. Tsuji T, Matsuyama Y, Goto M et al (2002) Knee-spine syndrome: correlation between sacral inclination and patellofemoral joint pain. J Orthop Sci 7(5):519–523
28. Jensen R, Hystad T, Baerheim A (2005) Knee function and pain related to psychological variables in patients with long-term patellofemoral pain syndrome. J Orthop Sports Phys Ther 35(9):594–600
29. Post WR (1999) Clinical evaluation of patients with patellofemoral disorders [current concepts]. Arthroscopy 15:841–851
30. Schulz B, Brown M, Ahmad CS (2010) Evaluation and imaging of patellofemoral joint disorders. Oper Tech Sports Med. doi:10.1053/j.otsm.2009.12.015
31. Dejour H, Walch G, Nove-Josserand L et al (1994) Factors of patellar instability: an anatomic radiographic study. Knee Surg Sports Traumatol Arthrosc 1:19–26
32. Merchant AC (2001) Patellofemoral imaging. Clin Orthop Relat Res 389:15–21
33. Beaconsfield T, Pintore E, Maffulli N et al (1994) Radiological measurements in patellofemoral disorders. Clin Orthop Relat Res 308(18–28):1994

34. Thomas S, Rupiper D, Stacy GS (2014) Imaging of the patellofemoral joint. Clin Sports Med 33(3):414–436
35. Collins NJ, Bisset LM, Crossley KM et al (2012) Efficacy of nonsurgical interventions for anterior knee pain: systematic review and meta-analysis of randomised trials. Sports Med 42:31–49
36. Oliveira VC, Henschke N (2013) Multimodal physiotherapy is effective for anterior knee pain relief. Br J Sports Med 47:245–246
37. Al-Hakim W, Jaiswal PK, Khan W et al (2012) The non-operative treatment of anterior knee pain. Open Orthop J 6(Suppl 2: M10):320–326
38. Earl JE, Hoch AZ (2011) A proximal strengthening program improves pain, function, and biomechanics in women with patellofemoral pain syndrome. Am J Sports Med 39(1):154–163
39. Coppack RJ, Etherington J, Wills AK (2011) The effects of exercise for the prevention of overuse anterior knee pain: a randomized controlled trial. Am J Sports Med 39(5):940–948
40. Collado H, Fredricson M (2010) Patellofemoral pain syndrome. Clin Sports Med 29(3):379–398
41. Clark DI, Downing N, Mitchell J et al (2000) Physiotherapy for anterior knee pain: a randomised controlled trial. Ann Rheum Dis 59(9):700–704
42. McMullen W, Roncarati A, Koval P (1990) Static and isokinetic treatments of chondromalacia patella: a comparative investigation. J Orthop Sports Phys Ther 12:256–266
43. Harrison EL, Sheppard MS, McQuarrie AM (1999) A randomized controlled trial of physical therapy treatment programs in patellofemoral pain syndrome. Physiother Can 51(2):93–106
44. Heintjes E, Berger MY, Bierma-Zeinstra SM et al (2003) Exercise therapy for patellofemoral pain syndrome. Cochrane Database Syst Rev (4):CD003472
45. Harvie D, O'Leary T, Kumar S (2011) A systematic review of randomized controlled trials on exercise parameters in the treatment of patellofemoral pain: what works? J Multidiscip Healthc 4:383–392
46. D'hondt NE, Struijs PA, Kerkhoffs GM et al (2002) Orthotic devices for treating patellofemoral pain syndrome. Cochrane Database Syst Rev (2):CD002267
47. Warden SJ, Hinman RS, Watson MA Jr et al (2008) Patellar taping and bracing for the treatment of chronic knee pain: a systematic review and metaanalysis. Arthritis Rheum 59(1):73–83
48. Callaghan MJ, Selfe J (2012) Patellar taping for patellofemoral pain syndrome in adults. Cochrane Database Syst Rev. doi:10.1002/14651858
49. Powers CM, Ward SR, Chen YJ et al (2004) Effect of bracing on patellofemoral joint stress while ascending and descending stairs. Clin J Sport Med 14(4):206–214
50. Vicenzo B, Collins N, Crossley K et al (2008) Foot orthoses and physiotherapy in the treatment of patellofemoral pain syndrome: a randomised clinical trial. BMC Musculoskelet Disord 9:27–34
51. Wiener-Ogilvie S, Jones RB (2004) A randomised trial of exercise therapy and foot orthoses as treatment for knee pain in primary care. Br J Podiatry 7(2):43–49
52. Hossain M, Alexander P, Burls A et al (2011) Foot orthoses for patellofemoral pain in adults. Cochrane Database Syst Rev (1): CD008402
53. Heintjes E, Berger MY, Bierma-Zeinstra SM et al (2004) Pharmacotherapy for patellofemoral pain syndrome. Cochrane Database Syst Rev (3):CD003470
54. Kettunen JA, Harilainen A, Sandelin J et al (2007) Knee arthroscopy and exercise versus exercise only for chronic patellofemoral pain syndrome: a randomized controlled trial. BMC Med 13(5): 38–45
55. Kettunen JA, Harilainen A, Sandelin J et al (2012) Knee arthroscopy and exercise versus exercise only for chronic patellofemoral pain syndrome: 5-year follow-up. Br J Sports Med 46(4):243–246
56. Panni AS, Tartarone M, Patricola A et al (2005) Long-term results of lateral retinacular release. Arthroscopy 21(5):526–531
57. Post WR (2005) Anterior knee pain: diagnosis and treatment. J Am Acad Orthop Surg 13(8):534–543
58. Aglietti P, Buzzi R, De Biase P et al (1994) Surgical treatment of recurrent dislocation of the patella. Clin Orthop 308:8–17
59. Panni AS, Alam M, Cerciello S et al (2011) Medial patellofemoral ligament reconstruction with a divergent patellar transverse 2-tunnel technique. Am J Sports Med 39(12):2647–2655
60. Kasim N, Fulkerson JP (2000) Resection of clinically localized segments of painful retinaculum in the treatment of selected patients with anterior knee pain. Am J Sports Med 28:811–814
61. Kartus J, Magnusson L, Stener S et al (1999) Complications following arthroscopic anterior cruciate ligament reconstruction. A 2- to 5-year follow-up of 604 patients with special emphasis on anterior knee pain. Knee Surg Sports Traumatol Arthrosc 7:2–8
62. Shino K, Nakagawa S, Inoue M et al (1993) Deterioration of patellofemoral articular surfaces after anterior cruciate ligament reconstruction. Am J Sports Med 21:206–211

Knee Arthritis in Athletes

47

Stefano Zaffagnini, Tommaso Roberti di Sarsina, Alberto Grassi, Federico Raggi, Tommaso Bonanzinga, Giulio Maria Marcheggiani Muccioli, and Maurilio Marcacci

47.1 Etiology

Osteoarthritis is the most common joint disease. Being exposed to a high-intensity and prolonged sports activity, athletes are particularly vulnerable to osteoarthritis as a long-term effect of such vigorous physical stress and results in irreversible pathological changes in affected joints.

The main symptoms are joint pain, stiffness, dysfunction, instability, deformity, swelling, and crepitus. These symptoms can be presented early in an athlete's career and lead to sports dismission.

The etiology of osteoarthritis is multifactorial and not fully understood. Age is the major independent risk factor of osteoarthritis; however, aging and osteoarthritis are interrelated, not interdependent. Cartilage senescence is related to other factors, both intrinsic (e.g., alignment, overloading) and extrinsic (e.g., genetics) to the joint [1]. In the young patient, the pathogenesis of knee osteoarthritis is predominantly related to an unfavorable biomechanical environment at the joint, which results in mechanical demand that exceeds the ability of a joint to repair and maintain itself, predisposing the articular cartilage to premature degeneration [2].

Systemic risk factors are thought to make the joint vulnerable to local factors and are thereby associated with the development of OA. They include age, gender, hormonal status, and genetics. Local risk factors cause abnormal biochemical loading on joints and include obesity, occupational activities (squatting, kneeling, lifting), joint injury, and high-level sports participation.

In this chapter, we will focus on those mechanical factors that accelerate this process in the younger sportive population.

S. Zaffagnini (✉) • M. Marcacci
Clinica Ortopedica e Traumatologica II, Laboratorio di Biomeccanica ed Innovazione Tecnologica, Istituto Ortopedico Rizzoli, Via di Barbiano, 1/10, Bologna 40136, Italy
e-mail: stefano.zaffagnini@biomec.ior.it

T. Roberti di Sarsina • A. Grassi • F. Raggi • T. Bonanzinga
G.M. Marcheggiani Muccioli
Clinica Ortopedica e Traumatologica II, Laboratorio di Biomeccanica ed Innovazione Tecnologica, Istituto Ortopedico Rizzoli, Bologna, Italy
e-mail: tommaso.robertidisarsina@hotmail.com

47.2 Injury Mechanism

47.2.1 Malalignment

Varus or valgus malalignment of the lower extremity results in an abnormal load distribution across the medial and lateral tibiofemoral compartment. For example, a 4–6 % increase in varus alignment increases loading in the medial compartment by up to 20 % [3].

Articular cartilage and subchondral bone are subjected to an increased stress, suggesting that axial malalignment plays an important role in the development of early OA.

However, studies examining the relationship between malalignment and early knee osteoarthritis have produced conflicting results.

A possible relationship between the incidence of early osteoarthritic changes and axial malalignment is only supported by limited evidence so far.

In contrast, the correlation between the progression of early osteoarthritic changes and axial malalignment has been well established. Both conventional radiological and MRI [4] studies found that axial malalignment is a potent risk factor for progression of early osteoarthritic changes in patients with axial malalignment. Articular cartilage loss and subchondral bone changes may lead to an increased malalignment. Cicuttini et al. [5] reported that the degree of varus knee angle was associated with a reduction in the volume of both femoral and tibial articular cartilage in the medial tibiofemoral compartment of the knee over a 1.9-year follow-up period. Similar results were seen in the lateral tibiofemoral compartment.

The rationale for high tibial osteotomy (HTO) is slowing or preventing early osteoarthritic changes by restoring a more favorable biomechanical situation, thereby reducing local

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_47,

compartmental overload, via correction of malalignment. However, the long-term effectiveness of HTO for treatment of medial compartment osteoarthritis has not been experimentally confirmed to date.

Everything suggests that malalignment is related to increased occurrence and faster progression of osteoarthritic changes, and high tibial osteotomy shows promising results even though there is no evidence of such change prevention. Such practice must be studied deeper to increase our knowledge and to justify this practice.

47.2.2 Loss of Meniscal Tissue

Any substantial loss of meniscal tissue from injury or iatrogenic meniscectomy permanently alters knee joint biomechanics and biology [6]. Subtotal or total meniscectomy increases the risk of secondary osteoarthritic changes by a factor 14 when compared to matched controls [7], eventually resulting in radiographic changes in 30–70 % of patients [8]. The role of concomitant cartilage damage related to the trauma that resulted in meniscal injury in the first place, or of iatrogenic damage related to the meniscectomy procedures, has not been determined, but is likely to play a role as well. The younger the patients, the worse the outcomes, especially in those with associated articular comorbidities such as chondral damage, ligamentous instability, and malalignment [9].

47.2.3 Meniscectomy

Initially described as vestigial, nonfunctional tissue, the menisci have since been found to play a vital role in load transmission in the knee. They transmit 50 and 70 % of the medial and lateral compartment load, respectively, with the knee in extension. This increases to almost 85 % when the knee is flexed to 90° [10]. The menisci also serve as an important secondary restraint to anteroposterior joint translation in unstable knees, that is, knees with deficient anterior cruciate ligament. Biomechanical studies have demonstrated significant alteration in load transmission with meniscal deficiency or mismatch [9].

Biomechanical and animal models of meniscal repair demonstrated near-normal load transmission [11], providing a rationale for meniscal repair when possible. The same study, however, pointed out the challenges posed by radial tears, which even after successful healing demonstrated decreased contact area.

Clinical data reflect the biomechanical changes observed experimentally. Radiographic changes in the knee joint after meniscectomy were noticed as early as 1939; however, Fairbank was the first to describe in 1948 a consistent pattern of ridge formation, femoral flattening, and joint space narrowing in 107 patients that had undergone open, most likely complete meniscectomy [12]. Jackson in 1968 reviewed 577 meniscectomized knees, demonstrating increasing numbers of patients with degenerative changes and osteoarthritic symptoms with longer follow-up, reaching 67 and 33 %, respectively, at 30-year follow-up [13]. These findings were confirmed subsequently by multiple authors, supporting Fairbank's theory that meniscectomy predisposes the knee to osteoarthritis, especially when concomitant injuries or abnormalities are present, such as instability or malalignment. Relatively few experimental studies on this subject have been performed in vivo besides Voloshin and Wosk [14] who were able to demonstrate a 20 % reduction in the shock absorption capacity of the knee after meniscectomy.

47.2.4 Meniscal Transplantation

The changes in biomechanical and biological environment of the knee joint following loss of meniscal tissue, either traumatic or following meniscectomy, and the deleterious consequences of those changes on the articular cartilage are well known.

This leads to the meniscal transplantation rationale: to restore the optimal biomechanical environment that has demonstrated an improved contact area and peak stresses and a reduction in tibial translation and thus in ACL strains, being the menisci a secondary stabilizer of the knee.

Of course sizing, positioning, and fixation technique for meniscal transplants appear to have important impact on biomechanical results.

In literature, it is reported that even though degenerative changes were not avoided, they were reduced in comparison to meniscectomized controls.

Overall, patients needing a meniscal transplant without malalignment or following high tibial osteotomy had far better results.

47.2.5 Cartilage Defects

Cartilage lesions should be evaluated carefully in order to assess their depth: they are divided into partial-thickness and full-thickness defects, plus osteochondral lesions.

Partial-thickness lesions are usually less symptomatic (symptoms are usually related to bone and periarticular tissue changes), and there is little evidence regarding their progression onto osteoarthritis, while full-thickness chondral or osteochondral lesions are believed to predispose to premature osteoarthritis [15–17].

This kind of lesions are of a common finding in asymptomatic patients (up to 20 % during arthroscopy and up to 40 % as MRI findings), and it is not clear which lesion and under which circumstances progress to osteoarthritis.

Various animal models have helped us to understand the biology of cartilage repair; however, due to the anatomical and biomechanical differences, they cannot give evidence on the natural history of cartilage defects in humans.

An unanswered issue still remains: do our cartilage repair strategies stop or slow down osteoarthritis process?

At present, only the classical autologous chondrocyte implantation (ACI) technique has a prospective follow-up of over 10 years.

What we know is that the repair tissue does not have the mechanical properties of native hyaline cartilage, leaving the rim of the defect exposed to increased stress. Clinical data with sufficient long-term follow-up regarding different treatment options are not yet available. However, what seems to be the aim of treatment still remains the restoration of the biomechanical environment to near normal.

47.2.6 Joint Instability or Laxity

We can define instability as a shift from the primary load bearing areas to a different location, resulting in overloading of part of the articular cartilage, with a change in both static and dynamic loading with increased stress through the articular cartilage.

The ACL is the most commonly injured knee ligament and it is a primary constraint to anteroposterior joint translation, and isolated lesions are uncommon. Frequently, other ligamentous structures or the menisci are affected, leading to further compromission of joint stability.

However, there is a lack of evidence that anterior cruciate ligament reconstruction or meniscus repair prevents the development of osteoarthritis in the long term. There is evidence of radiographic osteoarthritic changes in 50–80 % of injured knees even after adequate ACL reconstruction [18].

This can be due to a persistent excessive tibial rotation during demanding activity. This is the case of athletes whose return to high functional, demanding sport is allowed by ACL reconstruction.

In conclusion, joint instability or laxity seems to play an important role in the development of early osteoarthritis even though more studies are needed to better understand and justify our everyday handling of ligamentous injuries.

47.3 Treatment Strategy

There are two approaches to early osteoarthritis, conservative and surgical.

Conservative approach is usually the first choice in early knee degenerative processes, with no clear lesions or associated abnormalities requiring surgical procedures.

If we exclude the oral medication (NSAIDs, COX inhibitors) and exercise, of which we will not cover here, conservative treatment trend nowadays involves injective treatments.

Corticosteroid intra-articular therapy was firstly described in 1951 by Hollander [19] and the first clinical trial by Miller et al. [20]. Since then, short-term benefits of intra-articular corticosteroids are well established and universally accepted; however, long-term benefits have not been confirmed and repeated use is controversial, since they might facilitate tissue atrophy, joint destruction, or cartilage degeneration.

Viscosupplementation, which involves the use of intra-articular injections of hyaluronic acid (HA), adds this glycosaminoglycan to the joint, providing lubrication and shock adsorbency, and acts as a backbone for the proteoglycans of the extracellular matrix.

HA on the market differ in molecular weight, method of preparation, dose instructions, biologic characteristics, and possible clinical outcome. Clinical trials do not confirm that differences in molecular weight have any impact on clinical efficacy, and it has not been shown that higher molecular weight is related to higher efficacy [21].

Another injective procedure based on the use of autologous blood derivatives has been developed since the mid-1990s in order to obtain an injectable material enriched with endogenous interleukins or growth factors that can lead to cartilage repair or at least reduced cartilage degeneration.

Platelet-rich plasma (PRP) is gaining more and more attention due to the pools of growth factors stored in platelet α-granules that take part in cartilage regulation.

Blood is harvested and centrifuged to separate and concentrate platelets, which are injected into the joint. Kon et al. [22] published a pilot study on 100 patients, with evidence of safety, pain reduction, and improved function. The evaluation performed at 2-year follow-up [23] showed an overall worsening and showed a median duration of the beneficial effect of 9 months. It is interesting that better results were found in patients with no clear signs of osteoarthritis, suggesting indication for early osteoarthritis.

However, no well-designed high-level studies have been found in literature to support its efficacy, and one of the main reasons can be found in the heterogeneous products used.

Conservative management with physical therapy should be prescribed for at least 3–6 months before thinking about surgery, and it must comprehend activity modification and weight normalization.

Injective therapy must be considered especially for those patients eligible for joint replacement within few years.

Preoperative counseling is fundamental in order to find the best patient-fitting solution and to set reasonable expectation.

Surgical intervention is considered after failure of conservative management and lack of other alternatives. Moreover, patients with systemic inflammatory disease, heavy smokers, and obese are not good candidates for cartilage repair.

47.4 Surgical Treatment

As stated before, varus or valgus malalignment of the lower extremity results in an abnormal load distribution across the medial and lateral tibiofemoral compartment, thus leading to unicompartmental OA. It is caused by local overload exceeding the resilience of the osteochondral unit, resulting in accelerated tissue degeneration.

Osteotomy is a very old surgical technique that remains an important procedure for salvage surgery in patients with unicompartmental OA, cartilage defects, and ACL or meniscal lesions. Surgical treatment of such lesions without malalignment correction leads often to poor results.

Indications for osteotomy are malalignment associated with unicompartmental OA, cartilage or meniscal lesions, and ligament instability.

Generalized OA affecting multiple compartments is considered a contraindication, and such patients should be considered for arthroplasty.

This procedure does not require permanent activity restriction, thus being suitable for young active patients.

Closing-wedge high tibial osteotomy in association with ACL reconstruction and/or meniscal allograft transplantation has shown good results in varus-angulated knees (combine ACL reconstruction and closing-wedge HTO for varus-angulated ACL-deficient knees).

Postoperatively, patients are instructed to wear a long-leg brace for the first 4 weeks after surgery.

Rehabilitation protocol, starting from the day after surgery, comprehends quadriceps muscle isometric exercises, straight leg raises, patellar mobilization, and electric muscle stimulation.

After 2 weeks, passive range-of-motion exercises (0–90°) with motorized hardware. Complete range of motion should be achieved at week 6. Patients are allowed to toe-touch weight bearing for the first 4 weeks to prevent excessive forces on the osteotomy site and, then, progressive weight bearing. Swimming pool exercise and stationary bike should start from week 5, running 4 months after surgery, and return to sports activity after 8 months [24].

To address focal chondral defects, osteochondral autograft transfer represents a good single-stage technique that involves harvesting and implanting of autologous osteochondral plugs.

From a small incision, plugs are harvested from lesser weight-bearing regions such as the medial and lateral margins of the trochlea, the intercondylar notch, or the sulcus terminalis of the lateral femoral condyle [25].

Patients should not be over 50 and present with a full-thickness focal chondral defect that should be smaller than 4 cm^2. This procedure should not be performed in case of advanced OA, inflammatory disease, uncorrectable ligamentous instability, or malalignment.

Rehabilitation starting from day one after surgery should engage in passive motion without ROM limitations, except in case of patellofemoral lesions.

Toe-touch weight bearing for the first 6 weeks, then progressive.

If muscle mass is restored and ROM is complete, full athletic activity is permitted 4 months after surgery, and a return to sport at the preoperative level should be achieved after 6–8 months.

However, donor site morbidity has been reported, and up to 50 % of patients after surgery reported pain in the donor site.

Marcacci et al. [26] focused on the correlation between lesion size and outcomes suggesting to apply only a limited number of plugs. However, in literature, good results have been reached in lesions up to 4 cm^2. Also, clinical trials showed that this technique is superior to microfracture and as good or better than autologous chondrocyte implantation (ACI) in small to medium lesions [27, 28].

In patients with bigger lesions, results are expected to be inferior than for focal lesion; however, they have few alternative treatment options. Autologous chondrocyte implantation (ACI) is one of those.

This technique developed almost 20 years ago addresses large chondral defects. This technique utilizes nowadays biodegradable scaffold, mostly collagen or hyaluronan-based, as cell carriers, facilitating implantation and maintaining chondrocyte-differentiated phenotype, which are mostly done arthroscopically.

This surgical technique is indicated for young active patients with large chondral lesions, after a careful evaluation of comorbidities such as malalignment and meniscal or ligamentous insufficiency that should be addressed concurrently or before ACI.

The implant is very delicate and vulnerable for at least the first 6 weeks, so weight-bearing restriction and limited motion is suggested.

A CPM machine is used for 6 weeks in association with isometric exercise for the quadriceps. Strengthening and proprioceptive exercises are added after 6 weeks, when the use of a stationary bike and elliptical trainer is also allowed. Return to running and contact sports is delayed until at least 12–18 months to allow graft maturation.

Cavallo et al. [29] showed that hyaluronan is able to recreate an ideal environment for the cells. Their results suggest that the scaffold might favor the activation of anabolic factors, which induce chondrocyte differentiation and reduce the expression and production of catabolic molecules, thus negating the differences between cells derived from normal and degenerated cartilage. Histological and biochemical analysis showed that OA does not inhibit the regeneration process, confirming an important role for bioengineering.

Filardo et al. [30] analyzed a group of patients with degenerative cartilage lesions treated with arthroscopic second-generation hyaluronan scaffold ACI. All the scores evaluated showed a statistically significant improvement at medium-term follow-up. However, the number of failures was quite high: 18.5 % at 6-year follow-up.

Aging and joint overuse, as in athletes, may lead to degenerative or traumatic meniscal lesions. Treatment may include meniscal resection, meniscal suture, or meniscal replacement using scaffolds or allografts.

Meniscal scaffolds are indicated in case of history of meniscal injury with loss of >25 % of meniscal tissue both traumatic and iatrogenic, in absence or minimal chondral damage. They require some residual meniscal tissue for attachment, thus being contraindicated in meniscectomized patients without anterior/posterior horn attachments and a circumferential rim.

Meniscal allograft transplantation is indicated in young patients with history of meniscectomy and pain in the meniscus-deficient compartment, in ACL-deficient patients with previous medial meniscectomy who can benefit from this second stabilizer, and in young athletic patients to avoid early joint degeneration, prior to symptoms onset.

In patients with advanced chondral degeneration or evidence of significant osteophyte formation or femoral condyle flattening, this procedure is contraindicated. Other contraindications are obesity, instability, synovial defects, inflammatory disease, and previous joint infection.

Rehabilitation guidelines for meniscal scaffolds include limited weight bearing and motion for the first 6–8 weeks and return to sports after 6 months.

In case of meniscal allograft transplantation, weight bearing is not permitted for 3 weeks followed by 3 weeks of partial weight bearing and progression to full weight bearing between weeks 6 and 10.

ROM is limited to 30° during the first 2 weeks and increased by 30° every 2 weeks. Proprioceptive training is started after week 3. Swimming is allowed after week 6 and biking after week 12. Running should not be introduced before week 2.

Meniscal scaffold has the general risks associated with meniscal repair, and allografts have specific risks related to the transplant itself that is disease transmission from the donor and injury to the patellar tendon due to the anterior approach.

Zaffagnini et al. [31] reported 10-year follow-up in 33 male patients after either Menaflex (ReGen Biologics, USA) or partial medial meniscectomy (PMM) alone based on patient choice. The Menaflex group showed significantly lower pain and higher objective IKDC, Tegner index, and SF-36 scores; the Lysholm score did not show any significant difference. Radiographic evaluation showed significantly less medial joint space narrowing in Menaflex patients, and MRI scores remained constant between 5 and 10 years after surgery.

Verdonk et al. reported on 52 patients after Actifit (Orteq, UK) implantation. At 3 months postimplantation, MRI showed evidence of tissue ingrowth in the peripheral half of the scaffold in 86 % of patients. At 12 months, MRI showed stable or improved cartilage scores compared to baseline, and statistically significant improvements were reported for IKDC functionality, Lysholm, VAS knee pain, and KOOS subscale at 6, 12, and 24 months after surgery.

There is clinical evidence to support meniscus allograft transplantation in meniscectomized painful knees. Significant pain reduction and functional improvement have been reported in a high percentage of patients and appear to be long lasting, preventing further cartilage degeneration [32]. There seems to be a strong rationale for adding meniscal transplantation to cartilage repair procedures motivated by the well-known deleterious effects of meniscal loss and positive outcomes.

Marcacci et al. [33] published the results after 36-month follow-up after arthroscopic meniscus allograft transplantation in male professional soccer players. What they showed is that at 36 months from surgery, 92 % of the players were able to return to play soccer and 75 % were able to return to their preinjury level of activity (Tegner score of 10) after arthroscopic meniscus allograft transplantation without bone plugs. It was to our knowledge the first study to report outcomes of arthroscopic MAT in male professional soccer players, suggesting its feasibility even in high-demanding athletes but with some limitations: the small sample size and the broad range of concomitant knee injuries, due to such complex patient knee comorbidities.

Conclusion

High functional demand and limited treatment options make early OA a challenging pathology to deal with. Conservative measures as physical therapy and injections are only palliatives that can provide short-term pain relief.

Cartilage repair represent a promising treatment option for such patients. We are seeing a rapid development of new promising technologies whose aim is to provide easier application techniques, less demanding rehabilitation, and better outcomes. Normalizing knee biomechanics with concurrent procedures such as meniscal transplantation and osteotomy still remains a crucial procedure to provide an adequate environment for these new technologies.

Joint replacement, indicated in older population, is controversial in younger patients who are less satisfied and experience a higher failure rate, because of a higher functional demand and thus a higher consumption and revision rate.

References

1. Loeser RF (2010) Age-related changes in the musculoskeletal system and the development of osteoarthritis. Clin Geriatr Med 26:371–386
2. Buckwalter JA, Martin JA, Brown TD (2006) Perspectives on chondrocyte mechanobiology and osteoarthritis. Biorheology 43:603–660
3. Tetsworth K, Paley D (1994) Malalignment and degenerative arthropathy. Orthop Clin North Am 25:367–377
4. Brouwer GM, van Tol AW, Bergink AP, Belo JN, Bernsen RM, Reijman M, Pols HA, Bierma-Zeinstra SM (2007) Association between valgus and varus alignment and the development and progression of radiographic osteoarthritis of the knee. Arthritis Rheum 56:1204–1211
5. Cicuttini F, Wluka A, Hankin J, Wang Y (2004) Longitudinal study of the relationship between knee angle and tibiofemoral cartilage volume in subjects with knee osteoarthritis. Rheumatology (Oxford) 43:321–324
6. Heijink A, Gomoll AH, Madry H, Drobnič M, Filardo G, Espregueira-Mendes J, Van Dijk CN (2012) Biomechanical considerations in the pathogenesis of osteoarthritis of the knee. Knee Surg Sports Traumatol Arthrosc 20(3):423–435
7. Roos EM, Ostenberg A, Roos H, Ekdahl C, Lohmander LS (2001) Long-term outcome of meniscectomy: symptoms, function, and performance tests in patients with or without radio- graphic osteoarthritis compared to matched controls. Osteoarthritis Cartilage 9:316–324
8. Alford JW, Lewis P, Kang RW, Cole BJ (2005) Rapid progression of chondral disease in the lateral compartment of the knee following meniscectomy. Arthroscopy 21:1505–1509
9. van Dijk CN, Tol JL, Struijs PAA (1997) Complications, rehabilitation and results of arthroscopic meniscectomy and meniscal repair: a review of the literature. J Sports Traumatol Rel Res 19:43–50
10. Ahmed AM, Burke DL (1983) In vitro measurement of static pressure distribution in synovial joints—part I: tibial surface of the knee. J Biomech Eng 105:216–225
11. Baratz ME, Fu FH, Mengato R (1986) Meniscal tears: the effect of meniscectomy and of repair on intraarticular contact areas and stress in the human knee. A preliminary report. Am J Sports Med 14:270–275
12. Fairbank TJ (1948) Knee joint changes after meniscectomy. J Bone Joint Surg Br 30B:664–670
13. Jackson JP (1968) Degenerative changes in the knee after meniscectomy. Br Med J 2:525–527
14. Voloshin AS, Wosk J (1983) Shock absorption of meniscectomized and painful knees: a comparative in vivo study. J Biomed Eng 5:157–161
15. Buckwalter JA (2002) Articular cartilage injuries. Clin Orthop Relat Res 402:21–37
16. Ding C, Cicuttini F, Scott F, Boon C, Jones G (2005) Association of prevalent and incident knee cartilage defects with loss of tibial and patellar cartilage: a longitudinal study. Arthritis Rheum 52:3918–3927
17. Heir S, Nerhus TK, Rotterud JH, Loken S, Ekeland A, Engebretsen L, Aroen A (2010) Focal cartilage defects in the knee impair quality of life as much as severe osteoarthritis: a comparison of knee injury and osteoarthritis outcome score in 4 patient categories scheduled for knee surgery. Am J Sports Med 38:231–237
18. Lohmander LS, Ostenberg A, Englund M, Roos H (2004) High prevalence of knee osteoarthritis, pain, and functional limitations in female soccer players twelve years after anterior cruciate ligament injury. Arthritis Rheum 50:3145–3152
19. Hollander JL (1951) The local effects of compound F (hydro cortisone) injected into joints. Bull Rheum Dis 2:3–4
20. Miller JH, White J, Norton TH (1958) The value of intraarticular injections in osteoarthritis of the knee. J Bone Joint Surg Br 40-B:636–643
21. Kon E, Filardo G, Drobnic M, Madry H, Jelic M, van Dijk N, Della Villa S (2012) Non-surgical management of early knee osteoarthritis. Knee Surg Sports Traumatol Arthrosc 20(3):436–449
22. Kon E, Buda R, Filardo G, Di Martino A, Timoncini A, Cenacchi A, Fornasari PM, Giannini S, Marcacci M (2010) Platelet-rich plasma: intra-articular knee injections produced favorable results on degenerative cartilage lesions. Knee Surg Sports Traumatol Arthrosc 18:472–479
23. Filardo G, Kon E, Buda R, Timoncini A, Di Martino A, Cenacchi A, Fornasari PM, Giannini S, Marcacci M (2011) Platelet-rich plasma intra-articular knee injections for the treatment of degenerative cartilage lesions and osteoarthritis. Knee Surg Sports Traumatol Arthrosc 19:528–535
24. Gomoll AH, Filardo G, Almqvist FK, Bugbee WD, Jelic M, Monllau JC, Puddu G, Rodkey WG, Verdonk P, Verdonk R, Zaffagnini S, Marcacci M (2012) Surgical treatment for early osteoarthritis. Part II: allografts and concurrent procedures. Knee Surg Sports Traumatol Arthrosc 20(3):468–486
25. Gomoll AH, Filardo G, de Girolamo L, Espregueira-Mendes J, Marcacci M, Rodkey WG, Steadman JR, Zaffagnini S, Kon E (2012) Surgical treatment for early osteoarthritis. Part I: cartilage repair procedures. Knee Surg Sports Traumatol Arthrosc 20(3):450–466
26. Marcacci M, Kon E, Delcogliano M, Filardo G, Busacca M, Zaffagnini S (2007) Arthroscopic autologous osteochondral grafting for cartilage defects of the knee: prospective study results at a minimum 7-year follow-up. Am J Sports Med 35: 2014–2021
27. Dozin B, Malpeli M, Cancedda R, Bruzzi P, Calcagno S, Molfetta L, Priano F, Kon E, Marcacci M (2005) Comparative evaluation of autologous chondrocyte implantation and mosaicplasty: a multicentered randomized clinical trial. Clin J Sport Med 15: 220–226
28. Hangody L, Dobos J, Baló E, Pánics G, Hangody LR, Berkes I (2010) Clinical experiences with autologous osteochondral mosaicplasty in an athletic population: a 17-year prospective multicenter study. Am J Sports Med 38:1125–1133
29. Cavallo C, Desando G, Facchini A, Grigolo B (2010) Chondrocytes from patients with osteoarthritis express typical extra- cellular matrix molecules once grown onto a three-dimensional hyaluronan-based scaffold. J Biomed Mater Res A 93:86–95
30. Filardo G, Kon E, Di Martino A, Patella S, Altadonna G, Balboni F, Visani A, Bragonzoni L, Marcacci M (2011) Second- generation arthroscopic autologous chondrocyte implantation for the treatment of degenerative cartilage lesions. A prospective 6-year follow-up Study. Knee Surg Sports Traumatol Arthrosc 20(9):1704–1713. doi:10.1007/s00167-011-1732-5
31. Zaffagnini S, Marcheggiani Muccioli GM, Lopomo N, Bruni D, Giordano G, Ravazzolo G, Molinari M, Marcacci M (2011) Prospective long-term outcomes of the medial collagen meniscus implant versus partial medial meniscectomy: a minimum 10-year follow-up study. Am J Sports Med 39:977–985
32. Zaffagnini S, Marcheggiani Muccioli GM, Grassi A, Bonanzinga T, Filardo G, Canales Passalacqua A, Marcacci M (2011) Arthroscopic lateral collagen meniscus implant in a professional soccer player. Knee Surg Sports Traumatol Arthrosc 19:1740–1743
33. Marcacci M, Marcheggiani Muccioli GM, Grassi A, Ricci M, Tsapralis K, Nanni G, Bonanzinga T, Zaffagnini S (2014) Arthroscopic meniscus allograft transplantation in male professional soccer players: a 36-month follow-up study. Am J Sports Med 42(2):382–388. doi: 10.1177/0363546513508763

Management of Anterior Cruciate Ligament Lesion in Adolescents

48

Corrado Bait, Matteo Denti, and Piero Volpi

Anterior cruciate ligament (ACL) lesions in adolescents represent a serious concern for the physician. The increased involvement of children and adolescents in organized sports beginning at an early age raises concern regarding risk and severity of sports injury. Inevitably with increased participation and training come an increasing number of sports injuries. Three types of youth sports injury, anterior cruciate ligament (ACL) injury, concussion, and physeal injury, are the focus of much recent media and scholarly attention given their frequency, potential for adverse long-term health outcomes, and escalating healthcare costs [1]. If not managed appropriately, they can also lead to long-term complications which could negatively affect ability to continue to participate in exercise and sports as well as threaten general health. Management of ACL injuries is an area of controversy because it can affect long-term growth and recovery as well as the ability to participate in sports. No well-designed epidemiologic studies to document ACL injury rates have been conducted in children younger than 14 years. Although there have been reports of sports-related ACL injuries in children as young as 5 years, the limited data available suggest that ACL disruptions in children younger than 12 years are rare [2]. In 1988, McCarroll et al. [3] found that of the 1,722 ACL injuries diagnosed over a 6-year period at their sports medicine center, 3 % were in children 14 years and younger; the authors conclude that a conservative approach, although reasonable, has not always been feasible in the young population who has a strong desire to pursue competitive athletics. The Norwegian ACL surgical registry collects data for all ACL surgeries performed at participating institutions nationwide. From 2004 to 2011, this registry recorded a total of only 8–9 ACL surgeries each year for children 11–13 years of age. This represents a small fraction (0.6 %) of the total number of ACL surgeries recorded each year (1,441) in this registry across all age groups. For the children who had surgery, the age at the time of injury ranged from 9 to 13 years. The ACL surgery rate for 12- to 13-year-olds (3.5 per 100,000 citizens) was substantially lower than that for 16- to 39-year-olds (85 surgeries per 100,000 citizens), the age group at highest risk [4]. Again, these numbers underestimate the actual injury rates, because they do not account for those treated nonoperatively. ACL injury risk begins to increase significantly at 12–13 years of age in girls and at 14–15 years of age in boys. Female athletes between 15 and 20 years of age account for the largest numbers of ACL injuries reported. The gender disparity in ACL injury rates among athletes begins to appear around the time of the growth spurt (12–14 years of age for girls and 14–16 years of age for boys), peaks during adolescence, and declines in early adulthood [5]. At the high school level, ACL injury rates in gender-comparable sports (soccer, basketball, baseball/softball, track, volleyball) are 2.5–6.2 times higher in girls compared with boys [6]. In college athletics, ACL injury rates are 2.4–4.1 times higher for women, and at the professional level, ACL injury rates for men and women are essentially equal [7]. The young athlete may be particularly vulnerable to sports injury because of the physical and physiological processes of growth; moreover, young athletes might also be at increased risk of injury because of immature or underdeveloped coordination, skills, and perception. Although ACL injuries account for approximately 3 % of all injuries in college sports, they account for 88 % of injuries associated with 10 or more days of time lost from sports participation. An ACL injury at an early age is a life-changing event. In addition to surgery and many months of rehabilitation, the treatment costs can be substantial ($17,000–$25,000 per injury), and the time lost from school and sports participation can have considerable effects on the athlete's mental health and academic performance [8].

C. Bait (✉) • M. Denti
Unità Operativa di Chirurgia del Ginocchio e Traumatologia dello Sport, Istituto Clinico Humanitas,
Via Manzoni 56, Rozzano, Milano 20089, Italy
e-mail: corrado.bait@humanitas.it; matteo@denti.ch.it

P. Volpi
Knee Orthopedics and Sport Traumatology Unit,
Humanitas Clinical Institute IRCCS, Milan, Italy

Head of Medical Staff FC Internazionale Milan, Milan, Italy
e-mail: piero.volpi@humanitas.it

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_48,

Beyond these more immediate effects, an ACL injury also has long-term health consequences. Regardless of the type of treatment, athletes with ACL injury are up to ten times more likely to develop early-onset degenerative knee osteoarthritis, a condition that not only limits one's ability to participate in sports but also often leads to chronic pain and disability. A systematic review of a series of long-term studies suggests that the rates of degenerative knee osteoarthritis 10–20 years after ACL injury are more than 50 % [9]. This means children and teenagers who suffer ACL injuries are likely to face chronic pain and functional limitations from knee osteoarthritis in their 20s and 30s.

48.1 Risk Factors

ACL injury risk in young athletes is probably multifactorial. Injury data from many fields demonstrate that numerous physical and psychological parameters affect ACL injury rates. Although ACL injury rates increase with age in both genders, girls have higher rates immediately after the growth spurt. It is likely that the increases in body weight, height, and bone length during pubertal development underlie the mechanism of increased risk of ACL injury with increasing age. During puberty, the tibia and femur grow at a rapid rate. This growth of the two longest levers in the human body translates into greater torques on the knee. In pubertal boys, testosterone mediates significant increases in muscular power, strength, and coordination, which affords them with greater neuromuscular control of these larger body dimensions. Pubertal girls do not experience this same growth spurt in muscular power, strength, and coordination, which likely explains their higher rates of ACL injuries compared with pubertal boys; the preadolescent athletes show no gender differences in ACL injury rates [10, 11]. Hormonal factors are also likely to play a role; however, results of studies investigating hormonal factors are both equivocal and controversial. Although the female knee appears to get slightly more lax, on the order of 0.5 mm, at mid-menstrual cycle, injuries tend to cluster near the start of menses at the polar opposite time in the cycle [12]. Activation of the quadriceps before the hamstrings, a pattern more frequently seen in female individuals, increases the anterior shear force that directly loads the ACL and also could be related to increased dynamic valgus alignment at initial contact during cutting and landing maneuvers. Although fatigue is often cited as a potential risk factor for ACL injury, there are relatively few published studies to support or refute this [13, 14]; certainly a greater weight and BMI have been associated with increased risk of ACL injury. A narrow intercondylar notch, where the ACL is housed, is proposed to increase ACL injury risk, because a narrow notch tends to be associated with a smaller, weaker ACL and also could cause increased elongation of the ACL under high tension. Some studies have shown that a narrow notch increases risk of ACL injury; however, others have shown no association between notch width and ACL injury [15, 16]. Subtalar joint overpronation, *flat foot*, has been associated with noncontact ACL injuries, likely because overpronation increases anterior translation of the tibia with respect to the femur, thereby increasing the strain on the ACL [17]. Generalized joint laxity and knee hyperextension were found to significantly increase the risk for ACL injury in female soccer players. Patients with ACL injury have significantly more knee recurvatum at 10° and 90° of hip flexion and an increased ability to touch palms to the floor. Athletes with generalized joint laxity had a 2.7 times greater risk of ACL injury than did those without generalized laxity, and those with increased anteroposterior laxity of the knee, as measured by a knee arthrometer, had an approximately three times greater risk of ACL injury than did those without such laxity. Joint laxity affects not only sagittal knee motion (hyperextension) but also coronal knee motion (valgus), which can strain the ACL and be related to increased risk in athletes [18].

48.2 ACL Tear

The most common mechanism of ACL injury is a noncontact pivoting motion on a fixed foot or a trauma with the knee in hyperextension or rotation. If a hemarthrosis develops within a few hours after the trauma in the absence of a bony injury, there is a 70 % chance of ACL injury [19]. The examiner should assess gait and alignment and range of motion and assess the affected joint and compare it with the contralateral joint, taking into account that most children may have hyperlaxity which decreases with maturity. Radiographs should be examined for bony injuries. Magnetic resonance imaging (MRI) can be useful, but may be no better than accurate clinical examination. In a pediatric athlete with an acute traumatic knee effusion, the Lachman test, anterior drawer test, and pivot shift test are clinical examinations that aid in making the diagnosis of an ACL tear. The Lachman test is considered the most accurate of the three commonly performed clinical tests for an acute ACL tear, showing a pooled sensitivity of 85 % (95 % confidence interval CI) and a pooled specificity of 94 % (95 % CI 92–95). The pivot shift test is very specific, namely, 98 % (95 % CI), but has a poor sensitivity of 24 % (95 % CI 21–27). Lastly, the knee arthrometer is an objective, accurate, and validated tool that measures, in millimeters, the amount of tibial translation relative to the femur while performing a Lachman test and, thus, augments the clinical examination when examining a patient with an ACL tear [20].

48.3 Management of ACL Tear

The management of ACL deficiency in skeletally mature children is still controversial, especially in terms of operative timing and surgical technique. Conservative

management is not recommended, as it is accompanied by marked reduction in activity, decline in functional performance, and development of early osteoarthritis. Historically, delayed anatomical ACL reconstructions were preferred [21] recommending extensive rehabilitation and return to activities with a brace to skeletal maturity and growth plate closure, to allow an anatomical adult-like reconstruction. The present trend favors early reconstruction, using either extra-physeal techniques in very young athletes or anatomical reconstruction techniques placing the tibial and femoral tunnels close to the center on the growth plate of the tibia and femur in young athletes closer to skeletal maturity [22]. An ACL tear in a child is not a surgical emergency; multiple timely discussions with the parents and the child about the appropriate management options and understanding their goals and expectations are very important. The general indications for surgery are the patient's inability to participate in his or her chosen sport, instability that affects activities of daily living, and an associated repairable meniscal tear or a knee injury with multiple torn ligaments. Treatment of ACL injuries in the skeletally immature patient remains controversial, because standard ACL reconstructions involve the use of drill holes that cross the open physes and may potentially cause growth disturbance, such as shortening or angulation of the child's leg. A meta-analysis of 55 studies suggested that the risk of leg length difference or angular leg deviations was approximately 2 % after ACL reconstruction in children and adolescents. The authors recommended randomized controlled trials to clarify this risk more accurately [23]. But ACL surgery is about 90 % successful in restoring knee stability and patient satisfaction. Ideally, surgical treatment of an ACL tear in a skeletally immature athlete would be postponed until skeletal maturity, and the athlete would not develop meniscal tears during that waiting time. Most recent literature now supports early surgery for pediatric athletes with an ACL-deficient knee and recurrent episodes of instability [24]. No consensus exists on the best method to treat an ACL tear in a pediatric athlete. Safe and effective surgical techniques continue to evolve. However, the current literature suggests reasonable, evidence-based management options that minimize the risks of iatrogenic growth plate injury [25, 26]. The two principal ACL surgery techniques performed on a pediatric athlete are physeal sparing or transphyseal and all inside.

48.3.1 Physeal Sparing Technique (Both Tibia and Femur)

Various physeal-sparing techniques have been described for primary repair of ACL. They were designed to avoid placing drill holes across both the tibial and the femoral growth physes because primary repair of ACL injury is associated with high rate of instability and failure. However, Brief [31] and recently Kocher et al. [32] have described a technique that avoids placing the tunnels across both the femoral and the tibial physes. This technique utilizes the distally attached semitendinosus and gracilis tendons or the iliotibial band graft by passing them under the anterior horn of the medial meniscus, through the intercondylar notch, passing over the top, and attaching with staples above the physes of the lateral distal femoral condyle. There were no reports of growth disturbances in these patients at 36-month follow-up. Eight of the nine patients in the study said they had no instability and were satisfied with the result. Micheli et al. [33] also described the use of the iliotibial band as a femoral and tibial physeal-sparing technique in 17 prepubescent children. However, the validity of these techniques was limited by the small size of the number of patients and a relatively short-term follow-up.

48.3.2 Transphyseal and "All-Inside" Technique

This involves the transphyseal tibial and femoral passage of the graft. Athletes who were close to skeletal maturity were treated with standard techniques as in adults. Aichroth et al. [34] reported results of a prospective study of 45 adolescent patients treated for ACL injuries, whose average chronological age was 12.5 years. They used the four-strand hamstring technique. The drill holes originated from the anatomical footprint of the ACL and were oriented to cross the physes as perpendicular as possible. The mean follow-up period was 49 months. There were neither any leg length discrepancies nor any physeal arrest during the follow-up of these patients [24]. This study documented that placement of transphyseal tunnels may not cause clinically significant growth plate arrest when anatomy and choice of fixation devices are carefully planned and considered. The young average age of the patients studied (12.5 years) indicates that these patients had remaining growth potential which was not affected by ACL reconstruction.

Recently, different surgeons have described [27, 28] "all-inside" techniques for ACL reconstruction using the innovative FlipCutter® (Arthrex Inc. – Naples, Fl), an all-in-one guide pin and reamer that allows minimally invasive socket creation from inside/out in the tibia and femur, as perpendicular as possible to the physes, using only a quadruple semitendinosus with good results.

An accurate understanding of the athlete's physical maturity by determining skeletal age and Tanner stage helps to identify which treatment is best for a specific patient. The most common method of measuring the patient's skeletal age is to compare an anteroposterior radiograph of the patient's left hand and wrist to an age-specific radiograph in the

Greulich and Pyle atlas. Tanner stage can be determined by self-assessment, which has been shown to be valid and reliable [10]. Patients with open physes at Tanner stage III and skeletal age of less than 14 in girls and less than 16 in boys can be offered the option of activity modification, functional bracing, rehabilitation, and careful follow-up. Surgery is indicated in skeletally immature patients with a torn ACL and an additional repairable meniscal injury and in patients who failed conservative care. Rehabilitation after ACL surgery may need to be modified for the individual patient and the particular surgical procedure. In general, a graduated rehabilitation program emphasizing full extension, immediate weight bearing, active range of motion, and strengthening of the quadriceps, hamstrings, hip, and core can be started in the first few weeks after surgery. Progressive rehabilitation during the first 3 months after surgery includes range-of-motion exercises, patellar mobilization, proprioceptive exercises, endurance training, and closed-chain strengthening exercises. Straight-line jogging, plyometric exercises, and sports-specific exercises are added after 4–6 months. Return to play typically occurs 7–9 months after surgery [29].

48.4 Conclusions and Guideline for Physician (From the American Academy of Pediatrics) [30]

1. The number of ACL injuries in young athletes has increased over the past two decades, coincident with the growing number of children and adolescents participating in organized sports, intensive sports training at an earlier age, and greater rate of diagnosis because of increased awareness and greater use of advanced medical imaging.
2. Intrinsic risk factors for ACL injury include higher BMI, subtalar joint overpronation, generalized ligamentous laxity, and decreased neuromuscular control of the trunk and lower extremities.
3. ACL injury rates are low in young children and increase sharply during puberty, especially for girls, who have higher rates of ACL injuries than boys do in similar sports.
4. Although there likely are multiple factors underlying the differences in noncontact ACL injury rates in male and female athletes, neuromuscular control may be the most important and most modifiable factor.
5. ACL injuries often require surgery and/or many months of rehabilitation and substantial time lost from school and sports participation.
6. The best physical examination test for an ACL tear is the Lachman test.
7. MRI can be valuable for diagnosing ACL tears and associated meniscal and chondral injury in the pediatric athlete whose physical examination is difficult to perform because of pain, swelling, and lack of cooperation.
8. An ACL tear in a young athlete is not a surgical emergency. Multiple discussions with the athlete and parents may be needed to understand the athlete's goals and parental expectations and to educate the family about possible treatment options.
9. The patient's skeletal age, measured by an anteroposterior radiograph of the left hand and wrist, and Tanner stage are helpful for the physician in deciding the most appropriate treatment of an ACL tear in a skeletally immature athlete.
10. Pediatricians and orthopedic surgeons treating young people with ACL injuries should advise them that regardless of treatment choice, they are at increased risk of early-onset osteoarthritis in the injured knee. Such discussions should be appropriately documented in the patient's medical record.
11. Musculoskeletal changes that decrease dynamic joint stability in high-risk female athletes and potentially lead to higher injury rates in this population could be modified if neuromuscular training interventions are instituted in early-middle adolescence, when the neuromuscular risk factors for ACL injury start to develop.
12. Neuromuscular training appears to reduce the risk of injury in adolescent female athletes by 72 %. Prevention training that incorporates plyometric and strengthening exercises, combined with feedback to athletes on proper technique, appears to be the most effective.
13. Pediatricians and orthopedic surgeons should direct patients at highest risk of ACL injuries (e.g., adolescent female athletes, patients with previous ACL injury, generalized ligamentous laxity, or family history of ACL injury) to appropriate resources to reduce their injury risk.
14. Pediatricians and orthopedic surgeons who work with schools and sports organizations are encouraged to educate athletes, parents, coaches, and sports administrators about the benefits of neuromuscular training in reducing ACL injuries and direct them to appropriate resources.

References

1. Caine D, Purcel L, Maffulli N (2014) The child and adolescent athlete: a review of three potentially serious injuries. Sports Sci Med Rehab 6:22
2. Shea KG, Pfeiffer R, Wang JH, Curtin M, Apel PJ (2004) Anterior cruciate ligament injury in pediatric and adolescent soccer players: an analysis of insurance data. J Pediatr Orthop 24(6):623–628
3. McCarrol JR, Rettig AC, Shelbourne KD (1988) Anterior cruciate ligament injuries in the young athlete with open physes. Am J Sports Med 16(1):44–47

4. Granan LP, Forssblad M, Lind M, Engebretsen L (2009) The Scandinavian ACL registries 2004–2007: baseline epidemiology. Acta Orthop 80(5):563–567
5. Renstrom P, Ljungqvist A, Arendt E et al (2008) Non contact ACL injuries in female athletes: an International Olympic Committee current concepts statement. Br J Sports Med 42(6):394–412
6. Comstock R, Collins C, McIlvain N (2013) National High School sports related injury surveillance study. 2009–2010 school year summary. Columbus. www.nationwidechildrens.org/cirp-rio-study-reports. Accessed 28 Feb 2013
7. Arendt E, Dick R (1995) Knee injury patterns among men and women in collegiate basketball and soccer. NCAA data and review of literature. Am J Sports Med 23(6):694–701
8. De Loes M, Dahlstedt LJ, Thomée R (2000) A 7-year study on risks and costs of knee injuries in male and female youth participants in 12 sports. Scand J Med Sci Sports 10(2):90–97
9. Lohmander LS, Englund PM, Dahl LL, Roos EM (2007) The long term consequence of anterior cruciate ligament and meniscus injuries: osteoarthritis. Am J Sports Med 35(10):1756–1769
10. Tanner JM, Davies PS (1985) Clinical longitudinal standards for height and height velocity for North American children. J Pediatr 107(3):317–329
11. Hewett TE, Myer GD, Ford KR (2004) Decrease in neuromuscular control about the knee with maturation in female athletes. JBJS Am 86-A(8):1601–1608
12. Hewett TE, Zazulak BT, Myer GD (2007) Effects of the menstrual cycle on anterior cruciate ligament injury risk: a systematic review. Am J Sports Med 35(4):659–668
13. Myer GD, Ford KR, Hewett TE (2005) The effects of gender on quadriceps muscle activation strategies during a maneuver that mimics a high ACL injury risk position. Electromyogr Kinesiol 15(2):181–189
14. Nylan JA, Caborn DN, Shapiro R, Johnson DL (1997) Fatigue after eccentric quadriceps femoris work produces earlier gastrocnemius and delayed quadriceps femoris activation during crossover cutting among normal athletic women. Knee Surg Sports Traumatol Arthrosc 5(3):162–167
15. Shelbourne KD, Davis TJ, Klootwyk TE (1998) The relationship between intercondylar notch width of the femur and the incidence of anterior cruciate ligament tears. A prospective study. Am J Sports Med 26(3):402–408
16. Lombardo S, Sethi PM, Starkey C (2005) Intercondylar notch stenosis is not a risk factor for anterior cruciate ligament tears in professional male basketball players: an 11-years prospective study. Am J Sports Med 33(1):29–34
17. Trimble MH, Bishop MD, Buckley BD, Fields LC, Rozea GD (2002) The relationship between clinical measurements of lower extremity posture and tibial translation. Clin Biomech 17(4):286–290
18. Soderman K, Alfredson H, Pietila T, Werner S (2001) Risk factors for leg injuries in female soccer players: a prospective investigation during one out-door season. Knee Surg Sports Traumatol Arthrosc 9(5):313–321
19. Boden BP, Dean GS, Feagin GA Jr, Garret WE Jr (2000) Mechanism of anterior cruciate ligament injury. Orthopedics 23:573–578
20. Kocher MS, Saxon HS, Hovis WD, Hawkins RJ (2002) Management and complications of anterior cruciate ligament injuries in skeletally immature patients: survey of Herodicus Society and The ACL study group. J Pediatr Orthop 22:452–457
21. Pressman AE, Letts RM, Jarvis JG (1997) Anterior cruciate ligament tears in children: an analysis of operative versus nonoperative treatment. J Pediatr Orthop 17:505–511
22. Kaeding CC, Flanigan D, Donaldson C (2010) Surgical techniques and outcome after anterior cruciate ligament reconstruction in preadolescent patients. Arthroscopy 26:1530–1538
23. Frosch KH, Stengel D, Brodhun T (2010) Outcomes and risks of operative treatment of rupture of anterior cruciate ligament in children and adolescents. Arthroscopy 26(11):1539–1550
24. Kocher MS, Smith JT, Zoric BJ, Lee B, Micheli LJ (2007) Transphyseal anterior cruciate ligament reconstruction in skeletally immature pubescent adolescent. JBJS Am 89(12):2632–2639
25. Volpi P, Galli M, Bait C, Pozzoni R (2004) Surgical treatment of anterior cruciate ligament injuries in adolescents using double-looped semitendinosus and gracilis tendons: supraepiphysary femoral and tibial fixation. Arthroscopy 20(4):447–449
26. Anderson AF (2004) Transepiphyseal replacement of the anterior cruciate ligament using quadruple hamstring grafts in skeletally immature patients. J Bone Joint Surg Am 86-A(pt 2 suppl 1):201–209
27. McCarthy MM, Graziano J, Green DW, Cordasco FA (2012) All epiphyseal, all-inside anterior cruciate ligament reconstruction technique for skeletally immature patients. Arthroscopy Tech 1(2):e231–e239
28. Lubowitz JH, Schwartzberg R, Smith P (2013) Randomized controlled trial comparing all-inside anterior cruciate ligament reconstruction technique with anterior cruciate ligament reconstruction with a full tibial tunnel. Arthroscopy 29(7):1195–1200
29. Ardern CL, Webster KE, Taylor NF, Feller JA (2011) Return to sport following anterior cruciate ligament reconstruction surgery: a systematic review and meta-analysis of the state of play. Br J Sports Med 45(7):596–606
30. LaBella CR, Henrikus W, Hewett TE (2014) Anterior cruciate ligament injuries: diagnosis, treatment and prevention. Pediatrics 133:e1437
31. Brief LP (1991) Anterior cruciate ligament reconstruction without drill holes. Arthroscopy 7(4):350–357
32. Kocher MS, Garg S, Micheli LJ (2006) Physeal sparing reconstruction of the anterior cruciate ligament in skeletally immature prepubescent children and adolescent. Surgical technique. J Bone Joint Surg Am 88(Pt 2 Suppl 1):283–293
33. Micheli LJ, Rask B, Gerberg L (1999) Anterior cruciate ligament reconstruction in patients who are prepubescent. Clin Orthop Relat Res (364):40–47
34. Duri ZA, Patel DV, Aichroth PM, (2002) The immature athlete. Clin Sports Med 21(3):461–482

Management of the First Patellar Dislocation

49

Matteo Coviello and Claudio Mazzola

49.1 Etiology

First patellar dislocation is defined as a clinical entity that usually causes a traumatic disruption of the previously uninjured medial restraints of the patella with consequent hemarthrosis of the knee [1]. Acute patellar dislocation is a frequent injury that usually occurs during sport and physical activities, most often in adolescents. The annual incidence is 5.8 per 100,000 in the general population. The incidence increases to 29 per 100,000 in the 10- to 17-year age group [2]. Patellar dislocation can occur without any pathologic structures in the patellofemoral joint when the femur rotates internally and the tibia externally with the foot fixed on the ground (Fig. 49.1). Quite often, however, patellar dislocation is associated with predisposing anatomic features for patellar instability as trochlear dysplasia, patella alta, increased femoral antetorsion, increased external tibial torsion, lateral patellar tilt, vastus medialis muscle hypoplasia, subtalar joint pronation-valgus alignment of the lower limb, and ligamentous laxity (Table 49.1). The risk factors for primary patellar dislocation are tall height and excess weight [3].

49.2 Injury Mechanism

A complex interaction between muscles, ligaments, bone morphology, and lower extremity alignment influences patellar motion. The most important stabilizer of the patella beyond 30° of knee flexion is bony stability of the femoral trochlea. Additional stability is provided by active and passive restraint. Actively, the rectus femoris and vastus intermedius muscles

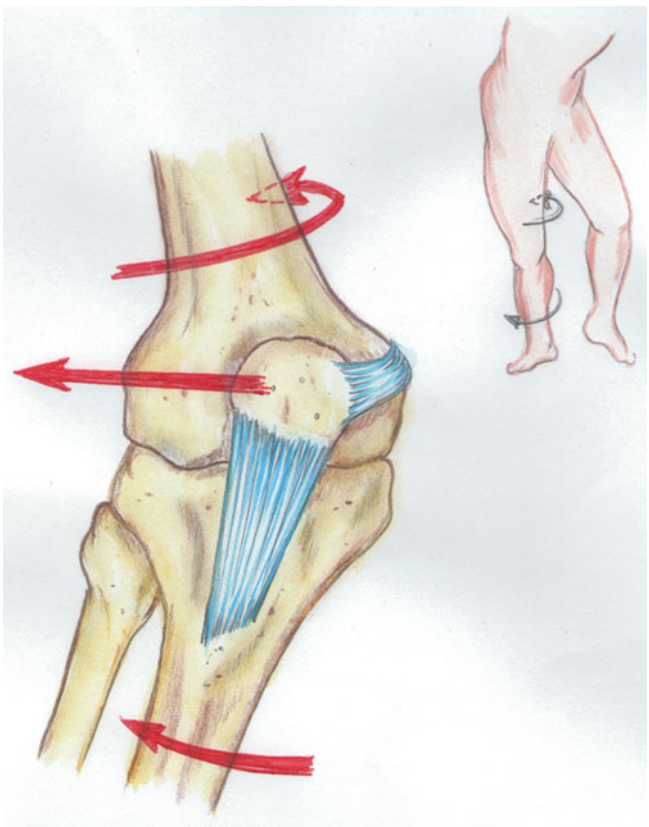


Fig. 49.1 Patellar dislocation can occur when the femur rotates internally and the tibia externally with the foot fixed on the ground

Table 49.1 Predisposing factors

Trochlear dysplasia
Patella alta
Increased femoral antetorsion
Increased tibial torsion
Lateral patellar tilt
Vastus medialis muscle hypoplasia
Subtalar joint pronation-valgus alignment of the lower limb
Ligamentous laxity
Tall height
Excess weight

M. Coviello (✉)
Clinica Ortopedica, IRCCS Azienda Ospedaliera Universitaria San Martino IST, Largo Rosanna Benzi 10, Genoa 16100, Italy
e-mail: coviellomatteo@hotmail.com

C. Mazzola
U.O.C. Ortopedia delle Articolazioni, E.O. Ospedali Galliera, Via Mura delle Cappuccine 14, Genoa 16128, Italy
e-mail: guimazz@alice.it

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_49,

act along the femoral axis, and the vastus lateralis and medialis help stabilize the patella in the medial/lateral plane. Passive restraints include patellar tendon, the medial menisco-patellar ligament, and the medial patellofemoral ligament (MPFL), a thin but relatively consistent ligament [4, 5]. The MPFL extends from the medial margin of the patella and attaches to the femur between the adductor tubercle and the medial epicondyle [6, 7]. The MPFL is important in initiating smooth entry into the trochlea with knee flexion and seems to be the major ligamentous restraint against patellar dislocation [8]. Traumatic primary patellar dislocation is almost always associated with hemarthrosis, MPFL injury, and medial retinacular disruption [3]. Clinically, 94–100 % of patients suffer from MPFL rupture after acute patellar dislocation [8].

49.3 Clinical and Diagnostic Examination

The initial evaluation of a first-time patellar dislocation should include an appropriate patient history, family history of patellar dislocation or hyperlaxity, and physical examination. Patients frequently report that their knee "gave way" and that they "felt a pop." Clinical findings include large effusion with tenderness about the medial retinaculum and hemarthrosis. The patella is usually spontaneously reduced, making the diagnosis less clear if the athlete cannot clearly identify what happened during injury. The clinician can rarely assist the reduction by applying a medially directed force with knee extension to push the patella over the lateral trochlea and back into the trochlear groove. If the patella does not easily reduce, the procedure should be aborted. A fracture fragment of the patella or condyle may limit the reduction and can be verified with radiographic studies [9].

Aspiration of the knee joint should be performed in patients with moderate to severe effusions. The presence of a hemarthrosis raises the likelihood that a significant osteochondral fracture has occurred. Clinical findings are not specific, so careful examination should be undertaken for anterior cruciate ligament, posterior cruciate ligament, and collateral and rotational laxity. Patellar apprehension and mobility should be assessed by medial and lateral patellar translation. A common physical examination sign to asses patella laxity is measuring the movement of the patella when passively stressed, recording the movement in quadrants of mobility as well as its endpoint firmness [10]. Increased lateral patella translation is suggestive of a lateral patellar dislocation.

Plain radiographs are always needed to evaluate patellar position and to asses osteochondral fractures, in particular, an AP-extended knee weight-bearing view, a Mercer-Merchant view (45° flexion weight-bearing view), and a 30° flexion lateral view. Both patellae should be included in the axial view. Even when the radiographs are normal, an osteochondral fracture may have occurred. Osteochondral fracture has been reported to be missed in 30–40 % of initial radiographs in both surgical and MRI studies [11, 12]. MRI is recommended to be performed quite soon after the injury to verify the diagnosis, evaluate additional injuries, asses the cartilage more precisely, classify the MPFL injury, and describe the anatomic factors of the patellofemoral joint (Fig. 49.2). MPFL injuries have been classified into three categories based on location: at the level of the MPFL patellar insertion, at the midsubstance, and at the femoral origin of the MPFL [13, 14]. Between 40 and 90 % of MPFL disruptions are located in the femoral attachment, whereas some studies have reported figures up to 50–60 % at the patellar insertion. The MPFL midsubstance region seems to be less frequently affected [15]. Recently, Sillanpää proposed a new classification for patellar insertion MPFL injuries: type P0 with ligamentous disruption, type P1 with bony avulsion fragment, and type P2 with bony avulsion involving articular cartilage surface from the medial facet of the patella [15].

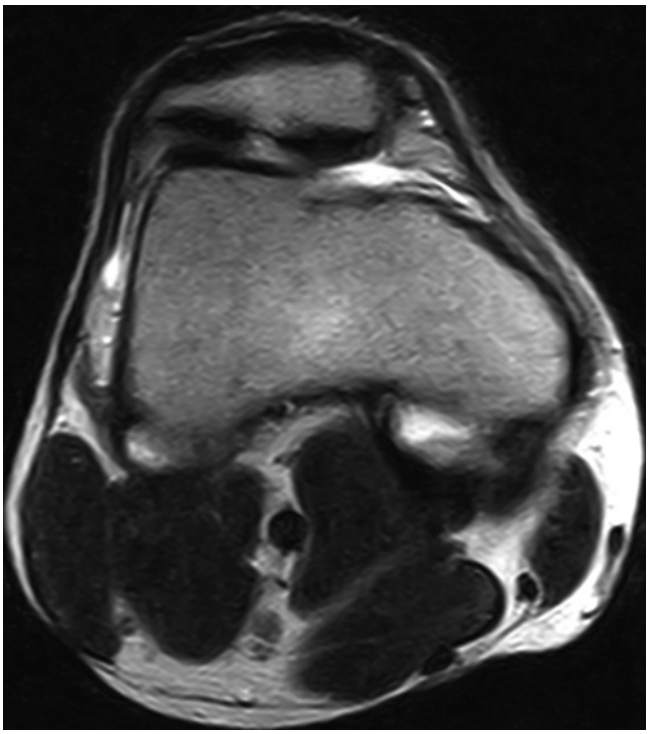


Fig. 49.2 Right knee MRI performed after first-time patellar dislocation. It can be noted a patellar osteochondral fracture at the MPFL insertion

49.4 Treatment Strategy

The initial management of a first-time traumatic patellar dislocation is controversial with no evidence-based consensus to guide decision-making. Conservative treatment has been historically suggested for patients with primary patellar dislocation. A short immobilization period is used for patient

comfort and is followed by formal physiotherapy. Therefore, the optimal conservative management has yet to be established [16]. The surgical treatment should be considered in specific conditions. These include the finding of an osteochondral fracture, a substantial disruption of the MPFL, and a laterally subluxated patella with normal alignment of the contralateral knee [17].

49.4.1 Nonoperative Treatment

Hemarthrosis can be aspirated for pain relief. It is important to prescribe rest, ice, compression, elevation, and NSAIDs. The drainage massage of the knee may be useful in order to reduce the post-traumatic edema. Common at this stage is the use of physical therapy for the resolution of edema and pain. The patient should be immobilized initially for comfort (3–4 weeks) to allow immediate weight bearing as tolerated on crutches with early mobilization. Immobilization in extension may allow for a better environment for MPFL healing, but this comes at the expense of stiffness, weakness, and loss of limb and proximal control [18]. The aims of physiotherapy are to restore knee range of motion and to strengthen the quadriceps muscles to restore the dynamic part of the patellar soft-tissue stabilizers. At 4–6 weeks, walking and knee range of motion should be normalized.

49.4.2 Surgical Options

Studies that observed a patellar dislocation recurrence rate of up to 44 %, findings of slow-developing symptoms with previous pain in the frontal region of the knee, and recurring instability rate above 50 % after nonoperative treatment led to an increase in initial treatment with surgical repair and reconstruction of the medial patellar stabilizers [19]. In literature, there is no sufficient evidence to confirm a significant difference in the outcome between surgical or nonsurgical initial management after first patellar dislocation [20]. Surgical intervention should be considered in specific conditions. These include the finding of an osteochondral fracture or major chondral injury, a substantial disruption of the MPFL-VMO-adductor mechanism, a laterally subluxated patella on the plain Mercer-Merchant view with normal alignment of the contralateral knee, a patient fails to improve with nonoperative management especially in the presence of predisposing factors to patellar dislocation, and subsequent redislocation [17].

There is a very high prevalence of MPFL lesion after first-time patellar dislocation. Clinically, up to 94–100 % of patients suffer from MPFL rupture [8]. As described earlier, MRI is suggested to classify MPFL injury. In literature, there are many methods described for the acute surgical repair, but no one seems to show better long-term outcomes compared to conservative treatment [21–25]. Camano et al. suggest that MPFL patellar or femoral attachment injury can be surgically reinserted with sutures or suture anchors with satisfying results and may lead to a better outcome than nonsurgical treatment [22]. In different prospective studies is yet described that operative treatment does not improve medium-term outcome [23–25]. Midsubstance MPFL injury should be repaired only in rare cases with extensive VMO fascial disruption in a high-energy dislocation when the VMO detaches from the medial patellar capsule, and the quadriceps pull vector may be significantly lateralized; therefore, the patella dislocates in extension when the quadriceps is activated [16].

Surgical open repair and fixation is recommended when the osteochondral fracture is greater than 10 % of the patella articular surface or part of the weight-bearing portion of the lateral condyle [26]. Fixation is performed with pins, screw sutures, or bioabsorbable nails. If the osteochondral fragment is smaller than 5×10 mm, especially when located in a low-pressure area, surgery treatment is not recommended. Small fracture that acts as a loose body and produces symptoms should be arthroscopically removed [27].

Mariani et al. described an arthroscopic technique for acute repair of patellar MPFL through reattachment at the patellar border with two trans-patellar sutures (Fig. 49.3). The potential benefit of performing an arthroscopic repair at medial hedge of the patella is to directly restore the MPFL to its anatomic position without associated surgical complications and to achieve a good tension of the ligament itself. In this prospective study, only one out of 17 patients treated with arthroscopic reinsertion of the MPFL reported one episode of patellar instability; 14 patients were able to return to sports at the same levels as before [28].

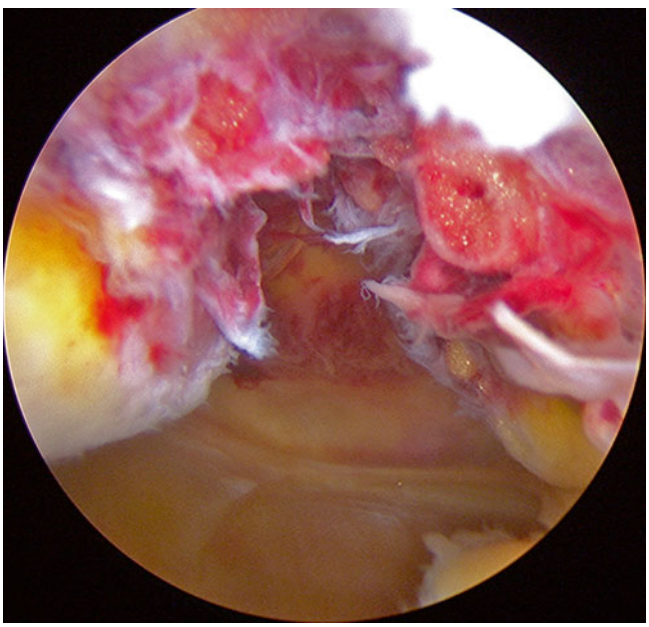


Fig. 49.3 MPFL injury arthroscopic view after the first patellar dislocation (Courtesy of Prof. PP Mariani)

In some cases, MPFL patellar or femoral attachment disruption can be accompanied by a midsubstance total or partial tear. Therefore, MPFL reconstruction may be the more reliable surgical method than MPFL repair. In a randomized controlled trial, Bitar et al. evaluated 44 patients with first patellar dislocation comparing the nonoperative treatment and MPFL reconstruction. They concluded that treatment with reconstruction of the MPFL with the patellar tendon produced better results, based on the analysis of posttreatment recurrences and the better final results of the Kujala questionnaire after a minimum follow-up period of 2 years [19].

Additional procedure involving osseous anatomy should be selected on an individual basis and is generally not suggested as a first-line treatment after first-time patellar dislocation. In this regard, skeletally immature patients and adolescents with primary dislocation could present severe trochlear dysplasia or alignment abnormalities that may need to be addressed whether surgery is performed after primary dislocation [29]. The clinical outcome of various acute patellar dislocation remains highly uncertain with regard to the factors that predispose a patient to patellar instability. Therefore, further randomized studies are required to stabilize whether the individual preexisting predisposing factors for instability and the pattern of the medial stabilizers injury could influence the operative or nonoperative management of the first acute patellar dislocation [30].

49.5 Rehabilitation and Return to Play

It is important to educate the patient about the importance of regaining muscle strength and dynamic stability. The VMO and glutes are the primary target of our work [31]. In order to achieve a proper and safe resumption of sports activities, there are critical points to achieve during rehabilitation: the strength of the lower limb muscles, especially the quadriceps and the gluteus medius. Therefore, the final goal of the rehabilitation program should be focused on the stability of the lower limb by the use of specific exercises on different surfaces, including cutting maneuvers, side hops, and sudden change of direction [32].

Testing exercises are mandatory to evaluate recovery and the competence of the injured or operated limb:

- Figure-of-eight test: the time taken to complete three laps around two circles each with a diameter of 4 m describing the shape of the number 8.
- Stair-running test: patient must run uphill and downhill on a flight of stairs to a total of 55 steps.

To make a judgment of suitability for the implementation of an athletic movement, we propose a series of three tests called functional performance tests (FPT):

- Co-contraction test: to make a series of jumps along a semicircle drawn on the ground while the subject is bound to the wall by an elastic cable. The protocol expected to perform five semicircles in the shortest possible time.
- Shuttle run test: the patient runs four routes of 6.1 m each, with stops and changes of direction. Tests are carried out for 3 times each and they are timed. The best times for each test are added together and constitute the FPT.

The monopodalic tests are valid indicators of strength and functional stability:

- Single-leg hop test: just one monopodalic jump.
- Triple-leg hop test: three consecutive monopodalic jumps; both limbs are tested by measuring the distance covered. Return to competition activity is considered appropriate when the difference between the two limbs is less than 10 %.
- Side-jump test: the knee is stressed not only in the sagittal plane as the previous ones but also in the transverse plane ensuring greater reliability to the evaluation of functional recovery [33].

Return to full activity can be suggested after 3 months of rehabilitation. The length of nonoperative treatment is recommended from 3 to 6 months, with emphasis that the exercise program should be continued over the long term to ensure optimal functioning [16].

References

1. Atkin DM, Fithian DC, Marangi KS et al (2000) Characteristics of patients with primary acute lateral patellar dislocation and their recovery within the first 6 months of injury. Am J Sports Med 28:472–479
2. Fithian DC, Paxton EW, Stone ML et al (2004) Epidemiology and natural history of acute patellar dislocation. Am J Sports Med 32:1114–1121
3. Sillanpaa P, Mattila VM, Iivonen T et al (2008) Incidence and risk factors of acute traumatic primary patellar dislocation. Med Sci Sports Exerc 40:606–611
4. Andrish J (2008) The management of recurrent patellar dislocation. Orthop Clin North Am 39:313–327
5. Bicos J, Fulkerson JP, Amis A (2007) Current concepts review: the medial patellofemoral ligament. Am J Sports Med 35:484–492
6. Laprade RF, Engerbretsen AH, Ly TV et al (2007) The anatomy of the medial part of the knee. J Bone Joint Surg Am 89:2000–2010
7. Warren LF, Marshall JL (1979) The supporting structures and layers on the medial side of the knee: an anatomical analysis. J Bone Joint Surg Am 61:56–62
8. Kang HJ, Wang F, Chen BC et al (2012) Nonsurgical treatment for acute patellar dislocation with special emphasis on the MPFL injury patterns. Knee Surg Sports Traumatol Arthrosc. doi:10.1007/s00167-012-2020-8
9. Skelley NW, McCormick JJ, Smith MV (2014) In-game management of common joint dislocations. Sports Health 6:246–255
10. Kolowich PA, Paulos LE, Rosenberg TD et al (1990) Lateral release of the patella: indications and contraindications. Am J Sports Med 18:359–365
11. Desio SM, Burks RT, Bachus KN (1998) Soft tissue restraints to lateral patellar translation in the human knee. Am J Sports Med 26:59–65

12. Stanitski CL, Paletta GA Jr (1998) Articular cartilage injury with acute patellar dislocation in adolescents. Arthroscopic and radiographic correlation. Am J Sports Med 26:52–55
13. Elias DA, White LM, Fithian DC (2002) Acute lateral patellar dislocation at MR imaging: injury patterns of medial patellar soft-tissue restraints and osteochondral injuries of the inferomedial patella. Radiology 225:736–743
14. Nomure E (1999) Classification of lesions of the medial patellofemoral ligament in patellar dislocation. Int Orthop 23:260–263
15. Sillanpaa PJ, Salonen E, Pihlajamaki H et al (2014) Medial patellofemoral ligament avulsion injury at the patella: classification and clinical outcome. Knee Surg Sports Traumatol Arthrosc 22(10):2414–2418. Epub ahead of print
16. Sillanpaa PJ, Maenpaa HM (2012) First-time patellar dislocation: surgery or conservative treatment? Sports Med Arthrosc 20:128–135
17. Stefancin JJ, Parker RD (2007) First-time traumatic patellar dislocation: a systematic review. Clin Orthop Relat Res 455:93–101
18. Buchner M, Baudendistel B, Sabo D et al (2005) Acute traumatic primary patellar dislocations: long term-results comparing conservative and surgical treatment. Clin J Sport Med 15:62–66
19. Bitar AC, Demange MK, D'Elia CO et al (2012) Traumatic patellar dislocation non operative treatment compared with MPFL reconstruction using patellar tendon. Am J Sports Med 40:114–122
20. Hing CB, Smith TO, Donell S et al (2011) Surgical versus non-surgical interventions for treating patellar dislocation. Cochrane Database Syst Rev (11):CD008106. doi:10.1002/14651858.CD008106.pub2
21. Sillanpaa PJ, Mattila VM, Maenpaa H et al (2009) Treatment with and without initial stabilizing surgery for primary traumatic patellar dislocation. A prospective randomized study. J Bone Joint Surg Am 91:263–273
22. Camanho GL, Viegas Ade C, Bitar AC et al (2009) Conservative versus surgical treatment for repair of the medial patellofemoral ligament in acute dislocations of the patella. Arthroscopy 25:620–625
23. Nikku R, Nietosvaara Y, Aalto K et al (2005) Operative treatment of primary patellar dislocation does not improve medium-term outcome: A 7-year follow-up report and risk analysis of 127 randomized patients. Acta Orthop 76:699–704
24. Palmu S, Kallio PE, Donell ST et al (2008) Acute patellar dislocation in children and adolescents: a randomized clinical trial. J Bone Joint Surg Am 90:463–470
25. Christiansen SE, Jakibsen BW, Lund B et al (2008) Isolated repair of the medial patellofemoral ligament in primary dislocation of the patella: a prospective randomized study. Arthroscopy 24: 881–887
26. Tsai CH, Hsu CJ, Hung CH et al (2012) Primary traumatic patellar dislocation. J Orthop Surg Res 7:21
27. Sillanpaa PJ, Peltola E, Mattila VM et al (2009) Femoral avulsion of the medial patellofemoral ligament after primary traumatic patellar dislocation predicts subsequent instability in men: a mean 7-year nonoperative follow-up study. Am J Sports Med 37: 1513–1521
28. Mariani PP, Liguori L, Cerullo G et al (2011) Arthroscopic patellar reinsertion of the MPFL in acute patellar dislocations. Knee Surg Sports Traumatol Arthrosc 19:628–633
29. Jain NP, Khan N, Fithian DC (2011) A treatment algorithm for primary patellar dislocations. Sports Health 3:170–174
30. Panni AS, Vasso M, Cerciello S (2013) Acute patellar dislocation. What to do? Knee Surg Sports Traumatol Arthrosc 21:275–278
31. McConnell J (2007) Rehabilitation and nonoperative treatment of patellar instability. Sports Med Arthrosc 15:95–104
32. Menetrey J, Putman S, Gard S (2014) Return to sport after patellar dislocation or following surgery for patellofemoral instability. Knee Surg Sports Traumatol Arthrosc 22(10):2320–2326. Epub ahead of print
33. Ricci G, Respizzi S (2011) Ritorno allo sport dopo trattamento conservativo. J Sport Traumatology 28:66–72

Chronic Ankle Instability

50

Alberto Ventura and Claudio Legnani

50.1 Anatomy and Biomechanics of the Ankle Ligamentous Complex

The *lateral ligamentous complex* of the ankle is composed by three major ligaments: the anterior talofibular ligament (ATFL), the calcaneofibular ligament (CFL), and the posterior talofibular ligament (PTFL) [1, 2]. The ATFL is continuous with the anterior joint capsule and runs from the anterior edge of the fibula lateral to the articular cartilage and inserts distal to the articular cartilage of the talar dome [1]. The ATFL is the primary restraint to inversion stress and is tensioned during increased plantar flexion. The CFL originates on the anterior edge of the fibula distal to ATFL and courses posteromedially deep to the peroneal tendons and inserts on the calcaneus distal to the subtalar joint. It acts as the primary lateral collateral ligament, and its strain is greatest in inversion and dorsiflexion; the CFL is considered one of the principal ligaments providing subtalar joint stability. The PTFL runs from the posteromedial aspect of the lateral malleolus to the lateral talar tubercle and is tensioned during increased dorsiflexion [2]. The *subtalar joint* has its own ligament system which comprises the interosseous talocalcaneal ligament which represents the pivot of rotatory stability and the cervical ligament which joins the astragalus to the lateral aspect of the calcaneus and is the first anterolateral stabilizer of the subtalar joint [3]. The syndesmosis is defined as the anterior inferior tibiofibular ligament (AITFL), running from the Chaput tubercle on the anterior tibia to the anterior distal fibula, the posterior inferior tibiofibular ligament (PITFL) which lies on the posterior aspect of the tibia and inserts on the posterior aspect of the distal fibula, the transverse tibiofibular ligament lying superior and anterior to the PITFL, and the interosseous membrane and ligament [4].

A. Ventura (✉) • C. Legnani
Minimally Invasive Articular Surgery Unit, G. Pini Orthopaedic Institute – Milan, P.zza A Ferrari, 1, Milan 20122, Italy
e-mail: alberto.ventura@doctoral.it

On the medial compartment, the strong deltoid ligament acts as a restraint against valgus tilt of the talus, while its deep fibers limit external rotation [5].

50.2 Etiology and Injury Mechanism

Within sportspeople, *ankle sprains* are the most frequently encountered injuries [6, 7]. This is especially true in basketball and soccer, with most of all injuries cited as ankle sprains [8]. It is well established that these injuries can have a considerable effect on athletic training schedules and can even interrupt participation in competitive sporting events as well as interfere with the simple activities of daily living. The most common mechanism of injury in lateral ankle sprains occurs with excessive plantar flexion and inversion of the ankle, with damage occurring to the lateral ankle ligamentous complex. Of all ankle sprains, nearly 85 % involve the lateral ligamentous complex [7]. The most commonly injured ligament is the anterior talofibular ligament (ATFL), followed by the calcaneofibular ligament (CFL). Syndesmotic injuries are seen with much less frequency (1–18 % of all ankle injuries) and may result from rotational injuries or forced abduction [4]. An injury of either the AITFL, PITFL, transverse ligament, or interosseous ligament leads to significant mechanical laxity.

Injuries to the medial ligamentous complex are usually caused by rotational injuries; different lesion patterns have been described: type 1 lesion consisting in a proximal avulsion of the deltoid ligament, type 2 in an intermediate tear, and type 3 resulting in a distal avulsion of the spring ligament [5]. Most patients with acute ankle sprains can be successfully managed with conservative treatment, such as bracing and physical therapy. However, approximately 15–20 % of patients will remain symptomatic, with the most commonly described complaints being ankle weakness, giving way, pain, and, occasionally, stiffness [9].

Chronic ankle instability (CAI) is commonly divided into functional and mechanical instability [3, 10]. Functional

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_50, © Springer International Publishing Switzerland 2016

instability is related to posture defects or tendon and muscle adjustment and usually occurs in the presence of proprioceptive deficits, without anatomic incompetence. Several authors have reported deficits in peroneal latency and electromechanical delay deficits following functional instability [11, 12]. Mechanical instability is characterized by anatomic abnormalities of the ankle and is usually related to *ligamentous laxity* [3, 10].

However, although this is the common classification reported in literature, it is not clear how many stages of mechanical instability exist. For prognostic purposes, it has recently been proposed to assess the severity of mechanical instability relying on the involvement of the anatomic structures of the ankle joint which are affected in this pathologic condition. It is important to recognize that subtalar instability is present in about 30 % of patients with chronic ankle instability. Therefore, it is essential to determine the integrity of the CFL both in the clinical setting and during surgery, since together with the cervical and the interosseous talocalcaneal ligament, it is the primary contributor to hindfoot stability [13]. For this reason, we suggested to distinguish between mild/moderate chronic ankle instability and severe chronic ankle instability, based on the involvement of the ATFL alone and of both ATFL and CFL [14]. The amount of ligament involved may be assessed through history, MRI findings, and clinical and radiographic stress tests.

50.3 Clinical and Diagnostic Examination

50.3.1 History and Physical Examination

The most common complaint is repeated giving way or repeated sprains in the ankle. Often it is accompanied with difficulty walking on uneven ground and sometimes with pain. Assessment of symptoms aids in the evaluation of the severity of CAI, together with physical examination.

Physical examination should include observation (presence of malalignment, effusion), range of motion (with any evidence of anterior or posterior impingement), and laxity tests.

The anterior drawer test measures ATFL laxity: anteroposterior displacement is compared between injured and uninjured ankles. Similarly, CFL laxity is assessed with the talar tilt test by inverting the hindfoot while maintaining maximum talocrural dorsiflexion.

However, the wide range of values in normal and injured ankles makes manual laxity tests inadequate to diagnose CAI. A previous study has found that *anterior drawer sign* has a sensitivity of 50 % and *talar tilt test* of only 36 % [15]. Thus, manual tests are reliable in identifying ankle instability, since specificity of both is 100 % [15], but they are not able to exclude the presence of ligament injury.

50.3.2 Imaging

Stress radiographs are also helpful for detecting joint instability. A displacement in the anterior drawer test is considered abnormal if it is greater than 3–5 mm, while the talar tilt test is considered pathologic for a side-to-side difference of more than 9°–10°. Increased values of talar tilt displacement suggest *subtalar instability* [16]. However, according to some studies, instrumented anterior ankle testing devices have been found unreliable in detecting increased ligament laxity [17]. Similar to manual stress tests, also radiographic talar tilt and anterior drawer stress tests are not reliable enough in the diagnosis of lateral ankle instability [18], and their diagnostic accuracy precludes their routine use [17].

Magnetic resonance imaging (MRI) and computed tomography (CT) scans can provide useful information on the integrity of the ATFL, CFL, muscle tendon units, and cartilage lesions on the talar dome. However, 2D images in most cases of chronic instability do not assess precisely the extent of the ligament lesion and do not give information on the degree of joint instability [19].

50.4 Treatment Strategies

50.4.1 Conservative Treatment

Usually nonoperative management is adopted in the initial presentation of ankle instability. It focuses on *proprioceptive training* and *peroneal muscle strengthening*. Joint taping may result in a more neutral ankle position during walking and jogging, and therefore this change in foot positioning and the mechanical properties of the tape act protectively in preventing lateral ankle sprains [20]. The use of bracing is debated for its risks of deconditioning the musculature as they are associated with changes in neuromuscular activity during walking [21]. Whenever the patient is unresponsive to conservative modalities, surgical repair is recommended to alleviate symptoms and resume activity.

50.4.2 Anatomical Direct Repair

The suture of the ATFL has been described by Broström and is considered an easy, inexpensive, anatomic, and minimally invasive procedure for the treatment of CAI [22]. It has led to promising results in the published literature [23]. However, as shown in the original paper, only the ATFL could be successfully repaired with excellent results [22]. Long-term follow-up reported a 16 % failure rate and 21–24 % of patients with fair or poor outcome score [24]. Thus, for improving ankle stability, the Gould modification of the Broström technique has been introduced [25]. The Broström

portion corrects the ATFL instability of the ankle, while the Gould portion reinforces the stabilization and indirectly affects subtalar instability. However, this technique can hardly be considered strictly "anatomical," since the rerouting of the extensor retinaculum alters the anatomy of the lateral ligamental complex. Prisk et al. demonstrated that neither lateral ligament reconstruction nor anatomic repair restores normal contact mechanics of the ankle joint, although anatomic graft reconstruction restores more closely ankle biomechanics and avoids increased contact areas beyond that of the normal ankle as it occurs after the Gould modification [26].

The Gould modification of the *Broström technique*, which dates back to the 1980s, is nowadays the most widespread procedure for the treatment of ankle instability disorders, and it is usually referred as the standard. However, no evidence exists supporting its role as a gold standard in the treatment of chronic ankle instability. It is certainly a simple, inexpensive technique, and its excellent outcomes have been supported by many papers. However, patients with previous failed ligament reconstruction, who are physically active and have a high BMI, or in cases where the ligament is absent or attenuated have reported poor outcomes with the use of this technique [27].

In addition, other pathologic features are frequently involved within the ankle joint that might not be treated by an isolated *Broström-Gould procedure* [28].

50.4.3 Tenodesis

Since common anatomical reconstruction techniques, such as the Broström procedure [22] and its modification according to Gould [25], may not provide adequate stability and lead to recurrence by using the weakened and scarred remnants, external ligament reconstruction techniques have been proposed [29–36]. *External ligamentoplasty* provides stability and reproduces the role of the main ankle stabilizers without having the disadvantages of using the weakened and scarred remnants of the ankle ligaments. Secondly these techniques allow to address subtalar joint instability brought by the CFL lesion. The use of autogenous rerouted tendons has the drawback of weaker muscle strength during eversion of the ankle. In order to allow the preservation of ankle stabilizing properties of the peroneus brevis muscle which is fundamental during early rehabilitation, the use of allogenic and synthetic grafts has been proposed [33, 34, 37]. Subtalar stiffness is a well-known disadvantage of restricting subtalar motion and represents the major criticism which is moved to these reconstructive procedures. Non-anatomical *tenodesis* techniques have been criticized for their invasiveness, related risks of neurovascular injuries, postoperative subtalar and tibiotalar joint stiffness, and altered biomechanics of and long-term degenerative joint disease [9]. For these reasons, common indications for non-anatomical procedures include patients with severe laxity, those with a previous failed stabilization surgery, those who impose heavy demands on the ankle, or those with associated subtalar joint instability. Several procedures of anatomical reconstruction of the lateral ligament complex have been recently proposed in order to restore the origin and insertion sites of native ligaments [34–36]. According to a recent meta-analysis, there is insufficient evidence (grade I recommendation) to support one form of treatment over the other in the management of chronic ankle instability [38].

50.4.4 Arthroscopy

Reliable arthroscopic procedures for the treatment of CAI are required with the current trend of minimally invasive surgery. Among the lateral ankle ligaments, the ATFL is the only one which is continuous with the joint capsule and therefore is easily accessible during *arthroscopy* [1]. Various reports documented successful outcomes after arthroscopic treatment of CAI [39–45]. Oloff et al. [39] first reported on the use of thermal stabilization procedure in the ankle joint of 10 patients. At 9 months follow-up, AOFAS score improved from 58.3 at baseline to 88.1 at follow-up; anterior drawer and talar tilt stress tests were significantly reduced compared to preoperative status. Khan and Fanton [40] observed in 23 patients an improvement in the AOFAS score from 57.5 preoperatively to 86.5 at follow-up. Similarly, Maiotti et al. [43] reported on 22 patients 2.5–5 years out after *thermal ankle shrinkage* reporting 86 % good to excellent results. Radiological anterior drawer sign reduced by 55 % and talar tilt test by 80 %. Recently it has been reported that the arthroscopic resection of the borders of the torn ligament, together with the debridement of the adjacent area and the capsular thermal shrinkage followed by immobilization produced an effective reduction in capsular volume and allows enhanced joint stability [45].

Several studies advocate an arthroscopic investigation of the joint in cases of instability. Apart from treating laxity itself, arthroscopy allows better visualization and allows diagnosis and treatment of concomitant underlying joint diseases (*synovitis*, soft tissue injuries, osseous pathology, or degenerative arthritis), and the high incidence of concomitant intra-articular lesions makes arthroscopy essential regardless of the repair procedures [46–48].

The complication rate has been found to be 5.7–9 % with ankle arthroscopy, with neurologic injury being the most common occurrence [45, 49]. Damage to a superficial branch of the peroneal nerve or deep peroneal injury may occur [45]. To try to prevent neurologic injuries, it is important to carefully consider neural structures close to the ankle joint capsule.

Table 50.1 Summary of previous studies reported in literature on arthroscopic procedures for chronic ankle instability

Author	Year	Ankles	Mean age	Follow-up	AOFAS score	Karlsson score	Anterior drawer stress test	Talar tilt stress test
Oloff et al. [39]	2000	10	34 (19–53)	9.6 months (6–21)	Preop: 58.3 (SD: 8.96) Postop: 88.1 (SD: 11.09)	N/a	Preop: 8.4 mm (SD: 2.61) Postop: 3.6 mm (SD: 1.60)	Preop: 8.3° (SD: 3.81) Postop: 5.5° (SD: 2.78)
Khan et al. [40]	2000	23	38.5	1–2.5 years	Preop: 57.5 (47–71) Postop: 86.5 (70–100)	N/a	Preop: 8.0 mm Postop: 2.4 mm	Preop: 9.1° Postop: 6.7°
Berlet et al. [41]	2002	16	N/a	14.5 months (9–20)	Preop: 60.2 (36–84) Postop: 88.5 (66–100)	N/a	N/a	N/a
Hyer et al. [42]	2004	4	29 (20–32)	6 months	Preop: 26[a] (SD: 11.52) Postop: 51[a] (SD: 10.23)	N/a	N/a	N/a
Maiotti et al. [43]	2005	22	18 (16–24)	42 months (32–56)	N/a	Preop: 52.27 (SD: 5.28) Postop: 89.27 (SD: 9.21)	Preop: 6.8 mm (SD: 0.6) Postop: 3.1 mm (SD: 1.2)	Preop: 11.2° (SD: 1.0) Postop: 4.2° (SD: 1.7)
Kim et al. [52]	2011	28	38.6 (22–55)	15.9 months (13–25)	Preop: 60.78 (SD: 13.38) Postop: 92.48 (SD: 6.14)	N/a	Preop: 3.59 mm (SD: 0.68) Postop: 0.61 mm (SD: 0.75)	N/a
Ventura et al. [45]	2012	88	32.4 (17–56)	4.2 years (1.1–9)	Preop: 63.51 (SD: 8.18) Postop: 92.31 (SD: 6.93)	Preop: 61.81 (SD: 11.07) Postop: 88.44 (SD: 8.81)	N/a	N/a
Cottom and Rigby [28]	2014	40	45.6 (15–83)	12.1 months (6–21)	Preop: 41.2 (23–64) Postop: 95.4 (84–100)	Postop: 93.6 (82–100)	N/a	N/a

[a]Modified AOFAS score on 60 points

Arthroscopic repair of the anterior talofibular ligament using a suture anchor has been described previously with good results [50–54]. Acevedo and Mangone [53] reported on 24 ankles treated with an arthroscopic lateral ligament repair technique after an average follow-up of 10.9 months. Subjectively, all subjects reported improvement in ankle stability, and no patient underwent revision surgery. Li et al. [54] recommended the use of bone anchors in high-level athletes as this patient group has a significantly greater load and demand on their ankle joints than the average population. Thus, they advocated using suture anchors with the modified Broström technique, because this provides an anatomic, yet structurally strong, result, allowing athletes to return to their original sports level. However, their findings did not determine how much biomechanical advantage the anchors actually provide. Shahrulazua et al. [55] promoted the use of bioabsorbable anchors which avoid potential problems such as migration and secondary chondral damage and the potential need for implant removal; in addition the use of bone anchors represents an advantage since the torn ligament ends are often difficult to suture. Outcomes following arthroscopic treatment of chronic ankle instability are summarized in Table 50.1.

50.4.5 Combined Procedures

Some authors underscore the need of identifying precisely the amount of ligaments involved and to provide either anatomical repair or ligament reconstruction according to the quality of the ligament remnants. For this reason, hybrid techniques have been proposed.

Kennedy et al. combined *ATFL reconstruction* with a split peroneus longus tendon graft direct anatomical repair and plication of the CFL [56]. Similarly, Peterson et al. suggest in patients with significant ligamentous instability, increased BMI, or failed primary repair a combination of the Broström-Gould repair augmented with a free autogenous split peroneus longus tendon graft [57].

In addition, combined arthroscopic and open procedures have been proposed in order to improve the diagnosis and management of intra-articular lesions and allow minimally invasive reconstruction of the lateral ligament complex [58]. Nery et al. recently described a technique providing arthroscopic *debridement of the ATFL* adhesions followed by arthroscopic-assisted anchor fixation of the lateral capsular and ligament remnants over the anteroinferior aspect of the lateral malleolus. Results were satisfying in most patients at 10 years follow-up with a 5 % failure rate [58].

50.5 Rehabilitation and Return to Play

Rehabilitation protocols following surgical repair usually consist of brace immobilization in a neutral position with non-weight-bearing for 2–3 weeks. Then peroneal muscle strengthening, proprioceptive, and complete ankle range of motion (ROM) exercises are started. Immobilization with cast or brace and non-weight-bearing are important in order to prevent lengthening of the treated tissue following *arthroscopic repair* and to allow the healing and reconstitution process to occur unimpeded. Various reports suggest non-weight-bearing for 2–3 weeks [40–43, 45, 52]. de Vries et al. [44] propose the use of a compression bandage only for 3–5 days and early weight-bearing 5–7 days postoperatively. Articular stiffness may result from prolonged brace immobilization and delayed functional rehabilitation program, and there is evidence that after surgical reconstruction, early functional rehabilitation appears to be superior to 6 weeks immobilization in restoring early function [38]. *Return to sports* is usually permitted 3 months postoperatively. Kim et al. [52] suggest return to straight running and functional activities 8 weeks postoperatively, and cutting and sport-specific drills are allowed by week 12. However, patients should be instructed to avoid premature return to sport activity that could affect the outcome.

References

1. Taser F, Shafiq Q, Ebraheim NA (2006) Anatomy of lateral ankle ligaments and their relationship to bony landmarks. Surg Radiol Anat 28:391–397
2. Burks RT, Morgan J (1994) Anatomy of the lateral ankle ligaments. Am J Sports Med 22:72–77
3. Bonnel F, Toullec E, Mabit C, Tourné Y et al (2010) Chronic ankle instability: biomechanics and pathomechanics of ligament injury and associated lesions. Orthop Traumatol Surg Res 96:424–432
4. Williams GN, Jones MH, Amendola A (2007) Syndesmotic ankle sprains in athletes. Am J Sports Med 35:1197–1207
5. Hintermann B, Knupp M, Pagenstert GI (2006) Deltoid ligament injuries: diagnosis and management. Foot Ankle Clin 11:625–637
6. Garrick JG (1977) The frequency of injury, mechanism of injury, and epidemiology of ankle sprains. Am J Sports Med 6:241–242
7. Ferran NA, Maffulli N (2006) Epidemiology of sprains of the lateral ligamentous complex. Foot Ankle Clin North Am 11:531–537
8. Yeung MS, Chan KM, So CH, Yuan WY (1994) An epidemiological survey on ankle sprains. Br J Sports Med 28:112–116
9. Baumhauer JF, O'Brien T (2002) Surgical considerations in the treatment of ankle instability. J Athl Train 37:458–462
10. Hertel J (2002) Functional anatomy, pathomechanics, and pathophysiology of lateral ankle instability. J Athl Train 37:364–375
11. Mitchell A, Dyson R, Hale T, Abraham C (2008) Biomechanics of ankle instability. Part 1: reaction time to simulated ankle sprain. Med Sci Sports Exerc 40:1515–1521
12. Hopkins JT, Brown TN, Christensen L, Palmieri-Smith RM (2009) Deficits in peroneal latency and electromechanical delay in patients with functional ankle instability. J Orthop Res 27:1541–1546
13. Keefe DT, Haddad SL (2002) Subtalar instability. Etiology, diagnosis, and management. Foot Ankle Clin 7:577–609
14. Ventura A, Terzaghi C, Legnani C, Borgo E (2014) Lateral ligament reconstruction with allograft in patients with severe chronic ankle instability. Arch Orthop Trauma Surg 134:263–268
15. Fujii T, Luo ZP, Kitaoka HB, An KN (2000) The manual test may be not sufficient to differentiate ankle ligament injuries. Clin Biomech (Bristol, Avon) 15:619–623
16. Karlsson J, Eriksson BI, Renström PA (1997) Subtalar ankle instability. A review. Sports Med 24:337–346
17. Frost SCL, Amendola A (1999) Is stress radiography necessary in the diagnosis of acute or chronic ankle instability? Clin J Sports Med 9:40–45
18. Harper MC (1992) Stress radiographs in the diagnosis of lateral instability of the ankle and hindfoot. Foot Ankle 13:435–438
19. Rijke AM, Goitz HT, McCue FC, Dee PM (1993) Magnetic resonance imaging of injury to the lateral ankle ligaments. Am J Sports Med 21:528–534
20. Chinn L, Dicharry J, Hart JM, Saliba S, Wilder R, Hertel J (2014) Gait kinematics after taping in participants with chronic ankle instability. J Athl Train 49:322–330
21. Barlow G, Donovan L, Hart JM, Hertel J (2015) Effect of lace-up ankle braces on electromyography measures during walking in adults with chronic ankle instability. Phys Ther Sport 16(1):16–21. [Epub ahead of print]
22. Broström L (1966) Sprained ankles: VI. Surgical treatment of "chronic" ligament ruptures. Acta Chir Scand 132:551–565
23. Bell SJ, Mologne TS, Sitler DF, Cox JS (2006) Twenty-six-year results after Broström procedure for chronic lateral ankle instability. J Athl Train 34:975–978
24. Maffulli N, Del Buono A, Maffulli GD, Oliva F, Testa V, Capasoo G, Denaro V (2013) Isolated anterior talofibular ligament Broström repair for chronic lateral ankle instability. 9-year follow-up. Am J Sports Med 41:858–864
25. Gould N, Seligson D, Gassman J (1980) Early and late repair of lateral ligament of the ankle. Foot Ankle 1:84–89
26. Prisk VR, Imhauser CW, O'Loughlin PF, Kennedy JG (2010) Lateral ligament repair and reconstruction restore neither contact mechanics of the ankle joint nor motion patterns of the hindfoot. J Bone J Surg Am J Sports Med 92:2375–2386
27. Corte-Real NM, Moreira RM (2009) Arthroscopic repair of chronic lateral ankle instability. Foot Ankle Int 30:213–217
28. Cottom JM, Rigby RB (2013) The "all inside" arthroscopic Broström procedure: a prospective study of 40 consecutive patients. J Foot Ankle Surg 52:568–574
29. Ahn JH, Choy WS, Kim HY (2011) Reconstruction of the lateral ankle ligament with a long extensor tendon graft of the fourth toe. Am J Sports Med 30:637–644
30. Coughlin MJ, Matt V, Schenck RC Jr (2002) Augmented lateral ankle reconstruction using a free gracilis graft. Orthopedics 25:31–35
31. Evans DL (1953) Recurrent instability of the ankle; a method of surgical treatment. Proc R Soc Med 46:343–344
32. Greer Richardson E (2001) Chronic lateral ligament laxity: reconstruction by the Chrisman-Snook and Watson-Jones peroneus brevis transfers and the modified Broström procedure. Oper Tech Sports Med 9:26–31
33. Jung HG, Kim TH, Park JY, Bae EJ (2012) Anatomic reconstruction of the anterior talofibular and calcaneofibular ligaments using a semitendinosus tendon allograft and interference screws. Knee Surg Sports Traumatol Arthrosc 208:1432–1437
34. Ellis SJ, Williams BR, Pavlov H, Deland J (2011) Results of anatomic lateral ankle ligament reconstruction with tendon allograft. HSS J 7:134–140

35. Pagenstert GI, Hintermann B, Knupp M (2006) Operative management of chronic ankle instability: plantaris graft. Foot Ankle Clin 11:567–583
36. Paterson R, Cohen B, Taylor D, Bourne A, Black J (2000) Reconstruction of the lateral ligaments of the ankle using semitendinosis graft. Foot Ankle Int 21:413–419
37. Jones AP, Sidhom S, Sefton G (2007) A minimally invasive surgical technique for augmented reconstruction of the lateral ankle ligaments with woven polyester tape. J Foot Ankle Surg 46:416–423
38. deVries J, Krips R, Sierevelt I, Blankevoort L, van Dijk CN (2006) Interventions for treating chronic ankle instability. Cochrane Database Syst Rev (4):CD004124
39. Oloff LM, Bocko AP, Fanton G (2000) Arthroscopic monopolar radiofrequency thermal stabilization for chronic lateral ankle instability: a preliminary report on 10 cases. J Foot Ankle Surg 39:144–153
40. Khan A, Fanton G (2000) Use of thermal energy in the treatment of ankle disorders. Sports Med Arthroscopy Rev 8:354–364
41. Berlet GC, Saar WE, Ryan A, Lee TH (2002) Thermal-assisted capsular modification for functional ankle instability. Foot Ankle Clin 7(3):567–576
42. Hyer CF, Vancourt R (2004) Arthroscopic repair of lateral ankle instability by using the thermal-assisted capsular shift procedure: a review of 4 cases. J Foot Ankle Surg 43:104–109
43. Maiotti M, Massoni C, Tarantino U (2005) The use of arthroscopic thermal shrinkage to treat chronic lateral ankle instability in young athletes. Arthroscopy 21:751–757
44. de Vries JS, Krips R, Blankevoort L, Fievez AW, van Dijk CN (2008) Arthroscopic capsular shrinkage for chronic ankle instability with thermal radiofrequency: prospective multicenter trial. Orthopedics 31(7):655
45. Ventura A, Terzaghi C, Legnani C, Borgo E (2012) Arthroscopic four-step treatment for chronic ankle instability. Foot Ankle Int 33:29–36
46. Ventura A, Terzaghi C, Legnani C, Borgo E (2013) Treatment of osteochondral lesions of the talus: a four-step approach. Knee Surg Sports Traumatol Arthrosc 21:1245–1250. Epub ahead of print
47. Hintermann B, Boss A, Schäfer D (2002) Arthroscopic findings in patients with chronic ankle instability. Am J Sports Med 30:402–409
48. Komenda G, Ferkel R (1999) Arthroscopic findings associated with the unstable ankle. Foot Ankle Int 20:708–713
49. Ferkel RD, Small HN, Gittins JE (2001) Complications in foot and ankle arthroscopy. Clin Orthop Relat Res 391:89–104
50. Kashuk KB, Carbonell JA, Blum JA (1997) Arthroscopic stabilization of the ankle. Clin Podiatr Med Surg 14(3):459–478
51. Hawkins RB (1987) Arthroscopic stapling repair for chronic lateral instability. Clin Podiatr Med Surg 4:875–883
52. Kim ES, Lee KT, Park JS, Lee YK (2011) Arthroscopic anterior talofibular ligament repair for chronic ankle instability with a suture anchor technique. Orthopedics 34(4):273
53. Acevedo J, Mangone P (2011) Arthroscopic lateral ankle ligament reconstruction. Tech Foot Ankle Surg 10:111–116
54. Li X, Killie H, Guerrero P, Busconi BD (2009) Anatomical reconstruction for chronic lateral ankle instability in the high-demand athlete: functional outcomes after the modified Broström repair using suture anchors. Am J Sports Med 37:488–494
55. Shahrulazua A, Ariff Sukimin MS, Tengku Muzaffar TM, Yusof MI (2010) Early functional outcome of a modified Brostrom-Gould surgery using bioabsorbable suture anchor for chronic lateral ankle instability. Singapore Med J 51(3):235–241
56. Kennedy JG, Smyth NA, Fansa AA, Murawski CD (2012) Anatomic lateral ligament reconstruction in the ankle. A hybrid technique in the athletic population. Am J Sports Med 40:2309–2317
57. Peterson KS, Catanzariti AR, Mendicino MR, Mendicino RW (2013) Surgical approach for combined ankle and subtalar joint chronic mechanical instability. J Foot Ankle Surg 52(4):537–542. Epub ahead of print
58. Nery C, Raduan F, Del Buono A, Asaumi ID, Cohen M, Maffulli N (2011) Arthroscopic-assisted Broström-Gould for chronic ankle instability: a long-term follow-up. Am J Sports Med 39: 2381–2388

Ankle Impingement (Anterior and Posterior)

51

Francesco Lijoi

Abbreviation

AAI	Anterior ankle impingement
AITFL	Anterior inferior tibiofibular ligament
AT	Achilles tendon
FHL	Flexor hallucis longus
PAI	Posterior ankle impingement
PLP	Posterolateral portal
PMP	Posteromedial portal
PTFL	Posterior talofibular ligament

Ankle impingement is a "collision" between bony or soft tissues and the anterior or posterior aspect of the tibia and/or of the talus. It is one of the most common disorders among sports population though the real incidence of this pathology is not known. While anterior and posterior forms of impingement are similar regarding etiology (soft or bony impingement), they are different in many other aspects (location, relation to trauma or to overuse, clinical presentation). It is therefore necessary to distinguish two clinical forms of impingement, anterior and posterior, and to analyze etiology, injury mechanism, clinical and diagnostic examination, and treatment strategies separately.

51.1 Anterior Ankle Impingement (AAI)

51.1.1 Etiology and Classification

The first description was given by Morris in 1943 [1]. In 1950, McMurray [2] called this condition "the footballer's ankle" for the frequency of this anatomical lesion in football players. O'Donague [3] described this impingement as a result of sports injuries in athletes of many other sports. Over the time, other studies have described this pathology in volleyball players, ballet dancers, jumpers, and other athletes.

The anatomic feature of the lesions described by these authors was the presence of osteophytes on the anterior part of the tibia and/or of the talus; the bony spurs were essential for the diagnosis of the pathology. In time AAI has become a painful syndrome that can be caused by the presence of bony spurs or of just hypertrophic synovial tissue (commonly called bony or soft tissue impingement [4]).

A classification of AAI may be done on topographic bases. The anterior aspect of the ankle is divided into three areas: a central area, between the tibialis anterior tendon and the peroneus tertius tendon; the lateral area, lateral to the peroneus tertius tendon; and the medial area, medial to the tibialis anterior tendon. In the lateral area, soft tissues are frequently the cause of impingement, while the central and the anteromedial areas are usually sites of bony impingement.

Other attempts to classify the AAI are specifically related to the radiographic aspects of the joint. Scranton and McDermott classification [5] is developed in four degrees based on the morphology of spurs: Grade I and Grade II describe tibial spurs that are inferior or superior to 3 mm, respectively, Grade III presents osteophyte formation also on the neck of the talus with an aspect of "kissing osteophytes," and Grade IV describes osteoarthritic changes of the joint with secondary osteophytes.

Van Dijk et al.'s [6] classification connects the osteophytes to possible joint space narrowing. This classification has four degrees, where Grade 0 and Grade 1 represent normal joint without joint space narrowing (slight subchondral sclerosis in Grade 0 and presence of osteophytes in Grade 1), Grade 2 represents a joint space narrowing with or without osteophytes, while Grade 3 shows a total or subtotal disappearance or arthritic deformation of the joint. This is a classification related to the degree of arthritic changes of the joint, but it is of great interest for the treatment because the removal of osteoarthritic osteophytes has worse prognosis compared to the removal of osteophytes without joint space narrowing.

F. Lijoi
Department of Orthopaedic-Trauma,
"L.Pierantoni- G.B.Morgagni" Hospital,
Via Carlo Forlanini, 34, Forlì 47121, Italy
e-mail: f.lijoi@ausl.fo.it

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_51,

The cause of pain of an impingement syndrome seems to be caused not by the direct conflict of the bony spurs but by the inflamed synovia that is caught between the osteophytes. This hypothesis is supported by some evidence: (a) tibial and talar spurs usually do not overlap each other [7]; (b) histological synovial chronic inflammatory aspects can be found in the soft tissue in the proximity of the site of impingement [8]; and (c) anterolateral impingement is a painful pathology without the presence of bony osteophytes.

51.1.2 Injury Mechanism

Repeated ankle inversion sprains are typical in the clinical history of the patient. During the inversion tilt of the talus inside the tibiofibular mortise, soft tissue damages involving lateral ligaments are frequent on the lateral side of the joint, while bony impaction between the medial side of the talus and the anterior border of the medial malleolus can take place on the medial side. Medially the repetitive sprains may determine impaction-related microtrauma of the chondral margin of the talus and of the medial malleolus. Over the time an attempt of repair by the fibrocartilage and a secondary formation of calcification and hyperostosis can develop, leading to an *anteromedial impingement*.

On the lateral side, the tear of the ligaments and of the capsule can cause a hypertrophy and a hyalinization of the synovia with possible production of chronic inflamed tissue in the anterior aspect of the lateral gutter [9]. This tissue can be entrapped during repetitive movements in dorsal flexion and plantar flexion producing swelling and pain, the classical symptoms of the anterolateral impingement. This pathological fibrous tissue can be usually seen in the front of the talus, fibula, and anterolateral tibia (*anterolateral impingement*), but sometimes it overlays the entire interosseus ligament of the syndesmosis inside the joint.

In other patients affected by an anterolateral impingement, a hard, fibrotic, meniscus-like band can be seen in the anterior lateral gutter, stretched between the lateral tibia and the anterior edge of the lateral malleolus. This arthroscopic finding has been defined as "meniscoid lesion" and is usually accepted as a posttraumatic hyaline tissue. The fibrotic band is actually an anatomical structure, the so-called Bassett ligament, a thickened distal fascicle of the anterior inferior tibiofibular ligament (AITFL) [10] (Fig. 51.1). This structure does not seem to be an accessory fascicle but a constant part of the AITFL and can impinge on the lateral edge of the talus for the following reasons [11]: (a) an increased anterolateral laxity with increased anterior extrusion of the talus and (b) a more distal insertion of the fascicle on the fibula, with a more vertical location and a possible greater contact of the fibrous band on the talus. Abrasion between the fascicle and the talus can lead to pain, and at arthroscopic inspection, the articular cartilage is generally of poor quality in the area of the fascicle contact [12].

In the "classic" AAI, anterior spurs were supposed to be traction osteophytes of the anterior capsule due to repetitive plantar flexion movements of the ankle during kicking [2]. The anterior joint capsule attaches actually more proximally to the tibia and more distally to the talus compared to the location of the spurs, and arthroscopy of the ankle allows to clearly inspect and to remove them without opening the capsule. It has been demonstrated through an experimental study [13] that in football players, the impaction-related microtrauma due to the repetitive ball impacts on the anterior aspect of the ankle can lead to the formation of hyperostosis in the distal tibia and in the talus through the cascade of

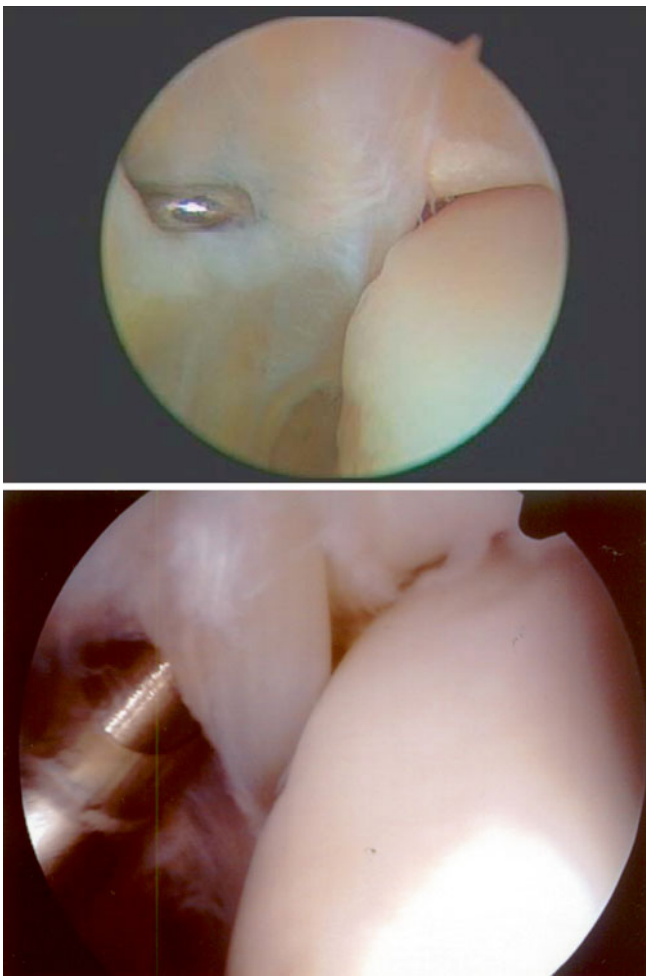


Fig. 51.1 The "Bassett ligament," also named "meniscoid lesion," is a type of anterolateral fibrous impingement. It is caused by an accessory fascicle of AITFL that has sometimes a vertical direction for a more distal insertion on the fibula

events described above for the pathogenesis of anteromedial impingement (fibrocartilage and secondary formation of calcification and hyperostosis).

51.1.3 Clinical and Diagnostic Examination

The symptoms of an AAI are usually a chronic anterior ankle pain and some kind of swelling after sports activity with a history of recurrent ankle sprains. Many times a slightly limited dorsal flexion can be detected. Anterolateral, anterior, or anteromedial impingement can be distinguished according to the site of the pain during activities and on palpation during physical examination.

The radiographic examination is essential for the diagnosis of the bony impingement. In the classic anterior impingement (the "footballer's ankle"), a lateral view of the ankle can detect the presence of hyperostosis of the anterior profile of the distal tibia and/or of the anterior border of the talar dome: the spurs can pinch the synovia during dorsiflexion and provoke pain and swelling (Fig. 51.2).

In anteromedial impingement, the presence of hyperostosis on the anterior aspect of the medial malleolus and on the medial border of the talus can be undetected on standard radiographs because of the prominence of the anterior border of the distal tibia and of the dorsal profile of the neck of the talus. Van Dijk et al. [14] described an anteromedial radiographic oblique view (described in Fig. 51.3) that can detect the anteromedial hyperostosis and allow the correct diagnosis.

Anterolateral impingement cannot be detected with radiographs as it is a soft tissue impingement. The diagnosis is done on clinical data: in a recent paper [15], the efficacy of conventional MRI in detecting soft tissue abnormalities in anterolateral gutter remains controversial, with a wide range of sensitivities (39–100 %) and specificities (50–100 %).

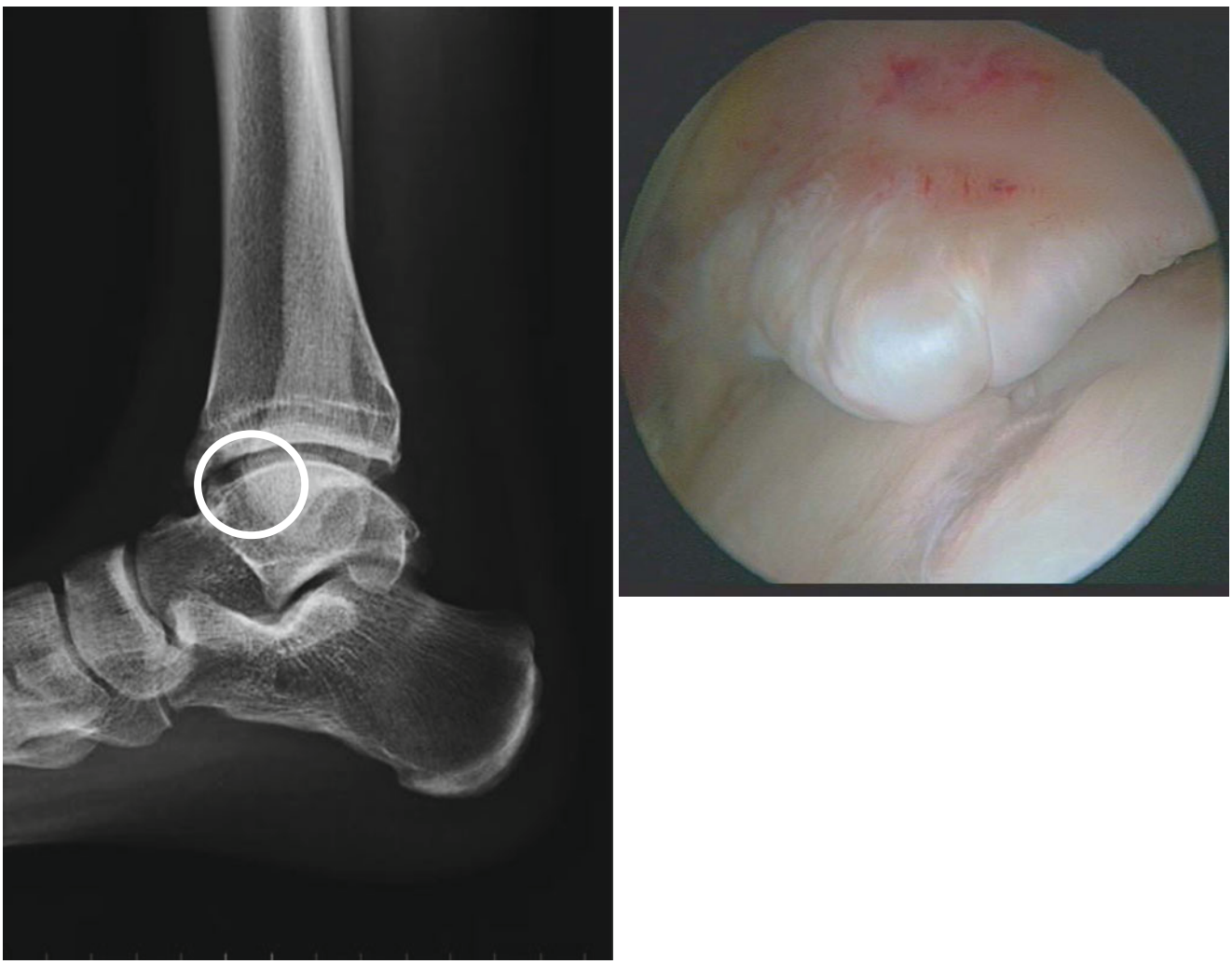


Fig. 51.2 "Classical" anterior ankle impingement. Presence of an anterior tibial spur seen on a radiographic lateral view and during an anterior arthroscopy ("the footballer's ankle")

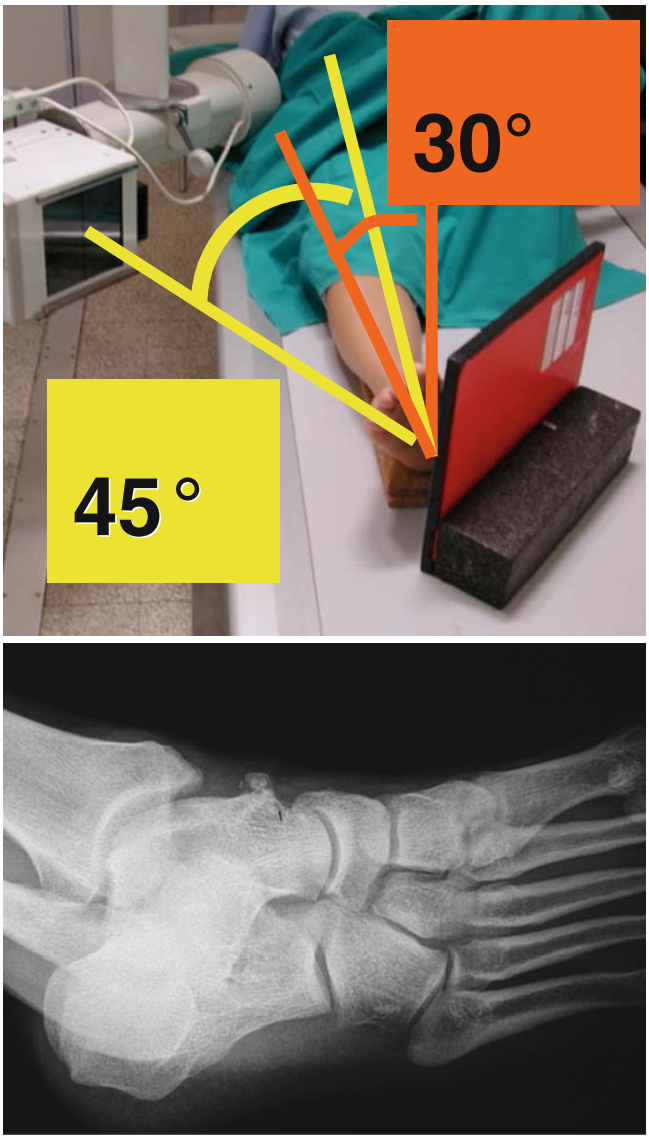


Fig. 51.3 The radiographic oblique view to detect an anteromedial bony impingement. The leg is positioned in 30° of external rotation; the radiographic beam is 45° oblique on the longitudinal axis of the leg, perpendicular to the radiographic film, centered on the ankle

51.1.4 Treatment Strategy

Pain in anterior ankle impingement seems to be due to the entrapment of the synovial tissue during the movements of the ankle, mainly in dorsal flexion. The presence of hyperostosis can reduce the anterior articular space and may facilitate this pathologic mechanism. Arthroscopy is theoretically the most useful tool to remove bony spurs and the synovial pathological tissue having fewer possibilities to create hypertrophic scars that might be painful again.

Anterior arthroscopy is not a trivial kind of surgery: accuracy is essential in positioning the patient, in performing the sites of entry portals, and in exchanging the arthroscope and instruments for the treatment of different types of impingement. Without dealing with the general technique of anterior ankle arthroscopy, we would like to emphasize some specific tips to consider in the treatment of an AAI.

Position of the patient: the patient must be supine, with the foot on the border of the table in order to be able to perform passively dorsal or plantar flexion during surgery. The foot must be exactly vertical on the plane of the table, and the operated leg must be slightly elevated compared to the other one to allow comfortable maneuvers of the instruments.

Sites of the portals: anteromedial portal is performed near the medial border of the tibialis tendon with the ankle in full dorsal flexion. A more medial site may create difficulties in viewing the lateral malleolus in case of lateral ossicles or peroneal hyperostosis for the convexity of the talar dome. The lateral portal is identified by the scope with a needle, on the lateral edge of the peroneus tertius muscle, avoiding the superficial peroneal nerve by dissecting the subcutaneous layer with a mosquito clamp. Also this portal must be performed as "central" as possible to allow a comfortable position of the instruments to work on the medial malleolus if it were required. If an anterolateral soft tissue impingement is suspected, it is recommended to locate this portal exactly at the joint level; to address a pathology of the medial malleolus, it is more useful to locate the anterolateral portal slightly more proximal in order to view more easily the malleolus [16].

Scope and instruments: after a routine exploration of the joint and the confirmation of the type of pathology, the portals must be used according to the pathology to treat. In anteromedial impingement, medial ossicles, hyperostosis of medial malleolus and of the neck of the talus, the medial portal is the working portal while the arthroscope is shifted in the anterolateral portal. In lateral pathology (anterolateral soft tissue impingement, removal of peroneal ossicles or osteophytes), instruments must be kept in the lateral portal. To remove an anterior bony impingement (tibial and/or talar), scope and instruments can be interchanged to address the spurs conveniently. It is not always clear how much bone must be resected to treat the bony impingement: tibial hyperostosis must be identified proximally to fully define the anatomic shape of the anterior tibia. In this way the proximal limit of the bony spur can be clearly addressed with a burr, performing the debridement in a proximal-distal direction. For the talar spurs, the distal base of implant of the spur must be identified to make a distal-proximal resection.

51.2 Posterior Ankle Impingement

51.2.1 Etiology and Classification

Posterior ankle impingement (PAI) is a painful syndrome in the posterior aspect of the ankle, and it is a mechanical conflict that arises in forced plantar flexion.

The talus extends posteriorly with a bony process that can cross laterally the midline in case of hypertrophy: it is called the posterolateral process, and in about 7 % of the adult population, it appears as a distinct accessory bone for a failed union of this ossification center [17]. This center is normally separated from the ossification center of the body of the talus until 8–10 years of life. In adults this distinct ossicle, if present, is called os trigonum, and it is usually linked to the talus through a fibrous synchondrosis. Medially to the posterolateral process, there is another smaller bony prominence of the talus, the medial tubercle: between these two bony prominences, there is a sulcus that is transformed in an osteofibrotic channel by a fibrous retinaculum that takes insertion on the tip of the two tubercles. The flexor hallucis longus (FHL), the most posterior of the posteromedial tendons, slides in this channel. At this level the channel acts as a pulley where the FHL changes its direction of sliding, from vertical to nearly horizontal: this is a point of weakness of the tendon, and pathologies that can involve the posterolateral talar process (or the os trigonum, if present) can also involve the tendon itself and cause a FHL tendinitis. The posterolateral process also gives insertion to the posterior talofibular ligament (PTFL), the strong and most posterior ligament of the lateral ligamentous complex of the ankle. This ligament is lax in plantar flexion and taut in maximal dorsal flexion, and for this reason, it can detach the process (or the os trigonum) from the talus during forced and sudden movements.

51.2.2 Injury Mechanism

PAI can be caused by trauma or overuse, and it has been typically associated with ballet dancers for the frequent "en pointe" or "demi-pointe" position assumed by these athletes. However, any sport that requires or any trauma that provokes forced plantar flexion or more rarely forced dorsal flexion can determine this syndrome. Running (especially downhill running), soccer, basketball, volleyball, and jumping (high, long, and triple jump) are the sports more frequently involved in this kind of pathology. Rarely the detachment of the posterior bony process can be caused by a sudden traction of the PTFL during a trauma in forced dorsal flexion. More frequently a forced plantar flexion of the ankle, sometimes in association with supination or pronation of the hindfoot, provokes a crush of the os trigonum or of the posterolateral process between the posteroinferior edge of the tibia and the calcaneus. The mechanism of this crush has been compared to a nut (the os trigonum) in a nutcracker (tibia and calcaneus). This kind of trauma can be acute and can determine the fracture of the posterior talar process or the sudden mobilization of the os trigonum. Other times the trauma can be repetitive during a long period of time and can cause an overuse syndrome. This different etiology is not academic, but it is of practical interest because of better prognosis of PAI in overuse syndromes compared to traumatic cases [18]. The presence of the os trigonum or of a hypertrophic posterolateral process is neither sufficient nor essential to determine this syndrome. If the bony process or the ossicle is present, they are not sufficient to determine the pain syndrome: sometimes the pain is caused only by the traumatic event or by efforts beyond the anatomical limit. In other patients the painful syndrome is caused by a soft tissue impingement: a thickened joint capsule, a rupture of the PTFL, and a bump of hypertrophic tissue may cause a PAI syndrome without bony prominence.

51.2.3 Clinical and Diagnostic Examination

PAI may be suspected in a patient who complains of posterior ankle pain and who plays sports that cause pronounced plantar flexion or have caused a sudden trauma in hyper dorsal flexion or hyper-plantar flexion of the ankle.

The typical clinical objective signs are:

- *Painful deep palpation* of the posterolateral area between the lateral malleolus and the lateral border of Achilles tendon at the height of the ankle: the patient recognizes this type of pain as his typical pain. Pain on palpation of the posteromedial area is less diagnostic because it may be due to the pathology of the neurovascular bundle or to the flexor tendons.
- Positivity of the passive *forced plantar flexion test*: quick, repetitive, passive hyperflexion movements in neutral rotation or sometimes in external or internal rotation of the foot relative to the tibia determine a sudden pain in the posterior aspect of the ankle.
- The passive forced plantar flexion test becomes negative after an infiltration of 4–5 cc of anesthetic performed from the posterolateral side to the posterior joint capsule (*positive xilo-test*).

Imaging may be diagnostic in the presence of bony impingement: AP view is generally normal, while the lateral view may show the presence of an os trigonum, a hypertrophic posterior talar process, a fracture, or a nonunion of it in case of trauma (Fig. 51.4). Sometimes the bony protrusion may be "hidden" because of the superimposition on the body of the talus. This generally occurs for the tendency of the posterior talar process to cross laterally the midline; a lateral view with the ankle in about 25° of external rotation can discover the bony spur. In difficult diagnosis or in posttraumatic cases, a CT scan can be useful to localize and correctly diagnose the type of the lesion (fracture, nonunion, os trigonum). In soft tissue PAI, the diagnosis is only clinical and arthroscopy may be the only tool to confirm the pathology.

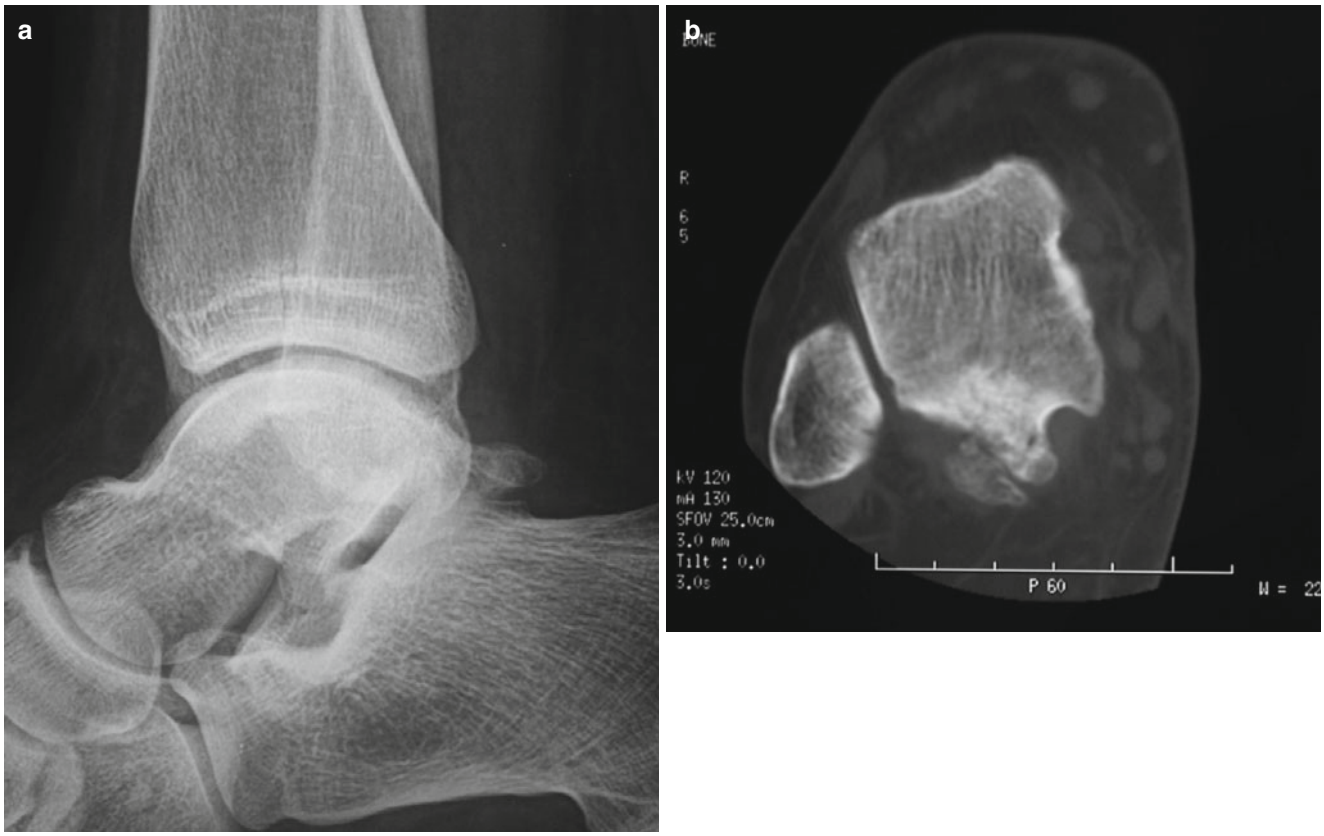


Fig. 51.4 (**a**) Lateral view of a posterior ankle impingement: presence of an os trigonum. (**b**) TC view showing the posterior process (*square*), the medial process (*star*), and the os trigonum (*circle*)

51.2.4 Treatment Strategy

Until the 2000s, operative treatment of PAI was performed by open surgery. Arthroscopy of the posterior aspect of the ankle was considered dangerous due to the presence of the posteromedial neurovascular bundle and the high possibility to create lesions during the introduction of instruments through the posteromedial portal. Van Dijk et al. [19] described a novel arthroscopic technique to address posterior ankle pathology, and Lijoi and al. [20] confirmed the safety and reproducibility of the technique by anatomical studies. Arthroscopic technique offers obvious advantages over open technique due to the precision of the surgical act, absence of immobilization, reduction of the time of recovery, and fewer risks of infections and of painful scars [21].

Posterior ankle arthroscopy is a demanding technique that requires arthroscopic skill and good knowledge of posterior ankle anatomy. This technique has been described by the authors [16, 19], and we would like to underline only some specific aspects of the technique.

Position of the patient: the patient is in prone position, with the foot outstanding the edge of the table and perpendicular to the floor. The ankle must be free to be dorsiflexed or plantar flexed.

Sites of the portals: they are two, the posterolateral portal (PLP) and the posteromedial portal (PMP), on the lateral edge and on the medial edge of the profile of AT, respectively. The PLP is the reference for the height of the portals: PLP is drawn in the angle delimited from the line of the lateral profile of the AT and the line drawn parallel to the lateral border of the foot, with the ankle at 90°, tangential to the tip of the lateral malleolus. At the same height, on the medial edge of the AT, the PMP is drawn without any reference with the medial malleolus.

Scope and instruments: after the incision only of the skin, a blunt dissection of the underling tissues is carried out with a mosquito clamp. The scope is introduced trough the PLP toward the posterior aspect of the talus, in a direction toward the second ray of the forefoot, touching the bone. Through the PMP the mosquito clamp is introduced horizontally compared to the axis of the foot. It bluntly dissects the tissue in front of the AT, touching the shaft of the scope on the lateral side of the AT, and then it slides on the shaft to the posterior aspect of the ankle (Fig. 51.5). In the same way, every instrument must be introduced. Van Dijk's first streategy is to go around the neurovascular bundle. The second strategy is to visualize the FHL and to work always lateral to it with the instruments: this tendon is always posterior and lateral to the

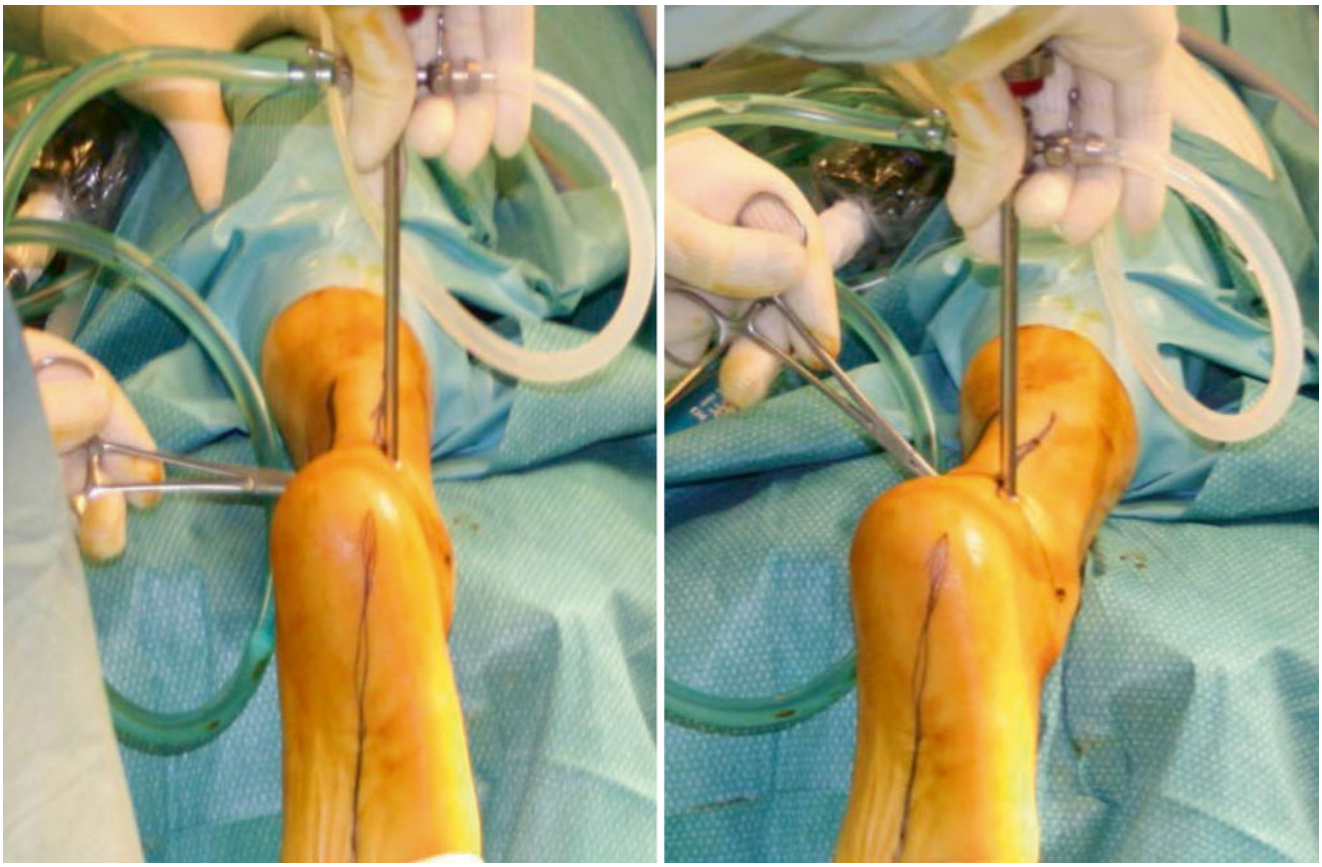


Fig. 51.5 The arthroscope and the instruments during a posterior arthroscopy of a right ankle. The trocar of the arthroscope is introduced through the posterolateral portal following the imaginary direction toward the second ray of the forefoot. A mosquito clamp is introduced in the posteromedial portal in a horizontal direction. It bluntly dissects the tissues in front of the Achilles tendon, until it touches the shaft of the trocar on the lateral side of the tendon, and then it slides on it to the posterior area of the ankle

tibialis posterior nerve, the structure of the bundle more close to the instruments [20]. In this way it is possible to work without any risk of dangers on the bundle.

51.2.5 Rehabilitation and Return to Play

After anterior or posterior ankle arthroscopy for the treatment of an impingement, we do not usually immobilize the ankle: only a compressive bandage is applied for 5–6 days, and the patient is invited to start active and passive mobilization to regain the maximum range of motion. The patient is instructed to take especially care of exercises in dorsal flexion of the ankle: this is the arch of movement that is limited before surgery in AAI, while it can be restricted for post-op scars after a posterior ankle arthroscopy. Weight bearing on crutches for 6–8 days is allowed if no chondral major treatment has been performed. After the first 10–15 days, rehabilitation is advised possibly with the use of a swimming pool, especially in the athletes: exercises are progressively increased without patient discomfort, until reaching normal range of motion and muscle strength. The last steps are recovery of good proprioception, correction of incorrect sports gestures, and sport-specific training.

The prognosis of anterior ankle impingement relates to some factors. The degree of osteoarthritis is the first prognostic factor to be taken into account, especially in anterior bony impingement: grade 0 and grade 1 according to Van Dijk's classification have good to excellent results in 83 % at long follow-up (100 % in grade 0), while good to excellent results were only 53 % in grade 2 (space narrowing) [22]. The presence of syndesmotic lesions and cartilage lesions and recurrence of inversion traumas are also bad prognostic factors in all other types of anterior impingement [23, 24]. On the other side, size and location of the hyperostosis are not related to the outcome and pain score [25].

In posterior ankle impingement, the overuse impingement has better scores at follow-up; patients' satisfaction is higher, and the return to work and sports activities results quicker than in posttraumatic cases [26].

References

1. Morris LH (1943) Report of cases of athlete's ankle. J Bone Joint Surg 25A:220
2. McMurray TP (1950) Footballer's ankle. J Bone Joint Surg 32:68–69
3. O'Donogue DH (1957) Impingement exostoses of the talus and tibia. J Bone Joint Surg 39A:835–852
4. Ferkel RD, Fasulo GJ (1994) Arthroscopic treatment of ankle injuries. Orthop Clin North Am 25:17–32
5. Scranton PE, McDermott JE (1992) Anterior tibiotalar spurs: a comparison of open versus arthroscopic debridement. Foot Ankle 13:125–129
6. van Dijk CN et al (1997) A prospective study of prognostic factors concerning the outcome of arthroscopic surgery for anterior ankle impingement. Am J Sports Med 25(6):737–745
7. Berberian WS et al (2001) Morphology of tibiotalar osteophytes in anterior impingement. Foot Ankle Int 22:313–317
8. Ferkel RD et al (1991) Arthroscopic treatment of anterolateral impingement of the ankle. Am J Sports Med 19:440–446
9. Wolin I (1950) Internal derangement of the talofibular component of the ankle. Surg Gynecol Obstet 91(2):193–200
10. Bassett FH III, Gates HS III, Billys JB et al (1990) Talar impingement by the anteroinferior tibiofibular ligament. A cause of chronic pain in the ankle after inversion sprain. J Bone Joint Surg Am 72(1):55–59
11. Golanò P et al (2010) Anatomy of the ankle ligaments: a pictorial essay. Knee Surg Sports Traumatol Arthrosc 18(5):557–569
12. Akseki D et al (2002) The anterior inferior tibiofibular ligament and talar impingement: a cadaveric study. Knee Surg Sports Traumatol Arthrosc 10:321–326
13. Tol JL et al (2002) The relationship of the kicking action in soccer and anterior ankle impingement syndrome. A biomechanical analysis. Am J Sports Med 30(1):45–50
14. van Dijk CN, Wessel RN, Tol JL, Maas M (2002) Oblique radiograph for the detection of bone spurs in anterior ankle impingement. Skeletal Radiol 31(4):214–221
15. Donovan A, Rosemberg ZA (2010) MRI of ankle and lateral hindfoot impingement syndromes. Am J Radiology 195:595–604
16. Van Dijk (2014) Ankle arthroscopy Springer, Berlin
17. Quirk R (1994) Common foot and ankle injuries in dance. Orthop Clin North Am 25:123–133
18. Stibbe AB et al (1994) The os trigonum syndrome. Acta Orthop Scand Suppl 262:59–60
19. Van Dijk CN et al (2000) A 2-portal endoscopic approach for diagnosis and treatment of posterior ankle pathology. Arthroscopy 16:871–876
20. Lijoi F, Lughi M, Baccarani G (2003) Posterior arthroscopic approach to the ankle: an anatomic study. Arthroscopy 19:62–67
21. van Dijk CN (2006) Hindfoot endoscopy. Foot Ankle Clin N Am 11:391–414
22. Tol JL et al (2001) Arthroscopic treatment of anterior impingement in the ankle. J Bone J Surg Br 83:9–13
23. Moustafa El-Sayed AM (2010) Arthroscopic treatment of anterolateral impingement of the ankle. J Foot Ankle Surg 49:219–223
24. Urguden M et al (2005) Arthroscopic treatment of anterolateral soft tissue impingement of the ankle: evaluation of factors affecting outcome. Arthoscopy 21(3):317–322
25. Moon JS et al (2010) Cartilage lesion in anterior bony impingement of the ankle. Arthroscopy 26(7):984–989
26. Sholten P et al (2008) Hindfoot endoscopy for posterior ankle impingement. J Bone J Surg Am 90(12):2665–2672

Ankle Osteochondral Lesions

52

Sandro Giannini, Roberto Emanuele Buda, Marco Cavallo, Pagliazzi Gherardo, Castagnini Francesco, and Francesca Vannini

52.1 Definition

Osteochondral lesion (OCL) of the ankle joint is an injury involving the chondral layer and, secondarily, the subchondral bone. It is usually localized on the talar dome, less frequently on the tibial plafond [1, 2].

52.1.1 Etiology

Ankle OCLs are usually traumatic in origin, mostly subsequent to ankle sprains or repetitive microtraumas [1]. Chondral lesions are present in 50 % of the acute ankle sprains and may be traced in 23 % of the lateral chronic instability of the ankle, causing persisting pain even after ligament reconstruction [3, 4]. Regarding the location, lateral lesions recognize a traumatic etiology in 93–98 % of the cases, whereas medial defects reported an ankle injury in only 60–71 % [1]. In a recent work by Orr, OCLs were centro-lateral (49 %) or centro-medial (33 %): specifically, the centro-lateral lesions were sent to surgery more frequently than the medial ones, which, nevertheless, tended to be larger [5]. The nontraumatic etiology can be confirmed for a small amount of cases, which have been addressed to various (weak) hypotheses as, for example, embolic, hereditary, endocrine, and idiopathic [6, 7].

S. Giannini • R.E. Buda
Orthopaedics and Traumatology,
University of Bologna,
Bologna, Italy

I Clinic, Rizzoli Orthopaedic Institute, Bologna, Italy
e-mail: giannini@ior.it; roberto.buda@ior.it

M. Cavallo (✉) • P. Gherardo • C. Francesco • F. Vannini
I Clinic, Rizzoli Orthopaedic Institute, Bologna, Italy
e-mail: marco.cavallo@ior.it; gpagliazzi@libero.it;
francescocastagnini@hotmail.it; france_vannini@yahoo.it

52.1.2 Injury Mechanism and Natural History

Traumas causing lateral OCLs are ankle inversion or inversion-dorsiflexion [1, 8]. As described by Berndt-Hardy, the forced inversion causes the talar dome to impact on fibular surface, damaging the articular surface through a shearing force. In this case, the lesion looks superficial and oval [1, 8]. Medial OCL is less correlated to traumas; nevertheless, a combined torsional impaction and axial loading (plantar flexion, anterior displacement, and internal rotation of the talus on tibia during inversion) is the advocated preponderant etiology. It usually appears deeper than the lateral one and is described as cup shaped [8]. The most affected areas are the centro-medial and centro-lateral, with the last localization seriously injured by the rotational forces. So, the centro-lateral lesions are more prone to a surgical treatment, despite the lower surface involved [5].

The OCL may be limited to the chondral tissue, or it may involve the subchondral bone or, after intense traumas, may even isolate a loose body [1]. From a histological perspective, after the impact, the chondral layer is found to be softened, with a significant chondrocyte apoptosis and matrix degeneration. The hyaline cartilage is progressively replaced by fibrocartilage during the healing process. The subchondral bone is strongly reshaped by an increased osteoclastic activity, with an ultimate bone stock loss [9]. The presence of bone bruise is a significant prognostic factor of chondral damage, causing cartilage irregularities, chondrocyte apoptosis, and matrix degeneration [10]. Classically, OCL may evolve to osteoarthritis and, when symptomatic and large defects are found, should be addressed to surgery in order to avoid progression [6–8]. The work by Guettler highlighted that not only OCL provides a local osteochondral disruption but alters the biomechanics of the surrounding cartilage as well, predisposing to arthritis [11]. In a work by Choi, in line with the classical theory of Berndt-Hardy, a critical size defect was traced at 150 mm^2 OCL, with good healing for defects with lower area [4, 8].

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_52, © Springer International Publishing Switzerland 2016

52.2 Clinical and Diagnostic Examination

Acute OCLs are frequently reported by young patients following a major trauma or ankle sprain during sport activity [1, 7, 12]. Swelling, pain, and symptoms related to lateral ligament lesions may be present. Locking, or catching, is associated with displaced fragments. Pain and limited range of motion (ROM) usually persist over 4–6 weeks after the acute event [1, 8, 12].

Chronic OCL most frequent symptom is a mild, continuous pain, mostly associated with physical activity [1, 8, 12]. Asymptomatic cases are not uncommon. Walking on uneven ground may increase the symptoms. Swelling, stiffness, weakness, and reduced ROM may be present, mostly in degenerated OCL. Patients may complain for the inability to load on the joint and, in case of loose bodies, for catching, locking, or clicking [1, 8, 12].

Palpation often evokes tenderness on posterior-medial or anterior-lateral areas of the talus [1, 8, 12]. The range of motion may be limited in half of the cases. Limpness, or antalgic gait, is relatively common. Anterior drawer and talar tilt test should be performed as sprains usually underlie OCL. Other tendon, vascular, and neurological pathologies should be ruled out (Table 52.1).

Routine X-ray is the first-line diagnostic tool also in order to rule out a fracture in acute cases [13]. Nevertheless, apart large lesions, OCL can easily be undiagnosed [14].

CT is valuable for the detection of subchondral bone injuries: it may clearly detect the size, shape, and extent of the localization (Fig. 52.1) [15].

MRI is the gold standard for OCL diagnosis, providing information about bone bruise, cartilage status, and soft tissues [15]. The sensitivity of MRI is high when correlated to arthroscopic findings (81–83 % or even higher) [16]. The most frequent features compatible with OCL are decreased signal intensity on T1-weighted images and increased intensity on T2-weighted images. In case of incomplete separation of the fragment, T2-weighted images may be confusing due to a high signal, with lower percentage of correlation with arthroscopic findings (55.6 %). In this case, the cartilage break discriminates (Fig. 52.2) [16].

Table 52.1 OCL classification according to Giannini divides the lesions into acute and chronic ones

Acute			
	Surface	**Extension**	**Treatment**
Type I	Damaged	<1 cm^2	Debridement
Type II	Damaged	>1 cm^2	Fixation
Chronic			
	Surface	**Extension**	**Treatment**
Type 0	Intact	Any	Retrograde Drilling
Type I	Damaged	<1.5 cm^2	Microfractures
Type II	Damaged	>1.5 cm^2	Cartilage Replacement
Type IIA	Damaged	>1.5 cm^2 >5 mm	CR+Bone Graft
Type III	Damaged	Anatomy disruption	Massive Graft

The area and the depth of the OCL are taken into account. For every kind of lesion, the most suitable treatment is suggested

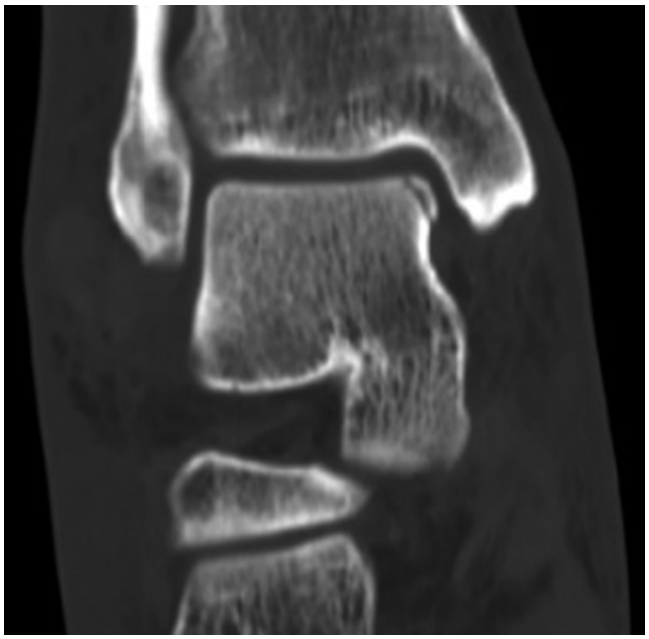


Fig. 52.1 Coronal view of a CT scan performed for OCL preoperative evaluation. CT scan is very useful to improve subchondral bone visualization

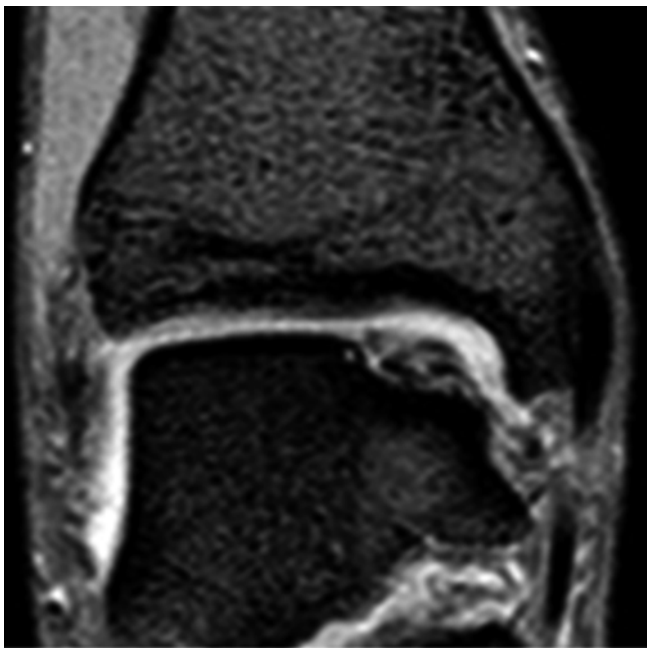


Fig. 52.2 Coronal view of MRI scan performed for chronic medial OCL. MRI is the best diagnostic tool, as it can visualize the cartilage and the subchondral bone as well as other soft tissue as ligaments

52.3 Treatment Strategy

No widely shared guidelines exist for OCL treatment [14, 17, 18]. A valid classification, focused on arthroscopic/MRI findings and corresponding treatments, considering the area and the depth of the lesions as well, was made by Giannini [19].

52.3.1 Acute Lesions

Conservative treatment is not successful in acute lesions, requiring arthroscopic procedures. Debridement and fragment excision are advised in case of acute lesions with fragment's dimensions inferior to 1 cm [19, 20]. Fragment fixation is performed in case of larger OCL using bioresorbable screws: good long-term results are achieved thanks to an effective vascular restoration [21]. Excision for larger fragment can dramatically raise osteoarthritis rates at long-term follow-up [20]. Recently osteochondral autografts have been adopted, with good results even in acute lesions [21].

52.3.2 Chronic Lesions

52.3.2.1 Conservative Treatment

The aim of conservative treatment is unloading the osteochondral layer, preventing the necrosis, and resolving the bone edema. To date, it should be reserved to small lesions with no fragment isolation in almost-asymptomatic patients [14, 17, 22]. In these cases, 45 % of the patients may benefit from a conservative approach. A possible beneficial approach in athletes may consist in rest, with sport activity restriction, and even a limited period of non-weight-bearing, lasting only a few weeks, according to the gravity of the lesion. In a work by Mei-Dan, hyaluronate and platelet-rich plasma (PRP) were injected intra-articularly in OCL, improving the clinical outcomes, with long-lasting results for PRP (at least 6 months) [23]. Intra-articular injections may be functional in athletes to delay the surgical treatment even in symptomatic lesions with no fragment isolation.

52.3.2.2 Retrograde Drilling

Retrograde drilling is mostly effective in lesions 0 according to Giannini's classification, with modest subchondral bone involvement and chondral layer continuity and viability [19]. The rationale consists in a stimulation of the repair depending on subchondral bone marrow cells [19, 24, 25]. The approach is made through sinus tarsi, drilling the subchondral bone without damaging the articular surfaces. An autologous calcaneal bone graft is then performed. Retrograde drilling may avoid the necrotic effect of the anterograde approach, preserving the chondral tissue [25]. Good results were reached in case of viable cartilage; nevertheless, it has been applied even in revision surgery [25].

52.3.2.3 Microfractures

Widely diffused microfractures are effective in OCL inferior to 1.5 cm^2 [6, 7, 19, 26]. The technique can be easily performed arthroscopically, penetrating the subchondral bone every 3–4 mm, using an awl [26]. Thanks to bone marrow stimulation, this procedure allows a good and rapid restoration of the osteochondral layer, but it generates fibrocartilage, with lower biomechanical properties and durability [14, 24]. Good clinical outcomes were reported by many authors, but medial lesions and larger and deep OCL tended to worsen over the time [26, 27].

52.3.2.4 Mosaicplasty

Osteochondral plugs, obtained from non-weight-bearing areas of the knee and, possibly, ankle, are implanted to restore the proper osteochondral layer [22, 24, 28]. This procedure often requires a malleolar osteotomy to improve the exposure. In the report by Hangody [22], the best OCL to treat is defined as approximately 10 mm large, positioned on the medial or lateral dome (not the central part of the talus), in a non-arthritic ankle. Clinical and bioptic results were promising, achieving remarkable outcomes even in athletes (63 % of the patients came back to sport activity at the same level, only 9 % gave up sport), with a slight deterioration over time, at around 10 years from the surgical procedure [28]. Nevertheless, mosaicplasty faces some drawbacks. First, it is a technical demanding technique, which includes a donor-site morbidity. Between osteochondral plugs, fibrocartilage is frequently found; moreover, not all lesions are successfully treated due to challenging locations (Fig. 52.3) [22, 24, 28].

52.3.2.5 Autologous Chondrocyte Implantation

Autologous chondrocyte implantation (ACI) has been intensively applied for OCL of the ankle, with successful clinical outcomes (90 %) [18]. Although no clear superiority has been established, ACI is considered the gold standard in regenerative procedures [18, 19]. The first-generation procedure was technically demanding, requiring an open-field approach, a malleolar osteotomy, and a periosteal flap suture [18, 29]. The second-generation procedure was performed arthroscopically, thanks to the development of specific instrumentation and scaffold [29]. Arthroscopic technique is performed with a two-step approach, with a first arthroscopy to debride the lesion and harvest the autologous chondrocytes doomed to expansion. A source of viable chondrocytes is the osteochondral fragment, the area around the OCL, or even a non-weight-bearing area of the knee [29, 30]. After the first arthroscopy, the chondrocytes are expanded in

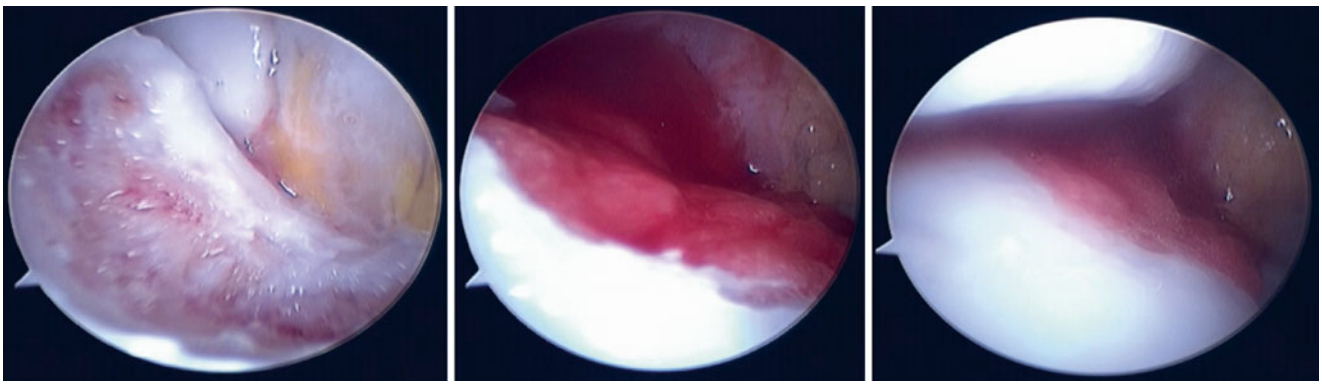


Fig. 52.3 Intraoperative arthroscopic images during BMDCT. This procedure can be performed using an arthroscopic one-step technique. First, the lesion is debrided, reaching a healthy subchondral bone. Then the biomaterial, a collagen membrane loaded with autologous mesenchymal stem cells, is implanted. Then a layer of PRF is sprayed on the biomaterial, to improve the stability of the implant and the cell differentiation and growth

culture and seeded on a hyaluronate membrane. After 3 weeks, the second step takes place, and the biomaterial is arthroscopically implanted onto the lesion. The hyaline regeneration was confirmed by histological and radiological outcomes [29, 30].

52.3.2.6 Bone Marrow-Derived Cell Transplantation

Bone marrow-derived cell transplantation (BMDCT) is a regenerative technique for bony and chondral layer, based on mesenchymal stem cells [19, 24, 31]. This technique may be performed in one step, in a same surgical session, or with more steps, with cells culture and enrichment: good results were achieved also in degenerated joints [19, 24, 31]. In the one-step technique, the cells are harvested from the iliac crest using a bone marrow needle. During the concentration, a standard arthroscopy of the ankle is performed; the joint and the defect is debrided. The cell concentrate is loaded on a collagen (or hyaluronate) membrane and then implanted in the joint using a specific instrumentation [31]. Then, a layer of platelet-rich fibrin (PRF) is sprayed on the biomaterial, to improve growth and differentiation and stability of the implant. Clinical results at medium-term follow-up are encouraging, with excellent outcomes even in athletes. Hyaline cartilage regeneration has been appreciated in bioptic samples and MRI qualitative scans [19, 31].

52.3.2.7 Allograft

Ankle allograft is a biological reconstruction, which should be reserved to high degenerated joints: it can be partial or total [19, 24]. Ankle may be approached laterally or anteriorly, with a fixation of both the articular surfaces using articular pins. The clinical and radiological outcomes are encouraging, and there is evidence of hyaline cartilage presence and colonization of host cells [19, 24]. Nevertheless, the indications of this procedure are selective and encompass young, active people with disrupted anatomy of the ankle [19].

52.4 Rehabilitation and Return to Play

Very few evidences about rehabilitation and return to play exist in literature, and many confounding factors may vary the outcomes [32]. Youth, small lesions, and lower BMI have been advocated as positive prognostic factors in a precocious sport comeback [32]. Nowadays, the two key points in cartilage rehabilitation are continuous passive motion, which may provide a good chondral nutrition, and careful weight-bearing, which may avoid the deleterious effects of overloading. Positive results may be achieved, thanks to pulse electromagnetic fields, bisphosphonates, or injective therapy with hyaluronate or platelet-rich plasma [23, 32]. A personalized program should always be recommended in athletes.

52.4.1 Fragment Fixation

A posterior splint or cast is advised in the first 2 weeks, then a passive continuous motion is recommended, and a partial weight-bearing, possibly with ankle in brace, is allowed not before 4 weeks [21].

52.4.2 Drilling

Active movements are encouraged since the day after surgery, while weight-bearing is proscribed for 6 weeks [25].

52.4.3 Microfractures

Rehabilitation after microfractures requires non-weight-bearing in ankle brace for 3 weeks. A progressive weight-bearing is then allowed, promoting exercises for proprioception and range of motion. Return to sport was advised not before 12 weeks [26]. In a work by Lee, early or delayed weight-bearing

(after 1 week or 6 weeks) after microfractures for OCL did not influence the final outcome [33].

52.4.4 Mosaicplasty

Continuous passive motion is allowed the day after surgery, but the ankle is kept non-weight-bearing for 3 weeks (6 weeks in case of osteotomy), which is necessary for graft incorporation [22, 28]. A progressive, partial weight-bearing is allowed. After 6 weeks complete weight-bearing is allowed, and athletic activities can be started after 6 months after surgery [22, 28].

52.4.5 ACI and BMDCT

Regenerative techniques require a specific timetable for rehabilitation, due to biological properties of the implanted cells [34]. For a large division and initial incorporation, chondrocytes require 6 weeks. Between 3 and 6 months, a primitive extracellular matrix is produced [19, 24, 34]. After 6 months, a progressive integration of the biomaterial with the subchondral bone occurs. Remodeling and maturation continue for 2–3 years [34]. The rehabilitation protocol for regenerative techniques should take into account this process, resulting in a mix of continuous passive motion, progressive weight-bearing, and muscular strengthening. Personalized schemes should be encouraged, but they have to share a precise program. The day after surgery, continuous passive motion is advised, and a Walker ankle brace is applied [34]. The period of non-weight-bearing lasts about 6 weeks; then a period of partial, progressive weight-bearing of 2 weeks follows. After 4 months from surgery, low-impact sport activities (swimming, cycling, etc.) can be safely performed. A progressive return to running and high-impact sport activities is not allowed before 10 months.

Conclusions

Athletes required effective treatments for OCL, with a rapid sport comeback. This aim is quite difficult to be achieved, as a good restoration of qualitative and durable hyaline cartilage can be achieved only through regenerative techniques. These procedures, due to biological reasons, need longer time to heal. Although clear guidelines for OCL in athletes do not exist, conservative treatment should be proposed only in very small, not painful OCL. Debridement in small acute OCL and fixation in larger defects are two effective procedures. Microfractures could be advised in symptomatic, small chronic OCL (1.5–2 cm^2). Larger chronic lesions may pose a serious challenge: microfractures may not achieve satisfying results. If regenerative techniques are suggested, athletes should be clearly warned of the longer times needed for rehabilitation after such procedures.

References

1. van Dijk CN, Reilingh ML, Zengerink M et al (2010) Osteochondral defects in the ankle: why painful? Knee Surg Sports Traumatol Arthrosc 18(5):570–580. doi:10.1007/s00167-010-1064-x
2. Schachter AK, Chen AL, Reddy PD, Tejwani NC (2005) Osteochondral lesions of the talus. J Am Acad Orthop Surg 13(3): 152–158
3. Saxena A, Eakin C (2007) Articular talar injuries in athletes: results of microfracture and autogenous bone graft. Am J Sports Med 35(10):1680–1687
4. Choi WJ, Park KK, Kim BS, Lee JW (2009) Osteochondral lesion of the talus: is there a critical defect size for poor outcome? Am J Sports Med 37(10):1974–1980. doi:10.1177/0363546509335765
5. Orr JD, Dutton JR, Fowler JT (2012) Anatomic location and morphology of symptomatic, operatively treated osteochondral lesions of the talus. Foot Ankle Int 33(12):1051–1057. doi:10.3113/FAI.2012.1051
6. Murawski CD, Kennedy JG (2013) Operative treatment of osteochondral lesions of the talus. J Bone Joint Surg Am 95(11): 1045–1054. doi:10.2106/JBJS.L.00773
7. Talusan PG, Milewski MD, Toy JO et al (2014) Osteochondritis dissecans of the talus: diagnosis and treatment in athletes. Clin Sports Med 33(2):267–284. doi:10.1016/j.csm.2014.01.003
8. Canale ST, Belding RH (1980) Osteochondral lesions of the talus. J Bone Joint Surg Am 62(1):97–102
9. Koch S, Kampen WU, Laprell H (1997) Cartilage and bone morphology in osteochondritis dissecans. Knee Surg Sports Traumatol Arthrosc 5(1):42–45
10. Johnson DL, Urban WP Jr, Caborn DN et al (1998) Articular cartilage changes seen with magnetic resonance imaging-detected bone bruises associated with acute anterior cruciate ligament rupture. Am J Sports Med 26(3):409–414
11. Guettler JH, Demetropoulos CK, Yang KH et al (2004) Osteochondral defects in the human knee: influence of defect size on cartilage rim stress and load redistribution to surrounding cartilage. Am J Sports Med 32(6):1451–1458
12. van Dijk CN, Reilingh ML, Zengerink M et al (2010) The natural history of osteochondral lesions in the ankle. Instr Course Lect 59:375–386
13. Dheer S, Khan M, Zoga AC et al (2012) Limitations of radiographs in evaluating non-displaced osteochondral lesions of the talus. Skeletal Radiol 4:415–421
14. Navid DO, Myerson MS (2002) Approach alternatives for treatment of osteochondral lesions of the talus. Foot Ankle Clin 7(3): 635–649
15. Ferkel RD, Flannigan BD, Elkins BS (1991) Magnetic resonance imaging of the foot and ankle: correlation of normal anatomy with pathologic conditions. Foot Ankle 11:289–305
16. Bae S, Lee HK, Lee K et al (2012) Comparison of arthroscopic and magnetic resonance imaging findings in osteochondral lesions of the talus. Foot Ankle Int 33(12):1058–1062. doi:10.3113/FAI.2012.1058
17. Verhagen RA, Struijs PA, Bossuyt PM et al (2003) Systematic review of treatment strategies for osteochondral defects of the talar dome. Foot Ankle Clin 8(2):233–242, viii–ix
18. Niemeyer P, Salzmann G, Schmal H et al (2012) Autologous chondrocyte implantation for the treatment of chondral and osteochondral defects of the talus: a meta-analysis of available evidence.

Knee Surg Sports Traumatol Arthrosc 20(9):1696–1703. doi:10.1007/s00167-011-1729-0
19. Giannini S, Buda R, Faldini C et al (2005) Surgical treatment of osteochondral lesions of the talus in young active patients. J Bone Joint Surg Am 87(Suppl 2):28–41
20. Badekas T, Takvorian M, Souras N (2013) Treatment principles for osteochondral lesions in foot and ankle. Int Orthop 37(9): 1697–1706. doi:10.1007/s00264-013-2076-1
21. Liu W, Liu F, Zhao W et al (2011) Osteochondral autograft transplantation for acute osteochondral fractures associated with an ankle fracture. Foot Ankle Int 32(4):437–442. doi:10.3113/FAI.2011.0437
22. Hangody L (2003) The mosaicplasty technique for osteochondral lesions of the talus. Foot Ankle Clin 8(2):259–273
23. Mei-Dan O, Carmont MR, Laver L et al (2012) Platelet-rich plasma or hyaluronate in the management of osteochondral lesions of the talus. Am J Sports Med 40(3):534–541. doi:10.1177/0363546511431238
24. Giannini S, Vannini F (2004) Operative treatment of osteochondral lesions of the talar dome: current concepts review. Foot Ankle Int 25(3):168–175
25. Taranow WS, Bisignani GA, Towers JD et al (1999) Retrograde drilling of osteochondral lesions of the medial talar dome. Foot Ankle Int 20:474–480
26. Ventura A, Terzaghi C, Legnani C et al (2013) Treatment of post-traumatic osteochondral lesions of the talus: a four-step approach. Knee Surg Sports Traumatol Arthrosc 21(6):1245–1250. doi:10.1007/s00167-012-2028-0
27. Yoshimura I, Kanazawa K, Takeyama A et al (2013) Arthroscopic bone marrow stimulation techniques for osteochondral lesions of the talus: prognostic factors for small lesions. Am J Sports Med 41(3):528–534. doi:10.1177/0363546512472979
28. Hangody L, Dobos J, Baló E et al (2010) Clinical experiences with autologous osteochondral mosaicplasty in an athletic population: a 17-year prospective multicenter study. Am J Sports Med 38(6):1125–1133. doi:10.1177/0363546509360405
29. Giannini S, Buda R, Ruffilli A et al (2014) Arthroscopic autologous chondrocyte implantation in the ankle joint. Knee Surg Sports Traumatol Arthrosc 22(6):1311–1319. doi:10.1007/s00167-013-2640-7
30. Battaglia M, Vannini F, Buda R et al (2011) Arthroscopic autologous chondrocyte implantation in osteochondral lesions of the talus: mid-term T2-mapping MRI evaluation. Knee Surg Sports Traumatol Arthrosc 19(8):1376–1384. doi:10.1007/s00167-011-1509-x
31. Giannini S, Buda R, Battaglia M et al (2013) One-step repair in talar osteochondral lesions: 4-year clinical results and t2-mapping capability in outcome prediction. Am J Sports Med 41(3):511–518. doi:10.1177/0363546512467622
32. van Eekeren IC, Reilingh ML, van Dijk CN (2012) Rehabilitation and return-to-sports activity after debridement and bone marrow stimulation of osteochondral talar defects. Sports Med 42(10): 857–870. doi:10.2165/11635420-000000000-00000
33. Lee DH, Lee KB, Jung ST (2012) Comparison of early versus delayed weightbearing outcomes after microfracture for small to midsized osteochondral lesions of the talus. Am J Sports Med 40(9):2023–2028. doi:10.1177/0363546512455316
34. Nho SJ, Pensak MJ, Seigerman DA et al (2010) Rehabilitation after autologous chondrocyte implantation in athletes. Clin Sports Med 29(2):267–282, viii. doi:10.1016/j.csm.2009.12.004

Ankle Arthritis in Athletes

53

Francesco Allegra and Stefano El Boustany

53.1 Background

Ankle bears load forces per cm^2 superior to other joints. Despite undergoing trauma more frequently than any other district, the prevalence of symptomatic ankle arthritis is approximately nine times lower than hip and knee [1]. The ankle moves mainly with a rolling mechanism and matches even at high loads: the knee, on the other hand, shows a combination of rolling, sliding, and rotation, preparing in this way to a higher frequency of appearance of arthritis [2, 3]. Articular cartilage has distinct characteristics with regard to thickness, mechanical properties, and metabolism compared to the knee and hip, which protect it from the onset of primitive osteoarthritis but predispose to post-traumatic arthritis if not anymore intact. Cartilage thickness has indirectly proportional correlation with its compression module; the more subtle the former, the higher the latter. Ankle articular cartilage has a uniform thickness between 1 and 1.7 mm, while knee varies from 1 to 6 mm. Therefore, ankle is the most consistent joint with thinner cartilage to better distribute the articular stresses homogeneously [4] and allows to carry loads up to 5 times the weight of the body during normal gait level. Any load is distributed approximately 75 % on talar dome and the remaining on medial and lateral facets [2]. There are numerous risk factors in practicing sports: they can be divided into intrinsic and extrinsic [5]. Intrinsic factors include gender, age, previous traumatic episodes, ligament laxity, joint instability, limitation of movement, strength loss, and finally alignment. Among the latter may be mentioned firstly environmental conditions and sports equipment, followed by level of competition, playing time, type of sports, and exercise. There are many sports that can help in inducing a post-traumatic ankle arthritis, especially contact ones, but even some of those, where the sport is exercised without risk of collisions with other athletes, recognize a particular frequency of injury due to the extreme use.

F. Allegra (✉) • S. El Boustany
Ortopedia Universitaria, Università La Sapienza, Polo Pontino, c/o I.C.O.T., Via Francesco Faggiana 1668, Latina 00040, Italy
e-mail: francescoallegra@tin.it; Stefano.elb@libero.it

53.2 Epidemiology

The ankle is the most frequently exposed joint to sports trauma: the percentage of hospital emergency room access varies considerably in the international statistics depending on the country. Not all sprained ankles are submitted to a treatment at first aid, being cared directly on sports field by team physician: for this reason, they can escape any official statistic. In general, the incidence of ankle trauma is 37 events/1,000 person with a progressive increasing trend. Ankle sprains represent 10–15 % of all sports injuries. Ankle arthritis can be induced firstly by bony fractures. The malleolar and tibial plafond fractures and the talar fractures associated or not with calcaneus are among the top causes of degenerative joint disease of the ankle [1]. Joint instability, due to chronic ligament laxity, medial or lateral, can lead to degenerative changes particularly on the medial side [6]. These, associated together, hold more than 80 % of all causes of ankle arthritis [1, 2, 6]. The remaining 20 % is determined by osteochondritis dissecans, osteochondral lesions, and chronic inflammatory diseases with mainly articular localization such as rheumatoid arthritis and psoriatic arthritis. Traumatic anterior compartment pathology is more diffused in sports and therefore more studied, more rarely is involved the posterior compartment [7].

The tibial plafond fractures are often as result of high-energy forces and are associated with a high percentage of post-traumatic arthritis, caused by the combination of joint damage, avascular necrosis of fragments of intra-articular fracture, post-traumatic infection, or sub-infection because of the difficulty of obtaining adequate surgical reduction of the fragments. Although the prevalence of ankle post-traumatic arthritis has a frequency as in other joints, an important predictive factor is severity in articular cartilage

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_53,

damage. Lindsjo [8] has investigated a series of more than 300 ankle fractures, in active population, treated with open reduction and internal fixation (ORIF) surgery and concluded that appearance of post-traumatic arthritis is direct consequence of fracture type, stood at 14 %. Lindsjo's study also showed that perfect reduction of fracture strongly influences the result of treatment. When arthritic changes appear following an ankle fracture, they tend to be highlighted on x-ray within the next 2 years. A strong correlation has been shown between the radiographic images, the severity of osteoarthritis, and clinical results. A separate consideration deserves talus fractures: even if they are most rare of tibial plafond and malleolus fractures, they can induce arthritis of the ankle and subtalar joint. Daniels [9] states that the frequency of appearance of osteoarthritis following fractures involving talus and subtalar joint ranges from 47 to 97 %. Among these, fractures of the neck are the direct result of joint damage by a collapse of subchondral bone, secondary to avascular necrosis or nonunion: the appearance of post-traumatic arthritis can also be extended in these districts. Initially present only on articular affected side, it is then extended to all other compartments in different time [9].

Ligament injuries secondary to ankle sprain have sequelae in chronic ankle instability, due to frequent episodes which athletes are involved during their sporting life. In a prospective study, Danish authors examined 766 patients affected with ankle sprain and submitted to first care at emergency room of the hospital where they were employed. This pathology represented the 4 % of all the hospital accesses on 1 whole year. The majority of sprains appears to be secondary to sports practice (45 %), but proceeding with age, different activities become dominant. External joint compartment was involved in 61 %. Average age was 24.4 for both sexes, and prevalent gender was male (58 %), but after age 40, the data reversed in favor of women. The male peak was between ages 20 and 30, but for female, the peak appeared between 10 and 20 years. The seasonal tendency was major during spring with an average of 62 % of all cases that happened in open air. Authors concluded the ankle is the most affected joint by sports trauma [10]. Effectively, ankle joint stability is essential for normal articular motion, minimizing the risk of joint trauma during game participations. The capability of static and dynamic stabilizers to keep their structural integrity is the major component of the whole normal gait cycle. This quality in sports assumes a major importance because of the increased range of motion and stresses which ankle joint is submitted to during different sport disciplines. Even though the ankle sprain average is high in active population, external ligament compartment is the most common traumatic disorder secondary to sports raising 25 % of all sports traumas [11].

Though sporters affected with sprain should come back to their functional activity in a fast way, almost 40 % of them continued to refer problems on affected side for a period of various months after first episode. Among all sports, the contact ones are first in statistics for determining ankle joint traumas, and soccer is absolutely the main cause [11].

In a Swedish prospective study [12], authors correlated the ankle sprains with time of game in soccer. Examining four male divisions at different levels of ability for 1 year, the exposition to ankle trauma risk and the accident number per player were higher in the first division, but their incidence and percentage were the same at all different levels of capability. Ankle sprains reached 17–20 % of all reported traumatic disorders, and their incidence was 1.7–2.0 events any 1,000 h of exposition to game. Any further episode represented an increased risk of recurrence during sport activity. Chronic ankle dysfunction due to ligament chronic disorder is a prevalent problem of sporters who have been formerly affected with and represents the cause of chronic ankle instability, strongly influencing the appearance in times of joint arthritis.

However, examining the complete ankle kinematics, some authors [13] correlated it with tibiotalar joint affected with arthritis, pointing out the effects on subtalar joint movements. They discovered that medial compartment arthritis can influence the kinematics of whole ankle joint complex. Compared to normal ankle behavior, their study suggested that the whole subtalar joint movements in all different planes have the tendency to shift in opposite direction in subjects with medial compartment arthritis, during the stance phase of gait. These reported conclusions represent a strong failure of normal joint motion, related to the physiological kinematics in any healthy articular complex in motion, thus limiting any performance and inducing joint symptoms.

53.3 Physical Examination

Clinical examination is generally emphasized by authors to point out the health condition of the patient. However, this view is opened to criticism because no particular sign is suggested in evaluating an arthritic ankle. The direct visual inspection of the ankle and foot will aim to identify the alteration of anatomical profile and conformation, comparing it with the other side. The normal profile is often altered by more prominence of malleolar edges because of deformity of previous fractures or of chronic instability. Generally, periarticular soft tissues are involved in little swelling. Sometimes it is possible to appreciate a bony prominence consistency on anterior side expression of a spur. An anamnestic fracture or many sprain episodes can lead to a correct diagnosis from the beginning: the perception of a sharp sound or feel of a bump to the ankle, the timing in the onset of pain as intensity and duration during activity, and the swelling onset with or without a bruise can help in diagnostic exam. Palpation will search the sore points of the bony profiles, ligaments, and tendons, progressing both front and rear, evaluating whether statically or passively moving the involved district.

Examining range of motion, particular attention is given to dorsal and plantar flexion of the foot and ankle, lateral movements in varus and in valgus, tibio-peroneal-talar supination and pronation, and the motility of subtalar and midtarsal joints, easily performed in chronic condition. Of great importance are the functional tests, which can highlight pathologic joint laxity secondary to ligament involvement. The test of supination highlights excessive movement on the affected side compared with the other one, forcing the joint in order to repeat a traumatic event. The search of talar "tilt" is performed with the examiner with one hand fixing the distal end of the leg and with the other hand grabbing the heel back, launching a forced movement in varus or valgus stress: a pathological ankle shifting is expression of a ligament involvement. The anterior drawer test tries to force the ankle ahead, giving a boost on the heel caught on the examiner hand, while the other one impresses an opposite movement: the presence of ligamentous injury in the ankle moves forward most of the talus against anterior tibial plafond. The "squeeze test" is a test to assess the involvement of the syndesmosis, squeezing with both hands the involved region determining pain. All these tests have the limit not to have a prognostic value on the treatment of ankle instability, which generally is well accepted by the athlete manifesting a particular compliance with this disorder, with few problems for their specific activity. Stiffness is generally present in a lot of arthritic ankles, expression of an involvement of whole joint in the degenerative changes, but not necessarily linked to a particular radiological aspect such as spurs or ossicles. Athlete complains of this symptom which is not well accepted because of strongly influencing sports performances during the game.

53.4 Diagnostics

The instrumental diagnosis is of great assistance to determine the extent of ankle arthritis. The x-ray plan scan in two positions allows to study the relationship of reciprocity between the various components of bone and highlights any alteration [14]. The bony profiles appear to be modified by the presence of anterior spurs both on tibial side and on talar one: it is very useful to submit the patient after surgery to a new scan to ensure its removal (see Figs. 53.1 and 53.2). Sometimes they appear as kissing lesions engaging during a dynamic study. Posterior compartment is not void of these lesions, appearing also in the hindfoot and involving tibial profile and talus at os trigonum area. However, the presence of spurs can be detected both anteriorly and posteriorly. Ossicles can be present on the malleolar tip. Loose bodies should be evidenced only if ossified, but cartilaginous ones do not relieve their presence on scans. The talar dome can be deformed and presents a flat shape such as the tibial plafond, even if the movement is maintained for quite a good range.

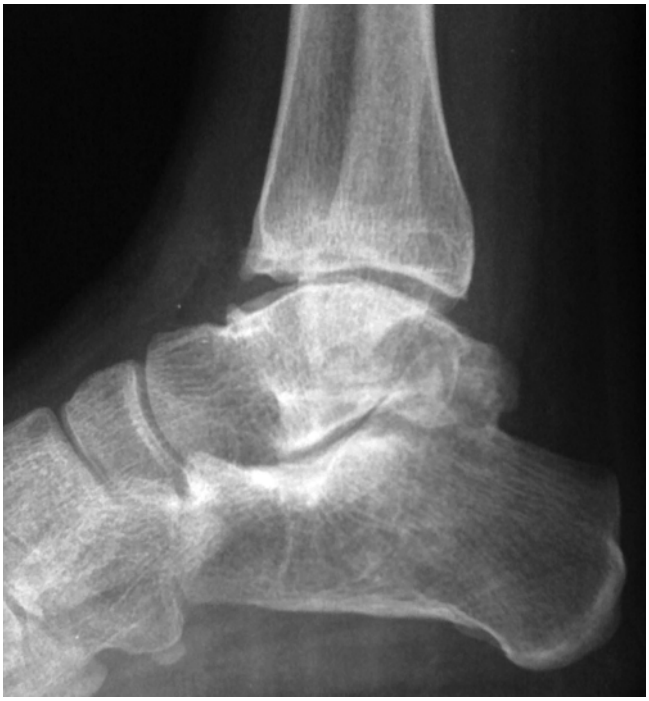


Fig. 53.1 The preoperative x-ray plan sagittal scan of arthritic ankle evidences the presence of anterior bony spur both on talar and tibial side

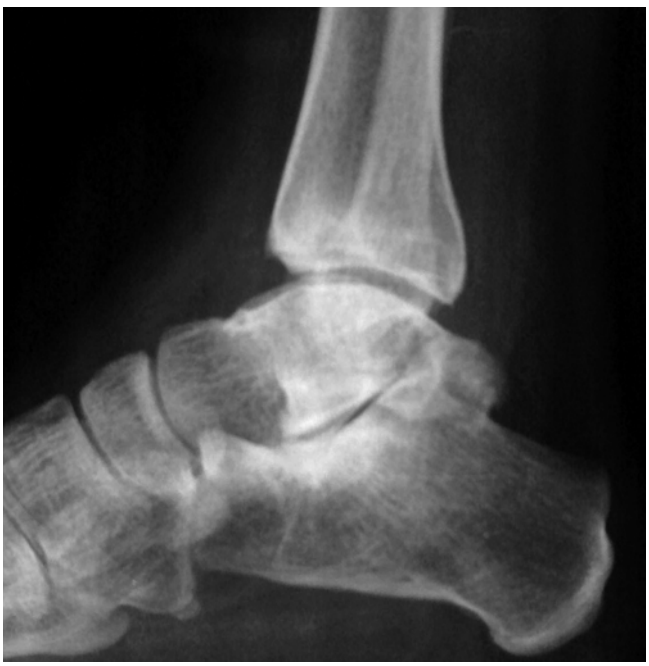


Fig. 53.2 The postoperative x-ray plan sagittal scan of arthritic ankle, submitted to arthroscopic debridement, confirms the surgical result of bony spur removal on talar and tibial side, recreating a good anterior articular space

In the case of arthritis secondary to chronic ankle instability, the functional radiographs give an added value to instrumental diagnostics, allowing to evaluate indirectly the capsule-

ligament chronic injured ankle, with the only limit represented by patient compliance due to pain. Submitting the patient to stress x-ray by the Telos tool, it is possible to measure in millimeters the anterior translation of the talus relative to the tibia (the so-called anterior drawer), detected at the posterior margin of the tibia and talus, and this lateral shifting in supination (the so-called talar tilt) measures the angle between the junction of the parallel line of the talar dome with the tibial plafond.

The ultrasound shows a high reliability because of the minimally invasive and big accuracy of the detected images, because it highlights not only the ligaments but also the associated tendon degenerative injuries. Magnetic resonance imaging provides the opportunity to study articular areas deeply, which escape the ultrasound examination and which are not detectable by traditional radiographs: its use in chronic condition is appropriate to particularly selected cases of low reliability with other instruments, both for appropriate treatment and for prognostic purposes.

53.5 Surgical Arthroscopic Procedure

Arthroscopy belongs indication for the first treatment of ankle arthritis, despite having gained over time its precise role in all the other joints as a tissue-saving procedure with lack of invasiveness. One of the first choice treatments is articular debridement performed by arthroscopy. Arthroscopy is addressed to evaluate the disorder (see Fig. 53.3), to remove synovial pathology, bone spurs, loose bodies (see Fig. 53.4), and ossicles that make symptomatic ankle joint as hindfoot [15, 16]. The reactive chondral debris-induced modifications of soft tissues are disabled with arthroscopic surgery. This treatment is limited to the initial ankle arthritis, without any deformity, any mechanical axis variation, and any joint space absence. Arthroscopy has the role of mini-invasive procedure and is respectful of disabling skin dystrophies as consequences of important traumas or bony profile deformity, thus avoiding the potential complications secondary to infection and to low capability of mortified skin to regenerate. It permits a selectivity of treatment to single sides of joint, not possible in open surgery because it is limited by surgical access, allowing to explore and to address surgery both anteriorly and posteriorly, laterally and medially, simply by exchanging the instruments inside the surgical portals, thus allowing wider angles of vision and work.

The association of surgical debridement with articular distraction by external fixation is reserved to younger patients with severe arthritis at the final stage, who are arthroplasty or arthrodesis candidates. The association between arthrodesis and arthroscopy gave fresh impetus to this surgical choice, ensuring a very low local aggressiveness despite the total big bone removal from the joint necessary for a good articular fusion. Arthrodesis is the treatment indicated at final stage of osteoarthritis and at failure of all other therapies, within the correct indication. However, it is not indicated for athletes in full sports activity because of the total lack of motion, representing the end of any ankle sports-related performance.

The arthroscopic procedure longer used is articular debridement (see Fig. 53.5); the term debris comes from

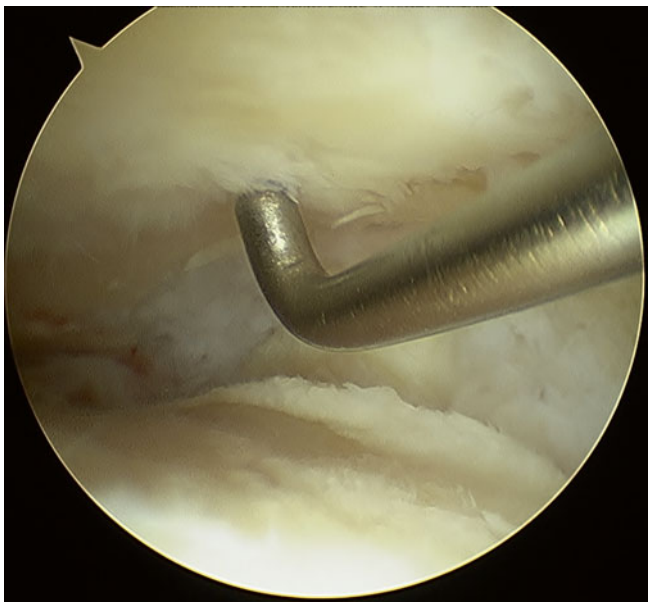


Fig. 53.3 The surgical picture shows the space between the two tibial (*above*) and talar (*below*) chondral surfaces: a big erosion of cartilaginous layer involves all the talar dome, while on tibial plafond, the detached cartilage is simply removed by probe which is going to appreciate the consistency of the articular several layers

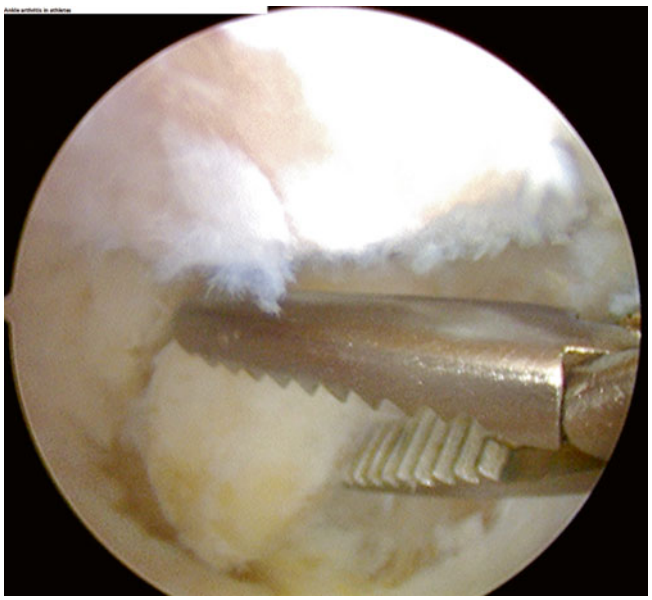


Fig. 53.4 An ossicle is found at the malleolar tip after cleaning up the joint and removing all the pathological tissue which hides the distal medial compartment: the punch grip is grabbing it out from the articular space

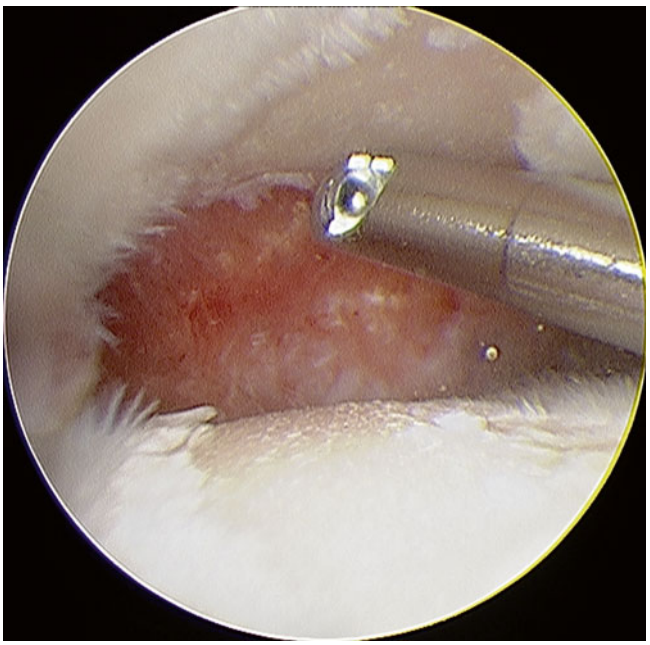


Fig. 53.5 A 4 mm shaver blade is inserted into the joint space to clean up the posterior compartment from anterior, to reaching a complete visualization of this area

French language and is reused literally in English language [17]. It is considered as the remaining of biological tissue, bone or cartilage, physiological or not, loose inside the joint, or linked to articular structures, which can assume a pathological significance according to its quantity, its placement, and its presence in critical areas subjected to movement or friction. Its presence becomes symptomatic triggering a widespread articular suffering, which often is associated to chondral surface pathology [18]. With the diffusion of arthroscopic removal of those debris and fragments, debridement is stated as one of the most common ankle joint surgical procedures. Technique [15] provides the patient's assessment supine with inferior limb on a leg holder and the ankle distracted, remaining free to perform movements on surgeon's needs, despite firmly wrapped in a noninvasive traction. If the joint region to be treated is posterior, the patient is assessed in prone position, with foot outside the surgical bed on a roll pillow placed below the pretibial region, in order to release any movement of the joint from bed [16]. In this case, ankle traction is not needed, just forcing movements to open ankle joint enough to be addressed by instruments. Alternating the position of scope and instruments on three selected portals, a targeted removal of pathological inflamed and fibrotic tissues is performed: tools such as basket or radio-frequency wands are needed to remove particularly tough and stiff tissues which make blind any visualization. Once cleaned up the spotted compartment, those loose bodies or ossicles in released articular recesses or gutters can be highlighted. If chronic inflammatory clinical aspects are diffused inside compartments, the association with synovectomy remains indicated, as largest as possible both in medial and lateral and frontal and rear compartment, although final results remain fair in short to medium term because of the possibility of inflammatory tissue recurrence. These surgical procedures make valid the arthroscopic debridement in solving a big part of symptoms of athletes. The rehabilitation protocol is very fast, permitting an immediate weight-bearing with crutches if needed by the patient: exercise to reinforce strength and to develop proprioception is immediately started. Better coordination between muscle and training activities addressed to the perception of balance makes possible to prevent the occurrence of further trauma, resulting in improved muscle strength and improved proprioceptive response from the operated ankle.

In some selected cases, with a small arthritic involvement of the joint, the association with removal of detached various-sized cartilage fragments should be associated with microfractures or perforations, aiming to revitalize the subchondral bone according to the lesion dimensions. A medium to long rehabilitation period will complete the treatment with respect to partial weight-bearing. The tibial or talar bony spurs reduce the joint space, reduce the range of motion, and cause erosions on the underlying or overlying cartilage, increasing joint damage and making more complex this surgical procedure. Their removal is easily performed by bur blades and cutters which remove the excess of bone under direct scope visualization. The painful giving way and joint blocks are often caused by the presence of loose bodies or malleolar ossicles, freely placed in the articular environment. However, their removal does not always resolve symptoms definitely and even prevents further damage related to their presence and their movement. Often, the loose bodies originate from detachments of cartilage layer portion, which remains damaged by sports-related overuse originating an osteochondral lesion. Therefore, their removal must often be associated with repair of those bare areas most subjected to load, if their extension is greater than 15–20 mm^2 and the suggested surgical procedure permits to do immediately. Sometimes the chondral damage is also extended to the tibial plafond, generating insidious kissing lesions whose treatment is still questionable in the evaluation of the outcomes.

The age at time of arthroscopy has been shown to be an important risk factor for clinical outcome. Some authors have demonstrated that young athletes have a better clinical outcome compared with older ones, probably because the former have a larger component of soft tissue placed on both sides of arthritic joint. However, some data show that age is not a limit for arthroscopic treatment and is not significantly associated with clinical outcome. Similarly, it had been reported that the shortest duration of symptoms was associated with better clinical outcome at arthroscopic treatment. Overweight athletes have higher risk of osteoarthritis: best

candidates for arthroscopy appear to be those with a BMI lower than 25. The type of osteoarthritis (degenerative or post-traumatic) is considered an important concomitant predictive factor. Association with other intra-articular pathologies affects the arthroscopy's outcome. The presence of intra-articular lesions is observed in a range from 20 to 72 %: a complete resolution of associated injuries can guarantee favorable clinical outcome.

Conclusions

Sports at professional level can be risky for evolution of ankle disorders in arthritis. At its appearance, the specific indications for arthroscopic joint debridement are still controversial. However, in many cases, it appears to have good results, despite that it is seldom necessary to submit athletes again to this procedure because of recurrence at 3–4 years. Overall, there are no studies showing what are the positive prognostic factors of this surgery. Despite the absence of data, possible risk factors have been assumed such as age, sex, duration of symptoms, body mass index, type of osteoarthritis, previous treatments, and the presence of associated ligament injuries.

References

1. Staufer RN, Chao EY, Rewster RC (1977) Force and motion analysis of the normal, diseased and prosthetic ankle joint. Clin Orthop 127:189–196
2. Treppo S, Koepp H, Quan EC, Cole AA, Keuttner KE, Grodzinsky AJ (2000) Comparison of biomechanical and biochemical properties of cartilage from human knee and ankle pairs. J Orthop Res 18:739–748
3. Sthepherd DE, Seedhom BB (1999) Thickness of human articular cartilage in joints of lower limb. Ann Biomed Eng 23:697–704
4. Baumhauer JF, Alosa DM, Renström PA, Trevino S, Beynnon B (1995) Test-retest reliability of ankle injury risk factors. Am J Sport Med 23(5):371–374
5. Harrington KD (1979) Degenerative arthritis of the ankle secondary to long-standing lateral ligament instability. JBJS 61-A:354–361
6. McAuley D et al (1999) Ankle injuries: same joint, different sports. J Sport Med 28(1):35–48
7. Kuijt MT, Inklar H, Gouttebarge V, Frings-Dresden MHW (2012) Knee and ankle osteoarthritis in former elite soccer players: a systematic review of the recent literature. J Sci Med Sport 15(6):480–487
8. Lindsjo U (1985) Operative treatment of ankle fracture-dislocations. A follow-up study of 306/321 consecutive cases. Clin Orthop 199:28–38
9. Daniels TR, Smith JW (1993) Talar neck fractures. Foot Ankle 14:225–234
10. Holrner P, Smdergaard L, Konradsen L, Torben Nielsen P, Nannestad Jwgensen L (1994) Epidemiology of sprains in the lateral ankle and foot. Foot Ankle Int 15(2):72–74
11. O'Loughlin PE, Muraski CD, Egan C, Kennedy JG (2009) Ankle instability in sports. Phys Sportmed 37(2):93–103
12. Ekstrand J, Tropp H (1990) The incidence of ankle sprains in soccer. Foot Ankle Int 11(1):41–44
13. Kozanek M, Rubash HE, Li G, de Asla RJ (2009) Effect of post-traumatic tibiotalar osteoarthritis on kinematics of the ankle. Foot Ankle Int 30(8):734–740
14. Cain LE, Nicholson LL, Adams RD, Burns J (2007) Foot morphology and foot and ankle injury in indoor football. J Sci Med Sport 10(5):311–319
15. Ferkel RD (1996) Arthroscopic surgery. The foot and the ankle, 1st edn. Lippincott/Raven Press, Philadelphia/New York
16. Van Dijk CN (2000) Hindfoot endoscopy. Arthroscopy 8:365–371
17. Allegra F (2005) Debridement artroscopico di caviglia. In: Pellacci F (ed) Chirurgia Artroscopica dell'arto inferiore. Mattioli Editore, Fidenza (PR), Tomo 2, sez.2 cap.B2
18. Kim SH, Ha KI (2000) Arthroscopic treatment for impingement of the anterolateral soft tissue of the ankle. J Bone Joint Surg Br 82:1019–1021

Return to Sport After Ankle Lesions

54

Antonio Zanini, Manuel Bondi,
Pierfrancesco Bettinsoli, Matteo Benedini,
Andrea Pizzoli, and Lodovico Renzi Brivio

54.1 Introduction

The most common traumatic ankle injuries in sports are sprains, as shown by Hootman et al. [1] comparing 15 different sports. Capsule-ligament sprains represent approximately 85 % of ankle lesions [2]; the mechanism of 85 % of these is inversion (adduction-varus-supination), 5 % is subversion (abduction-valgus-pronation), while the remaining 10 % involves the tibiofibular syndesmosis [3]. Cartilage injuries may result from these traumatic events, especially in males (70 %), and these kinds of lesions are bilateral in 10 % of cases [4].

Football, volleyball, basketball, and rugby are the most involved sports because they have statistically the largest number of athletes.

The rehabilitation process will be different according to pathology type that has affected the ankle and depending on the type of treatment that the surgeon has decided to undertake.

In fact, we can distinguish three main groups of injuries, which ensue different types of treatments in arthroscopic surgery:

1. Ankle instability, due to acute ligamentous injuries and constitutional laxity
2. Degenerative diseases and synovial and bone impingements sometimes in addition to cartilage lesions
3. Chronic instability with degenerative diseases

A. Zanini, MD • M. Bondi, MD (✉) • P. Bettinsoli, MD
A. Pizzoli, MD • L. Renzi Brivio, MD
Department of Orthopaedic and Traumatology, Carlo Poma Hospital, Strada Lago Paiolo 10, Mantova 46100, Italy
e-mail: antoniozanini.mn@libero.it; bondimanuel@gmail.com; pierfrancesco.bettinsoli@aopoma.it; andrea.pizzoli@aopoma.it; lodovico.renzibrivio@aopoma.it

M. Benedini, DPT
Physiotherapist Freelancer, Fisiocenter Multimedica, Via Gobetti, Mantova 46100, Italy
e-mail: matteo@fisiocentermultimedica.com

54.2 Ankle Instability Rehabilitation After Surgical Treatment

There are multiple surgical methodologies and they are all followed by a period of immobilization in cast or brace (walker type), typically for 2–4 weeks depending on the surgery applied. The surgeon will define the timing of immobilization with no weight bearing.

Even in such cases edema and pain after surgery are needed to be taken under control; therefore, it is advisable to apply the PRICE treatment, Tecar, and laser therapies. This Anglo-Saxon acronym refers to limb *protection*, relative *rest*, *ice* application, *compressive bandaging* application, and maintaining limb *elevation* [5]. The simultaneous oral administration of NSAIDs has also shown efficacy in controlling pain and edemas. Medical staff should avoid dexterity that acts directly on the capsule and ligaments, especially in ankle subversion cases. The passive movement should be started early trying to restore the maximum joint ROM, while the recovery of the full joint movement is not urgent; it should be postponed in order to avoid damaging stresses.

54.3 Degenerative Diseases Rehabilitation After Surgical Treatment

The purpose of the rehabilitation consists in the best and more rapid functional recovery in order to minimize the degree of disability [6, 7]. In the majority of impingement cases, it is recommended to keep the limb in discharge for a few days to allow the reabsorption of edema and recover the mobility of the ankle without pain.

Therefore, during the first 3–4 days, in order to reduce pain, swelling, and lesion extension, surgeons have to apply the previously described PRICE.

Usual of this step is the use of physical therapies for therapeutic purposes, even if the literature does not show evidence of clinical efficacy [8, 9]. Manetti et al. [10] support the efficacy of thermal shock that consists in the alternation

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_54, © Springer International Publishing Switzerland 2016

of hyperthermia and cryotherapy, promoting tissue regeneration and resolution of the painful inflammatory symptoms.

54.4 Tecar and Laser Therapy

CRET (Tecar) therapy, which stands for capacitive-resistive electric transfer, is common in particular for drainage of edema and for the treatment of muscle contractures, such as those of the triceps complex and those of the posterior tibial complex [11].

There are three key actions of the Tecar therapy system. They are executed by two modes of operation, namely, the capacitive mode and the resistive mode. Classically, it is considered that the capacitive mode of operation acts on the tissues and more hydrated structures such as muscles, blood vessels, and lymphatic vessels, whereas the resistive mode acts on tissues and structures with high density of connective tissue (e.g., joint capsules, cartilages, fibrotic cords, ligaments, bone, tendons, etc.).

Tecar therapy causes the ionization of molecules of the tissues; the atoms that are released collide between them, and by transmitting to each other the movement, they cause recordable acoustic waves (phonons). A portion of the energy released by this process is converted into vibrational energy and, thus, into heat.

The last-generation technologies allow to work at higher frequencies (from 0.5 up to 1 MHz), and the efficiency of energetic transfer is guaranteed by a reading system able to detect the effective absorption of energy by the tissue using feedback in histograms.

In this way it is possible to treat the tissues with handpieces and large plaques in both thermal and athermal modes, taking advantage of the benefits of oxygenation and drainage and vascularization without major, and sometimes harmful, thermal increments. It has been proposed to use a simple infrared thermometer (or more evolved thermography systems) to monitor the thermal response curve of the patient at the beginning, during, and after the treatments.

As previously mentioned, as a consequence of surgery, the formation of local edema responsible for pain can occur. Lymphatic drainage massage and the simultaneous involvement of CRET therapy are useful in order to reduce local edema.

Another frequent physical therapy is the laser therapy. The acronym laser means light amplification by stimulated emission of radiation. In simple terms, but not less realistic, the laser can be considered as a light amplifier.

The laser acts in accordance with the basic laws of light, according to which light travels in a straight line at a constant speed in space and it can be transmitted, reflected, refracted, and absorbed. The light can be positioned within the electromagnetic spectrum in relation to frequency and wavelength, which vary depending on the generator under consideration.

The therapeutic windows are characteristic of many areas of therapy and medical practice, and, in the case of laser, the therapeutic window is between 600 and 1,100 nm.

The principles of bioactivation have been proposed by Karu et al. [12] who reported and demonstrated some key factors. The author, in fact, has noted that some biomolecules are able to modify their activity in response to irradiation with visible light of low intensity, but that the same molecules are not able to directly absorb the light energy. The primary absorption would be carried out entirely by the cell membrane, where the intracellular effects would be produced by the action of second messengers, with a response of "cascade" type.

The magnitude of the photoresponse would be determined at least in part by the state of cells and tissues prior to irradiation; this concept is wonderfully exemplified by the assumption that "the hungry cells would be more photosensitive than the well-fed ones" [13].

The most recent methods of laser therapy involve simultaneous administration of multiple frequencies and high energies, but with low thermal impact and with different wavelengths. They are positioned at the beginning, in the middle, and at the end of the therapeutic window (laser FP3 – Bergamo). The entire process occurs without increasing the temperature of the tissues, considered as "waste" in the process of laser therapy. This is possible by specific sensors able to detect temperature and skin impedance in real time, by adapting parameters such as the power and the "blending" of the various wavelengths instant by instant.

Occasionally, excessive rehabilitative loads or excessive effort by the patient causes the formation of tendon pathology, especially of the anterior tibial and peroneal, and an inflammation in the surrounding areas of where the arthroscope is introduced. In this case, the laser therapy drastically reduces pain and inflammation since the earliest sessions.

54.5 Proprioception

Proprioception, however, should not be ignored. It is stimulated by exercises performed in a sitting position in two different ways: in a unidirectional way if during the surgical phase a capsular "shrinkage" has been performed and in a multidirectional way if no action has been taken on the capsular-ligamentous apparatus.

The discharge on the operated limb depends, of course, on the type of surgical treatment and on the treated tissue. In the case of the removal of osteophytes, active and passive mobilizations for 2 and 4 weeks, instead, in the case of treatment of osteochondral lesions are recommended. This is followed by re-education that starts at the beginning into the water

with gradual load for another 15 days and then switching to progressive load on the ground for a further 2 weeks with a full and progressive muscle strengthening.

The recovery of muscle strength can be started very early. Initially, with the foot in a neutral position, contractions will be performed in plantar and dorsal flexion and eversion/inversion; then the therapy involves elastic resistance exercises to be carried out also to domicile.

With the progress of the recovery, exercises in a closed chain will be introduced, initially from a sitting position and then standing. Muscle strengthening exercises are synergistic to the articular recovery and across all this stage; the progression of workloads will depend on edema and painful systems. It is recommendable to continue with the use of functional bandages (taping) during the recovery; the use of Canadian sticks is procrastinated until the "step pattern" is free of pain and analgesic compensation.

54.6 Discharge and Brace

After surgery a period of discharge is often necessary and it is decided by the surgeon depending on the method used. The discharge is often accompanied by the use of a plaster splint or brace (like walker) to protect the joint from trauma and involuntary movements.

In the case in which the surgical treatment was an arthroscopic "shrinkage," the patient is advised to wear a brace for 3 weeks; otherwise it may be sufficient a taping.

54.7 Rehabilitation in Water

Contrary to what is argued years ago, the movement is today considered crucial to accelerate tissue repair and the subsequent recovery of the injured area. Early rehabilitation of the patient, in safety conditions, favors the recovery of soft tissues; it properly orients the deposition of collagen, partially preserves tropism and muscular tone, and maintains the athletic condition, if possible [2, 14–16].

Muscles and bones are subjected to less tension into water: they have a subtotal or partial loss of weight. This allows a control of the magnitude and of the speed of movement, facilitating the edema and pain reduction, allowing the execution of movements free of pain [17, 18].

The biological effects are related to an improvement of the circle and to a normalization of the swelling, allowing, as a physiological effect, a better and more rapid formation of neovascularization and innervation. The balance of power between agonist and antagonist muscles is crucial. Any exercise performed into water allows to train both muscle areas involved in movement with a mutual control and without creating joint or tendon overloads (Fig. 54.1).

The posture is controlled by allowing an activity that involves an erect position and with proper coordination: the therapist can notice that the movement is not performed along the predetermined line, while the patient realizes how this movement is not monitored [17, 18].

During rehabilitation, water must have a temperature near 32 °C (89.6 °F). The flow of water, ropes, elastic bands,

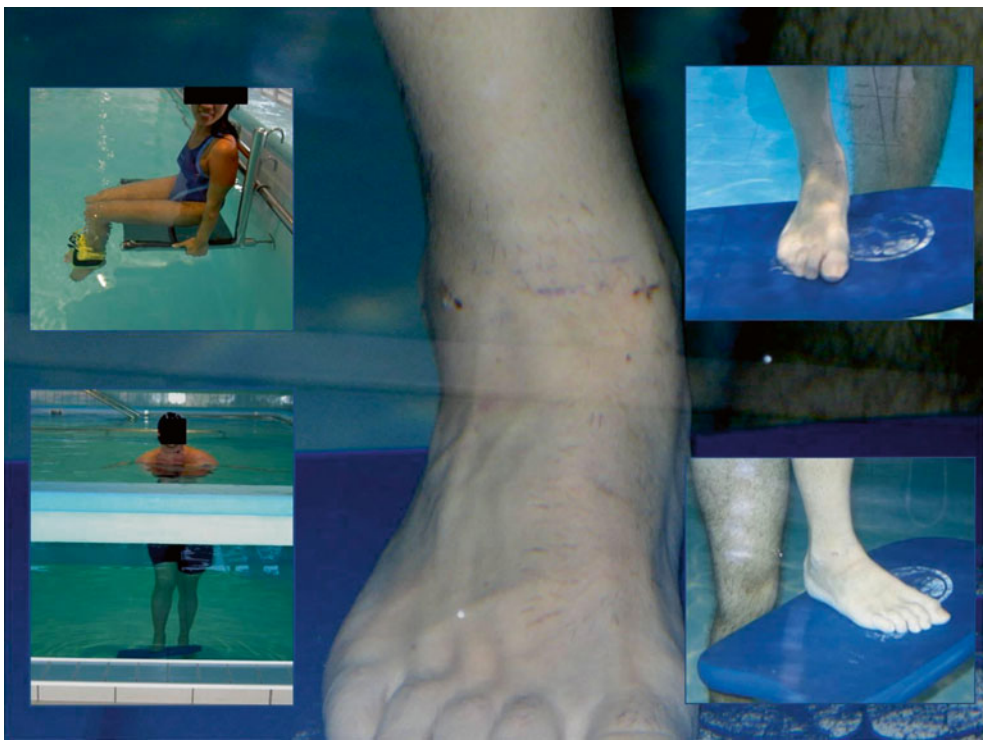


Fig. 54.1 Muscles and bones are subjected to less tension into water. It allows greater control of local edema, better mobilization, and greater muscle strengthening with a lower risk of functional overload

shovels, sticks, floats, weights, and even fins are the useful aids.

It should be noted that the density of water does not allow the speed increment; thereby, it is difficult to perform lactic acid muscle works that affect the anaerobic process. In this way the training activity involves a lower number of inflammations.

This type of rehabilitation is useful not only in the initial phase but also in the intermediate and final stages of the rehabilitation process for edema control.

54.8 NEURAC Rehabilitation

At the earliest stages of rehabilitation, we make use of a method called NEURAC (neuromuscular reactivation) that is carried out with a suspension system made by cables and ropes (defined as Redcord). This phase begins during unloading but can continue even after the giving of weight bearing. Such cables are applied in different points so that it is possible to create vectors of translation or rotation, as well as compression and decoaptation.

The peculiarity of this method is that it takes place in a closed kinetic chain emphasizing the role of the stabilizing musculature (type I fibers) and training the sensorimotor system.

It consists of the following:

1. Periarticular sensory afferents from bone, capsule, ligaments, tendons, and muscles
2. Interpretation of this information by the central nervous system and development of an efferent motor program
3. Periarticular muscles expressing the motor program prepared by the nervous system

After surgical treatment the sensorimotor system can be inefficient for several reasons: compensation previously established due to the pain, lack of peripheral afferents, lack of central ability to develop efficient motor programs, and insufficient periarticular muscle strength. Pain and injuries clearly accelerate, in a negative direction, the progression of these factors. Indeed, pain inhibits efficiency of the sensorimotor system. Any source of pain can interfere with the sensorimotor system producing a constant vicious circle "pain-sensorimotor deficit-pain." Therefore, it is important to reprogram the sensorimotor patterns of the whole fascial and muscular system, taking into account that the trunk and the pelvis (especially the transversus abdominis muscle, the quadratus lumborum, and the gluteus medius) are a very important key for the stability of the lower limbs and then the stability of the ankle (Fig. 54.2).

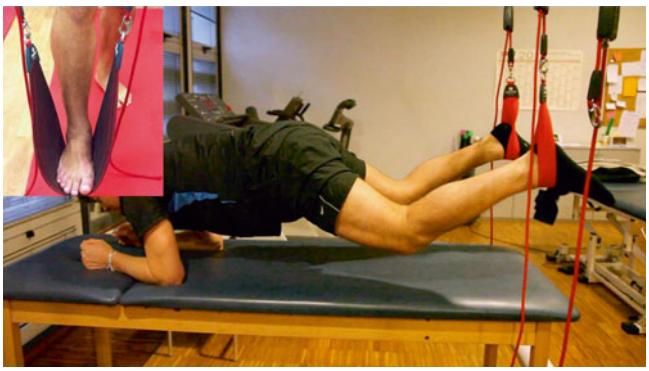


Fig. 54.2 NEURAC – record system. Is possible to improve patient's strength and functional capacity (coordination and balance)

They are useful exercises for all ages and improve patient's strength and functional capacity (coordination and balance). You can activate different muscle groups during a single exercise which is recommended for about 10 min 3 times week in relation to the patient's age.

54.9 Computerized Proprioception

This phase of training takes place after the giving of weight bearing.

Early mobilization promotes not only tissue nourishment but also a good orientation of the cicatricial fibers for capsular elasticity, and, subsequently, it allows to preserve a good muscle tone and tropism and maintain afferent and efferent neuromotor activities with a great advantage in the pursuit of the final goal, that is, the functional recovery.

The recovery of motor patterns and coordination is crucial, because only the reconquest of proprioceptive ability will allow a return to the sport. Afferent proprioceptive inputs are conveyed to different levels of the central nervous system [19], but most remain unconscious and only a very few (approximately one signal out of a million) are able to reach the conscious level [20]. The joint position sense and the joint movement sense (kinesthesia) are the expression of the conscious component, while postural control and joint

stability are mainly based on the unconscious component [19]. In the case of the antigravity movements, proprioceptive control is the expression of the effectiveness of stabilizing reflexes in controlling vertical stability [21]. By antigravity movements, we mean activities which require the individual to counteract gravity and postural instability with at least a phase of single-limb stance (walking, running, jumping, going up and down stairs, and so forth). Proprioceptive inputs are the most important sensory system in the maintenance of static postural stability at all ages.

Coupling single-stance stability with high-frequency instability of an electronic rocking board is able to reprogram proprioceptive control [22]. High-frequency instability is induced by the visual trace on the monitor (a feedback of the rocking movements of the board acting as feedforward) which tracks the subject and notably increases the number of corrections of the platform position. On the same rocking board, without visual tracking, you would experience instability only at low frequencies. The visual feedforward enhances the frequency of instability of the rocking base refining the postural reactions. In this way the postural adaptations can change from macroamplitude at low frequency to microamplitude at high frequency [22]. Besides the subcortical tracking, the visual information permits to assign specific tasks as trying to maintain the moving plate at an inclination corresponding to a certain level of supination or pronation (4–12° of supination, 4–8° of pronation) or passing from an inclination to another. These exercises of dynamic exploration are very effective to improve ankle functional stability and single-stance stability.

The stability tests and the high-frequency proprioceptive training can be performed by means of an electronic postural proprioceptive station (Delos Postural Proprioceptive System, Delos, Torino, Italy) (Fig. 54.3) [23] connected to a personal computer with a specific software. The station includes an electronic rocking board, an electronic postural reader, an infrared sensor bar, and a display. In case of risk of falling, the subject could lean on the bar placed in front of him to regain vertical control rapidly. The bar is equipped with an infrared sensor able to indicate when the subject leans on it. The electronic postural reader (DVC, Delos Vertical Controller), applied to the sternum, measures the trunk inclination in the frontal plane (x) and sagittal plane (y) by means of a two-dimensional accelerometer unit (Fig. 54.4). The electronic rocking board has a single degree of freedom on the frontal plane (range of motion: ±15°) and measures the inclination of its moving plate [24].

Fig. 54.3 Electronic postural proprioceptive station (Delos Postural Proprioceptive System, Delos, Torino, Italy). You can see the activity of the patient records on charts

54.10 Recovery of Athletic Movement and Return to Sport

For the athlete this is the most important phase: it is the only moment in which the athlete actually relives the return to sports. It should be noted that the training is a kind of specific

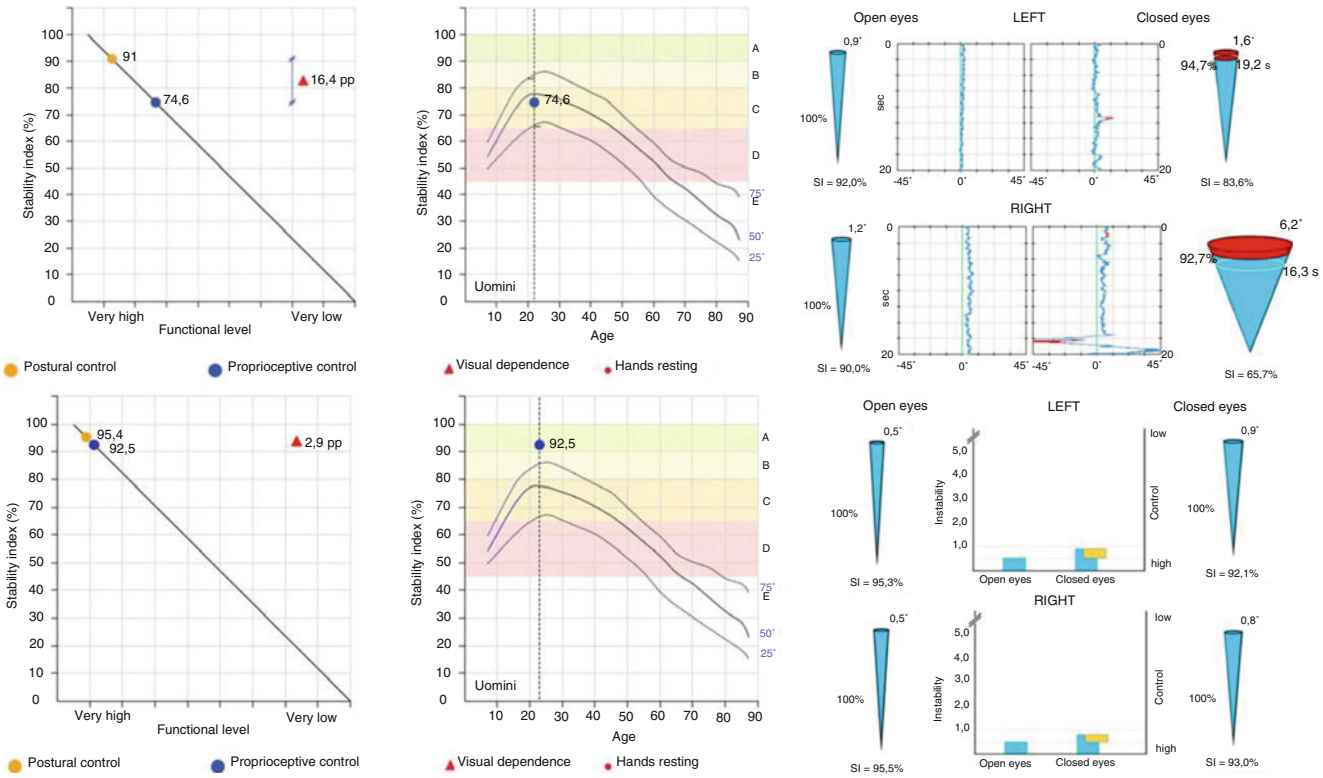


Fig. 54.4 Graphical comparison between the start and end of Delos therapy

Fig. 54.5 Isokinetic workup to increase muscular strength and isokinetic test to dynamically isolate the muscular group

sport and it is therefore essential to consult the team trainers to discuss about the purposes to be achieved and the time required.

The athlete can be asked to submit to an isokinetic workup of the ankle to increase strength with traditional isotonic exercise (Fig. 54.5).

At the last time of rehabiliation, we can use it as ankle test and threshold test. The first allows to dynamically isolate the muscular group under examination, assessing the performance across all the arc of contraction (constant resistance), estimating an average of deficit also for the dynamic recruitment. It is, however, a method that is not always easy to implement and difficult to find and that has a controversial utility. The second will give indications on metabolic conditions of the athlete.

Only when both tests give a positive outcome, the athlete will be allowed to return to sports [25].

The return to driving occurs usually between 2 weeks and 2 months.

References

1. Hootman JM, Dick R, Agel J (2007) Epidemiology of collegiate injuries for 15 sports: summary and recommendations for injury prevention initiatives. J Athl Train 42(2):311–319
2. Jones MH, Amendola AS (2007) Acute treatment of inversion ankle sprains: immobilization versus functional treatment. Clin Orthop Relat Res 455:169–172
3. McGuine TA, Greene JJ, Best T et al (2000) Balance as a predictor of ankle injuries in high school basketball players. Clin J Sport Med 10(4):239–244
4. Zengerink M, Struijs PA, Tol JL et al (2010) Treatment of osteochondral lesions of the talus: a systematic review. Knee Surg Sports Traumatol Arthrosc 18(2):238–246
5. Ivins D (2006) Acute ankle sprain: an update. Am Fam Physician 74(10):1714–1720
6. Willems TM, Witvrouw E, Delbaere K et al (2005) Intrinsic risk factors for inversion ankle sprains in male subjects a prospective study. Am J Sports Med 33(3):415–423
7. Kerkhoffs G, Struijs P, Marti R et al (2003) Functional treatments for acute ruptures of the lateral ankle ligament. Acta Orthop 74(1):69–77
8. Struijs PA, Kerkhoffs G (2006) Ankle sprain. Clin Evid 15: 1493–1501
9. van der Windt DA, van der Haijden GJ, van der Berg SG et al (2002) Ultrasound therapy for acute ankle sprains. Cochrane Database Syst Rev (1):CD001250
10. Manetti P, Dainelli S, Galanti G (2007) Smartherapy: supplying and subtracting thermal energy: first treatment trials on professional soccer players. In XVI Congresso Internazionale Isokinetic di Riabilitazione Sportiva e Traumatologia, Milano, pp 14–15
11. Weber T, Kabelka B (2012) Noninvasive monopolar capacitive-coupled radiofrequency for the treatment of pain associated with lateral elbow tendinopathies: 1-year follow-up. PM R 4(3):176–181
12. Karu TI, Piatibrat LV, Kalendo GS (1986) Radiation-modifying effect of UV and visible laser light. Radiobiologiia 27(6):804–809
13. Sun G, Tunér J (2004) Low-level laser therapy in dentistry. Dent Clin N Am 48(4):1061–1076
14. Clijsen R, Taeymans J, Clarys P et al (2007) Ist die sportphysiotherapeutische behandlung acuter inversionstraumata evidenzbasiert. Sportrverl Sportschad 21:71–76
15. Kerkhoffs GMMJ, Rowe BH, Assendelft WJ et al (2002) Immobilisation and functional treatment for acute lateral ankle ligament injuries in adults. Cochrane Database Syst Rev (3):CD003762
16. Kerkhoffs GM, Rowe BH, Assendelft WJ et al (2002) Immobilisation and functional treatment for acute lateral ankle ligament injuries in adults. Cochrane Database Syst Rev 3(3):CD002938
17. Norm A, Hanson B (eds) (1996) Aquatic exercise therapy. WB Sauders, Philadelphia
18. Ruoti RG, Morris DM, Cole AJ (eds) (1997) Aquatic rehabilitation. Lippincott Williams & Wilkins, Philadelphia
19. Riemann BL, Lephart SM (2002) The sensorimotor system, part I: the physiologic basis of functional joint stability. J Athl Train 37(1):71–79
20. Moruzzi G (1975) Physiology of relational life. Utet, Turin
21. Riva D, Mamo C, Fanì M et al (2013) Single stance stability and proprioceptive control in older adults living at home: gender and age differences. J Aging Res 2013:14
22. Riva D, Rossitto F, Battocchio L (2009) Postural muscle atrophy prevention and recovery and bone remodelling through high frequency proprioception for astronauts. Acta Astronaut 65(5): 813–819
23. Delos Postural Proprioceptive System. The devices to improve inner balance. Website: http://www.delos-international.com/prodotti.asp?sec=scheda&lang=eng
24. Germak A, Schiavi A, Mazzoleni F et al (2014) Metrological characterization of rocking boards and postural readers to assess single stance stability in human subjects. In: IMEKO 22ndTC3, 15thTC5 and 3rdTC22 international conferences, Cape Town
25. Roi GS, Nanni G, Tencone F (2006) Time to return to professional soccer matches after ACL reconstruction. Sport Sci Health 1(4):142–145

Viscosupplementation in Athletes

55

Vincenzo Salini, Andrea Pantalone, Daniele Vanni, and Michele Abate

55.1 Introduction

Besides acute musculoskeletal injuries due to traumatic events, chronic joint and tendon damage, due to overuse or repeated microtraumas, is common in athletes. The prevailing paradigm suggests that osteoarthritis (OA) results from the failure of the damaged cartilage repair process, due to biomechanical and biochemical changes in the joint, which follow, especially in athletes, repeated and stressful exercise and traumas [1]. Namely, OA is considered a disease of the whole joint involving subchondral bone changes with increased metabolism and sclerosis, chondrocyte death, and extracellular matrix catabolism, as well as primary or secondary changes in the synovium, including endothelial cell proliferation, macrophage infiltration, and inflammation with subsequent alterations in the molecular composition of the synovial fluid [1].

As far as tendons are concerned, it is well known that, in physiologic conditions, exercise has beneficial effects on tendon morphology and function [2]. Indeed, when the mechanical load is repeated and intense (as in athletes) but still in the physiologic window, anabolism prevails on catabolism. New extracellular matrix and collagen fibers are formed, so that, in the long run, tendon cross-sectional area increases, and the biomechanical properties are improved. However, when the individual threshold of loading frequency and magnitude is overcome, the tendon response reverses from beneficial toward degenerative [3]. An aberration in the proteoglycan metabolism is likely to drive the pathogenesis of tendon damage, with increased metalloproteinase expression, which favours the formation of degradation products. In addition, inflammatory molecules, such as interleukin1-beta, are released and may be implicated in the disease progression [4]. The chronic tendon damage is epidemiologically prevalent in comparison with acute damage (rupture), which can occur on tendons with evidence of degeneration or even in normal tendon when submitted to very intense exercise.

Quite all joints and tendons may be involved in athletes, some of them more frequently in relation to the peculiar sport activity: the shoulder in swimming, baseball, volleyball, and basketball, the elbow in tennis and golf, and the hip, knee, and ankle in sports requiring running and jumping. While the number of practitioners increases, the rate of sports injuries distressing the musculoskeletal system is growing and becoming a challenging problem. Indeed, these injuries can be the cause of a premature ending or of a long-standing stop of a professional career with ensuing economic damage or a limiting factor of leisure activities with a substantial impact on the quality of life.

The aim of the therapy is twofold: first, to get a physiologic and if possible complete healing process and, second, to shorten the recovery time, especially in professional athletes.

Several conservative treatments, such as oral and topical nonsteroidal anti-inflammatory drugs, glucosamine, chondroitin sulfate, and intra-articular or peritendinous corticosteroids, have been proposed as noninvasive solutions for pain treatment and improvement in function, with varying success rates. However, evidence suggests that they are not able to alter the natural history of the disease and some of them, while efficacious in the short term, may have deleterious local and systemic consequences [5]. With increasing understanding of cell signaling networks, current research is investigating new conservative methods seeking to provide an instructional environment for stimulating joint and tendon repair.

Among the emerging technologies for enhancing and accelerating tissue healing, a biocompatible and cost-effective approach involves the viscosupplementation with hyaluronic acid (HA). "Engineering" the synovial fluid with HA is a safe and effective procedure in the management of OA, as shown by a huge amount of clinical trials [1, 6], but

V. Salini • A. Pantalone • D. Vanni • M. Abate (✉)
Department of Medicine and Science of Aging,
University G. d'Annunzio Chieti-Pescara,
Via dei Vestini 31, Chieti Scalo 66013, Italy
e-mail: vsalini@unich.it; pantaloneandrea@libero.it;
danielevannimd@libero.it; m.abate@unich.it

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_55,

recently, encouraging results have been also reported in the treatment of tendon disorders [7].

The aim of this chapter is to show the current evidence about the use of HA in the treatment of joint and tendon injuries in athletes.

55.2 Rationale

Synovial fluid is essential for the normal joint functioning: it acts both as a lubricant during slow movement (e.g., in walking) and as an elastic shock absorber during rapid movement (e.g., in running). It also serves as a medium for delivering nutrition and transmitting cellular signals to articular cartilage [1]. HA is the major chemical component of synovial fluid. The native HA has a molecular weight of 4–10 million Daltons and is present in articular fluid in a concentration of about 0.35 g/100 ml [8]. It is essential for the viscoelastic properties of the fluid because of high viscosity and has a protective effect on articular cartilage and soft tissue surfaces of joints [9]. Furthermore, due to its abundant negative charges, HA absorbs a large amount of water at equilibrium, and this vast water domain helps to create the spaces through which the cells move and signaling molecules diffuse to reach their targets.

In pathological conditions, the concentration and molecular weight of HA are reduced, resulting in synovial fluid of lower elasticity and viscosity: the factors which contribute to its low concentrations are dilutional effects, reduced hyaluronan synthesis, and free radical degradation [9]. When viscoelasticity of synovial fluid is reduced, the transmission of mechanical force to cartilage may increase its susceptibility to mechanical damage. Therefore, the restoration of the normal articular homeostasis is the rationale for HA administration into OA joints.

The conceptual basis for the use of HA in the treatment of tendon disorders comes from physiopathologic considerations [4, 7]: first, because HA is an essential component of the tendon itself and, second, because sound experimental data show that an intense HA synthesis occurs during the healing phase after damage.

55.3 Therapeutic Activities of HA

The beneficial effects of HA are due to complex mechanisms [1]. Besides the restoration of the viscoelastic properties of the synovial fluid, basic research has shown that HA has several pleiotropic signaling properties [1, 10].

Actually, HA binds to a number of cell membrane receptors termed hyaladherins. The predominant and more widely expressed is CD44, which is provided of protective effects on tissue remodeling and damage progression [11]. Moreover, HA is provided of anti-inflammatory and antinociceptive activities and contributes to the normalization of endogenous HA synthesis. The analgesic properties of HA are attributed to a specific activity on opiod receptors [12]. In particular, it has stimulatory effects on the k receptors in a concentration-dependent manner, thus increasing the pain threshold by a direct action on synovial nerve endings.

In OA, the efficacy in the short term is mainly due to a substitution effect (viscosupplementation), i.e., to the restoration of the viscoelastic properties of the synovial fluid, such as cushioning, lubrication, and elasticity. On the contrary, the long-term effects are mainly dependent on the restoration of joint rheology (biosupplementation), i.e., the anti-inflammatory and antinociceptive activities, the normalization of endogenous HA synthesis, and chondroprotection [1]. The efficacy on tendon disorders can be ascribed to the improvement of the healing process and to the lubricating effect on the tendon sheaths, which has been clearly shown on hand flexors [7].

55.4 HA Preparations

At present, preparations with different molecular weight are commercially available. The enhanced penetration of low molecular weight (LMW) preparations (0.5–1.5 million Daltons) through the extracellular matrix of the synovium is thought to facilitate the interaction with target synovial cells, thus reducing the synovial inflammation [13]. HA preparations with high molecular weight (HMW) (6–7 million Daltons) [14], by means of their hydrophilic properties, retain higher amounts of fluid in the articular space and are also provided by anti-inflammatory activity, as shown by studies on migration of inflammatory cells in the joint and on reduced prostaglandin E2 and bradykinin concentration [15].

Recently, new preparations have been proposed, aiming to ameliorate the therapeutic efficacy of HA. New types of particulate carriers have been investigated for increasing the retention time of therapeutic agents within the joint cavity, among them, cationic polymeric nanoparticles that form diffuse ionically associated filamentous structures ("ionically cross-linked hydrogels") with resident hyaluronate in the synovial cavity after intra-articular injection [16]. Moreover, a preparation of celecoxib-loaded liposomes embedded in HA gel has been formulated [17]. The combination of these drugs, both efficient in the treatment of OA but with different mechanisms, injected into the joints, is expected to have synergistic effect, as preliminarily shown in animal models [18]. However, despite these promising results, high-quality clinical studies proving the superiority of new formulations toward the available preparations of HA are still lacking.

55.5 Clinical Trials

Information about the efficacy of HA in the treatment of joint and tendon pathologies in athletes comes in part from studies specifically performed in subjects, both professional and

amateurs, practicing sport activities and largely from trials, where young subjects with overuse and/or post-traumatic joint and tendon damage were included.

55.5.1 Knee

Viscosupplementation with HA in knee OA has been approved by the Food and Drugs Administration [19] and is recommended by OARSI for non-severe OA [20]. Guidelines are based on a meta-analysis of randomized saline-controlled trials, including a total of 29 studies representing 4.866 subjects (intra-articular HA, 2,673; saline, 2,193) [21, 22]. These trials were performed, single or double blind, with different types of HA (LMW and HMW). The number of injections ranged from 3 to 5 weekly, the doses from 15 to 60 mg, and the trials' length from 4 weeks to 18 months. HA injection resulted in very large treatment effects between 4 and 26 weeks for knee pain and function compared to preinjection values. The percentages of improvement from baseline were similar in the trials where LMW or HMW HA was used [21, 23]. However, the number of injections needed was in general lower for HMW preparation, and this is an advantage for patients and doctors [21].

The benefit of intra-articular HA injection has been also demonstrated in younger patients with acute knee damage, including symptomatic meniscal tears and isolated ACL injury with chondral injury [1, 24].

In athletic patients with patellar tendinopathy (stage 2 or 3 according to Blazina's classification), a mixture of 25 mg HA and 1 ml of 1 % lidocaine was injected blindly at the proximal interface between the posterior surface of the patellar tendon and the infrapatellar fat pad or into the region of maximum tenderness [25]. One week after the first injection, other injections were done on the patient's request; conservative treatments (exercises and instrumental therapies) were also prescribed. After treatment, 54 % of patients were rated in excellent conditions (return to previous athletic activities), while 40 % in good conditions complained some degree of limitation. The open design and the subjective evaluation methods used (no imaging) are important limitations of this study.

55.5.2 Hip

The experience about viscosupplementation of hip OA in athletes is scanty, because the modest epidemiological relevance of the disease in these subjects and in young patients in general. Therefore, the efficacy of the treatment cannot be definitely proved. Some trials have shown a reduction of pain, which, in general, becomes evident within 3 months and persists in the following months. However, it must be underlined that only few studies report longer follow-up periods (at 12 and 18 months) [26].

55.5.3 Ankle

The experience on ankle OA is limited. Positive results have been reported in studies performed without a control group, but it is not clear whether the pain reduction could be ascribed only to a placebo effect [27].

In other studies, HA injections have shown similar efficacy when compared to a 6-week exercise therapy (muscle strengthening and ankle range of motion exercises) [28] or arthroscopic lavage [29]. The limited efficacy of viscosupplementation in ankle OA can be partly explained by the fact that all studies, but one [27], were performed blindly, with any imaging guidance.

Ankle sprains are among the most common of all sports injuries, and their impact is of considerable concern to long-term function and performance of athletes beyond the acute event. At this regard, it is noteworthy a randomized, controlled, prospective trial, performed on 158 competitive athletes who suffered an acute grade I or II lateral ankle sprain [30]. The patients were randomly assigned at baseline (within 48 h of injury) to periarticular injection with HA and standard of care (rest, ice, elevation, and compression [RICE]) or placebo injection (PL) and RICE treatment. Periarticular HA treatment was highly satisfactory in the short and long term versus PL, being associated to reduced pain, more rapid return to sport activities, and fewer missed days from sport in a 24-month follow-up [30].

55.5.4 First Metatarsophalangeal Joint

First metatarsophalangeal joint OA is common in golfers and can lead to progressive reduction in range of motion and pain that can affect walking and recreational activity. A study performed on 47 older golfers (mean age 70 years), treated with a weekly intra-articular injection of 1 ml HA for 8 weeks, showed at week 9 a significant improvement in pain at rest and after tiptoe walking and an increased range of motion. These changes were maintained for all measures at 16 weeks [31].

55.5.5 Shoulder

HA is effective and well tolerated for the treatment of OA and persistent shoulder pain refractory to other standard interventions. In addition, most of the patients experience an improvement in the shoulder function score and in the activities of daily living [32, 33]. The efficacy of HA has been also demonstrated in the treatment of other shoulder diseases, such as subacromial bursitis and adhesive capsulitis [34]. The experience of HA use in rotator cuff tendon damage is more consistent [35, 36]. Patients with different rotator cuff diseases (full- or partial-thickness tear, tendinosis) were treated with HA injections and compared to placebo or active

treatments (steroids or physical therapies). A superior therapeutic effect was observed in comparison to placebo, but no significant difference was shown when steroids and physical therapy were used as controls. It can be supposed that in these conditions, HA helps in improving the articular and periarticular environment, because only surgery may be efficacious on tears.

55.5.6 Elbow

Tennis elbow, also called lateral epicondylitis, is characterized by degenerative changes related to overuse injury and repetitive stress, leading to microtearing and progressive degeneration of the common extensor origin at the attachment to the lateral epicondyle. During the past 10 years, several injection techniques have become available with positive results [37]. As far as HA is concerned, an important study, involving 341 competitive tennis athletes, has been performed. The patients were assigned randomly to HA and placebo group (treated with saline). Two injections (at baseline and a week later) were done blindly into the subcutaneous tissue and muscle, 1 cm from the lateral epicondyle toward the primary point of pain, using a two-dimensional fanning technique. Periarticular HA treatment was significantly better than control, after 1, 3, and 12 months, in improving pain at rest and after maximal grip testing. Moreover, HA treatment was deemed highly satisfactory by patients and physicians and resulted in better return to pain-free sport compared to controls [38]. Unfortunately, comparisons of HA with other available therapies (corticosteroids, platelet-rich plasma, prolotherapy, etc.) are still lacking [37].

Conclusions

On the basis of the published trials, viscosupplementation therapy with HA may be considered a safe and effective method in the management of OA occurring in athletes. The efficacy seems more limited in tendon disorders, and new trials are needed for a better selection of patients who can benefit from the treatment. This conclusion derives from studies specifically performed in subjects, both professional and amateurs, practicing sport activities, and indirectly from trials, where young subjects with overuse and/or post-traumatic joint and tendon damage were included.

Patients with mild morphological alterations and with preserved articular space are more responsive to treatment [39], while the results are less encouraging in patients with severe OA (Kellgren-Lawrence [K-L] IV). This is indirectly confirmed by the evolution of the serum levels of specific OA biomarkers (Coll2-1 and Coll2-1 NO2) after viscosupplementation. Indeed, the serum concentrations of the biomarkers are significantly higher in K-L III/IV patients compared to K-L I/II patients and significantly lower at baseline in responders than in nonresponders [40].

The published data suggest that intra-articular joint HA injections are more effective in younger patients, especially in the long term, whereas the results are less promising in older athletes.

The use of HA can be recommended as first-line therapy, or when corticosteroids and nonsteroidal anti-inflammatory drugs are inefficacious, contraindicated or badly tolerated. Viscosupplementation significantly reduces pain within 3 months, and this beneficial effect is maintained in the long term (12–18 months). The articular function improves, and, therefore, patients can rapidly come back to sports activities. Only a few trials have shown a very early improvement, which has been related to the lubricating effect of hyaluronate in "dry" joints, as reported in studies of viscosupplementation in knee OA, and/or to a short-term placebo effect [41].

The biological activity, shown by LMW and HMW HA preparations, is similar. However, an advantage of HMW HA may be the reduced number of the injections needed to obtain the therapeutic effect.

When the therapy is delivered by appropriately trained doctors, viscosupplementation is a safe procedure, without any systemic or local side effect, excluding the possible pain of the injection and a sensation of heaviness for a few hours/days after treatment. It is likely that adverse reactions may occur when injection is not properly performed. Indeed, they are more frequent in studies performed in blind conditions compared to those performed under imaging guidance. Vascular or nervous complications were never reported, and septic arthritis or aseptic synovial effusion occurred in a very limited number of cases.

References

1. Axe JM, Snyder-Mackler L, Axe MJ (2013) The role of viscosupplementation. Sports Med Arthrosc 21(1):18–22
2. Abate M, Oliva F, Schiavone C, Salini V (2012) Achilles tendinopathy in amateur runners: role of adiposity (Tendinopathies and obesity). Muscles Ligaments Tendons J 2(1):44–48
3. Thornton GM, Hart DA (2011) The interface of mechanical loading and biological variables as they pertain to the development of tendinosis. J Musculoskelet Neuronal Interact 11(2):94–105
4. Abate M, Silbernagel KG, Siljeholm C, Di Iorio A, De Amicis D, Salini V, Werner S, Paganelli R (2009) Pathogenesis of tendinopathies: inflammation or degeneration? Arthritis Res Ther 11(3):235
5. Briggs KK, Matheny LM, Steadman JR (2012) Can Hylan G-F 20 with corticosteroid meet the expectations of osteoarthritis patients? Am J Orthop (Belle Mead NJ) 41(7):311–315
6. Abate M, Pulcini D, Di Iorio A, Schiavone C (2010) Viscosupplementation with intra-articular hyaluronic acid for treatment of osteoarthritis in the elderly. Curr Pharm Des 16(6): 631–640

7. Abate M, Schiavone C, Salini V (2014) The use of hyaluronic acid after tendon surgery and in tendinopathies. Biomed Res Int 2014:783632
8. Weiss C, Band P (1995) Musculoskeletal applications of hyaluronan and hylan. Potential uses in the foot and ankle. Clin Podiatr Med Surg 12(3):497–517
9. van den Bekerom MP, Rys B, Mulier M (2008) Viscosupplementation in the hip: evaluation of hyaluronic acid formulations. Arch Orthop Trauma Surg 128(3):275–280
10. Andia I, Abate M (2014) Knee osteoarthritis: hyaluronic acid, platelet-rich plasma or both in association? Expert Opin Biol Ther 14(5):635–649
11. Li J, Gorski DJ, Anemaet W, Velasco J, Takeuchi J, Sandy JD, Plaas A (2012) Hyaluronan injection in murine osteoarthritis prevents TGFbeta 1-induced synovial neovascularization and fibrosis and maintains articular cartilage integrity by a CD44-dependent mechanism. Arthritis Res Ther 14(3):R151
12. Zavan B, Ferroni L, Giorgi C, Calò G, Brun P, Cortivo R, Abatangelo G, Pinton P (2013) Hyaluronic acid induces activation of the κ-opioid receptor. PLoS One 8(1):e55510
13. Bagga H, Burkhardt D, Sambrook P, March L (2006) Longterm effects of intraarticular hyaluronan on synovial fluid in osteoarthritis of the knee. J Rheumatol 33(5):946–950
14. Migliore A, Giovannangeli F, Granata M, Laganà B (2010) Hylan g-f 20: review of its safety and efficacy in the management of joint pain in osteoarthritis. Clin Med Insights Arthritis Musculoskelet Disord 3:55–68
15. Waddell DD (2007) Viscosupplementation with hyaluronans for osteoarthritis of the knee: clinical efficacy and economic implications. Drugs Aging 24(8):629–642
16. Morgen M, Tung D, Boras B, Miller W, Malfait AM, Tortorella M (2013) Nanoparticles for improved local retention after intra-articular injection into the knee joint. Pharm Res 30(1):257–268
17. Dong J, Jiang D, Wang Z, Wu G, Miao L, Huang L (2013) Intra-articular delivery of liposomal celecoxib-hyaluronate combination for the treatment of osteoarthritis in rabbit model. Int J Pharm 441(1–2):285–290
18. Chen B, Miller RJ, Dhal PK (2014) Hyaluronic acid-based drug conjugates: state-of-the-art and perspectives. J Biomed Nanotechnol 10(1):4–16
19. Hunter DJ, Lo GH (2008) The management of osteoarthritis: an overview and call to appropriate conservative treatment. Rheum Dis Clin North Am 34(3):689–712
20. Gallagher B, Tjoumakaris FP, Harwood MI, Good RP, Ciccotti MG, Freedman KB (2014) Chondroprotection and the prevention of osteoarthritis progression of the knee: a systematic review of treatment agents. Am J Sports Med 27
21. Colen S, van den Bekerom MP, Mulier M, Haverkamp D (2012) Hyaluronic acid in the treatment of knee osteoarthritis: a systematic review and meta-analysis with emphasis on the efficacy of different products. BioDrugs 26(4):257–268
22. Miller LE, Block JE (2013) US-approved intra-articular hyaluronic acid injections are safe and effective in patients with knee osteoarthritis: systematic review and meta-analysis of randomized, saline-controlled trials. Clin Med Insights Arthritis Musculoskelet Disord 6:57–63
23. Uçar D, Dıraçoğlu D, Süleyman T, Capan N (2013) Intra-articular hyaluronic acid as treatment in elderly and middle-aged patients with knee osteoarthritis. Open Rheumatol J 7:38–41
24. Huang MH, Yang RC, Chou PH (2007) Preliminary effects of hyaluronic acid on early rehabilitation of patients with isolated anterior cruciate ligament reconstruction. Clin J Sport Med 17(4):242–250
25. Muneta T, Koga H, Ju YJ, Mochizuki T, Sekiya I (2012) Hyaluronan injection therapy for athletic patients with patellar tendinopathy. J Orthop Sci 17(4):425–431
26. Abate M, Pelotti P, De Amicis D, Di Iorio A, Galletti S, Salini V (2008) Viscosupplementation with hyaluronic acid in hip osteoarthritis (a review). Ups J Med Sci 113(3):261–277
27. Abate M, Schiavone C, Salini V (2012) Hyaluronic acid in ankle osteoarthritis: why evidence of efficacy is still lacking? Clin Exp Rheumatol 30(2):277–281
28. Karatosun V, Unver B, Ozden A, Ozay Z, Gunal I (2008) Intra-articular hyaluronic acid compared to exercise therapy in osteoarthritis of the ankle. A prospective randomized trial with long-term follow-up. Clin Exp Rheumatol 26(2):288–294
29. Carpenter B, Motley T (2008) The role of viscosupplementation in the ankle using hylan G-F 20. J Foot Ankle Surg 47(5):377–384
30. Petrella MJ, Cogliano A, Petrella RJ (2009) Original research: long-term efficacy and safety of periarticular hyaluronic acid in acute ankle sprain. Phys Sportsmed 37(1):64–70
31. Petrella RJ, Cogliano A (2004) Intra-articular hyaluronic acid treatment for golfer's toe: keeping older golfers on course. Phys Sportsmed 32(7):41–45
32. Merolla G, Paladini P, Saporito M, Porcellini G (2011) Conservative management of rotator cuff tears: literature review and proposal for a prognostic. Prediction Score. Muscles Ligaments Tendons J 1(1):12–19
33. Merolla G, Sperling JW, Paladini P, Porcellini G (2011) Efficacy of Hylan G-F 20 versus 6-methylprednisolone acetate in painful shoulder osteoarthritis: a retrospective controlled trial. Musculoskelet Surg 95(3):215–224
34. Gross C, Dhawan A, Harwood D, Gochanour E, Romeo A (2013) Glenohumeral joint injections: a review. Sports Health 5(2):153–159
35. Ozgen M, Fırat S, Sarsan A, Topuz O, Ardıç F, Baydemir C (2012) Short- and long-term results of clinical effectiveness of sodium hyaluronate injection in supraspinatus tendinitis. Rheumatol Int 32(1):137–144
36. Merolla G, Bianchi P, Porcellini G (2013) Ultrasound-guided subacromial injections of sodium hyaluronate for the management of rotator cuff tendinopathy: a prospective comparative study with rehabilitation therapy. Musculoskelet Surg 97(Suppl 1):49–56
37. Krogh TP, Bartels EM, Ellingsen T, Stengaard-Pedersen K, Buchbinder R, Fredberg U, Bliddal H, Christensen R (2013) Comparative effectiveness of injection therapies in lateral epicondylitis: a systematic review and network meta-analysis of randomized controlled trials. Am J Sports Med 41(6):1435–1446
38. Petrella RJ, Cogliano A, Decaria J, Mohamed N, Lee R (2010) Management of Tennis Elbow with sodium hyaluronate periarticular injections. Sports Med Arthrosc Rehabil Ther Technol 2:4
39. Gaston MS, Tiemessen CH, Philips JE (2007) Intra-articular hip viscosupplementation with synthetic hyaluronic acid for osteoarthritis: efficacy, safety and relation to pre-injection radiographs. Arch Orthop Trauma Surg 127(10):899–903
40. Henrotin Y, Chevalier X, Deberg M, Balblanc JC, Richette P, Mulleman D, Maillet B, Rannou F, Piroth C, Mathieu P, Conrozier T (2013) Osteoarthritis Group of French Society of Rheumatology. Early decrease of serum biomarkers of type II collagen degradation (Coll2-1) and joint inflammation (Coll2-1 NO_2) by hyaluronic acid intra-articular injections in patients with knee osteoarthritis: a research study part of the Biovisco study. J Orthop Res 31(6):901–907
41. Brocq O, Tran G, Breuil V, Grisot C, Flory P, Euller-Ziegler L (2002) Hip osteoarthritis: short-term efficacy and safety of viscosupplementation by hylan G-F 20. An open-label study in 22 patients. Joint Bone Spine 69(4):388–391

Use of PRP in Sports Medicine

56

Pietro Simone Randelli, Chiara Fossati, Alessandra Menon, Vincenza Ragone, Riccardo D'Ambrosi, Paolo Cabitza, and Laura De Girolamo

Abbreviations

ACL	Anterior cruciate ligament
ACS	Autologous conditioned serum
AOFAS score	American Orthopaedic Foot and Ankle Society score
DASH score	Disabilities of the Arm, Shoulder and Hand score
MRI	Magnetic resonance imaging
PRP	Platelet-rich plasma
RC	Rotator cuff
VAS score	Visual analog scale score
VISA-A	Victorian Institute of Sport Assessment-Achilles questionnaire
VISA-P	Victorian Institute of Sport Assessment for patellar tendinopathy questionnaire
WADA	World Anti-Doping Agency

P.S. Randelli, MD (✉) • P. Cabitza
Dipartimento di Scienze Biomediche per la salute, Università degli Studi di Milano, Via Morandi 30, San Donato Milanese 20097, Italy
e-mail: pietro.randelli@unimi.it

C. Fossati • A. Menon, PhD • V. Ragone, MEng
R. D'Ambrosi
Unità Operativa Complessa Ortopedia II, IRCCS Policlinico San Donato, IRCCS Policlinico San Donato, Via Morandi 30, San Donato Milanese 20097, Italy
e-mail: chiara.fossati@hotmail.com; ale.menon@me.com; ragone.vincenza@inwind.it; riccardo.dambrosi@hotmail.it; paolo.cabitza@unimi.it

L. De Girolamo
Laboratorio di Biotecnologie applicate all'Ortopedia, IRCCS Istituto Ortopedico Galeazzi, Via Riccardo Galeazzi, 4, Milan 20161, Italy
e-mail: Laura.degirolamo@grupposandonato.it

56.1 Introduction

Musculoskeletal injuries are one of the most common causes of severe long-term pain and physical disability in sports medicine [1]. Healing of these injuries is often too slow and sometimes incomplete, decreasing performance of athlete and long-standing pain and discomfort [2]. The progressive understanding of mechanisms required for successful tissue repair has set the basis for the possibility of making injured tissues heal faster. Among the emerging technologies for enhancing and accelerating tissue healing, one of the most popular methods in the fields of orthopedic surgery and sports medicine includes the use of autologous blood products, particularly platelet-rich plasma (PRP). PRP is an autologous concentration of human platelets to supraphysiologic levels. It is produced from a patient's peripheral vein and centrifuged to achieve a high concentration of platelets within a small volume of plasma. It is then injected at a site of injury or inserted as a gel or other biomaterials during surgery [3]. Because of its autogenous origin, easy preparation, excellent safety profile, and reduced cost of platelet-based preparations, the advent of PRP is highly attractive for sports medicine where a fast recovery and return to competitions are often critical outcomes in patient care [4]. In the following paragraphs are presented some of the most interesting current approaches in the treatment of acute and chronic sports injuries.

56.2 Tendinopathy

56.2.1 Elbow Tendinopathy

Epicondylar tendinosis is frequent in athletes who perform repetitive wrist motions and strong gripping. PRP has advantages in the treatment of tennis elbow as demonstrated for the first time by Mishra and Pavelko in 2006. In this cohort study, the authors reported a clinical decrease of pain after PRP treatment in 15 patients having severe chronic

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_56,

tendinopathy of the elbow. The test group was compared to a control group of five patients receiving local anesthesia only. The patients were observed for 24 months. After 8 weeks of treatment, they noticed a 60 % improvement in pain scores for patients treated with PRP compared to 16 % improvement in control patients. At final follow-up, the PRP group reported 93 % reduction in pain. Although this study lacked a complete randomization and presented a reduced number of patients, the long follow-up and the absence of complications showed an excellent safety profile for PRP as a potential treatment option for sports-related injuries [5]. Perbooms et al. performed a double-blind randomized controlled trial of 100 patients undergoing lateral epicondylitis treatment with PRP (51 patients) or with corticosteroid (49 patients). They observed that a single injection of PRP significantly reduced pain (visual analog scale (VAS) score) and improved function (Disabilities of the Arm, Shoulder and Hand (DASH) score) at 6 months and 1 year follow-up better than corticosteroid treatment and without any complications [6]. Creaney and colleagues most recently published results of a prospective single-blind randomized trial on the efficacy of two types of blood injections. A group of 150 patients with elbow symptoms resistant to conventional treatment received either PRP injections or autologous conditioned plasma. The injections were repeated twice and both groups were compared. Unfortunately, there was no placebo control group. In both groups, they reported marked reduction in pain and increase in function but without any significant difference between groups after 6 months of follow-up [7].

56.2.2 Achilles Tendinopathy

Overuse injury of the Achilles tendon is a frequent problem that often affects sportsmen but also sedentary middle-aged individuals. Patients with Achilles tendinopathy who have failed physical therapy and multiple modalities of conservative treatment are candidates for PRP injections.

DeVos and colleagues were the first to use the PRP in the treatment of the Achilles tendon. In this randomized controlled double-blind study, 54 patients with mid-portion Achilles tendinosis were randomized to receive either injection with PRP or with saline associated with eccentric gymnastic exercises. As a result of the treatment, pain decreased, and functional scores, such as Victorian Institute of Sport Assessment-Achilles questionnaire (VISA-A), improved significantly in both groups. Even if results in the PRP group were somewhat higher, no statistical significance was found after 6 months between the two groups. There was also no difference in patient satisfaction or in the time taken to return to sport. Importantly, no relevant side effects were identified [8]. The same investigators subsequently extended the follow-up to 1 year, without finding any supplementary PRP benefit. Localized bleeding caused by the injecting syringe might have triggered the tissue healing process. A positive response could also be due to a placebo effect, as invasive procedures lead to higher expectancy of good results. Finally, in both groups, ultrasound showed that tendon structure and neovascularization improved significantly, without any group differences after 6 and 12 months. One of the main reasons for the absence of group differences is certainly the fact that standardized eccentric exercise training has already been shown to improve actual tendon structure [9]. Monto et al. treated 30 patients with Achilles tendinopathy who had failed 8 months of conservative treatment and physical therapy. The treatment involved the injection of PRP under ultrasound guidance. The pretreatment American Orthopaedic Foot and Ankle Society (AOFAS) score averaged 34, indicating significant pathology. AOFAS score increased from an average of 34 to 92 after 6 months of treatment. Ninety-three percent of patients were fully satisfied [10]. Gaweda and colleagues conducted a prospective case series on the efficacy of use of PRP injections in the treatment of a non-insertional Achilles tendinopathy. The authors showed that a single injection of PRP, under ultrasound guidance, determined a significant improvement of AOFAS and VISA-A scores in 14 patients (15 Achilles tendons) for up to 18 months [11]. Sanchez et al. showed a shorter time in the recovery of motion and return to sporting activities in athletes undergoing Achilles tendon surgical repair with augmentation of PRP than the control group. Controls were treated with an identical surgical procedure performed by the same surgeon, but they did not receive PRP during surgery. A fibrin scaffold was used in addition to PRP. The authors observed no wound complication and a faster return to jogging and training activities [12].

56.2.3 Patellar Tendinopathy

Jumper's knee (patellar tendinopathy) is common in athletes of sport disciplines where jumping is frequent, e.g., basketball, soccer, and volleyball. This pathology is characterized by angiofibroblastic hyperplastic changes within the substance of the tendon, which is typically located at the bone–tendon junction of the inferior pole of the patella.

Volpi and colleagues were the first to introduce a PRP injection to accelerate healing in chronic patellar tendinopathy of eight professional athletes. Participants, recalcitrant to conservative measures, received a single injection of PRP and were observed for 4 months, but only seven patients received a follow-up examination. The authors found a significant improvement of the VISA scores after the infiltration of PRP under ultrasound guidance. Furthermore, the nuclear magnetic resonance imaging (MRI) showed a reduction of

irregularities in the tendon [13]. In a recent report, Kon et al. reported similar results in a case series for the treatment of 20 patients who experienced recurrent patellar tendinopathy symptoms, over a 20-month period. Participants received three PRP injections within 1 month, showing statistically significant improvement in pain and physical function at 6 months follow-up [14]. Filardo et al. had good results with the use of PRP (type 1) for chronic patellar tendinopathy in 15 athletes [15]. Recently, a prospective study evaluated the influence of previous treatments on the effectiveness of PRP injections in 36 patients with chronic patellar tendinopathy. Assessment was done before and after injection of PRP using the Victorian Institute of Sport Assessment for patellar tendinopathy (VISA-P) questionnaire and VAS score. The first group (14 patients) had been treated with cortisone, ethoxysclerol, and/or surgical treatment before the injection, while the second group (22 subjects) had not received such treatments. A statistically significant improvement in both groups at 18 months was found but larger in the group without previous treatments. Thus, this study opens the question about a relationship between prior treatment and efficacy of PRP injections [16].

56.3 Rotator Cuff Tears

Rotator cuff (RC) injuries are a common source of shoulder pathology and result in an important decrease in quality of patient life. These tears mainly occur among individuals who constantly participate in overhead activities, such as swimming, baseball, football, tennis, or racquetball. Given the frequency of these injuries due to the population age increase, as well as the relatively poor result of surgical intervention, the use of PRP to improve RC tendon healing and clinical outcome has become more appealing over the last several years [17, 18]. Although conflicting results on the effectiveness of PRP use in RC tendon repair surgery were produced, making it now difficult to draw definitive conclusions, literature data suggest a beneficial effect on healing of arthroscopically repaired small and medium RC lesions (retear rate 7.9 % among patients treated with PRP, compared to 26.8 % of those treated without PRP) [19]. Furthermore, no complications have been reported from surgical use of PRP, with the exception of two cases of infection [20]. Therefore, it currently seems that PRP may improve healing of arthroscopically repaired small and medium RC lesions, which appear more prone to a biological response to treatment with growth factors [21–25].

In spite of this popularity and increasing use in clinical settings, we have found only two randomized controlled trials evaluating the use of PRP injections in RC tendinopathy reporting conflicting results [26, 27]. Further prospective randomized controlled trials (level 1 evidence) are necessary to define the role of PRP in healing process when applied during RC arthroscopic repair and of PRP injections in the subacromial space for treatment of RC tendinopathies.

56.4 Ligament Injuries

56.4.1 Anterior Cruciate Ligament

Anterior cruciate ligament (ACL) injuries are usually traumatic and sports related. Success rates of ACL reconstruction surgery vary between 73 and 95 %, and the rate of return to the preinjury level of activity varies from 37 to 75 % [28]. There has been increasing interest in improving these results, especially in the presence of graft failures. By acceleration of the biological integration of the graft by the use of PRP, patients could potentially advance through more intensive rehabilitation programs and return to sports more rapidly than patients treated with traditional surgical protocols.

In general, studies on ACL reconstruction focus more on the osteo-integration of ACL grafts and less on the graft itself. There have been a number of conflicting studies in humans; however, differences in techniques, type of PRP, and outcomes make direct comparison difficult. Radice et al., in a prospective single-blinded MRI study, have recently shown a 48 % reduction in the time needed to achieve a complete homogeneous graft signal, in 100 patients undergoing surgical ACL reconstruction with addition of PRP versus controls [29]. Also a case-control study indicated that the addition of PRP to ACL reconstruction led to improved graft remodeling and resulted in more mature graft tissue, based on arthroscopic and histologic evaluation [30]. Orrego and colleagues have also obtained similar results at 6 months of follow-up in patients treated with PRP. The authors reported a significant mature graft signal (100 %) in MRI from PRP group with respect to the control group, but did not find statistical differences in the osteoligamentous interface [31]. Not all studies, however, support the use of PRP to augment ACL healing. Silva et al. reported no differences in graft appearance at 3 months between patients treated with PRP and the control group. This study, however, did not use any scaffold [32]. Nin et al., in a prospective randomized double-blind study, did not show statistically significant difference in clinical and inflammatory parameters in patients treated with the addition of PRP in primary ACL reconstruction with allograft at 2 years of follow-up [33].

56.5 Muscle Injuries

Muscle strain and contusion injuries are common in sports and result in time loss from training and competition. However, despite advances in rehabilitation programs,

re-injury rates remain high. Historically, the management of muscle injuries has involved the use of various stretching and strengthening regimes underpinned by a graduated return to activity and subsequent return to sporting competition. These management strategies lack sound scientific support. Since the rapid return to functional activity and minimization of recurrence is the goal of any management intervention, PRP therapies represent a valid alternative to conventional approaches, because of the promise of accelerating muscle healing and reducing a player's injury time. Statistical power to support clinical evidence in humans is generally weak, due to the lack of methodologically robust studies. In effect, no randomized controlled studies conducted in humans could be identified, and only three other studies were performed on small samples with only few valuable controls.

The first description in humans did not strictly use PRP but rather autologous conditioned serum (ACS). Eighteen professional athletes suffering from muscle strains were treated by ACS muscle injection and compared with 11 athletes with similar injuries and previously treated by Actovegin or Traumeel injections. The study reported a significant reduction in return-to-field days (16 versus 22) but presented numerous limitations: small sample size, non-blinded, atypical controls, variable injury sites, and absence of long-term follow-up [34]. Two other case reports showed the return to sporting activities only 3–4 weeks after injury [35, 36]. Only one report evaluating PRP for muscle injuries is currently available. Sanchez et al. prospectively evaluated ultrasound-guided injections of PRP in 22 muscle injuries in 20 high-level professional athletes. The authors reported full functional recovery in all patients in half the time expected, and no fibrosis was seen [30].

56.6 PRP and Doping

The use of PRP in elite athletes has been discussed for a long time due to its content of growth factors that may be considered doping violation. The World Anti-Doping Agency's (WADA) mission is to lead a collaborative worldwide campaign for doping-free sport. Every year, WADA creates a list of substances and practices which are prohibited for use in competition, out of competition, and in particular sports. According to the World Anti-Doping Code, a substance or method is considered "doping" when two out of three criteria are fulfilled: (1) potential for performance enhancement, (2) risks to health, and (3) violates the spirit of sport. In 2010, PRP was specifically mentioned in the prohibited list for the first time and intramuscular PRP injections were prohibited. All other ways of administration, such as intra-articular and intra- or peritendinous, were permitted and required only a declaration of use. Growth factors are permitted only when part of platelet-derived preparations from the centrifugation of autologous whole blood. There was a concern by the WADA List Expert Group that growth factors contained in PRP may stimulate muscle satellite cells and increase muscular size and strength (beyond normal healing). However, the different PRP formulations and treatment methodologies, as they exist now, have not been found to increase muscle growth beyond return to a normal physiological state. There is a suggestion, but no compelling evidence, of systemic effects. The risk of adverse reactions (fibrosis, infection, carcinogenesis) is theoretical and has not been documented clinically. Hence, because the use of PRP injections for therapeutic purposes does not violate the spirit of sport, the prohibition for intramuscular injection of PRP has been removed from the 2011 prohibited list. This was an important issue to render the athletes treated with PRP therapy eligible for athletic participation.

Conclusion

The advent of regenerative medicine, aiming to rapidly translate the science into patient care using patients' own resources, has opened the door to new approaches. The biosafety and versatility of PRP approach has inspired and stimulated its therapeutic use in a wide range of medical and scientific fields and to an outstanding degree in orthopedics and sports medicine. Many studies assessed the effect of PRP on the musculoskeletal system during the last decade. Further studies are required to delineate correct dosage, timing, and quantification, as well as ideal techniques of PRP application, to understand the effect of PRP on the regenerative and healing processes of a given soft tissue and therefore support the use of PRP in clinical practice. However, PRP should not be considered first-line treatment. It takes 4–6 months to synthesize new tendon, so in sports settings, PRP should not to be considered an "in-season" treatment for chronic tendinopathy [37]. PRP and the field of regenerative treatments may serve to fortify the arsenal of nonoperative management of sports injuries, as well as have a role as an adjunct to improve postoperative healing.

References

1. Woolf AD, Pfleger B (2003) Burden of major musculoskeletal conditions. Bull World Health Organ 81(9):646–656
2. Cassell EP, Finch CF, Stathakis VZ (2003) Epidemiology of medically treated sport and active recreation injuries in the Latrobe Valley, Victoria, Australia. Br J Sports Med 37(5):405–409
3. Middleton KK, Barro V, Muller B, Terada S, Fu FH (2012) Evaluation of the effects of platelet-rich plasma (PRP) therapy involved in the healing of sports-related soft tissue injuries. Iowa Orthop J 32:150–163
4. Stanco D, Vigano M, Croiset SJ, De Girolamo L (2012) Applications and limits of platelet-rich plasma in sports related injuries. J Biol Regul Homeost Agents 26(2 Suppl 1):53s–61s

5. Mishra A, Pavelko T (2006) Treatment of chronic elbow tendinosis with buffered platelet-rich plasma. Am J Sports Med 34(11): 1774–1778
6. Peerbooms JC, Sluimer J, Bruijn DJ, Gosens T (2010) Positive effect of an autologous platelet concentrate in lateral epicondylitis in a double-blind randomized controlled trial: platelet-rich plasma versus corticosteroid injection with a 1-year follow-up. Am J Sports Med 38(2):255–262
7. Creaney L, Wallace A, Curtis M, Connell D (2011) Growth factor-based therapies provide additional benefit beyond physical therapy in resistant elbow tendinopathy: a prospective, single-blind, randomised trial of autologous blood injections versus platelet-rich plasma injections. Br J Sports Med 45(12):966–971
8. de Vos RJ, Weir A, van Schie HT et al (2010) Platelet-rich plasma injection for chronic Achilles tendinopathy: a randomized controlled trial. JAMA 303(2):144–149
9. de Vos RJ, Weir A, Tol JL, Verhaar JA, Weinans H, van Schie HT (2011) No effects of PRP on ultrasonographic tendon structure and neovascularisation in chronic midportion Achilles tendinopathy. Br J Sports Med 45(5):387–392
10. Monto RR (2012) Platelet rich plasma treatment for chronic Achilles tendinosis. Foot Ankle Int 33(5):379–385
11. Gaweda K, Tarczynska M, Krzyzanowski W (2010) Treatment of Achilles tendinopathy with platelet-rich plasma. Int J Sports Med 31(8):577–583
12. Sanchez M, Anitua E, Azofra J, Andia I, Padilla S, Mujika I (2007) Comparison of surgically repaired Achilles tendon tears using platelet-rich fibrin matrices. Am J Sports Med 35(2):245–251
13. Volpi P, Marinoni L, Bait C, De Girolamo L, Schoenhuber H (2007) Treatment of chronic patellar tendinosis with buffered platelet rich plasma: a preliminary study. Med Dello Sport 60(4):595–603
14. Kon E, Filardo G, Delcogliano M et al (2009) Platelet-rich plasma: new clinical application: a pilot study for treatment of jumper's knee. Injury 40(6):598–603
15. Filardo G, Kon E, Della Villa S, Vincentelli F, Fornasari PM, Marcacci M (2010) Use of platelet-rich plasma for the treatment of refractory jumper's knee. Int Orthop 34(6):909–915
16. Gosens T, Den Oudsten BL, Fievez E, van 't Spijker P, Fievez A (2012) Pain and activity levels before and after platelet-rich plasma injection treatment of patellar tendinopathy: a prospective cohort study and the influence of previous treatments. Int Orthop 36(9): 1941–1946
17. Bishop J, Klepps S, Lo IK, Bird J, Gladstone JN, Flatow EL (2006) Cuff integrity after arthroscopic versus open rotator cuff repair: a prospective study. J Shoulder Elbow Surg 15(3):290–299
18. Boileau P, Brassart N, Watkinson DJ, Carles M, Hatzidakis AM, Krishnan SG (2005) Arthroscopic repair of full-thickness tears of the supraspinatus: does the tendon really heal? J Bone Joint Surg Am 87(6):1229–1240
19. Chahal J, Van Thiel GS, Mall N et al (2012) The role of platelet-rich plasma in arthroscopic rotator cuff repair: a systematic review with quantitative synthesis. Arthroscopy 28(11):1718–1727
20. Bergeson AG, Tashjian RZ, Greis PE, Crim J, Stoddard GJ, Burks RT (2012) Effects of platelet-rich fibrin matrix on repair integrity of at-risk rotator cuff tears. Am J Sports Med 40(2):286–293
21. Randelli P, Arrigoni P, Ragone V, Aliprandi A, Cabitza P (2011) Platelet rich plasma in arthroscopic rotator cuff repair: a prospective RCT study, 2-year follow-up. J Shoulder Elbow Surg 20(4): 518–528
22. Gumina S, Campagna V, Ferrazza G et al (2012) Use of platelet-leukocyte membrane in arthroscopic repair of large rotator cuff tears: a prospective randomized study. J Bone Joint Surg Am 94(15):1345–1352
23. Jo CH, Shin JS, Lee YG et al (2013) Platelet-rich plasma for arthroscopic repair of large to massive rotator cuff tears: a randomized, single-blind, parallel-group trial. Am J Sports Med 41(10): 2240–2248
24. Castricini R, Longo UG, De Benedetto M et al (2011) Platelet-rich plasma augmentation for arthroscopic rotator cuff repair: a randomized controlled trial. Am J Sports Med 39(2):258–265
25. Weber SC, Kauffman JI, Parise C, Weber SJ, Katz SD (2013) Platelet-rich fibrin matrix in the management of arthroscopic repair of the rotator cuff: a prospective, randomized, double-blinded study. Am J Sports Med 41(2):263–270
26. Rha DW, Park GY, Kim YK, Kim MT, Lee SC (2013) Comparison of the therapeutic effects of ultrasound-guided platelet-rich plasma injection and dry needling in rotator cuff disease: a randomized controlled trial. Clin Rehabil 27(2):113–122
27. Kesikburun S, Tan AK, Yilmaz B, Yasar E, Yazicioglu K (2013) Platelet-rich plasma injections in the treatment of chronic rotator cuff tendinopathy: a randomized controlled trial with 1-year follow-up. Am J Sports Med 41(11):2609–2616
28. Ekdahl M, Wang JH, Ronga M, Fu FH (2008) Graft healing in anterior cruciate ligament reconstruction. Knee Surg Sports Traumatol Arthrosc 16(10):935–947
29. Radice F, Yanez R, Gutierrez V, Rosales J, Pinedo M, Coda S (2010) Comparison of magnetic resonance imaging findings in anterior cruciate ligament grafts with and without autologous platelet-derived growth factors. Arthroscopy 26(1):50–57
30. Sanchez M, Anitua E, Orive G, Mujika I, Andia I (2009) Platelet-rich therapies in the treatment of orthopaedic sport injuries. Sports Med 39(5):345–354
31. Orrego M, Larrain C, Rosales J et al (2008) Effects of platelet concentrate and a bone plug on the healing of hamstring tendons in a bone tunnel. Arthroscopy 24(12):1373–1380
32. Silva A, Sampaio R (2009) Anatomic ACL reconstruction: does the platelet-rich plasma accelerate tendon healing? Knee Surg Sports Traumatol Arthrosc 17(6):676–682
33. Nin JR, Gasque GM, Azcarate AV, Beola JD, Gonzalez MH (2009) Has platelet-rich plasma any role in anterior cruciate ligament allograft healing? Arthroscopy 25(11):1206–1213
34. Wright-Carpenter T, Klein P, Schaferhoff P, Appell HJ, Mir LM, Wehling P (2004) Treatment of muscle injuries by local administration of autologous conditioned serum: a pilot study on sportsmen with muscle strains. Int J Sports Med 25(8):588–593
35. Loo WL, Lee DY, Soon MY (2009) Plasma rich in growth factors to treat adductor longus tear. Ann Acad Med Singapore 38(8): 733–734
36. Hamilton B, Knez W, Eirale C, Chalabi H (2010) Platelet enriched plasma for acute muscle injury. Acta Orthop Belg 76(4): 443–448
37. Harmon KG, Rao AL (2013) The use of platelet-rich plasma in the nonsurgical management of sports injuries: hype or hope? Hematology Am Soc Hematol Educ Program 2013:620–626

Use of Scaffolds in Sports Medicine

57

Elizaveta Kon, Giuseppe Filardo, Giulia Venieri,
Francesco Perdisa, and Maurilio Marcacci

57.1 Introduction

Chondral and osteochondral injuries occur frequently in athletes, thus determining a higher risk of developing premature knee osteoarthritis (OA) with respect to the general population [1, 2]. Flanigan et al. [1] recently showed a mean 36 % prevalence of full-thickness focal chondral defects in athletes; moreover, Walczak et al. [3] found knee cartilage abnormalities in 89 % of asymptomatic professional basketball players using magnetic resonance imaging (MRI). Furthermore, it is well documented that articular cartilage lesions in a sports population commonly arise with concomitant injuries: meniscal (47 %) or anterior cruciate ligament (30 %) tears and medial or lateral collateral ligament tears (14 %) are the most commonly associated conditions [1]. Although the pathogenesis of early OA has not been clarified, it is recognized that traumatic injuries and chronic joint stress due to high-impact sports can lead to an unfavorable biomechanical environment, thus altering the natural homeostasis of articular cartilage [4]. Unfortunately, chondral injuries do not heal spontaneously and, if untreated, might cause a progression of joint degeneration with associated pain and functional limitation, which in turn will impede participating in sport and lead to disability [2, 5–7].

For such reasons, when treating young competitive athletes affected by articular cartilage defects, it is even more important to obtain the most effective and durable joint surface restoration, since the regenerated tissue must be able to withstand the significant joint stresses generated during sports activity [7–9].

E. Kon, MD (✉) • G. Filardo, MD, PhD
II Clinic – Nano-Biotechnology Laboratory,
Rizzoli Orthopaedic Institute, Via di Barbiano n. 1/10,
Bologna 40136, Italy
e-mail: e.kon@biomec.ior.it; g.filardo@biomec.ior.it

G. Venieri, MD • F. Perdisa, MD • M. Marcacci, MD
Biomechanics Laboratory, Rizzoli Orthopaedic Institute,
Via di Barbiano n. 1/10, Bologna 40136, Italy
e-mail: giulia.venieri@gmail.com; Francesco.perdisa@ior.it;
m.marcacci@biomec.ior.it

Techniques such as microfractures (MF) and mosaicplasty can successfully permit the athlete with articular cartilage injury to return to high-impact sports. MF represents the most frequently applied surgical approach in the clinical practice [10] and consists of arthroscopic bone marrow stimulation aimed at forming a clot of fibrin and precursor cells migrating from the adjacent bone marrow. Although the minimal invasiveness of this technique allows an early return to activity, it has been shown that this procedure offers mainly short-term benefits but tends to fail to provide long-lasting results, probably because of the poor mechanical properties of the newly formed fibrous tissue [11–13]. Mosaicplasty consists of the transplantation of osteochondral autologous cylinders harvested from a low weight-bearing area and implanted in the defect site by a press-fit technique [14]. Unfortunately, despite the positive results documented in the literature and the high percentage of return to sports activity [15], the issue of donor-site morbidity limits the indication of this technique for lesions smaller than 2–3 cm^2 [9].

Therefore, novel treatment concepts and techniques have been developed to overcome the limits of the classic approaches. Regenerative procedures are emerging which aim at restoring the articular cartilage defect by producing a high qualitative repair tissue, as similar as possible to the hyaline physiological one, and to allow immediate postoperative rehabilitation, which is directly correlated both with the short-term outcome and longer-lasting results [16–18].

This review briefly illustrates modern surgical strategies for articular cartilage regeneration, particularly focusing on scaffold-based procedures and the evidence for their use in athletic patients.

57.2 Articular Cartilage Scaffolds

Besides the satisfactory results obtained with the first generation of autologous chondrocyte implantation (ACI) in isolated femoral condyle lesions [19], numerous developments have been introduced taking advantage of bioengineering. In fact,

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_57, © Springer International Publishing Switzerland 2016

first-generation ACI presented several biological and surgical concerns, which led to the development of the so-called "matrix-assisted" autologous chondrocyte transplantation (MACT) [20]. Scaffolds are a temporary three-dimensional structure of biodegradable polymers, which are supposed to promote the growth of living cells and the maintenance of the differentiated chondrocyte phenotype and promote a more homogeneous distribution while avoiding the risk of chondrocyte leakage [21]. Moreover, the solid structure and easy handling of these engineered tissues allow a minimally invasive surgical application and then an early postoperative rehabilitation, which can favor and accelerate functional recovery [16, 22].

The ideal scaffold should fulfill a set of chemical, biochemical, and biophysical requirements able to meet several demands: it should consist of materials with controlled biodegradability or bioresorbability, characterized by adequate interconnecting pores to promote tissue integration and desired mechanical properties to match the site of implantation and handling; moreover, appropriate surface chemistry might promote cell attachment, proliferation, and differentiation. Finally, scaffolds should not induce adverse reactions and also be easily manufactured into a variety of shapes and sizes [23]. All these requirements explain why, despite the concept of "scaffolding" to support cells to infiltrate and regenerate the local tissue was firstly introduced more than one century ago [23], the introduction of scaffolds into clinical practice began only in recent decades [20]. Designing a material able to guide the tissue regeneration process is challenging, but advances in the field of molecular biology and material science have led to the development of numerous biomaterials [23]. Among the several scaffolds proposed in an attempt to better fulfill the requirements for cartilage regeneration, there are substantial differences regarding the materials chosen (natural or synthetic) and their physical forms (fibers, meshes, gels). Natural materials (hyaluronic acid, collagen derivatives, agarose, alginate, fibrin glue, and chitosan) have good biocompatibility, enhance cell proliferation, and are processed in a reliable and reproducible way. Concerning synthetic matrices, they are commonly made of polylactides, including polylactic and polyglycolic, and innovations in the chemistry of these materials have improved their biocharacteristics and biocompatibility.

Unfortunately, concerns about cell-based strategies, such as cost and time consumption because of the ex vivo cell processing [21], oriented research efforts toward different alternative solutions, developed to avoid manipulation of cells and the inherent regulatory obstacles. As an example of these new strategies, some scaffolds may have a potential themselves to promote tissue regeneration by exploiting the self-regenerative potential of the body. Thus, the use of cell-free scaffolds is the most recent trend nowadays. In fact, one-step cell-free approaches avoid problems related to the ex vivo chondrocyte culture and expansion in a scaffold, with marked advantages both from surgical and economic perspectives. The possibility of a cell-free implant, "smart" enough to provide the joint with the appropriate stimuli to induce orderly and durable tissue regeneration, is attractive, and new biomaterials and surgical strategies have been recently proposed to induce in situ cartilage regeneration after direct transplantation onto the defect site [24, 25]. Moreover, increasing awareness about the role of the subchondral bone in the pathogenesis of joint degeneration and early OA progression has led to the development of bilayer products to treat the entire osteochondral unit, thus reproducing the different biological and functional requirements for guiding the growth of both bone and cartilage tissues. In particular, this strategy becomes crucial when treating large chondral or osteochondral articular defects [24, 26].

Among the numerous osteochondral scaffolds developed and tested preclinically, only two have been reported for their clinical application [21]. One is a bilayer porous PLGA-calcium-sulfate biopolymer (TruFit®, Smith & Nephew, Andover, MA) in cylinder form, conceived as an alternative for mosaicplasty plugs [27, 28]. The second one is a three-layered nanostructured biomimetic collagen hydroxyapatite scaffold (MaioRegen®, Fin-Ceramica, Faenza, Italy) composed of the following: (1) a cartilaginous layer, consisting of type I collagen, with a smooth surface; (2) an intermediate layer, a combination of type I collagen (60 % of weight) and Mg-hydroxyapatite (HA) (40 % of weight); and (3) a lower layer that consists of a mineralized blend of type I collagen (30 % of weight) and Mg-HA (70 % of weight). Preclinical studies tested the safety and effectiveness of the implant to promote cartilage and bone tissue formation. A comparative analysis showed similar macroscopic, histological, and radiographic results for the scaffold either loaded with autologous chondrocytes or implanted alone, thus suggesting its ability to induce an in situ regeneration through cells coming from the surrounding bone marrow in the animal model and led to its introduction in the clinical practice as a cell-free approach [29, 30].

57.3 Clinical Evidence in Scaffold Application

MACT techniques were introduced in 1998 to improve the intrinsic limits of the traditional ACI technique. Their application was initially proposed for use in the knee joint, with a considerable number of clinical studies subsequently reported, and these techniques are more recently being applied also for the treatment of articular cartilage lesions in other joints [31]. However, only a few of these studies deal with long-term outcomes [32]. Filardo et al. [33] treated a series of 62 patients (48 M, 14 F, mean age 28.1 ± 11.4 years)

with hyaluronan-based MACT (Hyalograft C®, Fidia, Padova, Italy) for chondral lesions (ICRS grade III–IV, mean size 2.5 ± 1.0 cm^2) at the femoral condyles and prospectively evaluated them at 7 years' follow-up. A statistically significant improvement was observed in all the clinical scores from the basal evaluation to 84 months' follow-up. The IKDC subjective score increased from 39.6 ± 15.0 to 73.6 ± 18.8 at 12 months ($p < .005$), and a further slight improvement was observed at 24 months' follow-up (76.5 ± 20.7, ns), then the results were stable up to 77.3 ± 21.5 at final follow-up. Postoperative MRIs were performed in 42 cases, revealing a complete filling of the cartilage defect in 57 %. Further analysis showed that young men with higher pre-injury activity level reached better outcomes, whereas patients with degenerative lesions had worse results. Ibarra et al. documented a different arthroscopic approach based on matrix-encapsulated chondrocytes which obtained a positive clinical outcome in ten patients at 36 months [34]. Positive findings were confirmed by MRI using the MOCART score and T2 mapping quantitative evaluations, and patients were allowed to reach a satisfactory activity and sports level, as shown by the 5.8 mean Tegner score at 24 months, which remained stable at the final follow-up of 3 years [34].

In 2012, Kon et al. performed a comparative study to evaluate whether the regenerative cell-based approach allows highly demanding athletes a better functional recovery compared with the bone marrow stimulation [35]. Forty-one professional or semiprofessional male soccer players were treated and evaluated prospectively at 2 years and at a mean of 7.5 years' follow-up (minimum 4 years). Twenty-one patients received an arthroscopic MACI (Hyalograft C®), and 20 were treated with MF. Both groups had a significant improvement in all the scores used from preoperative evaluation to final follow-up. The IKDC subjective score showed similar results at 2 years, but the MACI group had a significantly better outcome at the final follow-up. In the MF group, the results decreased over time (from 86.8 ± 9.7 to 79.0 ± 11.6, $p < .0005$), whereas the Hyalograft C® group presented a more durable outcome, with stable results (90.5 ± 12.8 at 2 years and 91.0 ± 13.9 at final follow-up). Concerning the return to sports activity, 80 % of the patients treated with MF returned back to their previous level, playing the first official soccer game after a median of 8 months; in the Hyalograft C® group, 86 % of patients were back to previous level competitions in a median time of 12.5 months ($p = .009$). Despite the similar success rate in returning to competitive sport, it can be assumed that MF allows a faster recovery but presents a clinical deterioration over time, whereas arthroscopic MACT might delay the return of high-level male soccer players to competition, but at the same time offering a more durable clinical improvement. Moreover, MACT procedures might offer encouraging outcomes even for the treatment of patellofemoral full-thickness chondral defects, which are considered one of the most challenging lesions to treat. A study on 38 active patients treated with the same hyaluronan-based MACT showed a significant improvement in the clinical scores evaluated 2 and 5 years after surgery and a return to a satisfactory sports activity level, as evaluated by the Tegner score [36, 37].

This approach has even been tested for the treatment of degenerative lesions of the articular surface and gave encouraging clinical results but with a lower improvement and a higher number of failures with respect to patients affected by traumatic injuries [38]. Therefore, in recent years the focus on this kind of articular surface defects, usually linked to an altered joint environment, has extended to the role of the subchondral bone. Biphasic scaffolds were introduced to address both the cartilage and the subchondral bone issues. Currently, the use of just two scaffolds of this kind has been reported in the literature. The first one, a bilayer porous PLGA-calcium-sulfate, produced controversial results and thus is being abandoned in the clinical practice [39, 40].

Conversely, the implantation of the second one, a three-layered nanostructured biomimetic scaffold (MaioRegen®), is showing promising clinical results. A pilot study published by Kon et al. [41] involved 27 patients with chondral or osteochondral knee lesions (size 1.5–6.0 cm^2), which were treated with this osteochondral scaffold. The clinical evaluation performed prospectively for up to 60 months of follow-up showed a significant improvement in all clinical scores from basal evaluation to 24 months' follow-up, and the results were then stable over time. Furthermore, an MRI evaluation was performed on 23 lesions at both 24 and 60 months' follow-up, to assess the graft's appearance over time. The MOCART [42] score and a specifically designed score were used to assess the status of cartilage and subchondral bone, respectively. Both scores showed a significant improvement between the 2 follow-up times, even though no correlation with the clinical outcome was found. In detail, a complete integration of the graft was observed in 69.6 % of the cases, the repair tissue surface was intact in 60.9 %, and the structure of the repair tissue was homogeneous in 60.9 % at final follow-up. Further analysis, performed to investigate which patient characteristics might interfere with the outcome and recovery time, showed that active patients had a faster recovery compared to nonactive ones [24], even though they reached a similar final result. The effectiveness of such regenerative osteochondral technique has been successively confirmed also for the treatment of patients affected by osteochondritis dissecans (OCD), with a significant clinical improvement at 2 years' follow-up [43].

Following these promising results, the potential of this regenerative one-step procedure was tested also for the treatment of complex knee lesions, which more frequently occur in active or former athletes. A preliminary result showed the outcome of a 46-year-old active former soccer player, who

previously underwent anterior cruciate ligament (ACL) reconstruction. The osteochondral scaffold was implanted in four degenerative lesions at the medial femoral condyle (MFC), trochlea, and patella, in combination with closing-wedge high tibial osteotomy (HTO) to address varus misalignment [44], and positive findings were reported after 24 months, when the patient was pain-free and had almost fully recovered. A similar approach was applied to treat multifocal degenerative knee lesions in an Olympic-level woman athlete. A complex combined treatment was performed: implantation of the scaffold, autologous osteochondral grafting, patellar realignment, and meniscal allograft transplantation to address both joint surface lesions and associated comorbidities. The patient was able to return to high-level competition within 24 months postoperatively [45]. The validity of this combined biological and biomechanical approach was further investigated on 43 patients affected by unicompartmental OA (Kellgren-Lawrence grade 3) with full-thickness focal cartilage lesions in stable joints. Even in this larger cohort of patients, significant improvements were found in clinical outcome and activity level evaluations [46]. Other research groups also reported positive results using the same technique: Delcogliano et al. treated 19 patients for large lesions of the condyles and obtained a satisfactory clinical outcome and good function recovery at 24 months' follow-up, as confirmed by the Tegner score 5 (range 1–7); however, the patients did not reach their pre-injury activity level [47]. Berruto et al. showed similar results in a wider group of 49 patients affected by large lesions. Interestingly, a subanalysis of a subgroup of competitive athletes showed a significantly greater improvement ($p < .001$) in the subjective IKDC (86.5 ± 13.2) with respect to the nonathletic one (69.03 ± 19.41) at 24 months' follow-up [48]. Finally, a comparative study showed the application of this regenerative technique for the treatment of "complex cases" defined by the presence of at least one of the following characteristic: previous history of intra-articular fracture, tibial plateau lesion, concurrent knee axial realignment procedure, meniscal scaffold, or allograft implantation. Thirty-three patients (24 men, 9 women) were treated by implanting this osteochondral scaffold and, when needed, combined procedures to address axial misalignment and meniscal resection sequelae. Patients were prospectively followed up at 12 and 24 months, and a positive trend of improvement was revealed by all the scores used. These results were then compared with those of a homogeneous group of 23 patients previously treated and prospectively evaluated after the implantation of a chondral scaffold (MACT), which showed a significantly better outcome when the osteochondral approach was used for the treatment of complex lesions [49].

To conclude, it is important to highlight that an adequate rehabilitation program is crucial to optimize the results of cartilage surgery, since it may enhance cartilage repair and maturation, thus improving functional recovery and the ability to prevent the risk of reinjury [50]. A comparative analysis performed on 31 competitive athletes and 34 nonathletic patients treated by MACT showed that intensive rehabilitation might allow a faster return to sport in safety and even influence positively the clinical outcome in the midterm follow-up [16].

Currently available scaffolds showed promising results and offered new options to treat successfully both the traumatic chondral lesions in young athletes and their sequelae in former athletes. However, this treatment is still in its infancy and results are still preliminary. High-level studies should compare results with those of the more traditional procedures and identify the most appropriate indication criteria, and research should focus on the optimization of these surgical strategies to speed up and further improve the recovery of sports-active patients.

Conclusions

High-level athletes are exposed to an increased risk of joint injury, involving not only ligaments and menisci but also chondral and osteochondral structures. The repair of full-thickness articular cartilage defects with hyaline or hyaline-like tissue is essential to achieve a durable functional articular surface in the long term of such a demanding population. Of course, to optimize the final outcome, it is important not only to repair the osteochondral unit, but it is also crucial to restore the overall joint biomechanics, including the treatment of deformities, stability deficit, and menisci. Once the biomechanical environment is addressed, the osteochondral treatment can be performed with a better chance of success. Finally, outcome is determined not only by surgeons but also by an adequate, early, and intensive rehabilitation program. Among the several scaffolds that have been proposed over recent decades to fulfill the articular cartilage defects, none has demonstrated a perfect regenerative potential. However, encouraging outcomes, both clinical and radiological, have been reported in the literature at short- or midterm follow-up. Despite that, the superiority of one technique over another and the durability of these procedures have not been fully assessed. Further comparative studies and longer follow-ups are required to show clearly the efficacy of scaffolds in an athletic population, thus proving their real potential in allowing highly demanding and young patients to return to their previous sports activity level.

Elizaveta Kon Elizaveta Kon is a consultant for CartiHeal (2009) Ltd (Israel) and has stocks in it. She is a consultant and receives institutional support from Fin-Ceramica Faenza S.p.A (Italy). She also receives institutional support from Fidia Farmaceutici S.p.a (Italy), IGEA Clinical Biophysics (Italy), BIOMET (USA), and Kensey Nash (USA).

Giuseppe Filardo is a consultant and receives institutional support from Fin-Ceramica Faenza S.p.A. (Italy), Fidia Farmaceutici S.p.a. (Italy), and CartiHeal (2009) Ltd (Israel). He is also a consultant for EON Medica SRL (Italy). He receives institutional support from IGEA Clinical Biophysics (Italy), BIOMET (USA), and Kensey Nash (USA).

Giulia Venieri has no conflict of interests.

Francesco Perdisa has no conflict of interests.

Maurilio Marcacci receives royalties and research institutional support from Fin-Ceramica Faenza S.p.A. (Italy). He receives institutional support from Fidia Farmaceutici S.p.a. (Italy), CartiHeal (2009) Ltd (Israel), IGEA Clinical Biophysics (Italy), BIOMET (USA), and Kensey Nash (USA).

References

1. Flanigan DC, Harris JD, Trinh TQ, Siston RA, Brophy RH (2010) Prevalence of chondral defects in athletes' knees: a systematic review. Med Sci Sports Exerc 42(10):1795–1801
2. Kujala UM, Kettunen J, Paananen H, Aalto T, Battié MC, Impivaara O, Videman T, Sarna S (1995) Knee osteoarthritis in former runners, soccer players, weight lifters, and shooters. Arthritis Rheum 38(4):539–546
3. Walczak BE, McCulloch PC, Kang RW, Zelazny A, Tedeschi F, Cole BJ (2008) Abnormal findings on knee magnetic resonance imaging in asymptomatic NBA players. J Knee Surg 21(1):27–33
4. Heijink A, Gomoll AH, Madry H, Drobnič M, Filardo G, Espregueira-Mendes J, Van Dijk CN (2012) Biomechanical considerations in the pathogenesis of osteoarthritis of the knee. Knee Surg Sports Traumatol Arthrosc 20(3):423–435
5. Felson DT, Lawrence RC, Dieppe PA, Hirsch R, Helmick CG, Jordan JM, Kington RS, Lane NE, Nevitt MC, Zhang Y, Sowers M, McAlindon T, Spector TD, Poole AR, Yanovski SZ, Ateshian G, Sharma L, Buckwalter JA, Brandt KD, Fries JF (2000) Osteoarthritis: new insights. Part 1: the disease and its risk factors. Ann Intern Med 133(8):635–646
6. Buckwalter JA, Mankin HJ (1998) Articular cartilage: tissue design and chondrocyte-matrix interactions. Instr Course Lect 47:477–486
7. Buckwalter JA, Mankin HJ (1998) Articular cartilage: degeneration and osteoarthritis, repair, regeneration, and transplantation. Instr Course Lect 47:487–504
8. Vrahas MS, Mithoefer K, Joseph D (2004) The long-term effects of articular impaction. Clin Orthop Relat Res 423:40–43
9. McAdams TR, Mithoefer K, Scoop JM, Mandelbaum BR (2010) Articular cartilage injury in athletes. Cartilage 1(3):165–179
10. Brophy RH, Rodeo SA, Barnes RP, Powell JW, Warren RF (2009) Knee articular cartilage injuries in the National Football League: epidemiology and treatment approach by team physicians. J Knee Surg 22(4):331–338
11. Kon E, Gobbi A, Filardo G, Delcogliano M, Zaffagnini S, Marcacci M (2009) Arthroscopic second-generation autologous chondrocyte implantation compared with microfracture for chondral lesions of the knee: prospective nonrandomized study at 5 years. Am J Sports Med 37(1):33–41
12. Mithoefer K, Williams RJ 3rd, Warren RF, Potter HG, Spock CR, Jones EC, Wickiewicz TL, Marx RG (2005) The microfracture technique for the treatment of articular cartilage lesions in the knee. A prospective cohort study. J Bone Joint Surg Am 87(9): 1911–1920
13. Nehrer S, Spector M, Minas T (1999) Histologic analysis of tissue after failed cartilage repair procedures. Clin Orthop Relat Res 365:149–162
14. Szerb I, Hangody L, Duska Z, Kaposi NP (2005) Mosaicplasty: long-term follow-up. Bull Hosp Jt Dis 63(1–2):54–62
15. Marcacci M, Kon E, Zaffagnini S, Iacono F, Neri MP, Vascellari A, Visani A, Russo A (2005) Multiple osteochondral arthroscopic grafting (mosaicplasty) for cartilage defects of the knee: prospective study results at 2-year follow-up. Arthroscopy 21(4): 462–470
16. Della Villa S, Kon E, Filardo G et al (2010) Does intensive rehabilitation permit early return to sport without compromising the clinical outcome after arthroscopic autologous chondrocyte implantation in highly competitive athletes? Am J Sports Med 38:68–77
17. Mithoefer K, Hambly K, Logerstedt D, Ricci M, Silvers H, Della Villa S (2012) Current concepts for rehabilitation and return to sport after knee articular cartilage repair in the athlete. J Orthop Sports Phys Ther 42(3):254–273. doi:10.2519/jospt.2012.3665, Epub 2012 Feb 29. Review. PubMed PMID: 22383103
18. Mithoefer K, Hambly K, Della Villa S, Silvers H, Mandelbaum BR (2009) Return to sports participation after articular cartilage repair in the knee: scientific evidence. Am J Sports Med 37(Suppl 1):167S–176S. doi:10.1177/0363546509351650, Epub 2009 Oct 27. Review. PubMed PMID: 19861696
19. Brittberg M, Lindahl A, Nilsson A, Ohlsson C, Isaksson O, Peterson L (1994) Treatment of deep cartilage defects in the knee with autologous chondrocyte transplantation. N Engl J Med 331(14):889–895
20. Kon E, Verdonk P, Condello V, Delcogliano M, Dhollander A, Filardo G et al (2009) Matrix-assisted autologous chondrocyte transplantation for the repair of cartilage defects of the knee: systematic clinical data review and study quality analysis. Am J Sports Med 37(Suppl 1):156S–166S
21. Filardo G, Kon E, Roffi A, Di Martino A, Marcacci M (2013) Scaffold-based repair for cartilage healing: a systematic review and technical note. Arthroscopy 29(1):174–186
22. Marcacci M, Zaffagnini S, Kon E et al (2002) Arthroscopic autologous chondrocyte transplantation: technical note. Knee Surg Sports Traumatol Arthrosc 10:154–159
23. Gloria A, De Santis R, Ambrosio L (2010) Polymer-based composite scaffolds for tissue engineering. J Appl Biomater Biomech 8(2):57–67
24. Kon E, Delcogliano M, Filardo G, Busacca M, Di Martino A, Marcacci M (2011) Novel nano-composite multilayered biomaterial for osteochondral regeneration: a pilot clinical trial. Am J Sports Med 39(6):1180–1190
25. Gille J, Schuseil E, Wimmer J, Gellissen J, Schulz AP, Behrens P (2010) Mid-term results of Autologous Matrix-Induced Chondrogenesis for treatment of focal cartilage defects in the knee. Knee Surg Sports Traumatol Arthrosc 18(11):1456–1464
26. Carmont MR, Carey-Smith R, Saithna A, Dhillon M, Thompson P, Spalding T (2009) Delayed incorporation of a TruFit plug: perseverance is recommended. Arthroscopy 25(7):810–814
27. Melton JT, Wilson AJ, Chapman-Sheath P, Cossey AJ (2010) TruFit CB bone plug: chondral repair, scaffold design, surgical technique and early experiences. Expert Rev Med Devices 7(3):333–341
28. Williams RJ, Gamradt SC (2008) Articular cartilage repair using a resorbable matrix scaffold. Instr Course Lect 57:563–571
29. Kon E, Delcogliano M, Filardo G et al (2010) Orderly osteochondral regeneration in a sheep model using a novel nano-composite multilayered biomaterial. J Orthop Res 28(1):116–124
30. Kon E, Mutini A, Arcangeli E et al (2010) Novel nanostructured scaffold for osteochondral regeneration: pilot study in horses. J Tissue Eng Regen Med 4(4):300–308

31. Vannini F, Filardo G, Kon E, Roffi A, Marcacci M, Giannini S (2013) Scaffolds for cartilage repair of the ankle joint: the impact on surgical practice. Foot Ankle Surg 19(1):2–8
32. Kon E, Filardo G, Di Matteo B, Perdisa F, Marcacci M (2013) Matrix assisted autologous chondrocyte transplantation for cartilage treatment: a systematic review. Bone Joint Res 2(2):18–25
33. Filardo G, Kon E, Di Martino A, Iacono F, Marcacci M (2011) Arthroscopic second-generation autologous chondrocyte implantation: a prospective 7-year follow-up study. Am J Sports Med 39(10):2153–2160
34. Ibarra C, Izaguirre A, Villalobos E, Masri M, Lombardero G, Martinez V, Velasquillo C, Meza AO, Guevara V, Ibarra LG (2014) Follow-up of a new arthroscopic technique for implantation of matrix-encapsulated autologous chondrocytes in the knee. Arthroscopy 30(6):715–723. doi:10.1016/j.arthro.2014.02.032, Epub 2014 Apr 18. PubMed PMID: 24746406
35. Kon E, Filardo G, Berruto M, Benazzo F, Zanon G, Della Villa S, Marcacci M (2011) Articular cartilage treatment in high-level male soccer players: a prospective comparative study of arthroscopic second-generation autologous chondrocyte implantation versus microfracture. Am J Sports Med 39(12):2549–2557
36. Filardo G, Kon E, Di Martino A, Patella S, Altadonna G, Balboni F, Bragonzoni L, Visani A, Marcacci M (2012) Second-generation arthroscopic autologous chondrocyte implantation for the treatment of degenerative cartilage lesions. Knee Surg Sports Traumatol Arthrosc 20(9):1704–1713
37. Gobbi A, Kon E, Berruto M, Francisco R, Filardo G, Marcacci M (2006) Patellofemoral full-thickness chondral defects treated with Hyalograft-C: a clinical, arthroscopic, and histologic review. Am J Sports Med 34(11):1763–1773
38. Gobbi A, Kon E, Berruto M, Filardo G, Delcogliano M, Boldrini L, Bathan L, Marcacci M (2009) Patellofemoral full-thickness chondral defects treated with second-generation autologous chondrocyte implantation: results at 5 years' follow-up. Am J Sports Med 37(6):1083–1092
39. Dhollander AA, Liekens K, Almqvist KF, Verdonk R, Lambrecht S, Elewaut D, Verbruggen G, Verdonk PC (2012) A pilot study of the use of an osteochondral scaffold plug for cartilage repair in the knee and how to deal with early clinical failures. Arthroscopy 28(2):225–233
40. Joshi N, Reverte-Vinaixa M, Díaz-Ferreiro EW, Domínguez-Oronoz R (2012) Synthetic resorbable scaffolds for the treatment of isolated patellofemoral cartilage defects in young patients: magnetic resonance imaging and clinical evaluation. Am J Sports Med 40(6):1289–1295
41. Kon E, Filardo G, Di Martino A, Busacca M, Moio A, Perdisa F, Marcacci M (2014) Clinical results and MRI evolution of a nano-composite multilayered biomaterial for osteochondral regeneration at 5 years. Am J Sports Med 42(1):158–165
42. Marlovits S, Striessnig G, Resinger CT, Aldrian SM, Vecsei V, Imhof H, Trattnig S (2004) Definition of pertinent parameters for the evaluation of articular cartilage repair tissue with high-resolution magnetic resonance imaging. Eur J Radiol 52(3):310–319
43. Filardo G, Kon E, Di Martino A, Busacca M, Altadonna G, Marcacci M (2013) Treatment of knee osteochondritis dissecans with a cell-free biomimetic osteochondral scaffold: clinical and imaging evaluation at 2-year follow-up. Am J Sports Med 41(8):1786–1793
44. Kon E, Delcogliano M, Filardo G, Altadonna G, Marcacci M (2009) Novel nano-composite multi-layered biomaterial for the treatment of multifocal degenerative cartilage lesions. Knee Surg Sports Traumatol Arthrosc 17(11):1312–1315
45. Perdisa F, Filardo G, Di Matteo B, Di Martino A, Marcacci M (2014) Biological knee reconstruction: a case report of an Olympic athlete. Eur Rev Med Pharmacol Sci 18(1 Suppl):76–80
46. Marcacci M, Zaffagnini S, Kon E, Marcheggiani Muccioli GM, Di Martino A, Di Matteo B, Bonanzinga T, Iacono F, Filardo G (2013) Unicompartmental osteoarthritis: an integrated biomechanical and biological approach as alternative to metal resurfacing. Knee Surg Sports Traumatol Arthrosc 21(11):2509–2517
47. Delcogliano M, de Caro F, Scaravella E, Ziveri G, De Biase CF, Marotta D, Marenghi P, Delcogliano A (2014) Use of innovative biomimetic scaffold in the treatment for large osteochondral lesions of the knee. Knee Surg Sports Traumatol Arthrosc 22(6):1260–1269. doi:10.1007/s00167-013-2717-3
48. Berruto M, Delcogliano M, de Caro F, Carimati G, Uboldi F, Ferrua P, Ziveri G, De Biase CF (2014) Treatment of large knee osteochondral lesions with a biomimetic scaffold: results of a multicenter study of 49 patients at 2-year follow-up. Am J Sports Med 42(7):1607–1617
49. Filardo G, Kon E, Perdisa F, Di Matteo B, Di Martino A, Iacono F, Zaffagnini S, Balboni F, Vaccari V, Marcacci M (2013) Osteochondral scaffold reconstruction for complex knee lesions: a comparative evaluation. Knee 20(6):570–576
50. Creta S, Della Villa S, Roi GS (2006) Rehabilitation after arthroscopic autologous chondrocyte transplantation with three dimensional hyaluronan-based scaffolds of the knee. In: Zanasi S, Brittberg M, Marcacci M (eds) Basic science, clinical repair and reconstruction of articular cartilage defects: current status and prospects, vol 72. Timeo Editore, Bologna, Italy. pp 677–684

Arthroscopy and Sports Topics in Top Level Athletes: Use of a Brace

58

Angelo C.C. Di Giunta and Arcangelo Russo

58.1 Introduction

Bracing is a frequent component of the acute treatment of orthopaedic injuries. The main purposes of bracing are to immobilise, provide support, decrease pain, and/or to redirect forces away from the injured area. Indirectly, braces also play a role as a kinesthetic reminder to patients of their limitations. The use of bracing in sports traumatology can be preventive and for rehabilitation after surgical or conservative treatment.

In this chapter we deal with the use of tutors in large joints and the pathological conditions of overuse in trauma and in contact sports. While the recognised and widespread use of post-surgical or conservative braces has played an important role to date, their use in the prevention of joint injuries in sports is still very controversial because of the limitation of joint movement, the physical performance that they often determine and for some sports, because of federal regulations, as demonstrated by the recent international literature.

All braces place pressure on the skin; thus, appropriate fitting and education on adjustment and care are important, not only for skin integrity but also for patient compliance.

58.2 Shoulder

Slings can be used for many upper extremity injuries. In most cases, they are used for comfort and can be discontinued once pain subsides. They are used temporarily for clavicle fractures, acromioclavicular joint sprains, shoulder joint dislocation, proximal humerus fractures, and after the reduction of forearm fractures.

For several thousand years, even before Hippocrates, dislocated shoulders have been treated in a sling with the arm internally rotated. In spite of, and perhaps because of, using the same treatment for so long, there is little proof that it is effective.

The labrum acts as a chuck block, increasing the concavity of the glenoid and preventing translation of the humeral head. Thus, it stops the head sliding or rolling off the glenoid.

In a study of shoulder immobilisation in patients with Bankart lesions, Itoi et al. [1] used MRI to analyse shoulders immobilised during internal or external rotation. He found that immobilisation in internal rotation displaces the labrum. MRI findings demonstrated that, when patients' arms were immobilised during external rotation (mean, 35°), separation and displacement of the labrum decreased, and the detached area and opening angle of the antero-inferior portion of the capsule were smaller than when the arms were immobilised during internal rotation. The authors concluded that immobilisation during external rotation better approximates the Bankart lesion. However, in a recent randomised controlled trial, shoulder immobilisation during external rotation following primary dislocation did not reduce the rate of recurrence in patients with initial anterior shoulder dislocation. The compliance rate with immobilisation during external rotation was higher than compliance with immobilisation during internal rotation. The duration of immobilisation remains controversial.

Motion-limiting braces that prevent extreme shoulder abduction, extension, and external rotation are sometimes used once the athlete has returned to sport for anterior instability treatment or after dislocation treated incruently. These braces are designed to limit overhead motion and are best used in non-throwing athletes. Alternatively, neoprene sleeves can be used and are less restrictive than motion-limiting braces. These sleeves can be used in overhead athletes and motion-limiting straps can be

A.C.C. Di Giunta (✉)
Policlinico "G.B. Morgagni",
Via del Bosco 105, Catania 95124, Italy
e-mail: adigiunta@yahoo.com

A. Russo (✉)
Ospedale Umberto I – Enna,
C.da Ferrante, Enna 94100, Italy
e-mail: arcangelorusso@me.com

P. Volpi (ed.), *Arthroscopy and Sport Injuries: Applications in High-level Athletes*,
DOI 10.1007/978-3-319-14815-1_58,

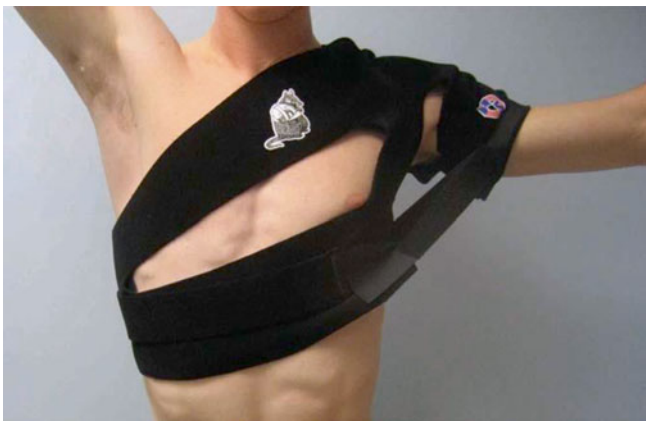


Fig. 58.1 Dinamic slink for anterior instability

incorporated if necessary (Fig. 58.1). Braces that restrict motion may be more effective in preventing future instability events; however, these braces can be cumbersome, potentially limiting the athlete's function and level of competitive play. Although bracing is associated with subjective improvement in stability, no studies have demonstrated a decreased rate of dislocation associated with bracing.

Moving on to the acromio-clavicular joint (ACJ), clavicle fractures make up 5 % of all fractures; thus, they are not uncommon. It is a common injury in contact sports (rugby, martial arts) and impact sports (such as horse, motor and bike racing). Most clavicle fractures are mainly treated in a sling for about 4–6 weeks, which may provide more comfort if applied correctly and align some fractures in a more stable position. However, complete healing can be slow and may take up to 3–6 months. The conservative treatment of midshaft clavicle fractures with simple slings vs figure-of-eight braces has been studied to examine whether one method is better than the other.

Concerning trauma, the ACJ in many contact sports, such as rugby, American football, ice hockey, basketball etc., is susceptible to frequent injury. Before the brace is applied, in terms of prevention, the foam pad is important in avoiding the impact on this small joint. Also, taping has been used for first- and second-degree sprains and ACJ separations are one of the most common injuries seen in orthopaedic and sports medicine practices, accounting for 9 % of all injuries to the shoulder girdle. Various operative and non-operative treatment schemes have been described for the management of ACJ injuries.

The ACJ consists of the articulation between the distal end of the clavicle and the acromion, functioning to anchor the clavicle to the scapula and shoulder girdle.

Early classification of AC joint injuries by Tossy and colleagues [2] and Allman [3] were based on radiographic displacement and the degree of ligamentous damage. They were initially graded I through III, and Rockwood and his

Fig. 58.2 AC joint stabilizer for acute dislocations

group [4] later added types IV through VI to the classification system.

Non-operative treatment is typically reserved for types I, II and acute type III injuries. This involves a 7- to 10-day period of rest, immobilisation and anti-inflammatory medications. Historically, braces, such as the Kenny–Howard brace, were used to treat these injuries. It acted to reduce the ACJ by applying a downward force on the clavicle and an upward force on the humerus. It was worn for 6–8 weeks and was associated with recurrence of deformity. Fourth-generation bracing was made for the non-operative treatments of the type I, II and for some type III ACJ dislocations for 4–6 weeks, with good compliance of the patients (Fig. 58.2).

58.3 Elbow

The most frequent injury is over-use syndrome and its effect on basic mechanics. The stress on the extensor muscles of the wrist (due to the frequent pronation and supination/extension movements against moderate or high resistance) cause microtrauma at the proximal osteotendinous insertion.

Painful symptoms play a role in the primary lesions of the extensor carpi radialis brevis, which is involved in 100 % of cases, but in 35 % of cases the extensor digitorum communis

Fig. 58.3 Supports for the treatment of the epicondilytis for day and night time

is also involved. This is termed epicondylitis and the aim of the therapy is to reduce or to eliminate the painful symptoms and recover the functionality of the arm. To enhance the individual effects, a route is created that applies more methods of treatment: pressure, cold, to the rest of the muscle at nighttime (Fig. 58.3).

Overuse of the wrist extensors is mentioned by many authors as a major factor in the development of lateral epicondylitis [5–9]. In tennis it has been shown that force and flexibility deficiencies in the forearm muscles and lack of movement accuracy lead to increased load at the lateral epicondyle [10]. These factors also correlate with the incidence of lateral epicondylitis.

Most braces on the market for the treatment of patients with epicondylitis claim to reduce the load at the lateral epicondyle. One type is clasp-based braces, which compress locally at the insertion of the wrist extensor tendons. In acute epicondylitis in particular, these braces are often not tolerated by the patient, because local compression at the insertion can be painful. Another type of brace applies compression over an area of several square centimetres with a silicone pad. A third type of brace was investigated in which a high-viscosity fluid pad is placed at the forearm over the extensor muscles. Conservative treatment of patients with lateral epicondylitis requires the limitation of repetitive stress to the origins of the two common extensors. It seems reasonable to consider the effectiveness of a brace in reducing the load at the lateral epicondyle as one of its quality criteria. The data show that the influence of a brace on the load at the lateral epicondyle depends on the characteristics of the product. Pad-based braces result in a much higher reduction of the load at the lateral epicondyle than braces with the a clasp. Placing a pad at the forearm, distal to the lateral epicondyle, seems to be superior to placing pads directly on the lateral epicondyle.

Hinged elbow braces may be used to provide medio-lateral support for elbow dislocations and collateral ligament sprains, while allowing a full range of motion. Postoperatively, a hinged elbow brace allows for a gradual increase in the range of motion. A dial on the hinge may be set to limit motion to a specific range, which can then be increased gradually during the healing process. The brace should run from the mid-bicep to the wrist. Straps should be secured from distal to proximal.

58.4 Wrist

A cock-up wrist brace is one of the most frequently used braces. It is appropriate for the treatment of wrist sprains and contusions. It may also be used for buckle (torus) fractures of the distal radius. Appropriate fitting requires the distal end of the splint to be just below the palmar crease so as not to restrict the movement of the meta-carpo-phalangeal joints.

For the treatment of distal radius buckle fractures, level I evidence studies demonstrate that a wrist splint is as good as a cast for the prevention of refracture and/or loss of alignment [11]. There was no difference in pain with use of a splint compared with a cast. Patients treated using the splint found it easier to bathe, had better function and did not need to return for cast removal.

A thumb spica splint or brace is used for the treatment of scaphoid injuries, gamekeeper's thumb (ulnar collateral

ligament sprain), De Quervain's tenosynovitis, and wrist and thumb sprains. The brace may be soft or rigid, depending on how much support is needed.

Appropriate fitting is for the distal end of the splint to sit just below the palmar crease and for the thumb portion to extend to the distal phalanx of the thumb to immobilise the interphalangeal joint.

58.5 Knee Braces

Controversy has long surrounded the use of knee braces in the practice of sports medicine. From their use as a prophylactic measure in high-risk sports to their prescription for ligamentous instabilities, knee braces have always been viewed with widely varying amounts of acceptance and scepticism. This has ranged from enthusiastic endorsement to outright condemnation. Braces for patellar-femoral disorders, osteoarthritis and postoperative rehabilitation have come to abound in the practice of sports medicine.

The idea of wearing a brace to prevent sports-induced injuries of the knee was first proposed in 1979 by Anderson and colleagues [12], who used a lateral, upright, dual-hinged device on National Football League players.

After it had been tested on nine players for short periods (one to nine games) without reinjury, this brace was proposed as a means of preventing ligamentous injury to the medial side of the knee. The authors believed that in addition to preventing a significant valgus stress to the knee, their brace also helped to restrict antero-posterior displacement. This report led to an explosion in the demand for prophylactic knee braces (PKBs) in football and other demanding sports, and a multitude of manufacturers sold off-the-shelf PKBs to meet the demand.

These can be classified into two basic types.

1. Single lateral upright and a single-axis, a dual-axis, or a polycentric hinge.
2. Bilateral (medial and lateral) uprights, a polycentric hinge and a band connecting the two units to make one [13] (Fig. 58.4).

Numerous laboratory studies investigating the effects of knee bracing have been performed. Some studies were designed to study PKBs, which are used to prevent knee injuries, whereas others looked at functional knee braces, which are structured to compensate for torn ligaments. Most studies are useful, but the reader must carefully consider the biomechanical parameter reproduced in the study and its subsequent effect on the ligaments of interest.

Cadaver studies are rarely performed at this time owing to longstanding criticism regarding the anatomical and age variations of specimens in addition to the increased expense.

In 1986, France and colleagues [14] published the results of loading experiments using 14 cadaver knees to test two

Fig. 58.4 knee brace 4 points

lateral upright PKBs. The authors concluded that these single upright braces were ineffective and possibly detrimental in resisting valgus forces.

These results were quoted widely and had a significant impact on how clinicians viewed these types of braces at the time. These same investigators subsequently demonstrated that this initial testing system was flawed, which added to the debate regarding the usefulness vs potential harmfulness of these types of braces [15].

Although these studies seemed to show that some types of braces could control antero-posterior translation and rotation of the knee, the loads used in these studies have uniformly been felt to be less than what would be seen under clinical, on-the-field playing conditions [16, 17]. Clinical studies using questionnaires, KT1000 measurements and physical examination to assess the effectiveness of functional braces have been similarly inconclusive.

Surrogates are testing fixtures made of metal and polymeric components that house a ligament substitute. Surrogates are designed to approximate closely and reproducibly the shape and the mechanical properties of the human knee. These have been shown to have less variability than cadaver specimens during testing. In addition, they are reusable because they have either replaceable ligaments or ligaments that are not destroyed during testing. This facilitates the use of increased sample sizes when comparing the effects of braces. Paulos and colleagues [18] engineered one of the first surrogate models and several types of prophylactic braces were tested. The authors concluded that a PKB was more effective when it was longer and displayed sufficient stiffness to distribute the impact away from the joint line and to divert it to the proximal thigh and the distal calf.

Paulos and coworkers, using the same mechanical surrogate developed by France and associates [15–19], showed that lateral knee braces could have a protective effect for both the medial collateral ligament (MCL) and the anterior cruciate ligament (ACL) against direct lateral blows to the knee. The ACL appeared to have better protection in this testing system.

Although there is no universal agreement, single upright PKBs probably do provide modest (20–30 %) MCL strain relief in the fully extended surrogate knee model when there is sufficient valgus force to cause mild-to-moderate joint-line opening in the unbraced specimen. It also appears that PKBs help to prevent abnormal motion in the sagittal plane, thereby also protecting the ACL. No change in current brace structures is likely to improve the effectiveness of this type of brace significantly. Although they are expensive and cumbersome, the greatest protection from MCL injury is likely to come from customised metal, dual upright functional braces like the Lenox Hill brace, which was tested. These braces apparently double the effectiveness of lateral upright knee braces, and they provide protection when the knee is flexed and when it is fully extended. A major contribution is made to the performance of this type of brace by the excellent fit to the thigh [20].

Few studies have been conducted on the effects of PKBs on knee function and athletic performance. It appears that knee braces do at least have the potential to restrict the performance of the athlete in high-speed running, but the effect is related to several factors. The weight of the braces in addition to its design features (e.g. type, number and resultant friction of the joints; how well it is contoured; tightness of the straps) appears to be important. With braces heavier than an elastic sleeve, the most measurable effects of the braces include increased muscular relaxation pressures, energy expenditure-related blood lactate levels, maximal torque outputs, and oxygen consumption and heart rate [12].

However, the pressure caused by the tension of the straps is a potential cause of increased intramuscular pressure. One successful method of brace suspension without necessarily increasing thigh and leg pressure is the use of Velcro to suspend the brace from the tight portion of the elasticised panty-girdle-type briefs that extends from the distal tight to above the iliac crests [21].

The most sophisticated studies on knee braces to date have reinforced the idea that players wearing PKBs are less likely to incur an MCL sprain. From the Big Ten Conference study alone, it is obvious that, far more important than whether or not a brace is worn, are the type of session, the position and the string to which the individual is assigned. All else being equal, preventive braces are probably clinically effective, but a much larger study is required for confirmation. The efficacy of PKBs remains in question, but recent studies have taught us enough to put their use into perspective. Although they may play some role, PKBs probably represent the least important factor in the likelihood of an MCL sprain. On the other hand, there is no evidence that such braces put added valgus pressure on some knees or that wearing a brace is associated with increased frequency or severity of knee or ankle injury [12].

58.6 Ankle Braces

External ankle stabilising devices (braces, tape) are commonly used for treatment, rehabilitation and prophylactic purposes in sport [22, 23]. There is evidence that the use of these devices (mainly semi-rigid braces) reduces the incidence of ankle sprains in high-risk sporting activities such as soccer or basketball in addition to tennis [24]. One potential reason is that devices are usually used in combination with shoes (Fig. 58.5). The overall stabilising effect may result from a combination of the stability of the external support device and the added effect of the shoe.

Fig. 58.5 Semi-rigid ankle brace

Ankle sprains are also frequent injuries in sports such as dancing and gymnastics in which no shoes are used. It remains unclear whether ankle braces provide the same stabilising effect in a barefoot situation as they do when used in combination with shoes [25–28].

The aim of the study by Eils was to evaluate the passive stability characteristics of the different external ankle stabilising devices with and without the influence of a sport shoe and to make recommendations regarding external stability devices in barefoot sports activities.

Under simulated barefoot conditions, a significantly reduced stabilising effect for inversion and eversion (19 and 29 % respectively) was found with the use of stirrup ankle braces. Small decreases were noted with the soft brace and tape, but these were not statistically significant. The passive stability characteristics of ankle braces depend to a great extent on being used in combination with a shoe. This is especially true for semi-rigid braces with a stirrup design. Therefore, it is recommended that soft braces be used in barefoot sports to restrict the passive range of motion of the foot and ankle complex.

Conclusion

The complexity of the joints has caused many of us to use different methods of prevention and protection through the use of externally aided devices. Even taping, strapping and bracing have become skills at which all of us involved in sports medicine are attempting to become more proficient. An external device can never restore the normal biomechanics of the injured joints. However, bracing usually supports traumatic injury and is very important for the correct adjunct for returning an athlete to competition.

References

1. Itoi E, Hatakeyama Y, Kido T, Sato T, Minagawa H, Wakabayashi I et al. A new method of immobilization after traumatic anterior dislocation of the shoulder: a preliminary study. J Shoulder Elbow Surg. 2003;12(5):413–5.
2. Tossy JD, Mead NC, Sigmond HM (1963) Acromioclavicular separations: useful and practical classification for treatment. Clin Orthop Relat Res 28:111–119
3. Allman FL Jr (1967) Fractures and ligamentous injuries of the clavicle and its articulation. J Bone Joint Surg Am 49(4): 774–784
4. Williams GR, Nguyen VD, Rockwood CA (1989) Classification and radiographic analysis of acromioclavicular dislocations. Appl Radiol 18:29–34
5. Coonrad RW, Hooper WR (1973) Tennis elbow: its course, natural history, conservative and surgical treatment. J Bone Joint Surg Am 55:1177–1182
6. Goldie I (1964) Epicondylitis lateralis humeri: a pathogenetical study. Acta Chir Scand 339:1–119
7. Brown A, Albright JP (2001) The use of knee braces in sports medicine. In: DeLee JC, Drez D (eds) Orthopaedic sports medicine: principles and practice, 2nd edn. Saunders, Philadelphia
8. Kaplan EB (1959) Treatment of tennis elbow. J Bone Joint Surg Am 41:147–151
9. Kelley JD, Lombardo SJ, Pink M, Perry J, Giangarra CE (1994) Electro-myographic and cinematographic analysis of elbow function in tennis players with lateral epicondylitis. Am J Sports Med 22:359–363
10. Roetert EP, Brody H, Dillman CJ, Groppel JL, Schultheis JM (1995) The biomechanics of tennis elbow. Integrated approach. Clin Sports Med 14:47–57
11. Plint AC, Perry JJ, Correll R et al (2006) A randomized, controlled trial of removable splinting versus casting for wrist buckle fractures in children. Pediatrics 117:691–697
12. Anderson G, Zeman SC, Rosenfeld RT (1979) The Anderson knee stabler. Phys Sports med. 7:125–127
13. Paulos LE, Drawbert JP, France EP et al (1986) Lateral knee braces in football: do they prevent injury? Phys Sports Med 14:119–124
14. France EP, Paulos LE, Jayaraman G et al (1987) The biomechanics of lateral knee bracing. II. Impact response of the braced knee. Am J Sports Med 15:430–438
15. Bollen SR (1992) Use of a knee-brace for control of tibial translation and rotation. A comparison in cadaver models [letter]. J Bone Joint Surg Am 74:154
16. Farmer MR (1989) Controlling anterior tibial displacement under static load: a comparison of two braces [letter]. Orthopedics 12:354–356
17. Alexander AH (1995) In search of the perfect ACL brace. Am J Orthop 24:328–336
18. Paulos LE, Cawley PW, France EP (1991) Impact biomechanics of lateral knee bracing: the anterior cruciate ligament. Am J Sports Med 19:337–342

19. Liggins AB, Bowker P (1991) A quantitative assessment of orthoses for stabilization of the anterior cruciate ligament deficient knee. Proc Inst Mech Eng H 205:81–87
20. Najibi S, Albright JP (2005) The use of knee braces. I. Prophylactic knee braces in contact sports. Am J Sports Med 33:602–611
21. Eils E (2005) Rehabilitation of the unstable ankle. J Orthop Sports Phys Ther 35:A13–A15
22. Kerkhoffs GM, Struijs PA, Marti RK, Blankevoort L, Assendelft WJ, van Dijk CN (2003) Functional treatments for acute ruptures of the lateral ankle ligament: a systematic review. Acta Orthop Scand 74:69–77
23. Handoll H, Rowe B, Quinn K, de Bie R (2001) Interventions for preventing ankle ligament injuries [Cochrane review]. Cochrane Database Syst Rev (3):CD000018
24. Alves JW, Alday RV, Ketcham DL, Lentell GL (1992) A comparison of the passive support provided by various ankle braces. J Orthop Sports Phys Ther 15:10–18
25. Eils E, Demming C, Kollmeier G, Thorwesten L, Völker K, Rosenbaum D (2002) Comprehensive testing of 10 different ankle braces: evaluation of passive and rapidly induced stability in subjects with chronic ankle instability. Clin Biomech (Bristol, Avon) 17:526–535
26. Shapiro MS, Kabo JM, Mitchell PW, Loren G, Tsenter M (1994) Ankle sprain prophylaxis: an analysis of the stabilizing effects of braces and tape. Am J Sports Med 22:78–82
27. Thonnard JL, Bragard D, Willems PA, Plaghki L (1996) Stability of the braced ankle: a biomechanical investigation. Am J Sports Med 24:356–361
28. Eils E, Imberge S, Volkler K (2007) Passive stability characteristics of ankle braces and tape in simulated barefoot and shod conditions. Am J Sports Md 35:282–287

Bibliography

Millet C, Drez D Jr (1987) Knee braces. Orthopedics 10:1777–1780