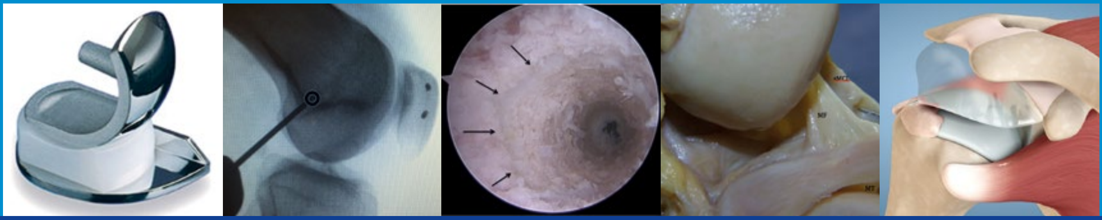
Gino M.M.J. Kerkhoffs
Fares Haddad
Michael T. Hirschmann
Jón Karlsson · Romain Seil
Editors



ESSKA Instructional Course Lecture Book

Glasgow 2018

European Society of Sports Traumatology, Knee Surgery & Arthroscopy
ESSKA

Springer

ESSKA Instructional Course Lecture Book

European Society of Sports Traumatology, Knee Surgery & Arthroscopy

ESSKA
Foundation

The ESSKA Instructional Course Lecture Book 2018
has been generously sponsored by the **ESSKA Foundation**.

The European Society for Sports Traumatology, Knee Surgery and Arthroscopy (ESSKA) established the ESSKA Foundation under the aegis of the Fondation de Luxembourg with the aim to raise the level of care and achieving excellence in the field of orthopedics, especially in sports medicine and degenerative joint disease in Europe, to improve musculoskeletal function and quality of life of patients.

To achieve its mission, the Foundation supports ESSKA education and research projects in the field of orthopedic and sports medicine.

For more information about ESSKA and ESSKA Foundation, please visit **www.esska.org**

Should you wish to donate to ESSKA Foundation for research or education in our field, please contact us at **info@esska.org**

ESSKA Foundation
Centre Médical
76 rue d'Eich, L-1460 Luxembourg
Phone: (+352) 4411-7026
Fax: (+352) 4411-7678

IBAN: LU47 0030 8907 9780 0000
Bank: BGL BNP Paribas S.A.
Swift Code: BGLL LULL

www.esska.org

Gino M.M.J. Kerkhoffs
Fares Haddad • Michael T. Hirschmann
Jón Karlsson • Romain Seil
Editors

ESSKA Instructional Course Lecture Book

Glasgow 2018

Springer

ESSKA

Editors
Gino M.M.J. Kerkhoffs
Orthopaedic Surgery
Academic Medical Center
AZ Amsterdam
The Netherlands

Michael T. Hirschmann
Kantonsspital Baselland
Bruderholz
Switzerland

Romain Seil
Clinique d'Eich
Centre Hospitalier de Luxembourg
Clinique d'Eich
Luxembourg
Luxembourg

Fares Haddad
Orthopaedics and Trauma
University College London Hospitals
London
United Kingdom

Jón Karlsson
Department of Orthopaedics
Sahlgrenska University Hospital
Mölndal
Sweden

ISBN 978-3-662-56126-3 ISBN 978-3-662-56127-0 (eBook)
https://doi.org/10.1007/978-3-662-56127-0

Library of Congress Control Number: 2017964627

© ESSKA 2018, corrected publication April 2018
This work is subject to copyright. All rights are reserved by the Publisher, whether the whole or part of the material is concerned, specifically the rights of translation, reprinting, reuse of illustrations, recitation, broadcasting, reproduction on microfilms or in any other physical way, and transmission or information storage and retrieval, electronic adaptation, computer software, or by similar or dissimilar methodology now known or hereafter developed.
The use of general descriptive names, registered names, trademarks, service marks, etc. in this publication does not imply, even in the absence of a specific statement, that such names are exempt from the relevant protective laws and regulations and therefore free for general use.
The publisher, the authors and the editors are safe to assume that the advice and information in this book are believed to be true and accurate at the date of publication. Neither the publisher nor the authors or the editors give a warranty, express or implied, with respect to the material contained herein or for any errors or omissions that may have been made. The publisher remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Printed on acid-free paper

This Springer imprint is published by Springer Nature
The registered company is Springer-Verlag GmbH Germany
The registered company address is: Heidelberger Platz 3, 14197 Berlin, Germany

Preface

Dear ESSKA members,

It is our great pleasure to offer you this instructional course lecture (ICL) book. It includes the contents of all the ICLs that will be given at the 2018 ESSKA Congress in Glasgow.

The book encapsulates the latest updates on surgical knowledge in the field of knee surgery, sports traumatology and arthroscopy.

A mixture of eminence and evidence-based material on the indications for surgical interventions, surgical tips and tricks, and management protocols should empower practitioners at every stage in their career.

In the light of the educational mission of ESSKA, we are excited to share this tome with you.

We hope that you will enjoy every aspect of it.

Amsterdam, The Netherlands	Gino M.M.J. Kerkhoffs
London, UK	Fares Haddad
Bruderholz, Switzerland	Michael T. Hirschmann
Mölndal, Sweden	Jón Karlsson
Luxembourg, Luxembourg	Romain Seil

Acknowledgements

The book represents the contents of all the instructional course lectures. We acknowledge the great contribution of the authors who allowed us to collate this book and present it at the 2018 ESSKA Congress. We would also like to acknowledge all those who have assisted us in the preparation and editing of the various chapters. A special word of thanks to Anne van der Made who did a great job in helping to motivate the authors to prepare their text and in keeping up the rhythm of the writing and editing.

Contents

1 Advances in Treatment of Complex Knee Injuries

Gilbert Moatshe, Jorge Chahla, Marc J. Strauss,
Robert F. LaPrade, and Lars Engebretsen

1.1 Introduction

Multi-ligament knee injuries are commonly defined as a tear of at least two of the four major knee ligament structures: the anterior cruciate ligament (ACL), the posterior cruciate ligament (PCL), the posteromedial corner (PMC), and the posterolateral corner (PLC) in the same incident [1, 2]. Knee dislocations often result in multi-ligament knee injuries, but some multi-ligament knee injuries are not knee dislocations. A knee dislocation is typically characterized by rupture of both cruciate ligaments, with or without an associated grade III medial- or lateral-sided injury [2, 3]. Knee dislocations with one of the cruciate ligaments intact have been reported, but these are less common [4, 5]. Multi-ligament injuries are heterogeneous and are often associated with other injuries in the ipsilateral limb and injuries to other organs. Therefore, a thorough diagnostic workup and treatment plan are mandatory when dealing with these injuries. The purpose of this chapter is to describe the principles of multi-ligament injuries including patient demographics and associated injuries, diagnosis and treatment approaches, surgical pearls for avoiding tunnel convergence, and grafts tensioning sequence, outcomes, and prevalence of osteoarthritis after knee dislocation surgery and future perspectives.

1.1.1 Classification

The most widely used classification system for the dislocated knee is based on the anatomical patterns of the ligaments torn and was described by Schenck et al. (Table 1.1) [3, 6]. The advantage of this classification is that it allows for identification of the torn ligaments

---

G. Moatshe (✉)
Department of Orthopaedic Surgery, Oslo University Hospital and University of Oslo, Oslo, Norway

OSTRC, The Norwegian School of Sports Sciences, Oslo, Norway
Steadman Philippon Research Institute, Vail, CO, USA
e-mail: gilbert.moatshe@medisin.uio.no

J. Chahla
Steadman Philippon Research Institute, Vail, CO, USA

M.J. Strauss
Department of Orthopaedic Surgery, Oslo University Hospital and University of Oslo, Oslo, Norway

R.F. LaPrade
Steadman Philippon Research Institute, Vail, CO, USA

The Steadman Clinic, Vail, CO, USA

L. Engebretsen
Department of Orthopaedic Surgery, Oslo University Hospital and University of Oslo, Oslo, Norway

OSTRC, The Norwegian School of Sports Sciences, Oslo, Norway

G.M.M.J. Kerkhoffs et al. (eds.), *ESSKA Instructional Course Lecture Book*,
https://doi.org/10.1007/978-3-662-56127-0_1

Table 1.1 Table with Schenck's knee dislocation classification [6]

KD I	Injury to single cruciate + collaterals
KD II	Injury to ACL and PCL with intact collaterals
KD III M	Injury to ACL, PCL, and MCL
KD III L	Injury to ACL, PCL, and LCL
KD IV	Injury to ACL, PCL, MCL, and LCL
KD V	Dislocation + fracture

Additional caps of "C" and "N" are utilized for associated injuries. "C" indicates an arterial injury. "N" indicates a neural injury, such as the tibial or, more commonly, the peroneal nerve

ACL anterior cruciate ligament, *PCL* posterior cruciate ligament, *MCL* medial collateral ligament, *LCL* lateral collateral ligament

and associated vascular, neurologic injuries, and fractures and also for planning of treatment.

1.2 State-of-the Art Treatment

1.2.1 Patient Demographics and Associated Injuries

Multi-ligament knee injuries were historically believed to be uncommon; however, Arom et al. recently reported an incidence of 0.072 per 100 patient-years based on a database with 11 million patients [7]. These injuries are often caused by both high-energy trauma [8], such as motor vehicle accidents and falls from heights, and low-energy trauma [9] including sporting activities. Engebretsen et al. reported that high-energy and sports-related injuries accounted for 51% and 47% of knee dislocations, respectively, based on a cohort of 85 patients with knee dislocations [10]. In a recent review of a large cohort of 303 patients with bicruciate knee dislocations, Moatshe et al. [11] reported equivalent rates of high- and low-energy trauma, with 50.3% and 49.7%, respectively. Miller et al. reported on multi-ligament knee injuries in obese individuals as a result of ultralow-velocity trauma [12]. These patients are reported to have a high prevalence of associated vascular and nerve injuries [12]. With obesity becoming a global problem, the incidence of these injuries will potentially increase.

Knees with both cruciate ligaments torn should be treated as knee dislocations, and the risk of vascular and neurologic injuries is high [13]. Furthermore, Geeslin and LaPrade reported that only 28% of posterolateral knee complex (PLC) injuries occur in isolation; hence patients presenting with PLC injuries should be evaluated for concomitant injuries [14]. Moatshe et al. [11] reported common peroneal nerve injuries and vascular injuries in 19% and 5%, respectively, in an evaluation of 303 patients with knee dislocations. Based on their cohort, the odds of having a peroneal nerve injury were 42 times higher among patients with posterolateral corner injury than those without, while the odds of having a popliteal artery injury were 9.2 times higher in patients with a posterolateral corner injury. Additionally, a peroneal nerve injury was significantly associated with a vascular injury with an odds ratio of 20.6. Thus, patients with peroneal nerve injuries should be examined thoroughly for an associated vascular injury, and the surgeon should have a low threshold for obtaining a CT angiogram. In a systematic review by Medina et al. [15], the frequencies of nerve and vascular injuries in knee dislocations were 25% and 18%, respectively. Becker et al. reported a comparable prevalence of peroneal nerve injuries (25%) but a higher prevalence of arterial injuries (21%) in a series of 106 patients [13].

A high prevalence of meniscal and focal cartilage injuries is reported in multi-ligament knee injuries. In a review of 121 patients (122 knees), Krych et al. reported that 76% of overall patients had a meniscal or chondral injury; 55% presented with meniscal tears, while 48% presented with a chondral injury in a follow-up of 121 patients (122 knees) [16]. However, Richter et al. reported a lower incidence (15%) of meniscal injuries in association with knee dislocations [17]. In a recent review of 303 patients with knee dislocations from a single center, Moatshe et al. [11] reported meniscal injuries in 37.3% of the patients and cartilage injuries in 28.3%. Patients treated for multi-ligament injuries in the chronic phase had higher prevalence of chondral lesions.

Medial-sided injuries are usually the most common injuries in multi-ligament knee injury patterns. Moatshe et al. [11] reported that medial-sided injuries constituted 52% of the injuries in 303 patients with knee dislocations. In their series, lateral-sided injuries constituted 28%, and bicruciate injuries with no other ligament involvement constituted only 5%. In a review by Robertson et al. [18], medial-sided and lateral-sided injuries were reported in 41% and 28%, respectively. In contrast, Becker et al. reported that lateral-sided injuries were the most common (43%) in a series of 106 patients [13]. What is common for these studies is that KD III injuries are the most common ligament injury pattern in knee dislocations.

1.2.2 Acute Treatment and Diagnostics

1.2.2.1 Acute Multiple-Ligament Knee Injuries Diagnostics

It is important to estimate the amount of energy involved in the injury. High-energy trauma can cause injuries distant to the knee, which can take the attention from the injured knee, leading to a missed or late diagnosis. Furthermore, associated limb or organ injuries can affect the treatment plan. It is recommended to apply the Advanced Trauma Life Support (ATLS) principles when treating high-energy injuries. Concomitant injuries to the popliteal artery (23–32%) [8, 19] and the common peroneal nerve (14–40%) [15, 20] are commonly observed in high-velocity knee dislocations.

For vascular assessment, foot pulses and skin color should be examined and compared with the uninjured side and monitored after admission for early detection of change in circulation. Physical examination with the presence of a normal vascular examination (normal and symmetrical pulses, capillary refill, normal neurological examination) is reported to be reliable to screen patients with knee dislocations for "selective" arteriography [21]. The ankle-brachial index (ABI) is useful as an adjunct to the physical examination to assess for vascular injuries, especially in patients where physical examination is not reliable such as those with neurological injuries and the obese. An angiography is recommended when the ankle-brachial index (ABI) is <0.9 (Fig. 1.1) [22, 23].

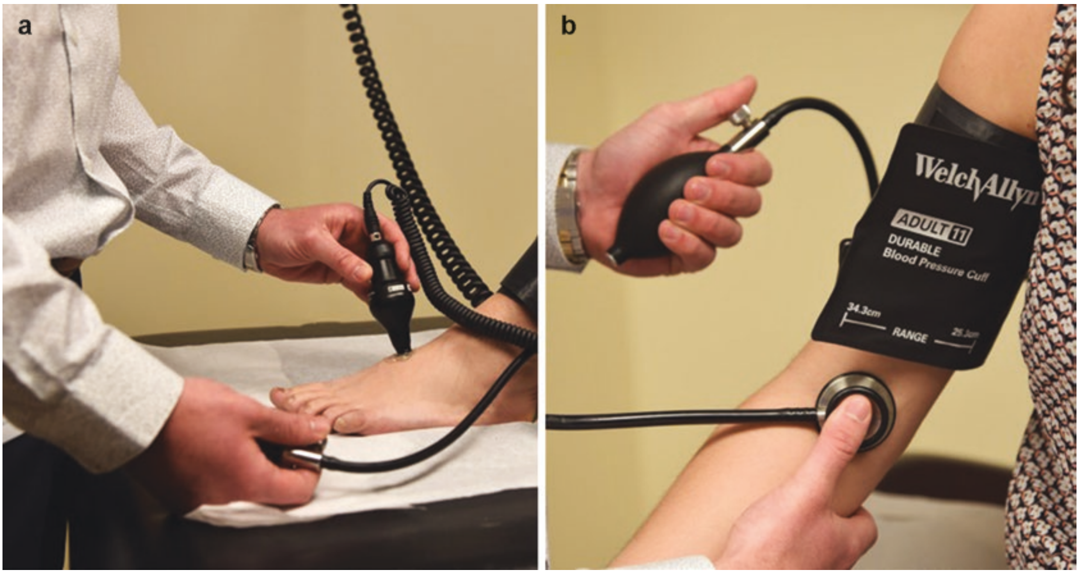


Fig. 1.1 Obtaining an (**a**) ankle- (**b**) brachial index (ABI) is important to have an objective evaluation of the vascular system. If the ABI is <0.9, angiography is recommended. Patients with peroneal nerve injuries have a higher odds of a concomitant vascular injury and should therefore be considered for CT angiography

In the obese patients with ultralow-velocity knee dislocations, one should have a low threshold for CT angiography examination because of the difficulty in physical examination and the previously reported high risk of vascular injuries [12, 24]. Some protocols recommend an ABI cut-off of <0.8 [25], while others recommend <0.9 to perform arteriography [21, 22]. The authors recommend a cutoff of <0.9 because ABI is easy and inexpensive to perform, while the consequences of not detecting vascular injury can be devastating. Patients with vascular injuries are initially treated with acute revascularization, and the knee is protected in an external fixator to protect the revascularization graft and to maintain knee reduction [25, 26]. The external fixator is usually removed at 2 weeks, and the knee is placed in a hinged brace to avoid pin infections and joint stiffness.

Magnetic resonance imaging (MRI) is performed to evaluate all the injured structures, including ligaments, menisci, and cartilage (Fig. 1.2). Stress radiographs are essential in the evaluation of the PCL, PLC, and the PMC but can be difficult to carry out in the acute phase due to patient guarding (Figs. 1.3 and 1.4) [27–29]. In cases where stress radiographs are difficult to perform, a mini C-arm can be utilized for the

Fig. 1.2 Preoperative magnetic resonance image (MRI) showing a posterior cruciate ligament (PCL) tear in a patient with multi-ligament injury

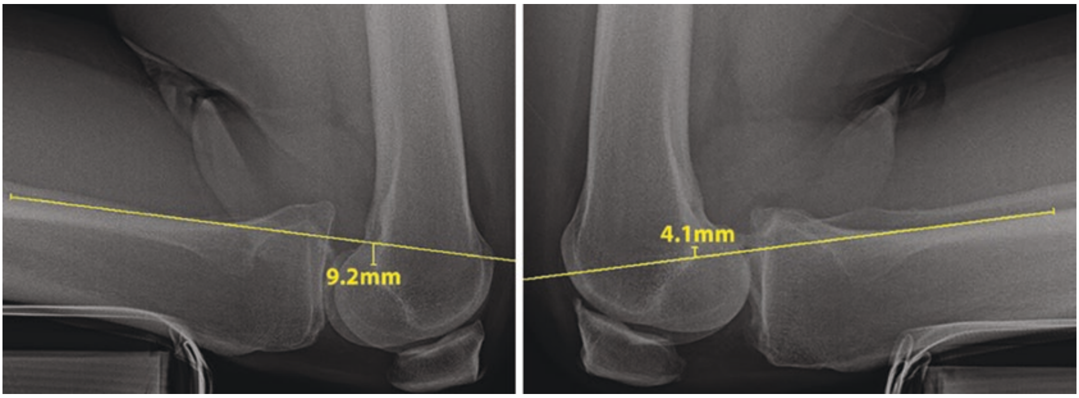


Fig. 1.3 Preoperative stress radiographs are important in evaluating patients with knee ligament injuries. In this patient, there was a 13.3 mm increase in posterior tibial translation on the left compared to the right knee, consistent with a combined PCL injury. To compare the posterior tibial translation, a point is identified along the posterior tibial cortex 15 cm distal to the joint line. A line is then drawn from this point parallel to the posterior cortex, through the femoral condyles. The most posterior point of Blumensaat's line is marked. A perpendicular line is drawn from the most posterior point of the Blumensaat's line to intersect the first line drawn parallel to the tibial cortex. This distance is compared to the contralateral side to give a side-to-side difference. A posterior translation side-to-side difference of 0–7 mm is usually due to partial PCL tear or in patients who are too sore to put sufficient weight on the knee; an 8–11 mm side-to-side difference is associated with a complete isolated PCL tear; and ≥12 mm is usually observed in patients with a complete PCL tear and additional ligament injury, usually the PLC or PMC but can also be seen in patients with decreased sagittal plane tibial slope

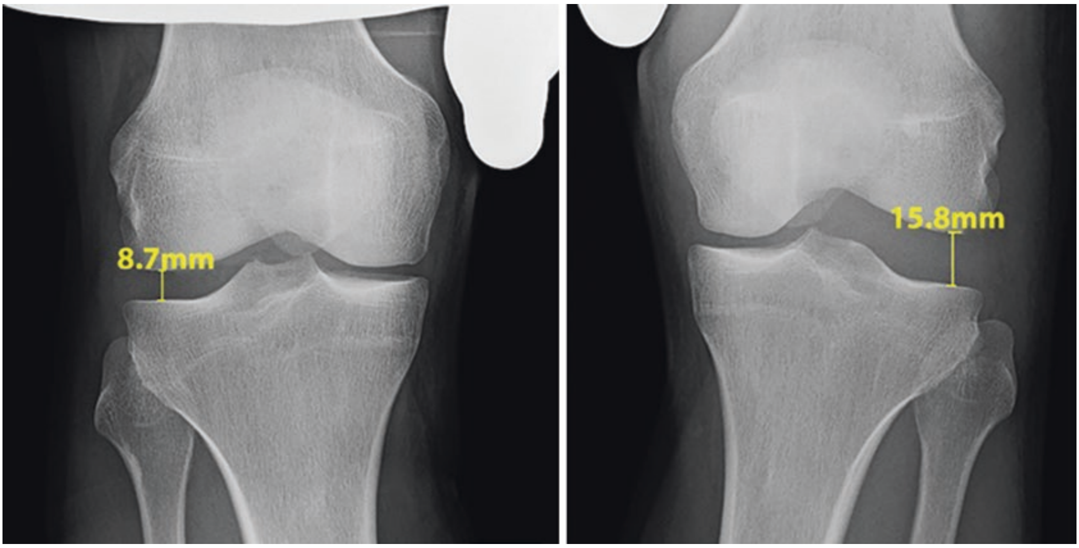


Fig. 1.4 Varus stress radiographs to evaluate the integrity of the posterolateral corner preoperatively. In this picture there is a 7.1 mm side-to-side difference consistent with a complete posterolateral corner (PLC) injury

examination under anesthesia at the time of surgery to objectively determine the amount of knee gapping. It is important to diagnose and treat collateral ligament injuries concurrently with cruciate ligament reconstructions because untreated collateral ligament injuries will lead to increased forces on the cruciate ligament reconstruction grafts, increasing the risk of graft failure [30, 31].

1.2.2.2 Treatment

It is commonly accepted that multi-ligament injuries should be treated with reconstruction of the torn ligaments. Non-operative treatment can be considered for the elderly, sedentary, and high surgical risk patients. Surgical treatment of the torn ligaments in multi-ligament injured knees improves patient-reported outcomes [17, 32, 33]. In a meta-analysis including 132 knees treated surgically and 74 treated nonsurgically, Dedmond and Almekinders reported better outcomes in the surgically treated group than the nonsurgical group, range of motion (123° in the surgical group vs. 108° in the nonsurgical group) and Lysholm scores (85.2 in the surgical group vs. 66.5 in the nonsurgical group) [32]. Richter et al. [17] reported significantly improved outcomes in the surgical group compared to the nonsurgical group in an evaluation of 89 patients with traumatic knee dislocations (63 patients treated with surgical repair or reconstruction, 26 patients treated nonsurgically) with a mean follow-up of 8.2 years. In a literature review by Peskun and Whelan [33] evaluating outcomes in 855 patients from 31 studies treated surgically, and 61 patients from 4 studies treated nonsurgically, functional outcomes, stability, and return to activity favored surgical treatment. In summary, the literature supports surgical treatment and postoperative functional rehabilitation of multi-ligament knee injuries.

1.2.2.3 Repair Versus Reconstruction

Several studies have demonstrated that reconstruction of the torn ligaments is superior to repair. Mariani et al. evaluated outcomes in a cohort of patients with multi-ligament injuries, 52 patients treated with repair of the ligaments versus 28 treated with reconstructions [34]. Patients with repair of cruciate ligaments had higher rates of flexion deficit, higher rates of posterior instability, and lower rates of return to preinjury activity levels. Studies by Stannard et al. and Levy et al. demonstrated high reoperation and failure rates in patients with posterolateral injuries treated with repair, further strengthening the argument for reconstruction of the collateral ligaments [35, 36]. Anatomic reconstruction of the injured structures using biomechanically validated techniques restores knee kinematics to near normal and yields improved patient outcomes [37–39]. Therefore, in the setting of multi-ligament injuries, reconstruction of all the torn ligaments is recommended. Repair of the collaterals is usually reserved for bony avulsions that are large enough to be fixed with hardware or suture anchors [40].

1.2.2.4 Timing of Surgery

Timing of surgery during multi-ligament injuries is a topic of debate, and there is still no consensus on the point of demarcation between acute and chronic. Some authors have used 3 weeks as the critical time to better identify and treat the structures before scar tissue forms, making dissection and identification of the structures difficult, and tissue necrosis affects outcomes [10, 34, 41, 42]. However, some authors have used a 6-week timeline to demarcate between acute and chronic injuries [37]. Studies have reported superior outcomes in acutely treated patients compared to chronic treated patients [1, 43]. Even though some surgeons are concerned about the risk of joint stiffness in acutely treated injuries, Levy et al. reported no difference in range of motion after acute and chronic surgery in a systematic review of literature that included five studies [1]. The authors preferred acute treatment of the injured structures to facilitate early rehabilitation [37]. In addition, staging the reconstruction can potentially alter joint kinematics and increase the risk of graft failure [30, 31, 44]. In high-energy trauma, surgery may be delayed because of injuries to the soft tissue about the knee and concomitant injuries to other vital organs. However, stiffness in these patients may be easier to treat than recurrent instability.

1.2.3 Surgical Treatment Pearls

1.2.3.1 Avoiding Tunnel Convergence

Reconstructing several reconstruction tunnels in the distal femur and proximal tibia poses a risk of tunnel convergence because of limited bone mass in these areas. Tunnel convergence increases the risk of reconstruction graft failure because of the potential damage to reconstruction grafts, fixation devices, and not having sufficient bone stock between the grafts for fixation and graft incorporation. Moatshe et al. reported a 66.7% tunnel convergence rate between the posterior oblique ligament (POL) tunnel and the PCL tunnel in the tibia when the POL tunnel was aimed at Gerdy's tubercle when evaluating the risk of tunnel convergence using biomechanically validated anatomic reconstruction techniques (Fig. 1.5). They recommended that the POL tunnels be aimed to a point 15 mm medial to Gerdy's tubercle to reduce risk of convergence with the PCL and that the superficial medial collateral ligament (sMCL) tunnel be aimed 30° distally to avoid convergence with the PCL tunnel [45].

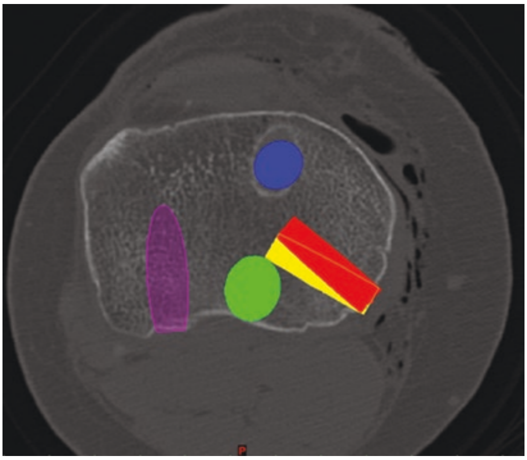


Fig. 1.5 There is a high risk of tunnel interference between the PCL (green) and POL (yellow) tunnels during multi-ligament knee reconstructions. Aiming the POL tunnel 15 mm anterior to Gerdy's tubercle (red) minimizes the convergence with the PCL tunnel (green). The anterior cruciate ligament (ACL) tunnel (blue) and the tunnel for the popliteus tendon and the popliteofibular ligament grafts (purple) are also shown. *PCL* posterior cruciate ligament, *POL* posterior oblique ligament, *ACL* anterior cruciate ligament

On the lateral femoral side, Moatshe et al. [46] performed a 3D imaging study varying the angles of the FCL and popliteus tunnels. A 35–40° angulation in the axial plane and 0° in the coronal plane was safe and avoided tunnel convergence (Fig. 1.6). On the medial side, aiming the sMCL

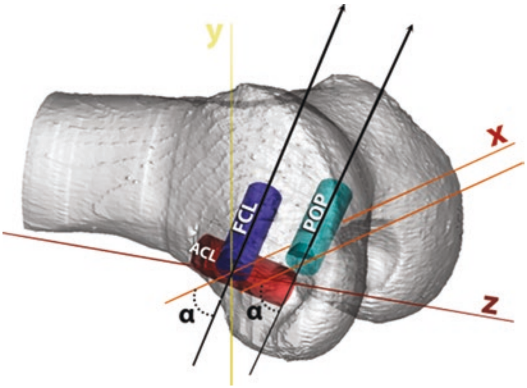


Fig. 1.6 Illustration demonstrating tunnels on the lateral femur condyle during multi-ligament knee reconstructions. Aiming the FCL (purple) and the popliteus (turquoise) 35–40° anteriorly minimizes the risk of tunnel convergence with the ACL (red) tunnel. *ACL* anterior cruciate ligament, *FCL* fibular collateral ligament, *POP* popliteus tendon tunnel (With permission from Moatshe G, Brady AW, Slette EL, Chahla J, Turnbull TL, Engebretsen L, LaPrade RF. Multiple Ligament Reconstruction Femoral Tunnels: Intertunnel Relationships and Guidelines to Avoid Convergence. Am J Sports Med. 2017 Mar;45(3):563–569.

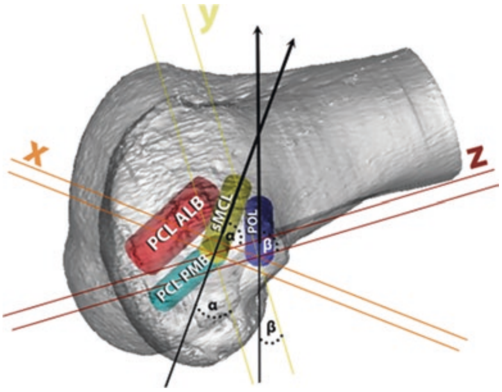


Fig. 1.7 Illustration demonstrating four tunnels in the medial femoral condyle. With four potential tunnels in the medial femoral condyle, the risk of tunnel convergence is high. Aiming the sMCL tunnel 40° anteriorly and 20–40° and the POL tunnel 20° anteriorly and proximally minimizes the risk of tunnel convergence (With permission from Moatshe G, Brady AW, Slette EL, Chahla J, Turnbull TL, Engebretsen L, LaPrade RF. Multiple Ligament Reconstruction Femoral Tunnels: Intertunnel Relationships and Guidelines to Avoid Convergence. Am J Sports Med. 2017 Mar;45(3):563-569.

tunnel 40° in the axial and coronal planes and the POL tunnel 20° in the axial and coronal planes was safe to avoid convergence with the double-bundle PCL tunnels (Figs. 1.7 and 1.8). In a laboratory study, Camarda et al. reported a high risk of tunnel convergence between the ACL and the FCL (69–75% depending on the length of the tunnel) and recommended aiming the FCL tunnel 0° in the coronal plane and 20–40° in the axial plane [47]. Gelber et al. evaluated tunnel convergence and optimal angulation of the tunnels on the medial femur condyle. They found that angulations of 30° in the axial plane and coronal plane reduced the risk of convergence with the PCL tunnels [48]. However, the diameter of their PCL tunnels was smaller than those used by Moatshe et al., and that can potentially explain the differences reported.

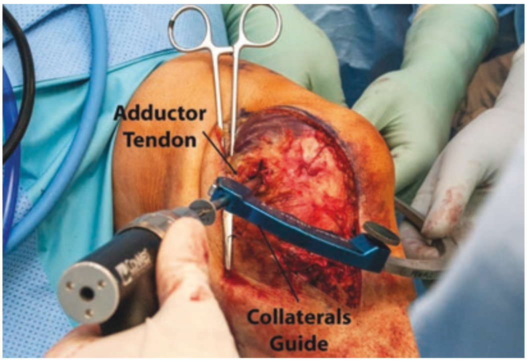


Fig. 1.8 An intraoperative picture demonstrating orientation of the sMCL tunnel on the femur to avoid convergence with the double-bundle PCL tunnels. The sMCL tunnel is aimed anteriorly and proximally to avoid convergence with the PCL tunnels. The adductors tendon is a “light house” on the medial side. The sMCL attaches 12 mm distal and 8 mm anterior to the adductor tubercle, which can be found just distal to the adductor tendon attachment. *sMCL* superficial medial collateral ligament, *PCL* posterior cruciate ligament

1.2.3.2 Tensioning Sequence

The tensioning sequence in multi-ligament injuries is a topic of debate, with different tensioning sequences having been reported in the literature. Some authors advocate for starting with the PCL to restore the central pivot and tibial step-off (Fig. 1.9), followed by the ACL in extension to ensure the knee can be fully extended, posterolateral corner, and the posteromedial corner last [49, 50].

Fig. 1.9 An intraoperative picture showing reduction of a right knee to restore tibial step-off prior to tensioning and fixing the anterolateral bundle (ALB) of the PCL. The PCL is tensioned first to restore tibial step-off, followed by the posterolateral corner (PLC) tension and fixation. The ACL is fixed after the PLC and PCL, and the PMC is fixed last. *ALB* anterolateral bundle, *PCL* posterior cruciate ligament, *PLC* posterolateral corner, *ACL* anterior cruciate ligament

In a posterolateral corner-deficient knee, tension during fixation of the ACL graft increased external tibial rotation of the tibia [44]. This change in tibiofemoral orientation would change joint mechanics and loading. Therefore, some authors advocate for fixing the posterolateral corner prior to the ACL to avoid external tibial rotation. Markolf et al. reported that the PCL should be fixed prior to the ACL to best restore graft forces, based on a biomechanical study of cadaveric bicruciate-injured knees [51]. Kim et al. retrospectively reviewed 25 patients with multi-ligament injuries, 14 with the PCL tensioned first, and 11 with simultaneous tension and fixing the ACL first and reported that posterior stress radiographs, Lysholm score, and IKDC scores favored fixing the ACL first [52]. There is currently no consensus regarding the optimal tensioning sequence, and there is a need for well-designed biomechanical studies [53]. Such biomechanical studies will lay ground for multicenter clinical studies to evaluate the optimal tensioning sequence. The author's preferred tensioning sequence is fixing the anterolateral bundle of the PCL at 90° to restore the normal tibial step-off, the posteromedial bundle of the PCL in extension, the FCL (LCL) at 20–30° of knee flexion, neutral rotation, and a slight valgus force, followed by the rest of the PLC structures at 60° of flexion and neutral rotation, the ACL near full extension, and finally the posteromedial corner. The PLC is fixed prior to the ACL to avoid external rotation of the tibia during tensioning of the ACL. Prepping the contralateral knee and using an intraoperative C-arm may aid when reducing the injured knee during graft tensioning and fixation.

1.2.3.3 Rehabilitation

Another key step for a successful outcome is a comprehensive and staged rehabilitation program starting from day 1 postoperative. The main goals are to protect the surgical reconstructions and to restore range of motion (ROM). All patients are instructed to remain non-weight bearing for 6 weeks while wearing a brace (dynamic brace for PCL reconstruction patients), followed by a 2-week period of weaning off crutches before achieving full weight bearing at 8 weeks' postsurgery. ROM exercises are probably the most important part of the rehabilitation to avoid stiffness and include patellofemoral joint mobilization and tibiofemoral flexion and extension from 0–90°. Additionally, all patients began quadriceps-setting exercises day 1 postsurgery to achieve symmetrical active knee extension at 6 weeks to facilitate a normal gait pattern. A stationary bike was initiated at 6 weeks postsurgery, depending on the range of motion. Although every rehabilitation protocol is customized to the patient, the periodization concept was utilized and included the following phases: muscular endurance, strength, and power development. Each phase consists of at least 6 weeks to allow for physiological adaptation to the exercise stimulus. Rehabilitation progress is assessed throughout the recovery, with clearance to return to activities provided once patients had achieved a quadriceps index greater than 90% and a passing grade on the Vail Sport Test [54].

1.2.4 Outcomes and Prevalence of Osteoarthritis After Knee Dislocation Surgery

Surgical management is recommended for multi-ligament knee injuries; therefore, this section will focus on outcomes after surgical management.

Good functional outcomes are reported in short to medium follow-up after surgical treatment of multi-ligament injuries [1, 10]. In a follow-up of 85 patients with knee dislocations at 2–9 years, Engebretsen et al. reported improved patient-reported outcomes with a mean Lysholm of 83, median Tegner activity score of 5, and mean IKDC 2000 subjective score of 64 [10]. Moatshe et al. [55] reported a mean Lysholm score of 84, Tegner score of 4, and subjective IKDC 73 in a follow-up of 65 patients with multi-ligament knee injuries at a minimum follow-up of 10 years demonstrating that good functional outcomes are possible at medium to long term. Geeslin and LaPrade [37] reported on 29 patients (30 knees), 8 knees had isolated posterolateral corner injuries, and 22 knees had combined ligament injuries involving the posterolateral corner. At a mean follow-up of 2.4 years, Cincinnati and IKDC subjective outcome scores improved from 21.9 to 81.4 and 29.1 to 81.5, respectively. Side-to-side varus gapping on stress radiographs improved from 6.2 mm preoperatively to 0.1 mm postoperatively [37]. Postoperative stress radiographs are an important objective method of evaluating stability (Fig. 1.10). Certain factors have been reported to correlate with poor outcomes including high-energy trauma [10], repair of medial-sided injury [56], age >30 years [55, 57], concomitant cartilage injury [58], and combined medial and lateral meniscal tears [58].

Despite good functional outcomes reported by these studies [37, 39, 43, 49, 59, 60], posttraumatic osteoarthritis (PTOA) is a common problem, reported to range from 23 to 87% [10, 43, 60] in the different studies (Fig. 1.11). Engebretsen et al. reported an 87% prevalence of PTOA, evaluated by the Kellgren-Lawrence (grade II or more) classification, after knee dislocation surgery of the patients in a cohort of 85 patients at 5–9 years' follow-up. In a follow-up of 68 patients at a median follow-up time of 12 years (range, 1–27 years), Hirschmann et al. reported a 31% prevalence of PTOA, and 16% had grade III and IV on Kellgren-Lawrence scale [43]. Fanelli et al. reported degenerative changes in 23% (10 of the 44) of the patients treated for knee dislocations at a mean follow-up of 10 years (range 5–22 years) [60].In a recent evaluation of knee dislocation patients treated surgically at a minimum follow-up time of 10 years, Moatshe et al. [55] reported that 42% of the cohort had radiologic osteoarthritis (KL ≥ 2) in the injured knee compared to only 6% in the uninjured knee.

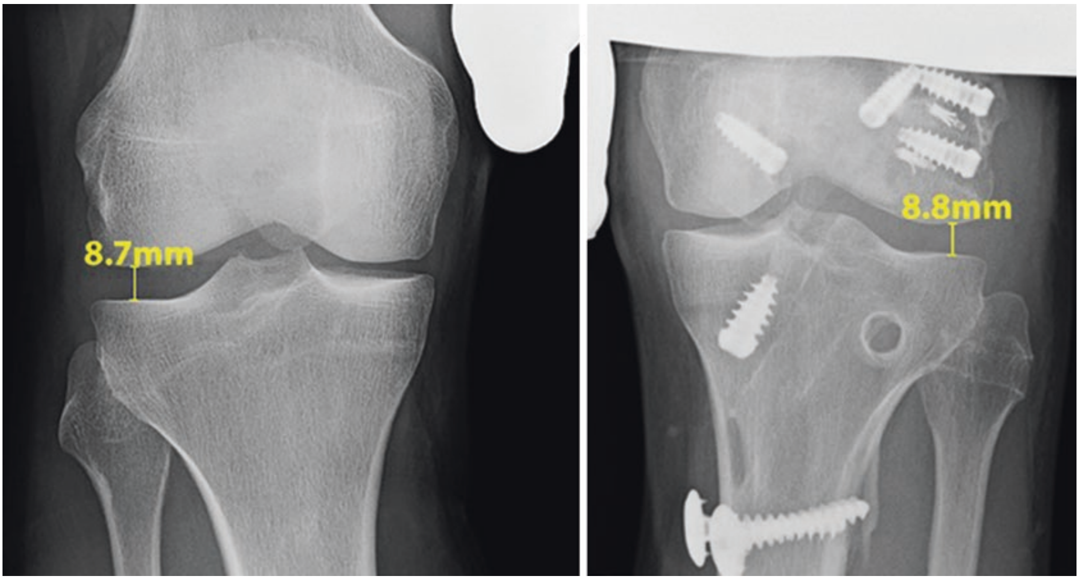


Fig. 1.10 Postoperative varus stress radiographs demonstrating a 0.1 mm side-to-side difference in the lateral compartment gapping compared to the normal contralateral knee Postoperative stress radiographs are valuable in evaluating knee stability.

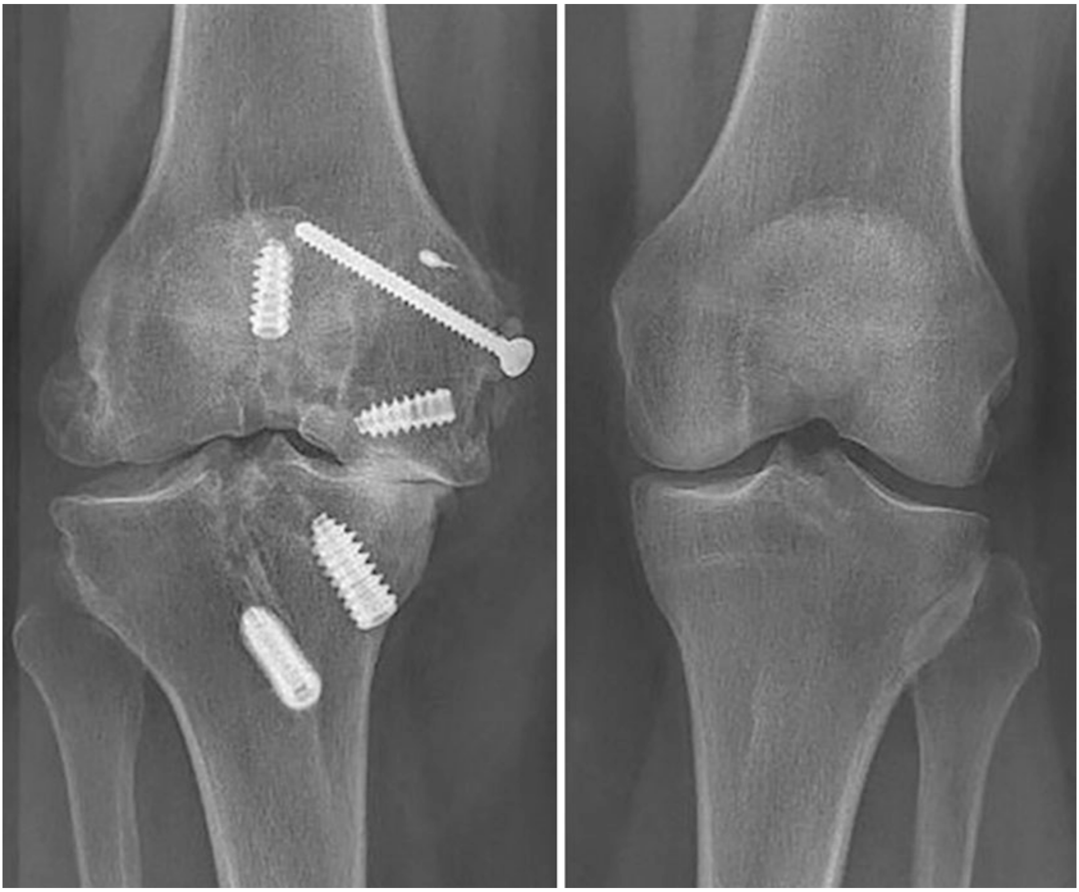


Fig. 1.11 A plain radiograph showing posttraumatic osteoarthritis on the right knee after knee dislocation surgery involving the ACL, PCL, and sMCL. The injured left knee has no sign of osteoarthritis. *ACL* anterior cruciate ligament, *PCL* posterior cruciate ligament, *sMCL* superficial medial collateral ligament

1.2.5 Future Treatment Options

Multi-ligament knee injuries are complex, and a high level of suspicion is required when evaluating these patients. Some of the concurrent ligament and meniscal injuries may be missed initially, and this requires a detailed history and clinical examination, supplemented with MRI and stress radiographs as part of the initial workup. Failure to treat all injured structures can lead to changes in knee kinematics and hence poorer outcomes and an increased risk of graft failure. Treating all the injured structures in the acute phase is recommended in order to facilitate early rehabilitation and better restoration of knee function. Biomechanical studies are necessary to evaluate the effects of the different tensioning orders to the knee kinematics. This will potentially pave the way for multicenter clinical studies to evaluate this in clinical settings. In addition, several reconstruction grafts are often needed during this type of surgery, posing a problem in areas where allografts are not available. Optimal reconstruction in the setting where allografts are not available is an area that needs further research. With the growing population and more grown-up people wanting to remain active, there is a need for research on enhancing healing of the reconstruction grafts because of poor healing potential that comes with age.

1.2.6 Take-Home Messages

- Multi-ligament injuries are challenging and require a detailed preoperative diagnosis, treatment plan, and a dedicated surgical and rehabilitation team to take care of the patients.
- Stress radiographs are valuable preoperatively to evaluate the torn ligaments and plan the surgery and postoperatively to evaluate the integrity of the ligament reconstructions.
- Posterolateral injuries are commonly associated with peroneal nerve and vascular injuries. Furthermore, the odds of vascular injuries are higher in the presence of a peroneal nerve injury. A high level of suspicion is advocated.
- Avoid tunnel convergence by detailed preoperative and intraoperative planning; the FCL tunnel should be aimed anteriorly or anteriorly and proximally to avoid convergence with the ACL tunnel. The sMCL tunnel and the POL tunnels should be aimed anteriorly and proximally to avoid convergence with the PCL tunnels.
- A well-designed, customized rehabilitation protocol is mandatory for good outcomes. The reconstruction grafts should be protected in a brace, while healing, and periodization of the rehabilitation is important.
- Treatment of these complex cases should be centralized and treated by dedicated teams with extensive surgical experience and volume.

References

1. Levy BA, Dajani KA, Whelan DB, et al. Decision making in the multiligament-injured knee: an evidence-based systematic review. Arthroscopy. 2009;25:430–8.
2. Wascher DC, Dvirnak PC, DeCoster TA. Knee dislocation: initial assessment and implications for treatment. J Orthop Trauma. 1997;11:525–9.
3. Schenck R. Classification of knee dislocations. Oper Tech Sports Med. 2003;11:193–8.
4. Bratt HD, Newman AP. Complete dislocation of the knee without disruption of both cruciate ligaments. J Trauma. 1993;34:383–9.
5. Shelbourne KD, Pritchard J, Rettig AC, McCarroll JR, Vanmeter CD. Knee dislocations with intact PCL. Orthop Rev. 1992;21:607–8. 610–601
6. Schenck RC Jr. The dislocated knee. Instr Course Lect. 1994;43:127–36.
7. Arom GA, Yeranosian MG, Petrigliano FA, Terrell RD, McAllister DR. The changing demographics of knee dislocation: a retrospective database review. Clin Orthop Relat Res. 2014;472:2609–14.
8. Shields L, Mital M, Cave EF. Complete dislocation of the knee: experience at the Massachusetts General Hospital. J Trauma. 1969;9:192–215.
9. Shelbourne KD, Klootwyk TE. Low-velocity knee dislocation with sports injuries. Treatment principles. Clin Sports Med. 2000;19:443–56.
10. Engebretsen L, Risberg MA, Robertson B, Ludvigsen TC, Johansen S. Outcome after knee dislocations: a 2-9 years follow-up of 85 consecutive patients. Knee Surg Sports Traumatol Arthrosc. 2009;17:1013–26.
11. Moatshe G, Dornan GJ, Løken S, Ludvigsen TC, LaPrade RF, Engebretsen L. Knee dislocations demographics and associated injuries: a prospective review of 303 patients. Orthop J Sports Med. 2017;5(5):2325967117706521.
12. Werner BC, Gwathmey FW Jr, Higgins ST, Hart JM, Miller MD. Ultra-low velocity knee dislocations: patient characteristics, complications, and outcomes. Am J Sports Med. 2014;42:358–63.
13. Becker EH, Watson JD, Dreese JC. Investigation of multiligamentous knee injury patterns with associated injuries presenting at a level I trauma center. J Orthop Trauma. 2013;27:226–31.
14. Geeslin AG, LaPrade RF. Location of bone bruises and other osseous injuries associated with acute grade III isolated and combined posterolateral knee injuries. Am J Sports Med. 2010;38:2502–8.
15. Medina O, Arom GA, Yeranosian MG, Petrigliano FA, McAllister DR. Vascular and nerve injury after knee dislocation: a systematic review. Clin Orthop Relat Res. 2014;472:2621–9.
16. Krych AJ, Sousa PL, King AH, Engasser WM, Stuart MJ, Levy BA. Meniscal tears and articular cartilage damage in the dislocated knee. Knee Surg Sports Traumatol Arthrosc. 2015;23:3019–25.
17. Richter M, Bosch U, Wippermann B, Hofmann A, Krettek C. Comparison of surgical repair or reconstruction of the cruciate ligaments versus nonsurgical treatment in patients with traumatic knee dislocations. Am J Sports Med. 2002;30:718–27.
18. Robertson A, Nutton RW, Keating JF. Dislocation of the knee. J Bone Joint Surg Br. 2006;88:706–11.
19. Wascher DC. High-velocity knee dislocation with vascular injury. Treatment principles. Clin Sports Med. 2000;19:457–77.
20. Molund M, Engebretsen L, Hvaal K, Hellesnes J, Ellingsen Husebye E. Posterior tibial tendon transfer improves function for foot drop after knee dislocation. Clin Orthop Relat Res. 2014;472:2637–43.

21. Kendall RW, Taylor DC, Salvian AJ, O'Brien PJ. The role of arteriography in assessing vascular injuries associated with dislocations of the knee. J Trauma. 1993;35:875–8.
22. Levy BA, Fanelli GC, Whelan DB, et al. Controversies in the treatment of knee dislocations and multiligament reconstruction. J Am Acad Orthop Surg. 2009;17:197–206.
23. Mills WJ, Barei DP, McNair P. The value of the ankle-brachial index for diagnosing arterial injury after knee dislocation: a prospective study. J Trauma. 2004;56:1261–5.
24. Carr JB, Werner BC, Miller MD, Gwathmey FW. Knee dislocation in the morbidly obese patient. J Knee Surg. 2016;29:278–86.
25. Klineberg EO, Crites BM, Flinn WR, Archibald JD, Moorman CT 3rd. The role of arteriography in assessing popliteal artery injury in knee dislocations. J Trauma. 2004;56:786–90.
26. Stuart MJ. Evaluation and treatment principles of knee dislocations. Oper Tech Sports Med. 2001;9:91–5.
27. Jackman T, LaPrade RF, Pontinen T, Lender PA. Intraobserver and interobserver reliability of the kneeling technique of stress radiography for the evaluation of posterior knee laxity. Am J Sports Med. 2008;36:1571–6.
28. James EW, Williams BT, LaPrade RF. Stress radiography for the diagnosis of knee ligament injuries: a systematic review. Clin Orthop Relat Res. 2014;472:2644–57.
29. Laprade RF, Bernhardson AS, Griffith CJ, Macalena JA, Wijdicks CA. Correlation of valgus stress radiographs with medial knee ligament injuries: an in vitro biomechanical study. Am J Sports Med. 2010;38:330–8.
30. LaPrade RF, Muench C, Wentorf F, Lewis JL. 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. 2002;30:233–8.
31. LaPrade RF, Resig S, Wentorf F, Lewis JL. The effects of grade III posterolateral knee complex injuries on anterior cruciate ligament graft force. A biomechanical analysis. Am J Sports Med. 1999;27:469–75.
32. Dedmond BT, Almekinders LC. Operative versus nonoperative treatment of knee dislocations: a meta-analysis. Am J Knee Surg. 2001;14:33–8.
33. Peskun CJ, Whelan DB. Outcomes of operative and nonoperative treatment of multiligament knee injuries: an evidence-based review. Sports Med Arthrosc Rev. 2011;19:167–73.
34. Mariani PP, Santoriello P, Iannone S, Condello V, Adriani E. Comparison of surgical treatments for knee dislocation. Am J Knee Surg. 1999;12:214–21.
35. Levy BA, Dajani KA, Morgan JA, Shah JP, Dahm DL, Stuart MJ. Repair versus reconstruction of the fibular collateral ligament and posterolateral corner in the multiligament-injured knee. Am J Sports Med. 2010;38:804–9.
36. Stannard JP, Brown SL, Farris RC, McGwin G Jr, Volgas DA. The posterolateral corner of the knee: repair versus reconstruction. Am J Sports Med. 2005;33:881–8.
37. Geeslin AG, LaPrade RF. Outcomes of treatment of acute grade-III isolated and combined posterolateral knee injuries: a prospective case series and surgical technique. J Bone Joint Surg Am. 2011;93:1672–83.
38. LaPrade RF, Griffith CJ, Coobs BR, Geeslin AG, Johansen S, Engebretsen L. Improving outcomes for posterolateral knee injuries. J Orthop Res. 2014;32:485–91.
39. Spiridonov SI, Slinkard NJ, LaPrade RF. Isolated and combined grade-III posterior cruciate ligament tears treated with double-bundle reconstruction with use of endoscopically placed femoral tunnels and grafts: operative technique and clinical outcomes. J Bone Joint Surg Am. 2011;93:1773–80.
40. Geeslin AG, Moulton SG, LaPrade RF. A systematic review of the outcomes of posterolateral corner knee injuries, part 1: surgical treatment of acute injuries. Am J Sports Med. 2016;44:1336.
41. Fanelli GC, Giannotti BF, Edson CJ. Arthroscopically assisted combined posterior cruciate ligament/posterior lateral complex reconstruction. Arthroscopy. 1996;12:521–30.
42. Harner CD, Waltrip RL, Bennett CH, Francis KA, Cole B, Irrgang JJ. Surgical management of knee dislocations. J Bone Joint Surg Am. 2004;86-A:262–73.
43. Hirschmann MT, Zimmermann N, Rychen T, et al. Clinical and radiological outcomes after management of traumatic knee dislocation by open single stage complete reconstruction/repair. BMC Musculoskelet Disord. 2010;11:102.
44. Wentorf FA, LaPrade RF, Lewis JL, Resig S. The influence of the integrity of posterolateral structures on tibiofemoral orientation when an anterior cruciate ligament graft is tensioned. Am J Sports Med. 2002;30:796–9.
45. Moatshe G, Slette EL, Engebretsen L, LaPrade RF. Intertunnel relationships in the tibia during reconstruction of multiple knee ligaments: how to avoid tunnel convergence. Am J Sports Med. 2016;44(11):2864–9.
46. Moatshe G, Brady AW, Slette EL, et al. Multiple ligament reconstruction femoral tunnels: intertunnel relationships and guidelines to avoid convergence. Am J Sports Med. 2017;45(3):563–9.
47. Camarda L, D'Arienzo M, Patera GP, Filosto L, LaPrade RF. Avoiding tunnel collisions between fibular collateral ligament and ACL posterolateral bundle reconstruction. Knee Surg Sports Traumatol Arthrosc. 2011;19:598–603.
48. Gelber PE, Masferrer-Pino A, Erquicia JI, et al. Femoral tunnel drilling angles for posteromedial corner reconstructions of the knee. Arthroscopy. 2015;31:1764–71.
49. Fanelli GC, Edson CJ. Surgical treatment of combined PCL-ACL medial and lateral side injuries

(global laxity): surgical technique and 2- to 18-year results. J Knee Surg. 2012;25:307–16.

50. Levy BA, Boyd JL, Stuart MJ. Surgical treatment of acute and chronic anterior and posterior cruciate ligament and lateral side injuries of the knee. Sports Med Arthrosc Rev. 2011;19:110–9.
51. Markolf KL, O'Neill G, Jackson SR, McAllister DR. Reconstruction of knees with combined cruciate deficiencies: a biomechanical study. J Bone Joint Surg Am. 2003;85-A:1768–74.
52. Kim SJ, Kim SH, Jung M, Kim JM, Lee SW. Does sequence of graft tensioning affect outcomes in combined anterior and posterior cruciate ligament reconstructions? Clin Orthop Relat Res. 2015;473:235–43.
53. Levy BA. CORR insights(R): does sequence of graft tensioning affect outcomes in combined anterior and posterior cruciate ligament reconstructions? Clin Orthop Relat Res. 2015;473:244–5.
54. Garrison JC, Shanley E, Thigpen C, Geary R, Osler M, Delgiorno J. The reliability of the vail sport test as a measure of physical performance following anterior cruciate ligament reconstruction. Int J Sports Phys Ther. 2012;7:20–30.
55. Moatshe G, Dornan GJ, Ludvigsen TC, Løken S, LaPrade RF, Engebretsen L. High prevalence of knee osteoarthritis at a minimum 10-year follow-up after knee dislocation surgery. Knee Surg Sports Traumatol Arthrosc. 2017;25(12):3914-3922.
56. King AH, Krych AJ, Prince MR, Pareek A, Stuart MJ, Levy BA. Surgical outcomes of medial versus lateral multiligament-injured, dislocated knees. Arthroscopy. 2016;32:1814.
57. Levy NM, Krych AJ, Hevesi M, et al. Does age predict outcome after multiligament knee reconstruction for the dislocated knee? 2- to 22-year follow-up. Knee Surg Sports Traumatol Arthrosc. 2015;23:3003–7.
58. King AH, Krych AJ, Prince MR, Sousa PL, Stuart MJ, Levy BA. Are meniscal tears and articular cartilage injury predictive of inferior patient outcome after surgical reconstruction for the dislocated knee? Knee Surg Sports Traumatol Arthrosc. 2015;23:3008–11.
59. Fanelli GC, Edson CJ. Arthroscopically assisted combined anterior and posterior cruciate ligament reconstruction in the multiple ligament injured knee: 2- to 10-year follow-up. Arthroscopy. 2002;18:703–14.
60. Fanelli GC, Sousa PL, Edson CJ. Long-term follow up of surgically treated knee dislocations: stability restored, but arthritis is common. Clin Orthop Relat Res. 2014;472:2712–7.

2

“Small” Fractures Below the Knee: Do Not Miss—Do Not Mistreat!

Pieter d’Hooghe, B. Krivokapic,
Gino M.M.J. Kerkhoffs, Christiaan van Bergen,
Peter G. van Doesburg, Laura Bloem,
Pietro Spennacchio, D. Cucchi, Joe Wagener,
Christopher diGiovanni, Tonya Dixon,
and Sjoerd Stufkens

2.1 Introduction

Many of the small fractures below the knee are known by eponyms. Although some are well known such as the Maisonneuve and Lisfranc fractures, several are less well known, such as the Cedell and Tillaux fractures. Unfamiliarity with these small fractures may result in failure of detection at initial emergency department surveys or treated suboptimally by lack of experience. This ICL chapter consists of an overview of several common “small” fractures of the foot and ankle, not to be missed, not to be mistreated.

2.1.1 Maisonneuve Fracture [1–15]

The Maisonneuve fracture is on this list because of its reputation to be overlooked, not because of its benign nature. On the contrary, it is an ankle fracture by definition; suboptimal treatment may predispose the ankle to the onset of posttraumatic osteoarthritis (Fig. 2.1). Pankovich appreciates five stages of the Maisonneuve fracture: rupture of the anterior talofibular ligament (ATFL), rupture of the interosseous membrane, fracture or rupture of the posterior talofibular ligament (PTFL), rupture of the anteromedial joint capsule, and fracture of the fibula and a rupture of the deltoid ligament or fracture of the medial malleolus. Since 7–15% of the body weight is transferred through the fibula, shortening will lead to lateral tibiotalar overload. Late repairs of syndesmotic injuries have less favorable outcome than primary stabilization.

2.1.2 Posterior Malleolus Fracture [16–21]

Approximately 7–44% of ankle fractures have involvement of a posterior tibial fragment. Patients with fractures that include a posterior tibial fragment tend to have a poorer prognosis than fractures

P. d’Hooghe • B. Krivokapic
Aspetar Orthopaedic and Sports Medicine Hospital,
Doha, Qatar
e-mail: pieter.orthopedie@gmail.com

G.M.M.J Kerkhoffs • S. Stufkens (✉)
Department of Orthopaedic Surgery, Academic
Center for Evidence-Based Sports Medicine,
Academic Medical Center,
Amsterdam, The Netherlands
e-mail: stufkens@gmail.com

C. van Bergen • P.G. van Doesburg • L. Bloem
Spaarne Gasthuis, Hoofddorp, The Netherlands

P. Spennacchio • D. Cucchi
Clinique du Sport, Centre Hospitalier de
Luxembourg, Luxembourg, Luxembourg

J. Wagener
Team Fuss und Sprunggelenk, Kantonsspital
Baselland, Liestal, Switzerland

C. diGiovanni • T. Dixon
Department of Orthopedics, Massachusetts General
Hospital, Boston, MA, USA

G.M.M.J. Kerkhoffs et al. (eds.), *ESSKA Instructional Course Lecture Book*,
https://doi.org/10.1007/978-3-662-56127-0_2

Fig. 2.1 Missed Maisonneuve fracture

without posterior involvement. Haraguchi and colleagues classified posterior malleolar fractures into three types, based on pathoanatomy of posterior malleolar fragments. The deep deltoid ligament can be attached to the posteromedial fragments, which has significant implications for stability. There seems a remarkable preference to fix Haraguchi type I fractures. These larger posterolateral fragments are best visible on plain lateral radiographs. Posteromedial fragments are at risk of being overlooked and undertreated and may lead to persisting medial instability in cases of malunion.

2.1.3 Tillaux Fracture [22–37]

Paul Jules Tillaux is credited to have discovered that an anterolateral distal tibial fracture was due to the pull of the anterior inferior tibiofibular ligament. In adolescents, physeal closure follows a predictable pattern from the anterolateral aspect of the medial malleolus to the posteromedial physis, then the posterolateral, and finally the anterolateral aspect. Because the distal lateral tibial growth plate is still open, adolescent Tillaux fracture is classified as a Salter-Harris type III epiphyseal fracture or, rarely, as a Salter-Harris IV fracture, of the distal tibia.

2.1.4 Osteochondral Talar Fracture [38–47]

Osteochondral talar fractures are rarely seen as a fresh injury. However, they are a commonly encountered foot and ankle disorder in an elective practice. In the majority of cases, patients with this pathology have a history of ankle sprains and/or fractures. Internal fixation of an osteochondral talar defect shows good results in the literature. However, in most studies, arthrotomies with or without a malleolar osteotomy were performed to fixate the osteochondral defects (OCDs).

2.1.5 Lateral Talar Process Fracture [48–59]

The lateral talar process provides stability to the ankle mortise and forms the talofibular and subtalar articulations. A lateral process fracture comprises 6% of all ankle fractures and 24% of fractures of the talar body. A lateral talar process fracture should be evaluated as an impact and crush injury instead of an avulsion injury. Because of the mechanism of injury, a lateral process fracture is often seen in snowboarders. Nonunion rates of 60% are found in missed or conservatively treated lateral talar process fractures. Nonunion rates of only 5% are found in lateral talar process fractures managed operatively.

2.1.6 Cedell Fracture [60–65]

Carl-Axel Cedell, a Swedish orthopedic surgeon, first described four cases of posteromedial talar tubercle fractures. This fracture is rare and often missed in the initial diagnostic setup. The mechanism of injury can be due to direct or indirect trauma. Patients usually present with clinical pain over the posteromedial aspect of the ankle. The physical examination reveals ecchymosis and tenderness over the posteromedial aspect of the talocalcaneal joint and the posterior aspect of the medial malleolus.

2.1.7 Anterior Calcaneal Process Fracture [66–71]

Anterior calcaneal process fractures are among the most frequently overlooked and underestimated foot injuries. It is held that these are either missed completely or not adequately diagnosed in about 30–40% of cases. The central bifurcate ligament acts as a pivot of the Chopart joint as a whole. Avulsion fractures of the anterior process should be operatively fixated whenever possible.

2.1.8 Lisfranc Injury [72–82]

Compared to many other injuries involving the musculoskeletal system, the overall incidence of Lisfranc injuries is low, with published rates approximating 0.2–0.4% of all midfoot injuries. Lisfranc injuries are still frequently missed by the unsuspecting clinician because initial radiographic evidence can be occult, especially with lower energy injury. The patients often exhibit plantar ecchymosis on examination of the midfoot region, which, when present, should mandate a high index of suspicion for possible Lisfranc injury.

2.2 State-of-the-Art Treatment

2.2.1 Maisonneuve Fracture [1–15]

Late repairs give satisfactory but less favorable outcome compared to properly treated acute injuries. In the largest series reported of operatively treated Maisonneuve fractures, 92% of patients had good or excellent clinical outcomes. Radiographic evidence of osteoarthritis was observed in 49% of patients. According to systematic reviews, the medial malleolus should be fixed in case of a fracture. For the fibula, one or two 3-and 4-cortical screws can be used. There is no recommendation for proximal fibula fracture fixation. Neither is there a recommendation for direct repair of the deltoid ligament. Suture buttons have become a viable alternative to screw fixation. A recent randomized controlled trial shows that syndesmotic screw and TightRope fixation (Fig. 2.2) result in a low malreduction rate (5%), and both methods maintained reduction well (syndesmotic screw 84% and TightRope 95%). Intraoperative or immediate postoperative control of fibular reduction in the mortise is necessary, since there is malreduction in 6–52% of the cases. It is not possible to conclude from the type of injury, type of treatment, or experience of the surgeon whether an increased risk of persistent dislocation is present. A nonanatomical reduction outcome must therefore be expected in many cases. The currently available literature does not support routine elective removal of syndesmotic screws. Secondary procedures increase overall healthcare costs and expose the patient to additional risk of complications. Therefore, in the absence of high-quality evidence, there appears to be little justification for routine removal of syndesmotic screws.

Fig. 2.2 Syndesmotic screw and TightRope fixation

2.2.2 Posterior Malleolus Fracture [16–21]

Diagnostic accuracy of measuring on plain lateral radiographs to assess articular involvement of posterior malleolar fractures is 22% (Fig. 2.3). Surgeons should no longer solely rely on plain lateral radiographs to judge the pathoanatomy of posterior fragments in ankle fractures. The size of the posterior malleolar fragment is long thought to be of relevance for decision-making. However, larger posterolateral fragments may be left unfixated, whereas smaller posteromedial fragments should be fixated since the deep deltoid ligament is attached to the posterior colliculus of the medial malleolus. Arthroscopically assisted percutaneous reduction and fixation of posterior malleolar fragments should be considered when the surgeon has the skills and ability. Fixation of a posterior malleolus provides 70% of stability whereas syndesmotic screws provide 40%.

2.2.3 Tillaux Fracture [22–37]

The correct diagnosis and appropriate treatment of the Tillaux fracture are of extreme importance because this fracture involves a major weight-bearing articular surface. However, treatment protocols in the literature are not uniform for this kind of fracture, and numerous case reports can be found describing various treatment methods. Anatomical reduction and internal fixation are required for every displaced epiphyseal fracture, especially in cases with more than 2 mm fragment displacement. This cutoff value is relevant because a gap of more than 2 mm on plain radiograph can lead to posttraumatic osteoarthritis.

Initial management with a closed reduction can be performed in the emergency room. To reduce a Tillaux fracture, the foot must be plantar flexed and then internally rotated; finally, the ankle must be maximally dorsiflexed. An assistant should stabilize the knee at 90° during this

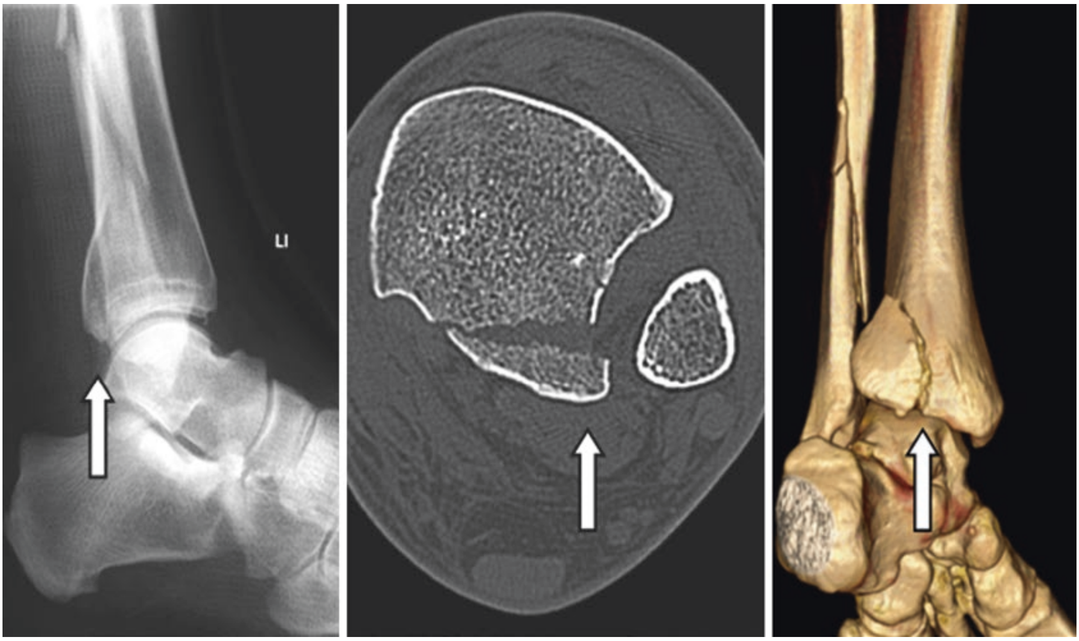


Fig. 2.3 Articular involvement of posterior malleolar fractures

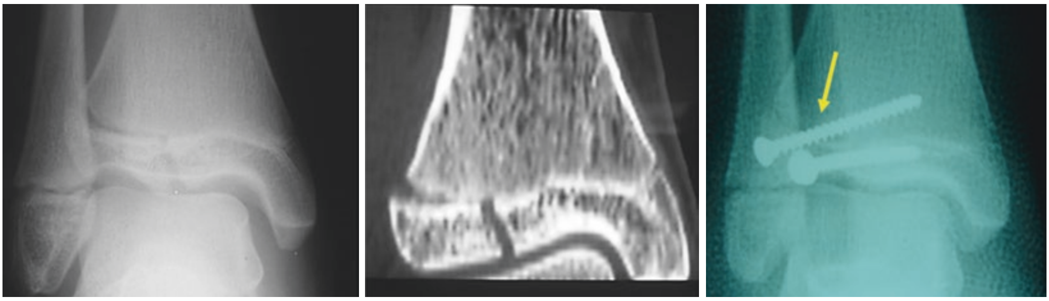
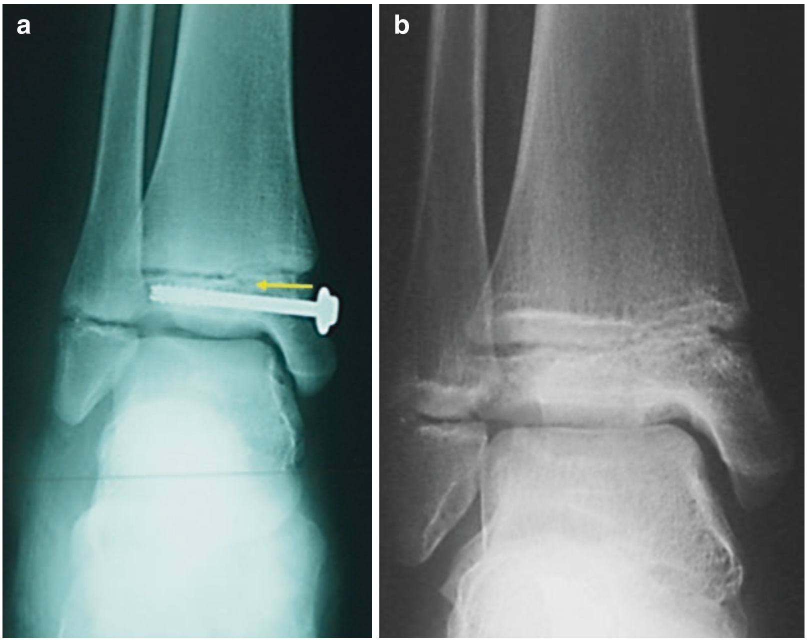


Fig. 2.4 Closed reduction and percutaneous fixation

Fig. 2.5 (**a**) Percutaneous screw fixation of a Tillaux fracture (yellow arrow), respecting the epiphyseal plate. (**b**) 7 months postoperative radiographic control after screw removal



manipulation. The closed reduction is followed by casting or splinting; a CT scan is then mandatory to confirm the adequacy of reduction, with a residual gap or step-off of <2 mm being considered acceptable.

If, after close reduction and casting, a residual step-off of >2 mm in any plane persists, operative treatment is indicated. Several surgical techniques have been described to treat displaced Tillaux fractures, which can be divided in closed reduction and percutaneous fixation (Figs. 2.4 and 2.5a, b), mini-open reduction and internal fixation, and open reduction and internal fixation.

A mini-open technique is defined as a small incision used to manipulate the fracture for reduction with an instrument under fluoroscopy but without formal articular visualization or exposure. More recently, arthroscopically assisted reduction and fixation has also been proposed. The treatment of choice of Tillaux fractures is dependent on the fracture displacement, stability of the fracture, articular congruity, and presence of associated injuries.

Early treatment is recommended, although acceptable results were also obtained with a 5-week delayed treatment. If screw fixation is performed, the screw should be removed within 1 year, since cannulated screw removal from the distal tibial epiphysis after more than 1 year postoperatively can be often complicated by screw breakage and screw head stripping.

2.2.4 Osteochondral Talar Fracture [38–47]

The distinction between fresh and chronic osteochondral lesions is difficult to make. There is a wide variety of treatment regimens for chronic lesions, whereas for fresh osteochondral fractures, fixation is to be preferred (Fig. 2.6a, b). There are several studies showing osteochondral lesions to occur as a result of ankle fractures. However, there is a paucity in the literature regarding the epidemiology of fresh osteochondral fractures. Probably, there is a role for conservative treatment, since 61.5% yields successful results in chronic lesions. For chronic lesions, none of the interventions for the treatment of primary osteochondral defects to the talus show clinical superiority over another or others. Internal fixation of a large enough fresh or chronic osteochondral talar defect is a good technique. The advantage is to restore the natural congruency of the subchondral bone and to preserve hyaline cartilage. However, often, a medial or lateral arthrotomy, often combined with a malleolar osteotomy, has to be performed to allow proper visibility and working access.

We advise an arthroscopic fixation technique for primary osteochondral talar defects: lift, drill, fill, and fix (LDFF). The contour of the anterior tibia can be identified and the distal tibia rim removed with a shaver to facilitate better access to the ankle joint. The arthroscopic portals should be interchangeably used to allow optimal vision. With a probe, the location of the OCD is identified, and a beaver knife is used to allow the making of a sharp osteochondral flap. The posterior side of the flap is left intact and can be used as a lever, allowing lifting from anteriorly with the use of a chisel (lift). The attached bone of the osteochondral flap and the osteosclerotic area of the bed can be debrided and drilled to promote revascularization (drill) in case of older lesions. After debridement and drilling, the defect can be filled with cancellous bone of the distal tibial metaphysis. Cancellous bone is harvested with a chisel by creating longitudinal particles that are transported into the defect with a grasp (fill). Finally, the osteochondral flap can be correctly aligned and fixed with a bio-screw (fix). Clinical success rates between 78% and 89% after fixation through an open procedure are reported.

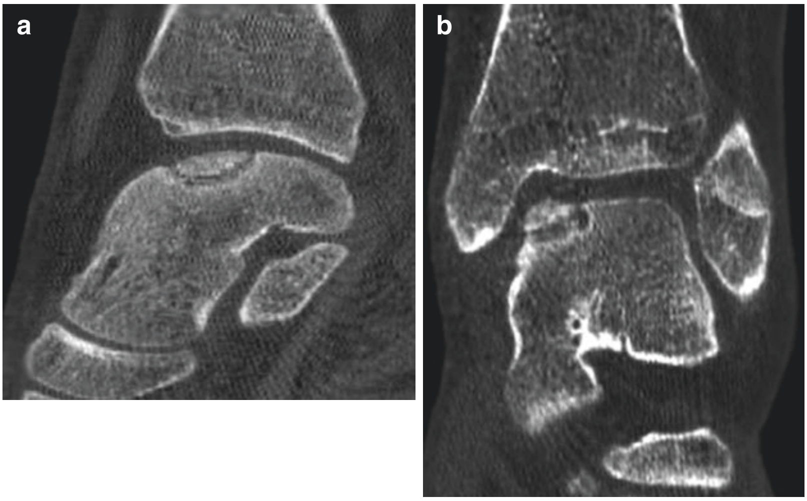


Fig. 2.6 (**a**) Large osteochondral fracture suitable for fixation. (**b**) Large osteochondral fracture suitable for fixation

Long-term outcomes after arthroscopic treatment are not yet available; however similar or higher values could be expected.

2.2.5 Lateral Talar Process Fracture [48–59]

A lateral talar process fracture is often misdiagnosed as an ankle sprain, assuming there is only soft tissue damage. Of all lateral talus fractures, 33–59% are missed on initial presentation. Misdiagnosis and undertreatment can lead to malunion or nonunion, eventually resulting in osteoarthritis. This can have severe consequences for the quality of life in the young and active patients suffering from this injury. Therefore, the diagnostic workup is essential.

The physician evaluating a patient with lateral ankle pain should suspect a lateral talar process fracture after a high-impact trauma or after snowboarding. Patients with a lateral talar process fracture present with pain, swelling, and hematoma. Palpation anteroinferior to the lateral malleolus is frequently painful. The lateral malleolus itself may also be painful. In most cases, the Ottawa ankle rules are positive in patients with a lateral talar fracture, leading to the first step in diagnostic imaging: radiography.

Standard radiography is false negative in 21–40% of the cases. The fracture is best established on a mortise view or Broden’s view. Chip fractures might overproject on the fibula and calcaneus and are therefore better seen on the lateral view.

An intact lateral process of the talus has a symmetrical V-shaped contour. A crooked or asymmetrical V-shape can be seen in a displaced fracture. Von Knoch et al. described this as a positive V-sign. A posterior subtalar effusion on the lateral ankle radiograph raises suspicion of a lateral talar process fracture. Holding the ankle in dorsiflexion and inversion can contribute to a better view of the fracture.

After diagnosing a lateral talar process fracture on plain radiography, a computed tomography (CT) scan should be made to visualize the type of fracture, amount of displacement, comminution, and involvement of the subtalar joint (Fig. 2.7). A CT scan can also be diagnostic in patients with a

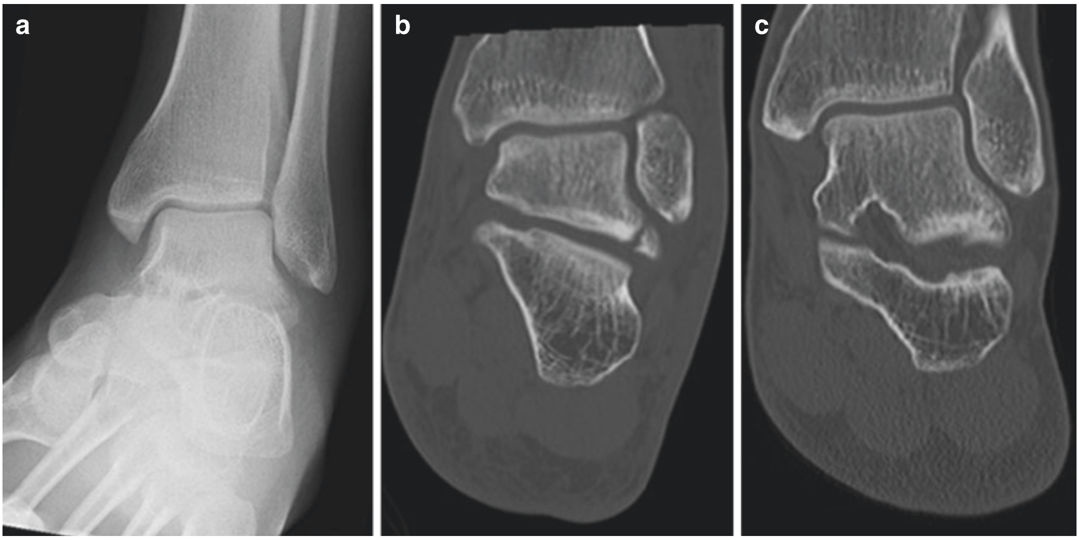


Fig. 2.7 A 30-year-old male with a lateral talar process fracture (**a**). The CT scan showed a Hawkins type I fracture with 3 mm dislocation (**b**). Despite the advice to perform open reduction and internal fixation, the patient chose a conservative treatment. Immobilization in a short-leg, non-weight-bearing cast for a period of 6 weeks was provided. The patient was seen 2 years after the initial treatment with persisting ankle pain. Physical examination revealed a limited ankle dorsiflexion. The CT scan showed a consolidated lateral talar process (**c**) and moderate subtalar arthrosis

high suspicion at clinical presentation but negative findings on radiography. Moreover, CT is able to detect early degenerative changes and visualize additional pathology like concomitant ankle fractures, joint dislocations, or soft tissue damage. In addition, Kramer et al. proposed magnetic resonance imaging to rule out syndesmosis injury.

To achieve normal ankle and subtalar function, it is necessary to restore the shape of the talus and the articular surfaces. The treatment of a lateral talar process fracture is dependent on the classification of Hawkins. Nondisplaced or minimally displaced type I and type III fractures can be managed without surgery. Type I and type III fractures with two or more millimeters of displacement, as well as most type II fractures, usually require surgery. In case of delayed diagnosis of the fracture, excision of the fragment is commonly recommended, although refixation might be possible in select cases. Subtalar arthrodesis might be necessary when the fracture has been neglected and the patient has developed posttraumatic arthrosis of the joint (Fig. 2.8).

Conservative or nonsurgical treatment includes immobilization in a short-leg, non-weight-bearing cast for a period of 6 weeks. After 6 weeks of immobilization, the initial treatment is followed by physiotherapy exercises to increase muscular strength, proprioception, and range of motion as well as to improve gait. Patients who fail conservative casting and immobilization with continued pain and symptoms should undergo a secondary debridement.

For large, displaced lateral talar process fractures, open reduction and internal fixation is recommended. For small, displaced, or highly comminuted lateral talar process fractures, every effort should be made to restore the articular congruity of the subtalar joint; only if the fragments are too small for secure fixation, fracture fragment debridement is indicated.

Lateral talar process fractures are approached through the sinus tarsi by an incision from the midpoint of the lateral malleolus to the calcaneocuboid joint, with distal reflection of the extensor brevis muscle. The incision goes directly over the lateral talar process and along the floor of the sinus tarsi. The subtalar joint capsule needs to be opened to inspect for loose fragments. Fixation of suitable fractures can be accomplished with (mini-fragment) screws and possibly plates. Fracture fragment debridement should be done only when the fragments are too small to support fixation. It is important to remove all intra-articular debris to reduce the risk of arthritis.

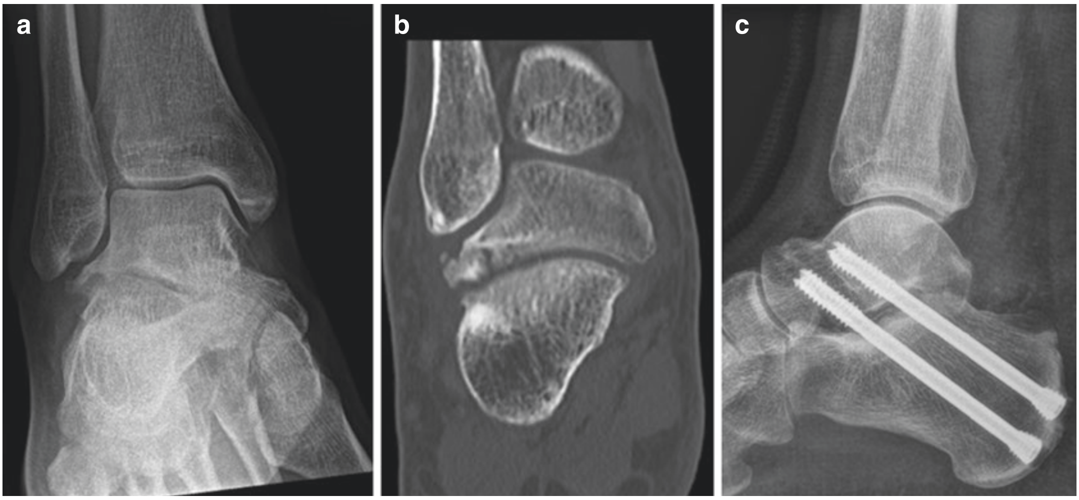


Fig. 2.8 A 44-year-old male who had had an ankle sprain 1 year prior, presented to the orthopedic department with persisting ankle pain. Physical examination revealed a swollen ankle joint and stiffness of the subtalar joint with recognizable pain. On the radiographs, an irregular surface of the lateral talar process was seen (**a**). The CT scan revealed a consolidated lateral talar process fracture as well as subtalar arthrosis (**b**). Because of the severe complaints, a subtalar fusion was performed (**c**)

2.2.6 Cedell Fracture [60–65]

Radiographic imaging is very important to confirm the clinical suspicion. A standard anteroposterior, lateral, and oblique radiograph should be obtained. The foot is placed on the film cassette as if a true anteroposterior view of the ankle is to be taken. Then the foot and ankle are externally rotated 40°. The beam is centered at 1 cm posterior and 1 cm inferior to the medial malleolus and is tilted caudal to cephalad at 10° (Fig. 2.9). A forced hyper-plantar flexion test with the patient sitting and with the knee flexed to 90° can raise a clinical suspicion to the diagnosis of a Cedell fracture (Fig. 2.10).

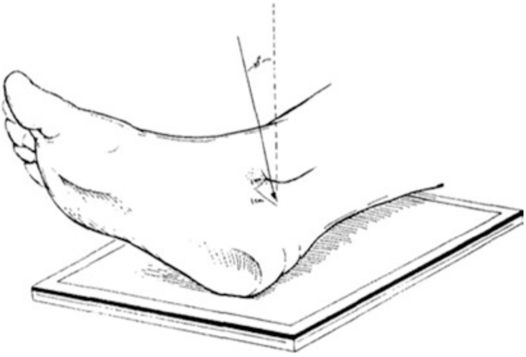


Fig. 2.9 A standard anteroposterior, lateral, and oblique radiograph should be obtained. The foot is placed on the film cassette as if a true anteroposterior view of the ankle is to be taken. Then the foot and ankle are externally rotated 40°. The beam is centered at 1 cm posterior and 1 cm inferior to the medial malleolus and is tilted caudal to cephalad at 10°

Fig. 2.10 A forced hyper-plantar flexion test with the patient sitting and with the knee flexed to 90° can raise a clinical suspicion to the diagnosis of a Cedell fracture

In his article on four patients in 1974, Cedell proposed operative treatment with bony fragment excision on all cases. An extra-articular or undisplaced small fracture of the medial tubercle is usually marginal, and best treatment remains a short-leg cast or orthosis and partial weight-bearing for 6 weeks. All displaced fractures and fractures involving the joint should be treated operatively to minimize the risk of early subtalar degenerative changes.

Therefore, small displaced fragments measuring <0.5 cm should be managed with subtalar arthroscopic excision. Displaced fragments measuring 0.5–1 cm should be evaluated arthroscopically while also evaluating possible chondral lesions, and according to the mentioned findings, they can be excided or reduced. All fracture fragments measuring >1 cm should be treated operatively whether through arthroscopic or ORIF fashion, depending on the surgical expertise and preference.

2.2.7 Anterior Calcaneal Process Fracture [66–71]

Standard radiographs with suspected Chopart joint injuries include dorsoplantar (anteroposterior), lateral, and 45°oblique projections of the foot. For obtaining the dorsoplantar view, the tube is tilted caudally 30°. If a bony injury at the Chopart joint is seen or suspected on plain radiographs, CT scanning should be used generously to reveal the exact fracture anatomy and to allow for adequate treatment planning.

Nondisplaced fractures can be treated conservatively by plaster immobilization for 6 weeks (in case of limited involvement of joint surfaces, type II fractures). An open reduction and internal fixation should be recommended however, when there is dislocation and joint surface involvement; small fragments and multifragment fractures are treated by excision. The anterior calcaneal process is visualized via an oblique anterolateral approach, ideally starting at the sinus tarsi and extending to the calcaneocuboid joint. The incision is carried out above and parallel to the peroneal tendons. Care is taken not to injure the sural nerve. The joint is visualized and the lateral column brought out to length using a mini-distractor (i.e., a Hintermann spreader) when necessary. In case of depressed fragments, the joint-bearing fragment is then mobilized toward the articular surface together with the subcortical bone using the uninjured surface of the cuboid as a template. The anterior process of the calcaneus is fixed with short T-shaped or L-shaped plates. The use of locking plates seems useful to sustain the length of the lateral column of the foot. Simple fractures of the upper part of the anterior process can be fixed with 1.5–2.7 mm screws. The decision whether an excision or reduction and fixation of the fragment is indicated can be made intraoperatively. Anatomic refixation of larger intra-articular fragments, which represents the optimal strategy, is technically challenging. Type III fractures usually show cartilage damage of the CC joint due to the often delayed diagnosis and treatment.

2.2.8 Lisfranc Avulsion Fracture [72–82]

The so-called Lisfranc ligament is actually only one of many ligaments in this complex, but it is the largest and most anatomically distinct of these structures, running obliquely from the medial cuneiform to the base of the second metatarsal—and it provides the greatest degree of ligamentous support to the metatarsal arch of all ligaments within the midfoot. While the midfoot capsuloligamentous complex in the Lisfranc region exists both dorsally and plantarly, the strongest and most important of these structures is plantar.

One anatomic pearl to remember about this midfoot region is that one of the more common reasons to develop compartment syndrome in the foot—an unusual but documented problem that can occasionally occur in the face of a Lisfranc injury—is that the first branch of the dorsalis pedis artery traverses the 1–2 interspace in this region and can be torn with injury or surgery in this region. Attention should be paid to this structure during any treatment of a Lisfranc injury (Fig. 2.11).

Fig. 2.11 First branch of the dorsalis pedis artery

The particular hallmark that can often be recognized in midfoot injuries when they occur in subtle fashion, however, is an avulsion fracture of the Lisfranc ligament involving the base of the second metatarsal—the so-called fleck sign (Figs. 2.12 and 2.13). While this small fracture does not occur in all of these injuries, not infrequently, it can represent the only manifestation of an important underlying condition that requires surgical attention and thus should not be missed. It can be argued that ensuring anatomic realignment and conferring stability to the midfoot after an occult Lisfranc injury (highlighted only by the presence of a midfoot avulsion fracture) are as functionally important as restoring Lisfranc midfoot integrity after a homolateral dislocation that involves tarsometatarsal (TMT) joints 1–5.

Traditionally, Lisfranc injury has been diagnosed when ≥2 mm displacement existed between normally congruent articulations within the TMT

Fig. 2.12 Weight-bearing radiographs now demonstrating increased gap between the first and second metatarsals; fleck sign is now visible off the second metatarsal

Fig. 2.13 Stress radiograph demonstrating clear instability of the middle column via disruption of the medial alignment of the second TMT joint. A positive fleck sign is seen here, demonstrating avulsion of the Lisfranc ligament. The first TMT joint can also be noted to have incongruency

complex, although in more recent times, Lisfranc injury is questioned if any irregularity or disruption exists between any of these normal anatomic relationships across TMT joints 1–5. Radiographs should be assessed carefully to assess alignment of the borders of each metatarsal base with its, respectively, articulating tarsal bone rather than any gapping between the metatarsals themselves, and attention should also be focused on the more proximal midfoot, since occasionally there can be superimposed intercuneiform instability as well. While the first and second articulations are best viewed on the anteroposterior foot radiograph, the third to fifth TMT joints are best viewed on the oblique radiograph. During stress examination for identification of occult Lisfranc injury (often seen with the so-called Lisfranc avulsion), the examiner applies an abduction force across the forefoot to stress the midfoot, which can often highlight subtle instability across one or more TMT joints radiographically.

Over the past few decades, a variety of treatment options have been described for this injury, and it is important to note that surgical approaches vary from percutaneous exposures to open approaches using one or several incisions, depending on the severity of the injury. Fixation options include Kirschner wires, trans-articular screws, dorsal plating, or suture button fixation—

although in recent years the former has somewhat fallen out of favor because of its decreased ability to maintain reduction over time. This is noteworthy since the outcome of Lisfranc surgery in many reports is most predicated not only on the nature or "personality" of the originating injury but also on the surgeon's initial ability to both obtain and maintain anatomic reduction over time. Over the past four decades, recommendations for fixation have thus interestingly evolved from Kirschner wires to trans-articular screws to dorsal plating (to avoid further articular damage) to primary fusion or suture button fixation.

For the majority of Lisfranc avulsion injuries (which typically involve the second TMT joint in isolation), the past 5 years has seen a surge of management with percutaneous or limited open reduction with either one or two screws or suture buttons across the unstable TMT (Fig. 2.14). It remains unclear as to which of these constructs offers greater advantage, and it remains controversial as to exactly where the hardware should be placed or whether or not it should be retained or removed following adequate time to allow for healing. Recovery from these injuries typically takes 2–3 months until weight-bearing progression in a regular shoe with arch support and 1 year to reach maximum medical improvement. Arthritic change in these joints can occur, but in the face of anatomic realignment, this often takes decades or more to be of any consequence to patients, and most do quite well with appropriate surgical management and rehabilitation.

Fig. 2.14 Postoperative anteroposterior radiograph of a 22-year-old male who sustained an avulsion type II TMT Lisfranc injury after being struck by a car while riding his motorcycle

2.3 Future Treatment Options

Regarding the treatment of Maisonneuve fractures, the future may lie in thoughts on preoperative evaluation. There may be advancements in imaging with weight bearing. Also, intraoperative tools such as 3D guiding or navigation for reduction may be beneficial. Guidelines on how to tension the suture buttons, which force to apply, and in which position of the foot are to be investigated. Arthroscopic evaluation and guidance for reduction could be useful. Hypothetically, the deltoid ligament could play a key role in Maisonneuve injuries, and an adequate deltoid ligament reconstruction would be stable enough to restore the reduction of the talus in the mortise. In his classic article, Boden already stated: Biomechanically, the syndesmosis is a secondary stabilizer to the primary restraint against talar translation: the deltoid ligament.

For posterior malleolar fractures, as for other intra-articular fractures, it is a fact that satisfactory outcomes are seen despite residual incongruity. Therefore, it is fair to question the need for perfect reduction in all cases. We do not know if some joints are able to tolerate incongruity better than others. Our ability to assess the quality of reduction is limited, and our understanding of tolerance for malreduction is still lacking. Recommendations for future studies include a prospective (long-term) follow-up of ankle fractures with posterior malleolar fragments including pre- and postoperative CT quantification. The influence of comminution, true fracture fragment size (mm^3), 3D fracture morphology, articular involvement (mm^2), residual gap, residual step-off, and other patient-related factors could then be analyzed to discover the most important predictors of functional outcome.

Due to the articular nature of Tillaux fractures, the arthroscopic-assisted approach guarantees the direct visualization of the reduction and fixation of the injury, with the advantages of a minimally invasive procedure.

Despite that available evidence fails to show improved outcome associated with this approach, we recommend it as the standard surgical strategy.

Regarding the osteochondral talar fractures, it must be said that the osteochondral autologous transfer system is theoretically an ideal solution. However, donor site morbidity (mostly of the knee) has raised reluctance both in patients and in surgeons. An alternative technique is to use a vascularized corticoperiosteal graft. This technique is not a new idea, but it was recently revived by Hintermann and Schäfer. They identified the medial condyle of the femur as an ideal site from which to harvest a vascularized bone graft, sufficiently large and solid, having a contour similar to that of the talar surface, a consistent perfusing artery and periosteal cover. This technique restores the contour of the talus with a firmly incorporated graft which retains its shape and size and develops an overlying layer of fibrocartilage. The osteoperiosteal graft could also be harvested at the iliac crest, in which case it is implanted nonvascularly.

There is no evidence of biologic therapy to improve outcomes of fresh osteochondral fractures. In chronic lesions, this remains a subject of ongoing debate. Based on available basic and clinical evidence, biologic agents and scaffold-based therapies might show improved clinical and radiological outcomes. A recent systematic review acknowledged that the majority of studies assessing clinical outcomes following operative treatment in osteochondral lesions are of poor methodological quality. Well-designed clinical trials with validated outcome measurements are needed.

Stimulating fracture healing in lateral talar process fractures can be an option as an adjunct in initial treatment or in the case of mal- or nonunion. To improve consolidation in fracture treatment, Buza et al. reviewed the latest treatment options in stimulating bone healing. The review discusses three major treatment options: physical stimulation, local strategies, and systemic biological factors. To date, there is little evidence for any of these treatment options, especially regarding ankle fractures. Nonoperative treatment with pulsed electromagnetic fields, low-intensity pulsed sonography, or shock wave therapy seems promising. Local administration of autologous bone marrow, growth factors, or bone morphogenetic proteins might be beneficial too. Systemic biological factors such as parathyroid hormone and bisphosphonates should be considered carefully, taking the patient and the severity of the local complaints into account.

Regarding Cedell fractures, recently Watenabe introduced a new classification (three types) according to the mechanism of injury and the fracture configuration: (1) avulsion type, (2) split type, and (3) comminuted type. A proposed algorithm for treatment is mentioned below according to this classification (Fig. 2.15).

Ideal treatment options for Lisfranc injuries continue to evolve as we learn more about this injury and evaluate the long-term effects of our various treatment options in different patient populations. The current consensus is that anatomic reduction and rigid fixation are necessary—regardless of implant choice—to maintain the alignment, midfoot arch stability, and sufficient ligamentous healing that promote optimized foot function in the long term. K-wire fixation is generally advised against in all TMT joint constructs except the lateral column, where these are still often employed as a temporary (6 weeks) form of fixation to be later removed in the presence of injury to the most mobile segment of the Lisfranc joint complex. While increasingly advocated by some surgeons as primary management for Lisfranc injuries even in low- or high-energy injuries, primary arthrodesis is still not considered the mainstream treatment choice for most of these patients except in the setting of highly

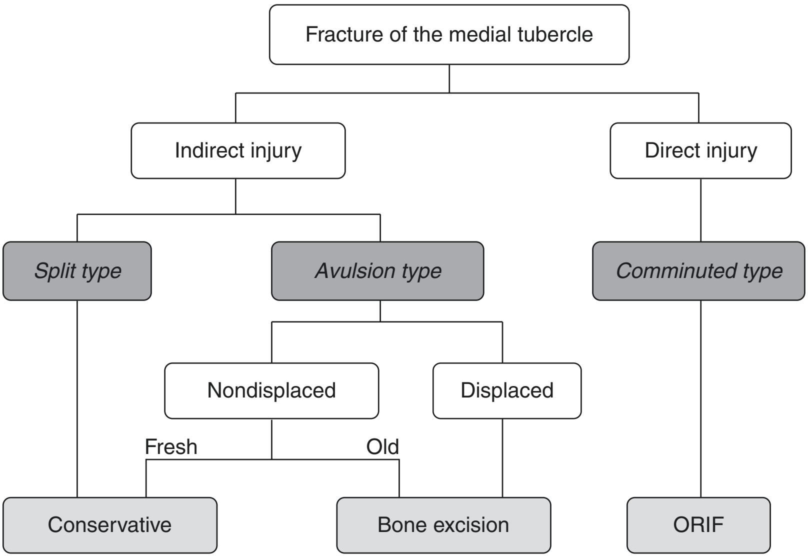


Fig. 2.15 Top one should be: Fracture of the medial tubercle Second row left one should be: Indirect injury Fourth row right one should be: Displaced

comminuted, intra-articular fractures. Debate also continues as to whether or not this should be the primary treatment choice for the elite athletic population and for patients who are expected to endure significant daily impact or loads to the midfoot based on career nature. Further study is necessary to answer these questions.

2.4 Take-Home Message

Maisonneuve fracture: should be suspected in external rotation injuries of the ankle joint. Screws or suture buttons can restore the original anatomy. However, caution is advised as malreductions may occur (approximately 25%). There is no recommendation for proximal fibula fracture fixation. Neither is there a recommendation for direct repair of the deltoid ligament.

Posterior malleolus fracture: involvement is severely misjudged on plain lateral radiographs. Overall, only 22% of measurements on plain radiographs are accurate. Posteromedial fragments are at risk of being overlooked and undertreated and may lead to persisting medial instability in cases of malunion. CTs are recommended in all trimalleolar ankle fractures.

Tillaux fracture: this is a Salter-Harris type III epiphyseal fracture, which demands high attention due to potential consequences for skeletal development. CT is recommended to determine the number of fragments and extent of fracture displacement. Operative treatment is indicated for fracture with displacement of more than 2 mm or articular step-off. Outcomes of Tillaux fracture treatment are generally excellent, provided that anatomic reduction is obtained.

Osteochondral talar fracture: arthroscopic fixation is the first choice for fragments of adequate size. In cases of small lesions, debridement and bone marrow stimulation are viable options. For failed treatment of larger lesions, consider replacement by osteoperiosteal allograft (vascularized or nonvascularized).

Lateral talar process fracture: uncommon ankle fracture with a higher incidence in snowboarders. The fracture is best seen on radiographic mortise view or Broden's view. Most displaced lateral talar process fractures require operative treatment. Long-term disability is especially seen in patients who did not receive proper treatment, as well as in patients who were initially misdiagnosed.

Cedell fracture: uncommon and frequently missed in the initial diagnostic setup. Cast treatment is recommended for nondisplaced fractures or fractures without significant subtalar involvement. Excision is recommended for symptomatic Cedell nonunions.

Anterior calcaneal process fracture: should be considered in the differential diagnosis of a lateral ankle sprain. Early diagnosis allows stable fixation of adequate fragment sizes and can accordingly prevent posttraumatic consequences such as osteoarthritis, collapse, or deformities of the CC joint.

Lisfranc avulsion fracture: even those that are occult, limited to one ray, or associated only with avulsion fracture and with slight incongruity, is best managed in active patient with surgical reduction and stabilization. Controversy persists as to retention or removal of hardware, type of hardware indicated, type of approach indicated, or the need for fusion, but what remains clear is that restoring anatomic alignment and conferring midfoot arch stability are paramount for good outcome in all such patients.

References

1. Pankovich AM. Maisonneuve fracture of the fibula. J Bone Joint Surg Am. 1976;58(3):337–42.
2. Lambert KL. The weight-bearing function of the fibula. A strain gauge study. J Bone Joint Surg Am. 1971;53:507.
3. Ramsey PL, Hamilton W. Changes in tibiotalar area of contact caused by lateral talar shift. J Bone Joint Surg Am. 1976;58:356.
4. Hughes JL, Weber H, Willenegger H, Kuner EH. Evaluation of ankle fractures: non-operative and operative treatment. Clin Orthop Relat Res. 1979; 138:111.
5. van den Bekerom MP, de Leeuw PA, van Dijk CN. Delayed operative treatment of syndesmotic instability. Current concepts review. Injury. 2009;40: 1137.
6. Stufkens SA, van den Bekerom MP, Doornberg JN, van Dijk CN, Kloen P. Evidence-based treatment of maisonneuve fractures. J Foot Ankle Surg. 2011;50:62.
7. Sagi HC, Shah AR, Sanders RW. The functional consequence of syndesmotic joint malreduction at a minimum 2-year follow-up. J Orthop Trauma. 2012;26:439.
8. Tucker A, Street J, Kealey D, McDonald S, Stevenson M. Functional outcomes following syndesmotic fixation: a comparison of screws retained in situ versus routine removal - is it really necessary? Injury. 2013;44:1880.
9. van Dijk CN, Longo UG, Loppini M, Florio P, Maltese L, Ciuffreda M, Denaro V. Conservative and surgical management of acute isolated syndesmotic injuries: ESSKA-AFAS consensus and guidelines. Knee Surg Sports Traumatol Arthrosc. 2016;24:1217.
10. Lintz F, Welck M, Bernasconi A, Thornton BSc J, Cullen NP, Singh D, Goldberg A. 3D biometrics for hindfoot alignment using weightbearing CT. Foot Ankle Int. 2017;38:684.
11. Probasco W, Haleem AM, Yu J, Sangeorzan BJ, Deland JT, Ellis SJ. Assessment of coronal plane subtalar joint alignment in peritalar subluxation via weight-bearing multiplanar imaging. Foot Ankle Int. 2015;36:302.
12. Boden SD, Labropoulos PA, McCowin P, Lestini WF, Hurwitz SR. Mechanical considerations for the syndesmosis screw. A cadaver study. J Bone Joint Surg Am. 1989;71:1548.
13. Franke J, von Recum J, Suda AJ, Vetter S, Grützner PA, Wendl K. Predictors of a persistent dislocation after reduction of syndesmotic injuries detected with intraoperative three-dimensional imaging. Foot Ankle Int. 2014;35(12):1323–8.
14. Kortekangas T, Savola O, Flinkkilä T, Lepojärvi S, Nortunen S, Ohtonen P, Katisko J, Pakarinen H. A prospective randomised study comparing TightRope and syndesmotic screw fixation for accuracy and maintenance of syndesmotic reduction assessed with bilateral computed tomography. Injury. 2015;46(6): 1119–26.
15. Dingemans SA, Rammelt S, White TO, Goslings JC, Schepers T. Should syndesmotic screws be removed after surgical fixation of unstable ankle fractures? A systematic review. Bone Joint J. 2016;98-B(11):1497–504.
16. de Muinck Keizer RO, Meijer DT, van der Gronde BA, Teunis T, Stufkens SA, Kerkhoffs GM, Goslings JC, Doornberg JN. Articular gap and step-off revisited: 3D quantification of operative reduction for posterior malleolar fragments. J Orthop Trauma. 2016;30(12):670–5.
17. van den Bekerom MP, Haverkamp D, Kloen P. Biomechanical and clinical evaluation of posterior malleolar fractures. A systematic review of the literature. J Trauma. 2009;66:279–84.
18. Ferries JS, DeCoster TA, Firoozbakhsh KK, Garcia JF, Miller RA. Plain radiographic interpretation in trimalleolar ankle fractures poorly assesses posterior fragment size. J Orthop Trauma. 1994;8:328–31.
19. Haraguchi N, Haruyama H, Toga H, Kato F. Pathoanatomy of posterior malleolar fractures of the ankle. J Bone Joint Surg Am. 2006;88:1085–92.
20. Weber M. Trimalleolar fractures with impaction of the posteromedial tibial plafond: implications for talar stability. Foot Ankle Int. 2004;25:716–27.
21. Meijer DT, Doornberg JN, Sierevelt IN, Mallee WH, van Dijk CN, Kerkhoffs GM, Stufkens SA, Ankle Platform Study Collaborative – Science of Variation Group. Guesstimation of posterior malleolar fractures on lateral plain radiographs. Injury. 2015;46:2024–9.
22. Choudhry IK, Wall EJ, Eismann EA, Crawford AH, Wilson L. Functional outcome analysis of triplane and

tillaux fractures after closed reduction and percutaneous fixation. J Pediatr Orthop. 2014;34:139–43.

23. Crawford AH. Triplane and tillaux fractures: is a 2 mm residual gap acceptable? J Pediatr Orthop. 2012;32(Suppl 1):S69–73.
24. Duchesneau S, Fallat LM. The tillaux fracture. J Foot Ankle Surg. 1996;35:127–33. discussion 189
25. Horn BD, Crisci K, Krug M, Pizzutillo PD, MacEwen GD. Radiologic evaluation of juvenile tillaux fractures of the distal tibia. J Pediatr Orthop. 2001;21:162–4.
26. Kim JR, Song KH, Song KJ, Lee HS. Treatment outcomes of triplane and tillaux fractures of the ankle in adolescence. Clin Orthop Surg. 2010;2:34–8.
27. Kumar N, Prasad M. Tillaux fracture of the ankle in an adult: a rare injury. J Foot Ankle Surg. 2014;53:757–8.
28. von Laer L. Classification, diagnosis, and treatment of transitional fractures of the distal part of the tibia. J Bone Joint Surg Am. 1985;67:687–98.
29. Landin LA, Danielsson LG, Jonsson K, Pettersson H. Late results in 65 physeal ankle fractures. Acta Orthop Scand. 1986;57:530–4.
30. Liporace FA, Yoon RS, Kubiak EN, Parisi DM, Koval KJ, Feldman DS, Egol KA. Does adding computed tomography change the diagnosis and treatment of tillaux and triplane pediatric ankle fractures? Orthopedics. 2012;35:e208–12.
31. Manderson EL, Ollivierre CO. Closed anatomic reduction of a juvenile tillaux fracture by dorsiflexion of the ankle. A case report. Clin Orthop Relat Res. 1992;276:262–6.
32. Miller MD. Arthroscopically assisted reduction and fixation of an adult Tillaux fracture of the ankle. Arthroscopy. 1997;13:117–9.
33. Nenopoulos A, Beslikas T, Gigis I, Sayegh F, Christoforidis I, Hatzokos I. The role of CT in diagnosis and treatment of distal tibial fractures with intra-articular involvement in children. Injury. 2015;46:2177–80.
34. Panagopoulos A, van Niekerk L. Arthroscopic assisted reduction and fixation of a juvenile tillaux fracture. Knee Surg Sports Traumatol Arthrosc. 2007;15:415–7.
35. Spiegel PG, Cooperman DR, Laros GS. Epiphyseal fractures of the distal ends of the tibia and fibula. A retrospective study of two hundred and thirty-seven cases in children. J Bone Joint Surg Am. 1978;60:1046–50.
36. Thaunat M, Billot N, Bauer T, Hardy P. Arthroscopic treatment of a juvenile tillaux fracture. Knee Surg Sports Traumatol Arthrosc. 2007;15:286–8.
37. Zatti G, D'Angelo F, Giughello A. Delayed diagnosis and treatment of tillaux fracture--a case report. Acta Orthop Scand. 2000;71:327–9.
38. Hintermann B, Regazzoni P, Lampert C, Stutz G, Gächter A. Arthroscopic findings in acute fractures of the ankle. J Bone Joint Surg Br. 2000;82(3):345–51.
39. Berndt AL, Harty M. Transchondral fractures (osteochondritis dissecans) of the talus. J Bone Joint Surg Am. 1959;41-A:988–1020.
40. Reilingh ML, van Bergen CJA, Blankevoort L, Gerards RM, van Eekeren ICM, Kerkhoffs GMMJ, van Dijk CN. Computed tomography analysis of osteochondral defects of the talus after arthroscopic debridement and microfracture. Knee Surg Sports Traumatol Arthrosc. 2016;24:1286–92.
41. Kerkhoffs GMMJ, Reilingh ML, Gerards RM, de Leeuw PAJ. Lift, drill, fill and fix (LDFF): a new arthroscopic treatment for talar osteochondral defects. Knee Surg Sports Traumatol Arthrosc. 2016;24:1265–71.
42. Schuh A, Salminen S, Zeiler G, Schraml A. Results of fixation of osteochondral lesions of the talus using K-wires. Zentralbl Chir. 2004;129:470–5.
43. Hintermann B, Wagener J, Knupp M, Schweizer C, J Schaefer D. Treatment of extended osteochondral lesions of the talus with a free vascularised bone graft from the medial condyle of the femur. Bone Joint J. 2015;97-B(9):1242–9.
44. Leontaritis N, Hinojosa L, Panchbhavi VK. Arthroscopically detected intraarticular lesions associated with acute ankle fractures. J Bone Joint Surg Am. 2009;91(2):333–9.
45. Hintermann B, Boss A, Schafer D. Arthroscopic findings in patients with chronic ankle instability. Am J Sports Med. 2002;30:402–9.
46. Stufkens SA, Knupp M, Horisberger M, Lampert C, Hintermann B. Cartilage lesions and the development of osteoarthritis after internal fixation of ankle fractures: a prospective study. J Bone Joint Surg Am. 2010;92(2):279–86.
47. Yasui Y, Wollstein A, Murawski CD, Kennedy JG. Operative treatment for osteochondral lesions of the talus: biologics and scaffold-based therapy. Cartilage. 2017;8(1):42–9.
48. Dale JD, Ha AS, Chew FS. Update on talar fracture patterns: a large level I trauma center study. AJR Am J Roentgenol. 2013;201(5):1087–92.
49. Vallier HA. Fractures of the talus: state of the art. J Orthop Trauma. 2015;29(9):385–92.
50. Kou JX, Fortin PT. Commonly missed peritalar injuries. J Am Acad Orthop Surg. 2009;17(12):775–86.
51. Wang P, Su W, Jou I. Lateral hindfoot impingement after nonunion of fracture of the lateral process of the talus. J Foot Ankle Surg. 2016;55(2):387–90.
52. Berkowitz MJ, Kim DH. Process and tubercle fractures of the hindfoot. J Am Acad Orthop Surg. 2005;13(8):492–502.
53. Valderrabano V, Perren T, Ryf C, Rillmann P, Hintermann B. Snowboarder's talus fracture: treatment outcome of 20 cases after 3.5 years. Am J Sports Med. 2005;33(6):871–80.
54. Perera A, Baker JF, Lui DF, Stephens MM. The management and outcome of lateral process fracture of the talus. Foot Ankle Surg. 2010;16(1):15–20.
55. von Knoch F, Reckord U, von Knoch M, Sommer C. Fracture of the lateral process of the talus in snowboarders. J Bone Joint Surg Br. 2007;89(6):772–7.
56. Ahmad J, Raikin SM. Current concepts review: talar fractures. Foot Ankle Int. 2006;27(6):475–82.

57. Boack D, Manegold S. Peripheral talar fractures. Injury. 2004;35(Suppl 2):35.
58. Parsons SJ. Relation between the occurrence of bony union and outcome for fractures of the lateral process of the talus: a case report and analysis of published reports. Br J Sports Med. 2003;37(3):274–6.
59. Buza J. Bone healing in 2016. Clin Cases Miner Bone Metab. 2016;13:101.
60. Cedell CA. Rupture of the posterior talotibial ligament with the avulsion of a bone fragment from the talus. Acta Orthop. 1974;45(1-4):454–61.
61. Saxena A. Sports medicine and arthroscopic surgery of the foot and ankle. London: Springer; 2013.
62. Kim DH, Hrutkay JM, Samson MM. Fracture of the medial tubercle of the posterior process of the talus: a case report and literature review. Foot Ankle Int. 1996;17(3):186–8.
63. Ebraheim NA, Skie MC, Podeszwa DA, Jackson WT. Evaluation of process fractures of the talus using computed tomography. J Orthop Trauma. 1994;8(4):332–7.
64. Boack DH, Manegold S. Peripheral talar fractures. Injury. 2004;35(Suppl 2):S-B23–35.
65. Watanabe H, Majima T, Takahashi K, Kawaji H, Takai S. Split fracture of the posteromedial tubercle of the talus: case report and proposed classification system. J Foot Ankle Surg. 2017;56(1):187–90.
66. Rammelt S, Schepers T. Chopart Injuries: when to fix and when to fuse? Foot Ankle Clin. 2017;22(1):163–80.
67. Kotter A, Wieberneit J, Braun W, et al. The Chopart dislocation. A frequently underestimated injury and its sequelae. A clinical study. Unfallchirurg. 1997;100:737–41. [in German]
68. Rammelt S, Grass R, Schikore H, et al. Injuries of the Chopart joint. Unfallchirurg. 2002;105:371–83. [in German]
69. Ochman S, Evers J, Rasehke MJ. Frakturen des processus anterior calcanei. Oper Orthop Traumatol 2013· 25:579–592
70. Degan TJ, Morrey BF, Braun DP. Surgical excision for anterior-process fractures of the calcaneus. J Bone Joint Surg Am. 1982;64:519–24.
71. Backman S, Johnson SR. Torsion of the foot causing fracture of the anterior calcaneal process. Acta Chir Scand. 1953;105:460–6.
72. Watson TS, et al. Treatment of Lisfranc joint injury: current concepts. J Am Acad Orthop Surg. 2010;18:718–28.
73. Easley M, Banerjee R, Nickisch F, DiGiovanni CW. Chapter 61. Foot injuries. In: Skeletal trauma. 4th ed. Philadelphia: Elsevier Saunders; 2008.
74. Schepers T, et al. Influence of approach and implant on reduction accuracy and stability in Lisfranc fracture-dislocation at the tarsometatarsal joint. Foot Ankle Int. 2013;34(5):705–10.
75. Kriz P, et al. Stress fracture of the second metatarsal and sprain of Lisfranc joint in a pre-professional ballet dancer. J Dance Med Sci. 2015;19(2):80–5.
76. Myerson MS, et al. Fracture dislocations of the tarsometatarsal joints: end results correlated with pathology and treatment. Foot Ankle. 1986;6(5):225–42.
77. Anderson RB, et al. Management of common sports-related injuries about the foot and ankle. J Am Acad Orthop Surg. 2010;18:546–56.
78. Myerson MS, et al. Current management in tarsometatarsal injuries in the athlete. Instr Course Lect. 2009;58:583–94.
79. Mulier T, et al. Severe Lisfranc injuries: primary arthrodesis or ORIF? Foot Ankle Int. 2002;23(10):902–5.
80. Sheibani-Rad S, et al. Arthrodesis versus ORIF for Lisfranc fractures. Orthopedics. 2012;35(6): 868–73.
81. Kuo RS, et al. Outcome after open reduction and internal fixation of Lisfranc joint injuries. J Bone Joint Surg Am. 2002;82-A(11):1609–18.
82. Resch S. The treatment of tarsometatarsal injuries. Foot Ankle. 1990;11(3):117–23.

Meniscal Injuries: Management and Outcome

3

S. Konan, M. McNicholas, P. Verdonk, T. Spalding,
A. Price, T. Holland, A. Volpin, I. Pengas, and P.E. Gelber

3.1 Introduction

Meniscal injuries are one of the most common injuries in orthopaedics. Currently, there is a great knowledge about the different type of injuries, as well as a more comprehensive treatment strategy. In this chapter, the most commonly performed treatments for meniscal pathologies are described.

3.2 State-of-the-Art Treatment

3.2.1 Meniscal Repairs and Return to Sport: Long-Term Outcomes

3.2.1.1 Patient Selection and Case Selection

The true prevalence of sports-related meniscal tears is perhaps under-reported. Epidemiologic studies have documented an incidence of meniscus tears requiring surgery at around 60–70 per 100,000 persons, with approximately one-third of these tears attributed to sports [1]. The main challenge for the surgeon when dealing with the athlete population is a return to pre-injury sporting level and minimizing the risk of reinjury.

Meniscal repair is the standard of care in athletic population for tears in the vascular zone; with some benefit in extending this to the less avascular zones [2]. In unstable knees and certain zones/patterns of tear, repair is unlikely to work, and careful selection of patient and case is the key to a successful outcome.

S. Konan
Department of Trauma and Orthopaedics, Organization/University: University College Hospital London, London, UK

M. McNicholas
Trauma and Orthopaedics, Organization/University: University Hospital Aintree, Liverpool, UK

P. Verdonk
Orthopaedic Surgery, Antwerp Orthopaedic Center, Monica Hospitals, Antwerp University Hospital, Antwerp, Belgium

T. Spalding
Trauma and Orthopaedics, Organization/University: University Hospital Coventry and Warwickshire NHS Trust, Coventry, UK

A. Price
Trauma and Orthopaedics, Organization/University: Nuffield Orthopaedic Centre NHS Trust, Oxford, UK

T. Holland
Trauma and Orthopaedics, University Hospital Aintree, Liverpool, UK

A. Volpin
Trauma and Orthopaedics, University College Hospital London, London, UK

I. Pengas
Trauma and Orthopaedics, Royal Cornwall Teaching Hospitals NHS Trust, London, UK

P.E. Gelber (✉)
Orthopaedics Surgery, Hospital de la Santa Creu i Sant Pau, Barcelona, Spain
e-mail: pablogelber@gmail.com

G.M.M.J. Kerkhoffs et al. (eds.), *ESSKA Instructional Course Lecture Book*,
https://doi.org/10.1007/978-3-662-56127-0_3

3.2.1.2 The Procedure: Tips and Tricks

When dealing with meniscal tears, the surgeon should use a combination of one or more techniques depending on the tear pattern and location. Traditionally, the gold standard for meniscal repair has been the inside-out technique. More recently, all-inside repairs have gained popularity and have few surgical risks. All-inside meniscal repair represents the treatment of choice for most surgeons. This technique has shown promising results in 312 repairs performed in 288 patients [3].

Newer arthroscopy instruments have, once again, made inside-out as well as outside-in repair techniques attractive options for meniscal horn tears or tears in white-red zones. It is important to remember that some patterns of tears have better outcomes than others. Similarly, in complex tears, in order to avoid a near-total meniscectomy, it may be beneficial to excise the unstable tears in the avascular edges and repair the tears in the vascularized periphery.

3.2.1.3 Isolated Meniscal Repairs Versus Repair with Ligament Reconstruction

Ligament injuries, in particular anterior cruciate ligament (ACL) injuries, often accompany meniscal tears. Meniscal repair in conjunction with ligament reconstruction has shown better outcomes. This is well documented in the literature (62–96% healing rate in ACL reconstructed group versus 17–22% in patients without ACL reconstruction) [3]. The stability provided by ACL reconstruction and the favourable healing environment from the post-surgery haemarthrosis may be responsible for this finding.

All-inside meniscal repairs have shown satisfactory results, with no particular difference in the type of suture used [4]. Retear of the meniscus following the repair is not uncommon and frequently due to persistent instability in the knee [5].

3.2.1.4 Outcomes in the General Population and in Elite Athletes

Repair of symptomatic meniscal tears in the appropriate patient has demonstrated successful mid- and long-term results with the goal of retaining as much native tissue as possible. Some authors [6] reported a 90.6% success rate with 2.3 years follow-up following meniscal repair. However, at 6.6 years, this success rate had declined to just 71.4% [7]. Another study [8] reported that 81% of 42 athletes, who underwent 45 meniscal repairs, returned to their main sport and most to a similar level at a mean time of 10.4 months after repair. In general, early to mid-term outcomes are excellent with failure usually associated with persistent high-level activity, participation in high-risk sports and/or persistent instability.

3.2.1.5 Rehabilitation

For years, rehabilitation protocols for meniscal repair have largely focused on limiting early postoperative weight-bearing and deep flexion (>90°) [9]. Recently, more aggressive approaches have been used with good success and put these traditional methods into question [10].

VanderHave et al. [11] reviewed outcome and reported both successful clinical outcomes following conservative rehabilitation (70–94%) and after an accelerated rehabilitation protocol with full weight-bearing and early range of motion (64–96%). However, lack of similar objective criteria and consistency among surgical techniques and existing studies made direct comparison quite difficult, and future randomized control trial studies are needed.

3.3 Meniscal Resection: What Next?

3.3.1 What Happens to the Knee After Meniscectomy?

Since King's paper reporting the degeneration of canine knees after meniscectomy in 1936 [12], there has been a gradual increase in the recognition of the importance of the meniscus as a protective structure in articular cartilage degeneration. His astute observation that the proportion of meniscus excised appeared to be related to the subsequent osteoarthritis is now accepted as common wisdom. Twelve years later, Sir Thomas Fairbank's seminal paper "Knee

Joint Changes After Meniscectomy" detailed his observations that the compartment which had been subjected to meniscectomy underwent radiographic degenerative change and suggested that the meniscus played a role in protecting the articular surface during weight bearing [13]. Over the preceding years, the importance of preservation of the meniscus gained momentum. In 1980 Fukubayashi and Kurosawa studied the contact pressures in the knee before and after removal of the menisci finding that the contact pressures rose 20–50% (from 3–4 to 6 MPa) after its removal [14]. The importance of the meniscus and its function in the knee was further supported by the work of Levy and Warren in 1982 demonstrating a role in stability of the ACL-deficient knee [15] (Fig. 3.1).

All these advances have led to our greater understanding of the importance of meniscal preservation, but they have not provided a framework on which we may proceed to do so. In 1991, Sommerlath reported results of meniscal repair in stable knees [16], which showed favourable results, and this work was further supported by Stein in 2010 [17] (Fig. 3.2).

However, there are still a large number of patients for whom meniscal repair is neither possible nor practical. For the young patient with the sub-total meniscectomy, there was a desire to do something more than removing the troublesome meniscus.

There is abundant evidence that meniscectomy leads to radiographic arthritis changes, but how relevant is this to the patient? Orthopaedics is largely a discipline where interventions are designed to aid lifestyle rather than preserve life.

Until fairly recently, there has been scant evidence to link radiographic changes in the menis-

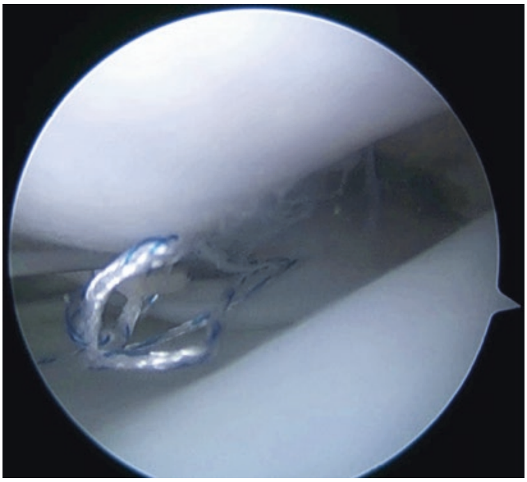


Fig 3.2 Medial meniscus horizontal tear repaired by all-inside suture technique

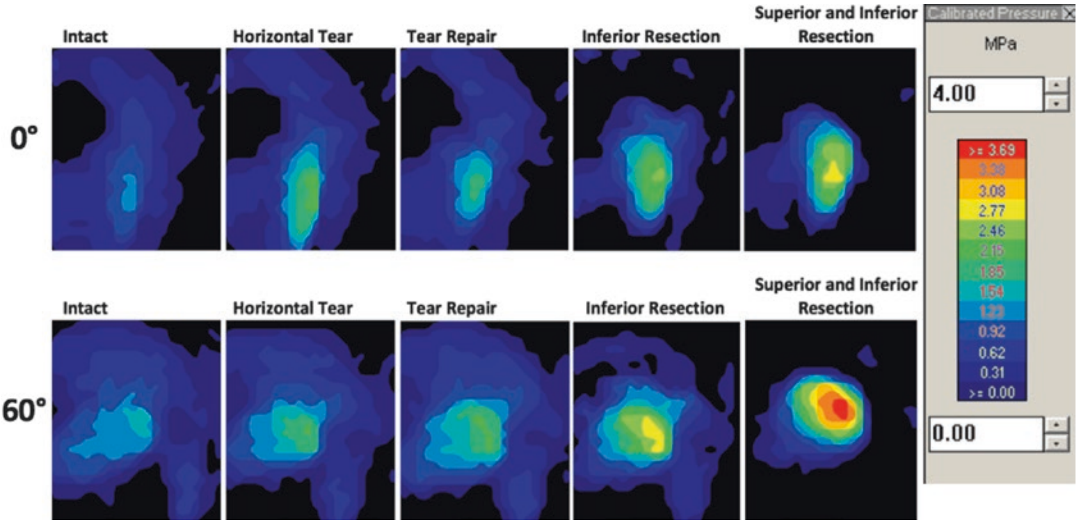


Fig. 3.1 Pressure map demonstrating consequences of medical meniscal lesions and meniscectomy on contact pressures

cectomized knee to decline in function or pain. The work by Pengas et al., published in 2012, has provided evidence that these changes are significant to the patient in terms of long-term knee function [18]. They identified 100 patients whom had undergone open meniscectomy aged under 19 years performed by Professor Iain Smillie in the 1960s and 1970s. They have follow-up data at an average of 17, 30 and 40 years. Thirty-one of these original cohorts were scrutinized at an average of 40-year follow-up, and this cohort was seen to be 132 times more likely to require total knee arthroplasty than an age-matched population. Pengas' work also found that there was an association between radical medial meniscectomy and development of fixed flexion deformity. In addition to this, Khan et al. have demonstrated an association with medial compartment osteoarthritis and progression to varus malalignment, as well as lateral compartment OA and progression of valgus [19]. While patients seldom complain of slight changes in alignment of the limb as their presenting complaint, we know that these changes in the morphology of the knee joint result in an alteration of the mechanical axis of the limb with subsequent alteration of articular wear pattern and a propensity towards osteoarthritis.

Partial meniscectomy is not a benign procedure. Contact stresses in the tibiofemoral joint are increased proportionally after partial meniscectomy [20]. There has been an inverse relationship demonstrated between the amount of meniscus removed and the subsequent knee function by a group in Copenhagen, Denmark [21]. These findings have since been echoed by a Swedish group in 2002 [10].

Allaire et al. showed that not only the extent of the meniscectomy matters but also the location of the excised meniscal tissue [22]. Their work on cadaveric knees found that tears of the posterior horn of the medial meniscus defunctioned the meniscus, leading to contact pressures and kinematic characteristics similar to those seen after total meniscectomy. These findings were reproduced in the Hede et al. paper [21]. This is thought to be as a result of the loss of the meniscus' ability to disseminate hoop stresses. It has become conventional wisdom over the years among knee surgeons that even limited meniscectomy leaves the knee at risk of degenerative joint disease.

These facts lead us to the inescapable conclusion that the meniscus should be preserved wherever possible and that, in the patient for whom meniscal preservation cannot be achieved, an alternative augmentation may be required to prevent hastening of articular cartilage wear.

3.4 Why Augment the Meniscus?

While we should endeavour to preserve the meniscus, it is inevitable that there will be patients whose meniscus is beyond preservation. Here there is a need for an alternative strategy to try to prevent long-term sequelae of meniscectomy. Meniscal augmentation is one such strategy. This was popularized in the early 1990s with the intention of allowing fibrocartilage to develop on a synthetic graft bearing the physical characteristics of the native meniscus [23]. These synthetic prostheses are called meniscal scaffolds. There are currently two meniscal scaffolds used outside of the United States. One of these is made of a porous collagen/GAG matrix. Ninety-seven percent purified type I collagen from bovine Achilles' tendon with approximately 3% GAG proteins attempting to mimic the native meniscus. The other available implant is a synthetic polymer of 80% biodegradable polycaprolactone+20% polyurethane.

3.4.1 How Does Meniscal Scaffold Work?

Animal models for both the collagen meniscal implant and the polyurethane scaffold have demonstrated regeneration of meniscus-like tissue after implantation [24–26]. Furthermore, a work by Verdonk et al. reported that 43 out of 44 patients who underwent implantation with polyurethane scaffolds had demonstrable tissue ingrowth at arthroscopic assessment. This included a biopsy 12 months post-implantation [27].

3.4.2 Who Should Have Meniscal Augmentation with Scaffold?

Patients who may be considered for treatment with meniscal scaffold should have a symptomatic knee with previous meniscal tissue resection of more than 25% of the meniscus, while preserving anterior and posterior roots. It must also maintain an intact meniscal rim as well as a stable knee. Posterior cruciate ligament (PCL) deficiency is a contraindication for scaffold augmentation, but ACL-deficient knees may have concomitant reconstruction or within 12 weeks of implantation. Likewise local/systemic infection, osteonecrosis of the knee and significant malalignment of the knee/limb (affecting the mechanical axis of the knee joint) are also contraindications for implantation. Grade IV articular cartilage damage may be treated with simultaneous surgery, but if left untreated then the scaffold should not be used. One question that remains unanswered is: Should we be treating the asymptomatic knee following sub-total meniscectomy in order to protect the knee? This is a matter for debate, and still there is no scientific data to support this indication.

3.4.3 Technical Considerations

Good arthroscopic skills are essential to performing complex surgery such as meniscal augmentation. Thorough examination of the stability of the knee is required along with arthroscopic assessment of the articular cartilage. Next, the area of damaged meniscus is debrided to a stable rim over the whole length of the defect one aims to address. This area should have “squared off” margins in order to allow for ease of fixation of the scaffold (Fig. 3.3).

The defect is measured (Fig. 3.4) and the graft is prepared by cutting with a sharp blade. It should be oversized by 10%. The scaffold is then placed into the defect and finally fixed by suturing to the squared off edges of the native meniscus (Fig. 3.5) and to the meniscal rim/joint capsule

Once the surgery is complete, the patient should undergo a rehabilitation strategy similar to that of a meniscal repair, avoiding deep flexion while using isometric quadriceps exercises to maintain strength. Resumption to open chain exercise is recommended from 12 weeks and return to sport at 6 months.

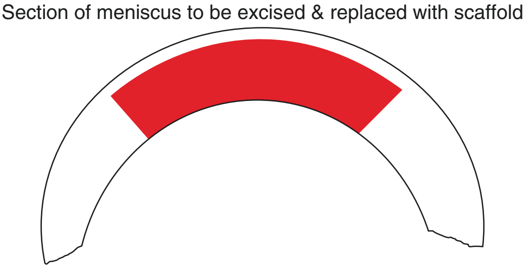


Fig. 3.3 Red section represents area that should be excised prior to scaffold insertion

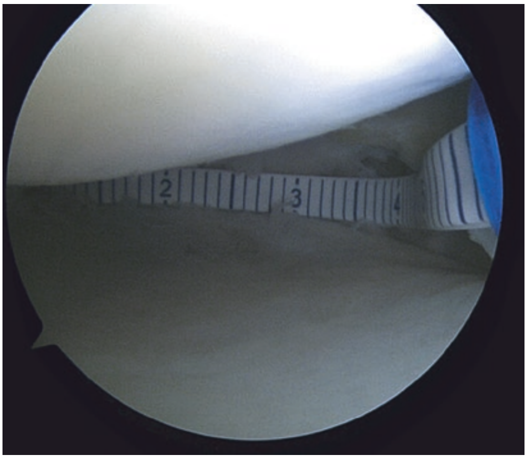


Fig. 3.4 The arthroscopic ruler used

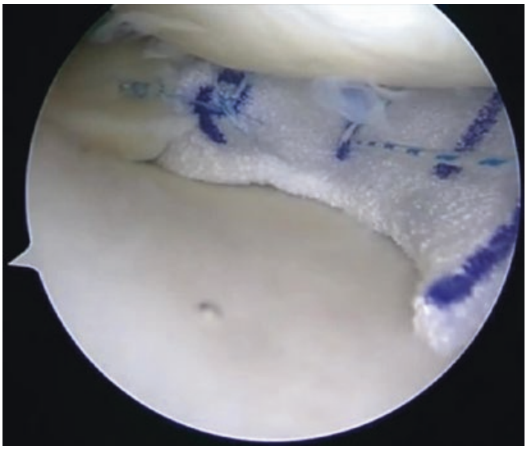


Fig. 3.5 Final position of scaffold (polyurethane scaffold)

3.4.4 Results of Meniscal Scaffold Implantation

The seminal paper often quoted when discussing implantation of the collagen meniscal implant implantation is the randomized multicentre trial by Rodkey et al. published in 2008 [28]. They reported 5-year outcomes of nearly 300 patients comparing the use of the collagen meniscal implant to a medial meniscectomy. Their work found that those patients with chronic symptoms with meniscal deficiency had better outcomes with lower reoperation rates than those who had acute meniscal tears. There is a paucity of evidence examining the polyurethane scaffold by comparison. This is largely due to its more recent authorization for use in Europe. However, short-term results from the work by Spencer et al. in 2012 have suggested that there is no significant difference between the results of the two implants [29]. Further follow-up of the work by Rodkey et al. is awaited with great interest as this will provide a useful insight into the longer-term results following implantation of the collagen meniscal implant.

One thing that can be agreed is that this is an operation indicated only in a specific group of patients who fulfil the criteria for meniscal scaffold use. While there is evidence suggesting that there are favourable results for both the collagen meniscal implant and polyurethane scaffold, more work is needed to validate their use in protection of the articular cartilage of the meniscectomized knee.

3.5 Meniscal Transplant

3.5.1 How Have We Got to Where We Are?

It seems somewhat incredible that the Scottish surgeon Thomas Annandale published the first description of meniscal repair in 1885 [30]. His use of catgut sutures to repair an avulsion of the anterior horn of the medial meniscus followed by 7 weeks in plaster of Paris might seem absurd in modern knee surgery, but he believed that the restoration of the native anatomy was superior to excision of the meniscus. While the techniques for repair of the meniscus have progressed substantially over the years, there are still significant challenges facing the surgeon intending to repair a torn meniscus. Failure of repair may be attributed to many things, including the orientation of the tear, the chronicity of the tear, satisfactory surgical technique for the repair and suitability of the tear for repair in the first instance. Even when all of the criteria are met and there is satisfactory repair of the meniscal tear, it is inevitable that there will be failures. This is where meniscal replacement has been suggested as the next step in management and an effort to avoid the otherwise inevitable degenerative changes seen following meniscectomy.

3.5.2 History of Meniscal Transplant

As with many advances in medical science, the initial work was undertaken in animal models including the work by a Canadian group working with canine knees [31]. Their work highlighted the need for proper sizing of the graft. Their work was quickly followed by the first human meniscal allograft transplant in 1984 by Milachowski in Germany [32]. Subsequent studies have demonstrated incorporation of the transplanted meniscus into the native knee with evidence of healing observed on arthroscopy [33, 34]. Further work from Toronto showed good to excellent Lysholm scores at an average of 31 months in 58/63 patients who had undergone meniscal transplant with allograft [33].

3.5.3 Patient Selection and Surgical Technique

Central to favourable outcome in all aspects of orthopaedics, proper consideration of the patient who will most likely benefit from meniscal transplant is paramount. There are three primary indications for consideration for meniscal transplant:

1. *Post-meniscectomy syndrome*—this is the presence of uni-compartmental pain after meniscectomy. Ideally, there is still minimal to moderate damage to the articular cartilage (ICRS grade I–III). A particular scenario is with the so-called pseudo post-meniscectomy syndrome, where the meniscal tissue was previously lost due to several non-surgical reasons (e.g. chronic bucket-handle meniscal tear in young patients).
2. *Prophylactic meniscal transplant*—this is generally considered in the young, active patient following traumatic meniscal tear requiring meniscectomy. However, there is no scientific data supporting this indication. One exception following the ACL Study Group's recommendation would be a medial meniscus transplant when the ACL is being reconstructed.
3. *Salvage procedures*—a more controversial area where the presence of arthritic changes may limit success. There is some spare evidence that this can prolong the need for arthroplasty. Stone et al. have shown improvement in pain and functional outcomes following transplant in the arthritic knee with mean time to failure of 4.4 years [24].

Patients should have a stable knee with functional ligamentous structures. This may require concomitant ligament reconstruction [35]. Equally, there is a strong argument for simultaneous meniscal transplant with ACL reconstruction when the medial meniscus has been lost. Another key consideration is the mechanical axis of the limb. The presence of mechanical axis deviation may necessitate osteotomy to protect the meniscal transplant and allow for restoration of good functional alignment of the knee. However, with a paucity of evidence, it is not clear from the literature whether simultaneous osteotomy with meniscal transplant or a staged meniscal transplant, should symptoms persist after HTO, is preferable.

Preoperative planning is key to surgical success. Incorrect sizing may lead to premature failure. Sizing of the required graft may be done by plain radiography [36], computed tomography scan [37] or by magnetic resonance imaging [38]. Meniscal transplant may be performed by fixation on a bone block (taken with the meniscus from the donor knee. Fig. 3.6) or by free soft tissue fixation where the meniscal root is sutured directly to the remnant of the recipient knee (Fig. 3.7). There is insufficient evidence to date to conclusively support one method of fixation over another.

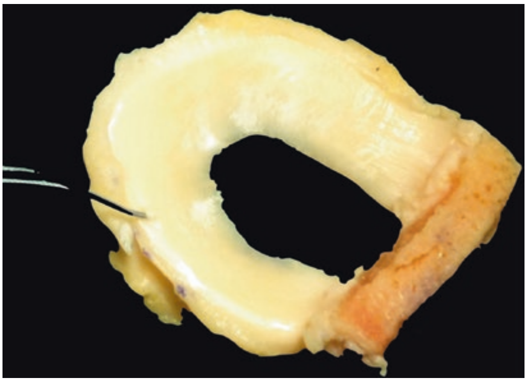


Fig. 3.6 Medial meniscus allograft with bone block

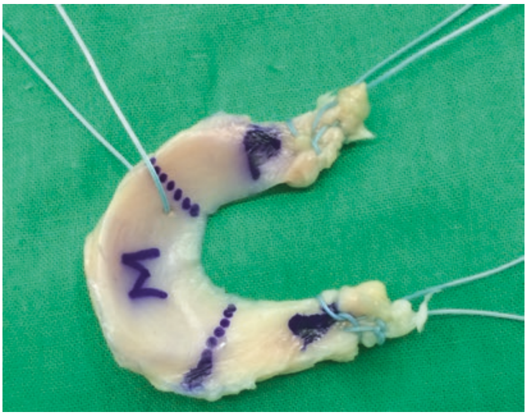


Fig. 3.7 Free lateral meniscus graft with whipstitch

3.5.4 Where Next for Meniscal Transplant?

One thing that may be agreed is that the surgical execution of meniscal transplant is technically challenging and requires a surgeon proficient in complex arthroscopic procedures. There is growing evidence to support this technique, but with its relatively high cost involved, the use of this technique in a publically funded healthcare system remains a matter of great debate. If transplant of the meniscus is to increase in use and become a financially acceptable surgical tactic, then there needs to be more research into how the benefits can be demonstrated to the hospital management and non-knee surgeon alike.

3.6 What Does Success Look Like?

3.6.1 Defining Success

There are abundant tools for scoring throughout orthopaedic practice. These are often quoted in the literature as justification for intervention or to validate results of studies. However, we must never forget that the most important outcomes are not those defined by surgeons but those most important to the patient. If the outcome of knee injury is the curtailment of activity of daily living, cessation of leisure pursuits or need for change of employment, then this is what the patient defines as success or failure.

3.6.2 So What Should We Do?

There is good evidence for meniscal repair throughout the orthopaedic literature. Even those tears previously thought "irreparable" in the white/white avascular zone have been shown to be repaired with satisfactory outcome. Gallacher's work at the Robert Jones and Agnes Hunt Hospital (Oswestry, UK) has demonstrated that, provided a certain number of criteria are met, repair of such tears resulted successful outcome (if reoperation on meniscus for any reason defined failure) in 68% of patients [39]. This echoes the work of Frank Noyes who published results of repair in the avascular zone in adolescents (aged under 20 years) with a 75% success rate [40]. While it can be agreed that meniscus preservation should be the default for the surgeon operating on the symptomatic meniscal tear, we know that this is not always possible. What should be done in those cases?

3.6.2.1 Meniscal Scaffold

While there have been promising early results in meniscal augmentation, this is still a technique which has a paucity of long-term evidence. A recent systematic review of meniscal scaffold use in athletes has suggested satisfactory outcome in approximately 70% of patients [41]. This makes it very difficult to justify in the young, active patient in whom we are trying to prevent early osteoarthritis. While the authors recognize that there is potential for this technique, it is our opinion that it is not presently justifiable to extend this treatment to the wider population.

3.6.2.2 Meniscal Allograft Transplant

Meniscal allograft is also relatively novel with a limited follow-up in the literature. There are papers stating the medium-term outcomes of meniscal transplant, which are very favourable indeed. Vundelinckx et al. followed up 39 patients following transplant assessing them with Lysholm, SF-36 and KOOS PROM data, all of which showed improvement in their scores at a mean 8.7 years [42]. Perhaps most importantly, all but one of these patients stated that they would be happy to have the operation again, which is a significant marker of how well the patient thinks they have done. However, meniscal transplant is a highly specialized procedure requiring significant surgical ability. As with all complex surgery, this is best done in centres where there are all of the ancillary support networks in place and by surgeons who have the right surgical skills set. Certainly from the perspective of a government-funded healthcare service (such as the National Health Service in the United Kingdom), there needs to be caution in extolling the virtues of such a procedure. This risks the non-specialist surgeon taking on these cases and performing them to an unsatisfactory standard thus affecting clinical outcomes.

3.6.3 Meniscal Surgery Is Specialist Surgery of the Knee

When considering the evidence presented for preserving the meniscus and subsequent management of the meniscectomized knee, the authors believe that this highlights a key area for consideration. If meniscal surgery is to be done properly, then it should be done by surgeons who are capable of meniscal repair and, perhaps more importantly, prepared to do it. The ESSKA have convened the "Meniscus Consensus Project" in order to harmonize the management of meniscal pathology. This project focuses on the evidence in the literature in order to bring together many varying opinions on management and bring them to a single set of guidelines. One of the problems with surgery such as knee arthroscopy is that there are so many different surgeons performing it. Some of these surgeons will be very well read, understand the current literature and be engaging in current trends and theorem, some will not. The problem comes when the surgeon who does not understand the evidence makes decisions about management of the meniscus lesion. One such example of this is surgery for degenerative meniscal tears. The landmark level I paper from the FIDELITY group in Finland highlighted that there was no significant difference between the meniscal debridement and sham surgery groups at 12 months [43]. Prior to this Moseley et al. published their work in the *New England Journal of Medicine*, demonstrating no benefit to arthroscopic lavage for osteoarthritis of the knee over placebo up to 24 months [44]. These papers have been widely misrepresented across the orthopaedic community, and by publishing a set of guidelines for surgeons, ESSKA hopes to consolidate the evidence from papers such as these into a more clear and concise format.

Perhaps in order that our patients receive the most appropriate treatment for their meniscal pathology, we should embrace the concept that surgery to the meniscus is a specialized area of knee surgery requiring a level of surgical competence and good understanding of the literature. Only then will we really begin to understand what successful meniscal surgery looks like.

3.7 Take-Home Message

These facts lead us to the inescapable conclusion that the meniscus should be preserved wherever possible and that in the patient whom meniscal preservation cannot be achieved, an alternative augmentation may be required to prevent hastening of articular cartilage wear.

While we should endeavour to preserve the meniscus, it is inevitable that there will be patients whose meniscus is beyond preservation. Here, there is need for an alternative strategy to try to prevent long-term sequelae of meniscectomy. Meniscal augmentation is one such strategy.

While the techniques for repair of the meniscus have progressed substantially over the years, there are still significant challenges facing the surgeon intending to repair a torn meniscus.

It is inevitable that there will be failures. This is where meniscal replacement has been suggested as the next step in management and an effort to avoid the otherwise inevitable degenerative changes seen following meniscectomy.

References

1. Manson TT, Cosgarea AJ. Meniscal injuries in active patients. Adv Stud Med. 2004;4:545–52.
2. Brelin AM, Rue JP. Return to play allowing meniscus surgery. Clin Sports Med. 2016;35(4):669–78.
3. Konan S, Haddad FS. Outcomes of meniscal preservation using all-inside meniscus repair devices. Clin Orthop Relat Res. 2010;468(5):1209–13.
4. Choi NH, Kim BY, Hwang Bo BH, Victoroff BN. Suture versus FasT-fix all-inside meniscus repair

at time of anterior cruciate ligament reconstruction. Arthroscopy. 2014;30(10):1280–6.

5. Matsushita T, Nagai K, Araki D, Tanaka T, Matsumoto T, Nishida K, Kurosaka M, Kuroda R. Factors associated with the status of meniscal tears following meniscal repair concomitant with anterior cruciate ligament reconstruction. Connect Tissue Res. 2017;58(3–4):386–92. [Epub ahead of print]
6. Gill SS, Diduch DR. Outcomes after meniscal repair using the meniscus arrow in knees undergoing concurrent anterior cruciate ligament reconstruction. Arthroscopy. 2002;18:569–77.
7. Lee GP, Diduch DR. Deteriorating outcomes after meniscal repair using the meniscus arrow in knees undergoing concurrent anterior cruciate ligament reconstruction: increased failure rate with long-term follow-up. Am J Sports Med. 2005;33:1138–41.
8. Logan M, Watts M, Owen J, Myers P. Meniscal repair in the elite athlete: results of 45 repairs with a minimum 5-year follow-up. Am J Sports Med. 2009;37(6):1131–4.
9. Lind M, Nielsen T, Faunø P, et al. Free rehabilitation is safe after isolated meniscus repair: a prospective randomized trial comparing free with restricted rehabilitation regimens. Am J Sports Med. 2013;41: 2753–8.
10. Andersson-Molina H, Karlsson H, Rockburn P. Arthroscopic partial and total meniscectomy: a long-term follow-up study with matched controls. Arthroscopy. 2002;18(2):183–9.
11. VanderHave KL, Perkins C, Le M. Weightbearing versus non-weightbearing after meniscus repair. Sports Health. 2015;7(5):399–402.
12. King D. The function of the semi-lunar cartilages. J Bone Joint Surg Am. 1936;18(4):1069–76.
13. Fairbank TJ. Knee joint change after meniscectomy. J Bone Joint Surg Br. 1948;30B(4):664–70.
14. Fukubayashi T, Kurosawa H. The contact area & pressure distribution pattern of the knee. A study of normal & osteoarthritic knee joints. Acta Orthop Scand. 1980;51(6):871–9.
15. Levy IM, Torzilli PA, Warren RF. The effect of medial meniscectomy on anterior-posterior motion of the knee. J Bone Joint Surg Am. 1982;64A(6):883–8.
16. Sommerlath KG. Results of meniscal repair and partial meniscectomy in stable knees. Int Orthop. 1991;15(4):347–50.
17. Stein T, Mehling AP, Welsch F, von Eisenhart-Rothe R, Jager A. Long term outcome after arthroscopic meniscal repair versus arthroscopic partial meniscectomy for traumatic meniscal tears. Am J Sports Med. 2010;38(8):1542–8.
18. Pengas IP, Assiotis A, Nash W, Hatcher J, Banks J, McNicholas MJ. Total meniscectomy in adolescents. A 40 year follow up. J Bone Joint Surg Br. 2012;94B:1649–53.
19. Khan FA, Koff MF. Effect of local alignment on compartmental patterns of knee osteoarthritis. J Bone Joint Surg Am. 2008;90(9):1961–9.
20. Baratz ME, Fu FH, Mengato R. Meniscal tears: the effect of meniscectomy and of repair on intra-articular contact areas and stress in the human knee. Am J Sports Med. 1986;14(4):270–5.
21. Hede A, Larsen E, Sandberg H. The long term outcome of open total and partial meniscectomy related to the quantity and site of the meniscus removed. Int Orthop. 1992;16(2):122–5.
22. Allaire R, Muriuki M, Gilbertson L, Hamer CD. Biomechanical consequences of a tear of the posterior root of the medial meniscus. Similar to total meniscectomy. J Bone Joint Surg Am. 2008;90(9): 1922–31.
23. Stone KR, Rodkey WG, Webber R, McKinney L, Steadman JR. Meniscal regeneration with polymeric collagen scaffolds. In vitro and in vivo studies evaluated clinically, historically and biochemically. Am J Sports Med. 1992;20(2):104–11.
24. Stone KR, Adelson WS, Pelsis JR, Walgenbach AW, Turek TJ. Long-term survival of concurrent meniscus allograft transplantation and repair of the articular cartilage: a prospective 2 to 12 year follow up report. J Bone Joint Surg Br. 2010;92(7):941–8.
25. Stone KR, Rodkey WG, Webber R, McKinney L, Steadman JR. Future directions. Collagen-based prostheses for meniscal regeneration. Clin Orthop Relat Res. 1990;252:129–35.
26. Tienen TG, Heijkants RG, de Groot JH, Pennings AJ, Schouten AJ, Veth RP, Buma P. Replacement of the knee meniscus by a porous polymer implant: a study in dogs. Am J Sports Med. 2006;34(1):64–71.
27. Verdonk R, Verdonk K, Huysse W, Heinrichs EL. Tissue ingrowth after implantation of a novel, biodegradable polyurethane scaffold for treatment of partial meniscal lesions. Am J Sports Med. 2011;39(4): 774–82.
28. Rodkey WG, DeHaven KE, Montgomery WH 3rd, Baker CL Jr, Beck CL Jr, Hormel SE, Steadman JR, Briggs KK. Comparison of the collagen meniscus implant with partial meniscectomy. A prospective randomised trial. J Bone Joint Surg Am. 2008;90(7):1413–26.
29. Spencer SJ, Saitha A, Carmont MR, Dhillon MS, Thompson P, Spalding T. Meniscal scaffolds: early experience and review of the literature. Knee. 2012;19(6):760–5.
30. Annandale T. An operation for displaced semilunar cartilage. BMJ. 1885;1(1268):779.
31. Canham W, Stanish W. A study of the biological behaviour of the meniscus as a transplant in the medial compartment of a dog's knee. Am J Sports Med. 1986;14(5):376–9.
32. Milachowski KA, Weismeier K, Wirth CJ. Homologous meniscus transplantation. Int Orthop. 1989;13(1):1–11.
33. Cameron JC, Saha S. Meniscal allograft transplantation for unicompartmental arthritis of the knee. Clin Orthop Relat Res. 1997;337:164–71.
34. de Boer HH, Koudstaal J. The fate of the cartilage after transplantation of cryopreserved nontissue-antigen-matched allograft. A case report. Clin Orthop Relat Res. 1991;266:154–1.

35. van Arkel ERA, de Boer HH. Survival analysis of human meniscal transplantations. J Bone Joint Surg Br. 2002;84(2):227–31.
36. Pollard ME, Kang Q, Berg EE. Radiographic sizing for meniscal transplantation. Arthroscopy. 1995;11:684–7.
37. McConkey M, Lyon C, Bennett DL, Schoch B, Britton C, Amendola A, Wolf B. Radiographic sizing for meniscal transplantation using 3-D CT reconstruction. J Knee Surg. 2012;25(3):221–5.
38. Yoon JR, Jeong HI, Seo MJ, Jang KM, Oh SR, Song S, Yang JH. The use of contralateral knee magnetic resonance imaging to predict meniscal size during meniscal allograft transplantation. Arthroscopy. 2014;30(10):1287–93.
39. Gallacher PD, Gilbert RE, Kanes G, Roberts SNJ, Rees D. White on white meniscal tears to fix or not to fix? Knee. 2010;17:270–3.
40. Noyes FR, Barber-Westin SD. Arthroscopic repair of meniscal tears extending into the avascular zone in patients younger than twenty years of age. Am J Sports Med. 2002;30(4):589–600.
41. Harston A, Nyland J, Brand E, McGinnis M, Caborn DN. Collagen meniscus implantation: a systematic review including rehabilitation and return to sports activity. Knee Surg Sports Traumatol Arthrosc. 2011;20(1):135–46.
42. Vundelinckx B, Bellemans J, Vanlauwe J. Arthroscopically assisted meniscal allograft transplantation in the knee: medium term subjective, clinical and radiological outcome evaluation. Am J Sports Med. 2010;38:2240–7.
43. Sihvonen R, Paavola M, Malmivaara A, Itälä A, Joukainen A, Nurmi H, Kalske J, Järvinen TLN. Finnish degenerative meniscal lesion study (FIDELITY) group. Arthroscopic partial meniscectomy versus sham surgery for a degenerative meniscal tear. BMJ Open. 2013;3:e002510.
44. Moseley JB, O’Malley K, Petersen NJ, Menke TJ, Brody BA, Kuykendall DH, Hollingsworth JC, Ashton CM, Wray NP. A controlled trial of arthroscopic surgery for osteoarthritis of the knee. N Engl J Med. 2002;347(2):81–8.

Basic Concepts in Hip Arthroscopy

4

O. Marin-Peña, B. Lund, O.R. Ayeni, P. Dantas,
D. Griffin, V. Khanduja, H.G. Said, M. Tey,
E. Dickenson, J. Kay, V. Mascarenhas,
M.A. Sadakah, K.H. Sunil Kumar, and M. Tahoun

4.1 Introduction

Hip arthroscopy has become very popular in the last decade. Numbers are increasing rapidly worldwide. More than 11,000 hip arthroscopies were performed at the English public health system from 2002 to 2013. It means an increase of more than 700%. Similar numbers were published in North America, with an increase of more than 350% in the period 2004–2009. During a similar period on the opposite side of the globe, Korean researches published a twofold increase between 2007 and 2010. In the midterm, there is a projected increase of 1388% by 2023 [1].

O. Marin-Peña (✉)
Orthopedic Surgery and Traumatology Department,
University Hospital Infanta Leonor, Madrid, Spain
e-mail: olivermarin@yahoo.es

B. Lund
Orthopedic Department, Hospitalsenheden Horsens,
Horsens, Denmark

O.R. Ayeni • J. Kay
Orthopedic Surgery and Traumatology Department,
McMaster University, Hamilton, ON, Canada

P. Dantas
Department of Orthopaedic Surgery, Hospital CUF
Descobertas, Lisbon, Portugal

D. Griffin • E. Dickenson
Department of Orthopaedic Surgery, Warwick
Medical School, University of Warwick,
Coventry, UK

V. Khanduja

Addenbrooke's – Cambridge University Hospital,
Cambridge, UK

University of Cambridge, Cambridge, UK

The Bone & Joint Journal, Cambridge, UK

H.G. Said
Orthopaedic and Trauma Surgery Department, Assiut
University Hospital, Asyut, Egypt

M. Tey • M. Tahoun
Department of Orthopaedic Surgery, Hospital del Mar,
UAB, Barcelona, Spain

V. Mascarenhas
MSK imaging Unit, Imaging Center, Hospital da Luz,
Lisbon, Portugal

M.A. Sadakah
Orthopaedic and Trauma Surgery Department, Tanta
University Hospital, Tanta, Egypt

K.H. Sunil Kumar
Addenbrooke's – Cambridge University Hospital,
Cambridge, UK

G.M.M.J. Kerkhoffs et al. (eds.), *ESSKA Instructional Course Lecture Book*,
https://doi.org/10.1007/978-3-662-56127-0_4

4.2 State-of-the-Art Treatment

Due to this increase of hip arthroscopy indications, it is very interesting to have a look at basic concepts that could be very useful for the beginners with this technique.

4.2.1 Good Indications for Hip Arthroscopy

The use of hip arthroscopy has recently developed and expanded more than in other joints such as the knee and shoulder. Several contributing factors are responsible for this delayed development. Anatomic constraints such as the femoral head sitting deeply recessed in the acetabulum, as well as the thick fibrocapsular and muscular envelope surrounding the hip, preclude large amounts of distension of the joint. Furthermore, the location of various neurovascular structures including the sciatic and lateral femoral cutaneous nerves makes portal placement in hip arthroscopy more complicated than in other joints. Moreover, the conditions for which hip arthroscopy is indicated such as femoroacetabular impingement (FAI), labral tears and extra-articular impingement have historically been poorly recognized and untreated [2]. Improvements in our understanding of these conditions about the hip, as well as enhanced imaging technology and arthroscopic tool development, have allowed for the recent expansion of indications for hip arthroscopic intervention. This chapter describes the common as well as emerging indications for hip arthroscopy.

4.2.1.1 Loose Body Removal

Etiologies of loose bodies within the hip joint include post-traumatic fragments of the femoral head or acetabulum, synovial chondromatosis, degenerative joint disease, avascular necrosis, osteochondritis dissecans, os acetabuli, calcium deposits inside a labral tear and foreign bodies such as bullets or pieces of surgical instruments. The diagnosis of a loose body within the hip joint involves a combination of patient history, clinical examination, diagnostic imaging and diagnostic intra-articular injections. Indications for arthroscopic removal include failure of nonoperative management after the diagnosis of a loose body within the joint. Access to the loose body may present a challenge for removal; however strategic positioning of portals such as direct anterior and posterolateral portals to access the acetabular fossa can improve access [3].

4.2.1.2 Septic Arthritis

The indications for arthroscopic management of septic arthritis of the hip include clinical, laboratory and imaging parameters. Clinical indications are pain to the anterior groin for less than 1 week, limited passive and active range of motion of the hip, inability to bear weight on the joint and/or pyrexia. Laboratory indications include leukocytosis, an elevated C-reactive protein level, an elevated erythrocyte sedimentation rate, purulent material on hip aspiration and/or a positive aspirate culture. When indicated, the arthroscopic procedure typically includes a thorough irrigation as well as debridement of any damaged or infected tissue [4].

4.2.1.3 Labral Tears

Labral tears can be traumatic or insidious in onset, and patients typically present with anterior groin pain that may radiate to the trochanteric or gluteal regions [5]. Physical examination manoeuvres that may indicate intra-articular pathology include passive log rolling of the affected leg, as well as the anterior impingement test. In terms of imaging, MRI is typically the preferred modality for labral tears as they allow for visualization of the labrum and surrounding soft tissues. A gadolinium-based contrast is added in magnetic resonance arthrography (MRA) to allow for separation of the labrum from the capsule, thereby enhancing visualization. A rigorous trial of non-surgical modalities including rest, medications, physical therapy and therapeutic injections should be completed prior to pursuing surgical intervention. The goal of surgery is to preserve the functional labral tissue with selective debridement and refixation of tissue (Fig. 4.1) [5]. A recent systematic review identified ten studies with a focus on the efficacy of hip arthroscopy for acetabular labral tears [6].

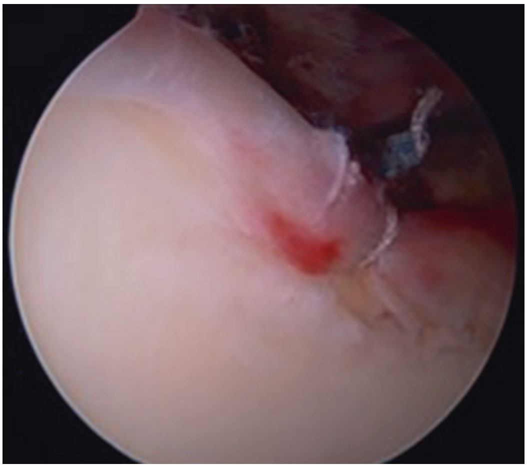


Fig. 4.1 Arthroscopic repair of a labral tear

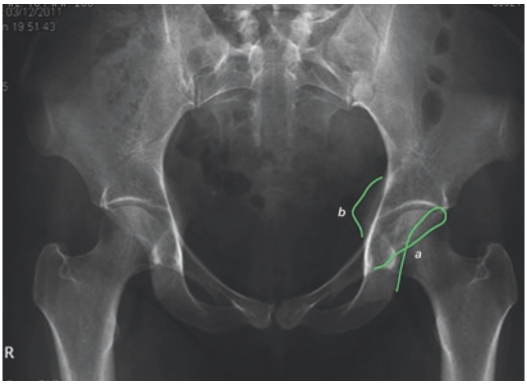


Fig. 4.2 FAI (pincer lesion) on an anteroposterior radiograph. (**a**) Crossover sign. (**b**) Medialization ischial spine

Fig. 4.3 FAI (cam lesion) on a frog-leg lateral radiograph

Good clinical outcomes have been described for patients with labral fixation and low-grade chondrolabral lesions [6]. Specific clinical hip scores should be used to evaluate the results after arthroscopic repair of hip labral injuries. Modified Harris Hip Score (mHHS), Hip Outcome Score (HOS), International Hip Outcome Tool 33 (iHOT 33) or Copenhagen Hip and Groin Outcome Score (HAGOS) are good clinical scores to measure clinical improvement after hip arthroscopy in young patients. Arthroscopic labral reconstruction may be considered in young and active patients who have undergone prior labral resection, with an irreparable or degenerative labrum, and a minimum of 2 mm joint space remaining [7].

4.2.1.4 FAI

The use of hip arthroscopy as a viable treatment option for FAI has expanded considerably in recent years. FAI is caused by a mismatch between the femoral head and acetabulum. Two subtypes of FAI, cam and pincer, involve abnormal morphologies of the femoral head and acetabular rim, respectively, with most patients presenting with a combination of these deformities (Figs. 4.2 and 4.3) [8].

The indications for arthroscopic management of FAI typically include a combination of pain, clinical examination findings, positive radiographic findings and diagnostic intra-articular injections. A positive impingement test was the commonest clinical examination manoeuvre used as an indication for surgery. Other special test manoeuvres such as FADIR (flexion, adduction and internal rotation), FABER (flexion, abduction and external rotation) (Fig. 4.4), C-sign and log roll have been reported to be involved in the decision-making process as well. Imaging modalities are important indications for arthroscopic management of FAI as well. In fact, it was found that 20% of studies that reported indications for hip arthroscopy used radiographic indications alone. The imaging modalities used include anteroposterior (AP) radiographs alone, as well as a combination of CT, MRI and MRA. Radiographic indications include, from most commonly used to least commonly used, cam or pincer lesions seen on AP radiographs, loss of sphericity of the femoral head (pistol-grip

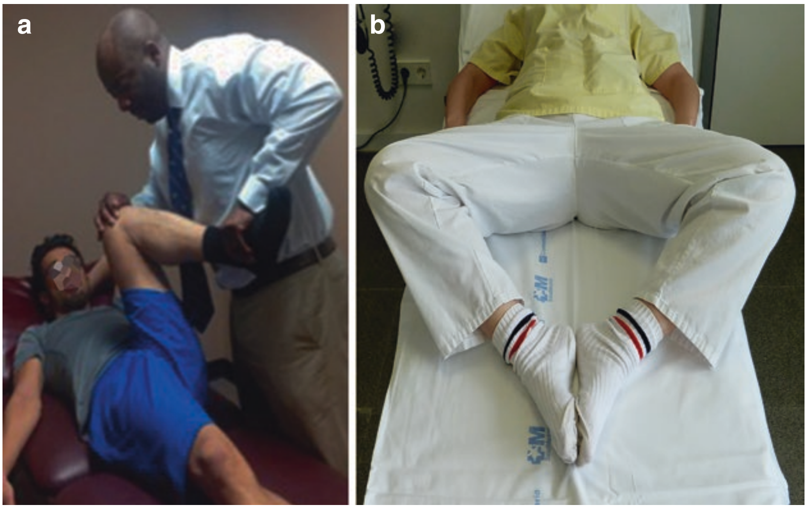


Fig. 4.4 (**a**) FADIR (flexion, adduction and internal rotation) and (**b**) FABER manoeuvres

deformity), acetabular retroversion, reduction of head-neck offset, alpha angle >50° and coxa profunda [9]. The duration of symptoms before consideration of surgical correction is typically reported as 6 months' time [9]. Arthroscopic surgical correction involves resection of the femoral head and neck, trimming of the osseous prominence on the acetabulum and repairing intra-articular damage such as cartilage and labral damage.

4.2.1.5 Extra-articular Disorders

The indications for the use of similar techniques for extra-articular pathology have continued to expand as well. Conditions include deep gluteal syndrome, internal snapping hip, external snapping hip and greater trochanteric pain syndrome [10]. In deep gluteal syndrome, entrapment of the sciatic nerve in the deep gluteal space causes pain in the buttock. Management with arthroscopic techniques involves exploration and decompression of the sciatic nerve from offending agents such as the piriformis, hamstring, obturator externus muscles or post-traumatic scar tissue. In two large series of 35 and 60 patients with deep gluteal syndrome, indications for hip arthroscopy were largely based on clinical symptoms and investigations such as imaging to rule out spinal pathology, diagnostic injections or the identification of an offending agent causing impingement on MRA [11, 12].

Sliding of the iliopsoas over the iliopectineal ridge or femoral head causes internal snapping hip. In symptomatic patients, a painful snapping sensation occurs with exercise. Indications for surgical management by means of arthroscopy involve failure of nonoperative management. First-line treatment consists of a rigorous trial of non-surgical modalities including rest, nonsteroidal anti-inflammatory drugs (NSAIDs), painkillers, physical therapy and steroid injections [13]. If patients continue to experience symptoms after nonoperative treatment, hip arthroscopy including release of the iliopsoas tendon at the pelvic brim or at its insertion to the lesser trochanter is appropriate [14]. Greater trochanteric pain syndrome includes a group of disorders at the lateral aspect of the hip including greater trochanteric bursitis, external snapping hip and gluteus medius/minimus tears. External snapping hip is caused by a thickened posterior iliotibial (IT) band, tensor fasciae latae or gluteus maximus sliding over the greater trochanter during flexion of the hip. First-line treatment for each of these conditions includes a trial of non-surgical modalities including rest, avoidance of inciting activities and anti-inflammatory medication and injections that were diagnostic and therapeutic in nature [15]. Failure of these nonoperative treatment options is the primary indication for surgical consideration [10]. Arthroscopic techniques are now more commonly used than open procedures, which include bursectomy, gluteus maximus tenotomy, IT band release, as well as abductor tendon debridement and repair [16].

4.2.1.6 Traumatic and Atraumatic Instability of the Hip

After traumatic dislocation of the hip, close reduction is the gold standard of treatment. If complete reduction is not achieved, arthroscopic surgery may be warranted to remove loose bodies or to manage accompanying labral tears and/or chondral injuries. Fractures of both the femoral head and acetabulum can be treated using arthroscopic-assisted techniques, assuming the patient is stable. Otherwise, patients with persistent pain and mechanical symptoms due to labral and/or chondral damage are suitable candidates for delayed arthroscopic management once stable [17]. Recently, hip arthroscopy has been considered for the treatment of recurrent, atraumatic instability as well. After a trial of nonoperative management, hip arthroscopy may be indicated when intra-articular injections provide significant relief in symptoms. However, patients undergoing arthroscopic management often undergo anatomic labral repair in addition to a capsule management procedure. Successful reduction in capsular volume, using either capsular plication or thermal capsulorrhaphy, is increasingly being reported. Connective tissue disorders such as Ehlers–Danlos syndrome that impact hip function can be treated with a capsular plication while addressing other concurrent lesions such as labral tears [18].

4.2.1.7 Arthroscopy in the Setting of Hip Arthroplasty

Another relatively novel indication for arthroscopic surgery of the hip involves its use after arthroplasty of the joint. The most common indication for arthroscopy in the setting of arthroplasty is in the management of patients with iliopsoas tendinopathy, commonly after a diagnostic injection of the iliopsoas tendon sheath. The second most common indication of this procedure in patients with a prior hip arthroplasty is to help with the diagnosis of persistent hip or groin pain [19].

4.2.1.8 Relative Contraindications

Although hip arthroscopy continues to expand with respect to its indications, contraindications to its use include patients with severe OA of the hip. Patients with no OA have substantially better pain and functional outcomes after hip arthroscopy than those with OA [20]. While medical history and physical examination may provide some indication as to when the extent of the OA has exceeded the allowable threshold, no definitive criteria have been established using these parameters. However, a recent systematic review has found that patients with radiographic parameters such as Tönnis grade 1 or higher or a joint space of 2 mm or less receive less benefit from hip arthroscopy and are more likely to ultimately covert to a total hip arthroplasty [21]. Other relative contraindications due to difficulties accessing the hip joint arthroscopically include obesity and significant bony deformities such as acetabular protrusio and severe heterotrophic ossification.

4.2.2 How to Best Learn Hip Arthroscopy: Dealing with the Learning Curve

Hip arthroscopy has been steadily gaining popularity since the 1970s and has seen a growth of magnanimous proportion in the last decade [22]. The concept of femoroacetabular impingement (FAI) was introduced in the late 1990s, and since then the number of publications relating to this subject has increased immensely [23]. FAI can be broadly classified into two types, cam and pincer; however, a majority of patients have a combination of both with one being a major abnormality [23]. Nonoperative measures to treat FAI include activity modification, analgesia and physiotherapy [23]. Ganz et al. proposed that early operative treatment of FAI led to slow the progression of arthritis in the hip joint [8]. Operative treatment primarily aims to treat the anatomical abnormality improving the clearance of motion of the hip joint and preventing abutment between the acetabular rim and femoral neck and secondarily to address the soft tissue damage caused by the impingement lesion [8]. The safe surgical dislocation with the trochanteric flip osteotomy was popularized by Ganz as a measure to address FAI, and of late, hip arthroscopy has become increasingly popular to achieve the same surgical goals [24]. On the other hand, in case of hip dysplasia, the surgical options available are either hip arthroscopy or periacetabular osteotomy for acetabular undercoverage. In addition to FAI and hip dysplasia, indications for hip arthroscopy include acetabular labral tears, ligamentum teres injuries, chondral defects in the hip joint, septic arthritis of the hip, loose bodies and synovial abnormalities of the hip and extra-articular indications such as snapping hip, trochanteric bursitis and gluteus medius tears, and the list keeps expanding [25]. This coupled with the fact that the risk of complications is around 3% makes the procedure attractive. However, it has a steep learning curve and is certainly not one for the inexperienced [26].

The senior author believes that one can achieve competence and become confident at performing hip arthroscopy only by having a structured approach to training. In addition to mastering the technical skills required for hip arthroscopy, understanding pathology and the decision-making process in the outpatient clinic are of utmost importance. This starts right from the assessment of a young adult with hip pain in clinic, arranging appropriate investigations and interpreting them correctly, and then finally making the decision of undertaking a hip arthroscopy if indicated. The role of a multidisciplinary team consisting of the hip surgeon, sports physician, radiologist and a physiotherapist cannot be underestimated in this regard.

Thus, not only performing the hip arthroscopic procedure safely and effectively is essential but also choosing the right patient for the procedure is paramount for a successful outcome. To progress in the technical art of arthroscopy of the hip and to become a safe and effective arthroscopist, one may consider the following avenues to master skills (Fig. 4.5):

- Simulation training
- Cadaveric skills training
- Fellowship in young adult hip surgery
- Mentored independent practice

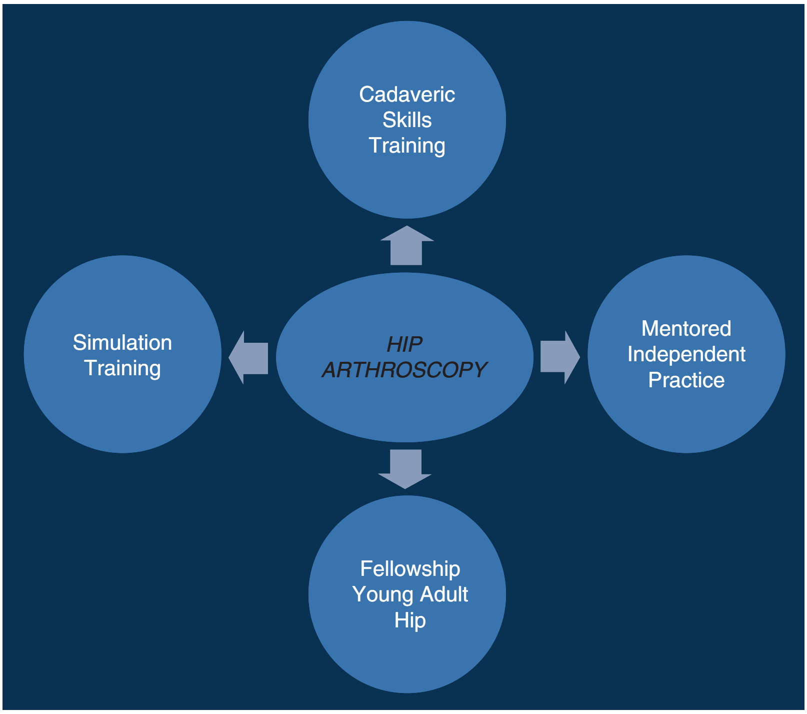


Fig. 4.5 Different aspects of a structured training in hip arthroscopy

The above is not an exhaustive list, and the surgeon may still find that his/her learning curve continues to improve throughout one's independent practice. Attending an educational meeting regularly during independent practice would give the surgeon the benefit of meeting up with their peers and discussing ideas, tips and tricks, advances in techniques and also any possible complications that one may have experienced.

4.2.2.1 Simulation Training

Virtual reality training has been shown to improve technical skills in orthopaedic surgery [27–29]. Therefore, the use of a virtual reality simulator in hip arthroscopy training has the potential to provide an additional benefit to a trainee surgeon [30]. In addition to the use of a simulator, adopting proficiency-based progression (PBP) in training has been shown to produce a superior arthroscopic skill set amongst the trainees [31]. PBP involves training to be undertaken in various key stages and several steps in each stage. A trainee then has to successfully progress through each stage. Training on an arthroscopic simulator helps with the trainee to go through a checklist making sure that one does not skip an essential step and also helps with improving hand-eye coordination [32]. In addition, simulation training can also be a useful tool to assess a trainee's judgment in a simulated setting, thereby assessing their cognitive and professional skills. This would in turn be helpful to provide a constructive feedback of both technical and non-technical skills [32]. Howells et al., in their study, showed improvement of knee arthroscopic psychomotor skills of orthopaedic trainees in theatre following a structured virtual reality knee simulator training programme [29].

4.2.2.2 Cadaveric Skills Training

With the introduction of European Working Time Directive (EWTD), there has been a significant reduction in total training time in surgical specialties in the UK from 30,000 h to around 6000 h [29]. Along with this reduction in training time, the surgical outcomes have been under public scrutiny, so one has to ensure that he/she is adequately trained in a specific procedure to provide safe and effective patient care [29]. Practicing the procedure in a cadaveric laboratory helps the trainee to experience the "feel" of the procedure in a controlled environment prior to undertaking the same procedure in a real patient [9]. Arthroscopic training in a cadaveric skills laboratory has been shown to be superior to virtual reality simulator training alone, in the case of knee arthroscopy, leading to a significantly faster progression to skill acquisition with cadaveric training [29]. The senior author chairs the Cambridge-ESSKA Hip Course, which is held in Cambridge (UK) annually and combines all the three aspects required for training in hip arthroscopy, i.e. didactic training via lectures and discussions, virtual reality hip arthroscopy simulation and alongside this state-of-the-art wet lab cadaveric training [33].

4.2.2.3 Fellowship in Young Adult Hip Surgery

Currently, the fivefold reduction in training time in Europe means that one has to undertake a specialist fellowship to consolidate clinical and surgical skills to bridge the gaps in training. In addition, hip arthroscopy is a very specialized procedure with only a select few centres and surgeons in Europe performing it in large numbers. Undertaking a specialist fellowship focused on treating hip problems in the young adult is essential for gaining adequate clinical exposure and improving surgical expertise. Travelling fellowships are invaluable, as well as in providing one with the experience from different centres of excellence, in particular a different approach to a familiar problem or tips and tricks to deal with a complex problem.

4.2.2.4 Mentored Practice During the Learning Curve

Setting up an independent surgical practice could be stressful in the beginning, especially if undertaking a highly specialized procedure like hip arthroscopy. Deitrich et al. have shown that with growing experience in performing hip arthroscopy, there was a decrease in the rate of complications [34]. At present, there is poor evidence to quantify the number of cases needed to overcome the learning curve; however some authors have reported that 30 cases are needed during this period [26, 35]. Furthermore, a newly appointed hip arthroscopy specialist may want to consider undertaking the procedure under the supervision of a senior surgeon, which has also been shown to significantly decrease the rate of complications [34]. Perez-Carro and Tey broadly classified the common pitfalls during the learning curve into three broad categories: (1) preoperative, (2) intraoperative and (3) postoperative [36]. Preoperative errors included improper or wrong patient selection and those related to improper patient positioning during hip arthroscopy [36]. Intraoperative errors included improper portal creation leading to nerve injury and/or difficult access to the hip joint and damage to intra-articular structures during introduction of instruments and those related to the total traction time [36]. These pitfalls can certainly be overcome by undertaking a structured training programme during a specialized hip arthroscopy fellowship in addition to virtual reality simulation training and cadaveric skills training which can also help with reduction in the learning curve.

4.2.2.5 Complications and Learning Curve

The rate of complications following hip arthroscopy is between 1 and 4%, for example, damage to femoral head, lateral femoral cutaneous nerve injury and neuropraxia of sciatic, femoral or pudendal nerve secondary to traction to name a few [37]. Harris et al. conducted a systematic review of 92 studies comprising of more than 6000 hip arthroscopies and found a major complication rate of 0.58% and minor complication rate of 7.5% [38]. It is therefore essential that during the initial part of the learning curve only non-complex, straightforward cases are selected to be performed via the arthroscopic route and open surgical dislocations performed for the complex cases. As the experience and the level of confidence increase, one can take on more challenging cases via the arthroscopic route. There is also evidence that with increasing experience, the rate of complications following hip arthroscopy decreases [38–40].

Adopting a structured approach to training is essential to gain an in-depth knowledge in the field of young adult hip surgery and hip arthroscopy in particular. Virtual reality simulation training and cadaveric skills training are useful adjuncts to a specialist hip arthroscopy fellowship. Finally, it would be an added benefit if one has a mentor during the initial phase of independent practice while going through the learning curve.

4.2.3 How to Get Safely into the Joint: Position and Portals

The main objective for beginners is to get a stable and comfortable setup for hip arthroscopy and obtain easy access through arthroscopic portals.

4.2.3.1 Patient Positioning

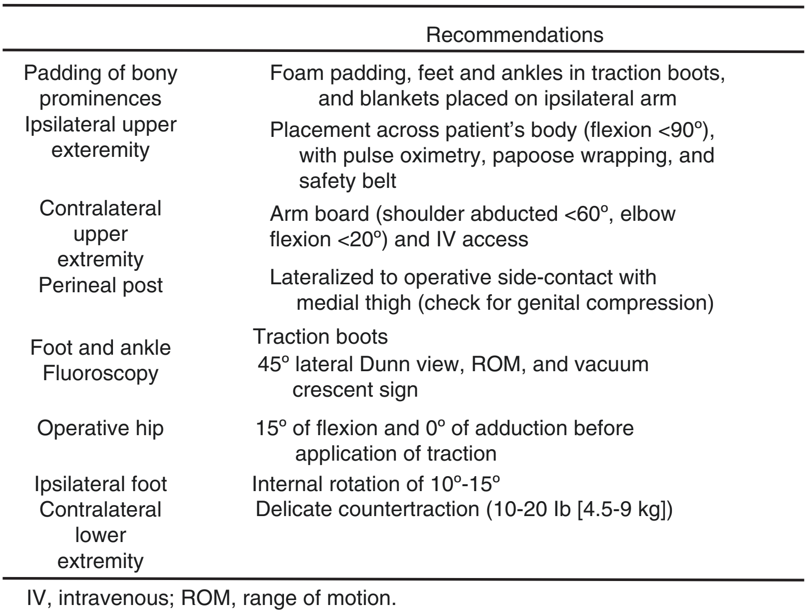
Hip arthroscopy can be effectively performed in a supine or lateral decubitus position. The position of the patient is largely dictated by the surgeon's preference and training. However, each position has its own benefits and drawbacks [41].

Supine Position

The patient is placed on a fracture table with the feet well-padded in traction boots and slightly lateralized towards the nonoperative leg, against a well-padded perineal post to limit the risk of pudendal nerve neuropraxia associated with traction. The nonoperative limb is abducted to approximately 45° with gentle traction applied to maintain the lateralized position [42, 43]. The supine position offers a familiar orientation of the joint to all orthopaedic surgeons. Also, a routine fracture table or a specialized traction table can be used [44]. Drawbacks to the supine position include difficult manoeuvrability in obese patients, and potentially decreased posterior access [45] (Fig. 4.6).

Lateral Position

A specialized traction table is usually needed. The patient must be stabilized in the decubitus position, usually with a beanbag, posts or a pegboard. Attention must be paid to padding downside bony prominences and placing an axillary roll to support the down arm. A perineal post is placed



	Recommendations
Padding of bony prominences	Foam padding, feet and ankles in traction boots, and blankets placed on ipsilateral arm
Ipsilateral upper exteremity	Placement across patient's body (flexion <90°), with pulse oximetry, papoose wrapping, and safety belt
Contralateral upper extremity	Arm board (shoulder abducted <60°, elbow flexion <20°) and IV access
Perineal post	Lateralized to operative side-contact with medial thigh (check for genital compression)
Foot and ankle	Traction boots
Fluoroscopy	45° lateral Dunn view, ROM, and vacuum crescent sign
Operative hip	15° of flexion and 0° of adduction before application of traction
Ipsilateral foot	Internal rotation of 10°-15°
Contralateral lower extremity	Delicate countertraction (10-20 lb [4.5-9 kg])

IV, intravenous; ROM, range of motion.

Fig. 4.6 Quick reference for hip arthroscopy procedure setup [43]

between the legs to allow for distraction and a lateral vector to the operative leg [44]. The lateral position is considered to have superior manoeuvrability in obese patients [46]. Also, it provides superior access to the posterior and inferior joint spaces compared with the supine position [47]. Disadvantages of lateral position include the extra time needed to position the patient and to adjust the perineal and traction posts in addition to the need to use special traction devices [41].

Most surgeons prefer general anaesthesia for hip arthroscopy because it provides adequate muscle relaxation for distraction, but regional anaesthesia can be a helpful adjunct in postoperative pain management [48]. Every effort should be made to maintain the patient's systolic blood pressure below 100 mmHg in order to optimize visualization [44].

4.2.3.2 Hip Joint Access

The hip joint space is separated into central and peripheral compartments by the acetabular labrum in addition to the extra-articular compartment around the hip joint (peritrochanteric space) [49]. So, the hip joint can be accessed either through:

1. "Central access first" is done under fluoroscopic guidance and under adequate joint distraction. This allows portal placement while minimizing the risk of injury to the labrum or cartilage [50]. However, there is a longer traction time with this technique, which leads to a higher risk of neurovascular and skin complication [51, 52].
2. "Peripheral access first", popularized by Dienst et al. [53], arthroscopic instruments are introduced along the anterior femoral neck region, which is devoid of articular cartilage and has a lower risk of injuring the labrum. This is followed by entry into the central compartment under vision without the need for fluoroscopy. No traction is needed during the arthroscopic access to the peripheral compartment. Masoud and Said [54] described anatomic surface landmarks for injection and access of the peripheral compartment that can markedly reduce or abolish the need for imaging guidance. This access is preferred by the author because it is easier and safer and involves less traction time (even acetabular recession could be started before traction) and because it is a very useful technique when central access fails.

3. “Outside-in technique”, in which anterior extra-capsular space of the hip is defined under fluoroscopy, then the anterior capsule is identified under arthroscopic visualization, and capsulotomy is performed to access the peripheral compartment. However, this technique is technically demanding and can be considered extensive relative to the other techniques, and complications such as fluid extravasation and dislocation can occur [55]. This technique is most useful when intra-articular access could not be achieved as in cases with intra-articular adhesions.

4.2.3.3 Portals

For the peripheral compartment, the proximal anterolateral portal (PAL) and distal anterolateral portal (DAL) are the primary working portals (Fig. 4.7). Their direction is marked on the skin under fluoroscopy before their establishment with their intersection projecting onto the femoral head-neck junction. PAL, as described by Dienst et al. [53], is oriented 45° caudally with the entry point lying at the junction between the medial and the middle third of a line drawn between GT and ASIS (soft spot). DAL is placed on a curved line running distally from the PAL with the centre of the curve being the greater trochanter (Figs. 4.8 and 4.9).

Fig. 4.7 Demonstrates bony landmarks (GT and ASIS) with the needles denoting surface drawings (vertical line from ASIS and perpendicular line to GT). Black dotted line demonstrates the direction of PAL portal. Note position of PAL, DAL and AL portals

Fig. 4.8 Curved red line (centred over GT) from PAL portal along which DAL portal is established

Fig. 4.9 Shows the direction of PAL and DAL portals

For central compartment, the anterior (A), anterolateral (AL) and posterolateral (PL) portals are the primary working portals [41]. The AL is established first. It is placed 1 cm proximal and anterior to the tip of the GT and directed parallel to the femoral neck [56]. The superior gluteal nerve is found an average of 4.4 cm above the level of the anterolateral portal [44]. The anterior (A) portal is placed at the intersection of a longitudinal line drawn distally from the ASIS and a transverse line across the superior edge of the GT. This portal is orientated 45° cephalic and 30° towards the midline [56]. This portal is often made slightly lateral to this intersection to avoid the branches of the lateral femoral cutaneous nerve that usually branch proximal to the anterior portal, but small lateral branches may be injured. The femoral nerve is found an average of 3.2 cm medial to the location of the anterior portal. The posterolateral portal is located 1 cm superior and posterior to the tip of the GT. The sciatic nerve is most at risk as it lays approximately 2–3 cm posterior to the portal. Other described accessory portals used to access the peripheral or lateral compartments should be placed under direct visualization if needed [44].

4.2.3.4 Author's Preferred Technique

The patient is placed in the supine position. A trial traction is done under fluoroscopy to assess sufficient distraction of the hip joint (8–10 mm distraction is sufficient). The traction is done in the abducted position and then completed by adduction of the limb to lever over the post. Once achieved, the traction is then released, and the hip can be prepped and draped in the standard fashion. The leg is flexed to 30–50° to relax the anterior capsule which facilitates access to the peripheral compartment and protects the cartilage. The post is removed during work in this position and reapplied before traction.

The author prefers to initially access the joint through the DAL portal as there is more space in the distal compartment of the joint, the soft tissues are less resistant, and there is less risk of scuffing the distal part of the femoral head. A spinal needle is inserted parallel to the femoral neck towards the head-neck junction under fluoroscopic guidance. Once the intra-articular location of the needle is established, a cannulated switching stick is passed over the guide wire. A standard arthroscopic cannula is passed over the switching stick followed by introduction of a standard 70° scope. A PAL portal is established under arthroscopic vision targeting an entry point 1 cm distal to the labrum. This is followed by thinning of the anterior capsule either by a shaver or radiofrequency. It should be mentioned that capsulotomy is not preferred at this step in order to maintain the ballooning effect of the capsule. Then, a "seven-step" tour of the peripheral compartment starts. Switching of viewing portal between PAL and DAL is important to see "around the curve" for any pathology. In addition, flexion to 70° and acetabular recession of the pincer lesion are usually started in this stage. This helps further reduce the traction time. For central compartment access, the post is reapplied, the leg is extended, and traction is done. The AL portal is established under arthroscopic vision. This further minimizes the risk of lateral head cartilage scuffing or labral injury compared with setting this portal under fluoroscopy guidance. Then inter-portal capsulotomy is established between the PAL and AL portals. The instruments from the DAL are redirected through the same skin incision for further work through the central compartment.

4.2.4 Hip Arthroscopy Anatomy and Variations at Central and Peripheral Compartment

The hip has two intra-articular compartments, the central and the peripheral, separated by the acetabular labrum [57]. The central compartment (CC) includes the acetabular fossa, teres ligament, lunate surface, labrum, fovea capitis and the articular surface of the femoral head in the weight-bearing area. The peripheral compartment contains the femoral neck, the non-weight-bearing cartilage of the femoral head, the medial and lateral synovial folds, perilabral recess or paralabral sulcus, the non-articular surface of the labrum and the articular capsule [58, 59]. There are several methods to localize intra-articular lesions in hip arthroscopy. The two most commonly used mapping systems are the "clock face" system—standardized to the right hip, where 3:00 clock position is anterior, and the 6:00 position corresponds to the middle point of the transverse acetabular ligament—and the "geographic zone method" which allows for a precise and more reproducible description of a lesion's position [58, 60] (Fig. 4.10). Both systems can also be used for mapping the femoral head, and the reference lines follow the same pattern used in the acetabulum, so the femoral locations will reflect the corresponding acetabular locations. The clock face system also allows a good correlation with the radial MRI findings [58, 60].

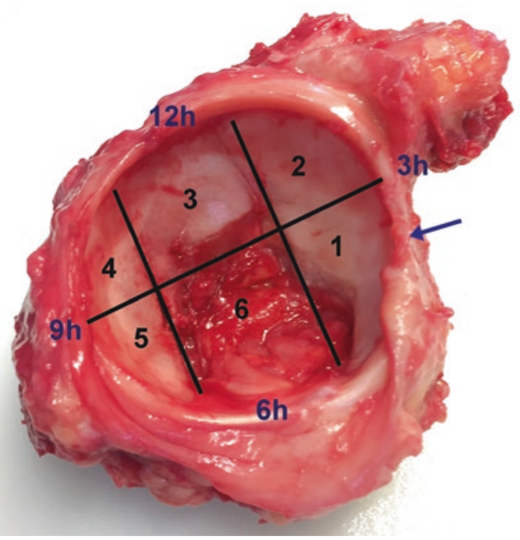


Fig. 4.10 The "clock face" system and the "geographic zone method" for the acetabular mapping. Arrow: the psoas U

4.2.4.1 Central Compartment

Acetabular Fossa

The acetabular fossa is located in the inferior region of the acetabulum and is surrounded by the horseshoe-shaped lunate surface. It is the non-articular surface of the acetabulum and is covered by synovium. The transverse ligament limits the inferior margin of the fossa and is in continuity with the anterior and the posterior labrum.

Ligamentum Teres

Arises from the transverse ligament and the ischial and pubic margins of the acetabular fossa and has a fascicular appearance with an anterior and posterior bundle. The ligamentum is trapezoidal at its base and runs to the femoral head where it becomes progressively round or oval shaped; it inserts in the fovea capitis. In the dynamic evaluation, the ligamentum is tight in hip external rotation, and head vascularity in adults is negligible [61, 62].

Fovea Capitis

The fovea capitis is a small area devoid of cartilage in the femoral head and is located slightly posterior and inferior to the centre of the femoral head cartilage. The articular cartilage thickness decreases from the centre to the periphery of the femoral head.

Labrum

The labrum is a fibrocartilage with a triangular cross section. Its base is inserted in the osseous acetabular rim, the articular or internal surface is in continuity with the acetabular cartilage, and the capsular or external surface is attached to the articular capsule. The labrum is in continuity with the transverse ligament antero-inferiorly and postero-inferiorly. The labral vascular supply arises from a periacetabular vascular ring with radial branches that course over the capsular surface of the labrum, terminating in its free edge [63]. This study did not show vessels entering the labrum from the adjacent subchondral bone. The clinical relevance of this fact is that the majority of the labral lesions are located in the chondrolabral junction with preservation of the vascularity, and the surgical labral detachment may interfere with its blood supply. The sublabral sulcus is an anatomic variant that should not be confused with a labral tear. In arthroscopy, it is a well-defined cleft between the labrum and the acetabular hyaline cartilage with smooth edges, with no signs of inflammation and no labral displacement or instability on probing. In the hips with a sublabral sulcus, the most frequent location is the postero-inferior (48%) and anterosuperior (44%) quadrant [64]. There is a normal concavity in the anterior acetabular rim in relation to the iliopsoas tendon (psoas U). It is universally present, and its superior aspect is a reliable arthroscopic landmark for the 3:00 clock position [58].

Physeal Scars

The ilio-pubic and the ilio-ischial physeal scars are remnants of the triradiate physis and can be found in the anterior and posterior lunate surfaces, respectively.

Stellate Crease and Supra-acetabular Fossa

The stellate crease is an anatomic variant and represents a focal fibrocartilaginous slitlike structure.

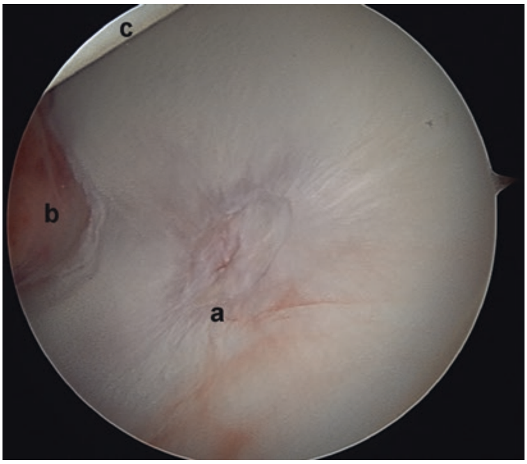


Fig. 4.11 Arthroscopic image of the left hip. The supra-acetabular fossa (**a**) separated from the acetabular fossa (**b**) and the femoral head (**c**)

Its location is variable but is usually found above the apex of the acetabular fossa at the 12:30 position. Another anatomic variant, more frequent in younger patients, is the supra-acetabular fossa located in the roof of the acetabulum at the 12:00 position on coronal and sagittal imaging. It can be detected in high-resolution hip MRI scans with normal marrow signal intensity and should not be confused with an osteochondral lesion (Fig. 4.11). The stellate crease is in continuity with the acetabular fossa, while supra-acetabular fossa is completely separated from it, but the former might be a residual scar left after obliteration of the supra-acetabular fossa [65, 66].

4.2.4.2 Peripheral Compartment

The peripheral compartment (PC) consists of the unloaded cartilage of the femoral head (FH); the femoral neck with the medial, anterior and posterolateral synovial folds (Weitbrecht's ligaments); the articular capsule with its intrinsic ligaments, including the zona orbicularis (ZO); the non-articular surface of the labrum and the perilabral recess or paralabral sulcus. PC of the hip can be divided routinely into the following areas: anterior neck area, medial neck area, medial head area, anterior head area, lateral head area, lateral neck area and posterior area (Fig. 4.12). Access to the CC can be undertaken by, initially, approaching the PC and, afterwards, accessing the CC under arthroscopic control [67]. Another technique to access the PC is an outside-in approach. The access to the PC is also important because loose bodies are commonly located here, and ultimately femoral osteoplasty is performed in the PC.

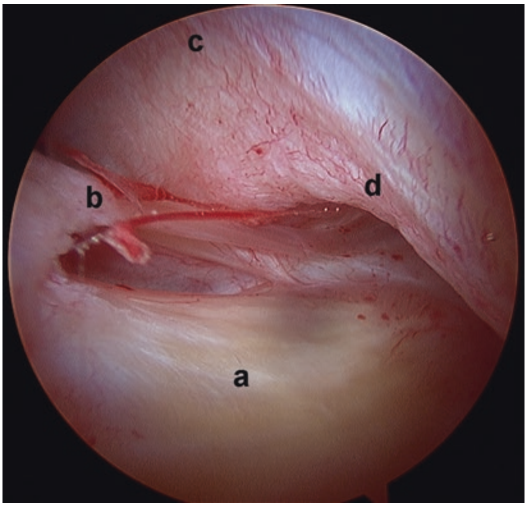


Fig. 4.12 Arthroscopic image of the peripheral compartment of a right hip. The anterior femoral neck (**a**), the medial synovial fold (**b**), the anterior capsule (**c**) and the zona orbicularis (**d**)

Labrum

The external surface of the labrum is observed from the PC enclosing the femoral head. At labral insertion lays the synovial membrane responsible for some of the labrum vascularization. In turn, the congruency of the labrum and the FH creates a perfect sealing mechanism. The acetabular labrum exhibits variability in shape, symmetry and dimensions. Labral shape can vary between triangular (66–69% of asymptomatic hips), round (11–16%) and flat (9–13%). It is thickest in the superior and posterior aspects and widest in the anterior and superior portions [68]. At the anterior and posterior margins, the hip joint capsule inserts directly at the base of the labrum. On the superior aspect of the PC, the anatomic space created between the joint capsule and labrum is the perilabral sulcus which is larger in this zone and also might be subject to dimensional variability [69].

Synovial Folds

Synovial folds are sheetlike structures of synovial and connective tissue that run longitudinally in various zones of the peripheral compartment and serve as important landmarks. The *medial synovial fold* ("iliopectineal fold") is located at the antero-medial aspect of the femoral neck but usually is not stuck to it. This structure is a very helpful landmark (guide to the 6:00 position), especially when visibility within the PC is limited by synovial disease. This fold passes proximally from the medial border of the femoral head, distal to the lesser trochanter [67]. The anterior synovial fold is adherent to the neck and only recognizable by its single fibres covering the bone of the neck. The lateral synovial fold is located at the junction between the lateral and the posterior femoral neck being a common landmark to the 12:00 position [58]. It runs from the greater trochanter upwards along the lateral side of the neck to the lateral margin of the head. It is often posteriorly stuck to the neck and forms a small pouch. It contains the lateral retinaculum and the intracapsular penetrating arteries from the lateral epiphyseal vessels arising from the terminal branches of the deep medial circumflex artery (the major responsible of femoral head blood supply). This fold usually marks the endpoint of trimming of the femoral head-neck junction in femoroacetabular impingement. Thus, preservation of these vessels is of primary importance, although cam deformities frequently extend beyond this point [70, 71].

Capsule

The anatomical structure that most influences the peripheral space is the joint capsule. It is a thick and tense fibrous sleeve extending from the outer neck to the acetabular rim. The inner surface is entirely covered with synovium. Some portions of the capsule have an increased thickness or are reinforced. Namely, (a) the superolateral part is reinforced by the reflected tendon of the rectus femoris, (b) the anterolateral part by the ilio-femoral ligament (y-shaped ligament of Bigelow), (c) the antero-medial part by the pubofemoral ligament and (d) the posterior capsule by the ischio-femoral ligament.

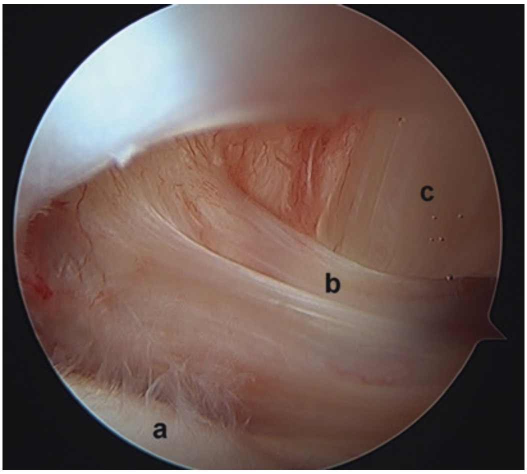


Fig. 4.13 Arthroscopic image of the peripheral compartment of a right hip with an articular communication with the iliopsoas tendon. The femoral head (**a**), the anterior capsule (**b**) and the iliopsoas tendon (**c**)

The zona orbicularis (ZO) is a major hip stabilizer and represents a circumferential thickening of the hip capsule forming a ring around the femoral neck. It consists of a thicker layer of annular fibres crossing the above-mentioned longitudinal ligaments and thus strengthening the capsule. Just cranial to the ZO, close to the anterior capsular recess, lies the psoas tendon. The iliopsoas bursa is located beneath the musculotendinous portion of the iliopsoas muscle, anterior to the hip joint capsule and lateral to the femoral vessels. The bursa separates the iliopsoas tendon from the articular capsule of the hip joint, and it may directly communicate with the PC in 15–20% of cases [69]. This finding may bear clinical relevance and can increase the risk of fluid extravasation during hip arthroscopy (Fig. 4.13).

4.2.5 How to Treat Cam Deformity: Tips and Tricks

Femoroacetabular impingement syndrome is most often associated with cam morphology. Successful treatment depends on precise reshaping of the proximal femur, whether done arthroscopically or by open surgery. We present ten steps to help get arthroscopic surgery right.

4.2.5.1 Be Sure of the Diagnosis

Not all patients with cam morphology have FAI syndrome, and coexisting diagnosis are common [72]. Be certain that this is the correct diagnosis before embarking on surgery. Local anaesthetic test injections and image intensifier examination under sedation, performed by the surgeon, and with pre- and postinjection exercise testing are very helpful. Be cautious of operating on patients with a negative injection test, and use the examination under sedation to inform the preoperative planning [73].

4.2.5.2 Preoperative Planning

We always perform CT with 3D reconstruction before surgery to reshape a cam. Modern CT protocols allow a low dose of radiation, without compromising the image quality. The 3D reconstruction acts as a map that can guide you around the hip (Fig. 4.14). Measurements from the scans allow accurate reshaping by quantifying depth and extent of bone resection. These scans should also include the distal femur so that femoral torsion can be measured and taken into account in planning the depth of resection.

Fig. 4.14 3D reconstruction of CT. A planned cam resection margin is marked

4.2.5.3 Setup

Surgery can be performed in supine or lateral positions. We prefer the lateral position as it is easy to achieve posterolateral access to the hip, the scrub nurse can work from other side of the patient, and the instruments do not fall out. Whichever approach is preferred, a range of motion from hyperextension to 100° flexion, abduction and internal and external rotation in flexion must be achievable. The foot must be easily detachable and reattachable from the traction system so that traction can be used where required while also allowing a wide range of motion (Fig. 4.15). We use a fully transparent drape with adhesive to allow visualization of the hip. An image intensifier, that can provide AP and lateral views, is required without interfering with the surgeon's position.

4.2.5.4 Portals

Most cases will need two to four portals depending on the technique and the shape and location of the cam. We typically use three portals: postero-superior, antero-superior and superior-lateral. Inject local anaesthetic with adrenaline into the skin before each incision to minimize bleeding. Avoid crowding by spacing out the entry points, and think carefully about where they penetrate the capsule more than the exact position of the skin

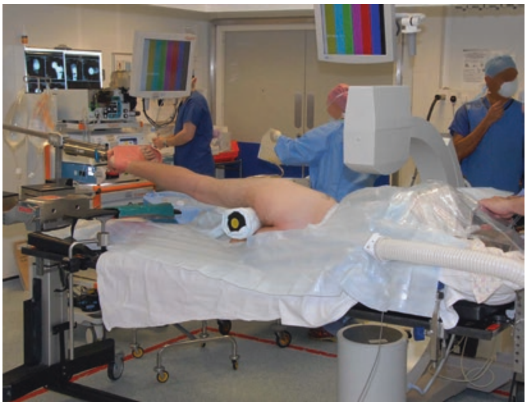


Fig. 4.15 Operating theatre set up with the patient in the lateral position. The surgeon stands behind the patient with the scrub nurse opposite. The C-arm, of the image intensifier, is under the radiolucent table, providing an AP view of the hip. The C-arm can rotate to provide a lateral view. When not in use the image intensifier moves to the distal end of the table

incision. Optimize triangulation by aiming for an angle of approximately 60° between each portal.

4.2.5.5 Capsulotomy

It is possible to reshape a cam without performing a capsulotomy, but for most cases a capsulotomy will improve visibility and access. We use an “L”-shaped capsulotomy extending from 11 to 1 o’clock, parallel to the labrum and from 1 o’clock towards the capsular insertion on the intertrochanteric line parallel to the femoral neck. This avoids dividing the ilio-femoral ligament and ensures postoperative stability of the hip. Only when the cam is large and extends well laterally does the second limb of the capsulotomy need to traverse the zona orbicularis. We control the capsulotomy flap with traction sutures and retractors inserted through the third portal.

4.2.5.6 Step-by-Step Reshaping

With the hip in traction, a 70° arthroscope in the postero-superior portal and a profile view of the postero-superior and superior head-neck junction, we begin resection just in front of the retinacular vessels. If the cam is very superior and lateral, then you may need to dissect these vessels as bone is removed, protecting them from injury. This is made easier by slightly abducting and extending the hip. Extend the intended line of the head-neck junction across the anterior aspect of the cam towards the inferior aspect of the neck, reducing traction and flexing the hip off traction to achieve a good view. Refer the 3D reconstruction of the CT to ensure the new head-neck junction is neither too medial nor too lateral. With various combinations of flexion and internal rotation of the hip, expose the more lateral aspect of the cam, and resect this right out to the normal neck surface. Make sure the resection extends all the way inferiorly by visualizing the medial synovial fold (Fig. 4.16). Frequently move the arthroscope, and change portals to properly appreciate the three-dimensional shape of the resection.

4.2.5.7 Create a Smooth Head-Neck Transition

Avoid a sharp edge at the junction between the femoral neck cartilage and the femoral neck, which might catch on the labrum during extension from a flexed position. Ideally, the profile should be an “S” shape with convexity at the edge of the femoral head and a concavity further lateral on the neck (Fig. 4.17). This will keep the labrum in contact

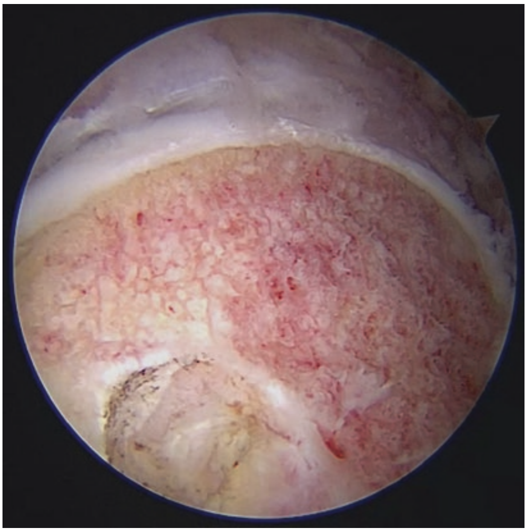


Fig. 4.16 View of antero-superior femoral head-neck junction following a cam resection

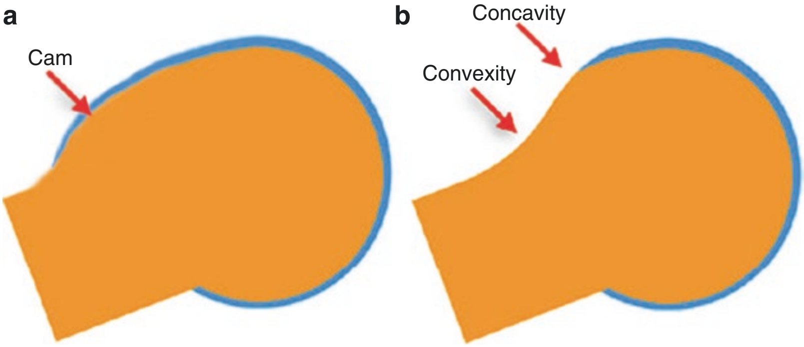


Fig. 4.17 Diagrammatic representation of cam morphology before (**a**) and after (**b**) resection. Note the “S”-shaped resection

with the head-neck junction throughout movement and help to maintain suction seal and stability.

4.2.5.8 Be Sure that the Shape Is Correct

Use an image intensifier to monitor progress of the resection, especially in the early part of your surgical experience. Always perform a dynamic impingement test observing the labrum and the head-neck junction through the arthroscope. Check in particular that the resection is adequate during flexion, adduction and internal rotation: flexion abduction and internal rotation and pure abduction.

4.2.5.9 Be Prepared to Close the Capsule

Hip instability is a devastating complication of hip arthroscopy, so we always consider whether to close the capsule. It may not be necessary in every patient, but we are more likely to do it if the patient has ligamentous laxity, a relatively shallow acetabulum, labral or ligamentum teres deficiency, performs a flexibility sport, has needed a more extensive capsulotomy dividing the zona orbicularis or has had a large resection. We close with several sutures including the corner of the "L" shape and the zona orbicularis.

4.2.5.10 Recovery and Rehabilitation

Use cryotherapy and CPM to facilitate early mobilization. Almost all patients can be fully weight bearing after surgery and can start exercising with a physiotherapist on the same day. We prefer to keep patients in the hospital for a day or two, so that they can begin with an intensive course of rehabilitation. Subsequent recovery is driven by milestones, which include a quiet hip with a full range of motion, normal core and hip muscle control and normal strength in dynamic movements. Most patients can return to full activity including sports training within 3 months.

Open surgery to correct cam morphology in FAI syndrome has been very successful where surgeons have been careful to achieve very accurate reshaping to a spherical head with a smooth head-neck transition [74]. Sometimes this is still the best approach. However, cam reshaping can be successfully performed in many patients by arthroscopic surgery [75]. Despite the extra challenges of a limited view and difficult access, arthroscopic surgery needs to achieve the same precision as an open approach. Only then does the less invasive technique provide an advantage. These ten steps provide a framework, but the best way to learn the surgical technique is in a hip preservation fellowship with a high-volume expert surgeon.

4.2.6 How to Manage a Chondro-Labral Lesion

The normal movement of the hip is purely rotatory due to the spherical congruency of the joint surfaces, so any change in this strict configuration will produce mechanical dysfunction and abnormal stresses on the articular cartilage. Cam deformity was described as a bony morphological change in the head-neck junction, mainly in the anterolateral area, that produces outside-in shear stress on the corresponding acetabular rim during the terminal flexion and internal rotation resulting in chondro-labral damage, usually labral detachment and chondral delamination (Fig. 4.18). On the other side, acetabular overcoverage may produce a pincer effect against the femoral neck on the terminal range of flexion.

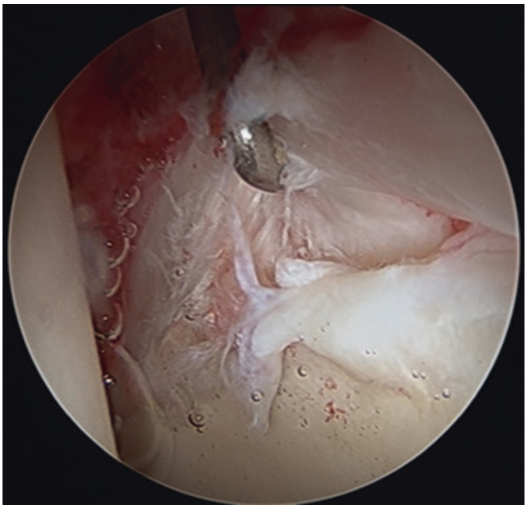


Fig. 4.18 Arthroscopic view of left hip with mixed type of FAI from anterolateral portal. The probe is showing labral detachment associated with a peripheral acetabular cartilage lesion

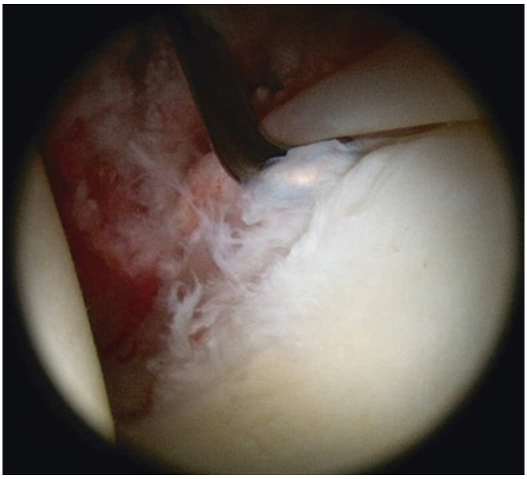


Fig. 4.19 Arthroscopic view of left hip with pincer type of FAI from anterolateral portal. A small labrum is typical in those cases, and detachment and degeneration due to impact at anterolateral zone are usually found

This compression mechanism against the labrum can lead to contusion and degeneration of it (Fig. 4.19). Secondarily, a levering mechanism of the head against the postero-inferior acetabular cartilage produces a contrecoup cartilage lesion [76, 77]. Instability is a wide term that includes the gross subluxation and dislocation caused by significant bony dysplasia as well as the new concept of microinstability caused by traumatic or atraumatic dysfunction of the soft tissues stabilizing the hip as the labrum, capsule, ligaments and muscles. In case of instability, there are abnormal translation movements of the femoral head producing inside-out shearing forces (mainly anterior to lateral) and causing chondrolabral injuries [78, 79] leading to detachment of the labrum in a similar way that cam lesion does.

4.2.6.1 Diagnosis

Chondro-labral lesions should be suspected in a young athletic patient presenting with groin pain during activity. As initial assessment, plain radiographs are used for detecting the underlying pathologies that predispose to chondro-labral lesions. Measurements of the centre-edge angle of Wiberg, acetabular index angle of Tönnis, alpha angle, head-neck offset and crossover sign of acetabular retroversion are reliable radiological findings for evaluating the bony morphology before and after any kind of hip preservative surgery [79].

Although the Tönnis classification is widely used for grading osteoarthritis based on specific radiographs, it cannot be used as an accurate predictor for condition of the cartilage in the early stages of the joint disease [80]. After development of magnetic resonance techniques, MRA with intra-articular injection of gadolinium has become the investigation of choice for detection of chondro-labral lesions with high sensitivity (71–100%) compared with standard MRI [81]. Nowadays, quantitative MRI techniques can map the concentration of glycosaminoglycans (GAGs) in the cartilage. Delayed gadolinium-enhanced MRI of cartilage (dGEMRIC) or T2 mapping is used for detecting early damage and for follow-up of patients after preservative hip surgeries [82].

4.2.6.2 Classification

Clinical interest towards hip pathologies produced many classification systems to describe the chondro-labral lesions. According to Beck et al. [76], labral condition is classified as (1) normal, (2) degeneration, (3) full-thickness tear, (4) detachment or (5) ossification, while articular cartilage is classified as (1) normal, (2) malacia, (3) debonding, (4) cleavage or (5) defect. Although most of these classification systems depend on the visual inspection of the lesion, the geographic description (Fig. 4.10) based on the six anatomical zones done by Ilizaliturri et al. [60] provides a simple and reproducible method with implications for prognosis and is commonly used for medical reporting.

4.2.6.3 Approach for Treatment

Since it was first described by Ganz et al. [83] in the early 2000s, surgical hip dislocation gained a worldwide popularity as a safe method for treatment of intra-articular hip lesions. However the evolution of arthroscopic techniques attracted the attention of hip surgeons, and today hip arthroscopy is a commonly performed procedure for intra-articular lesions and achieved favourable clinical outcomes compared with other surgical methods [84]. Cam deformity and acetabular overcoverage, if present, should be properly corrected as a first step of treatment to protect the repaired chondro-labral tissues and avoid further damage. Labral debridement was reported with satisfactory short-term clinical results in a few literature reports, but recent studies demonstrate better clinical outcomes in hip scores with labral preservation, becoming this option as the gold standard for labral damage repair. Biomechanical studies show that labral reattachment restores normal biomechanics. Labral preservation is the actual standard of care, so reattachment is performed when possible, and labral replacement should be considered when it is strongly degenerated or absent. The articular cartilage has poor intrinsic healing capacity, so the aim of treatment is to potentiate the cartilage healing and reproduce a new tissue with structural and biomechanical properties similar to the normal cartilage. Labral repair can be considered only in stable pocket lesions (Fig. 4.20), where viable chondrocytes can be demonstrated at the delaminated flaps of acetabular cartilage.

Debridement and bone marrow stimulation techniques are considered standard methods for small, focal full-thickness defect <2 cm^2 at the acetabular rim (Fig. 4.21), with improvement in the functional outcomes [85]. The microfracture technique depends on stimulation of subchondral bone marrow by penetrations that liberate undifferentiated stem cells, and then the blood clot formed in the defect provides a supporting environment for the cartilage progenitor cells and finally differentiates into stable fibrocartilage. Enhanced bone marrow stimulation techniques (EBMST) have been developed to improve the results of surgical standard methods of cartilage repair. For instance, AMIC technique adds membranes to the microfractured area, and chitosan-glycerol phosphate works as a scaffold material. These techniques augmented the biomechanical properties of clot formed in the microfractured area and provided more stable environment for growth and differentiation of hyaline cartilage. Both are recommended for full-thickness defect >2 cm^2 after adequate debridement and microfracture [86].

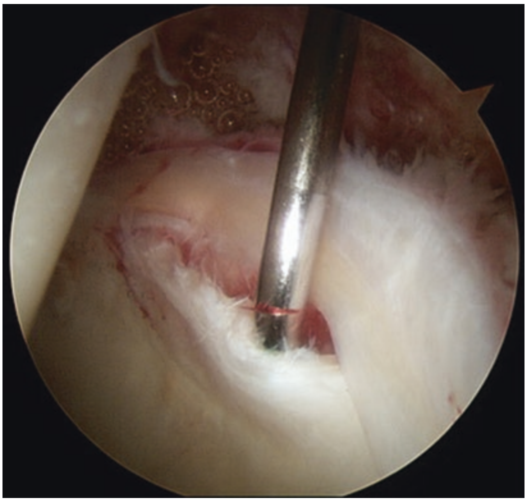


Fig. 4.20 Arthroscopic view of left hip with cam type of FAI from anterolateral portal. Cartilage delamination due to shear force named pocket lesion. Cartilage repair may be an option in these cases

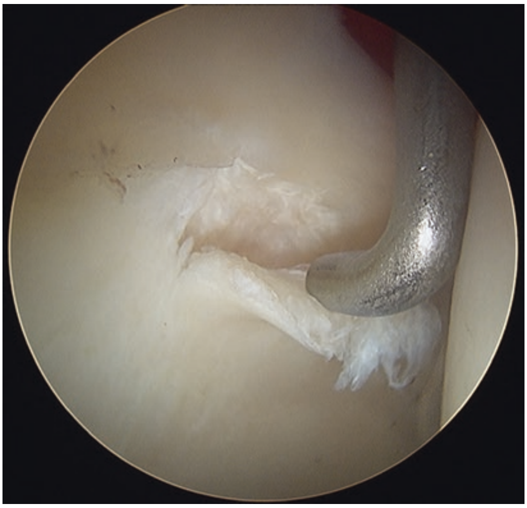


Fig. 4.21 Arthroscopic view of the right hip with cam type of FAI from anterolateral portal. Cartilage delamination has evolved to an unstable flap

4.3 Take-Home Message

Hip arthroscopy is an evolving technique that is expanding its indications. We should be very cautious during the learning curve period, and we must be very careful with the small details mentioned in this chapter. Special care should be taken to get a stable and comfortable initial setup of the patient. Knowledge of arthroscopic anatomy and its normal variants could facilitate identification of the anatomical deformities and its proper correction.

References

1. Maradit Kremers H, Schilz SR, Van Houten HK, Herrin J, Koenig KM, Bozic KJ, et al. Trends in utilization and outcomes of hip arthroscopy in the united states between 2005 and 2013. J Arthroplast. 2017;32(3):750–5.
2. Kelly BT, Williams RJ, Philippon MJ. Hip arthroscopy: current indications, treatment options, and management issues. Am J Sports Med. 2003;31(6):1020–37. http://www.ncbi.nlm.nih.gov/pubmed/14623676
3. Domb B, Botser I. Loose bodies: tips and pearls. In: Thomas Byrd JW, editor. Operative hip arthroscopy. New York: Springer; 2013. p. 161–9. https://doi.org/10.1007/978-1-4419-7925-4_12.
4. De Sa D, Cargnelli S, Catapano M, et al. Efficacy of hip arthroscopy for the management of septic arthritis: a systematic review. Arthrosc J Arthrosc Relat Surg. 2015;31(7):1358–70. https://doi.org/10.1016/j.arthro.2014.12.028.
5. Lynch TS, Terry MA, Bedi A, Kelly BT. Hip arthroscopic surgery: patient evaluation, current indications, and outcomes. Am J Sports Med. 2013;41(5):1174–89. https://doi.org/10.1177/0363546513476281.
6. Stevens MS, LeGay DA, Glazebrook MA, Amirault D. The evidence for hip arthroscopy: grading the current indications. Arthrosc J Arthrosc Relat Surg. 2010;26(10):1370–83. https://doi.org/10.1016/j.arthro.2010.07.016.
7. Ayeni OR, Alradwan H, de Sa D, Philippon MJ. The hip labrum reconstruction: indications and outcomes-a systematic review. Knee Surg Sport Traumatol Arthrosc. 2014;22(4):737–43. https://doi.org/10.1007/s00167-013-2804-5.
8. Ganz R, Parvizi J, Beck M, Leunig M, Nötzli H, Siebenrock KA. Femoroacetabular impingement: a cause for osteoarthritis of the hip. Clin Orthop Relat Res. 2003;417:112–20. https://doi.org/10.1097/01.blo.0000096804.78689.c2.
9. Ayeni OR, Wong I, Chien T, Musahl V, Kelly BT, Bhandari M. Surgical indications for arthroscopic management of femoroacetabular impingement. Anthroscopy. 2012;28:1170–9. https://doi.org/10.1016/j.arthro.2012.01.010.
10. Reich MS, Shannon C, Tsai E, Salata MJ. Hip arthroscopy for extra-articular hip disease. Curr Rev Musculoskelet Med. 2013;6(3):250–7. https://doi.org/10.1007/s12178-013-9177-8.
11. Park M, Yoon S-J, Jung S, Kim S-H. Clinical results of endoscopic sciatic nerve decompression for deep gluteal syndrome: mean 2-year follow-up. BMC Musculoskelet Disord. 2016;17(1):218. https://doi.org/10.1186/s12891-016-1062-3.
12. Martin HD, Shears SA, Johnson JC, Smathers AM, Palmer IJ. The endoscopic treatment of sciatic nerve entrapment/deep gluteal syndrome. Arthrosc J Arthrosc Relat Surg. 2011;27(2):172–81. https://doi.org/10.1016/j.arthro.2010.07.008.
13. Allen W, Cope R. Coxa saltans: the snapping hip revisited. J Am Acad Orthop Surg. 1995;3(5):303–8. http://www.ncbi.nlm.nih.gov/pubmed/10790668
14. Byrd JWT. Snapping hip. Oper Tech Sports Med. 2005;13(1):46–54. https://doi.org/10.1053/j.otsm.2004.09.003.
15. Shbeeb MI, O'Duffy JD, Michet CJ, O'Fallon WM, Matteson EL. Evaluation of glucocorticosteroid injection for the treatment of trochanteric bursitis. J Rheumatol. 1996;23(12):2104–6.
16. Domb BG, Nasser RM, Botser IB. Partial-thickness tears of the gluteus medius: rationale and technique for trans-tendinous endoscopic repair. Arthrosc J Arthrosc Relat Surg. 2010;26(12):1697–705. https://doi.org/10.1016/j.arthro.2010.06.002.
17. Newman JT, Saroki AJ, Philippon MJ. Hip arthroscopy for the management of trauma: a literature review. J Hip Preserv Surg. 2015;2(3):242–8. https://doi.org/10.1093/jhps/hnv047.
18. Shindle MK, Ranawat AS, Kelly BT. Diagnosis and management of traumatic and atraumatic hip instability in the athletic patient. Clin Sports Med. 2006;25(2):309–26. https://doi.org/10.1016/j.csm.2005.12.003.
19. Heaven S, Simunovic N, Williams DS, Naudie D, Ayeni OR. Hip arthroscopy in the setting of hip arthroplasty. Knee Surg Sport Traumatol Arthrosc. 2016;24(1):287–94. https://doi.org/10.1007/s00167-014-3379-5.
20. Kemp JL, MacDonald D, Collins NJ, Hatton AL, Crossley KM. Hip arthroscopy in the setting of hip osteoarthritis: systematic review of outcomes and progression to hip arthroplasty. Clin Orthop Relat Res. 2014;473(3):1055–73. https://doi.org/10.1007/s11999-014-3943-9.
21. Domb BG, Gui C, Lodhia P. How much arthritis is too much for hip arthroscopy: a systematic review. Arthrosc J Arthrosc Relat Surg. 2015;31(3):520–9. https://doi.org/10.1016/j.arthro.2014.11.008.
22. Palmer A, Malak TT, Broomfield J, Holton J, Majkowski L, Thomas GE, Taylor A, Andrade AJ, Collins G, Watson K, Carr AJ, Glyn-Jones S. Past and projected temporal trends in arthroscopic hip surgery in England between 2002 and 2013. BMJ Open Sport Exerc Med. 2016;2(1):e000082. eCollection 2016
23. Leunig M, Ganz R. The evolution and concepts of joint-preserving surgery of the hip. Bone Joint J. 2014;96-b(1):5–18.

24. Imam S, Khanduja V. Current concepts in the diagnosis and management of femoroacetabular impingement. Int Orthop. 2011;35(10):1427–35.
25. Bedi AK, Kelly BT, Khanduja V. Arthroscopic hip preservation surgery: current concepts and perspective. Bone Joint J. 2013;95-B(1):10.
26. Konan S, Rhee SJ, Haddad FS. Hip arthroscopy: analysis of a single surgeon's learning experience. J Bone Joint Surg Am. 2011;93(Suppl 2):52–6.
27. Aim F, Lonjon G, Hannouche D, Nizard R. Effectiveness of virtual reality training in orthopaedic surgery. Arthroscopy. 2016;32(1):224–32.
28. Boutefnouchet T, Laios T. Transfer of arthroscopic skills from computer simulation training to the operating theatre: a review of evidence from two randomised controlled studies. SICOT-J. 2016;2:4.
29. Howells NR, Gill HS, Carr AJ, Price AJ, Rees JL. Transferring simulated arthroscopic skills to the operating theatre: a randomised blinded study. J Bone Joint Surg Br Vol. 2008;90(4):494–9.
30. Khanduja VL, Lawrence JE, Audenaert E. Testing the construct validity of a virtual reality hip arthroscopy simulator. Arthroscopy. 2017;33(3):566–71.
31. Angelo RL, Ryu RK, Pedowitz RA, Beach W, Burns J, Dodds J, et al. A proficiency-based progression training curriculum coupled with a model simulator results in the acquisition of a superior arthroscopic Bankart skill set. Arthroscopy. 2015;31(10):1854–71.
32. Cannon WD, Garrett WE Jr, Hunter RE, Sweeney HJ, Eckhoff DG, Nicandri GT, et al. Improving residency training in arthroscopic knee surgery with use of a virtual-reality simulator. A randomized blinded study. J Bone Joint Surg Am. 2014;96(21):1798–806.
33. Sunil Kumar KL, Lawrence JE, Khanduja V. Training young adult hip surgeons for the future: the Cambridge vision. Bone Joint 360. 2016;5(6):8–12.
34. Dietrich F, Ries C, Eiermann C, Miehlke W, Sobau C. Complications in hip arthroscopy: necessity of supervision during the learning curve. Knee Surg Sports Traumatol Arthrosc. 2014;22(4):953–8.
35. Hoppe DJ, de Sa D, Simunovic N, Bhandari M, Safran MR, Larson CM, et al. The learning curve for hip arthroscopy: a systematic review. Arthroscopy. 2014;30(3):389–97.
36. Perez-Carro L, Tey M. What goes on during the learning curve? In: Marín-Peña O, editor. Femoroacetabular impingement. Berlin Heidelberg: Springer; 2012. p. 123–30.
37. Griffiths EJ, Khanduja V. Hip arthroscopy: evolution, current practice and future developments. Int Orthop. 2012;36(6):1115–21.
38. Harris JD, McCormick FM, Abrams GD, Gupta AK, Ellis TJ, Bach BR Jr, et al. Complications and reoperations during and after hip arthroscopy: a systematic review of 92 studies and more than 6,000 patients. Arthroscopy. 2013;29(3):589–95.
39. de Amorim Cabrita HA, de Castro Trindade CA, de Campos Gurgel HM, Leal RD, de Souza Marques RDF. Hip arthroscopy. Rev Bras Ortop. 2015;50(3):245–53.
40. Souza BG, Dani WS, Honda EK, Ricioli W Jr, Guimaraes RP, Ono NK, et al. Do complications in hip arthroscopy change with experience? Arthroscopy. 2010;26(8):1053–7.
41. Smart LR, Oetgen M, Noonan B, Medvecky M. Beginning hip arthroscopy: indications, positioning, portals, basic techniques, and complications. Arthroscopy. 2007;23(12):1348–53.
42. Vaughn ZD, Safran MR. Arthroscopic femoral osteoplasty/chielectomy for cam-type femoroacetabular impingement in the athlete. Sports Med Arthrosc Rev. 2010;18(2):90–9.
43. Mannava S, Howse EA, Stone AV, Stubbs AJ. Basic hip arthroscopy: supine patient positioning and dynamic fluoroscopic evaluation. Arthrosc Tech. 2015;4(4):e391–6.
44. Boyle JR, Silva JA, Mc Millan S, Busconi BD. Mechanics of hip arthroscopy. Clin Sports Med. 2011;30(2):285–92.
45. Byrd JW. Hip arthroscopy. J Am Acad Orthop Surg. 2006;14(7):433–44.
46. Shetty VD, Villar RN. Hip arthroscopy: current concepts and review of literature. Br J Sports Med. 2007;41(2):64–8. discussion 8
47. Mason JB, McCarthy JC, O'Donnell J, Barsoum W, Mayor MB, Busconi BD, et al. Hip arthroscopy: surgical approach, positioning, and distraction. Clin Orthop Relat Res. 2003;406:29–37.
48. Bond JL, Knutson ZA, Ebert A, Guanche CA. The 23-point arthroscopic examination of the hip: basic setup, portal placement, and surgical technique. Arthrosc J Arthrosc Relat Surg. 2009;25(4):416–29.
49. Dorfmann H, Boyer T, De Bie B. Arthroscopy of the hip. Rev Rhum Mal Osteoartic. 1988;55(1):33–6.
50. Cuellar Gutierrez R, Ruiz Iban MA, Cuellar Ayestaran A, Sanchez Sobrino A, Oteo-Alvaro A. Peripheral compartment as the initial access for hip arthroscopy in complex cases: technical note. Orthopedics. 2013;36(6):456–62.
51. Rupp R, Duggan B. Peripheral versus central compartment starting point in hip arthroscopy for femoroacetabular impingement. Orthopedics. 2012;35(2):e148–53.
52. Said HG, Steimer O, Kohn D, Dienst M. Vascular obstruction at the level of the ankle joint as a complication of hip arthroscopy. Arthrosc J Arthrosc Relat Surg. 2011;27(11):1594–6.
53. Dienst M, Seil R, Kohn DM. Safe arthroscopic access to the central compartment of the hip. Arthrosc J Arthrosc Relat Surg. 2005;21(12):1510–4.
54. Masoud MA, Said HG. Intra-articular hip injection using anatomic surface landmarks. Arthrosc Tech. 2013;2(2):e147–9.
55. Thaunat M, Murphy CG, Chatellard R, Sonnery-Cottet B, Graveleau N, Meyer A, et al. Capsulotomy first: a novel concept for hip arthroscopy. Arthrosc Tech. 2014;3(5):e599–603.

56. Thorey F, Ezechieli M, Ettinger M, Albrecht U-V, Budde S. Access to the hip joint from standard arthroscopic portals: a cadaveric study. Arthrosc J Arthrosc Relat Surg. 2013;29(8):1297–307.
57. Dorfmann H, Boyer T. Hip arthroscopy utilizing the supine position. YJARS. 1996;12(2):264–7.
58. Philippon MJ, Stubbs AJ, Schenker ML, Maxwell RB, Ganz R, Leunig M. Arthroscopic management of femoroacetabular impingement: osteoplasty technique and literature review. Am J Sports Med. 2007;35(9):1571–80.
59. Marín-Peña O, Fernández-Tormos E, Dantas P, Rego P, Pérez-Carro L. Anatomía y función de la articulación coxofemoral. Anatomía artroscópica de la cadera. Rev Esp Artrosc Cir Artic. 2016;23(1):3–10.
60. Ilizaliturri VM, Byrd JWT, Sampson TG, Guanche CA, Philippon MJ, Kelly BT, et al. A geographic zone method to describe intra-articular pathology in hip arthroscopy: cadaveric study and preliminary report. Arthroscopy. 2008;24(5):534–9.
61. Rao J, Zhou YX, Villar RN. Injury to the ligamentum teres. Mechanism, findings, and results of treatment. Clin Sports Med. 2001;20(4):791–9–vii.
62. Cerezal L, Kassarjian A, Canga A, Dobado MC, Montero JA, Llopis E, et al. Anatomy, biomechanics, imaging, and management of ligamentum teres injuries. Radiographics. 2010;30(6):1637–51.
63. Kalhor M, Horowitz K, Beck M, Nazparvar B, Ganz R. Vascular supply to the acetabular labrum. J Bone Joint Surg Am. 2010;92(15):2570–5.
64. Saddik D, Troupis J, Tirman P, O'Donnell J, Howells R. Prevalence and location of acetabular sublabral sulci at hip arthroscopy with retrospective MRI review. Am J Roentgenol. 2006;187(5):W507–11.
65. Dietrich TJ, Suter A, Pfirrmann C, Dora C. Supraacetabular fossa (pseudodefect of acetabular cartilage): frequency at MR arthrography and comparison of findings at MR arthrography and arthroscopy. Radiology. 2012;263(2):484–91.
66. Byrd JWT. Supraacetabular fossa. Radiology 2012;265(2):648–8.
67. Dienst M. Hip arthroscopy: technique and anatomy. Oper Tech Sports Med. 2005;13(1):13–23.
68. Nguyen MS, Kheyfits V, Giordano BD, Dieudonne G, Monu JUV. Hip anatomic variants that may mimic abnormalities at MRI: labral variants. Am J Roentgenol. 2013;201(3):W394–400.
69. Nguyen MS, Kheyfits V, Giordano BD, Dieudonne G, Monu JUV. Hip anatomic variants that may mimic pathologic entities on MRI: nonlabral variants. Am J Roentgenol. 2013;201(3):W401–8.
70. Mascarenhas VV, Rego P, Dantas P, Gaspar A, Soldado F, Consciência JG. Cam deformity and the omega angle, a novel quantitative measurement of femoral head-neck morphology: a 3D CT gender analysis in asymptomatic subjects. Eur Radiol. 2017; 27(5):2011–23. Epub 2016 Aug 30.
71. Rego PRA, Mascarenhas V, Oliveira FS, Pinto PC, Gaspar A, Ovídio J, et al. Morphologic and angular planning for cam resection in femoro-acetabular impingement: value of the omega angle. Int Orthop. 2016;40(10):2011–7. Epub 2015 Nov 18.
72. Griffin DR, Dickenson EJ, Agricola R, Awan T, Beck M, Dijkstra P, et al. The 2016 Warwick agreement on femoroacetabular impingement. Br J Sports Med. 2016;50(19):1169–76.
73. Khan W, Khan M, Alradwan H, Williams R, Simunovic N, Ayeni OR. Utility of intra-articular hip injections for femoroacetabular impingement: a systematic review. Orthop J Sports Med. 2015;3(9):2325967115601030.
74. Steppacher SD, Anwander H, Zurmühle CA, Tannast M, Siebenrock KA. Eighty percent of patients with surgical hip dislocation for femoroacetabular impingement have a good clinical result without osteoarthritis progression at 10 years. Clin Orthop Relat Res. 2015;473(4):1333–41.
75. Philippon M, Briggs K, Yen Y-M, Kuppersmith D. Outcomes following hip arthroscopy for femoroacetabular impingement with associated chondrolabral dysfunction. J Bone Joint Surg Br Vol. 2009;91(1):16–23.
76. Beck M, Kalhor M, Leunig M, Ganz R, Surgeon O. Hip morphology influences the pattern of damage to the acetabular cartilage femoroacetabular impingement as a cause of early osteoarthritis of the hip. J Bone Joint Surg Br. 2005;87:1012–8. https://doi.org/10.1302/0301-620X.87B7.
77. Martin DE, Tashman S. The biomechanics of femoroacetabular impingement. Oper Tech Orthop. 2010;20(4):248–54. https://doi.org/10.1053/j.oto.2010.09.015.
78. Shibata KR, Matsuda S, Safran MR. Is there a distinct pattern to the acetabular labrum and articular cartilage damage in the non-dysplastic hip with instability? Knee Surg Sport Traumatol Arthrosc. 2016:1–10. https://doi.org/10.1007/s00167-016-4342-4.
79. Philippon MJ. Microinstability of the hip: a previously unrecognized pathology. 2016;6(3):354–60.
80. Valera M, Ibañez N, Sancho R, Tey M. Reliability of Tonnis classification in early hip arthritis: a useless reference for hip-preserving surgery. Arch Orthop Trauma Surg. 2016;136(1):27–33. https://doi.org/10.1007/s00402-015-2356-x.
81. McCarthy JC, Glassner PJ. Correlation of magnetic resonance arthrography with revision hip arthroscopy. Clin Orthop Relat Res. 2013;471(12):4006–11. https://doi.org/10.1007/s11999-013-3202-5.
82. Lattanzi R, Petchprapa C, Glaser C, et al. A new method to analyze dGEMRIC measurements in femoroacetabular impingement: preliminary validation against arthroscopic findings. Osteoarthr Cartil. 2012;20(10):1127–33. https://doi.org/10.1016/j.joca.2012.06.012.
83. Ganz R, Gill TJ, Gautier E, Ganz K, Krügel N, Berlemann U. Surgical dislocation of the adult hip: A technique with full access to the femoral head and acetabulum without the risk of avascular necrosis. J Bone Joint Surg Br. 2001;83-B:1119–24.

84. Botser IB, Smith TW, Nasser R, Domb BG. Open surgical dislocation versus arthroscopy for femoroacetabular impingement: a comparison of clinical outcomes. Arthrosc J Arthrosc Relat Surg. 2011;27(2):270–8. https://doi.org/10.1016/j.arthro.2010.11.008.
85. MacDonald AE, Bedi A, Horner NS, et al. Indications and outcomes for microfracture as an adjunct to hip arthroscopy for treatment of chondral defects in patients with femoroacetabular impingement: a systematic review. Arthroscopy. 2016;32(1):190–200. https://doi.org/10.1016/j.arthro.2015.06.041.
86. Tey M, Mas J, Pelfort X, Monllau JC. Arthroscopic treatment of hip chondral defects with bone marrow stimulation and BST-CarGel. Arthrosc Tech. 2015;4(1):e29–33. https://doi.org/10.1016/j.eats.2014.10.002.

Visualization and Anesthesia in Shoulder Arthroscopy: How to Overcome Bleeding and Poor Exposure

Jens Agneskirchner, Nestor Zurita,
Malte Holschen, and Harald Pilger

5.1 Introduction

The evolution of surgical techniques for the treatment of shoulder injuries has made significant progress with the development of arthroscopic techniques. This evolution is supported by improvements in materials and expanding surgeons' experience, which results in an increasing number of indications for arthroscopy for both diagnostic and therapeutic purposes.

A correct performance of a shoulder arthroscopy requires an appropriate visualization using a variety of technical equipment. Among the many factors that can negatively impact the quality of the arthroscopic surgery, poor visualization due to swelling and bleeding is probably the most common avoidable cause. Shoulder arthroscopy, more than arthroscopy of any other joint, requires active measures for control of bleeding to enhance visualization.

During a shoulder arthroscopy, particularly procedures involving the subacromial space, bleeding is a frequent complication that limits the surgeon's field of view and affects the operative technique. Additionally, the duration of surgery can be greatly increased as a result of such a complication.

Visual clarity is essential to perform a safe and successful arthroscopic procedure [1]. The mixing of blood with the irrigation fluid during arthroscopic procedures is the most common factor influencing visual clarity.

The use of specific equipment such as thermal electrocautery devices and pressurized irrigation systems to control bleeding has shown a positive effect on visual clarity. Some studies have found that the use of epinephrine reduces bleeding and increases visual clarity.

In this chapter we describe some general essential parameters for shoulder arthroscopy to avoid obscured visualization and decrease the problem of bleeding. We divide these factors in four sections:

1. Technical equipment
2. Patient positioning
3. Surgical technique
4. Management of anesthesia

J. Agneskirchner (✉)
Gelenkchirurgie Orthopädie Hannover go:h,
Hannover, Germany
e-mail: jens.agneskirchner@g-o-hannover.de

N. Zurita
Arthroscopy Department, Medical Services
of the Royal Spanish Swimming Federation, Hospital
IMED Elche, Alicante, Spain

M. Holschen
OPPK Orthopädische Praxisklinik,
Münster, Germany

H. Pilger
Institut für Anästhesie und Schmerztherapie,
Hannover, Germany

G.M.M.J. Kerkhoffs et al. (eds.), *ESSKA Instructional Course Lecture Book*,
https://doi.org/10.1007/978-3-662-56127-0_5

5.2 State-of-the-Art Treatment

5.2.1 Technical Equipment

5.2.1.1 Fluid Management

We recommend the following issues for fluid management:

- Saline bag. The addition of epinephrine (0.33 mg/L) to the irrigation fluid significantly improves visual clarity in the most common types of therapeutic shoulder arthroscopy. A significant reduction in total operating time and use of irrigation fluid was observed [2, 3].
- Inflow tubing systems.
- A specialized automated pump (so-called dual-wave pump, controlling both inflow and outflow) [4, 5].
- Outflow tubing with or without pressure-sensing feedback to an automated pump.

Pump systems used for arthroscopic surgery have evolved over the years to provide improved intraoperative visualization [6].

Advantages of mechanical fluid irrigation system over gravity irrigation are:

- Consistent flow
- Greater and more reproducible degree of joint distention
- Improved visualization, especially when motorized operative instruments are used
- A tamponade effect on bleeding
- Decreased operative time

Disadvantages include the need for additional equipment with increased cost and maintenance, initial learning curve for the surgical team, and possible extraarticular fluid dissection leading to soft tissue swelling [7].

Although significant advances have been made in arthroscopic equipment, few investigations exist that compare different pump systems. Even though improvements in visualization have been noted with dual systems, more research is necessary to determine the exact clinical significance.

5.2.1.2 Radiofrequency Devices

Bleeding from smaller vessels during arthroscopic procedures of the shoulder joint is often unavoidable, especially while working in extra-articular (e.g., subacromial) space and in case of inflammatory tissue reaction. The use of thermal coagulation electrode (radiofrequency devices) devices is indispensable because it enables direct coagulation of bleeding vessels and tissue dissection with concomitant vessel ablation.

Most reconstructive arthroscopic procedures such as a rotator cuff repair (especially complex tears) are only possible using these thermal devices.

Several highly effective systems are available and offered as single-use devices by the industry. We recommend the following parameters:

- Bipolar electrode using rather low temperatures at the tip of the probe inside the shoulder (<65°)
- Plasma layer creating probe
- Slow profile probe with ablation angle of 90° related to instrument shaft
- Highly effective outflow tubing to evacuate air/gas bubbles
- Metal proximity detection feature to avoid damage to scope or other instruments
- Foot switch control

5.3 Patient Positioning

Any successful surgical procedure begins with correct positioning of the patient. In shoulder arthroscopy, two different options of patient positioning exist (Figs. 5.1 and 5.2):

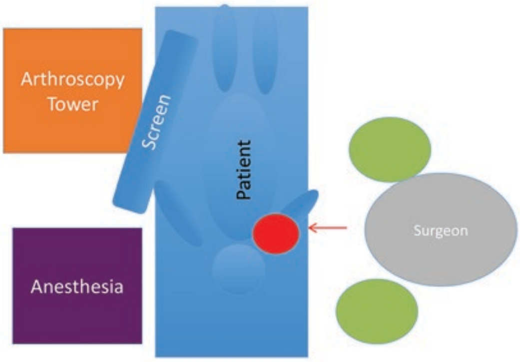


Fig. 5.1 Operation theater setup for shoulder arthroscopy: the arthroscopy tower and the screen are set up at the opposite side of the patient, facilitating access to the shoulder from a posterior, lateral, and anterior direction and leaving working space for the surgeon and assistants

Fig. 5.2 Operation theater setup for lateral decubitus position

- Lateral decubitus position
- The beach chair position

Whether one or the other is chosen is usually based on the surgeon's preference or in many centers a question related to the specific procedure, because each positioning offers specific advantages and disadvantages.

Special equipments such as positioning devices including arm holders are required for both types of positioning (Figs. 5.3, 5.4, 5.5, and 5.6).

Beach chair positioning is associated with the danger of cerebral oxygen desaturation and potential neurological complications. This is why regional cerebral oximetry should be utilized during shoulder arthroscopy in this position. In terms of potential neurological injury, the lateral decubitus position seems to be a safer option because cerebral blood flow is not compromised by the upright position of the head under general anesthesia (see also below).

However, in our experience, the majority of arthroscopic procedures are facilitated by the use of the beach chair position, including posterosuperior (supra- and infraspinatus) and anterior (subscapularis) tendon repairs. More complex extra-articular procedures such as the arthroscopic Latarjet procedure or plexus release require extensive anterior extra-articular shoulder dissection and access to the medial anterior shoulder, which is much easier to achieve in the beach chair position.

Fig. 5.3 Arthroscopy tower including video chain, automated pump, and radiofrequency console

The exact way to position the arm in the beach chair position is crucial to have adequate access to the subacromial space, e.g., during cuff repair.

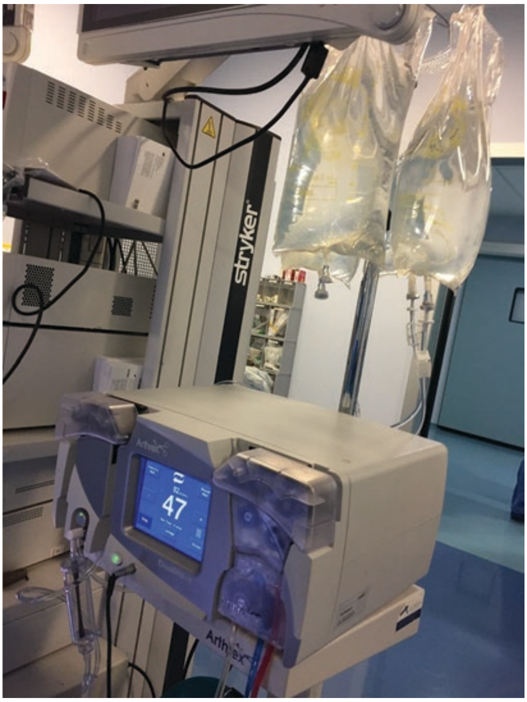


Fig. 5.4 Dual-wave pump, specifically suitable for shoulder arthroscopy. These pumps control both in- and outflow, thus minimizing turbulent flow

Fig. 5.5 High-flow arthroscopy shaft

We recommend putting the shoulder into a slight forward flexion (approximately 20°) and slight abduction while simultaneously applying slight (1–2 kg) traction of the upper arm parallel to the thorax. This will allow the subacromial space to open up and slightly move the humeral head backward and away from the narrow anterior part of the subacromial space. Usually, this allows excellent and wide access to the humeral and cuff (Fig. 5.7). Moreover, in the beach chair position, rotational maneuvers of the arm can be made to facilitate access and entrance for implants and instruments using different portals.

Fig. 5.6 Bipolar radiofrequency device (ArthroCare®)

Fig. 5.7 (Left) Wrong position of the arm in the beach chair. (Right) Correct position of the arm in slight flexion and abduction, which leads to subacromial opening and improved joint access

The lateral decubitus position (Fig. 5.8) allows for excellent visualization for intra-articular glenohumeral reconstruction such as instability repair. The use of double arm traction both distalizes and lateralizes the humeral space giving easier access to the inferior and posterior part of the labrum and capsule during instability repair. It seems that this is a reason why the lateral decubitus positioning is related to lower recurrence rates after arthroscopic Bankart repair than beach chair positioning.

Fig. 5.8 Double arm traction for lateral decubitus position

5.4 Surgical Technique

A careful surgical arthroscopic technique is another decisive factor to improve visualization during the procedure.

A very important principle is to minimize outflow of the irrigation fluid from the portals. This can be achieved by cannula placement or simply the assistant closing the portals with the fingers, thus eliminating outflow. Uncontrolled outflow will lead to turbulent flow inside the shoulder (Bernoulli effect), which opens small lacerated arterial vessels [8]. This will lead to bleeding and obscured vision.

We do not routinely place cannulas in each portal, so we often have several potential foci for the Bernoulli effect, which leads to its accompanying clouding of the arthroscopic field. We routinely have our surgical technician apply digital pressure over each of the non-cannulated portals, plugging the stream at each site. This simple maneuver (Fig. 5.9) can dramatically improve visualization, leaving the surgeon free to proceed with his operation instead of needlessly wasting valuable time “chasing bleeders” and uttering epithets. Recognizing and neutralizing the Bernoulli effect (Fig. 5.10) have helped us immensely to perform demanding subacromial techniques such as the repair of large and massive rotator cuff tears more rapidly and efficiently (Fig. 5.11).

We recommend some general arthroscopic principles in surgical technique:

- Reduce blood pressure if medical condition allows, to maintain systolic pressure of less than 90 mmHg (see below in detail).
- Incise the skin only and avoid deeper muscle laceration.
- Use a blunted conical trocar for penetration of the muscle, joint, and subacromial space.
- Avoid debridement of anterior medial acromion and the undersurface of the AC joint for as long as possible.
- Direct control of bleeding points by:
  - Thermal electrocautery devices (both monopolar and bipolar). Use them immediately when significant bleeders are encountered.
  - Locally infused vasoconstrictors (addition of epinephrine to the arthroscopy irrigation fluid).
- Indirect control of bleeding by:
  - Minimizing the fluid pressure differential between the patient’s blood pressure and the pressure of the infused irrigation fluid [9]. This is done by a combination of lowering the patient’s blood pressure if possible (hypotensive anesthesia; see below) and raising the pressure of the irrigation fluid.
  - The relation between subacromial pressure, blood pressure, and visual clarity during arthroscopic subacromial decompression was studied by Morrison et al. [7]. They concluded that a pressure of 49 mmHg is sufficient to prevent bleeding during surgery. In our experience, using a pump pressure between 40 and 45 mmHg, positioning the patient in the deckchair position, and using a saline/epinephrine solution, combined with sound knowledge of the vascular anatomy of the subacromial space, are sufficient to perform most interventions without difficulties. In the event of cumbersome bleeding, transient increase in the pump pressure may be necessary to identify and cauterize the offending vessels.

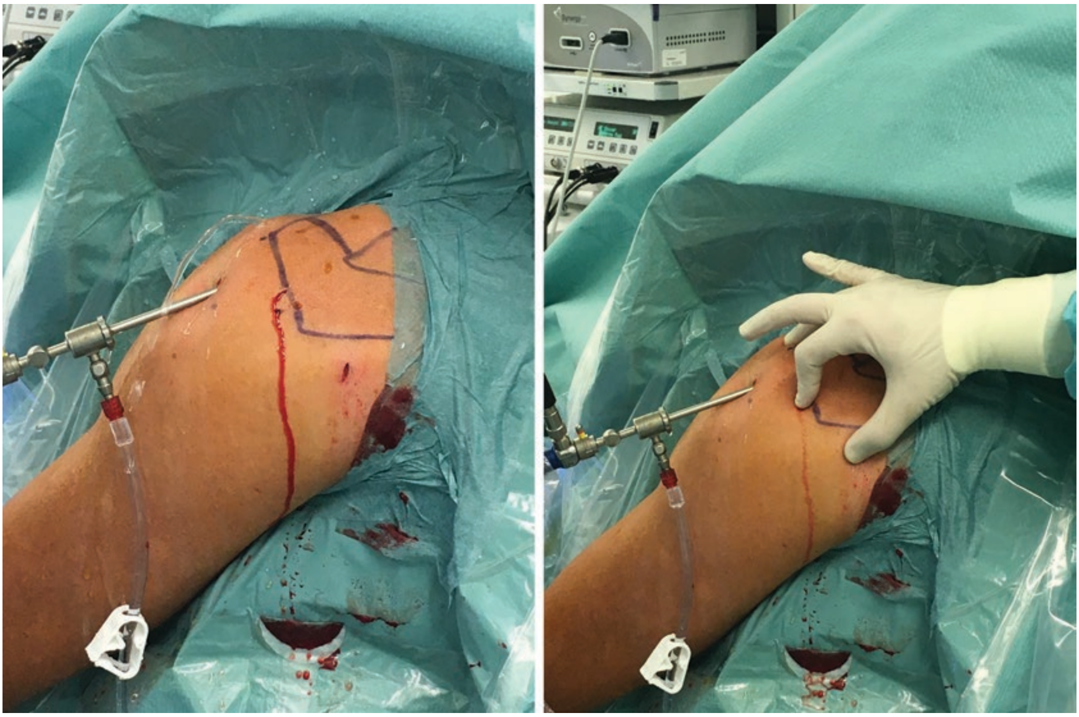


Fig. 5.9 (Left) Outflow of irrigation fluid leading to turbulent flow and bleeding (Bernoulli effect). By closing the portals with the fingers of the assistant (right), this can easily be avoided

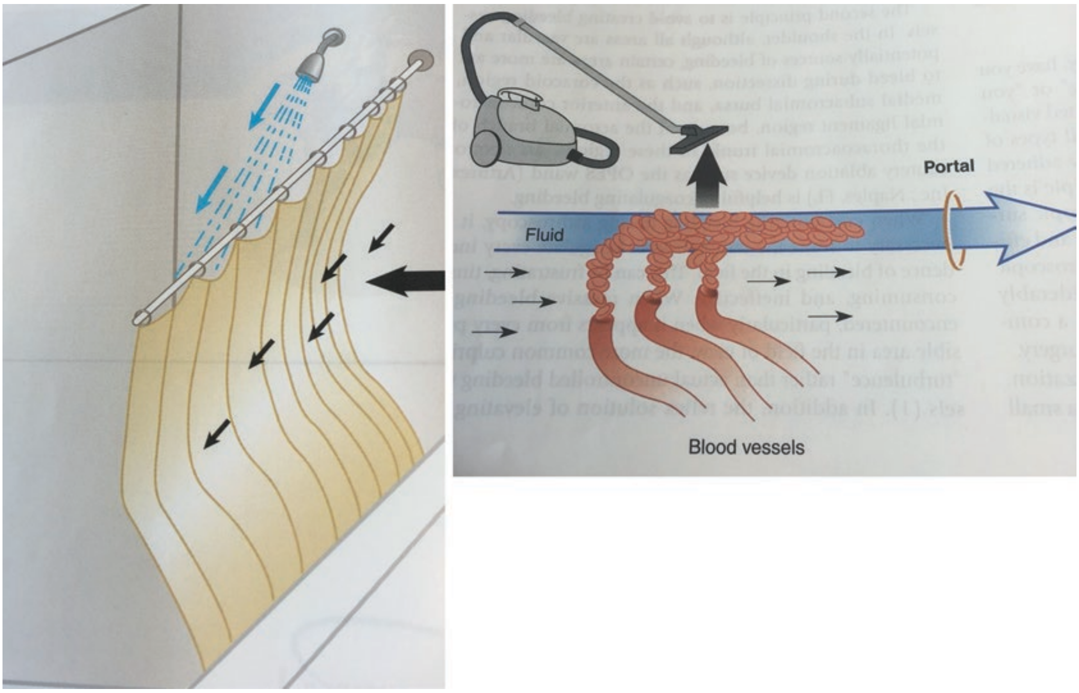


Fig. 5.10 Bernoulli effect: the uncontrolled outflow of fluid leading to turbulent flow will "suck" out the blood from small vessels and obscure the field (right)

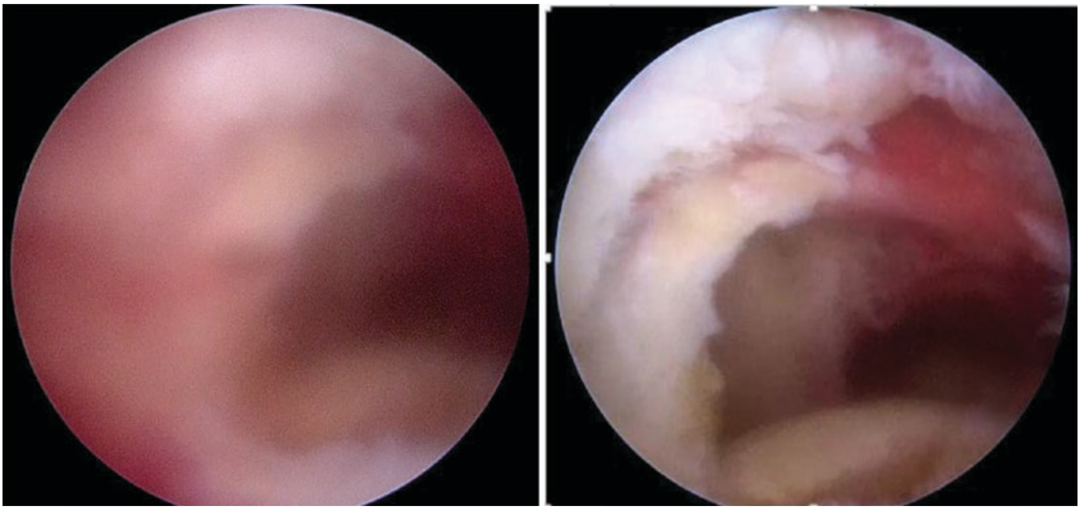


Fig. 5.11 (Left) Obscured vision during arthroscopic rotator cuff repair by turbulent flow. By closing the portals and eliminating outflow (right), the vision becomes clear

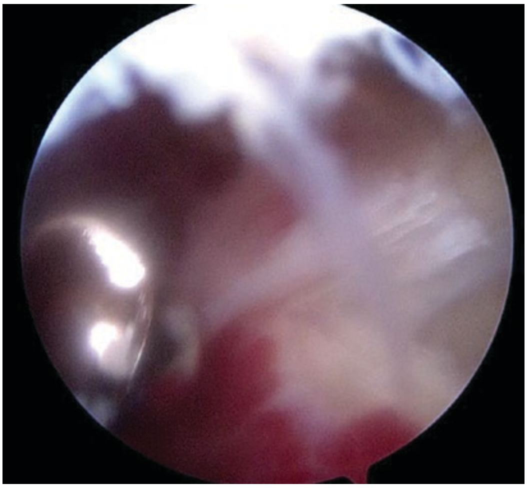


Fig. 5.12 Foresightful dissection: in inflammatory tissue, careful and slow preparation with a minimum of shaving is helpful

- Foresightful dissection technique [10]: depending on the case, careful and slow preparation of the tissue is mandatory. For example, it is clear that in case of highly inflammatory tissue reactions (bursitis, capsulitis), aggressive shaving of the tissue will lead to more bleeding and problems than in uninflamed tissues (Fig. 5.12). So in these cases, dissection with the radiofrequency (RF) probe is recommended. It is important to anticipate the risk of bleeding during tissue dissection and adapt the technique to the anatomic area and the degree of inflammation.

5.5 Anesthesia

Over the last few years, arthroscopic shoulder surgery has been increasingly performed by surgeons in a sitting position, thereby creating new challenges for anesthesiologists [11].

On the one hand, the surgeon's legitimate wish is for good visual conditions and thus low blood pressure. On the other hand, the anesthetist has to be aware of the redistribution of the blood volume due to gravity resulting from general anesthesia. This takes place at the normal measuring points of blood pressure measurement (NIBP) at the upper arm, which can therefore no longer be considered accurate.

The brain is a very ischemic-intolerant organ and particularly threatened by cerebral hypoperfusion. There are findings in literature of rare cases of cerebral damage, with partially fatal outcome because of permissive hypotonia and cerebral hypoperfusion [12].

Thus, we have developed so-called standard operating procedures (SOP) for shoulder arthroscopy, which enable safe and controlled hypotonic blood pressure values.

In a sitting position, an average vertical distance from the upper arm blood pressure cuff (NIBP) to the auditory canal or cranial base is 30–35 cm, resulting in a pressure difference of 20–25 mmHg. This difference will have to be subtracted from the measured NIBP values (Fig. 5.13).

It should be noted that the hydrostatic pressure gradient and a normally safe lower limit of 60 mmHg pressure (MAD) could lead to cerebral desaturation, while the conventional oxygen saturation (SaO_2) still denotes normal values at the finger clip [13].

The anesthesia often commences intravenously with propofol, opiate, and muscle relaxant, and then the patient is intubated and placed in an upright position.

A high incidence of 70–80% hypotonia below the known safe limits can be observed.

It has been shown that the hypnotics, such as propofol or sevoflurane alone, influence the redistribution phenomenon in a dose-dependent manner: in higher doses the usage of propofol has a characteristic "on-off effect" and may lead to increased awareness. At low doses, however, and used in combination with inhalational anesthetics, this characteristic is absent (Figs. 5.14 and 5.15).

Therefore, anesthesia continuation after introduction should be done with sevoflurane, which is associated with significantly less cerebral desaturation. Low doses of inhaled anesthetics disrupt cerebral autoregulation to a lesser extent [14].

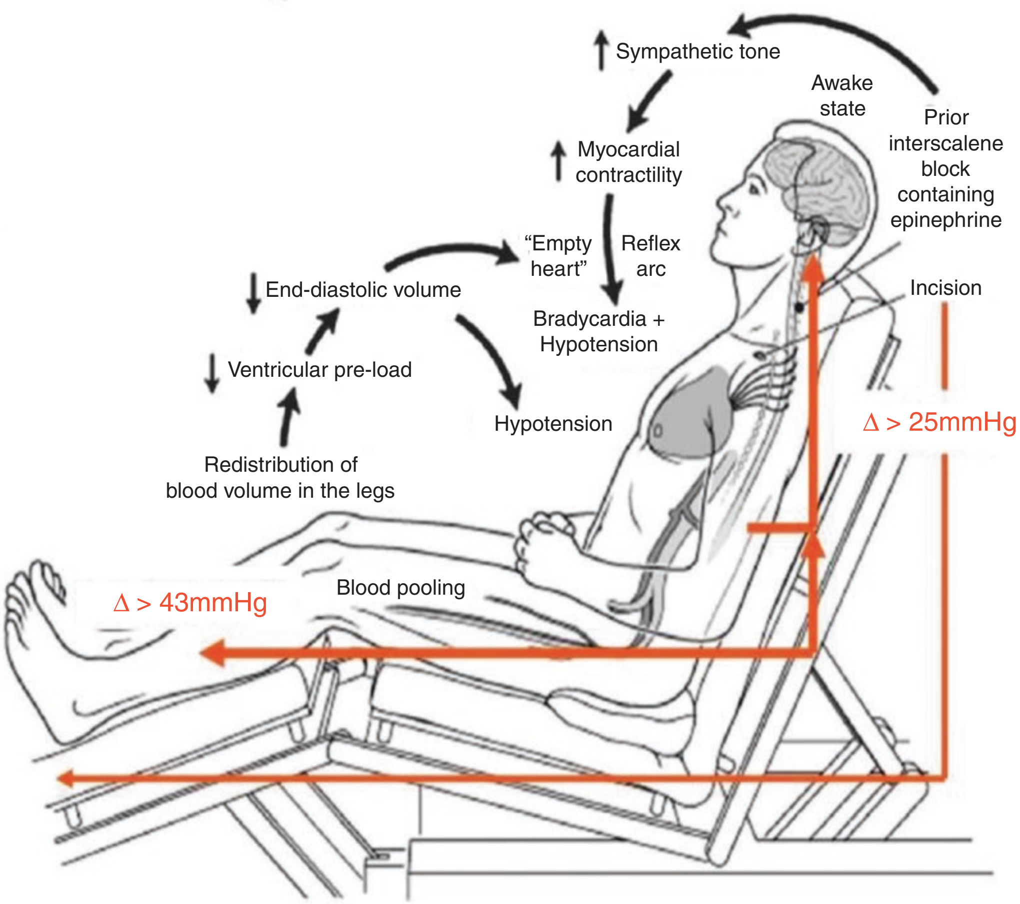


Fig. 5.13 Hydrostatic pressure difference (RR cuff at upper arm to cranial base) of 20–25 mmHg (30 cm H_2O = 22 mmHg) should be noted

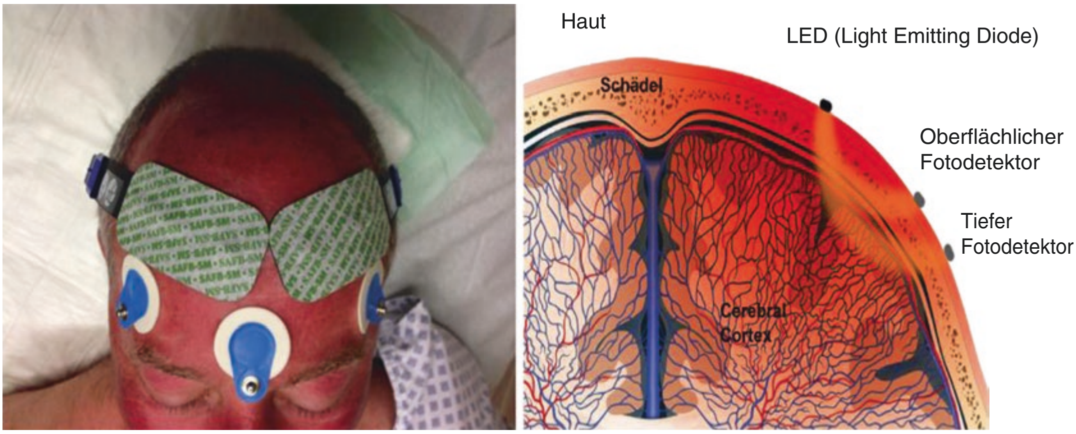


Fig. 5.14 Regulation of cerebral blood flow (CBF) as a function of inhalation anesthetics in different doses. In high doses, the autoregulation is abolished, and the CBF passively follows the perfusion pressure

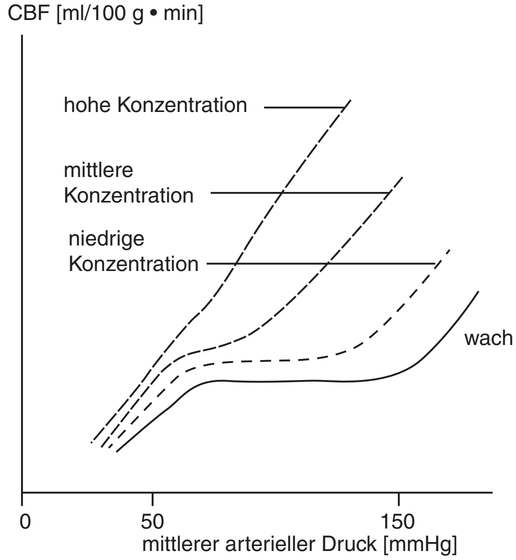


Fig. 5.15 Influence of MAP, CO_2, and O_2 on cerebral blood flow (CBF)

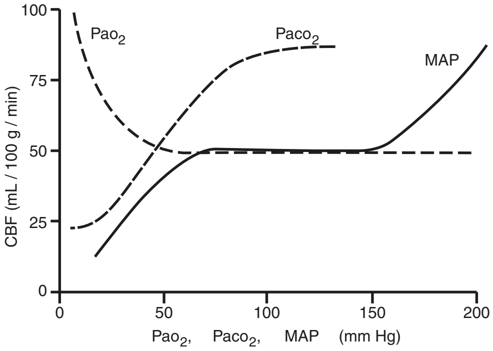


Fig. 5.16 Measurement of cerebral brain oxygenation (NIRS). (Left) *NIRS and Narcotrend electrodes*. (Right) NIRS function scheme

A moderate hypercapnia during ventilation denotes a notable effect and leads to a vasodilation with increase of the intracerebral blood volume and higher $rcSO_2$ values (regional cerebral oxygen saturation). Never hyperventilate with subsequent hypocapnia and cerebral hypoperfusion [15].

The cerebral blood flow (CBF) is affected by MAP, CO_2, and O_2. $rcSO_2$ using NIRS (near-infrared spectroscopy; Fig. 5.16) [16]. It should be measured before induction of the general anesthesia with the patient in sitting position which yields a baseline for each individual patient. Further the $rcSO_2$ should be continuously monitored throughout the whole surgical case so that critical drops of the saturation below the baseline can be early recognized and counteracted.

A so-called controlled hypotension (goal MAP of 60 mmHg) should be attained by utilizing all these measurements with adequate brain oxygen saturation being secured ($rcSO_2$ measurements) throughout the case (Fig. 5.17).

Fig. 5.17 Example of "ideal" conditions for shoulder arthroscopy using the setup mentioned in the text: systolic arterial pressure is very low; however NIRS indicated sufficient brain oxygenation at the same time

Checkbox with recommendations for anesthesia for shoulder arthroscopy:

Preop
Always regional anesthesia (interscalene block or catheter), decreasing pain and sympathotonus during the surgery [17]
Infusion bolus of approx. 500 mL crystalloids (electrolyte solution)
Sympathomimetics during anesthesia initiation (Akrinor® or ephedrine)
Antithrombosis prophylaxis with NMH the previous evening

Intra-op
If necessary sympathicomimetics during anesthesia
Always *low inhalational anesthesia* (*sevoflurane*) with end-tidal ca. 0.7% (age appropriate)
Moderately high end-expiratory CO_2 ($EtCO_2$) of 40–50 mmHg
Ventilation with 100% O_2
Cerebral oxygen saturation monitoring (NIRS)
NIBP interval 3 min for higher continuity, instead of arterial cannula

It has been shown that the consequent application of this neuroprotective procedure results in high safety as well as low hypotonic blood pressure.

5.6 Take-Home Message

Poor visualization continues to be a frustrating aspect in the field of arthroscopic shoulder surgery.

In this chapter we offer a concept (surgical equipment, patient positioning, surgical technique, anesthesia), which in our hands has tremendously helped to improve visualization by the decrease of intraoperative bleeding.

References

1. Noyes FR, Spievack ES. Extraarticular fluid dissection in tissues during arthroscopy: a report of clinical cases and a study of intraarticular and thigh pressures in cadavers. Am J Sports Med. 1982;10(6):346–51.
2. Funk RH, Wagner W, Rohen JW. The effect of epineph- rine on ciliary process vasculature and IOP studied by intraocular microendoscopy in the albino rabbit. Curr Eye Res. 1992;11:161–73.
3. Jensen KH, Werther K, Stryger V, Schultz K, Falkenberg B. Arthroscopic shoulder surgery with epinephrine saline irrigation. Arthroscopy. 2001;17:578–81.
4. Ampat G, Bruguera J, Copeland SA. Aquaflo pump vs FMS 4 pump for shoulder arthroscopic surgery. Ann R Coll Surg Engl. 1997;79:341–4.

5. Hsiao MS, Kusnezov N, Sie RN, Owens BD, Herzog JP. Use of an irrigation pump system in arthroscopic procedures. Orthopedics. 2016;39(3):e474–8.
6. Bomberg BC, Hurley PE, Clark CA, McLaughlin CS. Complications associated with the use of an infusion pump during knee arthroscopy. Arthroscopy. 1992;8(2):224–8.
7. Metcalf RW. Operative arthroscopy of the knee. Instr Course Lect. 1981;30:357–96.
8. van Montfoort DO, van Kampen PM, Huijsmans PE. Epinephrine diluted Saline irrigation fluid in arthroscopic shoulder surgery: a significant improvement of clarity of visual field and shortening of total operation time. A randomized controlled trial. Arthroscopy. 2016;32(3):436–44.
9. Morrison DS, Schaefer RK, Friedman RL. The relationship between subacromial pressure, blood pressure, and visual clarity during arthroscopic decompression. Arthroscopy. 1995;11:557–60.
10. Millstein ES, Snyder SJ. Arthroscopic management of partial, full-thickness, and complex rotator cuff tears: Indications, techniques, and complications. Arthroscopy. 2003;19(suppl 1):189–99.
11. Aguirre J, Borgeat A, Trachsel T, et al. Cerebral oxygenation in patients undergoing shoulder surgery at the beach chair position: comparing general to regional anesthesia and the impact on neurobehavioral outcome. Rev Esp Anestesiol Reanim. 2014;61:64–72.
12. Moerman AT, DeHert SG, Jacobs TF, et al. Cerebral oxygen desaturation. Eur J Anaesthesiol. 2012;29:82–7.
13. Pohl A, Cullen DJ. Cerebral ischemia during shoulder surgery in the upright position: a case series. J Clin Anesth. 2005;17:463–9.
14. Dagal A, Lam AM. Cerebral autoregulation and anesthesia. Curr Opin Anaesthesiol. 2009;22:547–52.
15. Murphy GS, Szokol JW, Avram MJ, et al. Effect of ventilation on cerebral oxygenation in patients undergoing surgery in the beach chair position: a randomized controlled trial. Br J Anaesth. 2014;113:618–27.
16. Jeong H, Jeong S, Lim HJ, et al. Cerebral oxygen saturation measured by near-infrared spectroscopy and jugular venous bulb oxygen saturation during arthroscopic shoulder surgery in beach chair position under sevoflurane-nitrous oxide or propofol-remifentanil anesthesia. Anesthesiology. 2012;116:1047–56.
17. Rohrbaugh M, Kentor ML, Orebaugh SL, et al. Outcomes of shoulder surgery in the sitting position with interscene nerve block: a single-center series. Reg Anesth Pain Med. 2013;38:28–33.

Fast Track in TKA Surgery: Where Are We Now?

6

Nanne P. Kort and Michael Clarius

6.1 Introduction

Care pathways in orthopedic surgery are designed to prepare and optimize patients before, during, and after surgery. As the total amount of arthroplasty procedures are increasing worldwide, these pathways are becoming more important. In the United States, 402.100 TKAs were performed in 2003, and the estimation is that by 2030 the annual demand will have grown by 673% to 3.48 million arthroplasties. To meet this high demand, proper clinical pathways are needed to provide safe and efficient arthroplasty procedures. The concept of clinical pathways has evolved over the past decades. So-called enhanced recovery pathways, or fast-track surgery pathways, are on the rise worldwide with successful results in terms of (serious or severe) adverse events, readmission rate, functional recovery, patient reported outcome measures, length of hospital stay, and costs.

A successful implementation of these clinical pathways requires the development of a selected number of scientifically supported procedures (e.g., multimodal pain protocol, early mobilization, prevention of blood loss, and postoperative nausea/vomiting, optimized logistic process), supported by the complete multidisciplinary team (e.g., anesthesiologist, physiotherapist, nurses, hospital managers). Within these pathways, evidence-based protocols are designed, evaluated, and recreated based on latest literature and experiences. The ongoing development of these pathways resulted in day-care surgery for a selected group of patients in the last years, in which patients were admitted and discharged on the day of surgery with no overnight stay. Expansion to a day-care surgery pathway involves an extensive change in mind-set, both for the surgeon as part of the multidisciplinary team and for the patients. The implementation process consists more than just the introduction and the use of new protocols itself. All the stakeholders involved in the day-care/fast-track surgery pathway need to understand and support the procedure. A stepwise implementation, starting with a fast-track surgery pathway with the aim to discharge patients on the day of surgery, will be more effective and safe toward the day-care procedure. Although evidence-based patient selection criteria are lacking, current literature shows acceptable clinical results in selected patients (e.g., low ASA score, primary arthroplasty, younger age, less comorbidities). Combining the high demand of arthroplasties in the future, with a correlated increase in economic burden on the public health system, an adequate and evidence-based clinical pathway is needed to

---

N.P. Kort (✉)
ACIBADEM International Medical Center,
Amsterdam, The Netherlands
e-mail: nanne@nannekort.com

M. Clarius
Vulpius Klinik GmbH,
Bad Rappenau, Germany

G.M.M.J. Kerkhoffs et al. (eds.), *ESSKA Instructional Course Lecture Book*,
https://doi.org/10.1007/978-3-662-56127-0_6

provide a safe and efficient knee arthroplasty procedure in terms of adverse events, readmissions, patient reported outcome measures, and costs.

In this chapter, the rise of fast-track surgery pathways will be discussed from a broad perspective with practical recommendations.

6.2 State-of-the-Art Treatment

6.2.1 Patient Demographics: Are All Patients Eligible for Fast-Track Surgery?

It is known that comorbidities can predispose patients to increased AEs, readmission rates, and increased length of hospital stay. So, when the orthopedic surgeon is intending arthroplasty surgery, preoperative patient selection seems to be the first crucial step in the prevention of AEs and readmissions in general.

When comparing an optimized pathway with a standard care pathway after primary arthroplasty in an unselected group of patients, fewer adverse events are reported. In particular, myocardial infarction, stroke, and death rates were lower in fast-track surgery pathways. This approach typically includes opioid-sparing anesthesia, local infiltration analgesia, and day of surgery mobilization (few hours after surgery). Therefore, all patients are eligible for the fast-track surgery pathway. As a matter of fact, patients with high comorbidity levels should benefit most from the fast-track surgery pathways as seen in nonelective surgery cases (e.g., femur fractures).

6.2.2 Fast Track: The Organization Aspect and Reducing Length of Stay

Several aspects of organizational origin should be taken into account when implementing a fast-track surgery pathway. Various studies showed that optimization of the existing organizational structure will decrease length of hospital stay after arthroplasty. In the first place, the ward should be optimized to an arthroplasty-specific environment. The involved multidisciplinary team needs to be trained and experienced in the care for arthroplasty patients. Furthermore, all resources should be available to reduce any delay in discharge from the hospital. Patient expectation and information is another important factor in optimizing the length of hospital stay. For example, preoperative home preparation has to be done to prevent a delay in discharge. Functional discharge criteria need to be implemented, which patients need to know to be motivated and to have a participating role in their discharge.

6.2.3 Fast Track: Getting Rid of Arthroplasty Traditions

Many traditions in orthopedic surgery exist, such as drain and bladder catheter use, adhesive drapes, continuous passive motion machines, tourniquet use, and flexion as discharge criteria, for example. Since there is less, or in some cases none, evidence for these principles and protocols, they should be omitted from the standard care for primary arthroplasty patients. The fast-track pathway is characterized by its evidence-based approach, which is an ongoing process. Protocols need therefore be reexamined and improved when new insights are available.

6.3 Future Treatment Options

6.3.1 Outpatient Joint Arthroplasty: A Bridge Too Far?

Outpatient joint arthroplasty pathways are on the rise all over the globe. It obtained increased interest from orthopedic surgeons with varying comments, in a still continuing discussion. Opponents of outpatient arthroplasty doubt the patients' safety after discharge from the hospital, where proponents claim that there are no differences between inpatients as long as there are proper selection criteria, evidence-based protocols and adaptation of the hospital, and logistic structures. These disagreements require top-class evidence to either confirm and support or disapprove the use of outpatient arthroplasty pathways. The first studies on outpatient arthroplasty are promising. Although these studies are of moderate quality, the first results are satisfying in terms of patient's safety and satisfaction. Further research, with randomized controlled data, is needed to draw final conclusions on the safety and efficacy of these pathways.

6.4 Take-Home Message

- Fast-track surgery is inevitable for every arthroplasty patient. Higher ASA classification and comorbidity levels mean more evidence to support fast-track surgery protocols.
- First better, then faster. Start with optimizing your arthroplasty pathway step by step before reducing the length of hospital stay. Quality before quantity.
- Teamwork. Involve the complete multidisciplinary team.
- If possible, perform outpatient joint arthroplasty on the younger and healthiest patients.

References

1. Argenson JN, Husted H, Lombardi A Jr, Booth RE, Thienpont E. Global forum: an international perspective on outpatient surgical procedures for adult hip and knee reconstruction. J Bone Joint Surg Am. 2016;98(13):e55. Review
2. Aynardi M, Post Z, Ong A, Orozco F, Sukin DC. Outpatient surgery as a means of cost reduction in total hip arthroplasty: a case-control study. HSS J. 2014;10(3):252–5.
3. Berger RA, Kusuma SK, Sanders SA, Thill ES, Sporer SM. The feasibility and perioperative complications of outpatient knee arthroplasty. Clin Orthop Relat Res. 2009;467(6):1443–9.
4. Berger RA, Sanders S, Gerlinger T, Della Valle C, Jacobs JJ, Rosenberg AG. Outpatient total knee arthroplasty with a minimally invasive technique. J Arthroplast. 2005;20:33–8.
5. Berger RA, Sanders SA, Thill ES, Sporer SM, Della Valle C. Newer anesthesia and rehabilitation protocols enable outpatient hip replacement in selected patients. Clin Orthop Relat Res. 2009;467(6):1424–30.
6. Chen D, Berger RA. Outpatient minimally invasive total hip arthroplasty via a modified Watson-Jones approach: technique and results. Instr Course Lect. 2013;62:229–36.
7. Cross MB, Berger R. Feasibility and safety of performing outpatient unicompartmental knee arthroplasty. Int Orthop. 2014;38(2):443–7.
8. Dervin GF, Madden SM, Crawford-Newton BA, Lane AT, Evans HC. Outpatient unicompartmental knee arthroplasty with indwelling femoral nerve catheter. J Arthroplast. 2012;27(6):1159–65.
9. Dorr LD, Thomas DJ, Zhu J, Dastane M, Chao L, Long WT. Outpatient total hip arthroplasty. J Arthroplast. 2010;25(4):501–6.
10. Gondusky JS, Choi L, Khalaf N, Patel J, Barnett S, Gorab R. Day of surgery discharge after unicompartmental knee arthroplasty: an effective perioperative pathway. J Arthroplast. 2014;29(3):516–9.
11. Goyal N, Chen AF, Padgett SE, Tan TL, et al. A multicenter, randomized study of outpatient versus inpatient total hip arthroplasty. Clin Orthop Relat Res. 2016;475(2):364–72.
12. den Hartog YM, Mathijssen NM, Vehmeijer SB. Total hip arthroplasty in an outpatient setting in 27 selected patients. Acta Orthop. 2015;86(6):667–70.
13. Husted H, Gromov K, Malchau H, Freiberg A, Gebuhr P, Troelsen A. Traditions and myths in hip and knee arthroplasty. Acta Orthop. 2014;85(6):548–55.
14. Husted H. Fast-track hip and knee arthroplasty: clinical and organizational aspects. Acta Orthop Suppl. 2012;83(346):1–39. Review
15. Khan SK, Malviya A, Muller SD, Carluke I, Partington PF, Emmerson KP, Reed MR. Reduced short-term complications and mortality following enhanced recovery primary hip and knee arthroplasty: results from 6000 consecutive procedures. Acta Orthop. 2014;85(1):26–31.
16. Kehlet H, Jørgensen CC. Rapid recovery after hip and knee arthroplasty—a transatlantic gap? J Arthroplast. 2015;30(12):2380.
17. Kolisek FR, McGrath MS, Jessup NM, Monesmith EA, Mont MA. Comparison of outpatient versus inpatient total knee arthroplasty. Clin Orthop Relat Res. 2009;467(6):1438–42.
18. Kort NP, Bemelmans YFL, Schotanus MGM. Outpatient surgery for unicompartmental knee arthroplasty is effective and safe. KSSTA. 2015. https://doi.org/10.1007/s00167-015-3680-y.
19. Kort NP, Bemelmans YFL, van der Kuy PH, Jansen J, Schotanus MGM. Patient selection criteria for outpatient joint arthroplasty. Knee Surg Sports Traumatol Arthrosc. 2016. https://doi.org/10.1007/s00167-016-4140-z.
20. Lovald ST, Ong KL, Lau EC, Joshi GP, Kurtz SM, Malkani AL. Patient selection in outpatient and short-stay total knee arthroplasty. J Surg Orthop Adv. 2014;23(1):2–8.
21. Lovald ST, Ong KL, Malkani AL, Lau EC, Schmier JK, Kurtz SM, Manley MT. Complications, mor-

tality, and costs for outpatient and short-stay total knee arthroplasty patients in comparison to standard-stay patients. J Arthroplast. 2014;29(3):510–5.

22. Malviya A, Martin K, Harper I, Muller SD, Emmerson KP, Partington PF, Reed MR. Enhanced recovery program for hip and knee replacement reduces death rate. Acta Orthop. 2011. https://doi.org/10.3109/17453674.2011.618911.
23. Schotanus MGM, Bemelmans YFL, Grimm B, Heyligers IC, Kort NP. Physical activity after outpatient surgery and enhanced recovery for total knee arthroplasty. Knee Surg Sports Traumatol Arthrosc. 2016. https://doi.org/10.1007/s00167-016-4256-1.

New Insights in Diagnosis and Treatment of Distal Biceps Pathology

7

Denise Eygendaal, Michel van den Bekerom,
Raul Barco, Paolo Arrigoni, Riccardo D'Ambrosi,
Davide Cucchi, Simone Nicoletti,
Pietro Simone Randelli, Kilian Wegmann,
and Lars Peter Müller

7.1 Introduction

7.1.1 Anatomical and Biomechanical Aspects of the Distal Biceps Tendon

Biceps brachii is composed of two separate heads and is innervated by a branch of the musculocutaneous nerve [1]. The proximal tendon of the long head is attached to the supraglenoid tubercle, and the proximal tendon of the short head is attached to the coracoid process. The biceps (muscle and tendon) rotates 90° externally from origin to insertion onto the bicipital tuberosity [2] and acts on three joints: the glenohumeral, ulnohumeral, and proximal radioulnar joints. A completely bifurcated distal tendon insertion is not uncommon [3, 4]. The short head of the distal biceps tendon was reported to insert more distally, and the long head was inserted more eccentric and medial. The moment arm of the long head was higher in supination, and the short head had a higher moment arm in neutral position and pronation [5]. These findings may allow functional independence and isolated rupture of each portion and may have consequences for restoring the native anatomy during a surgical repair. Several authors reported an isolated rupture of one of the two tendons in cases of bifurcated distal biceps tendons [4].

The blood supply for the proximal zone of the distal biceps tendon comes from the brachial artery by branches that extend across the musculotendinous junction. The distal zone has a separate blood supply by branches from the posterior interosseous recurrent artery. The middle zone receives vessels from both vessels but only through its paratenon cover [6]. The middle zone is considered as a transition area at which tendon repair mechanisms may be limited and is therefore more prone to injury and even rupture [6].

The lacertus fibrosus envelopes the forearm flexor muscles and serves as a stabilizer of the distal biceps tendon and particularly the short

---

D. Eygendaal (✉)
Department of Orthopedic Surgery, Amphia Hospital
and University of Amsterdam,
Amsterdam, The Netherlands
e-mail: denise@eygendaal.nl

M. van den Bekerom
Department of Orthopedic Surgery, OLVG,
Amsterdam, The Netherlands

R. Barco
Department of Orthopedic Surgery, University
Hospital La Paz, Madrid, Spain

P. Arrigoni • R. D'Ambrosi • D. Cucchi
S. Nicoletti • P.S. Randelli
Department of Orthopedic Surgery, Universtiy of
Milano, Milan, Italy

K. Wegmann • L.P. Müller
Department of Orthopedic Surgery, University of
Cologne, Cologne, Germany

G.M.M.J. Kerkhoffs et al. (eds.), *ESSKA Instructional Course Lecture Book*,
https://doi.org/10.1007/978-3-662-56127-0_7

head. As the forearm flexors contract, the lacertus is tensed, subsequently causing a medial pull on the biceps tendon and perhaps contributing to its rupture [7]. When intact, it may lessen the functional deficits and the need for surgical reconstruction of a distal biceps tendon tear in low-demand patients [8]. The need to preserve an intact lacertus fibrosus or even repair the lacertus at the time of surgery is very controversial [9]. A surgical technique, using the lacertus fibrosus, as a local graft source for chronic distal biceps tendon ruptures, to avoid harvest site morbidity was described [10].

The attachments of the two biceps brachii tendons are surrounded by the bicipitoradial bursa draped over the two tendons, lying between the brachialis muscle and the distal tendons when the elbow is extended and between the proximal radius and biceps tendon in pronated position [11]. This structure is of clinical importance during endoscopic evaluation of the distal tendon after injury in cases of uncertain rupture of the tendon [3]. Injection of the bursa under ultrasound control can be part of the conservative treatment regime.

7.1.2 Possible Complications

Posterior interosseous nerve injury is the most devastating neurologic complication of distal biceps tendon refixation [12, 13]. The posterior interosseous nerve pierces the supinator muscle in the proximal forearm and circumflexes the radius at approximately 1.0–1.5 cm distal to the center of the bicipital tuberosity [12]. The distance between the point where the posterior interosseous nerve crosses the radius and the radiocapitellar joint changes with forearm rotation: the mean distances are 4.2, 5.6, and 3.2 cm in neutral position, pronation, and supination, respectively [14]. The posterior interosseous nerve can be injured during the refixation of the tendon or become entrapped by scar tissue in more chronic tears [3, 12]. Dissection of the bicipital tuberosity should be performed with a supinated forearm, and caution is required when placing deep retractors around the tuberosity with the single-incision approach. It is advised not to use a Hohmann retractor at the lateral side of the tuberosity [15].

When approaching the bicipital tuberosity, dissection through the subcutaneous tissue requires caution in order to protect the lateral antebrachial cutaneous nerve. This is a sensory terminal branch of the musculocutaneous nerve. In chronic distal biceps ruptures with tendon retraction, the lateral antebrachial cutaneous and the posterior interosseous nerve can become entrapped within reactive inflammatory and scar tissue as it passes between the biceps and brachialis muscles [16]. Care must be taken to avoid forceful use of retractors nearby the lateral antebrachial cutaneous nerve and to protect the nerve at the time of refixation of the tendon to avoid iatrogenic entrapment by the repaired tendon or sutures.

7.2 State-of-the-Art Treatment

7.2.1 Partial Tears of the Distal Biceps: Workup and Treatment

Partial tears are rare injuries, occurring mostly in middle-aged men. While most of the pathology of the distal biceps is related to complete ruptures, partial tears or bursitis at the insertion site may present with mild pain in the antecubital fossa, so patients' diagnosis may be delayed. A high index of suspicion is needed in order to perform a timely diagnosis. The present paragraph reviews current aspects of diagnosis and treatment strategies. Patients presenting with pain at the antecubital fossa typically present with biceps tendinopathy. Other causes of pain include intra-articular problems such as rheumatoid arthritis, osteoarthritis, anterior capsular strain, loose body, or pronator syndrome, but these are rare. On clinical examination, patients usually show a full range of motion in both flexion-extension and pronation-supination but may show a very slight decrease in terminal extension with supination due to pain secondary to tendinopathy. The hook test is a very useful test to assess the integrity of the distal biceps. The patient is asked to bilaterally flex the shoulder to head level and to flex the elbow approximately to 90° while maintaining the forearm in supination. The index finger is then used to "hook" the distal biceps tendon [17].

Radiographs of the elbow are typically normal. Ultrasound (US) is accurate to diagnose complete tendon ruptures, but its role in diagnosing partial ruptures is less clear [18]. MRI may reveal tendinopathic changes or the presence of bicipitoradial bursitis. These changes may be best seen using the FABS (flexion-abduction-supination) view in which the patient is placed prone with the affected arm in flexion, abduction, and supination [19]. Of note, it may be difficult to distinguish between tendinosis and partial tears involving less than 50% of the tendon. Findings related to the presence of a partial tear include increased signal intensity in the distal biceps, the presence of peri-tendinous or intra-sheath fluid, and increased bone marrow signal at the tendon insertion site. A trial of 6 months of conservative management seems reasonable. High-grade tears with greater than 50% of the attachment site have more failures after conservative treatment.

Partial tears are typically a delayed diagnosis so patients may have tried different treatments at the time of consultation. A trial of 6 months of conservative management seems reasonable. Conservative management has not been clearly protocolized, and most authors use physical therapy, the cessation of aggravating activities (including splinting), NSAIDs, and the use of steroid/anesthetic injections. Progressive strengthening is recommended until patients can perform their desired activities. While this form of treatment can be useful in some patients, a recent systematic review of surgical outcomes of partial ruptures showed that only in 5 of 65 patients documented to have received conservative management; this form of treatment was effective [20]. High-grade tears with greater than 50% of the attachment site have more failures after conservative management, and some patients could benefit from early surgical repair.

7.2.1.1 Endoscopic Techniques

The use of endoscopy to treat distal biceps injuries has been recently advocated [21].

The use of endoscopy to treat distal biceps injuries has been recently reported using different techniques [3, 6]. The endoscopy can be utilized as a diagnostic aid in defining the extent of the rupture, for removing adjacent bursitis, to debride the partial biceps tear, or to complete and reattach the tendon. It is a complex technique and should be reserved for experienced arthroscopists.

The patient is placed supine, with the arm on an arm table. A tourniquet is helpful for visualization, and in partial ruptures the risk of not reaching the attachment site is nonexistent. The tendon can be palpated and it is usually central on the forearm. The incision can be made 3–4 cm distal to the elbow crease. Blunt dissection is carried out until the tendon is apparent. Injuries to the lateral antebrachial cutaneous nerve (LABCN) and the posterior interosseous nerve (PIN) are frequent complications. To decrease the rate of these complications, we recommend handheld retractors and avoid Hohmann retractors around the radial neck and tuberosity. Dissection of the LABCN is discretional to the surgeon, but not dissecting it may as well protect it further than dissecting it.

The scope is advanced to the bicipital tuberosity and the forearm is supinated to improve the working space. The medial fibers are usually intact in cases of a genuine partial tear. The distal short head of the biceps can be ruptured with preservation of the proximal long head of biceps insertion, and ganglions at the site of rupture are frequently seen [16].

Vandenberghe et al. suggest the following protocol to decide appropriate treatment of distal biceps tears [21]. Tears smaller than 25% are debrided; between 25 and 50%, they are partially repaired with the use of an anchor, and those greater than 50% are detached and fixed using a cortical bone technique. In the latter, the scope can be used to localize the proper insertion site, and, while removing the scope, the sheath can provide protection for the drills used for cortical preparation.

A guide wire is drilled in the center of the tuberosity through both cortices and must be directed straight posteriorly or with slight ulnar deviation. The guide wire is over-drilled with a bigger cannulated drill in the first cortex and a smaller drill on the second cortex (different systems may have different sizes). The guide wire may have trailing sutures, and it can be advanced through the posterior forearm to introduce the button and tendon into the drill site until the button has passed the second cortex. It can then be flipped by flexing and extending the elbow. Alternatively, an antegrade sliding technique can be used. In this technique, the sutures from the grasped tendon are passed through a button, which is advanced in an antegrade fashion with the use of a handle until the button has passed the second cortex. It is then deployed from the handle and toggling with the suture achieves flipping of the button. Sliding and tensioning of the limbs of the suture advance the tendon to the desired position. The sutures are then tied, and the position is locked. Otherwise, an interference screw can be used to secure the tendon and offset it to its lateral position in the radial tuberosity.

The patient is placed in supine position with the arm on an arm table. The tendon can be palpated and it is usually central on the forearm. The incision can be made 3–4 cm distal to the elbow crease. The scope is advanced to the bicipital tuberosity and the forearm is supinated to improve the working space.

As an alternative to the single anterior incision, a single posterior incision or a double incision can be used (see below).

7.2.2 Single- or Double-Incision Technique

The first surgical technique involved an anterior approach ("Henry's") with a single curvilinear incision centered on the antecubital fossa; in this approach, the radial nerve and the posterior interosseous nerve are at risk of injury. To avoid this risk, Boyd and Anderson developed a technique that included a double incision and an interosseous access to reinsert the biceps to the radius through a bone tunnel [16]. Kelly further modified the second access with a posterior approach through the muscle, dissociating the fibers of the extensor carpi ulnaris. The preparation of the radius, however, seems to increase the risk of postoperative calcification and radioulnar synostosis.

A subsequent modification of the single anterior approach was based on suture anchors in a narrow space bounded by the brachioradialis and the pronator teres. Suspensory cortical fixation with buttons demonstrated optimal mechanical properties. However, radial preparation is required, with major risks of calcification and synostosis. Moreover, once the button has passed the second cortex, it lays very close to the posterior interosseous nerve.

Single anterior incision techniques are associated with higher risk of nerve damage, while dual access or techniques that require radial preparation may lead to a greater risk of calcification and synostosis. The anterior approach may also be performed with a small transverse median incision, which could reduce the risk of nerve damage [13].

7.2.2.1 Fixation Techniques

Four different fixation methods are currently used:

- Intramedullary fixation with transosseous suture
- Tenodesis with interference screw
- Anchor suture
- Mono- or bicortical fixation with button

Simple elbow flexion to 90° generates a force of 90N at the tendon [14]. Tendon rupture occurs with a force of 204N. A technique based on suture anchors can be performed using a single anterior approach and keeping the forearm in supination: the tendon is reinserted with one or two anchors or with an interosseous screw after preparing the tuberosity. The perforation of the posterior cortex is thereby avoided. Gasparella reported good results in 14 patients at a mean follow-up of 26 months using two anchors. A deficit in supination [22] was found in two cases.

Transosseous fixation techniques with a cortical suspensory button were also developed. These techniques are based on the preparation of the radial tuberosity and the creation of a slot for the biceps tendon with a 4 mm hole [13]. A radiological control of the correct positioning of the button is mandatory. The suture fixation with cortical suspensory button turned out to be the most tolerant to the load: the fixation with a standard button has a breaking load of 270N, while screw anchors do not resist more than 57N. Mazzocca studied the cyclic load breaking, highlighting that the EndoButton technique presents a significantly higher load: 440N against 381N of suture anchors, 310N of the bone tunnel, and 232N of the interference screws [23]. Mazzocca also studied the cyclic mobilization of the different fixation techniques with inverse results: the interference screw presents minimal mobilization. These movements may delay or inhibit the healing process. In order to improve the fixation, other techniques have also been proposed that associate an interference screw fixation with a cortical suspensory button. In these cases, a bone tunnel of 8 mm is necessary [13]. None of these techniques are free from risk of complications related to access and posterior interosseous nerve protection.

7.2.2.2 Intramedullary Repair with Cortical Button

The patient is placed in supine position with an arm lying on a table with the tourniquet at the root of the arm. An anterior approach is created, with an oblique or longitudinal incision of approximately 6–8 cm in the middle of the forearm, about 2 cm distal to the elbow crease. This procedure is performed by blunt dissection under the skin, in order to avoid injury to the lateral antebrachial cutaneous nerve, which is retracted laterally. We proceed by separating the interval between the pronator teres muscle and brachioradialis, where it is common to see veins of large diameter that flow into the basilic and cephalic vein. In addition to these vessels, the radial artery is often present and should be protected. If the tendon is retracted, it can be gently mobilized to free it from post-traumatic adhesions. The degenerated distal portion is cut to about 0.5 cm. The tendon is then prepared with two ultrahigh molecular weight polyethylene Krackow sutures for 3 cm. The distal centimeter of the tendon is not prepared, but it is left as a shortening zone with the wires passing inside in a straight line (“sliding zones”). This will allow the tendon to be shortened and to lay the button easily on the cortex, which is secured with both sutures at about 2–3 mm from the tendon. Once the tendon is prepared, the peritenon of the biceps is opened up to the radial tuberosity. Serous fluid and hematoma may come out of the sheath. Once the tuberosity is prepared to bone, the forearm of the patient is supinated, and a slotted 1.5 mm wire is inserted at a 45° angle to the level of the tuberosity; this wire serves as a guide for dedicated cannulated cutters. A 4 mm bicortical tunnel (tunnel dimensions may vary depending on the device) is drilled first, followed by a proximal-to-distal 7–9 mm 1.5-cm-long half-tunnel (half-tunnel dimensions may vary according to the diameter of the tendon). A high-speed cutter or a Citelli can be used to broaden the entrance hole of the tendon proximally to an ellipsoidal shape to avoid conflicts or kinking of the repaired tendon. In this phase, it is essential to remove bone fragments with the suction to prevent heterotopic ossification. At this point, the traction sutures of the cortical button are inserted in the slot of the guide wire, which, once it crosses the soft tissues, comes out from the dorsal surface of the forearm and carries the sutures. The elbow is flexed to 100° and the suture is pulled with a more robust wire (traction suture). Once the cortical button passes the second cortex, the cortical button is flipped. With the image intensifier, it is mandatory to check the correct position. At this point, the elbow is completely extended to control the resistance. At the end of the surgical procedure, it is important to verify the correct tension on the tendon, which passes through the center of the surgical access. The elbow is immobilized at 90° for pain relief.

A recent systematic review identified all articles reporting distal biceps ruptures to compare outcomes between single- and double-incision techniques. In a total of 87 articles, lateral antebrachial cutaneous nerve neurapraxia was the most common complication in the single-incision group, occurring in 77 of 785 cases (9.8%). Heterotopic ossification was the most common complication in the double-incision group, occurring in 36 of 498 cases (7.2%). Posterior interosseous nerve palsy occurred in 2.7% (13/785) of single-incision procedures versus 0.2% (1/498) in the double-incision group. When combining heterotopic ossification and synostosis rates, the double-incision group demonstrated complications in 9.8% (47/498) of cases versus 3.2% (25/785) for single-incision cases. Additional complications in the single-incision group included superficial wound infection (11/785), nerve paresthesia (22/785), nerve dysesthesia (5/785), median nerve palsy (1/785), and other complications ranging from screw fractures to persistent elbow pain (49/785). In the double-incision group, additional complications included superficial wound infection (5/498), nerve paresthesia (2/498), nerve dysesthesia (3/498), posterior interosseous nerve palsy (1/498), ulnar nerve palsy (1/498), and other complications ranging from sterile stitch abscesses to lateral antebrachial cutaneous neuritis (30/498) [24].

7.3 Future Treatment Directions

More research is needed to assess whether or not separate reconstruction of the two bundles is better than single-strand reconstruction as described in this chapter. Handling of the tuberositas remains an interesting topic for the future. Reduction of the native bone of the tuberositas results in less tensioning of the biceps, whereas a pathologically thickened tuberositas may rub against the reinserted tendon and might be related to re-rupture of the biceps.

7.3.1 Rehabilitation After Distal Biceps Tendon Repair

A tear of the distal biceps tendon of its insertion at the radial tuberosity is a common soft tissue injury. With improved, stable surgical refixation techniques and the experiences of decreased rotation and flexion strength after conservative treatment, operative therapy is warranted. A whole variety of different surgical fixation techniques are available, with most of them being backed up by biomechanical evidence for sufficient primary stability of the construct. The most common technique now is the suture button fixation, which fixes the tendon on the tuberosity by a monocortical suture button. However, the postoperative treatment protocols vary significantly as there is few data available on their efficiency. The postoperative protocol should aim for protection of the repair by de-tensioning of the tendon. Usually, this is achieved by an immediate postoperative splint in flexion of at least 70°, followed by an orthosis providing an extension block. Forearm rotation also influences the tension of the distal biceps tendon, as the tendon wraps around the tuberosity in pronation and becomes tensed. In supination, the tendon unwinds off the proximal radius and thereby slackens. Hence, it is reasonable to place the forearm not only in flexion but also in supination.

The protocols also vary with respect to the administered time schedule. More cautious protocols advocate an extension block for 6 weeks, starting for 2 weeks in 90° flexion, followed by 2 weeks in 60°. After another 2 weeks of an extension limit of 30°, progressive range of motion is started. Full weight bearing should not be reached before 2 months. Heavy lifting and contact sports are allowed 6 months after the repair.

Another aspect of rehabilitation is the prevention of heterotopic ossification or radioulnar synostosis, which has been reported after distal biceps repair. The etiology of the ossification is not fully understood. It is unclear whether the amount of postoperative movement correlates with the development of heterotopic ossification. Even though there is only low-quality data on its use, the oral application of indometacine is part of many postoperative protocols. In a recent study, Costopoulos et al. reported a low percentage of less than 1%, after the administration of 75 mg of indometacine per day for a period of 10–42 days [25]. Prospective studies with conclusive study protocols are still missing.

7.4 Take Home Message

The short head of the distal biceps tendon inserts more distally and the long head inserts more medially. The moment arm of the long head is higher in supination, and the short head has a higher moment arm in neutral position and pronation. These findings may allow functional independence and isolated rupture of each portion and may have consequences for restoring the native anatomy during a surgical repair.

While most of the pathology of the distal biceps is related to complete ruptures, partial tears or bursitis at the insertion site may present with mild pain in the antecubital fossa so patients' diagnosis may be delayed.

Distal biceps tendon repair is a safe, replicable technique that offers optimal clinical results. Both the single- and double-incision techniques are safe and offer good clinical results. Patients gain full recovery of elbow articulation, strength, and resistance, with very low risk of complications. Endoscopic techniques could improve visualization, optimize the repair process, and reduce potential complications.

References

1. Pacha Vicente D, Forcada Calvet P, Carrera Burgaya A, Llusá Pérez M. Innervation of biceps brachii and brachialis: anatomical and surgical approach. Clin Anat. 2005;18:186–94.
2. Athwal GS, Steinmann SP, Rispoli DM. The distal biceps tendon: footprint and relevant clinical anatomy. J Hand Surg. 2007;32:1225–9.
3. van den Bekerom MP, Kodde IF, Aster A, Bleys RL, Eygendaal D. Clinical relevance of distal biceps insertional and footprint anatomy. Knee Surg Sports Traumatol Arthrosc. 2016;24(7):2300–7.
4. Koulouris G, Malone W, Omar IM, Gopez AG, Wright W, Kavanagh EC. Bifid insertion of the distal biceps tendon with isolated rupture: magnetic resonance findings. J Shoulder Elb Surg. 2009;18(6):e22–5.
5. Jarrett CD, Weir DM, Stuffmann ES, Jain S, Miller MC, Schmidt CC. Anatomic and biomechanical analysis of the short and long head components of the distal biceps tendon. J Shoulder Elb Surg. 2012;21(7):942–8.
6. Seiler JG, Parker LM, Chamberland PD, Sherbourne GM, Carpenter WA. The distal biceps tendon. Two potential mechanisms involved in its rupture: arterial supply and mechanical impingement. J Shoulder Elb Surg. 1995;4(3):149–56.
7. Miyamoto RG, Elser F, Millett PJ. Distal biceps tendon injuries. J Bone Joint Surg Am. 2010;92(11):2128–38.
8. Sutton KM, Dodds SD, Ahmad CS, Sethi PM. Surgical treatment of distal biceps rupture. J Am Acad Orthop Surg. 2010;18:139–48.
9. Landa J, Bhandari S, Strauss EJ, Walker PS, Meislin RJ. The effect of repair of the lacertus fibrosus on distal biceps tendon repairs: a biomechanical, functional, and anatomic study. Am J Sports Med. 2009;37:120–3.
10. Hamer MJ, Caputo AE. Operative treatment of chronic distal biceps tendon ruptures. Sports Med Arthrosc. 2008;16(3):143–7.
11. Eames MH, Bain GI, Fogg QA, van Riet RP. Distal biceps tendon anatomy: a cadaveric study. J Bone Joint Surg Am. 2007;89:1044–9.
12. Cohen MS. Complications of distal biceps tendon repairs. Sports Med Arthrosc. 2008;16(3):148–53.
13. Kodde IF, Baerveldt RC, Mulder PG, Eygendaal D, van den Bekerom MP. Refixation techniques and approaches for distal biceps tendon ruptures: a systematic review of clinical studies. J Shoulder Elb Surg. 2016;25(2):e29–37.
14. Calfee RP, Wilson JM, Wong AH. Variations in the anatomic relations of the posterior interosseous nerve associated with proximal forearm trauma. J Bone Joint Surg. 2011;93-A:81–90.
15. van Rheenen TA, van den Bekerom MP, Eygendaal D. The incidence of neurologic complications and associated risk factors in elbow surgery: an analysis of 2759 cases. J Shoulder Elb Surg. 2015;24(12):1991–7.
16. Ruch DS, Watters TS, Wartinbee DA, Richard MJ, Leversedge FJ, Mithani SK. Anatomic findings and complications after surgical treatment of chronic, partial distal biceps tendon tears: a case cohort comparison study. J Hand Surg. 2014;39(8):1572–7.
17. O'Driscoll SW, Goncalves LB, Dietz P. The hook test for distal biceps tendon avulsion. Am J Sports Med. 2007;35(11):1865–9.
18. Lobo Lda G, Fessell DP, Miller BS, Kelly A, Lee JY, Brandon C, et al. The role of sonography in differentiating full versus partial distal biceps tendon tears: correlation with surgical findings. Am J Roentgenol. 2013;200(1):158–62.
19. Festa A, Mulieri PJ, Newman JS, Spitz DJ, Leslie BM. Effectiveness of magnetic resonance imaging in detecting partial and complete distal biceps tendon rupture. J Hand Surg. 2010;35(1):77–83.
20. Behun MA, Geeslin AG, O'Hagan EC, King JC. Partial tears of the distal biceps brachii tendon: a systematic review of surgical outcomes. J Hand Surg. 2016;41(7):e175–89.

21. Vandenberghe M, van Riet R. Distal biceps ruptures: open and endoscopic techniques. Curr Rev Musculoskelet Med. 2016;9(2):215–23.
22. Gasparella A, Katusic D, Perissinotto A, Miti A. Repair of distal biceps tendon acute ruptures with two suture anchors and anterior mini-open single incision technique: clinical follow-up and isokinetic evaluation. Musculoskelet Surg. 2015;99(1): 19–25.
23. Mazzocca AD, Burton KJ, Romeo AA, Santangelo S, Adams DA, Arciero RA. Biomechanical evaluation of 4 techniques of distal biceps brachii tendon repair. Am J Sports Med. 2007;35(2):252–8.
24. Amin NH, Volpi A, Lynch TS, Patel RM, Cerynik DL, Schickendantz MS, Jones MH. Complications of distal biceps tendon repair: a meta-analysis of single-incision versus double-incision surgical technique. Orthop J Sports Med. 2016;4(10):2325967116668137.
25. Bhatia DN. Endoscopic distal biceps repair: endoscopic anatomy and dual-anchor repair using a proximal anterolateral “parabiceps portal”. Arthrosc Tech. 2015;4(6):e785–93.

8

Osteotomies: The Surgical Details You Want to Know

R.J. van Heerwaarden, S. Schröter,
Raghbir Singh Khakha, A. Wilson, D. Pape,
and Ph. Lobenhoffer

8.1 Surgical Planning

Surgical planning for osteotomies was first described as a comprehensive method of preparation for osteotomy surgery in a book on osteotomies for posttraumatic deformities [1] and in ESSKA's newsletter [2], both published in 2008. It was stated that a thorough history and careful physical examination may be more important factors than arbitrary radiographic examinations when advising patients as to the potential benefit of a corrective osteotomy for a lower limb deformity. In addition, surgery may not lead to a functional improvement of limb function without a thorough planning of deformity correction including choice of hardware for fixation and recognition of potential soft tissue problems during the procedure. Therefore, the surgeon treating limb deformities needs to have a surgical plan in which all of these factors are included before deformity correction is considered. The factors that are part of the surgical plan are displayed in Table 8.1 and will be described in more detail.

8.1.1 Physical Examination

The examination of all joints of the lower extremity remains important, even though a deformity may present itself only at one segment of the extremity. Limited function or ligamentous laxity of the hip, knee, patellofemoral, ankle, and subtalar joints has to be included in any preoperative planning which cannot be based on radiographs alone. Furthermore, range of motion should always be measured and the amount of excess or loss of motion documented to be able to correlate this with bone deformities found on radiologic examination.

It is important to distinguish between soft tissue and bone deformity, or a combination of both, as the cause for an abnormal range of motion. In the presence of symmetric (normal) legs, the pelvis is horizontal, the patellae are in the frontal plane of the knee, and the medial

---

R.J. van Heerwaarden (✉)
Centre for Deformity Correction and Joint Preserving Surgery, Kliniek ViaSana, Mill, The Netherlands
e-mail: vanheerwaarden@yahoo.com

S. Schröter
Department of Orthopedics and Reconstructive Surgery, University of Tübingen, Tübingen, Germany

R.S. Khakha • A. Wilson
University of Winchester and Basingstoke and North Hampshire Hospital, Basingstoke, UK

D. Pape
Centre Hospitalier de Luxembourg, Clinique d'Eich Akademisches Lehrkrankenhaus der Universität des Saarlandes, Luxembourg, Luxemburg

Ph. Lobenhoffer
Gelenkchirurgie Orthopädie Hannover, Hannover, Germany

G.M.M.J. Kerkhoffs et al. (eds.), *ESSKA Instructional Course Lecture Book*,
https://doi.org/10.1007/978-3-662-56127-0_8

Table 8.1 Schematic overview of a surgical plan for deformity correction of the lower limb

1.	Physical examination
2.	Radiological deformity analysis
	Frontal and sagittal plane
	(a) Weight-bearing line
	(b) Mechanical axis of femur and tibia
	(c) Joint orientation angles
	(d) Location of deformity (CORA)
	Transversal plane
	(a) CT limb rotation measurements
	(b) Patellar tracking analysis
	Multiplane deformities
3.	Correlation of physical examination and radiological deformity analysis
4.	Definition of deformity and aim of correction
5.	Planning of correction
6.	Hardware selection
7.	Description of surgical tactic

condyles of the femur and the medial malleoli are not separated more than 1–2 cm. The ankle is externally rotated relative to the knee, and the hind foot is in a slight valgus position.

Deformities in the sagittal plane can be recognized by clinical examination at the level of the hip, knee, and ankle (e.g., flexion contractures of the hip and knee, genu recurvatum, pes equinus). Rotation deformities can best be evaluated using the rotational profile according to Stahelin [3].

8.1.2 Radiological Deformity Analysis

The anatomical and mechanical axes of the lower extremities are assessed using a bilateral long-leg weight-bearing view and lateral radiographs of the affected limb. Attention to detail is essential when making the weight-bearing long-leg views to ensure that both knees are extended maximally and the patellae are pointing forward. Blocks are placed under the shorter leg to maintain a level pelvis. Besides standard anteroposterior and lateral radiographs and for hip and knee joint axial views, the weight-bearing radiographs of joints provide the best information to analyze osteoarthritis. Rotational deformities of both the upper and lower extremity are measured clinically using the rotational profile and by computer tomography using horizontal cuts obtained with the limb aligned, preferably in specific leg or foot holders. Scan images at each standard level of the limb or part of the limb are taken, while both sides are scanned for comparison to a normal side or in relation to normal values. MRI scans may add valuable information on cartilage condition; meniscus, ligament, and soft tissue damage; and the location of nerves and vessels relative to the malunion and the area of deformity correction.

The first step in frontal plane analysis is to draw a line from the center of the femoral head to the center of the tibial plafond at the ankle joint. This line should pass slightly medial of the center of the knee joint on the long-leg view through the medial eminence. In frontal plane deformities, this line passes lateral to the tibial spines in a valgus leg and medial to the tibial spines in a varus deformity. Measurements of leg length are made to identify a leg length discrepancy.

In the second step, the mechanical axes of the tibia and the femur are drawn. The femoral mechanical axis is the line between the center of the femoral head and the center of the knee joint and differs from the anatomical axis in the femur which is the mid-diaphyseal line. The tibial mechanical axis is a line drawn from the center of the tibial plafond to the center of the knee joint, which usually corresponds to the mid-diaphyseal anatomical axis in the tibia. The angle at the intersection of the tibial and femoral mechanical axes at the knee joint gives the magnitude of the whole leg deformity.

The third step of the analysis, defining the alignment of the joints of the lower leg, will reveal the location of the deformity. Hereto lines are drawn through the hip, knee, and ankle joint, defining the joint orientations. The angle between the femoral and tibial mechanical axis lines and the respective joint orientation lines identifies the bone segment responsible for the deformity. The normal values as defined by Paley [4] are most often used.

With the fourth step of the malalignment analysis, the location of the deformity within a bone segment can be found. For this, the anatomical axes of both the femur and the tibia are used. These axes are defined by the mid-diaphyseal lines of each bone segment. In a deformed bone segment, the proximal and distal anatomical axes intersect. Paley [4] defined this intersection point as the center of rotation of angulation (CORA) and described an extensive deformity analysis and planning method for deformity correction based on the CORA method.

In the sagittal plane, a deformity can be identified by intersecting the anatomical axes in the sagittal plane and establishing joint orientation angles at the proximal and distal femur and tibia using anatomical axes. Rotational deformities can be analyzed accurately using CT and related to reference values [5]. It should be noted that racial differences in normal values for lower leg rotation profile are present. Radiographs and CT measurements can also be used to analyze the patellar tracking. Specifically in patients with patellar instability and pain, it is important to measure parameters such as patellar height, patellar tilt, and the relation between the trochlea and tibial tuberosity in the transverse plane [6]. In the planning for corrective procedures, the effects for patellar tracking should be accounted for.

8.1.3 Correlation of Physical Examination and Radiological Deformity Analysis

Accurate range of motion measurements obtained during physical examination should now be compared to the measurements obtained at the radiological deformity analysis. This will give important information for the planning of deformity correction aimed at restoring limb function. Corrections based on only the bony deformity found in radiographs may cause an overcorrection or undercorrection of the limb deformity that may even worsen the limb function although the bone deformity may be corrected. Therefore, it is important to use both the findings at physical examination and radiologic measurements to define the deformity.

8.1.4 Definition of the Deformity and Aim of Correction

Angular deformities present themselves either in the frontal plane causing valgus or varus of the affected limbs or in the sagittal plane causing a recurvatum or procurvatum deformity. Rotation of a segment around its axis causes a rotational deformity, while shortening presents as a limb length discrepancy. These deformities on their own are termed uniplanar deformities [4]. If two or more deformities coexist in the same bone segment, these are termed biplanar and multiplanar deformities, respectively. The site of the deformity may be at the diaphysis or metaphysis or at the level of the joint and may be either unifocal or multifocal if the deformity coexists with another at more than one level within the same segment of the bone.

All factors described above should be taken into account before the aims of correction of the limb deformity with one or more osteotomies can be properly met. Different aims can be chosen varying from creation of “a leg to stand on” with the main purpose being the ability to bear weight to a purposely varus of valgus alignment unloading a unicompartmental osteoarthritis. Also a (near) anatomical correction in neutral alignment to recreate joint alignment and restore joint function or as an addition to cartilage reconstructive procedures can be the aim of a correction.

8.1.5 Planning of Deformity Correction

After the deformity has been described and the aim of the planning of the deformity correction is determined, the planning can be started. Different approaches can be used to plan the correction, and many techniques have been described in the literature. In the planning for corrections of rotational deformities, the patellar tracking should be accounted for [5, 6].

8.1.6 Hardware Selection

It is of major importance to follow the steps for the formation of a surgical plan in the correct order. Selecting a fixation method as a first step of the surgical plan may restrict the correction options and may even result in the creation of a secondary deformity after the correction [7]. It is most important to create a rigid fixation as this will allow for functional postoperative rehabilitation with early partial or full weight bearing. Regarding hardware selection for osteotomy fixation plates, intramedullary nails and several types of external fixators are available [8].

8.1.7 Description of Surgical Tactic

The surgical tactic is the outline of the sequential steps in the operating room, which will lead to the desired result. It is the final step of the surgical plan before surgery is performed, and the operative procedure is now well-planned. For optimal preparation of deformity correction, the following questions, adapted from fracture treatment planning [9], will help the surgeon to make the final preparations for the surgery:

1. Is the proposed osteotomy site surgically accessible?
2. Can the plan be carried out using intraoperative guides (e.g., k-wires, templates, saw guides) to enhance accuracy?
3. Is the location of the bone cuts biologically reasonable (living bone, no infection, extreme scarring, neurovascular compromised status, previous musculocutaneous flap surgery)?
4. Is stable fixation possible and, if not, how will additional fixation (e.g., cast, brace) be applied?
5. Can the soft tissues withstand the anticipated degree of bony correction (lengthening, shortening, straightening)?

In conclusion, the preparation for correction of lower limb deformities using diagnostics and planning of deformity correction will only lead to a predictable good result if this information is part of a surgical plan (Fig. 8.1). Formation of a surgical plan will not only protect the patient undergoing the deformity correction but will also help the surgeon who carefully prepared the surgical procedure in case complications arise from the operation.

8.2 Ten Bullet Points for HTO and DFO

Bullet points have been a subject in a previous ICL session of ESSKA. In that session, osteotomy experts were given a choice to make their own list of bullet points regarding high tibial osteotomy (HTO) and distal femoral osteotomy (DFO). Video registration of their lectures can be viewed at the ESSKA academy site. From these lectures it was concluded that it should be possible to formulate a bullet point list for the most commonly performed opening and closing wedge varus and valgus corrections around the knee. The purpose of the bullet point list is that it can be used as a reference for things to do or things not to forget when performing HTO and DFO. Here, a personal preference of bullet points is presented together with some background information. For further information on the specific bullet points, the reader is referred to textbooks and the ever-increasing list of articles published regarding osteotomies around the knee [10–15].

8.2.1 Medical History and Physical Exam

The medical history is, independent of the speciality in medicine, most important to arrive at the correct indication for treatment. Patients who are complaining of pain and limitation during walking have a disability in daily living. Depending on culture and living area problems, degenerative changes in the knee joint start earlier or later in life. In elderly patients, it is rather biological age than calendar age that is of importance for the choice of treatment.

Furthermore, the knowledge of the activity and pain level is fundamental, as well as the expectation of the patient [16]. Activity wish and pain tolerability of the patient should be discussed. Explanation of the possible result of

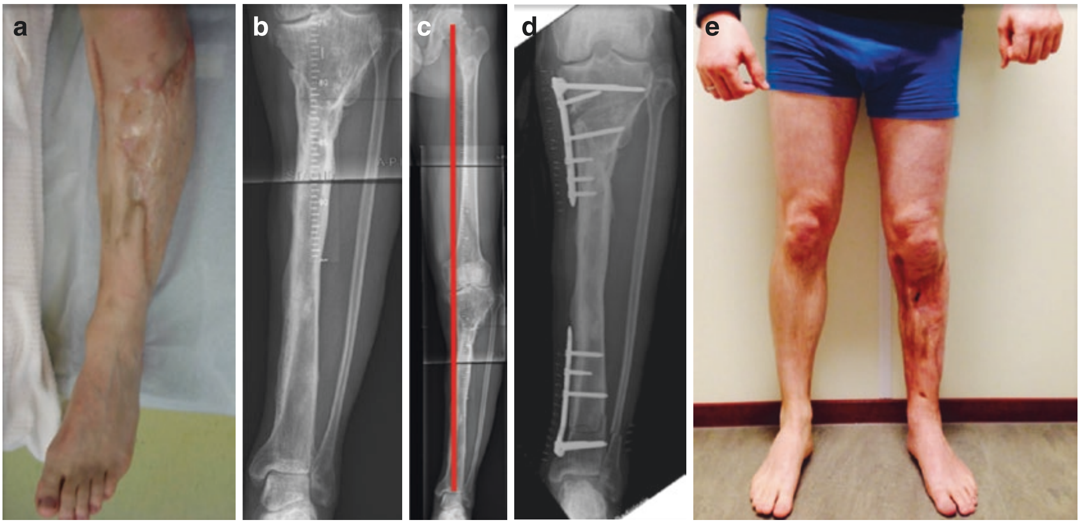


Fig. 8.1 Surgical plan for a patient with symptomatic varus medial compartment OA and 20° internal rotation malunion after motor vehicle accident and infected malunion treatments. Severe skin scarring after musculocutaneous flap transfers and internally rotated foot position (**a**), varus tibial deformity (**b**), and weight-bearing line passes medial side of the knee (**c**); after analysis the aim for correction is a valgus leg alignment unloading the medial compartment combined with an external rotation osteotomy to symmetrically normal external rotation foot position. Surgical tactics analysis: the soft tissue situation allows for a medial proximal tibial approach for opening wedge HTO however not for a combined proximal derotation osteotomy. Therefore a distal tibia and fibula derotation osteotomy was performed as part of a double-level tibial correction fixated with angle-stable implants (**d**). Postoperative picture shows corrected leg alignment (**e**)

the planned surgery and the possible complication is mandatory and will be relevant to the outcome.

Besides the standard knee examination including ligament laxity evaluation, observing the patient's gait pattern gives important information—fast, slow, limping, or even using crutches. Moreover, how the patient positions his foot (external, internal) and the knee is valuable information. Muscle forces and ligaments differ among patients and could be of relevance for the planned treatment.

8.2.2 Diagnostics (Radiographs, CT, MRI)

Basic diagnostics for all patients with possible osteoarthritis in the knee or cartilage defects are anteroposterior and lateral radiographs. In addition, long-leg full weight-bearing radiographs, patella views, and Rosenberg views are often indicated. If a torsional deformity is found at physical examination, a CT scan with measurements of transverse slides at standardized positions is mandatory. Nowadays, most patients present with an MRI scan of their knee already made, which may confirm pathology seen on radiographs as well as integrity of structures in other parts of the joint.

8.2.3 Deformity Analysis and Planning Depending on the Location of the Deformity

The basis for all osteotomies is a proper and perfect deformity analysis, which is based on a correct radiograph. Meanwhile, the deformity analysis is recommended according to Paley [17, 18]. He was not the inventor of how to measure angles, yet he defined Paley's systematic nomenclature of angles of the lower extremity. Depending on the result of the deformity analysis, a simulation of the deformity correction (osteotomy) is possible. A varus and a valgus bone deformity can be localized at the (distal) femur as well as in the (proximal) tibia or even involve both the tibia and femur. The planning for correction should always respect the location of the deformity. Not only the alignment should be restored according to the aim defined in the surgical plan, but also the joint angles should be (re) aligned, avoiding abnormal joint line obliquity.

8.2.4 Correction of the Frontal Plane: Biplanar HTO and DFO

Different osteotomy techniques correcting frontal plane, distal femoral, and proximal tibial deformities have been described. Currently, regardless of whether an opening or a closing wedge osteotomy is performed, a biplanar technique is recommended [13, 19, 20], not so much because of increased initial stability, but rather because of increased bone healing potential through increased contact healing and decreased gap healing. Furthermore, the biplane osteotomy guides the surgeon intraoperatively, indicating that when opening or when displacement of the biplanar osteotomy plane takes place, a secondary deformity should be corrected.

8.2.5 Multiple Plane Corrections

In case of multiple plane deformity, there is no consensus of the type of osteotomy and fixation technique. However, if a one-step correction is planned, the surgeon needs advanced skills in deformity correction. Techniques like oblique osteotomies and knowledge of the influence of rotation and lengthening to the frontal plane are mandatory to achieve a perfect result.

8.2.6 Osteotomies Combined with Ligament Reconstructions, Patella Stabilization, and Cartilage Repair

The combination of an osteotomy with ACL reconstruction is possible [21] and the results are successful. However, the success depends on the experience of the surgeon. Before starting a combined surgery, sufficient experience of simple osteotomy procedures is recommended. Combinations of osteotomies and stabilization procedures like MPFL reconstruction or tuberosity transfers in case of patella instability are considered complex osteotomies. When several different pathologies are present, a thorough analysis is required in these often young patients. It is not always necessary to treat all problems in the same procedure. If the surgeon is in doubt of multiple surgeries in one step, it is recommended to perform the treatment in several steps. Cartilage repair procedures for traumatic as well as chronic cartilage defects in the presence of a leg deformity causing overloading of the defect lead to superior results if surgery is combined with a (partially) unloading osteotomy [22, 23].

8.2.7 Fixation Technique

Today, many different types of implants are available. The most commonly used types of implants are plate fixators with angular stable locking screws and short spacer plates [24–26]. Complications related to correction loss reported for the short spacer plates are higher, especially in the presence of hinge fractures. Because of that, the plate fixator seems to have become the gold standard for closing wedge and opening wedge osteotomies. However, if there is no hinge fracture and the postoperative rehabilitation is adjusted to the less stable implant, comparable results are possible.

8.2.8 Gap Filling and Additional Bone Support

Preferences of surgeons differ, and cultural changes are present regarding the choices made to fill the gap created during opening wedge osteotomies of the tibia and the femur. Gaps may be filled with autologous [27–29] or allograft bone material [30, 31] or bone substitutes [32, 33]. Gap fillers may be used to decrease blood loss out of the gap and to promote bone healing. Depending on fixation strength of the osteotomy, fixation method gap filling may also be used to increase fixation strength. Bone support by additional bone interposition underneath the plate may be used as part of the procedure of closing wedge osteotomies of the tibia and the femur. Specifically when a large step-off is present between fragments while using malleable implants for fixation, bone support between the plate and bone is advised to prevent a hinge fracture during osteotomy fixation.

8.2.9 Prevent and Deal with Complications

Complications of osteotomies are not frequent, but can be very debilitating. Regarding surgery, one has to anticipate complications by careful preparation of surgery, prevention of infection, and treatment of intraoperative complications of the procedure. Infections can be prevented by preoperative administration of antibiotics, which is mandatory irrespective of the osteotomy or fixation method used.

Hinge fractures [34] present during surgery and should be dealt with if instability of the osteotomy results from the hinge fracture [35, 36]. In general, compression of the hinge point after opening the gap results in a more stable fixation and faster progression of bone healing. For opening wedge HTO, hinge fractures extending to the tibial plateau should be treated with additional fixation and adaptation of weight-bearing rehabilitation protocol. Hinge fractures extending distally in the proximal tibiofibular joint demand adaptation of hinge compression to prevent distraction and adaptation of weight-bearing rehabilitation [35, 36]. Additional fixation of hinge fractures can be performed with screws, staples, or plate fixation aimed at compression of the hinge point. Similar additional fixation methods can be used for hinge fractures in closing wedge HTO. However, in closing wedge osteotomy, one should anticipate on prevention of translation in the osteotomy during fixation, for example, by using interposing bone block or specific fixation materials. For hinge fractures in distal femoral osteotomies, one should realize that displacing forces are higher than in HTO. Therefore, additional hinge compression and fixation with plate fixation is even more important to prevent bone healing problems and fixation failures.

8.2.10 Rehabilitation Tailored to Osteotomy Technique and Strength of Fixation

Rehabilitation after osteotomies including exercises to regain function and full weight bearing depends on the type of osteotomy and the fixation technique. Physiotherapy without limitation of ROM until the patient achieves full mobilization is of importance, and in the first 6 weeks, a surgical stocking is helpful to reduce swelling and pain. Biplanar osteotomy techniques used for opening and closing wedge tibial and femoral osteotomies cause higher initial stability during surgery and the early postoperative phase. Different types of fixation have a difference in tolerance to full weight bearing [37, 38]. Besides that, the bone quality and the presence of hinge fractures should also be accounted for in the choice of rehabilitation protocol [34, 39]. Plate fixator fixation of osteotomies in general allows for immediate full weight bearing after wound healing (2 weeks after surgery). Whether the patient is able to follow this recommendation depends on the pain experienced by the patient. Small spacer plates are less stable and require a partial weight-bearing rehabilitation protocol between 8 and 12 weeks [40].

8.3 Filling the Gap

Medial opening wedge high tibial osteotomy (MOW HTO) remains a major undertaking for patients with significant levels of pain and swelling reported in the initial postoperative period. There are also concerns around the reported rates of delayed and non-union. To date, there has been a range of methods employed to fill the osteotomy site including autograft, allograft, and synthetics with variable reported outcomes. The primary use of grafting has been to promote healing as well as provide a structural support to the osteotomy [41].

8.3.1 Graft Options

Autologous bone graft offers significant advantages including osteogenic, osteoinductive, and osteoconductive properties. There are additional benefits of lower delayed or non-union rates compared to other sources of bone graft. Autograft remains an attractive choice in patients who are smokers or obese in order to reduce the risk of osteotomy failure [31, 42]. The disadvantage remains around the donor site morbidity, which can lead to prolonged pain. There are increased surgical time and an additional risk of infection and intraoperative complications due to the additional procedure to harvest the graft.

The use of allograft avoids donor site morbidity and has osteoconductive characteristics. The use of allograft in osteotomy surgery has been shown to have a predictable course with the use of femoral head shaped to fit the osteotomy gap [43]. There are, however, concerns related to disease transmission and immunological reaction and rejection. The reported figure for this is said to be less than that for a blood transfusion [44]. While these remain very low, this can influence decision making when consenting patients for surgery. In a recent systematic review comparing autograft, allograft, and synthetic bone grafts, allograft demonstrated the lowest rate of infection [42]. The rates of non-union were identical to autograft at 0.5%, with the synthetic group having a non-union rate of 1.1%.

Synthetic grafts largely consist of calcium and phosphate. They seek to mimic the porosity of cancellous bone in allowing infiltration of neovasculature and eventually osteoprogenitor cells. In the early phase after surgery, they may also provide structural support [31]. Collagen-based void fillers aim to provide a framework for bone cells to attach to and allow bone formation and may also contain bone morphogenetic proteins to encourage osteoconductive activity. Synthetic grafts have lower morbidity and cost in comparison with autograft and allograft. Concerns include the increased risk of infection and loss of correction when using synthetic grafts [45], particularly for adversely affecting the tibial slope.

8.3.2 Gap Size

Gap size may influence the use of graft in osteotomy surgery, with larger osteotomies potentially more likely to be filled with graft. Jung et al. demonstrated a 91% union rate at 3 months with an osteotomy gap of 7 mm using allograft [43], with larger osteotomies taking considerably longer [46]. It has been suggested that gaps greater than 13 mm should undergo filling of the osteotomy gap [47, 48]. Our experience in filling the gap for osteotomy agrees with the published data on union times. We have found it particularly useful in smaller osteotomies to increase the accuracy of the desired osteotomy gap. In shaping a femoral head allograft to the precise size required to fill the gap, we have had positive results in achieving the desired correction in both the coronal and sagittal plane.

Modern plates have been designed in such a way that bone graft is not required to fill the osteotomy gap [49]. In many countries due to cost implications, legal limitations, and local policy, the use of human allograft is prohibited. As a result, the plates have been designed to allow the osteotomy to heal without additional graft in the gap. There are, however, clear advantages to provide further structural support and allow early recovery. We suggest that grafting may also possess the properties suitable to reduce the postoperative swelling and pain by “plugging the gap.”

The philosophy as to whether one should fill the gap continues to be controversial. In certain instances, surgeons may choose to fill the gap depending on the plating system used. For example, the first-generation Puddu plate required additional structural support, and grafting was considered advantageous. There is also still a perception by many that you need to fill the gap to minimize the risk of failure of the plate.

The indications for filling the osteotomy gap remain unclear. While some authors have suggested filling the gap for smokers and larger osteotomies, others have demonstrated improved precision when using grafts for smaller osteotomy corrections [36, 50]. Revision osteotomy surgery remains a strong indication for using bone graft in order to reduce the risk of non-union.

There is convincing evidence that with modern fixed-angle plates, there is no need to graft and patients can weight-bear fully from day 1 without concern.

8.3.3 Graft Versus No Graft Research

We hypothesized that filling the gap would act like a cork in a bottle, to minimize hematoma formation and postoperative swelling. This would also provide additional structural support to make the MOW HTO patients feel less pain because of additional stability. We investigated the potential benefits of bespoke allograft bone wedges with respect to postoperative pain, initial swelling, length of stay, early outcome scores, surgical accuracy, and time to union.

We prospectively randomized two groups of patients either to receive a bespoke femoral head allograft (group 1) to fill the osteotomy gap or to leave the osteotomy gap empty (group 2). Both groups underwent osteotomy surgery in the standard fashion using the same implant. Femoral heads from donor total hip replacement patients were utilized as an allograft source from our established local bone bank.

In group 1, the femoral head allograft was fashioned to a bone wedge that matched the gap size as per our preoperative planning (Fig. 8.2). Both groups were asked to report pain and swelling using a visual analogue scale at the following stages postoperatively: day 1 and weeks 1, 2, 3, 6, 9, and 12. Postoperative long-leg alignment radiographs were analyzed against preoperative digital plans to assess accuracy of correction, and radiographs at week 12 were reviewed for osteotomy union. Subjective scoring using the Oxford Knee Score (OKS) and Knee Injury and Osteoarthritis Outcome Score (KOOS) was completed preoperatively and repeated at 12 weeks. Length of hospital stay and postoperative analgesic requirement were also recorded.

Patients were all followed in our research clinic as part of our standard management where we have a close to 1000 osteotomies being followed up.

Fig. 8.2 Femoral head allograft preparation (left) of a precision bone wedge (right)

There was a significant reduction in pain at day 1 and weeks 1, 2, and 3 in the patients who received the allograft femoral head bone wedges compared to those that didn’t ($p < 0.05$). The difference was still noted at later time points, but these scores were not statistically significant (Fig. 8.2). While we did not observe any statistical difference in accuracy of the long-leg alignment views postoperatively in either group, there were fewer outliers in the accuracy of surgical correction in group 1 (Fig. 8.3).

8.3.4 Future Treatment Options

Under- and overcorrection are potential pitfalls in osteotomy surgery. Advances in imaging and

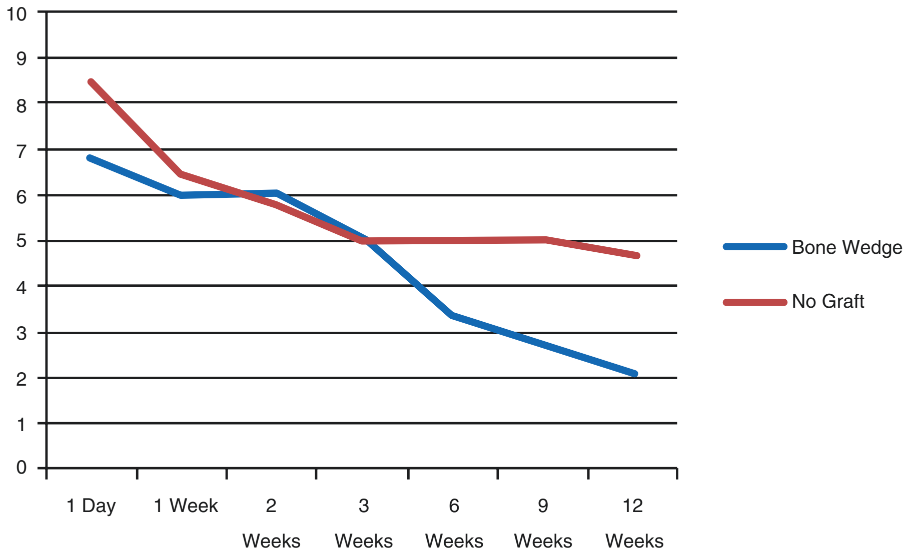


Fig. 8.3 Postoperative pain using femoral head allograft compared to no graft

planning software have allowed us to improve the accuracy of the osteotomies. The use of weight-bearing long-leg alignment views, CT, and gait biomechanics has improved our understanding of how one should aim to correct the deformity.

In the past, osteotomies have largely been used to correct deformities in the coronal plane. We have developed techniques in correcting for deformities in the sagittal plane, in particular by addressing the posterior tibial slope. A slope-changing osteotomy gives one the ability to correct for de-tensioned cruciate ligaments that can result in instability. This osteotomy can have a profound effect on the stability of the knee without necessarily needing to perform intra-articular surgery.

High-volume local anesthetics, cold compression therapy, and the recent use of bone wedge allograft have significantly reduced the postoperative pain experienced. This has accelerated the rehabilitation in the early postoperative period with improved patient satisfaction. As well as acting as a structural support and reducing pain, we have observed some patients healing at 3 months, allowing early removal of hardware if required.

We are conducting a biomechanical study to investigate the changes in structural stability when using bone wedge allograft.

In conclusion, precision bone wedge allograft augmentation of the gap in medial opening wedge high tibial osteotomy is a safe method of reducing patient pain scores in the early postoperative period. It also helps to reduce the number of outliers in the accuracy of your osteotomy corrections. Future development in osteotomy surgery may involve the use of biological enhancement means to accelerate the bone healing process and reduce the risk of non-union.

8.4 Risks and Complications of Osteotomies Around the Knee

Valgus-producing high tibial osteotomy is a cost-effective and joint-preserving treatment option for primary varus malalignment. Wrong indications, technical errors, and insufficient postoperative management can lead to serious complications such as loss of correction, non-union, and persistence of pain. Major risk factors for complications are wrong indications and poor planning for high tibial osteotomy. During surgical planning, long-leg standing radiographs with the correct limb rotation are mandatory. Joint space opening of the noninvolved compartment must be considered to avoid overcorrection [51]. Intraoperative opposite cortex fractures of the lateral hinge cannot be avoided in all patients: In osteotomies with larger correction angles, the capacity for elastic deformation is frequently exceeded, resulting in plastic deformation and fracture of the opposite cortex, which may lead to subsequent loss of correction or malunion. An anteroposterior drill hole at the apex of the horizontal osteotomy (i.e., the hinge point) is supposed to increase the capacity of the bony hinge for elastic deformation and ideally to prevent fractures of the opposite cortex. However, clinical and biomechanical studies proved that the hinge-protecting effect is restricted to small correction angles of 5° used, i.e., to unload cartilage repair regions in the absence of severe malalignment. For the treatment of a significant varus gonarthrosis, the fracture-protecting effect from a hinge drill is almost absent since correction angles above 8° are necessary [52].

Thus, implants need to be used which enable a temporary lag screw distal to the osteotomy to be able to reduce the fracture of the opposite cortex and preload the hinge until the angle-stable implant is secured.

In addition, vascular structures must be protected during the osteotomy of the posterior cortex. A variation of the origin of the anterior tibial artery with a course between the posterior tibial cortex and the popliteal muscle was found in 6% of all patients and predisposes to an accidental injury during osteotomy. The results in the literature and our own MRI findings suggest that a flexion angle of 90° facilitates anatomical dissection and osteotomy but cannot be regarded as a reliable protection against vascular injury [53]. Finally, reducing the amount of slow gap healing and simultaneously increasing the area of faster contact healing may be beneficial for osteotomy healing. Thus, biplanar rather than uniplanar osteotomy should be performed for osteotomy around the knee [54, 55].

In cases with non-union, high-energy shock wave therapy or autologous cancellous bone grafting is usually sufficient to support bone healing. Infections are frequently associated with the implant. Early infections may be treated with debridement and local or systemic antibiotics. Late infections usually require the removal of the implant and the use of an external fixator if the osteotomy is still unstable.

8.5 HTO vs Unicondylar Knee Prosthesis

High tibial osteotomy (HTO) and unicondylar knee prosthesis (UKP) both address unilateral osteoarthritis of the knee. Comparative studies demonstrate similar clinical results [33, 56]. The indications for HTO have been expanded and include nowadays also patients with bone-on-bone osteoarthritis on the involved side [2, 3, 57, 58] as well as patients with high age. The results of UKP have been proven to be very good even in patients with high activity level [59]. The question therefore remains what valid decision criteria should be used to choose between HTO and UKP?

8.5.1 Constitutional Deformity

UKP replaces the worn surfaces on the involved side of the knee. The procedure restores normal joint space height and corrects any frontal plane deformity resulting from the collapse of the joint space. The patient will receive the frontal plane alignment he had before he developed osteoarthritis (OA). UKP cannot correct any extra-articular deformity, usually from the metaphysis of tibia or femur. UKP is quite tolerant against frontal plane malalignment, but the patient will keep his pathological gait pattern and will continuously overload his UKP. Osteotomy may correct any metaphyseal malalignment and normalize gait pattern and load balance of the knee. Intra-articular defects may be compensated for by overcorrection. Significant constitutional deformity should be treated by osteotomy. Medial osteoarthritis without constitutional deformity should be treated by UKP (Fig. 8.4).

8.5.2 Ligament Stability

Normal collateral ligament status is an obligatory prerequisite for UKP. An intact anterior cruciate ligament is considered crucial for success of UKP by most authors. Chronic anterior cruciate deficiency results in a specific posteromedial OA type and chronic anterior tibia subluxation, making balance and tensioning of a UKP very difficult. If an osteotomy is indicated, frontal plane and sagittal plane correction can be combined with reduction of the tibial slope. Slope reduction has been proven to reduce anterior tibia translation significantly [60].

8.5.3 Grade of Osteoarthritis and Indications

Most studies found good and predictable results of UKP in cases with bone-on-bone defects of the involved compartment (grade IV OA, Kellgren-Lawrence scale). The results of UKP were unpredictable in less advanced OA, and this procedure should therefore be reserved for patients with bone-on-bone osteoarthritis. Osteotomy has good results in all grades of OA if a constitutional deformity is corrected [61].

Undebated indications for osteotomy around the knee are symptomatic metaphyseal frontal plane deformities with OA grade Kellgren-Lawrence II to III of the involved compartment.

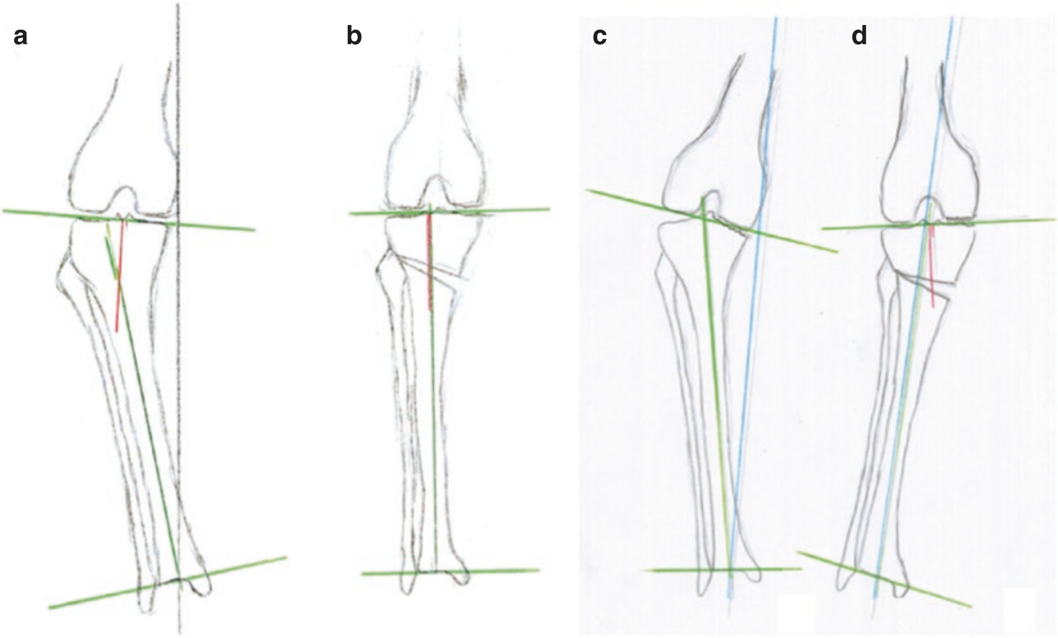


Fig. 8.4 Medial osteoarthritis with constitutional deformity (**a**) treated by an opening wedge HTO restores normal tibial anatomy (**b**). Medial osteoarthritis without constitutional deformity (**c**) treated by an opening wedge HTO creates abnormal anatomy with abnormal knee and ankle joint line orientation (**d**)

The indications can be expanded to cases with OA grade IV. Age plays no role for the result. Undebated indication for UKP is medial OA grade KL IV with no constitutional deformity and normal ligament status. Age and activity are no exclusion criteria. The indication for UKP can be expanded toward cases with moderate constitutional deformity (up to 5°).

Conclusions

Surgical details like the formation of a surgical plan, bulletpoints for HTO and DFO and the advantage of gap filling not only prevents risks and complications of osteotomy surgery but will also help to find the right indication for HTO in the treatment of unicompartmental osteoarthritis.

References

1. Van Heerwaarden RJ, Mast JW, Paccola CAJ. Diagnostics and planning of deformity correction: formation of a surgical plan. In: Marti RK, van Heerwaarden RJ, editors. Osteotomies for posttraumatic deformities. Stuttgart: Georg Thieme Verlag; 2008. p. 33–55.
2. Van Heerwaarden RJ. Osteotomies of the lower limb – the importance of making a surgical plan. ESSKA Newsletter December 2008; 2008. p. 4–8.
3. Stahelin L. Rotational problems of the lower extremities. Orthop Clin North Am. 1987;18(4):503–12.
4. Paley D. Principles of deformity correction. New York: Springer; 2002.
5. Van Heerwaarden RJ, et al. Derotation osteotomy for correction of congenital rotational lower limb deformities in adolescents and adults. Surg Tech Orthop Traumatol. 2003;55(10):575–85.
6. Dejour H, et al. Factors of patellar instability: an anatomic radiographic study. Knee Surg Sports Traumatol Arthrosc. 1994;2(1):19–26.
7. Van Heerwaarden RJ, Marti RK. Posttraumatic deformities and osteotomies. In: Marti RK, van Heerwaarden RJ, editors. Osteotomies for posttraumatic deformities. Stuttgart: Georg Thieme Verlag; 2008. p. 3–15.
8. Marti RK, Van Heerwaarden RJ, Van der Werken C. General considerations on indications, types of osteotomy, bone healing, and methods of fixation. In: Marti RK, van Heerwaarden RJ, editors. Osteotomies for post traumatic deformities. Stuttgart: Georg Thieme Verlag; 2008. p. 17–32.
9. Mast J, Jakob R, Ganz R. Planning and reduction technique in fracture surgery. Berlin: Springer; 1989.
10. Brinkman JM, Freiling D, Lobenhoffer P, Staubli AE, van Heerwaarden RJ. Supracondylar femur osteotomies around the knee: patient selection, planning, operative techniques, stability of fixation, and bone healing. Orthopade. 2014;43:988–99.
11. Brinkman JM, Lobenhoffer P, Agneskirchner JD, Staubli AE, Wymenga AB, van Heerwaarden RJ. Osteotomies around the knee: patient selection, stability of fixation and bone healing in high tibial osteotomies. J Bone Joint Surg Br. 2008;90(12):1548–57.
12. Lobenhoffer P, Agneskirchner J, Zoch W. Die öffnende valgisierende Osteotomie der proximalen Tibia mit Fixation durch einen medialen Plattenfixateur. Der Orthopade. 2004;33(2):153–60.
13. Lobenhoffer P, Agneskirchner JD. Improvements in surgical technique of valgus high tibial osteotomy. Knee Surg Sports Traumatol Arthrosc. 2003;11(3):132–8.
14. Lobenhoffer P, Kley K, Freiling D, van Heerwaarden R. Medial closed wedge osteotomy of the distal femur in biplanar technique and a specific plate fixator. Oper Orthop Traumatol. 2017;29(4):306–19.
15. Lobenhoffer P, Van Heerwaarden R, Agneskirchner J. Kniegelenknahe Osteotomien, vol. 2. Stuttgart: Thieme; 2014.
16. Takeuchi R, Ishikawa H, Aratake M, et al. Medial opening wedge high tibial osteotomy with early full weight bearing. Arthroscopy. 2009;25(1):46–53.
17. Paley D, Herzenberg JE, Tetsworth K, McKie J, Bhave A. Deformity planning for frontal and sagittal plane corrective osteotomies. Orthop Clin North Am. 1994;25(3):425–65.
18. Paley D, Tetsworth K. Mechanical axis deviation of the lower limbs. Preoperative planning of uniapical angular deformities of the tibia or femur. Clin Orthop Relat Res. 1992;280:48–64.
19. Freiling D, van Heerwaarden R, Staubli A, Lobenhoffer P. The medial closed-wedge osteotomy of the distal femur for the treatment of unicompartmental lateral osteoarthritis of the knee. Oper Orthop Traumatol. 2010;22(3):317–34.
20. Staubli AE, De Simoni C, Babst R, Lobenhoffer P. TomoFix: a new LCP-concept for open wedge osteotomy of the medial proximal tibia–early results in 92 cases. Injury. 2003;34(Suppl. 2):B55–62.
21. Hinterwimmer S, Mehl J. Combination of ACL-replacement and high tibial osteotomy. Oper Orthop Traumatol. 2014;26(1):43–55.
22. Bode G, Kloos F, Feucht MJ et al. Comparison of the efficiency of an extra-articular absorber system and high tibial osteotomy for unloading the medial knee compartment: an in vitro study. Knee Surg Sports Traumatol Arthrosc. 2017; 25(12):3695–703. https://doi.org/10.1007/s00167-016-4358-9.

23. Feucht MJ, Minzlaff P, Saier T, et al. Degree of axis correction in valgus high tibial osteotomy: proposal of an individualised approach. Int Orthop. 2014;38(11):2273–80.
24. Agneskirchner JD, Freiling D, Hurschler C, Lobenhoffer P. Primary stability of four different implants for opening wedge high tibial osteotomy. Knee Surg Sports Traumatol Arthrosc. 2006;14(3):291–300.
25. Diffo Kaze A, Maas S, Waldmann D, Zilian A, Dueck K, Pape D. Biomechanical properties of five different currently used implants for open-wedge high tibial osteotomy. J Exp Orthop. 2015;2(1):14.
26. Petersen W, Wall A, Paulin T, Park HU, Heymann L. Stability of two angular stable locking plates for open wedge high tibial osteotomy (HTO): TomoFix versus LOQTEQ(R) HTO plate. Arch Orthop Trauma Surg. 2014;134(10):1437–42.
27. Akiyama T, Okazaki K, Mawatari T, Ikemura S, Nakamura S. Autologous osteophyte grafting for open-wedge high tibial osteotomy. Arthrosc Tech. 2016;5(5):e989–95.
28. Schroter S, Mueller J, van Heerwaarden R, Lobenhoffer P, Stockle U, Albrecht D. Return to work and clinical outcome after open wedge HTO. Knee Surg Sports Traumatol Arthrosc. 2013;21(1):213–9.
29. Zorzi AR, Da Silva HG, Muszkat C, Marques LC, Cliquet A Jr, De Miranda JB. Opening-wedge high tibial osteotomy with and without bone graft. Artif Organs. 2010;35(3):301–7.
30. Haviv B, Bronak S, Thein R, Kidron A, Thein R. Mid-term outcome of opening-wedge high tibial osteotomy for varus arthritic knees. Orthopedics. 2012;35(2):e192–6.
31. Lash NJ, Feller JA, Batty LM, Wasiak J, Richmond AK. Bone grafts and bone substitutes for opening-wedge osteotomies of the knee: a systematic review. Arthroscopy. 2015;31(4):720–30.
32. Takeuchi R, Bito H, Akamatsu Y, et al. In vitro stability of open wedge high tibial osteotomy with synthetic bone graft. Knee. 2010;17(3):217–20.
33. Takeuchi R, Umemoto Y, Aratake M, et al. A mid term comparison of open wedge high tibial osteotomy vs unicompartmental knee arthroplasty for medial compartment osteoarthritis of the knee. J Orthop Surg Res. 2010;5(1):65.
34. Takeuchi R, Ishikawa H, Kumagai K, et al. Fractures around the lateral cortical hinge after a medial opening-wedge high tibial osteotomy: a new classification of lateral hinge fracture. Arthroscopy. 2012;28(1):85–94.
35. Schroter S, Ateschrang A, Ihle C, Stockle U, Konstantinidis L, Dobele S. Lateral hinge fractures in open wedge high tibial osteotomy. Orthopade. 2014;43(11):1000–7.
36. Schroter S, Freude T, Kopp MM, et al. Smoking and unstable hinge fractures cause delayed gap filling irrespective of early weight bearing after open wedge osteotomy. Arthroscopy. 2015;31(2):254–65.
37. Brinkman JM, Luites JW, Wymenga AB, van Heerwaarden RJ. Early full weight bearing is safe in open-wedge high tibial osteotomy. Acta Orthop. 2010;81(2):193–8.
38. Schroter S, Ateschrang A, Lowe W, Nakayama H, Stockle U, Ihle C. Early full weight-bearing versus 6-week partial weight-bearing after open wedge high tibial osteotomy leads to earlier improvement of the clinical results: a prospective, randomised evaluation. Knee Surg Sports Traumatol Arthrosc. 2017;25(1):325–32.
39. Nakamura R, Komatsu N, Murao T, et al. The validity of the classification for lateral hinge fractures in open wedge high tibial osteotomy. Bone Joint J. 2015;97-B(9):1226–31.
40. Nelissen EM, van Langelaan EJ, Nelissen RG. Stability of medial opening wedge high tibial osteotomy: a failure analysis. Int Orthop. 2010;34(2):217–23.
41. Smith JO, Wilson AJ, Thomas NP. Osteotomy around the knee: evolution, principles and results. Knee Surg Sports Traumatol Arthrosc. 2013;21(1):3–22.
42. Slevin O, Ayeni OR, Hinterwimmer S, Tischer T, Feucht MJ, Hirschmann MT. The role of bone void fillers in medial opening wedge high tibial osteotomy: a systematic review. Knee Surg Sports Traumatol Arthrosc. 2016;24(11):3584–98.
43. Jung KA, Lee SC, Ahn NK, Hwang SH, Nam CH. Radiographic healing with hemispherical allogeneic femoral head bone grafting for opening-wedge high tibial osteotomy. Arthroscopy. 2010;26(12):1617–24.
44. Buck BE, Resnick L, Shah SM, Malinin TI. Human immunodeficiency virus cultured from bone. Implications for transplantation. Clin Orthop Relat Res. 1990;251:249–53.
45. Gouin F, Yaouanc F, Waast D, Melchior B, Delecrin J, Passuti N. Opening wedge high tibial osteotomies: calcium-phosphate ceramic spacer versus autologous bonegraft. Orthop Traumatol Surg Res. 2010;96(6):637–45.
46. Santic V, Tudor A, Sestan B, Legovic D, Sirola L, Rakovac I. Bone allograft provides bone healing in the medial opening high tibial osteotomy. Int Orthop. 2010;34(2):225–9.
47. Galla M, Lobenhoffer P. Die öffnende valgisierende Umstellungsosteomie der proximalen Tibia mit dem TomoFix™-Plattenfixateur. Oper Orthop Traumatol. 2004;16(4):397–417.
48. Galla M, Riemer C, Lobenhoffer P. Die Osteosynthese posteromedialer Tibiakopffrakturen über einen direkten dorsalen Zugang. Oper Orthop Traumatol. 2009;21(1):51–64.
49. Aryee S, Imhoff AB, Rose T, Tischer T. Do we need synthetic osteotomy augmentation materials for opening-wedge high tibial osteotomy. Biomaterials. 2008;29(26):3497–502.

50. Schroter S, Ihle C, Elson DW, Dobele S, Stockle U, Ateschrang A. Surgical accuracy in high tibial osteotomy: coronal equivalence of computer navigation and gap measurement. Knee Surg Sports Traumatol Arthrosc. 2016;24(11):3410–7.
51. Holschen M, Lobenhoffer P. Komplikationen kniegelenknaher Umstellungsosteotomien. Der Orthopade. 2016;45:13–23.
52. Reyle G, Lorbach O, Diffo Kaze A, Hoffmann A, Pape D. Verhinderung einer Fraktur der Gegenkortikalis bei aufklappender Osteotomie. Der Orthopade. 2017;46:610–6. https://doi.org/10.1007/s00132-017-3418-2.
53. Gerich T, Lens V, Seil R, Pape D. Open wedge osteotomy of the tibial head. Management of vascular complications. Der Orthopäde. 2014;43:1008–15.
54. Pape D, Kohn D, van Giffen N, Hoffmann A, Seil R, Lorbach O. Differences in fixation stability between spacer plate and plate fixator following high tibial osteotomy. Knee Surg Sports Traumatol Arthrosc. 2013;21:82–9.
55. Pape D, van Heerwaarden RJ, Haag M, Seil R, Madry H. Kniegelenknahe Osteotomietechniken: Effekt auf Keilvolumina und knöcherne Kontaktflächen. Orthopade. 2014;43:966–75.
56. Stukenborg-Colsman C, Wirth CJ, Lazovic D, et al. High tibial osteotomy versus unicompartmental joint replacement in unicompartmental knee joint osteoarthritis: 7–10-year follow-up prospective randomised study. Knee. 2001;8:187–94.
57. Floerkemeier S, Staubli AE, Schroeter S, et al. Outcome after high tibial open-wedge osteotomy: a retrospective evaluation of 533 patients. Knee Surg Sports Traumatol Arthrosc. 2013;21:170–80.
58. Goshima K, Sawaguchi T, Sakagoshi D, et al. Age does not affect the clinical and radiological outcomes after open-wedge high tibial osteotomy. Knee Surg Sports Traumatol Arthrosc. 2017;25(3):918–23.
59. Price AJ, Dodd CA, Svard UG, et al. Oxford medial unicompartmental knee arthroplasty in patients younger and older than 60 years of age. J Bone Joint Surg Br. 2005;87(11):1488–92.
60. Bonnin M, Chambat P. Current status of valgus angle, tibial head closing wedge osteotomy in media gonarthrosis. Orthopade. 2004;33:135–42.
61. Niemeyer P, Koestler W, Kaehny C, et al. Two-year results of open-wedge high tibial osteotomy with fixation by medial plate fixator for medial compartment arthritis with varus malalignment of the knee. Arthroscopy. 2008;24:796–804.

The Role of Arthroscopy in Ankle Instability Treatment

9

Pietro Spennacchio, Gwen Vuurberg,
Stephane Guillo, Jón Karlsson, Jordi Vega,
and Helder Pereira

9.1 Introduction

Acute injuries of the ankle are one of the most common injuries seen by general practitioners and emergency departments [1]. They involvevapproximately 25% of all injuries of the musculoskeletal system. Inversion trauma, also referred to as lateral ankle sprains, constitutes a large proportion of these injuries [2–4].

Inversion sprain is typically associated with combined forced plantar flexion and inversion of the hindfoot around the externally rotated lower leg. This mechanism results in strain of the lateral ankle ligamentous complex. The anterior talofibular ligament (ATFL) is the first ligament to be damaged, followed by the calcaneofibular ligament (CFL) [5, 6].

In approximately 10–15% of all inversion injuries, there is a total rupture of the lateral ankle ligaments [7].

Considering lateral ankle ligament rupture, isolated lesion of the ATFL occurs in 65% of all injuries, while combined rupture of the ATFL and CFL occurs in approximately 20%. Isolated ruptures of the CFL are rare. The posterior talofibular ligament (PTFL), also a component of the lateral ligamentous complex, is usually not injured during inversion sprain [8, 9].

Although the natural history of ankle sprains is not fully known, it has been suggested that even untreated ligament ruptures might have a good prognosis. However, nonsurgical treatment

P. Spennacchio (✉)
Clinique du Sport, Centre Hospitalier Luxembourg,
Luxembourg, Luxembourg
e-mail: pietrospennacchio@gmail.com

G. Vuurberg
Department of Orthopedic Surgery,
Academic Medical Center, Amsterdam,
The Netherlands

S. Guillo
Centre de Chirurgie Orthopédique et Sportive,
Bordeaux-Mérignac, France

J. Karlsson
Department of Orthopaedics, Institute of Clinical
Sciences, Sahlgrenska Academy,
Gothenburg, Sweden

J. Vega
Foot and Ankle Unit, Hospital Quir.n Barcelona,
Barcelona, Plaza Alfonso Com.n 5, 08023,
Barcelona, Spain

Laboratory of Arthroscopic and Surgical Anatomy,
Department of Pathology and Experimental
Therapeutics (Human Anatomy Unit),
University of Barcelona, Barcelona, Spain

H. Pereira
Centro Hospitalar Póvoa de Varzim – Vila do Conde,
Póvoa de Varzim, Portugal

ICVS/3B's—PT Government Associated
Laboratory, University of Minho,
Braga-Guimarães, Portugal

Ripoll y De Prado Sports Clinic:
Murcia-Madrid—FIFA Medical Center
of Excellence, Madrid, Spain

G.M.M.J. Kerkhoffs et al. (eds.), *ESSKA Instructional Course Lecture Book*,
https://doi.org/10.1007/978-3-662-56127-0_9

fails in approximately 20% of patients after an inversion ankle sprain, and symptomatic chronic lateral ankle instability (CLAI) develops [10].

Patients with functional CLAI complain of an inability to depend on their ankle associated to repetitive episodes of "giving way" during which the joint exhibits pathologic inversion.

When a mechanical insufficiency of the lateral ligaments is present, surgical treatment of the ligaments may be considered in order to restore joint stability [11].

The surgical options to treat CLAI vary widely, from anatomical repair to non-anatomical reconstructions, with almost 80 different techniques described. The available literature shows that surgical strategy in terms of chronic lateral ankle instability is undergoing an evolution from traditional open procedures to minimally invasive techniques, with an increasing number of arthroscopic stabilization procedures being published [12–14].

9.1.1 Functional Anatomy and Biomechanics

The ankle complex comprises three articulations: the talocrural joint, the subtalar joint, and the distal tibiofibular syndesmosis. These three joints work together and are synchronized to allow coordinated movement of the hindfoot. The three major contributors to stability of the ankle joints are (1) the congruity of the articular surfaces when the joints are loaded, (2) the static ligamentous restraints, and (3) the musculotendinous units, which allow for dynamic stabilization of the joints [5, 15].

The relative contributions of these elements to joint stability vary significantly depending on the position and loading condition of the ankle complex [16].

In the neutral position, especially when coupled with compressive loads during weight-bearing, the bony architecture of the ankle joint is mostly important to create the stability of the joint. As the foot moves into plantar flexion, the ligamentous structures assume a crucial role in providing stability and become more susceptible to injury [9, 16].

The ATFL and the CFL are the key structures that contribute to lateral ankle stability [11]. Together with the PTFL, they form the lateral ligamentous complex and help to prevent inversion of the talus during plantar flexion and dorsiflexion of the foot [8], providing the primary static restraint to an inversion injury mechanism. In association with the medial ligamentous complex, the lateral ligaments also provide rotatory stability of the talus within the ankle mortise [16].

ATFL. The ATFL is a thickening of the tibiotalar capsule. It originates from the anterior margin of the lateral malleolus, with an average insertion 10 mm proximal to the tip of the fibula [17]. From its origin, the ATFL runs anteromedially to the insertion on the talar body immediately anterior to the joint surface occupied by the lateral malleolus. The ATFL is on average 7.2 mm wide and 24.8 mm long [17].

The ligament is virtually horizontal to the ankle in the neutral position but inclines upward in dorsiflexion and downward in plantar flexion. It is only in the latter position that the ligament comes under strain and is vulnerable to injury, particularly when the foot is inverted [7].

The ATFL demonstrates lower maximal load and energy to failure under tensile stress as compared with the PTFL, CFL, anterior inferior tibiofibular ligament, and deltoid ligament. This may explain why the ATFL is the most frequently injured of the lateral ligaments [18, 19].

In vitro kinematic studies have shown that the ATFL prevents anterior displacement of the talus from the mortise and excessive inversion and internal rotation of the talus on the tibia [5, 20–24]. After the ATFL is ruptured, the amount of transverse plane motion (internal rotation) of the hindfoot increases substantially, thus further stressing the remaining intact ligaments. This phenomenon has been described as "rotational instability" of the ankle and is often overlooked when considering laxity patterns in chronic ankle instability [25, 26].

CFL. The CFL originates from the anterior part of the lateral malleolus. Its origin is just below the lower band of the ATFL [27]. Frequently, fibers connecting these ligaments can be observed (Fig. 9.1). The confluency of the fibular insertion of the ATFL and the CFL furnishes the basis for proposed surgical procedures in which one common tunnel for fibular fixation of both the ATFL and CFL is created (Fig. 9.2) [11, 28].

The ligament runs obliquely downward and backward to attach to the posterior region of the lateral calcaneal surface [29, 30], bridging both the talocrural and subtalar joints.

The CFL is rounded, with a diameter of 6–8 mm, and its length is approximately 20 mm [27].

Most published anatomical observations identify the CFL as an extracapsular structure, rather than a capsular reinforcement, and it is intimately associated with the posteromedial part of the peroneal tendons sheath, which covers almost the entire ligament [29].

The CFL becomes horizontal during plantar flexion and vertical in extension and is most taut when the ankle is dorsiflexed [20, 22, 25], acting synergistically with the ATFL, which is under tension during plantar flexion (Fig. 9.3) [8].

Due to its anatomical course, the CFL restricts excessive motion of both the talocrural and subtalar joints [5]. It is widely accepted that the CFL plays a crucial role in stabilizing the subtalar joint [5, 31]. Increased inversion and internal rotation of the hindfoot after section of the calcaneofibular ligament have been shown to primarily take place in the talocalcaneal joint [32].

The biomechanical role of the CFL in the control of talocrural joint kinematics has been also demonstrated. Kerkhoffs et al. showed a constant increment of the anterior talar translation in the sagittal plane, associated with sequential cutting of the CFL after ATFL section [33].

The CFL is the second most injured of the lateral talocrural ligaments, after the ATFL [34].

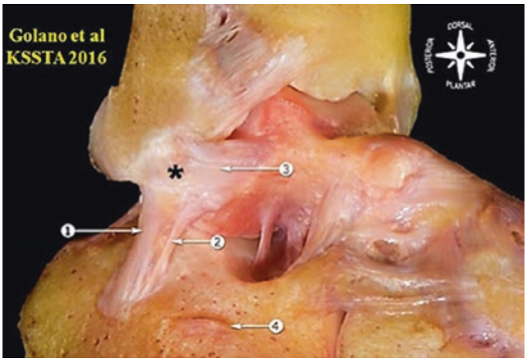


Fig. 9.1 Anatomic view of the lateral ligamentous complex [34]. (1) Calcaneofibular ligament; (3) anterior talofibular ligament; ("asterisk" partial confluency of the fibular insertions of the ATFL and the CFL)

Fig. 9.2 Schematic representation of anatomical lateral ligaments reconstruction with one common tunnel created for the fibular fixation of both the ATFL and CFL

Fig. 9.3 Synergistic action of the ankle lateral ligaments. The ATFL is taut in plantar flexion, whereas the CFL tightens in dorsiflexion [20, 22, 25]

Its injury usually follows the ATFL, depending on the energy and severity of the inversion sprain. In a cadaveric study, Rasmussen noted that the ATFL was always torn before the CFL was torn. Similarly, Broström found no isolated tears of the CFL in 60 patients examined surgically [35, 36].

9.1.2 Chronic Ankle Instability: Treatment Overview

Chronic ankle instability is a complex phenomenon difficult to qualify and quantify, with a recognized multifactorial etiology [37]. It is a consequence of both functional and mechanical factors, among which the posttraumatic ligamentous insufficiency might not always be the primary causative factor [5].

Lower leg proprioceptive deficits, disruption of normal arthrokinetic reflexes, and (peroneal) muscle weakness are frequently observed after an ankle ligament injury and considered major functional contributors to the persistence of the symptoms [5, 38]. Moreover subjective ankle instability can also exist in the absence of ligamentous insufficiency [6, 39, 40].

Based on these observations, a comprehensive rehabilitation program that emphasizes proprioceptive, neuromuscular control and balance training should always be considered as the first line of treatment for chronic ankle instability. Available data report success rates of 80–85% after proper functional ankle rehabilitation programs [8, 41–44].

When nonsurgical measures fail in patients with detectable posttraumatic mechanical ligamentous insufficiency, surgery should be considered in order to restore functional stability [45].

In particular cases, especially in high-demand athletes, surgery can be considered as a first-line treatment to ensure an early return to sport [46, 47].

Many surgical procedures have been suggested in the literature [12]. For several decades, pioneer surgeons proposed ligamentous reconstruction techniques using tendon procedures while sacrificing normal structures around the ankle.

The Evans, Watson-Jones, and Chrisman-Snook are common examples of these procedures, generically referred to as tenodesis [12, 38].

In 1966, Broström argued that these “non-anatomic” reconstructions of the ankle lateral ligaments did not allow a restoration of normal biomechanics and led to an altered function of the talocrural and subtalar joints. Accordingly, he proposed an anatomical repair of the injured ligamentous native tissue, through either direct end-to-end suture of the ligaments or ligamentous reattachment to the anterior fibular margin in case of proximal injuries. Broström reported complete restoration of function in the majority of his patients [35].

The direct anatomic repair of the ATFL and CFL has since gained popularity, and with the addition of the Gould modification, which includes reattachment of the lateral portion of the inferior extensor retinaculum (IER) to the distal fibula, it has become the preferred surgical approach to lateral ankle instability. The functional outcomes have been excellent, with success rates reported as high as 87–95% [35, 48–50].

Compared to anatomical repair, a tenodesis leads to inferior functional and mechanical laxity restoration, as well as overall satisfaction and sport performance [51–54]. This is probably due to the fact that tenodesis does not follow the orientation of the normal ligaments and thereby alters the biomechanics of the ankle complex, particularly at the level of the subtalar joint [53, 54].

A major concern about the anatomic techniques is related to the ability of the ligamentous native tissue to achieve a substantial repair, especially in cases of long-standing ligament insufficiency or generalized joint hypermobility [55]. Remnants of the ruptured ligaments most probably degenerate over time and therefore could be inadequate, because of both a weakness in tensile force and shortening with respect to normal length.

In their original studies, both Broström and Gould [35, 48] found that, even in long-standing instability, there is always some ligamentous tissue to repair, with the eventual addition of the nearby retinaculum [48].

Nevertheless, Karlsson et al. suggested that the ligament repair using native tissue should be used with great care in patients with generalized joint hypermobility and in patients with long-standing ligament insufficiency, associating these features with an increased risk for mechanical failure of the procedure [49, 50, 56].

Currently, the choice to rely on the native tissue in such critical cases remains a surgeon's decision.

In patients whose ligament remnants are inadequate for repair, anatomic reconstruction using a free tendon graft, usually one of the hamstring tendons, has been proposed [11, 57, 58]. These procedures benefit from the established biomechanical advantages over reconstruction tenodesis, with sparing important periarticular structures, namely, the peroneal tendons (Fig. 9.4) [11, 38, 59, 60].

Available clinical data indicate anatomical reconstructions (using free tendons) as a viable option for patients with generalized ligamentous laxity or long-standing ligamentous insufficiency or as a salvage procedure in a patient with a failed Broström-Gould lateral ligament repair [55, 61].

The need for an isolated ATFL or combined ATFL/CFL reconstruction represents another debated issue about anatomic surgical procedures, irrespective of repair or reconstruction.

Ex vivo biomechanical studies have identified different laxity patterns associated with isolated ATFL injury and more severe combined ATFL/CFL injury, respectively [16, 33, 54].

However, in current clinical practice, it can often be difficult to determine whether both the ATFL and the CFL ligaments are injured and whether they need simultaneous repair.

Clinicians frequently diagnose chronic ankle instability using the manual anterior drawer test and stress radiography [62, 63].

However, both physical and radiographic examinations often fail to reveal the extent of ankle laxity (Fig. 9.5) [63, 64].

Several authors have debated the diagnosis of lateral ankle ligament injuries using MRI [65, 66]. Nevertheless, despite the established usefulness of MRI for the evaluation of injuries commonly associated with CLAI, this static

Fig. 9.4 Cadaver specimen showing anatomical ligament ATFL/CFL reconstruction with free hamstring tendon graft

Fig. 9.5 Physical examination. A positive anterior drawer test (ADT) has a sensitivity of 73% and a specificity of 97% for ATFL rupture [62]. "*Asterisk*" the occurrence of a skin dimple when performing the ADT highly correlates with a rupture of the lateral ligaments (predictive value 94%) [62]

examination fails to reveal how unstable the affected ankle is [66, 67].

Clinical data on the subject are controversial. In a series of 60 patients, Karlsson et al. reported better functional results associated with simultaneous ligament reconstruction of both ATFL and the CFL; the authors concluded that if there is any doubt, reconstruction of both ligaments should be performed [50]. Okuda et al. proposed the isolated anatomical reconstruction of the ATFL (with a palmaris longus graft), to treat either combined ATFL/CFL injuries or an isolated ATFL injury [61]. The authors found no significant differences in terms of clinical and radiological outcomes between the two groups and concluded that CFL reconstruction is not necessary, even in patients with combined ATFL/CFL injuries.

Accordingly, Lee et al. reported 94% of good to excellent results in 88 patients suffering from CLAI, treated with isolated ATFL reconstruction and advancement of the inferior retinaculum [68]. In a following ex vivo biomechanical study, the same authors strengthened their clinical results, showing isolated ATFL reconstruction with IER advancement to provide as much initial stability as simultaneous ATFL/CFL reconstruction in cadavers after concomitant section of both ligaments [31].

9.2 State-of-the-Art Treatment

9.2.1 The Role of Arthroscopy in the Treatment of Ankle Instability

Ankle arthroscopy is the gold standard therapy for a variety of pathologies typically associated with ankle instability, such as posttraumatic synovitis, loose bodies, osteochondral lesions of the talus, and osteophytes. Due to the frequency of these concomitant lesions, as high as 95% [45, 61, 69, 70], some researchers suggest a routine arthroscopic exploration of the ankle before any stabilization procedures, in order to address lesions that might affect postoperative outcomes [45, 71].

Ferkel et al. concluded that excellent results can be expected in patients with CLAI, who undergo arthroscopic treatment of associated intra-articular pathology before an open Broström-Gould procedure [45].

Besides the well-established indications, recently, an increasing number of publications describing all-arthroscopic ankle stabilization procedures are found [13, 72, 73]. As for open surgery, reported techniques can be broadly divided in anatomic native tissue repair techniques, with or without local reinforcing using IER, often referred to as arthroscopic Broström-Gould technique and anatomic ligament reconstruction with a free tendon graft. Some available data on arthroscopic CLAI treatment deal with the results associated to the thermal shrinkage of the lateral capsular ligamentous complex [13].

9.2.1.1 Anatomic Local Tissue Repair Techniques

The anatomic repair of the local lateral ligamentous complex is the most extensively studied among arthroscopic techniques [13]. In available literature anatomic repair refers to ATFL repair [74], with, in some cases, additional Gould modification [75–78]. As it is difficult to objectify whether the IER is used during an arthroscopic repair procedure, some studies describe stitching the capsule and IER together over the ATFL as an augmentation [79, 80]. No reports of local tissue repair of the CFL have been reported. One report, from Vega et al, recently reported satisfactory results after combined arthroscopic repair of the ATFL and the anterior fascicle of the deltoid ligament, for patients diagnosed to suffer from a “rotational ankle instability” [81].

The repair of the lateral ligamentous complex is usually achieved through suture anchor fixation of the ATFL to the fibular footprint. Both the number and type of anchors being used vary [74–76, 79, 82–85]. Other than anchors, suture tape [86, 87], stapling, and bone tunnels [13] have been also proposed.

Analysis of the reported surgical techniques reveals some fundamental technical issues:

- Remnants of the lateral ligaments have to be thoroughly dissected. The full course of the ATFL must be visible and accessible (Fig. 9.6). A probe can be used to palpate, tension, and judge the quality of the tissue remnants (Fig. 9.7). Whenever these are inadequate for substantial repair, alternative procedures, i.e., ligament reconstruction, should be considered.
- An extensive debridement of the lateral gutter is mandatory to remove potential impingement tissue and to visualize the anterior distal face of the fibula, with clear definitions of its lateral and medial margin. This step is essential for proper positioning of anchors or bone tunnel (Fig. 9.8). The tip of the lateral malleolus is prepared until bleeding bone is achieved. Two anchors are inserted, to allow anatomical footprint fixation of the ATFL.

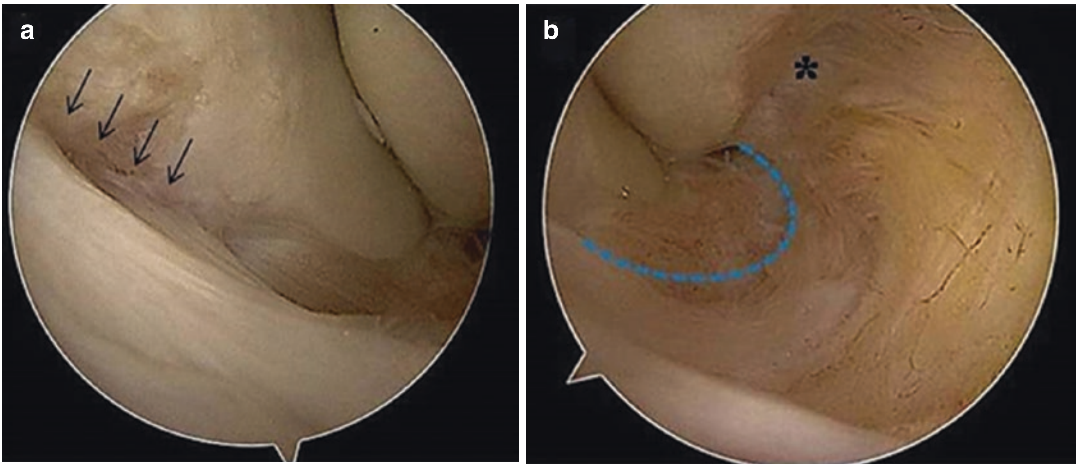


Fig. 9.6 Arthroscopic visualization of the ATFL (left ankle, scope in the AM portal). The ligament is inspected along the entire length: (**a**) ATFL talar insertion. (**b**) Fibular ATFL insertion showing irregularity “asterisk” at the level of the fibular footprint. The blue dotted line indicates the inferior margin of the ATFL

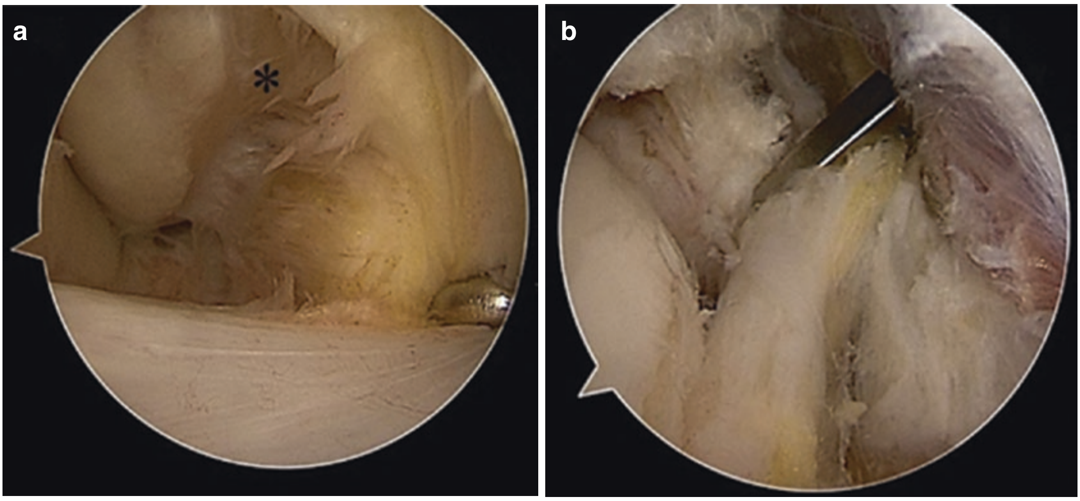


Fig. 9.7 Arthroscopic evaluation of the ATFL (same patient as Fig. 9.6, scope in the AM portal, instrumentation through AL portal). (**a**) Tensioning of the ligament confirms the ATFL fibular detachment “asterisk”. (**b**) The ability of the native ligament tissue to guarantee a substantial anatomical reduction is verified

– Suture passage through ligamentous tissue has to be carefully performed with sharp and thin instrumentations, avoiding multiple perforations to cause iatrogenic damage to the ligament remnants (Figs. 9.9 and 9.10).

Most studies dealing with arthroscopic repair procedures to treat CLAI are of low quality (levels IV and V), according to the criteria described by Wright et al. [13]. A few comparative studies have been published. Yeo et al., in a randomized controlled study, did not find any differences in terms of clinical and radiologic outcomes between the all-inside arthroscopic Broström-Gould technique and the equivalent open procedure, within a 1 year follow-up [83]. Similarly, in a retrospective comparative (level III) study, Matsui et al. noted similar clinical results at 1 year follow-up between arthroscopic and open repair of the ATFL with IER reinforcement, with earlier recovery after surgery in favor of the arthroscopic procedure [88]. Accordingly, some biomechanical observations suggest that there is a similar restoration of biomechanical function in the ankle after arthroscopic and open lateral ligament repairs [89].

Results of arthroscopic repair techniques (2010–2016) have shown good postoperative

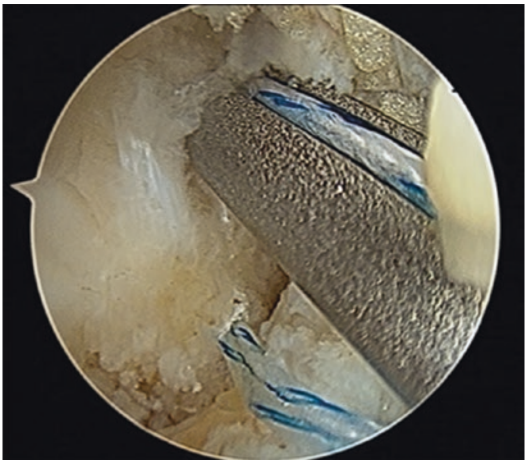


Fig. 9.8 Anchor insertion (left ankle, scope in the AM portal, instrumentation through accessory AL portal)

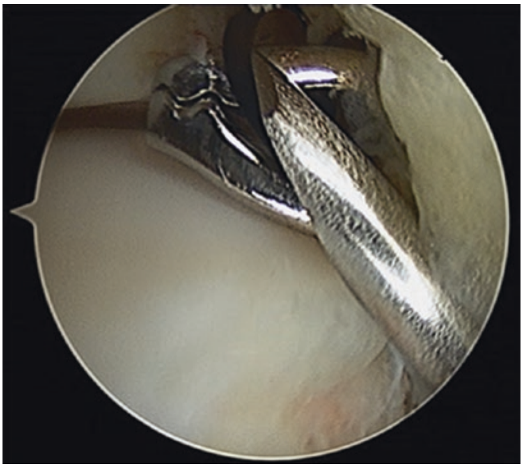


Fig. 9.9 ATFL repair (left ankle, scope in the AM portal, instrumentation through accessory AL and AL portals) An 18-gauge needle trespass the ATFL, bringing a nylon loop used as a suture passer

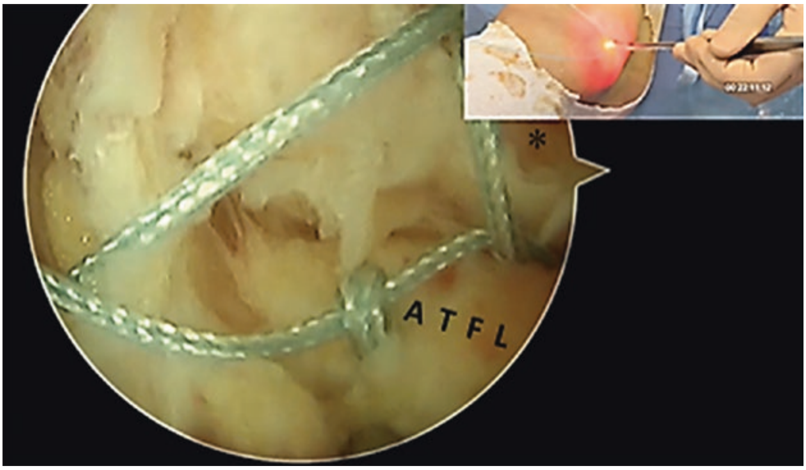


Fig. 9.10 ATFL anatomical repair (right ankle, scope in the AM portal) A suture-lasso technique is used to reduce the ATFL to the fibular footprint (*)

outcomes with a high satisfaction rate (94.5%). Cohort sizes ranged from 16 to 73 patients, with a mean age ranging from 23 to 44 years [13, 77, 79, 85, 86, 90, 91]. Follow-up ranged from 7 to 104 months [77, 79, 82, 85, 86, 90–92]. Outcomes were assessed using the Karlsson score (mean score increase (MSI) 13–61.9 points) [77, 79, 83], AOFAS (MSI 22.8–46.7) [77, 82, 83, 85, 86, 91, 92], Kaikkonen (MSI 45) [82], and VAS (minimal score decrease (MSD) 3.5–7.4) [77, 83].

Some researchers evaluated the mechanical laxity of the arthroscopically stabilized ankle, showing a significant reduction of laxity on stress radiographs [82, 86].

As for other arthroscopic procedures, complications such as infection and delayed wound healing are infrequent [71, 85, 90, 91]. However, due to lateral portal placement, the intermediate branch of the superficial peroneal nerve is at risk for entrapment by suture, occurring in up to 3% of cases [79]. Another complication is knot-related pain and/or asymptomatic prominent knot under the skin, with reported incidences of 0.5–0.8% [80, 85].

9.2.1.2 Anatomic Reconstruction

In terms of arthroscopic reconstruction, current available evidence is mainly limited to technical descriptions of the procedure in in vivo and ex vivo settings [13, 28, 29, 93, 94].

Surgical indications mirror those already proposed for similar open procedures, especially primary repair in patients with major long-standing instability and/or generalized ligamentous laxity, and revision in case of failed previous repair [75, 95, 96].

Both isolated ATFL and combined ATFL/CFL arthroscopic reconstruction have been proposed, with the use of an autologous hamstring graft as the most common choice [13].

The exact locations of the tunnels for graft placement, as well as the safety of the procedure with regard to the surrounding structures, have been analyzed in several anatomical studies [28–30, 97].

These analyses suggest that the arthroscopic equivalent of the well-recognized open procedure is feasible and reproducible across a wide number of surgeons that are experienced with arthroscopic techniques. Further research is needed to better identify the clinical value of the procedure.

At present, the ability to judge the quality of the ligament remnants through direct arthroscopic visualization, with no need to shift during surgery to an open procedure, in case a reconstruction was preferred over a repair, is considered a major advantage of this approach.

9.2.1.3 Capsular Shrinkage

Arthroscopic thermal capsular and ligament shrinkage has been proposed as a treatment for CLAI. By application of radiofrequent energy to the capsular ligamentous tissue, a shrinkage of collagenous structures is induced, resulting in ligamentous tightening.

Data about the shrinkage technique have been published between 2000 and 2012 [98–102], with 4–90 included patients per study. Mean age ranged from 18.1 to 43.1, and the mean follow-up duration ranged from 6 to 48 months. Postoperative outcomes after capsular shrinkage have been evaluated using both clinical and radiological outcome measures. Functional results were reported using the Karlsson score (MSI 26.6–37.2) [98–100], AOFAS score (MSI 25–29.8) [100–102], SF-36 physical (MSI 6.5) [99], Tegner (MSI 1.3–1.6) [99, 100], and Sefton scale (MSD 2.2) [100].

Reported complications in a total of 165 patients were injury of the superficial peroneal nerve leading to numbness (0.6%) or altered sensation (3%), reoperation (2%), tape allergy (1%), ROM restriction (2%), and persistent postoperative pain (0.6) [98–100, 103].

Besides high satisfaction rates and low complication rates, detailed biomechanical analysis from de Vries et al. [99] reported the technique achieved only a moderate reduction of the joint laxity. The researchers concluded that the functional improvement associated with arthroscopic shrinkage could be related to an improved proprioception and ankle coordination, caused by the debridement of the synovial tissue in the anterolateral joint gutter. In this regard, the relation between proprioception and functional ankle stability, with or without mechanical laxity in general, has been pointed out by several researchers [104–106].

Since de Vries' publication in 2008, only one report has been published related to CLAI treatment by arthroscopic shrinkage [100], and available data failed to clarify the best indications for this procedure.

9.3 Future Treatment Options

The current developing arthroscopic approach in the treatment of ankle instability mirrors the processes witnessed for the knee and shoulder during the last 40 years. Initially, stabilization procedures were performed in an open manner with non-anatomic methods applied externally to restrain abnormal motion. Such methods were followed by arthroscopic examination, followed by an open procedure. Finally, all-arthroscopic stabilization procedures have become the current standard of care for the knee and the shoulder [107, 108].

For many reasons, arthroscopic stabilization procedures represent an attractive option. First and foremost, there is a potential to lower morbidity and accelerate recovery, which is a characteristic for arthroscopic approaches in general [83, 88, 109]. Moreover, given the high incidence of associated intra-articular lesions, an arthroscopic approach enables the surgeon to address both intra-articular pathology and pathological laxity simultaneously through a single approach [45, 110–112].

Multiple studies that reveal equivalent clinical and biomechanical results for both traditional open and arthroscopic modified Broström ligament repair/reconstruction have been published. Therefore, evidence supporting the arthroscopic approach for CLAI treatment as a viable alternative to traditional open techniques is mounting [79].

In addition to encouraging clinical results, the arthroscopic approach to ankle instability offers a new insight, potentially able to provide improved knowledge of the intra-articular pathology.

The unique perspective of the pathoanatomical features of ligamentous structures of the ankle offered by arthroscopy has been advocated by several researchers.

Hintermann et al. highlighted that preoperative ankle arthroscopy allows precise identification of ligamentous abnormalities, both medially and laterally, corresponding with different entities of ankle instability [111].

Other researchers have underscored the ability of arthroscopy to depict subclinical pathological laxity, which is not always detectable in the diagnostic setting. In these patients, the arthroscopically diagnosed ATFL abnormalities have been associated with chronic anterior ankle pain and functional instability, proposing new clinical definitions referred to as microinstability or apparent instability of the ankle [113, 114].

These observations indicate a potential role for arthroscopy in the future definition of specific entities on the broad spectrum of ankle instability, able to clarify current unexplained clinical pictures.

The sharp direct evaluation of ligamentous structures permitted by arthroscopy might also be helpful to evaluate the quality of the ligament remnants [114], which still remains a critical issue, especially in demanding patients with long-standing CLAI and/or generalized ligamentous laxity. Due to the therapeutic consequences, the definition of objective criteria able to predict the ability of the endogenous tissue to achieve a substantial repair represents a primary purpose for orthopedic surgeons dealing with CLAI and warrants further research.

In this regard, Bauer et al. recently reported a retrospective analysis of preoperative ankle MRI, based on the arthroscopic evaluation of the lateral ligamentous complex state. On the basis of their observations, the authors defined MRI as a reliable and reproducible tool to judge the quality of the ligament remnants, allowing the preoperative decision as to whether a local tissue repair or a reconstruction procedure should be performed [115].

The arthroscopic accessibility of the CFL represents a potential weak point in the arthroscopic treatment of CLAI, due to the extracapsular nature of this ligament [111, 116].

Hintermann et al. reported that abnormalities of the CFL are difficult to appreciate arthroscopically from the anterior aspect of the joint because the ATFL and the capsular structures obstruct the view [111].

In a recent anatomical study, Thes et al. confirmed that the CFL can be visualized arthroscopically only after complete dissection of the ATFL [116].

Therefore, it is not surprising that current available data on arthroscopic reparative techniques only deal with isolated ATFL repair with occasional reinforcing by IER [13].

These observations raise the need for future clarifications about the ability of arthroscopy to guarantee an effective ankle stabilization for patients in which a simultaneous anatomic ATFL/CFL repair could be advisable.

However, as previously discussed, controversy still exists about the clinical benefits of a CFL ligament repair/reconstruction.

Several anatomic observations on lateral ligamentous complex have clearly demonstrated an anatomical confluence of the ATFL and CFL fibular insertions [27, 28]. In light of these findings, it could be argued that a wide fibular ATFL footprint reestablishment could lead to concomitant tightening of an insufficient CFL.

Nevertheless, until the development of a reliable clinical tool, able to measure ankle laxity and to associate it to the extent of the ligamentous injury, it will be difficult to improve and standardize current operative indications for CLAI, and to define possible contraindications for arthroscopic procedures.

9.4 Take-Home Messages

- Functional ankle instability is a multifactorial condition in which the posttraumatic ligamentous insufficiency—mechanical laxity—is not always the primary etiological factor.
- Arthroscopy offers a promising alternative for the treatment of chronic lateral ankle instability. Available outcomes are at least equal to published results for similar open techniques, with the added benefit of reduced invasiveness and surgical morbidity that characterize arthroscopic procedures.
- The inherent ability to evaluate the pathoanatomical features of the ligamentous structures by arthroscopy offers new insights into the pathology of chronic ankle instability, with potential improvement of the clinical understanding of this injury, as well as in therapeutic choices.
- Controversy about operative treatment of CLAI remains as to whether to repair or reconstruct the lateral ligamentous complex and whether the ATFL should be repaired alone or in combination with the CFL.
- The development of a reliable clinical tool able to define the entity of the ankle laxity could furnish a major future advancement in the treatment of ankle instability.

References

1. van Dijk CN. Enkelletsels (acuut). In: Het sportmedisch formularium, een praktische leidraad. Uitgever: Bohn Stafleu van Loghum; 2005. p. 97–116.
2. Garrick JG. The frequency of injury, mechanism of injury, and epidemiology of ankle sprains. Am J Sports Med. 1977;5(6):241–2.
3. Kannus P, Renstrom P. Treatment for acute tears of the lateral ligaments of the ankle. Operation, cast, or early controlled mobilization. J Bone Joint Surg Am. 1991;73(2):305–12.
4. Ekstrand J, Tropp H. The incidence of ankle sprains in soccer. Foot Ankle. 1990;11(1):41–4.
5. Hertel J. Functional anatomy, pathomechanics, and pathophysiology of lateral ankle instability. J Athl Train. 2002;37(4):364–75.
6. Brostrom L. Sprained ankles. I. Anatomic lesions in recent sprains. Acta Chir Scand. 1964;128:483–95.
7. Brostrom L. Sprained ankles. V. Treatment and prognosis in recent ligament ruptures. Acta Chir Scand. 1966;132(5):537–50.
8. Baumhauer JF, O'Brien T. Surgical considerations in the treatment of ankle instability. J Athl Train. 2002;37(4):458–62.
9. Lynch SA. Assessment of the injured ankle in the athlete. J Athl Train. 2002;37(4):406–12.
10. de Vries JS, et al. Interventions for treating chronic ankle instability. Cochrane Database Syst Rev. 2011;8:CD004124.
11. Takao M, et al. Anatomical reconstruction of the lateral ligaments of the ankle with a gracilis autograft: a new technique using an interference fit anchoring system. Am J Sports Med. 2005;33(6):814–23.

12. Di Matteo B, et al. A historical perspective on ankle ligaments reconstructive surgery. Knee Surg Sports Traumatol Arthrosc. 2016;24(4):971–7.
13. Matsui K, et al. Minimally invasive surgical treatment for chronic ankle instability: a systematic review. Knee Surg Sports Traumatol Arthrosc. 2016;24(4):1040–8.
14. Acevedo JI, Mangone P. Arthroscopic Brostrom technique. Foot Ankle Int. 2015;36(4):465–73.
15. Griffith JF, Brockwell J. Diagnosis and imaging of ankle instability. Foot Ankle Clin. 2006;11(3):475–96.
16. Watanabe K, et al. The role of ankle ligaments and articular geometry in stabilizing the ankle. Clin Biomech. 2012;27(2):189–95.
17. Burks RT, Morgan J. Anatomy of the lateral ankle ligaments. Am J Sports Med. 1994;22(1):72–7.
18. Attarian DE, et al. Biomechanical characteristics of human ankle ligaments. Foot Ankle. 1985;6(2):54–8.
19. Holmer P, et al. Epidemiology of sprains in the lateral ankle and foot. Foot Ankle Int. 1994;15(2):72–4.
20. Stormont DM, et al. Stability of the loaded ankle. Relation between articular restraint and primary and secondary static restraints. Am J Sports Med. 1985;13(5):295–300.
21. Renstrom P, et al. Strain in the lateral ligaments of the ankle. Foot Ankle. 1988;9(2):59–63.
22. Hollis JM, Blasier RD, Flahiff CM. Simulated lateral ankle ligamentous injury. Change in ankle stability. Am J Sports Med. 1995;23(6):672–7.
23. Stephens MM, Sammarco GJ. The stabilizing role of the lateral ligament complex around the ankle and subtalar joints. Foot Ankle. 1992;13(3):130–6.
24. Cass JR, Settles H. Ankle instability: in vitro kinematics in response to axial load. Foot Ankle Int. 1994;15(3):134–40.
25. Kjaersgaard-Andersen P, et al. The stabilizing effect of the ligamentous structures in the sinus and canalis tarsi on movements in the hindfoot. An experimental study. Am J Sports Med. 1988;16(5):512–6.
26. Hintermann B. Biomechanics of the unstable ankle joint and clinical implications. Med Sci Sports Exerc. 1999;31(7 Suppl):S459–69.
27. Golano P, et al. Anatomy of the ankle ligaments: a pictorial essay. Knee Surg Sports Traumatol Arthrosc. 2010;18(5):557–69.
28. Michels F, et al. Endoscopic reconstruction of CFL and the ATFL with a gracilis graft: a cadaveric study. Knee Surg Sports Traumatol Arthrosc. 2016;24(4):1007–14.
29. Matsui K, et al. Anatomy of anterior talofibular ligament and calcaneofibular ligament for minimally invasive surgery: a systematic review. Knee Surg Sports Traumatol Arthrosc. 2016;25:1892–902.
30. Lopes R, et al. Anatomic validation of the lateral malleolus as a cutaneous marker for the distal insertion of the calcaneofibular ligament. Knee Surg Sports Traumatol Arthrosc. 2016. https://doi.org/10.1007/s00167-016-4250-7.
31. Lee KT, et al. Biomechanical evaluation against calcaneofibular ligament repair in the Brostrom procedure: a cadaveric study. Knee Surg Sports Traumatol Arthrosc. 2008;16(8):781–6.
32. Kjaersgaard-Andersen P, Wethelund JO, Nielsen S. Lateral talocalcaneal instability following section of the calcaneofibular ligament: a kinesiologic study. Foot Ankle. 1987;7(6):355–61.
33. Kerkhoffs GM, et al. Anterior lateral ankle ligament damage and anterior talocrural-joint laxity: an overview of the in vitro reports in literature. Clin Biomech. 2001;16(8):635–43.
34. Renstrom PA, Konradsen L. Ankle ligament injuries. Br J Sports Med. 1997;31(1):11–20.
35. Brostrom L. Sprained ankles. VI. Surgical treatment of "chronic" ligament ruptures. Acta Chir Scand. 1966;132(5):551–65.
36. Rasmussen O. Stability of the ankle joint. Analysis of the function and traumatology of the ankle ligaments. Acta Orthop Scand Suppl. 1985;211:1–75.
37. Kaminski TW, Hartsell HD. Factors contributing to chronic ankle instability: a strength perspective. J Athl Train. 2002;37(4):394–405.
38. Rosenbaum D, Bertsch C, Claes LE. Tenodeses do not fully restore ankle joint loading characteristics: a biomechanical in vitro investigation in the hind foot. Chid Biomech. 1997;12(3):202–9.
39. Milgrom C, et al. Risk factors for lateral ankle sprain: a prospective study among military recruits. Foot Ankle. 1991;12(1):26–30.
40. McKay GD, et al. Ankle injuries in basketball: injury rate and risk factors. Br J Sports Med. 2001;35(2):103–8.
41. Holme E, et al. The effect of supervised rehabilitation on strength, postural sway, position sense and re-injury risk after acute ankle ligament sprain. Scand J Med Sci Sports. 1999;9(2):104–9.
42. Verhagen EA, van Mechelen W, de Vente W. The effect of preventive measures on the incidence of ankle sprains. Clin J Sport Med. 2000;10(4):291–6.
43. Wester JU, et al. Wobble board training after partial sprains of the lateral ligaments of the ankle: a prospective randomized study. J Orthop Sports Phys Ther. 1996;23(5):332–6.
44. Kerkhoffs GM, et al. Diagnosis, treatment and prevention of ankle sprains: an evidence-based clinical guideline. Br J Sports Med. 2012;46(12):854–60.
45. Ferkel RD, Chams RN. Chronic lateral instability: arthroscopic findings and long-term results. Foot Ankle Int. 2007;28(1):24–31.
46. Kerkhoffs GM, et al. Surgical versus conservative treatment for acute injuries of the lateral ligament complex of the ankle in adults. Cochrane Database Syst Rev. 2007;2:Cd000380.
47. Kerkhoffs GM, Van Dijk CN. Acute lateral ankle ligament ruptures in the athlete: the role of surgery. Foot Ankle Clin. 2013;18(2):215–8.
48. Gould N, Seligson D, Gassman J. Early and late repair of lateral ligament of the ankle. Foot Ankle. 1980;1(2):84–9.

49. Karlsson J, et al. Reconstruction of the lateral ligaments of the ankle for chronic lateral instability. J Bone Joint Surg Am. 1988;70(4):581–8.
50. Karlsson J, et al. Surgical treatment of chronic lateral instability of the ankle joint. A new procedure. Am J Sports Med. 1989;17(2):268–73; discussion 273–4
51. Krips R, et al. Anatomical reconstruction versus tenodesis for the treatment of chronic anterolateral instability of the ankle joint: a 2- to 10-year follow-up, multicenter study. Knee Surg Sports Traumatol Arthrosc. 2000;8(3):173–9.
52. Krips R, et al. Anatomical reconstruction and Evans tenodesis of the lateral ligaments of the ankle. Clinical and radiological findings after follow-up for 15 to 30 years. J Bone Joint Surg Br. 2002;84(2):232–6.
53. van der Rijt AJ, Evans GA. The long-term results of Watson-Jones tenodesis. J Bone Joint Surg Br. 1984;66(3):371–5.
54. Fujii T, et al. Comparison of modified Brostrom and Evans procedures in simulated lateral ankle injury. Med Sci Sports Exerc. 2006;38(6):1025–31.
55. Sammarco VJ. Complications of lateral ankle ligament reconstruction. Clin Orthop Relat Res. 2001; 391:123–32.
56. Karlsson J, et al. Comparison of two anatomic reconstructions for chronic lateral instability of the ankle joint. Am J Sports Med. 1997;25(1):48–53.
57. Elmslie RC. Recurrent subluxation of the ankle-joint. Ann Surg. 1934;100(2):364–7.
58. Coughlin MJ, et al. Comprehensive reconstruction of the lateral ankle for chronic instability using a free gracilis graft. Foot Ankle Int. 2004;25(4):231–41.
59. Bahr R, et al. Biomechanics of ankle ligament reconstruction. An in vitro comparison of the Brostrom repair, Watson-Jones reconstruction, and a new anatomic reconstruction technique. Am J Sports Med. 1997;25(4):424–32.
60. Coughlin MJ, Matt V, Schenck RC Jr. Augmented lateral ankle reconstruction using a free gracilis graft. Orthopedics. 2002;25(1):31–5.
61. Okuda R, et al. Reconstruction for chronic lateral ankle instability using the palmaris longus tendon: is reconstruction of the calcaneofibular ligament necessary? Foot Ankle Int. 1999;20(11):714–20.
62. van Dijk CN. Management of the sprained ankle. Br J Sports Med. 2002;36(2):83–4.
63. Cho JH, et al. Value of stress ultrasound for the diagnosis of chronic ankle instability compared to manual anterior drawer test, stress radiography, magnetic resonance imaging, and arthroscopy. Knee Surg Sports Traumatol Arthrosc. 2015;24:1022–8.
64. Rijke AM. MR imaging versus stress radiography in the evaluation of chronic ankle instability. Radiology. 1995;196(2):580–1.
65. Dimmick S, Kennedy D, Daunt N. Evaluation of thickness and appearance of anterior talofibular and calcaneofibular ligaments in normal versus abnormal ankles with MRI. J Med Imaging Radiat Oncol. 2008;52(6):559–63.
66. Schneck CD, Mesgarzadeh M, Bonakdarpour A. MR imaging of the most commonly injured ankle ligaments. Part II. Ligament injuries. Radiology. 1992;184(2):507–12.
67. Alparslan L, Chiodo CP. Lateral ankle instability: MR imaging of associated injuries and surgical treatment procedures. Semin Musculoskelet Radiol. 2008;12(4):346–58.
68. Lee K, et al. Modified Brostrom procedure without calcaneofibular ligament reconstruction for chronic ankle instability. J Korean Sports Med. 2002;20:209–14.
69. DiGiovanni BF, et al. Associated injuries found in chronic lateral ankle instability. Foot Ankle Int. 2000;21(10):809–15.
70. Komenda GA, Ferkel RD. Arthroscopic findings associated with the unstable ankle. Foot Ankle Int. 1999;20(11):708–13.
71. Cottom JM, Rigby RB. The “all inside” arthroscopic Brostrom procedure: a prospective study of 40 consecutive patients. J Foot Ankle Surg. 2013;52(5):568–74.
72. Wang J, et al. Arthroscopic repair of lateral ankle ligament complex by suture anchor. Arthroscopy. 2014;30(6):766–73.
73. Lee KT, et al. All-inside arthroscopic modified Brostrom operation for chronic ankle instability: a biomechanical study. Knee Surg Sports Traumatol Arthrosc. 2016;24(4):1096–100.
74. Takao M, et al. Arthroscopic anterior talofibular ligament repair for lateral instability of the ankle. Knee Surg Sports Traumatol Arthrosc. 2015;24: 1003–6.
75. Pereira H, et al. Arthroscopic repair of ankle instability with all-soft knotless anchors. Arthrosc Tech. 2016;5:e99–e107.
76. Cottom JM, Richardson PE. The “all-inside” arthroscopic Brostrom procedure augmented with a proximal suture anchor: an innovative technique. J Foot Ankle Surg. 2017;56:408–11.
77. Cottom JM, Baker JS, Richardson PE. The “all-inside” arthroscopic Brostrom procedure with additional suture anchor augmentation: a prospective study of 45 consecutive patients. J Foot Ankle Surg. 2016;55(6):1223–8.
78. Lui TH. Modified arthroscopic Brostrom procedure. Foot Ankle Surg. 2015;21(3):216–9.
79. Acevedo JI, Mangone P. Ankle instability and arthroscopic lateral ligament repair. Foot Ankle Clin. 2015;20(1):59–69.
80. Lui TH. Modified arthroscopic Brostrom procedure with bone tunnels. Arthrosc Tech. 2016;5(4):e775–80.
81. Vega J, Allmendinger J, Malagelada F, Guelfi M, Dalmau-Pastor M. Combined arthroscopic all-inside repair of lateral and medial ankle ligaments is an effective treatment for rotational ankle instability. Knee Surg Sports Traumatol Arthrosc. 2017. https://doi.org/10.1007/s00167-017-4736-y.
82. Maffulli N, et al. Isolated anterior talofibular ligament Brostrom repair for chronic lateral ankle instability:

9-year follow-up. Am J Sports Med. 2013;41(4):858–64.

83. Yeo ED, et al. Comparison of all-inside arthroscopic and open techniques for the modified Brostrom procedure for ankle instability. Foot Ankle Int. 2016;37(10):1037–45.
84. Matsui K, et al. Arthroscopic Brostrom repair with Gould augmentation via an accessory anterolateral port for lateral instability of the ankle. Arch Orthop Trauma Surg. 2014;134(10):1461–7.
85. Kim ES, et al. Arthroscopic anterior talofibular ligament repair for chronic ankle instability with a suture anchor technique. Orthopedics. 2011;34(4). https://doi.org/10.3928/01477447-20110228-03.
86. Yoo JS, Yang EA. Clinical results of an arthroscopic modified Brostrom operation with and without an internal brace. J Orthop Traumatol. 2016;17(4):353–60.
87. Prissel MA, Roukis TS. All-inside, anatomical lateral ankle stabilization for revision and complex primary lateral ankle stabilization: a technique guide. Foot Ankle Spec. 2014;7(6):484–91.
88. Matsui K, et al. Early recovery after arthroscopic repair compared to open repair of the anterior talofibular ligament for lateral instability of the ankle. Arch Orthop Trauma Surg. 2015;136(1):93–100.
89. Drakos MC, et al. Biomechanical comparison of an open vs arthroscopic approach for lateral ankle instability. Foot Ankle Int. 2014;35(8):809–15.
90. Corte-Real NM, Moreira RM. Arthroscopic repair of chronic lateral ankle instability. Foot Ankle Int. 2009;30(3):213–7.
91. Vega J, et al. All-inside arthroscopic lateral collateral ligament repair for ankle instability with a knotless suture anchor technique. Foot Ankle Int. 2013;34(12):1701–9.
92. Arroyo-Hernandez M, et al. Chronic ankle instability: arthroscopic anatomical repair. Rev Esp Cir Ortop Traumatol. 2016;61:104–10.
93. Guillo S, et al. Arthroscopic anatomical reconstruction of the lateral ankle ligaments. Knee Surg Sports Traumatol Arthrosc. 2016;24(4):998–1002.
94. Guillo S, et al. Consensus in chronic ankle instability: aetiology, assessment, surgical indications and place for arthroscopy. Orthop Traumatol Surg Res. 2013;99(8 Suppl):S411–9.
95. Ferran NA, Oliva F, Maffulli N. Ankle instability. Sports Med Arthrosc. 2009;17(2):139–45.
96. Guillo S, et al. Arthroscopic anatomical reconstruction of the lateral ankle ligaments. Knee Surg Sports Traumatol Arthrosc. 2015;24:998–1002.
97. Takao M, et al. Ankle arthroscopic reconstruction of lateral ligaments (ankle anti-ROLL). Arthrosc Tech. 2015;4(5):e595–600.
98. Maiotti M, Massoni C, Tarantino U. The use of arthroscopic thermal shrinkage to treat chronic lateral ankle instability in young athletes. Arthroscopy. 2005;21(6):751–7.
99. de Vries JS, et al. Arthroscopic capsular shrinkage for chronic ankle instability with thermal radiofrequency: prospective multicenter trial. Orthopedics. 2008;31(7):655.
100. Ventura A, et al. Arthroscopic four-step treatment for chronic ankle instability. Foot Ankle Int. 2012;33(1):29–36.
101. Hyer CF, Vancourt R. Arthroscopic repair of lateral ankle instability by using the thermal-assisted capsular shift procedure: a review of 4 cases. J Foot Ankle Surg. 2004;43(2):104–9.
102. Oloff LM, Bocko AP, Fanton G. Arthroscopic monopolar radiofrequency thermal stabilization for chronic lateral ankle instability: a preliminary report on 10 cases. J Foot Ankle Surg. 2000;39(3):144–53.
103. Berlet GC, et al. Thermal-assisted capsular modification for functional ankle instability. Foot Ankle Clin. 2002;7(3):567–76. ix
104. Hintermann B, Renggli P. Anatomic reconstruction of the lateral ligaments of the ankle using a plantaris tendon graft in the treatment of chronic ankle joint instability. Orthopade. 1999;28(9):778–84.
105. Jerosch J, et al. The influence of orthoses on the proprioception of the ankle joint. Knee Surg Sports Traumatol Arthrosc. 1995;3(1):39–46.
106. Hertel J. Functional instability following lateral ankle sprain. Sports Med. 2000;29(5):361–71.
107. Godin J, Sekiya JK. Systematic review of arthroscopic versus open repair for recurrent anterior shoulder dislocations. Sports Health. 2011;3(4):396–404.
108. Sedeek S, Gerard E, Andrew H. Evolution of arthroscopic shoulder stabilization: do we still need open techniques? OA Orthopaedics. 2013;1(1):1.
109. van Dijk CN, van Bergen CJ. Advancements in ankle arthroscopy. J Am Acad Orthop Surg. 2008;16(11):635–46.
110. Nery C, et al. Arthroscopic-assisted Brostrom-Gould for chronic ankle instability: a long-term follow-up. Am J Sports Med. 2011;39(11):2381–8.
111. Hintermann B, Boss A, Schafer D. Arthroscopic findings in patients with chronic ankle instability. Am J Sports Med. 2002;30(3):402–9.
112. Okuda R, et al. Arthroscopic findings in chronic lateral ankle instability: do focal chondral lesions influence the results of ligament reconstruction? Am J Sports Med. 2005;33(1):35–42.
113. Vega J, Pena F, Golano P. Minor or occult ankle instability as a cause of anterolateral pain after ankle sprain. Knee Surg Sports Traumatol Arthrosc. 2014;24:1116–23.
114. Takao M, et al. Arthroscopic and magnetic resonance image appearance and reconstruction of the anterior talofibular ligament in cases of apparent functional ankle instability. Am J Sports Med. 2008;36(8):1542–7.
115. Morvan A, et al. Fiabilité et validité de l'IRM préopératoire pour la prise de décision chirurgicale dans les instabilités latérales chroniques de cheville. Rev Chir Orthopéd Traumatol. 2016;102(8):S55–6.
116. Thes A, et al. Assessment of the feasibility of arthroscopic visualization of the lateral ligament of the ankle: a cadaveric study. Knee Surg Sports Traumatol Arthrosc. 2016;24(4):985–90.

Combined Meniscus and Cartilage Lesions

10

Peter Angele, Michael T. Hirschmann,
Sebastian Kopf, Henning Madry,
Philipp Niemeyer, and Peter Verdonk

10.1 Introduction

In the last decades, orthopaedic surgery has become increasingly specialised. The generalist orthopaedic surgeon, who is able to treat hip, knee, shoulder and ankle problems, has become a rare species. Most orthopaedic surgeons nowadays concentrate their activity on one or two joints, and even within the knee joint, there are surgeons who only focus on reconstructive or arthroplasty surgery. There is no doubt that the trend of specialisation has led to higher quality and more focused diagnostic and treatment strategies. However, there are backsides of this medal in particular in fields where different joint or treatment options could be involved.

It appears that it is in reconstructive knee surgery that we sometimes do not see the wood for the trees. It is not just fixing the meniscus or cartilage lesion. It is mandatory to see the whole picture, instead of just the meniscus or cartilage repair. The intimate connections between the menisci and the osteochondral unit of the femorotibial compartment are based on their specific anatomical structure. Understanding the underlying structural and anatomical basis helps to understand the causes why meniscal lesions (and meniscus extrusion) may lead to cartilage loss and why cartilage loss may also induce meniscal damage. In turn, the reconstructive surgical consequences are also based on these topographical correlations.

P. Angele (✉)
University Medical Center Regensburg,
Regensburg, Germany
e-mail: peter.angele@ukr.de

M.T. Hirschmann
Department of Orthopaedic Surgery and
Traumatology, Kantonsspital Baselland (Bruderholz,
Liestal, Laufen), Bruderholz, Switzerland
e-mail: michael.hirschmann@ksbl.ch,
michael.hirschmann@unibas.ch

S. Kopf
Center for Musculoskeletal Surgery, Charité –
University Medicine Berlin, Berlin, Germany
e-mail: mail@koepfchen.org

H. Madry
Center of Experimental Orthopaedics,
Saarland University, Homburg, Germany

Department of Orthopaedic Surgery, Saarland
University Medical Center, Homburg, Germany
e-mail: henning.madry@uks.eu

P. Niemeyer
OCM Clinic, Munich, Germany
e-mail: phniemeyer@gmail.com

P. Verdonk
Antwerp Orthopedic Center, Monica Hospitals,
Antwerp, Belgium
e-mail: pverdonk@yahoo.com

G.M.M.J. Kerkhoffs et al. (eds.), *ESSKA Instructional Course Lecture Book*,
https://doi.org/10.1007/978-3-662-56127-0_10

10.2 State-of-the-Art Treatment

10.2.1 Meniscus–Cartilage Unit

Both menisci are composed of fibrocartilage. They improve the congruency between the osteochondral units of the femur and the tibia. Both menisci are connected through their meniscal roots with the central tibial plateau. These roots, together with the menisci, may be affected during osteoarthritis (OA). The volume of the medial meniscus is significantly smaller than that of the lateral meniscus. In cross section, both menisci are wedge-shaped, and their surface facing the femur is concave, while the surface facing the tibial plateau is even [1, 2].

From a topographic standpoint, both tibial plateaus may be subdivided into a central (not covered by meniscus) and a peripheral (covered by meniscus) part. Interestingly, the human submeniscal articular cartilage in the peripheral tibial plateau is about 50% thinner than the centrally located cartilage, which is not covered by the meniscus. It has also different biomechanical properties [3]. Correspondingly, the subchondral bone plate is also characterised by similar differences [4]. Like the tibial plateaus, both femoral condyles articulate with the menisci. During knee flexion, both menisci adapt their form to the femoral condyles and move posteriorly [5].

Both menisci are protecting the peripheral articular cartilage of the tibial plateaus. Large animal studies have shown that spontaneous development of osteoarthritis only takes place in the central, not meniscus-covered tibial plateau [6]. However, the development of osteoarthritis in the peripheral, meniscus-covered tibial plateau correlates significantly with early degenerative changes of the meniscus [7]. Interestingly, osteoarthritis of the entire tibial plateau correlates with alterations of the microstructure of the subchondral bone plate [7].

10.2.2 Hierarchy of Treatment

There is a clear hierarchy for reconstructive treatment, which should be considered in every patient [8]. The highest priority is to achieve a balanced mechanical leg axis, followed by normal ligament laxity [8]. Thirdly, as much meniscus tissue as possible needs to be preserved or restored as it acts as shock absorber. All these aforementioned factors are a conditio sine qua non for successful cartilage repair [8].

10.2.2.1 Alignment

Correction of malalignment is the first priority in reconstructive knee surgery [8]. Mechanical alignment determines the loading distribution within the knee joint [9].

In a varus knee, about 70–90% of the joint loading runs through the medial compartment [9]. In these cases, addressing the cartilage and/or meniscus lesions alone will not result satisfying and good functional outcomes.

In a valgus knee, the loading is predominantly distributed through the lateral compartment. In these cases again, a pure meniscus and/or cartilage treatment is prone for failure. Clearly, the underlying pathology in these cases is mechanical malalignment [10]. The underlying cause of the problem needs to be addressed by an osteotomy [10]. In all cases with malalignment, correction of the alignment using an osteotomy is necessary to achieve good functional results [10].

10.2.2.2 Stability

Treatment of instability is the second priority in reconstructive knee surgery [8].

The anterior cruciate ligament (ACL) is the primary restraint against anterior tibial translation in relation to the femur [11]. The secondary role of the ACL is to resist internal tibial rotation, which is most pronounced in knee extension [11]. The ACL lets the tibia internally rotate during anterior tibial translation [11].

In an ACL-deficient knee, the meniscus (as secondary restraint for anterior tibial translation) is exposed to higher stresses leading to a higher likelihood of meniscal lesions [11]. In addition, the cartilage is also exposed to higher loads, which in midterm might result in cartilage lesions. In ACL-deficient patients with already existing meniscal or cartilage lesions, this effect also influences healing and restoration after surgical treatment, and hence a torn ACL needs to be repaired or reconstructed.

The previously described relationship of instability and healing of meniscal and chondral lesions is also true for the posterior cruciate ligament and collaterals. Actually all clinically relevant ligament tears including the periphery need to be addressed [12].

10.2.3 Meniscus Treatment Depending on Adjacent Cartilage

Treatment of meniscal pathologies is the third priority in reconstructive knee surgery [8]. In the early years of meniscal surgery, it was the gold standard to entirely excise the injured meniscus [13]. Since then, after recognising that a total or subtotal meniscectomy inevitably leads to development of osteoarthritis within 5–10 years after surgery, it was advocated that as much meniscus tissue as possible should be preserved [14]. Only the meniscus tissue which is identified as unrepairable should be excised [14]. The recommended strategy for treatment of meniscal lesions is to “preserve as much meniscus as possible”.

Meniscus lesions are divided into traumatic meniscus tears and degenerative meniscus lesions. Traumatic tears are associated with an adequate knee injury and demonstrate most likely a vertical tear pattern. They occur in rather younger patients, mainly without osteoarthritic changes. On the other hand, degenerative meniscus lesions develop over time without an adequate trauma, are rather found in elderly people and do not have to provoke symptoms. For example, in 50–59-year-old asymptomatic men, about 30 % have a meniscus lesion. These numbers increase up to about 50% in 70–90-year-old men. Tear patterns are mainly horizontal. Degenerative tears often occur with degenerative changes of the entire knee including the cartilage.

10.2.4 State-of-the-Art-Treatment

Traumatic meniscus tears should be repaired if possible. In case of a concomitant ACL reconstruction and a short tear of the lateral meniscus, it might be left alone. Partial or even total meniscectomy should only be performed as a salvage procedure. Meniscus repair is especially important in case of treatable focal chondral lesions. For osteoarthritic knees, found on a regular basis in multi-revision ligament reconstructed knees, there are no studies. Thus, it is discretionary of the surgeon. However, if the ligament is reconstructed, the meniscus should also be repaired. In case of a subtotal loss of the meniscus but with an intact rim, a partial meniscus replacement should be considered, which decreases the progression of osteoarthritic changes. Published indications suggest that cartilage defects should be <2° after the score of the International Cartilage Repair Society (ICRS). In case of a functional total loss of a meniscus, meniscus transplantation is a great option. Published indications suggest also that cartilage defects should be <2° after the score of the International Cartilage Repair Society (ICRS). However, OA changes progress.

Degenerative meniscus tears should initially be treated conservatively. A recent ESSKA consensus suggested a duration of 3 months. If this treatment fails, arthroscopic partial meniscectomy should be performed even in the knees with osteoarthritic changes. However, patients should be informed that the success rate decreases with increasing degenerative changes of the knee. It is important to check the limb alignment and treat it if appropriate. A WOMAC score with >40 points was identified as a predictor for failing conservative treatment and early arthroscopic partial meniscectomy [15].

10.2.5 Cartilage Treatment Depending on Adjacent Meniscus

Treatment of cartilage lesions is the fourth priority in reconstructive knee surgery [8]. It is important to realise that cartilage repair does nothing else than treating the surface. In some cases cartilage repair alone is able to solve a problem, but only if nothing else than the cartilage lesion was causing the symptoms of the patient. In most cases, a cartilage lesion is just the tip of the iceberg. It is of utmost importance to understand the mechanism of injury and knee pathology which led to the cartilage lesion.

While initial cartilage repair techniques such as autologous chondrocyte implantation (ACI), osteochondral transplantation or microfracturing have been introduced for the treatment of traumatic cartilage defects [16], it is questionable how much degeneration can be expected in a knee joint in order to treat cartilage defects with these regenerative treatment options. With regard to the data from the German Cartilage Registry (KnorpelRegister DGOU) [17], which represents a nationwide cohort study including more than 70 clinical centres and more than 1400 patients treated for cartilage defects of the knee between October 2013 and June 2014, the vast majority of the patients were treated for degenerative lesions and lesions with early osteoarthritis [18]. In a recent published multicentre trial of more than 400 patients treated with matrix-induced chondrocyte transplantation, almost 40% of the chondral defects were described as degenerative or chronic [19]. A significant improvement in clinical scores with a moderate increase in transplant failure after chondrocyte transplantation could be detected compared to traumatic cartilage lesions [19].

Focal cartilage defects and early- or late-stage osteoarthritis are associated with structural and mechanical changes of the meniscus, which may also contribute to further meniscal degeneration and meniscal extrusion [20]. Cartilage regenerative procedures can prevent further damage to the meniscus. Otherwise treatment of the meniscus lesion has to be performed in combination with the cartilage repair procedure. The long-term outcome and prognosis of knees with cartilage defects are therefore dependent on a successful cartilage regeneration with a proper meniscus function [20].

10.2.6 Clinical Scenarios

A varus-aligned leg with a cartilage lesion in the medial compartment of the knee needs a valgus osteotomy as most important part of treatment. Only then the loading within the knee will be reduced to allow proper healing of meniscus and/or cartilage repair or reconstruction tissue.

An ACL-deficient knee with a lateral meniscus lesion and a subsequent cartilage lesion on the lateral femoral condyle should be stabilised, and the lost meniscus tissue should be substituted.

Having understood the importance of the described treatment hierarchy of extra- and intraarticular conditions, it becomes obvious that orthobiologic treatment often results in an “a` la carte” approach [8].

10.2.7 Importance of Meniscus and Cartilage Function for Daily Activity and Sports

Since the vast majority of patients affected by focal cartilage defects are middle-aged and therefore represent an active patient population [21], return to daily activity and also return to sports have become an established outcome parameter with high clinical relevance to evaluate outcome of surgical treatment.

Recent meta-analysis revealed different return to sports rates in dependence of the type of surgical treatment applied. While higher return to sports rates have been described for patients who underwent autologous chondrocyte implantation (ACI) and autologous osteochondral transplantation (OCT), an inferior return to sports rate has been described for patients who underwent arthroscopic microfracture for focal cartilage defects [22, 23].

Also, for patients following partial meniscus resection, meniscus reconstruction and even meniscus transplantation, return to sports rates have been described and evaluated in scientific literature demonstrating that in the majority of the patients, sports participation can be achieved.

Nevertheless, for patients with cartilage defects as well as for patients with meniscus tears, time until return to sports is achieved differs in dependence of the individual pathology and the type of treatment applied. While in osteochondral transplantation a faster recovery is described, the longest time period has been described for autologous chondrocyte implantation focusing on the importance of the rehabilitation process [22].

Nevertheless, combined pathologies of cartilage and meniscus are really common in the femoro-tibial joint, and scientific data on return to sports rates are still lacking. Data from the German Cartilage Registry, which have not been published so far, demonstrate that these combined pathologies are associated with an inferior outcome and need to be considered more problematic, and duration of symptoms seems to influence return to sports probability [24]. Therefore, specific strategies for rehabilitation including return to sports recommendations need to be established in the future.

10.2.8 Future Treatment Options

Novel biologic treatment options will be developed to address these combined injuries. It is of utmost importance that a precise analysis of their usefulness in the clinical setup will be performed in clinical trials afterwards.

10.3 Take-Home Message

A meniscus–cartilage unit exists, which results in a defined close relationship between both components in the physiological and also in the pathophysiological situation. The recommended strategy for treatment of meniscal lesions is to "preserve as much meniscus as possible". Focal cartilage lesions should be addressed by regenerative treatment approaches. Even in early OA, a significant improvement could be achieved. In diffuse osteoarthritic joints, regenerative approaches to the cartilage still lack clinical improvement, and further innovative approaches have to be developed in the future.

In most cases, it takes more than meniscus and/or cartilage repair to bring a damaged knee back into its comfort zone called joint homoeostasis. The hierarchy of treatment (alignment, stability, meniscus, cartilage) has to be considered in every case.

For patients with cartilage defects, as well as for patients with meniscus tears, time until return to sports differs in dependence of the individual pathology and the type of treatment applied. Combined pathologies of cartilage and meniscus are a challenge for reconstructive surgery in order to bring our patients back to work and sport as early and safe as possible.

References

1. Bhatia S, Laprade CM, Ellman MB, et al. Meniscal root tears: significance, diagnosis, and treatment. Am J Sports Med. 2014;42:3016–30.
2. Bonasia DE, Pellegrino P, D'amelio A, et al. Meniscal root tear repair: why, when and how? Orthop Rev. 2015;7:5792.
3. Mckinley TO, English DK, Bay BK. Trabecular bone strain changes resulting from partial and complete meniscectomy. Clin Orthop Relat Res. 2003:259–67.
4. Ziegler R, Goebel L, Cucchiarini M, et al. Effect of open wedge high tibial osteotomy on the lateral tibiofemoral compartment in sheep. Part II: standard and overcorrection do not cause articular cartilage degeneration. Knee Surg Sports Traumatol Arthrosc. 2014;22:1666–77.
5. Chen HN, Yang K, Dong QR, et al. Assessment of tibial rotation and meniscal movement using kinematic magnetic resonance imaging. J Orthop Surg Res. 2014;9:65.
6. Madry H, Ziegler R, Pape D, et al. Structural changes in the lateral tibiofemoral compartment after high tibial osteotomy. Orthopade. 2014;43:958–65.
7. Ziegler R, Goebel L, Seidel R, et al. Effect of open wedge high tibial osteotomy on the lateral tibiofemoral compartment in sheep. Part III: analysis of the microstructure of the subchondral bone and correlations with the articular cartilage and meniscus. Knee Surg Sports Traumatol Arthrosc. 2015;23:2704–14.
8. Arnold MP, Hirschmann MT, Verdonk PC. See the whole picture: knee preserving therapy needs more than surface repair. Knee Surg Sports Traumatol Arthrosc. 2015;20(2):195–6. https://doi.org/10.1007/s00167-011-1733-4.
9. Hirschmann MT, Schön S, Afifi FK, Amsler F, Rasch H, Friederich NF, Arnold MP. Assessment of loading history of compartments in the knee using bone SPECT/CT: a study combining alignment and 99mTc-HDP tracer uptake/distribution patterns. J Orthop Res. 2013;31(2):268–74. https://doi.org/10.1002/jor.22206.
10. Mucha A, Dordevic M, Hirschmann A, Rasch H, Amsler F, Arnold MP, Hirschmann MT. Effect of high tibial osteotomy on joint loading in symptomatic patients with varus aligned knees: a study using SPECT/CT. Knee Surg Sports Traumatol Arthrosc. 2015;23(8):2315–23. https://doi.org/10.1007/s00167-014-3053-y.

11. Siebold R, Dejour D, Zaffagnini S. Anterior cruciate ligament reconstruction. A practical surgical guide. Heidelberg: Springer; 2014. https://doi.org/10.1007/978-3-642-45349-6.
12. Hirschmann MT, Müller W. Complex function of the knee joint: the current understanding of the knee. Knee Surg Sports Traumatol Arthrosc. 2015;23(10):2780–8. https://doi.org/10.1007/s00167-015-3619-3.
13. Scotti C, Hirschmann MT, Antinolfi P, Martin I, Peretti GM. Meniscus repair and regeneration: review on current methods and research potential. Eur Cell Mater. 2013;26:150–70. Review.
14. McGinity JB, Geuss LF, Marvin RA. Partial or total meniscectomy: a comparative analysis. J Bone Joint Surg Am. 1977;59(6):763–6.
15. Katz JN, Wright J, Spindler KP, Mandl LA, Safran-Norton CE, Reinke EK, Levy BA, Wright RW, Jones MH, Martin SD, Marx RG, Losina E. Predictors and outcomes of crossover to surgery from physical therapy for meniscal tear and osteoarthritis: a randomized trial comparing physical therapy and surgery. J Bone Joint Surg Am. 2016;98(22):1890–6.
16. Niemeyer P, Albrecht D, Andereya S, Angele P, Ateschrang A, Aurich M, Baumann M, Bosch U, Erggelet C, Fickert S, Gebhard H, Gelse K, Günther D, Hoburg A, Kasten P, Kolombe T, Madry H, Marlovits S, Meenen NM, Müller PE, Nöth U, Petersen JP, Pietschmann M, Richter W, Rolauffs B, Rhunau K, Schewe B, Steinert A, Steinwachs MR, Welsch GH, Zinser W, Fritz J. Autologous chondrocyte implantation (ACI) for cartilage defects of the knee: a guideline by the working group "clinical tissue regeneration" of the German Society of Orthopaedics and Trauma (DGOU). Knee. 2016;23(3):426–35.
17. Niemeyer P, Schweigler K, Grotejohann B, Maurer J, Angele P, Aurich M, Becher C, Fay J, Feil R, Fickert S, Fritz J, Hoburg A, Kreuz P, Kolombe T, Laskowski J, Lutzner J, Marlovits S, Muller PE, Niethammer T, Pietschmann M, Ruhnau K, Spahn G, Tischer T, Zinser W, Albrecht D. The German Cartilage Registry (KnorpelRegister DGOU) for evaluation of surgical treatment for cartilage defects: experience after six months including first demographic data. Z Orthop Unfall. 2015;153(1):67–74.
18. Angele P, Niemeyer P, Steinwachs M, Filardo G, Gomoll AH, Kon E, Zellner J, Madry H. Chondral and osteochondral operative treatment in early osteoarthritis. Knee Surg Sports Traumatol Arthrosc. 2016;24:1743–52.
19. Angele P, Fritz J, Albrecht D, Koh J, Zellner J. Defect type, localization and marker gene expression determines early adverse events of matrix-associated autologous chondrocyte implantation. Injury. 2015;46(Suppl 4):S2–9.
20. Verdonk R, Madry H, Shabshin N, Dirisamer F, Peretti GM, Pujol N, Spalding T, Verdonk P, Seil R, Condello V, Di Matteo B, Zellner J, Angele P. The role of meniscal tissue in joint protection in early osteoarthritis. Knee Surg Sports Traumatol Arthrosc. 2016;24(6):1763–74.
21. Niemeyer P, et al. Cartilage repair surgery for full-thickness defects of the knee in Germany: indications and epidemiological data from the German Cartilage Registry (KnorpelRegister DGOU). Arch Orthop Trauma Surg. 2016;136:891.
22. Mithoefer K, et al. Return to sports participation after articular cartilage repair in the knee: scientific evidence. Am J Sports Med. 2009;37(Suppl 1):167S–76S.
23. Campbell AB, et al. Return to sport after articular cartilage repair in athletes' knees: a systematic review. Arthroscopy. 2015;32:651.
24. Pestka JM, et al. Return to sports activity and work after autologous chondrocyte implantation of the knee: which factors influence outcomes? Am J Sports Med. 2016;44(2):370–7.

11

Osteotomies: Advanced and Complex Techniques

Susannah Clarke, Justin Cobb, Martin Jaere,
Gareth Jones, Kristian Kley, Philipp Lobenhoffer,
Christopher McCrum, Volker Musahl,
and Ryohei Takeuchi

11.1 3D Planned and Printed Patient Matched Osteotomy

11.1.1 Introduction

We started performing precise surgery based upon CT plans in the last century – the first embodiment of this approach was a robotic assistant built for total knee replacement, the "Acrobot" [1]. Abundant evidence now exists to confirm that assistive technologies enable surgeons to achieve their preoperative goals [2]. The concept of planned surgery is therefore not novel. Patient-matched instruments share several key elements with the robotic platform, and these formed the basis of this current project. The essential elements include image segmentation, planning, and registration. We applied the know-how of these dimensions to design and build patient-matched guides for a range of tasks using biocompatible polymer 3D printers. Having established a workflow for arthroplasty, the adaptation of the same principles to osteotomy was a short step, requiring software to be developed to deliver semiautomated useful information regarding limb segment alignment and the shapes of bones.

11.1.2 Method

To plan any procedure in 3D, images are acquired. Currently, we use CT for the bone model and EOS® to confirm both limb alignment and the impact of any shortening on the spine and entire body (Fig. 11.1). Using CT, the bone can be segmented out semiautomatically, using Hounsfield unit thresholds from a low-dose protocol [3]. This is rather easier in deformity correction than in arthrosis, as the joint spaces are better preserved. When segmenting for arthroplasty, in the presence of substantial arthrosis, separating out bone surfaces can be both tedious and time-consuming, as it needs significant human input to complete the task.

Having obtained the bone models, the task of planning can be semiautomated. We have already shown that there is in effect a lookup table of

---

S. Clarke • J. Cobb • M. Jaere • G. Jones
MSk Lab, Imperial College, London, UK

K. Kley (✉) • P. Lobenhoffer
GO-H (Gelenkchirurgie Orthopädie Hannover),
Hannover, Germany
e-mail: kristian.kley@g-o-hannover.de;
Philipp.lobenhoffer@g-o-hannover.de

C. McCrum • V. Musahl
Department of Orthopaedic Surgery, University
of Pittsburgh Medical Center, Pittsburgh, PA, USA
e-mail: musahlv@upmc.edu

R. Takeuchi
Joint Surgery Center, Yokosuka City Hospital,
Yokosuka, Kanagawa, Japan

G.M.M.J. Kerkhoffs et al. (eds.), *ESSKA Instructional Course Lecture Book*,
https://doi.org/10.1007/978-3-662-56127-0_11

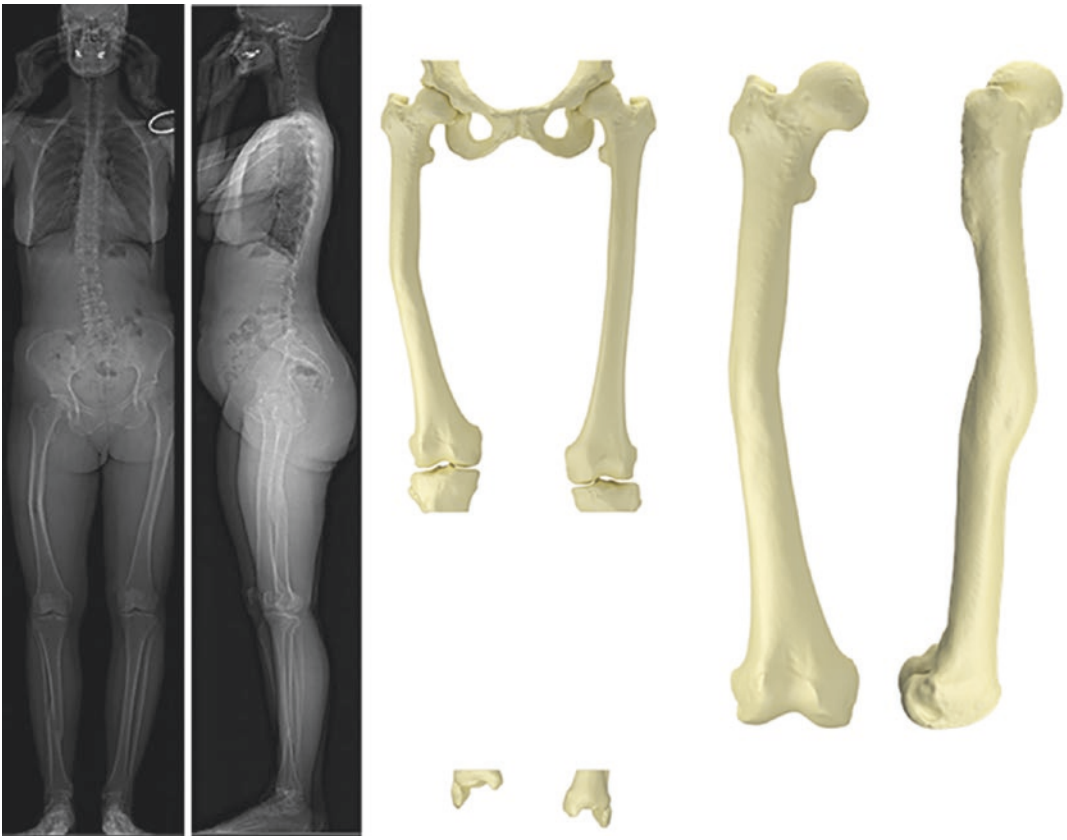
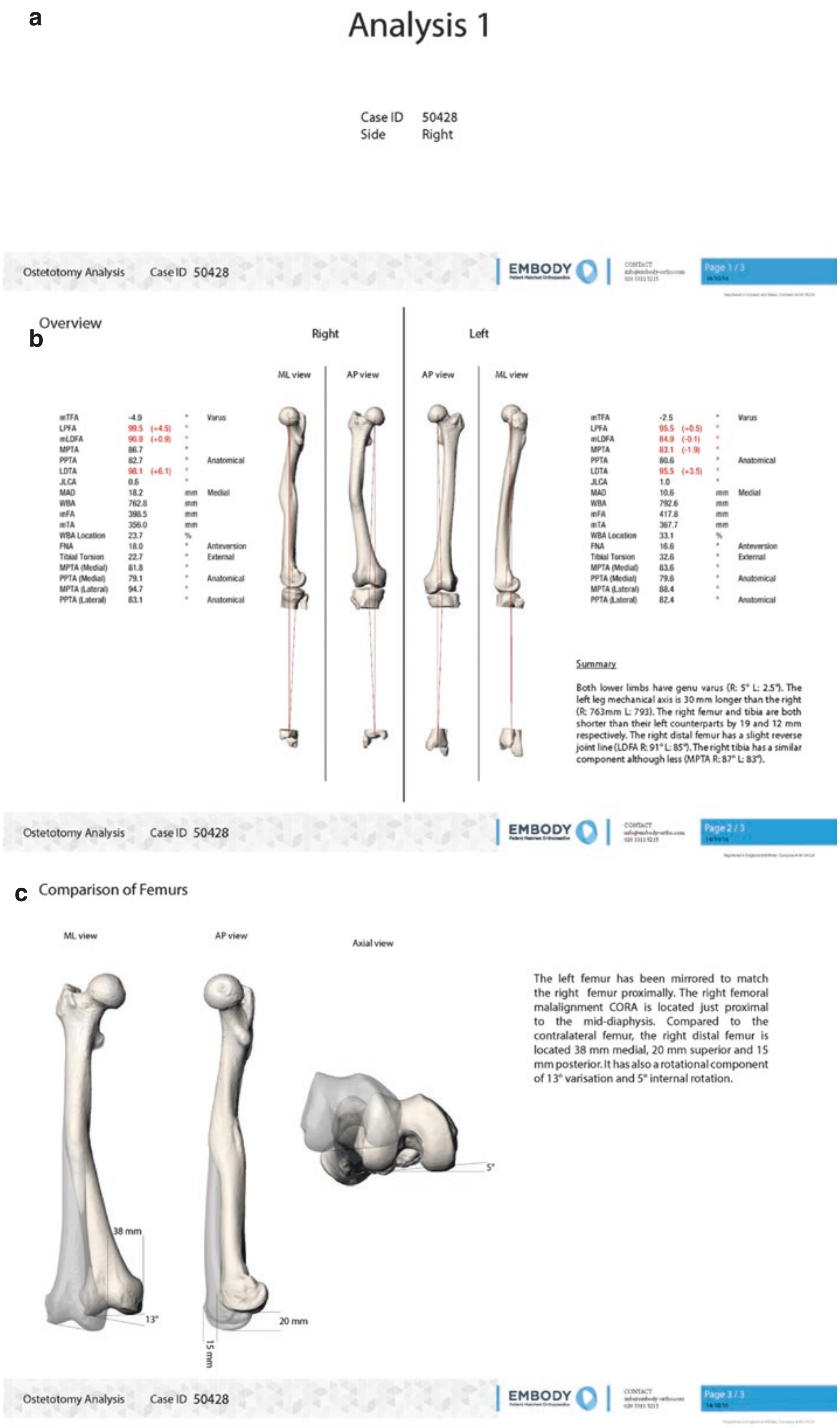


Fig. 11.1 Anteroposterior and lateral radiographs for assessment of limb alignment (left) and 3D model of EOS® imaging for preoperative planning (right)

deformity in varus: the supine deformity is increased by some 2° on weight bearing and by some 2° more in the stance phase of gait [4]. For corrective osteotomy of the proximal tibia, for instance, we are still exploring the impact of correction on gait, and our current understanding is less than ideal, especially when there is some chondral loss. So, as a group we do not support the "Fujisawa" point; instead we support offering the surgeon the option to correct alignment as much as he or she chooses, with correction to neutral alignment an option or any variant of this. Having undertaken less than 50 corrective osteotomies using full 3D planning, as a group we still are less than confident in how much correction is optimal for any one case.

In posttraumatic deformity correction, the task is somewhat simple. In general, the aim is simply to restore to the pre-fracture state. Using a Matlab script written by one of the coauthors (SJC), the good leg is simply flipped onto the bad leg, matching the larger bone segment. This allows the surgeon to appreciate the extent of the problem.

The deformity is then analyzed. In general, there is always a degree of rotation and translation, with the rotation always being "out of plane" of either an anteroposterior (AP) or a lateral projection, and the translation usually includes some substantial shortening. The exact extent of these angles and translations are provided semiautomatically with the other limb for comparison (Fig. 11.2). After describing the extent of the shortening and rotation, the planner then "simply" corrects the distal segment and chooses a plane for the correction that allows the bone segments to slide and rotate, restoring medullary continuity so that intramedullary fixation is an option at least (Fig. 11.3).



Analysis 1

Case ID 50428
Side Right

Overview

mTFA	-4.9		°	Varus
LPFA	99.5	(+4.5)	°	
mLDFA	90.9	(+0.9)	°	
MPTA	86.7		°	
PPTA	82.7		°	Anatomical
LDTA	98.1	(+6.1)	°	
JLCA	0.6		°	
MAD	18.2		mm	Medial
WBA	762.8		mm	
mFA	398.5		mm	
mTA	356.0		mm	
WBA Location	23.7		%	
FNA	18.0		°	Anteversion
Tibial Torsion	22.7		°	External
MPTA (Medial)	81.8		°	
PPTA (Medial)	79.1		°	Anatomical
MPTA (Lateral)	94.7		°	
PPTA (Lateral)	83.1		°	Anatomical

mTFA	-2.5		°	Varus
LPFA	95.5	(+0.5)	°	
mLDFA	84.9	(-0.1)	°	
MPTA	83.1	(-1.9)	°	
PPTA	80.6		°	Anatomical
LDTA	95.5	(+3.5)	°	
JLCA	1.0		°	
MAD	10.6		mm	Medial
WBA	792.6		mm	
mFA	417.8		mm	
mTA	367.7		mm	
WBA Location	33.1		%	
FNA	16.6		°	Anteversion
Tibial Torsion	32.6		°	External
MPTA (Medial)	83.6		°	
PPTA (Medial)	79.6		°	Anatomical
MPTA (Lateral)	88.4		°	
PPTA (Lateral)	82.4		°	Anatomical

Summary

Both lower limbs have genu varus (R: 5° L: 2.5°). The left leg mechanical axis is 30 mm longer than the right (R: 763mm L: 793). The right femur and tibia are both shorter than their left counterparts by 19 and 12 mm respectively. The right distal femur has a slight reverse joint line (LDFA R: 91° L: 85°). The right tibia has a similar component although less (MPTA R: 87° L: 83°).

Comparison of Femurs

The left femur has been mirrored to match the right femur proximally. The right femoral malalignment CORA is located just proximal to the mid-diaphysis. Compared to the contralateral femur, the right distal femur is located 38 mm medial, 20 mm superior and 15 mm posterior. It has also a rotational component of 13° varisation and 5° internal rotation.

Fig. 11.2 (**a**–**c**) Preoperative analysis and planning steps

EMBODY
Patient Matched Orthopaedics

CASE 50428

OSTEOTOMY PSI

Operative Technique and Parts List

Operative Technique - Case 50428

EMBODY
Patient Matched Orthopaedics

CONTACT
info@embody-ortho.com,
020 3311 5215

Page 1 / 8
05/02/17

Parts List

ADDITIONAL PARTS REQUIRED:
6 x 3.2 mm diameter Bone Pins

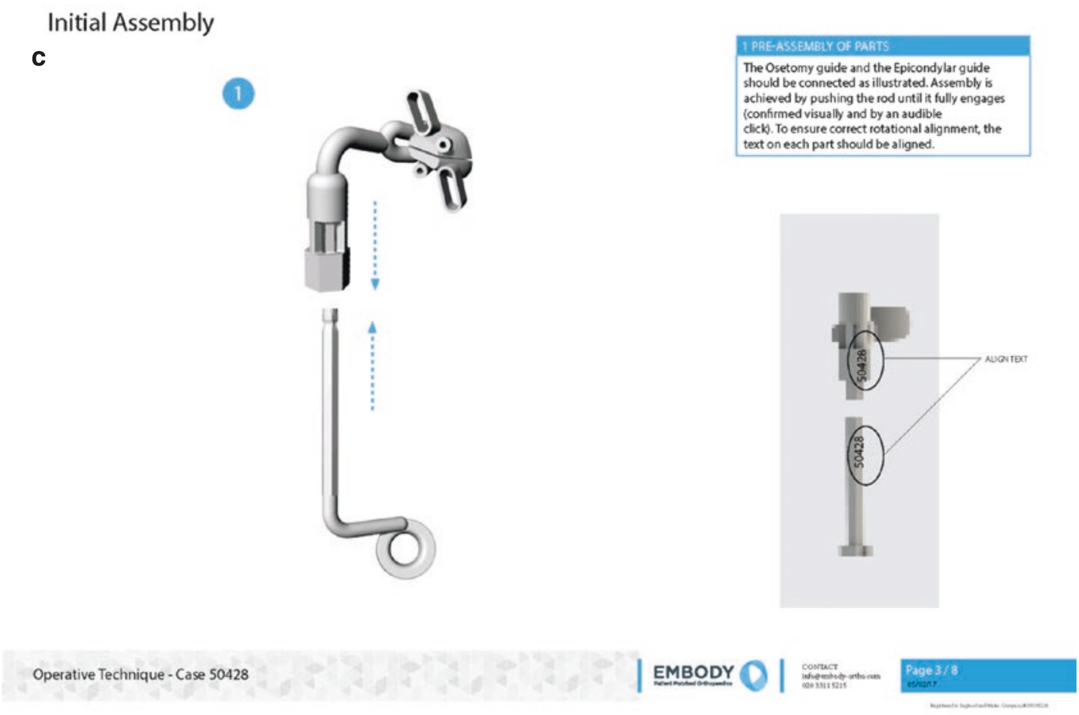
Operative Technique - Case 50428

EMBODY
Patient Matched Orthopaedics

CONTACT
info@embody-ortho.com
020 3311 5215

Page 2 / 8
05/02/17

Fig. 11.3 (**a**–**h**) Operative technique and parts list c



Initial Assembly

1 PRE-ASSEMBLY OF PARTS

The Osetomy guide and the Epicondylar guide should be connected as illustrated. Assembly is achieved by pushing the rod until it fully engages (confirmed visually and by an audible click). To ensure correct rotational alignment, the text on each part should be aligned.

Operative Technique - Case 50428

EMBODY
Patient Matched Orthopaedics

CONTACT
info@embody-ortho.com
020 3311 5215

Page 3 / 8
05/02/17

d

2 GUIDE LOCATION SKIN

Place the later epicondyle as a reference point for the epicondylar guide. This will guide the incision point.

3 GUIDE LOCATION BONE

Once the incision is made, place the guide osteotomy guide on the bone. The guide should match the bone surface.

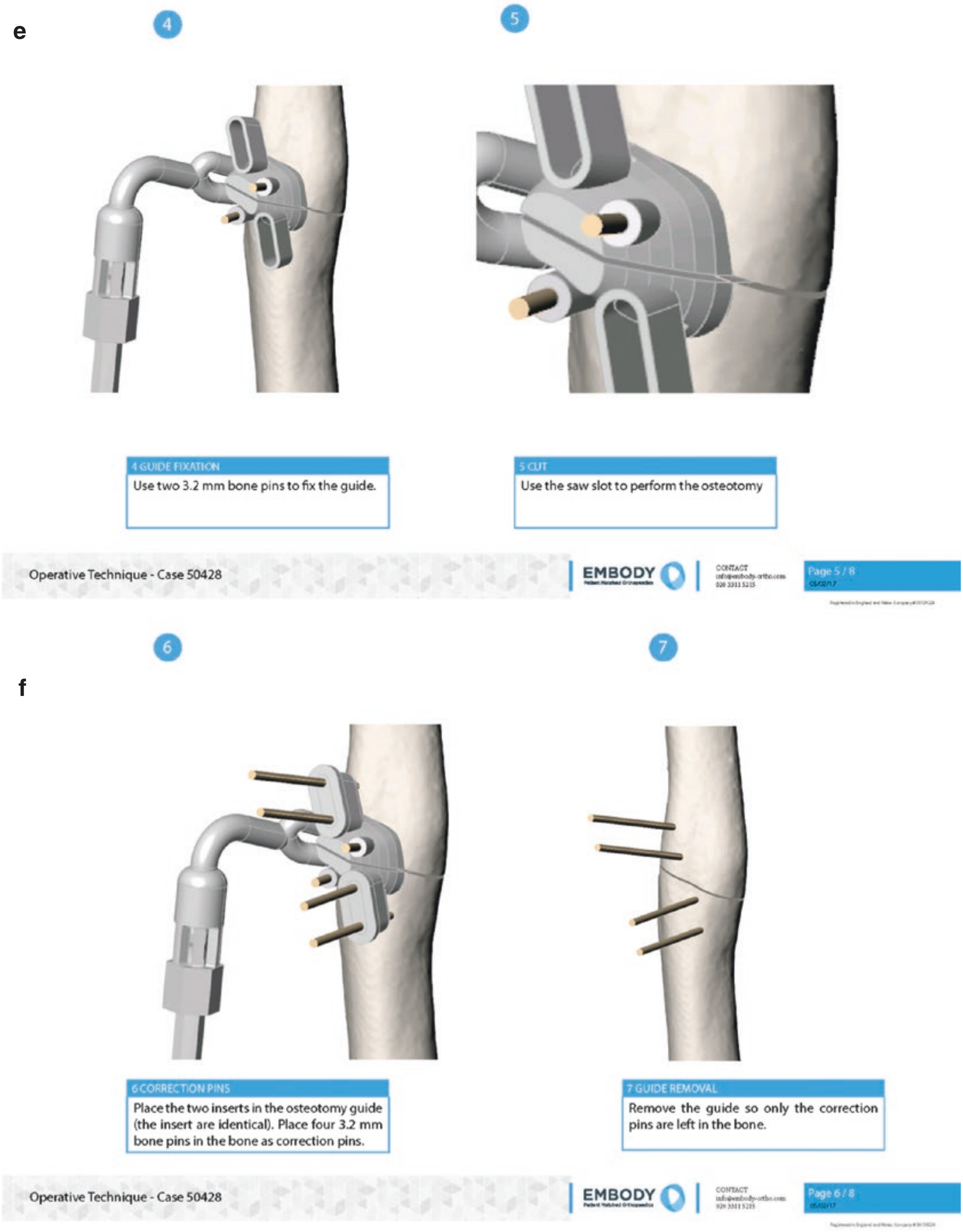
Operative Technique - Case 50428

EMBODY
Patient Matched Orthopaedics

CONTACT
info@embody-ortho.com
020 3311 5215

Page 4 / 8
05/02/17

Fig. 11.3 (continued)



4 GUIDE FIXATION

Use two 3.2 mm bone pins to fix the guide.

5 CUT

Use the saw slot to perform the osteotomy

Operative Technique - Case 50428

EMBODY
Patient Matched Orthopaedics

CONTACT
info@embody-ortho.com
020 3313 5215

Page 5 / 8
05/02/17

6 CORRECTION PINS

Place the two inserts in the osteotomy guide (the insert are identical). Place four 3.2 mm bone pins in the bone as correction pins.

7 GUIDE REMOVAL

Remove the guide so only the correction pins are left in the bone.

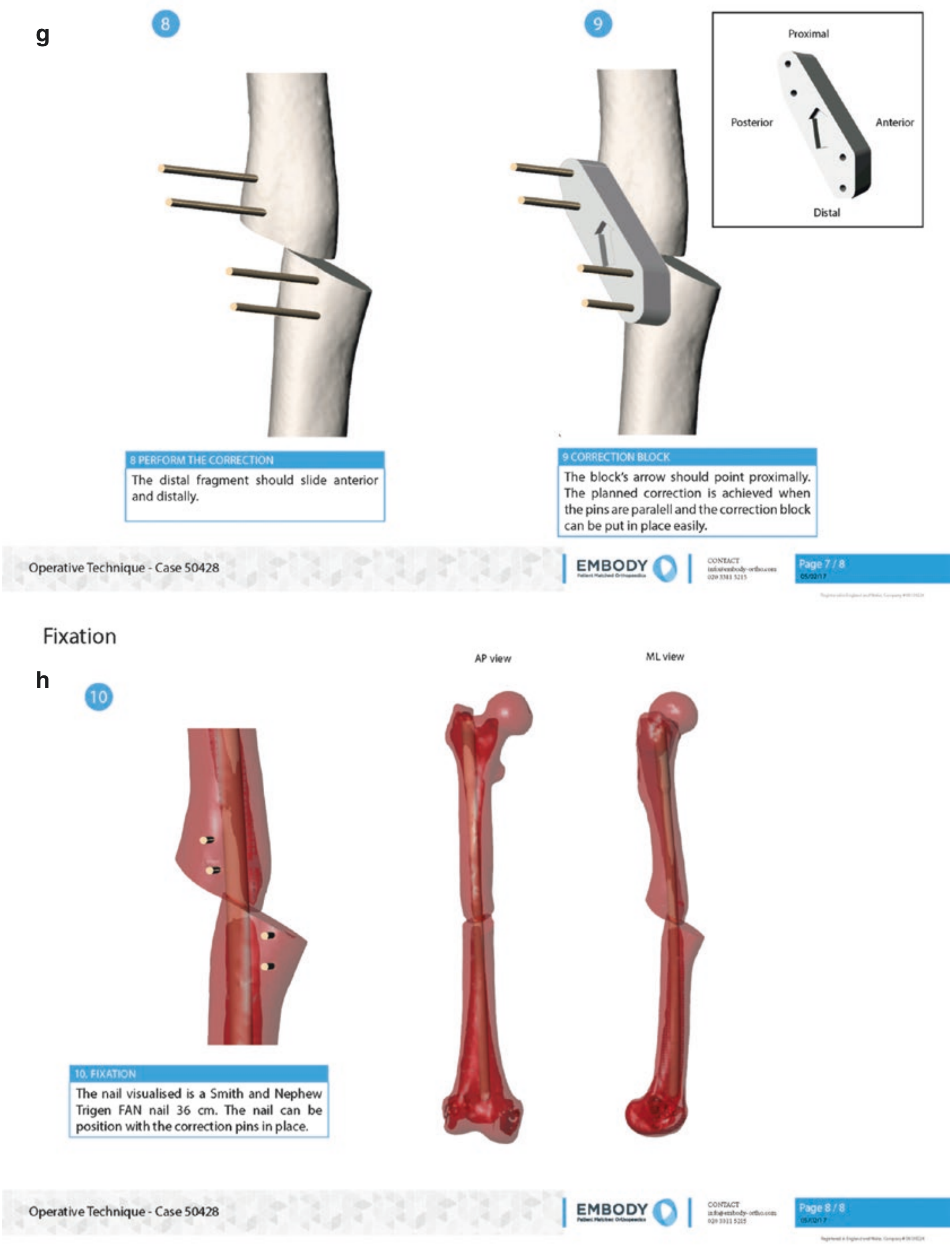
Operative Technique - Case 50428

EMBODY
Patient Matched Orthopaedics

CONTACT
info@embody-ortho.com
020 3313 5215

Page 6 / 8
05/02/17

Fig. 11.3 (continued)



8 PERFORM THE CORRECTION

The distal fragment should slide anterior and distally.

9 CORRECTION BLOCK

The block's arrow should point proximally. The planned correction is achieved when the pins are paralell and the correction block can be put in place easily.

Operative Technique - Case 50428

EMBODY
Patient Matched Orthopaedics

CONTACT
info@embody-ortho.com
020 3381 5215

Page 7 / 8
05/02/17

Fixation

10. FIXATION

The nail visualised is a Smith and Nephew Trigen FAN nail 36 cm. The nail can be position with the correction pins in place.

Operative Technique - Case 50428

EMBODY
Patient Matched Orthopaedics

CONTACT
info@embody-ortho.com
020 3381 5215

Page 8 / 8
05/02/17

Fig. 11.3 (continued)

Fig. 11.4 Fluoroscopy for confirmation of positioning

Guide design is generic in some features, so it can be semiautomated. Two elements need consideration: multipoint registration and soft tissue access. Ideally, subcutaneous features distant to the osteotomy are employed to improve gross positioning. Local features are also used, over as large an area as is practicable. Finally, fluoroscopic control is used to confirm positioning (Fig. 11.4).

Implant selection can be made during planning: the exact length of the fixation device and the exact screw lengths can be predicted with confidence, reducing the opportunity for error in screw length selection. Detailed planning of sizes at this stage reduces the cost and duration of the procedure by ensuring that exactly the right size components are ready beforehand.

Intraoperatively, both the detailed plan and the instruments provided for 3D planned corrective osteotomy reduce the level of anxiety considerably. At operation, the soft tissue access is of major importance. Inevitably, considerable manipulation of the bone fragments and the overlying muscle takes place, so an extensile approach should be considered and care taken to release any tethering of muscle or fascia before osteotomy and correction. We use infiltration of local anesthetic and do not employ a tourniquet.

11.1.3 Results

Since 2012, we have undertaken 49 corrective osteotomies. Eight of these have been combined osteotomies and arthroplasties. Two have combined a corrective osteotomy and total knee arthroplasty, while six have included a partial knee replacement. Two infections have occurred, both in soldiers who had sustained battlefield wounds. Both osteotomies have gone on to union. One plate has broken, requiring exchange for an intramedullary rod, and both of the distal tibial corrective osteotomies have united but only after considerable time. One of these, a rotational and lengthening osteotomy, required 4 months of exogen to ensure union.

11.1.4 Conclusion

3D planned and printed osteotomy is now an established methodology. By investing in detailed planning, the intraoperative process is simplified substantially, and the clinical outcomes are encouraging. The project has enabled some very complex reconstructions, and by automation, the time taken to produce a plan has fallen steadily.

11.2 Minimally Invasive Osteotomies Around the Knee

11.2.1 Introduction/Historical Background

Osteoarthritis is a very common cause of knee pain. It affects high percentages of patients above 60 years of age [5] but is also seen in younger patients as a result of different etiologies including rheumatoid arthritis, a genetic predisposition, poor cartilage quality, or obesity.

In addition, mechanical malalignment is widely accepted as a major source of osteoarthritis. Numerous studies have shown that at least 30% of the male population and almost 20% of the female population have a lower limb malalignment of more than 3° [5–7]. Regardless of the underlying cause of this malalignment, it then secondarily leads to high pressure loads and peak load areas resulting in mechanical abrasion. The patient then enters a vicious circle of progressive cartilage loss and worsening malalignment [8–11].

It is widely accepted that the best way of realigning and treating malalignment is with an osteotomy performed around the knee. Depending on the malalignment in the coronal plane, varus or valgus corrections can be achieved by open or closed-wedge osteotomies, which can be carried out laterally or medially at the level of the femur or tibia.

Regardless of the type of osteotomy, the aim is to correct the malalignment by changing the weight-bearing line and shift the peak load areas [12]. Recently, more attention was paid to the orientation of the joint line in relation to the Mikulicz line to restore the kinematic alignment profile [5, 13, 14]. To avoid creating a new deformity and malalignment of the joint line orientation, proper analysis is mandatory [15–17]. Having carried out the deformity analysis, it is not uncommon to find that there is a degree of deformity in both the distal femur and proximal tibia. And it is the experience of our unit and other centers [14] that the best results can be achieved by making the corrective realignment procedure at the level of the osteotomy. That, in many cases, is in both the distal femur and the proximal tibia with a double osteotomy. Though newer techniques of high tibial osteotomies (HTO) led to superior results, HTO and distal femoral osteotomy (DFO) are still considered to be difficult procedures with high potential risk in terms of complications [18, 19]. In the case of HTO, the number of recent outcome papers is low, and these are mostly based on historical techniques with relatively poor outcome long term [20, 21]. Again, in DFO surgery the procedure is considered technically challenging with higher complication rates, although a meta-analysis did not reveal supporting evidence [22].

It is now widely accepted that there is at least a 20% dissatisfaction rate with total knee replacement (TKR) surgery. There are also limitations of what can be achieved with a TKR, and we always need to have a plan "B" in the event of failure and think about the next procedure in an ever-aging population with high demands. In spite of this, this procedure is increasingly performed [23]. Paradoxically, osteotomy yields excellent results [19, 24, 25]; however the number of osteotomies carried out is decreasing. This could be explained by the feeling that these procedures are considered to be difficult and high risk. Furthermore, the shift to joint replacement surgeries over the last decades has been encouraged by the industry, and at the same time, we have seen relatively few centers of excellence and training initiatives for osteotomy surgery.

Taking that into consideration, there has been a need to adopt the recent major advantages of osteotomies around the knee and simplify the procedures to make them more reproducible and less traumatic.

11.2.2 State-of-the-Art Treatment/Biomechanical Problems HTO and DFO

One of the problems in a review of the literature of osteotomy surgery is the vast number of heterogeneous treatments and techniques that have been reported. As a result, there is a strong need to define a surgical standard that needs to be reproducible in terms of indication, planning, execution, and teaching. At the moment, the only standardized teaching and techniques to meet these criteria for osteotomy are those recommended by the Joint Preservation Expert Group (JPEG) of AO.

Correcting the tibia is achieved by medial open or closed-wedge technique for valgus or varus deformity. The more reproducible and accurate approach for high tibial osteotomy surgery is to carry out a medial opening wedge procedure of the proximal tibia. Unlike the lateral closing wedge technique, where there is a risk of common peroneal nerve injury, a need to perform a fibula osteotomy and more soft tissue dissection to the lateral compartment, the medial approach involves minimal soft tissue dissection, significantly less risk of neurovascular damage, and the open wedge approach allows “fine-tuning” of the osteotomy [26, 27].

For femoral malalignments, routinely closed-wedge osteotomies (either medially or laterally) are performed to correct coronal plane deformities. The distal femur shows different biomechanics than the proximal tibia. The surface at the level of the osteotomy is smaller on the femoral side. There is no natural “hinge-preserver” such as the fibers of the proximal tibiofibular joint in the area of the safe zone [28, 29], and the lever arm of the DFO is longer. As a result, DFO is inherently more unstable. To help with this problem of potential instability, we recommend, as a routine, that a proximal biplane second cut is made to provide more stability and also to help with the healing process [30, 31].

The biplanar technique for DFO and HTO (Fig. 11.5) has numerous advantages. Geometrically the volume of the osteotomy is reduced, the osteotomy can be performed closer to the metaphysis with better bone healing, and there is an inherent higher axial stability, protection against the potential issue of malrotation, and an option for reduction in case of a hinge fracture [27, 30, 31].

These biplanar techniques, along with angle-stable plate fixators, reproducibly showed very good midterm results and patient satisfaction [19, 32, 33]. A further technical advancement has been the introduction of a minimally invasive (MIS) approach to both, the proximal tibia and distal femur, which has been the standard in our department for the last 2 years.

11.2.3 MIS Technique/Future Options

The MIS biplanar technique we have developed is less invasive but still allows the procedures to be carried out safely in experienced hands. The key is to make the incision at the right location to allow optimal visualization (Fig. 11.6). This incision allows a window to be created, and like any MIS technique, this window is moved as is required. With increasing surgical experience in osteotomy surgery, it is possible to bring down

Fig. 11.5 Biplanar osteotomy at the distal femur (DFO) and the proximal tibia (HTO)

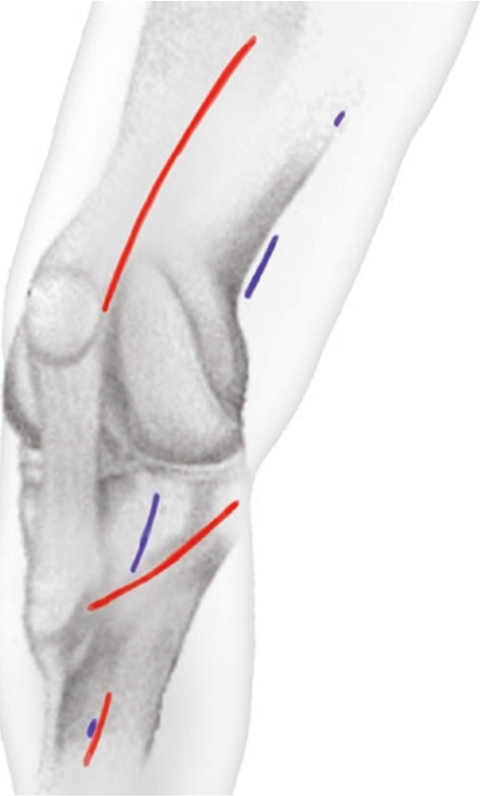


Fig. 11.6 Incisions for conventional (red) and MIS (blue) technique

the incisions to less than 4 cm for DFO and HTO. We do not advocate this technique for less experienced osteotomy surgeons at the start of their learning curve and would suggest a stepwise progression toward the MIS technique as the surgeon gains experience and confidence.

MIS in medial open wedge HTO is linked to the incision length. As there is no critical structure in the approach up to the MCL, MIS doesn't help to reduce the surgical trauma. But taking into consideration that osteotomy patients undergo revision surgery, the surgical pathways need to be planned thoughtfully. The skin bridge between two proximal tibial incisions should at least be 5 cm [34]. An incision shorter than that can be considered noncritical. Substantial change with the MIS approach is the direction of the incision, which at the distal end is medial to the tibial tubercle and goes slightly obliquely to the level of joint line and no longer to the posteromedial corner. The two osteotomy cuts need to be performed sequentially in a window-shift technique so as the placement of the drill sleeves being used for the plate fixator in the proximal part. The shaft screws are placed through a single-stab incision.

For the DFO procedure, the MIS approach moved significantly from a long median incision to a medial or lateral 4 cm incision at approximately 4 cm above the epicondyles. At the medial side, the fascia of the vastus medialis is then incised and longitudinally split. Blunt dissection around the muscle is performed to the dorsal circumference. At the lateral aspect, the upper border of the iliotibial band needs to be identified and divided in a longitudinal way. Care is to be taken at the distal parts around the vastus lateralis as branches of the lateral ascending genicular artery penetrate the intermuscular septum. These either need to be ligated or coagulated. From there, it is possible to lift the vastus medialis or lateralis to gain access to the medial or lateral intermuscular septum, which is then incised close to the femur. The key is to dissect the posterior aspect of the femur. That has to be revealed, and this can be done minimally invasively without lifting the whole muscles at each side. By doing so, there is minimal compromise to the vascularity of the area. It has been shown that conventional plating, when compared to MIPO (minimal invasive plate osteosynthesis), causes limitations for periosteal and bone marrow perfusion, leading to compromised osteotomy consolidation [35–37]. Performing the osteotomy and applying the plate like in HTO is achieved by window shifting. The placement of the shaft screws is also done by stab incision. To establish a safe portal with limited damage to the muscle, a cannula is placed in the stab incision. Following these MIS principles, in our hands surgical trauma has been reduced. Following deformity analysis, we have found significant numbers of varus patients with femoral deformity. Up to 20% of our varus deformities are located in the distal femur and are treated by lateral closing wedge DFO surgery. In conclusion, femoral osteotomy procedures are currently not carried out in sufficient numbers. Where appropriate, osteotomy needs to be performed in the femur and not always carried out in the tibia for varus deformity. The same is true in valgus deformity, where a significant number of patients have a deformity in the tibia as opposed to the femur, and therefore this is where the osteotomy should be carried out to prevent the procedure from creating an oblique joint line and a new deformity. In recent times, attention has been paid to the orientation of the joint line, along with the postoperative correction of the weight-bearing line [5, 14, 38]. From a dynamic perspective, the knee medializes during gait. This leads to a horizontal joint line orientation, when the joint line is medially inclined in stance. As a consequence, altering the MPTA to abnormal values to correct the weight-bearing line might lead to disturbed kinematics. Though at present there is no scientific evidence, our threshold for a postoperative MPTA is 93°. It is important to understand that the femur is decisive for joint line orientation. If the desired correction cannot be achieved within reasonable values for mLDFA and/or MPTA, we tend to plan a DLO (double-level osteotomy).

11.2.4 Take-Home Message

Biplanar osteotomy with angle-stable plate fixators produces excellent results when carried out for the right indications. As our understanding of malalignment, planning and execution of osteotomy surgery has evolved, the vast majority of the technical challenges of the past have been largely solved. Having said that, this field will continue to develop and improve with time. These interesting new concepts of correction philosophies have led to controversial discussion over the last years and led to promising concepts such as the MIS technique and focus on joint line orientation. The big challenge of the next decades will be to bring osteotomy back to a broader surgical society and to establish these working concepts as standard treatment pathways.

11.3 Intra-articular Osteotomies

11.3.1 Introduction/Historical Background

Osteotomy around the knee was traditionally focused on metaphyseal corrections, mainly treating deformities in the frontal plane. Indications for intra-articular osteotomy may be unilateral constitutional deformity, posttraumatic intra-articular deformity (Fig. 11.7), and unilateral deformity induced by osteoarthritis. Intra-articular osteotomies may be performed by hemiplateau osteotomy (wedge type, Chiba type) and by plateau osteotomy.

11.3.2 State-of-the-Art Treatment

11.3.2.1 Constitutional Deformities

Constitutional deformities suitable for intra-articular osteotomy are Blount disease, Ellis-van Creveld syndrome, and also some types of achondroplasia. In all these conditions, one tibia plateau will be depressed or angulated, causing significant axial deformity of the leg. Normal anatomy can be restored by the technique of hemiplateau osteotomy (Fig. 11.8). An osteotomy plane is created under the involved compartment with the hinge point at the borderline to the intact part of the tibia plateau. A wedge-type fragment is thus created, and by opening the osteotomy plane, correction is achieved. The gap is usually filled with an autologous bone graft from the iliac crest, and fixation is achieved by a locking plate [39].

The technique of open wedge hemiplateau osteotomy allows to restore the joint line but does not interfere with any metaphyseal deformity the patient may have in addition. In this case, the intra-articular osteotomy may be combined with a typical extra-articular osteotomy in the metaphyseal area, or the correction has to be planned in two stages [40].

11.3.2.2 Posttraumatic Intra-articular Deformities

Intra-articular Osteotomy

Intra-articular fractures may result in malunions, and intra-articular deformities are commonly seen after tibial plateau fracture treatment. A certain percentage of cases may be suitable for hemiplateau osteotomy. However, the defect of the joint surface may be irregularly shaped due to depressed osteochondral fragments. In this situation, a direct intra-articular osteotomy may be used, elevating

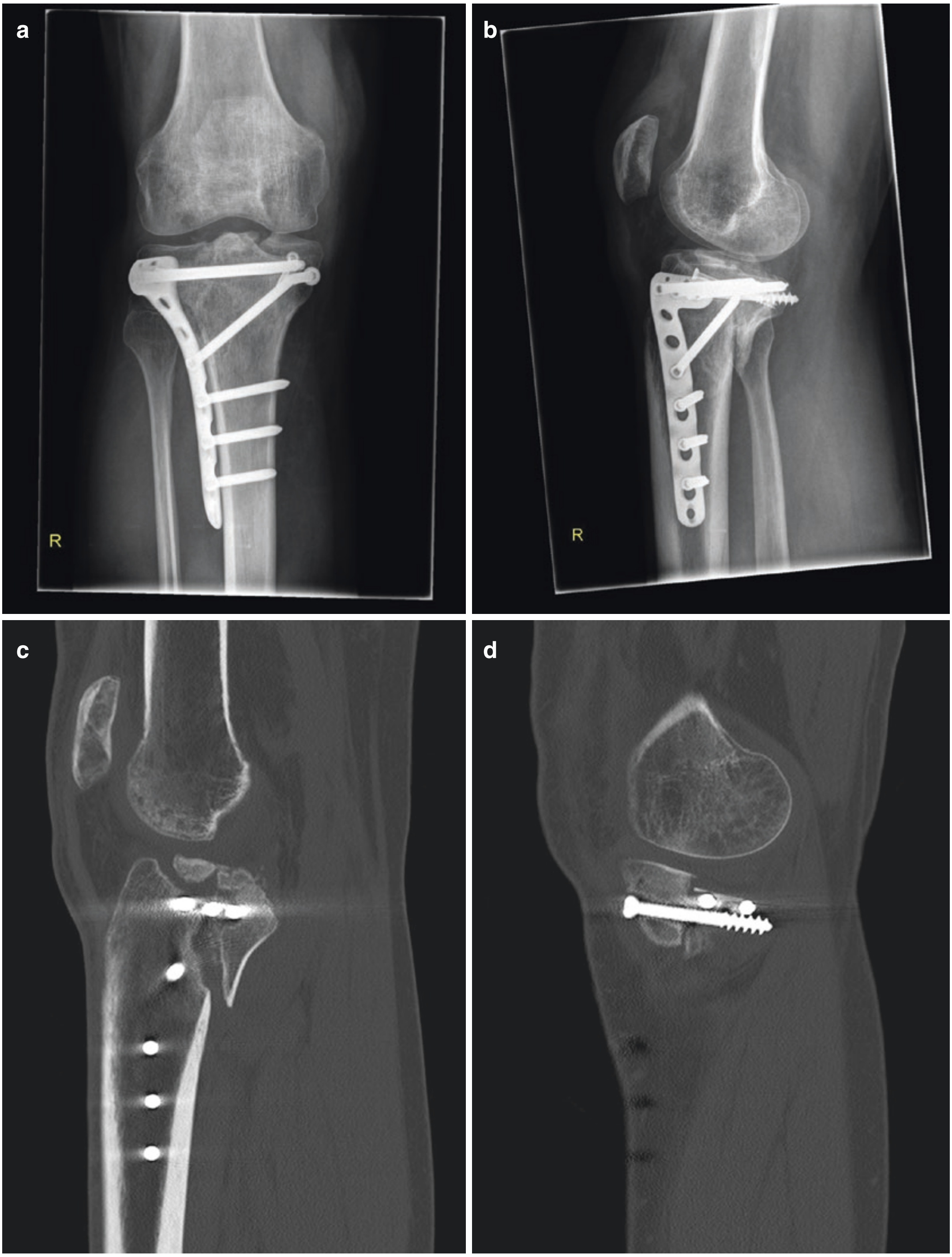


Fig. 11.7 (**a**–**h**) Intra-articular osteotomy. The images show a 34-year old patient after ORIF of bicondylar tibial plateau fracture (**a** and **b**). At 8 months, CT reveals a pseudarthrosis (**c**), with an articular step of 1 cm (**d**). Treatment included removal of medial hardware, mobilization of the pseudarthrosis (prone position, posteromedial approach), open reduction, fixation with posterior plate, and use of autologous bone graft from the posterior iliac crest. Postoperative radiographs (**e** and **f**) and after 9 months (**g** and **h**) are shown. The patient is now pain-free and is able to go mountain hiking without problems

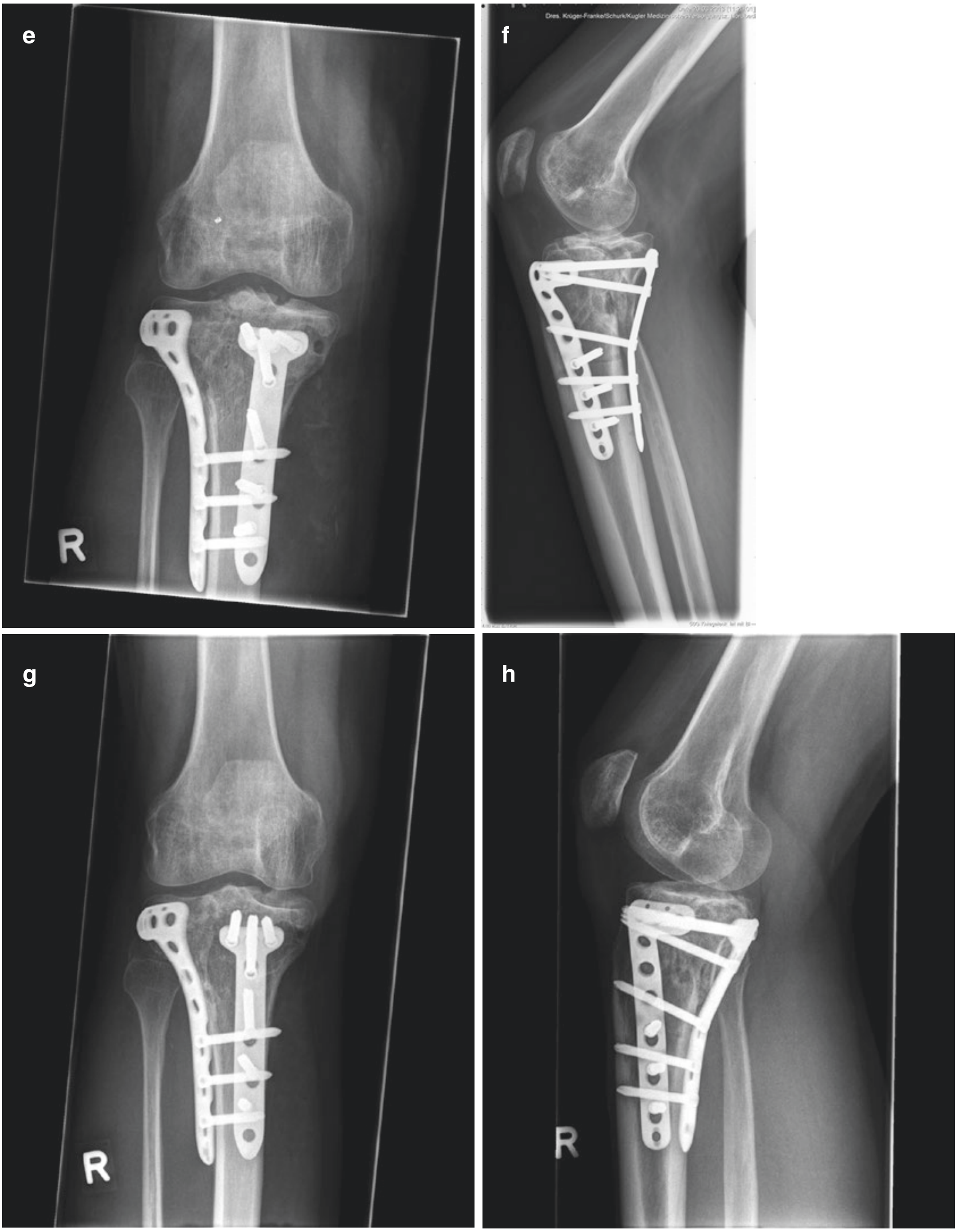


Fig. 11.7 (continued)

the depressed fragments and thus recreating the initial fracture situation. A specific extended exposure of the tibia plateau is mandatory to allow the surgeon to work under direct vision, and autologous bone grafts as well as locking plates are used to support the elevated areas. Again, this intervention can be combined with a metaphyseal osteotomy to correct an additional deformity [40].

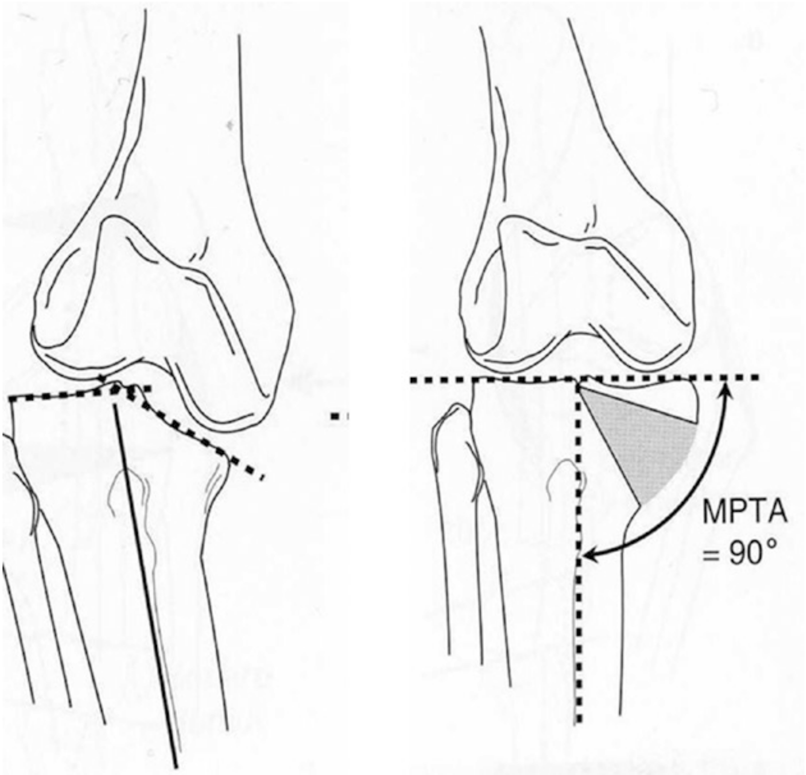


Fig. 11.8 Principle of hemiplateau osteotomy

Osteochondral Grafting and Metaphyseal Osteotomy

Open reduction and internal fixation may lead to necrosis of the osteochondral areas involved. In these defect situations, a reconstruction with local tissue is not possible any more. Fresh solid allogenic osteochondral grafts can be used to substitute for the defect if available. Such grafts are not available in our country, and we use a shaped cortico-cancellous graft from the iliac crest instead. The periosteal part of this graft faces the joint cleft. This graft is shaped individually to the defect for press fit; residual defects are filled with cancellous bone grafts. A locking plate can be used to support the graft by rafting screws. The overall alignment in the frontal plane has to be corrected, and any overload of the graft requires an additional metaphyseal osteotomy.

Intra-articular Osteotomy for Osteoarthritis of the Knee

Metaphyseal osteotomy around the knee is not recommended for patients with normal long bone configuration but gross intra-articular defects. An extra-articular correction of alignment will lead to an abnormal orientation of the joint line, creating shear stress on the cartilage. However, by elevating the involved side, the instability of the joint and the imbalance may be eliminated. This concept of intra-articular osteotomy in osteoarthritis was developed in Japan by Chiba (TCVO, tibia condylar valgus osteotomy) (Fig. 11.9). Medium-term results show patient benefit at a level of arthroplasty [39].

11.3.3 Future Treatment Options

Advanced digitizing and modelling technology will be helpful in planning of intra-articular osteotomies. PSI techniques will allow for individualized osteotomies based on advanced planning programs. Robotic technology may aid the surgeon to make individual grafts for large defects.

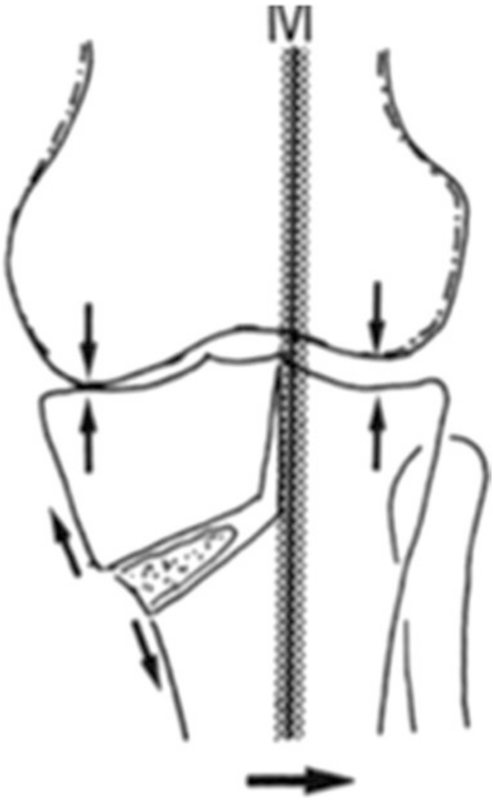


Fig. 11.9 Principle of tibial condylar valgus osteotomy (TCVO/Chiba)

11.3.4 Take-Home Message

Preservation of the knee joint is possible in many cases of intra-articular defects or posttraumatic malunion. The clinical results in appropriate selection are superior compared with arthroplasty which has a significant risk profile. Key points are restoration of the joint line and correction of any frontal plane deformity causing overload of the involved compartment.

11.4 Osteotomies for the Cruciate Deficient Knee

11.4.1 Anatomy and Biomechanics of Slope

In addition to stability imparted by ligamentous and soft tissue structures about the knee, the tibiofemoral joint articular surfaces contribute significantly to stability of the knee joint. In addition to coronal alignment, posterior tibial slope (PTS) plays a critical role in both the sagittal and rotational stability of the knee.

Generally, tibial slope is measured on the lateral radiograph and is defined as the angle between a line drawn parallel to the tibial plateau and a line along the longitudinal axis of the tibial. Conventionally, the line is drawn along the medial tibial plateau, but it is becoming more evident that the lateral tibial plateau slope is critical to the kinematics of the knee as well.

A wide range of tibial slope has been proposed in the literature. Although tibial slope varies between individuals, posterior tibial slope is generally accepted to be within the range from 6° to 11° in the sagittal plane, and it is generally believed to be pathologic if it is ≥12° [41–43]. This slope results in a shear force when compressive load is passed through the joint, which results in an anterior translation moment of the tibia relative to the femur [44, 45]. This anterior shift tends to be most pronounced with the knee in full extension and decreases with flexion of the knee. Furthermore, axial compressive load results in a proximal and anterior force, as a result of posterior tibial slope [46].

Osteotomy shifts the contact pressures and resting position of the tibia with respect to the femur, with an increase in the slope moving the resting point posterior and a decrease in the slope moving the point anterior. Thus, these changes can help biomechanically overcome PCL- and ACL-deficient knees, respectively [46]. While altering the slope in a ligament-intact knee may not significantly alter contact pressure of the femur on the tibia, altering tibial slope in a ligament-deficient knee can significantly increase the contact pressure of the knee. An increase in tibial slope of 5.5° will move the contact pressure up to 24% posteriorly [47].

11.4.2 High Tibial Osteotomy for the ACL-Deficient Knee

While the anterior cruciate ligament (ACL) is the main restraint to anterior translation of the tibia, malalignment of the extremity can leave an individual susceptible to ACL injury, as well as affect the stability of the ACL-deficient knee. Two patterns particularly exacerbate instability, as well as the rate of arthritic change in the ACL-deficient knee: osseous varus malalignment with medial compartment wear and increased tibial slope [48].

Increased tibial slope of both the medial and lateral plateaus has been shown to be a significant risk factor for ACL ruptures [49–55]. In conjunction with a natural posterior slope to the tibial plateau, the absence of the ACL results in anterior translation of the tibia with respect to the femur [56]. However, altering the tibial slope significantly changes the amount of anterior translation of the femur [46], with every 10° increase in posterior slope being associated with between a 3–6 mm increase in anterior translation of the tibia [44] and an increase of 4–5° resulting in a 2–3 mm increase in anterior translation [46].

Posterior tibial slope also appears to play a role in the rotational stability of the knee. Increased tibial slope is associated with increased Lachman and higher-grade pivot shift testing, and decreasing the slope is associated with increased rotational stability of the knee with decreased grade pivot shift, but no change in Lachman test [57, 58].

In conjunction with being intimately related to the stability of the knee, increased tibial slope ≥13° also puts increased stress on the medial compartment and leads to an increased risk of medial meniscus tears in the context of an ACL-deficient knee [59].

When comparing lateral closing-wedge (LCW) with medial opening-wedge (MOW) HTO in the ACL-deficient knee, LCW has shown to more reproducibly neutralize the posterior tibial slope and decrease anterior tibial translation in the context of ACL-deficiency, and, although it can also more often be associated with external tibial rotation and lateral patellar tilt, it may more significantly alter patellofemoral mechanics [60].

Generally, in ACL-deficient knees with varus alignment deformities, single-stage ACL reconstruction and HTO has demonstrated to be a reliable operation with good to excellent outcomes at long-term follow-up [61–63]. However, others argue that it may be prudent to stage HTO first and only perform the second stage of ACL reconstruction 6–12 months following surgery if instability persists. Especially in older patients, HTO alone can be an excellent, reproducible treatment option [64].

11.4.3 Deflexion Osteotomy for ACL Without Coronal Adjustment (Dejour)

There may be a role of HTO in the setting of appropriate coronal alignment of the knee. Dejour et al. recommend considering a deflexion osteotomy without coronal plane adjustment for patients with two or more failed ACL reconstructions and a pathologic tibial slope (generally ≥12°). In order to perform this technique, an anterior longitudinal incision is made just medial to the tibial tuberosity, with elevation of the soft tissues posterior to Gerdy’s tubercle. Osteotomy is recommended from the superior margin of the patellar tendon insertion and directed in an inferior direction, in order to avoid involving the tibial tubercle in the osteotomy. A second cut is made parallel to the joint line up to the posterior tibial cortex, which is then wedged for a goal slope of between 3° and 5° distally, with 1 mm of opening being equivalent to 1° of slope. Excellent results were reported at 4 years in conjunction with ACL reconstruction, with no revisions required and no postoperative complications, and only two of nine patients develop worsening in osteoarthritis at final follow-up [41]. An anterior closing wedge osteotomy, which involves the tibial tubercle, is also described. Although PTS was only improved from 13.6° preoperatively and 9.2° postoperatively in their series, they report excellent clinical outcomes at final follow-up [65].

11.4.4 High Tibial Osteotomy for the PCL-Deficient Knee

While decreasing the tibial slope can help improve the position of the tibiofemoral joint in the ACL-deficient knee, the converse is true in the PCL-deficient knee. Patients with PCL deficiency lack a restraint to posterior translation of the tibia on the femur. The degree of sag is related to the grade of PCL lesion, with grade I being 1–5 mm, grade II being 5–10 mm, and grade III being >10 mm. With loading, the change in resting position results in abnormal kinematics at the knee joint, which, over time, leads to increased strain and injury to the meniscus and wear due to higher contact pressures, especially in the anterior medial compartment [66, 67]. Furthermore, the posterior sag of the tibia can also result in osteoarthritic changes in the patellofemoral joint, particularly the lateral facet, with contact pressures in this joint increasing up to 16% [67].

PCL deficiency results in a posterior shift of the tibial resting position of about 8.4 mm at 90° flexion compared with a normal knee [68], which clinically manifests as posterior sag. However, slope altering osteotomy can help normalize contact pressures. By increasing the tibial slope from 9° to 14°, resting position is moved anteriorly 4 mm at 90° flexion and moves further anteriorly with axial compressive load [68]. As a result, load is transferred in a more anatomic nature with increased slope in the PCL-deficient knee, despite the absence of ligamentous stability.

Traditionally, closing wedge lateral HTO and dome osteotomies are thought to provide minimal opportunity to decrease the tibial slope [69], so medial opening wedge high tibial osteotomy has become the mainstay of treatment for PCL-deficient knees. The degree of correction in the sagittal plan can be evaluated by measuring the gap created by the osteotomy. It is important to consider that the shape of the proximal anteromedial tibia cortex is triangular and intersects the posterior cortex at about 45°, while the lateral tibial cortex is nearly perpendicular to the posterior tibia. As a result, an equal anterior and posterior osteotomy gap results in an increased slope, while slope does not change if the anterior gap is smaller than posterior [70]. Thus, for every 1 mm increase in the anterior gap, the posterior slope increases by about 2° [71].

Opening wedge HTO in the varus PCL-deficient knee has been shown to provide good clinical outcomes. Generally, the recommendation for varus PCL-deficient knees is to perform the osteotomy alone and only proceed with ligamentous reconstruction after 6–8 months if there is continued instability following corrective osteotomy [72]. However, further long-term studies are needed to truly evaluate the relationship between this treatment method and prevention of the progression of osteoarthritis.

11.4.5 Double and Triple Varus Knee

In order to classify abnormalities of instability and alignment of the knee, Noyes et al. created the terminology for primary varus, double varus, and triple varus. Primary varus is simply the varus osseous anatomy of the tibiofemoral joint, such as occurs with medial meniscectomy or medial compartment degenerative wear. Double-varus alignment occurs with the combination of varus tibiofemoral osseous alignment in combination of insufficiency of the lateral soft tissues of the knee, including the posterolateral ligament complex. Triple varus of the knee occurs when increased external tibial external rotation and hyperextension of the knee (varus recurvatum) is found in conjunction with the varus osseous alignment of the knee and insufficiency of the lateral soft tissues. This is often found with a significant varus thrust with gait [73, 74]. Furthermore, chronic triple varus is often found in conjunction with medial compartment posteromedial wear, due to chronic anterior subluxation of the tibia [75].

Much like in the PCL-deficient knee, tibial slope plays a critical role in stability of the triple varus knee, but it may not fully restore rotational stability. The literature demonstrates that combined sectioning of the PCL and PLC results in a 10.5 mm increase in posterior drawer, 15.5 mm increase in dial test at 30°, and 14.5 mm increase in dial test at 90°. Increasing the posterior slope by 5° tends to reduce translation by about 3 mm but does not have a significant impact on rotational stability when assessed by dial testing [76].

In the case of double or triple varus, HTO should always be performed before ligamentous reconstruction, and consideration should be given to wait 6–8 months to reliably evaluate the degree of instability imparted by the soft tissue injury.

Long-term varus stress, the stretched lateral structures, and proper tensioning of the soft tissues require the changes to the osseous anatomy to be performed first [74].

11.4.6 Take-Home Points

1. Posterior tibial slope is generally accepted to be within the range from 6° to 11° in the sagittal plane, and it is generally believed to be pathologic if it is ≥12°.
2. High tibial osteotomy can be utilized effectively for management of the ACL-deficient knee, with or without ACL reconstruction. Care should be paid to the PTS, with slopes ≥12° being an indication for altering slope.
3. Opening wedge HTO is generally preferred for PCL-deficient knees, due to a favorable ability to increase tibial slope. Slope should be normalized, and with every 1 mm increase in the anterior gap, the posterior slope increases by about 2°.
4. PCL deficiency can be found in conjunction with PLC injuries resulting posterolateral instability, defined as triple varus (osseous varus, incompetence of lateral soft tissues, external rotation, and hyperextension due to absence of PCL).
5. In the absence of coronal deformity, sagittal correction of tibial slope with a deflexion osteotomy should be considered in multiple failed ACL reconstructions with pathologic tibial slope ≥12°.

11.5 Simultaneous Bilateral High Tibial Osteotomy With Early Full Weight-Bearing Exercise

Many patients with osteoarthritis of the knee have symmetrical involvement and thus require a bilateral operation. Commonly, two-stage high tibial osteotomy has been performed during the same or in separate hospitalizations. Although simultaneous bilateral total knee arthroplasty is a common procedure these days, there is little report of simultaneous bilateral HTO. Because bone union takes a long time after conventional closed-wedge HTO surgery, patients are restricted from weight bearing for an extended period. However, development of open wedge HTO (OWHTO) and hybrid closed-wedge HTO (hybrid HTO) realized simultaneous bilateral HTO safely. The advantages of an HTO simultaneously performed for both knees in one operation with a single administration of anesthesia include the shortening of the hospitalization period, reduced costs, and lower anesthetic risks. Optimal postoperative rehabilitation following simultaneous bilateral HTO also allows early full weight bearing without any support, which can prevent the aggravation of osteoporosis and dementia. In OWHTO, combination of using beta-tricalcium phosphate (β-TCP, porosity of 60%) wedges and angle-stable plate fixation improved initial stability [77]. In hybrid HTO, a novel technique and new plate system realized early full weight bearing [78].

11.5.1 Postoperative Rehabilitation

The day after surgery, all patients are permitted standing exercise with full weight bearing, range of motion, and also muscle strengthening exercises are started actively and passively. Two days after surgery, partial weight bearing starts with the use of parallel bars. One or 2 weeks after surgery, every patient is allowed full weight bearing with or without a small cane according to their knee pain. From 3 to 6 months after surgery, patients are permitted to engage in sports activities. However, in OWHTO, rehabilitation schedules will be changed if an unstable type of lateral hinge fracture happens during surgery.

11.6 Open Wedge High Tibial Osteotomy for Spontaneous Osteonecrosis of the Knee

Spontaneous osteonecrosis of the medial condyle of the knee (SONK) occurs often in middle-aged patients and appears unilaterally in the medial femoral condyle of the knee. The clinical presentation of SONK includes the sudden onset of acute severe pain in the knee, which generally worsens at night.

11.6.1 Surgical Technique

Intra-articular procedures were performed arthroscopically. Damaged cartilage tissue was removed completely, the SONK lesion was curetted, and drilling of the necrotic area with a Kirschner wire of 1.6 mm was then performed. After that, OWHTO is performed and fixed with an angle-stable plate [79].

11.6.2 Treatment for SONK

SONK is a rare disease of the adult knee that was first described by Albäck in 1968 [80]. Various causes have been proposed for this disease, including an insufficiency fracture, a microfracture of the subchondral bone, degeneration of the meniscus, and vascular insufficiency in the distal femoral condyle [81, 82]. However, the precise etiology remains unknown. The incidence of SONK is also unknown, but the involvement of the knee has been reported to be approximately 10%. The clinical manifestation of SONK commonly includes the sudden onset of acute and severe pain which is frequently worse during the night and at rest. The clinical symptoms of SONK present typically in middle-aged and elderly patients as mild synovitis, mild effusion, and a minimal loss of range of motion [83]. These symptoms can therefore be difficult to distinguish from intra-articular diseases such as OA and meniscal tears.

Treatments for SONK, including conservative therapies, have been developed and most involve surgery. Some clinicians have advocated joint-preserving surgical procedures, such as core decompression and arthroscopic debridement, and reported that these procedures were successful in the early stage (no condyle collapse, no osteoarthrosis). There are other treatment options, such as unicompartmental knee arthroplasty (UKA) and total knee arthroplasty (TKA) for patients with severe osteoarthritis (Koshino's stage IV).

Aglietti et al. have reported that, in a cohort of 31 knees with SONK treated by HTO, 87% of the patients had a satisfactory evaluation with an average follow-up period of more than 6 years and that the ideal postsurgical FTA is 170° (10° anatomical valgus) [81]. Koshino et al. have reported that bone grafting or drilling into the necrotic lesion is effective in promoting healing in cases of osteonecrosis and recommend that surgical treatment is most effective when undertaken prior to the onset of osteoarthritic changes [83].

SONK is an acute disease whereas OA is a chronic disorder. Hence, the normal function of the knee, including range of motion of the knee joint, is not impaired in patients suffering from SONK as opposed to cases of osteoarthritis. Maintenance of a good range of knee motion is one of the most important considerations following knee surgery. However, there are currently few reports that describe the range of motion of the knee and cartilage regeneration in detail after a joint preservation surgery, HTO, for the treatment of SONK.

References

1. Jakopec M, Harris SJ, Rodriguez y Baena F, Gomes P, Cobb J, Davies BL. The first clinical application of a "hands-on" robotic knee surgery system. Comput Aided Surg. 2001;6(6):329–39.
2. Cobb JP, Kannan V, Brust K, Thevendran G. Navigation reduces the learning curve in resurfacing total hip arthroplasty. Clin Orthop Relat Res. 2007;463:90–7.
3. Henckel J, Richards R, Lozhkin K, Harris S, Rodriguez y Baena FM, ARW B, et al. Very low-dose computed tomography for planning and outcome measurement in knee replacement. The imperial knee protocol. J Bone Joint Surg Br. 2006;88(11):1513–8.
4. Duffell LD, Mushtaq J, Masjedi M, Cobb JP. The knee adduction angle of the osteo-arthritic knee: a comparison of 3D supine, static and dynamic alignment. Knee. 2014;21(6):1096–100.
5. Victor JMK, Bassens D, Bellemans J, Gürsu S, Dhollander AAM, Verdonk PCM. Constitutional varus does not affect joint line orientation in the coronal plane knee. Clin Orthop Relat Res. 2014;472(1):98–104.

6. Bellemans J, Colyn W, Vandenneucker H, Victor J. The Chitranjan Ranawat Award: is neutral mechanical alignment normal for all patients? The concept of constitutional varus. Clin Orthop Relat Res. 2012;470(1):45–53.
7. Cooke TD, Scudamore RA, Bryant JT, Sorbie C, Siu D, Fisher B. A quantitative approach to radiography of the lower limb. Principles and applications. J Bone Joint Surg Br. 1991;73(5):715–20.
8. Sharma L, Song J, Dunlop D, Felson D, Lewis CE, Segal N, et al. Varus and valgus alignment and incident and progressive knee osteoarthritis. Ann Rheum Dis. 2010;69(11):1940–5.
9. Felson DT, Jordan JM, Kington RS, Lane NE, Nevitt MC, Zhang Y, Sowers M, et al. Osteoarthritis: new insights. Part 1: The disease and its risk factors. Ann Intern Med. 2000;133(8):637–9.
10. Felson DT, Naimark A, Anderson J, Kazis L, Castelli W, Meenan RF. The prevalence of knee osteoarthritis in the elderly. The Framingham osteoarthritis study. Arthritis Rheum. 1987;30(8):914–8.
11. Brouwer RW, Jakma TSC, SMA B-Z, Verhagen AP, Verhaar J, et al. Osteotomy for treating knee osteoarthritis. Cochrane Database Syst Rev. 2005;1:CD004019.
12. Agneskirchner JD, Hurschler C, Wrann CD, Lobenhoffer P. The effects of valgus medial opening wedge high tibial osteotomy on articular cartilage pressure of the knee: a biomechanical study. Arthroscopy. 2007;23(8):852–61.
13. Cooke T, Pichora D, Siu D, Scudamore R, Bryant J. Surgical implications of varus deformity of the knee with obliquity of joint surfaces. J Bone Joint Surg Br. 1989;71-B(4):560.
14. Babis GC, An K-N, Chao EYS, Rand JA, Sim FH. Double level osteotomy of the knee: a method to retain joint-line obliquity. Clinical results. J Bone Joint Surg Am. 2002;84-A(8):1380–8.
15. Cooke TD, Sled EA, Scudamore RA. Frontal plane knee alignment: a call for standardized measurement. J Rheumatol. 2007;34(9):1796.
16. Paley D. Malalignment and malorientation in the frontal plane. In: Principles of deformity correction. Berlin: Springer; 2002. p. 19–30.
17. Paley D. Normal lower limb alignment and joint orientation. In: Principles of deformity correction. Berlin: Springer; 2002. p. 1–18
18. Saithna A, Kundra R, Modi CS, Getgood A, Spalding T. Distal femoral varus osteotomy for lateral compartment osteoarthritis in the valgus knee. A systematic review of the literature. Open Orthop J. 2012;6:313–9.
19. Rosso F, Margheritini F. Distal femoral osteotomy. Curr Rev Muscoskelet Med. 2014;7(4):302–11.
20. Felson DT, Niu J, Gross KD, Englund M, Sharma L, Cooke TD, et al. Valgus malalignment is a risk factor for lateral knee osteoarthritis incidence and progression: findings from the multicenter osteoarthritis study and the osteoarthritis initiative. Arthritis Rheum. 2013;65(2):355–62.
21. Nelissen EM, van Langelaan EJ, Nelissen RGHH. Stability of medial opening wedge high tibial osteotomy: a failure analysis. Int Orthop. 2010;34(2):217–23.
22. Chahla J, Mitchell JJ, Liechti DJ, Moatshe G, Menge TJ, Dean CS, et al. Opening- and closing-wedge distal femoral osteotomy: a systematic review of outcomes for isolated lateral compartment osteoarthritis. Orthop J Sports Med. 2016;4(6):2325967116649901.
23. W-Dahl A, Robertsson O, Lidgren L. Surgery for knee osteoarthritis in younger patients. Acta Orthop. 2010;81(2):161–4.
24. Spahn G, Hofmann GO, von Engelhardt LV, Li M, Neubauer H, Klinger HM. The impact of a high tibial valgus osteotomy and unicondylar medial arthroplasty on the treatment for knee osteoarthritis: a meta-analysis. Knee Surg Sports Traumatol Arthrosc. 2013;21(1):96–112.
25. Floerkemeier S, Staubli AE, Schroeter S, Goldhahn S, Lobenhoffer P. Outcome after high tibial open-wedge osteotomy: a retrospective evaluation of 533 patients. Knee Surg Sports Traumatol Arthrosc. 2013;21(1):170–80.
26. Lobenhoffer P, Agneskirchner J, Zoch W. Die öffnende valgisierende osteotomie der proximalen tibia mit fixation durch einen medialen plattenfixateur. Orthopade. 2004;33:153–60.
27. Brinkman J-M, Lobenhoffer P, Agneskirchner JD, Staubli AE, Wymenga AB, van Heerwaarden RJ. Osteotomies around the knee: patient selection, stability of fixation and bone healing in high tibial osteotomies. J Bone Joint Surg Br. 2008;90-B(12):1548–57.
28. Han SB, Lee DH, Shetty GM, Chae DJ, Song JG, Nha KW. A "safe zone" in medial open-wedge high tibia osteotomy to prevent lateral cortex fracture. Knee Surg Sports Traumatol Arthrosc. 2013;21(1):90–5.
29. Vanadurongwan B, Siripisitsak T, Sudjai N, Harnroongroj T. The anatomical safe zone for medial opening oblique wedge high tibial osteotomy. Singap Med J. 2013;54(2):102.
30. van Heerwaarden R, Najfeld M, Brinkman M, Seil R, Madry H, Pape D. Wedge volume and osteotomy surface depend on surgical technique for distal femoral osteotomy. Knee Surg Sports Traumatol Arthrosc. 2013;21(1):206–12.
31. Brinkman J-M, Hurschler C, Agneskirchner JD, Freiling D, van Heerwaarden RJ. Axial and torsional stability of supracondylar femur osteotomies: biomechanical comparison of the stability of five different plate and osteotomy configurations. Knee Surg Sports Traumatol Arthrosc. 2011;19(4):579–87.
32. Hofmann S, Lobenhoffer P, Staubli A, Van Heerwaarden R. Osteotomien am kniegelenk bei monokompartmentarthrose. Orthopade. 2009;38(8):755–70.
33. Bonasia DE, Governale G, Spolaore S, Rossi R, Amendola A. High tibial osteotomy. Curr Rev Muscoskelet Med. 2014;7:292.

34. Sanna M, Sanna C, Caputo F, Piu G, Salvi M. Surgical approaches in total knee arthroplasty. Joints. 2013;1(2):34–44.
35. Farouk O, Krettek C, Miclau T, Schandelmaier P, Guy P, Tscherne H. Minimally invasive plate osteosynthesis and vascularity: preliminary results of a cadaver injection study. Injury. 1997;28:A7–12.
36. Farouk O, Krettek C, Miclau T, Schandelmaier P, Tscherne H. The topography of the perforating vessels of the deep femoral artery. Clin Orthop Relat Res. 1999;368:255–9.
37. Farouk O, Krettek C, Miclau T, Schandelmaier P, Tscherne H. Effects of percutaneous and conventional plating techniques on the blood supply to the femur. Arch Orthop Trauma Surg. 1998;117(8):438–41.
38. Amis AA. Biomechanics of high tibial osteotomy. Knee Surg Sports Traumatol Arthrosc. 2013;21(1):197–205.
39. Marti RK, van Heerwaarden RJ. Osteotomies for posttraumatic deformities. Stuttgart: George Thieme Verlag; 2008. 704p.
40. Kerkhoffs GMM, Rademakers MV, Altena M, Marti RK. Combined intra-articular and varus opening wedge osteotomy for lateral depression and valgus malunion of the proximal part of the tibia. J Bone Joint Surg Am. 2008;90(6):1252–7.
41. Dejour D, Saffarini M, Demey G, Baverel L. Tibial slope correction combined with second revision ACL produces good knee stability and prevents graft rupture. Knee Surg Sports Traumatol Arthrosc. 2015;23(10):2846–52.
42. Hohmann E, Bryant A, Reaburn P, Tetsworth K. Is there a correlation between posterior tibial slope and non-contact anterior cruciate ligament injuries? Knee Surg Sports Traumatol Arthrosc. 2011;19(Suppl 1):S109–14.
43. Li Y, Hong L, Feng H, Wang Q, Zhang J, Song G, et al. Posterior tibial slope influences static anterior tibial translation in anterior cruciate ligament reconstruction: a minimum 2-year follow-up study. Am J Sports Med. 2014;42(4):927–33.
44. Dejour H, Bonnin M. Tibial translation after anterior cruciate ligament rupture. Two radiological tests compared. J Bone Joint Surg Br. 1994;76(5):745–9.
45. Torzilli PA, Deng X, Warren RF. The effect of joint-compressive load and quadriceps muscle force on knee motion in the intact and anterior cruciate ligament-sectioned knee. Am J Sports Med. 1994;22(1):105–12.
46. Giffin JR, Vogrin TM, Zantop T, Woo SLY, Harner CD. Effects of increasing tibial slope on the biomechanics of the knee. Am J Sports Med. 2004;32(2):376–82.
47. Rodner CM, Adams DJ, Diaz-Doran V, Tate JP, Santangelo SA, Mazzocca AD, et al. Medial opening wedge tibial osteotomy and the sagittal plane: the effect of increasing tibial slope on tibiofemoral contact pressure. Am J Sports Med. 2006;34(9): 1431–41.
48. Crawford MD, Diehl LH, Amendola A. Surgical management and treatment of the anterior cruciate ligament-deficient knee with malalignment. Clin Sports Med. 2017;36(1):119–33.
49. Feucht MJ, Mauro CS, Brucker PU, Imhoff AB, Hinterwimmer S. The role of the tibial slope in sustaining and treating anterior cruciate ligament injuries. Knee Surg Sports Traumatol Arthrosc. 2013;21(1):134–45.
50. Hashemi J, Chandrashekar N, Mansouri H, Gill B, Slauterbeck JR, Schutt RC, et al. Shallow medial tibial plateau and steep medial and lateral tibial slopes: new risk factors for anterior cruciate ligament injuries. Am J Sports Med. 2010;38(1):54–62.
51. Hudek R, Fuchs B, Regenfelder F, Koch PP. Is noncontact ACL injury associated with the posterior tibial and meniscal slope? Clin Orthop Relat Res. 2011;469(8):2377–84.
52. Rahnemai-Azar AA, Yaseen Z, van Eck CF, Irrgang JJ, Fu FH, Musahl V. Increased lateral tibial plateau slope predisposes male college football players to anterior cruciate ligament injury. J Bone Joint Surg Am. 2016;98(12):1001–6.
53. Stijak L, Herzog RF, Schai P. Is there an influence of the tibial slope of the lateral condyle on the ACL lesion? A case-control study. Knee Surg Sports Traumatol Arthrosc. 2008;16(2):112–7.
54. Vyas S, van Eck CF, Vyas N, Fu FH, Otsuka NY. Increased medial tibial slope in teenage pediatric population with open physes and anterior cruciate ligament injuries. Knee Surg Sports Traumatol Arthrosc. 2011;19(3):372–7.
55. Zeng C, Cheng L, Wei J, Gao S, Yang T, Luo W, et al. The influence of the tibial plateau slopes on injury of the anterior cruciate ligament: a meta-analysis. Knee Surg Sports Traumatol Arthrosc. 2014;22(1):53–65.
56. Shoemaker SC, Markolf KL. The role of the meniscus in the anterior-posterior stability of the loaded anterior cruciate-deficient knee. Effects of partial versus total excision. J Bone Joint Surg Am. 1986;68(1):71–9.
57. Brandon ML, Haynes PT, Bonamo JR, Flynn MI, Barrett GR, Sherman MF. The association between posterior-inferior tibial slope and anterior cruciate ligament insufficiency. Arthroscopy. 2006;22(8):894–9.
58. Voos JE, Suero EM, Citak M, Petrigliano FP, Bosscher MRF, Citak M, et al. Effect of tibial slope on the stability of the anterior cruciate ligament-deficient knee. Knee Surg Sports Traumatol Arthrosc. 2012;20(8):1626–31.
59. Lee J-J, Choi Y-J, Shin K-Y, Choi C-H. Medial meniscal tears in anterior cruciate ligament-deficient knees: effects of posterior tibial slope on medial meniscal tear. Knee Surg Relat Res. 2011;23(4):227–30.
60. Ranawat AS, Nwachukwu BU, Pearle AD, Zuiderbaan HA, Weeks KD, Khamaisy S. Comparison of lateral closing-wedge versus medial opening-wedge high tibial osteotomy on knee joint alignment and kinematics in the ACL-deficient knee. Am J Sports Med. 2016;44(12):3103–10.

61. Boss A, Stutz G, Oursin C, Gächter A. Anterior cruciate ligament reconstruction combined with valgus tibial osteotomy (combined procedure). Knee Surg Sports Traumatol Arthrosc. 1995;3(3):187–91.
62. Demange MK, Camanho GL, Pécora JR, Gobbi RG, Tirico LEP, da Mota e Albuquerque RF. Simultaneous anterior cruciate ligament reconstruction and computer-assisted open-wedge high tibial osteotomy: a report of eight cases. Knee. 2011;18(6):387–91.
63. Neuschwander DC, Drez D, Paine RM. Simultaneous high tibial osteotomy and ACL reconstruction for combined genu varum and symptomatic ACL tear. Orthopedics. 1993;16(6):679–84.
64. Lattermann C, Jakob RP. High tibial osteotomy alone or combined with ligament reconstruction in anterior cruciate ligament-deficient knees. Knee Surg Sports Traumatol Arthrosc. 1996;4(1):32–8.
65. Sonnery-Cottet B, Mogos S, Thaunat M, Archbold P, Fayard J-M, Freychet B, et al. Proximal tibial anterior closing wedge osteotomy in repeat revision of anterior cruciate ligament reconstruction. Am J Sports Med. 2014;42(8):1873–80.
66. MacDonald P, Miniaci A, Fowler P, Marks P, Finlay B. A biomechanical analysis of joint contact forces in the posterior cruciate deficient knee. Knee Surg Sports Traumatol Arthrosc. 1996;3(4):252–5.
67. Skyhar MJ, Warren RF, Ortiz GJ, Schwartz E, Otis JC. The effects of sectioning of the posterior cruciate ligament and the posterolateral complex on the articular contact pressures within the knee. J Bone Joint Surg Am. 1993;75(5):694–9.
68. Giffin JR, Stabile KJ, Zantop T, Vogrin TM, Woo SL-Y, Harner CD. Importance of tibial slope for stability of the posterior cruciate ligament deficient knee. Am J Sports Med. 2007;35(9):1443–9.
69. Maquet P. Valgus osteotomy for osteoarthritis of the knee. Clin Orthop Relat Res. 1976;120:143–8.
70. Noyes FR, Goebel SX, West J. Opening wedge tibial osteotomy: the 3-triangle method to correct axial alignment and tibial slope. Am J Sports Med. 2005;33(3):378–87.
71. Dugdale TW, Noyes FR, Styer D. Preoperative planning for high tibial osteotomy. The effect of lateral tibiofemoral separation and tibiofemoral length. Clin Orthop Relat Res. 1992;274:248–64.
72. Naudie DDR, Amendola A, Fowler PJ. Opening wedge high tibial osteotomy for symptomatic hyperextension-varus thrust. Am J Sports Med. 2004;32(1):60–70.
73. Noyes F, Simon R. The role of high tibial osteotomy in the anterior cruciate ligament-deficient knee with varus alignment. In: DeLee JC, Drez D, editors. Orthopaedic sports medicine principles and practice. Philadelphia: W.B. Saunders; 1994. p. 1401–43.
74. Noyes FR, Barber-Westin SD, Hewett TE. High tibial osteotomy and ligament reconstruction for varus angulated anterior cruciate ligament-deficient knees. Am J Sports Med. 2000;28(3):282–96.
75. Bonin N, Ait Si Selmi T, Donell ST, Dejour H, Neyret P. Anterior cruciate reconstruction combined with valgus upper tibial osteotomy: 12 years follow-up. Knee. 2004;11(6):431–7.
76. Petrigliano FA, Suero EM, Voos JE, Pearle AD, Allen AA. The effect of proximal tibial slope on dynamic stability testing of the posterior cruciate ligament- and posterolateral corner-deficient knee. Am J Sports Med. 2012;40(6):1322–8.
77. Takeuchi R. Simultaneous bilateral opening-wedge high tibial osteotomy with early full weight-bearing exercise. Knee Surg Sports Traumatol Arthrosc. 2008;16:1030–7.
78. Takeuchi R, Ishikawa H, Miyasaka Y, Sasaki Y, Kuniya T, Tsukahara S. A novel closed-wedge high tibial osteotomy procedure to treat osteoarthritis of the knee: hybrid technique and rehabilitation measures. Arthrosc Tech. 2014;3(4):e431–7.
79. Takeuchi R. Clinical results and radiological evaluation of opening wedge high tibial osteotomy for spontane osteonecrosis of the knee. Knee Surg Sports Traumatol Arthrosc. 2009;17:361–8.
80. Ahlbäck S. Spontaneous osteonecrosis of the knee. Arthritis Rheum. 1968;11:705–33.
81. Aglietti P, Insall JN, Buzzi R, Deschamps G. Idiopathic osteonecrosis of the knee. Aetiology, prognosis and treatment. J Bone Joint Surg Br. 1983;65(5):588–97.
82. Forst J. Spontaneous osteonecrosis of the femoral condyle: causal treatment by early core decompression. Arch Orthop Trauma Surg. 1988;117:18–22.
83. Koshino T. The treatment of spontaneous osteonecrosis of the knee by high tibial osteotomy with and without bone-grafting or drilling of the lesion. J Bone Joint Surg Am. 1982;64(1):47–58.

Patellofemoral Joint Instability: Where Are We in 2018?

12

Deiary Kader, David Dejour, Rebecca Stoner,
Stefano Pasqualotto, Bobby Anand,
Mirco Herbort, Jonathan Eldridge, Nick Caplan,
and Elizabeth Arendt

12.1 Introduction

Acute patella dislocation makes up 2–3% of all acute knee injuries, with a higher incidence in younger and athletic patients [1–3]. Risk of re-dislocation following first-time injury is 17–49% [2], rising to 44–71% in patients younger than 20 years [1, 3].

The stability of the patellofemoral (PF) joint is derived from a combination of local, distant, static and dynamic factors. Locally, static stability is provided by bone/cartilage geometry and ligaments, whilst dynamic stability is primarily maintained by the extensor muscles including vastus medialis obliquus (VMO) [4, 5].

The principle distant static factors are femoral anteversion (normal 5–15°), knee rotation (normal 3°) and external tibial torsion (25–30°), whilst the main distant dynamic factors are the iliotibial band complex, hip abductors/external rotators and foot malrotation such as excessive subtalar joint pronation, which generates a dynamic valgus force vector that displaces the patella laterally [6–9].

The bone geometry and cartilaginous structures of the patella and trochlea account for most of the patellofemoral joint stability in deeper knee flexion. The medial retinaculum consists of three distinct layers: investing fascia, medial patellofemoral ligament (MPFL) and superficial medial collateral ligament (MCL) and deep MCL and joint capsule. The MPFL is regarded as the primary passive stabiliser of the patella in early knee flexion (20–30°) [10]. It guides the patella into the trochlear groove and provides anywhere between 50 and 80% of the stability required to prevent lateral patella displacement [4, 10–12].

The MPFL has femoral and patellar attachments. It is well accepted that the MPFL becomes

---

D. Kader (✉) • R. Stoner
Academic Orthopaedic Department, South West London Elective Orthopaedic Centre, Epsom, UK
e-mail: deiary.kader@gmail.com

D. Dejour • S. Pasqualotto
Department of Orthopaedic & Sport-Medicine, Surgery Lyon-Ortho-Clinic,
Lyon, France

B. Anand
Croydon University Hospital, London, UK

Academic Orthopaedic Department,
South West London Elective Orthopaedic Centre,
Epsom, UK

M. Herbort
Klinik für Unfall-, Hand-, und Wiederherstellungschirurgie, Universitätsklinikum Münster, Münster, Germany

J. Eldridge
Bristol Royal Infirmary, University Hospitals Bristol,
Bristol, UK

N. Caplan
Faculty of Health Life Sciences, Northumbria University, Newcastle upon Tyne, UK

E. Arendt
Department of Orthopaedic Surgery, University of Minnesota, Minneapolis, MN, USA

G.M.M.J. Kerkhoffs et al. (eds.), *ESSKA Instructional Course Lecture Book*,
https://doi.org/10.1007/978-3-662-56127-0_12

conjoined with the deep portion of VMO before inserting into the upper two thirds of the medial patella (Fig. 12.1). However, there has been a lot of controversy regarding the femoral attachment site [13]. A previous anatomical study by Amis et al. in 2003 [14] concluded that the MPFL originated from the origin of the medial epicondyle of the femur. Desio et al. found that the femoral origin of the MPFL is 8.8 mm anterior to the line continuous with the posterior cortex of the femur and 2.6 mm proximal to a perpendicular line at the level of the proximal aspect of the Blumensaat line [10]. Schöttle [15], in his cadaver study, defined a radiographic point representing the MPFL femoral attachment. This was described on a lateral radiograph, with both posterior condyles projected in the same plane, as 1 mm anterior to the posterior cortex extension line, 2.5 mm distal to the posterior origin of the medial femoral condyle and proximal to the level of the posterior point of the Blumensaat line. However, McCarthy et al. reported that MPFL reconstruction using Schöttle's point does not correlate with improved functional outcomes [16].

Recent cadaveric dissections performed by this chapter's first author [13, 17, 18] showed that the MPFL attaches to a broad area between the medial epicondyle and the adductor tubercle on the femur (Fig. 12.2). When the centre of the attachment was marked radiologically, it corresponded to a point just anterior to the confluence of Blumensaat's line and the curving line off the posterior femoral cortex and posterior to the straight extension line from the posterior cortex in a true lateral radiograph of the knee (Fig. 12.3). Hence, it could be called the confluence point. This radiographic point is more than 5 mm distal and posterior to Schöttle's point [17–21] (Fig. 12.4). Interestingly, this point corresponds to the instant centre of knee rotation. This distinction between Schöttle's point and the confluence point is of paramount importance; hence, cadaver studies have shown that a

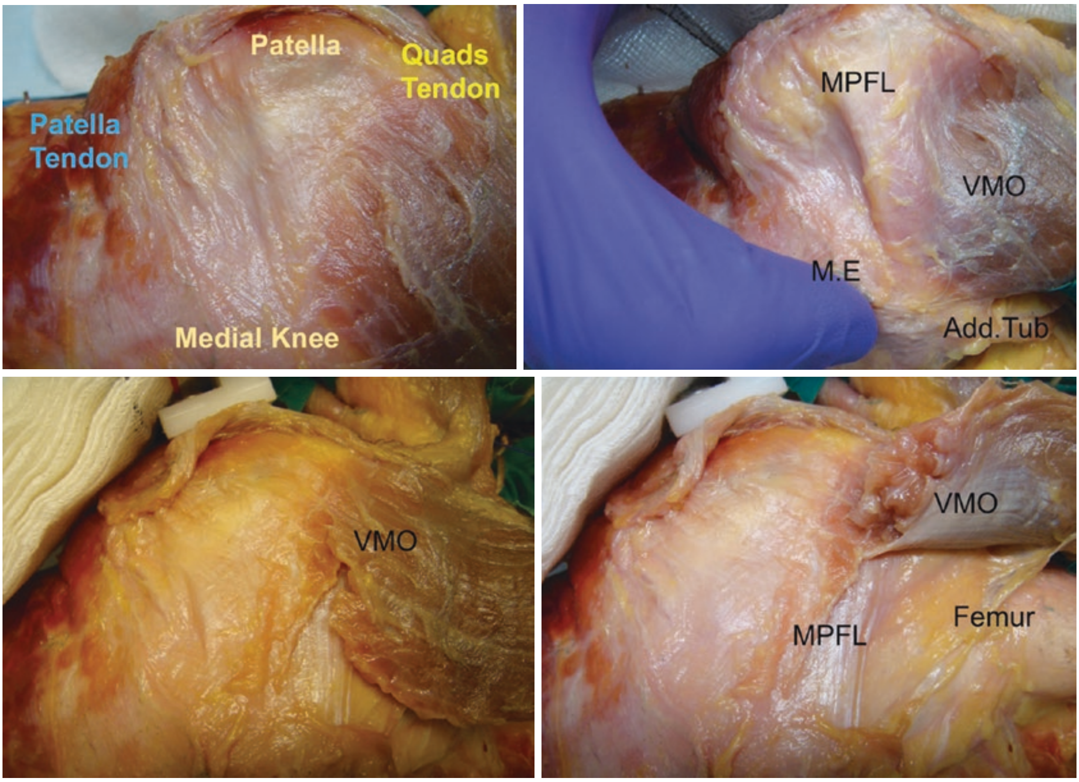


Fig. 12.1 Cadaveric dissections demonstrating that the MPFL attaches to a broad area between the medial epicondyle and the adductor tubercle

5 mm nonanatomic femoral attachment, either proximally or distally, causes a significant increase in medial contact pressures and medial patella tilt in flexion and extension, respectively [13]. The difference could be attributed to the quality of the cadavers and dissection techniques.

The aetiology of patellofemoral joint instability (PFJI) is complex and multifactorial. Several abnormal anatomical factors have been identified in patients with recurrent patella dislocation, including generalised hypermobility (24%) [22], patella hypermobility (51%) [22], increased femoral anteversion (27%), core and hip abductor weakness, abnormal knee rotation, trochlea dysplasia (53–71%), abnormal Q angle, patella alta (60–66%) [23], muscle and soft tissue imbalance, external tibial torsion and foot hyperpronation. In a recent magnetic resonance imaging (MRI)-based study, 58.3% of patients had multiple anatomical factors associated with recurrent patella dislocation [23].

The foundations for the management of PFJI have been laid out by the Lyonnaise school in their seminal paper, in which four principle factors were outlined, based on plain radiographs and slice imaging. These factors are patella height, patella tilt, trochlear groove-tibial tubercle distance (TT-TG) and trochlear morphology [5].

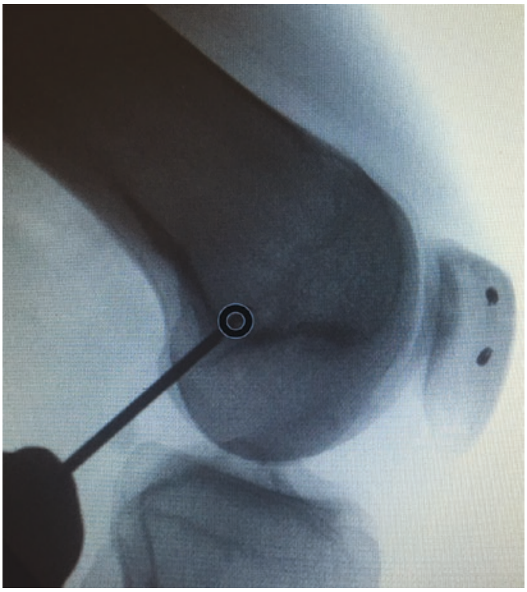


Fig. 12.2 True lateral intraoperative fluoroscopy image demonstrating the confluence point prior to drilling the femoral tunnel

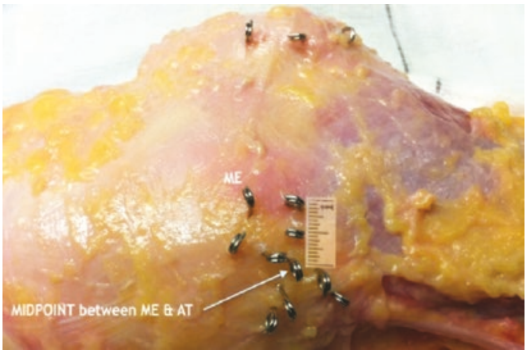


Fig. 12.3 Cadaveric dissection demonstrating pin marking details at various insertion points within 5 mm of each other to identify the optimum site for the femoral tunnel placement

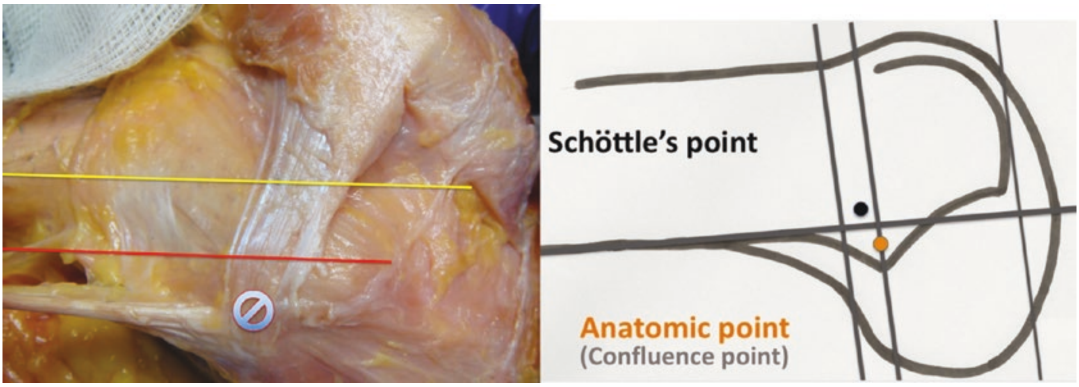


Fig. 12.4 Cadaveric dissection demonstrating the confluence point more than 5 mm distal and posterior to Schöttle's point

They divided PF disorders into three groups: objective patella instability, potential patella instability and painful patella syndrome. This study by Dejour et al. remains the largest follow-up in our literature on surgically treated patients with recurrent lateral patella dislocations.

In recent years there has been a renewed interest in PFJI, possibly related to the advances made in our understanding of various anatomic and dynamic factors that contribute to patella stability. The overall management of patella dislocation and instability has been linked with poor patient satisfaction, possibly due to a prolonged period of conservative treatment and the general tendency to delay surgical intervention [3].

12.1.1 Clinical Examination

Detailed clinical history and general hypermobility assessment by using the Beighton scoring system should be carried out. Patella examination typically includes the assessment of patella alignment (Q angle), height (alta/baja), hypermobility, dislocation in extension (reverse J sign), quadriceps function, hamstring tightness, para-patella tenderness, patella apprehension, trochlea depth in full flexion and PF joint crepitus.

The quadriceps angle (Q angle), first described by Brattström [24], represents the angle between the vector of action of the quadriceps and patella tendons. Traditionally, it is measured using the anterior superior iliac spine (ASIS), centre of the patella and centre of the tibial tuberosity as anatomical landmarks. With normal values estimated between 8 and 17° in males and 12 and 20° in females, an increased Q angle is thought to be associated with an increased risk of anterior knee pain and patella instability [25–27]. However, the Q angle has been found to be neither valid nor reliable as it can be affected by the anatomical points used to record the measurement and whether it is measured with a manual or digital goniometer [28]. Further, the measurement will be influenced by whether the patient is standing or supine, the rotation of the limb in relation to the pelvis, the degree of flexion of the knee and whether the quadriceps are relaxed or contracted [27, 29, 30]. Cooney et al. highlighted that the Q angle does not necessarily correlate with radiographic measures of patellar alignment (e.g. TT-TG). Therefore, Q angle should not be relied upon in isolation to identify PFJI [31].

12.1.2 Radiologic Assessment

Patella height is best assessed using a true lateral radiograph with the knee flexed to 30° according to the method of Caton-Deschamps (i.e. the ratio between the distance from the lower edge of the patella articular surface to the upper edge of the tibial plateau and the length of the patella articular surface) [32, 33]. A ratio of 1.2 or greater indicates patella alta, which predisposes the patient to patella instability due to late engagement of the patella in the trochlea as the knee flexes.

Rotational profile computed tomography (CT) scans [7] (Fig. 12.5) of the lower limbs in neutral rotation, as per Dejour's method [5], is very helpful in objectively assessing many anatomic factors that may contribute to the stability of the patella, such as femoral anteversion, knee rotation, external tibial torsion, tibial tuberosity-trochlear groove (TT-TG) distance, patella index, patella tilt, trochlea tilt and trochlea depth. The normal TT-TG distance is 2–9 mm, and it is generally accepted that a figure of >19 mm is pathological [34–36]. It is estimated that 42% of patients with PFJI have abnormal TT-TG [23]. Although TT-TG distance is regarded by many clinicians as one of the important measurements in assessing patella instability and deciding about distal realignment procedures, recent research has shown that it is not a decisive element in establishing therapeutic choices for instability [36, 37].

The TT-TG distance was originally called tibial tuberosity-patella groove (TT-PG) distance by Goutallier in 1978 [38]. The TT-PG distance was measured in three groups. The first group ($n = 16$) was aged over 65 years and had normal knees, the second group ($n = 30$) was aged under 65, suffering from PFJ arthritis, and the third group ($n = 24$) was aged under 65, suffering from patella dislocation. This was a descriptive paper on a

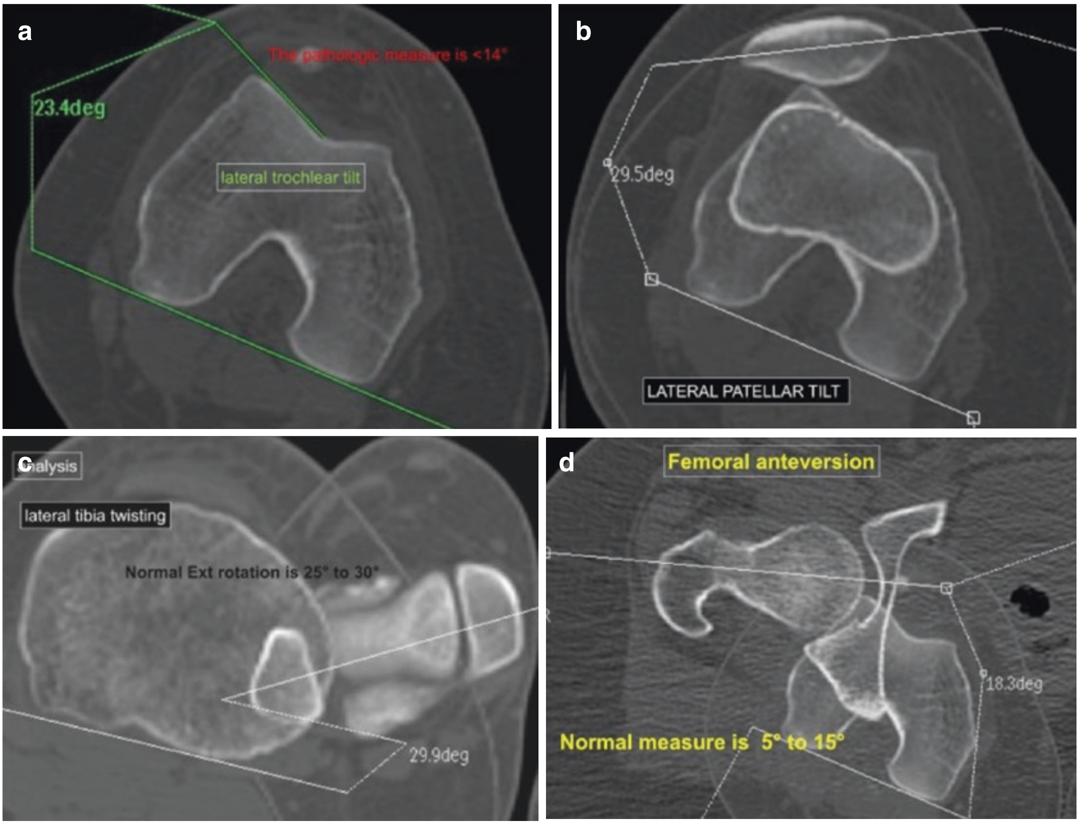


Fig. 12.5 Rotational profile CT images to demonstrate the multiple anatomical factors involved in destabilising the patella, including (**a**) lateral trochlear tilt, (**b**) lateral patellar tilt, (**c**) lateral tibia twisting and (**d**) femoral anteversion

heterogeneous population. Its methodology would have never passed the current stringent review process; thus, the TT-TG distance should be treated with caution based on this consideration alone. There are several potential problems with relying on TT-TG distance in isolation. There is a large variation in its normal value depending on patients' size and height. In a small person, a 20 mm distance will have a greater impact on PFJ kinematics in comparison with a larger person, as the TT-TG distance is recorded as an absolute distance rather than relative to the patient's knee size. The same values cannot be applied to both CT and MRI scans as the osseous and cartilaginous geometry of the patellofemoral joint frequently differ [39]. In addition, there is poor inter-rater reliability; measurement errors of 3–5 mm have been reported due to the difficulty in identifying the deepest point of the trochlea and the highest point of the tibial tuberosity, especially in dysplastic trochlea [36, 37]. Finally, the measurement is very much dependent on knee flexion angle and the weight-bearing status of the patient. Therefore, TT-TG distance should be interpreted with caution during clinical evaluation of patella instability [40].

Trochlear dysplasia has been linked to PFJI and was classified by Dejour based on trochlea morphology: type A, shallow trochlea; type B, flat or convex; type C, hypoplastic medial facet; and type D, asymmetrical facets with vertical links [41]. It is typically measured on a true lateral radiograph, with the knee flexed to 30°, at the point where the trochlear groove crosses both condyles, and this "crossing sign" was observed in 96% of patients with recurrent instability and in only 3% of controls [5]. Whilst dysplastic knees are correctly identified in the majority of the knees, low inter-rater reliability has been reported in the correct identification of trochlear morphology according to Dejour's classifications [42].

Despite a thorough clinical examination, radiographs, MRI and rotational profile CT, it is still difficult to quantify patella malalignment and malrotation. It is, therefore, recommended to use more than one clinical test and radiologic measurement to identify the main pathology that is causing the PFJI.

12.2 State-of-the-Art Treatment

12.2.1 Acute Patella Dislocation: Cast vs Early Immobilisation vs Surgery

Acute dislocation has been associated with osteochondral lesions in 49% of patients and with MPFL disruption in over 90–100% of patients [43–46]. There is high patient dissatisfaction after conservative treatment, with 58% reporting limitations in strenuous activities 6 months after treatment [47] and 55% of these patients failing to return to sporting activities. Chronic PFJI and recurrent dislocation may eventually lead to progressive cartilage damage if not treated adequately, and the risk of osteoarthritis (OA) has been found to be 35% after conservative treatment [48].

The best treatment of an isolated acute first patella dislocation is debatable. It is widely agreed that operative intervention is only recommended when there is evidence of a large osteochondral defects. In the past, isolated acute first patella dislocation was mainly treated conservatively, because older literature did not demonstrate any advantage of operative treatment in terms of re-dislocation rate [49–51].

Conservative treatment can take many forms, though most authors recommend an immobilising splint, cast or orthosis for 2–3 weeks, followed by physiotherapy focusing on building quadriceps. There is insufficient evidence to determine if weight-bearing restriction is necessary [51–54]. Most of the literature that is comparing operative with conservative treatment is unreliable due to variation in the reported surgical intervention. Long-term follow-up studies tend to include old-fashioned operative procedures that are no longer performed.

A recent systematic review and quantitative synthesis of literature found that re-dislocation rates were lower and short- to medium-term clinical outcomes were better after surgical treatment of primary acute patella dislocation, though no difference was seen in long-term follow-up [49]. A Cochrane review comparing surgical and non-surgical interventions reported that patients managed surgically had a significantly lower risk of recurrent dislocation following primary patella dislocation at 2–5 years follow-up. However, they concluded that adequately powered multicentre randomised controlled trials are needed [55].

Decision-making in management of acute patella dislocation, therefore, requires analysis of patient-specific instability predictors [56, 57]. Balcarek et al. suggested analysis of six parameters to determine the patellar instability severity score (PIS-score), which can identify patients who would benefit from operative management. The parameters are:

- Age
- Positive anamnesis of contralateral patella dislocation
- Patella tilt (<20°/>20°)
- Patella alta
- TT-TG distance
- Trochlea dysplasia

Patients with a PIS-score of 4 points or more have a higher risk of re-dislocation of the patella and, therefore, should receive operative treatment. As of yet, there are no long-term outcomes from the use of this classification, but individual analysis of patient factors in the decision-making process for operative or conservative treatment of dislocation and instability appears reasonable and is recommended by many experienced surgeons.

12.2.2 Is MPFL Reconstruction the Procedure of Choice?

A common current approach to patella stabilisation for recurrent lateral patella dislocation is MPFL reconstruction, for which many different techniques have been described [58].

The clinical challenge remains, however, in defining when isolated MPFL reconstruction (without a bony procedure) would provide consistent surgical success. To answer this question, studies are required to include evaluation and documentation of preoperative physical examination and imaging factors and relate these factors to measured surgical outcomes.

The vast majority of publications on "isolated MPFL reconstructions" define a relatively homogeneous population without excessive anatomic imaging factors that have resulted in successful surgical outcomes [59]. Current literature on MPFL reconstruction does not allow for strong evidence-based surgical decisions for those patients with anatomic instability factors above the previously established thresholds laid down by Dejour et al. [5], primarily due to the lack of reporting and/or inconsistent recording of pre- and postoperative anatomic variables.

Another barrier to clarity in the clinical approach to surgical management of lateral patella dislocation is the lack of specificity in the imaging measurements that are central to our current clinical algorithms. A recent systematic search with meta-analysis of MRI measurements revealed a wide range of imaging values within both controls and PFJI groups [60]. This showed that appropriate abnormality thresholds exist for anatomic patella instability MRI factors within groups of patients classified as having PFJI, indicating sensitivity. The wide range in the majority of measurements, especially in the control group, suggested poor specificity in most MRI measurements, indicating that these imaging measurements cannot be used in the absence of an appropriate history and physical examination to discriminate between patients with and without PFJI.

The clinical challenge that remains is detailing the anatomic thresholds for surgical correction of anatomic patella instability factors such as patella height, trochlear dysplasia and increased quadriceps vector (e.g. increased TT-TG) and determining which surgical procedure is most appropriate for correction of such factors. The question remains as to whether it is necessary to correct all identified factors.

The following guidelines are offered:

An ideal candidate for an isolated MPFL reconstruction, without bony procedures, should have a history consistent with recurrent dislocation and a physical examination demonstrating excessive lateral patella translation, with minimal or absent pain between episodes of instability and a normal or low-grade dysplastic trochlea (e.g. type A Dejour classification). There should be no radiological evidence of lateral PF load, tubercle sulcus angle between 0° and 5° valgus, and no excessive patella height (reasonable overlap of patella and trochlea surfaces on sagittal MRI measured by patella-trochlea index [61]). The Caton-Deschamps index up to 1.4 can be acceptable, except where there is a very short trochlea or significant knee hyperextension.

Where lateral retinacular tightness is present on clinical examination with TT-TG less than 20 mm, lateral retinacular lengthening, with or without partial lateral facetectomy, could be recommended in order to unload the lateral patellofemoral joint. However, where there is no retinacular tightness and TT-TG is more than 20 mm, medial tibial tubercle osteotomy is preferred.

Ultimately, surgical decisions involve a blend of imaging and physical examination features, combined with patient expectation and surgeon's experience and judgement.

12.2.3 Which Bony Procedure?

A large percentage of patients who suffer from PFJI can benefit from soft tissue procedures. However, in some patients this is not enough. Bony procedures are critical tools to address the underlying pathology and to ensure a successful outcome. It is clear that, in correctly selected patients and after careful technical considerations, soft tissue procedures can have high success rates, and this is evident by the variation in outcomes reported in the literature. After technical failures, one of the most important reasons for failure is often because a bony procedure was indicated, and an isolated soft tissue reconstruction was not the correct operation. So when and which bony procedure should we perform?

Failure to consider trochlea dysplasia and TT-TG is the common reasons for poor outcomes in soft tissue stabilisation procedures [62]. Dejour taught that it is vital to consider the major risk factors in patella instability carefully to plan the correct procedures [5].

The key bony procedures for patella instability are:

- Trochleoplasty
- Tibial tuberosity osteotomy
- Femoral osteotomy (derotation or angular)

Selecting the correct procedure or combination of procedures is the key to successfully treating these interesting and challenging patients [63].

Trochleoplasty surgery, either with a thick or thin flap technique, is a very powerful procedure to help treat patella instability in patients with significant trochlea dysplasia. Typically, this procedure is indicated in patients who have Dejour type B, C or D dysplasia. Usually, these patients present in their teens with atraumatic recurrent instability, significant apprehension on examination, easily dislocatable patella and a strongly positive J sign. They usually have mild patella alta. In Dejour type D, patients can be chronically dislocated or have significant patella tilt should also be considered for trochleoplasty surgery [64, 65]. The published outcomes of trochleoplasty in this patient group are promising. Good results have been reported with both the thick flap and thin flap techniques [66–68]. Dejour has reported good results with trochleoplasty in the revision setting [69].

Tibial tuberosity osteotomy (TTO) has developed a bad reputation over the years [70]. This is at least in part due to the indications it has been used for. The outcome of TTO that is performed for treating PFJ pain has been disappointing. However, the results of correcting instability are good, but often this is associated with increased pain and early onset osteoarthritis.

The TTO is usually reserved for patients with significant patella alta (Fig. 12.6) (Caton-Decamps index >1.3). It has been observed that is it rare to find a significantly increased TT-TG in the absence of trochlea dysplasia. Therefore, typically many clinicians use this procedure to distalise the patella only. The excellent work of Fulkerson and others have shown that the antero-medialisation osteotomy can yield good

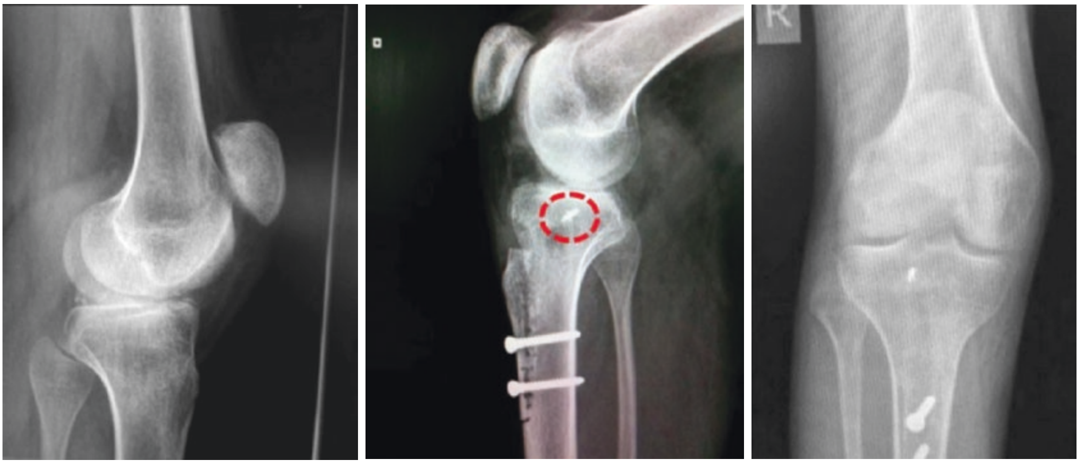


Fig. 12.6 Tibial tubercle distalisation for patella alta

results in the cases of increased TT-TG and trochlea dysplasia [71, 72]. This is an alternate option to trochleoplasty in mild/moderate trochlea dysplasia. Medialisation should be performed with care to avoid over medialisation as this can create a number of chronic problems.

Occasionally, a femoral osteotomy is required to address the patella instability. A derotational osteotomy is indicated in rare cases with significantly increased femur anteversion. It is important to assess the rotational profile of patients to ensure that this is not overlooked. An angular distal femoral osteotomy is occasionally indicated in patients who develop patella instability as a result of excessive valgus alignment.

12.2.4 Failed MPFL? What to Do Next?

There is no doubt that MPFL reconstruction for the treatment of objective patellofemoral dislocation has gained popularity in the last two decades. This rise in surgical intervention has brought about various complications. Recently, several surgical techniques have been described with various methods of fixation, knee flexion angle at the time of fixation, the choice of the graft and the tension which should be applied [73–80].

In the current literature, several studies have shown how the MPFL reconstruction provides significant improvement in patient-reported outcome measures and a high percentage of return to previous activity level [81–84]. However, despite its popularity, MPFL reconstruction is not free of complications. Indeed, in a systematic review of the literature, Shah et al. [85] found an overall complication rate of 26.1%, with almost one third (32%) of patients reporting recurrent instability. Meanwhile, the results published by Parikh et al. [86] reported complications in 16.2% of patients, with approximately half (47%) of them due to technical errors.

Almost all the complications could be categorised into two groups: complications that are due to an incorrect indication by failing to recognise the other risk factors that could have contributed to the dislocation and complications due to technical errors.

The first prerequisite to avoid complications and failure of an MPFL reconstruction is to properly select the patient. When evaluating a patient with patellofemoral complaints, it is mandatory to recognise that patellofemoral instability can present in a spectrum of manifestations. Therefore, it is important to differentiate between patients who have a documented true dislocation associated with haemarthrosis and those who report instability and "giving way" during low-energy activities which could be due to quadriceps inhibition following prolonged knee pain.

Anterior knee pain and excessive lateral patella tilt or lateral patella subluxation on imaging without a history and a physical examination for objective patella instability should never be treated with MPFL reconstruction.

On the other hand, the failure to take into account the major risk factors for patella instability [5] represents a common cause of failure of a MPFL reconstruction. Along with the clinical assessment, a complete imaging study is essential. One of the most relevant and major factors to consider is high-grade trochlear dysplasia, type C and D according to Dejour's classification [87], which can be responsible for an excessive laterally directed force on the patella [88] and overloading of the MPFL graft and fatigue rupture [64, 89, 90]. Therefore, high-grade trochlea dysplasia should be treated by trochleoplasty in order to avoid residual patellofemoral instability after isolated MPFL reconstruction [91, 92].

The patella height determines at which point the patella engages in the trochlea [93]. In patella alta, the engagement between patella and trochlea occurs at a higher degree of flexion and consequently with a lower contact area. For example, the contact area at 40° of knee flexion in patients with patella alta is comparable with the magnitude of contact area at 20° of knee flexion in patients with normal patella height. Hence, patients with patella alta have a mean of 19% less contact area than the control subjects over the range of 0–60° of flexion. Moreover, in patients with patella alta, the lateral patella tilt showed values of 39% higher than patients with normal patella height, and a 20% more lateral patella displacement has been reported [94].

Plain lateral radiographs are essential for the measurement of patella height by using different methods [33, 95, 96]. Recently, a new MRI index was introduced to assess the functional engagement between patella and trochlea in the sagittal plane [61, 97]. The sagittal patellofemoral engagement index, measured as the ratio between the articular cartilage of the patella and the trochlear cartilage length taken on two different MRI slices, may help to identify the cases where inadequate engagement is recorded despite the absence of patella alta, so that the need for tibial tuberosity osteotomy may be reassessed [97]. Therefore, the presence of patella alta with an insufficient functional sagittal patellofemoral engagement represents an indication to a distalisation of the tibial tuberosity in order to obtain a normal index.

The excessive TT-TG distance represents the third factor of patella instability and is a direct measure of the valgus alignment of the extensor mechanism and consequent valgus-displacing vector acting on the patella [98]. In particular, an excessive lateralized position of the tibial tuberosity reduces patella stability and increases patellofemoral joint contact pressure and lateral patella tracking. As a consequence of this, in the knees with overly lateralized position of the tibial tuberosity, the clinical maltracking may stretch the MPFL and allows lateral patella motion when the quadriceps are contracted, leading to failure of the graft and recurrent instability [99]. Different studies reported worse clinical and functional outcomes of isolated MPFL associated with high values of TT-TG distance [64, 92]. From a biomechanical point of view, a tibial tuberosity medialisation significantly reduces the lateral patella translation and the lateral patellofemoral joint contact pressure without increasing medial joint pressure. Therefore, when the TT-TG distance is increased over 20 mm, a tibial tuberosity medialisation osteotomy is performed in order to obtain a postoperative value between 10 and 15 mm [100]. Careful preoperative planning and an intraoperative confirmation of patella tracking are crucial to avoid complications resulting from overmedialisation.

It is crucial to keep in mind that trochleoplasty could reduce the TT-TG distance, acting as a proximal realignment [68] and that with a 10 mm distalisation 4 mm of medialisation is automatically achieved [87, 101].

The presence of an isolated patella tilt is not an indication for surgical treatment. However, the presence of a lateral patella tilt of more than 20° in patients with an objective patella instability associated with a negative medial patella tilt test could represent an indication to perform a lateral release.

Among the technical mistakes in MPFL reconstruction, the most recurrent and critical error is an incorrect femoral fixation point (Fig. 12.7), which is of crucial importance as it is

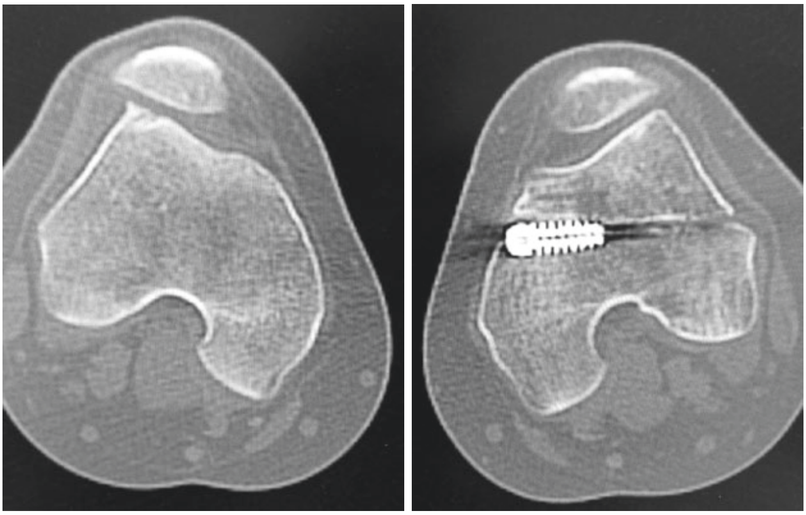


Fig. 12.7 Axial CT scan showing malpositioned femoral tunnel after MPFL reconstruction in the left knee

responsible for length change and graft tension during knee motion [18, 102]. In order to identify the anatomical femoral insertion of the MPFL, it is important to use intraoperative fluoroscopy. In this regard, it is imperative to obtain a true lateral radiographic image to identify the confluence point of the posterior cortex extension and the Blumensaat line [15, 103].

An excessively proximal or anterior femoral insertion is responsible for increased flexion tightness [104] which could lead to medial patellofemoral articular overload with pain and loss of flexion. This could even cause iatrogenic medial subluxation and subsequent recurrent lateral patella dislocation due to stretching of the graft [105]. Conversely, with an overly distal posterior tunnel placement, the graft may be loose in flexion and too tight in extension, causing pain with leg straightening, an extensor lag or stretching of the graft, with recurrent patella instability [104, 106].

Moreover, knee flexion angle during graft fixation is also crucial. If the graft is fixed in a misplaced tunnel whilst the knee is in extension, this leads to tightness and pain in flexion. If misplaced in flexion, the graft is loose and recurrent patella instability occurs in extension [91, 105].

Overtensioning the graft during fixation is another common technical error, which could lead to failure of MPFL reconstruction. Particularly, if the graft is too tight in flexion, it may increase medial patella facet pressure [107], causing pain, crepitus and loss of flexion, whilst the most frequent clinical presentation in cases of excessive tightness in extension is an extensor lag, with pain to fully straighten the leg [108]. Fithian and Gupta [109] described a medial gutter debridement in the case of medial knee pain after MPFL reconstruction. Meanwhile, Thaunat and Erasmus [108] described a gradual step-by-step percutaneous release of the graft. Both reported good resolution of pain and recovery of range of motion without instability.

Multiple studies have shown that the optimum tension for the MPFL graft is 2 newtons (204 g) at 30–60° knee flexion angle [18, 105, 108, 110]. In order to avoid excessive tension on the graft, intraoperatively the reconstructed MPFL should translate two to three patella quadrants and have a hard stop, without excessive constraint on the patella [85]. A comparison with the contralateral side can be helpful in determining appropriate graft tension [111].

Another major complication that leads to MPFL failure, generally due to technical error, is patella fracture. In literature, patella fractures after MPFL reconstruction have been categorised into three groups [112]:

- Type I fractures are transverse fractures generally associated with the patella tunnel or drill hole. These tunnels can act as stress risers and reduce the strength of the bone. Particularly, violation of the anterior patella cortex during tunnel creation represents the main cause of this complication, and, therefore, preservation of the anterior cortex of the patella is mandatory to avoid this complication. This kind of fracture is generally treated surgically using a tension-band wiring technique.
- Type II fractures are sleeve avulsion fractures or superior pole fractures, [110] generally encountered when proximal realignment, lateral release or excessive dissection at the superior aspect of the patella are performed. One of the suggested causes of this kind of fracture is vascular damage of the proximal part of the patella. Therefore, it is critical to perform an accurate dissection to preserve one or more of the genicular arteries during combined procedures on the medial and lateral sides of the patella. The treatment is similar to that of quadriceps tendon tears, consisting of suturing the quadriceps tendon to the superior pole of the patella through longitudinal drill holes.
- Type III fractures are medial rim avulsion fractures through the osseous bridge between the tunnels in the patella and are generally associated with recurrent lateral patella dislocation after patella stabilisation procedures [109, 113]. These fractures are generally treated with open reduction and internal fixation of the fragments with the use of screws or anchors.

Generally, in order to reduce the risk of a fracture after an MPFL reconstruction, it is better to avoid transverse patella tunnels, reduce tunnel diameter, maintain an adequate bone bridge, avoid devascularisation of the superior pole of the patella and perform an anatomic tunnel placement in the femur and the patella.

12.2.5 Patella Instability: Management Summary

12.2.5.1 Nonoperative Management

Functional rehabilitation is the mainstay of nonoperative management with particular focus on gait, core stability and quadriceps strengthening [55]. A small number of older randomised trials comparing operative and nonoperative treatment of initial patella dislocation found no benefit from immediate medial retinacular repair [51, 114].

Currently, nonoperative treatment is indicated in acute first-time dislocators without associated osteochondral fracture or loose bodies. Despite the high rate of re-dislocation, the benefit of acute soft tissue repair or reconstruction is yet to be established. Recent level one evidence studies, including six randomised controlled trials, showed that the rate of re-dislocation following surgical stabilisation was significantly lower than nonoperative treatment [46, 51–53, 115, 116]. However, it can be concluded from other level one evidence studies that the outcome of non-surgical treatment is less satisfactory, as 49% of the patients re-dislocated, nearly two thirds continued to have instability symptoms and anterior knee pain, with low patient satisfaction of 40%, and only 42% returned to pre-injury level [1–3, 47].

12.2.5.2 Operative Management

The principles of surgical management in patients with recurrent instability are to address the primary abnormal anatomical factor that contributes most to re-dislocation without creating a secondary pathoanatomy to compensate for it, as summarised in Table 12.1. Unfortunately, it is never as straightforward as the summary suggests. Often there are multiple abnormal anatomical factors that are interacting in the background. An event that leads to first-time dislocation disrupts knee homeostasis and causes it to decompensate. Homeostasis can be restored by simpler procedures such as MPFL reconstruction in more than 80% of the cases or tibial tuberosity distalisation in severe patella alta. However, in certain patients the patella is permanently dislocated or tracking in the lateral gutter, only relocating in full knee extension. This group of patients would require more than one procedure to achieve patella stability.

Table 12.1 The principles of “a la carte” surgical intervention based on the most contributing factor in PFJI

Pathoanatomy	Surgical options
Instability with malalignment	Tibial tuberosity medialisation
Instability without malalignment	MPFL reconstruction
Instability with patella alta	Tibial tuberosity distalisation
Trochlea dysplasia	Trochleoplasty
Rotational problems	Derotation osteotomy
Combined pathology	Multiple simultaneous surgical interventions

A variety of surgical techniques have been described to reconstruct the MPFL. Considering that the native MPFL resistance is around 208 N, the graft choice should reflect the required ultimate load to failure. It appears that the gracilis tendon has stiffness closer to that of the native MPFL compared to the semitendinosus tendon. One of the preferred ways is to fix the gracilis tendon autograft with a screw in the femur and either two suture anchors medially in a small patella, usually female patients, or a bony tunnel in the anterior patella in larger patients, normally male. There is still a paucity of studies presenting long-term data. In a recent meta-analysis, a total of 1065 MPFL reconstructions were identified in 31 studies, and it was found that autograft reconstructions were associated with greater postoperative improvements in Kujala scores when compared to allograft and that double-limbed reconstructions were associated with both improved postoperative Kujala scores and lower failure rate [117]. Overall, in the absence of significant malalignment, MPFL reconstructions appear to provide long-term functional improvement with improved Kujala scores, low rate of re-dislocation and decreases in apprehension and patellofemoral pain [76, 84, 118]. However, the current literature on MPFL outcomes has substantial methodological limitations with small sample sizes and limited follow-ups [119]. Standardising the surgical technique on an adequate sample size with long-term follow-up will be necessary for future outcomes studies.

The presence of trochlear dysplasia can be addressed with a trochlear groove deepening trochleoplasty procedure, as described by Dejour (Lyon's procedure) [120], or its variants which led to good clinical outcomes in the literature [34, 121–125]. Long-term studies on the effectiveness of trochleoplasty are scarce. In their series, Utting et al. [126] reported on 54 consecutive patients (59 knees) with PFJI secondary to trochlear dysplasia, who were treated by a trochleoplasty by a single surgeon. Overall, 92.6% of their patients were satisfied with the outcome of their procedure. Rouanet et al. [125] reported on their series of 34 patients, with an average of 15 years of follow-up who underwent deepening trochleoplasties using multiple outcome scores. They reported the restoration of patellofemoral stability, even in patients with severe dysplasia. However, it did not prevent patellofemoral osteoarthritis.

Distal realignment procedures include tibial tuberosity transfer, typically with distalisation and/or medialisation, to address patella alta and malalignment [84, 127] (Fig. 12.6). In a cadaveric study, it was found that in the knees with preoperative TT-TG distances of up to 15 mm, patellofemoral kinematics and contact mechanics can be restored with MPFL reconstruction [99]. However, for the knees with preoperative TT-TG distances greater than 15 mm, more aggressive surgery such as tibial tuberosity transfer may be indicated [99]. This, however, is difficult to translate to patients with PFJI as they normally have more than one anatomic abnormality unlike the cadavers studied, and their knees are subjected to various dynamic weight-bearing forces that are difficult to reproduce in laboratory investigations.

Contraindications of tibial tuberosity transfer include medial and/or proximal patellofemoral chondrosis that would be subjected to increased loading with a transfer of the tuberosity [128]. In a recent systematic review looking at MPFL reconstruction with concomitant tibial tuberosity transfer in five studies with 92 knees and a mean follow-up of 38 months (range 23–53), showed that the combined procedures are effective in the setting of malalignment [128].

12.3 Future Treatment Options

In the future, the graft choice may move towards synthetic or biologically engineered grafts to reduce the donor site morbidity and reduce operating time. In addition, in vivo intra-articular contact pressure and patella tracking measurement during bony or soft tissue realignment may be one of the ways to avoid the current problems with alignment accuracy and tunnel misplacement. Using an intraoperative graft tensioner, instead of eyeballing and manual dexterity, may overcome the problems with misjudging the graft tension.

12.4 Take-Home Message

Patellofemoral joint instability is relatively common. It can be caused by a range of factors including generalised hypermobility, patella hypermobility, increased femoral anteversion, core and hip abductor weakness, abnormal knee rotation, trochlea dysplasia, abnormal Q angle, patella alta, muscle and soft tissue imbalance, external tibial torsion and foot hyperpronation. Due to the multifactorial nature of PFJI, common clinical and radiological outcomes, such as the Q angle and TT-TG distance, cannot be relied upon in isolation. It is, therefore, vital to conduct a thorough clinical and radiological investigation to determine the main cause of instability, prior to treatment. Relatively simple surgical procedures, such as medial patellofemoral ligament reconstruction, can restore PFJ stability in a high proportion of unstable knees, especially in those with lower TT-TG distances. A deepening trochleoplasty is rarely indicated in isolation. Tibial tuberosity transfer can be used to address more significant instability, often in combination with MPFL reconstruction. A greater number of long-term investigations are needed to achieve a better understanding of patient outcomes following these procedures [129].

References

1. Casteleyn PP, Handelberg F. Arthroscopy in the diagnosis of occult dislocation of the patella. Acta Orthop Belg. 1989;55:381–3.
2. Hawkins RJ, Bell RH, Anisette G. Acute patellar dislocations: the natural history. Am J Sports Med. 1986;14:117–20.
3. Mehta VM, Inoue M, Nomura E, Fithian DC. An algorithm guiding the evaluation and treatment of acute primary patellar dislocations. Sports Med Arthrosc Rev. 2007;15:78–821.
4. Conlan T, Garth WP, Lemons JE. Evaluation of the medial soft tissue restraints of the extensor mechanisms of the knee. J Bone Joint Surg Am. 1993;75:682–93.
5. Dejour H, Walch G, Nove-Josserand L, Guier C. Factors of patellar instability: an anatomic radiographic study. Knee Surg Sports Traumatol Arthrosc. 1994;2:19–26.
6. Dierks TA, Manal KT, Hamill J, Davis IS. Proximal and distal influences on hip and knee kinematics in runners with patellofemoral pain during a prolonged run. J Orthop Sports Phys Ther. 2008;38:448–56.
7. Parikh S, Noyes FR. Patellofemoral disorders: role of computed tomography and magnetic resonance imaging in defining abnormal rotational lower limb alignment. Sports Health. 2011;3:158–69.
8. Redziniak DE, Diduch DR, Mihalko WM, Fulkerson JP, Novicoff WM, Sheibhani-Rad S, Saleh KJ. Patellar instability. Instr Course Lect. 2010;59:195–206.
9. Powers CM. The influence of altered lower extremity kinematics on patellofemoral joint dysfunction: a theoretical perspective. J Orthop Sports Phys Ther. 2003;33:639–46.
10. Desio SM, Burks RT, Bachus KN. Soft tissue restraints to lateral patellar translation in the human knee. Am J Sports Med. 1998;26:59–65.
11. Hautamaa PV, Fithian DC, Kaufmann KR, Daniel DM, Pohlmeyer AM. Medial soft tissue restraints in lateral patellar instability and repair. Clin Orthop. 1998;349:174–82.
12. Reider B, Marshall JL. The anterior aspect of the knee joint. J Bone Joint Surg Am. 1981;63:351–6.
13. Stephen JM, Lumpaopong P, Deehan DJ, Kader D, Amis AA. The medial patellofemoral ligament: location of the femoral attachment and length change patterns resulting from anatomic and non-anatomic attachments. Am J Sports Med. 2012;40:1871–9.
14. Amis AA, Firer P, Mountney J, Senavongse W, Thomas NP. Anatomy and biomechanics of the medial patellofemoral ligament. Knee. 2003;10:215–20.
15. Schöttle PB, Schmeling A, Rosenstiel N, Weiler A. Radiographic landmarks for femoral tunnel placement in medial patellofemoral ligament reconstruction. Am J Sports Med. 2007;35:801–4.
16. McCarthy M, Ridley TJ, Bollier M, Wolf B, Albright J, Amendola A. Femoral tunnel placement in medial patellofemoral ligament reconstruction. Iowa Orthop J. 2013;33:58–63.
17. Stephen JM, Kader D, Lumpaopong P, Deehan DJ, Anis AA. Sectioning the medial patellofemoral ligament alters patellofemoral joint kinematics and contact mechanics. J Orthop Res. 2013;31:1423–9.
18. Stephen JM, Kader D, Lumpaopong P, Deehan DJ, Amis AA. The effect of femoral tunnel position and graft tension on patellar contact mechanics after medial patellofemoral ligament reconstruction. Am J Sports Med. 2014;42:364–72.
19. Fujino K, Tajima G, Yan J, Kamei Y, Maryuma M, Takeda S, Kikuchi S, Shimamura T. Morphology of the femoral insertion site of the medial patellofemoral ligament. Knee Surg Sports Traumatol Arthrosc. 2015;23:998–1003.
20. Placella G, Tei MM, Sebastiani E, Crescenti G, Speziali A, Mazzola C, Georgoulis A, Cerulli G. Shape and size of the medial patellofemoral ligamentt for the best surgical reconstruction: a human cadaveric study. Knee Surg Sports Traumatol Arthrosc. 2014;22:2327.
21. Redfern J, Kamath G, Burks R. Anatomical confirmation of the use of radiographic landmarks in medial patellofemoral ligament reconstruction. Am J Sports Med. 2010;38:293–7.
22. Nomura E, Inoue M, Kobayashi S. Generalised joint laxity and contralateral patellar hyper mobility in unilateral recurrent patellar dislocators. Arthroscopy. 2006;22:861–5.
23. Steensen RN, Bentley JC, Trinh TQ, Backes JR, Wiltfong RE. The prevalence and combined prevalences of anatomic factors associated with recurrent patellar dislocation: a magnetic resonance imaging study. Am J Sports Med. 2015;43:921–7.
24. Brattström H. Patella alta in non-dislocating knee joints. Acta Orthop Scand. 1970;41:921–7.
25. Aglietti P, Insall JN, Ceruli G. Patellar pain and incongruence, 1: measures of incongruence. Clin Orthop Relat Res. 1983;176:217–24.
26. Wilson T, Kitsell F. Is the Q angle absolute or a variable measure? Measurement of the Q angle over one minute in healthy subjects. Physiotherapy. 2002;88:296–302.

27. Smith TO, Hunt NJ, Donell ST. The reliability and validity of the Q angle: a systematic review. Knee Surg Sports Traumatol Arthrosc. 2008;16:1068–79.
28. France L, Nester C. Effect of error in the identification of anatomical landmarks on the accuracy of Q angle values. Clin Biomech. 2001;16:710–3.
29. Biedert RM, Gruß C. Axial computed tomography of the patellofemoral joint with and without quadriceps contraction. Arch Orthop Trauma Surg. 1997;116:77–82.
30. Guerra JP, Arnold MJ, Gajdosik RL. Q angle: effects of isometric quadriceps contraction and body position. J Orthop Sports Phys Ther. 1994;19:200–4.
31. Cooney AD, Kazi Z, Caplan N, Newby M, St Clair Gibson A, Kader DF. The relationship between quadriceps angle and tibial tuberosity trochlear groove distance in patients with patellar instability. Knee Surg Sports Traumatol Arthrosc. 2012;20:2399–404.
32. Phillips CL, Silver DA, Schranz PJ, Mandalia V. The measurement of patellar height: a review of the methods of imaging. J Bone Joint Surg Br. 2010;92:1045–53.
33. Caton J, Deschamps G, Chambat P, Lerat JL, Dejour H. Patella Infera: apropos of 128 cases. Rev Chir Orthop Reparatrice Appar Mot. 1982;68:317–25.
34. Bollier M, Fulkerson JP. The role of trochlear dysplasia in patellofemoral instability. J Am Acad Orthop Surg. 2011;19:8–16.
35. Camp CL, Stuart MJ, Krych AJ, Levy BA, Bond JR, Collins MS, Dahm DL. CT and MRI measurements of tibial tubercle-trochlear groove distances are not equivalent in patients with patellar instability. Am J Sports Med. 2013;41:1835–40.
36. Pennock AT, Alam A, Bastrom T. Variation in tibial-trochlear groove measurement as a function of age, sex, size and patellar instability. Am J Sports Med. 2014;42:389–93.
37. Smith TO, Davies L, Toms AP, Hing CB, Donell ST. The reliability and validity of radiological assessment for patellar instability. A systematic review and meta-analysis. Skelet Radiol. 2011;40:399–414.
38. Goutallier D, Bernageau J, Lecudonnec B. The measurement of the tibial tuberosity. Patella groove distanced technique and results. Rev Chir Orthop Reparatrice Appar Mot. 1978;64:423–8.
39. Staubli HU, Durrenmatt U, Porcellini B, Rauschning W. Anatomy and surface geometry of the patellofemoral joint in the axial plane. J Bone Joint Surg Br. 1999;81:452–8.
40. Caplan N, Lees D, Newby M, Ewen A, Jackson R, St Clair Gibson A, Kader D. Is tibial tuberosity-trochlear groove distance an appropriate measure for the identification of knees with patellar instability? Knee Surg Sports Traumatol Arthrosc. 2014;22:2377–81.
41. Dejour H, Walch G, Neyret P, Adeleine P. Dysplasia of the femoral trochlea. Rev Chir Orthop Reparatrice Appar Mot. 1990;76:45–54.
42. Remy F, Chantelot C, Fontaine C, Demondion X, Miguad H, Gougeon F. Inter- and intra-observer reproducibility in radiographic diagnosis and classification of femoral trochlear dysplasia. Surg Radiol Anat. 1998;20:285–9.
43. Arendt EA, Dahm DL, Dejour D, Fithian DC. Patellofemoral jont: from instability to arthritis. Instr Course Lect. 2014;63:355–68.
44. Sallay PI, Poggi J, Speer KP, Garrett WE. Acute dislocation of the patella: a correlative pathoanatomic study. Am J Sports Med. 1996;24:52–60.
45. Buckens CF, Saris DB. Reconstruction of the medial patellofemoral ligament for treatment of patellofemoral instability: a systematic review. Am J Sports Med. 2010;38:181–8.
46. Sillanpaa PJ, Mattila VM, Maenpaa H, Kiuru M, Visuri T, Pihlajamaki H. Treatment with and without initial stabilising surgery for primary traumatic patella dislocation: a prospective randomised study. J Bone Joint Surg Am. 2009;91:263–73.
47. Atkin DM, Within CD, Marangi KS, Stone ML, Dobson BE, Mendelsohn C. Characteristics of patients with primary acute lateral patellar dislocation and their recovery within the first six months of injury. Am J Sports Med. 2000;28:472–9.
48. Maenpaa H, Lehto MU. Patellafemoral osteoarthritis after patellar dislocation. Clin Orthop Relat Res. 1997;339:156–62.
49. Longo UG, Ciuffreda M, Locher J, Berton A, Salvatore G, Denaro V. Treatment of primary acute patellar dislocation: systematic review and quantitative synthesis of the literature. Clin J Sport Med. 2017;27(6):511–23.
50. Nikku R, et al. Operative treatment of primary patellar dislocation does not improve medium-term outcome. Acta Orthop. 2009;76:699–704.
51. Nikku R, et al. Operative versus closed treatment of primary dislocation of the patella. Similar 2-year results in 125 randomized patients. Acta Orthop Scand. 1997;68:419–23.
52. Camanho GL, et al. Conservative versus surgical treatment for repair of the medial patellofemoral ligament in acute dislocations of the patella. Arthroscopy. 2009;25:620–5.
53. Petri M, et al. Operative vs conservative treatment of traumatic patellar dislocation: results of a prospective randomized controlled clinical trial. Arch Orthop Trauma Surg. 2012;133:209–13.
54. Bitar AC, et al. Traumatic patellar dislocation: nonoperative treatment compared with MPFL reconstruction using patellar tendon. Am J Sports Med. 2012;40:114–22.
55. Smith TO, Donell S, Song F, Hing CB. Surgical versus non-surgical interventions for treating patellar dislocation. Cochrane Database Syst Rev. 2015;(2).
56. Regalado G, et al. Six-year outcome after non-surgical versus surgical treatment of acute primary patellar dislocation in adolescents: a prospective randomized trial. Knee Surg Sports Traumatol Arthrosc. 2014;24:6–11.

57. Balcarek P, et al. Which patellae are likely to redislocate? Knee Surg Sports Traumatol Arthrosc. 2014;22:2308–14.
58. Burrus MT, Tompins M, Hinckel B, Diduch D, Arendt E. Chap. 68 Repair and reconstruction of the medial patellofemoral ligament for treatment of lateral patellar dislocations: surgical techniques and clinical results. In: Scott WN, editor. Insall & scott - surgery of the knee. 6th ed. Philadelphia: Churchill Livingstone; 2016.
59. Tompkins MA, Arendt EA. Patellar instability factors in isolated medial patellofemoral ligament reconstructions – what does the literature tell us? A systematic review. Am J Sports Med. 2015;43:2318–27.
60. Ridley TJ, Bremer Hinckel B, Kruckeberg BM, Agel J, Arendt EA. Anatomical patella instability risk factors on MRI show sensitivity without specificity in patients with patellofemoral instability: a systematic review. J ISAKOS: Jt Disord Orthop Sports Med. 2013;1:141–52.
61. Biedert RM, Albrecht S. The patellotrochlear index: a new index for assessing patellar height. Knee Surg Sports Traumatol Arthrosc. 2006;14:707–12.
62. Nelitz M, Theile M, Dornacher D, Wölfle J, Reichel H, Lippacher S. Analysis of failed surgery for patellar instability in children with open growth plates. Knee Surg Sports Traumatol Arthrosc. 2012;20:822–8.
63. Caplan N, Nassar I, Anand B, Kader DF. Why do patellofemoral stabilization procedures fail? Keys to success. Sports Med Arthrosc Rev. 2017;25:e1.
64. Wagner D, Pfalzer F, Hingelbaum D, Huth J, Mauch F, Bauer G. The influence of risk factors on clinical outcomes following anatomical medial patellofemoral ligament (MPFL) reconstruction using the gracilis tendon. Knee Surg Sports Traumatol Arthrosc. 2013;21:318–24.
65. Kita K, Tanaka Y, Toritsuka Y, Amano H, Uchida R, Takao R, Horibe S. Factors affecting the outcomes of double-bundle medial patellofemoral ligament reconstruction for recurrent patellar dislocations evaluated by multivariate analysis. Am J Sports Med. 2015;43:298–2996.
66. Fucentese SF, Zingg PO, Schmitt J, Pfirrmann CW, Meyer DC, Koch PP. Classification of trochlear dysplasia as predictor of clinical outcome after trochleoplasty. Knee Surg Sports Traumatol Arthrosc. 2011;19:1655–61.
67. Metcalfe AJ, Clark DA, Kemp MA, Eldridge JD. Trochleoplasty with a flexible osteochondral flap: results from an 11-year series of 214 cases. J Bone Joint Surg. 2017;99:344–50.
68. Ntagiopoulos PG, Byn P, Dejour D. Midterm results of comprehensive surgical reconstruction including sulcus-deepening trochleoplasty in recurrent patellar dislocations with high-grade trochlear dysplasia. Am J Sports Med. 2013;41:998–1004.
69. Dejour D, Byn P, Ntagiopoulos PG. The Lyon's sulcus-deepening trochleoplasty in previous unsuccessful patellofemoral surgery. Int Orthop. 2013;37:433–9.
70. Sherman SL, Erickson BJ, Cvetanovich GL, Chalmers PN, Farr JN, Bach BRJ, Cole BJ. Tibial tuberosity osteotomy: indications, techniques, and outcomes. Am J Sports Med. 2014;42:2006–17.
71. Tjoumakaris FP, Forsythe B, Bradley JP. Patellofemoral instability in athletes: treatment via modified Fulkerson osteotomy and lateral release. Am J Sports Med. 2010;38:992–9.
72. Chen H, Zhao D, Xie J, Duan Q, Zhang J, Wu Z, Jiang J. The outcomes of the modified Fulkerson osteotomy procedure to treat habitual patellar dislocation associated with high-grade trochlear dysplasia. BMC Musculoskelet Disord. 2017;18:73.
73. Nomura E, Inoue M. Surgical technique and rationale for medial patellofemoral ligament reconstruction for recurrent patellar dislocation. Arthroscopy. 2003;19:E47.
74. Steensen RN, Dopirak RM, Maurus PB. A simple technique for reconstruction of the medial patellofemoral ligament using a quadriceps tendon graft. Arthroscopy. 2005;21:365–70.
75. Schottle PB, Romero J, Schmeling A, Weiler A. Technical note: anatomical reconstruction of the medial patellofemoral ligament using a free gracilis autograft. Arch Orthop Trauma Surg. 2008;128:479–84.
76. Christiansen SE, Jacobsen BW, Lund B, Lind M. Reconstruction of the medial patellofemoral ligament with gracilis tendon autograft in transverse patellar drill holes. Arthroscopy. 2008;24:82–7.
77. Sillanpää PJ, Mäenpää HM, Mattila VM, Visuri T, Pihlajamäki H. A mini-invasive adductor magnus tendon transfer technique for medial patellofemoral ligament reconstruction: a technical note. Knee Surg Sports Traumatol Arthrosc. 2009;17:508–12.
78. Ahmad CS, Brown GD, Stein BS. The docking technique for medial patellofemoral ligament reconstruction: surgical technique and clinical outcome. Am J Sports Med. 2009;37:2021–7.
79. Siebold R, Chikale S, Sartory N, Hariri N, Feil S, Pässler HH. Hamstring graft fixation in MPFL reconstruction at the patella using a transosseous suture technique. Knee Surg Sports Traumatol Arthrosc. 2010;18:1542–4.
80. Deie M, Ochi M, Adachi N, Shibuya H, Nakamae A. Medial patellofemoral ligament reconstruction fixed with a cylindrical bone plug and a grafted semitendinosus tendon at the original femoral site for recurrent patellar dislocation. Am J Sports Med. 2011;39:140–5.
81. Schneider DK, Grawe B, Magnussen RA, Ceasar A, Parikh SN, Wall EJ. Outcomes after isolated medial patellofemoral ligament reconstruction for the treatment of recurrent lateral patellar dislocations: a systematic review and meta-analysis. Am J Sports Med. 2016;44:2993–3005.
82. Matic GT, Magnussen RA, Kolovich GP, Flanigan DC. Return to activity after medial patellofemoral ligament repair or reconstruction. Arthroscopy. 2014;30:1018–25.

83. Stupay KL, Swart E, Shubin Stein BE. Widespread implementation of medial patellofemoral ligament reconstruction for recurrent patellar instability maintains functional outcomes at midterm to long-term follow-up while decreasing complication rates: a systematic review. Arthroscopy. 2015;31:1372–80.
84. Valkering KP, Rajeev A, Caplan N, Tuinebreijer WE, Kader DF. An evaluation of the effectiveness of medial patellofemoral ligament reconstruction using an anatomical tunnel site. Knee Surgery, Sports Traumatology, Arthroscopy. 2017;25(10):3206–12.
85. Shah JN, Howard JS, Flanigan DC, Brophy RH, Carey JL, Lattermann C. A systematic review of complications and failures associated with medial patellofemoral ligament reconstruction for recurrent patellar dislocation. Am J Sports Med. 2012;40:1916–23.
86. Parikh SN, Nathan ST, Wall EJ, Eismann EA. Complications of medial patellofemoral ligament reconstruction in young patients. Am J Sports Med. 2013;41:1030–8.
87. Dejour D, Le Coultre B. Osteotomies in patellofemoral instabilities. Sports Med Arthrosc Rev. 2007;15:39–46.
88. Amis AA, Oguz C, Bull AM, Senavongse W, Dejour D. The effect of trochleoplasty on patellar stability and kinematics: a biomechanical study in vitro. J Bone Joint Surg Br. 2008;90:864–9.
89. Tischer T, Geier A, Lenz R, Woernle C, Bader R. Impact of the patella height on the strain pattern of the medial patellofemoral ligament after reconstruction: a computer model-based study. Knee Surg Sports Traumatol Arthrosc. 2016;25:3123.
90. Berard JB, Magnussen RA, Bonjean G, Ozcan S, Lustig S, Neyret P, Servien E. Femoral tunnel enlargement after medial patellofemoral ligament reconstruction: prevalence, risk factors, and clinical effect. Am J Sports Med. 2014;42:297–301.
91. Nelitz M, Williams RS, Lippacher S, Reichel H, Dornacher D. Analysis of failure and clinical outcome after unsuccessful medial patellofemoral ligament reconstruction in young patients. Int Orthop. 2014;38:2265–72.
92. Kita K, Tanaka Y, Toritsuka Y, Amano H, Uchida R, Takao R, Horibe S. Factors affecting the outcomes of double-bundle medial patellofemoral ligament reconstruction for recurrent patellar dislocations evaluated by multivariate analysis. Am J Sports Med. 2015;43:2988–96.
93. Tecklenburg K, Dejour D, Hoser C, Fink C. Bony and cartilaginous anatomy of the patellofemoral joint. Knee Surg Sports Traumatol Arthrosc. 2006;14:235–40.
94. Ward SR, Terk MR, Powers CM. Patella alta: association with patellofemoral alignment and changes in contact area during weight-bearing. J Bone Joint Surg Am. 2007;89:1749–55.
95. Insall J, Salvati E. Patella position in the normal knee joint. Radiology. 1971;101:101–4.
96. Blackburne JS, Peel TE. A new method of measuring patellar height. J Bone Joint Surg Br. 1977;59:241–2.
97. Dejour D, Ferrua P, Ntagiopoulos PG, Radier C, Hulet C, Rémy F. The introduction of a new MRI index to evaluate sagittal patellofemoral engagement. Orthop Traumatol Surg Res. 2013;99:391–S398.
98. Dejour D, Saggin P. Disorders of the patellofemoral joint. In: Scott WN, editor. Insall & scott surgery of the knee, 5th ed. Philadelphia: Churchill Livingstone/Elsevier; 2012. pp. 592–623.
99. Stephen JM, Dodds AL, Lumpaopong P, Kader D, Williams A, Amis AA. The ability of medial patellofemoral ligament reconstruction to correct patellar kinematics and contact mechanics in the presence of a lateralized tibial tubercle. Am J Sports Med. 2015;43:2198–207.
100. Servien E, Aït Si Selmi T, Neyret P. Subjective evaluation of surgical treatment for patellar instability. Rev Chir Orthop Reparatrice Appar Mot. 2004;90:137–42.
101. Servien E, Verdonk PC, Neyret P. Tibial tuberosity transfer for episodic patellar dislocation. Sports Med Arthrosc Rev. 2007;15:61–7.
102. Sanchis-Alfonso V, Ramirez-Fuentes C, Montesinos-Berry E, Domenech J, Martí-Bonmatí L. Femoral insertion site of the graft used to replace the medial patellofemoral ligament influences the ligament dynamic changes during knee flexion and the clinical outcome. Knee Surg Sports Traumatol Arthrosc. 2017;25:2433.
103. Servien E, Fritsch B, Lustig S, Demey G, Debarge R, Lapra C, Neyret P. In vivo positioning analysis of medial patellofemoral ligament reconstruction. Am J Sports Med. 2011;39:134–9.
104. Elias JJ, Cosgarea AJ. Technical errors during medial patellofemoral ligament reconstruction could overload medial patellofemoral cartilage: a computational analysis. Am J Sports Med. 2006;34:1478–85.
105. Bollier M, Fulkerson J, Cosgarea A, Tanaka M. Technical failure of medial patellofemoral ligament reconstruction. Arthroscopy. 2011;27:1153–9.
106. Elias JJ, Kelly MJ, Smith KE, Gall KA, Farr J. Dynamic simulation of the effects of graft fixation errors during medial patellofemoral ligament reconstruction. Orthop J Sports Med. 2016;4:2325967116665080.
107. Beck P, Brown NA, Greis PE, Burks RT. Patellofemoral contact pressures and lateral patellar translation after medial patellofemoral ligament reconstruction. Am J Sports Med. 2007;35:1557–63.
108. Thaunat M, Erasmus PJ. Management of overtight medial patellofemoral ligament reconstruction. Knee Surg Sports Traumatol Arthrosc. 2009;17:480–3.
109. Fithian DC, Gupta N. Patellar instability: principles of soft tissue repair and reconstruction. Tech Knee Surg. 2006;5:19–26.
110. Brennan SA, Walls R, Jackson M, Moran R. Superior pole sleeve fracture following patellar stabilisation. Knee. 2009;16:235–7.
111. Sanchis-Alfonso V, Montesinos-Berry E, Ramirez-Fuentes C, Leal-Blanquet J, Gelber PE, Monllau JC.

Failed medial patellofemoral ligament reconstruction: causes and surgical strategies. World J Orthod. 2017;8:115–29.
112. Parikh SN, Wall EJ. Patellar fracture after medial patellofemoral ligament surgery: a report of five cases. J Bone Joint Surg Am. 2011;93:e97(1-8).
113. Thaunat M, Erasmus PJ. Recurrent patellar dislocation after medial patellofemoral ligament reconstruction. Knee Surg Sports Traumatol Arthrosc. 2007;16(28):40–3.
114. Nikku R, Nietosvaara Y, Aalto K, Kallio PE. Operative treatment of primary patellar dislocation does not improve medium term outcome: a 7 year follow up report and risk analysis of 127 randomised patients. Acta Orthop. 2005;76:419–23.
115. Christiansen SE, Jakobsen BW, Lund B, Lind M. Isolated repair of the medial patellofemoral ligament in primary dislocation of the patella: a prospective randomised study. Arthroscopy. 2008;24:881–7.
116. Palmu S, Kallio PE, Donell ST, Helenius I. Acute patellar dislocation in children and adolescents: a randomised controlled trial. J Bone Joint Surg Am. 2008;90:463–70.
117. Weinberger JM, Fabricant PD, Taylor SA, Mei YM, Jones KJ. Influence of graft source and configuration on revision rate and patient reported outcomes after MPFL reconstruction: a systematic review. Knee Surg Sports Traumatol Arthrosc. 2017;25:2511.
118. Deie M, Ochi M, Sumen Y, Adachi N, Kobayashi K, Yasumoto M. A long term follow up study after medial patellofemoral ligament reconstruction using at the transferred semintendinosus tendon for patella dislocation. Knee Surg Sports Traumatol Arthrosc. 2005;13:82–7.
119. Smith TO, Walker J, Russell N. Outcomes of medial patellofemoral ligament reconstruction for patella instability: a systematic review. Knee Surg Sports Traumatol Arthrosc. 2007;15:1301–14.
120. Dejour D, Saggin P. The sulcus-deepening trochleoplasty - the Lyon's procedure. Int Orthop. 2010;34:311–6.
121. Banke IJ, Kohn LM, Meidinger D, Otto A, Hensler D, Beitzel K, Bimhoff B, Schottle PB. Combined trochleoplasty and MPFL reconstruction for treatment of chronic patellofemoral instability: a prospective minimum 2 year follow up study. Knee Surg Sports Traumatol Arthrosc. 2014;22: 2591–8.
122. Duncan TS, Noellen BS, Lattermann C. The role of trochleoplasty in patellofemoral instability. Sports Med Arthrosc Rev. 2012;20:171–80.
123. Faruqui S, Bollier M, Wolf B, Amendola N. Outcomes after trochleoplasty. Iowa Orthop J. 2012;32:171–80.
124. LaPrade RF, Cram TR, James EW, Rasmussen MT. Trochlear dysplasia and the role of trochleoplasty. Clin Sports Med. 2014;33:531–45.
125. Rouanet T, Gougeon F, Fayard JM, Remy F, Miguad H, Pasquier G. Sulcus deepening trochleoplasty for patellofemoral instability: a series of 34 cases after 15 years postoperative follow up. Orthop Traumatol Surg Res. 2015;101:443–7.
126. Utting MR, Mulford JS, Eldridge JD. A prospective evaluation of trochleoplasty for the treatment of patellofemoral dislocation and instability. J Bone Joint Surg. 2008;90:180–5.
127. Caton JH, Dejour D. Tibial tubercle osteotomy in patello-femoral instability and in patellar height abnormality. Int Orthop. 2010;34:305–9.
128. Burnham JM, Howard JS, Hayes CB, Lattermann C. Medial patellofemoral ligament reconstruction with concomitant tibial tubercle transfer: a systematic review. Arthroscopy. 2016;32:1185.
129. Kader DF, Matar HE, Caplan C. Patellofemoral joint instability: a review of current concepts. J Orthop Trauma. 2016;6:1.

Extra-articular Shoulder Endoscopy: A Review of Techniques and Indications

13

Roman Brzóska, Angel Calvo, Pablo Carnero,
Paweł Janusz, Viktoras Jermolajevas,
Laurent Lafosse, Thibault Lafosse, Hubert Laprus,
Olaf Lorbach, Paweł Ranosz, Alfredo Rodríguez,
and Nestor Zurita

13.1 Axillary Nerve and Musculocutaneous Nerve Anatomy After Latarjet Procedure

Roman Brzóska, Hubert Laprus, Paweł Ranosz, and Paweł Janusz

13.1.1 Introduction

Latarjet procedure is related with the highest rate of neurological complications from all of the procedures for anterior instability repair. Prevalence of these injuries is estimated at approximately 10% [1–3] in short-term results and 1.6% in long-term outcomes [4, 5]. Neurological complications most often involve musculocutaneous and axillary nerves. The changes in neuroanatomy and surgical mistakes during the operation play a main part in the etiology of nerve dysfunctions [6].

13.1.2 Neuroanatomy Before and After Latarjet Procedure

The musculocutaneous nerve arises from the lateral cord of the brachial plexus (C5–C7). Then, it descends laterally and enters the coracobrachialis muscle at the level of the latissimus dorsi tendon. Latarjet estimated the distance between the coracoid process and the entry point of musculocutaneous nerve into the coracobrachialis at 20–120 mm [7]. Further studies show that this distance is approximately 4.8–6.1 cm [8, 9]. The axillary nerve (C5–C6) originates from the posterior cord of the bra-

---

R. Brzóska • P. Janusz • H. Laprus • P. Ranosz
Department of Orthopedics, St. Luke's Clinic,
Bielsko-Biała, Poland

A. Calvo • P. Carnero • A. Rodríguez
Arthrosport, Zaragoza, Spain
e-mail: angelcalvo@me.com; pablocarneromds@hotmail.com; alfredorleon@gmail.com

V. Jermolajevas
Klaipėda Regional Hospital, Klaipėda, Lithuania

L. Lafosse (✉)
Clinique Générale, Annecy, France
e-mail: lafosseshoulder@yahoo.f

T. Lafosse
Service de Chirurgie de la Main, du Membre
Supérieur & des Nerfs Périphériques, SOS Mains,
Hopital Européen Georges Pompidou,
Paris, France

O. Lorbach
Sporthopaedicum Berlin-Straubing-Regensburg-München, Berlin, Germany
e-mail: olaf.lorbach@gmx.de

N. Zurita
IMED Elche Hospital, Alicante, Spain
e-mail: ntzurita@gmail.com

G.M.M.J. Kerkhoffs et al. (eds.), *ESSKA Instructional Course Lecture Book*,
https://doi.org/10.1007/978-3-662-56127-0_13

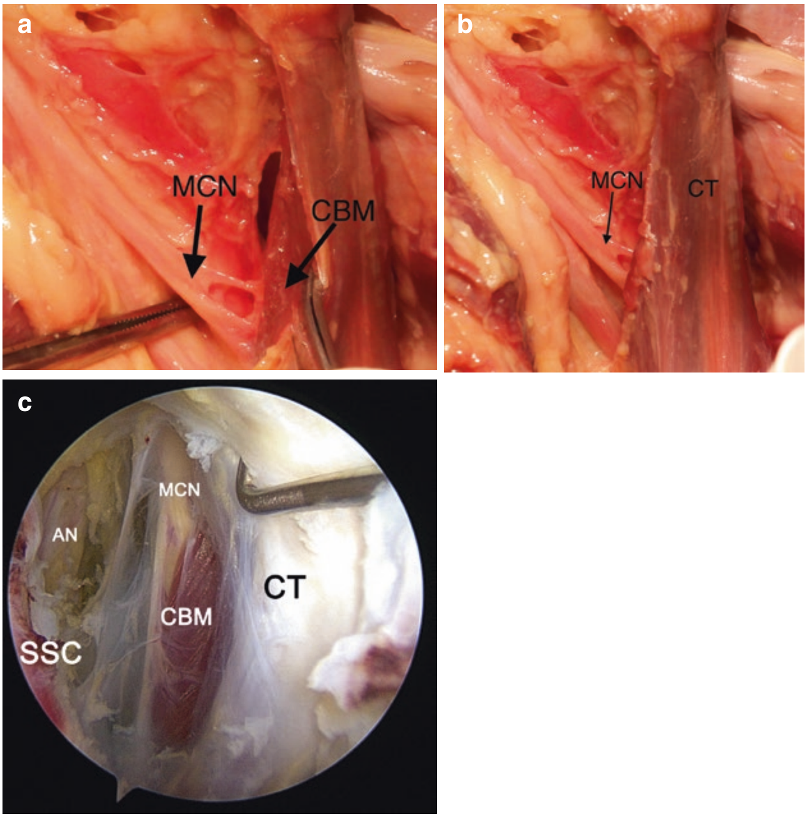


Fig. 13.1 Nerve pathways. *MCN* musculocutaneous nerve; *CBM* coracobrachialis muscle; *CT* conjoint tendon; *AN* axillary nerve; *SSN* suprascapular nerve

chial plexus at the level of the axilla. It lies posteriorly to the axillary artery and anteriorly to the subscapularis muscle. It descends laterally to the inferior border of the subscapularis muscle, where it folds around it in close proximity of brachial capsula, and then exits the axilla posteriorly via the quadrangular space, medial to the surgical neck of humerus, covered with deltoid muscle. In a cadaveric study, Freehill et al. proved that clinically significant changes occur in neuroanatomy after a Latarjet procedure. They found differences in the location of the axillary and the musculocutaneous nerves as they become more inferior and medial compared to their preoperative anatomy [6]. Divergent pathways of musculocutaneous and axillary nerves are seen in normal anatomy. This configuration is visible up to the curvature of the axillary nerve which goes under the subscapularis muscle (Fig. 13.1a–c). After a coracoid abutment procedure, anatomical changes result in consistent overlapping of both nerves and an apparent laxity of the conjoint tendon (Fig. 13.2). Clavert et al. performed a cadaveric study in which they found that, after the cora-

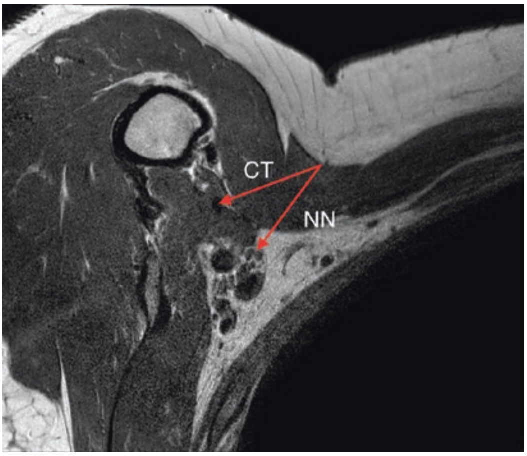


Fig. 13.2 CT image of the nerves and conjoint tendon

coid transfer, the angle between the axis of the coracobrachialis muscle and the musculocutaneous nerve is prolonged from 121° to 136° [6]. Modification of the angle of entry into the muscle along with elongation of the nerve after the Latarjet procedure might lead to transient lesions of the musculocutaneous nerve.

13.1.3 Analysis of Complications

Nerve palsy is the second frequent (after instability recurrence) and the most severe post-Latarjet complication. Neurological complications typically occur as a result of direct injury made by surgical tools, traction, and soft tissue conflict. Short course of musculocutaneous and axillary nerves from brachial plexus to the glenoid area [9] predisposes to traction injuries during conjoint tendon preparation and mobilization. To prevent musculocutaneous nerve injury in the above mentioned traction mechanism, the distance from the top of the coracoid process to the musculocutaneous nerve entry point should be known. However severe variability of entry point distance is proved [7–9], so there is no safe zone for blind conjoint tendon preparation, and musculocutaneous nerve visualization during this preparation is strongly recommended. Moreover, Apaydin et al. prove that entry point distance changes during shoulder joint movements. It is at its longest during abduction to 45° and shortest during abduction to 90° with internal rotation [8]. The axillary nerve which doesn’t have fixation point in any muscles is less exposed to injury. In addition, cadaveric study shows that its course and anatomical relations with other structures does not change during a Latarjet procedure [4].

Musculocutaneous and axillary nerves are placed in dangerous proximity to the subscapularis muscle that is split for coracoid transfer. Hawi et al. show that the musculocutaneous nerve (in 66%) and axillary nerve (in 50%)course within or in front of the split [4], exposing it to injuries. The least traumatic way to perform the split is to visualize tendinous cords, which is possible in arthroscopic technique during the intra-articular phase of the procedure. Analysis of anatomical variability of the tendinous cords shows that division between the superior 2/3 and inferior 1/3 of the subscapularis muscle is located on the level of the fourth tendinous cord counting from the top. The safest way to perform the split relies on increasing external rotation when switching to the stick that is placed inside the split. To protect musculocutaneous and axillary nerves from injury during insertion of the graft, the author creates an additional superior portal close to the anterior aspect of the acromioclavicular joint. Via this approach a specially made Langenbeck retractor is inserted to elevate upper part of the subscapularis which makes split space wider.

13.1.4 Discussion

The authors observed a partial transient musculocutaneous nerve palsy after arthroscopic Latarjet in one illustrative case. The 43-year-old patient developed a transient nerve palsy after 3 hours of driving a car and holding an arm in 30° of abduction on the armrest. A similar case was previously reported by Southam et al. [10]. The authors believe that the risk of neurological complications is related to the thickness of the coracobrachialis muscle at the location of its crossing at the subscapularis muscle split and contact of the coracobrachialis with the pectoralis minor fascia remnants. Endoscopic visualization after the Latarjet procedure shows that internal rotation movement of the shoulder in abduction increases the risk of compression of the musculocutaneous nerve and is further exacerbated by leaving the pectoralis minor fascia remnants. The fascia of pectoralis minor is an elastic structure, but its fibers lie perpendicular to the musculocutaneous and axillary nerves leading to nerve compression while abducting the shoulder. Additionally, this effect is intensified in the movement of internal rotation when the axillary nerve is strained and the musculocutaneous nerve is pressed against the coracobrachialis muscle (Figs. 13.3 and 13.4).

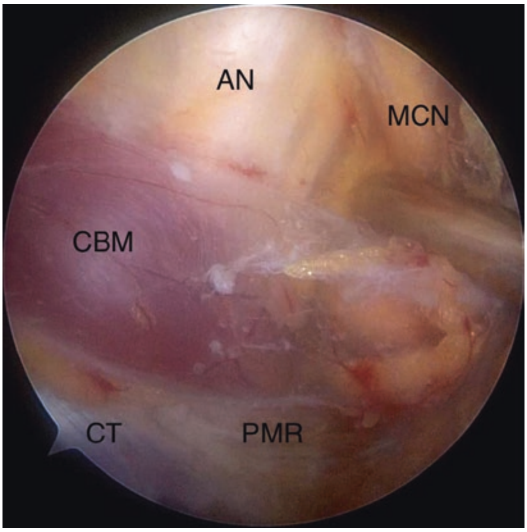


Fig. 13.3 Arthroscopic images demonstrating anatomical changes with motion. *MCN* musculocutaneous nerve; *CBM* coracobrachialis muscle; *CT* conjoint tendon; *AN* axillary nerve; *SSN* suprascapular nerve; *PMR* pectoralis minor fascia remnants

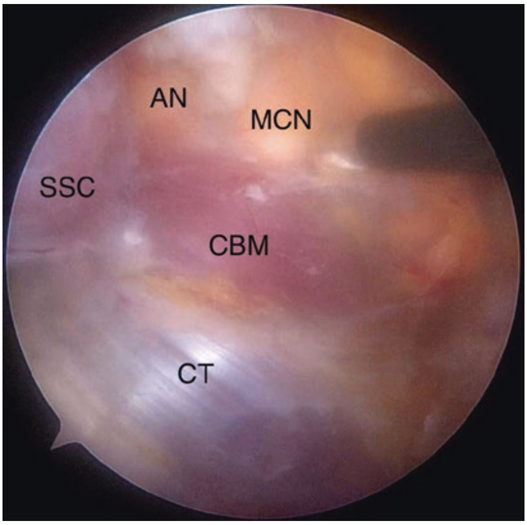


Fig. 13.4 Arthroscopic images demonstrating anatomical changes with motion. *MCN* musculocutaneous nerve; *CBM* coracobrachialis muscle; *CT* conjoint tendon; *AN* axillary nerve; *SSN* suprascapular nerve; *PMR* pectoralis minor fascia remnants

13.2 Arthroscopic Suprapectoral Biceps Tenodesis

Angel Calvo, Pablo Carnero, Alfredo Rodríguez, and Nestor Zurita

Abstract
Pathology of the long head of the biceps tendon is a common cause of anterior shoulder pain. Biceps tenodesis has been proposed as an effective surgical option for pain relief in young active patients while avoiding complications from tenotomy. Suprapectoral tenodesis can be performed as an all-arthroscopic procedure that places the fixation of the biceps tendon at the bicipital groove. This technique would prevent complication from open surgery such as surgical site infection and nerve injury.

13.2.1 Introduction

The long head of the biceps brachii tendon (LHBT) is a well-recognized source of pain in several pathological conditions of the shoulder, both isolated or associated with rotator cuff tears. Multiple congenital variations of intra-articular portion (Fig. 13.5) have been described and proposed as possible causes of pathology [11], making diagnosis a challenging issue. Function of LHBT is not related with active motion of the glenohumeral joint alone, as it seems to contribute with simultaneous elbow and forearm motion [12]. A proprioceptive role in shoulder movements and a secondary stabilization function have also been suggested.

When conservative management fails, surgical treatment is indicated. Biceps tenodesis is often used in active and younger patients in which cosmetic deformity due a Popeye deformity could negatively influence postoperative results. Avoiding arm cramping and maintaining the length-tension relation of the muscle belly are also reasons for choosing biceps tenodesis instead of tenotomy.

The main indication for biceps tenodesis is chronic painful tendinopathy with no improvement after adequate conservative treatment in young and active patients. Development of knowledge about the contribution of LHBT in pain, in rotator cuff tears, and in biceps insertion pathology has extended indication for tenodesis:

- Cuff tears involving LHBT
- Degenerative SLAP over 40 years old
- Painful LHBT instability
- Biceps pulley lesions
- SLAP repair failures

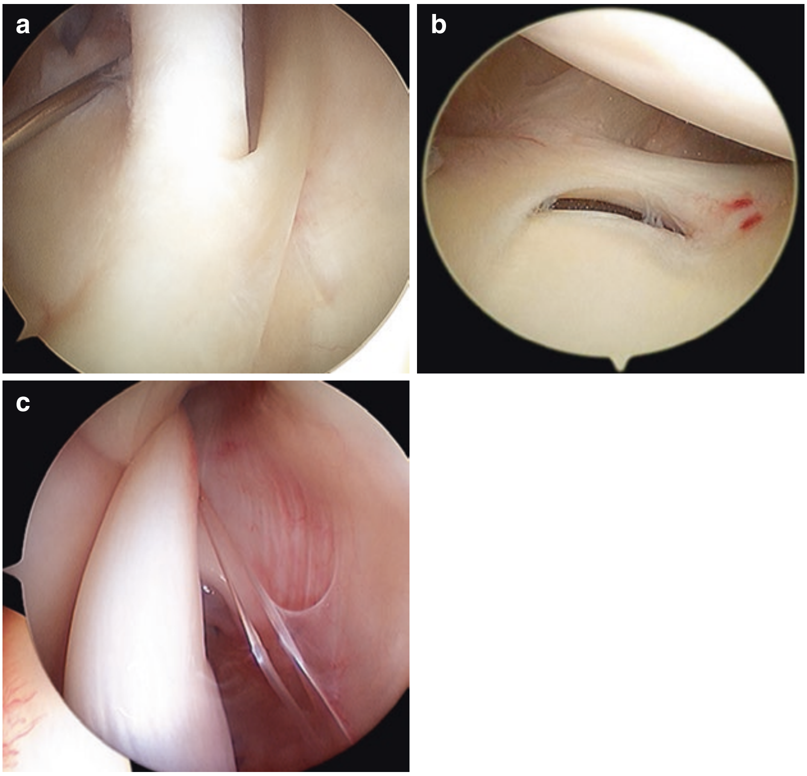


Fig. 13.5 Anatomical variations of intra-articular portion of LHBT

The ideal primary surgical procedure for SLAP acute tears in young athletes is unclear. Tenodesis has been proposed instead of repair as higher levels of patient satisfaction and higher rates of return to presurgical level of activity have been reported [13]. Furthermore, LHBT detachment has minimal effect on glenohumeral kinematics [14]. Further research is necessary to confirm the indication for biceps tenodesis in SLAP acute tears in young overhead athletes.

The optimal location for biceps tenodesis remains controversial. Suprapectoral tenodesis can be performed all arthroscopically and avoids complications of open surgery, which is often needed in subpectoral tenodesis. Risks of musculocutaneous and radial nerve injuries and humeral fractures have also been reported after subpectoral tenodesis [15], and risk is minimized with arthroscopic suprapectoral tenodesis. On the other hand, it is a highly technically demanding procedure that requires an experienced arthroscopist. Choosing the optimal point for tenodesis is critical, as an increase in tension due to malpositioning could evolve into an early failure of tenodesis [16].

Method of fixation is a key point in success of LHBT tenodesis. Biomechanical testing shows interference screws as the strongest construct [17] and better pullout. However, this technique is not free from complications like osteolysis around the implant and tendon injury or rupture, which may require latter revision.

We hereby describe an all-arthroscopic easy and reproducible procedure for suprapectoral LHBT intraosseous tenodesis with a suspensory fixation device.

13.2.2 Surgical Technique

13.2.2.1 Patient Position

Both lateral and beach chair position can be used, though we recommend the last option as anterior and posterior aspects of subacromial space are easily reached and external rotation of shoulder is allowed, in order to expose the bicipital groove.

Portals (Fig. 13.6):

1. Standard posterior portal: made in the soft spot between infraspinatus and teres minor muscles. This is the initial viewing portal.

2. Anterior standard portal: bone tunnel reaming and implant insertion are performed from here.
3. Anterolateral (AL) portal: aligned with the anterior edge of the acromion. Acromioplasty is performed from here and serves as the viewing portal during exposition of the bicipital groove.
4. Anterior accessory portal (AA): at the midpoint between the anterior and the anterolateral portals, just on the surface of the bicipital groove. Its main function is exposing the biceps, but it can act as the viewing portal, and it provides excellent view of the tunnel. Bone tunnels can also be reamed from here.

13.2.2.2 Technique

A complete examination of the glenohumeral joint is done with the arthroscope placed in the posterior portal. LHBT lesions are evaluated. An 8 mm cannula is placed in the anterior portal, and a suture is passed through the base of the LHTB using the lasso loop technique. This suture secures the end of the tendon and prevents its retraction. Additionally, a spinal needle is inserted through the AA portal and goes through the tendon. This needle will mark the bicipital groove in the subacromial space (Fig. 13.7). Then the tenotomy is completed.

The subacromial space is explored through the posterior portal. Bursectomy and acromioplasty are performed from the AL portal. This will help locate the needle. The cannula in the anterior portal will be visible as well (Fig. 13.8). Then the arthroscope is switched to the AL portal and the AA portal is made where the needle is inserted.

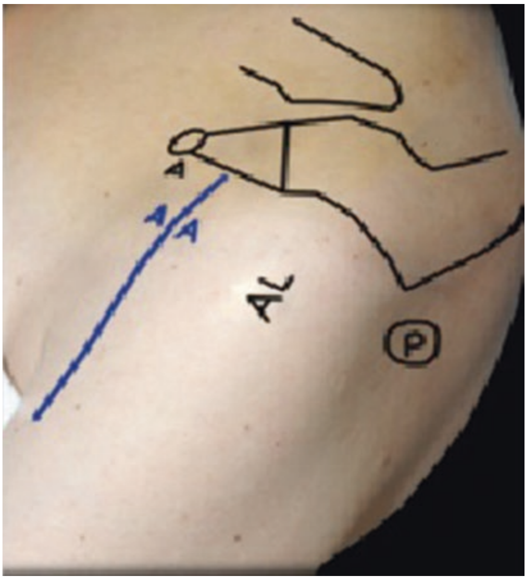


Fig. 13.6 Portals needed for the procedure

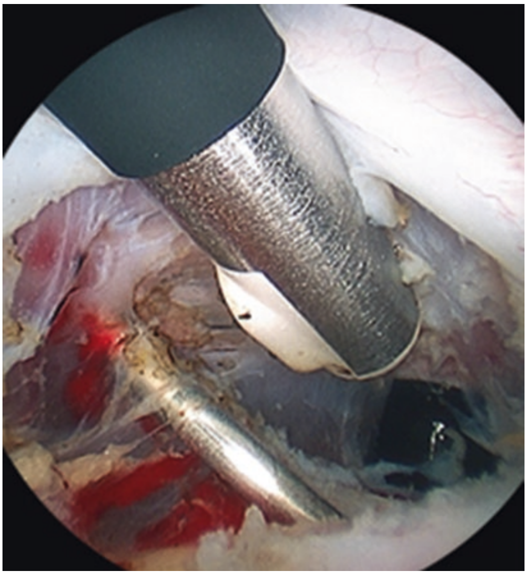


Fig. 13.8 The spinal needle is visible in subacromial space once bursectomy is completed

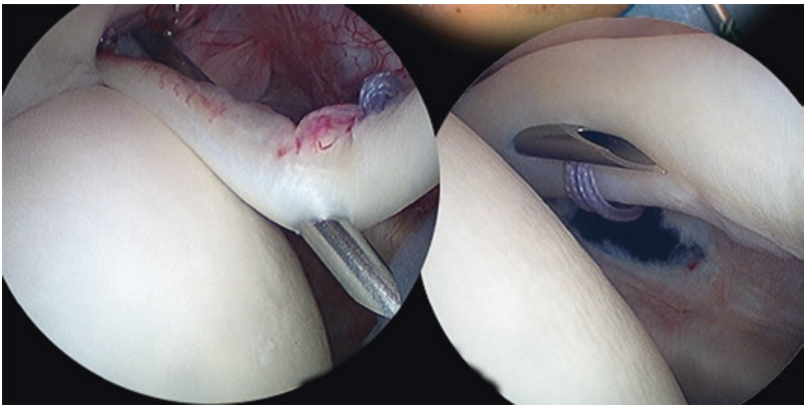


Fig. 13.7 LHBT is secured by the suture and the spinal needle. Both will prevent retraction when tenotomy is performed

The groove is located and opened with a radiofrequency electrode or a regular scalpel. The transverse ligament and surrounding tissue are released to make the LHBT visible in the groove (Fig. 13.9). It is important to extensively release the transverse ligament so that the tendon will be mobilized easily. LHBT is then grasped and exposed through the AA portal. A Krackow-type traction suture is used starting 30 mm of the free end of the tendon with a high-resistance suture (Fig. 13.10).

The arthroscope is kept in the AL portal to make the bone tunnels because a perfect view of the groove is obtained from here. A complete debridement of the floor of the groove is performed. Through the anterior portal, the guide wire with an eyelet is then inserted through the groove from the anterior aspect of the humerus to the posterior. The insertion site must be 2–3 cm from the articular cartilage start, centered in the groove and perpendicular, aiming to a point slightly lower than the posterior viewing portal (Fig. 13.11). The wire is exposed at the posterior part of the shoulder, tapping it gently in order to avoid injuries of the axillary nerve. Then a 30 mm length tunnel is drilled with a 7.0 mm drill bit. The base of the tunnel and the posterior cortex are drilled with a 4.5 mm drill bit. Total length of the tunnel is usually about 40 mm. A millimetric bit is used to confirm it. The arthroscope is switched to the AA portal to confirm the correct position of the tunnel and the integrity of the walls. All the debris tissue in the tunnel or around is removed, and the inferior edge of the tunnel entrance is smoothed in order to diminish friction between the tendon and the bone.

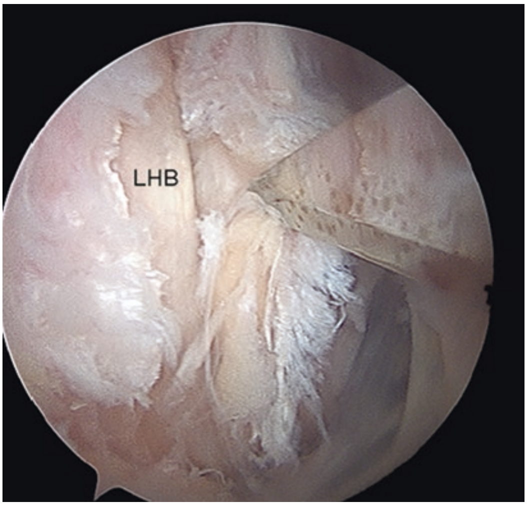


Fig. 13.9 The groove is opened to release the LHBT

Fig. 13.10 Preparation of the tendon exposed through the AA portal

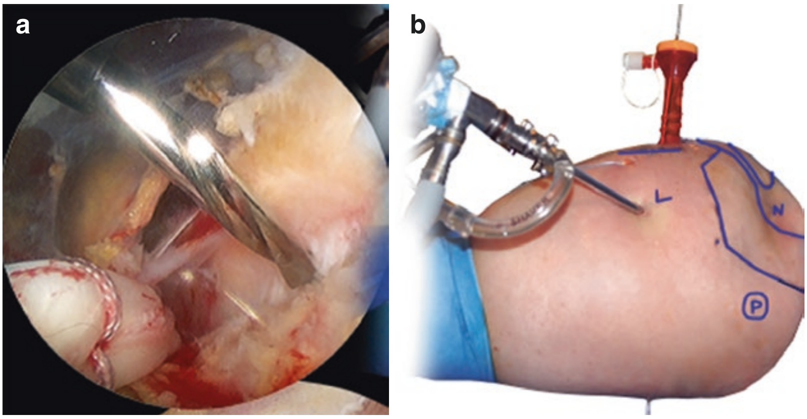


Fig. 13.11 Guide wire inserted in the groove. Note the direction in a saggital view

The tendon is then passed to the anterior portal. The suspension system used is made with a pen in the double loop at 40 mm, and the traction suture at the free end of the tendon is tied on the double loop of the device trying to leave 5 mm between the tendon and the loop. This distance corresponds to the base of the bone tunnel drilled by the 4.5 mm drill bit (Fig. 13.12). The traction suture is threaded to the guide needle eyelet.

Once the tendon is knotted to the device, a gentle pull is done from the guide wire at the posterior aspect of the shoulder. Arthroscopic vision through the AL portal will confirm correct entering of the implant at the bone tunnel. It will be felt that the implant crosses the posterior cortex when resistance decreases, and it will be confirmed if the 40 mm pen mark at the double loop reaches the entry of the tunnel (Fig. 13.13). Then the double loop is slightly pulled to make the implant rotate, and it will be locked to the posterior cortex of the humerus. Lastly, the sliding suture is pulled to shorten the double loop, and the tendon will be inserted in the tunnel with arthroscopic control. The tendon is inserted 30 mm length in the bone tunnel, coinciding with the beginning of the traction suture. It is quite important to confirm adequate tension and fixation of the tendon with a probe, avoiding overtension (Fig. 13.14).

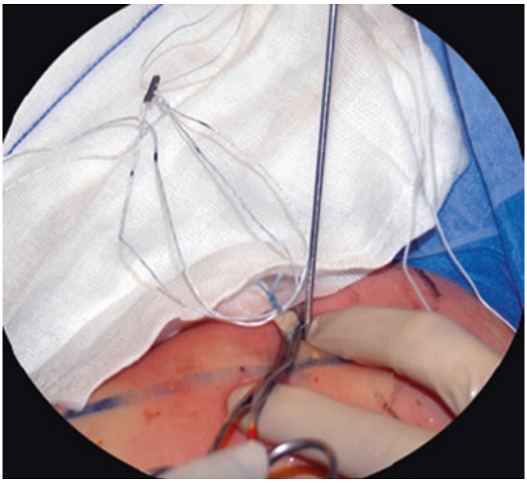


Fig. 13.12 Tendon sutured to the double loop of the anchor

13.2.3 Postoperative Care

A sling is used for pain control for a few days, and rehabilitation protocols can be started as soon as possible. The technique described provides a strong fixation that allows early active mobilization with reliability (Fig. 13.15). We recommend not performing active counter-resistance flexion of the elbow during the first 2 months.

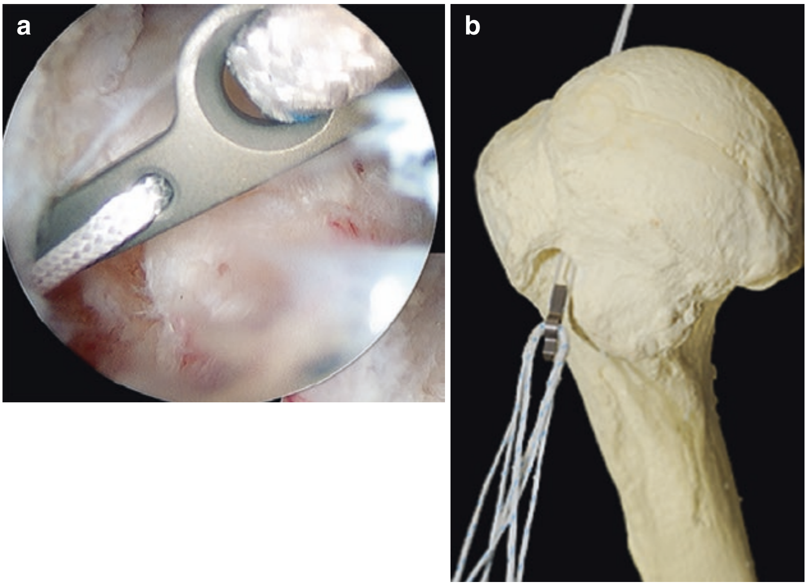


Fig. 13.13 Implant entry at the bone tunnel by traction of the guide wire

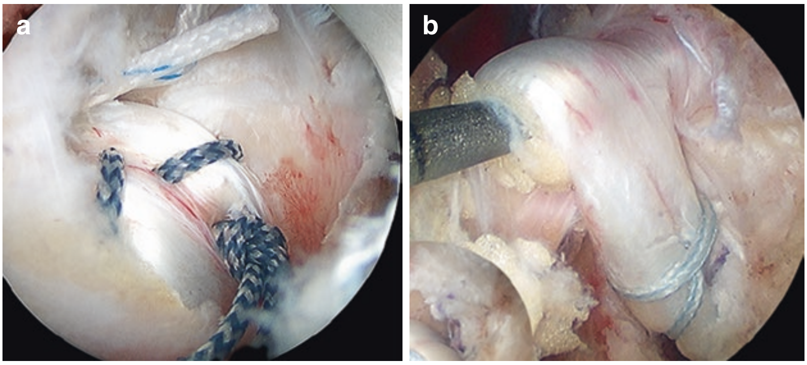


Fig. 13.14 Tendon inserted in the tunnel. Probe checking no overtension

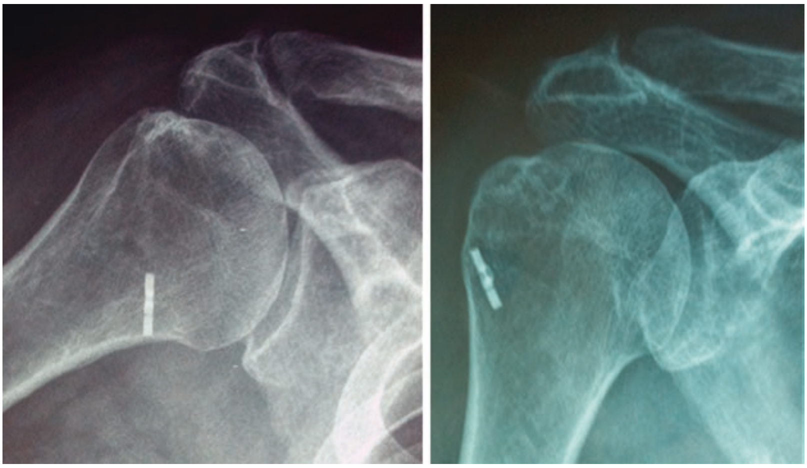


Fig. 13.15 Postoperative Rx

13.2.4 Conclusion

Biceps suprapectoral tenodesis is a viable option in biceps pathology in young active patients that can be performed entirely arthroscopically. Experienced surgeons are needed to obtain satisfactory results. We describe an easy and reproducible technique with a suspensory fixation device that allows early postoperative rehabilitation.

13.3 Brachial Plexus Endoscopic Release

Thibault Lafosse and Laurent Lafosse

Recent progress made in shoulder arthroscopy has led us to work beyond the limits of the glenohumeral joint (GH), around the coracoid process, and the brachial plexus (BP). Endoscopic work in this area requires a good visualization of the BP, which is very close. Therefore, in order to protect the BP, we became accustomed to approaching and neurolysing it endoscopically in our current practice during many different arthroscopic shoulder surgery procedures. Whenever working close to the BP, we encourage surgeons to visualize the nerves in order to protect it. We believe it will decrease the neurologic complication rate, particularly in arthroscopic Latarjet procedures, extended arthrolysis, and subscapularis repairs.

This led us to start treating neurogenic thoracic outlet syndrome (NTOS) endoscopically. NTOS is an uncommon condition occurring in rather young and healthy patients [18]. NTOS is caused by a neurologic compression of the BP which can occur at three preferred locations: the scalene triangle, the space between the first rib and the clavicle, and/or compression beneath the coracoid and the pectoralis minor (PM) muscle.

The symptoms of BP compression in NTOS are typified by pain, numbness, and paresthesia in the neck, occipital area, chest wall, arm, and hand. The examination shows reproducible exacerbation of the symptoms by elevation and external rotation of the upper extremity, along with an irritative pseudo Tinel's sign around the coracoid process. In most of the cases, the cause is not identified, and despite many studies, the treatment of NTOS is still controversial. We recently published our results on a series of 36 patients treated endoscopically for NTOS, along with the surgical technique [18].

13.3.1 Technique

13.3.1.1 Installation

The patient is set up in a beach chair position and operated on under general anesthesia, combined with an interscalenic locoregional anesthesia.

13.3.1.2 Endoscopic Portals (Fig. 13.16)

Eight endoscopic portals are needed, four supraclavicular and four infraclavicular.

Supraclavicular Portals

C and D are two subacromial portals, respectively, lateral, located at the level of the middle of the acromion 2 cm distal to its lateral border, and anterolateral, 2 cm distal to the anterior angle of the acromion. The two transtrapezial portals are located 2.5 cm posterior from the anterior border of the trapezius, with the lateral transtrapezial portals (TT1) at the level of the suprascapular notch (created under endoscopic control from C to D portals) and the medial transtrapezial portal (TT2) at the level of the middle of the clavicle (created under endoscopic control from D to TT1 portals).

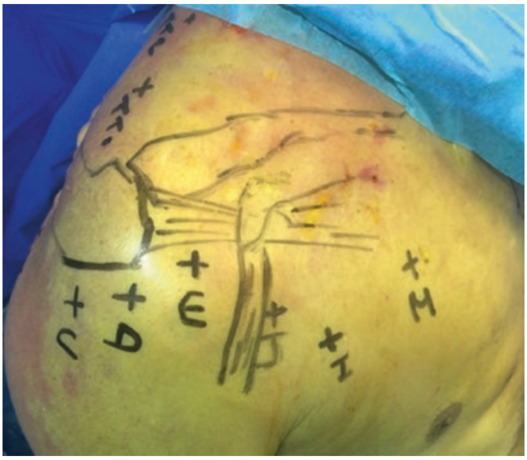


Fig. 13.16 Endoscopic portals

Infraclavicular Portals

E portal is anterior, 2 cm distal to the acromioclavicular joint, facing the rotator interval. I portal is in the axis of the coracoid process, 2–3 cm below. J portal is at mid-distance from I to E portals. Finally, M portal is medial, 4 cm distal to the clavicle and 3 cm medial to the coracoid process.

13.3.1.3 Supraclavicular Plexus and Interscalenic Triangle

Suprascapular Nerve Release

The first step is to release the suprascapular nerve (SSN) distally, from the transverse ligament with a subacromial approach. C portal is used as a visualization portal and D portal for instrumentation. The anterior border of the supraspinatus muscle is followed until the coracoclavicular ligaments. At this point, perpendicular to the coracoclavicular ligaments, the transverse ligament is identified, under which the SSN lies (Fig. 13.17). TT1 portal is created under endoscopic control, using a tracking needle before incising through the trapezius muscle. An endoscopic cutting device is then introduced in this portal toward the suprascapular notch, so the transverse ligament is cut and the suprascapular nerve is released.

Interscalenic Space

The TT2 portal is opened under endoscopic control, also using a tracking needle. The scope is switched to the TT1 portal, and the instrumentation goes through the TT2 allowing fulfilling proximally the dissection of the SSN up to the upper trunk. The upper trunk is released from the surrounding fibrous bands, exposing the middle and the lower trunks between in superior part of the interscalenic space.

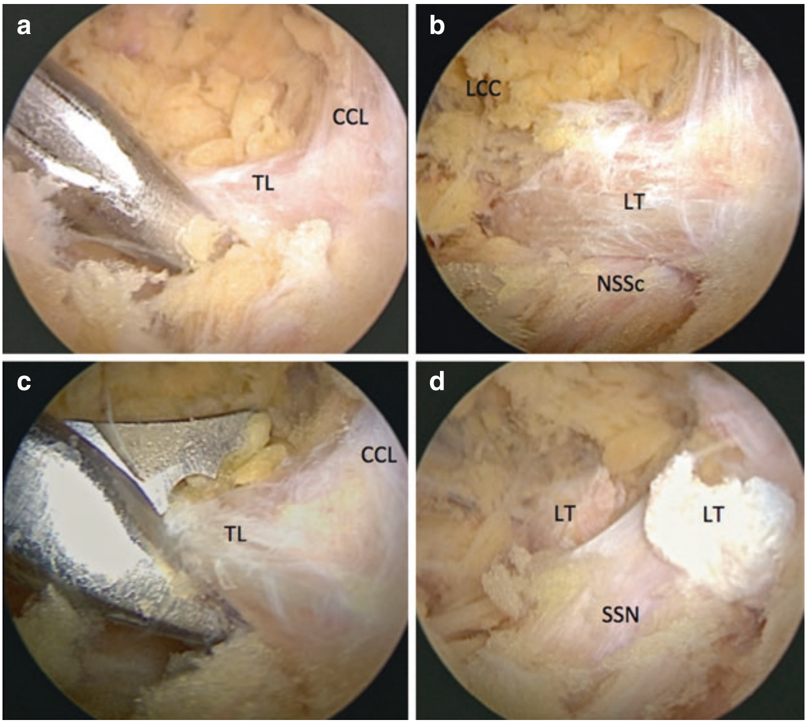


Fig. 13.17 Suprascapular nerve release

A smooth trocar is used to perform an intraneural dissection, but no scalenectomy, first rib resection, or root dissection is needed.

13.3.1.4 Infraclavicular Brachial Plexus

Exposition

The dissection is started from the subacromial area, with the scope through the C portal and the instruments through the D portal. The E portal is then opened under endoscopic control with a tracking needle. After opening the clavipectoral fascia, the conjoint tendon (CT) and the coracoid process are exposed to enlarge the retropectoral space anteriorly, between the coracoid process and the PM posteriorly and the pectoralis major anteriorly, using a smooth trocar and the water flow. Hemostasis is often needed and performed with radiofrequency assistance. Then, I portal is created under endoscopic control using a tracking needle, and J portal is opened at mid-distance from D to I portals. The scope is introduced in it, facing the coracoid process so the operator can see the PM muscle and the CT very clearly. The limit between the conjoint tendon and the PM muscle bellies can be difficult to visualize; however, since this is the only way to safely expose the musculocutaneous nerve lying below, it is of primary importance for the operator to identify it. The M portal is finally opened, allowing further proximal dissection, particularly to access the upper border of the PM and the medial border of the coracoid process.

Retro-pectoralis Minor Space (Fig. 13.18)

Finally, the space between the CT and the PM is opened to visualize the BP terminal branches. Only then, the PM tendon can be detached from the coracoid process. The three cords are dissected distally, by carefully cutting the surrounding fibrous bands.

The lateral cord (LC) allows the musculocutaneous nerve and the median nerve lateral branch, which are the first terminal branches to be visualized. After reclining these two nerves along with the axillary artery, the posterior cord (PC) and its two terminal branches, the axillary and radial nerves, are found heading posteriorly to the quadrilateral space.

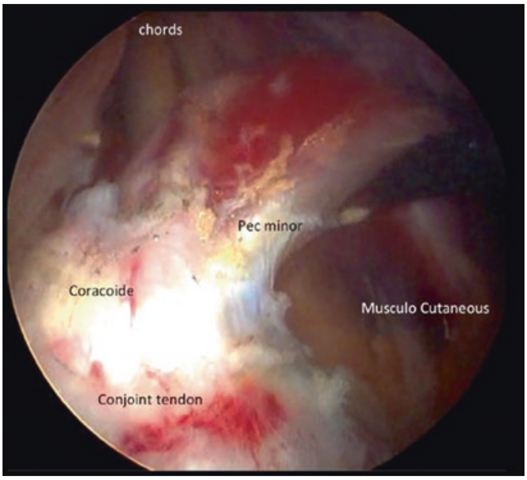


Fig. 13.18 Retro-pectoralis minor space

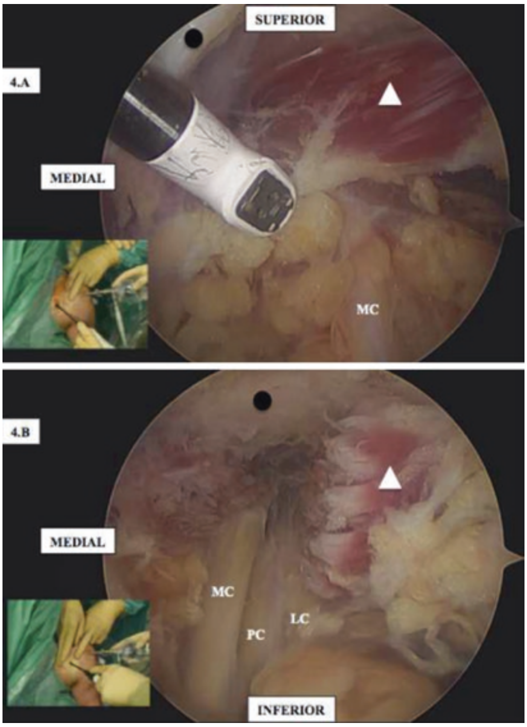


Fig. 13.19 Costoclavicular space. *MC* Medial cord; *LC* Lateral cord; *PC* Posterior cord

Costoclavicular Space (Fig. 13.19a, b)
Following the upper border of the PM tendon, the cords are found under the subclavian muscle. To increase the costoclavicular space, the subclavian muscle is detached from the clavicle, on a distance as large as the width of the three cords.

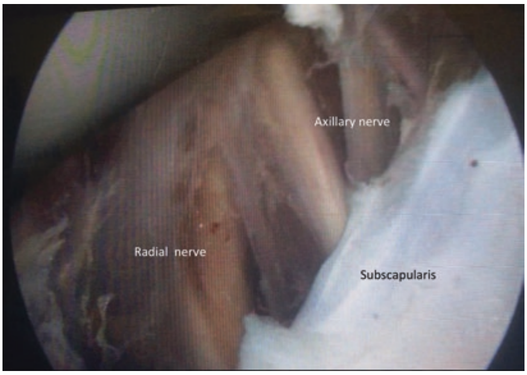


Fig. 13.20 Retrocoracoid approach

Retrocoracoid Approach (Fig. 13.20)
The release of the posterior cord and its terminal branches is completed with a posterior approach posterior through the C and E portals, following the anterior border of the subscapularis muscle. The axillary nerve and, immediately forward, the radial nerve are visualized.

13.3.2 Clinical Presentations, Indications

In our experience, more than 60 patients benefited from an endoscopic infraclavicular plexus release with a pectoralis minor section. More than 150 patients benefited from a suprascapular nerve release, many of which were released very proximally close to the superior trunk.

Two-thirds of them benefited from this procedure for an infraclavicular thoracic outlet syndrome, and one-third had it performed along with an extra-articular endoscopic procedure such as arthroscopic Latarjet, extensive subscapularis tears endoscopic repair, endoscopic arthrolysis, and endoscopic Latarjet revision with Eden-Hybinette bone block endoscopic procedure.

13.3.2.1 NTOS

For many surgeons, NTOS remains a difficult entity to treat endoscopically. Patients should be carefully selected as the results are expected for some very specific cases. Preoperative EMG, Doppler ultrasound on the subclavian vessels, cervical spine standard radiographs, and MRI should be performed and be normal in order to eliminate a cause for NTOS that would require an open procedure (e.g., cervical rib, hypertrophic transverse cervical process, arterial or venous component of the TOS). In our practice, we select the patients eligible for endoscopic NTOS release based on several clinical criteria: the complaint of a chronic pain lasting for more than 6 months and for which a conservative physiotherapy has failed, pain with criteria meeting those previously described of the NTOS symptoms, irritative pseudo Tinel's sign in the infraclavicular area, and reproducibility and exacerbation of the symptoms in abduction and external rotation that we call the BP stretch test. Very satisfactory results can be expected in patients meeting these requirements.

13.3.2.2 BP Exploration

In our nerve unit, we are frequently referred patients with BP palsies following shoulder dislocations, or more rarely, arthroscopic procedures, without an anatomic lesion on the MRI or ultrasound imaging of the nerve. Those patients are improved by the BP neurolysis, and may recover quicker, even though they could have recovered without any neurolysis. However, the indication of an open neurolysis of the BP is questionable, since it implies a great scar and a lengthy recovery and it makes a revision far more complicated. On the other hand, endoscopic release is a safe, quick, and postoperatively easy procedure, with similar to better results than an open procedure [18]. Our indications of BP release in cases of Sunderland 2 to 3 palsy have increased for those reasons.

13.4 Inferior to Shoulder-Arthroscopic Anatomy for Teres Major and Latissimus Dorsi Transfer

Viktoras Jermolajevas

In recent years several arthroscopic assisted or full arthroscopic latissimus dorsi transfers were developed [19–21]. This procedure remains one of the most difficult in the shoulder arthroscopy. Thorough knowledge of anatomy is mandatory, as several nerves and arteries are in close proximity during portal placement, latissimus dorsi release or space creation for transfer passage.

Standard portals are used in the beginning. To reach the posteroinferior part of the shoulder, a simple approach is used. The arthroscope is placed in the anterolateral (AL) portal. Using a shaver from the posterolateral (PL) portal, space is created between the deltoid (Del) and posterior cuff—infraspinatus (Inf)—. After removing the bursa, the deltoid fascia is incised horizontally at the Inf and teres minor (TM) muscle junction level. Fascia resection should be accompanied with blunt space enlargement and should be continued medial to the glenoid to spare the posterior branch of the axillary nerve (pAx) (Fig. 13.21: Posterior release). If visualization of the intermedius or anterior branch of the axillar nerve is needed, full resection of the posterior and posterolateral Del fascia is necessary [22]. These branches run 1–2 cm more lateral at the same level as the pAx nerve branch. Deeper dissection is done from the posterior portal (PP), looking from the PL portal. Shaving is performed medial to the pAx nerve branch. Fat tissue is resected following an inferomedial direction, and it is possible to dissect the vertical fibers of the posterior aspect of the long head of the triceps (pLTr). The circumflex scapular artery runs just medial to the pLTr and should be coagulated to prevent postoperative hematoma.

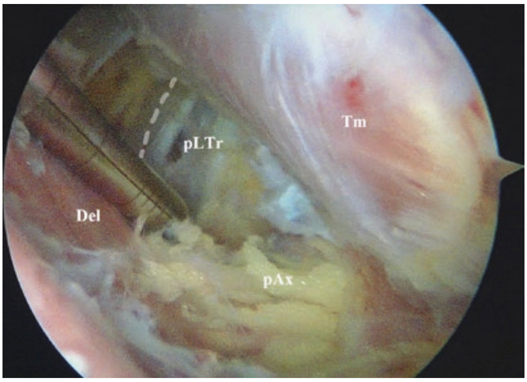


Fig. 13.21 Posterior Release: Creation of a space between the deltoid (Del) and the teres major (Tm) medial to the glenoid in order to protect the posterior branch of the axillary nerve (pAx). Posterior fibers of the triceps tendon (pLTr) are visible and may be partially dissected

More medially and inferiorly, after dissecting some fat tissue at this area, horizontal fibers of posterosuperior part of the teres major (TM) muscle are found on the posterior side, opposite to the triceps fibers. During shaving, more medial than inferior shaving should be done, to prevent axillar skin penetration. Inferior TM muscle fibers and pLTr angle should guide correct dissection. Here, a standard urinary catheter is inserted from the posterior portal and its balloon is inflated for future LD tendon subacromial passage.

Anteroinferior anatomy is more complex. The scope is switched to the anterosuperior (A) portal, looking distally, following the LHB tendon in its groove. For better visualization, arms are placed in adducted and flexed position. The lateral edge of the conjoined tendon (Con) should be used as a reference. Shaving is done through the AL portal, until the superior border of the pectoralis major tendon (PM) is clearly visible. Just lateral, about 1 cm to the PM insertion, the anterior branch of the Ax nerve enters the anterior deltoid muscle. At the same level medial to the PM insertion, circumflex vessels (3S), the so-called three sisters, are identified, as an inferior border of the subscapularis tendon (Fig. 13.22: Anterior release). Just underneath, the superior border of the LD tendon is visible. A specific suprapectoral portal (SP) just above the PM is created. The needle is placed 1 inch superior to the axillar crease and directed just above the PM tendon. In case of struggling with instruments, 1 cm release of the superior part of the PM tendon is done. It allows to bluntly create some space between three structures: anteriorly conjoined tendon, laterally PM, and posteriorly LD tendons. More space is created if release of the conjoint tendon lateral edge and PM posterior surface is done. Shaving is performed inferiorly to the “three sisters” until entire exposure of the LD tendon fibers running medial to lateral. The radial nerve, found inferiorly and crossing border of the TM tendon 2–3 cm medial to the humeral insertion, is an inferior limit for dissection [23]. The LD tendon is narrower than the TM. It does not cover the superior 5 mm of the TM muscle and 1 cm inferior TM tendon part. Medially LD tendon dissection is continued for as much as the scope and shaver length allow. LD neurovascular supply enters the muscle about 14 cm medial to the humeral insertion and standard scopes or instruments are not able to reach so medial.

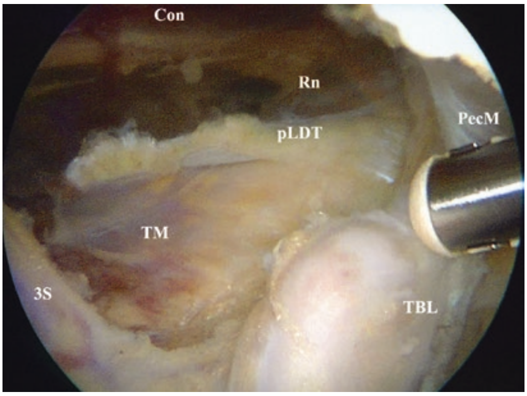


Fig. 13.22 Anterior Release: Space is bluntly created between 3 structures: anteriorly conjoined tendon (Con), laterally Pec. Major (PecM) and posteriorly Lat. dorsi tendons (pLDT). Inferiorly to “three sisters” (3S) after exposure of LD tendon fibers, Radial nerve (Rn) is found inferiorly and crossing border of TM tendon 2 to 3 cm medial to humeral insertion

13.5 Decompression of Suprascapular Nerve at the Spinoglenoid and Suprascapular Notches

Olaf Lorbach

13.5.1 Introduction

Entrapment of the suprascapular nerve is a rare disease, which may occur at the spinoglenoid and suprascapular notches. Anatomically, the nerve travels along the posterior border of the clavicle to reach the superior border of the scapula before diving into the suprascapular notch where the nerve and the artery are divided by the transverse scapular ligament before diving in the suprascapular notch. The roof of the notch is built by the transverse scapular ligament. Therefore, hypertrophy may lead to stenosis. Moreover, the geometry of the notch that is variable may lead to compression of the nerve as well. The nerve further travels along the supraspinatus fossa coming approximately 2 cm of the posterior glenoid rim at the level of the spine of the scapula. The suprascapular nerve then travels laterally around the scapular spine to descend into the infraspinatus fossa passing under the spinoglenoid ligament.

Reasons for a possible entrapment of the nerve include tumors, with encroachment of the suprascapular notch by intrinsic or extrinsic masses with ganglion cysts representing one of the most common of these lesions. Moreover, extremes of scapular motion, shoulder motion like hyperabduction, or repetitive microtrauma may affect the nerve as well.

13.5.2 Diagnosis

Often a history of trauma or of a sporting activity with repetitive use such as volleyball, basketball, tennis, weight lifting, and swimming is evident. In overhead athletes, injury to this nerve may occur from repetitive traction and microtrauma. Moreover, the spinoglenoid ligament tightens when the shoulder is in the position for overhead throwing, resulting in increased pressure on the suprascapular nerve. While sports activities can often lead to suprascapular neuropathy, workers may be plagued with this disease as well because of the nature of the repetitive overhead work they may perform daily.

During examination atrophy of the supraspinatus and/or infraspinatus may be evident. A patient may describe pain when reaching across his or her body. Moreover, tenderness may exist in the suprascapular notch between the clavicle and scapular spine, located approximately 3 cm medial and anterior to Neviaser portal.

Weakness of external rotation should be tested with the arm at the side and may be present without any significant pain. Furthermore, provocative tests especially looking for possible labral pathologies as well as the cross-arm adduction test need to be performed.

Magnetic resonance imaging (MRI) is a useful tool to identify soft tissue masses like a ganglion cyst and will give important information to identify their presence, location, and size. Muscle atrophy and fatty infiltration of the supraspinatus and infraspinatus can be visualized as well.

Furthermore, diagnostic injections using 1% of lidocaine may be used to differentiate between other pathologies and to confirm the diagnosis of suprascapular nerve entrapment with sometimes an almost immediate pain relief. However, a negative test does not rule out the disease. Diagnostic injections in other areas of the shoulder may also be helpful to identify other shoulder pathologies.

13.5.3 Treatment Options

13.5.3.1 Nonoperative Treatment

Initial treatment for an isolated suprascapular nerve compression is rest, activity modification, anti-inflammatory medications, physical therapy to maintain a normal range of motion, and strengthening of the shoulder girdle with return to sport after proprioceptive exercises. Often patients will need physical therapy to enhance scapular stability and resistive strengthening programs.

If there is, however, a space-occupying lesion, nonoperative treatment should not be continued for more than 6–8 weeks.

13.5.3.2 Operative Treatment

Operative treatment options include decompression of the suprascapular nerve by arthroscopic release of the transverse scapular ligament or endoscopic release of the spinoglenoid ligament. Moreover, several authors advocate for treating intra-articular lesions such as the labral tear. These authors believe that if the one-way valve mechanism is corrected, the cyst will never return. Some authors often just treat the SLAP tear and ignore the cyst as they believe it will decompress itself after correction of the intra-articular pathology. Other authors arthroscopically decompress the cyst, debride the frayed labrum, and repair and stabilize the SLAP lesion as well. If the labrum is intact, incision of the capsule above the labrum just posterior to the biceps can be performed in order to decompress the ganglion cyst.

Conflict of Interest The authors report no conflict of interest.

References

1. Shah AA, Butler RB, Romanowski J, Goel D, et al. Short-term complications of the Latarjet procedure. JBJS. 2012;94(6):495–501.
2. Gartsman GM, Jr WNW, Connor DPO, Elkousy HA, Edwards TB. Immediate and early complications of the open Latarjet procedure: a retrospective review of a large consecutive case series. J Shoulder Elb Surg. 2017;26(1):68–72.
3. Delaney RA, Bch MB, Freehill MT, Janfaza DR, Vlassakov KV, Higgins LD, et al. 2014 Neer Award Paper: neuromonitoring the Latarjet procedure. J Shoulder Elb Surg. 2014;23(10):1473–80.
4. Hawi N, Reinhold A, Suero EM, Liodakis E, Przyklenk S, Brandes J, et al. The anatomic basis for the arthroscopic Latarjet procedure. Am J Sports Med. 2016;44(2):497–503.
5. Griesser MJ, Harris JD, McCoy BW, et al. Complications and re-operations after Bristow-Latarjet shoulder stabilization: a systematic review. J Shoulder Elb Surg. 2013;22(2):286–92.
6. Clavert P, Lutz JC, Wolfram-Gabel R, Kempf JF, Kahn JL. Relationships of the musculocutaneous nerve and the coracobrachialis during coracoid abutment procedure (Latarjet procedure). Surg Radiol Anat. 2009;31(1):49–53.
7. Latarjet M, Neidhardt J, Morin A, Autissier J. L’entrée du nerf musculo-cutanédans le muscle coraco-brachial. C R Assoc Anat. 1967;138:755.
8. Apaydin N, Bozkurt M, Sen T, Loukas M, Tubbs RS, Ugurlu M, et al. Effects of the adducted or abducted position of the arm on the course of the musculocutaneous nerve during anterior approaches to the shoulder. Surg Radiol Anat. 2008;30(4):355–60.
9. Klepps SJ, Goldfarb C, Flatow E, Galatz LM, Yamaguchi K, Louis S, et al. Anatomic evaluation of the subcoracoid pectoralis major transfer in human cadavers. J Shoulder Elb Surg. 2001;10:453–9.
10. Southam JD, Greis PE. Delayed, transient musculocutaneous nerve palsy after the Latarjet procedure. J Shoulder Elb Surg. 2012;21(5):e8–11.
11. Dierickx C, Ceccarelli E, Conti M, Vanlommel J, Castagna A. Variations of the intra-articular portion of the long head of the biceps tendon: a classification of embryologically explained variations. J Shoulder Elb Surg. 2009;18(4):556–65.
12. Levy AS, Kelly BT, Lintner SA, Osbahr DC, Speer KP. Function of the long head of the biceps at the shoulder: electromyographic analysis. J Shoulder Elb Surg. 2001;10(3):250–5.
13. Boileau P, Parratte S, Chuinard C, Roussanne Y, Shia D, Bicknell R. Arthroscopic treatment of isolated type II SLAP lesions: biceps tenodesis as an alternative to reinsertion. Am J Sports Med. 2009;37(5):929–36.
14. Giphart JE, Elser F, Dewing CB, Torry MR, Millett PJ. The long head of the biceps tendon has minimal effect on in vivo glenohumeral kinematics: a biplane fluoroscopy study. Am J Sports Med. 2012;40(1):202–12.
15. Sears BW, Spencer EE, Getz CL. Humeral fracture following subpectoral biceps tenodesis in 2 active, healthy patients. J Shoulder Elb Surg. 2011;20(6):e7–11.
16. David TS, Schildhorn JC. Arthroscopic suprapectoral tenodesis of the long head biceps: reproducing an anatomic length-tension relationship. Arthrosc Tech. 2012;1(1):e127–32.
17. Ozalay M, Akpinar S, Karaeminogullari O, Balcik C, Tasci A, Tandogan RN, et al. Mechanical strength of four different biceps tenodesis techniques. Arthrosc J Arthrosc Relat Surg. 2005;21(8):992–8.
18. Lafosse T, Le Hanneur M, Lafosse L. All-endoscopic brachial plexus complete neurolysis for idiopathic neurogenic thoracic outlet syndrome: a prospective case series. Arthroscopy. 2017;33(8):1449–57.
19. Gervasi E, Causero A, Parodi PC, Raimondo D, Tancredi G. Arthroscopic latissimus dorsi transfer. Arthrosc J Arthrosc Relat Surg. 2007;23:1243.e1–4.
20. Grimberg J, Kany J. Latissimus dorsi tendon transfer for irreparable postero-superior cuff tears: current concepts, indications, and recent advances. Curr Rev Musculoskelet Med. 2014;7:22–32.
21. Jermolajevas V, Kordasiewicz B. Arthroscopically assisted latissimus dorsi tendon transfer in beach-chair position. Arthrosc Tech. 2015;4:e359.
22. Ball CM, Steger T, Galatz LM, Yamaguchi K. The posterior branch of the axillary nerve: an anatomic study. J Bone Joint Surg Am. 2003;85-A:1497–501.
23. Pearle AD, Kelly BT, Voos JE, Chehab EL, Warren RF. Surgical technique and anatomic study of latissimus dorsi and teres major transfers. J Bone Joint Surg Am. 2006;88:1524–31.

14 Unicompartmental Knee Arthroplasty

S. Lustig, S.T. Donell, G. Pagenstert, P. Henle,
S. Oussedik, J. Beckmann, and F. Haddad

14.1 Introduction

Unicompartmental knee arthroplasty (UKA) is now a well-established procedure in the armamentarium of an orthopaedic surgeon whose practice deals with managing the degenerating knee. Total knee arthroplasty (TKA) predominates in the management of knee osteoarthritis because it lies within the skills and competence of the generalist orthopaedic surgeon, has well-designed instrumentation, and the outcomes are reasonably predictable. The cost-effectiveness of UKA over TKA depends on the revision rates of the former, which tend to be higher than TKA [1]. UKA requires a different philosophical approach [2]. There are no soft tissue corrections permitted. Only the missing bearing surface is being replaced. The key is to restore the joint line accurately in all planes, which means matching the position of the meniscus. All the soft tissue ligaments are preserved, although Cartier allows the absence of an ACL when using a fixed-bearing implant [3, 4]. The underlying principle of UKA is that by restoring the native alignment of the knee, the remaining articular cartilage in the contralateral compartment of the knee becomes normally loaded and so stays healthy.

Medial UKA is classically indicated in the presence of three conditions:

- Advanced isolated pain at the medial knee joint space
- Marked isolated medial knee joint surface destruction, (bone-on-bone)
- Failure of conservative therapy

The classic definition of indications and contraindications for UKA was reported in 1989 by Kozinn and Scott [5]. Deschamps and Chol [6]

S. Lustig
Orthopaedic Department, Lyon North University Hospital, Lyon, France

S.T. Donell
Norwich Medical School, Faculty of Medicine and Health Sciences, University of East Anglia, Norwich, NR4 7TJ, UK

G. Pagenstert
University Clinics of Basel, Spitalstr. 21, Basel, 4031, Switzerland

P. Henle
Orthopädie Sonnenhof, Buchserstrasse 30, 3006, Bern, Switzerland

S. Oussedik
University College London Hospitals, 235 Euston Road, London NW1 2BU, UK

J. Beckmann (✉)
Sportklinik Stuttgart, Taubenheimstr. 8, 70372, Stuttgart, Deutschland, Germany
e-mail: drjbeckmann@gmx.de

F. Haddad
University College London Hospitals, 235 Euston Road, London NW1 2BU, UK

G.M.M.J. Kerkhoffs et al. (eds.), *ESSKA Instructional Course Lecture Book*,
https://doi.org/10.1007/978-3-662-56127-0_14

reported good and excellent results if UKA was indicated for:

- Isolated medial or lateral osteoarthrosis (OA) or osteonecrosis of the knee
- Age over 60 years
- Weight under 82 kg
- Lower leg deformity <15° of coronal knee deformity which needs to be correctable to neutral during surgery
- Extension loss <5°
- Total knee range of motion (ROM) at least 90°

Contraindications are high activity and systemic inflammatory joint disease.

14.2 Indications for UKA

14.2.1 Biological Factors

14.2.1.1 Age

Thompson et al. [7] analysed 229 UKAs performed at their institution for factors associated with poor outcome. They found that patients younger than 60 years did significantly better than older patients at 2 years follow-up (Knee score KSS 93 vs 77). Pennington et al. [8] reported a series of 46 consecutive UKAs under the age of 60 years at implantation; 93% of these UKAs had excellent results at mean of 11 years of follow-up. Other case series showed similar results [3, 9]. Thus, age under 60 years is no longer thought to be a contraindication for UKA. In a recent review about indications for UKA, Zuiderbaan et al. [10] proposed under age 40 years old as the new threshold.

14.2.1.2 Advanced Disease

- Rheumatoid arthritis (RA) is a systemic disease affecting all joints and therefore considered to be a contraindication for UKA. However, during the past two decades, the treatment of RA patients has changed considerably. The goal of therapy is no longer only symptom relief but rather the full prevention of structural joint damage and functional decline [11]. Today, no data exist in the literature reporting on the outcome of UKA in RA patients. However, further improvements in medical therapy may lead to an extended lifespan of these joints, eventually becoming candidates for partial knee arthroplasty. Today, progression of inflammatory joint disease is still believed to be less dependent on mechanical factors than on biological ones; therefore these patients are not suitable for UKA.
- Patellofemoral osteoarthritis (PFOA) visible on radiographs was thought to be a contraindication for UKA. Berger et al. [12] reported a series of 62 consecutive patients with medial UKA at minimum of 10 years of follow-up. Only two patients had to be revised but both for progressive PFOA. However, Berend et al. [13] reported on 638 consecutive knees with medial UKA and compared patients with no radiological evidence of PFOA with patients with such evidence. They found no patient was revised for advanced PFOA during follow-up. As well, they found no difference in outcome or revision rates for medial or lateral PFOA. In addition, Ma et al. [14] compared 100 consecutive medial UKAs with symptomatic anterior knee pain (AKP) in 43% of cases before surgery and looked for the location of PFOA. They found no significant difference between groups with or without preoperative AKP after a minimum of 50 months of follow-up. Patients with preoperative radiological medial PFOA had no significant difference in outcome to patients without PFOA. However, patients with lateral PFOA had a significantly poorer outcome compared to medial or no PFOA. A reason may be that UKA straightens the varus angle of the knee unloading the medial patellofemoral joint, whereas lateral PFOA is loaded more and may become more symptomatic. Thein et al. [15] studied the patellofemoral congruence angle before and after UKA. They found improved congruence after UKA suggesting that the medial PFOA is unloaded by the limb alignment correction. In conclusion, radiological PFOA with or without AKP is not a strong contraindication for UKA. However, severe degenerative PFOA with advanced patellar bone deformity and

destruction is still widely held to be a contraindication because these patients have not been included in the above-mentioned series.

- Lateral tibiofemoral TFOA visible on radiographs was thought to be a contraindication for medial UKA in varus OA. However, both UKA series of Berger et al. [12] and Pennington et al. [8] implanted UKA in the presence of asymptomatic lateral TFOA. They found little or slow progression of the disease over 10–15 years of follow-up without significant worsening of symptoms. Their patients had no progressive lateral joint pain and no revision for lateral TFOA. The key to this result may be the subtotal correction of the weight-bearing axis achieved. The mean femorotibial angle was 5°, which is a mild varus resulting in limited loading of the lateral TF joint space [8]. Marya and Thukral [16] took this concept to the limit by implanting a medial UKA in tricompartmental varus knee OA with symptoms confined to the medial tibiofemoral joint. In 45 low-demand older patients, medial UKA resulted in 96% survival and 95% good and excellent outcomes after 6 years mean follow-up. Therefore, candidates with isolated medial joint space symptoms for medial UKA that have visible early radiological signs of OA at the lateral tibiofemoral joint may still be treated successfully by isolated medial UKA.
- Minimal disease at the medial TF joint is a contraindication for medial UKA despite the presence of adequate symptoms. Niinimäki et al. [17], in their series of 113 consecutive medial UKAs, analysed the reoperation rate which was found to be independent of age, gender, obesity or arthroscopic degree of cartilage damage in the medial TF joint. However, if the medial knee compartment was thicker than 2 mm on standing anteroposterior radiographs, or more than 40% thickness of the unaffected lateral compartment, the reoperation rate was six times higher. Therefore, even in the presence of arthroscopically proven advanced cartilage loss at the medial compartment, if the medial joint space is radiologically intact, medial UKA must be avoided.

14.2.1.3 Physical Activity

Although Kozinn and Scott [5] recommended low activity and sedentary lifestyle in UKA, current practice changed. Pietschmann et al. [18] studied the preoperative activity level in relation to complications and outcome in their series of 131 consecutive patients. They found that higher preoperative activity was associated with higher postoperative activity with better overall outcome. In contrast, revision rates and complications were equal to the low-activity group after 4.2 years mean follow-up. Despite this encouraging comparative trials, van der List et al. [19] performed a recent large systematic review of 3967 UKA failures. They identified aseptic loosening and polyethylene wear as accounting for 50% of failure after UKA. Knowing that activity causes polyethylene wear and wear causes aseptic loosening, high-impact activity after UKA is still not recommended today.

14.2.1.4 Obesity

Obesity, defined as weighing more than 180 pounds (82 kg), was suggested as a contraindication for UKA in the past [5]. However, Cavaignac et al. [20] performed a retrospective study of 212 UKAs at mean follow-up of 12 years and found no significant influence of weight on revision rate or clinical outcome. Neither comparing patients up to 82 kg nor up to a BMI of 30 kg/m^2 revealed a significant difference. Thompson et al. [7] looked at even higher weight. They found that a BMI <35 kg/m^2 had no significant difference in revision rate but a significant better outcome score at 1 year follow-up. However, after 2 years of follow-up, this difference in outcome became insignificant, indicating slower recovery for patients with BMI >35. Therefore, currently there is no evidence-based threshold to deny UKA in obese patients.

14.2.2 Mechanical Factors

14.2.2.1 Anterior Cruciate Ligament Deficiency

Anterior cruciate ligament (ACL) deficiency in UKA has been reported to cause early failure and disappointing long-term results [21]. Therefore, in the absence of a functional ACL, combined procedures including UKA and ACL reconstruction have been conducted with success in 15 consecutive cases with excellent results [22]. However, recently, Boissonneault et al. [23] compared medial UKA in the ACL intact and not intact state and found no difference at mean follow-up of 5 years comparing 46 cases of each. Despite this, a recent systematic review looked at complications of UKA in ACL deficiency [24]. The revision rate of UKA in the presence of ACL deficiency was twice as high as in the group with UKA and combined ACL reconstruction. UKA with ACL reconstruction revealed lower outcome than UKA with intact ACL, but this difference was not significant. In conclusion, as for the native knee, if the patient has symptomatic knee instability, the ACL should be reconstructed, whereas the ACL rupture can be ignored in the medial arthritic knee with pain as the main complaint and no instability [25].

14.2.2.2 Mediolateral Subluxation

Mediolateral subluxation of the knee visible on weight-bearing radiographs has been described as a contraindication for medial UKA. This situation was defined either as advanced deformity or as mediolateral ligament insufficiency [6]. However, in a recent series reported by Khamaisy et al. [26] reporting on 174 medial UKAs, mediolateral subluxation could be reduced, and congruence of the lateral knee compartment was effectively restored by UKA. Thus, mild mediolateral ligament insufficiency caused by cartilage wear rather than by ligament insufficiency is not a contraindication for UKA.

14.2.2.3 Deformity and Restricted Range of Motion

Most authors agree that patients with knee varus deformity of more than 10–15° and restricted range of motion of less than 90° with combined extension—flexion or lack of extension of 5°—should not be treated by UKA [5, 19]. However, adequate studies comparing patient outcomes according to these preoperative variables are missing. Therefore, the influence of preoperative limb alignment may be questioned. However, the effect of postoperative limb alignment on UKA outcome has been studied in several case series and systematic reviews. Hernigou and Deschamps [27] analysed their series of 58 medial UKAs at 10–20 years follow-up and found overcorrection into valgus (hip-knee-ankle angle of more than 180°) associated with advanced degeneration of the uninvolved lateral knee compartment, and undercorrection with a hip-knee-ankle angle under 170° was associated with high wear. UKAs between 171° and 180° had better outcomes and lower revision rates. Vasso et al. analysed [28] their series and compared UKA alignment of mild varus (5–7°) with normal -2° to 1° and next to normal 2–4° of varus in 125 consecutive medial UKAs at mean 7.6 years. Mild varus limb alignment resulted in better outcome and no more complications than normal or next normal group. However, Zuiderbaan et al. [10], in their series of 104 consecutive medial UKAs, found better WOMAC scores in patients with a postoperative varus alignment of UKAs between 1° and 4° compared to UKAs with less than 1° or more than 4° of varus. In conclusion, over- and undercorrection with the UKA procedure should be avoided. Consequently, patients with medial compartment OA and valgus knee alignment are not ideal candidates for medial UKA.

14.2.3 Influence of Alternatives on UKA Indications

14.2.3.1 Total Knee Arthroplasty

Several advantages have been listed in case series and randomised controlled trials comparing outcome of UKA and TKA patients. Less perioperative morbidity, reduced blood loss, shorter postoperative rehabilitation, higher postoperative range of motion and reduced surgical costs favour UKA over TKA [29, 30]. In addition, patient-based outcome including the “forgotten knee joint score” is superior for UKA compared to TKA in limited medial knee OA [31]. However, in a recent meta-analysis, the revision rates of partial versus TKA have been compared. Medial UKA showed to have a 2.18-fold annual revision rate compared to TKA [32]. However, despite higher revision rates, UKA has shown to be more cost effective than TKA in a large study comparing 15.437 primary TKAs with 10.624 UKAs [33].

14.2.3.2 High Tibial Osteotomy

HTO was the established treatment for medial unicompartmental knee OA before UKA was available. The most obvious advantage of HTO is that it preserves the natural knee joint. Regarding the indications for UKA versus HTO, overlap exists in the current literature. Both surgical techniques have improved tremendously in accuracy, outcome and longevity. Some relatively clear cut-off variables can be defined in literature. Trieb et al. [34] compared HTO in 27 patients older than 65 years with 67 patients younger and found a 1.5-fold increased risk of failure at mean 13 years follow-up. In addition, the outcome was significantly worse in the older patients group. In two studies (BMI >27.5 kg/m^2 and >30 kg/m^2), HTO was associated with worse outcome and higher failure rate at 10–20 years follow-up after HTO [35, 36]. In conclusion, patients older than 65 years and obese should be candidates for UKA rather than HTO.

However, regarding deformity and ligament instability, in a recent systematic review, HTO showed superior survival comparing HTO and combined ACL reconstruction with UKA and combined ACL reconstruction [25]. In addition, HTO can stabilise a certain degree of medial collateral ligament insufficiency and can correct varus deformities above 15° or which cannot be corrected by manual reposition. Thus, HTO can be an option when UKA is contraindicated.

A recent meta-analysis of comparative trials between HTO and UKA by Fu et al. [37] comparing HTO and UKA for the treatment of unicompartmental knee OA found 8 studies including 461 patients. There was no overall difference in knee scores, but the postoperative functional subscore favoured UKA, but range of motion favoured HTO. However, HTO had a longer rehabilitation time with initially partial weight-bearing making UKA more attractive for the older and less active patients. Even though most studies showed a difference in complication rates between HTO and UKA, this meta-analysis including 4 studies with 301 patients found no significant difference.

14.2.3.3 Outcome of UKA and HTO Revision to TKA

Seven studies with 5641 patients compared the revision rates after HTO and UKA [37]. The reason for revision after HTO was mainly progressive OA. UKA was revised for loosening or breaking of the components, chronic pain and less frequently for progressive OA. After HTO, there was difficulty achieving correct tibial component position and adequate exposure of the knee. In UKA the most common difficulty was to manage bone defects in the tibia and femur. Compared to primary TKA or TKA after HTO, a significantly bigger polyethylene insert was needed after revision of UKA to TKA [38]. In another meta-analysis, Spahn et al. [39] compared time to TKA revision for patients with UKA and HTO. They found a significantly sooner time to revision of UKA at mean 8.2 years compared to 9.7 years for HTO. In contrast to HTO, the risk of revision of a UKA to TKA decreases with age. UKA patients under age 55 have a 3 times higher revision rate than above 55 years in the Swedish registry [40]. A possible reason was the higher activity and higher wear rate. It may also be that revising a UKA to a TKA is considered easier by general orthopaedic surgeons compared to TKA to TKA, and therefore a revision is more likely to be offered to a patient with an unsatisfactory UKA. Having said that the evidence suggests that in patients under 55 years old, one should favour treatment with an HTO rather than a UKA.

Summary of Indications

Medial UKA is indicated in symptomatic medial unicompartmental knee OA, with or without radiological signs of patellofemoral OA, in patients age over 55 years and with weight over 30 kg/m^2, a varus deformity no greater than 15° or loss of extension over 10°, no need for ACL or other ligament reconstructions and no interest in jumping and pivoting activities. Otherwise, HTO should be considered.

Medial UKA may be considered as well in patients with symptomatic medial knee OA and radiological evidence of OA at the patellofemoral or lateral compartment but without pain and limited bone destruction or deformity in the other compartments, plus a knee range of motion of more than 90°. Otherwise TKA should be considered.

In valgus knee alignment and medial knee OA, or limited medial knee OA with more than 2 mm preserved joint space, other treatment options besides UKA should be considered.

14.3 State-of-the-Art Treatment

Leaving aside the argument about the relevance of the state of the patellofemoral joint, UKA is suitable on radiological grounds when there is bone-on-bone arthritis in either the medial or lateral compartment. It should be noted that this is most likely to be demonstrated on the medial side with the knee flexed and weight-bearing at 30° flexion, whereas on the lateral side, the wear occurs on the posterior condyle. Therefore, lateral tibiofemoral wear may not be shown on an anteroposterior (AP) weight-bearing radiograph, since the knee would have to be flexed at 90°. Therefore, in lateral unicompartmental OA, the standing AP radiograph may appear normal. A provocation test with the knee in valgus flexed beyond 90° elicits pain and crepitus on the lateral tibiofemoral joint line confirms the diagnosis. In this instance, an MRI scan may be useful. It has been argued that the presence of bone marrow lesions aids the decision for UKA [40], but this has been disputed for medial compartment disease [41].

A minimal incision surgery (MIS) approach is justified in UKA and allows full visualisation of the compartment. Placing the incision correctly is important. If too close to the midline, it can be difficult exposing the tibia for its resection. Too far from the midline and the tibial sagittal cut may be impossible. Exposure can be improved by partially excising the infrapatellar fat pad and any patellar and notch osteophytes. The patella can have a sliver removed along its medial or lateral borders to help expose the femoral condyle and avoid excessive retraction. Removing the femoral-rim osteophytes improves exposure since it also relaxes the soft tissues. Evaluating the PFJ, ACL and lateral compartment provides reassurance, although should not lead to a change in the surgical plan as the preoperative workup should have excluded significant damage in the other compartments. Having said that, one should always have a TKA system available if there is a surprise, and a UKA is found to be contraindicated (Fig. 14.1).

Fig. 14.1 Mobile-bearing UKA

Table 14.1 NJR 10-year revision rates for total and unicompartmental knee arthroplasty

Implant	Revisions per 1000 patient-years	95% confidence interval
Total knee arthroplasty	3.86	3.80–3.93
Mobile-bearing UKA	13.40	12.96–13.85
Fixed-bearing UKA	12.10	11.43–12.81

As stated earlier, the mobile-bearing UKA has a number of theoretical advantages over the fixed-bearing design. These include better conformity through the flexion arc and therefore potential lower wear rates. The National Joint Registry of England and Wales (NJR) [42] shows that 70% of tibiofemoral UKA are mobile-bearing in the UK. Revision rates (all causes) are consistently greater than for TKA, and fixed-bearing designs have lower revision rates than mobile ones (Table 14.1.)

It should be noted that the confidence intervals between mobile- and fixed-bearing UKAs do not cross. Mobile-bearings have theoretical advantages with respect to wear that should occur between 10 and 20 years post-operation, and the

Fig. 14.2 Metal back fixed-bearing UKA

NJR only covers 10 years. The best performing implant in the NJR with 10 years follow-up is the fixed-bearing UKA (Fig. 14.2) with 6.31 revisions per 1000 patient-years (CI 95%, 5.16–7.70). The dominant UKA in the NJR (mobile-bearing UKA) has 12.02 revisions per 1000 patient-years (CI 95%, 11.51–12.54) reported. This may reflect the number of surgeons involved performing small numbers per year in the latter. The modes of failure appear similar, with the addition of bearing dislocation in mobile-bearing designs.

14.3.1 Technical Aspects of Medial Compartment Mobile-Bearing UKA

The degree of pre-existing laxity in the MCL should be evaluated to inform on the depth of tibial resection. The MCL should be normal, and therefore any laxity indicates the degree of articular cartilage and bone loss; the more lax the MCL, the less the resection.

After elevating the capsule from the most proximal part of the medial tibia, care must be taken to avoid damaging the deep MCL. Therefore, the soft tissue elevation medially should only be to the depth of the tibial resection. It must be remembered that the medial meniscus is attached to the MCL, so excision of the body of the meniscus should be undertaken with care, and a 1 mm rim should be left. Pulling hard on the anterior horn of the meniscus and then blindly sectioning the body with a scalpel can remove the deep MCL. If this happens, when the bearing size is assessed, it will be found to be much larger than expected. In this circumstance, either an MCL reconstruction will need to be performed or conversion to a constrained TKA. The MCL also has to be protected during the tibial bone resection. The posterior capsular attachment is the most difficult to elevate; the posteromedial corner can be mobilised safely with a small curved periosteal elevator. The tibial resection is critical. The alignment needs to be correct in the sagittal plane as well as the correct depth and slope. If the tibial resection is perfect, the rest of the operation is technically easy, including gap balancing and femoral component insertion. The tibial cut alignment can be improved through several key steps. The slope should be matched to the patient's own anatomy in both planes. The depth should be sufficient to allow a 7 mm feeler gauge (or the minimum depth to allow for the thickness of the tibial implant plus 1 mm laxity) to be inserted into the joint without gripping the gauge. The key is to get the new joint line back to the native joint line in all planes. Since the posterior femoral condyle has the full thickness of articular cartilage in medial compartment OA, then when the knee is flexed to 90°, this acts as the marker for the true joint line. Therefore, if the feeler gauge is at the right tension after the tibial resection, then one should be confident that the correct level has been achieved. This also means that achieving the correct posterior femoral cut is easy, since this is now just the thickness of the implant's posterior condyle, and is achieved using the relevant implant jig. The gap is now balanced in flexion. The MCL must be carefully protected during posterior femoral resection.

To achieve gap balance in extension, all tension must be removed from the soft tissues (i.e. the soft tissue retractors removed). The knee is then opened medially by a gentle valgus force to tension the MCL slightly. The extension gap can then be measured with feeler gauges where the tibial resection is the reference surface. The amount of distal femoral resection then allows for the thickness of the tibial implant and the distal femoral component thickness. Often this is 0 mm but more typically 2–3 mm. Femoral alignment may be improved with modern guides. Extramedullary rods avoid some of the pitfalls of incorrectly placed intramedullary rods. The pin guide must be flush against the condyle to provide accurate assessment of flexion-extension.

Uncemented implants may be used, but cemented implants are perhaps more forgiving. Tibial cement technique is critical and aims to produce 2–3 mm of bone penetration.

14.3.2 Technical Aspects of Medial Compartment Fixed-Bearing UKA

Fixed-bearing UKAs have a number of technical considerations to achieve a successful outcome [43]. Figure 14.3 shows an example which has an all-polyethylene tibial component. An all-polyethylene bearing allows for less bone resection and is easier to revise than a metal-backed one. Theoretically, the metal-back allows greater load transference and therefore may be more appropriate for younger, active patients.

Fig. 14.3 Full poly fixed-bearing UKA

The fixed-bearing surface needs to be flat to allow the curve of the femoral component to find its position on the insert after the wound has been closed; at this point the soft tissues will all be in a stable position and under their final tension. Any restraint caused by dishing the plastic risks overloading and early polyethylene wear. With a flat insert, the poly deforms by creep and so becomes dished and conforming without wear. It follows that it is important that the new implant is not inserted tight; slight laxity mimics the native knee.

Exposure is the same as for a mobile-bearing. Again, the key is to get the tibial cut right. Gap balancing is essentially the same. A key difference is that the femoral component needs to lie aligned with the tibial component in both flexion and extension. This means that the alignment on the femur does not match the femoral obliquity but is at right angles to the tibial alignment. If the tibial component has a varus slope, then the femoral component must match this (Fig. 14.4). It then follows that care must

Fig. 14.4 AP radiograph of a fixed-bearing UKA showing the varus slope of the tibial cut and the femoral component aligned at right angles

be taken that there is no overhang of the femoral component on the medial retinaculum at its most anterior point.

Finally the femoral component should be inserted flexed. This allows for greater knee flexion than if aligned with the femoral anatomical axis. With insertion with the knee at 90° flexion, impingement occurs between the posterior condyle of the femoral component and the posterior margin of the tibial component. Likewise, the posterior slope of the tibial component helps to avoid this conflict.

There is some evidence, including personal experience, that patients with fixed-bearing UKAs have less postoperative pain and more rapid recovery than mobile-bearings. This may possibly be due to soft tissue impingement by the mobile-bearing during knee motion. The 10-year follow-up data show no difference between the two types; however, the theoretical advantages of the mobile-bearing with respect to polyethylene wear are expected to be shown only after a longer time frame.

14.3.3 Technical Aspects of Lateral Compartment UKA

In the 1990s, lateral UKA was initially criticised by the proponents of the mobile-bearing UKA, mainly because it is difficult to balance the gaps as the lateral compartment is lax in flexion. Therefore, mobile-bearings tend to dislocate. Although the Oxford group have produced a domed-tibial mobile-bearing with some success in the designer hands, most surgeons favour a fixed-bearing design in the lateral compartment. Excellent long-term survival rates of 95–98% survival at 10-year follow-up have changed this perception [44, 45]. There are no differences between medial and lateral UKA when it comes to survival rates [46], if anything lateral fixed-bearing UKA is better as long as the different technical challenges are understood:

- In the MIS lateral parapatellar approach, removing the lateral patellar osteophytes along with a small partial lateral patellar facetectomy facilitates the exposure of the lateral tibiofemoral compartment and avoids the need for significant medial displacement of the patella. Like on the medial side, the tibial cut should reproduce the native slope. On the medial side, the tibia is dish-shaped but is domed on the lateral. It therefore does not have an obvious posterior slope. The rim should be exposed to the posterior edge. This is easier than medially as the LCL is extracapsular. The resection is usually less than the thickness of the tibial implant to allow for the lateral femoral hypoplasia that is present. The tibia typically has a neutral mechanical axis.
- The tibial sagittal cut should allow the tibial component to be positioned in internal rotation in order to compensate for the internal rotation of the external femoral condyle in extension (screw home mechanism). This may need to be achieved by creating the sagittal tibial cut with the saw blade passed through a separate stab incision through the patellar ligament. A malaligned tibial cut may induce impingement between the femoral component and the lateral tibial spine.
- The lateral femoral marginal osteophytes should be preserved in order to position the femoral implant condyle as lateral as possible. However, the notch osteophytes should be removed (to avoid continuing impingement on the ACL and the risk of later rupture).
- The posterior femoral condyle is worn, and so, if using standard medial UKA jigs for the posterior femoral cut, a suitable sized osteotome needs to be inserted between the bone and the jig to avoid an excessive flexion gap.
- Strict gap balancing is usually impossible; the lateral compartment opens up if the knee is placed in the Fig. 14.4 position. Resection of the distal femoral condyle needs to be minimal because of the hypoplasia. The tibial and femoral resections need to be such that on insertion of the implant, the knee valgus is less than normal. This reduces overload of the implant and early polyethylene wear [47].

Using these technical tips, the long-term results of the lateral fixed-bearing UKAs are extremely encouraging. The indications can now be extended to include young patients, the overweight and in some cases of posttraumatic OA, e.g. after fracture of the lateral tibial plateau [48].

14.3.4 Custom-Made UKA

Compared to TKA, MIS-UKA leads to faster recovery, lower complications, more “forgotten” joints and higher satisfaction but also, as shown above, has higher revision rates [49]. Reducing the number of revisions is an important goal considering the increasing need for artificial joints. Revision of a UKA in the first 3 years is usually due to surgical error; component malalignment and poor gap balancing being the commonest problems. These manifest as pain, stiffness and, in mobile-bearing designs, bearing dislocation. Poor tibial component fit can result in loosening and subsidence. Femoral component malposition can result in soft tissue impingement as well as poor gap balancing. It is therefore logical to consider whether this can be improved using patient-specific knee implants which are custom-made [50]. This is particularly apparent as the lateral compartment of the knee is biomechanically and anatomically completely different from the medial compartment. Most commercially available unicompartmental implants are not designed specifically for the lateral compartment. Patient-specific implants and the instruments needed for correct alignment and fitting are manufactured by virtual 3D reconstruction and 3D printing based on computed tomography (CT) scans. For the first time, implants are now matched to the individual knee and not vice versa. The aim is to achieve the best possible individual situation and geometry that includes coverage/fit, tibial slope and flexion gap balance.

However, this is currently in its infancy where the literature is sparse, and no long-term data are available.

14.3.5 Robotic Surgery

Many UKA instrument systems rely on manual placement of cutting blocks and extramedullary alignment rods. Open blocks use flexible saw blades; slotted blocks use rigid blades. One system places pins through the block, which is then removed, and the blade cuts on the pins. Accurate positioning is more difficult with MIS as less of the knee is visualised. Since the accuracy of the bone cuts is essential for a favourable outcome from UKA, it is logical to consider whether this would be improved by navigation aids. Computer-assisted navigation and tactile-robot assistance have been increasingly tried. Originally static referencing was used, which still had some implant placement variation between 1 and 2 mm and 2° and 5°, although overall alignment variance was less than 2° [51]. More recently, a dynamic referencing tactile-guidance robotic system has been trialled, which reduces set-up time and complexity [52]. These gave similar results. A retrospective comparative review of robot-assisted implantation versus standard techniques showed no difference in postoperative implant position or short-term outcomes [53]. Robot assistance added 20 min on average to the operation.

Robotic assistance is still experimental. The companies have not made a robot that is independent of the surgeon on the grounds that this would be unacceptable. The current systems are not suitable for normal clinical practice; cost-effectiveness as well as clinical effectiveness still needs to be confirmed, on top of surgeon acceptance. Experienced surgeons have similar outcomes with standard techniques.

14.4 Future Treatment Options

Although not new, there is a vogue for bicompartmental UKA in those patients with an intact ACL and bicompartmental (or even tricompartmental [54]) OA. The problem with bicompartmental tibiofemoral knee arthroplasty is that the medial and lateral tibial plateaux are not in the same orientation. Access is via a standard open access incision, unless the two sides are performed at different times. Navigation and robotics have a role. Cartier has been an enthusiast for bicompartmental UKA for many years [3].

Another extension is to combine an ACL reconstruction with a UKA in the younger patient. The problem is that to perform an excellent ACL reconstruction, the tibiofemoral joint needs to be intact, and to gap balance a UKA needs an intact ACL. Care needs to be taken to ensure that the ACL tibial tunnel does not pass through the UKA tibial cut; the tunnel is placed closer to the midline. Bioabsorbable screws are better than metal ones, as there is a risk of fretting corrosion against the tibial metal (and galvanic corrosion with dissimilar metals). One method is to make the ACL tunnels first arthroscopically. The posterior horn of the meniscus can also be removed. The ACL graft (ideally hamstrings) is then passed and anchored on the femoral end. The UKA is then cemented. Finally, the ACL graft is tensioned and fixed at the tibial end. Since ACL graft rupture is a risk, it is advisable to consider a fixed-bearing UKA. In addition, for medial UKA plus ACL reconstruction, lowering the tibial slope protects the graft. In fact, a medial tibial slope set at 0° using a fixed-bearing UKA without ACL reconstruction is another method for managing the ACL ruptured knee and unicompartmental disease [2]. A point to consider with lateral UKA plus ACL reconstruction is that the femoral tunnel can act as a stress riser; care must be taken not to fracture the lateral condyle, especially with a patellar retractor.

It should be emphasised that these treatments may fail, and the patient runs the risk of needing to undergo revision to a TKA. The objective is to keep the knee as mobile and functional as possible.

14.5 Take-Home Message

Unicompartmental knee replacement is a procedure for the dedicated knee specialist. To obtain good results, the surgeon and the surgical team need to be performing the operation routinely. Patient selection is the most important factor for a good outcome. As Cartier has stated that after a UKA, you will see “a forgotten knee” in an enthusiastic patient [4].

References

1. Chawla H, Ghomrawi HM, van der List JP, Eggman AA, Zuiderbaan HA, Pearle AD. Establishing age-specific cost-effective annual revision rates for unicompartmental knee Arthroplasty: a meta-analysis. J Arthroplast. 2017;32:326–35.
2. Donell ST. Unicompartmental knee arthroplasty. In: Bentley G, editor. European instructional lectures, vol. 10. Berlin: Springer; 2010. pp. 159–165.
3. Heyse TJ, Khefacha A, Peersman G, Cartier P. Survivorship of UKA in the middle-aged. Knee. 2012;19:585–91.
4. Cartier P. Story of my passion. Knee. 2014;21:349–50.
5. Kozinn SC, Scott R. Unicondylar knee arthroplasty. J Bone Joint Surg Am. 1989;71:145–50.
6. Deschamps G, Chol C. Fixed-bearing unicompartmental knee arthroplasty. Patients’ selection and operative technique. Orthop Traumatol Surg Res. 2011;97:648–61.
7. Thompson SA, Liabaud B, Nellans KW, Geller JA. Factors associated with poor outcomes following unicompartmental knee arthroplasty: redefining the “classic” indications for surgery. J Arthroplast. 2013;28:1561–4.
8. Pennington DW, Swienckowski JJ, Lutes WB, Drake GN. Unicompartmental knee arthroplasty in patients sixty years of age or younger. J Bone Joint Surg Am. 2003;85:1968–73.
9. Price AJ, Dodd CA, Svard UG, Murray DW. Oxford medial unicompartmental knee arthroplasty in patients younger and older than 60 years of age. J Bone Joint Surg Br. 2005;87:1488–92.
10. Zuiderbaan HA, van der List JP, Chawla H, Khamaisy S, Thein R, Pearle AD. Predictors of subjective outcome after medial unicompartmental knee arthroplasty. J Arthroplast. 2016;31:1453–8.
11. Goekoop-Ruiterman YP, de Vries-Bouwstra JK, Allaart CF, van Zeben D, Kerstens PJHazes JM, Zwinderman AH, Ronday HK, Han KH, Westedt ML, Gerards AH, van Groenendael JH, Lems WF, van Krugten MV, Breedveld FC, Dijkmans BA. Clinical and radiographic outcomes of four different treatment strategies in patients with early rheumatoid arthritis (the BeSt study): a randomized, controlled trial. Arthritis Rheum. 2005;52:3381–90.
12. Berger RA, Meneghini RM, Jacobs JJ, Sheinkop MB, Della Valle CJ, Rosenberg AG, Galante JO. Results of unicompartmental knee arthroplasty at a minimum of ten years of follow-up. J Bone Joint Surg Am. 2005;87:999–1006.
13. Berend KR, Lombardi AV Jr, Morris MJ, Hurst JM, Kavolus JJ. Does preoperative patellofemoral joint state affect medial unicompartmental arthroplasty survival? Orthopedics. 2011;34:e494–6.
14. Ma T, Cai MW, Xue HM, Liu XD, Tu YH. Preoperative assessment of the patellofemoral joint in

unicompartmental knee replacement using Oxford phase III and its influence on outcome. Zhonghua Wai Ke Za Zhi. 2013;51:1010–5.

15. Thein R, Zuiderbaan HA, Khamaisy S, Nawabi DH, Poultsides LA, Pearle AD. Medial unicondylar knee arthroplasty improves patellofemoral congruence: a possible mechanistic explanation for poor association between patellofemoral degeneration and clinical outcome. J Arthroplast. 2015;30:1917–22.
16. Marya SK, Thukral R. Outcome of unicompartmental knee arthroplasty in octogenarians with tricompartmental osteoarthritis: a longer followup of previously published report. Indian J Orthop. 2013;47:459–68.
17. Niinimäki TT, Murray DW, Partanen J, Pajala A, Leppilahti JI. Unicompartmental knee arthroplasties implanted for osteoarthritis with partial loss of joint space have high re-operation rates. Knee. 2011;18:432–5.
18. Pietschmann MF, Wohlleb L, Weber P, Schmidutz F, Ficklscherer A, Gulecyuz MF, Safi E, Niethammer TR, Jansson V, Muller PE. Sports activities after medial unicompartmental knee arthroplasty Oxford III-what can we expect? Int Orthop. 2013;37:31–7.
19. van der List JP, Zuiderbaan HA, Pearle AD. Why do medial unicompartmental knee arthroplasties fail today? J Arthroplast. 2016;31:1016–21.
20. Cavaignac E, Lafontan V, Reina N, Pailhe R, Wargny M, Laffosse JM, Chiron P. Obesity has no adverse effect on the outcome of unicompartmental knee replacement at a minimum follow-up of seven years. Bone Joint J. 2013;95:1064–8.
21. Goodfellow J, O’Connor J. The anterior cruciate ligament in knee arthroplasty. A risk-factor with unconstrained meniscal prostheses. Clin Orthop Relat Res. 1992;276:245–52.
22. Pandit H, Beard DJ, Jenkins C, Kimstra Y, Thomas NP, Dodd CA, Murray DW. Combined anterior cruciate reconstruction and Oxford unicompartmental knee arthroplasty. J Bone Joint Surg Br. 2006;88:887–92.
23. Boissonneault A, Pandit H, Pegg E, Jenkins C, Gill HS, Dodd CA, Gibbons CL, Murray DW. No difference in survivorship after unicompartmental knee arthroplasty with or without an intact anterior cruciate ligament. Knee Surg Sports Traumatol Arthrosc. 2013;21:2480–6.
24. Mancuso F, Dodd CA, Murray DW, Pandit H. Medial unicompartmental knee arthroplasty in the ACL-deficient knee. J Orthop Traumatol. 2016;17:267–75.
25. Mancuso F, Hamilton TW, Kumar V, Murray DW, Pandit H. Clinical outcome after UKA and HTO in ACL deficiency: a systematic review. Knee Surg Sports Traumatol Arthrosc. 2016;24:112–22.
26. Khamaisy S, Zuiderbaan HA, van der List JP, Nam D, Pearle AD. Medial unicompartmental knee arthroplasty improves congruence and restores joint space width of the lateral compartment. Knee. 2016;23:501–5.
27. Hernigou P, Deschamps G. Alignment influences wear in the knee after medial unicompartmental arthroplasty. Clin Orthop Relat Res. 2004;423:161–5.
28. Vasso M, Del Regno C, D’Amelio A, Viggiano D, Corona K, Schiavone Panni A. Minor varus alignment provides better results than neutral alignment in medial UKA. Knee. 2015;22:117–21.
29. Newman JH, Ackroyd CE, Shah NA. Unicompartmental or total knee replacement? Five-year results of a prospective, randomised trial of 102 osteoarthritic knees with unicompartmental arthritis. J Bone Joint Surg Br. 1998;80:862–5.
30. Noticewala MS, Geller JA, Lee JH, Macaulay W. Unicompartmental knee arthroplasty relieves pain and improves function more than total knee arthroplasty. J Arthroplast. 2012;27(8 Suppl):99–105.
31. Zuiderbaan HA, van der List JP, Khamaisy S, Nawabi DH, Thein R, Ishmael C, Paul S, Pearle AD. Unicompartmental knee arthroplasty versus total knee arthroplasty: which type of artificial joint do patients forget? Knee Surg Sports Traumatol Arthrosc. 2017;25:681–6.
32. Chawla H, van der List JP, Christ AB, Sobrero MR, Zuiderbaan HA, Pearle AD. Annual revision rates of partial versus total knee arthroplasty: a comparative meta-analysis. Knee. 2017;24:179–90.
33. Robertsson O, Borgquist L, Knutson K, Lewold S, Lidgren L. Use of unicompartmental instead of tricompartmental prostheses for unicompartmental arthrosis in the knee is a cost-effective alternative. 15,437 primary tricompartmental prostheses were compared with 10,624 primary medial or lateral unicompartmental prostheses. Acta Orthop Scand. 1999;70:170–5.
34. Trieb K, Grohs J, Hanslik-Schnabel B, Stulnig T, Panotopoulos J, Wanivenhaus A. Age predicts outcome of high-tibial osteotomy. Knee Surg Sports Traumatol Arthrosc. 2006;14:149–52.
35. Akizuki S, Shibakawa A, Takizawa T, Yamazaki I, Horiuchi H. The long-term outcome of high tibial osteotomy: a ten- to 20-year follow-up. J Bone Joint Surg Br. 2008;90:592–6.
36. Howells N, Salmon L, Waller A, Scanelli J, Pinczewski LA. The outcome at ten years of lateral closing-wedge high tibial osteotomy: determinants of survival and functional outcome. Bone Joint J. 2014;96:1491–7.
37. Fu D, Li G, Chen K, Zhao Y, Hua Y, Cai Z. Comparison of high tibial osteotomy and unicompartmental knee arthroplasty in the treatment of unicompartmental osteoarthritis: a meta-analysis. J Arthroplast. 2013;28:759–65.
38. Gill T, Schemitsch EH, Brick GW, Thornhill TS. Revision total knee arthroplasty after failed unicompartmental knee arthroplasty or high tibial osteotomy. Clin Orthop Relat Res. 1995;321:10–8.
39. Spahn G, Hofmann GO, von Engelhardt LV, Li M, Neubauer H, Klinger HM. The impact of a high tibial valgus osteotomy and unicondylar medial arthroplasty on the treatment for knee osteoarthritis: a meta-analysis. Knee Surg Sports Traumatol Arthrosc. 2013;21:96–112.
40. Park CN, Zuiderbaan HA, Chang A, Khamaisy S, Pearle AD, Ranawat AS. Role of magnetic resonance

imaging in the diagnosis of the painful unicompartmental knee arthroplasty. Knee. 2015;22:341–6.

41. Jacobs CA, Berend KR, Lombardi AV, Christensen CP. The location and severity of preoperative subchondral bone marrow lesions were not associated with inferior postoperative outcomes after medial unicompartmental knee arthroplasty or total knee arthroplasty. J Arthroplast. 2016;31:2476–80.
42. 13th Annual Report of the National Joint Registry for England. Wales: Northern Ireland and the Isle of Man. 2016.
43. Deschamps G, Bussière C, Donell S. Fixed bearing unicompartmental knee prosthesis: results, complications, and technical considerations. In: Bonnin M, Amendola NA, Bellemans J, et al., editors. The knee joint: surgical techniques and strategies. Paris: Springer Verlag; 2012, pp. 669–684.
44. Lustig S, Elguindy A, Servien E, Fary C, Munini E, Demey G, Neyret P. 5- to 16-year follow-up of 54 consecutive lateral unicondylar knee arthroplasties with a fixed-all polyethylene bearing. J Arthroplast. 2011;26:1318–25.
45. Argenson JN, Parratte S, Bertani A, Flecher X, Aubaniac JM. Long-term results with a lateral unicondylar replacement. Clin Orthop Relat Res. 2008;466:2686–93.
46. van der List JP, McDonald LS, Pearle AD. Systematic review of medial versus lateral survivorship in unicompartmental knee arthroplasty. Knee. 2015;22:454–60.
47. Lustig S, Lording T, Frank F, Debette C, Servien E, Neyret P. Progression of medial osteoarthritis and long term results of lateral unicompartmental arthroplasty: 10 to 18 year follow-up of 54 consecutive implants. Knee. 2014;21(Suppl 1):S26–32.
48. Lustig S, Parratte S, Magnussen RA, Argenson JN, Neyret P. Lateral unicompartmental knee arthroplasty relieves pain and improves function in posttraumatic osteoarthritis. Clin Orthop Relat Res. 2012;470:69–76.
49. Liddle AD, Judge A, Pandit H, Murray DW. Adverse outcomes after total and unicompartmental knee replacement in 101 330 matched patients: a study of data from the National Joint Registry for England and Wales. Lancet. 2014;384:1437–45.
50. Beckmann J, Steinert A, Zilkens C, Zeh A, Schnurr C, Schmitt-Sody M. Patientenspezifische Instrumente und Implantate beim teilgelenkersatz des kniegelenkes (ConforMIS iUni, iDuo). Orthopade. 2016;45:322–30.
51. Cobb J, Henckel J, Gomes P, Harris S, Jakopec M, Rodriguez F, Barrett A, Davies B. Hands-on robotic unicompartmental knee replacement: a prospective, randomised controlled study of the acrobot system. J Bone Joint Surg Br. 2006;88:188–97.
52. Dunbar NJ, Roche MW, Park BH, Branch SH, Conditt MA, Banks SA. Accuracy of dynamic tactile-guided unicompartmental knee arthroplasty. J Arthroplast. 2012;27:803–8.
53. Hansen DC, Kusuma SK, Palmer RM, Harris KB. Robotic guidance does not improve component position or short-term outcome in medial unicompartmental knee arthroplasty. J Arthroplast. 2014;29:1784–9.
54. Thienpont E, Cartier P. Tricompartmental resurfacing arthroplasty with a follow-up of more than 30 years. Knee. 2014;21:875–7.

Sports Injuries in Throwing Athletes

15

Oskar Zupanc, Nicolas Holzer, Claudio Rosso,
Nick F.J. Hilgersom, Luke S. Oh, Andreas Lenich,
Boris Hollinger, Meglič Uroš, Denise Eygendaal,
Raul Barco, Paolo Arrigoni, Marco Brioschi,
Pietro Randelli, Davide Cucchi,
and Hakan Turan Cift

15.1 Introduction

Upper extremity injuries are a common phenomenon in throwing athletes, as the throwing motion repetitively exposes the shoulder and elbow to extreme forces at extreme ranges of motion. Overuse injuries of the elbow and shoulder comprise 14.3% of all overuse injuries in high school athletes in the United States. This percentage increases among throwing sports

---

O. Zupanc (✉) • M. Uroš
Department of Orthopedic Surgery, University
Medical Centre Ljubljana, Zaloška 9,
1000 Ljubljana, Slovenia
e-mail: oskarzupanc@gmail.com;
uros.meglic@kclj.si

N. Holzer
Départment de Chirurgie, Hôpitaux Universitaires de
Genève, Genève, Switzerland
e-mail: nicolas.holzer@hcuge.ch

C. Rosso
ARTHRO Medics, Shoulder and Elbow Center,
Basel, Switzerland

University of Basel, Basel, Switzerland
e-mail: c.rosso@arthro.ch

N.F.J. Hilgersom
Department of Orthopaedic Surgery, Sports Medicine
Service, Massachusetts General Hospital, Harvard
University, Boston, USA

Department of Orthopaedic Surgery, Academic
Medical Centre, University of Amsterdam,
Amsterdam, The Netherlands
e-mail: nhilgersom@mgh.harvard.edu

L.S. Oh
Department of Orthopaedic Surgery, Sports Medicine
Service, Massachusetts General Hospital, Harvard
University, Boston, USA
e-mail: loh@mgh.harvard.edu

A. Lenich
Klinik fur Orthopädie, Unfall, Handchirurgie und
Sportorthopädie, HELIOS Klinikum München West,
Munich, Germany
e-mail: a.lenich@tum.de

B. Hollinger
ARCUS Sportklinik, Pforzheim, Germany
e-mail: b.k.hollinger@t-online.de;
hollinger@sportklinik.de

D. Eygendaal
Amphia Hospital Breda and University of
Amsterdam, Breda, The Netherlands
e-mail: deygendaal@amphia.ni

R. Barco
Upper Limb Unit, Hospital Universitario La Paz,
Madrid, Spain
e-mail: raulbarco@homtail.com

P. Arrigoni • M. Brioschi • P. Randelli • D. Cucchi
Department, Azienda Socio Sanitaria Territoriale,
Centro Specialistico Ortopedico Traumatologico,
Gaetano Pini-CTO, Milan, Italy

Department of Biomedical Sciences for Health,
University of Milan, Milan, Italy
e-mail: arrigoni.p@gmail.com

H.T. Cift
Central Hospital Kozyatagi, Mahallesi, Kocayol Cd.
No:19, 34742 Kadikoy/Istanbul, Turkey
e-mail: hakanturancift@yahoo.com

G.M.M.J. Kerkhoffs et al. (eds.), *ESSKA Instructional Course Lecture Book*,
https://doi.org/10.1007/978-3-662-56127-0_15

(i.e., softball, volleyball, baseball), up to 68% in male high school baseball players [1]. Among professional baseball players, the incidence of wrist, elbow, and shoulder injuries is 10%, 16.4%, and 21.2%, respectively [2]. Elbow and shoulder injuries are the two leading causes of time-out of play in professional baseball players [3]. Despite baseball-targeted prevention programs focused on the entire kinetic chain, the incidence of upper extremity injuries has remained the same in Major League Baseball from 1998 to 2015. Among the upper extremity injuries, a declining trend in shoulder injuries was observed but counteracted by an increase in elbow injuries [3].

The throwing motion is one of the most complex and rapid human movements. The entire kinetic chain from the feet up through the hips via the trunk and finally the upper extremity has to work in perfect harmony to generate and effectively dissipate the tremendous forces. During the acceleration phase, the elbow extends with over 2300°/s, and the shoulder internally rotates at over 6940°/s. Meanwhile the shoulder and elbow are exposed to excessive forces at extreme ranges of motion. Two critical moments in the throwing motion have been identified. The first is during late cocking and early acceleration, the second just after ball release. During these moments, the elbow is exposed to 64 Nm of valgus stress, 300 N of medial shear forces, and 900 N of lateral compressive force. The shoulder is exposed to a multitude of different directional forces, of which the most relevant are 67 Nm of internal rotation torque and 380 N of anterior shear force during late cocking and 1090 N of compressive forces along with 400 N of posterior shear force and 310 N of inferior shear force just after ball release. These forces place tensile stress along the medial side of the elbow and the posterior shoulder muscles, compressive forces on the radiocapitellar joint and subacromially, and shearing forces throughout the ulnohumeral and glenohumeral joint [4].

The proximal kinetic chain, consisting of the lower extremities and trunk, plays a significant role in generating the abovementioned forces and transferring them to the upper extremity during earlier phases of the throwing motion, the windup and stride phase, and dissipating these forces after ball release. Therefore, changes in biomechanical properties of the proximal kinetic chain, such as range of motion or timing sequence, are likely to alter the magnitude and distribution of forces throughout the upper extremity. In literature, an altered knee flexion at ball release and early trunk rotation have been proven to increase shoulder and elbow torques and thus risk for upper extremity injury [5].

The often numerous repetitions of the throwing motion may induce adaptive changes and possibly overload or microtraumata to the bony, muscular, and tendinous structures of the wrist (Paulo Arrigoni), elbow, and shoulder. Eventually this leads to specific adaptations and injuries such as ulnar collateral ligament insufficiency or rupture, valgus extension overload (Uroš Meglič, Oskar Zupanc, Andreas Lenich), lateral-sided elbow pain (Denise Eygendaal) such as osteochondritis dissecans or/and osteochondral lesions of the capitellum, glenohumeral internal rotation deficit (Boris Hollinger), and superior labrum anterior to superior tears. In the following sections, different parts of the spectrum of upper extremity injuries in the throwing athlete will be discussed into further detail, along with state-of-the-art treatments, rehabilitation (Hakan Turan), and future perspectives.

15.2 GIRD Phenomena in Throwing Athletes and the Impact on Elbow

The glenohumeral internal rotation deficit (GIRD) is a reduction in the internal rotation angle of the throwing shoulder in comparison to the non-throwing shoulder and is considered a primary factor in the development of shoulder injuries [6].

The set of adaptations that occur in the throwing shoulder is one of the reasons for shoulder pain in overhead atheletes [7]. Recent studies have contributed further information regarding the association of these adaptations and the injury mechanism in throwing athletes. It is currently believed that contracture of the posterior capsule and posterior band of the inferior glenohumeral ligament, caused by repetitive microtrauma during the deceleration phase of throwing, is the main cause of GIRD and, subsequently, future injuries.

Several authors [8–10] have stated that GIRD increases the load on medial compartment of the elbow and thus the ulnar collateral ligament, leading to potential subtle valgus instability of the elbow. This instability will eventually lead to injuries encountered in the throwing elbow, most commonly valgus extension overload syndrome (VEO) or ulnar collateral ligament injury (UCL) or less commonly ulnar neuritis, flexor-pronator injury, and medial epicondyle apophysitis or avulsion.

15.2.1 Literature Overview

Dines et al. [8] conducted a retrospective case-control study comparing range of motion (ROM) between baseball players in the clinic demonstrating UCL insufficiency with a healthy control group. The group with UCL insufficiency had less dominant-arm internal rotation and greater GIRD.

Garrison et al. [9] conducted a prospective case-control study of baseball players comparing patients in their clinic diagnosed with UCL tears with a control group of healthy players. The group of players with UCL tears exhibited less dominant-arm external rotation, as well as deficits in shoulder total rotation.

Wilk et al. [11] introduced the total rotation concept, where the amounts of external rotation and internal rotation at 90° of abduction are added together, and a total rotational motion arc (TROM) is determined [11]. They stated that a pitcher with more than a 5° difference in total rotation between his two shoulders had increased risk for shoulder injury [12]. In a later study, they were able to establish a correlation between total rotation deficit and elbow injuries. In a prospective study, they measured passive ROM of both throwing and non-throwing shoulder in baseball pitchers. They revealed that a pitcher with a deficit in throwing-shoulder TROM had an increased risk for elbow injury, and a pitcher with throwing-shoulder GIRD did not have an increased risk for elbow injury [10].

15.2.2 Take-Home Message

According to latest studies, a deficit in shoulder total rotational motion (TROM) greater than 5° and flexion has a significant effect on the risk for elbow injuries in a throwing athlete. However, in the overhead athlete, loss of glenohumeral rotation, termed glenohumeral internal rotation deficit (GIRD), is a normal phenomenon that should be expected. Pathologic GIRD is when there is a loss of glenohumeral internal rotation greater than 20° with a corresponding loss of TROM greater than 5° when compared bilaterally. Clinicians need to be aware of these problems and plan specific preventive rehabilitation programs that address these issues in hopes of reducing elbow injuries. They include exercises for the entire kinematic chain (trunk stabilization, glenohumeral, rotator cuff, and total arm strengthening), active and passive warm-up on an upper body ergometer, and active warm-up, manual therapy, and resisted and dynamic exercises for glenohumeral and scapulothoracic stability, stretching exercises, cryotherapy, and home exercises program.

15.3 Valgus Extension Overload of the Elbow

Valgus extension overload (VEO) among throwing athletes is a syndrome of symptoms and physical findings of the elbow that are commonly seen due to numerous repetitions of the throwing motion [13]. To understand the injury patterns and their treatment options, as well as the possible prevention of the injuries, clinicians must have a working knowledge of the elbow anatomy, biomechanics, and pathomechanics that lead to those common injuries.

15.3.1 Pathomechanics

Multiple biomechanical studies in throwing athletes have shown that during throwing motion the elbow is exposed to valgus stress, medial shear force, and lateral compressive force, and a spectrum of elbow lesions was encountered [14]. These extraordinary forces on the elbow joint leave the elbow especially vulnerable to injury [4]. The typical pattern of injury sustained is either due to repetitive microtrauma or chronic stress overload leading to injury and inflammation to the surrounding soft tissue structures of the elbow. However, ulnar collateral ligament (UCL) microtrauma may occur, leading to subtle valgus instability. This instability will lead to excessive force being transmitted to the lateral and posterior elbow compartments that are most significant in the late cocking and follow-through phases, as the elbow comes into extension. With the continuance of throwing in the setting of subtle instability, shear forces due to a combination of compressive and rotatory forces gradually increase, leading to synovitis and osteophyte formation. Osteophyte formation is hastened as abutment of the olecranon with the olecranon fossa that occurs as the elbow extends. This impingement can lead to a "kissing lesion" of chondromalacia in the olecranon fossa and loose body formation [15].

15.3.2 Examination

In the evaluation of athletes with elbow VEO syndrome, a thorough history typically involves a complaint of posterior or posteromedial pain during the follow-through phase of throwing. It is during this final phase of throwing that the elbow extends and the posterior osteophytes impinge. Pain that occurs earlier in the throwing cycle should raise suspicion for other pathologies such as UCL injury. If loose bodies are present, the athlete may also report mechanical symptoms such as locking or catching. Physical examination should focus on the evaluation of range of motion, especially as forced terminal extension usually leads to pain. Additionally, a "valgus extension overload test" elicits pain [16].

Imaging of athletes with elbow complaints should always initially include plain radiographs with anteroposterior, lateral, axial, and two oblique views of the affected side. An oblique axial view with the elbow in 110° of flexion is helpful to demonstrate posteromedial olecranon osteophytes. Comparison views of the opposite elbow may be done if necessary. If medial instability is suspected, stress AP radiographs can be performed [14].

Magnetic resonance imaging (MRI) is considered the gold standard imaging modality for the athlete's elbow. In cases of VEO, it identifies distinct bony and articular changes to the posterior trochlea and olecranon, along with posteromedial gutter synovitis. MRI findings of insertional tendinosis at the medial border of the triceps, sometimes with bone marrow edema in the olecranon, are common and may serve as an imaging clue to the diagnosis. Other associated findings may include loose bodies, as well as chronic changes to the UCL [17].

A CT scan can be helpful for detecting stress fractures of the olecranon [14].

15.3.3 Treatment

Nonoperative treatment of VEO syndrome consists of an initial period of active rest, icing, and anti-inflammatories. Once the initial pain resolves, rehabilitation including both shoulder and elbow exercises is initiated, with avoidance of throwing. As range of motion and strength improves, strengthening of the flexor-pronator musculature and a progressive supervised throwing program can be initiated.

The surgical procedure of choice in the throwing athlete that fails conservative treatment is osteophyte excision and exploration for loose bodies. Although this procedure was originally described as an open procedure [18], the current trend is for arthroscopic intervention. Elbow arthroscopy allows visualization of all compartments as well as arthroscopic evaluation of the UCL. With arthroscopy, osteophytes can be easily visualized and debrided in cases of posterior, posterolateral, and anterior elbow impingement. Also loose body excision, debridement of hypertrophic scar tissue, and synovium could be adequately performed. Osteochondral lesions especially of capitellum humeri may be treated with arthroscopic debridement or drilling.

15.3.4 Take-Home Message

Because of recent advances in arthroscopic surgical techniques, the prognosis for return to competition for this highly motivated patient population is generally good. However, continued exposure to high forces often results in symptom recurrence in the competitive thrower.

15.4 Lateral Elbow Pain in Athletes

Lateral elbow pain is the most common diagnosis in athletes presenting with elbow pain. Initially, it is important to differentiate between intra- or extra-articular causes. Intra-articular causes include osteochondral pathology of the radiocapitellar joint such as Panner's disease and osteochondral lesions (OCD), osteochondral fractures, and osteoarthritic spurs but also causes such as thickened, symptomatic synovial fold or diffuse synovitis. Pain from intra-articular causes will generally be felt at the radiocapitellar joint or just posterior to the epicondyle (posterolateral compartment of the joint). It can have a compromising effect on range of motion, which is rarely seen in extra-articular causes of lateral elbow pain. In these cases, the limited range of motion can sometimes be the result of an effusion with swelling, most notably at the "soft spot" on the posterolateral side of the elbow. Intra-articular causes are suggested by painful clicking during passive and active motions of the elbow. It should be noted that (extra-articular) ligamentous insufficiency should also be considered as a potential cause for these intra-articular symptoms. In young, overhead athletes, an OCD is often overlooked; each overhand sporting kid has an OCD until proven otherwise. Extra-articular causes include lateral epicondylitis (sometimes accompanied with an intra-articular component, i.e., a synovial fold), lateral collateral ligament complex injuries, radial tunnel syndrome, cervical root compression syndromes, and other causes of referred pain.

15.4.1 Lateral Epicondylitis

Lateral epicondylitis (LE) is the most common cause of lateral elbow pain, and it is typically diagnosed in people aged 35–50 [19]. The prevalence of LE in the general population is 1.3% [20]. While LE is called tennis elbow, tennis players only make up a mere 10% of all cases [21, 22]. On the other hand, 50% of the tennis players develop elbow pain, in which LE is the cause in 75% [22].

15.4.1.1 Pathophysiology and Anatomy

The symptoms in LE are the result of repetitive stress on the proximal origin of the tendons of the forearm extensor muscles, causing multiple microtraumata. These traumata lead to disruption of the internal structure of the tendon and cell matrix degeneration, which in turn causes tendinosis. Tendinosis, although not completely understood, is a process of degeneration without the presence of multiple macrophages, lymphocytes, and neutrophils [23]. At histological examination, a vast amount of fibroblasts, vascular hyperplasia, and disorganized collagen are found [24]. This makes epicondylitis a rather wrong term: epicondylosis would be more correct.

The origin of the extensor carpi radialis brevis (ECRB) muscle is usually the focal point of these symptoms [25]. Its tendon wraps around a convex surface—the lateral epicondyle—and is, especially in sports or work-related activity, exposed to repetitive tension. Another muscle regularly involved in LE is the extensor digitorum communis (EDC). The pathological changes at the ECRB origin are consistently present, while involvement of the EDC muscle is reported in one third of the cases [24]. In 5% of patients suffering from LE, there is concomitant radial nerve pathology, i.e., radial tunnel syndrome [26]. Another consideration is the potential presence of a synovial plica, folds in the synovial tissue, and remnants of the embryonic septa in normal articular development [27]. Injury to the synovium by direct blow, or chronic overloading such as repetitive extension (same cause for tennis elbow), can cause thickening of the plicae followed by impingement between the articular surfaces [27]. It is presumably due to this similar trauma mechanism that the synovial plica is a frequently found concomitant pathology in patients with LE. Characteristic findings are a click or snap with terminal elbow extension and forearm supination. However, some plicae can cause lateral elbow pain without the clicking or snapping symptoms which can make discrimination between a symptomatic plica and LE difficult.

15.4.1.2 Management

LE does not always demand active treatment, since it usually is a self-limiting disease. In 90% of the cases, symptoms will resolve after conservative treatment such as lowering or changing activity, physiotherapy, splints, or using local nonsteroidal anti-inflammatory drugs (NSAIDs). Local infiltration therapy is also widely employed. Injection therapy should be performed under ultrasound guidance in a standardized way. Corticosteroid injections should not be used anymore with the increasing amount of evidence of its harmful long-term effects. Other injectables such as dextrose, autologous blood, platelet-rich plasma, or dry-needling techniques are still being investigated [28].

Patients not responding to conservative treatment can be considered candidates for surgery. It is the surgeon's preference to perform *surgery in an open or arthroscopic way*. An advantage of an arthroscopic technique is complete visualization of the joint to treat concomitant intra-articular pathology such as a radiocapitellar plica, which can be treated in the same procedure if necessary. There is also a decreased chance of damaging the lateral collateral ligament complex in comparison to open surgery. It also shows an earlier return to activity [29].

Dunkow et al. [30] described a technique where a *percutaneous release* is done through a small 1 cm incision over the midpoint of the lateral epicondyle, lifting the common extensor origin, before releasing it. In their study containing 89 patients, they found significant improvements in return to work, DASH score, and sporting activities. It was compared to Nirschl's technique at 12 months follow-up. There is insufficient evidence to support the use of one operative procedure over another. This is mainly due to methodological limitations in the current studies, including high risk of bias in the available studies and small sample sizes.

15.4.2 Radial Tunnel Syndrome

Radial tunnel syndrome (RTS) is presumably caused by compression of the posterior interosseous nerve (PIN). It causes pain on the lateral side of the elbow and dorsal forearm. Pain may radiate proximately and distally. Usually, compression is mild, and no motor symptoms are present, although more severe compression (e.g., caused by lipoma, ganglion, or synovitis) can lead to motor weakness [24]. The incidence rate of RTS has been estimated as low as 0.03%, and it is mostly diagnosed in women aged 30–50 [25]. Other reports noted a coexisting RTS in up to 5% of the patients with LE [8].

15.4.2.1 Pathophysiology and Anatomy

The radial nerve originates from the brachial plexus, runs through the upper arm, passes the elbow, and enters the radial tunnel. The radial tunnel is approximately 5 cm long and extends from the radial head to the distal border of the supinator muscle. Boundaries of the radial tunnel are the brachioradialis, supinator, extensor carpi radialis longus, and ECRB [31]. The radial nerve splits into the PIN and a superficial branch. The PIN passes the leading aponeurotic edge of the supinator muscle, also called the arcade of Frohse, which is the most common site of compression [32]. Other potentially compressing structures are the sharp medial edge of the ECRB muscle, the radial recurrent blood vessels (leash of Henry), and the distal margin of the superficial layer of the supinator muscle [32].

In general, compression of the nerve causes the capillary bed in the nerve to be compromised. Hypoxia of the nerve trunk leads to dilation of the small vessels causing endoneurial edema. Edema, in turn, can increase the effect of the original compression [33].

The PIN, being purely a motor nerve, makes the cause of the pain a point of discussion. The usual absence of motor symptoms (in a compression syndrome of a motor nerve) and the fact that EMG and NVC studies are generally negative make the diagnosis somewhat controversial [31]. The PIN, however, does carry group IV unmyelinated fibers that have been associated with nociception which may explain the pain, and these fibers are not generally assessed with EMG [31].

15.4.2.2 Diagnosis

Patients present with pain at the lateral elbow and the dorsoradial aspect of the forearm which can radiate proximally and distally. The arcade of Frohse should also be palpated to localize a second focal point of tenderness. Pain could be aggravated by extending the elbow, pronating the forearm, or flexing the wrist. Resisted supination and hyperextension of the wrist against resistance are helpful diagnostic tests. Comparison with the opposite arm is necessary to decrease the chances of a false-positive test result. These tests, however, are very likely to be positive in LE as well. As noted earlier, EMG and NCV tests are typically normal. MRI can show muscle edema as a possible cause for compression or atrophy along the distribution of the radial nerve but is usually negative as well [31]. Injection of a local anesthetic can be helpful in establishing the diagnosis, when both a temporary paralysis of the PIN and pain relief are established. Relieve of the symptoms localized in the forearm, accompanied by a transient inability to extent the MCP joints, supports the diagnosis of a RTS. It should be noted that pain at the ECRB origin is not expected to diminish by this infiltration: infiltrating too proximally may lead to a temporary paralysis of the ECRB, giving a false-positive result. Asking for specifics when the patient reports a relieve of symptoms can clear this up in most cases. In addition, the use of ultrasound-guided infiltration could be considered to target the arcade of Frohse more accurately.

15.4.2.3 Management

Initial therapy is conservative. Modalities include splinting, using NSAIDs, physical therapy, and avoiding provocative maneuvers. Corticosteroid injections are used frequently. In a study consisting of 25 patients, 72% had pain relief of which 62% continued pain free for 2 years [34]. No clinical trials assessing the success rate and ideal duration of conservative therapy are available.

Surgical treatment for RTS consists mostly of decompression of the PIN and sometimes the superficial branch of the radial nerve as well. It is the key to release the nerve at the arcade of Frohse and negate other potentially compressive agents such as the radial recurrent blood vessels as the release is carried more distally [31]. Various approaches have been described: the dorsal approach between the wrist extensors and finger extensors, or between the brachioradialis and the wrist extensors, the anterior approach between the brachioradialis and biceps, and the transmuscular brachioradialis-splitting approach.

No clinical trials comparing various surgical approaches are available [35]. The overall success rate of RTS surgery ranges from 67% to 92% [36].

15.4.3 Lateral Collateral Ligament Injuries

The lateral collateral ligament (LCL) complex consists of four structures: the annular ligament (AL), the radial collateral ligament

(RCL), the lateral ulnar collateral ligament (LUCL), and the accessory lateral cross ligament. The RCL passes from the lateral epicondyle into the annular ligament; the AL originates and inserts at the anterior and posterior margins of the lesser sigmoid notch and encircles the radial head. The LUCL runs from the lateral epicondyle to the supinator crest of the ulna. An injury to the LCL complex can be caused by mechanical or iatrogenic trauma: *elbow dislocations* in various forms will predictably lead to a torn LUCL. It is common belief that simple elbow dislocations start with injury to the lateral complex before advancing through the anterior and posterior capsule and finally tearing the medial collateral ligament. In terrible triad injuries, the LUCL is also torn, but in addition a coronoid fracture and radial head fracture are present. *Iatrogenic causes* include damage during surgery for radial head fractures and surgery for LE. In a cohort of 13 patients, 3 out of the 13 patients showed evidence of laxity following surgical treatment for LE, indicating injury to the lateral collateral ligament complex [37]. LCL complex injuries can also result in posterolateral rotatory instability; the LUCL is considered to play a key role in the resulting instability pattern [38].

15.4.3.1 Diagnosis

Patients with an LCL injury can present with a variety of complaints such as lateral elbow pain, locking, clicking, or snapping. Symptoms can usually be provoked by activities with the forearm in supination, the elbow in extension, and valgus stress. Patients mostly present with a history of trauma including a fall on a fully extended arm or surgery on the lateral side of the elbow.

Varus stress itself is not a good test for lateral ligament injuries because it does not reproduce the forces on the lateral aspect of the elbow that are symptomatic in lateral ligament pathology. A good test would be the posterolateral rotatory pivot-shift test (mostly under anesthesia). Other tests are the posterolateral rotatory drawer test, tabletop relocation test, and active floor push-up sign. Radiographs can be made to assess the possibility of degenerative joint disease and previous trauma. MRI can show the location of ligament tears. However, it is not very specific: a negative MRI does not exclude instability.

15.4.3.2 Management

LCL complex injuries can be treated conservatively using activity modification and splinting or surgically. The most frequent indication for conservative treatment of an LCL injury is the simple elbow dislocation. When treating this injury surgically, it is either by direct repair (for the acute cases) or reconstruction (for the longer-standing cases).

In longer-standing cases, a repair is usually not feasible, and a reconstructive procedure is undertaken. There is no evidence for superiority of one reconstruction technique over the other due to a lack of comparative studies [39].

15.4.4 Radiculopathy

Radiculopathy that occurs at the C6 or C7 levels may cause referred pain to the lateral elbow area. It can also cause weakness and dysfunction of the biceps, triceps, wrist, and fingers. Most likely, pain cannot be increased by provocative tests stressing the muscles around the lateral epicondyle as in epicondylitis, and no other abnormalities will be found around the elbow during physical examination. Lee and Lee-Robinson [39] suggested that there is a correlation between existing radiculopathy of C6–C7 and the incidence of LE, most likely due to muscle weaknesses and imbalances caused by the radiculopathy. A correlation with C6–C7 radiculopathy is also present in medial epicondylitis [39].

15.5 Partial Tears of the Distal Biceps

Partial tears are rare injuries, occurring mostly in middle-aged men. While most of the pathology of the distal biceps is related to complete ruptures, partial tears or bursitis at the insertion site may present with mild pain in the antecubital fossa, so patient's diagnosis may be delayed. A high index of suspicion is needed in order to perform a timely diagnosis.

15.5.1 Clinical Findings

Patients presenting with pain at the antecubital fossa typically present with biceps tendinopathy. Biceps tendinopathy, including partial ruptures, may have a traumatic event or a more insidious onset indicating a degenerative disease of the tendon. As in other chronic tendinopathies, the traumatic event may be minor, so it is important to review possible events with the patient or prior symptoms. Some patients may report an overuse episode before the appearance of pain. On clinical examination, patients usually show a full range of motion in both flexion-extension and pronation-supination but may show a very slight decrease in terminal extension with supination and pain. The radial tuberosity can be painful when palpated, which is possible in thin patients with the elbow flexed and with the forearm pronated. The examiner passively pronates and supinates the forearm while pressing on the radial tuberosity, and if painful, it is a very clear sign of distal biceps pathology. *The hook test* is a very useful test to assess the integrity of the distal biceps [40]. The patient is asked to bilaterally flex the shoulder to head level and to flex the elbow approximately to 90° while maintaining the forearm in supination. While keeping this position, the examiner slides his index finger on the antecubital fossa and will hook on the distal biceps tendon. If the tendon is not present, the index finger will not hook, indicating a complete rupture of the distal biceps. If the tendon is present, but painful when "hooked," this is indicative of tendinopathy, and partial ruptures should be ruled out. Other clinical findings include pain in the antecubital fossa with resisted supination with the arms in almost full extension (mild cases) or with the elbow in 90° of flexion.

15.5.2 Imaging

Radiographs of the elbow are typically normal but may show indirect signs of tendinopathy, such as flattening of the radial tuberosity [41]. Ultrasound (US) is accurate to diagnose complete tendon ruptures, but its role in diagnosing partial ruptures is less clear [42]. Magnetic resonance imaging (MRI) is favored because it can evaluate the entire course of the distal biceps tendon and may assess the presence of a partial tear, the presence of tendinopathic changes, or the presence of bicipitoradial bursitis. These changes may be best seen using the FABS view (flexion-abduction-supination) in which the patient is placed prone with the affected arm completely abducted with the arm flexed and the forearm in supination [43]. Of note, it may be difficult to distinguish between tendinosis and partial tears involving less than 50% of the tendon. Findings related to the presence of a partial tear include increased signal intensity in the distal biceps, the presence of peri-tendinous or intra-sheath fluid, and increased bone marrow signal at the tendon insertion site [43].

15.5.3 Management

Partial tears are typically a delayed diagnosis, so patients may have tried different treatments at the time of consultation. A trial of 6 months of conservative management seems reasonable. Conservative management has not been clearly protocoled, and most authors use physical therapy, the cessation of aggravating activities (including splinting), NSAIDs, and steroid/anesthetic injections. Progressive strengthening is recommended until patients can perform their desired activities and it can be useful in some patients, a recent systematic review of surgical outcomes of partial ruptures showed that only in 5 of 65 patients who received conservative management that this form of treatment was effective [44]. High-grade tears involving more than 50% of the attachment site have more failures after conservative management, and some patients could benefit from early surgical repair.

15.5.3.1 Endoscopic Techniques

The use of endoscopy to treat distal biceps injuries has been recently reported using different techniques [41, 45]. Endoscopy can be utilized as a diagnostic aid in evaluating the extent of the rupture, for removing adjacent bursitis, to debride the partial biceps tear or to complete and reattach the tendon. However, it should be reserved for experienced arthroscopists. The patient is placed in supine position, with the arm on an arm table. A tourniquet is helpful for visualization, and in partial ruptures the risk of not reaching the attachment site is nonexistent.

The tendon can be palpated, and it is usually central on the forearm. The incision can be made 3–4 cm distal to the elbow crease. Blunt dissection is carried out until the tendon is apparent. Injury to the lateral antebrachial cutaneous nerve (LABCN) and the posterior interosseous nerve (PIN) is a frequent complication.

To decrease the rate of these complications, we recommended handheld retractors and avoid Hohman retractors around the radial neck and tuberosity. The scope is advanced to the bicipital tuberosity, and the forearm is supinated to improve the working space.

The medial fibers are usually intact in cases of a genuine partial tear. The distal short head of the biceps can be ruptured with preservation of the proximal long head of biceps insertion, and ganglions at the site of rupture are frequently seen [46]. Vandenberghe and van Riet suggest the following protocol to decide appropriate treatment of distal biceps tears [41]. Tears smaller than 25% are debrided; tears comprising between 25 and 50% are partially repaired with the use of an anchor; and those tears greater than 50% are detached and fixed using a cortical bone technique. In the latter, the scope can be used to localize the proper insertion side, and while removing the scope, the sheath can provide protection for the drills used for cortical preparation. A guide wire is drilled in the center of the tuberosity through both cortices and must be directed straight posteriorly or with slight ulnar deviation. The guide wire is over-drilled with a bigger cannulated drill in the first cortex and a smaller drill in the second cortex (different systems may have different sizes).

If we put trailing sutures on the guide wire, we can advanced it through the posterior forearm to introduce the button and tendon into the drill site until the button has passed the second cortex. It can then be flipped by flexing and extending the elbow. Alternatively, an antegrade sliding technique can be used. There, after we passed the button and tendon through the second cortex, we deployed it from the handle, and toggling with the suture achieves flipping of the button. Sliding and tensioning of the limbs of the suture advance the tendon to the desired position. The sutures are then tied, and the position is locked. Otherwise, an interference screw can be used to secure the tendon and offset it to its lateral position in the radial tuberosity.

15.5.3.2 Open Techniques

The techniques used for the treatment of partial distal biceps tears include a single anterior incision, a single posterior incision, or a double incision, but most cited authors in a recent systematic review use the single anterior incision approach [44]. Tendon fixation may be accomplished with a cortical button, a suture anchor, or transosseous sutures.

Load to failure and pullout strength are higher with an endobutton device when compared to other fixation devices, but accelerated protocols have been described with all kinds of fixation.

For a single anterior incision technique, the patient is placed supine with an arm tourniquet and the hand on an arm table. The incision is placed longitudinally along the medial border of the brachioradialis while protecting the LABC. This protection may be achieved by simple measures, including not dissecting it and avoiding self-retaining retractors.

The lacertus fibrosus is usually intact and can be detached for improved exposure. The biceps tendon is exposed and is followed distally toward the radial insertion. The decision-making process and techniques are similar to those presented under “Endoscopic techniques.”

15.5.4 Rehabilitation

Described postoperative protocols are very different and vary from a short course of immobilization, including splints, to the liberal use of the arm. At 3 months, the tendon is considered to have healed, and heavy strengthening can start.

During the first 12 weeks, the goals are to regain full range of motion and then add resistance training with small weights (1 kg), and eventually no restrictions are placed on the normal activity. It does not appear that the rehabilitation regime affects the results of surgery.

15.5.5 Results

Behun et al. presented outcome following surgical intervention for partial tears of the distal biceps in a systematic review [44]. Nineteen studies including 86 patients were reported. Surgery was performed with a single anterior incision in 50 cases, a single posterior incision in 11 cases, and two incisions in 6 cases, and the remainder was not specified. Fixation was performed using transosseous suture (seven studies), suture anchor (five studies), and cortical button (five studies). Surgery yielded satisfactory outcome in 94% of cases. There was one fixation failure (transosseous) that was revised using the suture anchor technique 4 years after the index operation. Two patients (2.3%) reported an unsatisfactory result due to weak supination, one of them being revised with fixation to the brachialis. Another patient reported an unsatisfactory result due to persistent LABCN paresthesia (1%).

15.5.6 Complications

Transient LABCN paresthesia was the most common complication, being encountered in 13 of 86 patients (15%), followed by transient PIN palsy in 5 patients (6%), elbow discomfort in 2 patients (2%), and asymptomatic HO in 1 patient (1%) [44].

15.5.7 Future Treatment Options

It remains to be seen if the use of biologics to treat partial tears of the distal biceps, as in other enthesopathies, will be successful or not. The use of (ultrasound-guided) PRP injection, with or without stem cell augmentation, could help modulate the inflammatory response and have a potential for regenerating damaged but still attached tendon. This strategy could prove useful for small tears. It is probable that as techniques evolve, the use of endoscopic techniques will increase over the future for larger tears, only limited by the safety of the approach.

15.6 Wrist Injuries in the Overhead Athletes

Overhead sports require high and unique physical demands and place athlete's wrists at risk of injury, leading to sport-specific and even position-specific injury patterns [47]. Up to 15% of all athletic injuries involve the hand or the wrist [48, 49]. Wrist injuries may be divided into two major groups according to anatomic localization (radial-sided or ulnar-sided) and injury mechanism (acute traumatic or overuse) [48]. Detailed classification systems have been specifically described for athletic wrist injuries: Mirabello et al. categorized them into throwing, weight-bearing, twisting, and impact injuries, being the first to use a *biomechanistic classification* [50]. The main overuse mechanisms in overhead athletes were throwing (with repetitive flexion/extension and radial/ulnar deviation) and twisting (with forceful rotation of the wrist). Werner and Plancher used a more *sports-specific classification* including impact (such as with a ball or another competitor), contact with a racket, stick, or club and external contact (e.g., gymnastics, weight lifting, and rock climbing) [51]. Due to the fact that many athletic wrist injuries can also occur in sports (such as soccer and running) where the hands are infrequently used, there is a lack of reviews and consensus in literature focusing only on the management and treatment of elite overhead athletes.

15.6.1 Biomechanics

Ryu et al. have reported that most daily activities can be executed with 40° of wrist extension, 40° of flexion, and a 40° arc of radial and ulnar deviation [52]. However, sport-specific movements may require a wider wrist range of motion (ROM) to effectively perform all phases of throwing gestures (cocking, acceleration, deceleration, and recovery). In basketball, for example, free throw shooting requires an average of 50° of extension (range, 40–56°) and 70° of flexion (range, 48–84°) for a total arc of 120° in the throwing hand. Furthermore, during the cocking phase, the wrist extends from neutral to 32° of extension, followed by rapid flexion over 94° during the 105 ms of the acceleration phase [53]. The knowledge of sport-specific ROM is crucial to predict return to sport following injuries and surgical procedures and to guide surgical and rehabilitation procedures but has not been defined yet for all sports [54]. Moreover, this knowledge has relevant implications when considering surgical interventions that may affect (i.e., limit) the range of motion in the wrist [48]. The ulnar variance also plays a fundamental role in the biomechanics of wrist injuries in the overhead athletes. In the ulna-neutral wrist, 82% of the load on the wrist joint passes across the radiocarpal joint. A relative increase in the ulnar length of only 2 mm in relation to the radius can shift the weight-bearing line toward the ulnocarpal joint and nearly double the load passing across it. This increase may lead to progressive alterations on the ulnar-sided structures, including the lunate, ulnar head, and the triangular fibrocartilage complex (TFCC) [55]. On the contrary, relative shortening of the ulna may increase the peak pressure on the distal radial ulna joint (DRUJ) [49]. Repetitive pronation and powerful grip can lead to a dynamic change in ulnar variance, with consequent change in the load distribution across the ulnocarpal joint and direct implications on overhead athletes' performance [56–58]. This has a relevant role in the immature athletes, in which repetitive axial loads across the wrist joint can cause premature physeal growth arrest of the distal radius, with higher probability of developing an ulna-positive variance at skeletal maturity [59, 60].

15.6.2 Scapholunate and Perilunate Injuries

The scaphoid, lunate, and triquetrum constitute the proximal carpal row of the wrist and are linked via the scapholunate (SL) and lunotriquetral (LT) interosseous ligaments. The mechanism responsible for most scapholunate and perilunate injuries is wrist extension, ulnar deviation, and carpal supination, most commonly resulting from a fall on an outstretched hand. Due to this common mechanism of injury, SL injury is the most common ligamentous injury in the wrist. *Perilunate instability* originating from SL tears progresses across four stages involving disruption of the scapholunate articulation, lunocapitate disruption, lunotriquetral disruption, and finally, dislocation of the lunate from the radius. A reverse perilunate injury pattern originating from LT tears, which progresses through dorsal ulnar midcarpal tear and eventually SL tear, has also been described [48]. If untreated, SL and LT injuries progress to a predictable pattern of osteoarthritic degeneration called *scapholunate advanced collapse* (SLAC) [61]. Since management of chronic injuries is associated with poor outcomes, early recognition of these injuries is paramount. However, especially in athletes, perilunate injuries are likely to be underdiagnosed and are often dismissed as simple "sprains" [47].

Overt instability on examination or localized pain over the SL or LT intervals should alert the clinician to consider additional diagnostic testing and appropriate imaging, which allows classifying perilunate injuries into "pre-dynamic," "dynamic," or "static" [62]. Suspected tears or partial tears with static and pre-dynamic instability can be managed conservatively. Dynamic SL injuries are considered an indication for arthroscopic surgery in the general population. Specific personal demands (e.g. despite the injury an athlete would like to finish the season) and coaching expectations may require postponing a surgical intervention to the end of the season in an elite overhead athlete: a specifically designed wrist orthosis has been reported to be a successful treatment in these cases been described through the use of [63].

Arthroscopy is considered the gold standard to diagnose intercarpal ligament tears: Geissler et al. developed an arthroscopic grading system to guide surgical management [64]. Surgical indications vary based on the severity of the instability, the chronicity of the injury, and the presence of degenerative changes. Recent reviews effectively summarized the spectrum of available surgical treatments, ranging from arthroscopic debridement or pinning to open salvage procedures [65]. When operating on elite overhead athletes, ROM-restricting procedures should be carefully discussed, and the patient should be informed regarding possible season-ending or even career-ending outcomes [47, 66].

15.6.3 Hamate Fractures

The hamulus, or hook of hamate, is a bony process, which is believed to function as a pulley for the flexor tendons during power grip. Fractures of the hamulus represent less than 3% of all carpal fractures and are most likely secondary to a direct blow by the counterforce to the butt end of a baseball bat or a racket [67]. Baseball is the most common athletic etiology. The dominant hand is more usually involved in tennis players and racquetball, whereas the nondominant hand is likely involved in baseball players [67]. Fractures of the hamate present with persistent ulnar-sided wrist pain and can be difficult to diagnose on routine radiographic views of the wrist; the carpal tunnel radiographic view can demonstrate pathology at either the pisiform or hamate hook, and CT and MRI are used to confirm the diagnosis or detect occult fractures. Appropriate index of suspicion, combined with directed physical examination, aids in early diagnosis [68]. Treatment of hook of the hamate fractures in athletes can range from casting to open reduction and internal fixation or excision [69]. Undisplaced hamulus fractures can heal uneventfully with cast immobilization [70], although with the risk of flexor digitorum profundus tendon lesion [71]. Due to the risk of nonunion, flexor tendon thinning, and neurovascular impingement, the excision of the fracture fragment through a palmar approach is usually considered the standard treatment [72, 73]. However, because of the possible decrease in flexion strength with the excision procedure, which may be undesirable for some overhead gestures, the open reduction through formal open palmar approach or percutaneous dorsal approach and internal fixation has been reported as a valid alternative technique [74]. Return to sport after surgery is often seen at 6–8 weeks postoperatively [75–77].

15.6.4 TFCC Lesions

The triangular fibrocartilage complex (TFCC) is an arrangement of structures composed of fibrocartilage and ligaments that originate from the sigmoid notch on the ulnar border of the articular surface of the distal radius and insert into the base of the ulnar styloid and fovea of the ulnar head [78]. Traumatic injuries of the TFCC result from a forced axial load to the wrist in an extension-pronation-ulnar deviation position, such as in a fall on an outstretched hand. Alternatively, a chronic mechanism of TFCC injury may occur from a distraction force applied to the volar forearm or wrist, as frequently seen in racket sports. Micro- or repetitive trauma from rapid supination-pronation of the ulnar deviated wrist (as seen with swinging a baseball bat) can cause peripheral tears to the TFCC [79]. Ulnar-positive variance is considered an anatomical risk factor for TFCC injury [55]. The injured patient typically reports pain that is aggravated by activity on the ulnar side of the affected wrist, with painful clicking or locking with supination and pronation [78]. A wait-and-see approach, frequently considered in the general population with suspected TFCC lesion, is not considered appropriate for the high-performance athlete [78]. MRI evaluation by an experienced radiologist is therefore recommended as an early step in the diagnostic algorithm when treating the elite athlete [80, 81]. Early arthroscopy is recommended for both diagnosis and possible treatment, with conservative treatment being accepted to avoid abrupt interruption of the season [78]. Immobilization for a period up to 3 months, with or without physical therapy, can be helpful for alleviating symptoms if extensor carpi ulnaris (ECU) tendinitis is associated [82]. Recalcitrant or recurring symptoms require arthroscopy for definitive classification as set forth by Palmer and treatment [83]. Symptomatic peripheral TFCC tears should be repaired, either open or with arthroscopic assistance, and typically require 3 months until the athlete is able to return to play [84–87]. Symptomatic tears of the central articular disk which fail conservative management can be treated with arthroscopic debridement (with or without a concomitant ulnar shortening osteotomy if indicated) but are not amenable to repair. Return to play is generally permitted at 2–3 months after surgery for all sports [78].

15.6.5 ECU Tendinopathy

The extensor carpi ulnaris (ECU) originates from the lateral epicondyle and inserts at the base of the fifth metacarpal. At the wrist, the ECU tendon is located in the sixth dorsal extensor compartment, held within the ECU groove on the dorsal ulna by the extensor retinaculum and its own separate subsheath [88]. The tension on this subsheath has been postulated to be greater during repetitive activities involving supination with a flexed and/or ulnarly deviated wrist [89], such as racket sports that require a snap of the wrist. These repetitive stresses on the ECU subsheath may cause synovitis, tearing, and subluxation/dislocation of ECU tendon.

On physical examination, patients with ECU lesions may present with a spectrum of symptoms, from vague dorsal ulnar wrist pain to reproducible dislocation of the tendon [90]. These lesions were classified into three groups to guide treatment: instability, tendinopathy, and tendon rupture [91].

Tendinopathy of the ECU is usually treated conservatively. Rest, immobilization in wrist extension, and ulnar deviation, followed by progression to isometric and eccentric exercises, may help athletes to return to their activities. In cases of ECU instability, conservative management is still initially employed, with a period of immobilization in a pronated, extended, and radially deviated wrist for 6–8 weeks in order to stabilize the ECU tendon in its own groove [92, 93].

Surgery is proposed as the first-line treatment for acute traumatic ECU instability [94]. Non-anatomic reconstruction of the subsheath with extensor retinaculum [89] or anatomic repair with reduction of the periosteum and subsheath back in the ulnar groove [95] was described as successful option to return to sports. After ECU sheath repair, a period of immobilization in a long arm cast for 4–6 weeks with the forearm in neutral rotation and elbow at 90° is recommended. Return to sport is permitted another 2 months after immobilization [94, 96].

15.6.6 Future Directions

Future research should be directed toward development of safe strategies, as well as identification of individual risk factors to anticipate or prevent injuries. Due to the articular or periarticular nature of most soft tissue wrist injuries in the overhead athlete, we recommend arthroscopy as the first-choice surgical strategy, since it guarantees direct visualization and correct classification, with the advantages of a minimally invasive procedure. Timing of treatment, postoperative rehabilitation, and return to sport in overhead athletes affected by wrist injuries are still based largely on expert experience, and development of a consensus is desirable. The best treatment option at the optimal time remains tricky in the management of the elite overhead athlete, requiring an overall understanding of the athlete's unique set of circumstances and priorities [78].

15.6.7 Take-Home Message

Early recognition of wrist injuries is important, because management of chronic injuries is associated with poorer outcomes. Timing and choice of treatment must be tailored to specific athlete's demands and coaching expectations. Conservative therapy with an appropriate orthosis can be accepted to postpone a surgical intervention to the end of the season. Arthroscopy is considered the gold standard for diagnosis and early treatment of intercarpal ligament and TFCC tears.

15.7 Rehabilitation of Upper Limb in the Overhead Athlete

Throwing injuries to the elbow are common in overhead athletes. During throwing, the medial aspect of the elbow undergoes tremendous (distraction) forces, while the lateral aspect is forcefully compressed. Throwing consists of six phases: windup, early cocking, late cocking, acceleration, release, and follow-through. A number of forces act on the elbow during the act of throwing. These forces are especially maximal during the acceleration phase. Valgus stress in particular creates tensile forces across the medial aspect of the elbow, which may eventually cause tissue breakdown and inability to throw. Compression forces are also applied to the lateral aspect of the elbow during the throwing motion. The posterior compartment is subject to tensile, compressive, and torsional forces during both the acceleration and deceleration phases, which may result in valgus extension overload within the posterior compartment, potentially leading to synovitis, osteophytes formation, and stress fractures of the olecranon.

15.7.1 General Rehabilitation Guidelines For Elbow Injuries

15.7.1.1 Phase 1: Immediate Motion

During the first phase, called the immediate motion phase, minimizing the effects of immobilization, along with reestablishing the range of motion (ROM), decreasing pain and inflammation, and retarding muscular atrophy are the goals. Techniques like cryotherapy, laser, and high-voltage simulation can be utilized. Four times 15 min/day-type stretch, referred to as TERT program, can be done, particularly in the case of stiff elbow patients. As a supplement to the abovementioned ROM exercises, certain joint mobilizations, such as Grade I and Grade II, may be used to minimize pain and decrease inflammation. During the early phase of rehabilitation, voluntary activation of the muscle and retarding muscular atrophy are also important. Elbow flexor/extensor, wrist flexor/extensor, and pronator/supinator muscle groups are treated with subpainful and submaximal isometrics. Also, shoulder isometrics can also be performed with caution against internal and external rotation exercises. Immediately after the injury, scapular muscle strengthening is initiated. In order to reestablish proprioception and neuromuscular control, alternating rhythmic stabilization drills for shoulder flexion/extension/horizontal abduction/adduction, shoulder internal/external rotation, and elbow flexion/extension/supination/pronation are to be performed.

15.7.1.2 Phase 2: Intermediate

Once the patient has full throwing ROM, minimal tenderness and pain, and a satisfactory muscle test (≥4/5) of the elbow flexor/extensor musculature, Phase 2 is started. The patient is instructed to continue doing the stretching exercises for elbow and wrist ROM. In order to stretch the capsular tissue at the end range, Grade III and IV techniques may be applied. The wrist flexion/extension, pronation, and supination progress are to be observed.

For athletes, particularly the throwing ones, elbow extension and forearm pronation flexibility are important. Internal and external at 90° rotation of abduction, flexion and horizontal abduction should be maintained. Specifically, external rotation at 90° abduction is to be emphasized. Strengthening exercises, including auxotonic contractions (from concentric to eccentric), such as Thrower's Ten for upper extremity, can be performed. Finally, neuromuscular control exercises are also initiated during this phase.

15.7.1.3 Phase 3: Advanced Strengthening

In Phase 3, the advance strengthening phase, the goal is to increase the strength, power, endurance, and neuromuscular control of the athlete to get him/her ready for sport participation. The criteria that must be met in this phase are full (non-painful) external and internal rotation ROM, no pain or tenderness, and 70% contralateral extremity strength. Gradual progression to higher resistance, functional movements, eccentric contraction, and plyometric are some of the recommended advanced strengthening activities for this phase. For the purpose of restoring the muscle balance and symmetry in the throwing athlete, a program that is tailored at throwing motion, high-level neuromuscular control, dynamic stabilization, muscular facilitation, endurance, and coordination may be adapted. In this phase, one other beneficial exercise that can be utilized is plyometric drills. These exercises may start with two hands and may progress to one-handed activities such as 90/90 throws with rhythmic stabilization at the end range, external and internal rotation throws at 0° of abduction into a trampoline, and wall dribbles. Regarding the forearm musculature, wrist flexion flips and extension grips can be adapted.

15.7.1.4 Phase 4: Return to Activity

The final phase is the return-to-activity phase. At the beginning of this phase, the athlete must exhibit full pain-free throwing ROM and no pain or tenderness and pass the isokinetic test which is utilized to determine the athlete's readiness for an interval sport program. Tests are performed at 180° and 300°/s and data showing that throwing arm's elbow flexion 10–20% at 180°/s, and the dominant extensors, 5–15% stronger, should be observed. Play ball drills, including one-hand wall throws stabilization and throwing into rebounder, are evaluated for pain, technique, and quality of the movement.

Once the abovementioned goals are achieved, the formal program may be initiated. These activities include warm-up and stretching, and performing one set of the exercise program before throwing and two additional sets of exercises after the throwing. The purpose is to provide adequate warm-up along with maintenance of ROM and flexibility of the shoulder joint. One day after, the thrower should also exercise his or her scapular muscles and external rotators and perform a core stabilization program.

15.7.2 General Rehabilitation Guidelines For Shoulder Injuries

General goals in shoulder rehabilitation include (1) relieving pain, (2) gaining full ROM, (3) the strengthening of peripheral muscles, and (4) the safe use of the joint proprioception. The methods and timings that will be applied to reach these goals depend on the type of treatment applied and the diagnosis of the problem. Conservative treatment programs and programs to be applied after surgery have the same basic objectives and principles but show differences in the timing and intensity of the methods used in the program. All extremity kinetic chain approach is applied as it is in other musculoskeletal problems in rehabilitation of shoulder problems. On the upper extremity, this chain is the trunk, scapulothoracic articulation, glenohumeral joint, and distal parts of the arm. In this approach, it is essential that all structures in the kinetic chain, not a single segment, be included in order to function reliably again.

1. *Relieving the pain*: Pain may arise from the shoulder problem itself or may develop after surgery. It is tried to be relieved through rest, avoidance of pain-causing movements, cold application or analgesic current treatment, and pain relievers. Pain inhibits motion by both restricting the patient and causing reflex muscle inhibition. Motion-enhancing exercises can be started by taking the pain under control.
2. *Motion enhancement*: Depending on the diagnosis and treatment, motion-enhancing exercises can be started with passive joint mobilization, stretching, active-assisted or active exercises. In the early stages of painful problems, painless motion is generally aimed between the limits of 90° of abduction and 90° of forward flexion. While exercising on a stretch of motion exercises, the patient is lying next to the arm, with a small pillow or towel under the elbow and with the elbow flexed up to 90°. This exercise position reduces the tension created in the shoulder joint by reducing

the effect of gravity and shortening the lever arm. Therefore, the joint can be worked more comfortably in order to open the movement. As the patient's pain-free range of motion increases, he or she can continue sitting and standing on the exercises.

3. *Strengthening of the muscles*: The appropriate time to start strengthening exercises for the muscles is determined according to the diagnosis and treatment. Various exercises can be used to strengthen muscles around the shoulder. In the early stage, it is safer to begin with closed kinetic chain exercises that allow early agonist and antagonist muscle groups to contract together. These exercises do not cause joint strain because they conform to normal physiological motor patterns. For closed kinetic chain exercises, the distal segment (hand) must be stabilized in a fixed area; this area for a shoulder joint may be a wall, a door, or a table, the purpose of which is to create resistance by movement of the shoulder and scapula. Strengthening the muscles that stabilize the scapula is also very important. Scapular strengthening starts with closed kinetic chain exercises and continues with open kinetic chain exercises.
4. *Proprioceptive neuromuscular stabilization exercises*: This can also be used to speed up recovery. An example of this type of exercise is the flexion/extension pattern of the upper extremity, during which the therapist can perform rhythmic stabilization with arm elevations of 30°, 60°, 90°, and 120°. In this way, the muscles that stabilize the GH joint are operated isometrically, thereby increasing the stability of the joint.

As the patient's complaints calm down and the range of motion increases, open kinetic chain exercises can be performed. During open kinetic chain exercises, some special positions need to be used during these exercises as the loads on shoulder joints increase. Positioning of the shoulders on the scapular plane is appropriate during open chain internal and external rotation exercises. Scapular plane position is achieved by taking the arm 30–60° before the coronal plane of the thorax or approximately midway between the patient's coronal plane and the frontal plane, which reduces loading on the capsule as it is very well suited for functional movements of the shoulder joint.

Rotational exercises should be initiated in the arm and should be increased by 90° according to the healing period and patient compliance. Exercise in different positions allows the dynamic stability of the muscles to be activated by changing the stability of the GH joint highest at the level of the head and at least 90°.

The most useful open kinetic chain exercises from the functional side are plyometric exercises. These exercises are added to rehabilitation programs after tissue healing and range of motion are completed, as the muscles stretch and twist during plyometric exercises, creating high tension on the tissues. Elastic tubes, treatment balls, and free weights are suitable materials that can be used for plyometric exercises. When shoulder joints are treated specially for rehabilitation of shoulder problems, it is important to bear in mind that the entire system constitutes a movement chain and that general fitness exercises such as stretching, strengthening, and endurance exercises for the entire system should not be neglected.

Beginning and some applications of the rehabilitation program are carried out by the therapists in the direction of the physician's recommendation, but some exercises are taught to the patient to complete the healing, and they are asked to apply these at home. In order for these exercises to be performed as intended, the patient must be well trained and motivated to perform the exercises regularly so that he/she takes responsibility for the healing process.

15.7.3 Take-Home Message

The elbow and shoulder joints are common sites of injury in the overhead athlete due to the repetitive microtraumatic injuries. In collision sports, elbow injury is caused by macrotraumatic forces resulting in fractures, dislocations, and ligamentous injuries. Rehabilitation of the elbow and shoulder, whether after injury or after surgical procedure, must be progressive and sequential to ensure that healing tissues are not overstressed but provide appropriate stress to promote proper collagen alignment. The rehabilitation program should limit immobilization and achieve full ROM early, especially elbow extension. The rehabilitation program must progressively restore strength and neuromuscular control while gradually incorporating sports-specific activities to successfully return the athlete to his or her previous level of function as quickly and safely as possible. The rehabilitation of the elbow and shoulder must include the entire kinetic chain to ensure the athlete's return to high-level sports participation. As most injuries predispose joints to stiffness, an early range of motion program followed by strengthening and then a gradual throwing program should be instituted as a part of all rehabilitation programs. Most shoulder and elbow injuries can be managed conservatively with activity modification and rehabilitation. Only rare traumatic ruptures or recalcitrant cases should be selected for operative intervention.

References

1. Schroeder AN, Comstock RD, Collins CL, Everhart J, Flanigan D, Best TM. Epidemiology of overuse injuries among high-school athletes in the United States. J Pediatr. 2015;166:600–6.
2. Posner M, Cameron KL, Wolf JM, Belmont PJ Jr, Owens BD. Epidemiology of major league baseball injuries. Am J Sports Med. 2011;39:1676–80.
3. Conte S, Camp CL, Dines JS. Injury trends in major league baseball over 18 seasons: 1998-2015. Am J Orthop (Belle Mead NJ). 2016;45:116–23.
4. Fleisig GS, Andrews JR, Dillman CJ, Escamilla RF. Kinetics of baseball pitching with implications about injury mechanisms. Am J Sports Med. 1995;23:233–9.
5. Chalmers PN, Wimmer MA, Verma NN, Cole BJ, Romeo AA, Cvetanovich GL, Pearl ML. The relationship between pitching mechanics and injury. Sports Health. 2017;9:216.
6. Almeida GP, Silveira PF, Rosseto NP, Barbosa G, Ejnisman B, Cohen M. Glenohumeral range of motion in handball players with and without throwing-related shoulder pain. J Shoulder Elb Surg. 2013;22(5):602–7.
7. Crockett HC, Gross LB, Wilk KE, Schwartz ML, Dugas JR, Meister K, et al. Osseous adaptation and range of motion at the glenohumeral joint in professional baseball pitchers. Am J Sports Med. 2002;30:20–6.
8. Dines JS, Frank JB, Akerman M, Yocum LA. Glenohumeral internal rotation deficits in baseball players with ulnar collateral ligament insufficiency. Am J Sports Med. 2009;37(3):566–70.
9. Garrison JC, Cole MA, Conway JE, Macko MJ, Thigpen C, Shanley E. Shoulder range of motion deficits in baseball players with an ulnar collateral ligament tear. Am J Sports Med. 2012;40(11):2597–603.
10. Wilk KE, Macrina LC, Fleisig GS, Aune KT, Porterfield RA, Harker P, Evans TJ, Andrews JR. Deficits in glenohumeral passive range of motion increase risk of elbow injury in professional baseball pitchers: a prospective study. Am J Sports Med. 2014;42(9):2075–81.
11. Wilk KE, Meister K, Andrews JR. Current concepts in the rehabilitation of the overhead throwing athlete. Am J Sports Med. 2002;30(1):136–51.
12. Wilk KE, Macrina LC, Fleisig GS, et al. Correlation of glenohumeral internal rotation deficit and total rotational motion to shoulder injuries in professional baseball pitchers. Am J Sports Med. 2011;39(2):329–35.
13. Dugas JR. Valgus extension overload: diagnosis and treatment. Clin Sports Med. 2010;29:645–54.
14. Cain EL, Dugas JR, Wolf RS, Andrews JR. Elbow injuries in throwing athletes: a current concepts review. Am J Sports Med. 2003;31(4):621–35.
15. Rossy WH, Oh LS. Pitcher's elbow: medial elbow pain in the overhead-throwing athlete. Curr Rev Muscoskelet Med. 2016;9:207–14.
16. Van der Bekerom MP, Eygendaal D. Posterior elbow problems in the overhead athlete. Sports Med Arthrosc Rev. 2014;22(3):183–7.
17. Cohen SB, Valko C, Zoga A, Dodson CC, Ciccotti MG. Posteromedial elbow impingement: magnetic resonance imaging findings in overhead throwing athletes and results of arthroscopic treatment. Arthroscopy. 2011;27(10):1364–70.
18. Bennet GE. Elbow and shoulder lesions of baseball players. Am J Surg. 1959;98:484–92.
19. Abrams GD, Renstrom PA, Safran MR. Epidemiology of musculoskeletal injury in the tennis player. Br J Sports Med. 2012;46:492–8.
20. Shiri R, Viikari-Juntura E, Varonen H, Heliövaara M. Prevalence and determinants of lateral and medial epicondylitis: a population study. Am J Epidemiol. 2006;164:1065–74.
21. Runge F. Zur genese und behandlung des schreiberskrampfes. Berl Klein Wochenschr. 1873;10:245–8.
22. De Smedt T, de Jong A, Van Leemput W, Lieven D, Van Glabbeek F. Lateral epicondylitis in tennis: update on aetiology, biomechanics and treatment. Br J Sports Med. 2007;41(11):816–9. https://doi.org/10.1136/bjsm.2007.036723.

23. Leadbetter WB. Cell-matrix response in tendon injury. Clin Sports Med. 1992;11:533–78.
24. Nirschl RP, Ashman ES. Tennis elbow tendinosis (epicondylitis). Instr Course Lect. 2004;53:587–98.
25. Cyriax JH. The pathology and treatment of tennis elbow. J Bone Joint Surg Am. 1936;18:921–40.
26. Werner CO. Lateral elbow pain and posterior interosseous nerve entrapment. Acta Orthop Scand Suppl. 1979;174:1–62.
27. Cerezal L, Rodriguez-Sammartino M, Canga A, et al. Elbow synovial fold syndrome. AJR Am J Roentgenol. 2013;201:W88–96.
28. Binder AI, Hazleman BL. Lateral humeral epicondylitis--a study of natural history and the effect of conservative therapy. Br J Rheumatol. 1983;22:73–6.
29. Longacre MD, Baker CL. Arthroscopic management of lateral epicondylitis. Oper Tech Sports Med. 2014;22:142–7.
30. Dunkow PD, Jatti M, Muddu BN. A comparison of open and percutaneous techniques in the surgical treatment of tennis elbow. J Bone Joint Surg Br. 2004;86:701–4.
31. Moradi A, Ebrahimzadeh MH, Jupiter JB. Radial tunnel syndrome, diagnostic and treatment dilemma. Arch Bone Jt Surg. 2015;3:156–62.
32. Naam NH, Nemani S. Radial tunnel syndrome. Orthop Clin North Am. 2012;43:529–36.
33. Rempel D, Dahlin L, Lundborg G. Pathophysiology of nerve compression syndromes: response of peripheral nerves to loading. J Bone Joint Surg Am. 1999;81:1600–10.
34. Sarhadi NS, Korday SN, Bainbridge LC. Radial tunnel syndrome: diagnosis and management. J Hand Surg (Br). 1998;23:617–9.
35. Peterson M, Butler S, Eriksson M, Svärdsudd K. A randomized controlled trial of exercise versus waitlist in chronic tennis elbow (lateral epicondylosis). Ups J Med Sci. 2011;116:269–79.
36. Huisstede B, Miedema HS, van Opstal T, de Ronde MT, Verhaar JA, Koes BW. Interventions for treating the radial tunnel syndrome: a systematic review of observational studies. J Hand Surg [Am]. 2008;33:72–8.
37. Morrey BF, An KN. Functional anatomy of the ligaments of the elbow. Clin Orthop Relat Res. 1985;(201):84–90.
38. Charalambous CP, Stanley JK. Posterolateral rotatory instability of the elbow. J Bone Joint Surg Br. 2008;90:272–9.
39. Lee A, Lee-Robinson A. Evaluating concomitant lateral epicondylitis and cervical radiculopathy: a correlation was found, suggesting comanagement of the disorders. J Muscoskelet Med. 2010;27:111.
40. O'Driscoll SW, Goncalves LB, Dietz P. The hook test for distal biceps tendon avulsion. Am J Sports Med. 2007;35(11):1865–9.
41. Vandenberghe M, van Riet R. Distal biceps ruptures: open and endoscopic techniques. Curr Rev Muscoskelet Med. 2016;9(2):215–23.
42. Lobo Lda G, Fessell DP, Miller BS, Kelly A, Lee JY, Brandon C, et al. The role of sonography in differentiating full versus partial distal biceps tendon tears: correlation with surgical findings. AJR Am J Roentgenol. 2013;200(1):158–62.
43. Festa A, Mulieri PJ, Newman JS, Spitz DJ, Leslie BM. Effectiveness of magnetic resonance imaging in detecting partial and complete distal biceps tendon rupture. J Hand Surg [Am]. 2010;35(1):77–83.
44. Behun MA, Geeslin AG, O'Hagan EC, King JC. Partial tears of the distal biceps brachii tendon: a systematic review of surgical outcomes. J Hand Surg [Am]. 2016;41(7):e175–89.
45. Bain GI, Johnson LJ, Turner PC. Treatment of partial distal biceps tendon tears. Sports Med Arthrosc Rev. 2008;16(3):154–61.
46. Ruch DS, Watters TS, Wartinbee DA, Richard MJ, Leversedge FJ, Mithani SK. Anatomic findings and complications after surgical treatment of chronic, partial distal biceps tendon tears: a case cohort comparison study. J Hand Surg [Am]. 2014;39(8):1572–7.
47. Gaston RG, Loeffler BJ. Sports-specific injuries of the hand and wrist. Clin Sports Med. 2015;34(1):1–10.
48. Rettig AC. Athletic injuries of the wrist and hand. Part 1. Traumatic injuries of the wrist. Am J Sports Med. 2003;31:1038–48.
49. Amadio PC. Epidemiology of hand and wrist injuries in sports. Hand Clin. 1990;6:379–81.
50. Mirabello SC, Loeb PE, Andrews JR. The wrist: field evaluation and treatment. Clin Sports Med. 1992;11:1–37.
51. Werner SL, Plancher KD. Biomechanics of wrist injuries in sports. Clin Sports Med. 1997;17:407–20.
52. Ryu JY, Cooney WP III, Askew JL, et al. Functional range of motion of the wrist joint. J Hand Surg [Am]. 1991;16:409–19.
53. Pappas AM, Morgan WJ, Schulz LA, et al. Wrist kinematics during pitching. A preliminary report. Am J Sports Med. 1995;23:312–5.
54. Rettig AC. Elbow, forearm and wrist injuries in the athlete. Sports Med. 1998;25(2):115–30.
55. Palmer AK, Werner FW. Biomechanics of the distal radioulnar joint. Clin Orthop Relat Res. 1984;(187):26–35.
56. Jarrett CD, Baratz ME. The management of ulnocarpal abutment and degenerative triangular fibrocartilage complex tears in the competitive athlete. Hand Clin. 2012;28(3):329–37.
57. Friedman SL, Palmer AK, Short WH, et al. The change in ulnar variance with grip. J Hand Surg [Am]. 1993;18(4):713–6.
58. Tomaino MM. The importance of the pronated grip X-ray view in evaluating ulnar variance. J Hand Surg [Am]. 2000;25(2):352–7.
59. DeSmet L, Claessens A, Lefevre J, et al. Gymnast wrist: an epidemiological survey of the ulnar variance in the elite female gymnasts. Am J Sports Med. 1994;22:846–50.
60. DiFiori JP, Caine DJ, Malina RM. Wrist pain, distal radial physeal injury, and ulnar variance in the young gymnast. Am J Sports Med. 2006;34:840–9.

61. Watson HK, Ballet FL. The SLAC wrist: scapholunate advanced collapse pattern of degenerative arthritis. J Hand Surg [Am]. 1984;9(3):358–65.
62. Moyer NT, Quinlan A, Shafritz N, Morrell AB. Scapholunate and perilunate injuries in the athlete. Curr Rev Muscoskelet Med. 2017;10:45.
63. Anderson H, Hoy G. Orthotic intervention incorporating the dart- thrower's motion as part of conservative management guidelines for treatment of scapholunate injury. J Hand Ther. 2016;29(2):199–204.
64. Geissler WB, Freeland AE, Savoie FH, et al. Intracarpal soft tissue lesions associated with an intra-articular fracture of the distal end of the radius. J Bone Joint Surg Am. 1996;78:357–65.
65. Paci GM, Yao J. Surgical techniques for the treatment of carpal ligament injury in the athlete. Clin Sports Med. 2015;34(1):11–35. https://doi.org/10.1016/j.csm.2014.09.010.
66. Nathan R, Blatt G. Rotary subluxation of the scaphoid. Revisited. Hand Clin. 2000;16:417–31.
67. Stark HH, Chao EK, Zemel NP, et al. Fracture of the hook of the hamate. J Bone Joint Surg Am. 1989;71:1202–7.
68. Fufa DT, Goldfarb CA. Sports injuries of the wrist. Curr Rev Muscoskelet Med. 2013;6:35–40.
69. Bishop AT, Beckenbaugh RD. Fracture of the hamate hook. J Hand Surg. 1988;13A:135–9.
70. Whalen JL, Bishop AT, Linscheid RL. Nonoperative treatment of acute hamate hook fractures. J Hand Surg [Am]. 1992;17(3):507–11.
71. Carroll RE, Lakin JF. Fracture of the hook of the hamate: acute treatment. J Trauma. 1993;34(6):803–5.
72. Aldridge JM III, Mallon WJ. Hook of the hamate fractures in competitive golfers: results of treatment by excision of the hook of the hamate. Orthopedics. 2003;26(7):717–9.
73. Trehan SK, Weiland AJ. Baseball and softball injuries: elbow, wrist, and hand. J Hand Surg [Am]. 2015;40(4):826–30.
74. Demirkan F, Calandruccio JH, DiAngelo D. Biomechanical evaluation of flexor tendon function after hamate hook excision. J Hand Surg [Am]. 2003;28(1):138–43.
75. Gupta A, Risitano G, Crawford R, et al. Fractures of the hook of the hamate. Injury. 1989;20:284–6.
76. Parker RD, Berkowitz MS, Brahms MA, et al. Hook of the hamate fractures in athletes. Am J Sports Med. 1986;14:517–23.
77. Chen NC, Jupiter JB, Jebson PJ. Sports-related wrist injuries in adults. Sports Health. 2009;1(6):469–77.
78. Ko JH, Wiedrich TA. Triangular fibrocartilage complex injuries in the elite athlete. Hand Clin. 2012;28:307–21.
79. Avery DM, Rodner CM, Edgar CM. Sports-related wrist and hand injuries: a review. J Orthop Surg Res. 2016;11:99.
80. Potter HG, Asnis-Ernberg L, Weiland AJ, et al. The utility of high-resolution magnetic resonance imaging in the evaluation of the triangular fibrocartilage complex of the wrist. J Bone Joint Surg Am. 1997;79:1675–84.
81. Blazar PE, Chan PS, Kneeland JB, et al. The effect of observer experience on magnetic resonance imaging interpretation and localization of triangular fibrocartilage complex lesions. J Hand Surg [Am]. 2001;26:742–8.
82. Geissler WB, Short WH. Repair of peripheral radial TFCC tears. In: Geissler WB, editor. Wrist arthroscopy. New York: Springer; 2005. p. 42–9.
83. Palmer AK. Triangular fibrocartilage complex lesions: a classification. J Hand Surg [Am]. 1989;14(4):594–606.
84. Trumble TE, Gilbert M, Vedder N. Isolated tears of the triangular fibrocartilage: management by early arthroscopic repair. J Hand Surg [Am]. 1997;22:57–65.
85. DeAraujo W, Poehling G, Kuzma G. New Tuohy needle technique for triangular fibrocartilage complex repair: preliminary studies. Arthroscopy. 1996;12:699–703.
86. Corso S, Savoie F, Geissler W, et al. Arthroscopic repair of peripheral avulsions of the triangular fibrocartilage complex of the wrist: a multicenter study. Arthroscopy. 1997;13:78–84.
87. Estrella EP, Hung LK, Ho PC, et al. Arthroscopic repair of triangular fibrocartilage complex tears. Arthroscopy. 2007;23:729–37.
88. Spinner M, Kaplan E. Extensor carpi ulnaris. Its relationship to the stability of the distal radio-ulnar joint. Clin Orthop Relat Res. 1970;68:124–9.
89. Campbell D, Campbell R, O'Connor P, Hawkes R. Sports-related extensor carpi ulnaris pathology: a review of functional anatomy, sports injury and management. Br J Sports Med. 2013;47(17):1105–11.
90. Pang EQ, Yao J. Ulnar-sided wrist pain in the athlete (TFCC/DRUJ/ECU). Curr Rev Muscoskelet Med. 2017;10:53.
91. Montalvan B. Extensor carpi ulnaris injuries in tennis players: a study of 28 cases. Br J Sports Med. 2006;40(5):424–9.
92. Stathopoulos IP, Raptis K, Ballas EG, Spyridonos S-PG. Recurrent dislocation of the extensor carpi ulnaris tendon in a water-polo athlete. Trauma Mon. 2016;21(1):e19551.
93. Burkhart S, Wood M, Linscheid R. Posttraumatic recurrent subluxation of the extensor carpi ulnaris tendon. J Hand Surg [Am]. 1982;7(1):1–3.
94. Inoue G, Tamura Y. Surgical treatment for recurrent dislocation of the extensor carpi ulnaris tendon. J Hand Surg Br. 2001;26(6):556–9.
95. MacLennan AJ, Nemechek NM, Waitayawinyu T, et al. Diagnosis and anatomic reconstruction of extensor carpi ulnaris subluxation. J Hand Surg [Am]. 2008;33:59–64.
96. Allende C, Le Viet D. Extensor carpi ulnaris problems at the wrist—classification, surgical treatment and results. J Hand Surg Br. 2005;30(3):265–72.

Decision-Making in Anterior Shoulder Instability

16

Michel P.J. van den Bekerom, Derek F.P. van Deurzen, Karin M.C. Hekman, Olivier Verborgt, Klaus Bak, Marco Brioschi, Chiara Fossati, Riccardo Compagnoni, Alessandra Menon, Hassanin Alkaduhimi, and Pietro Randelli

16.1 Introduction

The shoulder is the most commonly dislocated joint accounting for approximately 50% of all major joint dislocations [1, 2]. This can be explained by the unstable characteristics of the glenohumeral joint due to a shallow glenoid, which provides a wide range of motion. The incidence of shoulder dislocations is estimated at 11.2–56.3 per 100,000 persons-years [1, 3, 4]. The shoulder usually dislocates to the anterior direction (95–97%) and less commonly to the posterior (2–4%) or inferior direction (0.5%) [5]. There are over 23 different techniques with 17 modifications to reduce a dislocated shoulder. All these techniques use a form of traction, leverage, manipulation or a combination [6]. Of these techniques, the scapular manipulation method seems to be the most successful, fastest and least painful, a method wherein the inferolateral scapular edge is medially rotated upwards while the patient lies in a prone position with the arm hanging in 90° of anteversion [7].

After reduction, the patient can be treated operatively or nonoperatively. Management of a first-time shoulder dislocation needs understanding of the advantages and disadvantages of the different treatment options. One of the factors contributing to a higher recurrence rate is young age. Robinson et al. have reported that patients younger than 20 years have an 87% rate of recurrent dislocation, while this chance is approximately 30% in patients older than 30 years [8]. Sachs et al. have shown that patients who had a

---

The original version of this book was revised.
An erratum to this book can be found at
https://doi.org/10.1007/978-3-662-56127-0_24

M.P.J. van den Bekerom (✉) • D.F.P. van Deurzen
Orthopaedic Department, Onze Lieve Vrouwe
Gasthuis, Amsterdam, The Netherlands

Orthopaedic Department, Jan van Goyen Medical
Centre, Amsterdam, The Netherlands
e-mail: bekerom@gmail.com

K.M.C. Hekman
Orthopaedic Department, Jan van Goyen Medical
Centre, Amsterdam, The Netherlands

Interdisciplinary Center For Shoulder Rehabilitation,
IBC Amstelland, Amstelveen, The Netherlands

O. Verborgt
Orthopedic Department, AZ Monica,
Deurne, Belgium

K. Bak
Aleris Hamlet Parken, Copenhagen, Denmark

M. Brioschi • C. Fossati • R. Compagnoni
A. Menon • P. Randelli
1st Department, Azienda Socio Sanitaria Territoriale
Centro Specialistico Ortopedico Traumatologico
Gaetano Pini-CTO, Milan, Italy

Department of Biomedical Sciences for Health,
University of Milan, Milan, Italy

H. Alkaduhimi
Orthopaedic Department, Onze Lieve Vrouwe
Gasthuis, Amsterdam, The Netherlands

G.M.M.J. Kerkhoffs et al. (eds.), *ESSKA Instructional Course Lecture Book*,
https://doi.org/10.1007/978-3-662-56127-0_16

higher chance of recurrent instability after nonoperative treatment included those younger than 25 years, patients who participated in contact or collision sports and patients who used their arm at or above chest level in their occupation [9].

When choosing an operation, one must consider performing a soft tissue operation, (arthroscopic) Bankart repair or a bony procedure, like the Bristow-Latarjet. Previous studies have shown that an arthroscopic Bankart repair was accompanied by less reduction of the range of motion, decreased postoperative pain symptoms and improved cosmetic results while having a shorter duration of surgery compared to a bony procedure [10, 11]. A Bankart repair was associated with less complications including the risk of shoulder stiffness, wound infections, bony nonunion, screw migration and bending or breakage, osteoarthritis and neurological injury [10–13]. However, redislocation rates are considerably higher for Bankart repair (3.4–35%) [14, 15] when compared to a Latarjet procedure (0–8%) [12]. The risk of recurrence after a Bankart repair is even higher in collision athletes [16].

Balg and Boileau have developed a scale to aid in decision-making [17]. This checklist scores age, sports activity, shoulder hyperlaxity, presence of a Hill-Sachs lesion or presence of bone loss of the glenoid (Table 16.1). Previous studies have also stated that a high rate of recurrent instability is expected in patients with bony lesions (Hill-Sachs or glenoid bone loss) with more than 25% of articular surface bone loss [18, 19]. However, a recent study of Randelli et al. suggests that a Hill-Sachs lesion is not a predictor of failure for arthroscopic Bankart repair [20], and Schneider et al. have shown that there is poor interobserver variability in detecting Hill-Sachs lesions and choosing how to manage them [21]. Moreover, Garcia et al. have reported on a 68% consensus between surgeons, varying from 39.2 to 81.6% per case, in the selection of the desired operation for patients with shoulder dislocations, meaning there is still no consensus on which operation is preferable [22]. The surgical decision-making is difficult due to a paucity of large randomized studies. This emphasizes the difficulty of knowing the considerations to make the right decision. Further considerations will be explained in the following paragraphs.

Table 16.1 The instability severity index score

Prognostic factors	Points
Age at surgery (years)	
≤20	2
>20	0
Degree of sport participation (preoperative)	
Competitive	2
Recreational or none	0
Type of sport (preoperative)	
Contact or forced overhead	1
Others	0
Shoulder hyperlaxity	
Shoulder hyperlaxity (anterior or inferior)	1
Normal laxity	0
Hill-Sachs on anteroposterior radiograph	
Visible in external rotation	2
Not visible in external rotation	0
Glenoid loss of contour on AP radiograph	
Loss of contour	2
No lesion	0
Total (points)	**10**

16.2 The Indication for Surgical Treatment: How Long and How Should We Treat our Patients Nonoperatively?

Nonoperative management of recurrent shoulder instability needs a critical diagnostic approach before initiation of a treatment plan. Classification and functional testing can help to define the type of instability and to set goals and expectations for the content and outcome of treatment. Duration of conservative treatment is multifactorial and has many subjective variables. Nonetheless, validated objective findings should be scored and weighted, but eventually shared decision-making by the surgeon, the physiotherapist and the patient is desirable [23].

Clinical assessment starts with an accurate history evaluation, with emphasis on the mechanism of injury and the level of trauma applied to the shoulder at the initial dislocation or subluxation. But also age, activity level, frequency, subluxation or real dislocation, direction and aetiology should all be considered when deciding surgical or conservative treatment [24].

The Stanmore classification of shoulder instability provides a distinguished diagnostic model, in which the more structural elements caused by extrinsic trauma (polar I) can be differentiated from the more functional elements of nonsignificant trauma instability (polar II and III).

The advantage of this classification is the connection between the polarities demonstrated by the triangle figure (Fig. 16.1), which enables to diagnose combined types of instability. For instance a traumatic dislocation that occurs in a patient familiar with asymptomatic deliberate subluxations should be diagnosed as polar III/I. Also patients with primarily polar I/II type instability could present fear avoidance because of recurrence. Eventually these could develop muscle patterning changes over time and shift towards the bottom of the triangle (polar III).

These changes should be recognized, and it has been suggested to restore these by a specialized physiotherapist before surgical treatment, to avoid persistent undesirable muscle patterning, which could jeopardize the repair and cause recurrent instability [25].

Functional tests are based on aspects of mobility, strength, scapula kinetics, kinetic chain and coordination. The real challenge is to identify the main driver behind the instability, so therapy can focus on the most relevant aspect responsible for the recurrence for that particular individual [26].

Range of motion testing focuses on the glenohumeral rotational range. In throwing athletes, population proper glenohumeral internal rotation deficit might be present due to posterior tissue tightness [27]. A left-right difference of approximately 20 degrees or more is of clinical relevance [28], where a left-right difference of the total range of motion should not be more than 5° [29].

Scapula mobility needs to provide enough posterior tilt and upward rotation during elevation to provide a stable joint base. Tightness of the pectoralis minor, levator scapulae and rhomboids has been described to jeopardize this [29]. Mobility of the thoracic spine is also a factor that plays a role in the kinetic chain of movement. Especially in the hypermobile group, *hypo*mobility of the thoracic spine combined with a lack of upward scapular rotation could generate excessive glenohumeral mobility.

The Beighton score and Brighton [30] criteria are used to determine whether joint hypermobility syndrome (JHS) is an underlying factor for

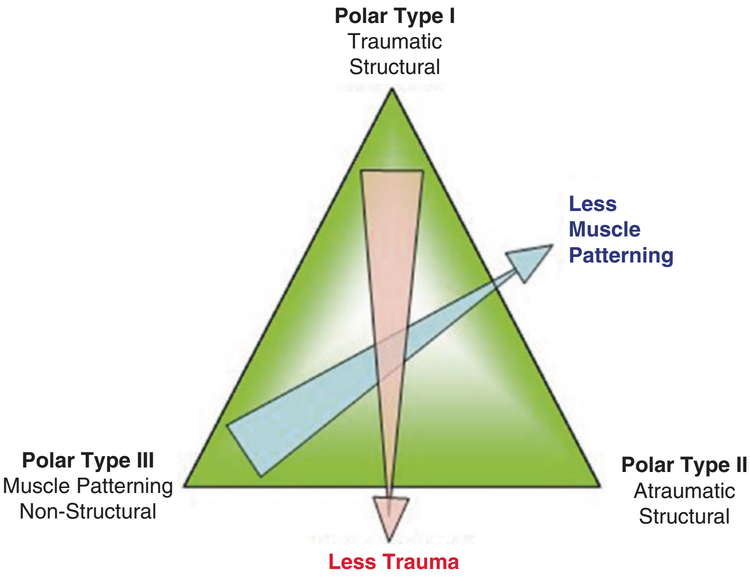


Fig. 16.1 Stanmore classification of shoulder instability

shoulder instability and differentiate the unidirectional instability from the multidirectional instable shoulder (MDI). Despite a negative Beighton/Brighton score, localized glenohumeral laxity could also indicate MDI. Recent research suggested no negative effect of JHS on the outcome after anterior stabilization [31]. However, the most common recommended treatment is exercise-based conservative treatment. Excessive joint laxity may give information about proprioceptive ability and joint position sense, which could be assessed with a two-point orientation discrimination or joint position tests [26] and indicates therefore the necessity for focus on neuromuscular control training.

16.2.1 Strength

Regarding *strength*, overhead sport-specific isometric concentric and eccentric normative data for external and internal cuff strength are available and should be used to recognize strength deficits in overhead athletes [32]. These tests should be performed bilaterally, in neutral position and in 90 degrees abduction. In general, external rotator strength should be at least 80% of internal rotator strength. The dominant side should be 10–15% stronger than the non-dominant side [33]. External rotation fatigue EMG tests are used in overhead athletes to determine whether external rotation fatigue alters scapula kinematics [34].

Although strength training is important to address, patients with recurrent instability show initially mainly deficiencies in neuromuscular control of the rotator cuff. The dynamic rotary stability test can be used to assess the rotator cuff ability to control the centralization of the humeral head into the glenoid cavity through the full range of movement [25]. Another important aspect of this neuromuscular control is the ability of the rotator cuff to quickly react on external forces in the whole range of movement especially in vulnerable positions. Unfortunately, reaction time tests have not yet been validated.

The first stage in rehabilitation should focus on these neuromuscular deficiencies. Ability to consciously contract the rotator cuff in all arm positions is a primary condition necessary before starting strength training.

16.2.2 Scapula Control

Scapular dyskinesia has been found to be related to shoulder instability and is mainly secondary to rotator cuff inhibition. Internal rotation and an increased downward rotation at onset of elevation have been associated with glenohumeral instability. Therefore, scapula-thoracic mobility, muscles coordination and strength play an important role in dynamic scapula stability to enhance rotator cuff strength. Since individuals show a high variability of motor strategies recruitment timing of force couples like upper and lower trapezius and serratus anterior through range of movement, surface EMG is suggested to be useful for identifying individual patterns and for biofeedback in training [35–37] (Fig. 16.2). Cools et al. published a clinical guideline for athletes and non-athletes to substantially build up the training sessions [38].

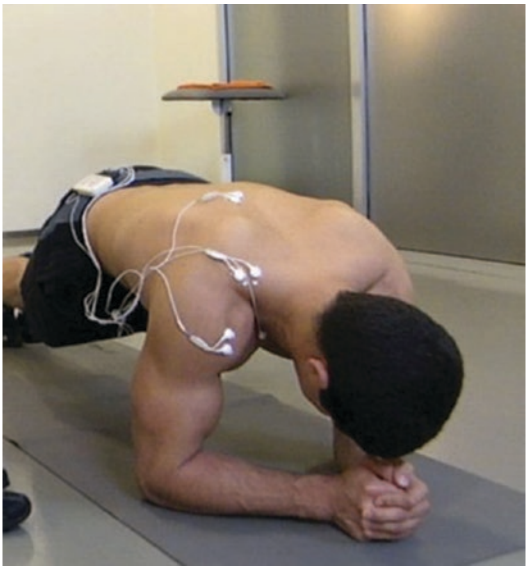


Fig. 16.2 EMG biofeedback for shoulder rehabilitation

16.2.3 Kinetic Chain

One weak link in the kinetic chain, i.e. hypomobility of the spine or hip, instability of the knee or core strength deficits, makes the shoulder more vulnerable to overuse injuries or instability. Optimal use of the chain means full range of motion, good motor control in all the joints in order to distribute forces. The lower extremities muscles are responsible for 50%, trunk muscles 30%, which leaves about 20% for shoulder muscles. This will avoid glenohumeral overuse and should therefore be evaluated with lower quadrant control tests (i.e. the one-leg squat test) [33]. Kinetic chain exercises can be prescribed in the early phase during trauma recovery and should be implemented during the whole rehabilitation process.

16.2.4 Pain and Fear

In case of persistent pain or fear for recurrence, normal movement patterns alter and could diminish scapula muscles and rotator cuff activation and could therefor maintain or worsen glenohumeral instability. As mentioned before, initial polar I/II instability could transfer towards the bottom of the triangle, where aberrant activation of larger muscles like pectoralis or latissimus dorsi suppresses the activation of the rotator cuff [25]. Creating painless and trustful training situations could be accomplished by using symptom modifications, like closed kinetic chain exercises, manual approximation of the humeral head in the glenoid or scapula assistance, postural muscle activation or external resistance for cuff activation on the arm while moving.

Avoidance to move towards risk positions may be a consequence of pain and injury remembrance, and patients should be gradually exposed into these positions during therapy. In case of longer duration of symptoms, alterations in the senso-motoric cortex and increased white matter connectivity within the pyramidal tracts lead to changes of proprioceptive behaviour. These patterns are also observed in chronic pain patients, and development of these changes over time should be avoided by providing the right initial treatment [39].

Altered proprioceptive ability, caused by associated peripheral nerve injury, present in 6% of the cases, [40] could give a delay of improvement and should therefore be recognized.

16.2.5 Treatment and Duration

Training should primarily focus on neuromuscular control of the rotator cuff and scapula in closed kinetic chain exercises and weight bearing to facilitate the cuff activation. Also external rotation resistance during open kinetic chain exercises stimulates the cuff and enhances stability through range. Core and lower quadrant strength exercises integrated into the treatment programme provide the base for cuff function and eventually resulting in glenohumeral joint stability [23, 29, 36, 41]. In the high-athletes population, several cut-off points for return to play have been described for glenohumeral ROM, cuff strength, pectoralis minor length and scapular upward rotation strength [29]. One could use the same objectives for finishing treatment in the nonathletes; however, in this group, particularly the non-traumatic instability, decision-making is much more based on symptomology and functional goals. Generally at least 3–6 months of training by a specialized physiotherapist should have been tempted before deciding whether additional stabilizing surgery is needed.

In cases with isolated anterior subluxations presented without a positive apprehension test, good neuromuscular control, optimal mobility and strength values, no scapula dyskinesia and a good kinetic chain function, but subluxation during horizontal abduction force, long rehabilitation might not be as effective, and early surgery should be discussed, though this expert opinion is not based on guiding literature.

16.2.6 Discussion

Boffano [42] provides a treatment algorithm where young age, highly demanding physical activities and the absence of soft tissue laxity are factors that support early surgery. In the young group without any overhead activities, conservative treatment is recommended until chronic instability becomes symptomatic. There is some evidence that an increased number of dislocations and a delayed time to stabilizing surgery are associated with an increased risk of postoperative recurrent instability. However, a consistent rehabilitation programme in time is suggested to minimize this [36].

Interesting is that even after surgical repair, 3–51% will stay apprehensive in position of abduction and external rotation [43]. The work of Lädermann et al. revealed that after glenohumeral stabilization, the anterior, superior and inferior translation of the humeral head from the centre of the glenoid during movement was not significantly reduced compared to preoperative values. This residual postoperative instability might explain the positive apprehension combined with pain or fear and trauma remembrance stored in the brain due to proprioceptive dysfunction or possible peripheral nerve injury, which reduces the ability to protect the joint in more extreme positions.

Considering the above, a positive or negative apprehension-relocation test does not indicate the need for surgery before at least all the above-mentioned functional elements have been addressed.

16.2.7 Take-Home Messages

- Diagnosing shoulder instability with the Stanmore Classification should be used to discover functional elements, like muscle patterning, that need to be addressed to get the right initial treatment.
- Cut-off values have been described for return to play for the overhead athlete, who could also be used for the nonathletes. However, for atraumatic instability, these values might not be applicable, and decision-making is more subjective and guided by individual functional goals.
- Neuromuscular control (timing, endurance, reaction time) in all arm positions should be trained in all kinds of instability before starting strength training.
- The value of the apprehension-relocation test might be taken into reconsideration for the treatment decision.

16.3 Surgical or Conservative Treatment for First-Time Dislocations in High-Level Athletes?

16.3.1 Introduction

It is generally well known that first-time glenohumeral dislocations in high-level athletes imply a greater risk of early recurrence. The recurrence rate of Bankart repair in contact athletes is two to three times higher than in non-contact athletes [44].

With regard to the risk factors involved in recurrent instability, existing literature strongly points to these aspects—age under 25 years, sports-active especially with contact, high-level athletes and significant associated bony lesions [45]. The balance of the treatment strategy can be delicate and should be individualized where clinical decision must be based on a thorough clinical evaluation including MR arthrography and CT scanning with 3D reconstruction and discussion with the athlete, the physiotherapist and the coach/parents. The treatment should be planned in a way that the risk of recurrence and possible new aggravated pathology are avoided and the treatment course matches the sports requirements of the athlete.

The sports associated with the highest risk of recurrent dislocation are collision sports such as American football and rugby. Overhead sports may have a lower risk of redislocation, but at the same time, there is a high demand for a stable glenohumeral joint when performing the sport. Table 16.2 shows the most common sports associated with a high risk of glenohumeral dislocation and recurrence. Bony Bankart and Hill-Sachs lesions are more frequent in collision sports, whereas a more complex pathology and dysfunction are seen in throwing sports. These are

Table 16.2 Sports associated with a high risk of glenohumeral dislocation and recurrence

High-risk collision sports
Rugby
American football
Aussie rules football
Soccer
European-type handball
Martial arts
Throwing sports with a high demand of stability
Racket sports
European-type handball
Javelin throwers
Cricket players
Baseball

important factors that should be taken into account when planning the treatment.

A number of factors have to be considered after primary glenohumeral dislocation in a high-level athlete. At initial clinical examination, the patient's age and activity demands as well as in-season timing should be discussed. In throwers, any dysfunction that may prolong rehabilitation or that may be necessary to correct in order to avoid recurrence must be assessed. An analysis of the kinetic chain as well as muscular weakness and scapula dysfunction should be integrated in the examination. An MR arthrography will show the extent of ligament lesion and associated lesions as well as indicating lesions and location of glenoid and humeral head bone lesions. Due to the high demands in high-level athletes, a CT scan with 3D reconstruction is needed in all cases to evaluate possible bone loss. After assessing the pathology and the athlete's demands, the sum of the clinical observations should lead to a discussion of the indication for and timing of surgery, the type of procedure and its prognosis and drawbacks. In some cases, a course of nonoperative treatment may be the initial choice after thoroughly instructing the athlete and the coach or trainer regarding the steps of progression in treatment and possible quick recurrence or lack of progression that can lead to reconsideration of the treatment plan. Another factor to be considered is to postpone the operation to the "off season", thus not affecting the work of professional athletes.

16.3.2 State-of-the-Art Treatment

(a) Nonoperative treatment: in a narrative review, Burns and Owens stated that the athlete is able to return to sports within 3 weeks after the injury and that surgical management results in a long absence from sports activity [46]. This illustrates some of the thoughts that should be considered when planning the treatment. Buss et al. showed that 26 out of 30 athletes with glenohumeral instability treated with physical therapy and a brace were able to return to sports for the entire season with an average time missed of 10 days [47]. One third, however, suffered from sports-related recurrent instability episodes during the observation period, and 16 out of the 30 underwent surgical stabilization [47]. In a similar study, Dickens et al. showed that 73% athletes returned to sport for either all or part of the season after a median 5-day absence from competing, while 27% successfully completed the season without recurrence [41]. Sixty-four percent of athletes returned to in-season play and had subsequent recurrent instability, including 11 recurrent dislocations and 10 recurrent subluxations. Athletes with subluxation were 5.3 times more likely to return to sport during the same season as compared to those with dislocations. Logistic regression analysis suggests that the Western Ontario Shoulder Instability Index and Simple Shoulder Test administered after the initial instability event are predictive of the ability to return to play [41]. Both studies underline that it is possible to return to sports after a nonoperative treatment of an instability episode, but the risk of recurrence is much higher than the chances of success. The risk of aggravated injuries with recurrence and a possible worse prognosis of surgical treatment should be taken into account.

(b) Operative treatment: although the literature supports that the risk of recurrence is lower and the quality of life is significantly greater after surgical treatment of anterior glenohumeral dislocation, the rehabilitation period is prolonged compared to nonoperative treatment, and this may be an obstacle for some athletes [48]. Depending on the sport and the pathology, the recovery time may vary from 4 to 6 months before sports activity can be resumed. On the other hand, as stated above, the risk of recurrence is dramatically reduced with surgical treatment, even in high-level athletes. According to the literature, the results of an open Bankart repair are superior compared to that of arthroscopic Bankart repair. Harris et al., however, in a comprehensive review, found that using suture anchors in an arthroscopic Bankart repair produces the same good outcome regarding return to sport and recurrence rate [49]. When all arthroscopic Bankart repair studies are pooled including transglenoid technique, the recurrence rate is higher [49]. Larrain et al. showed excellent results with a 4–9-year follow-up in collision athletes. The risk of recurrence was 5% in the acute dislocation group compared to 10% in the recurrent dislocation group [50].

(c) Today, there is no consensus on the preferred choice of the stabilising procedure. The procedure depends on the pathology and, not in the least, the possible involvement and extent of bony lesions—unipolar or bipolar. A simple arthroscopic Bankart repair can be performed for Bankart lesions without bone loss. An additional remplissage may be performed for instabilities with a significant Hill-Sachs lesion. For significant bone loss on the glenoid side, or in case of an obvious off-track lesion, a Latarjet procedure would be preferred. When these pathology-related treatment options have been considered, the surgeon should be aware of the sports-specific risk of recurrence. In high-level athletes active in collision and contact sports, the trend will lean more towards a Latarjet procedure due to the increased risk of a new high-impact trauma. Bessiere et al. compared the outcome of coracoid bone block transfer with arthroscopic Bankart repair and found a recurrence rate of 24% in the arthroscopic Bankart group compared to 12% in the Latarjet group, although this difference was not significant [51].

(d) Postoperative treatment and prognosis: the postoperative rehab should be planned by the surgeon and a specialized shoulder physiotherapist with emphasis on ROM exercises and scapular control for the first 6 weeks followed by increased strength training and sports-specific exercises. The prognosis differs with respect to the level of overhead involvement and risk of collision. Stein et al. divided their population in non-collision/non-contact athletes (G1), collision athletes (G2), overhead throwing athletes (G3) and martial arts (G4) and found significant longer recovery after arthroscopic Bankart in G3 and G4 athletes [52]. Petrera et al., when comparing the outcome after arthroscopic Bankart in collision and non-collision athletes after a minimum of 2 years follow-up, showed that there were no recurrences in the non-collision group, whereas the rate of recurrence was 9% in collision athletes [53]. Seventy-three percent of collision and 81% of non-collision athletes were able to return to sport at their preinjury levels [53].

16.3.3 Future Treatment Options

In order to enable high-level athletes to return to sport quickly and safely with a low recurrence risk, the future will provide us with more advanced diagnostic techniques combined with software assessing prognosis for the best individual treatment options.

16.3.4 Take-Home Messages

Currently, it is well-documented in the literature that high-level athletes involved in collision or throwing sports have a high risk of recurrence after a primary anterior glenohumeral dislocation. Physiotherapy and bracing can return the athlete to the same level sports in 3–4 weeks, but the risk of recurrence is approximately 50%. The choice of surgical procedure is based on correcting the pathology combined with an estimation of the sports-specific risks of a new episode of instability.

16.4 Bankart Versus Latarjet in Patients with Small or No (Glenoid and Humeral Head) Bone Loss

16.4.1 Introduction

The best surgical treatment for recurrent traumatic anterior shoulder instability still continues to be a challenge for orthopaedic surgeons, with several techniques being described over time [54].

Definitive management of anterior instability is crucial in order to allow recovery of shoulder function, return to daily and sports activity and prevent instability arthropathy (Fig. 16.3). Among the numerous available procedures, the repair of the capsulolabral tear (Bankart repair) and the coracoid bone transfer (Bristow-Latarjet) are the two most commonly performed techniques using open or arthroscopic approaches to treat anterior shoulder instability. Due to the high frequency of small or moderate bone loss (80%) [55, 56], new studies suggest to redefine the critical cut-off between soft tissue and bony procedures, showing potential inaccuracy of the diameter-based glenoid bone loss quantification [57]. Thus, the appropriate treatment choice remains tricky in case of preserved bone stock.

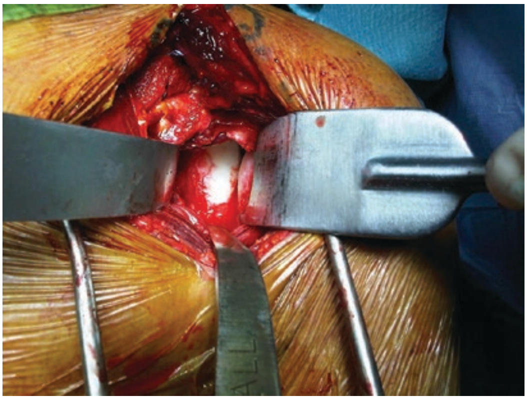


Fig. 16.3 Anterior glenoid erosion in recurrent shoulder instability

Historically, a bone loss of 20–25% of the largest anteroposterior glenoid width and an engaging Hill-Sachs have been described as the cut-off for choosing a bony augmentation procedure, due to the high rate of failure of the Bankart repair in these clinical conditions [18, 19]. In the past two decades, Bankart repair was considered the gold standard in patients without significant bone loss, but the redislocation rate could still exceed 13% [58, 59], with higher failure rates in collision and contact athletes [60–62].

Supporters of the Latarjet technique consider this procedure safer, especially for patients that are young and active or practice contact sport. On the other hand, Bankart repair restores the anatomy of the shoulder and preserves the range of motion (ROM); thus, supporters of this technique consider the Latarjet an overtreatment.

However, the literature about this topic is scarce, and no large randomized clinical trials directly comparing the two surgical techniques have been published. Most clinical evidence comes from retrospective studies considering just one single technique rather than direct comparison of the two procedures; only few comparative studies investigated the relationship between surgical treatment and patient outcomes in case of small (<25%) or no bone loss. This relative paucity of studies directly comparing these two procedures can be attributed to the confidence of the surgeon with just one of these procedures. Moreover, the rate of recurrence seems to be influenced not only by the surgical technique but also by several failure risk factors (such as young age, male sex, number of dislocations before surgery and competitive sport level) that may affect the possibility of definitive comparison among single-technique studies [15].

Some biomechanical investigations focused on the rationale of both techniques in case of small or no bone loss. In a cadaveric model, the reliability of Bankart repair was evaluated when the bone stock of the shoulder is preserved; on the contrary, the glenohumeral translation, rotational range of motion or humeral head position were not restored with the capsulolabral repair when glenoid defect was larger than 15% or more than the largest anteroposterior glenoid width [63]. In another cadaveric study, the stabilizing mechanism of the Latarjet in the setting of soft tissue insufficiency and preserved bone stock was tested: this technique limited anterior translation of the humeral head while preserving shoulder range of motion [64].

Latarjet procedure in patients without glenoid bone loss could fail because of coracoid osteolysis and fibrous nonunion. This could be explained by a diminished mechano-transduction effect at the bone healing site. In keeping with this, the coracoid bone graft seems to undergo much less osteolysis in patients with critical glenoid bone loss [65].

16.4.2 State-of-the-Art Treatment

At our knowledge, only three comparative papers dealing with both procedures in case of small or no bone loss were published, one considering the open Bankart repair and two considering the arthroscopic Bankart repair (Table 16.3). In all these studies, the Bristow-Latarjet procedures were performed using an open approach.

In a case-control matched study, Blonna et al. compared these two techniques, with particular focus on return to sport after surgery [66]. The patients with glenoid loss greater than 20% were excluded from the study.

After a mean follow-up of 5.3 years (range, 2–9 years), the main result of this study was that, despite less efficiency in terms of redislocation rate, the arthroscopic Bankart repair was associated with a better return to sport (SPORTS score, 8 vs 6 points; $p = 0.02$), better external rotation in the throwing position (ROM, 86 vs 79°; $p = 0.01$) and finally better subjective perception of the shoulder (subjective shoulder value, 86 vs 75%; $p = 0.02$). Nevertheless, more than 80% of the patients returned to their sport after both Bankart and Latarjet. The return to sport at a higher level score was significantly superior for the arthroscopic Bankart repair. The study also showed that patients playing demanding upper extremity sports (e.g. rugby, swimming) at a competitive level (Degree of Shoulder Involvement in Sports Scale: 9 or 10) had a lower level of return to sport with both techniques. The Latarjet procedure was associated with a significant loss of external rotation in the throwing position. The rate of recurrent instability was in favour of the Latarjet procedure, even though the difference between the two groups was not statistically significant (Bankart repair 10% vs Bristow-Latarjet 0%; $p = 0.25$).

In the randomized study of Zarezade et al., 2 equal groups of 20 patients without bone defects, treated with arthroscopic Bankart repair or open Bristow procedure, were compared [67].

The level of performance, range of motion, pain intensity, patient satisfaction, use of analgesics and range of internal rotation were in favour of the Bristow procedure, but these differences also did not reach statistical significance. However, this study has methodological limitations introducing risk of bias: the randomization process was unclear, the primary endpoint was not reported and the study was seriously underpowered.

Table 16.3 Comparative studies with small or no bone loss

Author	Year	Level of evidence	Bone loss	Technique	Follow-up (years)	No. Bankart	No. Latarjet
Blonna et al.	2016	III	< 20%	Arthroscopic Bankart vs open Latarjet	5.3	30	30
Zarezade et al.	2014	II	No bone defects	Arthroscopic Bankart vs open Latarjet	/	18	19
Aydin et al.	2012	III	No bony Bankart	Open Bankart vs open Bristow	5.5	25	13

The study of Aydin et al. compared the results of open Bankart repair and modified Bristow operation for the treatment of recurrent shoulder instability without glenoid bone loss [68].

The level of performance of the shoulder after surgery was satisfactory both in the open Bankart repair and in the modified Bristow group (ROWE score, 85.6 vs 81.9 points, respectively; $p > 0.05$). In terms of ROM, the open Bankart repair seemed to show slight better results compared to the Bristow technique. No recurrences were noted postoperatively. They concluded that both procedures are successful for the treatment of patients with capsular laxities.

16.4.3 Future Treatment Options

In the future, it will be challenging to combine the advantages of the two approaches in order to achieve the best clinical outcomes. The study of Russo et al. reported a new arthroscopic treatment consisting a tenodesis of the upper third of the subscapularis tendon associated with Bankart repair, defined as arthroscopic subscapularis augmentation (ASA) [69]. In particular, the authors compared their new technique to the open Latarjet in patients with recurrent shoulder instability with small glenoid bone loss (>5% but <23%).

Considering the functional level of the shoulder after surgery, the ASA group demonstrated to be superior compared to the Latarjet group, even if the difference was not statistically significant. Dislocation failure rate was 4% in the ASA group and 0% in Latarjet group. In the series of sportsmen, all patients returned to sports activities at the same preinjury level.

CT scans, performed at the final follow-up in the Latarjet group, showed a lower percentage of coracoid healing in patients with small glenoid bone loss. Signs of alteration of the glenoid chondral surface were noted in five patients. In the ASA group, MRI investigation revealed no signs of glenoid chondral damage but humeral head osteochondral alterations in two patients.

They concluded that ASA repair proved to be an effective procedure for the treatment of recurrent anterior shoulder instability with small glenoid loss in comparison with the open Latarjet.

16.4.4 Take-Home Messages

In summary, Bankart repair and Latarjet procedure are both effective and safe for the treatment of recurrent anterior shoulder instability with small or no bone loss. The lack of sound comparative studies between the two techniques does not allow drawing definitive conclusions on the superiority of one procedure over the other. A new arthroscopic treatment consisting of tenodesis of the upper third of subscapularis associated with Bankart repair, as a mechanical barrier to prevent anterior instability, reported comparable results to Latarjet procedure. More accurate comparative long-term clinical trials are needed to better understand clinical and instrumental outcomes of these procedure in patients with no or small bone loss.

16.5 Which Way to Go When Deciding Between Bony and Soft Tissue Procedure?

16.5.1 Introduction

Shoulder instability is a common problem, with a reported incidence of 1–2% in the general population [3, 70–72]. Anterior dislocations cause a detachment of the labrum and almost invariably a Hill-Sachs lesion in the posterosuperior area of the humeral head (Fig. 16.4). Patients with recurrent dislocations are often limited in performing sports or overhead activities due to an unstable shoulder joint [9, 72] and will likely seek surgical treatment to avoid recurrent dislocations. Recurrent dislocations can lead to further injury to soft tissue and bony structures of the anterior glenoid rim and humeral head [55].

In case of injury related only to the labrum with insignificant bony lesions, an arthroscopic anterior stabilization is mostly performed nowadays. Although arthroscopic labrum repair offers the advantages of a minimally invasive procedure

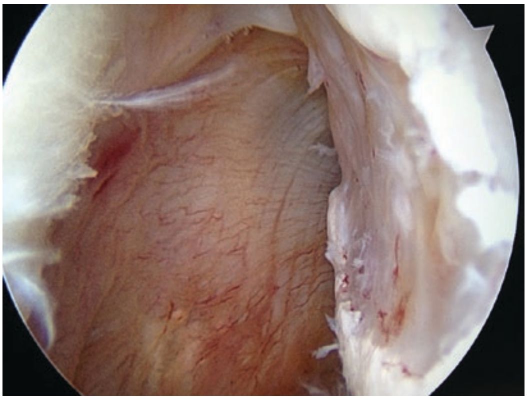


Fig. 16.4 Hill-Sachs lesion

with low morbidity and few complications, recent long-term studies reported relatively high recurrence rates [15, 73–75].

Several risk factors have been identified to bear an increased risk for recurrent instability [17]. Based on a study of 131 patients, several patient-specific factors were identified to have an increased risk for recurrent instability following arthroscopic Bankart repair such as age under 20 years, competitive and overhead sports, hyperlaxity and bony lesions to humeral head and glenoid, visible on radiographs. Based on their study, an instability severity index score (ISIS) of more than six points is a contraindication for an arthroscopic Bankart procedure, and a bony reconstruction such as the Bristow-Latarjet is advocated in those cases.

Although age and type of sport are readily assessable, the extent in which the bony lesions of the glenoid and humeral head occur is quite variable [55, 73, 76–79]. In the last decade, these bony lesions have been identified to play an important role in the risk of recurrent instability. Many studies have been conducted to optimize decision strategies with regard to the role of bony lesions of both glenoid and humeral head [80]. Probably even more important in the risk of recurrent instability is the interplay between the defect of the glenoid and humeral head [73, 81, 82].

To treat the Hill-Sachs lesion in order to diminish the risk of recurrent instability [83–85], the arthroscopic remplissage procedure of the Hill-Sachs lesion was proposed in 2008 by Wolf et al. [86]. In this procedure the posterosuperior defect of the humeral head is treated with a tenodesis of the infraspinatus tendon, decreasing the risk of engagement [73]. In case of large bony lesions of the humeral head and glenoid rim, the risk of recurrent instability after arthroscopic labrum repair is increased, and several bony procedures have been proposed to minimize the risk of recurrent instability. For example, the Bristow-Latarjet procedure (Fig. 16.5) concerns an open bony augmentation of the glenoid, with a low recurrence rate reported from 0 to 3.1%, but with a complication rate up to 30% [10, 87, 88].

16.5.2 State-of-the-Art Treatment

Most authors agree that bony procedures should be considered in the presence of certain conditions: glenoid bone loss >25%, a lesion involving >30% of the humeral head, an engaging Hill-Sachs lesion and a bipolar bone lesion even without engagement.

Careful imaging evaluation is therefore performed in order to assess the bone defects. Even though MRI has important additional value in the assessment of the glenoid labrum and rotator cuff, a CT scan is the examination of choice for studying bone defects.

Several methods have been proposed to assess bony lesions of the humeral head and glenoid rim such as plain radiography, MRI, plain CT, CT with three-dimensional reconstruction [17, 73, 77, 89–93] as well as measurement during arthroscopy [73, 79].

The Sugaya method is based on quantifying the size of the loose glenoid fragment and comparing it to the glenoid fossa (being >20%, 5–20% and <5%, respectively) [55]. Based on a cadaveric study, Itoi et al. [94] recommend making a West Point view followed by a CT scan if this is equivocal or hard to obtain due to pain or apprehension. The PICO method [95] draws a best-fit circle on the inferior portion of the uninjured, contralateral glenoid, which subsequently is superimposed onto the injured side. The area missing in the circle (the bony defect) is then

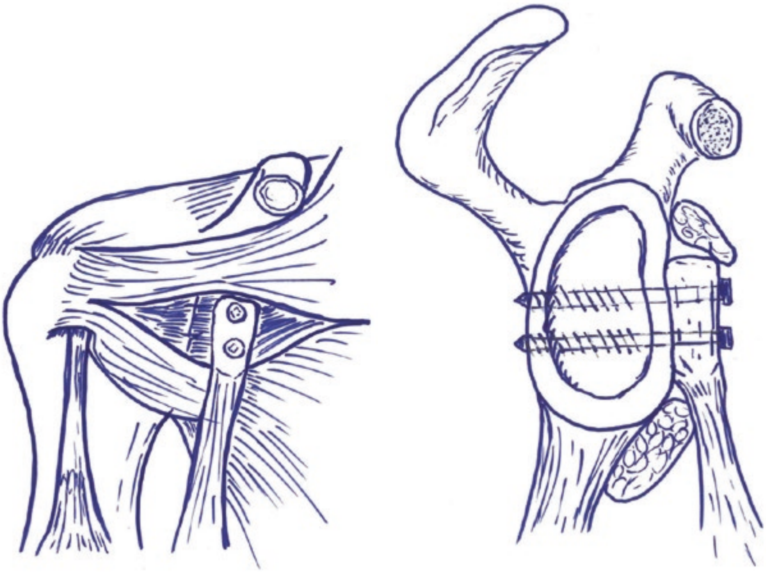


Fig. 16.5 Latarjet procedure, AP and lateral view

divided by the area of the best-fit circle to estimate the percentage of glenoid bone loss. An MRI-based method using OsiriX has also been suggested for measuring bony defects of the glenoid [90]. Despite several efforts to design a bone loss measuring tool, no consensus has been reached with regard to the cut-off value of the percentage of bone loss that determines whether to perform a bony or soft tissue procedure.

16.5.3 Future Treatment Options

The lack of consensus on cut-off values for bone loss may be due to the inherent difficulty when trying to calculate the dimensions of bone that is missing, rather than measuring structures that are remaining. Promising results have been presented based on studies with software modalities aiming to calculate contact area between humeral head and glenoid (Fig. 16.6).

16.5.4 Take-Home Messages

As bony lesions seem to play an important role in the selection process for the optimal procedure, adequate detection and quantification of these defects are indicated.

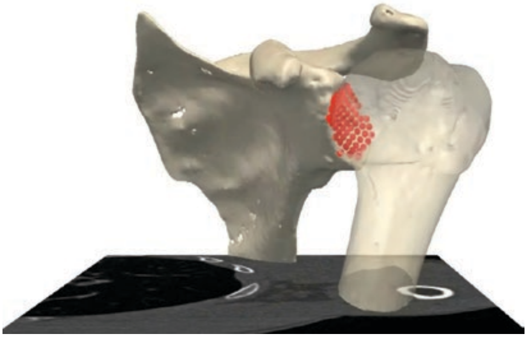


Fig. 16.6 Calculating contact area ratio of humeral head and glenoid

CT imaging, using the Glenoid Index or Pico Method, has good evidence for accurate quantification of glenoid bone loss.

16.6 Soft Issue or Bony Procedure for Recurrences?

16.6.1 Introduction

The patient with a failed instability procedure requires a thoughtful and systematic approach to achieve a good outcome. Goals of treatment should be defined, and realistic expectations should be set. Revision stabilization has a higher rate of recurrent instability, lower rates of return to play and lower clinical outcome scores.

Trauma, diagnostic errors and technical errors are the three major causes that can lead to surgical failure regardless of the type of index surgical procedure [15, 17, 96]. Overall, the best results with revision surgery are achieved when the failure had resulted from major trauma, when there had been only one prior attempt at stabilization and when there was no voluntary component to the instability [15, 97].

Fundamental to successful revision surgery is choosing the correct procedure. The decision is straightforward in athletes with clear factors that predict recurrence (significant glenoid bone loss, engaging Hill-Sachs lesions) because only a bony procedure can restore the articular arc of the glenoid. Arthroscopic revision Bankart repair may be appropriate in those athletes who have an obvious Bankart tear and no bone loss after a traumatic reinjury. The challenge for the shoulder surgeon is identifying the best surgery for the athlete who does not have such clear-cut indications. Each factor that has the potential to lead to a poor outcome needs to be collected and calculated. Patient factors (age, laxity, type and level of sport), injury factors (mechanism of injury, capsulolabral injury, glenoid bone loss, Hill-Sachs lesion) and technical factors (previous surgery performed, integrity of repair, scarring) must be integrated into the treatment algorithm. Based on this collection of factors, the shoulder surgeon should be prepared to provide the athlete with the surgery that provides the best chance to return to playing sports and the lowest risk of recurrent instability.

16.6.2 Revision Soft Tissue Repair

Although some consider open revision Bankart repair to be the gold standard for post-stabilization recurrence, several studies support the equivalence of arthroscopic revision for the appropriate indications [98–105]. However, revision Bankart repair should only be used in patients with minimal risk factors for recurrence and must not be used in the presence of significant glenoid bone loss and/or engaging Hill-Sachs lesion.

The Bankart repair attempts to return the injured capsulolabral structures to preinjury positions so that they may resume their stabilizing functions. The revision setting is frequently complicated by prior hardware, altered anatomy, glenoid bone loss, capsular attenuation and scar tissue. Essential to the repair is the restoration of the anteroinferior labral bumper. Even with intensive mobilization of scarred capsulolabral tissue from the glenoid neck, a robust labrum frequently cannot be restored. Attachment of the plicated capsule to the glenoid face may serve to recreate the labral bumper in cases of labral deficiency [98, 101]. After repair of the Bankart lesion and plication of the anterior capsule, persistent anterior laxity may be addressed with a rotator interval closure, whereas inferior or posterior laxity may require inferior or posterior capsular plication to reduce the volume of the axillary pouch and inferior capsule [98, 101].

Multiple anchors below the glenoid equator are recommended to secure the anteroinferior labrum, and some investigators recommend a low anteroinferior portal (5:00 or 5:30 position) through the subscapularis to facilitate anchor placement in the inferior glenoid [101, 103]. Orthogonal placement of the anchor into the glenoid improves pullout strength [106]. Double-loaded suture anchors have higher tensile strength than single-loaded anchors [107].

16.6.3 Bony Procedures

The coracoid transfer (Bristow-Latarjet procedure) possesses numerous advantages for athletes in the revision setting and will be the only option left in many cases. The multifactorial stabilizing effect of the transfer obviates the robust labral or capsular repair that is often difficult to achieve with a standalone revision Bankart repair. The grafted coracoid process addresses the anteroinferior glenoid deficiency commonly encountered in recurrent instability, restoring the articular surface and normalizing the glenohumeral contact pressures [108, 109]. Hill-Sachs engagement is eliminated by widening of the glenoid and extension of the glenoid arc [110]. The transferred conjoint tendon provides dynamic stability for athletes in the main position of apprehension [111]. The postoperative rehabilitation can be accelerated with no external rotation restrictions because the rigid fixation of the bone graft allows early motion, and the surgeon can have confidence in returning the athlete to play with radiographic union shown on CT [112].

Several complications specific to coracoid transfer have been reported [101, 113, 114]. Intraoperative fracture of the coracoid compromises its function as an anterior bone block. Screw breakage, loss of fixation and graft migration may result in recurrent postoperative instability. Cannulated screws have been implicated in cases of hardware failure. Postoperative pain is common in patients with graft or hardware problems. Neurovascular injury during the procedure may impair shoulder function.

In revision cases, glenoid bone loss may need to be addressed by structural bone grafting, such as in the Eden-Hybinette technique where autogenous tricortical iliac crest bone grafts are used to restore the anterior glenoid rim [115–117]. Especially after failed primary Latarjet procedures, this may be the only option left. The use of distal tibia grafts or glenoid allografts has also been suggested because of its anatomic similarity to the glenoid articular surface, conformity to the humeral head and capacity for secure fixation and incorporation [118].

Most Hill-Sachs lesions can be rendered inconsequential with an adequate anterior repair and capsular plication [119]. In large lesions that threaten stability, engagement may be eliminated with restoration of the articular arc through coracoid transfer or bone grafting [110]. Some investigators have recommended filling the defect with an osteochondral allograft to correct the humeral head deformity [84, 120]. The defect may also be filled (i.e. “remplissage technique”) with the posterior capsule and the adjacent infraspinatus tendon to prevent engagement and limit anterior translation of the humeral head [86].

16.7 Discussion

This chapter discusses several relevant factors, which are important to make the decision concerning anterior shoulder instability in daily clinical practice. It is not a guideline but can help you in choosing which diagnostic modalities and treatments are most optimal for your patient. Also, some recommendations for future research are made.

Conflict of Interest Pietro Randelli is consultant for Johnson & Johnson Medical, DePuy and Arthrex. Michel van den Bekerom and Derek van Deurzen receive institutional research support from Smith and Nephew. Klaus Bak is a consultant for Arthrex. The other authors declare no commercial associations (e.g. consultancies, stock ownership, equity interest, patent/licencing arrangements, etc.) that might pose a conflict of interest in connection with the submitted manuscript.

References

1. Zacchilli MA, Owens BD. Epidemiology of shoulder dislocations presenting to emergency Departments in the United States. J Bone Joint Surg Am. 2010;92(3):542–9. https://doi.org/10.2106/JBJS.I.00450.
2. Kazár B, Relovszky E. Prognosis of primary dislocation of the shoulder. Acta Orthop Scand. 1969;40(2):216–24.
3. Simonet WT, Melton LJ, Cofield RH, Ilstrup DM. Incidence of anterior shoulder dislocation in Olmsted County, Minnesota. Clin Orthop Relat Res. 1984;1984(186):186–91.
4. Liavaag S, Svenningsen S, Reikerås O, et al. The epidemiology of shoulder dislocations in Oslo. Scand J Med Sci Sports. 2011;21(6):e334–40.
5. Brady WJ, Knuth CJ, Pirrallo RG. Bilateral inferior glenohumeral dislocation: luxatio erecta, an unusual presentation of a rare disorder. J Emerg Med. 1995;13(1):37–42.
6. Alkaduhimi H, van der LJA, Flipsen M, van DDFP, van den BMPJ. A systematic and clinical guide on how to reduce a shoulder dislocation. Turk J Emerg Med. 2016;16(4):155–68. https://doi.org/10.1016/j.tjem.2016.09.008.
7. Alkaduhimi H, van der Linde JA, Willigenburg NW, van Deurzen DFP, van den Bekerom MPJ. A systematic comparison of the closed shoulder reduction techniques. Arch Orthop Trauma Surg. 2017;137(5):589–99. https://doi.org/10.1007/s00402-017-2648-4.
8. Robinson CM, Howes J, Murdoch H, Will E, Graham C. Functional outcome and risk of recurrent instability after primary traumatic anterior

shoulder dislocation in young patients. J Bone Jt Surg. 2006;88(11):2326. https://doi.org/10.2106/JBJS.E.01327.

9. Sachs RA, Lin D, Lou SM, Paxton E, Kuney M. Can the need for future surgery for acute traumatic anterior shoulder dislocation be predicted? J Bone Joint Surg Am. 2007;89(8):1665–74. https://doi.org/10.2106/JBJS.F.00261.
10. Griesser MJ, Harris JD, McCoy BW, et al. Complications and re-operations after Bristow-Latarjet shoulder stabilization: a systematic review. J Shoulder Elb Surg. 2013;22(2):286–92. https://doi.org/10.1016/j.jse.2012.09.009.
11. van der Linde JA, van Wijngaarden R, Somford MP, van Deurzen DFP, van Den Bekerom MPJ. The Bristow–Latarjet procedure, a historical note on a technique in comeback. Knee Surg Sport Traumatol Arthrosc. 2015;24(2):470. https://doi.org/10.1007/s00167-015-3704-7.
12. Bhatia S, Frank RM, Ghodadra NS, et al. The outcomes and surgical techniques of the Latarjet procedure. J Arthrosc Relat Surg. 2014;30(2):227–35. https://doi.org/10.1016/j.arthro.2013.10.013.
13. Longo UG, Loppini M, Rizzello G, Ciuffreda M, Maffulli N, Denaro V. Latarjet, Bristow, and Eden-Hybinette procedures for anterior shoulder dislocation: systematic review and quantitative synthesis of the literature. Arthroscopy. 2014;30(9):1184–211. https://doi.org/10.1016/j.arthro.2014.04.005.
14. Gerometta A, Rosso C, Klouche S, Hardy P. Arthroscopic Bankart shoulder stabilization in athletes: return to sports and functional outcomes. Knee Surg Sports Traumatol Arthrosc. 2014;24(6):1877–83. https://doi.org/10.1007/s00167-014-2984-7.
15. Randelli P, Ragone V, Carminati S, Cabitza P. Risk factors for recurrence after Bankart repair a systematic review. Knee Surg Sports Traumatol Arthrosc. 2012;20(11):2129–38. https://doi.org/10.1007/s00167-012-2140-1.
16. Alkaduhimi H, van der Linde JA, Willigenburg NW, Paulino Pereira NR, van Deurzen DFP, van den Bekerom MPJ. Redislocation risk after an arthroscopic Bankart procedure in collision athletes: a systematic review. J Shoulder Elb Surg. 2016;25(9):1549–58. https://doi.org/10.1016/j.jse.2016.05.002.
17. Balg F, Boileau P. The instability severity index score. A simple pre-operative score to select patients for arthroscopic or open shoulder stabilisation. J Bone Jt Surg Br. 2007;89:1470–7. https://doi.org/10.1302/0301-620X.89B11.18962.
18. Young AA, Maia R, Berhouet J, Walch G. Open Latarjet procedure for management of bone loss in anterior instability of the glenohumeral joint. J Shoulder Elb Surg. 2011;20(2):S61–9. https://doi.org/10.1016/j.jse.2010.07.022.
19. Shaha JS, Cook JB, Song DJ, et al. Redefining 'critical' bone loss in shoulder instability: functional outcomes worsen with 'subcritical' bone loss. Am J Sports Med. 2015;43(7):1719–25. https://doi.org/10.1177/0363546515578250.
20. Randelli P, Cucchi D, Ingala Martini L, Fossati C. Hill-Sachs lesion is not a significant prognostic factor for recurrence of shoulder redislocation after arthroscopic Bankart repair. J ISAKOS Jt Disord Orthop Sport Med. 2016;1(3):124–34. https://doi.org/10.1136/jisakos-2015-000017.
21. Schneider AK, Hoy GA, Ek ET, et al. Interobserver and intraobserver variability of glenoid track measurements. J Shoulder Elb Surg. 2016;26(4):573–9. https://doi.org/10.1016/j.jse.2016.09.058.
22. Garcia GH, Taylor SA, Fabricant PD, Dines JS. Shoulder instability management: a survey of the American shoulder and elbow surgeons. Am J Orthop. 2016;45(3):E91–7.
23. Gibson K, Growse A, Korda L, Wray E, MacDermid JC. The effectiveness of rehabilitation for nonoperative management of shoulder instability: a systematic review. J Hand Ther. 2004;17(2):229–42. https://doi.org/10.1197/j.jht.2004.02.010.
24. Lebus GF, Raynor MB, Nwosu SK, et al. Predictors for surgery in shoulder instability. Orthop J Sport Med. 2015;3(10):232596711560743. https://doi.org/10.1177/2325967115607434.
25. Jaggi A, Lambert S. Rehabilitation for shoulder instability. Br J Sports Med. 2010;44(5):333–40. https://doi.org/10.1136/bjsm.2009.059311.
26. Barrett C. The clinical physiotherapy assessment of non-traumatic shoulder instability. Should Elb. 2015;7(1):60–71. https://doi.org/10.1177/1758573214548934.
27. Whiteley R, Oceguera M. GIRD, TRROM, and humeral torsion-based classification of shoulder risk in throwing athletes are not in agreement and should not be used interchangeably. J Sci Med Sport. 2016;19(10):816–9. https://doi.org/10.1016/j.jsams.2015.12.519.
28. Burkhart SS, Morgan CD, Ben KW. The disabled throwing shoulder: spectrum of pathology part III: the SICK scapula, scapular dyskinesis, the kinetic chain, and rehabilitation. Arthroscopy. 2003;19(6):641–61.
29. Cools AM, Borms D, Castelein B, Vanderstukken F, Johansson FR. Evidence-based rehabilitation of athletes with glenohumeral instability. Knee Surg Sports Traumatol Arthrosc. 2016;24(2):382–9. https://doi.org/10.1007/s00167-015-3940-x.
30. Fikree A, Aziz Q, Grahame R. Joint hypermobility syndrome. Rheum Dis Clin N Am. 2013;39:419–30.
31. Koyonos L, Kraeutler MJ, O'Brien DF, Ciccotti MG, Dodson CC. A prospective cohort study investigating the effect of generalized joint hypermobility on outcomes after arthroscopic anterior shoulder stabilization. Phys Sportsmed. 2016;44(4):380–4. https://doi.org/10.1080/00913847.2016.1248225.
32. Cools AMJ, Vanderstukken F, Vereecken F, et al. Eccentric and isometric shoulder rotator cuff strength testing using a hand-held dynamometer:

reference values for overhead athletes. Knee Surg Sports Traumatol Arthrosc. 2016;24(12):3838–47. https://doi.org/10.1007/s00167-015-3755-9.

33. Ben KW, Thomas SJ. Pathomechanics of the throwing shoulder. Sports Med Arthrosc. 2012;20(1):22–9. https://doi.org/10.1097/JSA.0b013e3182432cf2.
34. Joshi M, Thigpen CA, Bunn K, Karas SG, Padua DA. Shoulder external rotation fatigue and scapular muscle activation and kinematics in overhead athletes. J Athl Train. 2011;46(4):349–57.
35. Gibson JC. Rehabilitation after shoulder instability surgery. Curr Orthop. 2004;18:197–209. https://doi.org/10.1016/j.cuor.2004.03.003.
36. Gibson J, Kerss J, Morgan C, Brownson P. Accelerated rehabilitation after arthroscopic Bankart repair in professional footballers. Should Elb. 2016;8(4):279–86. https://doi.org/10.1177/1758573216647898.
37. Struyf F, Cagnie B, Cools A, et al. Scapulothoracic muscle activity and recruitment timing in patients with shoulder impingement symptoms and glenohumeral instability. J Electromyogr Kinesiol. 2014;24(2):277–84. https://doi.org/10.1016/j.jelekin.2013.12.002.
38. Cools AM, Struyf F, De Mey K, Maenhout A, Castelein B, Cagnie B. Rehabilitation of scapular dyskinesis: from office worker to the elite overhead athlete. Br J Sports Med. 2014;48:692–7.
39. Lieberman G, Shpaner M, Watts R, et al. White matter involvement in chronic musculoskeletal pain. J Pain. 2014;15(11):1110–9. https://doi.org/10.1016/j.jpain.2014.08.002.
40. Atef A, El-Tantawy A, Gad H, Hefeda M. Prevalence of associated injuries after anterior shoulder dislocation: a prospective study. Int Orthop. 2016;40(3):519–24. https://doi.org/10.1007/s00264-015-2862-z.
41. Dickens JF, Owens BD, Cameron KL, et al. Return to play and recurrent instability after in-season anterior shoulder instability: a prospective multicenter study. Am J Sports Med. 2014;42(12):2842–50. https://doi.org/10.1177/0363546514553181.
42. Boffano M, Mortera S, Piana R. Management of the first episode of traumatic shoulder dislocation. EFORT Open Rev. 2017;2:35–40. https://doi.org/10.1302/2058-5241.2.160018.
43. Lädermann A, Denard PJ, Tirefort J, et al. Does surgery for instability of the shoulder truly stabilize the glenohumeral joint?: a prospective comparative cohort study. Medicine (Baltimore). 2016;95(31):e4369. https://doi.org/10.1097/MD.0000000000004369.
44. Yamamoto N, Kijima H, Nagamoto H, et al. Outcome of Bankart repair in contact versus non-contact athletes. Orthop Traumatol Surg Res. 2015;101(4):415–9. https://doi.org/10.1016/j.otsr.2015.03.008.
45. Wasserstein DN, Sheth U, Colbenson K, et al. The true recurrence rate and factors predicting recurrent instability after nonsurgical management of traumatic primary anterior shoulder dislocation: a systematic review. Arthroscopy. 2016;32(12):2616–25. https://doi.org/10.1016/j.arthro.2016.05.039.
46. Burns TC, Owens BD. Management of shoulder instability in in-season athletes. Phys Sportsmed. 2010;38(3):55–60. https://doi.org/10.3810/psm.2010.10.1808.
47. Buss DD, Lynch GP, Meyer CP, Huber SM, Freehill MQ. Nonoperative management for in-season athletes with anterior shoulder instability. Am J Sports Med. 2004;32(6):1430–3. https://doi.org/10.1177/0363546503262069.
48. Handoll HHG, Almaiyah MA, Rangan A. Surgical versus non-surgical treatment for acute anterior shoulder dislocation. Cochrane Database Syst Rev. 2004;1:CD004325. https://doi.org/10.1002/14651858.CD004325.pub2.
49. Harris JD, Gupta AK, Mall NA, et al. Long-term outcomes after Bankart shoulder stabilization. J Arthrosc Relat Surg. 2013;29(5):920–33. https://doi.org/10.1016/j.arthro.2012.11.010.
50. Larrain MV, Montenegro HJ, Mauas DM, Collazo CC, Pavón F. Arthroscopic management of traumatic anterior shoulder instability in collision athletes: analysis of 204 cases with a 4- to 9-year follow-up and results with the suture anchor technique. J Arthrosc Relat Surg. 2006;22(12):1283–9. https://doi.org/10.1016/j.arthro.2006.07.052.
51. Bessiere C, Trojani C, Pélégri C, Carles M, Boileau P. Coracoid bone block versus arthroscopic Bankart repair: a comparative paired study with 5-year follow-up. Orthop Traumatol Surg Res. 2013;99(2):123–30. https://doi.org/10.1016/j.otsr.2012.12.010.
52. Stein T, Linke RD, Buckup J, et al. Shoulder sport-specific impairments after arthroscopic Bankart repair: a prospective longitudinal assessment. Am J Sports Med. 2011;39(11):2404–14. https://doi.org/10.1177/0363546511417407.
53. Petrera M, Dwyer T, Tsuji MRS, Theodoropoulos JS. Outcomes of arthroscopic Bankart repair in collision versus noncollision athletes. Orthopedics. 2013;36(5):e621–6. https://doi.org/10.3928/01477447-20130426-25.
54. An VVG, Sivakumar BS, Phan K, Trantalis J. A systematic review and meta-analysis of clinical and patient-reported outcomes following two procedures for recurrent traumatic anterior instability of the shoulder: Latarjet procedure vs. Bankart repair. J Shoulder Elb Surg. 2016;25(5):853–63. https://doi.org/10.1016/j.jse.2015.11.001.
55. Sugaya H, Moriishi J, Dohi M, Kon Y, Tsuchiya A. Glenoid rim morphology in recurrent anterior glenohumeral instability. J Bone Joint Surg Am. 2003;85-A(5):878–84.
56. Beran MC, Donaldson CT, Bishop JY. Treatment of chronic glenoid defects in the setting of recurrent anterior shoulder instability: a systematic review. J Shoulder Elb Surg. 2010;19(5):769–80. https://doi.org/10.1016/j.jse.2010.01.011.

57. Bhatia S, Saigal A, Frank RM, et al. Glenoid diameter is an inaccurate method for percent glenoid bone loss quantification: analysis and techniques for improved accuracy. Arthroscopy. 2015;31(4):608–614.e1. https://doi.org/10.1016/j.arthro.2015.02.020.
58. Bacilla P, Field LD, Savoie FH. Arthroscopic Bankart repair in a high demand patient population. Arthroscopy. 1997;13(1):51–60.
59. Pagnani MJ, Dome DC. Surgical treatment of traumatic anterior shoulder instability in American football players. J Bone Joint Surg Am. 2002;84-A(5):711–5.
60. O'Neill DB. Arthroscopic Bankart repair of anterior detachments of the glenoid labrum. A prospective study. J Bone Joint Surg Am. 1999;81(10):1357–66.
61. Voos JE, Livermore RW, Feeley BT, et al. Prospective evaluation of arthroscopic Bankart repairs for anterior instability. Am J Sports Med. 2010;38(2):302–7. https://doi.org/10.1177/0363546509348049.
62. Berendes TD, Pilot P, Nagels J, Vochteloo AJH, Nelissen RGHH. Survey on the management of acute first-time anterior shoulder dislocation amongst Dutch public hospitals. Arch Orthop Trauma Surg. 2015;135(4):447–54. https://doi.org/10.1007/s00402-015-2156-3.
63. Shin S-J, Koh YW, Bui C, et al. What is the critical value of glenoid bone loss at which soft tissue Bankart repair does not restore glenohumeral translation, restricts range of motion, and leads to abnormal humeral head position? Am J Sports Med. 2016;44(11):2784–91. https://doi.org/10.1177/0363546516656367.
64. Dines JS, Dodson CC, McGarry MH, JH O, Altchek DW, Lee TQ. Contribution of osseous and muscular stabilizing effects with the Latarjet procedure for anterior instability without glenoid bone loss. J Shoulder Elb Surg. 2013;22(12):1689–94. https://doi.org/10.1016/j.jse.2013.02.014.
65. Di Giacomo G, de Gasperis N, Costantini A, De Vita A, Beccaglia MAR, Pouliart N. Does the presence of glenoid bone loss influence coracoid bone graft osteolysis after the Latarjet procedure? A computed tomography scan study in 2 groups of patients with and without glenoid bone loss. J Shoulder Elb Surg. 2014;23(4):514–8. https://doi.org/10.1016/j.jse.2013.10.005.
66. Blonna D, Bellato E, Caranzano F, Assom M, Rossi R, Castoldi F. Arthroscopic Bankart repair versus open bristow-latarjet for shoulder instability: a matched-pair multicenter study focused on return to sport. Am J Sports Med. 2016;44(12):3198–205. https://doi.org/10.1177/0363546516658037.
67. Zarezade A, Dehghani M, Rozati AR, Banadaki HS, Shekarchizade N. Comparison of Bristow procedure and Bankart arthroscopic method as the treatment of recurrent shoulder instability. Adv Biomed Res. 2014;12(3):256.
68. Aydin A, Usta M, Topal M, Uymur EY, Tuncer K, Karsan O. Comparison of open Bankart repair versus modified Bristow operation for the treatment of traumatic recurrent anterior dislocation and capsular laxity of the shoulder. Eurasian J Med. 2012;44(3):157–62. https://doi.org/10.5152/eajm.2012.37.
69. Russo R, Della Rotonda G, Cautiero F, et al. Arthroscopic Bankart repair associated with subscapularis augmentation (ASA) versus open Latarjet to treat recurrent anterior shoulder instability with moderate glenoid bone loss: clinical comparison of two series. Musculoskelet Surg. 2016;101(1):75–83. https://doi.org/10.1007/s12306-016-0446-8.
70. Hovelius L. Incidence of shoulder dislocation in Sweden. Clin Orthop Relat Res. 1982;166:127–31.
71. Nordqvist A, Petersson CJ. Incidence and causes of shoulder girdle injuries in an urban population. J Should Elb Surg. 1995;4(2):107–12.
72. te Slaa RL, Brand R, Marti RK. A prospective arthroscopic study of acute first-time anterior shoulder dislocation in the young: a five-year follow-up study. J Shoulder Elb Surg. 2003;12(6):529–34. https://doi.org/10.1016/S1058274603002180.
73. Burkhart SS, de Beer JF. 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. 2000;16(7):677–94. https://doi.org/10.1053/jars.2000.17715.
74. Itoi E, Lee SB, Berglund LJ, Berge LL, An KN. The effect of a glenoid defect on anteroinferior stability of the shoulder after Bankart repair: a cadaveric study. J Bone Joint Surg Am. 2000;82(1):35–46.
75. van der Linde JA, van Kampen DA, Terwee CB, Dijksman LM, KleinJan G, Willems WJ. Long-term results after arthroscopic shoulder stabilization using suture anchors: an 8- to 10-year follow-up. Am J Sports Med. 2011;39(11):2396–403. https://doi.org/10.1177/0363546511415657.
76. Bigliani LU, Newton PM, Steinmann SP, Connor PM, Mcllveen SJ. Glenoid rim lesions associated with recurrent anterior dislocation of the shoulder. Am J Sports Med. 1998;26(1):41–5.
77. Milano G, Grasso A, Russo A, et al. Analysis of risk factors for glenoid bone defect in anterior shoulder instability. Am J Sports Med. 2011;39(9):1870–6. https://doi.org/10.1177/0363546511411699.
78. Cho SH, Cho NS, Rhee YG. Preoperative analysis of the Hill-Sachs lesion in anterior shoulder instability: how to predict engagement of the lesion. Am J Sports Med. 2011;39(11):2389–95. https://doi.org/10.1177/0363546511398644.
79. Boileau P, Villalba M, Héry J-Y, Balg F, Ahrens P, Neyton L. Risk factors for recurrence of shoulder instability after arthroscopic Bankart repair. J Bone Jt Surg. 2006;88(8):1755. https://doi.org/10.2106/JBJS.E.00817.
80. Bak K, Wiesler ER, Poehling GG, ISAKOS Upper Extremity Committee. Consensus statement on

shoulder instability. Arthroscopy. 2010;26(2):249–55. https://doi.org/10.1016/j.arthro.2009.06.022.

81. Yamamoto N, Itoi E, Abe H, et al. Contact between the glenoid and the humeral head in abduction, external rotation, and horizontal extension: a new concept of glenoid track. J Shoulder Elb Surg. 2007;16(5):649–56. https://doi.org/10.1016/j.jse.2006.12.012.
82. Di Giacomo G, Itoi E, Burkhart SS. Evolving concept of bipolar bone loss and the Hill-Sachs lesion: from 'engaging/non-engaging' lesion to 'on-track/off-track' lesion. Arthroscopy. 2014;30(1):90–8. https://doi.org/10.1016/j.arthro.2013.10.004.
83. Chen AL, Hunt SA, Hawkins RJ, Zuckerman JD. Management of bone loss associated with recurrent anterior glenohumeral instability. Am J Sports Med. 2005;33(6):912–25. https://doi.org/10.1177/0363546505277074.
84. Chapovsky F, Kelly JD. Osteochondral allograft transplantation for treatment of glenohumeral instability. Arthrosc J Arthrosc Relat Surg. 2005;21(8):1007.e1–4. https://doi.org/10.1016/j.arthro.2005.04.005.
85. Shah AS, Karadsheh MS, Sekiya JK. Failure of operative treatment for glenohumeral instability: etiology and management. Arthroscopy. 2011;27(5):681–94. https://doi.org/10.1016/j.arthro.2010.11.057.
86. Purchase RJ, Wolf EM, Hobgood ER, Pollock ME, Smalley CC. Hill-Sachs "Remplissage": an arthroscopic solution for the engaging Hill-Sachs lesion. J Arthrosc Relat Surg. 2008;24(6):723–6. https://doi.org/10.1016/j.arthro.2008.03.015.
87. Allain J, Goutallier D, Glorion C. Long-term results of the Latarjet procedure for the treatment of anterior instability of the shoulder. J Bone Joint Surg Am. 1998;80(6):841–52. https://doi.org/10.1016/j.jse.2014.02.015.
88. Hovelius L, Vikerfors O, Olofsson A, Svensson O, Rahme H. Bristow-Latarjet and Bankart: a comparative study of shoulder stabilization in 185 shoulders during a seventeen-year follow-up. J Shoulder Elb Surg. 2011;20(7):1095–101. https://doi.org/10.1016/j.jse.2011.02.005.
89. Rerko MA, Pan X, Donaldson C, Jones GL, Bishop JY. Comparison of various imaging techniques to quantify glenoid bone loss in shoulder instability. J Shoulder Elb Surg. 2013;22(4):528–34. https://doi.org/10.1016/j.jse.2012.05.034.
90. Markenstein JE, Jaspars KCCJ, van der Hulst VPM, Willems WJ. The quantification of glenoid bone loss in anterior shoulder instability; MR-arthro compared to 3D-CT. Skelet Radiol. 2014;43(4):475–83. https://doi.org/10.1007/s00256-013-1780-7.
91. Huijsmans PE, Haen PS, Kidd M, Dhert WJ, van der Hulst VPM, Willems WJ. Quantification of a glenoid defect with three-dimensional computed tomography and magnetic resonance imaging: a cadaveric study. J Shoulder Elb Surg. 2007;16(6):803–9. https://doi.org/10.1016/j.jse.2007.02.115.
92. Kaar SG, Fening SD, Jones MH, Colbrunn RW, Miniaci A. Effect of humeral head defect size on glenohumeral stability: a cadaveric study of simulated Hill-Sachs defects. Am J Sports Med. 2010;38(3):594–9. https://doi.org/10.1177/0363546509350295.
93. Moroder P, Resch H, Schnaitmann S, Hoffelner T, Tauber M. The importance of CT for the preoperative surgical planning in recurrent anterior shoulder instability. Arch Orthop Trauma Surg. 2013;133(2):219–26. https://doi.org/10.1007/s00402-012-1656-7.
94. Itoi E, Lee S-B, Amrami KK, Wenger DE, An K-N. Quantitative assessment of classic anteroinferior bony Bankart lesions by radiography and computed tomography. Am J Sports Med. 2003;31(1):112–8. https://doi.org/10.1177/03635465030310010301.
95. Baudi P, Righi P, Bolognesi D, et al. How to identify and calculate glenoid bone deficit. Chir Organi Mov. 2005;90(2):145–52.
96. Porcellini G, Campi F, Pegreffi F, Castagna A, Paladini P. Predisposing factors for recurrent shoulder dislocation after arthroscopic treatment. J Bone Jt Surg. 2009;91(11):2537–42. https://doi.org/10.2106/JBJS.H.01126.
97. Boone JL, Arciero RA. Management of failed instability surgery: how to get it right the next time. Orthop Clin North Am. 2010;41(3):367–79. https://doi.org/10.1016/j.ocl.2010.02.009.
98. Arce G, Arcuri F, Ferro D, Pereira E. Is selective arthroscopic revision beneficial for treating recurrent anterior shoulder instability? Clin Orthop Relat Res. 2012;470(4):965–71. https://doi.org/10.1007/s11999-011-2001-0.
99. Kim S-H, Ha K-I, Kim Y-M. Arthroscopic revision Bankart repair. Arthrosc J Arthrosc Relat Surg. 2002;18(5):469–82. https://doi.org/10.1053/jars.2002.32230.
100. Cho NS, Yi JW, Lee BG, Rhee YG. Revision open Bankart surgery after arthroscopic repair for traumatic anterior shoulder instability. Am J Sports Med. 2009;37(11):2158–64. https://doi.org/10.1177/0363546509339015.
101. Boileau P, Richou J, Lisai A, Chuinard C, Bicknell RT. The role of arthroscopy in revision of failed open anterior stabilization of the shoulder. Arthroscopy. 2009;25(10):1075–84. https://doi.org/10.1016/j.arthro.2009.04.073.
102. Patel RV, Apostle K, Leith JM, Regan WD. Revision arthroscopic capsulolabral reconstruction for recurrent instability of the shoulder. J Bone Jt Surg (Br). 2008;90-B(11):1462–7. https://doi.org/10.1302/0301-620X.90B11.21072.
103. Bartl C, Schumann K, Paul J, Vogt S, Imhoff AB. Arthroscopic capsulolabral revision repair for recurrent anterior shoulder instability. Am J Sports Med. 2011;39(3):511–8. https://doi.org/10.1177/0363546510388909.

104. Sisto DJ. Revision of failed arthroscopic Bankart repairs. Am J Sports Med. 2007;35(4):537–41. https://doi.org/10.1177/0363546506296520.
105. Krueger D, Kraus N, Pauly S, Chen J, Scheibel M. Subjective and objective outcome after revision arthroscopic stabilization for recurrent anterior instability versus initial shoulder stabilization. Am J Sports Med. 2011;39(1):71–7. https://doi.org/10.1177/0363546510379336.
106. Ilahi OA, Al-Fahl T, Bahrani H, Luo Z-P. Glenoid suture anchor fixation strength: effect of insertion angle. Arthroscopy. 2004;20(6):609–13.
107. Kamath GV, Hoover S, Creighton RA, Weinhold P, Barrow A, Spang JT. Biomechanical analysis of a double-loaded glenoid anchor configuration: can fewer anchors provide equivalent fixation? Am J Sports Med. 2013;41(1):163–8. https://doi.org/10.1177/0363546512469090.
108. Piasecki DP, Verma NN, Romeo AA, Levine WN, Bach BR, Provencher MT. Glenoid bone deficiency in recurrent anterior shoulder instability: diagnosis and management. J Am Acad Orthop Surg. 2009;17(8):482–93.
109. Ghodadra N, Gupta A, Romeo AA, et al. Normalization of glenohumeral articular contact pressures after latarjet or iliac crest bone-grafting. J Bone Jt Surg. 2010;92(6):1478–89. https://doi.org/10.2106/JBJS.I.00220.
110. Burkhart SS, De Beer JF, Barth JRH, et al. Results of modified Latarjet reconstruction in patients with anteroinferior instability and significant bone loss. Arthroscopy. 2007;23(10):1033–41. https://doi.org/10.1016/j.arthro.2007.08.009.
111. Helfet AJ. Coracoid transplantation for recurring dislocation of the shoulder. J Bone Joint Surg (Br). 1958;40-B(2):198–202.
112. Neyton L, Young A, Dawidziak B, et al. Surgical treatment of anterior instability in rugby union players: clinical and radiographic results of the Latarjet-Patte procedure with minimum 5-year follow-up. J Shoulder Elb Surg. 2012;21(12):1721–7. https://doi.org/10.1016/j.jse.2012.01.023.
113. Butt U, Charalambous CP. Complications associated with open coracoid transfer procedures for shoulder instability. J Shoulder Elb Surg. 2012;21(8):1110–9. https://doi.org/10.1016/j.jse.2012.02.008.
114. Shah AA, Butler RB, Romanowski J, Goel D, Karadagli D, Warner JJ. Short-term complications of the latarjet procedure. J Bone Jt Surg. 2012;94(6):495–501. https://doi.org/10.2106/JBJS.J.01830.
115. Skogland LB, Sundt P. Recurrent anterior dislocation of the shoulder. The Eden-Hybbinette operation. Acta Orthop Scand. 1973;44(6):739–47.
116. Kraus N, Amphansap T, Gerhardt C, Scheibel M. Arthroscopic anatomic glenoid reconstruction using an autologous iliac crest bone grafting technique. J Shoulder Elb Surg. 2014;23(11):1700–8. https://doi.org/10.1016/j.jse.2014.03.004.
117. Verborgt O, Van den Bogaert G, Debeer P. Arthroscopic bone block augmentation of the anterior glenoid through the rotator interval: a cadaveric feasibility study. J Shoulder Elb Surg. 2013;22(4):458–62. https://doi.org/10.1016/j.jse.2012.04.017.
118. Provencher MT, Ghodadra N, LeClere L, Solomon DJ, Romeo AA. Anatomic osteochondral glenoid reconstruction for recurrent glenohumeral instability with glenoid deficiency using a distal tibia allograft. J Arthrosc Relat Surg. 2009;25(4):446–52. https://doi.org/10.1016/j.arthro.2008.10.017.
119. Burkhart SS, Debeer JF, Tehrany AM, Parten PM. Quantifying glenoid bone loss arthroscopically in shoulder instability. Arthroscopy. 2002;18(5):488–91. https://doi.org/10.1053/jars.2002.32212.
120. Kropf EJ, Sekiya JK. Osteoarticular allograft transplantation for large humeral head defects in glenohumeral instability. Arthrosc J Arthrosc Relat Surg. 2007;23(3):322.e1–5. https://doi.org/10.1016/j.arthro.2006.07.032.

Pediatric ACL Injuries: Treatment and Challenges

17

Rob Janssen, Martin Lind, Lars Engebretsen,
Håvard Moksnes, Romain Seil, Peter Faunø,
and Martha Murray

17.1 Introduction (Lars Engebretsen)

The number of publications on treatment of anterior cruciate ligament (ACL) injuries in the skeletally immature population has increased through the past decade [1–6]. However, opinions on whether pediatric ACL injuries should primarily be surgically reconstructed or conservatively treated are still divided within the pediatric orthopedic community [7, 8]. Evidence from high-level studies and randomized controlled trials is lacking [9], which leaves the field open for various treatment algorithms due to the lack of a

---

R. Janssen (✉)
Department of Orthopedic Surgery and Trauma,
Máxima Medical Center,
Eindhoven-Veldhoven, The Netherlands

Orthopedic and Sports Medicine Knee Research
Center, Orthopedic Associates Eindhoven
Greater Area, Eindhoven, The Netherlands
e-mail: rpajanssen@onsbrabantnet.nl

M. Lind • P. Faunø
Division of Sports Traumatology,
Department of Orthopedics,
Aarhus University Hospital,
Aarhus, Denmark

L. Engebretsen
Department of Orthopedic Surgery,
Oslo University Hospital,
University of Oslo, Oslo, Norway

Oslo Sports Trauma Research Center,
Oslo, Norway

International Olympic Committee (IOC),
Oslo, Norway

H. Moksnes
Oslo Sports Trauma Research Center,
The Norwegian School of Sport Sciences,
Oslo, Norway

The Norwegian Olympic Training Center
(Olympiatoppen), Oslo, Norway

The Norwegian Sports Medicine Center
(Idrettens Helsesenter), Oslo, Norway

R. Seil
Department of the Locomotor System, Centre
Hospitalier de Luxembourg, Luxembourg, Luxemburg

Sports Medicine Research Laboratory, Luxembourg
Institute of Health, Luxembourg, Luxemburg

M. Murray
Orthopedic Surgery, Harvard Medical School,
Boston, MA, USA

Orthopedic Surgeon, Boston Children's Hospital,
Boston, MA, USA

G.M.M.J. Kerkhoffs et al. (eds.), *ESSKA Instructional Course Lecture Book*,
https://doi.org/10.1007/978-3-662-56127-0_17

solid scientific knowledge base. Risk factors for ACL injuries in skeletally immature patients are unknown, although it seems that boys may be more prone to rupturing their ACL before skeletal maturity, while girls have an increased risk through and after puberty [10, 11]. Many authors argue that the incidence of pediatric ACL injuries is rising. However, no epidemiological studies are available to support this statement. Increased awareness and advances in diagnostic methods, in addition to higher participation rates and earlier specialization in sports, may have led to an increase in the incidence of pediatric ACL tears.

An ACL tear in a child or an adolescent is considered as a serious injury to the knee, which cannot be repaired back to normal. Pediatric ACL tears are rare, accounting for less than 5% of all ACL injuries, and rarely occur under the age of 9. The current evidence for treatment is low [9]. Thirty years ago, the problem was mainly managed nonoperatively or by suture repair, which frequently resulted in unsuccessful outcomes. At that time, the risk of pediatric ACL reconstruction had not been deeply evaluated, and the diagnostic possibilities were inferior to current standards. Therefore, many of these injuries were diagnosed late, and orthopedic surgeons were often confronted with a negative selection of ACL-injured children, presenting with secondary meniscus tears and cartilage lesions. Nowadays, we have learned that pediatric ACL reconstruction is a safe procedure with low complication rates, provided that surgery is performed correctly. This does not mean that all pediatric ACL reconstructions should be treated with surgery.

Pediatric ACL tears have a high impact on physical activity, with a significant risk of developing early posttraumatic osteoarthritis. In recent years, studies have emphasized the importance of prevention [12], and there are also studies showing that a high activity level may be kept with correct training without meniscal and cartilage injury in the first 2 years after the injury. However, osteoarthritis may be manifested as early as the age of 30–40 years with a higher risk in case of concomitant meniscal—or cartilage—damage. Clinical studies found concomitant injuries in as high as 50–65% of ACL tears in this young age group [13–15], although the quality of these studies can be questioned. Consequently, the primary objective must be to protect kids and adolescents from this potentially severe injury. Teachers at school and sports clubs have to be educated.

An acute ACL tear usually leads to immediate cessation of sports. The injured child and his/her parents often feel insecure about the situation and are afraid of permanent damage. To put their mind on ease, it is very important to spend time for consulting. The specialist physician has to check for additional injuries and has to establish an exact diagnosis. Therefore, a gentle examination and MRI are necessary. A decision for conservative or surgical treatment can then be taken. The final treatment decision should be made in discussion with the child and the family after a proper rehabilitation program has been undertaken for at least 6 months.

The rate of subsequent meniscal injury in this young group of active patients is not clear. Damage to the meniscus or cartilage may be as devastating as the ACL injury. Therefore, there is an international consensus that, in case of concomitant damage to the meniscus (bucket-handle type of injury) and/or persistent knee instability, an ACL reconstruction should be performed. The goal is to stabilize the knee, improve its function, repair the meniscus, and protect the knee from future episodes of giving way and injuries [15].

As the participant will learn in this ICL, an ACL tear is a severe injury and is considered a permanent damage to the young knee joint. A stable knee is important to protect the meniscus from secondary injury and early osteoarthritis. Return to high-level cutting and pivoting sports bears a high risk of reinjury and additional damage. The child and the family must be made aware of the danger of pivoting sports, regardless of surgical or nonsurgical treatment. As mentioned above, current evidence for treatment of pediatric ACL injuries is low. Finding the right treatment for each child is a matter of balance, patience, and thorough follow-up.

In order to look for answers to some of the remaining open questions in pediatric ACL injuries, the ESSKA Foundation started the “Pediatric ACL Monitoring Initiative” (PAMI), a combined, multicenter project on these relatively rare lesions. It started with a survey among ESSKA members and has been published in KSSTA [12]. Current efforts will be presented at the ESSKA congress in Glasgow. This current ESSKA ICL will give a closer insight in the challenging topic regarding prevention, conservative and operative treatment, risk of growth disturbance, clinical results, outcome measures, and future research.

17.2 State-of-the-Art Treatment

17.2.1 Pediatric ACL Treatment Algorithms (Håvard Moksnes)

Opinions on whether pediatric ACL treatment should primarily be surgically reconstructed or conservatively treated are still divided within the pediatric orthopedic community [7, 8]. It is commonly believed that the incidence of pediatric ACL injuries is rising [2, 16–19]. However, only one study has documented an increase [20], and there are no other epidemiological studies available to support this assumption. Advances in diagnostic methods and increased awareness, in addition to earlier specialization in sports and higher numbers being active, may have led to an increase in the incidence of pediatric ACL tears.

Weighing the risks and benefits between primary surgical treatment and primary conservative treatment is crucial for every surgeon involved in pediatric ACL decision-making [21]. Most authors follow algorithms in which the inability to be active in preferred activities, or repetitive episodes of giving way despite undergoing rehabilitation, will point toward advising an ACL reconstruction before skeletal maturity. However, a substantial number of clinical commentaries and other expert opinion publications recommend early reconstruction [22–24]. The risk of sustaining a secondary meniscus injury can be increased with persistent instability in the ACL-deficient knee, although it is uncertain whether time from injury to surgical treatment is an independent risk factor [10, 25]. Additionally, children who have a secondary repairable meniscus injury will usually undergo a meniscus repair with concomitant ACL reconstruction, as this is assumed to improve the prognosis of the meniscus repair [26]. The backside of performing ACL reconstructions in skeletally immature patients, however, is the risk of provoking a growth disturbance following transepiphyseal drilling [27] and the unknown development of the graft with postoperative growth [28, 29]. The awareness of these risks is high among orthopedic surgeons, and presumed safer surgical techniques have been developed and described in recent years [30, 31]. Still, a recent survey among European surgeons suggests that the number of growth disturbances is significant [12], which is also supported by several case publications [32–34].

Scandinavian groups have been more prone to follow a primary nonoperative treatment algorithm in skeletally immature patients [21]. This algorithm highlights that post-injury rehabilitation must be performed exhaustively before further treatment decisions are taken. Further treatment decision-making is based on the functional knee stability experienced by the child in its desired activities and supported by functional performance tests [21]. The adult ACL literature supports that supervised rehabilitation should be performed before a decision on further treatment is made for any ACL patient [35, 36]. Preoperative rehabilitation is beneficial because it increases the likelihood of a successful outcome after ACL reconstruction and is in many cases effective in restoring functional knee stability to a level that eliminates the need for a surgical ACL reconstruction [37, 38]. Children with ACL injuries should be monitored regularly and assessed by an orthopedic surgeon and a physical therapist in collaboration. This teamwork, supplemented by objective functional tests, secures that a structured rehabilitation program has been exhausted.

A prospective cohort study on 46 skeletally immature children has described that two out of three children were able to continue their activities for at least 2 years without suffering of instability or secondary injuries that required surgical treatment [6, 39]. For many children, 2 years of conservative treatment may be sufficient to avoid the risk of growth disturbances following skeletally immature procedures. To date, this study is the only prospective study on conservative management of ACL-injured children. Furthermore, three out of four studies on secondary meniscus injuries show numbers that are comparable to primary surgical treatment [15, 40, 41]. However, caution must be taken with regard to the long-term results. Likewise, there is a need for larger and prospective studies on different treatment algorithms in this population. The Pediatric Anterior Cruciate Ligament Monitoring Initiative (PAMI) has, under the umbrella of ESSKA, conducted a survey among orthopedic surgeons [12]. The survey describes that the number of pediatric ACL reconstructions in Europe is high and that there is a huge variance in both indications for surgery and preferred surgical techniques. Subsequent to this survey, the PAMI group has started the development of a multinational pediatric ACL registry. The registry is scheduled to be operative late in 2017 and will be a unique possibility to collect long-term prospective data that is much needed for the pediatric ACL community.

Conservative treatment and prevention of pediatric ACL patients were recently described by Moksnes and Grindem in the ESSKA journal [42]. Pediatric rehabilitation has to be performed in close collaboration between the parents, an experienced physiotherapist, and the orthopedic surgeon. The child and parents should consult their physical therapist regularly. A normal setup could be once a week throughout phase 1, every second week through phase 2, and once a month in phase 3. Rehabilitation is normally designed to enable home-based exercises, and it is recommended to limit the number of exercises to enhance the feasibility and adherence to the program [43]. Exercises and goals have to be adjusted compared to traditional adult rehabilitation protocols because children cannot be expected to perform unsupervised gym-based programs. Rehabilitation exercises are less focused on muscular strength and hypertrophy compared to adult rehabilitation. Thus, the primary focus should be neuromuscular stimulation and development of multi-joint functional stability [21, 44]. Pediatric ACL rehabilitation is progressed through the phases based on clinical reasoning, sequential functional achievements, and the achievement of functional milestones (Fig. 17.1).

Some milestones will be primary in each phase, for example, achieving full knee extension and quadriceps activation early after the knee injury in phase 1. Throughout the first two phases, the child should be guarded from pivoting activities, and possibly also wear a protective brace in school and training. Exercises to facilitate proper alignment and adequate landing techniques have been successfully implemented in injury prevention programs [45–47] and are also recommended through phase two and three of pediatric ACL rehabilitation. After completion of the rehabilitation milestones and passing functional test criteria (Fig. 17.2), the children usually return to their desired activities.

However, some children will not achieve a functionally stable knee, even with exhaustive rehabilitation. Children with persistent giving way episodes during, or after, nonoperative treatment should be counseled for a change in activity level and/or considered for surgical treatment. Additionally, an orthopedic surgeon must assess children with a very high demanding sports activity, symptomatic meniscus injuries, recurrent effusions, and/or restricted ROM for surgical treatment. A selection of neuromuscular exercises focusing on maintaining functional stability should be encouraged as a secondary prevention measure. Ideally, these exercises should be performed as part of their team warm-up routine before practice, which has been shown to be effective in preventing lower extremity injury rates by as much as 50% [48–50]. Several online resources are freely available such as the “Get Set—Train Smarter” app and the website https://hub.olympic.org/library/injury-prevention/. Additionally, it is imperative that the child and parents are provided with thorough information on the benefits and risks involved with both surgical and conservative treatment. Particularly, the option of continuing sports involving less pivoting actions until skeletal maturity is reached, when a reconstruction involving less risk can be performed.

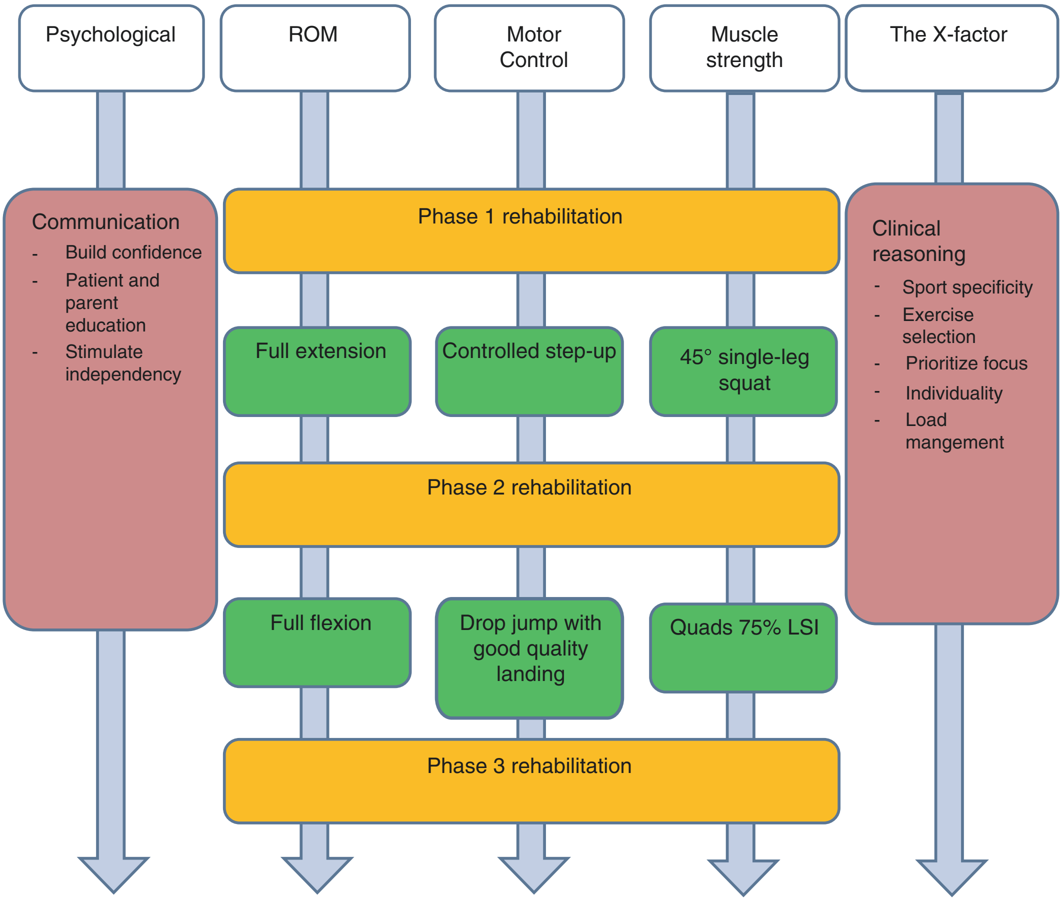


Fig. 17.1 Functional treatment algorithm with examples of rehabilitation milestones

Return to Sport criteria

Muscle strength measurements > 90%

Hop tests > 90%

With adequate strategy and quality

Performed gradual increase in sport specific training with adequate movement patterns and without pain, effusion or fear of re-injury

Fig. 17.2 Functional test criteria

17.2.2 Surgical Treatment Options in ACL Surgery with Open Physes (Romain Seil)

In the past, ACL injuries with open physes were mainly managed nonoperatively or by suture repair, which too frequently resulted in unsuccessful outcomes [15, 51]. At that time, the risk of pediatric knee ligament reconstruction had not been deeply evaluated yet, and the diagnostic possibilities were inferior to current standards. Therefore, many of these injuries were diagnosed late, and orthopedic surgeons were often confronted to a negative selection of ACL-injured children, presenting with secondary meniscus tears and cartilage lesions. Nowadays, it has been established that pediatric ACL reconstruction is a safe procedure with low complication rates, provided that surgery is performed correctly. In return, this does not mean that all pediatric ACL injuries should be treated operatively [9, 52].

In case of associated damage to the meniscus (i.e., bucket-handle tear, meniscal ramp lesion) and/or persistent knee instability, an ACL

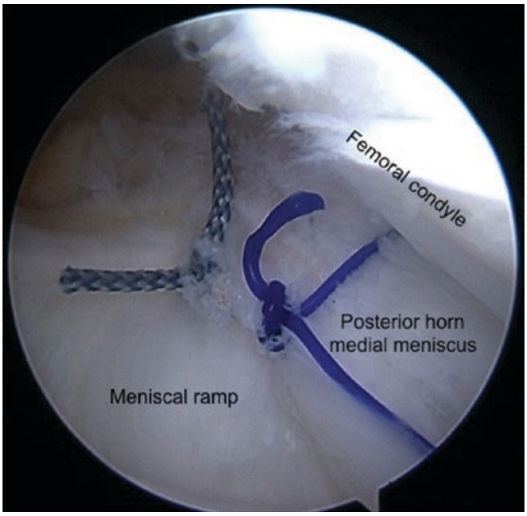


Fig. 17.3 Posteromedial view showing a repaired medial meniscus ramp lesion in a 12-year-old boy (typically the type of meniscus lesion which can be overlooked in children with ACL injury)

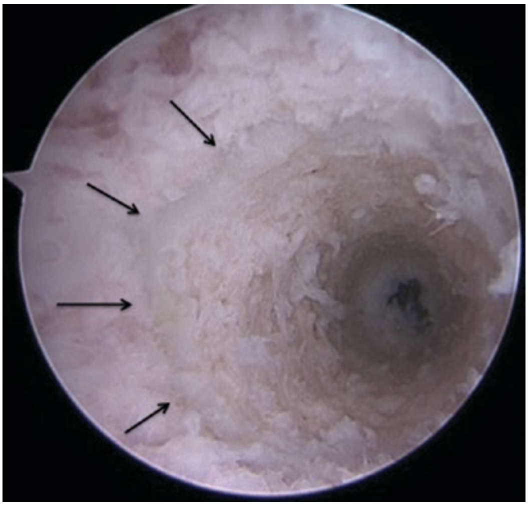


Fig. 17.4 Intraoperative view of the femoral tunnel in a transphyseal ACL reconstruction technique in the left knee of a 12-year-old boy. The growth plate is marked with arrows

reconstruction should be performed [53]. The goal is to stabilize the knee, improve its function, repair the meniscus, and protect the knee from future episodes of giving way and meniscus injuries (Fig. 17.3) [15, 51].

Many surgical techniques have been described in order to perform the best possible ACL replacement in children and, at the same time, reduce the surgically induced complication potential to a minimum. In contrast to the adult knee, an anatomic graft placement is difficult to obtain in children with the currently available techniques [54]. This is due to the presence of the growth plates, especially on the femoral side. According to the localization of the tibial and femoral tunnels, surgical techniques can be divided into three categories: transphyseal procedures, where the tunnels are drilled through the growth plates (Fig. 17.4); epiphyseal techniques, where the tunnels are located in the tibia and femoral epiphysis, not injuring the growth plate; and finally extra-epiphyseal techniques, where the graft is placed around the growth plate (Fig. 17.5).

A different type of graft placement can be chosen, either on the tibial or on the femoral side. Some authors differentiate their specific pediatric ACL reconstruction technique according to the amount of knee growth remaining [55]. In the rare advent of ACL injuries in very young children with a very high potential of remaining growth, some authors prefer using extra-epiphyseal techniques, whereas they use rather transphyseal options when knees are closer to growth plate fusion. However, this strategy is controversial and not based on evidence [56].

The most accepted and internationally widespread technique in case of open physis is an ACL reconstruction with a soft tissue graft, usually autologous doubled semitendinosus and gracilis tendons [1, 17, 57]. If hamstrings are used, it is important not to harm their periosteal attachment on the tibia to avoid fusion of the tibial tuberosity apophysis and subsequent development of a recurvatum knee [33, 58]. Graft diameter generally varies between 6 and 8 mm, but it may be thinner in some patients. Therefore, the surgeon should be prepared to change his graft harvesting strategy intraoperatively. Tunnel diameter should always be kept under 9 mm [59]. On the tibial side, care must be taken to position the tunnel entrance more medially than in adults, in order to protect the apophysis of the tibial tuberosity and to avoid subsequent development of a varus and/or a recurvatum knee [60]. On the

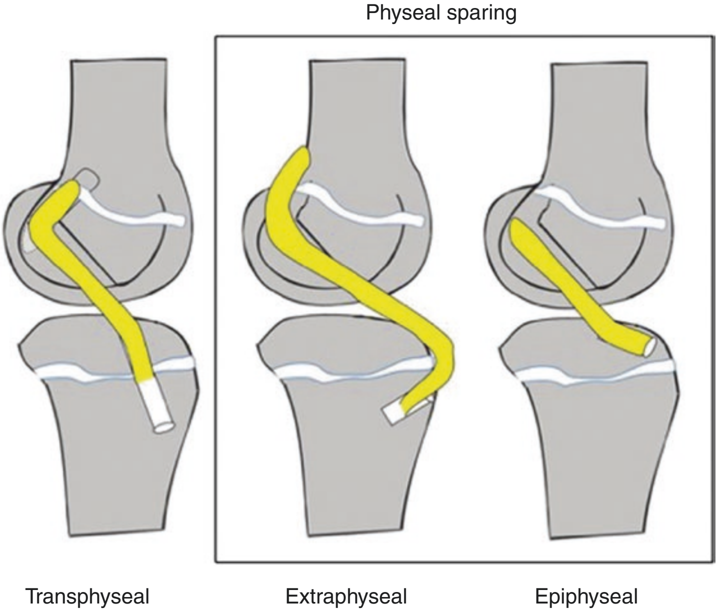


Fig. 17.5 Transphyseal and physeal-sparing ACL reconstruction techniques

femoral side, an injury to the perichondral structures should be avoided to prevent a posterolaterally located growth plate fusion and the iatrogenic development of a valgus knee [61]. Recently, the use of living donor allografts has been advocated [3], but this technique is controversial for ethical reasons. It is well known that allografts have a higher potential for retears of the reconstructed ACL in adolescents and young adults. For this reason, we refrain from using allografts in a pediatric population. Bone plugs or fixation material should not cross the physis to minimize the risk of growth disturbances [58, 62]. The use of permanent artificial grafts is prohibited as it may cause significant growth arrest as well as the need for complex, three-dimensional corrective surgeries for malalignment or leg length discrepancies. Every surgical technique bears its own, specific complication potential [17, 27, 32, 33, 36, 56, 63–65], but if all precautions are taken [58], the risk of a growth disturbance is very low [56]. It may, however, be underreported [12]. These possible side effects and complications of surgery should be thoroughly discussed with the child's family. Clinical follow-up visits should be maintained until the end of the growth period [12].

17.2.3 Clinical Outcome in Pediatric ACL Reconstruction (Peter Faunø)

A significant number of anterior cruciate ligament (ACL) injuries are seen in children and adolescents [66]. According to the Danish Knee Ligament Reconstruction Registry (DKRR), 6% of all ACL reconstructions are performed in patients younger than 15 years [67].

There is no consensus regarding treatment of ACL injuries in children, leaving the treating physicians with a therapeutic dilemma. Should the patient be treated conservatively, which has been done with some success [39] to avoid any growth disturbances, or should the child be treated operatively with an ACL reconstruction, increasing the risk of growth disturbances around the knee [31, 39].

The chance of successful outcome after ACL reconstruction (ACLR) in children and adolescents is poorer than surgery in adults for several reasons. Most of the length growth in children occurs around the knee. This makes the growth plates susceptible to injury due to hardware crossing or heat induced by drilling, resulting in growth disturbances [68]. Furthermore, it is to be expected that compliance to postoperative rehabilitation is lower in children due to psychological immaturity, resulting in more frequent failed rehabilitation or an untimely return to sport. Last, but not least, the pediatric patient has parents who might have difficulties accepting the implications of a serious knee injury such as an ACL injury. The expectations of sports-ambitious parents may have an impact on the final outcome of ACL reconstruction.

17.2.3.1 Clinical Outcome

There are several case series describing the results after ACLR in children and adolescents. It is difficult to compare and compile the results of these studies, since the number of patients often is limited and the surgical techniques, as well as the ages of the subjects, differ between studies. The most recent studies (summarized in Table 17.1) show a high failure rate among children and adolescents.

In the large-scale register studies, particularly from Scandinavia, the high failure rate among children and adolescents is confirmed. In the age group of 13–15 years, a revision rate of 6.7% was found compared to the adult ACLR group with only 2% revisions (Fig. 17.6). This is contrasted by higher subjective outcome scores in the 13–15-year age group [74].

Table 17.1 Studies on ACL pediatric reconstructions and results

	Patient number	Follow-up years	Age of operated patients (age span)	IKDC (Lysholm)	Failure rate (%)
Kohl et al. [69]	28	2.8	13 (9–15)	94 (Lysholm)	7
Calvo et al. [1]	27	10.6	13 (12–16)	95	15
Schmale et al. [70]	29	4	14 (13–15)	91 (Lysholm)	14
Kohl et al. [69]	15	4.1	12.8 (6.2–15.8)	94 (Lysholm)	0
Demange and Camanho [2]	12	15	10.7 (8.3–12.4)	83	25
Koizumi et al. [71]	15	3.1	14 (13–16)	97	13
Kocher et al. [31]	44	5.3	14.7 (11.6–16.9)	90	5
Willimon et al. [72]	22	3	11.8 (9.9–14.0)	96	14
Ho et al. [73]	561	–	15.4 (5–19)	–	10

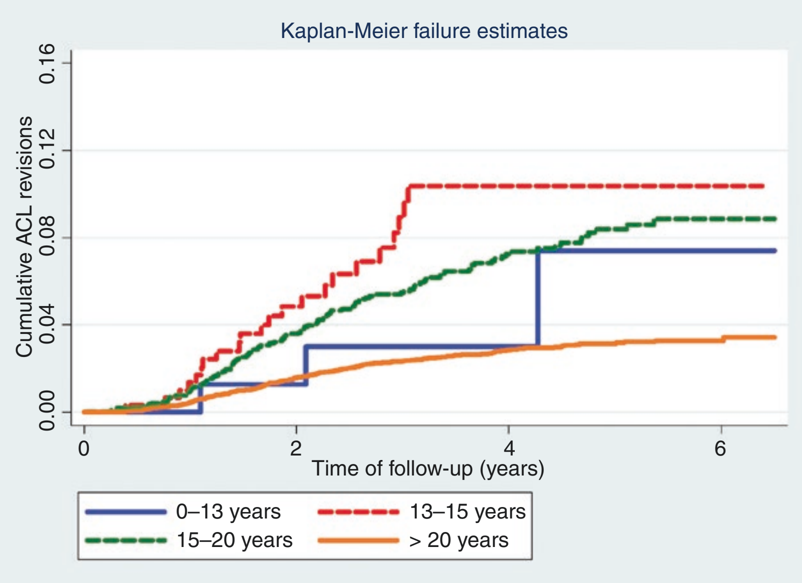


Fig. 17.6 Kaplan-Meier cumulative revision curve of primary ACL reconstructions in four different age groups. The highest revision age is seen in age group 13–15

The combination of age (13–19 years) and playing soccer increased the risk for an ACL revision even further up to three times compared to adult ACL reconstructed patients [75]. In a large retrospective study by Ho et al. [73], there was an average 9.6% failure rate. The authors also found twice the failure rate when a soft tissue graft was used compared to a patella tendon bone graft [73].

A systematic review comparing transphyseal (18 studies) and physeal-sparing (6 studies) techniques has failed to demonstrate any difference in clinical outcome such as growth disturbances or graft rerupture incidence [76]. The results of the pooled data were however weakened by lack of uniformity among the compared studies.

17.2.3.2 Growth Disturbances

There have been reports on growth disturbances after ACLR in the growing patient [17, 31, 77], which have lead authors to advocate a delay of ACLR until skeletal maturity [27, 41].

In a meta-analysis of children and adolescents, only 1.9% had leg length discrepancies after ACLR [17]. These results were, however, based on clinical judgment or standard radiographs, and the mean patient age was as high as 13 years. In the same study, it was shown that the use of hamstring grafts was less prone to induce growth disturbances compared to the use of patellar tendon graft. In a recent study of ACL transphyseal reconstructed knees, with a more accurate digital radiographic measurement in 33 patients with an index age of 11.7, it was found that 24% of the patients had more than 1 cm leg shortening compared to the unaffected leg. Mean femoral shortening in the operated leg was 3.5 mm compared to the nonoperated side. Furthermore, a significant increased femoral valgus deformity and an increased tibial varus deformity were seen [68].

Physeal-sparing ACLR techniques have evolved with the purpose of avoiding growth disturbances. However, a meta-analysis by Frosch et al. [17] showed a higher risk for growth disturbances among young patients operated with a physeal-sparing technique.

So far, there is no evidence that this approach has an advantage over the transphyseal technique with respect to limiting growth disturbances [76]. The current literature on ACLR in skeletal immature patients does not provide explicit recommendations whether a physeal-sparing technique is superior to a transphyseal technique when it comes to growth disturbances.

17.2.3.3 Concomitant Injuries

There is a high incidence of concomitant knee lesions in the ACL-injured child. In a large-scale register study from the United States, meniscal resection or meniscal repair was performed at time of ACLR in 39% and 28%, respectively, in patients aged 10–14 years [19]. The authors also found that some kind of cartilage procedure was done in 6% of the cases.

Several studies indicated that delayed ACLR increases the risk for concomitant lesions. Anderson et al. reviewed adolescents who underwent ACLR and found that those who waited more than 3 months after injury had a 2.8 times higher risk of lateral meniscal tear compared to those operated within 6–12 weeks [16]. Newman et al. [78] found that a delay to surgery correlated with increased severity of injury among both children and adolescents. A delay in surgery >3 months was the strongest predictor of the development of a concomitant injury among children [78]. In a review of long-term complications of delayed ACLR, Mansson et al. found that 55% of the patients had a meniscal tear at the time of ACLR if surgery was delayed by an average 11.6 months from the time of injury [79].

17.2.3.4 Summary

There is still no convincing evidence that demonstrates which technique should be used for ACL reconstruction in children and adolescents with respect to minimizing growth disturbances. Twenty-four percent of patients have >1 cm of growth reduction after ACLR, but only a small proportion has clinical symptoms of limp length discrepancy. Most studies report very good subjective and functional outcome after surgery, but there is an alarming high graft rupture rate up to 15% after 5 years in pediatric patients. Furthermore, a potential risk for physeal damage calls for good future studies.

17.2.4 Patient-Reported Outcome Measures (PROMs) in Children with ACL Injury (Rob Janssen)

PROMs are the patient's perceived health condition and treatment results [80]. Despite being developed for research purposes, clinicians use PROMs to enhance clinical management of individual patients [81]. PROMs offer specific benefits, since subjective assessment of reduction of symptoms and quality of life avoids observation bias [81].

There are two types of PROMs: disease-specific and generic PROMs [81]. Many disease-specific PROMs have been developed and focus on specific symptoms and impact on function of a specific condition [81]. The quality of PROMs is determined by three psychometric properties: reliability, validity, and responsiveness [80, 82–85]. Reliability refers to the reproducibility of an outcome measure between tests [80, 82]. Validity determines whether PROMs measure what they intend to measure [80, 82, 84]. Responsiveness assesses the ability of PROMs to detect changes in a patient's status over time or as a result of treatment [80, 82].

PROMs have become an important component to determine patient outcomes after sports-related knee injuries treated both surgically and nonsurgically [82–84, 86–89]. PROMs concerning musculoskeletal conditions have been validated in adult populations but are also being used for children [1, 80, 82–87, 89, 90]. Unfortunately, adult outcome measures are not necessarily appropriate or transferable to the pediatric population [82]. Furthermore, specific activity rating systems such as the Lysholm score and Tegner score have subset questions that measure work-related activities [89]. As such, they are less applicable to children.

17.2.4.1 Pedi-IKDC

Most research on PROMs in children and adolescents has focused on the Pediatric International Knee Documentation Committee (Pedi-IKDC) subjective knee evaluation form. The original adult version of the IKDC was developed and validated as a knee-specific, rather than a disease-specific, outcome instrument [87, 90, 91]. The Pedi-IKDC was developed in 2011 based on IKDC domains that children had difficulty in understanding [82, 86, 90, 91]. The 18 domains of the Pedi-IKDC are activity with pain, pain in the past 4 weeks, severity of pain, swelling, activity with swelling, catching, activity with giving way, highest activity, going upstairs, going downstairs, kneeling, squatting, sitting, rising, running, jumping, stopping, and overall knee function [82].

Nasreddine et al. determined normative Pedi-IKDC scores and found a strong association between Pedi-IKDC scores and prior knee surgery as well as recent activity limitations in the index knee [92]. The lack of a sex-based effect and the minor variation with age both simplify the interpretation and use of the Pedi-IKDC [92]. The authors concluded that Pedi-IKDC score distributions can be used to evaluate clinical outcomes as well as provide assumptions for use in sample size or power calculations for research [92]. Kocher et al. [82] demonstrated that the Pedi-IKDC has acceptable reliability, validity, and responsiveness as well as acceptable floor and ceiling effects if used as a complete instrument with an aggregate score [82, 91]. However, the high ceiling effects for the domains of catching, going upstairs, going downstairs, sitting, and rising do not allow reporting the patient's activity independently from within the score [82, 89]. Kocher et al. discussed that other PROMs may be more discriminating for these domains [82]. They recommend using Pedi-IKDC for knee injury outcome measurement in patients aged 10–18 years [82]. The Pedi-IKDC has been translated and validated in Danish and Dutch in children aged 10–15 years [90, 93]. A simple linear equation can convert Pedi-IKDC to adult IKDC allowing long-term tracking of patients [87].

The aforementioned authors did not validate the Pedi-IKDC in children younger than 10 years of age. A potential source of bias in young children is the fact that parents are often involved in explaining questions even though they are not allowed to answer for the patients [82, 84, 89]. One might argue that younger children might experience difficulty in understanding PROM questions and as a result have difficulty answering them. In terms of

PROMs, it is generally accepted that information provided by proxy respondents (parents) is not equivalent to patient self-report and that a parent report of function cannot be substituted for the child's report [84]. Schmitt et al. analyzed their data to find the lower age limit for which the self-reported Pedi-IKDC was still appropriate [84]. They concluded that the Pedi-IKDC is valid and consistent as PROM for symptoms, function, and sports activities for children and adolescents aged 6–18 years [84]. Further research on PROMs by very young children is still needed.

17.2.4.2 KOOS-Child

Örtqvist et al. have developed the Knee Injury and Osteoarthritis Outcome Score for Children (KOOS-Child) in 2012 [88]. In analogy to the IKDC, the authors showed that younger children (10–12 years) experienced difficulty answering the questions of the internationally validated KOOS score for adults [88]. The current KOOS-Child has 39 items divided in 5 domains: pain, other symptoms, activities of daily living, sports and recreation, and knee-related quality of life. The KOOS-Child is the first PROM for children with knee disorders designed to evaluate self-reported knee function that includes separate sports-related and knee-related quality of life subscales [83]. It is validated in Swedish, Danish, Dutch, and English [83, 93]. KOOS-Child is designed to assess and monitor groups and individuals over short time as well as long-time intervals [83]. The KOOS-Child demonstrated good psychometric properties in children aged 7–16 years with knee disorders [83, 93, 94].

17.2.4.3 CHQ

The Child Health Questionnaire (CHQ) is a generic PROM designed to assess the health-related quality of life in children and adolescents, validated for a range of pediatric conditions in children and adolescents aged 5–18 years [82, 91]. The questionnaire encompasses multiple aspects of physical, psychological, and social health with 12 domains [91]. Kocher et al. [82] found that Pedi-IKDC was significantly correlated with nine domains of the CHQ. The physical domains of the CHQ (physical functioning, physical limitations, and bodily pain) were best correlated. The domains of behavior and family cohesion were not correlated [82]. Boykin et al. [91] examined the association between the Pedi-IKDC and the CHQ in a group of pediatric patients with ACL injury. Seven of the 12 domains on the CHQ were significantly correlated with the Pedi-IKDC in adolescent patients. Self-esteem, mental health, emotional role, and social limitations categories were correlated with knee function suggesting that pediatric patients are affected differently than adults by ACL injuries. A further understanding of the psychosocial impact of injury may be useful in measuring outcome in children and adolescents [91].

Registration of the various domains of most pediatric PROMs requires cooperation of the child and may be time consuming in a busy outpatient sports medicine clinic [89]. In this regard, more concise PROMs have been developed for the pediatric population (Marx Activity Scale and Hospital for Special Surgery Pediatric Functional Activity Brief Scale) [89, 95–97].

The Marx Activity Scale is a simple and easily readable PROM with sports participation focus and prior use in sports medicine literature [89, 97]. It consists of only four questions regarding the frequency (<1 time/month, 1 time/month, 1 time/week, 2–3 times/week, and ≥4 times/week), each for running question (Q1), cutting (Q2), deceleration (Q3), and pivoting (Q4) in the last year [97]. Shirazi et al. [89] examined the reliability of the Marx Activity Scale in patients younger than 18 years. The Marx Activity Scale was statistically reliable in pediatric patients with knee injuries and lower extremity injury, though the scale was less reliable in patients younger than 14 years [89, 97]. Furthermore, there was a considerable ceiling effect with more than half of the patients with knee injuries reporting maximum scores of 16 [89]. This is likely caused by the high frequency of sports participation (in school, unorganized and organized sports league) by most teenagers, limiting the usefulness of the Marx Activity Scale in the pediatric population [89].

The more recently developed eight-item Hospital for Special Surgery Pediatric Functional Activity Brief Scale (HSS Pedi-FABS) [96] contains the four questions of the Marx Activity Scale [89, 97]. Additional measured items are duration and endurance of the patient, as well as level of competitive and supervised sports [89, 95, 96]. It is a simple, reliable, and valid PROM to assess activity in children and adolescents 10–18 years of age as a prognostic variable for clinical research studies [95, 96]. It was validated in healthy children as a current, though not baseline, activity scale [89].

Other pediatric activity scales are the Gross Motor Function Classification, Family Nutrition and Physical Activity (FNPA) screening tool, and Physical Activity Questionnaire (PAQ) [89]. These PROMs are not useful in children with ACL injuries because they do not specifically measure a patient's sports activity, nor do they register the patient's baseline pre-injury activity status [89].

17.2.4.4 Conclusion

The use of validated PROMs for children with an ACL injury may help improve the quality of clinical care for this pediatric population. Based on psychometric assessment, the Pedi-IKDC and KOOS-Child seem to best monitor the baseline and activity scale in patients aged 6–18 years with ACL injury. A further understanding of the psychosocial impact of injury may be useful in measuring outcome in children and adolescents.

17.3 Future Treatment

17.3.1 The Bridge-Enhanced ACL Repair (BEAR) Technique (Martha Murray)

Traditional surgery for an injured anterior cruciate ligament involves removing the torn ACL and replacing it with a graft of tendon. As we have noted earlier in this chapter, this operation has had good success in limiting additional injuries to the knee and returning patients to sport. However, it still requires using a graft, which in young patients is typically harvested from the patient (autograft). Thus, the pediatric or adolescent patient has to heal not only the graft after it is placed across the knee, but also the site the graft was harvested from. In addition, treatment with a graft does not diminish the risk of developing premature osteoarthritis of the knee after an ACL injury.

Enabling the ACL to heal after an injury, rather than being required to replace it, would eliminate the need to take a graft, and preclinical studies suggest that when successful ligament healing is achieved, the risk of premature osteoarthritis is significantly diminished [98]. The primary reason the ACL fails to heal is a premature loss of the provisional scaffold in the wound site [99]. When the MCL tears, the ends bleed, and the blood clot that forms in the tear site serves as a scaffolding for the ligament ends to grow into and a space for them to reconnect [100]. When the ACL tears, the ends bleed, but the fibrinolytic system in the synovial fluid prevents a clot from forming [101], and thus there is no scaffold to support repair of the ligament.

The Bridge-Enhanced ACL Repair (BEAR) procedure involves using a surgically implanted scaffold to hold clotted blood between the torn ligament ends, thus providing a structural support for the clot and a protected space in which the ligament ends can reconnect. This scaffold is combined with a suture repair for early mechanical stability of the knee [102] (Fig. 17.7). This procedure has been shown to be safe in an initial 20 patient study (NCT 02292004) [103], with no evidence of deep infection or loss of range of motion at 3 or 6 months after surgery (Fig. 17.8). Results from this small study suggest the knees treated with the BEAR procedure have similar stability to knees which have been treated with an autograft hamstring reconstruction and that patients who have a BEAR procedure have similar IKDC scores at 3 and 6 months when compared with patients undergoing ACL reconstruction (Fig. 17.9). A 100-patient randomized clinical trial for BEAR versus autograft ACL reconstruction began recruitment in May 2016 and is ongoing at Boston Children's Hospital (NCT 02664545). Future multicenter studies are planned if there is sufficient promise for this procedure from these initial studies.

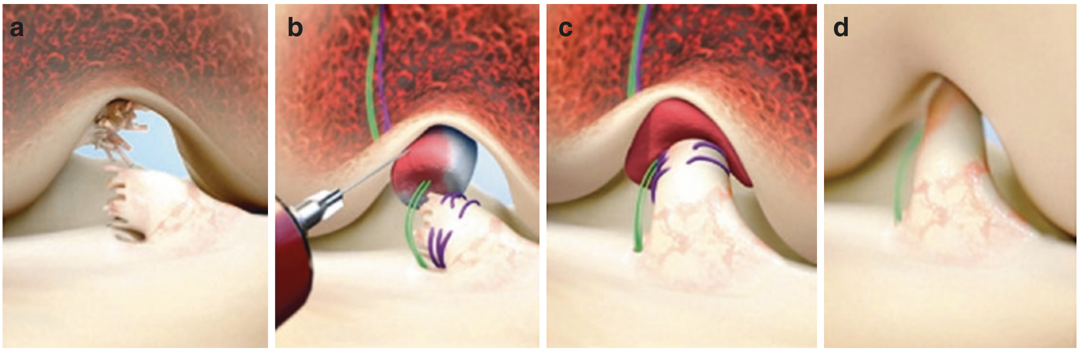


Fig. 17.7 Stepwise demonstration of the bridge-enhanced ACL repair (BEAR) technique using the BEAR scaffold. (**a**) Torn ACL. (**b**) A whipstitch is placed into the tibial stump of the ACL. Tunnels are drilled in the femur and tibia, and a cortical button with sutures attached to it is passed through the femoral tunnel and engaged on the proximal femoral cortex. The sutures are threaded through the BEAR scaffold, tibial tunnel, and secured in place with a second extracortical button. The BEAR scaffold is then saturated with 10 mL of the patient's blood and (**c**) the tibial stump pulled up into the saturated scaffold. (**d**) The ends of the torn ACL then grow into the BEAR scaffold and the ligament reunites. ACL, anterior cruciate ligament (Used with permission from Murray et al. [103])

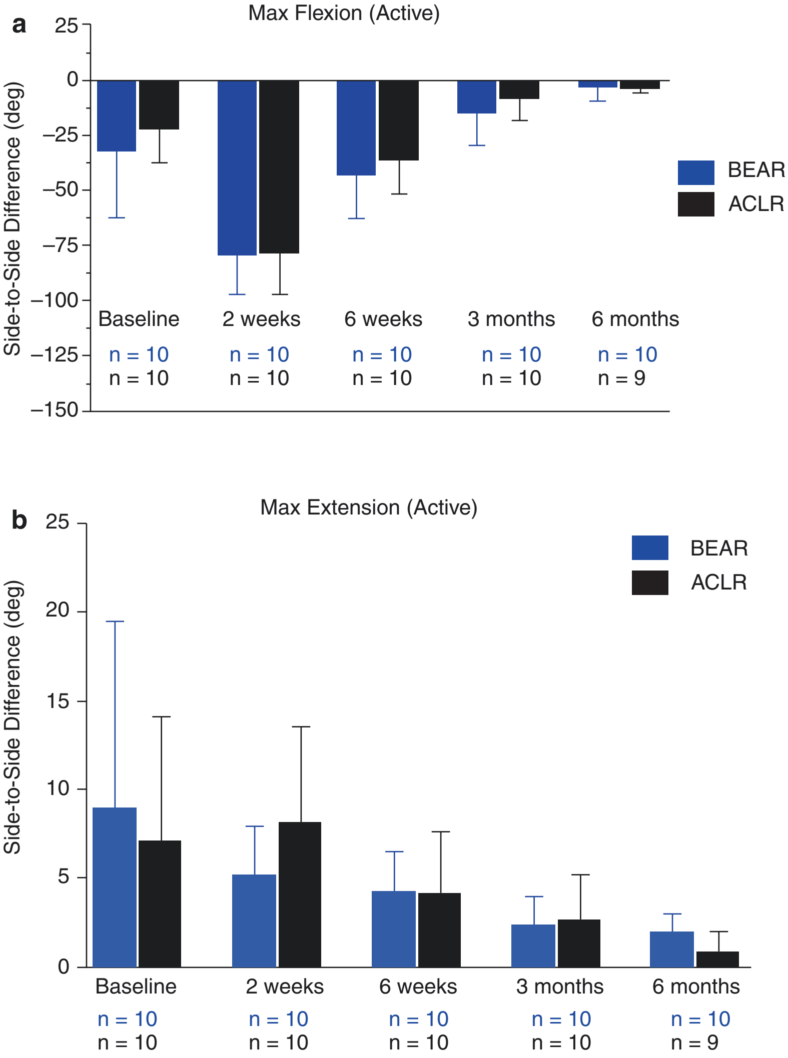


Fig. 17.8 Maximum active flexion (**a**) and extension (**b**) at time points up to 6 months after surgery for knees treated with Bridge-Enhanced ACL Repair (BEAR) and ACL autograft hamstring reconstruction (ACLR). No significant loss of range of motion was noted for any of the patients in the study.

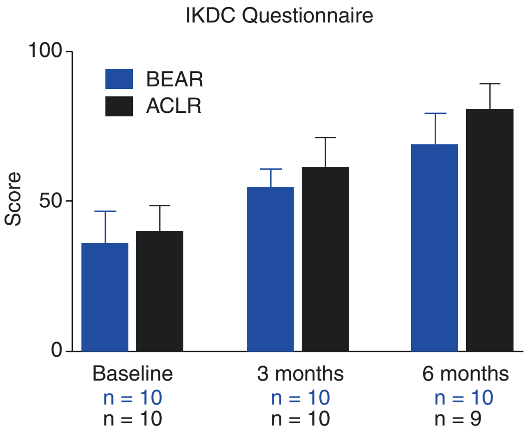


Fig. 17.9 International Knee Documentation Committee (IKDC) score for the patients treated with Bridge-Enhanced ACL Repair (BEAR) and ACL autograft hamstring reconstruction (ACLR). This is a validated patient-reported outcome measure for knee surgery. No significant difference was seen between the two groups of patients

17.3.2 The Future (Martin Lind)

The major unsolved issue in the management of pediatric ACL is graft failure after ACL reconstruction, up to 10% in 13–15-year-old patients at 5-year follow-up, which is 3 times higher than in adult patients. In females returning to contact sports, the graft failure rate is even higher. It is unlikely that new reconstruction techniques can improve this problem, and therefore better rehabilitation systems for girls wanting to return to contact sports are needed. Probably, there is also a need to counsel high-risk patients, such as girls with hyperlaxity, poor motor control, valgus knee deformity, and obesity, to find other sports activities than contact sports after an ACL lesion, irrespective of operative or nonoperative treatment. Nonoperative treatment rehabilitation principles have demonstrated that two-thirds of patients can return to demanding sports activities if intensive rehabilitation is performed and close long-term follow-up is instituted. There is still a need to define which patients do not function satisfactory with rehabilitation alone, so that these patients can receive early surgical treatment.

Another important unsolved issue is how to limit growth disturbances after pediatric ACL reconstruction. Despite the fact that recent studies have demonstrated up to 25% of patients having more than 1 cm limb shortening after ACL reconstruction, less than 5% of patients experience a symptomatic limp length discrepancy. Presently, physis-sparing ACL reconstruction techniques are being developed to hopefully reduce these problems. Level 1 studies are needed to fully elucidate the safety potential of physis-sparing reconstruction techniques. The PAMI initiative [12] aims for international multicenter data of pediatric ACL lesion management and could reveal important information on the impact of different reconstruction techniques. However, it is important to include high-quality radiographic outcome data in a PAMI database in order to evaluate growth disturbances properly.

ACL repair principles, as suggested by the BEAR technique, have demonstrated preclinical proof of concept as well as good safety and clinical proof of concept in a phase 1 study [102, 103]. An ongoing phase 3 study, comparing the BEAR treatment with hamstring autograft ACL reconstruction, will provide important information whether initial ACL repair should be used instead of autograft ACL reconstruction in the pediatric ACL-injured patient population.

17.4 Take-Home Messages

- Pediatric ACL tears are rare, accounting for less than 5% of all ACL injuries, and do rarely occur under the age of 9. Their current evidence for treatment is low.
- It is imperative that the child and parents are provided with thorough information on the benefits and risks involved with both surgical and conservative treatment.
- There is an international consensus that in case of concomitant damage to the meniscus (bucket-handle or meniscal ramp lesions) and/or persistent knee instability, an ACL reconstruction should be performed. The goal is to stabilize the knee, improve its function, repair the meniscus, and protect the knee from future episodes of giving way and injuries.
- According to the localization of the tibial and femoral tunnels, surgical techniques can be divided into three categories: transphyseal

procedures, where the tunnels are drilled through the growth plates; epiphyseal techniques, where the tunnels are located in the tibial and femoral epiphysis, not injuring the growth plate; and finally extra-epiphyseal techniques, where the graft is placed around the growth plate.

- There is still no convincing evidence that demonstrates which technique should be used in pediatric ACL reconstruction. Most studies report very good outcome after surgery, but there is an alarming high graft rupture rate.
- The backside of performing ACL reconstructions in skeletally immature patients is the risk of provoking a growth disturbance following transepiphyseal drilling and the unknown development of the graft with postoperative growth.
- The use of validated PROMs for children with ACL injury may help improve the quality of clinical care for this pediatric population. Based on psychometric assessment, the Pedi-IKDC and KOOS-Child are advised for patients aged 6–18 years with ACL injury.
- There is a need to improve current rehabilitation protocols for high-risk patients, such as girls with hyperlaxity, poor motor control, valgus knee deformity, and obesity.
- Preliminary results of the Bridge-Enhanced ACL Repair (BEAR) technique show promising short-term results for ACL repair techniques.
- In order to look for answers to the remaining open questions in pediatric ACL injuries, the ESSKA Foundation started the “Pediatric ACL Monitoring Initiative” (PAMI), a combined, multicenter project on these relatively rare lesions.

References

1. Calvo R, Figueroa D, Gili F, Vaisman A, Mococain P, Espinosa M, Leon A, Arellano S. Transphyseal anterior cruciate ligament reconstruction in patients with open physes: 10-year follow-up study. Am J Sports Med. 2015;43:289–94.
2. Demange MK, Camanho GL. Nonanatomic anterior cruciate ligament reconstruction with double-stranded semitendinosus grafts in children with open physes: minimum 15-year follow-up. Am J Sports Med. 2014;42:2926–32.
3. Goddard M, Bowman N, Salmon LJ, Waller A, Roe JP, Pinczewski LA. Endoscopic anterior cruciate ligament reconstruction in children using living donor hamstring tendon allografts. Am J Sports Med. 2013;41:567–74.
4. Kim SJ, Shim DW, Park KW. Functional outcome of transphyseal reconstruction of the anterior cruciate ligament in skeletally immature patients. Knee Surg Relat Res. 2012;24:173–9.
5. Kumar S, Ahearne D, Hunt DM. Transphyseal anterior cruciate ligament reconstruction in the skeletally immature: follow-up to a minimum of sixteen years of age. J Bone Joint Surg Am. 2013;95:e1.
6. Moksnes H, Engebretsen L, Eitzen I, Risberg MA. Functional outcomes following a non-operative treatment algorithm for anterior cruciate ligament injuries in skeletally immature children 12 years and younger. A prospective cohort with 2 years follow-up. Br J Sports Med. 2013;47:488–94.
7. Moksnes H, Engebretsen L. It takes more than timing: letter to the editor. Am J Sports Med. 2015;43:NP14–5.
8. Reider B. A matter of timing. Am J Sports Med. 2015;43:273–4.
9. Moksnes H, Engebretsen L, Risberg MA. The current evidence for treatment of acl injuries in children is low: a systematic review. J Bone Joint Surg Am. 2012;94:1112–9.
10. Funahashi KM, Moksnes H, Maletis GB, Csintalan RP, Inacio MC, Funahashi TT. Anterior cruciate ligament injuries in adolescents with open physis: effect of recurrent injury and surgical delay on meniscal and cartilage injuries. Am J Sports Med. 2014;42:1068–73.
11. Renstrom P, Ljungqvist A, Arendt E, Beynnon B, Fukubayashi T, Garrett W, Georgoulis T, Hewett TE, Johnson R, Krosshaug T, Mandelbaum B, Micheli L, Myklebust G, Roos E, Roos H, Schamasch P, Shultz S, Werner S, Wojtys E, Engebretsen L. Non-contact acl injuries in female athletes: an international olympic committee current concepts statement. Br J Sports Med. 2008;42:394–412.
12. Moksnes H, Engebretsen L, Seil R. The esska paediatric anterior cruciate ligament monitoring initiative. Knee Surg Sports Traumatol Arthrosc. 2016;24:680–7.
13. Aichroth PM, Patel DV, Zorrilla P. The natural history and treatment of rupture of the anterior cruciate ligament in children and adolescents. A prospective review. J Bone Joint Surg (Br). 2002;84:38–41.
14. Cohen M, Ferretti M, Quarteiro M, Marcondes FB, de Hollanda JP, Amaro JT, Abdalla RJ. Transphyseal anterior cruciate ligament reconstruction in patients with open physes. Arthroscopy. 2009;25:831–8.
15. Mizuta H, Kubota K, Shiraishi M, Otsuka Y, Nagamoto N, Takagi K. The conservative treatment of complete tears of the anterior cruciate ligament in skeletally immature patients. J Bone Joint Surg (Br). 1995;77:890–4.
16. Anderson AF, Anderson CN. Correlation of meniscal and articular cartilage injuries in children and

adolescents with timing of anterior cruciate ligament reconstruction. Am J Sports Med. 2015;43: 275–81.

17. Frosch KH, Stengel D, Brodhun T, Stietencron I, Holsten D, Jung C, Reister D, Voigt C, Niemeyer P, Maier M, Hertel P, Jagodzinski M, Lill H. Outcomes and risks of operative treatment of rupture of the anterior cruciate ligament in children and adolescents. Arthroscopy. 2010;26:1539–50.
18. Kaeding CC, Flanigan D, Donaldson C. Surgical techniques and outcomes after anterior cruciate ligament reconstruction in preadolescent patients. Arthroscopy. 2010;26:1530–8.
19. Werner BC, Yang S, Looney AM, Gwathmey FW Jr. Trends in pediatric and adolescent anterior cruciate ligament injury and reconstruction. J Pediatr Orthop. 2016;36:447–52.
20. Dodwell ER, Lamont LE, Green DW, Pan TJ, Marx RG, Lyman S. 20 years of pediatric anterior cruciate ligament reconstruction in new york state. Am J Sports Med. 2014;42:675–80.
21. Moksnes H, Engebretsen L, Risberg MA. Management of anterior cruciate ligament injuries in skeletally immature individuals. J Orthop Sports Phys Ther. 2012;42:172–83.
22. Anderson CN, Anderson AF. Management of the anterior cruciate ligament-injured knee in the skeletally immature athlete. Clin Sports Med. 2017;36: 35–52.
23. Dumont GD, Hogue GD, Padalecki JR, Okoro N, Wilson PL. Meniscal and chondral injuries associated with pediatric anterior cruciate ligament tears: relationship of treatment time and patient-specific factors. Am J Sports Med. 2012;40:2128–33.
24. Ramski DE, Kanj WW, Franklin CC, Baldwin KD, Ganley TJ. Anterior cruciate ligament tears in children and adolescents: a meta-analysis of nonoperative versus operative treatment. Am J Sports Med. 2014;42:2769–76.
25. Vavken P, Tepolt FA, Kocher MS. Concurrent meniscal and chondral injuries in pediatric and adolescent patients undergoing acl reconstruction. J Pediatr Orthop. 2016. https://doi.org/10.1097/BPO.0000000000000777.
26. Heckmann TP, Barber-Westin SD, Noyes FR. Meniscal repair and transplantation: indications, techniques, rehabilitation, and clinical outcome. J Orthop Sports Phys Ther. 2006;36:795–814.
27. Kocher MS, Saxon HS, Hovis WD, Hawkins RJ. Management and complications of anterior cruciate ligament injuries in skeletally immature patients: survey of the herodicus society and the acl study group. J Pediatr Orthop. 2002;22:452–7.
28. Astur DC, Arliani GG, Debieux P, Kaleka CC, Amaro JT, Cohen M. Intraarticular hamstring graft diameter decreases with continuing knee growth after acl reconstruction with open physes. Knee Surg Sports Traumatol Arthrosc. 2016;24:792–5.
29. Bollen S, Pease F, Ehrenraich A, Church S, Skinner J, Williams A. Changes in the four-strand hamstring graft in anterior cruciate ligament reconstruction in the skeletally-immature knee. J Bone Joint Surg (Br). 2008;90:455–9.
30. Kocher MS, Garg S, Micheli LJ. Physeal sparing reconstruction of the anterior cruciate ligament in skeletally immature prepubescent children and adolescents. J Bone Joint Surg Am. 2005;87:2371–9.
31. Kocher MS, Smith JT, Zoric BJ, Lee B, Micheli LJ. Transphyseal anterior cruciate ligament reconstruction in skeletally immature pubescent adolescents. J Bone Joint Surg Am. 2007;89:2632–9.
32. Chotel F, Henry J, Seil R, Chouteau J, Moyen B, Berard J. Growth disturbances without growth arrest after acl reconstruction in children. Knee Surg Sports Traumatol Arthrosc. 2010;18:1496–500.
33. Shifflett GD, Green DW, Widmann RF, Marx RG. Growth arrest following acl reconstruction with hamstring autograft in skeletally immature patients: a review of 4 cases. J Pediatr Orthop. 2016;36:355–61.
34. Zimmerman LJ, Jauregui JJ, Riis JF, Tuten HR. Symmetric limb overgrowth following anterior cruciate ligament reconstruction in a skeletally immature patient. J Pediatr Orthop B. 2015;24:530–4.
35. Kruse LM, Gray BL, Wright RW. Anterior cruciate ligament reconstruction rehabilitation in the pediatric population. Clin Sports Med. 2011;30: 817–24.
36. Mohtadi N, Grant J. Managing anterior cruciate ligament deficiency in the skeletally immature individual: a systematic review of the literature. Clin J Sport Med. 2006;16:457–64.
37. Grindem H, Granan LP, Risberg MA, Engebretsen L, Snyder-Mackler L, Eitzen I. How does a combined preoperative and postoperative rehabilitation programme influence the outcome of acl reconstruction 2 years after surgery? A comparison between patients in the delaware-oslo acl cohort and the norwegian national knee ligament registry. Br J Sports Med. 2015;49:385–9.
38. Lynch AD, Logerstedt DS, Grindem H, Eitzen I, Hicks GE, Axe MJ, Engebretsen L, Risberg MA, Snyder-Mackler L. Consensus criteria for defining 'successful outcome' after acl injury and reconstruction: a delaware-oslo acl cohort investigation. Br J Sports Med. 2015;49:335–42.
39. Moksnes H, Engebretsen L, Risberg MA. Prevalence and incidence of new meniscus and cartilage injuries after a nonoperative treatment algorithm for acl tears in skeletally immature children: a prospective mri study. Am J Sports Med. 2013;41:1771–9.
40. Moksnes H, Engebretsen L, Risberg MA. Performance-based functional outcome for children 12 years or younger following anterior cruciate ligament injury: a two to nine-year follow-up study. Knee Surg Sports Traumatol Arthrosc. 2008;16:214–23.
41. Woods GW, O'Connor DP. Delayed anterior cruciate ligament reconstruction in adolescents with open physes. Am J Sports Med. 2004;32:201–10.
42. Moksnes H, Grindem H. Prevention and rehabilitation of paediatric anterior cruciate ligament injuries. Knee Surg Sports Traumatol Arthrosc. 2016;24: 730–6.

43. Faber M, Andersen MH, Sevel C, Thorborg K, Bandholm T, Rathleff M. The majority are not performing home-exercises correctly two weeks after their initial instruction-an assessor-blinded study. Peer J. 2015;3:e1102.
44. Faigenbaum AD, Kraemer WJ, Blimkie CJ, Jeffreys I, Micheli LJ, Nitka M, Rowland TW. Youth resistance training: updated position statement paper from the national strength and conditioning association. J Strength Cond Res. 2009;23:S60–79.
45. Hewett TE, Ford KR, Myer GD. Anterior cruciate ligament injuries in female athletes: part 2, a meta-analysis of neuromuscular interventions aimed at injury prevention. Am J Sports Med. 2006;34:490–8.
46. McKay CD, Steffen K, Romiti M, Finch CF, Emery CA. The effect of coach and player injury knowledge, attitudes and beliefs on adherence to the fifa 11+ programme in female youth soccer. Br J Sports Med. 2014;48:1281–6.
47. Steffen K, Emery CA, Romiti M, Kang J, Bizzini M, Dvorak J, Finch CF, Meeuwisse WH. High adherence to a neuromuscular injury prevention programme (fifa 11+) improves functional balance and reduces injury risk in canadian youth female football players: a cluster randomised trial. Br J Sports Med. 2013;47:794–802.
48. Alentorn-Geli E, Myer GD, Silvers HJ, Samitier G, Romero D, Lazaro-Haro C, Cugat R. Prevention of non-contact anterior cruciate ligament injuries in soccer players. Part 1: mechanisms of injury and underlying risk factors. Knee Surg Sports Traumatol Arthrosc. 2009;17:705–29.
49. Olsen OE, Myklebust G, Engebretsen L, Holme I, Bahr R. Exercises to prevent lower limb injuries in youth sports: cluster randomised controlled trial. BMJ. 2005;330:449.
50. Soligard T, Myklebust G, Steffen K, Holme I, Silvers H, Bizzini M, Junge A, Dvorak J, Bahr R, Andersen TE. Comprehensive warm-up programme to prevent injuries in young female footballers: cluster randomised controlled trial. BMJ. 2008;337:a2469.
51. Millett PJ, Willis AA, Warren RF. Associated injuries in pediatric and adolescent anterior cruciate ligament tears: does a delay in treatment increase the risk of meniscal tear? Arthroscopy. 2002;18:955–9.
52. Streich NA, Barie A, Gotterbarm T, Keil M, Schmitt H. Transphyseal reconstruction of the anterior cruciate ligament in prepubescent athletes. Knee Surg Sports Traumatol Arthrosc. 2010;18:1481–6.
53. Henry J, Chotel F, Chouteau J, Fessy MH, Berard J, Moyen B. Rupture of the anterior cruciate ligament in children: early reconstruction with open physes or delayed reconstruction to skeletal maturity? Knee Surg Sports Traumatol Arthrosc. 2009;17:748–55.
54. McCarthy MM, Graziano J, Green DW, Cordasco FA. All-epiphyseal, all-inside anterior cruciate ligament reconstruction technique for skeletally immature patients. Arthrosc Tech. 2012;1:e231–9.
55. Fabricant PD, Jones KJ, Delos D, Cordasco FA, Marx RG, Pearle AD, Warren RF, Green DW. Reconstruction of the anterior cruciate ligament in the skeletally immature athlete: a review of current concepts: aaos exhibit selection. J Bone Joint Surg Am. 2013;95:e28.
56. Chotel F, Seil R. Growth disturbances after transphyseal acl reconstruction in skeletally immature patients: who is more at risk? Young child or adolescent? J Pediatr Orthop. 2013;33:585–6.
57. Lemaitre G, Salle de Chou E, Pineau V, Rochcongar G, Delforge S, Bronfen C, Haumont T, Hulet C. Acl reconstruction in children: a transphyseal technique. Orthop Traumatol Surg Res. 2014;100: S261–5.
58. Seil R, Weitz FK, Pape D. Surgical-experimental principles of anterior cruciate ligament (acl) reconstruction with open growth plates. J Exp Orthop. 2015;2:11.
59. Chotel F, Bonnard C, Accabled F, Gicquel P, Bergerault F, Robert H, Seil R, Hulet C, Cassard X, Garraud P. Résultats et facteurs pronostiques de la reconstruction de lca sur genou en croissance. A propos d'une série multicentrique de 102 cas. Rev Chir Orthop Reparatrice Apar Mot. 2007;93:3s131–8.
60. Shea KG, Apel PJ, Pfeiffer RP. Anterior cruciate ligament injury in paediatric and adolescent patients: a review of basic science and clinical research. Sports Med. 2003;33:455–71.
61. Seil R, Pape D, Kohn D. The risk of growth changes during transphyseal drilling in sheep with open physes. Arthroscopy. 2008;24:824–33.
62. Stadelmaier DM, Arnoczky SP, Dodds J, Ross H. The effect of drilling and soft tissue grafting across open growth plates. A histologic study. Am J Sports Med. 1995;23:431–5.
63. Koman JD, Sanders JO. Valgus deformity after reconstruction of the anterior cruciate ligament in a skeletally immature patient. A case report. J Bone Joint Surg Am. 1999;81:711–5.
64. McConkey MO, Bonasia DE, Amendola A. Pediatric anterior cruciate ligament reconstruction. Curr Rev. Musculoskelet Med. 2011;4:37–44.
65. Robert HE, Casin C. Valgus and flexion deformity after reconstruction of the anterior cruciate ligament in a skeletally immature patient. Knee Surg Sports Traumatol Arthrosc. 2010;18:1369–73.
66. Dorizas JA, Stanitski CL. Anterior cruciate ligament injury in the skeletally immature. Orthop Clin North Am. 2003;34:355–63.
67. Lind M, Menhert F, Pedersen AB. The first results from the danish acl reconstruction registry: epidemiologic and 2 year follow-up results from 5,818 knee ligament reconstructions. Knee Surg Sports Traumatol Arthrosc. 2009;17:117–24.
68. Fauno P, Romer L, Nielsen T, Lind M. The risk of transphyseal drilling in skeletally immature patients with anterior cruciate ligament injury. Orthop J Sports Med. 2016;4:2325967116664685.
69. Kohl S, Stutz C, Decker S, Ziebarth K, Slongo T, Ahmad SS, Kohlhof H, Eggli S, Zumstein M, Evangelopoulos DS. Mid-term results of transphyseal anterior cruciate ligament reconstruction in children and adolescents. Knee. 2014;21:80–5.

70. Schmale GA, Kweon C, Larson RV, Bompadre V. High satisfaction yet decreased activity 4 years after transphyseal acl reconstruction. Clin Orthop Relat Res. 2014;472:2168–74.
71. Koizumi H, Kimura M, Kamimura T, Hagiwara K, Takagishi K. The outcomes after anterior cruciate ligament reconstruction in adolescents with open physes. Knee Surg Sports Traumatol Arthrosc. 2013;21: 950–6.
72. Willimon SC, Jones CR, Herzog MM, May KH, Leake MJ, Busch MT. Micheli anterior cruciate ligament reconstruction in skeletally immature youths: a retrospective case series with a mean 3-year follow-up. Am J Sports Med. 2015;43:2974–81.
73. Ho B, Edmonds EW, Chambers HG, Bastrom TP, Pennock AT. Risk factors for early acl reconstruction failure in pediatric and adolescent patients: a review of 561 cases. J Pediatr Orthop. 2016. https://doi.org/10.1097/BPO.0000000000000831.
74. Fauno P, Rahr-Wagner L, Lind M. Risk for revision after anterior cruciate ligament reconstruction is higher among adolescents: results from the danish registry of knee ligament reconstruction. Orthop J Sports Med. 2014;2:2325967114552405.
75. Andernord D, Desai N, Bjornsson H, Ylander M, Karlsson J, Samuelsson K. Patient predictors of early revision surgery after anterior cruciate ligament reconstruction: a cohort study of 16,930 patients with 2-year follow-up. Am J Sports Med. 2015;43:121–7.
76. Pierce TP, Issa K, Festa A, Scillia AJ, McInerney VK. Pediatric anterior cruciate ligament reconstruction. Am J Sports Med. 2017;45:488–94.
77. Vavken P, Murray MM. Treating anterior cruciate ligament tears in skeletally immature patients. Arthroscopy. 2011;27:704–16.
78. Newman JT, Carry PM, Terhune EB, Spruiell MD, Heare A, Mayo M, Vidal AF. Factors predictive of concomitant injuries among children and adolescents undergoing anterior cruciate ligament surgery. Am J Sports Med. 2015;43:282–8.
79. Mansson O, Sernert N, Rostgard-Christensen L, Kartus J. Long-term clinical and radiographic results after delayed anterior cruciate ligament reconstruction in adolescents. Am J Sports Med. 2015;43:138–45.
80. de Vet HC, Terwee CB, Mokkink LB, Knol DL. Measurement in medicine. Cambridge: Cambridge University Press; 2011.
81. Black N. Patient reported outcome measures could help transform healthcare. BMJ. 2013;346:f167.
82. Kocher MS, Smith JT, Iversen MD, Brustowicz K, Ogunwole O, Andersen J, Yoo WJ, McFeely ED, Anderson AF, Zurakowski D. Reliability, validity, and responsiveness of a modified international knee documentation committee subjective knee form (pedi-ikdc) in children with knee disorders. Am J Sports Med. 2011;39:933–9.
83. Ortqvist M, Iversen MD, Janarv PM, Brostrom EW, Roos EM. Psychometric properties of the knee injury and osteoarthritis outcome score for children (koos-child) in children with knee disorders. Br J Sports Med. 2014;48:1437–46.
84. Schmitt LC, Paterno MV, Huang S. Validity and internal consistency of the international knee documentation committee subjective knee evaluation form in children and adolescents. Am J Sports Med. 2010;38:2443–7.
85. Terwee CB, Bot SD, de Boer MR, van der Windt DA, Knol DL, Dekker J, Bouter LM, de Vet HC. Quality criteria were proposed for measurement properties of health status questionnaires. J Clin Epidemiol. 2007;60:34–42.
86. Iversen MD, Lee B, Connell P, Andersen J, Anderson AF, Kocher MS. Validity and comprehensibility of the international knee documentation committee subjective knee evaluation form in children. Scand J Med Sci Sports. 2010;20:e87–95.
87. Oak SR, O'Rourke C, Strnad G, Andrish JT, Parker RD, Saluan P, Jones MH, Stegmeier NA, Spindler KP. Statistical comparison of the pediatric versus adult ikdc subjective knee evaluation form in adolescents. Am J Sports Med. 2015;43: 2216–21.
88. Ortqvist M, Roos EM, Brostrom EW, Janarv PM, Iversen MD. Development of the knee injury and osteoarthritis outcome score for children (koos-child): comprehensibility and content validity. Acta Orthop. 2012;83:666–73.
89. Shirazi CP, Israel HA, Kaar SG. Is the marx activity scale reliable in patients younger than 18 years? Sports Health. 2016;8:145–8.
90. Jacobsen JS, Knudsen P, Fynbo C, Rolving N, Warming S. Reproducibility and responsiveness of a danish pedi-ikdc subjective knee form for children with knee disorders. Scand J Med Sci Sports. 2016;26:1408–14.
91. Boykin RE, McFeely ED, Shearer D, Frank JS, Harrod CC, Nasreddine AY, Kocher MS. Correlation between the child health questionnaire and the international knee documentation committee score in pediatric and adolescent patients with an anterior cruciate ligament tear. J Pediatr Orthop. 2013;33: 216–20.
92. Nasreddine AY, Connell PL, Kalish LA, Nelson S, Iversen MD, Anderson AF, Kocher MS. The pediatric international knee documentation committee (pedi-ikdc) subjective knee evaluation form: normative data. Am J Sports Med. 2016;45(3):527–34. https://doi.org/10.1177/0363546516672456.
93. Van der Velden CA, Van der Steen MC, Leenders J, van Douveren FQ, Janssen RP, Reijman M. Pedi-ikdc or koos-child: which questionnaire should be used in Dutch children with knee disorders? A translation, cross-cultural adaptation, validation and reliability study. Br J Sports Med. 2017. In Review.

94. Dietvorst M, Reijman M, van Groningen B, van der Steen M, Janssen RP. PROMs in pediatric knee ligament injury: use the Pedi-IKDC and avoid using adult PROMs. Knee Surg Sports Traumatol Arthrosc. 2017;16:2017. https://doi.org/10.1007/s00167-017-4687-3.
95. Fabricant PD, Robles A, McLaren SH, Marx RG, Widmann RF, Green DW. Hospital for special surgery pediatric functional activity brief scale predicts physical fitness testing performance. Clin Orthop Relat Res. 2014;472:1610–6.
96. Fabricant PD, Robles A, Downey-Zayas T, Do HT, Marx RG, Widmann RF, Green DW. Development and validation of a pediatric sports activity rating scale: the hospital for special surgery pediatric functional activity brief scale (hss pedi-fabs). Am J Sports Med. 2013;41:2421–9.
97. Marx RG. Knee rating scales. Arthroscopy. 2003;19:1103–8.
98. Murray MM, Fleming BC. Use of a bioactive scaffold to stimulate anterior cruciate ligament healing also minimizes posttraumatic osteoarthritis after surgery. Am J Sports Med. 2013;41:1762–70.
99. Murray MM, Martin SD, Martin TL, Spector M. Histological changes in the human anterior cruciate ligament after rupture. J Bone Joint Surg Am. 2000;82-A:1387–97.
100. Frank C, Woo SL, Amiel D, Harwood F, Gomez M, Akeson W. Medial collateral ligament healing. A multidisciplinary assessment in rabbits. Am J Sports Med. 1983;11:379–89.
101. Harrold AJ. The defect of blood coagulation in joints. J Clin Pathol. 1961;14:305–8.
102. Murray MM, Magarian E, Zurakowski D, Fleming BC. Bone-to-bone fixation enhances functional healing of the porcine anterior cruciate ligament using a collagen-platelet composite. Arthroscopy. 2010;26:S49–57.
103. Murray MM, Flutie BM, Kalish LA, Ecklund K, Fleming BC, Proffen BL, Micheli LJ. The bridge-enhanced anterior cruciate ligament repair (bear) procedure: an early feasibility cohort study. Orthop J Sports Med. 2016;4:2325967116672176.

Return to Play Following Achilles Tendon Rupture

18

Michael R. Carmont, Jennifer A. Zellers,
Clare Ardern, Karin Grävare Silbernagel,
Jón Karlsson, and Hermann Mayr

18.1 Introduction

The incidence of Achilles tendon rupture has been increasing since this has featured in the literature in the 1980s [1, 2]. A greater appreciation of variations in incidence has occurred with the adoption of nationwide hospital [3–5] and provider group databases [6–9] together with the development of Achilles tendon rupture registries [10]. A mean annual increase in rupture rate of 2.4% has recently been reported [1]. The most recently reported injury rates are of 46 per 100,000 in 2013 [7].

The mechanism of injury is typically a rapid eccentric loading of the gastrocnemius-soleus complex. This typically occurs during sports activity [11] such as football and badminton in males and netball in females [12].

Whilst the majority of Achilles tendon ruptures are sustained during sports or athletic activity, they are not necessarily sustained by patients who would be considered to be athletes but most commonly the busy middle aged. The typical patient is in the mid-40s [12] and has recently returned to sports activity from either a period of work or looking after children, during which time the tendon has effectively become deconditioned. Males tend to be more frequently affected than females with a ratio of approximately 6:1 [13].

18.2 Consequences of Rupture

A rupture of the Achilles tendon has a prolonged recovery leaving a 10–30% reduction in functional calf strength [14–17] and endurance [18], despite increased muscle activity [19, 20]. The injury pro-

---

M.R. Carmont (✉)
Department of Orthopaedic Surgery, Princess Royal Hospital, Shrewsbury & Telford Hospital NHS Trust, Shrewsbury, UK

The Department of Orthopaedic Surgery, Institute of Clinical Sciences, Sahlgrenska Academy, University of Gothenburg, Gothenburg, Sweden
e-mail: mcarmont@hotmail.com

J.A. Zellers
The Department of Physiotherapy, University of Delware, Delaware, USA

C. Ardern
Department of Phyiscal Therapy, Linköping Univeristy, Linköping, Sweden

K.G. Silbernagel
The Department of Physiotherapy, University of Delware, Delaware, USA

The Department of Orthopaedic Surgery, Institute of Clinical Sciences, Sahlgrenska Academy, University of Gothenburg, Gothenburg, Sweden

J. Karlsson
The Department of Orthopaedic Surgery, Institute of Clinical Sciences, Sahlgrenska Academy, University of Gothenburg, Gothenburg, Sweden

H. Mayr
Department of Knee, Hip and Shoulder Surgery, Schoen Clinic Munich Harlaching, Munich, Germany

G.M.M.J. Kerkhoffs et al. (eds.), *ESSKA Instructional Course Lecture Book*,
https://doi.org/10.1007/978-3-662-56127-0_18

duces long-term limitations [4, 19, 21–23], and many patients fail to return to sports activities to the pre-injury level of performance [24].

In addition to the weakness following rupture, there are consequences and risks of the management option chosen, and there is also the potential for failure of treatment. These all need to be considered by the patient, in terms of their professional and sporting individual requirements. Examples here include the 55-year-old who trains and competes at masters sports events versus the early 1970s grandmother with plenty of family support and wants to ensure that she is able to get back to walking her dog.

18.3 The Achilles Tendon Has Been Ruptured: What Are the Management Options?

Any decisions in terms of management involve a discussion between the patient and the treating surgeon. This is a shared decision-making process, where the patient is aware that they are involved in the treatment selection. They need to have accurate information about the options and need to be guided to select the best method of treatment for them.

One of our roles as health-care providers is to explain the science and experience of those that have been before us and be able to accurately interpret the literature.

The management options for Achilles tendon rupture may be broadly split into two: nonoperative and operative treatments.

Nonoperative management now features a temporary or short-term cast followed by functional bracing with early weight-bearing [25, 26] and early movement. For the general population, this method produces low re-rupture rates and satisfactory outcomes for activities of daily living [27, 28].

Operative treatment may be divided into percutaneous, minimally invasive, open and augmented repairs. Following repair, full weight-bearing as able is encouraged, in addition to early movement [4, 29–32].

The majority of randomized controlled trials comparing nonoperative and operative treatment have shown no significant differences in terms of the avoidance of re-rupture, the primary outcome measure. This is usually due to small numbers of patients, giving an underpowered study for the dichotomous variable of re-rupture or no re-rupture. The re-rupture rate is around 0–3% after surgery and above 10% after nonoperative management. Many studies are adequately powered for current outcome variables, however, not for all the possible confounding factors, and as such may not yield accurate information on return to play.

The randomized controlled trials do, however, yield the following results:

Functional outcome: Patients who compete in sports activity, the equivalent to high-speed torque exercise, drop-counter movement jump and hopping assessments, will have a significantly less functional weakness after operative treatment [14, 17, 33, 34].

Failure and tendon elongation: Operative repair may be less prone to permanent elongation with interposed scar tissue or non-union and failure of treatment than nonoperative management [14, 35].

Re-rupture rates: The literature shows increased re-rupture rate after nonoperative treatment compared with operative treatment; however, there are fewer overall complications than after surgery. However, if accelerated rehabilitation is used, this risk of re-rupture decreases [36].

Return to play and activity: If treatment goes well and without complication(s), most patients are able to return to their previous level of sports activity [24]. If re-rupture occurs, patients rarely return to their previous level of sports activity [37]. The presence of complications significantly affects outcome.

Return to work and early function: Patients return to work [38] earlier and at 3 months [39] have improved function after operative treatment.

Minimally invasive and percutaneous surgery vs. open repair: Minimally invasive (MI) and percutaneous surgery provides good outcome and reduces the risk of wound breakdown and improves cosmesis compared with open surgery [40–42].

Open end-to-end vs. augmented repair: Simple end-to-end repairs have equal outcome scores, compared with repairs augmented with a fascial turndown flap. The use of a flap does not prevent tendon lengthening or muscle weakness [22, 43, 44].

The majority of Achilles tendon ruptures are sustained during sports or athletic activity; however, patients frequently have not participated in regular exercise. Similarly the exercise or activity during which the activity occurred is commonly relatively new to them. The typical patient is middle aged and has recently returned to sports activity either from a period of work or children care during which time the tendon has effectively become deconditioned.

Given the limited reporting of pre-injury sports participation [24], there are relatively few series of athletes who have sustained an Achilles tendon rupture, meaning that they are a specific subgroup for which the previous literature is too generalized from which to draw firm conclusions.

18.4 Management Decision-Making Process

Sportsmen and sportswomen will have different priorities in terms of treatment options compared with the general population. Many athletes appreciate that the injury has a prolonged recovery time; however, the presence of risks and in particular the risk of a poor outcome may be less understood. All patients wish to avoid complications, time loss and reduced functional outcome of re-rupture. Additionally competitive athletes wish to minimize any functional loss relating to their injury. The decision to choose operative treatment, or not, may be made on the basis of improved outcome together with the risks of a poor outcome and the risks and impact of complications rather than solely the risk of re-rupture.

Participants in competitive sports activities such as football are not usually affected by co-morbidities, e.g. obesity and diabetes, which would predispose to infection and wound healing problems. Olsson has determined that a high body mass index (BMI) leads to an increased risk of a poor outcome [45]; however in the athletic population, the BMI may be high although sportsmen may be healthy. This means that there is likely to be little risk of wound complication in athletes.

18.5 Outcome in Elite Athletes and Professional Sportsmen

A number of authors have published on the outcomes of professional or competitive recreational athletes returning to sport. These are case series rather than randomized controlled trials (RCT) as most professional athletes would not participate in a RCT due to the risk of being randomized to a group with a potentially worse outcome (Table 18.1).

Table 18.1 Series of patients reporting return to play related to the method of management

Author year	Number	Age	Repair method	Return to play	Notes
Martinelli 2000 [46]	30	30.5	TenoLig®	120–150 days	
Gigante et al. 2008 [47]	40	41	Pc (TenoLig® vs. Bunnell)	No difference between groups	
Parekh et al. 2009 [48]	31	29 NFL	Mixed	36% unable	
				50% drop in performance	
De Carli et al. 2009 [49]	20	39.7	Kakiuchi	76.4% same level	
Maffulli et al. 2011 [50]	17	34	Percutaneous	4.8 ± 0.9 months	11 swelling
					4 cramps
Amin et al. 2013 [51]	18	29.7 NBA	Mixed	30% DNR, 11/18 1 season only	
Jallageas et al. 2013 [52]	31	38	Pc vs. open	81% vs. 73.5% same level play	Overall time 153 days (91–246)
Vadala et al. 2014 [53]	36	29.7	Combined pc/mini-open	86% within 5 months	
McCullough et al. 2014 [54]	9	25.6 NFL	Pc	78% within 8.9 months	1 athlete
					166 days
Byrne et al. 2017 [55]	1	36	Pc	Medal winner 18 weeks	

Pc percutaneous, *DNR* did not return, *NBA* National Basketball Association

Ververidis et al. performed a systematic literature search of outcome following percutaneous repair in athletes. Ninety-one percent of patients returned to practising sports, and 78–84% returned to the same or a higher level of sports. From nine studies the average time to return to sports was 18 weeks. The most frequent complication was sural nerve injury (3.3%), and the re-rupture rate was low (2.1%) [56].

18.6 State-of-the-Art-Treatment

Since 2010 the benefits of early rehabilitation, involving early weight-bearing, brace use and early active movement, have been applied to both operative and nonoperative treatments with similar outcome scores and low re-rupture rates.

The most recent studies have focused on the optimization of gastrocnemius-soleus strength, by minimizing tendon lengthening, to reduce the risks of treatment failure.

Lengthening has been noted to occur after nonoperative and operative treatment despite repair augmentation [22, 43].

Cadaveric studies have shown that modes of failure include suture pull-out for non-locked sutures, predominantly from the distal stump, and knot failure for locked Krackow sutures [57–59]. This suggests that the use of locking sutures is advantageous.

State-of-the-art surgery includes using of locking sutures via a percutaneous surgery, direct anchoring into the calcaneum and the avoidance of knots (Table 18.2). The confidence of distal calcaneum fixation and, more recently, the avoidance of knots have contributed to the recent practice of brace-free rehabilitation (Fig. 18.1a, b) [55, 60–66].

18.7 Future Treatment Options

The aim of stable locked sutures with distal intracalcaneal fixation is to provide a stable repair, prevent lengthening and permit early return to activity. These repairs may offer stable initial fixation, but this stability may lead to increased stiffness affecting biomechanical properties during the organization and maturation phases of later healing. Stress shielding may make the tendon more susceptible to proximal ruptures at the musculotendinous junction.

18.8 Post-Operative Care and Rehabilitation Following Achilles Tendon Rupture

Rehabilitation following Achilles tendon rupture should be considered in several phases: controlled mobilization, early recovery, late recovery, and return to sport. Despite these distinctions, contributors to return to sport (i.e. strength deficits, psychosocial concerns) should be considered throughout the rehabilitative process [67, 68]. During later phases of rehabilitation, sport-specific guidelines have not been published in the context of Achilles tendon rupture.

18.8.1 Controlled Motion

The controlled mobilization phase starts directly following injury or surgery. The goal of this phase is to approximate tendon ends and facilitate tendon healing. Of particular concern is the avoidance of tendon elongation. Tendon elongation occurs during the first 8–12 weeks post-injury [29, 31] and results in long-term plantar flexor deficits [16], as well as changes in movement biomechanics, particularly with running and jumping [69]. Early weight-bearing has been associated with less tendon elongation [29]. Early weight-bearing is performed in plantar flexed positions using weight-bearing casts or boots. Later in this phase, the negative effects of immobilization can be addressed with joint mobilization techniques to the talocrural and subtalar joints, taking care not to put the tendon on stretch. Moreover, general hypotrophy can be addressed with active range of motion and isometric strengthening whilst avoiding maximal dorsiflexion.

18.8.2 Early Recovery

The early recovery stage starts when the patient is able to ambulate in sneakers with a wedge, typically around week 6–8. At this stage, slow, controlled weight-bearing exercise

Table 18.2 Outcomes of Achilles tendon repair. These techniques may be considered to provide stable repairs as post-operative brace protection was not used

Author year	Series	No. (*n*=)	Technique	Follow-up	Outcome	RTP	Score
Jennings et al. 2004 [60]	7.75 years	30	Modifed Bunnell and Kessler (transosseous) Using a 10 × 800 mm polyester tape	3.25 years (6 months to 8 years)	Median FWB 42 days Driving 49 days (11–123) Work 35.5 days (4–227)	5 months (3–7 months) 58% returned to ≥ same level activity Mean Tegner pre-injury 3.7, post-injury 3.0	
Yotsumoto et al. 2009 [61]	2004–2006	20	Side-locking loop—Polyblend	2.9 years (2–4.8)	DHR 6.3 weeks 20 SHR 9.9 weeks	14.4 weeks	ATRPS 98.3 at 24 weeks
Doral et al. 2009 [62]	1999–2005	62	Modified Bunnell PDS no. 5 FWB ≥ 3 weeks	46 months (12–78)	Regular work/rehab 11.7 weeks (10–13)	95% return previous activities	AOFAS 94.6
Doral 2013 [63]	1999–2012	69	Modified Bunnell PDS no. 5	28 months (12–72)	No side-to-side difference in hop or muscle strength test	All returned to same sports level	ATRS 86
Jielile et al. 2013 [64]	2001–2011	107	Pa-bone technique Polyglactin	Up to 24 months	Same ROM 7 weeks SHR 60 days SHR 12 weeks	Resume sports activities 13 weeks	ATRPS 93.9 ± 3.4 at 8.5 weeks ATRS 99 at 3 months
Groetelaers et al. 2014 [65]		32	Modified Bunnell proximally and transosseous distally using 1× no. 1 PDS and 1× no. 1 Vicryl	1 year	102% strength compared to uninjured side at 1 year	88% resumed sport 1 year	ATRPS 96% good/excellent at 1 year
Miyamoto et al. 2016 [66]	2011–2014	44	Double side-locking loop suture	24 weeks	DHR 8 ± 1.3 weeks 20SHR 10.9 ± 2.1 weeks	17.1 ± 3.7 weeks, all returned to pre-injury level activities	AOFAS 96.8 ± 4.7

DHR double heel rise, *ATRPS* Achilles Tendon Rupture Performance Score, *AOFAS* American Orthopaedic Foot and Ankle Score, *PDS* polydioxanone suture, *ATRS* Achilles tendon Total Rupture Score, *ROM* range of movement, *SHR* single heel rise

(such as the bilateral heel rise) is initiated to gradually load the tendon. Exercises to address impaired balance, reduced range of motion or strength deficits can also be added. The goal of this stage is to walk symmetrically without bracing and perform activities of daily living (stair negotiation, ambulation in community) without compensation.

18.8.3 Late Recovery

The late recovery stage starts when the patient is able to perform a unilateral heel rise with the goal of gradually progressing strengthening and returning to more dynamic activities, such as running. A running progression can be initiated when the patient is able to complete five

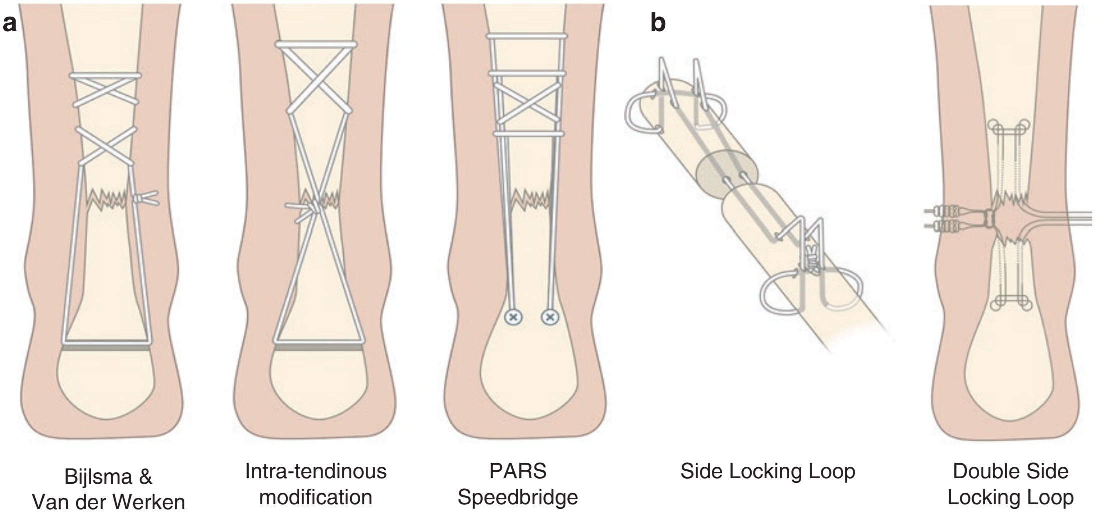


Fig. 18.1 Stable suture configurations, which have reported outcomes (Table 18.2) using either locking sutures or distal calcaneal attachment. (**a**) Bijlsma and van der Werken technique used in Groetelaers et al.'s series [60], modified Bunnell and distal transosseous intra-tendinous suture placement as used in Jennings et al.'s series [65], proximal locking sutures and distal calcaneal anchor fixation [55]. (**b**) Single side-locking loop as used in Yotsumoto et al.'s series [61] and double side-locking loop as used in Miyamoto et al.'s series [66]

unilateral heel rises at 90% of the available height on the ruptured side. If the patient is unable to achieve this milestone by 16 weeks, a running progression can be initiated if the patient is able to raise at least 70% of body weight during a unilateral heel rise [16]. Low-speed, low-intensity agility training (i.e. figure-of-eight jogging, low-speed carioca) can also be initiated during this phase.

The literature comments upon goals to be attained following Achilles tendon rupture. Saxena et al. stated specific targets such as five sets of 25 repetitions. These are introduced over time starting with three sets of ten based upon pain tolerance. The calf circumference, measured 10 cm distal to the inferior pole of the patella, should be comparable to the non-injured limb, and the ankle range of motion should be within 5° of the non-injured side [70]. Return to play was quoted as being between 12 and 26 weeks following Achilles tendon rupture [71].

In Hutchison et al.'s paper on a general population, patients were advised against sports activity until they could perform a single heel rise, sprint using the toe off phase of gait, a horizontal single leg hop three times for more than 75% of the non-injured leg and a vertical hop to greater than 75% of the non-injured side [27].

Return to play cannot be so precisely determined as much as the popular press would have us believe. Rehabilitation is a gradual process, which must follow graduated attainment of strength and functional activities.

How does a clinician determine when an athlete is ready to return to play? And is physical recovery alone enough for return to play?

The question of when to return to play, or more accurately when to return for training for selection to be included in the team play, is frequently considered.

Given that an athlete is likely to have a 6-month absence from his playing position, it is likely that the replacement player will become established in that position. This will also make return to play (RTP) data difficult to interpret.

The player will be keen to return to sports activity and play as well as to return to team training as there will be a considerable graduated increase in performance and play.

Players must have recovered optimal strength to the gastrocnemius-soleus complex for running, sprinting and jumping. The viscoelastic properties of the tendon will have to have optimized as the elastic recoil during loading with distraction could otherwise lead to re-rupture. Additionally, players will need to have an adequate psychological recovery to enable play without hesitation.

18.9 What Is a Successful Return to Play?

The patient or athlete may consider this to be able to perform at the desired level of activity in the desired sport. A number of methods have been used to determine the intensity of activity and sports performance.

The Tegner sport and activity scale was devised as a follow-up indicator after knee ligament injuries [72]. The Tegner score has been commonly used in lower limb sports injury surgery and after 25 years of use is considered to be a valid psychometric parameter. The score has acceptable responsiveness, with an intra-class correlation of 0.8 and minimal detectable change of 1. The Tegner score in its original form has been used for ankle ligament surgery [73], posterior ankle impingement [74] and plantaris injuries [71]. More specifically related to this chapter, the Tegner score has also been used following Achilles tendon injuries [75, 76].

Halasi et al. developed a new activity score for the evaluation of ankle instability in 2004 [77]. This included 53 sports, 3 working activities and 4 general activities inserted into a 0–10-point category scoring system. The level of participation is divided into top level/national team, lower competitive level and finally recreational level. Halasi score correlates with the Tegner score ($r = 0.7565$) and has been found to have high reliability. The ankle score differences were spread over a wider range (−1.18 ± 2.12) than the Tegner score differences (−0.68 ± 1.29), indicating that the new had higher sensitivity.

The Physical Activity Score (PAS) has been described by Grimby and Saltin to assess leisure time physical activity and divides the activity into intensity from light, moderate and hard or very hard exercise [78]. Some authors have also added duration and frequency requirements not included in the original version. The score has recently been found to have concurrent validity with respect to aerobic capacity and movement analysis. The score also has predictive validity to various risk factors for health conditions [78].

A simple method of determining whether patients have returned to the same standard of play is to rate their performance on a scale and to compare this rating with that achieved during follow-up.

Another subjective method is simply to ask patients if they have reached the same level of sports and physical activity or performance as before their injury. Patients are verbally given the options of not yet, the same or improved. This terminology was used so that the patient could decide about their own function in respect to their sports. For example, a competitive footballer may return to the same team, play in the same league and score the same number of goals, but they themselves may feel they have not yet reached the same level of function.

A systematic review and meta-analysis have been performed to identify RTP rates following Achilles tendon rupture and evaluate the measures used to determine RTP. A total of 108 studies encompassing 6506 patients were included for review. Eighty-five studies included a measure for determining RTP. The rate of RTP in all studies was 80% ($CI_{95\%}$, 75–85%). Studies with measures describing determination of RTP reported lower rates than studies without metrics described, with rates being significantly different between groups ($p < 0.001$). Eighty percent of patients returned to play following Achilles tendon rupture. However, the RTP rates are dependent on the quality of the method used to measure RTP. To further understand RTP after Achilles tendon rupture, a standardized, reliable and valid method is required.

The clinician may consider this to be the safe return without re-injury or long-term complications. The perspective of patients/athletes may be different; this could be considered to be to return to work as soon as possible with the avoidance of re-rupture or tendon elongation.

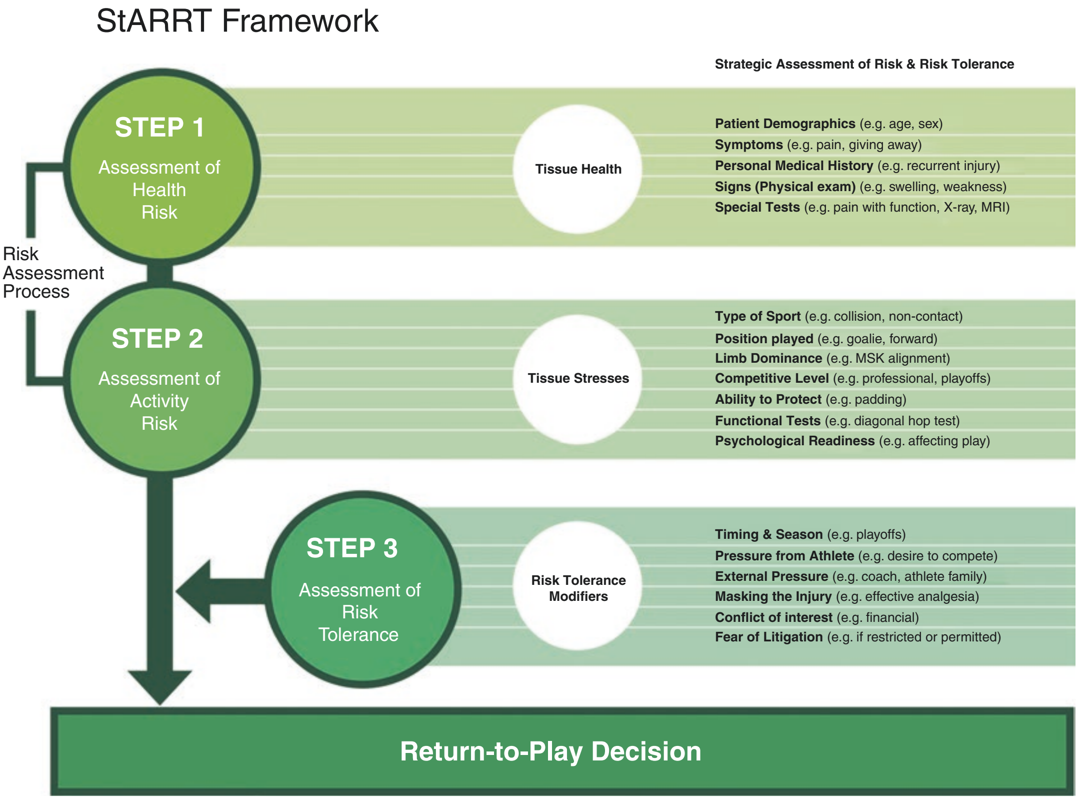


Fig. 18.2 Shrier's Strategic Assessment of Risk and Risk Tolerance (StARRT). Reproduced from Forsdyke et al. [68]

Psychological aspects are very important during the rehabilitation process and RTS. Information from TL data can be used to provide feedback to the player in terms of the ability to cope with on-field-specific activities. The sharing of information with athletes can provide consciousness of the ability to RTP and can reduce anxiety and stress and increase their motivation. Psychological readiness and the "fear of re-injury" may hinder return to the pre-injury level of play.

A useful strategy is to adopt Shrier's Strategic Assessment of Risk and Risk Tolerance (StARRT) framework (Fig. 18.2). This allows demographic, risk activity and risk tolerance all to be assessed to help determine RTP decisions. Additionally, viewing the considerations of the patient and the clinician together with the scientific evidence through an evidence-based practice lens gives additional support and guidance (Fig. 18.3) [68]. There are three key elements to be considered to help decide when an athlete is psychologically ready to return to play [68].

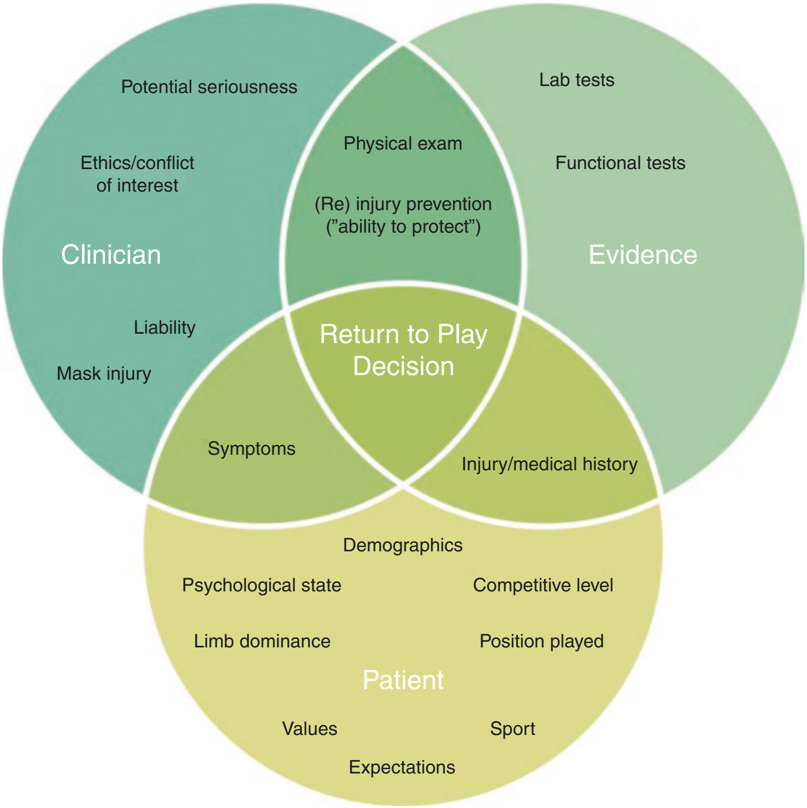
18.10 How Can the Practitioner Best Monitor Abilities?

These include tools for the assessment of performance [72, 77, 78], together with tools for psychological readiness, and one such tool is the Tampa Scale of Kinesiophobia.

18.11 Use the Working Knowledge of the Athlete

Frequently termed "knowing your athlete". The patient or athlete may be observed to be preoccupied, withdrawn or adopt movement patterns in terms of their state of function.

Fig. 18.3 The considerations of the patient and the clinician together with the scientific evidence through an evidence-based practice lens give additional support and guidance. Reproduced from Forsdyke et al. [68]



Adopting an interdisciplinary shared decision-making approach, this allows the patient considerations, the clinician concerns and the requirements of the coach to be borne in mind.

18.12 Take-Home Message

Achilles tendon ruptures are increasing in number and tend to occur in middle-aged persons or in professional athletes at the later stages of their sporting careers. Even when ruptures are "successfully" managed without complication in players, objective muscle strength weakness is almost always present.

In sports, where any loss of muscle strength leads to a considerable disadvantage, operative repair is most probably the management method of choice.

Although return to play at the same level is possible for many recreational athletes, this may be difficult for a professional footballer.

References

1. Lantto I, Heikkinen J, Flinkkilä T, Ohtonen P, Leppilahti J. Epidemiology of Achilles tendon ruptures: increasing incidence over a 33-year period. Scand J Med Sci Sports. 2015;25(1):e133–8.
2. Maffulli N, Waterston SW, Squair J, Reaper J, Douglas AS. Changing incidence of Achilles tendon rupture in Scotland: a 15 year study. Clin J Sport Med. 1999;9(3):157–60.
3. Huttunen TT, Kannus P, Rolf C, Felländer-Tsai L, Mattila VM. Acute Achilles tendon ruptures: incidence of injury and surgery in Sweden between 2001 and 2012. Am J Sports Med. 2014;42(10):2419–23.
4. Lantto I, Heikkinen J, Flinkkilä T, Ohtonen P, Kangas J, Siira P, Leppilahti J. Early functional treatment versus cast immobilization in tension after Achilles rupture repair: results of a prospective randomized trial with 10 years or more of follow up. Am J Sports Med. 2015;43(9):2302–9.
5. Mattila VM, Huttunen TT, Haapasalo H, Sillanpää P, Malmivaara A, Pihlajamäki H. Declining incidence of surgery for Achilles tendon rupture follows publication of major RCTs: evidence influenced change evident using the Finnish registry study. Br J Sports Med. 2015;49(16):1084–6.

6. Scott A, Grewal N, Guy P. The seasonal variation of Achilles tendon ruptures in Vancouver, Canada: a retrospective study. BMJ. 2014;4(2):e004320.
7. Sheth U, Wasserstein D, Jenkinson R, Moineddin R, Kreder H, Jaglal SB. The epidemiology and trends in management of acute Achilles tendon ruptures in Ontario, Canada: a population based study of 27,607 patients. Bone Joint J. 2017;99-B(1):78–86.
8. Suchak AA, Bostick G, Reid D, Blitz S, Jomha N. The incidence of Achilles tendon ruptures in Edmonton, Canada. Foot Ankle Int. 2005;26(11):932–6.
9. Wang D, Sandlin MI, Cohen JR, Lord EL, Petrigliano FA, SooHoo NF. Operative versus non-operative treatment of acute Achilles tendon rupture: an analysis of 12,570 patients in a large healthcare database. Foot Ankle Surg. 2015;21(4):250–3.
10. Ganestam A, Kallemose T, Troelsen A, Barfod KW. Increasing incidence of acute Achilles tendon rupture and a noticeable decline in surgical treatment form 1994-2013. A nationwide registry study of 33,160 patients. Knee Surg Sports Traumatol Arthrosc. 2016;24(12):3730–7.
11. Józsa L, Kvist M, Bálint BJ, Reffy A, Järvinen M, Lehto M, Barzo M. The role of recreational sport activity in Achilles tendon rupture. A clinical pathoanatomical and sociological study of 292 cases. Am J Sports Med. 1989;17(3):338–43.
12. Carmont MR, Silbernagel KG, Edge A, Mei-Dan O, Karlsson J, Maffulli N. Functional outcome of percutaneous Achilles repair: improvments in achilles tendon total rupture score during the first year. Orthop J Sports Med. 2013;1(1):2325967113494584.
13. Vosseller JT, Ellis SJ, Levine DS, Kennedy JG, Elliott AJ, Deland JT, Roberts MM, O'Malley MJ. Achilles tendon rupture in women. Foot Ankle Int. 2013;34(1):49–53.
14. Heikkinen J, Lantto I, Flinkkila T, Ohtonen P, Niinimaki J, Siira P, Laine V, Leppilahti J. Soleus atrophy is common after the non-surgical treatment of acute Achilles tendon ruptures: a randomized controlled trial comparing surgical and nonsurgical functional treatments. Am J Sports Med. 2017;45(6):1395–404.
15. Olsson N, Nilsson-Helander K, Karlsson J, Eriksson BI, Thomee R, Faxen E, Silbernagel KG. Major functional deficits persist 2 years after acute Achilles tendon rupture. Knee Surg Sports Traumatol Arthrosc. 2011;19:1385–93.
16. Silbernagel KG, Steele R, Manal K. Deficits in heel rise height and Achilles tendon elongation occur in patients recovering from an Achilles tendon rupture. Am J Sports Med. 2012;40(7):1564–71.
17. Willits K, Amendola A, Bryant D, Mohtadi NG, Giffin JR, Fowler P, Kean CO, Kirkley A. Operative versus non-operative treatment of acute Achilles tendon ruptures: a multicenter randomized trial using accelerated functional rehabilitation. J Bone Joint Surg Am. 2010;92:767–75.
18. Bostick GP, Jomha NM, Suchak AA, Beaupre LA. Factors associated with calf muscle endurance recovery 1 year after Achilles tendon rupture repair. J Orthop Sports Phys Ther. 2010;40(6):345–51.
19. Horstmann T, Lukas C, Merk J, Brauner T, Mundermann A. Deficits 10 years after Achilles tendon repair. Int J Sports Med. 2012;33(6):474–9.
20. Suydam SM, Buchannan TS, Manal K, Silbernagel KG. Compensatory muscle activation caused by tendon lengthening post Achilles tendon rupture. Knee Surg Sports Traumatol Arthrosc. 2015;23(3):868–74.
21. Barfod KW, Sveen TM, Ganestam A, Ebskov LB, Troelsen A. Severe functional debilitations after complications associated with acute Achilles tendon ruptures with 9 years of follow up. J Foot Ankle Surg. 2017;56(3):440–4.
22. Heikkinen J, Lantto I, Flinkkilä OP, Pajala A, Siira P, Leppilahti J. Augmented compared to non-augmented surgical repair after total Achilles rupture: results of a prospective randomized trial with 13 or more years of follow up. J Bone Joint Surg Am. 2106;98(2):85–92.
23. Mavrodontidis A, Lykissas M, Koulouvaris P, Pafilas D, Kontogeorgakos V, Zalavras C. Percutaneous repair of acute Achilles tendon rupture: a functional evaluation study with a minimum 10 year follow up. Acta Orthop Traumatol Turc. 2015;49(6):661–7.
24. Zellers J, Carmont MR, Grävare Silbernagel K. Return to play post Achilles tendon rupture: a systematic review and meta-analysis of rate and measures of return to play. Br J Sports Med. 2016;50(21):1325–32.
25. Barfod KW, Bencke J, Lauridsen HB, Ban I, Ebskov L, Troelsen A. Non-operative dynamic treatment of the acute Achilles tendon rupture: the influence of early weight-bearing on clinical outcome: a blinded randomized controlled trial. J Bone Joint Surg Am. 2014;96(11):1497–503.
26. Ecker TM, Bremer AK, Krause FG, Müller WM. Prospective use of a standardized non-operative early weight bearing protocol for Achilles tendon rupture: 17 years of experience. Am J Sports Med. 2016;44(4):1004–10.
27. Hutchison AM, Topliss C, Beard D, Evans RM, Williams P. The treatment of a rupture of the Achilles tendon using a dedicated management programme. Bone Joint J. 2015;97-B(4):510–5.
28. Wallace RG, Heyes GJ, Michael AL. The non-operative functional management of patients with a rupture of the tendo Achillis leads to low rates of re-rupture. J Bone Joint Surg. 2011;93(11):1362–6.
29. Kangas J, Pajala A, Ohtonen P, Leppilahti J. Achilles tendon elongation after rupture repair: a randomized comparison of two post operative regimens. Am J Sports Med. 2007;35(1):59–64.
30. Majewski M, Schaeren S, Kohlhaas U, Oschner PE. Postoperative rehabilitation after percutaneous Achilles tendon repair: early functional therapy versus cast immobilization. Disabil Rehabil. 2008;30(20–22):1726–32.
31. Mortensen H, Skov O, Jensen P. Early motion of Achilles after operative treatment of a rupture of the Achilles tendon: a prospective randomized clini-

cal and radiographic study. J Bone Joint Surg Am. 1999;81(7):983–90.

32. Suchak AA, Bostick GP, Beaupré LA, Durand DC, Jomha NM. The influence of early weight bearing compared with non-weight bearing after surgical repair of the Achilles tendon. J Bone Joint Surg Am. 2008;90:1876–83.
33. Lantto I, Heikkinen J, Flinkkilä T, Ohtonen P, Kangas J, Siira P, Laine V, Leppilahti J. A prospective randomized controlled trial comparing surgical and nonsurgical treatment of acute Achilles tendon ruptures. Am J Sports Med. 2016;44(9):2406–14.
34. Olsson N, Silbernagel KG, Eriksson BI, Sansone M, Brorsson A, Nilsson-Helander K, Karlsson J. Stable surgical repair with accelerated rehabilitation versus non-surgical management for acute Achilles tendon ruptures: a randomized controlled study. Am J Sports Med. 2013;41(12):2867–76.
35. Schepull T, Kvist J, Aspenberg P. Early E modulus of healing Achilles tendons correlates with late function: similar results with or without surgery. Scand J Med Sci Sports. 2012;22(1):18–23.
36. Soroceanu A, Sidhwa F, Aarabi S, Kaufman A, Glazebrook M. Surgical versus non-surgical treatment of acute Achilles tendon rupture: a meta-analysis of randomized trials. J Bone Joint Surg Am. 2012;94(23):2136–43.
37. Metz R, van der Heijden GJ, Verleisdonk EJ, Andrlik M, van der Werken C. Persistent disability despite sufficient calf muscle strength after re-rupture of surgically treated acute Achilles tendon ruptures. Foot Ankle Spec. 2011;4(2):77–81.
38. Erickson BJ, Mascarenhas R, Saltzman BM, Walton D, Lee S, Cole BJ, Bach BR Jr. Is operative treatment of Achilles tendon ruptures superior to non-operative treatment?: a systematic review of overlapping meta-analyses. Orthop J Sports Med. 2015;3(4):2325967115.
39. Keating JF, Will EM. Operative versus non-operative treatment of acute rupture of tendo Achillis: a prospective randomized evaluation of functional outcome. J Bone Joint Surg (Br). 2011;93(8):1071–8.
40. Del Buono A, Volpin A, Maffulli N. Minimally invasive versus open surgery for acute Achilles tendon rupture: a systematic review. Br Med Bull. 2014;109:45–54.
41. Li Q, Wang C, Huo Y, Jia Z, Wang X. Minimally invasive versus open surgery for acute Achilles tendon rupture: a systematic review of overlapping meta-analyses. J Orthop Surg Res. 2016;11(1):65.
42. McMahon SE, Smith TO, Hing CB. A meta-analysis of randomized controlled trials comparing conventional to minimally invasive approaches for repair of an Achilles tendon rupture. Foot Ankle Surg. 2011;17(4):211–7.
43. Pajala A, Kangas J, Siira P, Ohtonen P, Leppilahti J. Augmented compared with non-augmented surgical repair of a fresh total Achilles tendon rupture. A prospective randomized study. J Bone Joint Surg Am. 2009;91(5):1092–100.
44. Zhang YJ, Zhang C, Wang Q, Lin XJ. Augmented versus nonaugmented repair of acute achilles tendon rupture: a systematic review and meta-analysis. Am J Sports Med. 2017;2017:0363546517702872.
45. Olsson N, Petzold M, Brorsson A, Karlsson J, Eriksson BI, Silbernagel KG. Predictive of clinical outcome after acute Achilles tendon ruptures. Am J Sports Med. 2014;42(6):1448–55.
46. Martinelli B. Percutaneous repair of the Achilles tendon in athletes. Bull J Hosp Dis. 2000;59(3):149–52.
47. Gigante A, Moschini A, Verdenelli A, Del Torto M, Ulisse S, de Palma L. Open versus percutaneous repair in the treatment of acute Achilles tendon rupture: a randomized prospective study. Knee Surg Sports Traumatol Arthrosc. 2008;16(2):204–9.
48. Parekh SG, Wray WH III, Brimmo O, Sennett BJ, Wapner KL. Epidemiology and outcomes of Achilles tendon ruptures in the national football league. Foot Ankle Spec. 2009;2(6):283–6.
49. DeCarli A, Vadalà A, Ciardini R, Iorio R, Ferretti A. Spontaneous Achilles tendon ruptures treated with a mini-open technique: clinical and functional evaluation. J Sports Med Phys Fitness. 2009;49(3):292–6.
50. Maffulli N, Longo UG, Maffulli GD, Khanna A, Denaro V. Achilles tendon ruptures in elite athletes. Foot Ankle Int. 2011;32(1):9–15.
51. Amin NH, Old AB, Tabb LP, Garg R, Toossi N, Cerynik DL. Performance outcomes after repair of complete Achilles tendon ruptures in national basketball association players. Am J Sports Med. 2013;41(8):1864–8.
52. Jallageas R, Bordes J, Daviet JC, Mabit C, Coste C. Evaluation of surgical treatment for ruptured Achilles tendon in 31 athletes. Orthop Traumatol Surg Res. 2013;99(5):577–84.
53. Vadalà A, Lanzetti RM, Ciompi A, Rossi C, Lupariello D, Ferretti A. Functional evaluation of professional athletes treated with a mini-open technique for Achilles tendon rupture. Musc Ligam Tendons J. 2014;4(2):177–81.
54. McCullough KA, Shaw CM, Anderson RB. Min-open repair of Achilles rupture in the national football league. J Orthop Surg Adv. 2014;23(4):179–83.
55. Byrne PA, Hopper GP, Wilson WT, Mackay GM. Knotless repair of Achilles tendon rupture in an elite athlete: return to competition in 18 weeks. J Foot Ankle Surg. 2017;56(1):121–4.
56. Ververidis AN, Kalifis KG, Touzopoulos P, Drosos GI, Tilkeridis KE, Kazakos KI. Percutaneous repair of the Achilles tendon rupture in athletic population. J Orthop. 2015;13(1):57–61.
57. Clanton TO, Haytmanek CT, Williams BT, Civitarese DM, Turnbull TL, Massey MB, Wijdicks CA, LaPrade RF. A biomechanical comparison of an open repair and 3 minimally invasive percutaneous achilles tendon repair techniques during a simulated, progressive rehabilitation protocol. Am J Sports Med. 2015;43(8):1957–64.
58. Longo UG, Forriol F, Campi S, Maffulli N, Denaro V. A biomechanical comparison of the primary stabil-

ity of two minimally invasive techniques for repair of ruptured Achilles tendon. Knee Surg Sports Traumatol Arthrosc. 2012;20(17):1392–7.
59. Ortiz C, Wagner E, Mococain P, Labarca G, Keller A, Del Buono A, Maffulli N. Biomechanical comparison of four methods of repair of the Achilles tendon: a laboratory study with bovine tendons. J Bone Joint Surg (Br). 2012;94(5):663–7.
60. Jennings AG, Sefton GK, Newman RJ. Repair of acute rupture of the Achilles tendon: a new technique using polyester tape without external splintage. Ann R Coll Surg Engl. 2004;86:445–8.
61. Yotsumoto T, Miyamoto W, Uchio Y. Novel approach to repair of acute Achilles tendon rupture: early recovery without post-operative fixation or orthosis. Am J Sports Med. 2010;38(2):287–92.
62. Doral MN, Bozkurt M, Turhan E, Ayvaz M, Atay OA, Uzümcügil A, Leblebicioglu G, Kaya D, Avdog T. Percutaneosu suturing of the ruptured Achilles tendon with endoscopic control. Arch Orthop Trauma Surg. 2009;129(8):1093–101.
63. Doral MN. What is the effect of the early weight-bearing mobilization without using any support after endoscopy-assisted Achilles tendon repair? Knee Surg Sports Traumatol Arthrosc. 2013;21(6):1378–84.
64. Jielile J, sabirhazi G, Chen J, Aldyarhan K, Zheyiken J, Zhao Q, Bai J. Novel surgical technique and early kinesiotherapy for acute Achilles tendon rupture. Foot Ankle Int. 2012;33(12):1119–27.
65. Groetelaers RP, Jansson L, van der Velden J, Wieland AW, Amendt AG, Gaelen PH, Janzing HM. Functional treatment or cast immobilization after minimally invasive repair of an acute Achilles tendon rupture: prospective randomized trial. Foot Ankle Int. 2014;35(8):771–8.
66. Miyamoto W, Imade S, Innami K, Kawano H, Takao M. Acute Achilles tendon rupture treated by double side-locking loop suture technique with early rehabilitation. Foot Ankle Int. 2017;38(2):167–73.
67. Ardern CL, Glasgow P, Schneiders A, Witvrouw E, Clarsen B, Cools A, Gojanovic B, Griffin S, Khan KM, Moksnes H, Mutch SA, Phillips N, Reurink G, Sadler R, Silbernagel KG, Thorburg K, Wangensteen A, Wilk KE, Bizzini M. Consensus statement on return to sport from the first world congress in physical therapy, Bern. Br J Sports Med. 2016;50(14):853–64.
68. Forsdyke D, Gledhill A, Ardern C. Psychological readiness to retrun to sport: three key elements to help the practitioner decide whether the athlete is really ready. Br J Sports Med. 2017;51(7):555–6.
69. Willy RW, Brorsson A, Powell HC, Wilson JD, Tranberg R, Grävare Silbernagel K. Elevated knee joint kinematics and reduced ankle kinematics are present during jogging and hopping after Achilles tendon ruptures. Am J Sports Med. 2017;45(5): 1124–33.
70. Saxena A, Ewan B, Maffulli N. Rehabilitation of the operated Achilles tendon: parameters for predicting return to activity. J Foot Ankle Surg. 2011;50(1): 37–40.
71. Pollock N, Dijkstra P, Calder J, Chakraverty R. Plantaris injuries in elite UK track and field athletes over a 4 year period: a retrospective cohort study. Knee Surg Sports Traumatol Arthrosc. 2016;24(7): 2287–92.
72. Tegner Y, Lyholm J. Rating systems in the evaluation of knee ligament injuries. Clin Orthop Relat Res. 1985;198:43–9.
73. Matheny LM, Johnson NS, Liechti DJ, Clanton TO. Activity level and function after lateral ankle ligament repair versus reconstruction. Am J Sports Med. 2016;44(5):1301–8.
74. López Valerio V, Sejias R, Alvarez P, Ares O, Steinbacher G, Salient A, Cugat R. Endoscopic repair of posterior ankle impingement syndrome due to os trigonum in soccer players. Foot Ankle Int. 2015;36(1):70–4.
75. Ibrahim SA. Surgical treatment of chronic Achilles tendon rupture. J Foot Ankle Surg. 2009;48(3): 340–6.
76. Kosanovic M, Brilej D. Chronic rupture of Achilles tendon: is the percutaneous technique effective. Arch Orthop Trauma Surg. 2008;128(2):211–6.
77. Halasi T, Kynsburg A, Tállay A, Berkes I. Development of a new activity score for the evaluation of ankle instability. Am J Sports Med. 2004;32(4): 899–908.
78. Grimby G, Börjesson M, Jonsdottir IH, Schnohr P, Thelle DS, Saltin B. The “Saltin-Grimby physical activity scale” and its application to health research. Scand J Med Sci Sports. 2015;25(S4):119–25.

19

Management of Less Frequent and Multi-ligament Knee Injuries

Steve Bollen, Sam Oussedik, William Hage,
James Robinson, Manuel Leyes,
and Joan C. Monllau

Overview

The knee with multi-ligament injuries is a complex problem with a significant potential morbidity in terms of pain, instability and dysfunction. For some injury patterns, there is consensus as to whether to repair or reconstruct the damaged soft tissues, whilst for others, best treatment strategies remain unclear.

The following chapter aims to highlight a rational approach to these lesions drawn from both the experience of the authors and the current literature.

19.1 Isolated PCL Injury

Steve Bollen

19.1.1 Introduction

Isolated PCL injury occurs less frequently than other common ligament injuries. In Myasaka's 1991 paper [1], looking at 500 knees with "pathologic motion", about half the injuries were ACL tears, but isolated PCL injury only occurred in 18 patients. The injury may however be more prevalent as many patients can sustain isolated PCL injury and recover function without ever having the diagnosis made. Unlike the ACL, the PCL rarely ruptures mid-substance, and it frequently attenuates within its length, allowing the possibility of healing.

Patients may present in their 50s, complaining of medial and/or patellofemoral pain, with undiagnosed PCL laxity that they were completely unaware of. Going back through their history, there is often an injury they can recall that happened 20 or 25 years previously, which stopped them playing for 6–12 weeks. Isolated PCL injury has a high prevalence in professional sports such as rugby league. In the rugby league squads the author looks after, between 20 and 30% of the players have a ruptured PCL in one knee or the other and sometimes both! Many may continue to function at high levels and have a

S. Bollen
Yorkshire Clinic, Bingley, West Yorkshire, UK

S. Oussedik
University College Hospital, London, UK

W. Hage
North Cumbria University Hospitals, Cumbria, UK

J. Robinson
Avon Orthopaedic Centre, Bristol, UK

International Knee and Joint Center,
Abu Dhabi, UAE

M. Leyes
Clinica CEMTRO, Madrid, Spain

J.C. Monllau (✉)
Hospital del Mar and Hospital Universitari Dexeus,
UniversitatAutònoma de Barcelona, Barcelona, Spain
e-mail: JMonllau@parcdesalutmar.cat

G.M.M.J. Kerkhoffs et al. (eds.), *ESSKA Instructional Course Lecture Book*,
https://doi.org/10.1007/978-3-662-56127-0_19

normal longevity of their career without surgical intervention. Long-term follow-up (mean 18 years) of patients with isolated PCL laxity shows that good function and normal range of motion are often maintained and only 10% have moderate to severe arthritis. Importantly, this is not correlated with the degree of PCL laxity [2].

This is where the problem lies, as demonstrably it is possible to return to the very highest level of sporting performance without any surgical intervention.

19.1.2 Biomechanics

PCL deficiency leads to increased contact pressure in the patellofemoral and medial compartments leading, in the long term (usually 20–25 years), to osteoarthritis in these compartments.

Fixed posterior subluxation of the tibia may develop with time; however, Ogata et al. [3] showed that in the acute stage the knee will always reduce in extension providing the collateral structures are intact—the basis for conservative management.

19.1.3 Proprioception

Given that loss of proprioception is felt to be a major factor in symptomatology in ACL deficiency, it might be expected that PCL-deficient knees would have little proprioceptive deficit. However, an MD thesis project by Karen May in the mid-1990s found exactly the opposite, showing that the players with PCL deficiency had a marked proprioceptive deficit in the affected knee (Fig. 19.1)!

Fig. 19.1 Patients with PCL deficiency are found to have measurable proprioceptive deficits

19.1.4 History

Classically, this is a fall onto a flexed knee or a dashboard-type injury. In sport the player may be able to carry on and any swelling may be mild. There is often pain felt in the popliteal fossa made worse by knee flexion. Patients with PCL injury may not present following the acute injury, or the injury may be missed leading to delayed presentation. It is possible for the diagnosis to be made years later without them ever having been aware they had sustained a significant injury (Fig. 19.2).

In chronic cases, pain in both the patellofemoral joint and medial compartment is usually the predominant feature.

19.1.5 Examination

In acute cases, the most sensitive sign is reduced tibial step-off: palpating the distance between the anteromedial aspect of the tibial plateau and the medial femoral condyle when the knee is at 90 degrees of flexion. There is usually tenderness in the popliteal fossa and increased pain on knee flexion. Posterior tibial drawer at 90 degrees of knee flexion usually reveals a soft end feel. It is

Fig. 19.2 It is possible to participate in elite level sports with isolated PCL deficiency

important to ensure there is no coupled rotational laxity (i.e. the tibia moves directly posteriorly without rotating at the same time), indicating associated injury. Posterior drawer of >1 cm also points to associated injury of the posterolateral or posteromedial corners. It is vital to exclude associated laxity.

In chronic cases, posterior tibial sag with the knee at 90° of flexion is usually obvious.

19.1.6 Radiological Diagnostics

Plain radiographs may show an obvious sag on the lateral radiograph, and in more chronic cases, skyline views of the patellofemoral joint and AP weight-bearing films may show joint space narrowing in the patellofemoral joint and medial tibiofemoral joint.

In acute cases, plain radiographs may also identify a PCL avulsion fracture. It is important to pick this up, as early open reduction and fixation stand a very good chance of restoring near-normal laxity.

MRI may be able to identify injury to the PCL in acute cases but misses the diagnosis in about 50% of chronic cases [4]. In acute cases, it can also be useful in identifying associated injuries, but more than 12 weeks from index injury can miss associated posterolateral corner injury in most cases [5].

19.1.7 State-of-the-Art Treatment

With an acute injury, it is vital to exclude associated injury as this can influence management. Whilst MRI may help identify injury to associated structures, it does not assess laxity, so careful clinical assessment remains the cornerstone of decision-making.

Isolated PCL avulsion fracture is best treated by open reduction and fixation as the energy of the injury is dissipated through the fracture, and the ligament itself remains largely intact. The author prefers the modified Burks and Schaffer [6] approach to reattach the avulsed bone fragment (Fig. 19.3).

For cases of acute, isolated intra-substance injury, the author's preferred method of treatment

Fig. 19.3 Post-operative lateral radiograph following open reduction and internal fixation of a PCL tibial avulsion fracture

is to brace the knee in extension for 4 weeks as this generally results in a much-reduced posterior laxity. It is vital to check if the knee has reduced anatomically (which it will do if there are no associated injuries), as to brace a knee that is subluxed posteriorly will result in a fixed posterior subluxation—with poor results. Dynamic PCL bracing, for example, the Jack brace (Albrecht) or the PCL Rebound brace (Ossür), has become popular but may not be well tolerated and has also been shown not to confer any advantage in the immediate acute post-injury period according to Ogata et al. [3].

After 4 weeks, the knee can be mobilised. Open chain hamstring exercises should be avoided, and flexion may be slow to return. With increasing activity levels and return to running, players often develop some popliteal fossa pain. This usually fades with time, and return to playing is usually around the 12-week mark.

Very occasionally, patients develop persistent popliteal fossa pain, which may be associated with the development of granulation tissue around the injury site. Generally, this responds to guided injection.

In the author's 25 years' experience of managing isolated PCL injuries in sportsmen, the indications for early reconstruction are extremely rare, and only three PCL reconstructions for isolated rupture have been performed.

Functional problems are rare, but persistent patellofemoral pain, made worse on descending stairs and not responding to quad strengthening, is perhaps one of the few indications.

Whilst there are surgeons who advocate reconstruction for all PCL injuries, isolated or combined, there is little evidence that, for isolated injuries, the 20-year results are any better than conservative management. There is certainly no literature that shows PCL reconstruction prevents the progression of degenerative change.

19.1.8 Take-Home Message

Current techniques of PCL reconstruction are not reliable in returning knee laxity to normal and do not recreate normal knee biomechanics [7, 8]. They also have a definite morbidity and involve a very lengthy period of rehabilitation [8, 9].

The problem is that at the current time we do not have a way of identifying those patients who will become significantly symptomatic and therefore might benefit from early reconstruction. However, the reported high percentage of good results [10] from conservative treatment should be kept in mind.

19.2 PCL Reconstruction: Technical Aspects

Sam Oussedik

19.2.1 Introduction

Symptomatic PCL insufficiency is rarely seen in isolated injuries. Reconstruction outside of multi-ligament injuries is therefore unusual and the literature pertaining to PCL reconstruction less well-developed than ACL reconstruction. The varied nature of multi-ligament injuries makes unbiased evaluation of outcomes following different PCL reconstructive techniques much more difficult to achieve. Certain principles can, however, be determined from a review of the literature and established clinical practice.

19.2.2 Patterns of Injury and Diagnosis

The commonest mechanisms of injury are contact sports injuries and motor vehicle accidents. These impart large amounts of kinetic energy producing a posterior translation, rotation and/or hyperextension force on the knee.

A recent review of traumatic knee injuries reported that 79% of multi-ligament knee injuries involve the PCL [10]. Of these, the frequency of other injured ligaments includes 46% for ACL, 31% for MCL and 62% for concomitant PLC injuries [11].

Clinical examination should include tests for sagittal, coronal and axial plane laxity. PCL injury will result in posterior tibial sag and increased posterior translation on posterior drawer testing. Applied varus and valgus force should be used at 0 and 30 degrees of knee flexion to evaluate MCL and LCL integrity, as well as capsular integrity. Rotational laxity can be evaluated by use of the reverse pivot shift and dial test.

Imaging studies should include plain films, on which bony avulsions can be readily identified, angiography in the presence of reduced ankle-brachial pressure indices and CT in the presence of concomitant fractures. MRI offers the most accurate imaging of the damaged soft tissue structures [12].

A combination of clinical examination findings and imaging results is used to plan operative reconstruction.

19.2.3 PCL Reconstruction

Reconstruction techniques aim to recreate anatomy to a greater or lesser extent. The native PCL is a broad structure with two functional bundles, although many more anatomical bundles can be distinguished in cadaveric studies. No graft currently exists that can recreate this structure in all of its complexity [7, 8].

Double-bundle reconstruction techniques have evolved as a possible solution to recreating the functional anatomy of the native PCL. However, in the setting of multi-ligament injury and reconstruction, the additional benefits over single-bundle techniques are yet to be proven [13, 14]. The additional operative time and increased number of tunnels, particularly if further medial reconstructive procedures are required, mean that a single-bundle technique remains the author's choice in this setting.

Biomechanical studies have suggested that the anterolateral bundle (ALB) is the most powerful restraint to both posterior tibial translation and rotation [15], and therefore single-bundle reconstruction techniques aim to recreate this structure (Fig. 19.4).

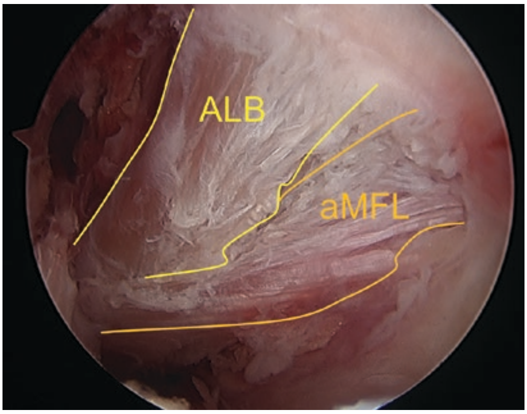


Fig. 19.4 Arthroscopic view of the lateral aspect of the medial femoral condyle. *ALB* anterolateral bundle, *aMFL* anterior meniscofemoral ligament

19.2.4 Technical Aspects of Reconstruction

Graft choices for PCL reconstruction in the setting of multi-ligament injury include autograft, allograft and synthetics. Each option has its own merits and limitations. With multiple reconstructions, autograft options can be exhausted, and the addition of donor site morbidity to the traumatic injury is often better avoided. Appropriately treated allograft material offers a good option both in terms of avoiding further trauma and allowing a larger graft length and diameter, closer to the native ligament's dimensions. Good results can be achieved with Achilles tendon allograft with the additional benefit of making use of the calcaneal bone block for osseous fixation or tibial inlay [16]. Although reasonable results have been reported with synthetic ligament substitutes, some concerns remain regarding possible synovitis and overconstraint [17].

Autograft options include ipsilateral hamstrings, contralateral hamstrings or quadriceps tendon.

19.2.5 Technical Tips and Pearls

Single-bundle reconstruction aims to recreate the largest, anterolateral bundle. Double-bundle techniques recreate both anterolateral and posteromedial functional bundles. Although testing data exist that show evidence of improved laxity measurements in double-bundle reconstruction, no good clinical data exist [7, 8]. Current practice is to offer double bundle in symptomatic isolated PCL rupture and single bundle for multi-ligament reconstruction [7–9].

The tibial side of the graft can be placed by transtibial drilling or by establishing a socket for inlay of the graft [10]. Inlay techniques can be open or arthroscopic. Inlay techniques have the advantage of avoiding the "killer turn" encountered with transtibial techniques [10–12] (Fig. 19.5).

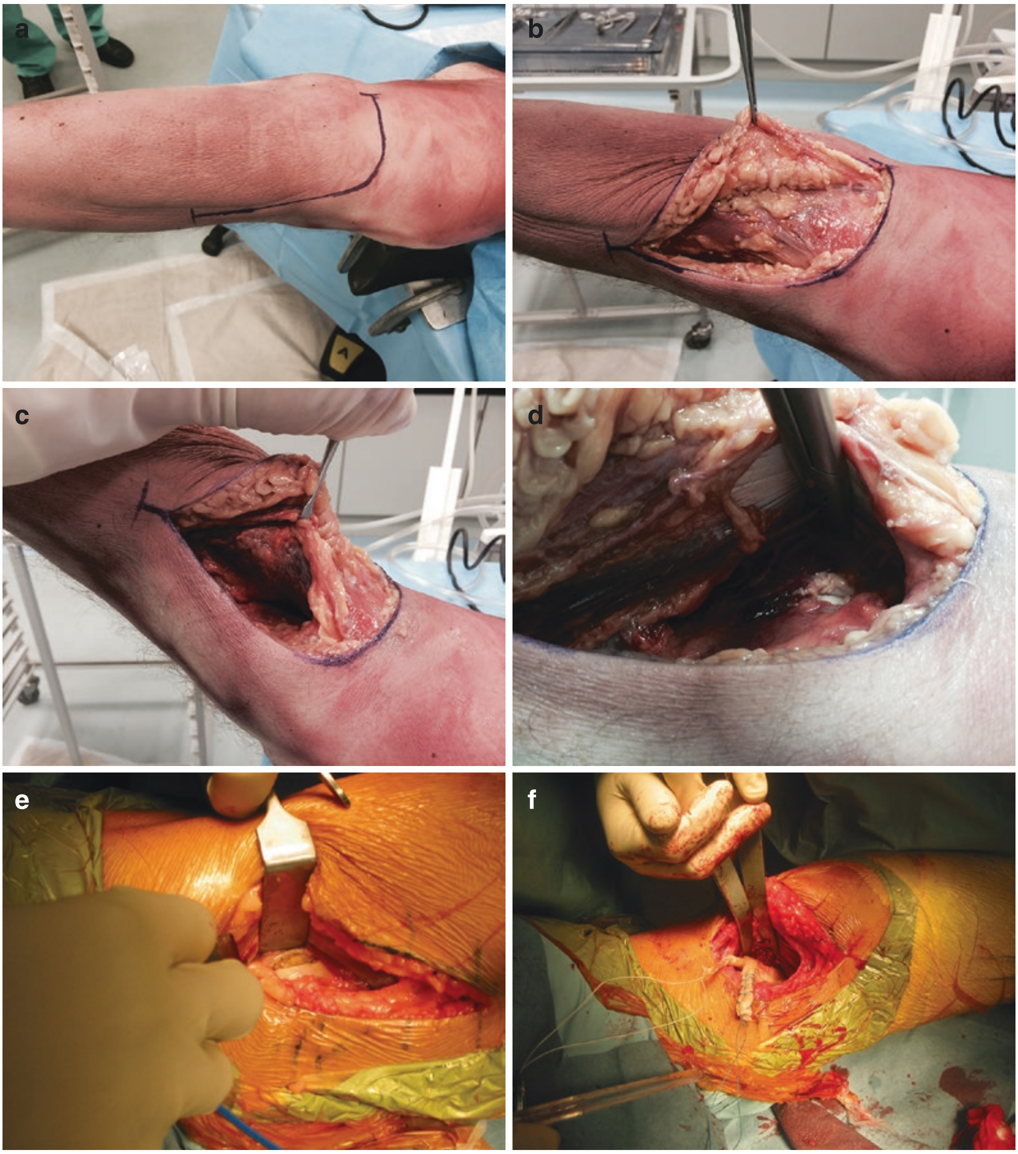


Fig. 19.5 (**a**) An incision is planned following the subcutaneous border of the medial gastrocnemius merging into the popliteal crease. (**b**) A full-thickness flap is developed. (**c**) The medial head of gastrocnemius is elevated. (**d**) The posterior capsule is incised at the level of the joint revealing the PCL fossa (arrow). (**e**) Intraoperative photo of the exposed fossa. Note a smaller incision may be employed in vivo. (**f**) The bone block of an Achilles allograft has been secured with two bone screws. The tendon has been prepared as two grafts, ready for a double-bundle reconstruction. These are fed into the intercondylar notch prior to closure, repositioning the patient supine and standard arthroscopic preparation of the femoral tunnels

Transtibial drilling can be achieved by arthroscopic visualisation through a posteromedial portal or using image intensifier control. Establishing the posteromedial portal (Fig. 19.6) is a simple task, and its location, superior to the joint line to allow access to the back of the tibia, is critical [12, 13]. Once established, an arthroscopic cannula can be passed to avoid losing the portal track, particularly useful in larger patients.

Posterior vascular structures are at risk during tibial drilling; the tip of the guide-wire must remain captured by an appropriate tool to avoid migration [12, 13]. The posterior capsule can be released to aid visualisation [14].

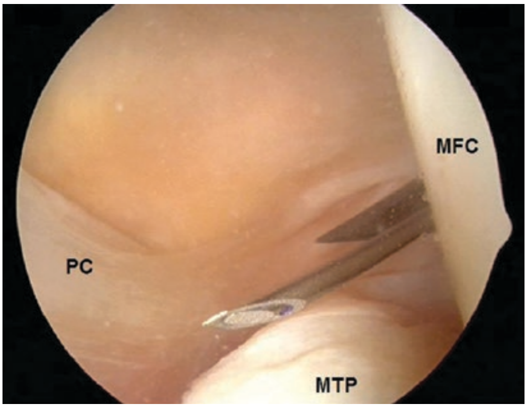


Fig. 19.6 Arthroscopic view of posteromedial portal placement. *MFC* medial femoral condyle, *MTP* medial tibial plateau, *PC* posterior capsule

Passing the graft can be challenging, particularly if a transtibial technique is employed. To this end, a smoother can be used to ease passage, together with placing a trochar through the posteromedial portal over which the graft can be levered.

If medial collateral surgery is also planned, it is important to avoid tunnel coalescence when preparing the femoral tunnels. To this end, outside-in guides can be employed for femoral drilling, through the medial MCL reconstruction incision, ensuring the tunnel aperture is distal to the medial epicondyle (Fig. 19.7).

A competent PCL is necessary to restore the normal tibiofemoral relationship. As such, fixation of the PCL reconstruction is undertaken prior to any other ligament reconstructions in multi-ligament injuries.

Post-operative rehabilitation avoids active hamstring exercises to reduce posterior translational force on the tibia and subsequent graft stretching. Passive prone flexion exercises are used for 6–12 weeks to regain range of motion whilst avoiding strong hamstring contraction [15].

Reproducible results can be achieved if these principles are respected [16].

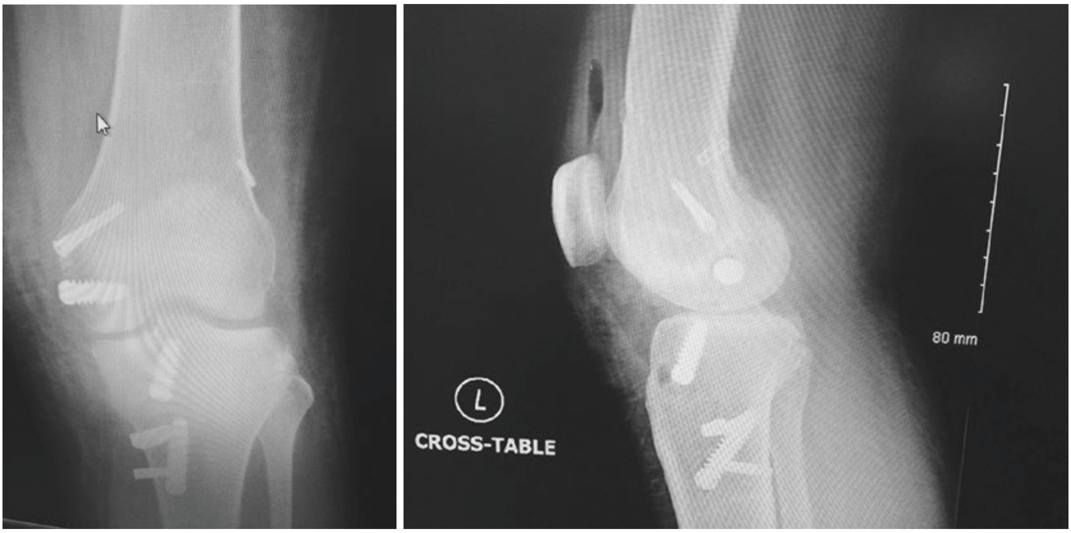


Fig. 19.7 AP and lateral radiographs showing ACL/PCL/MCL reconstructions. The relative positions of the PCL (red arrows) and MCL (yellow arrows) femoral tunnels avoid coalescence

19.2.5.1 Future Treatment

Current reconstruction techniques fail to restore normal knee kinematics and usually result in a persistent posterior sag. Future developments may well see advances in enhanced healing and regeneration of the native PCL, obviating the need for reconstruction, and improved graft selection to reduce persistent laxity. Restoration of normal knee kinematics remains an elusive goal, and the progression towards degenerative changes appears to be inexorable.

19.2.5.2 Take-Home Message

Arthroscopic PCL reconstruction has become a routine procedure for experienced knee surgeons. In the right hands, reproducible results can be achieved. However, persistent laxity remains a concern, and the restoration of normal knee kinematics is not yet achievable, in the setting of multi-ligament knee injury.

19.3 Posterolateral Corner Injuries and Management

William Hage

19.3.1 Introduction

The posterolateral corner (PLC) of the knee has an important role to play in the stability of the knee, and understanding its complexity is key to recognising injuries and reconstructing them, when necessary.

By 1918 in his seminal anatomy work, Henry Gray described the area as

"an inconstant bundle of fibers, the short fibular collateral ligament placed behind and parallel with the preceding (fibular collateral ligament)" [18].

Further work over the years including cutting studies and in vivo work has shown that to neglect this corner of the knee is to set surgical reconstruction of cruciate ligaments on an infirm footing.

19.3.2 Diagnosis

The anatomy of the PLC is now well defined to comprise three principle components: (1) the fibular (lateral) collateral ligament, (2) the popliteus tendon and (3) the popliteofibular ligament. These are complemented by various other not so discrete structures, such as the lateral capsule and its ligaments (meniscofemoral and meniscotibial), the fabellofibular ligament and the lateral coronary ligament (Fig. 19.8) [19, 20].

At a more superficial level, the PLC is thought to include the lateral head of the gastrocnemius, the biceps femoris tendon and the distal portion of the iliotibial band (ITB). When repairing an acute injury, it is often prudent to work sequentially through each of these in an attempt to reconstitute structures, starting outside with the ITB and proceeding deeper to the lateral meniscus at the depths of the posterolateral corner (Fig. 19.9) [19, 20].

Injuries of the PLC occur most commonly with a central cruciate injury, and therefore the commonest pattern of PLC injury is ACL/PLC. It has been reported [21], however, that up to 28% of PLC injuries occur in isolation. Furthermore, a large proportion of ACL/PLC injuries are simply diagnosed and reconstructed as single ACL injuries, leaving the unreconstructed PLC as a contributor to early and late graft failure and dissatisfaction with surgery.

The PLC composite acts to restrict and control varus movement at the knee and is key to stabilising posterolateral rotation of the tibia relative to the femur. This illustrates the rationale behind the commonly used examination methods. Recent efforts and consensus have tended to favour a graded system for quantifying the amount of PLC insufficiency and more importantly to consider the knee injury as a whole. Three of the more used grading systems in PLC injuries are the following [22, 23]:

Fanelli scale:

(a) Injury to the popliteofibular ligament (PFL) and popliteus tendon (PT)
(b) Injury to the PFL, PT and fibular collateral ligament (FCL)

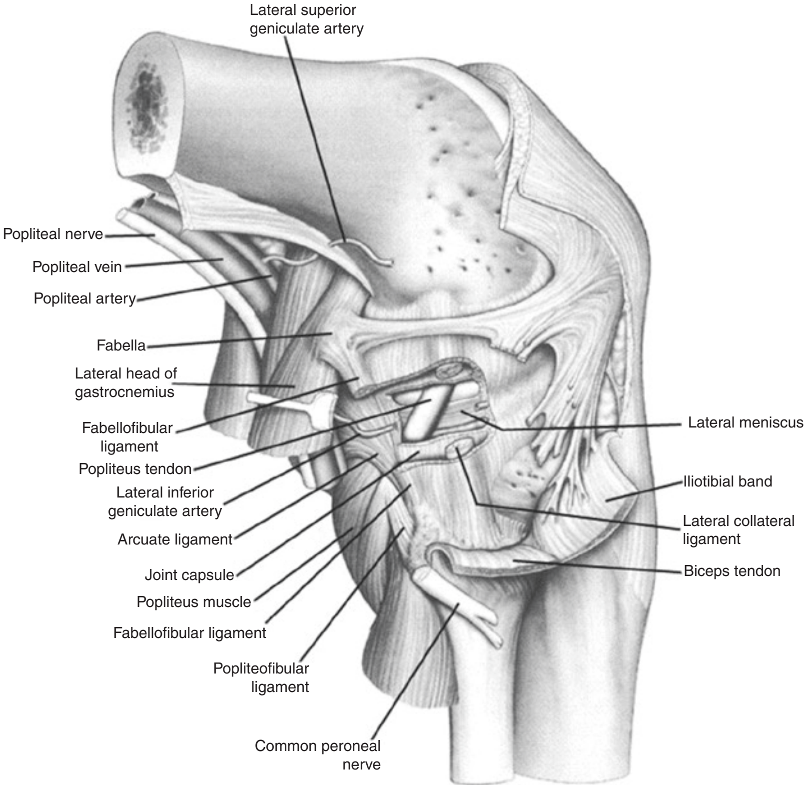


Fig. 19.8 Anatomy of the posterolateral corner

(c) Injury to the PFL, PT, FCL, lateral capsular avulsion and cruciate tear

Hughston scale (always compared to the uninjured contralateral knee):

(a) Varus opening 0–5 mm
(b) Varus opening 5–10 mm
(c) Varus opening >10 mm

With reference to the Schenck classification, these injuries would be classified as KD 1 (multi-ligament injury with involvement of ACL or PCL), but perhaps we need KD 1A, KD 1B, etc. to allow for quantities of rotatory instability attributable to the PLC, and thus a bespoke reconstruction could be planned for each case [24].

Examination of the PLC is performed in an awake cooperative patient and separately in the operating room under anaesthesia. The commonly used tests are easy to master, and most show good interobserver and intraobserver reliability. The most important principle is to consider the PLC when examining a knee injury and not simply to rest when discovering the ACL tear. “Where is the second ligament tear?” must be our mantra here. Subcutaneous bruising and pain at the lateral corner of the knee, particularly when the usually intra-articular ACL injury now tears the capsule (of the PLC), are a big clue. Gait inspection to look for a varus thrust is mandatory; to miss the need for osteotomy with ligament surgery is again a critical omission and likely to lead to failures [25].

19.3.2.1 State-of-the-Art Treatment

Acute injuries to the PLC need grading to ascertain the extent of damaged structures. If possible plain radiographs, MRI and clinical examination should be combined to quantify the amount of laxity. Severe injuries, graded III or above, need reconstruction to obtain the better outcomes [26],

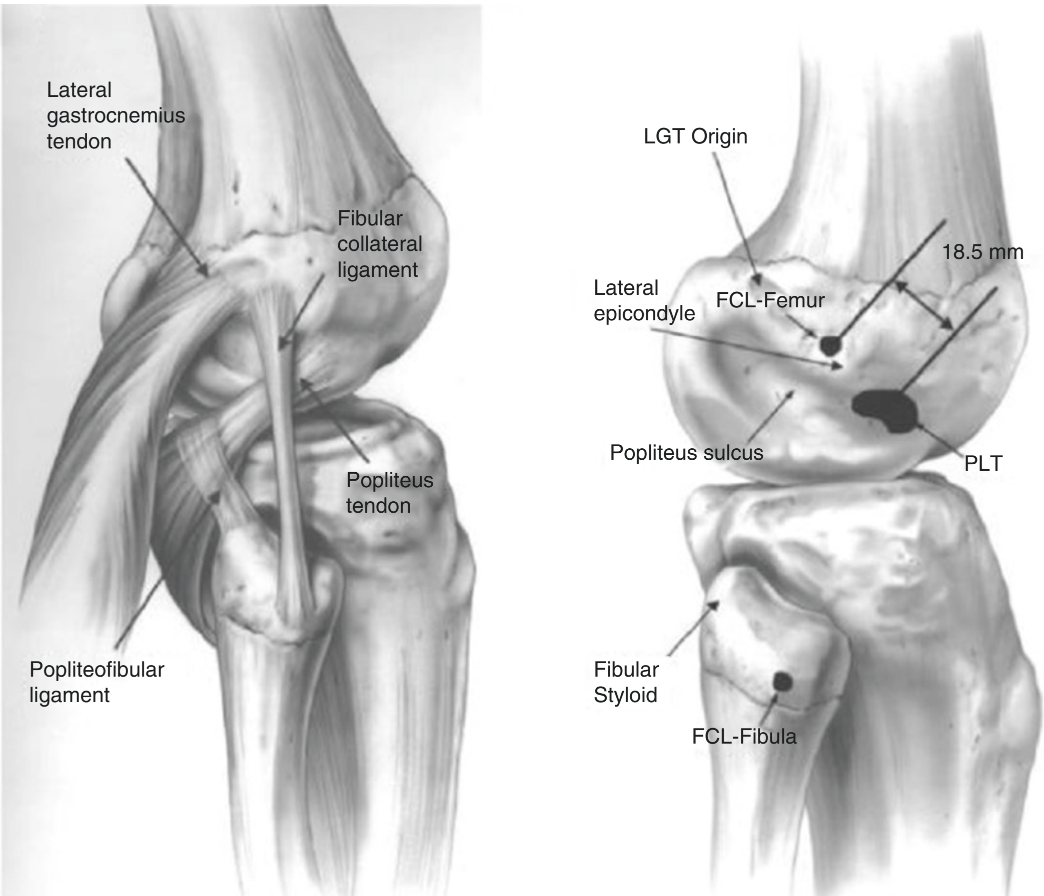


Fig. 19.9 Deeper structures of the posterolateral corner

and similarly minor sprains without division of structures, whilst rare, can often be managed non-surgically. In-between lies the area of controversy, of when to operate and when to brace, and there is little conclusive evidence on the matter.

Within the first 10 days of injury, it is often possible to explore an injury and identify torn structures of the PLC reliably before they fibrose. This then enables an accurate length reconstruction, through tissues that are receptive to surgery, with a decreasing inflammatory response and the benefits of putting native structures back together. Fractures at the proximal fibula, which often represent distal bony avulsions of the PLC, can be reliably fixed down and heal well with primary bone union. This often gives the best outcome for a PLC repair, as both length and strength are re-established quickly (Fig. 19.10).

The question of whether to augment such primary repairs of the PLC with an additional grafted surrogate PLC structure is also currently unanswered. Same sitting ACL or PCL reconstruction can be performed, after applying criteria for reconstruction of these ligaments on their own. This pays reference to such risks as overlying wounds and/or arthrofibrosis to name but a few.

Late reconstruction has been evaluated in more detail but follows a similar overview to the indications for acute reconstruction. The need to only reconstruct cruciates in the presence of minimal synovitis and a good range of movement is generally accepted, together with patient

Fig. 19.10 AP radiograph of a patient following PCL reconstruction and fixation of fibular head avulsion ("arcuate") fracture. The LCL and biceps tendon are attached to the fragment. Posterolateral stability is often well restored following ORIF

co-operation and a mature and established rehabilitation scheme. A thorough pre-op plan for reconstruction of the PLC must be made with gait analysis and an observed varus thrust corrected at the same sitting or before ligament surgery. It is accepted that an uncontrolled varus thrust, with weight bearing through a Mikulicz point in the medial compartment, will threaten any soft tissue surgery. Biplanar osteotomy to move the Mikulicz line laterally (to at least the 50% point) can be performed in series or parallel to ligament work.

The goal of late reconstruction is to reproduce the composite restraint provided by the discrete layers of the PLC, as described before. There are a small number of popular methods of reconstruction, using autograft and allograft more commonly than synthetic materials, and they are described in schematic detail here [27].

A commonly used technique, and one that has been evaluated in some detail by Mccarthy et al. [28], reconstructs the three main stabilisers at the PLC: the fibula or lateral collateral, the popliteus tendon and the popliteofibular ligament. Achilles allograft is suggested and isometric points identified with reference to known anatomical landmarks. It is described as an anatomical reconstruction (Fig. 19.11).

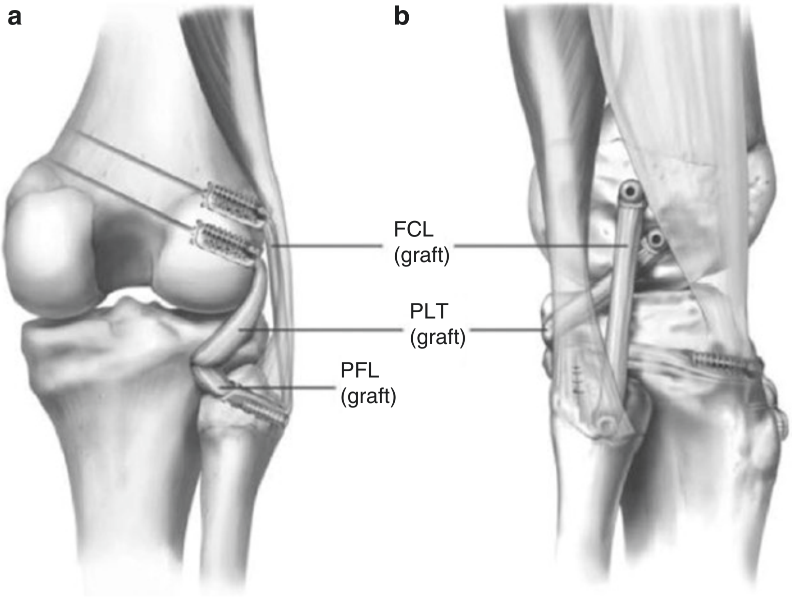


Fig. 19.11 Operative technique for reconstruction of the three main posterolateral corner stabilisers. (**a**) AP view. (**b**) Lateral view

Other techniques use a single loop of tendon to reconstruct a nonanatomical, but functional, static stabiliser with a simpler surgical approach. These are based on the Larson technique [27] (Fig. 19.12) and are modified to include two tendon origins on the femoral condyle (popliteus tendon and fibula collateral) and a tunnel through the fibula head serving as a surrogate attachment point. Zantop and Niki [29] have separately described these, and no firm data exists to prove or reproduce their superiority in practice. Their relative lower complexity in surgical technique may or may not allow a more reproducible outcome. Sometimes simpler is better, but what is needed clearly is a large RCT.

19.3.2.2 Future Treatment Options

Not all PLC injuries require surgery, and not all PLC injuries are diagnosed early enough to allow consideration of their relevance. Multiple methods for reconstruction exist without conclusive evidence of their superiority. Graft choice is unanswered, for what is principally an extra-articular repair. Early repair on its own, versus repair plus parallel augmentation, is also a question to be tested.

Fig. 19.12 Post-operative AP radiograph following a Larson-type posterolateral corner reconstruction using a single femoral tunnel. Whilst it is relatively easier to perform, the reconstruction is nonanatomical

Thus, future treatment options are pinned to a definition of a gold standard in posterolateral corner injuries, namely:

1. Identify all PLC injuries early enough to allow intervention.
2. Reconstruct as anatomically as necessary, to reproduce the restraints.
3. Limit incision size and risks of complications with elegant and timely surgical methods.
4. Use grafts with the least morbidity, including primary repair if possible when these are proven to function.
5. Always suspect the role of undetected PLC injuries in revision ACL/PCL, etc. cases.

19.3.2.3 Take-Home Message

The posterolateral corner has been the subject of much scientific analysis and surgical attention in the last 15 years. These efforts have built upon the work of the previous 30 years where its relevance was suspected in contributing to ligamentous instability of the knee. Given its likely underdiagnosis, the message is thus to always consider the PLC in a ligamentous injured knee and look for the disruption. To miss the peripheral PLC injury is to build your central cruciate reconstruction on shaky grounds.

19.4 MCL Injuries and Management

James Robinson

19.4.1 Introduction

The medial collateral ligament complex (MCL)/posteromedial corner (Fig. 19.13) is the most commonly injured ligamentous structure in the knee

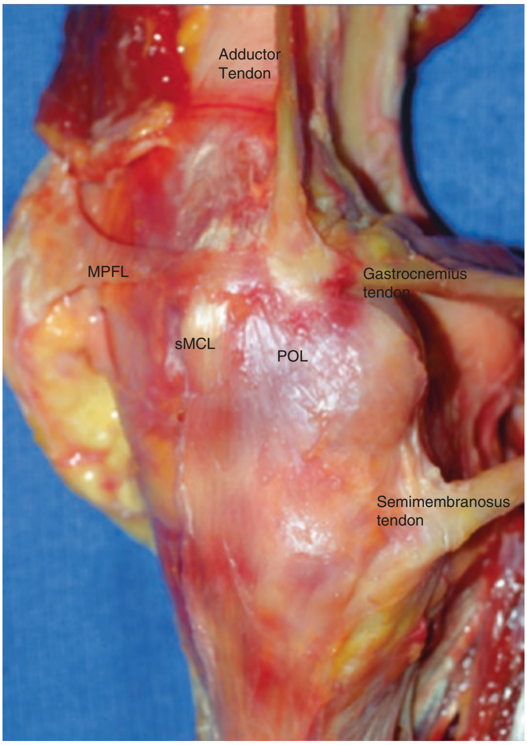


Fig. 19.13 Posteromedial corner (right knee). The patella is seen anteriorly. The meniscofemoral ligament (MPFL) is seen attaching in a saddle area between the adductor tubercle and the medial epicondyle, passing anteriorly and attaching to the medial side of the patella. Three tendons, those of the adductor magnus, the semimembranosus and the medial gastrocnemius are important landmarks during medial reconstruction

[30]. There is a wide consensus that the majority of grade I and II MCL injuries heal with rehabilitation alone [31–33]. Although grade III injuries may also heal without the need for surgery, it is recognised that a small percentage remain symptomatic following conservative treatment and may require operative intervention [30]. Similarly, some acute injuries may warrant surgical repair or reconstruction.

19.4.1.1 Functional Anatomy and Biomechanics of the Medial Side of the Knee

Three principal structural elements of the MCL have been described: the superficial MCL (sMCL), the deep MCL (dMCL) and the posterior oblique ligament (POL) [34, 35].

The sMCL is the most prominent ligamentous structure of the medial aspect of the knee and is a part of layer 2 (as described by Warren and Marshall [34]), lying deep to the sartorial facia (layer 1). The proximal attachment of the sMCL envelops the medial epicondyle [36]. The femoral attachment of the sMCL envelops the prominence of the medial femoral epicondyle running into the saddle area proximal/posterior to the epicondyle (anterior/distal) to the adductor tubercle. The deepest fibres run tangential to the bone surface, and they insert into the distal-facing slope of the epicondyle, whilst the more superficial fibres pass over and cover it and insert proximal posterior [37]. The distal attachment of the sMCL is to the anteromedial aspect of the tibia, typically 6 cm below the joint line. Approximately 2 cm long, it extends distally and slightly posteriorly in a linear attachment approximately 3 mm wide (Fig. 19.14). The sMCL remains taut with flexion/extension of the knee and is the primary knee against abduction moments at all angles of knee flexion [38].

The dMCL is a capsular ligament and is attached firmly to the medial rim of the medial meniscus with meniscotibial and meniscofemoral fibres, lying deep to the sMCL (layer 3) (Fig. 19.15) [36]. Injury to the meniscotibial fibres causes the meniscus to lift away from the

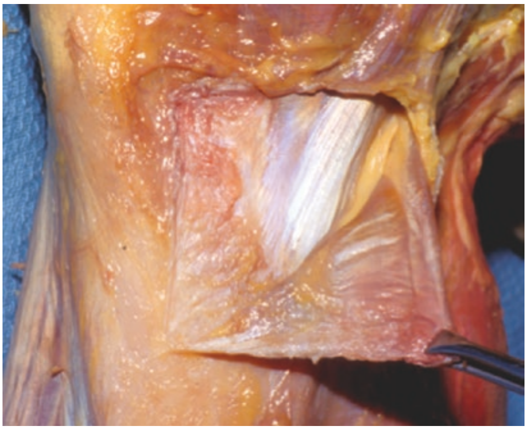


Fig. 19.14 The distal sMCL attachment (layer 2) to the tibia may be visualised under layer 1 (sartorial fascia). The tendons of the pes anserinus attach distally and anteriorly, with the tendons tying on the deep surface of the layer 1 fascia

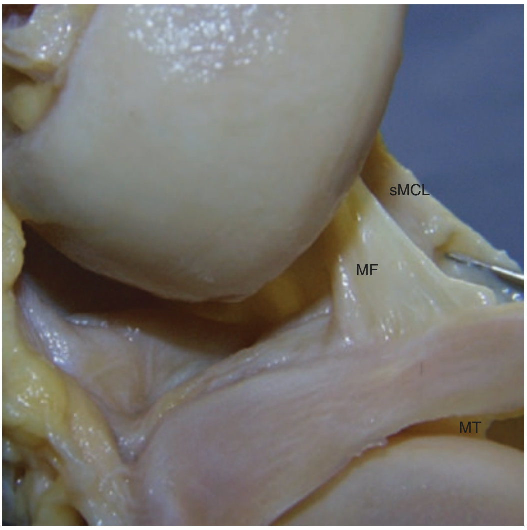


Fig. 19.15 The medial meniscus and the deep MCL viewed from within the joint—the sMCL is retracted. The deep MCL is a condensation of fibres in the capsule and has meniscotibial (MT) and meniscofemoral (MF) fibres

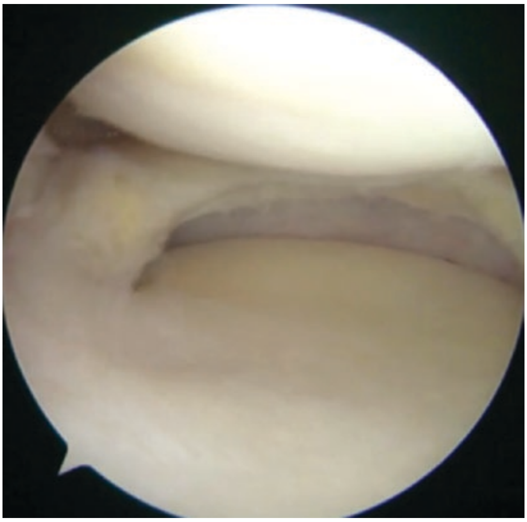


Fig. 19.16 MCL injury towards the femoral attachment causes the femur to lift away from the meniscus. Conversely, tibial-sided injury, with injury to the meniscotibial fibres of the dMCL, causes the meniscus to lift away from the tibia with the femur when valgus stress is applied to the knee

tibial plateau when the knee is subjected to an abduction moment. This can be visualised arthroscopically (Fig. 19.16). The femoral attachment is immediately distal to the epicondylar attachment of the sMCL, whilst the tibial attachment is to the medial rim of the tibial plateau and thus close to the joint line and proximal to the attachment of the anterior arm of the semimembranosus expansion. The posterior edge of the dMCL is marked by its blending with the posterior edge of the sMCL. Thus, at this boundary, layers 2 and 3 blend together to become a single capsular layer 3. The dMCL is tightened rapidly by tibiofemoral relative motion because its fibres are shorter than those of the other ligaments; it has a role in limiting tibial external rotation and anterior drawer in external rotation [38].

The femoral attachment is distal to the epicondylar attachment of the sMCL. The tibial attachment is 12 mm below the joint line just proximal to the attachment of the anterior arm of the semimembranosus expansion. At the posterior edge of the sMCL, layers 2 and 3 blend to form the POL.

The POL is a condensation of fibres within the posteromedial capsule of the knee and has been shown to be an important restraint to internal tibial rotation, posterior tibial translation and valgus rotation in the extended knee (Fig. 19.17) [38–42]. Several studies have postulated that it is injury to the POL that may be responsible for residual instability following conservative treatment of MCL injury [23, 32, 43, 44], in particular after combined grade III MCL and cruciate ligament injury [45].

Studies examining the strength of the three principle structures [46, 47] have shown that the sMCL is strongest with a yield load of >500 N at ultimate failure. Both the sMCL and POL are stronger than the dMCL. The sMCL has been shown to have higher tensile stiffness than the other two structures implying that it will take more of the load when an abduction (valgus) moment is imposed on the knee. The dMCL was shown to fail at significantly lower elongation than the other structures. The lower elongation to failure of the dMCL explains the clinical finding of dMCL rupture, whilst the knee remains stable against abduction (valgus) loading, when the sMCL has not ruptured. The earlier failure of the dMCL relates to its shorter fibres, so they are subjected to a higher percept strain elongation,

Fig. 19.17 The POL is tensed by full extension of the knee: the bulge of the posterior femoral condyle and the rim of the tibial plateau are seen. The posterior-distal orientation of the capsular fibres is shown. These fibres are an import restraint to valgus rotation, internal rotation and posterior translation in the extended knee

for a given tibiofemoral angulation, than the sMCL and posteromedial corner fibres. Isolated dMCL injury can be troublesome particularly in soccer players [48]. Kicking the ball with the side of the foot (tibial external rotation with coupled valgus, in a flexed knee) loads the dMCL and may cause pain. Jones et al. [48] reported that most patients respond well to "dry needling" (90%). If, after 12 weeks, pain persisted, it was thought reasonable to proceed to arthroscopic debridement of associated granulation tissue or operative repair. Narvarni et al. [49] suggested earlier repair may be offered in elite sportsmen in whom the dMCL may be refractory to conservative treatment and present with persistent symptoms after minor MCL injury.

The predominant site of ligamentous failure in posteromedial corner injury is on the femoral side [46, 47]. This distinction is functionally important, because rupture of the more distal part, including the dMCL, is associated with pathological mobility of the meniscus and is identifiable by the lifting of the meniscus away from the tibial plateau when the knee is subjected to a valgus load and the interior is viewed arthroscopically. The more common scenario is for femoral-sided injury resulting in the femur lifting away from the meniscus.

19.4.1.2 Clinical Evaluation

Classically, the MCL is injured where there is a combination of abduction, flexion and external rotation of the femur on the tibia [50]. The patient may experience a tearing sensation or hear a "pop", suggesting a high-grade injury [51]. Commonly, the MCL is injured by a direct blow to the lateral aspect of the knee, whilst the foot is in contact with the ground [52]. The MCL is however frequently not damaged in isolation, and injury is often associated with cruciate ligament damage, especially if a direct lateral blow to the slightly flexed knee is combined with rotation [53]. In a review of 265 patients, Fetto and Marshall [31] found that 78% of those with the most severe (grade III) MCL injuries had damage to other ligamentous structures, of which 95% were ACL injuries. It has also been noted in patients following ACL reconstruction that there is deterioration in function in those who have residual MCL laxity [54]. Indeed, one of the causes of an unsuccessful outcome following modern isolated cruciate ligament reconstruction may be because of an undiagnosed and untreated concomitant laxity of the posteromedial structures of the knee.

Rupture of the MCL may be detected clinically by palpation of the ligament along its course [55]. An hour after injury, tenderness and a spongy swelling [52] localise the lesion. Commonly, this is on the femoral side, approximately 2.5 cm above the joint line. It may take 30 minutes or more for the ligament to become tender with milder injuries—an athlete may have a transient inability to walk but then may be able to return to sporting activity for a few minutes. A knee joint effusion may be present; however, if there is significant capsular disruption, this may not be present. The site of ecchymosis and swelling may indicate the site of the lesion (Fig. 19.18) [51].

Fig. 19.18 Medial bruising following tibia avulsion of the sMCL

In general, valgus stress testing at between 20° and 30° of knee flexion is used as a primary test of MCL integrity (the test is not performed in greater flexion because rotation of the femur is difficult to control). Because there is variation amongst individuals, it is important to assess the contralateral, uninjured knee for comparison. Several grading systems for valgus laxity exist. The IKDC grading measures increased opening of the medial compartment: grade A (normal) 0–2 mm, grade B (nearly normal) 3–5 mm, grade C (abnormal) 6–10 mm and grade D (severely abnormal) > 10 mm. It may be difficult to determine this scoring system without the use of stress radiographs. O'Donoghue [56] classified MCL injuries as grade I, ligament sprained but intact; grade II, partial tearing with mild laxity; and grade III, complete tear with valgus laxity. Perhaps the most useful clinical classification is that used by Fetto and Marshall [31], grade I, no laxity; grade II, laxity at 30°; and grade III, laxity at 0° and 30° flexion. Valgus stress testing should be repeated with the knee in extension. Stability in full extension has been said to indicate that the posteromedial capsule/POL has no significant damage [53, 57]. Abnormal valgus laxity indicates MCL injury with concomitant posteromedial capsular/POL injury.

Slocum and Larson [58] characterised anteromedial rotatory instability as a positive anterior drawer sign that was accentuated when the test was repeated in 30° of external tibial rotation and reduced with the tibia in 15° of internal rotation. Similarly, Franklin et al. [59] described anterior translation of the tibia, with draw testing at 90° knee flexion, as being greater in tibial external rotation rather than in neutral or internal rotation when there was a combined injury to the posteromedial corner and ACL. Hughston also reported that resultant functional instability was due to "the abnormal excess opening of the medial joint space in 30° of knee flexion with simultaneous anteromedial rotatory subluxation of the medial tibial condyle on the central axis of the intact PCL" [23]. Engebretsen and Lind [60] described forward displacement of the medial tibial condyle, when a valgus stress was applied to the externally rotated knee. Increased tibial external rotation with the dial test must be carefully evaluated, and care must be taken to differentiate between posterolateral and anteromedial rotatory instabilities. Performing the test with the patient supine allows differential movement of the proximal tibia to be noted.

Physical examination should also include assessment of alignment. If valgus lower limb alignment is suspected, then long-leg (hip-to-ankle) standing radiographs should be taken. In cases of chronic MCL laxity with valgus, lower limb alignment, medial distal femoral closing wedge or medial tibial closing wedge osteotomy (Fig. 19.19) should be considered. Realignment alone may be enough to overcome the patient's feelings of instability [61].

19.4.1.3 Imaging of Medial-Sided Injury

The three layers on the medial side of the knee can be visualised on MRI (Fig. 19.20), and this imaging modality is regularly used to elucidate ligamentous knee injuries in the acute setting. Routine knee MRI sequences have been demonstrated to accurately detail all three components of the MCL complex [62]. The POL can be well identified on the axial and coronal images in a 1.5 T MRI scanner [63]. Radiologically, grade III MCL injury is represented by complete ligamentous discontinuity with laxity or waviness, suggesting disruption of all three components of the MCL [64]. Bone

Fig. 19.19 Interpretation of the dial test. Increased external rotation of the tibia at 30° knee flexion may be due to (**a**) anteromedial instability or (**b**) posterolateral corner laxity. Performing the test supine so that the rotation of the tibia can be observed. In the right knee, anteromedial subluxation of the tibia is seen

bruising in the lateral tibiofemoral compartment is often seen with acute MCL injury [65]. It is important to determine injury patterns as injury at the femoral attachment tends to heal well conservatively, mid-substance ruptures less so [66] and tibial-side injuries may require repair [67].

Stress radiography is important in the evaluation of posteromedial corner injury. It allows objective assessment of instability, validates the laxity pattern and may be used to assess post-operative outcomes. LaPrade et al. [68] demonstrated, in a cadaveric study, that medial gaping of >1.7 mm at 0° and >3.2 mm at 20° indicated grade III sMCL injury. For ruptures of all the passive medial restraining structures, medial gapping was >6.5 mm at 0° and >9.8 mm at 20° knee flexion.

Others have sought to radiologically determine combined cruciate and medial injury with differential movement of medial and lateral tibial condyles with an applied anteroposterior force. Stäubli and Jakob [69] studied 24 patients with PCL injuries using stress radiography. A posterior force was applied to the tibia with the knee near extension (10–15°), and lateral radiographs of the knee were taken. Combined posterior translation and tibial internal rotation (8 mm posterior translation of the lateral tibial plateau and 13 mm posterior translation of the medial tibial plateau) were said to represent PCL rupture with damage to the posteromedial corner. Lerat et al.

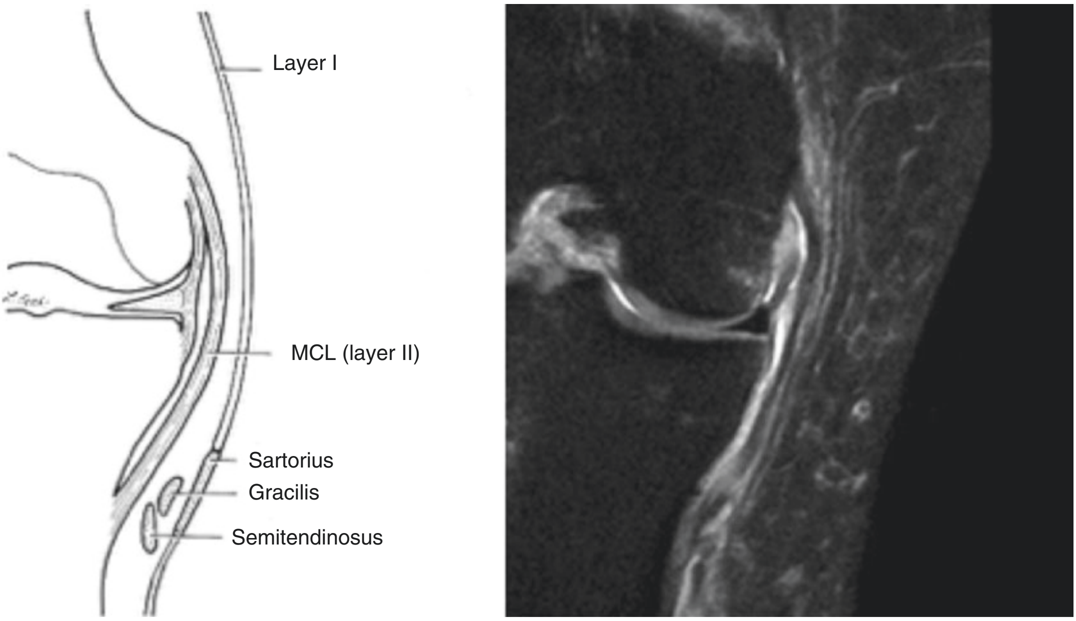


Fig. 19.20 The three layers of supporting structures on the medial side of the knee can be visualised on MRI

[70] suggested that anterior translation of the medial tibial condyle of >8 mm was suggestive of combined ACL and posteromedial laxity.

19.4.1.4 State-of-the-Art Treatment

Treatment of MCL Injuries

MCL injury (grades I, II and III) can be treated nonoperatively [31, 52, 71, 72]. There is a wide consensus that the majority of grade I and II MCL injuries heal with conservative rehabilitation alone [30–33]. Immediate RICE (rest, ice, compression, elevation) and use of crutches as required are followed by early range of motion in a hinged knee brace to protect the knee from valgus stress. Some authors recommend locking the brace in extension [67] or in slight flexion [73] for 1–2 weeks for high-grade injury. It is important to review the patient as prolonged immobilisation may lead to stiffness. Return to sport following grade I and II sprains is often achieved after a few weeks post-injury [74] (Holden reported an average of 3 [75]) with 74% of patients returning to normal function by 3 months post-injury [76]. Outcomes are excellent with Lundberg and Messner [73] reporting median Lysholm scores of 100 and 95 at 4 and 10 years, respectively. Isolated grade III injuries may also be similarly treated [30, 53, 71], but return to sport takes longer. There is some animal evidence that PRP may enhance healing [77, 78]. However, there is little evidence in humans. Evidence from animal models suggests that the use of NSAIDs is not deleterious [79], but smoking may impair MCL healing [80].

Some patients with grade III injuries do not respond well to nonoperative treatment [33], and it has been suggested that this may be due to the presence of an injury to the POL [55]. The POL has been demonstrated to be an important secondary stabiliser for rotation and valgus stress after isolated MCL injuries [41]. Combined sMCL and POL injuries may be associated with an increased incidence of ongoing MCL instability after grade III injury and have emphasised its reconstruction in order to restore stability [32, 43, 44].

Combined ACL and MCL Injuries

Delayed reconstruction of the ACL, following bracing and healing of the MCL injury, has been recommended by many [52, 81–83].

Petersen and Laprell (1999) found that delaying ACL reconstruction until >10 weeks post-conservative treatment of the MCL injury in a hinged knee brace was associated with improved Lysholm scores and lower rates of range of motion loss compared with patients where the ACL was reconstructed within 3 weeks of injury [83]. Others have also recommended the use of a collateral ligament brace for several weeks until a normal range of movement returns before reconstructing the ACL [52, 67]. Noyes and Barber-Westin [84] based their treatment of the combined MCL and ACL injury on the severity of medial ligamentous injury: in patients with complete rupture of both the ACL, sMCL and POL, reconstruction of the ACL and a medial side repair ± augmentation of the oblique fibres and capsule were performed. In patients with complete ruptures of the ACL and sMCL, the ACL was reconstructed, but the medial injury was treated conservatively. Hallinen et al. [85] demonstrated that nonoperative management of the MCL injury and surgical repair achieved similar results but that patients undergoing MCL repair took longer to recover strength and ROM. The tendency for patients to be stiff and potentially struggle to regain ROM leads to combined ACL/MCL surgery for acute injury becoming less popular. In a systematic review, Grant et al. [67] concluded that optimal results for patients with combined ACL/MCL injury were obtained after conservative treatment allowing healing of the MCL and the patient regaining full range of motion followed by delayed ACL. Following conservative treatment, studies have reported good results for isolated ACL reconstruction even in patients with ACL rupture and residual grade II MCL injury [86]. Patients who have residual, symptomatic MCL laxity following conservative treatment should undergo combined ACL/MCL. MCL reconstruction is indicated if there is symptomatic valgus instability or when stress radiography indicates grade III MCL laxity [67]. Radiological examination of medial laxity can be assessed intra-operatively (Fig. 19.21) with the use of an image intensifier.

Fig. 19.21 Stress radiographs allow objective assessment of instability, validation of instability patterns and assessment of post-op outcomes

Combined MCL and PCL Injuries

Combined MCL and PCL injuries account for 0.4–1% [87] of knee ligament injuries. Grade I and II MCL injuries with concomitant PCL rupture may be treated conservatively [88]. However, it has been recognised that combined PCL and grade III MCL injury may lead to increased internal tibial rotation laxity [89] and increased posterior translation that occurs when the POL is ruptured [90] as the POL is important in restraining posterior tibial translation in the extended knee [36]. Conservative treatment of isolated PCL injuries with braces that apply a dynamic anteriorly directed force has gained popularity. The best management for combined MCL/PCL injuries remains controversial, and there is no consensus. It is the authors' preference to manage combined injuries with conservative treatment in a PCL brace and then to assess the knee by stress radiographs. In our experience, a third of patients with grade III PCL/MCL tears managed conservatively go onto combined PCL/MCL reconstruction; thus early reconstruction of both ligaments, as has been advocated by others, may be reasonable.

Indications for Acute MCL Repair

1. Femoral attachment bony avulsion.
2. Intra-articular entrapment of the MCL. Operative treatment is mandatory if there is

intra-articular entrapment of the sMCL. This may occur following rupture of the sMCL at its distal tibial attachment [91, 92] and very rarely the femoral following femoral-sided injury. The avulsed portion or the ligament is retrieved from the joint and reattached to its attachment site with a suture anchor.

3. MCL "Stenner" lesion. The distal sMCL is replaced and reattached so that the pes anserinus tendons once again lie between layers 1 and 2.

Relative Indications for MCL Repair

1. Multi-ligament injury. In cases of ACL/PCL/MCL injury where reconstruction of the cruciate ligaments is undertaken acutely, the MCL should be repaired or reconstructed. Primary medial reconstruction should be performed if there is concern about the adequacy of surgical repair, as repair rates have been shown to have higher failure rates than primary reconstructions [93]. Avulsion injuries may be repaired by reattachment of the avulsed fragment. For midsubstance injuries, augmentation procedures are often required, commonly using semitendinosus or gracilis autograft. Some surgeons have advocated staged repair, reconstructing the PCL and MCL first and then the ACL [94].
2. Combined ACL and tibial MCL avulsions in athletes. This pattern of injury is reported to have a worse prognosis regarding the development of chronic medial laxity.

Some authors have suggested that MCL repair may be reinforced using braided ultrahigh-molecular-weight polyethylene/polyester suture tape and bone anchors [95]. Similar techniques have been used for tendon repair (rotator cuff and Achilles tendon). However, there are currently no published studies recommending indications or reporting results compared to conservative treatment, primary repair alone or reconstruction.

19.4.2 Posteromedial Corner Reconstruction

Reconstruction is indicated for chronic symptomatic medial instability following conservative treatment or for augmentation of acute primary repair where tissue quality is dubious. Many different medial reconstructions have been described, whereby just the sMCL or both sMCL and POL are reconstructed. These may involve re-routing the semitendinosus or use free autograft or allograft described diverting the semitendinosus around the medial epicondyle.

19.4.2.1 Tenodesis Procedures

The Bosworth procedure [96], described in 1952, involves detaching the semitendinosus proximally, leaving it attached distally, looping it around a screw placed in the medial epicondyle and stapling it to the tibia distally to reconstruct the sMCL. Kim et al. [97] described a modification in which the semitendinosus was looped around a screw in the epicondyle and the free end sutured to the semimembranosus, thereby producing a POL arm to the reconstruction (Fig. 19.22). Medial opening was reduced from 7.8 mm to <2 mm, and the mean post-op Lysholm score was 91.9 at 1 year post-op. Lind et al. [98] described a modification whereby the semitendinosus was left attached to the tibia and led proximally into a socket in the epicondyle (to reconstruct the sMCL) and the free end was led back to the posteromedial tibia (to reconstruct the POL) (Fig. 19.23). The results, using the technique for chronic medial instability, were reported

Fig. 19.22 Bosworth procedure in which the semitendinosus is left attached distally and is looped around a screw in the medial epicondyle. It must be noted that the distal attachment of the sMCL is posterior to the insertion of the semitendinosus, and thus the reconstruction is nonanatomical

Fig. 19.23 Reconstruction proposed by Lind et al. [98]

in 50 patients at 2 years follow-up. Ninety-one per cent were satisfied and with IKDC A or B in 98%. The technique has since been modified, as it has been accepted that the attachment of the semitendinosus is anterior to the tibial attachment of the sMCL. The semitendinosus tendon is first diverted posteriorly to the tibial attachment of the sMCL and fixed here with a suture anchor before being led proximally to the medial epicondyle. In addition, a further suture anchor is used 12 mm below the joint line to replicate the action of the dMCL.

19.4.3 Free Allograft/Autograft Procedures

Yoshiva et al. [99] reported the results of sMCL reconstruction (± POL imbrication) with autogenous hamstrings using a screw and washer in the femur and a tibial tunnel. All reconstructions were performed in patients with concomitant ACL or PCL reconstruction. At 27 months post-operatively, 88% of patients had IKDC A or B. Zhang et al. [100] reported 95% patients with normal/nearly normal knee stability following reconstruction of the sMCL with Achilles tendon allograft. The bone block was used on the tibial side, and the free end led to a femoral tunnel. Conversely, Marx and Hetsroni [101] reported the results of 14 patients undergoing combined ACL and MCL reconstruction with Achilles tendon allograft in which the bone block was used on the femoral side. All patients were reported to have achieved normal or near-normal MCL stability. Borden et al. [102] described the use of tibialis anterior allograft with a single femoral tunnel and two tibial tunnels. It was recognised that this was a nonanatomical reconstruction. Stannard [81] also described the use of free graft to reconstruct the sMCL and POL. A single tunnel was drilled at the femoral epicondyle, and the graft was taken distally to reconstruct the sMCL. The graft was looped around a screw placed 6 cm below the joint line and then passed proximally, under the semimembranosus, and back to the femoral socket to reconstruct the POL. Coobs et al. [40] described an anatomical medial reconstruction using semitendinosus autograft using separate femoral and tibial tunnels for the sMCL and POL grafts. Importantly, a suture anchor is added 12 mm below the joint line, and the sMCL graft is sutured in the position, replicating the function of the proximal tibial sMCL attachment/dMCL. LaPrade [103] reported on the results of this reconstruction in 28 patients at a mean 1.2 years follow-up. All had less than 2 mm medial opening on stress radiographs; the IKDC clinical score had improved from pre-operative score of 43.5 to a post-operative score of 76.2. It is important to note that the POL graft should be fixed with the knee in extension, as it is an anisometric structure that tightens in extension [38, 39]. Fixation of the POL graft in flexion captures the knee preventing full extension.

Comparing different MCL reconstruction techniques, Feeley et al. [104] tested, in vitro, the efficacy of restraint to valgus stress and tibial rotation at 0 and 30° of knee flexion. Results were favourable for reconstructions with two limbs—either modified tenodesis or anatomical reconstruction. In choosing which medial reconstruction to perform, the surgeon must consider whether there is sMCL or combined sMCL/POL laxity. Furthermore, POL laxity may be implicated in the development of chronic instability, and if the rotational instability of a torn posteromedial corner is not addressed, it frequently leads to a failure of the (MCL) reconstruction and may also lead to failure of associated ACL or PCL reconstructions [81]. The reconstruction proposed by LaPrade and colleagues (Fig. 19.24) has the best combination of an anatomical basis, biomechanical evaluation and published clinical outcomes and is the author's preferred method of MCL reconstruction. However, a balance needs to be struck between what is ideal and what is achievable, and particularly in the setting of multi-ligament reconstruction, a simpler reconstruction may be adequate. Similarly, in small knees, it may be challenging to separate femoral sMCL and POL tunnels, and in this situation, the author favours a single femoral tunnel.

19.4.3.1 Take-Home Message

The MCL complex is the most commonly injured ligamentous structure in the knee. A systematic physical examination as well as sophisticated imaging may help to characterise the injury profile. Most medial knee injuries can be treated conservatively with good results. However, when acute grade III MCL injury occurs, particularly in combination with other ligament damages, conservative treatment may not be the most suitable option. In these circumstances, anatomic surgical repair or reconstruction should be considered. Chronic valgus instability can be very disabling and so requires anatomical reconstruction of the deficient ligamentous structures.

19.5 Graft Selection in Multi-ligament Knee Injuries

Manuel Leyes

19.5.1 Introduction

The type of ligament graft selected may influence the outcome of patients with multi-ligament

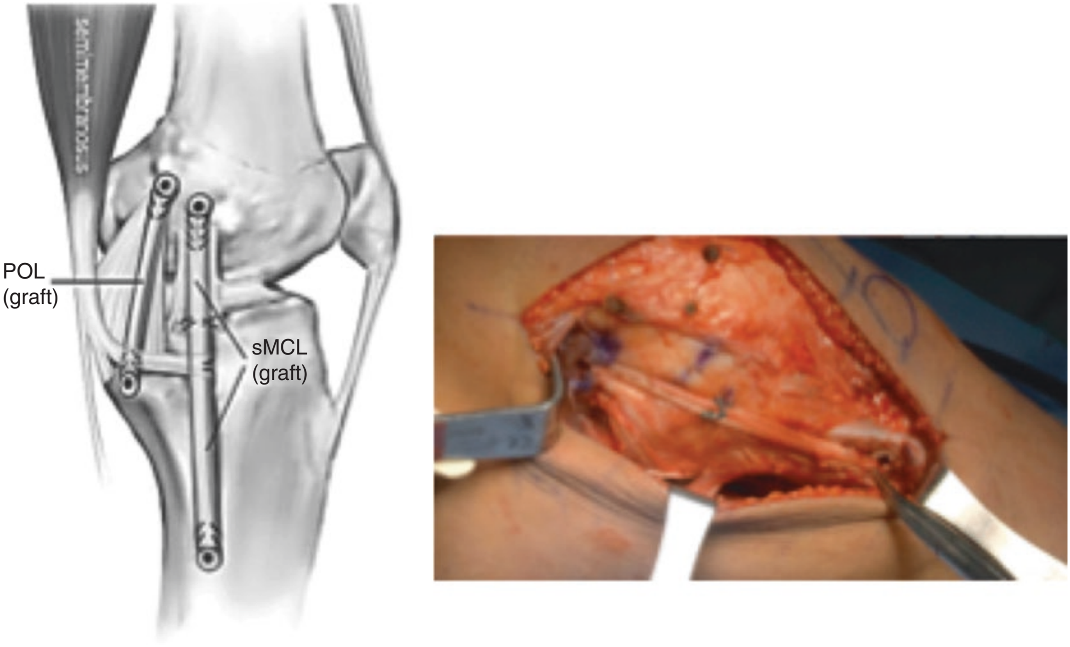


Fig. 19.24 (*Left*) Reconstruction proposed by LaPrade and co-workers (reproduced from Laprade et al., CORR 2012) reconstructs both sMCL and POL. A suture anchor placed 12 mm below the joint line (*right*) replicates the function of the dMCL in restraining tibial external rotation

knee injuries. The surgeon should be familiar with the different options and decide whether to create an additional injury to the ipsilateral knee to harvest the grafts or to use other options such as contralateral graft harvesting or allograft [105].

Graft choice depends on the surgeon's experience and preference, patient preference, tissue availability, the patient's desired activity level, profession, comorbidities, prior autograft tissue harvesting, prior tunnel placement, tunnel osteolysis, prior skin incisions, the number of ligaments requiring reconstruction or augmentation and the extent of each injury [106, 107].

The autograft options available include the patellar (PT) [106], hamstring (HT) and quadriceps tendons (QT) of either the ipsilateral or contralateral knee, whilst allograft choices are the Achilles, quadriceps, patellar, hamstring and anterior and posterior tibialis tendons as well as the fascia lata [96, 108].

No current indications exist for synthetic ligaments.

19.5.2 State-of-the-Art Treatment

19.5.2.1 Autograft

Autograft tissues do not require sterilisation and have no risk of infectious disease transmission and no risk of immune-mediated tissue rejection [108–110].

In the setting of the multi-ligament-injured knees, multiple grafts are usually needed, but the number of available autografts is limited and autograft tissue harvesting further damages the already injured knee [109]. Double ligament autograft reconstruction is usually not problematic. When more than two ligaments require reconstruction, graft harvesting from the contralateral side has been advocated, but this results in morbidity to the normal knee and patients often refuse this option. When two grafts are required, possible combinations include the PT and QT, the PT and HT or the QT and HT. Some authors have demonstrated excellent results in acute knee dislocations reconstructed in a staged fashion using contralateral hamstring autograft followed by ipsilateral hamstring and PT autografts 3 months after the index surgery.

The incorporation process of both auto- and allografts includes graft necrosis, cellular repopulation, revascularisation and collagen remodelling although it is at a slower rate in allografts.

PT grafts have the advantage of bone-to-bone healing, which is stronger and faster (6 weeks) than soft tissue healing (8–12 weeks) [106]. Besides the advantage of having bony fixation at both ends, the PT graft is strong and stiff. However, PT autografts cause higher graft site morbidity, primarily anterior knee pain and less cosmesis. The PT can be used to reconstruct the ACL, PCL or lateral collateral ligament.

HT grafts are better cosmetically, and harvesting results in less donor site morbidity but its incorporation is slower. They are smaller in size and the initial tibial fixation may be a problem. It is unclear whether HTs should be harvested in patients with associated medial knee instability [107].

QT provides a large tendinous graft with a bone plug on one end of the graft. A 10-mm-wide QT graft has a larger cross-sectional area than a similar-sized PT graft. QTs have intermediate morbidity and decrease the operative time but have the worst cosmesis and may have soft tissue fixation problems on the soft tissue end with slower incorporation. QT has been used to reconstruct the ACL and PCL and provides superior length, bulk and strength compared to the HTs [108–110].

19.5.2.2 Allografts

Allografts are currently becoming more popular due to improved sterilisation techniques and easier availability [108, 110]. In the past, the use of ethylene oxide sterilisation resulted in chronic effusions, whilst high-dose radiation used for sterilisation worsened the structural properties of allografts. Cryopreservation and gamma irradiation with less than 3.0 Mrad, the current sterilisation techniques, have no effect on the structural properties of ligaments and tendons. In some countries of Europe, no secondary sterilisation procedure is required as the graft sources are volunteer multi-organ donors that have been screened to rule out any infectious disease.

Additionally, the use of allografts shortens operative time as they can be prepared simultaneously before or during the surgical procedure.

The risk of viral or bacterial infectious disease transmission from musculoskeletal allograft implantation is extremely low. Freeze-drying and radiation decrease but may not eliminate the already low risk. Gamma irradiation to a level of greater than 3.5 Mrad is estimated to be required to eliminate HIV, but such a dose damages the graft's biomechanical properties. Nevertheless, there are no documented cases of HIV or HCV transmissions in the setting of appropriately screened donors and nucleic acid testing.

Over the last decade, the risk of bacterial infection from musculoskeletal allograft tissue, including clostridial infections, has been increasingly compared to the risk of viral transmission [110].

Autograft tissue incorporates faster than allograft tissues. It seems that a subtle immune response may occur after allograft implantation. This response may affect graft revascularisation and graft incorporation. It may take up to one and a half times longer for the allograft to completely remodel and gain comparable strength to an autograft. Despite the slower rate of incorporation, the eventual healing is almost identical to the healing of an autograft, and there is little evidence to suggest that this immune response plays a significant role in the clinical outcome of allografts [110].

It is difficult to compare studies on the graft's biomechanical properties because the results can vary markedly depending on the age of the donor, the size of the graft and the methods of testing. Most surgeons choose a graft with biomechanical properties superior to the native ligament that is being reconstructed. The fact that soft tissue autografts are known to undergo necrosis after implantation and may lose part of their intrinsic strength should be considered. Therefore, due to the current preference to use a large graft for PCL reconstruction, QT, double-stranded tibialis and Achilles tendon are usually chosen.

The author prefers to use a long tibialis anterior allograft for simultaneous central pivot and medial or lateral collateral ligament reconstruction. However, an Achilles tendon allograft is preferred in the case of a PCL reconstruction. A hybrid auto- and allograft reconstruction is commonly used in revision ACL surgeries and multi-ligament injuries.

Donor age has been proposed as a factor in the biomechanical strength of available allograft tissues. However, it has not been proved in donors up to age 55.

19.5.2.3 Future Treatment Options

Biodegradable biocompatible collagenic materials have powerful biological effects, but they do not have as much strength and stiffness as traditional grafts. Combining collagen with high mechanical strength artificial, biodegradable and biocompatible materials might overcome this problem.

These tissue-engineered three-dimensional implants could be designed with stem cell seeding and the selective addition of growth factors. Moreover, gene therapy might be able to enhance the behaviour of the seeded cells to improve the bone healing response. Novel braided scaffolds have already been used in animal models for ACL tissue engineering.

19.5.2.4 Take-Home Messages

We advocate for the use of allografts in the setting of multi-ligament knee injuries. The decreased surgical time and morbidity, easier rehabilitation and greater tissue availability when using allografts outweigh the higher costs and slower period of incorporation.

A concern with the use of allografts is the small but serious risk of viral and bacterial disease transmission. Allografts are also expensive and their availability may be limited.

Ultimately, the choice of graft is dependent on the surgeon's and the patient's preferences, the availability of graft sources as well as the number of ligaments requiring reconstruction or augmentation.

References

1. Myasaka KC, Daniel D, Stone ML, Hirschmann P. The incidence of knee ligament injuries in the general population. Am J Knee Surg. 1991;4:3–7.
2. Shelbourne KD, Gray T. Minimum 10-year follow-up of patients after an acute, isolated posterior cruciate ligament injury treated non-operatively. Am J Sports Med. 2013;41(7):1526–33.
3. Ogata K, McCarthy JA, Dunlap J, et al. Pathomechanics of posterior sag of the tibia in posterior cruciate deficient knees. An experimental study. Am J Sports Med. 1988;16(6):630–6.
4. Servant CTJ, Ramos JP, Thomas NP. The accuracy of magnetic resonance imaging in diagnosing chronic posterior cruciate ligament injury. Knee. 2004;11:265–70.
5. Pacheco R, Ayre C, Bollen SR. Posterolateral corner injury associated with ACL rupture a commonly missed diagnosis. J Bone Jt Surg. 2011;93B: 194–7.
6. Burks RT, Schaffer JJ. A simplified approach to the tibial attachment of the posterior cruciate ligament. Clin Orthop Relat Res. 1990;254:216–9.
7. Nuelle CW, Milles JL, Pfeiffer FM, et al. Biomechanical comparison of five posterior cruciate ligament reconstruction techniques. J Knee Surg. 2017;30(6):523–31.
8. Jung TM, Lubowicki A, Wienand A, Wagner M, Weiler A. Knee stability after posterior cruciate ligament reconstruction in female versus male patients: a prospective matched-group analysis. Arthroscopy. 2017;27(3):399–403.
9. Wajsfisz A, Christel P, Djian P. Does reconstruction of isolated chronic posterior cruciate ligament injuries restore normal knee function? Orthop Traumatol Surg Res. 2010;96(4):388–93.
10. Becker EH, Watson JD, Dreese JC. Investigation of multiligamentous knee injury patterns with associated injuries presenting at a level I trauma center. J Orthop Trauma. 2013;27(4):226–31.
11. Fanelli GC, Edson CJ. Posterior cruciate ligament injuries in trauma patients, part II. Arthroscopy. 1995;11(5):526–9.
12. Harner CD, Hoher J. Evaluation and treatment of posterior cruciate ligament injuries. Am J Sports Med. 1998;26(3):471–82.
13. Kim SJ, Jung M, Moon HK, Kim SG, Chun YM. Anterolateral transtibial posterior cruciate ligament reconstruction combined with anatomical reconstruction of posterolateral corner insufficiency: comparison of single-bundle versus double-bundle posterior cruciate ligament reconstruction over a 2- to 6-year follow-up. Am J Sports Med. 2011;39(3):481–9.
14. Kim SJ, Kim SH, Chun YM, Hwang BY, Choi DH, Yoon JY. Clinical comparison of conventional and remnant-preserving transtibial single-bundle posterior cruciate ligament reconstruction combined with posterolateral corner reconstruction. Am J Sports Med. 2012;40(3):640–9.
15. Kennedy NI, Wijdicks CA, Goldsmith MT, et al. Kinematic analysis of the posterior cruciate ligament, part 1: the individual and collective function of the anterolateral and posteromedial bundles. Am J Sports Med. 2013;41(12):2828–38.
16. Xu X, Huang T, Liu Z, et al. Hamstring tendon autograft versus LARS artificial ligament for arthroscopic posterior cruciate ligament reconstruction in a long-term follow-up. Arch Orthop Trauma Surg. 2014 Dec;134(12):1753–9.
17. Yang JH, Yoon JR, Jeong HI, et al. Second-look arthroscopic assessment of arthroscopic single-bundle posterior cruciate ligament reconstruction: comparison of mixed graft versus Achilles tendon allograft. Am J Sports Med. 2012 Sep;40(9):2052–60.
18. Gray H, Fedden WF, Jex-Blake AJ, Howden R. Anatomy, descriptive and applied. New York: Longmans, Green; 1918.
19. LaPrade RF, Ly TV, Wentorf FA, Engebretsen L. The posterolateral attachments of the knee: a qualitative and quantitative morphologic analysis of the fibular collateral ligament, popliteus tendon, popliteofibular ligament, and lateral gastrocnemius tendon. Am J Sports Med. 2003;31(6):854–60.
20. Ewan WJ, LaPrade C, LaPrade RF. Anatomy and biomechanics of the lateral side of the knee. Sports Med Arthrosc Rev. 2015;23(1):2–9.
21. Geeslin AG, Laprade RF, Geeslin AG, Laprade RF. Location of bone bruises and other grade III isolated and combined. Am J Sports Med. 2010;38(12):2502–8.
22. Fanelli GC. Treatment of combined anterior cruciate ligament-posterior cruciate ligament-lateral side injuries of the knee. Clin Sports Med. 2000;19(3):493–502.
23. Hughston JC, Andrews JR, Cross MJ, Moschi A. Classification of knee ligament instabilities. Part II. The lateral compartment. J Bone Jt Surg Am. 1976;58(2):173–9.
24. Schenck RC Jr. The dislocated knee. Instr Course Lect. 1994;43:127–36.
25. Schweller EW, Ward PJ. Posterolateral corner knee injuries: review of anatomy and clinical evaluation. JAOA. 2015;115(12):725–31.
26. Ricchetti ET, Sennett BJ, Huffman GR. Acute and chronic management of posterolateral corner injuries of the knee. Orthopedics. 2008;31(5):479–90.
27. van der Wal WA, Heesterbeek PJC, van Tienen TG, Busch VJ, van Ochten JHM, Wymenga AB. Anatomical reconstruction of posterolateral corner and combined injuries of the knee. Knee Surg Sports Traumatol Arthrosc. 2016;24(1):221–8.
28. Mccarthy M, Camarda L, Wijdicks CA, Johansen S, Engebretsen L, Laprade RF. Anatomic posterolateral knee reconstructions require a popliteofibular

ligament reconstruction through a tibial tunnel. Am J Sports Med. 2010;38(8):1674–81.

29. Niki Y, Matsumoto H, Otani T, Enomoto H, Toyama Y, Suda Y. A modified Larson's method of posterolateral corner reconstruction of the knee reproducing the physiological tensioning pattern of the lateral collateral and popliteofibular ligaments. Sports Med Arthrosc Rehabil Ther Technol. 2012;4(1):21.
30. Laprade RF, Wijdicks C. The management of injuries to the medial side of the knee. J Orthop Sports Phys Ther. 2012;42(3):221–33.
31. Fetto J, Marshall J. Medial collateral ligament injuries of the knee: a rationale for treatment. Clin Orthop Relat Res. 1978;132:206–18.
32. Jacobson KE, Chi FS. Evaluation and treatment of medial collateral ligament and medial-sided injuries of the knee. Sports Med Arthrosc. 2006;14(2):58–66.
33. Kannus P. Long-term results of conservatively treated medial collateral ligament injuries of the knee joint. Clin Orthop Relat Res. 1988;226:103–12.
34. Warren LFF, Marshall JLL. The supporting structures and layers on the medial side of the knee: an anatomical analysis. J Bone Jt Surg Am. 1979;61(1):56–62.
35. Hughston J, Andrews JR, Cross MJ, Moschi A. Classification of knee ligament instabilities * part I. The medial compartment and cruciate ligaments. J Bone Jt Surg. 1976;58-A(2):159–72.
36. Robinson JR, Sanchez-Ballester J, Bull AM, Thomas Rde W, Amis AA. The posteromedial corner revisited. J Bone Jt Surg. 2004;86(5):674–81.
37. Bonnin M, Amendola A, Bellemans J, MacDonald S, Ménétrey J, editors. The knee joint: surgical techniques and strategies. Paris: Springer; 2012.
38. Robinson JR, Bull AMJ, Thomas RRD, Amis A. The role of the medial collateral ligament and posteromedial capsule in controlling knee laxity. Am J Sports Med. 2006;34(11):1815–23.
39. Griffith C, LaPrade R, Johansen S, Armitage B, Wijdicks C, Engebretsen L. Medial knee injury: part 1, static function of the individual components of the main medial knee structures. Am J Sports Med. 2009;37(9):1762–70.
40. Coobs BR, Wijdicks CA, Armitage BM, et al. An in vitro analysis of an anatomical medial knee reconstruction. Am J Sports Med. 2010;38(2):339–47.
41. Tibor LM, Marchant MH, Taylor DC, Hardaker WT, Garrett WE, Sekiya JK. Management of medial-sided knee injuries, part 2: posteromedial corner. Am J Sports Med. 2011;39(6):1332–40.
42. Haimes JL, Wroble RR, Grood ES, Noyes FR. Role of the medial structures in the intact and anterior cruciate ligament-deficient knee: limits of motion in the human knee. Am J Sports Med. 1994;22(3):402–9.
43. Smyth MP, Koh JL. A review of surgical and nonsurgical outcomes of medial knee injuries. Sports Med Arthrosc. 2015;23(2):e15–22.
44. Phisitkul P, James SL, Wolf BR, Amendola A. MCL injuries of the knee: current concepts review. Iowa Orthop J. 2006;26(Mcl):77–90.
45. Hughston JC, Eilers AF. The role of the posterior oblique ligament in repairs of acute medial (collateral) ligament tears of the knee. J Bone Joint Surg Am. 1973;55(5):923–40.
46. Wijdicks CA, Ewart DT, Nuckley DJ, Johansen S, Engebretsen L, Laprade RF. Structural properties of the primary medial knee ligaments. Am J Sports Med. 2010;38(8):1638–46.
47. Robinson JR, Bull AMJ, Amis AA. Structural properties of the medial collateral ligament complex of the human knee. J Biomech. 2005;38(5):1067–74.
48. Jones L, Bismil Q, Alyas F, Connell D, Bell J. Persistent symptoms following non operative management in low grade MCL injury of the knee & the role of the deep MCL. Knee. 2017;16(1):64–8.
49. Narvani A, Mahmud T, Lavelle J, Williams A. Injury to the proximal deep medial collateral ligament: a problematical subgroup of injuries. J Bone Jt Surg Br. 2010;92(7):949–53.
50. Abbott LC, Saunders JB, Bost FC, Anderson CE. Injuries to the ligaments of the knee joint. J Bone Jt Surg. 1944;26(3):503–21.
51. Duffy PS, Miyamoto RG. Management of medial collateral ligament injuries in the knee: an update and review. Phys Ther Sport. 2010;38(2):48–54.
52. Reider B. Medial collateral ligament injuries in athletes. Sports Med. 1996;21(2):147–56.
53. Kakarlapudi TK, Bickerstaff DR. Knee instability: isolated and complex. West J Med. 2001;174(4):266–72.
54. Cross MJ, Wootton JR, Bokor DJ, Sorrenti SJ. Acute repair of injury to the anterior cruciate ligament. A long-term followup. Am J Sports Med. 1993;21(1):128–31.
55. Mazzoca AD, Nissen CW, Geary M, Adams DJ. Valgus medial collateral ligament rupture causes concomitant loading and damage of the anterior cruciate ligament. J Knee Surg. 2003;16(3):148–51.
56. O'Donoghue DH. Surgical treatment of injuries to ligaments of the knee. J Am Med Assoc. 1959;169(13):1423–31.
57. Noyes FR, Barber-Westin SD. A systematic review of the incidence and clinical significance of postoperative meniscus transplant extrusion. Knee Surg Sports Traumatol Arthrosc. 2014;23(1):290–302.
58. Slocum DB, Larson RL. Rotatory instability of the knee. J Bone Jt Surg Am. 1968;50(2):211–25.
59. Franklin JL, Rosenberg TD, Paulos LE, France EP. Radiographic assessment of instability of the knee due to rupture of the anterior cruciate ligament. A quadriceps-contraction technique. J Bone Joint Surg Am. 1991;73(3):365–72.
60. Engebretsen L, Lind M. Anteromedial rotatory laxity. Knee Surg Sports Traumatol Arthrosc. 2015;23(10):2797–804.
61. Phisitkul P, Wolf BR, Amendola A. Role of high tibial and distal femoral osteotomies in the treatment of lateral-posterolateral and medial instabilities of the knee. Sports Med Arthrosc. 2006;14(2):96–104.

62. Kurzweil PR, Kelley ST. Physical examination and imaging of the medial collateral ligament and posteromedial corner of the knee. Sports Med Arthrosc. 2015;14(2):67–73.
63. Loredo R, Hodler J, Pedowitz R, Yeh LR, Trudell D, Resnick D. Posteromedial corner of the knee: MR imaging with gross anatomic correlation. Skelet Radiol. 1999;28(6):305–11.
64. Schein A, Matcuk G, Patel D, et al. Structure and function, injury, pathology, and treatment of the medial collateral ligament of the knee. Emerg Radiol. 2012;19(6):489–98.
65. Miller MD, Osborne JR, Gordon WT, Hinkin DT, Brinker MR. The natural history of bone bruises a prospective study of magnetic resonance imaging-detected trabecular microfractures in patients with isolated medial collateral ligament injuries. Am J Sports Med. 1998;26(1):15–9.
66. Nakamura N, Horibe S, Toritsuka Y, Mitsuoka T, Yoshikawa H, Shino K. Acute grade III medial collateral ligament injury of the knee associated with anterior cruciate ligament tear. The usefulness of magnetic resonance imaging in determining a treatment regimen. Am J Sports Med. 2003;31(2):261–7.
67. Grant JA, Tannenbaum E, Miller BS, Bedi A. Treatment of combined complete tears of the anterior cruciate and medial collateral ligaments. Arthroscopy. 2012;28(1):110–22.
68. Laprade RF, Bernhardson AS, Griffith CJ, Macalena JA, Wijdicks CA. Correlation of valgus stress radiographs with medial knee ligament injuries: an in vitro biomechanical study. Am J Sports Med. 2010;38(2):330–8.
69. Staubli HU, Jakob RP. Posterior instability of the knee near extension. A clinical and stress radiographic analysis of acute injuries of the posterior cruciate ligament. J Bone Jt Surg Br. 1990;72(2):225–30.
70. Lerat JL, Moyen BL, Cladière F, Besse JL, Abidi H. Knee instability after injury to the anterior cruciate ligament. Quantification of the Lachman test. J Bone Jt Surg Br. 2000;82:42–7.
71. Ellsasser JC, Reynolds FC, Omohundro JR. The nonoperative treatment of collateral ligament injuries of the knee in professional football players. An analysis of seventy-four injuries treated non-operatively and twenty-four injuries treated surgically. J Bone Jt Surg Am. 1974;56(6):1185–90.
72. Indelicato PA. Non-operative treatment of complete tears of the medial collateral ligament of the knee. J Bone Jt Surg Am. 1983;65(3):323–9.
73. Lundberg M, Messner K. Ten-year prognosis of isolated and combined medial collateral ligament ruptures. A matched comparison in 40 patients using clinical and radiographic evaluations. Am J Sports Med. 1997;25(1):2–6.
74. Derscheid GL, Garrick JG. Medial collateral ligament injuries in football. Nonoperative management of grade I and grade II sprains. Am J Sports Med. 1981;9(6):365–8.
75. Holden DL, Eggert AW, Butler JE. The nonoperative treatment of grade I and 11 medial collateral ligament injuries to the knee. Am J Sports Med. 1983;11(5):340–4.
76. Lundberg M, Messner K. Long-term prognosis of isolated partial medial collateral ligament ruptures. A ten-year clinical and radiographic evaluation of a prospectively observed group of patients. Am J Sports Med. 1984;24(2):160–3.
77. Hildebrand KA, Woo SL, Smith DW, et al. The effects of platelet-derived growth factor-BB on healing of the rabbit medial collateral ligament an in vivo study. Am J Sports Med. 1998;26(4):549–54.
78. Batten ML, Hansen JC, Dahners LE. Influence of dosage and timing of application of platelet-derived growth factor on early healing of the rat medial collateral ligament. J Orthop Res. 1996;14(5):736–41.
79. Moorman CT, Kukreti U, Fenton DC, Belkoff SM. The early effect of ibuprofen on the mechanical properties of healing medial collateral ligament. Am J Sports Med. 1999;27(6):738–41.
80. Wright R, Mackey RB, Silva M, Steger-May K. Smoking and mouse MCL healing. J Knee Surg. 2010;23(4):193–9.
81. Stannard JP. Evaluation and treatment of medial instability of the knee. Sports Med Arthrosc. 2015;23(2):91–5.
82. Shelbourne DK. Management of combined and medial injuries collateral ligaments. J Bone Jt Surg Am. 1995;77(5):800–6.
83. Petersen W, Laprell H. Combined injuries of the medial collateral ligament and the anterior cruciate ligament. Early ACL reconstruction versus late ACL reconstruction. Arch Orthop Trauma Surg. 1999;119(1):258–62.
84. Noyes FR, Barber-Westin SD. The treatment of acute combined ruptures of the anterior cruciate and medial ligaments of the knee. Am J Sports Med. 1995;23(4):380–91.
85. Halinen J, Lindahl J, Hirvensalo E, Santavirta S. Operative and nonoperative treatments of medial collateral ligament rupture with early anterior cruciate ligament reconstruction: a prospective randomized study. Am J Sports Med. 2006;34(7):1134–40.
86. Hara K, Niga S, Ikeda H, Cho S, Muneta T. Isolated anterior cruciate ligament reconstruction in patients with chronic anterior cruciate ligament insufficiency combined with grade II valgus laxity. Am J Sports Med. 2008;36:333–9.
87. Kaeding CC, Pedroza AD, Parker RD, Spindler KP, McCarty EC, Andrish JT. Intra-articular findings in the reconstructed multiligament-injured knee. Arthroscopy. 2005;21(4):424–30.
88. Forsythe B, Mascarenhas R, Pombo MW, Harner CD. Combined injuries to the posterior cruciate ligament and medial collateral ligament of the knee. In: The knee joint: surgical techniques and strategies. Paris: Springer; 2012. p. 421–6.
89. Bergfeld JA, McAllister DR, Parker RD, Valdevit AD, Kambic H. The effects of tibial rotation on pos-

terior translation in knees in which the posterior cruciate ligament has been cut. J Bone Joint Surg Am. 2001;83-A(9):1339–43.

90. Petersen W, Loerch S, Schanz S, Raschke M, Zantop T. The role of the posterior oblique ligament in controlling posterior tibial translation in the posterior cruciate ligament-deficient knee. Am J Sports Med. 2008;36:495–501.
91. Desai R, Parikh S, Laor T. Intra-articular entrapment of the medial collateral ligament. Orthopedics. 2010;33(9):687.
92. Patel JJ. Intra-articular entrapment of the medial collateral ligament: radiographic and MRI findings. Skelet Radiol. 1999;28(11):658–60.
93. Stannard JP, Black BS, Azbell C, D a V. Posteromedial corner injury in knee dislocations. J Knee Surg. 2012;25(5):429–34.
94. Howells NR, Brunton LR, Robinson JR, Porteous AJ, Eldridge JD, Murray JR. Acute knee dislocation: an evidence based approach to the management of the multiligament injured knee. Injury. 2011;42(11):1198–204.
95. Lubowitz JH, Mackay G, Gilmer B. Knee medial collateral ligament and posteromedial corner anatomic repair with internal bracing. Arthrosc Tech. 2014;3(4):e505–8.
96. Bosworth D. Transplantation of the semitendinosus for repair of laceration of medial collateral ligament of the knee. J Bone Jt Surg Am. 1952;34:196–202.
97. Kim S-J, Lee D-H, Kim T-E, Choi N-H. Concomitant reconstruction of the medial collateral and posterior oblique ligaments for medial instability of the knee. J Bone Joint Surg (Br). 2008;90(10):1323–7.
98. Lind M, Jakobsen BW, Lund B, Hansen MS, Abdallah O, Christiansen SE. 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. 2009;37(6):1116–22.
99. Yoshiya S, Kuroda R, Mizuno K, Yamamoto T, Kurosaka M. Medial collateral ligament reconstruction using autogenous hamstring tendons: technique and results in initial cases. Am J Sports Med. 2005;33(9):1380–5.
100. Zhang H, Bai X, Sun Y, Han X. Tibial inlay reconstruction of the medial collateral ligament using Achilles tendon allograft for the treatment of medial instability of the knee. Knee Surg Sports Traumatol Arthrosc. 2014;22(2):279–84.
101. Marx RG, Hetsroni I. Surgical technique: medial collateral ligament reconstruction using Achilles allograft for combined knee ligament injury. Clin Orthop Relat Res. 2012;470(3):798–805.
102. Borden PS, Kantaras AT, Caborn DNM. Medial collateral ligament reconstruction with allograft using a double-bundle technique. Arthroscopy. 2002;18(4):E19.
103. Laprade RF, Wijdicks CA. Development of an anatomic medial knee reconstruction. Clin Orthop. 2012;470:806–14.
104. Feeley BT, Muller MS, Allen AA, Granchi CC, Pearle AD. Biomechanical comparison of medial collateral ligament reconstructions using computer-assisted navigation. Am J Sports Med. 2009;37(6):1123–30.
105. Kremen TJ, McAllister DR. Graft selection in multiple ligament injured knee surgery. In: Fanelli GC, editor. The multiple ligament injured knee: a practical guide to management, vol. 115. New York: Springer Science + Business Media; 2013. Chapter 9. p. 115–28.
106. Flahiff CM, Brooks AT, Hollis JM, Vander Schilden JL, Nicholas RW. Biomechanical analysis of patellar tendon allografts as a function of donor age. Am J Sports Med. 1995;23(3):354–8.
107. West RV, Harner CD. Graft selection in anterior cruciate ligament reconstruction. J Am Acad Orthop Surg. 2005;13:197–207.
108. Weiss NG, Kaplan LD, Graf BJ. Graft selection in surgical reconstruction of the multiple-ligament-injured. Oper Tech Sports Med. 2003;11(3):218–25.
109. Levy BA, Dajani KA, Whelan DB, et al. Decision making in the multiligament-injured knee: an evidence based systematic review. Arthroscopy. 2009;25(4):430–8.
110. Rodeo SA, Weber AE, Verdonk R, Monllau JC, Heinrichs E-L. The use of allografts in sports medicine. In: Warwick RM, editor. Tissue and cell clinical use. An essential guide. Hoboken: Blackwell Publishing Ltd; 2012.

20 How to Operatively Stabilize the Patella

Siebren Tigchelaar, Sebastiaan van de Groes, Nico Verdonschot, Marie Askenberger, Petri Sillanpää, and Sander Koëter

20.1 Introduction

The patellofemoral joint is an exceptional joint, unlike any other joint in the human body. The patella, albeit just a small part of the knee, has an important function as a fulcrum for the extensor mechanism of the knee. Patellofemoral problems account for a significant amount of consultations in both general and orthopedic practice. About 11–17% of all knee consultations in general practice concern patellofemoral complaints [1]. The incidence of primary patellar dislocations is 5.8 per 100,000 and increases to 29 per 100,000 in adolescents. Recurrent instability vigorously increases the number of patellar dislocations, as recurrent patellar instability occurs in 17% of persons after a first dislocation and in 49% after a second dislocation [2]. Unlike instability in other joints, patellofemoral instability is usually the direct consequence of congenital malformations of the patella and/or femoral trochlea. These malformations result from aberrations and disruptions in the evolutionary, embryological, and genetic development of the patellofemoral joint. The introduction of this chapter addresses the development of the patellofemoral joint in order to understand the etiology and causative factors of patellofemoral instability.

20.2 Congenital and Biomechanical Causes of Patella Instability

20.2.1 Congenital Aspects

Normal embryonic development of the anatomic structures of the patellofemoral joint is of crucial importance for proper patellar function and stability. The differentiation of the patella and the patellar tendon starts at day 37 with chondrification starting at day 45 of gestation. The patella increases in relative size up to the sixth month of fetal life, and after which it increases at the same rate as the other bones of the lower extremity. Initially, the medial and lateral patellar facets are equal in size,

S. Tigchelaar • S. Koëter (✉)
Department of Orthopaedic Surgery, Canisius Wilhelmina Ziekenhuis, Nijmegen, The Netherlands
e-mail: s.koeter@cwz.nl

S. van de Groes
Department of Orthopaedic Surgery, Radboud Universitair Medisch Centrum, Nijmegen, The Netherlands

N. Verdonschot
Orthopaedic Research Lab, Radboud University Medical Center, Nijmegen, The Netherlands

M. Askenberger
Department of Pediatric Orthopedics, Astrid Lindgren Children's Hospital, Karolinska University Hospital, Solna, Sweden

P. Sillanpää
Department of Orthopaedic Surgery, Dextra Koskisairaala Hospital, Tampere, Finland

G.M.M.J. Kerkhoffs et al. (eds.), *ESSKA Instructional Course Lecture Book*,
https://doi.org/10.1007/978-3-662-56127-0_20

but at week 23 of gestation, the lateral facet has become the more predominant, which is the key characteristic of the adult patella. Ossification of the patella usually starts at ages 5–6 but is sometimes visible on radiographs at ages 2–3 [3].

During human embryonic development, limb patterning is accompanied by rotation of the limbs. Initially, the upper and lower limb buds extend laterally from the body wall with the thumb and great toe facing cranially and the flexor surfaces facing ventrally. Subsequently, the limbs shift into a more ventral position with both the thumb and great toe still facing cranially, but the flexor surfaces are now facing medially. The limbs rotate around their proximo-distal axis between the sixth and eighth week of embryonic development. The upper and lower limbs rotate in opposite directions, the upper limbs rotate dorsally, and lower limb rotation occurs in the ventral direction. At this end stage of limb rotation, the flexor/palmar surfaces of the hands face ventrally, the flexor/plantar surfaces of the feet face dorsally, and the elbows and knees face outward (Fig. 20.1). Consequently, the patella, which primordial anlage is a dorsal structure, comes to lie ventrally during limb development [4]. It is argued that the patellar instability, which is always lateral, is frequently caused by a deficiency in this dorsoventral and rotational development.

In the embryo, the knee develops in a position of 90° flexion. This means that the patella initially conforms to the distal aspect of the femoral condyles, the part that will articulate with the tibial plateau in stance. The general adult form of the trochlear surface of the femur is achieved very early in fetal life, before movement has occurred. This means that it is not formed in contact with or in response to the patella but to the quadriceps musculature. As with most anatomic structures, form follows function and the final shape of both the patella and the femoral trochlea will be modified by use [5].

20.2.2 Biomechanical Aspects

The patella is the largest sesamoid bone in the human body, and its most important function is to facilitate extension of the knee by increasing the efficacy of the quadriceps muscle. This is achieved through the patella's function as a fulcrum, thus anteriorly displacing the line of pull and increasing the moment arm of the quadriceps muscle force in relation to the center of rotation

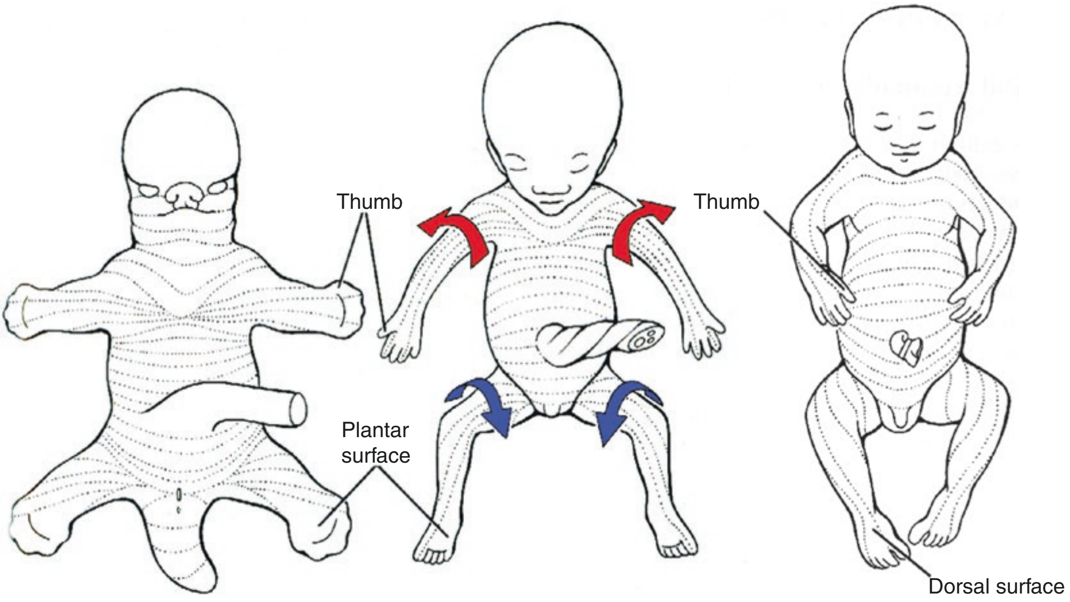


Fig. 20.1 Rotation of the upper and lower limbs between the sixth and eighth week of gestation

of the knee. The patella enhances the force of extension by as much as 50% throughout the entire range of motion (ref: Fulkerson). The function of the patellofemoral joint is normally maintained by a complex interaction between soft tissues and bony structures. The structures responsible for its stability can be divided into three groups: the *active* stabilizers represented by the quadriceps muscles, the *passive* stabilizers (particularly the retinacula, of which the medial and lateral patellofemoral ligaments are a part), and the *static* stabilizers represented by the articular surfaces. In normal knees, these structures act in harmony to maintain stability of the joint. In case of an imbalance between these stabilizers, patellofemoral dysfunction occurs.

Radiologic evaluation of patellofemoral dysfunction is traditionally performed using conventional radiographs and computed tomography (CT) scanning. Using these common radiologic modalities, excellent work by Dejour and his coworkers from the Lyon School has revealed four different factors that are significantly correlated with recurrent patellar instability and are currently considered to be the primary causes of patellar instability [6]. These four factors are:

- Patella alta
  In case of patella alta, or high-riding patella, the position of the patella is more proximal in relation to the trochlear groove. Due to this position of the patella, it engages in the trochlea only at greater flexion of the knee. Therefore, the patella has a greater trajectory in which there is no bony (static) restraint which prevents a lateral dislocation of the patella [7]. In this situation, the stability of the patellofemoral joint is completely dependent on the active (quadriceps muscles, in particular the m. vastus medialis obliquus) and passive (medial retinaculum) stabilizers of the patella.
- An increased tibial tubercle-trochlear groove (TT-TG) distance
  If the position of the patellar tendon insertion, the tibial tubercle, is located more lateral on the tibia, the distance between the trochlear groove and the tibial tubercle increases. This in turn increases the angle between the quadriceps muscles and the patellar tendon, the Q angle, creating a larger lateral force vector on the patella. A lateralized tibial tubercle therefore leads to an increased laterally oriented force on the patella. A higher lateral force on the patella may lead to either instability or initiate patellofemoral pain by raising the cartilaginous pressure from the patella on the lateral femoral condyle.
- Trochlear dysplasia
  In 96% of patients with patellar instability, trochlear dysplasia is present. Trochlear dysplasia is a common denominator for all types of aberrant anatomy of the femoral trochlea. All types of trochlear dysplasia share either a flat or convex trochlear groove which decreases lateral restraint and/or a bump at the entrance of the trochlea which prevents the patella from easy entry into the trochlea. A flat femoral trochlea reduces the bony lateral restraint by 70% at 20–30° of flexion, which makes the trochlea the largest contributor to patellofemoral stability from 20–30° up to full flexion.
- Medial patellofemoral ligament (MPFL) rupture
  The MPFL is a thin ligamentous structure within the medial capsule of the knee with its origin on the proximal half of the patella and its insertion just proximal and posterior to the medial epicondyle of the femur. After a first-time patellar dislocation, the MPFL is ruptured in 90–100% of patients. The MPFL is the single most important restraint in full or near full extension of the knee. It is responsible for 50–60% of the lateral restraint of the patella at 0–20° of knee flexion.

20.3 Clinical Relevance and State-of-the-Art Treatment

Primary patellar dislocations should be treated conservatively by brief immobilization, followed by active mobilization. Recurrent dislocations can be treated surgically. In clinical practice, usually a combination of factors leads to patella instability. The optimal, state-of-the-art, operative treatment is tailor-made (a la carte) and has to be individualized in every patient. Adequate physical examination, a good knowledge of the patients' specific (sports) goals, and different radiologic modalities—such as conventional radiographs, computed tomography (CT) scan, and magnetic resonance imaging (MRI)—are helpful in making the optimal surgical preoperative plan. In the following paragraphs is a short outline of the different treatment options and there indications.

20.3.1 MPFL Reconstruction: Novel Insights

After patella dislocation, the thin fibers of the MPFL are always affected. Although they have some healing potential, elongation and wearing of the structures almost always persist. Repair of this structure leads to poor residual restraint, and—in cases of recurrent dislocation—reconstructing of the MPFL has become a key procedure for stabilizing the patella. Different techniques to reconstruct the MPFL have been described: static techniques in which the graft is fixed rigidly to the bone or dynamic techniques with soft tissue fixation. Static MPFL reconstruction with the use of implants at both the patella and femoral side is most commonly used. However, dynamic reconstruction deforms more easily and presumably functions more like the native MPFL.

20.3.2 MPFL in the Young Patient

In young patients, with open physes, bony procedures are usually contraindicated because of the likelihood of postoperative growth disturbances. Soft tissue procedures may be indicated in patients with severe limitations caused by the patella instability. Various procedures have been described in literature, ranging from muscular transfer, patella tendon realignment, to capsular procedure. Examples of the latter are VMO transfer, Roux-Goldthwait procedures, and capsular reefing/lateral release. MPFL reconstruction has replaced much of the previously mentioned operations. The employed technique differs slightly from the techniques use in adults, but the basics are the same. In some cases the MPFL reconstructions can be combined with other soft tissue procedure.

20.3.3 Tibial Tubercle Transfer: Is It Still Indicated?

Historically, tibial tubercle transfer was the corner stone of patella surgery, but various articles showed poor long-term results of this treatment with high changes of developing osteoarthritis. The procedure developed a bad name, and its central role in the arsenal of the patella surgeon has now been overtaken by the MPFL reconstruction. The question arises: is there still a role for this procedure or is it outdated?

The transfer of the tibial tubercle has an enormous biomechanical effect on the force acting upon the patella, and by alteration of the tibial tubercle-trochlear groove (TT-TG) distance and/or the patella height, the stability can be improved. Most experienced surgeons use a tibial tubercle transfer in selected cases, but the TT-TG threshold for correction varies between surgeons and institutions, from 15 to 20 mm. The measurement has a measurement error of around 2 mm [6]. Care must be taken not to overcorrect the abnormality, because this can lead to unacceptably high retropatellar pressures and the development of osteoarthritis. We use a self-centering technique to prevent overcorrection; please refer to Fig. 20.2 for a brief description of this technique. Using this technique we found good improvement in functional scores (VAS pain, Lysholm, and Kujala scores are improved significantly compared to their preoperative values and do not deteriorate at final follow-up), low postoperative instability, and a limited deterioration in osteoarthritis (similar to the natural course of osteoarthritis after patellar dislocation without surgical treatment). Based on these results, we conclude that this self-centering tibial tubercle osteotomy provides good long-term results without inducing

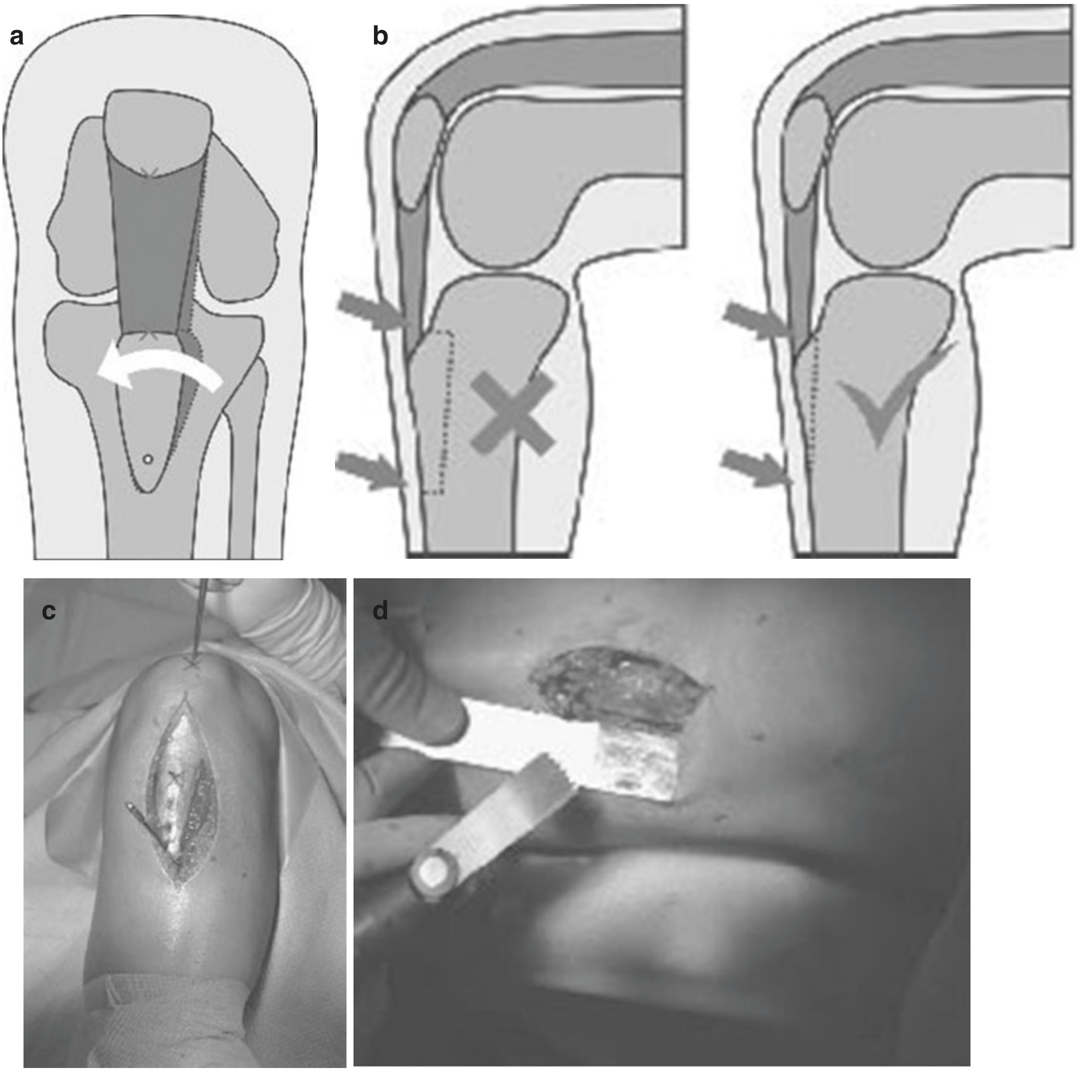


Fig. 20.2 Self-centering transfer technique. After a straight osteotomy, the knee is flexed and the tibial tuberosity medializes. Care must be taken to make a straight (and not a step cut) osteotomy because this will lead to stress rising in the tibia during weight bearing

progressive osteoarthritis [8]. Nowadays, it is usually combined with MPFL reconstruction, the cornerstone of patella-stabilizing surgery (Figs. 20.3 and 20.4).

20.3.4 Trochleoplasty for Patellar Instability

Trochlear dysplasia is a universally accepted primary anatomic risk factor for patellar instability. Trochlea dysplasia can be diagnosed on axial radiographs by measuring the sulcus angle or—on true lateral radiographs—by identifying a specific sign for trochlear dysplasia, the “crossing sign.” Trochlea dysplasia can be classified in four categories (Dejour A, B, C, or D), according to the severity of the dysplasia [7]. MRI gives more precise analysis of the trochlear shape, including the cartilaginous shape of the trochlea, which does not follow bony morphology in the patellofemoral joint. Therefore, advanced imaging by

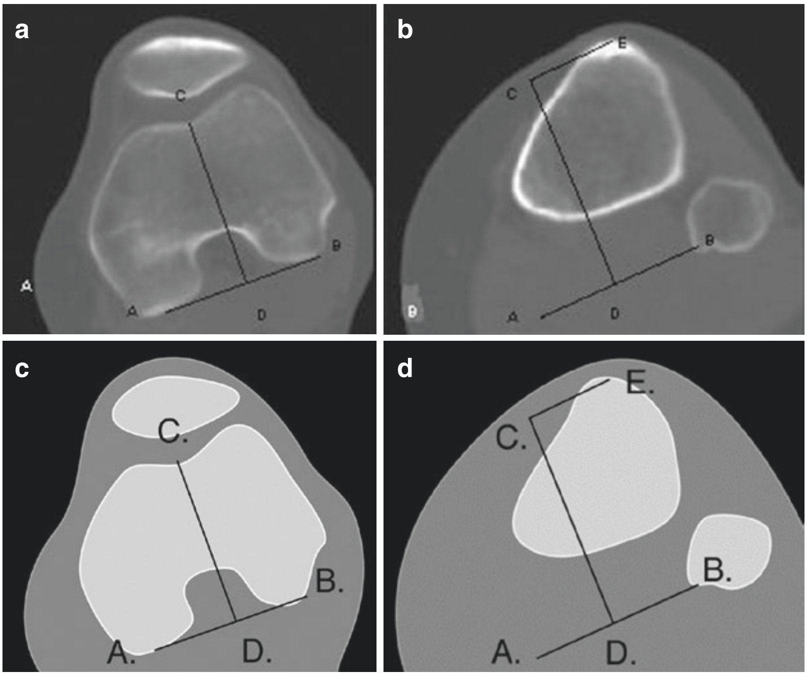


Fig. 20.3 Measurement technique for measuring the tibial tubercle-trochlear groove (TT-TG) distance on a CT or MRI. The deepest point of the sulcus is marked on Fig. A, the lines are transferred to the tibial tuberosity, and the medial point of the tuberosity is located, and the distance between C and E is the TT-TG

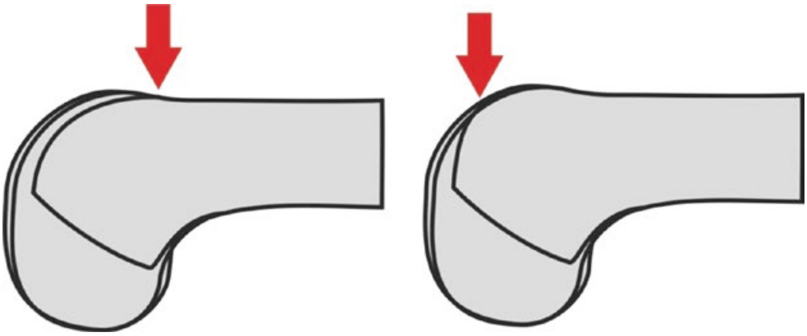


Fig. 20.4 The crossing sign can be seen best on a true lateral radiograph. The posterior condyles need to overlap; the crossing of the anterior part of the femoral trochlea with the line of the trochlea bottom is located at the arrow. The left figure is normal (no crossing of the lines), the right figure is dysplastic (with a crossing of the lines)

MRI is necessary when considering surgery for patellofemoral instability in patients with trochlea dysplasia [9].

There are no guidelines for when to correct the dysplastic trochlea by a trochleoplasty procedure. In high-degree trochlear dysplasia (Dejour B and D), with a highly abnormal shape of the trochlea, a prominent bump deformity, and complete lack of sulcus groove, other surgical methods may not provide adequate stabilization of the patella. In patients with mild trochlea dysplasia (Dejour A), the shallow trochlear groove does not significantly compromise patellar stability, and these patients can be treated with isolated MPFL reconstruction if there are no other major anatomical risk factors. Generally, trochleoplasty (as a primary or secondary procedure) is only indicated in patients with high-degree trochlea dysplasia. To gain optimal patella stabilization, trochleoplasty is usually combined with MPFL reconstruction and some form of lateral capsular structure lengthening.

Trochleoplasty aims at removing the trochlear bump and creating a normal or nearly normal sulcus groove. Different surgical techniques have been described in the literature, all of which deepen the trochlear groove by removing excessive bone after creating a cartilaginous flap. This cartilage flap can be created by various methods, either by splitting the cartilage (thick flap technique) in medial and lateral flaps or mobilizing the flap subchondrally from proximal without touching the cartilaginous surface (thin flap technique). After a trochlear groove has been created, the cartilage flap is fixed, and soft tissue balancing is performed at lateral structures, combined with medial soft tissue restraints (MPFL) reconstruction. Despite the fact that trochleoplasty is a relatively extensive surgery for the patellofemoral joint, complications such as cartilage viability issues are rare [10], and rehabilitation closely follows the standard postoperative MPFL reconstruction protocol to avoid arthrofibrosis (which has been described as a potential risk in earlier studies [11]). Recent outcomes for trochleoplasty report good results in terms of patellar stability, though long-term patient-reported outcomes in different surgical techniques are yet to be determined.

20.4 Future Directions

Since patella instability has a number of anatomic causes, the surgical plan has to be tailor-made (a la carte). The specific indications for surgery and the exact surgical plan can vary among surgeons and between countries, depending on regional traditions and expertise. Future research can be helpful in establishing which interventions provide the best results and the smallest change on complications and which preoperative workup is needed to define the underlying anatomical problem.

The first step can be a more dynamic approach to the problem. Nowadays, surgery is planned based on 2D images, but patellar instability is a dynamic problem. New imaging techniques are available for the real-time dynamic scanning of patients. This provides new insights in the patella tracking and the influence of, for instance, the position of the tibial tubercle on tracking throughout the whole range of motion. This data can be used to plan surgery in a patient-specific manner, leading to more advanced planning techniques. Advanced computer modeling techniques can be used to plan, for instance, the optimal position of the tibial tubercle to reduce cartilage stresses and optimize patellar tracking. New surgical techniques, like robotics and navigation, can provide the tools needed to execute the plan in theater.

20.5 Take-Home Message

Recurrent patella dislocations are caused by a (combination of) underlying anatomic disorders. The four most critical factors are patella alta, MPFL insufficiency, increased tibial tubercle-trochlear groove distance, and trochlea dysplasia. These anatomic abnormalities have to be identified before a good surgical plan to stabilize the patella can be made. The surgical plan is tailor-made (a la carte) and depends on the anatomic abnormalities and the patients' characteristics and needs.

References

1. Crossley KM, van Middelkoop M, Callaghan MJ, Collins NJ, Rathleff MS, Barton CJ. Patellofemoral pain consensus statement from the 4th International Patellofemoral Pain Research Retreat, Manchester. Part 2: recommended physical interventions (exercise, taping, bracing, foot orthoses and combined interventions). Br J Sports Med. 2016;50:844–52.
2. Fithian DC, Paxton EW, Stone ML, et al. Epidemiology and natural history of acute patellar dislocation. Am J Sports Med. 2004;32:1114–21.
3. Fox AJ, Wanivenhaus F, Rodeo SA. The basic science of the patella: structure, composition, and function. J Knee Surg. 2012;25:127–41.
4. Bongers EM, van Kampen A, van Bokhoven H, Knoers NV. Human syndromes with congenital patellar anomalies and the underlying gene defects. Clin Genet. 2005;68:302–19.
5. Fulkerson JP. Disorders of the patellofemoral joint. 4th ed. New York: Lippincott Williams and Wilkins; 2004.
6. Dejour H, Walch G, Nove-Josserand L, Guier C. Factors of patellar instability: an anatomic

radiographic study. Knee Surg Sports Traumatol Arthrosc. 1994;2:19–26.

7. Tavernier T, Dejour D. Knee imaging: what is the best modality. J Radiol. 2001;82(387–405):407–388.
8. Tigchelaar S, van Essen P, Benard M, Koeter S, Wymenga A. A self-centering osteotomy of the tibial tubercle for patellar malt racking or instability, results with ten years follow-up. Bone Joint J. 2015;97-B:329–36.
9. van Huyssteen AL, Hendrix MR, Barnett AJ, Wakeley CJ, Eldridge JD. Cartilage-bone mismatch in the dysplastic trochlea. An MRI study. J Bone Joint Surg. 2006;88(5):688–91.
10. Schottle PB, Schell H, Duda G, Weiler A. Cartilage viability after trochleoplasty. Knee Surg Sports Traumatol Arthrosc. 2007;15:161–7. https://doi.org/10.1007/s00167-006-0148-0.
11. Donell ST, Joseph G, Hing CB, Marshall TJ. Modified Dejour trochleoplasty for severe dysplasia: operative technique and early clinical results. Knee. 2006;13(4):266–73.

21

Massive Retracted Rotator Cuff Tear: Treatment Options

Giuseppe Milano, Maristella F. Saccomanno,
Paolo Avanzi, Bartlomiej Kordasiewicz,
Ladislav Kovacic, Vladimir Senekovic, Jan Kany,
Peter Domos, Bruno Toussaint, Jérôme Bahurel,
and Mustafa Karahan

21.1 Introduction

Arthroscopic rotator cuff repair grants successful and predictable outcomes for small- and medium-sized tears; however it remains challenging and controversial for large and massive tears, which represent 10–40% of all tears [1]. The ideal repair should provide high initial fixation strength, restoration of the anatomic footprint, minimization of gap formation and mechanical stability until biologic healing occurs. Unfortunately, despite an improved knowledge of rotator cuff biomechanics and biology, tendon healing drops considerably to 47% for massive cuff tears [2]. Therefore, one of the main questions after visiting a patient with a large to massive cuff tear is "Will it be possible to successfully repair this tear?" Some will argue that repairability may have more to do with the surgeon's experience and skill rather than anything else. However, even for the most talented surgeons, tendon mobility is already established to a certain extent by tear retraction and chronicity. The rate of irreparable rotator cuff tears has been estimated to be between 6.5 and 30% [3]. Many surgical procedures have been described to treat these lesions, including palliative options such as long head of the biceps (LHB) tenotomy and cuff debridement, functional rotator cuff repair, biological augmentation with scaffolds, superior capsule reconstruction, subacromial biodegradable spacer, tendon transfers and even reverse

---

G. Milano (✉) • M.F. Saccomanno
Department of Orthopaedics, Catholic University,
Service of Shoulder Surgery, "A. Gemelli" University
Hospital, Rome, Italy
e-mail: giuseppe.milano@unicatt.it

P. Avanzi
Department of Orthopaedic Surgery,
Sacro Cuore Hospital, Negrar, Italy

B. Kordasiewicz
Department of Traumatology and Orthopaedics,
Centre of Postgraduate Medical Education,
Prof. Adam Gruca Clinical Hospital,
Konarskiego Str. 13, 05-400, Otwock, Poland

L. Kovacic • V. Senekovic
Department of Traumatology, University Medical
Centre, Ljubljana, Slovenia

J. Kany
Shoulder Department, Clinique de l'Union,
Saint Jean, France

P. Domos
Sheffield Teaching Hospitals NHS Foundation Trust,
The Northern General Hospital, Sheffield, UK

B. Toussaint • J. Bahurel
Clinique Générale, Annecy, France

M. Karahan
Department of Orthopedics and Traumatology,
Acibadem University Faculty of Medicine,
Tekin sok. No:8, 34718, Acibadem, Istanbul, Turkey

G.M.M.J. Kerkhoffs et al. (eds.), *ESSKA Instructional Course Lecture Book*,
https://doi.org/10.1007/978-3-662-56127-0_21

shoulder arthroplasty as the last option. The purpose of this chapter is to provide an overview of the different treatments that can be considered in case of massive retracted irreparable rotator cuff tears.

21.2 State-of-the-Art Treatment

Exact definition of massive tears is somehow still controversial. DeOrio and Cofield [4] described them as lesions characterized by an anteroposterior or medio-lateral diameter greater than 5 cm on preoperative magnetic resonance imaging (MRI). Gerber et al. [5] defined them as tears involving two or more tendons. In a recent meta-analysis, Henry et al. [6] used a hybrid definition that accounts for both length and number of tendons: greater than 3 cm in the coronal plane but with complete detachment of both supra- and infraspinatus tendons or 4 cm in the coronal plane and the complete detachment of at least one tendon.

With advanced chronicity and tissue deterioration, massive tears can become irreparable. Irreparable rotator cuff tears are well-defined lesions consisting of massive retracted tears that cannot be repaired primarily to their insertion onto the tuberosities despite conventional techniques of mobilization and soft tissue releases.

Massive cuff tears usually occur in two distinct groups of patients: degenerative tears in elderly, low-demand patients that became symptomatic after a minor trauma and traumatic tears in younger, active patients, often in the fourth to sixth decade of life, who present with dramatic symptoms of pain and disability after an acute effective trauma. Two distinct anatomic patterns can be recognized: postero-superior tears involving the supraspinatus superiorly and the infraspinatus (and rarely the teres minor) posteriorly and antero-superior tears involving the supraspinatus superiorly and subscapularis anteriorly. Both patterns alter the concavity compression mechanism and the balance of force couple, and greater forces are then required by both the deltoid and the residual rotator cuff to maintain normal shoulder kinematics and to prevent superior migration of the humeral head during abduction [7]. Increase in muscle strain of those muscles may be the reason for shoulder pain. Furthermore, increased forces required to maintain joint stability contribute to the anterior and posterior tear propagation, particularly if the remaining tendons are of poor quality.

A number of studies focused on preoperative clinical and radiological factors which would predict repairability of massive cuff tears [8–13]. Infraspinatus atrophy with external rotation lag sign in both abduction and adduction or anterior-superior subluxation of the humeral head with pseudoparalysis has been claimed to suggest an irreparable tear. Plain radiographic findings of a narrowed (7 mm or less) acromiohumeral distance also suggest that a tear may not be repairable. On MRI, fatty infiltration classified as Goutallier stage 3 or greater suggests that a cuff tear is unlikely to be repairable [8]. Sugihara et al. [13] noted that a tear size >4 cm, atrophy and degeneration of the supraspinatus muscle, and changes in signal intensity of the infraspinatus muscle would predict an irreparable tear. Holtby et al. [9] identified that the shape and size of the tear, the tissue quality of the rotator cuff, the preoperative American Shoulder and Elbow Surgeons (ASES) pain and function scores and the range of active external rotation were significantly correlated to repairability. Koh et al. [12] reported that the size of the tear, measured on coronal MRI scans, was the only factor that had an independent effect on complete footprint coverage. Kissenberth et al. [11] showed that patients with a positive tangent sign had an 82.3% post-test probability of having an irreparable tear, whereas probability in those with a negative tangent sign was only 1.6%. Moreover, tangent sign seems to be related to the chronicity of the lesion, since it has been reported to appear 4.5 years after the initial onset of shoulder symptoms [14]. Kim et al. [10] recently proposed a scoring system based on a retrospective review of the preoperative MRI and surgical records of 87 patients who underwent arthroscopic repair of a large to massive rotator cuff tear. It included the following variables: medio-lateral diameter greater than 4.2 cm, anteroposterior diameter greater than 3.7 cm, Warner's grade of muscle atrophy greater than 3, and Goutallier's grade of fatty infiltration greater than 3. The so-called repairability index showed that the degree of muscle atrophy and fatty infiltration are two to four times more likely to influence repairability than tear size. However, reliability and validity of the score have to be demonstrated. Currently, arthroscopy is the best and probably the only way to ascertain cuff repairability. Particularly, tendon reducibility is only appreciable when moving the tendons into the reduction position.

21.2.1 Scaffold Augmentation

Over the last decades, besides palliative options and functional repair, clinicians and researchers focused on biological strategies to improve repairability of massive contracted tears. The most recent biological augmentation procedures include the use of scaffolds. The goals of scaffold augmentation are structural stability, improved biochemical environment, and complete biocompatibility. Scaffolds reinforce and protect the repair warranting an increased failure load without increasing its stiffness. They were shown to bear 45% of the total load at the fixation site [15].

Various types of scaffolds have been popularized: autografts, allografts, synthetic, and xenografts. Each scaffold type presents specific limitations that influence their diffusion. Autografts require tissue harvesting, thus implying some potential donor site morbidity. Allografts arose concerns about the presence of residual DNA that could increase inflammatory response and degeneration. Synthetic scaffolds received increasing attention due to the good clinical outcomes [16] but showed limited ingrowth potential and a negative effect on cellular proliferation and differentiation of osteoblasts [17]. Moreover, some safety issues are related to possible acute inflammatory response and chronic inflammation due to foreign material reactions [17]. Xenografts are extracellular matrices derived from xenogenic material, decellularized to warrant immunogenicity but with enhanced molecule liberation. Porcine acellular dermis is the most used scaffold source; it showed repopulation and revascularization, minimal inflammatory host response and a propensity to remodel to a fascia-like architecture by 6 months. Integration was demonstrated at 24 months without macrophages and giant cell infiltration and without areas of calcification, fibrocartilage and ectopic bone and appearing similar to a mature tendon-to-bone insertion [18]. Xenografts also promote the expression of type I and III collagen in the tenocytes, molecules responsible for tendon strength, healing and fibrosis. Recent clinical studies showed very high healing rate without complications [19].

21.2.2 Superior Capsule Reconstruction

The superior capsule of the shoulder is a membranous layer beneath the rotator cuff that ranges from 4.4 to 9.1 mm at its attachment, which constitutes a substantial area, from 30% to 61% of the greater tuberosity [20]. It has been proven that the superior capsule plays an important role in the stability of the glenohumeral joint. Ishihara et al. [21] reported in their biomechanical study that a deficient superior capsule increased glenohumeral translation in all directions, particularly superior translation at 5° and 30° of abduction. In massive cuff tears, it has been postulated that the lack of both dynamic (tendons) and static (capsule) components of glenohumeral stabilization is responsible for superior humeral head migration and development of the rotator cuff tear arthropathy [22]. Mihata et al. [23] proposed the superior capsule reconstruction (SCR) to restore the static component. In a cadaver model of irreparable supraspinatus tear, SCR restored the superior stability by enabling the action of the remaining tendons and creating some “spacer” effect [24]. In another biomechanical study, Mihata et al. [25] showed that 8 mm graft fixed to the bones at 15 and 45° of shoulder abduction (corresponding to 10 or 30° of glenohumeral abduction) normalized the superior shoulder stability. Mihata et al. [26] also proved that acromioplasty could play a protective role for the reconstructed superior capsule in opposition to the risk related with the coracoacromial ligament resection in massive cuff tears.

SCR is performed arthroscopically with the patient placed in beach chair position. In the first study reported by Mihata et al. [23], arthroscopic SCR was performed using an autologous fascia lata folded twice or three times to achieve about

6–8 mm of graft thickness. The graft size was evaluated intraoperatively after partial rotator cuff repair (subscapularis tendon) and assessment of infraspinatus mobility (the sutures for repair could derive from the anchors used for graft fixation). Then, the graft was fixed medially to the glenoid superior tubercle (10–11 and 12–1 o'clock in a right shoulder) with a single row of suture anchors and laterally to the greater tuberosity using the double row combined with the suture-bridge technique (two anchors placed at the cartilage proximity and two laterally). Some other authors recommended graft fixation with up to six anchors in the greater tuberosity (three anchors medially and three laterally), depending on the size of the graft [27]. The final fixation was performed at 45° of shoulder abduction [23]. The graft was also sutured to the infraspinatus posteriorly and to the anterior supraspinatus or subscapularis anteriorly with side-to-side sutures in order to restore anterior and posterior force couples. Authors recommended very gentle suturing of the anterior supraspinatus and subscapularis as not to limit the range of motion. Other authors replaced the fascia lata graft with a human acellular dermal allograft.

Hirahara and Adams [28] were the first to report the use of an acellular dermal allograft in 2015. Burkhart et al. [29] also reported this modification and additionally proposed a manoeuvre to avoid problems with large graft introduction—a "zip-line" technique, which was recommended for dermal allografts >40 mm in any dimension. A third glenoid anchor, placed between the two above described, was inserted, and the sutures pulled through the Neviaser portal facilitated graft positioning into the joint followed by fixation. Burkhart et al. [29] recommended fixation of the graft with the remnants of the cuff in the posterior and the anterior area (Fig. 21.1). The anterior fixation to the cuff should be performed as long as anteriorly some supraspinatus or "comma" tissue was left intact. Direct fixation to the subscapularis tendon is somehow contraindicated.

Most authors recommend very slow rehabilitation after 4–6 weeks of immobilization [27–29]. It is related to the observation that, in a canine model, the acellular dermal allografts in the shoulder undergo significant remodelling and become weaker before they get stronger [30].

Promising clinical results of SCR were reported in 24 patients, who underwent arthroscopic SCR for irreparable rotator cuff tears at an average follow-up of 34.1 months (range, 24–51 months). Acromiohumeral distance increased from 4.6 mm preoperatively to 8.7 mm postoperatively, and mean active elevation increased significantly from 84 to 148°, and the ASES score improved from 23.5 to 92.9. MRI at follow-up revealed integrity of the graft and of the repaired tendons in 20 of 24 shoulders (83.3%). Additionally, no progression of osteoarthritis (OA) nor rotator cuff muscle atrophy was reported. Three patients (12.5%) with severe fatty degeneration of the infraspinatus tendon had retears of the repaired infraspinatus tendon 3 months after surgery [23]. Advantages and disadvantages of SCR are summarized in Table 21.1.

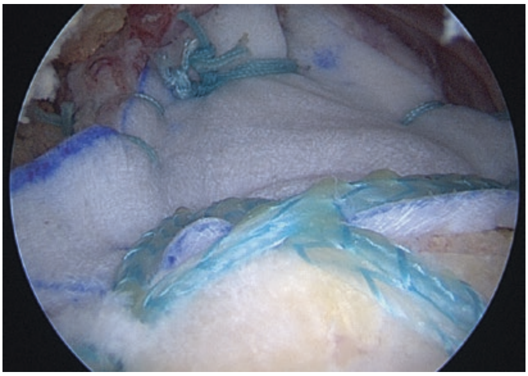


Fig. 21.1 Superior capsule reconstruction. The graft is secured to the postero-superior and antero-superior cuff with side-to-side sutures

Table 21.1 SCR: pros and cons

SCR advantages	SCR disadvantages
Arthroscopic minimally invasive technique, with very low infection rate	Technically demanding procedure
Restores stable fulcrum	Restores stability and mobility of the shoulder rather than strength
Non-"burning bridges" technique does not preclude future tendon transfer or reverse shoulder arthroplasty	Large number of implants is necessary (however the costs are still lower than reverse shoulder arthroplasty)
Lower morbidity comparing to reverse shoulder arthroplasty	Some immobilization due to the prolonged graft healing is necessary

21.2.3 Biodegradable Subacromial Spacer

The deployment of a real spacer in the subacromial space has also been described as a viable option to improve shoulder function by lowering the humeral head, reducing the forces required to achieve stable abduction, and facilitating humeral gliding against the acromion, thus reducing subacromial friction during shoulder abduction [31, 32] (Fig. 21.2).

Standard shoulder arthroscopy in a beach chair or lateral decubitus position is performed. After debridement and bursectomy, the rotator cuff is assessed for repairability. Tendon mobility to the footprint region and quality of the tendon are assessed. Once deemed irreparable, the correct size of the subacromial spacer is selected by measuring the distance from the lateral border of the acromion to approximately 1 cm medial to the glenoid apex. If LHB is still present, biceps tenotomy is advised. The rolled-up spacer is then inserted through the lateral portal and inflated with saline. Once optimized by determined passive full range of movements, the appropriate inflation volume is left in situ by sealing the device.

The spacer begins to degrade approximately 2–3 months post-implantation and fully disintegrates within 12 months, while pain and functional scores continue to improve beyond the period of spacer disintegration (Fig. 21.3). Contraindications for this treatment option include glenohumeral OA, allergies to the device materials and active infection [32].

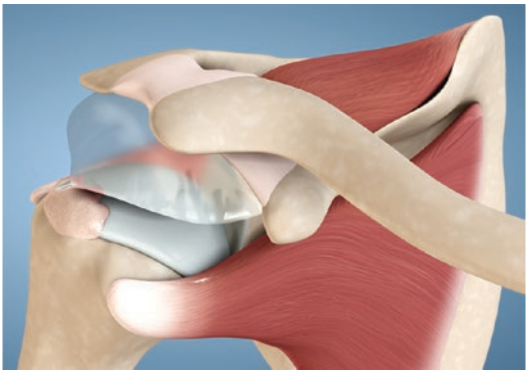


Fig. 21.2 An illustration of biodegradable subacromial spacer (InSpace™) inserted in the subacromial space (with permission of OrthoSpace)

It must be highlighted that subacromial spacer cannot restore the force couples in the transverse plane; thus subscapularis tendon and teres minor should be intact or at least repairable before considering this option.

Some clinical studies showed that outcomes of subacromial spacer are comparable to those obtained with arthroscopic debridement and subacromial decompression alone. Subacromial spacer provided significant improvement in pain, range of motion (ROM) and ability to perform activities of daily living, albeit no improvement in abduction strength was observed. Interestingly, results for subacromial spacer improved over time, whereas subacromial decompression and acromioplasty alone showed clinical worsening over time, while progression of OA was not delayed [33, 34].

Senekovic et al. reported on 24 patients that underwent subacromial spacer treatment. At 5-year follow-up, 84.6% showed significant improvement from baseline [32]. It was also demonstrated that, in contrast with other treatment options, the results of subacromial spacer were not influenced by biceps tenotomy [32].

Potential complications include foreign body response, local irritation or inflammation, tissue necrosis and displacement of the device displacement. However, no complications or unexpected device-related adverse events were recorded in clinical experiences [31].

21.2.4 Tendon Transfers

Different tendon transfers can be considered as proper options for treating massive retracted rotator cuff tears. The most commonly described procedure is the latissimus dorsi transfer (LDT) with or without teres major transfer (LDTMT). While no upper age limit has yet been reported, a recent systematic review noted a mean age of 59 years. However, this indication can be extended to elderly patients with even Hamada stage 3 OA. Previous papers emphasized the importance of the integrity of the deltoid and subscapularis for a good outcome, as forward elevation and shoulder stability drastically decrease with subscapularis insufficiency. Atrophy and fatty

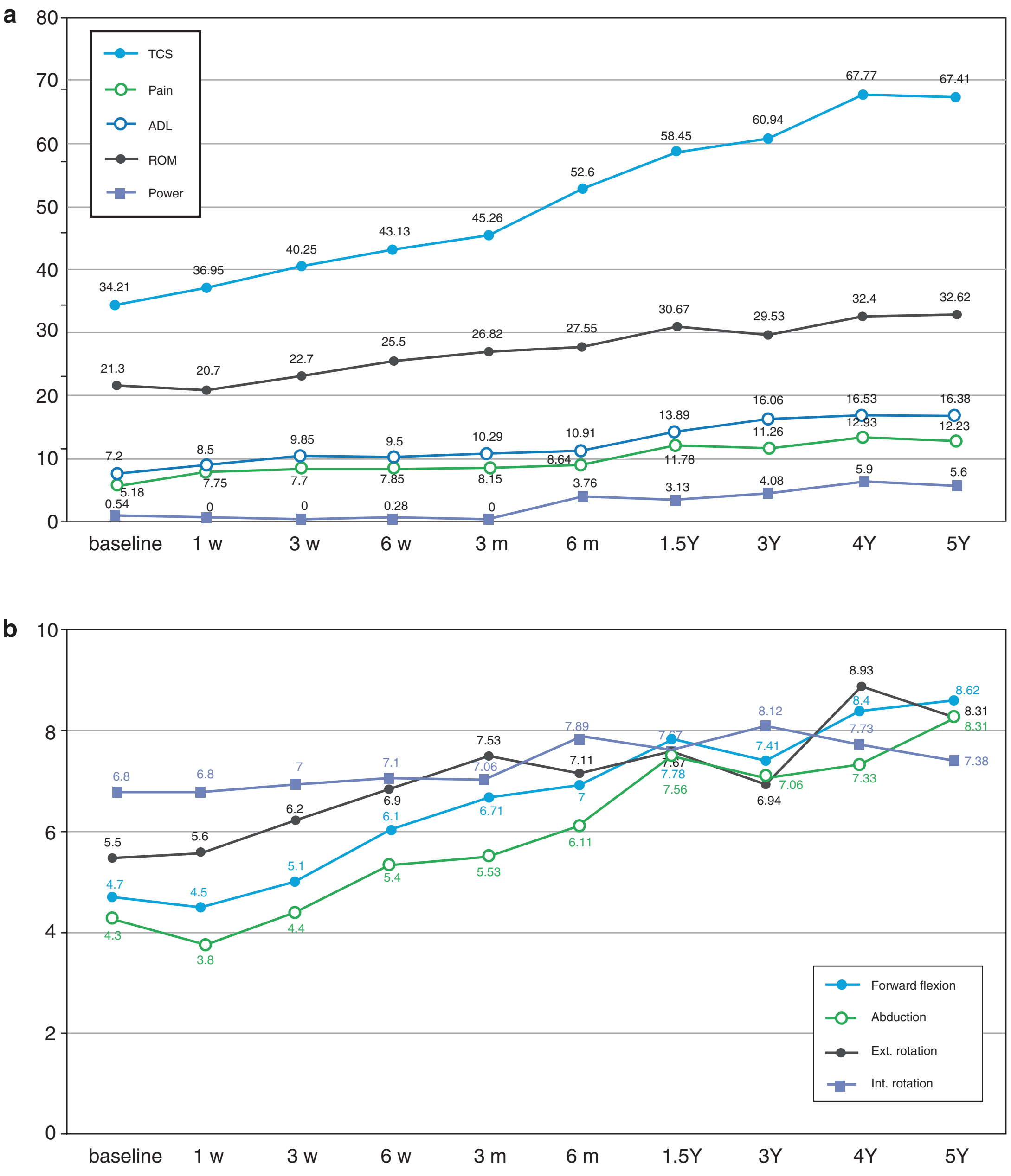


Fig. 21.3 (**a**) Clinical results following biodegradable spacer insertion presenting total Constant score and its subscores: pain, activity of daily living (ADL), range of motion (ROM) and power of abduction. Change in mean scores from baseline to 5 years postoperatively is presented. (**b**) Presentation of ROM score variables over a 5-year period of time. The values are presented as means and represent two points per every 30° of active, pain-free ROM (0 = worst, 10 = best)

infiltration (Goutallier grade 3 or higher) of the teres minor were also reported by several authors as associated with worse postoperative outcomes and with poor recovery of active external rotation (ER). Good preoperative ROM is also essential, specifically passive forward flexion (FF) and abduction over 80–90°. A pseudoparalytic shoulder has been associated with poorer outcomes.

Other transfers for irreparable postero-superior rotator cuff tears include isolated teres major transfer (TMT) and the recently described lower trapezius transfer (LTT). TMT was designed as a transfer for infraspinatus deficiency [35]. The authors recommended patients under the age of 55 with intact subscapularis and anterior supraspinatus cable.

Antero-superior rotator cuff tears or subscapularis tears are less common, but the subscapularis is essential to the proper function of the shoulder. If subscapularis tear increases to involve the supraspinatus, the proximal humerus may escape antero-superiorly with active elevation of the shoulder. Several options to treat irreparable antero-superior RCTs have been studied, including pectoralis major transfer (PMT), pectoralis minor transfer (PmT) and LDT with or without teres major transfer (LDTMT).

21.2.5 Tendon Transfers for Postero-superior Irreparable Cuff Tears

21.2.5.1 Latissimus Dorsi Transfer

The earliest and most studied transfer is the LDT, originally described in Gerber's landmark paper [36], which showed how LDT can be used in massive cuff tears using a double-incision technique. The transfer is fixed anteriorly to the subscapularis tendon and laterally to the greater tuberosity by transosseous sutures, and the latissimus dorsi (LD) is changed into an abductor and external rotator of the humerus. Different techniques were also developed with time. Habermeyer [37] described a single-incision approach with a more posterior attachment of the transfer on the humeral head. Moursy [38] reported a modification of the technique by using small bone chips rather than simple tenotomy during LD harvesting. Kany [39] reported an arthroscopically assisted approach that provided better mechanical resistance to traction due to the tubularized tendon and its fixation into a bone tunnel. The published results were comparable with the Gerber two-incision method [40] (Fig. 21.4).

21.2.5.2 Teres Major Transfer

This technique is performed using a two-incision technique similar to the LDT, but if the supraspinatus is torn, it can be combined with a trapezius transfer. Isolated infraspinatus involvement and a functional teres minor showed better outcomes.

21.2.5.3 Lower Trapezius Transfer

The two-incision technique of LTT prolonged with tendon autograft (hamstrings) or allograft (Achilles tendon) has been recently published as an alternative option to the LDT for the irreparable posterior rotator cuff tear and in cases of chronic isolated musculotendinous tear of the infraspinatus [41]. The line of pull of its muscle fibres replicates more closely that of the infraspinatus (Fig. 21.5). Furthermore, the tension and excursion forces of the trapezius are quite similar to the infraspinatus. In a cadaveric study, Omid et al. [42] concluded that the LTT was superior to LDT, and Hartzler et al. [43] also found improved ER with the arm at the side compared with the LDT, but LDT is a more appropriate technique to restore forward elevation or ER at 90° of abduction. In spite of these theoretical advantages, LTT is limited by the fact that a graft must be used to improve its excursion so that the problems related to healing (tendon necrosis) must be taken into account.

21.2.6 Tendon Transfers for Antero-superior Irreparable Cuff Tears

21.2.6.1 Pectoralis Major Transfer

Wirth and Rockwood [44] originally described and published the PMT (anterior to the conjoined tendon) in 1997. Resch et al. [45] modified this technique to a transfer involving only the superior two-thirds of the tendon under the conjoined tendon, until it was further developed to a transfer involving the entire pectoralis major tendon (Fig. 21.6).

Subcoracoid placement of the graft appears much better from a biomechanical standpoint, as traction lines of subscapularis might be better restored. Unfortunately, poor outcomes are

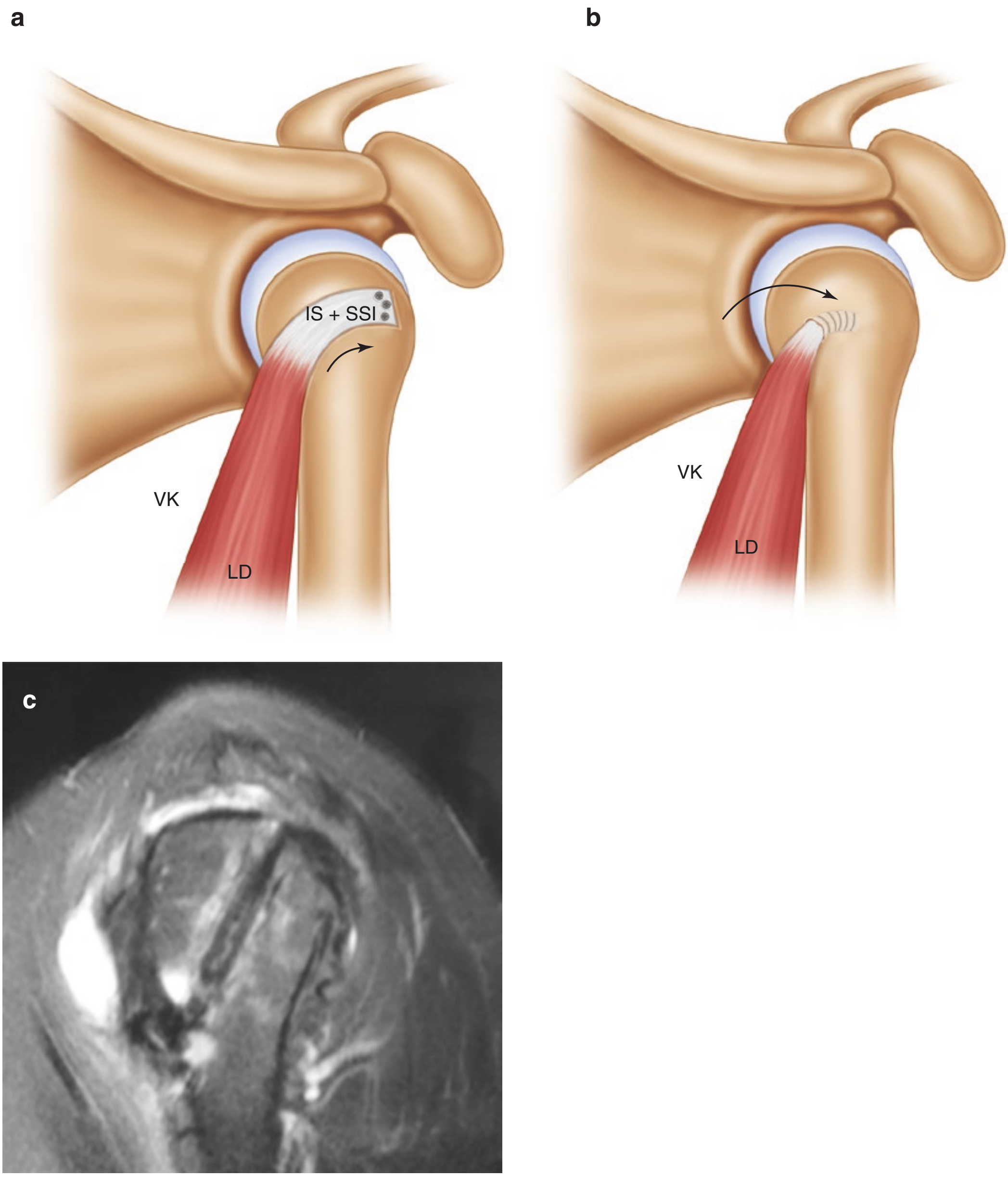


Fig. 21.4 Latissimus dorsi transfer for postero-superior cuff tear. (**a**) Graft fixation with suture anchors. (**b**, **c**) Graft fixation within a bone tunnel

reported in the literature regardless of the muscle part used (either clavicular or sternocostal) or graft placement (either above or below the conjoined tendon). This is probably due to the fact that the force vectors of the pectoralis major muscle are completely different from those of the subscapularis, and this is not in compliance with the principles of tendon transfers.

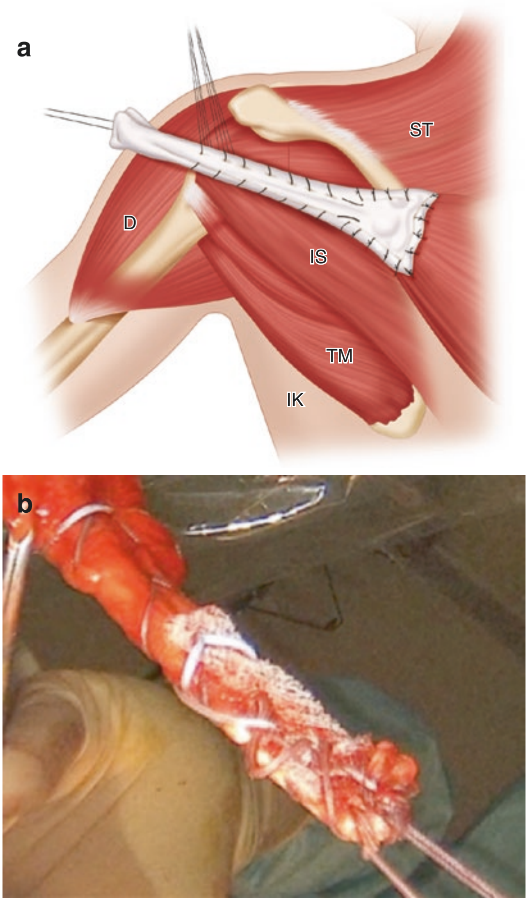


Fig. 21.5 (**a**, **b**) Lower trapezius transfer for posterosuperior cuff tear

21.2.6.2 Pectoralis Minor Transfer

If supracoracoid PmT is performed, it can leave patients with positive belly-press and lift-off test [35]. Wirth et al. [44] first described PmT in 1997, but Paladini et al. [46] used a subcoracoid PmT with a small cortical piece of the coracoid for the combined superior two-thirds subscapularis and irreparable supraspinatus tears. Valenti et al. recently described an arthroscopically assisted PmT technique (*unpublished data*). This technique is associated with less ER loss and positive belly-press test postoperatively, with adequate restoration of strength in IR.

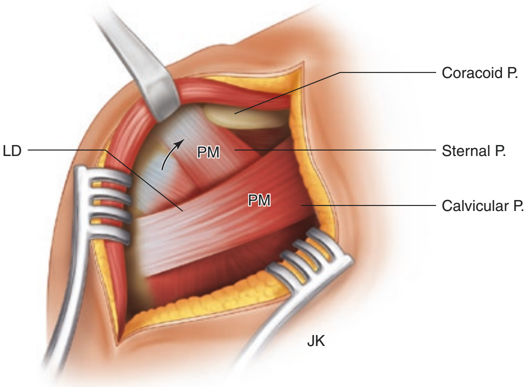


Fig. 21.6 Pectoralis major transfer for subscapularis deficiency

21.2.6.3 Latissimus Dorsi Transfer

Elhassan et al. [47] biomechanically investigated the potential of LDT, TMT, and LDTMT to the subscapularis footprint for anterior cuff deficiencies. A recent anatomical cadaveric study proved that both isolated and simultaneous latissimus dorsi and teres major transfers were feasible and safe to reconstruct subscapularis tear, as the force vector of the LD is anatomically similar to that of the subscapularis muscle. This technique can be used in patients with an anteriorly subluxated humeral head by transferring the latissimus dorsi proximally and/or the teres major distally on the subscapularis insertion simultaneously or alone.

Recently, the arthroscopically assisted LDT was also described in the literature by Kany et al. [48] for cases of irreparable subscapularis (±supraspinatus) tears without any significant anterosuperior static instability of the humeral head. This technique takes advantage of the tubularized tendon with a strong fixation and of tension adjustment of the muscle belly, which are supposed to act as dynamic restraints against the superior escape of the humeral head. This technique showed promising short-term results with complete recovery of the previously positive clinical signs (belly press) at 1 year or later after surgery.

21.2.7 Reverse Total Shoulder Arthroplasty

Improved imaging of massive rotator cuff tears allows detecting eccentric glenohumeral OA with progressive erosion of the upper part of the glenoid. Reverse prostheses were designed for pseudoparalytic massive rotator cuff tears, with or without associated eccentric glenohumeral OA [22, 49–52]. Reverse total shoulder arthroplasty (RTSA) was initially indicated in patients older than 70–75 years, in light of the limited survivorship beyond 10 years. Nowadays, the indications are extended to younger patients in whom alternative therapeutic options cannot be employed. Indication to RTSA can be limited by insufficient glenoid bone stock for the implantation of the glenosphere and by impaired deltoid function [53–55], albeit muscular transfers to counter any insufficiency of the deltoid muscle have been suggested [56]. Combining a LDT with RTSA allows recovery of external rotation in cases of complete external rotation deficit related to a combined deficiency of the infraspinatus and teres minor muscles [56, 57].

Two surgical approaches are possible: the delto-pectoral route and the MacKenzie antero-superior approach. The delto-pectoral route allows for a wide approach to the humeral shaft for stem arthroplasty and repairs for bone loss in the anterior part of the glenoid. However, it exposes the anterior fibres of the deltoid muscle during the preparation of the glenoid, which requires posterior and inferior dislocation of the humerus. This manoeuvre places tension on the neurovascular structures. Furthermore, the approach requires sectioning of the subscapularis muscle often a partial tenotomy of the pectoralis major muscle.

The MacKenzie antero-superior approach allows for simple exposure of the glenoid without the need to luxate the humerus downwards and so without exerting significant traction on the neurovascular structures and especially on the axillary nerve. It also respects the lower part of the humeral insertion of the subscapularis muscle. The 135° cut provides easier access to the lower part of the glenoid, making it possible to place the prosthetic glenoid with an inferior tilt, and hence more easily a press-fit large glenosphere. Correction for central and posterior bone loss is facilitated by the antero-superior approach. However, this approach passes through the fibres of the deltoid, which creates a risk of postoperative muscle weakness and requires precise muscle repair by trans-acromial bone reinsertion.

The preparation of the humerus begins with a humeral cut guided by a centromedullary cutting guide, which ensures the angulation of the humeral rim and the retroversion of the humeral implant. This cut is more or less largely depending on the design of the implant and the desired lowering of the humeral epiphysis. The preparation of the humeral diaphysis begins with reamers, which can serve as a support to guide the cutting of the head. This preparation is simple and is further simplified in case of stemless models. This is followed by preparation of the glenoid, made possible by resection of the humeral head. This preparation is sometimes difficult due to deformations of the glenoid and possible peripheral osteophytes. CT scans and planning software allow for 3D reconstruction of the glenoid and the scapula, making it possible to precisely measure the ideal position of the metaglenoid [58–62]. For very large deformations of the glenoid with great loss of bone substance, the planning software helps calculate the volume and shape of the bone graft or design custom implants to compensate for such bone loss [63, 64]. The metaglenoid position should be as low as possible, with a minimal inferior tilt, so as not to diminish the strength of the lower pillar of the scapula, and no superior tilt, which would weaken the bone fixation of the glenoid implants. Fixation of the glenosphere can be ensured by different methods depending on the different types of prostheses [65].

The next step is to lower the humerus, implanting the humeral stem into the diaphysis by either press fit or cementing, which limits the possible insertion of the humeral prosthesis into the diaphysis and commensurately reduces the lowering of the humerus. The introduction of a test of polyethylene component makes it possible to assess the lowering and to evaluate the stability. Tensioning the conjoint tendon by hand is the best way to assess the elongation and stability of the prosthesis, given the extent of muscle relaxation of the deltoid muscle under anaesthesia. Polyethylene in various height options and elevators make it possible to improve humeral prosthesis lowering and stability.

Reinsertion of the muscles is favourable but is not always possible. The tendons of the teres minor muscle and of the lower part of the infraspinatus should be reattached if possible. The subscapularis muscle must also be attached to the front. This gesture is often made difficult by the lateralization and the lowering of the humeral epiphysis and the lesser tuberosity, on which it is normally inserted. It is not always possible to attach the subscapularis tendon, even after extensive muscle release.

Postoperative immobilization for simple prostheses makes use of an abduction pillow with early passive- and active-assisted rehabilitation.

21.3 Future Treatment Options

Clear repairability criteria are of utmost importance in order to define both clinical and radiological variables that can predict a successful rotator cuff repair and subsequently improve the surgical decision-making process. In case of an irreparable retracted cuff tear, maximizing biological strategies by adding autogenous or synthetized molecules to the repair site is surely the next step. Platelet-rich plasma was the first product studied, but no benefit was noted, and its application is therefore not recommended. Growth factors (BMP-2, BMP-7, bFGF) and stem cells from the bone marrow or adipose tissue are providing compelling in vitro and in vivo evidences in animal models, with a promising potential to enhance rotator cuff repairs [66]. However, well-designed clinical studies comparing the results in different existing treatment options are still lacking.

Properties of biodegradable spacers still need to be elucidated. It is uncertain how long the spacer remains inflated, albeit the current results demonstrate that the positive effect continues beyond the time of spacer degradation period [32]. Although the reason for this remains unclear, limited animal studies may support the hypothesis that an inflammatory response around the implant may provide a (fibrotic) barrier between the humeral head and the acromion to reduce pain.

Fig. 21.7 Short-stem reverse total shoulder arthroplasty

Although tendon transfers seem to provide promising clinical results, anatomical healing of transferred tendons on MRI is currently difficult to analyse.

Reverse prostheses based on the concepts proposed by Grammont have resulted in spectacular improvement in the management of massive irreparable rotator cuff tears, cuff tear arthropathy and eccentric glenohumeral OA. Nonetheless, irreparable rotator cuff tear is the condition associated with the lowest complication rate of RTSA, whereas revision surgery is associated with the highest complication rate. In order to reduce problems related to peri-prosthetic fractures, short-stem or stemless prostheses have been designed (Fig. 21.7). The initial results are satisfactory and do not show any complications specific to the design of the humeral component [67–70]. These results are promising and encourage an extension of the indications for reverse prostheses, particularly to younger patients in situations where all other therapeutic possibilities have failed.

21.4 Take-Home Message

Treatment of irreparable massive rotator cuff tears should be based on patient factors and associated pathology. The decision-making process includes surgeon personal experience as well as scientific evidence. A thorough knowledge of existing treatment options and indications is crucial to achieve the best outcome. Irreparable rotator cuff tears can be debilitating, and failed cuff repairs are still a surgical challenge. When possible, biological augmentation by using a scaffold can mechanically reinforce a rotator cuff repair, ultimately providing a better tissue healing. Otherwise, arthroscopic SCR should be taken into consideration. It is a minimally invasive procedure that can be converted into any other treatment if the results are not satisfactory (tendon transfers or RTSA). However, the procedure is relatively new, and more clinical studies with longer follow-up are required.

Alternatively, patients with predominant symptoms of pain but relatively preserved active elevation are probably ideal candidates for a biodegradable subacromial spacer. Good clinical results can be expected in patients with preserved force couple in the transverse plane in absence of shoulder OA. The spacer in the subacromial space will only influence the force couple in the coronal plane by lowering the humeral head, facilitating humeral gliding against the acromion and reducing subacromial friction during shoulder abduction. The procedure is simple, safe, reliable and minimally invasive with a lower complication rate compared to other treatment options.

Proposed benefits of tendon transfers are pain relief and some increased ROM with a potential increase in strength to improve quality of life and shoulder function. Different techniques have been developed. For postero-superior rotator cuff tears, the LDT remains the most commonly used method. However, LTT transfer is promising and should be considered to restore ER. Isolated subscapularis deficiencies seem to be well managed with PMT and may potentially be even better with the recently reported LDT.

Reverse prostheses radically changed the course of treatment for massive and retracted rotator cuff tears, offering a reliable and reproducible solution. Nevertheless, RTSA requires adequate glenoid bone stock and must only be used as a last resort in the treatment of massive and retracted rotator cuff tears.

References

1. Godenèche A, Freychet B, Lanzetti RM, Clechet J, Carrillon Y, Saffarini M. Should massive rotator cuff tears be reconstructed even when only partially repairable? Knee Surg Sports Traumatol Arthrosc. 2016;25(7):2164–73.
2. Nho SJ, Delos D, Yadav H, Pensak M, Romeo AA, Warren RF, MacGillivray JD. Biomechanical and biologic augmentation for the treatment of massive rotator cuff tears. Am J Sports Med. 2010;38:619–29.
3. Moore DR, Cain EL, Schwartz ML, Clancy WG. Allograft reconstruction for massive, irreparable rotator cuff tears. Am J Sports Med. 2006;34:392–6.
4. DeOrio JK, Cofield RH. Results of a second attempt at surgical repair of a failed initial rotator-cuff repair. J Bone Joint Surg Am. 1984;66:563–7.
5. Gerber C, Fuchs B, Hodler J. The results of repair of massive tears of the rotator cuff. J Bone Joint Surg Am. 2000;82:505–15.
6. Henry P, Wasserstein D, Park S, Dwyer T, Chahal J, Slobogean G, Schemitsch E. Arthroscopic repair for chronic massive rotator cuff tears: a systematic review. Arthroscopy. 2015;31:2472–80.
7. Parsons IM, Apreleva M, FH F, Woo SLY. The effect of rotator cuff tears on reaction forces at the glenohumeral joint. J Orthop Res. 2002;20:439–46.
8. Bedi A, Dines J, Warren RF, Dines DM. Massive tears of the rotator cuff. J Bone Joint Surg Am. 2010;92:1894–908.
9. Holtby R, Razmjou H. Relationship between clinical and surgical findings and reparability of large and massive rotator cuff tears: a longitudinal study. BMC Musculoskelet Disord. 2014;15:180.
10. Kim S-J, Park J-S, Lee K-H, Lee B-G. The development of a quantitative scoring system to predict whether a large-to-massive rotator cuff tear can be arthroscopically repaired. Bone Jt J. 2016;98–B:1656–61.
11. Kissenberth MJ, Rulewicz GJ, Hamilton SC, Bruch HE, Hawkins RJ. A positive tangent sign predicts the repairability of rotator cuff tears. J Shoulder Elb Surg. 2014;23:1023–7.
12. Koh KH, Lim TK, Park YE, Lee SW, Park WH, Yoo JC. Preoperative factors affecting footprint coverage in rotator cuff repair. Am J Sports Med. 2014;42:869–76.
13. Sugihara T, Nakagawa T, Tsuchiya M, Ishizuki M. Prediction of primary reparability of massive tears of the rotator cuff on preoperative magnetic resonance imaging. J Shoulder Elb Surg. 2003;12:222–5.

14. Melis B, DeFranco MJ, Chuinard C, Walch G. Natural history of fatty infiltration and atrophy of the supraspinatus muscle in rotator cuff tears. Clin Orthop Relat Res. 2010;468:1498–505.
15. Aurora A, McCarron JA, van den Bogert AJ, Gatica JE, Iannotti JP, Derwin KA. The biomechanical role of scaffolds in augmented rotator cuff tendon repairs. J Shoulder Elb Surg. 2012;21:1064–71.
16. Proctor CS. Long-term successful arthroscopic repair of large and massive rotator cuff tears with a functional and degradable reinforcement device. J Shoulder Elb Surg. 2014;23:1508–13.
17. Mikos AG, McIntire LV, Anderson JM, Babensee JE. Host response to tissue engineered devices. Adv Drug Deliv Rev. 1998;33:111–39.
18. Nicholson GP, Breur GJ, Van Sickle D, Yao JQ, Kim J, Blanchard CR. Evaluation of a cross-linked acellular porcine dermal patch for rotator cuff repair augmentation in an ovine model. J Shoulder Elb Surg. 2007;16:S184–90.
19. Barber FA, Burns JP, Deutsch A, Labbé MR, Litchfield RB. A prospective, randomized evaluation of acellular human dermal matrix augmentation for arthroscopic rotator cuff repair. Arthroscopy. 2012;28:8–15.
20. Nimura A, Kato A, Yamaguchi K, Mochizuki T, Okawa A, Sugaya H, Akita K. The superior capsule of the shoulder joint complements the insertion of the rotator cuff. J Shoulder Elb Surg. 2012;21:867–72.
21. Ishihara Y, Mihata T, Tamboli M, Nguyen L, Park KJ, McGarry MH, Takai S, Lee TQ. Role of the superior shoulder capsule in passive stability of the glenohumeral joint. J Shoulder Elb Surg. 2014;23:642–8.
22. Neer CS, Craig EV, Fukuda H. Cuff-tear arthropathy. J Bone Joint Surg Am. 1983;65:1232–44.
23. Mihata T, Lee TQ, Watanabe C, Fukunishi K, Ohue M, Tsujimura T, Kinoshita M. Clinical results of arthroscopic superior capsule reconstruction for irreparable rotator cuff tears. Arthroscopy. 2013;29:459–70.
24. Mihata T, McGarry MH, Pirolo JM, Kinoshita M, Lee TQ. Superior capsule reconstruction to restore superior stability in irreparable rotator cuff tears: a biomechanical cadaveric study. Am J Sports Med. 2012;40:2248–55.
25. Mihata T, McGarry MH, Kahn T, Goldberg I, Neo M, Lee TQ. Biomechanical effect of thickness and tension of fascia lata graft on glenohumeral stability for superior capsule reconstruction in irreparable supraspinatus tears. Arthroscopy. 2016;32:418–26.
26. Mihata T, McGarry MH, Kahn T, Goldberg I, Neo M, Lee TQ. Biomechanical effects of acromioplasty on superior capsule reconstruction for irreparable supraspinatus tendon tears. Am J Sports Med. 2016;44:191–7.
27. Petri M, Greenspoon JA, Millett PJ. Arthroscopic superior capsule reconstruction for irreparable rotator cuff tears. Arthrosc Tech. 2015;4:e751–5.
28. Hirahara AM, Adams CR. Arthroscopic superior capsular reconstruction for treatment of massive irreparable rotator cuff tears. Arthrosc Tech. 2015;4:e637–41.
29. Burkhart SS, Denard PJ, Adams CR, Brady PC, Hartzler RU. Arthroscopic superior capsular reconstruction for massive irreparable rotator cuff repair. Arthrosc Tech. 2016;5:e1407–18.
30. Adams JE, Zobitz ME, Reach JS, An K-N, Steinmann SP. Rotator cuff repair using an acellular dermal matrix graft: an in vivo study in a canine model. Arthroscopy. 2006;22:700–9.
31. Senekovic V, Poberaj B, Kovacic L, Mikek M, Adar E, Dekel A. Prospective clinical study of a novel biodegradable sub-acromial spacer in treatment of massive irreparable rotator cuff tears. Eur J Orthop Surg Traumatol. 2013;23:311–6.
32. Senekovic V, Poberaj B, Kovacic L, Mikek M, Adar E, Markovitz E, Maman E, Dekel A. The biodegradable spacer as a novel treatment modality for massive rotator cuff tears: a prospective study with 5-year follow-up. Arch Orthop Trauma Surg. 2017;137:95–103.
33. Anley CM, Chan SK, Snow M. Arthroscopic treatment options for irreparable rotator cuff tears of the shoulder. World J Orthop. 2014;5:557–65.
34. Greenspoon JA, Petri M, Warth RJ, Millett PJ. Massive rotator cuff tears: pathomechanics, current treatment options, and clinical outcomes. J Shoulder Elb Surg. 2015;24:1493–505.
35. Valenti P. Tendon transfer for irreparable cuff tear. Paris: Springer-Verlag; 2011.
36. Gerber C, Vinh TS, Hertel R, Hess CW. Latissimus dorsi transfer for the treatment of massive tears of the rotator cuff. A preliminary report. Clin Orthop Relat Res. 1988;232:51–61.
37. Habermeyer P, Magosch P, Rudolph T, Lichtenberg S, Liem D. Transfer of the tendon of latissimus dorsi for the treatment of massive tears of the rotator cuff: a new single-incision technique. J Bone Joint Surg (Br). 2006;88:208–12.
38. Moursy M, Forstner R, Koller H, Resch H, Tauber M. Latissimus dorsi tendon transfer for irreparable rotator cuff tears: a modified technique to improve tendon transfer integrity. J Bone Joint Surg Am. 2009;91:1924–31.
39. Kany J, Kumar HA, Chang VK, Grimberg J, Garret J, Valenti P. Mini invasive axillary approach and arthroscopic humeral head interference screw fixation for latissimus dorsi transfer in massive and irreparable posterosuperior rotator cuff tears. Tech Shoulder Elb Surg. 2010;11:8–14.
40. Grimberg J, Kany J, Valenti P, Amaravathi R, Ramalingam AT. Arthroscopic-assisted latissimus dorsi tendon transfer for irreparable posterosuperior cuff tears. Arthroscopy. 2015;31:599–607.e1.
41. Elhassan BT, Wagner ER, Werthel J-D. Outcome of lower trapezius transfer to reconstruct massive irreparable posterior-superior rotator cuff tear. J Shoulder Elb Surg. 2016;25:1346–53.
42. Omid R, Heckmann N, Wang L, McGarry MH, Vangsness CT, Lee TQ. Biomechanical comparison between the trapezius transfer and latissimus transfer for irreparable posterosuperior rotator cuff tears. J Shoulder Elb Surg. 2015;24:1635–43.

43. Hartzler RU, Barlow JD, An K-N, Elhassan BT. Biomechanical effectiveness of different types of tendon transfers to the shoulder for external rotation. J Shoulder Elb Surg. 2012;21:1370–6.
44. Wirth MA, Rockwood CA Jr. Operative treatment of irreparable rupture of the subscapularis. J Bone Joint Surg Am. 1997;79:722–31.
45. Resch H, Povacz P, Ritter E, Matschi W. Transfer of the pectoralis major muscle for the treatment of irreparable rupture of the subscapularis tendon. J Bone Joint Surg Am. 2000;82:372–82.
46. Paladini P, Campi F, Merolla G, Pellegrini A, Porcellini G. Pectoralis minor tendon transfer for irreparable anterosuperior cuff tears. J Shoulder Elb Surg. 2013;22:e1–5.
47. Elhassan B, Christensen TJ, Wagner ER. Feasibility of latissimus and teres major transfer to reconstruct irreparable subscapularis tendon tear: an anatomic study. J Shoulder Elb Surg. 2014;23:492–9.
48. Kany J, Guinand R, Croutzet P, Valenti P, Werthel JD, Grimberg J. Arthroscopic-assisted latissimus dorsi transfer for subscapularis deficiency. Eur J Orthop Surg Traumatol. 2016;26:329–34.
49. Drake GN, O'Connor DP, Edwards TB. Indications for reverse total shoulder arthroplasty in rotator cuff disease. Clin Orthop Relat Res. 2010;468:1526–33.
50. Hartzler RU, Steen BM, Hussey MM, Cusick MC, Cottrell BJ, Clark RE, Frankle MA. Reverse shoulder arthroplasty for massive rotator cuff tear: risk factors for poor functional improvement. J Shoulder Elb Surg. 2015;24:1698–706.
51. Laurence M. Replacement arthroplasty of the rotator cuff deficient shoulder. J Bone Joint Surg (Br). 1991;73:916–9.
52. Neri BR, Chan KW, Kwon YW. Management of massive and irreparable rotator cuff tears. J Shoulder Elb Surg. 2009;18:808–18.
53. Formaini NT, Everding NG, Levy JC, Santoni BG, Nayak AN, Wilson C, Cabezas AF. The effect of glenoid bone loss on reverse shoulder arthroplasty baseplate fixation. J Shoulder Elb Surg. 2015;24:e312–9.
54. Jones RB, Wright TW, Zuckerman JD. Reverse total shoulder arthroplasty with structural bone grafting of large glenoid defects. J Shoulder Elb Surg. 2016;25:1425–32.
55. Marinello PG, Amini MH, Peers S, O'Donnell J, Iannotti JP. Reverse total shoulder arthroplasty with combined deltoid reconstruction in patients with anterior and/or middle deltoid tears. J Shoulder Elb Surg. 2016;25:936–41.
56. Shi LL, Cahill KE, Ek ET, Tompson JD, Higgins LD, Warner JJP. Latissimus dorsi and teres major transfer with reverse shoulder arthroplasty restores active motion and reduces pain for posterosuperior cuff dysfunction. Clin Orthop Relat Res. 2015;473:3212–7.
57. Puskas GJ, Germann M, Catanzaro S, Gerber C. Secondary latissimus dorsi transfer after failed reverse total shoulder arthroplasty. J Shoulder Elb Surg. 2015;24:e337–44.
58. Dallalana RJ, McMahon RA, East B, Geraghty L. Accuracy of patient-specific instrumentation in anatomic and reverse total shoulder arthroplasty. Int J Shoulder Surg. 2016;10:59–66.
59. Heylen S, Van Haver A, Vuylsteke K, Declercq G, Verborgt O. Patient-specific instrument guidance of glenoid component implantation reduces inclination variability in total and reverse shoulder arthroplasty. J Shoulder Elb Surg. 2016;25:186–92.
60. Stephens BF, Hebert CT, Azar FM, Mihalko WM, Throckmorton TW. Optimal baseplate rotational alignment for locking-screw fixation in reverse total shoulder arthroplasty: a three-dimensional computer-aided design study. J Shoulder Elb Surg. 2015;24:1367–71.
61. Van Haver A, Heylen S, Vuylsteke K, Declercq G, Verborgt O. Reliability analysis of glenoid component inclination measurements on postoperative radiographs and computed tomography-based 3D models in total and reversed shoulder arthroplasty patients. J Shoulder Elb Surg. 2016;25:632–40.
62. Wylie JD, Tashjian RZ. Planning software and patient-specific instruments in shoulder arthroplasty. Curr Rev. Musculoskelet Med. 2016;9:1–9.
63. Holcomb JO, Cuff D, Petersen SA, Pupello DR, Frankle MA. Revision reverse shoulder arthroplasty for glenoid baseplate failure after primary reverse shoulder arthroplasty. J Shoulder Elb Surg. 2009;18:717–23.
64. Ivaldo N, Mangano T, Caione G, Rossoni M, Ligas A. Customized tantalum-augmented reverse shoulder arthroplasty for glenoid bone defect and excessive medialization: description of the technique. Musculoskelet Surg. 2016;100:13–8.
65. Leuridan S, Goossens Q, Pastrav L, Van Tongel A, De Wilde L, Debeer P, Denis K, Desmet W, Vander Sloten J. A nondestructive method to verify the glenosphere-baseplate assembly in reverse shoulder arthroplasty. J Shoulder Elb Surg. 2016;25:e156–65.
66. Lorbach O, Baums MH, Kostuj T, Pauly S, Scheibel M, Carr A, Zargar N, Saccomanno MF, Milano G. Advances in biology and mechanics of rotator cuff repair. Knee Surg Sports Traumatol Arthrosc. 2015;23:530–41.
67. Ang C-Y, Lai K-W, Tjiauw Tjoen DL, Chang Chee Cheng P. Reverse shoulder arthroplasty: the Singapore General Hospital experience and a simple method of measuring change in the center-of-rotation. J Orthop. 2015;12:97–101.
68. Fink Barnes LA, Grantham WJ, Meadows MC, Bigliani LU, Levine WN, Ahmad CS. Sports activity after reverse total shoulder arthroplasty with minimum 2-year follow-up. Am J Orthop. 2015;44:68–72.
69. Frankle M, Siegal S, Pupello D, Saleem A, Mighell M, Vasey M. The reverse shoulder prosthesis for glenohumeral arthritis associated with severe rotator cuff deficiency. A minimum two-year follow-up study of sixty patients. J Bone Joint Surg Am. 2005;87:1697–705.
70. Wirth B, Kolling C, Schwyzer H-K, Flury M, Audigé L. Risk of insufficient internal rotation after bilateral reverse shoulder arthroplasty: clinical and patient-reported outcome in 57 patients. J Shoulder Elb Surg. 2016;25:1146–54.

22

Instability After Total Knee Arthroplasty

Michael T. Hirschmann, Antonia F. Chen, Sandro Kohl, Pier Francesco Indelli, Kimberly E. Hall, and Roland Becker

22.1 Introduction

National registries have been established in certain European countries, Canada, Australia, and New Zealand, to monitor the rates of revision replacement surgery. The average and median rates of primary and revision (combined) total knee arthroplasty (TKA) were 175 and 149 procedures/100,000 population, respectively, and ranged between 8.8 and 234 procedures/100,000 population for Romania and the United States, respectively [1]. In the United States alone, the increasing number of patients undergoing primary TKA (720,000 in 2010) has been accompanied by a similar increase in the number of revisions (70,000 in 2011).

When early TKA failure causes are infection and instability, equally, instability alone is the leading cause (up to 22%) of late revision TKAs [2].

Instability after TKA is one of the top three reasons as to why revision TKA is performed worldwide, especially in the early postoperative period [3–5]. Prominent causes of early instability include mismatch between the gaps (extension and flexion), improper component alignment in all three planes, and iatrogenic loss of ligamentous integrity. On the other hand, late instability is usually secondary to loss of fixation with concomitant mild/moderate/severe bone loss related to osteolysis formation from polyethylene wear. Since the objective of revision arthroplasty is anatomical and functional restoration of the knee joint, the gravity of bone loss and ligamentous and capsular laxity might compromise the final outcome. During revision TKA for asymmetric mediolateral instability, increasing levels of implant constraint are mandatory in order to obtain anteroposterior, mediolateral, and rotational stability of the prosthetic knee joint.

M.T. Hirschmann (✉)
Department of Orthopaedic Surgery and Traumatology, Kantonsspital Baselland (Bruderholz, Liestal, Laufen), Ch-4101, Bruderholz, Switzerland
e-mail: michael.hirschmann@unibas.ch

A.F. Chen
Orthopaedic Surgery, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA

S. Kohl
Trauma Zentrum Hirslanden, Klinik Hirslanden, Witelliker Strasse 40, CH-8032, Zürich, Switzerland

P.F. Indelli, MD, PhD • K.E. Hall, MD
Department of Orthopaedic Surgery and Bioengineering, Stanford University School of Medicine, Palo Alto Veterans Affairs Health Care System (PAVAHCS), Palo Alto, CA, USA

R. Becker
Department of Orthopedics and Traumatology, Centre of Joint Replacement, Medical Director, Hospital Brandenburg, Medical School "Theodor Fontane", Hochstrasse 2, 14770, Brandenburg, Germany

G.M.M.J. Kerkhoffs et al. (eds.), *ESSKA Instructional Course Lecture Book*,
https://doi.org/10.1007/978-3-662-56127-0_22

22.2 Patients at Risk for Instability After TKA

There are multiple predisposing patient factors that can lead to instability after TKA. These include patient medical conditions, anatomic considerations, and patient function including preoperative instability, muscle strength, and range of motion.

22.2.1 Medical Conditions

There are predisposing medical conditions that may contribute to increased instability after TKA. Connective tissue diseases, such as Ehlers-Danlos syndrome and Marfan syndrome, may lead to increased tissue laxity that may result in instability after TKA [6, 7]. Other conditions, such as multiple sclerosis, may also lead to TKA instability resulting in dislocations, and the use of revision components may be recommended in these patients to provide greater constraint [8–10]. Fibromyalgia patients commonly have persistent pain after TKA and are often revised for symptomatic instability or arthrofibrosis [11].

22.2.2 Anatomic Considerations

Anatomically, the ligaments of the knee contribute to knee stability. The anterior cruciate ligament (ACL) is often sacrificed during TKA, but the posterior cruciate ligament (PCL) may be retained to provide increased stability. Thus, if a PCL is ruptured postoperatively or if a PCL sacrificing implant is used, then there can be increased instability after TKA [12]. In a study by Montgomery et al., 3 out of 150 sustained a postoperative PCL rupture, and these patients experienced pain and chronic instability and eventually underwent revision TKA [13].

22.2.3 Function

Similar to range of motion, preoperative subjective instability leads to an increased risk of postoperative instability after TKA [14]. Of 390 participants, 72% reported preoperative instability, while 32% complained of persistent instability 6 months after undergoing TKA. Subjective knee instability was measured by the Activities of Daily Living Scale of the Knee Outcome Survey and was associated with increased pain, younger age (<60 years), low power with stair climbing, and increased comorbidities [14]. The authors concluded that improving lower extremity strength prior to surgery could reduce the amount of subjective instability after TKA. This was confirmed by Vince et al., who stated that neuromuscular pathology, including quadriceps weakness or hip abductor weakness, may contribute to instability and should be addressed prior to surgery [15].

Using falls as a proxy for instability after TKA, Matsumoto et al. compared TKA patients who fell to those who did not [16]. They found that those that fell had decreased knee flexion and ankle plantar flexion, indicating that increased range of motion could improve stability of the lower extremity when ambulating after TKA. By enhancing range of motion and muscle strength, the likelihood of instability decreases with diligent exercise.

22.3 How to Avoid Instability After TKA?

Although the pertinent question on how to avoid instability in patients after TKA has kept generations of knee surgeons busy and a considerable amount of research has been done to answer this question, the problem remains unsolved.

A detailed analysis of preoperative alignment, bony anatomy, and ligament laxity is crucial for a successful TKA [17]. The preoperative analysis typically starts with the assessment of preoperative leg alignment. In particular, severe varus or valgus deformity should be noted. Also, a flexion or extension deficit, as well as genu recurvatum, should be recognized [17].

Analysis of bony anatomy aims to predict bony wear. It is mandatory to perform weight-bearing anterior-posterior, lateral, and Rosenberg view radiographs. It is important to note if there is flexion facet osteoarthritis (OA), extension facet OA, or a combination of both [17].

Finally, ligament laxity should be evaluated in extension and 30 and 90° flexion. A comparison to the contralateral side is highly recommended [17].

Based on the aforementioned criteria, TKA is planned using standard planning software. In addition, the adequate level of constraint is chosen [17]. In terms of constraint, a cruciate-retaining (CR) TKA should only be used in patients with a competent PCL [17]. If the PCL is insufficient, a posterior cruciate-substituting (PS) TKA is indicated [17]. For both CR and PS TKA, the collateral ligaments should be competent. If the lateral collateral ligament is insufficient, a limited condylar constrained knee (LCCK) might be used [17]. If the medial collateral ligament is insufficient, a rotating-hinge TKA should be used [17].

At the time of surgery, the knee surgeon should then meticulously execute his plan. A profound insight into ligament balancing of TKA is necessary. There are numerous balancing methods and philosophies to achieve a stable and well-functioning knee after TKA. Adequate ligament balancing is a challenging demand to every surgeon. One major problem is the fact that, intraoperatively, only passive ligament laxity testing can be done. Navigation might add information on the laxity envelope before and after TKA.

22.4 Diagnostic Algorithm

Establishment of the correct diagnosis and characterization of the type of instability are difficult, even for experienced orthopedic surgeons [17]. The three pillars of diagnostics, consisting of patient's history, clinical examination, and radiological evaluation, cannot be overestimated for diagnosis of instability after TKA [17].

22.4.1 Patient's History

The patient's history brings the attention to the differential diagnosis "instability" [17]. Typically, patients suffering from instability after TKA complain about problems and pain when descending stairs [17]. The pain pattern is typically medial, lateral, or anterior [17]. This depends on the type of instability [17].

Fig. 22.1 Lateral asymmetric flexion instability in a patient after CR TKA

Patients with instability in extension often suffer from pain at the iliotibial tract insertion site, at Gerdy's tubercle [17]. Patients with instability in flexion either suffer from pain at the medial, lateral, or anterior knee compartment [17] (Fig. 22.1). Often, anterior knee pain is reported [17].

In the case of an insufficiency of the PCL in CR knees, typically anterior knee pain due to patellofemoral overloading is found [17].

22.4.2 Clinical Examination

Clinical examination is one of the cornerstones of diagnostics [17]. The diagnostic hypothesis, which is based on the patient's history, is specifically checked one by one [17]. It is important to standardize the test battery of clinical examination to gather experience with assessment of patients after TKA [17]. A comparison to the contralateral side is always recommended.

The four pillars of clinical examination are inspection, palpation, range of motion, and specific tests [17]. Inspection aims to identify erythema, skin coloration, scars, and deformities. In addition, the patient should be investigated while walking, including heel and toe gait. In particular, in heel gait, an instability in extension is represented by varus or valgus thrust.

Active and passive range of motion is noted. Anterior and posterior drawer tests help to identify flexion as well as anterior-posterior instability. Varus-valgus testing near extension and 30° flexion is recommended to reveal asymmetric medial-lateral instabilities. The posterior sag sign is also found in patients with posterior instability after TKA.

22.4.3 Radiological Evaluation

Primary radiological evaluations are two-plane radiographs (anterior-posterior and lateral views) with the patient standing on the affected leg and long-leg views with body weight equally distributed on both lower limbs [17]. These radiographs sometimes seem to reveal a more open joint space medially or laterally, which could be a sign of extension gap asymmetry or femoral condyle lift-off but could also just be a matter of projection [17].

On weight-bearing lateral radiographs, the femorotibial TKA contact point should be in the posterior two-thirds of the tibial plateau length [17]. However, in radiographs, only frank signs of static instability or TKA component malposition are seen [17]. Long-leg radiographs are necessary to determine changes while weight bearing and assessment of leg alignment (Fig. 22.2). Special radiographs such as the Kanekasu view are helpful to detect asymmetric flexion instability.

3D-reconstructed CT in combination with specific analysis software should be considered as the gold standard to evaluate TKA component position [17].

For assessment of more subtle findings of instability after TKA, stress radiographs should be used. Stress radiographs should be done in full extension and 20° flexion for varus-valgus stress and 15° and 90° flexion for anterior-posterior stress [17]. If stress radiographs are not available, fluoroscopic stress radiographs should be done (Figs. 22.3 and 22.4).

Fig. 22.2 Weight-bearing long-leg radiographs showing a medial instability in an obese patient after TKA

However, it represents not only a major challenge to quantify the degree of laxity and to correctly identify the type of instability but also to interpret the measured laxity values seen in stress radiographs [17]. The amount of normal laxity before and after TKA is still a matter of debate. A safe zone for laxity values after TKA has yet not been established [17].

In our opinion, increased laxity in any direction only represents instability when there are matching clinical symptoms and findings [17]. It

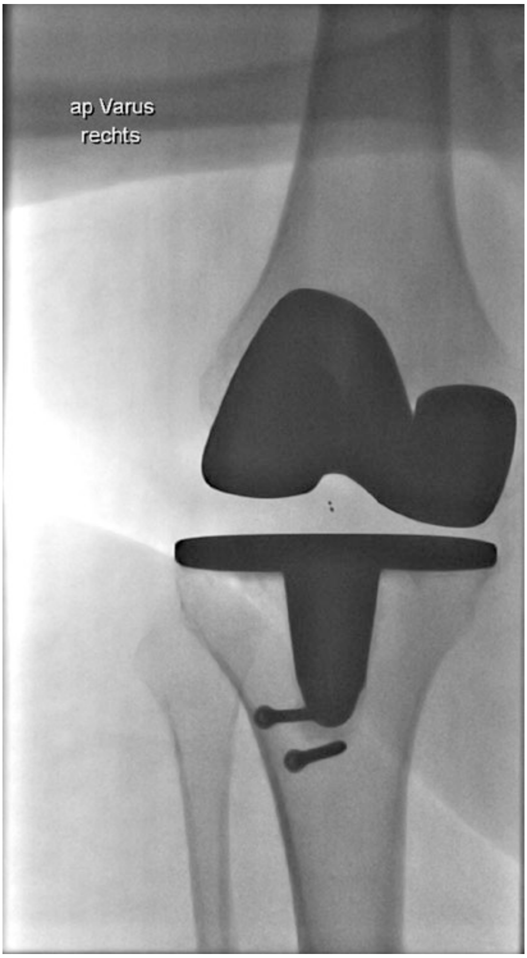


Fig. 22.3 Fluoroscopic varus stress radiographs showing a lateral instability near extension

is always the synopsis of patient's history, clinical findings, and radiological investigation [17].

22.4.4 Laboratory Tests

A periprosthetic joint infection (PJI) algorithm should always be followed to rule out infection: laboratory tests including C-reactive protein and erythrocyte sedimentation rate and analysis of the synovial fluid including arthrocentesis with cell count, culture, and sensitivity have been suggested by many authors [18]. Recently [19], a new leukocyte esterase test performed analyzing synovial fluid has been proposed to detect an otherwise negative PJI.

Fig. 22.4 Stress radiographs showing a lateral instability in extension

22.5 Classification of Instability after TKA

The classification of knee instability can be based on:

1. Time point—preoperative, intraoperative, postoperative
2. Plane of instability—instability in the sagittal plane (anteroposterior instability) or coronal plane (mediolateral instability)

3. Anatomical site—instability of one compartment such as the medial (valgus instability) or lateral (varus instability) compartment = asymmetrical instability; instability of the medial and lateral compartment = symmetrical instability
4. Flexion/extension gap—instability in knee extension, flexion, or mid-flexion
5. Compartment—instability at the femorotibial, patellofemoral, or both compartments

A precise analysis of the cause of the instability is mandatory for planning of surgery. Most often, both components require removal, and a higher constraint of TKA is chosen such as total stabilized or rotating-hinge design. However, in some cases change of single components or soft tissue reconstructive surgery might be sufficient.

22.5.1 Time Point

Instability before surgery is one of the indications for primary TKA. Instability may also occur during surgery and mainly affects the medial or lateral collateral ligament or the posterior cruciate ligament. Damage to the medial collateral ligament during surgery occurs in about 1% and is thus a very rare complication [20]. Stability is predominantly provided by the superficial medial collateral ligament. The deep part of the medial collateral ligament is generally resected during surgery because the insertion site is about 5 mm below the joint line [21]. In case of damage of the medial collateral ligament, one may consider conservative treatment keeping the patient in a valgus brace for 6 weeks, because the ligament shows good vascularization and healing capability [22].

In contrast, damage of the lateral collateral ligament should not be treated conservatively. Reconstructive surgery should be performed using the semitendinosus or gracilis tendon.

Iatrogenic damage of the PCL during preparation of the tibial plateau is not unusual. The insertion site of the PCL is resected in up to 70% during primary surgery [23]. It depends from the level and the posterior slope of the tibial cut.

The surgeon should be aware that damage of the posterior cruciate ligament may affect the flexion gap as shown by Schnurr et al. [24]. He found an increase in flexion gap of less than 2 mm in 44% and more than 3 mm in 36% of the patients. Twelve percent even showed an increase of more than 5 mm.

Instability may also occur after surgery due to trauma caused by fracture involving the insertion site of the medial or lateral collateral ligament or due to rupture of the collateral or posterior cruciate ligament.

22.5.2 Plane of Instability

Instability may occur in the sagittal plane due to insufficiency of the PCL, causing an increase in anteroposterior femorotibial translation. Insufficiency of the PCL during surgery requires the usage of either an ultracongruent or posterior-stabilized design. In case of secondary traumatic damage of the PCL and symptomatic instability, revision is required with the change of the components.

The most common instability is in the coronal plane due to insufficiency of the medial and/or lateral collateral ligament. Loss of function of the medial collateral ligament will cause valgus instability due to opening of the medial femorotibial compartment. A variety of different techniques have been introduced to reconstruct the medial collateral ligament [25, 26].

Insufficiency of the lateral collateral ligament causes varus deformity due to opening of the lateral compartment. There are several techniques to reconstruct the lateral collateral ligament, by using the semitendinosus or gracilis tendon [27–29] or a strip of the iliotibial band according to Bosquet [30]. The semitendinosus or gracilis tendon might be used for reconstruction (Figs. 22.5, 22.6, and 22.7).

The use of a thicker liner is not recommended due to the high risk of creating a patella infera. Alternatively, in some cases the intraoperative change to a higher constrained design such as total stabilized or rotating hinged knee might be required (Fig. 22.8).

Fig. 22.5 There was a history of an acute trauma. Correct component placement prior to revision surgery was proven. The lateral collateral ligament was reconstructed using the gracilis tendon

Fig. 22.6 A tunnel was drilled through the fibular head and the insertion site at the medial epicondyle was identified using an imaging intensifier

Combined instability may also occur at the medial and lateral site due to inappropriate flexion or extension gap, a complication caused by the surgeon.

Fig. 22.7 An interference screw serves for graft fixation at the femoral site

22.5.3 Anatomical Site

Isolated instability shows varus or valgus deformity and is mainly caused by either severe bone loss in one of the two femorotibial compartments or ligamentous insufficiency [17]. Varus deformity for instance can be caused by bone loss at the medial compartment due to severe osteoarthritis or osteonecrosis but also due to insufficiency of the lateral collateral ligament [17]. On the other side, valgus deformity can be caused due to bone loss at the lateral compartment or damage of the medial collateral ligament [17]. In contrast, symmetric instability affects both the medial and lateral joint space and in addition rotation of the knee as well [17]. Complex instability is most often caused by inappropriate ligament balancing and a mismatch between the flexion and extension gap [17].

22.5.4 Flexion and Extension Gap (Fig. 22.9)

Mismatch between the flexion and extension gap is predominantly caused by erroneous bony resection at the femoral or tibial site [17]. It is recommended to measure the resected bone after each cut, which will allow the surgeon to be aware of any abnormalities during the entire

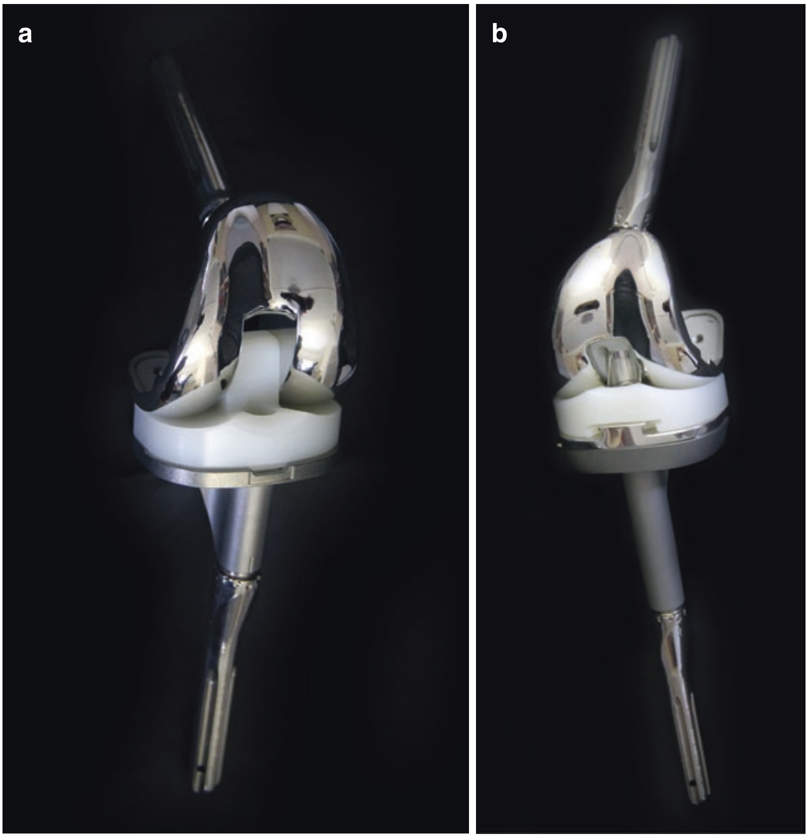


Fig. 22.8 Total stabilized (**a**) and rotating hinged (**b**) knees are the more constrained design used in total knee arthroplasty

Flexion Extension	Tight	Normal	Loose
Tight	1. More tibial resection 2. Decrease the poly thickness	1. More resection of distal femur	1. More distal femoral resection 2. Augment at posterior femoral condyles
Normal	1. More resection of posterior femoral condyles		1. Augment at posterior femoral condyles
Loose	1. More resection at posterior femoral condyles 2. Augment at the distal femur	1. Augment at distal femur	1. Increase the poly thickness 2. Augment at distal and posterior femoral condyles

Fig. 22.9 The table shows the treatment options for joint gap difference or asymmetry in flexion and/or extension

surgical procedure [17]. In computer surgery, this information is provided when the resection needs to be confirmed by the surgeon before the computer will allow the next step of the procedure.

Isolated extension instability (mediolateral asymmetry with the knee in extension) usually arises from an originally poor extension gap balancing: the main cause is usually excessive distal femoral resection that was not originally managed with a large polyethylene insert. Azzam et al. [31] suggested the use of distal femoral augments to facilitate the proper positioning of the revision components and to regain the necessary extension symmetry, restoring the correct joint line level at the same time. On the other hand, excessive tibial resection usually increases both gaps equally: in this case, the use of a thicker polyethylene insert may fill the gaps, but this may also lead to patellar impingement during deep knee flexion.

Another reason for mediolateral asymmetric TKA instability in extension is an excessive intraoperative medial or lateral soft tissue release to compensate for an excessively tight compartment, leading to varus-valgus instability in extension: this scenario can be treated by increasing the level of implant constraint (i.e., revision to a PS or semi-constrained implant). In this scenario, the level of constraint should be decided according to the degree of soft tissue laxity: Azzam et al. [31] do not recommend, when this scenario is present, soft tissue reconstruction, like a collateral ligament repair or soft tissue advancement, because of a high failure rate of such procedures.

Flexion instability usually occurs when the flexion gap is significantly larger than the extension gap [17]. Causes of pure flexion gap instability include excessive resection of the posterior femoral condyles, a small femoral component on the AP plane, an insufficient distal femoral resection with an appropriate posterior femoral resection combined with an adjusted tibial cut to create a normal extension gap, and a progressive attenuation of the PCL when a CR design is used [17]. At the time of the preoperative evaluation, an intraoperative anterior drawer of more than 5 mm with a relocated extensor mechanism may raise suspicion of flexion instability. As a solution for this, a more conservative approach suggests the use of a thicker polyethylene insert to fill the flexion gap, providing better flexion stability but increasing tightness in the extension gap, often resulting in a permanent flexion contracture. Because of this possible complication, in the presence of pure flexion instability, we recommend revising the femoral component too: posterior femoral augments and/or an upsized femoral component is the gold standard for flexion instability correction during revision TKA.

Mid-flexion instability is increasingly raising concerns as a cause of TKA revision [17]. This kind of knee instability is usually characterized by a knee that is stable in both full extension and in flexion at 90°, but instability develops during the 30–90° arc of motion. Such instability may be not recognized in most cases because of the subtle nature of complaints of the patient, like development of anterior knee pain while rising from a chair or descending a flight of stairs. In a well-balanced knee, soft tissue tension should be equal, not only mediolaterally but also anteroposteriorly. In fact, the main stabilizer of the knee in extension is the posterior capsule and in flexion are the collateral ligaments: it is important to understand the interplay between these soft tissue structures at the time of revision surgery.

Usually, the main two causes for mid-flexion instability are the inappropriate balancing of the soft tissues (medial collateral ligament (MCL) and/or posterior capsule) and malpositioning of the implant, both leading to poor ligamentous isometry [17]. The anterior portion of the MCL has been shown to be the main knee stabilizer between 30 and 60° of flexion [32]. On the other side, malpositioning of the implant modifies the tibiofemoral geometry and the final position of the epicondyles, influencing collateral ligaments isometry and leading to mid-flexion instability too.

Mid-flexion instability associated with correct implant positioning can be described in three types: (1) over-released MCL and normal posterior capsule, (2) normal MCL with a tight posterior capsule, and (3) over-release of both the MCL and the posterior capsule.

Another frequent cause of mid-flexion instability is original components downsizing: it has been demonstrated that also a mild components downsizing (2 mm on the femoral and 1 mm on the tibial component) has a similar increase in laxity in mid-flexion [33]. Treatment of mid-flexion instability usually includes increasing the femoral condylar offset, proximalization of the joint line, and decreasing the posterior tibial slope.

A clear understanding of flexion/extension balancing is mandatory before performing either primary or revision surgery.

22.5.5 Suboptimal TKA Component Position

Evaluation of component malposition must include preoperative radiographic [34] and intraoperative visual assessment. Component malposition is generally defined as a coronal plane malalignment of >5°, a sagittal plane tibial component malalignment of <0° (anterior slope) or >10° (excessive posterior slope), or axial malalignment of the femoral component of >5° internal rotation. Component malpositioning can gradually interact with the soft tissue environment, leading to a secondary cause of instability: for example, component malpositioning could attenuate the periarticular soft tissues environment and be combined with previously isolated ligament and extensor mechanism insufficiency. In order to fully understand component malpositioning, surgeons must be able to explain the direction and pattern of instability.

22.5.6 Isolated Ligament Insufficiency

Isolated ligament insufficiency usually includes postoperative traumatic rupture or chronic functional attenuation of the PCL in a PCL-retaining TKA, of the medial collateral ligament (MCL) or of the lateral collateral ligament (LCL) [17]. The identification of isolated ligament insufficiency etiology is often difficult and not always useful for planning revision TKA surgery. On the other side, the identification of the anatomical feature of the isolated ligament insufficiency and chronicity is rather useful. The specific differentiation of the insufficient ligament could make the selection of constraint prosthesis more reasonable.

22.5.7 Extensor Mechanism-Related Instability

Extensor mechanism insufficiency includes acute or chronic patellar or quadriceps tendon rupture, patellar bone fracture with displaced nonunion, patellar dislocation, or dissociation of the patellar component [17]. According to the cause of patellofemoral-related instability (patellar component problems, patellar bone and bone integrity issues, soft tissue imbalance, or instability of the patellofemoral joint), the revision strategy and the degree of constraint might change consequently.

22.5.8 Loosening of Components

Components loosening must be first assessed by preoperative radiographic evaluation and by intraoperative findings subsequently. Historically, radiographically loose components are those with a progressive complete radiolucent line of >2 mm in width around the component, a visible fracture of the cement around the components, or a change in component position including subsidence. Knees with component loosening may progress to multidirectional instability: the right degree of constraint must to be chosen at the time of revision surgery in order to obtain neutral alignment, accurate components position, proper components sizing, and fixation [17].

22.5.9 Global Instability

Global instability is defined as combined mediolateral and both flexion and extension instability. Knees with global instability can be divided in three subcategories: (1) soft tissue attenuation due to chronic synovitis, recurrent hemarthrosis, or undersizing of polyethylene (PE) insert, (2) direct negative effect of PE insert such as post-fracture or wear, and (3) knee dislocation. Tibiofemoral dislocation is the extreme form of global instability: this is usually due to progression of severe imbalance of flexion gap and extensor mechanism insufficiency.

Generally, the timing of instability symptoms manifestation could be related to the causes of instability itself. Intraoperative technical errors such as flexion/extension gap mismatch and component malposition tend to present early in the postoperative period. Other instabilities have a more subtle appearance, tending to produce attenuation of the soft tissues environment first and becoming clinically evident. In a review of consecutive TKA revisions, it was demonstrated that the interval between the primary and the revision surgery was significantly shorter in the gap mismatch and component malpositioning category groups than in other category groups.

22.6 Treatment of Instability

Surgical treatment modalities for instability do not include a single procedure, but several different procedures, according to the instability etiology: treatments range from a simple exchange of PE insert to a full TKA revision with variable levels of constraint in often complex combined surgeries. It has been recommended to use a minimally constrained type of prosthesis to regain knee stability [35, 36].

Four basic treatment options have been described to treat TKA instability:

1. Isolated polyethylene exchange
2. Single-component revision
3. Full-component revision
4. Hinged arthroplasty

22.6.1 Isolated Polyethylene Exchange

Isolated polyethylene exchange is an attractive, low-morbidity solution for TKA instability [17]. The prerequisites for isolated polyethylene exchange are proper knee alignment and stable fixation of the retaining components. It is most helpful in cases of global instability where it appears that the polyethylene originally placed was not thick enough: in this scenario, the patient knee will likely go into recurvatum in extension and will have anteroposterior instability at 90° of flexion too. Another possible scenario manifests itself when PCL insufficiency occurs in a patient with a posterior cruciate-retaining implant: in this case, if the manufacturer has an ultracongruent polyethylene insert available, a single polyethylene exchange might be sufficient to regain adequate knee stability. A final yet quite rare type of instability, where isolated polyethylene exchange is still an appropriate option, is pure mediolateral instability because of varus/valgus ligamentous imbalance: in this scenario, the soft tissues on the tight compartment can be gradually released to catch up with the stretched compartment and a thicker polyethylene be placed to appropriately balance the knee. Unfortunately, the current literature [13, 37–39] demonstrated that the results of isolated polyethylene exchange are usually poor and unpredictable.

22.6.2 Single-Component Revision

The prerequisite for the use of this surgical technique is stable fixation and proper axial alignment of the retained modular implant [17]. The primary indication for this technique is in a posterior cruciate-retaining knee when the PCL becomes secondarily incompetent: in this scenario, the femoral component can be revised to a posterior-stabilized implant, and a posterior-stabilized polyethylene insert is placed to address the instability issue. Another indication for an isolated femoral component revision is the scenario of isolated medial collateral ligament incompetence: the femoral component can be revised to a constrained condylar design, and a constrained tibial insert can be placed into the stable well-fixed and well-aligned tibial baseplate. Early isolated femoral loosening in the picture of an otherwise stable, well-fixed, and well-aligned knee is another indication for single-femoral component revision (Fig. 22.10). An isolated femoral revision allows surgeons to adjust the joint line height, to balance flexion/extension gap, and to increase the level of constraint.

Fig. 22.10 Right TKA. Femoral (CR) component loosening: visible fracture of the cement (left). Femoral component revision: a short cemented stem and a PS polyethylene insert have been used. *CR* cruciate retaining, *PS* posterior stabilized

Isolated tibial revision is indicated in the event of isolated tibial loosening (Fig. 22.11) with a well-fixed and well-aligned retained femoral implant: if this scenario is caught early, there might still be little damage to the soft tissue knee environment, allowing for the use of a posterior stabilized implant without increasing the level of constraint.

22.6.3 Complete Revision of TKA Components

Full-component revision is generally indicated when there is a clear malalignment of the components, a poor track record of the retained implant, or inadequate constraint options of the existing implant [17]. The revision of femoral and tibial components allows realignment, correct flexion/extension gaps balancing, and restoration of the joint line [17].

Complete TKA revision is a stepwise procedure: first, the entity of bone loss needs to be detected and addressed. Second, femoral and tibial implants need to be chosen. Third, the level of constraint needs to be intraoperatively selected.

Treatment of bone loss during revision TKA has evolved considerably over the past couple of decades, primarily due to the emergence and rapid adoption of metaphyseal fixation implants. To date, there are two basic categories of metaphyseal fixation devices [40]: (1) porous-coated solid sleeves that are unitized to an intended implant stem and (2) highly porous cones that are implanted into the metaphyseal region separate from the intended final implant (Fig. 22.12).

The selection of the tibial implant should allow reconstruction of a stable platform without overhanging and stem impingement and with good stem support [17]. Wedges (Fig. 22.13) should allow bone substitution and raise the joint line to its natural height. The selection of the femoral

Fig. 22.11 Left total knee arthroplasty. Loosening of the tibial component and overall varus malalignment (top); the tibial component has been revised with a metaphyseal porous-coated solid sleeve and a cementless stem: a posterior-stabilized (PS) polyethylene insert has been used (bottom)

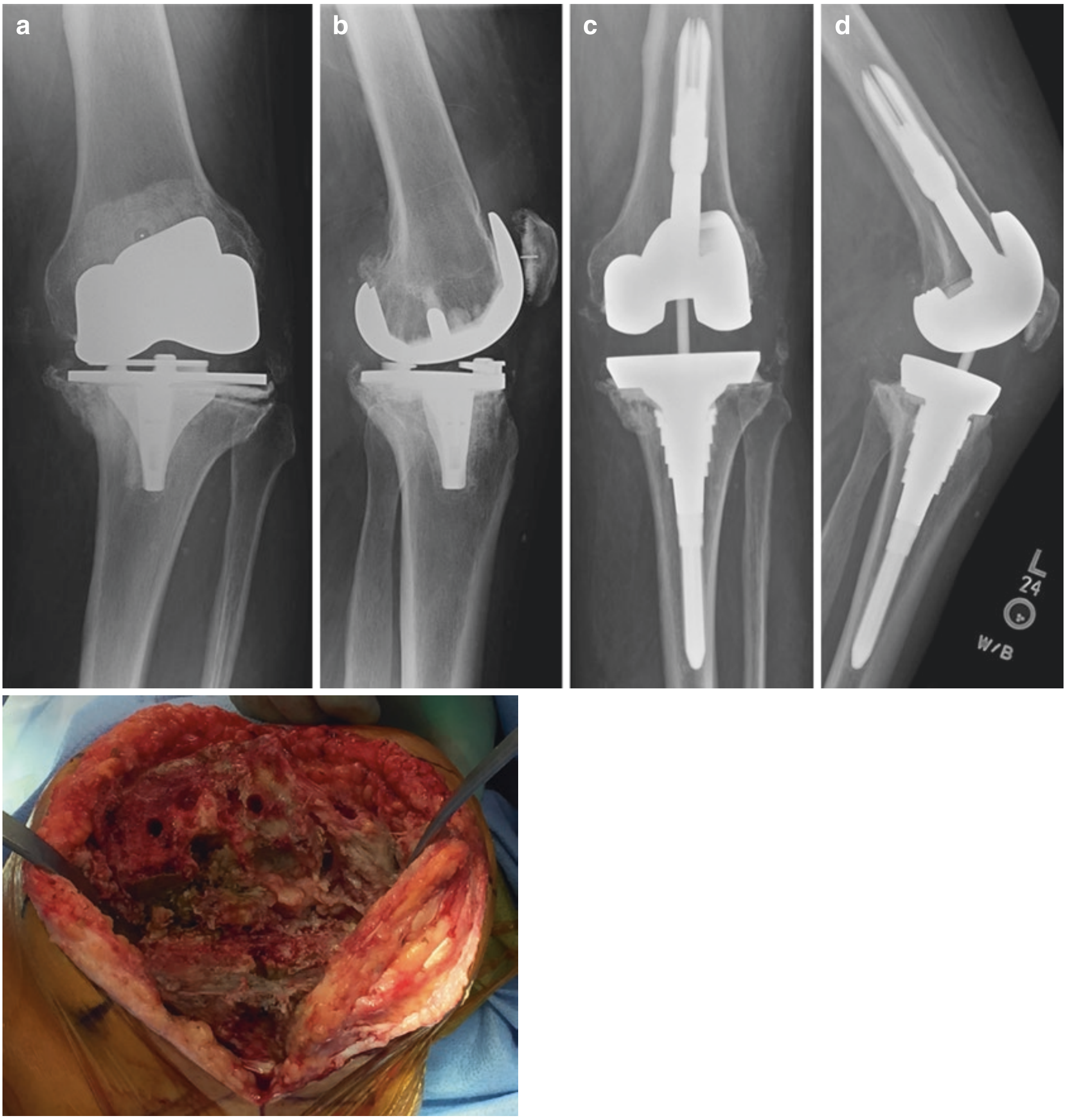


Fig. 22.12 (**a**) Left TKA. (A) Weight-bearing anteroposterior view showing severe poly wear, loosening of the tibial component and severe bone loss. (B) Lateral view; (C) revision TKA: anteroposterior view showing the use of a titanium metaphyseal sleeve. (D) Revision TKA: lateral view. (**b**) Left RKA revision: intraoperative image showing the amount of bone loss after components removal

implant should allow recreation of the natural posterior condylar offset and the normal flexion gap, improving stability in flexion. Sometimes, the use of a long straight femoral stem should be avoided because of the tendency to increase the anterior offset leading to over-resection of the posterior condyles and an increase in flexion gaps: this happens because the diaphyseal fit of the stem leads to a relative extension position of the femur compared to the relative natural anterior bow of the native distal femur [17]. In this scenario, in order to recreate a symmetric flexion gap, a thicker polyethylene insert is occasionally needed, and thus a more proximal femoral resection is

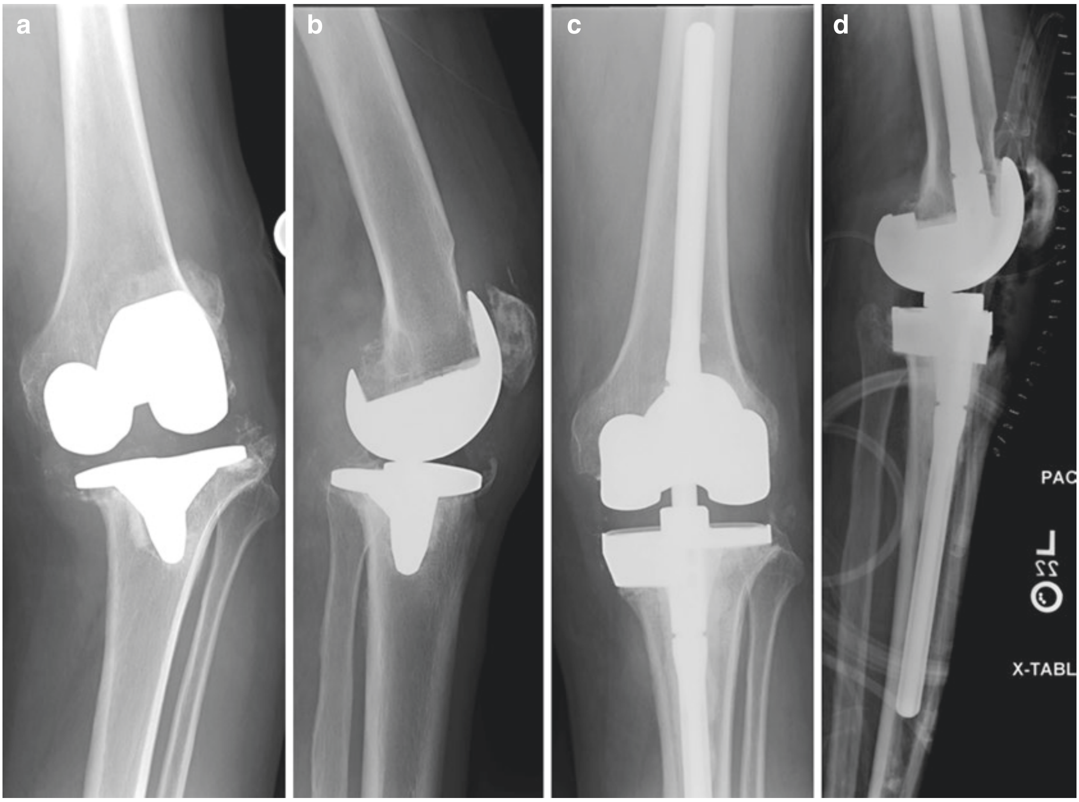


Fig. 22.13 Left knee. (**a**) Anteroposterior weight-bearing view of a left TKA showing poly wear, definite loosening of the tibial component, possible loosening of the femoral component with anterior cortical "notching," and severe tibial bone loss. (**b**) Lateral view; (**c**) revision TKA: anteroposterior view showing the use of a trabecular metal wedge to fill the bone loss. (**d**) Revision TKA: lateral view

required to fit the same thick polyethylene in extension: this can occasionally lead to a mild to moderate patella infera [17].

Historically, the guiding principle for the surgical treatment of TKA instability is to use the least constrained implant to solve the instability problem [41]. Unfortunately, in most unstable TKA, because of the periprosthetic soft tissue damage, it is practically impossible to achieve stability without implanting a semiconstrained or fully constrained prosthesis [42]. In fact, posterior-stabilized implants provide sufficient anteroposterior stability but little mediolateral stability: they are mainly used to substitute the posterior cruciate ligament. Differently, a constrained insert can help regain TKA stability in multiple planes: it provides anteroposterior and mediolateral stability, and it is primarily designed to substitute deficient collateral ligaments. The height of the post (different according to the manufacturer) avoids posterior knee dislocation and is generally taller than a posterior stabilized construct. Constraint differs between manufacturers with regard to varus/valgus constraint, rotational constraint, and post height [43].

A complete revision of the components is often necessary in a scenario of patellar maltracking or instability: this is required because patellofemoral instability results most frequently from internal malrotation of the femoral or tibial components [44]. Patellar-"unfriendly" designs have been related to a high revision rate because of severe extensor mechanism complications (Fig. 22.14).

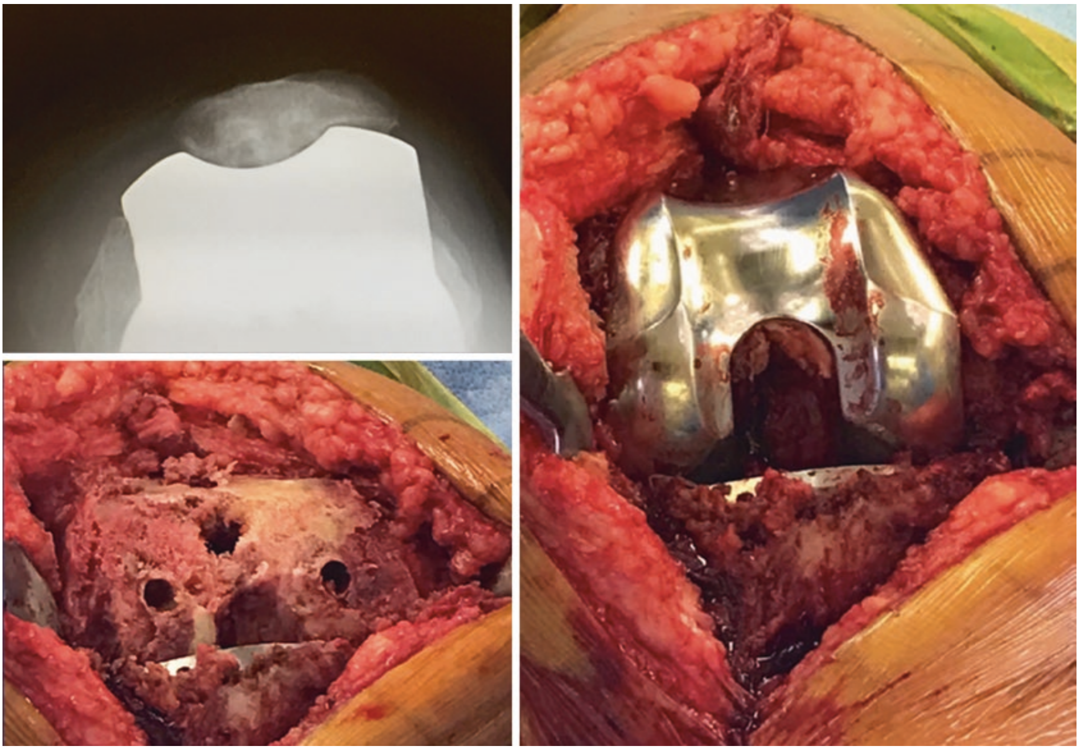


Fig. 22.14 Right TKA revision because of severe anterior knee pain and patellar lateral facet chondropathy. A patellar-"unfriendly" design of the femoral groove can be noted

22.6.4 Revision TKA with a Hinged System

Primary indications for using a hinged arthroplasty are severe distal femoral bone loss (Fig. 22.15); severe flexion gap instability, which cannot be matched by the extension gap; and presence of a totally disrupted MCL in an elderly patient. However, in a younger patient population, the use of a constrained condylar design augmented by a MCL allograft reconstruction has been advocated as a better choice. In reality, hinged total knee implants have limited usage among relatively young and active patients because they have been associated with increased risk of a secondary revision due to early loosening caused by excessive stress at the fixation interface [45]. When a surgeon is forced to use a hinged device, a rotating bearing design has been advocated because of the theoretical advantage to diminish stress at the implant-bone interfaces. Last, when using a hinge, the surgeon must be aware of the variability in disengagement potential between manufacturers [46].

Few studies demonstrated that patients undergoing complete revision had a better outcome than those undergoing isolated polyethylene exchange [31, 38]. Revision of all components offers the opportunity of increasing the level of intra-articular constraint, improving final stability. Controversy exists with regard to different levels of constraint in revision TKA. Hass et al. [47] and Hwang et al. [48] reported better clinical outcomes when PS systems were chosen over more constrained implants. On the other side, Shen et al. [49] and Lachiewicz et al. [50] showed inferior clinical results when an unconstrained prosthesis was utilized in the Anderson Orthopaedic Research Institute (AORI) [51] type I bone defect. There is also controversy regarding the outcome of unlinked constrained knees and hinged knee prostheses. Barrack et al. [52], Hossain et al. [53], and Kim et al. [54] all demonstrated comparable results between condylar constrained prosthesis

Fig. 22.15 Left knee. Antibiotic-loaded cement spacer following left total knee arthroplasty septic loosening (top); revision total knee arthroplasty with a hinged implant (bottom)

and hinged prosthesis. Shen et al. [49] suggested unlinked constrained prostheses offering superior results when used in aseptic AORI type II and type III patients; on the other side, the septic AORI type II and type III patients were found to have a better outcome when hinged prostheses were utilized.

22.7 Take-Home Message

Instability after TKA is one of the most important problems after TKA. A detailed analysis of preoperative alignment, bony anatomy, and ligament laxity is crucial for a successful TKA. The adequate level of constraint needs to be carefully chosen based on the aforementioned evaluation. A profound insight into ligament balancing of TKA is necessary to avoid instability or stiffness after TKA.

In patients with pain after TKA, instability is one of the most important causes. Diagnostics is challenging and consists of a detailed patient's history, thorough clinical examination, radiological investigations, and lab work. Treatment depends on the type of instability and ranges from a simple exchange of PE insert to a full TKA revision with variable level of constraint in often complex combined surgeries.

References

1. Kurtz SM, Ong KL, Lau E, Widmer M, Al e. International survey of primary and revision total knee replacement. Int Orthop. 2011;35(12):1783–9.
2. Sharkey PF, Hozack WJ, Rothman RH, Shastri S, Jacoby SM. Insall award paper: why are total knee arthroplasty failing today? Clin Orthop Relat Res. 2002;404:7–13.
3. Hardeman F, Londers J, Favril A, Witvrouw E, Bellemans J, Victor J. Predisposing factors which are relevant for the clinical outcome after revision total knee arthroplasty. Knee Surg Sports Traumatol Arthrosc. 2012;20(6):1049–56.
4. Le DH, Goodman SB, Maloney WJ, Huddleston JI. Current modes of failure in TKA: infection, instability, and stiffness predominate. Clin Orthop Relat Res. 2014;472(7):2197–200.
5. Koh IJ, Cho WS, Choi NY, Kim TK, Kleos Korea Research Group. Causes, risk factors, and trends in failures after TKA in Korea over the past 5 years: a multicenter study. Clin Orthop Relat Res. 2014;472(1):316–26.
6. Rose PS, Johnson CA, Hungerford DS, McFarland EG. Total knee arthroplasty in Ehlers-Danlos syndrome. J Arthroplast. 2004;19(2):190–6.
7. Wolf JM, Cameron KL, Owens BD. J Am Acad Orthop Surg. 2011;19(8):463–71.
8. Bron JL, Saouti R, De Gast A. Posterior knee dislocation after total knee arthroplasty in a patient with multiple sclerosis. A case report. Acta Orthop Belg. 2007;73(1):118–21.
9. Dawson-Bowling S, Tavakkolizadeh A, Cottam HL, Butler-Manuel PA. Multiple sclerosis and bilateral dislocations of total knee replacements: a case report. Knee Surg Sports Traumatol Arthrosc. 2008;16(2):148–51.
10. Rao V, Targett JP. Instability after total knee replacement with a mobile-bearing prosthesis in a patient with multiple sclerosis. J Bone Joint Surg (Br). 2003;85(5):731–2.
11. D'Apuzzo MR, Cabanela ME, Trousdale RT, Sierra RJ. Primary total knee arthroplasty in patients with fibromyalgia. Orthopedics. 2012;35(2):e175–8.
12. Kim SM, Jang SW, Seo JG, Lee SS, Moon YW. Comparison of cruciate retaining and PCL sacrificing TKA with respect to medial and lateral gap differences in varus knees after medial release. J Arthroplast. 2015;30(1):26–30.
13. Montgomery RL, Goodman SB, Csongradi J. Late rupture of the posterior cruciate ligament after total knee replacement. Iowa Orthop J. 1993;13:167–70.
14. Fleeton G, Harmer AR, Nairn L, Crosbie J, March L, Crawford R, van der Esch M, Fransen M. Self-reported knee instability before and after Total knee replacement surgery. Arthritis Care Res. 2016;68(4):463–71.
15. Vince KG, Abdeen A, Sugimori T. The unstable total knee arthroplasty: causes and cures. J Arthroplast. 2006;21(4 Suppl 1):44–9.
16. Matsumoto H, Okuno M, Nakamura T, Yamamoto K, Hagino H. Fall incidence and risk factors in patients after total knee arthroplasty. Arch Orthop Trauma Surg. 2012;132(4):555–63.
17. Hirschmann MT, Becker R. Unhappy total knee replacement. A comprehensive review and management guide. Switzerland: Springer International Publishing; 2015. https://doi.org/10.1007/978-3-319-08099-4.
18. Volpe L, Indelli PF, Latella L, Poli P, Yakupoglu KJ, Marcucci M. Periprosthetic joint infections (PJI): a clinical practice algorithm. Joints. 2014;2(4):169–74.
19. Aggarwal VK, Tischler E, Ghanem E, Parvizi J. Leukocyte esterase from synovial fluid aspirate: a technical note. J Arthroplast. 2013;28:193–5.
20. Bohl DD, Wetters NG, Del Gaizo DJ, Jacobs JJ, Rosenberg AG, Della Valle CJ. Repair of intraoperative injury to the medial collateral ligament during primary total knee arthroplasty. J Bone Joint Surg Am. 2016;98:35–9.
21. Maes M, Luyckx T, Bellemans J. Does a conservative tibial cut in conventional total knee arthroplasty violate the deep medial collateral ligament? Knee Surg Sports Traumatol Arthrosc. 2014;22:2735–279.

22. Woo SL-Y, Vogrin TM, Abramowitch SD. Healing and repair of ligament injuries in the knee. J Am Acad Orthop Surg. 2000;8:364–72.
23. Matziolis G, Mehlhorn S, Schattat N, Diederichs G, Hube R, Perka C, Matziolis D. How much of the PCL is really preserved during the tibial cut? Knee Surg Sports Traumatol Arthrosc. 2012;20:1083–6.
24. Schnurr C, Eysel P, König DP. Is the effect of a posterior cruciate ligament resection in total knee arthroplasty predictable? Int Orthop. 2012;36:83–8.
25. Adravanti P, Dini F, Calafiore G, Rosa MA. Medial collateral ligament reconstruction during TKA: a new approach and surgical technique. Joints. 2015;3:215–7.
26. Lind M, Jakobsen BW, Lund B, Hansen MS, Abdallah O, Christiansen SE. 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. 2009;37:1116–22.
27. Fanelli GC. Surgical treatment of lateral posterolateral instability of the knee using biceps tendon procedures. Sports Med Arthrosc. 2006;14:37–43.
28. Larson R. Isometry of the lateral collateral and popliteofibular ligaments and techniques for reconstruction using a free semitendinosus tendon graft. Oper Tech Sports Med. 2001;9:84–90.
29. Zantop T, Petersen W. Modified Larson technique for posterolateral corner reconstruction of the knee. Oper Orthop Traumatol. 2010;22:373–86.
30. Becker R. Posterolaterale Rekonstruktion des Kniegelenks. Arthroskopie. 2014;27:208–12.
31. Azzam K, Parvizi J, Kaufman D, Purtill JJ, Sharkey PF, Austin MS. Revision of the unstable total knee arthroplasty: outcome predictors. J Arthroplast. 2011;26(8):1139–44. https://doi.org/10.1016/j.arth.2011.02.028.
32. Phisitkul P, James SL, Wolf BR, Amendola A. MCL injuries of the knee: current concepts review. Iowa Orthop J. 2006;26:77–90.
33. Muller JK, Wentorf FA, Moore RE. Femoral and tibial insert downsizing increases the laxity envelope in TKA. Knee Surg Sports Traumatol Arthrosc. 2014;22(12):3003–11.https://doi.org/10.1007/s00167-014-3339-0.
34. Ryd L, Albrektsson BE, Carlsson L, et al. Roentgen stereophotogrammetric analysis as a predictor of mechanical loosening of knee prostheses. J Bone Joint Surg (Br). 1995;77(3):377.
35. Bugbee WD, Ammeen DJ, Engh GA. Does implant selection affect outcome of revision knee arthroplasty? J Arthroplast. 2001;16(5):581.
36. Scuderi GR. Revision total knee arthroplasty: how much constraint is enough? Clin Orthop Relat Res. 2001;392:300–5.
37. Babis GC, Trousdale RT, Pagnano MW, Morrey BF. Poor outcomes of isolated tibial insert exchange and arthrolysis for the management of stiffness following total knee arthroplasty. J Bone Joint Surg Am. 2001;83(10):1534–6.
38. Babis GC, Trousdale RT, Morrey BF. The effectiveness of isolated tibial insert exchange in revision total knee arthroplasty. J Bone Joint Surg Am. 2002;84-A(1):64.
39. Waslewski GL, Marson BM, Benjamin JB. Early, incapacitating instability of posterior cruciate ligament-retaining total knee arthroplasty. J Arthroplast. 1998;13(7):763.
40. Haidukewych GJ, Hanssen A, Jones RD. Methaphyseal fixation in revision total knee arthroplasty: indications and techniques. J Am Acad Orthop Surg. 2011;19(6):311–8.
41. Berend ME, Bertrand T. The role of implant constraint: not too little, not too much. Orthopedics. 2007;30(9):793.
42. Pritsch M, Fitzgerald RH Jr, Bryan RS. Surgical treatment of ligamentous instability after total knee arthroplasty. Arch Orthop Trauma Surg. 1984;102:154–8.
43. Lombardi AV, Berend KR. Posterior cruciate ligament retaining, posterior stabilized, and varus/valgus posterior stabilized constrained articulations in total knee arthroplasty. Instr Course Lect. 2006;55:419–27.
44. Malo M, Vince KG. The unstable patella after total knee arthroplasty: etiology, prevention, and management. JAAOS. 2003;11(5):364–71.
45. Easley ME, Insall JN, Scuderi GR, Bullek DD. Primary constrained condylar knee arthroplasty for the arthritic valgus knee. Clin Orthop Relat Res. 2000;380:58–64.
46. Ward WG, Haight D, Ritchie P, Gordon S. Eckardt: dislocation of rotating hinge total knee prostheses. J Bone Joint Surg. 2003;85(A):448–63.
47. Haas SB, Insall JN, Montgomery W III, et al. Revision total knee arthroplasty with use of modular components with stems inserted without cement. J Bone Joint Surg Am. 1995;77:1700.
48. Hwang SC, Kong JY, Nam DC, et al. Revision total knee arthroplasty with a cemented posterior stabilized, condylar constrained or fully constrained prosthesis: a minimum 2-year follow-up analysis. Clin Orthop Surg. 2010;2:112.
49. Shen C, Lichstein PM, Austin MS, Sharkey PF, Parvizi J. Revision knee arthroplasty for bone loss: choosing the right degree of constraint. J Arthroplast. 2014;29:127–31.
50. Lachiewicz PF, Soileau ES. Fifteen-year survival and osteolysis associated with a modular posterior stabilized knee replacement. A concise follow-up of a previous report. J Bone Joint Surg Am. 2009;91:1419.
51. Engh GA, Parks NL. The management of bone defects in revision total knee arthroplasty. Instr Course Lect. 1997;46:227.
52. Barrack RL. Evolution of the rotating hinge for complex total knee arthroplasty. Clin Orthop Relat Res. 2001;392:292.
53. Hossain F, Patel S, Haddad FS. Midterm assessment of causes and results of revision total knee arthroplasty. Clin Orthop Relat Res. 2010;468:1221.
54. Kim YH, Kim JS. Revision total knee arthroplasty with use of a constrained condylar knee prosthesis. J Bone Joint Surg Am. 2009;91:1440.

23

Dance Orthopaedics, Ballet Injuries and When to Perform Surgical Treatment

Duncan E. Meuffels, Rintje Agricola,
and James Calder

23.1 Introduction

Ballet injuries are either injuries that occur whilst performing ballet or are musculoskeletal injuries that happen to ballet dancers. In either case, a thorough knowledge is necessary of the specific demands that ballet asks of the artist, who is now your patient. An orthopaedic surgeon should be specialised in sports medicine and should understand that the demands of the dancer are very specific due to the nature of the prescribed dance technique and due to the extremely demanding professions that is ballet.

It starts with knowing the appropriate nomenclature and finding out what the demand of the dancer is. The treating physician should combine this knowledge with the specific anatomical knowledge of the lesion and the professional surgical skills as a sports orthopaedic surgeon. This chapter focuses on injuries that result from dancing and require surgical intervention that is specific for professional ballet dancers.

Dance is a very common pastime and comes in a wide variety of forms and shapes. Ballet is a very specific form of dance, and professional ballet dancers are a much more rare breed. Most countries have a national classical ballet company and will have one or more other companies, which will use classical ballet as a technique, incorporated in many of the choreographies that they perform annually. In the Netherlands, the National Ballet consists of around 90 dancers, and some of the other companies using classical technique but maybe no point work are the Nederlands Dans Theater, Scapino Ballet Rotterdam and Introdans comprising of in total 250 dancers. These dancers are full-time dancers and have devoted their whole life to the art of dance. They will rehearse daily, starting with a barre, and will then rehearse the performance at hand and the new choreography that will be performed the next month [1]. In general they will perform around 100 times a year and will tour nationally and internationally for 2–3 months. Performances can be six times a week or even up to twice daily (matinee and evening performances). There is no periodization or performing at a friendly pace, which makes an injury difficult to accept, and it is never at the right moment. More than half of the professional dancers rehearse and perform with pain, which they have come to tolerate and accept as part of their dance life [2].

Little has been published specifically on surgical treatment for ballet injuries. A narrative review on what is known and what experience the faculty

---

D.E. Meuffels (✉) • R. Agricola
Orthopaedic Department, Erasmus MC,
University Medical Centre Rotterdam,
Rotterdam, The Netherlands
e-mail: d.meuffels@erasmusmc.nl

J. Calder
Fortius Clinic, London, UK

G.M.M.J. Kerkhoffs et al. (eds.), *ESSKA Instructional Course Lecture Book*,
https://doi.org/10.1007/978-3-662-56127-0_23

has with this specific problem in this specific population has been summarised in this chapter.

23.1.1 The Technical and Physical Demands of Ballet

What makes ballet different from dance and from sport is that these athletes are foremost artists and performers for which they trained and formed their body through more than a decade of specific training to fulfil the specific demands. The basis is that all movement is performed with an externally rotated hip and foot position, which is called 'en dehors'. That means that jumps, landings and turns start and end in that position.

There are five classical foot positions (Fig. 23.1), the most common are first, second, fourth and fifth.

Secondly, female dancers dance on point shoes (Fig. 23.2).

Fig. 23.2 Pas de deux, with female dancer on pointe

The point shoe enables them to balance on the small surface at the top of the shoe which is very flexible at the back and more rigid at the front. The shoe does not relieve pressure, but creates a small even end, so they can be on point (maximal plantarflexion of the foot with the weight on the distal phalanges as shown in Fig. 23.3).

Fig. 23.3 X-ray left foot on pointe

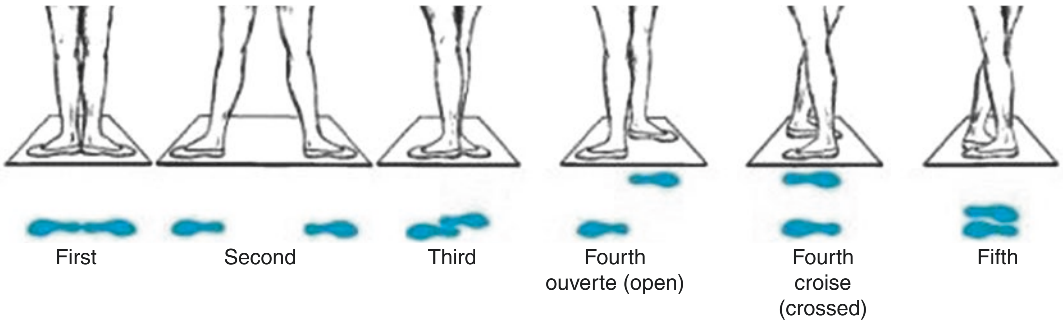


Fig. 23.1 The five classical foot positions

Fig. 23.4 Grand jeté

This and numerous jumps and lifts day in day out (Fig. 23.4) ask their toll on the dancer's body.

This position was historically created from the time of Louis XIV (the Sun King) as to achieve an aesthetically more pleasing position to lengthen the leg and foot. The actual point shoe with its blocked toes was introduced around 1880 in St. Petersburg during the period of Petipa [1].

Long and hard exercise and training are required for artistic top performances under the scrutiny of the audience and the stress of the media. Dance requires maximal propriocepsis and coordination, extreme precision and fine motor control, combined with stamina and perseverance.

Hypermobility is an asset and a prerequisite for dancers, who usually score 5 or more points of the nine Beighton criteria.

As a general principle of treatment, the physician must respect the 'passion' of the dancer and never give an injured dancer the advice to stop dancing.

23.1.2 Dance Injury Incidence

Injuries to dancers often result from overuse. Constant repetition of prescribed movements required by the practice or dance can wear body parts down to the point they give way. The reported injury incidence is 1.29 injury per 1000 dance hours [2]. An approach to dealing with the available time and costs is looking at severity versus likelihood. This ranges from more likely to occur such as cuts and bruises, which are less significant, to more unlikely injuries such as anterior cruciate ligament injuries that have a more severe impact. In professional football, prospective data registration shows that in general 12% of the team is not available during the season due to injury [3]. No data is available for ballet, as a prospective data registry does not yet exist for professional dance companies.

Table 23.1 Dance injuries reported per year in a professional dance company and at a pre-professional level

Anatomic location	Professional ballet dancer (%)	Dance student (%)
Neck	23	11
Shoulder	15	8
Arms/hands	10	5
Thoracic spine	11	7
Lumbar spine	47	42
Pelvis	15	16
Thigh	14	15
Lower leg	26	23
Knees	32	30
Ankles	42	30
Feet	33	21

Most injuries are caused by repetitive movement, and a smaller amount of injuries are caused by direct or indirect trauma. In a professional dance company, 40% of the injuries are traumatic injuries. In male dancers, this traumatic injury rate is higher; nearly 50% of their overall injury rate is traumatic of origin [2]. Very little has been published on when to operate on professional dancers and the outcome.

Because the lower extremity is the most often affected (Table 23.1), the current chapter focuses on these injuries.

23.2 Injury of the Lower Extremity in Ballet

23.2.1 Foot and Ankle Injuries

The intensity of training and performance with the additional high prevalence of nutritional problems creates an optimal environment for injuries to the foot and ankle in dance and ballet [4]. Stress injuries are frequently encountered and may be overlooked or 'hidden' by dancers reluctant to miss important career opportunities, and this may lead to the development of stress fractures [5]. Conservative management is certainly the optimal management for the treatment of a stress response to the metatarsals and navicular, but this may require a protracted period of time with adjusted load-bearing and avoidance of dance for up to 3 months in order to prevent progression to a fracture. Some advocate the use of bone stimulators such as low-intensity pulsed ultrasound (LIPUS) and Exogen, but a full medical workup for bone health is imperative as low vitamin D3 is not an uncommon finding [5]. The timing of when it is safe to return to dance is difficult to determine as MRI findings may remain abnormal for several months, and it is probably best to adjust according to symptoms and clinical findings – there is little evidence in the literature to guide on this, and a high rate of non-union (26% in the navicular) is reported without cast immobilisation and strict adherence to non-weight-bearing [6]. CT scan at 3 months may indicate healing of a fracture, but for the navicular, this may be far from normal for even a year, if ever [7].

Stress fractures that fail to respond to conservative treatment after 6–12 weeks may require surgical fixation. There is one report of 2 s metatarsal base stress fractures in ballet dancers healing with extracorporeal shockwave therapy [8]. Internal fixation with or without bone grafting is the more usual approach for the treatment of these and navicular fractures, whilst intramedullary screw fixation of fifth metatarsal fractures has a high incidence of union and return to sporting activities although little is written about this in the dance population [9–15].

Acute injuries to the midfoot with subtle instability of the Lisfranc joints may be difficult to assess. A high index of suspicion is required in order to avoid misdiagnosis of a midfoot 'sprain'. The plantar ecchymosis sign is said to be pathognomonic, and weight-bearing radiographs may be difficult to obtain initially because of pain [16]. A delayed examination with full single-stance weight-bearing radiographs (Stork views) may be more helpful 5–10 days later, and this may demonstrate subluxation of the Lisfranc joints. Injuries with more than 2 mm displacement require surgical fixation [17, 18].

Posterior ankle impingement (PAI) pain is a problem particularly encountered in ballerinas because of the requirement to perform en pointe. PAI may be secondary to an os trigonum or from flexor hallucis longus (FHL) tenosynovitis and associated tendinosis in 63–85% of cases [19–21]. FHL problems frequently occur due to irritation from the os trigonum or from a tight neck in the retinaculum surrounding the FHL leading to a 'ratcheting' effect, which may be palpable and even audible. Physiotherapy to improve strength and also posture and stability may eradicate symptoms, and an ultrasound-guided injection may help alleviate symptoms [22–24]. However, ultimately if pain persists, surgery is indicated [25–27]. Posterior ankle arthroscopy for excision of an os trigonum and/or release of the FHL has a high success rate and also has a quicker return to dance when compared with open surgical procedures [28].

Ankle instability following acute lateral ligament injury may occur in 15–20% of individuals. Functional rehabilitation is supported by the literature except in elite athletes or when there is gross clinical laxity with rupture of both the anterior talo-fibular ligament (ATFL) and calcaneofibular ligament (CFL), in which case acute surgical reconstruction may be considered [29]. The problem arises in dancers who frequently have a degree of generalised joint laxity and a significantly lax anterior draw test. Careful comparison with the contralateral ankle is required, and assessment at 5–10 days following acute injury is preferred [30–32]. The more common presentation is chronic instability with a history of repeated giving-way and inability to dance despite physiotherapy strengthening and proprioceptive training. Surgical management with a modified Broström-Gould repair provides a high success rate in stabilising the ankle allowing return to dance with minimal restriction in range of movement post-operatively [29]. Recent reports have suggested encouraging results using arthroscopic and percutaneous techniques, but routine use of augmentation techniques has not yet been justified [33–35].

The forefoot remains a common region for injury and complaints. Whilst hallux valgus deformity may frequently be treated nonoperatively until after retirement from professional dance, surgical treatment of hallux rigidus has a high success rate with open or minimally invasive cheilectomy [36, 37]. Ultrasound-guided injection for Morton's neuroma may provide complete symptomatic relief in up to 87% of patients, although only 31% may continue to have a good result at 2 years. Surgical excision may be considered if symptoms persist [38]. Freiberg's disease usually affects the second metatarsal and, less frequently, the third and is more commonly encountered in adolescent girls [39]. The majority of symptoms will settle with off-loading the affected metatarsal head, but it may take many months or even a year before full dance may recommence, necessitating careful explanation to the patient and parents. Occasionally, surgery is required if fragmentation of the metatarsal head occurs [40].

23.2.2 Knee Injuries

In a professional dance company, knee injuries are common. In a season, one-third of the dancers have missed class or could not perform due to knee complaints. The majority of these are overuse injuries such as patellofemoral complaints, iliotibial band injury, prepatellar bursitis and patella tendinopathy [1].

A professional dancer will not have knee complaints in the same manner as a beginning or pre-professional ballet dancer, where technique issues due to a forced turnout caused by lack of external hip rotation will give rise to 'screwing your knees'. This refers to forcing the knees in an externally rotated position by locking the feet in hyperpronation. Due to the tough selection process, dancers who do not have a sufficient turn out or have found a way to deal with this on a daily basis will not have reached the professional ranks.

Two knee injuries require surgery frequently: traumatic meniscal lesions and knee instability due to anterior cruciate ligament (ACL) rupture [41].

Classical ballet has been shown to have a higher chance of anterior cruciate ligament rupture than contemporary dance, probably due to the higher number of jumps and landing in en dehors with the knee in valgus. More than 90% of the reported ACL injuries in ballet occurred at the landing on one leg after a jump. Seventy-five percent happened during the second half of the performance or at the end of the day. The incidence rate of 3.2 symptomatic ACL ruptures per 100,000 dance-working hours is nearly as high as the well-recognised high risk of ACL injury in professional skiers [41].

A more frequent knee injury requiring surgery is the traumatic meniscal lesion. No true incidence for meniscal injury is known for ballet. This injury which can give clear physical impairment such as a locked knee disabling the dancer to weight-bear or stretch the knee to full extension or hyperextension will not be missed primarily. Meniscal injuries that give less clear physical impairment but can give rise to pain in the joint line or swelling after dancing can be initially difficult to diagnose. In a classic textbook about 'disease and injuries of ballet dancers', Eivind Thomasen describes 73 meniscal injuries during his working life whilst treating 750 dancers in this period spanning more than 25 years [1]. We have learned from prospective data registries in football players that lateral meniscal lesions need longer time to recover than medial meniscectomy before return to play is feasible. A multidisciplinary approach is essential in dealing with these work-threatening injuries, starting with prompt evaluation by the company physiotherapist, swift referral to a trained sport orthopaedic surgeon and a combined rehabilitation programme in close contact and cooperation with the companies' director and ballet masters.

23.2.3 Hip Injuries

Ballet exposes the hip joint to repetitive loading in extreme ranges of movement and may therefore predispose to hip pain and injury. A recent survey amongst retired professional ballet dancers in the UK showed that over one-third (36%)

retired from ballet due to musculoskeletal injury [42]. Of this subgroup, the most common injury causing dancers to retire was hip pain. Furthermore, 91% of all respondents reported muscle and joint pain post-retirement, with the hip being one of the most affected joints.

Due to the supra-physiological hip range of motion required for ballet dancing, femoroacetabular subluxation and femoroacetabular impingement (FAI) are frequently found. Femoroacetabular subluxation or instability is predominantly caused by soft tissue laxity of the musculotendinous structures around the hip, hip capsule laxity, labral tears, ligamentum teres tears and/or a bony morphology with (mild) acetabular dysplasia. FAI is normally caused by excessive acetabular coverage or acetabular retroversion (pincer morphology) or a non-spherical femoral head (cam morphology). However, FAI can occur with normal bony hip anatomy in ballet dancers due to the excessive range of motion experienced [43]. This was shown in a study of 11 dancers who performed six different dancing movements (arabesque, développé devant, développé a la seconde, grand écart facial, grand écart latéral and grand plié) which were registered by a Vicon motion capture system [43]. The hip joint kinematics were then applied to reconstructed 3D models of acquired MRI scans in order to simulate the range of motion and congruency of the hip joint.

Four dancing movements développé a la seconde, grand écart facial, grand écart latéral and grand plié induced both impingement and subluxation of up to 5 mm, resulting in significant stress in the hip joint. More than 80% of the dancers' hips showed cartilage thinning and degenerative labral lesions, with participants having an average age of only 25 years. Interestingly, the location of these pathological findings corresponded to the computed zones of impingement in the superior and postero-superior quadrant of the acetabulum. Despite a certain level of uncertainty resulting from the segmented images and simulated range of motion, it seems that a hip with normal anatomy can impinge in extreme positions and become incongruent resulting in translation/subluxation within the joint.

A study comparing 30 symptomatic and asymptomatic female ballet dancers with 14 asymptomatic non-dancing women showed a significantly higher rate of acetabular cartilage lesions >5 mm as quantified on MRI in the dancer's hips (29% versus 7%) [44]. Also in this study, the lesions were located at the superior side of the acetabulum. Labral lesions were also common among the dancers and more prevalent than in the controls, whilst morphological characteristics associated with FAI such as cam or pincer morphology were rare in the dancers [44]. Interestingly, they also obtained MRI of the dancer's hip in the split position (grand écart latéral) and observed a mean femoroacetabular subluxation of 2 mm, corresponding to the findings of the simulated motions as described above. In an additional study using the same cohort, no correlation between the imaging findings and pain in the dancers was found [45]. Mitchell et al. used radiographs in the split position to study the degree of subluxation [46]. They found that an increasing alpha angle (more femoral head asphericity) increased the magnitude of subluxation in men and women, as well as increasing severity of acetabular dysplasia in men [46]. The split position also generates higher peak pressures to the cartilage and labrum, as shown by finite element analysis [47].

Mayes et al. studied 49 male and female current and retired ballet dancers and compared them with 49 age- and sex-matched non-dancing athletes [48–50]. They showed that the prevalence of cartilage defects as quantified on MRI was 61% in the ballet dancers. However, this percentage was not different from the non-dancing athletes [48]. Labral tears were also a common finding with a prevalence of 65% in ballet dancers but not different from the non-dancing athletes [49]. Also in this study, no relationship between labral tears and hip pain was found. Interestingly, ligamentum teres tears were more frequently seen in ballet dancers with a prevalence of 55% as compared to 22% in the non-dancing athletes [50]. As the ligamentum teres functions as one of the hip stabilisers, micro-instability of the hip due to extreme range of motion and/or secondary to FAI might be a cause of the higher rate of ligamentum teres tears in ball ballers should be ballet dancers.

In summary, ballet dancers require extreme ranges of motion in their hips. The prevalence of cam and pincer morphology is lower in dancers than reported in other athletes [51]. However, due to a lack of prospective data, it is unknown whether they do not develop this morphology during adolescence like other athletes or if it is a natural selection as cam and pincer morphology limit the range of motion [52–54]. Despite these findings, it is known that dancers experience FAI with a normal hip anatomy at the end points of their range of motion during different dancing movements. At the locations where this impingement occurs, the labrum is compressed, and higher stresses on the cartilage are placed, especially at the superior part of the acetabulum. During the end points of the range of motion, subluxation also occurs as shown by multiple studies [43, 44, 46, 47]. Various factors might cause the subluxation such as secondary to FAI, dysplasia, capsular laxity and ligamentum teres tears. Ballet dancers have a higher rate of chondral and labral injury than the general population, but these injuries are probably similar to non-dancing athletes. To date, the clinical relevance of labral and ligamentum tears found on imaging is unknown.

23.3 State-of-the-Art Treatment

Any treatment of a dancer should be selected with their artistic background in mind, in which the body is the tool to express the part or roll they are performing. The art of dance is their life, and all efforts should be directed to performing and getting back on stage as safely and as quickly as possible. The team effort has to council and inform the dancer of all the possibilities and the pros and cons of operative treatment.

Assessment of the dancer's technique and style is necessary in order to understand the mechanism of injury and to successfully work towards preventing reinjury.

The role of a company physiotherapist is continuous screening, guiding and treating injuries. The close collaboration with an orthopaedic surgeon is essential to address urgent traumatic lesions but also to discuss when continuing with an overuse injury is not tolerable anymore and the timing for surgery should be discussed.

Recognising is step one, but deciding when to operate or not to operate is maybe even more essential. Dancers have an uncanny ability to tell you the exact diagnosis but will also remind you what your surgery has altered when they are recuperating and performing again.

23.3.1 Foot and Ankle Injury Treatment

Overall, despite the high frequency of foot and ankle conditions in ballet, most may be treated nonoperatively with careful management of load and prevention of reinjury through strength and proprioceptive training whilst maintaining realistic expectations of time to return to dance. Certain acute injuries require early accurate diagnosis and treatment in order to avoid long-term problems, and reassessment at 5–10 days following an injury may be useful, and even a further re-evaluation a week later may be justified to ensure optimal management.

23.3.2 Knee Injury Treatment

As for any injury, operative treatment is only one side of the coin, as a perfect operation without proper and well-managed rehabilitation will have a poorer outcome than with a multidisciplinary approach.

This is very clearly true for ACL surgery. After prompt and adequate diagnosing of an ACL tear with the possible comorbidity such as other ligamentous lesions, meniscal damage and chondral damage, counselling is of the utmost importance. The dancer should be aware of the impact of the injury to his knee at short term and for the future at a longer time span. The dancer is at an increased risk of re-rupture of up to 25%, depending on age and gender, and an increased risk (5% in the next 5 years) for rupturing his ACL in the noninjured knee. Little is known of the return to dance rate although the literature shows percentages equal to professional football players of 90–95% [18]. An ACL-injured knee has a tenfold increased risk of developing radiological osteoarthritis in 10 years post-trauma. Unfortunately, this risk is not yet lowered by ACL reconstruction.

ACL reconstruction, however, will reduce the risk of consecutive meniscal lesions and will create a stable knee, without giving-way moments in more than 90% of the procedures. Essential for a ballet dancer is a stable knee that does not limit knee flexion and above all does not decrease the pre-trauma extension or hyperextension. Hamstring autograft has lower comorbidity for anterior knee pain than bone-patellar tendon-bone autograft and is the author's preferred choice for this specific artist.

23.3.3 Hip Injury Treatment

Both nonoperative and operative treatments have an important role in returning injured ballet dancers to their previous level. The field of hip arthroscopy is quickly evolving. Hip arthroscopy in ballet dancers can be considered for ballet dancers with cam and/or pincer morphology, labral tears, ligamentum teres tears or rupture, capsular laxity and recurrent anterior hip dislocation. Except a case report for recurrent anterior hip dislocation, there are no studies available that specifically investigated surgery in ballet dancers.

In general, cam and/or pincer morphology with or without labral pathology can be addressed similar to other athletes. Indications include prolonged hip pain together with the presence of cam and/or pincer morphology with or without labral tears not responding to nonoperative management [55]. The main contraindication is advanced osteoarthritis of the hip.

To date, there are no studies available that directly compare nonoperative versus operative management in a blinded randomised setting. A systematic review on surgical management for FAI in athletes included nine case series [56]. A total of 440 athletes were analysed who were predominantly male with a mean age of 26 years in which cam morphology was most frequently addressed. Of these athletes, 92% returned to sports, and 88% returned to their pre-injury level of activity at a minimum of 6 months follow-up. These percentages were somewhat higher for professional athletes and somewhat lower for recreational athletes. Another systematic review which also included nonathletic populations concluded that the current literature shows improvement in symptoms and bone shape after FAI surgery (either open or arthroscopically), but all these studies have a high risk of bias, and no blinded comparative studies with conservative therapy or sham surgery were available [57].

A systematic review on ligamentum teres injuries found only case reports or small case series eligible for inclusion [58]. People who were operated for this condition were predominantly women (80%), and surgery consisted in almost all cases of debridement of the ligamentum teres. Of the included studies in this systematic review, six cases of ligamentum teres reconstruction were described using a synthetic knee medial collateral ligament, a semitendinosus autograft or an iliotibial band autograft. Five out of these six patients were able to return to their preoperative level of activities or sports. Both debridement and reconstruction showed a short-term relief of pain, but long-term results are unknown.

A case of recurrent anterior hip dislocation was described which, after prompt reduction, was treated arthroscopically with anterior suture capsular plication, repair of an anterior superior labral tear and a partial release of the iliopsoas [59]. For atraumatic hip instability, thermal capsulorrhaphy in conjunction with partial labral resection has been described in some professional athletes, but long-term outcomes are unknown [60].

Thus, good outcomes have been described for surgery to address cam morphology, pincer morphology and/or labral tears in athletes. However, caution is warranted as no randomised trials are available to date. When surgery is considered in ballet dancers, one must be aware that FAI and FAS can still occur when the 'normal' anatomy is restored. There is a lack of literature on other indications for surgical treatment in ballet dancers such as ligamentum teres tears, capsular laxity and hip (sub)luxation.

23.4 Future Treatment Options

Combining knowledge from different disciplines that are involved in treating dancers is important to enhance mutual knowledge and improve outcome. Professional dancers are rare, and improving our knowledge on incidence, conservative and operative treatment impact and outcome of ballet injuries still has a long way to go. A central international registry of ballet injuries would be an important step, which would enable dance orthopaedic surgeons to learn from not only their own experience but from a host of other engaged and professional colleagues.

23.5 Take-Home Message

In dance orthopaedics, being aware of orthopaedic techniques is not enough. A thorough understanding of dance styles, the specific requirements and ballet technique is a necessity. A dancer is not just another athlete; they are artists in their own right.

References

1. Thomase E. Disease and injuries of ballet dancers. Aarhus: Universitetsforlaget I Aarhus; 1982. isbn:87-504-0480-6
2. Smith PJ, Gerrie BJ, Varner KE, McCulloch PC, Lintner DM, Harris JD. Incidence and prevalence of musculoskeletal injury in ballet: a systematic review. Orthop J Sports Med. 2015;3(7):2325967115592621.
3. Ekstrand J, Hagglund M, Walden M. Injury incidence and injury patterns professional football: the UEFA injury study. Br J Sports Med. 2011;45(7):553–8.
4. Sousa M, Carvalho P, Moreira P, Teixeira VH. Nutrion and nutritional issues for dancers. Med Probl Perform Art. 2013;28(3):119–23. Review
5. Wolman R, Wyon MA, Koutedakis Y, Nevill AM, Eastell R, Allen N. Vitamin D status in professional ballet dancers: winter vs. summer. J Sci Med Sport. 2013;16(5):388–91.
6. Khan K, Fuller P, Brukner P, Kearney C, Burry H. Outcome of conservative and surgical management of navicular stress fractures in athletes. Am J Sports Med. 1992;20:657–66.
7. Torg JS, Pavlov H, Cooley LH, et al. Stress fractures of the tarsal navicular. A retrospective review of twenty-one cases. J Bone Joint Surg Am. 1982;64:700–12.
8. Silk ZM, Alhuwaila RS, Calder JD. Low-energy extracorporeal shock wave therapy to treat lesser metatarsal fracture nonunion: case report. Foot Ankle Int. 2012;33(12):1128–32. https://doi.org/10.3113/FAI.2012.1128.
9. Bica D, Sprouse RA, Armen J. Diagnosis and management of common foot fractures. Am Fam Physician. 2016;93:183–91.
10. Boutefnouchet T, Budair B, Backshayesh P, Ali SA. Metatarsal fractures: a review and current concepts. Trauma. 2014;16:147–63.
11. Fitch KD, Blackwell JB, Gilmour WN. Operation for non-union of stress fracture of the tarsal navicular. J Bone Joint Surg (Br). 1989;71-B:105–10.
12. Hong CC, Pearce CJ, Ballal MS, Calder JD. Management of sports injuries of the foot and ankle: an update. Bone Joint J. 2016;98-B(10):1299–311. Review
13. Roche AJ, Calder JD. Treatment and return to sport following a Jones fracture of the fifth metatarsal: a systematic review. Knee Surg Sports Traumatol Arthrosc. 2013;21:1307–15.
14. Saxena A, Fullem B. Navicular stress fractures: a prospective study on athletes. Foot Ankle Int. 2006;27:917–21.
15. Thevendran G, Deol RS, Calder JD. Fifth metatarsal fractures in the athlete: evidence for management. Foot Ankle Clin. 2013;18(2):237–54. https://doi.org/10.1016/j.fcl.2013.02.005. Review
16. Ross G, Cronin R, Hauzenblas J, Juliano P. Plantar ecchymosis sign: a clinical aid to diagnosis of occult Lisfranc tarsometatarsal injuries. J Orthop Trauma. 1996;10:119–22.
17. Deol RS, Roche A, Calder JD. Return to training and playing after acute lisfranc injuries in elite professional soccer and rugby players. Am J Sports Med. 2016;44:166–70.
18. Eleftheriou K, Rosenfeld PF, Calder JD. Lisfranc injuries in sports – an update and algorithm for treatment. Knee Surg Sports Traumatol Arthrosc. 2013;21(6):1434–46.
19. Hamilton WG, Geppert MJ, Thompson FM. Pain in the posterior aspect of the ankle in dancers: differential diagnosis and operative treatment. J Bone Joint Surg Am. 1996;78:1491–500.
20. Roche A, Calder JD, Williams L. Posterior ankle impingement in dancers and the athlete. Foot Ankle Clin. 2013;18(2):301–18.
21. Scholten PE, Sierevelt IN, van Dijk CN. Hindfoot endoscopy for posterior ankle impingement. J Bone Joint Surg Am. 2008;90:2665–72.
22. Albisetti W, Ometti M, Pascale V, et al. Clinical evaluation and treatment of posterior impingement in dancers. Am J Phys Med Rehabil. 2009;88:349–54.
23. Messiou C, Robinson P, O'Connor PJ, et al. Subacute posteromedial impingement of the ankle in athletes: MR imaging evaluation and ultrasound guided therapy. Skelet Radiol. 2006;35:88–94.
24. Robinson P, Bollen SR. Posterior ankle impingement in professional soccer players: effectiveness of sono-

graphically guided therapy. AJR Am J Roentgenol. 2006;187:W53–8.

25. Calder J, Pearce C, Sexton S. Return to training and playing after posterior ankle arthroscopy for posterior impingement in elite professional soccer. Am J Sports Med. 2010;38(1):120–4.
26. van Dijk CN, Scholten PE, Krips R. A 2-portal endoscopic approach for diagnosis and treatment of posterior ankle pathology. Arthroscopy. 2000;16:871–6.
27. Galla M, Lobenhoffer P. Technique and results of arthroscopic treatment of posterior ankle impingement. Foot Ankle Surg. 2011;17:79–84.
28. Ballal MS, Roche A, Brodrick A, Williams RL, Calder JD. Posterior endoscopic excision of Os trigonum in professional national ballet dancers. J Foot Ankle Surg. 2016;55(5):927–30.
29. Guillo S, Bauer T, Lee J, Takao M, Kong S, Stone J, Mangone P, Malloy A, Perera A, Pearce C, Michels F, Tourne Y, Ghorbani A, Calder J. Consensus in chronic ankle instability: aetiology, assessment, surgical indications and place for arthroscopy. Orthop Traumatol Surg Res. 2013;99(8 Suppl):S411–9.
30. Friden TZR, Lindstrand A, et al. A stabilometric technique for evaluation of lower limb instabilities. Am J Sports Med. 1989;17:118–22.
31. Lentell GKL, Walters MR. The relationship between muscle function and ankle instability. J Orthop Sports Phys Ther. 1990;11:605–11.
32. Tourné Y, Besse JL, Mabit C. Sofcot. Chronic ankle instability. Which tests to assess the lesions? Which therapeutic options? Orthop Traumatol Surg Res. 2010;96(4):433–46. https://doi.org/10.1016/j.otsr.2010.04.005
33. Glazebrook M, Stone J, Matsui K, Guillo S, Takao M, Batista J, Bauer T, Calder J, Choi J, Ghorbani A, Molloy A, Nery C, Ozeki S, Pearce C, Perera A, Pereira H, Pijnenburg B, Raduan F, Stone JW, Tourne Y. Percutaneous ankle reconstruction of lateral ligaments (Perc-anti RoLL). Foot Ankle Int. 2016;37(6):659–64.
34. Guillo S, Takao M, Calder J, Karlson J, Michels F, Bauer T, Ankle Instability Group. Arthroscopic anatomical reconstruction of the lateral ankle ligaments. Knee Surg Sports Traumatol Arthrosc. 2016;24(4):998–1002.
35. Takao M, Matsui K, Stone JW, Glazebrook MA, Kennedy JG, Guillo S, Calder JD, Karlsson J. Ankle instability group. Arthroscopic anterior talofibular ligament repair for lateral instability of the ankle. Knee Surg Sports Traumatol Arthrosc. 2016;24(4):1003–6.
36. van Dijk CN, Veenstra KM, Nuesch BC. Arthroscopic surgery of the metatarsophalangeal first joint. Arthroscopy. 1998;14(8):851–5.
37. Mulier T, Steenwerckx A, Thienpont E, Sioen W, Hoore KD, Peeraer L, Dereymaeker G. Results after cheilectomy in athletes with hallux rigidus. Foot Ankle Int. 1999;20(4):232–7.
38. Grice J, Marsland D, Lomax A, Ballal M, Lee J, Mitchell M, Calder J. Efficacy of foot and ankle corticosteroid injections. Foot Ankle Int. 2016;38(1):8–13.
39. Mizel MS, Yodlowski ML. Disorders of the lesser metatarsophalangeal joints. J Am Acad Orthop Surg. 1995;3(3):166–73.
40. Sproul J, Klaaren H, Mannarino F. Surgical treatment of Freiberg’s infraction in athletes. Am J Sports Med. 1993;21(3):381–4.
41. Meuffels DE, Verhaar JA. Anterior cruciate ligament injury in professional dancers. Acta Orthop. 2008;79(4):515–8.
42. Smith TO, de Medici A, Oduoza U, Hakim A, Paton B, Retter G, et al. National survey to evaluate musuloskeletal health in retired professional ballet dancers in the United Kingdom. Phys Ther Sport. 2017;23:82–5.
43. Charbonnier C, Kolo FC, Duthon VB, Magnenat-Thalmann N, Becker CD, Hoffmeyer P, et al. Assessment of congruence and impingement of the hip joint in professional ballet dancers: a motion capture study. Am J Sports Med. 2011;39(3):557–66.
44. Kolo FC, Charbonnier C, Pfirrmann CW, Duc SR, Lubbeke A, Duthon VB, et al. Extreme hip motion in professional ballet dancers: dynamic and morphological evaluation based on magnetic resonance imaging. Skelet Radiol. 2013;42(5):689–98.
45. Duthon VB, Charbonnier C, Kolo FC, Magnenat-Thalmann N, Becker CD, Bouvet C, et al. Correlation of clinical and magnetic resonance imaging findings in hips of elite female ballet dancers. Arthroscopy. 2013;29(3):411–9.
46. Mitchell RJ, Gerrie BJ, McCulloch PC, Murphy AJ, Varner KE, Lintner DM, et al. Radiographic evidence of hip microinstability in elite ballet. Arthroscopy. 2016;32(6):1038–44.e1.
47. Assassi L, Magnenat-Thalmann N. Assessment of cartilage contact pressure and loading in the hip joint during split posture. Int J Comput Assist Radiol Surg. 2016;11(5):745–56.
48. Mayes S, Ferris AR, Smith P, Garnham A, Cook J. Professional ballet dancers have a similar prevalence of articular cartilage defects compared to age- and seks matched non-dancing athletes. Clin Rheumatol. 2016;35(12):3037–43.
49. Mayes S, Ferris AR, Smith P, Garnham A, Cook J. Similar prevalence of acetabular labral tear in professional ballet dancers and sporting participants. Clin J Sport Med. 2016;26(4):307–13.
50. Mayes S, Ferris AR, Smith P, Garnham A, Cook J. Atraumatic tears of the ligamentum teres are more frequent in professional ballet dancers than a sporting population. Skelet Radiol. 2016;45(7):959–67.
51. Frank JM, Harris JD, Erickson BJ, Slikker W III, Bush-Joseph CA, Salata MJ, et al. Prevalence of femoroacetabular impingement imaging findings in asymptomatic volunteers: a systematic review. Arthroscopy. 2015;31(6):1199–204.
52. Agricola R, Heijboer MP, Ginai AZ, Roels P, Zadpoor AA, Verhaar JA, et al. A cam deformity is gradually acquired during skeletal maturation in adolescent and young male soccer players: a prospective study with minimum 2-year follow-up. Am J Sports Med. 2014;42(4):798–806.

53. Agricola R, Weinans H. What causes cam deformity and femoroacetabular impingement: still too many questions to provide clear answers. Br J Sports Med. 2016;50(5):263–4.
54. Roels P, Agricola R, Oei EH, Weinans H, Campoli G, Zadpoor AA. Mechanical factors explain development of cam-type deformity. Osteoarthr Cartil. 2014;22(12):2074–82.
55. Griffin DR, Dickenson EJ, O'Donnell J, Agricola R, Awan T, Beck M, et al. The Warwick agreement on femoroacetabular impingement syndrome (FAI syndrome): an international consensus statement. Br J Sports Med. 2016;50(19):1169–76.
56. Alradwan H, Philippon MJ, Farrokhyar F, Chu R, Whelan D, Bhandari M, et al. Return to preinjury activity levels after surgical management of femoroacetabular impingement in athletes. Arthroscopy. 2012;28(10):1567–76.
57. Fairley J, Wang Y, Teichtahl AJ, Seneviwickrama M, Wluka AE, Brady SR, et al. Management options for femoroacetabular impingement: a systematic review of symptom and structural outcomes. Osteoarthr Cartil. 2016;24(10):1682–96.
58. de Sa D, Phillips M, Philippon MJ, Letkemann S, Simunovic N, Ayeni OR. Ligamentum teres injuries of the hip: a systematic review examining surgical indications, treatment options, and outcomes. Arthroscopy. 2014;30(12):1634–41.
59. Epstein DM, Rose DJ, Philippon MJ. Arthroscopic management of recurrent low energy anterior hip dislocation in a dancer: a case report and review of literature. Am J Sports Med. 2010;38(6):1250–4.
60. Philippon MJ. The role of arthroscopic thermal capsulorrhaphy in the hip. Clin Sports Med. 2001;20(4):817–29.

Erratum to: Decision-Making in Anterior Shoulder Instability

Michel P. J. van den Bekerom, Derek F. P. van Deurzen, Karin M. C. Hekman, Olivier Verborgt, Klaus Bak, Marco Brioschi, Chiara Fossati, Riccardo Compagnoni, Alessandra Menon, Hassanin Alkaduhimi, and Pietro Randelli

Erratum to:
Chapter 16 in: G.M.M.J. Kerkhoffs et al. (eds.), *ESSKA Instructional Course Lecture Book*, https://doi.org/10.1007/978-3-662-56127-0_16

The spelling of the author's name was wrong in chapter 16. The correct name is: Marco Brioschi

---

The updated original online version for this chapter can be found at https://doi.org/10.1007/978-3-662-56127-0_16

M. P. J. van den Bekerom (✉) · D. F. P. van Deurzen
Orthopaedic Department, Onze Lieve Vrouwe Gasthuis, Amsterdam, The Netherlands

Orthopaedic Department, Jan van Goyen Medical Centre, Amsterdam, The Netherlands
e-mail: bekerom@gmail.com

K. M. C. Hekman
Interdisciplinary Center For Shoulder Rehabilitation, IBC Amstelland, Amstelveen, The Netherlands

Orthopaedic Department, Jan van Goyen Medical Centre, Amsterdam, The Netherlands

O. Verborgt
Orthopedic Department, AZ Monica, Deurne, Belgium

K. Bak
Aleris Hamlet Parken, Copenhagen, Denmark

M. Brioschi · C. Fossati · R. Compagnoni
A. Menon · P. Randelli
1st Department, Azienda Socio Sanitaria Territoriale Centro Specialistico Ortopedico Traumatologico Gaetano Pini-CTO, Milan, Italy

Department of Biomedical Sciences for Health, University of Milan, Milan, Italy

H. Alkaduhimi
Orthopaedic Department, Onze Lieve Vrouwe Gasthuis, Amsterdam, The Netherlands

G.M.M.J. Kerkhoffs et al. (eds.), *ESSKA Instructional Course Lecture Book*,
https://doi.org/10.1007/978-3-662-56127-0_24

Index

G.M.M.J. Kerkhoffs et al. (eds.), *ESSKA Instructional Course Lecture Book*,
https://doi.org/10.1007/978-3-662-56127-0