Stefano Zaffagnini
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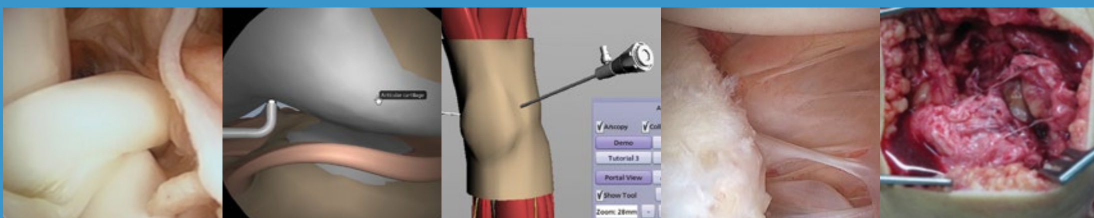

ESSKA Instructional Course Lecture Book

Amsterdam 2014

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EUROPEAN SOCIETY OF SPORTS TRAUMATOLOGY, KNEE SURGERY AND ARTHROSCOPY

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ISBN 978-3-642-53982-4 ISBN 978-3-642-53983-1 (eBook)
DOI 10.1007/978-3-642-53983-1
Springer Heidelberg New York Dordrecht London

Library of Congress Control Number: 2014937002

Printed on acid-free paper

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

Preface

Dear ESSKA-members,

We are gratified to offer you this instructional course book that represents a latest update on the current knowledge on knee surgery, sports traumatology and arthroscopy. The book includes the contents of all the instructional course lectures. We acknowledge the great efforts of the authors that allowed us to present this book at the 2014 ESSKA congress.

In the light of the educational aspirations of ESSKA, it is a great pleasure to share this knowledge with all of you.

Stefano Zaffagnini
Roland Becker
Gino M.M.J. Kerkhoffs
C. Niek van Dijk
João Espregueira Mendes

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Part I

Instructional Courses: Upper Limb

Osteoarthritis in Young Patients and Current Treatments

1

Roman Brzóska, Adrian Błasiak,
Polydoor E. Huijsmans, Anthony Miniaci,
Giuseppe Porcellini, Wojciech Solecki,
and Catherine van der Straeten

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1.1 Current Concepts on Shoulder Early Arthritis

Giuseppe Porcellini

The articular cartilage of the shoulder is not endowed with intrinsic repair abilities; therefore in the presence of diseases, like instability or cuff injury, even minor lesions may rapidly lead to early glenohumeral joint arthritis. The presence of cartilage lesions is not unusual even in young patients, and often they are found during arthroscopic procedures in several pathologic conditions. Less common conditions include glenoid dysplasia and osteochondritis dissecans. The varying thicknesses of the joint cartilage and the different resistance properties of the subchondral bone result in lesions of different depths and widths depending on the resistance offered by the

S. Zaffagnini et al. (eds.), *ESSKA Instructional Course Lecture Book*,
DOI 10.1007/978-3-642-53983-1_1, © ESSKA 2014

articular surface. Minor cartilage lesions associated with rotator cuff or glenohumeral ligament damage will induce topographically different types of stress on the various areas of the articular surface. Recent and older research findings showed in the shoulder as in the knee, a correlation between cartilage wear and lesion site and between site and symptoms. Several conservative options available to manage shoulder arthritis are directed to alleviate pain, reduce inflammation, and, especially, halt or at least slow down the evolution of arthritis. Such therapies entail changes in lifestyle and systemic and topical drug administration. Viscosupplementation using hyaluronic acid may constitute a helpful treatment option in patients who have shoulder osteoarthritis with an intact rotator cuff, while lesser satisfactory results have been showed in case of rotator cuff tears or advanced osteoarthritis. Several surgical options are available to manage primary shoulder arthritis, including simple arthroscopic joint debridement and more complex techniques such as resurfacing using the fascia lata or meniscus, osteochondral autologous transplantation, resurfacing arthroplasty, and total arthroplasty.

1.2 Articular Cartilage Injuries (Clinical Treatment Options and Basic Science)

Anthony Miniaci

1.2.1 Objectives

At the end of this presentation, the participant will be able to:

- Understand the history and evolution of the arthroscope in the treatment of cartilage disorders
- Identify the efficiency of the various surgical techniques used to treat chondral defects and arthrosis and indications for each
- Have an understanding of the “newer techniques” available for the treatment of cartilage lesions
- Identify the limitations and strengths of various techniques used to treat chondral pathology
- Evaluate basic science of cartilage transplantation and clinical application and determine the relevance to their own clinical practice

1.2.2 Introduction

- Degenerative OA results in loss of function and motion and pain.
- No cure available yet.

1.2.3 Articular Cartilage Basic Science

- Primary cell: chondrocyte
- Function: decreases friction, distributes load, and exhibits stress shielding of solid matric components
- Avascular, aneural, and alymphatic with minimal reparative potential
- Nourished by synovial fluid at surface and subchondral bone at base
- Composition: water > collagen > noncollagen protein > cells
- Four types of articular cartilage:
  - Hyaline cartilage
  - Fibroelastic cartilage
  - Fibrocartilage
  - Elastic cartilage
- Zones of articular cartilage:
  - Superficial zone: collagen parallel to surface
  - Transitional zone: random fibers
  - Deep zone: collagen perpendicular to surface
  - Tidemark (subchondral bone and cancellous bone)

1.2.4 Treatment Options

- Nonsurgical
  - Decreased activity
  - Weight loss
  - NSAIDs
  - Physiotherapy aids
  - Steroids, viscotherapy
  - Platelet-rich plasma

- Surgery
  - Realignment procedures
  - Replacement
  - Arthrodesis
  - Arthroscopy – growing area

1.2.5 Arthroscopy

- Least invasive surgical approach
- As adjuvant to realignment procedure

Types of Procedures

Historical: lavage, debridement, abrasion, carbon fiber

Current practice

- Microfracture
- Osteochondral autograft transfer system (OATS)/mosaicplasty
- Autologous chondrocyte implantation
- Focal resurfacing

1.2.5.1 Lavage

- Beneficial effects
- Removes all irritants and floating debris
- 74 % good results (Sprague et al. [1])

1.2.5.2 Debridement

- More aggressive than lavage.
- Removes loose chondral flaps.
- Beneficial effects (Magnuson [2]).
- Jackson demonstrated debridement better than lavage alone (Jackson et al. [3]).
- Generally good short-term results, 75 % at 2 years except:
  - Poor alignment
  - Duration of disease
  - Severe X-ray changes (Salisbury et al. [4]; Baumgartner et al. [5])

1.2.5.3 Abrasion Arthroplasty

- In conjunction to debridement
- Stimulates fibrocartilage repair
- Open chondroplasty
- Arthroscopic
- Limited usefulness with questionable biomechanical integrity of fibrocartilage
- Perhaps no benefit over debridement alone

1.2.5.4 Carbon Fiber Implantation

- Improves biochemical integrity of fibrocartilage repair
- Good early clinical results

1.2.5.5 Microfracture

- Used to treat small (<2 cm) symptomatic lesions.
- Creating a contained lesion is imperative to create a stable base for filling defect.
- Involves removing calcified cartilage layer and perforation of subchondral plate to recruit mesenchymal stem cells into lesion; these cells differentiate into fibrocartilage.
- Procedure success is correlated with formation of stable blood clot that maximally fills defect.

Outcomes

- JKS: 103 prospective patients with 6-year follow-up
  - Procedure found to be technically simple with minimal complications.
  - Defects <4 cm^2 tend to do better.
- Steadman et al. [6]: 75 knees with average 11-year follow-up
  - 80 % of patients rated themselves “improved.”
  - Multivariate analysis – age was a predictor of functional improvement.
- Mithoefer et al. [7]: 48 patients with 2-year follow-up
  - 67 % good to excellent results
  - Poor results correlated to:
    - BMI > 30
    - Poor fill on MRI
    - Symptoms >12 months
- Frank et al. [8]: 16 shoulders with average 28-month follow-up
  - Significant relief in pain and shoulder function with isolated, full-thickness chondral injuries (average 5.07 cm^2).
  - Three patients required subsequent procedures.
- Millett et al. [9]: 31 shoulders with average 47-month follow-up
  - Greatest improvement for smaller, isolated lesions of the humerus.
    - Worse results in patients with bipolar lesions
  - Six shoulders required additional surgery.

- Goyal et al. [10]: Systematic review of 15 level I and II studies
  - Patients with small lesions and low post-op demands had good clinical outcomes.
  - Beyond 5 years, failure after microfracture could be expected regardless of lesion size.
  - Younger patients had better clinical outcomes.

1.2.6 Osteochondritis Dissecans

Localized lesion characterized by separation of a segment of articular cartilage and its underlying subchondral bone.

1.2.6.1 Incidence

- Poorly understood entity with no universally accepted etiology. True incidence unknown as often spontaneous resolution without presentation. Multiple joints reported but knee accounts for 75 % [11].
- Male/female 2:1 [11].

1.2.6.2 Etiology

Two types based around physeal closure: juvenile OCD (5–15 year), adult OCD (15–50 year).

1.2.6.3 Causes

- *Trauma*
  - *Direct* **–** as the posterolateral portion of the medial femoral condyle is affected in 85 % of knees, direct trauma is unlikely [12].
  - *Indirect* **–** odd facet of the patella articulating with area [12] and/or impingement of tibial spine on area in internal rotation [13].
- *Ischemia*
- *Defects of Ossification: Due to MED* [14]
  Abnormal end-artery theory of subchondral bone susceptible to emboli and ischemia has been suggested [15]. Other studies found blood supply of subchondral femur rich in anastomoses and the histology of loose bodies and resected fragments to NOT demonstrate evidence of OCD [16].
- *Genetic*
  Suggested that OCD may represent a mild subgroup of epiphyseal dysplasia [17]
  - Associations with Perthes and achondroplasia.
  - Familial relationships have been recorded.
  - Ossification defects (juvenile OCD).

1.2.6.4 Presentation

Symptoms dependent on stage of lesion:

- Early – vague pain +/– swelling, activity related
- Late – if flap or loose body, catching and giving way
- Effusion, tenderness, and crepitus

1.2.6.5 Imaging

- Plain X-ray.
- Tc-99m scan – previously described to identify and follow the course/recovery of OCD [18].
- MRI now is the best modality for diagnosis and following progress of lesion.

MRI Classification

Stage I – thickening of articular cartilage and low signal change

Stage II – articular cartilage breached and low signal rim behind fragment

Stage III – articular cartilage breached and high signal changes behind fragment

Stage IV – loose body (Dipaola)

Arthroscopic Classification [19]

Grade I – depressed subchondral fracture

Grade II – OC fragment attached by an osseous bridge

Grade III – a detached non-displaced fragment

Grade IV – displaced fragment

1.2.6.6 Management

Controversy and confusion in literature are present as often works are based on small numbers, mix juvenile and adult cases, and few prospective trials have been performed.

Juvenile OCD

Lesions in knees with open physes usually heal with conservative treatment; those that do not are due to continued activity [18]. Ideal initial management is conservative.

Protected weight bearing/restriction of activities (90° casts), affected children often athletic and difficult to fully restrict. Try to avoid impact activities. Chances of success with nonoperative treatment decrease as time of physeal closure nears. 50 % of them will heal in 12 months.

Follow progress with serial MRI.

Indications for Operative Intervention

- Symptoms persist for 6–12 months despite adequate nonoperative treatment.
- Loose fragment.
- Progression of defect radiologically (MRI).
- Predicted physeal closure within 6–12 months.

Adult OCD

- Symptomatic lesions rarely heal with nonoperative measures.
- Lower tolerance for operative intervention after failure of conservative measures.

1.2.6.7 Operative Intervention

- Stabilization/re-fixation of fragment.
- Clanton II, III, and selected IV.
- Excision of fragment/reconstruction of OCD defect (Clanton Type II/III (IV)).
  - Principles: rigid fixation, enhance blood supply, reestablish congruency
  - Arthroscopic drilling is controversial for most lesions, but good results have been reported in Juvenile Clanton II lesions [20].
- *Internal fixation: open or arthroscopic*
  - Pins [21]
  - K-wires [18]
  - Herbert screws [22]
  - Biodegradable rods [23]
  - Corticocancellous bone pegs [24]
- *Additional bone grafting under articular cartilage (Anderson)*

OCD Defect

- Poor results after excising lesion and leaving defect [18].
- Fibrocartilage is biomechanically less resilient than articular cartilage predisposing to degenerative changes [25].

Fragment Fixation Technique [26]

(a) *Clanton and DeLee Type II and III Lesions*

- Unstable plugs that fail conservative management
- Can be used in prepubertal and postpubertal patients
- Theoretical considerations:
  1. Stabilize fragment with K-wire, and remove after fixed with one or two plugs.
  2. Drill holes – stimulates blood supply.
  3. Press fit 3.5 or 4.5 mm plugs results in stable fixation.
  4. Place peripheral plugs between native vascular bone and fragment so that healing of fragment can occur.
  5. Plug serves as a source of bone graft.
  6. Cartilage cap on “plug” restores articular surface so end result will have continuous articular cartilage surface.
  7. Central plug should be used for ultimate stability. This should be long enough to traverse OCD fragment into underlying vascular bone.

     Measure depth preoperatively.

     Ultimately provides blood supply – drilling, stability-interference fixation, bone grafting of fibrous layer, and congruent articular cartilage surface.

     Clinical results > 20 cases of OCD, 100 % healing rates, no additional fixation, return to activity and sports by 3–4 months, and complete healing by 6–9 months.
  8. *Type IV lesions*: where suitable for fixation- debride bed. Initial stabilization with K-wires is required before plug insertion.

(b) *Chronic Lesions*

Indications for treatment

- Symptomatic defect (trial of debridement)
- Stable knee
- Normal biomechanical alignment
- Minimal degenerative changes

Defect Reconstruction

- Large osteochondral grafts
- Autografts [27]
- Mosaic autografts [26, 28, 29]

- Allografts [30]
- Chondrocyte culture implantation – not as effective as a result of bone defect

1.2.7 Filling of Defect: Mosaicplasty Reconstruction

Technique – when fragment is lost, BE CAREFUL.

- Aim to recreate joint curvature and congruence.
- Fill defect with grafts from the periphery inwards. This allows for assessment of joint curvature and for central graft support.
- Central pegs will need to be longer to account for the greater height of curvature and depth of crater.
- Need to sit central plugs higher, since you are reconstructing both a bone and cartilage defect.
- If plugs in center are not higher, then reconstruction will be flat.
- Measure center of defect on MR preoperatively to determine size and length of plugs.
- Graft harvest from edge of the patellofemoral joint in both knees as necessary (10–12 4.5 mm grafts from each knee).

Postoperative Treatment

- Allow knee motion but strict non-weight bearing for 6 weeks.

Focal Traumatic Osteochondral Lesions

- Similar principles to OCD.
- In acute lesions can use plugs to fix osteochondral lesions.
- Femoral condyles the easiest.
- Tibial lesions difficult, not practical.
- Trochlear lesions usually require arthroscopy.

Donor Sites

- Plug harvest location is important.
- Keep out tibiofemoral joint.
- 5 mm on periphery of patellofemoral joint is optimal (less contact) and avoids reciprocal arthrosis.
- Large plugs, >5 mm fill incompletely, with fibrosis tissue.
- Causes reciprocal OA in areas of weight bearing contact.

Recipient Sites

- Hole preparation is crucial.
- Preserve bone stock, need stable construct.
- Drilling holes cause thermal necrosis.
- Dilating holes preserve bone stock and reduce thermal necrosis.
- Press fit plug to hole for stable construct.
- Bottom out hole to avoid subsidence and cyst formation.
- Fill defect with as much articular cartilage as possible, reduces percentage of fibrocartilage.
- Put plugs even with surrounding articular surface, too proud causes loosening and cyst formation, recessed covered with fibrocartilage.

Graft Harvest and Delivery

Harvest: do not use power trephine for harvest, causes cell necrosis. Multiple small plugs allow for better reconstruction of curved surface. After harvest inspect plug integrity, fractures, and obliquity. Measure depth of plug.

Plug delivery: press fit plugs, flush with surrounding articular surface, and bottom out hole; manual or light pressure only to insert plugs; impaction causes cell necrosis; reconstruct curved surfaces (center higher than periphery); tendency is for flat reconstructions.

Outcomes

- Histologically Type II collagen preserved; bone healing of plugs provides solid structure.
- Subchondral cyst formation is a concern.
- Hangody and Fules [31]: The largest series of mosaicplasties (831) for grade III or IV chondral lesions.
- Good to excellent results for 92 % femoral lesions, 87 % tibial lesions, and 79 % patellofemoral lesions.
- 80 % of second-look arthroscopies in 85 patients showed congruent articular cartilage surfaces and histologic evidence of transplanted hyaline cartilage.

- Ozturk et al. [32]: 19 patients with grade IV lesions.
- 85 % rate of good-to-excellent results at 32-month follow-up.
- MRI showed excellent fill and congruency < 1 mm without fissuring or delamination in 84 % of cases.
- Miniaci and Tytherleigh-Strong [26]: Mosaicplasty in unstable OCD lesions.
- Twenty knees were studied and scored clinically normal by 18 months.
- Serial MRI showed healing of osseous lesion by 6 months, continuous articular cartilage by 9 months.
- Kircher et al. [33]: OATS in the shoulder, seven patients at 9-year follow-up.
- Improved mean Constant scores; Lysholm score remained excellent.
- Significant progression of OA.
- Not related to size, number of plugs, or Constant score.
- MRI showed congruent joint surface on all except one shoulder.
- No further surgeries needed.

Outcomes Compared to Microfracture

- Krych et al. [34]: Level III, 96 patients at 5-year follow-up
  - Mosaicplasty and microfracture resulted in significantly improved general health and knee function scores.
  - Higher activity level post-op with mosaicplasty compared to microfracture.
- Gudas et al. [35]: Level I, 60 patients at 10.4-year follow-up
  - OATS technique allowed for higher rate of return to and maintenance of sports at pre-injury level compared with microfracture.
- Ideal patient is a young active patient with full-thickness chondral defect between 2 and 10 cm^2 that is surrounded by stable and healthy cartilage.
  - Useful in treating injuries that have failed debridement
  - Best suited for unifocal femoral condyle lesions but may be used for multifocal disease
- 2-stage procedure to implant chondrocytes to produce repair tissue that most closely replicated the composition and function of normal hyaline cartilage, restoring the durability and function of the joint articular surface:
  - Stage 1: arthroscopic biopsy of healthy chondral tissue
  - In vivo culture expansion
  - Stage 2: chondrocyte implantation

1.2.8 Autologous Chondrocyte Implantation

- First generation: periosteal patch utilized that leads to high reoperation rates.
- Second generation: utilizes a specialized bilayer collagen membrane instead of periosteal flap to cover implanted chondrocytes within the treated chondral defect.
- Third generation: three-dimensional scaffold acts as a carrier for implanted chondrocytes.

Outcomes

- Peterson et al. [36]: 61 patient at average 7.4-year follow-up
  - Isolated femoral condyle or patella.
  - After 2 years, 50 of 61 patients had good or excellent results.
  - At 5–11 years, 51 of 61 had good or excellent results.
  - 8/12 biopsies showed hyaline cartilage.
- Micheli et al. [37]: 32 patients at average 4.3-year follow-up after ACI of the distal femur
  - Mean changes in scale scores measuring overall condition, pain, and swelling were 3.8, 4.1, and 3.4 respectively.
  - One patient had implantation failure.
- *AJSM*: systematic review of level I and II studies of ACI versus other restoration/repair techniques
  - ACI versus MF
    - 3/7 studies found better clinical outcomes in ACI at 1–3 years.

        - 1/7 studies had better outcomes than MF at 2 years.
        - 2/7 studies no difference.
        - 3/7 deterioration in MF at 18–24 months compared to ACI.
        - No difference histologically.
    - ACI versus OC allograft
        - Two studies compared the two: quicker relief in OC allograft with overall similar results.
    - ACI versus ACI
        - Higher complication rate with open, 1st-generation, periosteal cover technique
    - Overall
        - Improved outcome in younger patients < 30 and shorter preoperative symptoms
        - Possible worse outcome with prior treatment but not measured in all studies
        - Defect size > 4 cm^2, better outcome with ACI

Focal Resurfacing

- Treatment of localized full-thickness articular cartilage lesions in middle-aged patients remains challenging.
- Previous biological treatment options may have failed.
- Other articular surfaces still intact – conventional joint replacement may be over-indicated.
- Nonoperative management delaying conventional arthroplasty.

1.2.9 HemiCAP® for Focal Full-Thickness Articular and Osteochondral Defects

- Designed to match the shape and contour of the patient's individual cartilage surface.
    - Ti fixation screw and CoCr articular component
- Device is a "patch" for focal arthrosis and necrosis designed to provide load sharing and contoured new surface while protecting the remaining, healthy cartilage.
    - Essentially, placement of an articular component matching the patient's individual joint surface geometry
- Components: fixation component and modular articular component connected with a Morse taper.
    - Fixation uses titanium cancellous screw with full-length cannulation.

Indications

- Focal full-thickness articular cartilage
- Osteochondral defects
- Early degenerative defects
- Localized avascular necrosis

Operative Technique

Standard deltopectoral approach with tenotomy of subscapularis and capsule taken down off the lesser tuberosity. Articular cartilage lesion is identified and measured with a guide pin placed at the center of the defect. The central post is placed before measuring the radius of curvature. Cartilage is scored and reamed before trial placement to verify the depth of the post; the peak height of the cap should be flushed with the surrounding cartilage. Final prosthesis is secured by Morse taper interlock and gently impacted.

Outcomes

- Bollars et al. [38]: 18 patients treated with knee femoral condyle resurfacing at average 34-month follow-up
    - Excellent results for pain and function in middle-aged, well-selected patient with full thickness and OCD
- Becher et al. [39]: 21 patients at 5.3-year follow-up
    - Improved KOOS pain, symptoms, return of activities, and quality of life.
    - Radiographic results demonstrated solid fixation, preservation of joint space, and no change in osteoarthritis stage.
- Uribe and Botto-van Bemden [40]: 12 shoulders with AVN of the humeral head at average 30-month follow-up
    - Improved VAS for pain as well as functional range of motion
    - No complications

1.3 Arthroscopic Soft Tissue Interpositioning for Patients with Glenohumeral Osteoarthritis

Polydoor E. Huijsmans and
Catherine van der Straeten

1.3.1 Introduction

When conservative treatment (analgesics, NSAIDs, corticoid injections, hyaluronic acid injections, physiotherapy) fails to control pain in glenohumeral osteoarthritis, surgical treatment remains the only option. However, especially in young and active patients, the surgical treatment with a total shoulder prosthesis is not always successful.

High percentages of glenoid loosening and disappointing subjective results in this group make us look for other options.

Arthroscopic treatment to postpone the need for shoulder replacements would be of enormous benefit in this group of patients.

One of the existing arthroscopic options is soft tissue interpositioning.

In this presentation our results with this technique are presented.

A small randomized study between debridement alone and debridement in combination with a human acellular skin graft showed better results for the patients with the interposition graft.

Therefore, we continued with this technique and have data of 45 patients with a minimum of 1 year post-op.

1.3.2 Technique Outline (Figs. 1.1, 1.2, 1.3, 1.4, 1.5 and 1.6)

- Arthroscopy in lateral decubitus position
- Removal of cartilage remains
- Creating a bleeding surface
- The labrum normally in good condition
- Posterior sutures in position
- Anterior loop for sutures through the anterior labrum

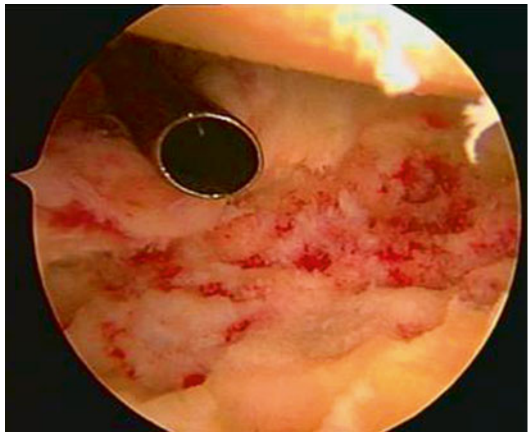

Fig. 1.1 Bony bed of the lesion after removal of cartilage remains

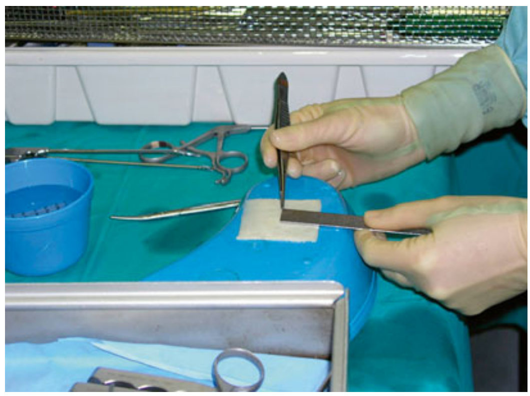

Fig. 1.2 Measuring of the graft

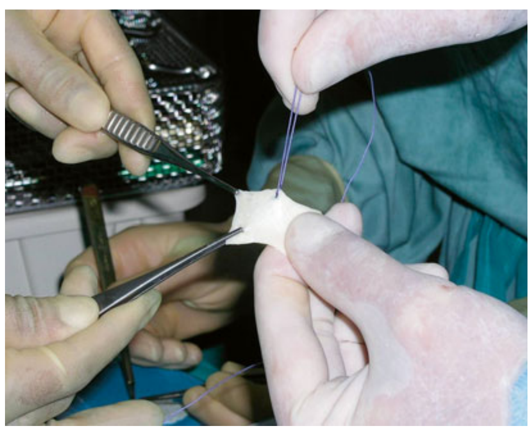

Fig. 1.3 Graft preparation

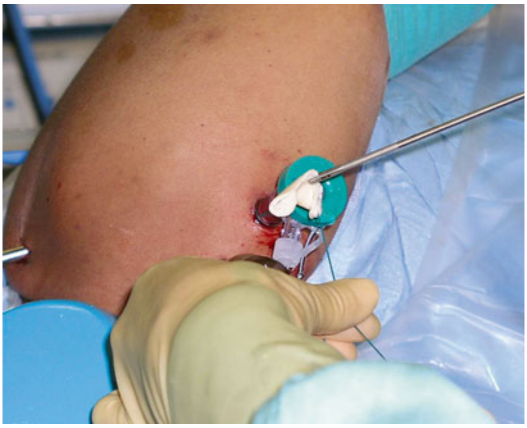

Fig. 1.4 Introduction of collagen matrix

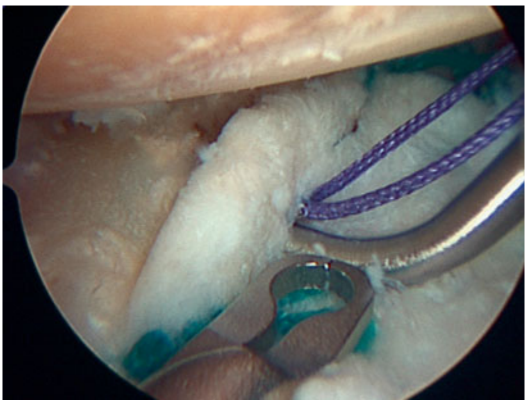

Fig. 1.5 Sutures tied to the labrum

Fig. 1.6 Graft grown into the treated lesion

- Introduction of collagen matrix
- Sutures tied on the labrum

1.3.3 Postoperative Regimen

- Three weeks passive mobilization, started in the first postoperative week.
- After 3 weeks gradual active movements of the shoulder in ADL circumstances.
- After 6 weeks muscle strengthening exercises are added, and once the pain is gone, activity may be increased.

1.4 Prevention of Shoulder Osteoarthritis in Young Patients

Roman Brzóska, Wojciech Solecki, and Adrian Błasiak

1.4.1 Introduction

The number of early recognized cartilage lesions of glenohumeral joint in young patients has increased in recent years with spread of arthroscopic surgery. Therefore, a research on finding methods of prevention of osteoarthritis development and its effective treatment is being conducted.

In our practice, we observe young patients developing shoulder osteoarthritis mainly as a result of intra-articular fractures, OCD, and metabolic and systemic diseases or as a consequence of rotator cuff tears and shoulder instability.

1.4.2 Intra-articular Fracture Management

We adopted arthroscopic techniques as a standard procedure in the treatment of patients with intra-articular fractures within the shoulder (Fig. 1.7). In our practice we use anchors, screws (Fig. 1.8), and cannulated Herbert screws, always making sure that the obtained fusion is stable as possible. For patients with clinically significant glenoid or humeral head bone loss, bone block grafts are used. Fracture union and joint congruency are evaluated in CT scan postoperatively (Fig. 1.9).

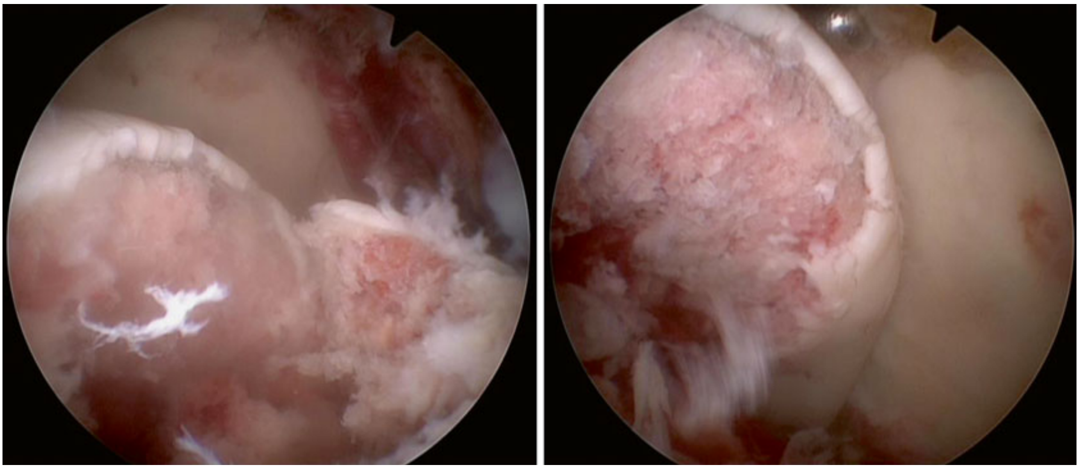

Fig. 1.7 Arthroscopic view of humeral head fracture

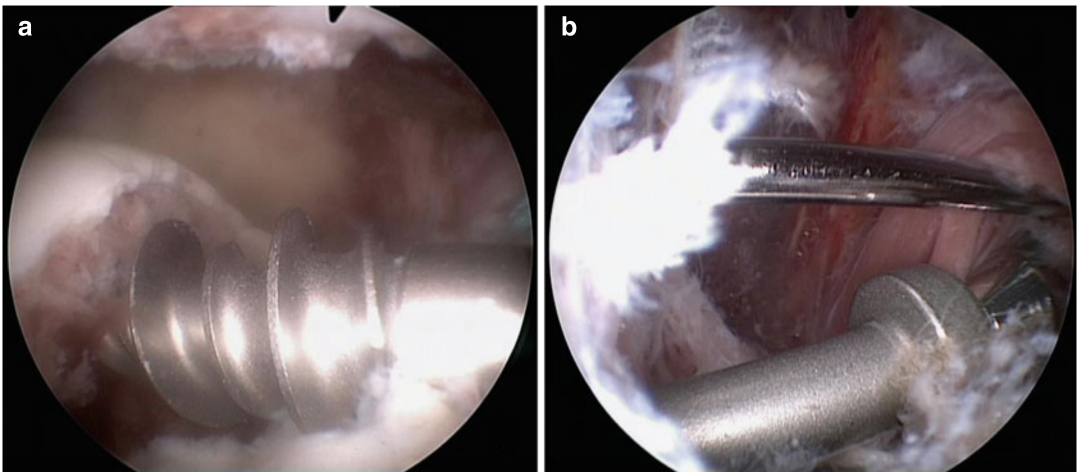

Fig. 1.8 Stabilization of the fracture with a suture anchor (**a**) and a screw (**b**)

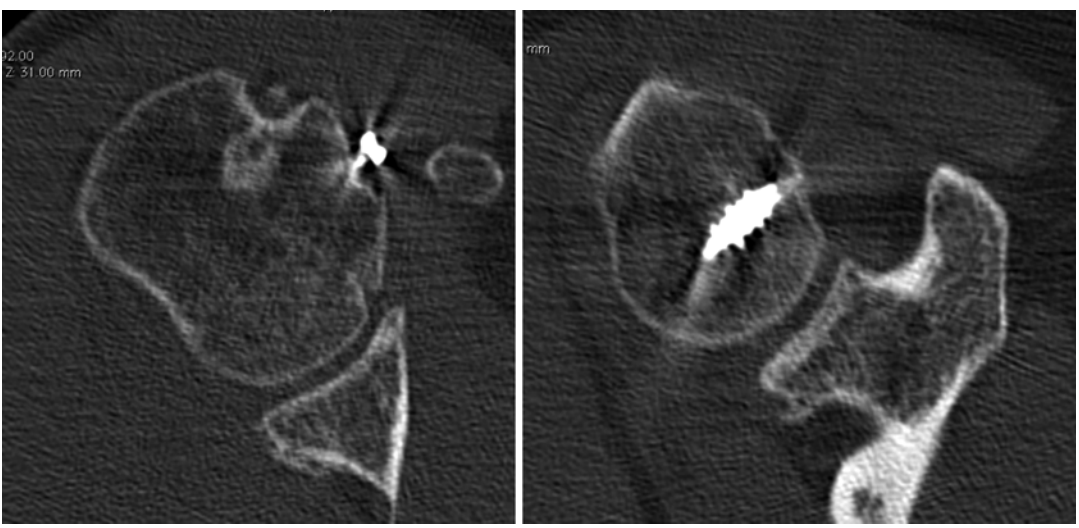

Fig. 1.9 CT scan 1 year after minor tubercle fracture treatment

So far we performed 11 surgeries achieving complete union of the fractures; in at least 1-year follow-up, we observed osteoarthrosis in one case.

1.4.3 OCD and Systemic Diseases

In patients with osteochondral defects due to already diagnosed systemic or metabolic diseases, method of treatment is chosen according to the extent and cause of the lesion. It should be noted that in case of large ischemic lesions, resurfacing or anatomic prosthesis remains as an alternative.

In patients with defects smaller than 1 cm^2, with either systemic, traumatic, or OCD background, iliac crest autograft or AMIC technique seems to be an effective treatment. The drawbacks of arthroscopic technique are limited access to some locations of the lesion, difficult debridement of deep bony defects, and difficult matrix insertion.

We performed arthroscopic repair of cartilage lesion using collagen matrix in four patients and repair of osteochondral lesion using autologous iliac crest bone graft with periosteum in five patients (Fig. 1.10). Early results were evaluated in Constant and VAS score and compared with radiological findings (MRI) (Fig. 1.11).

In both groups of patients, we observed improvement in range of motion and reduction of pain. Due to a short follow-up time, comparative assessment of both methods regarding subsequent development of shoulder osteoarthritis cannot be performed.

1.4.4 Surgical Techniques Preserving Glenohumeral Joint Cartilage

The rotator cuff tear management is a well-known technique not only restoring the function of the shoulder but also preventing the development or progression of osteoarthritis. Currently used techniques for treatment of shoulder instability are characterized by a high rate of early osteoarthritis (Fig. 1.12). According to Harris 19–56 % of patients or as Kavaja states up to 68 % of patients develop shoulder osteoarthritis subsequent to surgical treatment.

We performed a retrospective study of 55 patients treated for glenohumeral instability using Between the Glenohumeral Ligaments and Subscapularis Tendon (BLS) technique with 5-year follow-up. Clinical and radiological outcomes including recurrence of instability and presence of glenohumeral osteoarthritis are still under evaluation.

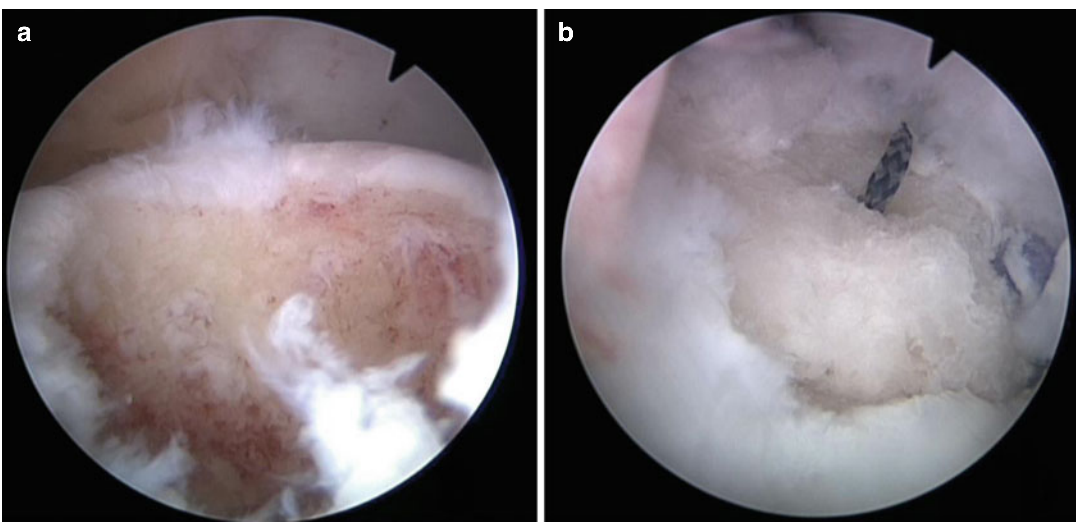

Fig. 1.10 Osteochondral defect of humeral head before (**a**) and after insertion of an iliac crest graft (**b**)

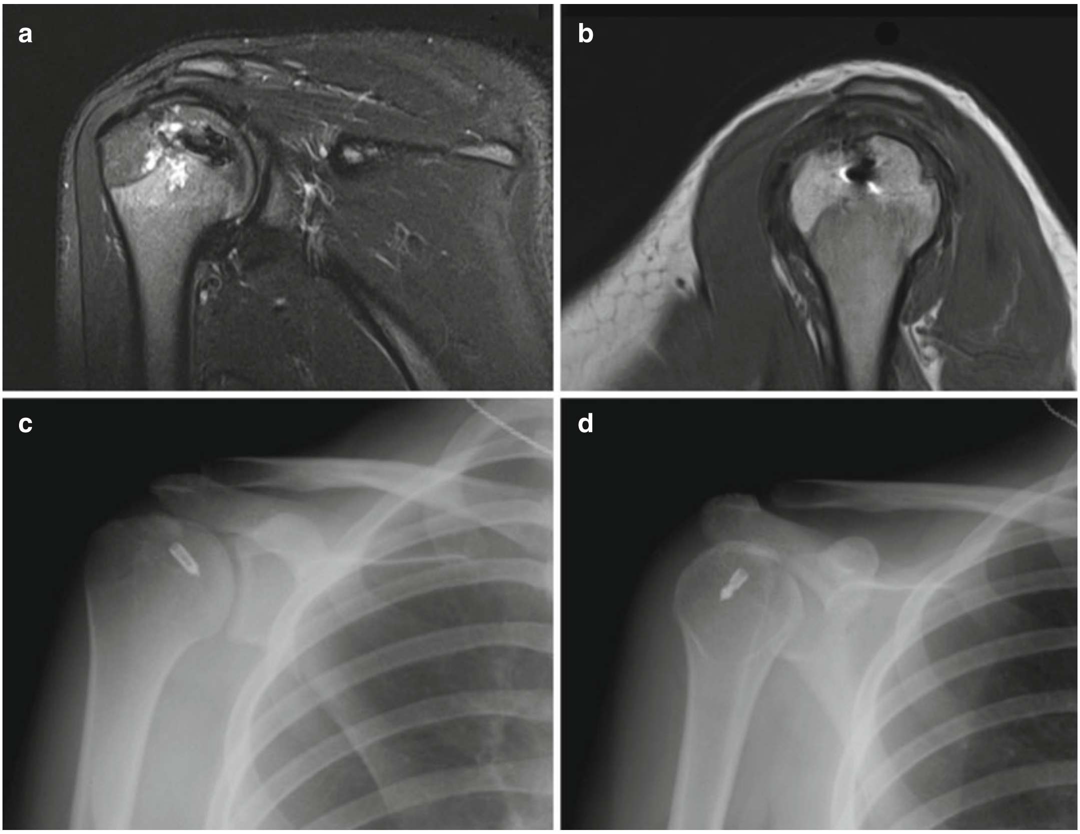

Fig. 1.11 Post-operative MRI (**a**, **b**) and X-ray (**c**, **d**)

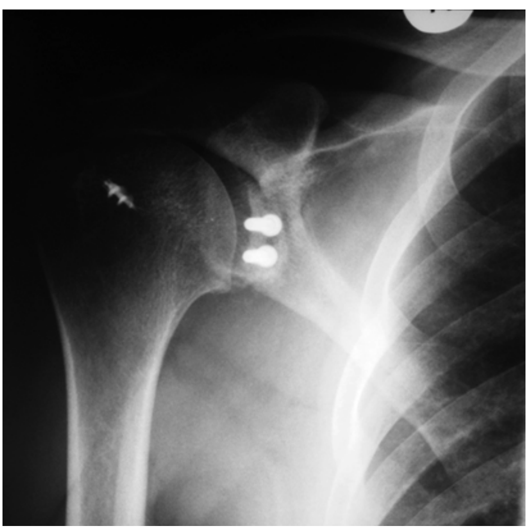

Fig. 1.12 Early osteoarthrosis 2 years post-op Latarjet procedure

Preliminary results are satisfactory and suggest that the elimination of contact between the humeral head cartilage and sutures seems to protect the joint against cartilage damage, which may slow the development of osteoarthritis. However, long-term observations are needed to confirm this hypothesis.

Conclusions

Early outcomes of biological joint-preserving techniques are promising; however, due to dynamic changes of the radiological presentations, further investigation with higher number of cases and longer follow-up is required. Benefits of shoulder cartilage and osteochondral defect treatment using biological methods such as AMIC and MSC techniques and microfractures and transplantation of iliac crest graft with periosteum need further investigation.

It is our duty to develop cartilage-preserving surgical techniques as well as to continue long-term observation of our patients based on multicenter studies.

References

1. Sprauge et al. Arthroscopic debridement for degenerative knee joint disease. Clin Orthop 1981;160: 118–23.
2. Magnuson PB. Joint debridement: a surgical treatment of degenerative arthritis. Surg Gynecol Obst 1941;73:1–9.
3. Jackson RW, Marans HJ, Silver RS. The arthroscopic treatment of degenerative arthritis of the knee. J Bone Joint Surg [Br] 1988;71–B:332.
4. Salisbury RB, Nottage WM, Gardner V. The effect of alignment on results in arthroscopic debridement of the degenerative knee. Clin Orthop 1985;198:268–72.
5. Baumgaertner MR, Cannon WD Jr, Vittori JM, Schmidt ES, Maurer RC. Arthroscopic debridement of the arthritic knee. Clin Orthop 1990;253:197–202.
6. Steadman JR, Briggs KK, Rodrigo JJ, Kocher MS, Gill TJ, Rodkey WG. Outcomes of microfracture for traumatic chondral defects of the knee: average 11-year follow-up. Arthroscopy. 2003;19(5): 477–84.
7. Mithoefer K, Williams III RJ, Warren RF, Potter HG, Spock CR, Jones EC, Wickiewicz TL, Marx RG. The microfracture technique for the treatment of articular cartilage lesions in the knee. A prospective cohort study. J Bone Joint Surg Am. 2005;87(9):1911–20.
8. Frank RM, Van Thiel GS, Slabaugh MA, Romeo AA, Cole BJ, Verma NN. Clinical outcomes after microfracture of the glenohumeral joint. Am J Sports Med. 2010;38(4):772–81.
9. Millett PJ, Huffard BH, Horan MP, Hawkins RJ, Steadman JR. Outcomes of full-thickness articular cartilage injuries of the shoulder treated with microfracture. Arthroscopy. 2009;25(8):856–63.
10. Goyal D, Keyhani S, Lee EH, Hui JH. Evidence-based status of microfracture technique: a systematic review of level I and II studies. Arthroscopy. 2013; 29(9):1579–88.
11. Pappas AM. OCD. Int Orthop. 2012;36(11): 2243–8.
12. Aichroth P. OCD of the knee. J Bone Joint Surg. 1971;53-B:440–7.
13. Fairbank HA. OCD. Br J Surg. 1933;21:67–82.
14. Mubarak SJ, Carroll NC. Juvenile OCD of the knee: etiology. Clin Orthop. 1981;157:200–11.
15. Campbell CJ, Ranawat CS. OCD: the question of etiology. J Trauma. 1966;6:201–21.
16. Chiroff RT, Cooke CP. OCD: microradiographic analysis. J Trauma. 1975;15:369–96.
17. Ribbing S. Hereditary ME. Disturbances. Acta Orthop Scand. 1955;24(4):286–99.
18. Cahill B. Treatment of juvenile OCD & OCD of the knee. Clin Sports Med. 1985;4:367–84.
19. Clanton TO, DeLee JC. OCD. History, pathophysiology & current treatment concepts. Clin Orthop Relat Res. 1982;167:50–64.
20. Aglietti P, et al. Arthroscopic drilling in juvenile OCD. Arthroscopy. 1994;10:286–91.
21. Smillie IS. The treatment of OCD. J Bone Joint Surg Br. 1957;39-B:248–60.
22. Mackie IG, et al. Arthroscopic use of the Herbert screw in OCD. J Bone Joint Surg Br. 1990;72-B:1076.
23. Dervin GF, et al. Biodegradable rods in adult OCD of knee. Clin Orthop Relat Res. 1998;356:213–21.
24. Victoroff BN, et al. Arthroscopic bone peg fixation in the treatment of OCD in the knee. Arthroscopy. 1996;12:506–9.
25. Landells JW. The reaction of injured human cartilage. J Bone Joint Surg. 1957;39-B:548–62.
26. Miniaci A, Tytherleigh-Strong G. Fixation of unstable osteochondritis dissecans lesions of the knee using arthroscopic autogenous osteochondral grafting (mosaicplasty). Arthroscopy. 2007;23(8):845–51.
27. Outerbridge HK, et al. OCD defects in the knee. Clin Orthop Relat Res. 2000;377:145–51.
28. Bobic V. Arthroscopic osteochondral autograft transplantation in anterior cruciate ligament reconstruction: preliminary clinical study. Knee Surg Sports Traumatol Arthrosc. 1996;3:262–4.
29. Hangody L, Kish G, Karpati Z, Szerb I, Udvarhelyi I, Toth J, et al. Autogenous osteochondral graft technique for replacing knee cartilage defects in dogs. Orthopaedics. 1997;5:175–81.
30. McDermott GB, et al. Fresh small fragment osteochondral allografts. Long term follow up on 1oo cases. Clin Orthop Relat Res. 1985;197:96–102.
31. Hangody L, Fules P. Autologous osteochondral mosaicplasty for the treatment of full-thickness defects of weight-bearing joints: ten years of experimental and clinical experience. J Bone Joint Surg Am. 2003;85-A Suppl 2:25–32.
32. Ozturk A, Ozdemir MR, Ozkan Y. Osteochondral autografting (mosaicplasty) in grade IV cartilage defects in the knee joint: 2- to 7-year results. Int Orthop. 2006;30(3):200–4.
33. Kircher J, Patzer T, Magosch P, Lichtenberg S, Habermeyer P. Osteochondral autologous transplantation for the treatment of full-thickness cartilage defects of the shoulder: results at nine years. J Bone Joint Surg Br. 2009;91(4):499–503.
34. Krych AJ, Harnly HW, Rodeo SA, Williams III RJ. Activity levels are higher after osteochondral autograft transfer mosaicplasty than after microfracture for articular cartilage defects of the knee: a retrospective comparative study. J Bone Joint Surg Am. 2012;94(11):971–8.
35. Gudas R, Gudaite A, Pocius A, Gudiene A, Cekanauskas E, Monastyreckiene E, Basevicius A. Ten-year follow-up of a prospective, randomized clinical study of mosaic osteochondral autologous transplantation versus microfracture for the treatment of

osteochondral defects in the knee joint of athletes. Am J Sports Med. 2012;40(11):2499–508.
36. Peterson L, Brittberg M, Kiviranta I, Akerlund EL, Lindahl A. Autologous chondrocyte transplantation. Biomechanics and long-term durability. Am J Sports Med. 2002;30(1):2–12.
37. Micheli LJ, Moseley JB, Anderson AF, Browne JE, Erggelet C, Arciero R, Fu FH, Mandelbaum BR. Articular cartilage defects of the distal femur in children and adolescents: treatment with autologous chondrocyte implantation. J Pediatr Orthop. 2006;26(4):455–60.
38. Bollars P, Bosquet M, Vandekerckhove B, Hardeman F, Bellemans J. Prosthetic inlay resurfacing for the treatment of focal, full thickness cartilage defects of the femoral condyle: a bridge between biologics and conventional arthroplasty. Knee Surg Sports Traumatol Arthrosc. 2012;20(9):1753–9.
39. Becher C, Kalbe C, Thermann H, Paessler HH, Laprell H, Kaiser T, Fechner A, Bartsch S, Windhagen H, Ostermeier S. Minimum 5-year results of focal articular prosthetic resurfacing for the treatment of full-thickness articular cartilage defects in the knee. Arch Orthop Trauma Surg. 2011;131(8):1135–43.
40. Uribe JW, Botto-van Bemden A. Partial humeral head resurfacing for osteonecrosis. J Shoulder Elbow Surg. 2009;18(5):711–6.

Shoulder Pathology in Sports

2

Norman D'Hondt, Lennard Funk, Jo Gibson, Srinath Kamineni, Tom Clement Ludvigsen, Puneet Monga, and Nestor Zurita

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2.1 Introduction

Sports injuries are on the increase, with the increasing interest and involvement in sports around the world. In Europe there is a large diversity of sports, with many sports being more prominent in certain countries. Although injury pathology is similar (e.g. a labral tear), the injury mechanisms are often unique to that sport and the rehabilitation and demands are also unique to the sport. In this Instructional Course chapter, we hope to show you some examples of this and highlight some unique and important current issues in the sports shoulder in 2014.

2.2 Shoulder Injuries in European Handball

Tom Clement Ludvigsen

European handball is a tough contact sport with high-energy collisions between opponents.

A male handball player is normally about 190 cm tall and weighs 90–100 kg; the game is very fast and includes numerous cutting movements, rapid direction changes, jumping and running. However, in contrast to other similar tough

S. Zaffagnini et al. (eds.), *ESSKA Instructional Course Lecture Book*,
DOI 10.1007/978-3-642-53983-1_2, © ESSKA 2014

contact sports as ice hockey, rugby and American football, the vast majority of collisions occur face to face, and tackles from behind and the side are less frequent.

Both the collisions and a very high number of throws and passes make the shoulder region vulnerable to acute injuries as well as overuse injuries.

Very few studies on the epidemiology of shoulder injuries in handball have been published and most of them are focused on acute injuries. Gohlke et al. [1] and Seil et al. [2] reported on a high number of shoulder pain in mail elite handball players. In 2011 Myklebust et al. reported a high prevalence of shoulder pain (57 %) amongst elite Norwegian female handball players, and in 2012 Moller et al. reported shoulder injuries to be ranked third after ankle and knee as the most common injuries in a mixed population of male and female elite Danish handball players. Clarsen et al. reported in 2012 that 51 % of male elite Norwegian handball players experienced shoulder problems in the dominant arm throughout the 1-year season; 23 % of them had serious problems leading to reduced performance and/or time out of play.

Despite the high energy involved in the collisions and tackles, fractures of the shoulder region are quite rare and soft tissue injuries are dominating.

Sprains and contusions occur as a result of the collisions. Sprains of the AC joint do occur, but AC joint dislocations are quite rare.

A relatively typical injury pattern in handball is a forced hyperextension of the arm, either in full extension or abducted 90°. This is the mechanism behind the majority of anterior shoulder dislocations and also subluxations, which in turn may cause stretching of the anterior passive stabilisers.

Another acute injury frequently reported is that an opponent is hooking the throwing arm from behind in the exact moment when the throw is initiated, which is when the arm is in abduction and maximum external rotation with the throwing muscles fully activated, this leading to considerable stress on anterior structures and in some rare cases isolated avulsion of the superior glenohumeral ligament from the anterosuperior glenoid rim.

The majority of reported shoulder injuries are, however, shoulder pain. Ninety-five percent of these injuries affect the dominant throwing arm, of which two thirds have a gradual onset.

The high number of throws and passes exposes the handball players to the same pathogenesis as reported in other overhead athletic activities, amongst whom the baseball pitchers, and maybe javelin throwers, probably have been most frequently studied. This pathology leads to loss of scapula control, internal impingement, SLAP lesions, weakness of internal rotators and stretching of anterior stabilising structures. In addition, however, the handball players may have had numerous minor injuries as described above, which increases the risk of developing these type of symptoms. Accordingly, Myklebust et al. found that 60 % of the players with current pain had a positive apprehension and relocation test indicating a shoulder instability problem.

In conclusion, shoulder injuries are a frequent finding in handball players. The majority of the injuries have a gradual onset and affect the throwing (dominant) arm. Treatment of the injuries should be in accordance with the generally accepted outlines for treating overhead throwing athletes, and great care should be taken to avoid surgery not founded on strong indications. One should be aware of the fact that even a successful operation rarely can bring such an athlete back to pre-injury level of performance.

2.3 Pectoralis Major Ruptures

Puneet Monga

The pectoralis major is a large fan-shaped muscle having a clavicular and sternocostal origin over the anterior aspect of the chest wall. It inserts onto the lateral lip of the bicipital groove along a 5 cm narrow longitudinal insertion. It is innervated by the medial and lateral pectoral nerve and is a strong flexor, adductor and internal rotator of the shoulder. Enthusiasts commonly use resistance training, typified by the 'bench press' or 'push-up' action, to build up this muscle. All types of swimming strokes along with rowing are other aerobic forms of training the pectoralis major. It is hardly surprising therefore that pectoralis major injury is commonly related to these sports. Like other tendon injuries, ruptures are the most common during eccentric contraction, and the role of overtraining and fatigue is often debated.

An acute pectoralis major rupture presents typically with a sudden onset pain in the pectoral region during strenuous activity. The ‘bench press’ is a commonly reported activity. Patients commonly report a ‘pop’ or a ‘snap’ along with swelling and loss of definition of the anterior axillary fold. Bruising is commonly seen on the medial/distal aspect of the arm and the medial axillary wall. The diagnosis of pectoralis muscle ruptures is usually evident from the typical history and examination findings in a specialist setting, but lack of awareness of such injuries in a generic casualty setting can lead to delayed diagnosis. The rupture most commonly occurs with the tendon avulsing off the humerus although musculotendinous junction ruptures and intramuscular injuries are sometimes seen.

Musculotendinous and intramuscular injuries may not be associated with the typical loss of pectoral contour, and an MR scan is best to confirm diagnosis. MR scan is also useful to pick up associated injuries and delineate the extent of the tear. Surgical treatment for acute ruptures is offered to patients desirous of return to sports and heavy manual work. Surgical repair of the tendon to the lateral lip of the bicipital groove is achieved using bone anchors with an aim of achieving a longitudinal area of footprint to recreate the original pectoralis major attachment. Such tears may extend into the musculotendinous junction and intramuscular part of the pectoralis major, and repair of these should be concomitantly addressed as such. Chronic presentations may require Achilles tendon allograft to bridge the gap generated by retraction and help achieve an adequately tensioned repair.

The key determinant of a good outcome for athletes with complete pectoralis major ruptures is early diagnosis and anatomical repair. Surgical repair is associated with better outcomes related to strength and return to sporting activity, and nonoperative treatment has a limited role in the active sporting population. Repairs for chronic ruptures are associated with higher risks and relatively inferior outcomes as compared to acute repairs, but may be considered in conjunction with Achilles tendon allograft for the symptomatic athlete.

2.4 The Swimmer’s Shoulder

Nestor Zurita

Swimming is a very demanding sport, and when practiced at a good competition level, there is a high risk of injury. Due to the nature of the sport, the most common place of injury is the shoulder of the athlete. In the year 2000, Jobe stated that, on average, 50 % of competitive swimmers suffer shoulder pain severe enough to prevent them from swimming for 3 weeks or more at some point in their sporting career.

The great complexity of the pathological phenomena that taking place in what we define as swimmer’s shoulder creates a challenge for practitioners when planning the therapeutic approach to these injuries.

Therefore, we set the objectives to define the pathogenetic and physiopathology mechanisms of swimmer’s shoulder injury and the therapeutic approach towards treatment. We used a sample of 50 swimmers from the national swimming team of the Royal Spanish Swimming Federation (RFEN). We developed a 4-year observational design, in which we collected all musculoskeletal injuries that were subsidiaries of treatment from the medical services of the Royal Spanish Swimming Federation, classified them according to their location, type and length of treatment. The diagnosis and treatment of the injuries were performed according to clinical protocols established by the medical services of the RFEN.

From our data, shoulder pain was the most common problem in RFEN swimmers, representing 94.4 % of the injuries in the upper limb and 57.62 % of the total incidents, coinciding with other series. This distribution practically remains the same in each of the different styles of swimming. However, the variations between them are determined by the biomechanical characteristics of each of the different swimming strokes.

The factors influencing the presence of shoulder dysfunction will be those associated with unusually high number of revolutions, approximately 500,000 for each arm, added to the extreme arches needed for each revolution and the general state of laxity in these athletes.

Other influences are the result of repetitive microtrauma to the input wave motion in the water. The factors of fatigue or weakness to the muscles of the anterior wall and stretching of capsular and ligamentous structures both contribute to each other until instability occurs.

Thus we define the swimmer's shoulder as a double impingement mechanism of the rotator cuff with glenoid and the acromion, added to a base of instability due to extreme ranges of motion in this sport. Characteristically, impingement of the rotator cuff against the acromion occurs mostly during the beginning of the stroke, when the hand seeks entry into the water and this is located too low in butterfly and front crawl. Another risk position is when the hand enters the water, at which time there is an impingement between the lower surface of the rotator cuff and the anterior glenoid rim.

Other points of friction include the top edge of the glenoid and the coracoid process, with increased flexion, internal rotation and adduction of the arm.

Generally, swimmer's shoulder injuries are usually due to the muscle and tendon overuse. Therefore, we always choose orthopaedic treatment as the first option. It consists of physiotherapy, a technical study and treatment of sporting gesture intended to balance biomechanical muscle function, so that we compensate the instability.

The surgical option is reserved for swimmers that are entering into the in the final stages of their career or the presence of specific lesions of the labrum and/or ligamentous and tendon structures.

2.5 Mechanics of Throwing

Norman D'Hondt

Since the overhead athlete's shoulder is often exposed to extreme movement ranges, repetitive motion and high peak loads, there is no doubt that its high demand forms a potential risk to injuries.

The origin of most pathology found in the athlete's shoulder (e.g. SLAP lesions, PASTA lesions, biceps pulley lesions) is referred to as internal impingement mechanisms.

In general it is thought that those pinching mechanisms are due to excessive translations of the humeral head to a specific direction, caused by capsular laxity or tightness and that these mechanisms are typically manifest in the late cocking phase of the throwing cycle.

As part of a kinetic chain, it is also thought that a lack of scapular stability and retraction and malposition of the scapula, in popular terms 'scapular dyskinesia', play a major contributing role in provoking the pathomechanism. As the kinetic chain literally extends to the tip of the toe, alterations elsewhere in the body are presumed to be of clinical importance as well.

From the perspective that not only the shoulder, but that the whole body participates in the throwing motion, clinical analysis and decision making are often based on the results of various tests that try to identify each of these presumed contributing factors.

2.5.1 State-of-the-Art Treatment

Many authors recommend a full kinetic chain approach during a rehabilitation programme of injured overhead athletes. Using an ideal, symmetrical posture and a movement pattern accordingly as a reference standard, these programmes include optimising hip flexibility, training core stability, stretching of posterior glenohumeral structures, scapula setting and stabilisation exercises, restoring UT/LT muscle imbalance with specific muscle exercises and muscle strengthening.

When rehabilitation does not succeed, surgical procedures are considered to be the next approach. In general these are mainly focussed on repairing the damaged structures.

2.5.2 Future Treatment Options

From a classical point of view, the throwing cycle serves as the reference standard for evaluating throwing mechanics.

But since performance of a throw depends on many variables, it is not that simple.

For instance, morphological properties of the object that is about to be thrown (e.g. a javelin, a dart, a handball or a baseball) and the environmental circumstances (target position and distance, actual speed of the athlete, mental state, etc.) are not always equal.

As well are morphological properties of the musculoskeletal system and movement coordination skills individually unique and subject to adaptation.

Therefore, these are serious determining factors that have to be taken into consideration when interpreting a patient's problem.

Unfortunately, not all clinical measurements are able to accurately reflect the true nature of the athlete's problem. For example, using a step test to investigate the core stability of a swimmer with shoulder complaints seems a bit awkward.

This puts their use and their consequences with respect to treatment into debate.

2.5.3 Take Home Message

Hence, the existence of an ideal posture or movement pattern is questionable; we believe that after having examined the athlete's unique constraints of movement, an analysis of how the athlete uses these constraints, preferably in the sports context (e.g. video analysis or simulation of the provocative task), will result in a better understanding of how and why tissue damage occurs.

This ICL will discuss how a whip phenomenon causes trouble in an overhead athlete's shoulder.

2.6 Shoulder Injuries in Soccer

Lennard Funk

Shoulder injuries in football are not nearly as common as lower limb injuries such as the hip, knee or ankle. The incidence in the reported literature is approximately 2–4 % of all football injuries. However, shoulder injuries are generally more serious than many of the other more common injuries sustained and result in a longer time off play than other joint injuries.

Due to the paucity of literature, we undertook a study to analyse the incidence and treatment of shoulder injuries in professional football in the UK (2010). Anonymous and summarised data was courtesy of Health Partners Europe Limited, medical insurers to the PFA (Professional Footballers' Association). Data was provided over a 3-year period from January 2007 to January 2010. There were 35,000 claimed injuries in this period, of which 3.3 % were shoulder injuries. This is a total of 1,155 shoulder injuries or 385 serious shoulder injuries per year, which is still a significant number of injuries.

The percentage of shoulder injuries per year over the total shoulder injuries increased from 35 % in the 2006/2007 season to 85 % in the 2008/2009 season. There was also a steady increase in the number of MRI scans, but a similar number of MR arthrograms were requested over the study period. There was a significant increase in the number of surgical interventions from 12 in 2006/2007 to over 20 in the 2009/2010 season. The vast majority of the surgical procedures were arthroscopic stabilisations (29 %) and labral repairs (17 %) comprising 46 % of the surgical cases.

The commonest mechanism of injury is forced abduction external rotation injury with anterior to posterior applied force on the arm resulting in an anterior dislocation or subluxation. We have seen this typically occur in outfield players when jumping for the ball with an opponent and their arms in ABER position or when being struck by a ball with their arm in this position. In goalkeepers diving for the ball with their arm extended overhead and being forced back or in contact with an opposing striker can lead to this mechanism of injury as well.

Posterior injuries can occur when the flexed arm is in internal rotation usually as coming down from a high jump or when landing after a tackle. We have not seen any posterior labral injuries or posterior dislocations in goalkeepers yet.

Funk and Sargent [20] looked at all the professional football players we treated over a 3-year period assessing the injury mechanisms and pathologies. Over that period there were 25 professional footballers of which 15 were outfield players and 10 goalkeepers. It was interesting to note that on MR scan the rotator cuff showed a chronic pathology in five goalkeepers (50 %) compared to two outfield players (13 %). However, there were ten Bankart lesions in the outfield players (67 %) compared to one in the goalkeepers (10 %). Of the rotator cuff pathologies, the goalkeepers had predominantly partial-thickness rotator cuff tears or tendonosis or partial-thickness rotator cuff tears and another three players had significant tendonosis. Thus in total 80 % of the goalkeepers had some rotator cuff pathology.

The treatment of the rotator cuff pathologies depended on the severity of the pathology as well as the significance of the symptoms of the patient. Mild impingement symptoms with bursitis were treated with rehabilitation and subacromial steroid injection if required. Partial-thickness rotator cuff tears were treated with arthroscopic debridement or repair depending on the severity of the tear. However, for early or mid-season players able to continue playing although with significant discomfort used platelet-rich plasma injections. For full-thickness rotator cuff tears, arthroscopic repair was undertaken.

Most players with a labral injury and associated pathologies require surgical repair. Some may be able to cope through the season and can be repaired at the end of the season. These are usually the less significant injuries with lesser pathology on MR arthrogram. Large structural lesions such as bony Bankart injuries, rotator cuff tears and large Hill-Sachs lesion generally require early surgical repair. It is also better to perform an early repair on injuries sustained later in the season to avoid absence from play in the forthcoming season due to ongoing unresolved pathology.

Soft tissue labral capsular and rotator cuff repairs are generally performed arthroscopically with early return to play and excellent outcomes with recurrence rates to be expected of less than 7 %. Significant bony lesions, particularly large chronic bony Bankart lesions with glenoid deficiency, require bony reconstruction procedures, and the most popular procedure at the moment is the Latarjet procedure.

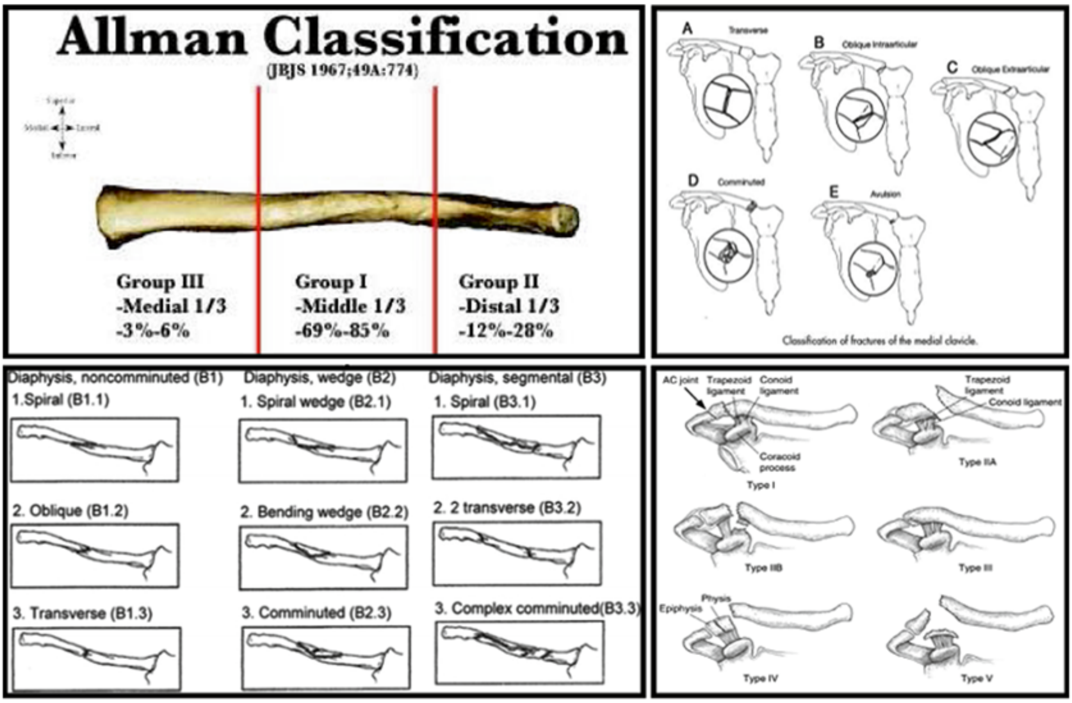
Rehabilitation after the surgery should be sport specific, and we prefer to start the sports-specific rehabilitation immediately. A striker will have different rehabilitation requirements to a defender and significantly different requirements to a goalkeeper. Although the basic principles are the same, their requirements and goals will be different. We prefer to follow a phase- or goal-based approach to progressing through the rehabilitation schedule rather than a time-based approach. A player should return when he is ready, fully rehabilitated and it is safe to do so rather than at a predetermined time. The rehabilitation will depend on a number of factors, these being the surgeon preference, type of pathologies and procedure performed, fixation techniques and implants used, patient age, requirements, compliance, physiotherapist experience and availability and, most importantly, good communication and co-operation between patient, therapist and surgical team.

2.7 Clavicle Fracture in Sports

Srinath Kamineni

Clavicle fractures represent a common cause of loss of playing and training time. Since the bone is subcutaneous, diagnosis and deformity are often straightforward. The commonest mechanisms are a direct fall and contact with the shoulder tip with either a downward force along the arm or a medially directed force along the shaft of the clavicle. Less commonly a direct impact and fall on an outstretched hand are responsible for sporting fracture.

Whereas Allman broadly classified clavicle fractures according to geographic location, medial-middle-lateral, more comprehensive classifications have been developed for medial, midshaft, and lateral third fractures.



The majority of mid-shaft and medial third fractures are treated conservatively with slings and figure-of-eight braces, with restoration of good motion and strength. Early return to play, especially contact sports, is associated with a high refracture rate. Strapping of clavicle fractures can improve fracture position. Displaced fractures often require operative fixation especially if associated with comminution, shortening greater than 2 cm (which has been associated with longer-term weakness), other injuries, e.g. floating shoulder, and skin integrity (impending or breach).

Plate and screw fixation (non-locking or locking) is the gold standard method of operative fixation, but is associated with more side effects compared to intramedullary fixation. Biomechanical data suggests plating strength can be optimised by locating it anteroinferiorly, compared to the traditional superior placement, additionally serving to decrease skin complications and improve its biomechanics.

Several recent intramedullary devices have gained popularity, but none (Rockwood pin, Sonoma nail, Hagie pin, etc.) have significant documented complications. However, where rotational control is necessary, plate fixation has the advantage compared to intramedullary devices.

Methodology to accelerate the bony healing process has already gained impetus with attempts with low-intensity pulsed ultrasound that has been trailed without significant positive effect. Stem cell therapy also holds promise for bone healing acceleration. The other major directions for the future are more biomechanically compatible implants (extra- and intramedullary), both in regard to modulus of elasticity vs. required strength, and biodegradability, circumventing the need to implant removal.

In summary, until the biology of fracture healing can be accelerated, we have to respect the timeline of bony union. Currently a coherent treatment algorithm is based on several factors, e.g. sport specific, part of season in which injury occurred, demands of sport, fracture pattern and soft tissue integrity. A cautionary message to remember: trying to accelerate return to full sporting activity may lead to a greater, potentially career-ending injury.

2.8 Sports-Specific Rehabilitation

Jo Gibson

Shoulder injuries in sport present a considerable challenge to the rehabilitation specialist. Evidence suggests that there is an increasing prevalence of shoulder injuries within the sporting population, and significantly these are associated with a high recurrence rate. It is of note that when comparing return to play data in different sports, a greater proportion of shoulder injuries result in prolonged periods away from sports participation compared to lower quadrant injuries. Patterns of recovery post surgery differ according to specific sports; in the contact athlete there is a higher incidence of recurrence but a relatively quick return to play; in the overhead athlete authors report that return to play is much slower and that deficits in shoulder function can persist for up to 2 years post injury. The functional demands of differing sports are therefore an essential consideration when designing effective rehabilitation programmes.

The loads and shear stresses imposed on the glenohumeral joint during swimming or throwing clearly differ markedly from those in a rugby tackle situation. Nonetheless, there are well-reported commonalities in terms of deficits in the dynamic system. Traditionally much emphasis has been placed on strength ratios within the sporting population and are stipulated as key in terms of return to play criteria. Irrespective of the specific sport, inhibition or relative weakness in the external rotators is well reported in many sports-related shoulder injuries. However, increasingly it would seem that the stabilisation function of the rotator cuff and its ability to maintain the humeral head relatively centred within the glenoid during functional activity are of paramount importance. A loss of translational control and inability of the rotator cuff to stabilise pre-movement onset are key features of many shoulder injuries. This initial 'preset' is fundamental in engaging the dynamic system.

Scapula dyskinesis has been a frequently described feature of upper limb dysfunction and has been demonstrated in up to 85 % of throwing athletes with shoulder pain relating to instability. It has been stipulated that a lack of a stable base potentially increases loads and stresses across the glenohumeral joint during upper limb activities. However, whilst it has been commonly assumed that scapula dyskinesis is a primary feature of shoulder pathology, current literature challenges this supposition. Increasingly evidence suggests that scapula dyskinesis is actually a secondary feature of shoulder pathology resulting from rotator cuff dysfunction, i.e. a scapula 'lag'. Consequently exercises employed to rehabilitate dynamic control of the shoulder complex should work the scapula and rotator cuff in unison, not in isolation.

The role of the kinetic chain in shoulder pathology and its impact on glenohumeral stability must be considered. Deficits in the kinetic chain resulting in deficient proximal activation or inefficient force transfer can result in increased load across the shoulder potentially presenting an injury risk and impaired function. Increasingly the effects of the kinetic chain and poor movement patterns are recognised as key factors in shoulder pathology necessitating a more holistic approach to patient management. However, the other advantage of utilising a more dynamic approach to rehabilitation is its impact on the sensorimotor system. The sensorimotor system (SMS) encompasses the processing and integration of both afferent and efferent signals by the central nervous system, which is constant and dynamic. These feedforward and feedback mechanisms provide neuromuscular control and facilitate dynamic joint stability. However, these vital SMS functions have been shown to be impaired by both fatigue and injury. Consequently rehabilitation should aim to maximise utilisation of the SMS and enhance fatigue resistance. A kinetic chain approach emphasises SMS components whilst optimising efficiency of force transfer.

The challenge for the rehabilitation specialist is to address each of these parameters in a sports-specific manner. The motor learning literature is clear that exercises that have functional relevance to the individual patient and are dynamic will result in quicker and more effective movement re-education than non-specific ones. Similarly those that enhance input to the SMS in a functionally relevant manner establish transfer to function much more quickly therefore potentially improving rate of recovery and return to sport. Our increased understanding of the factors impacting dynamic stability of the shoulder complex and the demands of individual sports should underpin the design of any sports-specific rehabilitation programme.

Bibliography

1. Gohlke F, Lippert MJ, Keck O. Instability and impingement of the shoulder of the high performance athlete in overhead stress. Sportverletz Sportschaden. 1993;7(3):115–21.
2. Seil R, Rupp S, Tempelhof S, Kohn D. Sports injuries in team handball. A one-year prospective study of sixteen men's senior teams of a superior nonprofessional level. Am J Sports Med. 1998;26:681–7.
3. Myklebust G, Hasslan L, Bahr R, Steffen K. High prevalence of shoulder pain among elite Norwegian female handball players. Scand J Med Sci Sports. 2011;23(3):1–7.
4. Moller M, Attermann J, Myklebust G, Wedderkopp N. Injury risk in Danish youth and senior elite handball using a new SMS text messages approach. Br J Sports Med. 2012;46(7):531–7.
5. Clarsen B, Bahr R, Myklebust G. Overuse injuries of the shoulder in male elite Norwegian handball players Abstr. In: Annual Norwegian sports medicine conference. Oslo, Norway. 2012.
6. Anderson MW, Alford BA. Overhead throwing injuries of the shoulder and elbow. Radiol Clin North Am. 2010;48(6):1137–54.
7. Burkhart SS, Morgan CD, Kibler WB. The disabled throwing shoulder: spectrum of pathology. Part I: Pathoanatomy and biomechanics. Arthroscopy. 2003;19(4):404–20.
8. Mihata T, et al. Effect of posterior shoulder tightness on internal impingement in a cadaveric model of throwing. Knee Surg Sports Traumatol Arthrosc. 2013;1–7.
9. Mueller AM, Entezari V, Rosso C, McKenzie B, Hasebrock A, Cereatti A, Della Croce U, Deangelis JP, Nazarian A, Ramappa AJ. The effect of simulated scapular winging on glenohumeral joint translations. J Shoulder Elbow Surg. 2013;22(7):986–92.
10. Mihata T, Jun BJ, Bui CN, Hwang J, McGarry MH, Kinoshita M, Lee TQ. Effect of scapular orientation on shoulder internal impingement in a cadaveric model of the cocking phase of throwing. J Bone Joint Surg Am. 2012;94(17):1576–83.
11. Hodges PW, Tucker K. Moving differently in pain: a new theory to explain the adaptation to pain. Pain. 2011;152(3 Suppl):S90–8.
12. D'hondt NE, Hacquebord ST, van Schoot L, Kiers H, Terwee CB, Veeger HEJ. Measurement properties of clinical tests to assess cleidoscapulothoracic kinematics in patients with shoulder disorders. A systematic review. (in progress)
13. Veeger HEJ, D'hondt NE. De schoudergordel in evenwicht (Dutch) (The shouldergirdle in balance). In: van Wilgen CP, Calders P, Geraets JJXR, Nijs J, Veehof C, van Weegen EEH, editors. Jaarboek Fysiotherapie/Kinesitherapie 2013 (Yearbook of physiotherapy 2013). Bohn Stafleu Van Loghum, Amsterdam, Netherlands. p. 33–48.
14. Clabbers KM, Kelly JD, Bader D, Eager M, Imhauser C, Siegler S, Moyer RA. Effect of posterior capsule tightness on glenohumeral translation in the late-cocking phase of pitching. J Sport Rehabil. 2007;16(1):41–9.
15. Rizio L, Garcia J, Renard R, Got C. Anterior instability increases superior labral strain in the late cocking phase of throwing. Orthopedics. 2007;30(7):544–50.
16. Morais NV, Pascoal AG. Scapular positioning assessment: is side-to-side comparison clinically acceptable? Man Ther. 2013;18(1):46–53.
17. Crockett HC, Gross LB, Wilk KE, Schwartz ML, Reed J, O'Mara J, Reilly MT, Dugas JR, Meister K, Lyman S, Andrews JR. Osseous adaptation and range of motion at the glenohumeral joint in professional baseball pitchers. Am J Sports Med. 2002;30(1):20–6.
18. Sweitzer BA, Thigpen CA, Shanley E, Stranges G, Wienke JR, Storey T, Noonan TJ, Hawkins RJ, Wyland DJ. A comparison of glenoid morphology and glenohumeral range of motion between professional baseball pitchers with and without a history of SLAP repair. Arthroscopy. 2012;28(9):1206–13.
19. Ekstrand J, Hägglund M, Waldén M. Injury incidence and injury patterns in professional football: the UEFA injury study. Br J Sports Med. 2009. doi:10.1136/bjsm.2009.
20. Funk L, Sargent M. Shoulder injuries and management in Premier League Professional Football Players – ISSSMC conference, Newcastle, 18–20 Aug 2011.

Management of Failed Traumatic Anterior Instability Repair

3

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3.1 Failure of the Analysis at First Repair

Antonio Cartucho

Not all recurrences after shoulder surgical stabilisation are due to surgical error. It is impossible to prevent the dislocation of a shoulder with normal function. Error analysis and evidence-based medicine turned to be one of the most powerful tools of scientific progress.

Shoulder instability is one of the most frequent clinical entities in sports traumatology.

S. Zaffagnini et al. (eds.), *ESSKA Instructional Course Lecture Book*,
DOI 10.1007/978-3-642-53983-1_3, © ESSKA 2014

Due to advances in basic science and clinical investigation, there has been a constant evolution in concepts. At the same time, patients' expectations evolved and the need to shoulder better function is higher. The assumption of these expectations by the surgeon is mandatory because the risks taken by the patient after the healing process will have a definitive influence on the results. A judgment error at this stage is as important as a technical error at surgery. This being said, overhead and contact sports are a negative predictive factor and the chosen treatment must take this fact into consideration. Age is also considered a negative predictive failure [1]. This is due to the "normal" risk activity of the youngest. Although currently considered, the number of dislocations was not found statistically significantly responsible for the recurrence of instability in operated patients [2]. Hyperlaxity is also considered a risk for recurrence of instability after reconstructive surgery.

Complementary exams are important to determine the structural damage. Plain X-ray can determine a Hill-Sachs lesion and an anterior glenoid erosion. The presence of those lesions, age under 20, hyperlaxity and overhead sports were considered bad prognostic factors for arthroscopic reconstruction [3]. The ISIS score can be a valuable tool in order to decide the type of surgery [4]. Humeral head and glenoid amount of bone loss must be evaluated by CT scan like Sugaya showed us [5] or by arthro-CT scan [6]. More than 25 % of glenoid bone loss must be reconstructed by bone block techniques [7], and quantification of humeral head bone loss may point the need to use soft tissue techniques or bone block techniques to reduce recurrence rate [8]. MRI and arthro-MRI can be useful to determine the soft tissue lesions. ALPSA and HAGL lesions are associated with higher recurrence rates in arthroscopically treated patients.

Prior to reoperation, the described issues must be determined. Sometimes this fact allied with surgical report is sufficient to analyse the error committed at first surgery in order to correct it with a new procedure.

At the time of surgery, evaluation of the labrum and capsule healing and/or correct repair, correct positioning of the anchors, correction of capsular redundancy and repair of bony Bankart is mandatory. Once this is done, the correction of the errors committed at the first surgery can be achieved.

3.2 What to Do After the Recurrence?

Emilio Calvo and Diana Morcillo

3.2.1 Introduction

Anterior shoulder instability is the more frequent dislocation and it is estimated to affect 1.7 % of the population [9]. Satisfactory results have been obtained with open surgical stabilisation techniques, despite their risk of external rotation limitation, and they have been regarded traditionally as the "gold standard". However, with the advent of new arthroscopic techniques and the development of new implants, arthroscopic shoulder stabilisation is regarded as the ideal technique for this condition, with recurrence rates similar to those of open surgery. Despite these results, reported recurrence rates after open or arthroscopic shoulder stabilisation range between 5 and 15 % [10, 11].

Patients with postoperative shoulder instability should be carefully evaluated not only to diagnose the failure but also to clearly identify the underlying causes that determined the outcome and to establish a successful therapeutic strategy. Any patient with surgical treatment failure for shoulder instability may be allocated in one of the following groups. The first group is composed of patients in whom the problem was misdiagnosed, either because surgery was not indicated (i.e. voluntary instability), because the specific joint abnormalities to be corrected at surgery were not precisely identified, or because the direction of instability was not adequately defined (i.e. patients with multidirectional instability treated only for anterior instability). Another group of subjects includes properly diagnosed patients in whom the treatment was inadequate in terms of selection of the procedure or technical execution (Fig. 3.1). Obviously, there could also

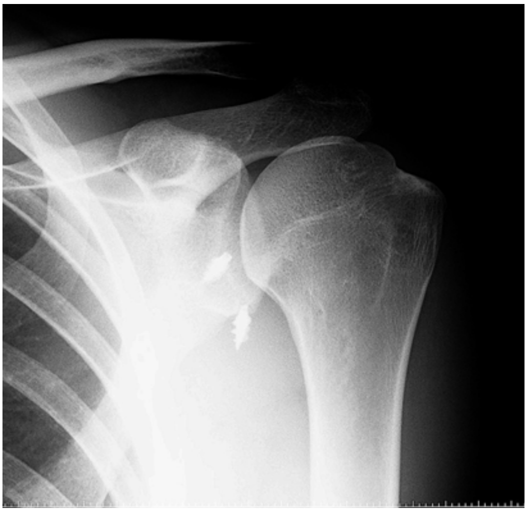

Fig. 3.1 Inadequate procedure with bone anchor

be patients composing a third group in whom both the diagnosis of the aetiology and problems associated to shoulder instability as well as the treatment performed were inadequate. Finally, the fourth group includes those patients that were properly diagnosed and in whom joint abnormalities were recognised and corrected with the optimal procedure, but that suffered a new trauma in the postoperative that caused a new shoulder dislocation [12, 13].

3.2.2 State of the Art

According to the previous outline, it is mandatory to have a clear idea on what the reasons are for this new dislocation. For this purpose, we must be able to correctly answer the following questions:

3.2.2.1 What Are the Characteristics of the Patient?

Prior to evaluating the shoulder and the potential reasons for postoperative shoulder instability, it is necessary to discard other problems different to shoulder instability, like stiffness, pain or weakness. In this setting, conditions like cervical problems, neurologic injuries, adhesive capsulitis, osteoarthritis, subscapularis muscle injuries, etc., must be ruled out.

Once the diagnosis of postoperative recurrence of shoulder instability is confirmed, several authors have suggested to initially conservative treatment including a period of immobilisation in external rotation followed by a progressive physical therapy centred on the rotator cuff, deltoid muscle and periscapular musculature [13]. Nonoperative treatment should be followed by an education about the positions and activities that can put the shoulder at risk. Marquardt et al. compared the results of surgical versus conservative treatment in 24 patients with failed anterior shoulder stabilisation [14]. Patients revised surgically had significantly better results on the Rowe scale, although the number of procedures affected negatively the final outcome and patient subjective satisfaction.

Other factors like age and athletic activity are key therapeutic decisions since young patients practising risky sports carry a higher risk of failure. In these patients, it is important to perform a surgical technique that allows the athlete to resume this activity [7, 15]. Regarding those subjects who practise contact or overhead sports, Neyton et al. reported excellent results with the technique of Latarjet [16]. In elderly patients, the possibility of rotator cuff tear associated to shoulder instability should always be kept in mind.

3.2.2.2 What Are the Shoulder Abnormalities to Be Repaired?

Rowe et al. [13] demonstrated that in more than 85 % of the shoulders with postoperative instability, a lesion responsible for this poor result could be identified. Moreover, Meeham and Petersen [12] proved that in almost half of the cases, there were multiple lesions. First, to properly address failed surgical treatment of shoulder instability, it is essential to recognise the direction of the instability, as well as the abnormalities and associated factors responsible for the poor results. Clinical history and meticulous physical examination allow to identify the direction of the instability and provide evidence about the possible cause of failure and potential associated lesions. Whereas instability symptomatic in trivial situations like while sleeping or in daily activities suggests a persistent capsular laxity or a glenoid bone defect, a posttraumatic dislocation that requires a reduction suggests a rupture of previous surgical stabilisation or large Hill-Sachs lesion. Physical examination should be performed compared to the contralateral shoulder. Subscapularis muscle function should always be explored, especially in patients with previous open surgery. Sachs et al. [17] found that 25 % of the patients undergoing open surgery had a deficient subscapularis function and only 57 % of them obtained good or excellent results after revision surgery.

Imaging exams are essential to identify articular abnormalities and to quantify possible bone defects. Conventional radiographs, anteroposterior and axillary views, identify bone defects in the glenoid and in the humeral head, and numerous specific radiographic views have been proposed to assess bone defects. These modalities have been overtaken by other diagnostic methods. The computed tomography (CT) scan is ideal to quantify bone defects. 3D tomography with humeral head subtraction facilitates quantification of glenoid bone defect related to the total area and depth of the defect. Arthro-CT and arthro-MRI can identify soft tissue injuries, rotator cuff tears, HAGL lesions, capsulolabral lesions and laxity or rupture of the joint capsule [18].

3.2.2.3 Did the Patient Have a New Trauma?

Some patients that were adequately diagnosed and treated sustained a postoperative episode of instability simply because they had a new trauma. This group of patients should be approached like primary cases searching for potential new injuries and managing them accordingly.

3.2.3 Treatment Options

Several authors have reported the distribution of anatomical changes found in open and arthroscopic revision surgery of shoulder instability. Capsular hyperlaxity, a persistent Bankart lesion or a Bankart lesion medially healed were the most frequent abnormalities in addition to bone defects [13, 19].

It is known that the recurrence of instability is significantly higher in patients with anterior glenoid bone defects [7, 15, 20]. The morphology of the glenoid in inverted pear is a clear risk factor, and we observed that a bony Bankart involving 15 % of the articular surface can increase significantly the risk of recurrence, and should be reconstructed [7]. The results of techniques based on the reconstruction of the glenoid joint surface using bone grafting have been satisfactory, but a correct position of the graft is critical. Whereas medial positioning of the graft has been associated with persistent instability, a position too lateral takes a higher risk of glenohumeral osteoarthritis. The possibility of performing these techniques arthroscopically with good preliminary results has been recently published [21, 22]. Other techniques to reconstruct the bony Bankart using autograft from the iliac crest or allograft from cadaveric distal tibia or glenoid have also been described [23, 24].

Bone defects in the humeral head have also been incriminated as a cause of failure of shoulder stabilisation surgery, especially in large Hill-Sachs lesions that engage in the anterior glenoid rim in external rotation [25]. On the other hand, the rationale of the techniques usually recommended to treat engaging Hill-Sachs lesions is filling the bone defect. The technique of remplissage consists of filling the bone defect by capsulotenodesis including the posterior joint capsule and the infraspinatus muscle tendon to prevent engagement making the bone injury in extra-articular lesion [26].

Addressing soft tissue problems is also extremely important in revision surgery for instability since technical errors, especially labrum reattachment in a medial position, are common (Fig. 3.2). In addition, it is necessary to repair the anteroinferior capsulolabral complex. When a nonanatomical repair is encountered, glenoid labrum dissection and reinsertion to its anatomical position are recommended. In other cases, a redundant joint capsule, a capsule with poor quality tissue or with an unbalanced asymmetric repair can be involved in recurrent instability. Several authors have recommended that surgical revision of failed shoulder stabilisation should be performed with

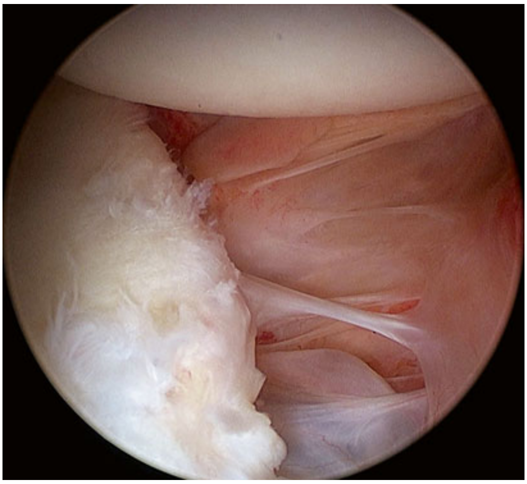

Fig. 3.2 Labrum reattachment in a medial position

open surgery, but numerous studies have provided data on the superiority of arthroscopic revision. Arthroscopy permits direct visualisation of the pathology responsible for the recurrence, including unexpected causes that can be corrected at the same procedure, like SLAP or HAGL lesions, loose bodies, rotator cuff tears or chondral lesions. Furthermore, some bony lesions on the surface glenoid or humeral head or severe weak capsular can be solved by arthroscopy [27, 28].

3.3 Arthroscopic Management of Failed Bankart Procedure with Bone Block Procedure

Ettore Taverna, Guido Garavaglia, Carlo Perfetti, Henri Ufenast, Ferdinando Battistella, Laura Broffoni, and Riccardo D'Ambrosi

3.3.1 Introduction

Eventually patients with significant bone loss at the glenoid, given the unacceptably high rate of recurrent dislocation and subluxation after arthroscopic soft tissue repair, are candidates for open or arthroscopic "bony procedures" [21, 29, 30].

An erosion of the glenoid is quite a common phenomenon in recurrent anterior shoulder instability [5]. Biomechanical studies have found an inverse relationship between the size of the glenoid defect and the stability of the shoulder. The stability of the shoulder progressively decreases as the size of the osseous defect increases [31].

A large defect of the glenoid must be treated with bone grafting to the glenoid when a Bankart procedure is performed [32]. Many authors recommend coracoid transfer if the glenoid rim deficiency involves 25 % of the anterior-posterior diameter of the glenoid. Others suggest that measures to restore the arc of glenoid concavity may be beneficial, in terms of both stability and motion, for patients who have a glenoid defect whose width is at least 21 % of the glenoid length [30, 31]. Only recently arthroscopic techniques, positioning a bone graft at the anterior-inferior aspect of the glenoid, have been described [21, 30].

3.3.2 Surgical Technique: The Arthroscopic Bone Block Procedure (Fig. 3.3)

The arthroscopic anterior bone block procedure described by Taverna et al. [33] combines an arthroscopic Bankart repair with the transfer of the tip of the iliac crest graft that is passed through a cannula placed in the rotator interval and fixed on the glenoid rim under the equator. The efficacy of this procedure is related to the bone block effect provided by the tricortical bone graft that increases the size of the glenoid surface and the

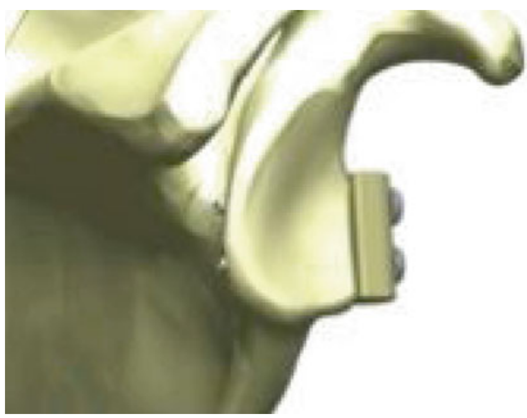

Fig. 3.3 The arthroscopic bone block procedure by Taverna

concavity recreation provided by the labral repair and capsular and ligaments shift and repair. The goal of the procedure is to restore the normal anatomy of the unstable shoulder with bone defects.

The advantages of this procedure are the associated repair of the glenoid labrum and tensioning and shift of capsule and ligaments. As for the Bristow-Latarjet procedure, the bone block is placed in an extra-articular position, preventing synovial fluid from coming in contact with the bone graft, thus avoiding the potential contact between the humeral head and the coracoid bone block with the screw, which can cause pain and glenohumeral osteoarthritis. This is an anatomical procedure with the restoration of normal glenohumeral anatomy, increasing the damaged glenoid bony support and the normal insertion of the labrum, ligaments and capsule. Compared to the Bristow and Latarjet procedure, the damage of the subscapularis fibres is minimal. The weakest point of this procedure is the impossibility to face the combination of an unstable shoulder with glenoid bone defects and capsular-labrum-ligaments inconsistency. In this case, the dynamic musculotendinous sling effect created by the conjoint tendon passing over the inferior part of the subscapularis of the Latarjet and Bristow procedures is mandatory.

3.3.3 Rehabilitation

Postoperatively, we recommend to keep the patients in a sling for a period of 3 weeks. After the immobilisation, there is no limitation in passive motion and the patients are allowed to a full recovery in elevation and external rotation. After complete wound healing is obtained, swimming pool active exercises are recommended and resumed working activities are allowed. Progressive strengthening exercises are started after 6–8 weeks. Return to overhead and contact sports are generally allowed after 4–6 months post surgery.

3.3.4 Conclusion

The effect of a glenoid defect on shoulder stability continues to be investigated. To date, there is no consensus when and how bony procedures are needed to stabilise a glenohumeral joint. Further investigations are needed to determine the amount of bone loss that significantly affects the recurrence rate of an isolated soft tissue repair in an unstable shoulder [34].

For the future, we think that the goal of an arthroscopic procedure for stabilising a glenohumeral joint should involve restoration of the arc of glenoid concavity and when it is possible along with labrum repair and capsule and ligamentous tension.

3.4 Arthroscopic Management of Failed Anterior Instability Repair with Latarjet-Lafosse Procedure

J. Leuzinger, Ch. Sternberg, and D. Meraner

There is still an ongoing controversial discussion about how to treat failed anterior shoulder instability repair. Open Bankart repair with refixation of the labrum at the glenoid neck was the golden standard for decades. Since the 1990s, this procedure has also been performed using the arthroscopic technique. Subsequent studies showed higher re-instability rates up to 15 % [35], depending on the age and activity level of the patients.

The Latarjet procedure, a modification of the Bristow procedure, has been indicated for the treatment of anterior glenohumeral instability with associated glenoid bone loss of up to 40 % as well as for patients who have undergone anterior shoulder stabilisation procedures that were unsuccessful [36].

The coracoid transfer was first described in 1954 by Latarjet [37]. The coracoid and conjoint tendon are transferred by a subscapularis tendon split and refixed to the glenoid with screws. The benefit of this so-called nonanatomical procedure is a stable fixation on the glenoid and the conjoint tendon used as a hammock to avoid further dislocation of the humeral head. This procedure has excellent results described in literature [38] with low redislocation rates and good postoperative range of motion. Even if it is a nonanatomical procedure, there seems to be no higher osteoarthrosis rate.

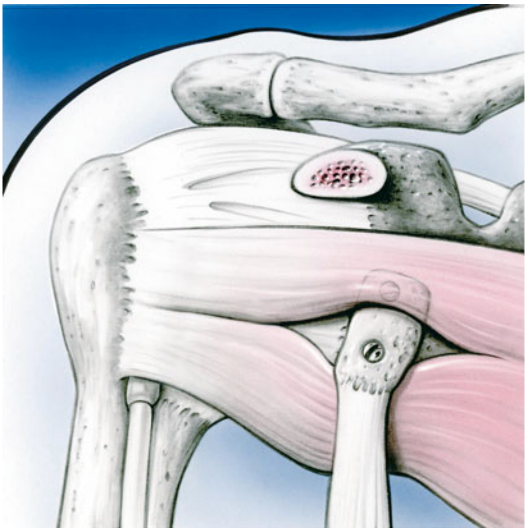

Fig. 3.4 Latarjet procedure

Fig. 3.5 Pseudarthrosis of Latarjet procedure and breakage of the screw

Since 2003, the Latarjet procedure has also been performed using the arthroscopic technique as described first by Lafosse [22] (Fig. 3.4). The first results of this technique seem to be promising [21, 22].

We have been using the Latarjet-Lafosse procedure since 2008 and we find it a very useful and practicable procedure for failed traumatic anterior instability. In our opinion, it is also a good technique for treating traumatic glenohumeral instability, even without severe bone loss or previous failed surgery in young active patients with an ISIS score >6 [3]. The advantage of an arthroscopic procedure is less trauma for the soft tissue and a good visibility of the axillary and musculocutaneous nerve to avoid any damage to these important structures. We use the DePuy Mitek instruments for the standardised procedure. Getting back of the coracoid after screw fixation with the pistol. New fixation to the ventral glenoid after preparing the bone stock with two screws. Postoperatively, we allow free mobilisation depending on the patient's pain, only using an arm sling in public.

We believe that this procedure provides a very good alternative to classic Bankart repair or other anatomical bone block procedures and might even be useful in cases of hyperlaxity of the glenohumeral joint. Further studies will show the long-term efficacy of this procedure. A very shallow learning curve of this technique should limit its use experienced shoulder surgeons.

3.5 Management of Failed Latarjet Procedure

Christophe Charousset

Latarjet shoulder stabilisation is an effective surgical treatment for recurrent shoulder instability associated or not with glenoid bone loss. The Latarjet procedure guarantees a very moderate level of recurrence [38, 39]. Griesser and Al made a systematic review of multiple medical databases which included studies reporting outcomes with complication and reoperation rates following original or modified versions of the Bristow or Latarjet. They found that original or modified Bristow and Latarjet procedures have a 30 % complication rate. Rates of recurrent dislocation and reoperation were 2.9 and 7 %, respectively.

1. Potential complications that may even be of greater entity such as mobilisation or breakage of the screws, absorption of the bone block (Fig. 3.5). These complications can be reoperated by the modified Eden-Hybinette [40] operation as a salvage procedure.

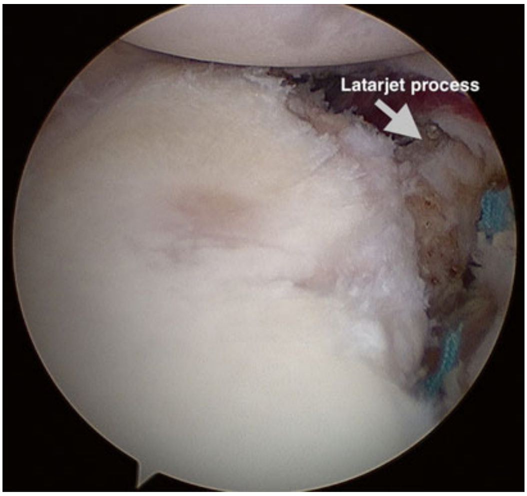

Fig. 3.6 Labral reattachment and capsuloligamentous retensioning with suture anchors

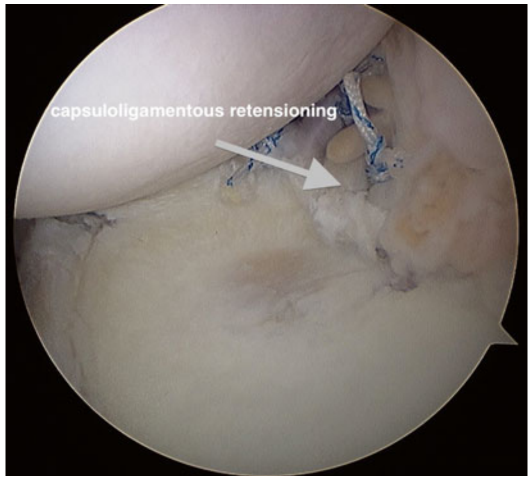

Fig. 3.7 Labral reattachment and capsuloligamentous retensioning with suture anchors

All modified Eden-Hybinette operations were done through a deltopectoral approach, and the subscapularis was approached from the lateral side, taking care to stay on the muscle. The subscapularis was split, allowing removal of preexisting hardware and preparation of the anterior glenoid neck. A wedge-shaped bicortical graft, typically measuring 2–3 cm, was taken from the outer table of the iliac crest. The graft was fixed to the anterior glenoid, flushed or slightly overhanging the joint, with two malleolar screws.

We obtained excellent and good results, but some patients developed glenohumeral arthritis, and rare patients may still experience a sensation of apprehension.

2. The other potential complication is, in some cases, a persistent shoulder pain and/or a globally unstable joint, which would be termed a non-success rather than a complication in itself [41]. In these cases, the question that arises is what technique to apply, considering that the Latarjet procedure tends to determine significant subversion of the shoulder anatomy. Arthroscopy can be beneficial for these subjects and guarantees satisfactory results.

   In our technique, labral reattachment and capsuloligamentous retensioning with suture anchors were performed in all cases (Figs. 3.6 and 3.7). Boileau et al. [27] reported that arthroscopic revision of failed open anterior shoulder stabilisation provides satisfactory results in a selected patient population. Some persistent pain and osteoarthritis progression remain concerns. The main advantages of the arthroscopic approach are the avoidance of anterior dissection in front of the subscapularis, which places the axillary nerve at risk, and the ability to address the various soft tissue pathologies encountered.

3.6 Management of Multioperated Patients

L. Neyton

3.6.1 Introduction

The management of failed anterior instability repair in multioperated patients is a challenge. The need for multiple surgeries in a single unstable patient can ensue from wrong initial diagnosis, wrong indication, technical error or postoperative complication. Many times, the reason for failure of multiple surgeries is a combination of all these reasons. Therefore, a precise analysis of the patient's situation is mandatory. The choice of the new treatment option will be conditioned by the clinical presentation at the time of consultation but also by the unique history of each patient of this difficult-to-treat population.

3.6.2 Analysis of Patient's Situation

3.6.2.1 Initial Diagnosis

What was the diagnosis for the first operation? Recurrent dislocations, subluxations? Pain? Hyperlaxity? Combined instability and hyperlaxity?

Did the patient have a bone loss on the glenoid side? Hill-Sachs lesion? Bankart lesion? HAGL lesion?

3.6.2.2 Previous Surgeries

How many procedures were already performed? It is of importance to obtain the operative notes of any previous surgeries. What kind of procedure was performed? Arthroscopic or open procedure? Soft tissue repair (labrum, capsule, remplissage, HAGL)? Bone block procedure? Coracoid transfer, iliac crest?

Devices used (metallic or absorbable anchors, screws).

What were the approaches? How was the subscapularis managed? (Section, discission) How many times?

What was the reason for failure of the procedure? Fracture, non-union, malposition of a bone block? Recurrence after isolated Bankart procedure in a patient with bone defect?

What were the sequences of the multiple surgeries?

3.6.3 Actual Situation

3.6.3.1 Clinical Examination

Range of motion (focus on hyperlaxity in external rotation, i.e. passive external rotation with elbow at side >85°), specific tests of instability and rotator cuff (focus on subscapularis). Neurologic tests.

Skin examination locates the previous scars.

What are the symptoms? Recurrent anterior instability? Pain? Weakness?

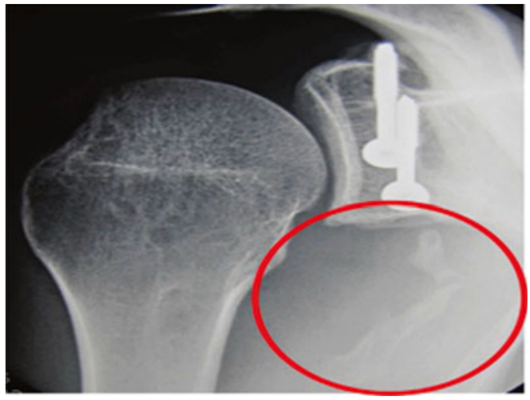

Fig. 3.8 Fracture and non-union of Latarjet coracoid bone block. Large Hill-Sachs lesion

Oftentimes, multioperated patients will present a combination of these symptoms.

3.6.3.2 Imaging

- Plain X-rays (AP views in neutral, internal, external rotation, axillary Bernageau view, outlet view): Assessment of glenohumeral joint space, acromiohumeral distance. Status of the coracoid process. Previous surgeries (anchors, screws, bone block, glenoid defects, Hill-Sachs lesion) (Figs. 3.8 and 3.9).
- MRI scan is helpful in the situation of the use of non-metallic devices during previous surgeries.
- CT arthrogram +++ analysis of glenoid bone status. Size of Hill-Sachs lesion, cuff tendon and muscle status (subscapularis+++).

EMG study is required in case of clinical doubt.

3.6.4 Treatment

First, the physician must explain to the patient his own situation. Why the previous operations failed. We recommend the physician may take some time to consider the case and not take a decision alone. Analysis of these difficult cases should be better discussed with other physicians. Any additional investigation should be performed to have a thorough analysis.

Fig. 3.9 Partial superior lysis of the bone block and excessive anterior pouch

Second, the physician should ask himself if a new operation is required and why should a new operation be better than the previous ones.

Third, every surgical technique, arthroscopic or open, should be considered as an option.

Soft tissue procedures [27]: capsular/labral reattachment, plication, Hill-Sachs remplissage [26], posterosuperior rotator cuff repair, subscapularis repair, imbrication, and pectoralis major transfer.

Bony procedures: coracoid transfer (Latarjet), iliac crest bone block (Eden-Hybinette) [40, 42], allograft of Hill-Sachs lesion, humeral derotational osteotomy (Weber) and fusion.

3.6.5 Conclusion

Fortunately, the vast majority of unstable patients will be stable after a single operation. However, recurrence of instability can occur after surgery. Management of failed multioperated patients is a challenge. Beyond surgical options, careful analysis of the unique situation of the patient is required. Benefits/risks balance of a new surgical operation must be discussed and chance of success estimated. When a new surgery is scheduled, technical options should be chosen in the light of the previous surgeries and actual situation of the patient.

References

1. Dowdy PA, O'Driscoll SW. Shoulder instability. An analysis of family history. J Bone Joint Surg Br. 1993;75:782–4.
2. Hovelius L, et al. The coracoid transfer for recurrent dislocation of the shoulder. Technical aspects of the Bristow-Latarjet procedure. J Bone Joint Surg Am. 1983;65:926–34.
3. 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 Joint Surg Br. 2007;89:1470–7.
4. Thomazeau H, et al. Can we improve the indication for Bankart arthroscopic repair? A preliminary clinical study using the ISIS score. Orthop Traumatol Surg Res. 2010;96:S77–83.
5. Saito H, et al. Location of the glenoid defect in shoulders with recurrent anterior dislocation. Am J Sports Med. 2005;33:889–93.
6. Charousset C, et al. Can we improve radiological analysis of osseous lesions in chronic anterior shoulder instability? Orthop Traumatol Surg Res. 2010;96:S88–93.
7. Calvo E, Granizo JJ, Fernández-Yruegas D. Criteria for arthroscopic treatment of anterior instability of the shoulder: a prospective study. J Bone Joint Surg Br. 2005;87:677–83.
8. Skendzel JG, Sekiya JK. Diagnosis and management of humeral head bone loss in shoulder instability. Am J Sports Med. 2012;40:2633–44.
9. Romeo AA, Cohen BS, Carreira DS. Traumatic anterior shoulder instability. Orthop Clin North Am. 2001;32:399–409.
10. Franceschi F, et al. Arthroscopic salvage of failed arthroscopic Bankart repair: a prospective study with a minimum follow-up of 4 years. Am J Sports Med. 2008;36:1330–6.
11. Millar NL, Murrell GAC. The effectiveness of arthroscopic stabilisation for failed open shoulder instability surgery. J Bone Joint Surg Br. 2008;90:745–50.
12. Meehan RE, Petersen SA. Results and factors affecting outcome of revision surgery for shoulder instability. J Shoulder Elbow Surg/Am Shoulder Elbow Surg. 2005;14:31–7.
13. Rowe CR, Zarins B, Ciullo JV. Recurrent anterior dislocation of the shoulder after surgical repair. Apparent causes of failure and treatment. J Bone Joint Surg Am. 1984;66:159–68.
14. Marquardt B, et al. Outcome after failed traumatic anterior shoulder instability repair with and without

surgical revision. J Shoulder Elbow Surg/Am Shoulder Elbow Surg. 2007;16:742–7.
15. Boileau P, et al. Risk factors for recurrence of shoulder instability after arthroscopic Bankart repair. J Bone Joint Surg Am. 2006;88:1755–63.
16. Neyton L, 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 Elbow Surg/Am Shoulder Elbow Surg. 2012;21:1721–7.
17. Sachs RA, Williams B, Stone ML, Paxton L, Kuney M. Open Bankart repair: correlation of results with postoperative subscapularis function. Am J Sports Med. 2005;33:1458–62.
18. Probyn LJ, White LM, Salonen DC, Tomlinson G, Boynton EL. Recurrent symptoms after shoulder instability repair: direct MR arthrographic assessment – correlation with second-look surgical evaluation. Radiology. 2007;245:814–23.
19. Shah AS, Karadsheh MS, Sekiya JK. Failure of operative treatment for glenohumeral instability: etiology and management. Arthrosc J Arthrosc Relat Surg Off Publ Arthrosc Assoc North Am Int Arthrosc Assoc. 2011;27:681–94.
20. Voos JE, et al. Prospective evaluation of arthroscopic bankart repairs for anterior instability. Am J Sports Med. 2010;38:302–7.
21. Boileau P, Mercier N, Roussanne Y, Thélu C-É, Old J. Arthroscopic Bankart-Bristow-Latarjet procedure: the development and early results of a safe and reproducible technique. Arthrosc J Arthrosc Relat Surg Off Publ Arthrosc Assoc North Am Int Arthrosc Assoc. 2010;26:1434–50.
22. Lafosse L, Boyle S. Arthroscopic Latarjet procedure. J Shoulder Elbow Surg/Am Shoulder Elbow Surg. 2010;19:2–12.
23. Warner JJP, Gill TJ, O'hollerhan JD, Pathare N, Millett PJ. Anatomical glenoid reconstruction for recurrent anterior glenohumeral instability with glenoid deficiency using an autogenous tricortical iliac crest bone graft. Am J Sports Med. 2006;34:205–12.
24. 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. Arthrosc J Arthrosc Relat Surg Off Publ Arthrosc Assoc North Am Int Arthrosc Assoc. 2009;25:446–52.
25. 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. Arthrosc J Arthrosc Relat Surg Off Publ Arthrosc Assoc North Am Int Arthrosc Assoc. 2000;16:677–94.
26. Purchase RJ, Wolf EM, Hobgood ER, Pollock ME, Smalley CC. Hill-sachs 'remplissage': an arthroscopic solution for the engaging hill-sachs lesion. Arthros J Arthrosc Relat Surg Off Publ Arthrosc Assoc North Am Int Arthrosc Assoc. 2008;24:723–6.
27. Boileau P, Richou J, Lisai A, Chuinard C, Bicknell RT. The role of arthroscopy in revision of failed open anterior stabilization of the shoulder. Arthrosc J Arthrosc Relat Surg Off Publ Arthrosc Assoc North Am Int Arthrosc Assoc. 2009;25: 1075–84.
28. Arce G, Arcuri F, Ferro D, Pereira E. Is selective arthroscopic revision beneficial for treating recurrent anterior shoulder instability? Clin Orthop. 2012;470: 965–71.
29. Milano G, et al. Analysis of risk factors for glenoid bone defect in anterior shoulder instability. Am J Sports Med. 2011;39:1870–6.
30. Piasecki DP, et al. Glenoid bone deficiency in recurrent anterior shoulder instability: diagnosis and management. J Am Acad Orthop Surg. 2009;17: 482–93.
31. 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;82:35–46.
32. Yamamoto N, et al. Effect of an anterior glenoid defect on anterior shoulder stability: a cadaveric study. Am J Sports Med. 2009;37:949–54.
33. Taverna E, Golanò P, Pascale V, Battistella F. An arthroscopic bone graft procedure for treating anterior-inferior glenohumeral instability. Knee Surg Sports Traumatol Arthrosc Off J ESSKA. 2008;16:872–5.
34. Beran MC, Donaldson CT, Bishop JY. Treatment of chronic glenoid defects in the setting of recurrent anterior shoulder instability: a systematic review. J Shoulder Elbow Surg/Am Shoulder Elbow Surg. 2010;19:769–80.
35. Koss S, Richmond JC, Woodward Jr JS. Two- to five-year followup of arthroscopic Bankart reconstruction using a suture anchor technique. Am J Sports Med. 1997;25:809–12.
36. Shah AA, et al. Short-term complications of the Latarjet procedure. J Bone Joint Surg Am. 2012;94: 495–501.
37. Latarjet M. Treatment of recurrent dislocation of the shoulder. Lyon Chir. 1954;49:994–7.
38. 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;80:841–52.
39. Hovelius LK, et al. Long-term results with the Bankart and Bristow-Latarjet procedures: recurrent shoulder instability and arthropathy. J Shoulder Elbow Surg/Am Shoulder Elbow Surg. 2001;10:445–52.
40. Lunn JV, Castellano-Rosa J, Walch G. Recurrent anterior dislocation after the Latarjet procedure: outcome after revision using a modified Eden-Hybinette operation. J Shoulder Elbow Surg/Am Shoulder Elbow Surg. 2008;17:744–50.
41. Griesser MJ, et al. Complications and re-operations after Bristow-Latarjet shoulder stabilization: a systematic review. J Shoulder Elbow Surg/Am Shoulder Elbow Surg. 2013;22:286–92.
42. Walch G, Charret P, Pietro-Paoli H, Dejour H. Anterior recurrent luxation of the shoulder. Postoperative recurrences. Rev Chir Orthop Reparatrice Appar Mot. 1986;72:541–55.

Elbow Arthroscopy: From Basic to Advance

4

Paolo Arrigoni, Alexander Van Tongel, Denise Eygendaal, Grzegorz Adamczyk, and Luigi Pederzini

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4.1 Introduction

Elbow arthroscopy has dramatically improved following clinical and cadaveric studies with understanding of portal placement and the relative positions of surrounding neurovascular structures. Thanks to the more precise surgical techniques and instrumentation (e.g., arthroscopic pumps, trocars, motorized instruments), elbow arthroscopy is nowadays a more safe and effective treatment modality for several elbow pathologies. Most of the indications for open surgery of the elbow could be considered to be indications for arthroscopic or arthroscopically assisted management. As with any procedure, there is a learning curve involved in establishing safe portals and avoiding neurovascular injury.

4.1.1 Positioning

The ideal position for elbow arthroscopy is ultimately determined by the experience and comfort level of the surgeon; however, several general

S. Zaffagnini et al. (eds.), *ESSKA Instructional Course Lecture Book*, DOI 10.1007/978-3-642-53983-1_4, © ESSKA 2014

Fig. 4.1 Intraoperative picture of anterior compartment work

principles apply to all positions. The patient's bony prominences should be well padded and the antecubital space of the operative arm should remain free of constriction to allow for distention of the anterior capsule.

The elbow should be positioned in 70–80° of flexion allowing for maximal distention of the elbow (Fig. 4.1). Finally, a tourniquet should be placed proximal sufficient on the arm to prevent constriction of the antecubital space, yet allow for hemostasis and visualization of the joint. Patients may be positioned in one of four ways for elbow arthroscopy: supine, supine-suspended, prone, and lateral decubitus. Each position has its advantages and disadvantages. Prone or lateral are probably the most accepted ones at the moment.

4.1.2 Instrumentation

The preferred arthroscopic system used in elbow arthroscopy is a 4.0-mm, 30° arthroscope. On occasion, a 2.7-mm arthroscope can be helpful for navigating through smaller spaces or joints. Careful monitoring of fluid pressures using gravity or pump pressure control (30 mmHg) is required to minimize risk of fluid extravasation and compartment syndrome. Retractors and switching sticks can be very helpful and further instrumentation (e.g., biters, shavers, osteotomes) depends on the procedure.

4.1.3 Portals

The bony and ligamentous anatomy of the elbow joint serves to divide it into three major arthroscopic compartments: anterior, posterior, and posterolateral. The initial portal for joint visualization is a matter of surgeon preference but is dictated to some extent by the underlying pathology to be addressed. Multiple portal sites have been described in the literature; however, the most common portals utilized are the anterolateral, midlateral "soft spot portal," anteromedial, proximal anteromedial, proximal anterolateral, straight posterior, and posterolateral.

Before trocar placement, a key facet of portal creation is the distention of the joint with normal saline (15–20 ml). This serves to increase the maximum distance from the articular surface to the neurovascular structures and, hence, decreases the risk of iatrogenic injury.

4.1.4 Safety Considerations

Significant disruption of the normal anatomy as a result of trauma or deforming arthritis may be a relative contraindication. Subluxation of the ulnar nerve is not longer considered as a contraindication. In these patients, the proximal anteromedial portals can be established by reducing and holding the nerve behind the epicondyle with a thumb while establishing or entering the portal. Arthroscopy of the elbow after ulnar nerve transposition must be based on the degree of certainty with which the nerve can be localized by palpation in the region of the planned portal. Arthroscopy should not be done in the presence of local soft tissue infection in the area of the portal sites. As in open procedures, a perfect knowledge of the anatomy, good visualization (use of retractors), and a clear safety-driven strategy are the most important factors for increasing the safety of this procedure.

4.2 State-of-the-Art Treatments

4.2.1 Tennis Elbow

The name tennis elbow is derived from the description of "lawn tennis arm," introduced by Morris in 1882. Tennis, however, contributes in only 5–10 % of all cases. It is rather related to manually intensive work, requiring forceful and repetitive rotation of the forearm and wrist extension or flexion (e.g., in mechanics, butchers, construction workers). The incidence of lateral humeral epicondylitis in general practice is estimated at 4–7 per 1,000 patients per year, with a peak between 35 and 54 years of age. The prevalence ranges from 1 to 3 % in the general population. Usually, the dominant arm is involved. Men and women are equally affected, according to most studies. The actual pathophysiology of tennis elbow is still not completely understood. Although many researchers have proposed numerous theories regarding the pathophysiology, the current consensus is that repetitive trauma results in microtears and hypoxic degeneration of the common extensor tendon, especially the extensor carpi radialis brevis (ECRB) portion. Microscopic evaluation of the involved tissue shows disruption of the normal collagen architecture with ingrowth of fibroblastic and granulation tissue.

Conservative treatment of tennis elbow will yield excellent results in the vast majority of patients. Elbow tendinosis is self-limiting within 12 months in up to 90 % of patients. The "wait and see" policy has many advantages in the well-informed patient. Most surgeons will not consider surgery before a period of 9–12 months of "adequate" conservative treatment has been offered to the patient. However, there is no consensus in the literature and among surgeons on which means of conservative treatments has the best chance of success.

Many options have been suggested ranging from rest and activity modification to injections of various substances and ESWT. Shock wave therapy (ESWT) is no longer considered to be effective.

The most important aspect of conservative therapy of tendinosis of the elbow is counseling the patient about the pathology. If possible, overuse or disuse should be identified and the cause should be addressed. Furthermore, the patient should be informed about relatively benign character of the pathology and possible treatment options.

Some studies have shown a positive effect of physiotherapy. Evidence on the use of acupuncture, botulinum toxin therapy, PRP, or autologous blood injection for treatment of lateral epicondylitis is still insufficient, and therefore no conclusions can be drawn regarding its role in treating lateral epicondylitis.

Orthoses are more controversial. There are many designs, and marketing or commerce may play a role in prescribing braces in the treatment of elbow tendinosis. Although the basic pathology of the tendon origin may not be inflammatory, oral NSAIDs may decrease symptoms. No difference between different NSAIDs has been shown nor have NSAIDs been shown to alter the natural course. The long-term use of NSAIDs cannot be recommended in the treatment of elbow tendinosis due to the potentially severe side effects. The use of topical NSAIDs is under investigation and may prove to be a safer alternative.

Surgical techniques are various, including open, percutaneous, and arthroscopic treatment. Method of treatment consists mainly of tenotomy (tendon release) or excising pathological tissue. Research investigating which approach is superior reveals that the less-invasive approaches (percutaneous or arthroscopic) allow faster return to work than the open procedure. Although every surgical procedure has its advantages and disadvantages, no technique appears to be superior.

4.2.2 Posterolateral Rotatory Instability, Role of R-LCL and Arthroscopic Diagnosis

Injury to the LCL complex resulting in a posterolateral rotatory instability has been reported. Some authors have attributed the cause of PLRI to the U-LCL. However, anatomical studies have indicated that more than half of the cadavers lack an obvious and thick lateral ulnar collateral ligament. A recent arthroscopic cadaver model series has shown that the U-LCL can be transected without inducing posterolateral rotatory instability of the elbow and that both injuries to the R-LCL and the U-LCL are necessary to cause significant PLRI. This observation is supported by a recent in vivo study on the isometric point of the lateral ligament of the elbow. The authors highlight that the R-LCL is essentially isometric, while U-LCL is not isometric in the flexion-extension arc. They conclude that the R-LCL appears to be more important than the U-LCL in preventing PLRI. An arthroscopic technique is more accurate than an open "classic" one to evaluate the possible laxity of R-LCL.

4.2.3 Associated Pathology in Minor Instability

The advantage of an arthroscopic technique is the detection and treatment of 69 % of the associated pathologies like plicae, loose bodies, or degenerative changes.

4.2.4 Arthroscopic Evaluation of Medial Instability

Arthroscopy has been found as an unvaluable tool to evaluate medial elongation/incompetency of the medial collateral ligament too. At 70° of flexion, a valgus stress can be applied evaluating the degree of medial opening. Based on this consideration, it becomes possible to decide for a full MCL reconstruction versus simple plications/shaving.

Fig. 4.2 MRI showing a radial head fracture

4.2.5 Posteromedial Impingement (Valgus Overload)

Posteromedial impingement is a progressive loss of extension and ulnar nerve symptoms. The starting condition is an elongation of the medial collateral ligament that determines a valgus opening in flexion. Progressively, a posteromedial osteophyte limits the extension. The treatment includes open ulnar nerve release, arthroscopic excision of posteromedial osteophyte, and evaluation of medial collateral ligament elongation.

4.2.6 Fractures

Arthroscopy is definitively gaining a role in understanding and treating some types of articular fractures such as median single fragmented radial head fractures and single fragmented coronoid fractures (Fig. 4.2).

4.2.7 Elbow OCD

Osteochondritis dissecans of the elbow is a pathology of the cartilage and subchondral

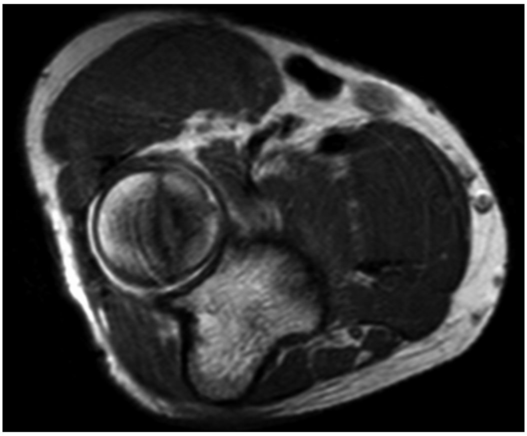

Fig. 4.3 OCD plug detaching from the capitellum

bone with a high incidence between 10 and 15 years involving generally the capitulum humeri. It begins after the complete ossification of capitulum humeri and must be distinguished by Panner disease. This is an osteochondrosis of the capitulum in patients between 7 and 12 years of age, during the ossification phase. The initial symptom is moderate pain with an extension deficit of 5–20°. Pain can be greater at the lateral side of the elbow. Symptoms can solve spontaneously without sequelae. OCD symptoms include unlocalized pain and muscular contraction. Initially, the x-ray may be negative, while MRI is the gold standard imaging system (Fig. 4.3) then intra-articular fragments may appear, and modifications of the shape of capitulum humeri are often present.

Nonsurgical treatment is an initial option. Surgical treatment varies widely and includes arthroscopic drilling of intact lesions, securing of cartilage flap lesions with pins or screws, drilling, mosaicplasty, and the osteoarticular transfer system (OATS).

Surgical treatment is indicated in grades 3–4 ICRS, when the OCD plug becomes unstable. Arthroscopic drilling may be performed by an anterograde (from the front) or by a retrograde (from behind) approach.

4.2.8 The Stiff and Degenerative Elbow and the Osteocapsular Arthroplasty

Stiff elbows can be classified as intrinsic, extrinsic, or mixed. Posttraumatic or degenerative causes can also bring to, respectively, arthrofibrotic or arthritic elbow. A possible posttraumatic initial cause can determine a degenerative clinical picture. Arthroscopic osteocapsular arthroplasty is the choice of treatment when bone congruency is maintained. In case of bony ossifications of the capsule, combined retraction of medial and lateral collateral ligaments, and previous surgery with transposition of the nerves, open surgical treatment is recommended.

The arthroscopic technique in stiff elbow mainly consists of:

- Prone/modified lateral decubitus
- Ulnar nerve isolation (open)
- Three posterior portals and two anterior portals (associated portals for retractors can be used posteriorly or anteriorly)
- Posterior and anterior debridement
- Removal of osteophytes, hypertrophic olecranon, and coronoid
- Posterior and anterior capsulectomy

A 5-rule progressive technique has to be kept in mind:

1. Ulnar nerve isolation.
2. Enter the joint from posterolateral.
3. Use two accessory portals in under tricipital recess.
4. Start medial to lateral in the anterior compartment to perform capsulectomy.
5. Use retractors to avoid complications.

In the postoperative phase, a dedicated rehabilitation program is indicated:

- Maintain axillary plexus anesthesia.
- Backslab in full extension and removal after 10 days.
- CPM five times per day for 40 min.
- Active assisted mobilization 1 day post-op.
- Fans (indomethacin).

Complications can concern neurological, vascular major complications. Synovial fistula and minor transient neurological deficit are described.

4.3 Future Treatment Options

Elbow arthroscopy is a technique in progression. Indications are expanding and safety rules are becoming more and more universally accepted and applied. It's much more a reproducible technique respect to 10 years ago. In the next years, we'll observe an implementation in understanding minor instability problems and their treatment.

4.4 Take-Home Messages

- Prone or modified lateral decubitus are the most consolidated positions for complex elbow arthroscopy.
- Low pump pressure and use of retractors through dedicated portals is indicated.
- Significant disruption of the normal anatomy as a result of trauma or deforming arthritis may be a relative contraindication to arthroscopy.
- In contrast to what is widely thought, tennis contributes in only 5–10 % of all cases of tennis elbow.
- Tennis elbow is rather degenerative than inflammatory; therefore, the term "tendinosis" is more appropriate.
- When OCD plugs become unstable, the operative approach to stabilize or remove of the fragments is indicated.
- Rehabilitation and postoperative treatment with indomethacin is crucial after arthroscopic treatment of stiff and degenerative elbow.

Bibliography

1. Andrews JR, Carson WG. Arthroscopy of the elbow. Arthroscopy. 1985;1(2):97–107.
2. Bauer M, Jonsson K, Josefsson PO, et al. Osteochondritis dissecans of the elbow. A long-term follow-up study. Clin Orthop Relat Res. 1992;284:156–60.
3. Baumgarten TE, Andrews JR, Satter-White YE. The arthroscopic classification and treatment of osteochondritis dissecans of the capitellum. Am J Sports Med. 1998;26:520–3.
4. Blonna D, Wolf JM, Fitzsimmons JS, O'Driscoll SW. Prevention of nerve injury during arthroscopic capsulectomy of the elbow utilizing a safety-driven strategy. J Bone Joint Surg Am. 2013;95:1373–81.
5. Budge MD, Armstrong AD. Elbow arthroscopy: set up, portals, and tools for success. Oper Tech Orthop. 2009;19:209–19.
6. Dodson CC, Nho SJ, Williams III RJ, Altchek DW. Elbow arthroscopy. J Am Acad Orthop Surg. 2008;16:574–85.
7. Duthie RB, Houghton GR. Constitutional aspects of the osteochondroses. Clin Orthop. 1981;158:19–27.
8. Gardiner JB. Osteochondritis dissecans in three family members of one family. J Bone Joint Surg. 1955;37B:139–42.
9. Gugenheim Jr JJ, Stanley RF, Woods GW, Tullos HS. Little League survey: the Houston study. Am J Sports Med. 1976;4(5):189–200.
10. Indelicato PA, Jobe FW, Kerlan RK, Carter VS, Shields CL, Lombardo SJ. Correctable elbow lesions in professional baseball players: a review of 25 cases. Am J Sports Med. 1979;7(1):72–5.
11. Morrey BF, Askew LJ, Chao EY. A biomechanical study of normal functional elbow motion. J Bone Joint Surg Am. 1981;63(6):872–7.
12. Pill SG, Ganley TJ, Flynn JM, et al. Osteochondritis dissecans of the capitellum. Arthroscopic-assisted treatment of large, full thickness defects. Arthroscopy. 2003;19(2):222–5.
13. Sahajpal DT, Blonna D, O'Driscoll SW. Anteromedial elbow arthroscopy portals in patients with prior ulnar nerve transposition or subluxation. Arthroscopy. 2010;26:1045–52.
14. Savoie III FH. Guidelines to becoming an expert elbow arthroscopist. Arthroscopy. 2007;23:1237–40.

Part II

Instructional Courses: Lower Limb

3D Anatomy Versus Arthroscopy Versus Navigation

5

Gianluca Camillieri, Pau Golano,
and Stefano Zaffagnini

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5.1 Computer Functional and Biomechanical Consideration on ACL, PCL and MPFL

5.1.1 Introduction

Computer-assisted surgery (CAS) has been extensively used over the course of the last few decades. At present, ligaments surgery—with great care to anterior cruciate ligament (ACL) injury—and joint arthroplasty represent the major field of application in orthopaedics. The first step in using CAS technology for knee surgery was addressed to improve the accuracy of the intervention (limb alignment or isometric graft) and enhance surgical outcome [3, 18], using information collected pre-operatively via computed tomography (CT) or magnetic resonance imaging (MRI) scans. Indeed, CAS for orthopaedic purposes has entered the mainstream only in the last 10 years, providing, in addition to surgical guidance for reconstructive surgery based on anatomical features, a valuable feedback for kinematic analysis [5]. In fact, with the aid of imageless intra-operative registration, CAS systems are allowed to evaluate the passive range of motion and laxity associated with each specific reconstruction, both ligamentous and prosthetic ones. Besides achieving the correct anatomical features, a global and accurate functional evaluation should be recommended in order to reach the joint parameters as close as possible to its normal function.

S. Zaffagnini et al. (eds.), *ESSKA Instructional Course Lecture Book*,
DOI 10.1007/978-3-642-53983-1_5, © ESSKA 2014

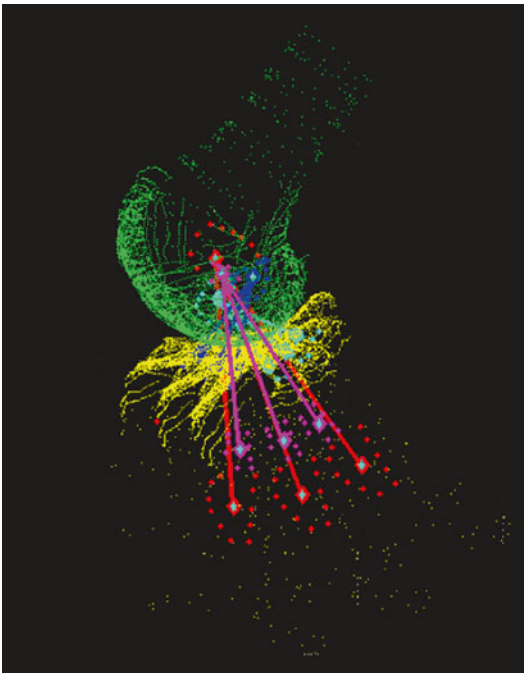

Fig. 5.1 Graphical representation of the data available by using navigation systems: bones and ligaments anatomy and kinematics

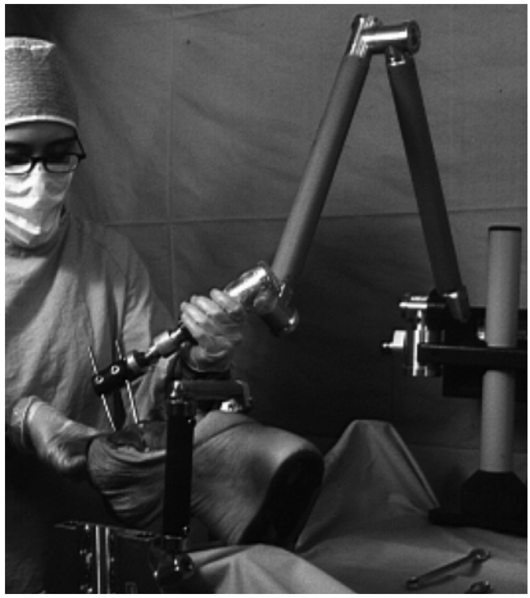

Fig. 5.2 Cadaver setup during the testing of a mechanical system used to assess joint kinematics in anterior cruciate ligament (ACL) reconstructions (Courtesy of Istituto Ortopedico Rizzoli, Bologna, Italy)

At the very beginning CAS systems, even based on mechanical systems, provided also an optimal methodology to perform biomechanical studies, exploiting its potentialities above all cadaver laboratory design. The last products of CAS technology, i.e. the navigation systems, can be instead used during the surgery following an intra-operative approach and providing information about the anatomy of the patient and the stability and function of the joint both before and after the reconstruction.

Specifically this chapter aims to highlight the advantages given by using a CAS approach in analysing from both a biomechanical and a clinical point of view:

- Anterior cruciate ligament (ACL)
- Posterior cruciate ligament (PCL)
- Medial patellofemoral ligament (MPFL)

Hereinafter details about several biomechanical and clinical studies will be therefore provided, including both the anatomical and functional features (Fig. 5.1).

5.1.2 State of the Art

5.1.2.1 ACL

Anterior cruciate ligament (ACL) anatomy and its biomechanics and kinematics have been widely investigated since the nineteenth century, due to its functional importance inside the knee joint. The information presented in literature certainly improved knowledge about it and consequently the efficacy of its surgical reconstruction. As reported by Zaffagnini et al. [19], in general, knee ligaments have been studied by several approaches, including anatomical observation, mathematical modelling, force measurement and displacement measurement. ACL insertion areas and ligament isometry, elongation and orientation during normal and pathological conditions have also been widely investigated for their importance in improving clinical results of ACL reconstruction.

In 1998 Martelli et al. [12], starting from the acquisition of the anatomy and kinematics of the knee joint by means of a mechanical system (Fig. 5.2), proposed a biomechanical ACL model including mechanical and geometrical features

(Fig. 5.3), specifically based on viscoelastic curvilinear fibres, through a quasi-static simulation.

In that study, the simulation of ACL deformations during passive motion predicted a fibre strain of less than 20 %, low insertion forces and an isometric behaviour of the anterior fibres. This proposed model was able to clearly explain the actions of ACL fibres, introducing the concepts of viscoelasticity in curvilinear fibres (Fig. 5.4).

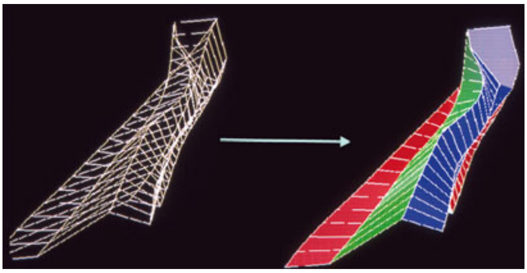

Fig. 5.3 Schematic description of the approach proposed by Martelli et al. [12]: from anatomy to biomechanical modelling

Furthermore, in 2004 Zaffagnini et al. [19] performed a quantitative analysis in a cadaver study, examining ACL elongation and its 3D orientation. The performed analysis was based on a computer elaboration of anatomical and kinematic data obtained by an electrogoniometer, thus enabling a mathematical and statistical evaluation of the 3D behaviour of the ACL (Fig. 5.5).

Their data confirmed the isometric behaviour of ACL; specifically the ACL slope with respect to the tibial plateau decreases with flexion, whereas its orientation with respect to the femoral notch increases with flexion. They also provided information about the importance of anteromedial (AM) and posterolateral (PL) bun-

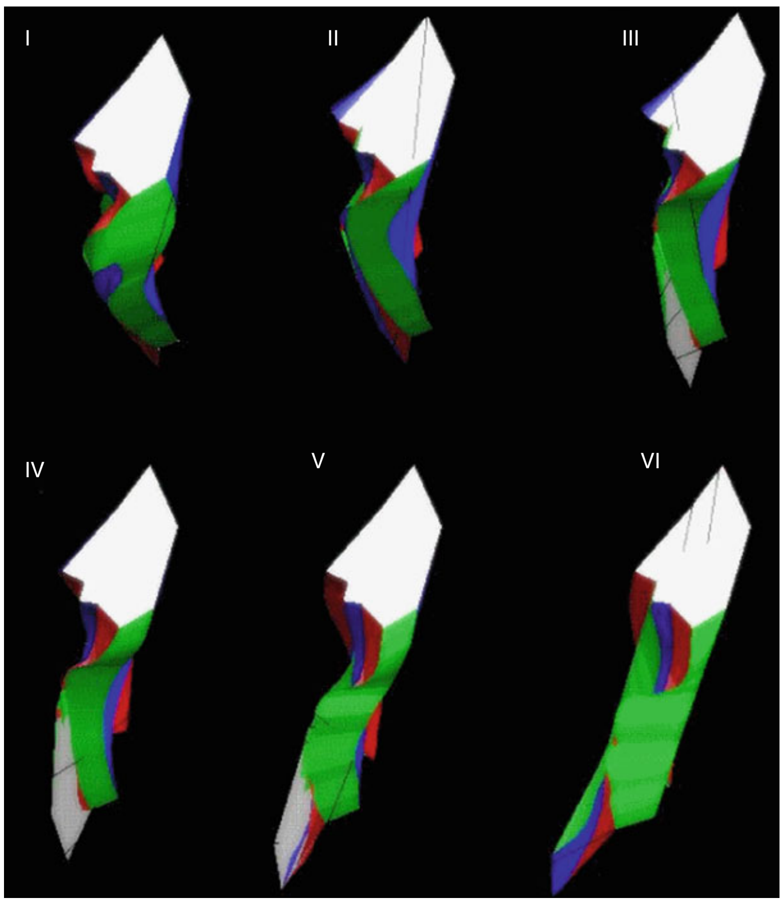

Fig. 5.4 Viscoelastic model for ACL fibres, simulated for a flexion movement of the knee joint (from position I to VI)

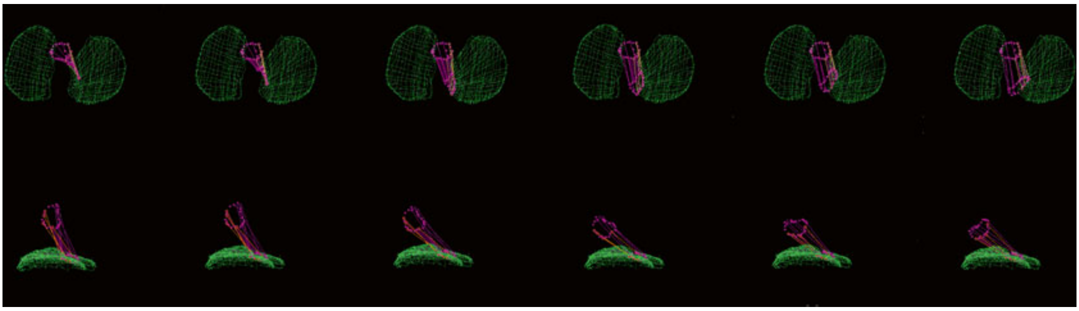

Fig. 5.5 Anterior cruciate ligament (PCL) fibres behaviour acquired by the CAS system during a passive range of motion

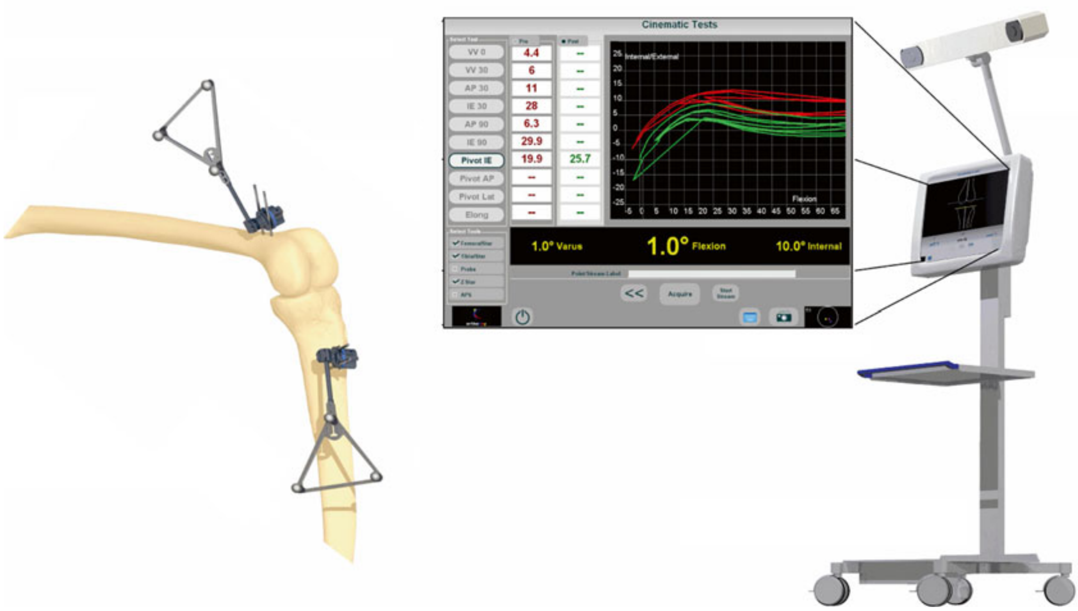

Fig. 5.6 Navigation system details: localiser, user interface and positioned trackers (Courtesy of Orthokey LLC, Lewes, DE, USA)

dle; specifically they have different angular variations, mainly with respect to the tibial plateau and mediolateral direction.

These biomechanical studies provided quantitative and qualitative information on the ACL behaviour, thus increasing not only the knowledge on the ACL anatomy but also developing or improving the surgical strategy for ACL reconstruction. More recently with the introduction of intra-operative navigation system (Fig. 5.6), besides the anatomical features, the kinematic analysis in ACL surgery acquired also a great impact in the evaluation of the knee stability related to ligament surgery. Several analyses have been performed on ACL surgery using intra-operative navigation system, highlighting the advantages given by the introduction of this technology and the obtained clinical outcomes.

As reported by [6, 13–16, 21], the 6° of freedom (DoF) of the knee joint can be computed by analysing the relative motion of the tibial frame with respect to the femoral one. Knee kinematics can be real-time evaluated for each performed test in ACL surgery. In particular

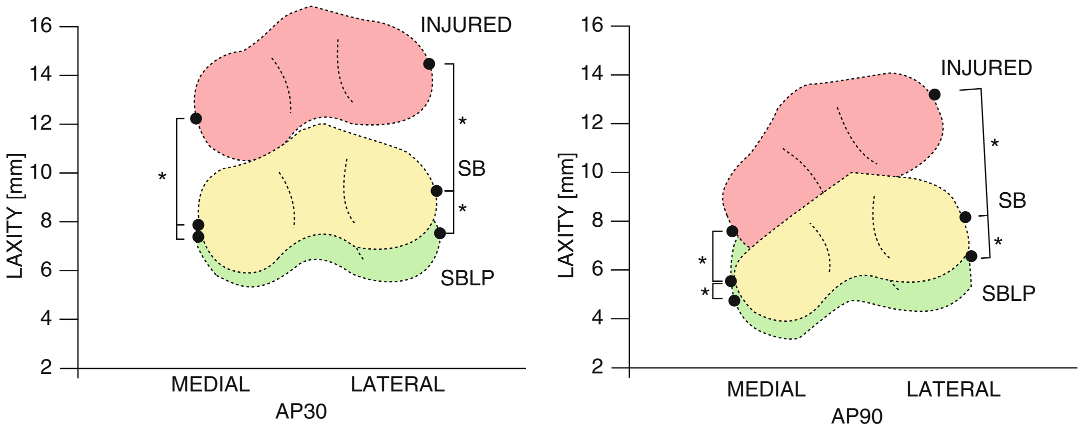

Fig. 5.7 Knee AP laxity decomposition. Medial and lateral plateau displacements are highlighted during AP30 and AP90 test and corresponding translations are reported. * $p<0.05$

the system computed varus/valgus (VV) laxity as the difference between maximum and minimum instantaneous rotations achieved during VV tests at 0° (VV0) and 30° (VV30) around the anterior-posterior axis; internal/external (IE) rotational laxity as the difference between maximum and minimum instantaneous rotations achieved during IE tests at 30° (IE30) and 90° (IE90) around the proximo/distal axis; and the anterior-posterior (AP) laxity as the difference between maximum and minimum instantaneous displacements achieved during Lachman's (AP30) and drawer (AP90) tests along the anterior-posterior axis. Since pivot-shift (PS) test is a complex manoeuvre, the decomposition of the kinematics corresponding to this test involves the analysis of both IE rotations and AP displacement, specifically of the lateral compartment.

This new approach led to the possibility of answering, on a quantitative basis, to different kinds of clinical questions, such as:

- *The influence of combined lesions in ACL injury on knee laxity*: In 2007 Zaffagnini et al. [22] intra-operatively quantified the knee laxity in AP and VV directions in isolated ACL injury compared with combined ACL and medial collateral ligament (MCL) grade II sprain. To verify the hypothesis that some residual laxities may remain in patients with combined ACL and MCL injuries even after ACL reconstruction, they compared the differences in knee stability between the two groups. The obtained results confirmed the hypothesis that in those patients with associated lesions, VV laxity at 30° and AP laxity at 90° of flexion were approximately 1° and 1.3 mm greater, respectively, with respect to the control group.
- *The influence of an extra-articular lateral plasty during single-bundle ACL reconstruction on knee laxity*: In 2009 Bignozzi et al. [1] analysed the kinematic behaviour of an arthroscopic single-bundle ACL reconstruction performed with a hamstring tendon technique with additional extra-articular procedure [11]. The laxity tests were performed before the reconstruction, after the single-bundle (SB) graft insertion and after the extra-articular procedure. As highlighted in Fig. 5.7, the analysis of medial and lateral compartment during AP stress showed that, at 30° of flexion, the SB graft causes a similar reduction of laxity in both compartments. The additional extra-articular procedure does not reduce the AP laxity in the medial compartment, while it controls the lateral compartment reducing AP displacement. On the contrary, at 90° of flexion, the SB graft reduces AP laxity more in the lateral compartment than in the medial one. The additional extra-articular procedure causes a further reduction of knee laxity in both compartments.

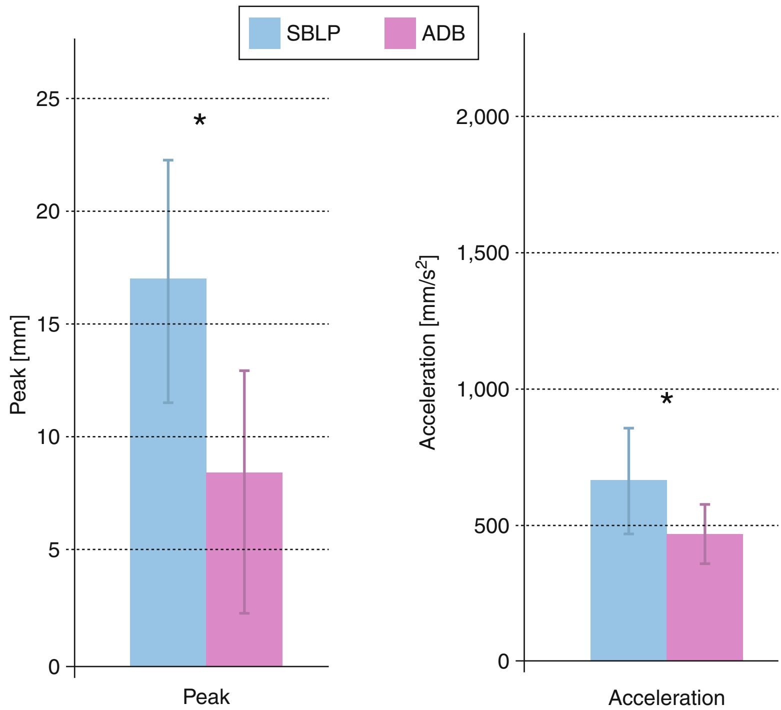

Fig. 5.8 Displacement peak and acceleration reached by the lateral tibial compartment during pivot-shift test in the single bundle with lateral plasty and anatomical double-bundle reconstructions. * $p < 0.05$

- *Knee rotational laxity in anatomical double-bundle ACL reconstruction*: In 2010, Lopomo et al. [7] evaluated the knee joint rotational laxity in anatomical double-bundle ACL reconstruction, introducing possible quantifications of the pivot-shift test, specifically the decomposition of AP translation, IE and VV rotations with respect to flexion/extension angle. For each decomposition they evaluated the areas included by the curves (the 'hysteresis' of the unstable joint) and the difference in the coupled peaks before and after the surgery at a specific flexion angle. In a comparison with respect to single-bundle ACL reconstruction with external tenodesis [24], anatomical double bundle showed its better behaviour for what concerns rotational laxities during pivot-shift test (Fig. 5.8), presenting lower peaks in lateral compartment displacement and corresponding acceleration reached after reduction.

Furthermore, due to the importance that double-bundle ACL reconstruction has been gaining in the last year [23], navigation systems are providing their support to identify also the correct position and orientation for the tunnels, starting from the anatomy and functional analysis of the joint, using also the isometry maps (Fig. 5.9).

5.1.2.2 PCL

Whereas ACL reconstructions have provided predictable and reliable results, reporting from good to excellent results in over 90 % of their patients, unfortunately, the treatment of PCL rupture still remains unclear. As reported by Zaffagnini et al. [20], this is related to the lack of knowledge about the PCL with respect to ACL, regarding anatomy, biomechanics, kinematics and biology, and the fact that the incidence of PCL lesion is lower and clinical symptoms are fewer. Moreover, the surgical technique is technically demanding. In order to improve PCL reconstruction, it is necessary to better understand its anatomy, function and behaviour. A variety of studies have looked at the fibres isometry during range of motion (ROM), as well as their elongation under different load conditions. Zaffagnini et al. [20] analysed also the exact orientation of PCL fibres by means of a computer method, thus focusing on PCL—and its anterolateral (AL) and posteromedial (PM) bundles—role in knee kinematics (Fig. 5.10).

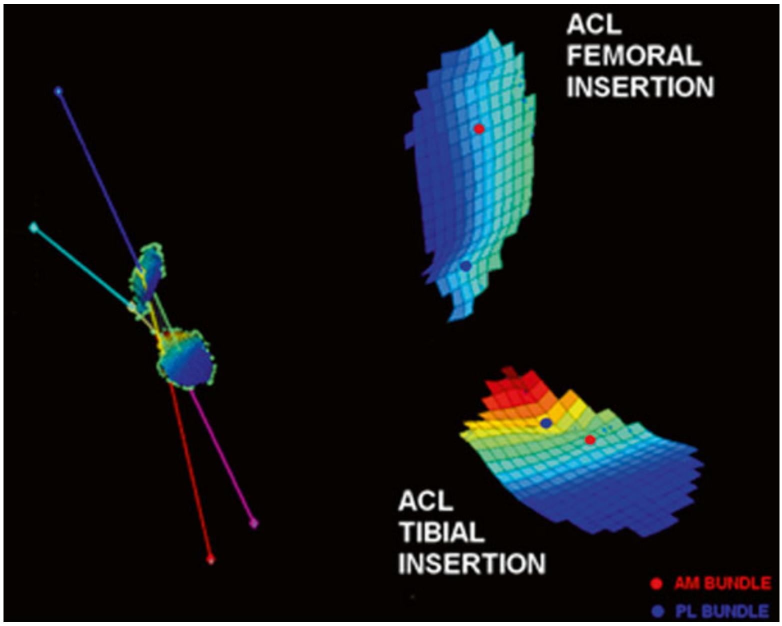

Fig. 5.9 Isometry maps for tibial and femoral ACL insertions, with the highlighted position of the tunnels in double-bundle reconstruction

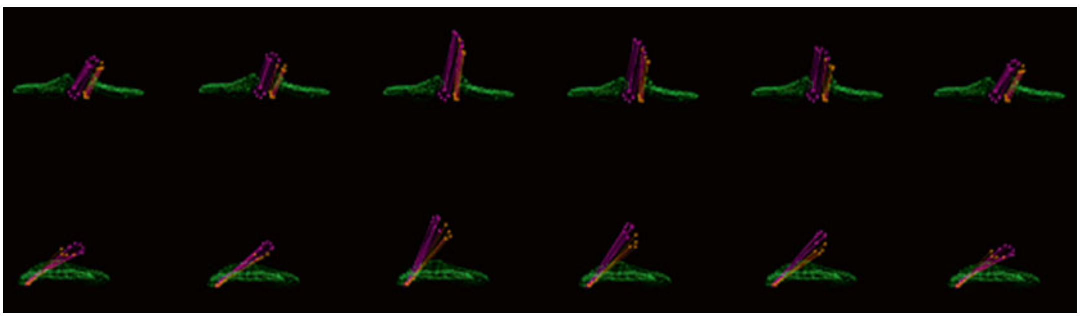

Fig. 5.10 Posterior cruciate ligament (PCL) fibres behaviour acquired by the CAS system during a passive range of motion. Anterolateral (AL) and posteromedial (PM) bundles are highlighted with different colours

Zaffagnini et al. [20] specifically reported that the anterolateral (AL) and posteromedial (PM) bundles of the PCL showed an increasing angle with respect to the tibial plateau during flexion and a statistically significant difference between them (Fig. 5.11). PCL bundles showed similar orientation with respect to the femur and varied 14° average during flexion; that study also measured 84° twisting of PCL bundles during passive range of motion.

Moreover they found that in each specimen, AL and PM fibres behave differently for what concerned fibres elongations; in fact the AL bundle elongates by 25 ± 11 % average in flexion with respect to its length in extension, while the PM bundle slackens by 4.5 ± 4 % during flexion (Fig. 5.12).

Summarising the data Zaffagnini et al. [20] acquired and analysed from a biomechanical point of view could provide new information for a better understanding of the global kinematic behaviour of the PCL and knee joint as well as to ensure a more physiologic PCL reconstruction.

5.1.2.3 MPFL

Retinacular restraints have a critical role in patellar tracking, limiting the movement of the patella in the trochlear groove. The medial patellofemoral ligament (MPFL) is one of the well-recognised primary static stabiliser of lateral patellar dislo-

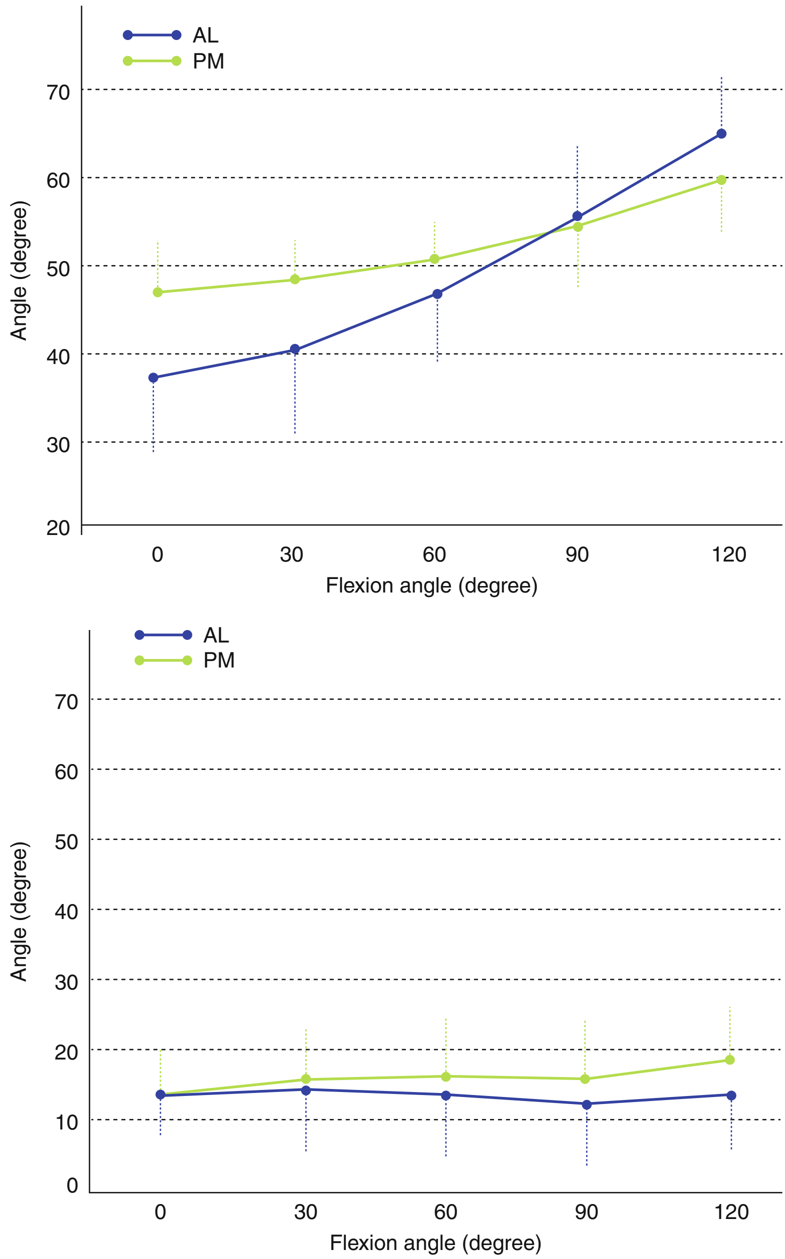

Fig. 5.11 Angle between anterolateral (AL) and posteromedial (PM) bundle in PCL and the tibial plateau (left) and the plane through the lateral wall of the medial femoral condyle. Values are the average of all fibres and specimens, and their standard deviation is computed on the mean of the eight knees [20]

cation and is always found to be deficient or ruptured in acute and chronic cases of patellar dislocation [17]. At present few studies are focused on MPFL role on patellofemoral kinematics and patellar stability.

In a recent work, Zaffagnini et al. [25] analysed in an in vitro study the influence of the MPFL on the function of the patellofemoral joint and patellar stability, from a biomechanical point of view, using a nonimage-based navigation system and integrating anatomical and kinematics data (Fig. 5.13).

They specifically reported that the MPFL-intact state showed a patellar shift in medial direction during the first degrees of knee flexion—which disappeared in MPFL-cut condition—followed by a lateral shift, similar to that of MPFL-cut condition. Tilt analysis showed that

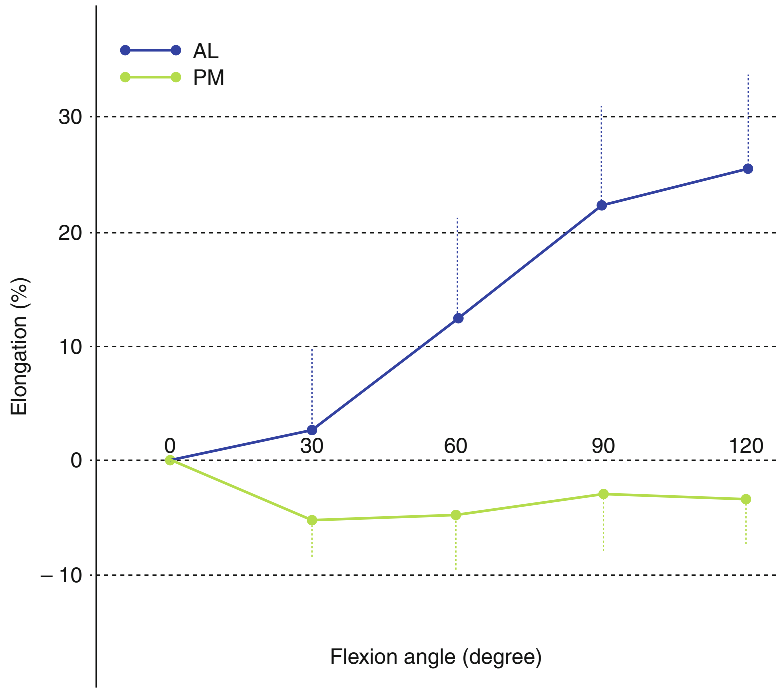

Fig. 5.12 Percentage elongation of the anterolateral (AL) and posteromedial (PM) bundle in PCL. Values are the average of all fibres and specimens, and their standard deviation is computed on the mean of the eight knees [20]

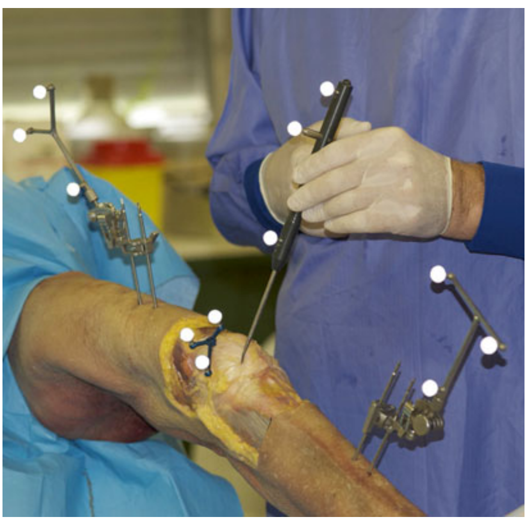

Fig. 5.13 Cadaver laboratory setup for MPFL and patellofemoral kinematic analysis [25]

patella is rotated laterally until 85° of knee flexion for intact MPFL condition and until 70° for MPFL-cut condition and after rotated medially. Static tests showed that patellar stability was significantly affected by MPFL-cut condition in particular at 30° and 60°. A good simulation of the MPFL fibres behaviour is reported in Fig. 5.14.

They concluded that MPFL has an aponeurotic nature, thus it works as a restraint during motion, with an active role under high stress on lateral side, but with a small contribution during neutral knee flexion. Its biomechanical behaviour under loading conditions should be kept into account when performing surgical reconstruction of this ligamentous structure.

In fact, since the importance of MPFL in patellar stability has been documented, several authors have studied also its anatomic reconstruction, either as an isolated procedure or combined with other patellofemoral surgery. Ntagiopoulos et al. [17] specifically evaluated the biomechanical results from the in vitro reconstruction of MPFL using a navigation-assisted technique on a cadaveric model and its effects on patellar stability and kinematics (Fig. 5.15).

The authors investigated the hypothesis that patellar kinematics after reconstruction with a tubular graft are not optimal when compared with the original fan-shaped MPFL. They found that there was a comparable medial to lateral patellar translation and tilting of the patella in the MPFL-intact and the MPFL-reconstructed state.

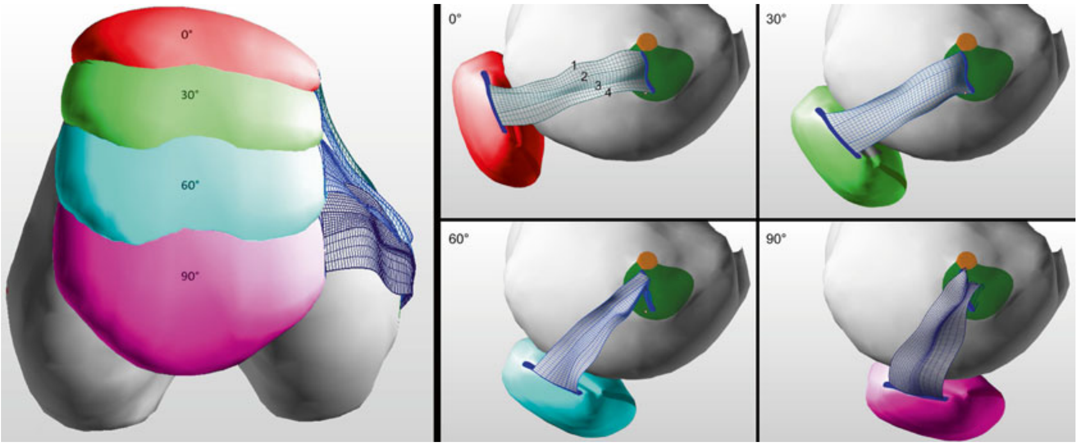

Fig. 5.14 Three-dimensional elaboration of the medial patellofemoral ligament (MPFL) and medial collateral ligament (MCL) insertions and simulated motion of its fibres (1, 2, 3, 4) during a tibiofemoral flexion

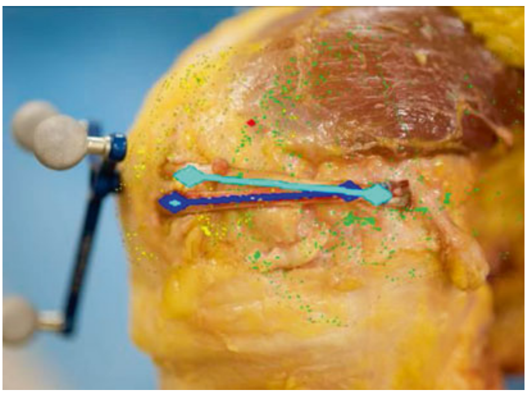

Fig. 5.15 Close-up of the performed MPFL reconstruction [17], superimposing bony and ligamentous structures acquired by the navigation system; superior (*light blue line*) and inferior (*dark blue arrow*) bundle; the used patellar tracker is evident on the *left side*

Static patellar translation in the MPFL-reconstructed state, with and without the application of load, was comparable to patellar translation in the MPFL-intact state. The dynamic patellofemoral shift kinematics recorded an under-constraint in early flexion and over-constraint in late flexion, while an opposite effect was recorded in patellar tilt. However, these differences were not statistically significant.

This study confirmed the major role of the MPFL in case of medial loading between 0° and 60°, by focusing on the kinematic importance of identifying the proper femoral point for fixation. While the study demonstrates the importance of kinematic determination of the proper femoral point of fixation, the anatomical insertion remains difficult to identify. Even in dissected cadavers, the authors recorded a slightly anterior placement than native MPFL. After reconstruction, patellar stability in terms of lateral translation and tilt was similar to the intact MPFL, but patellar kinematics were not optimal with the use of a smaller and tubular graft than the native wider and fan-shaped MPFL (Fig. 5.16).

5.1.3 Future Treatment Options

5.1.3.1 In Vivo Evaluation of Patellofemoral Kinematics and Stability

The patellofemoral joint biomechanics is still under analysis, above all for what concerns its behaviour in in vivo conditions. The navigation systems could contribute to better understand the biomechanics behind this joint using specific trackers (Fig. 5.17). This knowledge should then be reported to active movements as well.

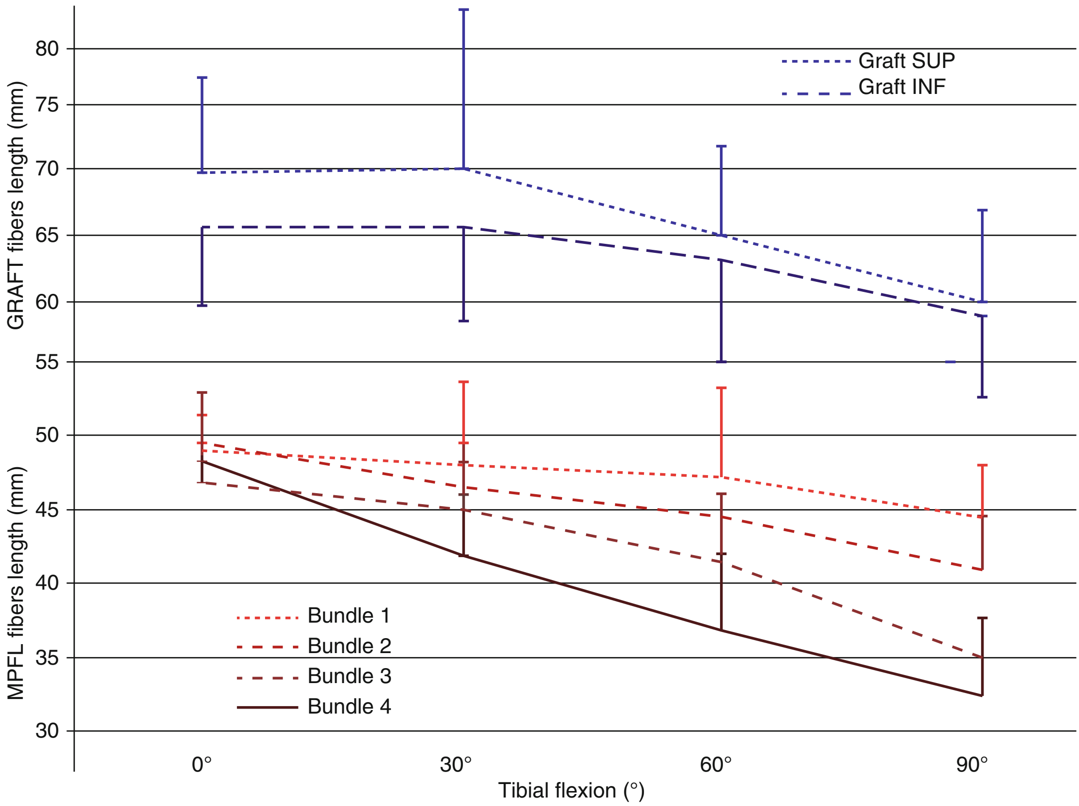

Fig. 5.16 Global length of MPFL bundles and corresponding two-graft MPFL reconstruction

Fig. 5.17 Prototypal patellar tracker (*circle*) in an intra-operative setup

5.1.3.2 Forces Measurements During Intra-operative Clinical Examinations

At present most navigation systems have introduced the ability to record knee joint kinematics, but to move forward, this remains the next step in intra-operative analysis and an open challenge for system developers, even if some researchers are working on it [2].

5.1.3.3 Non-invasive Assessment of Knee Stability

Pivot-shift test seems to be the most relevant test in the analysis of knee rotational laxity. In the last years several efforts have been done in order to use non-invasive systems based on inertial [8–10] or image analysis [4] in quantifying pivot-shift test, thus allowing a quantitative assessment also in ambulatory setup, where CAS technology still has a lot to give.

5.1.4 Take-Home Message

The navigation systems, based on an opto-electronic localiser and combining specific functional-dedicated acquisition software, can allow to perform a set of very different analysis of knee joint kinematics, thus focusing on ligaments behaviour. These systems have been characterised and their clinical reliability verified. The CAS approach, with the combination of measuring knee kinematic properties, anatomical features, and allowing intra-operative standard clinical tests, helped both to improve our knowledge on the knee joint biomechanics and to distinguish between healthy and pathological knees in in vivo conditions, supporting the surgery. The development of the kinematic approach has showed its huge potentiality and has been contributing in significant manner to several research works in different clinical field; specifically the functional methodology was successfully used in the analysis of the comparison between the outcomes of different ligamentous reconstructions.

5.2 3D Stereoscopy and Arthroscopic Anatomy of the Knee: Portal Toward the Future

5.2.1 Introduction

'Why looking through a hole when you can open the door'. This sentence has been widely used in the past by sceptics referring themselves to first steps of a new surgical technique to indirectly observe, magnify and operate joints: arthroscopy. Since the pioneers as Takagi, Bircher, Nordentoft and Watanabe, arthroscopy has grown and conquered a main role into orthopaedic surgery, improving our knowledge regarding anatomy, functional anatomy and surgical techniques. Less morbidity and invasivity are concepts that have married arthroscopic surgery since the beginning, reaching in the third millennium, outcomes that were unthinkable 20 years ago.

Optical and digital technology played a main role to support and improve the visualisation and magnification of the anatomical structures observed and eventually treated by arthroscopy. We started watching directly on the back of the optic, through the analogic cameras and video to the modern HD systems. With a power of magnification up to 30 times, we are able to better discriminate anatomy and move with higher precision inside the joints. Limits of arthroscopy are the closed field of action (inner joint), poor visualisation of ligaments and tendons lying on the outer aspect of the joint capsule and visualisation of flat images, decreasing the perception of the deepness of the different layers.

Technology is moving fast: we are already able to record and view anatomy and surgical procedures with 3D stereoscopic technology. This is based on the human system of vision: two eyes, two optic devices. If we use two cameras or a camera with two lenses, we can reproduce the stereoscopic view recording a left and right image and putting it on a dedicated monitor with a particular refresh rate. These devices represent a terrific step forward into the field of teaching/learning anatomy, surgical approaches and procedures. The commercialisation of the 3D monitor for desktop and laptop computer will give us the opportunity to build e-learning platforms based on this technology.

The question is if this technology can be applied to arthroscopy. We know that advanced projects for laparoscopy with stereoscopic optic devices are available on the market. Patents for stereoscopic arthroscopy have been registered but without any commercial or scientific use. Development of stereoscopic arthroscopy might remove the limit of two-dimensional visualisation allowing a proper discrimination of the field deep. Surgical procedure might be executed more precisely and even intra-articular measuring and probing.

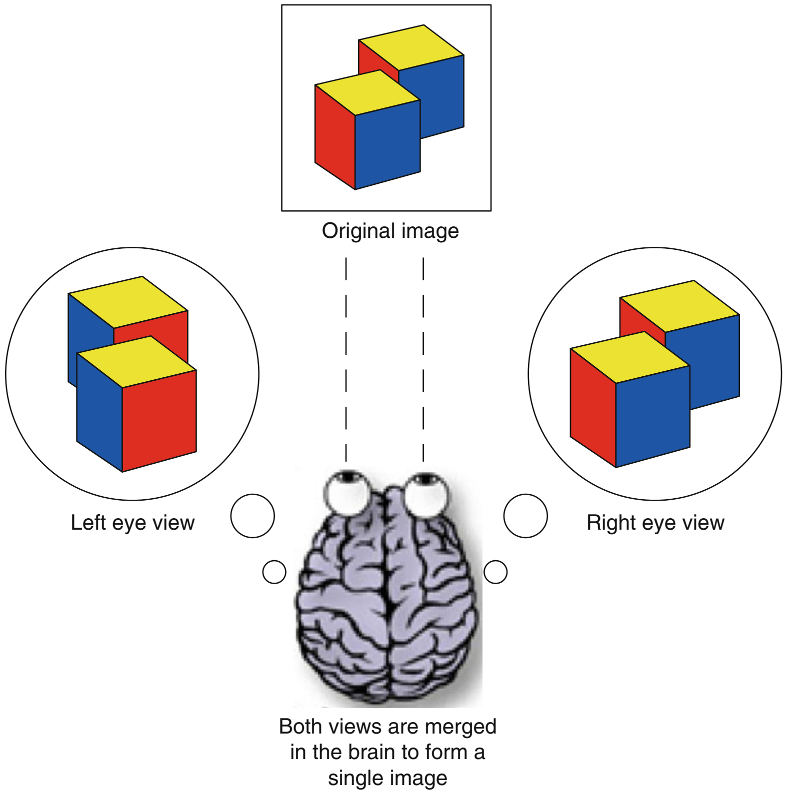

Fig. 5.18 How the brain elaborates images from left and right eye

We can already get 3D stereoscopic still images by arthroscopy. Recording a left and right still picture of a same frame is not difficult; dedicated software is capable to elaborate a stereoscopic image. This process is useful for arthroscopic anatomy comprehension and joined to 3D stereoscopic open anatomy pictures, representing a powerful tool for education and learning.

The aim of this section of ICL is to show the potentiality of this technology confronting 3D stereoscopic and flat images by arthroscopy and open anatomy from standard and accessory portals of the knee.

5.2.2 3D Stereoscopic Imaging

Human beings, like most other creatures, are equipped with two eyes, situated close together and side by side. This positioning means that each eye has a view of the same area from a slightly different angle. You can check this out by focusing on a distant object and viewing through each eye alternately—see how some things seem to change position slightly. The brain takes the information from each eye and unites them into one picture, interpreting the slight differences between each view as *depth*. This produces a three-dimensional picture: one with height, width and depth (Fig. 5.18).

With stereoscopic vision, we see exactly where our surroundings are in relation to our own bodies, usually with considerable precision. We are particularly good at spotting objects that are moving toward or away from us, and the positioning of our eyes means we can see partially around solid objects without needing to move our heads. Anatomically, there are three levels of binocular vision required to view stereo images:

1. Simultaneous perception
2. Fusion (binocular 'single' vision)
3. Stereopsis

These functions develop in early childhood. Some people who have strabismus disrupt the

Fig. 5.19 Two video cameras for recording two streams (*left* and *right*)

Fig. 5.20 (**a**) Dedicated camera with two optics for recording one multi-format streaming. (**b**) Consumer video and photo camera with two optics

development of stereopsis; however, orthoptics treatment can be used to improve binocular vision. A person's stereoacuity determines the minimum image disparity they can perceive as depth. It is believed that approximately 12 % of people are unable to properly see 3D images, due to a variety of medical conditions. According to another experiment up to 30 % of people have very weak stereoscopic vision preventing them from depth perception based on stereo disparity. This nullifies or greatly decreases immersion effects of stereo to them.

The process of recording and reproducing the world with stereoscopic 3D vision starts obtaining a left and a right frame like the eyes do. We can use two separate cameras (Fig. 5.19) or dedicated cameras with built-in double optic system, usually at the same focal distance of the human eyes (6.5 cm) (Fig. 5.20a, b).

Technology is moving faster and faster to develop hardware that could trick human brain reproducing perception of 3D stereoscopic vision on flat surfaces (monitor, television, cinema screen). The method is based on giving to eyes/brain right and left images with a particular frame rate or activating right and left eye alternatively.

There are two categories of 3D viewer technology, active and passive. Active viewers have electronics which interact with a display. Passive viewers filter constant streams of binocular input to the appropriate eye.

5.2.2.1 Active Shutter Systems

A shutter system works by openly presenting the image intended for the left eye while blocking the right eye's view, then presenting the right-eye image while blocking the left eye, and repeating this so rapidly that the interruptions do not interfere with the perceived fusion of the two images into a single 3D image. It generally uses liquid crystal shutter glasses. Each eye's glass contains a liquid crystal layer which has the property of becoming dark when voltage is applied, being otherwise transparent. The glasses are controlled by a timing signal that allows the glasses to alternately darken over one eye, and then the other, in synchronisation with the refresh rate of the screen (Fig. 5.21).

5.2.2.2 Passive Polarisation Systems

To present stereoscopic pictures, two images are projected superimposed onto the same screen through polarising filters or presented on a display

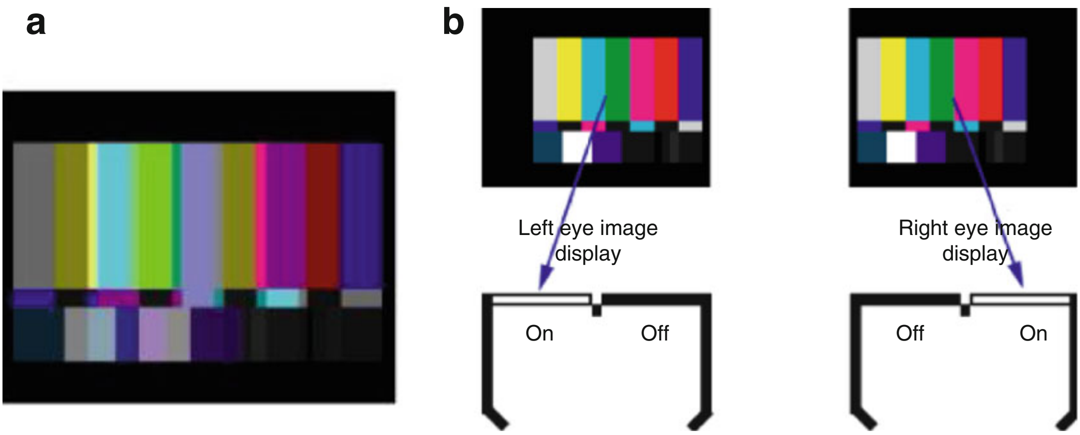

Fig. 5.21 (**a**, **b**) Mixed left and right frames (**a**) are sent alternatively to the right and left eye by glasses synchronised with the monitor (active glasses)

with polarised filters. For projection, a silver screen is used so that polarisation is preserved. The viewer wears low-cost eyeglasses which also contain a pair of opposite polarising filters. As each filter only passes light which is similarly polarised and blocks the opposite polarised light, each eye only sees one of the images, and the effect is achieved.

Interference Filter Systems

This technique uses specific wavelengths of red, green and blue for the right eye and different wavelengths of red, green and blue for the left eye. Eyeglasses which filter out the very specific wavelengths allow the wearer to see a full-colour 3D image. It is also known as *spectral comb filtering or wavelength multiplex visualisation or super-anaglyph*. Dolby 3D uses this principle.

Colour Anaglyph Systems

Anaglyph 3D is the name given to the stereoscopic 3D effect achieved by means of encoding each eye's image using filters of different (usually chromatically opposite) colours, typically red and cyan. Anaglyph 3D images contain two differently filtered coloured images, one for each eye. When viewed through the 'colour-coded anaglyph glasses', each of the two images reaches one eye, revealing an integrated stereoscopic image. The visual cortex of the brain fuses this into perception of a three-dimensional scene or composition (Fig. 5.22).

Fig. 5.22 Anaglyph 3D glasses

Fig. 5.23 ChromaDepth 3D glasses

Chromadepth System

ChromaDepth Glasses with Prism-Like Film

The ChromaDepth procedure of American Paper Optics is based on the fact that with a prism, colours are separated by varying degrees. The ChromaDepth eyeglasses (Fig. 5.23) contain special view foils, which consist of microscopically small prisms. This causes the image to be translated a certain amount that depends on its colour.

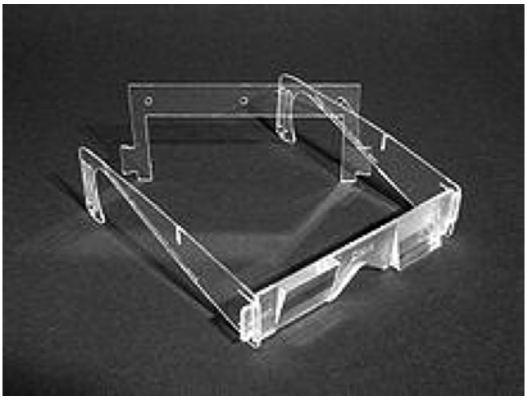

Fig. 5.24 KMQ stereo prismatic viewer with openKMQ plastics extensions

If one uses a prism foil now with one eye but not on the other eye, then the two seen pictures—depending upon colour—are more or less widely separated. The brain produces the spatial impression from this difference. The advantage of this technology consists above all of the fact that one can regard ChromaDepth pictures also without eyeglasses (thus two dimensional) problem-free (unlike with two-colour anaglyph). However, the colours are only limitedly selectable, since they contain the depth information of the picture. If one changes the colour of an object, then its observed distance will also be changed.

Over/Under Format

Stereoscopic viewing is achieved by placing an image pair one above one another. Special viewers are made for over/under format that tilts the right eyesight slightly up and the left eyesight slightly down. The most common one with mirrors is the View Magic. Another with prismatic glasses (Fig. 5.24) is the KMQ viewer. A recent usage of this technique is the openKMQ project.

A step beyond is represented by systems that do not need any glasses or 'interface' to trick the brain vision. These systems are going to substitute the glasses-mediated devices into the near future.

Autostereoscopy

Autostereoscopic display technologies use optical components in the display, rather than worn by the user, to enable each eye to see a different image. Because headgear is not required, it is also called 'glasses-free 3D'. The optics split the images directionally into the viewer's eyes, so the display viewing geometry requires limited head positions that will achieve the stereoscopic effect. Automultiscopic displays provide multiple views of the same scene, rather than just two. Each view is visible from a different range of positions in front of the display. This allows the viewer to move left to right in front of the display and see the correct view from any position. The technology includes two broad classes of displays: those that use head tracking to ensure that each of the viewer's two eyes sees a different image on the screen and those that display multiple views so that the display does not need to know where the viewer's eyes are directed. Examples of autostereoscopic displays technology include lenticular lens, parallax barrier, volumetric display, holography and light field displays. The Nintendo 3DS uses parallax barrier autostereoscopy to display a 3D image.

Holography

Laser holography, in its original 'pure' form of the photographic transmission hologram, is the only technology yet created which can reproduce an object or scene with such complete realism that the reproduction is visually indistinguishable from the original, given the original lighting conditions [citation needed]. It creates a light field identical to that which emanated from the original scene, with parallax about all axes and a very wide viewing angle. The eye differentially focuses objects at different distances, and subject detail is preserved down to the microscopic level. The effect is exactly like looking through a window. Unfortunately, this 'pure' form requires the subject to be laser-lit and completely motionless—to within a minor fraction of the wavelength of light—during the photographic exposure, and laser light must be used to properly view the results. Most people have never seen a laser-lit transmission hologram. The types of holograms commonly encountered have seriously compromised image quality so that ordinary white light can be used for viewing, and non-holographic intermediate imaging processes are almost always resorted to, as an alternative to using powerful and hazardous pulsed lasers, when living subjects are photographed.

Although the original photographic processes have proven impractical for general use, the combination of computer-generated holograms (CGH) and optoelectronic holographic displays, both under development for many years, has the potential to transform the half-century-old pipe dream of holographic 3D television into a reality; so far, however, the large amount of calculation required to generate just one detailed hologram and the huge bandwidth required to transmit a stream of them have confined this technology to the research laboratory.

5.2.2.3 Laser Plasma Volumetric Display

Volumetric Displays

Volumetric displays use some physical mechanism to display points of light within a volume. Such displays use voxels instead of pixels. Volumetric displays include multiplanar displays, which have multiple display planes stacked up, and rotating panel displays, where a rotating panel sweeps out a volume.

Other technologies have been developed to project light dots in the air above a device. An infrared laser is focused on the destination in space, generating a small bubble of plasma which emits visible light.

Integral Imaging

Integral imaging is an autostereoscopic or multiscopic 3D display, meaning that it displays a 3D image without the use of special glasses on the part of the viewer. It achieves this by placing an array of microlenses (similar to a lenticular lens) in front of the image, where each lens looks different depending on viewing angle. Thus rather than displaying a 2D image that looks the same from every direction, it reproduces a 4D light field, creating stereo images that exhibit parallax when the viewer moves.

Wiggle Stereography

Wiggle stereoscopy is an image display technique achieved by quickly alternating display of left and right sides of a stereogram and found in animated GIF format on the web. Online examples are visible in the New York Public Library stereogram collection. The technique is also known as 'Piku-Piku'.

5.2.3 3D Stereoscopic Imaging of the Knee

Comprehension of knee anatomy starts with reading books, papers and anatomic tables and proceeds with direct observation on cadavers and in vivo on patients involved in surgical procedures. Working on cadavers represents the best learning experience, but it is not costless and in some country not easily accessible. Specific cadaver labs allow us to learn surgical technique and improve our anatomic knowledge as well as our handy skill with instruments and procedures. But if we limit our experience to the simple observation of the knee anatomy, 3D stereoscopic imaging is a promising tool for teaching/learning anatomy and surgical procedures. This technology is founded on digital electronics and, for this peculiarity, is available everywhere carried by web, flash memories, optical discs or any digital recording device. Live and relive surgery could be showed by 3D stereoscopic beamers giving to the audience a deeper immersion and feedback.

In this paragraph we are going to meet 3D stereoscopic images of the knee anatomy recorded at University of Barcelona and arthroscopic images of the knee elaborated by software and transformed into stereoscopic images (anaglyph green-cyano).

5.2.3.1 MPFL

Medial patellofemoral ligament has attracted a lot of research for developing less invasive but more efficient techniques of reconstruction. Anatomical studies are fundamental in defining the exact origin and insertion of this ligament (Figs. 5.25a, b, 5.26a, b and 5.27a, b).

5.2.3.2 ACL (Figs. 5.28, 5.29 and 5.30)

5.2.3.3 3D Stereoscopic Images by Arthroscopy

Knee joint has the biggest volume allowing us to create and access numerous portals for arthroscopy. Scoping the knee with different optic degrees (30°, 45° and 70°) and taking advantage of the cone view (optic rotation) allow us to reach the whole inner surface of the joint. Thus,

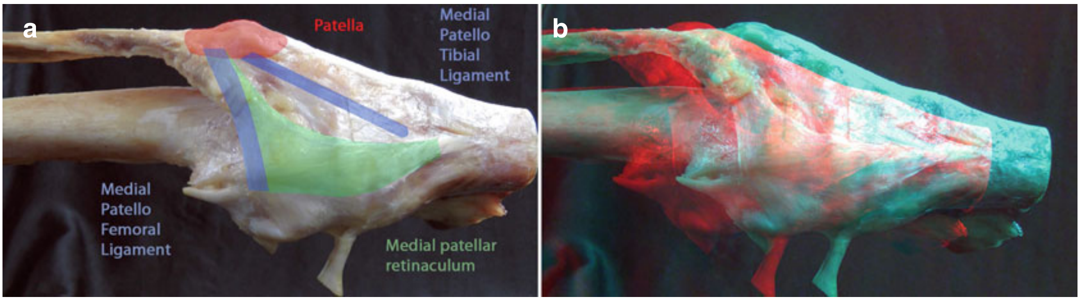

Fig. 5.25 Medial aspect of the knee, full extension (**a**) and same stereoscopic frame (**b**) (anaglyph g-c)

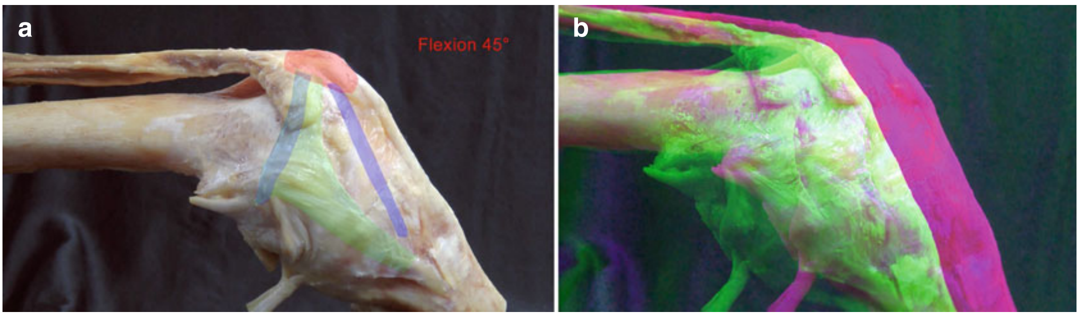

Fig. 5.26 Medial aspect of the knee, flexion 45° (**a**) and same stereoscopic frame (**b**) (anaglyph g-c)

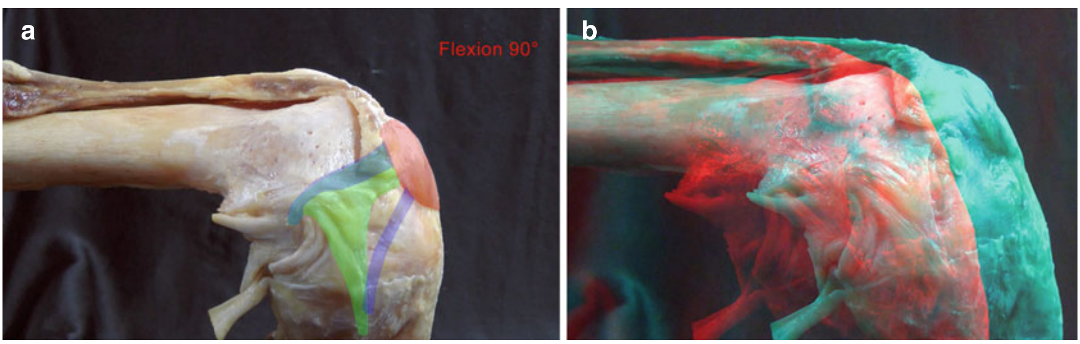

Fig. 5.27 Medial aspect of the knee, flexion 90° (**a**) and same stereoscopic frame (**b**) (anaglyph g-c)

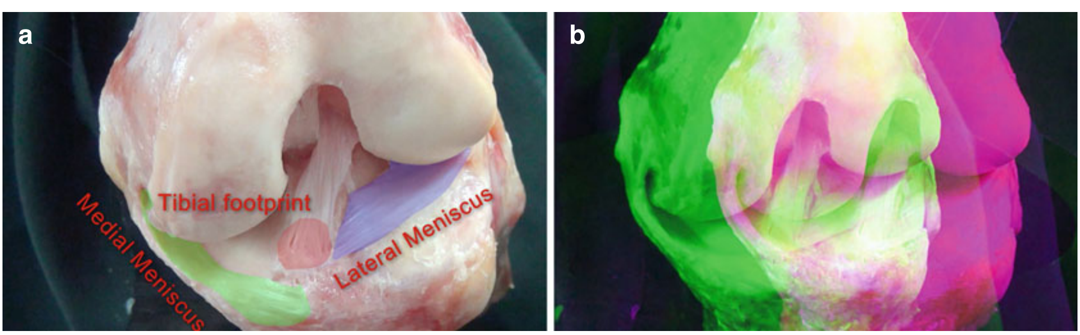

Fig. 5.28 Front-up view of the anterior aspect of the knee, flexion 90° (**a**) and same stereoscopic frame (**b**) (anaglyph g-c)

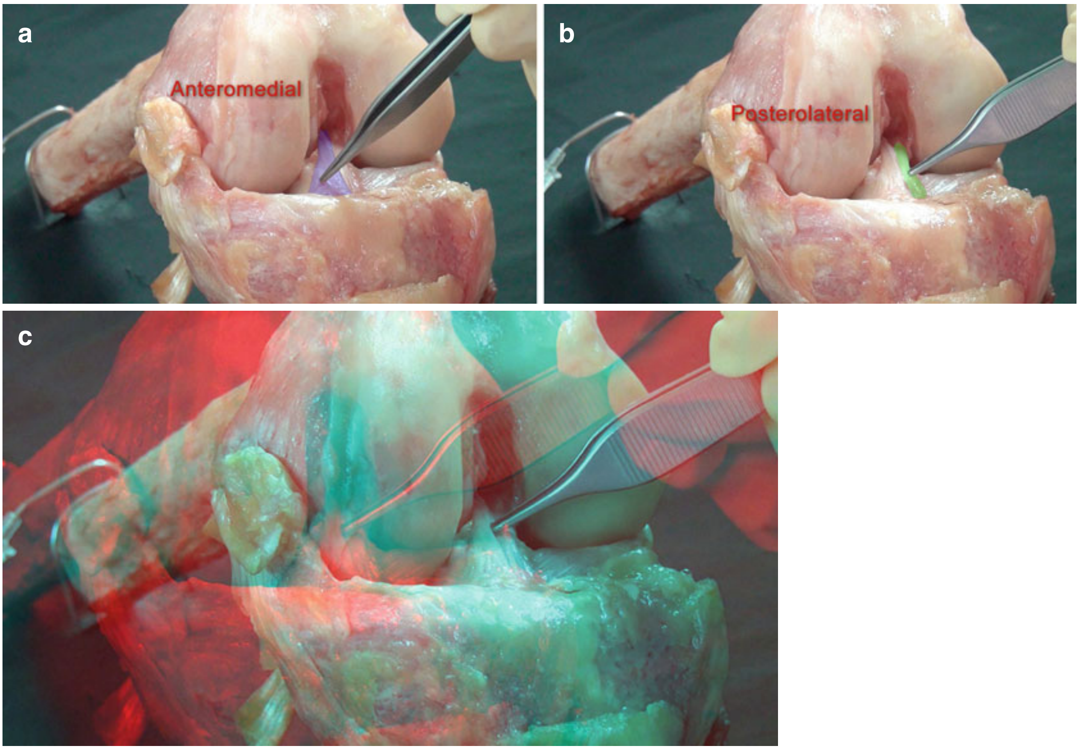

Fig. 5.29 Front view of the anterior aspect of the knee, flexion 90° (**a**) AL and PL (**b**) band of ACL (**c**)

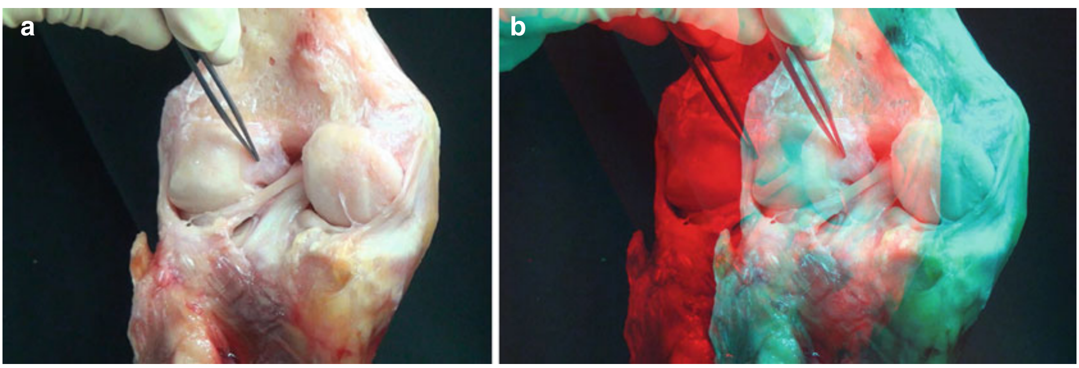

Fig. 5.30 Posterior aspect of the knee, full extension, showing the femoral origin of ACL (**a**) and same stereoscopic frame (**b**) (anaglyph g-c)

we can see and touch directly and indirectly the ligamentous structures of the deepest layer of the knee.

The following images show the potential power as didactic and practical instrument of 3D stereoscopic arthroscopy (Figs. 5.31, 5.32, 5.33, 5.34 and 5.35).

5.2.4 Portal Toward the Future

Even though projects and patents for a 3D optics for arthroscopy already exist (Fig. 5.36), the use of this new technology seems to be reserved to laparoscopy and surgical endoscopy in general, but not arthroscopy.

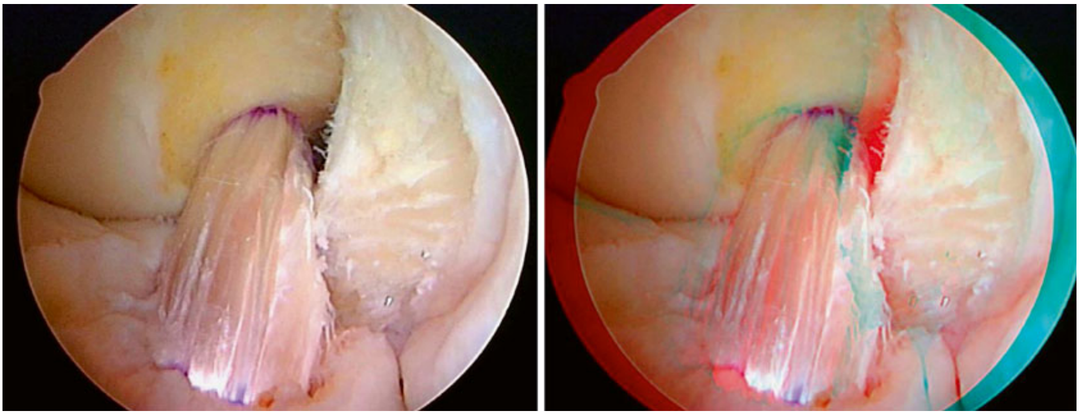

Fig. 5.31 ACL graft from AM portal and same stereoscopic frame (anaglyph g-c)

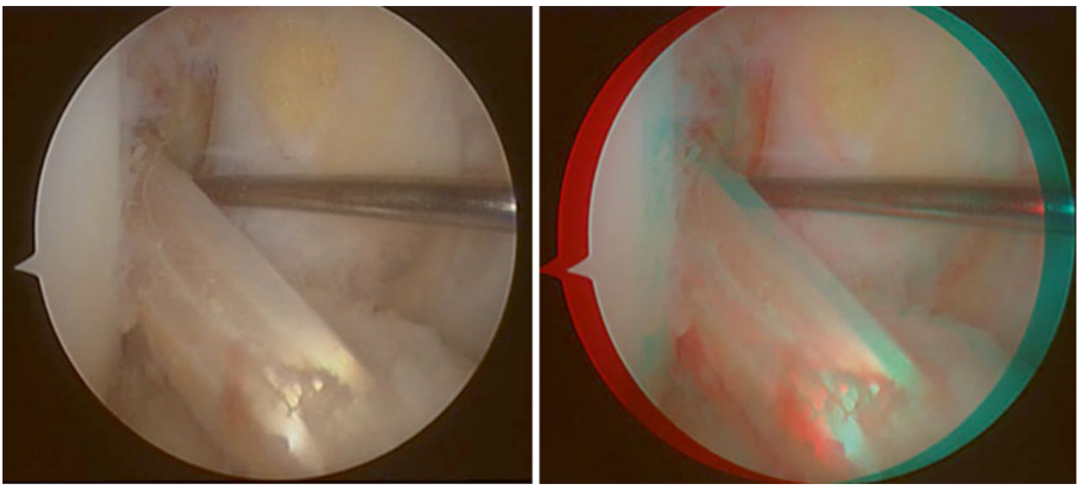

Fig. 5.32 ACL graft from AL portal and same stereoscopic frame (anaglyph g-c)

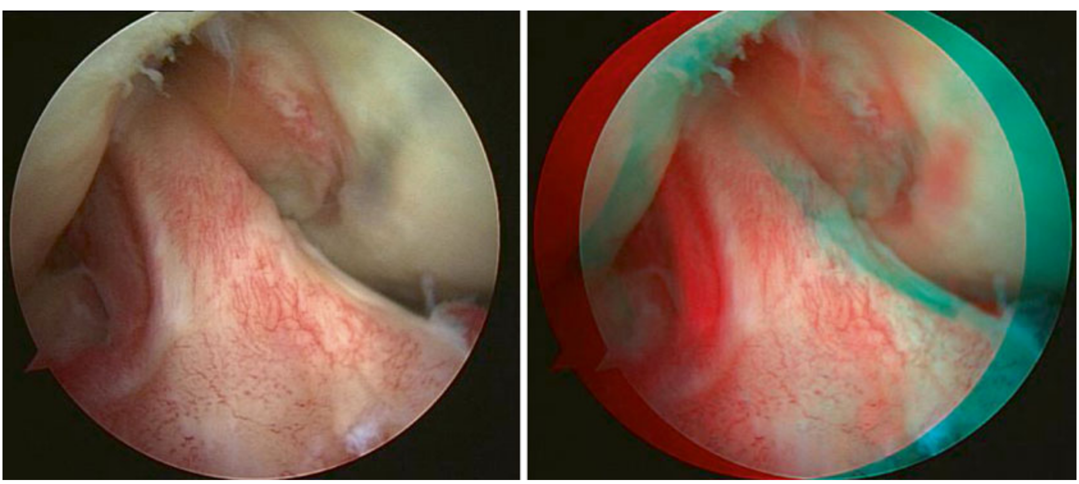

Fig. 5.33 ACL from AL portal and same stereoscopic frame (anaglyph g-c)

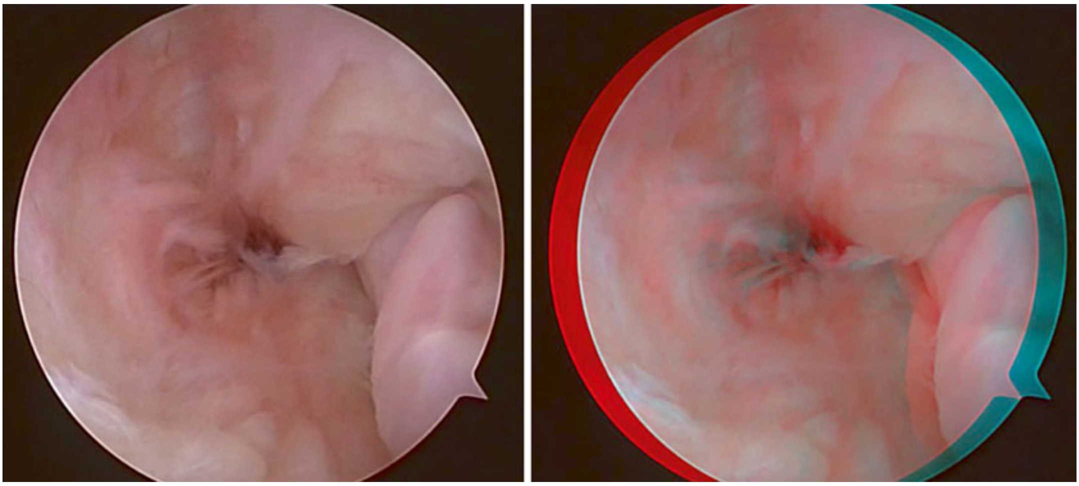

Fig. 5.34 Popliteus tendon from high-AL portal and same stereoscopic frame (anaglyph g-c)

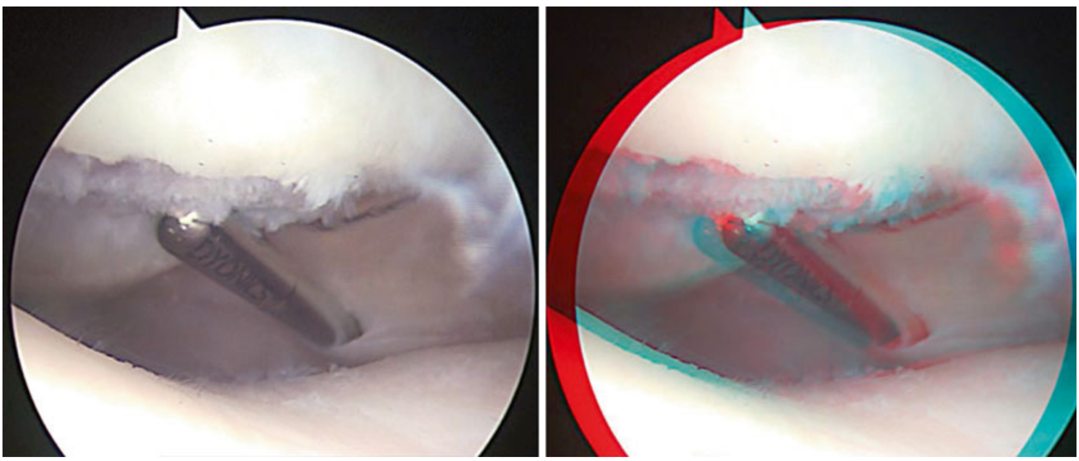

Fig. 5.35 Patella from AL portal and same stereoscopic frame (anaglyph g-c)

We started with a European project (Fig. 5.37) to build a complete arthroscopic set based on stereoscopic technology. We hope to contribute to a better knowledge, comprehension and utilisation of arthroscopic surgery.

3D stereoscopic imaging is not only entertainment: reproducing reality in remote sites (cinema, TV, monitor, etc.) can really improve our knowledge, surgical skill and procedures' precision.

Arthroscopy has been a terrific advancement for orthopaedic surgery in the last decades. It will keep this role driving and driven by new technologies.

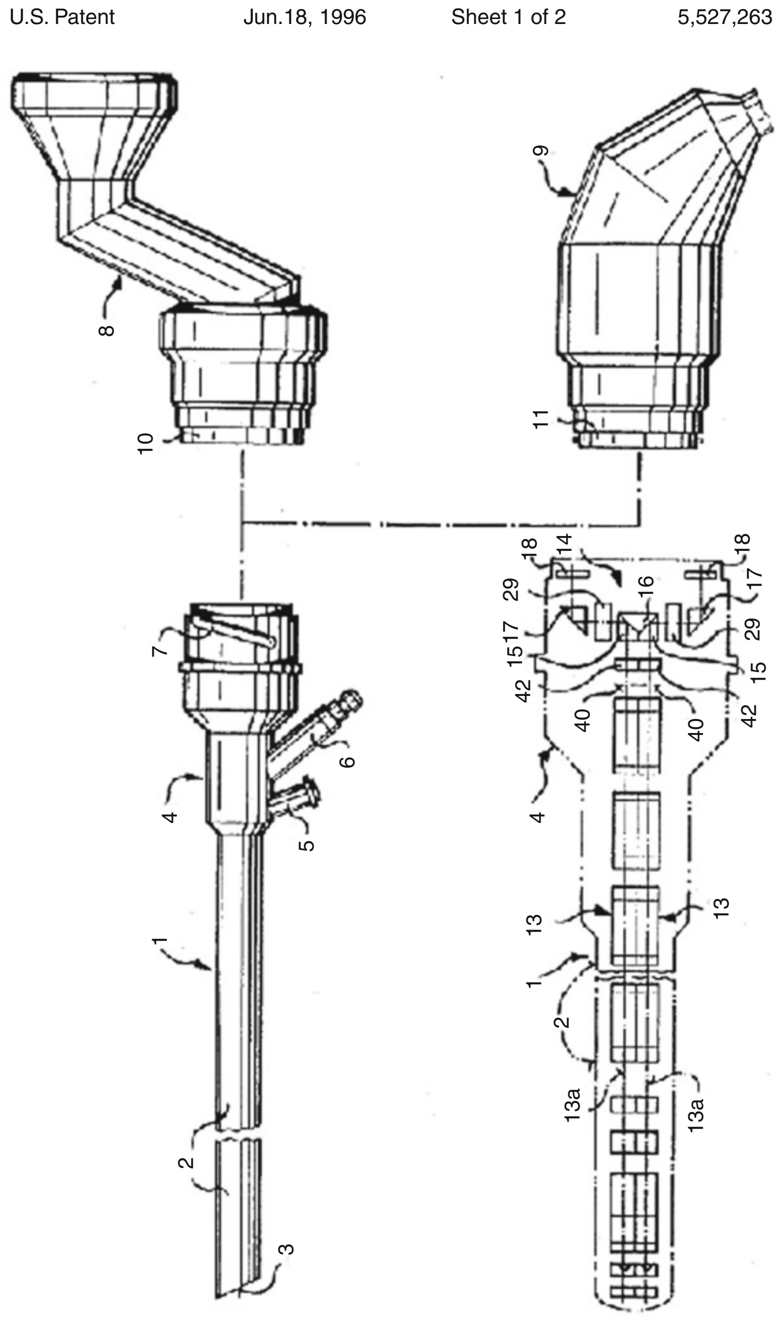

Fig. 5.36 Stereoscopic optics (US patent 5.527.263)

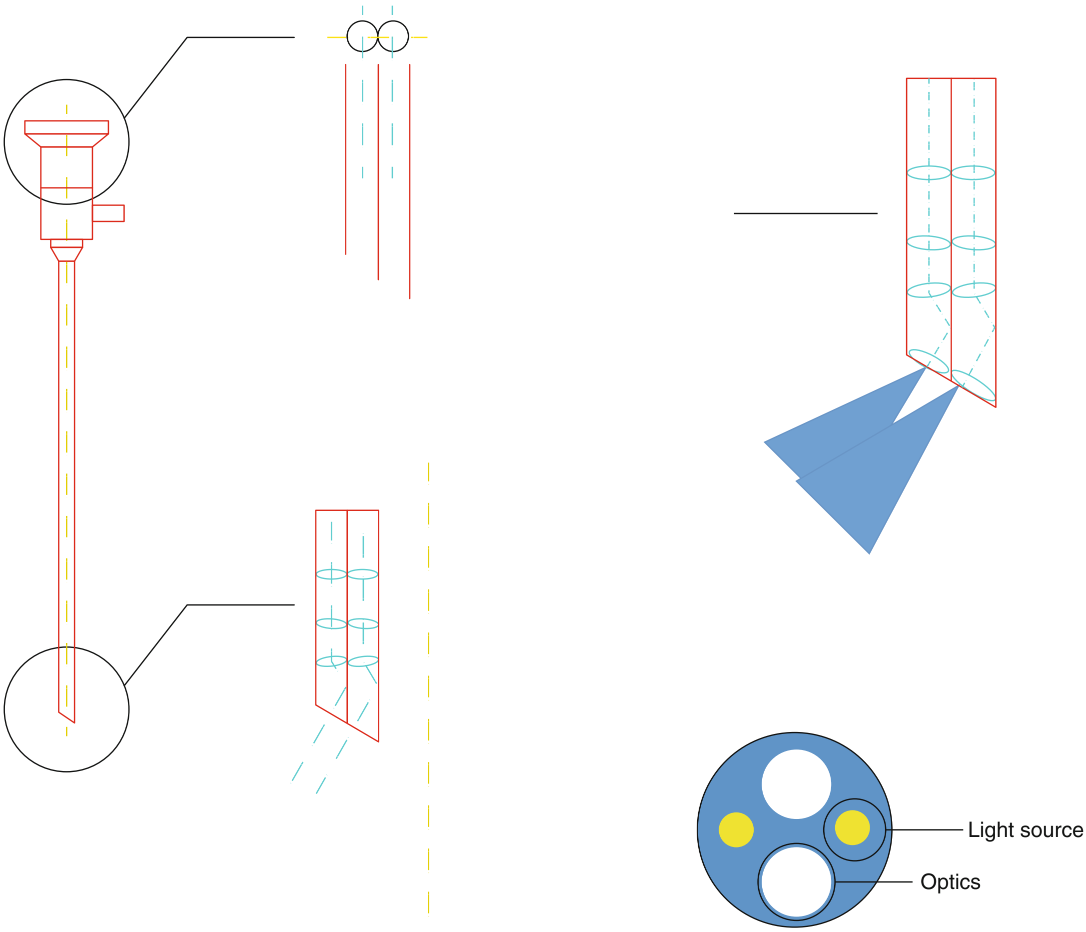

Fig. 5.37 Stereoscopic optics 3D for arthroscopy (Courtesy of G. Camillieri)

References

1. Bignozzi S, Zaffagnini S, Lopomo N, Martelli S, Iacono F, Marcacci M. Does a lateral plasty control coupled translation during antero-posterior stress in single-bundle ACL reconstruction? An in vivo study. Knee Surg Sports Traumatol Arthrosc. 2009;17(1):65–70.
2. Cammarata S, Cammarata M, Lopomo N, Bignozzi S, Cerveri P, Ferrigno G, Zaffagnini S, Visani A. Accuracy characterization of an integrated optical-based method for loads measurements in computer aided surgery. J Mech Med Biol. 2010;10:577–91.
3. Chauhan SK, Scott RG, Breidahl W, Beaver RJ. Computer-assisted knee arthroplasty versus a conventional jig-based technique. A randomised, prospective trial. J Bone Joint Surg Br. 2004;86(3):372–7.
4. Hoshino Y, Araujo P, Irrgang JJ, Fu FH, Musahl V. An image analysis method to quantify the lateral pivot shift test. Knee Surg Sports Traumatol Arthrosc. 2012;20(4):703–7.
5. Klein GR, Parvizi J, Rapuri VR, Austin MS, Hozack WJ. The effect of tibial polyethylene insert design on range of motion: evaluation of in vivo knee kinematics by a computerized navigation system during total knee arthroplasty. J Arthroplasty. 2004;19(8):986–91.
6. Lopomo N, Bignozzi S, Martelli S, Zaffagnini S, Iacono F, Visani A, Marcacci M. Reliability of a navigation system for intra-operative evaluation of antero-posterior knee joint laxity. Comput Biol Med. 2009;39(3):280–5.
7. Lopomo N, Zaffagnini S, Bignozzi S, Visani A, Marcacci M. Pivot-shift test: analysis and quantification of knee laxity parameters using a navigation system. J Orthop Res. 2010;28(2):164–9.
8. Lopomo N, Zaffagnini S, Signorelli C, Bignozzi S, Giordano G, Marcheggiani Muccioli GM, Visani A. An original clinical methodology for non-invasive assessment of pivot-shift test. Comput Methods Biomech Biomed Engin. 2012;15(12):1323–8.

9. Lopomo N, Signorelli C, Bonanzinga T, Marcheggiani Muccioli GM, Visani A, Zaffagnini S. Quantitative assessment of pivot-shift using inertial sensors. Knee Surg Sports Traumatol Arthrosc. 2012;20(4):713–7.
10. Maeyama A, Hoshino Y, Debandi A, Kato Y, Saeki K, Asai S, Goto B, Smolinski P, Fu FH. Evaluation of rotational instability in the anterior cruciate ligament deficient knee using triaxial accelerometer: a biomechanical model in porcine knees. Knee Surg Sports Traumatol Arthrosc. 2011;19(8):1233–8.
11. Marcacci M, Zaffagnini S, Iacono F, Neri MP, Loreti I, Petitto A. Arthroscopic intra- and extra-articular anterior cruciate ligament reconstruction with gracilis and semitendinosus tendons. Knee Surg Sports Traumatol Arthrosc. 1998;6(2):68–75.
12. Martelli S, Joukhadar A, Zaffagnini S, Marcacci M, Lavallee S, Champleboux G. Fiber-based anterior cruciate ligament model for biomechanical simulations. J Orthop Res. 1998;16(3):379–85.
13. Martelli S, Zaffagnini S, Bignozzi S, Bontempi M, Marcacci M. Validation of a new protocol for computer-assisted evaluation of kinematics of double-bundle ACL reconstruction. Clin Biomech (Bristol, Avon). 2006;21(3):279–87.
14. Martelli S, Lopomo N, Bignozzi S, Zaffagnini S, Visani A. Validation of a new protocol for navigated intraoperative assessment of knee kinematics. Comput Biol Med. 2007;37(6):872–8.
15. Martelli S, Zaffagnini S, Bignozzi S, Lopomo N, Marcacci M. Description and validation of a navigation system for intra-operative evaluation of knee laxity. Comput Aided Surg. 2007;12(3):181–8.
16. Martelli S, Zaffagnini S, Bignozzi S, Lopomo NF, Iacono F, Marcacci M. KIN-Nav navigation system for kinematic assessment in anterior cruciate ligament reconstruction: features, use, and perspectives. Proc Inst Mech Eng H. 2007;221(7):725–37.
17. Ntagiopoulos PG, Sharma B, Bignozzi S, Lopomo N, Colle F, Zaffagnini S, Dejour D. Are the tubular grafts in the femoral tunnel in an anatomical or isometric position in the reconstruction of medial patellofemoral ligament? Int Orthop. 2013;37(10):1933–41.
18. Sparmann M, Wolke B, Czupalla H, Banzer D, Zink A. Positioning of total knee arthroplasty with and without navigation support. A prospective, randomised study. J Bone Joint Surg Br. 2003;85(6):830–5.
19. Zaffagnini S, Martelli S, Acquaroli F. Computer investigation of ACL orientation during passive range of motion. Comput Biol Med. 2004;34(2):153–63.
20. Zaffagnini S, Martelli S, Garcia L, Visani A. Computer analysis of PCL fibres during range of motion. Knee Surg Sports Traumatol Arthrosc. 2004;12(5):420–8.
21. Zaffagnini S, Bignozzi S, Martelli S, Imakiire N, Lopomo N, Marcacci M. New intraoperative protocol for kinematic evaluation of ACL reconstruction: preliminary results. Knee Surg Sports Traumatol Arthrosc. 2006;14(9):811–6.
22. Zaffagnini S, Bignozzi S, Martelli S, Lopomo N, Marcacci M. Does ACL reconstruction restore knee stability in combined lesions?: An in vivo study. Clin Orthop Relat Res. 2007;454:95–9.
23. Zaffagnini S, Klos TV, Bignozzi S. Computer-assisted anterior cruciate ligament reconstruction: an evidence-based approach of the first 15 years. Arthroscopy. 2010;26(4):546–54.
24. Zaffagnini S, Signorelli C, Lopomo N, Bonanzinga T, Marcheggiani Muccioli GM, Bignozzi S, Visani A, Marcacci M. Anatomic double-bundle and over-the-top single-bundle with additional extra-articular tenodesis: an in vivo quantitative assessment of knee laxity in two different ACL reconstructions. Knee Surg Sports Traumatol Arthrosc. 2012;20(1):153–9.
25. Zaffagnini S, Colle F, Lopomo N, Sharma B, Bignozzi S, Dejour D, Marcacci M. The influence of medial patellofemoral ligament on patellofemoral joint kinematics and patellar stability. Knee Surg Sports Traumatol Arthrosc. 2013;21(9):2164–71.

ICL: Anatomy of the ACL and Reconstruction

6

Christian Fink, Timo Järvelä, Rainer Siebold, Robert Śmigielski, and Kazunori Yasuda

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S. Zaffagnini et al. (eds.), *ESSKA Instructional Course Lecture Book*,
DOI 10.1007/978-3-642-53983-1_6, © ESSKA 2014

6.1 Anatomy and Biomechanics of the Mid-substance and Fan-like Extension Fibres of the Femoral ACL Attachment

Kazunori Yasuda

6.1.1 Functional Anatomy of the Femoral Attachment Fibres of the ACL [14]

1. The ACL has two types of attachment margins. One is the relatively narrow oval attachment margin of the mid-substance (MS) fibres of ACL, and the other is the broader attachment margin of the "fan-like extension(FLE)" fibres, which extend from the MS fibres and broadly spread out on the posterior condyle [10, 15].
2. At the full extension position, the FLE fibres, which appear to be thin and coarse, are aligned parallel to the intercondylar roof without curving. The distinct border between the MS and FLE fibres cannot be identified.
3. At 15° and 30° of knee flexion, the border between the MS fibres and the FLE fibres becomes obvious. At 45° and 60° of knee flexion, the curving of the ACL fibres becomes an obvious fold. At 90° the whole fold becomes deeper.
4. Because the FLE fibres are adhered to the bone surface, the fibre location and orientation in relation to the femoral surface do not change, regardless of the knee flexion angle, while the orientation of the MS fibres in relation to the femur changes during knee motion. These two different structures form the fold at the border between the MS fibres and the FLE fibres.

6.1.2 Histologic Observation of the MS and FLE Fibres [14]

1. At the full extension position, the thin FLE fibres, which extend from the MS fibres, are widely attached to the postero-proximal aspect of the lateral condyle.
2. At 120° of knee flexion, a fold is observed in the MS fibres several millimetres from the bone surface. The thin FLE fibres are adhered to the bone surface in the same manner as observed in the full extension position.
3. The insertion of the MS fibres involves a cartilaginous zone, which is regarded as the direct insertion. The FLE fibres directly attach onto the bone without forming a transitional cartilaginous zone, which is regarded as the indirect insertion.

6.1.3 Discussion

1. These above-described results suggest that the load distribution mechanism from the ACL mid-substance to the femur is more complex than the previously thought.
2. As for clinical relevance of this study, it is considered to be difficult to reconstruct the natural function of the FLE fibres by creating a tunnel in the attachment area of the fan-like extension fibres, although such tunnel creation is recommended in a few clinical papers. On the other hand, the MS fibres can be reconstructed by creating a tunnel at the femoral and tibial ends of the AM or PL bundle.
3. Our recent biomechanical study (submitted) showed that, at the full extension position, a part of the force applied to the MS fibres was distributed to the FLE fibres. However, when the knee was flexed, the force was not distributed to the FLE fibres due to the presence of the fold. Therefore, it is considered to be of less value to reconstruct the fan-like extension fibres.
4. What should be anatomically reconstructed in "Anatomic ACL reconstruction"? Since 2004, we have emphasized that we should anatomically reconstruct the MS fibres of the AM and PL bundles, which can be seen in the intercondylar space [25]. The above-described facts suggest that to reconstruct the function of the MS fibres as physiologically as possible, the

two femoral tunnels should be created at the centre of each MS fibre attachment.

6.2 Ribbon-like Anatomy of Mid-substance and Tibial Crescent-Shaped ACL Insertion

Robert Śmigielski

Introduction: Previous publications described the shape of the ACL tibial insertion to be oval, regardless the concept of single- or double-bundle anatomy. The purpose of this anatomical study was to evaluate the macroscopic appearance of the tibial ACL insertion and the ACL mid-substance.

Methods: The tibial insertion of the ACL and its mid-substance was dissected in 111 human fresh frozen cadaver knees and the shape was described. The anatomical findings were documented on digital photographs as well as on video. Detailed anatomical measurements were taken. 30 knees were sent for computed tomography (CT) and magnetic resonance imaging (MRI).

Results: We observed a flat ribbon-like structure of the ACL within its mid-substance. The tibial insertion was a continuation of that flat ribbon appearance in the shape of a crescent. We observed three main types of the shape of tibial insertion site: C-type (67 % of knees), J-type (24 %) and Cc-type (9 %). The fibres of the ACL tibial footprint surrounded the attachment of the anterior horn of the lateral meniscus. 3D CT and MRI examination confirmed above findings.

Discussion: This is a detailed anatomical study describing the ribbon-like structure of the ACL from its mid-substance to the tibial attachment. The shape of the ACL was found to be wide but thin resembling a ribbon, including its tibial insertion site, which was crescent shaped.

Conclusion: In our study the shape of ACL tibial insertion site and mid-substance differs from previous studies and these findings may change the approach to anatomical ACL reconstruction.

6.3 Consequences of Ribbon-like ACL Anatomy for ACL Reconstruction Using Hamstrings

Rainer Siebold

There is rising evidence from anatomical studies that the tibial and femoral direct insertions of the mid-substance fibres of the ACL are long but narrow. On the femoral side the mean length and width are reported to be 16 mm and 4–5 mm, respectively [22]. On the tibial side the direct insertion of the mid-substance fibres is crescent shaped showing a similar length and width like on the femur (see Smigielski et al. above). The direct mid-substance ACL fibres fan out into tibial and femoral indirect insertion fibres [15, 19, 22]. The mid-substance of the ACL resembles a "ribbon-like" flat structure [15, 22].

Sasaki et al. [19] concluded from microscopic evaluations of the femoral ACL insertion that whereas the indirect insertion plays a role as a dynamic anchorage of soft tissue to bone allowing certain shear movements, the strength of anchoring is weaker than the direct insertion. Therefore the authors recommend to make the femoral tunnel on the direct insertion. Mochizuki et al. [15] concluded from their anatomical study that it is difficult to reconstruct the natural fan-like (indirect) extension fibres by creating a tunnel at the femoral and tibial ends of each fibre bundle, although the mid-substance fibres can be reconstructed by such procedures.

In a previous study on ACL footprint reconstruction, we could show that the type of ACL reconstruction does strongly influence the shape of the reconstructed footprint and ACL mid-substance [21]: The flat configuration of the ACL including its direct insertion sites cannot be anatomically reconstructed by a round SB ACL reconstruction with hamstrings. The reconstructed footprint and mid-substance ligament will usually be too wide but not long enough. A flat and long reconstruction of the mid-substance including its direct insertion sites may only be achieved a double-bundle reconstruction. In this case the reconstruction is less wide but longer [21]. A flat but wide patella tendon or quadriceps tendon graft may be a good alternative too.

In conclusion the findings of a flat ACL ligament will change our surgical approach to ACL reconstruction. A flat but long reconstruction of both insertion sites is more anatomical. When using hamstrings a DB ACL reconstruction seems to be the better choice to achieve this goal.

6.4 Alternatives for ACL Reconstruction Using Patella Tendon or Quadriceps Tendon

Christian Fink

In the 1990s patellar tendon bone-tendon-bone (BTB) autograft was termed the "gold standard" for anterior cruciate ligament (ACL) reconstruction [20]. Since 2000 hamstring grafts have become more popular [18]. Until today the graft choice for ACL reconstruction remains still a controversial matter. In a recent meta-analysis [16, 18], patellar tendon and hamstring grafts did not show significant differences in the clinical outcome – with hamstring grafts associated with slightly less morbidity and patellar tendon grafts with slightly better results for laxity.

While many knee surgeons use the quadriceps tendon (QT) as a graft for ACL revision surgery [7], it has never achieved universal acceptance for primary ACL reconstruction. Long-term outcomes over 20 years are documented for BTB grafts [5, 9, 12], whereas QT has been for a long time by far the least-studied autograft. However, during the last years QT gained increasing interest amongst orthopaedic surgeons, and in several studies QT was found to be a viable and reliable graft with minimal donor site morbidity [1–6, 8, 11, 17].

In comparison to hamstring grafts, patellar tendon and QT are more predictable when it comes to graft dimensions. Especially the QT is extremely versatile with respect to length and diameter [13, 23, 24].

The potential reason why QT has had little attention as a primary ACL graft source is that QT graft harvest is technically more demanding and a scar on the thigh cosmetically less favourable. With the use of a minimally invasive harvesting technique, these issues can be markedly reduced [6]. Adapting the geometry of the bone tunnel (squared tunnels versus round tunnels), the biomechanics of the BTB and QT reconstruction can be further improved [6].

Therefore we think that QT is becoming an increasingly attractive graft for primary ACL reconstruction and BTB will continue to play its role as a reliable graft choice.

6.5 Outcome of Randomized Controlled Trials Comparing Double-Bundle to Single-Bundle ACL Reconstruction

Timo Järvelä

According to the 21 prospective randomized studies found from the English literature and published so far, six studies (29 %) did not find any significant differences in the clinical results between double-bundle and single-bundle ACL reconstructions. However, 15 studies (71 %) reported significantly better results with the double-bundle technique than with the single-bundle technique. Nine of these trials reported better rotational stability, seven trials noted better anterior stability, three trials showed better objective knee scores, three trials had better subjective knee scores, four trials had fewer graft failures, one trial less tunnel enlargement, and one trial was found to have less degenerative changes in the knee joint with double-bundle technique compared to the single-bundle technique. In addition, none of the studies found that the single-bundle technique had better results in any of these evaluations than the double-bundle technique.

All the above randomized, controlled trials comparing double-bundle and single-bundle ACL reconstruction are summarized in the Table 6.1.

Table 6.1 Randomized controlled trials comparing double-bundle (DB) and single-bundle (SB) ACL reconstruction

Authors	Number of patients	Follow-up time	Results
Adachi et al. [26]	108	2 months	No difference
Aglietti et al.	75	2 years	Better rotational and anterior stability in DB group
Aglietti et al.	70	2 years	Better anterior stability and subjective and objective knee scores in DB group
Järvelä	65	14 months	Better rotational stability in DB group
Järvelä et al.	77	2 years	Better rotational stability and fewer graft failures in DB group
Järvelä et al.	60	2 years	Less tunnel enlargement in DB group
Yagi et al.	60	1 year	Better rotational stability in DB group
Muneta et al.	68	2 years	Better rotational and anterior stability in DB group
Streich et al.	50	2 years	No difference
Siebold et al.	70	19 months	Better rotational and anterior stability and objective knee scores in DB group
Sastre et al.	40	2 years	No difference
Zaffagnini et al.	100	3 years	Better anterior stability and objective and subjective knee scores in DB group
Zaffagnini et al.	79	8 years	Better functional scores and fewer graft failures and less degenerative changes in DB group
Wang et al.	64	10 months	No difference
Ibrahim et al.	218	29 months	Better rotational and anterior stability in DB group
Suomalainen et al.	153	2 years	Fewer graft failures in DB group
Araki et al.	20	1 year	No difference
Fujita et al.	55	2 years	No difference
Hussein et al.	281	51 months	Better rotational and anterior stability in DB group
Lee et al.	42	2 years	Better rotational stability in DB group
Suomalainen et al.	90	5 years	Fewer graft failures in DB group

References

1. Antonogiannakis E, Yiannakopoulos CK, Hiotis I, et al. Arthroscopic anterior cruciate ligament reconstruction using quadriceps tendon autograft and bioabsorbable cross-pin fixation. Arthroscopy. 2005;21:894.
2. Barie A, Huber J, Streich NA. Implant-free replacement of the anterior cruciate ligament – quadriceps tendon versus patellar tendon – long-term results of a prospective randomized clinical study. Knee Surg Sports Traumatol Arthrosc. 2012;20:34.
3. Chen CH, Chuang TY, Wang KC, et al. Arthroscopic anterior cruciate ligament reconstruction with quadriceps tendon autograft: clinical outcome in 4–7 years. Knee Surg Sports Traumatol Arthrosc. 2006;14:1077–85.
4. De Angelis JP, Fulkerson JP. Quadriceps tendon – a reliable alternative for reconstruction of the anterior cruciate ligament. Clin Sports Med. 2007;26:587–96.
5. Eriksson E. Patellar tendon or quadriceps tendon grafts for ACL reconstruction. Knee Surg Sports Traumatol Arthrosc. 2007;15:1283.
6. Fink C, Hoser C. Einzelbündeltechnik: Quadricepssehne in Portaltechnik. Arthroskopie. 2013;26:35–41.
7. Garofalo R, Djahangiri A, Siegrist O. Revision anterior cruciate ligament reconstruction with quadriceps tendon-patellar bone autograft. Arthroscopy. 2006;22:205–14.
8. Geib TM, Shelton WR, Phelps RA, et al. Anterior cruciate ligament reconstruction using quadriceps tendon autograft: intermediate-term outcome. Arthroscopy. 2009;25:1408–14.
9. Gorschewsky O, Klakow A, Putz A, et al. Clinical comparison of the autologous quadriceps tendon (BQT) and the autologous patella tendon (BPTB) for the reconstruction of the anterior cruciate ligament. Knee Surg Sports Traumatol Arthrosc. 2007;15:1284–92.

10. Hara K, et al. Anatomy of normal human anterior cruciate ligament attachments evaluated by divided small bundles. Am J Sports Med. 2009;37:2386.
11. Hoeher J, Balke M, Albers M, et al. Anterior cruciate ligament (ACL) reconstruction using a quadriceps tendon autograft and press-fit fixation has equivalent results compared to a standard technique using semitendinosus graft: a prospective matched-pair analysis after 1 year. Knee Surg Sports Traumatol Arthrosc. 2012;20:147.
12. Kim SJ, Kumar P, Oh KS. Anterior cruciate ligament reconstruction: autogenous quadriceps tendon-bone compared with bone-patellar tendon-bone grafts at 2-year follow-up. Arthroscopy. 2009;25:137–44.
13. Lippe J, Armstrong A, Fulkerson JP. Anatomic guidelines for harvesting a quadriceps free tendon autograft for anterior cruciate ligament reconstruction. Arthroscopy. 2012;28:980–4.
14. Mochizuki T, Fujishiro H, Nimura A, Mahakkanukrauh P, Yasuda K, Muneta T, Akita K. Anatomic and histologic analysis of the mid-substance and fan-like extension fibres of the ACL during knee motion, with special reference to the femoral attachment. Knee Surg Sports Traumatol Arthrosc. 2014;22: 336–44.
15. Mochizuki T, Muneta T, Nagase T, Shirasawa S, Akita KI, Sekiya I. Cadaveric knee observation study for describing anatomic femoral tunnel placement for two-bundle anterior cruciate ligament reconstruction. Arthroscopy. 2006;22(4):356–61.
16. Mohtadi NG, Chan DS, Dainty KN, et al. Patellar tendon versus hamstring tendon autograft for anterior cruciate ligament rupture in adults. Cochrane Database Syst Rev. 2011;(9):CD005960.
17. Mulford JS, Hutchinson SE, Hang JR. Outcomes for primary anterior cruciate reconstruction with the quadriceps autograft: a systematic review. Knee Surg Sports Traumatol Arthrosc. 2013;21:1882–8.
18. Samuelsson K, Andersson D, Karlsson J. Treatment of anterior cruciate ligament injuries with special reference to graft type and surgical technique: an assessment of randomized controlled trials. Arthroscopy. 2009;25:1139–74.
19. Sasaki N, Ishibashi Y, Tsuda E, Yamamoto Y, Maeda S, Mizukami H, Toh S, Yagihashi S, Tonosaki Y. The femoral insertion of the anterior cruciate ligament: discrepancy between macroscopic and histological observations. Arthroscopy. 2012;28(8):1135–46.
20. Shelton WR, Fagan BC. Autografts commonly used in anterior cruciate ligament reconstruction. J Am Acad Orthop Surg. 2011;19:259–64.
21. Siebold R, Schuhmacher P. Restoration of the tibial ACL footprint area and geometry using the Modified Insertion Site Table. Knee Surg Sports Traumatol Arthrosc. 2012;20(9):1845–9.
22. Smigielski R, et al. Ribbon concept of the anterior cruciate ligament. KSSTA J. 2013 (accepted for publication).
23. Stäubli H, Schatzmann L, Brunner P, et al. Mechanical tensile properties of the quadriceps tendon and patellar ligament in young adults. Am J Sports Med. 1999; 27:27–34.
24. Xerogeanes JW, Mitchell PM, Karsev PA, et al. Anatomic and morphological evaluation of the quadriceps tendon using 3-dimensional magnetic resonance imaging reconstruction: applications for anterior cruciate ligament autograft choice and procurement. Am J Sports Med. 2013;41(10):2392–9.
25. Yasuda K, et al. Anatomic reconstruction of the anteromedial and posterolateral bundles of the anterior cruciate ligament using hamstring tendon grafts. Arthroscopy. 2004;20:1015.
26. Adachi N, Ochi M, Uchio Y et al. Reconstruction of the anterior cruciate ligament. Single- versus double-bundle multistranded hamstring tendons. J Bone Joint Surg Br. 2004;86:515–20.

How to Address Multi-ligament Injuries?

7

Paolo Adravanti, Bent Wulff Jakobsen, Robert G. Marx, D.J. Santone, D.B. Whelan, and Andy Williams

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S. Zaffagnini et al. (eds.), *ESSKA Instructional Course Lecture Book*,
DOI 10.1007/978-3-642-53983-1_7, © ESSKA 2014

7.1 Initial Management of the Dislocated Knee

D.B. Whelan and D.J. Santone

7.1.1 Introduction

A knee dislocation – or disarticulation of the tibiofemoral articulation – is formally defined as an injury to three or more of the main stabilising ligaments around the knee, including both cruciates [1]. This definition becomes necessary with the recognition that most knee dislocations spontaneously reduce prior to assessment. Although these injuries are rare, they can result in serious injury that can jeopardise the viability of the limb and/or the future stability of the knee. The assessing physician should maintain a high suspicion for knee dislocation and the sequelae thereof for any high-energy trauma patient that presents to the trauma bay with an acute haemarthrosis and a normal x-ray [1].

7.1.2 Classification and Mechanism of Knee Dislocation

Knee dislocations can be classified via several criteria: (1) mechanism of injury (high energy, low energy, ultra low energy), (2) acuity of presentation (acute = <3 weeks and chronic = >3 weeks), (3) direction of dislocation (based on the location of tibia relative to the femur – anterior, posterior, medial, lateral or rotatory) or (4) anatomically (based on the ligamentous structures torn on MRI) (Table 7.1) [2]. Traditionally the directional classification was most popular, with anterior being the most commonly reported type followed by posterior. Anterior dislocations are usually the result of hyperextension injuries, whereas posterior dislocations are more often due to an anterior force on a flexed knee driving the tibia posteriorly (typical 'dashboard' injury). Rotatory knee dislocations also occur and involve a combination of the aforementioned tibial displacement positions but were not well described by the directional system [2].

Table 7.1 Frequency of direction of knee dislocation

Direction of dislocation	Frequency of occurrence (%)
Anterior	40
Posterior	33
Lateral	18
Medial	4

Adapted from Rihn et al. [2]

Table 7.2 Schenck classification of knee dislocations

Type	Description
KD I	Dislocation associated with multi-ligament injuries that do not include both cruciate ligaments (ACL/PCL + collateral)
KD II	Dislocations associated with bicruciate injury only (ACL + PCL, collaterals intact)
KD III L	ACL + PCL + LCL/PLC (MCL intact)
KD III M	ACL + PCL + MCL (LCL/PLC intact)
KD IV	Dislocations associated with tears of both cruciate ligaments and both posteromedial and posterolateral ligaments
KD V	Dislocation associated with a periarticular fracture

More recently the anatomical classification (originally proposed by Schenck and later modified by Wascher) has been widely adopted and is based on MRI-confirmed ligamentous pathology [3]. This system is subdivided into five subtypes. These are described in Table 7.2.

7.1.3 Neurologic and Vascular Injuries Following Knee Dislocation

Due to the proximity of major neurovascular structures around the knee, dislocations can result in limb-threatening injury. Vascular injury (popliteal artery injury) occurs in 10–40 % of knee dislocations particularly with anterior and posterior mechanisms [4]. The anatomy at the posterior aspect of the knee explains the predisposition to arterial injury. Fibrous tethering of the vessel at the adductor hiatus proximally and the soleal arch distally limits artery excursion during dislocation, causing injury with excessive anterior or posterior translation [5].

Neurologic injury following knee dislocation has been reported with frequencies of 10–35 %. The common peroneal is the most commonly injured nerve [6]. Similar to the popliteal artery, the peroneal nerve is tethered distally at the fibular neck which may enhance the incidence of a traction injury during joint distraction [7]. The nerve is somewhat superficially positioned at the fibular neck, and blunt trauma may also be a factor. The mechanism resulting in neurologic injury is most often a varus stress, whereas sagittal forces are more likely to contribute to vascular injuries.

7.1.4 Emergency Evaluation and Management of the Dislocated Knee

Knee dislocations are frequently the result of high-energy trauma, and thus patients should initially be managed according to the Advanced Trauma Life Support principles [8]. Concomitant life-threatening injuries to the head, chest and abdomen are the priority prior to the assessment of the dislocated joint. If a dislocation is present on the initial presentation in the emergency room and has not yet been reduced, then a thorough neurologic and vascular exam is completed and documented, followed by an urgent reduction of the knee under conscious sedation. Reduction most commonly follows the application of axial traction and extension of the joint. Sedation and anaesthetic also afford a thorough examination of joint laxity which can direct future treatment. A thorough neurovascular examination is then repeated and documented post reduction as is an assessment of the integrity of the soft tissue envelope and extensor mechanism.

In rare circumstances, the joint may prove irreducible via closed manipulation. This is encountered with posterolateral dislocations and usually suggests incarceration of the medial capsule and MCL within the medial compartment. In this case, open reduction is necessary to clear the entrapped tissue via a limited medial approach. The MCL can be repaired simultaneously and the medial compartment inspected [9].

Following reduction of the dislocated knee, the limb is generally stable in an extended position and should be immobilised in this position. Radiographic confirmation of reduction is imperative. Associated fractures are common and x-rays should be scrutinised for the same, especially of the fibular head, femoral epicondyles and tibial plateau [10]. The application of an external fixator may be necessary to maintain the reduction in a fractured or hypermobile knee or in the case of a vascular injury requiring repair but should not be routinely applied.

Open wounds and fractures are unfortunately common and can further complicate the definitive management of multi-ligament knee injury, especially with respect to the timing of ligament repair and/or reconstruction. Open wounds mandate serial irrigation and debridements at a minimum, and potentially coverage procedures such as flaps of grafts, to ensure an intact and aseptic soft tissue envelope. Periarticular fractures may require preliminary skeletal fixation prior to restoration of ligamentous stability via reconstructive surgery.

7.1.5 Vascular Assessment

Throughout the assessment of a knee dislocation, serial clinical vascular examinations are critical. An ankle brachial index is a reasonable screening exam in all cases of recognised dislocation or high-energy multi-ligament joint injury. Angiography (via CT or otherwise) is recommended if any vascular disturbance is appreciated clinically at any point during assessment. Figure 7.1 depicts an algorithm for the management of dislocated knees and diagnosis of arterial injury.

The presence of distal pedal pulses and a reduced dislocation should not exclude the concern of vascular injury, as collateral circulation in the lower limb can mask injuries such as occlusive popliteal thrombus and intimal tears, both of which may progress to compartment syndrome and limb-threatening ischaemia and amputation [10]. For this reason, all confirmed knee dislocations should be admitted and monitored for 24 h,

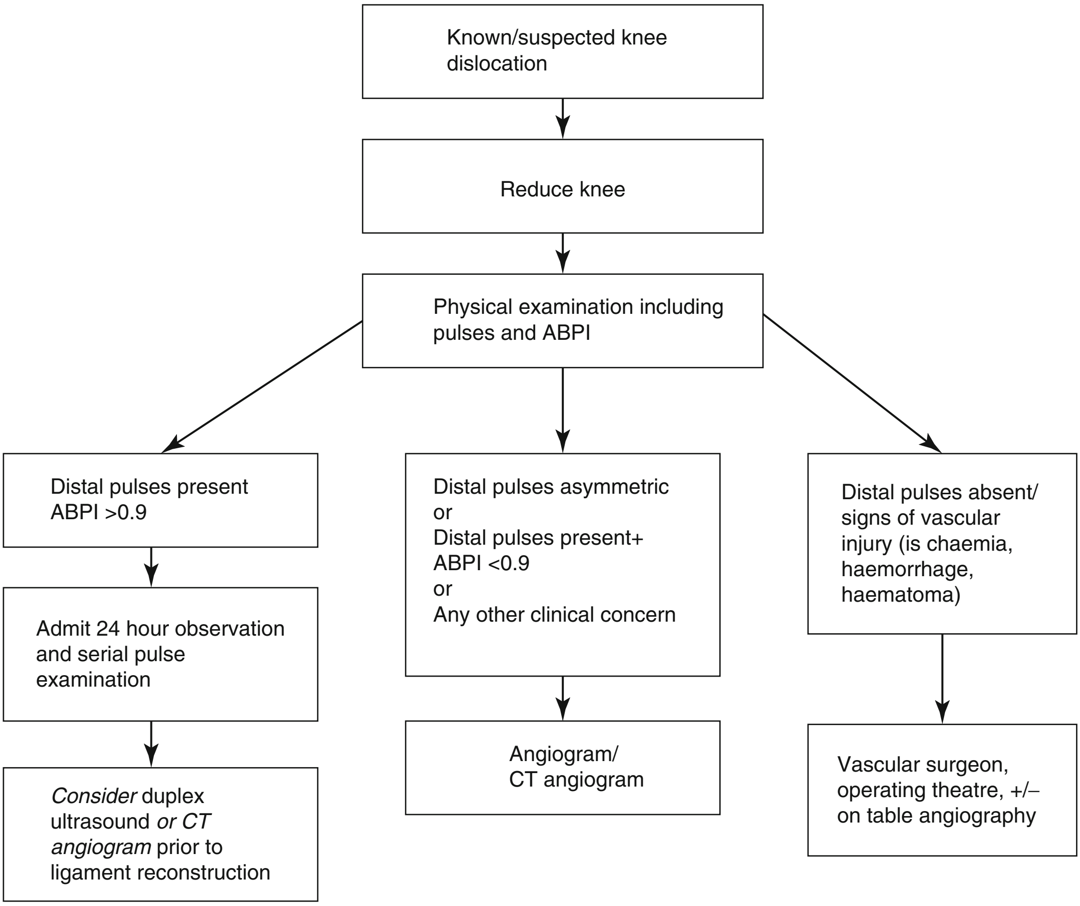

Fig. 7.1 Algorithm for diagnosis of vascular injury post knee dislocation (Adapted from Howells et al. [10]). *ABPI* ankle brachial pressure index, *CT* computerised tomography

with serial vascular exams [11–13]. An ABI is strongly recommended as a screening tool. An ABI >0.9 is considered normal and has a 100 % negative predictive value for a major arterial lesion. An ABI <0.90 requires further arterial screening [14].

Conclusion

Although knee dislocation is not a common injury, the consequences of poorly managed dislocations are serious. Management begins with identification of the injury. Determination of vascular status is imperative. Definitive delayed management of the multi-ligament injured knee is controversial.

7.2 PCL Management in the Multi-ligament Injured Knee

Paolo Adravanti

7.2.1 Introduction

Posterior cruciate ligament (PCL) injuries are rare, but they often require reconstruction, especially in the setting of combined ligamentous knee injury. In contrast to anterior cruciate ligament (ACL) tears, since PCL injuries are relatively infrequent, the information on the natural history and treatment outcomes have been limited. In a review, it has been reported that over a 10-year period, ACL injuries occurred 31 times more often than PCL ones [15]. Recent research has provided informations on the anatomy and biomechanics of the PCL, the merits and drawbacks of the transtibial compared with the tibial inlay technique, the use of single- or double-bundle reconstruction and the different graft options for reconstruction.

7.2.2 Anatomy

The PCL is divided in anterolateral and a posteromedial bundle. The anterolateral bundle is tight inflexion, while the posteromedial bundle is tight in extension. The PCL is the primary restraint to posterior tibial translation and a secondary restraint to external tibial rotation. The meniscofemoral ligaments act as secondary stabilisers to posterior translation of the tibia. With the knee at 90° of flexion, the meniscofemoral ligaments provide approximately 28 % of the total knee force resisting posterior tibial translation [16].

7.2.3 Diagnosis

7.2.3.1 Clinical Tests

There are several tests that can be used to examine for posterior instability. The posterior drawer test is the most accurate physical examination test to assess PCL injury, with a sensitivity of 90 % and specificity of 99 % [17]. The degree of posterior translation is used to determine the grade of injury: grade I (1–5 mm), grade II (6–10 mm) and grade III (>10 mm). To avoid incorrectly diagnosing an ACL injury in lieu of a PCL tear, the examiner must position the tibia in its natural position, with the medial femoral condyle resting approximately 1 cm anterior to the femur before applying the posteriorly directed force. The posterior sag test is performed with the ipsilateral hip and knee both flexed to 90°. If the PCL is torn, there may be an abnormal sag evident at the proximal anterior tibia when observed from a lateral view. The quadriceps active test is performed in supine position with the knee flexed at 90°. The patient is asked to contract the quadriceps or slide his foot down the table; the tibia will then translate anteriorly from a posteriorly subluxated position.

7.2.3.2 Imaging

MRI remains the diagnostic imaging modality of choice for the multi-ligament injured knee. Fluoroscopic or radiographic stress examination is often helpful but difficult to perform in the acute injured knee. On the other hand, in a chronic scenario, it can further clarify the extent of the ligament injuries. Posterior stress radiographs are obtained at 90° of knee flexion with an applied posterior force. Stress radiographs are evaluated according to a technique described by Jacobsen [18] using a bone peripheral bone landmarks to determine the tibial displacement relative to the tibia [19].

7.2.4 Management Strategies

Based on the current data on the natural history of PCL injury and the outcomes of operative treatment, we currently hypothesise nonoperative management for isolated grade I or II PCL tears. Operative management is reserved for PCL avulsion fractures, PCL tears associated with additional knee ligament injuries and isolated grade III PCL tears which fail nonoperative management. Treatment options including single-bundle transtibial, tibial inlay and double-bundle transtibial reconstruction should be considered in the setting of a multi-ligament injured knee. A systematic literature review examining transtibial versus tibial inlay techniques did not reveal any significant differences [20]. In the transtibial technique, the tibial and femoral tunnels are drilled, and the graft must make an acute turn as it surfaces from the tibial tunnel and changes direction before entering the knee joint. Subsequent graft abrasion and attenuation may result in graft rupture or laxity. This sharp turn has been implicated in the residual posterior knee laxity observed clinically after transtibial PCL reconstruction. The tibial inlay was developed as an alternative to the transtibial technique to avoid this problem. In the tibial inlay reconstruction, the graft is fixed directly to the PCL's native insertion site on the tibia. Recently a number of authors have also described arthroscopic tibial inlay PCL reconstruction [21]. Proposed advantages of this technique include avoiding the open posterior approach to the tibia while simultaneously avoiding unwanted effects of the acute angle on graft abrasion in tibial tunnel reconstruction. Potential disadvantages of the inlay tibial technique in the setting of the multi-ligament injured knee are the complexity of the technique, the risk of complication and the time consumed in a long surgical procedure. A wider debate regarding single-bundle versus double-bundle PCL reconstruction exists in the current literature. To date, there are no clinical studies proving an advantage of one technique over the other in the multi-ligament scenario. Markolf studied 13 cadaveric knees that had either single- or double-bundle reconstruction [22]. They found that the anterolateral bundle restores normal laxity between 45° and 90° of knee flexion; however, between extension and 30° of flexion, the anterolateral bundle is approximately 2 mm lax. They concluded that this laxity in 0–30° range may not be worth the increased operative time and extra hardware. When combined with medial-sided reconstructions, it is necessary to pay attention to the second tunnel in the MFC. In the largest study to date comparing the two techniques, Fanelli and colleagues performed a retrospective comparison of 45 single-bundle PCL reconstructions and 45 double-bundle reconstructions using the transtibial technique in a cohort of PCL-based multi-ligament injured knees [23]. No statistically significant differences were observed between the two techniques using stress radiography at 90° of knee flexion or KT-1000 measurements at 90°, 70° and 30° of knee flexion, as well as in Lysholm, Tegner and Hospital for Special Surgery Knee (HSS) scores.

The most appropriate graft choice for PCL reconstruction remains controversial because no ideal graft exists to replace this large ligament with large-footprint bony insertion-site anatomy. Hoher et al. [24] defined an ideal graft for PCL reconstruction as having the following properties: structural properties identical to an intact PCL; identical geometric shape; no harvest-site morbidity; easy graft passage; secure fixation in an anatomic position; and fast graft incorporation. Autografts and allografts have been used in PCL reconstruction, each presenting advantages and disadvantages. Achilles tendon allograft, with its large cross-sectional area, is currently the most frequently used graft for acute (43 %) and chronic (50 %) PCL reconstructions [25]. Recently, Maruyama compared bone-tendon-bone with semitendinosus and gracilis tendon autograft in a single-bundle transtibial PCL reconstruction. No differences in improvement in functional scores or posterior laxity were observed [26].

7.2.5 Surgical Technique

We routinely use a single-bundle transtibial technique in the setting of the multi-ligament injured knee. We use hamstring autograft or tibialis anterior or posterior allograft to reconstruct the PCL. Since only the anterolateral bundle of the PCL will be reconstructed, it is important to consider the anatomy of the AL bundle which inserts on the femur between 12 and 2 o' clock positions (right knee). The PCL femoral tunnel is created from an inside-out technique through a low lateral arthroscopic portal after having determined the central insertion of the AL bundle, which is not more than 7 mm away from the edge of the medial condyle cartilage. The placement of the tibial tunnel and identification of the PCL insertion require preliminary debridement of the posterior joint space through a posteromedial portal and controlling with a mirror (Fig. 7.2). All the PCL tissue should not be resected as it will provide covering for the PCL graft. We create a connection between the anterior and posterior joint compartments removing soft tissue behind the PCL scar in the high notch part. After preparation of the dorsal capsule, a guide pin is placed slightly lateral to the tibial insertion approximately 2 cm below the joint line. The graft is pulled through the bone tunnels with a passing suture. Thus, the passing suture must be routed through the tibial tunnel and then through the femoral tunnel. A PCL elevator is inserted through the posteromedial portal, and the graft passage is greatly facilitated by a careful backward motion. We use a hybrid fixation in the femur with a suspension device plus a bioabsorbable interference screw. We check the position of the graft in the back of the tibia and fix the graft into the tibia at approximately 70° of flexion with a bioabsorbable screw and backup fixation with a button.

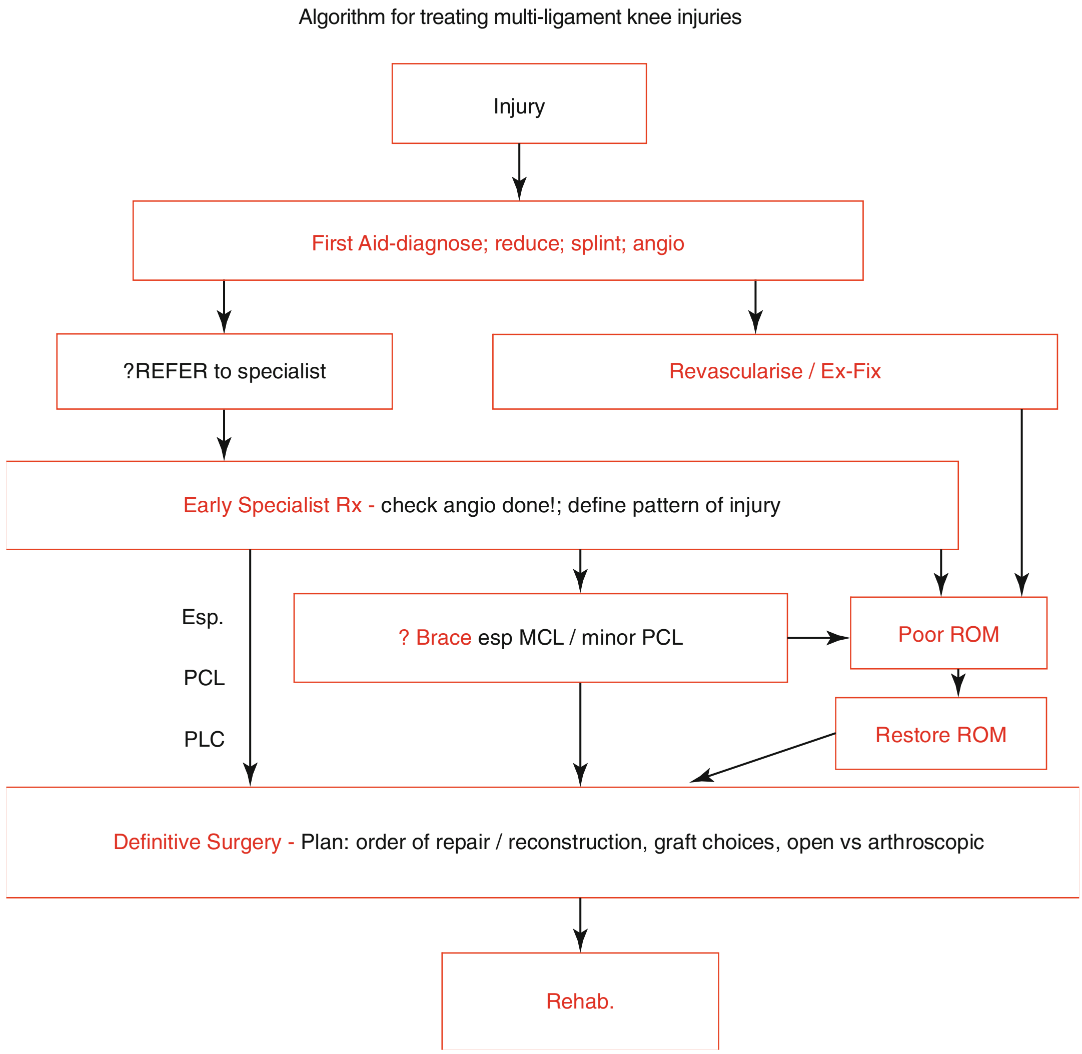

Fig. 7.2 Algorithm for treating multi-ligament knee injuries

7.3 Instructional Course Lecture: Multi-ligament Knee Injuries

7.3.1 Medial Collateral Ligament Injuries

Robert G. Marx

7.3.1.1 MCL Function

The medial collateral ligament (MCL) is the primary restraint to valgus stability of the knee. At 20–30° flexion, it provides approximately 80 % of the restraining force, whereas at full extension, it provides approximately 60 % of the restraining force with the posteromedial capsule, posterior oblique ligament (POL) and ACL providing the remaining restraint [27]. Failure in recognising dysfunction of this ligament in the setting of concomitant ligament reconstruction surgery (ACL or PCL) can result in excessive valgus stresses applied to the reconstructed ligaments, subsequently leading to graft stretch-out and failure.

7.3.1.2 MCL Anatomy

The MCL has three major components:

1. The superficial MCL which is the largest component, originating 3.2 mm proximal

and 4.8 mm posterior to the medial epicondyle and inserting on the proximal tibia, just anterior to the posteromedial crest of the tibia and posterior to the pes anserinus insertion [28].

2. The deep MCL which is a thickened part of the medial joint capsule, lying deep to the superficial part of the MCL, and has meniscotibial and meniscofemoral components. The femoral attachment is 12.6 mm distal and deep to the femoral attachment of the superficial MCL, and the tibial attachment lies just distal to the edge of the articular cartilage of the medial tibial plateau, 3.2 mm distal to the medial joint line [28].
3. The POL, functioning as an additional medial knee restraint when the knee is extended. This is a fibrous extension of the distal aspect of the semimembranosus that blends with the posteromedial joint capsule. Its major and central portion attaches on the femur 7.7 mm distal and 2.9 mm anterior to the gastrocnemius tubercle. This is just proximal and posterior to the femoral insertion of the superficial MCL [28].

7.3.1.3 Assessment of MCL Dysfunction

Physical Examination

Physical examination should begin with assessing alignment and gait. When excessive valgus is identified in the injured limb and confirmed with AP hip-to-ankle axis view, varus-directed osteotomy to correct the alignment should be thought of as a first step before ligament reconstruction is considered [29]. This may result in decreasing valgus moments and consequently may lead to the resolution of the sense of instability. Following assessment of alignment and gait, the knee is thoroughly examined, including laxity assessment of all ligaments. The uninjured contralateral knee is used as a baseline for comparison. MCL laxity should be tested and graded with valgus stress applied at 0° and at 20–30° knee flexion. MCL laxity grade 0 corresponds to 0–2 mm side-to-side medial opening difference, grade 1+ corresponds to 3–5 mm difference, grade 2+ corresponds to 6–10 mm difference and grade 3+ corresponds to more than 10 mm difference [30–32].

Imaging

Stress x-rays can also be used to provide further quantification of medial laxity. However, the amount of medial opening on stress x-rays that correlates with a specific grade of MCL laxity has not been well documented in vivo. Recently, reference values were provided, but this was tested in an old-aged population cadaveric model which may not apply to young or middle-aged living humans [33].

Examination Under Anaesthesia

The operated knee should be examined under anaesthesia and compared with the nonoperated side for range of motion and ligament laxity prior to surgery. While in the awakened patient, physical examination of MCL laxity relies both on the patient's ability to relax and the clinician's skill to detect the amount of medial opening and existence or absence of an endpoint, in the anaesthetised patient, the former is avoided allowing the clinician a more objective evaluation of ligament laxity without muscle guarding.

Arthroscopic Evaluation

Following an orderly arthroscopic examination of the knee, emphasis is applied to the medial compartment in cases where increased medial constraints laxity is suspected. Quantitative assessment of medial compartment opening can then be performed using the tip of the arthroscopic probe as a scale after its length is measured and confirmed outside the knee. Medial compartment opening of above 5 mm is suggestive of grade 2+ MCL laxity [34], whereas 10 mm or more medial opening is suggestive of grade 3+ MCL laxity [35].

7.3.1.4 Surgical Approaches to Address MCL Dysfunction

Direct Repair

An absolute indication for direct repair of the MCL is bony avulsion with displacement of the femoral insertion of the ligament.

Medial and Posteromedial Plication/Reefing:

In cases where residual medial laxity at the end of a cruciate reconstruction remains and arthroscopic assessment reveals grade 2+ medial laxity (medial opening >5 mm), reefing of the medial constraints should be considered, unless chronic distal lesions with poor-quality scarring exclude this alternative [34].

MCL Reconstruction

In cases where grade 3+ medial laxity is observed (medial opening >10 mm), reconstruction of the MCL should be considered. Surgical techniques which have been described to reconstruct the MCL include semitendinosus autograft with preservation of the tibial insertion [36–39], allograft tissues [40, 41] and double-bundle reconstructions [32, 38, 40–42]. Drawbacks related to these techniques include a long incision across the medial aspect of the knee with up to 20° loss of knee flexion or extension in 20 % of the operations [39], keeping the semitendinosus insertion distally and using it as an MCL graft [36–39] resulting in a too anterior tibial attachment (i.e. the tibial insertion of the MCL should be posterior to the pes anserinus [28, 43]), harvesting a dynamic medial stabiliser that applies adduction moment during gait (i.e. semitendinosus) in a knee with an already medial instability, and the relative complexity of double-bundle reconstructions, compared to single-bundle reconstructions, corresponding to their need for multiple attachment sites on the femur as well as on the tibia, more graft tissue and number of fixation devices (i.e. screws, washers, staples, etc.) required [32, 38, 40–42]. Recently, a new technique to reconstruct the MCL has been described that uses Achilles tendon allograft [35]. Benefits include avoiding donor site morbidity, secure fixation with bone-to-bone healing on the femur, small skin incisions that do not cross the knee and isometric reconstruction.

Surgical Technique [35]

With the patient under anaesthesia, after confirming MCL laxity that requires reconstruction as indicated previously by physical examination and arthroscopic examination, the following steps are carried out (after fixing the cruciate graft on the femur): (1) the Achilles allograft is prepared creating a 9-mm diameter by 18-mm length bone plug; (2) a 3-cm longitudinal skin incision is made over the medial femoral epicondyle; (3) a guide pin is inserted 3–5 mm proximal and posterior to the medial femoral epicondyle, parallel to the joint line, and in a 15° anterior direction to avoid the intercondylar notch. Location of the pin is confirmed with fluoroscopy; (4) the skin is undermined from the femoral guide pin to the anatomic MCL insertion on the tibia, creating a tunnel for the graft under the subcutaneous fat; (5) a nonabsorbable suture loop is placed around the guide pin and brought distally under the skin through the tunnel just created; (6) the distal suture is held against the tibia at the estimated anatomic insertion, just posterior to the pes anserinus insertion. Isometricity is tested through knee motion from 0° to 90°. The tibial insertion point is modified, if needed, until the loop is isometric; (7) the isometric point is marked on the tibia with a Bovey; (8) soft tissue around the guide pin is debrided to allow for insertion of the Achilles bone plug into a socket created around this pin later; (9) a 9-mm diameter reaming is performed over the guide pin to a depth of 20 mm; (10) the Achilles bone plug is inserted into the femoral socket and fixed with a 7-mm diameter by 20-mm length metal interference screw; (11) the Achilles tendon tissue is passed under the skin and distally; (12) the cruciate graft is now tensioned and fixed on the tibia; (13) the MCL graft is tensioned with the knee at 20° flexion under varus stress and fixed at the isometric point on the tibia with a 4.5-mm cortical screw and a 17-mm spiked washer; and (14) subcutaneous tissue and skin are closed. Tunnels position and hardware placement are confirmed postoperatively with radiographs.

Outcomes After MCL Reconstruction

In the literature, except for this recent description of an MCL reconstruction technique using Achilles allograft [35], there are only two studies reporting ROM and function in patients that had MCL reconstruction with one similar graft tissue in all patients and a similar specifically described MCL reconstruction technique in a combined MCL and another cruciate reconstruction [38, 39]. Both described a technique that uses the semitendinosus tendon with preservation of the insertion site at the pes anserinus on the tibia, creating anterior and posterior limbs to reconstruct the MCL. However, in both studies, the group of patients was heterogeneous and included isolated

MCL reconstructions as well as concomitant cruciate reconstructions, but ROM was reported for all patients as one group, not differentiating the combined reconstructions from the isolated MCL reconstructions. In one of these, which included six cases of isolated MCL reconstruction and 18 cases of MCL with another cruciate reconstruction, the investigators found motion limitation between 5° and 10º in extension or in flexion in five patients (21 % of the patients) [38], whereas in the other study, which included 11 cases of isolated MCL reconstruction and 39 cases of MCL with another one or both cruciate ligament reconstructions or posterolateral corner reconstruction, the investigators noticed motion loss of between 5° and 20º in extension or in flexion in 10 patients (20 % of the patients) [39]. Both studies did not report ROM specifically for the combined reconstructions, and therefore the comparison to the technique described here, using the Achilles allograft, is limited. The fact that all MCL grafts, using the Achilles allograft technique [35], demonstrated grade 0–1+ valgus laxity on physical examination is comparable to previous reports after double-bundle MCL reconstruction in a combined ligament reconstruction scenario that described grade 0–1+ valgus laxity in more than 90 % of their cases [38, 39]. Mean IKDC-subjective and Lysholm knee scores demonstrated excellent (i.e. above 90 points) [44–46] function in patients with MCL reconstruction and primary ACL reconstruction, using the Achilles allograft technique [35], and are comparable to the mean Lysholm score reported by others when creating a double-bundle MCL reconstruction with the semitendinosus, preserving its tibial insertion [38]. Mean KOOS subscores in the Achilles allograft technique were between 77 and 96 for the five categories of the score in cases with primary ACL reconstruction, which is comparable to another study that created a double-bundle MCL reconstruction and reported mean KOOS subscores between 75 and 89 for MCL reconstruction in a multi-ligament reconstruction scenario, the vast majority of which were MCL with ACL reconstructions [39]. In patients with the Achilles allograft MCL reconstruction with revision ACL reconstruction, IKDC-subjective, Lysholm and KOOS subscores demonstrated inferior outcome [35]. Tegner and Marx activity level scores demonstrated patients with concomitant primary ACL reconstruction were able to return to pre-injury activity levels, which were at means of between 6 and 7 points, indicating that cutting and pivoting sports on a recreational level may be a realistic goal after this type of Achilles allograft MCL reconstruction, but when this technique is performed in the setting of revision ACL reconstruction, return to pre-injury activity levels may not be achieved despite regaining normal knee laxity.

7.4 Managing Lateral Collateral Ligament and Posterolateral Corner Injuries

Bent Wulff Jakobsen

7.4.1 LCL and Posterolateral Compartment Function

The lateral collateral ligament (LCL) is the primary restraint to varus stress in the knee. At 5° of knee flexion, it provides 55 % of restraint, and at 25° it provides 69 %. The popliteus structure limits posterior tibial translation, external tibial rotation and varus rotation.

Injuries to the LCL and posterolateral compartment result when a force is directed at the medial side of the knee or leg. These injuries occur in about 7–16 % of all knee ligament injuries. They are reported less commonly than injuries to the medial collateral ligament as they are less frequently recognised and are usually prevented by the presence of the opposite leg which can block direct blows to the knee's medial side.

7.4.2 LCL and Posterolateral Corner Anatomy

The lateral compartment of the knee contains both dynamic and static stabilisers. The dynamic stabilisers include the biceps femoris, the iliotibial band, the popliteus muscle and the lateral head of the gastrocnemius muscle. The lateral collateral ligament, the popliteo-fibular ligament and the arcuate ligament make up the static ligamentous complex.

The lateral capsular complex of the lateral aspect is further divided into three parts. The anterior third is attached to the lateral meniscus anterior to the LCL. The middle third is attached proximally at the femoral epicondyle and distally at the proximal tibia, while the posterior third is found posterior to the LCL.

7.4.3 Evaluation of Lateral Compartment Injuries

7.4.3.1 History

A direct force applied to the weight-bearing knee which causes excessive varus stress, external tibial rotation and/or hyperextension can cause lateral ligament injury. This force commonly demonstrates as a posterolaterally directed force to the medial tibia while the knee is in an extended position. This frequently occurs during motor vehicle accidents and athletic injuries. Injury to the lateral and posterolateral structures often occurs in conjunction with cruciate injury rather than as isolated injuries. Patients with this injury present with instability of the knee near full extension. They may have difficulty ascending and descending stairs and performing cutting or pivoting movements. They may also complain of lateral joint line pain.

7.4.3.2 Physical

The injured extremity should be examined by performing adduction stress at both 0° and 30° of knee flexion. Signs of posterolateral injury include footdrop, peroneal nerve injury, posterolateral corner tenderness and pain with posterior internal rotation of the tibia. If the knee demonstrates isolated laxity at 30°, injury to the LCL is likely. Laxity at both 0° and 30°, however, is indicative of combined injury to the LCL and ACL, PCL or arcuate complex.

In chronic injuries, it is also important to evaluate their gait. This can help determine whether there is a varus or hyperextension thrust. Neurovascular injuries must also be assessed, as peroneal nerve deficits have been reported along with acute posterolateral corner injuries in up to 29 % of cases.

7.4.3.3 Imaging

Plain radiographs should be taken for all patients suspected to have injuries to their LCL and posterolateral corner. These images are used to rule out associated osteochondral fracture, fibular head avulsion, Gerdy tubercle avulsion and fracture of the tibial plateau. MRI is the preferred imaging tool to assess the integrity of the LCL, popliteus tendon and cruciate ligaments. MRI can be used to determine the severity and location of the knee injury.

7.4.3.4 Classification

Isolated injury to the LCL rarely results in coronal plane laxity. Rotatory instability demonstrating as multiplanar laxity can be seen with combined injury to the LCL and other structures such as the ACL and mid-third capsular ligament or arcuate ligament, popliteus tendon and fabellofibular ligament. Chronic isolated injury to the LCL rarely occurs. Most patients with this type of injury will eventually develop injury patterns of the other posterolateral corner structures.

Grading of posterolateral corner injuries (grade I, II or III) is determined based on the degree of ligament disruption – minimal, partial or complete. However, a more precise method exists which relies on quantification of lateral joint opening with varus stress.

7.4.4 Treatment Approaches

7.4.4.1 Nonsurgical

Nonsurgical treatment is indicated by grade I and II (partial) isolated injuries of the LCL. Such patients have little functional instability. Nonsurgical treatment consists of limited immobilisation with protected weight bearing during the first 2 weeks after injury. As the patient improves, progressive ROM, quadriceps strengthening and functional rehabilitation are encouraged. The patient may return to sports in about 6–8 weeks. If associated injuries to the posterolateral structures fail to be identified, nonsurgical treatment can, however, lead to progressive varus/hyperextension laxity.

7.4.4.2 Surgical

Surgical treatment is indicated by complete injuries or avulsions of the LCL, rotatory instabilities of the LCL and arcuate ligament, popliteus tendon and fabellofibular ligament and combined instability patterns of the LCL/posterolateral corner and ACL or PCL.

Acute injuries can be treated surgically by primary repair of torn or avulsed structures or reconstruction of the tissue. Direct repair can be hindered by the formation of scar tissue and distortion of the tissue planes, so it must be done within 2 weeks if performed. Reconstruction has been shown to have a lower failure rate than isolate repair in two comparative studies.

Surgical treatment of chronic LCL and posterolateral corner dysfunction can be done using allograft tissue to form a single-stranded graft. Some authors advocate a separate graft to reconstruct the popliteo-fibular ligament from the femur to the tibia, but no comparative studies have been done.

Full-length upright radiographs of both lower extremities of patients with chronic instability should be taken to determine whether or not varus mechanical axis is present. If so, a high tibial osteotomy may be indicated.

7.4.5 Complications

Chronic injuries can result in persistent varus or hyperextension laxity due to advancement of attenuated lateral and posterolateral structures. Surgical treatment may cause peroneal nerve injury as a result of exposure of the fibular neck or during drilling or graft passage. Hardware irritation at the lateral femoral condyle may also occur, and knee range of motion may be lost, as can occur after the reconstruction of multiple knee ligaments.

7.5 Multi-ligament Injuries in Elite Athletes

Andy Williams

7.5.1 Background

There are multi-ligament injuries: those resulting from a tibiofemoral dislocation are devastating, while an ACL + MCL may effectively be no more than an ACL rupture. Traditionally, knee dislocations have had bad outcomes and for elite sportsmen have been career ending. Nonsurgical treatment results in stiffness, fixed subluxation deformities and instability. My view has been developed after my training and with an experience of over 300 multi-ligament cases treated surgically in the past 13 years in my public and private practice.

I am certain that these injuries should have rapid referral to specialist centres for surgical repair/reconstruction. The vast majority should have this within 2–3 weeks from injury.

I have an unusual practice such that 70 % of all patients I see are professional athletes, and I am the primary knee surgeon for 50 % English Premier League Football clubs and 75 % of English Premiership Rugby among many other teams. As a result, although multi-ligament injuries are relatively uncommon, I have acquired a series of over 40 cases in elite athletes. The results at minimum 2 years follow-up will be available at ESSKA 2014.

7.5.2 'Immediate' Management

Many cases receive suboptimal early management which can affect outcome long term. While not ignoring other injuries if part of a multiply injured patient, specifically considering the knee injury, the following is suggested:

1. Reduce the knee
2. Establish neurovascular status – arteriogram (Doppler US does not show intimal tears). If there is a significant arterial injury, then vascular surgeons should deal with the artery

first, and ligament reconstruction should be delayed 6–12 weeks. The knee should be held with an external fixator spanning the knee. This is the only reason for using an external fixator. It is essential that the knee is perfectly reduced during this period. Sadly it is often not in my experience. As a result, contracture develops with a fixed subluxation – usually posterior tibial subluxation from PCL insufficiency. Ideally the knee is held fully extended. If this is not acceptable to the vascular surgeon, then the period during which the knee is held flexed should be minimised.

3. Establish the pattern of ligament injury by clinical examination (later confirmed by examination under anaesthetic when the patient goes for definitive surgery – EUA alone is rarely needed) and x-ray/MRI.
4. Treat soft tissues. Compression boots, e.g. Flowtron, massage, ice and active movements especially in the leg will reduce swelling. Controlled movement is encouraged. Full extension must be restored as a priority. Flexion helps reduce swelling and post-op stiffness. This can be undertaken on CPM or be assisted by the physiotherapist. Anterior drawer movements should be undertaken to prevent development of fixed posterior subluxation which frequently accompanies complete PCL disruption. The gastro-soleus complex should be stretched especially if there is common peroneal nerve palsy and a resting splint used to prevent contracture – it is remarkable how frequently this is not done, and the patient has irreversible equinus contracture which requires surgical release prior to dealing with the knee.
5. Do not undertake transpatellar Steinmann pin fixation to the tibia – 'olecranonisation'.
6. Brace in a hinged brace set according to muscular control. If poor stability, then fix straight for walking. If good, then can allow movement. Weight bearing is good if possible to encourage neuromuscular control and stretch the calf.
7. Plan surgery – Which structures need repair/reconstruction? Which grafts – auto- or allograft?
8. Counsel patient.

7.5.3 Early Management

My philosophy for early surgery is to provide the joint with early congruent reduction and sufficient ligamentous stability to allow early controlled motion which is good for articular surface, prevention of contracture and limb neuromuscular function. Leaving some structures lax (e.g. leaving the PCL component) will lead to overstress of the ligaments that have been repaired/reconstructed, and they will fail.

The ideal time for surgery is 10–14 days. Any sooner and the risk of compartment syndrome is very high, and later and early scarring makes anatomical repairs difficult and puts the common peroneal nerve at risk in cases including PLC disruption. Three weeks after injury is the likely limit for repair surgery.

While I have always done surgery as a single procedure, I realise that prolonged operative times lead to rising complication rates and staged surgery can be advantageous.

It is important to note that I have learnt that NOT all cases need early surgery – some ACL + PCL + MCL tears (with grade 1 or even 2 PCL injury and favourable MCL disruption) can be braced to allow healing of the MCL and possibly PCL and therefore only require ACL reconstruction several weeks later. In elite sportsmen, an important concept is of doing as little as possible to get the result. However, if there is a grade 3 (and most grade 2 PCL laxity) or a significant posterolateral corner injury, early surgery is best. In my series of 141 multi-ligament injuries in the general population, only those operated upon acutely achieved high-level function.

In the nonathlete population, significant varus/valgus often benefits from a corrective osteotomy. However, this magnitude of surgery may preclude return to elite sport. I have only successfully returned an elite skier to competition at the same level after HTO.

At surgery, the following are the key points:

1. EUA to confirm the ligaments which need addressing.
2. Arthroscopy and meniscal/chondral surgery. Beware compartment syndrome – continually

check calf tension. If the calf fills with fluid, significantly deflate the tourniquet and convert to open surgery. Stopping fluid and operating with 'dry arthroscopy' is useful safeguard when possible.

3. Prepare grafts – my preference is autograft especially hamstrings. I only use patellar tendon for a third ligament, having harvested bilateral thighs first for hamstrings, for reasons of speed and morbidity. Since graft harvest is now rapid, I use less allograft than before.
4. All injured ligaments are dealt with at one operation if possible. This should help maintain a congruent reduction, which helps the soft tissue envelope to heal with proper tension and allows early protected joint motion which optimises soft tissue healing, reduces problems of stiffness and fat pad contracture and helps nourish the articular cartilage.
5. Cruciate ligaments – sometimes these can be repaired, e.g. avulsion fractures or peel-off of ligament from bone. If both are torn, then PCL is the priority; if there is only time to do one, do the PCL. The notion that leaving the PCL but reconstructing the other ligaments leaves a knee with 'isolated PCL deficiency' is completely wrong – the PCL rupture in these cases is associated with much greater damage to secondary restraints such as the capsule. If the anterior meniscofemoral ligament is seen to be intact, then I now preserve it when reconstructing the PCL as the posteromedial bundle of the PCL is likely to be preserved. Use an intraoperative x-ray to optimise tunnel placement – ACL as well as PCL. The graft(s) is/are passed and fixed in the femur. Always consider open surgery if fluid extravasation occurs – it is easy and the morbidity minor.
6. Collateral ligament injuries are repaired with the tourniquet deflated. Medially repair is expected and helped by detailed understanding of the three-layer anatomy (Warren and Marshall *JBJS* (*Am*) 1979). Laterally, repair is also undertaken. For mid-substance ruptures, I always add a reconstruction to protect the repair while it is healing. Exceptions are for repairs of avulsed ligaments back to bone where the repair is often sufficiently robust on its own.
7. Ligament tensioning and fixation is as follows: PCL with knee at 70°, then ACL with knee straight and finally collaterals at 30°.
8. Brace – fixed straight for first 24 h, then released according to surgery undertaken. With PCL reconstruction, early flexion range is restricted (or knee held straight).
9. IV antibiotics.
10. Physiotherapy protocol.

7.5.4 The Recovery

I expect to obtain normal ligament laxity at surgery-end.

Many months are required. The final result takes about 2 years to occur.

7.5.5 Prognosis

The best results are in motivated, slim, healthy, young, patients. Fat, older, diabetic, unathletic patients have poor outcomes. The presence of significant chondral damage is very bad in terms of prognosis. The smaller the ligament injury, the better. Previous unsuccessful surgery is problematic. A persistent footdrop is a limiter of outcome when present and for the elite athlete career ending.

Effective and good early surgery produces the very best outcome. Bad surgery is worse than none!

Some patients get back to a high-level function including sports, but I have learned that this is not common and is perhaps ill-advised for those not earning their living from it. These joints are never normal and have permanent and significant proprioceptive deficit. Like a Charcot joint, the chondral surfaces can't cope with compromised neuromuscular control. I have had some patients get back to sport only to then steadily and depressingly rapidly deteriorate. I now try to stop nonathlete patients returning to running sports. The prognosis is also much better with an experienced surgeon. There is a 'learning curve'. My early cases had significantly less certain outcomes than those now.

I am grateful to those many early patients who allowed me to build experience and to those who kindly referred them.

7.5.6 Rehabilitation and Return to Sports after Multi-ligament Injuries

The first important message for the patient is that the final result from multi-ligament reconstruction will probably take 2 years as a minimum to occur. Furthermore, the knee will never be normal. It is impossible to restore normal proprioceptive function with reconstruction. For this reason, whenever possible, tissue should be repaired in the hope that proprioception is optimised. As stated above, even in the best cases when ligament laxity is minimal, the knee will have major proprioceptive deficit and so is effectively a 'Charcot joint'. This fact must be made clear to patients so that sports involving repetitive loading such as distance running are discouraged.

Rehabilitation of acute repair/reconstruction is much harder than rehab following surgery for chronic cases. The reason is the amount of inflammation due to the massive soft tissue injury.

Another important principle is that the PCL surgery dominates the rehabilitation. Note that the PCL is tensioned with flexion angles above 60° and stressed by gravity tending to produce a posterior tibial translation. The PCL is also tensed by open-chain hamstring activity. Not surprisingly, it is prone to stretching.

7.5.7 Swelling

Flowtron and Game Ready ice/compression can be helpful.

7.5.8 Soft Tissue Stiffness

Early patellar mobilisations are important to prevent a fat pad contracture. Full active and passive extension should be encouraged as soon as possible. Although collateral ligaments are tensioned in terminal extension, surgical technique should be good enough to allow for this. If the PCL is normal, then flexion is regained as tolerated. If, however, PCL reconstruction has been undertaken, then flexion has to be restricted early on as the PCL hamstring graft will stretch. Currently I use the PCL Jack brace which pushes the tibia forward with a spring-loaded mechanism during flexion. I am therefore happy for cases with PCL reconstruction to flex up to 60° from the outset. In the absence of this brace, I would advocate bracing the knee straight for 3/4 weeks. In cases using the PCL Jack brace with PCL reconstruction, I increase flexion to 90° at the end of the fourth week. After 6 weeks, flexion is steadily increased to full at between 12 and 16 weeks. Due to the effect of gravity, if the patient is supine, then flexion exercises should be undertaken with the therapist pulling the tibia forward. Alternatively flexion exercise can be done with the patient prone.

7.5.9 Weight-Bearing Status

If the limb has neutral alignment, then I am happy for the patient to partially weight-bear for 4 weeks followed by full weight bearing. In chronic cases, of course, I wouldn't accept malalignment and osteotomy would be undertaken, but in acute cases one has to accept it. If there is any significant varus/valgus that would compromise a ligament repair/reconstruction, then the patient remains touch weight bearing for 4 weeks and partial weight bearing for 2 weeks.

7.5.10 Strengthening

If a PCL reconstruction has been undertaken with soft tissue, I do not allow isolated active hamstring exercises for 12 weeks. In addition, due to the hamstring co-contraction that occurs with closed-chain quadriceps' work, I do not allow loaded flexion exercises past 60° for 12 weeks. Around 70° of flexion, the net effect of the hamstring pull and the quadriceps' pull via the patella tendon is to produce posterior tibial translation. This will obviously stress the PCL graft.

For the quadriceps, open-chain knee extensions are allowed if no ACL reconstruction has been undertaken. If the ACL is reconstructed, then open-chain quadriceps' work is left until after 3 months. Closed-chain quadriceps' work is the mainstay. This is limited to 60° if PCL reconstruction is undertaken as above. Furthermore, due to the effect of gravity, the use of the leg press machine can be problematic. Closed-chain quadriceps' work therefore is largely done with the patient in a vertical stance.

7.5.11 Proprioceptive Control

The use of balance drills is instituted as soon as single-leg full weight-bearing stance is allowed. The reason for the very slow recovery following surgery for these terrible injuries is largely due to the slowness in restoration of proprioception. Structures other than those reconstructed have to upregulate their proprioceptive function.

7.5.12 Trunk/Pelvic Stability

From the early stage, it is vital to work on trunk and pelvic stability as this has a major effect on the stability and shock absorbency of the knee.

7.5.13 Return to Sport

Multi-ligament reconstruction obviously encompasses a wide spectrum of magnitude of injury. Many of our skiers have combined ACL and MCL injury, but of course only the minority require surgery to the MCL. The vast majority of two ligament injuries will return to sport even at the highest level. Some three ligament injury cases return to sport at the highest level but only a few four ligament injuries. I have the largest single-surgeon series of multi-ligament reconstructions and the largest series multi-ligament reconstructions in professional sportsmen. Data from these series of this ongoing study will be presented.

The incomplete results are:

Thirty-seven (there are over 40) patients met our criteria; this included 9 rugby players, 22 footballers and 6 from other sports. Thirteen patients played sport at international level.

Eight athletes required reconstruction of the ACL/MCL. Seventy-five percent (six cases) returned to their pre-injury level of sport at a mean time of 11 months post surgery. Of the two who failed to return to pre-injury levels, one reinjured the ACL and underwent revision surgery, returning to professional football at a lower division. The second patient had associated chondral injuries treated with microfracture. At 18 months post surgery, he is yet to return to competitive sport. (Minimum 2 year data will be presented.)

Eleven of the 15 patients (73 %) who underwent ACL and PLC/LCL reconstructions returned to their pre-injury level of sport at a mean time of 12.4 months post surgery. Three footballers returned to professional sport at a lower level, and one failed to return to professional sport. One professional rugby player had reconstructions of ACL/LCL and MCL. He returned to his pre-injury level in 11 months and improved to international level at 18 months post surgery. A footballer with the same injury also made a full recovery.

Four out of the six patients (67 %) that sustained bicruciate ligament injuries returned to pre-injury level sport at a mean of 14 months post surgery. One returned at lower level, and one gave up professional sport.

References

1. Scheid D. Treatment of the multiple ligament injured knee and knee dislocations: a trauma perspective. Instr Course Lect. 1993;52:409–11.
2. Rihn J, Cha P, Groff Y, Harner CD. The acutely dislocated knee: evaluation and management. J Am Acad Orthop Surg. 2004;12:334–46.
3. Wascher DC. High velocity knee dislocation with vascular injury. Treatment principles. Clin Sports Med. 2000;19:457–77.
4. Stannard JP, Shells TM, Lopez-Ben RR, McGwin G, Robinson JT, Volgas DA. Vascular injuries in knee dislocations: the role of physical examinations in determining the need for angiography. J Bone Joint Surg Am. 2004;86-A(5):910–5.

5. Good L, Johnson RJ. The dislocated knee. J Am Acad Orthop Surg. 1995;3(5):284–92.
6. Niall DM, Nutton RW, Keating JF. Palsy of the common peroneal nerve after traumatic dislocation of the knee. J Bone Joint Surg Br. 2005;87(5):664–7.
7. Peskun CJ, Levy BA, Fanelli GC, Stannard JP, Stuart MJ, MacDonald PB, Marx RG, Boyd JL, Whelan DB. Diagnosis and management of knee dislocations. Phys Sportsmed. 2010;4(38):101–11.
8. Merritt AL, Wahl C. Initial management of the acute and chronic multiple-ligament injured (dislocated) knee. Sports Med Arthrosc. 2011;19(2):93–103.
9. Silverberg DA, Acus R. Irreducible posterolateral knee dislocation associated with interposition of the vastus medialis. Am J Sports Med. 2004;32:1313–6.
10. Howells NR, Brunton LR, Robinson J, Porteus AJ, Eldridge JD, Murray JR. Acute knee dislocation: an evidence based approach to the management of the multiligament injured knee. Injury. 2011;42:1198–204.
11. Lynch K, Johansen K. Can Doppler pressure measurement replace "exclusion" arteriography in the diagnosis of occult extremity arterial trauma? Ann Surg. 1991;214:737–41.
12. Johansen K, Lynch K, Paun M. Non invasive vascular tests reliably exclude occult arterial trauma in injured extremities. J Trauma. 1991;31:515–9.
13. 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.
14. Stannard JP, Schenck RC Jr., Fanelli GP. Knee dislocations and fracture-dislocations. Rockwood and Green's Fractures in Adults 7th ed. Bucholz RW, Heckman JD, Court-Brown CM, Tornetta P, McQueen M, Ricci WM, eds. Lippincott Williams & Wilkins, Philadelphia; 2009.
15. Majewski M, Susanne H, Klaus S. Epidemiology of athletic knee injuries: a 10-years study. Knee. 2006;13:184–8.
16. Gupte CM, Bull AM, Thomas RD, Amis AA. The meniscofemoral ligaments: secondary restraints to the posterior drawer. Analysis of anteroposterior and rotary laxity in the intact and posterior-cruciate-deficient knee. J Bone Joint Surg Br. 2003;85:765–73.
17. Rubinstein Jr RA, Shelbourne KD, McCarroll JR, VanMeter CD, Rettig AC. The accuracy of the clinical examination in the setting of posterior cruciate ligament injuries. Am J Sports Med. 1994;22: 550–7.
18. Jacobsen K. Stress radiographical measurement of the anteroposterior, medial and lateral stability of the knee joint. Acta Orthop Scand. 1976;47(3):335–4.
19. Schulz MS, Russe K, Lampakis G, Strobel MJ. Reliability of stress radiography for evaluation of posterior knee laxity. Am J Sports Med. 2005; 33:502–6.
20. May JH, Gillette BP, Morgan JA, Krych AJ, Stuart MJ, Levy BA. Transtibial versus inlay posterior cruciate ligament reconstruction: an evidence–based systematic review. J Knee Surg. 2010;23:73–9.
21. Kim SJ, Choi CH, Kim HS. Arthroscopic posterior cruciate ligament tibial inlay reconstruction. Arthroscopy. 2004;20 Suppl 2:149–54.
22. Markolf KL, Feeley BT, Jackson SR, McAllister DR. Biomechanical studies of double-bundle posterior cruciate ligament reconstructions. J Bone Joint Surg Am. 2006;88:1788–94.
23. Fanelli GC, Beck JD, Edson CJ. Single compared with double bundle PCL reconstruction using allograft tissue. J Knee Surg. 2012;25:59–64.
24. Hoher J, Scheffer S, Weiler A. Graft choice and graft fixation in PCL reconstruction. Knee Surg Sports Traumatol Arthrosc. 2003;11:297–306.
25. Dennis MG, Fox JA, Alford JW, Hayden JK, Bach Jr BR. Posterior cruciate ligament reconstruction: current trends. J Knee Surg. 2004;17:133–9.
26. Maruyama Y, Shitoto K, Baba T, Kaneko K. Evaluation of the clinical results of posterior cruciate ligament reconstruction-a comparison between the use of the bone tendon bone and semitendinosus and gracilis tendons. Sports Med Arthrosc Rehabil Ther Technol. 2012;4:30.
27. Grood ES, Noyes FR, Butler DL, Suntay WJ. Ligamentous and capsular restraints preventing straight medial and lateral laxity in intact human cadaver knees. J Bone Joint Surg Am. 1981;63:1257–69.
28. La Prade RF, Engebretsen AH, Ly TV, Johansen S, Wentorf FA, Engebretsen L. The anatomy of the medial part of the knee. J Bone Joint Surg Am. 2007;89:2000–10.
29. 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;14:96–104.
30. Fanelli GC, Harris JD. Late medial collateral ligament reconstruction. Tech Knee Surg. 2007;6:99–105.
31. 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;55:923–40.
32. Wijdicks CA, Griffith CJ, Johansen S, Engebretsen L, La Prade RF. Injuries to the medial collateral ligament and associated medial structures of the knee. J Bone Joint Surg Am. 2010;92:1266–80.
33. 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.
34. Canata GL, Chiey A, Leoni T. Surgical technique: does mini-invasive medial collateral ligament and posterior oblique ligament repair restore knee stability in combined chronic medial and ACL injuries? Clin Orthop Relat Res. 2012;470(3):791–7.
35. 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.
36. Azar FM. Evaluation and treatment of chronic medial collateral ligament injuries of the knee. Sports Med Arthrosc. 2006;14:84–90.

37. Bosworth DM. Transplantation of the semitendinosus for repair of laceration of medial collateral ligament of the knee. J Bone Joint Surg Am. 1952;34: 196–202.
38. Kim SJ, Lee DH, Kim TE, Choi NH. Concomitant reconstruction of the medial collateral and posterior oblique ligaments for medial instability of the knee. J Bone Joint Surg Br. 2008;90:1323–7.
39. 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.
40. Borden PS, Kantaras AT, Caborn DNM. Medial collateral ligament reconstruction with allograft using a double-bundle technique. Arthroscopy. 2002;18:E19.
41. Fanelli GC, Tomaszewski DJ. Allograft use in the treatment of the multiple ligament injured knee. Sports Med Arthrosc. 2007;15:139–48.
42. 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;37:1123–30.
43. Feeley BT, Muller MS, Allen AA, Granchi CC, Pearle AD. Isometry of medial collateral ligament reconstruction. Knee Surg Sports Traumatol Arthrosc. 2009;17:1078–82.
44. Mitsou A, Vallianatos P, Piskopakis N, Maheras S. Anterior cruciate ligament reconstruction by over-the-top repair combined with popliteus tendon plasty. J Bone Joint Surg Br. 1990;72:398–404.
45. Moller E, Weidenhielm L, Werner S. Outcome and knee-related quality of life after anterior cruciate ligament reconstruction: a long term follow-up. Knee Surg Sports Traumatol Arthrosc. 2009;17: 786–94.
46. Odensten M, Lysholm J, Gillquist J. Long-term follow-up study of a distal iliotibial band transfer (DIT) for anterolateral knee instability. Clin Orthop Relat Res. 1983;176:129–35.

8 How to Share Guidelines in Daily Practice on Meniscus Repair, Degenerate Meniscal Lesion, and Meniscectomy

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Surgeries involving the meniscus are currently the most common procedures in orthopedic surgery. Meniscectomy is a routine surgery affecting all age groups. Alongside, meniscal preservation has been advocated for a long time since it preserves knee homeostasis and protects the cartilage from the detrimental effects of meniscectomy.

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S. Zaffagnini et al. (eds.), *ESSKA Instructional Course Lecture Book*, DOI 10.1007/978-3-642-53983-1_8, © ESSKA 2014

This concept has been well documented by numerous high-level studies.

The purpose of this Instructional Course Lecture (ICL) is to analyze the gap between the scientific publications and the daily practice and to demonstrate how certain guidelines or recommendations may promote surgeons to preserve the meniscus. Two main chapters will be therefore covered: (1) traumatic meniscal tears where the repair of the meniscus could be an alternative to meniscectomy and (2) degenerative meniscal lesions where conservative treatment would be our first choice of treatment.

8.1 There Is a Need for Setting Guidelines

8.1.1 "Scientific Reports" and "Daily Practice"

We all know about the considerable gap, both in terms of time and also of space, between the scientific publications and the daily practice. As far as finding the "ideal treatment," the purpose of scientific publications is to demonstrate the efficacy, or sometimes the lack of it (!), of a given procedure. Authors who are considered as experts in these fields usually produce these data. But even in the case of a perfectly designed study, the adoption of the described technique is not always guaranteed. It takes a considerable amount of time for a newly described technique to be diffused in the surgical population. This occurs predominately because of the required time for mastering a new procedure, the availability of certain tools and implants, insurance reimbursement policies, etc.

Meniscal surgery is a perfect example of such a gap between what "should be done" and what is "actually done." We have known since many years that preserving the meniscus allows to retain both the mechanical and structural properties and thus to avoid disrupting knee function and cartilage integrity. There are a lot of well-executed studies on both traumatic meniscal tears (meniscus repair versus meniscectomy) and degenerative meniscal lesions (conservative treatment versus meniscectomy). However, the daily practice doesn't still correspond to the conclusions derived from these studies. Meniscectomy still remains the most frequent or one of the most frequent orthopedic procedures worldwide [18]. On the contrary, meniscus repair is not yet widely adopted. Table 8.1 shows the data provided by the French National Healthcare System. It demonstrates a recent yet slow increase of meniscus repair rate which, though, remains low compared to the 20 % theoretical incidence of repairable menisci in stable knees [87].

Table 8.1 Meniscectomy and meniscus repair in stable knees (ACL tears excluded) in France

	Meniscectomy	Repair	%
2009	126,045	4,050	3.20
2012	122,517	14,781	12.06

The probable large abuse of meniscectomy in the field of degenerative meniscal lesions should equally produce the same skepticism. The latter is, actually, very difficult to evaluate with precision, because the alternative treatment option is not the easy-to-trace "meniscal repair" but the "conservative treatment," which is far more difficult to record. The incidence of this "benign neglect" of degenerative meniscal tears is challenging to evaluate in the daily practice. The prevalence of self-prescription or self-treatment among patients, the treatment by the non-orthopedic surgeon (general practitioners, sports medicine physicians, physiotherapists, etc.), is truly unknown. What we know for sure is that conservative treatment should be the first choice in degenerative meniscal lesions.

8.1.2 Design of Guidelines

The cornerstone for setting guidelines among physicians is the conclusion from high-level scientific articles (e.g., randomized controlled trials, RCT). In fact, we need such studies to strongly establish guidelines, but these studies per se cannot be considered as guidelines or recommendations [60]. Personal studies of any expert cannot be considered as guidelines or recommendations. Guidelines are the "official" text published by a public health authority [7], or by a scientific society [13, 17, 58], or by a national healthcare insurance system. Guidelines require setting a specific design that is usually dependent on different healthcare policies and ethical committees' rules among countries. Two aspects must be pointed out: first, guidelines and recommendations usually cover a large field of the pathology, while an RCT or high-level study is usually focused on a specific point. The goal of guidelines is to assist the surgeon in each aspect of his/her practice. Second, guidelines and recommendations must be approved by the users. This means that their users must be included in the decision-making process. The French ACL and Meniscal Treatment Guidelines [7] could be taken as an example. The Haute Autorité de Santé (HAS; High Health Authority) intended to establish guidelines in this field; two surgeons (P Beaufils et C Hulet) were asked:

- To build questionnaires in order to cover the existing ACL and meniscal treatments.
- To review the literature (N Pujol and G Nourissat). More than 1,500 articles were reviewed.
- To build a group of experts consisting of surgeons, but that also included sports medicine specialists, radiologists, general practitioners, and rheumatologists.

A primary draft was written, based on the literature data. When the literature on a specific point was level 1, the recommendation was regarded as grade A, level 2 – grade B, and so on. But sometimes there were no high-level publications while the asked question was important in terms of daily practice. Recommendations coming from level 3 or 4 publications were regarded as grade C, and experts opinions were considered as grade D. Scientifically speaking, grade C or grade D recommendations may have of course a lower strength, but in terms of daily practice, they can help the surgeon in the decision-making process. Then the draft was submitted to 50 independent reviewers coming from different specialties. Comments were analyzed and were included, if necessary, in the final text which was approved by the ad hoc HAS group. Then 3 different articles were published: the whole text, including the methodology, and the detailed reviewed literature, another more practical second short text with algorithms, and last a summary. All these papers, and mainly the short text, can be reprinted in scientific journals.

8.1.3 How to Share Guidelines on Meniscus Pathology and Treatment

Regarding the pathology of the meniscus, guidelines of treatment must be distinguished according to the etiology: traumatic and degenerative meniscal lesions. In the setting of a traumatic lesion, the goal here is to evaluate the particular indications for meniscectomy and for meniscus repair. In the case of degenerative meniscal lesions, the goal is to promote to surgeons the conservative treatment instead of meniscectomy, whenever this is applicable. The following sections will cover these two conditions.

8.2 What Is a Traumatic Meniscal Lesion?

Menisci can be traumatically torn during sports activities or high-energy trauma, possibly combined with fractures around the knee [82]. Meniscal injuries are common in young and active individuals, particularly those who are involved in contact level 1 sports that involve frequent pivoting, such as soccer, rugby, or American football [74]. Conversely, they can also be injured by apparently innocuous activities such as walking or squatting [4]. The traumatic action is most often a twisting movement at the knee while the leg is bent. Acute tears can lead to pain and/or swelling of the knee joint. Particularly acute injuries might originate displaced tears which more often cause mechanical symptoms such as clicking, catching, or locking during motion of the knee joint [74].

An injury mechanism of valgus impact with external rotation of the tibia can also lead to a triad of injuries involving meniscal damage with associated medial collateral and anterior cruciate ligament disruption [19, 89]. Epidemiological studies have shown that all meniscal lesions, in different sports, involve the medial meniscus in 24 % of cases, while lateral meniscus is implicated in around 8 % and 20–30 % of meniscal lesions are associated with other ligament injuries [34].

The patient's chief complaints are usually knee pain and swelling. These are worse when the knee bears higher loads (e.g., when running). Another typical complaint is joint locking, with patients referring that are unable to straighten the leg completely. This can be accompanied by a sense of "clicking." Some patients also refer an impression of giving away [34].

The patient can often remember a specific trauma, activity, or movement during which the meniscus tear was sustained.

Diagnosis is based on clinical examination. MRI is useful but should be reserved for cases in which an experienced clinician necessitates further information before reaching a diagnosis [84]. Current indications for arthroscopy include mainly therapeutic actions.

Meniscus tears can be classified in various ways: by anatomic location, by proximity to blood supply, etc. Various tear patterns and configurations have been described [9]. These include:

- Radial tears
- Flap or parrot beak tears
- Peripheral, longitudinal tears
- Bucket-handle tears
- Horizontal cleavage tears
- Complex, degenerative tears

The functional importance of these classifications, however, is to ultimately determine whether a meniscus is repairable.

The possibility to repair a meniscal injury is dependent upon a number of factors [9]. These include:

- Age/strength
- Activity level
- Tear pattern
- Chronicity of the tear
- Associated injuries (anterior cruciate ligament injury)
- Healing potential

More recently, the ISAKOS classification of meniscal tears has been developed for pooling of data from international clinical trials designed to evaluate the outcomes of treatment. The method has shown sufficient interobserver reliability [2].

The literature indicates a number of risk factors leading to either degenerative or acute meniscal tears, with some of these factors being potentially modifiable [94].

There is moderate evidence that weight bearing during trauma is an important risk factor for acute meniscal tears [33]. Overall, sport seems to be a relevant risk factor for acute meniscal tears [4, 5, 94]. Some contact sports such as soccer or rugby have been correlated with increased risk. However, swimming has also been identified as a risk factor for acute tears despite its low-contact profile [5] and minimal evidence for running also as a risk factor [94]. Generic joint laxity is a risk factor for meniscal tears, which despite not being modifiable might be subject of pre-participation prevention program [4].

An overall *odds ratio* of 3.50 for medial meniscal tears is observed when anterior cruciate ligament (ACL) surgery is performed more than 12 months after the ACL injury compared to less than 12 months after ACL injury (Fig. 8.1) [94]. Concerning lateral meniscus tears, minimal to no evidence was found for the amount of time between ACL injury and reconstruction surgery as a risk factor [94]. These findings should be understood considering the different roles of medial and lateral menisci within knee joint, particularly the role of medial meniscus as secondary restrictor of anterior tibial displacement and the relatively higher mobility of lateral meniscus [59]. Moreover, a delay in surgical treatment is also associated with a higher incidence of medial meniscal tears in pediatric and adolescent populations [62]. Pediatric patients treated >150 days after injury for ACL tears have a higher rate of MMT than those treated ≤150 days after injury. Increased age and weight are independently associated with a higher rate of MMT [21].

Symptomatic horizontal meniscal tears in young patients are a singular entity which often present isolated severe meniscus injuries. It has

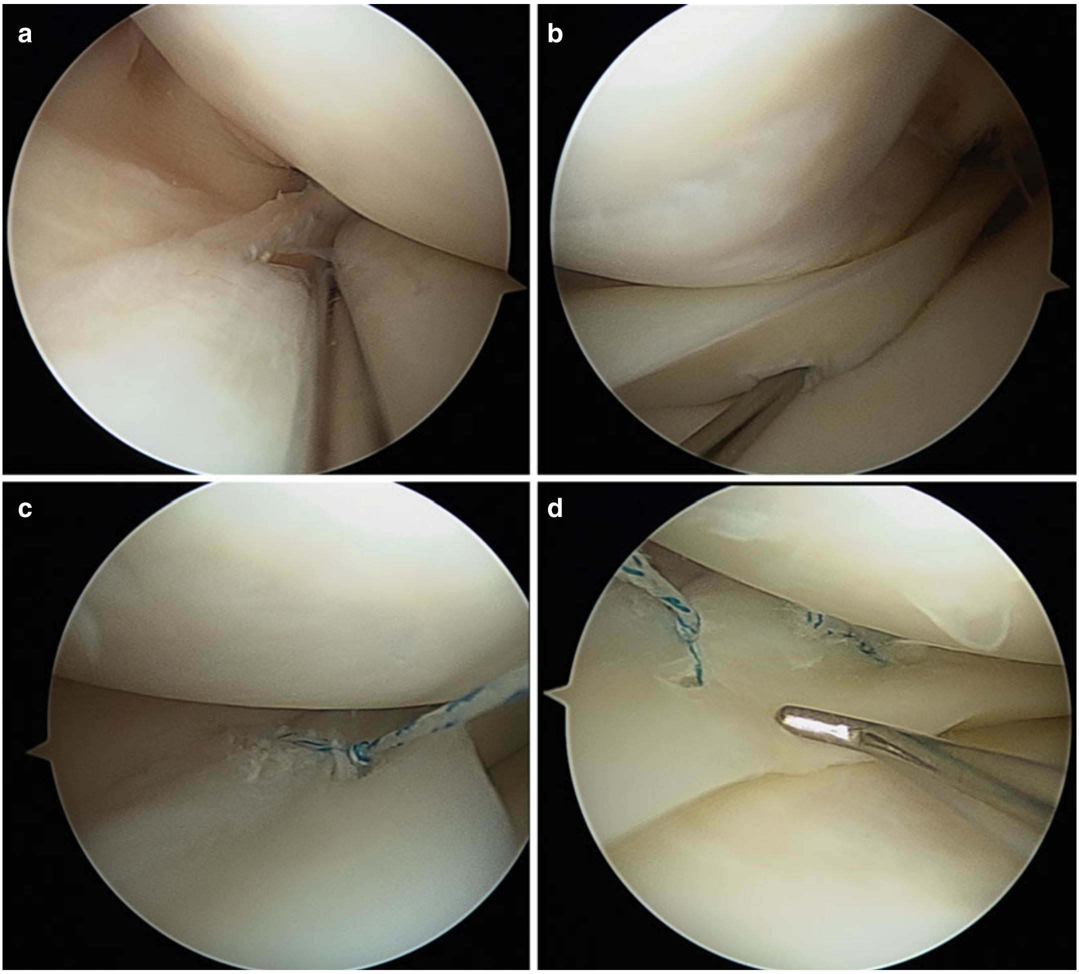

Fig. 8.1 (**a**, **b**) Traumatic vertical lesion of the posterior segment of the medial meniscus in the red–red zone. (**c**, **d**) Treatment by meniscus repair

been considered as an overuse syndrome [78]. Repair of complete radial meniscal tears is a key to restoring the mechanical integrity necessary to maintain hoop tension in the meniscus [14], but the biomechanical structural properties of different repair techniques for complete radial meniscal tears remain unknown. Meniscal root tears (MRTs) may be traumatic or degenerative. Traumatic root tears are often associated with ACL tear, especially on the lateral meniscus. The disruption of collagen fibers, which provide hoop strength, eventually results in extrusion of the menisci, altering their biomechanical properties. Located into the vascularized zone of the meniscus, management is preferentially arthroscopic, aimed at repairing the lesions with arthroscopic transosseous sutures or suture anchors [66]. Considering severe trauma implicated in acute tibial plateau fractures, joint-line depression is a potential predictor of specific meniscal (and ligamentous) injuries [95].

8.3 Meniscus Repair: Guidelines

When an orthopedic surgeon is faced with a meniscal lesion that is assumed to be responsible for the patient's symptoms, two fundamental questions need to be answered: (1) Is it necessary to treat this lesion surgically? Refraining from operative treatment must be seriously considered. (2) If there is a need for surgical treatment, should meniscectomy or meniscal repair be performed? The purpose of meniscus repair is to enhance functional outcomes and protect cartilage, with an acceptable rate of failure [6, 56, 64, 68, 75, 77, 78, 79, 90].

The most important guideline in the decision-making process is the principle of meniscal sparing. Therefore, meniscectomy should only be contemplated when neither of the above options is applicable.

8.3.1 Longitudinal Vertical Lesion in a Stable Knee

Surgical removal of the torn fragment is most commonly performed because in the vast majority of cases, the tear is located in the avascular zone of the meniscus. Patients usually recover rapidly and uneventfully.

The long-term prognosis is favorable, provided that the meniscus has not been totally removed (which would mean excision of meniscal tissue as far as the peripheral zone) and that the resection has not been extended too far anteriorly or posteriorly. As a rule, asymptomatic lesions should be left alone. Meniscal repair should always be considered when the anatomical conditions are favorable (lesion located within the red–red or red–white zone), when the time from injury is less than 3 months, especially if the patient is young and also if the patient's morphotype is disadvantageous (varus knee for medial meniscectomy and valgus knee for lateral meniscectomy) [7, 8, 22]. Particular attention must be paid to the possible detrimental effect of lateral meniscectomy on the affected joint as secondary cartilage degeneration is common, not to mention rapid chondrolysis in a young and active patient. Indications for repair should, therefore, be widened for the lateral meniscus (hypermobile meniscus, true traumatic lesion). It is in these cases that the techniques of stimulating healing (fibrin clot, abrasion, synovial or membrane flap) are most applicable, particularly for long-standing, more extensive lesions and red–white lesions.

8.3.2 Traumatic Vertical Lesion in ACL-Deficient Knee

Every effort should be made to avoid subsequent meniscectomy, which is known to compromise functional performance, joint stability, and cartilage, whether it is associated with ACL reconstruction or not. Let the meniscus alone or surgical repair be considered as the best solution, since these lesions are most often located in the peripheral vascularized zone of the meniscus and have the best chance to heal. These lesions fall into one of the following categories:

Symptomatic anterior laxity of the knee (functional instability) in an active individual practicing sports, in whom ACL reconstruction is strongly indicated. In this situation, the meniscal lesion is diagnosed before or during surgery and is treated simultaneously (Fig. 8.2). The postoperative protocol is not altered, regardless of the treatment of the meniscus, which may involve surgical repair or let the meniscus alone. The ACL surgery is aimed at optimally restoring joint function and protecting the cartilage, mainly attributable to meniscal tissue preservation.

Anterior laxity of the knee associated with minor symptoms in an active individual who is not engaged in high-demand sports activities. In this case, the indications for ACL reconstruction are not straightforward considering the functional limitation of the patient. A diagnosis of a reparable meniscal lesion may be an important argument in favor of surgery. The goal of ACL reconstruction then is to protect the articular cartilage and to improve the natural history of the knee joint. A simple meniscectomy without ACL reconstruction can only be considered in case of a symptomatic meniscal lesion in a sedentary middle-aged patient who does not present functional instability.

Meniscal repair or leave the meniscal tear alone without treatment.

A commonly shared opinion is that unstable or symptomatic meniscal tears should be surgically repaired at the time of ACL reconstruction,

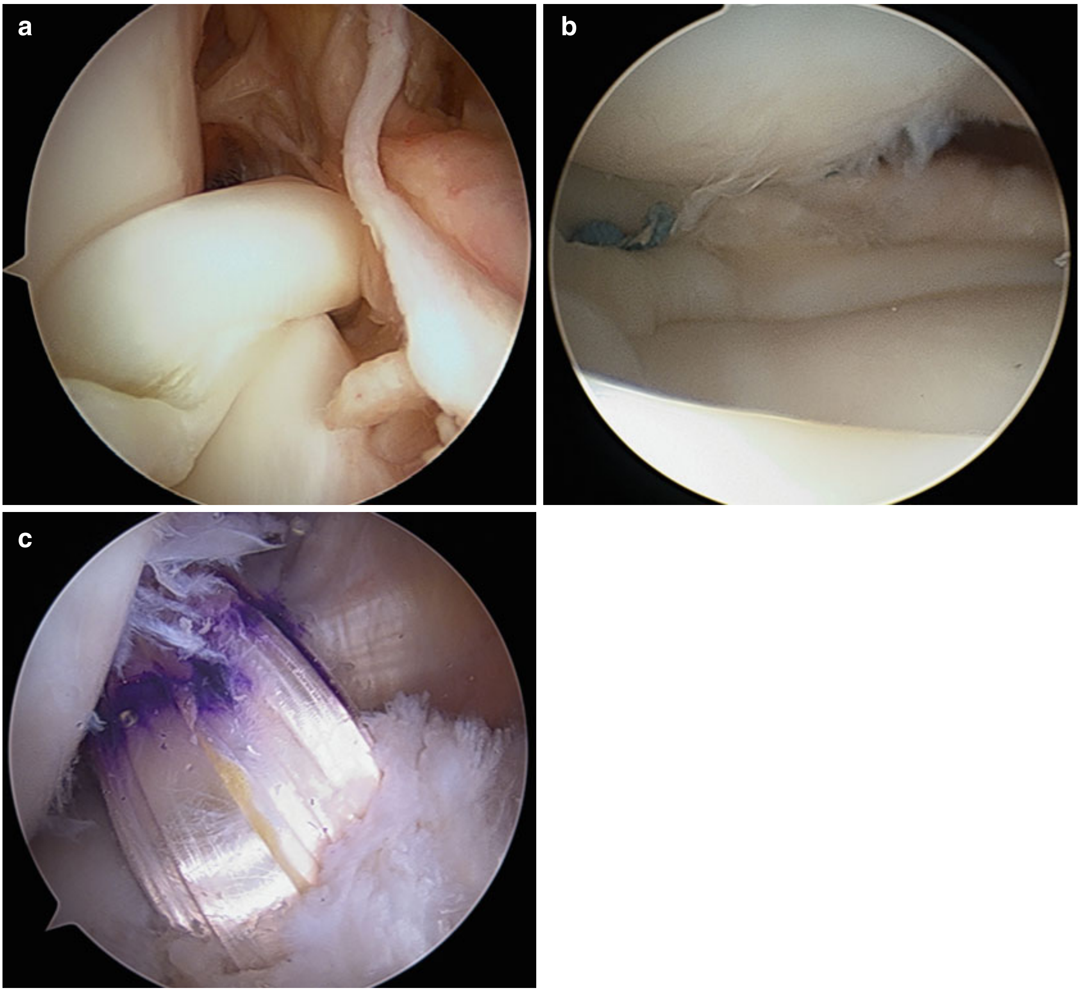

Fig. 8.2 (**a**) Bucket handle of the medial meniscus associated with ACL tear. (**b**) Meniscus repair and (**c**) ACL reconstruction

while stable asymptomatic tears should be left untreated. However, lesion instability has not been clearly defined, and the problem of establishing proper criteria (e.g., size of lesion and abnormal mobility of the meniscus) remains unsolved. At most, Pujol and Beaufils [76] can assume that the indications for surgical repair can be widened for the medial meniscus (increased risk of secondary meniscectomy if left alone), even for small stable lesions whereas for the lateral meniscus let the meniscus alone can be the preferred approach (low risk of subsequent meniscectomy), again for such small stable lesions.

8.3.3 Horizontal Cleavage of the Meniscus in the Young Athlete

It may be considered as an overuse lesion and should be differentiated from the well-known degenerate lesion in older patients. It appears as an intra-meniscal (grade 2) or extra-meniscal (grade 3) horizontal cleavage sometimes associated with a peripheral meniscal cyst [12]. Rather than an arthroscopic repair, Pujol et al. [78, 79] perform an open technique (Fig. 8.3) which allows the debridement of the intra-meniscal lesion close to the horizontal cleavage, and they insert vertical strong bioabsorbable stitches. In a

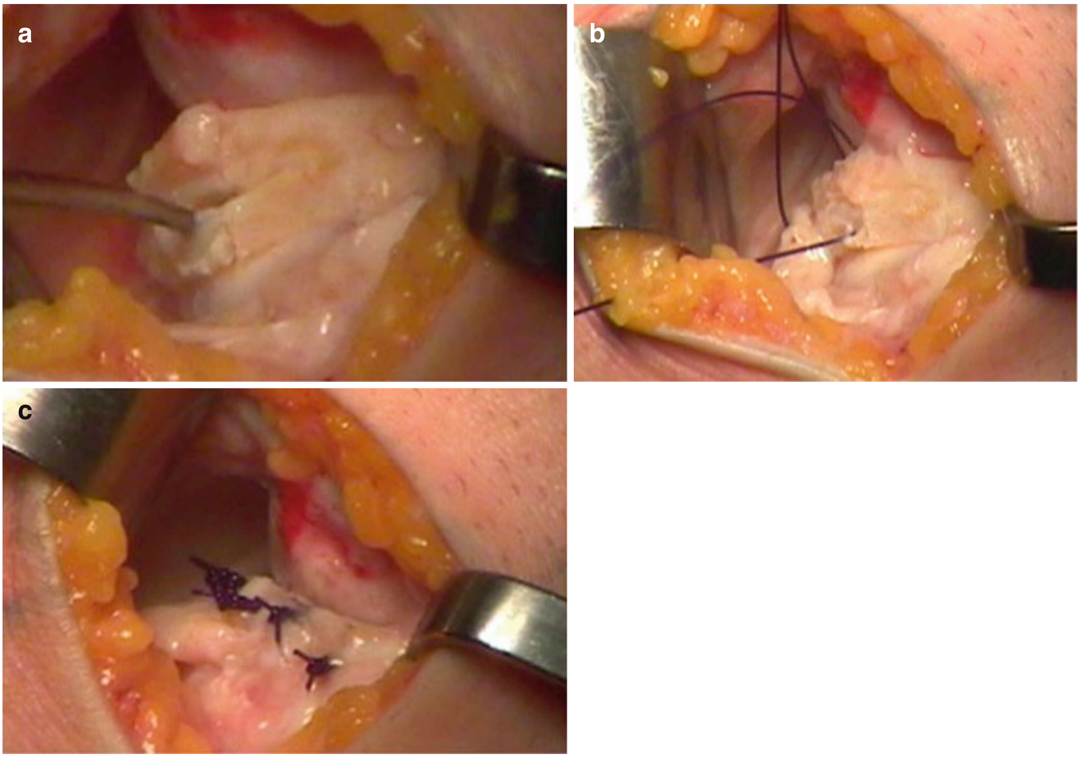

Fig. 8.3 Open meniscus repair of the medial meniscus (post segment) in young athletes. (**a**) After posterior arthrotomy and meniscosynovial junction release, the horizontal cleavage is clearly visible on the peripheral wall of the meniscus. (**b**) vertical bioabsorbable stitches (PDS). (**c**) After closure of the capsule on the meniscus. Knots are intra-articular

review of 30 knees with 40 months FU, Pujol et al. [78, 79] demonstrate the meniscus is preserved in 80 %; functional results deteriorate in patients older than 30. The rate of secondary meniscectomy is low (12 %).

8.3.4 Radial Tears

Radial tears in young patients could be considered for repair when extended to the periphery, specially on the lateral meniscus. It could be done with horizontal hybrid suture devices or with a suture hook and a knot pusher.

8.3.5 Meniscal Root Tears

Root tears have been recently described. Frequent degenerate root tears must be differentiated from true traumatic root tears which are rare. These traumatic root tears are often associated with an ACL tear especially on the posterior horn of the lateral meniscus. They have been ignored for a long time and should be systematically assessed during an ACL reconstruction. They can be treated by tibial refixation, using a transtibial tunnel [1].

8.3.6 Far Posterior Meniscal Tears

These lesions can be suspected by MRI and anterior arthroscopy during an anterior cruciate ligament reconstruction. But it is often misdiagnosed. These lesions are located in the meniscocapsular junction and can be diagnosed by a posterior arthroscopic approach. It is now recommended to repair such meniscal tears in order to support additional stability after ACL reconstruction.

There are several methods of repairing the meniscus: inside-out, outside-in, and all-inside. For far posterior medial meniscal tears, vertical sutures are placed through a posteromedial portal with a suture hook.

8.3.7 In Summary

According to these rules, we can assume the rate of meniscectomies should decrease, and the rate of repair or conservative treatment should increase in the future. Techniques evolved, and indications are more and more accurate; this contributes to current good clinical outcomes of meniscal repairs. Several options to enhance meniscus repair are currently under intense investigation [70] based in growing knowledge related to meniscus tissue [69]. Enhancement of suture techniques includes the use of hydrogels, scaffolds, growth factors, cells, and nanotechnology in an attempt to control the release or activity of the former [69–72].

8.4 Etiology of Degenerative Meniscus Tear and Its Consequences

8.4.1 Epidemiology and Etiology of Degenerative Meniscal Lesions

About every third knee of middle-aged and elderly persons has a meniscus with a degenerative meniscal tear [25] (Fig. 8.4). Typical configurations of these are horizontal cleavages and/or flap tears/oblique tears involving medial meniscal body and or the posterior horn. Most meniscal tears exist in persons without knee symptoms. Hence, meniscal tears are an extremely common incidental finding on magnetic resonance imaging of the knee [25].

Risk factors associated with the development of such degenerative lesions besides self-reported knee injury are malalignment of the knee (the more loaded compartment) and the presence of signs of hand osteoarthritis suggesting systemic or potentially a common environmental factor [29]. Further, in cross-sectional studies, floor layers have been found to have a higher prevalence than graphic designers suggesting occupational load also may contribute [83].

When a meniscus is damaged, degenerative processes and the risk of knee osteoarthritis dramatically increase, probably due to loss of meniscal function in load distribution and shock absorption. In middle-aged and elderly patients, the meniscal tear may often be considered a signifying feature of incipient osteoarthritis.

The prevalence of meniscal tears increased with increasing age, ranging from 16 % in the knees in 50- to 59-year-old women to over 50 % in the knees of men aged 70–90 years [25]. In addition, in this population-based sample, 10 % had partial destruction or complete absence of normal meniscal tissue, which is not classified as meniscal tear but is a finding typically associated with radiographic evidence of osteoarthritis. A prevalence of meniscal pathology of about 70–90 % has been reported in knees of patients with symptomatic knee osteoarthritis [11, 24, 50, 54]. These results are important in a couple of aspects. First, the studies demonstrate the very high prevalence of meniscal damage in the general population. Second, most of meniscal tears do not cause knee symptoms as over 60 % of tears were seen in knees of subjects without knee pain, aching, or stiffness [25]. Hence, a meniscal tear is a common incidental finding when performing MR imaging of the knee (Fig. 8.5). These tears may further act as key factors in the early-stage development of knee osteoarthritis, although often not directly linked to knee pain [24]. Having such meniscal damage on MRI is, for example, associated with an almost sixfold increased odds ratio for development of radiographic knee osteoarthritis over 30 months [26].

Another critical aspect of the meniscus in addition to its morphologic integrity is its positioning within the knee joint. Meniscal tears, for example, are often accompanied by varying degree of meniscal extrusion, i.e., radial displacement of the meniscus outside the joint margin [53]. Several investigators have reported of more frequent meniscal extrusion of the

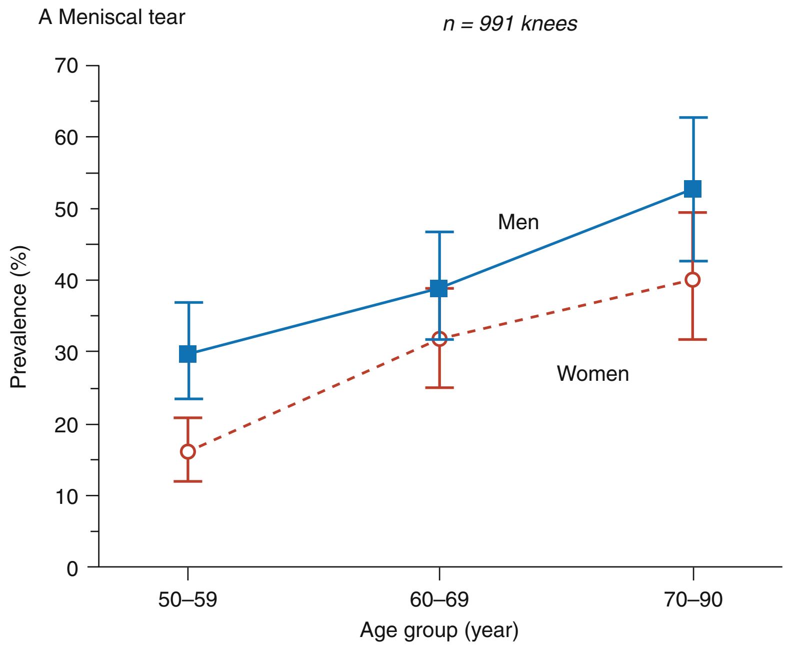

Fig. 8.4 Prevalence of meniscal lesions according to age and gender [25]

Fig. 8.5 Grade 3 hypersignal of the posterior horn (medial meniscus) associated with a parameniscal cyst

meniscal body in the osteoarthritic knee [36, 47, 96]. Also, meniscal extrusion and low coverage of the tibial surface have been reported to be a potent risk factor for cartilage loss [42, 43, 88]. Further, medial meniscal body extrusion is a strong risk factor for the development of bone marrow lesions [28]. Importantly, meniscal extrusion is also a contributing factor to joint space narrowing seen on conventional tibiofemoral radiographs [42–44], making it an integral part of knee osteoarthritis as the disease is often clinically defined by the combination of symptoms and radiographic evidence of knee osteoarthritis.

8.4.2 A Causal Chain of Events to Knee Osteoarthritis

Knee osteoarthritis is often a result of increased biomechanical loading in susceptible individuals and the pathological response of joint tissues to such abnormal biomechanical stress [27]. Knee malalignment, obesity, and occupational hazards could result in chronic overloading. Such overloading, coupled with degenerative meniscal matrix changes possibly related to early-stage osteoarthritis, could lead to meniscal fatigue, rupture, and extrusion [25, 67, 83]. Once the meniscus loses its critical function in the knee joint, the increased biomechanical loading patterns on joint cartilage may result in cartilage loss [10, 42, 43], bone alterations including trabecular bone changes [51, 73, 99], increased bone mineral density [55], development of subchondral bone marrow lesions [24, 26, 27], and increasing malalignment. The vicious cycle of knee osteoarthritis is in motion.

The biomechanical effect of loss of meniscal function is well documented in multiple biomechanical studies [35, 52, 85, 86, 91, 97]. Cartilage loss predominantly occurs in the vicinity of where the meniscal damage is located suggesting a firm cause and effect relationship between the meniscal damage and structural disease progression [15].

8.4.3 Symptoms and Degenerative Meniscal Pathologies

The association between meniscal tears and knee symptoms is complex in knees with osteoarthritis as well as in knees without osteoarthritis [11, 25, 65]. Healthcare professionals also need to be aware of the fact that meniscal tears may also be asymptomatic per se in a patient with knee pain. Just because there is a tear, visible on knee MRI or at arthroscopy, it does not necessarily mean surgical resection of torn meniscal tissue will resolve the patient's pain or aid the patient in the long term [39, 49, 63]. This is particularly true in patients' with preexisting knee osteoarthritis where the pain, for example, may be a result of the compromised meniscal function leading to increased stress on joint cartilage and subchondral bone, which may result in bone marrow lesions [28]. Bone marrow lesions have been found to be highly associated with knee pain and fluctuations in knee pain [32, 101]. Meniscus tears are also often associated with synovitis which may be painful [80]. Recently, increased vascular penetration and nerve growth have also been reported of the menisci obtained from osteoarthritic knees [3]. Although a cross-sectional study indicated meniscal extrusion to be more frequent in painful knees than the contralateral non-painful knee of similar radiographic osteoarthritis stage, it is still largely unknown if meniscal extrusion may be directly associated with pain due to, for example, stretching/irritation of the synovial capsule [98].

8.5 Treatment of Degenerative Meniscus Tear: Current Concepts and Evidence

Arthroscopic partial meniscectomy (APM) is the most common orthopedic procedure [18]. The aim of APM is to relieve symptoms attributed to meniscus lesion by removing torn meniscus fragments and trimming the meniscus back to a stable rim. However, there is a dire lack of evidence on its efficacy [30]. Most treated meniscus lesions are degenerative in nature and associated with various degrees of degenerative knee disease – anything from mild chondral changes not visible in normal x-rays to established knee osteoarthritis (OA) [61]. The number of knee arthroscopies to treat established knee OA, without or with a concomitant meniscus lesion, has decreased dramatically in the past 15 years [41, 48]. This trend has been attributed to the findings of two controlled trials [49, 63] demonstrating lack of efficacy of arthroscopic surgery. However, the number of APMs has concurrently increased by 50 % [48]. At approximately 700,000 per year in the USA alone [18] and costing over $6,000 per case, the estimated annual direct medical costs of these APMs amount to about $4 billion.

8.5.1 Existing Evidence

One randomized, placebo surgery controlled trial in the arthroscopic treatment of degenerative knee disease exists [63]. In this seminal trial, Moseley et al. compared the efficacy of arthroscopic lavage, debridement, and placebo surgery (skin incisions only) in patients with established knee osteoarthritis. The outcomes after arthroscopic lavage or debridement were no better than those after a placebo procedure. However, concerns regarding the methodological choices and patient selection of this trial prompted Kirkley et al. [49] to reassess the role of arthroscopic surgery in patients with knee OA. A comparison of arthroscopic surgery coupled with optimized physical and medical therapy showed no additional benefit to optimized physical and medical therapy alone. In the three previous trials specifically assessing the benefit of

APM in the treatment of degenerative meniscus tear [40, 46, 100], arthroscopic surgery and exercise therapy were not superior to exercise therapy alone in patients with varying degrees of knee OA. The elementary problem regarding the abovementioned randomized controlled trials (RCTs) assessing the outcome of APM in patients with degenerative meniscus tear is that all these studies have been effectiveness trials in design – addressing whether arthroscopic surgery works in ordinary healthcare settings – including typical degenerative knee disease patients with varying degrees of knee OA. Considering the fact that the existing literature actually provides no (efficacy) evidence in support of the current practice of performing APM for patients with a degenerative meniscus tear, a study using a sham surgery controlled design and enrolling a sample with optimal anticipated response to APM – patients with a degenerative medial meniscus tear [16, 38] and no concomitant advanced knee degeneration [31] – are urgently needed. A detailed elaboration of the strengths and limitations of such trial, including ethical and feasibility analysis on the use of the sham surgery design, has been published previously [93], and at least another such trial is currently under way [37].

The scientific validity of the entire clinical scheme for diagnosing and treating patients with a suspected degenerative meniscus tear is under scrutiny. Medial joint line symptoms are currently commonly attributed to meniscus tears. If the clinical diagnosis is "confirmed" in MRI, APM is typically advocated, particularly for patients without concomitant knee OA [57]. However, this may be too simplistic a strategy: Englund et al. [24] showed no association between the presence of meniscal damage and the development of frequent knee pain in middle-aged and older adults. The authors concluded that the association seems to be present because both meniscal damage and knee pain are related to OA and not because of a direct link between the two. This finding, along with other similar evidence, has led numerous authors to propose that degenerative meniscus tear is an early sign of knee OA rather than a separate clinical entity [11, 20, 25, 26]. On a closely related issue, there is mounting evidence that indicates that the occurrence of mechanical symptoms preoperatively predicts poor patient satisfaction and functional outcome and APM/arthroscopic surgery provides poor relief from these symptoms [92].

A counterargument to findings challenging the justification of any medical intervention is that previous studies – albeit often using trial designs with higher risk of bias – have consistently shown the intervention to be beneficial. In this respect, it should be noted that all randomized trials addressing APM showed a marked (and statistically significant) improvement on all outcome measures, but no benefit of APM over conservative treatment has been shown. It should also be noted that beyond the question of symptomatic relief, there is also concern – based on evidence from cohort studies – that arthroscopic partial meniscectomy may actually accelerate, rather than prevent, the progression of OA [23, 81]. However, as the respective effects of surgery (APM) and tear cannot be disentangled in such study designs [45], only the long-term follow-ups of ongoing sham surgery controlled RCTs [37, 93] will eventually resolve this most important issue.

8.5.2 In Summary

In summary, the results of existing high-quality evidence (from randomized, controlled trials) have failed to provide evidence to support the current practice of performing arthroscopic partial medial meniscectomy for patients with a degenerative meniscus tear. Currently millions of people worldwide are annually being exposed to APM without evidence of benefits while there are risks and substantial costs inherent in surgery.

References

1. Ahn JH, Wang JH, Yoo JC. Arthroscopic all-inside suture repair of medial meniscus lesion in anterior cruciate ligament-deficient knees: results of second-look arthroscopies in 39 cases. Arthroscopy. 2004;20:936–45.

2. Anderson AF, Irrgang JJ, Dunn W, Beaufils P, Cohen M, Cole BJ, et al. Interobserver reliability of the International Society of Arthroscopy, Knee Surgery and Orthopaedic Sports Medicine (ISAKOS) classification of meniscal tears. Am J Sports Med. 2011;39:926–32.
3. Ashraf S, Wibberley H, Mapp PI, Hill R, Wilson D, Walsh DA. Increased vascular penetration and nerve growth in the meniscus: a potential source of pain in osteoarthritis. Ann Rheum Dis. 2011;70(3):523–9.
4. Baker P, Coggon D, Reading I, Barrett D, McLaren M, Cooper C. Sports injury, occupational physical activity, joint laxity, and meniscal damage. J Rheumatol. 2002;29:557–63.
5. Baker P, Reading I, Cooper C, Coggon D. Knee disorders in the general population and their relation to occupation. Occup Environ Med. 2003;60:794–7.
6. Beaufils P, Cassard X. Meniscal repair. Rev Chir Orthop Reparatrice Appar Mot. 2004;90:3S49–75.
7. Beaufils P, Hulet C, Dhénain M, Nizard R, Nourissat G, Pujol N. Clinical practice guidelines for the management of meniscal lesions and isolated lesions of the anterior cruciate ligament of the knee in adults. Orthop Traumatol Surg Res. 2009;95:437–42.
8. Beaufils P. Synthesis-indications. In: Beaufils P, Verdonk R, editors. The meniscus. Berlin/New York: Springer; 2010. p. 235–8.
9. Bernstein J. In brief: meniscal tears. Clin Orthop Relat Res. 2010;468:1190–2.
10. Berthiaume MJ, Raynauld JP, Martel-Pelletier J, Labonte F, Beaudoin G, Bloch DA, et al. Meniscal tear and extrusion are strongly associated with progression of symptomatic knee osteoarthritis as assessed by quantitative magnetic resonance imaging. Ann Rheum Dis. 2005;64(4):556–63.
11. Bhattacharyya T, Gale D, Dewire P, Totterman S, Gale ME, McLaughlin S, et al. The clinical importance of meniscal tears demonstrated by magnetic resonance imaging in osteoarthritis of the knee. J Bone Joint Surg Am. 2003;85-A(1):4–9.
12. Biedert RM. Treatment of intrasubstance meniscal lesions: a randomized prospective study of four different methods. Knee Surg Sports Traumatol Arthrosc. 2000;8:104–8.
13. Brown GA. AAOS clinical practice guideline: treatment of osteoarthritis of the knee: evidence-based guideline, 2nd edition. J Am Acad Orthop Surg. 2013;21(9):577–9.
14. Brucker PU, Favre P, Puskas GJ, von Campe A, Meyer DC, Koch PP. Tensile and shear loading stability of all-inside meniscal repairs: an in vitro biomechanical evaluation. Am J Sports Med. 2010;38:1838–44.
15. Chang A, Moisio K, Chmiel JS, Eckstein F, Guermazi A, Almagor O, et al. Subregional effects of meniscal tears on cartilage loss over 2 years in knee osteoarthritis. Ann Rheum Dis. 2011;70(1):74–9.
16. Chatain F, Adeleine P, Chambat P, Neyret P. A comparative study of medial versus lateral arthroscopic partial meniscectomy on stable knees: 10-year minimum follow-up. Arthroscopy. 2003;19(8):842–9.
17. Committee on Ethics and Standards Arthroscopy Association of North America; Board of Directors of the Arthroscopy Association of North America. Suggested guidelines for the practice of arthroscopic surgery. Arthroscopy. 2011;27(9):A30.
18. Cullen KA, Hall MJ, Golosinskiy A. Ambulatory surgery in the United States, 2006. Natl Health Stat Rep. 2009;11:1–25.
19. Dacombe PJ. Shelbourne's update of the O'Donoghue knee triad in a 17-year-old male Rugby player. BMJ Case Rep. 2013;2013.
20. Ding C, Martel-Pelletier J, Pelletier JP, Abram F, Raynauld JP, Cicuttini F, et al. Meniscal tear as an osteoarthritis risk factor in a largely non-osteoarthritic cohort: a cross-sectional study. J Rheumatol. 2007;34(4):776–84.
21. 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.
22. Eggli S, Wegmuller H, Kosina J, Huckell C, Jakob RP. Long-term results of arthroscopic meniscal repair. An analysis of isolated tears. Am J Sports Med. 1995;23:715–20.
23. Englund M, Lohmander LS. Risk factors for symptomatic knee osteoarthritis fifteen to twenty-two years after meniscectomy. Arthritis Rheum. 2004;50(9):2811–9.
24. Englund M, Niu J, Guermazi A, Roemer FW, Hunter DJ, Lynch JA, et al. Effect of meniscal damage on the development of frequent knee pain, aching, or stiffness. Arthritis Rheum. 2007;56(12):4048–54.
25. Englund M, Guermazi A, Gale D, Hunter DJ, Aliabadi P, Clancy M, et al. Incidental meniscal findings on knee MRI in middle-aged and elderly persons. N Engl J Med. 2008;359(11):1108–15.
26. Englund M, Guermazi A, Roemer FW, Aliabadi P, Yang M, Lewis CE, et al. Meniscal tear in knees without surgery and the development of radiographic osteoarthritis among middle-aged and elderly persons: the Multicenter Osteoarthritis Study. Arthritis Rheum. 2009;60(3):831–9.
27. Englund M. The role of biomechanics in the initiation and progression of OA of the knee. Best Pract Res Clin Rheumatol. 2010;24(1):39–46.
28. Englund M, Guermazi A, Roemer FW, Yang M, Zhang Y, Nevitt MC, et al. Meniscal pathology on MRI increases the risk for both incident and enlarging subchondral bone marrow lesions of the knee: the MOST Study. Ann Rheum Dis. 2010;69(10):1796–802.
29. Englund M, Felson DT, Guermazi A, Roemer FW, Wang K, Crema MD, et al. Risk factors for medial meniscal pathology on knee MRI in older US adults: a multicentre prospective cohort study. Ann Rheum Dis. 2011;70(10):1733–9.
30. Englund M, Roemer FW, Hayashi D, Crema MD, Guermazi A. Meniscus pathology, osteoarthritis and the treatment controversy. Nat Rev Rheumatol. 2012;8(7):412–9.

31. Fabricant PD, Rosenberger PH, Jokl P, Ickovics JR. Predictors of short-term recovery differ from those of long-term outcome after arthroscopic partial meniscectomy. Arthroscopy. 2008;24(7):769–78.
32. Felson DT, Chaisson CE, Hill CL, Totterman SM, Gale ME, Skinner KM, et al. The association of bone marrow lesions with pain in knee osteoarthritis. Ann Intern Med. 2001;134(7):541–9.
33. Friden T, Erlandsson T, Zatterstrom R, Lindstrand A, Moritz U. Compression or distraction of the anterior cruciate injured knee. Variations in injury pattern in contact sports and downhill skiing. Knee Surg Sports Traumatol Arthrosc. 1995;3:144–7.
34. Frizziero A, Ferrari R, Giannotti E, Ferroni C, Poli P, Masiero S. The meniscus tear. State of the art of rehabilitation protocols related to surgical procedures. Muscles Ligaments Tendons J. 2012;2:295–301.
35. Fukubayashi T, Kurosawa H. The contact area and pressure distribution pattern of the knee. A study of normal and osteoarthrotic knee joints. Acta Orthop Scand. 1980;51(6):871–9.
36. Gale DR, Chaisson CE, Totterman SM, Schwartz RK, Gale ME, Felson D. Meniscal subluxation: association with osteoarthritis and joint space narrowing. Osteoarthritis Cartilage. 1999;7(6):526–32.
37. Hare KB, Lohmander LS, Christensen R, Roos EM. Arthroscopic partial meniscectomy in middle-aged patients with mild or no knee osteoarthritis: a protocol for a double-blind, randomized sham-controlled multi-centre trial. BMC Musculoskelet Disord. 2013;14(1):71.
38. Hede A, Larsen E, Sandberg H. Partial versus total meniscectomy. A prospective, randomised study with long-term follow-up. J Bone Joint Surg Br. 1992;74(1):118–21.
39. Herrlin S, Hallander M, Wange P, Weidenhielm L, Werner S. Arthroscopic or conservative treatment of degenerative medial meniscal tears: a prospective randomised trial. Knee Surg Sports Traumatol Arthrosc. 2007;15(4):393–401.
40. Herrlin SV, Wange PO, Lapidus G, Hallander M, Werner S, Weidenhielm L. Is arthroscopic surgery beneficial in treating non-traumatic, degenerative medial meniscal tears? A five year follow-up. Knee Surg Sports Traumatol Arthrosc. 2013;21(2):358–64.
41. Holmes R, Moschetti W, Martin B, Tomek I, Finlayson S. Effect of evidence and changes in reimbursement on the rate of arthroscopy for osteoarthritis. Am J Sports Med. 2013;41:1039–43.
42. Hunter DJ, Zhang YQ, Niu JB, Tu X, Amin S, Clancy M, et al. The association of meniscal pathologic changes with cartilage loss in symptomatic knee osteoarthritis. Arthritis Rheum. 2006;54(3):795–801.
43. Hunter DJ, Zhang YQ, Tu X, Lavalley M, Niu JB, Amin S, et al. Change in joint space width: hyaline articular cartilage loss or alteration in meniscus? Arthritis Rheum. 2006;54(8):2488–95.
44. Hunter DJ, Buck R, Vignon E, Eckstein F, Brandt K, Mazzuca SA, et al. Relation of regional articular cartilage morphometry and meniscal position by MRI to joint space width in knee radiographs. Osteoarthritis Cartilage. 2009;17(9):1170–6.
45. Katz JN, Martin SD. Meniscus – friend or foe: epidemiologic observations and surgical implications. Arthritis Rheum. 2009;60(3):633–5.
46. Katz JN, Brophy RH, Chaisson CE, de Chaves L, Cole BJ, Dahm DL, et al. Surgery versus physical therapy for a meniscal tear and osteoarthritis. N Engl J Med. 2013;368:1675–84.
47. Kenny C. Radial displacement of the medial meniscus and Fairbank's signs. Clin Orthop Relat Res. 1997;339:163–73.
48. Kim S, Bosque J, Meehan JP, Jamali A, Marder R. Increase in outpatient knee arthroscopy in the United States: a comparison of National Surveys of Ambulatory Surgery, 1996 and 2006. J Bone Joint Surg Am. 2011;93:994–1000.
49. Kirkley A, Birmingham TB, Litchfield RB, Giffin JR, Willits KR, Wong CJ, et al. A randomized trial of arthroscopic surgery for osteoarthritis of the knee. N Engl J Med. 2008;359:1097–107.
50. Kornaat PR, Bloem JL, Ceulemans RY, Riyazi N, Rosendaal FR, Nelissen RG, et al. Osteoarthritis of the knee: association between clinical features and MR imaging findings. Radiology. 2006;239(3):811–7.
51. Kraus VB, Feng S, Wang S, White S, Ainslie M, Brett A, et al. Trabecular morphometry by fractal signature analysis is a novel marker of osteoarthritis progression. Arthritis Rheum. 2009;60(12):3711–22.
52. Kurosawa H, Fukubayashi T, Nakajima H. Load-bearing mode of the knee joint: physical behavior of the knee joint with or without menisci. Clin Orthop. 1980;149:283–90.
53. Lee DH, Lee BS, Kim JM, Yang KS, Cha EJ, Park JH, et al. Predictors of degenerative medial meniscus extrusion: radial component and knee osteoarthritis. Knee Surg Sports Traumatol Arthrosc. 2011;19(2):222–9.
54. Link TM, Steinbach LS, Ghosh S, Ries M, Lu Y, Lane N, et al. Osteoarthritis: MR imaging findings in different stages of disease and correlation with clinical findings. Radiology. 2003;226(2):373–81.
55. Lo GH, Niu J, McLennan CE, Kiel DP, McLean RR, Guermazi A, et al. Meniscal damage associated with increased local subchondral bone mineral density: a Framingham study. Osteoarthritis Cartilage. 2008; 16(2):261–7.
56. Lozano J, Ma CB, Cannon WD. All-inside meniscus repair: a systematic review. Clin Orthop Relat Res. 2007;455:134–41.
57. Lyman S, Oh LS, Reinhardt KR, Mandl LA, Katz JN, Levy BA, et al. Surgical decision making for arthroscopic partial meniscectomy in patients aged over 40 years. Arthroscopy. 2012;28(4):492–501.e1.
58. Mayr H. Recommendations of the AGA (German-speaking Arthroscopy Association) on equipment of facilities, process quality and qualification of surgeons for arthroscopic interventions. Unfallchirurg. 2010;113(11):960–3.

59. McDermott ID, Masouros SD, Amis AA. Biomechanics of the menisci of the knee. Curr Orthop. 2008;22:193–201.
60. McIntyre LF, Beach WR, Higgins LD, Mordin MM, Mauskopf J, Sweeney CT, Copley-Merriman C. Evidence-based medicine, appropriate-use criteria, and sports medicine: how best to develop meaningful treatment guidelines. Arthroscopy. 2013;29(7):1224–9. doi:10.1016/j.arthro.2013.01.018.
61. Metcalf MH, Barrett GR. Prospective evaluation of 1485 meniscal tear patterns in patients with stable knees. Am J Sports Med. 2004;32(3):675–80.
62. 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.
63. Moseley JB, O'Malley K, Petersen NJ, Menke TJ, Brody BA, Kuykendall DH, et al. A controlled trial of arthroscopic surgery for osteoarthritis of the knee. N Engl J Med. 2002;347(2):81–8.
64. Nepple JJ, Dunn WR, Wright RW. Meniscal repair outcomes at greater than five years: a systematic literature review and meta-analysis. J Bone Joint Surg Am. 2012;94:2222–7.
65. Noble J, Erat K. In defence of the meniscus. A prospective study of 200 meniscectomy patients. J Bone Joint Surg Br. 1980;62-B(1):7–11.
66. Papalia R, Vasta S, Franceschi F, D'Adamio S, Maffulli N, Denaro V. Meniscal root tears: from basic science to ultimate surgery. Br Med Bull. 2013;106:91–115.
67. Pauli C, Grogan SP, Patil S, Otsuki S, Hasegawa A, Koziol J, et al. Macroscopic and histopathologic analysis of human knee menisci in aging and osteoarthritis. Osteoarthritis Cartilage. 2011;19(9):1132–41.
68. Paxton ES, Stock MV, Brophy RH. Meniscal repair versus partial meniscectomy: a systematic review comparing reoperation rates and clinical outcomes. Arthroscopy. 2011;27:1275–88.
69. Pereira H, Frias AM, Silva-Correia J, Oliveira JM, Espregueira-Mendes J, Reis RL. Human Meniscus segmental characterization: building the basis for tissue engineering. Knee Surg, Sports Traumatol, Arthroscop. 2012;20(suppl 1): S3.
70. Pereira H, Frias AM, Oliveira JM, Espregueira-Mendes J, Reis RL. Tissue engineering and regenerative medicine strategies in meniscus lesions. Arthroscopy. 2011;27:1706–19.
71. Pereira H, Silva-Correia J, Oliveira JM, Reis RL, Espregueira-Mendes J. Future trends in the treatment of meniscus lesions: from repair to regeneration. In: Verdonk R, Espregueira-Mendes J, Monllau JC, editors. Meniscal transplantation. Heidelberg/New York/Dordrecht/London: Springer; 2013. p. 103–14.
72. Pereira H, Silva-Correia J, Oliveira JM, Reis RL, Espregueira-Mendes J. The meniscus: basic science. In: Verdonk R, Espregueira-Mendes J, Monllau JC, editors. Meniscal transplantation. Heidelberg/New York/Dordrecht/London: Springer; 2013. p. 7–14.
73. Podsiadlo P, Dahl L, Englund M, Lohmander LS, Stachowiak GW. Differences in trabecular bone texture between knees with and without radiographic osteoarthritis detected by fractal methods. Osteoarthritis Cartilage. 2008;16(3):323–9.
74. Poulsen MR, Johnson DL. Meniscal injuries in the young, athletically active patient. Phys Sportsmed. 2011;39:123–30.
75. Pujol N, Panarella L, Ait Si Selmi T, Neyret P, Fithian D, Beaufils P. Meniscal healing after meniscus repair: a CT arthrography assessment. Am J Sports Med. 2008;36:1489–95.
76. Pujol N, Beaufils P. Healing results of meniscal tears left in situ during anterior cruciate ligament reconstruction: a review of clinical studies. Knee Surg Sports Traumatol Arthrosc. 2009;17:396–401.
77. Pujol N. Meniscus repair. In: Beaufils P, Verdonk R, editors. The meniscus. Berlin/New York: Springer; 2010. p. 229–34.
78. Pujol N, Bohu Y, Boisrenoult P, Makdes A, Beaufils P. Clinical outcomes of open meniscal repair of horizontal meniscal tears in young patients. Knee Surg Sports Traumatol Arthrosc. 2013;21(7):1530–3.
79. Pujol N, Tardy N, Boisrenoult P, Beaufils P. Long-term outcomes of all-inside meniscal repair. Knee Surg Sports Traumatol Arthrosc. 2013 [Epub ahead of print].
80. Roemer FW, Guermazi A, Hunter DJ, Niu J, Zhang Y, Englund M, et al. The association of meniscal damage with joint effusion in persons without radiographic osteoarthritis: the Framingham and MOST osteoarthritis studies. Osteoarthritis Cartilage. 2009;17(6):748–53.
81. Roos EM, Ostenberg A, Roos H, Ekdahl C, Lohmander LS. Long-term outcome of meniscectomy: symptoms, function, and performance tests in patients with or without radiographic osteoarthritis compared to matched controls. Osteoarthritis Cartilage. 2001;9(4):316–24.
82. Ruiz-Iban MA, Diaz-Heredia J, Elias-Martin E, Moros-Marco S, Cebreiro Martinez Del Val I. Repair of meniscal tears associated with tibial plateau fractures: a review of 15 cases. Am J Sports Med. 2012;40:2289–95.
83. Rytter S, Jensen LK, Bonde JP, Jurik AG, Egund N. Occupational kneeling and meniscal tears: a magnetic resonance imaging study in floor layers. J Rheumatol. 2009;36(7):1512–9.
84. Ryzewicz M, Peterson B, Siparsky PN, Bartz RL. The diagnosis of meniscus tears: the role of MRI and clinical examination. Clin Orthop Relat Res. 2007;455:123–33.
85. Seedhom BB, Dowson D, Wright V. Proceedings: functions of the menisci. A preliminary study. Ann Rheum Dis. 1974;33(1):111.
86. Seedhom BB, Hargreaves DJ. Transmission of the load in the knee joint with special reference to the role of the meniscus. Part I+II. Eng Med. 1979;4: 207–28.

87. Servien E, Acquitter Y, Hulet C, Seil R, French Arthroscopy Society. Lateral meniscus lesions on stable knee: a prospective multicenter study. Orthop Traumatol Surg Res. 2009;95(8 Suppl 1):S60–4.
88. Sharma L, Eckstein F, Song J, Guermazi A, Prasad P, Kapoor D, et al. Relationship of meniscal damage, meniscal extrusion, malalignment, and joint laxity to subsequent cartilage loss in osteoarthritic knees. Arthritis Rheum. 2008;58(6):1716–26.
89. Shelbourne KD, Nitz PA. The O'Donoghue triad revisited. Combined knee injuries involving anterior cruciate and medial collateral ligament tears. Am J Sports Med. 1991;19:474–7.
90. Shelbourne KD, Dersam MD. Comparison of partial meniscectomy versus meniscus repair for bucket-handle lateral meniscus tears in anterior cruciate ligament reconstructed knees. Arthroscopy. 2004;20:581–5.
91. Shrive NG, O'Connor JJ, Goodfellow JW. Load-bearing in the knee joint. Clin Orthop. 1978; 1978(131):279–87.
92. Sihvonen R, Järvinen T. Mechanical symptoms as an indication for knee arthroscopy in patients with degenerative meniscus tear. American Academy of Orthopaedic Surgeons Annual Meeting 2014, New Orleans, LA, USA [abstract].
93. Sihvonen R, Paavola M, Malmivaara A, Jarvinen TL. Finnish Degenerative Meniscal Lesion Study (FIDELITY): a protocol for a randomised, placebo surgery controlled trial on the efficacy of arthroscopic partial meniscectomy for patients with degenerative meniscus injury with a novel 'RCT within-a-cohort' study design. BMJ Open. 2013;3(3):e002510.
94. Snoeker BA, Bakker EW, Kegel CA, Lucas C. Risk factors for meniscal tears: a systematic review including meta-analysis. J Orthop Sports Phys Ther. 2013;43:352–67.
95. Spiro AS, Regier M, Novo de Oliveira A, Vettorazzi E, Hoffmann M, Petersen JP, et al. The degree of articular depression as a predictor of soft-tissue injuries in tibial plateau fracture. Knee Surg Sports Traumatol Arthrosc. 2013;21:564–70.
96. Sugita T, Kawamata T, Ohnuma M, Yoshizumi Y, Sato K. Radial displacement of the medial meniscus in varus osteoarthritis of the knee. Clin Orthop Relat Res. 2001;387:171–7.
97. Walker PS, Erkman MJ. The role of the menisci in force transmission across the knee. Clin Orthop. 1975;109:184–92.
98. Wenger A, Englund M, Wirth W, Hudelmaier M, Kwoh K, Eckstein F. Relationship of 3D meniscal morphology and position with knee pain in subjects with knee osteoarthritis: a pilot study. Eur Radiol. 2012;22:211–20.
99. Wolski M, Podsiadlo P, Stachowiak GW, Lohmander LS, Englund M. Differences in trabecular bone texture between knees with and without radiographic osteoarthritis detected by directional fractal signature method. Osteoarthritis Cartilage. 2010;18(5): 684–90.
100. Yim JH, Seon JK, Song EK, Choi JI, Kim MC, Lee KB, et al. A comparative study of meniscectomy and nonoperative treatment for degenerative horizontal tears of the medial meniscus. Am J Sports Med. 2013;41(7):1565–70.
101. Zhang Y, Nevitt M, Niu J, Lewis C, Torner J, Guermazi A, et al. Fluctuation of knee pain and changes in bone marrow lesions, effusions, and synovitis on magnetic resonance imaging. Arthritis Rheum. 2011;63(3):691–9.

The Medial Patellofemoral Ligament

9

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S. Zaffagnini et al. (eds.), *ESSKA Instructional Course Lecture Book*,
DOI 10.1007/978-3-642-53983-1_9, © ESSKA 2014

9.1 Introduction

The medial patellofemoral complex, consisting of the medial patellofemoral ligament (MPFL) and the medial patellotibial ligament, is the main passive stabiliser of the patellofemoral joint. Since it has been shown that rupture of the MPFL is the main pathological consequence of patellar dislocation and biomechanical studies have demonstrated that the MPFL is the main restraint against lateral patellar displacement, reconstruction of the MPFL has become a widespread technique for restoration of patellofemoral stability.

Different authors have reported on the results of different surgical techniques. The techniques and outcomes vary widely. This ICL aims to provide an overview of the indication for isolated MPFL reconstruction, advantages, and disadvantages of surgical technique and reasons for failure.

9.2 State-of-the-Art Treatment

9.2.1 Biomechanical Considerations: The Normal Anatomy and Biomechanics of the MPFL and of MPFL Reconstruction

Joanna Stephen, Punyawan Lumpaopong, David Deehan, Deiary Kader, and Andrew Amis

The most important anatomical factor is the femoral attachment, which was shown to be midway between the medal epicondyle and the adductor tubercle. If the attachment is too proximal, then it leads to elongation when the knee flexes, and a too distal attachment leads to elongation when the knee extends. In a normal knee, the femoral attachment of the MPFL can be defined by X-ray: if the anterior–posterior size of the femoral condyle is 100 %, then the MPFL attachment is 40 % from the posterior limit, 50 % from the distal limit, and 60 % from the anterior limit [1].

The MPFL is known to be the principal passive soft tissue restraint to patellar lateral displacement. If the MPFL was transacted, it caused significant changes in patellar kinematics, with increased lateral tilt and translation. This change was matched by significant increases in lateral trochlear articular contact stresses and reduction of the medial facet contact stresses. These changes make a case for the desirability to restore MPFL function [2].

When MPFL reconstruction was studied, using a 2-strand gracilis tendon graft from a single femoral tunnel to a groove along the medial edge of the patella, it was found that the best graft tension was only 2 N, that is, only just take out the slackness from the graft, and do not pull the patella medially. Any higher graft tension led to elevated cartilage contact pressures on the medial facet of the PF joint. If a central, anatomical graft tunnel position was found on the femur, the MPFL graft was close to isometry across the range of knee flexion–extension, with approximately 2 mm elongation in the last 15° of knee extension. So the angle where the graft was tensioned did not matter, in the range from 30° to 60° knee flexion, to restore the articular contact pressures to normal across the medial and lateral facets of the PF joint. If the femoral graft tunnel was placed 5 mm proximal or 5 mm distal from the anatomical site, it caused significant joint contact pressure elevation, in line with the length changes of the natural MPFL. That is, 5 mm proximal attachment led to elevated pressures on the medial facet of the PF joint in the flexed knee, and 5 mm distal attachment led to elevated pressures in the extended knee [3].

9.2.2 Indications for Isolated MPFL Reconstruction and the Influence of the Soft Tissue Anatomy on the Diagnosis and Treatment: When Is a Soft Tissue Procedure Sufficient for Patella Stabilisation?

Elizabeth A. Arendt

To describe the ideal patient who would benefit from an MPFL reconstruction to guard against recurrent lateral patella dislocation (LPD) is not a difficult task on paper. What makes this a difficult task in practice is that in the patellofemoral joint, there is not a black and a white answer, but rather numerous shades of grey. This is due to the varied soft tissue and bony dysplasias of the patellofemoral joint that are associated with lateral patella dislocations. Alteration of anatomy, of both soft tissue and bone, can make any one patient a blend of normal, near normal, slightly dysplastic, and highly dysplastic features.

Although we have many measurement schemes to help to objectify our decisions, the type of surgical decision will involve a blend of imaging and physical exam features, combined with patient expectation and surgeon's experience and judgement.

This handout will discuss evidence (Level V) of when a soft tissue procedure sufficient for patella stabilisation against recurrent lateral dislocations. In addition it will assess if current literature helps the clinician in this clinical dilemma.

An ideal candidate for an isolated MPFL reconstruction (without bony work, e.g. tibial tubercle osteotomies/trochleoplasties) might have the following profile of risk factors [4–6]:

- A normal trochlea and a low-grade dysplasia. Low-grade dysplasia can be described as type A (D. Dejour classification) or those patients with normal tracking of the patella through an active arc of motion (no significant J sign or excessive quadriceps pull sign).
- No 'excessive' lateral vector. Though the role of tibial tubercle medialisation as a necessary component of patella stabilisation is being challenged by our current surgical indications, an excessive lateral vector force usually implies a more dysplastic joint.

  A tubercle sulcus angle of 0–5° valgus on physical exam and TT-TG <20 mm (no significant malalignment of the patellofemoral joint) are good candidates for 'isolated' MPFL.

  These patients usually have a reduced patella on axial imaging in early flexion in a non-effused knee.
- No 'excessive' patella height. Though our most common measurement schemes are tibial based, for me it comes down to 'reasonable' overlap of the patella and trochlea surfaces on sagittal MR (functional patella engagement with the trochlea).

In regard to lateral lengthening, the author reviews the following preoperative factors to aid in the decision of how to manage the lateral soft tissue structures.

- Lateral patella tilt less than 20° utilising axial image with posterior femoral condyles as a reference, measured on an image without notable knee effusion (in a non-acute injury setting), usually does not need lateral lengthening.
- If the non-acute axial image in full extension shows increased lateral patella tilt but the tilt corrects in early flexion (20° Laurin's view or a 30° Merchant's view), the patella rarely needs lateral structures lengthened.
- Axial radiographs taken in early flexion reveal excessive lateral tilt on both sides, with no injury to the opposite (non-injured) knee; this is a strong sign that lateral-sided deforming forces are present.
- Patella tilt that has no lateral tightness on physical exam after the patella is relocated does not need lateral side lengthening (This may be necessary to evaluate intra-operatively.)

9.2.3 Surgical Technique

9.2.3.1 MPFL Reconstruction Using the Superficial Quad Technique

Deepak Goyal and Christian Fink

Introduction

MPFL reconstruction using hamstring grafts is quite popular but carries an important inherent risk of complications associated with patellar bony procedure. Patella fracture, violation of anterior cortex or chondral surface of patella, irritation due to hardware, gradually rising stress-riser effect, etc. are commonly reported problems [7–13]. Improper point of isometry at the femur and failure to keep optimum length of the graft are other important reasons for development of complications like patellofemoral overload [14], medial patellofemoral arthritis [15], persistent lateral instability [16], and loss of postoperative movement [11].

Superficial slip of quadriceps tendon is a broad and thin graft similar to native MPFL and has biomechanical properties similar to MPFL. Its use eliminates hardware fixation at the patella. Steensen et al. [17] had used a partial thickness graft from quadriceps tendon. Goyal presented a modification called 'the superficial quad technique' during 14th ESSKA congress, Oslo, 2010, and later reported midterm results of his series in 2013 [18]. The modifications involved providing anatomical fixation points at the patella without use of any hardware, passage of graft through anatomical subvastus space, and isometric fixation at femur and keeping optimum length of the graft instead of tensioning it. As there was no patellar bony procedure required with the superficial quad technique, there were no patellar complications. Fink developed a further modification by developing a technique for the close harvest of the graft.

Surgical Technique

MPFL reconstruction should always be preceded by arthroscopic joint assessment and management of intra-articular pathologies. The superficial slip of quadriceps tendon is actually the anterior most lamina of the three-layered quadriceps tendon structure. It is best dissected few centimetres above the upper pole of patella. A surgical plane of separation exists a few centimetres above the upper pole of patella, from where a broad strip of tendon, around 10 mm wide, can be dissected as far as tendino-muscular junction proximally. Distally one must take care to avoid the penetration of the joint, and thin/broad anterior lamina dissection is carried as an oblique dissection on the anterior surface of patella. The lateral subperiosteal dissection is carried out till midpoint of supero-inferior length of patella on lateral side, while medial dissection ends at the level of superomedial corner of patella. Now the graft is rotated medially and is passed through subvastus space. While Steensen et al. [17] leaves the graft attached on anterior surface of patella, Goyal [18] fixes the graft at superior half of medial border of patella at its anatomical attachment. Fink recommends forming a subperiosteal sleeve of tissue on the medial part of anterior surface and passing the graft underneath the sleeve to have more secured fixation.

There are various methods of fixation of superficial slip of the graft on femoral side. While Goyal uses Farr and Schepsis technique to fix the graft on isometric point, others use Redfern's technique [19] or Schoettle's technique [20]. The superficial quad technique described by Goyal stresses importance of keeping optimum length of the graft instead of tensioning the graft. Any undue tension can lead to immediate postoperative stiffness, pain, loss of motion, and late onset of medial patellofemoral arthritis.

As there are secure fixations on both the sides using the superficial quad technique, there is no need to immobilise the joint. A simple brace for comfort of the patient is enough. Patient is encouraged to start ROM exercises on the same day or as soon as pain is under control. Walking is also allowed once patient regains a good quadriceps control.

Discussion and Anatomical Considerations

Literature confirms that MPFL is a thin, broad, sheetlike ligament with length varying from 45 to 65 mm and width varying from 10 to 32 mm (Smirk and Morris [21], Nomura et al. [22], Tuxøe et al. [13]). Average length of semitendinosus and gracilis is 250 and 220 mm, respectively, and these are cordlike grafts. According to Andrikoula et al. [23], the mean length of superficial slip of quadriceps tendon is 68 mm (range, 50–85) with knee flexed and distance measured from superior border of patella to myoaponeurotic junction of rectus femoris. The average width is 41 mm at superior aspect of patella and 22 mm at middle of tendon. Hence superficial slip of quadriceps tendon is more anatomical match of native MPFL.

Wide area of attachment at patella will have better rotational control on the patella, during flexion–extension movement. Also, it will avoid abnormal biomechanical stresses at the graft–patella junction. Hamstrings tendons, being cordlike structures, can only provide a single-point (or two point) fixation, while superficial slip of quadriceps tendon, being broad in structure, can provide continuous attachment on the medial border of the patella.

Biomechanical Considerations

Mountney et al. [24] studied strength of various fixation methods in MPFL reconstruction along with strength of native MPFL itself. While strength of sutures alone was average 37 N, that of bone anchors combined with sutures was 142 N. Both were remarkably less than strength of MPFL itself that is mean 208 N. When using blind-tunnel tendon graft, it was 126 N, and with through-tunnel graft, it was mean 195 N. The strength of various fixation methods in MPFL reconstructions is remarkably less than the strength of MPFL itself. That means a graft that is fixed at patella using any means is more likely to fail at the patella fixation point rather than at mid-substance, even if its strength and stiffness is similar to native MPFL. On the other hand, a reconstruct which is stronger and stiffer than original MPFL will put more loads on patella when subjected to a severe stress. The loads will be much higher in presence of other persistent anatomical abnormalities like trochlea dysplasia, patella alta, abnormal TTTG distance, etc. To avoid such overload and overstress at patella, stiffness and strength of an ideal graft should be as near to the native MPFL as possible.

Mean strength of the MPFL is 208 N with a mean stiffness of 24 N/mm. Hamner et al. [25] found that the mean strength and stiffness of single-strand gracilis was 402 and 666 % higher, respectively, than the native MPFL. Similarly mean strength and stiffness of double-strand gracilis was 745 and 1,400 % higher, respectively, than the native MPFL. On the similar considerations, it was found that mean strength and stiffness of single-strand semitendinosus was 509 and 887 % higher, respectively, than native MPFL. Similarly mean strength and stiffness of double-strand semitendinosus was 1,120 and 1,954 % higher, respectively, than native MPFL. Herbort et al. [26] found out the mean strength of superficial slip of quadriceps is 204 N, compared to that of MPFL being 190 in his study. The mean stiffness value for the superficial slip of quadriceps was 33 N, compared to that of MPFL being 29 in his study. Studies of Hamner and Herbort confirm that superficial slip of quadriceps tendon is much better biomechanical match of native MPFL.

Patellar Complications Due to Bony Fixation

Patella fracture is the most devastating complication reported after MPFL reconstruction with a hamstring graft. According to one study, up to 90 % strength reduction in the bone can occur depending on the geometry and the size of the bony defects [27]. A weak medial patellar ridge has a potential for late patella fracture. As discussed previously, a stronger and a stiffer graft puts more load on patellofemoral joint as against a native MPFL. The extra load put on the graft–patella junction by a stronger graft will have further deteriorating effect on weak medial patellar ridge. This continuous load over a period can cause stress-riser effect and lead to late patella fractures after many years [12]. Patella fracture is a complication that cannot be accepted from a surgery that was aimed to treat patella instability.

Other complications can be accidental damage of the anterior or the chondral surface while creating the bone tunnels. Fixation devices such as suture anchors or buttons may lead to foreign-body reaction or other hardware-related intraoperative or postoperative problems [11]. This can lead to poor postoperative result and early onset of patellofemoral arthritis. A graft that can eliminate bony fixation at patella indirectly eliminates all associated complications.

Conclusion

The superfi.cial slip of quadriceps tendon is a better anatomical and biomechanical match to the native MPFL. It provides anatomical patellar fixation without any requirement of patellar hardware and thus avoids hardware/bony procedure related to patellar complications.

9.2.3.2 Surgical Technique: MPFL Reconstruction Using a Double-Bundle Gracilis Tendon with Swivel Lock Fixation

Philip Schoettle

Reconstruction of the MPFL has become a widespread technique for restoration of patellofemoral stability. The main reason that MPFL reconstruction became popular is the fact that distal realignment procedures such as transfer of the tibial tuberosity or release at the lateral patellar retinaculum/capsule have provided inadequate restoration of patellofemoral stability in every patient, frequently leading to increased mediolateral instability, increased patellofemoral pressure, or arthritic degeneration.

Therefore, numerous techniques for reconstruction of the medial patellofemoral complex have been described with promising clinical results. However, since it is known that a nonanatomical reconstruction of the MPFL can lead to non-physiological patellofemoral loads and kinematics, the goal of a surgical intervention must be an anatomical reconstruction. Since the femoral insertion of the MPFL has been evaluated anatomically, biomechanically, and radiologically, the complications of increased patellofemoral pressure in flexion associated with nonanatomical femoral graft fixation that is too anterior/proximal can be avoided. Upon careful observation of the anatomical shape of the original MPFL, it is apparent that the patellar insertion is much wider than the femoral one. Additionally, Amis et al. have proven double-bundle structure provides a more stable proximal and distal ligamentous structure. Respecting this anatomical condition, a double-bundle reconstruction at the patellar side is reasonable to restore native ligamentous morphologic and biomechanical properties; moreover, this method lessens the patellar rotation during flexion–extension movement that may occur during single-bundle reconstruction. Under these conditions, the double-bundle reconstruction, described earlier shows very satisfying clinical results. As we know from ACL reconstruction, direct anatomical/aperture fixation provides the highest time-zero fixation by avoiding elongation of the graft or ‘bungee’ effect, resulting in the possibility of early rehabilitation with a full range of motion. In a similar manner, these concepts may be applied to MPFL reconstruction.

Although most of the actual surgical techniques utilise a free tendon graft to reconstruct the MPFL as the only method for anatomical double-bundle graft fixation, an all aperture fixation has not yet been described.

Recent studies have described an anatomical double-bundle reconstruction, using an aperture fixation at the femoral insertion, while the patellar fixation remains relatively indirect resulting in the eventual risk of postoperative micromotion and subsequent loosening. Patellar graft fixation has been described either with an anchor system, attaching the graft into a bony rim, or by tying the attached graft sutures to each other at the lateral patellar edge; however, this method may potentially result in graft slippage by degloving.

Until today, only one technique described anatomical patellar fixation by looping graft through bone tunnels without any additional fixation device. This technique appears to produce stable fixation at the patella. However, in soft bone, a widening of the tunnel could occur in the long term; moreover, in patients with a short gracilis graft, the tendon length may not be long enough to reach the anatomical femoral insertion.

The double-bundle technique described here offers an aperture fixation at the patella and the femur, providing a high initial stability on both insertions, resulting in improved bony ingrowth and, consequently, an earlier return to full range of motion.

Harvesting and Preparing of the Gracilis Tendon

After completion of the arthroscopy, a 2 cm long oblique incision is performed at the pes anserinus. After incising the sartorius aponeurosis, the gracilis tendon is harvested and used as an autograft. The load to failure force of the Gracilis graft – even as a single bundle – exceeds the failure to load of the MPFL (208 N.) The usable part of the tendon should be at least 18 cm long. After harvesting the tendon with the stripper and removing the muscle tissue, the doubled tendon diameter is determined, and both ends are whipstitched with an absorbable braided suture over a length of 15 mm.

Preparing the Soft Tissue Layer

A 2 cm skin incision is performed from the superomedial corner to the end of the medial margin of the patella, where the patellar MPFL insertion is located. As the MPFL is situated central to the vastus medialis obliquus (VMO) in the second layer of the medial patellofemoral complex, the central part of the VMO is identified, and a scissor is brought along to the medial femoral epicondyle in between the VMO and the joint capsule, cautiously avoiding any injury to the joint. After the opened scissors are removed, a right-angle clamp is brought into the separated layer, and the tip is directed towards the skin in the area of the adductor tubercle, where the femoral MPFL insertion is located. Then a small longitudinal skin incision is performed over the tip in 30° knee flexion, the position where the graft will be finally fixed. Finally, in preparation for passing the final graft, a suture loop is inserted in between the second and the third layer using the right-angle clamp.

Preparing the Femoral Insertion Site

To avoid non-physiological patellofemoral forces, the femoral MPFL insertion has to be very accurate. Therefore, a guide wire with an eyelet is placed slightly posterior to the midpoint of the medial epicondyle and the adductor tubercle, and the entering point into the bone is marked with a clamp. Then the guide wire placement is controlled by a picture intensifier on a straight lateral view to obtain the correct anatomical femoral insertion; if the graft is placed too anterior or proximal, abnormal graft tensioning will lead to increased patellofemoral pressures during flexion. Therefore, we use the radiographic landmark of the anatomical MPFL insertion which has been shown to be located slightly anterior to an elongation of the posterior femoral cortex in between the proximal origin of the medial condyle and the most posterior point of Blumensaat's line. If necessary, the guide wire entry point is corrected before overdrilling to the contralateral cortex with a drill diameter 1 mm larger than that of the graft loop.

Preparing the Patellar Tendon Insertion Site

To achieve aperture fixation at the patellar side, the free graft ends have to be fixated directly to the patella. Therefore, the medial patellar margin is prepared, and two guide wires are drilled tangentially into the patella at the proximal and distal end of the medial edge. The guide wires are subsequently overdrilled with a cannulated 4 mm drill to a depth of 20 mm.

Graft Fixation

The two free-sutured graft ends are fixed into the patellar holes one after each other, using a 4.75 × 15 mm Swivel Lock (Fa. Arthrex), achieving a direct anatomical graft fixation. To accomplish this, the graft sutures are pulled through the PEEK eyelet of the Swivel Lock and pushed into the drill holes. Keeping the suture under tension, the graft ends are fixed with the 4.75 × 15 mm Swivel Lock screw. In this way, a double-bundle aperture fixation at the patellar side is achieved, leaving the graft loop free.

The suture loop is then used to pull the graft in between layer 2 and 3 to the femoral insertion. Next, a nitinol wire is inserted into the femoral drill hole, and the suture loop of the graft is pulled laterally using the guide wire. Finally, while maintaining equal tension on both bundles, the graft is pulled into the femoral socket. Since biomechanical studies have shown that the MPFL has its maximal length and restraint against patella lateralisation in 30° of flexion, femoral fixation is performed in 30° of flexion with the lateral patellar edge positioned in line with the lateral trochlear border using a bioresorbable interference screw. An anatomical femoral insertion avoids an overcorrection, since an overtension of the graft can only occur if the femoral tunnel is placed too far anterior or proximal. In this case, the insertion point would move towards posterior in flexion, leading to a lengthening of the distance between patellar and femoral insertion, increasing the load onto the graft and, consequently, onto the patellofemoral joint.

If adequate medial restraint has been restored, lateral patellar dislocation should no longer be possible, and routine skin closure is performed after reattaching the aponeurosis of the VMO back to the medial edge of the patella with resorbable sutures.

Postoperative Treatment

Compared to other techniques, this aperture fixation with a biotenodesis screw at the patellar insertion provides an immediate stable tendon to bone fixation with an ultimate load to failure force at the patellar side higher than the 208 N needed to rupture an intact MPFL. Weight bearing is allowed, however, no more than 20 kg until wound healing, while leg raising and quadriceps setting exercises can be started immediately with a free range of motion as tolerated.

Low-impact activities such as running or cycling are allowed at 6 weeks post-op; full activity is permitted at 3 months.

9.2.3.3 Surgical Technique Using a Double-Bundle Dynamic MPFL Reconstruction with a Free Gracilis Tendon Autograft

A. Rood, K. Groenen, A. Lentinga,
N. Verdonschot, A.V. Kampen, and Sander Koëter

Why Reconstruct the MPFL?

The medial patellofemoral ligament (MPFL) is the most frequently injured soft tissue structure following acute lateral patellar dislocation. MPFL reconstruction has become a popular option to restore patellar stability following lateral patellar dislocation. The goal is to reconstruct the MPFL in a way that mimics the pre-traumatic condition of the patellofemoral joint in safe and reproducible way with minimal change of complications. In some papers complication rate can be as high a 25 %, with stiffness of the knee and patellofemoral pain being the most common. Some complications are influenced by the operative technique chosen.

Why Use the Gracilis Tendon?

Graft choice depends on graft length, graft stiffness, graft strength, and graft fixation possibilities. The ideal graft has qualities that resemble the native MPFL in strength, elasticity, and length. An advantage of using the gracilis is that the mechanical properties resemble the native MPFL more than those of the quadriceps. The tensile strength of the MPFL is 208 N (SD 90) at 26 mm (SD 7) of displacement. The gracilis has a tensile strength of 800 N, while the quadriceps tendon graft has a thicker cross-sectional area and a much higher tensile strength of 2,352 N to failure. The stiffness of the native MPFL is 8 N/mm, the stiffness of the gracilis is 171 N/mm and that of the quadriceps is even greater. Another advantage of gracilis tendon harvest is that the extensor mechanism is not violated with a hamstring harvest which causes less initial quadriceps atrophy. This advantage may not be clinically relevant, since extensor mechanism wasting is usually more caused by the postoperative treatment.

Which Fixation Technique?

Fixation of the graft can be with bone anchors, interference screws, or sutures. The optimal fixation is rigid enough to prevent dislocation but allows full range of motion and does not influence the patellofemoral pressure.

To evaluate which fixation technique of the MPFL approaches the original situation best in terms of patellofemoral pressure, we performed a biomechanical in vitro study. We measured the patellofemoral pressure at different angles of knee flexion in normal, undamaged cadaveric knees and compared this to the pressure after cutting the MPFL and after three reconstructive techniques of the MPFL (a fully dynamic reconstruction, a partial dynamic fixation through bone tunnels in the patella, and a static fixation on both the femoral and patellar side). Seven fresh frozen knee specimens were tested in an in vitro simulation using a knee joint motion and loading apparatus. We harvested a gracilis tendon to use as a graft for reconstruction of the MPFL later on. We divided the quadriceps muscles into three groups. A total load of 50 N was applied to the three muscle groups. We created a constant 20 N force on the hamstrings. The knee joint was opened by a small medial arthrotomy. A pressure-sensitive film was fixed to the retropatellar cartilage with skin glue, covering the whole surface of the patella, where after the knee joint was closed using sutures to restore intact conditions. The knee was inserted into the knee loading and motion apparatus. We started the pressure and orientation measurements in fixed flexion angles from 0 to 110 in the following five conditions: A, normal knee condition; B, transected MPFL; C, complete dynamic MPFL reconstruction where we attached the graft only to soft tissues on both femoral and patellar side; D, partial dynamic reconstruction where the patellar attachment was altered by fixing the graft through bony tunnels; and E, static MPFL reconstruction, stapling the graft onto the femur at the location of the isometric point. As primary outcome measurement we looked at patellofemoral mean pressure and peak pressure. As secondary outcome measurements we evaluated centre of force (COF) and contact area. We compared all different reconstructions and the situation where the MPFL was cut (group B–E) to baseline conditions (group A). Mean patellofemoral pressure increased slightly with deeper flexion in all conditions, but it was highest after static reconstruction (E). The pressures in condition B and C looked most similar to baseline condition (A). The peak pressure also differed the most in condition E. In this static reconstruction the peak pressure in deep flexion was much higher than in the other situations. The dynamic reconstruction (C) showed the most similar peak pressure compared to the original situation. In all conditions the contact area increased when the knees were more flexed. There were no significant differences between the groups.

This study shows that after a dynamic reconstruction patellofemoral pressures return to the normal situation. Rigid fixation causes higher peak and mean pressures. This could suggest that after a dynamical method, the risk of pressure-related complications is lowest.

Which Operative Technique?

We use a gracilis tendon through bone tunnel in patella. The bone tunnels do not completely transverse the patella to minimise the risk of fracture. The MPFL is not a genuine ligament (like the ACL) but rather a thicker part of the medial retinaculum. Please see Fig. 9.1 for more details.

What Are the Results?

We retrospectively evaluated the results. Between 2009 and 2013, 117 patients were operated. Redislocation was seen in two patients; in both cases patients were treated with a trochlea osteotomy. No clinical significant decrease in ROM was noted. A patella fracture was seen in two cases, in both cases after a fall (during field hockey at 8 weeks post-op and during physical therapy at 3 months post-op). One patient was treated conservatively; the other was operated.

> **Conclusion**
> This is one of the many techniques for reconstructing the MPFL. Advantages of this technique include its biomechanical advantages; it is not technically demanding and seems relatively safe.

9.2.3.4 Surgical Technique for MPFL Reconstruction After TKA

S. van Gennip, J.J. Schimmel, G.G. van Hellemondt, K.C. Defoort, and A.B. Wymenga

Maltracking of the patella after total knee arthroplasty (TKA) remains a well-recognised problem. The medial patellofemoral ligament (MPFL) has shown to be important for patellar stabilisation, and reconstructions of the MPFL have already shown excellent functional outcomes for patellar instability of the native knee. Nevertheless, there is only limited literature on using an MPFL reconstruction for correction of patellar maltracking after TKA. In this retrospective study, a consecutive case series was evaluated.

Between 2007 and 2010, nine patients (nine knees) with anterior knee pain and symptomatic (sub)luxations of the patella after primary or revision TKA were treated by reconstruction of the MPFL in combination with a lateral release. In two cases, an additional tibial tuberosity transfer was performed, due to insufficient preoperative correction. Preoperative workup included a CT scan to rule out component malrotation and disorders in limb alignment. Pre- and postoperative patellar displacement and lateral patellar tilt were measured on axial radiographs. Clinical outcome was evaluated using the visual analogue scale (VAS) satisfaction, VAS pain, dislocation rate, and Bartlett patella score.

Median patellar displacement improved from 29 mm (0–44) to 0 mm (0–9) postoperatively. Median lateral patellar tilt was 45° (23–62) preoperative and changed to a median 15° (−3 to 21)

Fig. 9.1 Anatomic double bundle MPFL reconstruction. The MPFL is a thicker part of the retinaculum and its attachment is close to the adductor insertion (at the scissors). A gracilis tendon is prepared. A separate incision is made at the patella and at the medial epicondyle. If necessary avulsion fragments can be removed. The adductor tubercle is prepared. Two patellar tunnels are made. The graft is fixed using resorbable sutures

postoperative. Median VAS satisfaction was 8 (5–9), and only one patient reported a subluxing feeling afterwards. The Bartlett patella score displayed a diverse picture.

Patellar maltracking after primary or revision TKA without malrotation can effectively be treated by MPFL reconstruction in combination with a lateral release. Only in limited cases, an additional tibial tuberosity transfer is needed.

9.2.4 The Failed MPFL

D. Dejour

There are various reasons for failure of the reconstructed MPFL. Before revision procedures you should evaluate the reason for failure. Reasons can be underlying bony pathology and technical problems. This session will address management of the failed MPFL.

9.3 Future Directions for Research and Clinical Decision-Making Based on Literature Review

Elizabeth A. Arendt

In regard to understanding in what clinical setting is an isolated MPFL reconstruction of value, one would hope to be able to turn to the literature for some answer. A recent systematic review of PubMed on MPFL reconstructions was performed at the University of Minnesota.

Inclusion criteria were 'isolated' MPFL reconstructions without any bony procedure, at least ten patients in the cohort and in a journal with an impact factor >2.

We found 24 articles that fit these criteria. The main points of the systematic review are outlined below.

What kind of outcomes were reported? $n=24$

- Rate of redislocation – nearly all
- Kujala score (pain scale) – nearly all
- VAS (pain) – few
- Return to activity – most commented on this but without a metric
- QOL scale – few (KOS, IKDC, Cincin.)
- PF instability scale – none

What kind of preoperative variables were reported? $n=24$

- # of patients in study (range 12–193) 5>45 patients
- Age M 23, range 10–52
- 'Children': variably reported as either open physis or <18 y/o

Preoperative variables recorded in M & M (% of all studies, $n=24$)

Mechanism of injury (A vs T)	71 %
Cartilage status	71 %
Activity level (1° Tegner)	50 %
Prior procedures	71 %

Preoperative variables recorded in M & M

Physical exam	
(+) Apprehension sign and/or quad. translation	56 %
Hyper laxity syndrome	21 %
Version	25 %

Preoperative variables recorded in M & M Imaging measurements

Patella height	72 % C/D and/or I/S methods
Trochlear dysplasia (TD)	80 %
Most studies used sulcus angle to determine TD	
TT-TG	80 %
Tilt (variably measured)	54 %

MPFL Reconstruction: In what population was 'isolated' procedures performed?

Were anatomical instability factors accounted for in the patient selection?

Increased Q Angle: patients were excluded if TT-TG was elevated:

Wang et al. [28]	>15 mm
Howell et al. [29]	>18 mm
Kang et al. [30]	>20 mm

Patella alta and selection criteria were discussed:

- Steiner et al. [31]
  - 'Significant alta' excluded
  - M I/S 1.16
- Nomura et al. [32]
  - I/S: M 1.08
  - Range (.98–1.23)
- Thaunat/Erasmus [33]
  - 'Severe' patella alta with extensor lag
- Patella alta: excluded if >1.2 (I/S)
  - Ronga et al. [34]
  - Kang et al. [30]
  - Goyal [18]
  - Wang et al. [35]

9.3.1 Gaps of Knowledge in Current Literature and Possible Recommendations for Future Research

- Current literature on MPFL reconstruction contains variable selection bias in the patients that they are reporting on that influences the results.
- Current literature on MPFL reconstructions contains non-uniform methods of reporting preoperative variables and outcomes.
- More clarity in reporting methodology is needed to be useful for the treating clinician.

9.4 Take-Home Message

- The MPFL is nearly always damaged after patellar dislocation.
- MPFL reconstruction seems a valuable technique in case of recurrent dislocation.
- There is no consensus regarding the exact indication for isolated MPFL reconstruction in literature.
- There are various operative techniques available, all with their different advantages and disadvantages (with regard to stability, ease of conduct, specific complications, and costs).

References

1. Stephen J, Lumpaopong P, Deehan DJ, Kader D, Amis AA. The medial patellofemoral ligament: location of femoral attachment and length change patterns resulting from anatomic and non-anatomic attachments. Am J Sports Med. 2012;40:1871–9.
2. Stephen JM, Kader D, Lumpaopong P, Deehan DJ, Amis AA. Sectioning the medial patellofemoral ligament alters patellofemoral joint kinematics and contact mechanics. J Orthop Res. 2013;31:1423–9.
3. Stephen J, Kader D, Lumpaopong P, Deehan DJ, Amis AA. The effect of femoral tunnel position and graft tension on patellar contact mechanics and kinematics following MPFL reconstruction. Am J Sports Med. 2013;42(2):364–72.
4. Biedert RM, Albrecht S. The patellotrochlear index: a new index for assessing patellar height. Knee Surg Sports Traumatol Arthrosc. 2006;14:707–12.
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. Arendt EA, Dejour D. Patella instability: building bridges across the ocean a historic review. Knee Surg Sports Traumatol Arthrosc. 2013;21:279–93.
7. 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(1):82–7.
8. Ellera Gomes JL, Stigler Marczyk LR, César de César P, Jungblut CF. Medial patellofemoral ligament reconstruction with semitendinosus autograft for chronic patellar instability: a follow-up study. Arthroscopy. 2004;20(2):147–51.
9. Fithian DC, Gupta N. Patellar instability: principals of soft tissue repair and reconstruction. Tech Knee Surg. 2006;5:19–26.
10. Mikashima Y, Kimura M, Kobayashi Y, Miyawaki M, Tomatsu T. Clinical results of isolated reconstruction of the medial patellofemoral ligament for recurrent dislocation and subluxation of the patella. Acta Orthop Belg. 2006;72(1):65–71.
11. 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(11):1542–4.
12. Thaunat M, Erasmus PJ. Management of overtight medial patellofemoral ligament reconstruction. Knee Surg Sports Traumatol Arthrosc. 2009;17(5):480–3.
13. Tuxøe JI, Teir M, Winge S, Nielsen PL. The medial patellofemoral ligament: a dissection study. Knee Surg Sports Traumatol Arthrosc. 2002;10(3):138–40.
14. Elias JJ. Technical errors during medial patellofemoral ligament reconstruction could overload medial patellofemoral cartilage: a computational analysis. Am J Sports Med. 2006;34(9):1478–85.

15. Arendt EA, Fithian DC, Cohen E. Current concepts of lateral patella dislocation. Clin Sports Med. 2002;21(3):499–519.
16. Schöttle PB, Fucentese SF, Romero J. Clinical and radiological outcome of medial patellofemoral ligament reconstruction with a semitendinosus autograft for patella instability. Knee Surg Sports Traumatol Arthrosc. 2005;13(7):516–21.
17. Steensen RN, Dopirak RM, Maurus PB. A simple technique for reconstruction of the medial patellofemoral ligament using a quadriceps tendon graft. Arthroscopy. 2005;21(3):365–70.
18. Goyal D. Medial patellofemoral ligament reconstruction: the superficial quad technique. Am J Sports Med. 2013;41(5):1022–9. doi:10.1177/0363546513477828.
19. 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(2):293–7.
20. 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(5):801–4.
21. Smirk C, Morris H. The anatomy and reconstruction of the medial patellofemoral ligament. Knee. 2003; 10(3):221–7.
22. Nomura E, Horiuchi Y, Kihara M. Medial patellofemoral ligament restraint in lateral patellar translation and reconstruction. Knee. 2000;7(2):121–7.
23. Andrikoula S, Tokis A, Vasiliadis HS, Georgoulis A. The extensor mechanism of the knee joint: an anatomical study. Knee Surg Sports Traumatol Arthrosc. 2006;14(3):214–20.
24. Mountney J, Senavongse W, Amis AA, Thomas NP. Tensile strength of the medial patellofemoral ligament before and after repair or reconstruction. J Bone Joint Surg Br. 2005;87(1):36–40.
25. Hamner DL, Brown Jr CH, Steiner ME, Hecker AT, Hayes WC. Hamstring tendon grafts for reconstruction of the anterior cruciate ligament: biomechanical evaluation of the use of multiple strands and tensioning techniques. J Bone Joint Surg Am. 1999;81(4): 549–57.
26. Herbort M, et al. Bio-mechanical properties of a New MPFL reconstruction technique using quadriceps tendon in comparison to the intact MPFL. A human cadaveric study. Presented during 9th biennial ISAKOS Congress, Toronto, Canada. 2013.
27. Clark CR, Morgan C, Sonstegard DA, Matthews LS. The effect of biopsy-hole shape and size on bone strength. J Bone Joint Surg Am. 1977;59(2):213–7.
28. Wang JL, Li HP, Liu YJ, Wang N, Li ZL, Wang ZG, et al. Reconstruction of the medial patellofemoral ligament with a suture-tie technique of patellar side fixation. Chin Med J (Engl). 2012;125(11):1884–8.
29. Howells NR, Barnett AJ, Ahearn N, Ansari A, Eldridge JD. Medial patellofemoral ligament reconstruction. Bone Joint Surg Br. 2012;94-B: 1202–8.
30. Kang H, Cao J, Yu D, Zheng Z, Wang F. Comparison of 2 different techniques for anatomic reconstruction of the medial patellofemoral ligament: a prospective randomized study. Am J Sports Med. 2013;41(5): 1013–21. doi:10.1177/0363546513480468.
31. Steiner TM, Torga-Spak R, Teitge RA. Medial patellofemoral ligament reconstruction in patients with lateral patellar instability and trochlear dysplasia. Am J Sports Med. 2006;34(8):1254–61.
32. Nomura E, Inoue M. Hybrid medial patellofemoral ligament reconstruction using the semitendinous tendon for recurrent patellar dislocation: minimum 3 years' follow-up. Arthroscopy. 2006;22(7):787–93.
33. Thaunat M, Erasmus PJ. The favourable anisometry: an original concept for medial patellofemoral ligament reconstruction. Knee. 2007;14(6):424–8.
34. Ronga M, Oliva F, Longo UG, Testa V, Capasso G, Maffulli N. Isolated medial patellofemoral ligament reconstruction for recurrent patellar dislocation. Am J Sports Med. 2009;37(9):1735–42. doi:10.1177/0363546509333482.
35. Wang CH, Ma LF, Zhou JW, Ji G, Wang HY, Wang F, et al. Double-bundle anatomical versus single-bundle isometric medial patellofemoral ligament reconstruction for patellar dislocation. Int Orthop. 2013;37(4):617–24. doi:10.1007/s00264-013-1788-6.

ICL-15 Cartilage Lesion and the Patellofemoral Joint

10

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Patellar cartilage lesions are one of the most frequent articular cartilage defects seen in knee arthroscopic procedures [1]. Today, the understanding of articular cartilage structure, biochemistry, and biomechanics has significantly improved, and most problems of the patellofemoral joint have been clarified. However, lesions of the articular cartilage of the patellofemoral joint still pose a clinical and therapeutical problem.

The present chapter resumes the presentations of the Instructional Course Lectures at the 2014 ESSKA Congress on the evaluation and management of cartilage injuries affecting the patellofemoral joint.

10.1 Anatomy and Biomechanics of the Patellofemoral Joint

The patella is the largest sesamoid bone in the body. It is a flat triangular bone and functions to direct forces of the quadriceps, protect the deeper knee joint anatomy, and protect the quadriceps tendon from frictional forces [2, 3]. The patella has a rounded base, proximally to which the quadriceps tendon is attached. Distally, the patella narrows to a pointed end, which attaches to the patellar ligament. Anteriorly, the patella is

S. Zaffagnini et al. (eds.), *ESSKA Instructional Course Lecture Book*,
DOI 10.1007/978-3-642-53983-1_10, © ESSKA 2014

convex, with a roughened surface for tendinous attachments. Posteriorly, the patella is covered by thick hyaline cartilage. This articular surface also has a midline ridge that is congruous with the trochlear groove. The distal 25 % of the patella's undersurface is non-articulating.

The articular surface of the patella is divided into two large facets, medial and lateral. Facet morphology can be classified into one of the three groups on the basis of the Wiberg classification scheme. Type 1, patellae have concave medial and lateral facets and are equal in size. This makes up 10 % of patella morphology. Type 2, patellae have a flat or convex medial facet that is much smaller in size than the lateral facet. This makes up 65 % of patella morphology. Type 3, patellae have a convex medial facet that is slightly smaller than the lateral facet. This is seen in 25 % of patellae [2, 4].

The femoral trochlea is a 5.5-mm-deep groove in the distal aspect of the femur that intimately articulates with the patella. The lateral facet is larger and extends more proximally and anteriorly than the medial facet. The trochlear groove is covered by a 2- to 3-mm-thick cartilage cap, which tends to be thinner medially. Functionally, the trochlea provides a lateral buttress to lateral subluxation of the patella, starting at approximately 15–20° of knee flexion [2–4]. Patellar contact area changes with increasing knee flexion. In general, the contact area reaches a maximum at 90° of knee flexion and moves proximally on the patella from extension to 90° flexion. At 90° flexion, the proximal aspect of the patella is in contact with the femoral trochlea. As the knee flexes beyond 90°, the patellofemoral contact area decreases, and the tendo-femoral contact area increases. Contact pressure is the ratio of the contact area and the patellofemoral joint reaction force. Force increases from extension to 90° of flexion at a greater rate than contact area increases. The maximum compressive pressures occur at 60–90° of flexion [5, 6].

In the frontal plane, the angle between the quadriceps and patellar tendons has been defined as the Q angle. Its normal range is between 10° and 15° with the knee in full extension. With the knee in flexion, the Q angle decreases because of internal rotation of the tibia relative to the femur. Contraction of the quadriceps creates a bowstring effect, which displaces the patella in a lateral direction, producing a contact force against the lateral margin of the femoral trochlear groove.

Abnormal tracking of the patella, which allows lateral subluxation of only a few millimeters, decreases the contact area and increases the stress on the lateral margin of the trochlear groove and the lateral facet of the patella [7]. Medial patellofemoral ligament (MPFL) disruption may alter patellofemoral joint kinematics and contact pressures, potentially causing pain and articular cartilage degeneration [6].

10.2 Physiopathology of Patellar Chondral Lesions

Patellofemoral cartilage lesions are due to different causes including sport trauma, traffic accidents, osteochondritis dissecans, patellofemoral malalignment, and idiopathic chondromalacia.

Patellofemoral malalignment or maltracking encompasses a number of conditions, isolated or variously associated, such as increased Q angle, high-riding patella (patella alta), trochlear dysplasia, increased femoral anteversion, excessive tension of lateral retinaculum, absence of medial patellofemoral ligament, and vastus medialis obliquus hypotrophy [8–13]. Such disorders lead to altered articular congruence between patella and femoral trochlea. This leads to excess overload with sheer forces being applied to the articular cartilage in some areas, while underloading may occur in others. Both hyperpression and hypopression may be detrimental to nutrition of the cartilage progressing to osteoarthritis.

The term "chondromalacia patellae" describes a pathological alteration of articular cartilage of the patellofemoral joint which, when mild, may be reversible but when severe can lead ultimately to osteoarthritis. However, since it is most often seen on the medial facet of the patella and osteoarthritis on the lateral facet, it can be argued that the one does not necessarily lead to the other [14].

Chondromalacia is classified in four grades according to Outerbridge [15] by arthroscopic evaluation:

Grade 1. Closed disease. The articular cartilage surface looks normal, but the probe reveals a soft spongy feel or pitting edema. The surface is intact and the softening probably reversible.

Grade 2. Open disease. The probe reveals fissures which may be unrecognized at first sight.

Grade 3. Exuberant fibrillation appearance of the articular surface.

Grade 4. Full-thickness fibrillation and the erosion of the articular cartilage extend down to the bone which may be exposed. This is one of the typical features of osteoarthritis.

There is conflicting evidence as to the true pathological nature of chondromalacia. Goodfellow et al. [16] described primary changes in the deep and intermediate layers of the articular cartilage and referred to the process as "basal degeneration." This seems to fit well with the appearance at arthroscopy in grade 1 disease. Bentley [17], however, has biopsied chondromalacic areas on the medial facet and provided evidence of surface fibrillation and loss of superficial matrix that could be the initial chondromalacic change. About the pathogenesis, from a biomechanical point of view, cartilage can be considered as a compressed matrix: the type II collagen fibers tightly bind together the proteoglycans which carry a very high negative charge and are strongly hydrophilic. If the collagen breaks down, the unconstrained proteoglycans will inevitably draw more water into the hydrated matrix causing boggy swelling. Fissuring is the next step in this destructive process, followed by fragmentation and ulceration.

Ficat et al. [14] provided a description of chondral alterations associated with known patellofemoral disorders, such as a lateral patellar tilt around the central ridge secondary to retraction of the lateral retinaculum. The critical zone is that situated around the central ridge of the patella with a certain extension to the lateral facet. Insall et al. [18] found that the most frequent location of chondromalacia had an elliptical area with the major axis lying transverse, bridging over the central part of the patella, with the upper and lower third of the articular surface almost always spared (Fig. 10.1). A topographic study of 105 patients with anterior knee pain or simple malalignment documented that the most frequent site of cartilage lesion (71 %) is a roughly elliptical area with the major diameter parallel to the transverse axis of the patella, not affecting the upper and lower thirds of the patella, i.e., a diffuse lesion (type IV). The lateral and medial facets are affected in 7 and 21 % of cases, respectively [18].

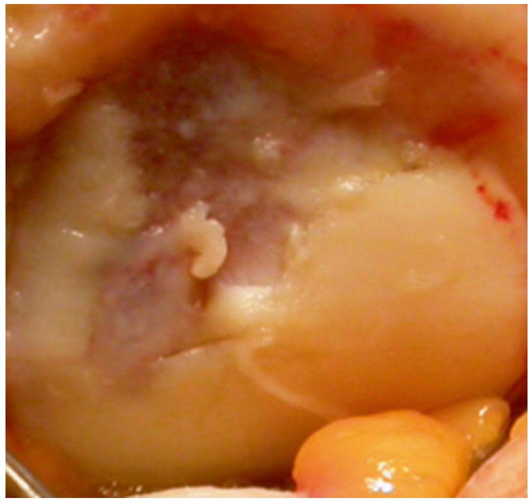

Fig. 10.1 Elliptical area of chondromalacia with the major axis lying transverse, bridging over the central part of the patella, with the upper and lower third of the articular surface almost always spared

10.3 Epidemiology

In a study of 31,000 consecutive knee arthroscopies, Curl et al. reported that 63 % of patients had a chondral defect [19]. In a similar study of 1,000 consecutive knee arthroscopies, Hjelle et al. found that 61 % of their patients had chondral or osteochondral lesions present [20]. The majority of these lesions were found on the medial femoral condyle (58 %), but chondral lesions affecting the patella were the second most common, present in 11 % of cases [7, 20]. Most of the patients (95 %) with acute lateral patellar dislocations treated surgically were found to have chondral or osteochondral lesions affecting the patella [21].

10.4 Presentation and Physical Exam

Patients with patellofemoral cartilage injuries typically present with activity-related anterior knee pain and intermittent swelling and may report mechanical symptoms, including catching with knee flexion. A pain in the anterior knee is often described, especially after sitting for prolonged periods of time ("movie theater sign"). Activity-related pain is associated with knee flexion, including ascending or descending stairs [22]. Occasionally, patients will present with patellar acute instability and a history of traumatic subluxation or dislocation [23].

Clinical examination should include standing assessment of the alignment of the lower limb, the presence of varus or valgus deformity and observation of the patella during movement of the knee, and restriction of flexion by tight structures or shortening of the rectus muscle. Examination of the patellar position should be made from the front while the patient is seated, in order to understand whether it is in a high or lateral position and whether there is lateral patellar tilt as the knee is extended from a flexed position of 90° to full extension.

In the supine position, the patella should be examined by palpation along the undersurface of the medial facet and medial retinacula. The examiner should carry out a hypermobility test with the knee flexed to 30°. Firm pressure is exerted along the medial patella to push it over the lateral femoral condyle. The test is positive if the patient experiences apprehension and discomfort.

Examination of the hip should be made for rotation and to determine femoral anteversion.

10.5 Imaging

10.5.1 X-rays

At least ten different radiological views of this joint have been described. Standing anteroposterior (AP) views offer little information about chondromalacia; however, they are useful to assess for the presence of osteoarthritis, fractures, or other lesions. Tunnel views give insight into femoral condylar disease, including osteochondritis dissecans, and a clearer assessment for the presence of osteoarthritis. The lateral view gives useful information of a high or low patella in adults and children.

In the axial projection the measurement of the sulcus angle (SA) is 142° in mean in normal subjects. Merchant [24] described the measurement of the congruence angle (CA) for patellar subluxation. This is the relationship of the patella to its intercondylar sulcus.

Dejour et al. [25] described a view of the knee in 30° of flexion, with perfect superimposition of the condyles posteriorly which permitted to distinguish three types of trochlear dysplasia important in the evaluation and management of patellar instability.

However, the traditional x-ray views provide static images from which it is difficult to understand fully the dynamics of patellar tracking.

10.5.2 Computed Tomography (CT)

The main advantage of CT when compared with axial radiography is its ability to image the knee in greater degrees of extension. Schutzer et al. [26] identified three patterns of malalignment. Type I is characterized by subluxation without patellar tilt, type II by both subluxation and tilt, and type lll by tilt without subluxation.

CT can also be used to measure the distance between the trochlear groove and tibial tuberosity (TGTT) in millimeters and rotational deformities of the lower limb [27].

CT can be performed in static and dynamic (maximal voluntary contraction of the quadriceps) conditions in order to better visualize dynamic malalignment [28].

10.5.3 Magnetic Resonance Imaging (MRI)

Studies similar to those made with CT can be done using MRI, without exposure to radiation. Various MRI sequences have been developed to provide important information regarding the presence of articular cartilage injuries and the calculation of trochlear depth, sulcus angle, lateralization, and inclination of the patella. Additionally, T2 images can evaluate defect-associated bone marrow edema lesions. In addition, meniscal injuries, synovial plicae, and patellar tendinitis can be visualized [29, 30].

10.5.4 Arthroscopy

This is the most valuable method for evaluating a diagnosis of chondromalacia patellae. The cartilage on the patella and trochlea should be assessed according to Outerbridge classification. Arthroscopic assessment of patellar tracking is undertaken through an anterolateral or superolateral portal. It is possible to also observe overhang of the lateral facet.

10.6 Conservative Management

Basic nonsurgical management is used as an initial treatment modality to treat chondral lesions of the patellofemoral joint for at least 6 months [31]. It is usually best for patients without significant pain and those without mechanical symptoms related to a loose chondral or osteochondral fragment [32]. Nonsteroidal anti-inflammatory medications, intra-articular corticosteroid injections, hyaluronic acid viscosupplementation, and platelet-rich plasma injection can be used as needed to relieve inflammation and pain.

Activity modification to avoid exacerbating symptoms and weight loss may also help [33]. In addition, strengthening of muscles crossing the knee through formal physical therapy helps absorb physiologic loads and may improve patient symptoms. In a study by Chiu et al., patellofemoral pain improved with isometric and isokinetic weight training focusing on quadriceps strengthening [34]. Quadriceps exercise is considered the cornerstone intervention for the management of patellofemoral pathologies given the intimate relationship between the patella within the quadriceps complex [35]. A variety of exercises have been advocated to address imbalances in the recruitment, timing, or general strength of the vastus medialis obliquus over the vastus lateralis [36].

Rest is also used to decrease knee pain symptoms. The goal is to eliminate activities that aggravate the patellofemoral joint.

Patellar taping during exercise has been reported to promote vastus medialis function through enhanced proprioceptive feedback and to decrease pain [37]. However, Gigante et al. suggested that taping does not medialize the patella and that a biomechanical mechanism for any change in symptoms is unclear [38]. A recent Cochrane review concluded that there was limited evidence to suggest that taping can significantly improve outcomes compared to other exercise-based interventions not incorporating taping [39].

Use of electric biofeedback systems in collaboration with VMO-specific training regimes has been introduced firstly to improve clinical outcome. After that, a large number of other electrotherapy modalities such as ultrasound, laser, interferential, and transcutaneous nerve stimulation have been described in the literature. Based on the current evidence, there remains inconclusive evidence that such electrotherapy modalities provide benefit when used in isolation [40].

There is limited evidence to support the use of immobilization or restriction of range of motion with knee orthoses or casting techniques, compared to permitting range of motion without a brace, on clinical outcomes or recurrent dislocation events [41]. Barton et al.'s systematic review [42] has reported limited pain, better outcome, and significantly improved function with the use of foot orthoses in addition to exercise.

Heintjes et al.'s Cochrane review [43] concluded that only nonsteroidal anti-inflammatory drugs (NSAIDs) demonstrated the effectiveness in the pharmacological management of short-term symptoms. Simple analgesics such as aspirin produced no significant differences in clinical symptoms and signs compared to a placebo.

10.7 Surgical Treatment

Surgical treatment is entertained when a patient has persistent and functionally limiting symptoms despite an adequate trial of nonoperative treatment. Surgical options depend on the lesion size, depth, location, and status of the underlying subchondral bone.

General indications for surgery include young and active patients with severe discomfort related to a deep focal chondral or osteochondral lesion. Lesions appropriate for surgical management are typically larger than 0.5 cm^2. Contraindications to surgery are obesity and chronic inflammatory diseases. Malalignment and instability can be treated at the time the chondral defect is addressed [44].

When operation is indicated, the choice lies between the correction of maltracking if this has been demonstrated, a local procedure to repair or improve the articular surface, and a biomechanical maneuver to reduce load on the patellofemoral joint. A combination of approaches may be appropriate [45].

Pure patellar tilt is a particular type of patellofemoral incongruence and is comparable to excessive lateral pressure syndrome first recognized by Ficat et al. [14]. In these cases a tight retinaculum places the lateral patellar facet under increased load leading to a patellar tilting without lateral subluxation or maltracking. The subchondral bone under the lateral facet is sclerotic, while the medial third of the patella is osteoporotic, leading to the so-named “one facet patella.” This is one of the few indications for lateral release alone.

Surgical procedures in the presence of an immature skeleton should involve the soft tissues only because of the risk of premature fusion and genu recurvatum. ln the Roux–Goldthwait operation, the patellar tendon is divided longitudinally; the lateral half is detached from the tibial tuberosity and transplanted medially. In the Galeazzi technique, the semitendinosus tendon is fixed to the patella to act as a tenodesis. In many cases these procedures cannot offer a permanent solution, and after growth is complete, further surgery is required.

10.7.1 Surgery for Maltracking

Several surgical techniques have been proposed in the treatment of patellar maltracking. They should be selected on the basis of his type and severity of malalignment [46]. Lateral subluxation of the patella can be treated with proximal or distal realignment.

In proximal realignment, quadriceps plasty described by Insall et al. [47] combines advancement of vastus medialis with a lateral release.

Medialization of the tibial tuberosity in the form of an Elmslie–Trillat procedure is used in patellar lateralization if the distance between the tibial tuberosity and the trochlear groove is more than 20 mm. Excessive medial transposition should be avoided since this can cause increased medial facet patellofemoral contact pressures in flexion.

Fulkerson’*s technique* is a modification of the Maquet procedure and consists of an anteromedialization of the tuberosity.

10.7.2 Chondral Surgery

Patellar shaving has not been found to be a rewarding procedure. Nevertheless, when there is severe fibrillation with effusion and small fragments of articular cartilage in the joint, it does seem reasonable to try to tidy up the articular surface.

Treatment approaches to repair patellar cartilage lesions have received less attention, probably due to the poor range of cartilage repair options available until not long ago. In particular, few studies have addressed the effectiveness of combined distal realignment associated with autologous chondrocyte implantation (ACI) in repairing isolated patellar cartilage lesions.

Different operative procedures are currently applied for articular cartilage repair, including microfractures, subchondral drilling, periosteal transplantation, mosaicplasty, and ACI [48–53]. First introduced in 1994 by Brittberg et al. [54], who used a periosteal flap, the ACI technique has

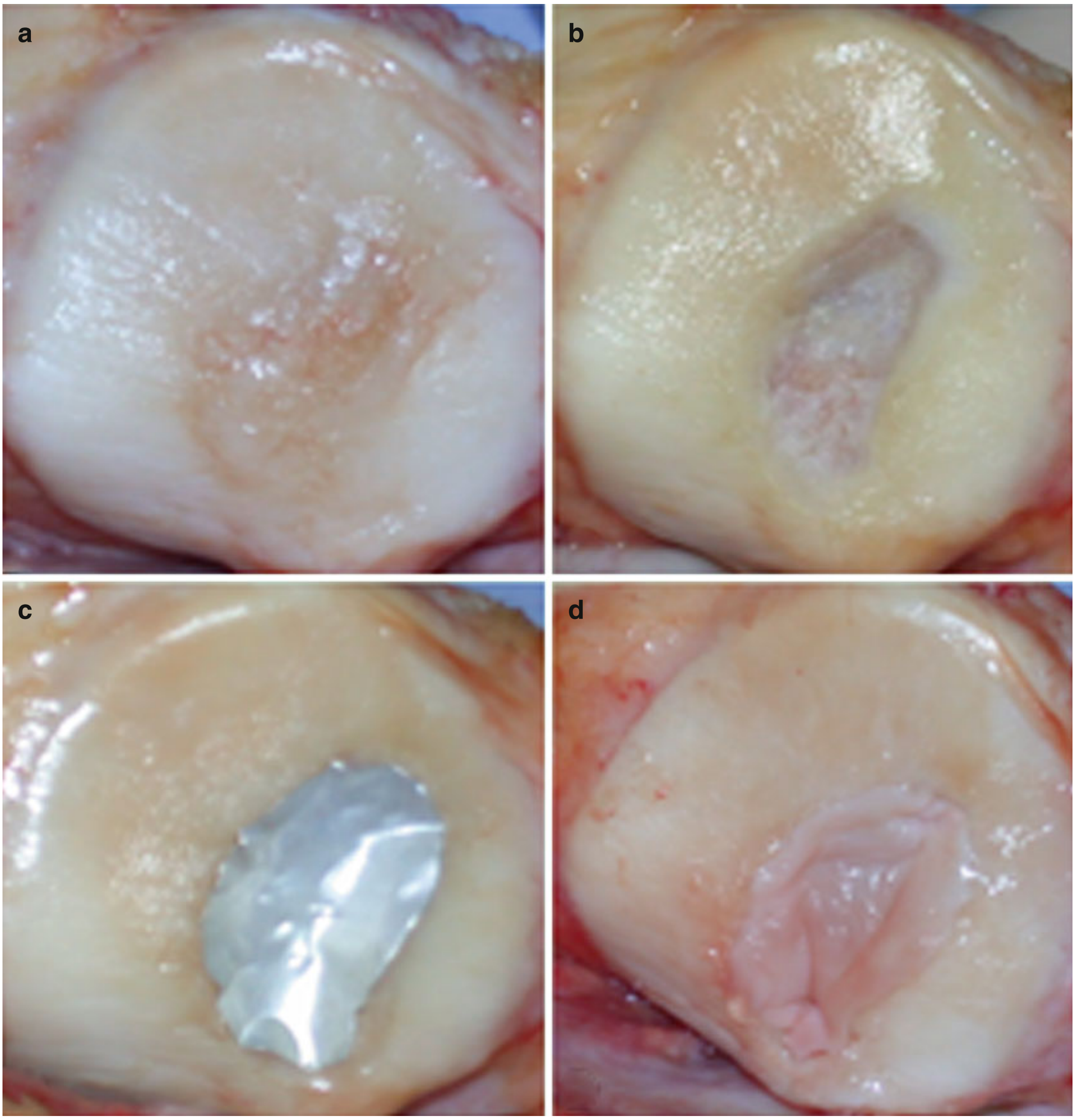

Fig. 10.2 Elliptical area of patellar chondromalacia (**a**) treated with curettage of the lesion (**b**), templating (**c**), and autologous chondrocyte implantation seeded on collagen membrane (MACI) (**d**)

evolved to include the utilization of chondrocytes grown on synthetic scaffolds, thus obviating the need for harvesting periosteum and affording a less invasive surgical wound. Some recent studies have failed to document significant differences between the clinical outcomes of first-generation ACI versus its variant using a collagen membrane as a scaffold (MACI) (Fig. 10.2) [55, 56].

Despite the good long-term clinical outcomes [57], both techniques involve two surgical steps, the high cost of cell culture and, for MACI, the cost of the membrane itself (Fig. 10.2). Their actual cost-effectiveness compared with more traditional techniques is therefore still unclear [52]. Bentley et al. have shown that ACI is far superior to mosaicplasty in repairing patellar cartilage lesions [25], while Knutsen et al. have found no significant differences in cartilage lesion repair between ACI and microfractures at 5 years [52]. However, none of the existing cartilage repair techniques have proved to be superior to the others in the long term, although there are no works comparing different techniques for the patella. Some studies [36, 45, 57–60] have considered the clinical outcomes of combined distal realignment and ACI/MACI.

A small number of recent studies have addressed the effectiveness of the ACI or similar techniques in treating patellofemoral joint cartilage defects, documenting variable and heterogeneous outcomes. Brittberg et al.'s early results achieved only 29 % good to excellent outcomes [54]. Lorentzon et al. reported 96 % good to excellent results in isolated patellar cartilage defects treated by periosteal transplantation, using a particular periosteal suture, associated with continuous passive motion in the immediate postoperative period. They did not use chondrocyte cultures and did not perform realignment [61]. Peterson and co-workers [57, 62] obtained 76 % good to excellent results at 10 years by associating, where necessary, an operative realignment of the extensor mechanism. Minas et al. [60]. described 71 % good to excellent results in a fairly heterogeneous population. Gobbi et al. [59], using a hyaluronic acid synthetic scaffold instead of the periosteal flap, obtained 90 % A and B results according to the IKDC knee ligament standard evaluation at 2 years. In a retrospective cohort study, Niemeyer et al. compared the outcomes of periosteal patch-covered conventional ACI, collagen membrane-covered ACI, and matrix-associated ACI in treating 70 patients with retropatellar cartilage defects. Patients with patellofemoral malalignment (assessed on axial knee x-rays) were excluded in order to obtain a homogeneous sample. The authors reported good to excellent result in approximately 70 % of patients and found larger lesions, lesions on the medial patellar facet and diffuse lesions to have a poorer prognosis than smaller lesions and lesions on the lateral patellar facet. The clinical outcomes of the three surgical techniques used were not significantly different [56]. In a recent study, Henderson et al. [63] compared two groups of 22 patients, one with patellofemoral malalignment and patellar cartilage defects and the other with patellar cartilage defects only. The two groups were treated with ACI combined with distal or proximal realignment or with ACI alone, respectively. The authors reported 86 % good to excellent outcomes in the first group versus 55 % in the second. They ascribed the difference to unloading due to the distal osteotomy or to incorrect preoperative evaluation of the extent of malalignment in the patients of the second group and concluded that patellofemoral joint unloading with a realignment procedure is desirable to maximize the clinical outcome of ACI, even when no tracking anomalies are identified clinically. Their reoperation rate was 52 % (10/22 and 13/22 patients, respectively). Farr [58] considered 38 patients with patellar and/or trochlear cartilage lesions, of whom 28 underwent distal realignment prior to or simultaneously with ACI. Thirteen of the 28 patients had isolated patellar cartilage lesions, but no data were provided as to the type of malalignment treated, and patellofemoral congruence was assessed only by a lateral radiograph and a Merchant axial view, without specification of the amount of subluxation. The two patients with a tilted and subluxated patella and those with no clear subluxation received patellar realignment followed by a rehabilitation program permitting earlier range of motion recovery compared with our patients; our rehabilitation protocol was more aggressive in allowing earlier full weight bearing. Overall, 25 of Farr's 38 patients required additional surgery, 18 for procedures other than screw removal. Nonetheless, their median modified Cincinnati scores ranged from 3 to 6 and the Lysholm score from 56 to 86.

The relative effectiveness of realignment and ACI in treating malalignment associated with patellar cartilage lesions is at present difficult to assess, due to the fact that the outcomes of ACI alone and of ACI with an extensor mechanism correction have often been reported together. In addition, the lack of a control group does not allow to establish whether one or the other procedure alone can

provide both pain relief and functional improvement, but ethical reasons suggest that both conditions should be treated. Gigante et al. focused on a small subgroup of patients with ante-

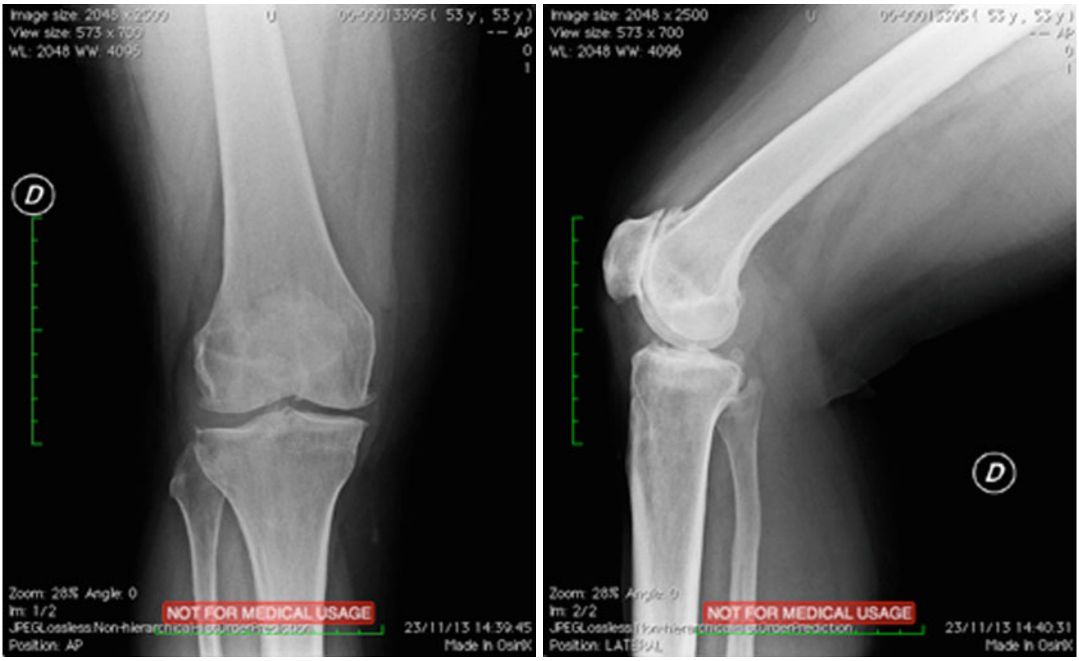

Fig. 10.3 Isolated symptomatic patellofemoral arthritis

rior knee pain and tilted and subluxated patella with TT–TS (20 mm on preoperative CT scans associated with large, isolated patellar cartilage lesions). In such cases the surgical approach should address both conditions, because transfer of the tibial tuberosity alone cannot solve the cartilage problem and could thus fail to provide pain relief [64]. On the other hand, it is disputed whether cartilage defects should be repaired without addressing the biomechanical imbalance [63]. A combination of MACI and transfer of the tibial tuberosity achieved good results in this selected group of patients, with significant increases in all outcome scales (Fig. 10.2).

AMIC plus autologous matrix-induced chondrogenesis (AMIC) combined with platelet-rich plasma gel is feasible for the treatment of symptomatic patellar cartilage defects and resulted in a clinical improvement in all patients [65].

10.7.3 Biomechanical Surgery

Maquet [66] described how anterior displacement of the tibial tubercle reduces the forces acting across the patellofemoral joint. Extreme advancement is no longer recommended, but elements of the principle of Maquet are incorporated into the Fulkerson anteromedial realignment as well as the Elmslie–Trillat procedure which invariably leaves a slightly elevated tubercle.

10.7.4 Patellofemoral Arthroplasty

Some authors showed that among symptomatic knees, isolated radiographic patellofemoral arthritis was present in 21 % of patients older than 60 years. Therefore, isolated symptomatic patellofemoral arthritis, which was considered rare in the past, is probably frequent in elderly patients (Fig. 10.3). However, the number of cases in which isolated patellofemoral arthroplasty is indicated remains small, probably about 10 % of the cases that come to any form of knee replacement.

Patellofemoral arthroplasty is a treatment option for isolated arthritis in the patellofemoral joint, and, compared to total knee arthroplasty, it has a number of advantages. Patellofemoral arthroplasty preserves the structural integrity of the tibiofemoral joint by sparing the condylar surfaces, menisci, and cruciate ligaments. It is assumed to be a limited procedure with less dissection, less blood loss, and more rapid recovery after surgery. However, the procedure did not gain widespread use because of initial design limitations [67].

Over the past decade, second-generation PFAs incorporated changes in implant design and instrumentation and have shown promising results when used in the properly selected patient population. Despite the successful, durable results, concern remains for using TKA in patients with isolated PF OA, as TKA requires a more extensive surgical exposure and bone resection, a longer recovery time, and a potentially more complex revision than that required for a patient with a failed PFA. It is controversial as to whether PFA or TKA should be used to treat patients with isolated PF OA [68]. The quality of evidence supporting both procedures has been objectively classified in a recent systematic review as low quality. Most of the published studies are retrospective, non-comparative, or nonrandomized and report on a small number of patients.

10.7.5 Ablative Surgery

Patellectomy is indicated when the articular surface of the patella is severely damaged over a wide area and the rest of the knee looks normal. After patellectomy, patients can expect little problem in day-to-day living, but sporting activities will likely be curtailed. Even with vigorous rehabilitation, some wasting of the quadriceps will remain, and various authors have estimated that extensor power will never be better than 70 % of normal. The complications include rupture or persistent subluxation of the quadriceps mechanism and pain after the procedure.

10.8 Take-Home Messages

1. Chondral lesions of the patellofemoral joint are frequent and are often related to patellofemoral maltracking.
2. CT and MRI can well define, respectively, malalignment and chondral lesions.
3. Conservative treatment should be considered in the management of pain and function in early cartilage lesions and not severe maltracking.
4. Surgical options should be selected on the type and severity of malalignment.
5. Surgical treatments should correct patellar maltracking and at the same time repair cartilage defects.

References

1. Mandelbaum BR, Browne JE, Fu F, Micheli L, Mosely Jr JB, Erggelet C, Minas T, Peterson L. Articular cartilage lesions of the knee. Am J Sports Med. 1998;26(6):853–61.
2. Fox AJSA, Wanivenhaus FF, Rodeo SAS. The basic science of the patella: structure, composition, and function. J Knee Surg. 2012;25(2):127–41.
3. Tecklenburg KK, Dejour DD, Hoser CC, et al. Bony and cartilaginous anatomy of the patellofemoral joint. Knee Surg Sports Traumatol Arthrosc. 2006;14(3):235–40.
4. Stäubli HUH, Dürrenmatt UU, Porcellini BB, et al. Anatomy and surface geometry of the patellofemoral joint in the axial plane. J Bone Joint Surg Br. 1999;81(3):452–8.
5. Zwerver JJ, Bredeweg SWS, Hof ALA. Biomechanical analysis of the single-leg decline squat. CORD Conf Proc. 2007;41(4):264–8.
6. Stephen JM, Kader D, Lumpaopong P, Deehan DJ, Amis AA. Sectioning the medial patellofemoral ligament alters patellofemoral joint kinematics and contact mechanics. J Orthop Res. 2013;31(9):1423–9.
7. Widuchowski WW, Widuchowski JJ, Trzaska TT. Articular cartilage defects: study of 25,124 knee arthroscopies. Knee. 2007;14(3):177–82.
8. Aglietti P, Giron F, Cuomo P. Insall & Scott surgery of the knee. 4th ed. Philadelphia: Churchill Livingstone; 2006. p. 848.
9. Arendt EA, Fithian DC, Cohen E. Current concepts of lateral patella dislocation. Clin Sports Med. 2002;21(3):499–519.
10. Dejour H, Walch G, Nove-Josserand L, Guier C. Factors of patellar instability: an anatomic radiographic study. Knee Surg Sports Traumatol Arthrosc. 1994;2(1):19–26.
11. Desio SM, Burks RT, Bachus KN. Soft tissue restraints to lateral patellar translation in the human knee. Am J Sports Med. 1998;26(1):59–65.
12. Fithian DC, Paxton EW, Cohen AB. Indications in the treatment of patellar instability. J Knee Surg. 2004;17(1):47–56.

13. Peterson L, Karlsson J, Brittberg M. Patellar instability with recurrent dislocation due to patellofemoral dysplasia. Results after surgical treatment. Bull Hosp Jt Dis Orthop Inst. 1988;48(2):130–9.
14. Ficat P, Ficat C, Bailleux A. Syndrome d'hyperpression externe de la rotule (SHPE): Son interet pour la connaissance del l'arthrouse. Rev Chir Orthop. 1975; 61:39.
15. Outerbridge RE, Dunlop JA. The problem of chondromalacia patellae. Clin Orthop Relat Res. 1975;110: 177–96.
16. Goodfellow J, Hungerford DS, Woods C. Patello-femoral joint mechanics and pathology. 2. Chondromalacia patellae. J Bone Joint Surg Br. 1976;58(3): 291–9.
17. Bentley G. Articular cartilage changes in chondromalacia patellae. J Bone Joint Surg Br. 1985;67(5):769–74.
18. Insall JJ, Falvo KAK, Wise DWD. Chondromalacia patellae. A prospective study. J Bone Joint Surg Am. 1976;58(1):1–8.
19. Curl WW, Krome J, Gordon ES, et al. Cartilage injuries: a review of 31,516 knee arthroscopies. Arthroscopy. 1997;13(4):456–60.
20. Hjelle K, Solheim E, Strand T, et al. Articular cartilage defects in 1,000 knee arthroscopies. Arthroscopy. 2002;18(7):730–4.
21. Strauss EJ, Galos DK. The evaluation and management of cartilage lesions affecting the patellofemoral joint. Curr Rev Musculoskelet Med. 2013;6:141–9.
22. Gallo RA, Feeley BT. Cartilage defects of the femoral trochlea. Knee Surg Sports Traumatol Arthrosc. 2009;17(11):1316–25.
23. Post WR. Clinical evaluation of patients with patellofemoral disorders. Arthroscopy. 1999;15(8):841–51.
24. Merchant AC. Classification of patello femoral disorders. Arthroscopy. 1988;4:235.
25. Dejour H, Walch G, Neyret P, Adeline P. La dysplasie de la trochle´e femorale. Rev Chir Orthop Reparatrice Appar Mot. 1990;96:45.
26. Schutzer SF, Ramsby GR, Fulkerson JP. The evaluation of patellofemoral pain using computerized tomography. A preliminary study. Clin Orthop Relat Res. 1986;204:286–93.
27. Gigante A, Bevilacqua C, Bonetti MG, Greco F. Increased external tibial torsion in Osgood-Schlatter disease. Acta Orthop Scand. 2003;74(4):431–6.
28. Guzzanti V, Gigante A, Di Lazzaro A, Fabbriciani C. Patellofemoral malalignment in adolescents. Computerized tomographic assessment with or without quadriceps contraction. Am J Sports Med. 1994;22(1):55–60.
29. Schoettle PB, Zanetti M, Seifert B, et al. The tibial tuberosity– trochlear groove distance; a comparative study between CT and MRI scanning. Knee. 2006; 13(1):26–31.
30. Smith TO, Davies L, O'Driscoll M-L, et al. An evaluation of the clinical tests and outcome measures used to assess patellar instability. Knee. 2008;15(4):255–62.
31. Kramer K. Management of patellar and trochlear chondral injuries. Oper Tech Orthop. 2007;17(4):10.
32. Post WR. Patellofemoral pain: results of non operative treatment. Clin Orthop Relat Res. 2005;436:55–9.
33. Strauss EJE, Fonseca LEL, Shah MRM, et al. Management of focal cartilage defects in the knee—is ACI the answer? CORD Conf Proc. 2011;69(1): 63–72.
34. Chiu JKWJ, Wong Y-MY, Yung PSHP, et al. The effects of quadriceps strengthening on pain, function, and patellofemoral joint contact area in persons with patellofemoral pain. Am J Phys Med Rehabil. 2012;91(2):98–106.
35. Bolgla LA, Boling MC. An update for the conservative management of patellofemoral pain syndrome: a systematic review of the literature from 2000 to 2010. Int J Sports Phys Ther. 2011;6:112–25.
36. Chester R, Smith TO, Sweeting D, Dixon J, Wood S, Song F. The relative timing of VMO and VL in the aetiology of anterior knee pain: a systematic review and meta-analysis. BMC Musculoskelet Disord. 2008;9:64.
37. Gilleard W, McConnell J, Parsons D. The effect of patellar taping on the onset of vastus medialis obliquus and vastus lateralis muscle activity in persons with patellofemoral pain. Phys Ther. 1998;78:25–32.
38. Gigante A, Pasquinelli FM, Paladini P, Ulisse S, Greco F. The effects of patellar taping on patellofemoral incongruence. A computed tomography study. Am J Sports Med. 2001;29:88–92.
39. Callaghan MJ, Selfe J. Patellar taping for patellofemoral pain syndrome in adults. Cochrane Database Syst Rev. 2012;(4):CD006717.
40. Lake DA, Wofford NH. Effect of therapeutic modalities on patients with patellofemoral pain syndrome: a systematic review. Sports Health. 2011;3:182–9.
41. Smith TO, Davies L, Donell ST. Immobilization regime following lateral patellar dislocation: a systematic review and meta-analysis of the current evidence base. Eur J Trauma Emerg Surg. 2010;36:353–60.
42. Barton CJ, Munteanu SE, Menz HB, Crossley KM. The efficacy of foot orthoses in the treatment of individuals with patellofemoral pain syndrome: a systematic review. Sports Med. 2010;40:377–95.
43. Heintjes E, Berger MY, Bierma-Zeinstra SMA, Bernsen RMD, Verhaar JAN, Koes BW. Pharmacotherapy for patellofemoral pain syndrome. Cochrane Database Syst Rev. 2004;(3):CD003470.
44. Versier G, Dubrana F, French Arthroscopy Society. Treatment of knee cartilage defect in 2010. Orthop Traumatol Surg Res. 2011;97(8 Suppl):S140–53.
45. Gigante A, Enea D, Greco F, Bait C, Denti M, Schonhuber H, Volpi P. Distal realignment and patellar autologous chondrocyte implantation: mid-term results in a selected population. Knee Surg Sports Traumatol Arthrosc. 2009;17(1):2–10.
46. Fulkerson JP, Shea KP. Disorders of patellofemoral alignment. J Bone Joint Surg Am. 1990;72(9):1424–9.
47. Insall J, Bullough PG, Burstein AH. Proximal "tube" realignment of the patella for chondromalacia patellae. Clin Orthop Relat Res. 1979;144:63–9.

48. Bentley G, Biant LC, Carrington RW, Akmal M, Goldberg A, Williams AM, Skinner JA, Pringle J. A prospective, randomized comparison of autologous chondrocyte implantation versus mosaicplasty for osteochondral defects in the knee. J Bone Joint Surg Br. 2003;85(2):223–30.
49. Gobbi A, Nunag P, Malinowski K. Treatment of full thickness chondral lesions of the knee with microfracture in a group of athletes. Knee Surg Sports Traumatol Arthrosc. 2005;13(3):213–21.
50. Hangody L, Kish G, Ka´rpa´ti Z, Udvarhelyi I, Szigeti I, Be´ly M. Mosaicplasty for the treatment of articular cartilage defects: application in clinical practice. Orthopedics. 1998;21(7):751–6.
51. Knutsen G, Engebretsen L, Ludvigsen TC, Drogset JO, Grøntvedt T, Solheim E, Strand T, Roberts S, Isaksen V, Johansen O. Autologous chondrocyte implantation compared with microfracture in the knee. A randomized trial. J Bone Joint Surg Am. 2004;86-A(3):455–64.
52. Knutsen G, Drogset JO, Engebretsen L, Grøntvedt T, Isaksen V, Ludvigsen TC, Roberts S, Solheim E, Strand T, Johansen O. A randomized trial comparing autologous chondrocyte implantation with microfracture. Findings at five years. J Bone Joint Surg Am. 2007;89:2105–12.
53. Mc Ginley BJ, Cushner FD, Scott WN. Debridement arthroscopy. 10-year followup. Clin Orthop Relat Res. 1999;367:190–4.
54. Brittberg M, Lindahl A, Nilsson A, Ohlsson C, Isaksson O, Peterson L. Treatment of full-thickness cartilage defects in the human knee with cultured autologous chondrocytes. N Engl J Med. 1994;331:889–95.
55. Bartlett W, Skinner JA, Gooding CR, Carrington RW, Flanagan AM, Briggs TW, Bentley G. Autologous chondrocyte implantation versus matrix-induced autologous chondrocyte implantation for osteochondral defects of the knee: a prospective, randomised study. J Bone Joint Surg Br. 2005;87(5):640–5.
56. Niemeyer P, Steinwachs M, Erggelet C, Kreuz PC, Kraft N, Köstler W, Mehlhorn A, Südkamp NP. Autologous chondrocyte implantation for the treatment of retropatellar cartilage defects: clinical results referred to defect localisation. Arch Orthop Trauma Surg. 2008;128:1223–31.
57. Peterson L, Minas T, Brittberg M, et al. Two- to 9-year outcome after autologous chondrocyte transplantation of the knee. Clin Orthop Relat Res. 2000;374:212–34.
58. Farr J. Autologous chondrocyte implantation improves patellofemoral cartilage treatment outcomes. Clin Orthop Relat Res. 2007;463:187–94.
59. Gobbi A, Kon E, Berruto M, Francisco R, Filardo G, Marcacci M. Patellofemoral full-thickness chondral defects treated with Hyalograft-C: a clinical, arthroscopic, and histologic review. Am J Sports Med. 2006;34(11):1763–73.
60. Minas T, Bryant T. The role of autologous chondrocyte implantation in the patellofemoral joint. Clin Orthop Relat Res. 2005;436:30–9.
61. Lorentzon R, Alfredson H, Hildingsson C. Treatment of deep cartilage defects of the patella with periosteal transplantation. Knee Surg Sports Traumatol Arthrosc. 1998;6(4):202–8.
62. Peterson L, et al. Two- to 9-year outcome after autologous chondrocyte transplantation of the knee. Clin Orthop. 2000;374:212–34.
63. Henderson IJ, Lavigne P. Periosteal autologous chondrocyte implantation for patellar chondral defect in patients with normal and abnormal patellar tracking. Knee. 2006;13(4):274–9.
64. Pidoriano AJ, Weinstein RN, Buuck DA, Fulkerson JP. Correlation of patellar articular lesions with results from anteromedial tibial tubercle transfer. Am J Sports Med. 1997;25(4):533–7.
65. Dhollander AA, De Neve F, Almqvist KF, Verdonk R, Lambrecht S, Elewaut D, Verbruggen G, Verdonk PC. Autologous matrix-induced chondrogenesis combined with platelet-rich plasma gel: technical description and a five pilot patients report. Knee Surg Sports Traumatol Arthrosc. 2011;19(4):536–42.
66. Maquet P. Advancement of the tibial tuberosity. Clin Orthop Relat Res. 1976;115:225–30.
67. Baker PN, Refaie R, Gregg P, Deehan D. Revision following patello-femoral arthroplasty. Knee Surg Sports Traumatol Arthrosc. 2012;20:2047–53.
68. Dy J, Franco N, Ma y, Mazumbar M, McCarthy MM, Della Valle AG. Complications after patello-femoral versus total knee replacement of isolated patellofemoral osteoarthritis. A meta-analysis. Knee Surg Sports Traumatol Arthrosc. 2012;20:2174–90.

ICL 16: Subchondral Bone and Reason for Surgery

11

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S. Zaffagnini et al. (eds.), *ESSKA Instructional Course Lecture Book*,
DOI 10.1007/978-3-642-53983-1_11, © ESSKA 2014

Table 11.1 Presentations from the Instructional Course Lecture "Subchondral bone and reason for surgery" included in this book chapter

Speaker	City	Country	Title of presentation
Deepak Goyal	Ahmedabad	India	Subchondral bone: healthy soil for the healthy plant
Henning Madry	Homburg/Saar	Germany	Pathological alterations of the subchondral bone in cartilage repair
Jacques Menetrey	Geneva	Switzerland	Epidemiology and imaging of the subchondral bone
Elisaveta Kon	Bologna	Italy	Biomimetic scaffolds for osteochondral regeneration in the knee joint
C. Niek van Dijk	Amsterdam	The Netherlands	How to treat subchondral bone pathologies in the ankle joint

11.1 Introduction

The subchondral bone plays a key role in the integrity and repair of the entire osteochondral unit. Its unique anatomical structure is well suited to support the articular cartilage, even providing nutrition for its basal layers. The subchondral bone becomes a problem in reconstructive cartilage surgery when violated, for example, following osteochondral fractures or in osteochondritis dissecans (OCD), thus leading to an osteochondral lesion. Such osteochondral defects are difficult to treat because the subchondral bone and the articular cartilage have very dissimilar intrinsic healing capacities [35–37]. Recent clinical evidence also pointed to the role of the subchondral bone alterations in the course of spontaneous and surgical cartilage repair [24]. In the treatment of cartilage defects, it is imperative to establish the etiology of the subchondral bone lesion and then address the specific pathology accordingly. This chapter will focus on the subchondral bone and its relevance for reconstructive cartilage surgery. It is based on individual presentations from the Instructional Course Lecture "Subchondral bone and reason for surgery" (Table 11.1).

11.2 Pathological Alterations of the Subchondral Bone in Cartilage Repair

The osteochondral unit is composed of the articular cartilage and the underlying subchondral bone [56]. The subchondral bone is formed by the subchondral bone plate and the subarticular spongiosa (Fig. 11.1). The line separating the hyaline articular cartilage from the calcified cartilage is called the tidemark, while the cement line divides the calcified cartilage from the subchondral bone plate [56]. This connection is of major importance for maintaining the osteochondral integration. Here, no collagen fibrils extend from the calcified cartilage (which contains mainly type II and X collagen) into the subchondral bone plate (which contains mainly type I collagen). From a clinical standpoint, Drobnic et al. have shown that when the hyaline articular cartilage and calcified cartilage layer are surgically removed—as mandatory prior to applying marrow stimulation techniques and autologous chondrocyte implantation—these small canals are opened, at least to some extent, allowing the influx of marrow elements from the subchondral bone into the cartilage defect [19]. This junction is also a site of active remodeling. A high number of arteries, veins, and nerves send small branches through canals in the subchondral bone plate into the calcified cartilage [56]. This plexus follows the undulations of the subchondral–cartilage border and reaches the calcified cartilage, eventually perforating it. Nutrients can therefore reach chondrocytes in the calcified and hyaline articular cartilage [72]. Of note, the anatomy of the subchondral region is highly variable and depends on the location within a joint surface and therefore on the local mechanical environment. Studies of subchondral bone mineralization have also shown that changes in stress distribution are followed by changes in morphology [21]. Thus, the osteochondral connection is not impermeable, but a structural and functional unit that is an active site of remodeling allowing both mechanical and biochemical interactions [56].

The integrity of the subchondral bone can be disrupted in traumatic osteochondral defects,

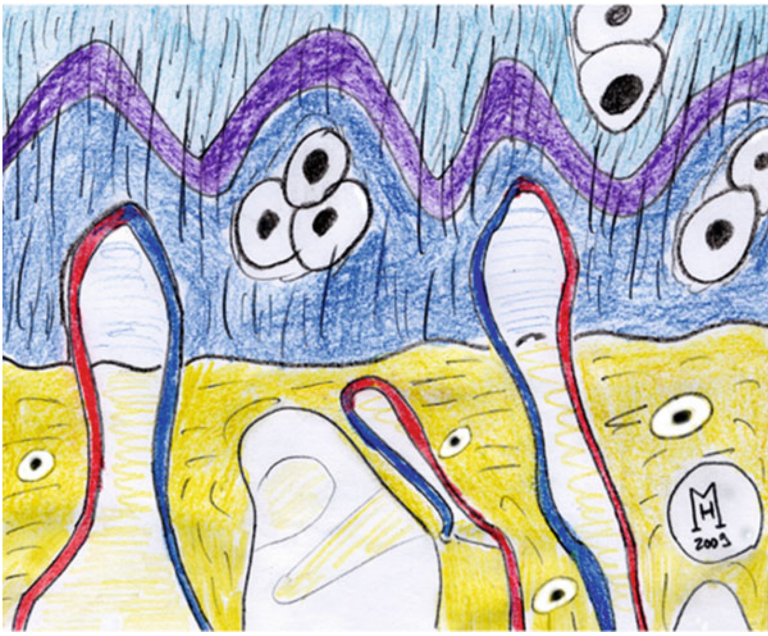

Fig. 11.1 Schematic drawing of the calcified cartilage layer and the subchondral bone plate with articular vascular plexus vessels (From Madry et al. [56], with permission). The tidemark is crossed by collagen fibrils that extend from the hyaline articular cartilage into the calcified cartilage. No collagen fibrils connect the calcified cartilage to the subchondral bone plate. Blood vessels from the subchondral region can extend into the overlying calcified cartilage through canals in the subchondral bone plate

OCD, ON, and OA. Osteochondral defects primarily arise from the subchondral bone in the case of OCD and osteonecrosis (ON). They secondarily extend into the subchondral bone as a result of osteochondral fracture and large degenerative osteoarthritic (OA) cartilage lesions. Even focal chondral defects have been shown to increase the mechanical stress on the edge of the lesion and grow in size over time, producing concomitant changes of the underlying subchondral bone structure [73]. The healing of an osteochondral defect in terms of subchondral bone plate restoration is only incompletely understood [31, 56, 70, 73]. Besides constituting the new cartilaginous repair tissue in the surface layer, mesenchymal cells coming from the bone marrow can also differentiate into osteocytes in the deeper area of the lesion [39, 86].

11.2.1 Pathological Alterations of the Subchondral Bone

Very recently, complex structural changes of the subchondral bone have been found to be associated either with spontaneous osteochondral repair or with the use of different cartilage repair procedures. In general, they can be categorized as four different pathological alterations (Fig. 11.2) [70]. Such subchondral bone changes include (1) the upward migration of the subchondral bone plate, (2) the formation of intralesional osteophytes, (3) the appearance of subchondral bone cysts, and (4) the generalized impairment of the subchondral bone microarchitecture below an articular cartilage defect (Table 11.2).

The upward migration of the subchondral bone plate (Fig. 11.3) is defined as the expansion of the osteochondral junction above its original level with the elevation of the subchondral bone plate into the cartilaginous repair tissue as a result [69]. Henderson and La Valette found an upward migration of the subchondral bone plate in 34 % of chondral defects at 3 years after ACI. Interestingly, there was a significant association between upward migration of the subchondral bone plate and a larger defect size [103].

Intralesional osteophytes are defined as focal, newly formed bone located apical to the original cement line and projected into the cartilaginous

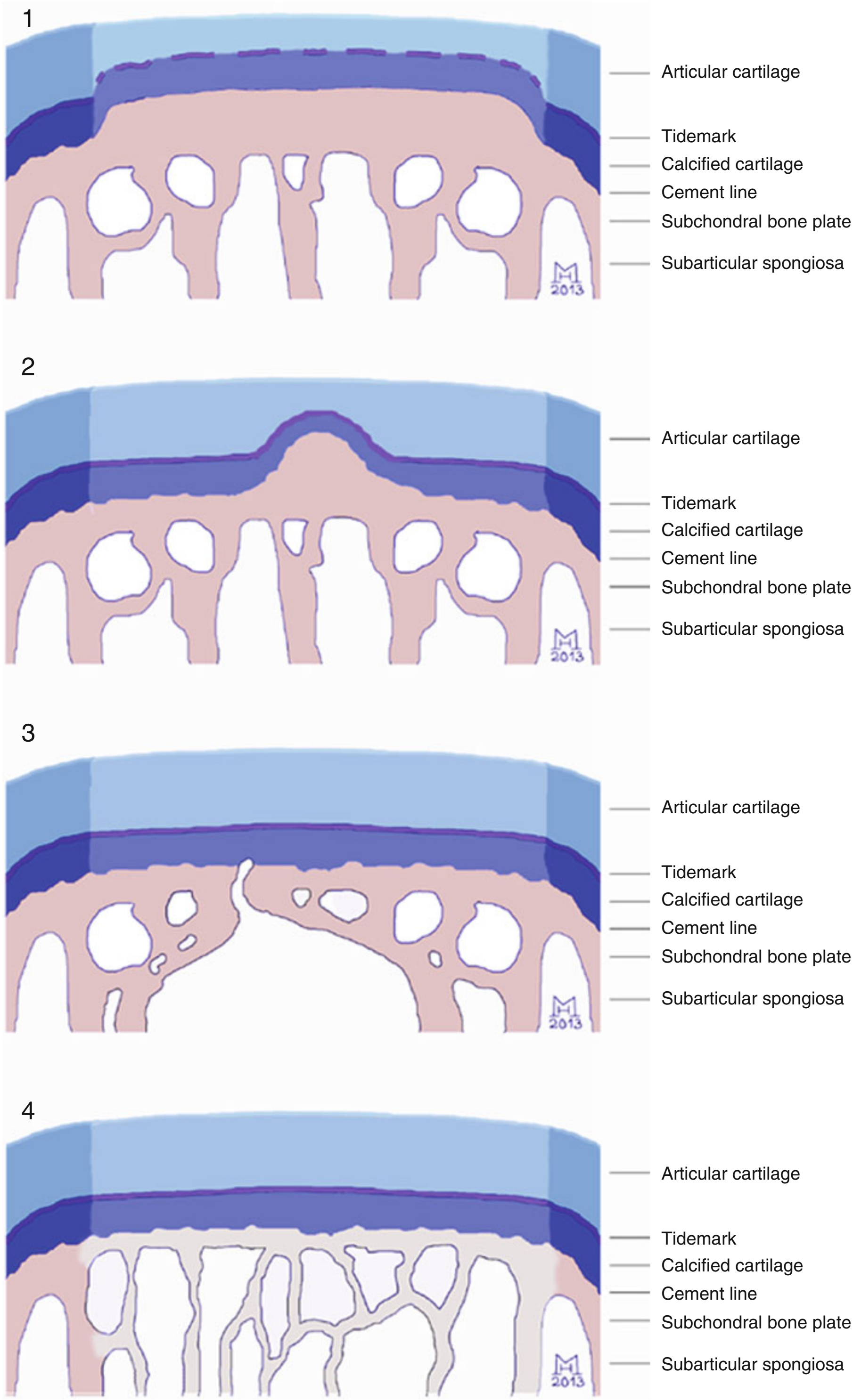
1
Articular cartilage
Tidemark
Calcified cartilage
Cement line
Subchondral bone plate
Subarticular spongiosa
2
Articular cartilage
Tidemark
Calcified cartilage
Cement line
Subchondral bone plate
Subarticular spongiosa
3
Articular cartilage
Tidemark
Calcified cartilage
Cement line
Subchondral bone plate
Subarticular spongiosa
4
Articular cartilage
Tidemark
Calcified cartilage
Cement line
Subchondral bone plate
Subarticular spongiosa

Table 11.2 Clinical evidence of subchondral bone alterations following the use of cartilage repair procedures

Pathology	Surgical index procedure	Detection method	Follow-up (months)	Number of patients	Incidence per defect (%)	Reference
Upward migration	Microfracture	MRI	12	24	25.0	[66]
	Microfracture	MRI	36	44	52.0	[82]
	ACI	MRI	36	41	25.0	[82]
	ACI	MRI	36	179	33.5	[33]
Intralesional osteophyte	Drilling (type I/type III collagen, PRP gel)	MRI	24	5	60.0	[17, 18]
	Microfracture	MRI	36	70	27.1	[52]
	Microfracture	MRI	15	80	48.8	[8]
	Microfracture	MRI	0.8	13	0.0	[11]
			6	13	53.8	
			12	13	69.2	
			24	8	75.0	
	ACI	MRI	13	34	23.3	[8]
	ACI	MRI	155	31	63.9	[96]
Cyst	Microfracture	MRI	0.8	13	0.0	[11]
			6	13	15.4	
			12	13	38.5	
			24	8	37.5	
	ACI	MRI	155	31	38.8	[96]

MRI magnetic resonance imaging, *ACI* autologous chondrocyte implantation, *PRP* platelet-rich plasma

repair tissue layer [11, 71]. Cole et al. observed intralesional osteophytes in 75 % at 2 years after microfracture. Intralesional osteophytes were also detected at a mean follow-up of 13 years after ACI in 64 % of cartilage defects [96].

Subchondral bone cysts, an entity usually related to late-stage OA [104], have been reported in conjunction with marrow stimulation procedures. Following microfracture subchondral cysts were seen by MRI in 38 % of defects at 2 years postoperatively [11] and in 39 % of defects a mean follow-up of 13 years after ACI [96].

Significant alterations of the microarchitecture of the subchondral bone have been discovered in preclinical large animal models [71]. Here, significantly reduced bone volume, trabecular thickness, and bone mineral density—consistent with patterns observed in osteoporosis and OA—were

Fig. 11.2 Summary of the complex alterations of the subchondral bone in the course of osteochondral repair (repair tissue can be identified by its less intense color). These alterations occur sporadically and are not inevitable consequences of subchondral bone plate perforation. They can be categorized as (*1*) generalized upward migration of the subchondral bone plate, (*2*) formation of focal intralesional osteophytes, (*3*) emergence of subchondral bone cysts, and (*4*) impairment of the microarchitecture of the subchondral bone. The upward migration of the subchondral bone plate (*1*) leads to a consecutive thinning of the articular cartilage layer and to an extension of the subchondral bone plate volume into the cartilaginous repair tissue. Intralesional osteophytes (*2*) are defined as focal newly formed bone located apical to the original cement line. An intralesional osteophyte can be either located in a central or peripheral location within the articular cartilage defect. The subchondral bone cyst (*3*) has its largest expansion within the subarticular spongiosa and is surrounded by a sclerotic rim. Changes occur also in the entire microarchitecture of the subchondral bone (*4*), for example, a generalized thinning of the subchondral bone plate, a reduced trabecular thickness, and an overall decreased subchondral bone volume

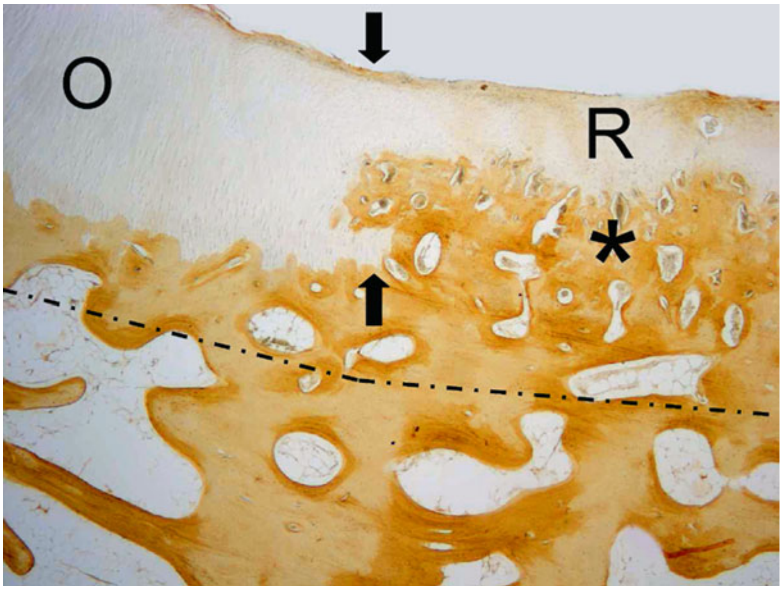

Fig. 11.3 An upward migration of the subchondral bone plate leads to an extension of the subchondral bone plate volume into the cartilaginous repair tissue and to a consecutive thinning of the articular cartilage layer. Note the relative decreased thickness of the repair cartilage (*R*) in relation to the original adjacent articular cartilage (*O*). The *two arrows* show the vertical cartilaginous integration zone, the subchondral bone plate advancement is denoted with an *asterisk*, the bone above the *dotted line* is the subchondral bone plate, and the bone below is the subarticular spongiosa. Sheep trochlea, anti-type-I-collagen immunohistochemistry. Magnification ×4

reported after 6 months. No clinical investigation has examined the microarchitecture of the subchondral bone below articular cartilage defects so far. Imaging methods that are accurate for the study of cartilage lesions and cartilage loss also need to provide information on the subchondral bone and marrow changes.

11.3 Epidemiology and Imaging of the Subchondral Bone

Few epidemiological data exists regarding the epidemiology of defects extending into the subchondral bone, in particular as they relate to diseases such as traumatic osteochondral defects, OCD, ON, and OA. Imaging of the subchondral bone needs to fit into the concept of the functional cartilage–subchondral bone unit as articular cartilage, and bone health appears to be tightly associated [38]. Ample evidence is found for bone changes during progression of OA, including, but not limited to, increased turnover in the subchondral bone; thinning of the trabecular structure, osteophytes, and bone marrow lesions; and sclerosis of the subchondral bone plate.

11.3.1 Location of Osteochondral Lesions

The preferential site for focal chondral or osteochondral lesions was the medial femoral condyle (58 %) in a study encompassing 1,000 knee arthroscopies [34]. The remaining lesions were situated on the patella (11 %), the lateral tibia (11 %), the lateral femoral condyle (9 %), the trochlea (6 %), and the medial tibia (5 %). Those findings are consistent with the observations made by Curl et al. [14], who showed that the most common locations for full-thickness chondral lesions with exposed bone (grade IV lesions based on a modified Outerbridge scale) were the medial femoral condyle (about 32 % of patients), the patella (about 21 % of patients), and the lateral femoral condyle (about 20 % of patients). Less than 5 % of all patients had grade IV lesions in the medial and lateral tibial plateau.

11.3.2 Size and Numbers of the Lesions

Regarding the size of the lesion, the mean chondral or osteochondral defect area was 2.1 cm^2 (range, 0.5–12; SD, 1.5) [34]. Of all chondral or osteochondral defects, 88 % were less than 4 cm^2 (19 % of the defects were less than 1 cm^2; 26 % ranged from 1 to 2 cm^2; 42 % ranged from 2 to 4 cm^2; and 12 % were more than 4 cm^2). An average of 2.7 lesions per knee was reported by Curl et al. [14]. According to Hjelle et al. [34], when narrowing down those lesions to focal chondral lesions only (as opposed to OA, chondromalacia patella OCD), 80 % of the injuries were unique, 12 % were double, and 8 % were triple.

11.3.3 Prevalence

It is difficult, if not impossible, to establish who, in the general population, suffers from chondral lesions of the knee. An unknown number of people who sustain articular surface injuries will never develop symptoms or seek medical treatment [63].

In a study of 200 arthroscopies performed on symptomatic patients, Zamber et al. [101] found that 62 % of them presented with at least one cartilage lesion. The retrospective study by Curl et al. [14] reviewed 31,516 knee arthroscopies. Chondral lesions were found in 63 % arthroscopies. The prospective study of 1,000 arthroscopies of the knee conducted by Hjelle et al. [34] has also considerably contributed to mapping the chondral pathology of the knee. In the collective of 1,000 patients requiring knee arthroscopy for various reasons, 61 % of them revealed chondral or osteochondral lesions, of which 44 % were OA, 28 % focal chondral lesions, 23 % chondromalacia patellae, 2 % OCD, and 3 % others. Curl et al. [14] identified 1 % lesions caused by OCD, 1 % by articular fractures, 10 % by grade I chondromalacia, 28 % by grade II chondromalacia, 41 % by grade III chondromalacia, and 19 % by full-thickness defects with the exposed subchondral bone. Grade III lesions (partial-thickness chondral lesions with deep fissures) were the most common lesions in patients over 30 years of age. Focal chondral or osteochondral lesions were found in 19 % of the arthroscopies.

11.3.4 Patient Age

Zamber et al. reported that 76 % of patients suffering from chondral lesions were older than 30 years [67, 101]. According to Curl et al. [14], the average age of the patients with lesions was 43 years, predominantly in male patients (62 vs. 38 % for female patients). Overall, the majority (72 %) of grade IV lesions was found in patients over 40 years of age. Patients under 40 years of age with grade IV lesions accounted for 5 % of all arthroscopies.

11.3.5 Clinical Imaging of the Subchondral Bone

The subchondral bone cannot readily be evaluated during arthroscopy. Different imaging techniques are available, and MRI is actually considered the most accurate method for the evaluation of the cartilage and underlying subchondral bone.

Radiography still is the golden standard for imaging features of OA. Objective assessment, however, of osteoarthritis features such as joint space narrowing, subchondral sclerosis, and osteophyte formation is highly important. Therefore, radiographic acquisition needs to be standardized: apart from a standard lateral knee view, a posteroanterior, weight-bearing, fixed flexion radiography with 10° caudal beam angulation needs to be acquired [23, 51].

Bone scintigraphy using diphosphonate derivatives radiolabeled with Tc-99m targets the bone response that results from the abnormal biomechanics of joint motion when the articular cartilage is damaged and has therefore a high sensitivity in detecting bone reaction to the pathology of OA [64].

Both computed tomography (CT) and CT arthrography can show subchondral bone changes, such as subchondral bone sclerosis and osteophytes. Both techniques can show central osteophytes, associated with more severe changes of OA than marginal osteophytes [61]. CT arthrography, performed after direct intra-articular injection of iodine contrast, is the most accurate method for the evaluation of cartilage thickness and cartilage defects, thanks to its high spatial resolution and high contrast ratio [22]. However, purely intrachondral lesions, without communication with the surface, cannot be detected [78]. CT scan is a valuable technique to evaluate suspected spontaneous ON about the knee (differentiation with adult onset OCD; eventual depression of the subchondral bone plate and appearance of intra-articular loose bodies). CT arthrography is highly accurate to evaluate the stability of the osteochondral fragment in OCD. Major drawback for CT is the exposition of the patient to ionizing radiation and the need for invasive intra-articular punction to perform a CT arthrography examination.

Magnetic resonance imaging is actually considered the most accurate method for the evaluation of the cartilage and subchondral bone. Cartilage-specific sequences, such as intermediate-weighted FSE and especially T2-weighted FSE with fat saturation, are ideal to detect non-cystic bone marrow lesions (BML) in their maximum extent [105]. Gradient-recalled echo-type sequences with robust water excitation are insensitive to diffuse marrow abnormalities because of trabecular magnetic susceptibility, but are very sensitive in delineating subchondral cysts [20]. Additionally, for correct assessment of sclerotic lesions, a non-fat-saturated T1-weighted sequence is required. It is important to have these 4 basic sequences as they serve for optimal cartilage and subchondral bone scoring [81]. Additional in vivo biochemical imaging such as dGEMRIC, T2 mapping, and diffusion-weighted imaging makes functional analysis of cartilage possible [4, 92]. For example, Krusche-Mandl et al. described that T2 mapping correlated with the modified Lysholm score in patients with autologous osteochondral transplantation [53]. A baseline MRI examination in a patient with clinical suspicion of a cartilage or osteochondral defect should be able to give—apart from an overall knee joint evaluation—a detailed description of the cartilage or osteochondral defect (Fig. 11.4).

The adequate assessment of cartilage repair tissue on MRI has led to the definition of pertinent variables for the description of articular cartilage repair tissue after different repair techniques (MOCART: magnetic resonance observation of cartilage repair tissue) as described by Marlovits et al. [59]. Additional modifications regarding the subchondral bone marrow and the subchondral bone plate evaluation are needed. The original MOCART scoring system evaluates the subchondral bone either as intact (attributed score = 1) or not intact (attributed score = 0) meaning edema, granulation tissue, cysts, or sclerosis. It seems appropriate to differentiate pure bone marrow edema-like signal from associated subchondral cysts since emerging evidence shows that subchondral cysts develop in preexisting regions of subchondral bone marrow edema-like signal [10, 12, 13].

In the recent literature, novelties have been described regarding imaging of the cartilage and subchondral bone. Welsch et al. showed that 3D PD SPACE is the best MRI sequence in terms of acquisition time and cartilage repair assessment [100]. Bousson et al. have developed a new integrated 3D segmentation and analysis tool for quantitative CT of the knee (Medical Image Analysis Framework (MIAF)-Knee) to determine the subchondral bone mineral density (BMD) and bone texture. Bone marrow lesions (BMLs) in MRI have been associated with pain and progression of knee OA, and in their study (population with Kellgren–Lawrence 2 and 3) there was a significant association between the occurrences of BMLs and increased local BMD [5]. Liukkonen et al. reported that arthroscopic ultrasound was feasible and that could assess the quantity of cartilage and the stiffness of the subchondral bone in vivo. This technique used the ultrasound reflection to determine the trabecular thickness [54].

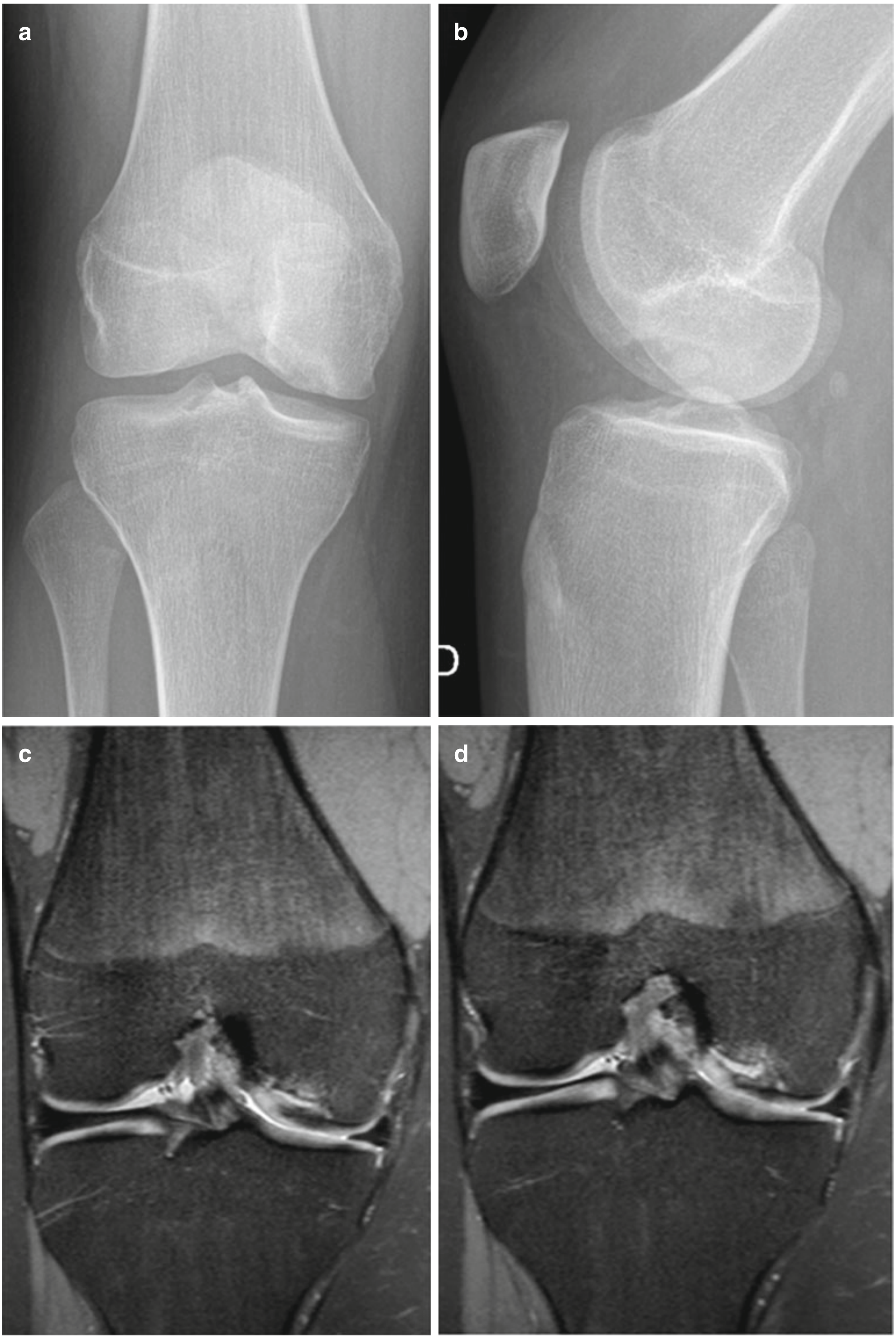

Fig. 11.4 Radiographs of a 19-year-old woman suffering from OCD of her right knee. (**a**, **b**) AP and lateral radiographs show an OCD. (**c**, **d**) MRI of the lesion with dissection within the subchondral bone

11.4 Biomimetic Scaffolds for Osteochondral Regeneration in the Knee Joint

The rationale to provide a temporary three-dimensional structure for the growth of living cells, able to guide tissue formation, led to the development of specific biopolymers, assembled in form of scaffolds for the regeneration of the damaged articular surface. The leading idea is that scaffolds should mimic biology, architecture, and functional properties of the native tissue, with the aim to facilitate cell infiltration, attachment, proliferation, and differentiation. Moreover, optimal biocompatibility and biodegradability through safe biochemical pathways at suitable time intervals are key: in fact, the scaffold should support the early phases of tissue formation and then be gradually replaced by regenerating tissue [49]. The ideal graft in clinical use should be an off-the-shelf product, thus avoiding the limits related to cell manipulation. In fact, despite overall satisfactory clinical results up to a medium- to long-term follow-up [27, 29] have been reported for cell-seeded scaffolds, the most recent trend is moving toward one-step products, possibly cell-free.

This emerging strategy, which involves the implant of various biomaterials for "in situ" cartilage repair, relies on the differentiation of bone marrow stem cells thanks to the scaffold properties [49]. These implants may be "smart" enough to be implanted cell-free directly into the defect site, providing the joint with the appropriate stimuli to induce orderly and durable tissue regeneration by the host resident cells. Among these cell-free techniques, Autologous Matrix-Induced Chondrogenesis (AMIC®) combines microfractures with the implantation of a porcine collagen type I/type III bilayer matrix as a 1-step procedure for the treatment of cartilage defects: satisfactory results were reported in 87 % of patients at mean 37 months' follow-up, in association with positive MRI findings in most cases [30]. A different kind of augmentation to microfractures involves an absorbable nonwoven polyglycolic acid textile treated with hyaluronic acid (BioTissue AG, Zurich, Switzerland) [75] that was applied to hold the progenitor cells into the blood clot in a 6 cm^2 posttraumatic medial femoral condyle defect, with good results at 2 years. Finally, several other surgical options are being tested to increase the healing potential, such deep subchondral perforations (to provide a greater number of MSCs to the membrane), or the addition of platelet-derived growth factors to further enhance the healing processes [17, 47, 74, 84]. However, despite some promising preliminary results and the increasing clinical application of such procedures, the results are still preliminary, and indications remain currently limited to small focal defects, whereas the real benefit in different conditions is yet not demonstrated.

Techniques traditionally used for osteochondral repair mainly originate from procedures initially developed for the cartilage layer: besides marrow stimulation procedures and modified ACI [26], involving the use of autologous bone to fill the bone defect, the transplantation of an entire osteochondral unit, either autologous or allogeneic, offers an immediate one-step alternative able to provide a viable bone and cartilage tissue directly into the lesions [3]. These techniques can potentially produce good clinical results, but several limitations emerged when addressing large lesions. This led surgeons and researchers to take advantage by the most recent achievements in biomaterial technology in order to develop a new treatment strategy focusing precisely on both bone and cartilage tissues. The development of new products with a bilayer structure allowed the entire osteochondral unit to be treated by reproducing the different biological and functional requirements in order to guide the growth of both bone and cartilage tissues, respectively.

Many scaffolds have been proposed, integrating distinct layers to address both cartilage and bone tissues [60]. The aim of such devices is to provide the right stimuli to regenerate the entire osteochondral unit, by the in situ presence of materials able to support and guide cell differentiation toward bone and cartilage. First, integrated bilayered osteochondral scaffolds have been proposed combining a ceramic component (i.e., hydroxyapatite, tricalcium phosphate) with a-hydroxy acid polymers (i.e., polylactic acid, polylactic-coglycolic acid); then biphasic but monolithic materials were formed by freeze-drying and chemical cross-linking collagen-based materials (i.e., mineralized or coupled with hyaluronic acid), as well as by ionotropic gelation of alginate-based materials (i.e., containing or not hydroxyapatite ceramic particles), allowing to achieve specific mechanical properties (i.e., elasticity or compression strength) [31, 83, 88]. Among the different scaffolds commercialized for clinical application and specifically developed to reproduce the different biological and functional requirements of bone and cartilage, the use of just two of them has been currently reported in literature.

The first osteochondral scaffold is a bilayer scaffold made of a porous PLGA–calcium sulfate biopolymer (TruFit, Smith & Nephew, Andover, MA) in form of cylinders, thus giving a surgical alternative to the mosaic-like osteochondral autograft transplantation (OAT). This technique showed to produce controversial clinical results and currently lacks of long-term follow-up studies, paired with unsatisfactory MRI findings [2, 9, 18, 42, 62].

The second osteochondral scaffold is a three-layer nanostructured implant consisting of collagen and hydroxyapatite, specifically designed to mimic the structure of cartilage and subchondral bone tissue, respectively. This composite material resembles the composition of the extracellular matrices of cartilage and bone tissue, and it is based on nucleation of hydroxyapatite (HA) nanocrystals onto self-assembled collagen fibers [91] to generate a chemically and morphologically graded hybrid biomaterial. Equine type I collagen acts as an organic matrix for the mineralization process, due to its good physicochemical stability and processability and high safety and biocompatibility profile, related to the removal of all potentially immunogenic telopeptides. The mineral phase is represented by magnesium–hydroxyapatite (Mg–HA). Magnesium ions were introduced to increase the physicochemical, structural, and morphological affinities of the composite with newly formed natural bone [85]. The cartilaginous layer surface is smooth, and it entirely consists of type I collagen. The intermediate layer is made of a combination of type I collagen and Mg–HA in 60 and 40 % weight ratio, whereas the lower layer consists of a mineralized blend of type I collagen and Mg–HA in 30 and 70 % weight ratio. Each layer is separately synthesized, and then the layers are physically combined on top of a Mylar sheet and finally freeze-dried and gamma-sterilized at 25 Kgray [91]. In vitro and animal studies [45, 50] tested this biomaterial, showing good results with cartilage and bone tissue formation. Similar macroscopic, histological, and radiographic results have been obtained by implanting scaffolds loaded with autologous chondrocytes or scaffolds alone, thus concluding that in the animal model the scaffold is able to induce an in situ regeneration through progenitor cells coming from the surrounding bone marrow. Following the results of these preclinical studies, this scaffold has been introduced in the clinical practice as a cell-free approach.

11.4.1 Clinical Investigations in Osteochondral Regeneration

The first clinical investigation (Table 11.3) in osteochondral regeneration regarding surgical technique and the intrinsic mechanical stability of the biomimetic construct was conducted in 13 patients [46], documenting with high-resolution MRI the graft adherence at 5–8 weeks after implantation and then further graft stability and maturation at 6 months. The pilot clinical study [44] involved 30 consecutive patients with lesion size within 6.0 cm^2, and a significant improvement was reported at 24 months of follow-up. The same results but a slower recovery was observed in patients who presented adverse events, older ones, patients with previous surgery, and for lesions located in the patella. A faster recovery was associated to higher pre-injury activity level. Most of the evaluated MRIs showed complete filling of the cartilage and complete integration of the graft. Twenty-seven patients of the same series [48] were recently evaluated at 5 years' follow-up, showing that the

Table 11.3 Osteochondral regeneration with a biomimetic scaffold

Scientific publication	Type of study	Number of patients	Minimum follow-up	Summary—results
Kon et al., *KSSTA* [43]	Case report	1	1 year	Treatment of multiple osteochondral lesions in an active 46-year-old patient who previously underwent anterior cruciate ligament (ACL) reconstruction. Treatment with scaffold implants for degenerative lesions of MFC, trochlea, and patella, with associated closing wedge HTO. At 1-year follow-up, the patient was pain-free, had full range of motion, and returned to his preoperation tennis level. MRI at 6 and 12 months after surgery: the implants were stable and showed a hyaline-like signal with good restoration of the articular surface after 6 months; subchondral edema progressively decreased over time, and at 12 months it was barely evident
Kon et al., *Injury* [46]	Case series	13	6 months	Early stability pilot clinical trial. Lesion site (15 defects): 4 MFC, 2 LFC, 5 patella, and 4 trochlea. Defect size: 2.8 cm^2. At 4–8-week MRIs, 13 implantation sites had complete attachment and adherence, while in two patients only partial attachment was found. In these two patients, 6-month MRIs showed partial reabsorption of the graft in one case with an incomplete cartilage layer and a complete subchondral structure, while in the other case, an inhomogeneous tissue filled the entire treated area. Histological analysis (two cases): presence of perfectly formed subchondral bone and complete biomaterial reabsorption
Kon et al., *AJSM* [44]	Case series	28	2 years	Pilot clinical trial. Lesion site (34 defects): 8 MFC, 5 LFC, 12 patella, 7 trochlea, and 2 lateral tibial plateau. Defect size: 2.9 cm^2. Significant clinical improvement in IKDC subjective and objective scores and in Tegner score. At MRI complete filling and integration in 70 % of the cases. Slower recovery but the same results for patients who presented with adverse events (6 swelling, 1 bleeding, 2 fever, 2 joint stiffness), for older patients, for patients with previous surgery, and for those with patellar lesions. In contrast, a faster recovery was observed in active patients
Filardo et al., *Cartilage* [25]	Case report	1	5 years	Treatment of a Schatzker type II tibial plateau fracture in a 50-year-old woman. Previous surgery: reduction and osteosynthesis of the fracture 3 years before. Surgical technique: open-wedge elevation osteotomy of the tibial lateral plateau, homologous bone graft, osteochondral scaffold, and external fixator. Significant improvement both clinically (IKDC subjective from 40.2 to 88.5, EQ-VAS from 49 to 83, Tegner score from 0 to 4) and at MRI evaluation. After 5 years from surgery, the patient did not complain of knee pain, had a full range of motion, and returned to her previous activities
Kon et al., *AJSM* [48]	Case series	27	5 years	Pilot clinical trial. Lesion site (32 defects): 7 MFC, 5 LFC, 11 patella, 7 trochlea, and 2 lateral tibial plateau. Defect size: 2.9 cm^2. Both Tegner (from 1.6 to 4.1) and IKDC subjective (from 40.0 to 77.1) scores improved significantly from the baseline evaluation up to 5 years. MRI evaluation (23 lesions): significant improvement in both MOCART score and subchondral bone status from 2 to 5 years. At 5 years: 78.3 % complete filling, 69.6 % complete integration, 60.9 % intact repair tissue surface, and 60.9 % homogeneous structure of the repair tissue
Filardo et al., *AJSM* [26]	Case series	27	2 years	Treatment of skeletally mature patients affected by symptomatic knee osteochondritis dissecans of the femoral condyles. Improvement in IKDC subjective (from 48.4 to 82.3), IKDC objective (from 40 to 85 % of normal knees), and Tegner scores (from 2.4 to 4.5). MRI evaluation: good defect filling and implant integration but also inhomogeneous regenerated tissue and subchondral bone changes in most patients. No correlation between MOCART score and clinical outcome

Marcacci et al., *KSSTA* [58]	Case series	43	2 years	Evaluation of an integrated biomechanical and biological approach as alternative to metal resurfacing for unicompartmental osteoarthritis. Surgical procedure: osteochondral scaffold, synthetic meniscal scaffold, meniscal allograft, tibial/femoral osteotomy in multiple combinations. Significant improvement using IKDC subjective (from 47.3 to 79.6), VAS (from 6.1 to 2.3), and Tegner scores (from 2 to 4). Positive outcome confirmed in all the treatment subgroups, with a higher clinical improvement in patients under the age of 40 years
Filardo et al., *Knee* [28]	Comparative study	33	2 years	Comparison between chondral and osteochondral scaffold for the treatment of complex cases. Inclusion criteria: previous clinical history of intra-articular fracture, lesion located at the tibial plateau, concurrent knee axial realignment procedure, concurrent meniscal scaffold or allograft implantation, and multiple articular surface lesions treated. Significant improvement in osteochondral scaffold group in IKDC subjective (from 40.4 to 75.5), VAS (from 4.0 to 7.3) and Tegner scores (from 1.9 to 4.5). Better subjective IKDC score for the group treated with the osteochondral scaffold with respect to the group treated with the chondral scaffold
Delcogliano et al., *KSSTA* [16]	Case series	19	2 years	Treatment of large osteochondral defects. Lesion site (20 defects): MFC 10, LFC 7, and tibial plateau 3. Defect size: 5.2 cm^2. Significant improvement in IKDC subjective score (from 35.7 to 72.9) and EQ-VAS; 85 % of the patients were satisfied with the clinical outcome. MRI evaluation: MOCART score 63.2, subchondral lamina was never considered intact, and subchondral bone changes were observed in all cases. No correlation was found analyzing MOCART score and IKDC subjective evaluation or the Tegner score. Slower recovery in patients with degenerative lesions and better results for MFC and tibial plateau with respect to LFC. Two failures
Perdisa et al., *ERPS* [76]	Case report	1	2 years	Treatment of complex degenerative lesions of the knee due to subtotal meniscal loss and patellar overload in a 31-year-old Olympic fencer. Surgical technique: articular resurfacing with OC scaffold (patella and lateral condyle) and OAT (trochlea) in combination with lateral meniscal allograft transplantation and patellar realignment. Clinical improvement in both knee functional status and pain. Return to official competition 17 months after surgery

good results obtained at 24 months were stable over time. However, even though MRI findings (analyzed in 18 patients who had both 24 months and final follow-up) improve over time, some abnormalities persisted, but no correlation was found between imaging and clinical results.

This procedure has been reported to be successful also for OCD lesions at short-term follow-up [26]; this articular disease primary involves the subchondral bone, often resulting in separation and instability of the overlying articular cartilage; thus, an osteochondral approach is mandatory. The MRI evaluations in this peculiar group of patients were positive in terms both of defect filling and implant integration, whereas most patients presented inhomogeneity of the regenerating tissue and the presence of subchondral bone changes. Also in this study a correlation between MOCART (magnetic resonance observation of cartilage repair tissue) score and clinical outcome was not found. A recent study by Delcogliano et al. evaluated 19 patients successfully treated with the same biomimetic scaffold for big size articular defects, confirming its potential also in large osteochondral lesions at 24 months of follow-up [16]. The potential of the biomimetic osteochondral approach has even been demonstrated in a group of patients affected by complex lesions, where the subchondral bone is clearly involved in the etiopathogenetic process. Among them, the first report was published on a former athlete, a 46-year-old patient [43], reporting good clinical results 12 months after the implantation in multifocal osteochondral lesions of the medial femoral condyle, trochlea, and patella and concurrently by restoring the correct alignment of the varus knee through a closing-wedge high tibial osteotomy. The MRI performed 6 and 12 months after surgery showed a hyaline-like signal and anatomical restoration of the articular surface, together with a progressive decrease of subchondral edema over time. Similar findings were reported up to 4 years postoperatively in a 50-year-old woman affected by an osteochondral defect at the tibial plateau following a Schatzker type II fracture [24]. Besides filling the bone and cartilage defect with this three-layered implant, the tibial plateau was elevated by an opening-wedge osteotomy and filled with homologous bone graft, and finally a dynamic external distractor was applied to allow early mobilization. Twelve months later the patient was pain-free and returned to a satisfactory activity level maintained stable until the medium-term follow-up. Finally, the implantation of this scaffold as part of a complex treatment to address multifocal degenerative knee lesions of an Olympic-level athlete, concurrently with patellar realignment, meniscal allograft transplantation, and the use of an autologous osteochondral graft, allowed the patient to return to high-level competitions within 24 months postoperatively [76]. This combined approach aims at restoring the previous anatomical features with both mechanical and biological treatments, and these promising cases suggested a more extensive application for the treatment of complex osteochondral lesions. Following this rationale, a study was performed, specifically focusing on 33 patients affected by challenging knee lesions [28] defined as "complex" according to defined criteria, where concurrent procedures were necessary to address axial misalignment and meniscal resection sequelae. This approach produced a good clinical outcome at 24 months, and the final clinical results were higher than those of a homogeneous group of 23 patients previously treated with a similar protocol but with the implantation of a chondral scaffold. Finally, the biomimetic scaffold was tested as an alternative to metal resurfacing for unicompartmental OA in young patients [58]. This population is maybe the most challenging in the field of joint reconstructive surgery, due to a combination of high functional demands and great expectations regarding their recovery, but a limited choice of treatment options on the other hand. At present day, the main surgical indication in a young and active population affected by unicompartmental OA consists in unloading osteotomies or unicompartmental metal resurfacing, depending on the age of the patient and stage of the disease [32]; however, the high functional requests and the young age of this kind of patient increase the risk of prosthetic revision. Therefore, a solution to delay or even avoid metal resurfacing is highly desirable. The osteochondral scaffold was implanted in a group of 43 unicompartmental OA patients (Kellgren–Lawrence 3) with full-thickness focal cartilage lesions in stable knees, and concurrent procedures were performed when requested (15 osteotomies, 11 meniscal scaffolds, and 9 meniscal allograft implantation), leading to a significant clinical improvement from pre-op to the 3-year follow-up. Best benefits were obtained in patients younger than 40 years, showing the promising results of this surgical approach as new treatment option even in young OA patients.

Taken together, several procedures aiming to regenerate articular tissues are being tested, showing good results at short- and medium-term follow-up. However, they are mainly indicated for the treatment of focal and traumatic cartilage defects. The damage of the articular surface often involves the subchondral bone, and there is a need for treatment strategies providing its correct restoration. Thus, different osteochondral scaffolds have been created. Among these, a biomimetic nanostructured osteochondral scaffold, reproducing the requirements of both bone and cartilage, has been reported to potentially offer good clinical results at midterm follow-up. Findings were promising even for complex knee lesions, so that it can be considered as a valid option for both large chondral and osteochondral lesions. On the other hand, imaging evaluation showed some controversial findings, with the persistence of an altered signal and a slow maturation process of the osteochondral structure. Thus, even if satisfactory clinical outcomes have been already shown by the literature, some elements might be further improved in order to obtain a faster and more effective healing process.

11.5 How to Treat Subchondral Bone Pathologies in the Ankle Joint

The development of a symptomatic osteochondral defect in the ankle joint depends on various factors, including the damage and insufficient repair of the subchondral bone Plate. A defect may heal, remain asymptomatic, or progress to deep ankle pain on weight bearing, prolonged joint swelling, and formation of subchondral bone cysts. The ankle joint has a high congruency. During loading, compressed cartilage forces its water into the microfractured subchondral bone, leading to a localized high increased flow and pressure of fluid into the subchondral bone. This will result in local osteolysis and can explain the slow development of a subchondral cyst. The pain does not arise from the cartilage lesion, but is caused by repetitive high fluid pressure during walking, which results in stimulation of the highly innervated subchondral bone underneath the cartilage defect. Malalignment of the hindfoot plays an important role in the development of further degeneration. Plain radiographs may disclose the lesion. Modern imaging technology has enhanced the ability to fully evaluate and accurately determine the size and extent of the lesion, which are important for proper treatment. Concerning diagnosis, CT scan and MRI have similar accuracy. CT scan however is better for preoperative planning.

Treatment options for osteochondral ankle defects are diverse, and up to the present there is no consensus. The following guidelines, guided by the size of the lesion, are based on the current literature. Asymptomatic or low-symptomatic lesions are treated nonoperatively. The primary surgical treatment of defects up to 15 mm in diameter consists of arthroscopic debridement and bone marrow stimulation. For large cystic talar lesions, retrograde drilling combined with a bone graft is an alternative. In adolescents or in (sub)acute situations in which the fragment is 15 mm or larger, fixation of the fragment is preferred. Osteochondral autograft transfer and autologous chondrocyte implantation, with or without a cancellous bone graft, are recommended for treatment of secondary cases as well as large lesions. Resurfacing implants are a promising novel technique for medial defects. Although these are often successful, malalignment may be the cause of persistent symptoms. Correction osteotomy may be suitable for osteochondral defects in selected cases. Of note, Cochrane Review did not find evidence for superiority of any of the techniques for cartilage repair, and it was concluded that the majority of studies on cartilage have a low methodological quality repair [99]. A systematic review [57] on treatment of focal cartilage defects identified five randomized controlled trials, and it was concluded that no technique consistently had superior results compared to other techniques. No technique available today has been able in clinical studies to prove the regeneration of normal hyaline cartilage. Tissue engineering has a great potential and probably in the future could have superior results compared to bone marrow stimulation techniques. For the moment, all tissue engineering techniques and introduction of new scaffolds should be carefully evaluated in randomized clinical trials before they can be generally adopted [55].

11.5.1 Relationship Between Cartilage Thickness and Joint Congruency

The thickness of the cartilage differs between joints. When compared to other joints, the ankle joint has the thinnest cartilage. The average cartilage thickness of the talar dome is 1.11 mm (±0.28 mm) in women and 1.33 mm (±0.22 mm) in men [87, 90]. It has been demonstrated that there is a linear relationship between cartilage thickness and joint congruency [6]. Joints with a high congruency have thin cartilage, whereas incongruent joints have the thickest cartilage coverage. A thick layer of cartilage can more easily deform thereby increasing the load-bearing area and decrease the stress per unit area.

11.5.2 Mechanism of Repair in Traumatic Osteochondral Defects

By definition osteochondral defects involve both the articular cartilage and the underlying subchondral bone, while chondral defects are restricted to the articular cartilage. In both situations the MRI can show bone bruising. Bone bruises are seen as decreased signal intensity on T1-weighted MRI studies and an elevated intensity on T2-weighted MRI. Bone bruises can be classified as reticular lesions or geographic lesions. The reticular bone bruise is not continuous with the articular service [7, 68, 97]. This type of bone bruise heals from the periphery to the center [15]. The geographic bone bruises are continuous with the articular service. Spontaneous healing is impaired or can be absent [68, 80, 97].

The bone marrow edema will resolve when the subchondral bone plate heals. In cases where the subchondral bone plate did not heal, the initial traumatic defect will lead to a symptomatic osteochondral defect with cyst formation. The mechanism is as follows. Cracks or other local defects in the subchondral bone remain, and the water content of the cartilage is forced into the subchondral spongiosa with every step. Intermittent fluid pressure of 150 mmHg executed on the bone has demonstrated to cause bone resorption [1, 93]. The formation of symptomatic subchondral bone cysts thus can be explained [94, 95].

In cases of traumatic osteochondral defects of larger size, healing will depend on whether the osteochondral fragment stays in situ or not. If the defect remains in situ, it might heal. In case it leaves its donor site, then spontaneous repair of the osteochondral defect will usually take place. This depends on the size of the lesion. Smaller defects are more likely to be completely repaired compared to larger defects. Alignment plays a role. Progression of an osteochondral lesion depends on the type of the lesion. A superficial defect with sheared-off flakes with intact subchondral bone plate has a good prognosis in a congruent joint. Since the bone plate is intact, these lesions have a very limited chance of becoming symptomatic. An osteochondral defect is spontaneously filled with a blood clot that forms by cells entering the defect from the bone marrow. Differentiation of mesenchymal cells takes place. Part of the mesenchymal cells transform to osteoblasts forming immature bone starting from the edges of the bone defect. This new bone formation can restore part of the subchondral bone injury. The mesenchymal cells will differentiate to chondrocytes, and the rest of the defect will be filled with fibrocartilage. Restoration of the subchondral bone is crucial in order to allow for this new articular cartilage formation.

11.5.3 Therapy of Chronic Lesions

Arthroscopic techniques aiming to preserve joint function include debridement of the defect with removal of unstable articular cartilage flaps. The penetration of the subchondral bone plate to grant access of cells from the bone marrow to the articular cartilage defect is an important step for many clinical procedures in articular cartilage repair [40, 77, 89]. Correct restoration of large defects in the subchondral bone probably plays a crucial role in achieving satisfactory articular cartilage repair. Techniques for inducing a fibrocartilaginous repair include marrow stimulation techniques as abrasion arthroplasty, microfracturing, or Pridie drilling. All these methods rely on a perforation of the subchondral bone plate in order to grant access of the bone marrow to the defect. Other treatment options are retrograde drilling, fixation of fragment, osteochondral autograft fragmentation, autologous chondrocyte implantation (ACI), or limited prosthetic replacement (HemiCAP). The goal of all treatments is to resolve the symptoms and ideally to prevent the development of osteoarthritis on the long term. However, there are no long-term follow-up studies of untreated osteochondral defects that demonstrate progress or deterioration of the ankle joint. Reports of patients that are undergoing an ankle replacement or ankle arthrodesis following an OCD are rare. The natural history of talar OCD lesions is mostly benign. Treatment of a symptomatic osteochondral defect therefore has to concentrate on relief of symptoms. The main symptom is pain. The cause of pain in an osteochondral defect arises from the subchondral bone [94, 95]. Treatment strategies therefore need to concentrate on the subchondral bone rather than on the cartilage. Detection of the bone lesion is likewise as important as detecting of the cartilage lesion itself. For detection of a talar osteochondral defect, CT scan and MRI have shown similar accuracy [98]. A CT scan is the diagnostic strategy of choice. For preoperative planning, CT scan is preferred since it demonstrates the exact size and location of the subchondral bone lesion. It is the subchondral bone lesion that has to be treated in order to treat the pain. Overview of the literature has demonstrated that excision, curettage, and bone marrow stimulation are the current treatment strategies of choice [102]. The overall success rate has reported to be 85 %.

11.6 Philosophical Remarks on the Subchondral Bone: Healthy Soil for the Healthy Cartilage

Relationship between cartilage and subchondral bone is very much similar to a relation between plant and the soil underneath. A healthy plant cannot be imagined without the healthy soil. Soil provides base for the plant and scaffold for its roots, supplies nutrition to it, and gives ability to withstand strong winds (shear forces) to avoid being uprooted out. On the other side, plants provide necessary coverage to the soil so that soil erosion is prevented from direct stresses of harsh weather and keep soil bound together with its roots [79]. Soil is actually made up of four layers: topsoil, subsoil, regolith, and the rock bed. Above all these four layers is the covering by a layer called organic layer and by plantations. An osteochondral unit also consists of calcified cartilage, cement line, subchondral bone plate, and subarticular spongiosa, all four layers having a superficial covering of articular cartilage. The purpose of comparison of subchondral bone–cartilage relationship with soil–plant relationship is to find out if the subchondral bone is as important to the healthy cartilage as the soil is for the healthy plant. An established relation can help surgeons to give more importance to subchondral bone while dealing with cartilage pathologies. Following incidences were studied to scrutinize the relationship.

Certain diseases of the cartilage are actually diseases of the subchondral bone rather than a disease of the cartilage *per se*. It is akin to a bad soil causing loss of healthy plantation on it. OCD is one such disease where the pathological process starts in the subchondral bone, gradually causing separation of the affected bone from the surrounding healthy subchondral bone. An unstable and probably unviable subchondral bone causes overlying cartilage to stop functioning in its normal capacity. Gradually the whole osteochondral unit separates with overlying cartilage. A similar disease process might be affecting the cases with osteonecrosis. Compare it with a condition in the backyard garden, where a disease affects soil first. For example, an area of soil gets desiccated (state of extreme dryness due to lack of water) and that will lead to loss of the entire healthy plantation on it. Any attempt to salvage the dying plant is useless unless a proper treatment for the ailing soil is done. If the process continues, then after some time desiccated soil separates from the surrounding soil. A dried-up piece of soil that was devoid of water actually becomes useless and needs to be replaced by fresh soil. This is simply because of the reason that a soil deprived of water has also lost many nutrients and thus has also lost its regenerating capacity. It not only becomes hard but also develops cracks. It is similar to OCD in that when the piece becomes sclerotic and fragmented, the defect needs to be reconstructed using bone graft and cartilage repair surgery.

The cartilage has a protective role for the underlying bone from the joint stresses, and on the other side, the subchondral bone has a supportive role to play for the cartilage. This is a synergistic role where both work in a perfect homeostatic balance and neutralize abnormal stresses on each other. For example, an isolated chondral injury can alter the homeostatic balance of the joint leading to progression of surrounding cartilage damage. It also makes the area more vulnerable to the shear forces, ultimately leading to increase in the size of focal chondral defect and changes in the otherwise normal underlying subchondral bone plate in form of overgrowth or bone loss. Here, the protective role of the cartilage is lost, leading to damage to the underlying bone. On the other hand, a subchondral bone fracture also leads to changes in homeostatic balance of the joint and leads to unsupported cartilage over it. The supportive role of the subchondral bone is lost leading to the damage to the overlying cartilage. There are similar incidences when the soil is normal, but there is loss of overlying plantation, thus exposing the soil to harsh weather conditions. An exposed superficial area of land gradually gets eroded due to shear forces of the strong winds. Gradually roots of dead plants fail to keep the soil intact and cause loss of soil crust. On the other side, a healthy and integrated soil is a must for a healthy plant. It provides a structured support for the plant to grow. Any break in soil crust can result in loss of support of the overlying plant.

It has been shown that blood vessels from the subchondral region can extend into the overlying calcified cartilage, and nutrients reach chondrocytes in the calcified zone. Their diffusion can occur in a similar way that can occur between synovial fluid and the superficial cartilage layers [56]. Roots of the plant in the soil also serve the same purpose of bringing nutrition to the plant, however, to the greater extent in plants than in cartilage. Plants however also take its nutrition from photosynthesis, something similar to cartilage taking its nutrition from synovial fluid.

Though collagen fibers cross between calcified and uncalcified cartilage, there is no crossing of collagen fibers between calcified cartilage and subchondral bone plate. In addition, there is a considerable change in the biomechanical properties of calcified and uncalcified articular cartilage in adults due to mineralization, making this area weak. This is the reason for a high incidence of isolated chondral separation occurring due to shear forces in adults [41]. A shear force and pullout force applied to a plant lead to its uprooting with or without soil mass surrounding the roots. This difference depends on the weak interface that might exist between roots and soil and between nearby soil and far soil.

The regenerative tissues in the repaired cartilage need support from the healthy subchondral bone; otherwise, the overlying cartilage repair will fail [56]. A new plant grows well only if sown into a healthy soil. Soil not only supports in form of scaffolding of its roots but also by constant supply of essential nutrients. Occasionally an outside support in form of a small stick is needed to the plantation, till its roots are mature enough to support the plant. In the regenerating cartilage, collagen fibers must also develop cross-links so as to provide a similar scaffolding support till a weak chondral or osteochondral construct gains maturity. An external brace support and limited weight bearing are needed till the time collagen fibers make the scaffold strong.

A lesson on soil–plant relationship thus helps in understanding the relation that the cartilage has with the subchondral bone. A proper attention to the subchondral bone is important to maintain a healthy cartilaginous tissue and to regenerate a healthy cartilage in case of cartilage loss, as both tissues work as one unit.

Conclusion

The treatment of osteochondral lesion continues to remain a challenge. Epidemiological studies need to provide more data on osteochondral defects that are caused by trauma, OCD, ON, and OA. In the diagnostic workup of patients with clinical suspicion for a cartilage or osteochondral defect, apart from standard radiographs, an MRI examination should be performed. Understanding and respecting the natural history of each type of subchondral bone lesion is a key to select the optimal surgical therapy. Further clinical investigations at longer follow-up with a higher number of patients are required to confirm the efficacy and durability of the clinical outcome of osteochondral biomimetic scaffolds over time and to understand which indication is the most appropriate for their use. Altogether, a better understanding of the basic science and pathophysiology of the osteochondral unit, together with additional investigations in animal models and patients, will translate into improved strategies for the repair of cartilage defects arising from or extending into the subchondral bone.

Acknowledgments The authors thank B. Di Matteo, G. Venieri, F. Balboni: II Clinic, Biomechanics Laboratory, Rizzoli Orthopaedic Institute, Bologna, Italy. Mona Eldracher, Gertrud Schmitt: Center of Experimental Orthopaedics, Homburg, Germany for helpful discussions.

References

1. Aspenberg P, Van der Vis H. Migration, particles, and fluid pressure. A discussion of causes of prosthetic loosening. Clin Orthop Relat Res. 1998;352:75–80.
2. Bedi AF, Foo LF, Williams III RJ, Potter HG, The Cartilage Study Group. The maturation of synthetic scaffolds for osteochondral donor sites of the knee: an MRI and T2-mapping analysis. Cartilage. 2010;1(1):20–8.
3. Bentley G, Bhamra JS, Gikas PD, Skinner JA, Carrington R, Briggs TW. Repair of osteochondral defects in joints–how to achieve success. Injury. 2013;44 Suppl 1:S3–10. Review.
4. Bining HJ, Santos R, Andrews G, Forster BB. Can T2 relaxation values and color maps be used to detect chondral damage utilizing subchondral bone marrow edema as a marker? Skeletal Radiol. 2009;38:459–65.
5. Bousson V, Lowitz T, Laouisset L, Engelke K, Laredo JD. CT imaging for the investigation of subchondral bone in knee osteoarthritis. Osteoporosis Int. 2012;23 Suppl 8:S861–5.
6. Braune W, Fischer O. Die Bewegungen des Kniegelenks nach einer neuen Methode am lebenden Menschen gemessen. Abhandlungen der mathematisch-physischen Klasse der Köninglich-Sächsischen Gesellschaft der Wissenschaften 17–2, Leipzig, S Hirzel; 1891. p. 75–150.
7. Bretlau T, Tuxoe J, Larsen L, Jorgensen U, Thomsen HS, Lausten GS. Bone bruise in the acutely injured knee. Knee Surg Sports Traumatol Arthrosc. 2002;10:96–101.
8. Brown WE, Potter HG, Marx RG, Wickiewicz TL, Warren RF. Magnetic resonance imaging appearance of cartilage repair in the knee. Clin Orthop Relat Res. 2004;422:214–23.
9. Carmont MR, Carey-Smith R, Saithna A, Dhillon M, Thompson P, Spalding T. Delayed incorporation of a TruFit plug: perseverance is recommended. Arthroscopy. 2009;25(7):810–4.
10. Carrino JA, Blum J, Parellada JA, Schweitzer ME, Morrison WB. MRI of bone marrow edema-like signal in the pathogenesis of subchondral cysts. Osteoarthritis Cartilage. 2006;14:1081–5. OARS, Osteoarthritis Research Society.
11. Cole BJ, Farr J, Winalski CS, Hosea T, Richmond J, Mandelbaum B, De Deyne PG. Outcomes after a single-stage procedure for cell-based cartilage repair: a prospective clinical safety trial with 2-year follow-up. Am J Sports Med. 2011;39:1170–9.
12. Crema MD, Roemer FW, Marra MD, Niu J, Lynch JA, Felson DT, Guermazi A. Contrast-enhanced

MRI of subchondral cysts in patients with or at risk for knee osteoarthritis: the MOST study. Eur J Radiol. 2010;75:e92–6.
13. Crema MDR, Roemer FW, Marra MD, Niu J, Zhu Y, Lynch J, Lewis CE, El-Khoury G, Felson DT, Guermazi A. 373 MRI-detected bone marrow edema-like lesions are strongly associated with subchondral cysts in patients with or at risk for knee osteoarthritis: the most study. Osteoarthritis Cartilage. 2008;16:S160. World Congress on Osteoarthritisheld.
14. Curl WW, Krome J, Gordon ES, Rushing J, Smith BP, Poehling GG. Cartilage injuries: a review of 31,516 knee arthroscopies. Arthroscopy. 1997;13:456–60.
15. Davies NH, Niall D, King LJ, Lavelle J, Healy JC. Magnetic resonance imaging of bone bruising in the acutely injured knee-short-term outcome. Clin Radiol. 2004;59:439–45.
16. Delcogliano M, de Caro F, Scaravella E, Ziveri G, De Biase CF, Marotta D, Marenghi P, Delcogliano A. Use of innovative biomimetic scaffold in the treatment for large osteochondral lesions of the knee. Knee Surg Sports Traumatol Arthrosc. 2013. doi 10.1007/s00167-013-2717-3 PMID: 24146051. [Epub ahead of print].
17. Dhollander AA, De Neve F, Almqvist KF, et al. Autologous matrix-induced chondrogenesis combined with platelet-rich plasma gel: technical description and a five pilot patients report. Knee Surg Sports Traumatol Arthrosc. 2011;19(4):536–42.
18. Dhollander AA, Liekens K, Almqvist KF, Verdonk R, Lambrecht S, Elewaut D, Verbruggen G, Verdonk PC. A pilot study of the use of an osteochondral scaffold plug for cartilage repair in the knee and how to deal with early clinical failures. Arthroscopy. 2012;28(2):225–33.
19. Drobnic M, Radosavljevic D, Cor A, Brittberg M, Strazar K. Debridement of cartilage lesions before autologous chondrocyte implantation by open or transarthroscopic techniques: a comparative study using post-mortem materials. J Bone Joint Surg Br. 2010;92:602–8.
20. Duc SR, Koch P, Schmid MR, Horger W, Hodler J, Pfirrmann CW. Diagnosis of articular cartilage abnormalities of the knee: prospective clinical evaluation of a 3D water-excitation true FISP sequence. Radiology. 2007;243:475–82.
21. Eckstein F, Muller-Gerbl M, Steinlechner M, Kierse R, Putz R. Subchondral bone density of the human elbow assessed by computed tomography osteoabsorptiometry: a reflection of the loading history of the joint surfaces. J Orthop Res. 1995;13:268–78.
22. El-Khoury GY, Alliman KJ, Lundberg HJ, Rudert MJ, Brown TD, Saltzman CL. Cartilage thickness in cadaveric ankles: measurement with double-contrast multi-detector row CT arthrography versus MR imaging. Radiology. 2004;233:768–73.
23. Felson DT, Nevitt MC, Yang M, Clancy M, Niu J, Torner JC, Lewis CE, Aliabadi P, Sack B, McCulloch C, Zhang Y. A new approach yields high rates of radiographic progression in knee osteoarthritis. J Rheumatol. 2008;35:2047–54.
24. Filardo G, Di Martino A, Kon E, Delcogliano M, Marcacci M. Midterm results of a combined biological and mechanical approach for the treatment of a complex knee lesion. Cartilage. 2013;3(3):288–92.
25. Filardo G, Kon E, Berruto M, Di Martino A, Patella S, Marcheggiani Muccioli GM, Zaffagnini S, Marcacci M. Arthroscopic second generation autologous chondrocytes implantation associated with bone grafting for the treatment of knee osteochondritis dissecans: results at 6 years. Knee. 2012;19(5):658–63.
26. Filardo G, Kon E, Di Martino A, Busacca M, Altadonna G, Marcacci M. Treatment of knee osteochondritis dissecans with a cell-free biomimetic osteochondral scaffold: clinical and imaging evaluation at 2-year follow-up. Am J Sports Med. 2013;41(8):1786–93.
27. Filardo G, Kon E, Di Martino A, Iacono F, Marcacci M. Arthroscopic second-generation autologous chondrocyte implantation: a prospective 7-year follow-up study. Am J Sports Med. 2011;39(10):2153–60.
28. Filardo G, Kon E, Perdisa F, Di Matteo B, Di Martino A, Iacono F, Zaffagnini S, Balboni F, Vaccari V, Marcacci M. Osteochondral scaffold reconstruction for complex knee lesions: a comparative evaluation. Knee. 2013;20(6):570–6.
29. Filardo G, Kon E, Roffi A, Di Martino A, Marcacci M. Scaffold-based repair for cartilage healing: a systematic review and technical note. Arthroscopy. 2013;29(1):174–86.
30. Gille J, Schuseil E, Wimmer J, Gellissen J, Schulz AP, Behrens P. Mid-term results of Autologous Matrix-Induced Chondrogenesis for treatment of focal cartilage defects in the knee. Knee Surg Sports Traumatol Arthrosc. 2010;18(11):1456–64.
31. Gomoll AH, Farr J, Gillogly SD, Kercher J, Minas T. Surgical management of articular cartilage defects of the knee. J Bone Joint Surg Am. 2010;92:2470–90.
32. Gomoll AH, Filardo G, de Girolamo L, Espregueira-Mendes J, Marcacci M, Rodkey WG, Steadman JR, Zaffagnini S, Kon E. Surgical treatment for early osteoarthritis. Part I: cartilage repair procedures. Knee Surg Sports Traumatol Arthrosc. 2012;20(3):450–66.
33. Henderson IJ, La Valette DP. Subchondral bone overgrowth in the presence of full-thickness cartilage defects in the knee. Knee. 2005;12:435–40.
34. Hjelle K, Solheim E, Strand T, Muri R, Brittberg M. Articular cartilage defects in 1,000 knee arthroscopies. Arthroscopy. 2002;18:730–4.
35. Huey DJ, Hu JC, Athanasiou KA. Unlike bone, cartilage regeneration remains elusive. Science. 2012;338:917–21.
36. Hunziker EB. Articular cartilage repair: are the intrinsic biological constraints undermining this process insuperable? Osteoarthritis Cartilage. 1999;7:15–28.
37. Hunziker EB. The elusive path to cartilage regeneration. Adv Mater. 2009;21:3419–24.
38. Imhof H, Sulzbacher I, Grampp S, Czerny C, Youssefzadeh S, Kainberger F. Subchondral bone

and cartilage disease: a rediscovered functional unit. Invest Radiol. 2000;35:581–8.
39. Jackson DW, Lalor PA, Aberman HM, Simon TM. Spontaneous repair of full-thickness defects of articular cartilage in a goat model. A preliminary study. J Bone Joint Surg Am. 2001;83A:53–64.
40. Johnson LL. Arthroscopic abrasion arthroplasty: a review. Clin Orthop. 2001;391:306–17.
41. Johnson-Nurse C, Dandy DJ. Fracture-separation of articular cartilage in the adult knee. J Bone Joint Surg Br. 1985;67:42–3.
42. Joshi N, Reverte-Vinaixa M, Díaz-Ferreiro EW, Domínguez-Oronoz R. Synthetic resorbable scaffolds for the treatment of isolated patellofemoral cartilage defects in young patients: magnetic resonance imaging and clinical evaluation. Am J Sports Med. 2012;40(6):1289–95.
43. Kon E, Delcogliano M, Filardo G, Altadonna G, Marcacci F. Novel nano-composite multi-layered biomaterial for the treatment of multifocal degenerative cartilage lesions. Knee Surg Sports Traumatol Arthrosc. 2009;17(11):1312–5.
44. Kon E, Delcogliano M, Filardo G, Busacca M, Di Martino A, Marcacci M. Novel nano-composite multilayered biomaterial for osteochondral regeneration: a pilot clinical trial. Am J Sports Med. 2011;39(6):1180–90.
45. Kon E, Delcogliano M, Filardo G, Fini M, Giavaresi G, Francioli S, Martin I, Pressato D, Arcangeli E, Quarto R, Sandri M, Marcacci M. Orderly osteochondral regeneration in a sheep model using a novel nano-composite multilayered biomaterial. J Orthop Res. 2010;28(1):116–24.
46. Kon E, Delcogliano M, Filardo G, Pressato D, Busacca M, Grigolo B, Desando G, Marcacci M. A novel nano-composite multi-layered biomaterial for treatment of osteochondral lesions: technique note and an early stability pilot clinical trial. Injury. 2010;41:693–701.
47. Kon E, Filardo G, Delcogliano M, Fini M, Salamanna F, Giavaresi G, Martin I, Marcacci M. Platelet autologous growth factors decrease the osteochondral regeneration capability of a collagen-hydroxyapatite scaffold in a sheep model. BMC Musculoskelet Disord. 2010;11:220.
48. Kon E, Filardo G, Di Martino A, Busacca M, Moio A, Perdisa F, Marcacci M. Clinical results and MRI evolution of a nano-composite multilayered biomaterial for osteochondral regeneration at 5 years. Am J Sports Med. 2014;42:158–65. PubMed PMID: 24114751.
49. Kon E, Filardo G, Roffi A, Andriolo L, Marcacci M. New trends for knee cartilage regeneration: from cell-free scaffolds to mesenchymal stem cells. Curr Rev Musculoskelet Med. 2012;5(3):236–43.
50. Kon E, Mutini A, Arcangeli E, et al. Novel nanostructured scaffold for osteochondral regeneration: pilot study in horses. J Tissue Eng Regen Med. 2010;4(4):300–8.
51. Kothari M, Guermazi A, von Ingersleben G, Miaux Y, Sieffert M, Block JE, Stevens R, Peterfy CG. Fixed-flexion radiography of the knee provides reproducible joint space width measurements in osteoarthritis. Eur Radiol. 2004;14:1568–73.
52. Kreuz PC, Steinwachs MR, Erggelet C, Krause SJ, Konrad G, Uhl M, Südkamp N. Results after microfracture of full-thickness chondral defects in different compartments in the knee. Osteoarthritis Cartilage. 2006;14:1119–25.
53. Krusche-Mandl I, Schmitt B, Zak L, Apprich S, Aldrian S, Juras V, Friedrich KM, Marlovits S, Weber M, Trattnig S. Long-term results 8 years after autologous osteochondral transplantation: 7 T gagCEST and sodium magnetic resonance imaging with morphological and clinical correlation. Osteoarthritis Cartilage. 2012;20:357–63. OARS, Osteoarthritis Research Society.
54. Liukkonen J, Hirvasniemi J, Joukainen A, Penttila P, Viren T, Saarakkala S, Kroger H, Jurvelin JS, Toyras J. Arthroscopic ultrasound technique for simultaneous quantitative assessment of articular cartilage and subchondral bone: an in vitro and in vivo feasibility study. Ultrasound Med Biol. 2013;39:1460–8.
55. Lopa S, Madry H. Bioinspired scaffolds for osteochondral regeneration. Tissue Eng Part A. 2014 Jan 29. [Epub ahead of print] PMID: 24476065.
56. Madry H, van Dijk CN, Mueller-Gerbl M. The basic science of the subchondral bone. Knee Surg Sports Traumatol Arthrosc. 2010;18:419–33.
57. Magnussen RA, Dunn WR, Carey JL, Spindler KP. Treatment of focal articular cartilage defects in the knee: a systematic review. Clin Orthop Relat Res. 2008;466:952–62.
58. Marcacci M, Zaffagnini S, Kon E, Marcheggiani Muccioli GM, Di Martino A, Di Matteo B, Bonanzinga T, Iacono F, Filardo G. Unicompartmental osteoarthritis: an integrated biomechanical and biological approach as alternative to metal resurfacing. Knee Surg Sports Traumatol Arthrosc. 2013;21(11):2509–17.
59. Marlovits S, Striessnig G, Resinger CT, Aldrian SM, Vecsei V, Imhof H, Trattnig S. Definition of pertinent parameters for the evaluation of articular cartilage repair tissue with high-resolution magnetic resonance imaging. Eur J Radiol. 2004;52:310–9.
60. Martin I, Miot S, Barbero A, et al. Osteochondral tissue engineering. J Biomech. 2007;40:750–65 [11].
61. McCauley TR, Kornaat PR, Jee WH. Central osteophytes in the knee: prevalence and association with cartilage defects on MR imaging. AJR Am J Roentgenol. 2001;176:359–64.
62. Melton JT, Wilson AJ, Chapman-Sheath P, Cossey AJ. TruFit CB bone plug: chondral repair, scaffold design, surgical technique and early experiences. Expert Rev Med Devices. 2010;7(3):333–41.
63. Menetrey J, Unno-Veith F, Madry H, Van Breuseghem I. Epidemiology and imaging of the subchondral bone in articular cartilage repair. Knee Surg Sports Traumatol Arthrosc. 2010;18:463–71.
64. Merrick MV. Investigation of joint disease. Eur J Nucl Med. 1992;19:894–901.

65. Minas T, Gomoll AH, Rosenberger R, Royce RO, Bryant T. Increased failure rate of autologous chondrocyte implantation after previous treatment with marrow stimulation techniques. Am J Sports Med. 2009;37:902–8.
66. Mithoefer K, Williams 3rd RJ, Warren RF, Potter HG, Spock CR, Jones EC, Wickiewicz TL, Marx RG. The microfracture technique for the treatment of articular cartilage lesions in the knee. A prospective cohort study. J Bone Joint Surg Am. 2005;87:1911–20.
67. Mont MA, Baumgarten KM, Rifai A, Bluemke DA, Jones LC, Hungerford DS. A traumatic osteonecrosis of the knee. J Bone Joint Surg Am. 2000;82:1279–90.
68. Nakamae A, Egebretsen L, Bhar R, Krosshaug T, Ochi M. Natural history of bone bruises after acute knee injury: clinical outcome and histopathological findings. Knee Surg Sports Traumatol Arthrosc. 2006;14:1252–8.
69. Orth P, Cucchiarini M, Kaul G, Ong MF, Gräber S, Kohn D, Madry H. Temporal and spatial migration pattern of the subchondral bone plate in a rabbit osteochondral defect model. Osteoarthritis Cartilage. 2012;20(10):1161–9.
70. Orth P, Cucchiarini M, Kohn D, Madry H. Alterations of the subchondral bone in osteochondral repair-translational data and clinical evidence. Eur Cell Mater. 2013;25:299–316.
71. Orth P, Goebel L, Wolfram U, Ong MF, Graber S, Kohn D, Cucchiarini M, Ignatius A, Pape D, Madry H. Effect of subchondral drilling on the microarchitecture of subchondral bone: analysis in a large animal model at 6 months. Am J Sports Med. 2012;40:828–36.
72. Pan J, Zhou X, Li W, Novotny JE, Doty SB, Wang L. In situ measurement of transport between subchondral bone and articular cartilage. J Orthop Res. 2009;27:1347–52.
73. Pape D, Filardo G, Kon E, van Dijk CN, Madry H. Disease-specific clinical problems associated with the subchondral bone. Knee Surg Sports Traumatol Arthrosc. 2010;18(4):448–62.
74. Pascarella A, Ciatti R, Pascarella F, et al. Treatment of articular cartilage lesions of the knee joint using a modified AMIC technique. Knee Surg Sports Traumatol Arthrosc. 2010;18(4):509–13.
75. Patrascu JM, Freymann U, Kaps C, Poenaru DV. Repair of a post-traumatic cartilage defect with a cell-free polymer-based cartilage implant: a follow-up at two years by MRI and histological review. J Bone Joint Surg Br. 2010;92(8):1160–3.
76. Perdisa F, Filardo G, Di Matteo B, Di Martino A and Marcacci M. Biological knee reconstruction: a case report of an Olympic athlete. Eur Rev Pharmacol Sci. 2014 (in press).
77. Pridie KH. A method of surfacing knee joints. Proceedings of the British orthopaedic association. J Bone Joint Surg Br. 1959;41:618–9.
78. Rand T, Brossmann J, Pedowitz R, Ahn JM, Haghigi P, Resnick D. Analysis of patellar cartilage. Comparison of conventional MR imaging and MR and CT arthrography in cadavers. Acta Radiol. 2000;41:492–7.
79. Reynolds JF, Smith DM, Lambin EF, Turner 2nd BL, Mortimore M, Batterbury SP, Downing TE, Dowlatabadi H, Fernández RJ, Herrick JE, Huber-Sannwald E, Jiang H, Leemans R, Lynam T, Maestre FT, Ayarza M, Walker B. Global desertification: building a science for dryland development. Science. 2007;316(5826):847–51.
80. Roemer FW, Bohndorf K. Long-term osseous sequelae after acute trauma of the knee joint evaluated by MRI. Skeletal Radiol. 2002;31:615–23.
81. Roemer FW, Frobell R, Hunter DJ, Crema MD, Fischer W, Bohndorf K, Guermazi A. MRI-detected subchondral bone marrow signal alterations of the knee joint: terminology, imaging appearance, relevance and radiological differential diagnosis. Osteoarthritis Cartilage. 2009;17:1115–31. OARS, Osteoarthritis Research Society.
82. Saris DB, Vanlauwe J, Victor J, Almqvist KF, Verdonk R, Bellemans J, Luyten FP. Treatment of symptomatic cartilage defects of the knee: characterized chondrocyte implantation results in better clinical outcome at 36 months in a randomized trial compared to microfracture. Am J Sports Med. 2009;37 Suppl 1:10S–919.
83. Scheck RM, Taboas JM, Segvich SJ, Hollister SJ, Krebsbach PH. Engineered osteochondral grafts using biphasic composite solid free-form fabricated scaffolds. Tissue Eng. 2004;10:1376–85.
84. Schiavone Panni A, Cerciello S, Vasso M. The management of knee cartilage defects with modified amic technique: preliminary results. Int J Immunopathol Pharmacol. 2011;24(1 Suppl 2):149–52.
85. Serre CM, Papillard M, Chavassieux P, et al. Influence of magnesium substitution on a collagen-apatite biomaterial on the production of a calcifying matrix by human osteoblasts. J Biomed Mater Res. 1998;42:626–33.
86. Shapiro F, Koide S, Glimcher MJ. Cell origin and differentiation in the repair of full-thickness defects of articular cartilage. J Bone Joint Surg Am. 1993;75:532–53.
87. Shepherd DE, Seedhom BB. Thickness of human articular cartilage in joints of the lower limb. Ann Rheum Dis. 1999;58:27–34.
88. Sherwood JK, Riley SL, Palazzolo R, et al. A three-dimensional osteochondral composite scaffold for articular cartilage repair. Biomaterials. 2002;23:4739–51.
89. Steadman JR, Rodkey WG, Rodrigo JJ. Microfracture: surgical technique and rehabilitation to treat chondral defects. Clin Orthop. 2001;391:S362–9.
90. Sugimoto K, Takakura Y, Tohno Y, Kuami T, Kawate K, Kadono K. Cartilage thickness of the talar dome. Arthroscopy. 2005;21:401–4.
91. Tampieri A, Sandri M, Landi E, et al. Design of graded biomimetic osteochondral composite scaffolds. Biomaterials. 2008;29:3539–46.

92. Trattnig S, Millington SA, Szomolanyi P, Marlovits S. MR imaging of osteochondral grafts and autologous chondrocyte implantation. Eur Radiol. 2007;17:103–18.
93. Van der Vis HM, Aspenberg P, Marti RK, Tigchelaar W, Van Noorden CJ. Fluid pressure causes bone resorption in rabbit model of prosthetic loosening. Clin Orthop Relat Res. 1998;350:201–8.
94. Van Dijk CN, Reilingh ML, Zengerink M, Van Bergen CJA. The natural history of osteochondral lesions in the ankle. Instr Course Lect. 2010;59: 375–86.
95. Van Dijk CN, Reilingh ML, Zengerink M, Van Bergen CJA. Osteochondral defects in the ankle: why painful? Knee Surg Sports Traumatol Arthrosc. 2010;18(5):570–80.
96. Vasiliadis HS, Danielson B, Ljungberg M, McKeon B, Lindahl A, Peterson L. Autologous chondrocyte implantation in cartilage lesions of the knee: Long-term evaluation with magnetic resonance imaging and delayed gadolinium-enhanced magnetic resonance imaging technique. Am J Sports Med. 2010;38:943–9.
97. Vellet AD, Marks PH, Fowler PJ, Munro TG. Occult posttraumatic osteochondral lesions of the knee: prevalence, classification, and short-term sequelae evaluated with MR imaging. Radiology. 1991;178: 271–6.
98. Verhagen R. Diagnostic guidelines for chronic ankle pain. Amsterdam: Thesis; 2004.
99. Wasiak J, Clar C, Villanueva E. Autologous cartilage implantations for full thickness articular cartilage defects of the knee. Cochrane Database Syst Rev. 2006;(3):CD003323.
100. Welsch GH, Zak L, Mamisch TC, Paul D, Lauer L, Mauerer A, Marlovits S, Trattnig S. Advanced morphological 3D magnetic resonance observation of cartilage repair tissue (MOCART) scoring using a new isotropic 3D proton-density, turbo spin echo sequence with variable flip angle distribution (PD-SPACE) compared to an isotropic 3D steady-state free precession sequence (True-FISP) and standard 2D sequences. J Magn Reson Imaging. 2011;33:180–8.
101. Zamber RW, Teitz CC, McGuire DA, Frost JD, Hermanson BK. Articular cartilage lesions of the knee. Arthroscopy. 1989;5:258–68.
102. Zengerink M, Struijs P, Tol J, Van Dijk CN. Treatment of osteochondral lesions of the talus: systematic review. Knee Surg Sports Traumatol Arthrosc. 2010;18(2):238–46.
103. Henderson I, Lavigne P, Valenzuela H, Oakes B. Autologous chondrocyte implantation: superior biologic properties of hyaline cartilage repairs. Clin Orthop Relat Res. 2007;455:253–61.
104. Pritzker KP, Gay S, Jimenez SA, Ostergaard K, Pelletier JP, Revell PA, Salter D, van den Berg WB. Osteoarthritis cartilage histopathology: grading and staging. Osteoarthritis Cartilage. 2006;14:13–29.
105. Zanetti M, Bruder E, Romero J, Hodler J. Bone marrow edema pattern in osteoarthritic knees: correlation between MR imaging and histologic findings. Radiology. 2000;215(3):835–40. doi:10.1148/radiology.215.3.r00jn05835. PubMed PMID: 10831707.

Hip Arthroscopy: Basis From Access to Repair in Femoroacetabular Impingement

12

Ali S. Bajwa, Nicolas Bonin, Michael Dienst, Frédéric Laude, Marc Philippon, Hassan Sadri, and Hatem Said

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12.1 Get into the Joint: 3 Ways to Manage the Capsule

12.1.1 How to Make an Easy Extracapsular Approach

Frédéric Laude

Deep and complex anatomic situation of the hip joint makes the establishment of arthroscopic portals more difficult and risky than in other joints. We describe a new technique to improve the efficiency and the safety of establishing standard hip arthroscopy portals (anterolateral, anterior +/− posterolateral portals): the extra-articular approach. Although special techniques and specialized instruments have been developed to make the hip arthroscopic procedure safer and easier, surgeons still need to use an image intensifier to

S. Zaffagnini et al. (eds.), *ESSKA Instructional Course Lecture Book*,
DOI 10.1007/978-3-642-53983-1_12, © ESSKA 2014

guide the approach. However, this procedure has several disadvantages, including exposure to radiation (surgeon and patients). In addition, fluoroscopy provides only 2-dimensional images for the surgeon, especially because it is not convenient to change the C-arm machine position during the operation which can lead to potential damage to nerves, vessels, or cartilage during the approach.

12.1.1.1 Technique

Patients were positioned supine on the traction table under general anesthesia. The affected limb was in a slight flexion without any traction on the limb at the beginning. The surface projections of anatomic structures around the hip joint were marked. The anterolateral portal was first placed at the level of the trochanter. The arthroscopic trocar was introduced under the fascia lata until the contact with the femoral neck. Keeping the contact with the anterior femoral neck, a 70° scope was introduced. The anterior portal, which was at least 5 cm away from the first one (anterior to the fascia lata and more distal), was then established. A fad-pat area (muscle-free zone) was then identified and removed by placing a shaver in the anterior portal. The fad-pat was removed; the capsule was exposed and incised with an electrocoagulation device. The capsulotomy was performed in a "T" shape, and the labrum and cartilage surfaces were identified before traction on the hip. If necessary, the posterolateral portal was established by the same procedure.

12.1.1.2 Results

From September 2010 to May 2011, we routinely performed this arthroscopic approach for hip impingement treatment in 80 patients (47 males and 33 females). The mean age was 30 years (12–52). We never had to use fluoroscopy to establish the different portals. The mean duration of the surgery was 57 min (30–105 min), and the time during which we applied traction to the hip was restricted to an average of 18 min (maximum 50 min – minimum 5 min). The mean time to access to the capsule and to enter the joint was 12 min (3–25 min). No nerve or vessel complication occurred in any of our 80 cases. Labral and chondral injury do not occur, because of the direct arthroscopic visualization of the joint before traction and entrance in the joint. Because of the short duration of the traction, no perineal soft tissue complication occurred.

Conclusion

We believe that extra-articular arthroscopic approach without fluoroscopy is a safe and reproducible method to establish hip arthroscopy portals. For surgeons this procedure can reduce the risk of potential complications mainly due to traction time and is easy to incorporate into practice.

12.1.2 How to Make an Easy Peripheral Compartment Approach

Hatem Said

Peripheral compartment hip arthroscopy was described by Klapper et al. [1], Dorfmann and Boyer [2] and later popularized by Dienst et al. [3].

It has many advantages, as you are entering into a wider space, which makes it technically easier and reduces the steep learning curve associated with learning hip arthroscopy. It does not use traction [3], thus avoiding most of the complications of hip arthroscopy like neurovascular problems [4] and perineal injuries. After completion of peripheral compartment procedures like cam osteoplasty, synovectomy, or loose body removal, you can then apply traction and enter the central compartment under vision, thus reducing the risk of labral or chondral injury.

12.1.2.1 Technique

Peripheral access mostly uses the position of hip flexion to relax the anterior capsule; thus to establish the portals, the hip is flexed to 30°. The main working portals are the PAL (proximal anterolateral portal) and the mid-anterior or DAL (distal anterolateral portals). The PAL lies 1/3 of the distance on a line from ASIS to the greater trochanter. The direction is towards the intersection of line A from ASIS distally and B from the greater trochanter anteriorly. Capsular resistance is encountered, and then the neck is felt, which is then slid on the anterior surface of the neck. The DAL is performed about the same distance from the greater trochanter but in a more distal manner.

The direction of entry is almost perpendicular to the skin or directed slightly proximal. Once the femur is felt, the needle is directed just anterior to the neck to enter the capsule, which is thinner than in the PAL and provides less resistance.

The easiness of this technique allows that we do it with or without C-Arm guidance, by using the anatomic surface landmarks [5]. To gain access to all parts of the peripheral compartment, the leg position varies from extension to flexion 70°. to visualize different parts of the head and neck, while abduction and adduction aims at relaxing the lateral or medial capsule. Capsular thinning by the shaver and radio frequency is done to ease visualization and maneuvering, while capsulotomy is avoided at this stage to maintain fluid distention. The diagnostic round trip is then performed to visualize all parts of the head and neck [6]. After completion, the leg is extended, traction is applied, and the central compartment is accessed under direct vision.

In this presentation, we present the technique of gaining easy access to the peripheral compartment of the hip.

12.1.3 How to Make an Easy Central Compartment Approach

Marc Philippon

The key to making an easy central compartment approach is following a number of steps that will ensure proper muscle relaxation, patient positioning, adequate pelvis stabilization, sufficient joint distraction, and precise portal placement.

12.1.3.1 Muscle Relaxation

Full muscle relaxation is of paramount importance. To obtain this, the anesthesiologist may choose to do a general anesthesia with intubation plus an intravenous muscle relaxant (neuromuscular blocker). Another option is to use a spinal, with or without a combined epidural, which allows the patient to remain awake with mild sedation.

12.1.3.2 Patient Positioning

We typically perform hip arthroscopy in a modified supine position. The effected leg is placed in 10° of flexion, 30° of internal rotation, 10° of lateral tilt, and neutral abduction. Internal rotation makes the femoral neck parallel to the floor, and a slight lateral tilt will help to prevent the patient from sliding laterally when traction is applied. Both feet must be padded and wrapped in a tight and secure manner, while avoiding excessive pressure that may compromise blood circulation. Foot slippage is a common cause for distraction loss, so the foot wrapping must be adequate.

12.1.3.3 Pelvis Stabilization

To keep the pelvis in place, it is necessary to have a properly sized and padded perineal post. Proper padding is important to prevent pudendal nerve neuraraxia as well as skin injuries. Countertraction is applied to the non-affected side, to prevent the pelvis from tilting sideways. This leg will typically be placed at about 45° of abduction and neutral rotation.

12.1.3.4 Joint Distraction

The next step is to apply proper joint distraction. First, limb positioning must be rechecked – if traction is applied under adduction or abduction, proper distraction is more difficult to obtain. Then, traction is applied progressively, up to around 30 lb of force. A pop may be felt; a joint separation is confirmed under fluoroscopy. Adduction is applied under traction, to add a lateral force vector that will ensure the joint to be distracted properly. Initially, about 1 cm of distraction is sufficient.

12.1.3.5 Portal Placement

The first portal is the anterolateral one. This portal is made 1 cm proximal and 1 cm anterior to the tip of the greater trochanter. Careful palpation is necessary to assure the skin incision is made at the right point. A spinal needle is inserted and should pierce the capsule as close as possible to the femoral neck. By keeping it away from the acetabular rim, labrum perforation can be avoided. If in doubt, a C-arm can be used at this step to ensure the spinal needle is at the correct position. A nitinol guide wire is passed and a trocar is inserted, followed by the arthroscopic camera.

The camera should then be turned anterosuperiorly and a “V” should be visualized: the proximal border is formed by the anterior labrum and the distal border by the femoral head. Between them, the anterior capsule should be visible, and this is where the mid-anterior portal should be made.

Attention is turned to the skin: the correct position for the mid-anterior portal is around 4 cm distal and 4 cm medial to the anterolateral portal, at a 45° angle from a longitudinal line passing through the anterolateral portal. The mid-anterior portal is made halfway between a longitudinal line passing through the ASIS and a longitudinal line passing through the anterolateral portal.

The spinal needle is then inserted, and the point where it touches the capsule is visible with the scope from within the joint. At this point, one can carefully choose the exact point where to pierce the capsule. The next step is to use an arthroscopic blade to cut the capsule between these portals (interportal capsulotomy). This will allow for ample access to the central compartment, from the two portals.

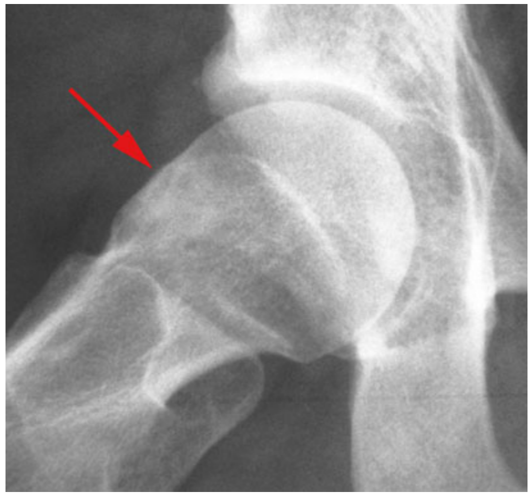


Fig. 12.1 Anterosuperior cam deformity (*arrow*) visible on Ducroquet view

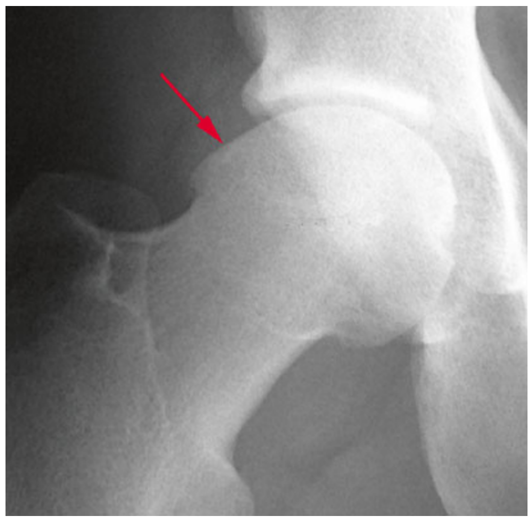


Fig. 12.2 Superior cam deformity (*arrow*) visible on AP view

12.2 How to Manage the Cam

Nicolas Bonin

Cam impingement generally results from excessive bone (bump) at the anterosuperior femoral head–neck junction or when there is a flattening and loss of the normal anterior offset concavity between the femoral head and neck [7–9] (Fig. 12.1). With a better understanding of FAI, we now know that cam can be at the superior or the inferior part of the neck (Fig. 12.2). The aim of the cam management is to restore the normal sphericity of the femoral head that is perturbed.

12.2.1 Cam Assessment

The first step is an adequate assessment of the cam deformity. Radiographic methods for defining cam impingement have advanced in the last few years. Cam lesion is well quantified by the alpha angle measurement [8] on MRI and flattening of the normal anterior offset by the head–neck anterior offset measurement. You must evaluate the limits of the cam deformity from the posterosuperior to the anteroinferior part of the neck but also from proximal to distal in order to make a complete resection. Radial CT or MR imaging (around the femoral neck) is of great help. Prior to surgery, you should have a perfect planning in mind to know exactly what to take down.

12.2.2 Technique

You can either start with the peripheral compartment to do the cam or start with the central compartment to treat acetabular chondrolabral lesions before performing cam Resection.

I start my cam resection looking at the posterosuperior aspect of the head under traction, so I can detect the beginning of the flat part of the head, which can be sometimes under the labrum. At this moment, the 70° scope is in the mid-anterior portal, looking posteriorly, and the burr is in the anterolateral portal. The burr is placed at the beginning of the flat aspect of the head in order to make the proximal landmark, and then it can slowly go distally to join the normal neck. There is almost always a step you can find between the cam and the normal neck that tells you when to stop proximally and deeply. As soon as posterosuperior cam is done, traction can be released and the hip flexed to 30°. Again, the nonspherical aspect of the head is searched and burred to join the normal neck. Step by step the hip is flexed at 50° and then 70° to use the capsulotomy as a mobile window to go down to the neck, avoiding to make it larger. Once you cannot go farther, portals are switched to have the best vision and access to the anteroinferior part of the neck. You should stop searching for cam only once you have reached the inferior part of the neck located by the medial synovial fold. With the scope in the anterolateral portal, you come back to extension, step by step, to control head sphericity from another view and to complete resection if needed.

At the end of procedure, when you are back to extension, the hip is tested under direct scope vision, in flexion and internal rotation and in abduction, internal, and external rotation, to confirm your resection is adequate. In case of any doubts, you should use fluoroscopy (Fig. 12.3).

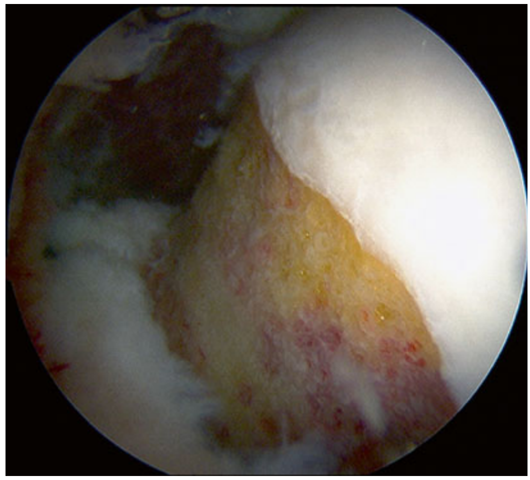


Fig. 12.3 Arthroscopic final view of the cam resection

Conclusion

Residual cam is one of the most common causes of failed hip arthroscopy [10]. Visualizing abnormal head sphericity limits under arthroscopy is not effortless, and preoperative landmarks must be well known prior to surgery. Fluoroscopy should be used during the procedure in case of any doubts during the Resection.

12.3 How to Manage the Pincer

Ali S. Bajwa

Pincer lesion or over-coverage of the acetabulum can lead to femoroacetabular impingement (FAI) on its own or in combination with a cam lesion. The exact definition of pincer is controversial; however, a center-edge (CE) angle of >39° is generally considered as a pincer lesion [11]. This should not distract the surgeon from the fact that pincer is a 3-dimensional phenomenon. Pincer lesion can be due to global over-coverage such as in coxa-profunda and protrusio acetabuli. There can indeed be focal anterior or posterior over-coverage, the former present in a classic acetabular retroversion.

12.3.1 Pincer Assessment

The first step is an adequate assessment of the pincer deformity. This starts with careful history and clinical examination. It is imperative to find out how the structural deformity of the hip is affecting the patient. This takes into account patient's sports and daily activities. The radiological assessment includes a plain radiograph of the pelvis in anteroposterior (AP) and a cross-table lateral planes, MR arthrogram, and CT scan in selected cases. Often there is an associated cam lesion, giving rise to the mixed picture.

12.3.2 Origin of Symptoms

Symptoms may result purely from the impingement phenomenon or an underlying labral tear or chondral damage. There is often a mixed picture with chondrolabral damage circumferentially in a global pincer.

12.3.3 Technique

The arthroscopic treatment plan includes gaining access in a rather snug joint, assessment of damage, correction of bony structural over-coverage, repair or reconstruction of the labrum, and addressing the chondral damage with a suitable chondral regeneration technique. A final dynamic assessment is essential and is best seen from the peripheral compartment. I undertake arthroscopy in the lateral position with a dedicated hip distractor system using distal anterolateral, posterolateral, and superolateral portals for pincer lesion. For mild to moderate pincer lesion, I generally start from the central compartment. However, for severe and some moderate pincer with varus femoral neck, I start from the peripheral compartment. Occasionally the hip is not distractable; however, the acetabular overhang is such that the central compartment can be difficult to access safely. In this instance, again a peripheral first approach is used. Most of mild to moderate pincers can be treated without formal labral takedown while starting the rim trimming from the retro-labral sulcus. However, for severe global pincer, labral takedown may be required but only if the labrum is not ossified! In many global pincer lesions, the labrum is rudimentary or ossified, and the resection directly starts with the burr. It is important to keep checking under image intensifier to avoid iatrogenic dysplasia.

Labrum is assessed and if needed stabilized with suture anchors 8 mm apart. The cartilage regeneration procedures are undertaken at the end as they may require a dry field.

12.4 How to Manage the Labrum

Michael Dienst

In pincer FAI, the technique of trimming the acetabular rim depends on the condition of the acetabular labrum (Fig. 12.4). In many cases, the labrum can be saved but needs to be temporarily detached because of an unstable chondrolabral separation and refixed after the acetabular rim is trimmed. If the chondrolabral junction is intact, the labrum bruised, and the width of the labrum sufficient, the labrum can be recessed. By doing this, the labrum may be destabilized, thus additional refixation is needed. If the labrum is of bad quality, small, or ossified, the labrum needs to be removed before the rim is reduced. Usually, where sufficient acetabular coverage and overall hip stability is achieved, the result will be good without the need of additional procedures. However, there may be cases where the hip is destabilized by removing the labrum and/or rim. In those cases, subsequent, staged procedures such as labral reconstruction or a bony reorientation of the acetabulum may be necessary.

12.5 Arthroscopic Treatment of Cartilage Lesions in the Hip

Hassan Sadri

12.5.1 Rational of Arthroscopic Treatments of Chondral Lesions of the Hip

Fully arthroscopic treatment of chondral lesions of the hip is feasible. Most chondral lesions of the hip are focal lesions and associated with

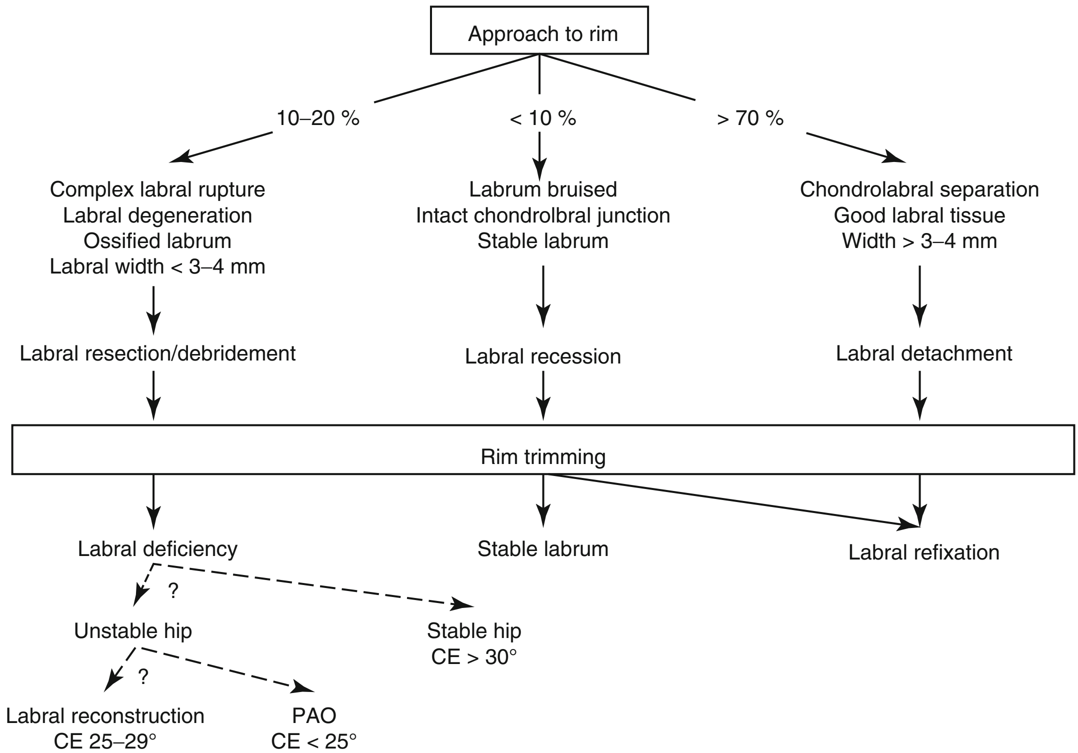
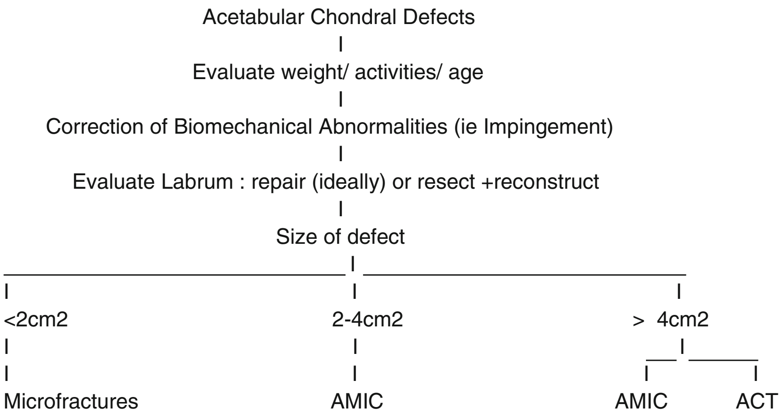


Fig. 12.4 Management of the acetabular labrum in pincer FAI

femoroacetabular impingement. These lesions are most often located in the anterosuperior acetabulum but can be located on the head. The goal of the treatment is to fill the chondral defect by the best available filler, i.e., the best quality fibrocartilage or cartilage. Some studies have however shown that after a period of 2 years, the fibrocartilage transforms into cartilage in the AMIC technique. By filling the defect, the increase of pressure on the adjacent living cartilage is reduced by 200 %, thus protecting it. When chondral flaps are present, some authors have suggested that these contain living chondrocytes and have suggested that these be "glued" in with collagen glue. This would again be the best defect filler available in these cases. In cases of stage IV lesions, the treatment is divided in function of the size of the lesion after debridement to stable margins (see flow chart below). After debridement, it is essential to remove the calcified subchondral bone layer. As in other joints mechanical abnormalities have to be treated (i.e., correction of femoroacetabular impingement) and if possible the labrum repaired or reconstructed. In these cases, the simultaneous arthroscopic femoroacetabular correction, labral repair/reconstruction and cartilage repair (AMIC and ACT) can be challenging and should be performed by very experienced arthroscopic hip surgeons. Standard portals are used (anterolateral, posterolateral, and anterior). The necessary time of traction is usually longer than 2 h, and complications related with traction have to be neutralized. It is also essential to proceed to a structured rehabilitation protocol as this has been shown to produce better healing.



12.5.2 Rehabilitation

The postoperative management following abrasion, microfracture, AMIC, and ACT is as critical as the respective surgical procedure. Postoperative weight bearing consists of 5–10 kg (toe touch) for 6–8 weeks. Additionally, they are provided with continuous passive motion (CPM) set at one cycle per minute using the largest range of motion tolerated for 8 weeks during 8 h per day. If CPM is unavailable, patients are instructed to perform full hip active assisted range of motion 300 (30 times every hour) times daily, for example, by keeping their heels on a bed in the supine position and flexing their hips from 0° to 90° as tolerated.

Following the 6- to 8-week period of protected weight bearing, patients are instructed to begin active ROM exercises and progress to full weight bearing. No cutting, twisting, or jumping sports are allowed until at least 6 months postoperatively.

Up to 8 weeks of non-weight bearing is required to promote early fibrous tissue maturation.

The beneficial effect of motion on the healing of articular injuries is well documented. The principles of CPM include enhanced nutrition and metabolic activity of articular cartilage, the stimulation of pluripotent mesenchymal cells to differentiate into articular cartilage rather than fibrous tissue or bone, and the acceleration of healing of both articular cartilage and periarticular tissues. A slower rate of motion is superior to a faster rate.

Other studies demonstrate that in animals, CPM promotes early and more complete cartilage metaplasia and that pressure-dependent matrix flow from the surrounding articular surfaces may positively influence this metaplasia. Additionally, defects exposed to CPM rather than immobilization or intermittent motion exhibit greater defect fill.

Synovial fluid is a source of nutrition for cartilage proliferation and may contain chondrotrophic properties. The pumping action of CPM improves cartilage nutrition and helps to clear hemarthrosis. Moreover, physiologic exercise increases the volume of synovial fluid and immobilization is associated with reduced synovial fluid.

References

1. Klapper R, Dorfmann H, Boyer T. Hip arthroscopy without traction. In: Byrd JWT, editor. Operative hip arthroscopy. New York: Thieme; 1998. p. 139–52.
2. Dorfmann H, Boyer T. Arthroscopy of the hip: 12 years of experience. Arthroscopy. 1999;15(1):67–72.
3. Dienst M, Seil R, Kohn DM. Safe arthroscopic access to the central compartment of the hip. Arthroscopy. 2005;21(12):1510–4.
4. Said HG, Steimer O, Kohn D, et al. Vascular obstruction at the level of the ankle joint as a complication of hip arthroscopy. Arthroscopy. 2011;27(11):1594–6.
5. Masoud MA, Said HG. Intra-articular hip injection using anatomic surface landmarks. Arthrosc Tech. 2013;2(2):e147–9.
6. Dienst M, Gödde S, Seil R, Hammer D, Kohn D. Hip arthroscopy without traction: In vivo anatomy of

the peripheral hip joint cavity. Arthroscopy. 2001;17(9):924–31.
7. Siebenrock KA, Schoeniger R, Ganz R. Anterior femoro-acetabular impingement due to acetabular retroversion. Treatment with periacetabular osteotomy. J Bone Joint Surg Am. 2003;85-A(2):278–86.
8. Ito K, Minka 2nd MA, Leunig M, et al. Femoroacetabular impingement and the cam-effect. A MRI-based quantitative anatomical study of the femoral head-neck offset. J Bone Joint Surg Br. 2001;83(2):171–6.
9. Notzli HP, Wyss TF, Stoecklin CH, et al. The contour of the femoral head-neck junction as a predictor for the risk of anterior impingement. J Bone Joint Surg Br. 2002;84(4):556–60.
10. Bogunovic L, Gottlieb M, Pashos G, et al. Why do hip arthroscopy procedures fail? Clin Orthop Relat Res. 2013;471(8):2523–9.
11. Tonnis D, Heinecke A. Acetabular and femoral anteversion: relationship with osteoarthritis of the hip. J Bone Joint Surg Am. 1999;81(12):1747–70.

Achilles Tendon Rupture Treatment: Still a Weak Spot?

13

Umile Giuseppe Longo and James Calder

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13.1 Introduction

The Achilles tendon (AT), the largest and strongest human tendon, is also the most frequently ruptured [8]. The incidence of AT ruptures has increased in the last decade and usually occurs in sedentary population playing sport occasionally, especially in their third or fourth decade of life. Most of AT ruptures occur during sport activities, and biomechanical and biochemical changes related to ageing may play a significant role [7]. Even though many theories have been developed to explain the aetiology of AT rupture, no agreement was found in the literature. Gastrocnemius-soleus dysfunction, gender, age, changes in training pattern, poor technique, previous injuries, footwear, poor tendon vascularity, and degeneration are frequently attributed to AT ruptures. Infective diseases, hyperthyroidism, neurological conditions, renal insufficiency, arteriosclerosis, inflammatory and autoimmune conditions, hyperuricaemia, genetically determined collagen abnormalities, and high serum lipid concentration can be associated with AT ruptures [9].

Two main theories have been proposed to clarify the aetiology of the AT ruptures: the "degenerative theory" and the "mechanical theory". The first theory states that the chronic degeneration of the tendon leads to a rupture without excessive loads being applied. The second theory states that degeneration of the tendon may be caused by factors such as chronic overloading or microtrauma, physiological alterations in the tendon, and pharmacological treatments [1].

S. Zaffagnini et al. (eds.), *ESSKA Instructional Course Lecture Book*,
DOI 10.1007/978-3-642-53983-1_13, © ESSKA 2014

The diagnosis of AT ruptures starts with a collection of the history followed by physical exam. Nevertheless, the 20–25 % of AT ruptures are not diagnosed by the first examining doctor [9].

Patient with ruptured AT usually report a history of pain in the affected leg and the feeling that, at the time of injury, they had been kicked in the posterior aspect of the lower leg. The inability to weight bear and the weakness or stiffness of the affected ankle is common.

On clinical examination, diffuse oedema and bruising are usually present, and, unless the swelling is severe, a palpable gap may be felt along the course of the tendon, most frequently 2–6 cm proximal to the insertion of the tendon. Inspection and palpation should be followed by other tests to confirm the diagnosis, such as Simmonds and Matles test and O'Brien and Copeland tests. Ultrasonography is largely considered the primary imaging method for the diagnosis of AT ruptures.

13.2 State-of-the-Art Treatment

The management of a patient with an acute rupture of the AT can be classified into operative and conservative and usually depends on the preference of the surgeon and the patient. The surgical management consist on minimally invasive or open while the conservative management consist on immobilisation or functional bracing [12, 14]. More evidence is available for percutaneous techniques instead of open surgery and also for the use of early mobilisation [9–11].

Surgery has been the method of choice in the last two decades especially in athletes and young people or in case of delayed ruptures, while conservative management in nonathletic patients. Open operative management of acute AT ruptures significantly reduces the risk of re-rupture compared with nonoperative treatment, while is associated with a significantly higher risk of wound healing problems that can be reduced by performing surgery percutaneously [8].

The objective of management of AT rupture is to minimise the morbidity of the injury, optimise rapid return to full function, and prevent complications.

13.3 Conservative Management

The immobilisation in a below-knee plaster cast in gravity equinus position for 4 weeks followed by a more neutral position for a further 4 weeks is considered the most common nonoperative protocol of management of AT rupture. The reports on early functional treatment suggest good functional outcome and low re-rupture rates.

Following immobilisation, a profound alteration of muscle morphology and physiology occurs. Despite the gastrocnemius muscle, which is a bi-articular muscle able to move when a short leg cast is used, the soleus muscle is particularly susceptible to immobilisation. Given the presence of a high proportion of type I muscle fibres in human soleus, this is particularly susceptible to atrophy if immobilised.

13.4 Minimally Invasive Operative Treatment

Minimally invasive repair of acute traumatic ruptures of the Achilles tendon may produce lower complications compared to open repair [3, 4]. In 1977, Ma and Griffith were the first that proposed the percutaneous repair for the management of AT ruptures, in order to find a compromise between the open surgical and nonsurgical managements. By producing six small stab incisions across the medial and lateral borders of the tendon, they passed a suture through the tendon using these incisions. In their series, there were not re-rupture while two minor, non-infectious skin complications occur.

Carmont and Maffulli described a technique in which three transverse incisions are used, minimising the chance of sural nerve injury, allowing an even less invasive approach to the tendon that permits the accurate apposition of the tendon ends. Ismail et al. [5] compared the mechanical properties of the Achillon mini-incision technique with the long established Kessler method, concluding that the strength of the repair was related to tendon diameter and that there were no differences between the two techniques.

The primary stability of two minimally invasive procedures of Achilles tendon (AT) repair, namely, a modified percutaneous repair of ruptured AT and the Achillon suture configuration, provided similar biomechanical performance [6].

13.5 Open Surgery

In the last years, open surgical repair was considered the gold standard for the management of AT ruptures in young fit individuals. Moreover, the numerous advances in surgical techniques, such as in postoperative rehabilitation protocols have encouraged many surgeons to favour direct tendon repair [13]. Besides, the excellent results of surgical repair concerning re-rupture rate and tendon strength, such as calf trophism, may help many athletes to return to pre-injury physical activities [2].

Different kinds of operative techniques can be performed to repair ruptured ATs, ranging from simple end-to-end suturing by Bunnell- or Kessler-type sutures to more complex repairs using fascial reinforcement or tendon grafts, artificial tendon implants, using materials such as absorbable polymer-carbon fibre composites, xenograft ECM scaffolds, Marlex® mesh, and collagen tendon prostheses. Primary augmentation of the repair with the plantaris tendon, a single central or two (one medial, one lateral) gastrocnemius fascial turndown flaps, the peroneus brevis tendon, the gracilis tendon, the bone-patellar tendon, the bone-quadriceps tendon, the semitendinosus tendon, and the free hamstring tendon transfer was also performed. However, there is no evidence that, in acute AT ruptures, this is better than a non-augmented end-to-end repair. Recently, platelet-rich plasma (PRP) was also used alone for the management of chronic AT tendinopathy or in association with open repair in case of acute AT ruptures. The randomised control trials that evaluated the role of PRP in acutely ruptured AT or in chronic AT tendinopathy concluded that PRP is not useful for the management of these pathologies. In our opinion, the use of augmentation should be preferred only when dealing with delayed repairs and chronic tears and in the management of re-ruptures.

13.6 Future Treatment Options

In the last few decades, several emerging strategies including tissue engineering with mesenchymal stem cells have been proposed to enhance tendon healing. They hold the promise to yield more successful outcomes for the management of patients with tendon pathology. Current in vitro studies support the application of these cell-based therapies for the regeneration of tendon tissues. However, these cell-based strategies have been investigated only in preclinical studies, and the role of stem cells needs to be confirmed.

13.7 Take-Home Message

In the last decades, the incidence of AT ruptures is increased while the evidence for best management is still debated. Conservative treatment and early mobilisation achieves excellent results in elderly or selected patients, but the re-rupture rate which is associated is not acceptable in young individuals. Open surgery is frequently associated with higher risk of superficial skin breakdown and wound problems, which can be prevented by performing percutaneous repair. The percutaneous repairs, performed under local anaesthesia and followed by early functional rehabilitation, are becoming increasingly common and must be taken into account in some selected patients.

References

1. Ames PR, Longo UG, Denaro V, Maffulli N. Achilles tendon problems: not just an orthopaedic issue. Disabil Rehabil. 2008;30:1646–50. doi:10.1080/09638280701785882.
2. Carmont MR, Silbernagel KG, Nilsson-Helander K, Mei-Dan O, Karlsson J, Maffulli N. Cross cultural adaptation of the Achilles tendon Total Rupture Score with reliability, validity and responsiveness evaluation. Knee Surg Sports Traumatol Arthrosc. 2013;21:1356–60. doi:10.1007/s00167-012-2146-8.
3. Doral MN. What is the effect of the early weight-bearing mobilisation without using any support after endoscopy-assisted Achilles tendon repair? Knee Surg Sports Traumatol Arthrosc. 2013;21:1378–84. doi:10.1007/s00167-012-2222-0.

4. Doral MN, Bozkurt M, Turhan E, Dönmez G, Demirel M, Kaya D, Ateşok K, Atay OA, Maffulli N. Achilles tendon rupture: physiotherapy and endoscopy-assisted surgical treatment of a common sports injury. Open Access J Sports Med. 2010;1:233–40. doi:10.2147/OAJSM.S10670.
5. Ismail M, Karim A, Shulman R, Amis A, Calder J. The Achillon achilles tendon repair: is it strong enough? Foot Ankle Int. 2008;29:808–13. doi:10.3113/FAI.2008.0808.
6. Longo UG, Forriol F, Campi S, Maffulli N, Denaro V. A biomechanical comparison of the primary stability of two minimally invasive techniques for repair of ruptured Achilles tendon. Knee Surg Sports Traumatol Arthrosc. 2012;20:1392–7. doi:10.1007/s00167-011-1868-3.
7. Longo UG, Petrillo S, Maffulli N, Denaro V. Acute achilles tendon rupture in athletes. Foot Ankle Clin. 2013;18:319–38. doi:10.1016/j.fcl.2013.02.009.
8. Longo UG, Ronga M, Maffulli N. Acute ruptures of the achilles tendon. Sports Med Arthrosc. 2009;17:127–38. doi:10.1097/JSA.0b013e3181a3d767.
9. Maffulli N, Longo UG, Maffulli GD, Khanna A, Denaro V. Achilles tendon ruptures in diabetic patients. Arch Orthop Trauma Surg. 2011;131:33–8. doi:10.1007/s00402-010-1097-0.
10. Maffulli N, Longo UG, Maffulli GD, Khanna A, Denaro V. Achilles tendon ruptures in elite athletes. Foot Ankle Int. 2011;32:9–15. doi:10.3113/FAI.2011.0009.
11. Maffulli N, Longo UG, Ronga M, Khanna A, Denaro V. Favorable outcome of percutaneous repair of achilles tendon ruptures in the elderly. Clin Orthop Relat Res. 2010;468:1039–46. doi:10.1007/s11999-009-0944-1.
12. Nilsson-Helander K, Silbernagel KG, Thomeé R, Faxén E, Olsson N, Eriksson BI, Karlsson J. Acute achilles tendon rupture: a randomized, controlled study comparing surgical and nonsurgical treatments using validated outcome measures. Am J Sports Med. 2010;38:2186–93. doi:10.1177/0363546510376052.
13. Nilsson-Helander K, Swärd L, Silbernagel KG, Thomeé R, Eriksson BI, Karlsson J. A new surgical method to treat chronic ruptures and reruptures of the Achilles tendon. Knee Surg Sports Traumatol Arthrosc. 2008;16:614–20. doi:10.1007/s00167-008-0492-3.
14. Olsson N, Silbernagel KG, Eriksson BI, Sansone M, Brorsson A, Nilsson-Helander K, Karlsson J. Stable surgical repair with accelerated rehabilitation versus nonsurgical treatment for acute Achilles tendon ruptures: a randomized controlled study. Am J Sports Med. 2013. doi:10.1177/0363546513503282.

Tips and Pitfalls in Unicompartmental Knee Arthroplasty (UKA)

14

Jean-Noel Argenson, John Bellemans, Christopher Dodd, Julian Dutka, Nikolay Kornilov, David Murray, Sebastian Parratte, and Pawel Skowronek

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14.1 Introduction

Unicompartmental arthroplasty (UKA) is a well-established treatment option for unicompartmental osteoarthritis of the knee. There are many advantages of UKA over total knee arthroplasty (TKA). In particular UKA has as good pain relief if not better, there is a better "feel," the range of motion is greater, UKA has normal kinematics, and the complications are fewer with UKA and less severe when compared to TKA.

There is, however, one major disadvantage of UKA. In all national registries, there is a higher revision rate with UKA. This factor, and others, has limited the use of UKA to about 5–8 % of knee replacements. Despite this major disincentive, there are many who wish to pursue UKA and wish to optimize it as a procedure in their practices.

This course will provide a review of how to optimize the treatment of patients who require UKA by addressing many of the important issues confronting the UKA surgeon.

S. Zaffagnini et al. (eds.), *ESSKA Instructional Course Lecture Book*,
DOI 10.1007/978-3-642-53983-1_14, © ESSKA 2014

14.2 Tips for Preoperative Assessment Before Unicompartmental Knee Replacement

David Murray

In order to optimize preoperative assessment, it is necessary to have a clear understanding of the principles underlying selection of patients for UKA. Traditionally the decision about whether to do a UKA or a TKA depended on the balance of indications and contraindications, the indication being damage to a single compartment of the knee, with pain localized to that part of the knee. Contraindications are not well defined and vary between authors but tend to relate to the knee and the patient in general. Knee-related contraindication includes damage to the lateral compartment, the patellofemoral joint, and the ligaments. These may be identified in many ways such as the presence of pain or radiographic changes related to these compartments, lateral thrust or subluxation, and large (>10°) or non-correctable deformities. Patient-related contraindications include being young, very old, active, or obese. With this approach about 5 % of knees needing replacement are considered ideal for UKR, and it is believed that if the rate is increased, the revision rate will also increase.

Thirty years ago John Goodfellow proposed a fundamentally different approach for the mobile-bearing Oxford UKR. His view was that if patients had significant symptoms related to the knee and a specific disease entity, anteromedial osteoarthritis (AMOA), then these patients should have UKA and there should be no contraindications. The features of AMOA are well defined and include bone-on-bone medial osteoarthritis, functionally intact ligaments, and full-thickness cartilage laterally. It has subsequently been shown that with this approach, none of the contradictions described above compromise the outcome so they can all safely be ignored (except for severe damage to the lateral part of the patellofemoral joint with bone loss and subluxation). With this approach about 50 % of knees needing replacement are appropriate for UKA.

The diagnosis of AMOA is made radiographically. On a well-aligned lateral radiograph (femoral condyles overlapping), if the tibial defect does not reach the back or cannot be seen, then there is a 95 % chance that the ACL is functionally intact. Medial bone on bone must be identified: this can be done using an AP weight bearing, a Rosenberg, or a varus stress radiograph; if bone on bone is not seen radiographically, then a UKA should not be done unless bone on bone is demonstrated arthroscopically. A valgus stress radiographs (in 20° flexion) should be used to determine if there is full-thickness cartilage laterally and a functionally intact MCL (correctable intra-articular deformity). Although it has not been proven, the two most important features are likely to be a functionally intact ACL and bone on bone medially.

Recently, data from the England and Wales National Joint Registry has been analyzed to identify the optimal usage of UKA [1]. The majority of surgeons use UKR for 5 % or less of their knee replacements (ratio UKA:TKA is 5 %:95 %), in other words they appear to be using the traditional approach to UKA. When the UKA usage is 5 % or less, the 5-year survival of UKA is 90 %. With increasing usage up to 20 %, the results improve dramatically. Therefore, surgeons using the traditional approach to UKR should either stop doing UKR or increase their usage to 20 %. It is however not clear, with fixed-bearing UKR, what the optimal indications and contraindications are to achieve 20 % usage. With the Oxford knee, the results continue to improve with increased usage reaching a maximum at about 50 %, at which stage the 5-year survival is 96 %. This would suggest that, for the Oxford knee, Goodfellow's approach was correct and that all patients with AMOA should have a UKR. These patients will then benefit from all the advantages of UKA without the disadvantage of a high revision rate.

14.3 Tips for Fixed UKA

Sebastian Parratte

14.3.1 Patient Selection and Indications

Preoperative physical exam should ensure a range of knee flexion >100°, full knee extension, and a stable knee in the anteroposterior (AP) and sagittal planes. The patellofemoral joint should be clinically asymptomatic.

The radiological analysis should include full-length x-rays, AP and ML view of the knee, skyline views, and stress radiographs. The radiographic analysis should ensure that there is no patellofemoral loss of joint space on skyline views at 45° of flexion and the presence of full-thickness articular cartilage in the uninvolved compartment, with full correction of the deformity to neutral on stress radiographs.

On the lateral view of the tibia, the tibial slope should be calculated to try to reproduce the same angle during the procedure or not. The height of the patella analyzed as a patella baja will limit the exposure during MIS procedures.

14.3.2 Implant Selection

A cemented pegged metal-backed implant is recommended to ensure the stability of the tibial tray.

To limit wear over time, which can be a problem with fixed-bearing implants in young patients, cross-linked polyethylene is recommended.

High-flexion femoral implant with round design limits the risk of overloading the tibial component.

14.3.3 Technique

Limited incisions are now currently used but the size should be large enough to ensure proper intraoperative visualization to optimize implant positioning.

Osteophytes should be properly removed including those in the notch to avoid any impingement against the ACL over the time.

Linked distal and proximal cuts can be used to ensure proper alignment in extension.

Tibial frontal cut should be done around 2–4 mm below the lowest point of the tibial plateau to preserve the strength of the subchondral bone. Too distal a tibial cut will lead to pain.

The slope should be controlled. If there is any doubt concerning the ACL, less slope should be applied into the implant.

The sagittal tibial cut should preserve the ACL.

Rotation of the femur should be checked in flexion and in extension to obtain at the end of the procedure the middle of the femur in the middle of the tibia both in flexion and in extension.

Trials are crucial and range of motion and stability must be evaluated with floating implants trials. A 2 mm laxity in the frontal plane is mandatory.

The knee should be at 45° of flexion during the curing of the cement to avoid any posterior liftoff that can be observed with the knee hyperextended [2–4].

Conclusions

Indication, implant selection, and surgical technique are the three steps to obtain good and long-lasting objective and subjective results in fixed-bearing UKA.

14.4 Tips and Tricks in UKA

John Bellemans

Unlike TKA, UKA is an anatomic and therefore more physiologic procedure. During UKA the cruciate ligaments, collateral soft tissues, patellofemoral mechanism, and joint line orientation are basically preserved or restored to its pre-diseased status.

This requires a fundamentally different technical approach by the surgeon, when compared to performing a TKA.

In UKA the primary objective is therefore to restore the joint to its anatomic, physiologic, and kinematic conditions as was the case before the degeneration or damage occurred.

Three key factors are thereby fundamental: (1) the restoration of joint line position, (2) the restoration of natural soft tissue interaction, and (3) the restoration of the patient's constitutional leg alignment. These three factors are at the same time interrelated as well as synergistic towards the final outcome.

Restoration of the joint line starts with the tibial cut. It should be low enough to compensate for the loss of cartilage with at least the thinnest polyethylene thickness available, but at the same time high enough to provide sufficient bone strength for supporting the component (tibial bone strength decreases distally). Together with proximodistal restoration, correct orientation of sagittal tibial slope is important, since it determines correct restoration of the sagittal anatomy, stability, as well as flexion kinematics.

Restoration of the femoral joint line is technically simpler than on the tibial side and aims at replacing the summation of resected and eroded bone plus cartilage.

Once both of these are achieved, the interprosthetic gap needs to be filled with the appropriate insert thickness to restore natural soft tissue tightness (or laxity), which in its turn automatically restores natural (constitutional) alignment of the patient, provided that no iatrogenic soft tissue damage has occurred.

All of the above can be obtained through different methodologies or strategies. Whether one uses a tibia-first or femur-first technique, dependent or independent femorotibial cuts, conventional or patient-specific guides, or even navigated or robotic cuts, these three general principles stay the same.

Some generic tips may further help obtaining the optimal result. First, an adequate judgment of preoperative tibial bone loss, femoral bone loss, alignment, and soft tissue status should be obtained. These will teach you how the joint is just prior to the procedure and will also give you an estimate on how it was in its pre-diseased state. The latter will then serve as your goal during UKA surgery.

Second, adequate visualization of the different steps during surgery is mandatory. An incision of sufficient length and an arthrotomy approach and patient positioning that allows adequate visualization of the operative field and the different surgical steps are extremely important. The surgeon should therefore explore which of the several methodologies he/she can familiarize in order to achieve this. Additionally, adequate intraoperative assessment tools are key to judge joint line level, ligament status, and alignment objectives. Certainly for the beginner UKA surgeon, these are critical. When experience however grows, they become less critical since each of the above reported key factors (joint line/soft tissue status/alignment) is interrelated and errors in one of them will influence the others, which will become rapidly apparent even during the surgery. It is one of the reasons why experience is of so much benefit during knee replacement surgery.

14.5 Tips on Assessment of Failed UKA

Nikolay Kornilov

UKA not always leads to expected outcome. Systemic approach to the patient helps to find the reasons of UKA failure and it is based on five steps: complaints, history, physical examination, imaging studies, and laboratory tests.

14.5.1 Complaints

Listen to the patient attentively and define the main problem: pain, instability, stiffness, swelling, or combination of symptoms. Knee pain after UKA has a broad spectrum of etiologies that can be divided into two categories: intra-articular and extra-articular. Pain characteristics such as localization, irradiation, intensity, nature, duration, and relation to activity should always be taken into consideration.

14.5.2 History

Time of symptoms onset and their change after primary surgery are very important. If they appeared immediately after surgery, remember the so-called four i rule:

- Wrong *indication* (extra-articular etiology, especially if the same nature)
- Early *infection*
- *Instability* due to inadequate soft tissue balancing
- *Impingement* of soft tissue or mobile bearing

If there was some period of asymptomatic function, it is necessary exclude late infection or aseptic loosening. Some of somatic comorbidities (vascular, neurologic, etc.) and psychiatric disorders, especially depression, may also have influence on knee function. General symptoms like fever, rigors, lethargy, or disturbed sleep often accompany surgical infection. Often, patients already used some medications so their efficiency should be evaluated.

14.5.3 Physical Examination

During examination pay attention to walking, limp, alignment, internal/external rotation of foot, joint swelling, erythema, and sinus with/without discharge. Palpate joint line, patella edges, and tendon and ligament insertions. Check joint stability (medial/lateral and sagittal laxity) and active/passive range of motion (flexion/extension contracture, hyperextension, extension lag). Investigate general patella mobility, tracking (lateralization, subluxation, dislocation), crepitation or clunks, and pain after compression or percussion. Briefly examine other joints: lumbar spine, hip, ankle, and foot deformity.

14.5.4 Imaging Studies

Radiography is the first imaging study that includes whole leg weight-bearing x-rays, standard AP and lateral view, and axial patella view and allows analysis of component size (overstuffing, medial/lateral overhang), component positioning (varus/valgus, flexion/extension) or migration, joint line and patella level, lucencies, osteolysis, component fracture, poly wear, and periprosthetic fractures. Signs of implant loosening are radiolucent line progression on implant/bone interface (>2 mm), zones of osteolysis, migration of components, cement mantle fractures, and reaction around the tip of stemmed component.

Fluoroscopy is useful in the diagnosis of early osteolytic changes. Besides that, knee stability could be evaluated under fluoroscopic control.

Computed tomography demonstrates rotation of femoral and tibial components, extent of osteolytic areas, and overall leg alignment but in non-weight-bearing conditions.

MRI after artifact suppression allows establishing soft tissues state (meniscus, cartilage, ligaments) in non-replaced knee compartments. Radionuclide bone scans with technetium, indium-labeled white blood cells and sulfur-colloid marrow may also be useful, especially when combined together.

14.5.5 Laboratory Tests

For infection exclusion synovial fluid aspiration for cytology and culturing as well as hematologic tests (leucocytes, erythrocyte sedimentation rate, C-reactive protein, interleukin-6) must be routinely performed.

For differentiation of pain origins, intra-articular injection of local anesthetic or combination anesthetic with corticosteroids may be also performed. Arthroscopy is the last but not least diagnostic tool that can also be helpful for the treatment of meniscus or cartilage damage and loose body removal [5].

14.5.6 Take Home Message

- Systemic approach.
- Remember extra-articular sources of pain.
- Always exclude infection first.
- Revision without definitive diagnosis leads to poor results: >60 % of patients

14.6 Tips on Revision UKA

Pawel Skowronek and Julian Dutka

14.6.1 Introduction

The key issues concerning UKA conversion to TKA consist of the proper implantation including implant sizing, rotation, level of cuts, and joint line restoration. Exposure, implant removal, and bone loss are usually minor problems. It is important to identify the reason for UKA failure and also have good knowledge of the principles of knee arthroplasty. The idea of UKA revision is to convert revision surgery into primary total knee arthroplasty. UKA should not be revised to UKA.

14.6.2 Seven Steps for Successful Revision

1. *Planning:* Find the reason for failure – consider previous surgery, previous scars, infection, instability, stiffness, fractures, and bone defects.
2. *Implant selection:* The idea of UKA revision is to convert revision surgery into primary total knee arthroplasty. The aim of the revision is to use a less constrained implant to achieve good knee stability. In most cases primary knee implants can be used.
3. *Exposure:* Appropriate exposure is needed for UKA revision. Use an old incision, lengthen it, and avoid additional skin incisions to prevent problems with the skin healing.
4. *Implant removal:* Techniques for component removal are the same as in revision knee arthroplasty. Be patient; preserve as much bone as possible. In the first step of the operation, it is recommended to leave components in place if they are not loose. It is helpful to determine the size and position of the implant. Thorough debridement is also needed to remove all unhealthy tissue and cement.
5. *Bone loss:* Types of bone stock restoration include augments and bone grafts. It is important to achieve a good initial stability of the implant.
6. *Proper implant position:* Follow the principles of total knee arthroplasty (sizing, rotation, ligament balancing, joint line). It is necessary to use bone landmarks for proper implant position.

   Femoral cuts should be assessed with the implant left in place. After the distal cut, the implant can be removed. The use of Whiteside's and epicondylar lines as a reference and the augmentation of the posterior condyle resection are needed for the proper rotational axis of the femoral implant.

   A tibial bone resection should be based on the lateral condyle level using an extramedullary cutting device as with primary TKA. The next step is assessing bone loss in the medial compartment and preparing for augmentation if needed.
7. *Balancing the knee:* Flexion/extension gap assessment.

   Results: The revision of failed UKA to TKA is technically easier than revision of failed TKA. Most series show survivorship similar to that of primary TKA but the patient-reported outcome measures are not as good as the primary TKA [6–17].

14.6.3 Take Home Message

Identify the reason for UKA failure.

Convert revision surgery into primary total knee arthroplasty.

Use the least constrained implant to achieve good knee stability.

Do not revise UKA to UKA.

References

1. Liddle AD, et al. Optimal usage of unicompartmental knee replacement: an analysis of 41,986 cases from the National Joint Registry for England and Wales. International Congress of Arthroplasty Registries, 2013.

2. Argenson JN, Parratte S, Flecher X, Aubaniac JM. Unicompartmental knee arthroplasty: technique through a mini-incision. Clin Orthop Relat Res. 2007;464:32–6.
3. Parratte S, Pauly V, Aubaniac JM, Argenson JN. No long-term difference between fixed and mobile medial unicompartmental arthroplasty. Clin Orthop Relat Res. 2012;470:61–8.
4. Parratte S, Argenson JN, Pearce O, Pauly V, Auquier P, Aubaniac JM. Medial unicompartmental knee replacement in the under-50s. J Bone Joint Surg Br. 2009;91:351–6.
5. Goodfellow JW, et al. Unicompartmental arthroplasty with the Oxford knee. Oxford: Goodfellow Publishers; 2011. 194 p.
6. Berend KR, George J, Lombardi Jr AV. Unicompartmental knee arthroplasty to total knee arthroplasty conversion: assuring a primary outcome. Orthopedics. 2009;32(9).
7. McAuley JP, Engh GA, Ammeen DJ. Revision of failed unicompartmental knee arthroplasty. Clin Orthop Relat Res. 2001;392:279–82.
8. Levine WN, Ozuna R, Scott RD, Thornhill TS. Conversion of failed modern unicompartmental arthroplasty to total knee arthroplasty. J Arthroplasty. 1996;11(7):797–801.
9. Springer BD, Scott RD, Thornhill TS. Conversion of failed unicompartmental knee arthroplasty to TKA. Clin Orthop Relat Res. 2006;446:214–20.
10. Saragaglia D, Estour G, Nemer C, Colle PE. Revision of 33 unicompartmental knee prostheses using total knee arthroplasty: strategy and results. Int Orthop. 2009;33(4):969–74. Epub 2008 Jun 18.
11. Saldanha KA, Keys GW, Svard UC, White SH, Rao C. Revision of Oxford medial unicompartmental knee arthroplasty to total knee arthroplasty-results of a multicentre study. Knee. 2007;14(4):275–9.
12. Parvizi J. Unicompartmental knee replacement did not differ from total knee replacement with regard to clinical outcomes at 15 years. J Bone Joint Surg Am. 2009;91(8):2012.
13. Baker PN, Petheram T, Avery PJ, Gregg PJ, Deehan DJ. Revision for unexplained pain following unicompartmental and total knee replacement. J Bone Joint Surg Am. 2012;94(17):e126.
14. Bohm I, Landsiedl F. Revision surgery after failed unicompartmental knee arthroplasty: a study of 35 cases. J Arthroplasty. 2000;15(8):982–9.
15. Johnson S, Jones P, Newman JH. The survivorship and results of total knee replacements converted from unicompartmental knee replacements. Knee. 2007;14(2):154–7.
16. Sierra RJ, Kassel CA, Wetters NG, Berend KR, Della Valle CJ, Lombardi AV. Revision of unicompartmental arthroplasty to total knee arthroplasty: not always a slam dunk! J Arthroplasty. 2013;28(8 Suppl):128–32. pii: S0883–5403(13)00474–9.
17. Hang JR, Stanford TE, Graves SE, Davidson DC, de Steiger RN, Miller LN. Outcome of revision of unicompartmental knee replacement. Acta Orthop. 2010;81(1):95–8.

Part III

Instructional Courses: Education

How to Do Proper Research

15

Kristian Samuelsson, Camilla Halewood, Andrew A. Amis, Sebastian Kopf, Eduard Alentorn-Geli, and Volker Musahl

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15.1 How to Ask, Plan and Apply

Proper research follows the "cycle of surgical research" described by Robert Salter (Fig. 15.1) [1]. The first step in a research project is recognizing an unsolved clinical problem. This requires researchers to work closely together with clinicians or have expertise in both fields. Nowadays, most universities offer MD/PhD programs to train so-called clinician scientists. This provides medical students from an early point in their career both the clinical and research methodological tools required to be successful in surgical research.

Before a review of the scientific literature is undertaken, one should think about possible problems and their solutions. For example, if desired to study unstable knees following injury and occurrence of hemarthrosis, the thought should be about patient-related factors and objective outcome tools that will be helpful in analyzing the natural history of knee instability.

The art of asking intelligent questions is best learned in the conference room. Preliminary thoughts and speculations should be presented in a lab meeting. Questions will occur both ways, from mentor to student and equally importantly from student to mentor. Questions will also occur from peer to peer, whereby a diverse background of the audience, with respect to educational level and expertise, is helpful and will enhance feedback for the presenter.

A scientific hypothesis needs to be carefully formulated to reflect both a question of scientific

S. Zaffagnini et al. (eds.), *ESSKA Instructional Course Lecture Book*,
DOI 10.1007/978-3-642-53983-1_15, © ESSKA 2014

merit and a question that is statistically testable. It is important that a hypothesis be formulated before the research protocol is being planned. The alternative hypothesis is used, which states that there will be a difference or relation between two expected outcomes. For example, patella tendon anterior cruciate ligament (ACL) ACL reconstruction will lead to fewer graft ruptures compared to hamstring ACL reconstruction. Statistical tests are used to determine how likely it is that the overall effect would be observed if no real relation as hypothesized exists. If the likelihood is sufficiently small, i.e., less than 5 % ($p<0.05$), it may be assumed that the relation exists and the alternative hypothesis can be accepted.

The research protocol needs to be planned in as much detail as possible. This will start with the location and personnel required to perform the test. The strength of the research team will be their experience and diversity. One should utilize methods that have been well established and can be compared to. A power analysis should be performed as well. Data analysis and employing of statistical tests is best done by independent observers to avoid observer bias.

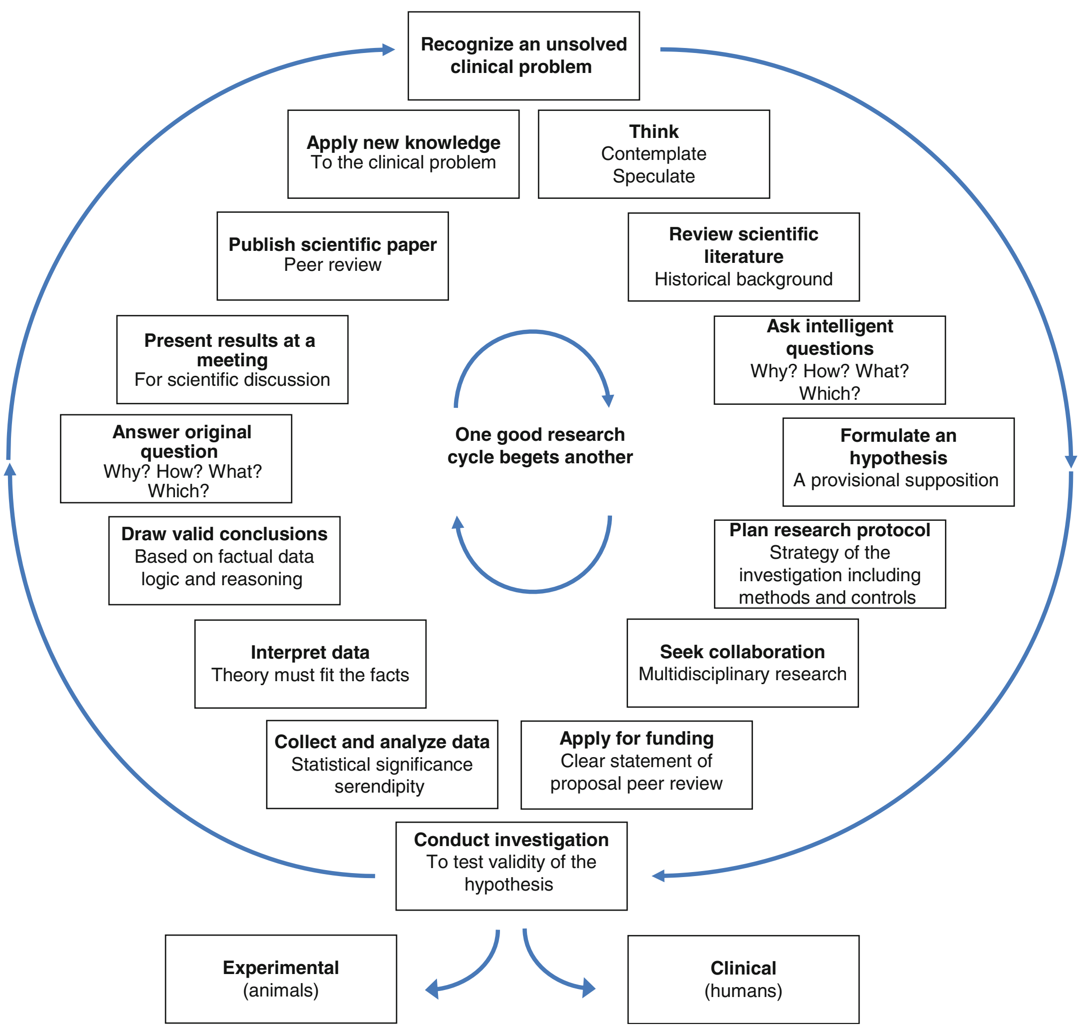

Fig. 15.1 Dr. Robert Salter's Cycle of medical research. To find the solution to an unsolved clinical problem

The application for funding of a research study should start well in advance of conducting investigations. The most important piece of the application is a well-thought thorough research plan including a paragraph on the pertinent knowledge of the subject to be tested, e.g., ACL reconstruction. There should be one paragraph outlining how this research study is going to impact the field, followed by a paragraph that explains the expertise of this research team and unique tools available to accomplish the planned study. The lack of available data on an unsolved clinical problem should then lead to a scientific objective and hypothesis. The final part of the one-page research plan is a summary statement explaining clinical significance of this research. The following pages (usually 10–20) include all the necessary details, such as background and significance, preliminary data, methods, ethics approval, literature list, biographical sketches, and a budget with justification.

In summary, a good researcher has to be a meticulous planner and has open collaboration that should be both intra- and extramural. A diverse research team will enrich the knowledge to be gained from answers addressing the original question of an unsolved clinical problem. Plan for every possible outcome, and you will not be surprised!

15.2 Evidence-Based Medicine

Modern medicine, both in the clinical setting and in the field of research, is characterized by a need for well-founded information on diagnosis, treatment, prevention, and prognosis for numerous patients with countless conditions and diseases. This has formed the basis of a shift in the general conception of the nature of scientific endeavor.

Sackett et al. proposed a system in 1986 for grading different levels of medical evidence and introduced the concept of evidence-based medicine (EBM) [2]. It was described as "the conscientious, explicit, and judicious use of the current best evidence in making decisions about the care of individual patients." Historically, the philosophical origin of EBM extends back to mid-nineteenth-century Paris and earlier [3].

As a concept and practice, EBM has woven its way into the fabric of most if not all fields of medicine today and orthopedic surgery is no exception. Two fundamental principles exist that form the backbone of presumed sound evidence: internal and external validity. Internal validity describes the contingent relationship between two variables; in the case of medicine, these variables are intervention/exposure and the resultant outcome. The presence of internal validity is in turn quantified by three main factors: the power of a study, subject allocation, and blinding. External validity refers to the consistency or replicability of results within a given population or setting.

There are multiple versions of the hierarchy for level of evidence and none that is unanimously used. However, there is a consensus on the strength from different study types relative to each other. One of the most common grading system can be found at the Oxford Centre for Evidence-Based Medicine website, www.cebm.net (Table 15.1). The system categorizes a study from one to five on the basis of its design and as one of four different types on the basis of its content. Based on this, the paradigm of EBM has proposed a hierarchy of study designs in ascending order of bias control whereby the presence of three vital features, each in itself contributing to such bias control, raises the strength of the studies within the hierarchy and thereby its level of evidence. These features are randomization, prospective follow-up, and, finally, replication of evidence. The scale is built according to a hierarchy in which Level I is the highest level of evidence, which includes high-quality randomized clinical trials, and Level V, which is the lowest level of evidence, includes so-called expert opinions (Fig. 15.2). The higher the level of evidence, the more reproducibility and applicability to the general patient there is. Levels of evidence are important not only in determining whether one study is of higher quality than another, they also give the reader an immediate sense of how much weight the results of the study should be given. Thus, it is more likely to find a final answer to a

Table 15.1 Levels of evidence for primary research question. A complete assessment of quality of individual studies requires critical appraisal of all aspects of the study design

	Types of studies			
	Therapeutic studies	Prognostic studies	Diagnostic studies	Economic and decision analyses
	Investigating the results of treatment	Investigating the effect of a patient characteristic on the outcome of disease	Investigating a diagnostic test	Developing an economic or decision model
Level I	High-quality randomized clinical trial (RCT) with statistically significant difference or no statistically significant difference but narrow confidence intervals	High-quality prospective study[c] (all patients were enrolled at the same point in their disease with ≥80 % follow-up of enrolled patients)	Testing of previously developed diagnostic criteria on consecutive patients (with universally applied reference "gold" standard)	Sensible costs and alternatives; values obtained from many studies; with multiway sensitivity analyses
	Systematic review[a] of Level I RCTs (and study results were homogenous[b])	Systematic review[a] of Level I studies	Systematic review[a] of Level I studies	Systematic review[a] of Level I studies
Level II	Lesser quality RCT (e.g., <80 % follow-up, no blinding, or improper randomization)	Retrospective[f] study	Development of diagnostic criteria on consecutive patients (with universally applied reference "gold" standard)	Sensible costs and alternatives; values obtained from limited studies; with multiway sensitivity analyses
	Prospective[c] comparative study[d]	Untreated controls from an RCT	Systematic review[a] of Level II studies	Systematic review[a] of Level II studies
	Systematic review[a] of Level II studies or Level I studies with inconsistent results	Lesser quality prospective study (e.g., patients enrolled at different points in their disease or <80 % follow-up)		
		Systematic review[a] of Level II studies		
Level III	Case–control study[e]	Case–control study[e]	Study of nonconsecutive patients; without consistently applied reference "gold" standard	Analyses based on limited alternatives and costs; and poor estimates
	Retrospective[f] comparative study[d]		Systematic review[a] of Level III studies	Systematic review[a] of Level III studies
	Systematic review[a] of Level III studies			
Level IV	Case series[g]	Case series	Case–control study	Analyses with no sensitivity analyses
			Poor reference standard	
Level V	Expert opinion	Expert opinion	Expert opinion	Expert opinion

[a] A combination of results from two or more prior studies
[b] Studies provided consistent results
[c] Study was started before the first patient enrolled
[d] Patients treated one way (e.g., cemented hip arthroplasty) compared with a group of patients treated in another way (e.g., uncemented hip arthroplasty) at the same institution
[e] Patients identified for the study based on their outcome, called "cases" (e.g., failed total arthroplasty), are compared to those who did not have that outcome, called "controls" (e.g., successful total hip arthroplasty)
[f] The study was started after the first patient enrolled
[g] Patients treated one way with no comparison group of patients treated in another way

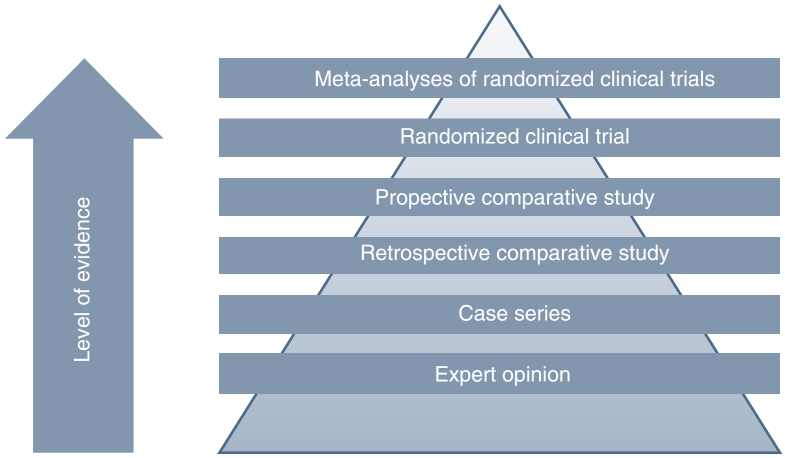

Fig. 15.2 The hierarchy of evidence

research question the higher you move up the hierarchy. The grading system is widely accepted and utilized by most orthopedic journals as it ensures that the best available evidence is used in patient care. It has become the foundation of evidence-based medicine.

There is no doubt that evidence-based medicine has forever changed modern medicine both in the clinical setting and in the field of research. In an international survey from the British Medical Journal to the global medical community, the EBM concept was voted as one of the top 15 medical breakthroughs in the last 160 years [4]. This places a perspective on the importance and the geniality of EBM.

15.3 How to Do a Proper Biomechanical Study

A well-conducted biomechanical study can deliver valuable information about orthopedic surgical techniques, implant performance, and treatment options that in vivo or in silico work cannot provide alone.

There are three broad types of biomechanical studies that can be carried out in the laboratory using cadaver specimens: (1) testing of the intrinsic mechanical properties of a material; (2) assessment of implant, graft, or fracture fixation; and (3) examination of joint biomechanics. Within each of these categories is a raft of existing tests that could be conducted as well as the scope for planning bespoke work.

Proper planning of a biomechanical study is critical, made even more so when using donated cadaver specimens. Ethical approval must be sought to use cadavers and legislation regarding the storage and management of cadaveric tissue varies with country and is tightly regulated; therefore, individuals should check this with local authorities before planning a study.

There are a number of key questions that must be considered before embarking on a biomechanical study, to ensure that it is well planned:

- What is the research hypothesis?
- What are the independent and dependent variables?
- What kind of controls will be used?
- How many specimens are required for the study to be adequately powered?
- What kind of statistical analysis will be made?
- Can it be a repeated measures design?
- Is there an existing test method?
- Is materials testing machine required?
- Is a testing rig or fixture needed?
- Is another hardware or software required?
- How long will each test take to complete?
- What are the limitations?

Preliminary testing, using plastic bone or animal cadaver substitutes, is recommended in order to finalize the testing procedure and ensure that every donated human specimen is fully utilized. In some cases, animal specimens can be used for the actual testing, but care must be taken to ensure the validity of such a model. In particular, it should be considered that animals are put down at a young age, meaning that their soft tissue material properties may differ significantly from human tissue.

A key consideration of most biomechanical studies is loading. Joint forces result mainly from muscles; the directions and magnitudes of these tensions must be derived from the literature prior to finalizing the test method. In order not to damage cadaver specimens, the loading magnitudes may well have to be scaled down from the physiological.

Specimen management during testing is critical. The use of fresh-frozen cadavers is preferable to embalmed ones, as formalin-fixed specimens are stiffer and the mechanical properties are altered, making them unrepresentative of in vivo conditions. Specimens must be kept moist throughout testing; particular care should be taken of water-rich structures such as cartilage and menisci, which can permanently dehydrate rapidly.

Biomechanical studies can be powerful tools when in vivo measurements are not possible. However, care must be taken to properly plan the study, to make the experiments as realistic as possible, and to be aware of the limitations of working with cadaver specimens (Fig. 15.3).

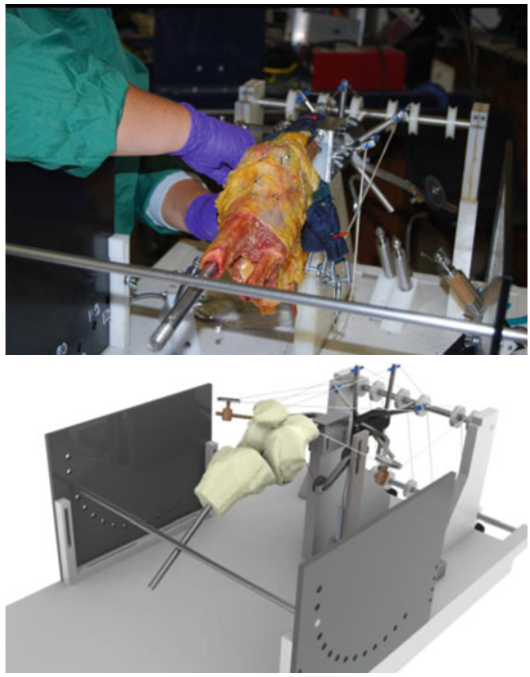

Fig. 15.3 *Top*: a cadaver knee being prepared for testing in a knee extension rig, with the heads of the quadriceps divided for loading and LVDTs for ligament length change measurements. *Below*: a model of the experimental setup. This kind of rig can also be used in conjunction with optical tracking equipment for 6 Degrees of freedom (DoF) kinematic measurements and pressure-sensitive film for contact pressure and area measurements

15.4 How to Conduct a Clinical Trial

Conducting a clinical trial starts with an interesting, worthwhile research question that has not been answered or that needs to be double-checked as well as improves patient outcome. The hypothesis should be defined. One of the utmost important steps is a meticulously performed literature search including old papers and occasionally books before the time of PubMed. To further design the study, ideally a statistician should be involved in deciding about the important details of the study design, e.g., controlled, prospective vs. retrospective, case–control or case series, and correct randomization. The aim is to choose the best level of evidence for the study design to assure a high impact of its findings. A crucial step is the calculation of a sample size. Appropriate outcome tools that show a holistic picture of the evaluated study objective, e.g., evaluating a new surgical method in ACL reconstruction, include not only knee-specific questionnaires but also functional tests and general health questionnaires and evaluate the rate of osteoarthritis onset. Before starting the clinical trial, the ethical committee must approve the study. Patient information and informed consents are important for prospective studies. Furthermore, financing of the entire study should be secured. A pilot study might be introduced to gain data for calculating the sample size and expose possible sources of error. The study should be conducted based on the Good Clinical Practice Guideline (http://www.ich.org/fileadmin/Public_Web_Site/ICH_Products/Guidelines/Efficacy/E6_R1/Step4/E6_R1__Guideline.pdf). Data collection should be anonymized or pseudonymized. The statistical analysis should be done with appropriate statistical methods, ideally by a statistician, e.g., checking the data for their distribution, using appropriate tests, and presenting the data accordingly in the paper (e.g., normal distribution with mean or 95 % confidence interval vs. median with range or interquartile range). The hypothesis should be confirmed or declined and investigators should be aware of the fact that statistical differences do not have to be clinically significant. In order to have a meaningful message of the paper, a high rate (above 90 %) is recommended. The authorship should be clarified, and the first author should write the manuscript and is generally responsible for submitting. Declaring the authorship before starting the trial helps to avoid confusion later.

15.5 How to Prepare a Systematic Review

The available information in medical literature is huge and unmanageable in most of their fields. Therefore, health-care providers, patients, researchers, and any other potential reader may not have the time, skills, and resources to find, appraise, interpret, and incorporate this information for decision-making [5]. Review articles are especially useful for clinicians and researchers to keep up-to-date in their fields (continuing medical education), develop practice guidelines, and develop ideas for future research [6]. These articles may be nonsystematic (narrative) or systematic reviews. Systematic reviews are reviews "of a clearly formulated question that uses systematic and explicit methods to identify, select, and critically appraise relevant research, and to collect and analyze data from the studies that are included in the review" [6]. The use of statistical methods (meta-analysis) is common in systematic reviews to analyze and summarize the results of the included studies. Meta-analyses provide more precise estimates of the effects of health care and facilitate investigation of consistency of evidence and exploration of differences across studies [5]. The use of statistics to report the results of included studies is not mandatory in systematic reviews. Systematic reviews provide more reliable conclusions than "narrative" reviews due to its inherent characteristics [5]:

- Clearly stated set of objectives with predefined eligibility criteria for studies
- Explicit and reproducible methodology
- Systematic literature search that attempts to identify all studies that would meet the eligibility criteria
- Assessment of the validity of the findings of the included studies
- Systematic presentation and synthesis of the characteristics and findings of the included studies

In 2005, a meeting of experts was held in Canada to revise and expand the Quality of Reporting of Meta-analyses (QUOROM) checklist and flow diagram [6]. The results of this meeting were renamed Preferred Reporting Items for Systematic Reviews (PRISMA) statement [6]. This statement provides an essential checklist with recommended information in each of the following items to prepare systematic reviews and meta-analyses [6]:

- Title
- Abstract
- Introduction: rationale and objectives
- Methods: protocol and registration, eligibility criteria, information sources, search, study selection, data collection process, data items, risk of bias in individual studies, summary measures, synthesis of results, risk of bias across studies, and additional analyses
- Results: study selection, study characteristics, risk of bias within studies, results of individual studies, synthesis of results, risk of bias across studies, and additional analysis
- Discussion: summary of evidence, limitations, and conclusions
- Funding

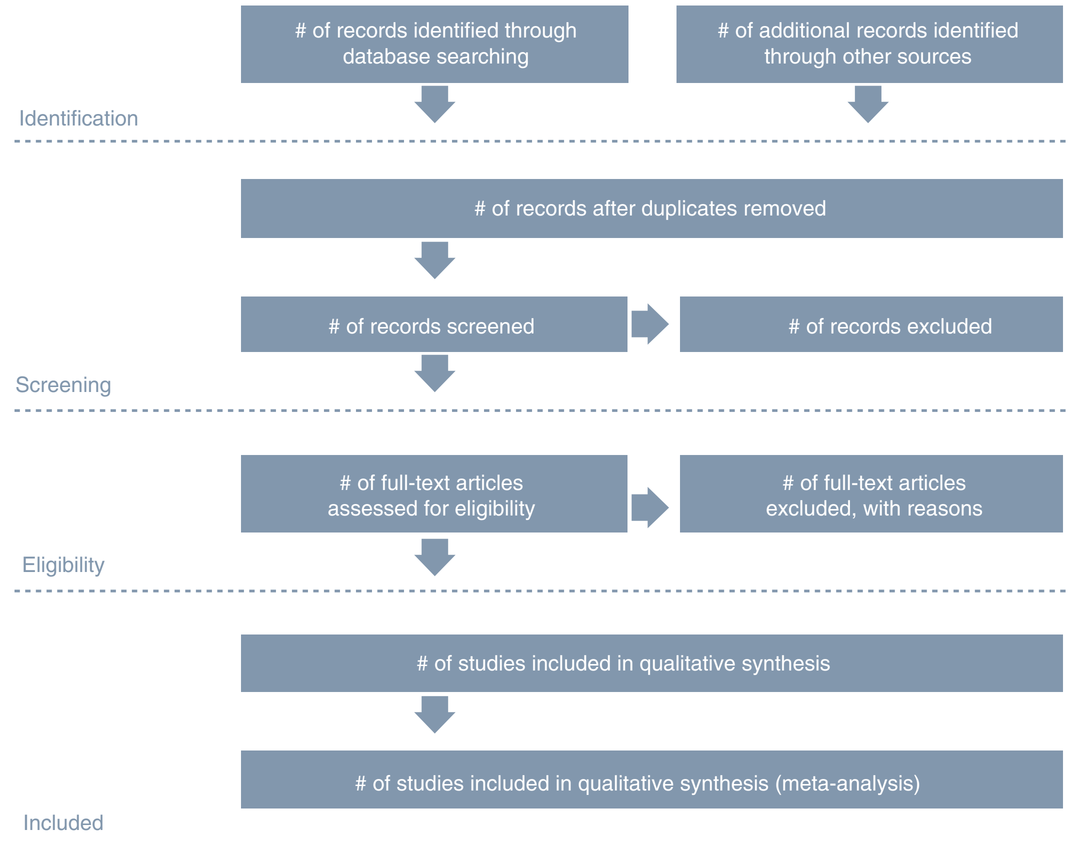

Fig. 15.4 Flow of information through the different phases of a systematic review

A complete explanation with useful examples for each of these items can be found in another publication by the same group [7]. A flow of information through the different phases (identification, screening, eligibility, and inclusion of studies) of a systematic review should be presented as a figure in the article [6] (Fig. 15.4). Following these guidelines and recommendations will homogenize the existing literature and may increase the quality of systematic reviews. This may in turn result in an improvement in medical decision-making, with a consequent improvement in the patient's care.

References

1. Salter RB. Textbook of disorders and injuries of the musculoskeletal system. 3rd ed. Baltimore: Lippincott Williams & Wilkins; 1999.
2. Sackett DL. Rules of evidence and clinical recommendations on the use of antithrombotic agents. Chest. 1986;89(2 Suppl):2S–3.
3. Sackett DL, Rosenberg WM, Gray JA, Haynes RB, Richardson WS. Evidence based medicine: what it is and what it isn't. BMJ. 1996;312(7023):71–2.
4. Committee IS, Audige L, Ayeni OR, Bhandari M, Boyle BW, Briggs KK, et al. A practical guide to research: design, execution, and publication. Arthroscopy. 2011;27(4 Suppl):S1–112.
5. Green S, Higgins JPT, Alderson P, Clarke M, Mulrow CD, Oxman AD. Cochrane reviews. Introduction: systematic reviews. In: Higgins JPT, Green S, editors. Cochrane handbook for systematic reviews of interventions. Chichester: Wiley-Blackwell; 2008. p. 3–9.
6. Moher D, Liberati A, Tetzlaff J, Altman DG, Group P. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. Ann Intern Med. 2009;18:264–9.
7. Liberati A, Altman DG, Tetzlaff J, Mulrow CD, Gotzsche PC, Ioannidis JPA, et al. The PRISMA statement for reporting systematic reviews and meta-analyses of studies that evaluate health care interventions: explanations and elaboration. Ann Intern Med. 2009;151:W65–94.

Arthroscopic Simulation in Skills Training: European Initiatives

16

Mustafa Karahan and Gabrielle Tuijthof

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16.1 Introduction

Arthroscopic surgery has become the leading operative therapy for a growing number of injuries [1, 2]. Obtaining arthroscopic skills is quite challenging, due to reduced visibility and dexterity of instruments, disturbed eye-hand coordination and loss of force and tactile feedback [3–5]. That is why learning arthroscopic skills takes considerable time and implicates an increased risk of surgical errors during the early stages of the learning curve when operating on patients [6–8]. As patient safety and competence in surgery are of utmost importance to fulfil requirements of high-quality healthcare [9], there is a need for efficient and effective training.

It is known that training of psychomotor skills, such as arthroscopic skills, can be optimally performed by actual instrument handling [10, 11]. This is based on the theory which states that skilled motor behaviour relies on accurate predictive models of our own body and the environment we interact with (e.g. instruments) [10, 11] (Fig. 16.1). These models are stored in our central nervous system. For execution of a certain task, the best available internal model is selected. A key feature in this theory is that these internal models are tuned, updated and learned by providing feedback from our sensory organs (vision and proprioception) (Fig. 16.1).

This is why arthroscopic training traditionally is performed in the operating room in a one-to-one apprentice model [6, 9, 12]. The arthroscopic skills are learned by observing skilled surgeons

S. Zaffagnini et al. (eds.), *ESSKA Instructional Course Lecture Book*,
DOI 10.1007/978-3-642-53983-1_16, © ESSKA 2014

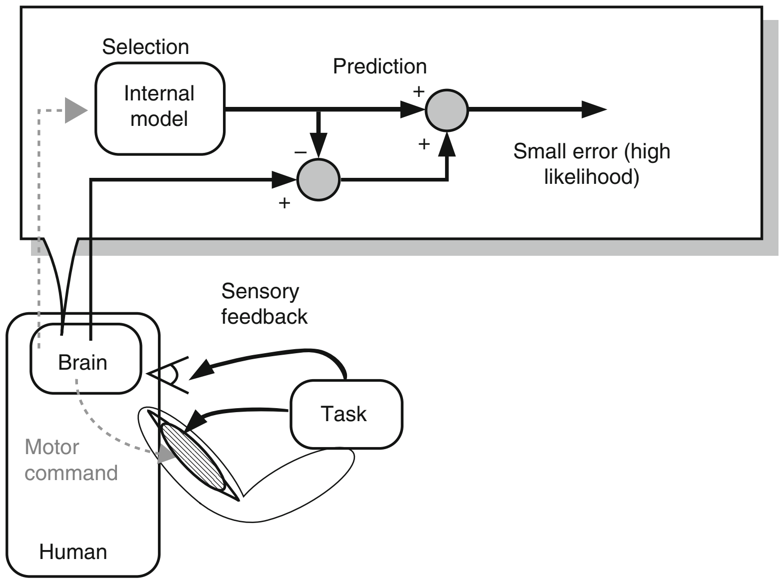

Fig. 16.1 Illustration of the theory on motor learning. The internal model is chosen that is most likely to predict the smallest estimation error. An internal model predicts both the spatial state of your body and the sensory signals. The latter combined with the actual sensory signals are used to correct the initial estimate

in the operating room initially, which gradually expands to instrument handling whilst the resident's proficiency improves [6]. However, at the start of residents' training process, the operating room is no optimal learning environment. The resident is not allowed to make errors and learn from them, because those errors would compromise patient safety [12–14].

Today, simulation training for surgical procedures is increasingly seen as an essential component of the curriculum. For example, in the UK, compulsory simulation training is for the first time being introduced into the curriculum by the regulator, the General Medical Council. It is almost certain that this trend will continue and expand also towards arthroscopic skills. This chapter presents the state of the art of training modalities based on an educational framework for well-designed training programmes.

16.2 State of the Art in Training Arthroscopy

From an educational point of view, a key feature for a well-designed training programme is that the three training elements are formulated explicitly and are aligned [15]: learning objectives (what should be trained?), means (how should it be trained?) and assessment (is the objective achieved?).

16.2.1 What Should Be Trained?

The first step is addressing the learning objectives. The opinion of faculty was documented on how many repetitions an average resident needs in the operating room to become proficient in arthroscopic procedures [8]. The results were as follows: on average, 50 (standard deviation (SD) 46) repetitions for partial medial meniscectomy, 61 (SD 53) for anterior cruciate ligament (ACL) reconstruction, 48 (SD 44) for diagnostic shoulder scope, and 58 (SD 56) for subacromial decompression [8]. An interesting result was that the absolute minimum number of repetitions needed to achieve proficiency was indicated to be 5–8 for any arthroscopic procedure [8]. Thus, at least up to eight patients are at risk at the start of each resident training programme. An ideal situation is that before residents continue their training in the operating room, they should have achieved a competency level that guarantees safe arthroscopic treatment on their first patient. Logically, this should be one of the primary learning objectives for training arthroscopic skills in a simulated environment.

Next, this question needs to be answered: 'What skills are crucial for a resident to possess before continuing safe training in the operating room?' [16]. Safir and co-workers [16] consulted a group of experienced orthopaedic surgeons from the Canadian Orthopaedic Association.

Table 16.1 Ranking of importance for trainee to possess ability prior to performing in the operating room

Rank	Surgeons [16] $n = 101$	Score (1–5)	Residents [17] $n = 67$	Score (1–5)	Surgeons-residents NVA $n = 20$	Score (1–5)
1	Anatomic knowledge	3.86*	Anatomic knowledge	4.4	Anatomic knowledge	4.70
2	Triangulation/depth perception	3.34*	Spatial perception	4.3	Spatial perception	4.15
3	Spatial perception	2.77*	Triangulation/depth perception	4.2	Tactile sensation	4.15
4	Manual dexterity	2.86*	Manual dexterity	4.2	Manual dexterity	4.00
5	Tactile sensation	2.05*	Tactile sensation	3.7	Triangulation	3.75

*Significantly different ($p < 0.001$) [16]

Supported by the Dutch Arthroscopy Society (NVA), a similar questionnaire was conducted in The Netherlands amongst the experienced arthroscopists and residents to determine the presence of cultural differences. To this end, an online survey was set up consisting of questions regarding (a) five general skills (Table 16.1) and (b) detailed skills such as placement of the tourniquet, creation of a portal, removal of loose fragments, specific anatomic knowledge and navigation. Surgeons were asked to indicate the importance of each arthroscopic task on a five-point Likert scale ranging from 'not important at all' (score 1) to 'very important' (score 5).

The preliminary results of the Dutch survey are presented together with the results from Safir and co-workers [16] and Hui and co-workers [17] (Table 16.1). These results cannot give a conclusive interpretation on cultural differences.

In all three surveys, knowledge on anatomy of the knee joint is ranked as priority number one (Table 16.1). As the term 'knowledge' already indicates, this skill does not require actual instrument handling during training. As performing arthroscopy is largely dependent on visual cues received from the monitor, arthroscopic anatomy is suited to be taught outside the operating room, for example, using interactive e-learning modules that incorporate arthroscopic movies, pictures and animated joint structures or using virtual reality simulators which also provide movies and sometimes specific exercises focused on anatomy in combination with spatial perception [14, 18]. One other solution being explored is to use online simulators, where the programme is held on a central server and where the simulator addresses those aspects of a surgical task that do not require a complex end-user controller that is expensive and fixed in one geographical location [19]. The other skills do require instrument handling [20]. In general, the top five of specific skills to be trained reflects the basic steps required to gain access and navigate into the joint. This seems straightforward as knowing your way in the joint will contribute to safe performance of the therapy.

16.2.2 How Can Arthroscopic Skills Be Assessed?

The second step is to set up an assessment method that is capable to discriminate the proposed competency level. Some evidence now highlights that not all individuals are able to reach competence in some basic arthroscopic skills despite training and practice [21]. Therefore, it is sensible and necessary we ensure that objective, validated and reliable assessment methods are employed during arthroscopic skills training. So far, the development of a skills assessment method that can be used for examination or credentialing has not been achieved [22]. Psychomotor skills are complex and it is difficult to translate the execution of skills into a set of measurable parameters that also make sense when providing feedback. On the other hand, the available methods of skills assessment can be used to monitor trainee's performance and provide feedback. Trainee's performance consists roughly of three categories: psychomotor skills inherently present in each person, ability of effective and efficient task completion and ability to guarantee patient safety [11, 23, 24]. Each will be discussed.

First, the training of basis motor skills in relation with arthroscopy will be illustrated by presenting a 2-day arthroscopy course consisting of six steps [25]. The first step includes interactive presentations about arthroscopic technology and basic knee pathologies. The second step shows video presentations of basic arthroscopic procedures. The third step consists of basic motor skill exercises. The search to meet today's educational needs has required the combination of motor skill training, kinesiology and the science of learning [26]. Basic motor skill acquisition can be applied to teach surgical skills [27, 28]. Some of the basic motor skills that constitute arthroscopic skills is listed in Table 16.2 and some instruments that simulate or evoke these skills are shown in Fig. 16.2 [24]. In a previous study, instruments to practise and perform basic motor skills were used to compare the differences between experienced and inexperienced orthopaedic surgeons [24]. The conclusion was that experienced surgeons react faster and more judiciously compared to novices, which provides evidence that a correlation is present between competency in basic psychomotor skills and competency in arthroscopic skills. The fourth step consists of triangulation on dry knee joint models. The fifth step is the wet lab that is mainly designed to mimic a real arthroscopic procedure with the use of a cow knee (Fig. 16.3). In this wet lab, basic arthroscopic procedures such as diagnosis, synovectomy, loose body extraction, meniscectomy, microfracture, etc. can be completed by the guidance of an instructor (Fig. 16.3). The final step is the knot station, in which all participants can train surgical knot tying. This basic arthroscopy course focused on (basic) motor skill training and arthroscopic procedures on cow knees is an effective, reproducible, safe and inexpensive method that improves the performance of trainees.

Table 16.2 Basic motor skills presented in the task analysis of arthroscopy

Skills	Task analysis of arthroscopy
Triangulation	Procedures performed by two hands
Depth perception	Ability to differentiate the position of objects in two-dimensional images
Response orientation	Ability to rapidly select a response from a number of alternatives
Reaction time	Ability to rapidly initiate a response to a stimulus
Grip strength	Strength of hand muscles

Fig. 16.2 Examples of basic motor skill exercising instruments, Lafayette Instrument Company, IN

Fig. 16.3 Right cow knee prepared for wet lab and cow knee arthroscopy setup

Second, measures are discussed that reflect the ability of effective and efficient task completion. This category is by far the most widely used set of measures to reflect performance with the time to complete a task being the strongest objective discriminator between novice and expert levels [1, 12, 20, 29, 30]. This has been proven in many studies and for many endoscopic disciplines [22]. Task time registration is widely used, easy to measure and easy to interpret during feedback, but task time does not necessarily reflect the quality of the executed task. Other parameters that were proposed are path length which has a high correlation with task time [31, 32]; a Likert scale to indicate task (in)completion, for example, in knot tying [33]; and the number of identified anatomic landmarks [34]. Motion analysis is a common method for assessing arthroscopic skill in the simulation laboratory. It has been validated and subsequently used to demonstrate the construct validity of arthroscopic tasks on benchtop simulators [35]. Furthermore, it has proved useful in monitoring the learning curves of trainees during simulated arthroscopic tasks [36–38]. Arthroscopists with higher technical skill are able to perform a task with fewer hand movements and shorter path length and in a shorter time. It does not rely on an assessor to evaluate skill and so is entirely objective. However, it does not provide specific feedback on performance to the trainee and cannot be used in the real operating theatre. Another type of instrument to monitor task completion in a more holistic approach is a global rating scale (GRS). This instrument has been developed for structured, objective feedback during training and may potentially contribute to steeper learning curves [33, 39]. For arthroscopy, two GRS have been proposed: Basic Arthroscopic Knee Skill Scoring System (BAKSSS) [40] and Objective Structured Assessment of Technical Skills Global Rating Scale (OSATS) [30]. Both GRS have been validated during arthroscopic benchtop model simulator training [30, 40]. By recording arthroscopic video and anonymising the subject, scoring can be performed remotely by raters, making these assessment tools more flexible and economical. In addition to these formal scoring systems, some other novel parameters have also been identified and used to objectively assess arthroscopic skill [41].

Third, measures are discussed that reflect patient safety. Patient safety can be defined as the prevention of damaging healthy tissues in the intra-articular joint space and tissues surrounding the access portals when performing surgical treatment [23]. Especially delicate tissues that have little to no healing potential (meniscal and cartilage tissue) need to be monitored as they are frequently probed even outside the arthroscopic camera view. Measures that were introduced that reflect safe tissue manipulation are motion smoothness [13], number of instrument-tissue collisions [12, 29, 42, 43] and the applied force on tissues [20, 23, 44]. It is essential that the safety measures are measured in real time during task execution and immediately feedback is given to the trainee when safe tissue manipulation levels are crossed. The feedback can be performed by auditive cues but also by tactile or visual cues such as colouring red the entire screen [45] or an colored arrow that represents the force direction [46].

16.2.3 How Can Arthroscopic Skills Be Trained in a Safe Environment?

The third step is to select or to design an adequate environment that allows training to achieve the learning objectives and monitoring of trainees' progress. When you consider the broadest definition of medical simulation, it has been practised in primitive forms for centuries [47]. Three main types of simulation environments for training arthroscopic skills are available: hands-on cadaver courses, anatomic benchtop models and virtual reality simulators. The main reasons to use a simulated environment instead of traditional training in the operating room are patient safety [1, 6, 7, 16, 30, 48–50], improved educational experience caused by easy access and better availability [51], cost-efficiency [51] and lastly simulators offer the opportunity to measure performance and training progress objectively [23, 31, 40, 51].

Questioning experts and residents what training means they prefer, cadaver courses are ranked number one, followed by high-fidelity simulators (e.g. synthetic knee), virtual reality simulators and box trainers [16]. So far, ESSKA has accredited 67 training centres in Europe, which predominantly focus on cadaver training (www.esska.org). Some companies have invested major sources (financial and intellectual) in human cadaver labs. They are officially run by professionals appointed by the company itself and academic curriculum is set by company related surgeons. Time slots are booked in far advance which shows that education in these facilities is highly popular. Despite its high reputation and acceptance, their role in training has not been academically evaluated. 'industry, education, arthroscopy' keywords did not retrieve any results. In order to receive the highest benefit from these facilities, a high level of academic relationship is required between academics and the industry. The largest asset of cadaveric specimens is that they offer the most realistic simulation environment in terms of tissue appearance and sensory feedback. Per specimen natural clinical variation is present, and tissue can be cut or sutured similarly as in the patient. Drawbacks of cadaver specimen are that they offer limited possibilities to provide feedback on performance and unpredictability of the natural variation [52]. Furthermore, human cadaver teaching is restricted because of limits on availability due to local regulations. Although animal cadavers are more readily available, their usage is limited mostly due to indifference.

Various anatomic models of joints are available. These models provide a consistent training environment for all participants, allow training of tissue manipulation (e.g. probing and cutting) and can be widely available due to their relatively low cost. Drawbacks are that these models lack realism and as a consequence are considered not challenging enough for training. Besides, they are not equipped with registration devices to track performance. To overcome these drawbacks, several research groups demonstrated that anatomic models can be very well used in combination with tracking equipment and their realism can be improved [1, 44]. Of course this comes at extra costs. The main asset of anatomic models is that they do offer haptic feedback, which is considered crucial in arthroscopic training [29, 50].

The last category is virtual reality systems (VR), which are inspired by the airline and, more recently, the gaming industry [3, 13, 29, 31]. The VR offer the ability of providing direct feedback on performance based on computer-generated outcome measures and can combine computer-based learning with part-task training. In theory, they also allow a wide range of natural clinical variation both in terms of pathology and joint shape and appearance, but only the former is offered in commercial systems [13]. A huge challenge for VR simulation of arthroscopy is still the implementation of realistic instrument-tissue behaviour, which is considered essential especially when training cutting, suturing and drilling [29].

To determine the potential of arthroscopic simulators, their educational value and user-friendliness have been evaluated and validated for different types of validity (internal, face, construct and content validity) [1, 2, 7, 13, 14, 29–31]. Modi and co-workers [2] indicated a range of limitations on the methodology used in these evaluation studies: the use of poorly validated outcome measures. In an effort to overcome a number of these limitations, we have set up a general protocol to assess face and construct validity of arthroscopic simulators [14]. This protocol enables evaluation and relative comparison of any type of simulator (virtual or benchtop model). At this stage, we have evaluated ToLTech Knee Arthroscopy Simulator™ (Touch of Life Technologies, Aurora, CO, USA: Simulator A), ArthroMentor™ (Simbionix, Cleveland, Ohio USA, previously known as the InsightArthroVR1 Arthroscopy Simulator (GMV, Madrid, Spain): Simulator B), VirtaMed ArthroS™ (VirtaMed AG, Zurich, Switzerland: Simulator D) and our own development the PASSPORT simulator (Delft University of Technology and Academic Medical Centre, The Netherlands: Simulator C). Participants were recruited and grouped in different experience levels; we only present the results of novices who had never performed an arthroscopic procedure and experts who had performed more than 60 arthroscopies [8]. Between 6 and 11 participants were present in each experience group for each simulator. All participants were scheduled a maximum period of 30 min.

Construct validity was assessed based on a single predefined navigation task. Nine anatomic landmarks had to be probed sequentially: medial femoral condyle, medial tibial plateau, posterior horn of the medial meniscus, midsection of the medial meniscus, ACL, lateral femoral condyle, lateral tibial plateau, posterior horn of the lateral meniscus and midsection of the lateral meniscus [1]. The task trial times were recorded by separate digital video recording equipment to guarantee uniformity in data processing. All participants performed the navigation task five times. Construct validity was determined with the Kruskal-Wallis test by calculation of overall significant differences in task time between the three groups for each of the five task trials. The significance level was adjusted for multiple comparisons with the Bonferroni-Holm procedure ($\alpha = 0.05$) [53]. Mann–Whitney U tests were used for pairwise comparisons to highlight significant differences. Construct validity was shown for Simulators C and D, as the novices were significantly slower than the experts in completing all five trials (Fig. 16.4). For Simulator A, only 2 out of 11 novices could complete all task trials within the set time limit. This indicates a clear distinction between novices and experts, which unfortunately cannot be supported by actual measurements. Simulator B partly demonstrated construct validity as the experts were faster in the second and third trials compared to the novices.

Face validity (realism), educational value and user-friendliness of the simulators were determined by giving the participants a second task in which up to three exercises had to be performed that were characteristic for that particular simulator and by asking them to fill out a questionnaire afterwards [14]. Questions were answered using a 10-point numerical rating scale (NRS) (e.g. 0 = completely unrealistic and 10 = completely realistic). Only the answers of the intermediates

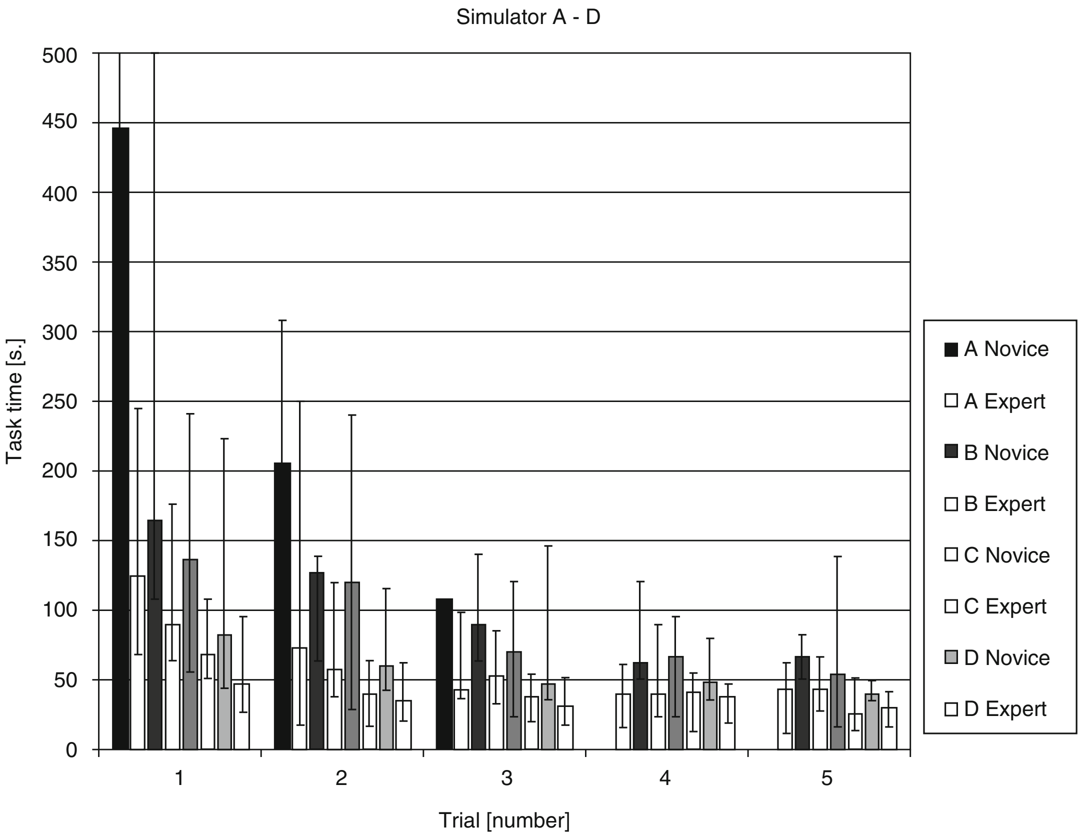

Fig. 16.4 Construct validity of four simulators for one navigation task

and experts regarding face validity and educational value were included. A value of 7 or greater was considered sufficient. Face validity of the outer appearance was demonstrated for all simulators, but only Simulator C demonstrated face validity for intra-articular joint realism and instrument realism (Fig. 16.5). This result was significantly different from Simulator B for intra-articular joint realism and significantly different between all simulators for instrument realism ($p < 0.05$). The explanation is that Simulator C is the only system that uses real instruments and a knee benchtop model to mimic sense of touch, which was considered the biggest asset by the participants. Simulators B and D have good user-friendliness, with the difference between Simulator A and B being significant ($p < 0.05$). All virtual reality simulators needed improvement of the sense of touch. All simulators could benefit from more realistic structures but were considered as valuable training tool in the beginning of the residency curriculum.

Deaneries in the UK have spent varying amounts of money developing simulation centres, but problems remain with the cost of buying and maintaining highly sophisticated simulators and the difficulty of ensuring geographical access to such simulators by all trainees. The skills involved can be divided into 'cognitive' and 'haptic'. The cognitive aspects could be trained with an online simulator. The concept of a 'cognitive trainer' for arthroscopy of the knee was explored in a joint project between the Royal College of Surgeons of England and Primal Pictures. This pilot study showed that it is feasible to develop a tutorial-based simulator that uses a simple and cheap interface to address the cognitive ('non-haptic') components of arthroscopic knee surgery [19]. The pilot VATMAS simulator, building on earlier work on the VE-KATS

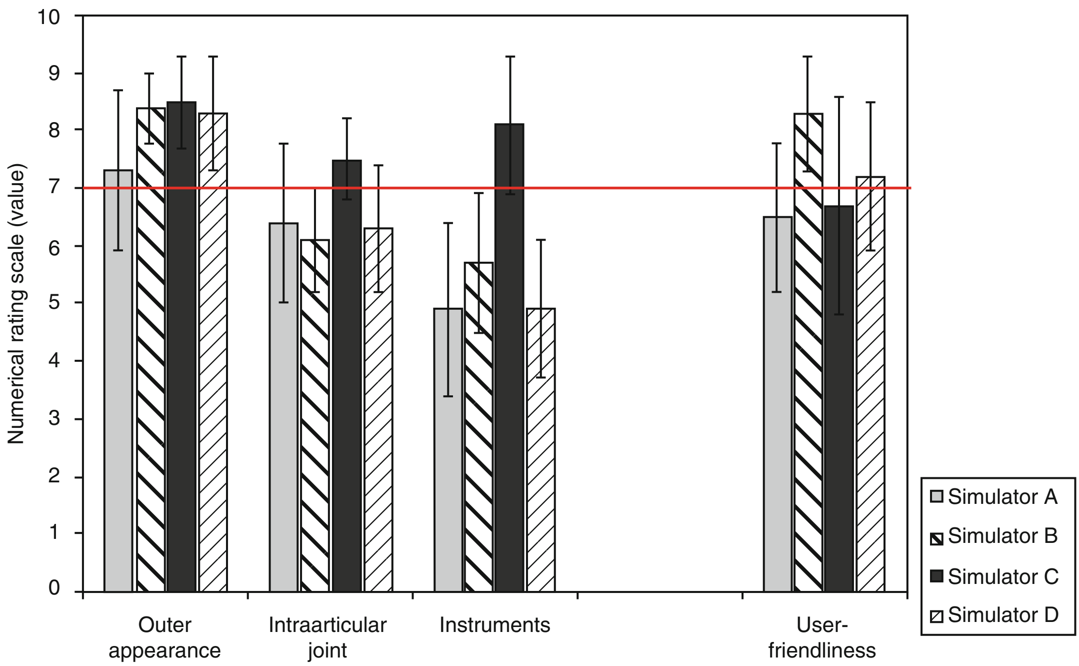

Fig. 16.5 Face validity and user-friendliness of four simulators

simulator (Fig. 16.6) [31], showed that it is possible to reproduce arthroscopic optics, with individual rotation of the camera and arthroscope. The arthroscope and probe can be independently manipulated, with indication of contact with hard and soft surfaces. An 'overview' can be provided if the trainee became disorientated (Fig. 16.6). Such a simulator can take the trainee through a series of tutorials whilst providing automated feedback based on time, accuracy and efficiency. From any point, images or videos from real arthroscopies can be called up. Focus group evaluation gave directions for further development.

16.3 Industry Involvement

Arthroscopic skills education is one of the most sophisticated zones of orthopaedic training which require compound efforts and resources. Despite extensive programming and detailed education environment, return may be low [54]. Teaching tools and environments should be categorised primarily on their fidelity, availability, acceptability and cost. Basic skills instruments, dry models, animal cadaver models, human cadaver models, virtual reality simulators and hands-on teaching in increasing order of fidelity are the teaching tools that are currently in use. Their utilisation differs from institution to institution based on the institution's education strategy, type of the hospital setting (whether it is a primarily teaching institution or not), motivation of the teaching staff and most importantly resources.

Industry involvement in orthopaedic practice is an approved manner under strict regulations [55]. Commercial companies supporting orthopaedic research, supply in patient care and education is highly welcome in the orthopaedic communities with academic hesitance. Companies provide educational support to the practising surgeon through company employee, peer to peer, printed matter (booklets, books), online materials (education platforms, surgical technique archives), company meetings (live surgery based), reserved sessions in society meetings and time in company running cadaver labs.

Visits to the clinics and accompanying surgeries by company employees are a well-established mode of educational support. Educational activity

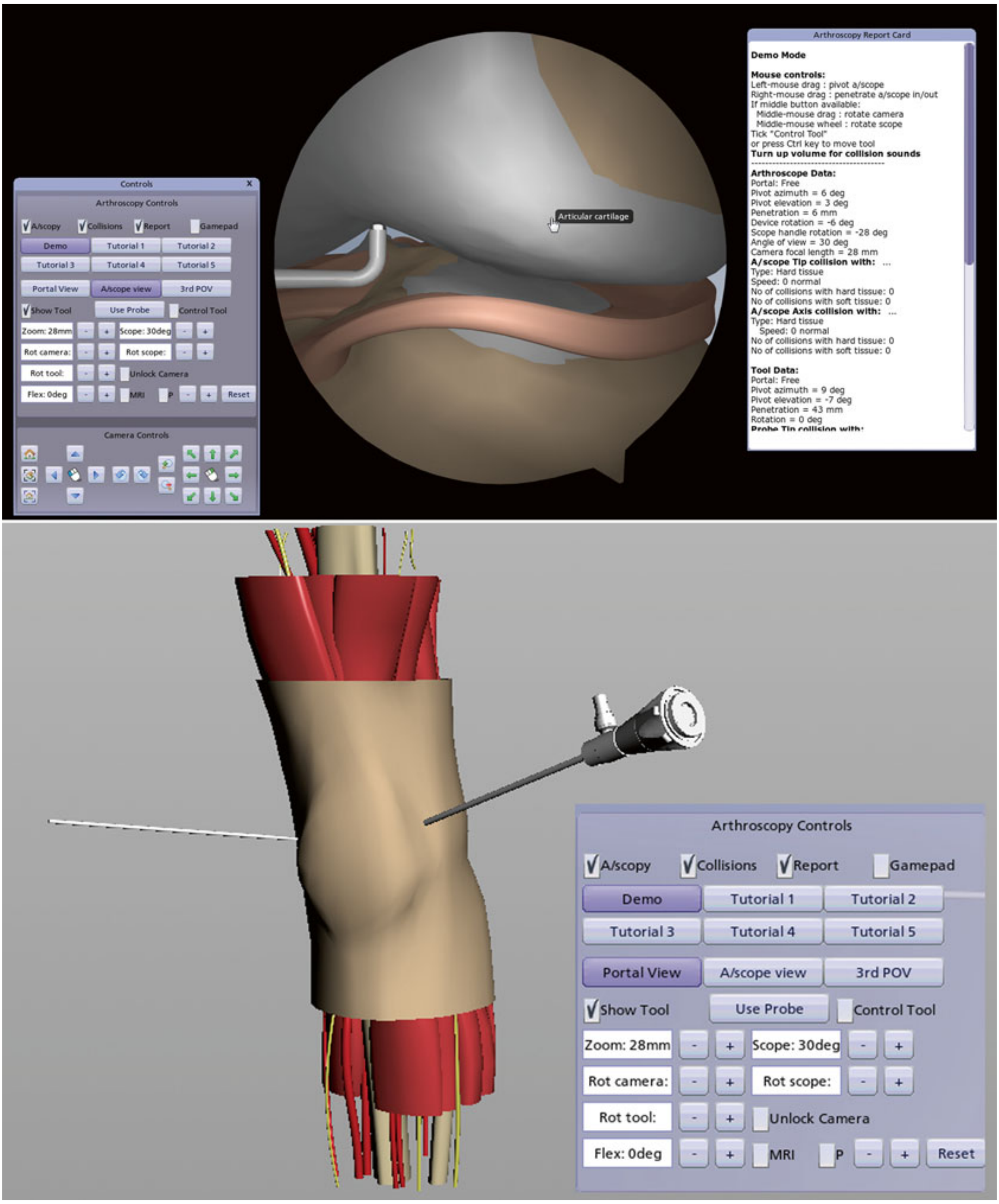

Fig. 16.6 Screenshot of VATMAS online simulator and screenshot of Epicardio simulator

performed by a surgeon who has disclosed relationship with a certain company is probably one of the most enjoyable educational setting for a young surgeon. The young surgeon has a personal relationship with the experienced surgeon. Company printed matters are usually neglected by the surgeons, and their use is limited to support of the verbal encounter that is taking place at the time. Online material is on the increasing edge for a long while and it is expected to increase further. Obvious reasons of having immediate access at all times to any need for information are indispensable at our digital age. Company meetings that promote an exceptional surgeon in a live surgery setting to a high number of attendants are also an attractive educational environment. Academic quality is expected to increase one notch if a similar setting takes place in a society meeting.

16.4 Take-Home Messages

Knowledge on anatomy, portal placement and identification of both condylar compartments are skills residents should have mastered before continuing training in the OR.

Sufficient measures are available to monitor training performance. However, some of these still have some subjectivity to them and are time consuming. Any development of future automated objective measures for the OR will prove useful and popular.

Thresholds for task completion and safety parameters should be determined with experts to provide validated training curricula.

Arthroscopic simulators are suited to train eye-hand coordination tasks such as navigation with arthroscope and probe, which is best trained in the beginning of the residency curriculum.

For all simulators, improvement is desired, especially the realism of joint space and sense of touch and the possibility to train portal placement.

Validation studies need to focus on concurrent and transfer validity to provide evidence that simulated training provides the required competency level when continuing training in the OR.

Online surgical simulators offer much potential in cost saving and accessibility, but further work needs to be done to demonstrate construct validity and predictive validity and to establish the optimal fidelity required for learning.

It must also be remembered that non-technical skills also need assessing, and the development of objective assessment methods for these non-technical skills is also required.

References

1. Tuijthof GJ, van Sterkenburg MN, Sierevelt IN, van Oldenrijk J, van Dijk CN, Kerkhoffs GM. First validation of the PASSPORT training environment for arthroscopic skills. Knee Surg Sports Traumatol Arthrosc. 2010;18(2):218–24.
2. Modi CS, Morris G, Mukherjee R. Computer-simulation training for knee and shoulder arthroscopic surgery. Arthroscopy. 2010;26(6):832–40.
3. Pedowitz RA, Esch J, Snyder S. Evaluation of a virtual reality simulator for arthroscopy skills development. Arthroscopy. 2002;18(6):E29.
4. Tuijthof GJM. Technical improvement of arthroscopic techniques. Delft University of Technology, Department of Design, Engineering and Production, Section Man–machine systems, Delft; 2003.
5. Barrett DS, Green RG, Copeland SA. Arthroscopic and endoscopic skills: a method of assessment. Ann R Coll Surg Engl. 1991;73(2):100–4.
6. Cannon WD, Eckhoff DG, Garrett Jr WE, Hunter RE, Sweeney HJ. Report of a group developing a virtual reality simulator for arthroscopic surgery of the knee joint. Clin Orthop Relat Res. 2006;442:21–9.
7. McCarthy AD, Moody L, Waterworth AR, Bickerstaff DR. Passive haptics in a knee arthroscopy simulator: is it valid for core skills training? Clin Orthop Relat Res. 2006;442:13–20.
8. O'Neill PJ, Cosgarea AJ, Freedman JA, Queale WS, McFarland EG. Arthroscopic proficiency: a survey of orthopaedic sports medicine fellowship directors and orthopaedic surgery department chairs. Arthroscopy. 2002;18(7):795–800.
9. Morris AH, Jennings JE, Stone RG, Katz JA, Garroway RY, Hendler RC. Guidelines for privileges in arthroscopic surgery. Arthroscopy. 1993;9(1):125–7.
10. Wolpert DM, Ghahramani Z. Computational principles of movement neuroscience. Nat Neurosci. 2000;3(Suppl):1212–7.
11. Kaufman HH, Wiegand RL, Tunick RH. Teaching surgeons to operate–principles of psychomotor skills training. Acta Neurochir (Wien). 1987;87(1–2):1–7.
12. Gomoll AH, Pappas G, Forsythe B, Warner JJ. Individual skill progression on a virtual reality simulator for shoulder arthroscopy: a 3-year follow-up study. Am J Sports Med. 2008;36(6):1139–42.
13. Bayona S, Fernandez-Arroyo JM, Martin I, Bayona P. Assessment study of insight ARTHRO VR arthroscopy virtual training simulator: face, content, and construct validities. J Robot Surg. 2008;2(3):151–8.
14. Tuijthof GJ, Visser P, Sierevelt IN, van Dijk CN, Kerkhoffs GM. Does perception of usefulness of arthroscopic simulators differ with levels of experience? Clin Orthop Relat Res. 2011;469:1701–8.
15. Biggs J. Constructing learning by aligning teaching: constructive alignment. In: Biggs J, editor. Teaching for quality learning at university. 2nd ed. Berkshire: Open University Press; 2003. p. 11–33.

16. Safir O, Dubrowski A, Mirsky L, Lin C, Backstein D, Carnahan A. What skills should simulation training in arthroscopy teach residents? Int J Comput Assist Radiol Surg. 2008;3(5):433–7.
17. Hui Y, Safir O, Dubrowski A, Carnahan H. What skills should simulation training in arthroscopy teach residents? A focus on resident input. Int J Comput Assist Radiol Surg. 2013;8:945–53.
18. Obdeijn MC, Bavinck N, Mathoulin C, van der Horst CM, Schijven MP, Tuijthof GJ. Education in wrist arthroscopy: past, present and future. Knee Surg Sports Traumatol Arthrosc. 2013.
19. Hurmusiadis V, Rhode K, Schaeffter T, Sherman K. Virtual arthroscopy trainer for minimally invasive surgery. Stud Health Technol Inform. 2011;163:236–8.
20. Chami G, Ward JW, Phillips R, Sherman KP. Haptic feedback can provide an objective assessment of arthroscopic skills. Clin Orthop Relat Res. 2008; 466(4):963–8.
21. Alvand A, Auplish S, Khan T, Gill HS, Rees JL. Identifying orthopaedic surgeons of the future: the inability of some medical students to achieve competence in basic arthroscopic tasks despite training: a randomised study. J Bone Joint Surg Br. 2011;93(12): 1586–91.
22. van Hove PD, Tuijthof GJ, Verdaasdonk EG, Stassen LP, Dankelman J. Objective assessment of technical surgical skills. Br J Surg. 2010;97(7):972–87.
23. Tuijthof GJ, Horeman T, Schafroth MU, Blankevoort L, Kerkhoffs GM. Probing forces of menisci: what levels are safe for arthroscopic surgery. Knee Surg Sports Traumatol Arthrosc. 2011;19(2):248–54.
24. Karahan M, Unalan PC, Bozkurt S, Odabas I, Akgun U, Cifcili S, et al. Correlation of basic motor skills with arthroscopic experience. Acta Orthop Traumatol Turc. 2009;43(1):49–53.
25. Unalan PC, Akan K, Orhun H, Akgun U, Poyanli O, Baykan A, et al. A basic arthroscopy course based on motor skill training. Knee Surg Sports Traumatol Arthrosc. 2010;18(10):1395–9.
26. Dubrowski A, Backstein D. The contributions of kinesiology to surgical education. J Bone Joint Surg Am. 2004;86-A(12):2778–81.
27. Neiman Z. Teaching specific motor skills for conducting to young music students. Percept Mot Skills. 1989;68(3 Pt 1):847–58.
28. Neequaye SK, Aggarwal R, Brightwell R, Van Herzeele I, Darzi A, Cheshire NJ. Identification of skills common to renal and iliac endovascular procedures performed on a virtual reality simulator. Eur J Vasc Endovasc Surg. 2007;33(5):525–32.
29. Moody L, Waterworth A, McCarthy AD, Harley P, Smallwood R. The feasibility of a mixed reality surgical training environment. Virtual Real. 2008;12: 77–86.
30. 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. 2008;90(4):494–9.
31. Sherman KP, Ward JW, Wills DP, Sherman VJ, Mohsen AM. Surgical trainee assessment using a VE knee arthroscopy training system (VE-KATS): experimental results. Stud Health Technol Inform. 2001;81:465–70.
32. Sherman KP, Ward JW, Wills DP, Mohsen AM. A portable virtual environment knee arthroscopy training system with objective scoring. Stud Health Technol Inform. 1999;62:335–6.
33. O'Connor A, Schwaitzberg SD, Cao CG. How much feedback is necessary for learning to suture? Surg Endosc. 2008;22(7):1614–9.
34. Bliss JP, Hanner-Bailey HS, Scerbo MW. Determining the efficacy of an immersive trainer for arthroscopy skills. Stud Health Technol Inform. 2005;111:54–6.
35. Howells NR, Brinsden MD, Gill RS, Carr AJ, Rees JL. Motion analysis: a validated method for showing skill levels in arthroscopy. Arthroscopy. 2008;24(3): 335–42.
36. Pollard TC, Khan T, Price AJ, Gill HS, Glyn-Jones S, Rees JL. Simulated hip arthroscopy skills: learning curves with the lateral and supine patient positions: a randomized trial. J Bone Joint Surg Am. 2012;94(10):e68.
37. Jackson WF, Khan T, Alvand A, Al-Ali S, Gill HS, Price AJ, et al. Learning and retaining simulated arthroscopic meniscal repair skills. J Bone Joint Surg Am. 2012;94(17):e132.
38. Alvand A, Auplish S, Gill H, Rees J. Innate arthroscopic skills in medical students and variation in learning curves. J Bone Joint Surg Am. 2011;93(19):e115–9.
39. Harewood GC, Murray F, Winder S, Patchett S. Evaluation of formal feedback on endoscopic competence among trainees: the EFFECT trial. Ir J Med Sci. 2008;177(3):253–6.
40. Insel A, Carofino B, Leger R, Arciero R, Mazzocca AD. The development of an objective model to assess arthroscopic performance. J Bone Joint Surg Am. 2009;91(9):2287–95.
41. Alvand A, Khan T, Al-Ali S, Jackson WF, Price AJ, Rees JL. Simple visual parameters for objective assessment of arthroscopic skill. J Bone Joint Surg Am. 2012;94(13):e97.
42. Mabrey JD, Gillogly SD, Kasser JR, Sweeney HJ, Zarins B, Mevis H, et al. Virtual reality simulation of arthroscopy of the knee. Arthroscopy. 2002;18(6): E28.
43. McCarthy AD, Hollands RJ. A commercially viable virtual reality knee arthroscopy training system. Stud Health Technol Inform. 1998;50:302–8.
44. Tashiro Y, Miura H, Nakanishi Y, Okazaki K, Iwamoto Y. Evaluation of skills in arthroscopic training based on trajectory and force data. Clin Orthop Relat Res. 2009;467(2):546–52.
45. Tuijthof G, Sierevelt IN, Dijk C, Kerkhoffs G. Is the presence of a dummy leg essential in the SIMENDO arthroscopy trainer? Knee Surg Sport Tr A. 2010;18(1):16.

46. Horeman T, Rodrigues SP, van den Dobbelsteen JJ, Jansen FW, Dankelman J. Visual force feedback in laparoscopic training. Surg Endosc. 2012;26(1):242–8.
47. Rosen KR. The history of medical simulation. J Crit Care. 2008;23(2):157–66.
48. Michelson JD. Simulation in orthopaedic education: an overview of theory and practice. J Bone Joint Surg Am. 2006;88(6):1405–11.
49. Poss R, Mabrey JD, Gillogly SD, Kasser JR, Sweeney HJ, Zarins B, et al. Development of a virtual reality arthroscopic knee simulator. J Bone Joint Surg Am. 2000;82-A(10):1495–9.
50. Ward JW, Wills DPM, Sherman KP, Mohsen AMMA. The development of an arthroscopic surgical simulator with haptic feedback. Future Generation Comput Syst. 1998;14(3–4):243–51.
51. Kunkler K. The role of medical simulation: an overview. Int J Med Robot. 2006;2(3):203–10.
52. Meyer RD, Tamarapalli JR, Lemons JE. Arthroscopy training using a "black box" technique. Arthroscopy. 1993;9(3):338–40.
53. Holm S. A simple sequentially rejective multiple test procedure. Scand J Stat. 1979;6(2):65–70.
54. Hall MP, Kaplan KM, Gorczynski CT, Zuckerman JD, Rosen JE. Assessment of arthroscopic training in U.S. orthopedic surgery residency programs–a resident self-assessment. Bull NYU Hosp Jt Dis. 2010;68(1):5–10.
55. Poehling GG, Lubowitz JH, Brand R, Buckwalter JA, Wright TM, Canale ST, et al. Patient care, professionalism, and relations with industry. Arthroscopy. 2008;24(1):4–6.

Part IV

Orthopaedic Sports Medicine Review Course

Upper Limb Injuries in Athletes

17

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17.1 Introduction

Upper extremity injuries usually occur in the shoulder, elbow, or wrist of the athletic population, especially in overhead disciplines. Traumas, biomechanical imbalance due to improper technique, and overuse cover the majority of the epidemiological factors in this population. Common types of injury include tendon problems, bone fractures, sprains, and dislocations.

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S. Zaffagnini et al. (eds.), *ESSKA Instructional Course Lecture Book*,
DOI 10.1007/978-3-642-53983-1_17, © ESSKA 2014

In this instructional course chapter, we try to summarize the most relevant pathologies that a sport physician can encounter in his/her daily practice.

Shoulder Injuries
- *Rotator Cuff Injuries*
- *SLAP Lesions and Biceps Pathology*
- *Glenohumeral Instability*
- *Acromioclavicular Joint Injuries*
- *Clavicle Fractures*
- *Humeral Head Fractures*

Elbow Injuries

Triangular Fibrocartilage Complex Injuries (Wrist)

17.2 Shoulder Injuries

17.2.1 Rotator Cuff Injuries

There are numerous lesions to rotator cuff that can occur in athletes. The classification of rotator cuff injury is based on the knowledge of the pathophysiology of events leading to rotator cuff failure.

Primary subacromial impingement is caused by violation of the rotator cuff between the greater tuberosity and the coracoacromial arch. Abnormal acromial morphology, acromial spurs, and acromioclavicular joint arthritis are identified as predisposing factors.

The compression of the rotator cuff between the coracoacromial arch and the humeral head may lead to inflammation and tears of the rotator cuff tendons.

Neer classified three stages of the impingement syndrome as (1) edema and hemorrhage in the subacromial space and supraspinatus tendon, (2) thickening and fibrosis in the distal tendon insertion, and (3) full-thickness rotator cuff tears. Primary impingement is typically diagnosed in older overhead athletes with a stable shoulder, whereas it is rare in young throwers. The spectrum of cuff pathology ranges from tendinopathy to partial- or full-thickness tears.

Secondary impingement is a very common cause of pain in the young overhead athlete (swimmers, throwers, tennis players) and often results from preexisting ligamentous laxity or acquired traumatic capsular laxity. Because of this pathologic laxity, the humeral head translates anteriorly, producing impingement of the supraspinatus tendon against the coracoacromial arch.

Internal impingement is characterized by contact of the articular surface of the rotator cuff with the posterior and superior glenoid rim and labrum in the extremes of shoulder abduction and external rotation.

In normal throwers there is no significant contact between the posterior cuff and the adjacent glenoid. However, a mild instability that results from chronic stretching of the anterior capsular allows repetitive impactions to occur.

Tears of the rotator cuff are common in overhead athletes mostly due to overuse and rarely because of trauma. The cause is often multifactorial; tensile overload, outlet impingement, and internal impingement are common causes of cuff pathologic conditions in this population [1].

The physical examination shows weakness and pain related to the tendon involved, mostly again resistance. A positive painful arc test result and a positive external rotation resistance test result were the most accurate findings for detecting rotator cuff tears, whereas the presence of a positive lag test (external or internal rotation) result was most accurate for the diagnosis of a full-thickness rotator cuff tear [2]. Of course ER against resistance, Jobe test, Whipple test, and lift-off test offers great help in order to locate the damage among the different tendons.

The most reliable imaging is actually MRI-arthrogram, but due to its cost and invasivity, a standard MRI is mostly used. Standard X-rays are valuable especially in differential diagnosis among glenohumeral arthritis and calcific tendonitis.

The management of cuff problems is initially conservative with a personalized physical therapy program of at least 3 months. Injections are not indicated in this population especially with steroids.

Surgical intervention is considered if nonoperative management fails or if a full-thickness tear is observed that should lead to an immediate (nonurgent) operation.

Partial-thickness rotator cuff tears of less than 50 % may benefit from surgical debridement. Partial-thickness tears greater than 50 % or full-thickness tears should be repaired. In patients with mild instability and secondary impingement, a glenohumeral stabilization procedure should be considered as a crucial component of the surgical management. In patients without instability, an arthroscopic subacromial decompression can be performed.

A PubMed search assessed treatment options providing expedited recovery time and return to competition. Twelve of 231 articles fit the objective criteria; 90.5 % of professional contact athletes, 40 % of professional overhead athletes, and 83.3 % of recreational athletes fully recovered following rotator cuff tear surgical repair [3].

Prompt surgical treatment for full-thickness rotator cuff tears may be appropriate for contact athletes and recreational overhead athletes. Although professional overhead athletes have low recovery rates, surgical repair of full-thickness rotator cuff tears may still be indicated.

17.2.1.1 Cuff Repair: Conservative Versus Arthroscopic and Open Treatment

The clinical results of reconstructions of rotator cuff tears are described as good to excellent in the literature. In order to further improve the outcome, minimally invasive technique like the mini-open approach or all arthroscopic repairs of the rotator cuff were introduced.

Minimally invasive techniques may have the potential to reduce postoperative pain, postoperative stiffness, and damage of the deltoid muscle. Especially in large and massive tears, a more controlled release of the retracted tendon might be achieved. Moreover, the decision if a tear is repairable can be decided without damaging the deltoid muscle which might have a potential impact on following procedures like a reverse shoulder arthroplasty. Finally, there is a clear improvement for the minimally invasive approach concerning the cosmetic appearance compared to traditional open approaches.

A critical review of the literature, however, questions the superiority in the clinical results of minimally invasive techniques compared to open rotator cuff repairs and even to conservative treatment.

Conservative treatment of rotator cuff tears also leads to acceptable midterm results in the literature. However, there is an increase of fatty muscle infiltration and decrease of the acromiohumeral distance. Moreover, some repairable rotator cuff tears become irrepairable over time.

Randomized controlled trials are necessary in order to critically analyze potential benefits of minimally invasive techniques to traditional approaches as well as conservative treatment, especially as minimally invasive techniques increase the overall cost by increased surgical time and higher implant costs and are technically more demanding. Moreover, a critical analysis is necessary, which tears need to be repaired and which tears might be treated conservatively.

17.2.2 SLAP Lesions and Biceps Pathology

Several disorders involving the biceps tendon have been identified as common sources of shoulder pain in the overhead athlete.

Patients with biceps tendonitis have anterior shoulder pain intensified with overhead activities. The most common cause is subacromial impingement.

Subluxation or dislocation of the biceps tendon from its groove can occur in conjunction with a subscapularis tendon disruption.

Tendon debridement, release, or tenodesis is indicated if conservative treatments fail in the patients with an associated subscapularis tear.

Injuries to the superior labrum at the biceps–labral anchor are common in athletes. This lesion can result from repetitive microtrauma as in a throwing athlete or direct trauma.

SLAP lesions can be classified into four types: type 1, fraying of the superior labrum; type 2, the biceps anchor that is disrupted; type 3, bucket-handle tear of the labrum; and type 4, bucket-handle labral tear that extends into the biceps tendon [4].

If conservative treatment fails, type I and III lesions are treated with debridement and careful evaluation for glenohumeral instability. Type II lesions can be treated with arthroscopic fixation of the biceps anchor with good results. Good outcomes and full return to their pre-injury level of sport participation have been observed in athletes who have received a surgical stabilization of their SLAP II lesions.

17.2.3 Glenohumeral Instability

Primary acute shoulder dislocation is a common orthopedic injury, with an incidence rate of 1.7 % in the general population [5]. Recurrence of instability, defined as a single dislocation or subluxation event, and pain preventing the return to sport activities are the most common reasons for concern [6]. There is growing interest in identifying the best treatment in patients with primary dislocation of the shoulder, especially in populations at higher risk of recurrence, such as young physically active adults [7].

In case of primary acute dislocation of the shoulder, one of the approaches most widely used is the reduction of the glenohumeral joint and immobilization followed by a variable period of rehabilitation to restore shoulder range of motion and strength [8]. Immobilization has been performed in either internal or external rotation, with discordant results. Despite enthusiastic results proposed with the use of external rotation bracing [9], it has been proposed that it may not be as effective as claimed in preventing recurrent anterior dislocation of the shoulder.

Surgery has generally been used for chronic recurrence/instability. However, whether surgical management of primary dislocation is warranted for a first-time traumatic anterior dislocation of the shoulder is still debated.

Although, once a dislocation has occurred, the shoulder is less stable and more susceptible to redislocation [10], the risk of recurrent instability (defined as a single dislocation or subluxation event) after any type of treatment is higher in males and young people [11–13].

Several studies demonstrated the youth have a major risk to develop two or more recurrent dislocations [14, 15]. Patients who are from 23 to 29 years old at the time of the original injury have a risk of 0.5 in comparison with the patients who had been 12–22 years old. Instead the risk was reduced to 0.15 when patients were 30–40 years old at the time of the injury compared with those who had been 12–22 years old [16]. On the other hand, the role of sport activities is controversial. Some authors suggested that sport participation can improve the risk of recurrence, whereas others did not confirm this correlation by using the age-adjustment logistic regression analysis [17].

Finally, dislocation of the shoulder can be associated with frequent injury patterns, such as the classical Bankart lesion and the Hill–Sachs lesion. In terms of soft tissue injuries, the Bankart lesion can be found in 35 % of the shoulders and the rotator cuff tear in 10 % [8, 18–37]. Less frequent injuries include labral, humeral avulsion glenohumeral ligament (HAGL), superior labral tear from anterior to posterior (SLAP), and anterior labral periosteal sleeve avulsion (ALPSA) lesions. In terms of bony lesions, the glenoid defect can be found in 18 % of the shoulders, the humeral head defect in 30 %, and the combination of these in 22 % [8, 16, 18–21, 23–35, 37–40]. This finding could affect the clinical outcomes of patients and the rate of recurrence [18, 19, 21].

The best management of the primary acute shoulder dislocation has not been clarified yet. Both conservative and surgical managements have been proposed; however, the current literature fails to provide a definitive recommendation to treat these patients.

Conservative management usually consists of immobilization in internal rotation (IR) for a period of time raging from 3 to 6 weeks. However, several authors proposed shorter periods or no immobilization at all [41]. Paterson et al. [42] showed that the immobilization in conventional sling for more than 1 week does not provide benefit in younger patients with primary anterior shoulder dislocation. The recurrence rate is strictly related with the age of the patient, and people less than 30 years at the time of injury have a very high risk of recurrence.

Some authors proposed an immobilization with 10° of external rotation and abduction [9, 43, 44], whereas others used an immobilization up to 15–20° of external rotation [45].

In patients who underwent a conservative treatment, the risk of recurrent instability including subluxation and dislocation has been estimated from 25 % up to more than 90 % [8, 9, 16, 19, 21, 22, 25, 28, 29, 34–37, 39, 40, 43–50]. The great variability in the recurrence rate is likely related to different patients enrolled and follow-up length through the studies. Patients such as top-level athletes and military cadets have the highest risk to develop a recurrent instability [49, 50].

The position of the shoulder during the immobilization period significantly affects the recurrence rate. The internal rotated position is associated with a risk ranging from 30 % [9] to 70 % [28], while the external rotated position with a risk ranging from 0 % [9] to 37 % [45]. In the systematic review by Paterson et al. [42], clinically important benefits for the bracing in external rotation over the traditional sling immobilization have been found, despite no statistically significant difference in recurrence rates reported.

The superiority of external rotation over internal rotation is also confirmed by radiological studies [20, 32] that show the external rotation of arm is associated with a decrease of hemarthrosis, reduction of anterior capsule detachment, and labral lesions.

Finally, a cadaveric study showed that there is no glenolabral contact when the shoulder is held with 60° of internal rotation in the shoulder affected by a Bankart lesion. The labrum–glenoid contact force increases when the arm passes from internal rotation to neutral rotation, reaching a maximum contact at 45° of external rotation [51]. Although these data support the use of an externally rotated immobilization to provide an anatomic healing, the compliance of the patients can be difficult with high degrees of external rotation [9]. No recommendations about the degree of external rotation to immobilize the arm with the best clinical outcome can be drawn; however, protocols with immobilization in a slight external rotated position can be more successful.

Several authors investigated the arthroscopic shoulder stabilization for the management of first-time acute dislocation [19, 21, 25, 46]. The soft tissue stabilization aims to restore the native capsulolabral anatomy and is performed as a unique treatment when no or mild bone defects can be found. The recurrence of instability with this procedure has been estimated around 10 % [19, 25]. Some authors also evaluated the role of arthroscopic lavage reporting different results in terms of the recurrence rate that ranged from 20 % [35] to 55 % [27].

Robinson et al. [27] performed a prospective double-blind randomized clinical trial comparing arthroscopic washout alone with arthroscopic stabilization in patients with ages between 15 and 35 years old. At 2-year follow-up time, the authors reported a significantly lower recurrence rate in patients managed with surgical stabilization (7 % vs. 38 %).

The management of the bone loss in traumatic anterior glenohumeral instability is extremely challenging. Some authors provided algorithms to choose the appropriate surgical treatment according to the size and the location of the defect [52–54]. The glenoid bone loss less than 25 % is most frequently managed with arthroscopic osseous Bankart repair or capsulolabral repair. On the other hand, the glenoid bone loss more than 25 % is mainly managed by open reconstruction with bone graft, Bristow, or Latarjet procedure [38]. Recently, arthroscopic coracoid transfer has been described [55]. If the coracoid is no longer available, such as in revision cases, iliac crest bone autograft or allograft bone can be used for the bony augmentation [56]. Arthroscopic bone block procedures have also described [57, 58]. Finally, the remplissage technique has been proposed to manage the engaging Hill–Sachs lesions by performing a capsulotenodesis of the posterior infraspinatus tendon and posterior capsule to fill the Hill–Sachs defect. The aim is to prevent humeral defect from engaging with the anterior glenoid [59, 60].

Burkhart and De Beer [61] in a landmark study found a recurrence rate of 67 % in patients with significant bone defects in whom a soft tissue repair was performed. The management of bone defects allows to reduce the recurrence rate with a risk of 7 % associated with glenoid bony defect, 13 % with humeral bony defect, and 6 % with both glenoid and humeral involvement [38].

A Cochrane review [10] comparing surgical versus nonsurgical management found limited evidence supporting primary surgery for young adults, usually male, participating in highly demanding physical activities who had sustained their first acute traumatic shoulder dislocation.

In case of primary acute dislocation of the shoulder, the conservative management still remains the most widely performed consisting in the reduction and immobilization of the glenohumeral joint. Although the shoulder can be immobilized in both internal and external rotated positions, the external rotation provides lower rates of recurrence. To increase the compliance of the patients for the external rotated immobilization, protocols with immobilization in a slight external rotated position should be recommended.

The available evidence from RCTs supports primary surgery for the management of primary acute traumatic shoulder dislocation in young adults participating in highly demanding sport or work activities. However, no recommendation can be drawn on the best surgical approach in terms of clinical outcomes and recurrence rate.

Finally, there is no evidence available to determine the superiority of surgery over conservative management in patients at lower risk of redislocation. Recurrence rates are age related and may be associated with male gender, bone defects, and sport activities. Therefore, future studies have to consider patients for gender, type of occupation or sports, and type of bone defects to provide the actual relationship between these factors and the increase of the rate of recurrence.

17.2.4 Acromioclavicular Joint Injuries

Injuries to the acromioclavicular (AC) joint are common in sports and may lead to instability or degenerative changes requiring surgical intervention.

The AC articulation is an arthrodial joint between the acromial end of the clavicle and the medial margin of the acromion of the scapula. Articular joint capsule with superior and anterior AC ligaments provides AC joint horizontal (anteroposterior) stability. Vertical stability and

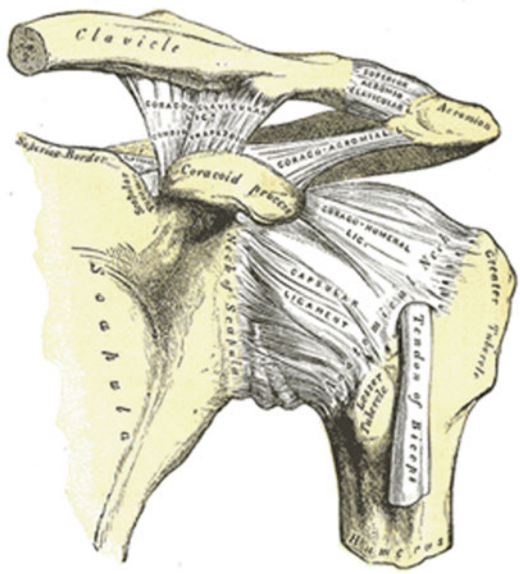

Fig. 17.1 Shoulder anatomy

compression resistance across the AC joint are achieved by the coracoclavicular (CC) ligaments—trapezoid and conoid. The deltoid and trapezius muscles are dynamic stabilizers of AC joint movements that consist of gliding motion of the articular end of the clavicle on the acromion and rotation of the scapula forward and backward upon the clavicle. The articular disk is rarely presented in AC joint (Fig. 17.1).

The characteristic history for an AC joint injury is a direct blow to the lateral shoulder. This frequently occurs from a fall with an adducted arm and rarely from a fall on an outstretched arm or flexed elbow that may cause a superiorly directed force through the humeral head to the acromion resulting in an AC injury. The most common sports associated with AC joint injuries include cycling, skiing, hockey, rugby, and football. The severity of injury is based on the direction and degree of forces across the joint. The spectrum of injury ranges from sprain to disruption of the AC ligaments—typically injured first—and CC ligaments, being disrupted with more significant force [59, 60].

The physical examination is notable for localized tenderness over the AC joint, with or without an obvious deformity and the prominence of the

AC joint that is due to the shoulder complex being displaced inferiorly. Although glenohumeral motion is preserved, it is frequently decreased secondary to pain, and it is most notable with cross-body adduction or resisted abduction. Also, the horizontal component of AC joint instability is indicated by increased distal clavicle posterior translation with the acromion fixed by the other hand [62].

Diagnostics include imaging studies: AP (panorama) stress view of both AC joints, axillary dynamic radiological evaluation in patient's supine position, Alexander modified scapular lateral view to demonstrate the horizontal instability, and Zanca view with the X-ray beam tilted at 10° in caudo-cranial dislocation. MRI should not be the imaging modality of first choice, but it could be useful in assessing clinically low-grade injuries that have not settled, thus excluding higher-grade injury, or if associated glenohumeral soft tissue injuries are assumed [63].

Instead of the obsolete Tossy–Allman classification, nowadays the Rockwood classification system is almost universally used and is based on the degree and direction of disrupted anatomy (Fig. 17.2).

Type I AC joint injury is a strain to the AC ligament without presenting significant instability. Type II reveals a complete tear of the AC ligaments with intact CC ligaments and includes a slight vertical separation of the AC joint. In type III, IV, and V AC joint separations, both sets of ligaments are disrupted. A type III injury occurs when the distal clavicle is completely displaced, while in a type IV injury there is posterior displacement of the clavicle through the trapezius muscle. In type III–VI injuries, the deltoid and trapezius muscles are detached from the distal clavicle. In type V AC joint separation gross displacement, often between 100 and 300 % of the width of the clavicle, is present. In a type VI injury, the distal clavicle is inferiorly displaced, to be either subacromial or subcoracoid [60, 64].

The type of injury dictates the treatment modality.

Nonoperative treatment should be symptomatic in the acute phase and functional in the subacute/chronic phase. For all acute type I and most type II injuries, nonoperative treatment with rest, immobilization in cast or sling (1–2 weeks), cryotherapy, and early motion are recommended. A key pillar of physical rehabilitation programs represents the strengthening of the spino-scapulo-humeral function chain. Main focus should be kept on the periscapular muscles to stabilize the scapula actively due to the lack of passive ligamentous suspension to the clavicle.

Although there is a possibility for skin and soft tissue-related pitfalls of nonoperative treatment with external immobilization, the most common complication of conservative therapy is a chronic pain that increases under physical activity. It is presented in certain numbers of conservatively treated type II and III AC joint dislocations as mainly a result of primarily misdiagnosed persistent horizontal instability. The reason for chronic pain could be also lesion of articular disk or posttraumatic osteoarthritis due to chondropathy, subchondral bone marrow edema, and cysts. Today, a biologic treatment with stem cells, cytokines, and growth factors from serum/plasma injections improves the status of a posttraumatic arthritic AC joint [65].

Operative treatment consists of early surgical AC joint stabilization or chronic AC joint dislocation therapy.

It is mostly indicated for acute type IV–VI injuries within a time frame of 2–3 weeks after injury. The operative treatment of type III AC injuries remains controversial. It varies on a case-by-case basis, and if it is not operated, most commonly it is initially treated nonoperatively with reserve for surgical stabilization in chronically symptomatic injuries. A recent meta-analysis indicates that operative treatment in such cases results in better cosmetic outcome but longer duration of sick leave compared to nonoperative treatment. No difference regarding the strength, pain, throwing ability, and incidence of AC joint osteoarthritis has been observed between both treatment groups [66].

Multiple open stabilization procedures for the AC joint have been described. Many of these techniques, including AC joint transfixation (with Kirschner wires, Steinman pins, or screws) and

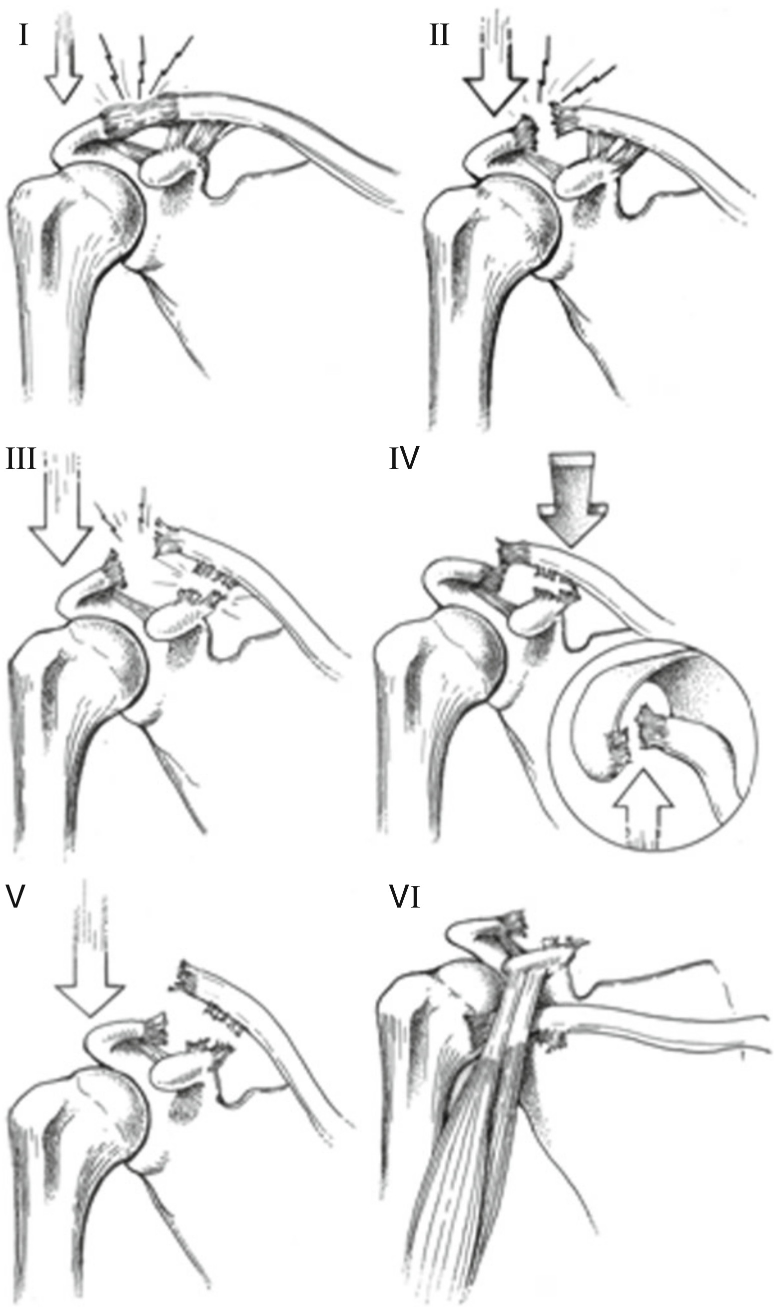

Fig. 17.2 The Rockwood classification system

dynamic muscle transfers, have fallen out of favor due to high complication rates (K wires migration, AC joint redislocation, infection, etc.).

Lately, anatomic as well as minimally invasive repair techniques with major focus on restoration of the CC ligaments have been described for AC joint reconstruction.

Operative treatment of acute AC dislocation includes CC stabilization with different techniques of fixations: Bosworth screw, hook plate, PDS sling, and TightRope (Arthrex, USA) or MINAR (Karl Storz, Germany) system, with repair or reconstruction of the CC ligaments. Those techniques could be utilized to assist

Fig. 17.3 TightRope fixation

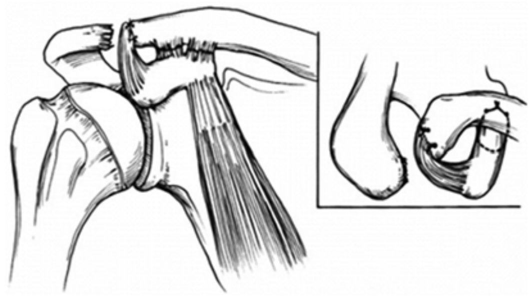

Fig. 17.4 Modified Weaver–Dunn procedure

stabilization in chronic injuries, but should not be used alone. Possible disadvantages and complications of these procedures are as follows:

- Malpositioning, screw breakage, damage of the CC repair, and necessity to remove a screw represent disadvantages of Bosworth implant.
- Possible loss of reduction, acromion osteolysis or fractures, and the need for plate removal after 3 months are the handicaps of transarticular hook plate [67].
- Requirement a large exposure with soft tissue damage and redislocation rate upon it is very high in technique of PDS sling around the coracoid and clavicle [60].

The TightRope or MINAR system presents the mostly popular method of CC fixation performed with mini-open procedure. It includes replacing the conoid and trapezoid ligaments separately with nonabsorbable sutures and titanium buttons on the superior clavicular side and inferior coracoid side (Fig. 17.3).

Nowadays, arthroscopically assisted techniques improve anatomic AC joint reconstruction by providing initial static and dynamic stability in both the vertical as well as the horizontal plane, superior to the native CC ligaments. Lately, due to frequent episodes of recurrent AC joint dislocation, two TightRope systems of new generation with drill holes directed in the anatomic course of CC ligament are recommended. Finally, in types IV and V, repair and suture of the superior AC joint ligament and delto-trapezoidal fascia are performed [60].

While the arthroscopic procedure allows for contemporaneous diagnostic and therapeutic treatment of intra-articular glenohumeral lesions, mini-open procedure gives a better overview on coracoid drill holes and the possibility of delto-trapezoidal fascia, which is ignored in all arthroscopic techniques [68].

The modified Weaver–Dunn procedure is the most common reconstruction technique of chronic AC joint dislocation. The procedure is done to essentially replace the CC ligament with the CA ligament. It includes clavicle resection (optionally), detaching the acromial end of the CC ligament, and possibly shortening it, and attaching the remaining ligament to the remaining clavicle with sutures. Distal clavicle removal at the time of CC ligament reconstruction is generally favored because of higher rates of AC joint arthrosis with distal clavicle preservation. This procedure is not indicated for acute cases when CC and AC ligaments are likely to heal spontaneously after repair (Fig. 17.4).

Other methods for stabilization of chronic AC joint dislocation are CC ligament reconstruction with HS tendon grafts or artificial ligaments and bone grafting between the clavicle and coracoid (Gene-Wolf procedure).

However, biomechanical and clinical data proved anatomic CC ligament reconstruction using autologous semitendinosus tendon to be superior to the Weaver–Dunn procedure for chronic cases [60].

Following surgery, exceptional protection of the AC joint repair has to be guaranteed in the immediate postoperative period, which minimizes the risk of redislocation. It is crucial to provide a sufficient support to the forearm and elbow to neutralize CC gravity distraction forces. The patient is placed into a sling with a waist support in an adducted and internally rotated position for 4–6 weeks. A limited range of movements (rotations) is allowed out of the sling by physiotherapeutic instruction only. Upon achievement of full, pain-free passive and active range of motions, the patient could start with strengthening exercises, but not until 8 weeks after surgery. Carrying of weight on the hanging arm is still not allowed during this time. High compliance of the patient is imperative to the rehabilitation process. Return to contact sports is avoided for approximately 5–6 months [69].

17.2.5 Clavicle Fractures

The clavicle is an S-shaped, membrane bone that connects the sternum and scapula/glenohumeral joint. It is subcutaneous and can be easily seen and palpated. It is connected to the sternum through the sternoclavicular joint and with the acromion at the acromioclavicular joint. The lateral third is flattened, which is the optimal shape for the attachment of ligaments, muscles, and aponeurosis, and the medial two-thirds are tubular, a shape that provides optimal axial-load bearing. Many ligaments attached to the clavicle provide stability of the articulations. At the sternal side there are anterior and posterior capsules like primary stabilizers and interclavicular and costoclavicular ligament. On the acromial side, at the AC joint, stability is provided by the AC ligament and coracoclavicular ligament. The coracoclavicular ligament is actually formed from two separate ligaments, the conoid and the trapezoid that are attached from the coracoid to the inferior surface of the lateral clavicle. The conoid ligament predominantly restrains superior and anterior loads to the AC joint, and the main role of the trapezoid ligament is posterior load restrain. The AC ligament is attached to the superior-lateral side of the clavicle and overlies the AC joint.

Three muscles originate from the clavicle: the sternohyoid, the pectoralis major, and the deltoid. As well, three muscles insert into the clavicle: the sternocleidomastoid, the subclavius, and the trapezius. The forces of the muscles may be the reasons for bone fracture, by deforming forces applied to the bone, and fragment displacement depends on the muscular and ligamentous attachments. The middle third is the weakest part of the bone, and several of the fractures occur at that part. The clavicle is in close contact with many other important structures that can be injured with the fracture: the subclavian artery and vein, the brachial plexus, and the apices of the lung.

The minimum force that leads to clavicle fracture during axial loading is two to three times the average body weight, and the clavicle is weakest in the middle third.

Clavicle fractures are common injuries, representing about 4–10 % of all adult fractures and 35–45 % of all fractures that occur in the upper limb girdle. The most frequent site of injury is at the middle third (group I fractures), accounting for approximately 72–80 % of all fractures of the clavicle. Approximately 25–30 % of clavicle fractures occur at the lateral clavicle (group II). Fractures of the medial clavicle are quite rare, accounting for 2 % of all clavicle fractures. Fractures are commonest in males under 30 years, and incidence increases in the very elderly where it is almost equal in males and females. The most common cause of fracture is falls; actually, the most common mechanism for clavicle fractures is a fall directly onto the shoulder; and in the young age group, the causes of clavicle fractures are sports, falls from the high, and road traffic accidents. For sport injury, male to female ratio is 7:1.

Plenty of classification schemes have been presented. However, the Allman classification scheme with the Neer modification is the most commonly used and is listed in detail below [70, 71]:

Group I—Fracture of the middle third
Group II—Fracture of the distal third
- Type I—Minimally displaced/interligamentous
- Type II—Displaced due to fracture medial to the coracoclavicular ligaments
  - IIA—Both the conoid and trapezoid remain attached to the distal fragment
  - IIB—Either the conoid is torn or both the conoid and trapezoid are torn

Type III—Fractures involving articular surface
Type IV—Ligaments intact to the periosteum with displacement of the
proximal fragment
Type V—Comminuted
Group III—Fracture of the proximal third
Type I—Minimal displacement
Type II—Displaced
Type III—Intra-articular
Type IV—Epiphyseal separation (observed in patients aged 25 years and younger)
Type V—Comminuted

A new classification was developed by Robinson based on radiological review of the anatomic site and the extent of displacement, comminution, and articular extension [72]. Fractures of the medial fifth (type 1), undisplaced diaphyseal fractures (type 2A), and fractures of the outer fifth (type 3A) usually had a benign prognosis. The incidence of complications of union was higher in displaced diaphyseal (type 2B) and displaced outer fifth (type 3B) fractures. In addition to displacement, the extent of comminution in type 2B fractures was a risk factor for delayed nonunion of fractures (Figs. 17.5, 17.6, 17.7, 17.8, 17.9, and 17.10).

When the patient with a fractured clavicle presents at the ER, usually steps to determine diagnosis are anamnesis, clinical examination, and diagnostic imaging.

The first thing is to find out about the injury and how it occurred. The clavicle is subcutaneous, and there is usually visible deformity at the

Fig. 17.6 Lateral third clavicle fracture minimally displaced (3A)

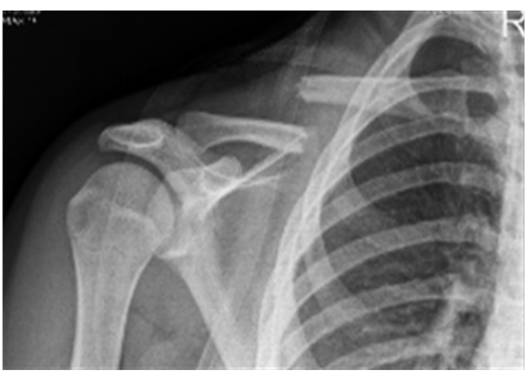

Fig. 17.5 Midshaft right clavicle fracture with dislocation (2B)

Fig. 17.7 Comminution of lateral end right clavicle (3B)

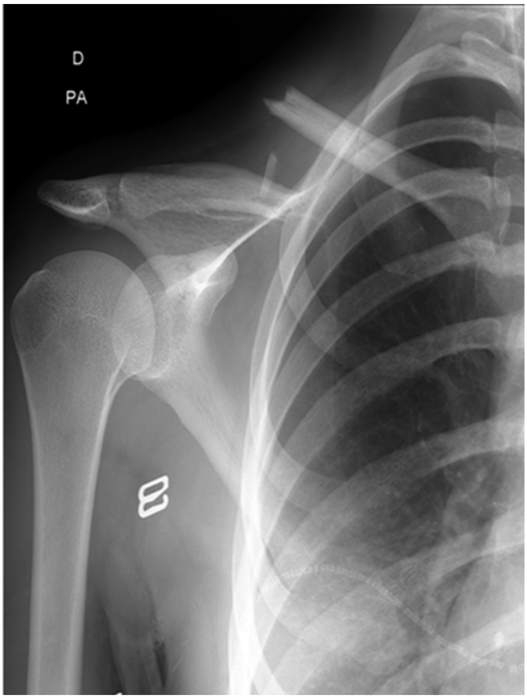

Fig. 17.8 Severe displacement of middle third, right clavicle fracture

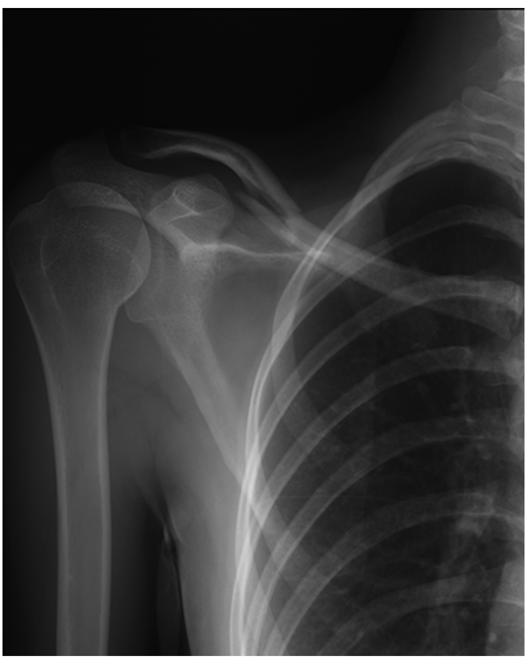

Fig. 17.9 Midshaft right clavicle fracture minimally displaced (2A)

Fig. 17.10 Lateral end left clavicle fracture, no displacement (3A)

initial observation. The mechanism of injury is usually direct blow to the shoulder, by falling onto the shoulder, or in a traffic accident. Clavicle fractures can be very painful and may make it hard for the patient to move his/her arm. Additional symptoms include sagging shoulder (down and forward), inability to lift the arm because of pain, a grinding sensation if an attempt is made to raise the arm, a deformity or “bump” over the break, bruising, swelling, and/or tenderness over the clavicle. There is usually an obvious deformity, or “bump,” at the fracture site. Gentle pressure over the break will bring about pain. Although a fragment of the bone rarely breaks through the skin, it may push the skin into a “tent” formation. We must be careful of the presence of nerve or blood vessel injuries. The shoulder is internally rotated, protracted, and inferiorly displaced.

Diagnostic imaging includes two plain radiographs of the entire shoulder: anteroposterior and 45° cephalic tilt anteroposterior views. Also it is useful to make the whole chest radiographs while standing, to compare the relative positions of the scapulae and clavicle shortening. CT scan is used in the evaluation of nonunion and malunion and in the medial third fractures when standard radiographs make it hard to determine the medial part.

The goal of clavicle fracture treatment is to restore the anatomic position of the fractured fragments to gain stability of the shoulder girdle.

Treatment of midshaft displaced clavicular fractures traditionally was nonoperatively. For almost all clavicle fractures, the best treatment option is the one that is used nowadays: to support the arm while achieving acceptable fragment alignment and to avoid complication. A satisfactory function and united fracture were the goals, despite some cosmetic deformity, shortening, and a lump. The perfect method for nonoperative treatment is still not clear. A simple sling and figure-of-eight bandage are most common. In a study by Andersen et al. [73], the functional and cosmetic results were identical, and the initial displacement of the fragments remains unchanged after the fracture has healed. Less discomfort and fewer complications were seen with a simple sling. In a randomized controlled trial Hoofwijk and van der Werken [74] found limited evidence that there is no difference in pain between the two methods after 2 weeks and 6 months.

However, not all the fractures healed, and in some cases clavicular nonunion occurred. There was increasing interest in identifying the types of fractures that might lead to potential nonunion. The current stance is that the lateral third fracture, a more lateral multifragmentation, and more than 15 mm displacement are more common in nonunion cases.

Previous opinion was that some operative management of fresh fracture increased the possibility of nonunion. Early operative management of certain clavicular fractures was taken into consideration, and internal fixation techniques were developed. Several operative treatments are used to stabilize clavicle fractures. Two of the most commonly used are intramedullary pin fixation and internal plate fixation. For plate fixation different types of plates are available: dynamic compression plates (DCP) and tubular or reconstruction plates. The Kirschner wires, Knowles pin, Rockwood pin, elastic stable intramedullary nailing (ESIN), and titanium elastic nailing (TEN) are vailable to performe a intramedullary fixation.

Plate fixation and intramedullary fixation both have advantages and disadvantages. According to recently published prospective randomized trials [75], functional results after operative treatment seem to be better than conservative for displaced clavicular fractures. Plate fixation provides more rigid fixation, allows earlier exercise and rehabilitation, and is technically easy to perform. Disadvantages of use of plate fixation include: implant failure (breakage of the implant), deep infections, implant prominence, poor cosmetics (hypertrophic scars), nonunion and refracture because of removal of the plate, symptomatic malunion, angulation, and refracture after plate removal. Minor complications are superficial wound infection and neurovascular problems (brachial plexus symptoms and regional pain syndromes) that seem to pass over time by reinnervation.

Compared to plate fixation, intramedullary fixation is technically more demanding; in approximately 50 % of the patients, open reduction was necessary to reduce the fracture. The main complications are migration and perforation of the device and brachial plexus injury (it is described only as iatrogenic).

Searching in literature, implant-related problems after plate fixation of clavicular fractures occur frequently. Infection rates have been reported from 5 to 22 %. Nonunion rates diverge from 3 to 13 %, and significant rates of implant-related problems with irritation or failures of the plate requiring plate debridement or removal/revision surgery are reported in almost every study, on average ranging from 9 to 64 %. A second operation with plate debridement or removal/revision surgery was required at best in one out of every ten patients treated, in some studies even up to one out of two patients. There is a relatively small risk of refracture after plate removal, between 1 and 5 %.

Wijdicks et al. point out in their systematic review that based on the overall low numbers of reported nonunion and symptomatic malunion, plate fixation is a safe treatment option for displaced clavicular fractures. In three studies, there is no difference in functional outcome and complications after plate fixation or intramedullary fixation for DMCF [76].

In general, the disadvantage of clavicular surgery is the need for implant removal and a second operation. The number of plate removal cases differs between studies from 0 up to 74 % [77].

Concerning two operating treatments, there is very limited evidence of postoperative pain, function after 1 year, the need for reoperation after initial treatment with locking plate or non-locking plate, and the difference in complications in treatment with pin or plate fracture fixation and moderate evidence that the method of osteosynthesis has no effect on the incidence of delayed union or nonunion.

When comparing operative versus nonoperative treatment according to literature, there is limited evidence that surgery has substantial effect in pain relief after 1–5 months and low effect at 6–7 months. Using function, the effect of surgery is better at 6 weeks, but after a 6-month follow-up, there is no major difference. Disability was greater in nonoperative treatment after 6 weeks, but after 6 months, no relevant difference was found. Both methods have similar risk of mild complication. There was moderate quality of evidence found that after nonoperative treatment, there was an increase in risk of delayed union and nonunion.

Virtanen et al. in their paper in 2012 [78] concluded that there is moderate-quality evidence that operative treatment of middle third clavicular fractures has slightly better functional results after short-term follow-up. The benefits of operative treatment after 6 months were very small. Patients treated nonoperatively also recovered after the same period with good functional results, pain relief, and union rates. Fracture union was better after surgery. Operative treatment should be considered for young, active patients who need to restore their previous level of activity as quick as possible.

The best method of treatment of fractures of the clavicle is still unclear. There is a need for randomized controlled studies comparing plate fixation, intramedullary nailing, and nonoperative treatment. In addition, there is a need for randomized controlled studies of lateral and medial clavicle fractures. The shape and type of plates need to be determined. There is still an open question on the impact of fracture union or nonunion on functional results.

Do we have to operate only on patients with symptomatic nonunion of the clavicle?

Moreover, the old question in the new manner is to operate or not to operate.

17.2.6 Humeral Head Fractures

Proximal humeral fractures are most common between the ages of 11 and 17 years, and 20 % of these injuries occur as a result of a traumatic event during athletic participation.

Because of the probable remodeling of the proximal humerus, many authors do not recommend closed reduction or surgery for proximal humerus fractures. However, older patients with less growth potential may need a closed reduction with significant displacement or angulation.

A stress fracture of the proximal humeral physis or osteochondritis is common in the athlete with an immature skeleton. Repetitive stress caused by torque during the acceleration phase of throwing may lead to tendinitis in adults and stress fractures in youths. Young athletes with stress fractures usually present with pain produced by throwing. There may be focal pain over the deltoid insertion and perhaps the general rotator cuff without any instability or impingement signs. The radiographic finding is a widening of the proximal humeral physis compared to the normal shoulder. Treatment should consist of possibly limited immobilization, ice, and physical therapy.

17.3 Elbow Injuries

The elbow is a hinge joint consisting of three articulations: the ulnohumeral, the radiocapitellar, and the proximal radioulnar joints. Except the bony anatomy, stability of the elbow is provided by soft tissue restraints, like the joint capsule, and surrounding muscles and ligaments. The two main ligamentous structures which are essential for elbow stability are the medial collateral ligament (MCL) complex and the lateral collateral ligament (LCL) complex.

The number of participants in overhead and throwing sports, like baseball, volleyball, tennis, javelin, and discus throw, has increased dramatically in the last years. Similarly, the number of elbow injuries related to these activities has increased also. The most common elbow injuries in athletes include (1) lateral epicondylitis (tennis elbow), (2) medial (ulnar) collateral ligament tears, (3) flexor pronator muscle injuries, (4) valgus extension overload syndrome, (5) ulnar neuritis, (6) medial epicondyle apophysitis, (7) olecranon stress fractures, and (8) osteochondritis dissecans.

During throwing motion the ligament restraints provide the majority of elbow stability. The combination of large valgus loads with elbow extension produces tensile stress along the medial compartment structures, shear forces in the posterior compartment, and compression forces on the lateral aspect of the elbow. The combination of these forces known as "valgus extension overload syndrome" produces the vast majority of injuries around the elbow in athletes. Repetitive valgus forces in sports like baseball, tennis, javelin, and discus throw result in microtrauma and inflammation to the MCL complex which may lead to ligament attenuation and failure, injuries to the flexor–pronator muscle, traction neuropathy of the ulnar nerve, and medial epicondyle apophysitis. Extension and compression forces in the posterior and lateral compartment, respectively, will produce osteophyte formation at the fossa or olecranon tip, loose bodies, and olecranon stress fractures. Shear stress from wrist extension at the extensor radialis brevis in sports such as tennis, racket sports, or archery is responsible for lateral epicondylitis.

A thorough history including type of sports, duration and onset of symptoms, and location and severity of pain is mandatory to guide further investigation. Active and passive range of motion of both elbows should be assessed. Pain in the medial side of the elbow can indicate MCL or flexor–pronator—flexor carpi radialis—injury. The ulnar nerve must be evaluated at the medial side of the elbow. Numbness or tingling in the hand or fingertips may be early signs of ulnar neuropathy. In the lateral part, tenderness just anterior and distal to the lateral epicondyle is indicative of lateral epicondylitis, while palpation of the radiocapitellar joint and LCL will reveal/exclude pathology of these structures. Posteriorly, olecranon tenderness and loss of full extension are indicative of valgus extension overload syndrome and osteophyte formation. Finally palpation of the distal biceps tendon anteriorly should be performed to evaluate its integrity. Specific tests for MCL (valgus stress test, milking maneuver) and pivot shift test for posterolateral instability are part of the physical examination.

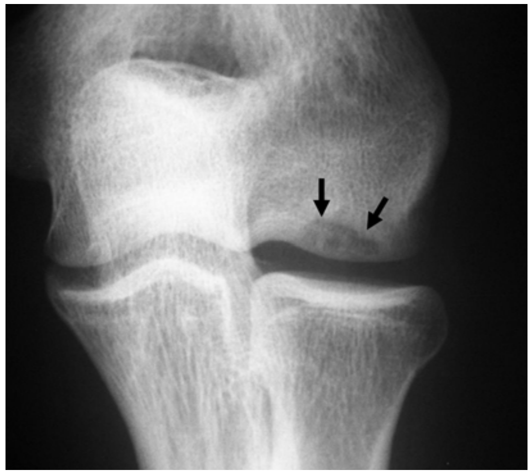

Fig. 17.11 Osteochondritis dissecans of the capitellum (*arrows*) in a 22-year-old male athlete

Plain radiographs providing an overview of the osseous structures and of course injuries to the soft tissues are not visualized. Standard views include AP and lateral projections and two oblique views if necessary. Stress views should be obtained if ligament disruption is suspected. Radiographs could reveal olecranon osteophytes, loose bodies, or osteochondritis dissecans of the capitellum (Fig. 17.11). Ultrasound is limited in its ability to evaluate the articular surfaces of the

Fig. 17.12 FS PD TSE coronal MR image demonstrating a partial tear of the MCL

elbow, but it may be used for the evaluation of elbow effusion or imaging of superficial muscle and tendon tears. Computed tomography provides excellent osseous detail and can be very helpful to determine stress fractures or osteochondral defects and loose bodies. Finally, MRI is the modality of choice to evaluate soft tissue structures, such as ligaments (Fig. 17.12), tendons and muscles, or intra-articular abnormalities (Fig. 17.13) such as chondral defects (Fig. 17.14).

Treatment of elbow injuries depends on the type and chronicity of the injury, level and age of the athlete, as well as imaging studies. Nonoperative treatment of ulnar collateral ligaments is generally indicated in non-throwing athletes. After pain and inflammation are controlled, a period of active rest (2–6 weeks) with functional exercises and strengthening of the shoulder and scapula stabilizers should be followed. Return to throwing is allowed when the athlete is free of pain. However, high-demand athletes do not respond well to nonoperative treatment, and operative treatment is warranted when a tear of the MCL is determined by history, clinical examination, and imaging studies.

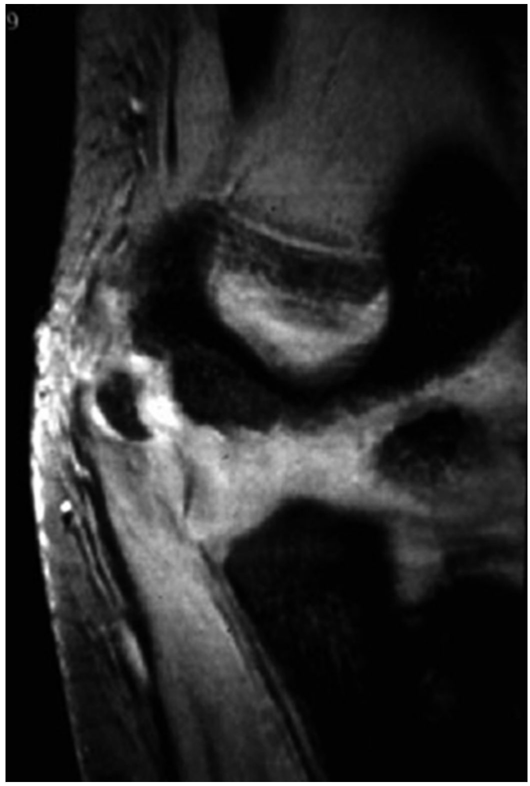

Fig. 17.13 FS PD TSE coronal MR image demonstrating avulsion fracture of the medial humeral epicondyle (little leaguer's elbow)

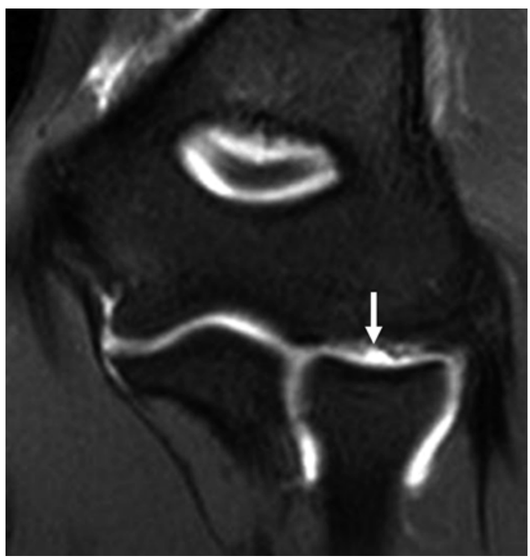

Fig. 17.14 FS T1-w SE coronal MR arthrography image demonstrating cartilage lesion of the capitellum

Reconstruction of the MCL is performed with the palmaris longus tendon as a graft which is then placed in the ulna and medial epicondyle through bone tunnels. Similarly, ulnar neuritis can be managed conservatively with anti-inflammatory medication and gradual return to throwing. If nonoperative treatment fails, surgical transposition of the ulnar nerve is indicated. Subcutaneous instead of submuscular transfer of the nerve is preferred since it provides better results. Flexor–pronator injuries generally respond well to conservative treatment, and gradual return to throwing is expected after 2–3 weeks. Valgus extension overload syndrome usually requires operative treatment especially when posteromedial osteophyte formation and loss of full extension are present. Arthroscopic osteophyte debridement with capsular release results in restoration of extension with excellent clinical results. Operative intervention and osteosynthesis with a 6.5 or 7.2 mm cannulated screw are the treatments of choice for olecranon stress fractures. Treatment of osteochondral lesions and osteochondritis dissecans of the capitellum is based on the stability of the osteochondral lesion. Operative treatment consists of arthroscopic debridement, abrasion chondroplasty, and mosaicplasty. In cases of failed nonoperative treatment of lateral epicondylitis, arthroscopic debridement of the anterolateral capsule and extensor carpi radialis brevis insertion are the preferred treatments in our days.

A thorough understanding of the elbow anatomy and biomechanics is essential to understand the spectrum of its pathology. The combination of large valgus loads, shear forces in the posterior compartment, and compression forces on the lateral aspect of the elbow is responsible for the underlying pathology in the throwing athlete. Operative treatment is indicated when conservative treatment fails. Elbow arthroscopic surgery has expanded its indications in the last years, and treatment of lateral epicondylitis, osteochondritis dissecans, valgus extension overload syndrome, and elbow contracture can be performed with minimal morbidity.

17.4 Triangular Fibrocartilage Complex Injuries

TFCC means triangular fibrocartilage complex. The TFC (triangular fibrocartilage) is an articular structure that lies over the distal ulna. The term "complex" indicates the relationship between the central disk and the ligaments that surround it. The central portion consists of chondroid fibrocartilage; the peripheral portion of the TFCC is well vascularized, while the central portion has no blood supply. There is a strong attachment to the base of the ulnar styloid. The radioulnar ligaments, palmar and dorsal, are the principal stabilizers of the distal. These ligaments arise from the distal radius sigmoid facet and insert at the ulna styloid and the fovea. Ulnocarpal ligaments prevent dorsal migration of the distal ulna.

The TFCC is important in load transmission across the ulnar aspect of the wrist and stabilization of the ulnar head. The ulnar variance influences the amount of load that is transmitted through the distal ulna. The load transmission is directly proportional to this ulnar variance. With positive ulnar variance the load is increased. This variance occurs in pronation.

Patients with a TFCC injury usually experience pain or discomfort located at the ulnar side of the wrist, often just above the ulnar styloid. However, there are also some patients who report diffuse pain throughout the entire wrist. Extension and ulnar deviation usually enhance the symptoms (Fig. 17.15).

The application of an extension–pronation force to an axial-load wrist, such as in a fall on an outstretched hand, causes most of the traumatic injuries of the TFCC. Perforations and defects in the TFCC are not all traumatic. There is an age-related correlation with lesions in the TFCC, but many of these defects are asymptomatic. These lesions commonly occur in patients with positive ulnar variance. Chronic and excessive loading through the ulnocarpal joint causes degenerative TFCC tears. These tears are a component of ulnar impaction syndrome. In cadaveric examinations, 30–70 % of the cases had TFCC perforations and chondromalacia of the ulnar head, lunate, and

Fig 17.15 Painful extension and ulnar deviation express a high probability of TFCC lesion

Table 17.1 Classification of TFCC tears

Type 1: Traumatic	
1a	Central disk perforation
1b	Peripheral ulnar-sided tear
1c	Distal TFCC disruption (ulnocarpal ligaments)
1d	Radial TFCC disruption
Type 2: Degenerative	
2a	TFCC wear
2b	TFCC with lunate or ulnar chondromalacia
2c	TFCC with lunate or ulnar chondromalacia + ulnotriquetral ligament perforation
2e	TFCC with lunate or ulnar chondromalacia + ulnotriquetral ligament perforation + arthritis

triquetrum. Palmer classification is the most recognized classification; it divides TFCC lesions into these two categories: traumatic and degenerative (Table 17.1).

MRI is, together with physical examination, a helpful diagnostic tool to assess the condition of the TFCC. Nevertheless the incidence of false-positive and false-negative MRI results is high. Arthroscopy is an invasive diagnostic tool, but still it remains to this day the most accurate way to identify TFCC lesions.

The initial treatment for both traumatic and degenerative TFCC lesions, with a stable DRUJ, is conservative. Patients may wear a temporary splint to immobilize the wrist and forearm for 4–6 weeks. Oral NSAIDs and corticosteroid joint injections can be prescribed for pain relief. TFCC surgery is indicated when conservative treatment fails, usually after 8–12 weeks.

The central part of the TFCC has no blood supply and therefore has no healing capacity. Removing the damaged tissue (debridement) is then indicated. Arthroscopic debridement is at the moment the treatment of choice. In case of degenerative scenarios, a wafer resection, shortening the most distal 4 mm of the ulnar head, is indicated.

Suturing TFCC ligaments can also be performed arthroscopically, either with simple capsular knots or, in case of a complete detachment from the fovea, with an anchor-based technique. This is at the moment the standard of care.

More complex techniques most of the time rely on open surgery including reconstruction of the ligament component with a free palmaris graft.

Return to sports: 64 % of high-level athletes return to the previous level (even racket sports) in the major series.

Conclusion

TFCC has two components: a central disk and a peripheral ligament structure.

Lesion diagnosis is mainly formulated with clinical assessment rather than imaging.

Arthroscopic treatment is, at the moment, the standard of care.

References

1. Economopoulos KJ, Brockmeier SF. Rotator cuff tears in overhead athletes. Clin Sports Med. 2012;31(4):675–92.
2. Hermans J, Luime JJ, Meuffels DE, Reijman M, Simel DL, Bierma-Zeinstra SM. Does this patient with shoulder pain have rotator cuff disease? The Rational Clinical Examination systematic review. JAMA. 2013;310(8):837–47.
3. Plate JF, Haubruck P, Walters J, et al. Rotator cuff injuries in professional and recreational athletes. J Surg Orthop Adv. 2013;22(2):134–42.
4. Snyder SJ, Karzel RP, Del Pizzo W, Ferkel RD, Friedman MJ. SLAP lesions of the shoulder. Arthroscopy. 1990;6(4):274–9.
5. Romeo AA, Cohen B, Carreira DS. Traumatic anterior shoulder instability. Orthop Clin North Am. 2001;32(3):399–409.

6. Franceschi F, Longo UG, Ruzzini L, Rizzello G, Maffulli N, Denaro V. Arthroscopic salvage of failed arthroscopic Bankart repair: a prospective study with a minimum follow-up of 4 years. Am J Sports Med. 2008;36(7):1330–6.
7. Longo UG, Huijsmans PE, Maffulli N, Denaro V, De Beer JF. Video analysis of the mechanisms of shoulder dislocation in four elite rugby players. J Orthop Sci. 2011;16(4):389–97.
8. Jakobsen BW, Johannsen HV, Suder P, Sojbjerg JO. Primary repair versus conservative treatment of first-time traumatic anterior dislocation of the shoulder: a randomized study with 10-year follow-up. Arthroscopy. 2007;23(2):118–23.
9. Itoi E, Hatakeyama Y, Kido T, et al. A new method of immobilization after traumatic anterior dislocation of the shoulder: a preliminary study. J Shoulder Elbow Surg. 2003;12(5):413–5.
10. Handoll HH, Almaiyah M, Rangan A. Surgical versus non-surgical treatment for acute anterior shoulder dislocation. Cochrane Database Syst Rev. 2004;(1):CD004325.
11. Handoll HH, Hanchard NC, Goodchild L, Feary J. Conservative management following closed reduction of traumatic anterior dislocation of the shoulder. Cochrane Database Syst Rev. 2006;(1):CD004962.
12. Porcellini G, Campi F, Pegreffi F, Castagna A, Paladini P. Predisposing factors for recurrent shoulder dislocation after arthroscopic treatment. J Bone Joint Surg Am. 2009;91(11):2537–42.
13. Vermeiren JHF, Casteleyn PP, Opdecam P. The rate of recurrence of traumatic anterior dislocation of the shoulder. A study of 154 cases and a review of the literature. Int Orthop. 1993;17(6):337–41.
14. Goss TP. Anterior glenohumeral instability. Orthopedics. 1988;11(1):87–95.
15. Hovelius L. Incidence of shoulder dislocation in Sweden. Clin Orthop Relat Res. 1982;166:127–31.
16. Hovelius L, Olofsson A, Sandstrom B, et al. Nonoperative treatment of primary anterior shoulder dislocation in patients forty years of age and younger. a prospective twenty-five-year follow-up. J Bone Joint Surg Am. 2008;90(5):945–52.
17. Kralinger FS, Golser K, Wischatta R, Wambacher M, Sperner G. Predicting recurrence after primary anterior shoulder dislocation. Am J Sports Med. 2002;30(1):116–20.
18. Boszotta H, Helperstorfer W. Arthroscopic transglenoid suture repair for initial anterior shoulder dislocation. Arthroscopy. 2000;16(5):462–70.
19. Bottoni CR, Wilckens J, DeBerardino TM, D'Alleyrand JC, Rooney RC, Harpstrite JK, Arciero RA. A prospective, randomized evaluation of arthroscopic stabilization versus nonoperative treatment in patients with acute, traumatic, first-time shoulder dislocations. Am J Sports Med. 2002;30(4):576–80.
20. Chetouani M, Ropars M, Marin F, Huten D, Duvauferrier R, Thomazeau H. Is MRI useful to assess labral reduction following acute anterior shoulder dislocation? Orthop Traumatol Surg Res. 2010;96(3):203–7.
21. DeBerardino TM, Arciero R, Taylor DC, Uhorchak JM. Prospective evaluation of arthroscopic stabilization of acute, initial anterior shoulder dislocations in young athletes. Two- to five-year follow-up. Am J Sports Med. 2001;29(5):586–92.
22. Edmonds G, Kirkley A, Birmingham TB, Fowler PJ. The effect of early arthroscopic stabilization compared to nonsurgical treatment on proprioception after primary traumatic anterior dislocation of the shoulder. Knee Surg Sports Traumatol Arthrosc. 2003;11(2):116–21.
23. Hart WJ, Kelly CP. Arthroscopic observation of capsulolabral reduction after shoulder dislocation. J Shoulder Elbow Surg. 2005;14(2):134–7.
24. Law BK, Yung PS, Ho EP, Chang JJ, Chan KM. The surgical outcome of immediate arthroscopic Bankart repair for first time anterior shoulder dislocation in young active patients. Knee Surg Sports Traumatol Arthrosc. 2008;16(2):188–93.
25. Kirkley AGS, Griffin S, Richards C, Miniaci A, Mohtadi N. Prospective randomized clinical trial comparing the effectiveness of immediate arthroscopic stabilization versus immobilization and rehabilitation in first traumatic anterior dislocations of the shoulder. Arthroscopy. 1999;15(5):507–14.
26. Robinson CM, Akhtar A, Mitchell M, Beavis C. Complex posterior fracture-dislocation of the zshoulder. Epidemiology, injury patterns, and results of operative treatment. J Bone Joint Surg Am. 2007;89(7):1454–66.
27. Robinson CM, Jenkins PJ, White TO, Ker A, Will E. Primary arthroscopic stabilization for a first-time anterior dislocation of the shoulder. A randomized, double-blind trial. J Bone Joint Surg Am. 2008;90(4):708–21.
28. 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 Joint Surg Am. 2006;88(11):2326–36.
29. Robinson CM, Kelly M, Wakefield AE. Redislocation of the shoulder during the first six weeks after a primary anterior dislocation: risk factors and results of treatment. J Bone Joint Surg Am. 2002;84-A(9):1552–9.
30. 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 Elbow Surg. 2003;12(6):529–34.
31. Salmon JM, Bell S. Arthroscopic stabilization of the shoulder for acute primary dislocations using a transglenoid suture technique. Arthroscopy. 1998;14(2):143–7.
32. Siegler J, Proust J, Marcheix PS, Charissoux JL, Mabit C, Arnaud JP. Is external rotation the correct immobilisation for acute shoulder dislocation? An MRI study. Orthop Traumatol Surg Res. 2010;96(4):329–33.

33. Valentin A, Winge S, Engström B. Early arthroscopic treatment of primary traumatic anterior shoulder dislocation. A follow-up study. Scand J Med Sci Sports. 1998;8(6):405–10.
34. Wintzell G, Haglund-Akerlind Y, Tidermark J, Wredmark T, Eriksson E. A prospective controlled randomized study of arthroscopic lavage in acute primary anterior dislocation of the shoulder: one-year follow-up. Knee Surg Sports Traumatol Arthrosc. 1996;4(1):43–7.
35. Wintzell G, Haglund-Akerlind Y, Ekelund A, Sandström B, Hovelius L, Larsson S. Arthroscopic lavage reduced the recurrence rate following primary anterior shoulder dislocation. A randomised multicentre study with 1-year follow-up. Knee Surg Sports Traumatol Arthrosc. 1999;7(3):192–6.
36. Wintzell G, Haglund-Akerlind Y, Nowak J, Larsson S. Arthroscopic lavage compared with nonoperative treatment for traumatic primary anterior shoulder dislocation: a 2-year follow-up of a prospective randomized study. J Shoulder Elbow Surg. 1999;8(5):399–402.
37. Larrain MV, Botto G, Montenegro HJ, Mauas DM. Arthroscopic repair of acute traumatic anterior shoulder dislocation in young athletes. Arthroscopy. 2001;17(4):373–7.
38. Longo UG, Loppini M, Rizzello G, Romeo G, Huijsmans PE, Denaro V. Glenoid and humeral head bone loss in traumatic anterior glenohumeral instability: a systematic review. Knee Surg Sports Traumatol Arthrosc. 2014;22(2):392–414.
39. Hovelius L, Augustini B, Fredin H, Johansson O, Norlin R, Thorling J. Primary anterior dislocation of the shoulder in young patients. A ten-year prospective study. J Bone Joint Surg Am. 1996;78(11):1677–84.
40. Liavaag S, Brox JI, Pripp AH, Enger M, Soldal LA, Svenningsen S. Immobilization in external rotation after primary shoulder dislocation did not reduce the risk of recurrence: a randomized controlled trial. J Bone Joint Surg Am. 2011;93(10):897–904.
41. Smith GC, Chesser TJ, Packham IN, Crowther MA. First time traumatic anterior shoulder dislocation: a review of current management. Injury. 2013;44(4):406–8.
42. Paterson WH, Throckmorton TW, Koester M, Azar FM, Kuhn JE. Position and duration of immobilization after primary anterior shoulder dislocation: a systematic review and meta-analysis of the literature. J Bone Joint Surg Am. 2010;92(18):2924–33.
43. Taskoparan H. Immobilization of the shoulder in external rotation for prevention of recurrence in acute anterior dislocation. Acta Orthop Traumatol Turc. 2010;44(4):278–84.
44. Itoi E, Hatakeyama Y, Sato T, et al. Immobilization in external rotation after shoulder dislocation reduces the risk of recurrence. A randomized controlled trial. J Bone Joint Surg Am. 2007;89(10):2124–31.
45. Finestone A, Milgrom C, Radeva-Petrova DR, Rath E, Barchilon V, Beyth S, Jaber S, Safran O. Bracing in external rotation for traumatic anterior dislocation of the shoulder. J Bone Joint Surg Br. 2009;91(7):918–21.
46. Arciero RA, St Pierre P. Acute shoulder dislocation. Clin Sports Med. 1995;14(4):937–53.
47. Chalidis B, Sachinis N, Dimitriou C, Papadopoulos P, Samoladas E, Pournaras J. Has the management of shoulder dislocation changed over time? Int Orthop. 2007;31(3):385–9.
48. Kirkley A, Werstine R, Ratjek A, Griffin S. Prospective randomized clinical trial comparing the effectiveness of immediate arthroscopic stabilization versus immobilization and rehabilitation in first traumatic anterior dislocations of the shoulder: long-term evaluation. Arthroscopy. 2005;21(1):55–63.
49. Larrain MV, Montenegro HJ, Mauas DM, Collazo CC, Pavon 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. Arthroscopy. 2006;22(12):1283–9.
50. Taylor DC, Arciero RA. Pathologic changes associated with shoulder dislocations. Arthroscopic and physical examination findings in first-time, traumatic anterior dislocations. Am J Sports Med. 1997;25(3):306–11.
51. Miller BS, Sonnabend DH, Hatrick C, et al. Should acute anterior dislocations of the shoulder be immobilized in external rotation? A cadaveric study. J Shoulder Elbow Surg. 2004;13(6):589–92.
52. Bollier MJ, Arciero R. Management of glenoid and humeral bone loss. Sports Med Arthrosc. 2010;18(3):140–8.
53. Provencher MT, Bhatia S, Ghodadra NS, et al. Recurrent shoulder instability: current concepts for evaluation and management of glenoid bone loss. J Bone Joint Surg Am. 2010;92 Suppl 2:133–51.
54. Piasecki DP, Verma NN, Romeo AA, Levine WN, Bach Jr BR, Provencher MT. Glenoid bone deficiency in recurrent anterior shoulder instability: diagnosis and management. J Am Acad Orthop Surg. 2009;17(8):482–93.
55. Lafosse L, Boyle S, Gutierrez-Aramberri M, Shah A, Meller R. Arthroscopic latarjet procedure. Orthop Clin North Am. 2010;41(3):393–405.
56. Weng PW, Shen HC, Lee HH, Wu SS, Lee CH. Open reconstruction of large bony glenoid erosion with allogeneic bone graft for recurrent anterior shoulder dislocation. Am J Sports Med. 2009;37(9):1792–7.
57. Taverna E, Golanò P, Pascale V, Battistella F. An arthroscopic bone graft procedure for treating anterior-inferior glenohumeral instability. Knee Surg Sports Traumatol Arthrosc. 2008;16(9):872–5.
58. Sugaya H, Moriishi J, Kanisawa I, Tsuchiya A. Arthroscopic osseous Bankart repair for chronic recurrent traumatic anterior glenohumeral instability. Surgical technique. J Bone Joint Surg Am. 2006;88(Suppl 1 Pt 2):159–69.
59. Salter Jr EG, Nasca RJ, Shelley BS. Anatomical observations on the acromioclavicular joint and

supporting ligaments. Am J Sports Med. 1987;15(3): 199–206.
60. Tauber M. Management of acute acromioclavicular joint dislocations: current concepts. Arch Orthop Trauma Surg. 2013;133(7):985–95.
61. 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.
62. Renfree KJ, Wright TW. Anatomy and biomechanics of the acromioclavicular and sternoclavicular joints. Clin Sports Med. 2003;22(2):219–37.
63. Bossart PJ, Joyce SM, Manaster BJ, Packer SM. Lack of efficacy of 'weighted' radiographs in diagnosing acute acromioclavicular separation. Ann Emerg Med. 1988;17(1):20–4.
64. Szalay EA, Rockwood Jr CA. Injuries of the shoulder and arm. Emerg Med Clin North Am. 1984;2(2): 279–94.
65. Reid D, Polson K, Johnson L. Acromioclavicular joint separations grades I-III: a review of the literature and development of best practice guidelines. Sports Med. 2012;42(8):681–96.
66. Schlegel TF, Burks RT, Marcus RL, Dunn HK. A prospective evaluation of untreated acute grade III acromioclavicular separations. Am J Sports Med. 2001; 29(6):699–703.
67. Sim E, Schwarz N, Hocker K, Berzlanovich A. Repair of complete acromioclavicular separations using the acromioclavicular-hook plate. Clin Orthop Relat Res. 1995;314:134–42.
68. Venjakob AJ, Salzmann GM, Gabel F, et al. Arthroscopically assisted 2-bundle anatomic reduction of acute acromioclavicular joint separations: 58-month findings. Am J Sports Med. 2013;41(3): 615–21.
69. Cote MP, Wojcik KE, Gomlinski G, Mazzocca AD. Rehabilitation of acromioclavicular joint separations: operative and nonoperative considerations. Clin Sports Med. 2010;29(2):213–28, vii.
70. Allman Jr FL. Fractures and ligamentous injuries of the clavicle and its articulation. J Bone Joint Surg Am. 1967;49(4):774–84.
71. Neer 2nd CS. Nonunion of the clavicle. J Am Med Assoc. 1960;172:1006–11.
72. Robinson CM, Court-Brown CM, McQueen MM, Wakefield AE. Estimating the risk of nonunion following nonoperative treatment of a clavicular fracture. J Bone Joint Surg Am. 2004;86-A(7): 1359–65.
73. Andersen K, Jensen PO, Lauritzen J. Treatment of clavicular fractures. Figure-of-eight bandage versus a simple sling. Acta Orthop Scand. 1987;58(1):71–4.
74. Hoofwijk AG, van der Werken C. Conservative treatment of clavicular fractures. Z Unfallchir Versicherungsmed Berufskr. 1988;81(3):151–6.
75. Zlowodzki M, Zelle BA, Cole PA, Jeray K, McKee MD. Treatment of acute midshaft clavicle fractures: systematic review of 2144 fractures: on behalf of the Evidence-Based Orthopaedic Trauma Working Group. J Orthop Trauma. 2005;19(7):504–7.
76. Ferran NA, Hodgson P, Vannet N, Williams R, Evans RO. Locked intramedullary fixation vs plating for displaced and shortened mid-shaft clavicle fractures: a randomized clinical trial. J Shoulder Elbow Surg. 2010;19(6):783–9.
77. Chen CE, Juhn RJ, Ko JY. Anterior-inferior plating of middle-third fractures of the clavicle. Arch Orthop Trauma Surg. 2010;130(4):507–11.
78. Virtanen KJ, Malmivaara AO, Remes VM, Paavola MP. Operative and nonoperative treatment of clavicle fractures in adults. Acta Orthop. 2012;83(1): 65–73.

Lower Limb

18

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S. Zaffagnini et al. (eds.), *ESSKA Instructional Course Lecture Book*,
DOI 10.1007/978-3-642-53983-1_18, © ESSKA 2014

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18.1 Introduction

Lower limb injuries are most common in athletes and involve fractures and ligament or tendon injuries. Running, jumping and throwing produce tremendous ground reactive forces that can create various musculoskeletal injuries and dysfunction.

In this chapter, we will discuss the different types of common lower limb injuries. In particular, we will focus on:

Hip/groin/thigh injuries:
- *Hip pathology*
- *Groin pain and muscle tears*
- *Symphysis pubis pathology*
- *Pelvic fractures*
- *Soft tissue pathology*
- *Pelvic complex syndrome*
- *Femoroacetabular impingement*
- *Hamstring muscle injuries*

Knee injuries:
- *ACL rupture*
- *MCL injury*
- *Osteochondral defects*

Ankle and foot injuries:
- *Ankle sprain*
- *Syndesmosis injury*
- *Talar dome chondral and osteochondral lesions*
- *Achilles tendon injuries*
- *Midfoot soft tissue injury*
- *First metatarsophalangeal joint injury*
- *Fifth metatarsal base fracture*
- *Distal metatarsal shaft fractures*
- *Plantar fasciitis*

18.2 Hip and Groin Pain

The athlete presenting with hip and groin pain is a challenge. The assessment of the hip region is complex. Presentation can be diverse for the same pathology and be similar for different pathologies. The history and exam are important especially as pathologies can coexist. This is certainly true of hip and lumbar spine disease and in >35 year age group. As one region fails, the athlete loses ability to compensate so symptoms and treatment may change over time. A hip joint disorder can therefore go undetected for several months before it is recognised. Often, injuries take time to heal which frustrates the patient and the doctor. It is difficult to rest the pelvis.

Identifying the pathological area within the pelvis is often a problem, and we should be aware of pain referred from sacroiliac joint or lumbar spine. Chronic pain raises possibility of systemic cause for loss of homeostasis and pelvic organ pathology. Care needs to be taken with image interpretation as existing or degenerative pathology may be asymptomatic. In the absence of trauma, the diagnostic possibilities are endless.

The patient does not know it is the hip and may describe groin or thigh pain. Acute injury particularly if associated with bruising usually radiating to inner thigh signifies muscle distraction tear (Fig. 18.1). Enquiry about pain associated with certain motion limits may point to relevant muscle groups such as internal or external hip rotators or short hip flexors but beware that muscles acting on pathological bone will be weak and painful. Immediate disability such as difficulty weight-bearing may point to a fracture or hip distraction. With chronic injury, there is variable disability and symptoms over time which may lead to different diagnoses. Sportspeople often present late and struggle through pain with impaired performance. Pain exacerbated by straining may point to potential hernia. The most vulnerable patients are those who participate in sports that involve excess hip motion. We should distinguish between active daily living and sport ability.

Acute groin injuries are more common in sports with acceleration, hip rotation and pivoting and include soccer, hockey, hurdling, skiing and dancing. Young athletes are susceptible to

Fig. 18.1 Adductor bruising

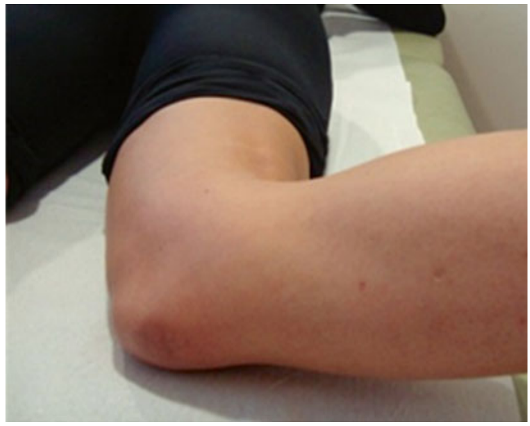

Fig. 18.2 Femoral anteversion

Fig. 18.3 Leunig–Ganz test

bone trauma and avulsions. Slipped capital femoral epiphysis should be suspected and excluded in 10–15 year age group when presenting with pain and disability. Older athletes tend to have muscle injuries and symphysis pubis stress.

Initial exam requires observation and ability to perform certain tasks. A significant deficit can be acknowledged without touching the patient and alerting the examiner to a gentle exam approach. The focus of pain may be an indicator such as the Byrd C-sign. Passive hip motion and presence of femoral anteversion should be assessed. Pain on log roll test is a reliable pointer to active hip joint disease. Leunig–Ganz test for potential cam femoroacetabular impingement [FAI] and reverse test for potential pincer FAI are always included (Figs. 18.2 and 18.3). Assess pelvic muscle actions in supine position and watch for contralateral signs. Tenderness of the symphysis pubis and those muscles inserting onto it may point to pathology. Superficial inguinal rings are checked for disruption and hernia. The lumbar spine and sacroiliac joint is part of the routine exam, and the genital region, abdomen and lower limb neurological exam may be indicated.

X-ray AP erect pelvis is the first-line investigation and can highlight degenerative joint disease. Note is made on the bone contours and alignment with documentation of cross-over, posterior wall and prominent iliac spine signs which are prognostic indicators for hip joint disease. Flamingo views for symphysis instability and modified Dunn view can further clarify joint pathology.

Discrete pathology can be missed on standard MRT sequences and often we require MRT arthrography with intra-articular local anaesthetic for potential hip joint pathology. SPECT-CT is now preferred for clarifying pain generator in the pelvis especially where we suspect several pathologies.

Fig. 18.4 XR femoral neck stress fracture

Fig. 18.5 XR femoral neck fracture

US scanning and dynamic mode are useful for enthesopathy and identifying bursae. In non-trauma patients, it is wise to check labs with general profiles and inflammatory markers. Neurophysiology studies help to define an associated radiculopathy. Lumbar spine and pelvic organ imaging may supplement our investigations.

18.2.1 Hip Pathology

18.2.1.1 Acute Hip Fractures

These occur in collision and high-velocity sports. Acetabular stress fractures present with chronic pain, increasing disability and positive hip impingement signs. A case of a soccer player with groin pain for 1 year is discussed and treated conservatively.

18.2.1.2 Femoral Neck Stress Fractures

These are important to diagnose and can have intrinsic, extrinsic and endocrine factors. Groin and thigh pain with disability and possible knee pain is a common presentation, and we have high suspicion index in runners. These are occult fractures and initial x-rays may appear normal, but look for discrete periosteal reaction on the inferior neck. Early diagnosis is paramount with careful treatment to prevent complications (Fig. 18.4). Significant symptoms and signs and stress fracture line through the neck confirmed on MRT in patients may be indicator for surgery (Figs. 18.5 and 18.6). Less disabled patients with observed weak pelvic muscle actions and more diffuse bone oedema on MRT can be treated conservatively (Fig. 18.7).

Fig. 18.6 MRT femoral neck fracture

Patients at risk should take oral mineral supplementation over 3 months with dietary advice on increasing calcium and protein intake. Patients

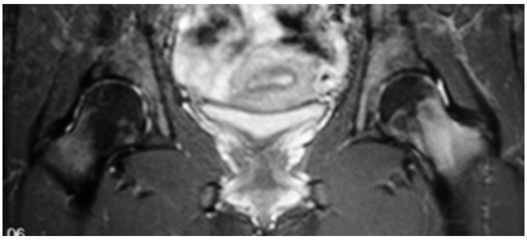

Fig. 18.7 MRT femoral neck oedema

Fig. 18.8 XR AP pelvis SCFE

are warned of those products which can impair bone healing.

18.2.1.3 Slipped Femoral Capital Epiphysis

There is a well-recognised aetiology with preponderance of 11–15 year age group, M–F 2:1, 95th centile for weight and African heritage. The presentation of groin/hip pain and disability in this age group should raise awareness. It is a cause of knee pain. XR and urgent orthopaedic referral for decision on operative care are mandatory in this group of patients (Fig. 18.8).

18.2.1.4 Acetabular Labral Tears

An isolated labral tear is rare. Fifty-five percent have associated articular damage and all may have FAI. Cam impingement is more common and associated with anterior superolateral labral tears. History is variable and patient may not complain of hip pain, but clinical signs point to the likely pathology and may be more reliable than imaging. Unhappy patients tend to have chondropathy. Arthroscopy for these lesions has around 80 % success, but 5 % can be made worse (Figs. 18.9 and 18.10).

Fig. 18.9 XR pelvis cam lesion

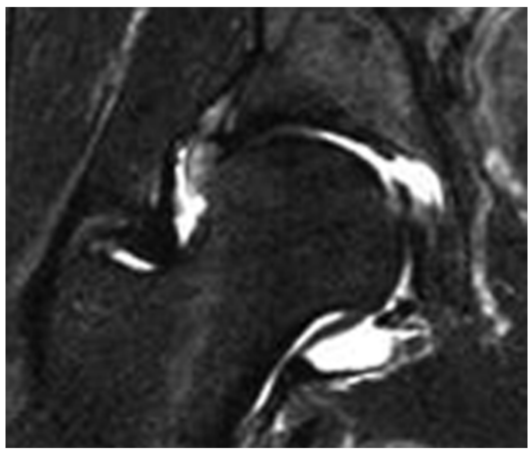

Fig. 18.10 MRTA hip labral tear

18.2.1.5 Ligamentum Teres Tear

The ligament is strong, bundled and intra-articular [cf ACL]. Injury may be more common than we realise (4–15 % incidence at arthroscopy). The ligament is an important hip stabiliser, and most injuries occur in forced abduction (Fig. 18.11).

18.2.1.6 Other Hip Pathology

Developmental hip dysplasia and degenerative hip joint disease present with chronic hip pain

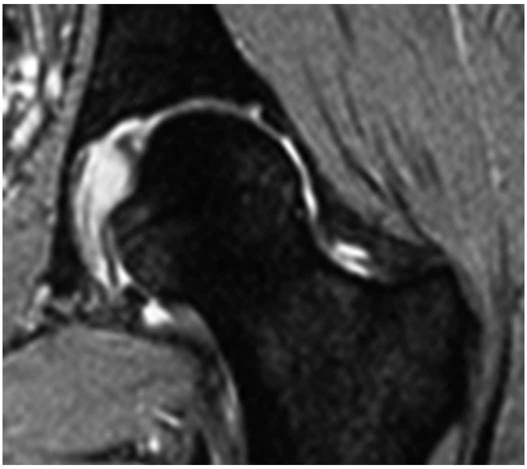

Fig. 18.11 MRTA hip–ligamentum teres tear

Fig. 18.12 XR developmental dysplasia

Fig. 18.13 OA hip

and increasing disability, and young athletes sometimes fall into these groups. X-ray AP erect should clarify the diagnosis (Figs. 18.12 and 18.13). Avascular necrosis presents with hip pain when symptomatic, and the history may point to diagnosis. Serious pathology such as tumours may be found when one is suspecting a more benign cause for hip pain.

Table 18.1 Adductor healing times

Weeks from injury	% add tears heal
<4	24 %
5–12	29 %
13–20	6 %
20+	41 %

18.2.2 Groin Pain and Muscle Tears

Most muscle tears are distraction injuries such as in soccer and rugby. Adductor strains are the most common and acute injuries easy to define. Obturator tears occur when there is hip rotation. Aetiology includes intrinsic factors and hip joint disease and may coexist with symphysis pubis stress. Most resolve with conservative care and active physical training, but warn patients that 24 % resolve <4 weeks and that 41 % may still be symptomatic >20 weeks (Table 18.1).

We can define individual muscle tears on MRT including adductors, obturators, rectus abdominis, pectineus, quadratus femoris and gemelli. Apophyseal injury to rectus femoris insertion at anterior inferior iliac spine normally presents acutely, and most of these injuries resolve with conservative care and settle (Figs. 18.14, 18.15 and 18.16).

18.2.3 Symphysis Pubis Pathology

Stress injuries occur in many sports with rotation and sustained impact such as soccer and running. Chronic groin pain is often associated with low abdominal and/or adductor pain with gradual deterioration in function and performance, leading to a complex of symptoms and signs. Is it a primary disorder or secondary to other pathology such as hip disease?

Four clinical types of ‘symphysis syndrome’ or ‘pubalgie du sportif’ are recognised:

1. Pubic osteoarthropathy – symphysitis
2. Inguinal canal pain – superficial inguinal ring and conjoint tendon
3. Rectus abdominis tendinopathy
4. Adductor tendinopathy

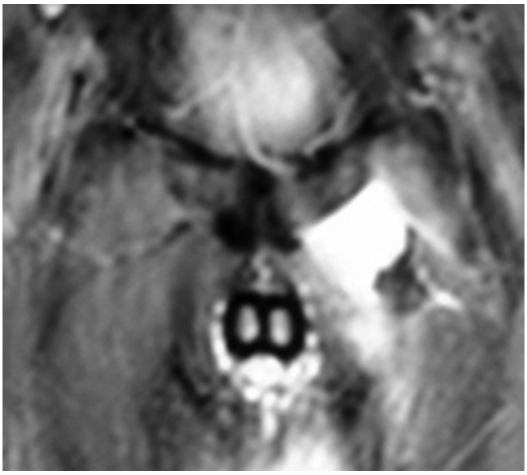

Fig. 18.14 MRT adductor tear

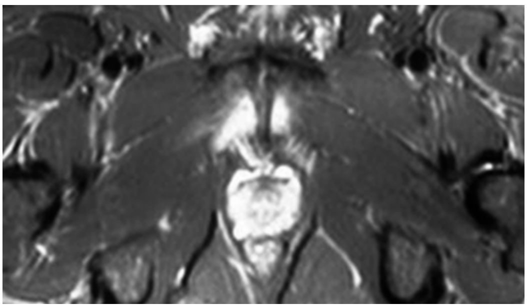

Fig. 18.15 MRT obturator tear

Frequently, there is recurrent pain in pubic zone that is sport related. We exclude multidisciplinary causes and aim for conservative treatment. Surgery may involve Nesovic-type procedure, insertion of mesh, laparoscopy and neurolysis of genitofemoral nerve. Whom to operate on and timing of surgery is debatable (Figs. 18.17, 18.18, 18.19, and 18.20).

18.2.4 Pelvic Fractures

Acute avulsion fractures are seen in adolescents, and stress fractures may occur from inappropriate training and cumulative exercise.

18.2.4.1 Inferior Pubic Ramus

This can present with acute groin pain with difficulty weight-bearing in activity but also in

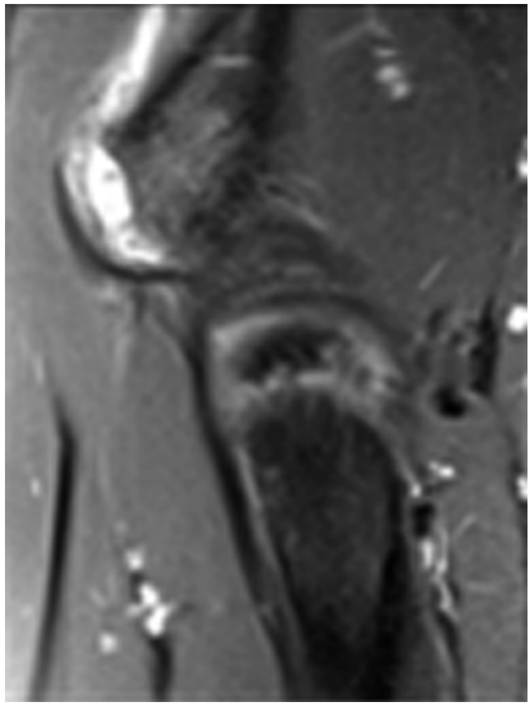

Fig. 18.16 MRT rectus femoris apophyseal tear

chronic case with gradual increasing groin pain and disability. They are common in slim female runners and dancers. Grade IV stress fractures are associated with pelvic muscle oedema and most can take several months to resolve (Fig. 18.21).

18.2.4.2 Superior Pubic Ramus

These stress fractures tend to present 3–4 months with pain, hip stiffness and increasing disability. Symphysis pubis and adductor cleft signs may be seen on MRT. Functional deficit is often present, and these injuries can take up to 1 year to resolve depending on sport level and participation (Fig. 18.22).

18.2.5 Soft Tissue Pathology

Bursitis is diagnosed on exclusion of more serious pathology. There may be history of contusion, and most bursal cysts are amenable to aspiration under US guidance. Bursal distension

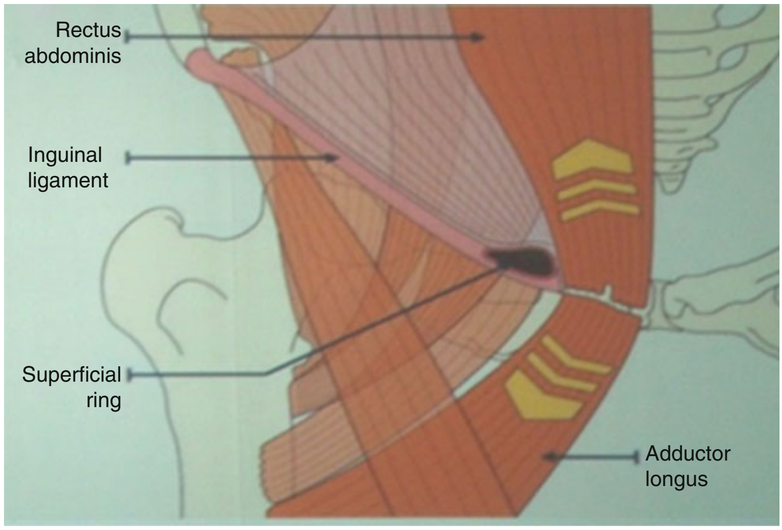

Fig. 18.17 Inguinal canal region

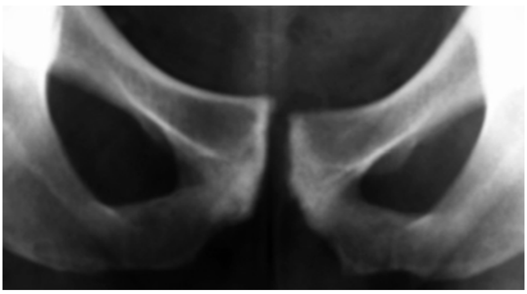

Fig. 18.18 XR symphysis pubis instability

Fig. 18.19 XR symphysis osteoarthropathy

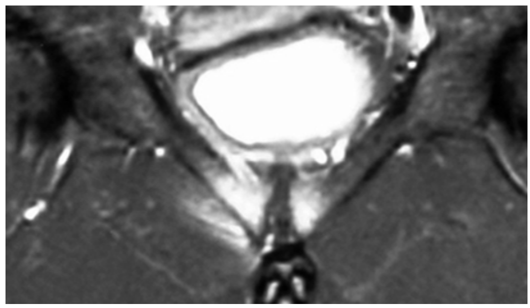

Fig. 18.20 MRT pelvis bone stress

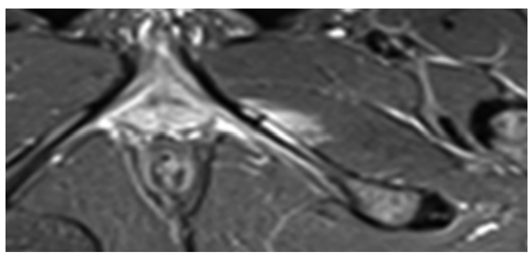

Fig. 18.21 MRT inferior pubic ramus fracture

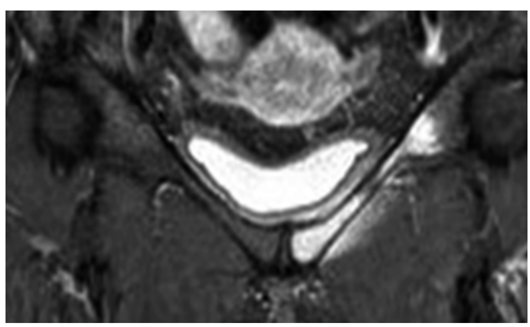

Fig. 18.22 MRT superior pubic ramus fracture

along the iliopectineal line is usually more symptomatic and impairs hip flexion (Fig. 18.23).

18.2.6 Pelvic Complex Syndrome

Athletes presenting with groin/hip pain may have a variety of pathologies, often in combination. We

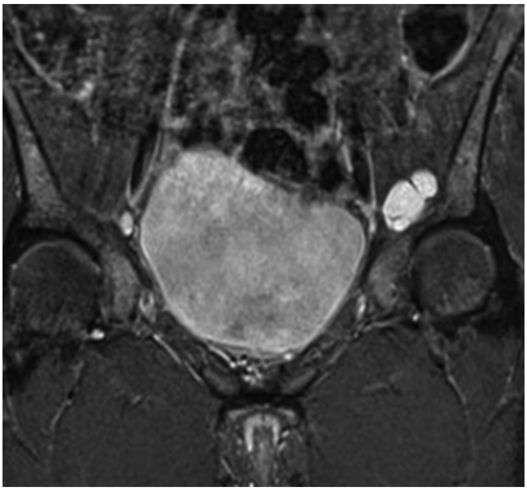

Fig. 18.23 MRT iliopsoas bursitis

Fig. 18.24 XR pelvic complex syndrome. *Arrows* show the combination of a variety of pathologies associated with groin/hip pain. The *circle* indicates the symphysis pubis

have learned about hip impingement over the last decade, but does primary hip pathology, with loss of motion and function, lead to other symptoms? We should use the term 'pelvic complex syndrome.'

Acute injuries are succinct but beware of the '3-month' presentation of symptoms. There should always be a high suspicion of stress fracture and occasional serious pathology. There is no such thing as a 'groin strain' (Fig. 18.24).

18.2.7 Femoroacetabular Impingement

Femoroacetabular impingement (FAI) has been described initially in the 1990s. FAI was extensively evaluated and demonstrated that it is caused by recurring compression of a morphologically abnormal proximal femur and/or acetabulum during terminal range of motion of the hip. Depending on the osseous abnormality, FAI results in typical damage to the labrum and acetabular cartilage. The osseous abnormality may be due to loss of the normal femoral head–neck offset resulting in cam impingement or may be due to loss of acetabular coverage resulting in pincer impingement. The third type is referred to as mixed or combined impingement.

Most FAI patients are young, athletically active and usually present with unilateral symptoms. Anterior groin or hip pain most often accompanies limitation of hip flexion and internal rotation. The pain may be described in the greater trochanter area and lateral thigh or may be referred to the knee. Plain x-rays are used to diagnose abnormalities with FAI and assess the hips to rule out osteoarthritis, avascular necrosis and other articular conditions. A true AP pelvic view and a cross-table lateral view of the proximal femur and hip x-ray in 45° of hip flexion are also used. MR imaging is essential in the assessment of the labrum and articular cartilage, especially when performed with gadolinium injection. Femoral torsion can be estimated from axial computed tomography (CT) or axial MRI images performed through the proximal and distal femur.

Conservative management provides limited relief. Conservative treatment includes activity modification, core muscle strengthening, anti-inflammatory medication and intra-articular local anaesthetic and corticosteroid injections.

FAI was initially addressed by open dislocation technique with varying success. Mini-open method with concomitant arthroscopic surgery was popularised to resolve the morbidities associated with the open technique. Arthroscopic management of the FAI has proven to be effective in elimination of the symptoms. The technique addresses the impingement by removing the morphologic abnormalities involving the acetabulum and/or the femoral head–neck junction. Tears of the labrum may either be repaired or debrided if there is extensive damage. Articular cartilage lesions on both sides may be addressed by debridement and microfracture. Earlier intervention before the occurrence of irreversible joint damage results in optimum long-term outcome.

Complications of surgery for correction of FAI include minor ectopic calcification, non-union of the greater trochanteric osteotomy, fracture, nerve damage, adhesions, avascular necrosis and persisting pain [1–3].

18.2.8 Hamstring Muscle Injuries

The diagnosis of a hamstring injury is rather straightforward and mainly based on history taking and physical examination. The key requirements in diagnosing an acute hamstring injury are (1) a history of acute onset of posterior thigh pain, (2) localised pain on manual palpation, (3) increased pain on muscle contraction and (4) pain on muscle stretch [4]. The initial clinical evaluation is preferably performed within 2–5 days after the injury, as symptoms may rapidly decline over time.

Complementary imaging with ultrasound or MRI is usually not required for the diagnosis. There is an exception in case of a suspected complete rupture (grade III), as these severe injuries have a different management strategy, including possible indication for a surgical treatment. Nonetheless, ultrasound and MRI are increasingly used to confirm the diagnosis and provide a prognosis on the recovery time, especially in the elite athlete [5].

There are several clinical and imaging measures suggested as indicators for the recovery time, but the large variation from days up to months challenges estimating a prognosis.

There is only limited evidence for the prognostic value of measures from clinical evaluation. Factors shown to be associated with an increased recovery time are the inability to walk pain free >1 day after injury [6] and proximity of the maximal pain on palpation to the ischial tuberosity [4]. Conflicting evidence is reported for deficits in hamstring flexibility and strength as a prognostic factor.

A substantial amount of studies have been performed on the prognostic value of imaging, especially MRI. The key MRI feature is the presence of intramuscular increased signal on fluid sensitive sequences (often referred as 'oedema').

Table 18.2 MRI grading

Injury grade	MRI features	Prognosis
Grade 0	Clinical diagnosis, no MRI abnormalities	Days–weeks
Grade I	Oedema, no fibre rupture visible	Days–months
Grade II	Partial rupture	
Grade III	Complete muscle rupture	Months–years

A MRI classification commonly used in clinical practice discriminates four grades of injury (Table 18.2) [7]. As grades I and II injuries in this classification do not differ in prognosis and management, a clinically relevant classification is limited to three levels of injury severity: (1) no MRI abnormalities, (2) increased signal without total rupture and (3) total rupture.

The strongest prognostic factor is the absence of increased signal, often denominated as MRI negative or grade 0 injuries, which is associated with a reduced injury time. On the other end of the spectrum are the less common total muscle ruptures, which can take up to years to recover and may result in lasting functional impairment. Another MRI feature associated with a prolonged recovery is the involvement of tendinous structures [4, 8]. Conflicting evidence is reported for the extent of the increased signal as a prognostic factor.

We would emphasise that an accurate prediction of the recovery time in the individual athlete cannot be established with the current scientific knowledge. We therefore suggest refraining from such a prediction, as it gives rise to unrealistic expectations.

Treatment of acute muscle injuries is mainly an expert practice, as it has a very limited research base. In the times ahead, the current practice will probably be challenged and changed, as on-going and future research will provide necessary evidence for a better understanding of the treatment of hamstring injuries.

There is general consensus among experts that a thorough progressive rehabilitation program is the basis of the treatment of acute hamstring injuries, in which progression should be based on functional criteria rather than based on a specific time frame.

The best available evidence indicates that the rehabilitation should focus on trunk stabilisation exercises, lengthening exercises, eccentric training, agility exercises and sport-specific training [9–11]. We recommend to guide progression through rehabilitation by both symptoms (pain free) and functional performance criteria.

In recent years, numerous medical treatment modalities have been suggested, including NSAIDs, losartan, hyperbaric oxygen therapy and injections with platelet-rich plasma, corticosteroids, Actovegin and Traumeel. Given the limited research base and the lack of evidence on their efficacy in muscle injuries, we cannot recommend the use of these medical treatment modalities and believe that it should remain restricted to experimental settings [12].

Indication for surgical intervention is limited to total hamstring ruptures. Besides injury-related factors, such as the number of muscles/tendons ruptured and the amount of muscle retraction, patient and sport-related factors, such as the level of sports and the desired activity level, should be considered in the decision for surgical intervention.

18.3 Knee Injuries

18.3.1 ACL Rupture

18.3.1.1 Immediate Versus Delayed Reconstruction

There has been considerable debate regarding both the earliest time for anterior cruciate ligament (ACL) reconstruction performed with safety and the time that surgery can be delayed without increasing the risk of developing secondary knee lesions.

When ACL reconstruction is performed within the first weeks after injury, knee effusion, ROM deficits and rehabilitation protocols more strongly affect the clinical outcomes. Mayr et al. [13] retrospectively reviewed a large cohort of 156 ACL-reconstructed patients with postoperative arthrofibrosis. They found that knee irritation, effusion and swelling following the acute injury significantly correlated with the development of arthrofibrosis. Similarly, Cosgarea et al. [14] suggested that arthrofibrosis was significantly more likely in patients with a preoperative motion deficit of 10° or greater compared to those without.

Accumulated evidence from the existing body of literature suggests a significant relationship between the duration of ACL deficiency and the incidence of secondary chondral and medial meniscal injuries. Many studies have shown that the incidence of medial meniscal injuries and the number and grade of cartilage lesions increase with time after ACL rupture [15–18]. The medial femoral condyle is the most commonly affected.

Although prospective comparisons are lacking, most authors suggest that to avoid the risk of additional damage, ACL reconstruction should preferably be performed within 6 months from injury. In a national survey among UK orthopaedic surgeons in the early 2000s [19], 81 % advocated that surgery should ideally be performed between 1 and 6 months post injury. However, it was acknowledged that only 35 % of ACL reconstructions are performed within this time frame in National Health Service (NHS) hospitals.

Additional concerns have been raised in the paediatric and adolescent population where the risk of damaging open physes should be weighed against that of secondary meniscal and chondral injury. It is clear that establishing the optimal timing for ACL reconstruction is particularly important for both the treating surgeons and the patients.

18.3.1.2 Single- or Double-Bundle ACL Reconstruction

Conventional ACL reconstruction is carried out with a single-bundle method, in which only the anteromedial bundle is reconstructed. It has been reported in the literature that there is still residual rotational laxity after the operation. Moreover, longer follow-up studies have shown that the conventional method does not prevent osteoarthritis of the knee (OA), partly because the OA develops as a result of the primary trauma [20]. The double-bundle technique with anteromedial and posterolateral bundle reconstruction was developed to overcome these disadvantages.

Table 18.3 Level I randomised controlled studies on single-bundle versus double-bundle ACL reconstruction

Study	Year	Patients	Follow-up (months)	Results
Adachi et al. [30]	2004	108	33	SB had more notchplasties than DB group
Aglietti et al. [21]	2010	70	24	DB had better VAS, anterior stability and final objective IKDC score
Hussein et al. [22]	2012	281	51	DB had the best anterior and rotational stability, the DB group also had better IKDC and Lysholm scores
Ibrahim et al. [23]	2009	200	29	DB had the best anterior and rotational stability
Jarvela [25]	2007	65	14	DB had better rotational stability
Jarvela et al. [24]	2008	77	24	DB had better rotational stability than either of the SB procedures
Muneta et al. [26]	2007	68	24	DB had better anterior and rotational stability
Sastre et al. [31]	2010	40	24	No difference
Siebold et al. [27]	2008	70	19	DB had better anterior and rotational stability and better objective IKDC score
Streich et al. [32]	2008	49	24	No difference
Suomalainen et al. [33]	2011	152	24	DB had fewer revisions
Suomalainen et al. [34]	2012	90	60	DB had fewer revisions
Zaffagnini et al. [28]	2008	72	36	DB had better anterior stability and better subjective, objective and functional evaluations
Zaffagnini et al. [29]	2011	79	96	DB had better rotational stability, ROM and functional scores, fewer degenerative changes and fewer reoperations

So far, 14 level I prospective randomised studies have been presented on single-bundle versus double-bundle ACL reconstructions. They report rather short-term (1–8 years) results comparing these two reconstruction methods (Table 18.3).

The main finding in some of these studies is the superior stability in the double-bundle group especially in the rotational plane [21–29], but other studies have not confirmed this conclusion [30–32].

Another important finding in favour for the double-bundle group is the better graft durability. There were seven graft failures in the SB Group (9 %) and only one in the DB Group (1 %) leading to ACL revision surgery during the 2-year follow-up ($p=0.04$). This was still significant after 5 years follow-up [33, 34].

18.3.1.3 Hamstrings or Patellar Tendon in Sport Population

There are 16 RCTs comparing hamstring grafts with BTB grafts. Seven studies (12–120 months of follow-up) reported no statistical differences between these two graft materials in any of the measurements [35–41].

Eight studies (4–96 months of follow-up) reported more donor site morbidity in the BTB graft group [42–49], and one, with follow-up of 132 months, reported more OA in the BTB group [50]. On the other hand, Aune et al. [42] and Maletis et al. [48] found that their hamstring tendon group had more hamstring muscle weakness. Complications using the BTB include patellar fractures, quadriceps weakness and patellar tendon inflammation or rupture [51].

18.3.2 MCL Injury

MCL is the most commonly injured ligament about the knee. Isolated injuries often exist, but MCL tears can also be associated with other ligamentous or intra-articular pathology [52]. The Fetto and Marshall [53] classification divides medial side knee injuries in grade I (no valgus laxity), grade II (valgus laxity at 30° of flexion) and grade III (valgus laxity at 0° and 30°). According to the amount of medial joint opening, the American Medical Association classification divides the grade II injuries according to Fetto and Marshall in (1) type I sprains (1–4 mm), (2) type II (5–9 mm) and (3) type III (10–15 mm). Valgus laxity in full extension is highly indicative of other ligamentous lesions associated with MCL injury (mostly the posteromedial corner and the ACL). The chronicity of the lesion is another essential factor to consider in MCL laxity. MCL tears can be classified in acute (<3 weeks), subacute (3–6 weeks) and chronic (>6 weeks).

In general, grades I and II tears of the MCL are managed conservatively with good results [52]. Management of grade III MCL injuries is much more controversial. Early authors advocated acute primary repair of complete MCL tears [54–56]. Recent literature, however, has focused more on nonoperative treatment of grade III MCL injuries [53, 57–61].

In the acute setting, surgical treatment is indicated in some grade III injuries, according to the surgeon preference, when there are large bony avulsions or in case of MCL entrapment. The treatment is controversial also in case of ACL and grade III MCL injuries and the options include (1) conservative treatment of MCL and delayed ACL reconstruction, (2) early ACL reconstruction followed by conservative MCL treatment and (3) acute MCL repair combined with ACL reconstruction. Different options can be considered also in case of ACL, PCL and grade III MCL injuries, and these include (1) conservative MCL treatment and delayed ACL/PCL reconstruction; (2) acute MCL repair and PCL reconstruction with delayed ACL reconstruction and (3) acute combined MCL repair, PCL and ACL reconstruction.

In case of chronic medial laxity, MCL reconstruction or capsular procedures (retensioning or en masse elevation) with or without auto- or allograft augmentation are indicated in case of neutral or varus alignment. In case of valgus alignment, a distal femoral varus osteotomy should be considered.

Grade I MCL injuries. Weight-bearing as tolerated with crutches if necessary. Active ROM exercises as soon as tolerated. Active strengthening exercises as tolerated. Progress to agility, proprioceptive and sport-specific drills as tolerated. Return to sports when strength, agility and proprioception are equal to the uninvolved side [62].

Grade II MCL injuries. Long leg brace for ambulation with weight-bearing as tolerated with crutches. Brace may be locked in extension for 1–2 weeks depending upon pain, valgus opening and anatomic alignment. Crutches can be discontinued when patient attains a nonantalgic gait. Active ROM exercises are started immediately. Electrical stimulation to the quadriceps, quad sets and single leg raise are initiated immediately. Long leg brace is opened at the end of the third week and full weight-bearing is encouraged. Severe grade II injuries may require 6 weeks of bracing. Once full ROM and functional strength are attained, proprioceptive and agility drills can be initiated [62].

Grade III MCL injuries. Immobilisation in long leg brace locked in extension for 3–6 weeks depending on anatomic alignment. Non-weight-bearing for 3 weeks in patients with valgus alignment. Toe-touch weight-bearing for neutral or varus. Immediate ROM out of the brace 2–3 times a day for neutral or varus alignment and after 3 weeks for patients with valgus. Strengthening is done throughout the 6-week period in the form of quad sets, single leg raise and electrical stimulation. Closed chain exercises are initiated at the appropriate time depending upon the patient's weight-bearing status [62].

Platelet-rich plasma (PRP) has been proposed as a treatment method to assist regenerative healing processes in acute ligament injuries. In animal studies, there is some evidence that PRP improves healing and mechanical strength in the early stages of acute MCL injury [63–68]. However, a marked drop in the effectiveness of platelet-derived growth factor was observed when administered more than 24 h after injury [65]. However, no prospective studies on PRP application in MCL lesions in humans are available [69].

In the acute setting, MCL repair is the treatment of choice with the correct indications (see above). The injured structures are identified and should be repaired from the deepest structure outward. A peripheral tear of the medial meniscus is commonly seen (33 %) and repaired with an open technique [70]. The deep MCL can be directly repaired using sutures alone or suture anchors. If injured, the posterior oblique ligament (POL) is repaired by direct suture back to the femur. Repair of the deep structures is completed with the knee held in varus and full extension. Femoral avulsion of the sMCL leaves the best tissue for repair using suture anchors, staples or a screw/washer. However, repair in this location is associated with postoperative stiffness more than in other locations. Acute complete avulsions off of the tibia can be repaired using either suture anchors or staples. The semimembranosus portion of the POL can be repaired with interrupted absorbable sutures and sutured to the posterior border of the MCL. Occasionally, midsubstance and tibial-sided injuries require augmentation due to the poor soft tissue quality. The sMCL is fixed at 30° of knee flexion [70].

In the subacute setting, capsular procedures can be considered. The goal of medial structure retensioning is to remove the laxity from the injured posteromedial structures by creating increased distance between the origin and insertion. The lax structures are attached to an adjacent intact structure in a pants-over-vest fashion. Otherwise, an en masse elevation can be performed. This procedure is indicated when a generalised laxity of the posteromedial structures is present. The structures must be released as an entire tendon/ligament unit (en masse). This unit must be armed with sutures, retensioned and fixed back to an isometric point on the bone with staples or suture anchors [71].

In the chronic setting with varus or neutral alignment, MCL reconstruction is indicated. Many techniques have been described [72–78]. In some of them, only the MCL is reconstructed, while others entail both MCL and POL reconstruction. Most techniques describe the use of hamstring autograft, but in multiligament knees allografts might be the best graft option. No reconstruction technique has shown superior results over the others. However, independently on the chosen technique, isometricity is crucial in obtaining good results and in reducing the risk of postoperative stiffness [71]. One of the most commonly used MCL reconstruction procedure is Kim's technique [72]. The semitendinosus is harvested, preserving the tibial attachment. A K wire is inserted on isometric point of the distal femur. The semitendinosus tendon is looped around the wire, and isometricity (<2 mm migration) is tested by pulling the suture at the tendon and moving the knee through a full ROM. A 6.5 mm cancellous screw with washer is used for proximal fixation, with the knee in 30° of flexion and varus stress. The free end of the graft is pulled under direct head of the semimembranosus tendon and sutured to the tendon itself, in 30° of knee flexion.

18.3.3 Osteochondral Defects

Treatment of knee cartilage defect, including articular cartilage defect (ACD) and osteochondral defect (OCD), has always been and is still a difficult challenge. The different methods of repair have been a concern in recent centuries. In 1743, Hunter [79] recognised that no technical method is sufficient to ensure the return of normal cartilage. In 1853, J. Paget made the same observation for joint fractures where consolidation cartilage was impossible. In 1997, faced with the difficulty and specificity of cartilage repair was created the ICRS (International Cartilage Repair Society, www.icrs.org). More recently, in 2001, some authors studied the natural history over a 30-year period and the evolution of osteochondritis dissecans of the femoral condyles. For the 20 cases occurred in patients compared to baseline (mean age 17 years) and still active in largest follow-up (mean age 49 years), he found osteoarthritis evolution in 60 % of cases regardless of the treatment achieved. When the lesion was greater than 2 cm^2, the percentage of osteoarthritis was then 78 %.

The natural history of cartilage lesions is marked by painful episodes of hydarthrodial with blockages in the short- and medium-term to long-term osteoarthritis. The International Cartilage Repair Society (ICRS) cartilage injury grading system was selected to classify articular cartilage defects. In the preop evaluation, MRI is very relevant. The MRI scans were used with the following protocols: sagittal oblique; coronal and axial PD TSE fs (proton density turbo spin-echo with fat saturation) sequences, with a repetition time (TR) and an echo time (TE); a sagittal oblique PD TSE (proton density turbo spin-echo without fat saturation) sequence; coronal and axial T1 SE (T1-weighted spin-echo) sequences; and a slice thickness of 3 mm. The magnetic resonance observation of cartilage repair tissue (MOCART) score system was used to grade articular cartilage repair quality (degree of defect repair and filling, integration to border zone, surface of the repair tissue, structure of the repair tissue, subchondral lamina, subchondral bone, adhesions, effusions).

The activity level of ICRS is interesting to evaluate the return to sport participation after articular cartilage repair in the knee: level I (high competitive athlete), level II (well-trained and frequently sporting at least 6 times per week), level III (sport recreational athlete) and level IV (non-sporting).

It is essential regarding cartilage damage; using the arthroscopic classification of the ICRS allows a descriptive analysis in four grades. It also specifies the size of the lesion and location (unilateral or bifocal) and should be left to the preoperative evaluation of the lesion. The ICRS grade I is where the fibrillation surface or surface with or without chondromalacia cracks. The ICRS grade II is where the cartilage is abnormal with cracks less than 50 % of the thickness of the cartilage. Lesions reach more than 50 % of the thickness of the cartilage in the ICRS grade III and sometimes reach the subchondral bone. In the ICRS grade IV, lesions reach the subchondral bone with involvement of the surface plate and the alteration of bone surface. Only grades III and IV with painful symptomatic knees are relevant for surgical treatment. Therefore, osteoarthritis lesions and 'mirror' lesions were excluded from the surgical options.

What is the incidence of chondral lesions of the knee with an area greater than 2 cm^2? This was achieved through the study of a large number of arthroscopies performed. From 993 consecutive arthroscopies, Aroen et al. [80] found a cartilage lesion in 66 % of cases. The lesion is localised in 20 % of cases, and only 11 % of patients have localised ICRS grade III and IV defect. However, this localised lesion ICRS grades III and IV has a size greater than 2 cm^2 in only 6 %.

The goal of treatment is to remove these painful crises and to delay the degenerative arthritic changes with long-term follow-up. The ability of articular cartilage repair to return the injured athlete to demanding sport participation is also a key factor regarding the success of these surgical procedures. We must distinguish regeneration techniques or seek to replace the cartilage with an identical tissue repair techniques where the cartilage is replaced by a non-strictly identical to the original cartilage tissue (rich in PG and collagen type II).

In our therapeutic arsenal, we have at our disposal four techniques: microfracture (MF), cartilage autografts (allografts massive cartilage), autologous chondrocyte cultures (ACI) and osteochondral autograft transfer (OAT).

The purpose of this chapter is to provide clinical results of the four methods and then answer the question 'is it realistic to return to sports?'

18.3.3.1 Microfracture

The principle is based on the stimulation of stem cells from the bone marrow then with the formation of a fibrin cruoric clot, which is gradually colonised by the potential of bone marrow cells. This method advocated by Steadman et al. [81] is the equivalent or close to old techniques performed in orthopaedics: Pridie's perforations and the abrasive chondroplasty [82]. This technique is performed arthroscopically after preparation of the lesion where the cartilage fragments were excised. After removal of the cartilage debris, once a clean bed is created within the lesion site and a good supporting peripheral rim of cartilage exists, small holes are created through the subchondral plate to allow the extravasation of marrow stem cells. Angled awls were used to make holes through the subchondral bone plate approximately 3–4 mm apart. Marrow elements accessed by the subchondral bone could be seen as they essentially came from all microfracture holes after the inflow was stopped. It forms a fibrocartilage (type I collagen) repair in 6–8 weeks with a composition and different from those of normal cartilage strength.

Steadman et al. [81] reviewed 72 patients with an average follow-up of 11 years. Lysholm scores were 59 preoperatively and 89 postoperatively. Tegner scores also showed improvement with a preoperative score of 3.1 and a postoperative score of 5.8. The preoperative pain score (3.4) improved as well after surgery to 1.9. Steadman found 75 % good or very good results but with a deterioration of the quality of the results after the second or third year. As previously mentioned, younger patients overall did better than older patients in this study. All authors recommend this technique only for small (less than 2 cm^2) and uni-focal lesions, among patients with few or no sports. The main prognostic factor is initial age at surgery of less than 35 years. However, this is one simple inexpensive technique, which is useful in several circumstances but do not especially cut the bridges for the realisation of other techniques later in case of failure.

Gudas et al. [83] in his comparative prospective study between MF and OAT evaluates the outcomes of the procedures in young active athletes, with activity level II and I and the mean age was 25 years (range, 15–45). Statistically, significantly better results were detected in patients in the OAT group compared with those in the MF group at 10 years ($p < .005$). At 10-year follow-up, there were 15 failures (26 %), including 4 failures (14 %) of the OAT and 11 failures (38 %) of MF treatment ($p < .05$). The survival probability was different in both groups: More than 85 % in the OAT group compare to 50 % in the MF group and this difference was significant. The ICRS and Tegner scores of younger athletes (25 years at the time of primary surgery) remained significantly higher after 10 years compared with older patients ($p < .05$).

18.3.3.2 Massive Allografts Cartilage for Lesion Greater than 4 cm²

This technique depends on the availability of soft tissue bank with fresh-stored allograft obtained in accordance with the screening guidelines of the association of tissue banks. This is a salvage procedure and often, associated surgeries were required.

The Gross et al. [84] team has extensive experience on the subject with many publications. They reported their experience of 125 cases since 1972 for patients who had an average age of 27 at the time of the intervention. It was in the majority of cases of post-traumatic injuries with loss of unipolar major substances (at least 3 cm in diameter and 1 cm deep). It is a massive fresh osteochondral allograft in accordance with the guidelines of the American Association of Tissue Banks. For the entire series, one case of deep infection was found. For the femur, 72 massive allografts were performed with equal distribution between the medial and lateral condyle. Associated realignment osteotomy was required in 68 % of cases. Twelve failures were often identified after the sixth year of evolution. The actuarial survival curve of the femoral allograft was 85 and 75 %, respectively, and 10 and 15 years of hindsight. Radiological evaluation showed that the spacing was long preserved. Severe osteoarthritic change was observed in 26 % of cases the greatest decline. Secondary conversion with the establishment of a total knee replacement was required in 15 % of the decline in 15 years. For the tibia, 72 transplants were performed (medial rather than lateral). Associated realignment osteotomy was made in 58 % of cases and a meniscal allograft (60 %). The current curve is 86 and 65 %, respectively, and 10, 15 and 46 %, respectively, at 20 years of follow-up. Severe osteoarthritis was observed in 39 % of the maximum decline, and conversion with implantation of TKA was required in 44 % of cases 20 years later.

Shaha et al. [85] evaluated the durability of return to activity in a high demanding population with the OATS technique in large areas of cartilage defect. Thirty-eight patients were included in the study; they all were involved in a single military institution. The overall rate to return to full duty activity was 29 and 29 % were able to limited activity with permanent modification. Forty-two percent were unable to return to high demand because of their knees. Regarding return to sport activity, only 5.3 % were able to return to their primary level. A little bit better 7 % when no other concomitant procedures were performed.

It was performed for bigger lesions, and this is really a challenge for the orthopaedic surgeons. This study is in contrast to the other two large series in the literature that report on return to activity after OATS similar procedures.

McCulloch et al. [86] described a series of 25 patients (25 knees) with a mean age of 35 years and a mean follow-up of 35 months. They reported improvements in the International Knee Documentation Committee score, Lysholm score, all 5 KOOS components and SF-12 physical component score. Overall, they reported rates of 84 % satisfaction and 79 % function compared with the contralateral knee. This study did not specify the level of activity in their population.

Similarly, a second large case series by Krych [87] reported on 43 isolated OATS procedures. The study population had a mean age of 33 years, a 79 % rate of return to sport at the pre-injury level and an 88 % rate of return to sport at any level. These authors specified activity level as defined by the Cincinnati sports activity scale (CSAS). They found age of less than 25 years as well as a short duration of symptoms to be significant predictors of a good outcome.

One explanation for the differences between the different series may lie in the definition of activity. The definition of return to sports really needs discussion to find a general agreement to talk about the same characteristics. This is an effective palliative surgery that is available but not used due to lack of plug-ins. This is a useful alternative to not ignore. Therefore in very large defects, the surgery improves pain and function but healing a chondral defect remains a challenging condition and further investigation in treatment is needed.

18.3.3.3 Osteochondral Autografts or Mosaicplasty (OAT)

This is an autograft with a single operation of transferring functional living units with a cylindrical form [88]. The goal is to transplant living chondrocytes and get coverage of more than 70 % hyaline cartilage of the defect. This is also based on an autologous bone support. This involves the harvest of autologous osteochondral plugs of tissue from other area of the knee.

Technically, the principles are the following:

- Preparation of the bed area with a curette and shaver. The defect was debrided of macroscopically damaged cartilage, and the edges were cut vertically to better accept the OATS.
- Estimate, during the procedure, the surface of the defect in order to choose the number and diameter of the cylinders. The harvest of the grafts is done using the appropriate ancillary.
- The osteochondral cylinder, which is 15–20 mm thick, is then impacted without rotation in the receiving bed to flush the healthy area. Care was taken not to put the plug below the level of the host cartilage in order to allow nutrition and good congruency during further knee movement.

The stability of the OATS is checked, and filling the gap between the plugs is a real challenge. It is recommended to use grafts of 7–8 mm diameter by 15 mm long and not exceed six transplants [88]. The technique can be used by arthroscopy for small lesions in a shortest possible arthrotomy.

Several questions remain unanswered. Are few large diameter grafts with better coverage better than several small diameter grafts? Choosing the contralateral area preferable to avoid the pain of the harvest site? What is the best way to restore the curvature?

Technical and radiological results are favourable for osteochondritis effects and lesions of the femoral condyle; the surface is less than 3 cm^2.

Lefort et al. [89] have reported the results of 98 mosaicplasty for OCD. The average age was 26 years, and the surface of the damaged area was 3.3+2.5 cm^2. These procedures were performed either arthroscopically or by arthrotomy. Four failures were taken (mobilisation of the graft, release of a foreign body) and 2 chondrolysis were observed. At follow-up, pain in 12 % pain from the harvest's site was reported. Clinical results with the IKDC score were good (87+4 pts.). They are even better when the age is less than 28 years and/or if the coverage exceeds 70 % of the defect.

Horas et al. [90] reported better results with 2 years of follow-up using OATS than ACI. However, there was no control group and the follow-up is short.

With long-term follow-up, Gudas et al. [83] reported better results with OATS versus MF. Bentley et al. [91] with more than 10 years of follow-up reported a randomised comparison of the two techniques in 100 patients at a minimum follow-up of 10 years. The mean age of the patients at the time of surgery was 31.3 years (16–49). The functional outcome of the patients with ACI demonstrated significantly better Cincinnati scores ($p = 0.02$). The number of patients whose repair had failed at 10 years was ten of 58 (17 %) in the ACI group and 23 of 42 (55 %) in the mosaicplasty group ($p < 0.001$). However, no return to sport activity was described in this long-term series.

It is a technique for young patients with a lesion of less than 3 cm^2 condylar reached without mirror aspect lesion or associated lower limb malalignment. It is also a reliable technique.

18.3.3.4 Cultured Chondrocytes or Autologous Chondrocytes Implantation (ACI)

This is a recent technique of tissue regeneration based on the biological dynamics of complex chondrocyte periosteum [92]. The principle is to insulate and culture the patient's own chondrocytes and then injecting them into the injured on a support that allows them to regenerate the loss of cartilage defect area. The first experimental work on tissue engineering goes back to Grande in 1987 [93]. Brittberg reported the first application in human clinical practice in the *New England Journal of Medicine* [94] in 1994.

We must distinguish several generations and changes in cultured chondrocytes.

- The first generation: the periosteal flap technique [94]. The idea is to take a 200 mg of arthroscopic biopsy of the healthy cartilage rom a non-weight-bearing zone with use of a curette from patients. Then, in a laboratory approved for cell culture, setting of specific cell culture is performed. After 3–6 weeks of culture, we obtained cell culture that is returned to the surgeon. Each sterile pellet contains ten million cells with a viability of 80 %. Finally the last step, the implantation of cells with arthrotomy with a periosteal flap taken from the tibia is sutured to cover the defect and filled the area with the chondrocytes cultivated. One of the difficulties is the realisation of the periosteal flap to allow the integration of cultivated chondrocytes. He reported good and excellent result on more than 100 cases with 2–9 years of follow-up. There is production of 'hyaline-like' cartilage with 88 % satisfactory results.

  In France, Robert published [95] results from a national prospective multicenter study supported by the French Arthroscopy Society of 28 cases with 2 years follow-up and histological control. The mean age was 28 years and the average size 4.9 cm^2. There was no infection and no thromboembolic complications. There was a significant clinical improvement with a gain of ICRS score of 80 % (4 pts. → 74 pts.). MRI analysis showed 11 cases of hypertrophy, and histologically, there is a satisfactory repair with mostly fibrocartilage tissue, but only 1 case is hyaline-like.

  This technique of periosteal flap is marketed under different names: Genzyme® (USA), Codon® and Verigen® (Germany). The need for the second surgical site for the removal of periosteal flap and complications such as hypertrophic periosteal flap were gradually abandoned by this technique.
- The second generation [92]: the periosteal flap as a collagen membrane patch that allows for better tissue repair replaces a barrier cell. Several membranes are being proposed by the industry:
  - Chondro-Gide® (Geistlich)
  - Chondroseal®, with ChondroCelect® membrane (TiGenix)
  - MCI® (Verigen)

  These methods have limitations (significant cost, quality of implanted chondrocytes) then the culture of the third generation has been developed through research and tissue engineering. However, the results of the second-generation techniques are encouraging.
- The third generation: tissue-engineering techniques have enabled the development of cell culture in three-dimensional artificial matrix. These matrices must have mechanical and biological properties similar to those

of normal cartilage. These matrixes or 'scaffolds' can cover large areas and facilitate cell proliferation with a more close to that of normal cartilage matrix. The specifications of these biomaterials are important. They must be biocompatible, biodegradable and fitted relationship to allow cell adhesion, adhesion properties and bio-integration. Finally, the biomaterial should not be cytotoxic, and its physical properties (size, volume, elasticity) should allow good filling of the defect whether partial or total.

These matrices or support three dimensions are numerous and can be of different types: synthetic protein or polysaccharide.

Synthetic matrices: PLA or PGA (Fleece, BioSeed® (Germany)), collagen membrane (ACI-Maix, Matricel®, Germany)

Protein nature collagen, gelatin or fibrin (Cores T Cais®, NeoCart®)

Polysaccharide type: with agarose (Cartipatch®, France (23)), alginate, chitosan (TSB CartGel®) and hyaluronate (Hyalograft®, Hyalf-11®)

All these researches in tissue engineering are based on two important and complementary concepts: applied research to develop implantable human matrices and basic research on cell culture (quality of chondrocyte phenotype, cell differentiation and growth factors).

In this technique, schematically four steps are necessary:

1. Evaluation of preoperative and intraoperative lesions also associated with taking 200 mg of cartilage in safe area (first arthroscopy).
2. Production in 6 weeks following the rules of accredited cell cultures from 20 to 40 million cells and integration of these cells in 3D matrices to create implants size and shape adapted to the defect.
3. Site preparation and implementation by arthrotomy to fill the defect (second surgery). The objective is to obtain a smooth articular surface without tear or absence of tissue continuity.
4. Passive rehabilitation and no support within 4 weeks. The recovery of the support is then progressive.

In 2010, Harris et al. [96] did a systematic review of autologous chondrocyte implantation with 13 studies and more than 917 subjects were included. Patients underwent autologous chondrocyte implantation ($n = 604$), microfracture ($n = 271$) or osteochondral autograft ($n = 42$). All surgical techniques demonstrated improvement in comparison with the preoperative status. This review demonstrated that there are patient-specific and defect-specific factors that do influence clinical outcome after autologous chondrocyte implantation.

However, regarding more specifically the results, there are some differences.

Regarding ACI versus MF, Saris et al. [97] randomised 118 patients to either characterised chondrocyte implantation ($n = 57$) or microfracture ($n = 61$). He reported a mean improvement from baseline to 36 months in terms of the overall KOOS score that was greater in the characterised chondrocyte implantation group. Significant differences were demonstrated supporting characterised chondrocyte implantation over microfracture in terms of the KOOS score ($p = 0.048$).

Kon et al. [98] performed a non-prospective cohort study in high-level sport athlete of 41 professional and semi-professional athletes. The mean age was close to 15 years old and the mean follow-up was 7.5 years. Twenty-one patients were treated with arthroscopic second-generation autologous chondrocyte implantation (Hyalograft C) and 20 with the microfracture technique. A significant improvement in all clinical scores from preoperative to final follow-up was found in both groups. The percentage of patients who returned to competition was similar: 80 % in the microfracture group and 86 % in the Hyalograft C group. Patients treated with microfracture needed a median of 8 months before playing their first official soccer game, whereas the Hyalograft C group required a median time of 12.5 months ($p = .009$). The International Knee Documentation Committee (IKDC) subjective score showed similar results at 2 years' follow-up but significantly better results in the Hyalograft C group at the final evaluation ($p = .005$). In fact, in the microfracture group, results decreased over time, whereas the Hyalograft C group presented a more durable outcome with stable results at the final follow-up. The success in returning to competitive sport is high in both groups, but microfracture allows a faster recovery but present a clinical deterioration over time. At the opposite, arthroscopic second-generation autologous chondrocyte implantation delays the return of high-level male soccer players to competition but offer more durable clinical results.

Regarding ACI versus OAT, there were few studies available in the literature to performed scientific comparison, and with the two studies, there is no conclusion available in this comparison. Horas [90] found a slight difference but it was not significant at 2 years. Finally, Bentley et al. [99] reported better results with cultured chondrocytes versus osteochondral autograft cartilage for larger lesions (4.6 cm^2).

Comparison between first- and second-generation ACI shown equivalent short-term clinical outcomes [28], and also we did not find any difference regarding open versus arthroscopic treatment [29].

18.3.3.5 Is It Realistic to Return to Play?

There is part of the answer in the question itself. In clinical practice, the objective of the treatment is to improve joint function and decrease pain. But there is lack of information on the ability to return the injured-knee athlete to demanding sports. Often, the methodology of the studies does not allow this kind of answer, and the definition of return to sport really needs discussion to find a general agreement to talk about the same characteristics. The evaluation using the ICRS level is part of the answer. The ICRS level classification allows further comparison between before and after the surgery with a sufficient long-term follow-up.

What are the informations provided by the literature?

Steadman et al. [81]. showed that 95 % of players returned to professional soccer the season following microfracture surgery and continued to play for an average of 5 years (range, 1–13 years) but the age was not consistent.

Gudas et al. [83] showed that 15 of 20 patients (75 %) in the OAT group and 8 of 22 patients (37 %) in the MF group maintained the same physical activity level. However, in both groups, a decrease in sport activity from 3 to 10 years was documented. He concluded that the OAT technique for ACD or OCD repair in the athletic population allows for a higher rate of return to and maintenance of sports at the pre-injury level compared with MF. Most of the athletes changed their level of activity because of a lifestyle change, or they decided to stop professional sports because of age limits or other reasons.

For massive allografts, the return rate to duty reported by Shaha et al. [85] is similar to that reported by Scully et al. [100] in the only other published series that examines return to duty after OATS in a military population. They found 1 of 16 included patients able to return to pre-injury activity.

One systematic review performed by Mithoefer et al. [101] focused more specifically on the ability to return to sports after articular cartilage repair; 1,410 patients were included with, respectively, 787 MF, 362 ACL and 261 OAT and the mean average follow up was 42 months. The average rate to return to sport activity was 73 %, and the average time to return to sports varied between 7 and 18 months (Table 18.4). The rate of return to sports at the initial pre-injury level was 68 %, and continued sport participation was possible in 65 %. This needs to be compared with the return to sport rates after several other common orthopaedic procedures such as ACL reconstruction (71 % (range 53–81)) and meniscal repair (74 % (range 56–85)).

In this review, several important factors were found to influence the ability to return to sports after articular cartilage repair in the knee. Therefore, these factors are very relevant to define the best profile regarding the patients when facing the challenging situation of knee cartilage defect.

These factors are listed below:

- Younger age resulted in better rates of return to sport participation with all surgical techniques. The reported age threshold for increased return to sports varied between 25 and 40 years.
- The length of the time interval between injury and surgery: the duration of symptoms with a time threshold of 12 months after injury and below one year of evolution.

Table 18.4 Comparison of return to sport rates in percentage according to Mithoefer systematic review [100]

	N cases	Lesions size (cm^2)	Return to sport participation (percentage)	Time to return to sports (months)	Return to sports at the pre-injury level	Best durability of return to sports
Overall	1,410	3.6 + 0.4	73 + 5	7–18	68 + 4	
MF	787	3.2 + 0.4	66 + 6 * SD	8 + 1	68 + 5	52 + 6
ACI	362	5.1 + 0.8	67 + 17	18 + 4 * SD	71 + 12	96 + 4 * SD
OAT	261	2.4 + 0.2	91 + 2 * SD	7 + 2	70 + 3	52 + 21

- Lesion size of less than 2 $cm^{2.}$
- The type of cartilage defect had influence: articular cartilage defect (ACD) did better results than osteochondral defect (OCD).
- Lesion location is less important but still relevant: lateral femoral condyle superior to medial femoral condyle.
- Athletes without prior surgical intervention were more likely to return to high-impact sports after microfracture.
- Athlete's skill level.
- Concomitant procedures.
- Repair tissue morphology.

In conclusion, the ideal candidate for cartilage repair could be a young competitive athlete with high-level sports with small defect size, short duration of symptoms and fewer prior surgical interventions. All these parameters should be addressed during this clinical situation in order to perform cartilage repair and propose a 'Menu à la carte'.

Autologous chondrocyte implantation may be the best option for large defects in young, active patients with a short duration of symptoms and no previous cartilage surgery. Autologous chondrocyte implantation may provide a more durable repair tissue than microfracture, with preservation of clinical outcome success in the long term. Microfracture remains indicated for smaller defects in young, active patients. Osteochondral autograft may provide more rapid improvement in terms of clinical outcome than autologous chondrocyte implantation or microfracture but is limited by donor site. Massive allograft cartilage for lesion greater than 4 cm^2 is a salvage procedure, and often associated surgeries were required. In this situation, return to sports is very questionable.

A complete rigorous evaluation with ICRS package is a must in 2013 with specific attention to five parameters when facing a young patient with knee pain related to cartilage defect.

1. Lesion analysis with ICRS package and quality imaging (MRI with specific sequences of cartilage gradient echo T1 and T2 FSE associated with FAT-SAT, axial and coronal).
2. Evaluate other joint elements: state of the ACL, menisci and the overall morphotype lower limbs.
3. Correlate the anatomical lesions found with the symptoms expressed by the patient; it is not optimal to treat asymptomatic and isolated lesion. Similarly, it is not reasonable to treat a degenerative lesion alone as osteoarthritis or pre-arthrosis lesion.
4. Treat other associated lesions: preliminary ACL reconstruction, realignment osteotomy (varus greater than 5°), gesture meniscal preservation and then propose a gesture for cartilage PDS after an observation period of at least 3–6 months.
5. Patient's motivation, skills and adherence to the surgical treatment.

Cartilage injuries in a high demand, athletic population remain a clinical challenge.

18.4 Foot Injuries

18.4.1 Ankle Sprain

This chapter will attempt to cover common ankle and foot sport trauma injuries and chronic conditions. For each pathology, the diagnosis and definition; epidemiology, history and mechanism of injury; examination findings; imaging studies; management and treatment options; outcomes and in particular return to sport will be discussed.

An ankle inversion injury is one of the most common sport injuries sustained, with over occurring one per 10,000 people per day. The majority of injuries occur in those aged 15–19 years and most occur in those less than 35 years old. Three quarters of the ligaments injured are the lateral ligaments, the anterior talofibular ligament and the calcaneofibular ligament, together with the deltoid ligament [102]. The anterior inferior tibiofibular ligaments, the interosseous ligament and the posterior inferior tibiofibular ligament at the syndesmosis may also be injured [103]. The majority of these will settle with a short period of immobilisation, with either cast or brace, and reduced weight-bearing, ice therapy and simple analgesics [104]. The use of nonsteroidal anti-inflammatory drugs is contentious in the early stages and can lead to increased bleeding [105]. However after 48 h, these medications may reduce inflammation and be a valuable synergist to analgesics.

Associated injuries include ankle instability and pain and swelling due to loose bodies, synovial thickening and chondral lesions.

Examination is most accurate at 5 days following injury when the initial bruising and swelling have reduced [106]. The ankle can be assessed for local tenderness and ligamentous laxity. Clinical tests include the anterior drawer test and an inversion stress test to determine integrity of the anterior talofibular ligament and the calcaneofibular ligament, respectively. These should be compared with the uninjured side and interpreted based upon the time line of the injury.

Patients with symptoms of instability have been found to have altered function of the stabilising peroneal musculature [107]. This delayed latency of muscular contraction can be targeted with physiotherapy but if patients continue to report symptoms, surgical stabilisation is an option.

Acute repair of lateral ligament rupture may lead to the same recovery to pre-injury activity level corresponding to functional treatment. Surgery does appear to reduce the risk of reinjury but may lead to an increased risk of osteoarthritis [108]. Ankle stabilisation can be categorised to ankle and subtalar joint instability [109]. Surgery can involve procedures to shorten the elongated anterolateral capsule following a rupture of the anterior talofibular ligament. This is the operation described by Broström [110] for which numerous variations exist. Arthroscopically assisted Broström procedures have been demonstrated to be effective. Nery reported that all patients had excellent or good postoperative function (AOFAS 90) [111]. These are consistent with Lee's finding of a postoperative AOFAS of 91 and talar tilt and anterior drawer was reduced to comparable to the non-injured side (talar tilt 3° & 2.5°, anterior translation 6.9 mm vs. 6.1 mm). High demand athletes with a Tegner score ≥ 6 underwent Gould-modified Broström procedure for chronic instability, and by 1 year the score had returned to a mean of 8.2 and by 2 years 8.6. Mean Karlsson scores were 92 and 95. There was a 6 % complication rate [112].

Additional symptomatic laxity can be stabilised by ligament reconstruction using either local tendon transfer from the peroneal tendons or hamstring or extensor digitorum longus fourth toe autograft [113]. Autograft [114] or allograft [115] would permit a more anatomic reconstruction to be performed. When anatomic and check-rein tenodesis were performed in the athletic population, the FAOS improved from 58 points to 89. All patients achieved mechanical stability, but 12 % demonstrated functional instability. This was found to functionally influence a failure to return to sport at the previous level [116]. Return to sport was achieved in 80 % patients following the Chrisman-Snook procedure at a median of 6 months following surgery. The mead FAOS was 74 and Kaikkonen 77 [117].

18.4.2 Syndesmosis Injury

Syndesmosis injury is a general term that encompasses any form of trauma to the distal tibiofibular joint, ranging from a simple ligamentous sprain to frank diastasis with concomitant bone, osteochondral and/or soft tissue injury. The incidence of syndesmotic injury ranges from 1 to 18 % of ankle sprains [118].

Isolated syndesmotic sprains, with no concomitant fractures, also called 'high ankle sprains', are the variety most frequently found in the athlete. Of all ankle sprains, 'high ankle sprains' have been shown to be associated with the longest recovery period and the greatest functional impairment [119].

These sprains are more prevalent in high-energy contact sports or in sports that involve wearing a stiff boot or skate, such as hockey or skiing. The most commonly described mechanism of syndesmotic injury involves an external rotation moment at the ankle with the foot positioned in dorsiflexion and pronation. The talus in the ankle mortise is forcibly externally rotated against the fibula; this tensions and ruptures the AITFL (anterior inferior tibiofibular ligament) first. As external rotation continues, disruption of the IOL (interosseous ligament) follows, and finally rupture or avulsion of the PITFL (posterior inferior tibiofibular ligament) occurs. The fibula may fracture in the final stage of this injury with persistently applied force.

The definition of the injury mechanism is the first guiding clue toward differentiating a syndesmotic injury from other lateral ankle injuries, especially because symptoms of pain and swelling in the anterolateral ankle and difficulty with ambulation are general to most ankle trauma.

The classic physical examination finding in patients with acute syndesmosis injuries is well-localised tenderness in the anterior space between tibia and fibula. Yet, according to some authors, specificity of local tenderness in acute setting is questionable [120]. The extension of the pain from the AITFL proximally up the anterolateral leg is suggestive of a more significant injury.

Numerous clinical tests have been described for syndesmotic injuries, but their clinical utility is still to be established. The squeeze test and the external rotation test are the most popular provocative tests [121]. Both use the reproduction of symptoms as the basis for a positive test (Fig. 18.25). de Cesar reported low sensitivity rates with specificity of 93.5 and 84.5 % for the squeeze test and the external rotation test, respectively [122]. The fibular translation test and the cotton test are dynamic evaluations of the syndesmosis, with the aim to directly assess the degree of bony translation available [121]. However, examiner's interpretation of 'looseness' and the need to perform these tests in precise positions and in specific directions of stress call into question their accuracy.

Fig. 18.25 Dorsiflexion external rotation test. With the knee in approximately 90° of flexion and stabilising the leg steady, the examiner applies an external rotation force to the ankle kept in dorsiflexion. The test result is positive if there is pain in the region of the distal syndesmosis ligaments or interosseous membrane, *black arrow*

Standard radiographs of the ankle, comprising weight-bearing anteroposterior (AP), mortise and lateral views, are the first set of investigation for a patient with clinically suspected syndesmosis injury. In cases of fracture or frank diastasis, these examinations are conclusive (a fracture and/or frank diastasis may be readily seen). The ability to detect less severe injuries on plain films is questionable. The major radiographic parameters

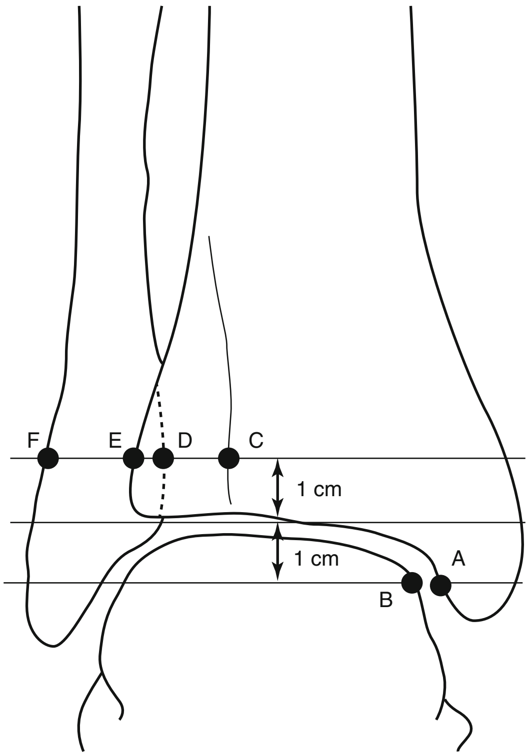

Fig. 18.26 Rx parameters for syndesmosis evaluation: *AB* medial clear space, distance between medial talar border and lateral medial malleolus border; *CD* tibiofibular clear space, distance between medial fibular border and lateral border of posterior tibial malleolus; *DE* tibiofibular overlap, distance between medial fibular border and lateral tibial border

to assess syndesmotic integrity – the medial clear space widening, the reduced tibiofibular overlap and the increased tibiofibular clear space (Fig. 18.26) – depend on the position of rotation, and it is actually not possible to place every ankle in the exact position for radiography to view the syndesmosis and the relationship of the tibia with the fibula [123]. Moreover, individual variability and gender-specific differences exist, which complicates matters, especially in cases of latent diastasis [124]. Because of these differences, comparison radiographs of the contralateral ankle may be valuable in reducing confusion in diagnosis. Some investigators have advocated the use of stress radiographs as the next step in exposing the injury [124], but also the role of this tool is controversial with reported low sensitivity rate for less severe injuries [125].

MRI and CT are currently the imaging modalities of choice. CT provides excellent bone detail, and the thin slices allow for associated bony avulsions to be appreciated. CT imaging has also improved the accuracy in detection of diastases of as small as 1 mm, not previously identifiable on plain radiographs [126]. MRI allows accurate delineation of the syndesmotic ligaments [127]. Its wide field of view allows detection of concomitant injuries, with no radiation burden to the patient. Arthroscopy is a useful diagnostic and therapeutic tool. Instability and the presence of AITFL and PITFL injuries can be identified and confirmed [127, 128] (Fig. 18.27). Besides, any dynamic instability may be addressed at the same time with diagnostic arthroscopy that allows the direct visualisation of the reduction [129].

Syndesmosis injuries have been classified chronologically, radiographically and clinically. At present, there is no classification system that allows clear definition of the degree of injury, guidance of treatment or prediction of outcome.

The temporal classification in acute (<6 weeks), subacute (6 weeks to 6 months) or chronic (>6 months) is general to several traumatic musculoskeletal conditions and helps to guide treatment [130].

Gerber et al. [131] provided a classification system based on clinical examination and radiographic parameters referred to as the West Point Ankle grading system (I–III). Grade I is a mild sprain to the AITFL with no instability, grade II refers to slight instability and tear of the AITFL and partial tear of the IOL, and grade III is definite instability characterised by the complete disruption of all the ligaments.

A grade II injury poses a particular diagnostic challenge, as its occult instability could be difficult to detect clinically and radiologically. MRI and arthroscopy may be helpful to define the real extent of the injury and to discriminate between group I and group II injuries, reducing the risk of undertreatment, that, especially in the athlete, can have serious consequences, as unacceptable instability may ensue [128, 132].

Stable syndesmotic injuries with no diastasis are adequately managed conservatively. The return to sports in a safely and timely manner at

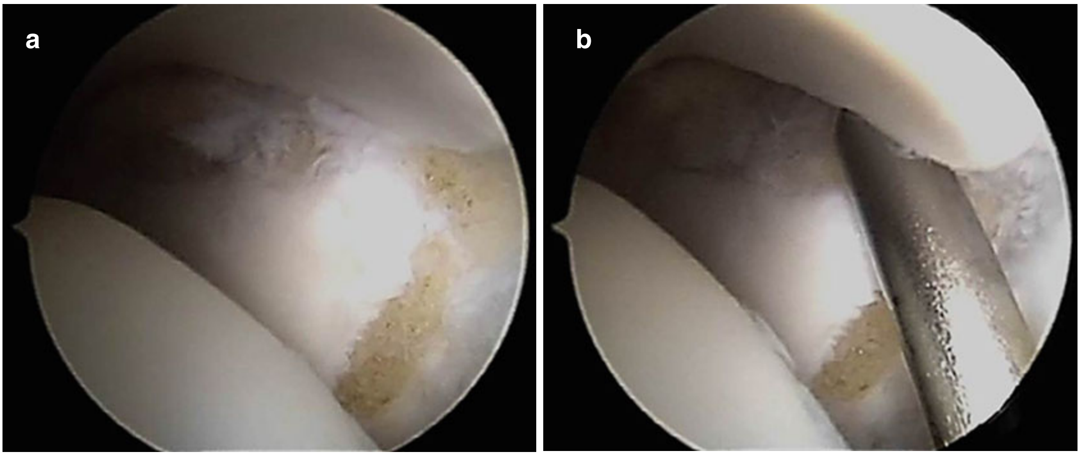

Fig. 18.27 (**a**) Arthroscopic exploration of chronic syndesmotic instability, left ankle, arthroscope in the anteromedial portal; (**b**) the smooth entry of the shaver blade in the anterior area of the syndesmosis reveals an unopposed diastasis of 4.0 mm, shaver blade diameter

the same pre-injury level is the major issue in treating an athlete. This is decided through careful consideration of the patient's report, functional testing results and clinical examination. Conservative treatment of syndesmosis sprains has an overall reported rate of 86–100 % good to excellent outcomes, and almost all cases achieve full return to sports [131, 133]. The recovery period after syndesmotic ankle sprains is highly variable, usually at 6–8 weeks post-injury, overall typically longer than the one following lateral ankle injuries [119, 131, 134].

No controversy about the management of syndesmotic injury with a displaced, widened mortise exists. Regardless the presence of a concomitant fracture, these conditions warrant operative fixation. The goal of surgical stabilisation is to restore and maintain the normal tibiofibular relationship to allow appropriate healing of the ligamentous structures of the syndesmosis. The pursuit of the perfect mortise is mandatory, as the existing data clearly point to necessary anatomic restoration of the syndesmosis for prevention of a debilitating late syndesmotic instability and development of post-traumatic arthritis [135]. The prognosis of treatment of acute ruptures is good [130].

However, these clear indications for surgical intervention represent a small percentage of the population. Taylor et al. [136] observed that only 1.7 % of syndesmotic sprains are grade III injuries with unstable radiographs and, as mentioned before, stress x-ray do not effectively improve the radiographic ability to detect an instability [137].

Controversies prevail with regard to whether nonoperative treatment or early surgery should be recommended in those whose syndesmosis injuries do not demonstrate widening on radiographs, in the presence of clinical suspicion of dynamic instability, as MRI examination could suggest on the basis of significant disruption of the syndesmotic stabilisers. Some authors advocate arthroscopic exploration for these grade II injuries in professional athletes, with eventual fixation if an instability is demonstrated [118]. Conservative treatment remains a viable option for grade II injury, especially in case of amateur sportspeople, with an expected prolonged time to return to sports.

When surgical stabilisation of an acute injury is decided, operative options include screw fixation, dynamic fixation with a suture button or anatomical repair of the AITFL [138]. Physiologic motion at the syndesmosis and the potential for an earlier return to motion, with no concern about screw breakage, are the theoretical advantages of the suture button device over the screw fixation. In a recent systematic review, Schepers showed the TightRope suture button device (TightRope®

syndesmotic repair kit; Arthrex, Inc., Naples, FL) to have similar outcome compared with the syndesmotic screw, with an earlier return to work and less frequent need for implant removal [139]. However, the longest follow-up for the TightRope system is currently approximately 3 years and it has to be continued to better define long-term effects. For several authors, metallic syndesmotic screw fixation is still considered the 'gold standard' [139]. There are no uniform recommendations regarding the technical aspects of syndesmotic screw fixation. Areas of controversy include the optimal number of cortices, the appropriate size of the screws, the position of the ankle joint during screw insertion, the use of one or two screws, the position of the screw(s) relative to the tibiotalar joint and the need for and timing of implant removal [138, 140]. Regarding the latter, literature data show no absolute need for the removal of syndesmotic screw, with no negative effects associated to screw breakage and/or loosening [141].

Chronic sprains with recalcitrant pain and functional instability are another indication for surgical fixation of the syndesmosis. Proposed procedures vary from the arthroscopic debridement of the syndesmosis for symptomatic relief [142, 143] to the creation of a synostosis to stabilise the distal tibiofibular joint [130]. For the athlete, the restoration of stability is a vital part of the management strategy; at this regard the literature suggests that it is not easy to regain complete stability by means of syndesmotic late repairs, with less favourable outcome as compared to properly treated acute injuries [130].

The literature review about syndesmotic injuries suggests several prospective areas for additional investigations. Currently, we do not have standardised methods of diagnosing and grading the severity of ligament damage at the time of injury nor guidelines to use these informations to choose the proper treatment to maximise each patient's return to activity. The biggest issue, especially in the athlete, remains to recognise and treat acutely the latent diastasis prompt to evolve in future instability. At this regard, MRI and arthroscopy has showed promising results [118, 127, 144].

Regarding operative procedures, the use of bioabsorbable screws for syndesmosis fixation has proven to be safe and effective, with the advantage of no need for implant removal and reduced hardware-related complications, as screw breakage and/or loosening [138, 145].

18.4.2.1 Conclusion

- Early recognition of a syndesmotic injury is mandatory for a successful outcome, as inadequate treatment of an acute syndesmotic injury may result in unacceptable chronic instability.
- The rehabilitation period of syndesmotic injuries tends to be longer than lateral ankle injuries.
- Acute grade II injuries in an athlete can be better evaluated by arthroscopy in order to definitely recognise latent instability.
- Late repairs for chronic syndesmotic instability give less favourable outcome as compared to properly treated acute injuries.

18.4.3 Talar Dome Chondral and Osteochondral Lesions

These occur with incidence of 27 per 100,000 person years and this frequency appears to be increasing [146]. The most common location for symptomatic OCLT was the central third of the lateral talar dome followed by the central third of the medial talar dome. Anterolateral and posteromedial lesions accounted for relatively few OCLTs. Compared with lateral OCLTs, medial OCLTs were significantly larger in transverse and anteroposterior diameter and surface area but no significant differences existed with regard to lesion depth. Overall, the majority of lesions were MRI stage II; however, stage II lesions were more likely located laterally, whereas stage III lesions were more likely located medially [147].

Treatment options include nonoperative reduced weight-bearing, splintage, and hyaluronic acid and platelet-rich plasma injections [148]. Surgical options include excision and curettage, filling the defect with autogenous cancellous bone graft, drilling both antegrade and retrograde and fixation osteochondral and iliac crest transplantation [149] and autologous chondrocyte implantation. Lesion depth of more than 7.8 mm, age greater than 80 years and uncovered medial lesions predict unsatisfactory outcome following arthroscopic debridement and bone marrow stimulation [150].

Postoperatively, early weight-bearing can be recommended for patients treated by microfracture for small to midsized osteochondral lesions of the talus [151].

Return to sport activity has been divided into four phases of increasing intensity: walking, jogging, return to non-contact sports (running without swerving) and return to contact sports (running with swerving and collision).

18.4.4 Achilles Tendon Injuries

Mid-portion Achilles tendinopathy is a common problem affecting 2.35 per 1,000 Dutch adults, with 35 % of these related to sports [152]. The terminology is now clear in that tendinopathy consists of a thickened, painful Achilles tendon, which results in loss of function. Surrounding swelling has been termed paratendinopathy and the structural histological change and imaging changes termed tendinosis [153]. The best clinical test for tendinopathy is pain on palpation of the Achilles tendon (sensitivity 84 %, specificity 73 %, kappa 0.74–0.96) and subjective reporting of pain 2–6 cm above the insertion of the calcaneum (sensitivity 78 %, specificity 77 %, kappa 0.75–0.96) [154]. The cause is thought to be due to injury followed by an incomplete healing response.

The Victorian Institute of Sport Assessment-Achilles score has been shown to be a validated, reliable and reproducible outcome score, which can be used to assess treatment [155]. Early studies noted that neovascularisation is associated with painful tendinosis but not in normal tendons [156]. The efficacy of management has been based on improvement in outcome score, reduction in tendon size and neovascularity. It has been uncertain as to why the tendinopathic tendons are painful. Nerve tissue has been considered to pass from the tendon alongside the neovascularisation from the ventral paratendinous tissue in chronic Achilles tendinosis [157].

Eccentric exercises have been thought to place the tendon under increased strain improving symptoms and reducing tendon thickness [158]. Even after 5 years, whilst VISA-A outcome scores had significantly improved, only 40 % of patients are pain free [159]. This contrasts with a comparable series suggesting that the majority of patients recover fully when treated with exercise alone [160]. Eccentric loading exercises have been the standard by which other treatment options are compared.

Further treatments include the topical application of glyceryl trinitrate patches [161] to improve blood flow to the tendon and extracorporal shock wave therapy. The application of a 1.25 mg patch of topical glyceryl trinitrate for 24 h led to improved pain, including at night, tenderness, hop testing, increased ankle plantar flexion work and continued efficacy at 3 years [162]. Other groups have not shown any additional benefit offered over standard treatments [163]. Extracorporal shock wave therapy has shown satisfactory treatment but produced significantly improved results on combining with eccentric loading exercises [164].

Injection treatments include sclerosant injections, high volume saline, local anaesthetic and steroid injections guided by ultrasound.

Steroid injections into and around the tendon have been associated with increased tendency to rupture. Given that the aetiology is considered to be a poor healing response, it seems logical that injections to promote healing are likely to be effective in treating the pathology. In a double-blind randomised study, no benefit of PRP over saline was found for the management of symptoms at 1 year [165, 166]. They did show improved tendon structure in both groups; however, this was thought to be due to concurrent eccentric exercises. Owens in a small study did find an improvement with the use of PRP but only a modest one.

Recent editorials have, however, suggested that the neovascularisation is a red herring [167]. Neovascularisation in Achilles tendinopathy is weakly related to clinical severity, mainly based upon the functional domain of the VISA-A score [168].

High volume image-guided injections have shown good short-term results with reduced VISA-A (46.3–84.1 $p < 0.001$), neovascularisation and decreased tendon diameter (8.7–7.6 mm, $p < 0.001$) for resistant non-insertional Achilles tendinopathy [169]. Sclerosant polidocanol injections in mid-portion Achilles tendinosis: remaining good clinical results and decreased tendon thickness at 2-year follow-up [170].

Surgical interventions include stripping the paratenon, percutaneous tenotomy and open decompression and tendon transfer.

Minimal invasive stripping has been used to remove peritendinous adhesions [171]. Percutaneous tenotomy has been performed in middle and long-distance runners with results rated as excellent in 25 out of 48 patients and good in 12 and complications occurred in 9 patients consisting of haematomas, sensitive scars, infections and hypertrophic scars. Strength and endurance were within 10 % of the operated side [172]. Tendons operated on with ultrasound guidance remained thickened and had abnormal structure even 8 years after the procedure. Isometric maximal muscle strength and isometric endurance gradually returned to values similar to the contralateral un-operated tendon [173]. When followed up at 17 years, the calf circumference and strength in the operated side were significantly lower than the contralateral side. On average the level of sports was 60 % less than before the onset of symptoms. Seventy-seven percent reported good or excellent outcome according to Boyden assessment. The tendon was generally thicker (7.0 vs. 8.7 mm). The mean VISA-A score was 78.5 [174].

More recently, the involvement of the adjacent plantaris tendon has been postulated because of the predisposition for pain on the medial side of the Achilles tendon [175]. A thickened plantaris was located close to medial Achilles 80 % of tendinopathic tendons [176]. Tenoscopic release lead to an improvement in AOFAS from 68 to 92 and SF-36 increased from 76 to 87 [177]. Interestingly, unilateral release has been shown to lead to bilateral pain relief and improvement.

Reconstruction has been reported using hamstring tendons, flexor hallucis longus and peroneus brevis transfer and bone patellar tendon bone graft [178, 179].

Using a peroneus brevis transfer, the ATRS at a mean of 48 months following surgery was 92.5 although calf circumference and strength were reduced [180]. These deficiencies are maintained in the long term, 15.5 years. The use of a hamstring autograft did not alleviate these weaknesses [181]. At 8.2 years follow-up, the mean ATRS was 88 using a less invasive technique. Twenty-two patients returned to their pre-injury level of activity at a mean of 6.7 months after surgery, and most had returned to their pre-injury level of sport activity within 9 months from surgery. Using a minimally invasive reconstruction, ATRS improved from 42 to 86 at 31.4 months and strength values remained lower than the non-affected side, but all patients were able to walk tiptoes and returned to their pre-injury occupation [174].

The transfer of flexor hallucis longus tendon has led to alleviation of pain; however, limitations of plantar flexion strength remain and AOFAS improved from 62 to 89 points. The outcome of FHL transfer was studied in a group of patients with mean age of 54 and BMI of 34 over 2 years. Overall pain intensity, SF-36, ankle osteoarthritis scale and performance of a single heel rise improved significantly. The passive range of motion of the first MTPJ decreased from 85° to 68°. There were 13 complications reported in 48 patients [178]. A cohort of patients with tendinosis scored an average of 96 at 27 months after surgery, and 95 % of patients were satisfied with their outcome [182].

Achilles tendon rupture occurs with incidence of 10/100,000 patients [183, 184]. Ruptures occur four times as commonly in males compared to females. The median age is 45 years; younger patients predominantly sustain a rupture participating in sport activity, whereas rupture in the elderly tends to occur during activities of daily living. The risk of contralateral rupture is 200 times [185].

Randomised studies have so far failed to show a difference in outcome between operative and nonoperative management of Achilles tendon rupture [186, 187]. A recent study showed improved plantar flexion strength and outcome score at 3 months in operatively treated patients; however, most outcomes are measured at 6 and 12 months following injury [188]. Meta-analyses and systematic reviews have consistently reported reduced re-rupture rates for patients managed with operative intervention at the expense of increased incidence in minor complications [189, 190].

Meta-analyses comparing minimally invasive and percutaneous repair with open repair has shown no significant difference in rates of re-rupture, tissue adhesion, sural nerve injury, deep infection and deep vein thrombosis, while minimally invasive surgery has shown considerably reduced risk of superficial wound infection and a three times greater patient satisfaction rate [189, 191].

The assessment of outcome following Achilles repair has focussed on re-rupture rates, complication rates and patient subjective scores of function. Most scores are determined at a single point of follow-up and restoration of function for the cohort or function at 1–2 years following injury and repair. Two studies from separate centres have reported very similar ATRS scores at 3, 6, 9 and 12 months following surgical repair of 43, 73, 83 and 89 [187, 192]. This is likely to be the functional outcome over time following repair. In Olsson's comparison study, there was a tendency to improved outcome at 3 months and improved hop and counter movement jump at 12 months. Keating and Wall's series showed improved score and plantar flexion strength at 3 months. Bergqvist's comparison study tended toward significance for the operative treatment of Achilles ruptures in elderly females [193]. Metz has reported that patients are unlikely to participate in sport activity at pre-injury levels if re-rupture occurs [194]. This suggests that surgical repair offers improved early outcome and improved function in athletes and elderly females.

18.4.5 Midfoot Soft Tissue Injury

Midfoot dislocations, termed Lisfranc injury, occur with incidence of 1 in 55,000. Males are two to four times more likely to sustain these injuries compared to females. Injury mechanisms typically associated with collision sports such as American Football involve hyper-plantar flexion force. The injury can occur in as much as 4 % of NFL players per year. Although these injuries are commonly described in dancers, they actually occur rarely in this group [195].

The Lisfranc ligament passes between the base of the second metatarsal and the medial cuneiform. There are no ligaments directly between the base of the first and second metatarsal. The dorsal ligaments have a third of strength of the Lisfranc ligament. Rupture can lead to subtle instability and widening between the first and second metatarsal leading to the loss of the stabilising key stone arrangement of the transverse and longitudinal arches of the foot.

Rupture can lead to persistent foot pain and ultimately osteoarthritis. The foot is swollen and bruised. Plane AP radiographs can reveal widening of the 1st–2nd metatarsal interspace. Oblique radiographs reveal alterations in the alignment of the third ray. Low energy injuries have been described in the athletic population [196]. These have been graded with grade I having pain, no opening to the interspace but unable to participate in sports, stage 2 reveals 1–5 mm opening and finally stage 3 with >5 mm separation, loss of midfoot height and arch, together with reduced distance between the medial cuneiform and the fifth metatarsal. Stage1 managed non-weight-bearing and stages 2 and 3, those with diastasis anatomic reduction and stabilisation. In their case series of 15 athletes, 93 % had an excellent outcome when followed up at 27 months.

18.4.6 First Metatarsophalangeal Joint Injury

Injuries to the first metatarsophalangeal joint have been termed turf toe [197]. The principal mechanism is forced hyperextension in 85 % cases. There is increased prevalence on synthetic surfaces and with lighter more flexible shoes [198]. In 12.8 % there is an associated osteocartilagenous injury [199]. The hallux is important in pivoting and cutting [200], and following injury the joint may have a reduced range of movement. Associated injuries include sesamoid disruption and differences of greater than 3 mm or more high likelihood of having sustained injury to at least 3 of the 4 ligaments of the plantar plate complex [201]. Irreducible dislocation may require open relocation and plantar plate repair. Elastoplast spica immobilisation followed by a metatarsal shoe may be used until symptoms settle.

18.4.7 Fifth Metatarsal Base Fracture

Since Sir Robert Jones has been reported sustaining a fifth metatarsal base fracture whilst dancing vigorously [202], the management of this fracture has been discussed. There is commonly an overuse component and symptoms of a dull ache to the region receding acute injury. The general incidence has been estimated at 1.8 per 1,000 person years. In professional footballers, the incidence is 0.04 injuries per 1,000 h exposure and thus a team of 25 players might expect a 5th MT fracture every fifth season. Forty-five percent of players had prodromal symptoms [203]. Fractures may be classified according to location, termed zones and chronic non-union changes. Tip avulsion fractures, extending into the metatarsal cuboid joint and are related to traction of a lateral band of the plantar aponeurosis, are considered to be zone 1. Fractures that more proximally occur in the metaphyseal bone and extend into the intermetatarsal joint are zone 2. Zone 3 injuries occur in the diaphysis. Non-union and chronic changes have been described by Torg. This classification is based upon the presence of a visible fracture line, sclerosis of the bone ends and the obliteration of the medullary cavity [204]. The blood supply is a key factor in these injuries with a recurrent blood supply supplying the diaphyseal–metaphyseal junction forming an intraosseous nutrient vessel. The biomechanical aspects of this injury suggest that there is an adductor moment arm leading to fracture so that the body weight loads the fifth metatarsal when the heel is off the ground, leading to a fracture between peroneus brevis and tertius proximal to the intermetatarsal ligament. The resultant fractures tend to open up laterally rather than plantarwards.

In injuries identified at the NFL combine, 61 % were located in the Jones area, 20 % the proximal diaphyseal area and 19 % of indeterminate location. Varus alignment predisposed to injury [205, 206].

Treatment options: nonoperative, with or without weight-bearing; operative includes debridement and bone grafting [207], intramedullary screw fixation and tension band wire stabilisation [208, 209].

After surgical treatment, the fractures healed faster (75 % vs. 33 %, $p<0.05$) [210]. Mologne has compared internal fixation to cast immobilisation in athletes and showed that those treated with screw fixation returned to sports by 8 weeks compared to 15 weeks for those immobilised in cast.

Return to sports after intramedullary fixation for acute fractures ranged from 4 to 18 weeks. Acute fractures treated nonoperatively had a union rate of 76 %, whereas in fractures treated with a screw it was 96 %. Delayed unions treated nonoperatively had a union rate of 44 and 97 % treated operatively. Non-unions treated with screw fixation healed in 97 % of cases. Early return to play in athletes prior to full radiological union is not advised in case of refracture [211]. Lee et al. report on a series of 42 patients with acute injuries and confirm union at a mean of 75 days. There were four refractures, four delayed-unions and one non-union [209].

Hunt recommends the use of a large solid screw 5.5 mm and autologous bone grafting for symptomatic refractures and non-unions in elite athletes [212]. In their comparison series, Porter showed the effectiveness of the 5.5 mm screw but were unable to demonstrate improvement over the 4.5 mm screw [213]; however, 4.5 mm screws had been shown to yield reliable and effective healing [214]. Alternatively symptomatic non-unions have been excised with good return to function in a small group of athletes with symptomatic non-unions [215].

Bone grafting recommended as in Popovic's series: 3 out of 11 fractures treated by percutaneous fixation refractured compared to none of the 7 treated by open fixation and bone grafting.

18.4.8 Distal Metatarsal Shaft Fractures

Fractures of the distal metatarsal shaft have been termed 'dancers fractures.' The principal mechanism in dancers is rotating with load on the ball of the foot during demi-pointe. A similar fracture may be sustained by slipping off a step and landing directly on the ball of the foot, whilst rotating, on the surface below. O'Malley's cohort of 35 dancers all reported to dancing with no long-term sequelae [216]. A recent study of 142 fractures showed that the nonoperative management of these fractures resulted in excellent long-term functional outcomes with only 2 painful non-unions that required open reduction and internal fixation with bone grafting [217].

18.4.9 Plantar Fasciitis

Plantar fascia is the principal static and dynamic stabiliser of the longitudinal arches of the foot.

It also acts as a shock absorber and helps to protect the underlying soft tissues. Degenerative changes can cause acute and chronic inflammation and may also cause calcification at the origin of the plantar fascia and bony traction spur formation [218].

Plantar fasciitis is usually an overuse injury at the origin of the plantar fascia caused by excessive stress to the foot or biomechanical abnormalities of the foot such as excessive pronation.

This condition may cause inflammatory or degenerative changes of the fascia and periostitis of the medial calcaneal tubercle [219].

The differential diagnosis of plantar fasciitis includes significant disorders such as calcaneal stress fracture, entrapment neuropathies (e.g. tarsal tunnel syndrome), calcaneal tumour, Paget's disease and systemic arthritides [219].

Traditionally, the first line of treatment of plantar fasciitis has been rest, analgesics with nonsteroidal drugs, physiotherapy, night splints and orthoses. Patients not responding to this treatment are usually advised with infiltration with corticosteroids or extracorporeal shock wave therapy. Surgery in the form of fasciectomy, neurolysis of the nerve to abductor digiti minimi and excision of the heel spur has been successfully used in resistant cases [220, 221].

Recently, promising results were reported with the use of platelet-rich plasma (PRP) injections for treating muscle and tendon injuries and degeneration [222].

In this chapter, we tried to resume all recent evidence in diagnostic procedures and treatment of plantar fasciitis.

Plantar fasciitis is described as a painful inflammatory process, generally at the origin of the plantar fascia on the calcaneus. It can also be central in the plantar arch or, less commonly, distal.

Approximately 10–16 % of the population suffer from plantar fasciitis [223]. Mechanical overload is generally believed to be fundamental to the development of the condition.

Obesity not only increases the risk of plantar fasciitis, but also increases the level of disability, which is proportional to the body mass index. Work-related weight-bearing and biomechanical abnormalities in the foot such as tight Achilles tendon and reduced ankle dorsiflexion are common predisposing factors. Normal gait requires a minimum 10° of dorsiflexion at the ankle joint to clear the ground. If the patient has less than 10° of dorsiflexion, the foot will compensate by pronating and increasing the tensile load on the plantar fascia [223].

The most common exam finding in plantar fasciitis is pain at the medial calcaneal tubercle, which may be exacerbated with passive ankle dorsiflexion or first digit extension. Pain is particularly severe with the first few steps taken in the morning or after a period of rest, and usually lessens after a period of warming up. Occasionally, the pain may spread to the whole of the foot including the toes.

If paresthesia occurs with percussion inferior to the medial malleolus, a possible nerve entrapment or tarsal tunnel syndrome must be suspected [224].

The diagnosis is usually clinical and rarely needs to be investigated further.

Ultrasonography, bone scintigraphy and MRI have been used to evaluate plantar fasciitis.

Increased thickness, hypoechogenicity and biconvexity are the main diagnostic findings on sonography. Bone scintigraphy shows diffuse increased activity along the fascia during dynamic and blood pool stages and focal increased activity at the inferior calcaneal surface in the late static phase. Common MRI findings are thickened plantar fascia, peritendinous oedema, bone marrow oedema of the calcaneus and fascial tears [225–227].

Conservative therapies remain the preferred approach to treating plantar fasciitis, successfully managing 85–90 % of cases [228].

Noninvasive treatment includes plantar fascia-specific stretching, nonsteroidal anti-inflammatory drugs, orthotics and night splints and shock wave therapy.

Plantar fascia-specific stretching is useful in treating chronic recalcitrant heel pain; use of nonsteroidal anti-inflammatory drugs has not shown evidence of pain reduction, both prefabricated and custom-made foot orthotics can decrease pain [229].

Extracorporeal shock wave therapy is indicated if there is failure of other conservative modalities such as stretching exercises, casting or night splinting. As this is a relatively safe procedure with no clinically relevant side effects, it could be considered before any surgical treatment and may be preferable to try before steroid infiltration.

Local infiltration of steroids to the plantar fascia can produce short-term alleviation of pain.

Long-term complications of steroid treatment are fat pad atrophy and rupture of plantar fascia [230].

Recently, promising results were reported with the use of platelet-rich plasma (PRP) injections for treating muscle and tendon injuries and degeneration [222]. This technique is gaining popularity in treatment of plantar fasciitis and has been shown to be as effective in reducing pain scores at 3 weeks and 6 months [231].

Recalcitrant cases where symptoms persist for more than 6–12 months, even after adequate conservative treatment, are usually selected for surgery. Open release and endoscopic fasciectomy are various modes of plantar fascia release. Patients undergoing surgery should expect a return to normal activity in approximately 2–3 months, and up to 35 % of patients may continue to have symptoms after surgical intervention.

Plantar fasciitis is a common cause of heel pain with many possible treatments.

Stretching the plantar fascia, using prefabricated orthotics and wearing night splints, have clinical evidence of benefit.

Corticosteroid injections must be reserved to patients that need a fast return to activity and are willing to accept the risks associated with this procedure.

Extracorporeal shock wave therapy improves pain and function scores; PRP has limited but promising evidence for patients with chronic pain.

Surgery is limited to recalcitrant cases where symptoms persist for more than 6–12 months, even after adequate conservative treatment.

References

1. Dimmick S, Stevens KJ, Brazier D, Anderson SE. Femoroacetabular impingement. Radiol Clin North Am. 2013;51(3):337–52.
2. Anderson CN, Riley GM, Gold GE, Safran MR. Hip-femoral acetabular impingement. Clin Sports Med. 2013;32(3):409–25.
3. Byrd JW. Femoroacetabular impingement in athletes: current concepts. Am J Sports Med. 2013 Aug 27. [Epub ahead of print] PMID:23982400 doi: 10.1177/0363546513499136.
4. Askling CM, Tengvar M, Saartok T, Thorstensson A. Acute first-time hamstring strains during slow-speed stretching: clinical, magnetic resonance imaging, and recovery characteristics. Am J Sports Med. 2007;35(10):1716–24.
5. Kerkhoffs GM, van Es N, Wieldraaijer T, Sierevelt IN, Ekstrand J, van Dijk CN. Diagnosis and prognosis of acute hamstring injuries in athletes. Knee Surg Sports Traumatol Arthrosc. 2013;21(2):500–9.
6. Warren P, Gabbe BJ, Schneider-Kolsky M, Bennell KL. Clinical predictors of time to return to competition and of recurrence following hamstring strain in elite Australian footballers. Br J Sports Med. 2010;44(6):415–9.
7. Ekstrand J, Healy JC, Walden M, Lee JC, English B, Hagglund M. Hamstring muscle injuries in professional football: the correlation of MRI findings with return to play. Br J Sports Med. 2012;46(2):112–7.
8. Comin J, Malliaras P, Baquie P, Barbour T, Connell D. Return to competitive play after hamstring injuries

involving disruption of the central tendon. Am J Sports Med. 2013;41(1):111–5.
9. Silder A, Sherry MA, Sanfilippo J, Tuite MJ, Hetzel SJ, Heiderscheit BC. Clinical and morphological changes following 2 rehabilitation programs for acute hamstring strain injuries: a randomized clinical trial. J Orthop Sports Phys Ther. 2013;43(5):284–99.
10. Askling CM, Tengvar M, Thorstensson A. Acute hamstring injuries in Swedish elite football: a prospective randomised controlled clinical trial comparing two rehabilitation protocols. Br J Sports Med. 2013;47(15):953–9.
11. Mason DL, Dickens VA, Vail A. Rehabilitation for hamstring injuries. Cochrane Database Syst Rev. 2012;(12):CD004575.
12. Reurink G, Goudswaard GJ, Tol JL, Verhaar JA, Weir A, Moen MH. Therapeutic interventions for acute hamstring injuries: a systematic review. Br J Sports Med. 2012;46(2):103–9.
13. Mayr HO, Weig TG, Plitz W. Arthrofibrosis following ACL reconstruction – reasons and outcome. Arch Orthop Trauma Surg. 2004;124(8):518–22.
14. Cosgarea AJ, Sebastianelli WJ, DeHaven KE. Prevention of arthrofibrosis after anterior cruciate ligament reconstruction using the central third patellar tendon autograft. Am J Sports Med. 1995;23(1):87–92.
15. Papastergiou SG, Koukoulias NE, Mikalef P, Ziogas E, Voulgaropoulos H. Meniscal tears in the ACL-deficient knee: correlation between meniscal tears and the timing of ACL reconstruction. Knee Surg Sports Traumatol Arthrosc. 2007;15(12):1438–44.
16. Yoo JC, Ahn JH, Lee SH, Yoon YC. Increasing incidence of medial meniscal tears in nonoperatively treated anterior cruciate ligament insufficiency patients documented by serial magnetic resonance imaging studies. Am J Sports Med. 2009;37(8):1478–83.
17. Kennedy J, Jackson MP, O'Kelly P, Moran R. Timing of reconstruction of the anterior cruciate ligament in athletes and the incidence of secondary pathology within the knee. J Bone Joint Surg Br. 2010;92(3):362–6.
18. Tandogan RN, Taser O, Kayaalp A, et al. Analysis of meniscal and chondral lesions accompanying anterior cruciate ligament tears: relationship with age, time from injury, and level of sport. Knee Surg Sports Traumatol Arthrosc. 2004;12(4):262–70.
19. Francis A, Thomas RD, McGregor A. Anterior cruciate ligament rupture: reconstruction surgery and rehabilitation. A nation-wide survey of current practice. Knee. 2001;8(1):13–8.
20. Oiestad BE, Engebretsen L, Storheim K, Risberg MA. Knee osteoarthritis after anterior cruciate ligament injury: a systematic review. Am J Sports Med. 2009;37(7):1434–43.
21. Aglietti P, Giron F, Losco M, Cuomo P, Ciardullo A, Mondanelli N. Comparison between single-and double-bundle anterior cruciate ligament reconstruction: a prospective, randomized, single-blinded clinical trial. Am J Sports Med. 2010;38(1):25–34.
22. Hussein M, van Eck CF, Cretnik A, Dinevski D, Fu FH. Prospective randomized clinical evaluation of conventional single-bundle, anatomic single-bundle, and anatomic double-bundle anterior cruciate ligament reconstruction: 281 cases with 3- to 5-year follow-up. Am J Sports Med. 2012;40(3):512–20.
23. Ibrahim SA, Hamido F, Al Misfer AK, Mahgoob A, Ghafar SA, Alhran H. Anterior cruciate ligament reconstruction using autologous hamstring double bundle graft compared with single bundle procedures. J Bone Joint Surg Br. 2009;91(10):1310–5.
24. Jarvela T, Moisala AS, Sihvonen R, Jarvela S, Kannus P, Jarvinen M. Double-bundle anterior cruciate ligament reconstruction using hamstring autografts and bioabsorbable interference screw fixation: prospective, randomized, clinical study with 2-year results. Am J Sports Med. 2008;36(2):290–7.
25. Jarvela T. Double-bundle versus single-bundle anterior cruciate ligament reconstruction: a prospective, randomize clinical study. Knee Surg Sports Traumatol Arthrosc. 2007;15(5):500–7.
26. Muneta T, Koga H, Mochizuki T, et al. A prospective randomized study of 4-strand semitendinosus tendon anterior cruciate ligament reconstruction comparing single-bundle and double-bundle techniques. Arthroscopy. 2007;23(6):618–28.
27. Siebold R, Dehler C, Ellert T. Prospective randomized comparison of double-bundle versus single-bundle anterior cruciate ligament reconstruction. Arthroscopy. 2008;24(2):137–45.
28. Zaffagnini S, Bruni D, Russo A, et al. ST/G ACL reconstruction: double strand plus extra-articular sling vs double bundle, randomized study at 3-year follow-up. Scandinavian J Med Sci Sports. 2008;18(5):573–81.
29. Zaffagnini S, Bruni D, Marcheggiani Muccioli GM, et al. Single-bundle patellar tendon versus non-anatomical double-bundle hamstrings ACL reconstruction: a prospective randomized study at 8-year minimum follow-up. Knee Surg Sports Traumatol Arthrosc. 2011;19(3):390–7.
30. Adachi N, Ochi M, Uchio Y, Iwasa J, Kuriwaka M, Ito Y. Reconstruction of the anterior cruciate ligament. Single- versus double-bundle multistranded hamstring tendons. J Bone Joint Surg Br. 2004;86(4):515–20.
31. Sastre S, Popescu D, Nunez M, Pomes J, Tomas X, Peidro L. Double-bundle versus single-bundle ACL reconstruction using the horizontal femoral position: a prospective, randomized study. Knee Surg Sports Traumatol Arthrosc. 2010;18(1):32–6.
32. Streich NA, Friedrich K, Gotterbarm T, Schmitt H. Reconstruction of the ACL with a semitendinosus tendon graft: a prospective randomized single blinded comparison of double-bundle versus single-bundle technique in male athletes. Knee Surg Sports Traumatol Arthrosc. 2008;16(3):232–8.
33. Suomalainen P, Moisala AS, Paakkala A, Kannus P, Jarvela T. Double-bundle versus single-bundle anterior cruciate ligament reconstruction: randomized clinical and magnetic resonance imaging

study with 2-year follow-up. Am J Sports Med. 2011;39(8):1615–22.
34. Suomalainen P, Jarvela T, Paakkala A, Kannus P, Jarvinen M. Double-bundle versus single-bundle anterior cruciate ligament reconstruction: a prospective randomized study with 5-year results. Am J Sports Med. 2012;40(7):1511–8.
35. Aglietti P, Giron F, Buzzi R, Biddau F, Sasso F. Anterior cruciate ligament reconstruction: bone-patellar tendon-bone compared with double semitendinosus and gracilis tendon grafts. A prospective, randomized clinical trial. J Bone Joint Surg Am. 2004;86-A(10):2143–55.
36. Ahlden M, Kartus J, Ejerhed L, Karlsson J, Sernert N. Knee laxity measurements after anterior cruciate ligament reconstruction, using either bone-patellar-tendon-bone or hamstring tendon autografts, with special emphasis on comparison over time. Knee Surg Sports Traumatol Arthrosc. 2009;17(9):1117–24.
37. Beard DJ, Anderson JL, Davies S, Price AJ, Dodd CA. Hamstrings vs. patella tendon for anterior cruciate ligament reconstruction: a randomised controlled trial. Knee. 2001;8(1):45–50.
38. Gifstad T, Sole A, Strand T, Uppheim G, Grontvedt T, Drogset JO. Long-term follow-up of patellar tendon grafts or hamstring tendon grafts in endoscopic ACL reconstructions. Knee Surg Sports Traumatol Arthrosc. 2013;21:576–83.
39. Liden M, Ejerhed L, Sernert N, Laxdal G, Kartus J. Patellar tendon or semitendinosus tendon autografts for anterior cruciate ligament reconstruction: a prospective, randomized study with a 7-year follow-up. Am J Sports Med. 2007;35(5):740–8.
40. Taylor DC, DeBerardino TM, Nelson BJ, et al. Patellar tendon versus hamstring tendon autografts for anterior cruciate ligament reconstruction: a randomized controlled trial using similar femoral and tibial fixation methods. Am J Sports Med. 2009;37(10):1946–57.
41. Holm I, Oiestad BE, Risberg MA, Aune AK. No difference in knee function or prevalence of osteoarthritis after reconstruction of the anterior cruciate ligament with 4-strand hamstring autograft versus patellar tendon-bone autograft: a randomized study with 10-year follow-up. Am J Sports Med. 2010;38(3):448–54.
42. Aune AK, Holm I, Risberg MA, Jensen HK, Steen H. Four-strand hamstring tendon autograft compared with patellar tendon-bone autograft for anterior cruciate ligament reconstruction. A randomized study with two-year follow-up. Am J Sports Med. 2001;29(6):722–8.
43. Barenius B, Nordlander M, Ponzer S, Tidermark J, Eriksson K. Quality of life and clinical outcome after anterior cruciate ligament reconstruction using patellar tendon graft or quadrupled semitendinosus graft: an 8-year follow-up of a randomized controlled trial. Am J Sports Med. 2010;38(8):1533–41.
44. Ejerhed L, Kartus J, Sernert N, Kohler K, Karlsson J. Patellar tendon or semitendinosus tendon autografts for anterior cruciate ligament reconstruction? A prospective randomized study with a two-year follow-up. Am J Sports Med. 2003;31(1):19–25.
45. Feller JA, Webster KE, Gavin B. Early post-operative morbidity following anterior cruciate ligament reconstruction: patellar tendon versus hamstring graft. Knee Surg Sports Traumatol Arthrosc. 2001;9(5):260–6.
46. Ibrahim SA, Al-Kussary IM, Al-Misfer AR, Al-Mutairi HQ, Ghafar SA, El Noor TA. Clinical evaluation of arthroscopically assisted anterior cruciate ligament reconstruction: patellar tendon versus gracilis and semitendinosus autograft. Arthroscopy. 2005;21(4):412–7.
47. Laxdal G, Kartus J, Hansson L, Heidvall M, Ejerhed L, Karlsson J. A prospective randomized comparison of bone-patellar tendon-bone and hamstring grafts for anterior cruciate ligament reconstruction. Arthroscopy. 2005;21(1):34–42.
48. Maletis GB, Cameron SL, Tengan JJ, Burchette RJ. A prospective randomized study of anterior cruciate ligament reconstruction: a comparison of patellar tendon and quadruple-strand semitendinosus/gracilis tendons fixed with bioabsorbable interference screws. Am J Sports Med. 2007;35(3):384–94.
49. Matsumoto A, Yoshiya S, Muratsu H, et al. A comparison of bone-patellar tendon-bone and bone-hamstring tendon-bone autografts for anterior cruciate ligament reconstruction. Am J Sports Med. 2006;34(2):213–9.
50. Sajovic M, Strahovnik A, Dernovsek MZ, Skaza K. Quality of life and clinical outcome comparison of semitendinosus and gracilis tendon versus patellar tendon autografts for anterior cruciate ligament reconstruction: an 11-year follow-up of a randomized controlled trial. Am J Sports Med. 2011;39(10):2161–9.
51. Lee GH, McCulloch P, Cole BJ, Bush-Joseph CA, Bach Jr BR. The incidence of acute patellar tendon harvest complications for anterior cruciate ligament reconstruction. Arthroscopy. 2008;24(2):162–6.
52. Fanelli GC, Harris JD. Surgical treatment of acute medial collateral ligament and posteromedial corner injuries of the knee. Sports Med Arthrosc. 2006;14(2):78–83.
53. Fetto JF, Marshall JL. Medial collateral ligament injuries of the knee: a rationale for treatment. Clin Orthop Relat Res. 1978;132:206–18.
54. O'Donoghue DH. Reconstruction for medial instability of the knee. J Bone Joint Surg Am. 1973;55(5):941–54.
55. Collins HR. Reconstruction of the athlete's injured knee: anatomy, diagnosis, treatment. Orthop Clin North Am. 1971;2(1):207–30.
56. Kannus P. Long-term results of conservatively treated medial collateral ligament injuries of the knee joint. Clin Orthop Relat Res. 1988;226:103–12.
57. Indelicato PA, Hermansdorfer J, Huegel M. Nonoperative management of complete tears of the medial collateral ligament of the knee in intercollegiate football players. Clin Orthop Relat Res. 1990;256:174–7.
58. Indelicato PA. Non-operative treatment of complete tears of the medial collateral ligament of the knee. J Bone Joint Surg Am. 1983;65(3):323–9.

59. Jones RE, Henley MB, Francis P. Nonoperative management of isolated grade III collateral ligament injury in high school football players. Clin Orthop Relat Res. 1986;213:137–40.
60. Sandberg R, Balkfors B, Nilsson B, Westlin N. Operative versus non-operative treatment of recent injuries to the ligaments of the knee. A prospective randomized study. J Bone Joint Surg Am. 1987; 69(8):1120–6.
61. Reider B, Sathy MR, Talkington J, Blyznak N, Kollias S. Treatment of isolated medial collateral ligament injuries in athletes with early functional rehabilitation. A five-year follow-up study. Am J Sports Med. 1994;22(4):470–7.
62. Edson CJ. Conservative and postoperative rehabilitation of isolated and combined injuries of the medial collateral ligament. Sports Med Arthrosc. 2006;14(2):105–10.
63. Eirale C, Mauri E, Hamilton B. Use of platelet rich plasma in an isolated complete medial collateral ligament lesion in a professional football (soccer) player: a case report. Asian J Sports Med. 2013;4(2):158–62.
64. 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.
65. 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.
66. Yoshioka T, Kanamori A, Washio T, et al. The effects of plasma rich in growth factors (PRGF-Endoret) on healing of medial collateral ligament of the knee. Knee Surg Sports Traumatol Arthrosc. 2013;21(8): 1763–9.
67. Ueshima K, Kitaoka K, Nakase J, Xu Q, Matsumoto K, Tsuchiya H. Promotion of rabbit ligament healing by local delivery of hepatocyte growth factor. J Orthop Sci. 2011;16(4):451–7.
68. Saiga K, Furumatsu T, Yoshida A, et al. Combined use of bFGF and GDF-5 enhances the healing of medial collateral ligament injury. Biochem Biophys Res Commun. 2010;402(2):329–34.
69. Paoloni J, De Vos RJ, Hamilton B, Murrell GA, Orchard J. Platelet-rich plasma treatment for ligament and tendon injuries. Clin J Sport Med. 2011;21(1):37–45.
70. Phisitkul P, James SL, Wolf BR, Amendola A. MCL injuries of the knee: current concepts review. Iowa Orthop J. 2006;26:77–90.
71. Bonasia DE, Bruzzone M, Dettoni F, et al. Treatment of medial and posteromedial knee instability: indications, techniques, and review of the results. Iowa Orthop J. 2012;32:173–83.
72. Kim SJ, Lee DH, Kim TE, Choi NH. 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.
73. Stannard JP. Medial and posteromedial instability of the knee: evaluation, treatment, and results. Sports Med Arthrosc. 2010;18(4):263–8.
74. 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.
75. 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.
76. Wijdicks CA, Griffith CJ, Johansen S, Engebretsen L, LaPrade RF. Injuries to the medial collateral ligament and associated medial structures of the knee. J Bone Joint Surg Am. 2010;92(5):1266–80.
77. 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.
78. Borden PS, Kantaras AT, Caborn DN. Medial collateral ligament reconstruction with allograft using a double-bundle technique. Arthroscopy. 2002;18(4):E19.
79. Hunter W. Of the structure and disease of articulating cartilages. 1743. Clin Orthop Relat Res. 1995;317:3–6.
80. Aroen A, Loken S, Heir S, et al. Articular cartilage lesions in 993 consecutive knee arthroscopies. Am J Sports Med. 2004;32(1):211–5.
81. Steadman JR, Briggs KK, Rodrigo JJ, Kocher MS, Gill TJ, Rodkey WG. Outcomes of microfracture for traumatic chondral defects of the knee: average 11-year follow-up. Arthroscopy. 2003;19(5):477–84.
82. Johnson LL. Arthroscopic abrasion arthroplasty historical and pathologic perspective: present status. Arthroscopy. 1986;2(1):54–69.
83. Gudas R, Gudaite A, Pocius A, et al. Ten-year follow-up of a prospective, randomized clinical study of mosaic osteochondral autologous transplantation versus microfracture for the treatment of osteochondral defects in the knee joint of athletes. Am J Sports Med. 2012;40(11):2499–508.
84. Gross AE, Shasha N, Aubin P. Long-term followup of the use of fresh osteochondral allografts for posttraumatic knee defects. Clin Orthop Relat Res. 2005;435:79–87.
85. Shaha JS, Cook JB, Rowles DJ, Bottoni CR, Shaha SH, Tokish JM. Return to an athletic lifestyle after osteochondral allograft transplantation of the knee. Am J Sports Med. 2013;41(9):2083–9.
86. McCulloch PC, Kang RW, Sobhy MH, Hayden JK, Cole BJ. Prospective evaluation of prolonged fresh osteochondral allograft transplantation of the femoral condyle: minimum 2-year follow-up. Am J Sports Med. 2007;35(3):411–20.
87. Krych AJ, et al. Return to athletic activity after osteochondral allograft transplantation in the knee. Am J Sports Med. 2012;40(5):1053–9. doi: 10.1177/0363546511435780. Epub 2012 Feb 7.
88. Hangody L, Fules P. Autologous osteochondral mosaicplasty for the treatment of full-thickness defects of weight-bearing joints: ten years of experimental and clinical experience. J Bone Joint Surg Am. 2003;85-A Suppl 2:25–32.

89. Lefort G, Moyen B, Beaufils P, et al. Osteochondritis dissecans of the femoral condyles: report of 892 cases. Rev Chir Orthop Reparatrice Appar Mot. 2006;92:2s97–92s141. France.
90. Horas U, Pelinkovic D, Herr G, Aigner T, Schnettler R. Autologous chondrocyte implantation and osteochondral cylinder transplantation in cartilage repair of the knee joint. A prospective, comparative trial. J Bone Joint Surg Am. 2003;85-a(2):185–92.
91. Bentley G, Biant LC, Carrington RW, et al. A prospective, randomised comparison of autologous chondrocyte implantation versus mosaicplasty for osteochondral defects in the knee. J Bone Joint Surg Br. 2003;85(2):223–30.
92. Galois L, Freyria AM, Herbage D, Mainard D. Cartilage tissue engineering: state-of-the-art and future approaches. Pathol Biol (Paris). 2005; 53(10):590–8.
93. Grande DA, Singh IJ, Pugh J. Healing of experimentally produced lesions in articular cartilage following chondrocyte transplantation. Anat Rec. 1987;218(2):142–8.
94. Brittberg M, Lindahl A, Nilsson A, Ohlsson C, Isaksson O, Peterson L. Treatment of deep cartilage defects in the knee with autologous chondrocyte transplantation. N Engl J Med. 1994;331(14):889–95.
95. Robert H, Bahuaud J, Kerdiles N, et al. Treatment of deep cartilage defects in the knee with autologous chondrocyte transplantation: a review of 28 cases. Rev Chir Orthop Reparatrice Appar Mot. 2007;93(7): 701–9.
96. Harris JD, Siston RA, Pan X, Flanigan DC. Autologous chondrocyte implantation: a systematic review. J Bone Joint Surg Am. 2010;92(12):2220–33.
97. Saris DB, Vanlauwe J, Victor J, et al. Treatment of symptomatic cartilage defects of the knee: characterized chondrocyte implantation results in better clinical outcome at 36 months in a randomized trial compared to microfracture. Am J Sports Med. 2009;37 Suppl 1:10s–9.
98. Kon E, Filardo G, Berruto M, et al. Articular cartilage treatment in high-level male soccer players: a prospective comparative study of arthroscopic second-generation autologous chondrocyte implantation versus microfracture. Am J Sports Med. 2011;39(12):2549–57.
99. Bentley G, Biant LC, Vijayan S, Macmull S, Skinner JA, Carrington RW. Minimum ten-year results of a prospective randomised study of autologous chondrocyte implantation versus mosaicplasty for symptomatic articular cartilage lesions of the knee. J Bone Joint Surg Br. 2012;94(4):504–9.
100. Scully WF, Parada SA, Arrington ED. Allograft osteochondral transplantation in the knee in the active duty population. Mil Med. 2011;176(10):1196–201.
101. Mithoefer K, Hambly K, Della Villa S, Silvers H, Mandelbaum BR. Return to sports participation after articular cartilage repair in the knee: scientific evidence. Am J Sports Med. 2009;37 Suppl 1:167s–76.
102. Golano P, Vega J, de Leeuw PA, et al. Anatomy of the ankle ligaments: a pictorial essay. Knee Surg Sports Traumatol Arthrosc. 2010;18(5):557–69.
103. Hermans JJ, Beumer A, de Jong TA, Kleinrensink GJ. Anatomy of the distal tibiofibular syndesmosis in adults: a pictorial essay with a multimodality approach. J Anat. 2010;217(6):633–45.
104. Kerkhoffs GM, van den Bekerom M, Elders LA, et al. Diagnosis, treatment and prevention of ankle sprains: an evidence-based clinical guideline. Br J Sports Med. 2012;46(12):854–60.
105. Chen MR, Dragoo JL. The effect of nonsteroidal anti-inflammatory drugs on tissue healing. Knee Surg Sports Traumatol Arthrosc. 2013;21(3):540–9.
106. van Dijk CN, Lim LS, Bossuyt PM, Marti RK. Physical examination is sufficient for the diagnosis of sprained ankles. J Bone Joint Surg Br. 1996;78(6):958–62.
107. Hopkins JT, Brown TN, Christensen L, Palmieri-Smith RM. Deficits in peroneal latency and electromechanical delay in patients with functional ankle instability. J Orthop Res. 2009;27(12):1541–6.
108. Pihlajamaki H, Hietaniemi K, Paavola M, Visuri T, Mattila VM. Surgical versus functional treatment for acute ruptures of the lateral ligament complex of the ankle in young men: a randomized controlled trial. J Bone Joint Surg Am. 2010;92(14):2367–74.
109. Barg A, Tochigi Y, Amendola A, Phisitkul P, Hintermann B, Saltzman CL. Subtalar instability: diagnosis and treatment. Foot Ankle Int. 2012;33(2): 151–60.
110. Brostrom L. Sprained ankles. VI. Surgical treatment of "chronic" ligament ruptures. Acta Chir Scand. 1966;132(5):551–65.
111. Nery C, Raduan F, Del Buono A, Asaumi ID, Cohen M, Maffulli N. Arthroscopic-assisted Brostrom-Gould for chronic ankle instability: a long-term follow-up. Am J Sports Med. 2011;39(11):2381–8.
112. Li X, Killie H, Guerrero P, Busconi BD. Anatomical reconstruction for chronic lateral ankle instability in the high-demand athlete: functional outcomes after the modified Brostrom repair using suture anchors. Am J Sports Med. 2009;37(3):488–94.
113. Ahn JH, Choy WS, Kim HY. Reconstruction of the lateral ankle ligament with a long extensor tendon graft of the fourth toe. Am J Sports Med. 2011;39(3):637–44.
114. Coughlin MJ, Schenck Jr RC, Grebing BR, Treme G. Comprehensive reconstruction of the lateral ankle for chronic instability using a free gracilis graft. Foot Ankle Int. 2004;25(4):231–41.
115. Ellis SJ, Williams BR, Pavlov H, Deland J. Results of anatomic lateral ankle ligament reconstruction with tendon allograft. HSS J. 2011;7(2):134–40.
116. Kennedy JG, Smyth NA, Fansa AM, Murawski CD. Anatomic lateral ligament reconstruction in the ankle: a hybrid technique in the athletic population. Am J Sports Med. 2012;40(10):2309–17.
117. Kramer D, Solomon R, Curtis C, Zurakowski D, Micheli LJ. Clinical results and functional

evaluation of the Chrisman-Snook procedure for lateral ankle instability in athletes. Foot Ankle Spec. 2011;4(1):18–28.
118. McCollum GA, van den Bekerom MP, Kerkhoffs GM, Calder JD, van Dijk CN. Syndesmosis and deltoid ligament injuries in the athlete. Knee Surg Sports Traumatol Arthrosc. 2013;21(6):1328–37.
119. Wright RW, Barile RJ, Surprenant DA, Matava MJ. Ankle syndesmosis sprains in national hockey league players. Am J Sports Med. 2004;32(8):1941–5.
120. van Dijk CN, Mol BW, Lim LS, Marti RK, Bossuyt PM. Diagnosis of ligament rupture of the ankle joint. Physical examination, arthrography, stress radiography and sonography compared in 160 patients after inversion trauma. Acta Orthop Scand. 1996;67(6):566–70.
121. Alonso A, Khoury L, Adams R. Clinical tests for ankle syndesmosis injury: reliability and prediction of return to function. J Orthop Sports Phys Ther. 1998;27(4):276–84.
122. de Cesar PC, Avila EM, de Abreu MR. Comparison of magnetic resonance imaging to physical examination for syndesmotic injury after lateral ankle sprain. Foot Ankle Int. 2011;32(12):1110–4.
123. Beumer A, van Hemert WL, Niesing R, et al. Radiographic measurement of the distal tibiofibular syndesmosis has limited use. Clin Orthop Relat Res. 2004;423:227–34.
124. Edwards Jr GS, DeLee JC. Ankle diastasis without fracture. Foot Ankle. 1984;4(6):305–12.
125. Ogilvie-Harris DJ, Reed SC. Disruption of the ankle syndesmosis: diagnosis and treatment by arthroscopic surgery. Arthroscopy. 1994;10(5):561–8.
126. Ebraheim NA, Lu J, Yang H, Mekhail AO, Yeasting RA. Radiographic and CT evaluation of tibiofibular syndesmotic diastasis: a cadaver study. Foot Ankle Int. 1997;18(11):693–8.
127. Oae K, Takao M, Naito K, et al. Injury of the tibiofibular syndesmosis: value of MR imaging for diagnosis. Radiology. 2003;227(1):155–61.
128. Ogilvie-Harris DJ, Gilbart MK, Chorney K. Chronic pain following ankle sprains in athletes: the role of arthroscopic surgery. Arthroscopy. 1997;13(5):564–74.
129. Mei-Dan O, Kots E, Barchilon V, Massarwe S, Nyska M, Mann G. A dynamic ultrasound examination for the diagnosis of ankle syndesmotic injury in professional athletes: a preliminary study. Am J Sports Med. 2009;37(5):1009–16.
130. van den Bekerom MP, de Leeuw PA, van Dijk CN. Delayed operative treatment of syndesmotic instability. Current concepts review. Injury. 2009;40(11):1137–42.
131. Gerber JP, Williams GN, Scoville CR, Arciero RA, Taylor DC. Persistent disability associated with ankle sprains: a prospective examination of an athletic population. Foot Ankle Int. 1998;19(10):653–60.
132. Harper MC. Delayed reduction and stabilization of the tibiofibular syndesmosis. Foot Ankle Int. 2001;22(1):15–8.
133. Amendola A, Williams G, Foster D. Evidence-based approach to treatment of acute traumatic syndesmosis (high ankle) sprains. Sports Med Arthrosc. 2006;14(4):232–6.
134. Boytim MJ, Fischer DA, Neumann L. Syndesmotic ankle sprains. Am J Sports Med. 1991;19(3):294–8.
135. Leeds HC, Ehrlich MG. Instability of the distal tibiofibular syndesmosis after bimalleolar and trimalleolar ankle fractures. J Bone Joint Surg Am. 1984;66(4):490–503.
136. Taylor DC, Tenuta JJ, Uhorchak JM, Arciero RA. Aggressive surgical treatment and early return to sports in athletes with grade III syndesmosis sprains. Am J Sports Med. 2007;35(11):1833–8.
137. Press CM, Gupta A, Hutchinson MR. Management of ankle syndesmosis injuries in the athlete. Curr Sports Med Rep. 2009;8(5):228–33.
138. van den Bekerom MP, Raven EE. Current concepts review: operative techniques for stabilizing the distal tibiofibular syndesmosis. Foot Ankle Int. 2007;28(12):1302–8.
139. Schepers T. Acute distal tibiofibular syndesmosis injury: a systematic review of suture-button versus syndesmotic screw repair. Int Orthop. 2012;36(6):1199–206.
140. van den Bekerom MP, Hogervorst M, Bolhuis HW, van Dijk CN. Operative aspects of the syndesmotic screw: review of current concepts. Injury. 2008;39(4):491–8.
141. Schepers T. To retain or remove the syndesmotic screw: a review of literature. Arch Orthop Trauma Surg. 2011;131(7):879–83.
142. Han SH, Lee JW, Kim S, Suh JS, Choi YR. Chronic tibiofibular syndesmosis injury: the diagnostic efficiency of magnetic resonance imaging and comparative analysis of operative treatment. Foot Ankle Int. 2007;28(3):336–42.
143. Pritsch M, Lokiec F, Sali M, Velkes S. Adhesions of distal tibiofibular syndesmosis. A cause of chronic ankle pain after fracture. Clin Orthop Relat Res. 1993;289:220–2.
144. Sikka RS, Fetzer GB, Sugarman E, et al. Correlating MRI findings with disability in syndesmotic sprains of NFL players. Foot Ankle Int. 2012;33(5):371–8.
145. Kaukonen JP, Lamberg T, Korkala O, Pajarinen J. Fixation of syndesmotic ruptures in 38 patients with a malleolar fracture: a randomized study comparing a metallic and a bioabsorbable screw. J Orthop Trauma. 2005;19(6):392–5.
146. Orr JD, Dawson LK, Garcia EJ, Kirk KL. Incidence of osteochondral lesions of the talus in the United States military. Foot Ankle Int. 2011;32(10):948–54.
147. Orr JD, Dutton JR, Fowler JT. Anatomic location and morphology of symptomatic, operatively treated osteochondral lesions of the talus. Foot Ankle Int. 2012;33(12):1051–7.
148. Mei-Dan O, Carmont MR, Laver L, Mann G, Maffulli N, Nyska M. Platelet-rich plasma or hyaluronate in the management of osteochondral lesions of the talus. Am J Sports Med. 2012;40(3):534–41.

149. Leumann A, Valderrabano V, Wiewiorski M, Barg A, Hintermann B, Pagenstert G. Bony periosteum-covered iliac crest plug transplantation for severe osteochondral lesions of the talus: a modified mosaicplasty procedure. Knee Surg Sports Traumatol Arthrosc. 2013 Jul 13. [Epub ahead of print] PMID:23851923.
150. Angthong C, Yoshimura I, Kanazawa K, et al. Critical three-dimensional factors affecting outcome in osteochondral lesion of the talus. Knee Surg Sports Traumatol Arthrosc. 2013;21(6):1418–26.
151. Lee KT, Park YU, Kim JS, Kim JB, Kim KC, Kang SK. Long-term results after modified Brostrom procedure without calcaneofibular ligament reconstruction. Foot Ankle Int. 2011;32(2):153–7.
152. de Jonge S, van den Berg C, de Vos RJ, et al. Incidence of midportion Achilles tendinopathy in the general population. Br J Sports Med. 2011;45(13):1026–8.
153. Maffulli N, Khan KM, Puddu G. Overuse tendon conditions: time to change a confusing terminology. Arthroscopy. 1998;14(8):840–3.
154. Hutchison AM, Evans R, Bodger O, et al. What is the best clinical test for Achilles tendinopathy? Foot Ankle Surg. 2013;19(2):112–7.
155. Robinson JM, Cook JL, Purdam C, et al. The VISA-A questionnaire: a valid and reliable index of the clinical severity of Achilles tendinopathy. Br J Sports Med. 2001;35(5):335–41.
156. Ohberg L, Lorentzon R, Alfredson H. Good clinical results but persisting side-to-side differences in calf muscle strength after surgical treatment of chronic Achilles tendinosis: a 5-year follow-up. Scand J Med Sci Sports. 2001;11(4):207–12.
157. Andersson G, Danielson P, Alfredson H, Forsgren S. Nerve-related characteristics of ventral paratendinous tissue in chronic Achilles tendinosis. Knee Surg Sports Traumatol Arthrosc. 2007;15(10):1272–9.
158. Alfredson H, Spang C, Forsgren S. Unilateral surgical treatment for patients with midportion Achilles tendinopathy may result in bilateral recovery. Br J Sports Med. 2012 Nov 28. [Epub ahead of print] PMID:23193327 doi:10.1136/bjsports-2012-091399.
159. van der Plas A, de Jonge S, de Vos RJ, et al. A 5-year follow-up study of Alfredson's heel-drop exercise programme in chronic midportion Achilles tendinopathy. Br J Sports Med. 2012;46(3):214–8.
160. Silbernagel KG, Brorsson A, Lundberg M. The majority of patients with Achilles tendinopathy recover fully when treated with exercise alone: a 5-year follow-up. Am J Sports Med. 2011; 39(3):607–13.
161. Paoloni JA, Appleyard RC, Nelson J, Murrell GA. Topical glyceryl trinitrate treatment of chronic noninsertional Achilles tendinopathy. A randomized, double-blind, placebo-controlled trial. J Bone Joint Surg Am. 2004;86-A(5):916–22.
162. Paoloni JA, Murrell GA. Three-year followup study of topical glyceryl trinitrate treatment of chronic noninsertional Achilles tendinopathy. Foot Ankle Int. 2007;28(10):1064–8.
163. Kane TP, Ismail M, Calder JD. Topical glyceryl trinitrate and noninsertional Achilles tendinopathy: a clinical and cellular investigation. Am J Sports Med. 2008;36(6):1160–3.
164. Al-Abbad H, Simon JV. The effectiveness of extracorporeal shock wave therapy on chronic achilles tendinopathy: a systematic review. Foot Ankle Int. 2013;34(1):33–41.
165. de Jonge S, de Vos RJ, Weir A, et al. One-year follow-up of platelet-rich plasma treatment in chronic Achilles tendinopathy: a double-blind randomized placebo-controlled trial. Am J Sports Med. 2011;39(8):1623–9.
166. Owens Jr RF, Ginnetti J, Conti SF, Latona C. Clinical and magnetic resonance imaging outcomes following platelet rich plasma injection for chronic midsubstance Achilles tendinopathy. Foot Ankle Int. 2011;32(11):1032–9.
167. Tol JL, Spiezia F, Maffulli N. Neovascularization in Achilles tendinopathy: have we been chasing a red herring? Knee Surg Sports Traumatol Arthrosc. 2012;20(10):1891–4.
168. Sengkerij PM, de Vos RJ, Weir A, van Weelde BJ, Tol JL. Interobserver reliability of neovascularization score using power Doppler ultrasonography in midportion Achilles tendinopathy. Am J Sports Med. 2009;37(8):1627–31.
169. Humphrey J, Chan O, Crisp T, et al. The short-term effects of high volume image guided injections in resistant non-insertional Achilles tendinopathy. J Sci Med Sport. 2010;13(3):295–8.
170. Lind B, Ohberg L, Alfredson H. Sclerosing polidocanol injections in mid-portion Achilles tendinosis: remaining good clinical results and decreased tendon thickness at 2-year follow-up. Knee Surg Sports Traumatol Arthrosc. 2006;14(12):1327–32.
171. Longo UG, Ramamurthy C, Denaro V, Maffulli N. Minimally invasive stripping for chronic Achilles tendinopathy. Disabil Rehabil. 2008; 30(20–22):1709–13.
172. Maffulli N, Testa V, Capasso G, Bifulco G, Binfield PM. Results of percutaneous longitudinal tenotomy for Achilles tendinopathy in middle- and long-distance runners. Am J Sports Med. 1997;25(6):835–40.
173. Testa V, Capasso G, Benazzo F, Maffulli N. Management of Achilles tendinopathy by ultrasound-guided percutaneous tenotomy. Med Sci Sports Exerc. 2002;34(4):573–80.
174. Maffulli N, Oliva F, Testa V, Capasso G, Del Buono A. Multiple percutaneous longitudinal tenotomies for chronic Achilles tendinopathy in runners: a long-term study. Am J Sports Med. 2013;41(9):2151–7.
175. van Sterkenburg MN, Kerkhoffs GM, Kleipool RP, Niek van Dijk C. The plantaris tendon and a potential role in mid-portion Achilles tendinopathy: an observational anatomical study. J Anat. 2011; 218(3):336–41.
176. Alfredson H. Midportion Achilles tendinosis and the plantaris tendon. Br J Sports Med. 2011;45(13):1023–5.

177. Pearce CJ, Carmichael J, Calder JD. Achilles tendinoscopy and plantaris tendon release and division in the treatment of non-insertional Achilles tendinopathy. Foot Ankle Surg. 2012;18(2):124–7.
178. Schon LC, Shores JL, Faro FD, Vora AM, Camire LM, Guyton GP. Flexor hallucis longus tendon transfer in treatment of Achilles tendinosis. J Bone Joint Surg Am. 2013;95(1):54–60.
179. Miyamoto W, Takao M, Matsushita T. Reconstructive surgery using autologous bone-patellar tendon graft for insertional Achilles tendinopathy. Knee Surg Sports Traumatol Arthrosc. 2012;20(9):1863–7.
180. Maffulli N, Spiezia F, Longo UG, Denaro V. Less-invasive reconstruction of chronic Achilles tendon ruptures using a peroneus brevis tendon transfer. Am J Sports Med. 2010;38(11):2304–12.
181. Maffulli N, Spiezia F, Pintore E, et al. Peroneus brevis tendon transfer for reconstruction of chronic tears of the Achilles tendon: a long-term follow-up study. J Bone Joint Surg Am. 2012;94(10):901–5.
182. Elias I, Raikin SM, Besser MP, Nazarian LN. Outcomes of chronic insertional Achilles tendinosis using FHL autograft through single incision. Foot Ankle Int. 2009;30(3):197–204.
183. 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.
184. Nyyssonen T, Luthje P, Kroger H. The increasing incidence and difference in sex distribution of Achilles tendon rupture in Finland in 1987-1999. Scand J Surg. 2008;97(3):272–5.
185. Aroen A, Helgo D, Granlund OG, Bahr R. Contralateral tendon rupture risk is increased in individuals with a previous Achilles tendon rupture. Scand J Med Sci Sports. 2004;14(1):30–3.
186. Cetti R, Christensen SE, Ejsted R, Jensen NM, Jorgensen U. Operative versus nonoperative treatment of Achilles tendon rupture. A prospective randomized study and review of the literature. Am J Sports Med. 1993;21(6):791–9.
187. Olsson N, Silbernagel KG, Eriksson BI, et al. Stable surgical repair with accelerated rehabilitation versus nonsurgical treatment for acute Achilles tendon ruptures: a randomized controlled study. Am J Sports Med. 2013;41:2867–76.
188. Keating JF, Will EM. Operative versus non-operative treatment of acute rupture of tendo Achillis: a prospective randomised evaluation of functional outcome. J Bone Joint Surg Br. 2011;93(8):1071–8.
189. Khan RJ, Fick D, Keogh A, Crawford J, Brammar T, Parker M. Treatment of acute Achilles tendon ruptures. A meta-analysis of randomized, controlled trials. J Bone Joint Surg Am. 2005;87(10):2202–10.
190. Jiang N, Wang B, Chen A, Dong F, Yu B. Operative versus nonoperative treatment for acute Achilles tendon rupture: a meta-analysis based on current evidence. Int Orthop. 2012;36(4):765–73.
191. McMahon SE, Smith TO, Hing CB. A meta-analysis of randomised controlled trials comparing conventional to minimally invasive approaches for repair of an Achilles tendon rupture. Foot Ankle Surg. 2011;17(4):211–7.
192. Carmont MR, Maffulli N. Less invasive Achilles tendon reconstruction. BMC Musculoskelet Disord. 2007;8:100.
193. Bergkvist D, Astrom I, Josefsson PO, Dahlberg LE. Acute Achilles tendon rupture: a questionnaire follow-up of 487 patients. J Bone Joint Surg Am. 2012;94(13):1229–33.
194. Metz R, van der Heijden GJ, Verleisdonk EJ, Kolfschoten N, Verhofstad MH, van der Werken C. Effect of complications after minimally invasive surgical repair of acute Achilles tendon ruptures: report on 211 cases. Am J Sports Med. 2011;39(4):820–4.
195. Eleftheriou KI, Rosenfeld PF, Calder JD. Lisfranc injuries: an update. Knee Surg Sports Traumatol Arthrosc. 2013;21(6):1434–46.
196. Nunley JA, Vertullo CJ. Classification, investigation, and management of midfoot sprains: Lisfranc injuries in the athlete. Am J Sports Med. 2002;30(6):871–8.
197. Bowers Jr KD, Martin RB. Turf-toe: a shoe-surface related football injury. Med Sci Sports. 1976;8(2):81–3.
198. Rodeo SA, O'Brien S, Warren RF, Barnes R, Wickiewicz TL, Dillingham MF. Turf-toe: an analysis of metatarsophalangeal joint sprains in professional football players. Am J Sports Med. 1990;18(3):280–5.
199. Kaplan LD, Jost PW, Honkamp N, Norwig J, West R, Bradley JP. Incidence and variance of foot and ankle injuries in elite college football players. Am J Orthop (Belle Mead NJ). 2011;40(1):40–4.
200. Frimenko RE, Lievers W, Coughlin MJ, Anderson RB, Crandall JR, Kent RW. Etiology and biomechanics of first metatarsophalangeal joint sprains (turf toe) in athletes. Crit Rev Biomed Eng. 2012;40(1):43–61.
201. Waldrop 3rd NE, Zirker CA, Wijdicks CA, Laprade RF, Clanton TO. Radiographic evaluation of plantar plate injury: an in vitro biomechanical study. Foot Ankle Int. 2013;34(3):403–8.
202. Jones RI. Fracture of the base of the fifth metatarsal bone by indirect violence. Ann Surg. 1902;35(6):697–700.692.
203. Ekstrand J, Torstveit MK. Stress fractures in elite male football players. Scand J Med Sci Sports. 2012;22(3):341–6.
204. Torg JS, Balduini FC, Zelko RR, Pavlov H, Peff TC, Das M. Fractures of the base of the fifth metatarsal distal to the tuberosity. Classification and guidelines for non-surgical and surgical management. J Bone Joint Surg Am. 1984;66(2):209–14.
205. Carreira DS, Sandilands SM. Radiographic factors and effect of fifth metatarsal Jones and diaphyseal stress fractures on participation in the NFL. Foot Ankle Int. 2013;34(4):518–22.
206. Pecina M, Bojanic I, Smoljanovic T, Ivkovic A, Mirkovic M, Jelic M. Surgical treatment of diaphyseal stress fractures of the fifth metatarsal in competitive athletes: long-term follow-up and

computerized pedobarographic analysis. J Am Podiatr Med Assoc. 2011;101(6):517–22.
207. Sarimo J, Rantanen J, Orava S, Alanen J. Tension-band wiring for fractures of the fifth metatarsal located in the junction of the proximal metaphysis and diaphysis. Am J Sports Med. 2006;34(3):476–80.
208. Hulkko A, Orava S, Nikula P. Stress fracture of the fifth metatarsal in athletes. Ann Chir Gynaecol. 1985;74(5):233–8.
209. Lee KT, Park YU, Young KW, Kim JS, Kim JB. Surgical results of 5th metatarsal stress fracture using modified tension band wiring. Knee Surg Sports Traumatol Arthrosc. 2011;19(5):853–7.
210. Ekstrand J, van Dijk CN. Fifth metatarsal fractures among male professional footballers: a potential career-ending disease. Br J Sports Med. 2013;47(12):754–8.
211. 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(6):1307–15.
212. Hunt KJ, Anderson RB. Treatment of Jones fracture nonunions and refractures in the elite athlete: outcomes of intramedullary screw fixation with bone grafting. Am J Sports Med. 2011;39(9):1948–54.
213. Porter DA, Rund AM, Dobslaw R, Duncan M. Comparison of 4.5- and 5.5-mm cannulated stainless steel screws for fifth metatarsal Jones fracture fixation. Foot Ankle Int. 2009;30(1):27–33.
214. Porter DA, Duncan M, Meyer SJ. Fifth metatarsal Jones fracture fixation with a 4.5-mm cannulated stainless steel screw in the competitive and recreational athlete: a clinical and radiographic evaluation. Am J Sports Med. 2005;33(5):726–33.
215. Ritchie JD, Shaver JC, Anderson RB, Lawrence SJ, Mair SD. Excision of symptomatic nonunions of proximal fifth metatarsal avulsion fractures in elite athletes. Am J Sports Med. 2011;39(11):2466–9.
216. O'Malley MJ, Hamilton WG, Munyak J. Fractures of the distal shaft of the fifth metatarsal. "Dancer's fracture". Am J Sports Med. 1996;24(2):240–3.
217. Aynardi M, Pedowitz DI, Saffel H, Piper C, Raikin SM. Outcome of nonoperative management of displaced oblique spiral fractures of the fifth metatarsal shaft. Foot Ankle Int. 2013;34:1619–23.
218. Thordarson DB, Kumar PJ, Hedman TP, Ebramzadeh E. Effect of partial versus complete plantar fasciotomy on the windlass mechanism. Foot Ankle Int. 1997;18(1):16–20.
219. Healey K, Chen K. Plantar fasciitis: current diagnostic modalities and treatments. Clin Podiatr Med Surg. 2010;27(3):369–80.
220. Roberts WO. Plantar fascia injection. Phys Sportsmed. 1999;27(9):101–2.
221. Jarde O, Diebold P, Havet E, Boulu G, Vernois J. Degenerative lesions of the plantar fascia: surgical treatment by fasciectomy and excision of the heel spur. A report on 38 cases. Acta Orthop Belg. 2003;69(3):267–74.
222. Foster TE, Puskas BL, Mandelbaum BR, Gerhardt MB, Rodeo SA. Platelet-rich plasma: from basic science to clinical applications. Am J Sports Med. 2009;37(11):2259–72.
223. Riddle DL, Pulisic M, Pidcoe P, Johnson RE. Risk factors for Plantar fasciitis: a matched case-control study. J Bone Joint Surg Am. 2003;85-a(5):872–7.
224. Glazer JL. An approach to the diagnosis and treatment of plantar fasciitis. Phys Sportsmed. 2009;37(2):74–9.
225. Akfirat M, Sen C, Gunes T. Ultrasonographic appearance of the plantar fasciitis. Clin Imaging. 2003;27(5):353–7.
226. Ozdemir H, Ozdemir A, Soyucu Y, Urguden M. The role of bone scintigraphy in determining the etiology of heel pain. Ann Nucl Med. 2002;16(6):395–401.
227. Grasel RP, Schweitzer ME, Kovalovich AM, et al. MR imaging of plantar fasciitis: edema, tears, and occult marrow abnormalities correlated with outcome. AJR Am J Roentgenol. 1999;173(3):699–701.
228. Gill LH. Plantar fasciitis: diagnosis and conservative management. J Am Acad Orthop Surg. 1997;5(2):109–17.
229. Lee SY, McKeon P, Hertel J. Does the use of orthoses improve self-reported pain and function measures in patients with plantar fasciitis? A meta-analysis. Phys Ther Sport. 2009;10(1):12–8.
230. Acevedo JI, Beskin JL. Complications of plantar fascia rupture associated with corticosteroid injection. Foot Ankle Int. 1998;19(2):91–7.
231. Aksahin E, Dogruyol D, Yuksel HY, et al. The comparison of the effect of corticosteroids and platelet-rich plasma (PRP) for the treatment of plantar fasciitis. Arch Orthop Trauma Surg. 2012;132(6):781–5.

Head, Low-Back and Muscle Injuries in Athletes: PRP and Stem Cells in Sports-Related Diseases

19

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S. Zaffagnini et al. (eds.), *ESSKA Instructional Course Lecture Book*,
DOI 10.1007/978-3-642-53983-1_19, © ESSKA 2014

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19.1 Introduction

In this chapter, we will discuss some particular topics about sports medicine. In particular, we will focus on:

Head injury:
- *Skull fracture*
- *Facial injury*
- *Twitch on the pitch*
- *Concussion*

Low-back pain and sacroiliac joint disorders:
- *Strains and sprains*
- *Degenerative disc disease*
- *Disc herniation*
- *Spondylolysis and spondylolisthesis*
- *Posterior element overuse syndrome*
- *Sacroiliac joint dysfunction*

Muscle injuries:
- *Sites of injury*
- *Epidemiology*
- *Treatment*
- *Complication*
- *Injury prevention*

PRP and stem cells:
- *PRP*
- *Stem cells*

19.2 Head Injury

Head injury is an inherent component of combat/fighting sports such as boxing, karate and tae kwon do and may occur as a result of unintentional contact during sports such as rugby football and soccer. In other sports such as American football (NFL), the head may be used as a weapon to strike the opponent.

Concussion is thought to occur most commonly in girls soccer, American football and ice hockey [1]. Concussion comprised 33 % of the injuries sustained in amateur and professional boxers observed over a 12-month period [2]. In tae kwon do championships, the head and neck was identified as the second most common area injured with rates of 18.3/1,000 athlete exposures [3]. Concussion injury rates during professional rugby union matches were 4.1/1,000 player-hours, and most of these were sustained tackling head on (28 %), collisions (20 %) or being tackled head on (19 %) [4]. In international football, the incidence of all head and neck injuries was 12.5/1,000 player-hours with aerial challenges being the most common cause of injury (55 %) [5]. Head injury rates in collegiate American football are not directly comparable although head injuries occurred at a rate of 0.21 per 100,000 participants [6].

During the sport of horse racing, riders may both sustain falls from above head height onto the head and in addition suffer the risk of being kicked or trodden on by the horse. Professional horse racing suffered six deaths due to head injuries between 1975 and 2000. Concussion occurred in 1.8–2.7 per 100 falls from 1992 to 2000. In jump racing, 126 concussive injuries occurred per 100,000 rides [7].

The majority of this chapter involves standard pitch or event side and prehospital management, which are reproduced in texts and course manuals.

The initial assessment of head-injured athletes follows the same standard safe to approach with airway and consideration and control of the cervical spine, breathing with supplementary oxygen therapy, circulation assessment with control of exsanguination, disability assessment and exposure and environmental control.

The response of the player following the head injury may change rapidly, and so a careful repeated assessment is needed particularly with airway opening and the administration of supplementary oxygen therapy.

When assessing disability, the level of consciousness may be determined by using the AVPU scale [8]. These are graduated scales of consciousness based upon the patient's response to verbal and painful tactile stimuli. Limb movement can be noted at the point of injury together with looking at the player's eyes. The presence of movement and alteration of papillary size with time may represent intracranial haemorrhage and increased pressure over time. Noting these at the point of injury provides a baseline for future reference.

During the secondary survey, a more formal Glasgow Coma Scale can be determined [9]. As can be seen below, the GCS is complicated and difficult to remember; however, there are several key important thresholds. These are whether the player is confused or drowsy (GCS 13 and 14) or the patient is so deeply unconscious that they are unable to maintain their own airway (GCS ≤ 8) approximately equal to unresponsive on AVPU. Patients who are not verbalising may be considered to be unconscious.

The deterioration of patients on this scale may be more reliably noted than the finite score.

Glasgow Coma Scale		AVPU
Eyes		
Open spontaneously	4	Alert
Open to speech	3	Responds to voice
Open to painful stimulus	2	Responds to pain
Remain closed	1	Unresponsive
Voice		
Appropriate conversation	5	
Confused conversation	4	
Inappropriate words	3	
Incomprehensible sounds	2	
No response	1	
Pain		
Responds appropriately	6	
Localises to pain	5	
Flexes to pain	4	
Decorticate posturing	3	
Decerebrate posturing	2	
No response	1	

The management of confused players is difficult as they may not comply with simple advice and instructions. This can be a difficult area regarding cervical spine immobilisation as they have potential for a cervical spine injury at the time of their head injury and yet may not tolerate oxygen therapy or cervical spine immobilisation. Players who are combative should clearly not have their head immobilised for fear of causing iatrogenic damage. In practice, combative players should be treated with care to calm them, make them compliant and encourage them from the field of play with the consideration that a cervical spine injury may have occurred and immobilised once they have calmed to the extent that they can now be managed. A combative player is unlikely to have sustained an unstable cervical spine fracture. The greatest deformation and risk to the cord is likely to have occurred during injury. Protection against subsequent injury must be undertaken.

Event side providers are able to respond within seconds of a player sustaining a head injury. The majority of episodes of loss of consciousness are short lived, and by manually opening the airway, providing supplementary oxygen therapy with cervical spine immobilisation for a short period of time, the player rapidly recovers to an alert state in which their airway is no longer compromised. Remember not all head injuries result in a loss of consciousness and the player may present with an altered state of consciousness. These players then need to be transferred from the field of play with cervical spine precautions to allow a more thorough assessment to exclude concussion and cervical spine injury.

During a prolonged episode of unconsciousness, the patient's airway may be considered to be in jeopardy. Manual airway opening techniques or simple adjuncts can successfully maintain the airway for short periods of time; however, for prolonged unconsciousness, a definitive airway may be required. In these situations, the player will be transferred to the hospital for further care, treatment and investigation.

Careful observation and repeated assessment must be performed in any player who sustains a head injury. Intracranial bleeding has been noted to occur after minimal force [10] and may take some time for its effects to be realised. The bleeding may be either venous or arterial with rupture of the extradural middle meningeal artery following a skull fracture. The increase in pressure from the bleeding may present as delayed-onset drowsiness following a head injury after the player has recovered from the loss of consciousness associated with the initial impact. These patients have been described as 'talking and dying' on account of the lucid interval. Other signs of increased intracranial pressure include reduced pupil reflexes in response to light and a fixed dilated pupil due to third cranial nerve compression. The sluggish response upon shining a penlight into the pupil in the presence of an intracranial collection is caused by compression of the outer parasympathetic neural fibres of the oculomotor nerve with sparing of the central sympathetic nerves. Subdural accumulation is usually caused by the gradual collection of venous blood commonly presenting as drowsiness. Intracerebral haemorrhage may present with a focal neurological deficit. The distinction between these at the event side is academic, and it is vital to note deterioration early and seek expert help.

19.2.1 Skull Fracture

Typical signs of a skull fracture include the presence of a scalp haematoma, palpable step or deformity to the cranial vault; however, other less obvious signs may become apparent as time progresses. These include:

Visual changes including field defects and double vision/diplopia due to either bleeding within the occipital cortex or optic nerve compression. Diplopia due to orbital muscle dysfunction may also occur.

Bruising around the eyes in the absence of facial injury. This is commonly known as raccoon/panda eyes and together with a retro-auricular haematoma is a sign of skull fracture. However, this is a late sign and not usually seen pitchside.

A persistent clear discharge from the nose or ear. This is a leak of cerebrospinal fluid producing CSF rhinorrhoea or otorrhoea.

Hearing change due to blood accumulating behind the tympanic membrane: haemotympanum.

A facial muscle weakness could occur due to cranial nerve VII compression together with loss of facial nerve sensation. This could also occur with an inferior orbital blowout fracture.

19.2.2 Facial Injury

Clearly, any player sustaining a facial injury has received an injury above the clavicle and as such has the potential to both be concussed and have a cervical spine injury. Haemorrhage and fracture from facial injuries have the potential to place the patient's airway in jeopardy. Alert players with such injuries typically wish to sit forward so that any blood can be spat out or can drain from the mouth. In the primary assessment of these patients, airway management is the first concern with consideration of the cervical spine. If the patient wishes to sit forward, he may be considered to have head control. In this situation, the injured player will be unwilling to tolerate triple cervical spine immobilisation, and the application of this may be detrimental to their overall care. Once again, consideration that cervical spine injury may have occurred is required.

19.2.3 Twitch on the Pitch

It is common for players to suffer a short seizure or convulsive episode following a head injury. When this occurs in sport, it is commonly termed a 'twitch on the pitch'. These are benign, do not represent a form of epilepsy and usually settle after a few seconds. If this is still in progress by the arrival of the medical team, supplementary oxygen therapy can be provided. During this seizure activity, the cervical spine should be considered while the airway is maintained; however, the rigid immobilisation of the cervical spine during a convulsion may actually be detrimental.

Most seizure activity is short lived and will terminate within a few seconds particularly after an airway manoeuvre and the provision of supplementary oxygen. The Rugby Football League mandatory equipment list includes ampoules of diazepam for IV/rectal administration to stop seizures; however, this is primarily for prolonged epileptic seizures. Seizure activity relating to a head injury is likely to spontaneously terminate particularly with oxygen therapy. Again the straight pointing of an upper or lower limb, called a 'fencing response', may be indicative of a head injury and may allow recognition from afar. A brief loss of consciousness and impact seizures do not reliably predict outcomes following concussion although a cautious approach should be adopted following their occurrence [11].

19.2.4 Concussion

Over the past decade, there have been advances in the understanding and terminology of concussion; however, concussion is considered by many to be the most complex injury in sports medicine to diagnose, assess and manage. Experts in concussion research have met at three international conferences to produce consensus statements and guidelines for the management of concussion. The first of these was in Vienna in 2001 [12], the second in Prague in 2004 [13] and the most recent in Zurich in 2008 [14]. At each meeting, the groups have appraised and modified their initial statements in light of advances in research. A fourth symposium convened in Zurich in November 2012 [15]. The American Medical Society for Sports Medicine and the American Academy of Neurology have recently produced guidelines for the diagnosis and treatment of concussion in sport [16, 17]. The American Medical Society for Sports Medicine reviewed the literature and made recommendations based upon the Strength of Recommendation Taxonomy (SORT). The American Academy of Neurology performed a systematic review based upon a Grading of Recommendations, Assessment, Development and Evaluation (GRADE) based upon evidence-based methodology and a subsequent Delphi process. The distinction of the Zurich Consensus working group is that the group provided recommendations for all aspects of concussion management even if there is little or no evidence.

West and Marion have recently compared these three published guidelines. Despite the different methodologies used [1], key similar guidelines have been produced in each case, and these will be discussed below.

> Concussion is defined as a complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces. Several common features that incorporate clinical, pathologic and biomechanical injury constructs that may be utilised in defining the nature of a concussive head injury include:
>
> Concussion may be caused either by a direct blow to the head, face, neck or elsewhere on the body with an "impulsive" force transmitted to the head.
>
> Concussion typically results in the rapid onset of short-lived impairment of neurologic function that resolves spontaneously.
>
> Concussion may result in neuropathological changes but the acute clinical symptoms largely reflect a functional disturbance rather than a structural injury.
>
> Concussion results in a graded set of clinical symptoms that may or may not involve loss of consciousness. Resolution of the clinical and cognitive symptoms typically follows a sequential course; however it is important to note that in a small percentage of cases however post concussive symptoms may be prolonged.
>
> No abnormality on standard structural neuroimaging studies is seen on concussion.

The consensus groups have also given guidelines on the on-field or sideline evaluation of acute concussion. The key principles are based upon the recognition of injury, assessment of symptoms, cognitive and cranial nerve function and balance tests. It is recommended that when a player shows any features of a concussion:

The player should be medically evaluated onsite using standard emergency management principles, and particular attention should be given to excluding a cervical spine injury.

The appropriate disposition of the player must be determined by the treating health-care provider in a timely manner. If no health-care provider is available, the player should be safely removed from practice or play and urgent referral to a physician or emergency department arranged.

Once the first aid issues are addressed, then an assessment of the concussive injury should be made using the Sport Concussion Assessment Tool 3 (SCAT3) or other similar tool. Comparison with a preseason baseline is advantageous.

The player should not be left alone following the injury, and serial monitoring for deterioration is essential over the initial few hours following injury.

A player with diagnosed concussion should not be allowed to return to play on the day of the injury.

Players are well aware that if they are concussed they will be removed from the field of play and so may not complain of symptoms. Any player therefore sustaining a head injury should be carefully observed for the signs of concussion.

Observable signs include behavioural changes and cognitive impairment, as demonstrated by them lacking their usual clarity of thinking while performing their sport.

The pitchside assessment of concussion may be formed by brief neuropsychological test batteries assessing attention and memory function. These tests include the commonly used Maddocks questions [18] and the Standardized Assessment of Concussion. Standard orientation questions have been shown to be unreliable in the sporting situation when compared to memory assessment. The Maddocks questions are more relevant to athletes during a game and more specific than standard orientation questions. The use of the SCAT3 card and Pocket SCAT3 will help determine whether a player has been concussed and give good advice following a concussion.

Even in the presence of negative neuropsychological test results, the clinical impression of the attending sports physician is paramount. Knowledge of individual players and athletes, in particular to their mood, mannerisms and character, is much more important.

The Maddocks Questions:

Which ground are we at?

Which half is it?

Which side scored the last goal?

Which team did we play last week?

Did we win last week?

The player can then be removed from the field of play for assessment or substituted. In some sports, officials have the ability to remove players from the field of play following injury, irrespective of the player's or the coach's opinion.

In this respect, although a loss of consciousness has been acknowledged as a predictor of outcome in moderate to severe traumatic brain injury, it is not noted as a marker of severity for concussion. The most recent consensus meeting determined that prolonged (>1 min duration) LOC would be considered as a factor that may modify management. LOC is however a useful objective marker that a head injury has occurred and that the player is likely to be concussed. Thus, the consensus group recommends that those sustaining a loss of consciousness do not return to play. However, a player may still demonstrate concussive symptoms and need to be removed from the field of play, even if they have not sustained an LOC.

Asking patients about symptoms is facilitated by the use of a checklist as patients may be unable to recall if they have had the symptom or not.

It involves three balance tests, which should be performed on the SCAT3 form: the double-leg stance, the single-leg stance and the tandem stance.

The mainstay of concussion management involves physical and cognitive rest until all symptoms resolve and a graded programme of exertion prior to medical clearance and return to play.

The player should be observed during this period of rest and ensure they are well hydrated, eat regularly and take simple analgesics, e.g. paracetamol. In more severe cases, minimising cognitive input and maintaining a regular routine should be advised. It must be remembered that the effects of concussion can have delayed onset.

Patients should be referred to an emergency facility if they:

Have worsening headache

Become very drowsy or cannot be easily awakened

Cannot recognise people or places

Develop significant nausea or vomiting

Behave unusually more confused or irritable

Develop seizures

Develop weakness or numbness in the arms or legs

Develop slurred speech or unsteadiness of gait

The graded programme should typically take about a week following a concussion with 24 h required to complete each stage. The player/athlete should only progress a stage onwards if symptoms do not reoccur. The additional use of neuropsychological testing and the interpretation of the results are beyond the scope of this manual.

Since the guidelines in 2013, the consensus is that any player who has sustained or is suspected to have sustained a concussion may not return to play on the same day as injury. 'When in doubt, sit them out!' is a useful mantra to follow. From the work within the NFL, it must be noted that the majority of players sit out rather than the 15.2 % of players who return immediately and 39 % of those playing in high school and collegiate football admit to playing with residual symptoms from a prior head injury.

Nonelite athletes may not have the same resources available as elite athletes (such as the presence of trained medical staff during practice and competition, a concussion programme as part of sideline preparedness, the benefit of neuropsychological or postural testing as well as consultants with expertise readily available) and as a result tend to be managed more conservatively. Younger athletes often have a greater incidence of concussion with longer recovery time frames; however, they are often managed with less expertise and with limited resources. It is safer to not return these athletes to play on the same day and be more conservative with their management.

Legislation now mandates that any child or adolescent removed from the field of play or game with suspected concussion may not return. Unfortunately, data on this group of patients is limited.

Ten to fifteen percent of patients will have ongoing post-concussive symptoms for more than 10 days. This could be even higher in some sports typically ice hockey and some populations of children. It must be remembered that many of the symptoms of concussion are not specific to concussion and it is important to consider and manage coexistent pathologies. Further investigation is required consisting of formal neuropsychological and conventional neuroimaging to exclude structural pathology. There is insufficient evidence for the use of advanced neuroimaging or genetic markers. There is potential benefit of sub-symptom threshold activity as part of a comprehensive rehabilitation programme.

Patients should be managed in a multidisciplinary manner by health-care providers with experience in sports-related concussion. Important components of management after an initial period of physical and cognitive rest include associated therapies such as cognitive vestibular physical and psychological therapy assessment for other forms of prolonged symptoms and consideration of a graded exercise programme at a level that does not exacerbate symptoms [11].

The assessment of any player after a head injury should progress along the management principles of airway with consideration of the cervical spine, breathing, circulation with haemorrhage control, disability assessment and finally exposure and environmental control.

Assessment of concussion is complex and is facilitated with the use of the Standardised Assessment Concussion Tool (SCAT3).

Any player who is concussed or has symptoms of concussion should be removed from the field of play. They may only return once they are symptom-free at rest and on exertion and preferably after being reviewed by a physician or experienced clinician.

19.3 Low-Back Pain and Sacroiliac Joint Disorders

Not only sedentary individuals are afflicted by back pain; it has significant effects on athletes as well, but they differ from nonathletic population in their will to return to activity that may vary from will to win through significant financial considerations. Low-back pain represents one of the most common reasons for missed playing time by professional athletes.

Athletes are typically well conditioned in spite of low-back pain, because of greater flexibility of the lumbar spine and higher pain thresholds, but they have higher demand on their lumbar spine and cannot tolerate any kind of limitations on their activities.

Low-back pain is a common musculoskeletal disorder that is almost ubiquitary and that may be either acute or chronic. It is important to remember that it's a symptom, not a diagnosis. Pain could be related to problems with the lumbar spine, intervertebral disc, ligaments around spine and discs, spinal cord and nerves, paravertebral muscles, pelvic and abdominal organs or skin (Table 19.1); in most cases of low-back pain, an anatomical abnormality is not identifiable.

Accurate history and physical examination are essential for evaluating low-back pain in an

Table 19.1 Low-back pain in athletes: differential causes

Spinal pathologies	Nonspinal pathologies
Muscle strain/ligament sprain	Sacroiliac joint dysfunction
Degenerative disc disease	Intrapelvic, gynaecologic conditions (e.g. ovarian cysts)
Isthmic spondylolysis (no slip)	Renal disease
Isthmic spondylolisthesis	
Facet syndrome	
Ring apophyseal injury (adolescents)	
Sacral stress fracture	
Central disc herniation (without radiculopathy)	
Sacralisation of L5/transverse process impingement	
Facet stress fracture	
Acute traumatic lumbar fracture	
Discitis/osteomyelitis	
Neoplasm	

athlete. In case of acute symptoms, it is important to understand the mechanism of injury; otherwise, duration of symptoms, location and irradiation and rate of onset of symptoms must be investigated in case of chronic low-back pain. If pain is localised in the lower back, it's more probably a mechanical back pain, while if symptoms predominantly afflict the legs, it may be due to nerve compression/irritation.

Lifetime prevalence of low-back pain in the general adult population is estimated to be between 85 and 90 %. Back pain is common in competitive athletes, with an estimated prevalence ranging from 1 to 30 %, and represents a common cause of missing playing time. Low-back injuries are established to account among 10–15 % of all athletic injuries [19]. Low-back injuries most frequently involve soft tissue surrounding the spine but also intervertebral disc and pars interarticularis.

Pattern of low-back pain in young athletes is significantly different from those in adult athletes: a young athlete is more frequently affected by pars interarticularis injuries occurring up to 47 %, while disc-related disorders are quite uncommon; instead an adult athlete often suffers low-back pain due to degenerative conditions (48 %) or to unspecified causes [20]. In order to establish a proper diagnostic process and treatment, it's important to underline that common causes of low-back pain in athletes are different between ages [21], as shown in Table 19.2.

Prevalence of low-back pain was shown to be different depending on the practised sport: Granhed and Morelli [22] found a significantly higher prevalence of low-back pain in retired wrestlers and heavyweight lifters than in a control population. Other studies [19, 20] showed a different occurrence of low-back pain in football players (27 %), artistic gymnasts (50 %) and rhythmic gymnasts (86 %). Similarly, also other causes of low-back pain such as sacroiliac disorders show a higher prevalence in rowers [23].

Low-back pain can occur either from an acute traumatic injury, more commonly in athletes, or from a repetitive microtrauma that produces an overuse injury. It's immediately understandable that contact sports tend to produce acute injuries from high-energy impacts, whereas sports involving repetitive flexion, extension and torsion of the spine result in overuse injuries [20].

Large forces are produced in the lumbar spine region during various athletic manoeuvres; these forces act on the muscles and ligaments, on the intervertebral disc and on the posterior elements.

Mechanical strain on spinal ligaments leads to tears and produces pain. This pain leads to decreased motor unit recruitment and reduced activity due to fear of producing pain that produce muscle wasting and weakness. This condition creates a muscle imbalance that leads to further mechanical disruption and muscle wasting [24].

During sports, the intervertebral disc is always under load as a result of body weight and muscle activity; under certain conditions, these loads exceed the tolerance of the disc and may cause degenerative or traumatic changes (degenerative disc disease or disc herniation) that are represented by progressive disc dehydration and loss of proteoglycans in the nucleus; these changes reduce the ability of the nucleus to absorb loads and could decrease the space available for the

Table 19.2 Causes of low-back pain by age

Prepubescent	Adolescent	Adult	Elderly
Infection	Trauma	Discogenic	Osteoarthritis
Tumour or other malignancy	Spondylosis	Mechanical back pain	Spinal stenosis
Trauma	Hyperlordosis back pain	Unspecified	Discogenic
Developmental	Discogenic	Osteoarthritis	Medical cause

neural elements producing irritation or compression [19]. In this case, a long history of low-back pain is usually reported by the athletes, with or without radicular pain; over time, the radicular pain becomes more severe because initially the disc material pushes on the annulus, activating pain receptors, and then the disc material herniates and irritates or compresses nerve roots [24]. Other authors found that these changes are sports and training related; in particular, they found that lumbar flexion was the most influential factor in developing disc degeneration.

Many authors [25, 26] consider that pars interarticularis lesions arise from mechanical stress due to repetitive hyperextension and axial loading applied to posterior elements of the spine, and they believe that the increased rate of spondylolysis in athletes is related to increased forces acting on the lumbar spine during sport activities.

In a young athlete, growth cartilage and secondary ossification centres are particularly vulnerable to injury; these areas are susceptible to compression, distraction and torsion injury. An incomplete ossification of the posterior column could be present in the lower lumbar vertebrae, particularly L5, predisposing to spondylolytic stress fractures; the presence of an unknown spina bifida represents an additional risk factor for spondylolysis [26]. So a growing athlete may potentially be at higher injury risk, in particular in contact sports [20]. Vulnerability of the growing spine was shown by Goldstein et al. [27] in an MRI study of female gymnasts and swimmers: they found that back pain complaints were more common in gymnasts, with increased age and level of competition, than in swimmers that did not have such a repetitive loading on their spine.

As mentioned previously, the type of sport or activity and position played must be elicited

Fig. 19.1 Ileo-psoas abscess (*red circle*)

because it correlates with the prevalence of low-back pain [19, 20]. Also athletic background should be explored: volume of training and level of competition should be investigated, as well as any increase of volume or intensity of training because it could reveal a poor conditioning, an excessive or repetitive loading, an improper techniques or an inadequate equipment [20].

History of back pain should be investigated: onset and duration of symptoms should differentiate between acute trauma and overuse injuries. Pain quality, location and severity should be determined, as well as eliciting any associated neurological symptoms and aggravating factors. Previous episodes of low-back pain should be investigated: as reported by Greene et al. [28], athletes with previous history of low-back pain had three times the risk for subsequent episodes if compared to those with no history of pain.

It is also very important to consider other more sinister causes of back pain, such as infection (Figs. 19.1 and 19.2), tumours or inflammatory conditions, in particular if 'red flag' symptoms

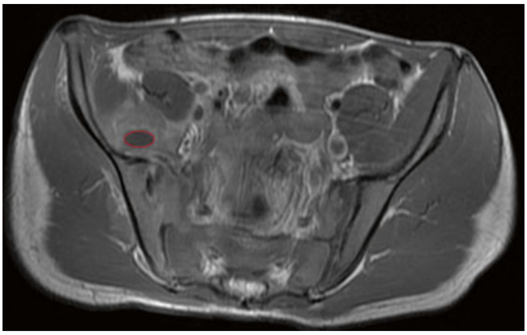

Fig. 19.2 Ileo-psoas abscess 2

Fig. 19.3 FABER test

such as fever, malaise, weight loss, neurological abnormalities, night pain and morning stiffness are present. Moreover, numerous conditions can affect bone metabolism contributing to stress fractures or metabolic bone pain: steroids (asthma, allergies), endocrinological dysfunctions (amenorrhoea, thyroid irregularities, disordered eating, illicit use of hormones) and chronic disease (inflammatory bowel, HIV) [20].

Physical examination should include inspection of the spine and also athlete's gait and posture; any ataxia, antalgic gait, limp or Trendelenburg gait should be noted.

Looking the patients from behind shoulders and pelvis should be aligned; bony and soft tissue should be symmetrical on both sides of the spine. Any abnormalities such as cysts, hairy patches, dimples, growth, haemangiomas or café au lait spots could be associated with congenital malformation (e.g. tethered cord).

Scoliosis, kyphosis or excess lordosis should also be noted: a gentle lumbar lordosis should be appreciable from the side. A forward-bending test should be performed to asses for any asymmetry suggestive of scoliosis.

Range of motion in all plans should be evaluated in flexion, extension, rotation and lateral bending. It's supposed that an athlete should be able to flex forward and come close to touch his toes without bending knees; tight hamstring can cause a reduction of forward flexion. If pain is evoked with flexion, an injury of the anterior elements of the spine is suggested or a muscle strain/spasm or a disc-related condition. Pain with extension indicates an involvement of the posterior elements or the sacroiliac joint; also a single-legged hyperextension test can be performed [20].

The spine and sacroiliac joint should be palpated looking for any tenderness point. Also paraspinal muscles and buttocks should be palpated for any tenderness or muscle spasm.

Examination of the lumbar spine should include special tests like figure-4 or FABER (flexion, abduction, external rotation) (Fig. 19.3) and Gaenslen sign (Fig. 19.4). If these tests provoke ipsilateral pain, this is suggestive of a sacroiliac joint pathology.

Neurological examination is necessary to complete investigation and should include deep tendon reflexes and lower extremity strength (heel and toe walking); during physical examination for low-back pain and radiculopathy, most useful tests are the straight leg raise (Fig. 19.5) and the cross-table straight leg raise; moreover, if used together [29], any abnormalities indicated a compression or irritation of nerves that could be originated from a degenerative disc disease or a disc herniation.

Any kind of imaging studies cannot substitute an accurate and complete physical examination. In selected patients with acute low-back pain and without signs or symptoms of an underlying serious condition, imaging studies are not indicated, as shown by several authors [24, 30], because they did not find any improvement in clinical outcome in these patients. Henschke et al. [30, 31] evaluate that in nonathletic patients even if some weaker 'red flags' are present, a 4–6-week period of treatment is suggested before any imaging study; but if an athlete suffers low-back pain,

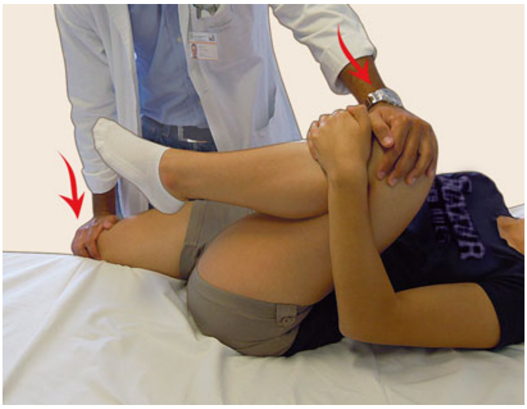

Fig. 19.4 Gaenslen sign

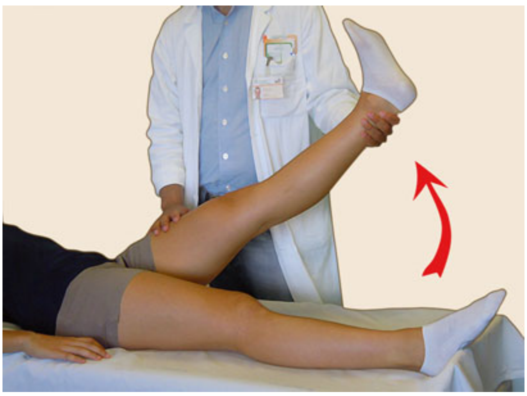

Fig. 19.5 Straight leg raise test

in particular if he is an elite athlete, a proper and rapid treatment is requested. So an imaging study, to avoid any delay in diagnosis and treatment and soon return to sport activity, is maybe suggested when this kind of patient is involved.

Plain radiographs in two projections could be useful for screen for serious conditions; because of low sensitivity and specificity, they had little diagnostic value but could provide information to rule out any structural abnormalities: oblique view allows evaluation of pars interarticularis, while flexion/extension radiographs assist in assessing dynamic instability.

MRI represents the gold standard for the lumbar spine and sacroiliac joint, in particular if neurological symptoms are present or a serious condition is suspected. An alternative could be represented by CT scan, if MRI is contraindicated or unavailable [30], but it's necessary to remember that they are different exams: MRI is more useful in evaluating discs, neural structures or other soft tissues and can also provide information regarding possible occult fractures or the presence of neoplastic disease, while CT helps define bony structures [32], and it should be reserved for confirming diagnosis or in case of significant trauma or to evaluate spinal canal impingement [24, 33].

Bone scan is very sensitive to rule out metabolic activity, such as with a neoplastic lesion, fractures of indeterminate age or spondylolytic defects. Scintigraphic tracer is abnormally accumulated where repetitive stress causes local bone remodelling [34].

A good clinician should never forget that any findings in MRI or computer tomography, or in any other imaging study, should be correlated with clinical findings, especially in older patients because the likelihood of false positives is increased [30]. Moreover, as shown by Graw and Wiesel [19], a 'nonspecific low-back pain' should be included in differential diagnosis: patients who suffer low-back pain could have no specific signs of any pathology on imaging studies, or they could present abnormalities that don't correlate with symptoms; instead, patients without low-back pain could present abnormalities on MRI nevertheless. These possibilities underline the importance of correlating imaging and clinical findings together; otherwise, the risks of incorrect diagnosis and under- or overtreatment increase.

The majority of low-back pain cases are self-limiting in the general population and resolve within 6 weeks regardless of treatment, but 5–10 % of patients will develop chronic back pain [30]. An athlete hardly accepts any kind of limitation on his sport activity, which can lead to a shortened career and financial loss, so an adequate treatment should be rapidly undertaken in order to return to play as soon as possible and reduce the possibility of developing chronic pain.

Although low-back pain is a common cause of inability in general population, there are few data in literature that analyse proper treatment and criteria for return to play when an athlete is involved.

19.3.1 Strains and Sprains

Strains and sprains are stretch injuries that involve respectively ligaments and muscles. These are quite common injuries in athletes that cause low-back pain, but diagnosis is usually made by exclusion.

Strains and sprains occur when loads exceed the resistance of involved structure. Typically, pain is acute and worst in the first 24–48 h and improves with time; chronic strains and sprains present a gradual onset of symptoms persisting for longer periods [32]. Muscle spasm is often present, and point of tenderness is generally localised; in particular, they are worsened by particular movements. Neurological examination doesn't show any abnormalities.

Acute strains and sprains could be simply diagnosed by acquiring accurate history of injury and performing physical examination. Any imaging study is not necessary, and the result is generally negative. When chronic strains and sprains occur, onset of symptoms is gradual and insidious, and pain persists for a longer period; in these cases, imaging studies are still generally negative, but they are used to rule out other pathologies [32].

These kinds of injuries represent the principal cause of low-back pain in athletes.

These are typically acute injuries, and patients report pain immediately consequent to the injury that increases in few hours (increased stiffness and pain the morning following the incident). Conservative treatment is the choice. Cryotherapy and heat offer benefits in decreasing spasm and pain; transcutaneous electric nerve stimulation and electric stimulation (high-voltage pulse galvanic stimulation) may be useful in the acute stages, but their efficacy has not been conclusively demonstrated [32].

A brief period of rest is needed: it should be limited to 1–2 days to prevent muscle atrophy that could lead to muscle imbalance and consequent lengthening of the period before restart of sport activity [24, 32].

Medications could be administrated; nonsteroidal anti-inflammatory drugs (NSAIDs) and COX-2 inhibitors are a good choice: in a large Cochrane review [35], they showed statistically better effects compared with placebo; NSAIDs have more side effects than COX-2 inhibitors.

Glucocorticosteroids are not more effective than placebo in acute injuries with straight leg raise test negative at 1 month [24]; moreover, they are banned by the World Antidoping Association.

Other drugs could be used. There is little evidence on the efficacy of muscle relaxants on low-back pain, and they have a high incidence of side effects (oversedation) that can hinder their use [36]. Also antidepressants could be administrated, but results are not consistent with all of them; the most effective antidepressant in reducing pain in chronic condition is selective serotonin reuptake inhibitor, in particular duloxetine [35].

These drugs have significantly higher adverse events: drowsiness, dry mouth, dizziness and constipation are the most common [35].

One trial comparing opioid to naproxen found that they were significantly better for relieving pain but not improving function. They commonly cause headaches and nausea; moreover, they are banned by the World Antidoping Association.

These initial interventions are followed by a physical therapy programme: as reported by Burns et al. [36], it would be started with direction-specific exercises; patients are invited to perform exercises or movements that decrease pain, and the primary goal is centralisation of symptoms (from distal to proximal). In another study Long et al. [37], 84 % of patients had significant reduction in pain and disability within the first 2 weeks of treatment.

When pain disappears or is at least reduced and centralised, the rehabilitation programme is continued with trunk, back and lower extremity strengthening, stretching exercises to restore function and then progressive return to sport [38]. In order to have better outcome results, physical therapy should be individualised and supervised [35].

To prevent recurrence of low-back pain, it is important that the athlete understands the importance of adherence to the rehabilitation programme and maintains a proper training; avoidance of overtraining is essential [36].

19.3.2 Degenerative Disc Disease

As in general population, degenerative disc disease is a segmental dysfunction: initially, pain originates from synovitis of facets or intervertebral disc (circumferential or radial annular tears); afterwards disc annular functionality, due to dehydration, is reduced and facet capsules become lax, influencing vertebral stability, and finally the degenerative process may be accompanied with listhesis and segment instability [19, 32].

History and physical examination are relatively nonspecific of degenerative disc disease. This pathology could be suggested by worsening of symptoms during flexion activities and improving with extension. So to diagnose degenerative disc disease, imaging studies are necessary.

Neural elements, including the spinal dura centrally and the nerve roots in the lateral recess, could be compressed by annulus fibrosus and by ligamentum flavum and facet joint capsule hypertrophy, with consequent symptoms irradiated to lower limb [19].

Plain radiograph may demonstrate an indirect sign of disc degenerative changes represented by loss of disc space height. MRI (Fig. 19.6) is the gold standard in demonstrating degeneration of the disc: loss of disc hydration on T2-weighted images on sagittal plane is the typical sign of degenerated disc [32]. According to Pfirrmann's classification [39] based on the evaluation of disc homogeneity, height, signal intensity and distinction between the nucleus and annulus, there are 5 MRI degrees of lumbar intervertebral disc degeneration; de Schepper et al. [40] showed how low-back pain is strongly associated with disc space narrowing more than other radiographic features, in particular if more spaces are involved.

Considering athletic population, several studies [32] found a significantly higher prevalence of disc signal changes in athletes than in nonathletes, confirming the highest demand on the spine of athletes.

Discogenic low-back pain should be initially treated nonsurgically. Resolution of acute symptoms generally occurs within weeks [32].

First of all, abstinence from practice and competition is recommended; the period of inactivity should be balanced against the risk of losses in trunk muscle and general fitness [24].

Fig. 19.6 DDD RMN

Pain relief is achieved with the administration of NSAIDs with or without muscle relaxants, in relation with muscle spasm presence. In consideration of symptoms and possible side effects, other drugs could be used, as previously described [24, 35].

The use of lumbosacral corset or orthoses is still controversial: although it may offer benefits in controlling extremes of motion, muscle wasting can occur [30].

After the initial period of rest, patients should be encouraged to regain mobility through passive and active stretching exercises, followed by isometric exercises, in particular emphasising abdominal muscles and lumbar extensors. Once neutral position could be attained and maintained, muscle strengthening is encouraged to gain greater strength. Then, sport-specific exercise is emphasised to hasten return to sport and prevent recurrence [32].

If conservative treatment doesn't obtain pain relief or recurrences are frequent, surgery should be considered. The choice of spinal fusion is not optimal for an athlete, because the loss of function of one segment should be compensated by other segments leading to an overloading and increasing the risk of degenerative changes to those levels; moreover, the lengthy postoperative period is poorly tolerated by most athletes [41]. Possibility of return to play following a spinal fusion is related to the level and the type of sport: most authors suggest to return to play in limited contact sports at least 1 year after fusion; full contact sports or highly competitive is much less likely, especially if 2 or more levels are involved, due to increased risk of subsequent injury to the adjacent levels [24].

A new surgical option is represented by total disc replacement (TDR). Some authors conducted a randomised trial comparing lumbar fusion and TDR: both groups showed significant improvements after surgery, but they were greater in the TDR group; at 2-year follow-up, there were no more differences between groups. Siepe et al. [42] reviewed 39 athletes who underwent TDR: return to play rate was 94.9 %, and 9 of 12 professional or elite athletes returned to play at competitive level and reported they achieved full recovery and peak fitness at an average of 5.2 months postoperatively. Tumialan et al. [43] compared 12 military patients who underwent TDR to an age- and level-matched cohort who underwent lumbar fusion: 83 % of TDR group were able to return to unrestricted full duty at an average of 22.6 weeks, while in the fusion group only the 67 % return to full duty at an average of 32.4 weeks.

These studies showed a better outcome at short follow-up of TDR if compared to spinal fusion. Longer follow-up is still not available, but these promising results lead to the possibility of employing total disc replacement in athletes.

19.3.3 Disc Herniation

Disc herniation is due to the escape of nuclear material from the annulus fibrosus. It is often related to peripheral annulus injury associated with traumatic disruption; initially, only low-back pain could be present that could progress to radicular symptoms if irritation or compression of surrounding neurological structures is present.

Patients with disc herniation present radicular symptoms: most common levels involved are L4–L5 and L5–S1. Pain is worsened by flexion and Valsalva manoeuvre and improved by lying supine.

Motor and sensitive tests are mandatory: if L5 root is involved, weakness of ankle and toe dorsiflexion could be noted, and sensory changes may be experienced on the lateral aspect of the lower leg and middorsum of the foot; if S1 root is involved, weakness in ankle eversion and plantar flexion, sensory changes on the lateral aspect of the foot and a decrease in Achilles tendon reflex should be present. Tests to investigate radiculopathy, such as straight leg raise test, are typically positive.

Cauda equina syndrome, caused by the compression of the nerves in the lower portion of the spinal canal, is uncommon. Usually, it presents with the characteristic findings of saddle paresthesia, loss of bowel and bladder function [20].

X-rays could be useful to rule out other pathologies. MRI (Figs. 19.7 and 19.8) represents the study of choice because it can demonstrate the herniation and its extension and moreover can show any nerve root compression [20]. MRI could be oversensitive for disc herniation, so it's important to correlate with clinical symptoms [30].

As shown by Hsu et al. [44], 82 % of athletes that suffered lumbar disc herniation successfully returned to play regardless of treatment: performance scores within each group were equal before and after treatment, either conservative or surgical. Anakwenze et al. [45] compared NBA players who underwent lumbar discectomy to a control group matched for experience, position, age and BMI who did not require surgery and found that both groups achieved the same level of performance.

As shown by these results, it's initially suggested to treat patients conservatively, including limited rest, activity modification, NSAIDs and

Fig. 19.7 EDD sagittal

exercises (extension and isometric exercises are performed first, and when sufficient strength and pain relief are achieved, flexion exercises are begun). Surgery is needed only if severe or progressive neurological deficit is present and if pain is refractory to conservative care. As long as patient shows improvements, surgery is deferred [24].

Microdiscectomy is widely recommended in athletes with lumbar disc herniation, because of less invasiveness and less disruption to muscles, bone and neural structure.

Wang et al. [46] showed that patients who underwent single-level microdiscectomy returned

Fig. 19.8 EDD axial

to play in 90 % of cases, whereas multiple-level microdiscectomy showed the worst rate of returning to play.

In literature, all authors emphasise the importance of postoperative physical therapy and rehabilitation; Watkins et al. [47] permitted professional and Olympic athletes who underwent lumbar microdiscectomy once they complete truck stabilisation programme, achieve an excellent aerobic condition and perform sport-specific stretching and strengthening exercises; Eck and Riley [24] allow return to sport once sufficient pain relief and range of motion are achieved.

Typically, 6–8 weeks are needed to return to noncontact sports and 4–6 months to contact sports postoperatively [41].

Spinal fusion is recommended for cases of multiple recurrences or if spinal instability coexists; this procedure increases the risk of degenerative changes and injury to adjacent levels; moreover, the ability to return to sport is related to level of play and type of sport involved, as previously said for disc degenerative disease.

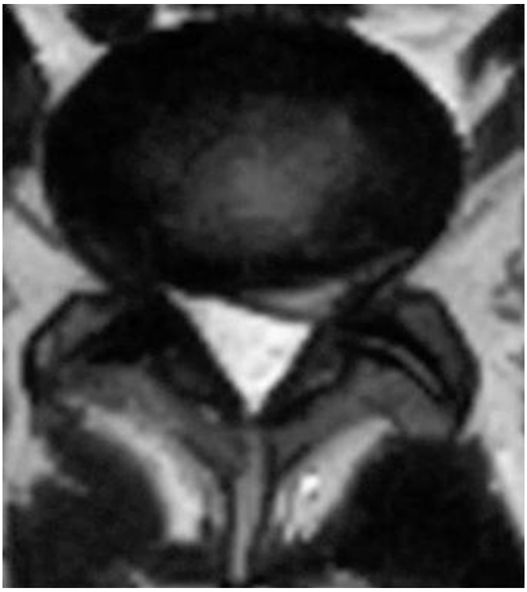

Fig. 19.9 Single-legged hyperextension test

Fig. 19.10 Spondylolisthesis rx

19.3.4 Spondylolysis and Spondylolisthesis

Spondylolysis is defined as a defect of the pars interarticularis; sometimes a forward slipping of one vertebra on another (spondylolisthesis) is associated. Spondylotic spondylolisthesis is the most common in athletes, interesting in most cases L5 and sometimes L4.

Spondylolysis is classified in four groups: dysplastic, developmental, traumatic (acute or chronic) and pathological. Typically, athletes suffer from spondylolysis due to a stress fracture (chronic traumatic) [38].

Risk of injury of the posterior elements is higher in sports involving repetitive extension and rotation of the lumbar spine: dancers, figure skaters, gymnasts and football linemen [32].

Insidious onset of low-back pain, in particular if extension related, is typical in athletes with spondylolysis and often associated with hamstring flexibility reduction that produces gait described as 'stiff-legged'. Pain is enhanced by spine extension: single-legged hyperextension test (Fig. 19.9) can localise spondylolysis when standing on the ipsilateral leg [32].

Pain irradiation to lower limb, with numbness or weakness, is occasionally present; in this case, differential diagnosis with disc herniation should be considered [20].

Classical sign of spondylolysis on radiographs is a radiolucent defect in the pars interarticularis: in an acute injury, edges are irregular and gap is narrow, whereas in a chronic injury, lesions are smooth and rounded [48]. When the defect is large, on radiographs, in particular on oblique view, diagnosis is easy (the 'Scotty Dog' visual is a useful aid, with the defect appearing at the dog's neck that represents the pars interarticularis); however, if the defect is narrow, spondylolysis could be missed, because a 45° oblique view demonstrates a pars defect well only if it is perpendicular to the pars [38].

When forward displacement of the vertebra on the other occurs, spondylolisthesis is diagnosed on lateral view radiograph (Fig. 19.10) and classified according to the percentage of anterior slippage (Meyerding system). If spondylolisthesis is suspected with normal static radiographs, flexion and extension views should be obtained: a dynamic translation greater than 3.0 mm is considered abnormal, suggesting spinal instability [24].

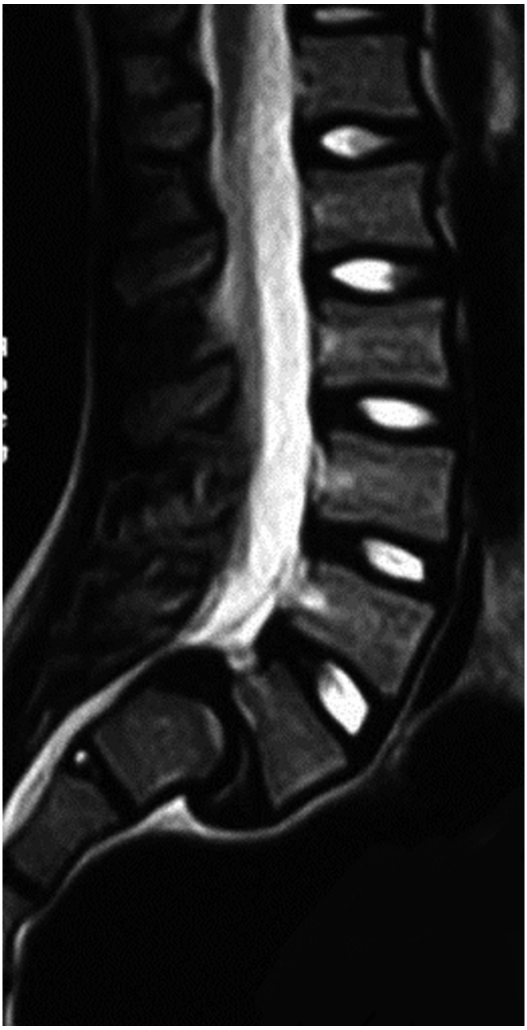

Fig. 19.11 Spondylolisthesis RMN

On axial CT scans, an unclosed neural arch is diagnostic of spondylolysis (incomplete ring sign). At this level, it is important to differentiate spondylolysis from facet joint analysing cortical contours, cortical margins and landmarks of joint capsule; on sagittal views, differences between pars defect and facet joint are more evident, and also incomplete fractures are better diagnosed [48].

The use of MRI (Fig. 19.11) is still unclear, because it has limitation in evidencing the cortical integrity of an incomplete stress fracture, but, as reported by Sairyo et al. [33], high signal on T2-weighted images is useful in early diagnosis of spondylolysis and could be used as a good predictive indicator of bony healing after conservative treatment. MRI has the advantage of being noninvasive and the lack of radiation exposure, but Masci et al. [49] reported a significant major number of false negatives if compared with single photon emission computed tomography (SPECT). Also bone scintigraphy is very sensitive for detection of bone stress, but it's not useful for diagnosis; moreover, once forward slippage develops, bone stress couldn't be any more present (however, bone remodelling and tracer uptake may occur at the pars interarticularis immediately above or below the level of fracture).

Nowadays, when spondylolysis or spondylolisthesis is suspected, SPECT in addition to bone scintigraphy increases sensitivity and improves disease localisation without exposing the patient to additional radiation. Moreover, SPECT can identify early pars stress before any bone abnormality is detected with CT [34].

Spondylolysis and low-grade spondylolisthesis (<grade 3), associated with a recent injury and acute back pain, could be successfully treated conservatively: first of all, restriction to sport activity is necessary for at least 2 months or until patients can achieve painless lumbar extension [48]; the use of bracing is suggested in acute or delayed symptomatic spondylolysis, low-grade spondylolisthesis and unilateral pars fractures: Steiner and Micheli [50] treated adolescent athletes with spondylolysis or low-grade spondylolisthesis for a mean of 2.5 years with a modified Boston brace and noted good or excellent results in 78 % of cases despite a union rate on radiographs of only 25 % at follow-up. Anderson et al. [51] noted that patients with spondylolysis showing greater signal intensity on SPECT had a better outcome with bracing treatment than patients with lower intensity signal indicating that early bracing may offer advantages compared to delayed treatment.

In addition to bracing, a physical therapy programme is suggested: it is started with flexion and stretching exercises that improve abdominal strength and flexibility, reducing hamstring contracture; extension exercises are initially avoided. NSAIDs are administrated if radicular pain complaints are present [48].

As reported in literature, majority of athletes can be effectively treated conservatively, but a long period of sport inactivity and physiotherapy is needed that could not be accepted by professional athletes. Generally, surgery should be suggested if back pain persists despite conservative treatment, or if spondylolisthesis is >grade 3 or progressive, or if neurological symptoms are present [32].

There two main options of surgical treatment: spinal fusion and direct pars repair. Spinal posterolateral fusion, with or without instrumentation, is widely studied in literature in general population, but few information are available on athletes. Recently, interbody fusion has been reported more frequently, but no clinical advantages are not yet demonstrated if compared with posterolateral fusion. Proceeding with a spinal fusion leads to loss of motion of that segment overloading adjacent segments, and return to sport could be conditioned.

Direct pars repair offers a major advantage on spinal fusion: less restriction on motion of the segment involved with reduced subsequent overloading of adjacent ones.

Several techniques have been described for direct pars repair including Scott wiring technique, hook–wire constructs, translaminar interfragmentary screw (i.e. Buck screw) technique and pedicle screw–rod–hook constructs with autogenous bone grafting [48].

Nozawa et al. [52] describe better outcomes of Scott wiring technique in 20 competitive athletes with spondylolysis or grade 1 spondylolisthesis; all athletes returned to sport, but not all to their preoperatory level of activity, while Debnath et al. [53] showed a better outcome in athletes treated with Buck screw technique.

Other series [41] of competitive athletes treated with both techniques showed good or excellent results with over 90 % of patients who returned to the same level of sport activity. In the achievement of good success with multiple techniques, it is likely that the critical aspect of surgery is resection of fibrous tissue within the defect, decortications to a bleeding surface and autogenous bone grafting of the defect. Once healing is achieved, a rehabilitation programme, as previously described, should be considered [48].

Return to sport criteria after surgery for spondylolysis or spondylolisthesis is still debated: Radcliff et al. [54] proposed a rehabilitation protocol with core strengthening and nonimpact aerobic activity at 2 weeks postoperatively with exercises performed with a neutral spine during the first 3 months; after those higher-impact exercises are started, sport-specific training could be introduced at 4–6 months. Return to play is allowed when athletes have normal strength and range of motion and they are pain-free during sport activity; this usually occurs from 6 to 12 months after surgery.

Recommendations for return to noncontact or low-impact sports after spinal fusion are controversial. Many authors allowed their patients to return to low-impact sports after 6 months; Eck and Riley [24] disagree and suggested delaying return to noncontact sport after 1 year. Criteria to return to high-impact sports are even more controversial: some authors did not recommend return to this kind of sports after spinal fusion; otherwise, there are authors who permitted return to high-impact sport after 1 year [41].

After spondylolisthesis fusion, many authors agree to forbid sports that require extreme mobility or involve heavy loads such as gymnastic, football, rugby, wrestling, weightlifting, skydiving and bungee jumping. Other authors [48, 54] did not forbid return to these sports, but they advise that athletes could be limited after surgery.

19.3.5 Posterior Element Overuse Syndrome

It is known also as hyperlordotic low-back pain, mechanical low-back pain or muscular low-back pain [26, 38]. After spondylolysis, it is the most common cause of low-back pain in adolescents.

This syndrome is not a well-defined condition: a lot of structures such as muscle–tendon units, ligaments, facet joints and joint capsules could be involved in causing low-back pain. Clinical presentation of athletes with posterior element overuse syndrome is completely similar to spondylolysis [49].

This syndrome is clinically similar to spondylolysis, and the same imaging studies are suggested, but differently to spondylolysis, they are negative. Imaging studies are necessary to exclude other causes of low-back pain originating from posterior elements [20].

Treatment does not require surgery. In order to control pain, a brief period of rest is suggested, but muscle wasting should be prevented. The use of ice and NSAIDs may be helpful in reducing inflammation. A lumbar support brace, which often is required for up to 6 weeks, may help to reduce pain and reduce hyperlordosis, which often is the cause of this syndrome. Physical therapy consists of abdominal strength exercises, antilordotic exercises and hamstring and lumbodorsal fascia stretching; extension exercises are initially avoided. Return to sport is allowed; pain-free extension is obtained, but training should be modified to avoid recurrences [20].

19.3.6 Sacroiliac Joint Dysfunction

The sacroiliac joint functions as a shock absorber and disperses forces between the trunk and the lower extremities. Sacroiliac joint dysfunction can be a cause of low-back pain due to disease, inflammation, movement dysfunction (hypermobility or hypomobility) or leg length discrepancy. The influence of proximal or distal structures is poorly appreciated but must be considered. A sacral stress fracture could be a very rare cause of pain in sacroiliac joint region, in particular in elite athletes [55]. Provocative tests for the sacroiliac joint are numerous, such as distraction test, FABER test, thigh thrust test and Gaenslen test, but they are not specific, so it's necessary to perform them together to make a proper diagnosis. Signs and symptoms of seronegative spondyloarthropathies, such as Crohn's disease, psoriatic arthritis and juvenile ankylosing spondylitis, should be addressed when the sacroiliac joint is involved [23].

A specific gold standard imaging study to evaluate sacroiliac joint dysfunction is still not identified, because of joint location and presence of overlying structures.

If pain has been present for more than 3 weeks, standard radiographs taken at a 25–30° from AP axis and lateral view should be obtained: they may show degenerative changes, ankylosis, demineralisation or a fracture. On radiographs, degenerative changes are usually first on the iliac side; if sclerosis involves the lower two-thirds of the joint on both sides, sacroiliitis is common [23]. Irregularity and widening of the sacroiliac joint could be present in an adolescent patient.

Computerised tomography is useful in identifying fractures, degenerative changes and osteoid osteomas. The high sensibility of bone scans in detecting osteoblastic activity could be exploited in the suspect of infection, fracture or a metabolic process [20].

Magnetic resonance imaging helps to diagnose soft tissue pathology, lumbar disc disease, fractures and tumours. MRI is helpful and the most sensitive for diagnosing inflammatory sacroiliitis.

Few information are available on management of sacroiliac joint dysfunction.

Ice and drugs, in particular NSAIDs, help alleviate pain and inflammation. If a sacral stress fracture is present, protected weight bearing is necessary until pain resolves. The use of bracing could be useful to stabilise the joint [20].

The influence of proximal and distal structures should be considered during physical therapy. To achieve a successful return of the athlete to sport, its mechanical demands must be evaluated carefully. Rehabilitation must focus on postural retraining of the entire abdomino–lumbo–sacro–pelvic–hip complex. The transversus abdominis has been shown to be the key muscle to functional retraining: some studies [23] demonstrate lower recurrence rates and lower pain. Recently, Richardson et al. [56] analyse that clinical benefits focusing on the transversus abdominis occur due to significantly reduced laxity in the sacroiliac joint.

Before starting any postural retraining, considering the entire abdomino–lumbo–sacro–pelvic–hip complex, a leg length discrepancy should be corrected.

Intra- or periarticular sacroiliac joint injections of corticosteroids and/or anaesthetic, with or without fluoroscopy, have not been consistently shown to be effective [23], although they theoretically should help to treat sacroiliac joint dysfunction.

Return to sport is allowed when pain is controlled and good balance of the abdomino–lumbo–sacro–pelvic–hip complex is achieved through postural retraining.

19.4 Muscle Injuries

Muscle injuries within sport are both a common and problematic occurrence. This will be acknowledged by everybody that works within sport, regardless of which sport they are involved whether that be soccer, swimming, cricket, rugby, canoeing, athletics, fencing, Australian rules football, gymnastics and Gaelic football, to list those whose epidemiology has been described within the literature since the start of 2012 [57–65]. Nevertheless, muscle injury potentially affects all sport participants, and an understanding of the injury, subsequent treatment and ultimately any injury prevention mechanisms is essential for the physician working within sport.

The category of muscle injuries often incorporates the entire musculotendinous complex and thus concerns injuries anywhere along the length of a muscle from the bone and tendon interface at the origin, along the tendon, the proximal musculotendinous junction, the muscle belly itself, the distal musculotendinous junction and the insertion of the distal tendon into bone. The general acceptance is that tendon avulsions and tears in the sportsperson are best treated by surgical intervention whether it is the Achilles [66], quadriceps [67], hamstrings [68] or biceps [69]. Therefore, the focus of this piece will be regarding injuries to the muscle belly.

It has been previously suggested that little descriptive information exists regarding the description and therefore classification of muscle injuries. Indeed, the term 'muscle strain' has been interesting commonplace yet also vague and nondescriptive. To grade muscle injuries into one of three groups has gained popularity and has been described by more than one author: O'Donoghue in 1962, Takebayashi in 1995, Peetrons in 2002 and Stoller in 2007 [70–73]. However, each of these whether being clinically based or imaging based (either ultrasound or MRI) has a very broad middle grade, which is essentially nondescriptive. This was the stimulus for the introduction of a much more descriptive classification system that was formed from a consensus of a group of experts within the field of sports medicine. The Munich classification [74] has been introduced and displays a more detailed and consistent description of muscle injuries that has already been shown to be useful and ultimately validated by the UEFA Study Group when describing return to play following muscle injury in elite soccer players [59]. It is suggested that the research community adopt the Munich classification for the foreseeable future in order to permit progression in our understanding of muscle injuries.

19.4.1 Sites of Injury

19.4.1.1 Adductor Muscles ('Groin Strain')

Collectively, the adductor muscle groups may be divided into further three muscle groups by depth of layer of muscle. The superficial group may comprise of the pectineus, gracilis and adductor longus muscles; the middle layer, of the adductor brevis; and the deep layer, of the adductor magnus. However, pain in the groin region may be attributed to more than one of these muscles or potentially none of them at all. As a result, the presentation of a patient with groin pain requires very specific attention and exclusion of other pathologies such as abdominal wall pathology (e.g. hernia), intra-abdominal disorders (e.g. appendicitis or inflammatory bowel disease), genito-urinary pathology (e.g. urinary tract infection, sexually transmitted infection, testicular abnormalities), lumbosacral pathology (e.g. nerve root irritation or compression from bony or disc disease) or hip pathology (e.g. labral tear, femoroacetabular impingement, osteoarthritis, osteochondritis dissecans, iliopsoas tendinopathy) [75].

As a result, a thorough history, examination and imaging studies are important in deciding what the source of the pain is. Regarding an abductor strain, which is one of the more common muscle injuries among soccer players [60], a patient may present with decreased abductor range of movement and decreased abductor strength. It has also been speculated that biomechanical irregularities of the lower limb, such as overpronation and fatigue of accessory muscles around the hip, contribute to the risk of adductor injuries [76]. The physical examination findings are essentially tenderness to palpation of the muscle and pain on resisted contraction, in this case resisted abduction. Imaging is thought to be useful in determining the difference in the potential diagnoses; however, it has been suggested that more consistent and systematic terminology be used when describing imaging findings [77] in order to achieve a better framework for treatment planning.

19.4.1.2 Quadriceps Muscle

The quadriceps muscles are formed by the rectus femoris, vastus lateralis, vastus intermedius and vastus medialis. Collectively, an injury to the quadriceps muscles accounts for the most common muscle injury in soccer [60]. In particular, as the quadriceps (rectus femoris) spans two joints in the hip and the knee, it is susceptible to a higher rate of injury as it will be caused by excessively forceful hip flexion and/or knee flexion [75]. An increased rate of injury is observed within the player's kicking leg, with a history of previous injury and an increase in player age.

Rupture of the tendinous origin or insertion at the tendon bone interface is identifiable as this is the area of pain and tenderness. An injury to the muscle itself is well described by the patient as during a manoeuvre of forceful quadriceps contraction as a sharp pain in the area of muscle tear with or without the sensation of a popping or tearing sensation. There is tenderness in the area of muscular tear with associated swelling and ecchymosis that may be visible after 24 h [78].

19.4.1.3 Hamstrings

The hamstring complex comprises of the semitendinosus, semimembranosus and biceps femoris, both short and long heads. All but the short head of the biceps femoris are bi-articular. This is likely to be a contributing factor into why injuries of the hamstrings are common in soccer and demonstrated no bias between kicking and non-kicking legs [60]. Commonly the injury is during running, and it is hypothesised that this injury occurs as a result of high eccentric forces and moderate muscle strain [79]. In addition, many other factors have been suggested as being contributory to hamstring injury. These include anatomical factors, muscle fibre type distribution, muscle architecture and degree of anterior pelvic tilt [79] in addition to the phase of the activity involved [80]. Of specific interest is the high rate of hamstring injuries in Australian rules football that may be accounted for by the nature of the sport where a sprinting player may have to reach down for the ball [80]. There is again an increase in the rate of injury in those that have suffered a previous hamstring injury [81].

Presentation of these injuries is often following a classical history of injury that is seen with a sudden acceleration followed by pain in the posterior thigh. It is often accompanied by a tearing sensation. Tenderness over the area of the tear is seen with associated swelling and ecchymosis as seen with other types of muscle tear. A review of the imaging of hamstring injuries suggested that the majority (86 %) occur in the muscle belly and within the biceps femoris (80 %) [82].

19.4.1.4 Gastrocnemius (Calf Strain)

The 'calf' muscle refers to the gastrocnemius and soleus complex. While again being observed at a fairly common rate in soccer [83], this injury type may again be observed in many different sports. In particular, the term 'tennis leg' [84] has been mentioned to describe this injury in the tennis player. This refers to the serving motion that fully extends the knee with sudden ankle dorsiflexion, which results in the maximum stretch of the gastrocnemius. Tennis leg typically refers to the distal myotendinous junction although the term may be used when describing any injury to the calf region. Again the fact that the muscle is bi-articular is likely to be a reason as to why it is a fairly commonly injured muscle [82].

The clinical presentation is consistent with other types of muscle injury; a particular event is often noted with pain and potential tearing sensation felt. Then follow the examination findings of swelling and ecchymosis with tenderness to palpation over the area of torn muscle fibres.

The injury tends to occur in younger patients following a period of heavy exercise, whereas in older patients, it may occur following a much more insidious episode such as stepping out of a car which may not have registered with the patient as being a specific injury [82]. As a result, an important differential diagnosis includes deep venous thrombosis (DVT), particularly in the absence of a specific injury, as this is also more likely in older patients. Interestingly in a study of patients with ultrasound evaluation of tennis leg, 10 % of patients were found to have a DVT without any other finding and a further 5 % in addition to another finding [85]. Another important differential diagnosis is an Achilles tendon rupture, which should be differentiated upon clinical examination with a palpable gap at the site of tendon failure. In the event of any doubt regarding the diagnosis, an ultrasound scan is suggested, as the management of the two possibilities is extremely different, with the mainstay of muscle belly injury being nonoperative compared to a low threshold for surgical intervention in the athlete with Achilles tendon rupture.

19.4.2 Epidemiology

In essence, any of these injuries may occur in any sport. Indeed, if one considers the functional chain to be of significance, then any muscle injury will have an impact on any sport, e.g. a quadriceps injury in the overhead athlete. The next section will summarise some of the epidemiology findings specific to some of the most common sports in the world.

19.4.2.1 Soccer

The world game has been investigated extensively with regard to the epidemiology of muscle injury, mainly in the form of the UEFA Study Group. A review of 2,123 muscle injuries concluded that intrinsic factors found to increase muscle injury rates in professional soccer were previous injury, older age and kicking leg and that injury rates varied during different parts of the season and also depending on match location [60]. However, importantly, further UEFA studies have shown that while ligament injury rate has decreased over the last decade, muscle injury remains high [60]. This may be associated with another finding from the group that has shown fixture congestion to be associated with high muscle injury rates [86].

19.4.2.2 Swimming

A recent study has shown the rates of injury in elite Paralympic swimmers with visual impairment. As one may expect, the muscular injuries are more prevalent in the trunk and spine muscles and the upper than the lower limb although of interest 19.9 % of injuries were seen in the lower limbs [87].

19.4.2.3 Rugby Union

In rugby, and in particular sevens rugby union (an Olympic sport in 2016), the rate of injury has been described as being 55.4 injuries per 1,000 playing hours with 14.6 % of all injuries being in the lower extremity in terms of site and 10.4 % being muscular injuries in terms of type [62]. The sport of rugby itself is interesting, as it has moved from an amateur era into a modern professional sport. This change has accounted for an increase in injury risk [88]. A meta-analysis has confirmed that an increase rate of injury is observed with an increase in the level of rugby that is played; in addition, most injuries were muscular and equivalent in rate to other collision sports [89].

19.4.2.4 Tennis

An extensive review article concerning tennis has shown a general trend that acute injuries in tennis were more likely to occur in the lower extremity rather than the upper extremity. There was no association with age, sex, skill level and injury rate although the volume of play was associated with the risk of injury. Tennis leg itself accounted for between 4 and 9 % of all tennis injuries discussed [90].

19.4.2.5 Ice Hockey

A study of 1292 National Hockey League (NHL) players demonstrated that low levels of sport-specific training in the off-season (as compared to high levels) and previous injury were factors associated with increased risk of groin injury [91]. This was the basis for potential injury prevention strategies to be introduced in the sport.

19.4.2.6 Australian Rules Football

While not being potentially a worldwide sport, 'Aussie rules' has led the way in terms of injury surveillance with over 20 years of data from the elite league governing 13,606 injuries [64]. During this time, hamstring injuries have featured most commonly, and interestingly recurrence rates of injury have decreased each season. The authors concluded that such annual surveillance of injury rates was well received and crucially was instrumental in aiding rule changes that improved player safety.

19.4.3 Treatment

19.4.3.1 Traditional Methods

The mainstay immediate treatment of muscle injury for many years has been the 'RICE' method incorporating rest, ice, compression and elevation [92]. The aim of which is principally to decrease the risk of injury-induced bleeding into the muscle beyond the initial zone of injury with the result to minimise the extent of the injury [93]. This also decreases the inflammatory process in its earliest phase and helps with the pain that a patient will experience following injury. Subsequently to RICE, the term PRICE has evolved in order to add the use of 'protection' in the form of soft padding to the injured area of muscle to decrease the risk of impact injury with other objects [93].

It is recommended that the rest phase of activity restriction be present for 48–72 h post injury and that through this time ice be applied in 15- to 20-min spells every 60–90 min. It is also suggested that the compression bandage be applied during the periods where icing is not used (ACSM). The duration of rest and immobilisation is limited to this time as it is a period sufficient enough to produce a form of scar with sufficient strength to bear the forces that will be experienced by the next phase of treatment, namely, commencing mobilisation [93].

Beyond 72 h and dependent upon the severity of the injury, the next phase of rehabilitation will begin with a gradual increase in activity, which may begin with gentle movement of the muscle, mild resisted exercise, proprioception exercise and continued icing. Following this phase, a gradual increase in activity is permitted with pain being the indicator of speed of progression (ACSM). The recommendation is to progress from isometric to isotonic to isokinetic muscle contraction training through this time with again pain being the guiding factor [93].

An early return to activity is required in order to optimise the regeneration of healing muscle and promote the recovery of flexibility and strength of the injured skeletal muscle to pre-injury levels. The rehabilitation protocol should also incorporate core stability exercises, as these exercises appear to result in a better outcome for injured skeletal muscle than programmes based exclusively on stretching and strengthening of the injured muscle alone [93].

Following this early phase of treatment, the specifics of the muscle rehabilitation are dependent upon the muscle group involved, e.g. the use of Nordic hamstring exercises [94, 95]. Such exercises can be initiated in order to help rehabilitation and prevent recurrence.

In spite of these treatments being considered traditional and the basis of most management plans for the treatment of muscle injury, little in the way of objective evidence exists within the literature to suggest this is the optimum treatment. As a result, other techniques have been suggested and attempted in an effort to decrease the healing time from injury.

19.4.3.2 Medication

Few controlled studies exist to demonstrate the effects of nonsteroidal anti-inflammatory drugs (NSAIDs) or glucocorticoids in the patient with muscle injury. Some have suggested a transient improvement in recovery from exercise-induced muscle injury [96]. In addition, short-term use of

NSAIDs in the early phase of muscle healing has been shown to lead to a decrease in inflammatory cell reaction without any adverse effect on muscle healing, tensile strength or contraction [93]. However, the long-term use of NSAIDs is potentially detrimental to the regenerating skeletal muscle, with the suggestion that it is harmful in the eccentric contraction-induced strain injury model [97].

19.4.3.3 Ultrasound

Once again, while being well recognised as a treatment for muscle injury, the role of ultrasound has very little supportive objective evidence existing within the literature. Some animal-based studies suggest a benefit from continuous therapeutic ultrasound in accelerating the healing response [98], whereas others suggest that while ultrasound will promote the satellite cell proliferation phase of myoregeneration, it did not display any benefit in the overall morphology of muscle regeneration [99].

In the absence of any randomised controlled trial that offers definitive evidence as to the effects of ultrasound in muscle healing, studies continue to emerge with the most recent in the literature again supporting the use of low-intensity pulsed ultrasound (LIPUS). The suggestion is that it induced an organised tissue structure at the site of injury and stimulates expression of cyclooxygenase-2 (COX-2) and the formation of new muscle fibres [100].

19.4.3.4 Hyperbaric Oxygen Therapy

Scientifically, it makes perfect sense that by increasing the concentration of oxygen within the body, there would then be more oxygen availability for the cellular processes of muscle healing and regeneration [93]. However, in spite of some experimental studies showing that its use may significantly improve the rate of repair of injured skeletal muscle [101], a subsequent Cochrane review has stated that there is insufficient evidence from randomised controlled trials that will identify the effects of hyperbaric oxygen therapy on delayed-onset muscle soreness [102], which was the closest review found in the literature to muscle injury in the athlete.

19.4.3.5 Kinesio Tape

The use of colourful taping has gained popularity among athletes in recent times. This has particularly been seen in the elite athlete population. That being said, very little exists in terms of scientific evidence as to the benefits of the taping. A systematic review performed in October 2011 and published in November 2012 concluded that there was insufficient evidence to support the use of kinesio tape following musculoskeletal injury due to there being few high-quality studies that have investigated its use [103]. Interestingly, however, the review was unable to discount a perceived benefit that the athlete may have. No ill effects were perceived; therefore, it may be that the use of such taping may provide a placebo effect for the athlete.

19.4.3.6 Platelet-Rich Plasma (PRP)

PRP has become very popular as a method of treatment for a number of different diagnoses. One is to treat muscle injuries. The possibility has received much coverage in the medical literature without any convincing conclusion to whether or not it carries a benefit. Scientifically, it stands to reason that PRP injections into muscle would carry a benefit; however, comment is made of a single poster presentation that showed benefit in a case series of 20 professional athletes with hamstring injury without subsequent publication [104]. Little else exists within the literature that would convincingly suggest a beneficial effect from PRP injection into muscle in the clinical setting. A review article has suggested that no PRP formula has yet been proven to result in good-quality evidence of muscle healing and recovery following injury in sports [105]. This feeling appears to be shared by the International Olympic Committee Sports Medicine consensus group as well, with a warning to proceed with caution in the use of PRP and the statement that further clinical trials are required [106]. Interestingly, one study is progressing and appears to be of suitable design quality in order to provide and answer to the question as to how much benefit PRP will provide as treatment to an injured muscle.

19.4.4 Complication

19.4.4.1 Myositis Ossificans

Although being rare, this is an important complication of muscle injury or repetitive muscle injury where bone or cartilage is laid down at the site of trauma. Clinically, it should be suspected if pain and swelling have not subsided by 10–14 days after a muscle injury and the normal pattern of injury recovery is not being observed. The ossification itself is unlikely to be visible on radiographs for around 6 weeks, as per any standard bone formation. The treatment is debated, and NSAIDs may be used in an attempt to decrease the rate of ossification as demonstrated within the literature for the process of heterotopic ossification [93]. Other treatments that have been successful, albeit on a case report basis, include extracorporeal shock wave therapy [107] and acetic acid ionophoresis, which has been described previously [108] and again in recent times [109].

19.4.5 Injury Prevention

It was said by Benjamin Franklin that an ounce of prevention is worth a pound of cure. This certainly appears to be the trend with muscle injury as well. It has been shown that the use of eccentric hamstring exercises (Nordic hamstring exercise) as a part of a 10-week prophylactic training programme resulted in a reduction in the rate of overall, new and recurrent hamstring injuries in professional and amateur male soccer players in a randomised controlled trial [95]. Similarly, a spreventative exercise programme has been shown to be of benefit in reducing adductor muscle injuries in soccer as well [110].

The FIFA 11+ is a series of warm-up exercises designed by the FIFA Medical Assessment and Research Centre (F-MARC) that has since been popularised and integrated into the framework of soccer coaching and subsequently adopted by many soccer associations across the world due to its success at decreasing the rate of a number of injuries [111]. It has been demonstrated to be of benefit in terms of reducing a large spectrum of lower limb injuries in the sport of soccer, including muscle injuries [112, 113]. Further, widespread incorporation of the FIFA 11+ is likely to be of benefit in further decreasing the rate of injury in other sports as has already been demonstrated in basketball [114].

Finally, core stability appears to have an associated effect of risk of injury to the lower limbs. A study in American football suggested that suboptimal core stability might be a contributory factor towards an increased risk of injury in a preliminary study [115] and the previously mentioned study that decreased the frequency of adductor muscle injuries also utilised a programme that improved core stability [76].

Ultimately, muscle injury has a traditional approach to its treatment, which is accepted. It also has a number of potential improvements to this standard treatment that may yet be shown to be of benefit. However, it would appear that the greatest benefit is seen with a structured preventative exercise programme and as such should be considered by those involved with the medical care of sports teams.

19.5 PRP and Stem Cells

In recent years, many developments on the treatment of musculoskeletal injuries/diseases have been done within the scope of regenerative medicine. Several reports have been published regarding the use of platelet-rich plasma (PRP) and stem cells, alone or in combination with delivery carriers and/or biomaterial matrices also known as scaffolds. These have shown great promise for treatment of damaged meniscus, ligaments, tendons, skin, bone, cartilage and osteochondral tissues. The main aim of this review is to highlight the most recent and relevant reports on the use of PRPs and current role of stem cells in clinical orthopaedics (autologous treatments). Herein, the tremendous advances in the clinical applications of PRP, namely, the aspects related with the processing techniques, formulations and administration, are briefly overviewed. Moreover, the regenerative potential of stem cells (and its sources) and the control of their cellular fate by means of using different biomolecules and clinical use (mostly in knee joint) are also analysed. Finally, new promising regenerative treatment solutions based on tissue engineering (TE) strategies are also briefly discussed. The global concept of tissue engineering requires the combined roles of a triad: cells, scaffolds and bioactive molecules (including several growth factors). When trying to figure out 'how any tissue functions' and what is necessary to put its 'repair mechanisms' at work, one cannot focus only on an isolated factor. It is necessary to understand the different roles of these different protagonists at different times to envision influencing the role of events necessary for tissue repair. There is no such thing as a 'panacea' of modern times capable to solve every problem using the same method. It is still necessary to further investigate the tissue's biology in order to achieve more effective and reproducible methods of their repair.

For the present time, it is not possible to provide neither evidence-based indications nor guidelines concerning the clinical use of PRP and/or MSCs. Despite promising early results, these are still in the early phase of its clinical experience, and there is a long way in the field of research concerning these issues.

19.5.1 PRP

Autologous platelet-rich plasma (PRP) is a generic term referring to any sample of autologous plasma with platelet concentrations above baseline blood values [116]. Being a source of concentrated autologous platelets, after degranulation of their alpha granules, PRP provides a source of several different growth factors and other cytokines [117, 118].

PRP has been under development as a theory since the 1990s and has been increasingly used in clinical applications [118, 119]. However, one must understand that this technology is still in the early phase of its development and further research is required in either basic science or clinical perspectives. PRP technology basically relies in providing fibrin and high concentrations of a 'cocktail' of growth factors with 'a certain' potential to aid in selected tissue healing, including bone and soft tissues [119]. The role of multiple growth factors (GFs) in different biologic repair mechanisms as well as the variety and raised concentrations of these GFs found within PRP is the theoretical basis supporting the use of PRP in tissue repair [120]. The growth factors and other cytokines present in PRP include platelet-derived growth factor (PDGF), transforming growth factor beta (TGF-β), fibroblast growth factor (FGF), insulin-like growth factors 1 and 2 (ILGF-1, ILGF-2), epidermal growth factor (EGF), interleukin 8 (IL-8), vascular endothelial growth factor (VEGF), hepatocyte growth factor (HGF), keratinocyte growth factor (KGF) and connective tissue growth factor (CTGF), among many others [121]. However, there is no current effective method to control the availability or concentration of any of them prior to application. There are inter- and intra-individual differences as well as inherent to the method of preparation [122].

Moreover, according to Langer and Vacanti, the principles of tissue engineering and biologic repair mechanisms not only depend on the presence of bioactive proteins (e.g. growth factors) but also include cells and scaffolds [123]. It is necessary to make this 'magic triad' work properly in order to achieve successful tissue regeneration [124].

Considering the aforementioned, one should have no doubts about the critical role of 'growth factors' in biologic repair mechanisms. What must be appreciated and better understood is the effectiveness of all clinical methods aiming to use these proteins in clinical setting. The rationale supporting this method is that by delivering an amount of GFs, we might influence the cascade of events leading to the recovery of a tissue after an injury. One effective way to jeopardise a promising technology is to promote its incorrect use since the beginning of its application.

One should be aware that, up to now, it has not been possible to confirm in large-scale and long-time controlled clinical trials all the observations and suggestions arising from basic science research and preclinical studies [125].

There is no consensus on the ideal dosage of platelets to be delivered through PRP preparations. However, the principle of 'the more, the better' does not apply in this sense [126]. A wide range of platelet concentrations from 200 × 103 platelets/mL up to 1,000 × 103 platelets/mL are considered therapeutic for tissue healing, whereas much higher counts appear to have biologic deleterious effects [127–129]. The platelets collected in PRP can be activated by the addition of factors such as thrombin and/or calcium chloride, which induces the release of these factors from alpha granules.

Plasma, the fluid portion of blood, contains ions, inorganic and organic molecules as well as same proteins which assist in healing process of connective tissues [130]. Plasma is different from serum once plasma still contains fibrinogen as well as certain clotting factors. When plasma is exposed to thrombin, either by the adding exogenous thrombin or by contact with tissue thromboplastin, the clotting cascade is initiated and platelets are activated [130].

Creating such a platelet-rich fibrin matrix, one might achieve benefits in selected situations. A fibrin scaffold might play the role of conductive matrix for cells' migration as well as provide a GF reservoir that indirectly binds growth factors, thus prolonging their delivery [126].

Differences between preparations further include the presence or absence of white blood cells (WBCs). WBCs are known to play a role on the initial phases of inflammation, namely, removal of bacteria and/or biologic debris [126]. There is no consensus on this issue, with some methods developed for intentional removal of leukocytes while others promote leucocyte-rich PRP preparations [122]. Some in vitro studies have reported some antibacterial effects related to inclusion of WBCs in PRP [131, 132]. However, it is also recognised that WBCs can release matrix metalloproteinases and produce reactive oxygen species that might lead to increased tissue damage [133]. This represents another key issue, determining a significant difference among the standard methods for the PRP preparation.

It is possible to describe PRP preparation according to the method of production. Single- and two-step centrifugation processes have been described to fraction whole blood and concentrate the platelets [122]. PRP preparation systems have recently gathered FDA approval for clinical use (http://www.fda.gov/).

Some proponents of PRP therapy argue that negative clinical results are associated with poor-quality PRP produced by inadequate devices; however, there are poor evidences or guidelines favouring any method over another [122]. There are inter- and intra-individual differences in human donors affecting the quality and quantity of PRP preparations [122]. Moreover, variability in platelet-concentrating techniques may alter platelet degranulation characteristics that could affect clinical outcomes [134].

Dose–effect is also a relevant variable to consider. In a recent in vitro study [135], certain doses of PRP could inhibit adipogenic differentiation while inducing the proliferation and osteogenic differentiation of bone marrow mesenchymal stem cells derived from osteoporotic bone marrow, thus promoting fracture healing. However, high concentration of PRP inhibited osteogenic differentiation and callus remodelling [135].

Combining all the previous, the possibility of 'a la carte' PRP seems somewhat feasible, in a near future [136]. That is, if several methods exist, if different products are obtained accordingly, maybe, after development of our ability to fine-tune and control these differences, one might choose the 'type' of PRP product that best adapts to a specific clinical model. It must be recognised and understood that this plethora of methods to harvest GFs from PRP products creates difficulties in evaluating clinical outcome and in interpretation of published literature.

The use and clinical validation of PRP in orthopaedics and sports medicine are still in their early stages [137]. There are few controlled clinical trials that have adequately evaluated the safety and efficacy of PRP treatments. Most published literature remains on the level of expert's opinion or small case series without control [125].

PRP has a promising profile for injection therapy permitting minimally invasive, nonoperative approaches for several conditions [138, 139]. Its proposed applications include shoulder cuff

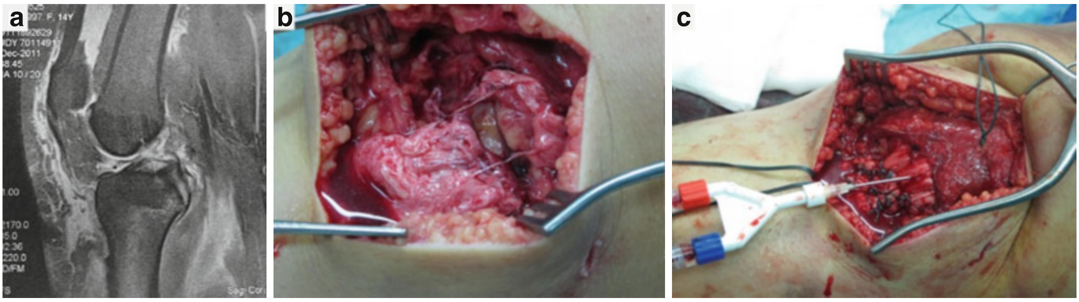

Fig. 19.12 PRP application in bone fracture. (**a**) Bone fracture, (**b**) surgical treatment, (**c**) PRP application

pathology, epicondylitis, Achilles tendinopathy, plantar fasciitis, jumpers knee, muscle injuries or even joint arthritis [138]. There has been some controversy around clinical results. However, one must consider that these results have been obtained from different injuries or pathological models, involving different tissues, many times different preparations of PRP which limits the possibility for further widespread conclusions or guidelines [119]. Once PRP is not supposed to be the 'panacea' of modern times, it is difficult to expect that it will always have to 'work' no matter the strategy of therapeutic model.

A recent meta-analysis considering rotator cuff tears suggests that PRP has no benefits on the overall clinical outcomes and retear rate after arthroscopic repair of large- and massive-sized defects. However, it was observed a decrease in the rate of retears among patients treated with PRP for small- and medium-sized rotator cuff tears (level II study) [140]. Focused on lateral epicondylitis, a small controlled trial described promising results for 'tennis elbow' after failure of previous conservative treatment [141]. Some limitations inherent to study protocol must be considered including the small number of patients considered for results.

However, concerning Achilles tendon disease, modest improvement in functional outcome measures was observed in a small case series following PRP injection [142]. On another clinical trial enrolling patients with chronic Achilles tendinopathy (after failure of physiotherapy), PRP injection did not result in better outcome in pain nor activity when compared to a saline injection [143].

In a randomised trial with a small number of patients, PRP presented favourable results in improving patellar tendon healing after harvesting for ACL repair [144]. A small case series without control reported clinical improvement after PRP in treatment of jumper's knee [145]. Once more, design of the studies must be carefully considered.

Contradictory results have been reported regarding the influence of PRP in graft integration after ACL repair. Tendon-to-bone healing (osteoligamentous interface) after ACL repair has presented nonsignificant differences [146, 147]. Nevertheless, some improvement in MRI signal concerning graft maturation has been described [147].

A recent single-blinded, randomised, controlled study comparing PRP injections to dextrose prolotherapy for the treatment of chronic recalcitrant plantar fasciitis achieved similar results with only slight better initial improvement in function for PRP group [148].

Once more, in favour of clinical application for PRP, intra-articular knee injections produced favourable functional results on degenerative cartilage lesions as well as pain decrease and quality of live improvement [149].

PRP has also been tempted to improve outcome after traumatic injuries (Fig. 19.12) including fracture healing. PRP combined with allograft achieved similar outcome when compared to autograft and significantly better than allograft alone in the treatment of displaced intra-articular calcaneal fractures [150].

In brief, PRP technology has been recently tested in a wide range of musculoskeletal pathologies in orthopaedics but also in sports medicine particularly aiming to improve outcome and fasten recovery in high-level athletes.

The issue concerning PRP implications in antidoping rules by either the World Antidoping Agency or the International Olympic Committee (IOC) has been continuously appreciated [151, 152]. It has been discussed if local injections of PRP could have a systemic impact affecting doping tests as well as possible anabolic effects affecting performance [152]. In 2011, the World Antidoping Agency removed intramuscular injections of PRP from its prohibitions after determining that there is a '… lack of any current evidence concerning the use of these methods for purposes of performance enhancement' [153]. Similarly, the IOC tolerates the use of PRP given the lack of evidence concerning systemic effects affecting performance and however recommends caution with the use of these methods [152, 154]. Conversely, the use of PRP is still illegal according to medical care law in countries such as Korea [155].

PRP technology is a promising technology for sports medicine and musculoskeletal application. It is clear that GFs can influence tissue's repair mechanism. PRP is a method for harvesting autologous GFs whose source each one of us always carries around. What remains unclear is the best method for each tissue, what must be included and what should be excluded in each specific clinical case. Moreover, the timing, the dose and the conjunction with other therapeutic agents are also issues that should be considered. It is necessary to develop appropriate guidelines and increase evidence level prior to its widespread application as treatment option for joint, tendon, ligament and muscle injuries [125]. Results of clinical studies on PRP are difficult to interpret given the methodological quality and limitations of most published clinical trials [154]. More attention should be paid to methodological issues when designing, performing and reporting clinical trials assessing outcome of PRP technology.

To improve clinical application, several authors have been trying to tune and control some aspects. It has already been shown that an innocuous agent like anti-VEGF antibody can play a role to modulate and control some effects of PRP [116]. Moreover, a combination of PRP injections and oral administration of losartan (an anti-fibrotic agent) could enhance muscle healing by stimulating muscle regeneration and angiogenesis and by preventing fibrosis in skeletal muscle from mice model [156]. Research in this field still has a long way to go.

Future directions of PRP application may concentrate on seeking an appropriate method to control their effects (either by favouring one effect over another or by controlling different effects at different times). Moreover, given the multiplicity of different methods providing different PRP products, in future we might be able to provide guidelines in selection of different products best suited for specific effects: the 'a la carte' PRP technology. It is now consensual that more controlled trials are required describing properly the method of PRP tested in order to permit higher level of evidence and indications for use.

19.5.2 Stem Cells

The different types of cells found in our body can be generally categorised as germ cells, somatic cells and stem cells. Germ cells give rise to gametes, while somatic cells are the differentiated cells that constitute the adult body. By its turn, stem cells are characterised by cell types possessing the ability of dividing indefinitely, in vitro. Actually, stem cells may become a specialised cell upon differentiation, under the action of specific growth factors/bioactive agents. Therefore, stem cells can proliferate and differentiate beyond the tissues in which they normally reside or may be implanted. An interesting work has been reported by López-Ruiz et al. [157], which showed that chondrocytes extract from patients with OA can induce chondrogenesis in infrapatellar fat pad-derived stem cells.

In respect to its body location, stem cells have been identified and isolated from germ cells, embryo, fetus and many adult tissues (e.g., adipose tissue, skeletal muscle, Wharton's jelly) [158], including those of diarthrodial joints.

Nevertheless, stem cells that express embryonic and adult stem cell markers have also been found in amniotic fluid, for example [159].

Thus, stem cells may be classified according to the tissue source or, alternatively, their capacity of differentiation (plasticity), as follows:

- Totipotent stem cells are present in the early embryo, and it can differentiate in all types of specialised cells of the body, including the entire fetus and placenta (e.g. zygote and immediate daughter cells).
- Pluripotent stem cells are isolated from the fetus, and it can differentiate in several cell types of all three germ layers (ectoderm, mesoderm and endoderm), but not the whole organism (e.g. embryonic stem cells derived from the isolated inner cell masses of mammalian blastocytes, embryonic germ cells (until 8th week) and embryonic carcinoma cells are some examples of pluripotent cells).
- Multipotent stem cells are cells that can differentiate in a limited type of specialised cells (e.g. mesenchymal stem cells (MSCs), neural stem cells and haematopoietic stem cells).
- Unipotent stem cells are also obtained from adult organs giving rise to one differential cell lineage (e.g. immature oligodendrocytes and keratinocytes).

Recently, Yamanaka's group [160, 161] has found that mature specialised cells also known as induced pluripotent stem (iPS) cells can be reprogrammed into immature cells capable of differentiating into all tissues of the body. iPS cells are particularly interesting for use in in vitro models of disease since it does not present the ethical problems of the use of human embryos. Another advantage of iPS cells is that creation of cell lines that are genetically tailored to a patient became also possible. With this significant progress, musculoskeletal regenerative medicine can be fully addressed. Still, several hurdles have to be overcome before its use in therapeutic applications, namely, the need for a fast and efficient reprogramming [162]. Besides, this technology makes use of traditional viral vectors (some are oncogenes), which should be avoided.

Despite the great developments in stem cell research for orthopaedics [163], fundamental studies are still needed to better understand its tissue location, differentiation pathways, paracrine signalling, mechanism of action and more importantly the regenerative potency of different stem cell types [164]. Another important issue is related to the poor knowledge of stem cell fate once implanted at a defect site. Therefore, all these issues must be clarified before it's fully acceptance by clinicians and regulators as potential therapy.

Joint disease and repair (trauma) are some of the major challenges that orthopaedic surgeons have to face in their daily activities. Most treatments include pain control with administration of drugs and injection of biomaterials such as hyaluronan or chondroitin sulphate (viscosupplementation strategy) and other natural-based polymers [165–168]. Despite symptomatic improvements in most cases, these treatment solutions have failed in regenerative results, thus leading to progressive loss of joint functioning and ultimately requiring patients' joint replacement [169].

It is well known that mammal's tissue repair mechanisms result from the activation of pre-existing stem cells or progenitors cells. In addition, endogenous MSCs contribute to the maintenance of healthy tissues by playing as repairing reservoirs or immunomodulators to reduce inflammation processes [170, 171]. Thus, stem cells use promise to revolutionise traditional clinical practice with a striking impact on patients' quality of life [172], i.e. from simple symptom control and repair approaches towards the whole tissue or organ regeneration. Despite the use of stem cell-based therapies in clinics is still limited due to the legislative/regulatory constraints and socio-economic factors [162, 173], adult stem cell application has been increasing in the area of joint disease/repair, in the last few years. Actually, adult stem cells have been found in bone marrow, synovium, synovial fluid, meniscus, articular cartilage, ligament and fat pad [174, 175]. Figure 19.13 shows the typical stem cells after being isolated from human knee fat pad.

Clinical trials using MSCs have been stimulated [176–178] by animal studies that showed a beneficial effect of MSC's transplantation to the osteoarthritic or damaged joint. The clinical trials

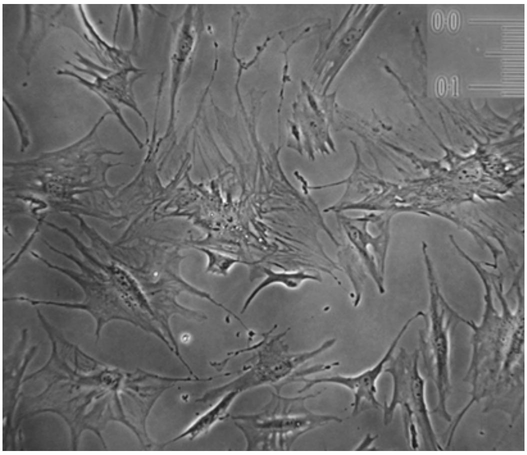

Fig. 19.13 Stem cell isolation

were aimed to investigate the therapeutic effects of autologous MSCs as intra-articular injection (mostly in the knee) [175] or after transplantation upon cells seeding into matrices for the treatment of diseased/damaged cartilage tissue. In the following sections, a comprehensive overview of the clinical reports indicating the potential for stem cell therapies and combination of stem cells with scaffolds (TE strategy) in the treatment of joint defects/disorders will be provided.

As aforementioned, MSCs can be found in almost all tissues of the diarthrodial joints and have been exploited for their self-renewal capacity and ability to differentiate towards osteogenic, chondrogenic and adipogenic lineages and apparent myogenesis [174].

A great deal of attention has been given to stem cell-based therapies by means of delivering MSCs in vitro cultured into the defect/diseased areas. Recently, Mirabella et al. [170] has shown that amniotic fluid stem cells (AFSCs) are not osteogenic in vivo, although AFSCs can recruit more host CD31- and VEGF-R2-positive cells as compared to BMSCs. In that work, the authors concluded that AFSCs do not contribute to the deposition of de novo bone, but it can modulate a host response beneficial for enhancing the vascularisation of the bone environment.

Another beneficial application of autologous MSCs has also been reported for meniscus repair. Centeno et al. [179] showed regeneration of meniscus in a knee treated after percutaneous implantation of MSCs.

McIlwraith et al. [180] has investigated the effect of intra-articular injection of MSCs for treatment of microfractured chondral defects, in a horse model. In this preclinical study, a histological improvement was evident, which indicates that intra-articular injection of BMSCs enhances cartilage repair quality. In respect to clinical cartilage regeneration, Saw et al. [181] reported on a pilot study involving 180 patients (grade III and IV lesions of the knee joint, ICRS) that underwent arthroscopic subchondral drilling. Intra-articular injections of autologous peripheral blood progenitor cells (PBPCs) in combination with hyaluronic acid (HA) (8: 2 ml) were administered 1 week after surgery. A total of 5 weekly intra-articular injections were given. Five patients were evaluated postoperatively. The authors obtained encouraging results as articular hyaline cartilage regeneration was observed at second-look arthroscopies and histological characterisation.

It is in the treatment of OA that stem cell-based therapies are most appealing [182]. In that review, it is the author's opinion that bone marrow-stimulating techniques and ACI can improve pain relief to the patients and are superior to no treatment. Similar in their clinical outcome, they induce fibrocartilaginous repair tissue which possibly can progress to hyaline cartilage formation with time. MSCs isolated from infrapatellar fat pad have also been investigated as potential therapy [183] for treatment of knee OA. In that study, stem cell injections ($n = 25$; mean of 1.89×106 stem cells prepared with ~3.0 ml of PRP) combined with arthroscopic debridement were administered to OA patients. Results demonstrated that the mean Lysholm, Tegner activity scale and VAS scores of patients treated with stem cells improved significantly at the last follow-up, as compared to control groups (patients had undergone arthroscopic debridement and PRP injection without stem cells). Therefore, the authors found that MSC's therapy with intra-articular injections is safe and provides assistance in reducing pain and improving function in patients with knee OA. In a recent study,

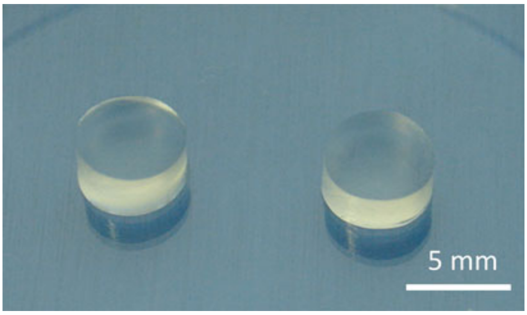

Fig. 19.14 Cell-laden hydrogels

Koh et al. [177] reported once similar findings showing that MSC's injections can improve symptoms of OA knee.

Interestingly, Barry and Murphy [174] reported that 13 clinical trials addressing research on OA were ongoing in 2012. In most of the studies, MSCs isolated from bone marrow and adipose tissue are administered by intra-articular injection (1–4 × 107 cells/injection), after in vitro expansion. In these cases, no scaffolds were used as support for cell growth and differentiation, but hyaluronan was often used as the delivery carrier.

Interestingly, preclinical studies have been exploiting combinatory strategies by means of using cell-laden hydrogels (Fig. 19.14) mimicking the extracellular matrix, which can be further loaded with micro-/nanoparticle systems for the delivery of drugs aimed at promoting cell proliferation and differentiation, at the defect site [184–186].

The pioneering work of Ohgushi et al. [187] showed that the use of MSCs isolated from the patient's iliac crest followed by seeding onto implants contributed to improve the bone–prosthesis interface, with no inflammatory reactions. These preliminary results indicated that a TE strategy using MSCs (autologous approach) can contribute for preventing aseptic loosening of the total ankle arthroplasty. Similarly, Bertram et al. [188] demonstrated the validity of matrix-assisted cell transfer for intervertebral disc cell therapy. Another report also demonstrated that BMSCs combined with porous beta-tricalcium phosphate may be beneficial in posterior spinal fusion [189].

Marcacci et al. [190] reported on a pilot study involving stem cells associated with hydroxyapatite scaffolds for the repair of critical-sized long bone defects. That study showed the long-term durability of bone regeneration achieved by a bone TE approach at 6- to 7-year follow-up.

Different works have been reporting that microfracture procedure shows comparable clinical results to those treated with ACI (autologous chondrocyte implantation) in the treatment of small cartilage defects. In addition, microfracture procedure is only able to produce fibrocartilage tissue. Thus, novel strategies based on matrix-guided stem cell implantation for treatment of chondral lesions are more appealing as compared to cellular strategies as it has the promise to improve outcomes and produce mature tissues [191]. In another preclinical study, Yamada et al. [192] reported on an investigation involving bone regeneration with stem cells isolated from several sources (e.g. deciduous teeth, extracted from puppies and grafted them into a parent canine mandible as an allograft, parent dental pulp, and bone marrow by tissue engineering and regenerative medicine technology using platelet-rich plasma as an autologous scaffold and signal molecules). In that study, it was evident that stem cells from deciduous teeth, dental pulp and bone marrow with PRP have the ability to form bones. Apparently, bone formation with puppy deciduous teeth stem cells can have the potential to produce a graft between a child and parent.

There is a general consensus that one-stage procedure is advantageous over the conventional autologous chondrocytes implantation. Girolamo et al. [193] reported on the treatment of chondral defects of the knee with one-step matrix-assisted technique improved by means of using autologous concentrated bone marrow.

In another two-case report, Kasemkijwattana et al. [178] also investigated the therapeutic effect of implanting BMSCs in cartilage defects (grade III and IV lesions of the knee joint, ICRS). Despite the need for long-term follow-up, that study corroborated the previous findings [194] that BMSC's implantation after seeding into a collagen scaffold showed a great potential for the treatment of large cartilage defects. More recently, Richter and Zech [195] reported on the clinical evaluation of MASI procedure and 2-year follow-up in chondral defects of the foot and ankle, but no control group was included. Evaluation comprised size and location of the chondral defects, method-associated problems and the Visual Analogue Scale Foot and Ankle (VAS FA) before treatment and at follow-up. This interesting work comprised evaluation of 25 patients revealing good clinical scores and no major complications. The authors concluded that MASI is a safe and effective method for the treatment of chondral defects and presents the advantage of being a single-procedure methodology as compared to ACI and MACI.

Driven by the good preclinical results, stem cell clinical trials are still ongoing. The latter are mostly following the simpler strategy of stem cell implantation using a scaffold-free method, but the cell number and density, dosage and matrices/gels/hydrogels for cells delivery have not yet been established. In a near future however, we firmly believe that clinical trials will evolve to advanced tissue engineering strategies, i.e. making use of stem cells combined with scaffolds and possibly also supplemented with PRPs or other growth factors. Nevertheless, these will present some surgical concerns for efficient delivery and many regulatory constraints. Finally, much knowledge is still needed, namely, that related to regulation of stem cell differentiation, role of host cell recruitment, type of transplanted cells, defect-filling materials and growth factor formulations in order to help improve surgical outcomes in patients presenting large defects and showing advanced signs of disease.

References

1. West TA, Marion DW. Current recommendations for the diagnosis and treatment of concussion in sport: a comparison of three new guidelines. J Neurotrauma. 2014;31(2):159–68.
2. Zazryn T, Cameron P, McCrory P. A prospective cohort study of injury in amateur and professional boxing. Br J Sports Med. 2006;40(8):670–4.
3. Kazemi M, Pieter W. Injuries at the Canadian National Tae Kwon Do Championships: a prospective study. BMC Musculoskelet Disord. 2004;5:22.
4. Kemp SP, Hudson Z, Brooks JH, Fuller CW. The epidemiology of head injuries in English professional rugby union. Clin J Sport Med. 2008;18(3): 227–34.
5. Fuller CW, Junge A, Dvorak J. A six year prospective study of the incidence and causes of head and neck injuries in international football. Br J Sports Med. 2005;39 Suppl 1:i3–9.
6. Boden BP, Tacchetti RL, Cantu RC, Knowles SB, Mueller FO. Catastrophic head injuries in high school and college football players. Am J Sports Med. 2007;35(7):1075–81.
7. Turner M, McCrory P, Halley W. Injuries in professional horse racing in Great Britain and the Republic of Ireland during 1992-2000. Br J Sports Med. 2002;36(6):403–9.
8. McNarry AF, Goldhill DR. Simple bedside assessment of level of consciousness: comparison of two simple assessment scales with the Glasgow Coma scale. Anaesthesia. 2004;59(1):34–7.
9. Teasdale G, Jennett B. Assessment of coma and impaired consciousness. A practical scale. Lancet. 1974;2(7872):81–4.
10. Carmont MR, Mahattanakul W, Pigott T. Acquisition of a chronic subdural haematoma during training for competitive race walking? Br J Sports Med. 2002; 36(4):306–7.
11. Makdissi M, Cantu RC, Johnston KM, McCrory P, Meeuwisse WH. The difficult concussion patient: what is the best approach to investigation and management of persistent (>10 days) postconcussive symptoms? Br J Sports Med. 2013;47(5):308–13.
12. Aubry M, Cantu R, Dvorak J, et al. Summary and agreement statement of the first international conference on concussion in sport, Vienna 2001. Phys Sports Med. 2002;30(2):57–63.
13. McCrory P, Johnston K, Meeuwisse W, et al. Summary and agreement statement of the 2nd International Conference on Concussion in Sport, Prague 2004. Br J Sports Med. 2005;39(4):196–204.
14. McCrory P, Meeuwisse W, Johnston K, et al. Consensus statement on Concussion in Sport: the 3rd International Conference on Concussion in sport held in Zurich, November 2008. Br J Sports Med. 2009;43 Suppl 1:i76–90.
15. McCrory P, Meeuwisse WH, Aubry M, et al. Consensus statement on concussion in sport: the 4th International Conference on Concussion in Sport held in Zurich, November 2012. Br J Sports Med. 2013;47(5):250–8.
16. Harmon KG, Drezner J, Gammons M, et al. American Medical Society for Sports Medicine position statement: concussion in sport. Clin J Sport Med. 2013;23(1):1–18.
17. Giza CC, Kutcher JS, Ashwal S, et al. Summary of evidence-based guideline update: evaluation and management of concussion in sports: report of the Guideline Development Subcommittee of the American Academy of Neurology. Neurology. 2013;80(24):2250–7.

18. Maddocks DL, Dicker GD, Saling MM. The assessment of orientation following concussion in athletes. Clin J Sport Med. 1995;5(1):32–5.
19. Graw BP, Wiesel SW. Low back pain in the aging athlete. Sports Med Arthrosc. 2008;16(1):39–46.
20. Purcell L, Micheli L. Low back pain in young athletes. Sports Health. 2009;1(3):212–22.
21. Micheli LJ, Wood R. Back pain in young athletes. Significant differences from adults in causes and patterns. Arch Pediatr Adolesc Med. 1995;149(1):15–8.
22. Granhed H, Morelli B. Low back pain among retired wrestlers and heavyweight lifters. Am J Sports Med. 1988;16(5):530–3.
23. Brolinson PG, Kozar AJ, Cibor G. Sacroiliac joint dysfunction in athletes. Curr Sports Med Rep. 2003;2(1):47–56.
24. Eck JC, Riley 3rd LH. Return to play after lumbar spine conditions and surgeries. Clin Sports Med. 2004;23(3):367–79. viii.
25. Ward CV, Latimer B. Human evolution and the development of spondylolysis. Spine (Phila Pa 1976). 2005;30(16):1808–14.
26. d'Hemecourt PA, Gerbino 2nd PG, Micheli LJ. Back injuries in the young athlete. Clin Sports Med. 2000;19(4):663–79.
27. Goldstein JD, Berger PE, Windler GE, Jackson DW. Spine injuries in gymnasts and swimmers. An epidemiologic investigation. Am J Sports Med. 1991;19(5):463–8.
28. Greene HS, Cholewicki J, Galloway MT, Nguyen CV, Radebold A. A history of low back injury is a risk factor for recurrent back injuries in varsity athletes. Am J Sports Med. 2001;29(6):795–800.
29. van der Windt DA, Simons E, Riphagen II, et al. Physical examination for lumbar radiculopathy due to disc herniation in patients with low-back pain. Cochrane Database Syst Rev. 2010;(2):CD007431.
30. Casazza BA. Diagnosis and treatment of acute low back pain. Am Fam Physician. 2012;85(4):343–50.
31. Henschke N, Maher CG, Refshauge KM, et al. Prevalence of and screening for serious spinal pathology in patients presenting to primary care settings with acute low back pain. Arthritis Rheum. 2009;60(10):3072–80.
32. Lawrence JP, Greene HS, Grauer JN. Back pain in athletes. J Am Acad Orthop Surg. 2006;14(13):726–35.
33. Sairyo K, Katoh S, Takata Y, et al. MRI signal changes of the pedicle as an indicator for early diagnosis of spondylolysis in children and adolescents: a clinical and biomechanical study. Spine (Phila Pa 1976). 2006;31(2):206–11.
34. Zukotynski K, Curtis C, Grant FD, Micheli L, Treves ST. The value of SPECT in the detection of stress injury to the pars interarticularis in patients with low back pain. J Orthop Surg Res. 2010;5:13.
35. Petering RC, Webb C. Treatment options for low back pain in athletes. Sports Health. 2011;3(6):550–5.
36. Burns SA, Foresman E, Kraycsir SJ, et al. A treatment-based classification approach to examination and intervention of lumbar disorders. Sports Health. 2011;3(4):362–72.
37. Long A, May S, Fung T. Specific directional exercises for patients with low back pain: a case series. Physiother Can. 2008;60(4):307–17.
38. Kobayashi A, Kobayashi T, Kato K, Higuchi H, Takagishi K. Diagnosis of radiographically occult lumbar spondylolysis in young athletes by magnetic resonance imaging. Am J Sports Med. 2013; 41(1):169–76.
39. Pfirrmann CW, Metzdorf A, Zanetti M, Hodler J, Boos N. Magnetic resonance classification of lumbar intervertebral disc degeneration. Spine (Phila Pa 1976). 2001;26(17):1873–8.
40. de Schepper EI, Damen J, van Meurs JB, et al. The association between lumbar disc degeneration and low back pain: the influence of age, gender, and individual radiographic features. Spine (Phila Pa 1976). 2010;35(5):531–6.
41. Li Y, Hresko MT. Lumbar spine surgery in athletes: outcomes and return-to-play criteria. Clin Sports Med. 2012;31(3):487–98.
42. Siepe CJ, Wiechert K, Khattab MF, Korge A, Mayer HM. Total lumbar disc replacement in athletes: clinical results, return to sport and athletic performance. Eur Spine J. 2007;16(7):1001–13.
43. Tumialan LM, Ponton RP, Garvin A, Gluf WM. Arthroplasty in the military: a preliminary experience with ProDisc-C and ProDisc-L. Neurosurg Focus. 2010;28(5):E18.
44. Hsu WK, McCarthy KJ, Savage JW, et al. The Professional Athlete Spine Initiative: outcomes after lumbar disc herniation in 342 elite professional athletes. Spine J. 2011;11(3):180–6.
45. Anakwenze OA, Namdari S, Auerbach JD, et al. Athletic performance outcomes following lumbar discectomy in professional basketball players. Spine (Phila Pa 1976). 2010;35(7):825–8.
46. Wang JC, Shapiro MS, Hatch JD, Knight J, Dorey FJ, Delamarter RB. The outcome of lumbar discectomy in elite athletes. Spine (Phila Pa 1976). 1999;24(6):570–3.
47. Watkins RG, Williams LA, Watkins 3rd RG. Microscopic lumbar discectomy results for 60 cases in professional and Olympic athletes. Spine J. 2003;3(2):100–5.
48. Leone A, Cianfoni A, Cerase A, Magarelli N, Bonomo L. Lumbar spondylolysis: a review. Skeletal Radiol. 2011;40(6):683–700.
49. Masci L, Pike J, Malara F, Phillips B, Bennell K, Brukner P. Use of the one-legged hyperextension test and magnetic resonance imaging in the diagnosis of active spondylolysis. Br J Sports Med. 2006;40(11):940–6. discussion 946.
50. Steiner ME, Micheli LJ. Treatment of symptomatic spondylolysis and spondylolisthesis with the modified Boston brace. Spine (Phila Pa 1976). 1985;10(10):937–43.
51. Anderson K, Sarwark JF, Conway JJ, Logue ES, Schafer MF. Quantitative assessment with SPECT imaging of stress injuries of the pars interarticularis and response to bracing. J Pediatr Orthop. 2000; 20(1):28–33.

52. Nozawa S, Shimizu K, Miyamoto K, Tanaka M. Repair of pars interarticularis defect by segmental wire fixation in young athletes with spondylolysis. Am J Sports Med. 2003;31(3):359–64.
53. Debnath UK, Freeman BJ, Gregory P, de la Harpe D, Kerslake RW, Webb JK. Clinical outcome and return to sport after the surgical treatment of spondylolysis in young athletes. J Bone Joint Surg Br. 2003;85(2):244–9.
54. Radcliff KE, Kalantar SB, Reitman CA. Surgical management of spondylolysis and spondylolisthesis in athletes: indications and return to play. Curr Sports Med Rep. 2009;8(1):35–40.
55. Skaggs DL, Avramis I, Myung K, Weiss J. Sacral facet fractures in elite athletes. Spine (Phila Pa 1976). 2012;37(8):E514–517.
56. Richardson CA, Snijders CJ, Hides JA, Damen L, Pas MS, Storm J. The relation between the transversus abdominis muscles, sacroiliac joint mechanics, and low back pain. Spine (Phila Pa 1976). 2002;27(4):399–405.
57. Crowley J, Jordan J, Falvey E. A comparison of Gaelic football injuries in males and females in primary care. Ir Med J. 2011;104(9):268–70.
58. Franklin RC, Leggat PA. The epidemiology of injury in canoeing, kayaking and rafting. Med Sport Sci. 2012;58:98–111.
59. Ekstrand J, Askling C, Magnusson H, Mithoefer K. Return to play after thigh muscle injury in elite football players: implementation and validation of the Munich muscle injury classification. Br J Sports Med. 2013;47(12):769–74.
60. Hagglund M, Walden M, Ekstrand J. Risk factors for lower extremity muscle injury in professional soccer: the UEFA Injury Study. Am J Sports Med. 2013;41(2):327–35.
61. Ranson C, Hurley R, Rugless L, Mansingh A, Cole J. International cricket injury surveillance: a report of five teams competing in the ICC Cricket World Cup 2011. Br J Sports Med. 2013;47(10):637–43.
62. Lopez Jr V, Galano GJ, Black CM, et al. Profile of an American amateur rugby union sevens series. Am J Sports Med. 2012;40(1):179–84.
63. Chung WM, Yeung S, Wong AY, et al. Musculoskeletal injuries in elite able-bodied and wheelchair foil fencers – a pilot study. Clin J Sport Med. 2012;22(3):278–80.
64. Orchard JW, Seward H, Orchard JJ. Results of 2 decades of injury surveillance and public release of data in the Australian Football League. Am J Sports Med. 2013;41(4):734–41.
65. Grapton X, Lion A, Gauchard GC, Barrault D, Perrin PP. Specific injuries induced by the practice of trampoline, tumbling and acrobatic gymnastics. Knee Surg Sports Traumatol Arthrosc. 2013;21(2):494–9.
66. Maffulli N, Longo UG, Maffulli GD, Khanna A, Denaro V. Achilles tendon ruptures in elite athletes. Foot Ankle Int. 2011;32(1):9–15.
67. Langer PR, Selesnick FH. Floating patella: combined quadriceps tendon, retinacula, and patellar tendon ruptures in a high-performance elite athlete. Am J Orthop (Belle Mead NJ). 2010;39(9):446–9.
68. Konan S, Haddad F. Successful return to high level sports following early surgical repair of complete tears of the proximal hamstring tendons. Int Orthop. 2010;34(1):119–23.
69. Kokkalis ZT, Sotereanos DG. Biceps tendon injuries in athletes. Hand Clin. 2009;25(3):347–57.
70. Peetrons P. Ultrasound of muscles. Eur Radiol. 2002;12(1):35–43.
71. Takebayashi S, Takasawa H, Banzai Y, et al. Sonographic findings in muscle strain injury: clinical and MR imaging correlation. J Ultrasound Med. 1995;14(12):899–905.
72. O'Donoghue DH. Introduction. Treatment of injuries to athletes. Clin Orthop Relat Res. 1962;402:3–8.
73. Stoller DW. MRI in Orthopaedic & Sports Medicine. 3rd edition. Philadelphia: Wolters Kluwer/Lippincott 2007.
74. Mueller-Wohlfahrt HW, Haensel L, Mithoefer K, et al. Terminology and classification of muscle injuries in sport: the Munich consensus statement. Br J Sports Med. 2013;47(6):342–50.
75. Morelli V, Weaver V. Groin injuries and groin pain in athletes: part 1. Prim Care. 2005;32(1):163–83.
76. Holmich P, Larsen K, Krogsgaard K, Gluud C. Exercise program for prevention of groin pain in football players: a cluster-randomized trial. Scand J Med Sci Sports. 2010;20(6):814–21.
77. Branci S, Thorborg K, Nielsen MB, Holmich P. Radiological findings in symphyseal and adductor-related groin pain in athletes: a critical review of the literature. Br J Sports Med. 2013;47(10):611–9.
78. Faltus J, Boggess B, Bruzga R. The use of diagnostic musculoskeletal ultrasound to document soft tissue treatment mobilization of a quadriceps femoris muscle tear: a case report. Int J Sports Phys Ther. 2012;7(3):342–9.
79. Opar DA, Williams MD, Shield AJ. Hamstring strain injuries: factors that lead to injury and re-injury. Sports Med. 2012;42(3):209–26.
80. Orchard JW. Hamstrings are most susceptible to injury during the early stance phase of sprinting. Br J Sports Med. 2012;46(2):88–9.
81. Engebretsen AH, Myklebust G, Holme I, Engebretsen L, Bahr R. Intrinsic risk factors for acute knee injuries among male football players: a prospective cohort study. Scand J Med Sci Sports. 2011;21(5):645–52.
82. Armfield DR, Kim DH, Towers JD, Bradley JP, Robertson DD. Sports-related muscle injury in the lower extremity. Clin Sports Med. 2006;25(4):803–42.
83. Hagglund M, Walden M, Magnusson H, Kristenson K, Bengtsson H, Ekstrand J. Injuries affect team performance negatively in professional football: an 11-year follow-up of the UEFA Champions League injury study. Br J Sports Med. 2013;47(12):738–42.
84. Arner O, Lindholm A. What is tennis leg. Acta Chir Scand. 1958;116(1):73–7.
85. Delgado GJ, Chung CB, Lektrakul N, et al. Tennis leg: clinical US study of 141 patients and anatomic investigation of four cadavers with MR imaging and US. Radiology. 2002;224(1):112–9.

86. Bengtsson H, Ekstrand J, Walden M, Hagglund M. Match injury rates in professional soccer vary with match result, match venue, and type of competition. Am J Sports Med. 2013;41(7):1505–10.
87. Magno e Silva M, Bilzon J, Duarte E, Gorla J, Vital R. Sport injuries in elite paralympic swimmers with visual impairment. J Athl Train. 2013;48(4):493–8.
88. Brooks JH, Kemp SP. Recent trends in rugby union injuries. Clin Sports Med. 2008;27(1):51–73, vii-viii.
89. Williams S, Trewartha G, Kemp S, Stokes K. A meta-analysis of injuries in senior men's professional Rugby Union. Sports Med. 2013;43(10):1043–55.
90. Abrams GD, Renstrom PA, Safran MR. Epidemiology of musculoskeletal injury in the tennis player. Br J Sports Med. 2012;46(7):492–8.
91. Emery CA, Meeuwisse WH. Risk factors for groin injuries in hockey. Med Sci Sports Exerc. 2001;33(9):1423–33.
92. Rizzo Jr TD. Using RICE for injury relief. Phys Sports Med. 1996;24(10):33–4.
93. Jarvinen TA, Jarvinen TL, Kaariainen M, Kalimo H, Jarvinen M. Muscle injuries: biology and treatment. Am J Sports Med. 2005;33(5):745–64.
94. Schache A. Eccentric hamstring muscle training can prevent hamstring injuries in soccer players. J Physiother. 2012;58(1):58.
95. Petersen J, Thorborg K, Nielsen MB, Budtz-Jorgensen E, Holmich P. Preventive effect of eccentric training on acute hamstring injuries in men's soccer: a cluster-randomized controlled trial. Am J Sports Med. 2011;39(11):2296–303.
96. O'Grady M, Hackney AC, Schneider K, et al. Diclofenac sodium (Voltaren) reduced exercise-induced injury in human skeletal muscle. Med Sci Sports Exerc. 2000;32(7):1191–6.
97. Mishra DK, Friden J, Schmitz MC, Lieber RL. Anti-inflammatory medication after muscle injury. A treatment resulting in short-term improvement but subsequent loss of muscle function. J Bone Joint Surg Am. 1995;77(10):1510–9.
98. Karnes JL, Burton HW. Continuous therapeutic ultrasound accelerates repair of contraction-induced skeletal muscle damage in rats. Arch Phys Med Rehabil. 2002;83(1):1–4.
99. Rantanen J, Thorsson O, Wollmer P, Hurme T, Kalimo H. Effects of therapeutic ultrasound on the regeneration of skeletal myofibers after experimental muscle injury. Am J Sports Med. 1999;27(1):54–9.
100. Montalti CS, Souza NV, Rodrigues NC, Fernandes KR, Toma RL, Renno AC. Effects of low-intensity pulsed ultrasound on injured skeletal muscle. Braz J Phys Ther. 2013;17(4):343–50.
101. Best TM, Loitz-Ramage B, Corr DT, Vanderby R. Hyperbaric oxygen in the treatment of acute muscle stretch injuries. Results in an animal model. Am J Sports Med. 1998;26(3):367–72.
102. Bennett M, Best TM, Babul S, Taunton J, Lepawsky M. Hyperbaric oxygen therapy for delayed onset muscle soreness and closed soft tissue injury. Cochrane Database Syst Rev. 2005;(4):CD004713
103. Mostafavifar M, Wertz J, Borchers J. A systematic review of the effectiveness of kinesio taping for musculoskeletal injury. Phys Sports Med. 2012;40(4):33–40.
104. Harmon KG. Muscle injuries and PRP: what does the science say? Br J Sports Med. 2010;44(9):616–7.
105. Andia I, Sanchez M, Maffulli N. Platelet rich plasma therapies for sports muscle injuries: any evidence behind clinical practice? Expert Opin Biol Ther. 2011;11(4):509–18.
106. Engebretsen L, Steffen K. To PRP or not? Br J Sports Med. 2010;44(15):1071.
107. Torrance DA, Degraauw C. Treatment of post-traumatic myositis ossificans of the anterior thigh with extracorporeal shock wave therapy. J Can Chiropr Assoc. 2011;55(4):240–6.
108. Wieder DL. Treatment of traumatic myositis ossificans with acetic acid iontophoresis. Phys Ther. 1992;72(2):133–7.
109. Gard K, Ebaugh D. The use of acetic Acid iontophoresis in the management of a soft tissue injury. N Am J Sports Phys Ther. 2010;5(4):220–6.
110. Holmich P, Uhrskou P, Ulnits L, et al. Effectiveness of active physical training as treatment for long-standing adductor-related groin pain in athletes: randomised trial. Lancet. 1999;353(9151):439–43.
111. Bizzini M, Junge A, Dvorak J. Implementation of the FIFA 11+ football warm up program: how to approach and convince the football associations to invest in prevention. Br J Sports Med. 2013;47(12):803–6.
112. Grooms DR, Palmer T, Onate JA, Myer G, Grindstaff T. Comprehensive soccer-specific warm-up and lower extremity injury in collegiate male soccer players. J Athl Train. 2013;48:782–9.
113. Steffen K, Emery CA, Romiti M, et al. 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(12):794–802.
114. Longo UG, Loppini M, Berton A, Marinozzi A, Maffulli N, Denaro V. The FIFA 11+ program is effective in preventing injuries in elite male basketball players: a cluster randomized controlled trial. Am J Sports Med. 2012;40(5):996–1005.
115. Wilkerson GB, Giles JL, Seibel DK. Prediction of core and lower extremity strains and sprains in collegiate football players: a preliminary study. J Athl Train. 2012;47(3):264–72.
116. Zhu Y, Yuan M, Meng HY, et al. Basic science and clinical application of platelet-rich plasma for cartilage defects and osteoarthritis: a review. Osteoarthritis Cartilage. 2013;21:1627–37.
117. Amable PR, Carias RB, Teixeira MV, et al. Platelet-rich plasma preparation for regenerative medicine: optimization and quantification of cytokines and growth factors. Stem Cell Res Ther. 2013;4(3):67.
118. Anitua E, Andia I, Ardanza B, Nurden P, Nurden AT. Autologous platelets as a source of proteins for

healing and tissue regeneration. Thromb Haemost. 2004;91(1):4–15.
119. Martinez-Zapata MJ, Marti-Carvajal AJ, Sola I, et al. Autologous platelet-rich plasma for treating chronic wounds. Cochrane Database Syst Rev. 2012;(10):CD006899.
120. Mei-Dan O, Lippi G, Sanchez M, Andia I, Maffulli N. Autologous platelet-rich plasma: a revolution in soft tissue sports injury management? Phys Sportsmed. 2010;38(4):127–35.
121. Anitua E, Sanchez M, Nurden AT, Nurden P, Orive G, Andia I. New insights into and novel applications for platelet-rich fibrin therapies. Trends Biotechnol. 2006;24(5):227–34.
122. Mazzocca AD, McCarthy MB, Chowaniec DM, et al. Platelet-rich plasma differs according to preparation method and human variability. J Bone Joint Surg Am. 2012;94(4):308–16.
123. Langer R, Vacanti J. Tissue engineering. Science. 1993;260:920–6.
124. Pereira H, Frias AM, Oliveira JM, Espregueira-Mendes J, Reis RL. Tissue engineering and regenerative medicine strategies in meniscus lesions. Arthroscopy. 2011;27(12):1706–19.
125. Sheth U, Simunovic N, Klein G, et al. Efficacy of autologous platelet-rich plasma use for orthopaedic indications: a meta-analysis. J Bone Joint Surg Am. 2012;94(4):298–307.
126. Arnoczky SP, Delos D, Rodeo SA. What is platelet-rich plasma? Oper Tech Sports Med. 2011;19:142–8.
127. Foster TE, Puskas BL, Mandelbaum BR, Gerhardt MB, Rodeo SA. Platelet-rich plasma: from basic science to clinical applications. Am J Sports Med. 2009;37(11):2259–72.
128. Nikolidakis D, Jansen JA. The biology of platelet-rich plasma and its application in oral surgery: literature review. Tissue Eng Part B Rev. 2008;14(3):249–58.
129. Lopez-Vidriero E, Goulding KA, Simon DA, Sanchez M, Johnson DH. The use of platelet-rich plasma in arthroscopy and sports medicine: optimizing the healing environment. Arthroscopy. 2010; 26(2):269–78.
130. Mann KG. Biochemistry and physiology of blood coagulation. Thrombosis Haemost. 1999;82(2):165–74.
131. Bielecki TM, Gazdzik TS, Arendt J, Szczepanski T, Krol W, Wielkoszynski T. Antibacterial effect of autologous platelet gel enriched with growth factors and other active substances: an in vitro study. J Bone Joint Surg Br. 2007;89(3):417–20.
132. Moojen DJ, Everts PA, Schure RM, et al. Antimicrobial activity of platelet-leukocyte gel against Staphylococcus aureus. J Orthop Res. 2008; 26(3):404–10.
133. Tidball JG. Inflammatory processes in muscle injury and repair. Am J Physiol Regul Integr Comp Physiol. 2005;288(2):R345–353.
134. Marx RE. Platelet-rich plasma: evidence to support its use. J Oral Maxillofac Surg. 2004;62(4):489–96.
135. Chen L, Yang X, Huang G, et al. Platelet-rich plasma promotes healing of osteoporotic fractures. Orthopedics. 2013;36(6):e687–694.
136. Evans CH. Platelet-rich plasma a la carte: commentary on an article by Satoshi Terada, MD, et al.: "use of an antifibrotic agent improves the effect of platelet-rich plasma on muscle healing after injury". J Bone Joint Surg Am. 2013;95(11):e801–802.
137. Nguyen RT, Borg-Stein J, McInnis K. Applications of platelet-rich plasma in musculoskeletal and sports medicine: an evidence-based approach. PM R. 2011;3(3):226–50.
138. Tinsley BA, Ferreira JV, Dukas AG, Mazzocca AD. Platelet-rich plasma nonoperative injection therapy—a review of indications and evidence. Oper Techn Sports Med. 2012;20(2):192–200.
139. Papalia R, Vasta S, Zampogna B, Tecam A, Maffulli N. Platelet-rich plasma injections and surgery: short-term outcomes and long-term prognosis. Oper Tech Orthop. 2012;22(2):71–7.
140. Zhang Q, Ge H, Zhou J, Cheng B. Are platelet-rich products necessary during the arthroscopic repair of full-thickness rotator cuff tears: a meta-analysis. PLoS One. 2013;8(7):e69731.
141. Mishra A, Pavelko T. Treatment of chronic elbow tendinosis with buffered platelet-rich plasma. Am J Sports Med. 2006;34(11):1774–8.
142. Owens Jr RF, Ginnetti J, Conti SF, Latona C. Clinical and magnetic resonance imaging outcomes following platelet rich plasma injection for chronic midsubstance Achilles tendinopathy. Foot Ankle Int. 2011;32(11):1032–9.
143. de Vos RJ, Weir A, van Schie HT, et al. Platelet-rich plasma injection for chronic Achilles tendinopathy: a randomized controlled trial. JAMA. 2010;303(2):144–9.
144. de Almeida AM, Demange MK, Sobrado MF, Rodrigues MB, Pedrinelli A, Hernandez AJ. Patellar tendon healing with platelet-rich plasma: a prospective randomized controlled trial. Am J Sports Med. 2012;40(6):1282–8.
145. Kon E, Filardo G, Delcogliano M, et al. Platelet-rich plasma: new clinical application: a pilot study for treatment of jumper's knee. Injury. 2009; 40(6):598–603.
146. Silva A, Sampaio R. Anatomic ACL reconstruction: does the platelet-rich plasma accelerate tendon healing? Knee Surg Sports Traumatol Arthrosc. 2009;17(6):676–82.
147. Orrego M, Larrain C, Rosales J, et al. Effects of platelet concentrate and a bone plug on the healing of hamstring tendons in a bone tunnel. Arthroscopy. 2008;24(12):1373–80.
148. Kim E, Lee JH. Autologous platelet-rich plasma versus dextrose prolotherapy for the treatment of chronic recalcitrant plantar fasciitis. PM R. 2013. pii: S1934-1482(13)00369-9. doi:10.1016/j.pmrj.2013.07.003.
149. Kon E, Buda R, Filardo G, et al. Platelet-rich plasma: intra-articular knee injections produced favorable results on degenerative cartilage lesions. Knee Surg Sports Traumatol Arthrosc. 2010;18(4):472–9.
150. Wei LC, Lei GH, Sheng PY, et al. Efficacy of platelet-rich plasma combined with allograft bone in the management of displaced intra-articular

calcaneal fractures: a prospective cohort study. J Orthop Res. 2012;30(10):1570–6.
151. Borrione P, Gianfrancesco AD, Pereira MT, Pigozzi F. Platelet-rich plasma in muscle healing. Am J Phys Med Rehabil. 2010;89(10):854–61.
152. Engebretsen L, Schamasch S. The use of platelet-rich plasma in sports medicine—the International Olympic Committee Opinion. Oper Tech Orthop. 2012;22(1):43–8.
153. WADA. WADA the World Anti-Doping Code. The prohibited list 2011. Montreal: World Anti-Doping Agency; 2011.
154. Engebretsen L, Steffen K, Alsousou J, et al. IOC consensus paper on the use of platelet-rich plasma in sports medicine. Br J Sports Med. 2010;44(15):1072–81.
155. Park YG, Han SB, Song SJ, Kim TJ, Ha CW. Platelet-rich plasma therapy for knee joint problems: review of the literature, current practice and legal perspectives in Korea. Knee Surg Relat Res. 2012;24(2):70–8.
156. Terada S, Ota S, Kobayashi M, et al. Use of an antifibrotic agent improves the effect of platelet-rich plasma on muscle healing after injury. J Bone Joint Surg Am. 2013;95(11):980–8.
157. López-Ruiz E, Perán M, Cobo-Molinos J, et al. Chondrocytes extract from patients with osteoarthritis induces chondrogenesis in infrapatellar fat pad-derived stem cells. Osteoarthr Cartil. 2013;21(1):246–58.
158. Verfaillie CM. Adult stem cells: assessing the case for pluripotency. Trends Cell Biol. 2002;12(11):502–8.
159. Cananzi M, Atala A, De Coppi P. Stem cells derived from amniotic fluid: new potentials in regenerative medicine. Reprod BioMed Online. 2009;18(Supplement 1):17–27.
160. Okita K, Ichisaka T, Yamanaka S. Generation of germline-competent induced pluripotent stem cells. Nature. 2007;448(7151):313–7.
161. Takahashi K, Yamanaka S. Induction of pluripotent stem cells from mouse embryonic and adult fibroblast cultures by defined factors. Cell. 2006;126(4):663–76.
162. Li M, Chen M, Han W, Fu X. How far are induced pluripotent stem cells from the clinic? Ageing Res Rev. 2010;9(3):257–64.
163. Kim S-J, Shettey AA. (ii) Stem cell research in orthopaedic and trauma surgery. Orthop Trauma. 2011;25(3):168–73.
164. Im II G, Shin Y-W, Lee K-B. Do adipose tissue-derived mesenchymal stem cells have the same osteogenic and chondrogenic potential as bone marrow-derived cells? Osteoarthr Cartil. 2005;13(10):845–53.
165. Muzzarelli RAA, Greco F, Busilacchi A, Sollazzo V, Gigante A. Chitosan, hyaluronan and chondroitin sulfate in tissue engineering for cartilage regeneration: a review. Carbohydr Polym. 2012;89(3):723–39.
166. Šoltés L, Mendichi R, Machovà E, et al. Cyclodextrin derivative of hyaluronan. Carbohydr Polym. 1999;39(1):17–24.
167. Tytherleigh-Strong G, Hurtig M, Miniaci A. Intra-articular hyaluronan following autogenous osteochondral grafting of the knee. Arthroscopy. 2005;21(8):999–1005.
168. Wobig M, Dickhut A, Maier R, Vetter G. Viscosupplementation with Hylan G-F 20: a 26-week controlled trial of efficacy and safety in the osteoarthritic knee. Clin Ther. 1998;20(3):410–23.
169. McGuire DA, Carter TR, Shelton WR. Complex knee reconstruction: osteotomies, ligament reconstruction, transplants, and cartilage treatment options. Arthroscopy. 2002;18(9, Supplement 2):90–103.
170. Mirabella T, Gentili C, Daga A, Cancedda R. Amniotic fluid stem cells in a bone microenvironment: driving host angiogenic response. Stem Cell Res. 2013;11(1):540–51.
171. van Buul GM, Villafuertes E, Bos PK, et al. Mesenchymal stem cells secrete factors that inhibit inflammatory processes in short-term osteoarthritic synovium and cartilage explant culture. Osteoarthr Cartil. 2012;20(10):1186–96.
172. Bagaria V, Patil N, Sapre V, Chadda A, Singrakia M. Stem cells in orthopedics: current concepts and possible future applications. Indian J Med Sci. 2006; 60(4):162–9.
173. Miller RH, Bai L. Translating stem cell therapies to the clinic. Neurosci Lett. 2012;519(2):87–92.
174. Barry F, Murphy M. Mesenchymal stem cells in joint disease and repair. Nat Rev Rheumatol. 2013;9(10):584–94. doi:10.1038/nrrheum.2013.109.
175. Lubowitz JH, Provencher MT, Poehling GG. Stem cells in the knee. Arthroscopy. 2013;29(4):609–10.
176. McGonagle D, English A, Jones EA. (iii) The relevance of mesenchymal stem cells in vivo for future orthopaedic strategies aimed at fracture repair. Curr Orthop. 2007;21(4):262–7.
177. Koh Y-G, Jo S-B, Kwon O-R, et al. Mesenchymal stem cell injections improve symptoms of knee osteoarthritis. Arthroscopy. 2013;29(4):748–55.
178. Kasemkijwattana C, Hongeng S, Kesprayura S, Rungsinaporn V, Chaipinyo K, Chansiri K. Autologous bone marrow mesenchymal stem cells implantation for cartilage defects: two cases report. J Med Assoc Thai. 2011;94(3):395–400.
179. Centeno CJ, Busse D, Kisiday J, Keohan C, Freeman M, Karli D. Regeneration of meniscus cartilage in a knee treated with percutaneously implanted autologous mesenchymal stem cells. Med Hypotheses. 2008;71(6):900–8.
180. McIlwraith CW, Frisbie DD, Rodkey WG, et al. Evaluation of intra-articular mesenchymal stem cells to augment healing of microfractured chondral defects. Arthroscopy. 2011;27(11):1552–61.
181. Saw K-Y, Anz A, Merican S, et al. Articular cartilage regeneration with autologous peripheral blood progenitor cells and hyaluronic acid after

arthroscopic subchondral drilling: a report of 5 cases with histology. Arthroscopy. 2011;27(4):493–506.
182. Richter W. Cell-based cartilage repair: illusion or solution for osteoarthritis. Curr Opin Rheumatol. 2007;19(5):451–6.
183. Koh Y-G, Choi Y-J. Infrapatellar fat pad-derived mesenchymal stem cell therapy for knee osteoarthritis. Knee. 2012;19(6):902–7.
184. Oliveira JM, Sousa RA, Kotobuki N, et al. The osteogenic differentiation of rat bone marrow stromal cells cultured with dexamethasone-loaded carboxymethylchitosan/poly(amidoamine) dendrimer nanoparticles. Biomaterials. 2009;30(5):804–13.
185. Silva-Correia J, Correia SI, Oliveira JM, Reis RL. Tissue engineering strategies applied in the regeneration of the human intervertebral disk. Biotechnol Adv. 2013;31(8):1514–31. doi:10.1016/j.biotechadv.2013.07.010.
186. Pereira DR, Silva-Correia J, Caridade SG, et al. Development of gellan gum-based microparticles/hydrogel matrices for application in the intervertebral disc regeneration. Tissue Eng Part C Methods. 2011;17(10):961–72.
187. Ohgushi H, Kotobuki N, Funaoka H, et al. Tissue engineered ceramic artificial joint—ex vivo osteogenic differentiation of patient mesenchymal cells on total ankle joints for treatment of osteoarthritis. Biomaterials. 2005;26(22):4654–61.
188. Bertram H, Kroeber M, Wang H, et al. Matrix-assisted cell transfer for intervertebral disc cell therapy. Biochem Biophys Res Commun. 2005;331(4):1185–92.
189. Gan Y, Dai K, Zhang P, Tang T, Zhu Z, Lu J. The clinical use of enriched bone marrow stem cells combined with porous beta-tricalcium phosphate in posterior spinal fusion. Biomaterials. 2008;29(29):3973–82.
190. Marcacci M, Kon E, Moukhachev V, et al. Stem cells associated with macroporous bioceramics for long bone repair: 6- to 7-year outcome of a pilot, clinical study. Tissue Eng. 2007;13(5):947–55.
191. Wegener B, Schrimpf FM, Pietschmann MF, et al. Matrix-guided cartilage regeneration in chondral defects. Biotechnol Appl Biochem. 2009;53(Pt 1):63–70.
192. Yamada Y, Ito K, Nakamura S, Ueda M, Nagasaka T. Promising cell-based therapy for bone regeneration using stem cells from deciduous teeth, dental pulp, and bone marrow. Cell Transplant. 2011;20(7):1003–13.
193. de Girolamo L, Bertolini G, Cervellin M, Sozzi G, Volpi P. Treatment of chondral defects of the knee with one step matrix-assisted technique enhanced by autologous concentrated bone marrow: in vitro characterisation of mesenchymal stem cells from iliac crest and subchondral bone. Injury. 2010;41(11):1172–7.
194. Guyton GP, Miller SD. Stem cells in bone grafting: trinity allograft with stem cells and collagen/beta-tricalcium phosphate with concentrated bone marrow aspirate. Foot Ankle Clin. 2010;15(4):611–9.
195. Richter M, Zech S. Matrix-associated stem cell transplantation (MAST) in chondral defects of foot and ankle is effective. Foot Ankle Surg. 2013;19(2):84–90.

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DOI 10.1007/978-3-642-53983-1, © ESSKA 2014

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