

Pietro Randelli, Vincenza Ragone,  
Alessandra Menon, Paolo Arrigoni, Mauro Ciuffreda,  
Nikica Darabos, Vincenzo Denaro, Michael Hantes,  
Vaso Kecojevich, Umile Giuseppe Longo,  
Mattia Loppini, Olaf Lorbach, Elena Azzalini,  
Nicola Maffulli, Giacomo Rizzello, Paolo Cabitza,  
and Giuseppe Banfi

## Contents

17.1	<b>Introduction</b> .....	211
17.2	<b>Shoulder Injuries</b> .....	212
17.2.1	Rotator Cuff Injuries .....	212
17.2.2	SLAP Lesions and Biceps Pathology .....	213
17.2.3	Glenohumeral Instability .....	214
17.2.4	Acromioclavicular Joint Injuries.....	216
17.2.5	Clavicle Fractures .....	220
17.2.6	Humeral Head Fractures .....	224
17.3	<b>Elbow Injuries</b> .....	224
17.4	<b>Triangular Fibrocartilage Complex Injuries</b> .....	227
	<b>Conclusion</b> .....	228
	<b>References</b> .....	228

P. Randelli (✉)  
Dipartimento di scienze biomediche per la salute,  
Università degli studi di Milano, Milan, Italy

2nd Department of Orthopaedic and Traumatology,  
IRCCS Policlinico San Donato, Milan, Italy  
e-mail: [pietro.randelli@unimi.it](mailto:pietro.randelli@unimi.it)

V. Ragone  
Dipartimento di scienze mediche per la salute,  
IRCCS Policlinico San Donato, San Donato, Italy

A. Menon • E. Azzalini  
2nd Department of Orthopaedic and Traumatology,  
IRCCS Policlinico San Donato, Milan, Italy

P. Arrigoni  
Department of Orthopaedics and Traumatology,  
Policlinico San Donato, Università degli Studi di  
Milano, Milan, Italy

M. Ciuffreda • V. Denaro • U.G. Longo • M. Loppini  
G. Rizzello  
Department of Orthopaedic and Trauma Surgery,  
Campus Bio-Medico University, Rome, Italy

## 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.

N. Darabos  
Clinic for Traumatology Clinical Hospital Center  
Sisters of Charity Medical School University of  
Zagreb, Zagreb, Croatia

M. Hantes  
Department of Orthopaedic Surgery,  
University of Thessalia, University Hospital  
of Larissa, Volos, Greece

V. Kecojevich  
Department of Orthopaedic Surgery and Traumatology,  
Clinical Center Vojvodina, University of Novi Sad,  
Novi Sad, Serbia

O. Lorbach  
Department of Orthopedic Surgery,  
Saarland University, Saarbrücken, Germany

N. Maffulli  
Department of Musculoskeletal Disorders, Centre for  
Sports and Exercise Medicine, Barts and The London  
School of Medicine and Dentistry, London, UK

P. Cabitza  
Dipartimento di scienze biomediche per la salute,  
Università degli studi di Milano, Milan, Italy

G. Banfi  
Dipartimento di scienze mediche per la salute,  
IRCCS Istituto Ortopedico Galeazzi, Milan, Italy

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 against 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 irreparable 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 ranging 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 hemiarthrosis, 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 been described [57, 58]. Finally, the remplissage technique has been proposed to manage the engaging Hill–Sachs lesions by performing a capsulotomodesis 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



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.

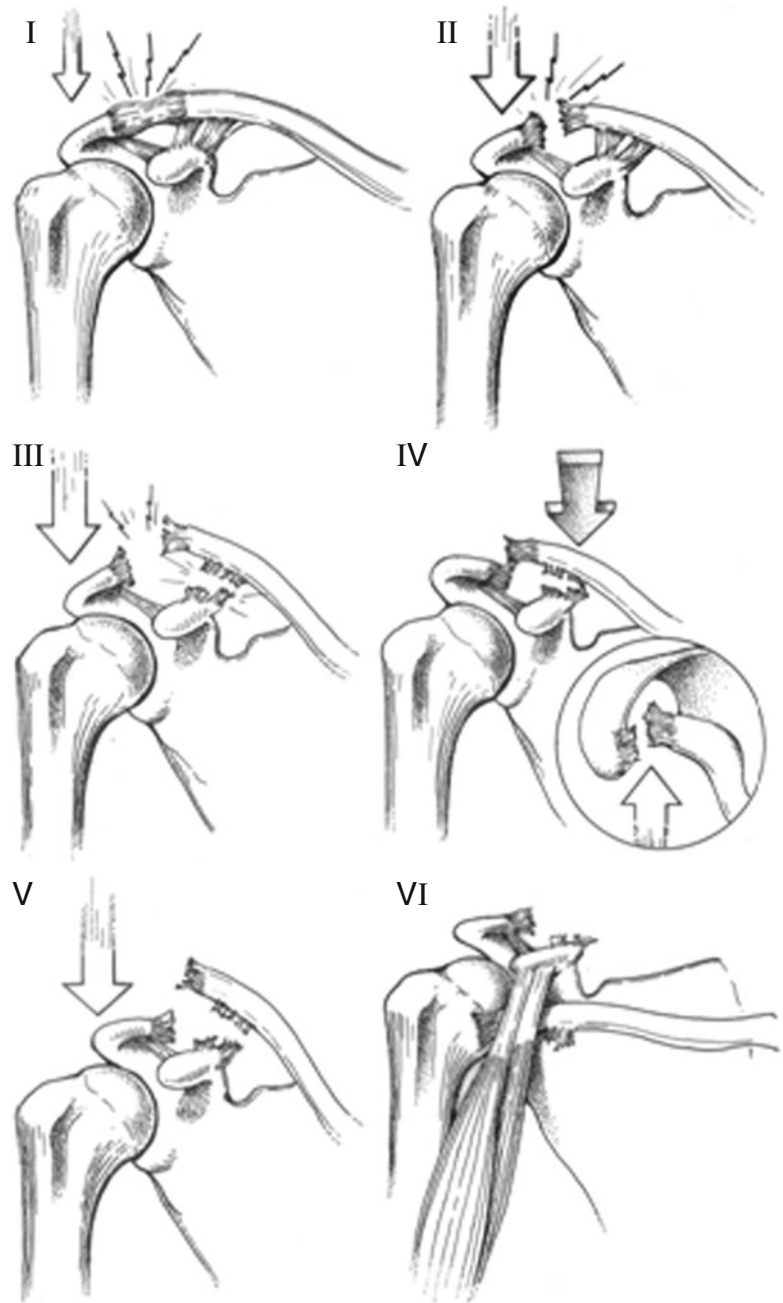
Although there is a possibility for skin and soft tissue-related pitfalls of nonoperative treatment with external immobilization, the most common complication of conservative therapy is a chronic pain that increases under physical activity. It is presented in certain numbers of conservatively treated 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





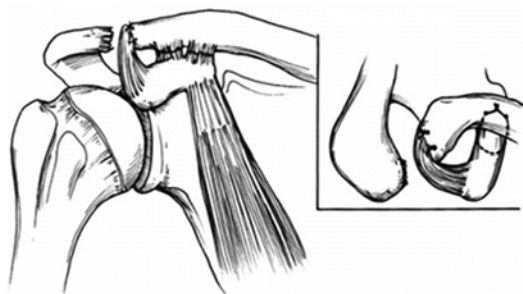
**Fig. 17.3** TightRope fixation

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].



**Fig. 17.4** Modified Weaver–Dunn procedure

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 restraint. 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



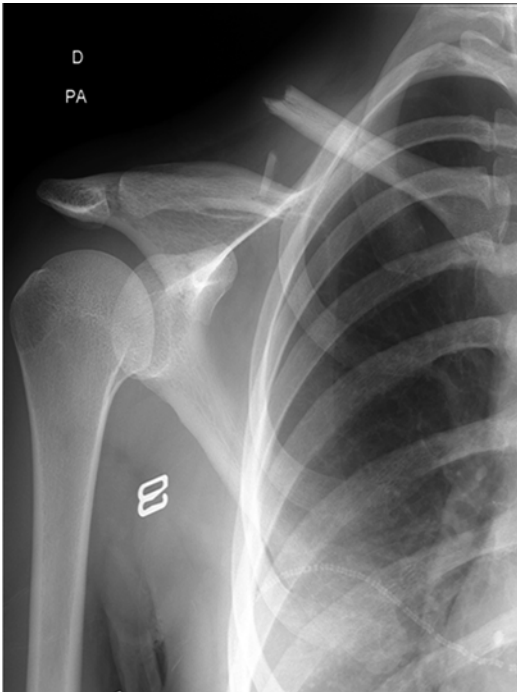
**Fig. 17.6** Lateral third clavicle fracture minimally displaced (3A)



**Fig. 17.7** Comminution of lateral end right clavicle (3B)



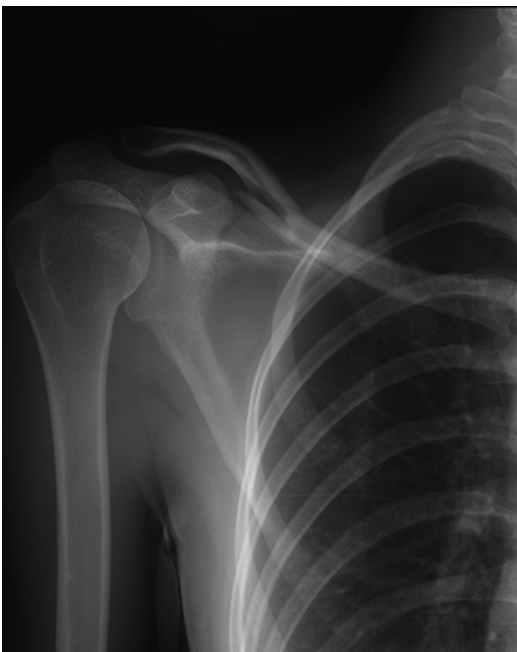
**Fig. 17.5** Midshaft right clavicle fracture with dislocation (2B)



**Fig. 17.8** Severe displacement of middle third, right clavicle fracture



**Fig. 17.10** Lateral end left clavicle fracture, no displacement (3A)



**Fig. 17.9** Midshaft right clavicle fracture minimally displaced (2A)

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 available to perform an 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 micro-trauma 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



**Fig. 17.11** Osteochondritis dissecans of the capitellum (arrows) in a 22-year-old male athlete

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.

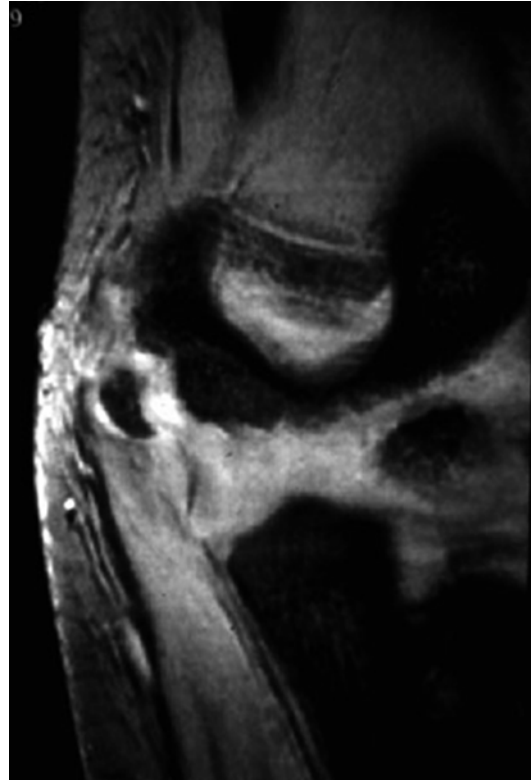
Plain radiographs providing an overview of the osseous structures and of course injuries to the soft tissues are not visualized. Standard views include AP and lateral projections and two oblique views if necessary. Stress views should be obtained if ligament disruption is suspected. Radiographs could 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



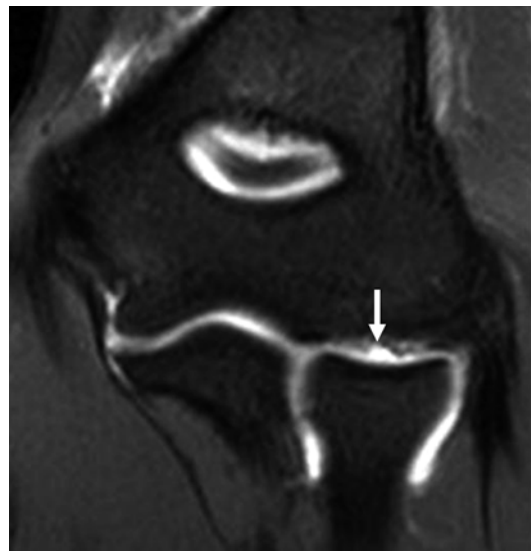
**Fig. 17.12** FS PD TSE coronal MR image demonstrating a partial tear of the MCL

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, Pegreffì 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, Hutten 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. Redirection 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.