

# Sports Injuries of the Shoulder

Lennard Funk  
Mike Walton  
Adam Watts  
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*Editors*

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# Clinical Anatomy and Biomechanics of the Sporting Shoulder

Giulio Maria Marcheggiani Muccioli,  
Carbone Giuseppe, Grassi Alberto,  
Zaffagnini Stefano,  
and Marcacci Maurilio

## Key Learning Points

- The shoulder joint is the most mobile joint in the body; however, it is also the most unstable.
- Strength and stability of the joint are highly dependent on both static and dynamic restraints.
- The constitutional trait of laxity facilitates extensive motion in multiple planes and may be essential to athletic performance.
- The scapulothoracic muscles transfer the potential energy of the trunk to kinetic energy in the shoulder. The scapula is a key link in the kinetic chain between the trunk and the shoulder.

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## 1.1 Introduction

The shoulder complex is an association of 5 joints, 8 ligaments and 30 muscles working together to achieve hand positioning in

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the space. This complex is the most movable of the human body, at the price of a great unstableness [1].

Its movements can be described through anatomic coordinate systems: internal and external rotations are the movements in the transversal plane and they can be described as the rotation around humerus long axis; abduction and adduction are the movements in the frontal plane; flexion and extension are the movements in the sagittal plane. Moreover, the glenohumeral joint can translate in anterior-posterior, superior-inferior and medial-lateral directions. The combination of the elementary movements generates circumduction, described as a complex circle movement, whose trajectory is an irregular cone with the apex centred on the glenohumeral joint.

This large range of motion is possible because of a balanced and synchronised interplay between static and active stabilisers. Static stabilisers include: bony, cartilaginous, capsular and ligamentous factors. Active stabilisers include: muscles (both glenohumeral and scapulothoracic musculature) and neural feedback between capsular and ligamentous structure and muscle.

A slight borderline exists between normal laxity and pathologic instability.

Laxity, defined as asymptomatic translation of humeral head on the glenoid, may be essential to reach good athletic performance, especially in sports that require wide motion of the shoulder. Shoulder instability is defined as an abnormal translation associated with a functional deficit and symptoms like pain and apprehension.

In athletes, glenohumeral instability can occur because of chronic overuse injuries or after an acute traumatic event. In any case, shoulder instability as a result of damage or deficiency in normal shoulder stabilisers is often counterbalanced by neuromuscular control. If it fails, because of acute or chronic worsening, shoulder instability is established.

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## 1.2 Static Stabilisers

The static stabilisers comprise bones, labrum, capsule and the vacuum effect. Static stabilisers could be divided into bony

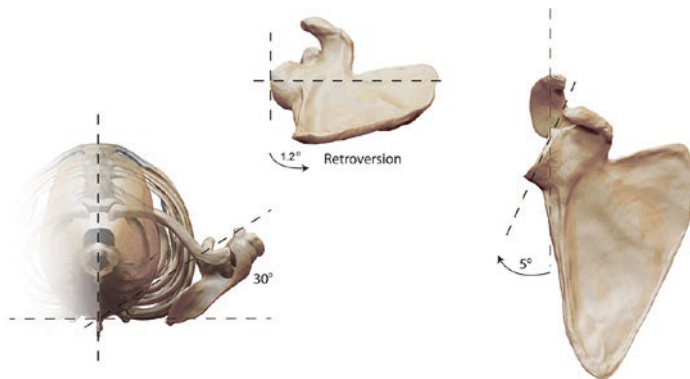


stabilisers (humeral head and glenoid) and soft tissue stabilisers (glenoid labrum, glenohumeral ligaments and joint capsule, rotator interval, negative intracapsular pressure, adhesion cohesion mechanism and acromio-clavicular joint system). They keep the shoulder in joint when at rest.

### 1.2.1 Bony Static Stabilisers

The humeral head is extremely variable in shape and size: it is retroverted on average  $19^\circ$  (range  $9^\circ$  to  $31^\circ$ ) and inclined on average  $41^\circ$  (range  $34$ – $47^\circ$ ); head radius averages 23 mm (range 17–28 mm) and medial and posterior head centre offset are on average 7 mm (range 4–12 mm) and 2 mm (range 1–8 mm), respectively [2] (Fig. 1.1). The humeral head is covered by a layer of hyaline articular cartilage; articular surface ending is lined by the anatomic neck, a bony transition from cartilage to capsular attachment and tendinous insertion. Laterally to the anatomic neck, greater and lesser tuberosity are the insertion point of the rotator cuff tendons, delimit the bicipital groove and help to maintain the long-head biceps in place.

The glenoid is a shallow socket that holds humeral head; its mean depth is 2.5 mm on anteroposterior direction and 9 mm in



**Fig. 1.1** Bony geometry of the scapula and glenoid (courtesy of Lennard Funk, <http://www.shoulderdoc.co.uk>)

superior inferior direction. It is retroverted on average  $1.2^\circ$  (range  $9.5^\circ$  of anteversion to  $10.5^\circ$  of retroversion) and inclined superiorly on average  $5^\circ$  (range  $7^\circ$  of inferior inclination to  $15.8^\circ$  of superior inclination) [3]. Friedman et al. [4] reported that its bending radius is larger than humeral head radius in 93% of examined joints; the remainder have glenoid and humeral head with the same bending radius.

Only a maximum of 30% of the humeral articular surface articulates with glenoid articular surface at any time [5]; bearing in mind the importance of soft tissue static and dynamic restrains in shoulder stability. The glenohumeral ratio shows a dimensional relationship between humeral head and glenoid: it's the result of the division between the maximum diameter of the glenoid and the maximum diameter of the humeral head. It's different according to different planes: 0.75 in the sagittal plane and 0.6 in the coronal plane [6].

All the bony characteristics influence stability, therefore changes in bony anatomy could result in shoulder instability. An excessive retroversion of the glenoid could be a rare cause of posterior instability, but more frequently it is only a contributory factor.

Most important bony lesions that result in instability occur after traumatic events and involve the anterior-inferior glenoid rim and the posterolateral aspect of the humeral head, called a bony Bankart lesion and a Hill–Sachs lesion, respectively (Fig. 1.2).

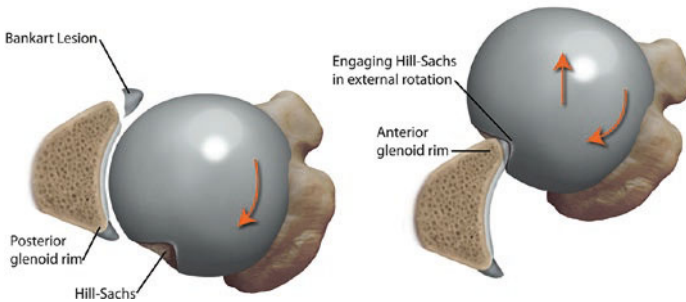
Bony Bankart lesions become significant when they involve more than 20% of the length of the glenoid and are predisposed to recurrence despite correct soft tissues repair; if the bony Bankart lesion involves more than 50% of the length of the glenoid, shoulder stability is reduced by more than 30% [7]. Bony Bankart lesions are classified as described by Bigliani et al. [8]: type I, a displaced avulsion fracture with attached capsule; type II, a medially displaced fragment malunited to the glenoid rim; type III, an erosion of the glenoid rim lower than 25% (III A) and more than 25% (III B). If a bone fragment is present it will be reabsorbed within a year [9]. The PICO method, suggested by Baudi et al. [10], could be used to calculate bone deficiency produced by a bony Bankart lesion: it needs Computed Tomography Multiplanar

**Fig. 1.2**  
Bony Bankart  
lesion (courtesy  
of Lennard  
Funk, [http://  
www.  
shoulderdoc.  
co.uk](http://www.shoulderdoc.co.uk))



Reconstruction of both shoulder and defects and is calculated as a ratio between the surface of the damaged glenoid and the surface of not damaged glenoid.

A Hill–Sachs lesion is an impact fracture occurring after one or more traumatic anterior shoulder dislocations and involves the posterior-lateral articular surface of the humeral head (Fig. 1.3). Smaller Hill–Sachs lesions don't influence stability; the level of influence on shoulder instability depends on the size of lesion and its location. According to their size, Hill–Sachs lesions are classified as mild ( $2 \times 0.3$  cm), moderately severe ( $4 \times 0.5$  cm) and severe ( $>4 \times 0.5$  cm) [11]. In addition, Burkhart and De Beer [12] classified them according to their orientation as engaging or not engaging (the impact fractures that extend to the area of contact between articular surfaces of the glenohumeral joint during abduction, external rotation and extension have a higher risk of engagement). Naturally, risk of engagement is higher if the gle-



**Fig. 1.3** Hill-Sachs lesion of the posterior humeral head (courtesy of Lenard Funk, <http://www.shoulderdoc.co.uk>)

noid surface is reduced. An arthroscopic classification of Hill–Sachs lesions by Calandra et al. [13] can be used to identify 3 types of defects: Grade I, that doesn't involve subchondral bone; Grade II, that involves subchondral bone; Grade III, that involves subchondral bone widely. Similar but specular lesions occur in posterior traumatic instability: the posterior glenoid rim could be fractured after acute traumatic dislocation or eroded after repeated subluxations (reverse bony Bankart lesion) [14] and the humeral head could be fractured in its anterior articular surface (reverse Hill–Sachs lesion or McLaughlin lesion) [15]. Reverse Hill–Sachs lesions could be engaging during adduction, flexion and internal rotation if they extend into the zone of contact between articular surfaces during that motion [16].

Considering bony stabilisers, it's important to underline the glenoid track concept, defined as a contact area between glenoid and humeral head, created by shifting of the glenoid from the inferomedial to the posterolateral portion of the posterior articular surface of the humeral head when the arm moves in maximum external rotation, extension and abduction. This area's width is 84% of the glenoid width, therefore, any glenoid articular surface loss (as in bony Bankart lesions) greatly influences the width of the glenoid track. The glenoid track influences the risk of engagement of a Hill–Sachs lesion: if the bony loss in the humeral head remains within the glenoid track there is no possibility that the Hill–Sachs

lesion overrides the glenoid rim. On the contrary, if a Hill–Sachs lesion extends over the medial margin of the glenoid track, risk of engagement rises according to the lesion’s position [17, 18].

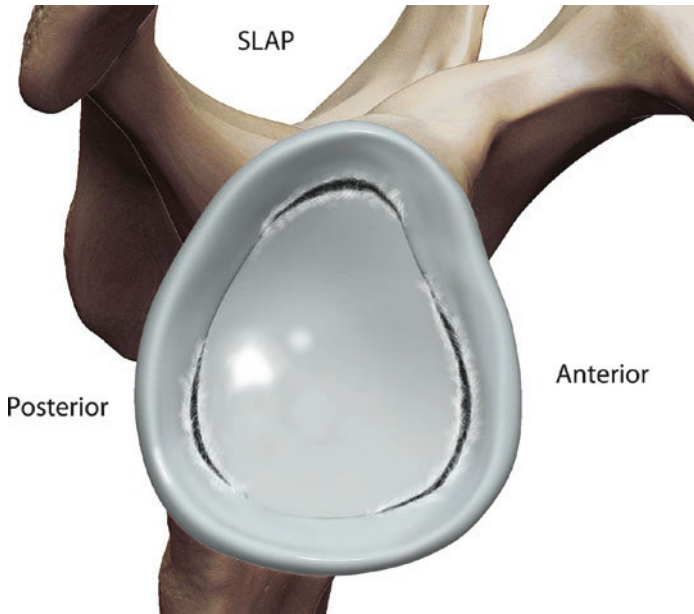
### 1.2.2 Soft Tissue Static Stabilisers

Soft tissue static stabilisers include glenoid labrum, glenohumeral capsule, glenohumeral ligaments, rotator interval, negative intra-capsular pressure and the adhesion-cohesion mechanism.

The glenoid labrum is a triangular section ring around the glenoid rim to which it’s connected by fibrocartilage and fibrous bone. The superior half of glenoid labrum is more movable than the inferior half that is tenaciously connected to the glenoid rim. Its superior border blends with the origin of the long head of the biceps. Its jobs are to make the glenoid socket deeper, to increase contacting area and congruity, to generate a suction effect, to function as an insertion area for capsular-ligamentous structures and to help muscles to compress the humeral head within the glenoid. The glenoid labrum acts on the humeral head like a plunger: loss of the glenoid labrum reduces depth of the glenoid socket more than 50%, reducing stability [19].

There are different kinds of labrum lesions and it’s very important not to confuse tears with anatomical variants that don’t require surgical repair, like sublabral foramen associated with cord-like middle glenohumeral ligament or meniscoid labrum [20] (Fig. 1.4).

The most common injury to the labrum, found in more than 90% of traumatic anterior instability [21], is a Bankart lesion. It is defined as a detachment of the anteroinferior aspect of the labrum and its attached portion of the inferior glenohumeral ligament. Despite its frequency, it cannot be considered a cause of instability in isolation, seeing that a concomitant plastic deformation needs to produce certain instability [22]. Green and Christensen [23] classified Bankart lesions in 5 arthroscopic types: type 1 refers to an entire labrum; type 2 is a simple detachment of labrum with no other significant lesions; type 3 is an intraparenchymal tear of labrum; type 4 and 5 are complex tears with a significant or



**Fig. 1.4** Labral tears occur in the antero-inferior labrum, posterior labrum and superior labrum (SLAP) (courtesy of Lennard Funk, <http://www.shoulderdoc.co.uk>)

complete degeneration of the inferior glenohumeral ligament, respectively. This classification has a prognostic value: type 4 and 5 has a good chance (87%) of recurrent instability after arthroscopic Bankart procedure.

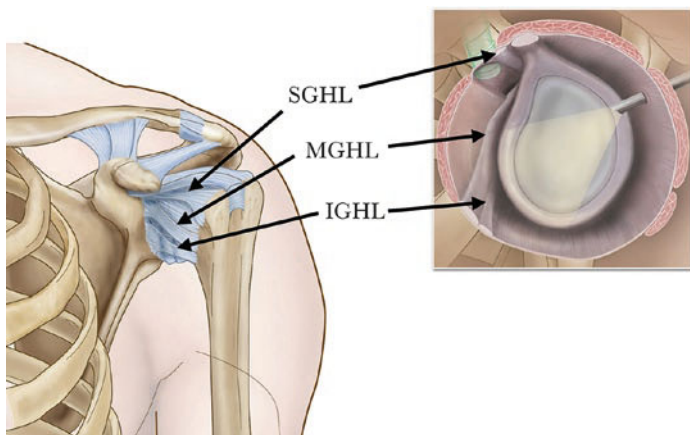
Another lesion that involves antero-inferior aspect of the labrum is the anterior labro-ligamentous periosteal sleeve avulsion (ALPSA) lesion: the anterior labro-ligamentous complex rolls up in a sleeve-like fashion and becomes displaced medially and inferiorly on the glenoid neck [24]. ALPSA lesions probably have a higher risk of redislocation than undisplaced Bankart tears, as the normal bumper and capsule that stabilise the front of the shoulder are displaced and the anterior glenoid is deficient of a capsule and labrum.

Specular lesions can be described for the posterior aspect of the labrum: a reverse Bankart lesion involves the posterior labrum and the posterior band of inferior glenohumeral ligament [25]; a POLPSA is a posterior labroligamentous sleeve avulsion, that if chronic could become a Bennett lesion (an extraarticular calcification along the posteroinferior glenoid neck close to the posterior band of the glenohumeral ligament) [26]. Reverse Bankart lesions are quite frequent in athletes, in particular contact athletes such as rugby players, being reported with a 20% incidence in a study of 142 elite rugby player shoulder arthroscopies [27]. The mechanism of injury could trace back to a direct blow to the anterior and lateral aspect of the shoulder, while the arm is adducted; a rare mechanism of injury is a posterior blow to the arm, while holding a tackle shield [28].

As far as the superior labrum is concerned, a very common lesion in throwing overhead athletes is the SLAP (superior labrum anterior and posterior) tear. Described for the first time by Snyder et al. [29], SLAP lesions occur during the ending deceleration phase of throwing, because of a traction force wielded by the long head of biceps on the glenoid labrum. Snyder has classified SLAP tear in 4 different types: type II and IV are the most significant in determining instability because they involve both labrum and long head of the biceps, so resulting in an increased total range of motion, particularly in antero posterior and superior inferior translation. Moreover, SLAP lesions are common in contact athletes: Funk and Snow [30] reported a 35% incidence of SLAP tears, arthroscopically diagnosed, in 51 rugby players' shoulders.

Capsuloligamentous structures include the joint capsule, whose mean thickness is 5 mm, and glenohumeral ligaments (superior, middle and inferior), described as located at the thickening of the capsule (Fig. 1.5). These structures have received great attention and many cadaveric and clinic studies have tried to clarify their anatomical and biomechanical characteristics and their relationship with dynamic stabilisers.

The constitutional trait of laxity facilitates extensive motion in multiple planes and may be essential to athletic performance. On the other hand, capsular stretching is noted along with a Bankart



**Fig. 1.5** The anterior glenohumeral joint ligaments: Superior (SGHL), Middle (MGHL) and anterior band of the Inferior (IGHL) (courtesy of Lennard Funk, <http://www.shoulderdoc.co.uk>)

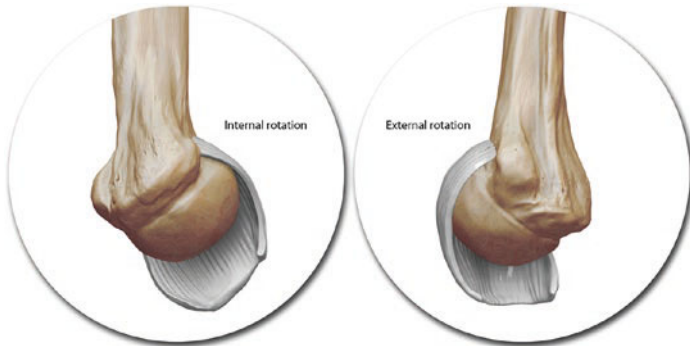
lesion and it's present in up to 28% of patients with recurrent anterior instability [31].

Glenohumeral ligaments act at maximum degrees of range of motion, when they appear in tension; at middle degrees of motion, when they are slack, stability depends on rotator cuff and long head biceps activities, those compress the humeral head inside the glenoid concavity.

Superior and middle glenohumeral ligaments, together with the coracohumeral ligament, long head of the biceps and a thin layer of capsule, help to form rotator interval and they will be discussed in detail later.

The inferior glenohumeral ligament, better-called the inferior glenohumeral ligament complex (IGHLC), is formed by 3 parts: two thicker bands on the anterior and posterior and an axillary thinner recess, assuming a sling-like structure. During abduction, external rotation and extension the IGHLC moves anteriorly, forming a restraint to anterior translation of the humeral head (Fig. 1.6).





**Fig. 1.6** The effect of internal and external rotation on the IGHL (courtesy of Lennard Funk, <http://www.shoulderdoc.co.uk>)

On the other hand, during adduction, flexion and internal rotation, the IGHL moves posteriorly, forming a restraint to posterior translation. The IGHL suffers an initial plastic deformation during initial dislocation, but the damage becomes more critical after several episodes [32]. It could be damaged more frequently at the glenoid insertion (anteroinferior glenoid rim), but also in the middle part or at the humeral insertion [33]. The incidence of humeral avulsion of the glenohumeral ligament (HAGL) has been reported as high as 10%, but they are often unrecognised [34].

Usually capsular stretching is noted along with a Bankart lesion and it's present in up to 28% of patients with recurrent anterior instability [31]. The posterior capsular also can be damaged, seeing that recurrent posterior subluxations or luxations produce capsular redundancy and increase joint volume, resulting in posterior instability. Capsular redundancy, both anterior and inferior and posterior, is a very common find in atraumatic multidirectional instability.

The rotator interval is a triangular space, with medial base and lateral apex, limits of which are the coracoid medially, the long head of biceps and its groove laterally, the superior fibres of subscapularis inferiorly and the anterior fibres of supraspinatus supe-

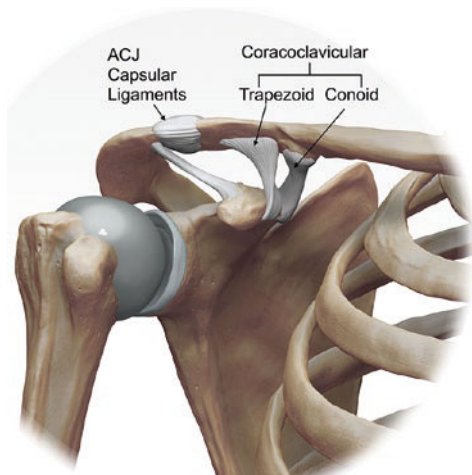
riorly. The rotator interval is composed of the coracohumeral ligament (CHL) and superior and middle glenohumeral ligaments deeper, even if the middle glenohumeral ligament contribution is relatively variable (different studies has reported its absence, from 10 to 40% of cases). Usually, it is larger in males than in females and becomes smaller with internal rotation. It is an important inferior stabiliser and its insufficiency could be clinically appreciated with sulcus sign examination. A rotator interval defect could be a little foramen or could reach larger size, influencing significantly inferior stability [35].

Negative intracapsular pressure plays a role in shoulder stability. Intracapsular pressure is about  $-42$  mmHg  $H_2O$  and it acts especially when rotator cuff muscles are not contracted and glenohumeral ligaments and capsular structure are not in tension. Loss of intracapsular negative pressure manifests itself as augmented anterior translation; this factor could be marginal when muscles are contracted and capsuloligamentous structures are in tension, especially in athletes [36].

Furthermore, synovial fluid generates the adhesion-cohesion mechanism: when two articular cartilage wet surfaces, such as the humeral head and glenoid, come into contact with each other this creates an adhesion-cohesion bond that provides stability to the glenohumeral articulation [37]. The suction effect of the glenoid labrum, the negative intracapsular pressure and the adhesion-cohesion mechanism are the three mechanisms providing the vacuum effect.

The acromioclavicular system (ACS) is formed by a complex of ligaments (conoid, trapezoid and acromioclavicular capsular ligaments) that stabilize the acromioclavicular joint (Fig. 1.7). The conoid and the trapezoid are attached from the distal clavicle to the coracoid. The ACS helps to prevent excessive superior translation of the shoulder. An acromion-clavicular dislocation up to Rockwood type 3 could require surgical repair as these cause pain and functional restrictions [38], with good results even in athletes [39].

**Fig. 1.7**  
Acromioclavicular  
system of  
ligaments (courtesy  
of Lennard Funk,  
[http://www.  
shoulderdoc.co.uk](http://www.shoulderdoc.co.uk))



## 1.3 Dynamic Stabilisers

The dynamic stabilisers are the muscles and proprioception. Rotator cuff muscles act to compress the humeral head on the glenoid surface and tightening the capsuloligamentous structures, those have direct insertion on rotator cuff tendons. The scapular rotators allow the glenoid to modify its orientation in such a way as to follow the humeral head during motion. The long head of biceps and scapulo-thoracic rhythm boost this mechanism.

### 1.3.1 Proprioception

It has been shown that the glenohumeral joint capsule has numerous mechanoreceptors particularly within the anterior and inferior capsule. In abduction and external rotation these mechanoreceptors are most likely activated as the humeral head comes into contact with the capsule sending a signal to the stabilising muscles of

the shoulder providing containment and stability of the humeral head [40]. Moreover, there is a close relationship between the glenohumeral ligament and rotator cuff muscles, as muscle contraction acts as pretensioners or cotensioners for the capsular ligament. In fact, instability could also derive from an incoordinate contraction of the rotator cuff; especially in overhead athletes, as the cuff acts as an important decelerator of anterior translation [41].

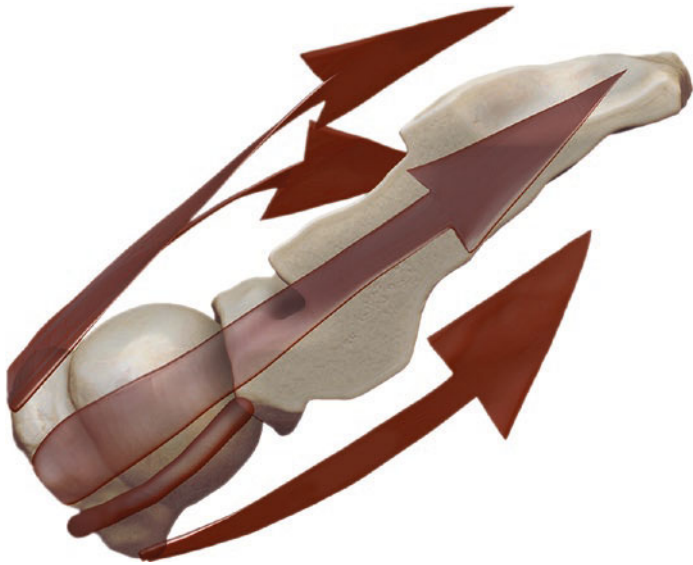
### 1.3.2 Rotator Cuff Muscles

The Rotator cuff is composed of four muscles (subscapularis, supraspinatus, infraspinatus and teres minor) that origin from the scapula and insert on the humeral head.

Subscapularis origin is on the anterior face of the scapula and insertion medially on the lesser tuberosity; supraspinatus origins in the fossa up to the scapular spine and inserts on the anterior facet of the greater tuberosity; infraspinatus origins in the fossa under the spine and inserts on the middle facet of the greater tuberosity; teres minor origins from the lateral border of the scapula and inserts on the posterior facet of the greater tuberosity.

The rotator cuff muscles provide significant stability to the shoulder joint, almost hugging the joint to the glenoid (Fig. 1.8). Wuelker et al. [42] showed that a 50% decrease in the rotator cuff muscle forces resulted in nearly a 50% increase in anterior displacement of the humeral head in response to external loading at all glenohumeral joint positions. The subscapularis muscle provides anterior stability when the arm is in neutral, but less so as the arm comes into abduction [43]. The infraspinatus and teres minor act together to reduce the strain on the antero-inferior glenohumeral ligament in abduction and external rotation [44].

Lesions to the rotator cuff can occur after a single traumatic event or after degeneration because of overuse, causing rising of the humeral head during abduction and, if a massive lesion occurs, an excessive anterior translation. Degeneration of the rotator cuff could occur because of external or internal impingement: external impingement derived from an abnormal contact between coracoacromial arc and superior surface of the rotator cuff; internal



**Fig. 1.8** Active compression effect of the rotator cuff to stabilise the humeral head on the glenoid (courtesy of Lennard Funk, <http://www.shoulderdoc.co.uk>)

impingement, defined as abnormal contact between rotator cuff articular surface and the posterosuperior glenoid rim, is common in throwing athletes. It could result in tearing of both rotator cuff and labrum. Origin of internal impingement is much discussed and it has been attributed to anterior microinstability and tightness of the posterior capsule; others underline that posteroinferior capsular contracture results in posterosuperior instability and a peel-back to the superior labrum and a tearing of the rotator cuff [45].

### 1.3.3 Long Head of Biceps

Long head of biceps (LHB) is a secondary stabiliser, its role is predominant if a rotator cuff or capsuloligamentous deficiency coexist. This tendon, originating from the supraglenoid tubercle

and passing through the bicipital groove, acts as anterior stabiliser during internal rotation and posterior stabiliser during external rotation; during the late loading phase of throwing the LHB reduces anterior translation, helping to prevent excessive torsion of the glenohumeral joint in rotation with a flexing elbow. These concepts could explain why Slap lesion type II or IV are very common in throwing athletes and why hypertrophic tendons are found in patients with insufficient rotator cuff [46].

### 1.3.4 Scapulothoracic Muscles

Trapezius, rhomboids, latissimus dorsi, serratus anterior and levator scapulae belong to the scapular rotators. The scapulothoracic joint is constituted by a sliding surface between the anterior face of scapula and thoracic cage. The coordinated movement between the scapulothoracic joint and the glenohumeral joint has been defined by Codman as “scapulothoracic rhythm” [5]. The ratio between glenohumeral and scapulothoracic joint motion is approximately 2:1, but it’s higher during lower degrees and lower at the extreme degrees of motion [47, 48]. The scapulothoracic muscles transfer the potential energy of the trunk to kinetic energy in the shoulder. The kinetic train is a concept describing the transfer of energy from the trunk to the shoulder and arm. The scapula is a key link in the kinetic chain between the trunk and the shoulder [49] (Fig. 1.9).

Any alteration in scapulothoracic rhythm could predispose to shoulder joint pathology. In particular, in pitchers the weakness of the serratus anterior predispose to development of rotator cuff tendinitis because of an abnormal contact with the coracoacromial arch or atraumatic shoulder instability [50]. Scapulothoracic rhythm recovery by appropriate scapular rotator rehabilitation is essential in younger patients with rotator cuff tendinitis or atraumatic instability.

With progressive instability there will be more proprioceptive loss from the capsule with increased capsular stretch. This proprioceptive disorganisation will lead to muscle patterning problems, repeated continued dislocations and subluxations and can progress



**Fig. 1.9** Kinetic chain (courtesy of Lennard Funk, <http://www.shoulderdoc.co.uk>)

to bony glenoid wear and affect core stability and the full kinetic chain. It would, therefore, make sense that early treatment and stabilisation would be advantageous based on the principles above.

---

## 1.4 Conclusion

The glenohumeral joint is a complex articulation with high freedom of motion but inherent instability. Many structures provide to maintain stability and a balanced and synchronised interplay between passive and dynamic restrains is essential to counteract the forces that could destabilise the glenohumeral joint. Damage to one struc-

ture is most likely to have a knock-on effect to the others and treatment should be directed accordingly. It's interesting to note how different injuries could appear with a similar clinical presentation and how narrow the borderline is between normal anatomical and pathological variants. Only a deep knowledge of anatomy and biomechanical principles will help the surgeon to recognise pathology, choose the best treatment and adapt it according to the pathoanatomy of the patient and their individual demands.

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## Q&A

- (1) Why is the shoulder the most unstable joint in the body?

The humeral head is larger than the glenoid socket, thus unconstrained. It's stability comes from the soft tissues and muscles.

- (2) What are the roles of the rotator cuff muscles?

The rotator cuff muscles provide dynamic stability to the gleno-humeral joint and assist in movement by centralising the humeral head on the glenoid.

- (3) Why is the shoulder inherently lax, with extensive motion in multiple planes?

The excessive mobility of the shoulder is to allow for overhead motions, particularly throwing. This was initially for hunting and survival, but now mainly for athletic activities.

- (4) What are the roles of the scapulothoracic muscles?

The scapulothoracic muscles transfer the potential energy of the trunk to kinetic energy in the shoulder. The scapula is a key link in the kinetic chain between the trunk and the shoulder.

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## References

1. Rockwood CAJ, Matsen FA. The shoulder. 4th ed. Philadelphia: Saunders-Elsevier; 2009.
2. Robertson DD, Yuan J, Bigliani LU, Flatow EL, Yamaguchi K. Three-dimensional analysis of the proximal part of the humerus: relevance to arthroplasty. *J Bone Joint Surg.* 2000;82-A(11):1594–602.



3. Churchill RS, Brems JJ, Kotschi H. Glenoid size, inclination, and version: an anatomic study. *J Shoulder Elb Surg.* 2001;10(4):327–32. <https://doi.org/10.1067/mse.2001.115269>.
4. Friedman RJ, An Y, Chokeski R, Kessler L. Anatomic and biomechanical study of glenohumeral contact. *J Shoulder Elb Surg.* 1994;3:S35.
5. Codman EA. *The shoulder.* Boston: Thomas Todd; 1934.
6. Saha AK. Dynamic stability of the glenohumeral joint. *Acta Orthop Scand.* 1971;42(6):491–505.
7. Piasecki DP, Verma NN, Romeo AA, Levine WN, Bach BR Jr, Provencher MT. Glenoid bone deficiency in recurrent anterior shoulder instability: diagnosis and management. *J Am Acad Orthop Surg.* 2009;17(8):482–93.
8. Bigliani LU, Newton PM, Steinmann SP, Connor PM, McLlveen SJ. Glenoid rim lesions associated with recurrent anterior dislocation of the shoulder. *Am J Sports Med.* 1998;26(1):41–5.
9. Nakagawa S, Mizuno N, Hiramatsu K, Tachibana Y, Mae T. Absorption of the bone fragment in shoulders with bony Bankart lesions caused by recurrent anterior dislocations or subluxations: when does it occur? *Am J Sports Med.* 2013;41(6):1380–6. <https://doi.org/10.1177/0363546513483087>.
10. Baudi P, Righi P, Bolognesi D, Rivetta S, Rossi Urtoler E, Guicciardi N, Carrara M. How to identify and calculate glenoid bone deficit. *Chirurgia Degli Organi Movimento.* 2005;90(2):145–52.
11. 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.* 1984;66(2):159–68.
12. 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.
13. Calandra JJ, Baker CL, Uribe J. The incidence of Hill-Sachs lesions in initial anterior shoulder dislocations. *Arthroscopy.* 1989;5(4):254–7.
14. Fronek J, Warren RF, Bowen M. Posterior subluxation of the glenohumeral joint. *J Bone Joint Surg.* 1989;71(2):205–16.
15. McLaughlin HL. Posterior dislocation of the shoulder. *J Bone Joint Surg.* 1952;24A(3):584–90.
16. Goudie EB, Murray IR, Robinson CM. Instability of the shoulder following seizures. *J Bone Joint Surg.* 2012;94(6):721–8. <https://doi.org/10.1302/0301-620X.94B6.28259>.
17. Di Giacomo G, De Vita A, Costantini A, de Gasperis N, Scarso P. Management of humeral head deficiencies and glenoid track. *Curr Rev Musculoskelet Med.* 2014;7(1):6–11. <https://doi.org/10.1007/s12178-013-9194-7>.
18. Yamamoto N, Itoi E, Abe H, Minagawa H, Seki N, Shimada Y, Okada K. Contact between the glenoid and the humeral head in abduction, external rotation, and horizontal extension: a new concept of glenoid track. *J Shoulder Elb Surg.* 2007;16(5):649–56. <https://doi.org/10.1016/j.jse.2006.12.012>.

19. Howell SM, Galinat BJ. The glenoid-labral socket. A constrained articular surface. *Clin Orthop Relat Res.* 1989;243:122–5.
20. Rao AG, Kim TK, Chronopoulos E, McFarland EG. Anatomical variants in the anterosuperior aspect of the glenoid labrum: a statistical analysis of seventy-three cases. *J Bone Joint Surg.* 2003;85-A(4):653–9.
21. Owens BD, Nelson BJ, Duffey ML, Mountcastle SB, Taylor DC, Cameron KL, Campbell S, DeBerardino TM. Pathoanatomy of first-time, traumatic, anterior glenohumeral subluxation events. *J Bone Joint Surg.* 2010;92(7):1605–11. <https://doi.org/10.2106/JBJS.I.00851>.
22. Speer KP, Deng X, Borrero S, Torzilli PA, Altchek DA, Warren RF. Biomechanical evaluation of a simulated Bankart lesion. *J Bone Joint Surg.* 1994;76(12):1819–26.
23. Green MR, Christensen KP. Arthroscopic Bankart procedure: two- to five-year followup with clinical correlation to severity of glenoid labral lesion. *Am J Sports Med.* 1995;23(3):276–81.
24. Neviaser TJ. The anterior labroligamentous periosteal sleeve avulsion lesion: a cause of anterior instability of the shoulder. *Arthroscopy.* 1993;9(1):17–21.
25. Kim SH, Ha KI, Park JH, Kim YM, Lee YS, Lee JY, Yoo JC. Arthroscopic posterior labral repair and capsular shift for traumatic unidirectional recurrent posterior subluxation of the shoulder. *J Bone Joint Surg.* 2003;85-A(8):1479–87.
26. Van Tongel A, Karelse A, Berghs B, Verdonk R, De Wilde L. Posterior shoulder instability: current concepts review. *Knee Surg Sports Traumatol Arthrosc.* 2011;19(9):1547–53. <https://doi.org/10.1007/s00167-010-1293-z>.
27. Badge R, Tambe A, Funk L. Arthroscopic isolated posterior labral repair in rugby players. *Int J Shoulder Surg.* 2009;3(1):4–7. <https://doi.org/10.4103/0973-6042.50875>.
28. McDonough A, Funk L. Critical reflection of the advanced rehabilitation of an elite rugby league player sustaining a posterior Bankart lesion. *Phys Ther Sport.* 2013;14(1):60–7. <https://doi.org/10.1016/j.pts.2012.01.002>.
29. Snyder SJ, Karzel RP, Del Pizzo W, Ferkel RD, Friedman MJ. SLAP lesions of the shoulder. *Arthroscopy.* 1990;6(4):274–9.
30. Funk L, Snow M. SLAP tears of the glenoid labrum in contact athletes. *Clin J Sport Med.* 2007;17(1):1–4. <https://doi.org/10.1097/JSM.0b013e31802ede87>.
31. Rowe CR, Patel D, Southmayd WW. The Bankart procedure: a long-term end-result study. *J Bone Joint Surg.* 1978;60(1):1–16.
32. Robinson CM, Dobson RJ. Anterior instability of the shoulder after trauma. *J Bone Joint Surg.* 2004;86(4):469–79.
33. Bigliani LU, Pollock RG, Soslowsky LJ, Flatow EL, Pawluk RJ, Mow VC. Tensile properties of the inferior glenohumeral ligament. *J Orthop Res.* 1992;10(2):187–97. <https://doi.org/10.1002/jor.1100100205>.
34. Wolf EM, Cheng JC, Dickson K. Humeral avulsion of glenohumeral ligaments as a cause of anterior shoulder instability. *Arthroscopy.* 1995;11(5):600–7.

35. Jost B, Koch PP, Gerber C. Anatomy and functional aspects of the rotator interval. *J Shoulder Elb Surg.* 2000;9(4):336–41. <https://doi.org/10.1067/mse.2000.106746>.
36. Kumar VP, Balasubramaniam P. The role of atmospheric pressure in stabilising the shoulder. An experimental study. *J Bone Joint Surg.* 1985;67(5):719–21.
37. Terry GC, Chopp TM. Functional anatomy of the shoulder. *J Athl Train.* 2000;35(3):248–55.
38. Collins DN. Disorders of the acromioclavicular joint. In: Rockwood CAJ, Matsen FA, editors. *The shoulder*, vol. 1. Philadelphia: Saunders-Elsevier; 2009. p. 453–526.
39. Marcheggiani Muccioli GM, Manning C, Wright P, Grassi A, Zaffagnini S, Funk L. Acromioclavicular joint reconstruction with the LARS ligament in professional versus non-professional athletes. *Knee Surg Sports Traumatol Arthrosc.* 2014;24(6):1961–7. <https://doi.org/10.1007/s00167-014-3231-y>.
40. Jerosch J, Thorwesten L, Teigelkotter T. Proprioception of the shoulder joint in young tennis players. *Sportverletz Sportschaden.* 1997;11(1):1–9. <https://doi.org/10.1055/s-2007-993356>.
41. Lugo R, Kung P, Ma CB. Shoulder biomechanics. *Eur J Radiol.* 2008;68(1):16–24. <https://doi.org/10.1016/j.ejrad.2008.02.051>.
42. Wuelker N, Korell M, Thren K. Dynamic glenohumeral joint stability. *J Shoulder Elb Surg.* 1998;7(1):43–52.
43. Turkel SJ, Panio MW, Marshall JL, Girgis FG. Stabilizing mechanisms preventing anterior dislocation of the glenohumeral joint. *J Bone Joint Surg.* 1981;63(8):1208–17.
44. Cain PR, Mutschler TA, Fu FH, Lee SK. Anterior stability of the glenohumeral joint. A dynamic model. *Am J Sports Med.* 1987;15(2):144–8.
45. Kirshhoff C, Imhoff AB. Posterosuperior and anterosuperior impingement of the shoulder in overhead athletes-evolving concepts. *Int Orthop.* 2010;34(7):1049–58. <https://doi.org/10.1007/s00264-010-1038-0>.
46. Pagnani MJ, Deng XH, Warren RF, Torzilli PA, O'Brien SJ. Role of the long head of the biceps brachii in glenohumeral stability: a biomechanical study in cadavera. *J Shoulder Elb Surg.* 1996;5(4):255–62.
47. Halder AM, Itoi E, An KN. Anatomy and biomechanics of the shoulder. *Orthop Clin North Am.* 2000;31(2):159–76.
48. Poppen NK, Walker PS. Forces at the glenohumeral joint in abduction. *Clin Orthop Relat Res.* 1978;135:165–70.
49. Kibler WB. The role of the scapula in athletic shoulder function. *Am J Sports Med.* 1998;26(2):325–37.
50. Glousman R, Jobe F, Tibone J, Moynes D, Antonelli D, Perry J. Dynamic electromyographic analysis of the throwing shoulder with glenohumeral instability. *J Bone Joint Surg.* 1988;70(2):220–6.



# Shoulder Injuries in Overhead Athletes

Teruhisa Mihata

## Key Learning Points

- Shoulder symptoms in overhead athletes are related mainly to failure of the kinetic chain—specifically at the hip joint, trunk, and scapula—as well as to anatomical failure at the shoulder or elbow joint. Once the kinetic chain fails, shoulder biomechanics may change, resulting in overstress of specific soft tissues, including tendons, ligaments, and muscles, or at the joint surface (cartilage or subchondral bone), thus reducing throwing performance.
- In the early stage of the pathologic kinetic chain, shoulder pain is generated without anatomical failure in the shoulder. Most early-stage symptoms can be effectively treated non-operatively.

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- When the pathologic kinematic chain (including scapular dyskinesis, muscle imbalance, posterior tightness, and increased anterior laxity) is ameliorated with physical therapy, shoulder pain during throwing decreases or disappears in most cases. An understanding of the interactions in the upper-extremity kinetic chain, together with determination of the precise pathologic condition in each athlete, is necessary for physical therapy to succeed.
- If the pathologic kinetic chain is not appropriately treated in the early stage, soft tissue or cartilage in the shoulder joint may fail, leading to the advanced stage.
- If physical therapy fails, surgical treatment needs to be considered. The best surgical option needs to be determined on the basis of the patient's background (gender, age, sport, and occupation) and the results of physical examination, including of shoulder laxity and stiffness.

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## 2.1 Aetiology

Overhead throwing motion is developed through a kinematic chain of sequential body positions and motions [1, 2]. A fully functional kinetic chain provides distal arm mobility on a stable proximal base at the scapula, core, and lower leg, together with transfer of the maximum force developed in the large muscles of the core and lower leg to the hand [3]. In the late cocking and acceleration phases of the throwing motion, a tremendous force is created on the glenohumeral joint [4]. Therefore, repetitive throwing motion may cause micro-damage of the tendons and ligaments in the glenohumeral joint, even with a fully functional kinematic chain.

Shoulder symptoms in overhead athletes are related mainly to failure of the kinetic chain—specifically at the hip joint, trunk, and scapula—as well as to anatomical failure. Once the kinetic chain fails, shoulder biomechanics may change, resulting in overstress of specific soft tissues, including tendons, ligaments, and muscles, or at joint surfaces (cartilage or subchondral bone), thus reducing throwing performance. In the early stage of the pathologic kinetic chain, shoulder pain is generated without anatomical failure at the

shoulder joint. If the pathologic kinetic chain is not appropriately treated in the early stage, soft tissue or cartilage in the shoulder may fail, leading to the advanced stage. Stiffness in the hip joint is associated with shoulder injury and poor throwing mechanics [5]. Scapular dyskinesis is associated with rotator cuff disease [6], sub-acromial impingement [7, 8], and internal impingement [9, 10]. In a mathematical study, a 20% reduction in trunk kinetic energy development resulted in a requirement for 33% more velocity in the distal segments to maintain the same energy at ball impact [1].

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## **2.2 Diagnosis**

### **2.2.1 Early Stage of the Pathologic Kinetic Chain**

Shoulder symptoms can be seen in the absence of anatomical failure, especially in the early stage of the pathologic kinetic chain. In these cases, the clinical diagnosis is likely to be inflammation in the shoulder, disabled throwing shoulder, or pathologic kinetic chain syndrome. Most symptoms in the early stage can be effectively treated non-operatively.

### **2.2.2 Advanced Stage of the Pathologic Kinetic Chain**

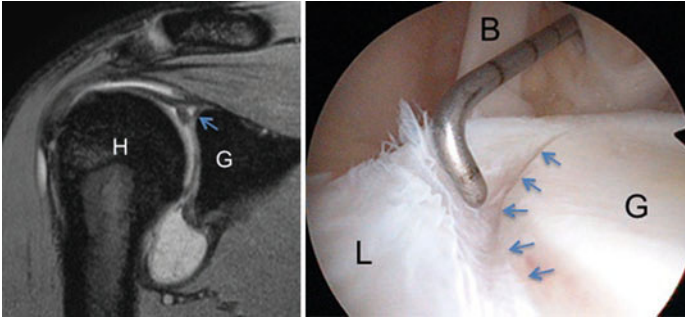
In the advanced stage, anatomical failure should be evaluated by using imaging studies such as X-ray, CT, MRI, and ultrasonography and added to the patient's diagnosis.

#### **2.2.2.1 Shoulder Injury**

##### **SLAP Lesion and Biceps Tendinitis**

A type II superior labrum anterior-posterior (SLAP) lesion is defined as an avulsion of the superior labrum and the biceps anchor from the glenoid (Fig. 2.1) [11].

Type II SLAP lesions cause shoulder instability [11–13] and pain [11, 14, 15]. According to previous biomechanical studies, an

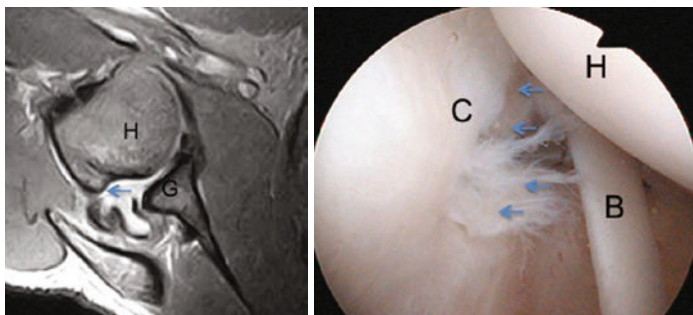


**Fig. 2.1** Type II SLAP lesion. (Left) MR arthrography; (Right) arthroscopic view. *B* biceps tendon, *G* glenoid, *H* humeral head, *L* superior labrum

isolated type II SLAP lesion results in a subtle increase in glenohumeral translation [13, 16]. However, excessive external rotation at the glenohumeral joint—one of the pathomechanical mechanisms behind type II SLAP lesions—elongates the shoulder anterior capsule [16, 17]. When the combination of a type II SLAP lesion and excessive anterior capsular laxity occurs, shoulder symptoms may become more severe because of an apparent increase in glenohumeral translation [16]. SLAP lesions can be detected with MRI or MR arthrography. Most symptomatic type II SLAP lesions are associated with a positive O'Brien test or pain at the end range of shoulder motion (e.g., in maximal shoulder abduction or maximal shoulder external rotation in the abducted position).

### Rotator Cuff Tear

Rotator cuff injury is common among overhead athletes, including baseball players [18] and tennis players [19]. Repetitive throwing motion leads to fatigue of the rotator cuff muscles and rotator cuff tears over time, usually involving the undersurface of the posterior half of the supraspinatus and superior half of the infraspinatus. Whereas articular-sided partial-thickness rotator cuff tears (PASTA lesion) are common in overhead athletes (Fig. 2.2) [20–22], full-thickness tears are diagnosed much less frequently.



**Fig. 2.2** PASTA lesion (articular-sided partial-thickness rotator cuff tear). (Left) MR arthrography in the abduction—external rotation position; (right) arthroscopic view. *B* biceps tendon, *C* superior capsule underlying supraspinatus and infraspinatus tendons, *G* glenoid, *H* humeral head

A recent anatomical study has shown that the superior shoulder capsule is attached to a substantial area (30–61%) of the greater tuberosity [23]. This suggests that articular-sided partial-thickness tears of the supraspinatus and infraspinatus tendons include detachment of the superior shoulder capsule from the greater tuberosity. It also suggests that low-grade partial tears found to involve less than 50% of the tendon thickness are not rotator cuff tears but just superior capsular tears, although they have been traditionally diagnosed as rotator cuff tears. By using MRI or ultrasonography, rotator cuff tears can be diagnosed very accurately in terms of whether they are partial or complete, along with their size and location. In a high-grade (more than 50% of the tendon thickness) partial thickness tear or complete tear of the supraspinatus tendon or infraspinatus tendon, patients have a positive subacromial impingement test (Neer test [24], Hawkins test [25], or Yocum test [26, 27]) and decreased muscle strength in shoulder abduction or external rotation. Cadaveric biomechanical study showed that a tear in the superior capsule at the greater tuberosity, which may be seen with partial rotator cuff tears, increased anterior and inferior translations [28]. For the treatment



of articular-sided partial-thickness tears, the shoulder laxity should be evaluated.

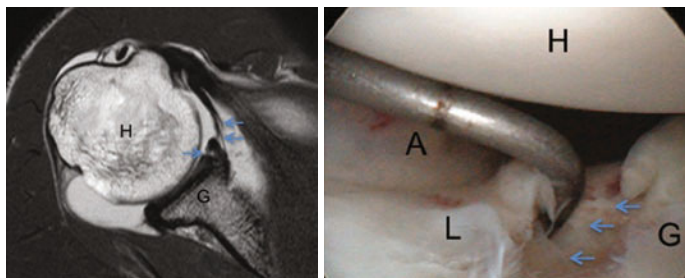
### **Anterior Capsular-Ligament Tear or Elongation**

Anterior shoulder instability due to dysfunction of the anterior capsular ligaments [14, 29, 30] may disable the throwing shoulder. Although traumatic subluxation causes anterior labral or capsular tears in some throwing athletes [31, 32], most cases of excessive anterior shoulder laxity (hypermobility of the humeral head) result from repeated stretching of the anterior capsular ligaments during the throwing motion [32–35]. Excessive anterior capsular laxity (elongation of the anterior capsular ligaments) is thought to cause shoulder subluxation during acceleration of the throwing motion, thus disabling the throwing shoulder. Anterior capsular-ligament tear or elongation can be diagnosed by using MR arthrography (Fig. 2.3).

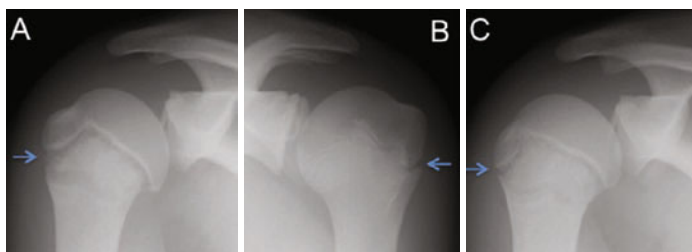
Anterior capsular-ligament tear or elongation causes shoulder pain, rather than shoulder instability, through the application of excessive external rotation torque in the abducted shoulder position. This is because most tearing or elongation in overhead athletes is not as severe as that in patients with traumatic anterior shoulder dislocation, and a subtle increase in shoulder laxity, as seen in overhead athletes, causes shoulder pain. Some athletes have anterior shoulder pain due to anterior labrum tear, and some have posterior shoulder pain due to shoulder internal impingement; the latter is exacerbated by increased anterior shoulder laxity [36].

### **Little League Shoulder (Proximal Humeral Epiphysiolysis)**

Repetitive throwing motion in adolescent throwing athletes can lead to epiphyseal plate injuries, because the epiphyseal plate is weaker than the surrounding tendons and ligaments. This injury is called proximal humeral epiphysiolysis, or Little League shoulder. Proximal humeral epiphysiolysis causes shoulder pain localized to the proximal humerus during throwing and is diagnosed from radiographic or ultrasonographic evidence of widening of the proximal humeral epiphysis (Fig. 2.4) [37, 38]. Most patients feel tenderness of the epiphyseal plate in the proximal humerus.



**Fig. 2.3** Anterior labrum tear and capsular-ligament elongation. (Left) MR arthrography; (right) arthroscopic view. *A* anterior band of the inferior glenohumeral ligament, *G* glenoid, *H* humeral head, *L* anterior labrum

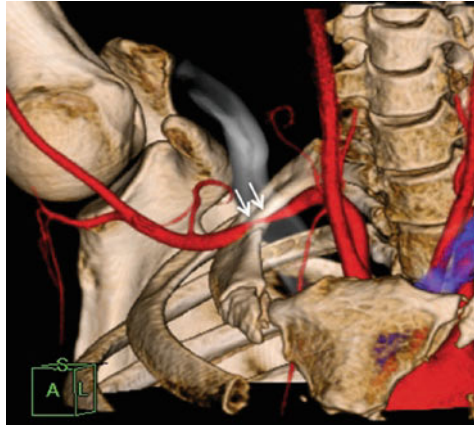


**Fig. 2.4** Radiographic findings in Little League shoulder (proximal humeral epiphysiolysis). (a) Widening of the right proximal humeral epiphysis at the first visit to our clinic; (b) intact epiphyseal plate of the left proximal humerus; (c) healed epiphyseal plate of the right proximal humerus after cessation of baseball for 3 months

### Neurovascular Disease: Suprascapular Neuropathy, Quadrilateral Space Syndrome, Thoracic Outlet Syndrome, and Effort Thrombosis

In overhead athletes, less common causes of shoulder pain include quadrilateral space syndrome [39, 40] and suprascapular nerve entrapment [41–43], in addition to vascular problems such as effort thrombosis of the axillary artery or vein [44–47] and thoracic outlet syndrome [42, 43, 48, 49]. These neurovascular causes are difficult to diagnose and often

**Fig. 2.5** Three-dimensional CT angiography of thoracic outlet syndrome. White arrows show compression of the subclavian artery between the clavicle and first rib (Image courtesy of Dr. Kozo Furushima, Keiyu Hospital, Japan)



require specialised tests such as electromyography and arteriography (Fig. 2.5).

## 2.3 Relevant Pathology

### 2.3.1 Shoulder Internal Impingement

Shoulder internal impingement, namely impingement of the undersurface of the rotator cuff on the posterior superior labrum and the glenoid during the late cocking phase of throwing motion, is thought to be a cause of posterior rotator cuff injury and type II SLAP lesions [50, 51]. Whereas shoulder internal impingement is physiological, occurring in both throwing and non-throwing shoulders when the arm is in the abducted externally rotated position [52], forceful internal impingement can be pathological. Therefore, increased glenohumeral contact pressure is critical to the occurrence of pathological internal impingement. Previous biomechanical and electromyographic studies have shown that (1) excessive glenohumeral horizontal abduction [53], (2) increased anterior capsular laxity [36], (3) posterior capsular contracture [54], (4) rotator cuff muscle imbalance through decreased strength of the subscapularis mus-

cle [55, 56], (5) decreased internal rotator muscle strength [55], and (6) increased scapular internal rotation result in forceful internal impingement [10].

### **2.3.2 Shoulder Subacromial Impingement**

Subacromial impingement is mostly diagnosed in older overhead athletes. Several studies have reported a relationship between decreased upward scapular rotation and shoulder disorders caused by subacromial impingement [8, 57]. A previous biomechanical study showed that posteroinferior capsule tightness increased the contact pressure and area on the coracoacromial arch [58]. Radiographs in older overhead athletes sometimes show a prominent anterior acromion or acromial spur.

### **2.3.3 Peel-Back Mechanism**

Peel-back is the pathomechanism of type II SLAP lesions [14]. Cadaveric studies have shown that excessive humeral external rotation causes increased strain on [59], and detachment of [60, 61], the superior labrum, suggesting that increased humeral external rotation results in peel-back of the superior labrum. Although increased external rotation is often necessary to throw at a highly competitive level [17], it can cause type II SLAP lesions.

### **2.3.4 SICK Scapula**

Burkhart et al. [15] termed scapular dyskinesis in throwing athletes “SICK” scapula (scapular malposition, inferior medial border prominence, coracoid pain and malposition, and dyskinesis of scapular movement) and designated three types: type I (inferior medial scapular border prominence), type II (medial scapular border prominence), and type III (superior medial scapular border prominence). Medial scapular border prominence, which represents increased internal rotation of the scapula, can be a

cause of dead arm syndrome [15, 62, 63]. One cadaveric biomechanical study showed that increased internal scapular rotation (a possible cause of medial scapular border prominence) increased the pressure caused between the greater tuberosity and the glenoid by internal impingement during the late cocking phase of throwing motion, thereby increasing the risk of tearing the impinged rotator cuff tendons and superior labrum [10]. Decreased upward scapular rotation increased the internal impingement area. Altered scapular orientation is thought to result in alteration of the centre of rotation [63], diminished function of the kinematic chain between the upper and lower extremities [63, 64], and decreased shoulder muscle function [62, 63], thereby increasing the risk of shoulder injury [62, 63, 65].

### **2.3.5 Pathologic Shoulder Laxity**

Increased shoulder laxity due to dysfunction of the anterior capsular ligaments can disable the throwing shoulder [32–35, 66]. Jobe et al. [67, 68] postulated that, in overhead athletes, capsular laxity due to repetitive microtrauma may result in increased shoulder laxity with secondary pathologies such as labrum damage or partial rotator cuff tear (pathologic shoulder laxity). One cadaveric biomechanical study showed that excessive anterior capsular laxity, which was created by repeatedly applying excessive external rotational torque as seen in throwing athletes [34], significantly increased horizontal abduction and contact pressure in the glenohumeral joint [36]. These results suggest that excessive anterior capsular laxity can cause forceful internal impingement during the late cocking phase of throwing.

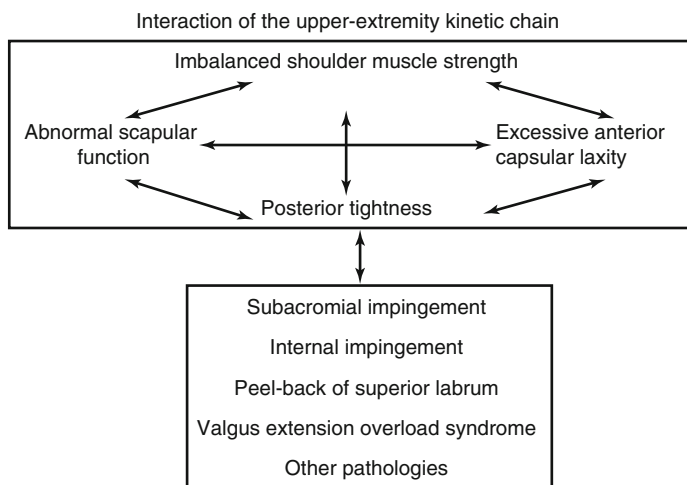
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## **2.4 Management Principles**

Physical therapy is the most important treatment for preventing surgery in overhead athletes—especially in the early stage of the pathologic kinetic chain—and for improving shoulder function

after surgery. When a pathologic kinematic chain, including scapular dyskinesis, muscle imbalance, posterior tightness, and increased anterior laxity, is ameliorated with physical therapy, shoulder pain during throwing decreases or disappears in most cases. An understanding of the interactions in the upper-extremity kinetic chain and determination of the precise pathological condition in each athlete are necessary for physical therapy to succeed (Fig. 2.6).

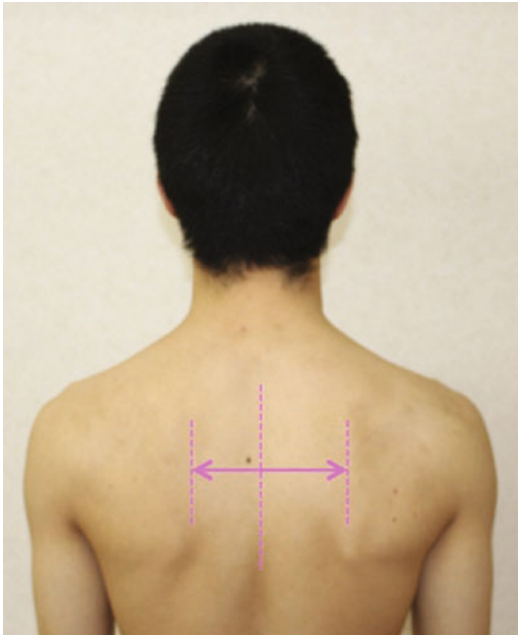
The main four pathological conditions—abnormal scapular function [10, 63], posterior tightness [14, 54], capsular laxity [33, 34, 36, 68], and imbalanced shoulder muscle strength [55, 56]—influence each other. These four pathological conditions also cause subacromial impingement [58], internal impingement [10,



**Fig. 2.6** Interactions in the upper-extremity kinetic chain. The main four pathological conditions—abnormal scapular function, posterior tightness, capsular laxity, and imbalanced shoulder muscle strength—influence each other. These four pathological conditions also cause subacromial impingement, internal impingement, peel-back of the superior labrum, valgus extension overload syndrome, and other pathologies, which may lead to anatomical failure. The reverse is also possible, i.e. impingements, peel-back, and overload can cause the four main pathological conditions

36, 54, 56], peel-back of the superior labrum [12, 14, 56], valgus extension overload syndrome [69], and other pathologies, which may lead to anatomical failure. The reverse is also possible, i.e. impingements, peel-back, and overload can cause the four main pathological conditions. The function of the trunk and lower extremity should be evaluated very carefully and treated. If physical therapy fails, surgical treatment needs to be considered.

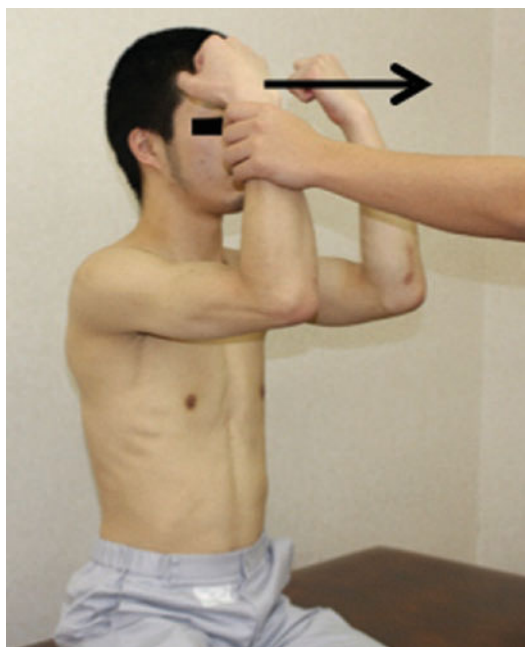
The Hara test is useful for assessing the upper-extremity kinetic chain for abnormalities leading to shoulder pain. The Hara test comprises 11 physical examinations relevant to the scapular and humeral kinetic chain: (1) scapula–spine distance (Fig. 2.7); (2) elbow extension test (Fig. 2.8); (3) elbow push test (Fig. 2.12);



**Fig. 2.7** In the scapula–spine distance test, the distance from the medial edge of the scapular spine to the spinous process of the thoracic spine is measured with the arms at the sides. The reference point on the thoracic spine is defined as the nearest spinous process. A difference of more than 1.0 cm between the left- and right-side measurements is considered abnormal

(4) manual muscle strength of abduction; (5) manual muscle strength of external rotation; (6) manual muscle strength of internal rotation; (7) combined abduction test (Fig. 2.9); (8) horizontal flexion test (Fig. 2.10); (9) capsular laxity tests; (10) subacromial impingement tests; and (11) hyper-external rotation test (Fig. 2.11). The total score (i.e., the number of “intact” results—see Fig. 2.12) for the Hara test and the abnormalities in each examination are evaluated.

The scapula–spine distance, elbow extension test, elbow push test, subacromial impingement tests, and manual muscle tests of shoulder abduction, external rotation, and internal rotation are



**Fig. 2.8** Elbow extension test for assessment of scapular stability. The elbow extension test is performed with the shoulders in  $90^\circ$  of forward flexion. The subject extends the elbow joint from  $90^\circ$  of flexion with maximum force while the examiner holds the subject's forearm to resist the extension force. The test is considered abnormal when the muscle strength on the dominant side is less than that on the non-dominant side

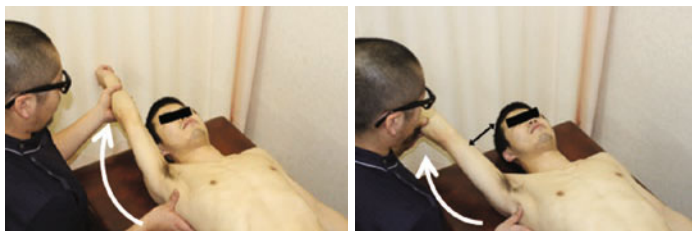




**Fig. 2.9** Elbow push test for assessment of scapular stability. The elbow push test is performed with the shoulders in  $90^\circ$  of forward flexion. While grasping the contralateral elbow with each hand, the subject pushes each elbow in turn anteriorly with maximum force. The examiner resists this pushing by holding the elbow. The test is considered to be abnormal when the muscle strength on the dominant side is less than that on the non-dominant side

assessed while the subject is sitting. Patients are supine for the combined abduction test, horizontal flexion test, capsular laxity tests, and hyper-external rotation test.

In the scapula–spine distance test, the distance from the medial edge of the scapular spine to the spinous process of the thoracic spine is measured with the arms at the sides (Fig. 2.10). The reference point on the thoracic spine is defined as the nearest spinous process. A difference of more than 1.0 cm between the left- and right-side measurements is considered abnormal. To assess the scapular stabilizers, the elbow extension test and elbow push test



**Fig. 2.10** Combined abduction test for assessment of posterior shoulder tightness. The examiner completely prevents any movement of the scapula by holding it. The humerus is passively abducted in the coronal plane. This test is considered abnormal when the upper arm fails to touch the head during glenohumeral abduction with a fixed scapula. Left: intact; right: abnormal



**Fig. 2.11** Horizontal flexion test for assessment of posterior shoulder tightness. The examiner completely prevents any movement of the scapula by holding it and horizontally flexes the humerus. This test is considered to be abnormal when, during shoulder horizontal flexion with a fixed scapula, the subject is unable to reach around the other shoulder to touch the bed. Left: intact; right: abnormal

are performed with the shoulders in  $90^\circ$  of forward flexion (Figs. 2.11 and 2.12). For the elbow extension test, the subject extends the elbow joint from  $90^\circ$  of flexion by using maximum force while the examiner holds the subject's forearm to resist the extension force (Fig. 2.11). For the elbow push test, while grabbing the contralateral elbow with each hand, the subject pushes each elbow in turn anteriorly with maximum force as the examiner resists the subject's pushing by holding the elbow (Fig. 2.12).



**Fig. 2.12** The hyper-external rotation test, which evaluates peel-back of the superior labrum and pathologic internal impingement, is performed in  $90^\circ$  of shoulder abduction with the elbow flexed at  $90^\circ$  in the supine position. The test is considered abnormal when the subject feels pain as the examiner applies external rotation torque beyond the maximum external rotation position

Muscle strength is evaluated by manual muscle testing on a scale of 0–5. We assess the muscle strength of shoulder abduction with the subject’s thumb up; this is known as the “full can position” [26, 27, 70]. We measure external rotation strength with the subject’s arm at his/her side [71]. To assess internal rotation strength, we record the subject’s strength in lifting his/her hand off his/her back [72]. We consider the results of the elbow extension test, elbow push test, and manual muscle tests of abduction, external rotation, and internal rotation to be abnormal when the muscle strength on the dominant side is less than that on the non-dominant side. To assess the posterior tightness of the shoulder joint, subjects perform the combined abduction test and horizontal flexion test while the examiner fixes the scapula and prevents it from moving by holding it. The humerus is passively abducted in the coronal plane for the combined abduction test (Fig. 2.13) and horizontally flexed for the horizontal flexion test (Fig. 2.15). If the subject’s upper arm fails to touch his/her head during glenohumeral abduction with a fixed scapula, the combined abduction test is graded as abnormal. The horizontal flexion test is considered abnormal when the subject is unable to reach around the other shoulder to touch the bed

## Hara test scoring sheet

Date of Examination \_\_\_\_\_

Name \_\_\_\_\_ Age \_\_\_\_\_ Sex \_\_\_\_\_

Dominant Hand (R) \_\_\_\_\_ (L) \_\_\_\_\_

Sport \_\_\_\_\_ Position \_\_\_\_\_ Years Played \_\_\_\_\_

## Instructions to examiners:

Please perform and score the following 11 physical examinations and then calculate the total score (i.e., the number of "intact" results).

Scapular function

Scapula–spine distance	Abnormal	Intact
Elbow extension test	Abnormal	Intact
Elbow push test	Abnormal	Intact

Manual muscle strength

Abduction	Abnormal	Intact
External rotation	Abnormal	Intact
Internal rotation	Abnormal	Intact

Posterior tightness

Combined abduction test	Abnormal	Intact
Horizontal flexion test	Abnormal	Intact

Capsular laxity tests

Abnormal Intact

Subacromial impingement tests

Abnormal Intact

Hyper external rotation test

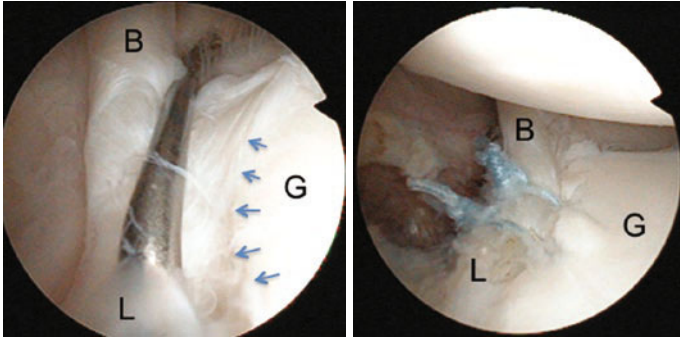
Abnormal Intact

Total Hara test score \_\_\_\_\_

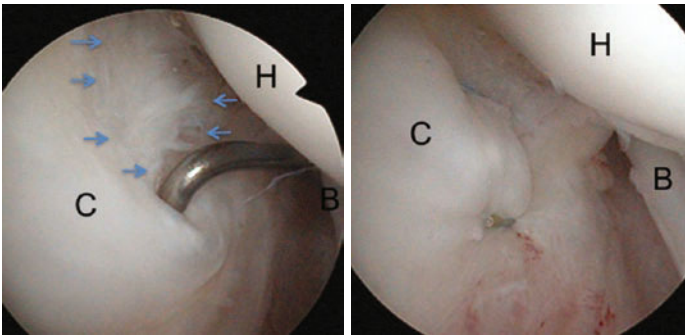
(number of "intact" results)

**Fig. 2.13** Hara test scoring sheet

during horizontal flexion with a fixed scapula. Capsular laxity is evaluated by load-and-shift testing in the anterior, posterior, and inferior directions; anterior apprehension and relocation tests are also done. When the dominant side shows increased laxity, or



**Fig. 2.14** Posterior view of shoulder arthroscopy. (Left) Type II SLAP lesion (blue arrows) before surgery. (Right) After repair of the detached superior labrum (SLAP repair). *B* biceps tendon, *G* glenoid, *L* superior labrum



**Fig. 2.15** Posterior view of shoulder arthroscopy. (Left) PASTA lesion (articular-sided partial-thickness rotator cuff tear). (Right) After trans-tendon repair of the PASTA lesion. *B* biceps tendon, *C* superior capsule underlying supraspinatus and infraspinatus tendons, *H* humeral head

when the subject feels that the shoulder is unstable during any test, capsular laxity is considered abnormal. To evaluate subacromial impingement, we perform the Neer [24], Hawkins [25], and Yocum [26, 27] tests. If the subject feels shoulder pain during any of these tests, subacromial impingement testing is graded as abnormal. The hyper-external rotation test (Fig. 2.15), which evaluates peel-back

of the superior labrum [12, 14, 16] and pathologic internal impingement [50, 53, 65], is performed in 90° of shoulder abduction with the elbow flexed at 90° in the supine position. The test is considered to be abnormal when a subject feels pain as the examiner applies external rotation torque beyond the maximum external rotation position. The number of “intact” results among the 11 physical examinations is recorded as the total Hara test score for each subject. The maximum total score (11 points) represents all “intact” results (i.e., no abnormality found) for all tests; subjects with lower scores are considered likely to have a problem in the upper-extremity kinetic chain.

Throwing athletes have increased external rotation and decreased internal rotation in the dominant shoulder when measurements are performed at 90° of abduction [15, 73, 74]. Changes in glenohumeral rotation in throwing athletes result from anterior capsular laxity [33, 34, 50], posterior capsular tightness [15, 54, 75, 76], muscular imbalance [56], scapula malposition [10, 77], or increased humeral retroversion [74, 78, 79]. Whereas increased humeral retroversion is thought to be an adaptation to throwing motion, excessive anterior capsular laxity, excessive posterior capsular contracture, and muscle imbalance may cause shoulder pathologies such as rotator cuff tear and superior labral tear from anterior to posterior (SLAP) lesions. To assess the capsular or muscular imbalance with the exclusion of side-to-side differences in humeral retroversion, an ultrasound-assisted measurement of glenohumeral range of motion by standardizing neutral rotation on the basis of the location of the bicipital groove is useful [61].

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## 2.5 Outcomes

### 2.5.1 Nonoperative Treatment

Only a few studies regarding the outcomes of physical therapy have been reported. Edwards et al. described the results of nonoperative treatment (physical therapy and nonsteroidal anti-inflammatory drugs) of SLAP lesions [80]. Of 15 overhead athletes, 10 (67%) were able to return to overhead athletics after nonoperative treatment.

We investigated the clinical outcomes of shoulder injuries after physical therapy without anti-inflammatory drugs in 25 competitive baseball players (11 with shoulder inflammation, 10 with an isolated SLAP lesion, 3 with a SLAP lesion and partial thickness rotator cuff tear, and 1 with a middle glenohumeral ligament tear). All pathologies were diagnosed by physical examination and MRI. Overall, 92% (23/25 athletes) could return to their previous levels of sport with proper physical therapy. Average time to return to the game was 53 days.

Most shoulder injuries in juvenile and adolescent baseball players, such as Little League shoulder (proximal humeral epiphysiolysis) can heal with nonoperative treatment, especially in the early stage of injury. Nonoperative treatment consists of active rest and physical therapy for the kinetic chain, with avoidance of throwing. Prevention is the key to the treatment of shoulder injuries in the young athlete.

### **2.5.2 Operative Treatment**

The clinical outcomes of arthroscopic SLAP repair vary: reported success rates range from 22% to 94% [81–87]. Conway [21] reported excellent clinical results of arthroscopic repair of partial thickness rotator cuff tears in nine baseball players. Seven were professional players and two were college players. Eight players (89%) returned to play at the same level or higher. However, most reports of rotator cuff repair in professional baseball players have demonstrated a poor prognosis, with substantial difficulty in returning to preinjury levels of play [18, 88, 89]. Debridement of partial thickness rotator cuff tears produces return-to-throw rates of 16–85% [20, 21, 90, 91].

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## **2.6 Surgery**

If physical therapy fails, surgical treatment typically is indicated. Most shoulder injuries in overhead athletes are treated arthroscopically. For SLAP lesions, debridement of the detached labrum, repair of the detached superior labrum (Fig. 2.14), or biceps tenodesis can be chosen.

Surgical treatment of articular-sided partial-thickness rotator cuff tears includes debridement of partial-thickness tears with or without acromioplasty [91–96], trans-tendon repair (Fig. 2.15) [90, 97–102], or conversion to a full-thickness tear followed by repair [98, 100, 103–105].

The best surgical option for each patient will differ and needs to be determined on the basis of the patient's background (including their gender, age, sport, and occupation) and the results of a physical examination, including of shoulder laxity and stiffness. Recently, Ito and Furushima recommended first rib resection for overhead athletes with thoracic outlet syndrome (personal communication).

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## Q&A

- (1) What is the commonest cause of shoulder pain in the repetitive throwing athlete?

Shoulder symptoms in overhead athletes are related mainly to failure of the kinetic chain. Once the kinetic chain fails, shoulder biomechanics may change, resulting in overstress of specific soft tissues, including tendons, ligaments, and muscles, or at the joint surface.

- (2) Do most shoulder problems in the overhead athlete need surgery?

No. Most shoulder pain is generated without anatomical failure in the shoulder. Most early-stage symptoms can be effectively treated non-operatively.

- (3) When would you consider surgery?

If the pathologic kinetic chain is not appropriately treated in the early stage, soft tissue or cartilage in the shoulder or elbow joint may fail, leading to the advanced stage. If physical therapy fails, surgical treatment needs to be considered.

- (4) What are the results of surgery?

The results are variable, with reported success rates of 22–94%. This huge variation is due to the complexity and multiple factors involved in the shoulder pain of a repetitive overhead athlete.



## References

1. Kibler WB. Biomechanical analysis of the shoulder during tennis activities. *Clin Sports Med.* 1995;14:79–85.
2. Putnam CA. Sequential motions of body segments in striking and throwing skills: descriptions and explanations. *J Biomech.* 1993;26(Suppl 1):125–35.
3. Lintner D, Noonan TJ, Kibler WB. Injury patterns and biomechanics of the athlete's shoulder. *Clin Sports Med.* 2008;27:527–51.
4. Fleisig GS, Andrews JR, Dillman CJ, Escamilla RF. Kinetics of baseball pitching with implications about injury mechanisms. *Am J Sports Med.* 1995;23:233–9.
5. Robb AJ, Fleisig G, Wilk K, Macrina L, Bolt B, Pajaczkowski J. Passive ranges of motion of the hips and their relationship with pitching biomechanics and ball velocity in professional baseball pitchers. *Am J Sports Med.* 2010;38:2487–93.
6. Kibler WB, Sciascia A. Current concepts: scapular dyskinesis. *Br J Sports Med.* 2010;44:300–5.
7. Graichen H, Stammberger T, Bonel H, Wiedemann E, Englmeier KH, Reiser M, Eckstein F. Three-dimensional analysis of shoulder girdle and supraspinatus motion patterns in patients with impingement syndrome. *J Orthop Res.* 2001;19:1192–8.
8. Ludewig PM, Cook TM. Alterations in shoulder kinematics and associated muscle activity in people with symptoms of shoulder impingement. *Phys Ther.* 2000;80:276–91.
9. Drakos MC, Rudzki JR, Allen AA, Potter HG, Altchek DW. Internal impingement of the shoulder in the overhead athlete. *J Bone Joint Surg Am.* 2009;91:2719–28.
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;94:1576–83.
11. Snyder SJ, Karzel RP, Del Pizzo W, Ferkel RD, Friedman MJ. SLAP lesions of the shoulder. *Arthroscopy.* 1990;6:274–9.
12. Mihata T, McGarry MH, Tibone JE, Abe M, Lee TQ. Type II SLAP lesions: a new scoring system--the sulcus score. *J Shoulder Elb Surg.* 2005;14:19S–23S.
13. Panossian VR, Mihata T, Tibone JE, Fitzpatrick MJ, McGarry MH, Lee TQ. Biomechanical analysis of isolated type II SLAP lesions and repair. *J Shoulder Elb Surg.* 2005;14:529–34.
14. Burkhart SS, Morgan CD. The peel-back mechanism: its role in producing and extending posterior type II SLAP lesions and its effect on SLAP repair rehabilitation. *Arthroscopy.* 1998;14:637–40.

15. Burkhart SS, Morgan CD, Kibler WB. The disabled throwing shoulder: spectrum of pathology part I: pathoanatomy and biomechanics. *Arthroscopy*. 2003;19:404–20.
16. Mihata T, McGarry MH, Tibone JE, Fitzpatrick MJ, Kinoshita M, Lee TQ. Biomechanical assessment of type II superior labral anterior-posterior (SLAP) lesions associated with anterior shoulder capsular laxity as seen in throwers: a cadaveric study. *Am J Sports Med*. 2008;36:1604–10.
17. Andrews JR, Dugas JR. Diagnosis and treatment of shoulder injuries in the throwing athlete: the role of thermal-assisted capsular shrinkage. *Instr Course Lect*. 2001;50:17–21.
18. Mazoue CG, Andrews JR. Repair of full-thickness rotator cuff tears in professional baseball players. *Am J Sports Med*. 2006;34:182–9.
19. Sonnery-Cottet B, Edwards TB, Noel E, Walch G. Rotator cuff tears in middle-aged tennis players: results of surgical treatment. *Am J Sports Med*. 2002;30:558–64.
20. Andrews JR, Broussard TS, Carson WG. Arthroscopy of the shoulder in the management of partial tears of the rotator cuff: a preliminary report. *Arthroscopy*. 1985;1:117–22.
21. Conway JE. Arthroscopic repair of partial-thickness rotator cuff tears and SLAP lesions in professional baseball players. *Orthop Clin North Am*. 2001;32:443–56.
22. Levitz CL, Dugas J, Andrews JR. The use of arthroscopic thermal capsulorrhaphy to treat internal impingement in baseball players. *Arthroscopy*. 2001;17:573–7.
23. Nimura A, Kato A, Yamaguchi K, Mochizuki T, Okawa A, Sugaya H, Akita K. The superior capsule of the shoulder joint complements the insertion of the rotator cuff. *J Shoulder Elb Surg*. 2012;21:867–72.
24. Neer CS. Anterior acromioplasty for the chronic impingement syndrome in the shoulder: a preliminary report. *J Bone Joint Surg Am*. 1972;54:41–50.
25. Hawkins RJ, Kennedy JC. Impingement syndrome in athletes. *Am J Sports Med*. 1980;8:151–8.
26. McFarland EG. Rotator cuff disease and impingement. In: McFarland EG, editor. *Examination of the shoulder*. New York: Thieme Medical Publishers, Inc.; 2005. p. 126–61.
27. McFarland EG. Strength testing. In: McFarland EG, editor. *Examination of the shoulder*. New York: Thieme Medical Publishers, Inc.; 2005. p. 88–125.
28. Ishihara Y, Mihata T, Tamboli M, Nguyen L, Park KJ, McGarry MH, Takai S, Lee TQ. Role of the superior shoulder capsule in passive stability of the glenohumeral joint. *J Shoulder Elb Surg*. 2013;23(5):642–8.
29. Cohen DB, Coleman S, Drakos MC, Allen AA, O'Brien SJ, Altchek DW, Warren RF. Outcomes of isolated type II SLAP lesions treated with

- arthroscopic fixation using a bioabsorbable tack. *Arthroscopy*. 2006;22:136–42.
30. Hawkins RJ, Schutte JP, Janda DH, Huckell GH. Translation of the glenohumeral joint with the patient under anesthesia. *J Shoulder Elb Surg*. 1996;5:286–92.
  31. Owens BD, Dickens JF, Kilcoyne KG, Rue JP. Management of mid-season traumatic anterior shoulder instability in athletes. *J Am Acad Orthop Surg*. 2012;20:518–26.
  32. Savoie FH, O'Brien MJ. Anterior instability in the throwing shoulder. *Sports Med Arthrosc*. 2014;22:117–9.
  33. Jobe FW, Giangarra CE, Kvitne RS, Glousman RE. Anterior capsulolabral reconstruction of the shoulder in athletes in overhand sports. *Am J Sports Med*. 1991;19:428–34.
  34. Mihata T, Lee Y, McGarry MH, Abe M, Lee TQ. Excessive humeral external rotation results in increased shoulder laxity. *Am J Sports Med*. 2004;32:1278–85.
  35. Ryu RK, Dunbar WH, Kuhn JE, McFarland EG, Chronopoulos E, Kim TK. Comprehensive evaluation and treatment of the shoulder in the throwing athlete. *Arthroscopy*. 2002;18:70–89.
  36. Mihata T, McGarry MH, Neo M, Ohue M, Lee TQ. Effect of anterior capsular laxity on horizontal abduction and forceful internal impingement in a cadaveric model of throwing shoulder. *Am J Sports Med*. 2015;43(7):1758–63.
  37. Carson WG, Gasser SI. Little Leaguer's shoulder. A report of 23 cases. *Am J Sports Med*. 1998;26:575–80.
  38. Ireland ML, Andrews JR. Shoulder and elbow injuries in the young athlete. *Clin Sports Med*. 1988;7:473–94.
  39. Hoskins WT, Pollard HP, McDonald AJ. Quadrilateral space syndrome: a case study and review of the literature. *Br J Sports Med*. 2005;39:e9.
  40. Todd GJ, Benvenisty AI, Hershon S, Bigliani LU. Aneurysms of the mid axillary artery in major league baseball pitchers--a report of two cases. *J Vasc Surg*. 1998;28:702–7.
  41. Gowan ID, Jobe FW, Tibone JE, Perry J, Moynes DR. A comparative electromyographic analysis of the shoulder during pitching. Professional versus amateur pitchers. *Am J Sports Med*. 1987;15:586–90.
  42. Safran MR. Nerve injury about the shoulder in athletes, part 1: suprascapular nerve and axillary nerve. *Am J Sports Med*. 2004;32:803–19.
  43. Safran MR. Nerve injury about the shoulder in athletes, part 2: long thoracic nerve, spinal accessory nerve, burners/stingers, thoracic outlet syndrome. *Am J Sports Med*. 2004;32:1063–76.
  44. Adams JT, DeWeese JA. "Effort" thrombosis of the axillary and subclavian veins. *J Trauma*. 1971;11:923–30.
  45. Nemmers DW, Thorpe PE, Knibbe MA, Beard DW. Upper extremity venous thrombosis. Case report and literature review. *Orthop Rev*. 1990;19:164–72.

46. Takach TJ, Kane PN, Madjarov JM, Holleman JH, Nussbaum T, Robicsek F, Roush TS. Arteriopathy in the high-performance athlete. *Tex Heart Inst J.* 2006;33:482–6.
47. Tullos HS, Erwin WD, Woods GW, Wukasch DC, Cooley DA, King JW. Unusual lesions of the pitching arm. *Clin Orthop Relat Res.* 1972;88:169–82.
48. Degeorges R, Reynaud C, Becquemin JP. Thoracic outlet syndrome surgery: long-term functional results. *Ann Vasc Surg.* 2004;18:558–65.
49. Nichols AW. The thoracic outlet syndrome in athletes. *J Am Board Fam Pract.* 1996;9:346–55.
50. Jobe CM. Superior glenoid impingement. Current concepts. *Clin Orthop Relat Res.* 1996;330:98–107.
51. Walch G, Boileau P, Noel E, Donell ST. Impingement of the deep surface of the supraspinatus tendon on the posterosuperior glenoid rim: an arthroscopic study. *J Shoulder Elb Surg.* 1992;1:238–45.
52. Halbrecht JL, Tirman P, Atkin D. Internal impingement of the shoulder: comparison of findings between the throwing and nonthrowing shoulders of college baseball players. *Arthroscopy.* 1999;15:253–8.
53. Mihata T, McGarry MH, Kinoshita M, Lee TQ. Excessive glenohumeral horizontal abduction as occurs during the late cocking phase of the throwing motion can be critical for internal impingement. *Am J Sports Med.* 2010;38:369–74.
54. Mihata T, Gates J, McGarry MH, Neo M, Lee TQ. Effect of posterior shoulder tightness on internal impingement in a cadaveric model of throwing. *Knee Surg Sports Traumatol Arthrosc.* 2015;23:548–54.
55. Glousman R, Jobe F, Tibone J, Moynes D, Antonelli D, Perry J. Dynamic electromyographic analysis of the throwing shoulder with glenohumeral instability. *J Bone Joint Surg Am.* 1988;70:220–6.
56. Mihata T, Gates J, McGarry MH, Lee J, Kinoshita M, Lee TQ. Effect of rotator cuff muscle imbalance on forceful internal impingement and peel-back of the superior labrum: a cadaveric study. *Am J Sports Med.* 2009;37:2222–7.
57. Ludewig PM, Cook TM. Translations of the humerus in persons with shoulder impingement symptoms. *J Orthop Sports Phys Ther.* 2002;32:248–59.
58. Muraki T, Yamamoto N, Zhao KD, Sperling JW, Steinmann SP, Cofield RH, An KN. Effect of posteroinferior capsule tightness on contact pressure and area beneath the coracoacromial arch during pitching motion. *Am J Sports Med.* 2010;38:600–7.
59. Pradhan RL, Itoi E, Hatakeyama Y, Urayama M, Sato K. Superior labral strain during the throwing motion. A cadaveric study. *Am J Sports Med.* 2001;29:488–92.
60. Kuhn JE, Lindholm SR, Huston LJ, Soslowsky LJ, Blasier RB. Failure of the biceps superior labral complex: a cadaveric biomechanical investigation comparing the late cocking and early deceleration positions of throwing. *Arthroscopy.* 2003;19:373–9.

61. Mihata T, Takeda A, Kawakami T, Itami Y, Watanabe C, Doi M, Neo M. Isolated glenohumeral range of motion, excluding side-to-side difference in humeral retroversion, in asymptomatic high-school baseball players. *Knee Surg Sports Traumatol Arthrosc.* 2014;24(6):1911–7.
62. Burkhart SS, Morgan CD, Kibler WB. The disabled throwing shoulder: spectrum of pathology part III: the SICK scapula, scapular dyskinesis, the kinetic chain, and rehabilitation. *Arthroscopy.* 2003;19:641–61.
63. Kibler WB. The role of the scapula in athletic shoulder function. *Am J Sports Med.* 1998;26:325–37.
64. Paine RM, Voight M. The role of the scapula. *J Orthop Sports Phys Ther.* 1993;18:386–91.
65. Warner JJ, Micheli LJ, Arslanian LE, Kennedy J, Kennedy R. Scapulothoracic motion in normal shoulders and shoulders with glenohumeral instability and impingement syndrome. A study using Moire topographic analysis. *Clin Orthop Relat Res.* 1992;285:191–9.
66. Jobe FW, Kvitne RS, Giangarra CE. Shoulder pain in the overhand or throwing athlete. The relationship of anterior instability and rotator cuff impingement. *Orthop Rev.* 1989;18:963–75.
67. Jobe FW, Pink M. Classification and treatment of shoulder dysfunction in the overhead athlete. *J Orthop Sports Phys Ther.* 1993;18:427–32.
68. Kvitne RS, Jobe FW, Jobe CM. Shoulder instability in the overhand or throwing athlete. *Clin Sports Med.* 1995;14:917–35.
69. Cain EL Jr, Dugas JR, Wolf RS, Andrews JR. Elbow injuries in throwing athletes: a current concepts review. *Am J Sports Med.* 2003;31(4):621–35.
70. Kelly BT, Kadrmaz WR, Speer KP. The manual muscle examination for rotator cuff strength. An electromyographic investigation. *Am J Sports Med.* 1996;24:581–8.
71. Daniels L, Worthingham C. *Muscle testing.* Philadelphia: WB Saunders; 1980. p. 118–20.
72. Gerber C, Krushell RJ. Isolated rupture of the tendon of the subscapularis muscle. Clinical features in 16 cases. *J Bone Joint Surg Br.* 1991;73:389–94.
73. Bigliani LU, Codd TP, Connor PM, Levine WN, Littlefield MA, Hershon SJ. Shoulder motion and laxity in the professional baseball player. *Am J Sports Med.* 1997;25:609–13.
74. 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;30:20–6.
75. Myers JB, Laudner KG, Pasquale MR, Bradley JP, Lephart SM. Glenohumeral range of motion deficits and posterior shoulder tightness in throwers with pathologic internal impingement. *Am J Sports Med.* 2006;34:385–91.

76. Ticker JB, Beim GM, Warner JJ. Recognition and treatment of refractory posterior capsular contracture of the shoulder. *Arthroscopy*. 2000;16:27–34.
77. Kibler WB, Kuhn JE, Wilk K, Sciascia A, Moore S, Laudner K, Ellenbecker T, Thigpen C, Uhl T. The disabled throwing shoulder: spectrum of pathology-10-year update. *Arthroscopy*. 2013;29:141–161.e26.
78. Osbahr DC, Cannon DL, Speer KP. Retroversion of the humerus in the throwing shoulder of college baseball pitchers. *Am J Sports Med*. 2002;30:347–53.
79. Reagan KM, Meister K, Horodyski MB, Werner DW, Carruthers C, Wilk K. Humeral retroversion and its relationship to glenohumeral rotation in the shoulder of college baseball players. *Am J Sports Med*. 2002;30:354–60.
80. Edwards SL, Lee JA, Bell JE, Packer JD, Ahmad CS, Levine WN, Bigliani LU, Blaine TA. Nonoperative treatment of superior labrum anterior posterior tears: improvements in pain, function, and quality of life. *Am J Sports Med*. 2010;38:1456–61.
81. Cohen SB, Sheridan S, Ciccotti MG. Return to sports for professional baseball players after surgery of the shoulder or elbow. *Sports Health*. 2011;3:105–11.
82. Field LD, Savoie FH. Arthroscopic suture repair of superior labral detachment lesions of the shoulder. *Am J Sports Med*. 1993;21:783–90.
83. Morgan CD, Burkhart SS, Palmeri M, Gillespie M. Type II SLAP lesions: three subtypes and their relationships to superior instability and rotator cuff tears. *Arthroscopy*. 1998;14:553–65.
84. Neri BR, ElAttrache NS, Owsley KC, Mohr K, Yocum LA. Outcome of type II superior labral anterior posterior repairs in elite overhead athletes: Effect of concomitant partial-thickness rotator cuff tears. *Am J Sports Med*. 2011;39:114–20.
85. Pagnani MJ, Speer KP, Altchek DW, Warren RF, Dines DM. Arthroscopic fixation of superior labral lesions using a biodegradable implant: a preliminary report. *Arthroscopy*. 1995;11:194–8.
86. Rhee YG, Lee DH, Lim CT. Unstable isolated SLAP lesion: clinical presentation and outcome of arthroscopic fixation. *Arthroscopy*. 2005;21:1099.
87. Yoneda M, Hirooka A, Saito S, Yamamoto T, Ochi T, Shino K. Arthroscopic stapling for detached superior glenoid labrum. *J Bone Joint Surg Br*. 1991;73:746–50.
88. Namdari S, Baldwin K, Ahn A, Huffman GR, Sennett BJ. Performance after rotator cuff tear and operative treatment: a case-control study of major league baseball pitchers. *J Athl Train*. 2011;46:296–302.
89. Tibone JE, Elrod B, Jobe FW, Kerlan RK, Carter VS, Shields CL, Lombardo SJ, Yocum L. Surgical treatment of tears of the rotator cuff in athletes. *J Bone Joint Surg Am*. 1986;68:887–91.

90. Ide J, Maeda S, Takagi K. Arthroscopic transtendon repair of partial-thickness articular-side tears of the rotator cuff: anatomical and clinical study. *Am J Sports Med.* 2005;33:1672–9.
91. Reynolds SB, Dugas JR, Cain EL, McMichael CS, Andrews JR. Debridement of small partial-thickness rotator cuff tears in elite overhead throwers. *Clin Orthop Relat Res.* 2008;466:614–21.
92. Budoff JE, Rodin D, Ochiai D, Nirschl RP. Arthroscopic rotator cuff debridement without decompression for the treatment of tendinosis. *Arthroscopy.* 2005;21:1081–9.
93. Cordasco FA, Backer M, Craig EV, Klein D, Warren RF. The partial-thickness rotator cuff tear: is acromioplasty without repair sufficient? *Am J Sports Med.* 2002;30:257–60.
94. Liem D, Alci S, Dedy N, Steinbeck J, Marquardt B, Mollenhoff G. Clinical and structural results of partial supraspinatus tears treated by subacromial decompression without repair. *Knee Surg Sports Traumatol Arthrosc.* 2008;16:967–72.
95. Park JY, Yoo MJ, Kim MH. Comparison of surgical outcome between bursal and articular partial thickness rotator cuff tears. *Orthopedics.* 2003;26:387–90.
96. Snyder SJ, Pachelli AF, Del Pizzo W, Friedman MJ, Ferkel RD, Pattee G. Partial thickness rotator cuff tears: results of arthroscopic treatment. *Arthroscopy.* 1991;7:1–7.
97. Castagna A, Delle Rose G, Conti M, Snyder SJ, Borroni M, Garofalo R. Predictive factors of subtle residual shoulder symptoms after transtendinous arthroscopic cuff repair: a clinical study. *Am J Sports Med.* 2009;37:103–8.
98. Franceschi F, Papalia R, Del Buono A, Vasta S, Costa V, Maffulli N, Denaro V. Articular-sided rotator cuff tears: which is the best repair? A three-year prospective randomised controlled trial. *Int Orthop.* 2013;37:1487–93.
99. Lo IK, Burkhart SS. Transtendon arthroscopic repair of partial-thickness, articular surface tears of the rotator cuff. *Arthroscopy.* 2004;20:214–20.
100. Shin SJ. A comparison of 2 repair techniques for partial-thickness articular-sided rotator cuff tears. *Arthroscopy.* 2012;28:25–33.
101. Spencer EE. Partial-thickness articular surface rotator cuff tears: an all-inside repair technique. *Clin Orthop Relat Res.* 2010;468:1514–20.
102. Waibl B, Buess E. Partial-thickness articular surface supraspinatus tears: a new transtendon suture technique. *Arthroscopy.* 2005;21:376–81.
103. Deutsch A. Arthroscopic repair of partial-thickness tears of the rotator cuff. *J Shoulder Elb Surg.* 2007;16:193–201.

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104. Kamath G, Galatz LM, Keener JD, Teefey S, Middleton W, Yamaguchi K. Tendon integrity and functional outcome after arthroscopic repair of high-grade partial-thickness supraspinatus tears. *J Bone Joint Surg Am.* 2009;91:1055–62.
  105. Porat S, Nottage WM, Fouse MN. Repair of partial thickness rotator cuff tears: a retrospective review with minimum two-year follow-up. *J Shoulder Elb Surg.* 2008;17:729–31.





# Shoulder Injuries in Contact Athletes

# 3

Sungjoon Lim and Lennard Funk

## 3.1 Introduction

The term ‘contact sports’ has been used as an elusive category throughout the literature without a universally accepted definition. Rice [1] subdivided contact sports into contact and collision sports to estimate the relative risk of injury). In collision sport, athletes purposely hit or collide with each other or inanimate objects, often with great force, as in rugby, American football and ice hockey. Contact sport involves the athlete making routine contact with each other with lesser force, as in basketball and soccer. Collision sport implies greater injury risk, but these terms have been used synonymously across the literature.

Due to the physical nature of these sports, they entail frequent musculoskeletal injuries. Studies from rugby and American football have shown that the shoulder is among the most common site of injury that is responsible for considerable loss of play-time. The acromioclavicular (AC) joint injury is the most common shoulder

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injury, accounting for 32–41% of collision sport injuries, followed by glenohumeral instability and rotator cuff injury [2, 3]. Among these injuries, shoulder dislocation and instability account for most play-time loss. Headey et al. [2] reported that the most severe type of shoulder injury in Professional Rugby Union, as assessed from days absent, was shoulder dislocation and instability (mean 81 days absent from play). Physicians caring for these patients must appreciate that other less common shoulder injuries can be the cause of the discomfort. Fractures around the shoulder (scapula, clavicle, or proximal humerus), long head of the biceps tendon injury, SLAP tears are some of the diagnoses that should not be overlooked.

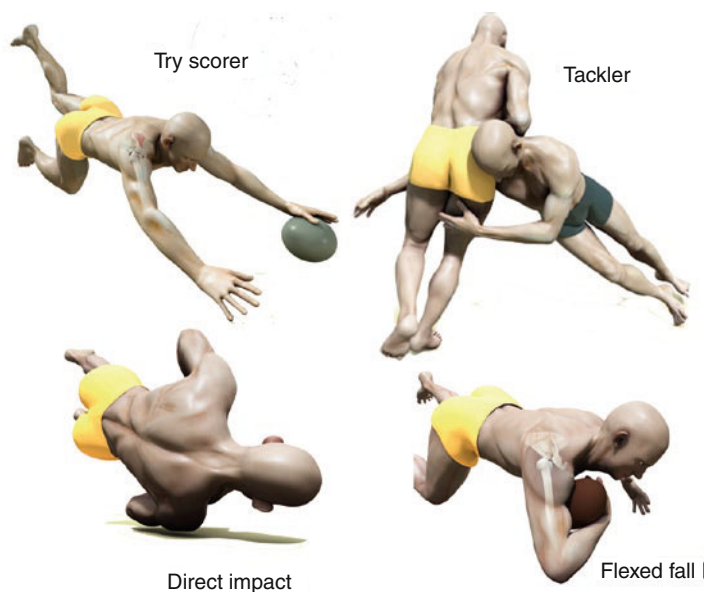
The goal of treatment should be returning the athlete to play safely with least amount of play-time lost whilst minimizing the chance of recurrence. Therefore, it is paramount to understand the nature of the injury and different treatment options to make the best possible decision for the injured athlete.

This chapter will discuss some of the most common injuries in contact athletes. Aetiology, evaluation, and treatment characteristics of AC joint injury, glenohumeral instability, rotator cuff injury will be discussed in the following sections.

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## 3.2 Aetiology

In contact athletes, shoulder injuries can be the result of a direct blow to the shoulder or indirectly landing on the affected shoulder. The majority of shoulder injuries are sustained in the tackling event (65% of all shoulder injuries) [2]. Video analysis studies have helped understand the common mechanisms of these sport injuries that result in predictable patterns of shoulder pathology [4]. In the video analysis study of twenty-four elite rugby players, Crichton et al. [4] identified the three different mechanisms of injury (Fig. 3.1): ‘Try-Scorer’, characterised by hyperflexion of the out-stretched ball-carrying arm to score a try; the ‘Tackler’, extension of the abducted arm behind the player while tackling an opponent; and the ‘Direct Impact’, a direct blow to the lateral aspect of the shoulder when held by the side in neutral or slight adduction. Posterior glenohumeral instability can result from a ‘blocking



**Fig. 3.1** Common mechanisms of shoulder injury in rugby (courtesy of Leonard Funk, <http://www.shoulderdoc.co.uk>)

injury', as can be seen commonly from an American football lineman block opposing force with the arm is placed in a flexed ( $90^\circ$ ) and internally rotated position [5, 6]. The physician should keep in mind that complex injuries can occur after a single traumatic event. Tischer et al. [7] reported 18.2% (fourteen of seventy-seven patients) of AC joint injuries showed the concomitant intra-articular injuries that required additional surgical intervention.

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## 3.3 Clinical Evaluation

### 3.3.1 History/Presentations

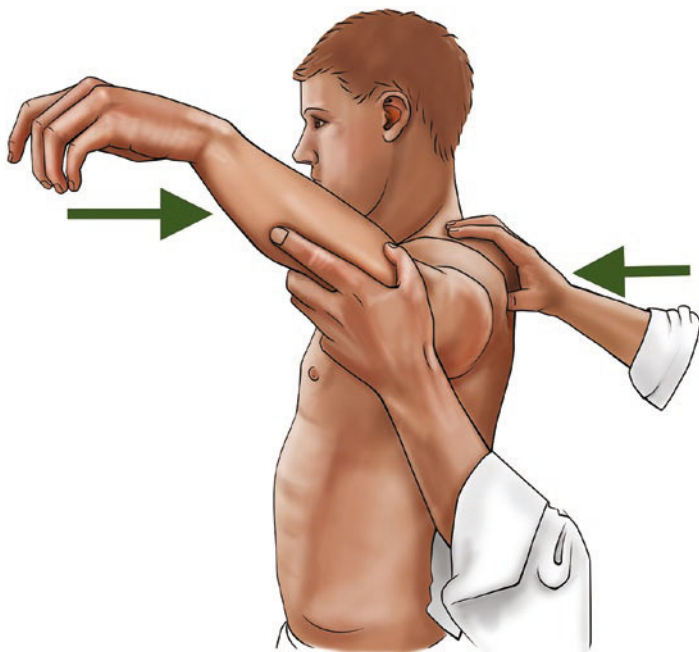
A thorough investigation of relevant history and physical examination are essential for making the correct diagnosis. Baseline information including the patient's age, dominant arm, type of the

sport and playing position are first obtained. It is also important to ask about the history of previous injury or treatment of the shoulder, any events of dislocation of other joints and the presence of known connective tissue disorders in the patient or family.

The mechanism of injury should be investigated in detail. The athlete with a shoulder dislocation may recall a specific traumatic instability event or incomplete subluxating events. The magnitude of the impact or collision, the position of the arm at the time of the trauma, how the shoulder was reduced (whether the shoulder was manually reduced or spontaneously reduced) are important. Patients with posterior shoulder instability primarily have pain and weakness rather than frank instability. However, a recent large-scale epidemiologic study showed that 54% of patients presented with subluxation/instability as primary complaint [8]. For patients who complain of recurrent instability, the number and frequency of recurrent episodes should be investigated. Whether the injury is affecting their activities of daily living as well sports are important factors in considering the surgical treatment.

### **3.3.2 Physical Examination**

The physical examination begins with inspection. Both shoulders need to be exposed for inspection of any deformity or muscle wasting. Active and passive range of motion testing must be performed and compared with the contralateral shoulder. Thorough palpation of the shoulder is carried out with special attention to the AC joint, biceps groove, rotator cuff insertion sites. Shoulder strength testing of each rotator cuff muscle must be assessed as well. It is essential to assess the neuromuscular status, with special attention to the axillary nerve (Deltoid muscle function and “regimental badge” paraesthesia). In addition, generalized ligamentous laxity has been associated with shoulder instability [9]. The Beighton score is used to determine the degree of generalized ligamentous laxity [10]. Special tests, including the Gagey’s test, sulcus sign, apprehension, relocation, and surprise tests may enable the physician to identify the shoulder instability pattern (Fig. 3.2).

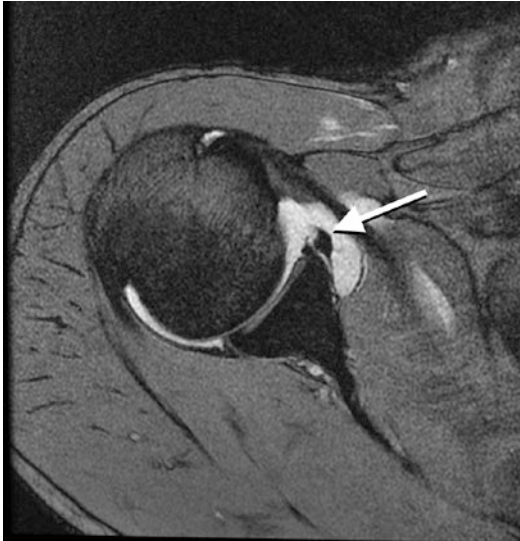


**Fig. 3.2** Anterior apprehension test (courtesy of Lennard Funk, <http://www.shoulderdoc.co.uk>)

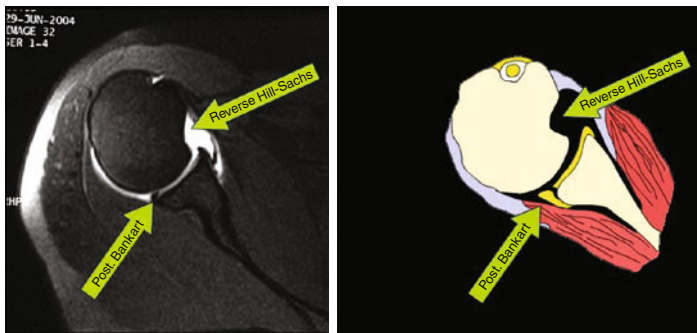
### 3.4 Imaging

Routine radiographic studies consist of orthogonal view of the shoulder joint: Anteroposterior (AP), axillary lateral and scapula Y view. The AC joint can be best visualized with a 15° cephalic tilt view known as the Zanca View. West Point axillary views often demonstrate the presence of a Hill-Sachs lesion of the humeral head and may demonstrate a loss of bone at the anterior surface of the glenoid. Stryker notch view can help detect humeral head bone loss (Hill-Sachs lesion).

Advanced imaging can be very helpful for defining the patho-anatomy and for planning the surgical approach. Magnetic resonance imaging (MRI) with arthrogram is an extremely useful tool to assess the soft tissue injuries. Classic Bankart lesion (Fig. 3.3), reverse Bankart lesion (Fig. 3.4), humeral avulsion of



**Fig. 3.3** Bankart tear on MR arthrogram (white arrow)



**Fig. 3.4** Reverse Bankart tear and reverse Hill-Sachs lesion on MR arthrogram



**Fig. 3.5** HAGL on coronal MR arthrogram (white arrow)

glenohumeral ligament (HAGL) can also be assessed (Fig. 3.5). The presence of a bone bruise or defect in the posterolateral aspect of humerus (Hill-Sachs lesion) may also be present. Rotator cuff tendon injury ranging from contusion to full thickness tear can be seen.

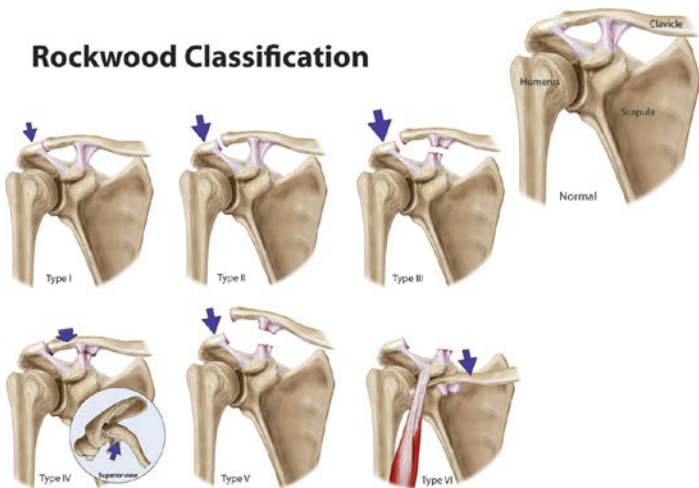
Computed tomography (CT) scan is best to detect bony injuries. Fractures of the clavicle, scapula, and proximal humerus can be better assessed. Three-dimensional (3D) CT reconstruction of the glenoid is useful to measure glenoid bone loss which has been recently understood to be a cause of failure of soft tissue surgery if not properly addressed.

## 3.5 Management

### 3.5.1 AC Joint Injury

The AC joint is a diarthrodial joint, stabilized by the AC ligament and the coracoclavicular (CC) ligaments. The AC ligament, particularly the superior and posterior ligament, provides stability in the AP plane whereas CC ligaments provide stability in the vertical plane. Injuries are typically classified using the Rockwood Classification [11]. Type I injuries are isolated sprains of the AC ligament. Type II injuries are a torn AC ligament and sprained CC ligaments. Type III–VI injuries are torn AC and torn CC ligaments with differential severity and location of AC displacement (Fig. 3.6).

Traditional treatment of AC joint injuries is based on the Rockwood classification, but there is no high level evidence to



**Fig. 3.6** Rockwood classification for AC joint injuries (courtesy of Lennard Funk, <http://www.shoulderdoc.co.uk>)



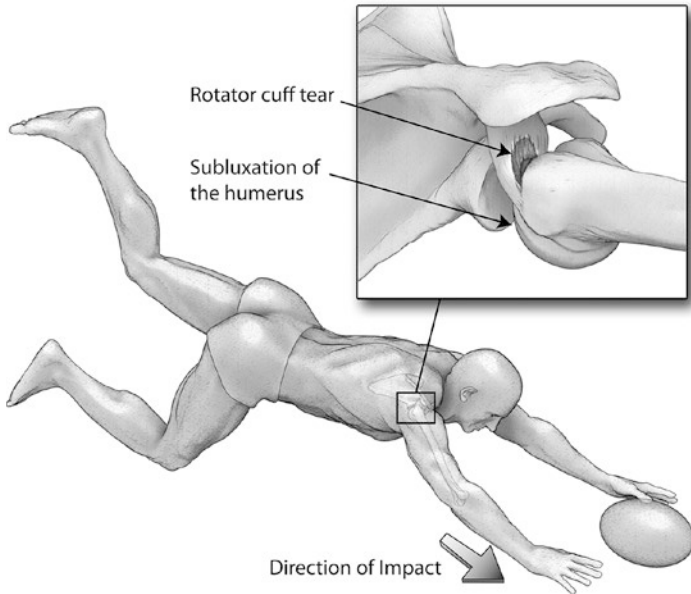
support this. Generally Type I and II injuries are treated non-operatively with brief period of immobilization, analgesia, cryotherapy, and physical therapy. Return to play is dependent on the player's comfort and ability to perform sport-specific activity. The management of Type III injuries remains controversial. Type III, IV and V injuries can be treated non-operatively initially, as with Type I and II injuries. If the athlete is not coping or progressing with rehabilitation then surgical stabilization is indicated. The time of season and specific sporting requirements should be taken in to consideration when timing surgery. Previous studies have shown that most AC joint injuries in contact athletes are low-grade (Types I and II) and only a small proportion of the injuries (1.7–2.4%) required surgical management [12, 13].

Numerous surgical techniques have been described in the treatment of AC joint injuries. There is little published research focusing on the surgical treatment of AC joint injuries in contact athletes specifically. Marcheggiani Muccioli et al. [14] reported on the outcome of the AC joint reconstruction with the LARS ligament in contact athletes. After reconstruction, the athletes were able to return to full contact sport at median of 4 months using an anatomical artificial ligament technique. Athletes who sustained low-grade injury (Type I or II) may develop posttraumatic osteoarthritis of the AC joint. This can be managed with distal clavicle excision [15].

### **3.5.2 Rotator Cuff Injury**

Injuries of the rotator cuff in contact athletes range from contusion to full thickness tear. A study of rotator cuff contusions in a North American professional football team found that contusions accounted for nearly half (47%) of all shoulder injuries, approximately 5.5 contusions per season [16].

The predominant mechanism of injury for full thickness tears is traumatic and usually associated with shoulder dislocations or subluxations [16, 17]. Tambe et al. [18] reported on the



**Fig. 3.7** Try scoring injury with anterior shoulder subluxation and rotator cuff tear (courtesy of Lennard Funk, <http://www.shoulderdoc.co.uk>)

arthroscopic repair of full thickness rotator cuff tear in professional rugby players. They found about half of the patients had concomitant labral injuries or bony Bankart lesions (Fig. 3.7).

Management of rotator cuff injury is dependent on the severity of the injury. Cuff contusions and partial tears can be managed with non-operative management including pain control and rotator cuff strengthening. Subacromial corticosteroid injections can be considered for athletes with persistent bursal inflammation and pain. Patients who do not respond to these conservative treatments, and those with full thickness cuff tears are indications for surgical management. Advancements in arthroscopic techniques has enabled the successful treatment of cuff tears in contact athletes with little soft tissue damage. A confident repair

of rotator cuff and other associated pathology allows the athlete to participate in early return to sport rehabilitation programme. In the series by Tambe et al., 91.7% (ten of eleven) of the elite rugby players were able to return to their pre-injury level of competition early (4.8 months) after arthroscopic rotator cuff repair [18].

### 3.5.3 Glenohumeral Joint Instability

Glenohumeral joint instability is common and often a disabling injury for the contact athlete. The direction of instability is an important factor for deciding treatment plan. Anterior instability comprises majority of shoulder instability cases, but recent studies have suggested that posterior instability is increasing and consists as much as 10–30% of traumatic instability in contact athletes [5]. Posterior instability is often associated with anterior instability and superior labral tears [8, 19]

If the injury occurs close to the end of the season or during the off-season, surgical stabilization can be easily selected with minimum play-time loss. The choice of early surgery or non-operative treatment should be tailored to the individual athlete with the mid-season injury, taking the following factors into consideration: The type of sport, level of competition, position of the athlete, age of the athlete, pathoanatomy (e.g. glenoid bone loss, Hill-Sachs lesion), timing of the injury in the competitive season, the athlete's career goal, expectation from the coaching staff or the family member. The orthopedic surgeon needs to gather all the necessary information, assess the risk of recurrence, and counsel the patient and family for the best treatment option.

Surgical stabilization can get the athletes side-lined for 4–6 months, which is usually season-ending. Athletes may be strongly motivated to play through the rest of the season, especially when external factors (contract, scholarship or personal goals etc) are involved. In this scenario, the decision to select non-operative treatment during the season with surgery reserved for the off-season is a viable option. For injured athletes who wish to

return to play with non-operative treatment, accelerated rehabilitation protocols can be employed [20, 21]. The immobilization period is minimized (none to 7 days), and supervised rehabilitation program is initiated as tolerated from the first day after injury. At first, gentle ROM exercises and cryotherapy to ease the discomfort are commenced. Once the full range of motion without discomfort is possible, rotator cuff and periscapular muscle strengthening exercises are initiated. When ROM and strength are symmetrical, sport-specific drills are initiated and return to play with a motion-limiting brace is considered when all the following criteria are met [22].

1. Little or no pain
2. Patient's subjective impression of stability
3. Near-normal ROM
4. Near-normal strength
5. Normal functional ability
6. Normal sport-specific skills

Buss et al. [23] managed 30 in-season athletes non-operatively with physical therapy and use of a motion-limiting brace, if appropriate. They reported that 27 of 30 athletes were able to return to play for either part or all of the remaining season, although 41% of the returned athletes experienced recurrence. Bottoni et al. [24] however, reported higher recurrence rate of 75% in athletes who were treated non-operatively.

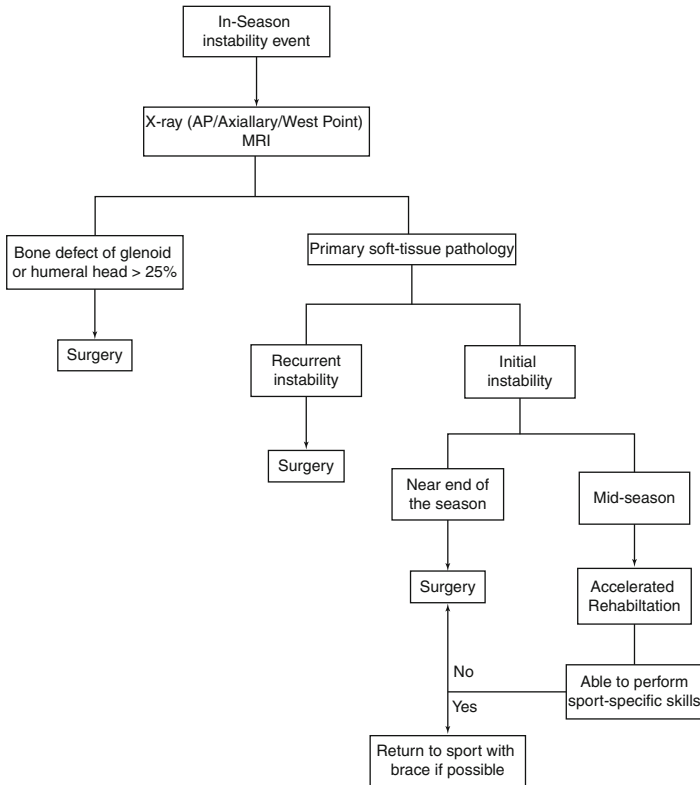
The failure of non-operative treatment, or the presence of high-risk factors (glenoid bone loss, engaging Hill-Sachs lesion), warrant early surgical treatment. The optimal surgical treatment option has been under constant debate. Historically, open Bankart repair with capsular shift had been considered as the gold standard for contact athletes over arthroscopic stabilization. However, with the development of new implants and advances in surgical techniques resulting in comparable outcome [25, 26], arthroscopic stabilization has gained popularity over open.

Recently, the risk factors for failure of arthroscopic surgery have been recognized. Burkhart and De Beer [27] reported that recurrence rate was much higher after arthroscopic Bankart repair for those patients who had glenoid bone loss and engaging Hill-Sachs lesions. They found that patients with these lesions have 67% recurrence rate when those without have only 4% recurrent rate. Subsequent studies have suggested that even small amounts of glenoid bone loss in contact athletes lead to recurrent instability in contact athletes with arthroscopic stabilization and a bone procedure is probably a better option [28–30]

The most commonly used bone-procedure is the Latarjet procedure, where coracoid process is transferred to the antero-inferior glenoid. Biomechanical studies have demonstrated the stabilizing mechanism of Latarjet procedure: bone-graft effect, the sling effect, and capsular-repair effect [31]. These procedures are non-anatomic and not without complications. Complication rates ranging from 5 to 30% have been reported [32–35].

Humeral head lesions are commonly present in patients with anterior shoulder instability. Burkhart and DeBeer [27] reported that some Hill-Sachs lesions ‘engage’ the anterior glenoid rim in the abduction and external rotation position, resulting in high recurrence rate after arthroscopic surgery. Yamamoto et al. [36] developed the glenoid track concept, which is the contact zone between humerus and glenoid throughout the range of motion. The glenoid track can be used to evaluate the risk of engagement and help guide clinical decision making. If the Hill-Sachs lesion is present within the glenoid track (on-track), there is no risk of engagement. If the Hill-Sachs lesion extends medially over the glenoid track (off-track), there is high a risk of engagement during functional movement. Engaging Hill-Sachs lesions or off-track lesions should be addressed if engagement is evident following glenoid augmentation [37]. Some of the procedures to address large humeral head defects include humeral head osteochondral allograft, and the infraspinatus remplissage procedure.

The following algorithm depicts the management of traumatic anterior shoulder instability during season (Fig. 3.8)



**Fig. 3.8** Algorithm for the management of traumatic anterior shoulder instability during season for the contact athlete

### 3.6 Conclusion

Shoulder injuries in young contact athletes are common. AC joint injury, rotator cuff injury and glenohumeral instability are more frequently encountered, but treating physicians should understand the variety and complexity of injury in this group of athletes. Obtaining the relevant history and a systematic physical examina-

tion are essential for diagnosis. Simple radiographs and advanced imaging can help detect the pathologic lesion and determine the choice of optimal treatment. Most AC joint injuries can be successfully managed non-operatively. Rotator cuff contusion and partial thickness tear can also be a good indication of conservative management with arthroscopic rotator cuff repair having good return to sports results in selected individuals. Full thickness rotator cuff tears generally require early surgical repair. Rotator cuff tears are often associated with other pathologies, particularly labral tears. Several factors should be considered when treating an in-season athlete with a shoulder instability. Athletes who wish to compete for the remaining season can be managed with accelerated rehabilitation and return to play within a few weeks if specific criteria are met. Motion-controlling braces may help stabilize the shoulder during sports activities. Athletes with bone loss, recurrent instability and end of the season injuries are candidates for surgical stabilization. Bony lesions that are associated with high recurrence rates require bone reconstruction procedures.

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## Q&A

- (1) What are the differences between contact and collision sports and does this differentiation important in studies of sports injuries of the shoulder?

Answer: Collision sports involve intentional collisions with opponents, such as American Football and rugby. Contact sports involve inadvertent contact with opponents, such as basketball and soccer.

- (2) What are the most common contact sports shoulder injuries and how frequent are concomitant problems?

Answer: The most common injuries are AC joint injuries, labral and rotator cuff injuries. Concomitant injuries are frequent, with 20% of AC joint injuries having an associated labral or cuff tear. Rotator cuff tears commonly are associated with a glenohumeral joint subluxation or dislocation in the collision/contact athlete.

(3) Which ACJ injuries are best treated surgically?

Answer: High grade injuries that are not settling in-season with a short period of rehabilitation and end of season injuries.

(4) Is open stabilisation surgery still the gold standard operation for instability in the athletes' shoulder?

Answer: It has been superseded by arthroscopic procedures, but bony reconstructions are indicated when there is bone involvement.

(5) How does the "glenoid track" concept aid decision making?

Answer: The glenoid track is the contact area between the humeral head and glenoid in the position of abduction and external rotation. If this is reduced by glenoid bone loss and/or a large medial Hil-Sachs lesion then the risk of recurrence is very high.

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## References

1. Rice SG, American Academy of Pediatrics Council on Sports M, Fitness. Medical conditions affecting sports participation. *Pediatrics*. 2008;121(4):841–8.
2. Headey J, Brooks JH, Kemp SP. The epidemiology of shoulder injuries in English professional rugby union. *Am J Sports Med*. 2007;35(9):1537–43.
3. Kaplan LD, Flanigan DC, Norwig J, Jost P, Bradley J. Prevalence and variance of shoulder injuries in elite collegiate football players. *Am J Sports Med*. 2005;33(8):1142–6.
4. Crichton J, Jones DR, Funk L. Mechanisms of traumatic shoulder injury in elite rugby players. *Br J Sports Med*. 2012;46(7):538–42.
5. Owens BD, Campbell SE, Cameron KL. Risk factors for posterior shoulder instability in young athletes. *Am J Sports Med*. 2013;41(11):2645–9.
6. Provencher MT, LeClere LE, King S, McDonald LS, Frank RM, Mologne TS, et al. Posterior instability of the shoulder: diagnosis and management. *Am J Sports Med*. 2011;39(4):874–86.
7. Tischer T, Salzmann GM, El-Azab H, Vogt S, Imhoff AB. Incidence of associated injuries with acute acromioclavicular joint dislocations types III through V. *Am J Sports Med*. 2009;37(1):136–9.
8. Lanzi JT Jr, Chandler PJ, Cameron KL, Bader JM, Owens BD. Epidemiology of posterior glenohumeral instability in a young athletic population. *Am J Sports Med*. 2017;45(14):3315–21.
9. Cameron KL, Duffey ML, DeBerardino TM, Stoneman PD, Jones CJ, Owens BD. Association of generalized joint hypermobility with a history of glenohumeral joint instability. *J Athl Train*. 2010;45(3):253–8.



10. Beighton P, Solomon L, Soskolne CL. Articular mobility in an African population. *Ann Rheum Dis.* 1973;32(5):413–8.
11. Nguyen V, Williams G, Rockwood C. Radiography of acromioclavicular dislocation and associated injuries. *Crit Rev Diagn Imaging.* 1991;32(3):191–228.
12. Dragoo JL, Braun HJ, Bartlinski SE, Harris AH. Acromioclavicular joint injuries in National Collegiate Athletic Association football: data from the 2004–2005 through 2008–2009 National Collegiate Athletic Association Injury Surveillance System. *Am J Sports Med.* 2012;40(9):2066–71.
13. Lynch TS, Saltzman MD, Ghodasra JH, Bilimoria KY, Bowen MK, Nuber GW. Acromioclavicular joint injuries in the National Football League: epidemiology and management. *Am J Sports Med.* 2013;41(12):2904–8.
14. Marcheggiani Muccioli GM, Manning C, Wright P, Grassi A, Zaffagnini S, Funk L. Acromioclavicular joint reconstruction with the LARS ligament in professional versus non-professional athletes. *Knee Surg Sports Traumatol Arthrosc.* 2016;24(6):1961–7.
15. Mouhsine E, Garofalo R, Crevoisier X, Farron A. Grade I and II acromioclavicular dislocations: results of conservative treatment. *J Shoulder Elb Surg.* 2003;12(6):599–602.
16. Cohen SB, Towers JD, Bradley JP. Rotator cuff contusions of the shoulder in professional football players: epidemiology and magnetic resonance imaging findings. *Am J Sports Med.* 2007;35(3):442–7.
17. Goldberg JA, Chan KY, Best JP, Bruce WJ, Walsh W, Parry W. Surgical management of large rotator cuff tears combined with instability in elite rugby football players. *Br J Sports Med.* 2003;37(2):179–81.
18. Tambe A, Badge R, Funk L. Arthroscopic rotator cuff repair in elite rugby players. *Int J Shoulder Surg.* 2009;3(1):8–12.
19. Engelsma Y, Willems WJ. Arthroscopic stabilization of posterior shoulder instability. *Knee Surg Sports Traumatol Arthrosc.* 2010;18(12):1762–6.
20. Dickens JF, Owens BD, Cameron KL, Kilcoyne K, Allred CD, Svoboda SJ, et al. Return to play and recurrent instability after in-season anterior shoulder instability: a prospective multicenter study. *Am J Sports Med.* 2014;42(12):2842–50.
21. Owens BD, Dickens JF, Kilcoyne KG, Rue JP. Management of mid-season traumatic anterior shoulder instability in athletes. *J Am Acad Orthop Surg.* 2012;20(8):518–26.
22. McCarty EC, Ritchie P, Gill HS, McFarland EG. Shoulder instability: return to play. *Clin Sports Med.* 2004;23(3):335–51.
23. Buss DD, Lynch GP, Meyer CP, Huber SM, Freehill MQ. Nonoperative management for in-season athletes with anterior shoulder instability. *Am J Sports Med.* 2004;32(6):1430–3.
24. Bottoni CR, Wilckens JH, DeBerardino TM, D'Alleyrand JC, Rooney RC, Harpriste JK, et al. 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.
25. Kim SH, Ha KI, Kim SH. Bankart repair in traumatic anterior shoulder instability: open versus arthroscopic technique. *Arthroscopy.* 2002;18(7):755–63.
  26. Potzl W, Witt KA, Hackenberg L, Marquardt B, Steinbeck J. Results of suture anchor repair of anteroinferior shoulder instability: a prospective clinical study of 85 shoulders. *J Shoulder Elb Surg.* 2003;12(4):322–6.
  27. 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.
  28. Piasecki DP, Verma NN, Romeo AA, Levine WN, Bach BR Jr, Provencher MT. Glenoid bone deficiency in recurrent anterior shoulder instability: diagnosis and management. *J Am Acad Orthop Surg.* 2009;17(8):482–93.
  29. Porcellini G, Campi F, Paladini P. Arthroscopic approach to acute bony Bankart lesion. *Arthroscopy.* 2002;18(7):764–9.
  30. Shaha JS, Cook JB, Song DJ, Rowles DJ, Bottoni CR, Shaha SH, et al. Redefining “critical” bone loss in shoulder instability: functional outcomes worsen with “subcritical” bone loss. *Am J Sports Med.* 2015;43(7):1719–25.
  31. Yamamoto N, Muraki T, An KN, Sperling JW, Cofield RH, Itoi E, et al. The stabilizing mechanism of the Latarjet procedure: a cadaveric study. *J Bone Joint Surg Am.* 2013;95(15):1390–7.
  32. Gartsman GM, Waggenspack WN Jr, O'Connor DP, Elkousy HA, Edwards TB. Immediate and early complications of the open Latarjet procedure: a retrospective review of a large consecutive case series. *J Shoulder Elb Surg.* 2017;26(1):68–72.
  33. Griesser MJ, Harris JD, McCoy BW, Hussain WM, Jones MH, Bishop JY, et al. Complications and re-operations after Bristow-Latarjet shoulder stabilization: a systematic review. *J Shoulder Elb Surg.* 2013;22(2):286–92.
  34. Young AA, Maia R, Berhouet J, Walch G. Open Latarjet procedure for management of bone loss in anterior instability of the glenohumeral joint. *J Shoulder Elb Surg.* 2011;20(2 Suppl):S61–9.
  35. Hovelius L, Sandstrom B, Saebo M. One hundred eighteen Bristow-Latarjet repairs for recurrent anterior dislocation of the shoulder prospectively followed for fifteen years: study II—the evolution of dislocation arthropathy. *J Shoulder Elb Surg.* 2006;15(3):279–89.
  36. Yamamoto N, Itoi E, Abe H, Minagawa H, Seki N, Shimada Y, et al. Contact between the glenoid and the humeral head in abduction, external rotation, and horizontal extension: a new concept of glenoid track. *J Shoulder Elb Surg.* 2007;16(5):649–56.
  37. Di Giacomo G, Itoi E, Burkhart SS. Evolving concept of bipolar bone loss and the Hill-Sachs lesion: from “engaging/non-engaging” lesion to “on-track/off-track” lesion. *Arthroscopy.* 2014;30(1):90–8.



# Scapular Dyskinesia in Athletes

# 4

W. Ben Kibler and Aaron Sciascia

## Key Learning Points

- The scapula is a critical link in the kinetic chain and is an integral part of overhead arm function.
- Scapular dyskinesia can alter kinetic chain function, thus decreasing athletic performance.
- Scapular dyskinesia is an impairment that can be considered a cause or an effect of shoulder injury.
- Scapular dysfunction should be assessed as part of the clinical examination of shoulder injury.
- In addition to improving scapular stability, rehabilitation for scapular dyskinesia should be performed addressing multiple aspects of kinetic chain function including strengthening and flexibility of both upper and lower extremity segments.

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## 4.1 Introduction

Skillful athletic activities involving the shoulder require precise coordinated motions of the anatomic components of the shoulder

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for efficient performance. Primary among them are the movements of the scapula and rotations of the glenohumeral joint. Coupling of these motions is key to normal shoulder function. Alterations in these motions, either separately in each component or coupled in both components, can lead to inefficient shoulder function with decreased performance and/or increased injury risk.

The scapula exhibits motion in 3 planes and translations in 2 directions as part of normal scapulohumeral rhythm [1–3]. The motions are upward/downward rotation around an anterior/posterior axis perpendicular to the scapula, internal/external rotation around a vertical superior to inferior axis along the medial border, and anterior/posterior tilt around a horizontal medial to lateral axis along the scapular spine [2]. The translations are upward/downward along the thorax and medial to lateral around the curvature of the thorax. The scapula rarely moves in only one of the motions and translations when accomplishing most scapular roles. However, loss of control of specific motions seems to alter glenohumeral kinematics and function more than others. Loss of control of posterior tilting, allowing more anterior tilt, and loss of control of external rotation, allowing more internal rotation, appear to be most commonly associated with altered function or injury [4–8]. Normal scapular resting position and active motion are altered in overhead athletes due to the repetitive motions. Studies have demonstrated increased posterior tilt and upward rotation in these athletes [7, 9].

Overhead tasks are performed through the utilisation and integration of multiple body segments and muscles. Sequential activation of specific muscle groups resulting in the performance of a specific dynamic action is known as kinetic chain function [10]. The scapula plays a major role in sports participation performance as a central segment in the kinetic chain. During throwing and serving tasks, the scapula is the pivotal link between the larger centralised body segments that produce stability and generate force and the smaller localised segments of the arm that produce mobility and precision, and apply force to the ball or racquet.

Proper utilisation of the kinetic chain allows the multiple body segments to optimally contribute to the performance or execution of the specific task. In the tennis serve, a specific set of sequential actions have been described that begin proximally in the lower extremity and end distally in the segments of the upper extremity

[11, 12]. The most effective serve motion creates adequate knee flexion, trunk rotation, and core stability, which allows the scapula to fully retract for increased energy storage and transference [12]. Similar use of the kinetic chain is necessary for overhead throwing in baseball. The scapula is positioned between the trunk and the arm, and to maximise its potential while minimising injury risk requires that the kinetic chain links preceding the arm be utilised appropriately. To achieve optimal scapular control, an overhead thrower must control the trunk over the back leg, have the forearm pronated during cocking, the front leg and hips directed at the target, and hip/trunk move synchronously in rotation towards the target [12]. This will allow maximal scapular retraction to occur in cocking, resulting in the ability to fully horizontally abduct and externally rotate the shoulder, increasing the ability to develop maximal velocity. In both scenarios, the larger muscles and segments serve as the initiators and regulators of function. However, alteration of a particular segment in the kinetic chain can result in either altered performance or injury to a more distal segment.

Sports participation results in slight differences in side-to-side motion and in scapular resting position in overhead athletes [7, 9, 13, 14]. The differences are increased or decreased upward rotation, increased internal rotation, and/or variable changes in anterior/posterior tilt. Recent evidence has confirmed that some groups of throwing athletes have specific compensations in position but display the same direction of motions during arm motion [15]. These findings require that side-to-side evaluation be done to check for abnormal asymmetries, and that observed alterations be treated only if they are found in association with injury. However, if alterations are found with injury, they should be addressed since the altered scapular positions have been hypothesised to have implications for decreases in muscle function and in injury [1, 9, 16–24].

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## 4.2 Scapular Dyskinesis in Athletes

### 4.2.1 Definition of Scapular Dyskinesis

Most scapular related problems in throwing athletes can be traced to loss of control of normal resting scapular position and dynamic

scapular motion, resulting in alterations in the position or motion that produce a position and motion of excessive protraction. This position and motion, in the face of functional demands of the throwing or overhead motion, can create inefficiencies and deficits in the kinematics of the shoulder, which can decrease performance and increase injury risk.

Altered dynamic motion is termed scapular dyskinesis (dys—alteration of; kinesis—motion) [25]. It is characterised by medial or inferior medial scapular border prominence, early scapular elevation or shrugging upon arm elevation, and rapid downward rotation upon arm lowering [6]. The most salient clinical manifestation of protraction is asymmetric prominence of the medial scapular border during lowering of an elevated arm commonly called “winging” (Fig. 4.1).

The clinical presentation of scapular winging can have multiple causes include neurologically based scapular winging, scapular muscle detachment, snapping scapula, and kinetic chain or muscle inhibition based scapular dyskinesis. Dyskinesis is found to be present in 67–100% of patients with shoulder injuries [26–28]. However, dyskinesis is best considered a potential

**Fig. 4.1**  
Illustration of  
typical  
observation of  
scapular  
dyskinesis



impairment of optimum shoulder function, and if it is found in association with shoulder symptoms, it should be further evaluated.

Scapular dyskinesis is important as a component of the disabled throwing shoulder [4, 5]. It is associated with labral tears [4, 5, 29], internal impingement (the combination of partial rotator cuff injury and labral tears) [30, 31], and elbow injuries [32]. It is considered part of the shoulder at risk, and should be checked as part of the routine pre-participation evaluation. Common causative factors for shoulder pain in sports participation are the deficits in glenohumeral internal rotation (GIRD) or total range of motion. These range of motion alterations can result from capsular, muscular, and possible osseous alterations [33]. In addition to affecting shoulder joint function, they create scapular dyskinesis in the form of scapular protraction due to a wind-up effect as the arm, while continuing into forward flexion, internal rotation, and horizontal adduction in follow through, pulls the scapula into internal rotation and anterior tilt. Since optimised scapular function is a key factor in optimal sports participation, recognition of dyskinesis and restoration of scapular retraction capability should be a standard part of injury prevention strategies. Also, scapular dyskinesis resulting from fatigue was shown to be an important factor in producing errors of arm proprioception [34]. However, the exact relationship between scapular position and/or motion and injury is unclear. Abnormal scapular motion or scapular dyskinesis has been described as a non-specific response to a painful condition in the shoulder rather than a specific response to or a definite cause of specific glenohumeral pathology [25]. Various shoulder soft tissue pathologies including impingement (internal and external) [35], anterior capsular laxity [36], labral injury [4, 5], and rotator cuff weakness [12] have been found in association with scapular dyskinesis in overhead athletes complaining of shoulder pain. However, the confounding issue is that scapular asymmetries have been noted in overhead athletes that are asymptomatic as well as those injured. At this time, it is unknown if scapular dysfunction is a cause and/or an effect of shoulder injury in overhead athletes and is, therefore, most appropriately characterised as a physical impairment [37].

## 4.2.2 Causative Factors for Scapular Dyskinesis

Most scapular dyskinesia results from alteration in coupled muscle activation within the upper trapezius, lower trapezius, rhomboids, and serratus anterior. Neurogenic causes include injury to the long thoracic or spinal accessory nerves, which appear to be relatively rare in throwing athletes. More commonly, the alterations are due to inhibition of activation due to pain from glenohumeral joint injury [38], strength imbalance among the scapular stabilisers [39], fatigue of muscle activation [40], or change in activation pattern [17]. In virtually every case, the serratus anterior and lower trapezius have been shown to be weak, display less activation intensity, or to be late in activation timing, while the upper trapezius displays increased activation and abnormal activation timing [41]. This results in less posterior tilt, less external rotation, and less upward rotation motions, but increased elevation translation [21, 41]. These results have been found in throwers with impingement [42], instability [28], and labral tears [4, 5].

Bony causes in athletes relate to alteration in the strut stabilisation function of the clavicle. This would include malunited (shortened, angulated, or malrotated) or non-united fractures, high-grade (type V, some type III) acromioclavicular separations, or excessive (greater than 5 mm) distal clavicle excision.

## 4.2.3 Scapular Dyskinesia and Specific Shoulder Injuries

### 4.2.3.1 Labral Injury

Scapular dyskinesia has a high association with labral injury [43, 44]. The altered position and motion of internal rotation and anterior tilt changes glenohumeral alignment, placing increased tensile strain on the anterior ligaments [36], increases “peel-back” of the biceps/labral complex on the glenoid [4, 5], and creates pathological internal impingement [44]. These effects are magnified in the presence of GIRD, which creates increased protraction due to “wind-up” of the tight posterior structures in follow-through. The demonstration of dyskinesia in patients with suspected labral



injury provides a key component of rehabilitation protocols. Correction of the symptoms of pain found in the modified dynamic labral shear test [45] can be frequently demonstrated by the addition of manual scapular retraction [46]. This indicates the presence of dyskinesia as part of the pathophysiology and the need for scapular rehabilitation to improve scapular retraction, including mobilisation of tight anterior muscles and institution of the scapular stability series of strengthening exercises.

### **Impingement**

Impingement is frequently seen in throwing athletes. Most commonly in this group, impingement is secondary to other pathology such as instability, labral injury, or biceps pathology. Scapular dyskinesia is associated with impingement by altering scapular position at rest and upon dynamic motion. Scapular dyskinesia in impingement is characterised by loss of acromial upward rotation, excessive scapular internal rotation, and excessive scapular anterior tilt [22, 47]. These positions create scapular protraction, which decreases the subacromial space [21] and decreases demonstrated rotator cuff strength [20, 24].

Activation sequencing patterns and strength of the muscles that stabilise the scapula are altered in patients with impingement and scapular dyskinesia. Increased upper trapezius activity, imbalance of upper trapezius/lower trapezius activation so that the lower trapezius activates later than normal, and decreased serratus anterior activation have been reported in patients with impingement [21–23]. Increased upper trapezius activity is clinically observed as a shrug manoeuvre, resulting in a variation of the scapular dyskinesia pattern. This causes impingement due to lack of acromial elevation. Frequently, lower trapezius activation is inhibited or is delayed creating impingement due to loss of acromial elevation and posterior tilt. Serratus anterior activation has been shown to be decreased in patients with impingement, creating a lack of scapular external rotation and elevation with arm elevation [19].

The pectoralis minor has been shown to be shortened in length in patients with impingement. This tight muscle creates a position

of scapular protraction at rest and does not allow scapular posterior tilt or external rotation upon arm motion, predisposing patients to impingement symptoms [48].

#### **4.2.3.2 Rotator Cuff Injury**

The rotator cuff is frequently clinically involved in throwers with shoulder symptoms and these symptoms can be exacerbated by dyskinesia. The dyskinetic position that results in an internally rotated and anteriorly tilted glenoid increases the internal impingement on the posterior superior glenoid with arm external rotation and increases the torsional twisting of the rotator cuff, which may create the under-surface rotator cuff injuries seen in throwers [4, 5, 35]. In addition, positions of scapular protraction have been shown to be limiting to the development of maximal rotator cuff strength. Recent work in laboratory models of rotator cuff disease has shown that surgically induced scapular dyskinesia results in changes in cell morphology, gene expression, and tendon characteristics that are similar to those seen in rotator cuff tendinopathy [49].

#### **4.2.3.3 AC Joint Injuries**

AC joint injuries are rare in throwing athletes except American football quarterbacks, but they can create major functional deficits due to the disruption of the important AC linkage. Dyskinesia is found in a high percentage of patients with high grade AC symptoms [26]. AC separations lessen, and high-grade AC separations remove, the strut function of the clavicle on the scapula. Loss of the strut function allows the “third translation” of the scapula, allowing it to move interior and medial to the clavicle, changing the biomechanical screw axis of scapulohumeral rhythm, allowing excessive scapular internal rotation and protraction and decreased dynamic acromial elevation when the arm is elevated. Iatrogenic AC joint injury due to excessive distal clavicle resection and detachment of the AC ligaments shortens the bony strut and allows excessive scapular internal rotation due to excessive anterior/posterior motion at the AC joint. The protracted scapular position creates many of the dysfunctional problems associated with chronic AC separations, including impingement and decreased demonstrated rotator cuff strength. However, scap-

ular and shoulder dysfunction can also occur in type II injuries if the AC ligaments are torn. This creates an anterior/posterior AC joint laxity and can be associated with symptoms of pain, clicking, decreased arm elevation, and decreased shoulder function.

If dyskinesia is demonstrated on the clinical exam, then increased attention should be directed towards correcting the biomechanical abnormality rather than just placing the arm in a sling. Treatment should include, not only CC ligament reconstruction, but also AC ligament reconstruction to completely restore the screw axis mechanism.

#### **4.2.3.4 Clavicle Fractures**

Clavicle fractures may produce dyskinesia if the anatomy is not completely restored. The dyskinesia can be associated with alterations in shoulder function such as decreased strength and decreased arm motion in elevation [50]. Shortened mal-unions or non-unions decrease the length of the strut, and alter the scapular position towards internal rotation and anterior tilt. In addition to changes in length, changes in clavicle curvature or rotation will affect scapular position or motion. Angulated fractures result in functional shortening and loss of rotation. The distal fragment in midshaft fractures often internally rotates, decreasing the obligatory clavicle posterior rotation and scapular posterior tilt during arm elevation. Dyskinesia can be a clinical sign of potentially harmful alteration of clavicle anatomy, and can provide information to clarify indications for operative treatment in these fractures.

#### **4.2.3.5 Scapular Muscle Detachment**

Scapular muscle detachment is a relatively rare and poorly characterized injury, and the diagnosis is often delayed or missed [51]. The pathoanatomy appears to be detachment of the lower trapezius and rhomboids from the spine and medial border of the scapula. The scapula usually presents in a position of protraction and lateral translation. The majority of cases present after an acute traumatic tensile load such as seat-belt-restrained motor-vehicle accidents, catching or lifting a heavy object with the arm at full extension, or pulling against a heavy object. In athletes, it may occur after a fall, or from an acute tear in throwing. The present-

ing symptom cluster is very uniform with early post-traumatic onset of localised and intense pain along the medial scapular border. There is a weakness of the rhomboids and lower trapezius, with difficulty in retracting the scapula, with resulting major limitations of arm use away from the body in forward flexion or overhead positions. Increased upper trapezius activity and spasm, resulting from lack of lower trapezius activity, creates migraine-like headaches. Neck and shoulder joint symptoms may be present due to dyskinesia and will often become the focus of treatment, including surgery that may not properly address the underlying pathology.

Surgical exploration for repair of scapular muscle detachment is indicated after failure to improve symptoms following a specific protocol of scapular retraction and depression exercises [52, 53]. The operative technique has been previously described in detail as well as mid-term outcome results [51, 54].

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### **4.3 Evaluation of the Scapula in the Throwing Athlete**

The history is an important part of the evaluation. Specific questions should be asked regarding past or present trauma to the scapula, clavicle, or AC joint, chronic or acute spinal symptoms, recent or remote hip or leg injuries, or any surgical procedures. It is also important to establish if the patients have had physical therapy for any of these conditions, or for a scapular condition to document the exact extent of the therapy, and to document the results. Therapy that emphasises modalities, early open chain rotator cuff exercises with resistance, shoulder shrugs, and shoulder protraction exercises have not been found to be effective for scapular dyskinesia.

The goals of the scapular examination are to establish the presence or absence of dyskinesia, the effect on symptoms of corrective manoeuvres, and to investigate possible bony, joint derangement, or muscle strength/flexibility causes for the dyskinesia.

The scapular exam should largely be accomplished from the posterior aspect. The scapula should be exposed for complete

visualisation. The resting posture should be checked for side to side asymmetry and obvious inferior medial or medial border prominence. If there is difficulty with determining the bony landmarks of the inferior medial or superior medial angles, marking the superior and inferior medial borders may help ascertain the position.

Visual dynamic assessment schemes of classifying the presence of scapular dyskinesia during shoulder motion have been developed in an attempt to resolve the issues with linear or static measures [8, 55, 56]. These methods are considered more functional and more inclusive with the ability to judge scapular movement in 3-dimensional patterns. Kibler et al. [55] were the first to describe a visually based system for rating scapular dysfunction that defined 3 different types of motion abnormality and one normal type. Reliability values for this system were too low to support clinical use and the test was subsequently refined [8, 56].

The scapular dyskinesia test [56] is a visual-based test for scapular dyskinesia that involves a patient performing weighted shoulder flexion and abduction movements while scapular motion is visually observed. This test consists of characterising scapular dyskinesia as absent or present and each side is rated separately. Good inter-rater reliability of this test (75–82% agreement; weighted kappa = 0.48–0.61) was achieved after brief standardised online training. Concurrent validity was demonstrated in a large group of overhead athletes, finding those judged as demonstrating abnormal motion using this system also demonstrated decreased scapular upward rotation, less clavicular elevation and less clavicular retraction when measured with 3-dimensional motion tracking [57]. These results support the assertion that shoulders visually judged as having dyskinesia utilising this system, demonstrate distinct alterations in 3-dimensional scapular motion, particularly during flexion.

Another dynamic test developed by Uhl et al. [8] used essentially the same criteria as the scapular dyskinesia test (winging or dysrhythmia) to classify an abnormality in scapular motion into the “yes” classification, and normal movement was classified as “no”. They studied both symptomatic patients with various soft tissue pathologies as well as an asymptomatic group. The “yes/

no” test was found to have superior inter-rater reliability (79% agreement; kappa = 0.41), and demonstrated better specificity and sensitivity values when using asymmetry found with 3-dimensional testing as a gold standard [8]. An important finding in this study was a higher frequency of multiple-plane dyskinesia during shoulder flexion in patients (54%) compared with asymptomatic subjects (14%), while no differences between groups were detected during scapular plane elevation. It appears that the optimum position for evaluating scapular dyskinesia dynamically is in forward flexion.

The scapular assistance test (SAT) and scapular retraction test (SRT) are corrective manoeuvres that can alter the injury symptoms and provide information about the role of scapular dyskinesia in the total picture of dysfunction that accompanies shoulder injury and needs to be restored. The SAT helps evaluate scapular contributions to impingement and rotator cuff strength, and the SRT evaluates contributions to rotator cuff strength and labral symptoms. In the SAT, the examiner applies gentle pressure to assist scapular upward rotation and posterior tilt as the patient elevates the arm (Fig. 4.2) [46].

This test has shown “acceptable” inter-rater reliability [58]. A positive result occurs when the painful arc of impingement symptoms is relieved and the arc of motion is increased. In the SRT, the

**Fig. 4.2**  
The scapular assistance test



examiner first grades the supraspinatus muscle strength following standard manual muscle testing procedures [46]. The examiner then places and manually stabilises the scapula in a retracted position (Fig. 4.3). A positive test occurs when the demonstrated supraspina-



**Fig. 4.3** The scapular retraction test

tus strength is increased or the symptoms of internal impingement in the labral injury are relieved in the retracted position [20].

Although these tests are not capable of diagnosing a specific form of shoulder pathology, a positive SAT or SRT shows that scapular dyskinesis is directly involved in producing the symptoms and indicates the need for inclusion of early scapular rehabilitation exercises to improve scapular control.

Coracoid based inflexibility can be assessed by palpation of the pectoralis minor and the short head of the biceps brachii at their insertion on the coracoid tip. The muscles will usually be tender to palpation, even if they are not symptomatic in use, can be traced to their insertions on the ribs as taut bands, and will create symptoms of soreness and stiffness when the scapulae are manually maximally retracted and the arm is slightly abducted to approximately 40–50°.

A major portion of the scapular exam is the evaluation of the proximal kinetic chain and distal glenohumeral joint structures that affect scapular position and motion. Kinetic chain screening can be accomplished by the one leg stability series—a combination of a standing balance test that assesses static control, and a single leg squat test that assesses dynamic control of the body over the planted leg [59]. In the standing balance test, the patient is asked to place their hands over their chest and stand on one leg with no other verbal cue. Deficits in balance and stability such as a Trendelenburg posture or internally or externally rotating the weight bearing limb indicates inability to control the posture and has been found to correlate with proximal core weakness especially in the gluteus medius [60–62]. The single leg squat is the next progressive evaluation. Assuming the same starting point as the standing balance test, the patient is asked to do repetitive partial half squats going down and returning to the standing position with no other verbal cues. Similar deviations in the quality of the movement are assessed as in the standing balance test. A Trendelenburg posture, which may not be noted on standing balance, may be brought out with a single leg squat. The patient may also use their arms for balance or may go into an exaggerated flexed or rotated posture—“corkscrewing”—in order to put the gluteal or short rotator muscles on greater tension to compensate for muscular weakness.



Standard glenohumeral exam techniques should be employed to evaluate for internal derangement and AC joint instability. Special attention should be paid to the examination for GIRD and the evaluation of labral injuries, both of which are associated with dyskinesia. To obtain accurate glenohumeral internal rotation measurements, the patient should be positioned supine on a flat level surface. A second examiner should be positioned behind the athlete in order to properly stabilise the scapula by applying a posteriorly directed force to the coracoid and humeral head to ensure that scapular movement does not occur [63, 64]. The humerus is supported on the surface with the elbow placed at 90° and the arm on a bolster in the plane of the scapula. A measurement is obtained using a standard bubble goniometer where the fulcrum is set at the olecranon process of the elbow, the stationary arm perpendicular to the table as documented by the bubble on the goniometer, and the moving arm in line with the styloid process of the ulna. The clinician passively moves the arm into internal and external rotation. Rotation is taken to “tightness”, a point where no more glenohumeral motion would occur unless the scapula would move or the examiner applies rotational pressure. This measurement should be taken bilaterally, and side-to-side differences are calculated. Side-to-side differences in internal rotation greater than 20° are considered clinical GIRD.

To evaluate labral injuries using the modified dynamic labral shear (M-DLS) test, position the patient standing [45]. Flex the elbow of the involved arm to 90°, abduct the humerus in the scapular plane to above 120°, and externally rotate to tightness. Gently guide the arm to maximal horizontal abduction. Apply a shear load to the joint by maintaining external rotation and horizontal abduction and lowering the arm from 120° to 60° abduction (Fig. 4.4). A positive test is indicated by reproduction of the pain and/or a painful click or catch in the joint line along the posterior joint line between 120° and 90° abduction.

Be cautious when placing the arm into maximal horizontal abduction as excessive overpressure and positioning can result in a false positive test and/or create pain throughout the entire motion. This test has been shown to have high clinical utility, with sensitivity = 0.72, specificity = 0.98, positive likelihood ratio = 31.57, and negative likelihood ratio = 0.2945.



**Fig. 4.4** The modified dynamic labral shear for evaluating the presence of superior labral injury

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#### 4.4 Specific Scapular Rehabilitation Exercises

Treatment of scapular dyskinesis must start with optimised anatomy. In many cases, the muscular anatomy is not disrupted so rehabilitation may begin early. However, local problems such as nerve injury or scapular stabiliser muscle detachment must be addressed with repair or muscle transfer, while bony and tissue derangement issues such as AC joint or clavicle injury, labral injury, rotator cuff disease, or glenohumeral instability must be repaired. Rehabilitation can then proceed on the optimised anatomy. A very effective protocol can be based around the protocol suggested by Ellenbecker and Cools [65]. It organises scapular rehabilitation into flexibility and strength protocols, and further orients strength into activation and strength areas.

Kinetic chain exercises for trunk and hip start from and end at the “ideal position” of hip extension/trunk extension. They include trunk/hip flexion/extension, rotation, and diagonal motions [53, 66]. Progressions include step up/down and increased weights. They may be started preoperatively when deficits have been identified and may be done while the shoulder is protected.

Specific areas to be addressed for flexibility include the anterior coracoid (pectoralis minor and biceps short head) and shoulder rotation. Tightness in these areas increases scapular protraction. Exercises include the open book and corner stretch for coracoid muscles (Fig. 4.5) and sleeper and cross body stretch for shoulder rotation (Fig. 4.6).

Peri-scapular strengthening should emphasise achieving a position of scapular retraction, as this is the most effective position to maximise scapular roles. Scapular retraction exercises may be done in a standing position to simulate normal activation sequences and allow kinetic chain sequencing. Scapular pinch and trunk extension/scapular retraction exercises may be started early in rehabilitation even when the shoulder is being protected, since there is minimal tensile load or shear on the glenohumeral joint in these exercise.

Several specific exercises have been shown to be very effective to activate the key scapular stabilisers—the lower trapezius and serratus anterior and minimise upper trapezius activation. They are the low row (Fig. 4.7), inferior glide (isometric exercises) (Fig. 4.8), fencing (Fig. 4.9), lawnmower (Fig. 4.10), and robbery (Fig. 4.11). These are collectively termed the scapular stability series.

**Fig. 4.5**  
Corner stretch  
for improving  
flexibility of  
anterior  
shoulder soft  
tissue structures



**Fig. 4.6** Cross body stretch for improving flexibility of posterior shoulder soft tissue structures



Once scapular control is achieved, integrated scapula/rotator cuff exercises such as punches and shoulder dumps that stimulate rotator cuff activation off a stabilised scapula are added. They may be done in various planes of abduction and flexion, with different amounts or types of resistance (beginning with lighter resistance elastic bands or tubing and progressing to free weights of no more than 3–5 lbs), and may be modified to be sport specific. Most activities, whether they are sports-related or normal daily movements, occur in the transverse plane. Therefore, the transverse plane should be exploited throughout the rehabilitation continuum, which is best accomplished with the patient standing. Exercises and manoeuvres that require supine or prone positions should be limited, as they do not allow for adequate utilisation of the kinetic chain. The protocol should progress to more unilateral planes as normal scapulohumeral kinematics are restored.



**Fig. 4.7** Low row exercise, which helps facilitate scapular retraction

Once strength and stabilisation has been achieved through controlled supervised rehabilitation, three areas of focus should be implemented to ensure an effective transition from controlled rehabilitation to advanced function: lower extremity muscle power and endurance, integrated sports-specific exercise, and upper extremity power and endurance [53]. High-repetition exercises designed to increase lower extremity muscle endurance should be employed first. For example, pitching is a task requiring



**Fig. 4.8** Active inferior glide exercise utilises muscular co-contraction for humeral head depression



**Fig. 4.9** Fencing manoeuvre utilises multiple kinetic chain segments to enhance proper muscle scapular muscle activation



**Fig. 4.10** Lawnmower exercise facilitates scapular retraction through trunk rotation

activation of multiple segments repetitively, adequate muscle endurance of all involved muscle groups is necessary for optimal performance. Focus on the gastrocnemius/soleus, quadriceps, hamstrings, and hip abductor muscle groups would be recommended. The next component would be the utilisation of integrated sports-specific exercise that encourages use of the improved lower extremity muscle strength and endurance to help facilitate upper extremity muscle activation. This is accomplished through synchronous single-leg and transverse-plane exercises, which aid in improving proprioception as well as muscle education. The final area, upper extremity power and endurance, is addressed via high-repetition, long-lever exercises performed in standing and prone positions.



**Fig. 4.11** Robbery exercise assists in achieving scapular retraction and depression through trunk extension and short lever arm motion

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## 4.5 Summary

The scapula plays multiple key roles in normal scapulohumeral rhythm and shoulder function. Alterations of scapular resting position and dynamic motion, collectively termed scapular dyskinesia, are associated frequently with many shoulder injuries in throwing athletes, including rotator cuff injury, impingement, labral injury, and clavicle and AC joint injuries. The clinical exam for presence or absence of scapular dyskinesia, utilising observation of medial scapular border prominence, has good clinical utility. If scapular dyskinesia is present, corrective manoeuvres may be used to determine the effect of dyskinesia on shoulder symptoms. Further investigations for kinetic chain muscular, neurological, glenohumeral joint, or bony causes of dyskinesia can then guide treatment of the dyskinesia as part of the treatment of the



entire shoulder problem. Scapular control in a position of retraction, external rotation, and posterior tilt should be a key detriment of return to play status.

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## Q&A

- (1) What are the 3 planes of normal scapular motion?  
Upward/downward rotation, internal/external rotation, anterior/posterior tilt
- (2) What is the position and motion that is most detrimental to normal scapular and shoulder function and what is the clinical finding?  
Excessive protraction, medial border prominence
- (3) How does scapular dyskinesia affect the pathophysiology of impingement and rotator cuff disease?  
Loss of acromial upward rotation and increased anterior tilt
- (4) What are the 2 clinical examination corrective manoeuvres that can provide information about the role of scapular dyskinesia in clinical dysfunction?  
Scapular assistance test (SAT) and scapular retraction test (SRT)
- (5) In rehabilitation of scapular dyskinesia, what should be the position of highest emphasis in order to maximize scapular roles in function?  
Scapular retraction

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## References

1. Kibler WB, Sciascia A, Wilkes T. Scapular dyskinesia and its relation to shoulder injury. *J Am Acad Orthop Surg.* 2012;20(6):364–72.
2. Ludewig PM, Phadke V, Braman JP, Hassett DR, Cieminski CJ, LaPrade RF. Motion of the shoulder complex during multiplanar humeral elevation. *J Bone Joint Surg.* 2009;91A(2):378–89.
3. McClure PW, Michener LA, Sennett BJ, Karduna AR. Direct 3-dimensional measurement of scapular kinematics during dynamic movements in vivo. *J Shoulder Elb Surg.* 2001;10:269–77.

4. Burkhart SS, Morgan CD, Kibler WB. The disabled throwing shoulder: spectrum of pathology part I: pathoanatomy and biomechanics. *Arthroscopy*. 2003;19(4):404–20.
5. Kibler WB, Kuhn JE, Wilk KE, Sciascia AD, Moore SD, Laudner KG, et al. The disabled throwing shoulder - spectrum of pathology: 10 year update. *Arthroscopy*. 2013;29(1):141–61.
6. Kibler WB, Ludewig PM, McClure PW, Uhl TL, Sciascia AD. Scapula summit 2009. *J Orthop Sports Phys Ther*. 2009;39(11):A1–A13.
7. Myers JB, Laudner KG, Pasquale MR, Bradley JP, Lephart SM. Scapular position and orientation in throwing athletes. *Am J Sports Med*. 2005;33(2):263–71.
8. Uhl TL, Kibler WB, Gecewich B, Tripp BL. Evaluation of clinical assessment methods for scapular dyskinesis. *Arthroscopy*. 2009;25(11):1240–8.
9. Laudner KG, Myers JB, Pasquale MR, Bradley JP, Lephart SM. Scapular dysfunction in throwers with pathologic internal impingement. *J Orthop Sports Phys Ther*. 2006;36(7):485–94.
10. Sciascia AD, Thigpen CA, Namdari S, Baldwin K. Kinetic chain abnormalities in the athletic shoulder. *Sports Med Arthrosc Rev*. 2012;20(1):16–21.
11. Kibler WB, Wilkes T, Sciascia A. Mechanics and pathomechanics in the overhead athlete. *Clin Sports Med*. 2013;32(4):637–51.
12. Lintner D, Noonan TJ, Kibler WB. Injury patterns and biomechanics of the athlete's shoulder. *Clin Sports Med*. 2008;27(4):527–52.
13. Laudner KG, Stanek JM, Meister K. Differences in scapular upward rotation between baseball pitchers and position players. *Am J Sports Med*. 2007;35:2091–5.
14. Oyama S, Myers JB, Wassinger CA, Ricci RD, Lephart SM. Asymmetric resting scapular posture in healthy overhead athletes. *J Athl Train*. 2008;43(6):565–70.
15. Seitz AL, Reinold M, Schneider RA, Gill TJ, Thigpen CA. No effect of scapular position on 3-dimensional scapular in the throwing shoulder of healthy professional pitchers. *J Sport Rehabil*. 2012;21(2):186–93.
16. Borich MR, Bright JM, Lorello DJ, Cieminski CJ, Buisman T, Ludewig PM. Scapular angular positioning at end range internal rotation in cases of glenohumeral internal rotation deficit. *J Orthop Sports Phys Ther*. 2006;36:926–34.
17. Cools AM, Witvrouw EE, DeClercq GA, Danneels LA, Cambier DC. Scapular muscle recruitment pattern: trapezius muscle latency with and without impingement symptoms. *Am J Sports Med*. 2003;31:542–9.
18. Cools A, Johansson FR, Cambier DC, Velde AV, Palmans T, Witvrouw EE. Descriptive profile of scapulothoracic position, strength, and flexibility variables in adolescent elite tennis players. *Br J Sports Med*. 2010;44:678–84.
19. Ebaugh DD, McClure PW, Karduna AR. Effects of shoulder muscle fatigue caused by repetitive overhead activities on scapulothoracic and glenohumeral kinematics. *J Electromyogr Kinesiol*. 2006;16:224–35.

20. Kibler WB, Sciascia AD, Dome DC. Evaluation of apparent and absolute supraspinatus strength in patients with shoulder injury using the scapular retraction test. *Am J Sports Med.* 2006;34(10):1643–7.
21. Ludewig PM, Reynolds JF. The association of scapular kinematics and glenohumeral joint pathologies. *J Orthop Sports Phys Ther.* 2009;39(2):90–104.
22. Ludewig PM, Cook TM. Alterations in shoulder kinematics and associated muscle activity in people with symptoms of shoulder impingement. *Phys Ther.* 2000;80(3):276–91.
23. Smith J, Kotajarvi BR, Padgett DJ, Eischen JJ. Effect of scapular protraction and retraction on isometric shoulder elevation strength. *Arch Phys Med Rehabil.* 2002;83:367–70.
24. Tate AR, McClure P, Kareha S, Irwin D. Effect of the scapula reposition test on shoulder impingement symptoms and elevation strength in overhead athletes. *J Orthop Sports Phys Ther.* 2008;38(1):4–11.
25. Kibler WB, Sciascia AD. Current concepts: scapular dyskinesia. *Br J Sports Med.* 2010;44(5):300–5.
26. Gumina S, Carbone S, Postacchini F. Scapular dyskinesia and SICK scapula syndrome in patients with chronic type III acromioclavicular dislocation. *Arthroscopy.* 2009;25(1):40–5.
27. Paletta GA, Warner JJP, Warren RF, Deutsch A, Altchek DW. Shoulder kinematics with two-plane x-ray evaluation in patients with anterior instability or rotator cuff tears. *J Shoulder Elb Surg.* 1997;6:516–27.
28. Warner JJP, Micheli LJ, Arslanian LE, Kennedy J, Kennedy R. Scapulothoracic motion in normal shoulders and shoulders with glenohumeral instability and impingement syndrome. *Clin Orthop Relat Res.* 1992;285(191):199.
29. Burkhart SS, Morgan CD, Kibler WB. The disabled throwing shoulder: spectrum of pathology part III: the SICK scapula, scapular dyskinesia, the kinetic chain, and rehabilitation. *Arthroscopy.* 2003;19(6):641–61.
30. Kibler WB, Dome DC. Internal impingement: concurrent superior labral and rotator cuff injuries. *Sports Med Arthrosc Rev.* 2012;20(1):30–3.
31. Mihata T, Jun BJ, Bui CN, Hwang J, McGarry MH, Kinoshita M, et al. Effect of scapular orientation on shoulder internal impingement in a cadaveric model of the cocking phase of throwing. *J Bone Joint Surg.* 2012;94(17):1576–83.
32. Dines JS, Frank JB, Akerman M, Yocum LA. Glenohumeral internal rotation deficits in baseball players with ulnar collateral ligament insufficiency. *Am J Sports Med.* 2009;37(3):566–70.
33. Kibler WB, Sciascia AD, Thomas SJ. Glenohumeral internal rotation deficit: pathogenesis and response to acute throwing. *Sports Med Arthrosc Rev.* 2012;20(1):34–8.
34. Tripp B, Uhl TL, Mattacola CG, Srinivasan C, Shapiro R. Functional multijoint position reproduction acuity in overhead athletes. *J Athl Train.* 2006;41(2):146–53.

35. Mihata T, McGarry MH, Kinoshita M, Lee TQ. Excessive glenohumeral horizontal abduction as occurs during the late cocking phase of the throwing motion can be critical for internal impingement. *Am J Sports Med.* 2010;38(2):369–82.
36. Weiser WM, Lee TQ, McQuade KJ. Effects of simulated scapular protraction on anterior glenohumeral stability. *Am J Sports Med.* 1999;27:801–5.
37. Kibler WB, Ludewig PM, McClure PW, Michener LA, Bak K, Sciascia AD. Clinical implications of scapular dyskinesis in shoulder injury: the 2013 consensus statement from the “scapula summit”. *Br J Sports Med.* 2013;47:877–85.
38. Falla D, Farina D, Graven-Nielsen T. Experimental muscle pain results in reorganization of coordination among trapezius muscle subdivisions during repetitive shoulder flexion. *Exp Brain Res.* 2007;178:385–93.
39. Cools AM, Witvrouw EE, Mahieu NN, Danneels LA. Isokinetic scapular muscle performance in overhead athletes with and without impingement symptoms. *J Athl Train.* 2005;40(2):104–10.
40. Tsai NT, McClure P, Karduna AR. Effects of muscle fatigue on 3-dimensional scapular kinematics. *Arch Phys Med Rehabil.* 2003;84:1000–5.
41. McQuade KJ, Dawson JD, Smidt GL. Scapulothoracic muscle fatigue associated with alterations in scapulohumeral rhythm kinematics during maximum resistive shoulder elevation. *J Orthop Sports Phys Ther.* 1998;28(2):74–80.
42. Cools AM, Witvrouw EE, DeClercq GA, Vanderstraeten GG, Cambier DC. Evaluation of isokinetic force production and associated muscle activity in the scapular rotators during a protraction-retraction movement in overhead athletes with impingement symptoms. *Br J Sports Med.* 2004;38:64–8.
43. Burkhart SS, Morgan CD, Kibler WB. Shoulder injuries in overhead athletes, the “dead arm” revisited. *Clin Sports Med.* 2000;19(1):125–58.
44. Myers JB, Laudner KG, Pasquale MR, Bradley JP, Lephart SM. Glenohumeral range of motion deficits and posterior shoulder tightness in throwers with pathologic internal impingement. *Am J Sports Med.* 2006;34:385–91.
45. Kibler WB, Sciascia AD, Dome DC, Hester PW, Jacobs C. Clinical utility of new and traditional exam tests for biceps and superior glenoid labral injuries. *Am J Sports Med.* 2009;37(9):1840–7.
46. Kibler WB. The role of the scapula in athletic function. *Am J Sports Med.* 1998;26:325–37.
47. Kebaetse M, McClure PW, Pratt N. Thoracic position effect on shoulder range of motion, strength, and three-dimensional scapular kinematics. *Arch Phys Med Rehabil.* 1999;80:945–50.
48. Borstad JD, Ludewig PM. The effect of long versus short pectoralis minor resting length on scapular kinematics in healthy individuals. *J Orthop Sports Phys Ther.* 2005;35(4):227–38.
49. Reuther KE, Thomas SJ, Tucker JJ, Yannascoli SM, Caro AC, Vafa RP, et al. Scapular dyskinesis is detrimental to shoulder tendon properties and joint mechanics in a rat model. *J Orthop Res.* 2014;32(11):1436–43.

50. McKee MD, Pedersen EM, Jones C, Stephen DJG, Kreder HJ, Schemitsch EH, et al. Deficits following nonoperative treatment of displaced mid-shaft clavicular fractures. *J Bone Joint Surg.* 2006;88:35–40.
51. Kibler WB, Sciascia A, Uhl T. Medial scapular muscle detachment: clinical presentation and surgical treatment. *J Shoulder Elb Surg.* 2014;23(1):58–67.
52. Kibler WB, McMullen J, Uhl TL. Shoulder rehabilitation strategies, guidelines, and practice. *Oper Tech Sports Med.* 2000;8(4):258–67.
53. Sciascia A, Cromwell R. Kinetic chain rehabilitation: A theoretical framework. *Rehabil Res Pract.* 2012;2012:1–9.
54. Kibler WB. Scapular surgery I-IV. In: Reider B, Terry MA, Provencher MT, editors. *Sports medicine surgery.* Philadelphia: Elsevier Saunders; 2010. p. 237–67.
55. Kibler WB, Uhl TL, Maddux JWQ, Brooks PV, Zeller B, McMullen J. Qualitative clinical evaluation of scapular dysfunction: a reliability study. *J Shoulder Elb Surg.* 2002;11:550–6.
56. McClure PW, Tate AR, Kareha S, Irwin D, Zlupko E. A clinical method for identifying scapular dyskinesia: part 1: reliability. *J Athl Train.* 2009;44(2):160–4.
57. Tate AR, McClure PW, Kareha S, Irwin D, Barbe MF. A clinical method for identifying scapular dyskinesia: part 2: validity. *J Athl Train.* 2009;44(2):165–73.
58. Rabin A, Irrgang JJ, Fitzgerald GK, Eubanks A. The intertester reliability of the scapular assistance test. *J Orthop Sports Phys Ther.* 2006;36(9):653–60.
59. Kibler WB, Press J, Sciascia AD. The role of core stability in athletic function. *Sports Med.* 2006;36(3):189–98.
60. Hardcastle P, Nade S. The significance of the trendelenburg test. *J Bone Joint Surg.* 1985;67(5):741–6.
61. Radwan A, Francis J, Green A, Kahl E, Maciurzynski D, Quartulli A, et al. Is there a relation between shoulder dysfunction and core instability? *Int J Sports Phys Ther.* 2014;9(1):8–13.
62. Reeser JC, Joy EA, Porucznik CA, Berg RL, Colliver EB, Willick SE. Risk factors for volleyball-related shoulder pain and dysfunction. *Phys Med Rehabil.* 2010;2(1):27–35.
63. Kibler WB, Sciascia AD, Moore SD. An acute throwing episode decreases shoulder internal rotation. *Clin Orthop Relat Res.* 2012;470:1545–51.
64. Wilk KE, Reinhold MM, Macrina LC, Porterfield R, Devine KM, Suarez K, et al. Glenohumeral internal rotation measurements differ depending on stabilization techniques. *Sports Health.* 2009;1(2):131–6.
65. Ellenbecker TS, Cools A. Rehabilitation of shoulder impingement syndrome and rotator cuff injuries: an evidence-based review. *Br J Sports Med.* 2010;44:319–27.
66. McMullen J, Uhl TL. A kinetic chain approach for shoulder rehabilitation. *J Athl Train.* 2000;35(3):329–37.



# Rotator Cuff Disorders in Athletes

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## Key Learning Points

- Rotator cuff muscles stabilise the glenohumeral joint and initiate glenohumeral motion in a way that allows deltoid and pectoralis muscles to be most effective.
- Rotator cuff injuries occur in athletes and must be appreciated to help these individuals return to optimum function.

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- Glenohumeral internal rotation deficit is a common finding in throwers and results from tightness of the posterior IGHL complex and posteroinferior capsule that leads to a posterosuperior shift of the point of glenohumeral articulation and centre of rotation.
- Most partial thickness rotator cuff tears can and should be managed nonoperatively. When surgical treatment is needed, debridement is often the preferred treatment, particularly in throwing athletes. Full-thickness rotator cuff tears usually require surgical repair.

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## 5.1 Biomechanics of the Rotator Cuff

The muscles of the rotator cuff play a crucial role in shoulder motion. Coordinated activities of the four muscles serve to maintain position of the humeral head in the glenoid cavity when the deltoid contracts. With the initiation of voluntary movement, electromyographic activity of the deltoid and pectoralis major muscle occurs after the activation of the rotator cuff muscles, supporting the notion that the rotator cuff muscles actively prepare the glenohumeral joint for movement [1]. Deltoid contraction leading to abduction generates shear forces that the rotator cuff muscles counteract. Resting tension of the rotator cuff is also an important component of glenohumeral stability.

In order for the rotator cuff to stabilise the glenohumeral joint, there must be a balance of forces between the coronal and axial plane as described by Burkhart et al. [2]. The native anatomic position of the four rotator cuff tendons allows for these two force planes to compress the humeral head within the glenoid fossa. The coronal force plane is between the deltoid and the inferior rotator cuff whereas the axial plane exists between the subscapularis anteriorly and the infraspinatus and teres minor posteriorly. Rotator cuff tears can disrupt these force couples, resulting in the inability to maintain the fulcrum for motion at the glenohumeral joint leading to severe limitations in active range of motion.

Tears of the rotator cuff require greater forces to be exerted by both the deltoid as well as the muscles of the rotator cuff itself to achieve stable abduction [1]. Forces required increase

with tear size and can contribute to the anterior or posterior extension of a tear. The part of the tendon that is torn cannot participate in load sharing of the exerted forces, therefore, increasing the tensile load on the remaining fibres. This can readily lead to tear propagation. Large retracted tears have been implicated to cause suprascapular nerve compression and may contribute to the progression of atrophy and fatty infiltration of the supraspinatus. Repair of these tears may release tension on the suprascapular nerve allowing for recovery of the nerve and improvement in function [3, 4].

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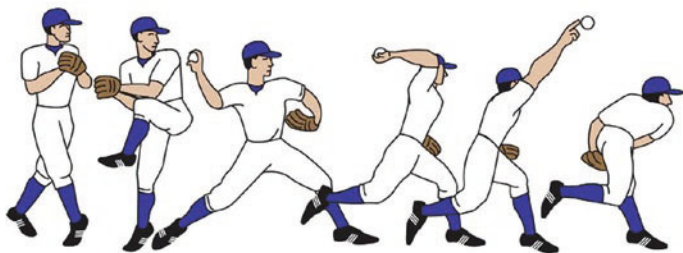
## 5.2 Aetiology and Relevant Pathology

A common cause of rotator cuff tears as well as other shoulder pathology in athletes is impingement. Impingement of the shoulder can be classified into two categories, external and internal impingement. External impingement is a relatively uncommon condition in young athletes. It is synonymous with subacromial impingement and is a result of narrowing of the subacromial space. Internal impingement often occurs in young athletes with overhead activity, especially in the dominant arm of throwing athletes (Javelin, Tennis, Volleyball, Baseball, American Football, Rugby) [5, 6]. However, internal impingement can also occur and be symptomatic in patients who do not participate in sports.

Often referred to as Thrower's shoulder, internal impingement occurs during the cocking phase of throwing (Fig. 5.1). Cocking consists of a combination of abduction, external rotation, and extension, leading to compression and impingement of the articular side of the rotator cuff between the humeral head and the posterior superior labrum [6]. These biomechanics are believed to serve as a physiologic restraint to prevent excessive external rotation, however, with repetitive overhead activity it may result in the development of pathology.

Posterior superior glenoid impingement by repetitive overhead activity can lead to rotator cuff injury in athletes as the posterosuperior labrum and rotator cuff can be compressed between the greater tuberosity and the glenoid rim (Fig. 5.2). Such glenoid





**Fig. 5.1** The six phases of the throwing motion. Phase 1 is the wind-up phase. Phase 2 is the early cocking phase, ending with planting of the striding foot. Phase 3 is the late cocking phase, in which the arm reaches maximum external rotation. In Phase 4, the ball is accelerated until Phase 5 starts with release of the ball and deceleration of the arm. Phase 6, the follow-through, rebalances the body until the motion stops (from Braun et al. [7])



**Fig. 5.2** Illustration of the pathomechanism behind the development of symptomatic internal impingement. Jobe and Walch et al. postulated that the posterosuperior cuff and labrum can become entrapped between the greater tuberosity and the posterosuperior glenoid resulting in partial-thickness articular-sided rotator cuff tears and posterosuperior labral tearing. Arrows indicate the resulting pathologic lesions associated with symptomatic internal impingement (from Spiegl et al. [8])

impingement may injure the superior labrum and superior glenoid bone, rotator cuff tendons, the greater tuberosity, the inferior glenohumeral ligament (IGHL) [7, 9, 10] and inferior labrum, or any combination of these [11] “Kissing lesions” of both the rotator cuff and the labrum are not uncommon.

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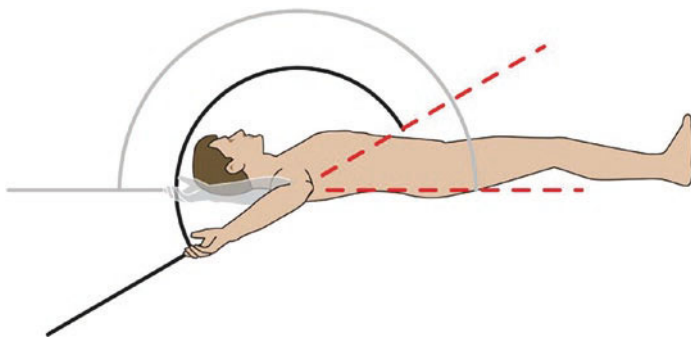
## 5.3 Diagnosis

### 5.3.1 Physical Examination

Patients usually present with pain in overhead activities and throwing. It is common for throwing athletes to present with shoulder pain without a specific acute injury. Clinical examination can show localised posterior shoulder pain on full external rotation and 90° abduction, positive impingement signs and signs of rotator cuff tears [6]. In the case of rotator cuff tears, weakness especially during abduction and pain with resistance are very common findings. Physical examination may also reveal anterior glenohumeral laxity without true instability. The Jobe relocation test can also be used to determine if a patient has symptomatic internal impingement [11].

Several physical examination manoeuvres can be used to recreate shoulder pain by compacting the muscles of the rotator cuff between the bony structures of the humeral head and the acromion or coracoid. These include Neer’s impingement sign and Hawkins’ impingement sign. However, a positive sign of pain may be due to other causes such as acromioclavicular or biceps pathology. If the labrum has sustained relevant damage, glenohumeral instability may occur. Sometimes a painful clicking or clunking can be provoked on examination.

Posterior capsular tightness as detected by glenohumeral internal rotational deficit (GIRD) may also be present. GIRD has been suggested by Burkhart et al. in 2003 as a clinical symptom and starting point of pathological changes of the shoulder [9]. The posterior IGHL complex and posteroinferior capsule undergo scarring, provoking a posterosuperior shift of the point of glenohumeral articulation and centre of rotation [9] (Fig. 5.3). With



**Fig. 5.3** The arc of motion of the throwing shoulder is shifted posteriorly, with increased external rotation and decreased internal rotation of the abducted shoulder (from Braun et al. [7])

chronic GIRD, it is not uncommon for overhead athletes to get articular sided rotator cuff pathology.

The active compression test, which is performed with the arm in  $90^\circ$  of forward flexion, starting in external rotation without pain and causing pain with internal rotation of the arm, is relatively specific in diagnosing labral tears [12]. Kibler's test, as well as many other physical exam manoeuvres can also be used to help in establishing a diagnosis.

### 5.3.2 Imaging

Ultrasound can detect rotator cuff tears, but strongly depends on the skills of the investigator and may miss subtle partial thickness tears. Magnetic resonance imaging (MRI) or MR arthrograms with intraarticular injection of contrast agent are often used to establish a diagnosis. Partial thickness rotator cuff tears, full thickness rotator cuff tears, SLAP (superior labrum anterior to posterior) lesions and lesions of the posterosuperior labrum can be detected by MR-arthrogram [6].

Standard MRI with the conventional 3 sequences (coronal, sagittal, and axial) is performed with the arm in adduction. Full thick-

ness rotator cuff tears can readily be detected. In this position, subtle labral tears as well as subtle partial thickness rotator cuff tears lying apposed to the intact tendon fibres may be missed [13].

To detect such partial thickness flap tears, the ABER view (Abduction External Rotation) for MRI of the shoulder has been introduced. This position tensions the anteroinferior glenohumeral ligament and labrum, while releasing tension on the cuff tendons compared with the normal coronal view with the arm in adduction.

Most conventional bore-style MRI scanners do not allow for the shoulder to be placed in the clinical position of apprehension, which is 90° of abduction and 90° of external rotation. This necessitates a modified position with the arm abducted and the hand tucked underneath the patient's head. Both the execution and the interpretation of ABER views can be challenging for technicians and physicians unfamiliar with its use [13].

While MRI is very useful in evaluating shoulder pathology, it can be misleading in throwing athletes as several studies have demonstrated significant amounts of shoulder pathology in asymptomatic athletes [14–18]. The incidence of full thickness tears was 0% in these studies, however, the partial thickness tears were detected in 20–86% of patients. Therefore, when evaluating a throwing athlete for shoulder pain, the physician must perform a thorough history and exam as the pain the patient may be experiencing may not be a result of rotator cuff pathology [19]. Furthermore, SLAP tears are a common finding in throwing athletes, which may not require immediate intervention [19].

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## 5.4 Management Principles

### 5.4.1 Non-surgical Treatment

The primary goal of intervention is to restore painless function to the affected shoulder. Conservative treatment should be employed for all patients prior to seeking surgical intervention, especially for throwing athletes. The first line of therapy consists of activity modification, non-steroidal anti-inflammatory drugs (NSAIDs),

and rehabilitation. This is an attempt to decrease inflammation and improve function.

Physical therapy with a stretching and strengthening programme, as well as sports and job specific training are performed. Physical therapy is sport- and/or job-specific and focuses on restoring normal glenohumeral kinematics. Exercises in the scapular plane with strengthening of the scapular stabilisers such as the serratus anterior and trapezius muscle, strengthening of the internal rotators of the shoulder, stretching of the posterior capsule, and isokinetic progressive endurance training should all be included (Fig. 5.4) [8].

Corticosteroid injections into the glenohumeral joint may provide both pain relief and serve as diagnostic tool by serial injections to differentiate glenohumeral joint pain from acromioclavicular joint pain and subacromial pain.

A recent randomised controlled study investigated the differences in outcomes at 5 years following physiotherapy only or



**Fig. 5.4** For the sleeper stretch, the individual lies on the involved side with the shoulder in approximately 90° of forward elevation. The other arm is used to internally rotate the involved shoulder until a stretch is achieved on the posterior aspect of the shoulder (from Braun et al. [7])

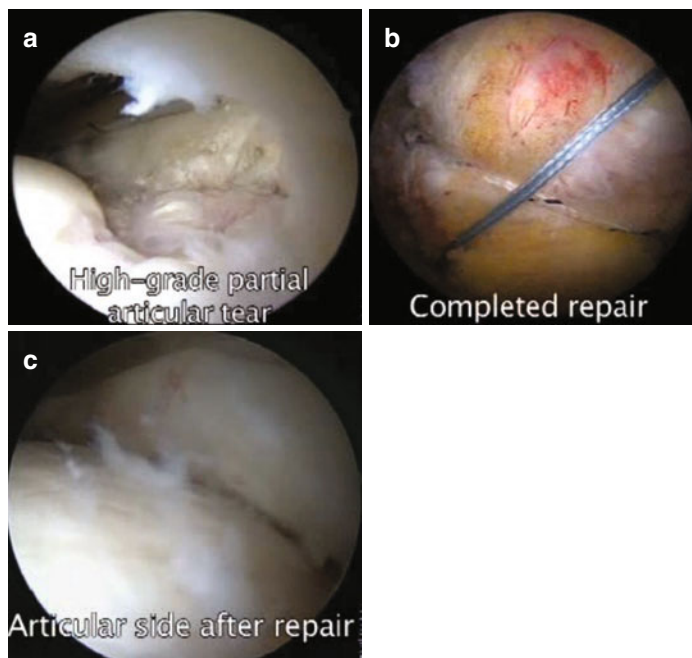
operative treatment for rotator cuff tears not exceeding 3 cm [20]. The investigators found that primary repair of small- and medium-sized rotator cuff tears were associated with better outcomes than the tears treated with physiotherapy, however, they concluded they may be below clinical importance. Importantly, 37% of tears treated with physiotherapy only, progressed by greater than 5 mm over 5 years. Given that these tears are now larger, surgical treatment is more challenging and outcomes less predictable [21–25].

### 5.4.2 Surgical Treatment

In case of failed non-operative treatment or proven full thickness rotator cuff tearing, surgery is indicated. Rotator cuff tears and SLAP lesions usually do not heal spontaneously [26]. Historically, open repairs of the rotator cuff and labrum were effective. However, with the improvement in arthroscopic equipment as well as surgical skill most rotator cuff tears and lesions of the labrum can readily be repaired arthroscopically [25, 27–29].

Bilateral examination under anaesthesia is performed first to assess range of motion and stability of the joint. Diagnostic arthroscopy enables visualisation of intraarticular pathologies throughout dynamic positioning of the shoulder. These pathologies include SLAP lesions, partial thickness tears of the undersurface of the rotator cuff, as well as full thickness tears. If scuffing of the articular side of the rotator cuff is noted on diagnostic arthroscopy, the physician should be aware that this can be a normal physiologic phenomenon in baseball players. This should, therefore, only be addressed if the clinical symptoms are concordant [15]. Posterior labral repair is rarely indicated in overhead athletes unless there is evidence of posterior glenohumeral instability.

The surgical management of partial thickness tears varies depending on the overhead demands of the patient (Fig. 5.5). Many surgeons advocate for simple debridement when the tear involves less than 50% of the thickness of the tendon while tears involving greater than 50% of the thickness should undergo repair. However, in the throwing athlete, outcomes of repair are less



**Fig. 5.5** (a) A high grade partial thickness articular sided tear of the supraspinatus tendon is visualized from the posterior portal. (b) View after repair of the bursal side of the rotator cuff from the lateral portal. (c) View after repair of the articular side of the rotator cuff from the posterior portal

favourable [19] and, therefore, the senior surgeon (PJM) prefers to manage tears involving up to 75% thickness with debridement.

Full-thickness rotator cuff tears are repaired in a stepwise manner that both reduces the torn rotator cuff tendon to its native footprint but also creates a favourable environment for healing. In progression, the rotator cuff footprint on the greater tuberosity is first decorticated to create a bleeding bony surface for healing [28–30]. Next, the tendon is mobilised to remove bursal adhesions and scarring that could prevent anatomic reduction. Third, suture anchors are placed into the humerus and sequentially passed

through the rotator cuff tendon. These anchors can be placed in a variety of configurations (single-row or double-row) depending on the biomechanical stability desired. Lastly, the sutures are secured to the tendon in either a knotless or knotted fashion while confirming anatomic reduction of the tendon to its footprint.

A level I meta-analysis conducted by Millett et al. [31] investigated the differences in clinical outcomes between rotator cuff tears that were repaired with a single row versus a double row technique. Although no statistically significant differences were found in clinical outcomes between the repair techniques, single row repairs were associated with higher re-tear rate.

In case of anterior subluxation and a distended anterior joint capsule, anterior or antero-inferior plication can be performed. The posterior capsule is usually released. Range of motion and stability are re-checked after the capsular procedures.

Derotational humeral osteotomy with a myorrhaphy of the subscapularis muscle is an option to be considered in athletes after failure of all other means of treatment, including arthroscopic management [32].

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## 5.5 Rehabilitation Following Surgery

Rehabilitation after surgery is an important aspect of treatment. Physical therapy is divided into four phases. Phase I focuses on establishing passive range of motion to reduce the risk of postoperative stiffness while focusing on protecting the integrity of the rotator cuff repair. Once passive motion has been established, typically about 4 weeks following surgery, the patient progresses to phase II which focuses on active assisted range of motion and active range of motion. At approximately 8 weeks postoperatively, phase III is begun which focuses on strengthening the rotator cuff followed by phase IV at approximately 12–16 weeks postoperatively at which advanced strengthening is begun. When phase IV is completed a gradual progressive return to sport rehabilitation plan is initiated prior to allowing the patient to return to competitive sporting activities.



## 5.6 Clinical Outcomes

### 5.6.1 Partial Thickness Tears

The management of partial thickness rotator cuff tears remains controversial. Studies have reported excellent overall outcomes after arthroscopic treatment of partial thickness rotator cuff tears, however, the ideal management remains an area of debate [31, 33–40].

Millett et al. [31] investigated the outcomes of treatment of partial thickness tears and found improvements in ASES, and SANE scores—79% of athletes in the cohort were able to compete in sports at or near preinjury levels of intensity. In addition, patients who underwent a concomitant acromioplasty had a significantly better SANE score ( $p = 0.043$ ). However, other investigators have advised to avoid anterior acromioplasty in patients with impingement due to unfavourable outcomes [41, 42]. Sonnery-Cottet et al. [43] investigated the outcomes of 28 tennis players with symptomatic posterosuperior glenoid impingement who underwent arthroscopic debridement for partial articular sided tendon avulsions. Despite a high level of satisfaction (82%) and high level of return to sport (79%), 82% of the players who returned to sport reported continued pain while competing.

In throwing athletes, the rate of return to sport at the same level following debridement of partial thickness rotator cuff tears has ranged from 16% to 76% [44–47]. Reynolds et al. [46] investigated outcomes of debridement for small partial thickness rotator cuff tears in elite pitchers. Sixty-seven out of eighty-two (82%) returned to sport, however, only 37/67 (55%) returned to sport at the same or higher level. Payne et al. investigated debridement of partial thickness tears in young athletes and found that for overhead athletes, they had significant pain relief from the surgery but only 45% returned to sport. Ide et al. [48] investigated outcomes or repair of partial thickness rotator cuff tears to the bone and found that 2/6 throwing athletes returned to the same level of sport. These studies highlighting return to sport levels are important and can be used in counselling patients and managing preoperative expectations.

### 5.6.2 Full Thickness Tears

Reported outcomes following surgical treatment of full thickness rotator cuff tears have been favourable across a wide spectrum of activity levels and age ranges [25, 30, 31, 40, 49–51]. In fact, a recent study conducted by Bhatia et al. [49] investigated the outcomes of arthroscopic repair of full thickness rotator cuff tears in recreational athletes aged 70 years or older. At a mean follow-up of 3.6 years, there were significant improvements in all subjective outcomes scores and no patients required revision rotator cuff repairs. The American Shoulder and Elbow Score (ASES) was comparable to age-matched individuals with normal shoulder function. In addition, 77% of patients were able to return to their recreational sport at a similar level of intensity to pre injury levels.

Full thickness tears in elite overhead athletes are uncommon, however they can be a career-threatening injury [52, 53]. Mazoue and Andrews [52] investigated outcomes in 12 professional baseball pitchers who underwent a mini-open repair on their dominant arm. Only 1 player (8%) was able to return to a high level of competition. Van Kleunen et al. [53] investigated outcomes following repair of combined SLAP and infraspinatus tears and found that 17% of patients with full thickness tears returned to the same sporting level. Given the low rates of return to sport following full thickness rotator cuff tears, it is extremely important to exhaust all conservative treatment options prior to engaging in surgical intervention.

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## 5.7 Conclusion

Rotator cuff tears are a common cause of shoulder pain and discomfort in the general population and athletes. An understanding of the biomechanics, aetiology, diagnostic testing, and outcomes can help the healthcare provider optimally treat patients and return them to a high level of function and activity. Athletes often suffer from partial thickness rotator cuff tears, however, outcomes are favourable following treatment. Full thickness tears are uncommon but career threatening in elite overhead athletes.

## Q&A

- (1) What is Glenohumeral Internal Rotation Deficit?  
Tightness of the posterior IGHL complex and posteroinferior capsule that is commonly seen in throwers
- (2) Which type of impingement is relatively uncommon in young athletes?  
External impingement
- (3) In rehabilitation of rotator cuff repairs, what type of motion is typically done first during rehab?  
Passive range of motion
- (4) The Abduction and External Rotation view (ABER view) is especially helpful during MRI to look for the presence of what kind of pathology?  
Partial thickness flap tears of the rotator cuff

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## References

1. Hansen ML, Otis JC, Johnson JS, Cordasco FA, Craig EV, Warren RF. Biomechanics of massive rotator cuff tears: implications for treatment. *J Bone Joint Surg Am.* 2008;90:316–25.
2. Burkhart SS, Nottage WM, Ogilvie-Harris DJ, Kohn HS, Pachelli A. Partial repair of irreparable rotator cuff tears. *Arthroscopy.* 1994;10:363–70.
3. Costouros JG, Porramatikul M, Lie DT, Warner JJ. Reversal of suprascapular neuropathy following repair of massive supraspinatus and infraspinatus rotator cuff tears. *Arthroscopy.* 2007;23(11):1152–61.
4. Mallon WJ, Wilson RJ, Basamania CJ. The association of suprascapular neuropathy with massive rotator cuff tears: a preliminary report. *J Shoulder Elb Surg.* 2006;15(4):395–8.
5. Paley KJ, Jobe FW, Pink MM, Kvitne RS, ElAttrache NS. Arthroscopic findings in the overhand throwing athlete: evidence for posterior internal impingement of the rotator cuff. *Arthroscopy.* 2000;16(1):35–40.
6. Walch G, Boileau P, Noel E, Donell ST. Impingement of the deep surface of the supraspinatus tendon on the posterosuperior glenoid rim: An arthroscopic study. *J Shoulder Elb Surg.* 1992;1(5):238–45.
7. Braun S, Kokmeyer D, Millett PJ. Shoulder injuries in the throwing athlete. *J Bone Joint Surg Am.* 2009;91:966–78.
8. Spiegl UJ, Warth RJ, Millett PJ. Symptomatic internal impingement of the shoulder in overhead athletes. *Sports Med Arthrosc.* 2014;22(2):120–9.

9. Burkhart SS, Morgan CD, Kibler WB. The disabled throwing shoulder: spectrum of pathology part I: pathoanatomy and biomechanics. *Arthroscopy*. 2003;19(4):404–20.
10. T treault P, Krueger A, Zurakowski D, Gerber C. Glenoid version and rotator cuff tears. *J Orthop Res*. 2004;22(1):202–7.
11. Jobe CM. Posterior superior glenoid impingement: expanded spectrum. *Arthroscopy*. 1995;11(5):530–6.
12. O'Brien SJ, Pagnani MJ, Fealy S, McGlynn SR, Wilson JB. The active compression test: a new and effective test for diagnosing labral tears and acromioclavicular joint abnormality. *Am J Sports Med*. 1998;25(6):610–3.
13. Iyengar JJ, Burnett KR, Nottage WM, Harwin SF. The abduction external rotation (ABER) view for MRI of the shoulder. *Orthopedics*. 2010;33(8):562–5.
14. Connor PM, Banks DM, Tyson AB, Coumas JS, D'Alessandro DF. Magnetic resonance imaging of the asymptomatic shoulder of overhead athletes: a 5-year follow-up study. *Am J Sports Med*. 2003;31(5):724–7.
15. Halbrecht JL, Tirman P, Atkin D. Internal impingement of the shoulder: comparison of findings between the throwing and nonthrowing shoulders of college baseball players. *Arthroscopy*. 1999;15(3):253–8.
16. Jerosch J, Castro WH, Drescher H, Assheuer J. Magnetic resonance morphologic changes in shoulder joints of world class water polo players. *Sportverletz Sportschaden*. 1992;7(3):109–14.
17. Jost B, Zumstein M, Pfirrmann CW, Zanetti M, Gerber C. MRI findings in throwing shoulders: abnormalities in professional handball players. *Clin Orthop Relat Res*. 2005;434:130–7.
18. Miniaci A, Mascia AT, Salonen DC, Becker EJ. Magnetic resonance imaging of the shoulder in asymptomatic professional baseball pitchers. *Am J Sports Med*. 2002;30(1):66–73.
19. Andrews JR, Wilcox JR. Decision making in the throwing athlete. *Sports Med Arthrosc Rev*. 2014;22(2):130–6.
20. Moosmayer S, Lund G, Seljom US, Haldorsen B, Svege IC, Henning T, Pripp AH, Smith HJ. Tendon repair compared with physiotherapy in the treatment of rotator cuff tears: a randomized controlled study in 103 cases with a five year follow-up. *J Bone Joint Surg Am*. 2014;96(18):1504–14.
21. Galatz LM, Ball CM, Teefey SA, Middleton WD, Yamaguchi K. The outcome and repair integrity of complete arthroscopically repaired large and massive rotator cuff tears. *J Bone Joint Surg Am*. 2004;86A:219–24.
22. Gerber C, Fuchs B, Hodler J. The results of repair of massive tears of the rotator cuff. *J Bone Joint Surg Am*. 2000;82:505–15.
23. Holtby R, Razmjou H. Relationship between clinical and surgical findings and reparability of large and massive rotator cuff tears: a longitudinal study. *BMC Musculoskelet Disord*. 2014;15:180.

24. Iagulli ND, Field LD, Hobgood R, Ramsey JR, Savoie FH III. Comparison of partial versus complete arthroscopic repair of massive rotator cuff tears. *Am J Sports Med.* 2012;40:1022–6.
25. Millett PJ, Warth RJ. Posterosuperior rotator cuff tears: classification, pattern recognition, and treatment. *J Am Acad Orthop Surg.* 2014;30:778–80.
26. Conway JE. Arthroscopic repair of partial-thickness rotator cuff tears and SLAP lesions in professional baseball players. *Orthop Clin North Am.* 2001;32(3):443–56.
27. Gaskill TR, Braun S, Millett PJ. Multimedia article. The rotator interval: pathology and management. *Arthroscopy.* 2011;27(4):556–67.
28. Vaishnav S, Millett PJ. Arthroscopic rotator cuff repair: scientific rationale, surgical technique, and early clinical and functional results of a knotless self-reinforcing double-row rotator cuff repair system. *J Shoulder Elb Surg.* 2010;19(2 Suppl):83–90.
29. Warth RJ, Greenspoon JA, Bhatia S, Millett PJ. Arthroscopic double-row rotator cuff repair using a knotless, interconnected technique. *Oper Tech Orthop.* 2015;25(1):1–6.
30. Ames JB, Horan MP, Van der Meijden OA, Leake MJ, Millett PJ. Association between acromial index and outcomes following arthroscopic repair of full-thickness rotator cuff tears. *J Bone Joint Surg Am.* 2012;94(20):1862–9.
31. Millett PJ, Warth RJ, Dornan GJ, Lee JT, Speigl UJ. Clinical and structural outcomes after arthroscopic single-row versus double-row rotator cuff repair: a systematic review and meta-analysis of level I randomized clinical trials. *J Shoulder Elb Surg.* 2014;23(4):586–97.
32. Riand N, Levigne C, Renaud E, Walch G. Results of derotational humeral osteotomy in posterosuperior glenoid impingement. *Am J Sports Med.* 1998;26(3):453–9.
33. Chung SW, Kim JY, Yoon JP, Lyu SH, Rhee SM, Oh SB. Arthroscopic repair of partial-thickness and small full-thickness rotator cuff tears: tendon quality as a prognostic factor for repair integrity. *Am J Sports Med.* 2014;43(3):588–96.
34. Duralde XA, McClelland WB Jr. The clinical results of arthroscopic transtendinous repair of grade III partial articular-sided supraspinatus tendon tears. *Arthroscopy.* 2012;28(2):160–8.
35. Eid AS, Dwyer AJ, Chamblar AF. Mid-term results of arthroscopic subacromial decompression in patients with or without partial thickness rotator cuff tears. *Int J Shoulder Surg.* 2012;6(3):86–9.
36. Franceschi F, Papalia R, Del Buono A, Vasta S, Costa V, Maffulli N, Denaro V. Articular-sided rotator cuff tears: which is the best repair? A three-year prospective randomised controlled trial. *Int Orthop.* 2013;37(8):1487–93.

37. Kim KC, Shin HD, Cha SM, Park JY. Clinical outcomes after arthroscopic trans-tendon suture-bridge technique in partial-thickness articular-side rotator cuff tear. *Knee Surg Sports Traumatol Arthrosc.* 2013;21(5):1183–8.
38. Kim KC, Shin HD, Cha SM, Park JY. Repair integrity and functional outcome after arthroscopic conversion to a full-thickness rotator cuff tear: articular- versus bursal-side partial tears. *Am J Sports Med.* 2014;42(2):451–6.
39. Peters KS, McCallum S, Briggs L, Murrell GA. A comparison of outcomes after arthroscopic repair of partial versus small or medium-sized full-thickness rotator cuff tears. *J Bone Joint Surg Am.* 2012;94(12):1078–85.
40. Sonnery-Cottet B, Edwards TB, Noel E, Walch G. Rotator cuff tears in middle-aged tennis players: results of surgical treatment. *Am J Sports Med.* 2002;30(4):558–64.
41. Mithofer K, Fealey S, Altchek DW. Arthroscopic treatment of internal impingement of the shoulder. *Tech Should Elbow Surg.* 2004;5:66–75.
42. Tibone JE, Jobe FW, Kerlan RK, Carter VS, Shields CL, Lombardo SJ, Yocum LA. Shoulder impingement syndrome in athletes treated by an anterior acromioplasty. *Clin Orthop Relat Res.* 1985;198:134–40.
43. Sonnery-Cottet B, Edwards TB, Noel E, Walch G. Results of arthroscopic treatment of posterosuperior glenoid impingement in tennis players. *Am J Sports Med.* 2002;30(2):227–32.
44. Andrews JR, Broussard TS, Carson WG. Arthroscopy of the shoulder in the management of partial tears of the rotator cuff: a preliminary report. *Arthroscopy.* 1985;1(2):117–22.
45. Payne LZ, Altchek DW, Craig EV, Warren RF. Arthroscopic treatment of partial rotator cuff tears in young athletes. A preliminary report. *Am J Sports Med.* 1997;25(3):299–305.
46. Reynolds SB, Dugas JR, Cain EL, McMichael CS, Andrews JR. Debridement of small partial-thickness rotatorcuff tears in elite overhead throwers. *Clin Orthop Relat Res.* 2008;466(3):614–21.
47. Riand N, Boulahia A, Walch G. Posterosuperior impingement of the shoulder in the athlete: results of arthroscopic debridement in 75 patients. *Rev Chir Orthop Reparatrice Appar Mot.* 2002;88(1):19–27.
48. Ide J, Maeda S, Takagi K. Arthroscopic transtendon repair of partial-thickness articular-sided tears of the rotator cuff: anatomical and clinical study. *Am J Sports Med.* 2005;33(11):1672–9.
49. Bhatia S, Greenspoon JA, Horan MP, Warth RJ, Millett PJ. Two-year outcomes following arthroscopic rotator cuff repair in recreational athletes over 70 years of age. *Am J Sports Med.* 2015;43(7):1737–42.
50. MacKenchia MA, Chahal J, Wasserstein D, Thoeoropoulos JS, Henry P, Dwyer T. Repair of full-thickness rotator cuff tears in patients aged younger than 55 years. *Arthroscopy.* 2014;30(10):1366–71.

51. Warth RJ, Dornan GJ, James EW, Horan MP, Millett PJ. Clinical and structural outcomes after arthroscopic repair of full-thickness rotator cuff tears with and without platelet-rich product supplementation: a meta-analysis and meta-regression. *Arthroscopy*. 2015;31(2):306–20.
52. Mazoue CG, Andrews JR. Repair of full-thickness tears in professional baseball players. *Am J Sports Med*. 2006;34(2):182–9.
53. Van Kleunen JP, Tucker SA, Field LD, Savoie FH. Return to high level throwing after combination infraspinatus, SLAP repair, and release of glenohumeral internal rotation deficit. *Am J Sports Med*. 2012;40(11):2536–41.



# Labral Injuries in Athletes

# 6

Jin Young Park and Jae Hyung Lee

## Key Learning Points

- Labral injuries commonly occur as a result of direct or indirect trauma to the glenohumeral joint in athletes
- Anterior dislocations and instability is most common
- Acute dislocations can often be managed non-operatively, but surgery should be considered where there are bony lesions and for recurrent instability
- Specific clinical tests can help determine the direction of instability and laxity, supported by MR arthrogram or CT arthrogram
- SLAP tears are not common in isolation and can be managed non-operatively initially.

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## 6.1 Instability of the Shoulder

Matsen et al. [1] described two types of shoulder instability: TUBS (traumatic, unidirectional, Bankart, surgery) and AMBRI (atraumatic, multidirectional, bilateral, rehabilitation, inferior capsular shift). However, most shoulder instability involves characteristics

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of both. Shoulder instability can be categorized in several ways: dislocation or subluxation; unidirectional or multidirectional; and traumatic or atraumatic. Most unilateral instability is the result of acute traumatic anterior dislocation, which leads to anterior instability of the shoulder. The presence of multidirectional instability refers to coexisting anterior, inferior, and posterior instability resulting from innate ligament laxity. The prevalence of multidirectional instability is not clear, but Gerber and Nyffeler [2] reported that it comprises less than 5% of all instability.

### **Editors Notes**

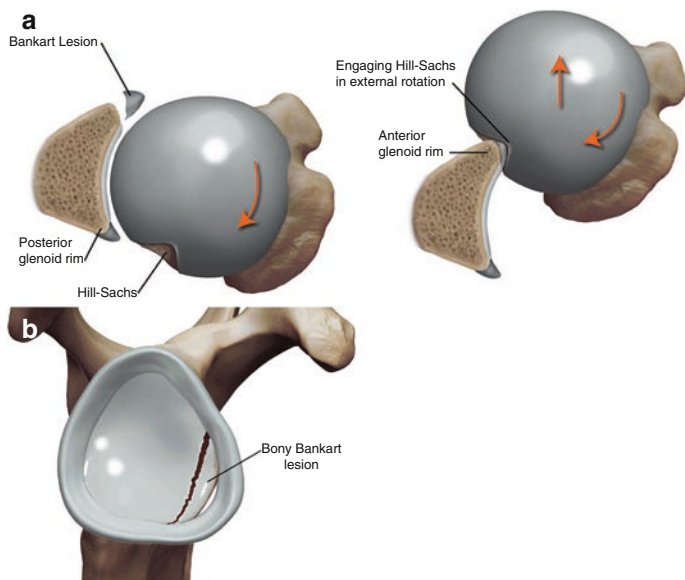
The role of dynamic stabilising factors is also very important to consider. This is highlighted in the classification system described by Lewis et al. [3]. The Stanmore Triangle has three polar groups; traumatic, atraumatic structural and non-structural muscle patterning or sequencing. Patients with abnormal muscle sequencing have instability generated by inappropriate action of the large torque producing muscles predominately Pectoralis Major and Latissimus Dorsi. They are not suitable for surgical intervention until this is addressed by highly specialist rehabilitation.

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## **6.2 Acute Anterior Dislocation**

One of the more common traumatic sports injuries is acute anterior shoulder dislocation, which usually results from excessive abduction and external rotation of the shoulder. In acute anterior shoulder dislocation, the anterior labrum or anterior glenoid is damaged (a Bankart or bony Bankart lesion). In addition, posterior humeral head compression fractures can occur due to impact with the anterior glenoid (Hill–Sachs lesion) (Fig. 6.1). Usually, the patient reports direct or indirect trauma and feels the shoulder popping out.

The physical examination reveals protrusion of the humeral head on the anterior aspect of the shoulder and sunken skin just below acromion. It may be accompanied by an axillary nerve injury which is easily diagnosed by assessing paresthesia over the lateral deltoid skin area.



**Fig. 6.1** Illustrations of (a) a Bankart tear and Hill-Sachs lesion with shoulder dislocation viewed from above (b) Bony Bankart lesion (courtesy of Leonard Funk, <http://www.shoulderdoc.co.uk>)

Before reducing the dislocation, plain radiographs should be taken to ensure an accurate diagnosis (Fig. 6.2). A prompt reduction is easier and non-steroidal anti-inflammatory drugs (NSAIDs) or muscle relaxants can be prescribed to make patients more comfortable. There are a number of described reduction techniques. Our preference is the Stimson manoeuvre [4] which is considered a safe reduction method (Fig. 6.3).

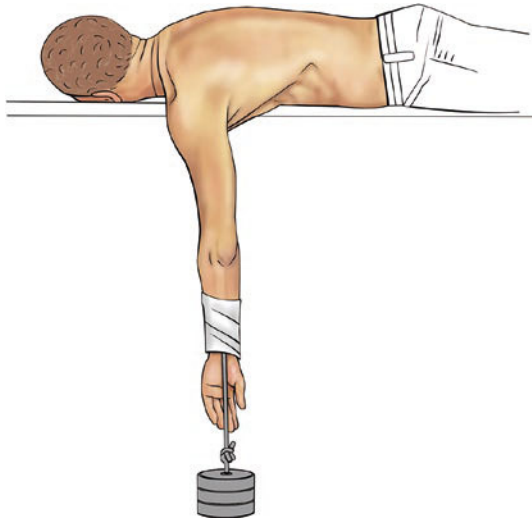
Once reduction is achieved, an arm sling is applied for comfort for 2–3 weeks. Although controversial, some papers report that internal rotation of the arm can aggravate a Bankart lesion, so we feel that an arm sling is beneficial after reducing the shoulder dislocation.

Anterior dislocation of the shoulder can be treated in several ways. However, most physicians start with immediate isometric exercises to strengthen internal rotation. After 3 weeks, the patient

**Fig. 6.2** Anterior shoulder dislocation on plain radiograph



**Fig. 6.3** Stimson manoeuvre (courtesy of Lennard Funk, <http://www.shoulderdoc.co.uk>)



is allowed to perform active external rotation, while abduction is not permitted until 6 weeks.

### **Editor's Note**

The longitudinal studies by Hovelius et al. [5] found that sling immobilisation did not alter the outcome of non-operatively managed dislocations and so many surgeons would advocate early movement and rehabilitation of dynamic stabilisation with specialist physiotherapy.

Young athletes in their teens and twenties have a greater tendency to re-dislocate after a first traumatic dislocation. Therefore, surgery can be considered in young athletes. The surgical treatment of choice is an arthroscopic Bankart repair. Twenty-four hours postoperatively, pendulum exercises are allowed and an abduction sling is applied for 4–6 weeks. To minimise anterior capsule contracture, the sling is applied with the shoulder in slight abduction and external rotation. After 6 weeks, active muscle strengthening is allowed; minor sports activities can be started at 3–4 months. The athlete can return to full activities at 6 months.

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## **6.3 Anterior Instability (Recurrent Anterior Dislocation)**

Anterior instability implies that there has been damage to the anteroinferior glenohumeral ligament, leading to dislocation or subluxation. A good analogy is to consider the shoulder as a golf ball on a tee; it is easily knocked off (or dislocated) with trauma. Anterior instability usually arises from acute dislocation and its prevalence is high in people in their teens and twenties. Most of the instability occurs within 2 years of a traumatic dislocation [6], and arm abduction, external rotation, and extension position can lead to dislocation. With frequent dislocation, some patients can reduce their shoulder themselves by using traction and rotation.

Bankart (anteroinferior glenoid labral tear) and Hill–Sachs lesions are common pathologies in anterior instability of the shoulder.

A detailed description of the first dislocation event is essential, including the nature of the trauma, the arm position during the

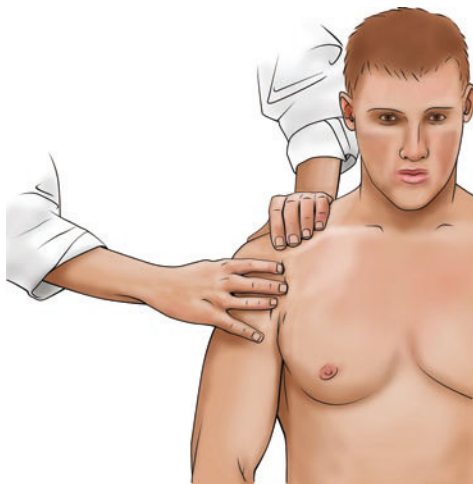
trauma, and the method of reduction and rehabilitation protocol. With anterior instability, patients fear anterior dislocation on abduction and external rotation, which makes the physical examination difficult. It is important to identify generalised hyperlaxity. The Beighton score metacarpophalangeal dorsiflexion, the distance from the thumb to the forearm with wrist flexion, elbow hyperflexion, and genu recurvatum must be measured [7]. The findings should be compared with those of the contralateral side.

### 6.3.1 Examination

Differential shoulder laxity can be assessed with the drawer tests and sulcus test.

**Drawer Test** With the patient sitting and resting, the surgeon holds the scapula with one hand and the humerus head and neck with the other. Then, the surgeon moves the humerus anteriorly (Fig. 6.4): Grade 0 is no translation; Grade 1 is translation just before the glenoid rim; Grade 2 is translation on the glenoid rim; and Grade 3 is translation past the glenoid rim.

**Fig. 6.4** Anterior drawer test (courtesy of Lennard Funk, <http://www.shoulderdoc.co.uk>)



**Fig. 6.5** Sulcus test (courtesy of Lennard Funk, <http://www.shoulderdoc.co.uk>)



**Sulcus Test** With the patient sitting and the arm in internal rotation, traction is applied to the arm (Fig. 6.5). Sunken skin between the acromion and humeral head is a positive sign. In a non-pathological shoulder, with the arm in external rotation, the sulcus sign disappears. A positive sulcus sign means that there is laxity of rotator interval: 1+ means subluxation <1 cm; 2+ means subluxation of 1–2 cm; and 3+ means subluxation >2 cm. Several apprehension tests are used to diagnose anterior instability of the shoulder.

**Fig. 6.6** Crank test  
(courtesy of Lennard  
Funk, [http://www.  
shoulderdoc.co.uk](http://www.shoulderdoc.co.uk))

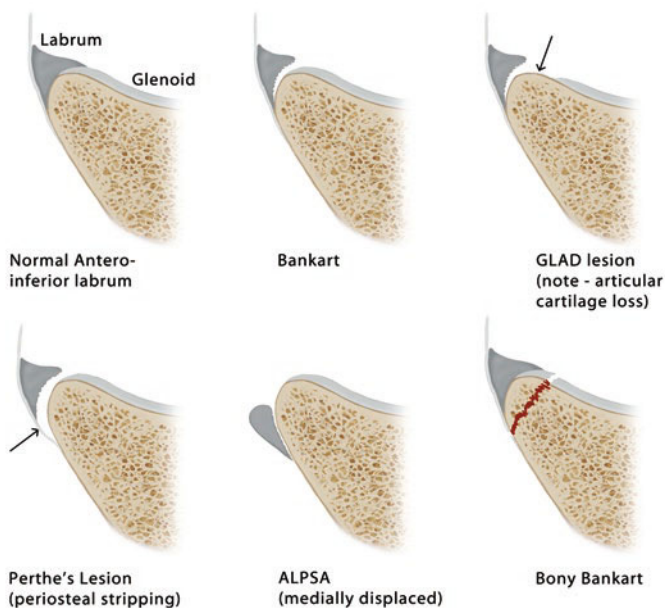


**Crank Test** With the patient sitting and resting, the surgeon holds the scapula with one hand and the arm with the other. Then, the arm is slowly abducted, externally rotated, and extended (Fig. 6.6). If the patient suffers pain or fears dislocation, the test is positive. This is called the fulcrum test when performed with the patient in the supine position.

**Relocation Test** After the fulcrum test, press the humerus back into the glenoid. If the patient's pain and anxiety are relieved, the sign is positive.

### 6.3.2 Imaging

Bony Bankart and Hill–Sachs lesions may be seen on plain radiographs. True anteroposterior (AP), apico-oblique, axillary lateral, West Point, and Stryker notch views should be obtained. The West Point and apico-oblique views reveal glenoid rim erosion, bone defects, and bony Bankart lesions; the Stryker notch view reveals Hill–Sachs lesions; and the axillary lateral view reveals the relationship between the glenoid and humeral head and articular surface. More detailed imaging such as magnetic resonance (MR) arthrogram or CT arthrogram will give a better representation of soft tissue injuries, particularly labral tears. Bony lesions are better visualized on CT.



**Fig. 6.7** Variants of labral tears (courtesy of Lennard Funk, <http://www.shoulderdoc.co.uk>)

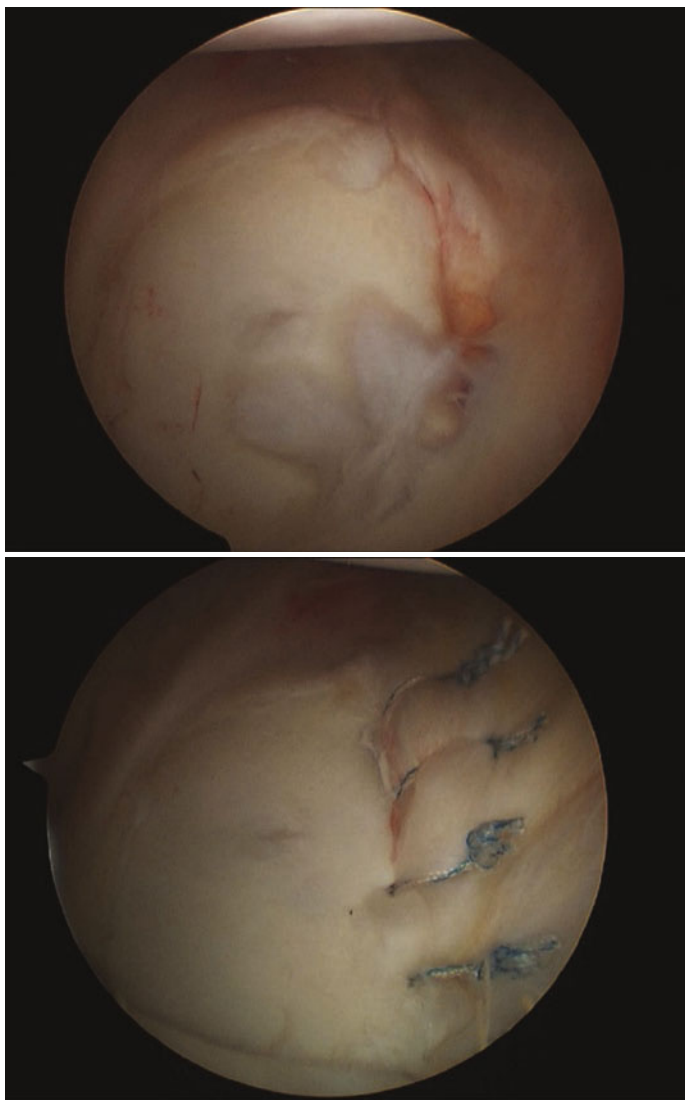
There are several types of glenoid labrum lesions (Fig. 6.7):

### 6.3.3 Treatment

Although there is some controversy over the use of surgical treatment for dislocation in young athletes, non-active patients are managed conservatively. There is no relationship between prolonged immobilisation and recurrent dislocation. Typically, after 3 months of isometric exercise, active muscle strengthening exercises are started. At 6 months, all sports activities are allowed.

If pain, loss of range of motion, and high recurrent dislocation rates persist with non-operative rehabilitation, surgeons should consider operative treatment. The arthroscopic Bankart repair (Fig. 6.8) is the gold standard for treating anterior instability and





**Fig. 6.8** Arthroscopic Bankart lesion and repair

the reported outcome is similar to that of open surgery [8]. After a Bankart repair, if laxity of the inferior capsule is present, then an inferior capsular shift should be performed. For a large bony Bankart lesion or a large glenoid rim defect, the bone transfer Latarjet) procedure is done [9]. Recent studies show that while a small Hill–Sachs lesion requires no direct treatment, a large Hill–Sachs lesion requires a remplissage procedure [10]. Postoperatively, minor sports activity starts at 3 months and a return to full competition is allowed at 6 months.

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## 6.4 Acute Posterior Dislocation

Acute posterior dislocation of the glenohumeral joint comprises 2–4% of all shoulder dislocations. In athletes, direct trauma, falling on an out-stretched arm, or a seizure are causes of posterior dislocation. With a posterior dislocation, there is loss of the normal muscle appearance and the arm is held in internal rotation and adduction. Limitation of external rotation is an important sign.

Posterior dislocation is easily missed on plain radiographs (Fig. 6.9). The lateral and, if possible, axillary lateral views help in the diagnosis.

**Fig. 6.9** Posterior dislocation X-ray



The shoulder is reduced by applying traction and anterior translation force. After reduction, a sling is used for 2–3 weeks and a return to minor sports activity is allowed at 3 months.

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## 6.5 Posterior Instability

Similar to acute posterior dislocation, posterior instability is rare. The traumatic mechanism or pathology is unclear and it is difficult to discriminate from other diseases.

The main reason for posterior shoulder instability is laxity of the posteroinferior glenohumeral ligament, tears of the posterior glenoid labrum, or excessive glenoid retroversion.

Distinctive symptoms of posterior shoulder instability are pain and crepitus. Crepitus occurs with flexion, adduction, and internal rotation of the arm. Like acute posterior dislocation, subluxation is frequent with adduction and internal rotation and mostly it occurs as a form of recurrent subluxation or instability. Many patients will complain of weakness in weight-bearing activities as opposed to true apprehension.

Apprehension tests are used to diagnose posterior instability of the shoulder.

**Jerk Test** The jerk test is one of the most important tests for diagnosing posterior instability. With the patient's arm in 90° of flexion, horizontal adduction, and the elbow at 90°, force is applied in a posterior direction, with the shoulder in adduction and internal rotation (Fig. 6.10). Posterior translation and subluxation of the patient's shoulder is a positive sign.

The drawer and sucks tests are also useful to assess directional laxity in posterior instability, as with anterior instability.

Surgical treatment for posterior instability is indicated if there is no response to 6 months of conservative management or severe pain during the activities of daily living. Without posterior subluxation or pain, the patient should start systemised rehabilitation of the posterior cuff and muscles. Arthroscopic repair is recommended for a reverse Bankart lesion and arthroscopic posterior



**Fig. 6.10** Jerk test (courtesy of Lennard Funk, <http://www.shoulderdoc.co.uk>)

capsular shift for posterior capsular laxity. Generally, the return to sports is similar to that in anterior instability.

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## 6.6 Multidirectional Instability

Multidirectional instability is subluxation or dislocation in two or more directions. Hyperlaxity is the main cause and is frequently accompanied by general laxity of other parts of the body. In athletes, however, repetitive micro-trauma can lead to acquired laxity of the shoulder capsule. Multidirectional instability in the mid-range of motion can result from a hypoplastic glenoid, excessive

retroversion of the glenoid, muscle imbalance, and generalised laxity. In the athletic population it is usually found in those that are hypermobile and their sport involves repetitive overhead trauma to the shoulder. This is covered in more detail in Chap. 2.

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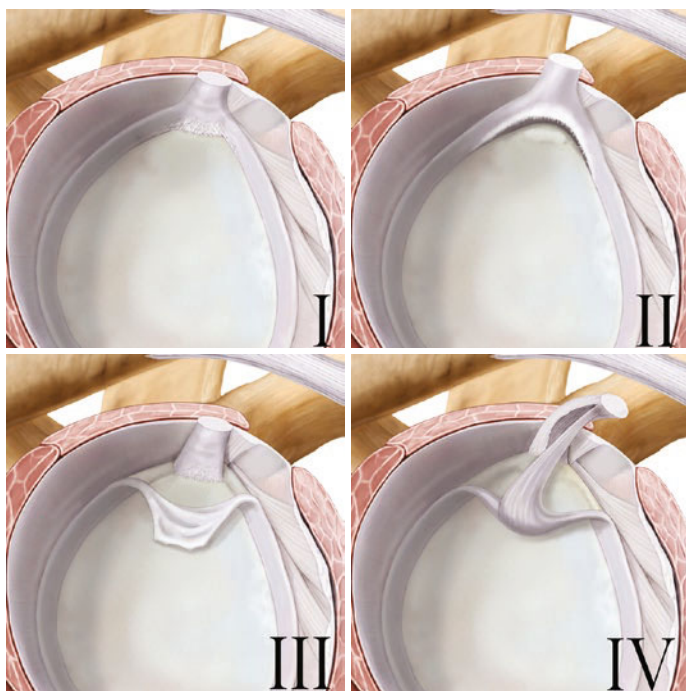
## 6.7 SLAP Lesions

The SLAP (Superior Labrum Anterior to Posterior) lesion was first described by Andrews et al. [11]. Andrews reported superior labral tears in athletes who perform overhead, near the insertion of the biceps long head tendon. In 1990, Snyder described an intra-articular biceps long head insertion rupture, and subsequently these lesions were named SLAP lesions.

The pathophysiology of SLAP lesions involves acute or chronic tension. Direct trauma to the shoulder in adduction and extension or sudden traction on the biceps tendon can lead to a SLAP lesion. Another important mechanism of injury in athletes is repetitive biceps and superior labrum complex tension, which makes the superior labrum translate medially. This is called the peel-back mechanism. In a cadaver study, abduction and external rotation of the arm placed the most tension on the superior labrum, which results in SLAP lesions. Recent studies report that athletes who throw overhead are at high risk of developing SLAP lesions due to the increased external rotation of the arm and decreased internal rotation. This phenomenon is called a glenoid internal rotation deficit (GIRD). SLAP lesions and internal impingement are explained as resulting from anterior capsule laxity and posterior capsule tightness.

Snyder classified SLAP lesions into four different types (Fig. 6.11): Type I is fibrillation and focal degenerative change in the superior labrum; Type II is unstable with abnormal movement of the fully detached biceps-superior labrum complex; Type III is a bucket-handle superior labrum tear; and Type IV is a bucket-handle superior labrum tear that extends into the biceps tendon.

Patients with a SLAP lesion feel pain when pushing objects above their head, and crepitus is felt during abduction and external rotation of the arm. SLAP lesions are often accompanied by

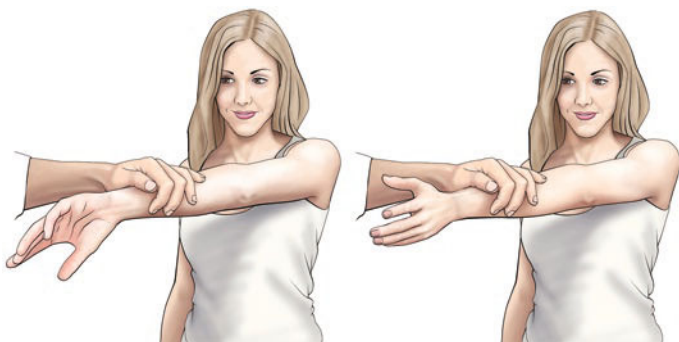


**Fig. 6.11** SLAP lesion type I/type II/type III/type IV (courtesy of Lennard Funk, <http://www.shoulderdoc.co.uk>)

other lesions of the shoulder, which makes this condition difficult to diagnose. The O'Brien test and the compression rotation test have high sensitivity for SLAP lesions.

**The O'Brien Test** [12] Arm flexed at 90° with the elbow fully extended, adduct the arm approximately 15° medially. Then the arm is internally rotated and the patient resists downward force of the examiner (Fig. 6.12). The test is repeat with supinated arm. The symptoms should be reduced in a positive test.

**Compression Rotation Test** With patient in supine position, manual compression of the shoulder joint is applied and passive



**Fig. 6.12** O'Brien test (courtesy of Lennard Funk, <http://www.shoulderdoc.co.uk>)

**Fig. 6.13**

Compression rotation test (courtesy of Lennard Funk, <http://www.shoulderdoc.co.uk>)



rotation of shoulder is performed in an attempt to trap labrum within the joint (Fig. 6.13).

**Speed Test** Arm flexed at  $60^\circ$ , the elbow is fully extended and forearm supinated. The patient tries to forward flex the humerus against resistance of the examiner.

MR arthrogram, including sequences performed with the arm in abduction and external rotation (ABER sequences) is the best investigation for detecting SLAP lesions. At meta-analysis overall sensitivity is 0.87 with specificity 0.92 [13].

The initial treatment of SLAP lesions should be conservative: avoiding activities that induce pain, using NSAIDs, and stretching the posterior capsule will help. If the patient still suffers pain after 4–6 months of rehabilitation, then operative treatment can be considered. Absolute surgical indications for SLAP lesions have not

been established. For Type I, debridement of the fibrillation is sufficient. For Type II, the SLAP lesion can be repaired under careful restricted indications. For Type III, the torn labrum should be resected, but in case of an unstable labrum, labrum repair helps. For Type IV, a biceps tendon tenotomy or tenodesis should be performed, depending on the patient's age [14].

Postoperatively, a sling is worn for 3–4 weeks, and shoulder range of motion exercises are then started to avoid a rigid shoulder. The patient must be careful to avoid abduction and external rotation, which can damage the surgical site. Internal rotation exercises must be performed to release the posterior capsule tightness. Six weeks postoperatively, rotator cuff and deltoid muscle strengthening exercises are started. Recently, the role of the periscapular muscle in SLAP lesions has been emphasised, so it is essential to strengthen the shoulder blade muscles. Return to full sports activities is allowed at 6 months.

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## Q&A

- (1) Is surgery necessary for first time dislocation of shoulder joint?

It is very controversial. For first time dislocation of the glenohumeral joint, conservative treatment is recommended. But, as mentioned earlier, young athletes in their teens and twenties have a greater tendency to re-dislocate after a first traumatic dislocation. Therefore, surgical procedure can be considered.

- (2) Which apprehension test is the best to diagnose anterior instability of the shoulder?

Unfortunately, no single test can represent labral tears or instability of the shoulder. Combination of mechanism or injury, physical examination and radiographs must be considered when making decisions.

- (3) What are the indications for remplissage procedure and Latarjet procedure?

Commonly, if the Hill-Sachs lesion is engaged on glenoid (off-track lesion), remplissage procedure is performed, and



glenoid bone loss of more than 25%, then Latarjet procedure is executed.

(4) SLAP lesion, is surgery really helpful?

Absolute surgical indications for SLAP lesions have not been established. Surgical treatment should be carefully approached. All types of SLAP lesion should be started with rehabilitation. After failure of rehabilitation, surgical procedure can be considered. For Type I, debridement of the fibrillation is sufficient. For Type II, the SLAP lesion can be repaired under careful restricted indications. For Type III, the torn labrum should be resected, but in case of an unstable labrum, labrum repair helps. For Type IV, a biceps tendon tenotomy or tenodesis should be performed, depending on the patient's age.

(5) For instability rehabilitation, what do we have to do?

Generally, thera-bands are used to strengthen rotator cuff muscles and deltoids. Also, periscapular exercises is important, focusing on lower trapezius, rhomboideus and serratus anterior.

**Editor's Note**

Rehabilitation of shoulder instability is a highly specialist field, especially in the context of atraumatic instability. Patients with muscle sequencing abnormalities or psychological components should be managed in specialist centres with multi-disciplinary teams of surgeons and physiotherapist.

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## References

1. Matsen FA, Harryman DT 2nd, Sidles JA. Mechanics of glenohumeral instability. *Clin Sports Med.* 1991;10(4):783–8.
2. Gerber C, Nyffeler RW. Classification of glenohumeral joint instability. *Clin Orthop Relat Res.* 2002;400:65–76.
3. Lewis A, Kitamura T, Bayley JIL. The classification of shoulder instability: new light through old windows. *Curr Orthop.* 2004;18:97–108.
4. Amar E, Maman E, Khashan M, Kauffman E, Rath E, Chechik O. Milch versus Stimson technique for nonsedated reduction of anterior shoulder dislocation: a prospective randomized trial and analysis of factors affecting success. *J Shoulder Elb Surg.* 2012;21(11):1443–9.

5. Hovelius L, Olofsson A, Sandström B, Augustini BG, Krantz L, Fredin H, Tillander B, Skoglund U, Salomonsson B, Nowak J, Sennerby U. 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.
6. 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.
7. Grahame R. The revised (Brighton 1998) criteria for the diagnosis of benign joint hypermobility syndrome (BJHS). *J Rheumatol.* 2000;27:1777–9.
8. Hobby J, Griffin D, Dunbar M, Boileau P. Is arthroscopic surgery for stabilisation of chronic shoulder instability as effective as open surgery? A systematic review and meta-analysis of 62 studies including 3044 arthroscopic operations. *J Bone Joint Surg Br.* 2007;89(9):1188–96.
9. Zimmermann SM, Scheyerer MJ, Farshad M, Catanzaro S, Rahm S, Gerber C. Long-term restoration of anterior shoulder stability: a retrospective analysis of arthroscopic Bankart repair versus open Latarjet procedure. *J Bone Joint Surg Am.* 2016;98(23):1954–61.
10. Lazarides AL, Duchman KR, Ledbetter L, Riboh JC, Garrigues GE. Arthroscopic Remplissage for Anterior Shoulder Instability: A Systematic Review of Clinical and Biomechanical Studies. *Arthroscopy.* 2019;35(2):617–28.
11. Andrews JR, Carson WG Jr, McLeod WD. Glenoid labrum tears related to the long head of the biceps. *Am J Sports Med.* 1985;13(5):337–41.
12. O'Brien SJ, Pagnani MJ, Fealy S, McGlynn SR, Wilson JB. The active compression test: A new and effective test for diagnosing labral tears and acromioclavicular joint abnormality. *Am J Sports Med.* 1998;26:610–3.
13. Arirachakaran A, Boonard M, Chaijenkij K, Pituckanotai K, Prommahachai A, Kongtharvonskul J. A systematic review and meta-analysis of diagnostic test of MRA versus MRI for detection superior labrum anterior to posterior lesions type II-VII. *Skelet Radiol.* 2017;46(2):149–60. <https://doi.org/10.1007/s00256-016-2525-1>.
14. Snyder SJ, Karzel RP, Del Pizzo W, Ferkel RD, Friedman MJ. SLAP lesions of the shoulder. *Arthroscopy.* 1990;6(4):274–9.



# Glenoid Bone Loss in Athletes

# 7

Deepak N. Bhatia and Joe F. De Beer

## Key Learning Points

- Understand the aetiology of glenoid bone injury in full-contact and overhead sports.
- Appreciate the techniques for identifying and quantifying glenoid bone loss
- Understand the role of clinical examination and the relevant clinical tests.
- Outline the treatments, particularly soft tissue procedures such as labroplasty and remplissage, and Bony procedures, such as Latarjet, and auto/allograft reconstruction.

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## 7.1 Introduction

Anterior shoulder instability in athletes is associated with varying degrees of humeral and glenoid bone loss. Contact athletes are a

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high-risk subgroup and bone defects in these athletes are usually significantly large at initial presentation. Historically, Burkhart and De Beer [1] first defined “significant” bone defects and used the terms “engaging Hill–Sachs” and “inverted-pear glenoid” to define significant humeral and glenoid bone loss. They evaluated the results of an arthroscopic soft tissue repair in contact athletes, and reported an 80% recurrence rate in the presence of significant bony lesions. Sugaya et al. [2] evaluated the glenoid rim morphology in 100 consecutive shoulders with recurrent anterior instability, and reported glenoid bone loss of varying severity in 90% of the glenoids.

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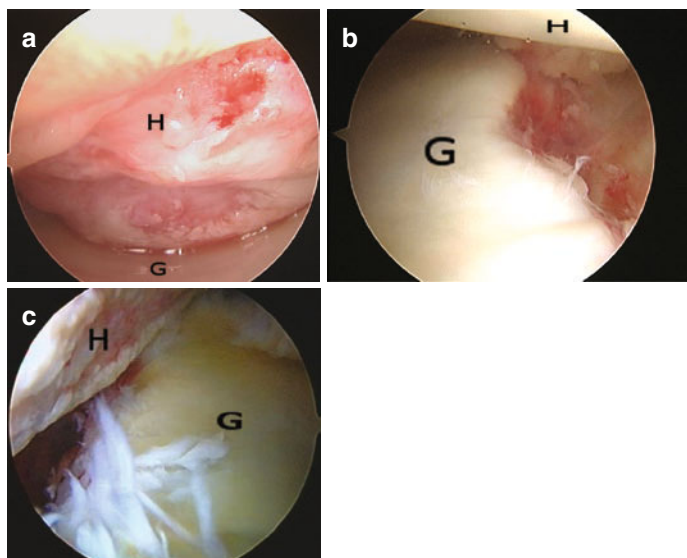
## 7.2 Aetiology

Recurrent anterior dislocations are common in athletes, possibly due to young age, high activity level, and potential for injury. Aggressive, full-contact and overhead sports (rugby, wrestling, judo, mixed martial arts, bodybuilding, weightlifting) result in high-energy traumatic dislocations, and usually have a high incidence of significant bone defects [3]. In contrast, significant defects are less common in non-contact athletes, and a higher incidence of soft tissue lesions (chondrolabral lesions, global labral tears) is seen. Video analysis of the on-field injury reveals the following mechanisms: (a) a direct fall onto the shoulder or when the horizontally abducted arm is forced posteriorly (‘straight-arm tackle’); (b) the player falls forward with the elbow flexed, and the elbow contacts the ground first; as the body falls forwards, the arm is forced posteriorly and this results in an anterior dislocation [3, 4]. Other conditions that are associated with significant glenoid defects include post-concussion seizures, vehicular sports (motocross and downhill mountain biking), and recreational gym injuries.

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## 7.3 Pathology

Traumatic glenohumeral dislocations result in bony and soft tissue lesions of varying severity over the glenoid and humeral head (Fig 7.1).



**Fig. 7.1** (a) A large humeral head defect (Hill–Sachs lesion) is shown (*G* glenoid, *H* humeral defect). (b) A significant glenoid defect that resembles an “inverted-pear” glenoid is shown (*G* glenoid, *H* humeral head). (c) An “off-track” humeral head defect is seen engaging over a significant glenoid defect (*G* glenoid, *H* humeral head)

Glenoid defects occur most frequently at the anterior rim (2.30–4.30 o’clock) and can extend down to the 6 o’clock position on the anteroinferior rim [5]. Glenoid defects can be quantified reliably on preoperative imaging and intraoperative arthroscopic measurements, and a defect measuring 25% of the total glenoid width, or 19–21% of the glenoid length, is considered significant [6, 7]. Sugaya et al. [2] demonstrated “fragment (50%)” and “erosive (40%)” types of glenoid defects, while Bigliani et al. [8] reported 3 types of these lesions (avulsion fracture, medially mal-united fracture, and erosive).

Humeral head defects (Hill–Sachs lesions) occur in 65–93% of anterior instability cases [9, 10]. The critical size of the humeral

defect that is considered significant is unclear (4 cm length, 20–25% of humeral head surface, 250–1000 mm<sup>3</sup> volume) [6, 11–14].

The concept of “engagement” was first put forth by Burkhart and De Beer, and they used dynamic air-arthroscopy to determine if the humeral head defect was an “engaging Hill–Sachs” lesion. Similarly, they used an antero-supero-lateral portal view to visualise and diagnose a significant “inverted-pear glenoid”, and described the bare spot as a landmark to quantify the glenoid defect [1, 15] (Fig. 7.1). Recently, Itoi et al. have described the concept of “Glenoid track” to assess bipolar bone loss. The glenoid track is defined as the “contact zone of the glenoid created on the humeral head along the end range of motion”; the medial margin of the glenoid track is located at a distance equivalent to 84% of the glenoid width in cadaveric shoulders and 83% in live shoulders [6, 7]. Based on this concept, Di Giacomo et al. [16] classified Hill–Sachs lesions as “on-track” and “off-track” Hill–Sachs lesions.

Glenoid bone loss in anterior instability may also be associated with soft tissue lesions. Arrigoni et al. [17] described associated pathological lesions in 73% of cases with significant bone loss. These included superior and posterior labral tears, loose bodies, rotator-cuff tears, and chondromalacia. The authors recommended arthroscopic evaluation prior to the surgical Latarjet procedure to treat these associated lesions. Bhatia and DasGupta [18] reported an 11% incidence of humeral avulsion of glenohumeral ligaments (HAGL) lesions in association with significant glenoid bone loss, and described a dual-window subscapularis-sparing approach to perform a combined Latarjet procedure and HAGL repair. Bernhardson et al. [19] have evaluated the association of an anterior labroligamentous periosteal sleeve avulsion (ALPSA) and glenoid bone loss; they found that “patients with anterior shoulder instability who have an ALPSA lesion have nearly twice the amount of glenoid bone loss as those with a standard Bankart tear (no ALPSA lesion)”.

## 7.4 Diagnosis

Significant bone loss should be suspected and evaluated in every athlete who presents with anterior shoulder instability. Clinical indicators of significant bone loss may be identified on history and physical examination, including; (1) frequent and easy dislocations, (2) dislocations in sleep, (3) high-energy traumatic dislocation, (4) failed previous stabilisation procedure, and (5) a positive “bony apprehension” test [20, 21].

Presence of associated lesions should be assessed on clinical examination, and tests for rotator cuff integrity, concomitant posterior instability, SLAP and biceps lesions, and acromioclavicular joint pathology should be performed.

Meticulous imaging is necessary to determine the extent of the labral tear and to quantify humeral and glenoid bone loss. Radiographic views (Table 7.1) that are useful in instability include; (1) a true anteroposterior view, (2) Garth view, (3) Bernageau view, and (4) Anteroposterior view with shoulder in external rotation [22, 23]

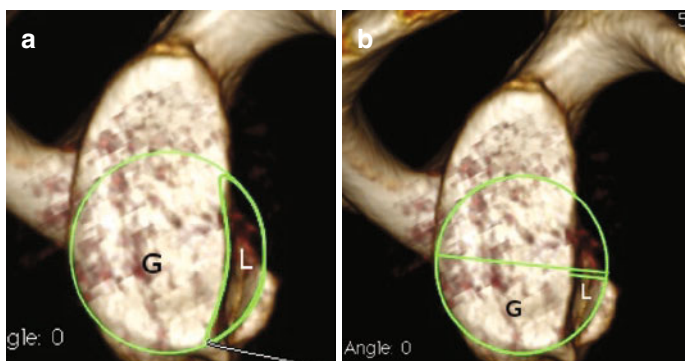
Magnetic resonance imaging and MR arthrography show the bone lesion and associated soft tissue pathology (Labral tears, HAGL lesions, rotator-cuff tears, bone bruising, chondral lesions). Bone loss may also be quantified accurately by MRI, both plain

**Table 7.1** Common radiographic views to assess glenoid and humeral bone loss

Radiographic view	Bone loss indicator
True anteroposterior view (Grashey view)	Positive LSGL sign ( <i>LSGL</i> loss of anterior sclerotic glenoid line)
Apical oblique-45° caudal tilt view (Garth view)	Anteroinferior bone fragment visualised
Bernageau view	Inferior glenoid profile and Hill–Sachs visualised
Anteroposterior view with shoulder in external rotation	Significant Hill–Sachs visualised

**Table 7.2** Common glenoid bone loss radiological measurement techniques

Bone-loss measurement method	Technique
Unilateral circle method (Chuang et al., Sugaya et al.)	A best-fit circle is drawn on 3D reconstruction of the inferior glenoid, and the defect is measured linearly (mm) or as area loss (mm <sup>2</sup> )
Bilateral Circle method (Pico Method, Baudi et al.)	Best-fit circle is drawn on inferior portion of the opposite normal glenoid and its surface area is digitally calculated This circle is superimposed onto the pathological glenoid, and surface area of defect is calculated
Bare area method (Sugaya et al.)	Bare area is approximated on computed tomography with use of intersecting lines, and distances are measured from bare area to anterior and posterior glenoid edges

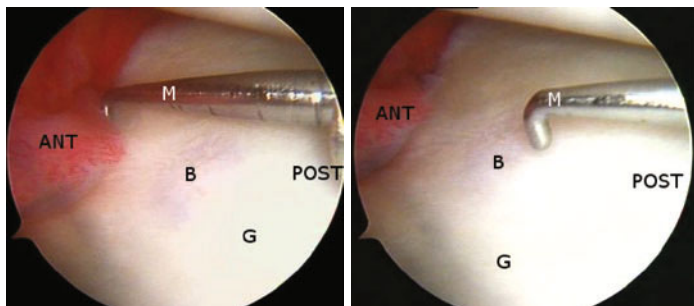


**Fig. 7.2** Measurement of glenoid bone loss on 3D CT images using the circle method is shown. (a) shows the area loss, (b) shows the linear length loss of glenoid bone

and arthrographic [24]. CT scans are indicated for 3D reconstruction analysis of bone loss using one of several methods (Table 7.2) that have been described [2, 25–28] (Fig. 7.2)

Arthroscopic suspicion of glenoid bone loss is based on visualisation of an “inverted-pear” glenoid, and dynamic “air-arthroscopy” is useful to assess engagement in abduction and





**Fig. 7.3** Arthroscopic evaluation of bone loss is demonstrated. The probe (M) first measures the linear distance between the anterior glenoid rim (ANT) and bare spot (B). This distance is then subtracted from the linear distance between the posterior glenoid rim (POST) and the bare spot, and this represents the glenoid bone loss

external rotation. Bone loss can be quantified with direct measurement using the bare spot as a reference [28, 29] (Fig. 7.3).

## 7.5 Management

Surgical treatment is recommended to treat bony instability, and the goal is to return the player to preinjury levels of overhead and contact sports. The decision making algorithm is broadly based on a combination of radiological quantification of bone loss, and the ISIS scoring system (Instability Severity Index Score) [29, 30]. Surgical techniques focus on preventing engagement of the humeral defect over the glenoid defect, and attempt to restore the glenoid track to normal (Table 7.3). This can be achieved using soft-tissue reconstruction (arthroscopic labroplasty  $\pm$  remplissage), or by reconstructing the incomplete glenoid arc (Latarjet procedure, Iliac crest bone graft) [29, 31–40] (Figs. 7.4 and 7.5).

Di Giacomo et al. [16] have developed a treatment paradigm with specific surgical criteria for all patients with anterior instability, both with and without bipolar bone loss. They suggested that “off track” engaging lesions with glenoid loss of  $<25\%$  could be

**Table 7.3** Surgical procedures used by the authors to treat bony instability in athletes

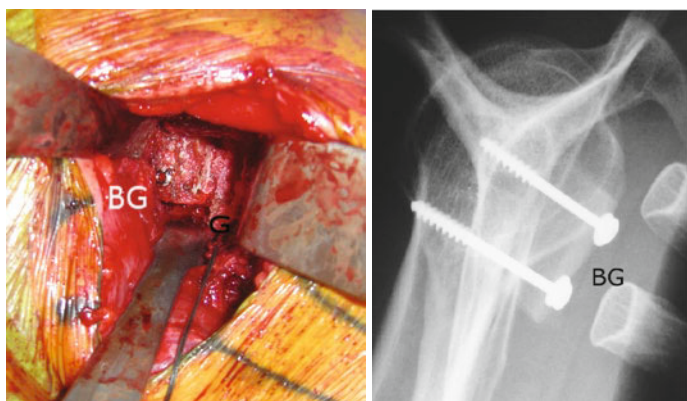
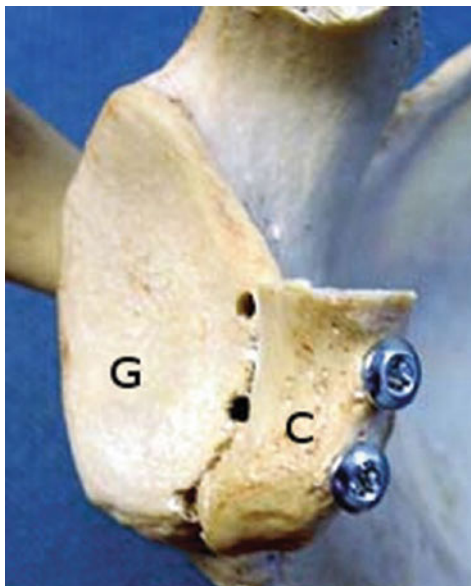
Surgical techniques	Description
Labroplasty [29]	Sequential tensioning of the capsulolabral complex to recreate a labral bump at the anterior glenoid rim
Remplissage [31, 32]	Capsulotenodesis of infraspinatus and posterior capsule into the Hill-Sachs defect
Mini-open Latarjet procedure [33]	Coracoid process transfer along with the conjoint tendon to the anterior glenoid rim using a mini-open subscapularis split approach
Mini-open congruent arc Latarjet procedure [1, 34]	Coracoid process transfer along with the conjoint tendon to the anterior glenoid rim using a mini-open subscapularis split approach. The coracoid block is “flipped” to orient the inferior coracoid surface along the articular glenoid surface, and a capsular shift is added
Arthroscopic Latarjet [35]	Coracoid process transfer along with the conjoint tendon to the anterior glenoid rim using an arthroscopic approach
Arthroscopic Latarjet and Capsular Shift [36]	Coracoid process transfer along with the conjoint tendon to the anterior glenoid rim and capsular shift using an arthroscopic approach
Arthroscopic/open bone grafting [37, 38]	Bone grafting procedure using autograft iliac bone or osteochondral allograft (distal tibia)

treated with a combined arthroscopic labral reconstruction and remplissage procedure; similarly, “off track” engaging lesions with glenoid loss of >25% could be treated with a Latarjet procedure. Additional associated lesions may be evaluated and treated with prior arthroscopy or with combined mini-open exposures [17, 41].

## 7.6 Outcomes

Burkhart and De Beer [1] first reported the results of arthroscopic Bankart repair in 194 consecutive shoulders (101 contact athletes). They found a 67% recurrence rate in shoul-

**Fig. 7.4** The coracoid transfer to the anterior glenoid rim (congruent arc Latarjet) is demonstrated on a bone model



**Fig. 7.5** An iliac crest bone grafting procedure is demonstrated. The radiograph shows the position of the block and fixation with 2 screws

ders with significant bone loss, and only a 4% recurrence rate in shoulders without significant bone loss. For contact athletes without significant bone defects, there was a 6.5% recurrence rate, whereas for contact athletes with significant bone defects, there was an 89% recurrence rate. The authors concluded that (a) In the absence of significant bone loss, arthroscopic Bankart repairs give results equal to open Bankart repairs, (b) Patients with significant bone deficits are not candidates for arthroscopic Bankart repair, (c) Contact athletes without structural bone defects may be treated by arthroscopic Bankart repair. However, contact athletes with bone deficiency require bone grafting procedures, and (d) The Latarjet procedure should be considered for patients with significant glenoid bone loss.

Burkhart et al. [42] analysed the results of the modified Latarjet procedure for 102 cases of shoulder instability associated with glenoid bone loss of 25% or more, or an engaging Hill–Sachs lesion. They reported a 4.9% recurrence rate in that same category of patient at a mean follow-up of 59 months, thereby validating their earlier recommendation of using the Latarjet procedure to reconstruct anterior glenoid bone loss.

De Beer et al. [43] recently reported an 89% return to previous sporting levels, and no recurrent instability episode after undergoing a congruent-arc Latarjet procedure. Additionally, the overall complication rate in the series was reported as 7%.

Arthroscopic soft tissue repairs (Bankart repair + remplissage) have been evaluated by Wolf and Arianjam [31]; in 55 patients with engaging Hill–Sachs lesions and glenoid bone loss <25%, they reported a 4.4% recurrent instability at an average follow-up of 58 months.

Kraus et al. [44] reported good to excellent early clinical results with an arthroscopic anatomic glenoid reconstruction using an all-arthroscopic, autologous tricortical iliac crest bone grafting technique in anterior instability patients.

Dumont et al. [35] reported a 1.6% recurrent instability rate and good WOSI score outcomes after arthroscopic Latarjet procedure at a minimum follow-up period of 5 years.

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## Q&A

- (1) What is the incidence of bone loss on the glenoid and humeral head?

Glenoid bone loss of varying severity occurs in 90% of the glenoids. Humeral head defects (Hill–Sachs lesions) occur in 65–93% of anterior instability cases

- (2) Which sports are associated with bony instability in athletes?

Aggressive, full-contact and overhead sports (rugby, wrestling, judo, mixed martial arts, bodybuilding, weightlifting) result in high-energy traumatic dislocations, and usually have a high incidence of significant bone defects. Other conditions that are associated with significant glenoid defects include post-concussion seizures, vehicular sports (motocross and downhill mountain biking), and recreational gym injuries.

- (3) Name five clinical “warning signs” of significant bone loss in instability.

(1) Frequent and easy dislocations, (2) Dislocations in sleep, (3) High energy traumatic dislocation, (4) Failed previous stabilization procedure, and (5) a positive “bony apprehension” test

- (4) Name 4 radiographic views used to evaluate bony instability.

(1) Grashey view, (2) Garth view, (3) Bernageau view, (4) True AP external rotation view.

- (5) What is the indication for a soft tissue repair procedure?

On-track Hill–Sachs (nonengaging) or Off-track Hill–Sachs (engaging) + <25% glenoid bone loss

- (6) What is the indication for a bone reconstruction procedure?

Off-track Hill–Sachs (engaging) + >25% glenoid bone loss

## References

1. 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.
2. Sugaya H, Moriishi J, Dohi M, Kon Y, Tsuchiya A. Glenoid rim morphology in recurrent anterior glenohumeral instability. *J Bone Joint Surg Am*. 2003;85-A(5):878–84.
3. De Beer J, Bhatia DN. Shoulder injuries in rugby players. *Int J Shoulder Surg*. 2009;3(1):1–3.
4. 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.
5. Saito H, Itoi E, Sugaya H, Minagawa H, Yamamoto N, Tuoheti Y. Location of the glenoid defect in shoulders with recurrent anterior dislocation. *Am J Sports Med*. 2005;33(6):889–93.
6. Itoi E, Yamamoto N, Kurokawa D, Sano H. Bone loss in anterior instability. *Curr Rev Musculoskelet Med*. 2013;6(1):88–94.
7. Yamamoto N, Itoi E, Abe H, Minagawa H, Seki N, Shimada Y, Okada K. Contact between the glenoid and the humeral head in abduction, external rotation, and horizontal extension: a new concept of glenoid track. *J Shoulder Elb Surg*. 2007;16(5):649–56.
8. Bigliani LU, Newton PM, Steinmann SP, Connor PM, McIlveen SJ. Glenoid rim lesions associated with recurrent anterior dislocation of the shoulder. *Am J Sports Med*. 1998;26(1):41–5.
9. Spatschil A, Landsiedl F, Anderl W, et al. Posttraumatic anteriorinferior instability of the shoulder: arthroscopic findings and clinical correlations. *Arch Orthop Trauma Surg*. 2006;126(4):217–22.
10. Yiannakopoulos CK, Mataragas E, Antonogiannakis E. A comparison of the spectrum of intra-articular lesions in acute and chronic anterior shoulder instability. *Arthroscopy*. 2007;23(9):985–90.
11. Buhler M, Gerber C. Shoulder instability related to epileptic seizures. *J Shoulder Elb Surg*. 2002;11:339–44.
12. Hardy P. Bony lesions influence on the result of the arthroscopic treatment of gleno-humeral instability. Symposium: shoulder instability limits of arthroscopic surgery: bone deficiency, shrink- age, acute instability. Read at 5th International Society of Arthroscopy, Knee Surgery and Orthopaedic Sports Medicine Congress, Auckland, March 10–14, 2003.
13. Miniaci A, Berlet G. Recurrent anterior instability following failed surgical repair: allograft reconstruction of large humeral head defects. *J Bone Joint Surg Br*. 2001;83(Suppl 1):19–20.

14. Voos JE, Livermore RW, Feeley BT, et al. Prospective evaluation of arthroscopic Bankart repairs for anterior instability. *Am J Sports Med.* 2010;38(2):302–7.
15. Burkhart SS, De Beer JF, Tehrany AM, Parten PM. Quantifying glenoid bone loss arthroscopically in shoulder instability. *Arthroscopy.* 2002;18(5):488–91.
16. Di Giacomo G, Itoi E, Burkhart SS. Evolving concept of bipolar bone loss and the Hill-Sachs lesion: from “engaging/non-engaging” lesion to “on-track/off-track” lesion. *Arthroscopy.* 2014;30(1):90–8.
17. Arrigoni P, Huberty D, Brady PC, Weber IC, Burkhart SS. The value of arthroscopy before an open modified Latarjet reconstruction. *Arthroscopy.* 2008;24(5):514–9.
18. Bhatia DN, DasGupta B. Surgical treatment of significant glenoid bone defects and associated humeral avulsions of glenohumeral ligament (HAGL) lesions in anterior shoulder instability. *Knee Surg Sports Traumatol Arthrosc.* 2013;21(7):1603–9.
19. Bernhardson AS, Bailey JR, Solomon DJ, Stanley M, Provencher MT. Glenoid bone loss in the setting of an anterior labroligamentous periosteal sleeve avulsion tear. *Am J Sports Med.* 2014;42(9):2136–40.
20. Bushnell BD, Creighton RA, Herring MM. Bony instability of the shoulder. *Arthroscopy.* 2008;24(9):1061–73.
21. Bushnell BD, Creighton RA, Herring MM. The bony apprehension test for instability of the shoulder: a prospective pilot analysis. *Arthroscopy.* 2008;24(9):974–82.
22. Edwards TB, Boulahia A, Walch G. Radiographic analysis of bone defects in chronic anterior shoulder instability. *Arthroscopy.* 2003;19(7):732–9.
23. Jankauskas L, Rüdiger HA, Pfirrmann CW, Jost B, Gerber C. Loss of the sclerotic line of the glenoid on anteroposterior radiographs of the shoulder: a diagnostic sign for an osseous defect of the anterior glenoid rim. *J Shoulder Elb Surg.* 2010;19(1):151–6.
24. Markenstein JE, Jaspars KC, van der Hulst VP, Willems WJ. The quantification of glenoid bone loss in anterior shoulder instability; MR-arthro compared to 3D-CT. *Skelet Radiol.* 2014;43(4):475–83.
25. Baudi P, Righi P, Bolognesi D, Rivetta S, Rossi Urtoler E, Guicciardi N, Carrara M. How to identify and calculate glenoid bone deficit. *Chir Organi Mov.* 2005;90:145–52.
26. Chuang TY, Adams CR, Burkhart SS. Use of preoperative three-dimensional computed tomography to quantify glenoid bone loss in shoulder instability. *Arthroscopy.* 2008;24:376–82.
27. Magarelli N, Milano G, Sergio P, Santagada DA, Fabbriani C, Bonomo L. Intra-observer and interobserver reliability of the ‘Pico’ computed tomography method for quantification of glenoid bone defect in anterior shoulder instability. *Skelet Radiol.* 2009;38(11):1071–5.

28. Sugaya H, Kon Y, Tsuchiya A. Arthroscopic repair of glenoid fractures using suture anchors. *Arthroscopy*. 2005;21:635.
29. Bhatia DN, De Beer JF. Management of anterior shoulder instability without bone loss: arthroscopic and mini-open techniques. *Should Elb*. 2011;3:1–7.
30. 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(11):1470–7.
31. Wolf EM, Arianjam A. Hill-Sachs remplissage, an arthroscopic solution for the engaging Hill-Sachs lesion: 2- to 10-year follow-up and incidence of recurrence. *J Shoulder Elb Surg*. 2014;23(6):814–20.
32. Bhatia DN. Double-barrel remplissage: an Arthroscopic all–intra-articular technique using the double-barrel knot for anterior shoulder instability. *Arthrosc Tech*. 2015;4(1):e65–70. <https://doi.org/10.1016/j.eats.2014.11.006>.
33. Young AA, Maia R, Berhouet J, Walch G. Open Latarjet procedure for management of bone loss in anterior instability of the glenohumeral joint. *J Shoulder Elb Surg*. 2011;20(2 Suppl):S61–9.
34. De Beer JF, Roberts C. Glenoid bone defects--open Latarjet with congruent arc modification. *Orthop Clin North Am*. 2010;41(3):407–15.
35. Dumont GD, Fogerty S, Rosso C, Lafosse L. The arthroscopic Latarjet procedure for anterior shoulder instability: 5-year minimum follow-up. *Am J Sports Med*. 2014;42(11):2560–6.
36. Bhatia DN. Arthroscopic Latarjet and capsular shift (ALCS) procedure: a new “freehand” technique for anterior shoulder instability associated with significant bone defects. *Tech Hand Up Extrem Surg*. 2014;19(1):11–7.
37. Haaker RG, Eickhoff U, Klammer HL. Intraarticular autogenous bone grafting in recurrent shoulder dislocations. *Mil Med*. 1993;158:164–9.
38. Taverna E, D’Ambrosi R, Perfetti C, Garavaglia G. Arthroscopic bone graft procedure for anterior inferior glenohumeral instability. *Arthrosc Tech*. 2014;3(6):e653–60.
39. 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. *Arthroscopy*. 2009;25:446–52.
40. Warner JJ, 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.
41. Bhatia DN. Dual-window subscapularis-sparing approach: a new surgical technique for combined reconstruction of a glenoid bone defect or Bankart lesion associated with a HAGL lesion in anterior shoulder instability. *Tech Hand Up Extrem Surg*. 2012;16(1):30–6.



42. Burkhart SS, De Beer JF, Barth JR, Cresswell T, Roberts C, Richards DP. Results of modified Latarjet reconstruction in patients with anteroinferior instability and significant bone loss. *Arthroscopy*. 2007;23(10):1033–41.
43. De Beer JF, et al. Evaluation of functional outcomes and complications following modified Latarjet reconstruction in athletes with anterior shoulder instability. *Should Elb*. 2015;7(3):168–73.
44. Kraus N, Amphansap T, Gerhardt C, Scheibel M. Arthroscopic anatomic glenoid reconstruction using an autologous iliac crest bone grafting technique. *J Shoulder Elb Surg*. 2014;23(11):1700–8.



# Acromioclavicular Joint Injuries

# 8

Lennard Funk and Mohamed A. Imam

## Key Learning Points

- Injuries to the ACJ are common in many different sports
- The classifications have poor inter- and intra-observer agreement. Therefore, surgical decisions should not be made on the grade of injury alone.
- Most injuries can be managed conservatively, except in overhead athletes
- Surgical reconstruction should be anatomical, biological and strong enough to allow early rehabilitation
- Good outcomes, with early return to sports can be achieved in most cases with low complication rates.

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## 8.1 Introduction

Acromioclavicular joint (ACJ) injuries are very common sports injuries with both acute and chronic presentations. This high rate correlates with the more than 100 reported surgical techniques described for management. The anatomy and configuration of the ACJ makes it a resilient joint that can resist a significant amount of force before disruption, yet the ACJ remains one of few major joints in the body, where dislocations are often managed without intervention. In this chapter, we aim to review the anatomy and biomechanics of the ACJ, as well as the assessment, diagnosis, and different treatment protocols including our preferred protocol for various ACJ injuries.

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## 8.2 Anatomy and Biomechanics

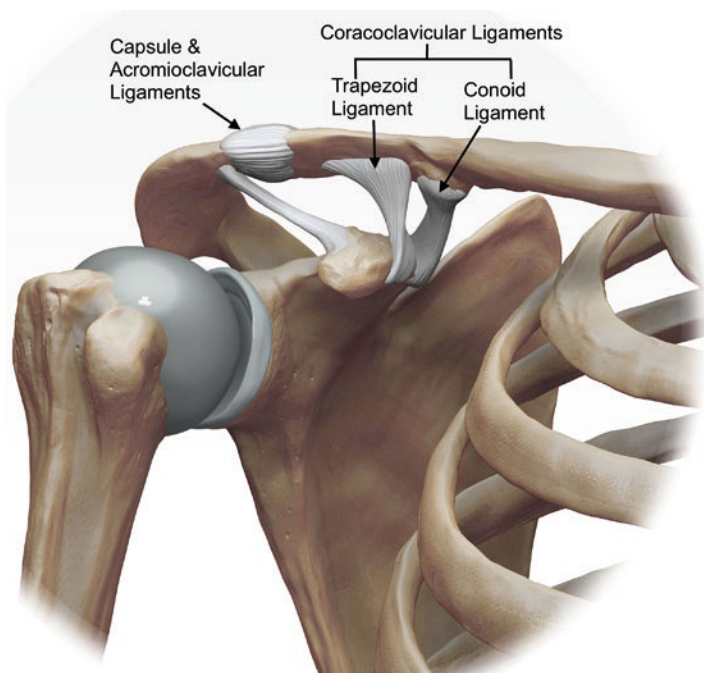
For the optimal management of ACJ injuries, it is imperative to be aware of the anatomy and biomechanics of the joint, so that basic principles of management can be applied. An understanding of these basic principles will allow the treating health care provider to evaluate various clinical presentations and apply bespoke treatment for the specific patient disorders and needs.

The ACJ is a diarthrodial, robust, synovial articulation that attaches the clavicle to the scapula. There is an intraarticular fibrocartilagenous disc. There are two types of discs: complete and partial (meniscoid). The disc undergoes rapid degeneration until it is essentially no longer functional beyond the fourth-decade [1–4]. The ACJ anatomy and supporting ligamentous structures have been described historically by Urist in 1946 [5], followed by DePalma in 1963 [6].

The stabilizers of the ACJ are categorized into dynamic and static stabilizers [7, 8]. The dynamic stabilizers include the muscles that cross the joint (Deltoid and Trapezius), providing dynamic suspensory support. The static stabilizers include: the acromioclavicular (AC) ligaments, coracoclavicular (CC) ligaments, deltotrapezial fascia and capsule (Table 8.1 and Fig. 8.1).

**Table 8.1** Acromioclavicular joint stabilizers

	Components	Direction of stability
Acromioclavicular ligament	(Superior, inferior, anterior, and posterior components). Superior ligament is strongest, followed by posterior	Horizontal stability
Coracoclavicular ligaments	Trapezoid and conoid ligaments	Vertical stability
	Trapezoid ligament inserts <2 cm from lateral end of clavicle	Horizontal and vertical stability
	Conoid ligament inserts 3.2 cm from the lateral end of clavicle at the posterior border	Vertical stability
Others	Deltotrapezial fascia, capsule	Horizontal and vertical stability

**Fig. 8.1** Static stabilisers of the acromioclavicular joint (courtesy of Lennard Funk, <http://www.shoulderdoc.co.uk>)

The importance of the dynamic stabilizers increases in the presence of torn ligaments.

The CC ligament complex is made up of the conoid and trapezoid ligaments. These are extremely strong, with a tensile strength of over 800 N. The anatomical landmarks and isometric points of the CC ligaments on the undersurface of the clavicle have been well described [9–11]. The trapezoid is at a mean of 14.7 mm and the conoid is a mean of 32.1 mm from the distal end of the clavicle. The origin of the CC ligaments varies between genders while keeping the same ratio of the origin to clavicular length [11].

Biomechanical studies have demonstrated that the ACJ capsule and ligaments act as a primary constraint for posterior displacement and posterior axial rotation of the clavicle [4]. Additional studies have confirmed that the ACJ ligaments and capsule provide the majority of anterior-posterior (Horizontal) stability whereas the CC ligaments provide a large percentage of superior-inferior (vertical) stability [5]. The trapezoid ligament is the primary restraint to axial loading of the ACJ. The ACJ ligaments are responsible of 90% the resistance to posterior loading forces, but also provide some vertical and rotational stability. An injury to any one structure does not specifically predict the type or direction of instability. Injuries of the static restraints does not occur in isolation, so single directional instability is unlikely. This highlights the complex three dimensional stability and importance of anatomic reconstruction of both the ACJ and CC ligaments. Fukuda et al. [4] affirmed that “if maximum strength of healing after an injury to the AC joint is the goal, all ligaments should be allowed to participate in the healing process.”

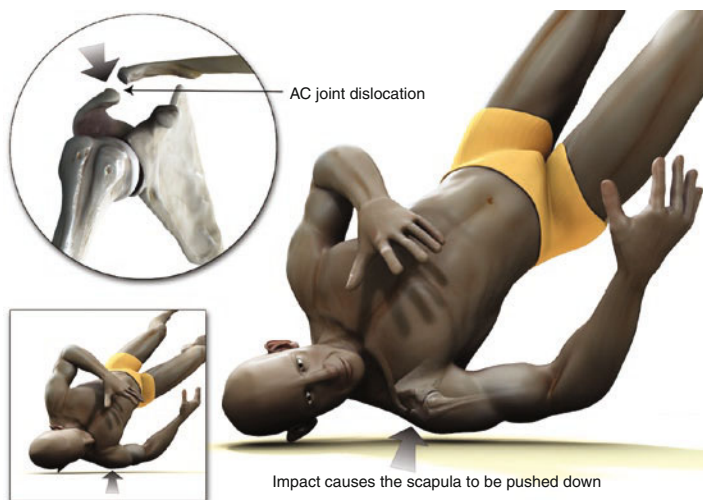
The ACJ acts as a pivot between the clavicle and the scapula, which allows a complex motion pattern that is not fully understood. The clavicle rotates 40–50° posteriorly with shoulder elevation with 8° of rotation through AC joint. The remainder is from scapula rotation and sternoclavicular motion. There is 5–8° of rotation, in line with the scapula, observed in the ACJ with forward elevation and abduction to 180° [3, 12–14]. Therefore, disruption of the ACJ is recognized as a scapula disorder [15].

Codman described the motion of the ACJ nicely as: “I have come to the conclusion, that the acromioclavicular joint moves very little indeed, but this motion may occur in many different planes. Its surfaces slide a little, rotate a little, tip apart a little and act like hinges to some degree” [16]. He highlighted the importance of having an intact ACJ for scapula motion to be synchronously coupled with arm motion by the clavicle; the CC ligaments guide this coupled motion. Subsequently, the ACJ should not be fixed, either by fusion (screws, plates, pins) or by CC screws. If these implants are used, motion will be lost, limiting shoulder function, or the hardware may eventually fail because of the obligatory coupling of clavicle rotation with scapula motion and arm elevation. This correlates with Gumina et al. [15], who found that longstanding type III ACJ injuries led to scapula dyskinesia in 71% of patients. Fifty-eighty percent had SICK scapula syndrome (Scapula malposition, Inferior medial border prominence, Coracoid pain and malposition, and dyskinesia of scapula movement). The authors proposed that dyskinesia is due to loss of function of the ACJ which is no longer a stable fulcrum of the shoulder girdle.

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### 8.3 Mechanism of Injury

The ACJ is a subcutaneous joint without a large sleeve of muscle protection. It is more prone to injury because the sternoclavicular joint is very stable. Direct and indirect trauma can cause ACJ injuries. Most ACJ disruptions are caused by direct trauma after a fall onto the point of the shoulder with the arm in the adducted position so that the acromion/scapula is forced inferiorly and anteriorly (Fig. 8.2). The clavicle is thus relatively displaced superiorly. As originally described by Codman, the AC ligaments and CC ligaments are sequentially torn, with damage to the ACJ, disruption of the delto-trapezial fascia or trapezius muscle. Indirect injury can also occur from falling on an adducted outstretched hand, pushing the humerus superiorly into the acromion [17].



**Fig. 8.2** Commonest mechanism of acromioclavicular joint dislocation, with a fall onto the point of the shoulder. The scapula is forced inferior and anterior, relatively displacing the clavicle postern-superiorly (courtesy of Lennard Funk, <http://www.shoulderdoc.co.uk>)

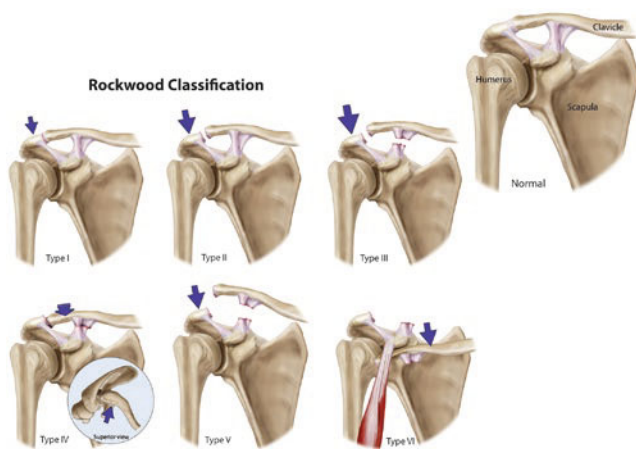
## 8.4 Classification of ACJ Injuries

In 1917, Cadenat [18] originally described the mechanisms of ACJ dislocation and the classical features of its presentation. He explained a sequential injury beginning with the AC ligaments disruption, progressing to the CC ligaments failure, and finally involving the deltoid and trapezial muscles and fascia. This formed the basis for future classifications.

The most commonly used classification is the Allman and Tossy classification [19, 20] who classified ACJ injuries into type I, II and III. In 1998, Rockwood's proposed a modification to this classification by adding grades IV, V and VI to complete the classification currently widely used globally. The grades essentially escalate with increasing soft tissue disruption about the AC joint (Table 8.2) (Fig. 8.3).

**Table 8.2** Rockwood classification of acromioclavicular joint injuries

Rockwood classification of ACJ injuries into six grades	
I	AC joint sprain
II	Subluxated ACJ with intact CC ligaments
III	Dislocated ACJ with disrupted CC ligaments
IV	Superiorly and posteriorly dislocated ACJ
V	Dislocated ACJ with 100–300% separation
VI	ACJ dislocated and Inferiorly displaced under the coracoid

**Fig. 8.3** Rockwood classification of acromioclavicular joint injuries (courtesy of Lennard Funk, <http://www.shoulderdoc.co.uk>)

Most physicians use radiographs to classify ACJ dislocations, but this has been proven to be an unreliable method for determining the pathological classification [21]. Visual assessment of radiographs is not reliable although bilateral panoramic digital comparative measurements are more accurate in determining the degree of vertical displacement [22]. Even with the addition of 3D CT scanning, the inter- and intra-observer reliability of the classification systems are poor [23]. The addition of MRI scans to the clinical and radiographic findings, may improve the accuracy [24].



In 2014 a consensus document was published by the ISAKOS Upper Limb Committee to diversify the Rockwood classification [25]. The group recognized the primary problem was a scapula disorder and focused on ACJ instability. They recognized that there was a lack of information to adequately identify the factors that made a patient more suitable for surgical intervention. Based on their combined experience and expertise, they subdivided the contentious Type III injuries into Type IIIA and Type IIIB, with IIIA being functional stable and IIIB functional unstable, when reviewed 3–6 weeks post-injury. The ‘stability’ was based on a number of clinical factors, comprising: ongoing pain (usually on the anterior acromion, rotator cuff, and medial scapular area), weakness during rotator cuff testing, decreased flexion and abduction range of motion, and demonstrable scapular dyskinesis on observation. Special radiographic views (i.e. cross-body stress view) may provide some objective information.

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## 8.5 Clinical Examination

Complete ACJ disruptions (Grade III-IV) are clinically obvious, with a classical deformity (Fig. 8.4). These may be ‘locked’ and not reducible as the clavicle overrides the acromion. A ‘locked’ dislocation may not allow full scapula excursion which leads to a variable limitation of elevation (Fig. 8.5). If type III, then these are likely to become type IIIB dislocations. However, some complete dislocations may be unstable but be easily reducible. We call these a ‘shocked’ dislocation, or type IIIA on the ISAKOS classification. Both type V and type III dislocations behave in this way. We therefore prefer to describe the injuries biomechanically as ‘locked’ and ‘shocked’ as this has more functional predictability and effect on management than the degree of vertical translation alone.

Other disruptions are less obvious and don’t fit the standard classifications (Grade II–II). They can clinically be diagnosed by asking the patient to adduct their arm across their chest.



**Fig. 8.4** Dropped right scapula and arm, with a complete acromioclavicular joint dislocation. Note that the clavicles remain in the same horizontal plane



**Fig. 8.5** Limited elevation of left shoulder due to scapula 'locked' under clavicle, thus limiting scapula and glenohumeral elevation

This manoeuvre accentuates the injury, displacing the clavicle superiorly and posteriorly. Standard radiographs may be normal, but the deformity can be demonstrated on adduction radiographs.

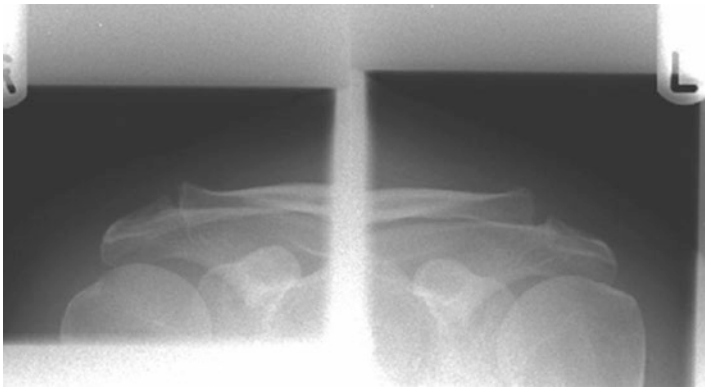
The lateral clavicle should also be assessed for both vertical and horizontal laxity and compared to the opposite (normal) side. This is done by direct manual palpation. Often excess laxity after injury is indicative of a significant Grade II injury and suggests instability without a true dislocation.

ACJ injury is identified by a triad of point tenderness, ACJ pain with cross-arm adduction, and pain relief by local injection of an anesthetic agent [17]. Walton et al. [26] described using the Paxinos test (thumb pressure at the posterior ACJ).

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## 8.6 Radiographic Evaluation

Although standard views taken for the shoulder can be helpful, the ACJ is not central and will often be over-penetrated (dark). Subsequently subtle lesions may be overlooked. The Zanca view is the most accurate view to assess the AC joint. This is performed 15° cephalad in the A-P plane with 50% of the standard penetration strength (Fig. 8.6). Nonetheless, it has not been shown to have good inter- or intra-observer reliability. An axillary view has been proposed to exclude posterior displacement, to differentiate grade III from IV,



**Fig. 8.6** Comparative Zanca views showing the subluxation of the left acromioclavicular joint

but this also has poor reliability. Weight-bearing views have not been proven to have good reliability either [27]. CT scan is the best form of imaging to appreciate the static bony displacement, however, clinical assessment is probably just as reliable. MRI can be useful to assess the soft tissue damage in the acute scenario. A Stryker notch view is also helpful if a coracoid fracture is suspected (normal CC distance with complete disruption of the ACJ). Bosworth [28] reported an average CC distance between 1.1 to 1.3 cm. An increase in the CC distance of more than 25% compared with the opposite normal side, indicates complete CC ligament injury [29, 30].

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## 8.7 Treatment

Traditional teaching for ACJ injury treatment has been to surgically repair type IV and V dislocations acutely, whilst managing type I and II non-operatively. Type III has been contentious. However, as we have seen above, classifying the injuries can be difficult with poor agreement. Recent evidence supports initial primary nonoperative treatment of complete ACJ dislocations. A review of 1172 patients reported successful outcomes in 88% of patients treated non operatively [31]. There was no difference when compared with an equivalent group managed operatively.

The ISAKOS Upper Limb Committee consensus approach to type III injuries is to reassess clinically at 3–6 weeks post-injury, which is a sensible approach since many patients will have improved by that time. The decision for surgery in the ISAKOS consensus article is then based on ‘overriding of the distal clavicle’ on cross body adduction radiographs. Unfortunately, there is no good evidence for this and the group admit that studies will be needed to support this consensus approach.

We therefore do not use the grade of ACJ injury as the primary determinant for defining treating options, given the poor inter/intra-observer reliability of the injury grades and no good correlation with clinical symptoms [21]. Many patients do well without surgery [25]. Surgical reconstruction should be reserved for those patients with high functional demands, symptomatic

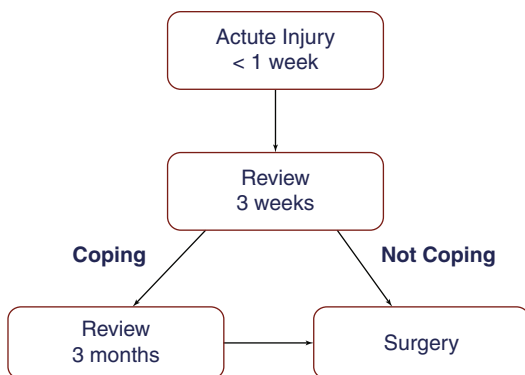
locked, or unstable (shocked) scapula, and failure to improve in the first 3–6 weeks. The patient's symptoms and early response to non-operative symptomatic management primarily defines the indication to offer surgery.

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## 8.8 Treatment Protocol

Acute injury (<1 week):

- Assess and diagnose.
- Sling for comfort only, analgesia and rehabilitation with early active mobilization as comfortable
- Surgery indicated if: Clearly in agony with clavicle button-holed through trapezius; Overhead athlete; Neurovascular injury; Open injury
- 3 week review:
  - Settling and improving—continue symptomatic management and gradually return to sports and manual activities. Arrange review at 3 months.
  - Not coping—offer early surgical reconstruction
- 3 month review:
  - Returned to sports and little symptoms—discharge
  - Not coping—offer surgical stabilization



## 8.9 Treatment

### 8.9.1 Nonoperative Treatment

Non-operative treatment of acute injuries includes simple analgesia, topical ice therapy and rest in a sling for comfort. The use of a supportive broad arm sling is preferable to a collar and cuff because it supports the elbow and supports the weight of the shoulder. It is recommended that the sling be discarded once the symptoms settle, usually within one week. Physiotherapy focuses on dynamic scapula stabilization and activity-specific rehabilitation. Contact sports and heavy lifting can be started as comfortable, usually about 6–12 weeks post-injury. Local discomfort may be felt with activity for up to 6 months. The literature reports that, at 1 year, there is a 17% chance of reduced bench press strength, although 80% of those patients do not find that a problem.

The results of non-operative treatment of grade III injuries has been variable in the literature. Tibone et al. found no significant difference in strength in patients with type III injuries, when treated nonoperatively versus operatively, at 2 years follow-up [32]. However, Schlegel demonstrated a 20% chance of suboptimal outcome with nonoperative treatment [33]. Types I and II injuries also have a 25% chance of requiring surgery by 2 years post-operatively [34].

However, in athletes, Cox [35] showed that a large proportion of ACJ injuries remained symptomatic at 6 months post-injury (36% of Grade I, 48% of Grade II and 69% of Grade III injuries). Also, 30% of overhead athletes are unable to continue at the same level of sport, and 9% had to change sport. Patients performing strength training, in particular climbers, and had to reduce their activities [36]. In contact athletes, many are able to return to sport but struggle with many of the strength and overhead training exercises required to keep them at high level sport.

### 8.9.2 Non-operative Rehabilitation Protocol

In 1997, Gladstone et al. [37] published a four-phase rehabilitation protocol for non-operative management for athletes. This protocol is still used with some modifications (Table 8.3)

**Table 8.3** Non-operative staged progression rehabilitation [37]

Phase	Protocol
I	Pain control, immediate protective range of motion, and isometric exercises
II	Strengthening exercises using isotonic contractions
III	Unrestricted functional participation with the goal of increasing strength, power, endurance, and neuromuscular Control
IV	Return to activity with sport-specific functional drills

## 8.10 Surgical Treatment

The purported advantage of surgical intervention, consistently borne out in the literature, is the increased probability of anatomic restoration. However the current literature has been unable to demonstrate a correlation between anatomical correction and improvement in pain, strength, or motion [38]. The aim of surgical intervention is to achieve a painless, stable shoulder, with adequate mobility, strength and muscle control for the individual desired level of activity and participation.

Over 100 different surgical techniques have been published to treat acute and chronic ACJ injuries. This is an indication of the lack of consensus in the literature as to the optimal treatment strategy. The described techniques include primary repair of the CC ligaments, augmentation with autogenous tissue like coracoacromial (CA) ligament, augmentation with absorbable and nonabsorbable suture as well as synthetic material, and CC stabilization with metallic screws. Many techniques have been described as open, arthroscopic and varying combined approaches and each of the different techniques have been used insolation or in combination with one or more of the others.

## 8.11 Surgical Techniques

### 8.11.1 The Weaver-Dunn Technique

The Weaver-Dunn technique involves excision of the distal clavicle and transfer of the CA ligament. The technique has been used extensively to manage both acute and chronic ACJ injuries.

Several modifications have been described with varying published outcomes. However, concerns have been published on the increased risk of failure and recurrence after surgery [39, 40]. Biomechanically, the use of the CA ligament only reproduces 25% of the ultimate load of the original CC ligament complex. The risk of failure of the Weaver-Dunn technique and the modified versions led to the development of recent techniques and newer concepts in reconstruction of the CC ligament.

### **8.11.2 Distal Clavicle Resection Without Coracoclavicular Ligament Reconstruction**

Numerous reports of satisfactory outcomes following arthroscopic resection of the distal clavicle with combined results of 92% good or excellent [41] for ACJ degenerative pathology. However the results have been significantly worse for resection in the presence of instability [17, 42, 43].

### **8.11.3 Hook Plate**

Plate fixation of the lateral clavicle with a subacromial hook offers stable reduction of the ACJ. It acts as an indirect mechanism of reduction and does not directly reconstruct the CC ligament complex. Whilst the reduction is rigid the subacromial hook can lead to impingement of the rotator cuff and may be poorly tolerated by the patient. The plate requires secondary removal. Metanalyses have shown the hook plate to be less well tolerated with inferior outcomes compared to coracoclavicular ligament fixation [44].

### **8.11.4 Coracoclavicular Fixation: Suture, Cerclage Wire, Slings, Screw Fixation and Suture Button Constructs**

Bosworth introduced the concept of CC ligament repair in 1941; he referred to it as a screw suspension procedure, which he performed percutaneously. Over the years, technical failure rate in



32% of the cases [45] has been reported. The use of Polydioxanone (PDS) cerclage repair of the CC ligament has been used and reported extensively with problems reported including failed reduction, loss of fixation and irritation of soft tissues. Early post-operative complications were reported in 43%, 58 % and 17% and recurrent ACJ instability was seen in 32%, 50% and 24% of patients treated with a tension band, hookplate, and PDS cord, respectively [46].

Techniques were developed based on the biomechanical observations that augmentations around/through the clavicle and coracoid to improve load to failure and stiffness of the reconstructions [17]. In one biomechanical study [47], bicortical screw augmentation, between the coracoid and the clavicle, produced higher strength and comparable stiffness to that of the CC ligaments. However, this study did not evaluate neither cyclic loading nor anterior-posterior translation. In another study looking at superior, anterior and posterior translation after the usage augmentation with three different constructs: suture sling, coracoacromial ligament transfer and screws. The authors concluded that these three surgical procedures do not have the appropriate stiffness to restore the stability of the intact joint before healing.

Suture button constructs have increased in popularity over recent years as they offer increased strength than cerclage sutures and permit more rotation than screws. They are also readily amenable to arthroscopic insertion techniques. Initial series were presented with high rates of success but subsequent series reports increased failure rates with loss of reduction and suture failure [48, 49]. This has led to the development of stronger constructs using multiple buttons [50] and tape sutures although there are limited published series of their efficacy.

### **8.11.5 Free Graft Augmentation/Reconstruction of the CC Ligament Complex**

The use of free graft augmentation of the CC ligament is used to improve surgical results by adding a biological graft into the CC ligament reconstruction. The use of a free tendon graft (Gracilis

tendon, toe extensor, or semitendinosus graft) is placed in an anatomic position in order to reconstruct trapezoid and conoid ligaments. This has been shown to have an ultimate failure load that is equivalent to the native intact CC ligaments but graft fixation techniques remain problematic.

### **8.11.6 Loop Reconstruction Techniques**

These include loop-wire, Dacron loop, Vicryl tape (Ethicon, Johnson & Johnson, USA), Surgilig/Lockdown or LARS. The purported advantages include early postoperative functional treatment and earlier recovery. The main concern of these constructs was reported in the old designs; these include clavicular osteolysis or coracoid erosion because of the knitted structure of the implants. However, improved implant technology seem to have minimized these problems in the latest generation of artificial ligaments [51].

### **8.11.7 Acromioclavicular Ligament Repair**

AC ligament repair and reinforcement of the superior ligament and capsule to supplement the repair is usually used in combination with other CC ligament reconstructive procedures. Based on biomechanical observations reported earlier in this chapter, and our experience, this is recommended in all cases of ACJ stabilization.

### **8.11.8 Repair of the Deltotrapezial Fascia**

Numerous reports stressed the importance of the imbrication of the delto-trapezial fascia in any surgical procedure addressing the ACJ disruption [52, 53]. Many published unsatisfactory results could be due to lack of repair of the trapezius and deltoid [54]. As with repair and plication of the AC joint capsule, we recommend this as a supplementation of all ACJ stabilization procedures.

### **8.11.9 Dynamic Muscle Transfer**

Transfer of the short head of the biceps tendon either with or without the coracobrachialis with the coracoid tip has been described. However, there is a risk of nonunion or injury to the musculocutaneous nerve with transfer of coracoid. Other complications include, including coracoid fragmentation, infection and pain.

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### **8.12 Current Trends**

Current trends are towards anatomical restoration of the CC and AC ligaments with some form of biological enhancement. The trend is towards strong constructs that allow for early mobilization and less risk of redisplacement. This is usually achieved by passing the graft through the isometric points of the CC ligaments in the clavicle and either through a drill-hole in the coracoid or looped under the coracoid. This is combined with a repair or reconstruction of the AC ligaments and repair of the delto-trapezial fascia. These techniques seem to show the best results, with lower complications. With advancing technologies, these will continue to evolve and improve.

The ideal reconstructive technique should:

- Restore the anatomical CC and AC ligament constructs
- Be strong, but flexible to allow some motion in all planes at the ACJ
- Allow early rehabilitation.
- Biological
- Avoid donor site morbidity
- Avoid the need for routine removal

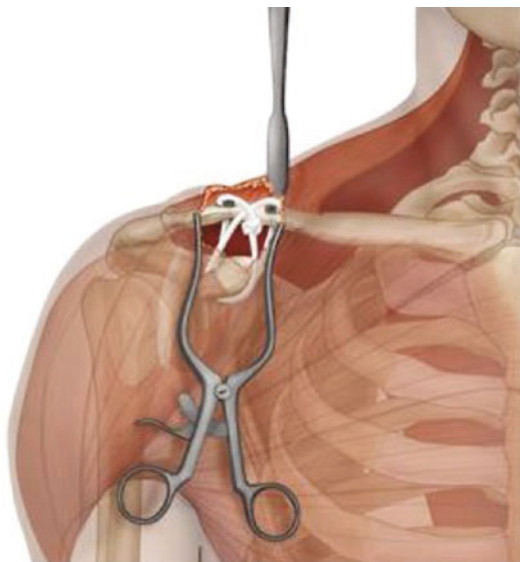
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### **8.13 Authors Preferred Technique**

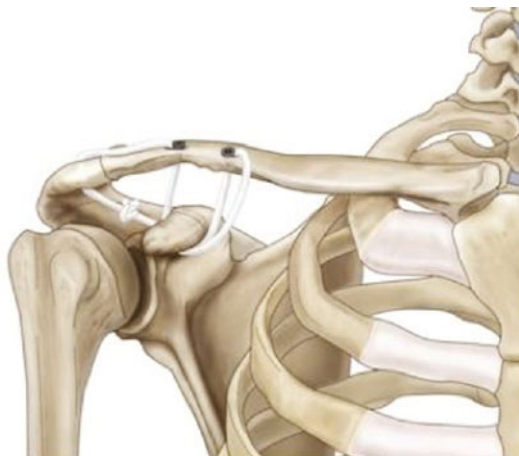
Based on the above criteria, our preferred method involves the use of a strong, washed, synthetic polyethylenetetraphthalate ligament, known as the Ligament Advancement Reinforcement

System (LARS Ligament, Corin). We use an anatomical construct through the clavicle, with a reefing repair of the ACJ capsule and delto-trapezial fascia. The LARS ligament exceeds the tensile strength of the native CC ligaments and has been shown to encourage fibroblast and collagen ingrowth [55]. In addition to the standard manufacturers technique we add a further figure-of-eight loop around the coracoid (Fig. 8.7) and/or over the ACJ through a drill-hole in the acromion (Fig. 8.8). We find both of these improve the horizontal stability and overall strength of the construct.

We evaluated our results of ACJ reconstruction, utilizing this LARS ligament technique, in professional and non-professional athletes at 2-year minimum follow-up [51]. All clinical scores and patient satisfaction improved significantly, with 2% loss of reduction at a 2-year minimum follow-up. Superior radiological outcomes in professionals were not correlated to improved clinical results.



**Fig. 8.7** LARS technique modification with second figure-of-eight loop under coracoid (courtesy of Lennard Funk, <http://www.shoulderdoc.co.uk>)



**Fig. 8.8** LARS ligament modified technique to reconstruct acromioclavicular ligaments via drill hole through the acromion and second loop under coracoid. This is mainly used for revision procedures, where the acromioclavicular joint cannot be indirectly stabilised (courtesy of Lennard Funk, <http://www.shoulderdoc.co.uk>)

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## 8.14 Post-operative Rehabilitation

- Phase 1:
  - Core stability and Scapula control
  - Proprioceptive exercises (minimal weightbearing below 90 degrees)
  - Active ROM as comfortable
  - No resistance exercises
- Phase 2:
  - Progress to light resistance exercises as tolerated
  - Sports-specific rehabilitation—Plyometrics and perturbation training
- Phase 3:
  - Regain scapula and glenohumeral stability working for shoulder joint control rather than range
  - Gradually Strengthening exercises

The progression through each phase is not time-based, but based on the patient's responses and ability to progress to the next phase (milestone based). This is a transitional, seamless process and supervised by experienced therapists.

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## 8.15 Return to Sports

Sports-specific rehabilitation starts within weeks of the rehabilitation process. The main aims are to apply the above rehabilitation principles in sports-specific manner. For example, rugby players will use a rugby ball for proprioceptive work and swimmers will do their rehabilitation in water. Return to sport participation and competition is done in a graduated way, with input from the surgeon, therapist and strength and conditioning coaches. In our experience this is usually achieved within 3–4 months for contact athletes and 6–9 months for overhead athletes. In our experience and practice reviews horse-riders and cyclists often return to sport less than 3 months post-surgery.

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## Q&A

- (1) Is there a role for MRI rather than standard radiographs in the diagnosis of AC joint injuries?

No well-conducted comparative study exists- as yet- to correlate radiographic analysis of ACJ injuries with MRI and correlate this with classifications or treatment of these injuries. Evidence confirms the ability of MRI to demonstrate ACJ injuries and assess the integrity of the CC ligaments (level IV). However, the sensitivity and specificity of injury detection and accuracy of classification via MRI compared to radiograph remains to be determined, and indications for MRI in this setting remain controversial. There is insufficient evidence to recommend use of MRI imaging for ACJ injuries. A detailed history and clinical examination (Coper versus non-coper) remains the mainstay of diagnosing these injuries

- (2) What is the optimal non-operative treatment of an AC joint injury?

Most patients with an ACJ injury especially in the milder forms would benefit from non-surgical intervention. These include: taping, bracing/splinting and slings.

- (3) When performing an arthroscopic distal clavicle excision for acromioclavicular joint arthrosis, which structures must be preserved to prevent post-operative anteroposterior instability of the clavicle?

Numerous biomechanical studies demonstrated that the primary restraint to AP translation of the clavicle is the ligamentous thickenings of the AC capsule. Debski et al. [56] showed that the strongest ligament is the superior one (50% of the strength against AP translation) and it is thickest in its posterior aspect. In addition, the posterior AC ligament provides extra 25% of the overall strength. For this reason, these ligaments should be preserved when performing a distal clavicle resection.

- (4) When should sports-specific rehabilitation start after reconstruction?

As long as the surgeon has achieved a strong and anatomical reconstruction, then incorporation of sport-specific exercises can be introduced very soon after surgery. Athletes can include aspects of their sports under supervision of their therapist and strength and conditioning coaches. Progressions through the rehabilitation phases should be personalized to the athlete and their sport, rather than being time-based.

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## References

1. Beaver AB, Parks BG, Hinton RY. Biomechanical analysis of distal clavicle excision with acromioclavicular joint reconstruction. *Am J Sports Med.* 2013;41(7):1684–8. <https://doi.org/10.1177/0363546513488750>.
2. Bontempo NA, Mazzocca AD. Biomechanics and treatment of acromioclavicular and sternoclavicular joint injuries. *Br J Sports Med.* 2010;44(5):361–9. <https://doi.org/10.1136/bjism.2009.059295>.
3. Buttaci CJ, Stitik TP, Yonclas PP, Foye PM. Osteoarthritis of the acromioclavicular joint: a review of anatomy, biomechanics, diagnosis, and treatment. *Am J Phys Med Rehabil.* 2004;83(10):791–7.

4. Fukuda K, Craig EV, An KN, Cofield RH, Chao EY. Biomechanical study of the ligamentous system of the acromioclavicular joint. *J Bone Joint Surg.* 1986;68(3):434–40.
5. Urist MR. Complete dislocation of the acromioclavicular joint. *J Bone Joint Surg.* 1963;45:1750–3.
6. Depalma AF. Surgical anatomy of acromioclavicular and sternoclavicular joints. *Surg Clin North Am.* 1963;43:1541–50.
7. Izadpanah K, Winterer J, Vicari M, Jaeger M, Maier D, Eisebraun L, Ute Will J, Kottler E, Langer M, Sudkamp NP, Hennig J, Weigel M. A stress MRI of the shoulder for evaluation of ligamentous stabilizers in acute and chronic acromioclavicular joint instabilities. *J Magn Reson Imaging.* 2013;37(6):1486–92. <https://doi.org/10.1002/jmri.23853>.
8. Lizaur A, Marco L, Cebrian R. Acute dislocation of the acromioclavicular joint. Traumatic anatomy and the importance of deltoid and trapezius. *J Bone Joint Surg.* 1994;76(4):602–6.
9. Stine IA, Vangsness CT Jr. Analysis of the capsule and ligament insertions about the acromioclavicular joint: a cadaveric study. *Arthroscopy.* 2009;25(9):968–74. <https://doi.org/10.1016/j.arthro.2009.04.072>.
10. Costic RS, Labriola JE, Rodosky MW, Debski RE. Biomechanical rationale for development of anatomical reconstructions of coracoclavicular ligaments after complete acromioclavicular joint dislocations. *Am J Sports Med.* 2004;32(8):1929–36.
11. Rios CG, Arciero RA, Mazzocca AD. Anatomy of the clavicle and coracoid process for reconstruction of the coracoclavicular ligaments. *Am J Sports Med.* 2007;35(5):811–7. <https://doi.org/10.1177/0363546506297536>.
12. Jerosch J. The acromioclavicular joint. *Der Orthopade.* 2000;29(10):895–908.
13. Lee S, Bedi A. Shoulder acromioclavicular joint reconstruction options and outcomes. *Curr Rev Musculoskelet Med.* 2016;9(4):368–77. <https://doi.org/10.1007/s12178-016-9361-8>.
14. 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. <https://doi.org/10.2165/11633460-000000000-00000>.
15. Gumina S, Carbone S, Postacchini F. Scapular dyskinesis and SICK scapula syndrome in patients with chronic type III acromioclavicular dislocation. *Arthroscopy.* 2009;25(1):40–5. <https://doi.org/10.1016/j.arthro.2008.08.019>.
16. Codman E. *The shoulder.* Malabar: Robert E. Krieger Publishing Company Inc; 1934.
17. Mazzocca AD, Arciero RA, Bicos J. Evaluation and treatment of acromioclavicular joint injuries. *Am J Sports Med.* 2007;35(2):316–29. <https://doi.org/10.1177/0363546506298022>.



18. Cadenat F. The treatment of dislocations and fractures of the outer end of the clavicle. *Int Clin.* 1917;1:145–69.
19. Allman FL Jr. Fractures and ligamentous injuries of the clavicle and its articulation. *J Bone Joint Surg.* 1967;49(4):774–84.
20. Tossy JD, Mead NC, Sigmund HM. Acromioclavicular separations: useful and practical classification for treatment. *Clin Orthop Relat Res.* 1963;28:111–9.
21. Chye Yew Ng EKS, Funk L. Reliability of the traditional classification system for acromioclavicular joint injuries by radiography. *Should Elb.* 2012;4(4):266–9.
22. Schneider MM, Balke M, Koenen P, Fröhlich M, Wafaisade A, Bouillon B, Banerjee M. Inter- and intraobserver reliability of the Rockwood classification in acute acromioclavicular joint dislocations. *Knee Surg Sports Traumatol Arthrosc.* 2016;24(7):2192–6.
23. Cho CH, Hwang I, Seo JS, Choi CH, Ko SH, Park HB, Dan J. Reliability of the classification and treatment of dislocations of the acromioclavicular joint. *J Shoulder Elbow Surg.* 2014;23(5):665–70.
24. Nemeč U, Oberleitner G, Nemeč SF, Gruber M, Weber M, Czerny C, Krestan CR. MRI versus radiography of acromioclavicular joint dislocation. *AJR Am J Roentgenol.* 2011;197(4):968–73. <https://doi.org/10.2214/AJR.10.6378>.
25. Beitzel K, Mazocco AD, Bak K, Itoi E, Kibler WB, Mirzayan R, Imhoff AB, et al. ISAKOS upper extremity committee consensus statement on the need for diversification of the Rockwood classification for acromioclavicular joint injuries. *Arthroscopy.* 2014;30(2):271–8.
26. Walton J, Mahajan S, Paxinos A, Marshall J, Bryant C, Shnier R, Quinn R, Murrell GA. Diagnostic values of tests for acromioclavicular joint pain. *J Bone Joint Surg.* 2004;86A(4):807–12.
27. 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.
28. Bosworth BM. Complete acromioclavicular dislocation. *N Engl J Med.* 1949;241(6):221–5. <https://doi.org/10.1056/NEJM194908112410601>.
29. Bearden JM, Hughston JC, Whatley GS. Acromioclavicular dislocation: method of treatment. *J Sports Med.* 1973;1(4):5–17.
30. Kovilazhikathu Sugathan H, Dodenhoff RM. Management of type 3 acromioclavicular joint dislocation: comparison of long-term functional results of two operative methods. *ISRN Surg.* 2012;2012:580504. <https://doi.org/10.5402/2012/580504>.
31. Phillips AM, Smart C, Groom AF. Acromioclavicular dislocation. Conservative or surgical therapy. *Clin Orthop Relat Res.* 1998;353:10–7.
32. Tibone J, Sellers R, Tonino P. Strength testing after third-degree acromioclavicular dislocations. *Am J Sports Med.* 1992;20(3):328–31. <https://doi.org/10.1177/036354659202000316>.
33. 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. <https://doi.org/10.1177/03635465010290060401>.

34. Mouhsine E, Garofalo R, Crevoisier X, Farron A. Grade I and II acromioclavicular dislocations: results of conservative treatment. *J Shoulder Elb Surg.* 2003;12(6):599–602. <https://doi.org/10.1016/S1058274603002155>.
35. Cox JS. The fate of the acromioclavicular joint in athletic injuries. *Am J Sports Med.* 1981;9(1):50–3. <https://doi.org/10.1177/036354658100900111>.
36. Rangger C, Hrubesch R, Paul C, Reichkendler M. Capacity to participate in sports after injuries of the acromioclavicular joint. *Der Orthopade.* 2002;31(6):587–90.
37. J Gladstone KW, Andrews J. Nonoperative treatment of acromioclavicular joint injuries. *Oper Tech Sports Med.* 1997;5:78–87.
38. Bradley JP, Elkousy H. Decision making: operative versus nonoperative treatment of acromioclavicular joint injuries. *Clin Sports Med.* 2003;22(2):277–90.
39. Tienen TG, Oyen JF, Eggen PJ. A modified technique of reconstruction for complete acromioclavicular dislocation: a prospective study. *Am J Sports Med.* 2003;31(5):655–9. <https://doi.org/10.1177/03635465030310050401>.
40. Balke M, Schneider MM, Akoto R, Bathis H, Bouillon B, Banerjee M. Acute acromioclavicular joint injuries. Changes in diagnosis and therapy over the last 10 years. *Unfallchirurg.* 2015;118(10):851–7. <https://doi.org/10.1007/s00113-013-2547-2>.
41. Rauschnig W, Nordesjo LO, Nordgren B, Sahlstedt B, Wigren A. Resection arthroplasty for repair of complete acromioclavicular separations. *Arch Orthop Trauma Surg.* 1980;97(3):161–4.
42. Baumgarten KM, Altchek DW, Cordasco FA. Arthroscopically assisted acromioclavicular joint reconstruction. *Arthroscopy.* 2006;22(2):228. e221–6. <https://doi.org/10.1016/j.arthro.2005.12.026>.
43. Beitzel K, Cote MP, Apostolakis J, Solovyova O, Judson CH, Ziegler CG, Edgar CM, Imhoff AB, Arciero RA, Mazzocca AD. Current concepts in the treatment of acromioclavicular joint dislocations. *Arthroscopy.* 2013;29(2):387–97. <https://doi.org/10.1016/j.arthro.2012.11.023>.
44. Qi W, Xu Y, Yan Z, Zhan J, Lin J, Pan X, Xue X. The tight-rope technique versus clavicular hook plate for treatment of acute acromioclavicular joint dislocation: a systematic review and meta-analysis. *J Invest Surg.* 2019;14:1–10. <https://doi.org/10.1080/08941939.2019.1593558>.
45. Tsou PM. Percutaneous cannulated screw coracoclavicular fixation for acute acromioclavicular dislocations. *Clin Orthop Relat Res.* 1989;243:112–21.
46. Gohring U, Matusewicz A, Friedl W, Ruf W. Results of treatment after different surgical procedures for management of acromioclavicular joint dislocation. *Chirurg.* 1993;64(7):565–71.
47. Harris RI, Wallace AL, Harper GD, Goldberg JA, Sonnabend DH, Walsh WR. Structural properties of the intact and the reconstructed coracoclavicular ligament complex. *Am J Sports Med.* 2000;28(1):103–8. <https://doi.org/10.1177/03635465000280010201>.
48. Motta P, Maderni A, Bruno L, Mariotti U. Suture rupture in acromioclavicular joint dislocations treated with flip buttons. *Arthroscopy.* 2011;27(2):294–8. <https://doi.org/10.1016/j.arthro.2010.09.009>.

49. Thiel E, Mutnal A, Gilot GJ. Surgical outcome following arthroscopic fixation of acromioclavicular joint disruption with the tightrope device. *Orthopedics*. 2011;34(7):e267–74. <https://doi.org/10.3928/01477447-20110526-11>.
50. Walz L, Salzmann GM, Fabbro T, Eichhorn S, Imhoff AB. The anatomic reconstruction of acromioclavicular joint dislocations using 2 TightRope devices: a biomechanical study. *Am J Sports Med*. 2008;36(12):2398–406. <https://doi.org/10.1177/0363546508322524>.
51. Marcheggiani Muccioli GM, Manning C, Wright P, Grassi A, Zaffagnini S, Funk L. Acromioclavicular joint reconstruction with the LARS ligament in professional versus non-professional athletes. *Knee Surg Sports Traumatol Arthrosc*. 2016;24(6):1961–7. <https://doi.org/10.1007/s00167-014-3231-y>.
52. Bartonicek J, Jehlicka D, Bezvoda Z. Surgical treatment of acromioclavicular luxation. *Acta Chir Orthop Traumatol Cechoslov*. 1988;55(4):289–309.
53. Bundens WD Jr, Cook JI. Repair of acromioclavicular separations by deltoid-trapezius imbrication. *Clin Orthop*. 1961;20:109–15.
54. Lizaar A, Sanz-Reig J, Gonzalez-Parreno S. Long-term results of the surgical treatment of type III acromioclavicular dislocations: an update of a previous report. *J Bone Joint Surg*. 2011;93(8):1088–92. <https://doi.org/10.1302/0301-620X.93B8.26775>.
55. Trieb K, Blahovec H, Brand G, Sabeti M, Dominkus M, Kotz R. In vivo and in vitro cellular in growth into a new generation of artificial ligaments. *Eur Surg Res*. 2004;36(3):148.
56. Debski RE, Parsons IM, Fenwick J, Vangura A. Ligament mechanics during three degree-of-freedom motion at the acromioclavicular joint. *Ann Biomed Eng*. 2000;28(6):612–8.



# The Sternoclavicular Joint

# 9

Graham Tytherleigh-Strong,  
Elizabeth Pinder, and Muiris Kennedy

## Key Learning Points

- Understand that the sternoclavicular joint is inherently very stable
- Dislocations can occur following injuries or atraumatically
- Acute posterior dislocations can lead to mediastinal compromise and are a potential medical emergency
- Most pathologies can be managed non-operatively
- Surgery is occasionally required for symptomatic instability and painful arthritis.

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## 9.1 Anatomy

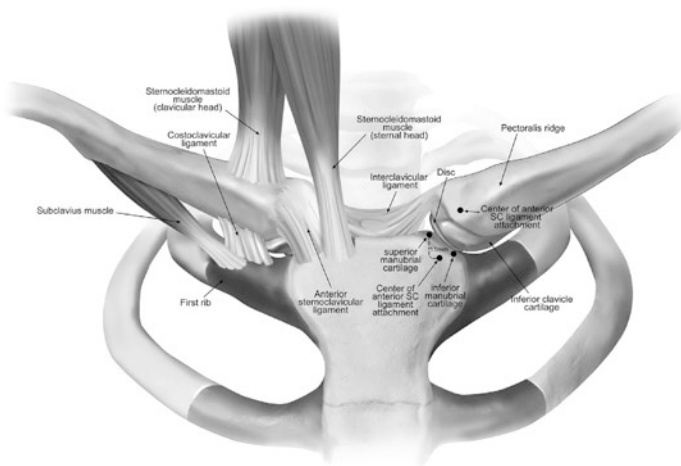
The sternoclavicular joint (SCJ) is formed by the articulation between the medial end of the clavicle and the sternal manubrium and plays a vital role in the attachment of the shoulder girdle to

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the body. It is the only true articular connection between the upper limb and the axial skeleton, as the scapulothoracic joint is not a true synovial joint.

The SCJ is a synovial joint with largely incongruent articular surfaces (Fig. 9.1). On the clavicular side the surface is saddle shaped with a concavity in the anteroposterior plane and convexity in the vertical plane [1, 2]. Between the articular surfaces lies a fibrocartilaginous disc [3], similar to the meniscus of the knee. This separates the joint into a medial and lateral compartment and is attached to the capsule at its periphery, to the superior surface of the medial end of the clavicle and the first costal cartilage inferiorly [2]. Despite the incongruent articular surfaces and small surface area of the joint, the SCJ is extremely stable owing to the effect of strong static (both intrinsic and extrinsic) and dynamic, soft tissue stabilisers (see Table 9.1) [4].



**Fig. 9.1** Sternoclavicular joint

**Table 9.1** Stabilisers of the SCJ

Static stabilisers	Dynamic stabilisers
Capsule <i>Intrinsic stabilisers</i> Intra-articular disc ligament Anterior sternoclavicular ligament Posterior sternoclavicular ligament <i>Extrinsic stabilizers</i> Interclavicular Costoclavicular	Subclavius muscle Sternocleidomastoid muscle Pectoralis major muscle

The anterior and posterior sternoclavicular ligaments are formed by thickenings in the capsule and are the most important contributors to antero-posterior stability [5]. The intra-articular fibrocartilagenous disc resists medial translation of the clavicle [4]. As a result, the disc can be prone to shearing injury, usually as a degenerate tear but occasionally as an acute incident.

The interclavicular ligament passes between the medial ends of both clavicles via the posterior aspect of the sternal notch and resists clavicular superior translation from gravity or forceful depression of the upper limb [4, 6]. The costoclavicular ligament passes from the inferior aspect of the medial clavicle to the first rib and/or first costal cartilage [7]. It is an important restraint when the clavicle is elevated.

The dynamic stabilisers form a musculo-tendinous envelope around the joint. The sternocleidomastoid and pectoralis major tendons lie anterior to the SCJ and play a role in anterior and posterior stability, whilst the subclavius passes from the inferior aspect of the clavicle to the first rib providing superior stability as well as an additional anterior/superior component.

A number of vital structures lie posterior to the SCJ including the great vessels of the neck, oesophagus and the trachea. These are at potential risk following posterior dislocations. A layer formed by the sternothyroid and sternohyoid muscles lies between these structures and the joint capsule [1, 2].

The epiphysis of the medial end of the clavicle is the first epiphysis to appear in utero and the last to close (25–31 years) [8, 9]. This is of relevance because the physis at the medial end of the clavicle is weaker than the SCJ ligaments. Significant traumatic injuries before physal closure, under the age of 25 years, may result in fracture through the physis rather than a true SCJ dislocation.

The SCJ moves in three planes: retraction/protraction, elevation/depression and rotation [10]. Movement at the SCJ and ACJ allows the scapula to move around the thorax to position the glenoid in the optimal location to maintain glenohumeral joint congruency for upper limb positioning.

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## 9.2 History and Examination

As with any upper limb complaint, it is important to consider the age, handedness, sport, aspirations and occupation of the patient. An acute injury typically involves a high-energy mechanism and an SCJ injury may be missed in the presence of more dramatic components. Details of the exact mechanism of injury including direction of impact should be sought. Up to 30% of acute posterior dislocations develop mediastinal compromise, concerning features include dyspnoea, dysphonia, dysphagia, coughing and venous congestion of the ipsilateral arm and should be considered as a medical emergency. Patients usually present with pain over the SCJ in the presence of a deformity, a prominence of the medial clavicle in anterior dislocations and a defect lateral to the sternum in a posterior dislocation.

In patients presenting with more chronic problems a history of previous trauma or a change of activity preceding the onset of symptoms may be relevant. In younger patients, complaints of pain, clicking, a feeling of instability or even recurrent dislocation in the absence of injury may suggest an atraumatic instability. A

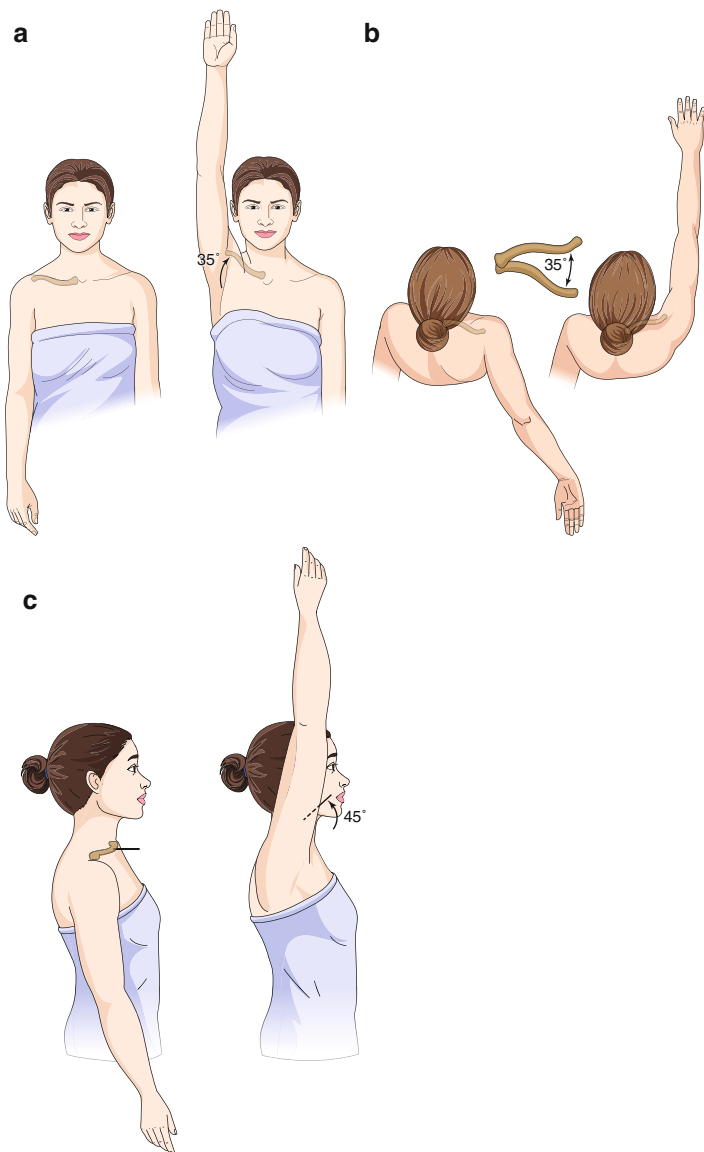
history of connective tissue disorders such as Ehlers-Danlos Syndrome maybe relevant. Older patients may present with a pain and restriction of movement associated with a swelling over the medial end of the clavicle.

SCJ examination is predominantly based on comparison and any asymmetry between sides. This requires exposure of the upper trunk to allow for comparison of both shoulder girdles including the clavicles, glenohumeral joints and scapulothoracic movements. There may be obvious asymmetry between the patient's SCJs with a lump present on the affected side. It is important to determine whether this is soft, representing an effusion or synovitis secondary to an inflammatory arthropathy or infection, or hard, which could represent either a chronic anterior dislocation of the medial end of the clavicle or an osteophyte secondary to osteoarthritis.

Movements at the SCJ are intimately related to the rest of the shoulder girdle, so that assessment of the ACJ, glenohumeral joint and scapulothoracic movements are essential to identify any confounding pathology. Both the SCJs should be examined and compared in 3 planes of movement. Protraction/retraction with the arms in full extension, elevation with the arms extended in maximal abduction. Rotation with the arms at 90°, abduction and the elbows flexed to 90° moves in 3 planes (Fig. 9.2).

It is important to also place a hand over the anterior joint to feel for any abnormal movement and clicking through the range of motion. Clicking, popping, or crepitus at the joint during movement may suggest degenerative changes or, in a younger patient, a disc tear. The medial end of the clavicle may sublux or even dislocate anteriorly in patients with instability. In this instance broader assessment of the stabilising soft tissue envelope, particularly looking at sternocleidomastoid and the sternal part of Pectoralis Major, for muscle sequencing over activity.





**Fig. 9.2** Examination of the sternoclavicular joint—3 planes of movement. (a) Superior and inferior elevation. (b) Anterior and posterior (protraction and retraction). (c) Rotation

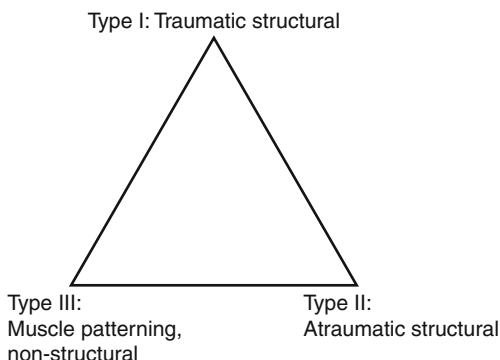
## 9.3 Sterno-Clavicular Joint Pathophysiology

### 9.3.1 Instability

Sterno-clavicular joint (SCJ) instability can be classified by direction (anterior or posterior), by severity (sprain, subluxation or dislocation—often referred to as type 1, 2 or 3) or by whether it is acute, recurrent or persistent (chronic/unreduced). Whilst these classifications are descriptive, none of them are able to take into account the traumatic or atraumatic nature of the instability. However, a classification system, that is a direct derivation of the Stanmore tri-polar instability triangle for the glenohumeral joint, has recently been described for the SCJ. In the Stanmore SCJ instability classification there are three polar groups: type I traumatic structural, type II atraumatic structural and type III muscle patterning (neuromuscular) (Fig. 9.3).

The type I traumatic structural group comprises traumatic subluxations and dislocations of the SCJ, as well as medial physal fracture displacements. The type II atraumatic structural group comprises conditions that lead to laxity of the restraining ligaments, and includes connective tissue disorders (Marfan's, Ehlers Danlos), degenerative arthritis, inflammatory arthritis, infection and clavicular shortening secondary to previous malunion. The type III muscle patterning group can occur in isolation and is most commonly due to an over active or aberrant pectoralis major

**Fig. 9.3** Stanmore triangle

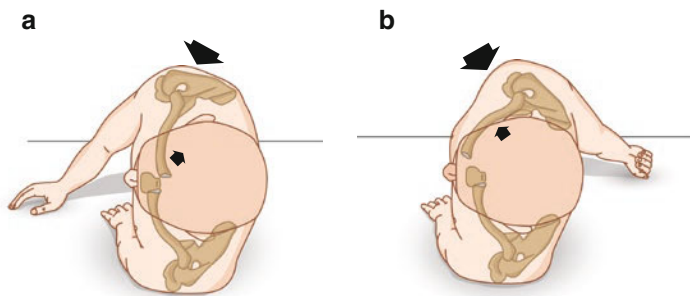


muscle but it can also develop secondary to a type I or type II disorder.

A continuum exists between the groups. Therefore, a patient with an initial type II cause of instability can develop secondary muscle patterning (type III) over time; this patient would be then classified as type II/III. The effect of any treatment can also be monitored using the Stanmore SCJ instability classification system. Patients 'migrate' around the triangle, depending on the presenting pathology, and how that changes over time as their treatment progresses.

### 9.3.1.1 Type I Traumatic Structural

Traumatic SCJ dislocations are rare, accounting for less than 1% of upper limb injuries, and usually occur as the result of a high-energy impact. The force is usually indirect and follows an impact either to the front or the back of the humeral head [11]. The force vector is then transferred along the clavicle resulting in disruption of the SCJ's restraining soft tissues. If the scapula is protracted at the time of impact a posterior dislocation is more likely and if the scapula is retracted it is more likely to dislocate anteriorly (Fig. 9.4). Less frequently a direct anterior blow to the clavicle can drive the medial end posteriorly into the mediastinum [12]. Biomechanical studies have shown that the force required to



**Fig. 9.4** Mechanism of injury. (a) Posterior dislocation: the scapula is protracted with an indirect force to the posterior shoulder. (b) Anterior dislocation: the scapula is retracted with an indirect force to the anterior shoulder

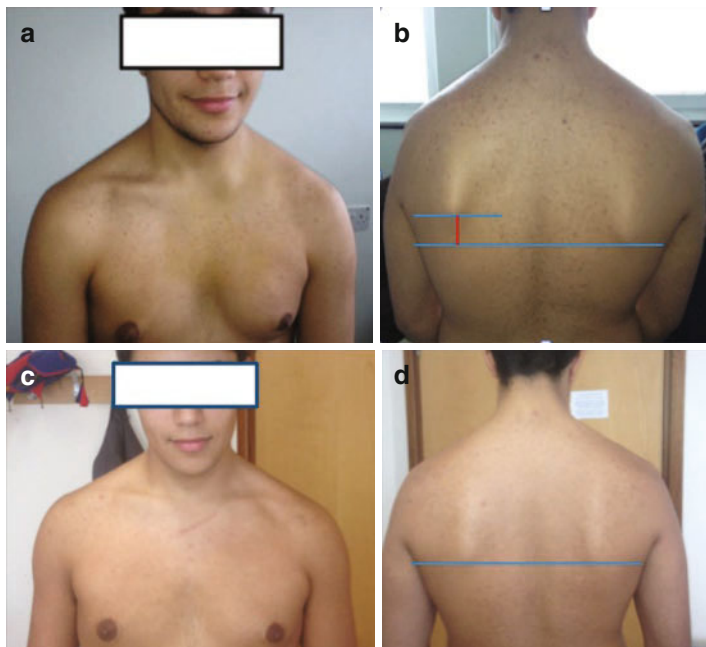
dislocate the SCJ posteriorly is 50% greater than that required to cause an anterior displacement [13].

A meta-analysis of 140 adolescents with posterior SCJ dislocations reported that 71% occurred during sporting activities [14]. Although still rare, this requires particular vigilance by pitch-side sports physicians and physiotherapists as over 30% of patients following an acute posterior SCJ dislocation develop mediastinal pressure symptoms. Acute symptoms include dyspnoea (14%) and dysphagia (22.5%) due to pressure on the trachea and oesophagus and venous congestion or oedema of the ipsilateral arm due to compression of the vessels (14%) [14]. Less common complications of posterior dislocations include mediastinal hematoma, vessel laceration (leading to death), stroke, pneumomediastinum, pneumothorax, and venous thromboembolism (0.72–2.90%) [14]. As a result, an acute posterior SCJ dislocation should be treated as a medical emergency.

Patients presenting more chronically often complain of pain and deformity over the SCJ. In certain patients, as the medial clavicle has been pushed posteriorly, the whole of the shoulder girdle has rotated anteriorly and superiorly. As a result, the scapular tends to sit in a more superior and protracted position. Patients may complain of problems with glenohumeral function and of asymmetrical scapular protraction which, for example, can make sitting in high-backed chairs uncomfortable as the medial scapula adopts a winged position (Fig. 9.5).

On examination an anterior SCJ dislocation presents with an obvious forward displacement of the clavicle, while a posterior dislocation demonstrates asymmetry compared with the contralateral side, with diminution of the entire clavicular contour on the affected side. However, there is often significant soft-tissue swelling in the days after an acute posterior dislocation, which may make this less obvious. It can also be difficult to clinically distinguish a medial clavicular physis fracture-dislocation from a true SCJ dislocation. A high clinical suspicion for medial clavicle physeal injury should remain for anyone under 25 years.

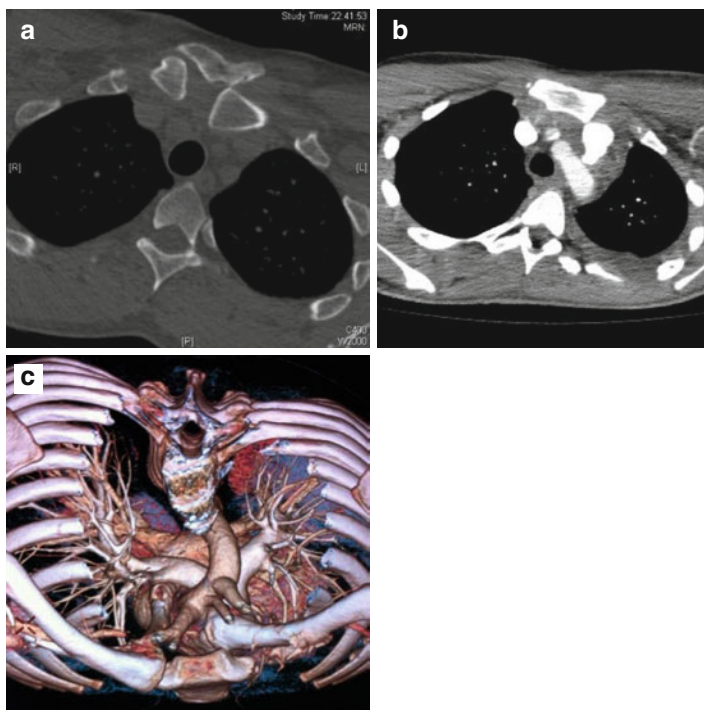
Traditionally initial investigations following an SCJ injury include plain radiography using either a Serendipity or Heinig view. However, these are often difficult to interpret. A plain chest



**Fig. 9.5** Posterior dislocation. A 16-year-old boy referred 4 weeks after sustaining a left posterior SCJ dislocation in a tobogganing accident. His CT scan confirmed an SCJ dislocation rather than an expected medial clavicular physal injury. **(a)** Anterior view: note the asymmetry and loss of clavicular contour on the left. **(b)** Posterior view. Note the elevated and winged scapula on the left hand side. 3 months following open reduction and stabilisation using a figure of eight gracilis graft. **(c)** Anterior view: clavicular symmetry has been returned. **(d)** Posterior view: the left scapula has now returned to its normal position

X-ray may be considered following an acute injury to check for an associated pneumothorax secondary to rib fractures. Currently the investigation of choice is a CT scan or, in the case of a posterior dislocation, a CT angiogram, this should be undertaken as a matter of urgency in the acute situation should there be any concern with regards to mediastinal compromise [15]. A CT scan can accurately assess the position of the medial end of the clavicle with regards to the sternum and the contralateral SCJ. It can also

differentiate between a dislocation and a medial physal injury. A CT angiogram additionally shows the arch of the aorta and great vessels in relation to the medial clavicle (Fig. 9.6). An MRI scan has poorer bony resolution than a CT scan but is able to more effectively demonstrate the ligamentous structures following subluxation and recurrent dislocation. It is also able to assess the intra-articular disc for injury and the condition of adjacent neurovascular anatomy.



**Fig. 9.6** CT scan (plain, angiogram and 3D reconstruction) of an acute posterior dislocation of the left SCJ. (a) Plain CT: axial view. (b) CT angiogram: axial view. The dislocated medial end of the left clavicle is abutting the arch of the aorta. (c) CT angiogram 3D reconstruction: The medial end of the clavicle is sitting on the arch of the aorta

Management of Type 1 SCJ instability depends on the severity of the injury, the direction of instability and the time from injury. Anterior and posterior undisplaced ligamentous sprains and subluxations of the SCJ (Grades 1 and 2) and minimally displaced medial physal fractures can usually be treated with conservative measures. Initial reassurance, oral analgesia, and ice—coupled with a short period of immobilisation in a sling—is usually sufficient. The patient should be advised to avoid re-injury for 3 months and should avoid contact sports or other high-risk activities until there is a resolution of clinical symptoms [16]. There is no brace or support that will provide any extra protection to the SCJ on return to contact sports.

The management of SCJ dislocations (Grade 3 injuries) is dependent on the direction and the time after injury (<48 h or later). For anterior dislocations that are less than 48 h post-injury, a closed reduction under sedation or general anaesthetic can be attempted. With a bolster placed under the patient between their scapulae, the clavicle is pushed in a posterior direction. The SCJ usually reduces easily but sometimes traction to the arm is necessary to pull the clavicle laterally. The arm should then be kept in a sling for four weeks in internal rotation [17]. Unfortunately, in over 50% of cases the SCJ re-dislocates. Some surgeons have advocated surgical stabilisation of the joint using a ligamentous sling as definitive primary treatment, however, there is little evidence for this [18]. The majority of surgeons adopt a wait and see policy following an anterior dislocation. Over the course of 3–6 months most patients' symptoms settle with conservative management, including a combination of physiotherapy and time [13]. However, in the unusual situation where a patient continues to have significant symptoms, despite an adequate period of conservative management once muscle patterning has been excluded as a contributing factor, then surgical reduction and stabilisation might be considered.

For posterior SCJ dislocations there is a greater need to reduce and maintain reduction of the joint. In the acute situation, in the face of mediastinal compromise, this is particularly the case. A chronic posterior dislocation may affect shoulder girdle function due to protraction of the scapula and there are concerns of poten-

tially developing erosion to the subclavian artery or thoracic duct injury and trachea-oesophageal fistula. Although these complications are rare, their probability will increase over time and so operative reduction and stabilisation may perhaps be of more consideration the younger the patient. Closed reduction is only generally considered if the injury is less than 48 h old. Closed reduction manoeuvres after 48 h are discouraged, as they may result in tearing of posterior structures, owing to the formation of adhesions. Closed reduction is undertaken using a general anaesthetic and a radio-translucent table allowing access for fluoroscopy. A bolster is placed posteriorly between the scapulae with the patient supine. Abduction, traction and extension are applied to the affected arm and a towel clip is used to grasp the medial clavicle and to pull it anteriorly [19].

Closed reduction of posterior dislocations are difficult, with a reported success rate of approximately 56% in those cases attempted within 48 hours and of 31% if undertaken between the second and fifth day [14]. Taking into account such a high potential failure rate for a closed reduction it is important to consider pre-operatively the requirement for an open reduction. This may mean that a patient requires transfer to an appropriate facility where cardiothoracic cover is available. If successful most closed reductions performed in the early acute phase remain stable in the long term, however, due to soft-tissue swelling and difficulties in interpreting fluoroscopy images around the SCJ, a repeat CT scan to confirm the reduction has been maintained should be undertaken the next day. Therefore, one should always plan for the eventuality of performing an open reduction at the same general anaesthetic if the closed option has failed. For this reason, the on-site cardiothoracic surgeon should be informed prior to booking a patient for closed reduction. If closed reduction is achieved, the patient can thereafter be kept in a figure of eight brace in order to keep the scapulae retracted for four weeks. Sports are avoided for at least 3–6 months.

Open reduction in the acute phase is usually technically easier due to the lack of adhesions, and the consequent diminished risk to the posterior mediastinal structures. In the chronic case, preoperative planning with a CT arteriogram with discussion and col-

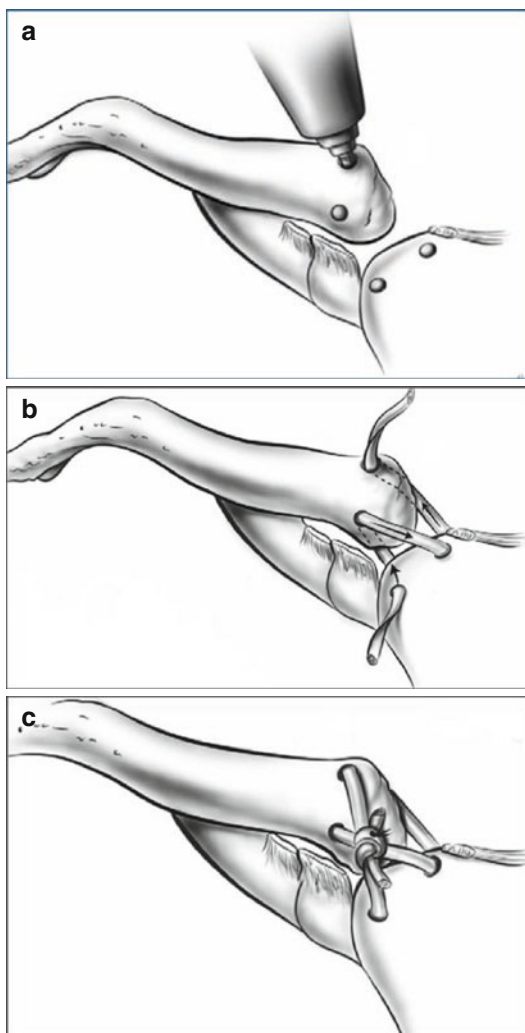


laboration with a cardiothoracic surgeon are essential. Any likely adhesions to the posterior mediastinal vascular structures, with the brachiocephalic veins in particular, can then be anticipated. A transverse incision is made over the SCJ and after freeing any adhesions, the clavicle is reduced by anterior and laterally directed traction applied through a towel clip or bone holding forceps. The normal capsular and ligamentous stabilisers are usually only partially repairable and would not be biomechanically sufficient to maintain the reduction and, as a result, an open reduction will usually require some form of additional reconstruction [5].

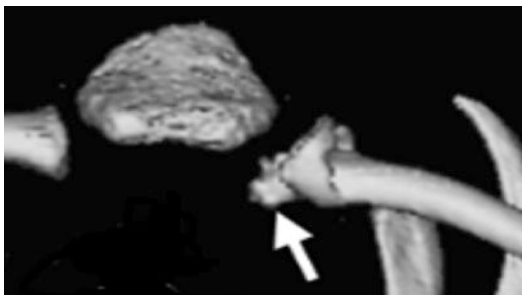
Previously, various types of wires and pins have been used to stabilise the joint, however, due to reported lethal complications, these techniques have, in the most part, been abandoned. Reconstruction using sutures alone through osseous drill holes or suture anchors have been reported, but with only marginal biomechanical results. The most recent trend has been towards reconstruction techniques using autograft (palmaris longus, semitendinosis, gracilis or sternocleidomastoid) or allograft. A number of techniques have been described and although satisfactory outcomes have been reported for most techniques, a figure-of-eight reconstruction appears to be biomechanically superior and may lead to better longer term outcomes. In this technique the graft is shuttled through 3.2 mm drill holes in the sternum and medial end of the clavicle. Synthetic ultra-strong synthetic braided sutures, such as Orthocord (DePuy Mitek, Raynham, MA) and Fibrewire (Arthrex, Naples, FL) may be useful in augmenting the graft. The ends of the graft are then tensioned and sutured together, any surrounding remnants of the capsule may be incorporated into the repair (Fig. 9.7).

### **9.3.1.2 Medial Physeal Clavicle Fractures**

The medial clavicular epiphysis does not ossify until between eighteen to 25-years-of-age. As a result, injury to the SCJ in patients younger than 25 years may actually lead to a displaced medial physeal fracture rather than a straightforward dislocation. A CT scan is the investigation of choice (Fig. 9.8). Fortunately, most physeal injuries are either un- or minimally displaced and rarely extend into the SCJ [20]. These injuries can be treated non-operatively with immobilisation in a sling.



**Fig. 9.7** SCJ reconstruction using a figure-of-eight hamstring tendon graft. (a) 3.2 mm drill holes are made in the medial end of the clavicle and the sternum. (b) Tendon graft is passed through the holes in a figure of eight. (c) The tendon ends are tensioned and sutured/tied together



**Fig. 9.8** CT 3D reconstruction of a right clavicular medial physal fracture in a 19-year-old man

More than 50% of patients with significantly displaced fractures that are treated non-operatively end up with persistent discomfort [13]. Some authors recommend an attempt at closed reduction for posteriorly displaced fractures within 7 days of injury. Open reduction should be reserved for injuries associated with mediastinal compressive symptoms [17]. Medial clavicle physal injuries are stable once reduced and usually do not require K-wire fixation [21]. Anterior physal injuries and posterior injuries presenting after 7 days may be treated symptomatically, with a degree of remodelling possible depending on the age of the patient.

### **9.3.1.3 Type II Atraumatic Structural**

Type II SCJ instability occurs as the result of either increased laxity or stretching out of the joint stabilising ligaments. It can be caused by a variety of pathologies including conditions that lead to ligamentous laxity (Marfan's, Ehlers-Danlos) or those that can weaken or stretch the ligaments such as degenerative and inflammatory arthritis, infection and clavicular shortening, secondary to fracture malunion. Correct diagnosis, therefore, requires an accurate history and careful local and systemic examination.

In cases of capsular laxity clinical evidence of a generalised ligamentous laxity secondary to conditions such as Ehlers-Danlos and Marfan's may be present. Typically, patients present in their teens with no specific history of trauma, with a prominence and subluxation of the medial clavicle and associated pain with over-

head activities. The majority of patients can be managed successfully with physiotherapy and corticosteroid injections. In the largest reported series [22], 29 of 37 patients (78%) returned to full activity when treated non-operatively. Eight of the patients (21%) had ongoing discomfort with evidence of persistent subluxation remaining in nearly all cases. The authors cautioned against surgical treatment of these cases, as all of the patients that were managed surgically reported unsatisfactory results.

Owing to the much stronger posterior capsular restraints [23], posterior atraumatic type II instability secondary to ligamentous laxity is much rarer than anterior [24]. However, in a similar way to the traumatic posterior dislocations, if at any point a patient's symptoms should become suggestive of retrosternal compression an open operative reduction is indicated.

The SCJ is a synovial joint and can be affected by any of the arthritides. Osteoarthritis is the most common, occurring most frequently in middle-aged women. As part of the osteoarthritic process a combination of degenerative changes within the ligamentous tissues and stretching, secondary to anterior osteophyte formation, can lead to capsular laxity. In certain patients this can manifest as clicking and subluxation of the joint as well as the associated pain. Whilst instability is usually not the primary complaint capsular laxity should be taken into consideration when surgical management with resection of the medial end of the clavicle, either open or arthroscopic, is undertaken.

Arthritic involvement of the SCJ has been reported in over 30% of patients with rheumatoid arthritis and in 90% of patients with severe psoriatic arthritis. The management of these conditions usually involves systemic pharmacological suppression and local intra-articular steroid injections. However, in severely affected cases debridement of the SCJ and stabilisation maybe considered.

Instability of the SCJ can occur following septic arthritis. Effusion and destruction of the anterior capsule and ligamentous structures can result in subluxation and even dislocation. Initial management requires complete eradication of the underlying infection and, although in most cases the joint will stiffen up, occasionally reconstruction of the damaged anterior soft-tissue structures.

Clavicular malunion resulting in relative anterior angulation of the medial end of the clavicle can give the appearance and sensation of anterior subluxation. This is particularly accentuated during retraction of the scapula and over time can lead to type II instability due to stretching out the anterior SCJ capsule. Other conditions that place the scapula in persistently abnormal positions, such as occurs with scoliosis, also predispose to atraumatic SCJ instability [17]. If the clavicular-malunion-induced SCJ symptoms are significant a corrective clavicular corrective osteotomy with a simultaneous SCJ stabilisation procedure may be necessary.

#### **9.3.1.4 Type III Muscle Patterning**

Type III instability is characterised by poorly coordinated afferent and efferent neuromuscular biofeedback loops in the presence of otherwise normal musculature and a structurally normal joint. The pectoralis major is the most commonly affected muscle and can be confirmed with EMG studies. Management focuses on re-learning the correct patterns of muscle contraction with proprioceptive feedback playing an important role [25]. Occasionally Botulinum toxin can be used to suppress hypertonicity in pectoralis major if slow progress is being made with physiotherapy treatment.

Muscle patterning can also arise secondary to type I or II instabilities, making the diagnosis lie on the I/III or II/III axis. In this situation it is important that the biofeedback loops are addressed prior to any stabilisation surgery. Botulinum toxin should be considered routinely pre-operatively in order to protect the ligamentous stabilisation in the first three months post-operatively.

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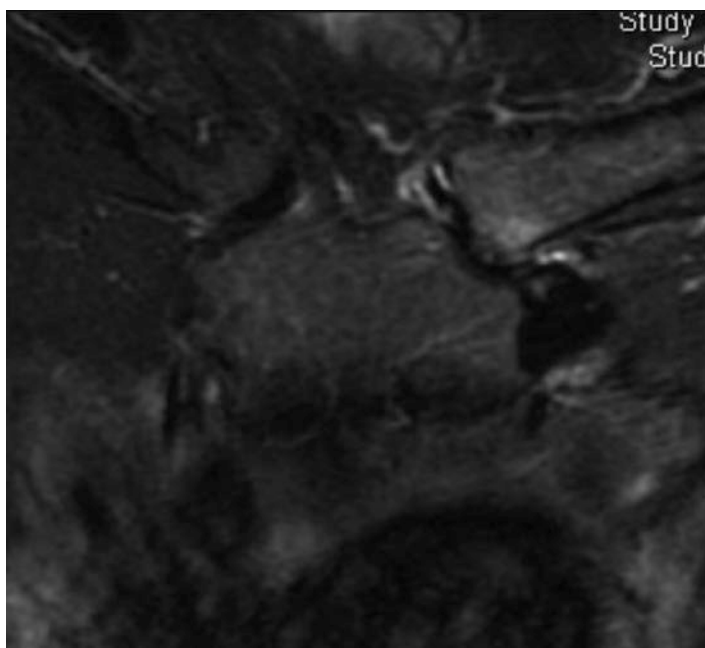
## **9.4 SCJ Disc Pathologies**

The SCJ is divided into medial and lateral halves by a complete fibrocartilaginous disc, which resembles a discoid meniscus in the lateral compartment of the knee. Although rare, damage to the disc can cause symptoms of pain and clicking of the joint on movement. Sometimes this clicking can be mistaken for minor anterior subluxation. In younger patients a shearing injury results in a complex tear in the middle part of an otherwise normal disc. This can occur when the joint is both loaded and twisting, such as

in serving in tennis. In older patients there is usually pre-existing degenerative change present and the disc usually has torn from the superior periphery of the joint. There are often associated degenerative articular changes present within the joint.

An MRI scan can usually demonstrate a disc tear, which has a characteristic wavy appearance when compared to the normal ipsilateral side (Fig. 9.9). A CT scan is not able to demonstrate the disc.

An ultrasound-guided cortisone injection can be tried as the first line of treatment. If this is unsuccessful resection of the torn disc is indicated. This has previously been done as an open procedure but can now be undertaken arthroscopically. At surgery the whole of the disc is resected back to a stable rim. In the presence of a degenerative tear, resection of the medial end of the clavicle may also be



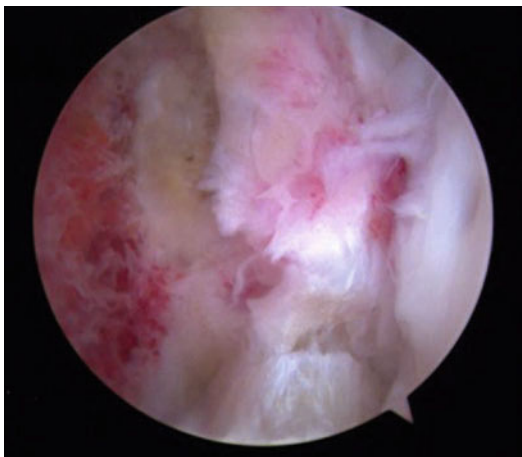
**Fig. 9.9** MRI scan (T2) demonstrating a wavy appearance of the superior disc with a small joint effusion. This represents a tear/detachment of the superior part of the disc from the capsule

undertaken if there are significant associated osteoarthritic symptoms.

## 9.5 SCJ Osteoarthritis

Osteoarthritis of the SCJ is relatively common in patients over the age of 50, particularly in women. It is usually asymptomatic and may present as a painless lump secondary to effusion and osteophytes. When symptomatic, patients complain of pain, crepitus and clicking. This is particularly on cross-body adduction and related to overhead sports such as tennis and golf.

Non-operative treatment including physiotherapy, NSAID medications and ultrasound-guided intra-articular steroid injection are adequate in the majority of cases. Occasionally, in patients with unremitting symptoms, resection of the degenerate disc and the medial end of the clavicle are indicated. When undertaken as



**Fig. 9.10** Arthroscopic excision of the medial end of the right clavicle. On the left of the image the medial end of the clavicle has been resected leaving exposed cancellous bone. The posterior rim of the resected disc can be seen centrally with the preserved articular cartilage of the sternum beside it

an open procedure the patient requires a period of immobilisation to protect the repaired anterior SCJ ligament. It is now possible to do this arthroscopically as a day case without immobilisation [26] (Fig. 9.10).

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## 9.6 Miscellaneous SCJ Pathologies

The SCJ can be the focus of a disparate group of other pathologies including inflammatory arthropathies, crystal-deposition arthropathies (gout and pseudogout), SAPHO syndrome (synovitis, acne, pustulosis hyperostosis and osteitis) and CRMO (chronic relapsing multifocal osteomyelitis). These conditions are all rare, but the physician should be mindful of them as a potential differential diagnosis for a painful swollen SCJ. Initial investigations would be screening inflammatory markers (CRP, ESR) and either an MRI or CT scan.

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## Q&A

- (1) Describe the stabilising structures of the sternoclavicular joint.

The stabilising structures are dynamic and static soft tissue structures. The static stabilisers are the inherent congruity of the articular surfaces of the medial end of the scapula and the sternal articulation, the anterior and posterior sternoclavicular ligaments, fibrocartilagenous disc, interclavicular and the costoclavicular ligament. The dynamic stabilisers make up the musculo-tendinous envelope around the joint.

- (2) How would you classify sternoclavicular joint instability?

The Stanmore triangle is a useful system that takes into account the range of instabilities of the SCJ.

- (3) What type of dislocation may lead to mediastinal compromise and what are the features?



Posterior mainly, with dysphagia, dyspnoea and/or vascular symptoms of venous congestion or oedema of the ipsilateral arm.

- (4) Why is atraumatic instability of the SCJ more commonly in an anterior direction?

A much greater direct force is required to achieve a posterior dislocation compared to anterior.

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## References

1. Lee JT, Campbell KJ, Mischalski MP, et al. Surgical anatomy of the sternoclavicular joint. *J Bone Joint Surg Am.* 2014;96(e166):1–10.
2. Warth RJ, Lee JT, Millett PJ. Anatomy and biomechanics of the sternoclavicular joint. *Oper Tech Sports Med.* 2014;22:248–52.
3. Emura K, Arakawa T, Terashima T, Miki A. Macroscopic and histological observations on the human sternoclavicular joint disc. *Anat Sci Int.* 2009;84(3):182–8.
4. Renfree KJ, Wright TW. Anatomy and biomechanics of the acromioclavicular and sternoclavicular joints. *Clin Sports Med.* 2003;22(2):219–37.
5. Spencer EE, Kuhn JE, Huston LJ, et al. Ligamentous restraints to anterior and posterior translation of the sternoclavicular joint. *J Shoulder Elb Surg.* 2002;11:43–7.
6. Tubbs RS, Loukas M, Slappegly JB, et al. Surgical and clinical anatomy of the interclavicular ligament. *Surg Radiol Anat.* 2007;29:357–60.
7. Tubbs RS, Shah NA, Sullivan BP, et al. The costoclavicular ligament revisited: a functional and anatomical study. *Romanian J Morphol Embryol.* 2009;50(3):475–9.
8. Koch MJ, Wells L. Proximal clavicle physeal fracture with posterior displacement: diagnosis, treatment, and prevention. *Orthopaedics.* 2012;35(1):e108–11.
9. Webb PA, Schuey JM. Epiphyseal union of the anterior iliac crest and medial clavicle in a modern multiracial sample of American males and females. *Am J Phys Anthropol.* 1985;68:4567–466.
10. Ludewig PM, Behrens SA, Meyer SM, et al. Three-dimensional clavicular motion during arm elevation: reliability and descriptive data. *J Orthop Sports Phys Ther.* 2004;34:140–9.
11. Mehta JC, Sachdev A, Collins JJ. Retrosternal dislocation of the clavicle. *Injury.* 1973;5:79–83.
12. Wirth MA, Rockwood CA. Acute and chronic traumatic injuries of the sternoclavicular joint. *J Am Acad Orthop Surg.* 1996;4:268–78.

13. Thut D, Hergan D, Dukas A, et al. Sternoclavicular joint reconstruction: a systematic review. *Bull NYU Hosp Jt Dis.* 2011;69:128–35.
14. Tepolt F, Carry PM, Heyn PC. Posterior sternoclavicular joint injuries in the adolescent population: a meta-analysis. *Am J Sports Med.* 2014;42(10):2517–24.
15. Deutsch AL, Resnick D, Mink JH. Computed tomography of the glenohumeral and sternoclavicular joints. *Orthop Clin North Am.* 1985;16:497–511.
16. Yeh GL, Williams GR. Conservative management of sternoclavicular injuries. *Orthop Clin North Am.* 2000;31:189–203.
17. Sewell MD, Al-Hadithy N, Le Leu A, Lambert SM. Instability of the sternoclavicular joint. Current concepts in classification, treatment and outcomes. *Bone Joint J.* 2013;95B(6):721–31.
18. Rockwood CA, Wirth MA. Injuries to the sternoclavicular joint. In: Rockwood CA, Green DP, Bucholz RW, Heckman JD, editors. *Rockwood and Green's fractures in adults.* 4th ed. Philadelphia: Lippincott-Raven; 1996. p. 1415–71.
19. Rockwood CA. Dislocations of the sternoclavicular joint. In: Evans EB, editor. *American Academy of Orthopaedic Surgeons instructional course lectures*, vol. XXIV. St Louis: CV Mosby; 1975. p. 144–59.
20. Nordqvist A, Petersson C. The incidence of fractures of the clavicle. *Clin Orthop Relat Res.* 1994;300:127–32.
21. Lyons FA, Rockwood CA Jr. Migration of pins used in operations on the shoulder. *J Bone Joint Surg Am.* 1990;72-A:1262–7.
22. Rockwood CA Jr, Odor JM. Spontaneous atraumatic anterior subluxation of the sternoclavicular joint. *J Bone Joint Surg Am.* 1989;71-A:1280–8.
23. Spencer EE, Kuhn JE. Biomechanical analysis of reconstructions for sternoclavicular joint instability. *J Bone Joint Surg Am.* 2004;86(1):98–105.
24. Martin SD, Altcheck D, Erlanger S. Atraumatic posterior dislocation of the sternoclavicular joint: a case report and literature review. *Clin Orthop Relat Res.* 1993;292:159–64.
25. Sonazaki H, Mitsui H, Miyanaga Y, et al. Clinical features of 53 cases with pustulotic arthro-osteitis. *Ann Rheum Dis.* 1981;40:547–53.
26. Tytherleigh-Strong G, Griffiths D. Arthroscopic excision of the sternoclavicular joint for the treatment of sternoclavicular osteoarthritis. *Arthroscopy.* 2013;29:1487–91.



# Clavicle Fractures

# 10

David Copas and Michael Walton

## Key Learning Points

- Clavicle fractures are common sports injuries due to the relative weakness of the mid shaft.
- Plain radiography is usually adequate although CT scanning can be useful in complex fracture patterns and medial fractures
- Prognostic indicators can guide decision-making based on union rates.
- If indicated, surgical fixation leads to high rates of union and return to sports

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## 10.1 Introduction

The shoulder is a complex closed chain mechanism comprised of four joints: the sternoclavicular, the acromioclavicular and the glenohumeral joints and the scapulothoracic articulation. Each joint is unique in both form and function but combine to provide a stable base to position the arm and hand in space. The clavicle is an s-shaped bone, which is longer in the male with more pronounced curves. It is thicker in the athlete or manual worker. The medial two-thirds are rounded with a forward convexity at the

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sternal end. The lateral third is flatter and curves back to join the scapula. The clavicle is the only skeletal attachment of the scapula to the torso, therefore, an injury to the clavicle affects the whole shoulder biomechanics.

The clavicle is the first bone to ossify in the foetus. This occurs from three centres. The two primary centres, medial and lateral, form the body from the fifth week in utero. The secondary centre forms the sternal end, which appears in the late teens and only fuses with the body of the clavicle by the 25th year. The medial end articulates with the manubrium and the cartilage of the first rib, forming a saddle type joint. This is a strong, stable joint allowing movement mostly in anteroposterior and vertical planes. Its stability arises from a complex arrangement of ligaments and capsule that require considerable force to disrupt them. The acromioclavicular joint comprises a laterally facing facet that articulates with the acromion. Stability relies on the capsule and the coracoclavicular ligaments, and in the joint is a meniscal homologue.

There are numerous muscle attachments to the clavicle. The sternocleidomastoid attaches to the medial side, and on the lateral side the pectoralis major and the deltoid have their origins. This leaves a potential exposed midshaft, which is also the thinnest segment. This area is, therefore, more predisposed to fracture.

Clavicle fractures have traditionally been treated conservatively. However, recent studies have suggested benefits with surgery of lower rates of non-union and symptomatic malunion [1]. This chapter will attempt to give a pragmatic approach to the fractured clavicle.

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## 10.2 Epidemiology of Fractures

Clavicle fractures are common with an incidence of up to 5% in the adult population [2, 3]. The vast majority arise in the midshaft (70–80%) [3, 4], with lateral-end injuries being next most frequent and medial being relatively rare.

The epidemiology varies with gender, with the male-to-female ratio being approximately 3:1 [3, 5]. In males, the annual incidence is highest under 20-years-of-age and then decreases every decade until the seventh. In females, the incidence, although

slightly higher in the teens and the elderly, is relatively constant. The young age group is highly associated with sports injuries, with a recent American study showing that sport was a factor in 45% of clavicle fractures [5].

The importance of high-energy clavicle fractures in the context of the polytrauma is being increasingly recognised. Early fixation is being instigated in patients with significant associated chest injury. Operative stabilisation has been associated with decreased length of stay in ITU following thoracic trauma.

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### 10.3 Presentation and Investigation

The diagnosis of a clavicle fracture is normally straight-forward. It is important to obtain an accurate assessment of the mechanism of injury as well as the patient's overall medical condition, recreational activities and occupation. It is important to understand the patient's expectations with regards to return to higher levels of physical functioning, especially repetitive overhead activity.

The biomechanics of the majority of clavicle fractures is a fall or direct blow to the shoulder leading to a compressive axial force [6, 7]. The zone of transition in the midshaft of the clavicle fractures as a result of its relative weakness. The fracture, if displaced, can then shorten as a result of unopposed muscular action. The sternocleidomastoid pulls the medial fragment superiorly and posteriorly, the lateral end moves inferiorly and laterally by the action of pectoralis major and the deltoid. This results in the usual deformity of the "ptotic" shoulder, representing the inferior and medial displacement of the distal fragment. In the case of significant deformity, care should be taken to assess the overlying skin to ascertain whether it is under threat. This "tenting" of the skin potentially affects the decision-making process.

It is possible to measure the length of the clavicle clinically from the sternal notch to the acromioclavicular joint on both sides and record the difference. Careful neurological examination of the involved arm is performed, as well as a assessment for any other associated injuries including sternoclavicular joint, AC joint, proximal humerus and pathology distally in the arm.

Investigations start with conventional radiography. AP and cephalic oblique (15–45°) views should be obtained. A serendipity view can be obtained to look at medial third fractures and injuries involving the sternoclavicular joint. This view is taken with the beam aimed at the manubrium with a 40° cephalic tilt.

Computed tomography is only occasionally required in the assessment of clavicle fractures. Its most important role is evaluation of medial clavicular fractures and injuries affecting the SC joint when plain images are not sufficient to do so. CT scans should include the SC joints and at least half of both clavicles to allow for side-to-side comparison. It may also be useful in segmental fractures to allow the morphology of the fracture to be studied in order to plan a surgical intervention. If vascular compromise is a concern, the study can be performed with intravenous contrast enhancement.

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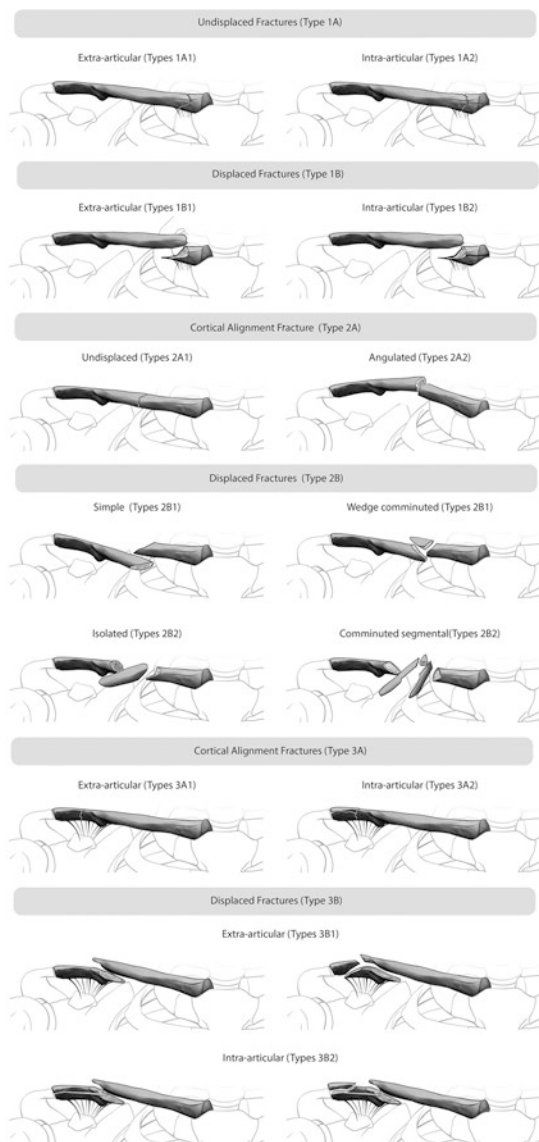
## 10.4 Classification

Classification systems should be reliable, reproducible, serve as a basis for treatment and ideally a predictor of outcome. It allows a common language to communicate and allows robust research. There are a number of classifications for fractures of the clavicle but most are, to some extent, compromised in delivering these goals [8, 9].

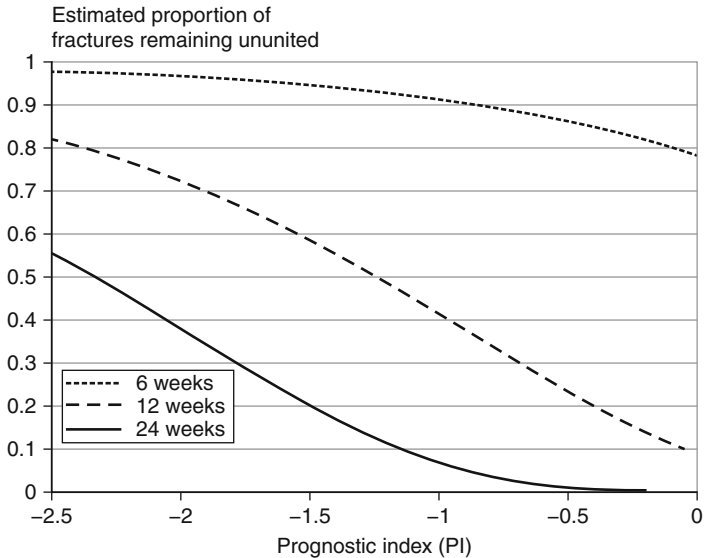
Our favoured system is the Robinson Classification [3]. This classification provides a reliable prognostic guide by identifying subgroups at risk of developing complications. It is possible to focus treatment and resources at these high-risk groups.

Three distinct regions of the bone are identified; the medial and lateral fifths, and the central three-fifths. The most common, mid-shaft, type 2, injuries are subdivided by angulation, displacement and comminution. The medial and lateral injuries are classified by the presence of intra-articular involvement. Robinson's paper demonstrated good reliability and reproducibility in his population of a 1000 fractures (Fig. 10.1).

The Robinson Classification system further has been used to identify risk factors for non-union ([10], Fig. 10.2). Age, sex,



**Fig. 10.1** Robinson classification of clavicle fractures



**Fig. 10.2** Prognostic index for union in mid-shaft clavicle fractures

degree of displacement and comminution were all associated with non-union. From these relative risks, a calculation tool was produced, in an attempt to predict the risk of non-union.

**Prognostic index** =  $[-0.85 \times (1 \text{ if displaced or } 0 \text{ if undisplaced})] + [-0.36 \times (1 \text{ if female or } 0 \text{ if male})] + [-0.37 \times (1 \text{ if comminuted fracture or } 0 \text{ if noncomminuted fracture})] + [-0.01 \times (\text{age of patient in years})]$ .

For example, a sixty-year-old woman with a displaced, comminuted diaphyseal fracture (Prognostic Index  $-2.18$ ) has an approximately 75% projected probability that the fracture will be un-united at 12 weeks and a 45% probability of non-union at 24 weeks; whereas a patient with a prognostic index of  $-1.05$  (for



example, a 20-year-old man with a displaced but noncomminuted fracture) has a 40% probability that the fracture will remain ununited at 12 weeks and only a 7.5% probability of non-union at 24 weeks. This suggests that earlier surgery might be beneficial for patients with a lower prognostic index.

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## **10.5 Management**

### **10.5.1 Midshaft (Robinson Type 2)**

Undisplaced midshaft fractures (type 2A) can be treated conservatively. They usually unite and patients return to excellent levels of function. Management of the displaced fracture, however, remains controversial. Historic evidence that the majority of fractures heal and patients are satisfied with their outcomes has been challenged with more recent studies suggesting that the non-union rate may be higher and that poorer outcomes may be present with conservatively treated displaced fractures.

### **10.5.2 Non-operative Treatment**

The broad arm sling is the most frequently used non-operative treatment. It has better patient satisfaction scores but identical functional outcomes when compared to the figure of eight bandage [11]. The latter also has a higher rate of non-union. It is important to note that neither sling type can reduce a displaced, shortened fracture and hence will always result in a degree of malunion.

The sling is used for comfort only. As function returns and pain recedes it can be discarded. This normally signifies bone healing and a return to activities.

### 10.5.3 Primary Operative Treatment

The Canadian Orthopaedic Trauma Association reported the results of a multicentre, randomised, controlled trial for conservative versus operative management for displaced midshaft clavicle fractures. They concluded that operative intervention resulted in better outcomes at all-time points with lower non-union rates [12]. However, a meta-analysis, by the same authors, assessing six randomised controlled studies totaling 412 patients, whilst concluding the superiority of operative over non-operative management, found no significant evidence that the long term functional outcome in the operated group was superior to the non-operative group [1]. A similar large multicentre UK trial [13] demonstrated that open reduction and internal fixation reduced the rates of non-union in displaced midshaft fractures when compared with non-operative treatment, and that this was associated with better functional outcomes. However, their principal conclusion was that the improved outcome scores resulted largely from the prevention of the non-union, which resulted in much poorer function. There was little functional difference between the groups of surgically and conservatively managed fractures that went on to union. The authors also highlighted the higher complication rates and the greater expense with surgical intervention.

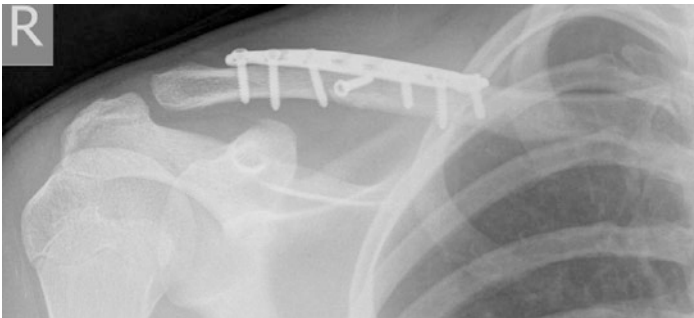
A Cochrane review in 2013 stated that there was limited evidence from RCTs on the relative effectiveness of surgical versus conservative treatment for acute middle-third clavicle fractures [14]. Their conclusions were that treatment should be chosen on an individual basis, after careful consideration of the relative benefits and risks of each modality. It is clear also that patients need to be included in the discussion to explore their expectations and demands.

Our current practice is for undisplaced fractures to be treated conservatively with broad arm sling, and clinical review with radiographs to ensure no further displacement, and progression to union. Displaced fractures are treated on an individual basis. In our practice we attempt to assess the likelihood and impact of a subsequent non-union principally in those patients with clinical or radiological shortening of more than 2 cm and usually with the typical “ptotic” appearance of the shoulder. This group of young, active patients, especially those who plan to return to high-level

athletic activities or occupations involving repetitive lifting or overhead activity, are likely to benefit from early surgery in terms of speed of recovery and predictability of outcome. Any intervention should be performed by an experienced surgeon to minimise the risk of complications.

### 10.5.4 Open Reduction and Internal Fixation

Plate fixation is the most common surgical intervention. Care is taken to preserve the supraclavicular nerve and its branches. Current modern implant technology has allowed the development of anatomically contoured specific clavicle fracture plates with dynamic compression locking screws. These allow anatomical restoration of the bony anatomy, whilst being less prominent and hence reducing the requirement for plate removal. The plates are typically placed on the superior surface of the clavicle, which has been shown to be biomechanically beneficial [15] and requires minimal soft tissue detachment. Lag screw techniques can be used to provide compression and to stabilise butterfly fragments in Type 2B1 and 2B2 injury patterns (Fig. 10.3). Anterior plating can also be utilized and proponents highlight that this approach reduces plate prominence and the need for subsequent removal.



**Fig. 10.3** Open reduction and internal fixation of a 2B1 clavicle fracture

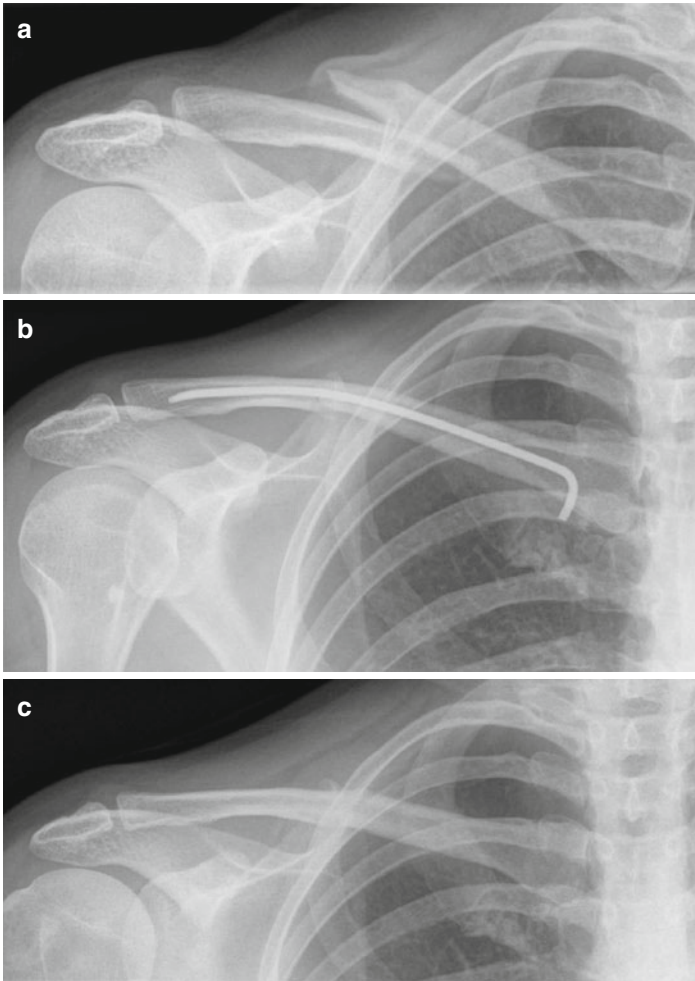
Care must be taken to protect the structures in the subclavicular region, as overzealous drilling and tapping could compromise inferior structures. Post-operatively radiographs are obtained and a neurovascular assessment of the limb is performed and documented. A poly sling is applied for comfort and simple pendular exercises are commenced. Most athletes can return to training between 3–6 weeks and competition between 6–12 weeks. Non-union following careful fixation is rare.

### **10.5.5 Intramedullary Fixation (IM Fixation)**

IM techniques have the advantages of using smaller incisions, less dissection and soft tissue stripping, relative protection of the supraclavicular nerves and potentially shorter operating times. Historical techniques using pins or modified nails have had serious reported complications, such as intra-thoracic migration and damage to the underlying structures. These were rigid devices inserted from the lateral fragment. Elastic titanium nails have become increasingly popular for the fixation of many long-bone fractures, particularly in children. They work by providing a three-point fixation in the medulla of the bone and are not prone to the problems of previous nails and pins. It is a relative stability construct that heals by callus. This can be rapid and there is evidence that it is useful in athletes, who seek an early return to sport [16]. There is increasing evidence for their use in clavicular fractures. However, the nails are unable to be statically locked, which results in the implant being axially and rotationally less stable. Therefore, the construct is significantly weaker than plate fixation. In our practice, we use IM fixation in longitudinally stable fractures (simple, displaced, midshaft, Type 2B1), in athletes and adolescents, giving good functional and cosmetic results. The nail tends to be removed at around three-six months.

Our technique involves the use of an antegrade titanium elastic nail. The fracture site is opened via a small incision over the fracture site. The fracture is then reduced under direct vision to confirm longitudinal stability. The nail is then passed retrograde into the medial fragment and out of the anteromedial cortex. The frac-

ture is then reduced and the nail passed antegrade into the distal fragment under fluoroscopic control. The wire is cut short and the wounds closed (Fig. 10.4a–c). The post-operative rehabilitation is the same as for plate fixation.



**Fig. 10.4.** (a) Preoperative film of displaced mid-shaft fracture. (b) 6-weeks post-operation. (c) After removal of nail at 6 months

## **10.6 Complications of Clavicle Fracture Management**

### **10.6.1 Non-union**

Historically, the rate of non-union of clavicle fractures was felt to be low but increasingly the rate has been shown to be more significant. In a large meta-analysis of all series of displaced mid-shaft clavicle fractures between 1975 and 2005, the non-union rate in non-operatively treated fractures was 15.1% compared with just 2.2% in those that were operatively treated [17]. Increasing age, female sex, shortening of greater than 2 cm, complete fracture displacement, smoking history and comminution are considered risk factors. The Robinson calculator in the classification section can be useful in trying to identify those at higher risk of non-union.

Non-unions present with pain, clicking, weakness and restriction of movement. The end result can be a significant reduction in function, affecting work, sport and daily activities. The diagnosis is clinical with radiological confirmation, either with plain radiographs or computed tomography.

In the presence of a symptomatic non-union, our preferred option is an open approach with plate fixation. In hypertrophic non-unions there is normally a failure of mechanical stability leading to a level of movement that precludes conversion of soft callus to hard callus. Rigid fixation should create an environment to promote bone union. In atrophic non-unions then there potentially exists a failure of biology and stability may be causal. Therefore, rigid fixation and biological stimulation is required, either by the process of judet decortication, drilling the medullary canals or bone grafting. The results of plate fixation are reliable and union rates post-surgery are high, and the complication rate is low [18, 19].

### **10.6.2 Malunion**

Clavicular malunions were believed to be of cosmetic interest but functionally they were well tolerated [20, 21]. However, with the advent of patient reported outcome measures it is becoming increasingly apparent that this is not always the case. Symptoms

of pain, loss of strength, rapid fatigability as well as cosmetic concerns have all been noted in the literature [22–24].

The shortening and rotational deformity that occurs with a malunion may lead to a number of biomechanical changes. The shortening of the clavicular lever arm of the shoulder leads to relative protraction of the scapula. This may lead to changes in the orientation of the glenoid and reciprocal winging of the scapula. The shortening of the clavicle may also have an effect on the muscle function around the shoulder. The muscle tension can be decreased and balance of the forces around the shoulder altered leading to weakness and decrease in endurance, with patients undergoing fatigue earlier [23, 25]. This may lead to functional problems, particular with overhead activities. It has also been suggested by Hill et al. [22] that an established malunion of the clavicle would lead to abnormal loading at the acromioclavicular and sternoclavicular joint. This, in turn, may lead to symptomatic arthrosis, early pain and decreased patient satisfaction.

Documentation of neurovascular problems and thoracic outlet syndrome has also been recorded following clavicle malunion. This can be due to bony encroachment on the neural structures by callus or fracture fragments. Again, these symptoms can be more profound in the overhead athlete [26].

Symptomatic malunion is uncommon, however if it does occur, the treatment algorithm follows a familiar course with non-operative treatments being utilised first. Patients may benefit from physiotherapy to improve muscle balance and power. However, for those who do not improve with therapy and lifestyle modification then surgery should be considered.

Surgery for malunions involves thorough pre-operative planning, with CT imaging being used to assess the plane for an osteotomy, which is typically in the original fracture plane. The set up for the procedure is similar to a primary approach, the malunion is exposed and a low-energy osteotomy performed. The medullary canals are opened with a drill to promote bony union following fixation and the fracture is reduced with careful attention to re-establishing original length and rotation. Plate fixation is used in a compression mode to create a rigid environment for direct bony healing. The literature suggests that corrective osteotomies can improve the symptoms from a malunion [27, 28].

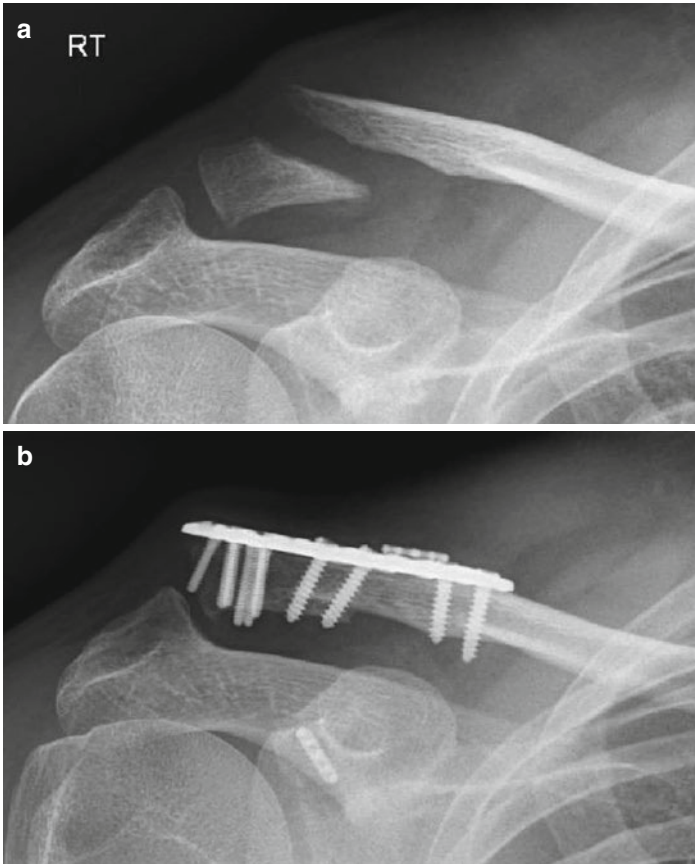
### 10.6.3 Lateral-Third Fractures

Lateral fractures of the distal clavicle occur in roughly one quarter of clavicle fractures. They are associated more with the elderly and females, and the mechanism is usually a simple fall. The majority of fractures occur around the coracoclavicular ligament complex and displacement occurs if this is defunctioned. The displaced lateral clavicle fracture has a very high rate of non-union (Fig. 10.5a) and consideration should be given to early fixation to stabilise the coracoclavicular complex (Fig. 10.5b). Surgery can be challenging due to the small quantity of bone stock. Non-displaced injuries, where the ligament complex integrity is maintained, can be managed non-operatively. The non-union rate is relatively high, at roughly a third. However, of these, only a third are symptomatic. Robinson et al. suggested a conservative approach to these injuries, using a sling. In cases of soft tissue compromise or a floating shoulder, a more aggressive surgical approach should be considered.

### 10.6.4 Medial-Third Fractures

These are rare injuries, that, due to the strong ligamentous structures, rarely displace. The vast majority can, therefore, be treated conservatively with a broad arm sling, analgesia and early mobilisation as pain allows. In those that do displace, careful assessment of the neurovascular structures is vital due to their close proximity posteriorly. If there is evidence of compromise due to posterior displacement, expeditious reduction should be performed in a setting capable of dealing with complications that might arise (vascular/thoracic surgical expertise). Displaced fractures may need open reduction and internal fixation.





**Fig. 10.5** (a) Lateral third clavicle fracture non-union. (b) Union following open reduction, internal fixation and coracoclavicular button supplementation

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## 10.7 Summary

Undisplaced fractures of the clavicle tend to be treated non-operatively. The management of the displaced fracture should be made on an individual basis. It is important to establish the expectations and the

demands of the patient and tailor the management accordingly. The young athlete, especially those who require repetitive overhead activity, do not tolerate non-union and malunion well. It is our practice, therefore, that patients with displaced, shortened or segmental fractures should be considered for early operative fixation to facilitate an early and predictable recovery.

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## Q&A

- (1) Describe the osteology of the clavicle?

The clavicle is an s-shaped bone, which is longer in the male with more pronounced curves. It is thicker in the athlete or manual worker. The medial two-thirds are rounded with a forward convexity at the sternal end. The lateral third is flatter and curves back to join the scapula

- (2) Why do clavicles most frequently fracture in the midshaft?

The sternocleidomastoid attaches to the medial side, and on the lateral side the pectoralis major and the deltoid have their origins. This leaves a potential exposed midshaft, which is also the thinnest segment. This area is, therefore, more predisposed to fracture

- (3) Which factors have a negative predictive value in the development of non-union?

Age, sex, degree of displacement and comminution were all associated with non-union.

- (4) Why do lateral third clavicle fractures displace?

The majority of fractures occur around the coracoclavicular ligament complex and displacement occurs if this is defunctioned.

- (5) Conservative management of clavicle fractures is best delivered with a broad arm sling, a collar and cuff sling or a Figure-8 bandage?

The broad arm sling is the most frequently used non-operative treatment. It has better patient satisfaction scores but identical functional outcomes when compared to the figure of eight bandage

## References

1. McKee RC, Whelan DB, Schemitsch EH, McKee MD. Operative versus nonoperative care of displaced midshaft clavicular fractures: a meta-analysis of randomized clinical trials. *J Bone Joint Surg Am.* 2012;94(8):675–84.
2. Postacchini F, Gumina S, De Santis P, Albo F. Epidemiology of clavicle fractures. *J Shoulder Elb Surg.* 2002;11(5):452–6.
3. Robinson CM. Fractures of the clavicle in the adult. Epidemiology and classification. *J Bone Joint Surg Br.* 1998;80(3):476–84.
4. Nordqvist A, Petersson C. The incidence of fractures of the clavicle. *Clin Orthop Relat Res.* 1994;300:127–32.
5. Van Tassel D, Owens BD, Pointer L, Moriatis Wolf J. Incidence of clavicle fractures in sports: analysis of the NEISS Database. *Int J Sports Med.* 2014;35(1):83–6.
6. Nowak J, Mallmin H, Larsson S. The aetiology and epidemiology of clavicular fractures. A prospective study during a two-year period in Uppsala, Sweden. *Injury.* 2000;31(5):353–8.
7. Stanley D, Trowbridge EA, Norris SHJ. The mechanism of clavicular fracture. A clinical and biomechanical analysis. *Bone Joint Surg Br.* 1988;70(3):461–4.
8. Allman FL Jr. Fractures and ligamentous injuries of the clavicle and its articulation. *J Bone Joint Surg Am.* 1967;49(4):774–84.
9. Neer CS. Fracture of the distal clavicle with detachment of the coracoclavicular ligaments in adults. *J Trauma.* 1963;3:99–110.
10. 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.
11. 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.
12. Canadian Orthopaedic Trauma Society. Nonoperative treatment compared with plate fixation of displaced midshaft clavicular fractures. A multicenter, randomized clinical trial. *J Bone Joint Surg Am.* 2007;89(1):1–10.
13. Robinson CM, Goudie EB, Murray IR, Jenkins PJ, Ahktar MA, Read EO, Foster CJ, Clark K, Brooksbank AJ, Arthur A, Crowther MA, Packham I, Chesser TJ. Open reduction and plate fixation versus nonoperative treatment for displaced midshaft clavicular fractures: a multicenter, randomized, controlled trial. *J Bone Joint Surg Am.* 2013;95(17):1576–84.
14. Lenza M, Buchbinder R, Johnston RV, Belloti JC, Faloppa F. Surgical versus conservative interventions for treating fractures of the middle third of the clavicle. *Cochrane Database Syst Rev.* 2013;6:CD009363.

15. Iannotti MR, Crosby LA, Stafford P, Grayson G, Goulet R. Effects of plate location and selection on the stability of midshaft clavicle osteotomies: a biomechanical study. *J Shoulder Elb Surg.* 2002;11(5):457–62.
16. Jubel A, Andemahr J, Bergmann H, Prokop A, Rehm KE. Elastic stable intramedullary nailing of midclavicular fractures in athletes. *Br J Sports Med.* 2003;37(6):480–3.
17. Zlowodzki M, Zelle BA, Cole PA, Jeray K, McKee MD, Evidence-Based Orthopaedic Trauma Working Group. 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.
18. Ebraheim NA, Mekhail AO, Darwich M. Open reduction and internal fixation with bone grafting of clavicular nonunion. *J Trauma.* 1997;42(4):701–4.
19. Olsen BS, Vaesel MT, Søjbjerg JO. Treatment of midshaft clavicular nonunion with plate fixation and autologous bone grafting. *J Shoulder Elb Surg.* 1995;4(5):337–44.
20. Nordqvist A, Redlund-Johnell I, von Scheele A, Petersson CJ. Shortening of clavicle after fracture. Incidence and clinical significance, a 5-year follow-up of 85 patients. *Acta Orthop Scand.* 1997;68(4):349–51.
21. Oroko PK, Buchan M, Winkler A, Kelly IG. Does shortening matter after clavicular fractures? *Bull Hosp Jt Dis.* 1999;58(1):6–8.
22. Hill JM, McGuire MH, Crosby LA. Closed treatment of displaced middle-third fractures of the clavicle gives poor results. *J Bone Joint Surg Br.* 1997;79(4):537–9.
23. McKee MD, Pedersen EM, Jones C, Stephen DJ, Kreder HJ, Schemitsch EH, Wild LM, Potter J. Deficits following nonoperative treatment of displaced midshaft clavicular fractures. *J Bone Joint Surg Am.* 2006;88(1):35–40.
24. Rosenberg N, Neumann L, Wallace AW. Functional outcome of surgical treatment of symptomatic nonunion and malunion of midshaft clavicle fractures. *J Shoulder Elb Surg.* 2007;16(5):510–3.
25. Ledger M, Leeks N, Ackland T, Wang A. Short malunions of the clavicle: an anatomic and functional study. *J Shoulder Elb Surg.* 2005;14(4):349–54.
26. Kitsis CK, Marino AJ, Krikler SJ, Birch R. Late complications following clavicular fractures and their operative management. *Injury.* 2003;34(1):69–74.
27. Hillen RJ, Eygendaal D. Corrective osteotomy after malunion of mid shaft fractures of the clavicle. *Strategies Trauma Limb Reconstr.* 2007;2(2-3):59–61.
28. McKee MD, Wild LM, Schemitsch EH. Midshaft malunions of the clavicle. *J Bone Joint Surg Am.* 2003;85-A(5):790–7.



# Glenohumeral Arthritis in Athletes

# 11

Peter A. D'Alessandro  
and Andrew L. Wallace

## Key Learning Points

- Awareness of signs and symptoms related to chondral damage allows for early diagnosis and optimum management
- Chondral damage may be associated with other shoulder pathology and may be difficult to accurately diagnose with imaging alone
- The athlete should be educated as to the natural history of arthritis, and manage their activity and expectations accordingly
- Management options focused on maintaining the athlete's career are generally non-operative, though arthroscopic procedures in particular can be beneficial
- Other operative options are aimed at career salvage at best; but may be career ending

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## 11.1 Introduction

The development of chondral damage in any major joint can be devastating for an athlete, and is a common cause for premature retirement [1]. Chondral damage can vary from isolated minimally symptomatic chondral lesions through to fulminant bipolar arthritis. Arthritis in the glenohumeral joint is generally defined as the presence of an Outerbridge grade III or IV chondral lesion on either side of the joint, signifying loss of more than 50% of the cartilage depth [2, 3]. This damage can lead to severe loss of function particularly in repetitive or throwing athletes and those involved in impact sports. This is compounded by the limited treatment options available to treat the disease in this group of patients [4]. Although surgical treatments including arthroplasty have been effective in managing this disease in older patients with low functional demands, the treatment of young patients with glenohumeral arthritis provides a significant challenge [5–7].

Most orthopaedic literature investigating glenohumeral arthritis is either focused on the elderly, in whom arthroplasty is clearly indicated and generally successful, or relatively ‘young,’ but still middle-aged patients who are leading a somewhat more active lifestyle [5, 8, 9]. However, the physical demands and functional expectations in these patients is significantly different from an even younger group of patients who are also professional or recreational athletes. These patients’ primary aim is generally to maximise function and performance whilst prolonging the length of their careers, and the treating team must be acutely aware of the individual goals of each patient to provide the best possible outcome in terms of function, performance and long-term outcome.

This chapter will specifically focus on the management of this rare but important cohort of professional and recreational athletes with glenohumeral chondral damage and arthritis. To assist the multidisciplinary team treating these patients, management options have been divided into three groups: those most effective at maintaining a career, salvage treatments that can be considered after failure of less invasive measures and finally, career-ending options that are not generally conducive with most sports and require cessation of athletic activities.

## **11.2 Aetiology**

### **11.2.1 Primary Osteoarthritis**

Whilst the specific causes of primary osteoarthritis are unknown, there is almost certainly a significant genetic component of the disease. Truly idiopathic primary disease of the glenohumeral joint is relatively rare, affecting less than 3% of all patients with arthritis [10]. This group of patients is more likely to have diffuse and bipolar disease, generally aged over 60 years, more likely to be female, and have degeneration affecting multiple joints [11]. Typically, this form of arthritis results in posterior glenoid wear and eventual posterior humeral head subluxation, while rotator-cuff tears in this population are uncommon [5].

### **11.2.2 Secondary Arthritis**

The role of isolated chondral lesions in the natural history of symptomatic glenohumeral arthritis is not clear, and many chondral lesions are tolerated well in young patients [5]. However, symptomatic chondral damage secondary to trauma, in particular acute or recurrent instability, is the most common cause of arthritis in the athletic population. Up to 10% of patients with instability have intra-operative evidence of a significant chondral injury [12]. Patients who have suffered a dislocation have up to a 20-times greater risk of developing glenohumeral arthritis than the general population [13, 14]. Athletes involved in repetitive overhead activities, such as in the dominant arm of tennis players or throwing athletes, can often develop internal impingement. This is associated with occult chondral trauma, which may also predispose to arthritis [10, 15, 16]. Chondral damage may also be caused by other pathological processes including osteonecrosis, osteochondritis dissecans or be the sequelae of infection [2].

Iatrogenic causes are rare but important, in particular chondrolysis following arthroscopic surgery. The implantation of bio-absorbable suture anchors and the use of radiofrequency probes have been suggested as possible contributing factors [17].

However, the vast majority of iatrogenic cases have been associated with the use of intra-articular infusion pumps containing the local anaesthetic bupivacaine during the 2000s. This led to fulminant chondrolysis and extensive glenohumeral arthritis in young patients within the months following their index procedure [18–20]. Importantly there has not been a similar association found with isolated injections of local anaesthetic, and awareness of the complications associated with intra-articular infusion pumps should reduce the future incidence of cases of post-operative chondrolysis [21, 22].

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## **11.3 Diagnosis**

### **11.3.1 Signs and Symptoms**

The athlete with glenohumeral arthritis may present with non-specific signs and symptoms that require a careful diagnostic assessment to determine the underlying cause. Other more common shoulder pathology including partial thickness rotator cuff tears, biceps tendinopathy, subacromial bursitis or labral tears may also be present. A narrow focus on these can potentially delay the diagnosis and management of chondral damage, which can have a deleterious impact on the athlete's career.

As with any patient, a detailed history should be taken from the athlete, with a particular focus on episodes of trauma, dislocation or previous surgery. It is important to elucidate more understated symptoms, including subtle episodes of instability or a slightly reduced ability to undertake previously performed tasks or activities. Night pain is common, with a deep ache associated with an inability to sleep on the affected side. Clicking, grinding and transient locking with sudden pain that limits activity are also frequently reported [10]. It is important to be aware that athletes may be functionally disabled by symptoms that would not necessarily affect the average member of the public [1]. Many of these athletes may continue to play and function, but will report gradual deterioration in their capability to perform at their expected level.



Examination findings include peri-scapular muscle wasting and crepitus, which may be audible or palpable. The joint line may be painful, with posterior joint-line tenderness in particular a more specific sign of chondral damage. Range of motion restriction may be quite subtle in the earlier stages of arthritis, with pain compounding this stiffness most commonly in external rotation. It is important to be aware of the patient with a ‘painful arc’ of movement who reproduces pain in the low-mid ranges of motion, as this may be more suggestive of chondral damage rather than mechanical impingement [2, 23].

## 11.3.2 Investigations

### 11.3.2.1 Radiographs

A series of plain radiographs including anteroposterior (Fig. 11.1), scapular lateral and an axillary view should be taken. Assessing the degree of arthritis is mostly reliably described using the

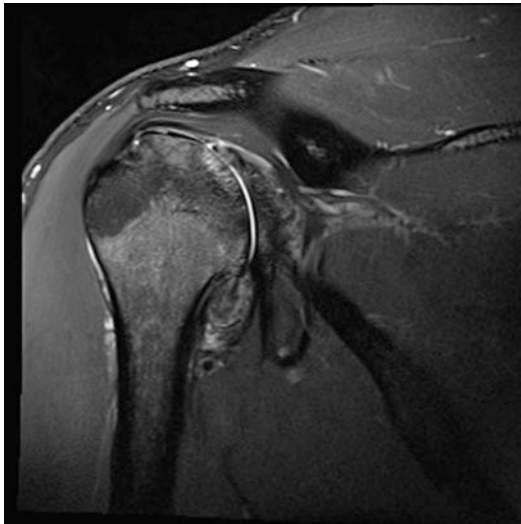


**Fig. 11.1** AP radiograph of the left shoulder demonstrating osteoarthritis with joint space narrowing, subchondral sclerosis and humeral osteophyte formation

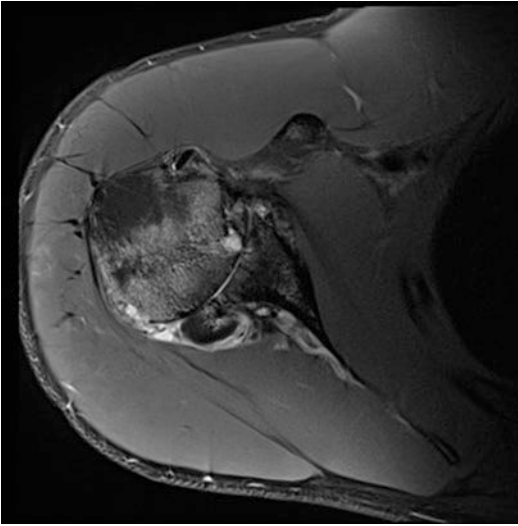
Samilson and Prieto classification based on the AP radiograph [24, 25]. It divides patients into categories of mild, moderate and severe depending on the size of osteophytes and extent of glenohumeral irregularity and joint space narrowing.

### 11.3.2.2 MRI and CT

The chondral surfaces in the glenohumeral joint are much thinner than those in the knee, measuring around 1.5 mm compared with up to 10 mm [26, 27]. Magnetic Resonance Imaging (MRI) scanning for arthritis lacks sensitivity and specificity, with even specialised cartilage sequences and MRI arthrography only having a moderate ability to correctly diagnose subtle chondral lesions [9, 28]. However, more profound chondral loss is more easily evident (Figs. 11.2 and 11.3).



**Fig. 11.2** T2 weighted coronal MRI scan demonstrating features of osteoarthritis



**Fig. 11.3** T2 weighted axial MRI scan demonstrating osteoarthritis with posterior humeral translation and almost complete chondral loss

Computed Tomography (CT) scans can also be helpful, particularly in quantifying glenoid wear or in the context of previously inserted metal soft tissue anchors leading to artefact and poor MRI image quality. Two and three dimensional image reconstructions can assist with more exact localisation of osteophytes and loose bodies (Fig. 11.4). Due to the limitations of imaging modalities in accurately diagnosing chondral damage, diagnostic arthroscopy remains the gold standard for diagnosis, particularly in joints such as the shoulder with thinner layers of articular cartilage [9, 29].



**Fig. 11.4** 3D CT reconstruction demonstrating circumferential humeral osteophyte formation

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## 11.4 Management Options

The principles and goals of managing the athlete with glenohumeral arthritis are understandably different from those of the general patient population. The competing professional athlete generally aims to maintain a career at the highest level for as long as possible. The recreational athlete may have slightly different priorities, and is often content to continue to participate in their chosen sport albeit at a lower level. Thus a solid understanding and appropriate management of the patient's aims, goals and expectations is critical to achieving a positive outcome in degenerative conditions such as arthritis that have a natural history of gradual deterioration. The athlete must be educated as to their condition, taught to interpret symptoms and contribute to their treatment plan so they can assume some responsibility and control

over its implementation. They should understand the aims of each treatment in the context of the overall prognosis of their condition, so they can adjust their expectations and in many cases, consider their career direction and planning for the future [1].

Treatments for degenerative joint disease have traditionally been divided into palliative, reparative, restorative and reconstructive options [9]. However, as this chapter focuses on the management of athletes, particularly those involved in professional or higher level recreational pursuits, we have categorised each treatment modality in the context of the athlete's ability to meet the functional demands required to continue their career.

### **11.4.1 Career Maintaining**

The following are all appropriate treatment options for the athlete with early or moderate stage arthritis who is aiming to continue to perform at the highest possible level. The objective is to mitigate against symptoms that may limit some aspects of higher level function. This group of treatments will be the most commonly utilised in athletes as they are less invasive with lower associated morbidity. Successful outcomes in the short term can often be achieved, however the natural history of deterioration of arthritis will generally determine the longer-term effect on the athlete's career. In professional sport, it is essential that the treating team liaise closely with the relevant governing body to ensure that all medications and injections used comply with the World Anti-Doping Agency (WADA) code, and if necessary a therapeutic use exemption obtained.

#### **11.4.1.1 Oral NSAIDs and Analgesics**

As function is paramount in the athlete, a key aim of treating their arthritis is to effectively manage pain during activity, as pain itself is a significant contributor to impaired function. Oral analgesics including paracetamol should be used as part of a regular tailored analgesic regime, whilst medications containing codeine are most appropriate for use at night. Intermittent use of non-steroidal anti-inflammatory medications (NSAIDs), particularly around high

intensity training sessions or on game day can be helpful, though care must be taken due to the risk of gastrointestinal, renal and possible cardiovascular side effects with prolonged use [30].

#### **11.4.1.2 Nutritional Supplements**

Glucosamine and chondroitin sulphate are components of the extracellular matrix of articular cartilage. The former is derived from crustacean exoskeletons, whilst the latter is extracted from animal cartilage [31]. A seminal paper based on a high-quality randomised controlled trial published in *The Lancet* in 2001 concluded that the use of glucosamine in patients with knee osteoarthritis could alter the natural history of the disease [32]. Since this time, the use of complementary medicines including glucosamine and chondroitin to treat arthritis has continued to rise. The largest multicentre, double-blinded trial published in the *New England Journal of Medicine*, however, concluded that the use of combined glucosamine/chondroitin had no overall effect on knee pain in a large cohort of patients, though there was a small benefit for the subgroup with moderate to severe pain [33]. Although their efficacy is uncertain, particularly in the shoulder, these supplements are approved for use in sport, and are safe with minimal side effects. This suggests that a trial of use in the athlete with painful glenohumeral arthritis is appropriate [31].

#### **11.4.1.3 Corticosteroid Injections**

The use of corticosteroid injections in sport is widespread, and often utilised in conjunction with long-acting local anaesthetics. Local anaesthetic is a helpful diagnostic tool while providing very short-term pain relief. A Cochrane review suggested that intra-articular steroid injections were superior to physiotherapy alone in providing pain relief in the short term. However, many studies are confounded by variability in injection type and site, associated medications and physiotherapy and differing activity levels. Hence assessing the true efficacy of injections in patients with arthritis has limitations [34].

The treating team should provide an environment that ensures the safest and most effective use of injections. The injection should be performed under sterile conditions with fluoroscopic

guidance to ensure correct intra-articular position. It is critical that injections are not used as short-term solutions to ‘get an athlete through a game.’ This approach can have devastating consequences and accelerate chondral degeneration due to the loss of the protective mechanism of pain. Furthermore, corticosteroids suppress collagen synthesis and may affect the integrity of the remaining cartilage. The objective should be to provide temporary dampening of the synovitis and pain associated with arthritis and to allow for a structured strengthening and rehabilitation programme. The athlete should not be expected to play or train at full intensity during this time. Therefore, injections should be used judiciously and ideally not more than twice per joint per year [1].

#### **11.4.1.4 Hyaluronic Acid Injections**

The intra-articular injection of various hyaluronic acid formulations has been utilised most commonly in the knee. There are some positive clinical outcomes reported as a pain reliever and a low side effect profile, particularly when compared to NSAIDs [35, 36]. There is less evidence regarding treatment of glenohumeral arthritis, likely in part due to regulatory approval in many countries (though not the European Union) being limited to the knee. There are two well designed randomised studies that show significantly improved pain for 6 months or more in patients receiving 3–5 weekly hyaluronic acid injections, with particular benefit in those without other concomitant shoulder pathology [37, 38].

#### **11.4.1.5 Physiotherapy**

The key aim of physiotherapy in these patients is to maximise range of motion and strength within the limitations rendered by the arthritic joint. This may focus on maintaining range of motion rather than attempting to increase it. Arthritic shoulders will often cause scapular dyskinesia, and a tailored programme focusing on scapular stabilisation and regaining excellent scapulohumeral rhythm throughout range of motion is paramount to maximising function. This should be associated with isometric strengthening and passive stretching to maintain range and function.

Hydrotherapy should play a key role in the rehabilitative process, as water provides buoyancy and resistance that aids strengthening, while minimising the stress on the joint [1]. Training regimes should be modified, in particular to avoid high load and repetitive impact activities.

#### 11.4.1.6 Arthroscopy

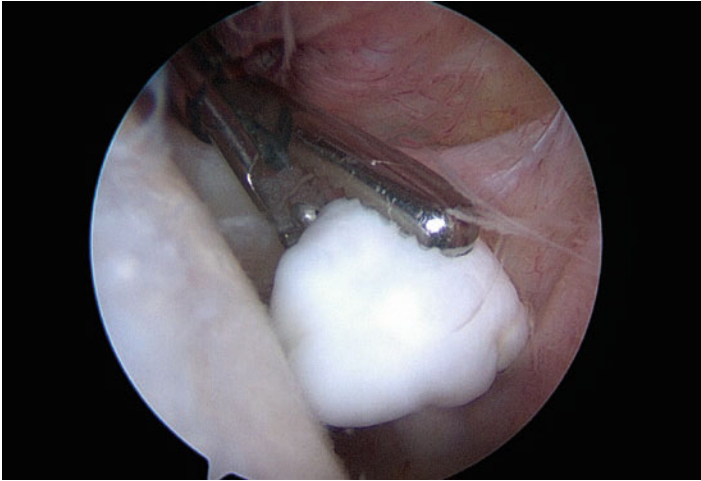
Arthroscopy is the gold standard for diagnosis of chondral lesions [9] (Fig. 11.5).

However, the therapeutic role of arthroscopy in patients with arthritis of any joint is controversial. The literature suggests that up to 80% of patients report good to excellent results over the short term following arthroscopy, however, many of these larger studies involve lower demand pre-arthroplasty patients who have a mean age of 50–60 years and are, therefore, a different cohort to the athletic population [39, 40]. The most convincing results of arthroscopic debridement and chondroplasty in young patients are found in those with isolated, symptomatic chondral lesions. The aim is to remove any unstable chondral flaps that are likely pain generators and may cause propagation of the defect. The benefit is greater in those patients with a congruent joint, minimal osteophyte or cyst formation and shallower lesions on only one side of the joint measuring  $<2 \text{ cm}^2$  [39].



**Fig. 11.5** Full thickness humeral chondral defect seen at arthroscopy





**Fig. 11.6** Loose body retrieved during arthroscopy

The adjunctive procedures utilised at time of arthroscopy in patients with earlier stage disease can include debridement, lavage and removal of loose bodies [2, 41, 42] (Fig. 11.6).

Other pain generators are appropriately addressed at the time of surgery. As the disease progresses, the anterior capsule in particular contracts leading to decreased external rotation and increased load on chondral surfaces due to abnormal motion [1]. Patients with more advanced disease may benefit from a more comprehensive procedure that includes a full capsular release, subacromial decompression, glenohumeral chondroplasty, AC joint excision, humeral osteoplasty and osteophyte resection plus a biceps tenodesis and axillary nerve neurolysis. Promising results have been described, with 85% of patients delaying arthroplasty by 2 years [40].

#### **11.4.2 Career Salvaging**

These options are indicated in the athlete with more severe or symptomatic degenerative disease. The athlete is likely to have

already undertaken multiple treatments in the career-maintaining group with diminishing success. Although high-impact activity is possible after these modalities, their overall success rates are circumspect, particularly in a high-load environment. Therefore, the athlete's expectations must be managed: they must be aware that although these procedures are an attempt to prolong a career, they may not recover sufficient function or symptom control to return to their desired level of activity or performance.

#### **11.4.2.1 Microfracture**

Due to the poor vascularity and cellular structure of articular cartilage, the ability for chondral defects to heal is poor [43]. The principle behind microfracture is to first create a favourable healing environment, followed by penetration of the subchondral plate and marrow underlying the defect to stimulate the body's injury response. This leads to a stimulation and proliferation of mesenchymal stem cells as part of an organising fibrin clot, and in conjunction with growth factors and platelets the formation of granulation tissue and eventually fibrocartilage [44]. The procedure is generally performed arthroscopically, however, crucial to its success is meticulous surgical technique and a slow, tailored rehabilitation programme [45]. Although microfracture treatment of full-thickness chondral defects have been utilised successfully in the knee and to a lesser extent the ankle, there is minimal literature regarding its use and efficacy in the shoulder [26]. The small series that have been published in the shoulder have found the best outcomes in isolated humeral-sided lesions, with promising improvement in pain, ability to work and play sport in young, active patients. Microfracture does not appear to compromise future reconstructive options, however, this is tempered by a failure rate of up to 20% [43, 46]. These results suggest that microfracture is a reasonable escalation of treatment in the symptomatic athlete, however, appropriate counselling must emphasise that a significant percentage of patients will not respond to this treatment resulting in likely premature retirement.

#### **11.4.2.2 Chondral/Osteochondral Grafting**

Replacing an area of damaged or missing cartilage with normal or near normal chondral tissue is the ideal endpoint for managing a

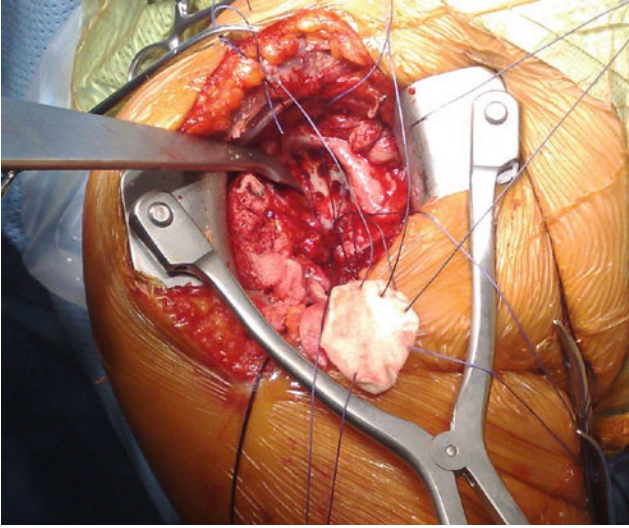
focal chondral lesion. Though difficult to achieve successfully, modalities include osteochondral transplant using allograft, primary grafting using autograft transfer (OATS), staged autologous chondrocyte implantation (ACI) or use of a synthetic scaffold.

Osteochondral grafting (OATS) into a humeral head defect has theoretical advantages including the healing potential of bone and use of autograft. However, there is also significant potential morbidity with open surgery required at both the shoulder and the donor site—normally the knee. The largest series, albeit in only 8 patients, shows good results with congruent joint lines and no further surgery at nine years following the index procedure [47]. For larger, uncontained defects, size and contour matched allograft from a glenoid or humeral head is a preferable option, though is limited by availability of fresh or fresh frozen grafts in some centres [5, 9]. Although ACI in the knee has shown promising results [48, 49] it remains unproven in the shoulder, with only two studies covering five patients in the literature [50]. This includes a case report in a teenage athlete who maintained a full range of motion and preserved function at one year after grafting [5, 51, 52].

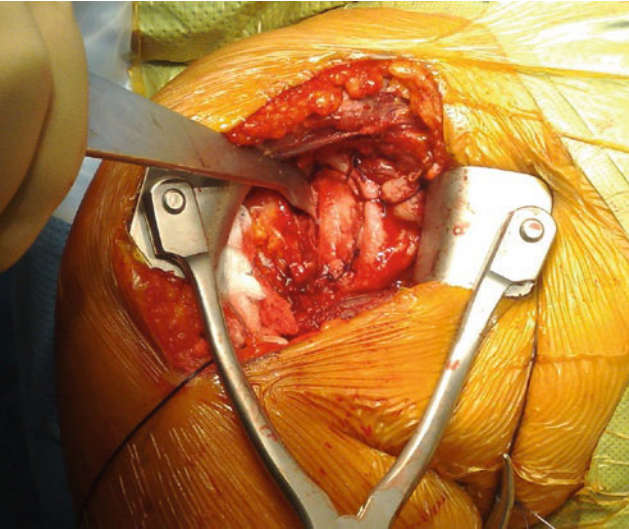
### 11.4.2.3 Biological Resurfacing

Resurfacing of an arthritic glenoid can be undertaken using a number of different tissues. Infolding of the patient's own anterior capsule, allograft such as fascia lata, achilles tendon, lateral meniscus or commercially available dermal tissue or biologic patches are some of the options. These are designed to provide at least an interposition of soft tissue on the arthritic surface and even perhaps a biological membrane that is incorporated into the host joint, and is, therefore, designed to be an alternative to prosthetic replacement [41] (Figs. 11.7 and 11.8). This is considered a viable alternative in a younger population as it is bone preserving and retains prosthetic options for later revision.

The procedure is generally performed open, requiring a subscapularis detachment and a prolonged rehabilitation, though some centres have developed advanced arthroscopic techniques for biological resurfacing. There are also sporadic reports in the literature regarding their use in athletes, and suggested good outcomes in the first couple of years' post-surgery [53–55].



**Fig. 11.7** Glenoid resurfacing-biological patch being prepared for fixation onto glenoid



**Fig. 11.8** Glenoid resurfacing-patch successfully implanted

Unfortunately, longer term outcome studies have not been able to replicate those promising early results, and the technical difficulty combined with a significant complication rate means that these procedures are now less commonly used [56].

#### **11.4.2.4 Arthrodesis**

The lack of shoulder girdle movement produced by a fused glenohumeral joint poses a restriction of function that most patients are not willing to consider, particular in the context of modern prosthetic implants producing excellent results. Therefore, arthrodesis has almost been consigned to historical significance for patients with glenohumeral arthritis, unless it is in the context of failed arthroplasty, infection or neurological injury [57]. In spite of the limitations created by the fused joint, a patient with a united arthrodesis can return to whatever level of sport they desire including full impact, as there are no overt contraindications to undertaking any activity. Hence in a small subgroup of patients, particular those with disease on their non-dominant side, whose priority is to maintain high-impact or loading, athletic or work-related activity rather than focus on basic function, a conversation regarding the possibility of arthrodesis as an alternative to arthroplasty should at least be undertaken.

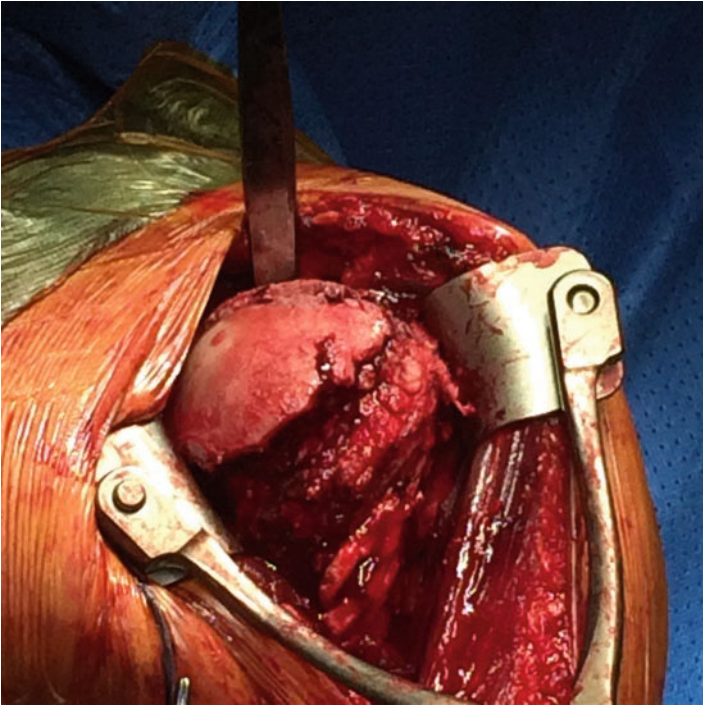
### **11.4.3 Career Ending**

#### **11.4.3.1 Activity Modification**

The cessation of activities that exacerbate pain is in the first line of management options in most patients. However, in the athlete, this is a last resort and generally a career-ending decision. This decision is usually made for one of three reasons: intractable pain, stiffness limiting function and performance, and finally a realisation that longer term quality of life will be significantly impacted by the continuation of a sporting career.

#### **11.4.3.2 Arthroplasty**

Glenohumeral arthroplasty produces the most consistently positive and reproducible outcomes in older patients with arthritis,



**Fig. 11.9** Arthritic humeral head seen intra-operatively prior to resection for arthroplasty

and with modern generation implants in particular is an excellent treatment option in this subgroup of patients [58, 59]. There is a spectrum of arthroplasty options available, with consideration given to whether the disease is on one or both sides of the joint or whether the patient is high or low demand.

Arthroplasty requires an extensive open exposure, and after capsular releases and osteophyte removal the humeral head and glenoid surfaces can be prepared (Fig. 11.9).

Partial arthroplasty options include cementless prosthetic humeral head resurfacing, stemmed hemiarthroplasty, and so-called 'ream and run' procedures, where the humeral head is replaced and the glenoid is not resurfaced but reamed to create a



**Fig. 11.10** Stemless total shoulder replacement for post traumatic arthritis (failed Latarjet procedure with secondary joint degeneration)

uniform concavity [57, 59]. The alternative is total shoulder arthroplasty utilising both humeral and glenoid components. Cementless resurfacing arthroplasty uses a metal or ceramic cap, the inside of which is usually coated with hydroxyapatite to

encourage bone ongrowth, and can be used in isolation or as part of a total shoulder arthroplasty [60] (Fig. 11.10).

These have become popular options for younger patients due to the preservation of bone stock, ease of revision and in the case of isolated humeral head resurfacing a requirement for fewer activity restrictions. Short- and medium-term results are generally positive [60–63]. However, this must be weighed against the generally superior overall results and longer history of survival of stemmed total arthroplasty, mostly due to progressive glenoid erosion and less predictable pain relief and function seen with partial implants [57, 58, 64, 65].

The increased surgical risks of any type of prosthetic arthroplasty and the high likelihood of at least one revision during the life of a younger patient leads to their indication and use only in those young patients with severe disease and intractable pain or profound loss of function [59]. The Mayo clinic has published the largest series of arthroplasties in young patients under 50 years and found more than half had unsatisfactory results [65]. The survival rate of the implant in young patients requiring arthroplasty after previous instability surgery (the most common cause of arthritis in this population) was only 61% at 10 years. This was due to implant failure and instability in a total arthroplasty and glenoid erosion in hemiarthroplasty [66]. Although use of newer generation prostheses with improved implant design should contribute to improved longevity, judicious use of arthroplasty in young patients is still essential [59, 67].

Most young, active patients will want to delay arthroplasty for as long as possible in order to maintain their higher level of activity [40, 68]. Sporting activities involving the upper limbs, including swimming, golf and tennis (if arthroplasty is performed on the non-dominant arm), that are undertaken at a non-strenuous level are still possible after arthroplasty [69]. However, for the professional or high-level recreational athlete, arthroplasty is only an option following the end of a career, as a prosthesis cannot tolerate the high forces and loads required by the vast majority of athletic pursuits involving the upper limbs. Although shoulder arthroplasty can be considered the gold standard of treatment for glenohumeral arthritis in the general population, the athlete must



be able to adjust their activities to cater for the precautions mandated by shoulder arthroplasty. As this will be a career-ending decision, alternative treatment options need to be utilised for as long as possible.

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## 11.5 Conclusion

Glenohumeral arthritis in the athlete is a disease that provides significant diagnostic and management challenges. It is most often encountered in athletes involved in high-contact sports with a history of previous instability. In mild cases that are diagnosed early and managed well, the athlete's career may be maintained with an adequate level of function. Much of this treatment will be non-operative, with use of NSAIDs, judicious use of injections and targeted physiotherapy. Surgery including arthroscopy, microfracture and occasionally more invasive grafting procedures can be successful in providing short to medium-term pain relief and adequate range of motion. Unfortunately however, this degenerative process often deteriorates rapidly and can have devastating physical and psychological consequences, leading to the truncation of the athlete's career due to pain and loss of function, or a requirement for career-ending surgery in itself.

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## Q&A

- (1) What are the important and sometimes subtle signs of early arthritis on examination?  
Understated symptoms include subtle episodes of instability or a slightly reduced ability to undertake previously performed tasks or activities. Night pain is common, with a deep ache associated with an inability to sleep on the affected side. Clicking, grinding and transient locking with sudden pain that limits activity are also frequently reported.
- (2) What is the gold standard tool for diagnosis of chondral damage?

Due to the limitations of imaging modalities in accurately diagnosing chondral damage, diagnostic arthroscopy remains the gold standard for diagnosis, particularly in joints such as the shoulder with thinner layers of articular cartilage.

- (3) How should intra-articular steroid injections be utilised relative to game day?

Regular injections should not be used as short-term solutions to 'get an athlete through a game.' This approach can have devastating consequences and accelerate chondral degeneration due to the loss of the protective mechanism of pain.

- (4) What are the possible adjunctive procedures in arthroscopy performed for glenohumeral arthritis?

The adjunctive procedures at time of arthroscopy in patients with earlier stage disease can include debridement, lavage and removal of loose bodies. Management of all possible pain generators should be considered as part of arthroscopic management including chondroplasty, subacromial decompression, AC joint excision, biceps tenodesis, humeral osteoplasty, capsular release and axillary nerve decompression.

- (5) Is glenohumeral arthroplasty indicated in the competing athlete?

For the professional or high-level recreational athlete, prosthetic arthroplasty is only an option following the end of a career, as a prosthesis cannot tolerate the high forces and loads required by the vast majority of athletic pursuits involving the upper limbs.

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## References

1. Reineck JR, Krishnan SG, Burkhead W. Early glenohumeral arthritis in the competing athlete. *Clin Sports Med.* 2008;27(4):803–19.
2. McCarty LP, Cole BJ. Nonarthroplasty treatment of glenohumeral cartilage lesions. *Arthroscopy.* 2005;21(9):1131–42.
3. Outerbridge R. The etiology of chondromalacia patellae. *J Bone Joint Surg.* 1961;43(4):752–7.
4. Gartsman GM, Brinker MR, Khan M, Karahan M. Self-assessment of general health status in patients with five common shoulder conditions. *J Shoulder Elb Surg.* 1998;7(3):228–37.

5. Bhatia S, Hsu A, Lin EC, Chalmers P, Ellman M, Cole BJ, et al. Surgical treatment options for the young and active middle-aged patient with glenohumeral arthritis. *Adv Orthop*. 2012;2012:846843.
6. Provencher MT, Barker JU, Strauss EJ, Frank RM, Romeo AA, III FAM. Glenohumeral arthritis in the young adult. *Instr Course Lect*. 2011;60:137–53.
7. Sayegh ET, Mascarenhas R, Chalmers PN, Cole BJ, Romeo AA, Verma NN. Surgical treatment options for glenohumeral arthritis in young patients: a systematic review and meta-analysis. *Arthroscopy*. 2015;31(6):1156–66.
8. Boselli KJ, Ahmad CS, Levine WN. Treatment of glenohumeral arthrosis. *Am J Sports Med*. 2010;38(12):2558–72.
9. Cole BJ, Yanke A, Provencher MT. Nonarthroplasty alternatives for the treatment of glenohumeral arthritis. *J Shoulder Elb Surg*. 2007;16(5):S231–S40.
10. Wallace A. Management of glenohumeral osteoarthritis in the young adult. *Should Elb*. 2010;2(1):1–8.
11. Walch G, Boulahia A, Badet R, Riand N, Kempf J. Primary glenohumeral osteoarthritis: clinical and radiographic classification. *Shoulder Arthroplast*. 1999;64(Suppl 2):46–52.
12. Buscayret F, Edwards TB, Szabo I, Adeleine P, Coudane H, Walch G. Glenohumeral arthrosis in anterior instability before and after surgical treatment incidence and contributing factors. *Am J Sports Med*. 2004;32(5):1165–72.
13. Hovelius L, Saeboe M. Neer award 2008: arthropathy after primary anterior shoulder dislocation—223 shoulders prospectively followed up for twenty-five years. *J Shoulder Elb Surg*. 2009;18(3):339–47.
14. Marx RG, McCarty EC, Montemurno TD, Altchek DW, Craig EV, Warren RF. Development of arthrosis following dislocation of the shoulder: a case-control study. *J Shoulder Elb Surg*. 2002;11(1):1–5.
15. Maquirriain J, Ghisi J, Amato S. Is tennis a predisposing factor for degenerative shoulder disease? A controlled study in former elite players. *Br J Sports Med*. 2006;40(5):447–50.
16. Paley KJ, Jobe FW, Pink MM, Kvitne RS, ElAttrache NS. Arthroscopic findings in the overhand throwing athlete: evidence for posterior internal impingement of the rotator cuff. *Arthroscopy*. 2000;16(1):35–40.
17. Levy JC, Virani NA, Frankle MA, Cuff D, Pupello DR, Hamelin JA. Young patients with shoulder chondrolysis following arthroscopic shoulder surgery treated with total shoulder arthroplasty. *J Shoulder Elb Surg*. 2008;17(3):380–8.
18. Hansen BP, Beck CL, Beck EP, Townsley RW. Postarthroscopic glenohumeral chondrolysis. *Am J Sports Med*. 2007;35(10):1628–34.
19. McNickle AG, L'Heureux DR, Provencher MT, Romeo AA, Cole BJ. Postsurgical glenohumeral arthritis in young adults. *Am J Sports Med*. 2009;37(9):1784–91.

20. Scheffel PT, Clinton J, Lynch JR, Warne WJ, Bertelsen AL, Matsen FA. Glenohumeral chondrolysis: a systematic review of 100 cases from the English language literature. *J Shoulder Elb Surg.* 2010;19(6):944–9.
21. Chu CR, Coyle CH, Chu CT, Szczodry M, Seshadri V, Karpie JC, et al. In vivo effects of single intra-articular injection of 0.5% bupivacaine on articular cartilage. *J Bone Joint Surg.* 2010;92(3):599–608.
22. Webb S, Ghosh S. Intra-articular bupivacaine: potentially chondrotoxic? *Br J Anaesth.* 2009;102(4):439–41.
23. Ellman H, Harris E, Kay SP. Early degenerative joint disease simulating impingement syndrome: arthroscopic findings. *Arthroscopy.* 1992;8(4):482–7.
24. Brox J, Lereim P, Merckoll E, Finnanger AM. Radiographic classification of glenohumeral arthrosis. *Acta Orthop.* 2003;74(2):186–9.
25. Samilson R, Prieto V. Dislocation arthropathy of the shoulder. *J Bone Joint Surg.* 1983;65(4):456–60.
26. 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.
27. Yeh L, Kwak S, Kim Y-S, Chou DS, Muhle C, Skaf A, et al. Evaluation of articular cartilage thickness of the humeral head and the glenoid fossa by MR arthrography: anatomic correlation in cadavers. *Skelet Radiol.* 1998;27(9):500–4.
28. Guntern DV, Pfirrmann CW, Schmid MR, Zanetti M, Binkert CA, Schneeberger AG, et al. Articular cartilage lesions of the glenohumeral joint: diagnostic effectiveness of MR arthrography and prevalence in patients with subacromial impingement syndrome I. *Radiology.* 2003;226(1):165–70.
29. Smith TO, Drew BT, Toms AP, Donell ST, Hing CB. Accuracy of magnetic resonance imaging, magnetic resonance arthrography and computed tomography for the detection of chondral lesions of the knee. *Knee Surg Sports Traumatol Arthrosc.* 2012;20(12):2367–79.
30. Moskowitz RW, Blaine TA. An overview of treatment options for persistent shoulder pain. *Am J Orthop.* 2005;34(12 Suppl):10–5.
31. Vangsness CT, Spiker W, Erickson J. A review of evidence-based medicine for glucosamine and chondroitin sulfate use in knee osteoarthritis. *Arthroscopy.* 2009;25(1):86–94.
32. Reginster JY, Deroisy R, Rovati LC, Lee RL, Lejeune E, Bruyere O, et al. Long-term effects of glucosamine sulphate on osteoarthritis progression: a randomised, placebo-controlled clinical trial. *Lancet.* 2001;357(9252):251–6.
33. Clegg DO, Reda DJ, Harris CL, Klein MA, O'Dell JR, Hooper MM, et al. Glucosamine, chondroitin sulfate, and the two in combination for painful knee osteoarthritis. *N Engl J Med.* 2006;354(8):795–808.
34. Buchbinder R, Green S, Youd J. Corticosteroid injections for shoulder pain. *Cochrane Database Syst Rev.* 2003;1:CD004016.

35. Bannuru RR, Vaysbrot EE, Sullivan MC, McAlindon TE. Relative efficacy of hyaluronic acid in comparison with NSAIDs for knee osteoarthritis: a systematic review and meta-analysis. *Semin Arthritis Rheum*. 2014;43(5):593–9.
36. Brzusek D, Petron D. Treating knee osteoarthritis with intra-articular hyaluronans. *Curr Med Res Opin*. 2008;24(12):3307–22.
37. Blaine T, Moskowitz R, Udell J, Skyhar M, Levin R, Friedlander J, et al. Treatment of persistent shoulder pain with sodium hyaluronate: a randomized, controlled trial. *J Bone Joint Surg*. 2008;90(5):970–9.
38. Kwon YW, Eisenberg G, Zuckerman JD. Sodium hyaluronate for the treatment of chronic shoulder pain associated with glenohumeral osteoarthritis: a multicenter, randomized, double-blind, placebo-controlled trial. *J Shoulder Elb Surg*. 2013;22(5):584–94.
39. Cameron BD, Galatz LM, Ramsey ML, Williams GR, Iannotti JP. Non-prosthetic management of grade IV osteochondral lesions of the glenohumeral joint. *J Shoulder Elb Surg*. 2002;11(1):25–32.
40. Millett PJ, Horan MP, Pennock AT, Rios D. Comprehensive arthroscopic management (CAM) procedure: clinical results of a joint-preserving arthroscopic treatment for young, active patients with advanced shoulder osteoarthritis. *Arthroscopy*. 2013;29(3):440–8.
41. Cole BJ, Mellano C. Is there a role for arthroscopy in the treatment of glenohumeral arthritis. *Am J Orthop*. 2014;43(2):1–3.
42. Elser F, Braun S, Dewing CB, Millett PJ. Glenohumeral joint preservation: current options for managing articular cartilage lesions in young, active patients. *Arthroscopy*. 2010;26(5):685–96.
43. 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.
44. Frisbie DD, Oxford JT, Southwood L, Trotter GW, Rodkey WG, Steadman JR, et al. Early events in cartilage repair after subchondral bone microfracture. *Clin Orthop Relat Res*. 2003;407:215–27.
45. Steadman JR, Rodkey WG, Rodrigo JJ. Microfracture: surgical technique and rehabilitation to treat chondral defects. *Clin Orthop Relat Res*. 2001;391:S362–S9.
46. 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.
47. 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*. 2009;91B(4):499–503.
48. Ebert JR, Robertson WB, Woodhouse J, Fallon M, Zheng MH, Ackland T, et al. Clinical and magnetic resonance imaging–based outcomes to 5 years after matrix-induced autologous chondrocyte implantation to address articular cartilage defects in the knee. *Am J Sports Med*. 2011;39(4):753–63.

49. Kon E, Gobbi A, Filardo G, Delcogliano M, Zaffagnini S, Marcacci M. Arthroscopic second-generation autologous chondrocyte implantation compared with microfracture for chondral lesions of the knee: prospective nonrandomized study at 5 years. *Am J Sports Med.* 2009;37(1):33–41.
50. Gross CE, Chalmers PN, Chahal J, Van Thiel G, Bach BR Jr, Cole BJ, et al. Operative treatment of chondral defects in the glenohumeral joint. *Arthroscopy.* 2012;28(12):1889–901.
51. Buchmann S, Salzmann GM, Glanzmann MC, Wörtler K, Vogt S, Imhoff AB. Early clinical and structural results after autologous chondrocyte transplantation at the glenohumeral joint. *J Shoulder Elb Surg.* 2012;21(9):1213–21.
52. Romeo AA, Cole BJ, Mazzocca AD, Fox JA, Freeman KB, Joy E. Autologous chondrocyte repair of an articular defect in the humeral head. *Arthroscopy.* 2002;18(8):925–9.
53. De Beer J, Bhatia D, van Rooyen K, Du Toit D. Arthroscopic debridement and biological resurfacing of the glenoid in glenohumeral arthritis. *Knee Surg Sports Traumatol Arthrosc.* 2010;18(12):1767–73.
54. Savoie FH, Brislin KJ, Argo D. Arthroscopic glenoid resurfacing as a surgical treatment for glenohumeral arthritis in the young patient: midterm results. *Arthroscopy.* 2009;25(8):864–71.
55. Williams G, Font-Rodriguez D, Baghlan S. Soft-tissue interposition without hemiarthroplasty as an alternative for degenerative shoulder arthritis in young, active patients. In: *Proceedings of the American Shoulder and Elbow Surgeons*; 2002.
56. Strauss EJ, Verma NN, Salata MJ, McGill KC, Klifto C, Nicholson GP, et al. The high failure rate of biologic resurfacing of the glenoid in young patients with glenohumeral arthritis. *J Shoulder Elb Surg.* 2014;23(3):409–19.
57. Denard PJ, Wirth MA, Orfaly RM. Management of glenohumeral arthritis in the young adult. *J Bone Joint Surg.* 2011;93(9):885–92.
58. Boileau P, Sinnerton RJ, Chuinard C, Walch G. Arthroplasty of the shoulder. *J Bone Joint Surg.* 2006;88B(5):562–75.
59. Johnson MH, Paxton ES, Green A. Shoulder arthroplasty options in young (<50 years old) patients: review of current concepts. *J Shoulder Elb Surg.* 2015;24(2):317–25.
60. Burgess DL, McGrath MS, Bonutti PM, Marker DR, Delanois RE, Mont MA. Shoulder resurfacing. *J Bone Joint Surg.* 2009;91(5):1228–38.
61. Bailie DS, Llinas PJ, Ellenbecker TS. Cementless humeral resurfacing arthroplasty in active patients less than fifty-five years of age. *J Bone Joint Surg Am.* 2008;90(1):110–7.
62. Iagulli ND, Field LD, Hobgood ER, Hurt JA, Charles R, O'Brien MJ, et al. Surface replacement arthroplasty of the humeral head in young, active patients: midterm results. *Orthop J Sports Med.* 2014;2(1):2325967113519407.
63. Levy O, Tsvieli O, Merchant J, Young L, Trimarchi A, Dattani R, et al. Surface replacement arthroplasty for glenohumeral arthropathy in

- patients aged younger than fifty years: results after a minimum ten-year follow-up. *J Shoulder Elb Surg.* 2015;24(7):1049–60.
64. Radnay CS, Setter KJ, Chambers L, Levine WN, Bigliani LU, Ahmad CS. Total shoulder replacement compared with humeral head replacement for the treatment of primary glenohumeral osteoarthritis: a systematic review. *J Shoulder Elb Surg.* 2007;16(4):396–402.
  65. Sperling JW, Cofield RH, Rowland CM. Minimum fifteen-year follow-up of Neer hemiarthroplasty and total shoulder arthroplasty in patients aged fifty years or younger. *J Shoulder Elb Surg.* 2004;13(6):604–13.
  66. Sperling JW, Antuna SA, Sanchez-Sotelo J, Schleck C, Cofield RH. Shoulder arthroplasty for arthritis after instability surgery. *J Bone Joint Surg.* 2002;84(10):1775–81.
  67. Raiss P, Aldinger P, Kasten P, Rickert M, Loew M. Total shoulder replacement in young and middle-aged patients with glenohumeral osteoarthritis. *J Bone Joint Surg.* 2008;90(6):764–9.
  68. Adams JE, Sperling JW, Hoskin TL, Melton LJ, Cofield RH. Shoulder arthroplasty in Olmsted County, Minnesota, 1976-2000: a population-based study. *J Shoulder Elb Surg.* 2006;15(1):50–5.
  69. Bühlhoff M, Sattler P, Bruckner T, Loew M, Zeifang F, Raiss P. Do patients return to sports and work after total shoulder replacement surgery? *Am J Sports Med.* 2015;43(2):423–7.



# Pectoralis Major Ruptures

# 12

Usman Butt and Puneet Monga

## Key Learning Points

- Pectoralis major ruptures occur predominantly in young adult males engaging in strenuous activity, classically during the bench press manoeuvre in weight training.
- Key examination findings are bruising over the chest wall and upper arm; a medially retracted muscle belly; a “dropped nipple” sign and loss of the anterior axillary fold.
- Magnetic resonance imaging is the imaging modality of choice for characterisation of the tear and to aid surgical planning.
- Early direct surgical repair within the first few weeks gives the best long-term outcomes and is the treatment of choice in the majority of cases.
- Surgery may be still considered in selected chronic cases.
- Early specialist referral is recommended for informed decision making and treatment.
- Post-operative care involves an initial period of rest in a sling followed by gradual return to increasing activity.

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## 12.1 Introduction

First described by Patissier in 1822, rupture of the pectoralis major (PM) tendon has traditionally been regarded as a rare injury with rather infrequent reports until the last decade, with over 260 cases described since 2000 [1]. It has been suggested that the rising incidence may reflect an increased public interest in health, fitness and strenuous sporting activities [2–4]. With an appropriate understanding of the presenting features, an early clinical diagnosis and referral for specialist treatment can positively influence the long-term outcome. In contrast, delayed diagnosis may alter subsequent management options leading to less favourable results [1, 4].

## 12.2 Anatomy and Function

The Pectoralis Major is comprised of two portions or “heads”. The clavicular head arises from the clavicle and the sternal head from the rib cage, sternum and the external oblique aponeurosis. The sternal head is the much larger of the two and is itself divided anatomically into several segments. The sternal and clavicular heads join to form a broad flat tendon consisting of two layers that inserts into the proximal humeral shaft (Fig. 12.1) [5–7].

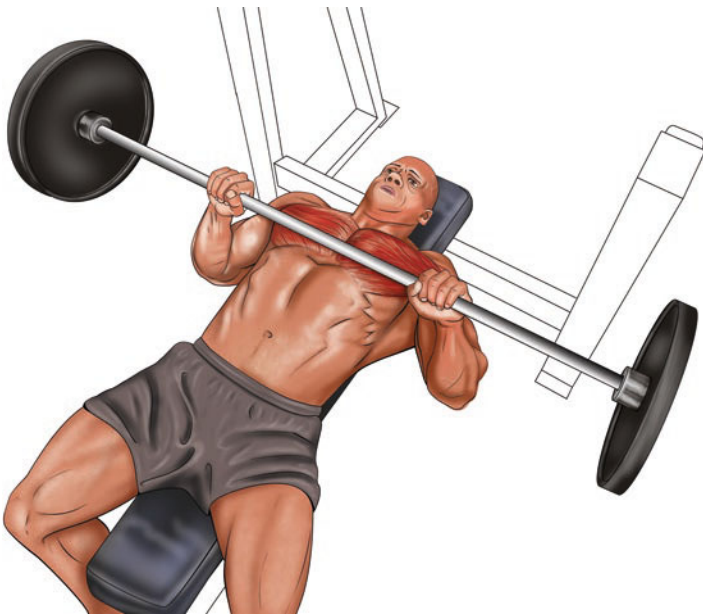


**Fig. 12.1** Pectoralis major anatomy: cadaveric dissection

Pectoralis major is a powerful adductor and internal rotator of the arm with some additional contribution to forward flexion [4, 7]. For the majority of mundane daily activities, the muscle is not considered essential; however, for more strenuous activity and particularly sporting endeavours, it becomes a necessity for maximal power production [8]. The muscle itself is under most stress when eccentrically loaded in extension with the inferior segments tending to fail first in a predictable sequence [9].

### 12.3 Aetiology

PM tendon ruptures typically occur in muscular, young, adult males aged between 20 and 40 years. The deep part of the bench press manoeuvre, used for weight training, is the most frequently associated mechanism of injury [2, 9] (Fig. 12.2). A number of



**Fig. 12.2** Schematic diagram depicting the bench press manoeuvre most commonly associated with ruptures of the pectoralis major tendon. Note the extended position of the arm with eccentric loading of the muscle

other demanding activities have also been reported to result in the injury, including rugby, wrestling, jujitsu, boxing and gymnastics [7, 9–12]. PM ruptures occur predominantly in males, the preponderance thought to be due to a lower tendon to muscle diameter and lower tendon elasticity, along with an engagement in higher energy activities than in females [1]. In a review of all reported cases before 2010, only 11 out of 365 cases occurred in females with an age range of 73–97 years; 10 of whom were nursing home residents [2]. The elderly, in general, form a less common subset of PM ruptures that usually occur during activities such as manual transfers [13].

An additional aetiological factor that is commonly associated with tendon ruptures, including those of the PM, is the use of anabolic steroids. Animal studies have suggested that anabolic steroids lead to alterations in collagen dysplasia and lower rupture stress values [14, 15]. It has also been suggested that with anabolic steroid use, there is a disproportionate increase in muscle strength relative to tendon strength [16].

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## 12.4 Presentation and Diagnosis

Patients commonly present early to healthcare professionals and can usually recall the exact mechanism and time of injury, frequently describing a “snap” or “pop” at the time of injury. The diagnosis is may not be appreciated however, resulting in late presentation for specialist treatment, and a case must be made for raising awareness of such injuries.

In the acute setting, physical examination may reveal a degree of swelling and bruising over the upper arm and chest wall. In comparison with the contralateral side, a “dropped nipple” [17], medially retracted pectoralis muscle belly, and loss of the anterior axillary fold are pathognomonic features and will usually be evident in both acute and chronic settings (Figs. 12.3 and 12.4). These features can be accentuated by passively abducting the arm or with attempted resisted adduction [7, 17].

The diagnosis is usually clear from the presenting history and examination findings. Where there is any doubt, ultrasound



**Fig. 12.3** Early presentation with bruising, swelling, “bunching of the Pectoral”, and a dropped nipple



**Fig. 12.4** Late presentation with loss of anterior Axillary fold, dimpling of the skin, and retraction of the pectoralis major tendon

assessment can be a useful adjunct but magnetic resonance imaging (MRI) is the preferred imaging modality. This will not only confirm the diagnosis but will provide information to aid in surgical planning, such as the degree of tendon retraction and presence of any intact portion of the tendon in the case of a partial tear [7]. Where an MRI is requested this should be done in consultation with a musculoskeletal radiologist so that the correct sequence, to image the entire Pectoralis Major, is performed. A standard shoulder MRI sequence will not suffice [18].

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## 12.5 Management and Outcomes

Complex classification systems have been described for PM ruptures [2, 19], but the key factors that determine the most appropriate course of management and subsequent outcome are the age and activity level of the patient, the chronicity of the injury and the location of the tear along the muscle tendon unit [1].

In frail, elderly patients with a sedentary lifestyle, non-operative management is favoured. In certain partial tears and tears of the muscle belly rather than tendon, non-operative management may also be indicated [1, 4, 7]. Initial management involves rest in a sling, cryotherapy and analgesia with passive exercises instituted as tolerated, followed sequentially by active-assisted and active exercises over a 6-week period. Resistance therapy is subsequently implemented and unrestricted activity allowed at 8–12 weeks [4, 7]. In the case of partial tears treated non-operatively there may be no cosmetic deficit, though in the case of complete tears there is likely to be a permanently visible deformity [4].

Surgical treatment is the management of choice for active individuals [1, 2, 4, 20], providing the best opportunity for restoring motion, strength and cosmesis to approach pre-injury levels [4, 7, 16, 21]. Ideally, surgery is instituted acutely, as there may be a degree of tendon retraction evident by as early as 3 weeks [1], highlighting the importance of early referral to a specialist with an interest in these injuries. Good results are still achievable in the delayed setting and should still be considered, particularly where

there is a functional deficit [1, 2, 4, 7, 20]. In the majority of cases a direct repair is possible with a variety of techniques described, including the use of anchors, buttons and trans-osseous sutures, though none of these techniques have been clearly shown to be superior to the other [1, 8, 22]. In the chronic setting, where a direct repair is not possible, additional releases and grafting techniques may be required [1, 23–28].

The surgery is usually undertaken through a skin crease incision in the deltopectoral groove, which achieves a cosmetic scar (Fig. 12.5). The authors prefer to use bone anchors to achieve repair. A safe range of motion is determined at the time of surgery, which helps inform the postoperative rehabilitation regime. Following surgery, the patient is initially placed in a sling. Rehabilitation regimes are personalised and based upon patient factors and characteristics of the tear and repair. In most cases, active hand, wrist and elbow exercises are allowed immediately. Early closed-chain mobilisation for the shoulder is started within the safe range determined at surgery. In general, external rotation and abduction is avoided initially. Over a variable period of 3–6 weeks, the sling is weaned off and progression aimed at instituting active motion. Return to sports and unrestricted activity is usually achieved between 3 to 6 months from the time of surgery.



**Fig. 12.5** Restoration of anterior axillary fold following a left Pectoralis Major tendon repair

## 12.6 Conclusion

Ruptures of the pectoralis major tendon are uncommon but important injuries that frequently present in a sporting setting. They occur mainly in the male population between the age of 20 and 40 years, commonly following bench press, but can result from any strenuous activity involving eccentric loads with the arm in an extended position. A careful history and examination will frequently allow the diagnosis to be made accurately on clinical grounds. The preferred imaging modality is MRI with dedicated sequencing, which not only helps confirm the diagnosis, but aids in surgical planning. Surgery is the treatment of choice for the young active population with the best outcomes achieved with early repair, preferably within the first few weeks. Early referral to a specialist with an interest in these injuries is paramount to allow informed discussion and the best possible long-term outcome.

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### Q&A

- (1) Which groups of patients commonly sustain ruptures of the pectoralis major tendon?

The injury most-commonly occurs in young muscular adult males between the age of 20 and 40. It has also been described in elderly frail patients.

- (2) How do ruptures of the pectoralis major tendon occur?

In the young and high demand patient, it typically occurs when the shoulder is extended and eccentrically loaded as in the depth of the bench press manoeuvre, but it can occur in any sporting activity.

- (3) What are the examination findings?

Key findings are the presence of bruising over the chest wall and upper arm; a medially retracted muscle belly; a “dropped nipple” sign and loss of the anterior axillary fold. These features are best appreciated by comparing to the contralateral

side and can be accentuated by passively abducting the arm or by resisted adduction. Partial tears may not demonstrate all of these features, and likewise swelling in the acute setting may obscure some of the findings. It is therefore important to consider the history carefully and maintain a high index of suspicion.

(4) What is the preferred method of treatment?

Initial management involves rest in a sling with analgesia and cryotherapy. Definitive management in the majority of cases involves surgical repair, which is best undertaken within the first few weeks of the injury. However, surgery can still be considered in selected cases where the diagnosis has been delayed or even in longstanding chronic cases.

(5) Who should manage this condition

These are uncommon injuries and as such tend to be managed by surgeons with a specialist interest in sports injuries. Initial and early referral to the most appropriate clinician will result in timely management.

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## References

1. Butt U, Mehta S, Funk L, Monga P. Pectoralis major ruptures: a review of current management. *J Shoulder Elb Surg.* 2015;24(4):655–62.
2. ElMaraghy AW, Devereaux MW. A systematic review and comprehensive classification of pectoralis major tears. *J Shoulder Elb Surg.* 2012;21:412–22.
3. Hasegawa K, Schofer JM. Rupture of the pectoralis major: a case report and review. *J Emerg Med.* 2010;38:196–200.
4. Petilon J, Ellingson CI, Sekiya JK. Pectoralis major muscle ruptures. *Oper Tech Sports Med.* 2005;13:162–8.
5. Fung L, Wong B, Ravichandiran K, Agur A, Rindlisbacher T, Elmaraghy A. Three-dimensional study of pectoralis major muscle and tendon architecture. *Clin Anat.* 2009;22:500–8.
6. Metzger PD, Bailey JR, Filler RD, Waltz RA, Provencher MT, Dewing CB. Pectoralis major muscle rupture repair: technique using unicortical buttons. *Arthrosc Tech.* 2012;1:e119–25.
7. Provencher CDRMT, Handfield K, Boniquit NT, Reiff SN, Sekiya JK, Romeo AA. Injuries to the pectoralis major muscle diagnosis and management. *Am J Sports Med.* 2010;38:1693–705.



8. Rabuck SJ, Lynch JL, Guo X, et al. Biomechanical comparison of 3 methods to repair pectoralis major ruptures. *Am J Sports Med.* 2012;40:1635–40.
9. Wolfe SW, Wickiewicz TL, Cavanaugh JT. Ruptures of the pectoralis major muscle an anatomic and clinical analysis. *Am J Sports Med.* 1992;20:587–93.
10. Merolla G, Paladini P, Artiaco S, Tos P, Lollino N, Porcellini G. Surgical repair of acute and chronic pectoralis major tendon rupture: clinical and ultrasound outcomes at a mean follow-up of 5 years. *Eur J Orthop Surg Traumatol.* 2015;25(1):91–8.
11. Ohashi K, El-Khoury GY, Albright JP, Tearse DS. MRI of complete rupture of the pectoralis major muscle. *Skelet Radiol.* 1996;25:625–8.
12. Zeman SC, Rosenfeld RT, Lipscomb PR. Tears of the pectoralis major muscle. *Am J Sports Med.* 1979;7:343–7.
13. Beloosesky Y, Grinblat J, Weiss A, Rosenberg PH, Weisbord M, Hendel D. Pectoralis major rupture in elderly patients: a clinical study of 13 patients. *Clin Orthop Relat Res.* 2003;413:164–9.
14. Inhofe PD, Grana WA, Egle D, Min K-W, Tomasek J. The effects of anabolic steroids on rat tendon an ultrastructural, biomechanical, and biochemical analysis. *Am J Sports Med.* 1995;23:227–32.
15. Tsitsilonis S, Panayiotis CE, Athanasios MS, et al. Anabolic androgenic steroids reverse the beneficial effect of exercise on tendon biomechanics: an experimental study. *Foot Ankle Surg.* 2014;20(2):94–9.
16. Bak K, Cameron EA, Henderson IJP. Rupture of the pectoralis major: a meta-analysis of 112 cases. *Knee Surg Sports Traumatol Arthrosc.* 2000;8:113–9.
17. Shah NH, Talwalker S, Badge R, Funk L. Pectoralis major rupture in athletes: footprint technique and results. *Tech Should Elbow Surg.* 2010;11:4–7.
18. Lee J, Brookenthal KR, Ramsey ML, Kneeland JB, Herzog R. MR imaging assessment of the pectoralis major myotendinous unit: an MR imaging—anatomic correlative study with surgical correlation. *Am J Roentgenol.* 2000;174:1371–5.
19. Tietjen R. Closed injuries of the pectoralis major muscle. *J Trauma Acute Care Surg.* 1980;20:262–4.
20. Schepsis AA, Grafe MW, Jones HP, Lemos MJ. Rupture of the pectoralis major muscle outcome after repair of acute and chronic injuries. *Am J Sports Med.* 2000;28:9–15.
21. Hanna CM, Glenny AB, Stanley SN, Caughey MA. Pectoralis major tears: comparison of surgical and conservative treatment. *Br J Sports Med.* 2001;35:202–6.
22. Sherman SL, Lin EC, Verma NN, et al. Biomechanical analysis of the pectoralis major tendon and comparison of techniques for tendo-osseous repair. *Am J Sports Med.* 2012;40(8):1887–94.

23. de Castro Pochini A, Ejnisman B, Andreoli CV, et al. Pectoralis major muscle rupture in athletes a prospective study. *Am J Sports Med.* 2010;38:92–8.
24. Joseph TA, DeFranco MJ, Weiker GG. Delayed repair of a pectoralis major tendon rupture with allograft: a case report. *J Shoulder Elb Surg.* 2003;12:101–4.
25. Schachter AK, White BJ, Namkoong S, Sherman O. Revision reconstruction of a pectoralis major tendon rupture using hamstring autograft a case report. *Am J Sports Med.* 2006;34:295–8.
26. Sikka RS, Neault M, Guanche CA. Reconstruction of the pectoralis major tendon with fascia lata allograft. *Orthopedics.* 2005;28:1199–202.
27. Zacchilli MA, Fowler JT, Owens BD. Allograft reconstruction of chronic pectoralis major tendon ruptures. *J Surg Orthop Adv.* 2012;22:95–102.
28. Zafra M, Muñoz F, Carpintero P. Chronic rupture of the pectoralis major muscle: report of two cases. *Acta Orthop Belg.* 2005;71:107–10.



# Principles of Sport-Specific Rehabilitation

# 13

Ian Horsley

## Key Learning Points

- Rehabilitation of the sporting shoulder is highly specialised
- Shoulder function depends upon energy transfer from proximal to distal segments and so rehabilitation of the whole kinetic chain is essential
- Athletes often develop a functional increase in external rotation and compensatory decrease in internal rotation (GIRD). Increasing GIRD may be associated with increased injury risk.
- Little evidence exists for return to play criteria or validated sports shoulder assessment tools
- Hand held dynamometry is a useful objective measure to guide rehabilitation

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## 13.1 Introduction

Athletes need shoulders with the functional mobility and stability necessary to cope with the speeds, loads, ranges and repetitions of their sports. Within some sports, the shoulder has been recorded to move at speeds of around 1500–10,000 deg/s [1, 2] and has been reported at attaining over 16,000 different positions [3].

Currently, there are no reported validated instruments that are designed to assess the function requirements of the upper limb in sporting athletes, although several authors have published recommendations as to what criteria need to be attained when rehabilitating the overhead athlete, including among others; full range of motion [4–8], full strength [4, 9–11], pain free motion [6, 8–11]. Several of these measures have been questioned as to whether they are indicative of functional recovery [12].

Although the exact make-up of the rehabilitation will be specific to the type of injury and the necessities of the sport to which the athlete is returning, it has been accepted that the rehabilitation needs to be divided into distinct stages based on specific entry and exit criteria. These criteria are specific for the area injured, the type of sport and specific for the patient. Every patient responds differently to an injury, and although the general time frames for healing have been established [13] reliance on a time-based rehabilitation programme rather than on a goal-driven programme could have detrimental consequences.

Like any joint injury, rehabilitation following an injury to the shoulder includes relevant pain management, reduction of inflammation, restoration of optimal muscle strength, and restoration of a functional joint range of motion. Rehabilitation should also progress along a continuum to include functional movements that replicate the demands of the sport. This progression is underpinned by a fundamental requirement for joint control and, as such, there is an inherent need to address proprioceptive awareness, dynamic stabilisation, feed-forward mechanisms (through anticipatory muscle responses), and reactive muscle function to athletic demands [14].

## 13.2 Return to Play Criteria

Return to play after a shoulder injury should be based on objective measurements [15] and the process include evaluation of the athlete's health status, participation risk and extrinsic factors [16].

The 'ideal' criteria for return to play were suggested by McCarty et al. [17] as being:

- Little/no pain
- Patient subjectivity
- Near-normal ROM
- Near-normal strength
- Normal functional ability
- Normal sport-specific skills

The popular proposed biomechanical model for hitting and throwing sports is an open-linked system of segments that operates in a proximal-to-distal sequence [18]. The purpose of these actions is to convey a high velocity or force on the distal segment. The final velocity of the distal segment is dependent upon the velocity of the proximal segment and the interaction between proximal and distal segments [19]. The proximal segments, the lower limbs and torso, accelerate the entire system and consecutively transfer momentum to the subsequent distal segment [18]. Hence, when assessing and rehabilitating the overhead athlete, each segment needs to be assessed to ascertain if there is the required range of movement and muscular control [20].

With this thought in mind, further attention needs to be paid to the position of the athlete at the time when optimal shoulder girdle function is required, and the requirements of that specific sport. Areas to consider are listed in Table 13.1.

Since the kinetic chain has a role in optimal function of the shoulder girdle, one must take into consideration the distal parts and their influence on local function; the scapula acts as a link between the lower limb and trunk (e.g. through the fascial connection between gluteus maximus and latissimus dorsi), the glenohumeral joint and upper limb, permitting effective transfer

**Table 13.1** Context for shoulder sporting function

Context	
What does the shoulder have to do?	
Anchor point	Foot
	Hip
	Trunk
Release point/action point	Above shoulder
	Align with shoulder
	Below shoulder
	Unilateral
	Bilateral
Transverse rotation	No
	Symmetrical
	Asymmetrical
Arm(s)	Single arm
	Double arm
Characteristic	High force
	High rate of force development (RFD)
	High endurance
	High speed

of forces and joint alignment [21]. Establishing a stable scapular platform is essential in minimising stresses to the shoulder during overhead movements, enabling the rotator cuff muscles to help stabilise the humeral head within the glenoid. The scapula will be influenced by the architecture and geometry of the thoracic spine, which will be influenced by the function of the lumbar spine, pelvis and lower limbs [22]. Therefore, it is essential that sub-optimal movement strategies elsewhere in the kinetic chain should be identified and included in the rehabilitation process.



In addition to the prerequisites of the sport, the requirements of the joints need to be considered. It has been proposed that within the kinetic chain, a balance is required between stability and mobility at joints with optimal performance being produced by an alternating sequence of mobility-stability from distal to proximal (Table 13.2) [23].

Integrating the components from Tables 13.1 and 13.2 will lead us to the required screening tools necessary to provide answers (Table 13.3).

**Table 13.2** Optimal demands of the kinetic chain




Joint	Requirement
Ankle	Mobility
Knee	Stability
Hip	Mobility
Lumbar spine	Stability
Thoracic spine	Mobility
Scapula	Stability
Glenohumeral	Mobility

**Table 13.3** Screening tools for sporting shoulder

Area	Key test	Reference
Thoracic rotation	Locked lumbar rotation	Johnson and Grindstaff [24] 
Thoracic extension	Combined elevation test	Dennis et al. [25] 



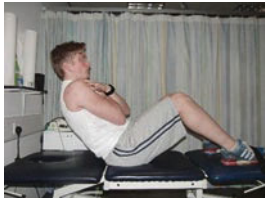


(continued)

**Table 13.3** (continued)

Area	Key test	Reference
Shoulder Internal rotation		Cools et al. [26] 
Shoulder External rotation		Cools et al. [26] 
Hip Internal/external rotation		Barbee-Ellison et al. [27] 





**Table 13.3** (continued)

Area	Key test	Reference
Trunk endurance	Extensor endurance test	Biering-Sorensen [28] 
	Lateral endurance test	McGill et al. [29] 
	The flexor endurance test	McGill et al. [30] 
	Flexion-rotation trunk test	Brotos-Gil et al. [31] 
Trunk muscle strength (the ability of the musculature to generate force through contractile forces and intra-abdominal pressure)	Double leg lowering test	Cutter and Kevorkian [32] 

(continued)

**Table 13.3** (continued)

Area	Key test	Reference
Trunk rate of force development	Front abdominal power test	Cowley and Swensen [33] 
	Side abdominal power test	Cowley and Swensen [33] 
Single leg power	Single leg counter movement jump	Hewit et al. [34]
Single leg force capacity	Single leg mid-thigh pull/ single leg isometric squat	Owens et al. [35]
Single leg reactive strength	Single leg reactive strength index 3 hop for distance	Stalboom et al. [36]
Single leg stability	SEBT	Gribble et al. [37]

### 13.3 Thoracic Spine (T-spine)

The T-spine is comprised of 12 vertebrae, which allow flexion, extension, and rotation within those 12 segments. The ribs attach from T1 to T10 and the T-spine has thinner intervertebral discs than the lumbar spine, which adds to its relative inflexibility. T-Spine movement is described as “coupled” such that lateral bending and rotation are obligated to occur together. The T-spine essentially works as two distinctly different subgroups. The upper T-spine (T1–T5) has ipsilateral coupling of the lateral bending and rotation whereas the mid-lower T-spine (T6–T12) has contralateral coupling i.e. lateral bending and rotation occur in opposite directions [38]. Crosbie et al. [39] report that the ratio of upper to lower thoracic extension during bilateral arm elevation was 1:3, and with unilateral arm elevation ipsilateral thoracic rotation occurs. Hence the clinical assessment of the spine needs to be incorporated into management.

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### 13.4 Rotation Range of Movement

The physical demands of sport-specific performance on an athlete’s body is responsible for specific musculoskeletal adaptation. Professional athletes are engaged for most of their sporting life in training and competition [40]. Repetitive muscular activity in the upper limb, necessary for optimal performance of overhead activities and specific movement patterns, leads to the development of sport-specific muscular adaptation in overhead players. Muscular imbalances within rotator-cuff and the peri-scapular muscles, combined with sub-optimal muscular endurance and inappropriate biomechanics can be responsible for overuse injury in the glenohumeral joint of overhead activity players [41].

Repetitive overhead movements commonly lead to the overuse injuries seen in athletes [42]. The deceleration phase of overhead sporting activity has been identified as being most damaging because of the extreme forces placed on the shoulder [20]. The issue of arm dominance has been reported in literature as being

**Table 13.4** Adaptive changes in shoulder rotation ranges in selected sports

Sport	Internal rotation	External rotation
Non-athlete	70°	90°
College swimming [44]	49°	100°
Professional baseball [45]	57°	109°
Junior tennis [46]	55°	105°

responsible for changes in the range of rotation in unilateral sports played above 90° of elevation [43]. Typically, these athletes present with functional increases in external rotation (ER) and concomitant decreases in internal rotation (IR) (Table 13.4).

Range of motion changes have also been reported in painful throwing shoulders [47–49]. Burkhart et al. [48] suggest that primary posterior inferior capsular contracture could be the potential source of the disabled throwing shoulder and that it can be measured by a glenohumeral internal rotation deficit (GIRD), and report that GIRD occurs before any other motion adaptation occurs.

Measurement of GIRD is assessed relative to the total arc of motion of the glenohumeral joint; total arc of motion is the sum of the measured glenohumeral IR + ER. It has been proposed that a healthy shoulder should present with a 180° arc of motion or, to be more functionally correct, the arc of motion should be equal bilaterally [11]. Previous researchers have documented <20° side difference for IR, and <10% side difference for total ROM as being acceptable values that are unlikely to contribute to pathology [50–54]. Predictive findings have been proposed as; loss of >25° into IR [55] in baseball and softball, and a loss of 20° IR and a loss of 5% in total ROM doubles the risk for injury in professional baseball pitchers. Although Clarsen et al. [56] were unable to find any associations between glenohumeral internal rotation deficits, external rotation deficits or total range of movement differences and injury.

## 13.5 Shoulder Strength

One of the contributing factors to shoulder injury, and detected on clinical assessment of symptomatic patients is reduction in shoulder strength around the rotator cuff and scapular muscles. The existence of an imbalance between the agonist and antagonist muscle groups has been shown to be one of the major risk factors for developing shoulder injuries [57], with a reduction in the external rotator strength conceivably causing an injury [58].

Controversy exists in the literature as to whether absolute strength or the IR:ER strength ratio should be utilised to quantify the ideal levels of dynamic shoulder stability, particularly in overhead athletes [59]. Several researchers [11, 46–48, 60] have advocated that the combination of forceful, eccentric contractions coupled with high distraction forces may cause microtrauma to the external rotators and posterior cuff during the follow through/deceleration that will re-model in accordance with Wolff's Law, which states that tissues will adapt to the stresses placed on them [61].

Many methods of assessing the strength around the shoulder girdle have been used; isokinetic dynamometers [62, 63], weight lifting [64], manual muscle testing (MMT) [65, 66], and hand held dynamometers (HHD) [26, 65].

Isokinetic dynamometers have been used as a clinical measure of muscle strength and endurance of the rotator cuff and the scapular stabilisers [26] and have the capability to measure strength at different speeds [67, 68], but these dynamometers are not readily available and the clinical validity of the results can be brought into question.

MMT has good clinically utility but is highly subject to user error and bias [65, 69] and it is difficult to assess small changes in muscle strength and present objective data utilising this method [65]. The results can be influenced by the experience and strength of the examiner [70, 71].

HHD is a more objective method of evaluation and is far superior to MMT when evaluating changes in muscle strength caused by dysfunction [72]. Numerous studies have reported the reliability of HHD to assess upper limb muscle strength including scapular muscles [65, 73, 74].

### 13.6 Methods of Testing

The reliability of HHDs has been examined in many studies and found to have reasonable inter-rater and intra-rater reliability of shoulder internal and external rotation [65, 75, 76]. Although several different positions have been reported in literature, recent investigations have shown good to excellent intra- and inter-tester reliability established for IR and ER isometric strength measurements, regardless of patient or shoulder position used [26, 77] (Tables 13.5 and 13.6).

**Table 13.5** Outcome measures and return to play decision

Outcome measurements and return to play decision	Glenohumeral joint [26, 78]	Scapulothoracic joint [51]
	GH IR and ER ROM	Scapular upward rotation
	GH rotator cuff strength	Strength of the scapular stabilisers
	Eccentric strength of the external rotators	PM length/PM index



**Table 13.6** Muscle function assessment

Muscle	Position	Reference
Upper trapezius recruitment	Seated HHD over upper border of scapula to resist shoulder elevation	Hislop et al. [79]
Middle trapezius recruitment	Prone lying arm at 90° abduction. HHD placed midway between root of spine of scapula and acromion to resist scapular retraction	Michener et al. [73]
Lower trapezius recruitment	Prone lying arm 120° elevation. HHD placed midway between root of spine of scapula and acromion to resist scapular adduction and depression	Michener et al. [73]
Serratus anterior recruitment	Supine shoulder flexion 90° and 110° adduction. HHD placed against flexed elbow to resist scapular protraction	Kendall et al. [80]
Posterior shoulder endurance	Subject lays prone with a weight equal to 2% bodyweight horizontally abducts arm at controlled cadence until unable to achieve 1 s hold in horizontal abduction	Moore et al. [81]

### 13.7 Muscular Endurance

In addition to glenohumeral and scapulothoracic control, emphasis needs to be placed on the assessment of muscular endurance, since shoulder muscle fatigue has been proposed to be associated with repeated arm use and the development of rotator cuff disorders [82]. The mechanism for this has been postulated as being due to fatigue and altered timing of the local muscle system around the shoulder girdle [82, 83]. Muscles that are fatigued absorb less energy before they are elongated and can result in injury [84]. Within literature there are no agreed definitions of exactly what localised muscle fatigue consists of due to the methodological problems in segregating the different components of fatigue [58]. Within a clinical setting, attention has primarily focused on localised muscle fatigue evidenced by an inability of the muscle(s) to sustain a desired force output, or increase output, even though the desired motion may continue to take place [85].

### 13.8 Proprioception

A vital element of the rehabilitation programme is explicit proprioceptive facilitation to aid the patient's motor re-learning and re-educate normal muscle recruitment patterns. Tactile and verbal cues can be utilised to augment the patient's understanding of the desired activation pattern required [86].

Observation and palpation of the humeral head during isometric muscle testing of the rotator cuff will regularly demonstrate translation in an anterior or posterior direction. This indicates poor ability of the cuff to maintain the humeral head in a centred position on the glenoid. The dynamic rotary stability test (DRST) can be performed in different degrees of elevation depending on symptomology, postoperative phase and functional relevance [87].

Proprioception has been defined as “a sensory afferent feedback mechanism that mediates joint position and movement sensibility with muscular reflex stabilisation” [88]. Mechanoreceptors within the capsule-labral complex around the shoulder detecting joint position and movement have been proposed to be damaged due to local tissue trauma, with deficiencies in joint proprioception ensuing [89, 90]. Reduced joint proprioception has been proposed as a significant contributing factor to re-injury [89–91].

Joint position sense (JPS) has been defined as “the appreciation and interpretation of information concerning one's joint position and orientation in space” [92]. Several different methods have been utilised in literature to measure this: isokinetic dynamometry [93, 94], customised devices [3, 90, 95], electronic tracking devices [96], inclinometers [97, 98] and photography [99], and several differing outcome measures have been reported:

- *Absolute Matching Error* denotes the actual difference between the target criterion angle and the angle recorded by the subject, but does not refer to whether the angle attained was less than (under shoot) or greater than (over shoot) the set angle [90, 93, 99, 100].



- *Constant Matching Error* describes the average mean error bias and reflects directional accuracy [3, 93]
- *Variable Matching Error* records the standard deviation of absolute matching errors over 3 trials [93, 101].

Many of these devices are not readily available in a clinical setting and have high costs attached, thus rendering these techniques impractical. Hence more utilitarian methods are required.

The assessment of proprioception using “reproduction of passive positioning” is a valid and established method reported by Barrett [102]. Clinically, joint angular replication tests—whereby the shoulder is placed in a position and the patient holds it in that position and consciously registers the position, then the arm is returned to a resting position. The subject is then asked to return the arm to the test position. This test has been described by Davies and Dickoff-Hoffman [103] and assesses both the static and dynamic shoulder joint stabilisers, providing a thorough afferent pathway assessment [104]. Other examples of open kinetic chain exercises are;

- **Joint angle repositioning:** the shoulder joint is taken to a specific position in space (generally a combination of abduction and external rotation) by the examiner. The subject (who has their eyes closed in order to negate visual cues) is asked to hold this position for 5 s, then the limb is moved to the starting position, and the subject is asked to move to the test position. The degree of error from the stated position is recorded.
- **Contra lateral limb mirroring:** the subject’s uninvolved shoulder is placed in a position in space (whilst they have their eyes closed) and the subject is asked to mirror that position with the “involved” limb. Once again the degree of error between the two sides is noted.
- **Balance Point Test:** developed at the Royal National Orthopaedic Hospital, Stanmore, London [105]. In side lying, the upper arm is passively placed in 90° of humeral abduction and held there via an isometric hold by the subject for 5 s. The subject then lowers the arm and tries to reproduce the 90° abduction angle. Note is made of any deviation from the vertical.

## 13.9 Conclusion

The sporting athlete represents an extremely specific population and, in addition to this, the specific sub-groups of sporting activity is even more specialised. Since force transfer to the shoulder is initiated via the kinetic chain, a full biomechanical screening process needs to be carried out.

Many of the current functional assessment tools reported in the literature are for use with a sedentary, non-sporting, population, and not suitable for use as a return-to-play assessment of athletes. Furthermore, using screening information from other sports is not always transferable to other (similar) sports due to the specialist movement characteristics of each sport.

Thus, it is recommended that the component parts of the specific sporting activity are analysed with respect to the activity of the shoulder as part of a pre-participation screening and return-to-play assessment.

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## Q&A

- (1) What is the kinetic chain?

A proposed biomechanical model for hitting and throwing sports is an open-linked system of segments that operates in a proximal-to-distal sequence. The purpose of these actions is to convey a high velocity or force on the distal segment

- (2) Describe general return to play criteria?

Little/no pain, near-normal ROM, near-normal strength, normal functional ability, normal sport-specific skills

- (3) Why is it important to assess glenohumeral joint range of rotation in the athletic shoulder?

Range of motion changes have also been reported in painful throwing shoulders suggesting that primary posterior inferior capsular contracture could be the potential source of the disabled throwing shoulder and that it can be measured by a glenohumeral internal rotation deficit (GIRD)

- (4) What means are available to objectively test shoulder strength? Many methods of assessing the strength around the shoulder girdle have been used including isokinetic dynamometers, weight lifting, manual muscle testing (MMT) and hand held dynamometers (HHD).

- (5) What clinical test are described to evaluate shoulder proprioception?

The assessment of proprioception using “reproduction of passive positioning” is a valid and established method reported by Barrett [102]. Clinically, joint angular replication tests—whereby the shoulder is placed in a position and the patient holds it in that position and consciously registers the position, then the arm is returned to a resting position.

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## References

1. Kibler WB. Biomechanical analysis of the shoulder during tennis activities. *Clin Sports Med.* 1995;14(1):79–85.
2. Werner SL, Gill TJ, Murray TA, Cook TD, Hawkins RJ. Relationships between throwing mechanics and shoulder distraction in professional baseball pitchers. *Am J Sports Med.* 2001;29(3):354–8.
3. Lephart SM. Re-establishing proprioception, kinesthesia, joint position sense and neuromuscular control in rehab. In: Prentice WE, editor. *Rehabilitation techniques in sports medicine.* 2nd ed. St. Louis: Mosby; 1994. p. 118–37.
4. Andrews JR, Dugas JR, Hackel JG, Reinold MM, Wilk KE. The thrower’s exostosis pathophysiology and management. *Tech Should Elbow Surg.* 2004;5(1):44–50.
5. Arroyo JS, Hershon SJ, Bigliani LU. Special considerations in the athletic throwing shoulder. *Orthop Clin N Am.* 1997;28(1):69–78.
6. Cavallo R, Speer K. Shoulder instability and impingement in throwing athletes. *Med Sci Sports Exerc.* 1998;30:18–25.
7. Kelly L, Terry GC. Team handball: shoulder injuries, rehabilitation, and training. *Sports Med Arthrosc Rev.* 2001;9(2):115–23.
8. O’Donnell C, Bowen J, Fossati J. Identifying and managing shoulder pain in competitive swimmers: how to minimize training flaws and other risks. *Phys Sportsmed.* 2005;33(9):27.
9. Andrews JR, Alexander EJ. Rotator cuff injury in throwing and racquet sports. *Sports Med Arthrosc Rev.* 1995;3:30–8.
10. Wilk KE, Obma P, Simpson CD, Cain EL, Dugas J, Andrews JR. Shoulder injuries in the overhead athlete. *J Orthop Sports Phys Ther.* 2009;39(2):38–54.

11. Wilk KE, Meister K, Andrews JR. Current concepts in the rehabilitation of the overhead throwing athlete. *Am J Sports Med.* 2002;30(1):136–51.
12. Kirkley A, Alvarez C, Griffin S. The development and evaluation of a disease-specific quality-of-life questionnaire for disorders of the rotator cuff: the Western Ontario Rotator Cuff Index. *Clin J Sport Med.* 2003;13(2):84–92.
13. Evans P. The healing process at cellular level: a review. *Physiotherapy.* 1980;66(8):256–9.
14. Lephart S, Henry T. Functional rehabilitation for the upper and lower extremity. *Orthop Clin N Am.* 1995;26(3):579–92.
15. Matheson GO, Shultz R, Bido J, Mitten MJ, Meeuwisse WH, Shrier I. Return-to-play decisions: are they the team physician's responsibility? *Clin J Sport Med.* 2011;21:25–30.
16. Creighton DW, Shrier I, Shultz R, Meeuwisse WH, Matheson GO. Return-to-play in sport: a decision based model. *Clin J Sport Med.* 2010;20:379–85.
17. McCarty EC, Ritchie P, Gil LHS, McFarland EG. Shoulder instability: return to play. *Clin Sports Med.* 2004;23:335–51.
18. Putnam CA. Sequential motions of body segments in striking and throwing skills: description and explanations. *J Biomech.* 1993;26:125–35.
19. Watkins RG, Dennis S, Dillin WH, et al. Dynamic EMG analysis of torque transfer in professional baseball pitchers. *Spine.* 1989;14:404–8.
20. Park SS, Loebenberg ML, Rokito AS, Zuckerman JD. The shoulder in baseball pitching biomechanics and related injuries—part 2. *Bull Hosp Jt Dis.* 2003;61(1–2):80–7.
21. Yamauchi T, Hasegawa S, Matsumura A, Nakamura M, Ibuki S, Ichihashi N. The effect of trunk rotation during shoulder exercises on the activity of the scapular muscle and scapular kinematics. *JSES.* 2015;24(6):955–64.
22. Sciascia A, Cromwell R. Kinetic chain rehabilitation: a theoretical framework. *Rehabil Res Pract.* 2012;2012:853037.
23. Boyle M. *Functional training for sports.* 2nd ed. Champaign: Human Kinetics; 2016.
24. Johnson KD, Grindstaff TL. Thoracic rotation measurement techniques: clinical commentary. *N Am J Sports Phys Ther.* 2012;5(4):252–6.
25. Dennis RJ, Finch CF, McIntosh AS, Elliott BC. Using field-based tests to identify risk factors for fast bowlers in cricket. *Br J Sports Med.* 2008;42:477–82.
26. Cools AM, Witvrouw EE, Danneels LA, et al. Test-retest reproducibility of concentric strength values for shoulder girdle protraction and retraction using the Biodex isokinetic dynamometer. *Isokinet Exerc Sci.* 2002;10:129–36.
27. Barbee-Ellison JB, Rose SJ, Sahrman SA. Patterns of hip rotation range of motion: comparison between healthy subjects and patients with low back pain. *Phys Ther.* 1990;70:537–41.
28. Biering-Sorensen E. Physical measurements as risk indicators for low-back trouble over a one-year period. *Spine.* 1984;9:106–19.

29. McGill SM, Grenier S, Kavcic N, Cholewicki J. Coordination of muscle activity to assure stability of the lumbar spine. *J Electromyogr Kinesiol.* 2003;13(4):353–9.
30. McGill SM, Childs A, Liebenson C. Endurance times for low back stabilization exercises: clinical targets for testing and training from a normal database. *Arch Phys Med Rehabil.* 1999;80:941–4.
31. Brotons-Gil E, García-Vaquero MP, Peco-González N, Vera-García FJ. Flexion-rotation trunk test to assess abdominal muscle endurance: reliability, learning effect, and sex differences. *J Strength Cond Res.* 2013;27(6):1602–8.
32. Cutter NC, Kevorkian CG. *Handbook of manual muscle testing.* 1st ed. New York: McGraw-Hill; 1999. p. 193–4.
33. Cowley P, Swensen T. Development and reliability of two core stability field tests. *J Strength Cond Res.* 2009;22(2):619–24.
34. Hewitt JK, Cronin JB, Hume PA. Asymmetry in multi-directional jumping tasks. *Phys Ther Sport.* 2012;13(4):238–42.
35. Owens EM, Serrano AJ, Ramsey MW, Mizuguchi S, Johnston B, Stone MH. Comparing lower-limb asymmetries in NCAA D-I male and female athletes. *J Strength Cond Res.* 2011;25:S44.
36. Stalbam M, Jonsson Holm D, Cronin JB, Keogh JWL. Reliability of kinematics and kinetics associated with horizontal single leg drop jump assessment. a brief report. *J Sports Sci Med.* 2007;6:261–4.
37. Gribble PA, Hertel J, Plisky P. Using the star excursion balance test to assess dynamic postural-control deficits and outcomes in lower extremity injury: a literature and systematic review. *J Athl Train.* 2012;47(3):339–57.
38. Panjabi MM, Brand RA, White AA. Mechanical properties of the human thoracic spine. *J Bone Joint Surg.* 1976;58(A):642–52.
39. Crosbie J, Kilbreath SL, Hollmann L, York S. Scapulohumeral rhythm and associated spinal motion. *Clin Biomech.* 2008;23:184–92.
40. 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.
41. Ellenbecker T, Robert EP. Age specific isokinetic glenohumeral internal and external rotation strength in elite junior tennis players. *J Sci Med Sport.* 2003;6(1):63–70.
42. Arnheim DD, Prentice WE, Arnheim DD. *Principles of athletic training.* St. Louis: McGraw-Hill; 1997.
43. Thomas SJ, Swanik KA, Swanik CB, Kelly JDT. Internal rotation deficits affect scapular positioning in baseball players. *Clin Orthop Relat Res.* 2010;468:1551–7.
44. Beach ML, Whitney SL, Dickoff-Hoffman SA. Relationship of shoulder flexibility, strength, and endurance to shoulder pain in competitive swimmers. *J Sports Phys Ther.* 1992;16(6):262–8.

45. Downar JM, Sauers EL. Clinical measures of shoulder mobility on the professional baseball player. *J Athl Train.* 2005;40(1):23–9.
46. Ellenbecker TS, Roetert EP, Piorkowski PA, Schulz DA. Glenohumeral joint internal and external rotation range of motion in elite junior tennis players. *J Orthop Sports Phys Ther.* 1996;24(6):336–41.
47. Burkhart SS, Morgan CD, Kibler WB. The disabled throwing shoulder: spectrum of pathology part I: pathoanatomy and biomechanics. *Arthroscopy.* 2003;19(4):404–20.
48. Burkhart SS, Morgan CD, Kibler WB. The disabled throwing shoulder: spectrum of pathology part III: the SICK scapula, scapular dyskinesis, the kinetic chain, and rehabilitation. *Arthroscopy.* 2003;19:641–461.
49. Kibler WB. Shoulder rehabilitation: principles and practice. *Med Sci Sports Med.* 1998;30:S40–50.
50. Braun S, Kokmeyer D, Millett PJ. Shoulder Injuries in the throwing athlete. *J Bone Joint Surg Am.* 2009;91(4):966–78.
51. Ellenbecker TS, Cools A. Rehabilitation of shoulder impingement syndrome and rotator cuff injuries: an evidence-based review. *Br J Sports Med.* 2010;44(5):319–27.
52. Reinold MM, Gill TJ. Current concepts in the evaluation and treatment of the shoulder in overhead-throwing athletes, part 1: physical characteristics and clinical examination. *Sports Health.* 2010;2(1):39–50.
53. Shanley E, Rauh MJ, Michener LA, Ellenbecker TS, Garrison JC, Thigpen CA. Shoulder range of motion measures as risk factors for shoulder and elbow injuries in high school softball and baseball players. *Am J Sports Med.* 2011;39(9):1997–2006.
54. Tate A, Turner GN, Knab SE, et al. Risk factors associated with shoulder pain and disability across the lifespan of competitive swimmers. *J Athl Train.* 2012;47(2):149–58.
55. Shanley E, Michener LA, Ellenbecker TS, Rauh MJ. Shoulder range of motion, pitch count, and injuries among interscholastic female softball pitchers: a descriptive study. *Int J Sports Phys Ther.* 2012;7(5):548–57.
56. Clarsen B, Bahr R, Haugsboe Andersson S, Munk R, Myklebust G. Reduced glenohumeral rotation, external rotation weakness and scapular dyskinesis are risk factors for shoulder injuries among elite male handball players: a prospective cohort study. *Br J Sports Med.* 2014;48(7):579.
57. Luttgens K, Deutsch H, Hamilton N. *Kinesiology: scientific basis of human motion.* 8th ed. Madison: Brown and Benchmark; 1992.
58. Donatelli RA, Ellenbecker T, Ekedahl S, Wilkes JS, Kocher PT, Adam J. Assessment of shoulder strength in professional baseball pitchers. *J Orthop Sports Phys Ther.* 2000;30:544–51.
59. McMaster WC, Long SC, Caiozzo VJ. Shoulder torque changes in the swimming athlete. *Am J Sports Med.* 1992;20(3):323–7.
60. Bigliani LU, Codd TP, Connor PM, Levine WN, Littlefield MA, Hershon SJ. Shoulder motion and laxity in the professional baseball player. *Am J Sports Med.* 1997;25(5):609–13.

61. Prentice W. Rehabilitation techniques for sports medicine and athletic training. Boston: McGraw-Hill; 2004.
62. Dvir Z. Isokinetics muscle testing: interpretation and clinical applications. Philadelphia: Churchill Livingstone; 1995. p. 171–93.
63. Ellenbecker TS, Davies GJ. The application of isokinetics in testing and rehabilitation of the shoulder complex. *J Athl Train.* 2000;35(3):338–50.
64. Sale DG. Testing strength and power. In: MacDougall JD, Wenger HA, Green HJ, editors. *Physiological testing of the high-performance athlete.* Champaign: Human Kinetics; 1991.
65. Hayes K, Walton JR, Szomor ZL, Murrell GA. Reliability of 3 methods for assessing shoulder strength. *J Shoulder Elb Surg.* 2002;11(1):33–9.
66. Murrell GAC, Walton J. Diagnosis of rotator cuff tears. *Lancet.* 2001;357:769–70.
67. Greenfield BH, Donatelli R, Wooden MJ, Wilkes J. Isokinetic evaluation of shoulder rotational strength between the plane of scapula and the frontal plane. *Am J Sports Med.* 1990;18:124–8.
68. Rabin SI, Post M. A comparative study of clinical muscle testing and cybex evaluation after shoulder operations. *Clin Orthop.* 1990;258:147–56.
69. Wadsworth DJ, Bullock-Saxton JE. Recruitment patterns of the scapular rotator muscles in freestyle swimmers with subacromial impingement. *Int J Sports Med.* 1997;18:618–24.
70. Aitkens S, Lord J, Bernauer E, Fowler WM, Lieberman JS, Berck P. Relationship of manual muscle testing to objective strength measurements. *Muscle Nerve.* 1989;12:173–7.
71. Bohannon RW. Testing isometric limb muscle strength with dynamometers. *Crit Rev Phys Rehabil Med.* 1990;2:75–86.
72. Schwartz S, Cohen ME, Herbison GJ, Shah A. Relationship between two measures of upper extremity strength. Manual muscle test compared to hand-held myometry. *Arch Phys Med Rehabil.* 1992;73(11):1063–8.
73. Michener LA, Boardman ND, Pidcoe PE, Frith AM. Scapular muscle tests in subjects with shoulder pain and functional loss: reliability and construct validity. *Phys Ther.* 2005;85:1128–38.
74. Celik D, Sirmen B, Demirhan M. The relationship of muscle strength and pain in subacromial impingement syndrome. *Acta Orthop Traumatol Turc.* 2011;45(2):79–84.
75. Leggin B, Neuman R, Iannotti J, Williams G, Thompson E. Intrarater and interrater reliability of three isometric dynamometers in assessing shoulder strength. *J Shoulder Elb Surg.* 1996;5:18–24.
76. Sullivan S, Chesley A, Hebert G, McFaul S, Scullion D. The validity and reliability of hand-held dynamometry in assessing isometric external rotator performance. *Phys Ther.* 1988;10:213–7.
77. Riemann BL, Davies GJ, Ludwig L, Gardenhour H. Hand-held dynamometer testing of the internal and external rotator musculature based on selected positions to establish normative data and unilateral ratios. *J Shoulder Elb Surg.* 2010;19:1175–83.

78. Johansson FR, Skillgate E, Lapauw ML, Clijmans D, Deneulin VP, Palmans T, Cools AM. Measuring eccentric strength of the shoulder external rotators using a hand-held dynamometer: reliability and validity. *J Athl Train*. 2015;50(7):719–25.
79. Hislop HJ, Montgomery J, Connelly B, Daniels L. *Daniel's and Worthingham's muscle testing techniques of manual examination*. Philadelphia: W.B. Saunders; 1995.
80. Kendall FP, McCreary EK, Provance PG. *Muscles testing and function*. 4th ed. Baltimore: Williams & Wilkins; 1993.
81. Moore SD, Uhl TL, Kibler WB. Improvements in shoulder endurance following a baseball-specific strengthening program in high school baseball players. *Sports Health*. 2013;5(3):233–8.
82. Fuller JR, Lomond KV, Fung J, Cote JN. Posture-movement changes following repetitive motion induced shoulder muscle fatigue. *J Electromyogr Kinesiol*. 2009;19:1043–52.
83. Blangsted AK, Sjogaard G, Madeleine P, Olsen HB, Sogaard K. Voluntary low-force contraction elicits prolonged low-frequency fatigue and changes in surface electromyography and mechanomyography. *J Electromyogr Kinesiol*. 2005;15:138–48.
84. Mair SD, Seaber AV, et al. The role of fatigue in susceptibility to acute muscle strain injury. *Am J Sports Med*. 1996;24(2):137–43.
85. Barry BK, Enoka RM. The neurobiology of muscle fatigue: 15 years later. *Integr Comp Biol*. 2007;47(4):465–73.
86. Shumway-Cook A, Woollacott MH. *Motor control: theory and practical applications*. 2nd ed. Philadelphia: Lippincott, Williams and Wilkins; 2001.
87. Magarey ME, Jones MA. Specific evaluation of the function of force couples relevant for stabilization of the glenohumeral joint. *Man Ther*. 2003b;8:247–53.
88. Lephart S, Pincivero D, Giraldo J, Fu F. The role of proprioception in the management and rehabilitation of athletic injuries. *Am J Sports Med*. 1977;25(1):130–7.
89. Lephart S, Warner J, Borsa P, Fu F. Proprioception of the shoulder joint in healthy, unstable and surgically repaired shoulders. *J Shoulder Elb Surg*. 1994;3:371–80.
90. Smith R, Brunolli J. Shoulder kinesthesia after anterior glenohumeral joint dislocation. *Phys Ther*. 1990;69:106–12.
91. Forwell L, Carnahan H. Proprioception during manual aiming in individuals with shoulder instability and controls. *J Orthop Sports Phys Ther*. 1996;23(3):111–9.
92. Myers JB, Laudner KG, Pasquale MR, Bradley JP, Lephart SM. Glenohumeral range of motion deficits and posterior shoulder tightness in throwers with pathologic internal impingement. *Am J Sports Med*. 2006;34:385–91.
93. Janwantanakul P. The effect of body orientation on shoulder proprioception. *Phys Ther Sport*. 2003;4:67–73.



94. Voight M, Hardin J, Blackburn T, Tippett S, Canner G. The effect of muscle fatigue on and the relationship of arm dominance to shoulder proprioception. *J Orthop Sports Phys Ther.* 1996;23:348–52.
95. Safran MR, Borsa PA, Lepahrt SM, et al. Shoulder proprioception in baseball pitchers. *J Shoulder Elb Surg.* 2001;10:438–44.
96. Barden JM, Balyk R, Raso J, Moreau M, Bagnall K. Dynamic upper limb proprioception in multidirectional shoulder instability. *Clin Orthop.* 2004;420:181–9.
97. Dover GC, Kaminski TW, Meister K, Powers ME, Horodyski MB. Assessment of shoulder proprioception in the female softball athlete. *Am J Sports Med.* 2003;31:431–7.
98. Dover G, Powers ME. Cryotherapy does not impair shoulder joint position sense. *Arch Phys Med Rehabil.* 2004;85:1241–6.
99. Herrington L, Horsley I, Rolf C. Evaluation of shoulder joint position sense in both asymptomatic and rehabilitated professional rugby players and matched controls. *Phys Ther Sport.* 2010;11(1):18–22.
100. Jerosch J, Prynka M. Proprioception and joint stability. *Knee Surg Sports Traumatol Arthrosc.* 1996;4:171–9.
101. Janwantanakul P, Magarey ME, Jones MA, et al. Variation in shoulder position sense at mid and extreme range of motion. *Arch Phys Med Rehab.* 2001;82:840–4.
102. Barrett DS. Proprioception and function after anterior cruciate reconstruction. *J Bone Joint Surg.* 1991;73:833–7.
103. Davies GJ, Dickoff-Hoffman S. Neuromuscular testing and rehabilitation of the shoulder complex. *J Orthop Sports Phys Ther.* 1993;18(2):449–58.
104. Lepahrt SM, Fu FH. Proprioception and neuromuscular control in joint stability. Champaign: Human Kinetics; 2000.
105. Jaggi A, Lambert S. Shoulder injuries in athletes: Rehabilitation for shoulder instability. *Br J Sports Med.* 2010;44:333–40.

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