

Sports Injuries of the Shoulder

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Mike Walton
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Contents

1 Clinical Anatomy and Biomechanics of the Sporting Shoulder	1
Giulio Maria Marcheggiani Muccioli, Carbone Giuseppe, Grassi Alberto, Zaffagnini Stefano, and Marcacci Maurilio	
2 Shoulder Injuries in Overhead Athletes	23
Teruhisa Mihata	
3 Shoulder Injuries in Contact Athletes	53
Sungjoon Lim and Lennard Funk	
4 Scapular Dyskinesis in Athletes	71
W. Ben Kibler and Aaron Sciascia	
5 Rotator Cuff Disorders in Athletes	99
Joshua A. Greenspoon, Maximilian Petri, Sanjeev Bhatia, and Peter J. Millett	
6 Labral Injuries in Athletes	117
Jin Young Park and Jae Hyung Lee	
7 Glenoid Bone Loss in Athletes	137
Deepak N. Bhatia and Joe F. De Beer	
8 Acromioclavicular Joint Injuries	153
Lennard Funk and Mohamed A. Imam	
9 The Sternoclavicular Joint	179
Graham Tytherleigh-Strong, Elizabeth Pinder, and Muiris Kennedy	

10	Clavicle Fractures	203
	David Copas and Michael Walton	
11	Glenohumeral Arthritis in Athletes	221
	Peter A. D’Alessandro and Andrew L. Wallace	
12	Pectoralis Major Ruptures	249
	Usman Butt and Puneet Monga	
13	Principles of Sport-Specific Rehabilitation	261
	Ian Horsley	
	Index	285



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.

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.

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° (range 9° to 31°) and inclined on average 41° (range 34 – 47°); 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

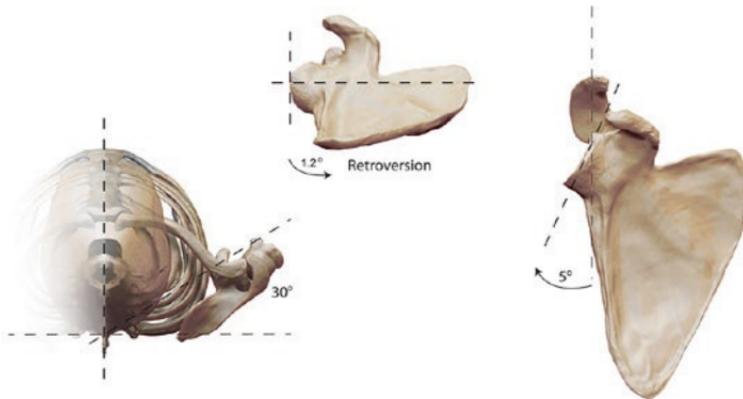


Fig. 1.1 Bony geometry of the scapula and glenoid (courtesy of Lennard Funk, <http://www.shoulderdoc.co.uk>)

superior inferior direction. It is retroverted on average 1.2° (range 9.5° of anteversion to 10.5° of retroversion) and inclined superiorly on average 5° (range 7° of inferior inclination to 15.8° 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 (2×0.3 cm), moderately severe (4×0.5 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-

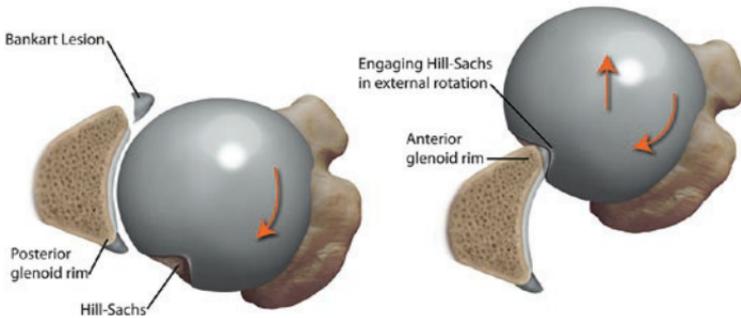


Fig. 1.3 Hill-Sachs lesion of the posterior humeral head (courtesy of Lenard Funk, <http://www.shoulderdoc.co.uk>)

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

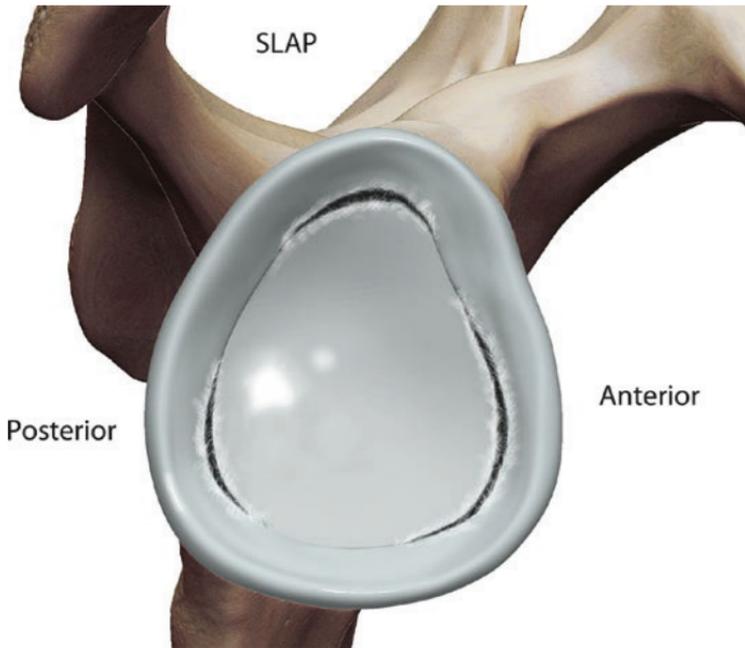


Fig. 1.4 Labral tears occur in the antero-inferior labrum, posterior labrum and superior labrum (SLAP) (courtesy of Lennard Funk, <http://www.shoulderdoc.co.uk>)

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

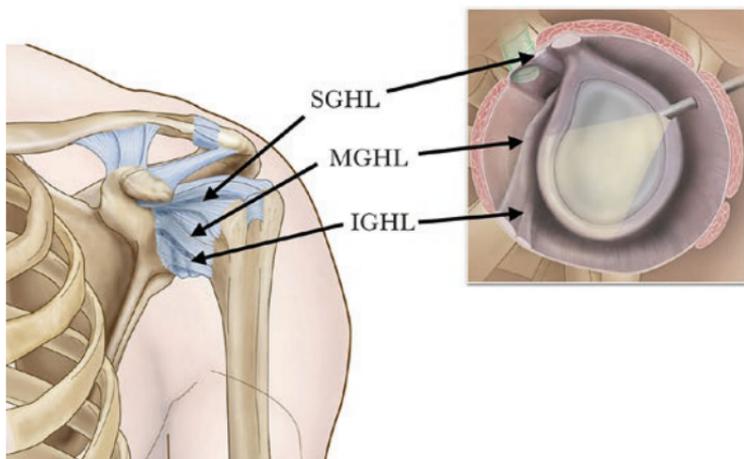


Fig. 1.5 The anterior glenohumeral joint ligaments: Superior (SGHL), Middle (MGHL) and anterior band of the Inferior (IGHL) (courtesy of Lennard Funk, <http://www.shoulderdoc.co.uk>)

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).

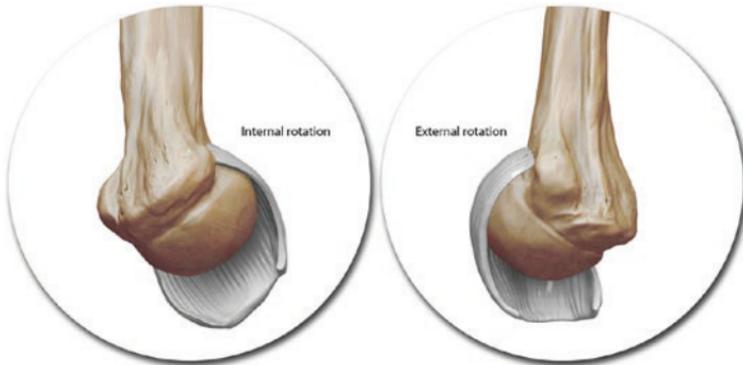


Fig. 1.6 The effect of internal and external rotation on the IGHLC (courtesy of Lennard Funk, <http://www.shoulderdoc.co.uk>)

On the other hand, during adduction, flexion and internal rotation, the IGHLC moves posteriorly, forming a restraint to posterior translation. The IGHLC 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-

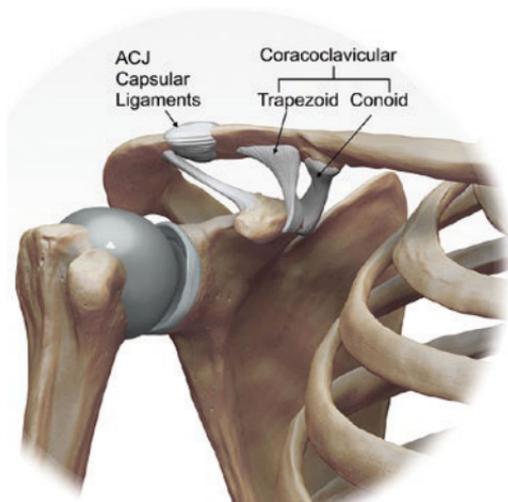
riorly. The rotator interval is composed of the coracohumeral ligament (CHL) and superior and middle glenohumeral ligaments deeper, even if the middle glenohumeral ligament contribution is relatively variable (different studies has reported its absence, from 10 to 40% of cases). Usually, it is larger 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

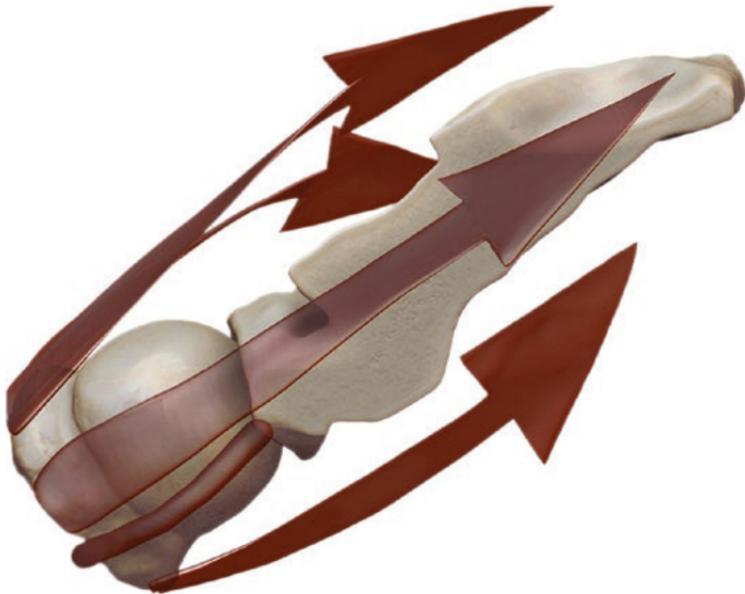


Fig. 1.8 Active compression effect of the rotator cuff to stabilise the humeral head on the glenoid (courtesy of Lennard Funk, <http://www.shoulderdoc.co.uk>)

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.

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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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.

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].

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