Functional Anatomy and Biomechanics of Shoulder Stability in the Athlete

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KEYWORDS

• Instability • Anterior • Posterior • Multidirectional

KEY POINTS

- The large range of motion afforded by the glenohumeral joint results in a propensity for instability.
- The constitutional trait of laxity facilitates extensive motion in multiple planes and may be essential to athletic performance.
- Range of motion and joint distractibility are increased in hyperlaxity, which is considered as instability when associated with loss of function.
- Strength and stability of the joint are highly dependent on both static and dynamic restraints.
- Soft tissue lesions associated with anterior instability include the Bankart lesion, superior labrum anterior and posterior detachment tear, humeral avulsion of the glenohumeral ligaments (HAGL), anterior labroligamentous periosteal sleeve avulsion, and defects of the rotator interval.
- Bony lesions associated with traumatic anterior instability include bony Bankart lesions and Hill-Sachs impression fractures.
- Soft tissue lesions associated with posterior instability include reverse Bankart lesions, plastic deformation of the capsule, Kim lesions, posterior labrocapsular periosteal sleeve avulsion lesions, Bennett lesions, and posterior HAGL.
- Posterior glenoid bone defects and reverse Hill-Sachs impression fractures have been associated with posterior instability.
- Atraumatic instability is most commonly associated with underlying ligamentous laxity or overuse injury but can also be associated with traumatic injury bringing subacute injury to clinical attention.

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INTRODUCTION

The glenohumeral joint provides greater freedom of motion than any other joint in the body at the expense of decreased stability. Glenohumeral joint movements include flexion-extension, abduction-adduction, circumduction, and rotation. Shoulder instability can occur in overhead throwing athletes (chronic overuse injuries) but more commonly occurs in contact athletes (acute traumatic dislocations). It can be conceptually regarded as a continuum of pathology with possible contributions from many of the bony and soft tissue intra-articular shoulder structures. Our understanding of the anatomy and pathologic entities has evolved significantly since initial descriptions of shoulder instability and this has facilitated an evolving repertoire of treatment options. This article reviews the functional anatomy and biomechanics of shoulder stability and outlines the bony and soft tissue lesions associated with shoulder instability in the athlete.

WHAT IS INSTABILITY?

The constitutional trait of hyperlaxity and the pathologic condition of instability represent distinct clinical entities.[1] Laxity is the asymptomatic passive translation of the humeral head on the glenoid and may be essential to athletic performance. In hyperlaxity, this range of joint motion and joint distractibility are increased without loss of function. Glenohumeral instability is defined as excessive translation of the humeral head on the glenoid associated with a functional deficit.[1] These abnormal translations of the humeral head can occur actively or passively and are classically accompanied by symptoms of pain and apprehension. In athletes, instability can present as frank dislocation, subluxation events, or more subtly as microinstability. Symptomatic instability and hyperlaxity both exhibit a spectrum of clinical diversity, and there is no single diagnostic test for the presence of either condition. Although they generally occur independently,[2] instability and hyperlaxity may coexist, particularly in elite athletes who are often hyperlax and prone to injury through sport. In the compensated athlete, static soft tissue and bony deficiency are often counteracted by advanced neuromuscular control. Symptomatic instability results from acute or chronic deterioration in the compensatory dynamic stabilizers of the glenohumeral joint or a frank traumatic event.

NORMAL SHOULDER STABILITY

The balance between stability and mobility within the shoulder is achieved through complex interactions involving static and dynamic restraints.[1] The bony static stabilizers include the glenoid, humeral head, and proximal humerus. The soft tissue passive stabilizers include the glenoid labrum, negative intra-articular pressure, articular cartilage surface, the glenohumeral ligaments, and the glenohumeral joint capsule. Soft tissue dynamic stabilizers are tendon-muscle complexes that provide both function and stability to the shoulder. The joint reaction force is primarily maintained by the rotator cuff muscles, long head of biceps, and the deltoid. However, all muscles that cross the glenohumeral joint, including pectoralis major and latissimus dorsi, can act as dynamic stabilizers.

Static Bony Stabilizers

- *Articular conformity*: The glenoid surface is pear-shaped; the inferior aspect of the glenoid takes the shape of a true circle and the superior aspect of the glenoid is 20% narrower.[3] The sphere-shaped humeral head has 3-fold the surface area

of the glenoid, with only 25% to 30% of the humeral head in contact with the glenoid surface in any one position.[4] This serves to highlight the importance of the soft tissues and muscles surrounding the joint in providing stability during shoulder function. The dimensional relationship between the humeral head and the glenoid can be expressed as the maximum diameter of the glenoid divided by the maximum diameter of the humeral head (glenohumeral ratio) (**Fig. 1**). This ratio is about 0.75 in the sagittal plane and 0.6 in the transverse plane.[5] The area of contact between the humeral head and glenoid moves superiorly with abduction.[5] The balance stability angle is the maximum angle that the humeral joint reaction force can make with the glenoid center line before dislocation results.[6] The effective glenoid arc is the arc of the glenoid (including the increased depth afforded by the labrum) able to support the net humeral joint reaction force. The shape of the underlying bone, cartilage, and labrum all influence the balance stability angle and the effective glenoid arc.[6]

- *Glenoid version*: Mean retroversion of the glenoid (see **Fig. 1**) has been reported as 1.23° (range 9.5° of anteversion to 10.5° of retroversion).[7] Excessive retroversion or anteversion can be associated with reduced stability. Average glenoid inclination has been reported to be 4.2° of superior inclination (range −7° to 15.8°).[7]
- *Coracoacromial arch*: The coracoacromial arch, composed of the coracoid, coracoacromial ligament, and acromion, acts to prevent anterosuperior migration of the humeral head.

Static Soft Tissue Stabilizers

- *Capsuloligamentous structures*: Different portions of the glenohumeral ligament and capsule are responsible for maintaining stability in each anatomic plane; cadaveric[8] and clinical studies[9] have confirmed the varying contributions of

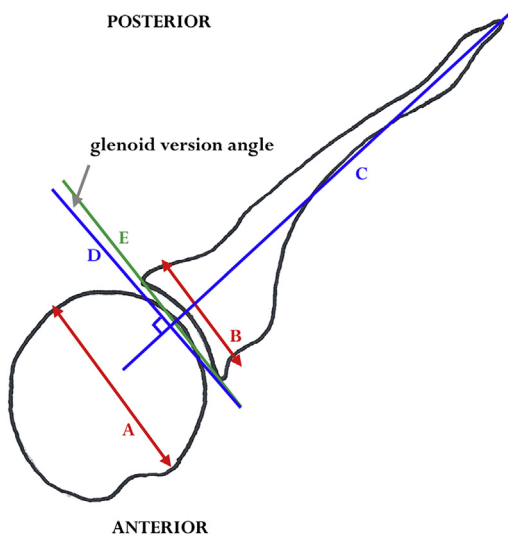

Fig. 1. Static bony stabilizers. The dimensional relationship between the humeral head and the glenoid can be expressed as the maximum diameter of the glenoid (*B*) divided by the maximum diameter of the humeral head (*A*). A method for measuring the glenoid version angle is also shown. Line *C* represents the plane of the body of the scapula and line *E* represents the plane of the osseous glenoid. The angle between line *E* and line *D* (line *D* is drawn perpendicular to line *C*) represents the glenoid version angle.

each structure with different shoulder positions (**Fig. 2**). The complex anatomy of each structure accommodates their role as a primary stabilizer as well as a secondary stabilizer in a position-dependent manner. The anterior and posterior bands of the inferior glenohumeral ligament form a slinglike structure that cradles the humeral head in position. As the shoulder is brought into abduction and external rotation, the anteroinferior aspect of the capsule and the anterior band of the inferior glenohumeral ligament are the most important static constraints to anterior translation of the humeral head. Conversely, with the arm positioned in adduction, flexion, and internal rotation, the posterior band of the inferior glenohumeral ligament and the posterior aspect of the capsule provide the major constraints against posterior instability. With the shoulder in adduction and neutral rotation, the rotator interval complex (comprising the superior glenohumeral ligament, the rotator interval capsule, and the coracohumeral ligament) and the inferior glenohumeral ligament complex resist inferior subluxation.[10] The middle glenohumeral ligament functions to limit both anterior and posterior translation of the arm at 45° of abduction and 45° of external rotation.[11] The functional

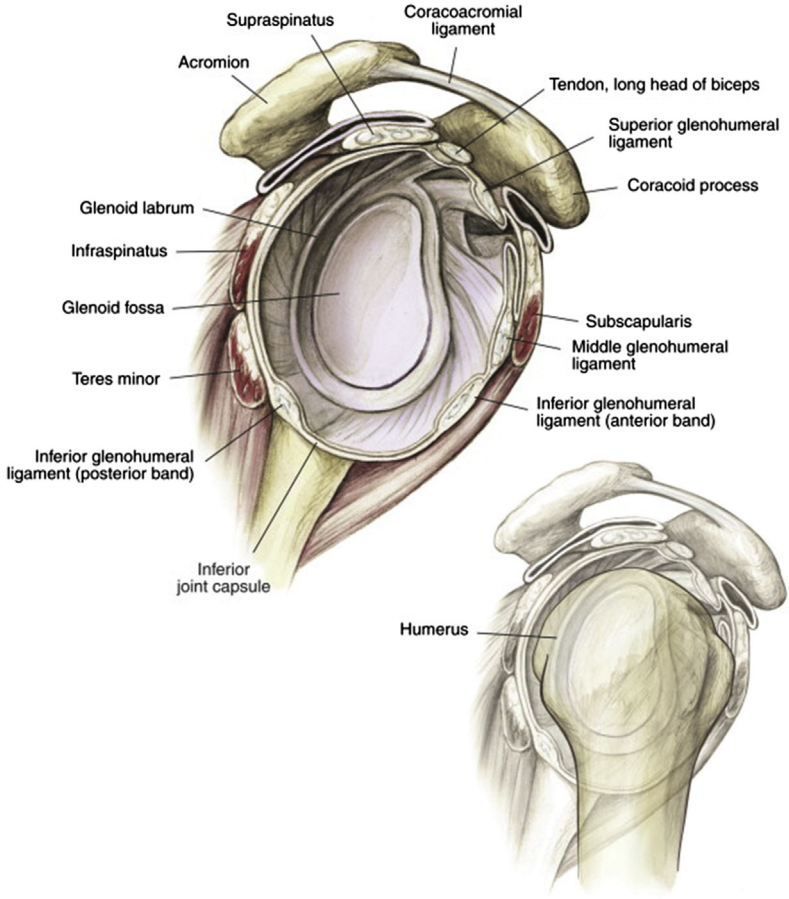

Fig. 2. Static soft tissue stabilizers. (*From* Miller MD. Orthopaedic surgical approaches. Philadelphia: Elsevier Saunders, 2008; with permission.)

interaction between stabilizers on opposite sides of the joint has been described as the "circle concept of ligamentous stability."[12]

- *The glenoid labrum*: The glenoid labrum is a triangular rim of fibrocartilaginous tissue that functions as an extension of the bony glenoid, increasing the depth and surface area of the glenohumeral articulation and contributing around 10% to glenohumeral stability.[13,14] The capsular attachments of the labrum provide further stability with contraction of the rotator cuff. Superiorly, the long head of the biceps tendon shares its origin with labral tissue and the supraglenoid tubercle.
- *The rotator interval:* The rotator interval is a triangular space within the glenohumeral joint capsule that comprises the coracohumeral ligament, the superior and middle glenohumeral ligaments, the long head of the biceps, and a thin layer of capsule. The coracohumeral ligament and associated lateral aspect of the subscapularis are important to maintain stability of the long head of the biceps tendon in the bicipital groove. It is bordered by the coracoid at its base, and the supraspinatis and subscapularis muscles, which converge to an apex laterally. The coracoid separates the subscapularis from the supraspinatus medial to the joint. These muscles insert laterally converging over the intertubercular sulcus.
- *Negative intra-articular pressure:* A degree of stabilization occurs through the vacuumlike effect produced within the closed shoulder compartment and the adhesion-cohesion effect produced by the synovial viscous fluid.[15]

Dynamic Stabilizers

- *Concavity compression:* The rotator cuff, deltoid, and long head of biceps work synergistically to compress the humeral head to the glenoid (concavity compression). The contribution of each depends on the conditioning and strength of the individual structures.[14] The neuromuscular presetting action of the rotator cuff provides the concavity compression required for stability before movement.[16] With their strong influence on shoulder stability, the muscles may contribute not only to stabilization of the shoulder joint but also instability.[17,18]
- *Rotator cuff:* Direct attachments to the capsule allow the rotator cuff muscles to contribute to stability by increasing articular tension; joint compression of the muscular structures provides additional support. In addition, rotator cuff muscles serve to depress the humeral head within the glenoid cavity, and a proprioceptive muscular reflex response counters capsular stretch and shoulder motion detected by sense receptors. Together with the coracoacromial arch, the supraspinatus guards the joint superiorly, the infraspinatus and teres minor stabilize it posteriorly, and the subscapularis protects the shoulder anteriorly.
- *Long head of biceps:* This tendinous structure serves to depress the humeral head while preventing excessive torsion of the glenohumeral joint in rotation with a flexing elbow.
- *Scapular rotators:* Trapezius, rhomboids, latissimus dorsi, serratus anterior, and levator scapulae ensure that the humerus and scapula move synchronously to maintain normal joint articulation throughout the range of motion.

CLASSIFICATION OF SHOULDER INSTABILITY

The cause of shoulder instability is complex and multifactorial, and although several classification systems have been suggested, there is no all-encompassing system that adequately serves as a guide to treatment, predicts outcome, or facilitates communication between clinicians. Instability has been described in terms of direction

(anterior, posterior, inferior, or multidirectional), degree of instability (dislocation, subluxation, or microinstability), chronology (acute, chronic, or acute on chronic), and whether instability is under voluntary control.[19,20]

Two typical groups of individuals who develop glenohumeral instability have been described based on the cardinal features of their condition.[21] The acronym TUBS describes patients with traumatic instability, who characteristically have unidirectional instability (*t*raumatic, *u*nilateral, with a *B*ankart lesion generally requiring *s*urgical treatment). The acronym AMBRI describes instability that is typically *a*traumatic, *m*ultidirectional, and *b*ilateral, responds to *r*ehabilitation, and occasionally requires an *i*nferior capsular shift. These patients classically have underlying ligamentous laxity and develop instability insidiously. It is now recognized that this system is oversimplistic; these 2 groups represent extremes in a spectrum of pathologic conditions, with many patients exhibiting overlapping traits (**Fig. 3**). Many patients with hyperlaxity who report instability without a traumatic precipitant have predominantly unidirectional or bidirectional instability.[2] Similarly, traumatic unidirectional instability occurs bilaterally in a quarter of patients with excessive capsular elastin also noted in many of these patients, implying an element of inherited predisposition.[22]

More recently, the FEDS classification (**Fig. 4**) which is based on the 4 cardinal features of instability used most frequently in existing classifications (*f*requency, *e*tiology, *d*irection, and *s*everity) has been demonstrated to have high intra-observer and inter-observer agreement.[23]

Many patients are able to voluntarily dislocate their shoulder. Although this can occur in those with anterior instability, it typically occurs in patients with posterior instability. Voluntary instability may be associated with an underlying psychological condition and outcomes from surgical treatment are rarely good unless the underlying emotional condition is addressed.[24]

The concept of instability being caused by a combination of structural (traumatic and atraumatic) and neurologic system disturbances has led to a classification of instability as a continuum of pathologies that can be displayed graphically as a triangle (the Stanmore classification) (**Fig. 5**).[25] The polar pathologies are labeled type I (traumatic instability), type II (atraumatic instability), and type III (neurologic dysfunctional or muscle patterning). Polar groups I and II and the axis I-II representing the spectrum

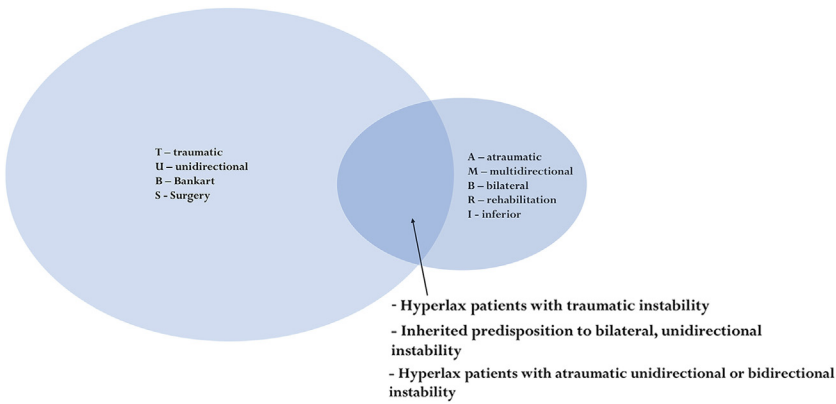

Fig. 3. The traditional dichotomy between TUBS and AMBRI patients is now recognized as being oversimplistic; many patients exhibit overlapping traits. Examples include hyperlax patients with traumatic unidirectional instability and ligamentously lax patients with traumatic injuries.

FREQUENCY–The patient is asked, *"How many episodes have you had in the last year?"*

Solitary – *'1 Episode'*
Occasional- *'2 -5 Episodes'*
Frequent – *'>5 Episodes'*

ETIOLOGY – The patient is asked, *'Did you have an injury to cause this?'*

Traumatic – *' Yes'*
Atraumatic – *' No'*

DIRECTION – The patient is asked, *' What direction does the shoulder go out most of the time?'*

Anterior- *'Out the Front'*
Inferior- *' Out the Bottom'*
Posterior- *'Out the Back'*

The direction is confirmed at the time of the physical examination using provocative tests. During translation testing, the physician asks, which one of the following directions most closely reproduces your symptoms, and then translates anterior, inferior, and posterior. To confirm, the physician may ask which one of these tests most closely reproduces your symptoms: and the anterior apprehension test, the sulcus test, and the posterior jerk test is performed. With the history and physical examination using provocative tests, the patient should be able to distinguish and identify the *primary direction* of his or her instability.

SEVERITY–The patient is asked,*'Have you ever needed help getting the shoulder back in joint?'*

Subluxation– *'No'*
Dislocation – *'Yes'*

Fig. 4. The FEDS classification of shoulder instability. (*From* Kuhn JE. A new classification system for shoulder instability. Br J Sports Med 2010;44(5):341–6; with permission.)

of injuries between them correspond to the TUBS-AMBRI classification. The direction of instability is not considered in the Stanmore classification, with the investigators arguing that it is less relevant to effective management whether the instability is structural, nonstructural, or both.[26]

Without an all-encompassing classification, we advocate an individual or algorithmic approach to diagnosis where the clinical manifestation of instability (chronicity, direction, presence of trauma, degree of instability, and volitional control) is combined with the underlying pathologic attributes on an injury (considering soft tissue and bony injuries).[1] In considering the most appropriate treatment, these features should be

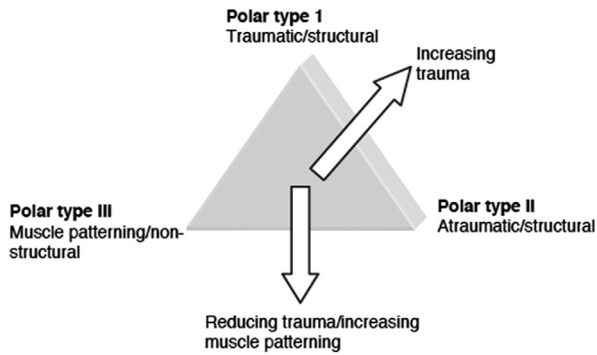

Fig. 5. Stanmore classification of shoulder instability. (*From* Jaggi A, Lambert S. Rehabilitation for shoulder instability. Br J Sports Med 2010;44(5):333–40; with permission.)

combined with a consideration of the athletic needs of an individual and the timing within the athletic calendar.

PATHOGENESIS OF INSTABILITY IN THE ATHLETE

Three broad etiologic categories have been implicated in instability of the shoulder: repetitive microtrauma to the shoulder, acute traumatic events, and purely atraumatic causes. It is crucial to identify the correct pathogenesis of instability so that treatment can be appropriately tailored to the patient's needs.

PATHOANATOMY OF TRAUMATIC ANTERIOR INSTABILITY

Traumatic anterior shoulder instability in the athlete usually occurs with a posteriorly directed force applied to the anterior aspect of an abducted, externally rotated arm. A direct blow (from posterior) can also cause a traumatic anterior dislocation. The humeral head is driven forward, producing a spectrum of soft tissue and bony lesions that are implicated in the pathogenesis of recurrent instability.

Soft Tissue Lesions

Bankart lesion

The Bankart lesion, with detachment of the anteroinferior labrum with its attached inferior glenohumeral ligament complex (IGHLC), was traditionally described as the "essential lesion" of anterior traumatic dislocation of the shoulder occurring in 90% of cases of anterior instability (**Fig. 6**). Although the Bankart lesion is almost always present in patients with traumatic instability,[27,28] it does not produce instability in isolation. The underlying cause is multifactorial, with plastic deformation of the IGHLC regarded central to development of recurrent anterior instability.[29]

Plastic deformation of the IGHLC

Plastic deformation of the IGHLC occurs during the initial dislocation before detachment of the labrum and becomes progressively more severe with recurrent episodes of instability.[30] In a cadaveric study, IGHLC specimens were loaded in tension to failure and 3 modes of failure were observed: at the site of the glenoid insertion (40%), in the mid-substance of the ligament (35%), and at the site of the humeral insertion (25%). Even when failure occurred at the site of the glenoid insertion, it occurred

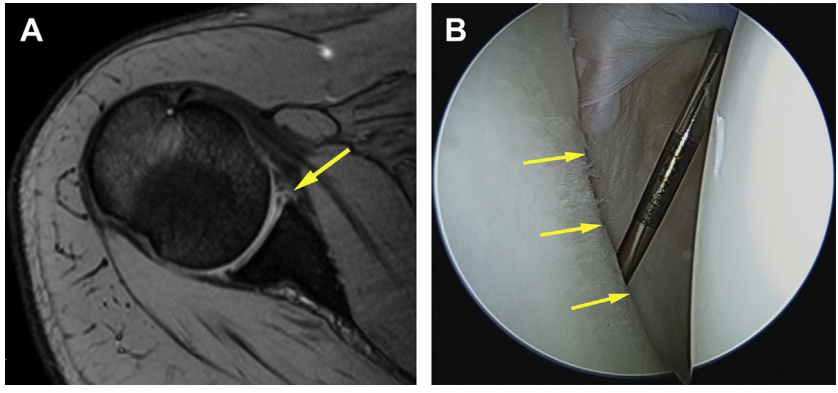

Fig. 6. Magnetic resonance image showing disruption of the anteroinferior glenoid labrum (*arrow, A*). Arthroscopic image confirming discontinuity of the glenoid surface and redundant anterior labrum (*arrows, B*).

only after significant elongation of the IGHLC.[31] Clinical studies have reinforced that capsular stretching can occur simultaneously with a Bankart lesion during an anterior dislocation, with an abnormal capsular redundancy reported in up to 28% of patients with recurrent anterior instability.[32]

Variations in anterior capsulolabral pathoanatomy

Various other patterns of capsulolabral injury have been described.

- Superior labrum anterior and posterior detachment (SLAP) (**Fig. 7**). This may occur in continuity with Bankart lesions and defects of the rotator interval. It is more common in throwing athletes perhaps because of the eccentric loads on the biceps anchor during the deceleration phase of throwing.[33,34] A cadaveric study demonstrated a significant effect on anteroposterior and superoinferior translation with complete lesions.[35]
- Humeral avulsion of the glenohumeral ligaments (HAGL) (**Fig. 8**). This lesion is typically recognized after first-time dislocations and probably represents a variant from the normal pattern of anterior capsular stretching or rupture.[36] It may occur in isolation or in conjunction with a Bankart lesion.
- Anterior labroligamentous periosteal sleeve avulsion (ALPSA) (**Fig. 9**). This is similar to the Bankart lesion, however, the anterior scapular periosteum does not rupture and the IGHLC, labrum, and periosteum are stripped and displaced in a sleeve-type fashion medial on the glenoid neck.[37]

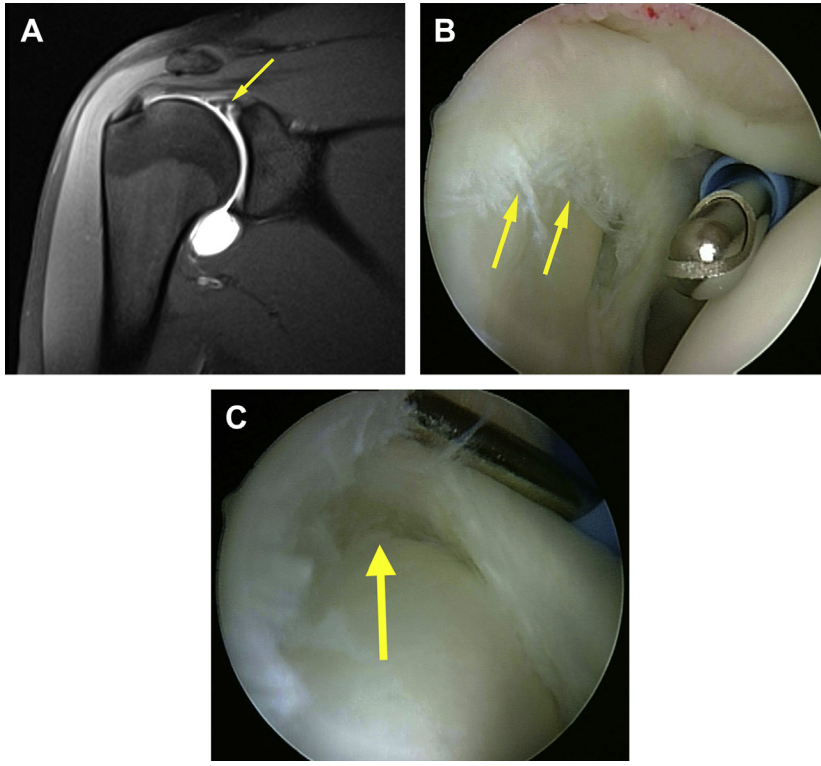

Fig. 7. Coronal magnetic resonance image demonstrating disruption of the superior labrum in keeping with an SLAP tear (*arrow, A*). On arthroscopic examination, the superior labrum can be seen to be detached from its attachment to the superior glenoid (*arrows, B* and *C*).

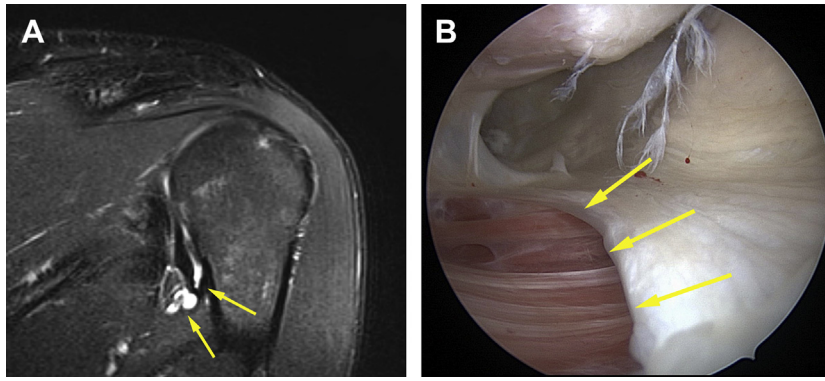

Fig. 8. Magnetic resonance image showing avulsion of the glenohumeral ligaments on from their proximal humeral insertion (HAGL lesion) (*arrows, A*). Arthroscopic appearance of an HAGL lesion (*arrows, B*).

Defects of the rotator interval

There is wide physiologic variability in the rotator interval capsule between individuals, from a small opening to a wide gap. It is therefore difficult to define what is abnormal and contributing to pathologic instability. Although open closure of isolated rotator interval defects has been described, it is not uncommon for patients to develop recurrent instability into a large, inferior pouch after this type of limited surgical repair.[38,39] There may be a role for arthroscopic rotator interval closure as an adjunct to other techniques to reduce failure rates.[40]

Disruption of neuromuscular control

Neuromuscular control plays an important role in the regulation of shoulder stability.[41] One study showed that proprioception of the affected shoulder was altered in patients with glenohumeral instability compared with the asymptomatic extremity.[42] This was eliminated after shoulder reconstruction, suggesting that surgery restores some of the proprioceptive characteristics. An arthroscopic study demonstrated a direct afferent neurologic pathway between the proprioceptive receptors in the joint capsule and

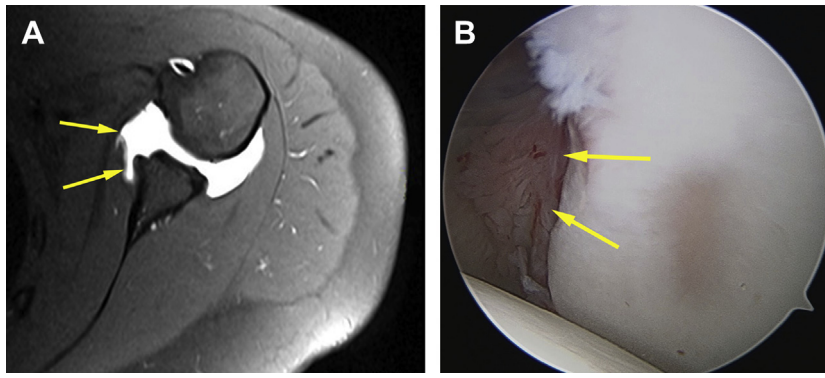

Fig. 9. Magnetic resonance (*A*) and arthroscopic (*B*) appearance of an ALPSA lesion. In this lesion the IGHLC, labrum and periosteum are stripped and displaced in a sleeve-type fashion medial on the glenoid neck (*arrows*).

the cerebral cortex.[43] More research is necessary to further our understanding of the role of proprioception in the unstable shoulder.

Bony Lesions

Bony Bankart lesion

As the humeral head dislocates anteriorly, it may produce a bony Bankart lesion (**Fig. 10**) a compression fracture, or wear of the glenoid rim (**Fig. 11**). Anterior glenoid defects result in loss of glenoid concavity and compromise the static shoulder restraints predisposing to instability.[44] Biomechanical studies demonstrate an inverse relationship between the size of the glenoid defect and joint stability.[45] Cadaveric studies report that glenoid lesions measuring more than half of the glenoid length reduced dislocation resistance by more than 30%; defects wider than 20% glenoid length predispose to recurrence despite Bankart repair.[19,45]

Hill-Sachs impression fracture

When the humeral head impacts on the anterior glenoid, a Hill-Sachs impression fracture is created on the posterolateral aspect of the humeral head. Most lesions are small to moderate in size and do not influence shoulder stability.[1] Hill-Sachs lesions can be classified according to their size as mild (2 cm $\times$ 0.3 cm), moderately severe (4 cm $\times$ 0.5 cm), and severe (4 cm $\times$ 1 cm or greater).[46] A Hill-Sachs lesion that extends into the zone of contact between the humeral head and glenoid during abduction, external rotation, and horizontal extension (glenoid track) has a risk of engagement, resulting in glenohumeral dislocation.[47] Because of the contribution of Hill-Sachs lesions to recurrent instability, the defect should be addressed in patients with severe defects or defects larger than 60% of the humeral head radius, and in those who engage in 90° of abduction and 90° of external rotation (the 90/90 position).[48,49]

PATHOANATOMY OF TRAUMATIC POSTERIOR INSTABILITY

The most frequent cause of recurrent posterior shoulder instability in the athlete is repetitive microtrauma to the posterior shoulder complex. In contrast to anterior instability, acute dislocation is usually not the most common initial presentation of posterior instability.[50] A spectrum of soft tissue and bony pathologies is encountered, the nature of which depends on the cause of the instability.

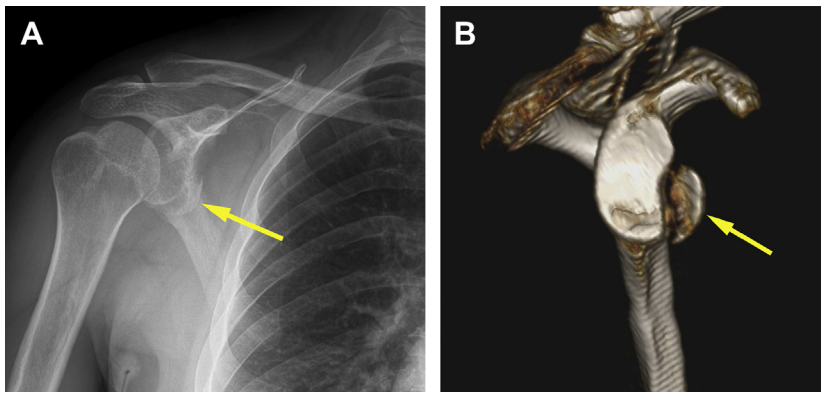

Fig. 10. Plain radiograph (*A*) and magnetic resonance image (*B*) illustrating an acute bony Bankart lesion with a large visible bony fragment (*arrows*).

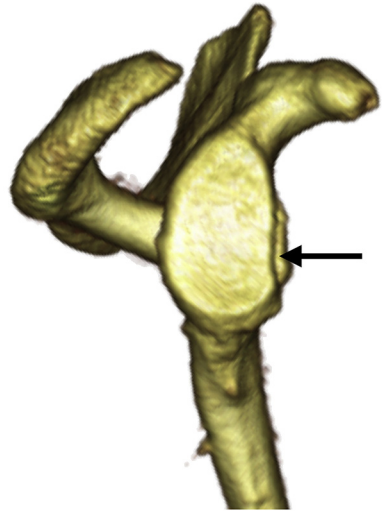

Fig. 11. Three-dimensional computed tomography reconstruction demonstrating anteroinferior glenoid bone loss (*arrow*).

Soft Tissue Lesions

Repetitive bench press lifting, overhead weight lifting, rowing, swimming, or other activities that involve repetitive loading of the shoulder in front of the body can be sources of repetitive microtrauma.[50] In these activities, the shoulder is repetitively placed in a flexed and internally rotated position. The resulting posterior load causes lesions of the posterior labrum, frequently accompanied by stretching of the posteroinferior aspect of the capsule.[51–53]

Reverse Bankart lesion

Tears of the posteroinferior aspect of the capsulolabral complex (reverse Bankart lesion) involving the posterior band of the inferior glenohumeral ligament are more common when there has been a discrete injury to the shoulder (**Fig. 12**).[54–57] They may be degenerative in origin, caused by recurrent episodes of instability.[52,58,59]

Plastic deformation of the capsule

With recurrent subluxation, the capsule undergoes plastic deformation, producing a patulous posteroinferior capsular pouch and increased joint volume. This excessive capsular laxity and large capsular recess can be a cause of posterior instability.[52,57,60]

Variations in posterior capsulolabral pathoanatomy

Several variations from the typical pattern of capsulolabral pathoanatomy have been described.

- Kim lesion. This incomplete and concealed avulsion of the posteroinferior aspect of the labrum may be associated with unidirectional or posteroinferior instability.[61]
- Posterior labrocapsular periosteal sleeve avulsion (POLPSA). In this lesion, the posterior labrum and the intact posterior scapular periosteum are stripped from the glenoid, producing a redundant recess that communicates with the joint space.[62]

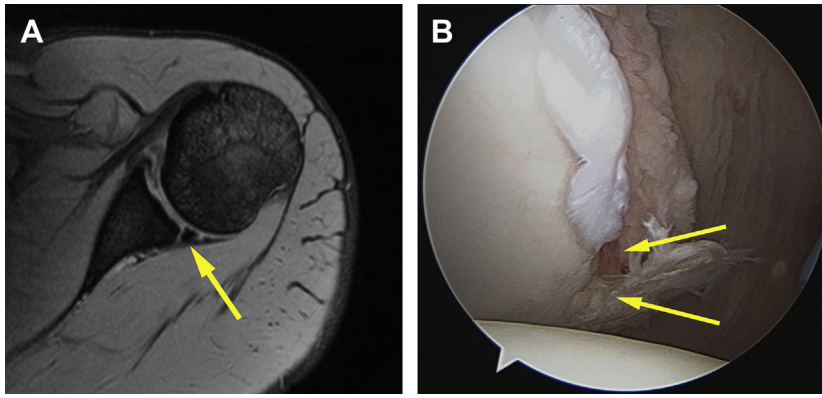

Fig. 12. Magnetic resonance image showing disruption of the posteroinferior glenoid labrum (*arrow, A*). Arthroscopic image confirming discontinuity of the glenoid surface and the posterior labrum (*arrows, B*).

- Bennett lesion. This is an extra-articular curvilinear calcification along the posteroinferior glenoid near the attachment of the posterior band of the inferior glenohumeral ligament.[63] It has been hypothesized that POLPSA may represent the acute stage of a Bennett lesion.[62]
- Posterior humeral avulsion of the glenohumeral ligament. This lesion represents an avulsion of the posterior band of the inferior glenohumeral ligament from its attachment on the humerus.[64]

Bony Lesions

Instability may theoretically occur through increased glenoid retroversion, posterior glenoid erosion, engaging anterior humeral head defects, localized posterioinferior glenoid hypoplasia, or increased humeral head retroversion. No clear association has been demonstrated between posterior instability and the latter 2 conditions.[53]

Glenoid retroversion

Glenoid version varies widely in the normal population and is often documented in patients with instability.[7,65] Studies of the degree of association between glenoid retroversion and posterior instability have produced conflicting results.[66–68] It is probable that excessive glenoid retroversion is rarely a primary cause of instability but should be considered a contributory factor.[53]

Posterior glenoid bone defects

Damaged posterior stabilizers may present as posterior rim fractures (reverse bony Bankart lesions) after an acute traumatic dislocation or erosions as a result of localized glenoid hypoplasia or repeated subluxations (**Fig. 13**).[52,69,70] There is a relationship between the extent of glenoid erosion seen on computed tomography and recurrent instability.[71]

Reverse Hill-Sachs impression fracture

Posterior dislocation often results in an osteochondral fracture of the anterior humeral head medial to the lesser tuberosity, in the region of the anatomic neck from impaction on the posterior glenoid rim (a reverse Hill-Sachs lesion). This may extend into the zone of contact between the humeral head and the glenoid during flexion, adduction, and internal rotation, producing subsequent engagement and subjective instability or dislocation.[72]

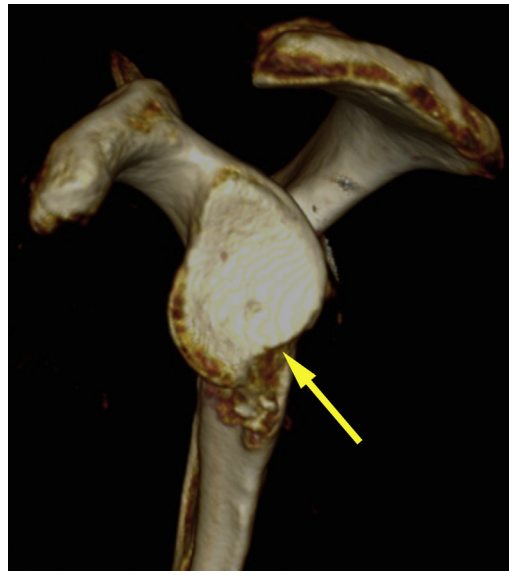

Fig. 13. Computed tomography reconstruction demonstrating posteroinferior glenoid bone loss (*arrow*).

PATHOANATOMY OF ATRAUMATIC INSTABILITY

It is challenging to define atraumatic instability because activities of daily living and improper shoulder mechanics may lead to damage at a molecular level. Many athletes with evidence of constitutional ligamentous laxity develop unilateral instability only after a discrete injury; whilst some degree of inherited predisposition to traumatic instability is implied by its occurrence bilaterally in a quarter of patients.[73] Atraumatic instability includes the diagnosis of multidirectional instability (MDI). Studies have shown an excessive elastin component in capsular tissue and skin of patients with MDI.[74] Patients with MDI tend to have a generalized increase in joint volume with posterior, inferior, and anterior capsular redundancy.[2] Bony abnormalities are not generally present, however pathologic findings may be present when a traumatic dislocation is superimposed in the setting of MDI. Muscle imbalance, especially rotator cuff weakness can lead to dependency on the capsule as the primary restraint to translational forces, which may ultimately result in fatigue failure beyond the visco-elastic material properties of the capsule. This ultimately leads to capsular stretching that may progress to symptoms of instability.[75]

SUMMARY

Glenohumeral joint motion results from a complex interplay between static and dynamic stabilizers that require intricate balance and synchronicity. Instability of the shoulder is a commonly encountered problem in active populations, especially young athletes. The underlying pathoanatomy predisposing to further episodes and the needs of individual athletes must be considered in determining the most appropriate treatment.

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