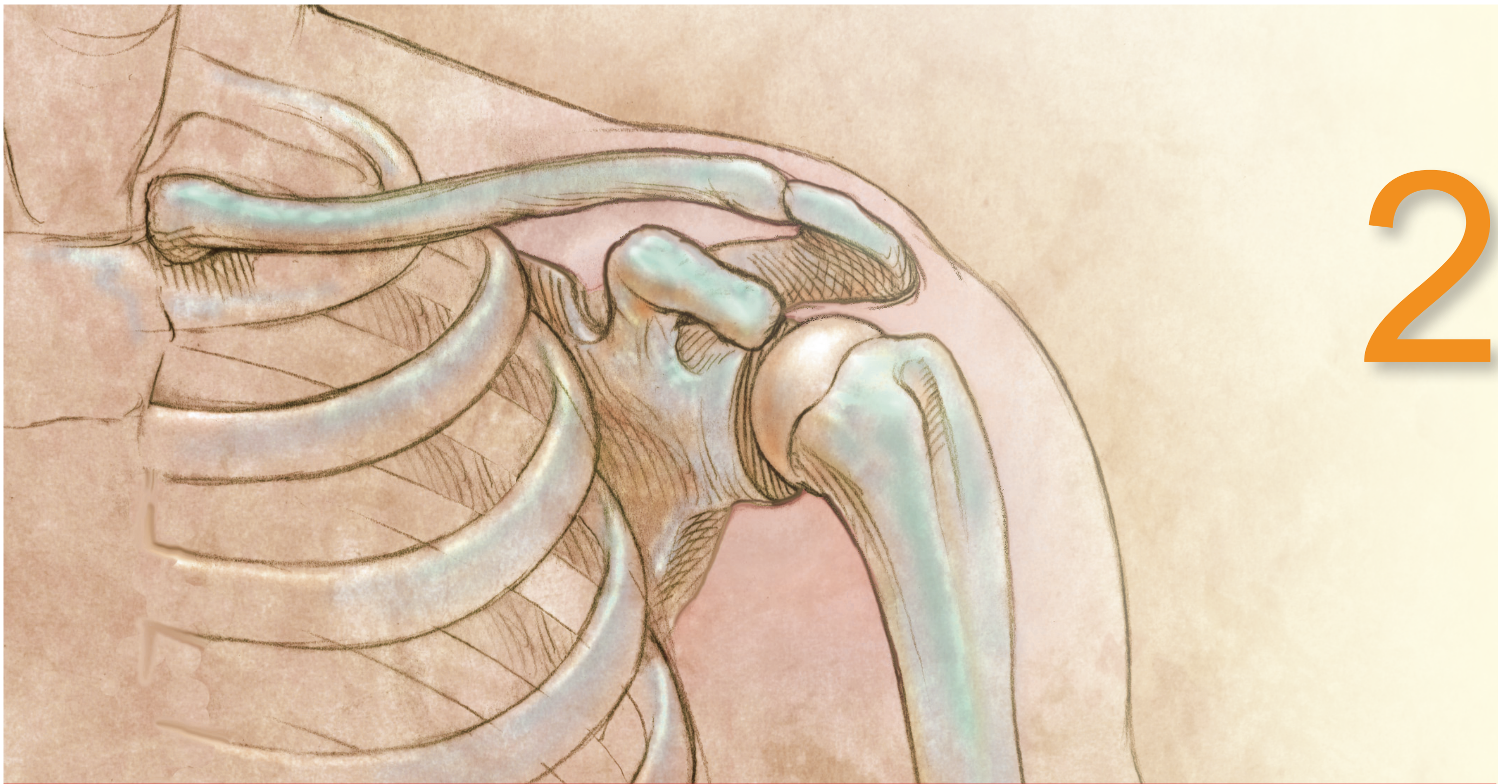

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Pathophysiology and Biomechanics of Glenohumeral Instability

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[AQ1]

Introduction

Hippocrates was the first to investigate the pathophysiology of the unstable shoulder (anterior instability and treatment) over 1,000 years ago (1). Early investigations attributed instability to traumatic events or congenital shoulder abnormalities (2). The capsuloligamentous complex of the glenohumeral joint was first described in 1829 as consisting of the superior, middle, and inferior glenohumeral ligaments (3). Subsequent studies in the early 1900s by Perthes (4) and Thomas (5) suggested that the capsule and glenohumeral ligaments play a role in shoulder stability. In 1923, Bankart (6,7) described the detachment of the anterior inferior capsule from the glenoid as the "essential" lesion in anterior glenohumeral instability. The modern-day term "Bankart lesion" is used to describe an avulsion of the anteroinferior glenoid labrum from its attachment to the inferior glenohumeral ligament complex (IGHLC). Subsequently, Turkel et al. (8) performed the classic biomechanical cadaver study to describe the contribution of the superior, middle, and inferior glenohumeral ligaments to shoulder stability at various degrees of shoulder abduction. Subsequently, Neer hypothesized that repetitive microtrauma to the shoulder capsule, as in the case of a high-demand overhead athlete, could also lead to overstretching and contribute to shoulder instability (9). Numerous biomechanical and clinical studies in the last decade have evaluated different contributing factors to shoulder instability (10–16).

The shoulder (glenohumeral joint) is minimally constrained and designed for mobility that allows for a tremendous range of motion in multiple anatomic planes to maximize function. However, this anatomic arrangement while allowing necessary motion for sports and overhead activities, places the glenohumeral joint at risk for instability. Translation of the humeral head in relation to the glenoid during activities of daily living or athletics is prevented by both the static and dynamic stabilizing mechanisms. Important static stabilizers include the articular anatomy of a joint with matched concavity and convexity of the ball-in-socket, as well as the glenoid labrum which broadens and deepens the socket depth. The vacuum seal of the closed joint capsule results in negative intra-articular pressure which may enhance the stabilizing effect of the capsular ligamentous structures. Dynamic stabilizers include the rotator cuff musculature, biceps tendon, scapulothoracic and humeral motions, and the deltoid muscle. The balance between the static and dynamic stabilizers determines the stability of the shoulder joint. An imbalance among

▲ FIGURE 2-1: A patient with clinical laxity increased range of motion in both external and internal rotations but without any symptoms of instability. External rotation at neutral measured 95 degrees **(A)**, internal rotation to T5 **(B)**, external rotation and internal rotation with the arm abducted at 90 degrees measured 100 degrees **(C)**, and 90 degrees **(D)**, respectively.

these stabilizing factors may result in instability occurring in the anterior, posterior, inferior directions or it may be multidirectional in nature (17–19). There is a spectrum of instability ranging from transient subluxation, dislocation that is self-reduced to locked dislocation requiring general anesthesia and muscle relaxation for reduction. In addition to the above dynamic and static factors, proprioception also plays a significant role in the pathoetiology of shoulder instability (20). Proprioception is the perception of motion of the joint and it is an important mechanism by which the muscles receive a message to contract and guard against instability. A failure of proprioceptive feedback may contribute to instability.

Finally, it is essential to distinguish between "laxity" and "instability." Some patients may be lax without actual instability (Fig. 2-1) and some individuals may be tight with episodes of instability. Laxity is the looseness of a joint necessary for normal shoulder motion and is often painless. It is variable between individuals. Instability is a sudden displacement of the humeral head out of the socket during shoulder motion and it is a pathologic event that is typically painful. Clinical manifestations of instability are the result of a combination of factors with both static and dynamic failure of stabilizing structures; therefore, it is essential to understand normal biomechanics if a surgeon is to formulate a logical approach to treatment on an individual patient basis. This chapter will clarify the static and dynamic contributions to stability of the shoulder joint complex and thus provide a framework for understanding surgical solutions for instability of the shoulder.

Spectrum and Direction of Instability

Instability and laxity are two separate terms describing two different entities of glenohumeral translation. Laxity is defined as asymptomatic translation of the humeral head on the glenoid surface that is seen in normal shoulders and it is a requirement for normal joint motion; however, the amount of translation may differ between individuals secondary to the status of the soft tissue about the shoulder. Instability is a clinical diagnosis manifested as excessive translation of the humeral head on the glenoid surface during active shoulder rotation or motion that is associated with symptoms, usually pain or apprehension (21–25).

A spectrum of instability also exists ranging from subluxation to complete dislocation requiring sedation and muscle relaxation for successful reduction. Subluxation is defined as symptomatic translation of the humeral head out of the glenoid socket but not to the point of actual dislocation. A dislocation is complete separation of the articular surfaces, which can also range from spontaneous self-reduction termed "transient luxation" to a fixed dislocation requiring sedation and muscle relaxation for reduction of the humeral head back into the glenoid (26). Owens et al. (26) defined "transient luxation" as complete dislocation that spontaneously self reduces. Most patients who experienced a "transient luxation" will present with both a Bankart lesion and a Hill-Sachs lesion on MRI.

The primary direction of instability can be anterior, posterior, inferior, or multidirectional. Often, patients presenting with anterior instability will also have associated injury to the posterior capsule and may have excessive posterior translation when examined under anesthesia. This phenomenon is explained by the "circle concept" and by the "load and shearing concept" of glenohumeral instability (Fig. 2-2) (27). Multidirectional instability (MDI) means symptomatic instability in more than one direction (28). Typically this produces symptoms in an anterior direction in combination with the inferior direction. Less commonly, it may be posteriorly associated with inferior direction of instability. While patients with MDI are rare and may present with a Bankart lesion and a Hill-Sachs lesion, the hallmark of this condition is a redundant inferior axillary pouch and deficient rotator interval (28–30).

History and physical examination help indicate the direction of instability and examination under anesthesia confirms the diagnosis. Typically, apprehension is elicited in the abduction–external rotation position in patients with anterior instability and in the adduction–internal rotation in patients with posterior instability. A significant symptomatic inferior translation on clinical examination in addition to either anterior or posterior symptoms is the hallmark of MDI (31–36). Biomechanically, Warner et al. (22) proved the primary restraint to inferior translation of

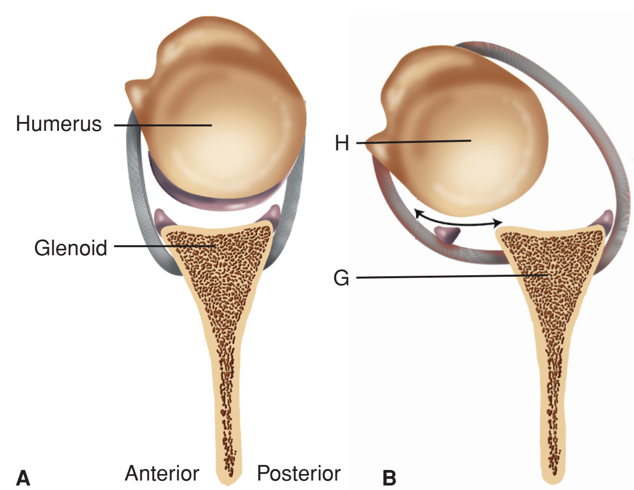

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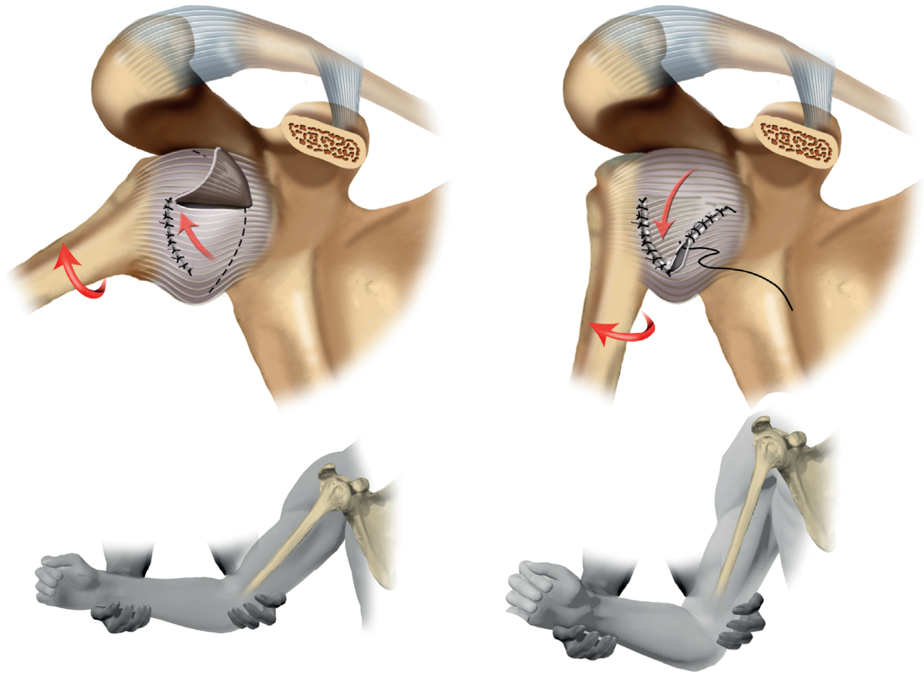
Figure 2-2: A: Normal glenohumeral joint with capsulolabral complex. **B:** Anterior dislocation of the humeral head (H) in relations to the glenoid (G) results in injury to both sides of the joint and capsulolabral complex.



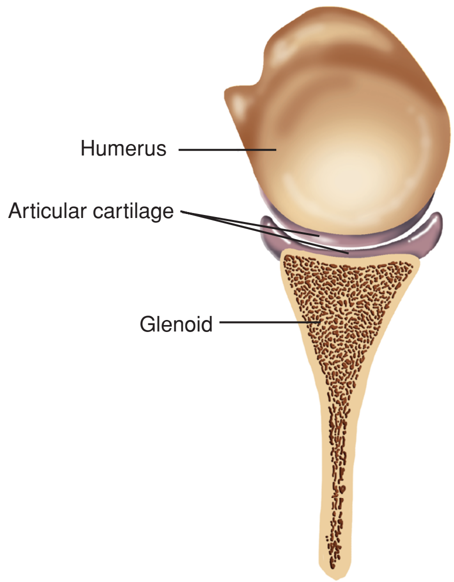
◀ FIGURE 2-3: Selective capsular shift in patients with both a Bankart lesion and inferior instability. The Bankart is repaired anatomically and the capsule is tightened with the arm in 30 degrees of abduction and 30 degrees of external rotation to prevent loss in range of motion.

the adducted shoulder is the superior glenohumeral ligament (SGHL). With progressive abduction of the arm, the anterior and posterior glenohumeral ligaments become the main static stabilizers in resisting inferior translation. Anterior portion was the primary restraint with the arm in 45 degrees of abduction and the posterior portion was the primary restraint with the arm in 90 degrees of abduction. Furthermore, Warner et al. (22) also showed that venting of the shoulder capsule resulted in significant inferior translation of the humeral head. Thus the so-called "sulcus sign" is the result of intra-articular vacuum effect and capsular laxity. In patients who present with both Bankart lesion and laxity, selective capsular repair with the arm in the 30 degrees of abduction and 30 degrees of external rotation (Fig. 2-3) have been recommended in the literature (37). Gerber et al. (38) have also demonstrated that total anterior capsular plication and posterior plication will significantly limit external rotation (>30 degrees) and internal rotation (>30 degrees), respectively. Inferior plication will limit shoulder range of motion in abduction, flexion, and rotation. Therefore, it is essential to diagnose the direction of instability or laxity and select the optimal location for capsular plication in order to maximize patient outcome.

STATIC STABILIZERS

Articular Geometry and Concavity

The glenohumeral joint comprises a large spherical humeral head that articulates with the smaller glenoid surface. Historically, the articular geometry was believed to contribute minimally to the overall stability of the glenohumeral joint. This conclusion was drawn from two observations. The first is the small area of the glenoid surface relative to the large humeral head; and the second is the relative mismatch of the bony curvature of the glenoid to the humeral head (39,40). The shape of the glenoid is smaller superiorly and larger inferiorly, much like a "pear." Average vertical and transverse dimensions are 35 and 25 mm, respectively, whereas the vertical and transverse humeral head articular surface average 48 and 45 mm, respectively (41). Thus, the above measurements produce a significant surface area and radius of curvature mismatch between the joint surfaces of the glenoid and the humeral head. Furthermore, unlike the hip joint, the glenoid does not enclose the humeral head and only up to 25% to 30% of the humeral head is in contact with the glenoid at various shoulder range of motion (42,43). Although the subchondral bone on the glenoid side is flatter than the humeral head, recent studies have demonstrated that the articular surface of the glenoid is actually highly congruent to the articular surface of the humeral head. Kelkar et al. (44) reported the average radii of curvature of the humeral head and glenoid articular surfaces were 25.5 ± 1.5 mm and 27.2 ± 1.6 mm, respectively. The articular surface of the glenoid is thin in the central bare area (1.2 mm average) and thick at the periphery (3.8 mm average). In contrast, the cartilage on the humeral head is thin in the peripheral region (0.6 mm average) and thick in the central region (2 mm average) (17,42,43). Thus, the mismatch in the articular cartilage in the glenoid and humeral head increases the conformity of the overall glenohumeral joint to within



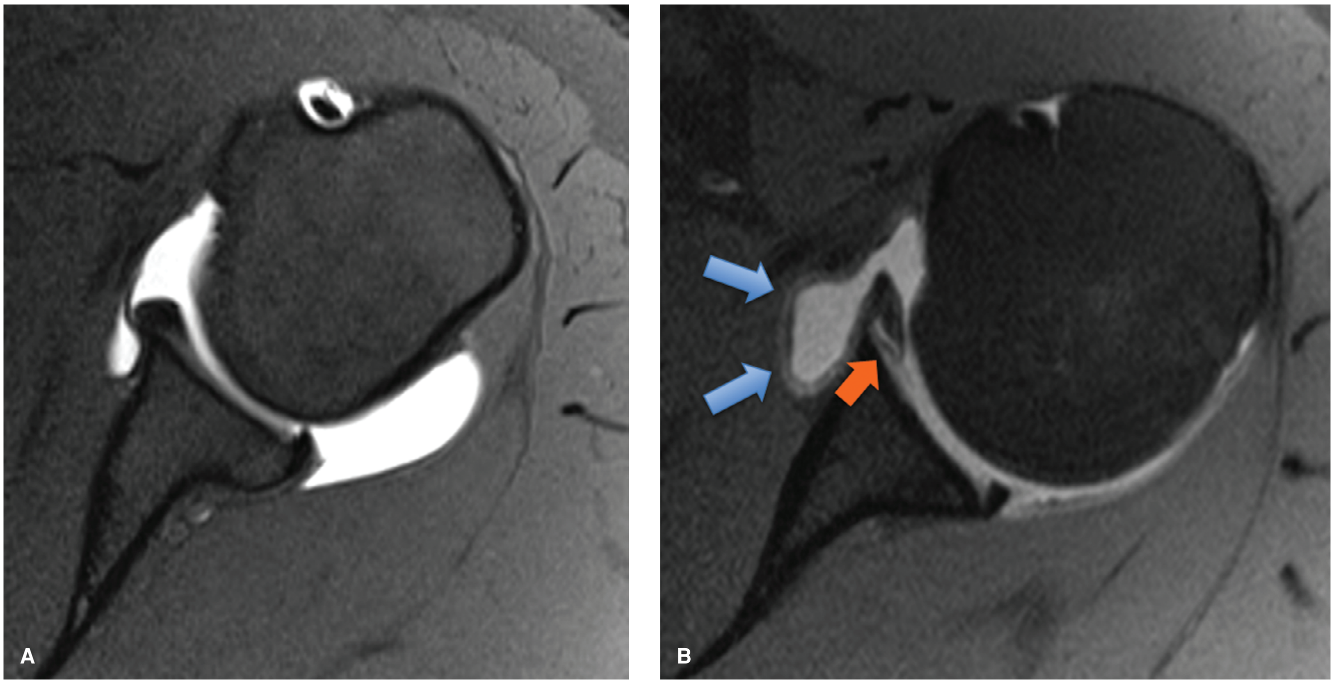
▲ **FIGURE 2-4:** Illustration demonstrating the conformity of the articular cartilage surfaces of the glenoid (G) with cartilage of the humeral head (H) despite the relative flat osseous glenoid.

3 mm (Fig. 2-4). Furthermore, the glenoid concavity is deepened by the labrum that is attached circumferentially around the glenoid on the outer rim (45). Biomechanical studies have demonstrated that joint conformity contributes more in controlling translations during active motions, whereas capsular constraints become more important during passive motions (46).

In terms of humeral version, there is minimal evidence that abnormal version will contribute significantly to glenohumeral instability (2).

Glenoid Labrum

The labrum is a fibrocartilaginous bumper that forms a circumferential ring around the glenoid and serves as an anchoring point for the capsuloligamentous structures. Attachment to the articular cartilage occurs via a narrow fibrocartilaginous transition zone but it is otherwise fibrous throughout the entire structure (45). It is loosely attached superiorly above the equator and significant anatomic variability exists in this particular region between individuals (47). In contrast, the anterior inferior labrum is intimately attached to the glenoid rim and any detachment would indicate an abnormality (Fig. 2-5) (19). Vascular supply occurs in the peripheral attachment to the joint capsule (47). The essential contribution of the labrum to glenohumeral stability is by deepening the anterior to posterior depth of the glenoid socket from 2.5 to 5 mm and increasing the glenoid concavity to 9 mm in the superior to inferior plane. A loss of the labrum will decrease the overall depth of the socket by up to 50% in all directions (48). The stabilizing effect of the labrum is similar to a "chock block" that is used to prevent a wheel on a car or plane from rolling downhill (48). Furthermore, the glenoid labrum also increases the surface area for humeral head articulation and increases the excursion distance required for glenohumeral instability (49,50).



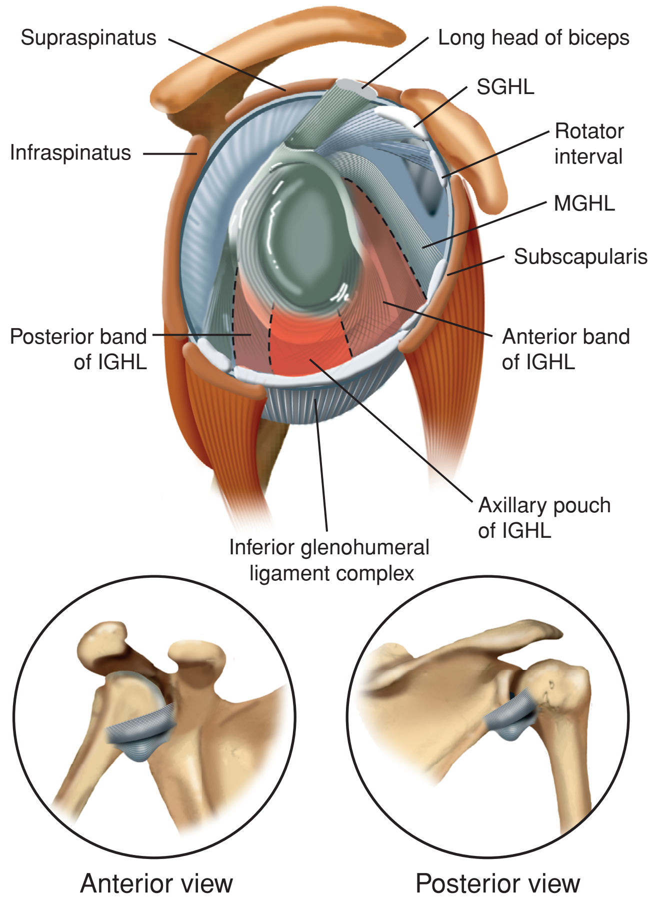
[AQ6] ▲ **FIGURE 2-5:** Axial T2-weighted MRI images of a normal patient **(A)** and a patient with a Bankart lesion (*orange arrow*) and associated anterior capsule stretch injury (*blue arrows*) secondary to the dislocation event **(B)**.

Biomechanical studies have shown that the concavity-compression effect of the labrum is the most effective stabilizing mechanism in resisting tangential forces. With the labrum intact, the humeral head will resist tangential forces of up to 60% of the compressive load. The degree of compression stabilization also varied according to the circumferential location of the glenoid, where the greatest magnitude was observed both superiorly and inferiorly. This effect may be attributed to the greater glenoid labrum depths in those two particular areas (49). The average contribution of the labrum to glenohumeral stability through the concavity compression is around 10%. This contribution also varies according to both arm position and direction of force with increased stability seen in the adducted position and inferior direction, respectively (51,52). Rodosky et al. (53) showed that with detachment of the superior glenoid labrum, resistance to torsion is decreased and more strain is placed on the inferior glenohumeral ligament (IGHL) which can contribute to dynamic anterior instability.

Another theory on the stabilizing effect of the labrum is its contribution to the intra-articular negative pressure of the shoulder. Habermeyer et al. (54) have compared the glenohumeral joint to a piston surrounded by a valve. The labrum works as a valve block that seals the joint from atmospheric pressure. Traction of the arm in a stable shoulder with intact labrum resulted in negative pressure that correlated to the amount of forces exerted. In contrast, in the unstable shoulder with detachment of the anterior inferior labrum, the above phenomenon does not exist, thus the piston and valve model is not valid. Thus the authors concluded that the absence of negative joint pressure disturbs joint mechanics and also the receptors that control motor feedback to protect the shoulder dynamically from dislocating forces. However, in contrast to the above study, restoring the "bumper" effect after Bankart repair to recreate the glenoid labrum has not been shown in a cadaver model to increase glenohumeral translational stability when compared to fixation at the glenoid rim (55).

Capsule and Glenohumeral Ligaments

The shoulder capsule has about twice the surface area of the humeral head and allows for shoulder range of motion (17). The anterior capsule is thicker than the posterior capsule. Ciccone et al. (56) found that the anterior shoulder capsule averaged 2.42 mm, inferior capsule averaged 2.8 mm, and posterior capsule averaged at 2.2 mm thick. The range in the study was 1.32 to 4.47 mm and with significant thinning laterally from the glenoid to the humerus. These distinct thickenings in the anterior capsule are called glenohumeral ligaments and play an important role in shoulder stability. Early cadaver studies have evaluated the role and function of these ligaments, which comprises SGHL, middle glenohumeral ligament (MGHL), and IGHL that is further separated into anterior and posterior components (Fig. 2-6). With rotation of the arm, specific ligaments tighten while others loosen. In the midranges of motion (everyday activities), the capsule and glenohumeral ligaments are in a lax state; therefore, does not contribute significantly to shoulder stability. However, at the extremes of range of motion, different glenohumeral ligaments will tighten according to the specific position of the arm and control humeral head translation to provide stability (17,19). The following subsections will discuss the contributions of each glenohumeral ligament to shoulder stability.



▲ FIGURE 2-6: Anatomic drawing of the superior, middle, and inferior glenohumeral ligaments. Both the intracapsular and extracapsular views are represented.

Rotator Interval

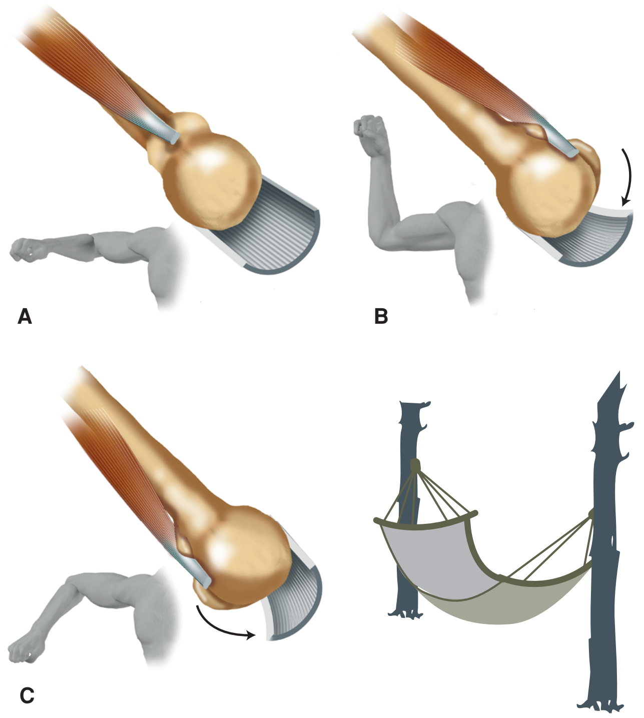
The "rotator interval" is a region that is between the superior border of the subscapularis tendon and the anterior border of the supraspinatus tendon. The two ligaments found within the rotator interval are the SGHL and the coracohumeral ligament (CHL) (57). The CHL is a dense fibrous structure that extends from the lateral aspect of the coracoid to the greater and lesser tuberosity of the humerus just adjacent to the bicipital groove (58). Portions of the CHL form a tunnel for the biceps tendon and blends inferiorly with the SGHL. Some investigators have demonstrated the CHL as a thin capsular fold without any ligamentous form (59), while others have suggested that the CHL may represent an accessory insertion of the pectoralis minor tendon (41). The SGHL originates from the supraglenoid tubercle anteroinferior to the origin of the long head of the biceps tendon and inserts onto the humerus on the proximal tip of the lesser tuberosity. Significant variations in the size and shape of the SGHL exist between individuals. In contrast to the CHL, Cooper et al. (59) demonstrated that the SGHL is a ligamentous structure with collagen bundles organized in a longitudinal direction. Both CHL and SGHL run parallel to each other in the rotator interval to limit inferior translation and external rotation in the adducted arm position or posterior translation with the arm in flexion, adduction, and internal rotation (17,19). Furthermore, deficiency or injury to the rotator interval may result in MDI, while contracture in this region may limit external rotation and forward flexion (60–62). Lee et al. (30) reported that in patients with MDI, the rotator interval width and depth were significantly greater than in normal patients on MRI. Furthermore, the capsular dimensions at the inferior and posteroinferior regions were larger as well. However, in contrast to the above findings, Provencher et al. (63) did not find a difference in the rotator interval distance between normal patients and instability patients. Furthermore, Mologne et al. (64) reported that with closure of the rotator interval in a cadaver model benefited or decreased anterior instability; however, posterior instability did not improve.

Middle Glenohumeral Ligament

The MGHL has the greatest variations among individuals and is absent in up to 30% of cases and poorly defined in another 10% (22,65,66). It typically originates from the superior glenoid just inferior to the SGHL between the 1-o'clock and 3-o'clock positions and blends in with the subscapularis tendon as its insertion approximately 2 cm medial to the lesser tuberosity (8,67). There are two variations to the MGHL that include a sheet-like structure that is confluent with the anterior band of the IGHL or a cord-like structure with a foraminal separation from the IGHL called a "Buford" complex (68,69). The MGHL primarily limits anterior humeral head translation with the arm abducted to 45 degrees and externally rotated. When the arm is in the adducted position, the MGHL functions to limit external rotation and inferior translation (8,17,70).

Inferior Glenohumeral Ligament

The IGHLC is a hammock-like structure that originates from the anterior inferior glenoid rim and labrum to insert below the MGHL on the inferior margin of the humeral articular surface and anatomic neck (Fig. 2-7).



▲ **FIGURE 2-7:** The inferior glenohumeral ligament complex provide support to the humeral head like a hammock **(A)**. Reciprocal tightening in external rotation **(B)** and internal rotation **(C)** provides barrier to prevent anterior and posterior instability, respectively.

The IGHLC is divided into three main components: A thick anterior band, a thinner posterior band, and the interposed axillary pouch between the two bands (65). Cadaver studies have found that the anterior band of the IGHLC averages 2.8 mm while the posterior portion of the IGHLC averages 1.7 mm in thickness. However, the authors could not identify a distinct posterior band (71). Recent studies also support that the posterior band of the IGHLC is less consistent than both the anterior band and the axillary pouch (72). The IGHLC function to support the humeral head and prevent translation when the arm is in the abducted position (73). Global stability requires function of all three components of the IGHLC. With abduction and external rotation of the arm, the entire complex becomes taut and moves beneath the humeral head to prevent anterior translation. However, with internal rotation and abduction, the IGHLC functions to limit posterior translation (17,19). When the arm is in the 90-degree abduction and extension (30 degrees), the anterior band of the IGHLC prevents excessive anterior and posterior translation. Conversely, with the arm in the 90-degree abduction and flexion (30 degrees) position, the posterior band of the IGHLC prevents excessive anterior and posterior translation (74).

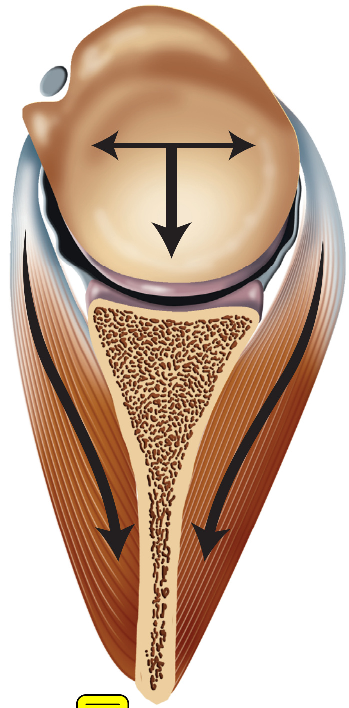
Tensile testing of the anterior band of the IGHLC in the position of clinical apprehension (abduction and external rotation) revealed that 66% of the specimens failed at the glenoid insertion while 34% failed at the midsubstance and humeral insertion.

Failure at the glenoid insertion region can be grouped into two separate pathologies. In one scenario, the failure occurred with the labrum completely avulsed from the glenoid bone (63%) and the other failure mode occurred at the ligament–labral junction with the labrum remained attached to the glenoid (37%). Before failure, all regions of the IGHLC experienced significant amount of strain (75). The ultimate load to failure of the anterior band (213 to 353 N) was not significantly different between the three modes of failure (glenoid, midsubstance, or humeral site). However, the amount of elongation was found to be greater at the glenoid and humeral insertion sites than specimens with midsubstance failures. Thus the yield strain at the glenoid and humeral region was larger than the midsubstance area prior to failure; however, permanent stretching of the anterior band IGHLC could never exceed a length greater than 4% strain (67,75). Several other studies have also investigated the strain of the IGHLC before failure and reported higher values of 9% to 11% (71,72). This difference may be attributed to the differences in the cadavers, modes of measurement, and equipment. Failure mode is also age dependent; in younger patients the disruption of the anterior band of the IGHLC typically occurs at the glenoid site while older individuals tend to fail at the midsubstance. Furthermore, the ultimate load of failure is significantly higher in the younger age group (76).

Dynamic Stabilizers

Rotator Cuff Musculature and Biceps Tendon

The rotator cuff musculature comprises supraspinatus, infraspinatus, teres minor, and subscapularis muscles. Contribution of the rotator cuff muscle group to glenohumeral stability occurs through three distinct mechanisms (39,77–79) that include: (1) joint compression, (2) coordinated contraction of the cuff muscle to guide the humeral head onto the center of the glenoid, and (3) dynamization of the glenohumeral ligament with shoulder range of motion through the cuff attachments (65). The rotator cuff muscles are well positioned to provide a coordinated compressive joint load to stabilize the shoulder throughout the different ranges of motion. Lippitt et al. (49) first described the effect of "concavity compression" in which compression of the humeral head into the glenoid cavity stabilizes it against translating forces (Fig. 2-8). With the labrum intact, the humeral head resisted tangential forces of up to 60% of the compressive load before instability. The greatest stabilizing effect was seen in the superior and inferior directions (52) while the least stable direction is anterior (51), which may be attributed to the glenoid depth in these regions respectively. Furthermore, resection of



▲ FIGURE 2-8: Cocontraction of the rotator cuff musculature results in compression of the humeral head onto the glenoid surface to improve dynamic stability of the glenohumeral joint.

the labrum decreased the effectiveness of the compression stabilization effect by approximately 20%. This value has been debated in literature, with a more recent study demonstrating the average contribution of the labrum to glenohumeral stability through the concavity compression is only 10%. Stability was also greater in the hanging arm position compared to arm abduction–external rotation under the concavity-compression mechanism (51). These findings indicate that the effect of concavity compression may be an important stabilizer of the glenohumeral joint in the midranges of motion when the capsuloligamentous structures are lax. When the arm is in the extremes of motion, the capsuloligament structures are stretched to enhance their contribution to stability.

Cocontraction of the cuff muscles with the long head of the biceps tendon enhances the conformity fit of the humeral head onto the glenoid and further stabilizes the glenohumeral joint (80–83). The stabilizing effect of the rotator cuff on glenohumeral dynamic stability has been well demonstrated in the literature. Kronberg et al. (84) revealed altered rotator cuff and deltoid EMG activity in patients with generalized ligamentous laxity and instability when compared to normal individuals. Warner et al. (85) further demonstrated rotator cuff muscle strength differences in patients with shoulder instability compared to normal. McMahon et al. (86) has also shown significantly reduced EMG activity in the supraspinatus muscle from 30 to 60 degrees of abduction in patients with anterior shoulder instability. In a dynamic shoulder model, 50% reduction in the rotator cuff forces resulted in increased anterior displacement by 46% and posterior displacement by 31%. However, a decrease in the rotator cuff strength did not significantly influence inferior instability (79).

Many investigators have studied the contribution of the biceps tendon to glenohumeral stability. The origin of the long head of the biceps tendon arose directly from both the supraglenoid tubercle and the superior glenoid labrum. Most of the attachment on the labrum is posterior in orientation (87). Itoi et al. (80) evaluated the stabilizing effect of the biceps tendon in a cadaver model and found that both the long and short head of the biceps have similar roles in preventing anterior shoulder instability with the arm in abduction and external rotation. Their role is further increased as the intrinsic shoulder stability decreases (capsule tear or Bankart lesion). Furthermore, the biceps becomes more important than the subscapularis in anterior stability as the stability from the capsuloligamentous structures decreases (81). Several other studies have also found that the magnitude of the joint compression stabilizing effect exceeds that of the static capsuloligamentous factors (49,52).

Deltoid Musculature

The deltoid muscle comprises three portions; anterior, middle, and posterior. It is a large triangular shaped bulky muscle which contributes to approximately 20% of all shoulder muscles (88). Morrey et al. (89) proposed the four essential muscle dynamic stabilizing effects contributing to shoulder stability. This includes: (1) passive tension from the muscle bulk, (2) muscle contraction that results in compression of the humeral head on the articular surface, (3) joint motion that tightens the passive ligaments of the shoulder, and (4) the barrier effect of the contracted muscle. Using a dynamic stability index, Lee and An (90) demonstrated the middle and posterior deltoid provided more stability by generating more compressive forces and lower shear forces than the anterior deltoid. Furthermore, the deltoid muscle produces more compressive force when the arm is elevated than in the neutral position. With the arm in external rotation, the insertion of the deltoid moves more posteriorly in relation to the glenohumeral joint, thus contraction at this position will produce a posteriorly directed compressive force and tensioning to reduce anterior instability. Kido et al. (91) also showed that with the capsule intact, anterior displacement is significantly reduced by application of load to the middle deltoid. However, with a simulated Bankart lesion, loading of each muscle portion significantly reduces anterior displacement. Thus the authors concluded that the stabilizing function of the deltoid becomes more essential as the shoulder becomes unstable.

Proprioception in Glenohumeral Stability

Placement of the upper extremity and hand in space for daily function is dependent on the perception of the shoulder joint position in space and motion. Capsule and ligaments function in joint stabilization by providing neurologic feedback that directly mediates joint position sensibility and muscle reflex stabilization. This sensory modality is called proprioception and mediated by receptors in the muscular and cutaneous structures of the shoulder joint. Specialized nerve endings and proprioceptive mechanoreceptors (Pacinian corpuscles, Ruffini endings, Golgi tendon endings, etc.) have been shown to exist in the capsule and ligaments (92,93). Stimulation of these mechanoreceptors result in muscle contraction around the joint that result in compressional forces which functions as an adaptive control for joint stabilization to sudden movements in acceleration or deceleration (20). It has been hypothesized that the receptors in the joint capsule responds to extremes in range of motion or deep pressure that may occur as a result of glenohumeral translation (94–96). Both Warner et al. (20) and Lephart et al. (97) have shown that the proprioception of the shoulder joint was disrupted in patients with glenohumeral instability compared to the asymptomatic shoulders. However, these differences were eliminated after surgical reconstruction. Laudner et al. (98) have also shown that shoulder proprioception at 75 degrees of external rotation decreases as the anterior glenohumeral laxity increases. After surgical reconstruction for shoulder instability, the joint position sense improved significantly in the position of abduction, flexion, and rotation from preoperative testing at greater than 5 years of follow-up. Interesting, the joint position in the contralateral shoulder also improved at final follow-up (99). Zuckerman et al. (100) also performed a similar study and reported that patients after open anterior stabilization procedure had 50% improvement of proprioceptive ability at the 6 months postsurgery time which improved to 100% or similar to the contralateral shoulder at the 1-year mark. Sullivan et al. (101) showed that patients after thermal or open capsulorrhaphy for anterior instability had significant better joint position sense than patients that had arthroscopic capsulorrhaphy. The authors attributed this finding to possible capsular retensioning and muscular scarring after the open and thermal capsulorrhaphy, respectively. Overall, the literature suggests that patients with recurrent shoulder instability will have a perceivable deficit in glenohumeral proprioception, which can be restored to normal after surgical repair or reconstruction. Capsuloligamentous structures may contribute to stability by providing the afferent feedback to reflexive muscle contraction of the rotator cuff, biceps, or deltoid. This reflexive contraction may serve as a protective mechanism via compressional forces against instability due to excessive glenohumeral translation or rotation.

SUMMARY

Successful management of shoulder instability requires knowledge of all factors responsible for stability and all of the potential factors that may contribute to instability. Both static and dynamic factors contribute to shoulder stability. Static factors include articular conformity of the glenohumeral joint and the negative intra-articular pressure. However, in the midranges of motion where the glenohumeral ligaments are lax, dynamic joint compression via rotator cuff muscle is responsible for joint stabilization. With the arm in the abducted and externally rotated position, the anterior portion of the IGHL is tensioned and provides a barrier to resist anterior instability. The rotator interval plays an additional role in limiting inferior translation of the humeral head when the arm in the adducted position. Cocontraction of rotator cuff musculature with the biceps tendon provides compression forces while enhancing the centering of the humeral head onto the glenoid to provide dynamic forces to stabilize the shoulder joint. Furthermore, both the deltoid muscle and proprioception also plays a dynamic role in shoulder stability. Abnormal version of either the glenoid or the humeral head has not been shown in the literature to contribute in glenohumeral instability. The spectrum of clinical instability can range from subluxation to transient luxation to dislocation. Most patients with a dislocation event will present with an injury to the anteroinferior capsulolabral complex or a "Bankart" lesion. In symptomatic patients, anatomic repair of the Bankart lesion is recommended. Some patients may also present with capsular laxity in combination to a Bankart lesion; therefore, a capsular shift may also be indicated in addition to a Bankart repair. It is essential to perform the capsular shift with the arm in specific positions (abduction of 30 degrees and external rotation of 30 degrees) to prevent overtightening and clinical loss in range of motion.

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AQ1: Please check whether all the head levels are given as intended.

AQ2: Please check whether the Reference 3 "Field (1997)" is OK as set.

AQ3: Please check whether the updated Author name and Book title are OK as set.

AQ4: Please check whether the updated Book title of Reference 41 "O'Brien et al. (1990)" is OK.

AQ5: Please provide editor name(s), Publisher name, and place of publication for reference "DePlama et al. (1949)", if possible.

AQ6: Please check the subpart label "(B)" has been added at the end of the caption of Figure 2-5 is OK.