Anatomy, Biomechanics, and Pathophysiology of Glenohumeral Instability

Brian J. Cole   **Clifford G. Rios**
Augustus D. Mazzocca   **Jon J. P. Warner**

INTRODUCTION

The complexity of the shoulder joint is best appreciated from our understanding of how shoulder anatomy and biomechanics are intrinsically related to the pathophysiology of shoulder instability. A multidisciplinary collaboration between surgeons, biomechanical engineers, anatomists, biochemists, and several other basic scientists is responsible for recent progress in these areas. Advances in experimental and clinical testing protocols have improved the current understanding of shoulder anatomy and biomechanics tremendously. No longer is our understanding of shoulder instability based on anecdotal and qualitative clinical experiences documenting shoulder pathology. A plethora of gross and histologic cadaveric studies, radiographic studies, and biomechanical studies now provides a sound foundation to understand how a minimally constrained articulation can balance mobility and stability. Alterations in any of the anatomic or biomechanical factors requisite for shoulder stability provide the pathogenesis for clinical instability.

The purpose of this chapter is to review the current anatomic and biomechanic factors that control glenohumeral joint motion and stability. Because anatomy and biomechanics are two interdependent disciplines, they will be discussed together as each of the relevant structures is reviewed. A deeper understanding of this relation will provide substantive data critical to classify and appreciate the pathophysiology of glenohumeral instability. More importantly, the application of anatomic and biomechanic principles provides a rational approach to the

treatment of glenohumeral instability for optimal functional restoration.

DEFINING THE PROBLEM

Laxity is asymptomatic, passive translation of the humeral head on the glenoid as determined by clinical examination and is unassociated with pain. Laxity is required for normal glenohumeral motion and may be affected by age,[62] gender, and congenital factors. In general, laxity changes with the position of the arm. At the extremes of rotation, the static restraints tighten and decrease laxity. Whether laxity is a risk factor for the development of clinical instability is a matter of debate.[184]

Instability is a pathologic condition that manifests as pain or discomfort in association with excessive translation of the humeral head on the glenoid fossa during active shoulder motion. Both clinical[48,62,73] and experimental studies[84,85,89,149,224,258,259,267] demonstrate a wide range of normal "play" in the glenohumeral joint; thus, it is the association with symptoms that clearly separates instability from excessive laxity. A spectrum of instability exists, representing increasing degrees of injury and dysfunction of the dynamic and static factors that function normally to contain the humeral head within the glenoid. Patients with multidirectional instability subluxate or dislocate in multiple directions, with concurrent reproduction of symptoms in at least two directions. Symptoms typically occur at midrange positions of glenohumeral motion, and often are associated with activities of daily living.[16,222] Recently, stability has been quantified as the force required to sublux the joint by a specified amount of translation. This has facilitated the study of the combined effects of muscle and capsular loads to joint stability and, therefore, represents an important advance in modeling the in vivo mechanical environment of the glenohumeral joint.[28,143,231]

ANATOMIC AND BIOMECHANIC CONSIDERATIONS

Anatomic control of glenohumeral joint stability can be divided into static (e.g., ligaments and tendons) and dynamic (e.g., muscular contraction) factors (Table 9-1). The cooperative role that these factors play is complex, and no single factor is responsible for glenohumeral joint stability. Similarly, no single lesion is responsible for clinical instability (Table 9-2). Contemporary approaches to the treatment of glenohumeral joint instability are directed at restoring normal anatomy and biomechanics, as opposed to simply constraining motion, as has been historically described.[46,64]

Establishing criteria for "normal" and "pathologic" conditions of the glenohumeral joint is often difficult owing

TABLE 9-1
FACTORS MAINTAINING JOINT STABILITY

Static Factors

Articular components
Articular version
Glenoid labrum

Negative intraarticular pressure
Adhesion–cohesion
Capsule and ligament
Rotator cuff

Dynamic Factors

Rotator cuff
Biceps brachii
Scapular rotators
Proprioception

TABLE 9-2
NORMAL AND ABNORMAL ANATOMY AND BIOMECHANICS

Stability Factor	Pathoanatomy
Glenoid version	Congenital: abnormal version; dysplasia Fracture causing abnormal version
Humeral version	Congenital: abnormal version; dysplasia Fracture/surgery causing abnormal version
Articular congruity	Congenital: dysplasia Acquired: fracture, Bankart lesion, osteoarthritis
Labrum	Bankart lesion "Fraying" secondary to laxity
Capsuloligamentous	Traumatic tear, cumulative microtrauma with plastic deformation Congenital laxity Loss of proprioceptive feedback
Negative intraarticular pressure	Capsular tear "Rotator interval" defect Lax capsule
Rotator cuff deficiency	Traumatic tear, cumulative microtrauma
Biceps	SLAP lesion Tendon rupture
Scapulothoracic motion	Dyskinesis: fatigue and weakness of serratus Long thoracic nerve palsy

SLAP, superior labrum from anterior to posterior.

to the considerable individual variation in capsuloligamentous anatomy[58,64,180,182,258,259] and in inherent shoulder laxity.[62,84,85,139] Dynamic factors (i.e., rotator cuff and biceps) are affected by their level of conditioning and strength. Scapulothoracic motion[263] is a more subtle, but equally important, dynamic factor helping to maintain shoulder stability. To add to this complex equation, an interaction between static capsuloligamentous factors and dynamic muscular factors, mediated through proprioception, has been postulated.[25,42,135,136,139,243,252]

Anatomic and biomechanic dysfunction leading to glenohumeral instability results from varying levels of applied stress (i.e., a single traumatic event vs. cumulative microtrauma), the relative risk of injury associated with an activity, the quality and integrity of the static stabilizers, and the strength and conditioning of the dynamic stabilizers. An individual's "susceptibility" for glenohumeral instability is dependent on these factors, each of which will be discussed in the following sections. The consequences of deficiency of any one component will be presented.

Static Factors

Articular Version

Both arthrographic and roentgenographic studies have characterized the relation between the humeral head and the glenoid surface of the scapula. With the arm hanging at the side in an adducted position, the scapula faces 30 degrees anteriorly on the chest wall and tilts 3 degrees upward relative to the transverse plane and 20 degrees forward relative to the sagittal plane (Fig. 9-1).[180] As described by Saha,[218] in 75% of persons the average glenoid orientation

is in 7 degrees of retroversion, with 25% of persons having anteversion ranging from 2 to 10 degrees. Churchill[43] et al. evaluated 334 cadaveric scapulae and found that Caucasians have more retroversion than African Americans. Further, there is no difference in retroversion seen between males and females. Saha[218] and others[14] have observed that the glenoid has an average superior tilt of 5 degrees (Fig. 9-2). Scapular inclination may have a contributory role in controlling inferior stability.[259] However, the difficulty in interpreting these studies arises from a wide range of interindividual variability, the reproducibility of techniques used to measure these factors, and an unknown relative contribution to clinical instability.

Recently, the anatomy of the proximal humerus has been significantly clarified. Saha[218] was one of the first to radiographically show that the neck-shaft angle averages 130 to 140 degrees and retroversion averages 30 degrees relative to the transepicondylar axis of the distal humerus (see Fig. 9-2).

Walch et al.,[254] using a micron precision probe and a computer to render a three-dimensional image of 65 humeri, determined that the inclination of the articular surface varies between 114 and 147 degrees with an average value of 130 degrees. In this same study, the average humeral retroversion measured 17.9 degrees (range, −6.5 to 47.5 degrees).

Articular Conformity

The glenohumeral joint in the adult consists of the humeral head and glenoid surface of the scapula. Understanding the glenoid and humerus as separate, but interdependent, congruent structures is integral to appreciate how
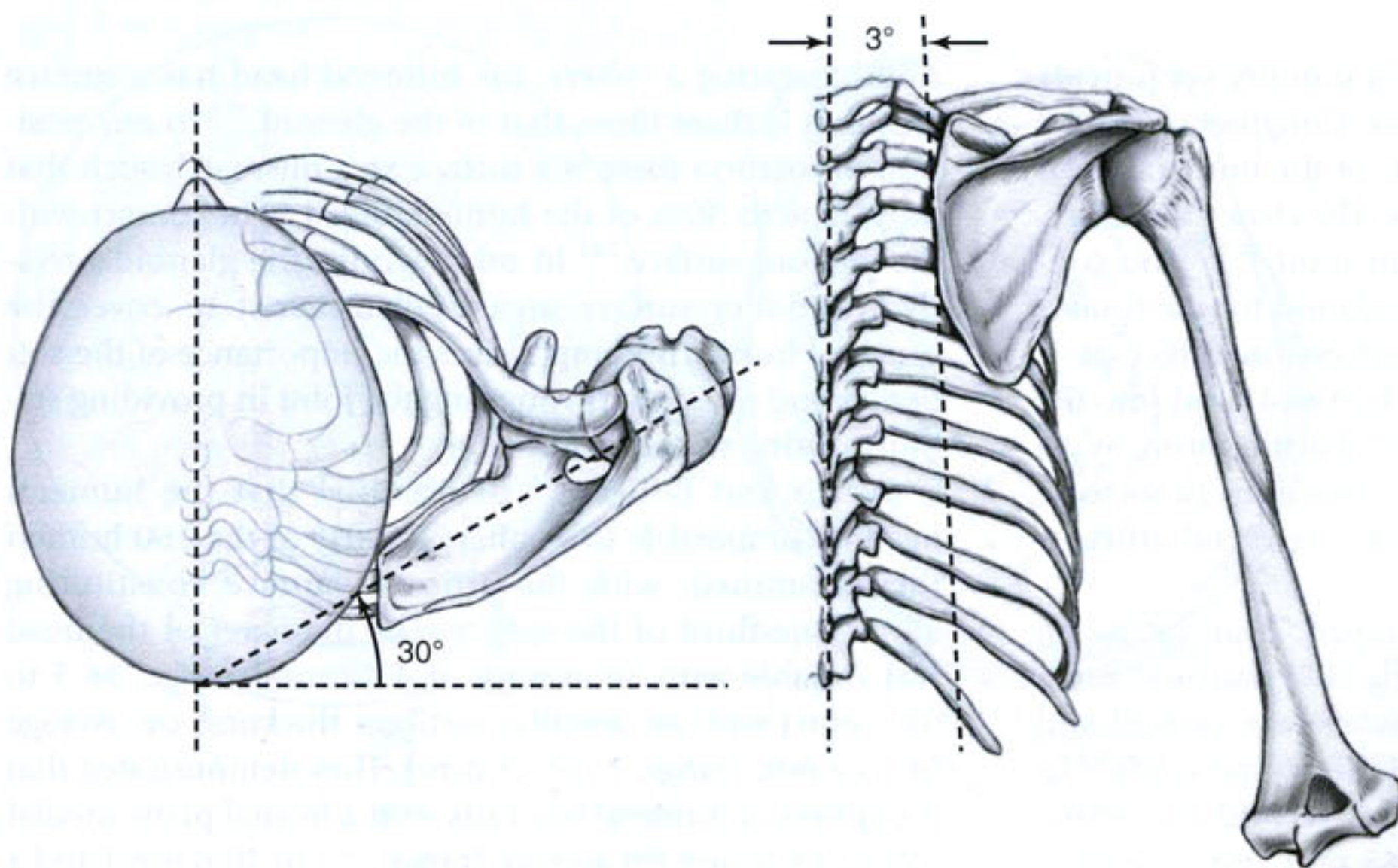

Figure 9-1 Scapular orientation on chest wall. **(Left)** 30 degrees anterior. **(Right)** 3 degrees upward. (Adapted from Warner JJP. The gross anatomy of the joint surfaces, ligaments, labrum and capsule. In: Matsen FA III, Fu FH, Hawkins RJ, eds. *The shoulder: a balance of mobility and stability.* Rosemont, IL: American Academy of Orthopaedic Surgeons, 1993:9.)

Figure 9-2 **(Left)** Superior tilt of the glenoid (see text). (From Warner JJP. The gross anatomy of the joint surfaces, ligaments, labrum and capsule. In: Matsen FA III, Fu FH, Hawkins RJ, eds. *The shoulder: a balance of mobility and stability.* Ro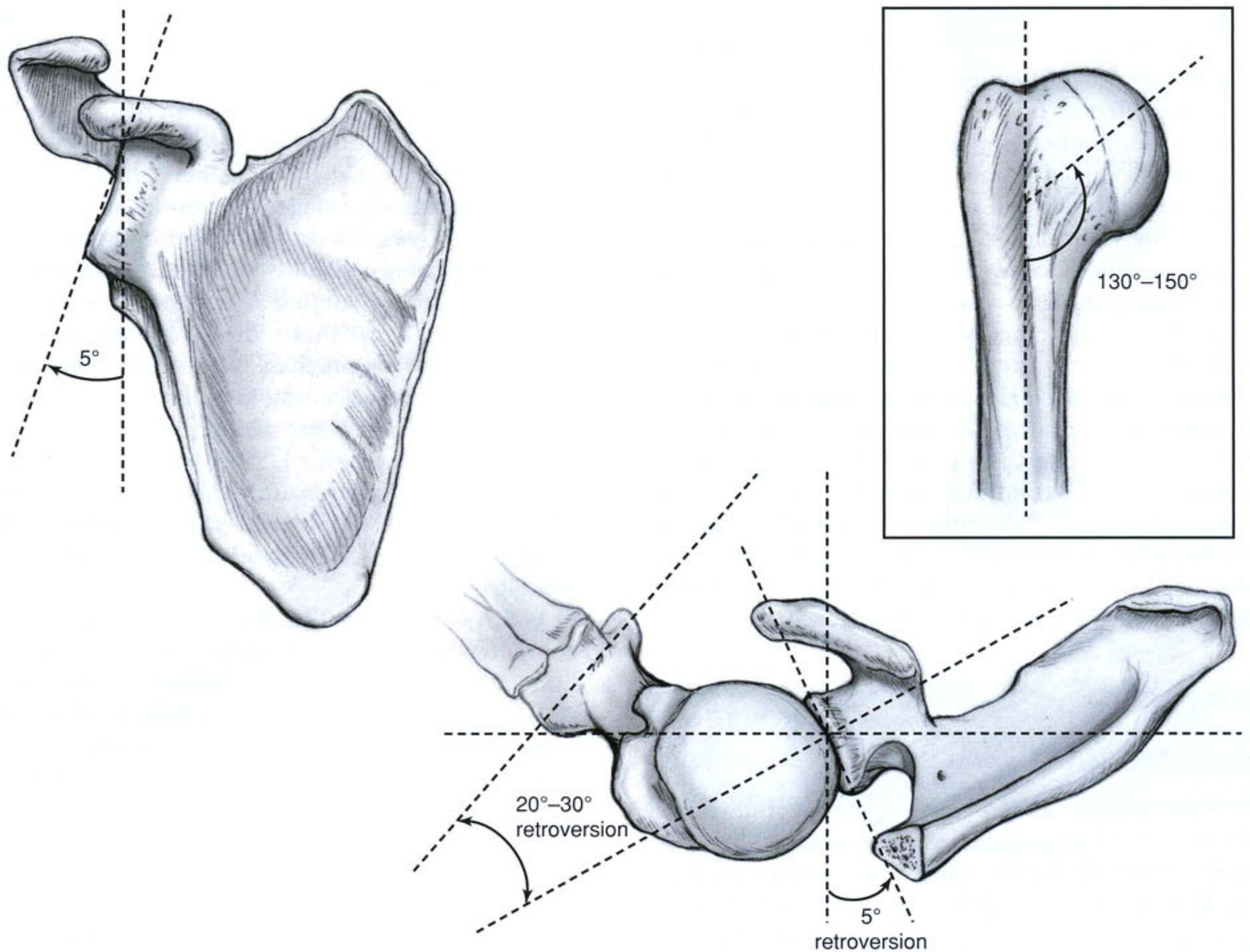semont, IL: American Academy of Orthopaedic Surgeons, 1993:9, with permission.) **(Center and right)** Glenoid and humeral version, and neck-shaft angle of proximal humerus (see text). (Adapted from Warner JJP, Caborn DNM. Overview of shoulder instability. *Crit Rev Phys Rehabil Med* 1992;4:145–198.)

these two joint surfaces can maintain stability yet provide for a relatively large range of motion. Congruence can be defined as the difference in the radii of the humeral head and the glenoid articulating surfaces. The closer the difference is to 0, the more congruent is the joint.[23,229] This congruent articulation provides the foundation for the rotator cuff to establish a concavity–compression effect as it dynamically compresses the convex humeral head into the matched concavity of the glenoid.[140] Furthermore, as an extension of the glenoid, the labrum functions to increase the depth and surface area of the glenohumeral articulation, enhancing this effect.[95]

The glenoid surface is "pear-shaped," similar to an inverted comma, being approximately 20% narrower superiorly than inferiorly (Fig. 9-3). The average vertical and transverse dimensions are 35 and 25 mm, respectively.[43] In contrast, the larger humeral head has vertical and transverse dimensions averaging 48 and 45 mm, respectively.[180]

Approximating a sphere, the humeral head has a surface area that is three times that of the glenoid.[229] In any position of rotation there is a surface area mismatch such that only 25% to 30% of the humeral head is in contact with the glenoid surface.[220] In other words, the glenoid's relatively smaller surface area is insufficient to cover the humeral head. This emphasizes the importance of the soft tissues and muscles surrounding the joint in providing stability during shoulder function.

Walch and Boileau[256] determined that the humeral head is comparable to a sphere in 90% of the 160 humeri they examined, with the articular surface constituting about one-third of the sphere. The diameter of the head was variable with an average of 43.2 mm (range, 36.5 to 51.7 mm) with an articular cartilage thickness on average of 15.2 mm (range, 12 to 18 mm). They demonstrated that the spherical humeral head sits with a frontal plane medial offset of 6.9 mm on average (range, 2.9 to 10.6 mm) and a
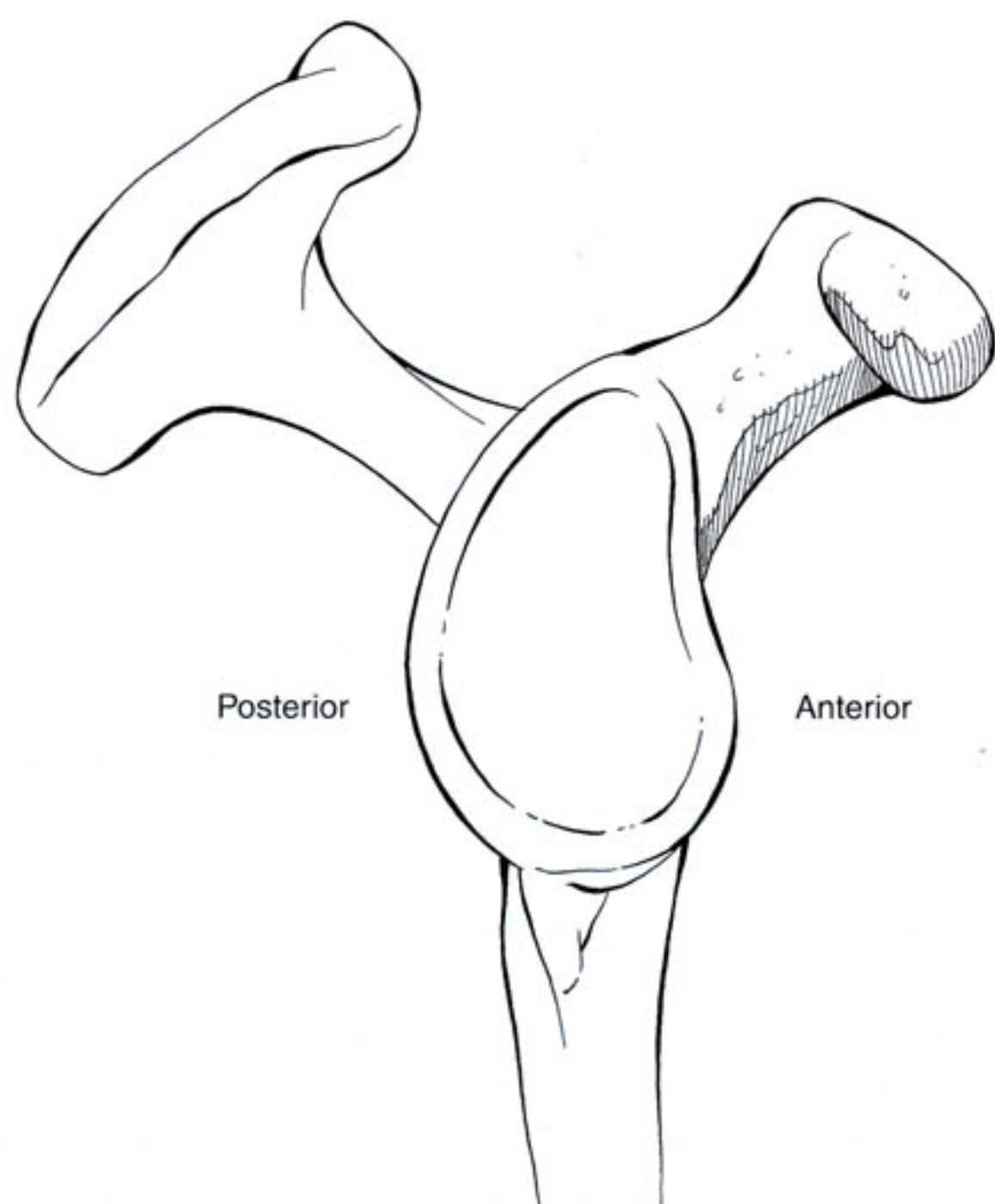
Figure 9-3 The "pear-shaped" face of the glenoid that articulates with the humeral head. (From Boardman ND III, Fu FH. Shoulder biomechanics. In: McGinty JB, Caspari RB, Jackson RW, Poehling GG, eds. *Operative arthroscopy.* Philadelphia: Lippincott-Raven, 1996:627, with permission.)

sagittal plane posterior offset of 2.6 mm (range, 0.8 to 6.1 mm). These parameters, however, may have more implications for shoulder arthroplasty design than for shoulder stability. Similarly, Iannotti et al.[100] reported that the humeral head approximates a sphere in the central articular areas and becomes slightly elliptical peripherally. Varia-

tions in these findings and their effect on the pathophysiology of shoulder instability are not clearly understood.

Conceptually, the glenohumeral joint has been compared to a "golfball sitting on a tee"[180] (Fig. 9-4). This analogy is based on historical beliefs sighting the relatively small area of the glenoid and its relative shallowness compared with the humeral head, allowing only a limited portion of the humeral head to contact the glenoid in any single shoulder position.[32,218,219] In fact, the articular surfaces of the humeral head and glenoid are almost perfectly matched with a congruence within 3 mm, with deviations from sphericity of less than 1%.[166,229] Additionally, the cartilage of the glenoid is thicker peripherally, and thus, plain radiographs tend to underestimate the relative concavity of the glenoid. This would imply that the glenohumeral joint would function similar to a ball-and-socket articulation as described by Kelkar et al.[118,119]

Although some coupled translation occurs at the extremes of glenohumeral rotation,[84,85,139] tracking of the geometric center of the cartilaginous articular surface with simulated muscle forces actually approximates ball-in-socket motion.[119] In the stable shoulder, external and internal rotation is associated with posterior and anterior humeral head translation, respectively. These relations may be altered in the unstable shoulder. McMahon et al.,[154] in an elegant model using a dynamic shoulder testing apparatus, measured muscle force values and tendon excursions across the glenohumeral joint during abduction in the scapular plane. They determined that humeral head translations on the glenoid were less than 2 mm under all testing conditions used and that the joint does behave kinematically as a "ball-and-socket" articulation during glenohumeral abduction. The importance of these findings is that articular incongruency is probably less of a predisposing factor for instability
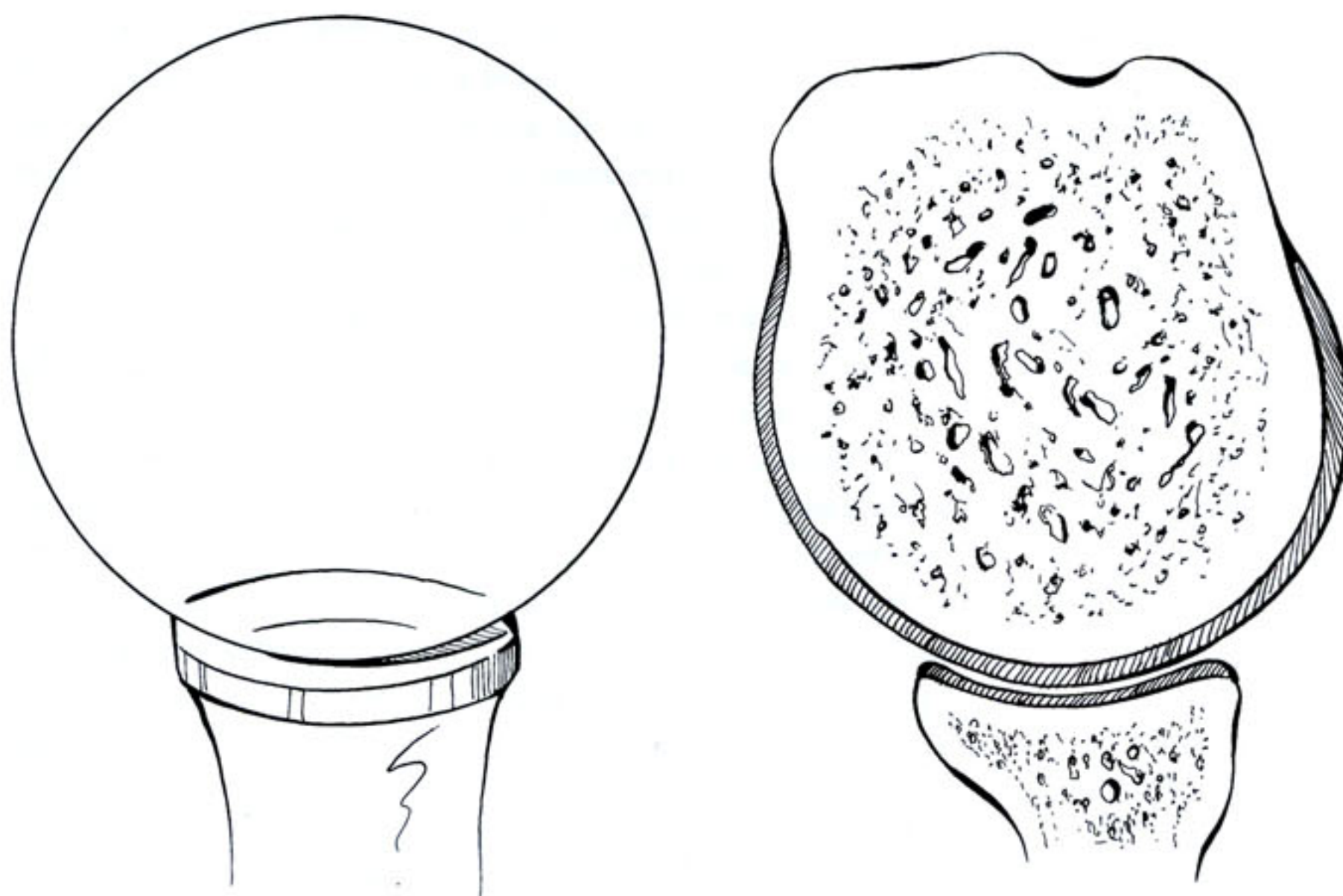

Figure 9-4 Analogy of the glenohumeral joint to a golf ball and tee. (From Boardman ND III, Fu FH. Shoulder biomechanics. In: McGinty JB, Caspari RB, Jackson RW, Poehling GG, eds. *Operative arthroscopy.* Philadelphia: Lippincott-Raven, 1996:627, with permission.)

than is surface area mismatch, as seen in glenoid dysplasia or glenoid fracture.[9,61,124,138,213,215] Additionally, the integrity of the soft tissues influences coupled translation, and this may also be a greater predisposing factor to instability than is articular incongruency.[35,84,94,95]

Glenoid Labrum

The relative lack of depth and surface area of the bony glenoid is compensated by the fibrous labrum acting to maintain normal glenohumeral biomechanics. As determined by Cooper et al.,[51] the labrum is a fibrous ring attaching to the glenoid articular cartilage through a narrow fibrocartilaginous transition zone. Above the glenoid equator, the labrum is relatively more mobile. In contrast, below the equator, the labrum is more consistently tightly attached to the glenoid articular cartilage. The tendon fibers of the long head of the biceps brachii blend with the superior labrum, and the inferior glenohumeral ligament (IGHL) blends into the inferior labrum. Cooper et al.[51] examined the vascular supply of the labrum and found that the superior and anterosuperior parts of the labrum were less vascular than the posterosuperior and inferior portions. Blood supply was limited to the periphery.

The labrum contributes to stability of the glenohumeral joint through several mechanisms. It acts as an anchor point for the capsuloligamentous structures.[51,164,204] Howell and Galinat[95] have shown how the labrum enhances stability by deepening the concavity of the glenoid socket to an average of 9 and 5 mm in the superoinferior and anteroposterior planes, respectively (Fig. 9-5). Loss of the labrum decreases the depth of the socket by 50% in either direction. Functionally, this acts as a "chock-block" preventing the head from slipping over the edge of the glenoid. Resection of the labrum reduces resistance to translation by 20%, and it is especially effective in doing so in combination with joint compression in the midrange of glenohumeral motion.[102,140] The labrum also acts to increase the surface area of contact, acting as a load-bearing

Figure 9-5 Glenoid labrum increases the surface area and depth of the glenoid socket. (Adapted from Warner JJP, Caborn DNM. Overview of shoulder instability. *Crit Rev Phys Rehabil Med* 1992;4:145–198.)

structure similar to the function of the meniscus in the knee.[34,230] Unlike the meniscus, however, the labrum lacks the microscopic architecture to disperse hoop stress and, therefore, is unlikely to effectively bear load.

Vanderhooft et al.[250] and Bowen et al.[35] have shown that the labrum plays a significant stabilizing role during rotator cuff contraction, facilitating the concavity–compression mechanism as the humeral head is compressed into the glenoid. Lazarus et al.[130] have defined a *stability ratio*, a measure of the effectiveness of concavity–compression in the stabilization of the glenohumeral joint, as the ratio between the maximum dislocating force that can be stabilized in a given direction and the load compressing the head into the glenoid. They determined that by creating a chondral–labral defect, an 80% reduction in the height of the glenoid occurred, with a concomitant reduction in the stability ratio of 65% for translation in the direction of the defect.

A Bankart lesion represents a lesion of the labrum corresponding to the detachment of the anchoring point of the IGHL and middle glenohumeral ligament (MGHL) from the glenoid rim.[12,213] A Bankart lesion disrupts the concavity–compression effect during rotator cuff contraction, eliminates the "chock-block" effect, and decreases the depth of the socket by 50% with detachment of the capsuloligamentous structures. This lesion should not be confused with the normal anatomic variants of a sublabral sulcus underneath a cord-like MGHL, the Buford complex,[275] or a loosely attached labrum superiorly. Whether the Bankart lesion is the "essential lesion" leading to recurrent anterior instability, as suggested by several authors, is currently a topic of interest.[12,32,164] Isolated detachment of the labrum as a singular entity leading to recurrent instability was challenged early in the literature by Townley[245] and then by Speer et al.[232] Normal variations of the capsulolabral complex occur commonly.

The difference between acute and chronic shoulder instability is important clinically. In acute shoulder instability, the authors feel that the labrum is the "essential" lesion. In a classic article by Arciero et al.,[8] repair of the Bankart significantly reduced the recurrence rate of anterior shoulder dislocation. In patients with chronic anterior instability, it is the capsule and labrum combined, or capsulolabral tissue, that is the essential lesion.[148]

Pagnani et al.[190] have demonstrated the importance of the superior portion of the glenoid labrum. In a cadaver study, isolated lesions of the anterosuperior portion of the labrum did not have a significant effect on glenohumeral translation. However, complete lesions of the superior portion of the labrum associated with destabilization of the biceps insertion resulted in significant increases in anteroposterior and superoinferior glenohumeral translations in the lower and middle ranges of elevation. The implications of these findings are that destabilization of the glenoid insertions of the superior glenohumeral ligament (SGHL), MGHL, and biceps insertion may be associated with subtle increases in translation and may be
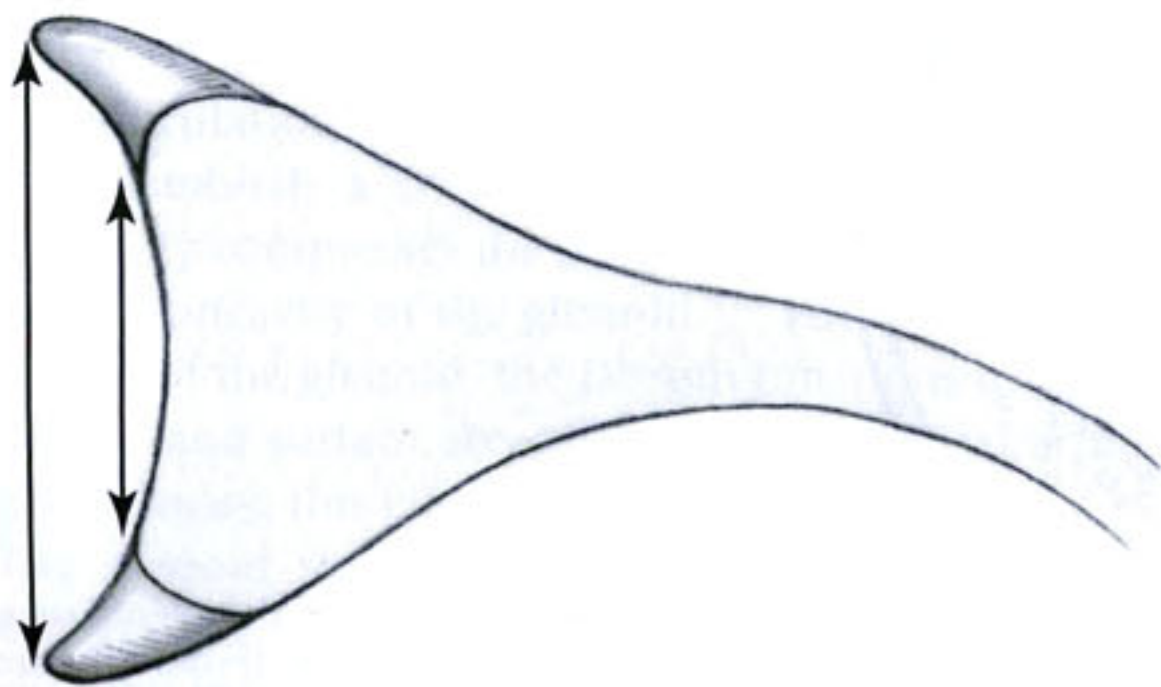
related to the symptoms present in patients who have isolated lesions of the superior labrum.[190,228]

Negative Intraarticular Pressure

In normal shoulders, a relative vacuum exists as a result of high osmotic pressure in the interstitial tissues, causing water to be drawn out of the glenohumeral joint.[147] As the articular surfaces are pulled apart, a suction effect develops to resist further displacement. The magnitude of this negative pressure has been shown to be about −42 cm of water in the adducted and relaxed shoulder. This increases to −82 cm of water during the application of a 25-N inferior force applied to the cadaver shoulder.[37] Negative intraarticular pressure becomes especially important when the rotator cuff is not contracting or when tension has not developed in the superior and coracohumeral ligaments during glenohumeral motion.

Pathologic conditions may include those that cause venting of the glenohumeral joint, leading to inferior subluxation, as has been shown experimentally by Warner et al.[257] and others.[127] Wuelker et al.[274] observed that venting of the joint increased displacement significantly in the anterior, posterior, and inferior planes. Anterior translation also increased by 55%[77] after capsular venting. This restraint becomes negligible, however, when the muscles contract with shoulder abduction or when the IGHL or superior capsular structures are under tension.[37,260] In a study by Helmig et al.,[91] venting of the capsule led to significant increases in anteroposterior translation and external rotation. The implications of their findings were that evaluation of shoulder stability in biomechanical investigations should be performed before violation of the negative intraarticular pressure mechanism occurs, or at the very least, measures should be corrected for this factor.

Thus, as a static restraint, negative intraarticular pressure appears to be important in limiting translation of the humeral head. Habermeyer et al.[82] noted that the presence of a Bankart lesion somehow eliminated the intraarticular seal to atmospheric pressure. A traumatic capsular rupture or an enlarged rotator interval capsular defect, possibly present at birth, presumably could lead to excessive glenohumeral translation, predisposing to instability.[49] Recently, Hashimoto et al.[87] have indicated that dynamic changes in intraarticular pressure can help differentiate patients with adhesive capsulitis, partial- and full-thickness rotator cuff tears, and instability. Practically, from a clinical perspective, reestablishing negative intraarticular pressure remains a theoretical concern and plays no role in the treatment of shoulder instability.

Adhesion–Cohesion

The glenohumeral joint contains less than 1 mm of synovial fluid that provides articular nourishment through diffusion and lubrication through several mechanisms (e.g., hydrodynamic, boundary, weeping, or boosted). Viscous and intermolecular forces help to create this adhesion–cohesion effect. Functionally, this is a stabilizing mechanism that permits sliding motion between the two joint surfaces while simultaneously limiting them from being pulled apart.[147] This is analogous to two glass plates separated by a thin film of water that slide easily over one another, but are difficult to separate. Negative intraarticular pressure and adhesive forces resulting from the presence of synovial fluid between the articular surfaces contribute static restraint, particularly when the capsule is lax and the muscles are relatively inactive.[101,127,145] Clinically, these factors probably play a minor role in maintaining glenohumeral stability and only at very low load levels.

Capsuloligamentous Structures

Few structures in the shoulder have received as much attention by investigators as the capsule and ligaments surrounding the glenohumeral joint. Traditionally, the ligaments of the capsule were described as discreet thickenings constituting the "glenohumeral ligaments."[58,180,183,221] Clinical observations at the time of surgery[4,32,70,164,169,192,213,215,217] or by cadaver shoulder dissections[12,45,58,64,95,126,133,174,182,213,217,246] Burkart and Debski[37a] have enhanced our understanding of these structures from an anatomic perspective. To obtain a concise appreciation of these structures, anatomic investigations must minimally distort the ligamentous relationships (Fig. 9-6). With increasing sophistication, the biomechanical function,[56,57,77,126,134,144,155,156,158,181,183,186,210,212,234,257,259] material properties,[21,29,56,83,117,134,144,155,156,158,207,210,234,244,257] and the interrelation of the rotator cuff and capsule have been described.[40,96,132,151,174,212,258] Synthesizing the available data into a cohesive algorithm applicable to the clinical setting of glenohumeral instability is a formidable task for most.

In a classic anatomic study, DePalma et al.[58] described the variability of the shoulder capsule, categorizing it into six basic types based on the pattern of the synovial recesses. Other anatomic studies have since confirmed and clarified the variable architecture of the glenohumeral ligaments.[64,164] Contemporary investigators suggest, as did DePalma et al.,[58] that some anatomic findings correlate with the risk of developing shoulder instability.[161,259] The basis for the functional roles of the capsular structures lies in their anatomic arrangement throughout the capsule. A significant advance in our knowledge came from Turkel et al.,[246] who confirmed by anatomic radiographic studies that different portions of the capsuloligamentous complex provided static stability that depends on arm position and the direction of the load applied to the proximal humerus.

The glenohumeral capsule is thin, less than 5 mm in thickness.[44] The glenohumeral ligaments function principally during rotation of the arm to reciprocally tighten and loosen, thus limiting translation and rotation in a
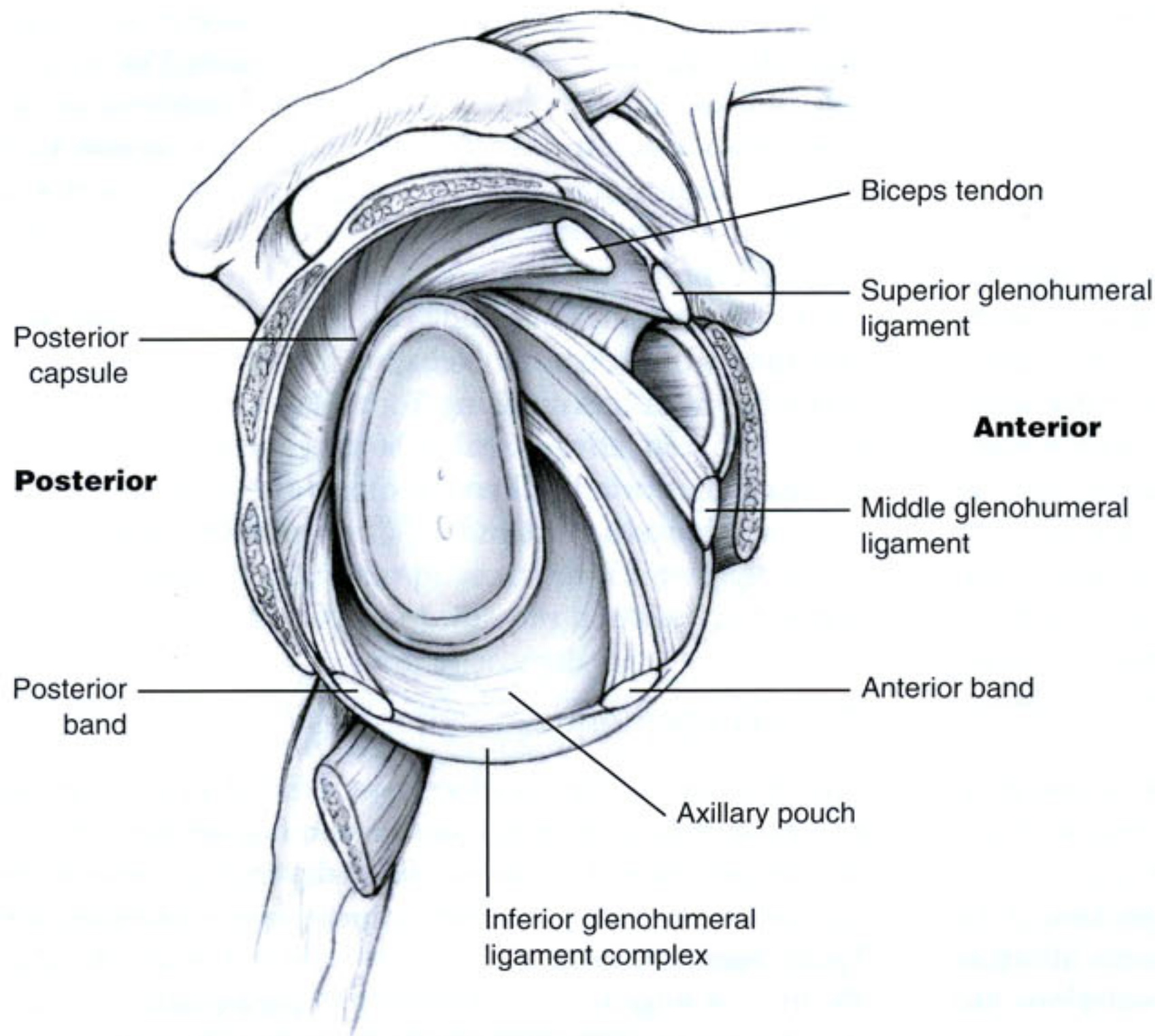
Posterior capsule

Posterior

Posterior band

Anterior

Biceps tendon

Superior glenohumeral ligament

Middle glenohumeral ligament

Anterior band

Axillary pouch

Inferior glenohumeral ligament complex

Figure 9-6 Capsuloligamentous anatomy viewed from the side with the anterior aspect to the right and the posterior aspect to the left. The humeral head has been removed, leaving the glenoid. The superior glenohumeral ligament and middle glenohumeral ligament are labeled. The inferior glenohumeral ligament complex consists of an anterior band, posterior band and interposed axillary pouch. The posterior capsule is the area above the posterior band. The biceps is also labeled. (Adapted from O'Brien SJ, Neves MC, Arnoczky SP, et al. The anatomy and histology of the inferior glenohumeral ligament complex of the shoulder. *Am J Sports Med* 1990;18: 449–456.)

load-sharing fashion.[261] In the midrange of rotation when these structures are relatively lax, stability is maintained primarily by the action of the rotator cuff and biceps through the concavity–compression effect across the glenohumeral joint.[85,140] The ligaments principally protect against instability when the joint is placed at the extremes of motion and become especially important when all other stabilizing mechanisms have been overwhelmed. [57,84,85,147,258]

Clinically, this becomes important during capsular reconstruction. Tensioning these structures in the midrange can potentially overtighten and constrain the joint, limiting rotation.[76,141,263] In the extreme case, this may lead to posterior humeral subluxation or arthritis.[18,22] The static role of each component of the capsuloligamentous structure is summarized in Table 9-3. In the sections that follow, each structure will be reviewed in terms of contemporary research describing the anatomy and biomechanics as well as a limited discussion on the relevant material properties.

Superior and Coracohumeral Ligaments

These structures are considered together because their anatomic courses are parallel and they constitute the reinforcing structures of the "rotator interval" region.[29,49,86,169,174,180,213] The rotator interval subtends a medially based triangular space, bordered superiorly by the anterior margin of the supraspinatus tendon, inferiorly by the superior border of the subscapularis tendon, medially

by the base of the coracoid, and laterally by the long head of the biceps tendon and sulcus. The floor of the rotator interval is normally bridged by capsule. Occasionally, a complete opening within the tissue spanning the rotator interval is present and is described as a "rotator interval capsular defect" (Fig. 9-7).

The coracohumeral ligament (CHL) is a dense fibrous extraarticular structure originating on the lateral surface of the coracoid process as a broad (1 to 2 cm) and thin structure. It inserts into the greater and lesser tuberosities adjacent to the bicipital groove and becomes intermingled with the tendinous edges of the supraspinatus and subscapularis, respectively.[58,86] Cooper et al.[52] questioned the significance of the CHL, describing it as a capsular fold creating a "pup tent" of capsule within the rotator interval. Others argue that the CHL is a well-defined structure that prevents excessive inferior translation of the adducted humerus in either position of humeral rotation.[171,186] While the specific function of the CHL remains disputed, it is known to be geometrically more robust and mechanically stiffer than the SGHL.[30] It also provides stability to the biceps tendon.

The SGHL lies deep to the CHL, is variable in size, and is present in over 90% of cases.[58,180,183,217,224,259] Usually quite diminutive, it originates from the superior glenoid tubercle just inferior to the biceps tendon and runs parallel to the CHL as it inserts into the superior aspect of the lesser tuberosity just medial to the bicipital groove.

TABLE 9-3
FUNCTIONS OF THE LIGAMENTS

Author	SGHL	CHL	MGHL	IGHLC	Other
Turkel et al.[246]	Little role in anterior stability		Primary stabilizer for anterior stability at 45 degrees of ABD; limits ER in mid-ABD	Primary stabilizer for anterior instability in ABD	Subscapularis is secondary stabilizer at 45 degrees of ABD
Ovesen and Nielsen[185–187]	Secondary stabilizer to posterior instability	Secondary stabilizer to posterior instability; primary stabilizer against inferior instability in ABD	Important for anterior instability at 45 degrees of ABD	Posterior capsule plays role in anterior and posterior stability	
Schwartz et al.;[224] O'Brien et al.[181]	Little role in stability			1-degree and 2-degree stabilizers against anterior and posterior instability in ABD	
Basmajian and Bazant[14]	Primary restraint to inferior translation in ADD	Primary restraint to inferior translation in ADD			
Warren et al.[266]	Secondary restraint to posterior instability in ADD, flexed, IR				Posterior capsule is primary restraint to posterior translation
O'Connell et al.[183]	Primary restraint to ER in ADD		Secondary restraint to ER in ABD; primary restraint to anterior instability at 45 degrees of ABD		
Ferrari[64]	Primary restraint to ER in lower range of ABD		Important restraint to ER at 60 and 90 degrees of ABD		
Helmig et al.[90]		Primary restraint to inferior instability			
Harryman et al.[84]		Primary restraint to ER			
Warner et al.[255]	Primary restraint to inferior translation in ADD	Minimal role in inferior stability	Secondary stabilizer for inferior translation in ADD	Primary stabilizer to inferior translation in ABD and secondary stabilizer in ADD	

ABD, abduction; ADD, adduction; CHL, coracohumeral ligament; ER, external rotation; IGHLC, inferior glenohumeral ligament complex; IR, internal rotation; MGHL, middle glenohumeral ligament; SGHL, superior glenohumeral ligament.

Opinions vary on the specific functions of these two ligaments. Harryman et al.[86] characterized the relative biomechanical contribution of the rotator interval capsule to shoulder stability in cadaver specimens. A transverse incision in the rotator interval region including the capsule, CHL, and SGHL allowed statistically significant increases in humeral head translations in all planes tested. Imbrication of the rotator interval decreased inferior translation in adduction and posterior translation in flexion to less than the intact state. No attempt was made to isolate the role of specific capsular ligaments. Burkart and Debski[37a] performed a selective sectioning study of these ligaments and concluded that the SGHL is an important stabilizer in the anterior direction. The SGHL also limits external rotation of the adducted arm.[37a] Basmajian and Bazant,[14] using electromyographic and anatomic dissections, showed that the
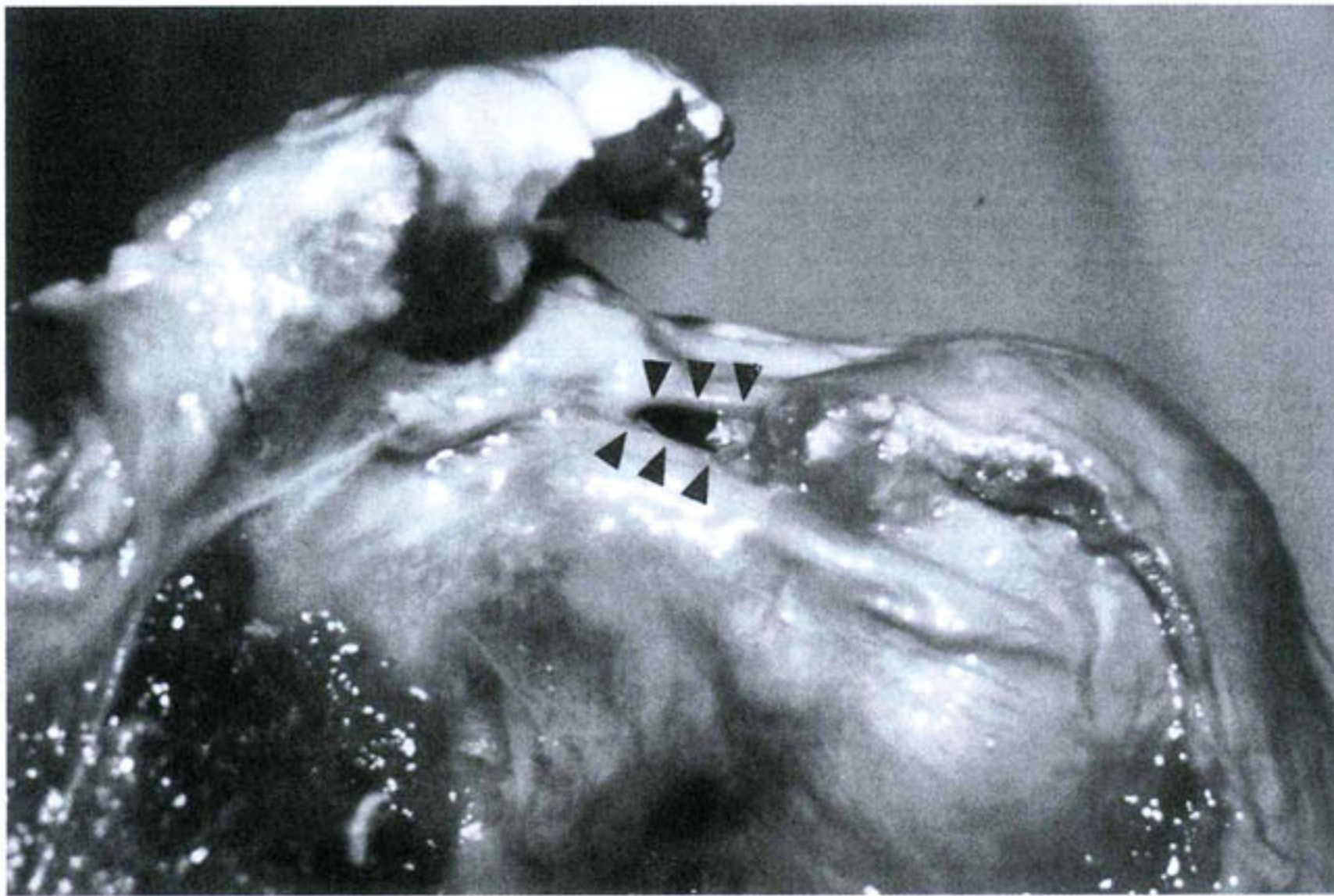
Figure 9-7 Gross anatomic specimen of a left shoulder demonstrating the opening within the rotator interval situated between the supraspinatus and subscapularis muscle tendons.

superior capsule and the CHL resisted downward displacement with the arm adducted, independent of load.

Patel et al.[193] described the CHL to consist of an anterior and posterior band originating at the coracoid and inserting into the lesser and greater tuberosities, respectively. During adduction and external rotation, the SGHL and anterior band of the CHL shortened from a maximally lengthened position. These changes were opposite those of the posterior band of the CHL that was maximally lengthened with adduction and internal rotation. Warner et al.[259] suggested that the SGHL resists inferior translation of the adducted shoulder and that the CHL is not important here. However, subsequent work by Boardman et al.[29] suggested that the CHL is the principal functional component of the capsule within the rotator interval. Observations by Lee et al.[133] suggest that the coracoacromial ligament has a role in static restraint of the glenohumeral joint as well. These authors propose that the coracoacromial ligament interacts with the CHL to prevent anterior and inferior translation, particularly between 0 and 30 degrees of abduction.

Despite varying opinions in the literature (see Table 9-3), the current consensus is that these two structures constrain the humeral head on the glenoid, limit inferior translation and external rotation when the arm is adducted, and limit posterior translation when the shoulder is in a position of forward flexion, adduction, and internal rotation. There has been renewed interest in this portion of the shoulder capsule because openings within the rotator interval have been associated with recurrent anteroinferior and multidirectional instability.[65,86,175,213] As suggested by several of these studies, addressing this pathology may be important in preventing recurrence. Conversely, contracture or scarring of this portion of the shoulder capsule has been associated with adhesive capsulitis.[86,171]

Middle Glenohumeral Ligament

As described by DePalma[58] and others,[180,182,224,258,259] the MGHL has the greatest variation in size and presence of all the ligaments of the shoulder. It is absent or poorly defined in 40% of individuals.[64,182] It originates from the supraglenoid tubercle and anterosuperior labrum, often along with the SGHL, and inserts just anterior to the lesser tuberosity, blending with the posterior aspect of the subscapularis tendon. Its variable morphology usually takes one of two forms: (a) sheet-like and confluent with the anterior band of the IGHL or (b) cord-like, with a foraminal separation between it and the anterior band of the IGHL. Moseley and Overgaard[164] reported that the MGHL originated from the scapular neck and formed an anterior pouch accommodating the humeral head in some patients with recurrent anterior instability.

It is generally believed that the MGHL functions as a passive restraint to both anterior and posterior translation of the humeral head when the arm is abducted in the range from 60 to 90 degrees in external rotation and limits inferior translation when the arm is adducted at the side. Those who are "MGHL dominant" individuals with a cord-like MGHL may be more dependent on this structure to provide a protective role against anterior instability.[161,259] Clinically, the MGHL may be detached from the anterior glenoid and constitutes the leading edge of a Bankart lesion, which typically includes the anterior band of the IGHL. However, the sublabral hole should not be confused with a detached labrum, for generally the labrum is more mobile above the equator of the glenoid.

Inferior Glenohumeral Ligament Complex

Originally described by DePalma,[58] several descriptions of the IGHL exist in the literature ranging from a triangular-shaped structure coursing from the labrum to the humeral neck, to one with well-defined thickenings at its leading edge.[182,244,246] Typically, it originates from the anteroinferior labrum or inferior half of the neck of the glenoid adjacent to the labrum and inserts just inferior to the MGHL at the humeral neck. Our current understanding has advanced to the point at which we now consider this structure to be quite developed, with very specific functions attributed to its individual components. O'Brien et al.[182] have defined this structure, through arthroscopic, gross, and histologic evaluation, as the inferior glenohumeral ligament complex (IGHLC) consisting of three components. They described discrete anterior and posterior bands (ligament) with an interposed thinner axillary pouch (see Fig. 9-6). The complex consists of three well-defined layers of collagen fibers extending from the glenoid to the humerus (inner and outer) and running circumferentially around the joint (middle). Ticker et al.[244] and Bigliani et al.[21] have recently challenged the presence of a discrete posterior band and

found all regions of the IGHLC to be thicker near the glenoid than the humerus.

The IGHLC contributes to glenohumeral stability in several ways. Recently, Kuhn et al. found that the IGHLC is a restraint to external rotation of the arm in neutral and abducted positions.[126] O'Brien et al.[182] suggested that the IGHLC functions as a hammock to support the humeral head as it undergoes reciprocal tightening–loosening with abduction or rotation as the orientation of the complex changes. In adduction, it forms a dependent fold, acting as a secondary restraint limiting large inferior translations.[182,259] In abduction, however, this complex moves underneath the humeral head, becoming taut, in the fashion of a hammock, effectively limiting inferior translation. As the arm is internally rotated, the complex moves posteriorly, and as the arm is externally rotated, the complex moves anteriorly, forming a barrier to posterior and anterior dislocation, respectively (Fig. 9-8). Horizontal flexion and extension in abduction will also tighten the posterior or anterior components, respectively, thereby limiting anteroposterior translation.[224] Another biomechanical study by O'Brien et al.[181] verified that the primary anteroposterior
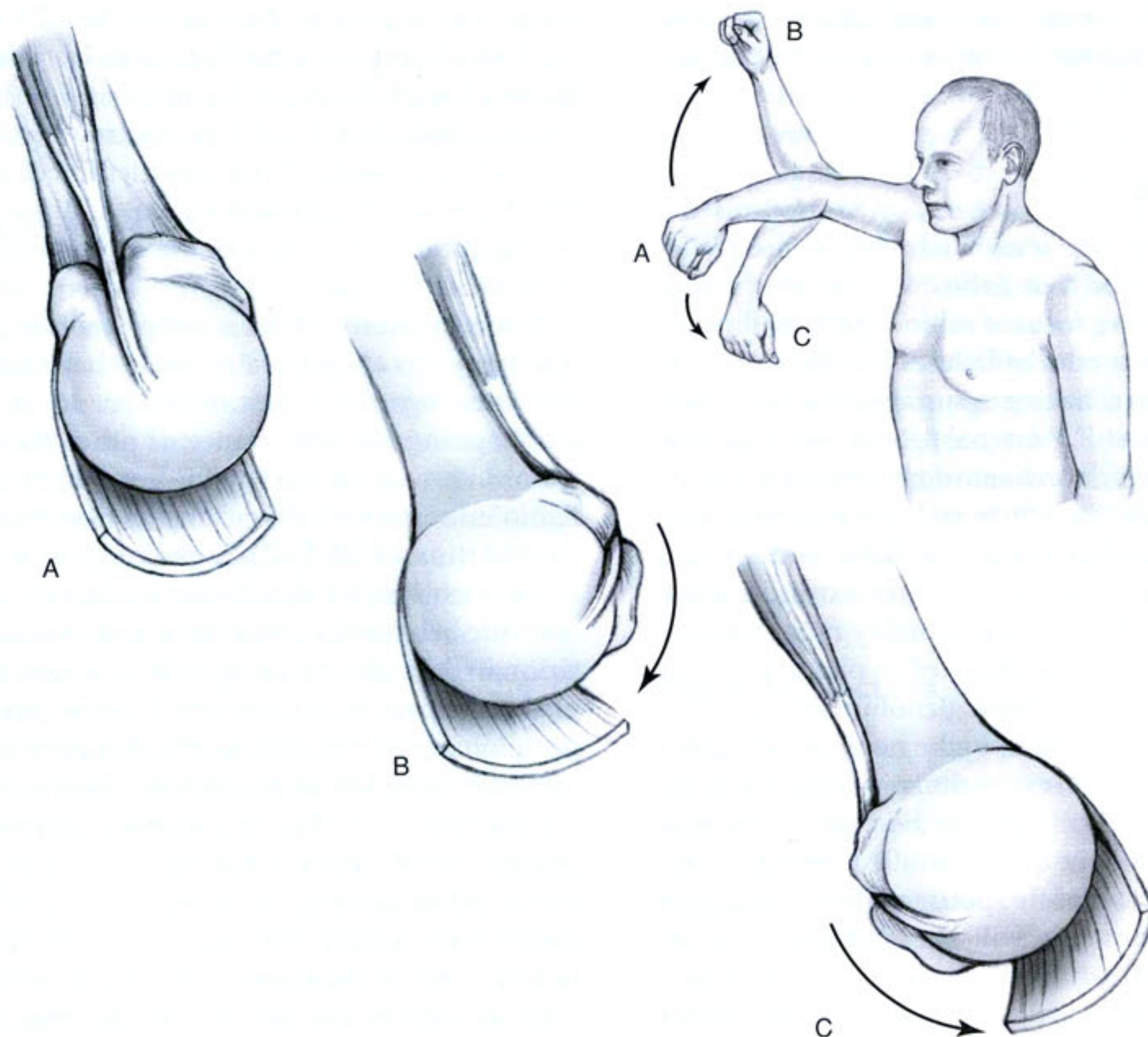

Figure 9-8 The "hammock"-like anatomy of the inferior glenohumeral ligament complex allows for reciprocal tightening of its anterior and posterior portions when the arm moves from neutral rotation in (**A**) abduction to external (**B**) and internal (**C**) rotation. (Adapted from Warner JJP, Caborn DNM. Overview of shoulder instability. *Crit Rev Phys Rehabil Med* 1992;4:145–198.)

stabilizer of the 90-degree abducted shoulder is the IGHL. The anterior band was the primary stabilizer in 30 degrees of horizontal extension and the posterior band at 30 degrees of horizontal flexion.

Injury to the IGHLC plays an integral role in the development of anterior instability. Surgical reconstruction directed at anatomic restoration of this part of the capsule has been advocated even in the earliest reports on the surgical management of shoulder instability.[12] Although all of the structures that define the shoulder capsule have at least a limited role, alterations of the IGHLC are believed by most to be a significant factor in the pathophysiology of anterior shoulder instability.

Posterior Capsule

This is the capsule extending from superior to the posterior band of the IGHLC to the intraarticular portion of the biceps tendon.[182] Other than the capsule found within the rotator interval, this is the thinnest region of the joint capsule.[49,182] There are no direct posterior ligamentous reinforcements. Its role is to limit posterior translation when the shoulder is forward-flexed, adducted, and internally rotated.[269] Clinically, this becomes relevant in patients who present with posterior instability. Unlike the other ligament structures about the glenohumeral capsule, the posterior capsule does not have a role in restraining external rotation.[5]

Material Properties

Because instability is often associated with failure of the static constraints (e.g., the capsuloligamentous structures), recent investigations have focused on the individual material properties and the modes of failure. The shoulder capsule is quite redundant, having a surface area two times that of the humeral head.[180] Material properties of the capsule refer to the intrinsic mechanical characteristics of its composition, molecular structure, and ultrastructure. Presumably anatomic variability (e.g., the SGHL and MGHL) may have clinical implications. In other words, a more robust ligament (e.g., IGHL) is presumably more tolerant of strain or force and would be expected to play a more significant role in helping to maintain glenohumeral stability.

Similar to other joint capsules in the body, the shoulder capsule is fibrous and rich in extracellular matrix. It is composed primarily of type I collagen, with lesser amounts of types II and III.[210] Debski et al.[56] quantified collagen fiber orientation in cadaveric capsule specimens and found that the collagen fibers of both the axillary pouch and the anterior band of the IGHL exhibited a random organization. Furthermore, there was no significant difference in fiber orientation seen in the bursal, middle, and articular portions of the axillary pouch. Malicky et al.[144] measured planar strains in the anteroinferior joint capsule and found considerable variability in maximum principal strains across specimens. The principal strain vectors were generally not aligned with the anterior band of the IGHL.[144] The results of these two recent studies strongly suggest that the IGHLC sustains loading in multiple directions rather than only along its length, as is the case with noncapsular ligaments.[259]

Reeves[207] determined that the average maximum tensile strength of the anteroinferior capsule measured in cadaver shoulders is 70 N (at least 20 kg), decreasing after age 50. Between the ages of 10 and 40, the anteroinferior labral insertion was the weakest portion of the whole complex, with more than two-thirds of the failures occurring there. Specimens in the fifth to seventh decades experienced capsular rupture and subscapularis tendon failure more frequently than failure at the labrum.[207] That the anteroinferior portion of the capsule fails first and capsular strength varies inversely with age has also been demonstrated by Kaltsas.[117] Hara et al.[83] evaluated the glenoid labrum and capsule and determined that the anteroinferior labrum close to the glenoid cartilage was weakest, rupturing with a mean force of 3.84 kg/5 mm.

The properties of the IGHL have been well described by Bigliani et al.,[21] who used tensile testing to analyze strength and failure modes in humerus–IGHLC–glenoid specimens. The region of the anterior band had the greatest thickness (average 2.8 mm), progressively decreasing in the axillary pouch (average 2.3 mm) and posterior capsular regions (average 1.7 mm). In contrast to O'Brien et al.,[182] no discrete posterior band was identified, and the axillary pouch was not the thickest region. Additionally, there were no significant differences in the resting length or width of these areas.[21]

Stress at failure of the anterior axillary pouch (average 5.5 MPa) was substantially lower than that described for the knee ligaments (estimated at 35 to 80 MPa198), emphasizing the importance of other stabilizing mechanisms in protecting the IGHLC from structural failure. Similar inferior strength characteristics have been described for the SGHL and CHL.[29] The superior band and anterior axillary pouch exhibit significant strain rate-dependent viscoelastic behavior. These effects were explained by compositional data determining that a proteoglycan content gradient exists, being greatest anterosuperiorly and least posteroinferiorly. Mechanically, this property leads to viscoelastic stiffening as the collagen fibers are "uncrimped" during tension.[21] This may also explain why the inferior glenohumeral ligament has the capacity to stretch considerably before ligament or insertion failure.[21] These investigators also determined that the predominant modes of failure were at the glenoid insertion, with slower strain rates as seen in the Bankart lesion, and in the midsubstance, with faster strain rates as seen with capsular laxity or stretching. This is explained by a nearly elastic behavior in the central region of the IGHL and principally viscoelastic behavior at the bony insertion.[21,244] These authors suggested

that viscoelastic behavior during tension and strain rate-dependent properties of the IGHL support its role as a humeral head stabilizer in the position of abduction and external rotation as force is rapidly applied. Subsequent tensile testing of the anterior band of the IGHLC in the apprehension position at substantially higher strain rates confirmed the viscoelastic property of increased failure stress that was previously noted.[133,155,156,158,234] Thus, functional adaptation may occur to stabilize the head during high-energy activities when other static or dynamic restraints are overwhelmed.

Morrey and Chao[163] have calculated that the anterior shear force in the position of apprehension is as high as 60 kg. To counteract these forces, contraction of the rotator cuff significantly reduces stress in the anterior capsule when the arm is in the maximally abducted and externally rotated position.[40] Thus, the dynamic restraints to stability function as a protective mechanism against structural failure of the static restraints. Although investigated to a lesser degree, the material properties of the restraints to posterior instability have been described. Weber and Caspari displaced the humeral head posteriorly in 90 degrees of flexion and full internal rotation, resulting in a horizontal split in the posterior capsule and posterior labral avulsion from the glenoid.[271] Because the results of these studies depend on a simulated mechanism of injury in the presence of an inactive rotator cuff, rigid interpretation and extrapolation to the pathoanatomy of instability is somewhat speculative.

Obligate Translations

A relatively new area of research interest is focused on understanding the relation between glenohumeral rotation and obligate translation caused by asymmetrical tightening–loosening of the capsuloligamentous structures. Tensile loading in either the anterior or superior structures is simultaneously accompanied by laxity in the posterior or inferior portion, respectively.[55,84,85,258] This is the so-called reciprocal load-sharing relationship of the capsule. Howell and Galinat[94] used axillary radiographs of patients to measure anteroposterior excursion during glenohumeral rotation. Except for maximal extension with external rotation, the humeral head remained centered on the glenoid. In normal subjects, the extended and externally rotated position caused the humeral head to translate posteriorly. In patients with anterior instability, posterior excursion did not occur. Taken a step further, Harryman et al.[85] monitored loads and translations with a magnetic-tracking device in cadaver specimens. Anterior translation occurred with flexion beyond 55 degrees, and posterior translation occurred with extension beyond 35 degrees. Interestingly, these authors found that surgical tightening of the posterior capsule resulted in increased anterior translation with flexion that occurred earlier in the arc of motion compared with normal specimens. Tightening of the rotator interval

also increased obligate anterior translation with flexion. The effects of Bankart repair and overtightened inferior capsular shifts were also investigated by Janevic et al.[110] These procedures shifted the humeral head and joint contact posteriorly during loading with abduction, extension, and external rotation. The importance of these findings is that static restraints may function in positions other than the extremes of rotation. It is conceivable that unidirectional tightness, primary (e.g., overhead athlete) or iatrogenic (e.g., anterior capsulorrhaphy), could lead to instability in the opposite direction. Moreover, excessive translation in one direction may require damage to restraints on the same and opposite sides of the joint.[68] These concepts, while requiring further investigation, add an additional layer of complexity to the diagnosis and treatment of shoulder instability.

Rotator Cuff as a Static Stabilizer

Passive tension within the rotator cuff musculotendinous structures appears to have some static role in preventing glenohumeral translation. The "posterior mechanism of dislocation" occurs in older patients who sustain supraspinatus and infraspinatus tendon tears, with or without capsular injury, in association with anterior dislocation.[64,207] Rupture of the subscapularis has also been noted in patients with recurrent dislocations who are older than 35 years of age.[172] The subscapularis statically limits anterior translation in lower ranges of abduction with similar limitations to posterior translation found from the infraspinatus and teres minor.[186,187] Recently, the contribution of passive bulk tissues and the deltoid to static inferior glenohumeral stability was investigated by Motzkin et al.[165] This study determined that in both humeral adduction and abduction, passive bulk tissues (i.e., all tissues superficial to the deltoid) and the deltoid did not provide significant stability to the shoulder joint. Thus, the rotator cuff appears to be one of the few dynamic restraints that have a concomitant passive role in preventing glenohumeral instability.

Dynamic Factors

Clinical experience suggests that static stabilizers by themselves may not be as important in enhancing glenohumeral stability as that provided by the dynamic stabilizers or the relation between them. Experimentally, specimens dissected free of the rotator cuff and long head of the biceps tend to demonstrate at least some degree of inferior subluxation.[127,186] Active contraction of these structures contributes to the dynamic stabilization of the glenohumeral joint through two mechanisms: (a) joint compression (e.g., concavity–compression) resulting from synergistic and coordinated rotator cuff activity and (b) ligament dynamization through direct attachments to the rotator

cuff muscles. Augmenting these mechanisms are the long head of the biceps brachii, coordinated scapulothoracic rhythm, and proprioception providing feedback about extremity position and movement.

Joint Compression

Contraction of the rotator cuff and long head of the biceps brachii augments joint stability by enhancing the conforming fit and increasing the load needed to translate the humeral head through compression of the humeral head into the glenoid.[40,140,147] The rotator cuff muscle forces are ideally aligned for effective compression of the glenohumeral joint at all shoulder positions.[132] Lippitt et al.[140] quantified the magnitude of the tangential forces required to produce glenohumeral dislocation in the setting of applied joint-compressive loads of 50 and 100 N. Tangential forces were as high as 60% of the applied joint-compressive load. The stability of the joint was markedly reduced if a portion of the labrum was removed. Vahey et al.[249] introduced the concept of "scapulohumeral balance" to illustrate that glenoid geometry coupled with joint compression is a major stabilizing force. Bowen et al.[35] determined that a joint compression load of 111 N was sufficient to stabilize the glenohumeral joint in the face of a 50-N force, despite sectioning of three-fourths of the joint capsule. It has been suggested from the results of ligament-cutting studies and direct quantification of the efficiencies of the dynamic stabilizers that joint compression is a more important stabilizer to translation than are static capsular constraints.[27,35]

Poppen and Walker[200] showed that the joint reaction force was a maximum of 0.89 times body weight directed into the face of the glenoid at 90 degrees of abduction using a simplified two-dimensional cadaveric model with digitized radiographs. Also, the subscapularis had a greater mechanical advantage at lower abduction angles (i.e., 60 degrees), whereas the deltoid had a greater advantage at higher abduction positions. McKernan et al.[151] have validated these findings and attributed them to the anterior location of the subscapularis tendon in lower ranges of elevation, making it a more effective stabilizer against a given translation. This effect is reduced as the shoulder is elevated and the line action of the subscapularis moves superior to the joint.

All portions of the rotator cuff are probably important in enhancing stability, as was shown by Blasier et al.[26] In their biomechanical study, omission of tension in any one of the rotator cuff muscles led to a substantial reduction in anterior joint stability. Labriola et al. reported that all rotator cuff muscles contribute equally to anterior stability when the glenohumeral joint is in the anatomic position; at end range, the subscapularis is less important.[107,128] This is supported by an investigation by Wuelker et al., which found 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.[278]

Rotator cuff tears result from either single traumatic or cumulative microtraumatic (i.e., overuse injuries) events. Because of age-related attrition, a dislocation in individuals older than the age of 40 is not uncommonly associated with a rotator cuff tear.[117,172,207] Rotator cuff tears result in superior translation of the humeral head during scapular plane abduction, and larger rotator cuff tears lead to increased displacement of the humeral head.[98] This demonstrates the importance of synchronous contraction of the entire cuff in maintaining containment of the humeral head in the glenoid.[200,267]

Increasing the joint compressive load appears to "center" the humeral head, reducing subsequent translation. This centering of the humeral head in the glenoid socket provides a stable fulcrum for elevation of the humerus.[67,97,240] Interestingly, the ability of isometric muscle contraction to "center" the humeral head is different in patients with traumatic instability compared to those with atraumatic instability. A recent evaluation of glenohumeral kinematics using magnetic resonance imaging revealed that such contraction led to recentering of the humeral head only in the patients with traumatic instability.[253]

In overhead athletes, for example, in whom the rotator cuff functions as an important decelerator to anterior translation, imbalanced muscle recruitment may play a role in those with more subtle forms of instability.[78,113] This has been validated by Warner et al.,[266] who demonstrated that patients with shoulder instability had altered rotator cuff strength patterns compared with normal controls. Asynchronous contraction of the rotator cuff, leading to voluntary instability, is an example in the extreme of the relative importance of the rotator cuff in enhancing dynamic stability of the glenohumeral joint.[214] Conversely, capsuloligamentous insufficiency could subject the rotator cuff to overuse, fatigue, and injury.

The importance of these findings is that rotator cuff–strengthening programs can improve the function of a weak or ineffective cuff by limiting translation of the humeral head on the glenoid during active shoulder motion.[39] Initial therapeutic approaches to shoulder instability, therefore, should emphasize strengthening, conditioning, and coordination of the rotator cuff as an integral part of the treatment program.

Ligament Dynamization

There appear to be direct connections between the rotator cuff tendons and the capsuloligamentous system.[45,64] Clark et al.[45] reported a complex anatomic relation between the tendons of the rotator cuff and the capsule adjacent to the humeral tuberosities. The joint capsule is adherent to the rotator cuff, except anterosuperiorly in the

rotator interval, found between the free margins of the suppaspinatus tendon superiorly and subscapularis tendon inferiorly. Conceptually, active shoulder motion may "dynamize" the capsule and ligaments, thereby becoming a significant stabilizing factor in the midranges of rotation at which the ligaments and capsule are relatively lax. Warner et al.,[258] in a dynamic shoulder model, were able to define and document the orientation and interrelation between the glenohumeral ligaments during simulated rotator cuff contraction. Although this study clearly elucidated the effect of shoulder rotation on the orientation of the undisturbed and intact capsuloligamentous system, the dynamic effects of rotator cuff contraction upon the ligaments remains unclear.

Pagnani et al.[189] suggested that because the biceps inserts into the relatively mobile superior labrum, it is conceivable that tension would be transmitted by the labrum to the SGHL and MGHL to dynamize these static structures and indirectly enhance stability. A similar relation may exist owing to the proximity of the triceps to the medial aspect of the axillary pouch of the IGHLC.[45,51] Anterosuperiorly, the subscapularis and supraspinatus interconnect with the CHL, providing an additional site for dynamic interaction between static and dynamic restraints.

Active rotation may also have the effect of altering capsular tension, potentially providing a protective mechanism against failure. For example, coupled posterior humeral head translation with active external rotation may actually reduce anterior ligamentous strain.[85,94] McKernan et al.[152] and others[40,211] have shown in cadaveric experiments that contraction of the posterior rotator cuff muscles (i.e., infraspinatus and teres minor) and biceps tendon reduced IGHL strain in the late cocking phase of throwing. These dynamic factors may provide relative protection of the IGHLC or other anterior structures as they contribute to anterior stability by dynamically increasing the resistance to torsional forces in the position of apprehension. Recently, however, the role of the infraspinatus, as determined by electromyographic (EMG) analysis in patients with recurrent anterior instability, was not believed to be a critical component in providing anterior stability.[96] Clinically, that a stabilizing relation may exist between the capsule and musculature about the shoulder signifies the importance in reestablishing length–tension relations by either operative or nonoperative means in patients with shoulder instability.

the dynamic-stabilizing role of the long head of the biceps brachii for the glenohumeral joint.[3,78,103,106-108,121,162,189,203,211] Rodosky et al.[211] showed that, in the late cocking phase of throwing, contraction of the biceps tendon can significantly reduce anterior translation and increase torsional rigidity of the joint helping to resist external rotation. Additionally, strain in the IGHL was noted to increase after sectioning of the tendon.

Pagnani et al.[189] determined that the effect of the long head of the biceps is dependent on the shoulder position being greatest in middle and lower elevation angles. The biceps tended to stabilize the joint anteriorly when the arm was internally rotated and served as a posterior stabilizer when the humerus was externally rotated (Fig. 9-9). Itoi et al.[107] found that anteroposterior translation was significantly decreased with biceps loading, particularly with external rotation. Superoinferior translation was also reduced with simulated contraction of the biceps, which was believed to help center the humeral head on the glenoid, thereby stabilizing the fulcrum and allowing more efficient arm elevation. Levy et al.[137] emphasized through dynamic EMG analysis that elimination of elbow flexion or supination resulted in complete inactivity of the biceps brachii. Thus, the role of biceps function at the shoulder is either due to a passive mechanism or depends on tension developing in association with elbow and forearm activity.

Kim et al.[121] conducted a thorough EMG analysis of the biceps brachii muscle in patients with anterior instability. The voltage of the biceps muscle was significantly greater in the unstable shoulder compared to the opposite arm in all positions of the arm. Moreover, activity increased in abduction and external rotation of the unstable shoulder; there was no change in activity in the stable shoulder placed in this position. These findings imply a secondary stabilizing function of the biceps muscle, which compensates for failed primary static restraints.[121]

These concepts may help explain why the biceps tendon or superior labrum may demonstrate lesions in throwers[3,228] and why it is occasionally found to be hypertrophied in the rotator cuff–deficient patient.[146,238] Extreme external rotation loads the long head of the biceps tendon, which predisposes the throwing athlete to biceps or biceps–labrum complex injuries.[126,162,203] Clinically, this suggests that nonoperative treatment of instability or rotator cuff deficiency should be directed at rehabilitation of the biceps brachii in addition to the rotator cuff muscles.

Long Head of the Biceps Brachii

As the tendon of the long head of the biceps passes to its insertion in the supraglenoid tubercle, it occupies an intraarticular position. The relative importance as a dynamic stabilizer probably becomes significant when the rotator cuff or capsuloligamentous structures are overwhelmed. Several experimental studies have demonstrated

Scapular Rotators

Until recently, scapulothoracic motion has been relatively ignored as an important dynamic factor maintaining stability of the glenohumeral joint. The scapular rotators include the following muscles: trapezius, rhomboids, latissimus dorsi, serratus anterior, and levator scapulae. Codman[47] first introduced the concept of "scapulohumeral
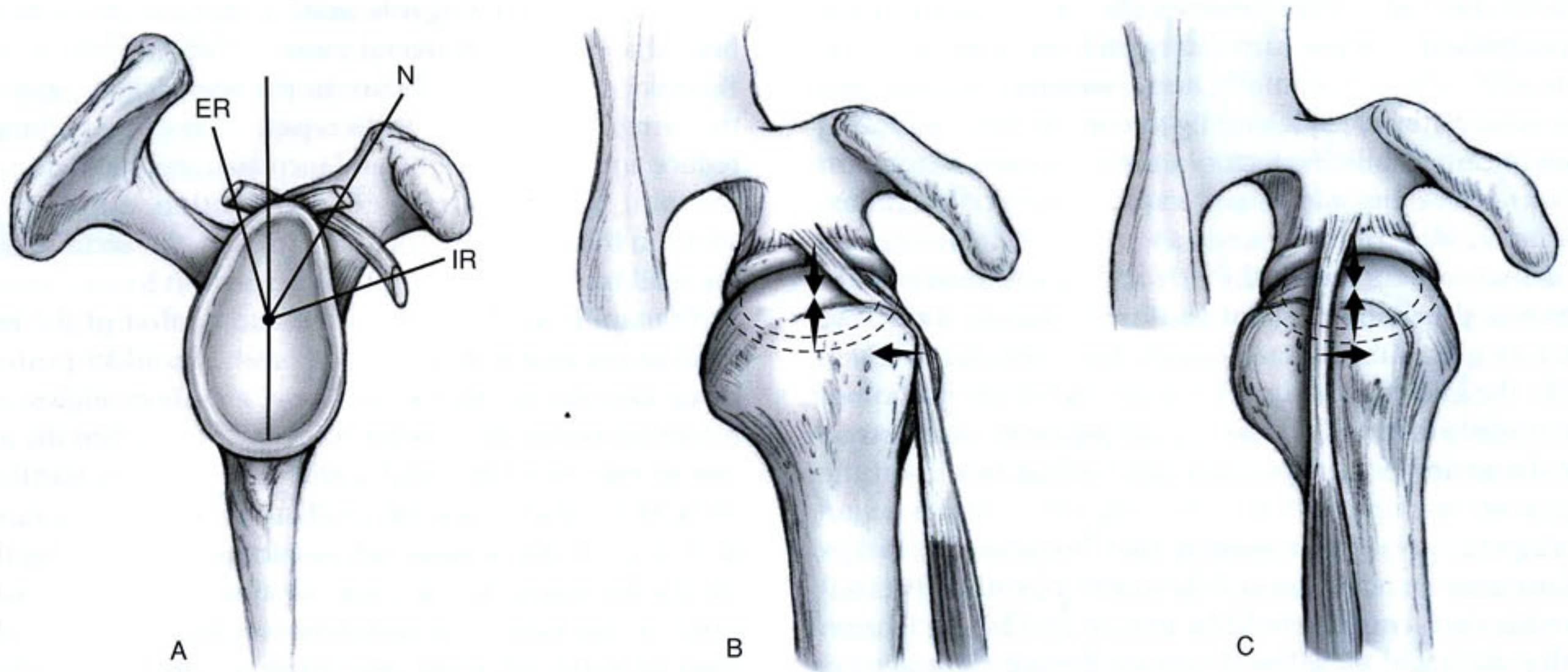
Figure 9-9   Diagrammatic representation of forces created with simulated contraction of long head biceps brachii. **(A)** Rotation of humerus changes orientation of biceps tendon relative to the joint. In neutral rotation (*N*) tendon generally occupies a slightly anterior position. With internal rotation (*IR*) the tendon lies anterior to joint. In contrast, the tendon occupies a slightly posterior position with external rotation (*ER*). **(B)** With internal rotation of humerus, the biceps appears to generate joint compressive forces (*paired arrows*) and posteriorly directed force (*single arrow*), which restrain glenohumeral translation. **(C)** With external rotation of the humerus, anteriorly directed force (*single arrow*) appears to accompany joint compressive forces (*paired arrows*). (Adapted from Pagnani M, Deng X-H, Warren R, Torzilli P, O'Brien S. Role of the long head of the biceps brachii in glenohumeral stability: a biomechanical study in cadavera. *J Shoulder Elbow Surg* 1996;5:255–262.)

rhythm," which has now been recognized by others to be an important contributor to joint stability.[14,105,218,265] Even though somewhat variable, the normal scapulohumeral rhythm motion relation is two of glenohumeral rotation for every one of scapulothoracic rotation during scapular plane abduction.[200,201] Clinical and radiographic studies have documented abnormal scapulothoracic motion in patients with shoulder instability.[188,265] EMG analysis of the scapulothoracic musculature has demonstrated fatigue of the serratus anterior and trapezius with repetitive overhead activities, leading to poor scapulothoracic control.[78,178] McMahon et al. demonstrated that patients with glenohumeral instability have decreased serratus anterior activity during abduction, scaption, and forward flexion.[157]

The scapular rotators function to provide a stable platform beneath the humeral head during shoulder motion (Fig. 9-10). These muscles allow the glenoid to adjust to changes in arm position. The scapular inclination angle is a significant factor preventing inferior translation of the adducted shoulder.[105] For example, the scapula normally rotates upward (i.e., protraction) in synchrony with arm elevation as the serratus anterior contracts. Thus, clinically, scapulothoracic weakness or dysfunction is associated with varying degrees of scapular winging, which is often found in patients with shoulder instability.[265] Warner et al.[265]

have hypothesized that scapulothoracic dysfunction may be a cause of "nonoutlet" impingement, as the advancing greater tuberosity is unable to avoid impingement on the coracoacromial arch during forward flexion. Presently, however, it is unclear if scapulothoracic dysfunction is a cause or product of shoulder instability. Despite these unanswered questions, nonsurgical management of shoulder instability must include rehabilitation of the scapular rotators.

The roles of the deltoid and pectoralis major muscles about the glenohumeral joint have been evaluated in the last several years. Kido et al.[120] evaluated the stabilizing function of the anterior, middle, and posterior deltoid muscle in normal shoulders and in shoulders with instability. In normal shoulders, tension on the middle deltoid reduced the amount of anterior translation substantially. When the joint capsule was vented, or when there was a simulated Bankart lesion, loading each of the three segments of the deltoid muscle decreased anterior displacement. The authors concluded that the deltoid muscle is an anterior stabilizer of the glenohumeral joint with the arm in the position of apprehension, and that this function may become more important in the unstable shoulder.[120]

Lee and An[131] similarly found that deltoid muscle activity increases glenohumeral joint stability. However, this
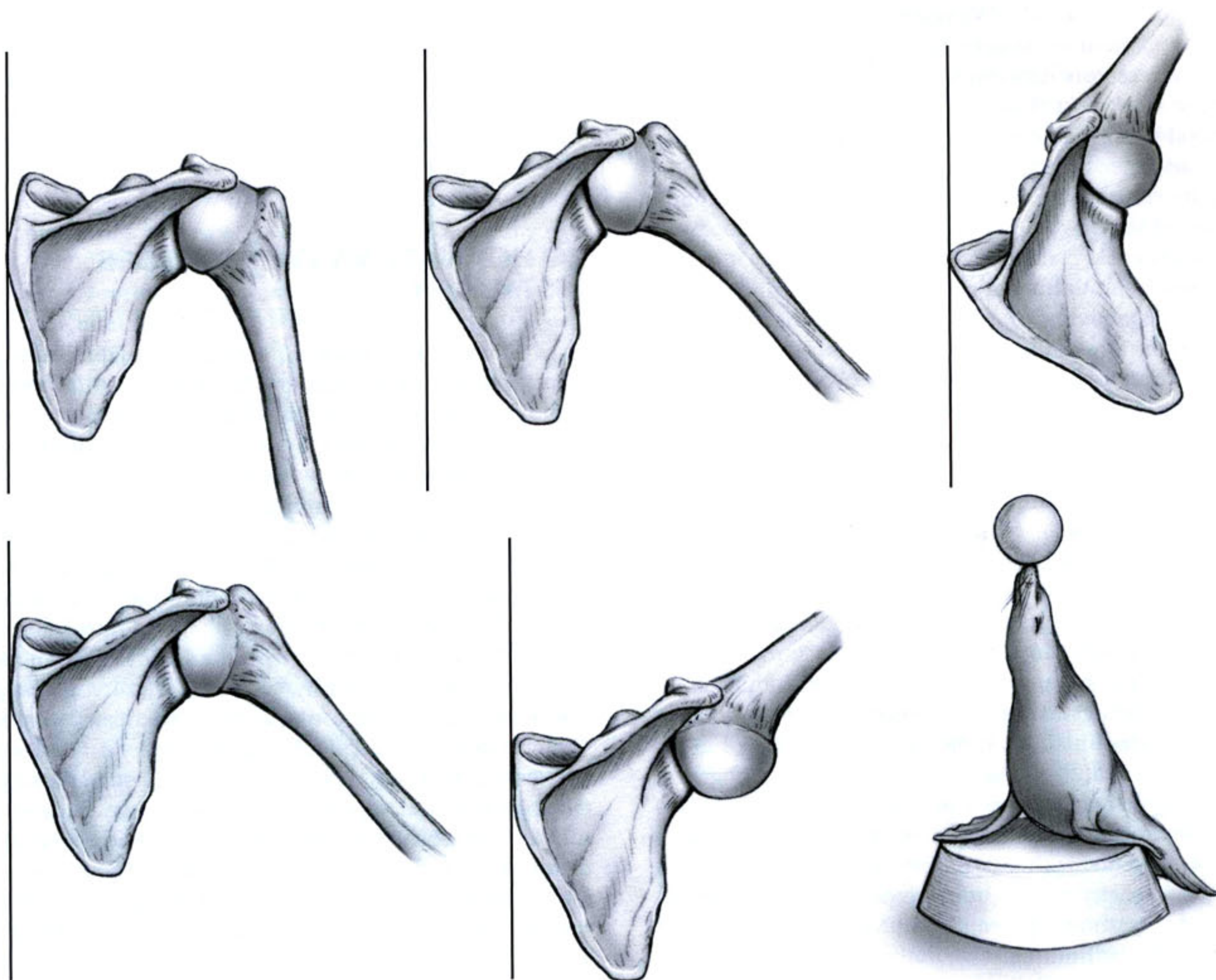
Figure 9-10 **(Top row)** Normal scapulothoracic rotation positions the glenoid underneath the humeral head so that it acts as a stable platform. **(Bottom row)** Failure of proper scapulothoracic motion results in loss of the stable glenoid platform underneath the humeral head. This is analogous to a seal balancing a ball on its nose. (Adapted from Warner JJP, Caborn DNM. Overview of shoulder instability. *Crit Rev Phys Rehabil Med* 1992;4:145–198.)

effect was most pronounced at 60 degrees of glenohumeral abduction in the scapular plane. Conversely, deltoid activity decreased glenohumeral stability when the glenohumeral joint was abducted to 60 degrees in the coronal plane. The role of the pectoralis muscle on glenohumeral joint stability is less clear, though Arciero and Cruser[7] reported a case of traumatic glenohumeral dislocation and pectoralis tendon rupture while bench pressing. They theorized that eccentric loading of the pectoralis led to both injuries. Sinha et al.[227] reported an irreducible glenohumeral dislocation that was successfully reduced only after paralysis of the pectoralis major with botulinum A toxin. Management of glenohumeral joint stability may be altered to consider these muscles as their role about the glenohumeral joint becomes clearer.

Proprioception

The perception of joint position and joint motion is termed *proprioception*. Proprioceptive interaction between ligaments and muscles may mediate a protective mechanism against capsular failure and instability.[25,42,123,135] Murakami et al.[167] described what were thought to be mechanoreceptors in the transition zone between the labrum and capsule in primates. Since then, others[112,251,252] have described similar findings in the capsule and ligaments of the glenohumeral joint.

Mechanoreceptors are specialized nerve endings (e.g., pacinian corpuscles, Ruffini endings, and Golgi tendon-like endings) that transduce mechanical deformation into electric signals that transmit information about joint

position and motion.[79,80] Vangsness et al.[252] evaluated the capsuloligamentous structures for the presence of these mechanoreceptors: Low-threshold, slow-adapting Ruffini afferents were most abundant overall, except in the glenohumeral ligaments where low-threshold, rapid-adapting pacinian-type afferents were more numerous. No mechanoreceptors were observed in the subacromial bursa or glenoid labrum.

Lephart et al.[135] and Warner et al.[264] have hypothesized that the capsuloligamentous structures may contribute to stability by providing an afferent feedback for reflex muscular contraction of the rotator cuff and biceps. It is plausible, as these authors discuss, that as these receptors respond to tension changes in the capsule during rotation, active stabilization may occur through reflex arcs from the capsule to the surrounding rotator cuff, allowing selective contraction of the rotator cuff and biceps muscles in response to changes in acceleration.

Several studies have found decreased proprioception in shoulders with instability.[13,25,135,136,168,243] Capsuloligamentous disruption combined with proprioceptive deficits contribute to functional instability.[136,243] Barden et al.[13] measured hand position error in patients with multidirectional instability (MDI) and compared this to patients without instability. The subjects with MDI had significantly greater hand position error than the control group. Interestingly, there was no difference between hand position error in the symptomatic arm and the contralateral arm in patients with MDI. The authors inferred that patients' capacity to use proprioception to refine upper-extremity movement is reduced in MDI.[13] Blasier et al.[25] reported similar findings.

With use of a specialized proprioception testing device, Lephart et al.[135] evaluated subjects with and without traumatic anterior instability preoperatively and postoperatively after arthroscopic or open Bankart repairs. In normal shoulders, the threshold to detect passive motion (TTDPM) averaged 1.5 to 2.2. In those with instability, TTDPM was 2.8. These differences were statistically significant. Postoperatively, patients' TTDPM was no different from normal. One criticism of this study was that the speeds at which patients were tested were much slower than the speeds that occur with overhead sports. Ito et al.[109] has stated that traumatic dislocation may, in fact, occur sooner than the response time of the rotator cuff stretch reflex. Thus, the relative importance of this proposed mechanism may be more significant in lower-energy situations when the rotator cuff or biceps has time to react to relative changes in capsular tension. Furthermore, it is postulated that proprioception may be a way to protect the capsuloligamentous structures from failure owing to repetitive microtrauma, leading to excessive translation or instability.[135,264]

Whether inherent deficits in proprioception predispose a patient to glenohumeral instability or instability reduces proprioceptive capacity remains unclear. It is interesting to find that surgical procedures that retension the capsuloligamentous structures improve glenohumeral joint proprioception.[135,202,243] One long-term follow-up study reported improvements in joint position sense for at least 5 years postoperatively; position sense may be comparable to normal, healthy shoulders.[202]

PATHOANATOMY OF SHOULDER INSTABILITY

In addition to the "essential lesion" (i.e., labral detachment), recurrent instability has been attributed to several pathologic entities. As indicated in Table 9-2, each of the factors already discussed plays a role in the pathogenesis of shoulder instability. Several authors have cited attenuation of the capsule and capsular ligaments[21,159,164,199,245,246] with associated histopathologic changes.[135,150,198,210,252] Impression fracture of the humeral head (Hill-Sachs or reverse Hill-Sachs lesion),[50,93,153] attenuation of the subscapularis tendon,[59,71,236] capsular rupture,[115,206,208] and humeral avulsion of the glenohumeral ligaments are other documented causes of anterior instability.[10,31,223,235,237,268,276] A thorough history is important to ascertain the mechanism of injury. A single violent trauma will focus the injury on a specific anatomic region (e.g., Bankart lesion or capsular damage). On the other hand, repetitive microtrauma may cause more subtle capsular stretch, emphasizing the pathoanatomic continuum of capsular injury. Clinically, appreciation of the pathoanatomy of shoulder instability is important because surgical intervention is ultimately directed at anatomic and biomechanic restoration.

Bankart Lesion

The most common form of shoulder instability is recurrent anterior subluxation or dislocation resulting from trauma. Perthes[195] and Bankart[12] (e.g., Perthes-Bankart lesion) originally described the detachment of the capsulolabral complex from the glenoid rim and scapular neck as the "essential lesion" leading to recurrent anterior dislocation. This has been challenged by Speer et al.,[232] who found that simulation of the Bankart lesion in cadaveric cutting studies resulted in only minimal increases in anterior translation. Baker et al.[11] established a classification system based on arthroscopic findings of initial anterior shoulder dislocations. Sixty-two percent had evidence of a Bankart or equivalent lesion, with all of these patients demonstrating gross instability with examination under anesthesia. Thirteen percent were stable on examination and demonstrated no evidence of labral detachment. Taylor and Arciero[237] studied first-time patients with traumatic anterior shoulder dislocations and determined arthroscopically that 97% had evidence of isolated detachment of the capsuloligamentous complex from the glenoid rim and neck, without evidence

of intracapsular injury. Others have noted a similarly high incidence of Bankart lesions at the time of surgery.[239]

Despite experimental and clinical evidence of increased anterior translation of the humeral head on the glenoid caused by a Bankart lesion, most patients present with recurrent anterior instability with additional pathology that may have developed or advanced over time. Additionally, plastic deformation or capsular injury in patients with first-time dislocations may not be appreciated by macroscopic evaluation because it may represent microscopic ultrastructural failure not visible to the naked eye. It is now believed that recurrent complete dislocation requires an additional pathoanatomic component (e.g., capsular plastic deformation or stretch).[12,21,213,232]

Recognition of this concept is clinically relevant. Isolated arthroscopic Bankart repair is technically challenging and with earlier reports associated with higher failure rates, possibly because of associated pathology, poor patient selection, and minimal scar formation.[6,53,129,160,233,254] Moreover, reduction in anterior translation, which is the goal of the procedure, is only significant with larger imbrication (5 mm) of the capsule. Larger imbrication leads to a more severe limitation of external rotation.[177] Conversely, open Bankart procedures that address "only" the labral detachment may create enough capsular scarring to prevent recurrence. Thus, the choice of operative procedure will depend on the patient's history, examination under anesthesia, arthroscopic anatomy, and appreciation for capsular injury. These tenants become more complex in the patient with multidirectional instability in whom capsular laxity is the dominant pathology, and Bankart lesions are less frequently found.

Capsular Injury

Traumatic Intrasubstance Injury

Clinical observations indicate that capsular injury is commonly associated with traumatic anterior shoulder dislocation. Capsular injury resulting from traumatic anterior shoulder dislocation was recognized as early as the 13th century. Reeves demonstrated capsular rupture by arthrography in 55% of the anterior dislocations he treated.[206,208] Symeonides[236] observed that 15% of his patients treated for anterior dislocation had both labral detachment and anterior capsular ruptures. Johnson[115] observed that 54% of his patients at the time of arthroscopy for anterior dislocation had torn glenohumeral ligaments. Conversely, others have shown that only minimal irrecoverable elongation of the inferior glenohumeral ligament occurs after traumatic unidirectional dislocations.[155,156,158,234]

Experimentally, Bigliani et al.[21] measured the stress–strain data at failure of the IGHLC in bone–IGHL–bone preparations and concluded that before failure, significant plastic deformation (e.g., strain) occurred. The implications of

these findings are that laxity of the IGHLC leading to instability is not only a congenital finding, but that it may be acquired through submaximal trauma (single or repetitive) without causing rupture or detachment. When the anterior shear force overcomes the capsular tensile strength or when the rotator cuff fatigues or cannot effectively contract (e.g., rotator cuff tears), the ligaments may fail on an ultrastructural level.[135,198,252] Rodeo et al.[210] provided evidence of ultrastructural changes in the joint capsules of unstable shoulders. In joint capsules from patients with instability, there are increases in the amount of stable and reducible cross-links (the latter of which is abundant in remodeling tissue) as well as the mean collagen fibril diameter (which correlates positively with tissue strength) compared to patients with stable shoulders.[210] Others have shown histologic changes in shoulder capsules of patients with traumatic instability, such as a denuded synovial layer, subsynovial edema, increased cellularity, and increased vascularity.[150]

Similarly, age-related attrition of the rotator cuff tissues is greater than in capsular tissues such that anterior dislocation commonly results in a rotator cuff tear, potentially leading to capsular injury in older patients.[207] Gamulin et al.[71] evaluated the histomorphometry of the subscapularis muscle in 52 patients operated on for recurrent traumatic anterior shoulder dislocation. They observed interstitial fibrosis within the subscapularis muscle and modifications in the ratio of fiber types that are characteristic of disuse atrophy. Clinically, capsular injury leading to laxity and labral detachment can be found to coexist, supporting concomitant capsulorrhaphy and Bankart repair in the surgical management of instability. Bigliani et al.[19] and Altchek and Dines[2] have advocated addressing capsular pathology as part of the surgical treatment of recurrent anterior shoulder instability.

Humeral Avulsion

First described in 1942 by Nicola,[173] avulsion of the capsule from the humerus can occur with forceful hyperabduction. Disruption of the lateral capsule from the humeral neck is probably rare, but has been reported in two cases by Bach et al.[10] and in one case by Taylor and Arciero[237] in association with anterior dislocation. Wolf et al.[276] has termed this a "HAGL lesion" representing humeral avulsion of the glenohumeral ligament. Appreciation for this variant of capsular injury at the time of arthroscopy can be difficult, though these authors advocate searching for it in patients with traumatic anterior instability who show no signs of a Bankart lesion.[276]

The HAGL lesion appears as a thickened, rolled edge of capsular defect, typically found in the inferior pouch of the shoulder below the level of the subscapularis muscle.[31] Associated glenohumeral abnormalities are common, most often in the form of rotator cuff tears; greater than 90% of these tears involve the subscapularis muscle.[10,31,223,268,276] Only 20% of HAGL lesions may be viewed radiographically.[10]

If it is visualized it likely represents a bony HAGL (BHAGL) lesion, which may mimic a bony Bankart lesion.[179] The BHAGL lesion, first described by Bach et al.,[10] is a HAGL lesion associated with bony avulsion of the humeral neck. Oberlander et al.[179] recommended evaluating such lesions with axillary or West Point views of the glenohumeral joint. In either of these views the BHAGL is seen superimposed on the proximal humerus, whereas the bony Bankart lesion is seen along the inferior half of the glenoid cavity. Stoller[235] described the J sign of the HAGL lesion on magnetic resonance arthrography, whereby the axillary pouch changes from a fluid-distended U-shaped structure to a J-shaped structure. This is attributed to inferior displacement of the anterior band of the inferior glenohumeral ligament.[235] Humeral avulsion of the glenohumeral ligament should be repaired anatomically at the time of surgical reconstruction.

Repetitive Injury

The overhead athlete (e.g., pitchers, throwers, swimmers, volleyball players, tennis players, water polo players, and javelin throwers) represents a special category of patients with complaints relating to instability. These patients subject their shoulder to repetitive stresses that potentially lead to microtrauma not readily appreciated at the time of arthroscopy. In a novel study, Pollock et al.[199] evaluated the response of the IGHL to a range of cyclic deformations and different levels of strain in an attempt to identify mechanical microdamage caused by repetitive loading. They found a significant decrease in the residual strain magnitude of the IGHL after cyclic loading compared to baseline. It was concluded that the cumulative effect of repetitive subfailure strain causes irreversible stretching of the IGHL. This may contribute to the development of shoulder instability.[199] Malicky et al.[142] also identified irreversible changes in strain magnitude of the anteroinferior capsule after 16 mm of humeral translation.

Repetitive rotational motion of the glenohumeral joint may also contribute to instability. Remia et al.[209] reported on an experimental model of multidirectional instability of the glenohumeral joint and found that application of internal and external rotational stretches to the capsule causes increased translation in all directions, without capsular disruption. Likewise, Mihata et al.[159] found increased shoulder laxity after nondestructive stretching of 30% beyond maximal humeral external rotation. This was attributed to a significant lengthening of the anterior band of the IGHL. Repetitive injury may be the cause of acquired laxity, as seen in gymnasts, and may present as multidirectional instability rather than pure unidirectional instability. These patients are often confused as having isolated subacromial impingement and inappropriately treated as such.[69,241] It is now believed that "subtle glenohumeral instability" may be associated with secondary subacromial impingement, and capsular laxity is the primary pathology that should be addressed.[242]

Capsular Laxity

Intrinsic Capsular Laxity

Capsular laxity is a prerequisite to allow a large range of glenohumeral motion. The degree of laxity varies among individuals, and attempts at correlating the extent and direction of laxity under anesthesia can be confusing, for the overlap between normal laxity and clinical instability is difficult to ascertain.[48,62,73,85,86,184,248,266] Interestingly, shoulders of asymptomatic patients can exhibit a range of rotational or translational motion comparable to that seen in patients diagnosed with symptomatic instability.[85,89,89] In the preadolescent shoulder joint, more than two-thirds may be asymptomatically subluxated on examination.[62] That asymptomatic subluxation or even dislocation may occur in the "normal" shoulder at the time of anesthesia was also appreciated by O'Driscoll and Evans[184] and Warner et al.[262] Thus, it is unclear if constitutional laxity is a risk factor for clinical instability of the shoulder joint. With a proper history of the mechanism and symptoms, correlation with drawer testing under anesthesia can be useful if one considers the effect of arm position on different portions of the capsule.

Inherited Disorders of Collagen

Inherited disorders of collagen are relatively rare, but present an unusual challenge in the management of glenohumeral instability. The collagen disorder most associated with shoulder instability is Ehlers-Danlos syndrome (EDS), which is characterized by increased laxity, problems with wound healing, and vascular anomalies. Several subtypes of EDS have been identified based on which type and which synthetic step of collagen is aberrant. EDS I is inherited in an autosomal dominant pattern and is most commonly associated with hyperlaxity. EDS II is a milder form of EDS I. The other subtypes of EDS predominantly affect the blood vessels. A survey of 42 patients with EDS revealed that they had experienced a combined 214 shoulder procedures. The indications were pain, instability, poor range of motion, or a combination of these.[89] Another case report describes a 19-year-old female with EDS and bilateral multidirectional shoulder instability who underwent multiple operations to achieve stability, all of which were ineffective.[149] Thus, shoulder instability with EDS remains a difficult area to manage even with contemporary techniques.

Humeral and Glenoid Bone Loss

Humeral Bone Loss

Articular abnormalities of the humeral head can disrupt the anatomic relation of the glenohumeral joint, predisposing to recurrent instability. In an evaluation of radiographs from 160 patients with chronic anterior shoulder instability, Edwards et al.[61] identified humeral impaction
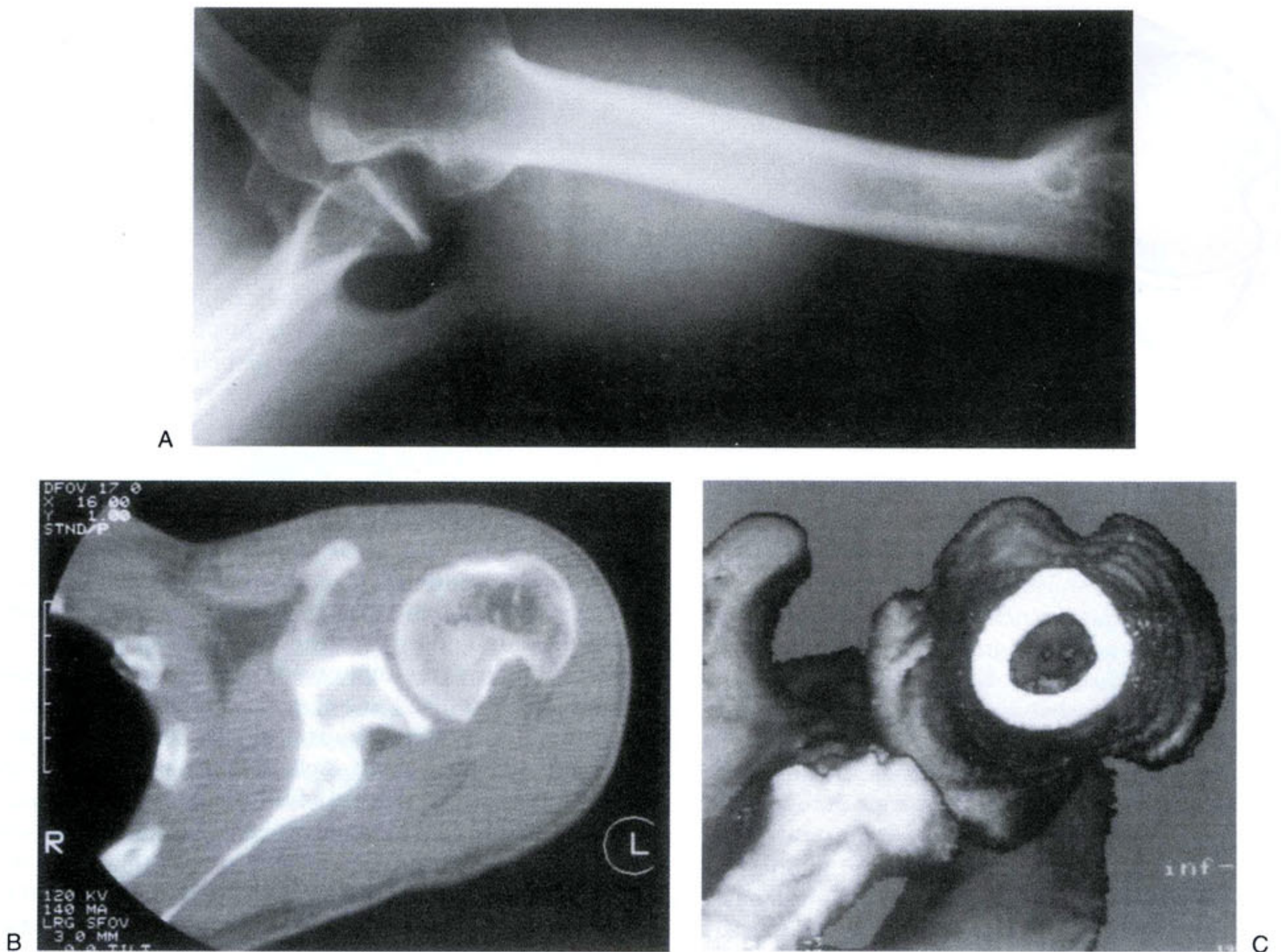
Figure 9-11 **(A)** Anterior dislocation in a patient with a large Hill-Sachs lesion (more than 30% of the humeral head). **(B)** Computed tomographic (CT) scan in the same patient. **(C)** A three-dimensional CT reconstruction also shows a large Bankart lesion in addition to the large Hill-Sachs lesion.

fracture in 117 shoulders (73%). A large Hill-Sachs or reverse Hill-Sachs impression fracture (Fig. 9-11) on the posterolateral or anterolateral margin of the humeral head, respectively, is created when the humeral head dislocates over the anterior or posterior glenoid rim.[41,194,213,216] This lesion is present in more than 80% of anterior dislocations and 25% of anterior subluxations.[41,194] Hill-Sachs lesions have been noted at the time of arthroscopy in patients with recurrent anterior instability up to 100% of the time.[41,176,237] Werner et al.[273] reported a prevalence of Hill-Sachs lesions approaching 60% in patients with atraumatic instability that did not respond to conservative therapy. The small Hill-Sachs lesions are not usually thought to be a major contributor to recurrent anterior instability.[41] The incidence with posterior instability is unknown.

The Hill-Sachs lesion is larger with dislocations of longer duration, recurrent dislocations, and inferior displacement of the humeral head.[66,92] In most instances this lesion is relatively small and plays little role in ongoing shoulder instability or its surgical management. Relatively

small lesions may be prevented from coming into contact with the anterior glenoid rim simply by performing a more generous anterior capsulorrhaphy (Fig. 9-12). Caution is warranted with this practice because excessively tight anterior repairs may be associated with the development of late arthrosis.[18,22] However, when the Hill-Sachs lesion involves more than 30% of the humeral articular surface, it may contribute to recurrent anterior instability, even with capsular repair.[215,216]

The mechanism for this persistent instability is that with increasing external rotation, the lesion slips over the anterior glenoid (i.e., as in the original injury) and sits in an anteromedial position outside of the glenoid cavity. Surgical treatment of these defects involves filling the defect with allograft bone (Fig. 9-13),[74,114] muscle tendon transfer,[50,66,153] (e.g., infraspinatus or subscapularis), or humeral head replacement in older individuals. Alternatively, the lesion can be rotated out of contact with the glenoid with proximal humeral osteotomy.[270] Decision making is predicated on the ability to perform an anatomic
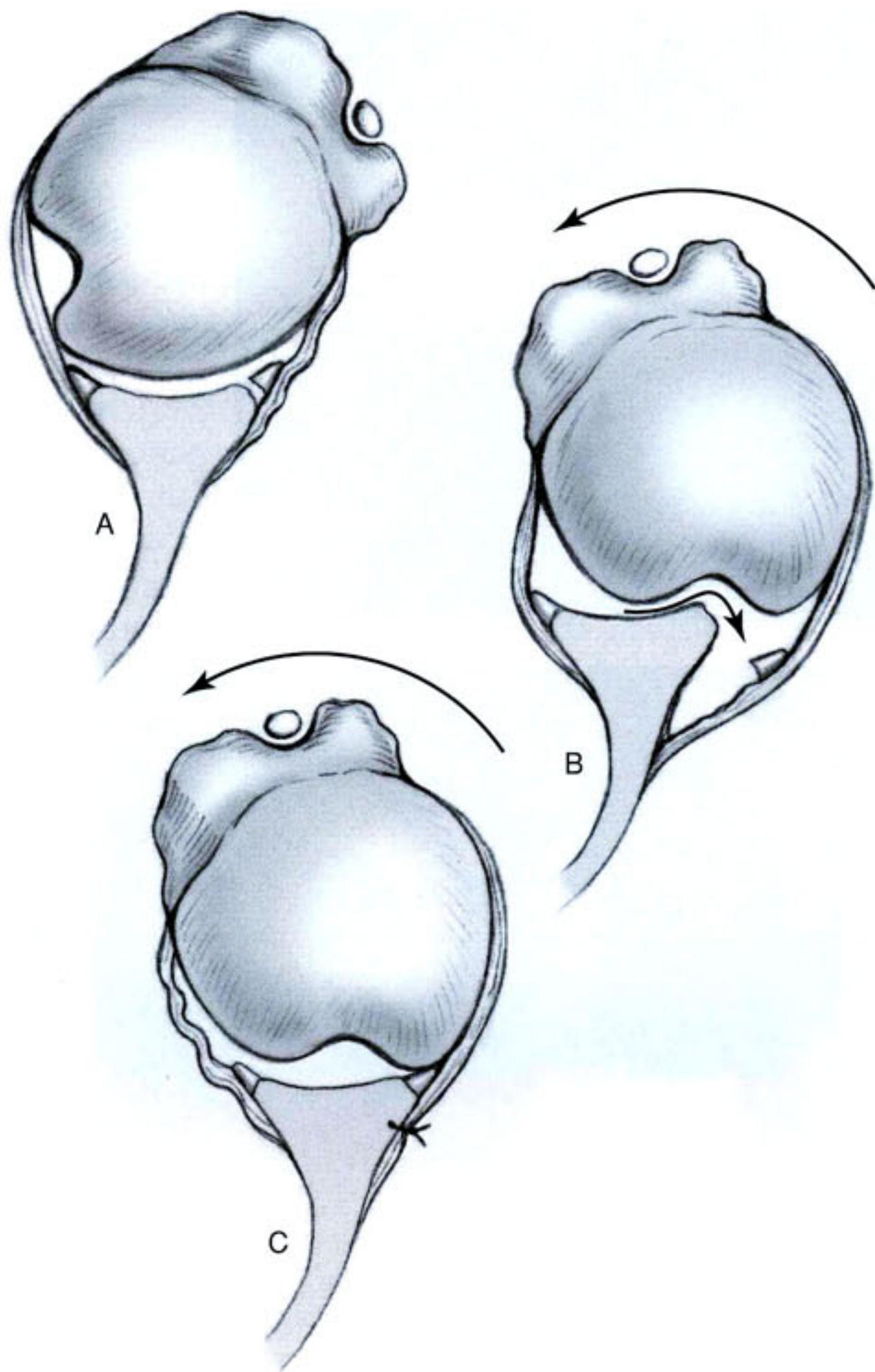
Figure 9-12 Role of the Hill-Sachs lesion in anterior shoulder instability. **(A)** With the arm in internal rotation, the Hill-Sachs lesion is not in contact with the glenoid. **(B)** With external rotation, the humeral head translates anteriorly because of the incompetent anterior capsular mechanism. This allows the humeral head to dislocate through the Hill-Sachs lesion. **(C)** An adequate Bankart repair keeps the Hill-Sachs lesion contained on the glenoid, unless it is greater than 30% of the humeral articular surface. (Adapted from Warner JJP, Schulte KR, Imhoff AB. Current concepts in shoulder instability. In: Stauffer RN, Erlich MG, Kostuik JP, Fu FF. *Advances in operative orthopaedics*, vol. 3. Philadelphia: Mosby-Year Book, 1995;217–247.)

reconstruction of the anteroinferior structures with an appropriate capsular plication followed by reassessment for the ability to engage the Hill-Sachs lesion in various positions of rotation with a simultaneously applied anterior force to the proximal humerus.

Glenoid Bone Loss

Glenoid bone loss has been implicated as a predisposing factor for recurrent instability after surgical repair.[24,104,247] Burkhart et al.[38] observed that glenoids with enough bone loss to convert the normally pear-shaped glenoid to an

inverted pear configuration are at particular risk for redislocation after surgical repair. Recent work by Gupta and Lee[81] offers a cause for posterior erosion of the glenoid. They evaluated glenoid–humerus contact forces in 12 overhead activity positions and found that there is a significant increase in contact pressure between the humeral head and posterior glenoid when the humerus is horizontally abducted to 70 degrees. They concluded that repetitive overhead activities may load the glenohumeral joint asymmetrically and lead to posterior glenoid erosion.[81]

Bony lesions of the anterior or posterior glenoid rim have also been described and are believed by some to be important enough to be formally reconstructed during open capsulorrhaphy. Edwards et al. found osseous lesions of the glenoid in 126 of 160 (78%) patients with chronic anterior glenohumeral instability.[61] These were seen on the glenoid profile view. These lesions are either due to an osseous Bankart or wear related to repeated instability. Pavlov et al.[194] described an osseous Bankart lesion of the anterior glenoid in 15% of patients with recurrent anterior dislocation and in approximately 50% of patients with recurrent anterior subluxation. Gerber[75] has advocated intraarticular iliac bone graft to formally reconstruct the glenoid cavity before capsular repair to restore normal anatomy of the glenoid. Bigliani et al.[24] believes that compromise of 25% or more of the glenoid surface warrants bony reconstruction. Burkhart et al.[38] recommend a coracoid process transfer (i.e., Latarjet procedure) when a bony Bankart lesion narrows the inferior half of the glenoid to a width that is less than that of the superior half of the glenoid (i.e., the inverted-pear configuration).

Defects smaller than 20% can be rendered extraarticular by repairing the capsule and labrum back to the edge of the intact glenoid. Larger fragments can be mobilized and fixed through traditional means.[33] Unlike the Hill-Sachs lesion, there are few data available to suggest which glenoid defects require repair, débridement, or neglect. Itoi et al.[104] investigated the effect of glenoid defect size on anteroinferior stability after Bankart repair. These authors found that with the arm in the position of apprehension, the size of the osseous defect does not affect stability of the arm. However, increasing the size of the osseous defect reduces stability when the arm is placed in abduction and internal rotation.[104] The overall aim of any reconstructive procedure directed at larger defects is to deepen the socket and support the capsule.

Articular Version Abnormalities

Clinically, excessive glenoid retroversion is thought to be a contributing factor to posterior instability and may infrequently be due to a variant of glenoid dysplasia. In most cases, however, excessive version is acquired from eccentric articular surface wear. Magnetic resonance imaging reveals that shoulders with posteroinferior instability have greater
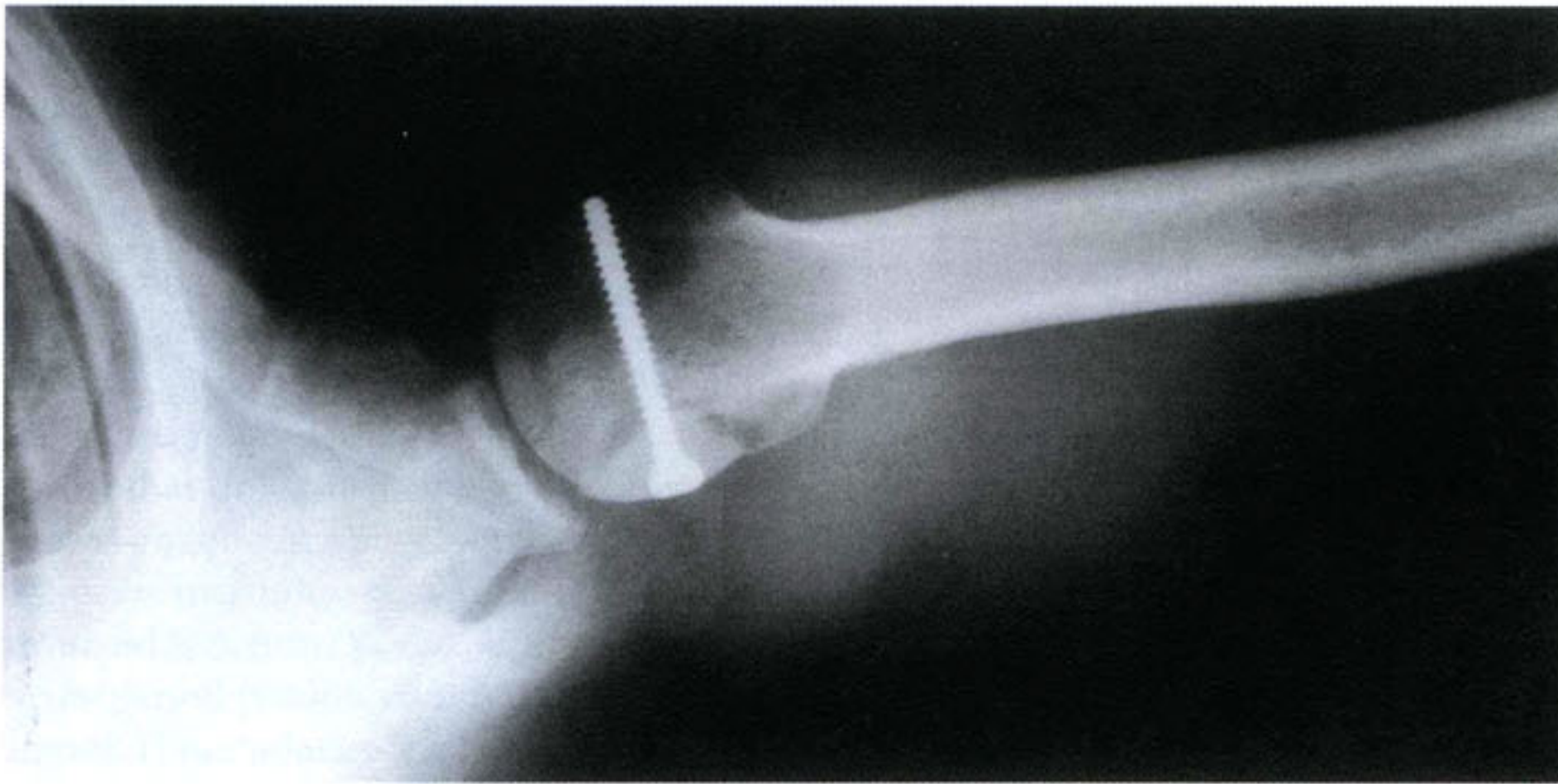
Figure 9-13 This large Hill-Sachs defect was treated with a Bankart repair and osteochondral allograft reconstruction of the humeral head.

retroversion of both the osseous and chondrolabral portion of the glenoid, and there is loss of height of the posterior portion of the labrum.[122] These features lead to loss in chondrolabral containment of the glenohumeral joint in patients with posteroinferior instability of the shoulder.[122]

Some surgeons recommend glenoid osteotomy in addition to soft tissue procedures.[36,125,225] Several other authors[125,205] have reported varying degrees of normal glenoid and humeral articular version, indicating that further study is needed to support a relation between the development of instability and bony alignment. Glenoid osteotomy and rotational humeral osteotomies, seemingly reasonable treatment options in the presence of articular version abnormalities, have been associated with the development of glenohumeral arthritis.[99,116] Currently, in North America, humeral rotational osteotomy or glenoid osteotomy is not commonly practiced, perhaps reflecting the unclear relation between these factors and clinical instability.

PUTTING IT ALL TOGETHER

Successful management of shoulder instability requires a thorough knowledge of all factors responsible for stability in addition to those pathologic factors contributing to instability. Shoulder instability may be viewed as any condition in which the balance of the various stabilizing structures is disrupted, leading to increased joint translation and the development of clinical symptoms.[89,191] Because the large spherical head of the humerus articulates with a relatively small and shallow glenoid, the glenohumeral joint requires several mechanisms to maintain stability while providing for a large range of motion. Static and dynamic stability is provided by the combined effects of the capsuloligamentous structures and rotator cuff and biceps. In the midranges of rotation, where the capsuloligamentous structures are lax, most joint stability is through the dynamic action of the rotator cuff and biceps tendons through

concavity–compression of the humeral head within the glenoid socket. The ligamentous structures, which are primarily capsular thickenings, function only at the extreme positions of rotation, preventing excessive rotation of the humeral head on the glenoid. Contraction of the muscles around the shoulder may act secondarily by protecting the relatively weak ligamentous structures from being overwhelmed from excessive tension. Because interpretations of the literature are often confusing, this section is an effort to synthesize the findings already discussed.

The labrum provides an attachment site for the glenohumeral ligaments and the tendon of the long head of the biceps. Its principal function is to increase the depth of the glenoid socket and to act as a chock block in preventing the head from rolling over the anterior edge of the glenoid. Recently, however, the role of the labrum in preventing translation or instability has been challenged. The Bankart lesion, by its anatomic definition, implies dysfunction of the IGHLC, and possibly the SGHL and MGHL. Thus, virtually all labral lesions, especially those below the glenoid equator, are thought to be associated with glenohumeral instability. However, plastic deformation, capsular rupture, abnormal laxity, periosteal stripping, or any combination of these lesions may also be associated with complete dislocation, with or without the Bankart lesion. Thus, one of the goals of reconstructive surgery for glenohumeral instability is to anatomically reconstruct both the labral and capsular deficiency independent of cause (e.g., genetic predisposition or extrinsic forces).

The role of the capsule and ligaments in preventing instability is quite complex and depends on shoulder position and the direction of the applied force. Generally, the anterior capsule becomes more important during extension and the posterior capsule during flexion. Extremes of internal and external rotation have the effect of winding up the capsular structures, leading to joint compression and increased stability owing to tension developing in the relevant structures. In general, the inferior capsular structures

are most functional near full elevation and the superior capsular structures near full adduction.

The IGHLC is the primary static check against anterior, posterior, and inferior translation between 45 and 90 degrees of glenohumeral elevation. The SGHL and MGHL limit anteroposterior and inferior translation in the middle and lower ranges of elevation as the arm approaches the adducted position. Experimentally, posterior translation in the flexed, adducted, and internally rotated position may require disruption of the anterosuperior capsule (including the SGHL) in addition to the posterior structures. Although controversial, the SGHL, CHL, and IGHL probably function together to limit inferior translation of the adducted shoulder and act as secondary restraints against posterior translation. Clinically, these structures are addressed during either arthroscopic or open Bankart repair or capsulorrhaphy. However, simply overtightening the capsule to limit the end ranges of motion to achieve stability may lead to pathologic limitation of shoulder motion and late arthrosis.

The rotator interval region between the subscapularis and supraspinatus may be associated with abnormal translation, especially inferior translation of the adducted arm and, possibly, anteroposterior translation. Contraction of the rotator cuff and long head of the biceps brachii affects both static and dynamic factors that enhance stability. Primarily, they act in concert to increase compression across the glenohumeral joint, increasing the loads required to translate the humeral head. These factors are especially important in the midranges of motion where the capsuloligamentous structures are more lax. The long head of the biceps brachii is a significant secondary stabilizer when the capsuloligamentous structures begin to fail. The scapulothoracic stabilizers help accurately time and position the glenoid beneath the humeral head. Dysfunction in any of these stabilizers can lead to subsequent instability as residual stabilizing mechanisms become overwhelmed. Furthermore, proprioceptive mechanisms help to coordinate and time this system and can be restored after instability surgery.

Finally, the effects of abnormal articular surfaces, articular version, negative intraarticular pressure, and adhesion–cohesion, either in part or in combination, can lead to or worsen shoulder instability. By themselves, however, they may play only a small role in the pathogenesis of shoulder instability. Rarely is bone loss significant enough to warrant surgical correction. Unfortunately, clinical data are lacking for most of these factors, and an algorithmic approach to their treatment is currently evolving as experimental models improve.

CLASSIFICATION

The importance of a classification system for shoulder instability is best appreciated from observations of treatment

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TABLE 9-4
SHOULDER INSTABILITY CLASSIFICATION

I. Degree
  A. Dislocation
  B. Subluxation
  C. Subtle
II. Frequency
  A. Acute (primary)
  B. Chronic
    1. Recurrent
    2. Fixed
III. Etiology
  A. Traumatic (macrotrauma)
  B. Atraumatic
    1. Voluntary (muscular)
    2. Involuntary (positional)
  C. Acquired (microtrauma)
  D. Congenital
  E. Neuromuscular (Erb's palsy, cerebral palsy, seizures)
IV. Direction
  A. Unidirectional
    1. Anterior
    2. Posterior
    3. Inferior
  B. Bidirectional
    1. Anteroinferior
    2. Posteroinferior
  C. Multidirectional

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failures resulting from improper matching of a surgical procedure with the appropriate pathology. Careful classification improves our ability to tailor individualized treatment programs for patients with glenohumeral instability. Although other classification systems exist for shoulder instability,[197] a system based on four factors is commonly employed: the degree of instability, the frequency of occurrence, direction, and cause of the instability (Table 9-4).

The degree of instability is proportional to the level of injury to the capsulolabral structures. *Dislocation* is defined as complete separation of the articular surfaces, often requiring a reduction maneuver to restore joint alignment. *Subluxation* is symptomatic instability without complete dislocation of the articular surfaces. These patients may complain of only pain without an appreciation for actual instability.

Subtle degrees of instability may be due to microtrauma, which may occur from overuse, as seen with repetitive overhead throwing. As in those with subluxation, some of these patients present with pain, with no knowledge of underlying instability. Additionally, patients with multidirectional or posterior instability may have associated tendinitis and pain without a sense of actual shoulder instability.[72,217,226] The avid overhead athlete may also present with pain in the posterior aspect (e.g., internal impingement of the posterior rotator cuff on the posterosuperior glenoid) of the shoulder

during the late cocking or early acceleration phase of throwing. Impingement, when present, is due to altered biomechanics and is a secondary phenomenon, rather than true mechanical impingement from the coracoacromial arch.[1,94,255] These can be difficult diagnostic and therapeutic problems.

The frequency of instability is described as acute or chronic. The temporal delineation between acute and chronic is not well defined in the literature, and those definitions that do exist may not have direct clinical relevance. However, for descriptive purposes, an *acute episode* of glenohumeral instability generally refers to the primary dislocation and is defined as one in which the patient is seen in the acute period (within several hours or even a few days) of the injury. These injuries may or may not need to be manually reduced, for occasionally they will reduce spontaneously. *Chronic instability* is in reference to either recurrent episodes of acute instability (a.k.a. recurrent instability) or dislocations that remain displaced for greater periods of time. The latter type may be more appropriately termed fixed or locked dislocations. Most commonly, this is seen in a missed or neglected posterior dislocation.[88]

The cause of instability may be categorized as traumatic, atraumatic, microtraumatic, congenital, or neuromuscular. As our understanding of the pathophysiology of shoulder instability has evolved, we now recognize that simplifying cause into atraumatic or traumatic is somewhat limiting. Thomas and Matsen[239] originally introduced the acronyms TUBS and AMBRI to help us think about the cause and treatment of most patients who have shoulder instability. The TUBS variety of instability describes a patient with macro*t*raumatic *u*nidirectional instability associated with a *B*ankart lesion that typically responds well to *s*urgery. The AMBRI variety of instability describes a patient with *a*traumatic *m*ultidirectional instability that is *b*ilateral and often responds to *r*ehabilitation; rarely, this type of instability requires an *i*nferior capsular shift.

Patients with this atraumatic instability may demonstrate the ability to voluntarily dislocate their shoulders. By selective muscle contraction and relaxation, these patients can position their shoulder to result in subluxation or dislocation. Most commonly, this is seen in cases of posterior and multidirectional instability, but pure anterior instability can also be produced. Rowe et al.[214] have observed that voluntary subluxation can be associated with emotional and psychiatric disorders of secondary gain. In general, this category of voluntary instability has a high rate of recurrence after surgical stabilization if the underlying psychopathology is not addressed.[169,214] A subtype of voluntary instability includes those who have an unconscious behavioral tic leading to selective muscular contraction. This form of voluntary instability may respond best to biofeedback techniques.[15]

Alternatively, dislocation may occur voluntarily with underlying involuntary instability, as with activities or even during sleep owing to instability that is positional. Although patients can voluntarily reproduce disabling instability just by positioning their arm, they prefer not to do so. Most commonly, these patients may have involuntary posterior instability that can be demonstrated by positioning the arm into flexion, adduction, and internal rotation. These patients often adapt by avoiding positions of risk where the shoulder might dislocate.[226] This positional type of involuntary instability, unlike voluntary instability caused by psychiatric factors or a behavior muscular tic, may respond well to surgical stabilization.[20,70]

Neer[170] recognized acquired instability that results from repetitive microtrauma (overuse) to the glenohumeral joint. These patients often provide a history of being an avid overhead athlete (e.g., baseball, swimming, tennis, and such) as they subject their anterior and inferior capsuloligamentous structures to repetitive injury and stretch causing symptomatic instability. These patients are not uncommonly found to have a preexistent constitutional congenital hyperlaxity.[60,62,184] Conceivably, these patients excel at their sport because of this excessive laxity and may develop symptomatic instability through subsequent trauma. Finally, neurologic disorders can lead to instability including stroke, Erb's palsy, and seizures, which can cause both anterior and posterior instability. Thus, rather than a discrete cause, there remains a spectrum of instability, with traumatic and atraumatic mechanisms occupying the extremes. This is reflected by the variety of findings observed at the time of surgery that may include any combination of a Bankart lesion, capsular laxity, or capsular rupture.

The direction of instability can be anterior, posterior, inferior, or any combination of these. Unidirectional instability occurs in only one of these directions. Multidirectional instability as in the AMBRI variety may demonstrate all three directions of instability in addition to generalized ligamentous laxity. The principal direction of instability in both the TUBS and AMBRI varieties of instability is usually anterior. However, the presence of inferior instability is the hallmark of the diagnosis of multidirectional instability. To add to already confusing nomenclature, Pollock and Bigliani[196] and Bigliani et al.[19] have described patients with an intermediate degree of instability, who demonstrate an inferior component in addition to an anterior or posterior component as bidirectional (e.g., anteroinferior or posteroinferior). This type of instability is more common in overhead athletes who expose their anterior and inferior capsular restraints to repetitive microtrauma, leading to plastic deformation and stretch. An additional subtlety is that patients with posterior instability often exhibit smaller degrees of inferior and even anterior instability.

Recognition of global capsular laxity and instability in more than one direction that is due to either of the extremes of causation (e.g., traumatic or atraumatic) is critical for determining appropriate surgical management so as not to exacerbate the instability in the direction left

unaddressed.[169] Often, it is the primary direction of instability that causes most of the patients' symptoms and is, therefore, most commonly addressed surgically. However, procedures that treat anterior capsular laxity by Bankart repair or capsular plication may not adequately manage the associated components of inferior and/or posterior instability. In the extreme, asymmetrical tightening during capsulorrhaphy can lead to a fixed subluxation in the opposite direction.[17,141] Thus, establishing the principal direction of the instability and acknowledging the lesser components by the time of surgical intervention is critical to obtain a successful outcome.

CONCLUSIONS

The aims of this chapter were to review the anatomy, biomechanics, and pathophysiology of shoulder instability. An understanding of what is "normal" provides a foundation for diagnosing and treating what is considered to be pathologic. Because current research endeavors have focused on the basic science of shoulder instability, we no longer have to rely on an anecdotal and qualitative account of the associated pathology noted at the time of treatment. We now have an organized and quantitative approach to the treatment of shoulder instability. Anatomic studies have provided abundant information on the macro- and ultra-structure of the static and dynamic restraints to stability. Experiments examining the biomechanics of shoulder instability have helped clarify the effects of articular version, the labrum, negative intraarticular pressure, the material properties and limits of function of the capsuloligamentous complex, and the dynamic interaction between static and dynamic restraints. There are still several unanswered questions. As technology is advancing, we must continue to evaluate how older and newer techniques correct anatomic and biomechanic abnormalities leading to glenohumeral instability. Newer forms of "heat therapy" and arthroscopic techniques are exciting means to perform less invasive surgery. However, meticulous analysis will be required to determine their value. Our current understanding of anatomy and biomechanics should greatly facilitate this goal.

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