Functional Anatomy and Biomechanics of Shoulder Instability

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KEYWORDS

• Glenohumeral joint • Stability • Biomechanics • Functional anatomy

KEY POINTS

- The glenohumeral joint is composed of static and dynamic stabilizers that allow the humeral head to remain centered on the glenoid.
- Glenoid bone loss can be measured with a variety of methods and should be addressed with a bony reconstruction procedure when bone loss reaches a critical value.
- Humeral bone loss is present in nearly all patients with recurrent dislocations and can be addressed with capsulotenodesis of the posterior cuff (remplissage).

INTRODUCTION

Anterior shoulder instability is a common diagnosis in young athletes. Shoulder stability is the result of a complex interplay between the static and dynamic stabilizers of the glenohumeral joint. Static stabilizers include the articular anatomy, negative intra-articular pressure, fibrocartilaginous labrum, and the glenohumeral ligaments. The dynamic stabilizers of the shoulder include the rotator cuff muscles and the scapulothoracic stabilizers. With increased utilization of advanced imaging and computed tomographic (CT) scans with 3 dimensional (3D) reconstructions, there is an improved understanding of the importance of bipolar bone loss on the outcomes following shoulder stabilization surgery. Herein, we provide a comprehensive review of the

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functional anatomy of the glenohumeral joint with a focus on scapular morphology and glenoid and humeral bone loss.

STATIC AND DYNAMIC STABILIZERS OF THE GLENOHUMERAL JOINT

The glenohumeral joint is composed of both static and dynamic stabilizers, which allow the humeral head to remain stable and centered on the relatively flat glenoid socket through a large range of shoulder motion. The static stabilizers of the glenohumeral joint include the articular anatomy of the glenoid and humeral head, the fibrocartilaginous labrum, the negative intra-articular pressure and the superior, middle, and inferior glenohumeral ligaments (superior glenohumeral ligament [SGHL], middle glenohumeral ligament [MGHL], and inferior glenohumeral ligament [IGHL]). The dynamic stabilizers of the shoulder joint include the rotator cuff muscles of the supraspinatus, infraspinatus, subscapularis, and the teres minor. With anterior instability, the subscapularis is particularly important in providing a dynamic restraint to anterior humeral translation. If the horizontal force couple is disrupted, this can lead to recurrent anterior instability (**Fig. 1**). The scapulothoracic stabilizer muscles are also a key dynamic stabilizer of the glenohumeral joint.

Understanding glenoid morphology plays an important role in evaluating the stability of the shoulder joint. The normal glenoid diameter measures 26.2 mm in women and 30.3 mm in men. The normal glenoid version ranges from -4° of retroversion to 7° of anteversion. The amount of inclination varies from 0° to 10° of inclination.[1–4] **Fig. 2** demonstrates a 3D CT reconstruction of the glenoid denoting the clock-face orientation often used when describing glenoid and labral morphology.

Cadaveric studies have demonstrated that the human anterior inferior glenoid labrum is a fibrous rounded structure, which is confluent with the hyaline cartilage of the glenoid articular surface.[5] Histologically, there is a narrow fibrocartilaginous transition zone between the glenoid hyaline cartilage and the fibrous labral tissue. This narrow transition zone is composed of numerous collagen bundles in a woven pattern within the hyaline cartilage.

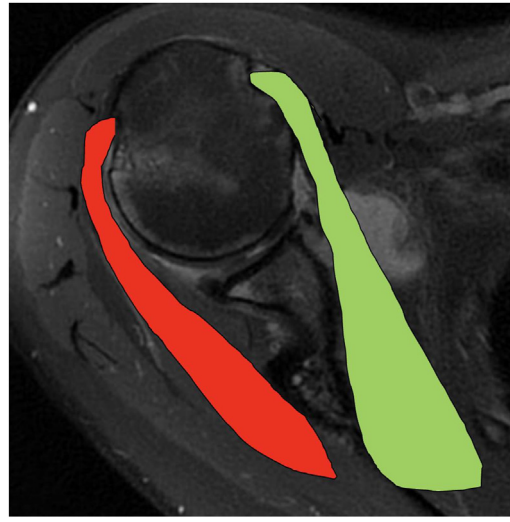

Fig. 1. Axial proton dense fat saturation image on an MRI that demonstrates the subscapularis (green) and the infraspinatus (red), which act as a force couple to balance the shoulder.

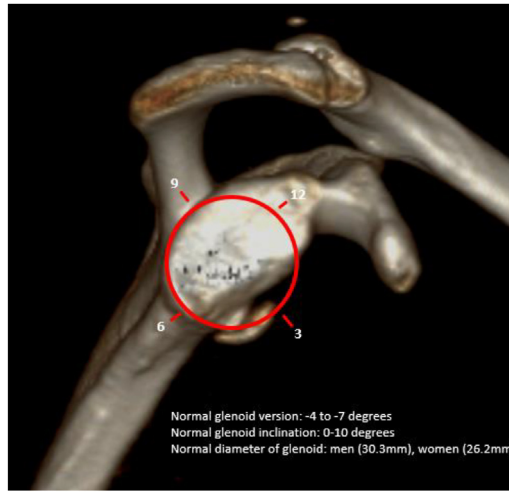

Fig. 2. Three-dimensional CT reconstruction of the glenoid and scapula demonstrating the clock-face orientation used for describing glenoid morphology.

The glenoid labrum provides depth to the glenoid and a bumper to prevent anterior inferior dislocation. Vaswani and colleagues performed a retrospective case–control study of patients undergoing primary anterior arthroscopic shoulder stabilization and matched those who sustained a failure of Bankart repair to those who did not. They found that a diffusely small labral morphology was a significant risk factor for sustaining a postoperative redislocation.[6]

The anterior inferior and posterior inferior glenoid labrum also serve as an important attachment site for the anterior and posterior bands of the inferior glenohumeral ligament (anterior-inferior glenohumeral ligament [AIGHL], posterior-inferior glenohumeral ligament [PIGHL]) that serve as a "hammock-like" structure to statically stabilize the shoulder.[7] The AIGHL and PIGHL undergo reciprocal shortening and lengthening in 90° of glenohumeral abduction and serve as a "check rein" to anterior and posterior translation.[8] Plastic deformation of the AIGHL with an associated Bankart is the classic pathologic lesion that occurs following anterior inferior glenohumeral dislocation. In addition to tearing of the anterior inferior labrum from the glenoid (Bankart lesion), the AIGHL can also tear at its attachment on the humerus, the so-called humeral avulsion of the glenohumeral ligament lesion.[9] Finally, the AIGHL can also rupture from both the labrum and the humerus, the "floating AIGHL."[10]

GLENOID BONE LOSS

The pathologic lesion of an anterior glenohumeral dislocation is an anterior inferior glenoid labral tear and a posterior humeral head impaction fracture (Hill–Sachs [HS] lesion).[11] However, commonly with recurrent anterior shoulder instability, patients develop anterior glenoid bone loss. Attritional glenoid bone loss has been well defined as a risk factor for failure of an arthroscopic soft tissue repair (Bankart).[12] Wiesel and colleagues[13] reported glenoid bone loss as the most common cause for failure of a primary arthroscopic soft-tissue stabilization procedure resulting in increased morbidity and cost.

Glenoid rim defects can be identified as glenoid rim fractures or attritional bone loss. Bigliani and colleagues[12] described a classification system for glenoid bone lesions

with 3 different types. Type I is a displaced avulsion fracture with attached capsule. Type II is a medially displaced fragment malunited to the glenoid rim. Type III can be defined as erosion of the glenoid rim with less than 25% (Type IIIA) or greater than 25% (Type IIIB) deficiency (Fig. 3).

In a prospective cohort study, Dickens and colleagues evaluated the degree of glenoid bone loss after initial and recurrent instability events. Glenoid bone loss after a first-time instability event was noted to be 6.8%, which increased to 22.8% in the setting of recurrent instability.[14] Milano and colleagues[15] analyzed a cohort of 161 patients with recurrent shoulder instability, glenoid bone defects were identified in 72% of patients with an average size of 6.9%. In a separate cohort of 218 patients, 71% of cases were noted to have bone loss with a mean size of 10.8%.[16] Shaha and colleagues[17] investigated the amount of glenoid bone loss in a young active-duty population noting 89.2% of their patients had bone loss with a mean of 14.5%. In comparison of these studies, it can be identified that the military population is a high-risk group for glenoid bone loss, which typically constitutes large defects.[14,17,18] Early stabilization is often recommended after an initial instability event in a young, active patient due to the risk of recurrent instability and increasing bone loss.

Initial assessment involves radiographs including anteroposterior (AP), Grashey, Axillary, and Scapular Y views. The West Point view and the Bernageau view can be obtained to assess for glenoid defects.[19,20] These radiographs can assist in identifying glenoid defects but are noted to have a reduced sensitivity compared to advanced imaging including CT and MRI.[21]

A CT scan with 3D reconstruction and humeral head subtraction allows for the identification and assessment of the size of the glenoid lesion through an en face view. In a cadaveric study, Huysman and colleagues[22] identified the inferior glenoid as a true circle and the bare spot as a means for identifying the center of that circle. A best-fit circle technique can be used to quantify the amount of bone loss within the area of circle that does not overlap bone as percentage of bone loss. There are several other methods of quantifying bone loss including the Pico method, circle line method, linear measurement percentage (LMP) method, and glenoid arc angle method (Table 1).[21,23–26] All these methods begin with the assumption that a best-fit circle can be placed on the inferior glenoid (Fig. 4).

There is evidence that supports these methods can be reliably performed on MRI as well; however, experts recommend 3D CT as the most current accurate study to assess bone loss.[27] Huijsmans and colleagues[28] performed a cadaveric study

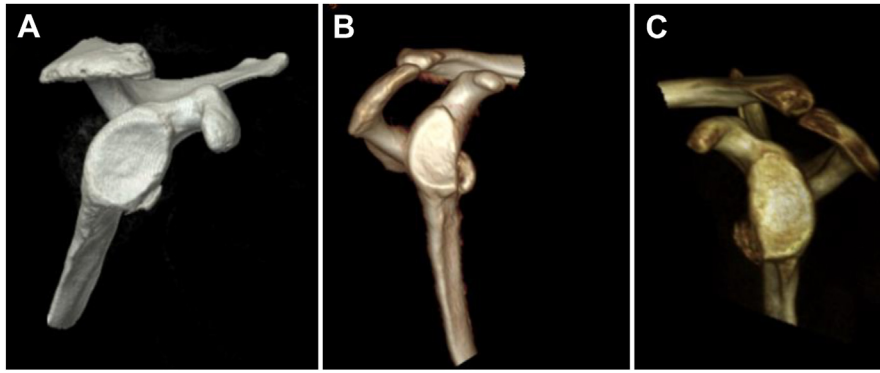

Fig. 3. Three-dimensional reconstructions demonstrating Bigliani glenoid rim defects with (*A*) type I, (*B*) type II, and (*C*) type III (*B*) lesions.

Table 1
Various methods of quantifying glenoid bone loss

Measurement Technique	Description	Equation
Best-fit circle method	True circle is centered on the inferior two-thirds of the glenoid, and the diameter of the circle and AP diameter of glenoid are measured at the same level	% GBL = (A – B)/A × 100% A = surface area of best-fit circle B = surface area of glenoid
Pico method	Circumference of the unaffected inferior glenoid based on a true circle is obtained, and then transferred to the affected glenoid; the glenoid defect is manually traced out in the affected shoulder to calculate surface area of bone loss	% GBL = (A – B)/A × 100% A = surface area of unaffected glenoid B = surface area of affected glenoid
Circle-line method	Best-fit circle is drawn as a line from center to the edge and compared with center to the bone defect; uses chord length to calculate area of a circular segment (ie, area of bone loss) and percent bone loss	Chord length = 2r sin (C/2) Area of bone loss (A) = $r^2/2$ (π/180C – sin C) % GBL = A/B × 100% A = area of bone loss B = total area of circle C = central angle r = radius of glenoid
LMP technique	Linear, ratio-based technique to measure the expected diameter of the glenoid based on a best-fit circle with a line drawn from center of circle to edge and compared with center to glenoid defect	% GBL = w/D × 100% D = best-fit circle diameter w = width of bone defect
Glenoid arc angle method	Center of the circle and central angle is defined by lines drawn to the upper and lower aspects of the glenoid defect; superior and inferior points at which the glenoid defect intersects the perimeter of the circle represents the central angle toward the center point of the circle	% GBL = [(α-sin α)/2 π] × 100% α = glenoid arc angle
Griffith's index	AP width measurements made perpendicular to a line through the vertical access of the glenoid from the supra-infra glenoid tubercle, which is compared between the affected and unaffected side to determined percent width loss	% GBL = B/A × 100% A = AP glenoid width of unaffected shoulder B = AP glenoid width of affected shoulder
Glenoid index (width) method	Using a best-fit circle, compare affected glenoid radius to unaffected native glenoid radius to give percentage bone loss, similar to arthroscopic bare spot technique	(R − r)/(2R) × 100% r = affected glenoid radius R = unaffected glenoid radius

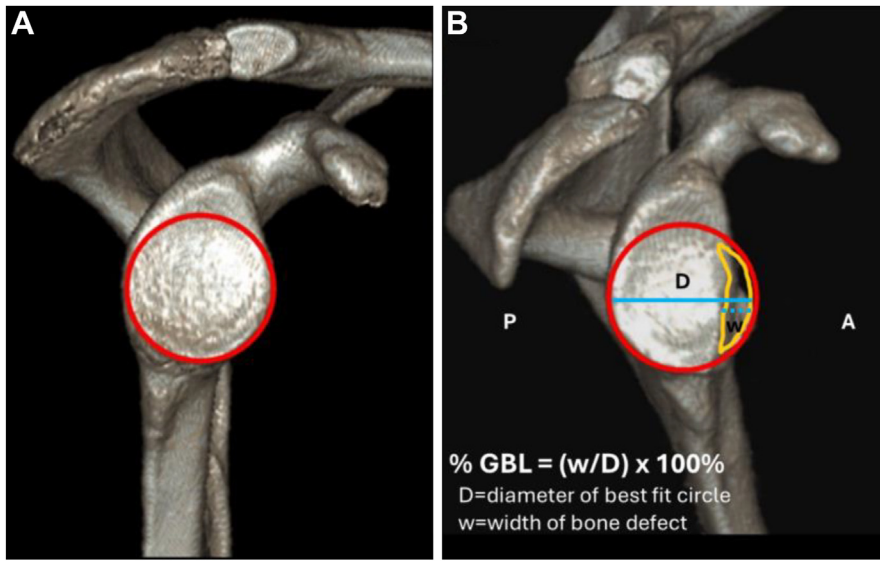

Fig. 4. A CT reconstruction with 3D reformat en face view of the glenoid demonstrating a best-fit circle drawn along the inferior portion of the glenoid shown on an intact glenoid without bone loss (*A*) as well as on a glenoid with attritional bone loss and medialized bone on the glenoid neck (*B*). A, anterior; P, posterior.

quantifying the amount of bone loss with the circle method and found no statistical differences between 3D CT and MRI. Magnetic resonance arthrography can allow for the assessment of soft-tissue injuries in addition to bone loss and has been found to have strong interobserver and intraobserver correlation of bone loss measurement compared to CT.[29]

Glenoid bone loss has been associated with failure of soft-tissue stabilization procedures resulting in worse clinical outcomes. Hettrich and colleagues prospectively followed 892 patients to assess predictors for bone loss in anterior shoulder instability. Anterior glenoid bone loss was identified in 20.7% of patients and was associated with an increased number of dislocations, increasing age, male sex, non-White race, and contact sports participation.[18] Milano and colleagues[15] evaluated a cohort of 161 patients with anterior shoulder instability and noted that the most significant predictors of bone loss were number of dislocations and age at first dislocation. It is important to identify bone loss because the failure rate of performing an arthroscopic Bankart repair alone in the setting of significant bone loss leads to increased failure rates.[30,31]

Boileau and colleagues[31] noted that glenoid bone loss greater than 25% was a risk factor for recurrent instability after arthroscopic stabilization. There have been several studies that analyze the amount of bone loss that indicates risk for recurrence of instability as well as the morphology of these glenoid rim lesions. Historically, glenoid defects have been classified as "critical" and necessitating bony augmentation procedures anywhere from 20% to 25%.[12,32–34] Itoi and colleagues performed a classic cadaveric study to assess the effect of different sizes of glenoid defects on stability and range of motion after Bankart repair. Their study demonstrated that an osseous defect of 21% may cause instability and limit range of motion.[35]

Further studies have evaluated "subcritical" amounts of bone loss. Shaha and colleagues analyzed a cohort of 72 anterior instability patients in a military population. In

their patient population, bone loss of greater than 13.5% yielded clinically significant differences in outcome scores even in the setting of patients that did not have recurrent instability.[17] Yamamoto and colleagues performed a follow-up study to Shaha and colleagues analyzing the amount of bone loss that led to worse outcome scores and recurrent rates in a cohort of 43 patients. This study demonstrated that a bone loss of greater than 17% was associated with worse Western Ontario Shoulder Instability Index (WOSI) scores especially in male patients that participated in sporting activities.[36] These studies indicate that the critical size necessitating bony augmentation procedures may be lower than the often cited threshold of 20% to 25%.[17,35,36]

Anterior instability and the morphology of glenoid defects has a characteristic pattern that is different from other forms of instability. Sugaya and colleagues performed a 3D CT study evaluating the glenoid rim morphology in 100 patients with recurrent instability. 90% had a glenoid rim defect with 50% being true bony Bankart lesions and 40% with loss of the normal circular configuration of the inferior glenoid.[37] The location of these lesions in anterior glenoid instability has been identified ranging from 12 to 6 o'clock on the glenoid clock face with the majority of lesions ranging between 2:30 and 4:20.[38]

Defects associated with anterior instability are vastly different than those associated with posterior instability. Ernat and colleagues quantitatively compared the anatomic and morphologic differences between anterior and posterior shoulder instability patients with the use of CT. Anterior glenohumeral instability was associated with anterior glenoid lesions that have a steeper slope, higher percentage of bone loss, and greater superior-inferior defect height. Posterior glenohumeral instability was primarily associated with a greater degree of retroversion rather than bone loss and occurs in the posterior inferior quadrant of the glenoid.[39]

As previously discussed, the amount of glenoid bone loss necessitating bony reconstruction surgery has been described as the "critical" amount of bone loss. Recent publications by Moroder and colleagues have challenged the concept of a specific threshold of bone loss that may necessitate these procedures. In a case–control study, Moroder and colleagues evaluated 96 shoulders with CT images to evaluate the bony glenoid concavity and the bony shoulder stability ratio (BSSR). The mean BSSR as depicted in the equation shown in **Fig. 5** was used to determine the stability between anterior shoulder instability and a control group.

In this cohort of patients, anterior shoulder instability was associated with a flattening of the normal glenoid concavity, which led to a significantly decreased BSSR.[40] In a more recent publication, Moroder and colleagues evaluated the capability of glenoid bone loss measurements to accurately depict the biomechanical effect of glenoid defects. Their study determined that conventional measurements with CT are unable to accurately estimate the biomechanical effect of glenoid defects due to the nonlinear relation between defect size and biomechanical effect as well as

$$BSSR = \frac{\sqrt{1-\left(\frac{r-d}{r}\right)^2}}{\frac{r-d}{r}}$$

Fig. 5. BSSR equation which is a mathematical equation depicting the stability of the shoulder joint based on CT measurements of the glenoid concavity. (*From*: Moroder P, Ernstbrunner L, Pomwenger W, Oberhauser F, Hitzl W, Tauber M, et al. Anterior Shoulder Instability Is Associated With an Underlying Deficiency of the Bony Glenoid Concavity. Arthrosc J Arthrosc Relat Surg Off Publ Arthrosc Assoc N Am Int Arthrosc Assoc. 2015 Jul;31(7):1223–31.)

the inability to assess the glenoid concavity shape differences. This study indicates that a "critical" threshold for bone loss does not accurately depict who may need a bony augmentation procedure and that glenoid bone restoration involves restoring both the width and concavity of the glenoid.[41]

HUMERAL BONE LOSS

An HS lesion, present in 70% of first-time dislocators and over 90% in recurrent dislocations, is defined by an impaction fracture of the posterior-superior humeral head against the anterior glenoid rim.[42] Although humeral bone defects have traditionally been considered less important than glenoid bone defects, there is increasing attention toward defects of the humeral head as a significant contributor to recurrent anterior shoulder instability.[23,43,44]

CT, especially with 3D reconstructions, is the gold standard for quantifying humeral bone defects,[45,46] and methods for measuring humeral bone loss continue to evolve over time. The first method used for quantifying HS lesions was performed on plain radiographs as a percentage of bone loss involvement in a 180° arc on the surface of the humeral head.[47] In this quantitative method, the HS depth index is calculated based on the ratio of the maximum depth of the notch defect (p) in internal rotation and the radius of the humeral head (R; **Fig. 6**).[48] This method has since been extrapolated to CT and MRI studies and was further expanded by Rowe and colleagues[49] to account for both the depth and width of bone loss to develop a grading scheme based on clinical severity. This classification system ranges from mild (<0.5 cm deep and <2 cm long), moderate (0.5–1 cm deep and 2–4 cm long) and severe (>1 cm deep and >4 cm long). The depth and width of humeral bone loss can be divided by the diameter of the humeral head and quantified as percentage of the humeral head diameter measured on both axial and coronal CT images, with bone loss greater than 40% being clinically significant.[50–52]

Increasing evidence suggests that the location and orientation of the HS lesion, extending from 0 mm to 24 mm from the top of the humeral head, in addition to the size, can affect clinical outcomes and provide prognostic value in predicting recurrent instability.[23] Twenty-five percent of humeral bone loss has been suggested as the critical threshold to recommend surgical intervention, mostly from studies that have

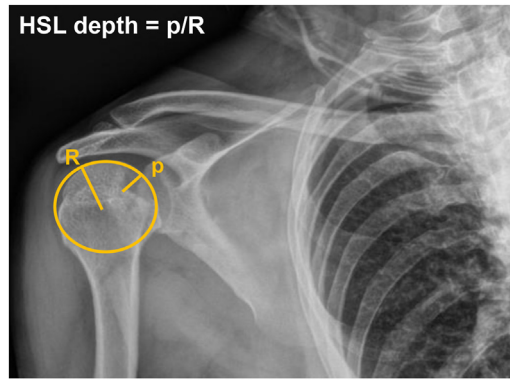

Fig. 6. An AP plain film of a right shoulder with a large Hill–Sachs lesion (HSL) demonstrating a quantitative method for measuring the HSL depth index, based on the ratio of the maximum depth of the notch defect (p) in internal rotation and the radius of the humeral head (R).

shown biomechanical alterations and clinical failure leading to instability at this threshold of humeral head volume loss.[43,44,53,54] In addition to volume of humeral head bone loss, the extent of bone loss in relation to the glenoid should also be considered, as studies support that humeral bone loss greater than 21% of the superior-inferior length of the glenoid or greater than 25% of the glenoid depth also leads to instability.[54] In recurrent instability, the posterolateral notch of the HS lesion increases in depth and the overall volume of humeral head bone loss increases.[35] Peebles and colleagues[45] additionally noted that glenoid bone loss was directly related to the characteristics of an HS lesion, and patients with smaller glenoid defects had narrower and deeper HS lesions with less humeral head surface area loss, while greater glenoid bone loss was associated with wider and shallower HS lesions.

The risk of an HS lesion engaging with the anterior rim of the glenoid is greatest at the end-range of abduction, external rotation, and horizontal extension because this is the position that the glenoid overrides the HS lesion.[35] The concept of glenoid track (GT) was introduced to describe the relationship of engagement of the glenoid and humeral head articular surfaces during movement at varying positions of abduction and external rotation.[36] This concept suggests that the location of the HS lesion is a significant contributing risk factor to recurrent instability, demonstrating that there is risk of engagement if the lesion extends beyond the medial margin of the zone of contact between the articular surfaces of the glenoid and humeral head.[36] In an on-track lesion, the HS defect stays within the GT and no engagement or dislocation occurs. Conversely, if the HS defect falls outside of the GT, termed an off-track lesion, the anterior rim of the glenoid may engage with the HS, leading to a recurrent dislocation.[55]

Glenoid bone loss has a high occurrence up to 100% in the presence of an HS lesion, described as a bipolar lesion.[43,55] In a bipolar lesion, the GT area is smaller and, therefore, the medial margin draws closer to the footprint, increasing the risk of engagement.[43,56] Nakagawa and colleagues[57] found one-third of all shoulders with primary instability had bipolar lesions, as did 60% of shoulders with recurrent instability, with a significant difference in recurrent instability noted in the setting of HS lesions and the inverted pear-shaped glenoid lesions.[58]

Three-dimensional CT scans are used in daily practice to measure on-track versus off-track lesions, using en face views of both the glenoid and posterior aspect of the humeral head (**Fig. 7**A).[45,59] This estimation is performed by a formula proposed by Di Giacomo and colleagues[59] and validated in several subsequent studies[35,55,60,61] (**Box 1**). The location of the medial margin of the GT is 83% of the entire glenoid width with the arm in 90° of abduction in normal shoulders.[62] As a result, the width of the GT in the setting of bone loss is defined as 0.83(D) – d, where D is the diameter of the inferior glenoid (**Fig. 7**B) and d is the width of the anterior glenoid bone loss (**Fig. 7**C). The GT width is applied to the posterior aspect of the humeral head. The Hill–Sachs interval (HSI) is subsequently measured as the distance from the rotator cuff attachment to the medial rim of the HS lesion, which is equal to the width of the HS lesion and the width of the intact bone bridge (BB) between the rotator cuff and the HS lesion. If the HSI is greater than the GT width, the medial rim of the HS extends past the glenoid rim with no bony support adjacent to the HS, identifying an off-track or engaging lesion. Contrarily, if the HSI is less than the GT width, there is bony support adjacent to the HS lesion and the medial rim of the HS is contained within the glenoid rim, identifying an on-track or nonengaging lesion. The effect of on-track and off-track lesions may potentially guide surgical decision-making in terms of arthroscopic soft tissue repair with or without the addition of humeral-sided procedures (ie, humeral head bone graft or remplissage).[46,63,64]

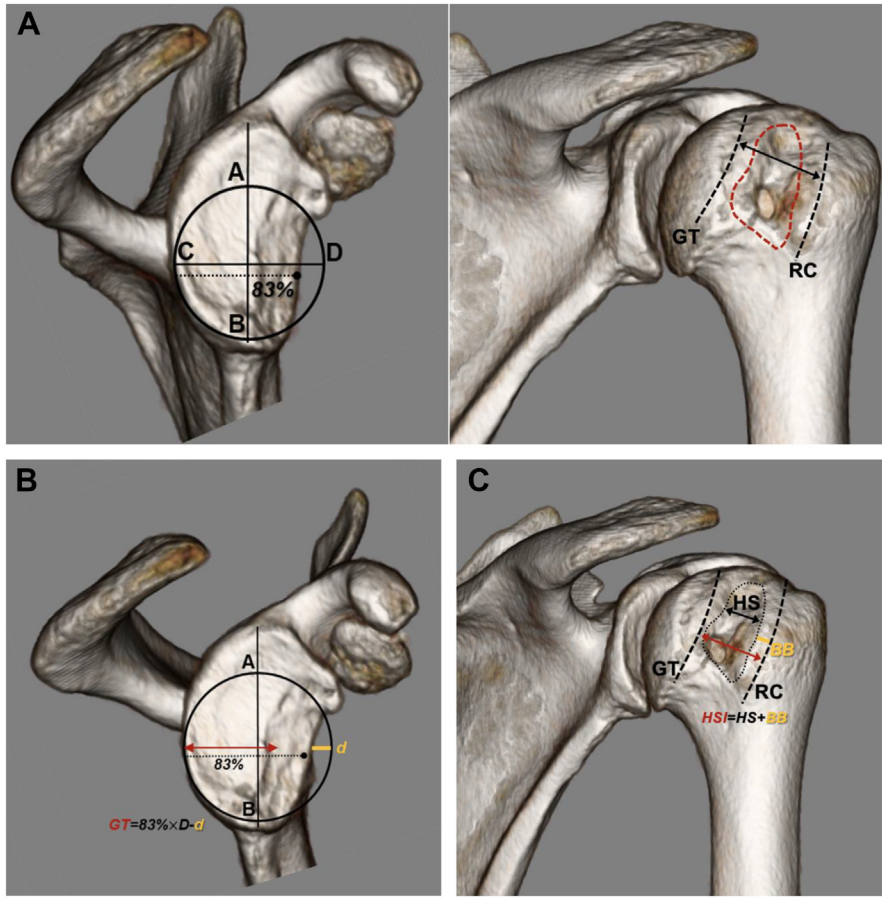

Fig. 7. A 3D CT scan utilized to measure GT in the presence of an HS lesion. (*A*) Three-dimensional CT scan with en face views of a left shoulder with glenoid bone loss (left) and posterior humeral head with a medium HS lesion (right). Line A–B represents the long axis of the glenoid, and the perpendicular line C–D is the diameter of the inferior glenoid. The width of a normal GT is defined as 83% of the inferior glenoid width (*dotted line*). Dotted line GT indicates the medial margin of the GT; dotted line RC indicates the medial margin of the posterior rotator cuff attachments. The boundary of the HS lesion is outlined in the red-dotted circle. (*B*) Three-dimensional CT scan with en face view of a left shoulder glenoid with bone loss of width d (*yellow line*). With glenoid bone loss, GT = 0.83 × D − d. D represents the inferior glenoid width of a normal glenoid without bone loss; line A–B is the long axis of the glenoid. The width of a normal GT without bone loss is defined as 83% of the inferior glenoid width (*dotted line*). (*C*) Three-dimensional CT scan with en face view of a left posterior humeral head with an HS lesion. The HSI is defined as the width of the HS lesion (boundary outlined in the red-dotted circle) plus the width of the intact BB between the HS and the posterior rotator cuff (RC) attachments. Dotted line GT indicates the medial margin of the GT; dotted line RC indicates the medial margin of the posterior RC attachments.

Historically, on-track and off-track lesions have been thought of as a binary variable, yet more recent studies describe this paradigm as a spectrum of risk of recurrent instability, introducing the term near-track lesions.[65–67] More specifically, on-track lesions with a small distance-to-dislocation (DTD) value, in which the medial edge of the

Box 1
Measurement of on-track versus off-track Hill–Sachs lesion

1. Obtain 3D CT scan with en face view of the glenoid (with humeral head subtraction) and the posterior view of the humeral head
2. Measure the diameter (D) of the inferior glenoid (perpendicular line to the long axis of the glenoid)
3. Measure the width of the anterior glenoid bone loss (d)
4. Calculate the width of the GT:
   $GT = 0.83 \times D–d$
5. Measure the width of the HS lesion
6. Measure the width of the remaining BB between the posterior rotator cuff attachment and lateral aspect of the HS lesion
7. Calculate the width of the HSI:
   $HSI = HS + BB$
8. If HSI > GT, HS lesion is off-track (engaging); if HSI < GT, HS lesion is on-track (nonengaging)

From: Di Giacomo G, Itoi E, Burkhart SS. Evolving concept of bipolar bone loss and the Hill-Sachs lesion: from "engaging/non-engaging" lesion to "on-track/off-track" lesion. Arthrosc J Arthrosc Relat Surg Off Publ Arthrosc Assoc N Am Int Arthrosc Assoc. 2014 Jan;30(1):90–8.

HS lesion is still within but in close proximity to the medial edge of the GT are still at a higher risk of failure and recurrent dislocation. The DTD is defined as the distance between the edge of the anterior aspect of the glenoid and the medial border of the HS lesion.[66] As the GT narrows and HS size increases, on-track lesions demonstrate a smaller DTD value and subsequently fall into the continuum of a near-track lesion.

The term near-track lesions suggests that not all on-track lesions are the same and the clinical implications of the DTD value require further investigation. Li and colleagues[66] demonstrated that a DTD of less than 8 mm in a near-track lesion is a risk factor for failure after arthroscopic Bankart repair, especially in patients aged 20 years or older, suggesting that the DTD value is a continuous marker to predict the risk of failure. Similarly, Barrow and colleagues[65] found that as the DTD approached 0 mm (the threshold for an off-track lesion), the risk of recurrent dislocation after arthroscopic Bankart repair increased. Furthermore, the authors demonstrated that a DTD of less than 10 mm was associated with an exponentially higher risk of failure, with a higher risk of recurrence for collision athletes at even higher DTD values compared to noncollision athletes. However, in a retrospective study of a Dutch military population by Verweij and colleagues,[67] there was no relationship demonstrated between a smaller DTD in predicting recurrent anterior instability, which may be due to the high-demand population or mid-range instability.

INFLUENCE OF SCAPULAR MORPHOLOGY

Variations in glenoid, coracoid, and acromial morphology have implications in anterior shoulder instability. Anterior instability is associated with a flattened AP radius of curvature of the glenoid, larger height to width index ratio, shallower cavity with loss of sphericity, and more anteversion and inferior inclination of the glenoid.[68–72] Glenoid version is thought to have a linear effect on the force required for dislocation. In a cadaveric study by Eichinger and colleagues,[73] increased glenoid anteversion of 10° resulted in spontaneous dislocation in 25% of specimens. A shallow glenoid in conjunction with a large

humeral head may also predispose patients to recurrent dislocation after arthroscopic Bankart repair.[74] Vaswani and colleagues[74] demonstrated patients with a shallow glenoid morphology (radius of curvature $\geq$24.5 mm) and a larger humeral head volume ($\geq$80 mm^3) had a 4 fold increased odds of postoperative dislocation. As understanding improves upon the relationship of glenohumeral instability and variations in glenoid morphology, there is increasing awareness that soft tissue stabilization procedures alone do not address the pathologic bony anatomy and may contribute to a patient's baseline predisposition to recurrent anterior instability.[74,75]

The sagittal morphology of the coracoacromial arch plays an increasingly recognized role in the pathogenesis of anterior instability. Owens and colleagues[69] identified the coracohumeral distance as an independent risk factor for traumatic anterior instability, noting a 20% increased risk of instability for every 1 mm increase in coracohumeral distance. Patients with anterior instability have been shown to have a more obtuse coracoacromial arch (decreased shoulder arch angle), in addition to a shorter coracoid with a superomedial offset and larger anterior coracoid tilt.[75] Altered contact between the conjoint tendon and the subscapularis as a result of altered coracoid morphology may lead to an altered line of force through the subscapularis, limiting the restraint to anterior humeral head translation during anterior shoulder dislocation.[76]

The acromion exhibits the highest variance of all periarticular scapular anatomic structures in relation to the glenoid.[76] An acromion with a more posterior origin of the scapular spine and more vertical orientation contributes to traumatic anterior shoulder instability.[76] Additionally, a flatter acromial roof and less containment of the humeral head can further contribute to traumatic instability.[68,75] A more vertical orientation of the acromion likely results in a greater percentage of the deltoid posterior to the center of the glenoid, providing less resistance to anterior translation. Increased posterior acromial coverage and posterior acromial tilt are described protective factors against recurrent anterior instability, suggesting that the anatomy of the entire arch, including both anterior and posterior elements, is important in anterior stability.[69,75]

CONTRIBUTION FROM THE LONG HEAD OF THE BICEPS TENDON

The long head of the biceps tendon (LHBT) is a dynamic stabilizer of the glenohumeral joint, with variable contribution to anterior shoulder instability.[77] While some studies have suggested that the LHBT plays a negligible role in dynamic stability of the shoulder, there is some evidence that the tendon offers some contribution to static stabilization of the shoulder as a humeral head depressor depending on the position of shoulder elevation in overhead activity.[78–80] As forward elevation of the shoulder stresses the posterior aspect of the unconstrained glenohumeral joint, biomechanical loading of the LHBT in cadaveric specimens demonstrated resistance to posterior translation of the humeral head during forward elevation, suggesting its role as a posterior stabilizer specifically at 60° of glenohumeral forward elevation.[81]

A recent review of 214 articles examining the contribution of the LHBT in shoulder stability found a minimal role of the LHB in glenohumeral stability in healthy controls.[82] However, hyperactivity of the LHB detected by electromyography in patients with rotator cuff failure or absent LHBT suggested a more significant role in shoulder stability and potential compensatory function in pathologic shoulders and instability. Ten studies evaluated the role of the proximal biceps in joint stability with variable results. One study of 20 asymptomatic volunteers noted a dynamic role of the LHBT in shoulder stability during low angles of shoulder elevation (<30°),[83] while another study of 5 patients undergoing LGBT tenodesis demonstrated no change in joint position or

activity compared to healthy controls.[84] In addition to rotator cuff failure, there is increasing awareness for potential LHBT lesions in acute traumatic dislocations, particularly in patients aged over 45 years. Feuerriegel and colleagues[85] noted 52% of patients with traumatic dislocations demonstrated pathology of the biceps pulley system, all of which showed associated partial tear of the rotator cuff. Furthermore, patients with recurrent anterior shoulder instability with greater than 5 dislocation have demonstrated increasing rates of associated biceps pathology requiring treatment at the time of surgical stabilization.[86]

BIOMECHANICS

Glenohumeral instability is a pathologic condition in which the humeral head is unable to maintain its position in the center of the glenoid fossa due to damage of 1 of more of these stabilizers.[87]

When considering glenoid reconstruction for anterior glenoid bone loss, it is important to understand the different reconstruction options and their effect on the glenohumeral biomechanics. Rauck and colleagues sought to evaluate the proportion of the glenoid width restoration necessary to restore glenohumeral stability after Latarjet. The authors measured anterior humeral heading translation and force distribution on the coracoid graft. They found that restoration of native glenoid width restored anterior stability while preventing coracoid graft overload and that restoration of 100% of native glenoid width should be the minimum goal when performing anterior glenoid reconstruction.[81] Distal tibial allograft (DTA) is a reasonable option for large glenoid reconstruction, as it offers a graft with a cartilaginous surface to reconstruct the joint (**Fig. 8**). Rodrigueza and colleagues evaluated DTA versus Latarjet for glenoid

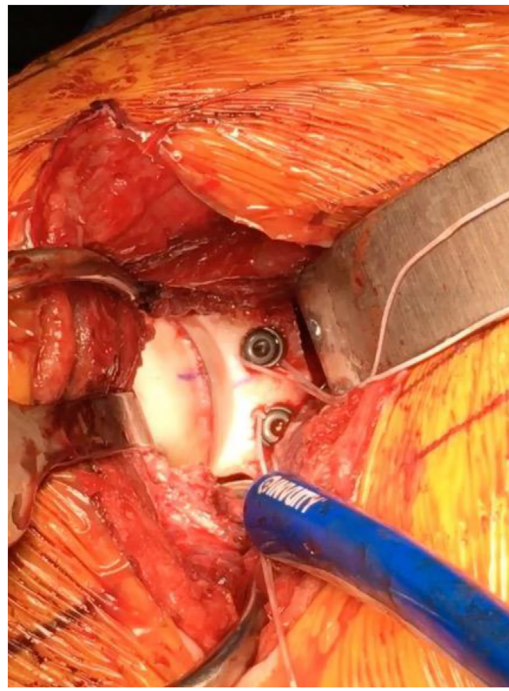

Fig. 8. Intraoperative photograph of right shoulder anterior glenoid reconstruction with distal tibia allograft demonstrating anatomic reconstruction of cartilaginous surface.

reconstruction with respect to joint kinematics and cartilage pressure mapping in shoulders undergoing 25% anterior glenoid-defect reconstruction. The authors concluded that Latarjet and DTA reconstructions exhibit similar glenohumeral kinematics regarding glenohumeral joint stability. However, joint compression load and articular contact pressure distribution may favor DTA reconstruction.[88] Also, when considering the biomechanics of anterior instability, the presence of a humeral head lesion must also be taken into consideration. Patel and colleagues designed a study to investigate the effects of Latarjet only reconstruction and the presence of varying humeral head defects. The authors found that anterior glenoid augmentation with a bone block can provide increased translation to dislocation in the presence of humeral defect sizes of 19% to 31% of the humeral head. However, Latarjet was not sufficient in defects greater than 31% and would require humeral augmentation.[89] Soft-tissue repair may be a valuable treatment option for patients with minimal bone loss; however, one must account for the negative and additive effect of humeral lesions. Arciero and colleagues evaluated the biomechanical efficacy of a Bankart repair in the setting of bipolar bone defects. The authors found that bipolar defects have an additive and negative effect on glenohumeral stability. As little as a 2 mm glenoid defect with a medium-sized HS lesion demonstrated a compromise in soft-tissue Bankart repair, while a small-sized HS lesion showed compromise of soft-tissue repair with 4 mm or more glenoid bone loss.[63]

SUMMARY

Shoulder stability is the result of a complex interplay between the static and dynamic stabilizers of the glenohumeral joint. It is important to understand the bipolar nature of bone loss from the glenoid and humeral head when evaluating the stability of the shoulder joint. While there are several cutoffs and treatment algorithms detailing options for treatment of bone loss, it is important to utilize a thorough history, physical examination, and evaluate the entirety of the glenohumeral joint stabilizers including the surrounding soft tissue structures as well as the morphology of the scapula.

DISCLOSURE

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