Management of Bone Loss in Glenohumeral Instability

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KEYWORDS

• Glenohumeral instability • Hill-Sachs • Bankart lesion • Latarjet

KEY POINTS

- With anterior dislocations, bony defects of the anterior glenoid and posterosuperior aspect of the humeral head occur with relative frequency.
- In shoulders sustaining a Hill-Sachs lesion at the initial dislocation, there exists a statistically significant association with recurrent dislocation.
- When a patient has symptomatic anterior instability associated with an engaging Hill-Sachs lesion with an articular arc deficit, treatment must be directed at both repairing the Bankart lesion, if present, and preventing the Hill-Sachs lesion from engaging the anterior glenoid.
- Glenoid bone loss often requires bone-block transfers using the coracoid (Bristow/Latarjet) or iliac crest autograft.
- Humeral bone loss can be addressed through a variety of surgical options, including humeroplasty, remplissage, partial resurfacing, allograft transfers, and total shoulder arthroplasty.

INTRODUCTION

The human shoulder is the most mobile joint in the body and consequently the glenohumeral joint is one of the most commonly dislocated joints in the body.[1] Glenohumeral instability affects approximately 2% of the general population, with anterior dislocations occurring 95% to 98% of the time.[2,3] With anterior dislocations, bony defects of the anterior glenoid and posterosuperior aspect of the humeral head occur with relative frequency (Fig. 1). These osseous injuries directly affect recurrent instability by altering joint-contact area, congruency, and function of the static restraints.[4–8] Thus, restoration of normal articular geometry should be considered when critical bone loss exists, especially in cases of failed soft-tissue stabilization procedures.

This review presents the epidemiology and pathophysiology of bone loss relevant to anterior shoulder instability, and summarizes the evaluation and management of this problem.

HUMERAL BONE LOSS

One of the first descriptions of the lesions found on the humeral head was by Flower in 1861, with many subsequent investigators reporting on these bony defects.[9,10] In 1940, 2 radiologists, Hill and Sachs, reported that these defects were actually compression fractures produced when the posterolateral humeral head impinged against the

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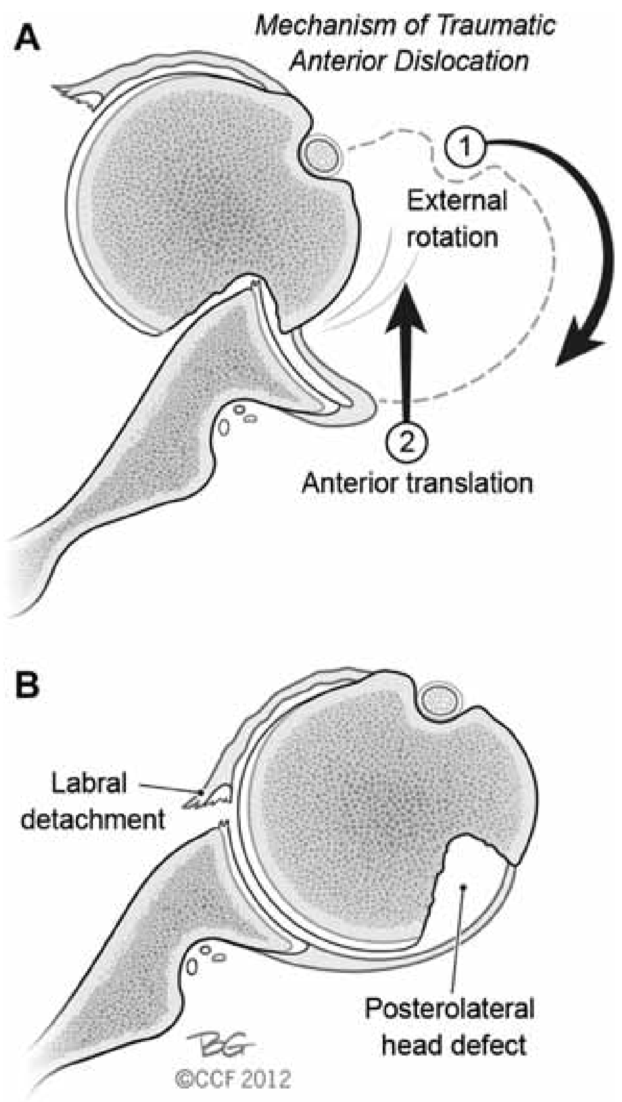

Fig. 1. Mechanism of traumatic anterior shoulder dislocation. (*A*) Combined forces in external rotation and anterior translation overcome internal restraints, resulting in anterior dislocation. (*B*) This process results in compression of the posterolateral aspect of the humeral head onto the anterior glenoid rim. (*Courtesy of* Cleveland Clinic Foundation, Cleveland, OH.)

anterior rim of the glenoid.[11] In their series of recurrent anterior glenohumeral instability, these lesions were found in 74% of patients. The true incidence of Hill-Sachs lesions is unknown; however, they are associated with approximately 40% to 90% of initial anterior glenohumeral dislocations.[12–16] The incidence in recurrent instability can vary from 70% up to 100%, with arthroscopy often identifying lesions not appreciated on imaging.[11,14] The management of Hill-Sachs lesions depends mainly on the size of the lesion and whether it is engaging.[17] Most lesions are small and clinically insignificant. Often, lesions that are clinically relevant may be indirectly managed with procedures aimed at addressing primary instability at the glenoid (ie, Bankart repair, glenoid reconstruction, and so forth).

GLENOID BONE LOSS

The characteristic anteroinferior capsulolabral injury (ie, Bankart lesion) associated with an acute anterior shoulder dislocation has been termed the essential lesion. Rowe and colleagues[13] first described glenoid bone loss as a “rim fracture” following anterior instability. Rowe’s key finding was the importance of the anterior glenoid rim in providing anterior shoulder stability, by creating a deepened concave surface of the glenoid and increased articular coverage. The importance of the rim fracture is shown in multiple studies by analyzing the relationship of the glenoid and humerus, especially in external rotation and abduction. Bigliani and colleagues[18] provided the first detailed description of osseous glenoid rim injuries, which included rim fractures and erosions (**Fig. 2**). In a radiographic study of patients with recurrent instability, 87% of shoulders involved the presence of either a glenoid rim fracture or erosion.[19] Griffith and colleagues[20] used 2-dimensional (2D) computed tomography (CT) to find glenoid bone loss in 41% of 66 patients with a first-time dislocation and 86% of 137 patients with recurrent instability. The predominant pattern of injury was attritional bone loss, with glenoid rim fractures reported in only 21% of 233 dislocated shoulders. However, using 3-dimensional (3D) CT to assess glenoid bone loss in 100 patients with recurrent instability, Sugaya and colleagues[21] found that only 40% of patients had erosive or attritional bone loss.

BIPOLAR BONE LOSS

The literature investigating osseous lesions of both the glenoid and humeral head is limited. The prevalence of combined bone defects is reported to be 64% to 70% in first-time anterior dislocations and 79% to 84% in recurrent anterior glenohumeral instability.[20,22]

PATHOPHYSIOLOGY

Knowledge of the pathoanatomy and biomechanics of glenohumeral bone loss and instability is crucial in the appropriate management to prevent recurrent instability. The most common mechanism of traumatic anterior shoulder dislocation occurs with an indirect force on the abducted and externally rotated arm. The humeral head externally rotates relative to the glenoid while translating anteriorly. The static glenohumeral restraints (ie, capsule, ligaments, labrum) are stretched or torn with further anterior translation, and dislocation, of the humeral head. The posterosuperolateral aspect of the humeral head then

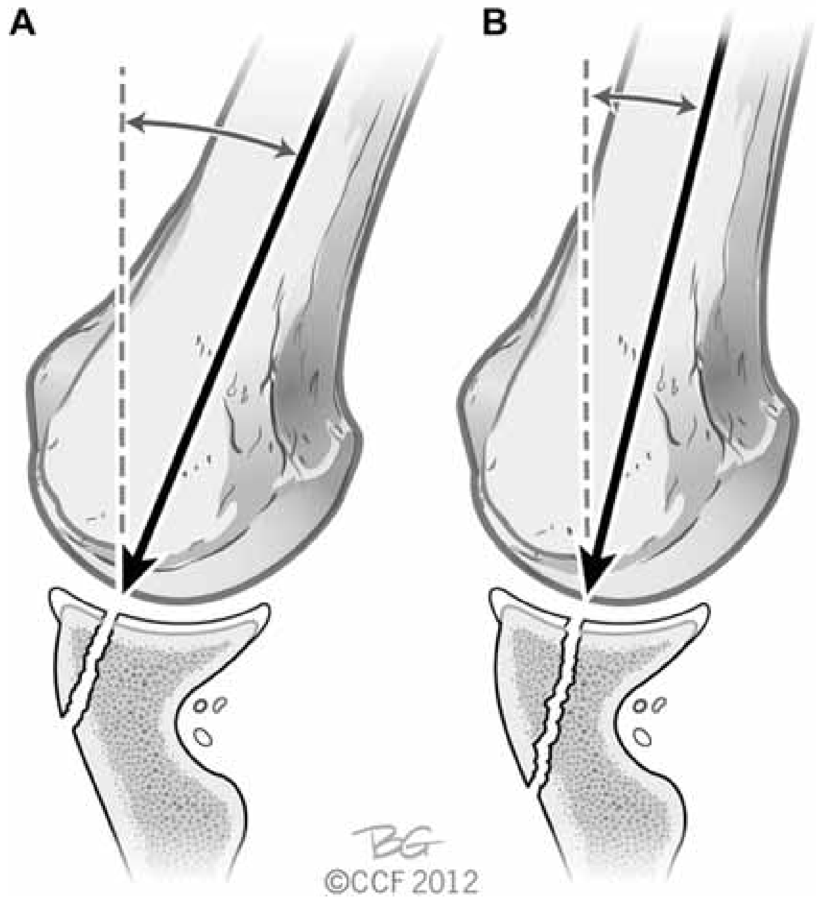

Fig. 2. Mechanism of glenoid fossa fractures. The force vector at the time of impact between the humeral head and glenoid fossa determines the morphology of the glenoid fracture. (*A*) Small rim-type fracture. (*B*) Larger fracture extending into the glenoid vault. (*Courtesy of* Cleveland Clinic Foundation, Cleveland, OH.)

impacts on the anterior aspect of the glenoid rim and can create a Hill-Sachs lesion and/or a bony Bankart lesion.

Richards and colleagues[23] investigated the location and depth of 28 arthroscopically confirmed Hill-Sachs lesions. On an axial view with 0° representing direct anterior, the typical Hill-Sachs lesion lies between 170° and 260° with a midpoint at 209°.[23] Saito and colleagues[24] used axial CT imaging to demonstrate that the normal bare area of the humeral head was located deeper than a typical posterolateral humeral head Hill-Sachs defect, allowing differentiation between the two.

Palmer and Widen[25] and Burkhart, Danaceau, and De Beer[17,26] described an "engaging" Hill-Sachs lesion as one that encounters the anterior glenoid rim with the arm in the "active" position of abduction (90°) and external rotation (0°–135°) and can lever the humerus from the glenoid concavity (**Fig. 3**).[17,25,26] These humeral head defects are parallel to the surface of the anterior glenoid when the arm is abducted and externally rotated.[27] This defect has been termed an articular arc deficit, as there is disruption of the glenohumeral articulation on motion.[17] Lesions that are not parallel to the glenoid rim in the active or athletic position do not engage, and are termed nonengaging lesions.[17,26] The Hill-Sachs defect passes diagonally across the anterior glenoid with external rotation; therefore, there is continual contact of the articulating surfaces and no engagement of the Hill-Sachs lesion by the anterior glenoid.[28]

Cho and colleagues[29] looked at 3D CT scans of 107 shoulders undergoing surgery for recurrent anterior instability to preoperatively predict engagement of a Hill-Sachs lesion. The mean width was 52% (range, 27%–66%) and depth 14% (range, 8%–20%) of the humeral head diameter on axial images. The magnitude of bone loss that coincides with a Hill-Sachs lesion depends on multiple factors including dislocation frequency, chronicity, and force. Cetik and colleagues[30] found an increasing percentage of articular surface involvement with increasing frequency of dislocations. Intraoperative assessment revealed an average involvement of 26% of the articular head in patients with greater than 20 dislocations. The size of the humeral head defect is also directly related to dislocations of longer duration, as seen with neglected and locked shoulder dislocations.[31–33]

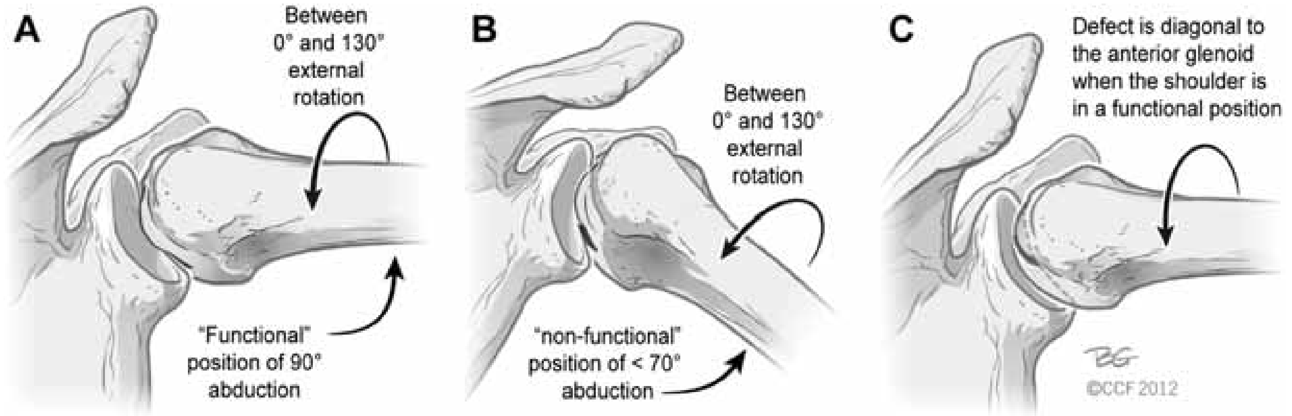

Fig. 3. Engaging and nonengaging Hill-Sachs lesions. (*A*) An engaging lesion is parallel to the anterior glenoid rim when the shoulder is in a functional position. (*B*) The "engagement point" of a nonengaging lesion occurs with the arm in a nonfunctional position. (*C*) In a functional position, a nonengaging lesion is diagonal and nonparallel to the anterior glenoid rim. (*Adapted from* Burkhart SS, De Beer JF. Traumatic glenohumeral bone defects and their relationship to failure of arthroscopic Bankart repairs: significance of the inverted-pear glenoid and the humeral engaging Hill-Sachs lesion. Arthroscopy 2000;16:677–94; *Courtesy of* Cleveland Clinic Foundation, Cleveland, OH.)

The critical limit or threshold of humeral bone loss on glenohumeral stability has been investigated in many ways. In a cadaveric study, Sekiya and colleagues[34] found that defects that were 25% of the humeral head diameter or larger revealed significantly ($P<.05$) less anterior translation before dislocation, and decreased stability ratios (displacing force divided by compressive load) when compared with the intact specimens. Furthermore, Kaar and colleagues[35] noted that defects that were greater or equal to five-eighths of the humeral head radius lead to decreased glenohumeral stability when tested in the functional position of abduction and external rotation.

Hill-Sachs lesions typically are accompanied with other abnormality including soft-tissue and/or bony Bankart lesions and anterior glenohumeral ligament disruption. In the clinical setting, there are essentially 2 types of anterior glenoid defects that occur after an instability event: rim fracture or avulsion, and compression fracture or erosive bone loss. The angle of the humeral head and shaft relative to the glenoid fossa, along with the energy, determine the extent of the resulting glenoid rim fracture (see **Fig. 2**). Recurrent or repetitive subluxations may have more shear and less axial load, leading to attritional bone loss rather than a large rim fracture that may accompany a high-energy axial load. When viewing the glenoid en face, the area of bone defect is nearly parallel to the long axis of the glenoid fossa. Saito and colleagues[36] found the average osseous glenoid injury to range from 12:08 to 6:32 on the clock-face scheme with the midpoint in line with 3:01. However, clinical bone loss can still occur in more anterior-inferior locations.

Glenoid defects are typically classified with large lesions accounting for greater than 20% of the glenoid fossa, medium lesions ranging from 5% to 15%, and small lesions usually less than 5% of the glenoid fossa.[21,37] Itoi and colleagues[38] performed a biomechanical analysis on amount of glenoid defect and force required to translate the humeral head to dislocation. The investigators made sequentially larger glenoid defects in the anterior-inferior glenoid (45° from the longitudinal axis) and found that stability progressively decreased as the size of the glenoid defect increased. Specifically, defects at least 21% of the glenoid length led to instability and limited the range of motion of the shoulder. Similarly, Yamamoto and colleagues[39] looked at anterior glenoid rim defects and found that the stability ratio significantly decreased with defects that were 20% or greater of the glenoid length. Optimal surgical management requires addressing these lesions and management of the clinically significant Hill-Sachs lesion.

The concept of the glenoid track was proposed by Yamamoto and colleagues in 2007 (**Fig. 4**), and serves to illustrate the dynamic of glenohumeral instability in cases of combined defects. The glenoid track represents the pattern of articular contact between the humeral head and the glenoid with the arm in a position of vulnerability for anterior dislocation. The width of the glenoid track was found to be 84% of the inferior glenoid surface. When a glenoid defect exists, the resulting glenoid width is multiplied by 0.84 to calculate the new glenoid track width. If a humeral head defect exists and remains within the glenoid track, there will be no engagement with the anterior glenoid rim.

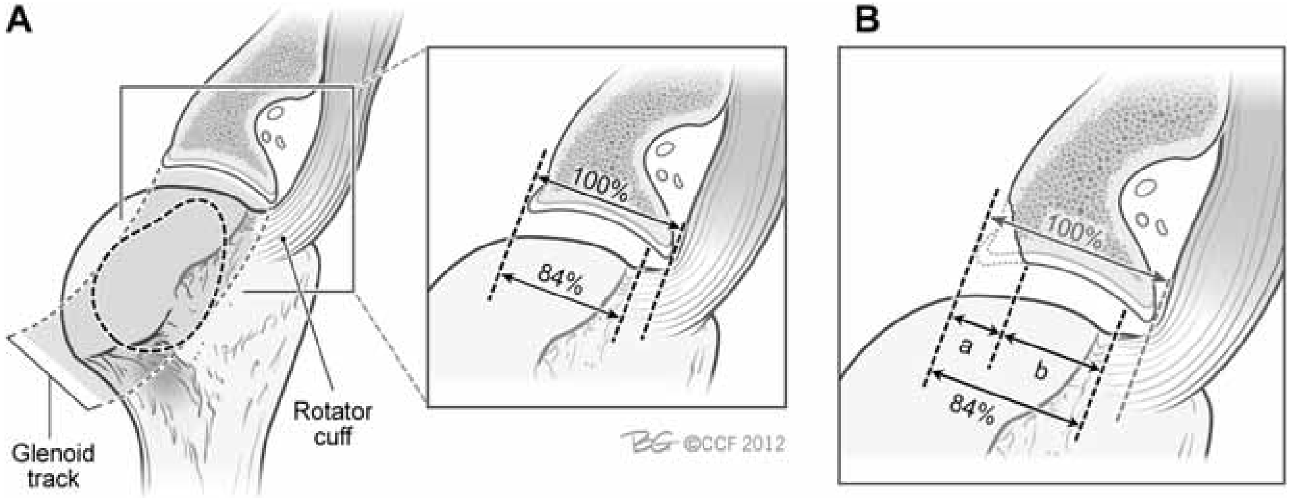

Fig. 4. Glenoid track concept. (*A*) In extremes of external rotation and abduction, the glenoid displaces the cuff tendon close to its footprint, creating a glenoid track that is close to 84% of the glenoid width. (*B*) When a glenoid defect exists, the defect width is subtracted from the 84% width obtained from the normal glenoid to calculate the true glenoid track width. (*Adapted from* Yamamoto N, Itoi E, Hidekazu A, et al. Contact between the glenoid and the humeral head in abduction, external rotation, and horizontal extension: a new concept of glenoid track. J Shoulder Elbow Surg 2007;16:649–56; *Courtesy of* Cleveland Clinic Foundation, Cleveland, OH.)

However, if even a margin of the humeral head defect extends beyond the glenoid track, there is a risk that it will engage the glenoid rim. This concept has proved valuable in understanding the clinical significance of bipolar bone loss.

NATURAL HISTORY

Hovelius and colleagues[40] prospectively followed 229 shoulder dislocations for 25 years. All patients were treated nonoperatively initially and prognostic factors, recurrence, and surgical intervention were monitored. At 10 years, 99 of 185 (53.5%) shoulders that were evaluated with radiographs had evidence of a Hill-Sachs lesion; of these 99 shoulders, 60 redislocated at least once and 51 redislocated at least twice during the 10-year follow-up,[41] compared with 38 (44%) of the 86 shoulders that did not have such a lesion documented (*P*<.04). However, at 25 years, the investigators concluded that a small humeral impression fracture at the time of initial dislocation did not influence the recurrence rate. Rowe and colleagues[13] analyzed the long-term results of Bankart repairs for recurrent instability, and found an overall recurrence rate of 3.4% (5 of 145); the recurrence rates were 4.7% and 6% for patients with moderately severe and severe Hill-Sachs lesions, respectively. Whereas Rowe and colleagues used depths of 3 mm, 5 mm, and greater than 10 mm to differentiate their size of Hill-Sachs lesions, various other methods of determining size and/or volume of the humeral head defect have been proposed without consensus; these include the Hill-Sachs quotient, articular arc circumference, and Hill-Sachs angle.[9,26,29,34,35,42–44]

Lo and colleagues[45] noted that bone loss of 25% or greater of the diameter of the inferior glenoid will create an “inverted pear” appearance, and recommended coracoid transfer when glenoid deficiency reached this magnitude. The inverted-pear glenoid had a poor prognosis in the study by Burkhart and De Beer[26] evaluating a series of 194 patients who underwent primary soft-tissue repair for anterior instability. Of the 21 patients with recurrent instability, 14 had either an engaging Hill-Sachs lesion (n = 3) or an inverted pear glenoid shape (n = 11).

HISTORY AND PHYSICAL EXAMINATION

A thorough orthopedic history must be obtained from the patient regarding shoulder instability. Specifics of the history that must be elucidated include the mechanism of instability and timing of initial symptoms. Arm position and amount of force required for instability may be an evolving process with progressively less rotation or force required for subsequent dislocations. Need and method of reduction of the glenohumeral joint may indicate the extent of laxity present. Presenting symptoms should be noted, including pain, frequency, instability, and level of function. Though infrequent, pertinent medical history including collagen disorders or epilepsy should be noted. Many patients will report a history of recurrent dislocations or multiple surgical attempts to correct the instability. All previous surgical procedures performed on the shoulder should be considered and, if possible, operative reports and photos should be obtained.

Physical examination should focus on inspection for previous scars, gross asymmetry, a thorough comparison of active and passive range of motion, strength testing, particularly evaluation of the integrity and strength of the rotator cuff, and axillary nerve function. The clinician should perform a detailed examination for glenohumeral laxity in the anterior, posterior, and inferior directions. Examination for apprehension should be performed in multiple positions (ie, sitting, standing, supine), as patients with large Hill-Sachs lesions usually exhibit apprehension that often occurs with the arm in significantly less than 90° abduction and 90° external rotation.[9,28] A positive anterior apprehension will be associated with anterior labral injuries. Moreover, apprehension with fewer degrees of abduction may indicate a significant and symptomatic bony contribution to the instability.

IMAGING AND OTHER DIAGNOSTIC STUDIES

The ideal imaging technique is easy to reproduce, with excellent reliability among physicians, and able to predict clinically significant bone defects. In addition to standard radiographs of the shoulder, specialized views allow for evaluation of bony defects of the glenoid and humeral head. Preoperative imaging includes a comprehensive radiographic evaluation with anteroposterior (AP), true AP, axillary, West Point axillary, and Stryker notch views of the involved shoulder (**Fig. 5**).

The Stryker notch view, in addition to AP internal rotation views, has been found to be most sensitive in detecting humeral head lesions on plain radiographs.[19,46] Based on these views, various quantification methods have been described (**Fig. 6**). However, Bois and colleagues[47] note that no method has been universally accepted because of the learning curve required to obtain

Fig. 5. Axillary view of the right shoulder shows congruency of the glenohumeral joint. This view also allows for evaluation of bone loss and glenoid version.

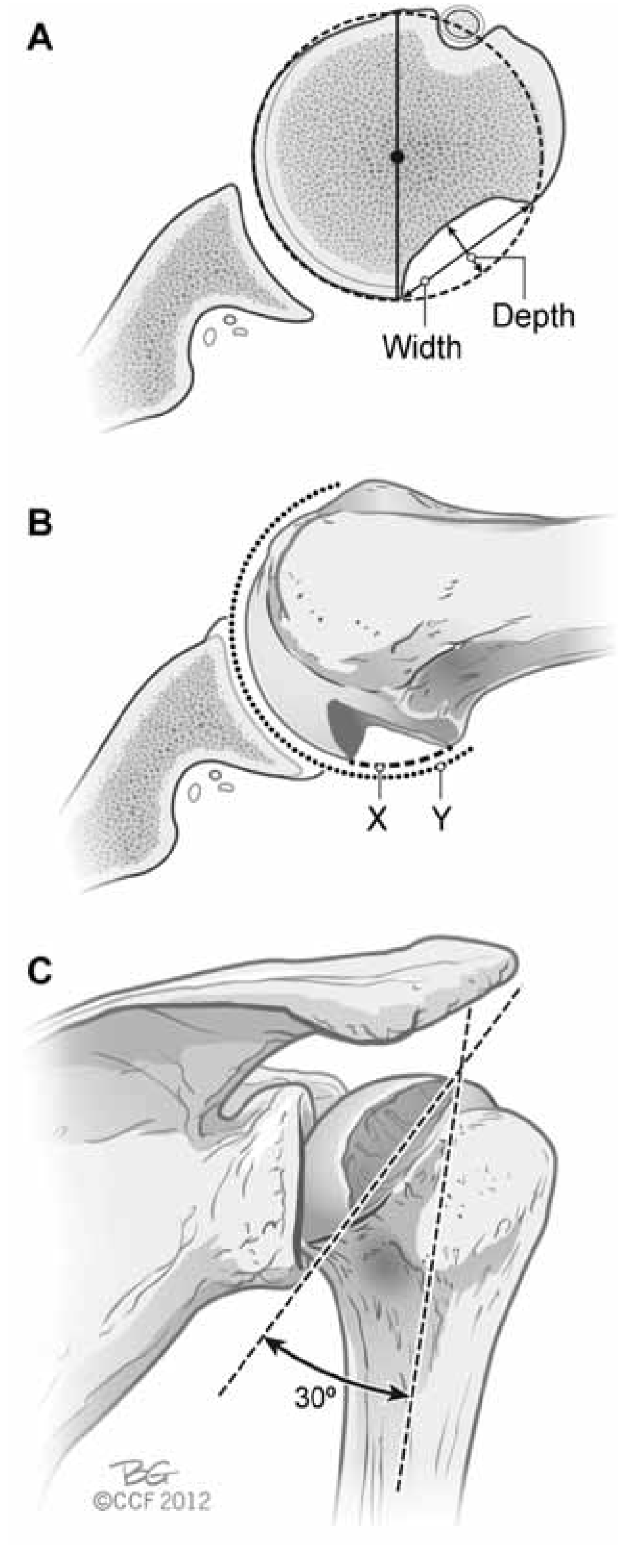

Fig. 6. Methods used to quantify Hill-Sachs lesions. Such defects may be quantified using (*A*) depth or width measurements, (*B*) percentage of humeral head involvement [(X/Y) × 100], and/or (*C*) measurement of the Hill-Sachs angle. (*Courtesy of* Cleveland Clinic Foundation, Cleveland, OH.)

these radiographic projections and the inconsistency often seen in special radiographic views.

Multiple variations of the axillary lateral view have been proposed to evaluate for glenoid defects on plain radiographs (ie, West Point, Bernageau, Garth, and Didiee).[46,48–50] The West Point view has been found to be the most accurate for demonstrating glenoid bone loss.[38]

Despite the vast array of radiographic views available, bone loss may often go undetected on plain radiographs. Preoperative advanced imaging study (CT and/or magnetic resonance imaging [MRI]) are obtained to better define the bony architecture of the glenoid and humeral head (**Fig. 7**). CT can offer the added value of providing better bony detail, with 1-mm slices and 3D reconstructions improving the accuracy of determining the true location and size of the defect.[47] Kodali and colleagues[51] investigated the reliability and accuracy of making width and depth measurements of different-sized Hill-Sachs lesions using axial, sagittal, and coronal 2D CT images. Measurements by 5 physicians were compared with measurements from a 3D laser scanner, and were found to be reproducible and most accurate in the sagittal and axial planes. However, on the glenoid side, in a study performed by Bois and colleagues[52] comparing various 2D and 3D methods of measurement of glenoid bone loss, there was variable agreement and inaccuracy for all 6 observers using 2D CT in measuring defect length and calculating the width/length ratio. Rather, 3D reconstruction is the most reliable and accurate imaging modality for the assessment of glenoid bone loss, and can be a useful tool to more clearly define the size and location of the defect and to estimate the amount of articular surface involved. Quantification of glenoid bone loss can be performed via either a linear method or measurement of surface area. Again, various methods of quantification for both the glenoid and humeral head have been described as being useful in preoperative planning, without universal acceptance.[51,53,54]

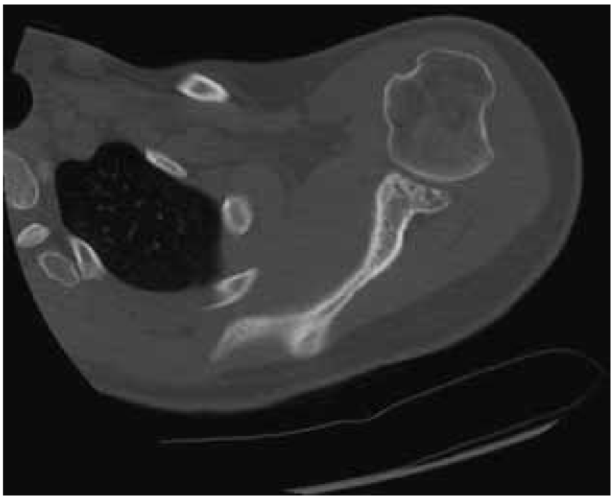

Fig. 7. Axial computed tomography image of the left shoulder showing anterior glenoid bone loss in addition to a large Hill-Sachs defect on the humeral head.

Despite the known advantages of 3D CT imaging techniques, the disadvantages include the financial burden to the institution and possibly the patient, the need for specialized computer software to quantify bone loss, and the lack of awareness in the orthopedic and radiology communities of the multiple measurement methods available and their general validity.[47]

Dynamic arthroscopy remains the gold standard for evaluation of bone loss, and can be useful for the preoperative planning of patients undergoing open osteoarticular allograft reconstruction to address bony deficiency.[29]

CLASSIFICATION OF BONE LOSS

Ideally classification schemes incorporate clinical, radiographic, and prognostic factors. In bone loss with anterior glenohumeral instability, few studies have validated classification schemes (**Table 1**). On the humeral side, Burkhart and De Beer[26] differentiated between engaging and nonengaging defects; clinically, patients with engaging lesions had a higher failure rate with soft-tissue stabilization procedures. This diagnostic sign has since been adopted by most investigators and surgeons as the method of classifying Hill-Sachs lesions. Glenoid defects were classified by Bigliani and colleagues[18] into 3 main types based on the nature of the rim fracture. This classification was later modified after the work of Boileau and colleagues[55] demonstrated a 75% recurrence rate in patients with a glenoid compression fracture and a stretched inferior glenohumeral ligament (**Fig. 8**).

SURGICAL INDICATIONS

Algorithms for the treatment of glenohumeral bone loss associated with anterior instability currently stem from clinical evidence levels IV and V. The lack of quality (level I or II) research limits the validity and generalizability of proposed treatment techniques. Traditionally, direct osseous injuries in instability were addressed with soft-tissue procedures; however, the negative biomechanical effects of bone loss have been correlated with failure and recurrence rates in soft-tissue stabilization procedures. Thus, recent trends toward addressing osseous defects on the glenoid and humeral head have led to greater discussion

Table 1
Classification schemes in bone loss associated with anterior glenohumeral instability

Authors,[Ref.] Year	Basis	Classification		
Humeral Head				
Rowe et al,[42] 1984	Size (length and depth)	Mild 2 × 0.3 cm	Moderate 4 × 0.5 cm	Severe ≥4 × 1.0 cm
Bigliani et al,[99] 1996	Percentage of head involvement	Mild <20%	Moderate 20%–45%	Severe >45%
Glenoid				
Bigliani et al,[18] 1998	Size of rim	Type I A displaced avulsion fracture with attached capsule	Type II A medially displaced fragment malunited to the glenoid rim	Type III Erosion of the glenoid rim with <25% (type IIIA) or >25% (type IIIB) bone loss

among surgeons managing these problems. The following are considered general indications for bone-augmentation procedures in anterior instability, but are neither validated nor widely accepted.

Humeral Head Bone Loss

- Absolute:
  - Displaced humeral head fracture with humeral fracture-dislocation and associated Hill-Sachs injury
  - Lesion greater than 30% to 40% of the humeral head with chronic dislocation or recurrent anterior instability
- Relative:
  - Engaging lesion greater than 20% to 25% of the humeral head
  - Lesion greater than 10% to 25% of the humeral head that does not remain well-centered in the glenoid fossa after arthroscopic instability repair

Glenoid Bone Loss

In most cases of glenoid bone loss, surgery is indicated when nonsurgical management or soft-tissue stabilization has failed to prevent instability and restore function.

- Absolute:
  - Active patients with acute fractures constituting greater than 30% loss of glenoid
- Relative:
  - Young (<25–30 years of age), active (overhead, contact) patients/athletes with bone loss greater than 25% to 30%

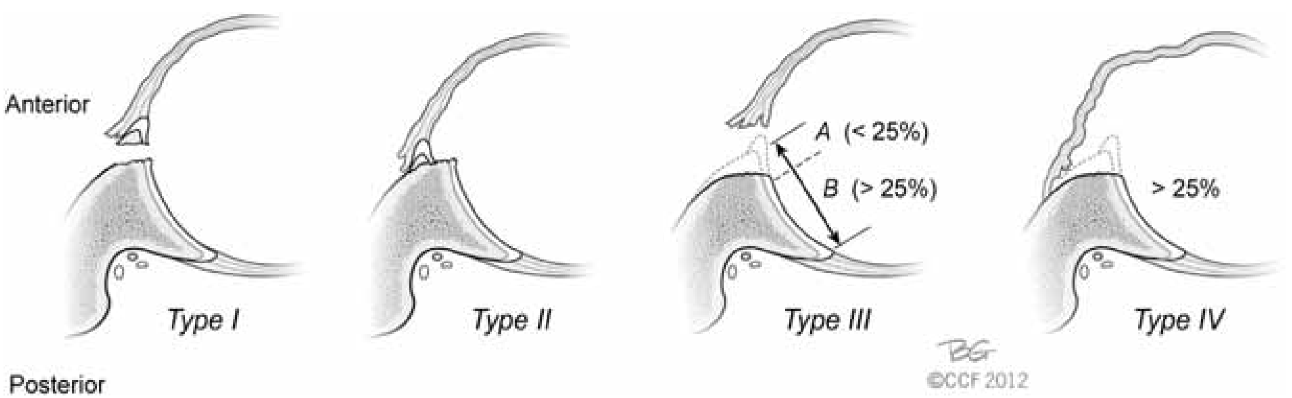

Fig. 8. Glenoid rim lesion types associated with anterior glenohumeral instability. Type I, a displaced avulsion fracture with attached capsule; type II, a medially displaced fragment malunited to the glenoid rim; type III, erosion of the glenoid rim with less than 25% (type IIIA) or greater than 25% (type IIIB) deficiency; type IV, erosion of the glenoid rim with greater than 25% deficiency combined with a stretched inferior glenohumeral ligament. (*Courtesy of* Cleveland Clinic Foundation, Cleveland, OH.)

HUMERAL HEAD TREATMENT OPTIONS

Nonoperative Treatment

Small osseous lesions and nonengaging Hill-Sachs lesions can be managed nonoperatively. Often, combined humeral head and glenoid injuries may be treated by addressing the primary defect alone (ie, Bankart, humeral avulsion of the glenohumeral ligament, or glenoid bone loss). A monitored rehabilitation program focusing on strengthening the dynamic stabilizers (deltoid, rotator cuff, and periscapular muscles) of the shoulder should be started after an initial brief period of immobilization.

Operative Treatment

Several techniques have been described in the literature to address symptomatic engaging Hill-Sachs lesions. Some of these techniques are considered historical and are performed infrequently (rotational osteotomies and east-west plications). Rotational proximal humeral osteotomies externally rotate the proximal humerus, reducing humeral retroversion and minimizing the potential for the defect to engage the anterior glenoid on internal rotation.[27] This technique is essentially of historical interest given the risk of complications and more successful alternatives.[13,17,26,56,57] Open anterior procedures, such as an east-west plication or capsulorrhaphy, shift the glenoid track medially and superiorly to limit external rotation, preventing the humeral head defect from engaging.[17,26] These soft-tissue-only techniques may not be adequate in the setting of a large humeral head defect; furthermore, concerns with restricted motion in young patients may prevent return to function and cause late arthrosis.[58]

Most surgical bone-augmentation procedures include the following:

1. Humeroplasty or disimpaction may be possible in the acute (<3 weeks) setting
2. Remplissage: Transfer of the infraspinatus into the defect to render the lesion essentially extra-articular[59,60]
3. Humeral head augmentation using either osteochondral bone plugs or size-matched bulk allograft transfers can be used to restore native anatomy
4. Humeral head augmentation with a prosthetic cap matched to defect size
5. In severe or failed reconstructive cases, prosthetic replacement using a hemiarthroplasty or total shoulder arthroplasty may become necessary[61]
6. Reconstruction of the anterior glenoid, even in cases without bone loss, to lengthen the glenoid articular arc to prevent engagement[62,63]

Bone-augmentation procedures have typically been performed as open surgery; however, the role of arthroscopic examination of the joint can prove vital in addressing associated disorder, particularly in cases of recurrent instability.

In acute injuries (<3 weeks) humeroplasty, or humeral head disimpaction, may be an option. Although this is a relatively new technique that requires further clinical and biomechanical research, it may be able to restore anatomy in cases with less than 40% of articular surface involved. The procedure can be performed in open fashion or, more commonly, percutaneously, and involves using a tamp or kyphoplasty balloon (Kyphon, Sunnyvale, CA, USA) to disimpact the humeral head lesion. Early results have shown promise. Stachowicz and colleagues[64] performed percutaneous balloon humeroplasties in 18 cadaveric shoulder Hill-Sachs lesions and regained 99.3% of the volume of initial defect. Kazel and colleagues[65] performed humeroplasties with tamps in cadaveric humeri in which they created Hill-Sachs lesions, and were able to reduce the lesions from -1755 mm^3 to -50 mm^3.

In French *remplissage* means "to fill." In shoulder instability with humeral bone loss, this term means to transfer a tendon into the humeral head defect, effectively turning the defect into an extra-articular defect with soft-tissue coverage to prevent engagement with the anterior glenoid rim (**Fig. 9**). Remplissage was originally described by Connolly as an open procedure by filling the Hill-Sachs lesion via transfer of the infraspinatus tendon with a portion of greater tuberosity.[60] An all-arthroscopic technique was first described by Wolf and Pollack,[66] which involved a posterior capsulodesis and infraspinatus tenodesis with transfer into the humeral head defect in conjunction with standard anteroinferior glenoid repair. This procedure is typically reserved for large Hill-Sachs lesions defects with associated glenoid loss of less than 25%; larger glenoid defects would require a conversion to open Latarjet. This approach was modified by Koo and colleagues[67] by using a double-pulley technique whereby 2 anchors were used to insert the infraspinatus tendon into the humeral head defect. This method allowed for the sutures to be tied over the tendon rather than through the tendon or on the muscle belly, allowing a more anatomic and tissue-preserving construct that is biomechanically stronger. Elkinson and colleagues[68] studied the effect of different anchor positions with the remplissage technique in a cadaveric model. Their biomechanical analysis showed that of the various suture techniques, medial suture passage through the infraspinatus muscle belly consistently had the greatest mean

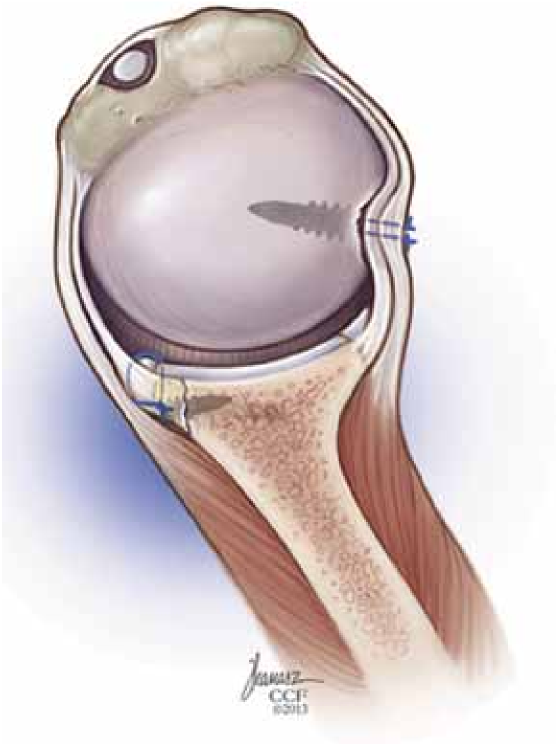

Fig. 9. Remplissage technique for humeral bone loss. The infraspinatus tendon is transferred into the humeral head defect, effectively turning the defect into an extra-articular defect with soft-tissue coverage to prevent engagement with the anterior glenoid rim. (*Courtesy of* Cleveland Clinic Foundation, Cleveland, OH.)

restriction of range of motion and highest stiffness value.

Despite criticism for the technique's nonanatomic nature and potential for loss of motion and subsequent revision surgery, clinical outcomes have been relatively successful. Early studies reported a 7% (2 of 24) incidence of recurrent instability with no loss of motion in any plane at 2-year follow-up.[56] Zhu and colleagues[69] evaluated 49 consecutive patients with a minimum 2-year follow-up. Patients had an increased mean of 8° of forward elevation with only an average loss of 1.9° of external rotation. Boileau and colleagues[70] studied 47 patients with a mean of 24-month follow-up who underwent arthroscopic remplissage. There was an average deficit of 8° (±7°) of external rotation and 9° (±7°) abduction, which was not functionally limiting. Of the 41 patients who participated in athletics before surgery, 37 (90%) returned to sport with 28 (68%) returning to the same level of sport, including overhead sports. A systematic review evaluated 7 studies (levels II, III, IV) of combined arthroscopic remplissage with Bankart repair with an average 26 month follow-up and a pooled rate of recurrent dislocation of 3.4%.[71] The investigators concluded there was no clinically significant loss of range of motion after remplissage. Furthermore, in 4 of the 7 studies postoperative imaging showed high rates of healing and tissue filling at the infraspinatus tenodesis. Similarly, an MRI investigation of 11 patients at an average follow-up of 18 months found evidence of tendon incorporation into humeral head defect as early as 8 months.[72]

Restoring the articular arc through anatomic allograft reconstruction has been described in young patients without osteoporosis or degenerative joint disease who meet the surgical indications.[28] There are 2 main categories of allograft reconstructions: osteochondral plug transfer and size-matched bulk graft. Only 2 case reports exist in the literature describing the technique of osteochondral plug transfer into the base of a humeral defect, both reporting good results after 12 months of follow-up.[73,74] The bulk graft reconstruction requires a size- and side-matched osteoarticular humeral head allograft, preferably a fresh-frozen cryopreserved graft, for optimal recreation of the radius of curvature of the humeral head (±2 mm). An extended deltopectoral approach and capsulotomy is made to expose, inspect, and address any abnormality at the anteroinferior capsulolabral complex and glenoid. The Hill-Sachs lesion is identified and osteotomized in a chevron fashion (Fig. 10). The matching allograft is then cut to fit the site of the humeral head osteotomy and is secured with countersunk screws in lag fashion.

Miniaci and Gish[9] and Miniaci and Martineau[28] reviewed 18 patients who underwent this procedure (16 fresh-frozen grafts, 2 irradiated grafts) after failing previous attempts at surgical stabilization, with an average follow-up of 50 months (range 24–96 months). There were no episodes of recurrent instability, and 16 of 18 (89%) patients returned to work. The average Constant score was 78.5 postoperatively while the WOSII, a validated quality-of-life scale specific to shoulder instability, decreased, indicating improvement. Complications included radiographic evidence of partial graft collapse in 2 of 18 patients, early evidence of osteoarthritis in 3 patients (marginal osteophytes), and 1 mild subluxation (posterior).[9,28] Furthermore, 2 patients required reoperation within 2 years to remove irritable screws. Diklic and colleagues[75] treated 13 patients with fresh-frozen femoral head allograft reconstructions for Hill-Sachs lesions between 25% and 50% of the humeral head. At an average of 54 months postoperatively, the mean Constant score for the cohort was 86.8. Twelve patients had stable shoulders and 1 patient had evidence of osteonecrosis. More long-term and higher-quality research is needed with allograft reconstructions, but may be limited because of the

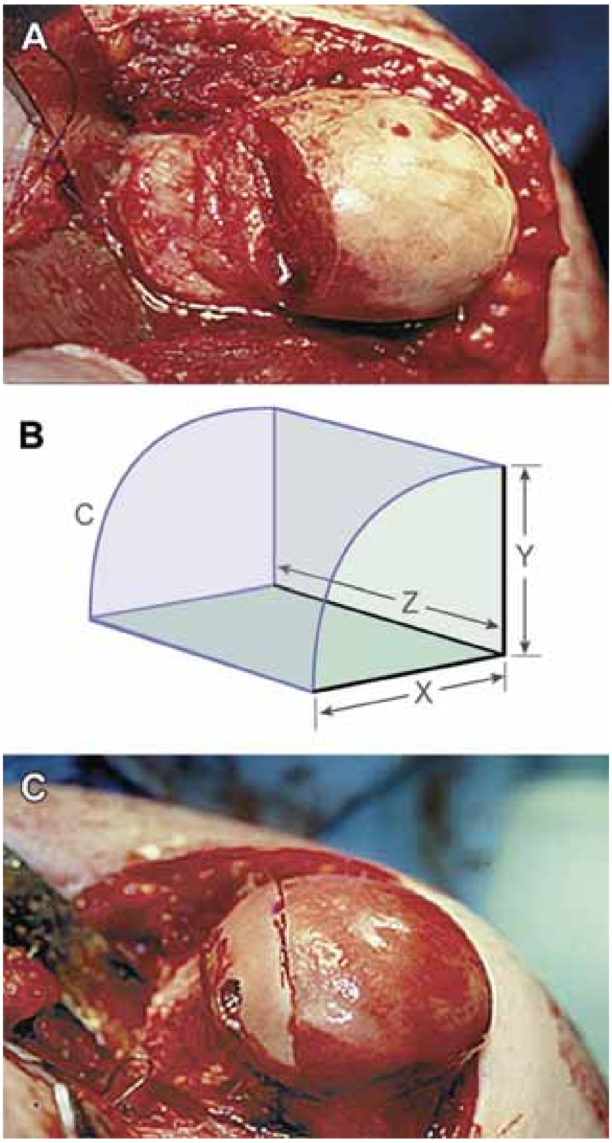

Fig. 10. (*A*) The Hill-Sachs lesion is identified and osteotomized in a chevron fashion. (*B*) The osteotomy is sized in a 3-dimensional pattern. (*C*) The matching allograft is then cut to fit the humeral head osteotomy site and secured with screws.

narrow indications and the required technical expertise.

Reconstituting the articular arc with prosthetic surface implants is another option. This technique uses a round cap-like cobalt-chrome articular component that fills the Hill-Sachs lesion on the posterosuperior humeral head (**Fig. 11**). The technique requires technical expertise and accuracy similar to those of allograft reconstruction without the associated potential complications of disease transmission, nonunion, and graft resorption.[27] Though not described in the limited literature, the use of prosthetic components potentially introduces elements of adverse reactions, hardware loosening, and glenoid wear. Moros and Ahmad[76] described a case report of a 50-year-old man with recurrent anterior shoulder instability and an engaging Hill-Sachs lesion. A Latarjet coracoid transfer and a partial humeral head resurfacing were performed with a successful result at 10-year follow-up. In another case series, Grondin and Leith[77] reported on 2 cases whereby a bony Bankart and a large Hill-Sachs were treated with a Latarjet procedure and partial humeral head resurfacing. In these 2 cases instability was reduced with the procedure, but only short-term follow-up was provided. In 2009, Raiss and colleagues[78] performed uncemented resurfacing arthroplasty in a series of 10 patients with chronic locked anterior shoulder dislocations with large Hill-Sachs defects. At mean follow-up of 24 months, the Constant score increased from 20 points preoperatively to 61 postoperatively ($P<.007$). There were 2 reoperations: one patient developed glenoid erosion and the other had a dislocation. Postoperative radiographs showed the humeral head centered on the glenoid in 9 of the 10 cases, and there were no signs of loosening appreciated.

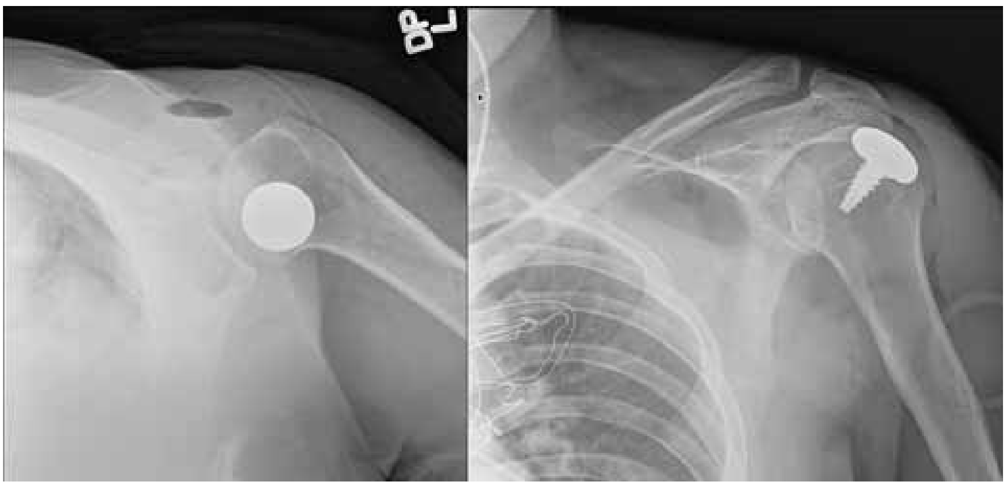

Fig. 11. Two radiographic views of prosthetic implant filling the void created by a large posterosuperior Hill-Sachs defect.

Partial resurfacing may be contraindicated in patients with osteoporosis or deficient bone stock. Scalise and colleagues[79] recommend sufficient quantity and quality of bone in the epiphyseal portion of the humerus to allow stable fixation of the implant, and suggest caution for its use in patients with severe Hill-Sachs lesions associated with chronic locked dislocations. Moreover, Copeland and colleagues[80] suggest a minimum of 60% of normal bone stock for a humeral resurfacing procedure. Elderly patients with osteoporotic bone and large defects (>40% of the humeral head) may have better outcomes with a stemmed prosthesis regardless of a degenerative joint.[32,81] Similarly, Armitage and colleagues[27] recommend a partial resurfacing in small to moderate lesions, but cautioned that future studies are needed.

As stated earlier, complete humeral head resurfacing or isolated humeral head arthroplasty (hemiarthroplasty) is an option in patients with Hill-Sachs defects greater than 40% of the articular surface.[61] Hemiarthroplasty or total shoulder arthroplasty (TSA) (if there is concomitant glenoid wear/erosion) may be particularly beneficial in elderly patients or those who are of low demand; however, indications are not well defined. In younger or more active patients, hemiarthroplasty or TSA should be used with caution because the likelihood of revision increases secondary to glenoid erosion, component wear, and loosening.[82] Pritchett and Clark[61] reported their outcomes of hemiarthroplasty and TSA in 7 patients with chronic dislocations and significant Hill-Sachs lesions. The average patient age was 55 years (range, 36–67 years) and average follow-up was only 2 years. Five of the 7 patients had good results, and there were no recurrent dislocations. These procedures should be reserved for older or less active patients with defects involving greater than 40% of the articular surface and/or significant degeneration of articular cartilage. Further research is needed to outline indications for age and activity levels.

Reconstruction of the anterior glenoid with bone augmentation has been used as the primary procedure for treatment of a Hill-Sachs lesion in recurrent anterior instability. The concept is to lengthen the glenoid articular arc to prevent engagement with the humeral head defect.[62,63]

GLENOID DEFECT TREATMENT OPTIONS

Nonoperative Treatment

As discussed earlier, small osseous lesions may be treated with monitored therapy focusing on strengthening the dynamic stabilizers. Hovelius and colleagues[40] followed the natural history of 229 shoulder dislocations for 25 years, and found that half of the patients between the ages of 12 to 25 years did not experience recurrent instability. Operative treatment of anterior shoulder instability has typically been reserved for large glenoid defects, extending past 25% of the surface area.[40,83,84]

Operative Treatment

Helfet[85] initially described a procedure known as the Bristow, whereby 1 cm of the distal coracoid and the conjoined tendon were transferred through a slit in the subscapularis on the anterior neck of the scapula. The transfer used suture fixation through the conjoined and subscapular tendons. A dynamic buttress was created across the anterior aspect of the glenoid to enhance shoulder stability in abduction and external rotation. Of the 30 patients reported, only 1 experienced continued instability after the coracoid bone-block transfer.[85] Schroder and colleagues[86] reported their findings on 52 Bristow procedures at an average follow-up of 26.4 years. In this cohort, 5 shoulders subsequently had dislocations, with 3 additional shoulders experiencing recurrent instability. Overall, 70% of the patients showed good to excellent results. However, the study only had 10 patients with clinical and radiographic follow-up, and found high rates of glenohumeral arthritis and loss of external rotation in 4 of 11 patients. Schauder and Tullos[87] reported that isolated Bristow results have been only 50% successful at preventing shoulder instability. Therefore, continued modifications to the Bristow procedure have been suggested and made to improve stability and decrease the rate of glenohumeral arthritis.

A similar bone-block technique was described by Latarjet to provide stability for anterior shoulder instability. The coracoid was secured to the anterior glenoid at the medial scapular neck using screw fixation. Since the early studies on bone block for anterior instability, several techniques have emerged for the Latarjet harvest, alignment, and fixation. Regardless of these techniques, Patte[88] described the location of the bone block being flush with the anterior glenoid rim as being a critical element in stabilization. To restore the articular concavity and contact pressures of the glenohumeral joint while avoiding ongoing instability and/or arthrosis, placement of the bone block at the level of the native glenoid fossa is necessary.[89]

Nevertheless, few high-quality studies have reported the results of Latarjet procedures. Allain and colleagues[90] found no instability at 14.3 years of follow-up in a series of 95 cases, but did note that 34 (37%) patients had glenohumeral arthritis.

Based on these findings they concluded that lateral placement of the bone block was a risk factor for higher rates of osteoarthritis. Walch and colleagues[91] found similar results on 160 patients who underwent a Latarjet with minimum 3-year follow-up. The investigators reported persistent instability at only 1%, but found that a laterally displaced coracoid graft led to higher rates of osteoarthritis.

In a study assessing clinical instability without radiographic review, Burkhart and colleagues[92] reported their findings on a modification of the original Latarjet by using the inferior surface of the coracoid in 102 patients, and found recurrent instability in 5 patients with a mean follow-up of 59 months. Hovelius and colleagues[93] reported outcomes of 118 patients at 15-year follow-up. One patient had recurrent shoulder instability within 2 years of follow-up; however, at final follow-up 14 patients had a dislocation or subluxation event. Nevertheless, 98% of the patients were very satisfied and/or satisfied with their results.

In 1948, Palmer and Widen reported their outcomes on the Hybbinette-Eden anterior bone-block procedure that used iliac crest autografting to prevent engagement of the Hill-Sachs lesion and recurrent instability. The idea of using the inner table of the iliac crest was that it would better match the articular contour and better restore the contact pressures within the glenohumeral joint.[94] Niskanen and colleagues[95] described a modification to the iliac crest technique, known as the Alvik glenoplasty, using press fixation at the anterior glenoid. Although the recurrence rate was 21%, there were degenerative changes within 52% of the shoulders. This finding demonstrates the importance of graft fixation and location on restoring the biomechanics of the glenohumeral joint in long-term outcomes.

Warner and colleagues[96] analyzed 11 cases with bony reconstruction for anterior glenoid bone loss at a mean follow-up of 33 months. CT scans with 3D reconstructions were obtained at 4 to 6 months postoperatively to demonstrate union of the bone graft. American Shoulder and Elbow Surgeons scores improved from 65 to 94, University of California Los Angeles scores improved from 33 to 18, and Rowe scores improved from 28 to 94. Two patients had pain with overhead activities, with no cases of recurrent instability.

However, Moroder and colleagues[97] studied the clinical and radiologic outcome of iliac crest autograft for anterior shoulder instability with glenoid bone loss in 9 patients with a mean follow-up of 34.6 months. Two patients reported the recurrence of instability and demonstrated a positive apprehension test. The overall glenoid surface increased 6.4% compared with preoperative findings, leading the investigators to suspect graft osteolysis and subsequent clinical instability. Moroder and colleagues[98] used a J-bone graft in 20 patients with a CT evaluation at 1-year follow-up, noting that the J-bone graft overcorrected the glenoid concavity and subsequently normalized as a result of remodeling processes.

Investigators continue to explore different types of bone blocks and fixation methods to restore optimal glenohumeral biomechanics. Further biomechanical and long-term follow-up studies are needed to not only reduce anterior shoulder instability but also decrease glenohumeral arthritis.

REFERENCES

1. Zacchilli MA, Owens BD. Epidemiology of shoulder dislocations presenting to emergency departments in the United States. J Bone Joint Surg Am 2010;92(3):542–9.
2. Hovelius L. Incidence of shoulder dislocation in Sweden. Clin Orthop Relat Res 1982;(166):127–31.
3. Mazzocca AD, Cote MP, Solovyova O, et al. Traumatic shoulder instability involving anterior, inferior, and posterior labral injury: a prospective clinical evaluation of arthroscopic repair of 270 degrees labral tears. Am J Sports Med 2011;39(8):1687–96.
4. Bollier MJ, Arciero R. Management of glenoid and humeral bone loss. Sports Med Arthrosc 2010;18(3):140–8.
5. Ghodadra N, Gupta A, Romeo AA, et al. Normalization of glenohumeral articular contact pressures after Latarjet or iliac crest bone-grafting. J Bone Joint Surg 2010;92(6):1478–89.
6. Greis PE, Scuderi MG, Mohr A, et al. Glenohumeral articular contact areas and pressures following labral and osseous injury to the anteroinferior quadrant of the glenoid. J Shoulder Elbow Surg 2002;11(5):442–51.
7. Matsen F 3rd, Chebli C, Lippitt S, American Academy of Orthopaedic Surgeons. Principles for the evaluation and management of shoulder instability. J Bone Joint Surg Am 2006;88:648–59.
8. Montgomery WH Jr, Wahl M, Hettrich C, et al. Anteroinferior bone-grafting can restore stability in osseous glenoid defects. J Bone Joint Surg 2005;87(9):1972–7.
9. Miniaci A, Gish MW. Management of anterior glenohumeral instability associated with large Hill–Sachs defects. Tech Shoulder Elbow Surg 2004;5(3):170–5.
10. Flower WH. On the pathological changes produced in the shoulder-joint by traumatic dislocation: as derived from an examination of all the

specimens illustrating this Injury in the Museums of London. Trans Pathol Soc London 1861;12:179.
11. Hill HA, Sachs MD. The grooved defect of the humeral head a frequently unrecognized complication of dislocations of the shoulder joint. Radiology 1940;35(6):690–700.
12. Calandra JJ, Baker CL, Uribe J. The incidence of Hill-Sachs lesions in initial anterior shoulder dislocations. Arthroscopy 1989;5(4):254–7.
13. Rowe CR, Patel D, Southmayd WW. The Bankart procedure: a long-term end-result study. J Bone Joint Surg Am 1978;60(1):1–16.
14. Saupe N, White LM, Bleakney R, et al. Acute traumatic posterior shoulder dislocation: MR findings. Radiology 2008;248(1):185–93.
15. Taylor DC, Arciero RA. Pathologic changes associated with shoulder dislocations. Arthroscopic and physical examination findings in first-time, traumatic anterior dislocations. Am J Sports Med 1997;25(3):306–11.
16. Yiannakopoulos CK, Mataragas E, Antonogiannakis E. A comparison of the spectrum of intra-articular lesions in acute and chronic anterior shoulder instability. Arthroscopy 2007;23(9):985–90.
17. Burkhart SS, Danaceau SM. Articular arc length mismatch as a cause of failed Bankart repair. Arthroscopy 2000;16(7):740–4.
18. Bigliani LU, Newton PM, Steinmann SP, et al. Glenoid rim lesions associated with recurrent anterior dislocation of the shoulder. Am J Sports Med 1998;26(1):41–5.
19. Edwards TB, Boulahia A, Walch G. Radiographic analysis of bone defects in chronic anterior shoulder instability. Arthroscopy 2003;19(7):732–9.
20. Griffith JF, Antonio GE, Yung PS, et al. Prevalence, pattern, and spectrum of glenoid bone loss in anterior shoulder dislocation: CT analysis of 218 patients. AJR Am J Roentgenol 2008;190(5):1247–54.
21. Sugaya H, Moriishi J, Dohi M, et al. Glenoid rim morphology in recurrent anterior glenohumeral instability. J Bone Joint Surg Am 2003;85-A(5):878–84.
22. Widjaja AB, Tran A, Bailey M, et al. Correlation between Bankart and Hill-Sachs lesions in anterior shoulder dislocation. ANZ J Surg 2006;76(6):436–8.
23. Richards RD, Sartoris DJ, Pathria MN, et al. Hill-Sachs lesion and normal humeral groove: MR imaging features allowing their differentiation. Radiology 1994;190(3):665–8.
24. Saito H, Itoi E, Minagawa H, et al. Location of the Hill-Sachs lesion in shoulders with recurrent anterior dislocation. Arch Orthop Trauma Surg 2009;129(10):1327–34.
25. Palmer I, Widen A. The bone block method for recurrent dislocation of the shoulder joint. J Bone Joint Surg Br 1948;30B(1):53–8.
26. Burkhart SS, De Beer JF. Traumatic glenohumeral bone defects and their relationship to failure of arthroscopic Bankart repairs: significance of the inverted-pear glenoid and the humeral engaging Hill-Sachs lesion. Arthroscopy 2000;16(7):677–94.
27. Armitage MS, Faber KJ, Drosdowech DS, et al. Humeral head bone defects: remplissage, allograft, and arthroplasty. Orthop Clin North Am 2010;41(3):417–25.
28. Miniaci A, Martineau PA. Humeral head bony deficiency (large Hill-Sachs). In: El Attrache NS, editor. Surgical Techniques in Sports Medicine. Philadelphia: Lippincott Williams & Wilkins; 2006.
29. Cho SH, Cho NS, Rhee YG. Preoperative analysis of the Hill-Sachs lesion in anterior shoulder instability: how to predict engagement of the lesion. Am J Sports Med 2011;39(11):2389–95.
30. Cetik O, Uslu M, Ozsar BK. The relationship between Hill-Sachs lesion and recurrent anterior shoulder dislocation. Acta Orthop Belg 2007;73(2):175–8.
31. Loebenberg MI, Cuomo F. The treatment of chronic anterior and posterior dislocations of the glenohumeral joint and associated articular surface defects. Orthop Clin North Am 2000;31(1):23–34.
32. Flatow EL, Miller SR, Neer CS 2nd. Chronic anterior dislocation of the shoulder. J Shoulder Elbow Surg 1993;2(1):2–10.
33. Kirtland S, Resnick D, Sartoris DJ, et al. Chronic unreduced dislocations of the glenohumeral joint: imaging strategy and pathologic correlation. J Trauma 1988;28(12):1622–31.
34. Sekiya JK, Wickwire AC, Stehle JH, et al. Hill-Sachs defects and repair using osteoarticular allograft transplantation biomechanical analysis using a joint compression model. Am J Sports Med 2009;37(12):2459–66.
35. Kaar SG, Fening SD, Jones MH, et al. Effect of humeral head defect size on glenohumeral stability a cadaveric study of simulated Hill-Sachs defects. Am J Sports Med 2010;38(3):594–9.
36. Saito H, Itoi E, Sugaya H, et al. Location of the glenoid defect in shoulders with recurrent anterior dislocation. Am J Sports Med 2005;33(6):889–93.
37. d'Elia G, Di Giacomo A, D'Alessandro P, et al. Traumatic anterior glenohumeral instability: quantification of glenoid bone loss by spiral CT. Radiol Med 2008;113(4):496–503 [in English, Italian].
38. Itoi E, Lee SB, Amrami KK, et al. Quantitative assessment of classic anteroinferior bony Bankart lesions by radiography and computed tomography. Am J Sports Med 2003;31(1):112–8.
39. Yamamoto N, Itoi E, Abe H, et al. Effect of an anterior glenoid defect on anterior shoulder stability: a cadaveric study. Am J Sports Med 2009;37(5):949–54.

40. Hovelius L, Olofsson A, Sandstrom B, et al. Nonoperative treatment of primary anterior shoulder dislocation in patients forty years of age and younger. a prospective twenty-five-year follow-up. J Bone Joint Surg Am 2008;90(5):945–52.
41. Hovelius L, Augustini BG, Fredin H, et al. Primary anterior dislocation of the shoulder in young patients. A ten-year prospective study. J Bone Joint Surg Am 1996;78(11):1677–84.
42. Rowe C, Zarins B, Ciullo J. Recurrent anterior dislocation of the shoulder after surgical repair. Apparent causes of failure and treatment. J Bone Joint Surg Am 1984;66(2):159.
43. Kralinger FS, Golser K, Wischatta R, et al. Predicting recurrence after primary anterior shoulder dislocation. Am J Sports Med 2002;30(1):116–20.
44. Chen AL, Hunt SA, Hawkins RJ, et al. Management of bone loss associated with recurrent anterior glenohumeral instability. Am J Sports Med 2005;33(6):912–25.
45. Lo IKY, Parten PM, Burkhart SS, et al. The inverted pear glenoid: an indicator of significant glenoid bone loss. Arthroscopy 2004;20:169–74.
46. Pavlov H, Warren RF, Weiss CB Jr, et al. The roentgenographic evaluation of anterior shoulder instability. Clin Orthop Relat Res 1985;(194):153–8.
47. Bois AJ, Walker RE, Kodali P, et al. Imaging instability in the athlete: the right modality for the right diagnosis. Clin Sports Med 2013;32(4):653–84.
48. Garth WP Jr, Slappey CE, Ochs CW. Roentgenographic demonstration of instability of the shoulder: the apical oblique projection. A technical note. J Bone Joint Surg Am 1984;66(9):1450–3.
49. Bernageau J, Patte D, Debeyre J, et al. Value of the glenoid profile in recurrent luxations of the shoulder. Rev Chir Orthop Reparatrice Appar Mot 1976;62(2 suppl):142–7 [in French].
50. Rokous JR, Feagin JA, Abbott HG. Modified axillary roentgenogram. A useful adjunct in the diagnosis of recurrent instability of the shoulder. Clin Orthop Relat Res 1972;82:84–6.
51. Kodali P, Jones MH, Polster J, et al. Accuracy of measurement of Hill-Sachs lesions with computed tomography. J Shoulder Elbow Surg 2011;20(8):1328–34.
52. Bois AJ, Fening SD, Polster J, et al. Quantifying glenoid bone loss in anterior shoulder instability: reliability and accuracy of 2-dimensional and 3-dimensional computed tomography measurement techniques. Am J Sports Med 2012;40(11):2569–77.
53. Baudi P, Righi P, Bolognesi D, et al. How to identify and calculate glenoid bone deficit. Chir Organi Mov 2005;90(2):145–52 [in English, Italian].
54. Burkhart SS, Debeer JF, Tehrany AM, et al. Quantifying glenoid bone loss arthroscopically in shoulder instability. Arthroscopy 2002;18(5):488–91.
55. Boileau P, Villalba M, Hery JY, et al. Risk factors for recurrence of shoulder instability after arthroscopic Bankart repair. J Bone Joint Surg Am 2006;88(8):1755–63.
56. Purchase RJ, Wolf EM, Hobgood ER, et al. Hill-Sachs "remplissage": an arthroscopic solution for the engaging hill-sachs lesion. Arthroscopy 2008;24(6):723–6.
57. Weber B, Simpson L, Hardegger F, et al. Rotational humeral osteotomy for recurrent anterior dislocation of the. J Bone Joint Surg Am 1984;66:1443–50.
58. Bigliani LU, Weinstein DM, Glasgow MT, et al. Glenohumeral arthroplasty for arthritis after instability surgery. J Shoulder Elbow Surg 1995;4(2):87–94.
59. Yagishita K, Thomas BJ. Use of allograft for large Hill–Sachs lesion associated with anterior glenohumeral dislocation: a case report. Injury 2002;33(9):791–4.
60. Connolly RS. Humeral head defects associated with shoulder dislocations: their diagnostic and surgical significance. Instr Course Lect 1972;21:42–54.
61. Pritchett JW, Clark JM. Prosthetic replacement for chronic unreduced dislocations of the shoulder. Clin Orthop Relat Res 1987;216:89–93.
62. Balg F, Boileau P. The instability severity index score. A simple pre-operative score to select patients for arthroscopic or open shoulder stabilisation. J Bone Joint Surg Br 2007;89(11):1470–7.
63. Millett PJ, Clavert P, Warner JJ. Open operative treatment for anterior shoulder instability: when and why? J Bone Joint Surg Am 2005;87(2):419–32.
64. Stachowicz RZ, Romanowski JR, Wissman R, et al. Percutaneous balloon humeroplasty for Hill-Sachs lesions: a novel technique. J Shoulder Elbow Surg 2013;22:e7–13.
65. Kazel MD, Sekiya JK, Greene JA, et al. Percutaneous correction (humeroplasty) of humeral head defects (Hill-Sachs) associated with anterior shoulder instability: a cadaveric study. Arthroscopy 2005;21(12):1473–8.
66. Wolf EM, Pollack ME. Hill-Sachs "remplissage": an arthroscopic solution for the engaging Hill-Sachs lesion. Arthroscopy 2004;20(Suppl 1):e14–5.
67. Koo SS, Burkhart SS, Ochoa E. Arthroscopic double-pulley remplissage technique for engaging Hill-Sachs lesions in anterior shoulder instability repairs. Arthroscopy 2009;25(11):1343–8.
68. Elkinson I, Giles JW, Boons HW, et al. The shoulder remplissage procedure for Hill-Sachs defects: does technique matter? J Shoulder Elbow Surg 2013;22(6):835–41.
69. Zhu YM, Lu Y, Zhang J, et al. Arthroscopic Bankart repair combined with remplissage technique for the treatment of anterior shoulder instability with

engaging Hill-Sachs lesion: a report of 49 cases with a minimum 2-year follow-up. Am J Sports Med 2011;39(8):1640–7.
70. Boileau P, O'Shea K, Vargas P, et al. Anatomical and functional results after arthroscopic Hill-Sachs remplissage. J Bone Joint Surg Am 2012;94(7):618–26.
71. Leroux T, Bhatti A, Khoshbin A, et al. Combined arthroscopic Bankart repair and remplissage for recurrent shoulder instability. Arthroscopy 2013;29:1693–701.
72. Park MJ, Garcia G, Malhotra A, et al. The evaluation of arthroscopic remplissage by high-resolution magnetic resonance imaging. Am J Sports Med 2012;40(10):2331–6.
73. Chapovsky F, Kelly JD 4th. Osteochondral allograft transplantation for treatment of glenohumeral instability. Arthroscopy 2005;21(8):1007.
74. Kropf EJ, Sekiya JK. Osteoarticular allograft transplantation for large humeral head defects in glenohumeral instability. Arthroscopy 2007;23(3):322.e1–5.
75. Diklic ID, Ganic ZD, Blagojevic ZD, et al. Treatment of locked chronic posterior dislocation of the shoulder by reconstruction of the defect in the humeral head with an allograft. J Bone Joint Surg Br 2010;92(1):71–6.
76. Ahmad CS, Moros C. Partial humeral head resurfacing and latarjet coracoid transfer for treatment of recurrent anterior glenohumeral instability. Orthopaedics 2009;32(8):602.
77. Grondin P, Leith J. Combined large Hill–Sachs and bony Bankart lesions treated by Latarjet and partial humeral head resurfacing: a report of 2 cases. Can J Surg 2009;52(3):249.
78. Raiss P, Aldinger PR, Kasten P, et al. Humeral head resurfacing for fixed anterior glenohumeral dislocation. Int Orthop 2009;33(2):451–6.
79. Scalise JJ, Miniaci A, Iannotti JP. Resurfacing arthroplasty of the humerus: Indications, surgical technique, and clinical results. Tech Shoulder Elbow Surg 2007;8:152–60.
80. Copeland S. The continuing development of shoulder replacement: "reaching the surface". J Bone Joint Surg Am 2006;88:900–5.
81. Flatow EL, Warner JI. Instability of the shoulder: complex problems and failed repairs: Part I. Relevant biomechanics, multidirectional instability, and severe glenoid loss. Instr Course Lect 1998;47:97–112.
82. Denard PJ, Raiss P, Sowa B, et al. Mid- to long-term follow-up of total shoulder arthroplasty using a keeled glenoid in young adults with primary glenohumeral arthritis. J Shoulder Elbow Surg 2013;22(7):894–900.
83. Hovelius L, Sandstrom B, Olofsson A, et al. The effect of capsular repair, bone block healing, and position on the results of the Bristow-Latarjet procedure (study III): long-term follow-up in 319 shoulders. J Shoulder Elbow Surg 2012;21(5):647–60.
84. Hovelius L, Sandstrom B, Saebo M. One hundred eighteen Bristow-Latarjet repairs for recurrent anterior dislocation of the shoulder prospectively followed for fifteen years: study II-the evolution of dislocation arthropathy. J Shoulder Elbow Surg 2006;15(3):279–89.
85. Helfet AJ. Coracoid transplantation for recurring dislocation of the shoulder. J Bone Joint Surg Br 1958;40-B(2):198–202.
86. Schroder DT, Provencher MT, Mologne TS, et al. The modified Bristow procedure for anterior shoulder instability: 26-year outcomes in Naval Academy midshipmen. Am J Sports Med 2006;34(5):778–86.
87. Schauder KS, Tullos HS. Role of the coracoid bone block in the modified Bristow procedure. Am J Sports Med 1992;20(1):31–4.
88. Patte D, Debeyre J. Luxations recidivantes de l'epaule. Encycl Med Chir Paris. Tech Chir Orthop 1980;44265. 4.4–02 [in French].
89. Lynch JR, Clinton JM, Dewing CB, et al. Treatment of osseous defects associated with anterior shoulder instability. J Shoulder Elbow Surg 2009;18(2):317–28.
90. Allain J, Goutallier D, Glorion C. Long-term results of the Latarjet procedure for the treatment of anterior instability of the shoulder. J Bone Joint Surg Am 1998;80(6):841–52.
91. Walch G, Boileau P. Latarjet-Bristow procedure for recurrent anterior instability. Tech Shoulder Elbow Surg 2000;1:256–61.
92. Burkhart SS, De Beer JF, Barth JR, et al. Results of modified Latarjet reconstruction in patients with anteroinferior instability and significant bone loss. Arthroscopy 2007;23(10):1033–41.
93. Hovelius L, Sandstrom B, Sundgren K, et al. One hundred eighteen Bristow-Latarjet repairs for recurrent anterior dislocation of the shoulder prospectively followed for fifteen years: study I–clinical results. J Shoulder Elbow Surg 2004;13(5):509–16.
94. Hutchinson MR, Dall BE. Midline fascial splitting approach to the iliac crest for bone graft. A new approach. Spine 1994;19(1):62–6.
95. Niskanen RO, Lehtonen JY, Kaukonen JP. Alvik's glenoplasty for humeroscapular dislocation. 6-year follow-up of 52 shoulders. Acta Orthop Scand 1991;62(3):279–83.
96. Warner JJ, Gill TJ, O'Hollerhan JD, et al. Anatomical glenoid reconstruction for recurrent anterior glenohumeral instability with glenoid deficiency using an autogenous tricortical iliac crest bone graft. Am J Sports Med 2006;34(2):205–12.

97. Moroder P, Blocher M, Auffarth A, et al. Clinical and computed tomography-radiologic outcome after bony glenoid augmentation in recurrent anterior shoulder instability without significant glenoid bone loss. J Shoulder Elbow Surg 2014;23:420–6.
98. Moroder P, Hitzl W, Tauber M, et al. Effect of anatomic bone grafting in post-traumatic recurrent anterior shoulder instability on glenoid morphology. J Shoulder Elbow Surg 2013;22(11):1522–9.
99. Bigliani LU, Flatow EL, Pollock RG. Fractures of the proximal humerus. In: Rockwood CA, Green DP, Bucholz RW, et al, editors. Fractures in adults. 4th edition. Philadelphia: Lippincott-Raven; 1996. p. 1055–107.